Textbook and Color Atlas of

# Traumatic Injuries to the Teeth

5th edition

Edited by
Jens O. Andreasen
Frances M. Andreasen
Lars Andersson

WILEY Blackwell

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Fifth Edition

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# Preface

It is now more than 50 years since the first edition of *Traumatic Injuries of the Teeth* was published. It has served clinicians in providing evidence-based treatment and also provided a complete overview of the scientific literature on traumatic dental injuries. The fourth edition came out in 2007 and this new, fifth, edition contains new information on the role of stem cells. This is presented together with a chapter updating us on research behind the creation of bio-roots attached by a functional periodontal ligament.

Traumatic dental injuries are very frequent and there are now data from almost every country in the world to support this. To reach a higher level of evidence we must, in the future, standardize data collection to enable new meta-analyses. For this reason we have expanded the chapter on classification and epidemiology, with standardized registration of important outcome variables according to the core outcome set as defined by the International Association of Dental Traumatology.

New data on root fractures and alveolar bone fractures are also included. The chapters on endodontics have been expanded and now also include procedures aimed at regenerative endodontics. In line with this, much emphasis has been placed on pulpal healing responses in the chapters dealing with tooth luxation and horizontal root fractures.

New chapters include those on reinforcing endodontically treated teeth and restoration using porcelain veneers. A special chapter is now devoted to the management of dentoalveolar ankylosis where emphasis is placed on preserving the alveolar bone rather than saving the tooth, which is especially important in the young patient who is still growing. The new interactive program Dental Trauma Guide is based on a database from Copenhagen. It illustrates the principles of diagnosis and treatment of traumatic dental injuries, and is presented in a special chapter. Finally, there is a new chapter presenting the International Association of Dental Traumatology.

We hope this new edition will continue to serve clinicians and researchers in this field in their efforts to provide optimal treatment for the benefit of victims of dental trauma.

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August 2018

# 1

# Wound Healing Subsequent to Injury

F. Gottrup & J. O. Andreasen

## Definition

The generally accepted definition of wound healing is: 'a reaction of any multicellular organism on tissue damage in order to restore the continuity and function of the tissue or organ'. This is a functional definition saying little about the process itself and which factors are influential.

Traumatic dental injuries usually imply wound healing processes in the periodontium, the pulp and sometimes associated soft tissue. The outcome of these determines the final healing result (Fig. 1.1). The general response of soft and mineralized tissues to surgical and traumatic injuries is a sensitive process, where even minor changes in the treatment procedure may have an impact upon the rate and quality of healing.

In order to design suitable treatment procedures for a traumatized dentition, it is necessary to consider the cellular and humoral elements in wound healing. In this respect considerable progress has been made in understanding the role of the different cells involved.

In this chapter the general response of soft tissues to injury is described, as well as the various factors influencing the wound healing processes. For progress to be made in the treatment of traumatic dental injuries it is necessary to begin with general wound healing principles. The aim of the present chapter is to give a general survey of wound healing as it appears from recent research. For more detailed information about the various topics the reader should consult textbooks and review articles devoted to wound healing (1–23, 607–612, 626, 631, 640–647).

## Nature of a traumatic injury

Whenever injury disrupts tissue, a sequence of events is initiated whose ultimate goal is to heal the damaged tissue. The sequence of events after wounding is: control of bleeding; establishing a line of defense against infection; cleansing the wound site of necrotic tissue elements, bacteria or foreign bodies; closing the wound gap with newly formed connective tissue and epithelium; and finally modifying the primary wound tissue to a more functionally suitable tissue.

This healing process is basically the same in all tissues, but may vary clinically according to the tissues involved. Thus wound healing after dental trauma is complicated by the multiplicity of cellular systems involved (Fig. 1.2).

During the last decades, significant advances have been made in the understanding of the biology behind wound healing in general and new details concerning the regulating mechanisms have been discovered.

While a vast body of knowledge exists concerning the healing of cutaneous wounds, relatively sparse information exists concerning the healing of oral mucosa and odontogenic tissues. This chapter describes the general features of wound healing, and the present knowledge of the cellular systems involved. Wound healing as it applies to the specific odontogenic tissues will be described in Chapter 2.

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**Fig. 1.1** Cells involved in the healing event after a tooth luxation. Clockwise from top: endothelial cell and pericytes; thrombocyte (platelet); erythrocyte; fibroblast; epithelial cell; macrophage; neutrophil; lymphocytes; mast cell.

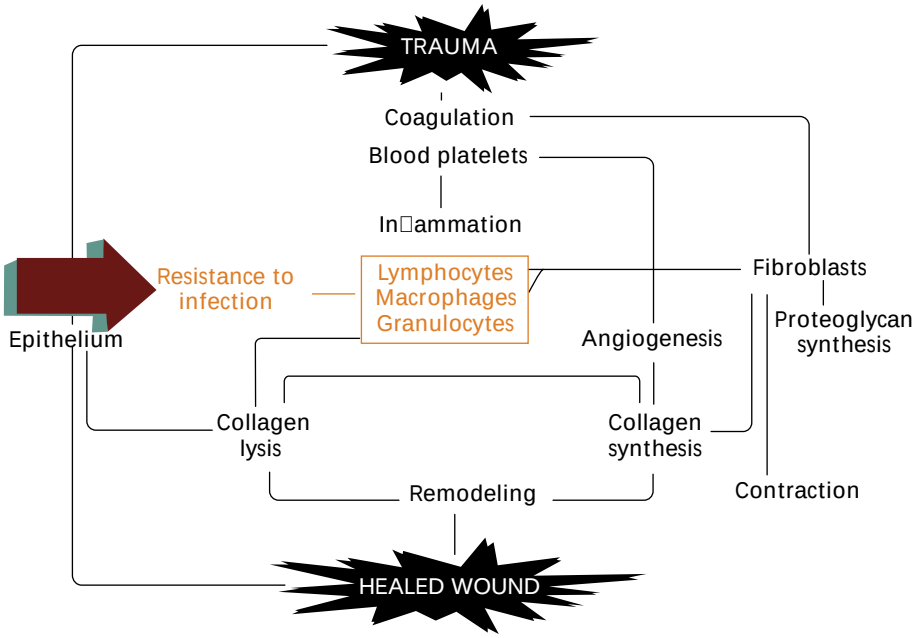


**Fig. 1.2** Modified Hunt flow diagram for wound healing.

## Wound healing biology

Wound healing is a dynamic, interactive process involving cells and extracelluar matrix and is dependent on internal as well as external factors. Different schemes have been used in order to summarize the wound healing process. With increasing knowledge of the involved processes, cell types, etc., a complete survey of all aspects will be hugely difficult to overview. The authors have for many years used a modification of the original Hunt flow diagram for wound healing (19) (Fig 1.2). This diagram illustrates the main events in superficial epithelialization and production of granulation tissue.

The wound healing process will be described in detail in the following section.

### Repair versus regeneration

The goal of the wound healing process after injury is to restore the continuity between wound edges and to re-establish tissue function. In relation to wound healing, it is appropriate to define various terms, such as *repair* and *regeneration*. In this context, it has been suggested that the term *regeneration* should be used for a biologic process by which the structure and function of the disrupted or lost tissue is completely restored, whereas *repair* or scar formation is a biologic process whereby the continuity of the disrupted or lost tissue is regained by new tissue which does not restore structure and function (14). Throughout the text, these terms will be used according to the above definitions. The implication of repair and regeneration as they relate to oral tissues is discussed in Chapter 2.

### Cell differentiation

Cell differentiation is a process whereby an embryonic non-functional cell matures and changes into a tissue-specific cell, performing one or more functions characteristic of that cell population. Examples of this are the *mesenchymal paravascular cells* in the periodontal ligament and the pulp, and the basal cells of the epithelium. A problem arises as to whether already functioning odontogenic cells can revert to a more primitive cell type. Although this is known to take place in cutaneous wounds, it is unsettled with respect to dental tissues (see Chapter 2). With regard to cell differentiation, it appears that extracellular matrix (ECM) compounds, such as proteoglycans, have a significant influence on cell differentiation in wound healing (25).

### Progenitor cells (stem cells)

Among the various cell populations in oral and other tissues, a small fraction are *progenitor cells*. These cells are self-perpetuating, non-specialized cells, which are the source of new differentiating cells during normal tissue turnover and healing after injury (17–19, 24). The role of these in wound healing is further discussed in Chapter 4.

### Cell cycle

Prior to mitosis, DNA must duplicate and RNA be synthesized. Since materials needed for cell division occupy more than half the cell, a cell that is performing functional synthesis (e.g. a fibroblast producing collagen, an odontoblast producing dentin or an epithelial cell producing keratin) does not have

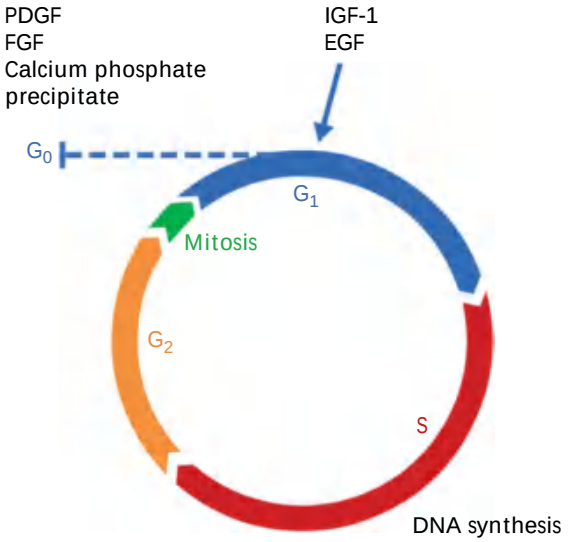


**Fig. 1.3** Cell cycle: $G_0$, resting phase; $G_1$, time before onset of DNA synthesis; S, replication of DNA; $G_2$, time between DNA replication and mitosis.

the resources to undergo mitosis. Conversely, a cell preparing for or undergoing mitosis has insufficient resources to undertake its functions. This may explain why it is usually the least differentiated cells that undergo proliferation in a damaged tissue, and why differentiated cells do not often divide (15).

The interval between consecutive mitoses has been termed the *cell cycle*, which represents an ordered sequence of events that are necessary before the next mitosis (Fig. 1.3). The cell cycle has been subdivided into phases such as $G_1$, the time before the onset of DNA synthesis; in the S phase the DNA content is replicated, $G_2$ is the time between the S phase and mitosis, and M the time of mitosis (Fig. 1.3). The cumulative length of S, $G_2$ and M is relatively constant at 10–12 hours, whereas differences occur among cell types in the duration of $G_1$ (26).

Cells that have become growth arrested enter a resting phase, $G_0$, which lies outside the cell cycle. The $G_0$ state is reversible and cells can remain viable in $G_0$ for extended periods.

*In vivo*, cells can be classified as continuously dividing (e.g. epithelial cells, fibroblasts), non-dividing post-mitotic (e.g. ameloblasts) and cells reversibly growth arrested in $G_0$ that can be induced to re-enter the proliferative cycle.

Factors leading to fibroblast proliferation have been studied in the fibroblast system. Resting cells are made *competent* to proliferate (i.e. entry of $G_0$ cells into early $G_1$ stage) by so-called *competence factors* (i.e. platelet-derived growth factor (PDGF), fibroblast growth factor (FGF) and calcium phosphate precipitates). However, there is no progression beyond $G_1$ until the appearance of progression factors such as insulin-like growth factor-1 (IGF-1), epidermal growth factor (EGF) and other plasma factors (26) (Fig. 1.3).

## Cell migration

Optimal wound repair is dependent upon an orderly influx of cells into the wound area. Directed cell motion requires polarization of cells and formation of both a leading edge that attaches to the matrix and a trailing edge that is pulled along. The stimulus for directional cell migration can be a soluble attractant (*chemotaxis*), a substratum-bound gradient of a particular matrix constituent (*haptotaxis*) or the three-dimensional array of ECM within the tissue (*contact guidance*). Finally there is a *free edge effect* which occurs in epithelial wound healing (27, 28).

Typical examples of cells responding to *chemotaxis* are circulating neutrophils and monocytes and macrophages. The chemoattractant is regulated by diffusion of the attractant from its source into an attraction-poor medium.

Cells migrating by *haptotaxis* extend lamellipodia more or less randomly and each of these protruding lamellipodia competes for a matrix component to adhere to, whereby a leading edge will be created on one side of the cell and a new membrane inserted into the leading edge. In that context, fibronectin and laminin seem to be important for adhesion (27).

*Contact guidance* occurs as the cell is forced along paths of least resistance through the ECM. Thus, migrating cells align themselves according to the matrix configuration, a phenomenon that can be seen in the extended fibrin strands in retracting blood clots, as well as in the orientation of fibroblasts in granulation tissue (29). In this context it should be mentioned that mechanisms also exist whereby spaces are opened within the extracellular area when cells migrate. Thus both fibroblasts and macrophages use enzymes such as plasmin, plasminogen and collagenases for this purpose (30).

During wound repair, a given parenchymal cell may migrate into the wound space by multiple mechanisms occurring concurrently or in succession. Factors related to cell migration in wound healing are described later for each particular cell type.

# Dynamics of wound repair

Classically, the events taking place after wounding can be divided into three phases, namely the *inflammation*, the *proliferation* and the *remodeling phases* (5, 13, 20–23, 31). The inflammation phase may, however, be subdivided into a *hemostasis* phase and an *inflammatory* phase. But, it should be remembered that wound healing is a continuous process where the beginning and end of each phase cannot be clearly determined and phases do overlap.

Tissue injury causes disruption of blood vessels and extravasation of blood constituents. Vasoconstriction provides a rapid, but transient, decrease in bleeding. The extrinsic and intrinsic coagulation pathways are also immediately activated. The blood clots together with vasoconstriction re-establish hemostasis and provide a provisional ECM for cell migration. Adherent platelets undergo morphologic changes to facilitate formation of the hemostatic plug and secrete several mediators of wound healing such as PDGF, which attract and activate macrophages and fibroblasts.

Other growth factors and a great number of other mediators such as chemoattractants and vasoactive substances are also released. The released products soon initiate the inflammatory response.

## Inflammation phase

Following the initial vasoconstriction, a vasodilation takes place in the wound area. This supports the migration of inflammatory cells into the wound area (Fig. 1.4).

These processes take place in the coagulated blood clot placed in the wound cavity. When prothrombin changes to thrombin, cleaving the fibrinogen molecule to fibrin, the clot turns into a fibrin clot, which later becomes the wound crust in open wounds. Fibronolytic activity is, however, also present in this early stage of healing. From plasminogen is produced plasmin which digests fibrin leading to the removal of thrombi. Fibrin has its main effect when angiogenesis starts and the restoration of vascular structure begins.

Neutrophils, lymphocytes and macrophages are the first cells to arrive at the site of injury. Their major role is to guard against the threat of infection, as well as to cleanse the wound site of cellular matrix debris and foreign bodies. The macrophages appear to direct the concerted action of the wound cell team (Fig. 1.4).

## Proliferative phase (fibroplasia)

This is called the *fibroplasia phase* or *regeneration phase* and is a continuation of the inflammatory phase, characterized by fibroblast proliferation and migration and the production of connective tissue. Once fibroblasts have migrated into the granulation tissue, their primary role is to rapidly produce new connective tissue ECM to re-establish tissue strength and function. It appears that fibroblast migration into the wound provisional matrix continues along an increasing but relatively low concentration gradient of a given chemoattractant, e.g. EGF or transforming growth factor (TGF)-band PDGF. It starts about day 2 after the tissue trauma and continues for 2–3 weeks after the trauma in the case of a closed wound. This phase can be extended significantly in the case of an open wound with severe tissue damage, where complete closure will require production of a large amount of connective tissue.

In response to chemoattractants created in the inflammation phase, fibroblasts invade the wound area and this starts the proliferation phase. The invasion of fibroblasts starts at day 2 after injury and by day 4 they are the major cell type in normal healing. Fibroblasts are responsible for replacing the fibrin matrix (clot) with collagen-rich new stroma often called *granulation tissue*. In addition, fibroblasts also produce and release proteoglycans and glycosaminoglycan (GAG), which are important components of the ECM of the granulation tissue. Vascular restoration uses the new matrix as a scaffold and numerous new capillaries endow the new stroma with a granular appearance (angiogenesis). Macrophages provide a continuing source of growth factors necessary to stimulate fibroplasia and angiogenesis. The structural molecules of newly formed ECM, termed *provisional matrix*, produce a scaffold or conduit for cell migration. These molecules include fibrin, fibronectin and hyaluronic acid. Fibronectin and the appropriate integrin receptors bind fibronectin, fibrin or both on fibroblasts, appearing to limit the rate of formation of granulation tissue.

Stimulated by growth factors and other signals, fibroblasts and endothelial cells divide, and cause a capillary network to move into the wound site which is characterized by ischemic-damaged tissue or a coagulum.

The increasing numbers of cells in the wound area induce hypoxia, hypercapnia and lactacidosis, due to the increased need for oxygen in an area with decreased oxygen delivery because of the tissue injury (32, 33).

At cellular level oxygen is an essential nutrient for cell metabolism, especially energy production. This energy is supplied by the coenzyme adenosine triphosphate (ATP), which is the most important store for chemical energy on the molecular/enzymatic level and is synthesized in mitochondria by oxidative phosphorylation. This reaction is oxygen dependent.

*NADPH-linked oxygenase* is the responsible enzyme for the respiratory burst that occurs in leukocytes. During the inflammatory phase of the healing process NADPH-linked oxygenase produces high amounts of oxidants by consuming high amounts of oxygen (34). The first event in wound healing is activation of an NADPH-linked oxidase (630). Successful wound healing can only take place in the presence of the enzyme, because oxidants are required for the prevention of wound infection.

Not only phagocytes, but almost every cell in the wound environment is fitted with a specialized enzyme to convert $O_2$ to *reactive oxygen species* (ROS), including oxidizing species such as free radicals and hydrogen peroxide ($H_2O_2$) (35, 36). These ROS act as cellular messengers to promote several important processes that support wound healing. Thus $O_2$ has a role in healing beyond its function as nutrient and antibiotic. Given the growth factors, such as PDGF, require ROS for their action on cells (35, 37), it is clear that $O_2$ therapy may act as an effective adjunct. Clinically this has been found in chronic granulomatous disease (CGD) where there are defects in genes that encode NADPH oxidase. The manifestations of this defect are increased susceptibility to infection and impaired wound healing (625).

Simultaneously, the basal cells in the epithelium divide and move into the injury site, thereby closing the defect. Along with revascularization, new collagen is formed which, after 3–5 days, adds strength to the wound. The high rate of collagen production continues for 10–12 days, resulting in strengthening of the wound. At this time healing tissue is dominated by capillaries and immature collagen.

The fibroblasts are responsible for the synthesis, deposition and remodeling of the ECM, which conversely can have an influence on the fibroblast activities. Cell movements at this stage into the fibrin clot or tightly woven ECM seem to require an active proteolytic system that can cleave a path for cell migration. Fibroblast-derived enzymes (collagenase,

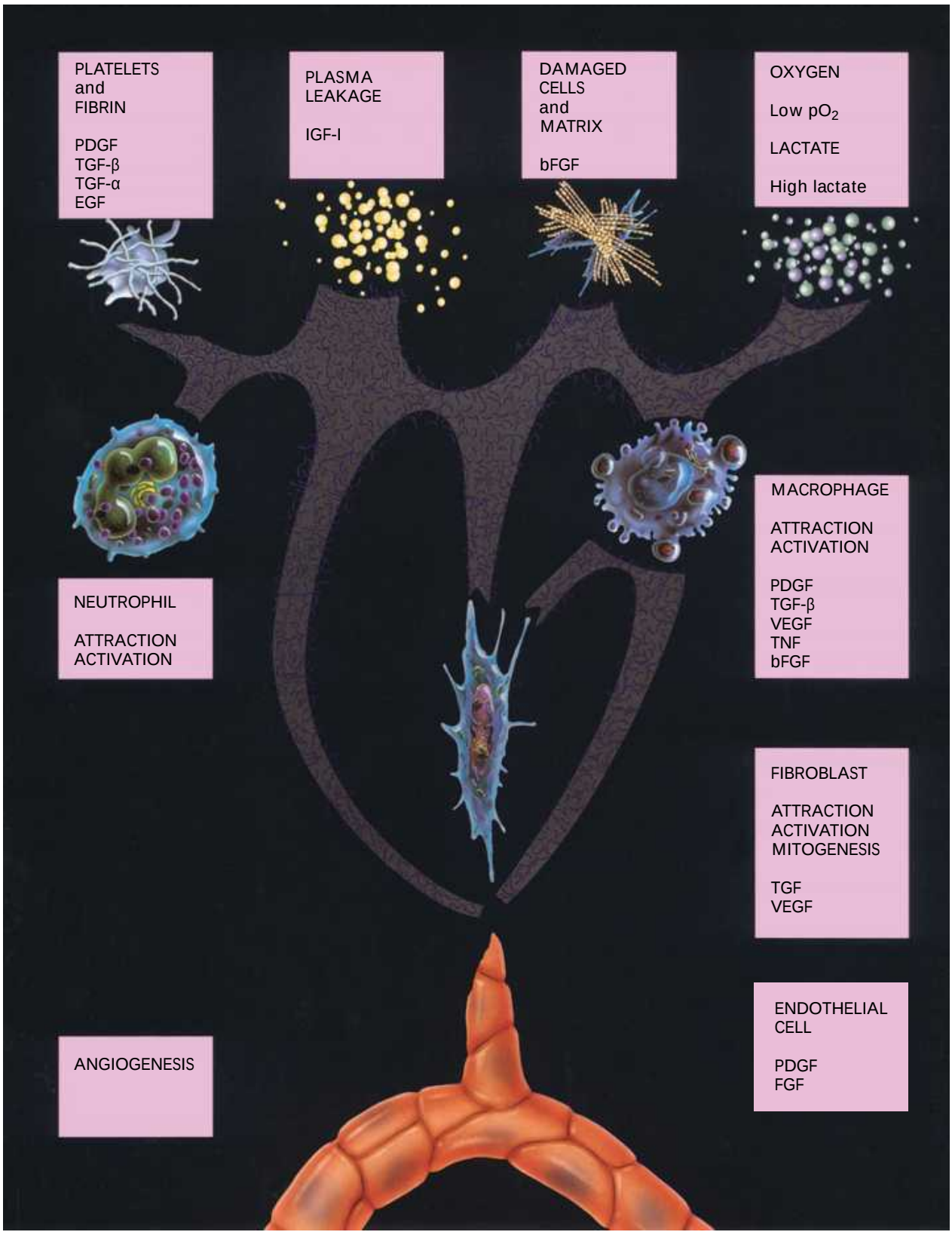


**Fig. 1.4** Cellular components and mediators in the wound healing module. Signals for wound healing are released by platelets, fibrin, plasma leakage, damaged cells and matrix. Furthermore low oxygen tension and a high lactate concentration in the injury site contribute an important stimulus for healing.

gelatinase A, etc.) and serum plasmin are potential candidates for this task (23).

After fibroblast migration into the wound cavity, the provisional ECM is gradually replaced with collagenous matrix. It appears that fibroblast migration into the wound provisional matrix continues along an increasing, but relatively low, concentration gradient of a given chemoattractant, e.g. EGF or TGF-band PDGF. New connective tissue begins to form approximately 2–4 days after wounding, and it is called granulation tissue due to its granular appearance when examined visually. Once an abundant collagen matrix has been deposited in the wound, the fibroblasts stop producing collagen, and the fibroblast-rich granulation tissue is replaced by a relatively acellular scar. Cells in the wound undergo apoptosis (cell death) triggered by unknown signals, but doing so the fibroblast dies without raising an inflammatory response. Deregulation of these processes occurs in fibrotic disorders such as keloid formation, morphea and scleroderma. Collagen synthesis and secretion requires hydroxylation of proline and lysine residues. Sufficient blood flow delivering adequate molecular oxygen is pivotal for this process.

Collagen production/deposition and development of strength of the wound is directly correlated to the partial pressure $pO_2$ of the tissue ($p_tO_2$) (38–40). Synthesis of collagen, crosslinking and the resulting wound strength relies on the normal function of specific enzymes (41, 42). The function of these enzymes is directly related to the amount of oxygen present, e.g. hydrolyzation of proline and lysine by hydroxylase enzymes (43).

Recently it has been shown that oxygen also may trigger the differentiation of fibroblasts to myofibroblasts, cells responsible for wound contraction (44).

### *Neovascularization/angiogenesis*

Early in the healing process there is no vascular supply to the injured area, but the stimulus for angiogenesis is present: growth factors released by especially macrophages, low oxygen and elevated lactate. The angiogenesis starts the day after the lesion. Angiogenesis is complicated, involving endothelial cells and activated epidermal cells. Proteolytic enzymes degrade the endothelial basement membrane allowing endothelial cells from the surroundings of the wound area to proliferate, migrate and form new vessels. The establishment of new blood vessels occurs by the budding or sprouting of intact venoles and the sprouts meet in loops (see p. 32) (259, 377). The presence of capillary loops within the provisional matrix provides the tissue with a red granular appearance. Once the wound is filled with new granulation tissue, angiogenesis ceases and many of the blood vessels disintegrate as a result of apoptosis. Angiogenesis is dependent upon the ECM (623, 624).

While hypoxia can initiate neovascularization, it cannot sustain it. Supplementary oxygen administration accelerates vessels' growth (35, 45). Vascular endothelial growth factor (VEGF) has been established as a major long-term angiogenetic stimulus at the wound site. Recently, the cell response to hypoxia has been further elucidated. Hypoxia inducible factor 1 (HIF-1) has been identified as a transcription factor that is induced by hypoxia (46, 48).

In the presence of normal oxygen tensions HIF-1 transcriptional activity is ubiquinated and degraded (47). HIF-1 seems to upregulate genes involved in glucose metabolism and angiogenesis under hypoxia and in a model of myocardial and cerebral ischemia the factor seems to protect cells from damage. The exact molecular mechanisms of how hypoxia is sensed by the cells are still unknown.

The arrangement of cells in the proliferative phase has been examined in rabbits using ear chambers where wounds heal between closely approximated, optically clear membranes (33, 49). It appears from these experiments that macrophages infiltrate the tissue in the dead space, followed by immature fibroblasts. New vessels are formed next to these fibroblasts that synthesize collagen. This arrangement of cells, which has been termed the *wound healing module*, continues to migrate until the tissue defect is obliterated. The factors controlling the growth of the wound healing module are described on p. 32.

### *Epithelialization*

Re-epithelialization of wounds begins within hours after injury. Within 24 hours after wounding, epithelial cells at the margin of the wound dissolve their hemidesmosomal adhesions and show the first signs of migration. In 48 hours, proliferation starts behind the leading edge, seeding more cells into the wound site. Epithelial cells migrate through the fibrin–fibronectin provisional matrix until they contact the front of leading cells coming from the other side of the wound (626).

If parts of the dermis layers are intact, epidermal cells from skin appendages such as hair follicles quickly remove clotted blood and damaged stroma and cover the wound space. This results in fast epithelialization. If the dermis is totally destroyed, the epithelialization only takes place from the wound edges and epithelialization can continue for a considerable time dependent on wound area.

The trauma of being wounded causes an activation of epithelial keratinocytes by exposure to the pro-migratory matrix molecules, growth factors and cytokines that are released, and wound-generated electrical fields. During epithelialization the cells undergo considerable phenotypic alteration including retraction of intracellular tonofilaments, dissolution of most intercellular desmosomes and formation of peripheral cytoplasmatic actin filaments, which allow cell movement. Furthermore, the cells no longer adhere to one another and the basement membrane. This allows migration of the cells dissecting the wound and separating scar from viable tissue. Integrin expression of the migrating epidermal cells appears to determine the path of dissection (23). Epidermal cell migration between collagenous dermis and the fibrin scar requires degradation of ECM. This is achieved by production of proteinases (collagenases, e.g. matrix metalloproteinase-1) and activation of plasmin by activators produced by epidermal cells. In well-adapted, non-complicated

surgical incisional wounds the first layers of epidermal cells move over the incisional line 1–2 days after suturing. At the same time, epidermal cells at the wound margin in open wounds begin to proliferate behind the actively migrating cells. The stimulus for migration and proliferation of epidermal cells is unknown, but the absence of neighbor cells at the margin of the wound (free edge effect), local release of growth factors and increased expression of growth factor receptors may be a suggestion.

During dermal migration from the wound margin, a basement membrane reappears in a zip-like fashion and hemidesmosomes and type VII collagen anchoring fibrils form. Epidermal cells firmly attached to the basement membrane and underlying dermis reverts to normal phenotype.

The production of epithelial tissue is primarily dependent on the degree of hydration and oxygen. While a moist wound environment increases the rate of epithelialization by a factor of two or three (50, 51), the optimal growth of epidermal cells is found at an oxygen concentration of 10–50% (52–54).

### *Wound contraction*

Wound contraction is a complex process, and beneficial because a portion of the lesion is covered by skin despite scar tissue and thus it decreases complications by decreasing the open skin wound area. In human skin, contraction can account for about 50% of wound closure, but in rodents it is more extensive and makes up to 90% of the wound closure (631). During the second week of healing, fibroblasts assume a myofibroblast phenotype characterized by large bundles of actin-containing microfilaments (55). The stimulus for contraction probably is a combination of growth factors, integrin attachment of the myofibroblasts to collagen matrix and crosslinks between collagen bundles (23). Wound contraction seems to be related to the early wound healing period and the effect decreases in time; in chronic unclosed wounds, no wound contraction exists.

### *Scar contracture*

As opposed to the process of wound contraction of skin edges, this is a late pathologic process in wound healing. It consists of a contraction of large amounts of scar tissue followed by immobilization of the affected area (e.g. a joint). In scar contracture, the wound area as well as adjacent tissue shrinks, as opposed to contraction where only the wound area is involved. The morbidity of scar contracture is a major problem in the rehabilitation of severely injured patients.

## Remodeling phase

The *remodeling phase* is also called the *moderation phase* or the *scar phase*.

The wound remodeling phase turns the abundant and poorly organized granulation tissue ECM into a mature connective tissue. Remodeling starts when wound contraction has assembled the collagen fibrils into thicker bundles and aligned them perpendicularly to the wound edges. During the remodeling stage, collagen crosslinking also gradually increases, improving the stability of the tissue, and there is a gradual maturation of the tissue so that the aligned collagen fiber bundles are reorganized to the typical and more resilient basketweave organization found in normal connective tissue (628). Remodeling continues slowly and can last for months or in some cases for years.

In closed wounds this phase starts 2–3 weeks after closure, while it does not start in open wounds before the wound has healed. Granulation tissue covered by epidermis is known to undergo remodeling earlier than uncovered granulation tissue. The length of this phase is unknown; some have argued 1 year but others have claimed the rest of the patient's life.

During this phase the granulation tissue is remodeled and matured to a scar formation. When granulation tissue is covered by epithelium it undergoes remodeling. Similarly, a wound covered by a graft will continue the remodeling phase. This results in a decrease in cell density, numbers of capillaries and metabolic activity (55). The collagen fibrils will be united into thicker fiber bundles. There is a major difference between dermis and scar tissue in the arrangement of collagen fiber bundles. In scar tissue, as in granulation tissue, they are organized in arrays parallel to the surface, while in dermis they are more in a basketweave pattern (21). This difference results in a more rigid scar tissue. The collagen composition change from granulation tissue to scar tissue, where there is collagen type III, decreases from 30% to 10%. In the remodeling phase the biomechanical strength of a scar increases slightly, despite no extra collagen being produced. This increase relates primarily to a better architectural organization of the collagen fiber bundles.

The epidermis of a scar differs from normal skin by lacking the rete pegs, which are anchored within the underlying connective tissue matrix (21). Furthermore there is no regeneration of lost subepidermal appendages such as hair follicles or sweat glands in a scar.

# Types of wound after injury

Wounds can be divided into different types, according to healing and associated wound closure methods (56–58) (Fig. 1.5). This distinction is based on practical treatment regimens while the basic biological wound healing sequences are similar for all wound types.

*Primary healing*, or healing by *first intention*, occurs when wound edges are anatomically accurately opposed and healing proceeds without complication. This type of wound heals with a good cosmetic and functional result and with a minimal amount of scar tissue. These wounds, however, are sensitive to complications, such as infection.

*Secondary healing*, or healing by *second intention*, occurs in wounds associated with tissue loss or when wound edges are not accurately opposed. This type of healing is usually

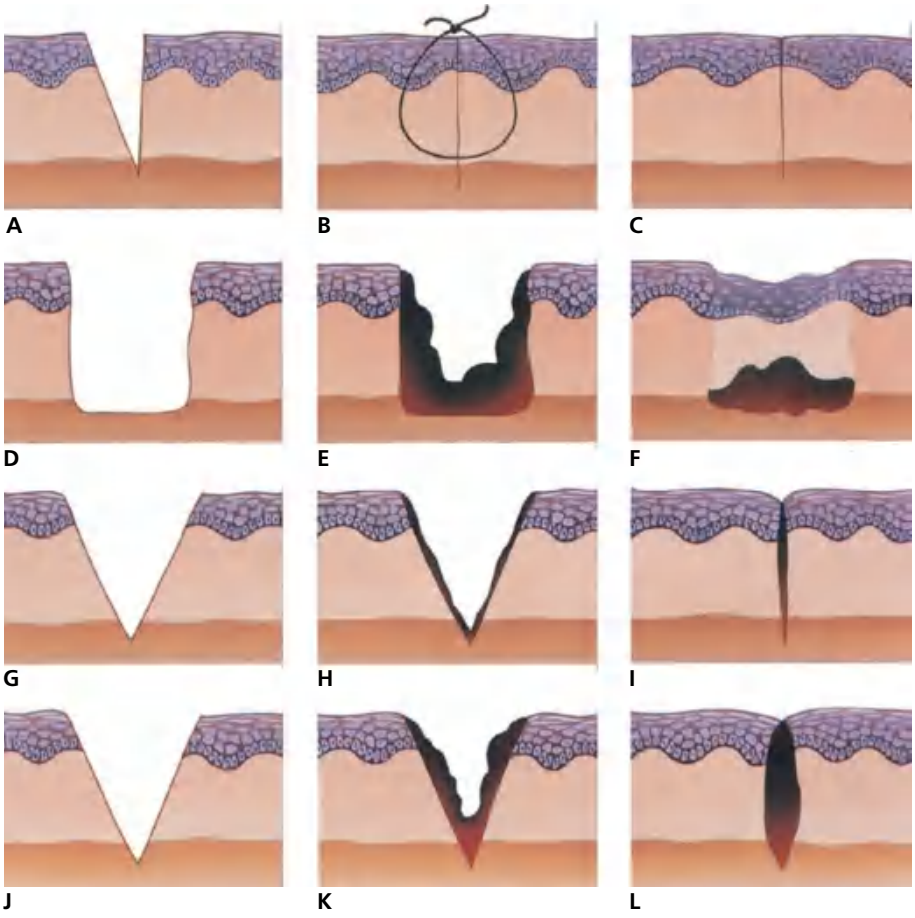


**Fig. 1.5** Wound healing events related to the type of wound and subsequent treatment. A–C. Incisional wound with primary closure. D–F. Open and non-sutured wound. G–I. Delayed primary closure. J–L. Secondary closure. From (56).

the natural biologic process that occurs in the absence of surgical intervention. The defect is gradually filled by granulation tissue and a considerable amount of scar tissue will be formed despite an active contraction process. The resulting scar is less functional and often sensitive to thermal and mechanical injury. Furthermore, this form of healing requires considerable time for epithelial coverage and scar formation, but is rather resistant to infection, at least when granulation tissue has developed.

Surgical closure procedures have combined the advantages of the two types of healing. This has led to a technique of *delayed primary closure*, where the wound is left open for a few days but closure is completed before granulation tissue becomes visible (usually a week after wounding) and the wound is then healed by a process similar to primary healing (59, 60, 435, 614). The resulting wound is more resistant to healing complications (primarily infection) and is functionally and cosmetically improved. If visible granulation tissue has developed before either wound closure or wound contraction has spontaneously approximated the defect, it is called *secondary closure*. This wound is healed by a process similar to secondary healing and scar formation is more pronounced than after delayed primary closure. The different closure techniques are shown in Fig. 1.5. The following section describes the sequential changes in tissue components and their interactions seen during the wound healing process.

## Tissues and compounds in wound healing

### Hemostasis phase and coagulation cascade

An injury that severs the vasculature leads to extravasion of plasma, platelets, erythrocytes and leukocytes. This initiates the coagulation cascade that produces a blood clot usually after a few minutes and which, together with the already induced vascular contraction, limits further blood loss (Fig. 1.6). The tissue injury disrupts the endothelial integrity of the vessels, and exposes the subendothelial structures and various connective tissue components. Exposure of type IV and V collagen in the subendothelium promotes binding and aggregation of platelets and their structural proteins (61, 62). Exposure of collagen and other activating agents provokes endothelial cells and platelets to secrete several substances, such as fibronectin, serotonin, PDGF, adenosine diphosphate (ADP), thromboxane A and others. Following this activation, platelets aggregate and platelet clot formation begins within a few minutes. The clot formed is impermeable to plasma and serves as a seal for the ruptured vasculature as well as to prevent bacterial invasion (62). In addition to platelet aggregation and activation, the coagulation cascade is initiated (Fig. 1.6).

The crucial step in coagulation is the conversion of fibrinogen to fibrin, which will create a thread-like network to entrap plasma fractions and formed elements. This fibrin

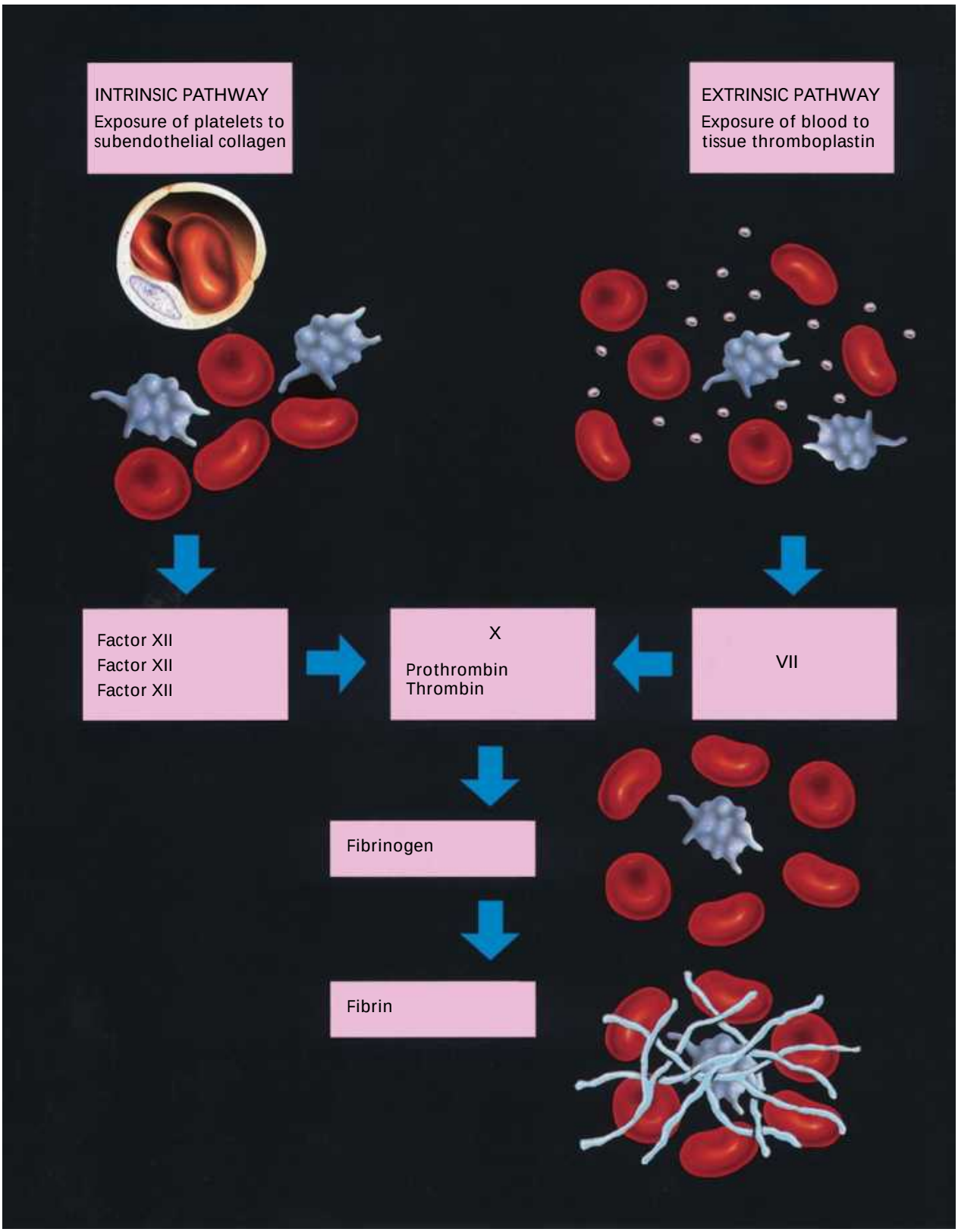


**Fig. 1.6** Extrinsic and intrinsic coagulation cascade.

blood clot is formed both intravascularly and extravascularly and supports the initial platelet clot (Fig. 1.6). Extrinsic and intrinsic clotting mechanisms are activated, each giving rise to cascades that will convert prothrombin to thrombin, and in turn cleave fibrinogen to fibrin which then polymerizes to form a clot (63).

The *extrinsic* coagulation pathway is initiated by tissue thromboplastin and coagulation factor VII, whereas the

initiator of the *intrinsic* coagulation cascade consists of Hageman factor (factor XII), prekallikrein and high molecular weight kinogen. The extrinsic coagulation pathway is the primary source of clotting, while the intrinsic coagulation pathway is probably most important in producing bradykinin, a vasoactive mediator that increases vascular permeability (64).

Products of the coagulation cascade regulate the cells in the wound area. Thus *intact thrombin* serves as a potent growth stimulator for fibroblasts and endothelial cells (65, 66) whereas *degraded thrombin* fragments stimulate monocytes and platelets (67–69). Through its chemotactic and mitogenic activities towards macrophages, fibroblasts and endothelial cells thrombin directly supports wound healing (632). Likewise, *plasmin* acts as a growth factor for parenchymal cells (69). *Fibrin* acts as a chemoattractant for monocytes (70) and induces angiogenesis (64). Other mediators created by blood coagulation for wound healing include *kallikrein*, *bradykinin*, and *C3a* and *C5a* through a spillover activation of the complement cascade, and most of these factors act as chemoattractants for circulating leukocytes. Thus apart from ensuring hemostasis, the clot also initiates healing (Fig. 1.4).

If the blood clot is exposed to air it will dry and form a scab which serves as a temporary wound dressing. A vast network of fibrin strands extends throughout the clot in all directions (Fig. 1.6). These strands subsequently undergo contraction and become reoriented in a plane parallel to the wound edges (71, 72). As the fibrin strands contract, they exert tensional forces on the wound edges whereby serum is extruded from the clot and the distance between wound edges is decreased. Contraction and reorientation of the fibrin strands later serve as pathways for migrating cells (see p. 24).

If proper adaptation of the wound edges has occurred, the extravascular clot forms a thin gel filling the narrow space between the wound edges and gluing the wound edges together with fibrin.

If hemostasis is not achieved, blood will continue to leak into the tissue, leading to a hematoma and a coagulum which consists of serum plasma fraction, formed elements and fibrin fragments. The presence of such a hematoma will delay the wound healing and increase the risk of infection (77).

## Coagulation

More extensive blood clot formation is undesirable in most wounds as the clots present barriers between tissue surfaces and force wounds that might have healed without a clot to heal by secondary intention. In oral wounds such as extraction sockets, blood clots are exposed to heavy bacterial colonization from the saliva (74). In this location neutrophil leukocytes form a dense layer on the exposed blood clot and the most superficial neutrophils contain many phagocytosed bacteria (75).

The breakdown of coagulated blood in the wound releases ferric ions into the tissue, which have been shown to decrease the non-specific host response to infection (76). Furthermore, the presence of a hematoma in the tissue may increase the chance of infection (77).

Clot adhesion to the root surface appears to be important for periodontal ligament healing. Thus an experiment has shown that heparin-impregnated root surfaces, which prevented clot formation, resulted in significantly less connective tissue repair and an increase in downgrowth of pocket epithelium after gingival flap surgery (78).

Coagulation and sustained thrombin production seem also to be essential for wound healing in genral. Mice deficient in FXIII or FIX have delayed wound healing because of continued bleeding into the granulation tissue and reduced thrombin production (633).

## Fibrin

Fibrin in the wound provisional matrix provides adhesion for leukocytes, fibroblasts and endothelial cells (693). In addition, fibrin provides a reservoir for many growth factors such as FGF-2 and VEGF that stimulate wound healing. During coagulation, fibrin is crosslinked to plasma fibronectin to create a plug for hemostasis. Fibrinogen is converted to fibrin which, via a fishnet arrangement with entrapped erythrocytes, stabilizes the blood clot (Figs 1.6 and 1.7).

In the early acute inflammatory period extravasation of a serous fluid from the leaking vasculature accumulates as an edema in the tissue spaces. This transudate contains fibrinogen which forms fibrin when acted upon by thrombin (Fig. 1.7). Fibrin plugs then seal the damaged lymphatics and thereby confine the inflammatory reaction to an area immediately surrounding the wound.

Formation of a fibrin clot is also essential for the initiation of wound healing. Fibrin has been found to play a significant role in wound healing by its capacity to bind to fibronectin (63). Thus fibronectin present in the clot will link to both fibrin and to itself (79, 80).

Fibrin clots and fibrinopeptides are weak stimulators of fibroblasts (81), an effect which is prevented by depletion of fibronectin (82). It has also been proposed that an interaction may take place between hyaluronic acid and fibrin which creates an initial scaffold on which cells may migrate into the wound (83).

The extravascular fibrin forms a hygroscopic gel that facilitates migration of neutrophils and macrophages, an effect which possibly reflects a positive interaction between the macrophage surface and the fibrin matrix. Fibrin has also been shown to elicit fibroblast migration and angiogenesis, both of which initiate an early cellular invasion of the clot (63, 64, 84–86).

Fibrin clots are continuously degraded over a 1–3 week period (73, 87, 88). This occurs during the fibrinolysis cascade, which is activated by the plasminogen present in damaged endothelial cells and activated granulocytes and macrophages (87–89) (Fig. 1.7).

In experimental replantation of teeth in monkeys it has been found that collagen fiber attachment to the root surface was preceded by fibrin leakage, and that this leakage was an initial event in the wound healing response (90).

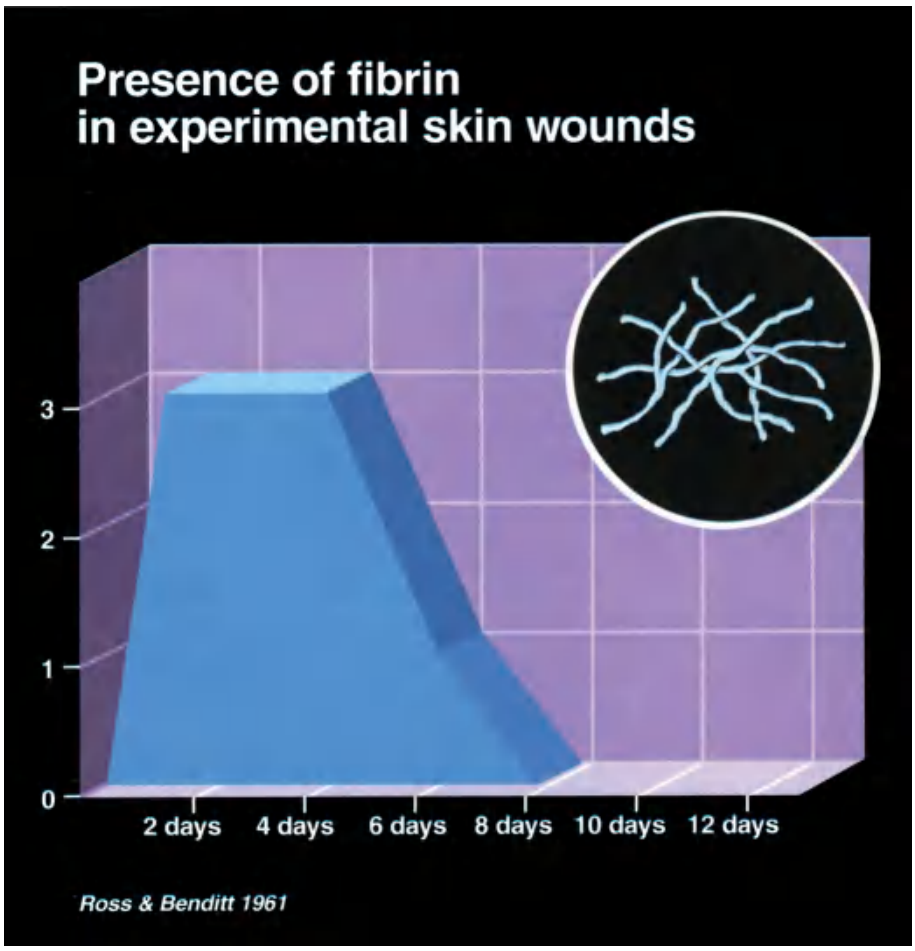


**Fig. 1.7** Schematic illustration of the presence of fibrin in experimental skin wounds in guinea pigs. The scale is semiquantitative, graded from 0 to 3. Adapted from (73).

In summary, the blood clot, apart from being responsible for hemostasis, also serves the purpose of initiating wound healing including functioning as a matrix for migrating connective cells.

## Fibronectin

Fibronectin is a complex glycoprotein, which can be present as soluble plasma fibronectin, produced by hepatocytes, or stromal fibronectin, found in basal laminae and loose connective tissue matrices where it is produced by fibroblasts, macrophages and epithelial cells (91, 92). During wound healing, fibronectin is also produced locally by fibroblasts (93), macrophages in regions where epidermal cell migration occurs (92), endothelial cells (94, 95), and by epidermal cells (96).

In normal resting adult epithelium, keratinocytes do not interact with fibronectin and thus do not typically express fibronectin receptors (629). However, in wound healing fibronectin plays many roles, including platelet aggregation, promotion of re-epithelialization, cell migration, matrix deposition and wound contraction (92, 97).

In wound healing, fibronectin is the first protein to be deposited in the wound (98) and therefore, together with fibrin, serves as a preliminary scaffold and matrix for migrating cells (99). Thus plasma fibronectin is linked to fibrin that has been spilled from damaged vessels or from highly permeable undamaged vessels (97, 100). The fibrin–fibronectin complex forms an extensive meshwork throughout the wound bed which facilitates fibroblast attachment and migration into the clot (80, 101–103). Furthermore, soluble fibronectin fragments are chemotactic for fibroblasts and monocytes (104).

Fibronectin appears also to guide the orderly deposition of collagen within the granulation tissue. Thus fibronectin serves as the scaffold for deposition of types III and I collagen (105–109) as well as collagen type VI (109). As dermal wounds age, bundles of type I collagen become more prominent at the expense of type III collagen fibronectin (106). Finally, fibronectin seems to represent a necessary link between collagen and fibroblasts, which makes it possible to generate the forces in wound contraction (92, 110).

Although plasma fibronectin does not appear to be essential for hemostasis (635), it can provide a substratum for epithelial and fibroblast migration towards the clot.

In the endothelium during wound healing, fibronectin is found in the basement membrane and reaches a maximum at approximately the same time as the peak in endothelial cell mitosis occurs, indicating a possible role of fibronectin in endothelial cell migration (111).

In epithelialization, it has been found that fibronectin is implicated in epidermal cell adhesion, migration and differ-

entiation (96, 111–116). Thus migrating epithelial cells are supported by an irregular band of fibrin–fibronectin matrix which provides attachment and a matrix for prompt migration (87, 108).

Clinically, fibronectin has been used to promote attachment of connective tissue to the exposed root and surfaces, thereby limiting epithelial downgrowth (117–123). Furthermore, fibronectin has been shown to accelerate healing of periodontal ligament fibers after tooth replantation (120). This effect has also been shown to occur in experimental marginal periodontal defects in animals (121, 122) as well as in humans (123).

### Complement system

The complement system consists of a group of proteins that play a central role in the inflammatory response. One of the activated factors, C5a, has the ability to cleave its C-terminal arginine residue by a serum carboxypeptidase to form C5a-des-arg which is a potent chemotactic factor for attracting neutrophils to the site of injury (124, 125).

### Necrotic cells

Dead and dying cells release a variety of substances that may be important for wound healing such as tissue factor, lactic acid, lactate dehydrogenase, calcium lysosomal enzymes and FGF (126).

### Matrix

#### *Proteoglycans and hyaluronic acid*

All connective tissues contain proteoglycans. In some tissues, such as cartilage, proteoglycans are the major constituent and add typical physical characteristics to the matrix (127).

##### Chondroitin sulfate proteoglycans

Chondrocytes, fibroblasts and smooth muscle cells are all able to produce these proteoglycans. Chondroitin sulfate impairs the adhesion of cells to fibronectin and collagen and thereby promotes cell mobility. Skin contains proteoglycans, termed dermatan sulfates, which are involved in collagen formation.

##### Heparin and heparan sulfate proteoglycans

Heparins are a subtype with an anticoagulant activity. Heparan sulfates are produced by mast cells and adhere to cell surfaces and basement membranes.

*Keratan sulfates* are limited to the cornea, sclera and cartilage. Their role in wound healing is unknown.

*Hyaluronic acid* is a ubiquitous connective tissue component and plays a major role in the structure and organization of the ECM. Hyaluronic acid has been implicated in the detachment process of cells that allows cells to move. Furthermore, hyaluronic acid inhibits cell differentiation. Because of its highly charged nature, hyaluronic acid can absorb a large volume of water (128).

The role of proteoglycans during wound healing is not fully understood (129). Heparin may play a role in the control of clotting at the site of tissue damage. Proteoglycans are also suspected of playing an important role in the early stages of healing when cell migration occurs. Thus *hyaluronic acid* may be involved in detachment of cells so that they can move (130). Furthermore, proteoglycans may provide an open hydrated environment that promotes cell migration (129, 131, 133, 135).

The proliferative phase of healing involves cell duplication, differentiation and synthesis of ECM components. Thus hyaluronidate has been found to keep cells in an undifferentiated state which is compatible with proliferation and migration (127). At this stage chondroitin and heparan sulfates are apparently important in collagen fibrillogenesis (127) and mast cell heparin promotes capillary endothelial proliferation and migration (132). Furthermore, when endothelium is damaged, a depletion of growth-suppressing heparan sulfate may allow PDGF or other stimuli to stimulate angiogenesis (133).

The combined action of substances released from platelets, blood coagulation and tissue degradation results in hemostasis, initiation of the vasculatory response and release of signals for cell activation, proliferation and migration.

The role of the anticoagulant heparin is to temporarily prevent coagulation of the excess tissue fluid and blood components during the early phase of the inflammatory response.

## Inflammatory phase mediators

The sequence of the inflammatory process is directed by different types of chemical mediators which are responsible for vascular changes and migration of cells into the wound area (Fig 1.6).

### Mediators responsible for vascular changes

Inflammatory mediators such as histamine, kinins and serotonin cause vasodilation unless autonomic stimulation overrules them.

The effect of these mediators is constriction of smooth muscles. This influences endothelial and periendothelial cells, providing reversible opening of junctions between cells and permitting a passage of plasma solutes across the vascular barrier. These mediators are released primarily during the process of platelet aggregation and clotting. The best known mediators related to the vascular response are shown in Table 1.1.

#### *Histamine*

The main sources of histamine in the wound appear to be platelets, mast cells and basophil leukocytes. The histamine release causes a short-lived dilation of the microvasculature (137) and increased permeability of the small venules. The endothelial cells swell and separations occur between the individual cells. This is followed by plasma leaking through the venules and the emigration of polymorphonuclear leukocytes (137–141).

**Table 1.1** Mediators of vascular response in inflammation. Adapted from (136).

| | Mediator | Originating cells |
|---|---|---|
| Humoral | Complement<br>Kallikrein–kinin system<br>Fibrin | |
| Cellular | Histamine | Thrombocytes<br>Mast cells<br>Basophils |
| | Serotonin | Thrombocytes<br>Mast cells |
| | Prostaglandins | Inflammatory cells |
| | Thromboxane $A_2$ | Thrombocytes<br>Neutrophils |
| | Leukotrienes | Mast cells<br>Basophils<br>Eosinophils<br>Macrophages |
| | Cationic peptides | Neutrophils |
| | Oxygen radicals | Neutrophils<br>Eosinophils<br>Macrophages |

### *Serotonin*

Serotonin (5-hydroxytryptamine) is generated in the wound by platelets and mast cells. Serotonin appears to increase the permeability of blood vessels, similarly to histamine, but appears to be more potent (139, 140). Apart from causing contraction of arterial and venous smooth muscles and dilation of arterioles, the net hemodynamic effect of serotonin is determined by the balance between dilation and contraction (137, 142).

### *Prostaglandins*

Other mediators involved in the vascular response are prostaglandins (PGs). These substances are metabolites of arachidonic acid and are part of a major group called eicosanoids, which are also considered primary mediators in wound healing (143). Prostaglandins are the best known substances in this group and are released by cells via arachidonic acid following injury to the cell membrane. These include $PGD_2$, $PGE_2$, $PGF_2$, thromboxane $A_2$ and prostacycline ($PGI_2$). These components have an important influence on vascular changes and platelet aggregation in the inflammatory response and some of the effects are antagonistic. Under normal circumstances, a balance of effects is necessary. In tissue injury, the balance will shift towards excess thromboxane $A_2$, leading to a shutdown of the microvasculature (143).

New research suggests that prostaglandins, and especially $PGF_2$, could be endogenous agents that are able to initiate repair or reconstitute the damaged tissue (144). Thus biosynthesis of $PGF_2$ has been shown to have an important effect on fibroblast reparative processes (145), for which reason this prostaglandin may also have an important influence on later phases of the wound healing process. The effect of prostaglandins on the associated inflammatory response elicited subsequent to infection is further discussed in Chapter 2.

### *Bradykinin*

Bradykinin released via the coagulation cascade relaxes vascular smooth muscles and increases capillary permeability leading to plasma leakage and swelling of the injured area.

### *Neurotransmitters (norepinephrine, epinephrine and acetylcholine)*

The walls of arteries and arterioles contain adrenergic and cholinergic nerve fibers. In some tissues the sympathetic adrenergic nerve fibers may extend down to the capillary level. Tissue injury will stimulate the release of neurotransmitters which results in vasoconstriction.

## Mediators with chemotactic effects

These mediators promote migration of cells to the area of injury and are thus responsible for the recruitment of the various cells that are involved in the different phases of wound healing (Fig. 1.4).

The first cells to arrive in the area are the leukocytes. The chemotactic effects are mediated through specific receptors on the surface of these cells. Complement-activated products like C5a, C5a-des-arg and others cause the leukocytes to migrate between the endothelial cells into the inflammatory area. This migration is facilitated by the increased capillary permeability that follows the release of the earlier mentioned mediators. Further leukocyte chemoattractants include kallikrein and plasminogen activator, PDGF and platelet factor 4.

Other types of chemotactic receptors are involved when leukocytes recognize immunoglobulin (Ig) and complement proteins such as C3b and C3bi. The mechanism appears to be that B lymphocytes, when activated, secrete immunoglobulin, which again triggers the activation of the complement system resulting in production of chemoattractants such as C5a-des-arg (146).

Other mediators involved in chemoattraction will be mentioned in relation to the cell types involved in the wound healing process.

# Growth factors

Growth factors are a group of polypeptides involved in cellular chemotaxis, differentiation, proliferation and synthesis of ECM during embryogenesis, postnatal growth and adulthood.

All wound healing events in both hard and soft tissues are influenced by polypeptide growth factors, which can be released from the traumatized tissue itself, can be harbored

in the quickly formed blood clot or brought to the area by neutrophils or macrophages.

Growth factors are local signaling molecules. They can act in a paracrine manner where they bind to receptors on the cell surface of neighboring target cells, leading to initiation of specific intracellular transduction pathways; or they can act in an autocrine manner, whereby the function is elicited on the secreting cell itself. Additionally, elevated serum levels have been demonstrated for a few growth factors which may indicate an endocrine effect. Complex feedback loops regulate the production of the individual growth. The effect of each growth factor is highly dependent on the concentration and on the presence of other growth factors. A growth factor can have a stimulatory effect on a specific cell type, whereas an increased concentration may inhibit the exact same cell type. Two different growth factors with a known stimulatory effect on a cell type can in combination result in both an *agonistic*, *synergistic* and even *antagonistic* effect.

Growth factors may have the potential to improve healing of traumatized tissues in several ways. First, some growth factors have the ability to recruit specific predetermined cell types and pluripotent stem cells to the wounded area by chemotaxis. Second, they may induce differentiation of mesenchymal precursor cells to mature secreting cells. Third, they often stimulate mitosis of relevant cells, and thereby increase proliferation. Fourth, several growth factors have the ability to increase angiogenesis, the ingrowth of new blood vessels. Finally, they can have a profound effect on both secretion and breakdown of ECM components.

The most important growth factors are listed in Table 1.2 and a brief summary of their characteristics, including their presumed role in wound repair and regeneration is given below.

Dentoalveolar traumas may involve a multitude of tissues like oral mucosa, periodontal ligament, root cementum, dentin, dental pulp, bone, skin, blood vessels and nerves. Only a few clinical studies have evaluated the use of growth factors specifically for oral and maxillofacial traumas (see Chapter 2).

### *Platelet-derived growth factor (PDGF)*

PDGF consists of two amino acid chains and comes in homo- and heterodimeric isoforms (AA, AB, BB, CC and DD, where AA, AB and BB are the best documented) (147). PDGF binds to two specific receptors: α and β. Differential binding of the different isoforms to the receptors contributes to the varying effects of PDGF. As the name implies, PDGF is released from platelets, where it is present in large amounts in α-granules. Platelets are activated by thrombin or fibrillar collagen. Other sources of PDGF are macrophages, endothelial cells and fibroblasts. PDGF was the first growth factor shown to be chemotactic for cells migrating into the wound area, such as neutrophils, monocytes and fibroblasts. Additionally, PDGF stimulates proliferation and ECM production of fibroblasts (148) and activates macrophages to debride the wound area (149).

### *Platelet-rich plasma (PRP)*

PRP has been advocated for periodontal regeneration as well as pulp regenerative therapy. PRP is prepared in the office from patients' own blood using centrifugation and platelets are then activated by thrombin (658). PRP contains several growth factors released mainly by thrombocytes such as PDGF-AB, PDGF-BB, TGF-β, IGF-1 and VEGF (657).

The use of PRP in regenerative periodontics has shown both positive and negative results (658, 659). PRP has also been used as a scaffold with growth factors in regenerative endodontic treatment. In case reports it seemed to have a positive effect (688–691); however, a larger clinical study could not support such a finding (692). The effect of PRP has been examined in furcation defects and sinus graft procedures, but it does not seem to optimize healing (670).

### *Transforming growth factors (TGFs)*

TGFs comprise a large family of cytokines with a widespread impact on the formation and development of many tissues (among those, the bone morphogenetic proteins, which are described separately). This factor has been divided into α and β subtypes, where the latter is the most important for the wound healing process (150). TGF-β is mainly released from platelets and macrophages as a latent homodimer that must be cleaved to be activated. This latent form is present in both wound matrix and saliva. TGF-β is known to be a strong promoter of ECM production of many cell types (e.g. collagen and mucopolysaccharide) including periodontal ligament fibroblasts. TGF-β encourages ECM reorganization and increased stability by collagen crosslinking (628).

Proliferation of fibroblasts is also induced by TGF-β, whereas mitogenesis of most other cell types is inhibited like keratinocytes, lymphocytes and most epithelial cells. Additionally, TGF-β plays a role in immune and inflammatory regulation. TGF-β is also deposited in bone matrix where it is released during bone remodeling or in relation to trauma and acts as a chemotactic on osteoblasts. The effects of TGF-β are extremely complex and strongly dependent on the concentration of the growth factor itself, the concentration of other growth factors and the differentiation state of the target cells.

### *Epidermal growth factor (EGF)*

EGF was one of the first growth factors to be isolated (151). It is produced by platelets, salivary glands and duodenal glands. TGF-α is today considered to be a member of the EGF family. The receptors for EGF have been found in oral epithelium, enamel organ, periodontal ligament fibroblasts and preosteoblasts (152, 153). Stimulation of the EGF receptor causes the cells to become less differentiated and to divide and grow rapidly. In wounds, EGF has been found to encourage cells to continue through the cell cycle. Such a cell proliferative effect has been demonstrated in epithelial cells (154), endothelial cells and periosteal fibroblasts (155). EGF has also been shown to be chemotactic for epithelial cells (156) and to stimulate fibroblast collagenase production

**Table 1.2** Characteristics of growth factors involved in healing after dental trauma.

| Growth factor | Originating cells | Target cells | Main effect | Tissue response |
|---|---|---|---|---|
| PDGF | Platelets<br>Macrophages<br>Endothelial cells<br>Osteoblasts | Neutrophils<br>Monocytes<br>Fibroblasts<br>Osteoblasts | Chemotaxis<br>Proliferation | Angiogenesis<br>Macrophage activation |
| TGF | Platelets<br>Macrophages<br>Fibroblasts<br>Lymphocytes<br>Osteoblasts | Fibroblasts<br>Monocytes<br>Neutrophils<br>Macrophages<br>Osteoblastic precursor cells | Chemotaxis<br>ECM production<br>Proliferation | Collagen production (scarring)<br>Downregulation of other cell types but fibroblasts<br>Immunoregulation |
| IGF | Hepatocytes<br>Osteoblasts | Fibroblasts<br>Osteoblasts<br>Epithelial cells | Proliferation<br>ECM production<br>Chemotaxis<br>Cell survival | Stimulated DNA synthesis<br>Growth promotion of committed cells |
| EGF | Platelets<br>Salivary glands | Epithelial cells<br>Enamel organ<br>Periodontal ligament fibroblasts<br>Preosteoblasts | Proliferation<br>Chemotaxis<br>ECM production | Epithelialization<br>Tooth eruption |
| FGF | Endothelial cells<br>Macrophages<br>Keratinocytes<br>Osteoblasts | Endothelial cells<br>Fibroblasts<br>Keratinocytes | Proliferation<br>Migration<br>ECM formation | Angiogenesis<br>Epithelialization |
| VEGF | Keratinocytes<br>Macrophages<br>Fibroblasts | Endothelial cells | Proliferation | Angiogenesis |
| BMP | Osteoblasts | Undifferentiated mesenchymal cells<br>Osteoblastic precursor cells<br>Osteoblasts | Differentiation<br>Proliferation<br>ECM production | Bone formation<br>Cementum formation<br>Dentin formation<br>PDL formation |
| GDF-5 | Osteoblasts | Fibroblasts<br>Osteoblasts<br>Chondroblasts | Proliferation | Recruitment of stem cells for ligament repair<br>Cartilage formation<br>Bone formation<br>Cementum formation<br>Periodontal ligament<br>Fibroblast proliferation |
| EMP | Ameloblasts | Angiogenesis<br>Macrophage activation<br>Periodontal ligament fibroblasts<br>Osteoblasts<br>Cementoblasts | Proliferation | Angiogenesis<br>PDL formation<br>Cementum formation<br>Bone formation |
| P-15 | Collagen cell binding region | Osteoblast precursors<br>Osteoblasts<br>Fibroblasts | Adhesion and proliferation of fibroblasts<br>Osteogenic differentiation | Bone formation |

ECM, extracellular matrix; PDL, periodontal ligament.

(157). In oral tissues it has been shown that EGF controls the proliferation of odontogenic cells (158) and accelerates tooth eruption (159).

### *Insulin-like growth factor (IGF)*

IGF is a single chain polypeptide which structurally is very similar to proinsulin. Two isoforms, IGF-1 and IGF-2 are mainly produced in the liver and exert their effects in autocrine, paracrine and endocrine manners. The endocrine effect is mainly controlled by growth hormone. Osteoblasts also produce IGF that is stored in the bone matrix and acts as paracrine and autocrine (160, 161). IGF alone has hardly any major effect on wound healing (162). Combinations with other growth factors, such as PDGF and FGF, have, however, been shown to have a pronounced stimulatory effect on fibroblast proliferation, collagen synthesis, bone formation and epithelialization (162).

### *Fibroblast growth factors (FGFs)*

FGFs comprise a growing family of polypeptides, currently consisting of more than 20 members. They are mainly produced by endothelial cells and macrophages. FGFs are mitogenic for several cell types involved in wound healing and support cell survival under stress conditions. FGFs are involved in angiogenesis and epithelialization. FGF-1 and FGF-2 (earlier known as acidic FGF and basic FGF) are potent stimulators of angiogenesis in the early formation of granulation tissue (days 1–3) by recruiting endothelial cells and inducing proliferation. Neither has a transmembrane sequence and can therefore not be secreted. Instead they are probably released from disrupted cells by tissue damage (163). After release, FGFs interact with heparin and heparan sulfate, with which they can be stored in the ECM. Here FGF can be activated when injury causes platelets to degranulate and among many other substances release heparin degrading enzymes.

### *Vascular endothelial growth factor (VEGF)*

VEGF is, as far as we know today, the only endothelial-specific growth factor enhancing cell proliferation, and its activity is therefore probably essential for angiogenesis in all tissues during both development and repair. VEGF is produced in large quantities by keratinocytes, macrophages and, to a lesser extent, fibroblasts in the epidermis during wound healing, where it seems to be critical for angiogenesis in the granulation tissue formation from days 4 to 7. Hypoxia, a hallmark of tissue injury, induces VEGF production. Reduced expression and accelerated degradation of VEGF has been shown to cause skin wound defects (163) and the addition of VEGF has promoted angiogenesis in skin wounds in diabetic mice (164).

*In vivo*, VEGF has resulted in increased capillary density and bone formation in standardized bone defects in rabbits (165).

No clinical studies have evaluated the effect of VEGF in relation to oral and maxillofacial trauma.

### *Bone morphogenetic proteins (BMPs)*

BMPs are members of the TGF-β superfamily. More than 20 different BMPs have been identified. BMPs are found in bone matrix and in periosteal cells and mesenchymal cells of the bone marrow (166). BMP-2, -4, and -7 (also called osteogenic protein-1 (OP-1)) are the most involved in bone healing, whereas increased BMP-6 has been described in skin wounds. The main task of BMP is to commit undifferentiated pluripotential cells to become bone or cartilage forming cells. BMPs are the only known factors that are capable of forming bone in extraskeletal sites, a phenomenon referred to as osteoinduction (167).

### *Growth and differentiation factor 5 (GDF-5)*

This growth factor is a member of the large BMP family. This factor has been shown to enhance bone and cartilage formation in a series of animal studies (662). Furthermore, in animal models GDF-5 used with a carrier (βTCP or PLGA) has been shown to augment cementum, periodontal ligament (PDL) and bone formation (663–667).

### *Enamel matrix proteins (EMPs)*

EMPs, commercially sold under the name Emdogain®, have been used for decades in periodontics to promote periodontal regeneration in relation to attachment loss, and many reviews have described their effects in relation to bone grafting or guided tissue regeneration (668–670). Several studies have shown that EMPs can stimulate the expression of TGF-β1 and IGF-1 in PDL fibroblasts (671). Furthermore, EMPs have been shown to stimulate phagacytic activity of macrophages in relation to tissue repair (672, 673) and angiogenesis (674).

## Experimental data indicating clinical implications of growth factors

### *Angiogenesis*

During healing after trauma, *de novo* formation of the disrupted vascular supply is a prerequisite for most of the healing events. This is supported by the finding that hyperbaric oxygen (HBO) is a potent stimulator of healing of both hard and soft tissue healing (168) in sites with a compromised healing potential such as diabetic ulcers and irradiated bone (169, 170). The primary long-term effect of HBO is increased angiogenesis. VEGF, FGF, TGF-β and PDGF are known to be involved in angiogenesis during wound healing (259). Exactly how these growth factors interact with the ECM environment in the blood clot and in granulation tissue, are, however, not known in detail. Revascularization of the dental pulp is necessary after both tooth fractures and luxation injuries. VEGF, PDGF and FGF have been identified in the soluble and insoluble part of human dentin matrix (171). These may be released during injury and contribute to pulpal wound healing.

### *Wounds in skin and oral mucosa*

In most instances, healing proceeds rapidly in healthy individuals. Research has therefore mainly been focused on situations where the healing potential is seriously compromised such as diabetes, malnutrition and infection. In skin wounds, PDGF is known to be chemotactic to neutrophils, monocytes and fibroblasts. In addition, PDGF is a mitogen for fibroblasts, which has led to US Food and Drug Administration (FDA) approval for the treatment of non-healing ulcers (172, 173). In addition, PDGF stimulates new vascularization of an injured area (174). Exogenously applied TGF-β has been demonstrated to induce fibroblast infiltration in the wound and increased collagen deposition (175), as well as angiogenesis and mucopolysaccharide synthesis (175, 176). This results in an accelerated healing of incisional wounds (177, 178). Due to the same mechanisms, however, TGF-β is also intimately related to scar formation. Thus the elimination of TGF-β from incisional wounds in rats (by neutralizing antibody) is able to prevent scar tissue

formation (179, 620–622). Furthermore, it has been shown that the effect of TGF-β can be potentiated by the presence of PDGF and EGF (180). In experimental skin wounds in *animals*, an acceleration of both connective tissue and epithelial healing was found after topical application of EGF (181, 182). However, results after topical application of EGF to experimental wounds in *humans* have shown contradictory results on re-epithelialization (178, 183–186). In the oral mucosa, salivary EGF has been shown to stimulate migration of oral epithelial cells (187).

An interesting observation in mice has been that saliva rinsing of skin wounds (by communal licking) both enhances coagulation and leads to acceleration of wound healing (182, 188–190). Due to the high concentration of EGF found in saliva (191) this effect has been suggested to be caused by EGF. Later experiments with induced tongue wounds in mice have shown that EGF (and possibly also TGF-β) is involved in healing of wounds of the oral mucosa (192, 193). Salivary EGF is suggested not only to accelerate wound healing in the oral cavity, but also to contribute to preserving integrity of the oral mucosa (194). Administration of IGF-1 in skin wounds has no influence upon fibroblast proliferation or activity, or upon epithelialization (195–197). However, if IGF-1 is administered together with PDGF or FGF, a marked fibroblast proliferation and collagen production can be observed as well as enhanced epithelialization (197).

### Periodontal healing

Experimental studies have suggested that PDGF-BB alone could have a regenerative effect on the formation of root cementum, periodontal ligament and alveolar bone (151, 198–200, 675, 676). PDGF has clinically, however, mainly been evaluated in combination with IGF-1 where an increased bone fill could be observed both around periodontally compromised teeth and in peri-implant defects (199, 201, 202, 677). IGF used alone, TGF-β used alone, and the combination IGF-2/FGF-2/TGF-β has not been able to generate noteworthy periodontal regeneration in experimental studies (197, 203, 204).

FGF-2 has resulted in increased periodontal regeneration compared to control sites in experimentally created defects (206). Its biologic action has recently been described in a review article (678). Clinically, FGF-2 has in a randomized multicenter study been shown to stimulate periodontal regeneration (679).

Experimental studies have reported regeneration of a periodontal ligament with Sharpey's fibers, inserted in the newly formed cementum and alveolar bone by using recombinant BMP-2, BMP-7 (OP-1) and recently also BMP-12. The treated periodontal defects have been either surgically created (207–209) or experimentally induced (210). This pronounced periodontal regeneration could not be obtained when BMP-12 was applied to extracted dog teeth before replantation. In contrast, ankylosis developed whether BMP-12 was applied or not (211). In cats ankylosis was also created when BMP-2 was applied to furcation defects (680). EMPs in the form of Endogain® have been tested for their capacity to promote healing in relation to periodontal healing in the replantation of extracted dog teeth. In one study a significantly better healing was found (681), whereas a similar designed study could not demonstrate any effect (682).

ABM/P-15 has been used in periodontal regeneration both in animals and in humans with good results in regard to bone healing and gain in attachment (683–687).

### Bone healing

Information of the role of growth factors in bone healing mainly comes from preclinical studies of periodontal lesions and bone augmentation procedures before or in relation to implant placement. Numerous growth factors are deposited in bone matrix during bone formation (e.g. PDGF, TGF-β, FGF-2 and IGF-1). These are released during bone remodeling and in relation to trauma (256).

Contradictory results have been reported regarding the bone regenerative potential of PDGF. Both inhibition and stimulation of bone formation has been observed in rat calvarial defects (212, 213). PDGF alone has little impact on bone healing *in vivo*. However, a couple of studies have reported significant bone regeneration in periodontal and peri-implant defects, when PDGF is combined with IGF (102, 201, 202, 214, 215). Likewise, IGF must be combined with other growth factors to promote bone healing. TGF-β has a strong impact on the healing of long bone fractures (216). Only a few clinical data from the use of BMP in humans exist (217, 218). Experimental data, however, suggest enhanced bone formation using BMP-2 and -7 for bone regeneration procedures (219–221).

### Pulp-dentin complex

Attempts to regenerate the pulp-dentin complex have mainly focused on the possibility of generating a hard tissue (dentin) closure to an exposed pulp in relation to pulp capping. The key question is how to induce uncommitted pulpal cells to differentiate into odontoblast-like cells secreting reparative dentin. TGF-βs, BMPs, FGFs and IGFs are harbored in dentin and are known to influence dentinogenesis during embryogenesis (222). BMP-2, BMP-4 and BMP-7 (OP-1) have all been shown to induce widespread dentin formation in the pulp, even leading to total occlusion of the pulp cavity when applied in high doses (223). Numerous studies have evaluated the revascularization of avulsed replanted teeth, but none have specifically studied the role of growth factors in this process.

### Platelet concentrate/platelet-rich plasma

In the past few years, utilization of platelet concentrate (PC), also called platelet-rich plasma (PRP), has been increasingly recommended in patients undergoing osseous reconstruction and periodontal regeneration. PRP has gained much attention since the presentation of very promising data for the resulting bone density by adding PC to iliac cancellous cellular bone marrow grafts in the reconstruction of mandibular continuity defects (224). An

accelerated graft maturation rate and a denser trabecular bone configuration were observed in defects where PRP had been added. It was speculated that the stimulating effect of PRP was due to the accumulation of autogenous platelets, providing a high concentration of platelet growth factors with a well-documented impact on bone regeneration (225, 226). The concept of using autogenous growth factors is attractive since there is no risk of disease transmission, and as it is relatively inexpensive compared to growth factors produced by recombinant techniques.

Additionally, one clinical study (227) and a series of clinical case reports and case series have presented the use of PRP in different applications (228–236), leading to divergent recommendations. Data from experimental studies, evaluating the addition of PRP to bone graft materials have also been conflicting (237–247). In these studies a wide range of different animal models and PRP preparation techniques have been used. Therefore, they are difficult to compare. Moreover, none of the studies have analyzed the growth factor content in the applied PRP. Just one study analyzed the influence of PRP platelet concentration in an *in vivo* model. The authors demonstrated a certain platelet concentration interval with the most positive biologic effect on bone regeneration, corresponding to a 3–5-fold increased concentration compared to whole blood. There was no effect using low concentrations (0–2-fold increased concentration), and there seemed to be an inhibitory effect on bone regeneration when higher concentrations were used (6–11-fold increased concentration compared to whole blood) (248).

The use of PRP in regenerative periodontics has shown both positive and negative results (658, 659). PRP has also been used as a scaffold with growth factors in regenerative endodontic treatment. In case reports it seemed to have a positive effect (688–691). However, a larger clinical study could not support such a finding (62).

In conclusion, no methods are currently available to produce standardized PRP in which a certain whole blood platelet count will result in PRP with a predictable amount of platelets and a predictable combination of growth factors. Use of autologous growth factors is simple and safe as compared with allogenic and xenogenic preparation methods. Consistent results, however, cannot be expected until the ideal concentration of platelet growth factors has been identified and reliable PC preparation methods have been developed.

### Carriers/delivery systems for growth factors

Growth factors are in general volatile and need carriers to ensure continuance of the growth factor at the relevant site, and to provide sustained release of the growth factor in therapeutic doses. A carrier must be biocompatible. In addition, the carrier should be substituted concurrently with healing of the traumatized tissue, without causing an inflammatory reaction. Collagen can bind and release bioactive substances with some predictability (249, 250). Like other natural polymers, however, collagen has limitations in clinical use due to difficulties in engineering its properties, handling problems, immunogenicity and lack of resorption resistance (251, 252). Synthetic carriers for tissue promotive agents have therefore been extensively investigated. Traditionally copolymers such as lactic and glycolic acid have been utilized as vehicles for bioactive molecules due to their handling properties and biodegradability. They may, however, be associated with protein denaturation and inflammatory reactions along with the degradation process. More hydrophilic materials with controlled network properties thus offer an attractive alternative, but problems with loading the bioactive protein into the material is a common limitation related to these materials. A new polyethylene glycol (PEG) hydrogel may meet these demands. This hydrogel polymer network is synthesized around the bioactive molecules without modifying its action; it is highly water soluble, non-toxic and non-immunogenic (253).

In bone regeneration, a certain mechanical stability of the carrier is often required in order to avoid collapse of soft tissue into the defect and to protect against pressure from the overlying periosteum. *In vitro* investigations have shown that both adsorption of the bioactive substance and release kinetics exhibit pronounced variation when different carriers and growth factors are combined (254). In addition, the growth factor may be inactivated in relation to the release (254). PDGF-BB has, compared to IGF-1, been shown to adsorb better, be released more completely and keep its bioactivity in combination with an anorganic bovine bone substitute material (255).

In dental traumatology a carrier will probably be needed in case of pulp and PDL regeneration.

## Cells in wound healing

### Platelets

Platelets (thrombocytes) are anucleate discoid fragments with a diameter of 2 µm (Fig. 1.8). They are formed in the bone marrow as fragments of cytoplasmatic buddings of

**Fig. 1.8** Activated platelet (thrombocyte).

megakaryocytes and have a life span of 7–10 days in the blood (260). Platelets contain various types of granules which, after release, have a number of effects upon hemostasis and initiation of wound healing processes (261–263) (Fig 1.4). Under physiologic conditions, platelets are limited to the intravascular space where they circulate in the blood without adhesion to each other or to the vessel walls.

The capacity of the platelets to adhere to exposed tissue surfaces as well as to each other after vessel injury is decisive for their hemostatic capacity (256–259). Adhesion and activation of platelets occurs when they contact collagen and microfibrils of the subendothelial matrix and locally generated factors such as thrombin, ADP, fibrinogen, fibronectin, thrombospondin and von Willebrand factor VIII. Platelet adhesion and aggregation are also influenced by the particular matrix proteins exposed during injury and by the local hydrodynamic conditions including shear stress and shear rate (632–635).

Platelet activation results in degranulation and release of ADP, serotonin, thromboxane, prostaglandins and fibrinogen. The release of these substances initiates binding of other platelets to the first adherent platelets whereby blood loss is limited during formation of a hemostatic platelet plug (264). The blood loss is further reduced by the vasoconstrictor effect of thromboxane and serotonin. Platelet activation during primary hemostasis also initiates the wound healing response via release of cytokines such as PDGF and TGF-β1 that serve as a chemotactic and stimulatory signal for many cell types essential for wound healing (627).

The inflammatory response is initiated by activation of platelets due to liberation of serotonin, kinins and prostaglandins which leads to increased vessel permeability.

The platelet release of cytokines such as PDGF, platelet-derived angiogenesis factor (PDAF), TGF-α, TGF-β and platelet factor 4 leads to an initiation of the wound healing process (Fig. 1.9). Thus PDGF has been shown to have a chemotactic and activating effect upon neutrophils, monocytes and fibroblasts as well as a mitogenic effect upon fibroblasts and smooth muscle cells (263, 265). The release of TGF-β has been found to induce angiogenesis and collagen deposition (266, 267). PDAF has been shown to cause new capillary formation from the existing microvasculature (268–270). Finally, platelet factor 4 has been found to be a chemoattractant for neutrophils (271).

In summary, the platelets are the first cells brought to the site of injury. Apart from their role in hemostasis, they exert an effect upon the initiation of the vascular response and attraction and activation of neutrophils, macrophages, fibroblasts and endothelial cells. As wound healing progresses, the latter tasks are gradually assumed by macrophages.

## Erythrocytes

The influence of erythrocytes upon wound healing is not adequately documented except for the effect of carrying oxygen to healing tissue (40, 42, 48, 618). In one study it was found that neovascularization was stimulated in areas with erythrocyte debris (272). Another effect of the breakdown of erythrocytes is the liberation of hemoglobin, which has been found to enhance infection (273–276). In addition, the heme part of hemoglobin may contribute to the production of oxygen free radicals that can produce direct cell damage (277).

In summary, the role of erythrocytes in wound healing is doubtful, apart from being oxygen carriers.

## Mast cells

Masts cells, distinguished by their large cytoplasmatic granules, are located in a perivenular position at portals of entry of noxious substances and are especially prominent within the body surfaces that are subject to traumatic injury, such as the mucosa and skin (278, 279) (Fig. 1.10).

The mast cell participates in the initial inflammatory response after injury via a series of chemical mediators such as histamine, heparin, serotonin, hyaluronic acid, prostaglandins and chemotactic mediators for neutrophils.

Mast cells, which reside in tissues in a resting state, may release mast cell mediators through direct trauma inflicted on the cell. Another means of activation after trauma appears to be when coagulation generates the mast cell activator bradykinin. An alternative means of mast cell activation appears to be the release of endotoxin during infection and the generation of C3a, C5a and cationic neutrophil protein during the inflammatory response (278).

The release of the mast cell mediators such as histamine, heparin, serotonin and slow reacting substance of anaphylaxis (SRS-A) results in active vasodilation of the small venules, which allows for the entrance of water, electrolyte and plasma proteins into the microenvironment. The maintenance of an open channel for this influx is promoted by the anticoagulant activity of heparin and by the proteolytic enzymes such as chymase. Histamine and heparin may also potentiate the angiogenesis when other angiogenic factors are present (280).

The liberation of a neutrophil chemotactic factor and a lipid chemotactic factor from activated mast cells both result in the attraction of neutrophils and the release of a platelet-activating factor which results in degranulation and aggregation of platelets. Finally, hyaluronic acid promotes cell movement and may be crucial for cell division, which is essential in this phase of wound healing (9).

In summary, the mast cell plays a role, together with platelets, in being the initiator of the inflammatory response. However, experiments with corneal wounds have shown that healing can proceed in the absence of mast cells (281) and in mice mast cells do not seem to play a role in the healing of full thickness excisional cutaneous wounds (639).

## Neutrophils

The first wave of cells entering the wound site are neutrophil leukocytes which migrate from the microvasculature (Figs 1.11 and 1.12). The primary function of neutrophils is to phagocytize and kill microorganisms present within the wound (282, 283). They then degrade tissue macromolecules

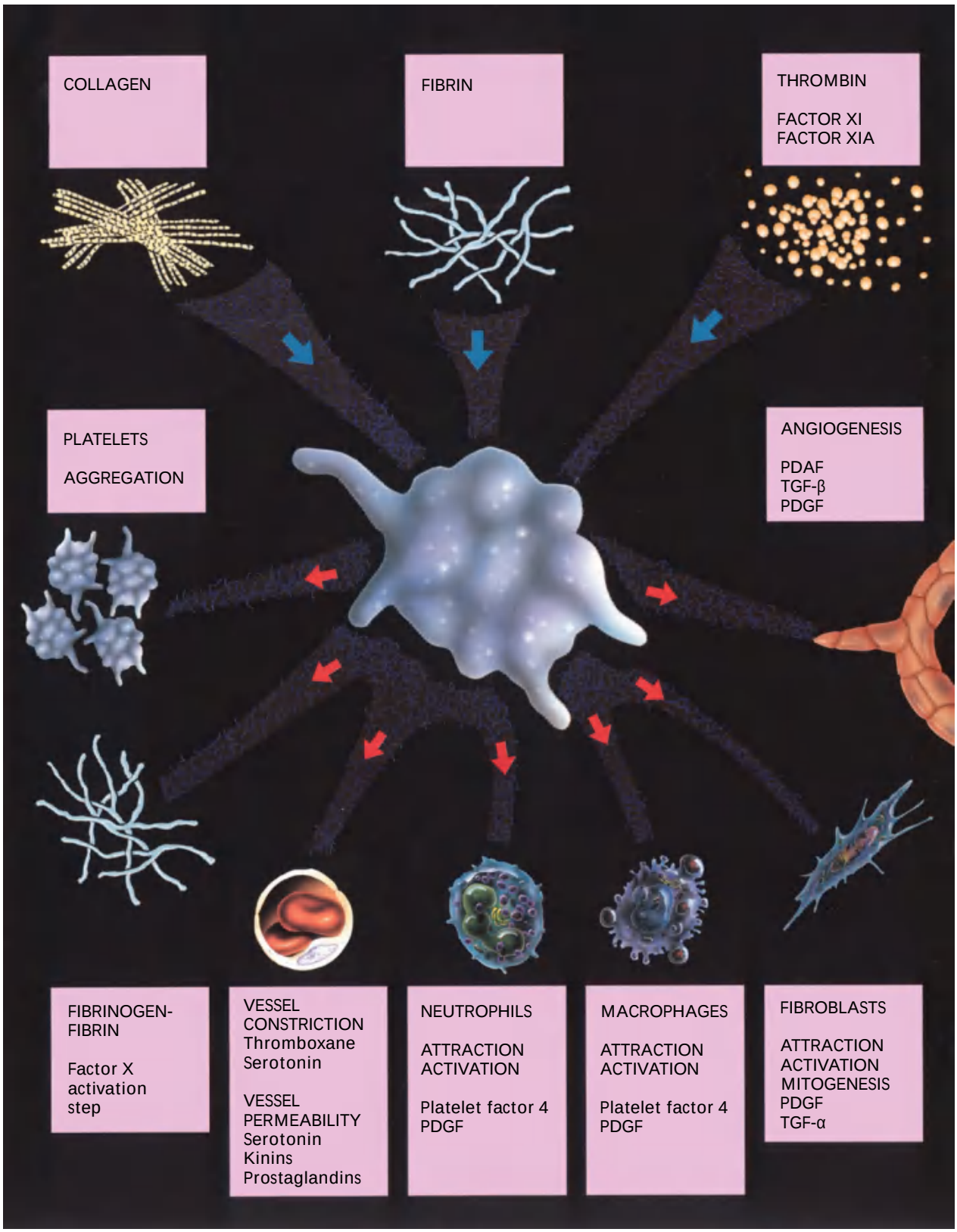


**Fig. 1.9** Role of platelets in wound healing. Exposure of platelets to collagen, fibrin, thrombin, factor XI and factor XI-A results in activation and degranulation. This then results in the release of a series of mediators influencing coagulation, vessel tone and permeability. Furthermore the initial cellular response of neutrophils, macrophages, fibroblasts and endothelial cells is established.

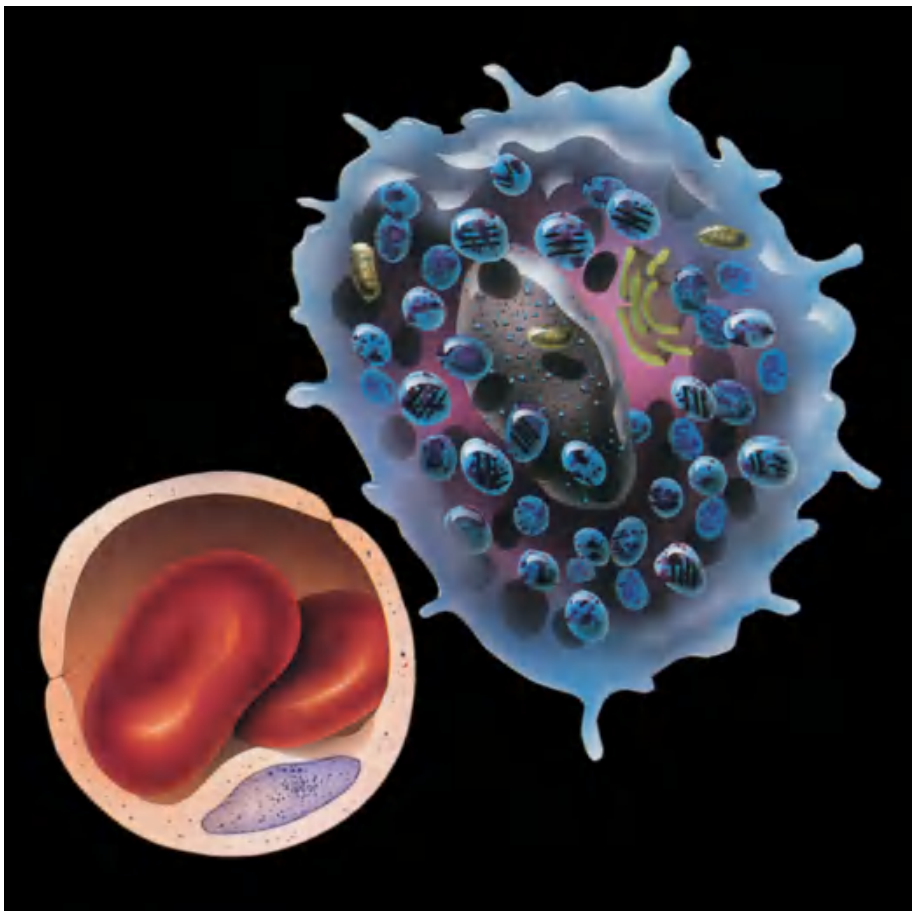

**Fig. 1.10** Mast cell in perivenular position.

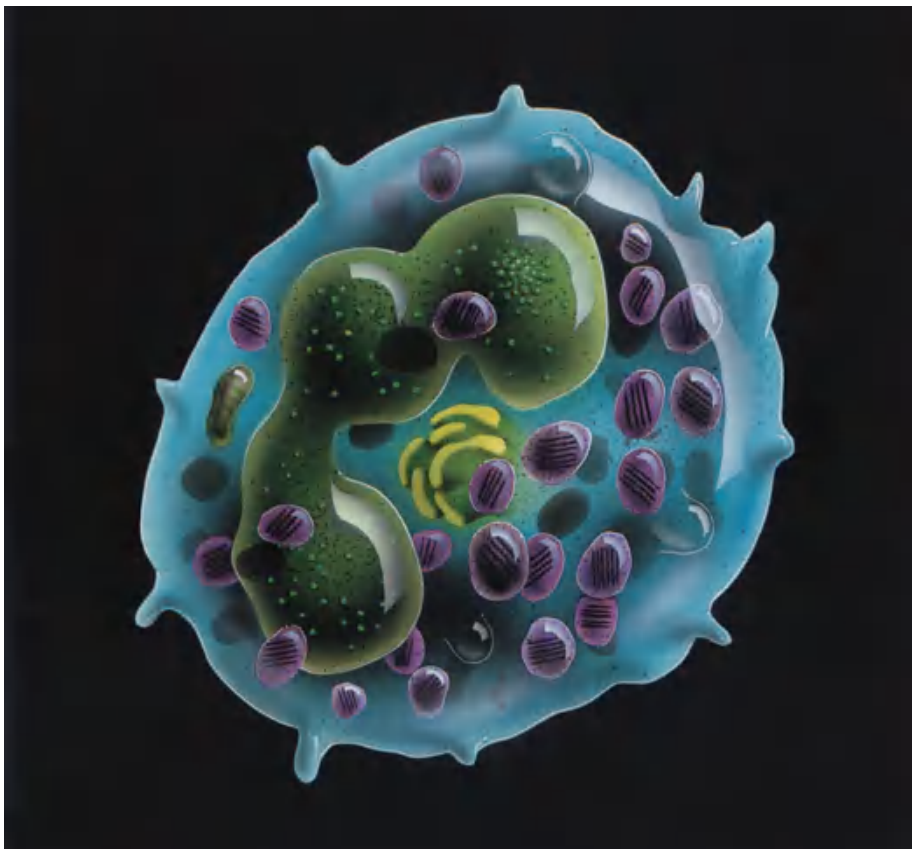

**Fig. 1.11** Neutrophil leukocyte.

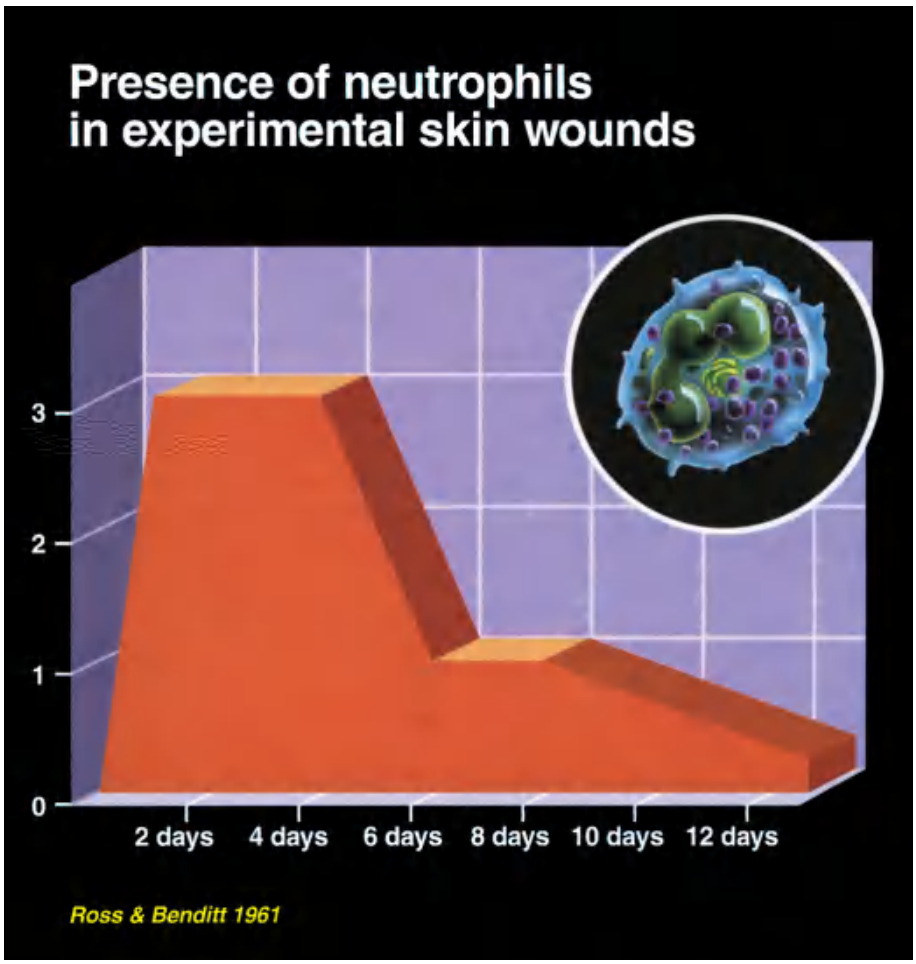


**Fig. 1.12** Schematic illustration of the presence of neutrophils in experimental skin wounds in guinea pigs. The scale is semiquantitative, graded from 0 to 3. Adapted from (73).

**Table 1.3** Neutrophil-produced degrading products.

| **Primary granules (unspecific, azurophilic)** |
|---|
| Cathepsin A, G<br>Elastase<br>Collagenase (unspecific)<br>Myeloperoxidase<br>Lyzozyme |
| **Secondary granules (specific)** |
| Lactoferrin<br>Collagenase (specific)<br>$B_{12}$ binding protein<br>Lyzozyme |
| **Other products** |
| Gelatinase<br>Kininogenase<br>Oxygen radicals |

such as collagen, elastin, fibrin and fibronectin by liberation of digestive enzymes (Table 1.3). Finally, neutrophils release a series of inflammatory mediators that serve as chemotactic or chemokinetic agents (Table 1.4, Fig. 1.4).

**Table 1.4** Some chemotactic and chemokinetic agents. Modified from (136).

| **Humoral** | |
|---|---|
| Chemotactic | C3 and C5 fragments<br>Fibrin degradation products<br>Kallikrein<br>Plasminogen activator<br>Fibronectin<br>Casein |
| Chemokinetic | C3 and C5 fragments<br>Acute phase proteins (Orosomucoid, $\alpha_1$-antitrypsin, $\alpha_2$-macroglobulin)<br>Hyaluronic acid |
| **Cellular** | |
| Chemotactic | Leukotriene $B_4$ (precursors and derivatives)<br>Platelet-activating factor (PAF)<br>Transforming growth factor-β (TGF-β)<br>Lymphokines<br>NCFs (neutrophil chemotactic factors)<br>ECFs (eosinophil chemotactic factors)<br>MCFs (monocyte chemotactic factors)<br>Formylated tripeptides (e.g. FMLP) |
| Chemokinetic | Cathepsin G |

Upon exposure to chemoattractants from the clot, such as platelet factor 4, PDGF kallekrein, C5a, leukotriene B4 (284, 285) and bacterial endotoxins (286–288), the granulocytes start to adhere locally to the endothelium of the venular part of the microvasculature next to the injury zone (289). Neutrophils begin to penetrate the endothelium between endothelial cells, possibly by active participation of the endothelium 2–3 hours after injury (290–293), and migrate into the wound area (291–293). Once a neutrophil has passed between endothelial cells, other leukocytes and erythrocytes follow the path (293, 294).

Once the neutrophil has passed between the endothelial cells it traverses the basement membrane by a degradation process and then moves into the interstitial tissue in the direction of the chemoattractant. This movement may be facilitated by proteolytic activity and enhanced by contact guidance. Thus neutrophils move preferentially along fiber alignments, suggesting that tissue architecture may be a significant determinant of the efficacy of cellular mobilization.

At this point the wound contains a network of fibrin, leukocytes and a few fibroblasts. By the end of the second day most of the neutrophils have lost their ameboid properties and have released their granula into the surrounding tissue. This event apparently triggers a second migration where plasma, erythrocytes and neutrophils again leave the venules (295). In the case of uncomplicated non-infected healing, the numbers of neutrophils decrease after 3–5 days (Fig. 1.12).

When the neutrophils have reached the site of injury they form a primary line of defense against infection by phagocytosis and intracellular killing of microorganisms (288). In this process each phagocyte may harbor as many as 30 or more bacteria (296).

Phagocytosis of bacteria by neutrophils induces a respiratory burst that produces toxic oxygen metabolites. These products include hypohalides, superoxide anion and hydroxyl radicals (288). Furthermore, they can generate chloramine formed by the reaction of hypochlorite with ammonia or amines (297). As a result of stimulation, phagocytosis or lysis, the neutrophils may release the content of their granules into the extracellular space. These granules contain oxygen radicals and neutral proteases, such as cathepsin G, elastase, collagenases, gelatinase and cationic proteins (288). All these products result in tissue damage and breakdown at an acid pH (298). A decrease or elimination of these products provided by experimental neutropenia has prevented the normally found decrease in wound strength in early intestinal anastomosis (299).

Despite these effects upon the wound, the presence of neutrophils is not essential for the wound healing process itself. Thus wound healing has been found to proceed normally as scheduled in uncontaminated wounds in the absence of neutrophils (283, 300). A recent study, however, demonstrating neutrophil expression of cytokines and TGF-β may indicate a positive influence on wound healing (301).

A role of neutrophils in healing of chronic wounds has been elucidated (302–308). A consistent feature of chronic wounds is chronic inflammation associated with increased neutrophil infiltration (302). Once initiated, the inflammatory response is perpetuated and gradually converted into a chronic inflammatory state. Morphologically, the chronic inflammatory infiltrate is predominantly composed of macrophages and lymphocytes (304, 305). Mast cells may also contribute to the fibrotic response (304). It is likely that different polypeptidic cytokines and growth factors mediate some of these processes. One candidate is the profibrotic and proinflammatory TGF-β1, which is also increased in the mRNA and protein levels in the lower leg skin of class 4 patients (305, 306).

It is thought that excessive local proteolytic activity results in the breakdown of the matrix components of the skin with the end result of an ulcer.

Proteinases in skin homeostasis have multiple biologic functions. Proteinases not only remodel ECM proteins but they also modulate the bioactivity of cytokines and growth factors by several different mechanisms (307, 308). In chronic wounds neutrophils are strong protease producers, delaying the wound healing process.

In summary, the main role of neutrophils in wound healing appears to be limited to the elimination of bacteria within the wound area.

## Macrophages

Following the initial trauma and neutrophil accumulation, monocytes become evident in the wound area (Fig. 1.13). These cells arise from the bone marrow and circulate in the blood (309, 310). In response to release of chemoattractants, monocytes leave the bloodstream in the same way as neutrophils. These cells are a heterogeneous group of cells which can express an almost infinite variability of phenotypes in response to changes (311–313).

Monocytes appear in the wound after 24 hours, reach a peak after 2–4 days, and remain in the wound until healing is complete (73) (Fig. 1.14). When monocytes invade the wound area, they undergo a phenotypic metamorphosis to macrophages (314, 315). It should be mentioned that tissue macrophages can proliferate locally (314–317) and possibly play a significant role in the initial inflammatory response.

The arrival of macrophages in the wound area is a response to various chemoattractants released from injured tissue, platelets, neutrophils, lymphocytes and bacteria (Fig. 1.4). In Table 1.4 a series of chemotactic factors are listed which have been shown to be chemotactic for macrophages. In this context, it should be mentioned that monocytes express multiple receptors for different chemotactic factors (317). As shown in Table 1.4, connective tissue fragments appear to be *chemotactic* for macrophages (i.e. forming gradients enabling directional movements) and *chemokinetic* (i.e. alter the rate of cell movements). These tissue fragments are possibly generated by neutrophils which precede the appearance of monocytes. It has been shown that neutrophils contain enzymes such as elastase, collagenase and cathepsin, which may

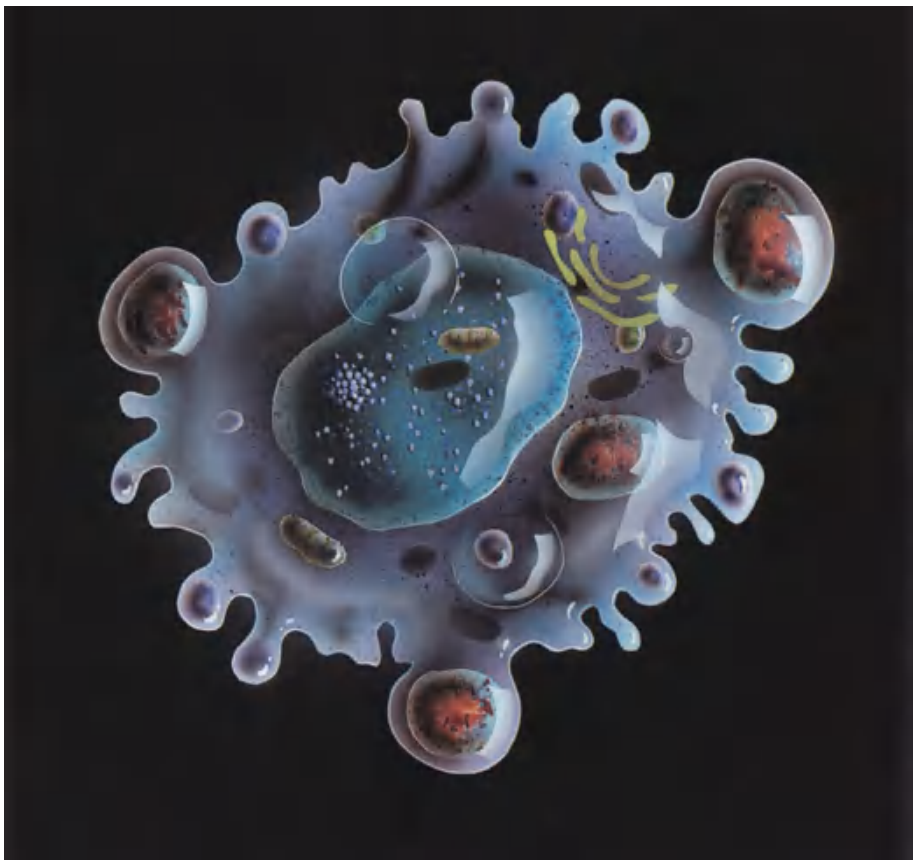

**Fig. 1.13** Macrophage.

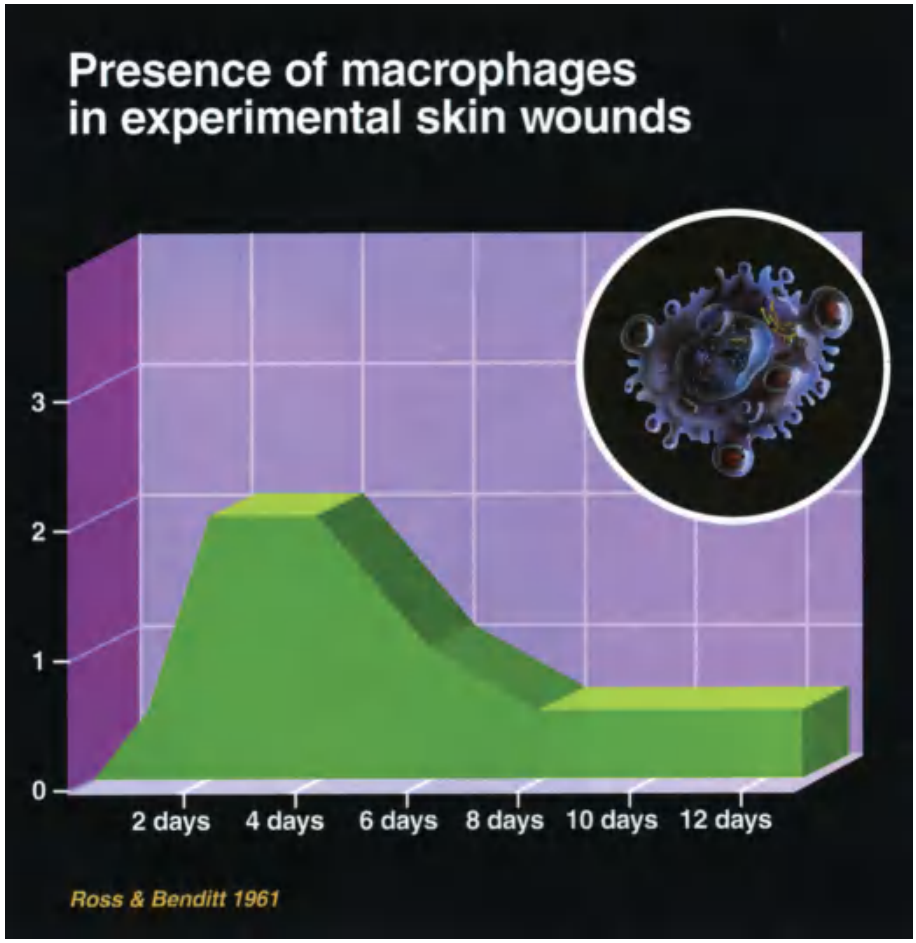


**Fig. 1.14** Schematic illustration of the presence of macrophages in experimental skin wounds in guinea pigs. The scale is semiquantitative, graded from 0 to 3. Adapted from (73).

degrade collagen and elastin, and fibronectin (Table 1.3) and thereby attract monocytes (317).

In contrast to neutrophils, depletion of circulating blood monocytes and tissue macrophages results in a severe retardation of tissue debridement and a marked delay in fibroblast proliferation and subsequent wound fibrosis (309). Macrophages therefore seem to have an important regulatory role in the repair process.

After migration from the vasculature into the tissue, the monocyte rapidly differentiates to an inflammatory macrophage, the mechanism of which is largely unknown. However, the binding of monocytes to connective tissue fibronectin has been found to drive the differentiation of monocytes into inflammatory macrophages (318, 319).

The regulatory and secretory properties of macrophages seem to vary depending on the state of activity: at rest, intermediate or in an activated state. Macrophage activation can be achieved by the already mentioned chemoattractants in higher concentrations. Further activation can be achieved through the products released from the phagocytotic processes described. These various stimuli induce macrophages to release a number of biologically active molecules with potential as chemical messengers for inflammation and wound repair (320) (Tables 1.1 and 1.2).

At the inflammatory site macrophages undertake functions similar to neutrophils, i.e. bacterial phagocytosis and killing and secretion of lysozomal enzymes and oxygen radicals $O_2$, $H_2O_2$ and OH (62). Activated inflammatory macrophages have been found to be responsible for the degradation and removal of damaged tissue structures such as elastin, collagen, proteoglycans and glycoproteins by the use of secreted enzymes such as elastase, collagenase, plasminogen activator and cathepsin B and D. Both extra- and intracellular tissue debridement can occur (317).

Macrophages have been shown to release growth factors such as PDGF, tumor necrosis factor (TNF) and TGF-β which stimulate cell proliferation in wound healing (321–323) (Fig. 1.4). These growth factors are collectively known as macrophage-derived growth factors (MDGFs). The level of MDGF can be significantly increased following stimulation of macrophages with agents such as fibronectin and bacterial endotoxin (324, 325).

Activation of macrophages has been found to lead to fibroblast proliferation (321, 326), increased collagen synthesis (322) and neovascularization (327–336).

Macrophages release their angiogenic mediator only in the presence of low oxygen tension in the injured tissue (i.e. 2–30 mmHg) (268, 269). However, as macrophages have been found to release lactate even while they are well oxygenated, the stimulus to collagen synthesis remains even during hyperoxygenation (331, 332), a finding which is of importance in the use of hyperbaric oxygen therapy.

Finally, macrophages can release a polypeptide, interleukin-1, that can function as a messenger for lymphocytes.

In summary, the macrophage seems to be the key cell in the inflammatory and proliferative phase of wound healing by secreting factors that stimulate the proliferation of fibroblasts and secretion of collagen, as well as stimulation of neovascularization (Fig. 1.4). The macrophages also act as scavengers in the wound area and remove traumatized tissue and bacteria and neutralize foreign bodies by forming giant cells that engulf or surround the foreign matter. Moreover, signals released by traumatized bone or tooth substances cause some monocytes to fuse and form osteoclasts which will subsequently resorb damaged hard tissue (see Chapters 2 and 7). Macrophages play an important role in the immune response to infection (see Chapter 2).

## Lymphocytes

Lymphocytes emigrating from the bloodstream into the injury site become apparent after 1 day and reach a maximum after 6 days (Figs 1.15 and 1.16). The role of these lymphocytes in the wound healing process has for many years been questioned as earlier investigations have pointed out that lymphocytes, like neutrophils, were not necessary for normal progression of healing in non-infected wounds (300). However, recent research has demonstrated that lymphocytes together with macrophages may modulate the wound healing process (333–335) (Fig. 1.17).

Lymphocytes can be divided into *T lymphocytes* (thymus-derived lymphocytes) and *B lymphocytes* (bone marrow-derived lymphocytes). Both types are attracted to the wound area, probably by activated complement on the surface of macrophages and neutrophils.

Lymphocyte infiltration in wounds is a dynamic process where both T-helper/effector and T-suppressor/cytotoxic lymphocytes are present in the wound after 1 week (336). These activated lymphocytes produce a variety of lymphokines of which interferon (IFN-α) and TGF-β have been shown to have a significant effect on endothelial cells and thereby may have an effect on angiogenesis. This effect may be secondary to other effects such as macrophage stimulation and activation (337). TGF-β is also a potent fibroblast chemotactic molecule and, in addition, induces monocyte chemotaxis and secretion of fibroblast growth factor and activating factors (338).

Recent studies have indicated that there are at least two populations of T cells involved in wound healing. One population bearing the T-cell marker appears to be required for successful healing, as shown by the impairment in healing caused by their depletion. The T-suppressor/cytotoxic subset appears to have a counterregulatory effect on wound healing, as their depletion enhances wound rupture strength and collagen synthesis (333–335, 337).

Based on present evidence, Barbul has postulated the following theory: macrophages exert a direct stimulatory effect on endothelial cells and fibroblasts (Fig. 1.17). A T-cell marker positive subset ($T^+$), which is not yet fully characterized, has a direct action on endothelial cells and fibroblasts and acts indirectly by stimulating macrophages. T-suppresser/cytotoxic cells (Ts/c) downregulate wound

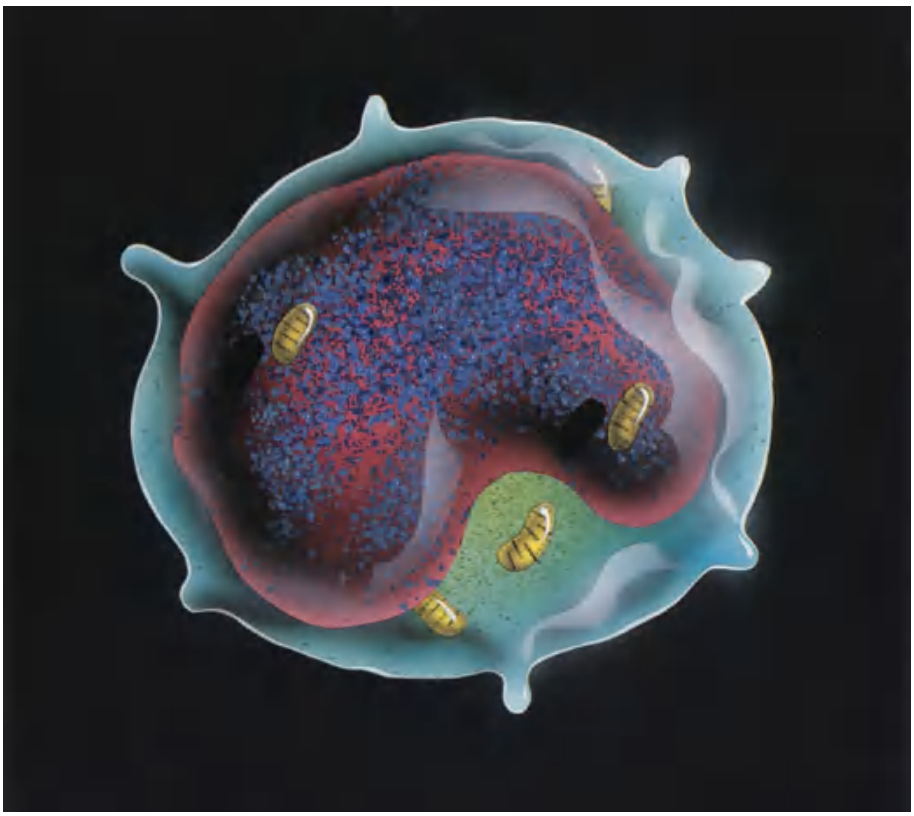

**Fig. 1.15** Lymphocyte.

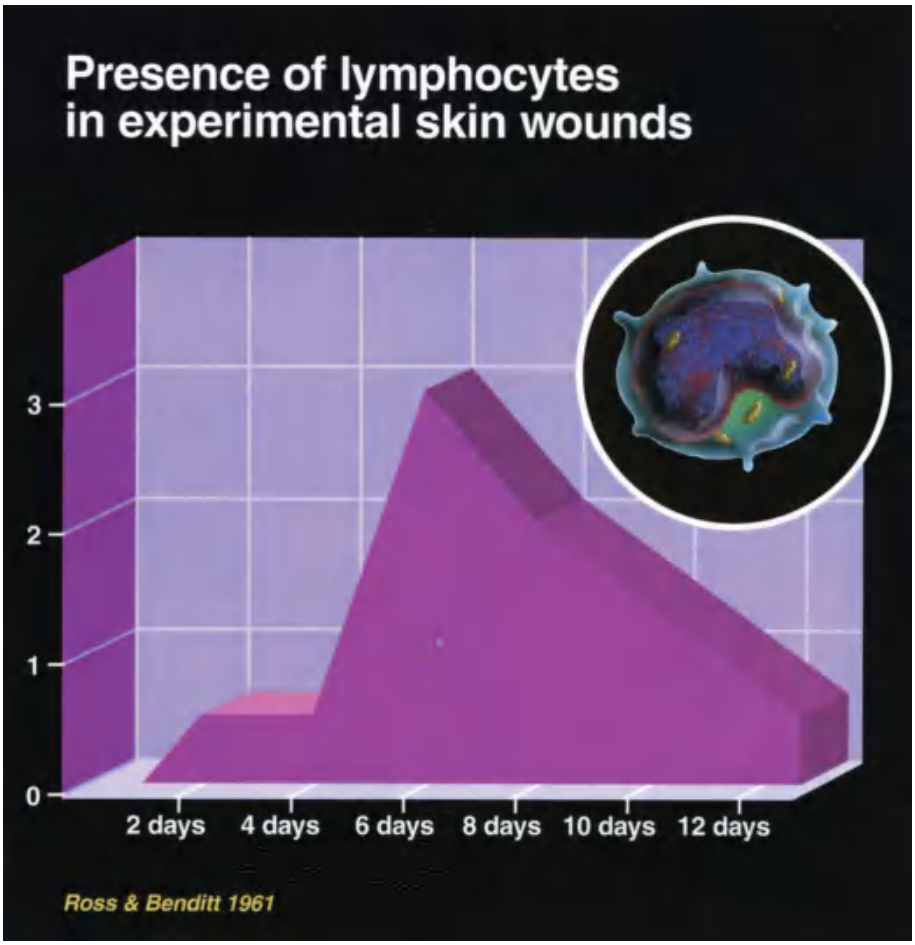


**Fig. 1.16** Schematic illustration of the presence of lymphocytes in experimental skin wounds in guinea pigs. The scale is semiquantitative, graded from 0 to 3. Adapted from (73).

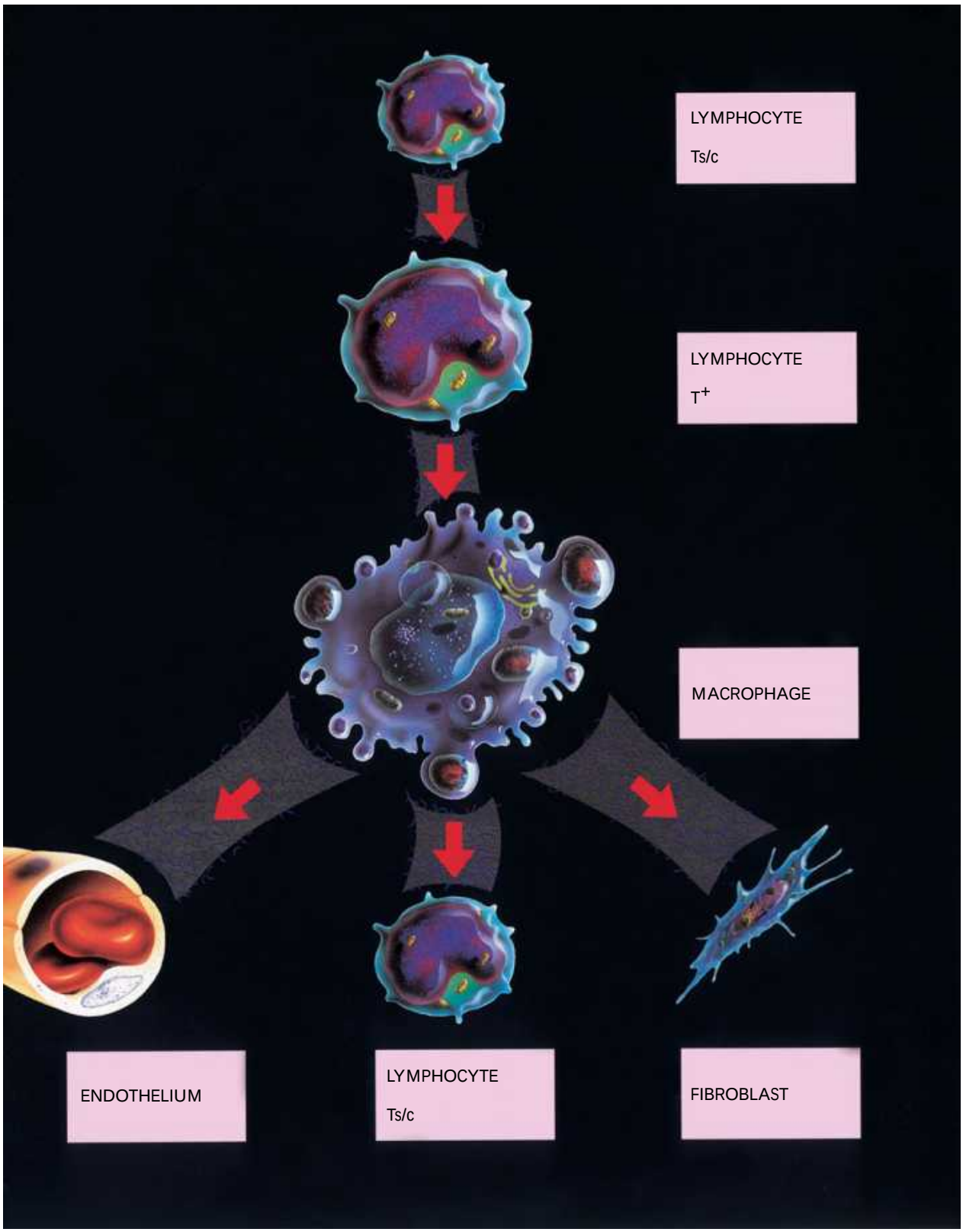


**Fig. 1.17** Role of lymphocytes in wound healing. Macrophages exert a direct stimulatory effect on endothelial cells and fibroblasts. A T-cell marker positive subset ($T^+$), which is not fully characterized, has direct action on endothelial cells and fibroblasts and acts indirectly by stimulating macrophages. T-suppressor/cytotoxic cells (Ts/c) downregulate wound healing by direct action on macrophages and T cells. Reproduced from (337) with permission.

healing by direct action on macrophages and T cells (337, 338) (Fig. 1.17).

There is presently no evidence to suggest that the humoral immune system (B lymphocytes) participates in the wound healing process. The influence of lymphocytes therefore seems primarily to be through T lymphocytes.

In summary, lymphocytes appear indirectly to influence the balance between the stimulatory and inhibitory signals to fibroblasts and endothelial cells via the macrophages.

## Fibroblasts

The fibroblast is a pleomorphic cell. In the resting, nonfunctional state it is called a *fibrocyte*. The cytoplasm is scanty and often difficult to identify in ordinary histologic sections. In the activated, mature form the cell becomes stellate or spindle shaped and is now termed a *fibroblast*. The most characteristic feature is now the extensive development of a dilated endoplasmic reticulum, the site of protein synthesis (339) (Fig. 1.18). In normal connective tissue, fibroblasts are attached to a fibrillar ECM mesh that contains an abundance of structural proteins such as crosslinked type I collagen and elastin produced by fibroblasts themselves (628). The ECM proteins present in the fibroblast microenvironment serve as a conduit for cell migration into the wound. In order to migrate, fibroblasts therefore need to modify their interaction with the existing ECM and be able to recognize and interact with the novel proteins present in the wound provisional matrix. To facilitate cell migration, fibroblasts use focalized proteolysis to release their adhesion to the pericellular ECM and to remodel the ECM (632, 633). The mode of fibroblast migration is different from that of leukocytes, which appear to use a more dynamic ameboid migration that does not require extensive ECM remodeling (634).

In the wound the fibroblast will produce collagen, elastin and proteoglycans (340). After injury, fibroblasts start to invade the area after 3 days, stimulated by platelets and macrophage products, and they become the dominating cell 6–7 days after injury, being present in considerable concentration until the maturation phase of the healing process (73) (Fig. 1.19).

Several studies have suggested that new fibroblasts arise from the connective tissue adjacent to the wound, principally from the perivascular undifferentiated mesenchymal cells (stem cells) (341–346) and not from hematogenous precursors (346).

Once fibroblast precursors receive the proper signal they begin to reproduce and a mitotic burst is seen between the 2nd and 5th day after injury (347). Proliferating fibroblasts develop through cell divisions every 18–20 hours and remain in the mitotic phase for 30 minutes to 1 hour. The primary function of the activated fibroblast in the wound area is to produce collagen, elastin and proteoglycans. However,

**Fig. 1.18** Active fibroblast.

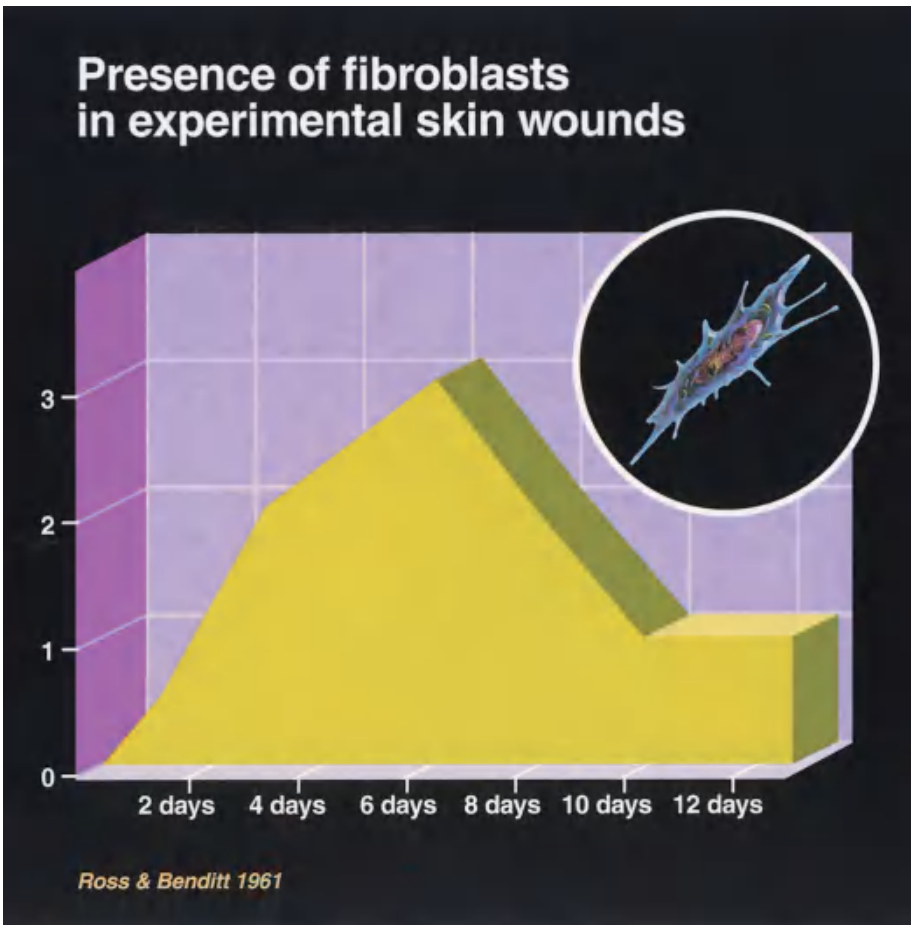


**Fig. 1.19** Schematic illustration of the presence of fibroblasts in experimental skin wounds in guinea pigs. The scale is semiquantitative, graded from 0 to 3. Adapted from (73).

during the mitotic phase, the fibroblast does not synthesize or excrete external components. Progression factors are necessary to stimulate the fibroblast to undergo replication. Before this can happen the fibroblast must be made competent. Factors that induce this competence are PDGF, FGF and calcium phosphate precipitates (26). PDGF-induced competence requires only transient exposure of cells to the factor. When competent, the fibroblast can replicate after stimulation by progression factors such as IGF-1, EGF and other plasma factors (26). This dual control of fibroblast proliferation explains why fibroblasts can remain in a reversible quiescent state in the presence of progression factor. The transition to a proliferative stage then awaits the release of competence factors by activated cells, such as platelets, macrophages and lymphocytes.

PDGF is released in response to injury by platelets and production is continued by activated macrophages, which can induce migration into the wound and proliferation of fibroblasts over an extended period of time (Fig. 1.4).

TGF-β is a growth stimulator for mesenchymal cells and has been found to accelerate wound healing in rats (348, 349) by direct stimulation of connective tissue synthesis by fibroblasts and indirect stimulation of fibroblast proliferation by PDGF (350, 351). Other factors that may be involved in fibroblast proliferation include TNF-α and TNF-β (26).

The best characterized inhibitor of fibroblast proliferation is β-fibroblast interferon (IFN-β). It has been suggested that IFN-β inhibits events involved in fibroblast competence and induction of competence of fibroblasts and by PDGF inhibition (352, 353).

Regulatory systems of fibroblast activity may operate through activation of macrophages, which generate an endogenous stimulus to fibroblast proliferation. Alternatively, fibroblast proliferation might be slowed by either inhibition of the release and activation of PDGF and TGF-β or by stimulation of inhibitory substances such as IFN-β. Clinically, this may suggest a specific stimulation of fibroblast proliferation by treatment with exogenous growth factors such as PDGF and TGF-β or by their activation (26).

Once fibroblasts have migrated into the wound, they produce and deposit large quantities of fibronectin, types I and III collagen and hyaluronidate. TGF-β is considered to be the most important stimulator of ECM production (354, 355).

Multiplication and differentiation of fibroblasts and synthesis of collagen fibers require oxygen as well as amino acids, carbohydrates, lipids, minerals and water. Collagen cannot be made in the mature fibroblast layer without oxygen (356–359). Consequently, the nutritional demands of the wound are greater than that of non-wounded connective tissue (315) and the demand is greatest at a time when the

local circulation is least capable of complying with that demand (316).

After collagen molecules are secreted into the extracellular space, they are polymerized in a series of steps in which the hydroxylysine groups of adjacent molecules are condensed to form covalent crosslinks. This step is rate dependent on oxygen tension and gives collagen its strength (356, 361).

Fibroblasts have been shown to have chemotactic attraction to types I, II, and III collagen as well as collagen-derived peptides, with binding of these peptides directly to fibroblasts (362, 363).

Fibroblasts have been known for many years to be involved in wound contraction. In relation to this phenomenon, a specific type of fibroblast has been identified which has the characteristics of both fibroblasts and smooth muscle cells for which reason they have been termed myofibroblasts. These cells are richly supplied with microfilament bundles that are arranged along the long axis of the cells and are associated with dense bodies for attachment to the surrounding ECM (364, 365). Besides numerous cytoplasmic microfilaments, large amounts of endoplasmic reticulum are also seen. In this respect these cells have characteristics of fibroblasts.

The myofibroblast has contractive properties and has been demonstrated in many tissues that form contracted and/or nodular scars (366). Myofibroblasts are present throughout granulation tissue and along wound edges at the time when active contraction occurs (367). For this reason the most generally accepted theory of wound contraction has involved a contribution by myofibroblasts (366).

Ehrlich and co-workers have presented a new theory for the phenomenon of wound contraction (368). In this fibroblast theory it is suggested that fibroblast locomotion is the mechanism that generates the contractive forces in wound contraction and that the connective tissue matrix is important in controlling these forces. It is suggested that the histologic existence of myofibroblasts is a transitional state of the fibroblast in granulation tissue.

In summary, the fibroblasts are latecomers in the inflammation phase of wound healing. Their main function is to synthesize and excrete the major components of connective tissue: collagen, elastin and proteoglycans. The fibroblast is also involved in wound contraction through a specific cell called the myofibroblast which has characteristics of both fibroblasts and smooth muscle cells.

### Endothelial cells and pericytes

The capillaries play an essential role in wound healing. The caliper of capillaries averages 9–12 µm, which is just enough to permit unimpeded passage of cellular elements. In cross-section the capillary wall consists of 1–3 endothelial cells (Fig. 1.20). Capillaries converge to form post-capillary venules of slightly larger size (15–20 µm). The endothelial cells are surrounded by a network of pericytes (Fig. 1.20). These cells appear to represent a pool of undifferentiated mesenchymal cells which have been found to participate in wound healing, and this applies also to the pulp and periodontium (see Chapter 5).

**Fig. 1.20** Endothelial cells and pericytes.

## Angiogenesis

Between 2 and 4 days after wounding, proliferation of capillaries and fibroblasts begins at the border of the lesion. However, studies have shown that blood cells and plasma perfuse the wound tissue several hours before the space is invaded by sprouting capillaries (295, 369). At first the blood cells move around randomly in the meshes of the fibrinous network but gradually preferential channels are formed in the wound through which cells pass more or less regularly. This phenomenon has been termed open circulation and it is suggested that the blood cells at this time are transported in a simple tube system which has not yet acquired an endothelial lining (295, 369, 370).

Angiogenesis is the process of formation of new blood vessels by directed endothelial migration, proliferation and lumen formation (262, 371–376, 628) (Fig. 1.21). In wound healing, angiogenesis is crucial for oxygen delivery to ischemic or newly formed tissue. New vessels arise in most cases as capillaries from existing vessels and only from venules (376, 377) (Fig. 1.21). In early granulation tissue, after the wound healing module is assembled, capillary sprouts move just behind the advancing front of macrophages. Collagen-secreting fibroblasts are placed between these sprouts and are nourished by the new capillaries (see Fig. 1.26).

Variants in healing of the vascular network are found according to the type of tissue involved and the extent of the injury. In skin or mucosal lacerations, primarily closed, existing vessels may anastomose spontaneously and thereby re-establish circulation. In wounds with tissue defects or in non-closed wounds, a new vascular network has to be created via granulation tissue. The third variant in vascular healing is

**Fig. 1.21** Neovascularization. New capillaries start as outgrowths of endothelial cells lining existing venules. Subsequent arcading sprouts unite and tubulization allows circulation to be established.

the revascularization of ischemic tissue as seen after skin grafting, tooth luxation, tooth replantation and transplantation. In these situations angiogenesis takes place in existing ischemic or necrotic tissue. The healing in these cases usually occurs as a mixture of gradual ingrowth of new vasculature combined with occasional end-to-end anastomosis between existing and ingrowing vessels (see Chapter 2).

Angiogenesis in wounds has been examined in different *in vivo* assays such as the rabbit ear chamber (269, 378), the Algire chamber where a transparent plastic window is placed in the dorsal subcutaneous tissue of a mouse (379) or the hamster cheek pouch (380, 381). Furthermore, angiogenesis has been tested in corneal pockets (379, 382, 383), and chicken chorioallantoic membrane (384). These assays have been used to describe the dynamic process of angiogenesis together with the influence of different types of external factors on vascular proliferation.

Our current knowledge of the biochemical nature of the signals that induce angiogenesis has been derived primarily from *in vitro* observations using cultured vascular endothelial cells. *In vitro* assays have been used extensively in the identification and purification of angiogenetic factors. In this context, as angiogenesis is considered to be a process of capillary growth, cultured capillary endothelial cells seem to be optimal for testing angiogenesis (385).

## Cellular events in angiogenesis

New capillaries usually start as outgrowths of endothelial cells lining existing venules. After exposure to an angiogenic stimulus, endothelial cells of the venules begin to produce enzymes that degrade the vascular basement membrane on the side facing the stimulus (636). After 24 hours the endothelial cells migrate through the degraded membrane in the direction of the angiogenic stimulus (Fig. 1.21). Behind the tip of the migrating wound edges, trailing endothelial cells divide and differentiate to form a lumen. The sprouts or buds can either connect with other sprouts to form vascular loops or can continue migrating. Capillary bud formation is found after 48 hours and these buds arise solely from venules (377).

When endothelial migration tips join to form capillary loops or join across a wound edge, blood flow begins within the formed lumen. As vessels mature, ECM components are laid down to form a new basement membrane (371, 376). Recent studies have shown that angiogenesis is closely related to fibroblast activity. Thus it appears that new vessels cannot grow beyond their collagenous support (356, 386).

The speed of neovascularization has been investigated in ear chambers and been found to range from 0.12 to 0.24 mm per day (272, 295, 387, 388). In dental pulps which become revascularized after replantation or transplantation, the speed is approximately twice this rate (see Chapter 2).

In raised and repositioned skin flaps, angiogenesis along the cut margins is rapid and capillary sprouts advance across the wound space from the host bed. In rats, new vascular channels across the wound margin can be demonstrated within 3 days; and in pigs, normal blood flow has been observed within 2–3 days (389–391). After tissue grafting, specific vascular healing processes take place. Thus it has been shown in skin transplants that after an initial contraction of the vessels, a so-called plasmatic circulation takes place in the zone next to the graft bed (392–400). This supply of fluids serves to prevent drying of the graft before blood supply has been restored (398). The role of the plasmatic circulation as a source of nourishment is, however, debatable (398).

Vascularization of skin transplants, although initially sluggish, takes place after 3–5 days (401–410). The role of already existing graft vessels is unsettled. Thus, in some studies, it has been shown that the original vessels act only as non-viable conduits for the ingrowth of new vessels (388), and that revascularization takes place primarily from invading new vessels (408, 411). In other experiments, however, it has been shown that, depending upon the degree of damage to the grafted tissues and on local hemodynamic factors, the original graft vessels may be incorporated in the established new vascular network (409–413).

Teeth have a vascular system which in some situations is dissimilar to skin. In replantation and transplantation procedures of immature incisors, the severed periodontal ligament and the pulp can be expected to become revascularized.

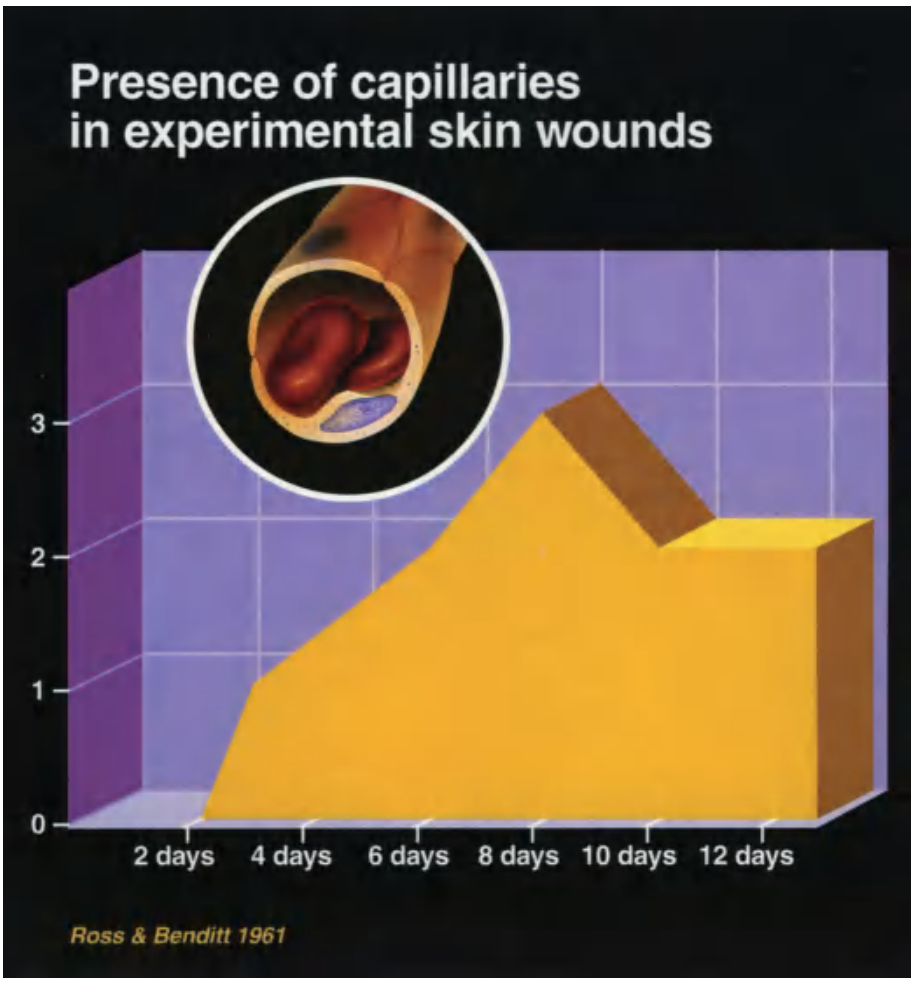


**Fig. 1.22** Schematic illustration of the presence of new capillaries in experimental skin wounds in guinea pigs. The scale is semiquantitative graded from 0 to 3. Adapted from (73).

The process of revascularization of the periodontal ligament seems to follow the pattern of skin grafts (see Chapter 2). There is limited diffusion of nutrients from the graft bed to the dental pulp due to its hard tissue confinement and extended length, which again leads to specific vascular healing events (see Chapter 2).

In a closed skin wound, circulation bridging the wound edges can be established as early as the 2nd or 3rd day and appears to be at a maximum after 8 days (73) (Fig. 1.22). The new vascular network is then remodeled. Some vessels differentiate into arteries and veins while others recede. The mechanism regulating this process is largely unknown. Active blood flow within the lumen may be a factor, as capillaries with decreased blood flow typically recede while those with active flow are usually maintained or expand into larger vessels (378, 414).

Factors determining angiogenesis represent a series of cellular and humoral events that lead to the initiation, progression and termination of angiogenesis (Figs 1.4 and 1.23). Initiation of angiogenesis appears to be related to signals released from activated platelets and fibrin at the site of vascular rupture (86, 285, 415, 416). During platelet activation, enzymes are released that degrade heparin and heparan sulfate components from the vascular basement membranes, whereby stored basic FGF (bFGF) is liberated (417–421). This liberation of FGF has been shown to induce angiogenetic activity (422). Other bFGF signals are released from injured cells and matrix (376). This growth factor is partly responsible for angiogenesis through initiating a cascade of events (423). Thus bFGF stimulates endothelial cells to secrete procollagenase, plasmin and plasminogen. Plasmin, as well as plasminogen, activates procollagenase to collagenase. Together, these enzymes can digest the blood vessel basement membrane. Subsequently, endothelial chemoattractants, such as fibronectin fragments generated from ECM degradation and heparin released from mast cells, draw endothelial cells through the disrupted basement membrane to form a nascent capillary bud (132).

A variety of growth factors, cytokines and lipid mediators produced in response to injury can stimulate angiogenesis. One of the most important proangiogenic mediators is vascular endothelial growth factor (VEGF or VEGF-A), and sufficient VEGF levels are believed to be essential for proper wound healing. VEGF is one of the most potent proangiogenic growth factors in the skin, and the amount of VEGF present in a wound can significantly impact healing. The activity of VEGF was once considered to be specific for endothelial cells lining the inside of blood vessels, partly because VEGF receptor (VEGFR) expression was believed to be restricted to endothelial cells. It is now known, however, that VEGFRs can be expressed by a variety of other cell types involved in wound repair. For example, keratinocytes and

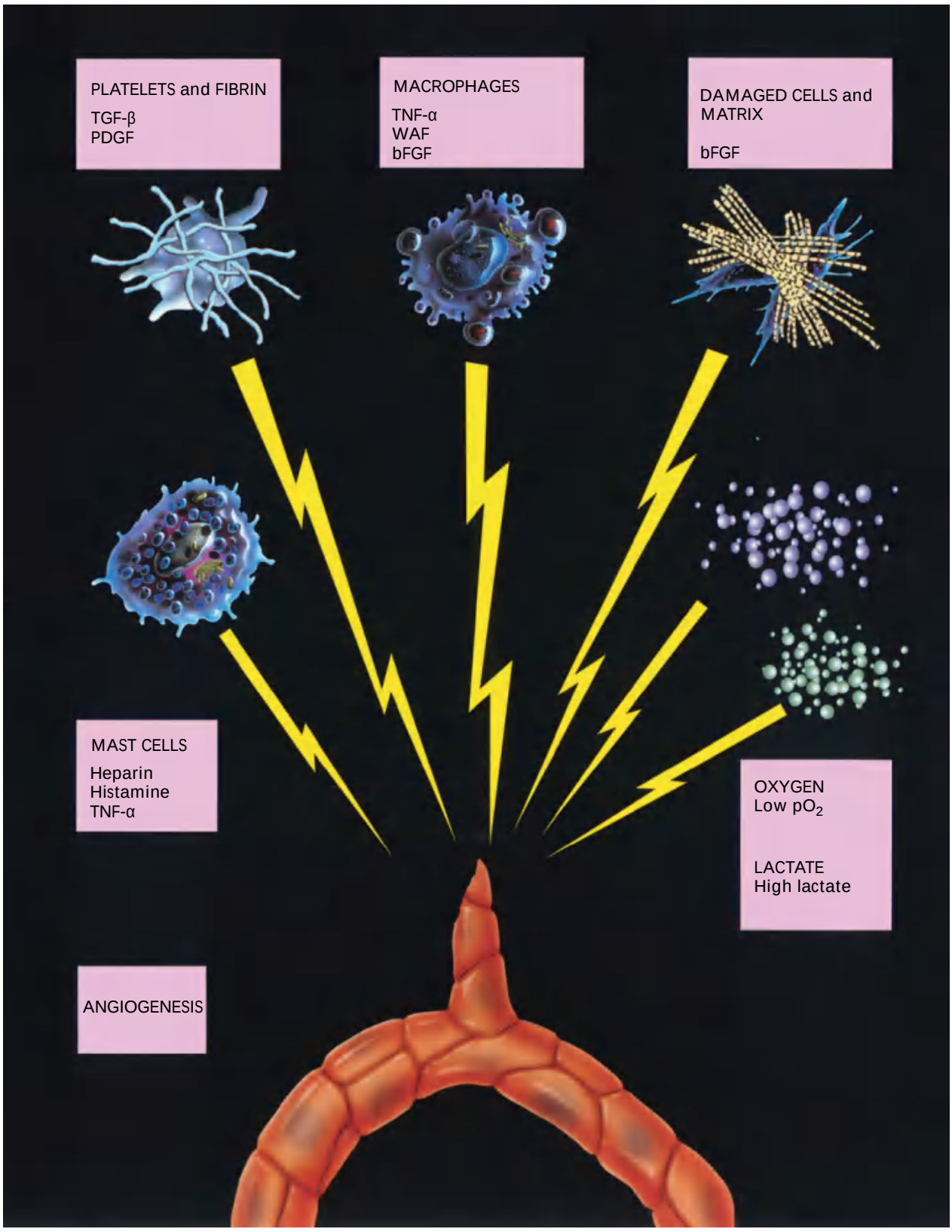


**Fig. 1.23** Cellular and humoral events leading to angiogenesis. Platelets, fibrin, mast cells, macrophages, injured cells and matrix all release angiogenic signals. Low oxygen tension and a high lactate concentration in the wound space represent also an important stimulation to angiogenesis.

macrophages, which both carry out important functions during wound healing, express VEGFRs and are capable of responding directly to VEGF (635).

Recruited and activated macrophages soon also promote angiogenesis (424) by liberating potent direct acting angiogenic factors such as TNF-α, wound angiogenesis factor (WAF) and FGF. The macrophage signal seems to diminish as angiogenesis proceeds (268, 269, 356, 361, 425–428). Recent studies indicate that the effect of hypoxia within the wound upon angiogenesis is possibly mediated via stimulated macrophages (see p. 42) (268, 429, 430).

### Other factors controlling angiogenesis

A number of angiogenetic factors have been isolated which either have a direct effect on endothelial cell migration/proliferation or have an indirect effect via other cells. The exact mechanisms behind indirect angiogenetic activity are not yet known, but it is possible that they cause accumulation of other types of cells, e.g. platelets or macrophages, that release direct acting factors (126, 376, 431).

Once new blood vessels form, they acquire a layer of pericytes and the composition of their basement membrane changes. Pericytes inhibit the growth of adjacent endothelial cells and thereby direct growth toward the site of attraction (432).

Finally, it should be mentioned that angiogenesis is dependent upon the composition of the ECM (433, 623, 624). Thus, fibrin appears to promote angiogenesis (64) and the fibrin–fibronectin extravascular clot serves as a provisional stroma providing a matrix for macrophages, fibroblasts and new capillary migration. In this way, the fibrin–fibronectin gel is transformed to granulation tissue.

## Wound strength development

Wound strength, from a functional point of view, is the most important property for a healing wound. The time interval from injury to a healed wound that is strong enough to resist mechanical stress in the tissue is important for the surgeon to consider. It is essential that an early return to normal life is facilitated with the development of significant wound strength. For ethical reasons this has to be based on investigations of the tensile strength of experimental wounds.

In early wounds the tensile strength is low and insufficient to keep the tissue edges together without sutures. The strength of the wound is in this stage mainly based on fibrin in the wound cavity. Later, in the proliferation phase, the strength increases rapidly as granulation tissue is formed. The strength of the wound lies in the collagen fibers, and is directly related to the collagen content of the tissue (434, 435) (Fig. 1.24). Some collagenous elements can be seen already after 2–3 days of injury, but the maximum period of collagen synthesis most often starts during the 5th to 6th days of healing. The collagen fibers are laid down in a random pattern and in the beginning possess little mechanical strength. Gradually, a more systematic pattern of collagen fibrils develops, leading to stabilization by crosslinking and assembly of fibers into a more correct anatomic pattern. Experimental studies have shown that the 'biochemical active zone' encompasses tissue 5–7 mm from the incisional line. Wound contraction also brings collagen fibrils together and organizes them perpendicular to the wound edges, increasing the mechanical strength of the tissue (644–646).

The resulting tensile strength of a wound is the combined strength of the old collagen (present in the wound area before injury), which diminishs through lyses of collagenases, and the increasing strength of the new-build collagen. The lowest tensile strength of a healing wound therefore occurs after some days of healing (20) (Fig. 1.25).

Wound strength can in a functional way be described as the relative tensile strength of the wound. This is the actual tensile strength of the wound in relation to the strength of intact tissue and is expressed as a percentage. Figure 1.25 shows the relative tensile strength of healing incisional wounds in different types of tissues. In tissues with a low collagen content in intact tissue (gastrointestinal tract, muscle) (436, 437), the relative tensile strength increases rapidly and reaches an intact level after 10–20 days. In tissues with a high collagen content (fascia, skin, tendon), the relative strength increases slowly and in skin and tendon the strength has after 60 days only reached 60% of the intact level (435) (Fig. 1.25).

Collagen constitutes the principal structural protein of the body and is the main constituent of ECM in all species. At least 13 types of collagen have been identified. Despite their differences, all collagen molecules consist of a triple helix matrix protein which gives the tissues their strength (15, 437–442). Literature on wound healing and collagen contains only sparse information about the different types of collagens (437).

Type I collagen is the major structural component of skin, mucosa, tendons and bone (440).

Type II collagen is located almost exclusively in hyaline cartilage.

Type III collagen, also called reticular fibers (443), is found in association with type I collagen although the ratio varies in different tissues (440). In a rat model, type III collagen could be demonstrated 10 hours after the start of wound healing in skin (444), and after 3 days in healing (PDL wounds (445, 446). The early appearance of type III collagen has been found associated with the deposition of fibronectin, indicating that type III collagen together with fibronectin may provide the initial scaffolding for subsequent healing events (447, 448).

In children, type III collagen can be detected between 24 and 48 hours in skin wounds, whereas no type I collagen is found in this type of wound (449). From 72 hours and onwards a substantial increase in type I collagen is found, together with the appearance of mature fibroblasts (449).

Type IV collagen, together with other components, including heparan sulfate, proteoglycans and laminin, makes up the basement membranes in both the epidermis and endothelium.

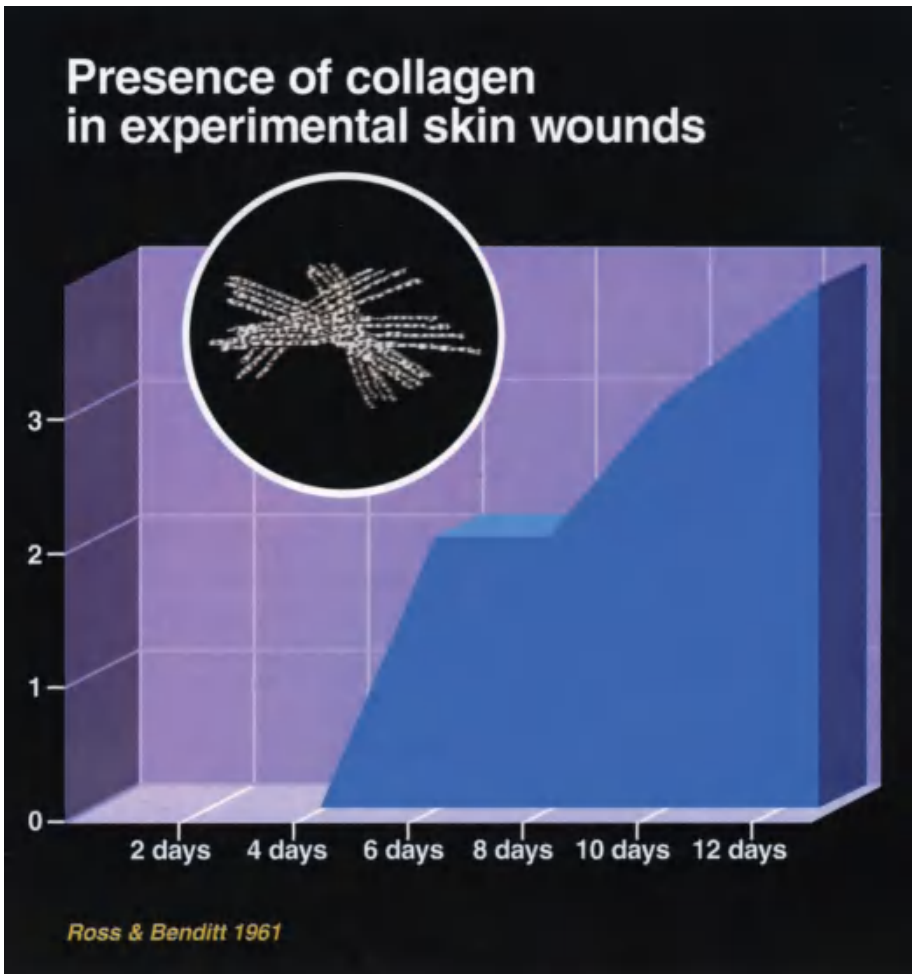


**Fig. 1.24** Schematic illustration of the presence of collagen in experimental skin wounds in guinea pigs. The scale is semiquantitative graded from 0 to 3. Adapted from (73).

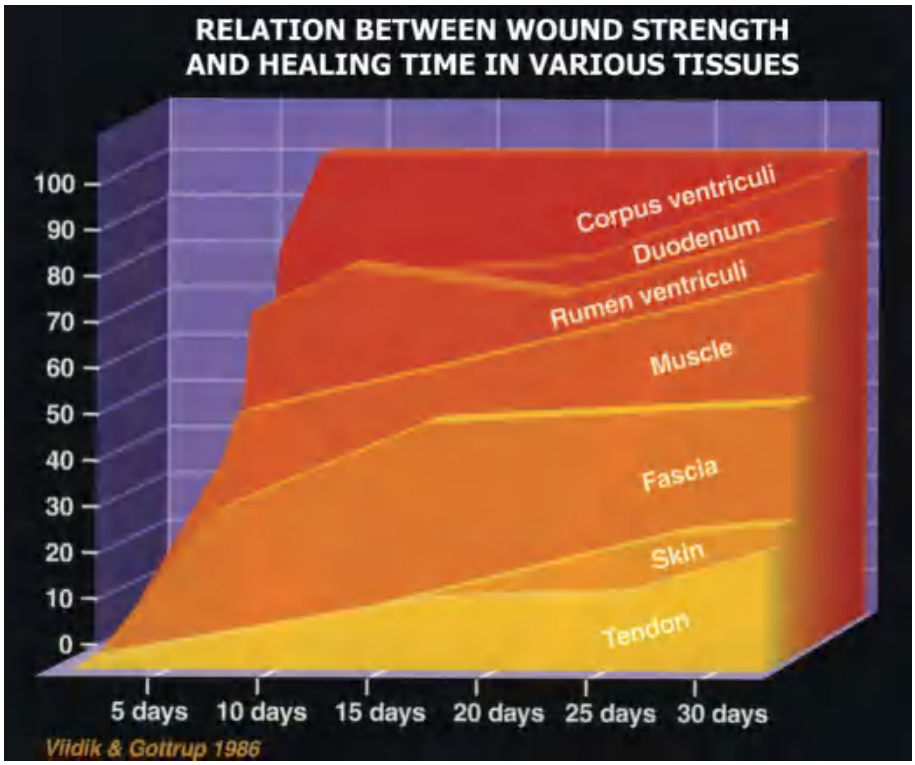


**Fig. 1.25** Relative healing rates for linear incisional wounds in different tissues in rats and rabbits; the tensile strength is calculated as a percent of that of the respective intact tissues (taken as 100%). Reproduced from (457) with permission.

In dermal wound healing, type IV collagen synthesis by epidermis is connected with the reformation of the basement membrane and is a relatively late event in the wound healing process (450–452).

Type V collagen is found in almost all types of tissue and has been proposed to be involved in the migration of capillary endothelial cells during angiogenesis (441). Type V collagen is synthesized while epidermal cells migrate; however, the regeneration of the basement membrane is delayed until the wound defect is covered and the epidermal cells are no longer in a migrating phase (451).

Type VII collagen has been found to be an anchoring fibril that attaches the basement membrane to the underlying connective tissue (454–456).

The remaining types of collagen are less known in relation to the wound healing process and are therefore not discussed.

Collagen represents a key component in wound healing. Thus, immediately after injury the exposure of collagen fibers to blood results in platelet aggregation and activation with resultant coagulation and the release of chemotactic factors from platelets that initiate healing (e.g. PDGF, platelet factor IV, IGF-1, TGF-β and an unidentified chemoattractant to endothelial cells). Collagen fragments are then degraded by the attracted neutrophils and leukocytes, which leads to attraction of fibroblasts.

Synthesis of new collagen in a wound starts with fibroblast proliferation and invasion and the deposition of a collagen-based ECM. This event takes place after 3–5 days and persists for 10–12 days. During this period of time, there is a rapid synthesis primarily of type III and later of type I collagen, resulting in an increase in the tensile strength of the wound which is primarily dependent on the build up of type I collagen (449, 458–460).

## Remodeling phase

Remodeling of the ECM is a continuous process that starts early in the wound healing process and turns the abundant and poorly organized granulation tissue ECM into a mature connective tissue. Thus most fibronectin is eliminated within 1 or 2 weeks after granulation tissue is established. Hyaluronidate is replaced or supplemented with heparan sulfate proteoglycans in basement membrane regions and with dermatan chondroitin sulfate proteoglycans in the interstitium (61).

Type III collagen fibers are gradually replaced by type I collagen, which becomes arranged in large, partly irregular, collagen bundles. These fiber bundles become oriented according to lines of stress and provide a slower increase in the tensile strength of the healing wound than that found during the proliferation phase (460). In most tissues, this remodeling phase ultimately leads to the formation of a scar.

The functional properties of the scar tissue vary considerably depending on the content of collagen in the intact original tissue. The healing rate, measured as the mechanical strength of the wound compared to adjacent intact tissue, therefore varies from one tissue type to another. In tissue with a low collagen content before injury (e.g. gastrointestinal tract (461) and other intra-abdominal organ systems), the primarily closed wound shows a rapid increase in relative strength (strength of wounded tissue compared to intact tissue) (457). As shown in Fig. 1.25, tensile strength close to that of intact tissue levels is reached after 10–20 days of healing in tissues with a low collagen content before injury. In tissues with a high collagen content (e.g. tendon and skin) the increase in relative strength is much slower; and more than 100 days of healing are needed to achieve half the strength of intact tissue. In the wounded PDL, a very rapid increase in tensile strength has been found after severance of Sharpey's fibers (see Chapter 2) Investigations of wounds closed 3–6 days after injury (delayed primary closure) has shown that this type of wound was significantly stronger than primarily closed wounds after 20 days of healing. After 60 days of healing, delayed primarily closed wounds were almost twice as strong as primarily closed wounds; and this difference persisted after 120 days (59, 60, 462). The mechanisms behind the different wound strength in primarily and delayed primarily closed wounds are probably related to an increase in tissue perfusion and oxygenation due to increased angiogenesis and oxygen delivery to the tissues (463).

Remodeling starts when wound contraction has assembled the collagen fibrils into thicker bundles and aligned them perpendicularly to the wound edges. The timing of the start of this stage depends on the size of the wound, and whether the wound healing occurs by primary or secondary intention. During the remodeling stage, collagen crosslinking also gradually increases, improving the stability of the tissue, and there is a gradual maturation of the tissue so that the aligned collagen fiber bundles are reorganized to the typical and more resilient basketweave organization found in normal connective tissue. Remodeling is a slow process and continues slowly and can last for months or in some cases for years (639, 640).

### Hypertrophic scar and keloid

As mentioned above, if remodeling does not lead to complete structural regeneration of the tissue, scar formation may occur. In the mildest form, scars may contain some excess of collagen-rich ECM where collagen is organized in thick parallel bundles instead of the mechanically stronger basketweave that is present in normal tissue. Therefore, scars can gain at best only about 70–80% of the breaking strength of normal skin. However, in more severe cases, ECM deposition and degradation is not balanced, leading to progressive ECM accumulation and formation of pathologic scars, namely keloids or hyperthrophic scars in the skin. Therefore, biologic processes that regulate the ECM abundance and organization are critical for appropriate wound healing outcome. At least four different mechanisms likely contribute to these processes: First, the number of cells secreting ECM is reduced. Second, cells degrade and

remodel ECM components to reduce their abundance. Third, ECM is reorganized and its stability is increased by collagen crosslinking. Fourth, secretion of ECM by the cells is downregulated (628).

Excessive deposition of scar tissue is a clinical problem that has been difficult to resolve because of the lack of reliable animal models. Hypertrophic scar and keloid are both characterized by excessive accumulation of ECM, especially collagen. The etiology is not known but abnormalities in cell migration and proliferation, inflammation, syntheses and secretion of ECM proteins and cytokines, and remodeling of wound matrix, have been described. Also, increased activity of fibrogenic cytokines (e.g. TGF-β1, interleukin-1), abnormal epidermal–mesenchymal interaction and mutations in regulatory genes have been proposed (464). In healed burns the development of hypertrophic scars seems not to be the result of a continued proliferation phase, rather an alteration of the remodeling phase (465).

The hypertrophic scar results from a full thickness injury and is characterized by a thick, raised scar that stays within the boundaries of the original injury. Keloids can develop from superficial injuries and exceed the boundaries of the initial injury. Histologically hypertrophic scars contain nodules and keloid does not. The collagen bundles on the surfaces of the nodule are arranged in parallel sheets, while randomly arranged fibrils occur within the centre (466). The collagen bundles of keloid are arranged in braided sheets running parallel to each other.

Hypertrophic scars most often regress over time and can be corrected by surgical intervention (620). Keloid scars as a rule do not regress over time and frequently recur after removal. Treatment of keloid scars is difficult, but pressure dressings and local application of glucocorticoids have been used with limited success. Silicone dressings and local use of calcium channel blockers have recently shown promising results.

## Epithelial cells

Different epithelial cells have various features of interest:

- Keratinocytes become exposed to novel ECM molecules in wounds.
- Integrins are signaling molecules that mediate cell adhesion to ECM and cell migration.
- Epithelial keratinocytes need to readjust their cell adhesion receptors to interact with the wound matrix and to modulate cellular signaling.
- Fibronectin receptor integrins regulate multiple aspects of re-epithelialization.
- Several types of cell adhesion molecules in different wound cells jointly contribute to wound healing.
- The EGF family of growth factors promote keratinocyte migration and proliferation.
- TGF-β1 promotes keratinocyte migration but inhibits proliferation.
- Wound-induced electrical fields direct re-epithelialization, but may fail to re-epithelialize chronic wounds (629).

Epithelium covers all surfaces of the body, including the internal surfaces of the gastrointestinal, respiratory and genitourinary tracts. The major function of epithelium is to provide a selective barrier between the body and the environment. The epithelial barrier is the primary defense against threats from the environment and is also a major factor in maintaining internal homeostasis. Physical and chemical injury of the epithelial layer must therefore be repaired quickly by cell proliferation.

After injury to the epidermis, wound protection is provided in two steps: within minutes there is a temporary coverage of the wound by coagulated blood which serves as a barrier to arrest the loss of body fluids. The second step is the movement of adjacent epithelium beneath the clot and over the underlying dermis to complete wound closure. Re-epithelialization of an injured surface is achieved either by movement or growth of epithelial cells over the wound area (467–469). In early phases of wound healing the most important process is cell migration, which is independent of cell division (470–472).

In deep wounds the new epithelial cover arises from the wound periphery, whereas shallow wounds usually heal from residual pilosebaceous or eccrine structures (405, 473–475).

The cellular response of epithelial cells to an injury can be divided into four basic steps: *mobilization* (freeing of cells from their attachment); *migration* (movement of cells); *proliferation* (replacement of cells by mitosis of pre-existing cells); and *differentiation* (restoration of cellular function, e.g. keratinization).

The first response of the epithelium after injury is *mobilization*, which starts after 12–24 hours. This involves detachment of the individual cells in preparation for migration. Epithelial cells lose hemidesmosomal junctions; the tonofilaments withdraw from the cell periphery; and the basal membrane becomes less well defined (15, 467, 476, 477). In addition, the cells of the leading edge become phagocytic, engulfing tissue debris and erythrocytes. Epithelialization occurs most rapidly in superficial wounds where the basal membrane is intact. Short tongues of epithelial cells grow out from the residual epithelial structures. By the 2nd or 3rd day, most of the wound base is covered with a thin epithelial layer; and, by the 4th day, by layered keratinocytes (453, 478, 479).

*Migration* of epithelial cells occurs as the movement of clusters or sheets of cells. This movement of epithelial cells has been proposed to take place in a 'leapfrog' fashion of epidermal sheet movement (480). Fibronectin in connection with fibrin seems to make a provisional matrix for cellular anchorage and self-propelling traction of the epithelial cell for migration. A speculative mechanism has been that fibronectin is produced in front of the wound edge by epidermal cells, and these then slide over the deposited fibronectin matrix and finally break down this matrix at a distance of some cell diameters behind the wound edge (481, 482). It seems that the motile cells use secreted fibronectin as a temporary basal membrane and use collagenase and plasminogen activators to facilitate passage through reparative connective tissue (483).

The specific signals or stimuli for epithelialization are unknown. However, it is known that the trauma of wounding causes an activation of epithelial keratinocytes by the exposure to pro-migratory matrix molecules, growth factors and cytokines that are released, and leads to wound-generated electrical fields (629).

Migration of epithelial cells takes place in a random fashion; however, orientation of the substrate on which the cells move, as well as the presence of other cells of the same type, are determinants of the extent and direction of cell movement. Furthermore, cell migration appears to be at least in part initiated by a negative feedback mechanism from other epithelial cells in the free edge of the wound (5). Substances in the substrate which are important for direction of the migration seem to be collagen fibers, fibrin and fibronectin, as earlier described. Fibronectin appears to be a substrate for cell movement and to have a binding capacity for epithelial cells as well as monocytes, fibroblasts and endothelial cells (101, 482–484).

Proliferation of epidermal cells starts after 1–2 days in the cells immediately behind the migrating edge, thereby generating a new pool of cells to cover the wound (472, 485). Mitosis in epidermal epithelium has a diurnal rhythm, being greatest during rest and inactivity. In normal epidermis, very few basal cells are in mitosis at any given time. Epidermal wounds, however, result in a change of the diurnal mitotic rhythm in cells adjacent to the wound, resulting in an absolute increase in mitotic activity and an increase in the size of epidermal cells (453). The maximal mitotic activity is found on the 3rd day and continues until epithelialization is complete and epithelial cells have reverted to their normal phenotype and reassumed their intercellular and basement membrane contacts by differentiation (486).

A number of stimuli for epidermal cell growth and thereby wound closure have been indentified, such as calcium in low concentration, interleukin-1, bFGF, EGF, PDGF and TGF-α (467, 482). The only factor known to block epithelial growth is TFG-β (478, 487). Most of these molecules are released from cells within the wound environment such as platelets, inflammatory cells, endothelial cells and smooth muscle cells (Fig. 1.4).

Another factor that influences epithelialization is oxygen tension. Thus a high $pO_2$ has been found to increase epithelialization (488, 489).

Epithelial repair differs temporally in different types of wound. In incisional wounds, mobilization and migration of epithelial cells is a rapid response compared to other events of the wound healing process. Already after 24–48 hours the epithelial cells have bridged the gap in clean incisional and sutured wounds (142). In small excised and non-sutured wounds which heal by secondary intention the surface is initially covered by a blood clot. Migrating epithelium does not move through the clot, but rather beneath it in direct contact with the original wound bed. Epithelial cells appear to secrete a proteolytic enzyme that dissolves the base of the clot and permits unimpeded cell migration.

In large excised wounds, all stages of epithelial repair may be seen simultaneously. In such wounds epithelialization will not be complete before granulation tissue has developed. Epithelial cells will use this bed for subsequent migration. Depending upon the size of wound, the surface will subsequently be covered by a scar epithelium which is thin, and lacks strong attachment to the underlying dermis as well as lacking Langerhans' cells and melanocytes.

One factor that has a strong influence upon epithelial healing is the depth of the wound. In superficial wounds, the regeneration from hair follicles coincides with epithelialization from the wound edges. In deeper wounds, all epidermal regrowth occurs from the wound edge.

Finally, it should be mentioned that re-epithelialization is significantly enhanced if the wound is kept moist (481).

In the oral cavity the morphological changes seen during epithelialization of the rat molar extraction socket appear to be similar to wounds that involve oral mucosa (74, 490–494). Thus the epithelium migrates down into and across the wound with either fibrin–fibronectin or granulation tissue–fibronectin below it and the superficial wound contents (i.e. neutrophil leukocytes, tissue debris, food elements and bacteria) above it. This layer is subsequently lost in the form of a scab following re-epithelialization (492).

## Microenvironment in wounds

Microenvironments in wounds are the sum of the single processes mentioned earlier. Of particular interest for the wound healing process is the influence of the wound microenvironment as an initiator, supporter and terminator of the wound healing processes (495).

Cellular activity in the wound has already been discussed in detail, but can be described as three waves of cells invading the wound area. Apart from their role in hemostasis, platelets serve as the initiators of wound healing by their release of substances such as growth factors (e.g. PDGF, platelet factor IV, IGF-1 and TGF-β) and an uncharacterized chemoattractant of endothelial cells at the moment of injury (Fig. 1.4). As the access of platelets to the wound area is limited by the coagulation process, the supply of these factors is limited.

The second set of cells, polymorphonuclear leukocytes, migrate into the wound after a few hours largely under the direction of complement factors. Their role in the wound healing process appears mainly to be the control of infection.

The third type of cell invading the wound area are the monocytes, which are attracted to the injury site by platelet factors, complement and fibrinopeptides. After entering the wound, these cells are transformed to macrophages and take over the control of healing processes. It would appear that macrophages have the capacity to detect and interpret changes in the wound environment and thereby initiate appropriate healing responses.

In the early wound, cells float around in the tissue fluid of the wound and their function and movement are directed by different growth factors (e.g. TGF-β, IL-6, IGF-1 and insulin produced by platelets and/or macrophages).

The amino acid content of wound fluids reflects to some extent the metabolic events. Amino acid concentrations are initially close to those of serum. Later, they approach those of inflammatory cells. After some time, levels of glutamine and glutamate in particular, rise well over those of serum, whereas arginine concentration falls to low levels due to conversion to ornithin and citrullin (495). Arginine has been shown to be active in influencing the wound healing process and seems to activate macrophages (496, 497).

It would seem that the wound fluid, with its mixture of growth factors, amino acids and other components, is conducive to cell proliferation. Thus, it has been found experimentally that cell growth was optimized in the presence of wound fluid compared to cell growth in serum (327).

The concentration of oxygen in the wound plays a very significant role in activation of the wound healing process and control of wound infection (648–653).

Wound microenvironments have been studied in rabbit ear chambers in which healing tissues can be visualized between closely approximated, optically clear membranes mounted under a microscope (Fig. 1.26). This narrow space, as thin as 50 µm, forces healing cells to travel in coherent order, so that one or two cells pass at a time (49). In this model, the influence of oxygen tension and lactate concentration could be measured by microprobes and has been characterized (49). An oxygen gradient from the central wound space in the chamber to the peripheral normal tissue has been described. While oxygen tension along the edges of the wound is very low, with hypoxia close to 0 mmHg, the oxygen tension at the periphery of the wound is up to 100 mmHg. This oxygen tension gradient seems to be important in the initiation of the wound healing process. Concurrently, lactate concentration in the wound space is 10–20 times that of venous blood, resulting in a fall in pH to 7.25 (498).

## Wound healing module

After the first phase of the inflammatory process occurs, a characteristic cell build-up is found after 3–4 days (Fig. 1.26). At the edge of a wound is a vanguard of scavenging cells, the majority of which in the non-infected wound are macrophages. If the wound is infected, these macrophages are accompanied by large numbers of neutrophilic leukocytes.

Beneath the phagocytes is a layer of immature fibroblasts, floating in a gelatinous, non-fibrillar matrix, which are unable to divide because of local hypoxia. Beneath this layer of fibroblasts is a group of dividing fibroblasts which are associated with the most distal perfused arcaded capillaries, and behind the first perfused capillary loops are more dividing fibroblasts which provide cells to form the new tissue.

Distal to this zone, blood vessels increase in size and become less dense, probably as a result of enlargement or coalescence of a few vessels, as the other channels recede from the pattern of vascular flow. Between these vessels lie mature fibroblasts and new fibrillar collagen. This arrangement of

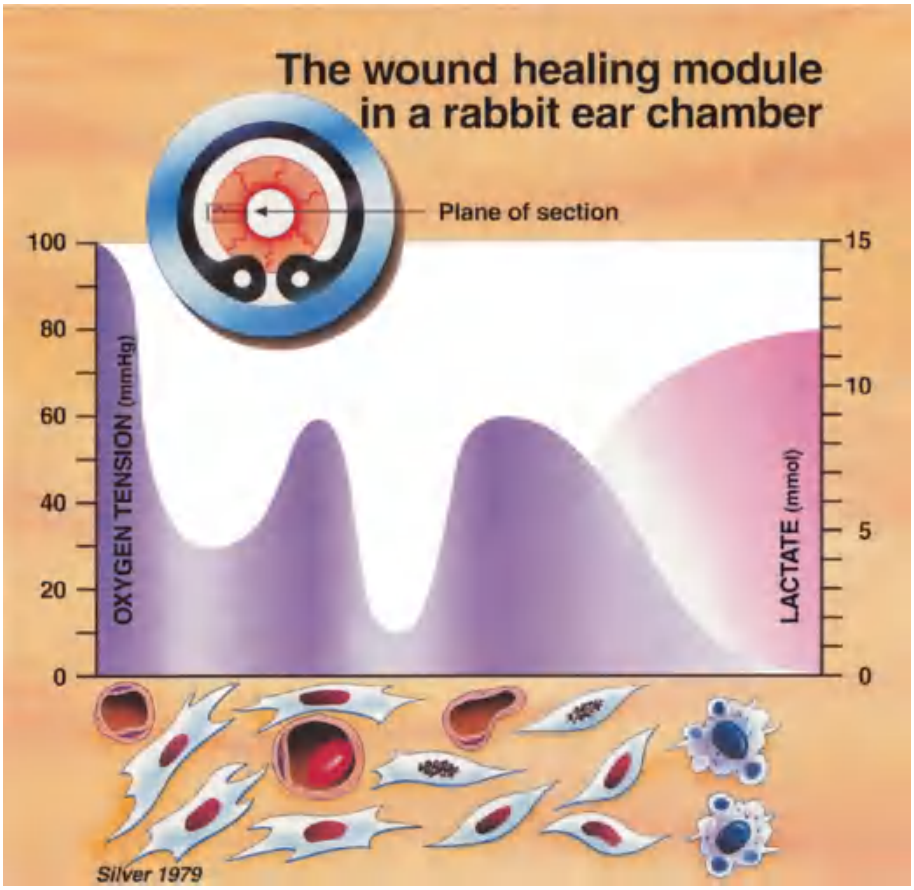


**Fig. 1.26** Cellular build up, oxygen tension and lactate concentration in a rabbit ear chamber The center of the wound is to the right on the drawing. At this location where macrophages operate there is very low oxygen tension and a high lactate concentration. It appears that oxygen tension is closely related to the location of the vasculature. Replication of fibroblasts takes place ahead of the regenerating vessels, whereas fibroblasts begin collagen production when the neovascular front reaches the proliferating fibroblasts. From (499).

cells creates an environment that is favorable to angiogenesis and collagen deposition and has been termed the *wound healing module* (Fig. 1.4).

Fibroblasts in mitosis are always found just ahead of the regenerating vessels, where the tissue oxygen tension is optimal for replication (i.e. about 40 mmHg). It is assumed that these fibroblasts behave as growth centers and that new fibroblasts remain at this location until reached by the neovascular zone, whereafter they initiate collagen production (499). The hyperemic neovascular front has a higher $pO_2$ (Fig. 1.26), which is optimal for collagen synthesis. Thus the fibroblasts replicate and produce collagen in different wound environments.

It has been found that hypoxia or exposure to a high lactate concentration increases the capacity of fibroblasts to synthesize collagen when they are subsequently placed in an oxygenated environment (500, 501). It has been suggested that lactate stimulates collagen synthesis (495). When lactate increases, the construction phase of healing starts by stimulating the transcription and post-translational modification of wound-related genes such as collagen genes, matrix metalloproteins and others (647). This carries healing on to its proliferative phase and thus collagen deposition. The lactate also enhances TNF production by lipopolysaccharide, collagen lysis (remodelling), hif production and angiogenesis (648).

Thus lactate has been shown to induce an increase in procollagen mRNA. Lactate concentration in test wounds is greatest in the central space and persists well into the zone of collagen synthesis (Fig. 1.26). It is therefore suggested that new vessels overtake the immature fibroblasts and change their environment from high lactate and low oxygen tension to high lactate and high oxygen tension and thereby increase both their collagen synthesis and their deposition. When the wound cavity is totally filled with granulation tissue, the hypoxia and high lactate concentration gradually diminish as does macrophage stimulation, whereupon the wound healing process will stop.

Oxygen tension in the wound has been shown to be important for collagen deposition and the development of the tensile strength of the wound (488, 502), for regulation of angiogenesis (369) and for the epithelialization of wounds (472). Oxygen in the wound's extracellular environment also has an important role in the intracellular killing of bacteria by granulocytes (503).

Neutrophil migration, attachment and ingestion of bacteria apparently are independent of oxygen; however, the killing of the most important wound pathogens is achieved by mechanisms that require molecular oxygen (504). These mechanisms are introduced through reducing extracellular molecular oxygen to superoxide which is then inserted into phagosomes. Here the superoxide is converted to high-energy bactericidal oxygen radicals for optimal bacterial killing (505).

Several studies in normal volemic animals have shown that they clear bacteria from wounds in proportion to the fraction of oxygen in the inspired gas. Infection was less invasive or failed to develop in hyperoxygenated guinea pigs when bacteria were injected into skin (505). Furthermore, infected skin lesions in dogs became invasive in tissues with an extracellular fluid oxygen tension under 60 mmHg; however, they remained localized if the tissue was better oxygenated, as in the case of higher inspiratory oxygen concentration (506). Experience in human subjects tends to support these findings, but no trial has yet been performed supporting these observations.

## Factors affecting the wound healing process

External factors have an influence on the wound healing process. The categorization is generally divided into local or systematic. In acute surgical wounds a classification based on patient- and surgical-related factors is most convenient. Patient-related factors are associated with the single patient and can alone, or together with other factors, inhibit or prevent wound healing. Surgical-related factors can be separated in pre-, peri- and postoperative influencing factors.

### Blood circulation and oxygenation of the wound

Continuous supply of oxygen to the tissue through a sufficient tissue perfusion is vital for the healing process as well as resistance to infection (360, 488, 502, 507–509, 648, 649). Collagen production and development of strength of the wound is directly correlated to the partial pressure $pO_2$ of the tissue ($p_tO_2$). Epithelialization is also dependent on oxygen, but the humidity of the wound healing environment seems of more importance. Moist wound healing increases the epithelialization by a factor of 2–3, while the optimal growth of epidermal cells is found at an oxygen concentration of between 10% and 50%.

Anemia with hematocrite values of 15–20 has, in experimental animal studies, in cases of normal function of the heart and normal tissue perfusion, been of minor importance for the $pO_2$ in the wound area and consequently for the healing. Evaluation of tissue perfusion and oxygenation is important in all types of wound. Monitoring systems should measure the hemodynamic situation and the ability of the cardiovascular system to deliver an adequate volume of oxygen to meet the metabolic demands of the peripheral tissue.

Tissue perfusion is determined by a variety of general and local factors. Peripheral tissue perfusion is influenced by multiple cardiovascular regulatory mechanisms. In response to hemorrhage, these mechanisms maintain blood flow to vital organs, such as the heart and brain, while blood flow to other tissues is decreased (510, 511). Circulatory adjustments are effected by local as well as systemic mechanisms that change the caliber of the arterioles and alter hydrostatic pressure in the capillaries. Detection of poor tissue perfusion, and especially tissue oxygenation, is crucial in the post-injury and care periods.

If wound edges show signs of ischemia, there is a risk of impaired wound healing with the development of wound leakage and infection. From the knowledge of healing wounds

of the oral cavity and the anus, it is obvious that perfusion is the major factor in resistance to infection. Despite contamination of both types of wound with bacteria, they almost always heal without infection if patients have a normally functioning immune system. The difference between these and other wounds (e.g. in the extremities) is not related to local immunity, but to differences in perfusion and oxygenation.

Increased oxygen tension improves resistance to infection through local leukocyte function (512). Thus, experimental data have shown that the killing capacity of granulocytes is normal only to the extent to which molecular oxygen is available (503, 513–519). Bacteria killing involves two major components. The first is degranulation of neutrophils in which bacteria located within the phagosome are exposed to various antimicrobial compounds from the granules. The effect of this system is unrelated to the environment of the leukocytes. The second system is the so-called oxidative killing and this depends upon molecular oxygen absorbed by the leukocytes and converted to high energy radicals such as superoxide, hydroxyl radical, peroxides, aldehydes, hypochlorite and hypoiodite – all substances which to varying degrees are toxic to bacteria. In this regard it is important to consider that the efficacy of oxidative bacteria killing is directly proportional to local oxygen tension (539).

The clinical relevance of blood flow and oxygen supply to healing and infection has been shown experimentally in skin flaps in dogs (32, 392, 395). In flaps with a high perfusion and tissue oxygen tension, no infection was found after injection of bacteria; whereas invasive, necrotizing infections were found in flap areas in which oxygen tension was less than 40 mmHg. When oxygen tension in inspired gas and arterial blood was raised or lowered, the infection rate corresponded to tissue oxygen tension, but not to oxygen-carrying capacity.

With respect to the healing of dental tissues, the oxygen tension may have a significant effect. Thus, several *in vivo* tissue culture studies have shown that low oxygen tension (e.g. a 5% oxygen atmosphere) results in reduced collagen and bone formation. A concentration of 35% $O_2$ was found to be optimal for collagen and bone formation, while a high oxygen concentration (95%) resulted in depression of collagen and bone formation as well as osteoclastic resorption of bone and cartilage (520–522). This *in vitro* relation between high oxygen concentration and osteoclastic activity may have an *in vivo* counterpart in vanishing bone disease (Gorham's disease or vanishing facial bone) (523, 524) as well as in the internal surface resorption phenomenon seen in revascularization of the pulps of luxated or root-fractured teeth (525). In both instances active local hyperemia and increased oxygen supply may be related to osteoclastic activity.

Among local factors which can control blood supply and tissue perfusion to the injured tissue, tension caused by splints or sutures may seriously jeopardize local circulation. Thus, splinting types exerting pressure on the periodontium may disturb or prevent uneventful PDL or pulp healing and lead to disturbances, such as root resorption, ankylosis and pulp necrosis (see Chapter 2). Furthermore, tension of sutured soft tissue wounds may lead to ischemia with subsequent risk of wound infection (see Chapter 21).

## Topical oxygenation

Locally, oxygen has been applied to the wound surface in order to increase regeneration of epithelium, but topical oxygen therapy approaches are not yet widely used in wound management anywhere in the world. While the effect of this treatment has been well documented, there has been scepticism largely because of the absence of randomized controlled trials. However, growing evidence of its effectiveness suggests it has the potential to form a regular part of adjunctive therapies in treatment regimens to speed up healing of acute and chronic wounds (650–652, 694).

## Hyperbaric oxygenation

Hyperbaric oxygen (HBO) has been introduced in the treatment of various oral conditions such as problem wounds subsequent to irradiation as well as in cases of grafting procedures where vascularization appears compromised, as well as in other types of chronic wounds (526, 617, 618, 694).

HBO is administered in pressurized tanks where the patients inhale 100% oxygen at a pressure of 2 atmospheres. The interaction of HBO with hypoxic tissue has a range of effects (7). The most significant effect of HBO is that it augments the oxygen gradient within the wound healing site, thus leading to increased fibroblast and endothelial activity (269, 527–530) as well as increased epithelialization (489). HBO may also suppress the growth of certain bacteria (512).

In experimental gingival wounds in rats, it has been found that HBO augments gingival connective tissue healing during the first 2 weeks, whereafter no difference was seen in comparison to wounds healed at normal atmosphere (531). In more extensive wounds in rats, where mandibular ramus osteotomy wounds severed the neurovascular supply in the mandibular canal, it was found that HBO reduced or prevented ischemic damage to pulp cells, ameloblastema and adjacent bone on a short-term basis (i.e. after 10 days). With an observation period of 30 days, HBO was found to stimulate osteodentin and bone formation in the zones of injury (532).

This beneficial effect of HBO on bone healing after injury is supported by a human study where acceleration of bone healing after osteotomy could be demonstrated after the use of hyperbaric oxygen (533, 619). Osteoradionecrosis of the jaws is another condition treated with HBO and the timing of surgical intervention is an important variable with respect to success (647). A standard protocol shown to be effective is 20 preoperative HBO treatments before oral surgery. When HBO is used without surgical intervention it has no lasting benefit (648). The effect of HBO in the treatment of diabetic foot lesions is, however, still debated. In a longitudinal observational cohort study Margolis et al. (654) it was concluded that the usefulness of HBO in the treatment of

diabetic foot ulcers needs to be better clarified, preferably using well-designed randomized controlled trials and perhaps using other healing-based end points other than a healed wound.

## Smoking and alcohol

Smoking influences the healing process by different mechanisms. Nicotine is quickly absorbed and starts a release of catecolamines resulting in a peripheral vascular constriction followed by a decrease in perfusion rate of 42% (534). Furthermore the CO in the cigarette smoke will reduce the oxygen content of the blood. These combined effects have been shown to decrease the tissue perfusion by more than 30% in more than 45 minutes in specific areas of the body (535). In such areas the production of collagen is 1.8 times higher in non-smokers compared to smokers (536). Leukocytes in smokers have also shown a decreased ability to kill bacteria resulting in a higher risk of wound infections in smokers. In surgical patients an increased risk of necrosis of the wound edge, diminished cosmetic result, increased risk of anastomic leakage after bowel surgery, and increased recurrence rate after hernia surgery have been described (49).

A study found that healthy smokers have a higher incidence of wound infections and wound ruptures than never-smokers, and 4 weeks of abstinence from smoking reduces wound infections to a level similar to that found in never-smokers (537). Smoking has to be considered within a pathophysiologic model as delaying healing and increaseing wound infection rate. Biologic markers of acute wound healing affected by smoking may appear to be valuable predictors for adverse wound events after surgery (650, 651, 655).

Alcohol has also been shown to increase the risk of postoperative infection, bleeding, exudation and wound/anastomoses rupture. The specific influence on the wound healing process is not known, but alcohol consumption decreases total protein but not collagen in artificial wounds. These changes are reversible after stopping alcohol intake (538).

## Infection

Infection is the most common complication that can disturb wound healing (342). Development of infection is determined by the number and type of contaminating organisms, host defense capability and local environment (540, 614).

When bacteria invade a wound, the final outcome of this event is related to the success of the initial phase of the inflammatory response in establishing an antibacterial defense (Fig. 1.27). Timing is critical and the fate of the contaminating bacteria appears to be determined within the first 3–4 hours after injury. During this period, the early inflammatory process is established and will usually result in the elimination of bacteria (539, 541, 542).

The risk of infection appears to be directly related to the number of bacteria initially present in the wound (540). If the bacteria are not eliminated during these first critical hours, a series of events will occur that affect wound healing. Thus the formation of fibroblasts will be disturbed in several ways and fibroblast proliferation is generally inhibited; but stimulation may occur in certain circumstances (541–547). Liberation of toxins, enzymes and waste products from bacteria decrease or inhibit collagen synthesis (548) and increase the synthesis of collagenase, resulting in lysis of collagen (549). Furthermore, some bacteria decrease the amount of oxygen available in the infected tissue (550, 551) whereby healing processes suffer. Collagen formation is reduced, cell migration is delayed or arrested, and cellular necrosis and microvascular thrombosis may result (552). Wounds have been classified as:

- *Clean wounds*, which are uninfected operative wounds in which no inflammation is encountered.
- *Potentially contaminated wounds*, in which the respiratory, alimentary or genitourinary tracts are entered under controlled conditions during surgery.
- *Contaminated wounds* where acute inflammation (without pus) is encountered or where there is a gross spillage from a hollow viscus during surgery.
- *Dirty wounds and infected wounds*, which are old traumatic wounds and operating wounds in the presence of pus or those involving clinical infection of perforated viscera (552).

The level of aerobic and anaerobic contamination expressed as bacteria in the wound (i.e. colony forming units per unit area) is for clean wounds 2.2, for potentially contaminated wounds $2.4 \times 10^1$, for contaminated wounds $1.1 \times 10^3$, and for dirty wounds $3.7 \times 10^3$ (553).

Oral wounds are associated with a high risk of contamination as saliva contains $10^{8-9}$ bacteria per milliliter (554).

The infective dose of bacteria that results in a microscopic infection has been found to be $10^5$ bacteria per gram of tissue (555). A correlation has been found in different types of wounds between preclosure bacterial density of aerobic and anaerobic bacteria and postsurgical wound infections (553). It is recognized that both aerobic and anaerobic organisms are implicated in most wound infections.

The use of antibiotics to supplement the natural host resistance (i.e. the early inflammatory response) has been found to be additive and sometimes even synergistic in bacteria killing. However, if antibiotics are given with more than a 3-hour delay, animal experiments have shown that the effect of antibiotics such as penicillin, erythromycin, chloramphenicol and tetracyclin was eliminated (556). The timing of antibiotics therefore seems to be of utmost importance.

Many factors are described as contributing to impaired wound healing as well as increased risk of infection. Some of these factors may have a direct influence on the healing process while other factors have an indirect influence by changing circulation and thereby oxygenation.

## Foreign bodies

The presence of foreign bodies can contribute to delayed healing, but is normally not by itself sufficient to prevent healing. Foreign bodies provide a focus for bacterial growth, and consequently a smaller amount of bacteria is needed to

cause infection in the wound area. More than 50 years ago it was observed that just a single silk suture present in the wound area increased the susceptibility to bacteria (*Staphylococcus*) by a factor of × 10 000 (557, 558). Other types of foreign materials, such as soil, clothing and drains have also been shown to increase the risk of postoperative infection and impaired wound healing (559–561). Recently, it has been found that bacteria may be camouflaged on artificial surfaces by producing an extracellular carbohydrate film (562). This film seriously affects the host response by inhibiting chemotaxis, bacterial engulfment and the oxidation response of the phagocytes.

Foreign bodies in oral and other soft tissue wounds consist mainly of soil and its contaminants (563), but tooth fragments can also be found. Soil has four major components: inorganic minerals, organic matter, water and air. The coarser components of soil are stone, gravel and sand. The smallest inorganic particle found in soil is clay. Not only does soil carry bacterial contamination into the wound, but the mere presence of inorganic and organic particles has been shown to lead to impairment of leukocyte ability to ingest and kill bacteria (559). Therefore very few bacteria are able to elicit purulent infections in the presence of foreign bodies. As there is no way of neutralizing the effect of soil, therapeutic efforts should be directed towards removing it from the wound area (see Chapter 24).

In traumatic wounds, foreign bodies can usually be removed, improving wound healing and decreasing the risk of infection. In surgical wounds, however, this may not always be the case. The most common foreign bodies in surgical wounds are sutures, drains and biological materials such as hematomas. A article emphasized that the knowledge of the foreign body reaction in tissue continuously needs to be re-established in the health care system especially in areas where implantation of foreign material is used (657).

## Sutures

The ideal suture can be described as free of infection, non-irritating to tissues, achieving its purpose and disappearing when the work is finished (435, 563). Such ideal sutures are still not available; but by choosing the best material, the complication rate provided by the suture material itself can be decreased. Bulky and braided suture materials are generally more likely to cause trouble than fine monofilament sutures (564, 565) (see Chapter 24).

In non-infected multifilamentous sutures, fibroblasts and giant cells appear early and the suture strands remain tightly bound in comparison to infected sutures where bacteria are entrapped within the braids, leading to pus formation (566).

The reaction around a monofilament suture is minimal and a fibrous capsule appears after 10 days, even in the presence of infection. Apart from the knots, there is no space for bacteria to lodge (559). The ideal suture is therefore a monofilament type of suture with sufficient strength to hold the wound edges together until significant healing has occurred, even in delayed healing. The use of absorbable and non-absorbable sutures in relation to wound healing and infection is still controversial (564–569). The use of sutures in soft tissue wounds is further discussed in Chapter 21.

## Distant wound response

For decades it has been known that a wound preceded by a previous injury heals faster than a primary wound. Thus from a mechanical point of view (wound strength), a second wound heals faster than the first (570). The explanation for this phenomenon is still uncertain. Another distant wound response is found when two wounds occur simultaneously in distant parts of the body. In these cases impaired blood circulation may be found in the wounds leading to impaired healing (571).

## Age

### *Fetal wound healing*

Healing of experimental oral wounds in a mammalian fetus differs greatly from the healing of similar wounds in adults (572–576). Thus accelerated healing without scarring is found in fetal wounds, even in defect wounds. The wound response appears to be without acute inflammation and with minimal fibroblast and endothelial cell proliferation (577). Furthermore the ECM appears collagen poor, and rich in hyaluronic acid.

Hyaluronic acid is laid down early in the matrix of both fetal and adult wounds, but sustained deposition of hyaluronic acid is unique to fetal wound healing (578). Hyaluronic acid is presently thought to play a decisive role in the regenerative process, as it provides a permissive environment for cell proliferation and mobility (572–579, 658, 659) and suppresses macrophage-effected postnatal repair (579). It has been shown that the elimination of TGF-β from healing wounds in adult rats reversed the typical fibrous scar formation to a stage of fetal wound healing without scar tissue (580). This implies that the control of selected cytokines may be a future approach to control scarring.

It is known that unique properties of fetal cells, ECM, cytokine profile and gene expression contribute to this scarless repair. However, the precise mechanism of fetal regeneration remains unclear with a number of differences identified between fetal and adult wound healing (651, 653). A number of potential antiscarring therapeutics have evolved from understanding fetal regeneration though to date none have completely prevented scar formation.

### *Adult wound healing*

The relationship between age and healing of skin wounds in respect to speed of healing has been studied experimentally in rats (581–588), rabbits (589, 590) and in humans (591, 592). Experiments examining the role of the age factor upon wounding of oral mucosa and gingiva have been performed in rats (593–597), mice (598) and humans (599–601). In the most comprehensive studies using experimental skin wounds in rats and gingival wounds in humans, it was found that healing of gingival defects is slower and regeneration is more incomplete in old than in young individuals. Furthermore,

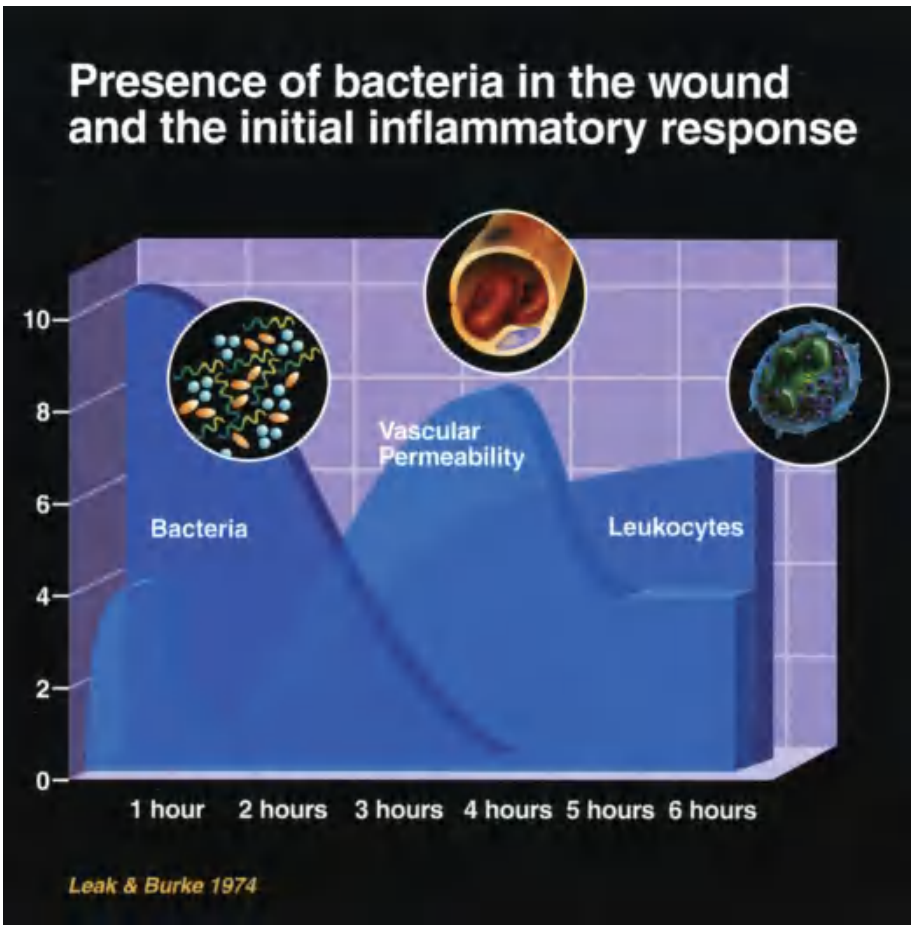


**Fig. 1.27** Bacteria and the initial inflammatory response. The period of active tissue antibacterial activity (the decisive period) relates well to the establishing of the inflammatory response, as reflected by increased vascular permeability and leukocyte migration into the wound site. From (539).

wound strength develops more slowly in old rats than in young rats, a finding which was related to the better functional arrangement of collagen fibers in the young animals (602). In the gastrointestinal tract, however, aging seems not to have an adverse influence on wound healing (603). Surveys of the current literature conclude that wound healing in the skin of healthy elderly people is delayed, but scar maturation is improved in comparison with young individuals. Impaired wound healing leading to scar formation is primarily associated with comorbidities, which are more prevalent in old age (615, 616). Nevertheless, age (>60 years) is an independent risk factor for less frequent closure of wounds (661).

Wound infection is also strongly related to age (604, 605). Thus, in a prospective study on wound infection subsequent to surgical wounds, a wound infection rate of 0.6% was found in children aged 1–14 years and this rate rose to a maximum of 3.8% in patients over 66 years of age (604).

## Optimizing oral wound healing

An important principle to consider in this context is that the mode of action of both normal wound healing and the response to infection seems to follow a general pattern which is sometimes in conflict with the regeneration of injured organs. This is apparent in skin wounds where the need for rapid wound closure (in order to prevent infection from invading microorganisms) usually results in the formation of a scar (613). In the dental organ, an effective response against bacteria takes priority with activation of the neutrophils, lymphocytes, macrophages and osteoclasts leading to frequent bone and tooth loss due to hard tissue resorption (see Chapter 2).

Presently, the most likely avenue whereby healing problems can be avoided appears to be careful tissue handling whereby tissue perfusion is re-established or stabilized and wound contaminants (e.g. foreign bodies and microbes) are reduced or eliminated.

To achieve this goal, various steps are necessary in the diffent phases of wound healing. In the coagulation phase, assistance in achieving hemostasis may be necessary. In performing this, it is important not to use excessive cautery, which results in tissue necrosis, or topical hemostatic agents (e.g. Surgicel®, Oxycel®, Gelfoam®) that may have a potentiating effect for infection (559). Instead, firm pressure exerted with a gauze sponge for several minutes usually results in hemostasis.

In handling oral wounds, a local anesthetic is usually necessary. In this regard it should be borne in mind that the vasoconstriction of the anesthetic solution increases the risk of infection of the wound due to interference with the inflammatory response in the critical first hours after injury (559, 606). Regional block anesthesia rather than local infiltration of the anesthetic solution is therefore to be recommended.

Wound debridement should be limited to the removal of foreign bodies and obviously damaged tissue that is not expected to survive or become revascularized (see Chapter 24).

The elimination and/or reduction in size of the blood clot should be attempted in order to facilitate wound healing, including revascularization. This applies to soft tissues as well as tooth and bone repositioning.

The value of complete immobilization of the wound edges is presently under debate so only a few treatment principles can be suggested.

In soft tissue wounds any sutures used to immobilize the wound edges must be regarded as foreign bodies which increases the risk of infection (276, 559, 652). Thus a minimal number of sutures should be used, and a suture type should be chosen which elicits minimal side effects (see Chapter 24).

In regard to hard tissue healing, splinting should generally be performed. These splints should not augment the risk of infection, whereby the application and the design of the splints becomes crucial (see Chapter 39).

The value of antibiotics in oral wound healing is presently unsettled (see Chapters 3 and 24). If indicated, antibiotics should be administered as early as possible and preferably not later than the first 3–4 hours after trauma and should only be maintained for a short period of time (540) (see Chapters 3, 21 and 24). Acceleration of oral wound healing by the use of growth factors is in its initial stages but has the potential to be an essential part of trauma treatment (see Chapter 2). A fascinating perspective of the use of growth factors is the achievement of an orchestrated healing response whereby certain parts of the cellular response are promoted (e.g. angiogenesis, fibrillogenesis, dentinogenesis, osteogenesis and epithelialization). The initial attempts at such an approach to oral wound healing appear very promising (see Chapter 24).

## Essentials

*Regeneration* is a process whereby the original architecture and function of disrupted or lost tissue is completely restored.

*Repair* is a process whereby the continuity of disrupted or lost tissue is restored by new tissue, which does not reproduce the original structure and function.

### General steps in wound healing

- Control of bleeding by the combined action of vasoconstriction and coagulation
- Inflammatory response, whereby leukocytes migrate into the wound in order to protect the area against infection and perform cleansing of the wound site
- Connective and epithelial tissue migration and proliferation, which obturate the wound defect and add mechanical strength to the wound
- Reorganization of the tissue by a remodeling process that results in more functionally oriented collagen fibers which increase the strength of the wound

### Main roles of the individual tissue cells

- *Platelets*, apart from their role in initial hemostasis and their activation of the coagulation cascade, serve as initiators of the wound healing process
- *Polymorphonuclear leukocytes* prevent bacterial infection within the wound site
- *Macrophages* are scavengers of tissue remnants and foreign bodies including bacteria and are the key cells in coordinating the cellular events in wound healing
- *Fibroblasts* produce collagen and ground substance which fills out the wound defect and adds mechanical strength to the wound
- *Endothelial cells* in the venules are the key cells in angiogenesis. By coordinated endothelial cell proliferation and migration, a new vascular network is formed at the wound site
- *Pericytes* represent a pool of undifferentiated mesenchymal cells
- *Epithelial cells* close the gap against the external environment by cell migration and proliferation

The coordinated action of the cells mentioned here is found in the *wound healing module* created a few days after injury where leading macrophages clear damaged tissue, foreign bodies and bacteria with trailing fibroblasts and newly formed capillaries.

Significant stimuli for the invasive growth of newly formed connective tissue and also termination of the wound healing module appear to be *growth factors* as well as *oxygen tension* and *lactate concentration* in the injury zone ahead of the wound healing module.

Of the many factors known to disturb wound healing, the following are the most likely candidates affecting mucosa and skin, as well as the periodontium and the pulp of traumatized teeth:

- *Low oxygen* delivery to the wound site due to the initial trauma and/or improper tissue handling technique (e.g. suturing, splinting or lack of repositioning of tissues)
- *Infection* due to contamination of the injury site
- *Foreign bodies* including inappropriate use of sutures and drains

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# 2

# Response of Oral Tissues to Trauma

J. O. Andreasen, H. Løvschall & D. Ricucci

## Repair and regeneration of oral tissues

An injury can be defined as an interruption in the continuity of tissues, and healing as the re-establishment of that continuity. The result of this process can either be *tissue repair*, where the continuity is restored but the healed tissue differs in anatomy and function, or *tissue regeneration*, where both anatomy and function are restored.

In lower vertebrates, the regeneration of appendages such as limbs, tails and fins is common. In mammals, this healing capacity has generally been lost, although it has been reported that, in humans, digits amputated distal to the distal interphalangeal joint can regenerate in children (1). The explanation for this general loss of healing capacity in mammals is presently under investigation. However, the rapid epidermal healing response in mammals, which optimizes wound closure in the skin and thereby limits the risk of infection, is currently believed to present an obstacle to tissue regeneration (2).

Healing of most wounds in humans, whether caused by trauma or surgery, includes repair with more or less fibrous scar tissue formation which subsequently leads to problems in the functioning of the particular organ affected. In the oral region, skin wounds and to a lesser degree the oral mucosa, are repaired with scar formation (see Chapter 24).

Dental tissues are unique in comparison to most other tissues in the body due to their marked capacity for regeneration. Thus tooth germs split by trauma or surgery may to a certain extent regenerate and the same applies to injured dentin, cementum, bone and gingiva (see later).

Injuries to the pulp and the periodontal ligament (PDL) may sometimes regenerate or alternatively show repair with fibrous scar tissue or bone.

Understanding the circumstances leading to repair and regeneration in oral tissues has been a formidable challenge (3). In this regard, wounding releases a variety of signals that induce neighboring cell populations to respond by proliferation, migration or differentiation.

The first prerequisite for tissue regeneration is that a tissue-specific cell population is present after wounding (e.g. pulp or PDL progenitor cells). If these cells are not present, repair rather than regeneration will take place. A typical example of this concept is the healing by ankylosis of teeth in which the PDL has been injured (see p. 85).

A second prerequisite for regeneration is that conditions exist that are conducive to migration of tissue-specific cells into the wound site. Thus, incomplete repositioning of a luxated tooth may lead to damage to the epithelial root sheath, thereby forcing ingrowth of PDL-derived cells and bone into the pulp canal. Another situation in which the topographic conditions of the wound may determine whether repair or regeneration take place is related to loss of periodontal attachment on a root or an implant. In this situation the insertion of a membrane may guide bone cells and/or PDL into the wound site (see Chapter 35).

A third factor that may determine tissue repair or regeneration in oral wounds is the presence of contaminating foreign bodies and/or bacteria. Inflammation related to a contaminated wound has been found to lead to repair rather than regeneration, possibly because contamination leads to formation of a non-tissue-specific, inflamed granulation tissue at the expense of the proliferation and migration of tissue-specific cells.

When assessing wound healing after trauma, it is not enough to understand the healing capacity of individual cell types; one must also consider the various tissue compartments, each consisting of different cellular systems. Differences in healing capacity and rates of healing can lead to competitive situations and thereby to variations in wound healing. Ischemia or the total destruction of cell layers may occur (2) (Fig. 2.1).

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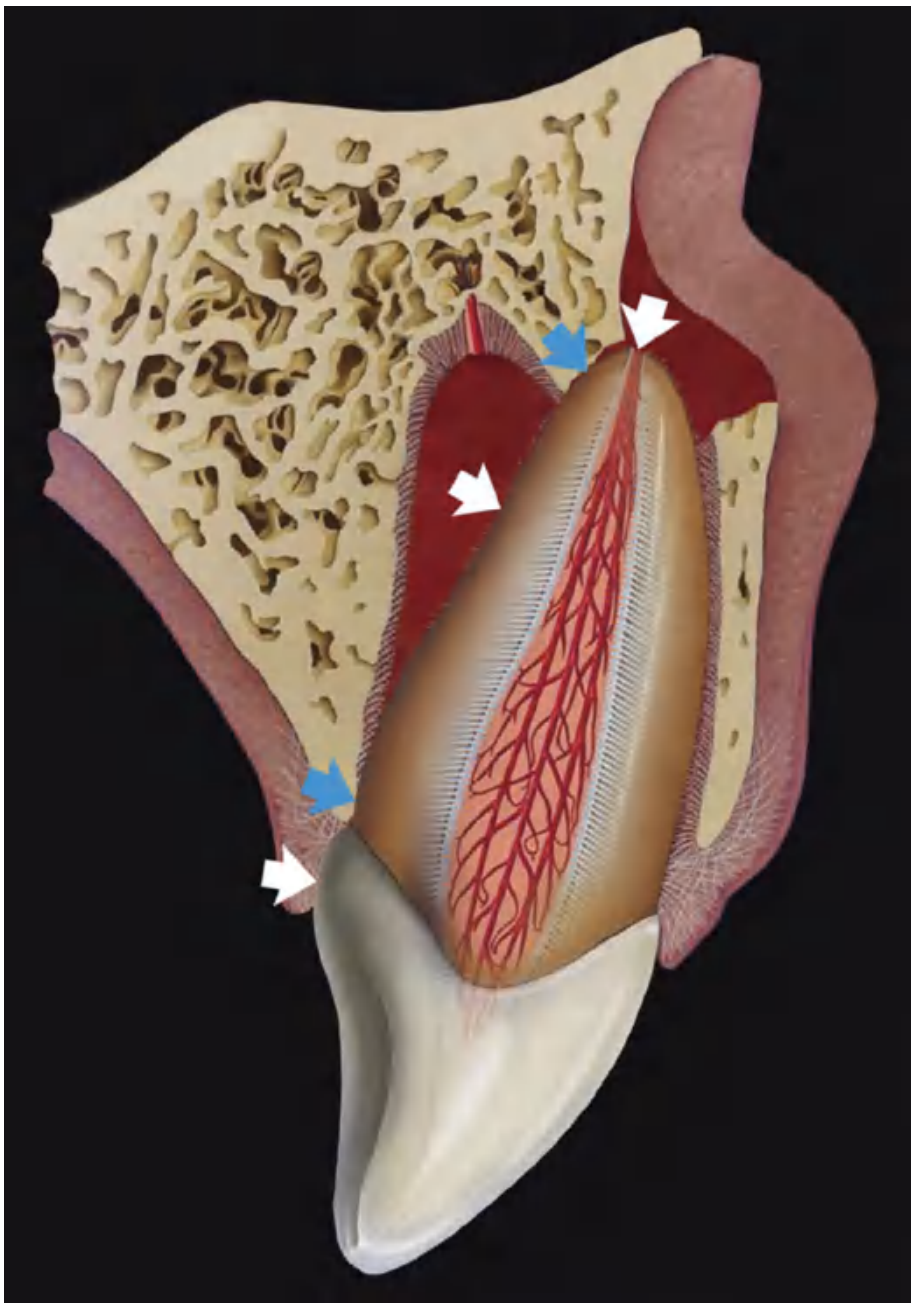


**Fig. 2.1** Injury zones after tooth luxation. A lateral luxation implies trauma to multiple cell systems in the periodontium and the pulp, as rupture (white arrows) or compression (blue arrows) or ischemia damage to these cellular compartments. The outcome of the healing processes is entirely dependent upon the healing capacity of the different cellular systems involved. From (3).

This chapter will present a brief description of the anatomy and function of cell compartments typically involved following a traumatic event. In the description of these compartments, anatomic borders have been chosen that are typically the result of separation lines or contusion locations subsequent to trauma. Using this approach, the following anatomic zones evolve in relation to teeth with completed root development: *gingival and periosteal complex*; *cementoperiodontal ligament complex*; *alveolar bone complex*; and *dentinopulpal complex*. In developing teeth, the following structures should be added: *dental follicle*; *enamel-forming organ*; and *Hertwig's epithelial root sheath*.

For each tissue compartment, a description will be given of its anatomy and healing responses to trauma and infection.

## Developing teeth

### Dental follicle

The dental follicle (dental sac) has traditionally been considered the formative organ of the periodontium (Fig. 2.2). This concept has been supported by experimental studies of transplanted tooth germs that show that the innermost layer (dental follicle proper, which is in contact with the tooth germ) give rise to all of the components of the periodontium (4–6). Based on these findings, it has been suggested that the term dental follicle should be reserved for the innermost layer of connective tissue separating the tooth germ from its crypt (7). The remaining peripheral tissue would therefore

be designated as the perifollicular mesenchyme (5, 8). However, until further research has definitely ruled out the possibility of perifollicular mesenchymal participation in the formation of the periodontal attachment, it appears justified to use the term dental follicle for all the mesenchymal tissue interposed between the tooth germ and alveolar bone.

In addition to the role of the dental follicle in the formation of cementum and PDL fibers, it also has a significant osteogenic capacity. Thus, in several experiments it has been shown that heterotopic transplantation of tooth germs, including their follicles, to soft tissue sites results in formation of a complete periodontium including cementum, PDL fibers and an adjacent shell of bone (alveolar bone proper) (3, 5, 8–15).

A number of events have been suggested to be responsible for tooth eruption, including root growth, dentin formation, pulp growth and changes in the dental follicle and the periodontal ligament (16–23). Recent experiments in dogs and monkeys have demonstrated that changes in the dental follicle (including the reduced enamel epithelium) are possibly responsible for the coordinated enlargement of the eruption pathway and movement of the tooth germ along this pathway in the initial phases of eruption (23–26). These findings, together with the established relationship between the dental follicle and distinct areas of bone resorption and bone formation, suggest that the dental follicle and/or the reduced enamel epithelium coordinate these processes during eruption (see Chapter 23) (21–30) (Fig. 2.3). Consequently, severe disturbances in eruption can be anticipated if there is damage to the follicle due to trauma or infection (see also Chapters 22 and 23).

### *Response to trauma*

Histologic studies have revealed a very close relationship between the primary teeth and the permanent dentition, especially in the initial phases of development (31, 32) (Figs 2.2 and 2.3). Traumatic injuries can therefore easily be transmitted from the primary to the permanent dentition (34).

While there is abundant clinical evidence to show that the dental follicle has a remarkable healing capacity after injury, certain limitations do exist. Thus, it has been shown that, when larger parts of the dental follicle are removed, an ankylosis is formed between the tooth surface and the crypt and eruption is arrested (23, 25) (Fig. 2.4). The extent to which a follicle can be damaged without leading to this complication is not known.

### *Response to infection*

Very little information exists on the reaction of the dental follicle to infection. In monkeys, it was found that follicles of permanent incisor tooth germs were resistant to short-term chronic periapical infection (i.e. 6 months' exposure) originating from the root canals of primary incisors (Fig. 2.5), and no influence could be demonstrated on the permanent successors with respect to enamel mineralization (33). In rare instances, acute infection has been found to spread to the entire follicle and to lead to sequestration of the tooth germ. Such events have been reported after traumatic injuries to the primary dentition, in jaw fractures where the line of fracture involves a tooth germ, or in cases of osteomyelitis that affect the bony regions containing dental follicles (34). Apart from these rare occurrences, the dental follicle appears to be rather resistant to infection.

## Enamel-forming organ

The formation and maturation of enamel in the permanent dentition is often disturbed or arrested by trauma transmitted from primary teeth after displacement and/or periapical inflammation resulting from infection. The outcome of such events depends primarily upon the stage of enamel formation at the time of the injury.

The usual stage at which primary tooth injuries interfere with odontogenesis of the permanent dentition is the beginning of mineralization of the incisal portion of the crown. At this stage of development, the enamel-forming organ consists of the cervical loop placed apically to the site of active enamel and dentin formation. Coronally, the enamel epithelium is divided into the inner and outer enamel epithelium with an intervening stratum intermedium and stellate reticulum between them. The reduced enamel epithelium is found more coronally, where the full enamel thickness has been formed and mineralization completed. In the following, a synopsis of the anatomy and function of these structures will be presented, as well their response to trauma and infection.

## Cervical loop

At the free border of the enamel organ, the inner and outer enamel epithelial layers are continuous and form the cervical loop (35, 36). Progression of tooth development is entirely dependent upon the growth and action of this structure.

### *Response to trauma*

The cervical loop is highly resistant to trauma. Simple separation of the cervical enamel and dentin matrix does not prevent further enamel or dentin formation (37, 38) (Figs 2.6 and 2.7). However, profound contusion of this structure, as after intrusion of a primary incisor into the developing successor, may result in total arrest of further odontogenesis (90).

### *Response to infection*

The response of the cervical loop to infection has not yet been studied.

## Inner enamel epithelium

According to function, the inner enamel epithelium (ameloblasts) evolves through a number of functional stages (39–41) (Fig. 2.8). The first is the *morphogenetic stage* whereby the future outline of the crown is determined. This stage is

**Fig. 2.2** Anatomy of a dental follicle, showing a dental follicle of a monkey maxillary central tooth germ. Note the close proximity between the primary tooth and the permanent successor as well as the loose structure of the follicle. ×12.

**Fig. 2.3** Eruption of a permanent maxillary central monkey incisor. Note bone apposition apically, while active bone and tooth resorption takes place coronally. ×12.

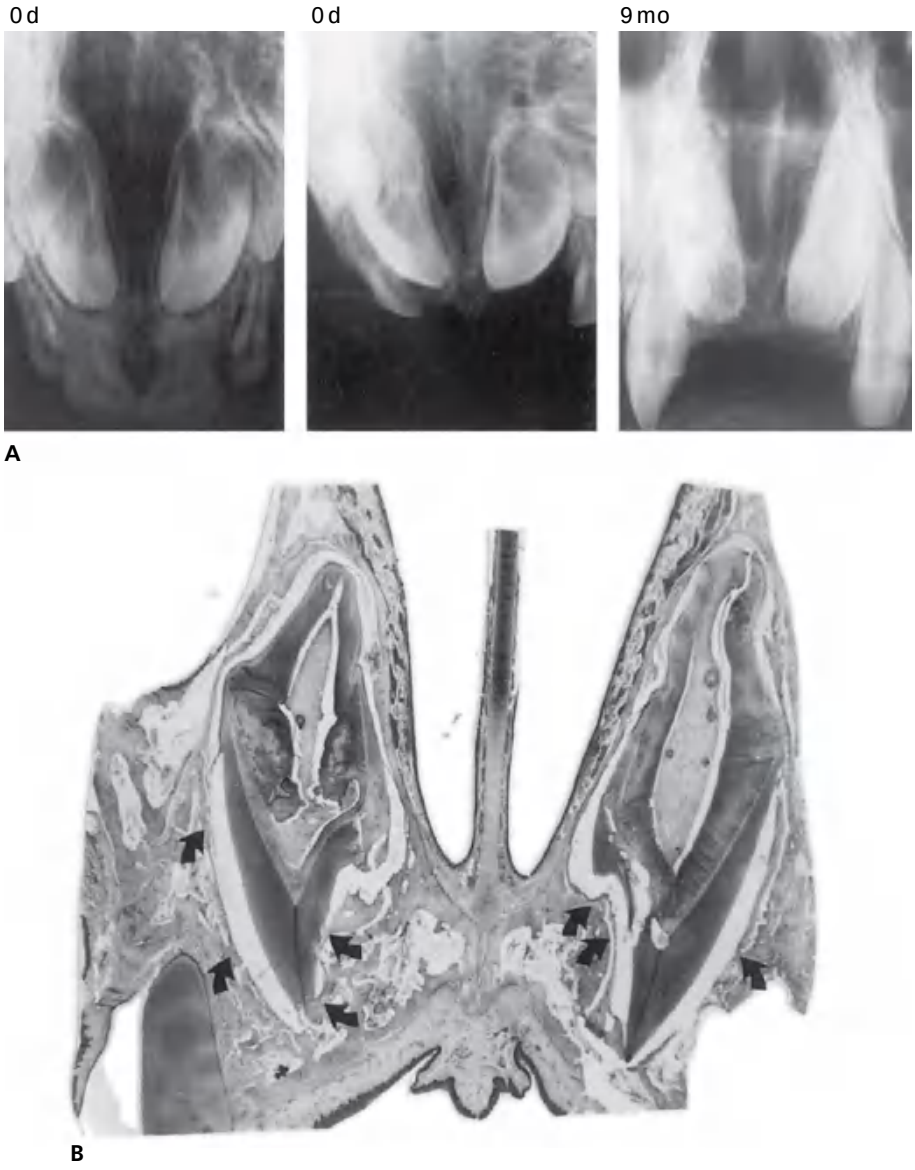



**Fig. 2.4** Replantation of maxillary monkey tooth germs with damaged follicles. A. Preoperative and postoperative radiographs and the condition after 9 months. No eruption has taken place. B. Low power view of both central incisors. Note the lack of eruption despite almost complete root formation. Furthermore note the ankylosis sites affecting the crowns of both teeth (arrows). Reproduced from (25) with permission.

followed by an *organizing stage*, with initiation of dentin formation. The *formative stage* is then reached, where enamel matrix formation as well as initial mineralization take place. The *maturation stage* follows enamel matrix formation. During this stage, there is partial removal of the organic enamel matrix accompanied by a complex mineralization process proceeding from the region immediately adjacent to the dentin and progressing outward (42). These are followed by the *protective* and *desmolytic stages*, which will be described later in this chapter. During these two stages, the tissue layer is now designated the *reduced enamel epithelium*.

### *Response to trauma*

To date, only limited knowledge exists concerning the response of the inner enamel epithelium to trauma (37, 38, 43–46). In the case of total loss of ameloblasts in the secretory phase, no regenerative potential exists (44). In the case of partial damage, the ameloblasts in the *secretory* stage may survive and continue enamel matrix formation, and later maturation may occur (42, 45–47) (Figs 2.6 and 2.7).

If there is total loss of the ameloblasts during the *maturation* stage, a hypomineralized area of enamel will develop. If partial damage occurs, the ameloblasts may recover and the result may be only a limited zone of hypomineralization (42).

### *Response to infection*

When chronic periapical inflammation develops due to necrosis of an infected pulp in primary teeth, the effect upon the permanent successors is very limited, at least over a short time (i.e. months) (33) (Fig. 2.5). When infection persists over longer periods (i.e. years), experimental and clinical studies have shown that enamel formation and maturation may be affected (48–50).

The response to acute infection is similar to the response to trauma and may lead to localized arrest of enamel formation (51–58). At the site of injury, a cementum-like tissue may later be deposited (59, 60). In this situation, the cementoblasts are most probably recruited from the follicle, which has been shown to have cementogenic potential (61).

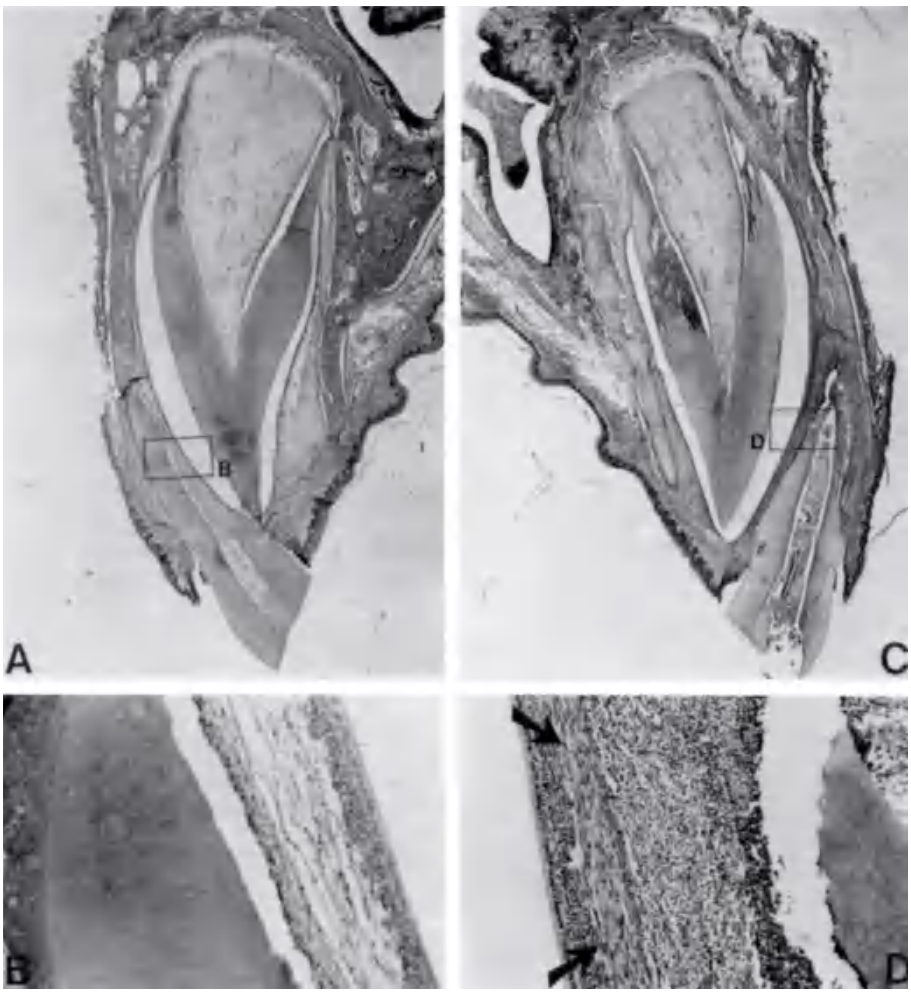



**Fig. 2.5 Follicular changes after pulp necrosis of a maxillary monkey central primary incisor**
Late stages of tooth development of permanent tooth germs. Low power view of control tooth (A) and experimental tooth (C). ×7.

**Changes in the follicle**
B. Normal reduced enamel epithelium in control tooth. In the experimental tooth (D) there is intense periapical inflammation demarcated from reduced enamel epithelium by a thin layer of fibrous tissue (arrows). ×75. Reproduced from (33) with permission.

## Reduced enamel epithelium

When enamel matrix formation is complete and enamel maturation has begun, the ameloblasts become compact and the stellate reticulum disappears, and a multilayered epithelium is formed of cuboidal or flattened cells. The following functions are currently linked to the reduced enamel epithelium: protection of the enamel against the follicle until the tooth erupts (62); regulation of osteoclastic activity in the follicle preparatory to eruption (62); and participation in the breakdown of the connective tissue overlying the crown (63–66). In addition, the reduced enamel epithelium may play an active part in fusion with the oral epithelium as the tooth emerges into the oral cavity (64).

### *Response to trauma*

Minor injury to the reduced enamel epithelium is repaired with a thin squamous epithelium (37) (Fig. 2.9). The influence of this event upon eruption is presently unknown, but with larger areas of destruction of the reduced enamel epithelium, ankylosis and tooth retention have been demonstrated (67) (Fig. 2.4).

### *Response to infection*

The short-term (i.e. months) effect of chronic inflammation on primary teeth appears to be negligible (33) (Fig. 2.5), while the long-term effect appears to be ectopic or accelerated eruption (50).

## Enamel and enamel matrix

Mature enamel has the highest mineral content of any tissue in the body (i.e. 96–98%), whereas enamel matrix is considerably less mineralized. Thus, if maturation is interrupted due to infection or trauma, enamel hypomineralization will result. One may see problems clinically with increased caries or perhaps with difficulties regarding acid-etch restorative techniques. This problem is further discussed in Chapter 23.

### *Response to trauma*

Trauma to a primary tooth may cause contusion of the permanent enamel matrix (37, 38). The ameloblasts will also be destroyed, thereby arresting enamel maturation and resulting in a permanent hypomineralized enamel defect (see Chapter 23). In mature enamel, the result of a direct impact will be enamel infraction, i.e. a split along the enamel rods usually ending at the dentino-enamel junction (see Chapter 13), or a fracture, whereby part of the crown is lost.

### *Response to infection*

The response of enamel matrix or enamel to infection has been discussed earlier.

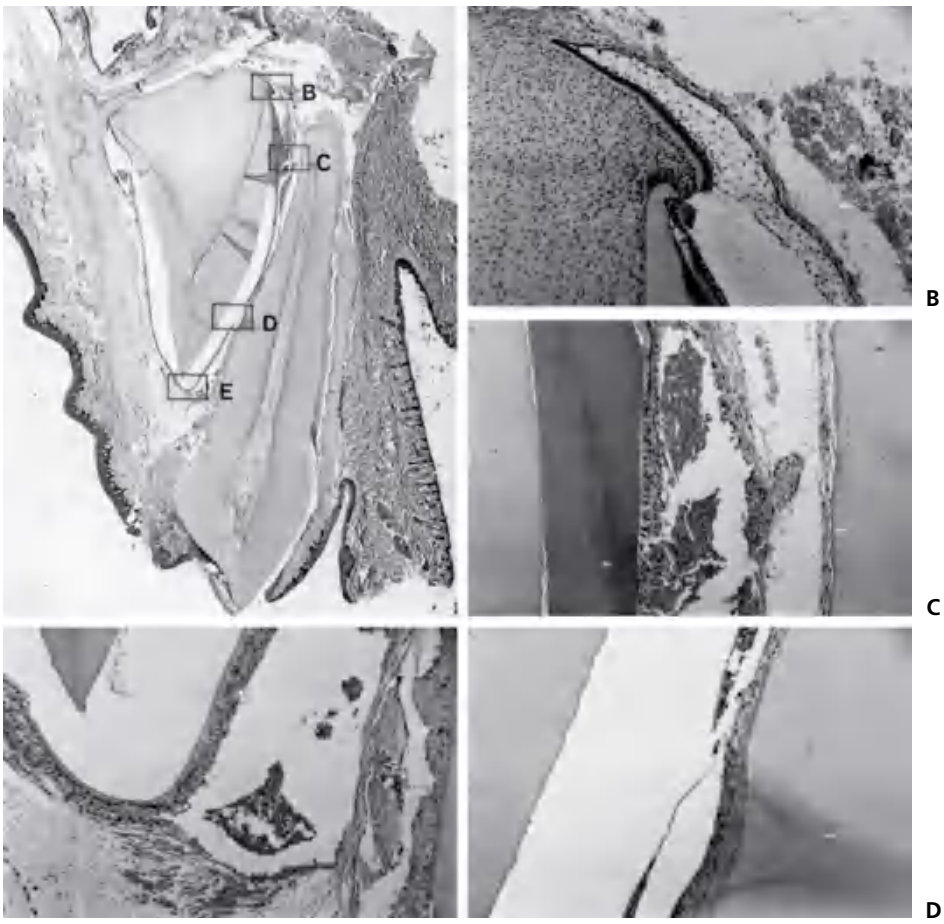



**Fig. 2.6** Immediate changes after intrusion of a primary monkey incisor. A. Low power view of specimen. ×8. B. Dislocation between mineralized tissues and cervical loop. ×75. C. Rupture and bleeding in stellate reticulum. ×75. D. Destruction of reduced enamel epithelium. ×75. E. Separation of reduced enamel epithelium from connective tissue. ×75. Reproduced from (37) with permission.

## Hertwig's epithelial root sheath

Hertwig's epithelial root sheath (HERS) is a continuous sleeve of epithelial cells that separates the pulp from the dental follicle. In all primates it consists of an inner layer of cuboidal cells and an outer layer of more flattened cells (68) (Fig. 2.10). Occasionally, there is an intermediate layer of elongated cells (69).

The HERS completely encloses the dental papilla except for an opening in its base, the *primary apical foramen*, through which the pulp receives its neurovascular supply. Root formation is determined by the activity of HERS (70–74), and root growth is dependent upon a continuous proliferation of epithelium (75, 76). More incisally, dentin and cementum develop synchronously, ensuring a relatively constant sheath length throughout root formation up to the final phases of root development, when the root sheath becomes considerably shorter.

The width of the primary apical foramen as well as the number of vessels entering it appears to be relatively constant until final root length has been achieved (74). Thus, in a luxation, replantation or transplantation situation, the chances of revascularization through the primary apical foramen should theoretically be the same throughout the early stages of root development, until apical constriction starts. Such a relationship has in fact been found for luxated (605) and replanted incisors (606) as well as for autotransplanted human premolars (77).

In the following, a closer look will be taken at the role of HERS in *dentin* and *cementum formation*. Odontoblastic differentiation takes place adjacent to the basal aspect of HERS, after which the first dentin matrix is deposited (*mantle dentin*). At this time, the innermost layer of cells in the root sheath secretes a material that combines with the mantle dentin to form the so-called *intermediate cementum layer*. This layer has been found to contain enamel matrix protein and later becomes hypercalcified (78–80). The intermediate cementum layer has attracted interest (81) and has been found to be an effective barrier against the penetration of noxious elements placed in the root canal to the periodontal ligament via the dentinal tubules and possibly also to toxins produced by bacteria within the root canal (82).

After odontoblast induction and formation of intermediate cementum, the root sheath degenerates and the epithelial cells migrate away from the root surface to form the epithelial rests of Mallassez (Fig. 2.11). At the same time, cells from the periodontal ligament migrate towards the root surface and become cementoblasts. In an extensive study of the role of HERS it is suggested that acellular and cellular cementum are synthesized by two different types of cells –first by HERS-derived cementoblasts forming acellular cementum and later by dental follicle-derived cementoblasts forming cellular cementum (618). These cells then synthesize collagen and other organic constituents of cementum. The role of the epithelial root sheath in the induction of cementoblasts, however, has been questioned (75, 83–85).

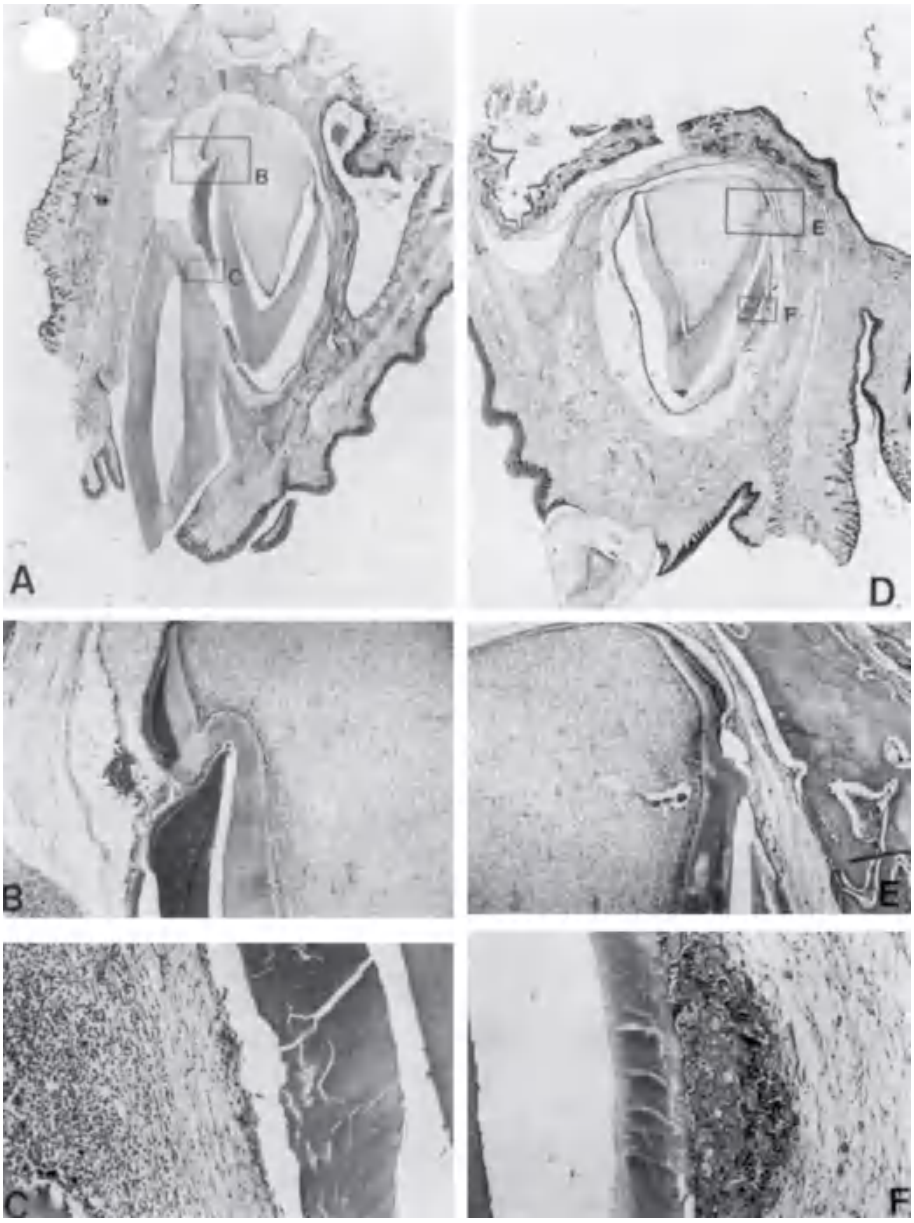



**Fig. 2.7 Late changes after intrusion of primary incisors in a monkey**
Low power view of a specimen where the intruded tooth is preserved (A), and the contralateral side where the intruded tooth was extracted (D). ×8.

**Changes in enamel and dentin**
B. Morphologic changes in enamel matrix and dentin. C. Destruction of enamel epithelium and abnormal matrix formation. E. Partial arrest of enamel matrix formation. ×30. F. Metaplasia of enamel epithelium and abnormal matrix formation. ×75. Reproduced from (37) with permission.

### *Response to trauma*

*Chronic trauma* to the HERS, such as orthodontic intrusion of immature teeth, often leads to its fragmentation. In these cases the epithelial fragments displaced into the pulp canal can induce true denticle formation (i.e. dentin-containing pulp stones) (86–90).

An injury close to the root sheath (e.g. a laceration in the PDL) may result in temporary hyperactivity of the root sheath, which can initiate rapid production of both dentin and cementum in the apical area (Fig. 2.12). However, activity of the root sheath will later return to normal.

*Acute trauma* to the epithelial root sheath is transmitted indirectly, for example by the intrusion of a primary tooth, or directly by forceful displacement of immature permanent teeth. This can damage the HERS and lead to partial or complete arrest of root development (91–94) (Fig. 2.13).

During replantation, the root sheath may be injured either during avulsion or during extraoral storage, or by the repositioning procedure itself. Following such injury, further root growth will be partially or totally arrested, and bone and PDL-derived tissue from the base of the socket may invade the root canal to form intraradicular bone which is separated from the canal wall by an *internal periodontal ligament* (see Chapters 13 and 17) (95) (Fig. 2.14). A similar bony invasion into the pulp of immature teeth is found when the epithelial root sheath is resected *in situ* (96, 97), or when the root sheath is injured by trauma (97) or chemically, as with devitalization procedures that employ formaldehyde (98–100).

### *Response to infection*

Hertwig's epithelial root sheath is rather resistant to inflammation in connection with partial pulp necrosis. Although

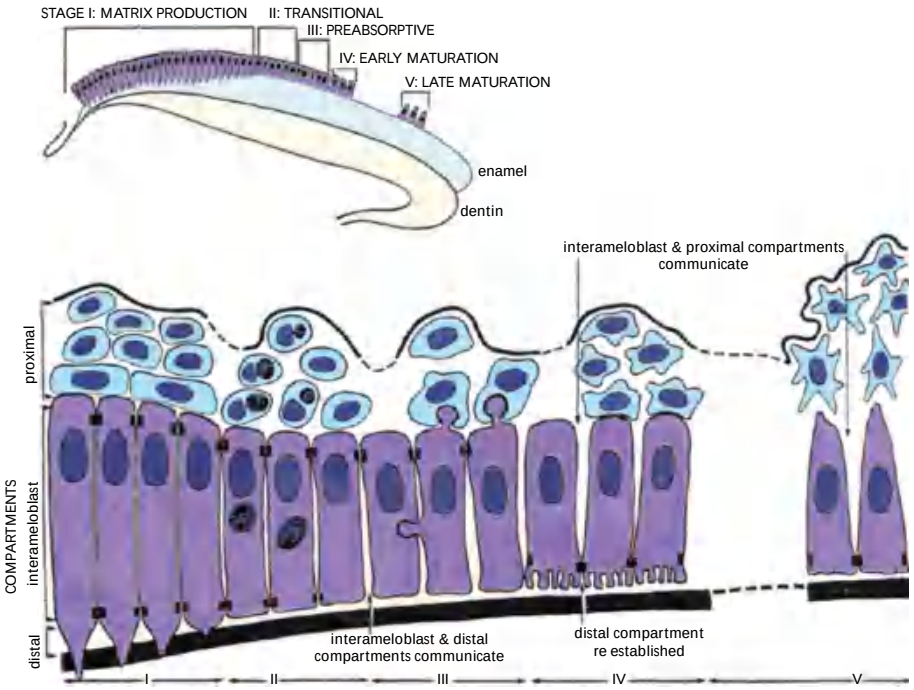



**Fig. 2.8** Ameloblasts and matrix production. Changes in the enamel epithelial compartment related to enamel matrix production. Reproduced from (39) with permission.

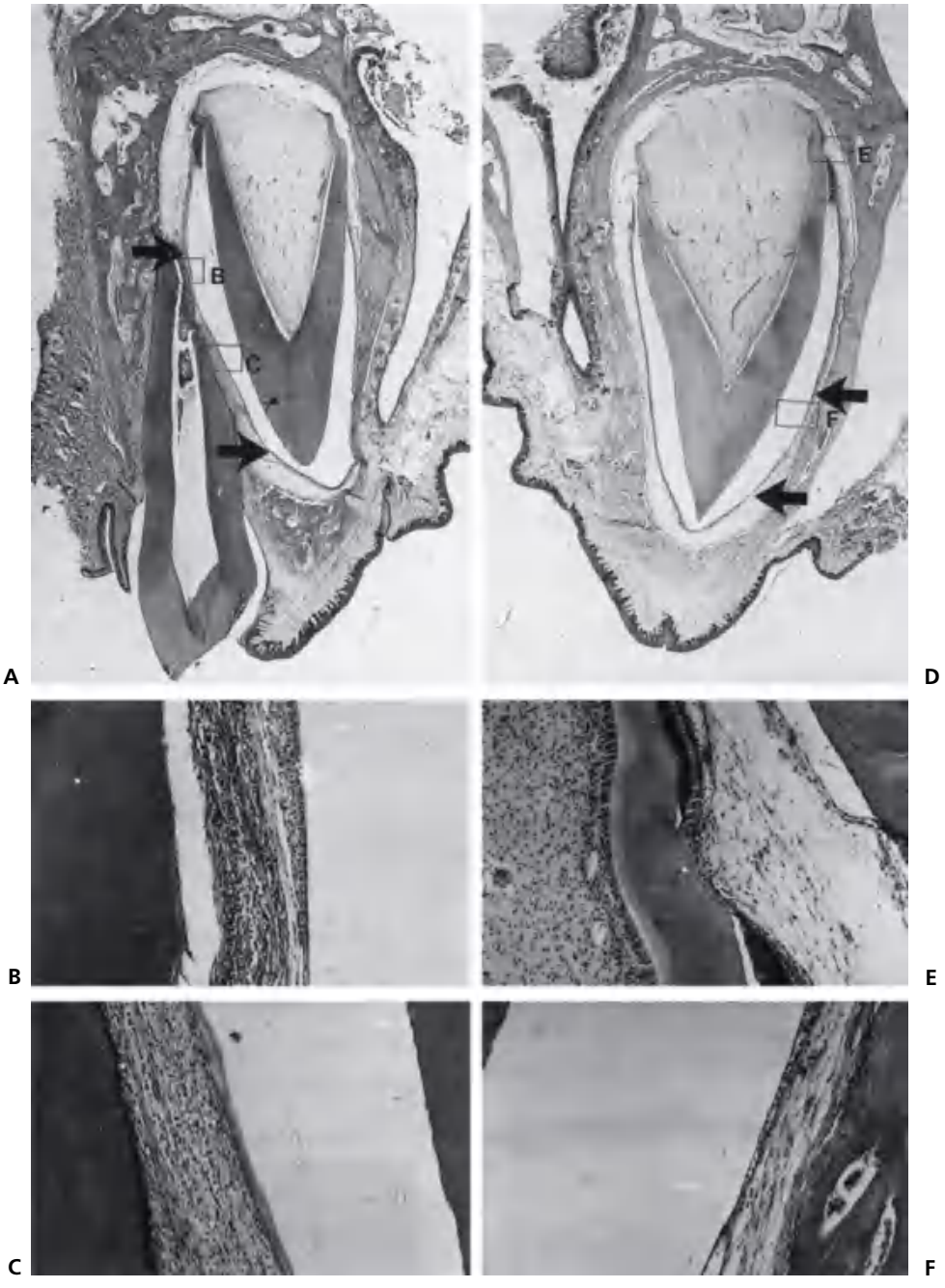



**Fig. 2.9 Late changes after intrusion in a monkey**
Low power view of a specimen where the intruded primary tooth was preserved (A), and the contralateral side where the intruded tooth was extracted (D). Arrows indicate length of disturbed reduced enamel epithelium. ×8.

**Changes in follicle and hard tissues**
B. Periapical inflammation and disturbed enamel epithelium. C. Amorphous eosinophilic substance deposited on enamel. E. Area with temporary arrest of enamel matrix formation. ×75. F. Metaplastic reduced enamel epithelium. Reproduced from (37) with permission.

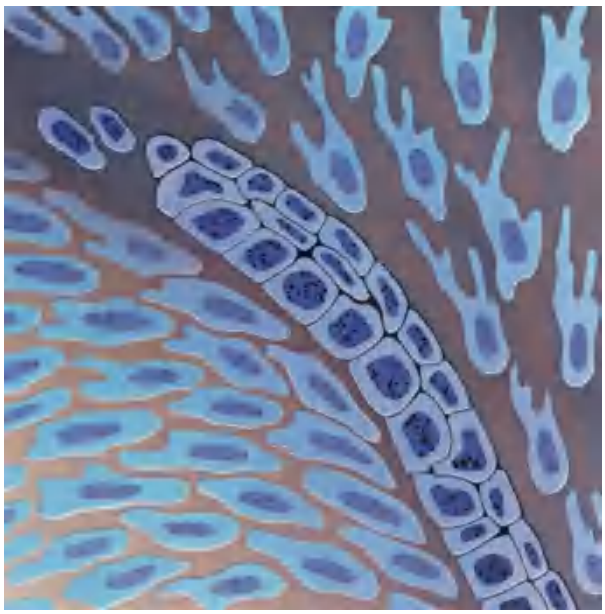


**Fig. 2.10** Hertwig's epithelial root sheath. The root sheath consists of two to three layers of epithelial cells.

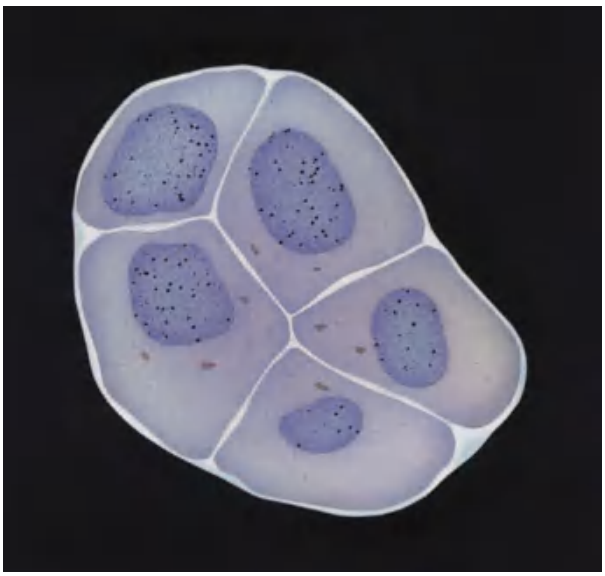
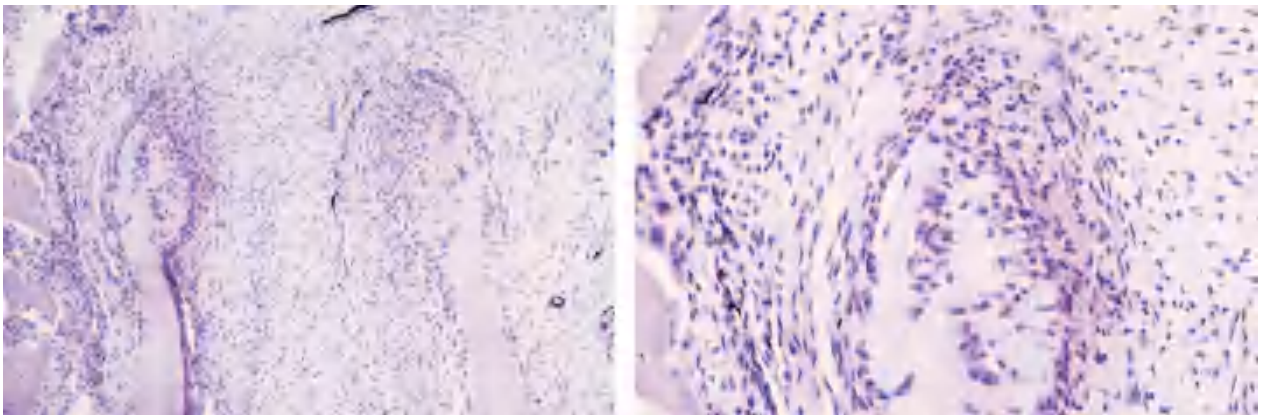
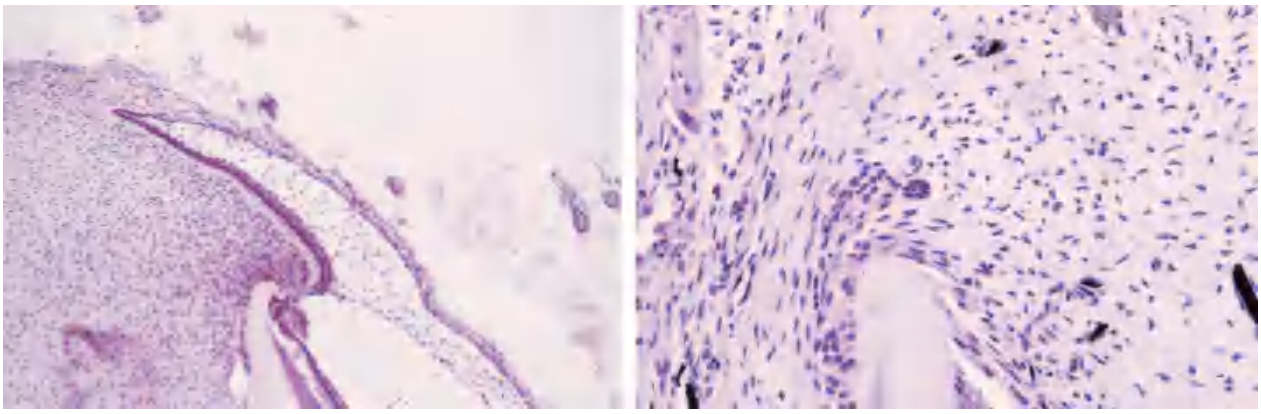


**Fig. 2.11** Epithelial island of Mallassez.



**A** **B**

**Fig. 2.12** Hyperactivity of HERS. Note excessive production of dentin after replantation of a permanent maxillary lateral incisor. A. ×25. B. ×75. Observation period: 1 week.



**A** **B**

**Fig. 2.13** Damage to the cervical diaphragm or the HERS subsequent to replantation of permanent lateral monkey incisors. A. Displacement of cervical diaphragm. ×25. B. Fragmentation of the root sheath. ×75. Observation period: 1 week.

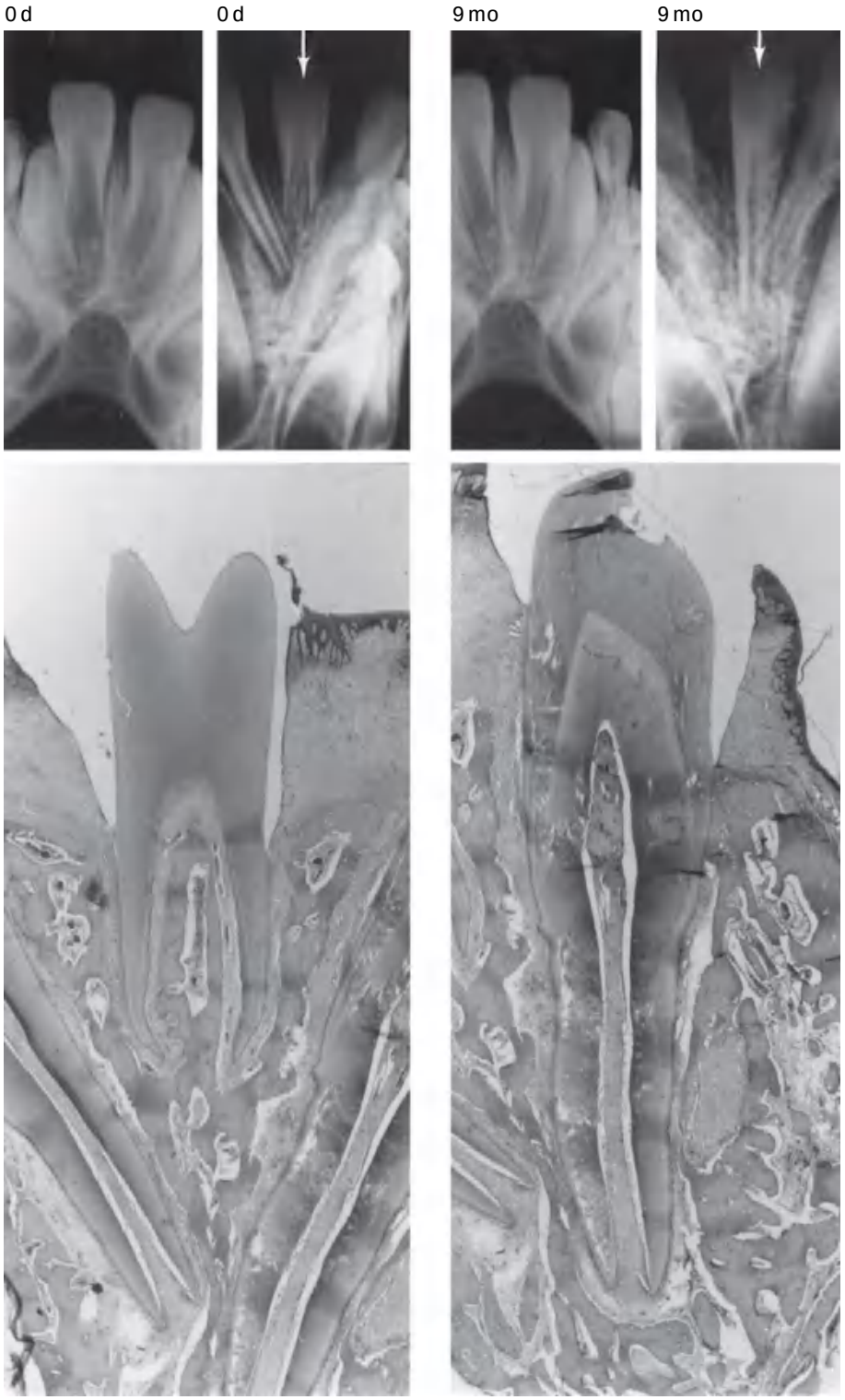
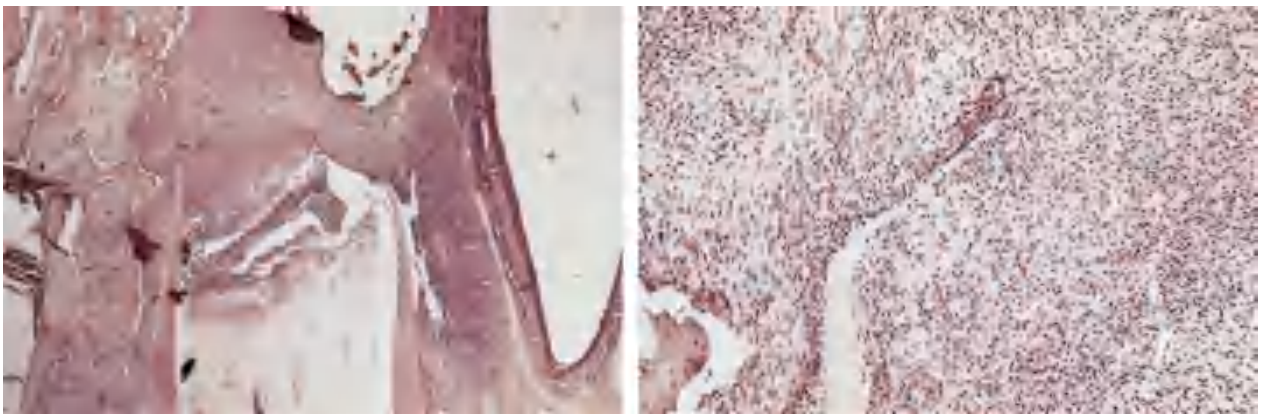



**Fig. 2.14** Effect of trauma to HERS upon root growth. Left: the right permanent central incisor in a monkey was extracted and the HERS was traumatized by pressing the apex against the socket wall before repositioning. The left central incisor was extracted and repositioned as a control. Right: condition 9 months after surgery in a monkey. Root development has stopped and bone has entered the root canal in the right central incisor. The left central incisor shows normal root development. From (95).

sometimes restricted, root formation has been found to occur in most cases of partial pulp necrosis irrespective of whether endodontic therapy has been instituted or not (see Chapter 25). This would seem to imply that HERS can continue to function despite inflammation (96, 101–108). In this context, there appears to be a critical distance between the root sheath and pulp resulting in pathologic changes in the pulp. If the distance is too short, inflammation will destroy the root sheath and root formation will be arrested (96) (Fig. 2.15). The fact that the epithelial root sheath can continue to function despite inflammation elicited by a partial pulp necrosis demonstrates that continued root development and apical closure cannot be taken as criteria for pulpal vitality (see Chapter 22).



A B

**Fig. 2.15** Critical distance between pulp necrosis and survival of HERS. A. The permanent central maxillary incisor was autotransplanted to the contralateral side in a monkey 1 week previously. ×25. B. Pulp necrosis extending to the apex mesially whereas the pulp necrosis zone is 1 mm short of the distal aspect of the apex. The HERS has survived and some root formation has occurred. ×75. From (95).

## Teeth with developed roots

### Gingival and periosteal complex

The gingiva is usually involved during crown-root and root fractures, luxation injuries and always during tooth avulsion. In addition, the periosteum is always involved during lateral luxation and alveolar bone fractures. In the following, the anatomy and function of the gingiva and periosteum will be described, as well as their response to trauma and infection.

#### Gingiva

The gingiva is defined from an anatomic point of view as either *free* or *attached*. The *free gingiva* comprises the vestibular and oral gingival tissue as well as the interdental papilla. Clinically, the apical border of the free gingiva is usually circumscribed by the free gingival groove (Fig. 2.16).

The *attached gingiva* is circumscribed coronally by the free gingival groove and apically by the mucogingival junction. The attached gingiva is firmly bound to the periosteum by collagenous fibers and is resistant to elevation.

The *alveolar mucosa* borders the attached gingiva and is loosely bound to the periosteum, thereby offering minimal resistance to the formation of a subperiosteal hematoma which can develop after lateral luxation, alveolar fracture or rupture from traction due to an impact parallel to the labial surface of the mandible or maxilla.

The function of the free gingiva is to seal, maintain and defend the critical area where the tooth penetrates its connective tissue bed and enters the oral cavity.

The gingival epithelium immediately adjacent to the tooth and *junctional epithelium* is designed to seal the periodontium from the oral cavity (Fig. 2.16).

Gingival epithelium displays a specific anatomy, being narrow and consisting of a few cell layers without rete pegs. The development of the junctional epithelium is closely related to tooth eruption. Thus, the junctional epithelium shows cell division originating in the basal layers, with cells being ultimately exfoliated into the gingival sulcus. Two zones can be recognized by transmission electron microscopy between the superficial cells of the junctional epithelium and the enamel; namely, the *lamina densa* and the *lamina lucida*, together called the *internal basement lamina*. Hemidesmosomes are formed by the epithelium adjacent to the lamina lucida, and these create an interphase with the enamel analogous to that with the subepithelial connective tissue (109).

Dental cuticle and sometimes a layer of fibrillar cementum can be seen interposed between this epithelium and the enamel. The *sulcular epithelium* faces the tooth without being in direct contact with it (Fig. 2.16). This epithelium is thicker than the junctional epithelium and has abundant rete pegs.

The *fibrillar system* of the gingiva is complex, comprising groups of collagen fibers with different sites of insertion (110–116) (Fig. 2.16).

The *dentinogingival fibers* originate in the cementum and insert in the free gingiva. The *dentoperiosteal fibers* start at the same site, but insert into the attached gingiva. The *transseptal fibers* extend from the cementum in the supra-alveolar region and insert into the supra-alveolar cementum of the adjacent tooth. The *circular fibers* encircle the tooth in a ring-like, supra-alveolar course. These fibers are responsible for the very rapid initial closure of an extraction wound (see Chapter 1), as well as the rapid adaptation of the gingiva around luxated or replanted teeth. Finally, *alveologingival fibers* emanate from the top of the alveolar crest and fan out into the attached gingiva.

The blood supply to the gingiva originates from three sources: the periodontal ligament, the crestal bone and the supraperiosteal blood vessels (see p. 81). This ensures survival of the marginal gingiva even after severe laceration or contusion (see Chapter 21).

#### Periosteum

The alveolar periosteum covers the alveolar process. In young individuals, at the time of active bone growth, it

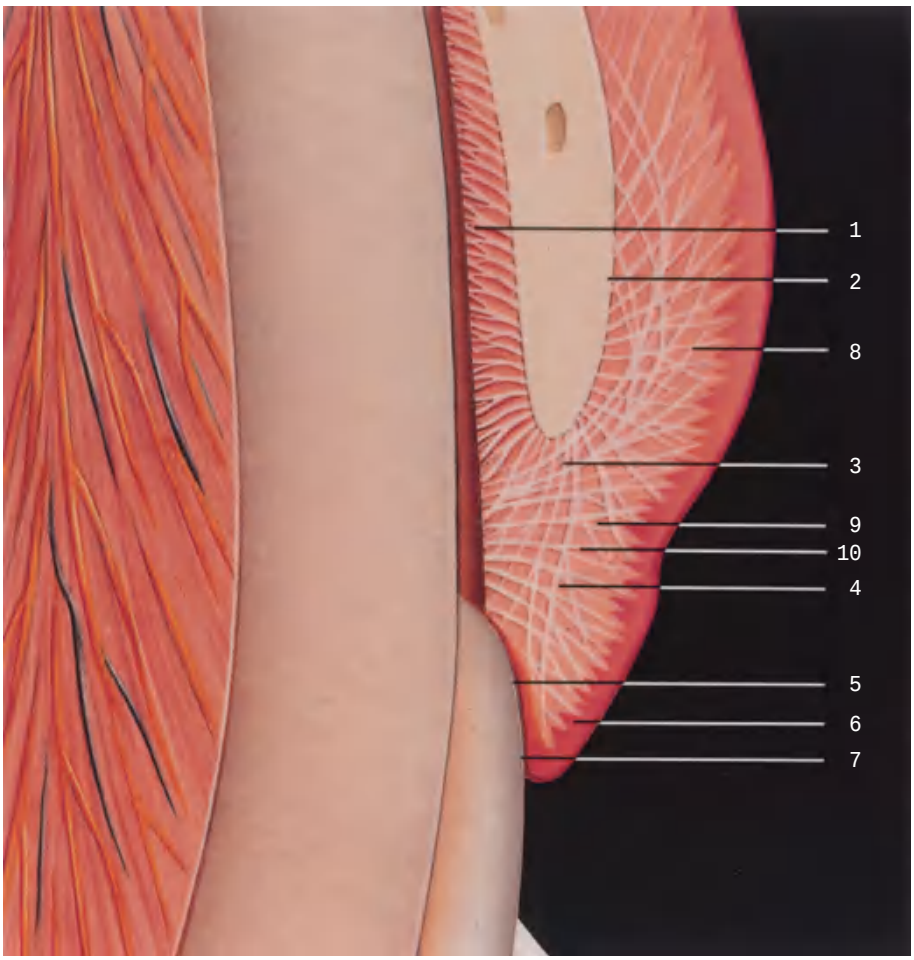
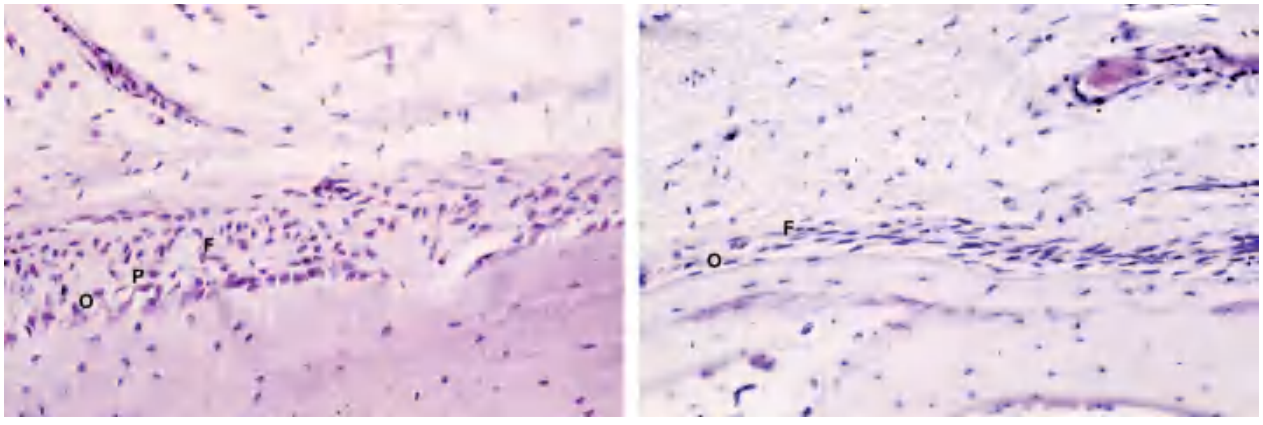



**Fig. 2.16** Anatomy of the gingiva and periosteal complex. 1, Sharpey's fibers; 2, dentoperiosteal fibers; 3, alveologingival fibers; 4, dentogingival fibers; 5, junctional epithelium; 6, gingival epithelium; 7, sulcular epithelium; 8, periosteogingival fibers; 9, intergingival fibers; 10, circular fibers.




A B

**Fig. 2.17** Differences in periosteum related to age. A. Cellular alveolar periosteum in a young monkey (root development of permanent incisors incomplete). F, fibroblasts; O, osteoblasts; P, precursor cells. ×75. B. Periosteum in an old monkey. Note the difference in the configuration of periosteum. ×75.

consists of an inner layer of angular osteoblasts followed by spindle-shaped precursor cells supported by loosely arranged collagen fibers (Fig. 2.17). In older individuals, where growth has ceased, the inner layer consists of flattened bone-lining cells followed by an outer fibrous layer of inactive osteoprogenitor cells which, however, still maintain their potential for cell division (117–119) (Fig. 2.17).

The periosteum serves an important function in appositional growth, remodeling and bone repair after injury. Furthermore, it anchors muscles and carries blood vessels, lymphatic vessels and nerves.

### *Response to trauma*

The gingival attachment is often torn during luxation injuries and always during avulsions. In an experimental replantation study in monkeys, the junctional epithelium showed increased autoradiographic labeling of cells as early as after

1 day, and reached a peak after 3 days. After 7 days, a new junctional epithelium was formed (120). In the connective tissue, the ruptured gingival and transseptal collagen fibers were united in the majority of cases after 1 week (121–123).

The response of periosteum to trauma is not very well described. Displacement of the attached gingiva or the alveolar mucosa involves injury to the periosteum and the underlying bone (124–161). Thus, the surface of bone is affected in several ways. Firstly, the cortical bone plate loses an important part of its vascular supply. Secondly, the cellular cover of bone provided by the innermost layer of periosteum is partially or totally removed. These two events invite an initial resorption of the bony surface which, however, is then followed by bony deposition to repair the initial loss.

In addition to bone loss due to displacement of the gingiva or alveolar mucosa, bone may also be lost directly due to trauma. In such cases, an important factor related to healing is the osteogenic potential of the periosteum, which is strongly influenced by age (152–154). Thus, when a periosteal flap is raised in *adult* animals, the osteogenic layer is usually disrupted and periosteal osteogenesis can only take place from the periphery of the wound where progenitor cells have not been disturbed (155–158), implying that bony repair will be limited and fibrous scar tissue will often form in its place (162, 163).

Conversely, in young animals, cells in the cambium layer of elevated flaps exhibit osteogenic potential and the bone contour is often fully repaired. In humans, surgical removal of large portions of the mandible, but with intentional preservation of the periosteum, has been seen to result in extensive new bone formation (160, 161).

### *Response to infection*

The general response of gingiva to infection has been the subject of numerous studies on plaque-induced gingivitis and is beyond the scope of this text. The significance of infection will be discussed only as it relates to the gingival attachment after traumatic or surgical injury. In monkeys, it has been shown that incisional wounds from the gingival sulcus to the alveolar crest lead to epithelial growth through most of the supracrestal area when gingival inflammation was induced (164).

The role of bacteria in gingival healing after surgical injury has been assessed only indirectly through the effect of antibiotic therapy. The use of antibiotics (tetracycline) in rats after injuries to pulpal, gingival and root tissues was shown to promote gingival and periodontal reattachment (165–167). In humans, it was demonstrated that the administration of erythromycin for 4 days after gingivectomy led to completed epithelialization after 1 week, in contrast to only two-thirds of the specimens when no antibiotic coverage was given (167). In conclusion, there is some evidence that bacteria-induced gingival inflammation has a negative influence upon wound healing after trauma and surgical injury.

If an odontogenic infection spreads from the bone marrow to the periosteal layer and an abscess is formed between the periosteum and cortical bone, the blood supply to the immediately underlying cortical bone can be compromised. The cortical bone can then undergo ischemic necrosis, resulting in an undermining resorption (168).

## Cementum–periodontal ligament complex

The anatomic border for the PDL is the most cervically located of the principal fibers (Sharpey's fibers) that insert into both cementum and alveolar bone (Fig. 2.18). In this context, only the anatomy and function of the more important cellular and fibrillar structures of the PDL will be considered. The reader is further referred to a series of review articles dealing with the cementum, PDL and alveolar bone (618–621).

#### Cementoblasts

Cementoblasts are spindle- or polyhedral-shaped cells whose long axes are usually oriented parallel to the root surface. The cytoplasm is basophilic and the nuclei are round or ovoid (Fig. 2.18). These cells are said to be active or resting according to the relative amount of cytoplasm. Resting cells contain less cytoplasm than active cementoblasts (169). Cementoblasts produce the organic matrix of cementum (i.e. intrinsic collagen fibers and ground substance), while the extrinsic fibers (i.e. Sharpey's fibers) are formed by fibroblasts from the PDL (170). If the cementoblast becomes incorporated into the mineralizing front, cellular cementum is formed. The deposition of cementum appears to occur rhythmically throughout life, at a speed of approximately 3 μm per year (171). Periods of activity alternate with periods of quiescence, thereby giving rise to incremental lines (172).

#### Periodontal fibroblasts

These cells are spindle-shaped, with several points of contact with adjacent cells. The nuclei are oval, containing one or more prominent nucleoli (Fig. 2.18). In sections parallel to Sharpey's fibers, fibroblasts appear as spindle-shaped cells with only occasional contact with other fibroblasts. In transverse sections, however, they are seen as stellate cells whose processes envelope the principal periodontal fiber bundles and connect with many other fibroblasts to form a cellular network (173). This intricate relationship between fibroblasts and Sharpey's fibers after injury may be important in the rapid degradation or reformation of Sharpey's fibers (175, 176). Anatomic (174) and *in vitro* (173) studies suggest that different fibroblast populations exist in the PDL (174, 177).

Fibroblasts are responsible for the formation, maintenance and remodeling of PDL fibers and their associated ground substance. By using tritiated thymidine labeling, it has been found that fibroblasts comprise a very active cell renewal system. In mice, for example, a cell turnover rate of 30% was seen when continuous labeling was used over a period of 25 days (178, 179). Furthermore, cell renewal took place as a clonal paravascular proliferation of cells. Together with programmed cell death (apoptosis) in the PDL, a steady state was created in the cellular content of the periodontal ligament (179–181).

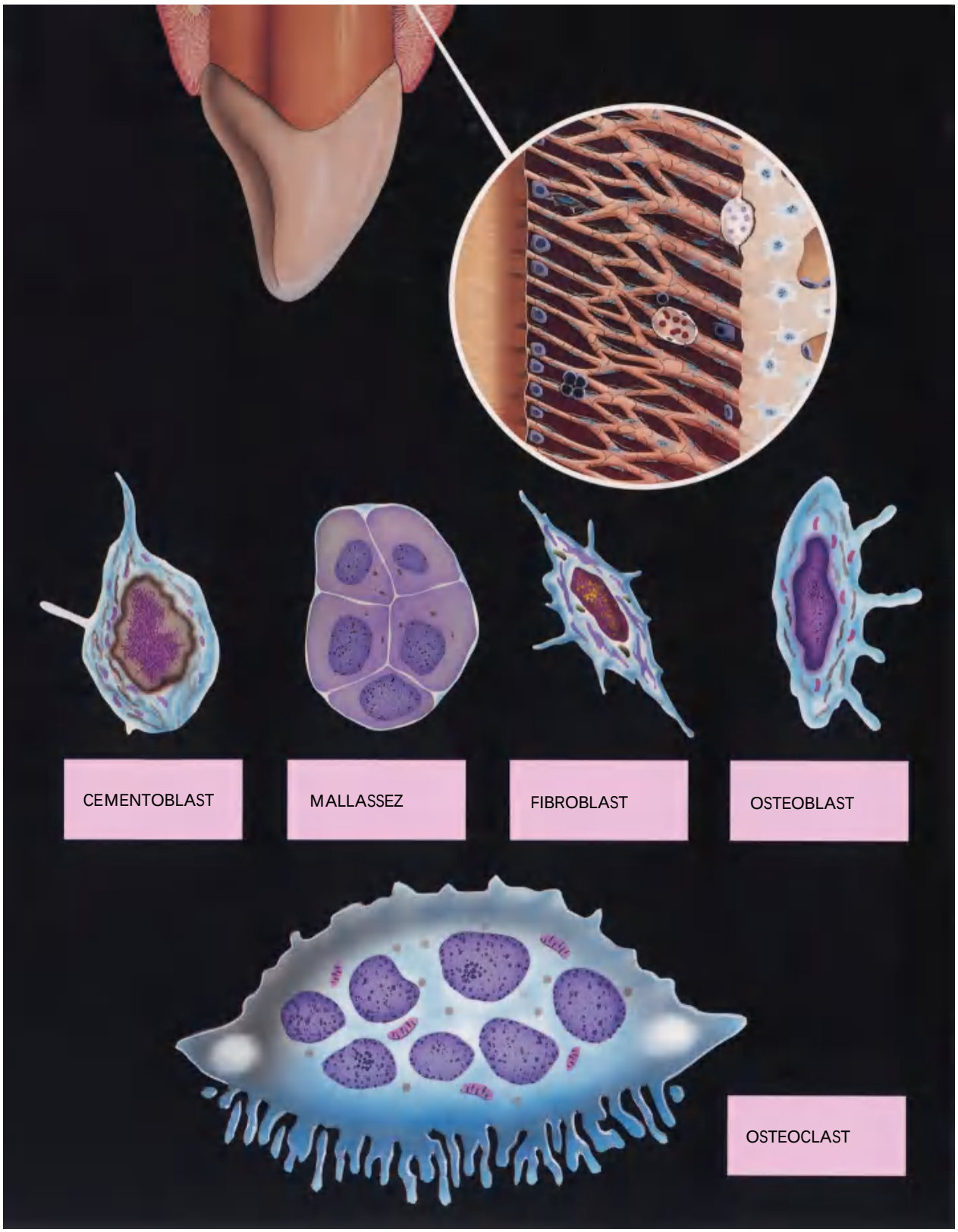



**Fig. 2.18** Anatomy of the periodontal ligament: Sharpey's fibers, cementoblast, Mallassez epithelial island, fibroblast, osteoblast and osteoclast.

As already stated, an important function of fibroblasts in the PDL is the maintenance of periodontal fibers. This function is manifested by very rapid collagen synthesis and degradation (turnover) (182–186), occurring primarily in the middle zone of the PDL (187). Thus, it has been shown that the half-life of collagen in the PDL of rats is about 6–9 days (188, 189). Moreover, collagen turnover is considerably more rapid in PDL collagen than in that of gingiva, pulp or other connective tissues in the body (190, 191). This very rapid turnover in the PDL is in agreement with findings

from studies in PDL wound healing, where very rapid healing of the periodontium has been demonstrated after surgical injury.

Finally, for the sake of completeness, it should be mentioned that periodontal fibroblasts are also responsible for synthesis and maintenance of other fibers, such as elastin and oxytalan.

#### Osteoblasts

The osteoblast is slightly larger than the cementoblast and has a circular or ovoid nucleus which is usually located eccentrically. The cytoplasm is abundant and very basophilic during osteogenesis (Fig. 2.18). Like the cementoblast, the osteoblast is found in an active or resting form, as revealed by less cytoplasm in the latter form.

#### Osteoclasts

Osteoclasts and osteoblasts are involved in both the formation and maintenance of bone (Fig. 2.18). The osteoclast, a bone-resorbing cell, also plays an important role in the healing events after trauma to bone and teeth as well as in the defense system established in respose to infection. Consequently, marked osteoclastic activity may be the explanation for a number of radiographic phenomena, including transient or permanent apical and marginal breakdown of the bony socket as well as root surface and root canal resorption (see Chapter 7).

#### Epithelial rests of Mallassez

A network of epithelial cells is found in the PDL positioned close to the root surface (Fig. 2.18). These cells originate from the successive breakdown of the HERS during root formation.

It has been proposed that these epithelial cells play a role in the homeostasis of the PDL (192–194, 198, 622, 623) as well as playing a crucial role in periodontal regeneration and cementum formation (623, 624). These cells also represent an important part of the defense system of the PDL against invading bacteria from the root canal. Bacterial invasion of both the main and lateral canals leads to proliferation and adherence of the epithelial cells to the root canal openings at the PDL to form an epithelial barrier against the invaders (201).

#### Periodontal ligament fibers

The vast majority of collagen fibers in the PDL are arranged in bundles, the so-called principal fibers (Sharpey's fibers) (Fig. 2.18). These fibers are embedded in both cementum and bone. In their course from cementum to alveolar bone, they are often wavy at their midpoint, giving the impression of an intermediate plexus, with interdigitation of fibers. However, recent scanning electron microscopic studies seem to indicate that the majority of the principal fibers span the entire PDL space, although they usually branch and join adjacent fibers to create a ladder-like architecture in the PDL (202–216) (Fig. 2.18). The principal fibers extending from cementum to bone can be classified according to their direction and location into horizontal, oblique and apical fibers. In the gingiva, the supra-alveolar (gingival) fibers can be classified into dentogingival fibers, dentoperiosteal fibers, interdental fibers and circular fibers (see p. 78) (Fig. 2.16).

The main function of the PDL is to support the tooth in its alveolus during function. Whenever functional demands are changed, corresponding adjustments take place in the architecture of the PDL, such that the orientation, amount and insertion pattern of the principal fibers are altered.

#### Periodontal vasculature

The blood supply to the PDL at the midportion of the root appears to arise from branches of the superior or inferior alveolar arteries. Before these arteries enter the apical foramen, they give off branches to the interdental bone. On their way to the alveolar crest, they give off multiple branches which perforate the socket wall and form a plexus that surrounds the root surface (217–222). This plexus is located in the interstitial spaces between Sharpey's fibers. In primates, the majority of these vessels are located close to the bone surface (221). *Apically*, the blood supply to the PDL originates from branches of the dental arteries which are released as they cross the PDL and enter the pulp. *Cervically*, however, anastomoses are formed with gingival vessels (Fig. 2.19).

The blood supply to the gingiva appears to arise primarily from the supraperiosteal blood vessels, which anastomose in the gingiva with vessels from the PDL and branches of the interdental arteries and which perforate the crestal bone margin or the labial or lingual bone plate.

#### Periodontal innervation

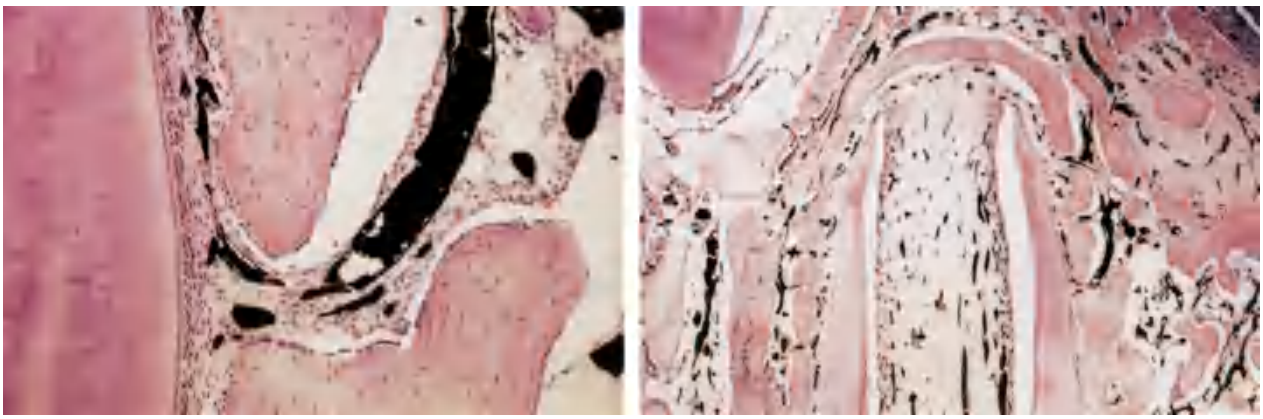
The PDL has receptors for pain, touch, pressure and proprioception, which all belong to the somatic nervous system. Autonomic nerves are also present, which innervate the blood vessels. In general, periodontal innervation follows the same pathways as the blood supply. In one experimental study it was also found that sensory fibers promote root resorption after pulpo-periodontal injury (223).

#### Homeostatic mechanism of the PDL

The PDL appears to exist throughout life with minimal variations in width (224, 225). This dimension is primarily controlled by age and function, but is also affected by pathology (226).

The homeostasis against resorption in the PDL space (and the pulp) has always been an enigma. Previously, it was hypothesized that the periodontal (227, 228, 255) and pulpal (229, 230) cells act as a barrier that prevents resorption of the roots by inhibiting the resorbing cells from gaining access to cementum and dentin (224, 229, 231–235).Apparently, these cells on the root surfaces, normally give protection against an osteogenic cell invasion. New molecular signals have recently been identified in various cell types (229, 230) such as PDL cells, odontoblasts, osteoblasts and cementoblasts (227), which may explain the homeostasis phenomenon.

Different cell types, including preosteoblasts, express active signal molecules in a membrane-bound (RANKL) and a soluble (sRANKL) form. The RANKL (receptor



A B

**Fig. 2.19** Vascular supply to the periodontium. A. Vascular supply to the PDL in a longitudinally sectioned permanent maxillary central incisor in a monkey shown by India ink perfusion. ×10. B. The interdental arteries give off multiple branches to the midportion of the PDL. ×100.

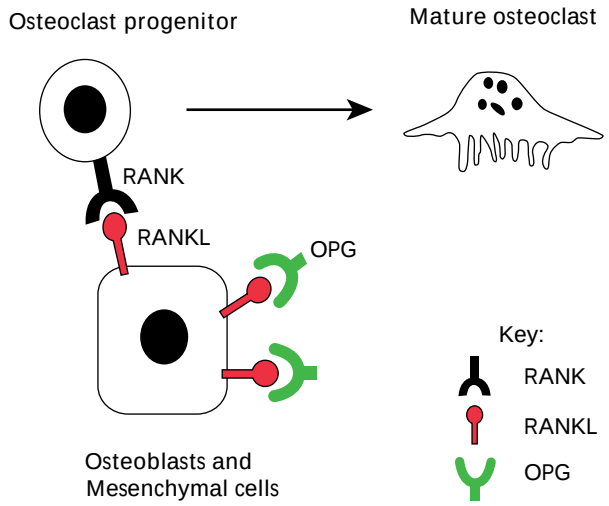



**Fig. 2.20** RANK, RANKL and OPG system monitoring osteoclast activity.

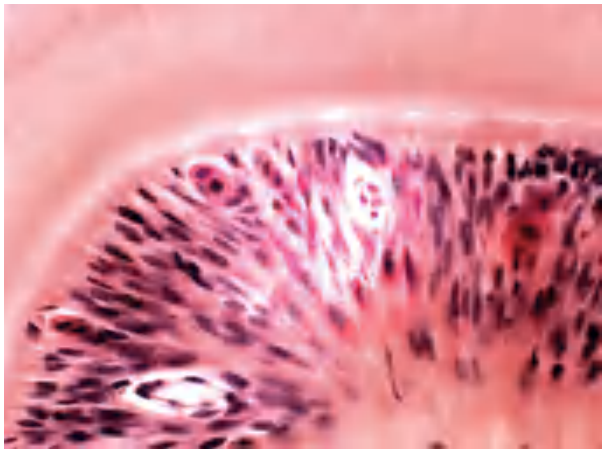


**Fig. 2.21** Intraradicular root surface of a rat molar where migration of osteoclasts is arrested in the periodontal ligament.

activator of nuclear factor κB ligand) molecules are ligands with the capacity to activate mononuclear osteclast progenitor cells with the receptor (RANK) attached to their surface. If these two molecules – the ligand and the receptor – meet, the osteoclast progenitor cells become differentiated and merge with other progenitor cells to form osteoclasts (236–241), which potentially may attack the root surface or the root canal.

A third molecule called osteoprotegerin (OPG) is a soluble molecule, i.e. a decoy receptor. The OPG is able to bind and cover both the RANKL molecules on adjacent cells and the soluble sRANKL, and OPG is thereby able to give protection to the root surface (236, 241) (Fig. **2.20**).

When the OPG/RANK/RANKL signaling pathway is in action it is able to both protect against or activate osteoclastic activity, and recent studies suggest this as a possible explanation for the protective action of the hard tissue covering cells (i.e. odontoblasts, cementoblasts and PDL cells) (Fig. 2.21). Furthermore, the OPG/RANKL ratio level can be upregulated or downregulated by growth factors, hormones and inflammatory factors (e.g. interleukins) which may explain a number of resorption phenomena such as osteoporosis and inflammatory root or bone resorption (see Chapters 7 and 8) (238).

Previous theories about periodontal homeostasis are supported with recent molecular evidence. This new knowledge about the OPG/RANK/RANKL system appears to explain central aspects of tooth eruption and shedding of primary teeth (241–244), progression of ankylosis (245–249), orthodontic tooth movement (250–253), periodontitis, periimplantitis and rheumatoid arthritis (253, 254).

Thus, the molecules RANKL/OPG have been found involved in physiologic primary tooth resorption (240) and in the follicles of erupting teeth (243, 244), and primary tooth shedding appears to be regulated by the RANKL/OPG signaling system (241).

In conclusion, observations on the OPG/RANK/RANKL system in the PDL seem to agree and support the previous hypothesis that the PDL cells act as a barrier that prevents resorption of the roots by inhibiting the resorbing cells (617). For a further discussion of the OPG/RANK/RANKL system the reader is referred to Chapter 7.

## *Response to trauma*

Following a severe dental injury (e.g. lateral luxation or intrusion) the PDL must respond to a variety of insults. These can include temporary compressive, tensile or shearing stresses, which result in hemorrhage and edema, rupture or contusion of the PDL. Each of these injuries can induce varying wound healing signals.

The cellular kinetics of healing in the PDL associated with a surgical wound have been examined using tritiated thymidine labeling of proliferating cells. Thus, in mice, paravascular cells were labelled within 100 μm of the margin of the injured PDL. Some of these cells moved into the injury zone 3–5 days after injury and divided (256). It would appear that in the PDL there is a population of paravascular stem cells that exhibits a high nuclear/cytoplasmic ratio and that remains stable throughout wound healing, but provides a front of new cells that migrate towards the wound and then divide (256–259) (Fig. 2.22). The identity of these progenitor cells is only partially known at present (260–262). It is possible that progenitor cells placed in the middle of the PDL supply the fibroblast population (256, 259), while progenitor cells close to the alveolar bone develop into osteoblasts (256–259, 263, 264). Cementoblasts precursors have not yet been identified; however, there is some indication that the progenitors for this cell population are located away from blood vessels (see Chapter 5) (259, 264).

### Bleeding and edema

Very little is known about healing events after bleeding or edema in the PDL subsequent to minor trauma (e.g. concussion or subluxation). However, in a clinical luxation study, a higher frequency of repair-related resorption was seen following concussion injury than after subluxation (265), suggesting that pressure from bleeding into the PDL after a mild injury might elicit minor areas of damage to the root surface. In the case of subluxations, where the impact is great enough to cause tooth loosening, this pressure can be relieved; however when no loosening results (i.e. concussion), pressure will be reflected in subsequent surface resorption.

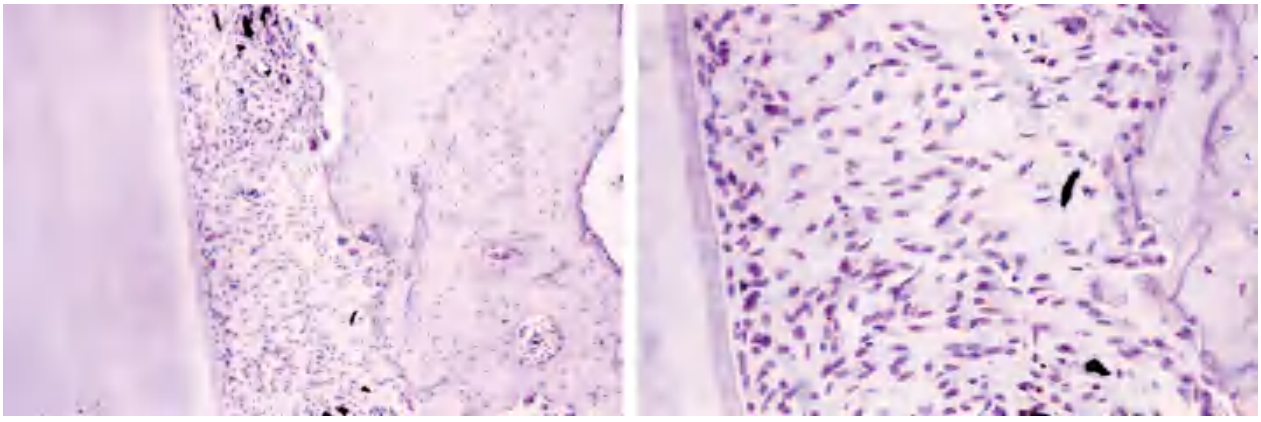
### Rupture of the PDL

There has been little investigation into the healing of a ruptured PDL. The few available studies are either of extrusive luxation (263, 266–268) or of extraction and subsequent replantation (120–122, 192, 269).

In monkeys, it appears that rupture of the fibers after extrusion or extraction usually occurs midway between the alveolar bone and the root surface. However, rupture can also be seen close to either the alveolar wall or the root surface. After 4 days, the cervical and apical part of the PDL appears revascularized (270). After 1 week, the split in the PDL is occupied by proliferating fibroblasts and blood vessels. In isolated areas, union of the principal fibers has already taken place (Fig. 2.22). After 2 weeks, a substantial number of principal fibers have healed. At that time, the mechanical properties of the injured PDL are about 50–60% of that of an uninjured PDL (266). With longer observation periods, an increased number of healed principal fibers can be seen. After 8 weeks, the injured PDL cannot be distinguished histologically from an uninjured control (266). If the alveolar bone is lost and there is an intact PDL covering the root, animal experiments have shown that the PDL will induce new bone (271, 272). In autotransplantation of teeth in humans the same phenomenon has been noted (see Chapter 33).

### Contusion injuries of the PDL

During intrusion, lateral luxation or avulsion with subsequent replantation, contusion of the PDL is a common occurrence (197). Wound healing is subsequently initiated when damaged tissue is removed by macrophage or osteoclast activity. During these events, not only are necrotic PDL tissue remnants removed, but sometimes also bone and cementum (273). The latter can lead to either *repair- or infection-related resorption* (surface or inflammatory resorption), depending upon the age of the patient, stage of root development and pulp status (224, 274) (Figs 2.23 and 2.24). When large areas of the PDL are traumatized, competitive wound healing processes begin between bone



A B

**Fig. 2.22** Healing of a ruptured periodontal ligament. A. Healing of the PDL after replantation of a permanent monkey incisor. The monkey was perfused with India ink at sacrifice 1 week after replantation. ×25. B. The PDL is very cellular and has lost its typical parallel arrangement of the fibroblasts. In a few places Sharpey's fibers appear united. ×100.

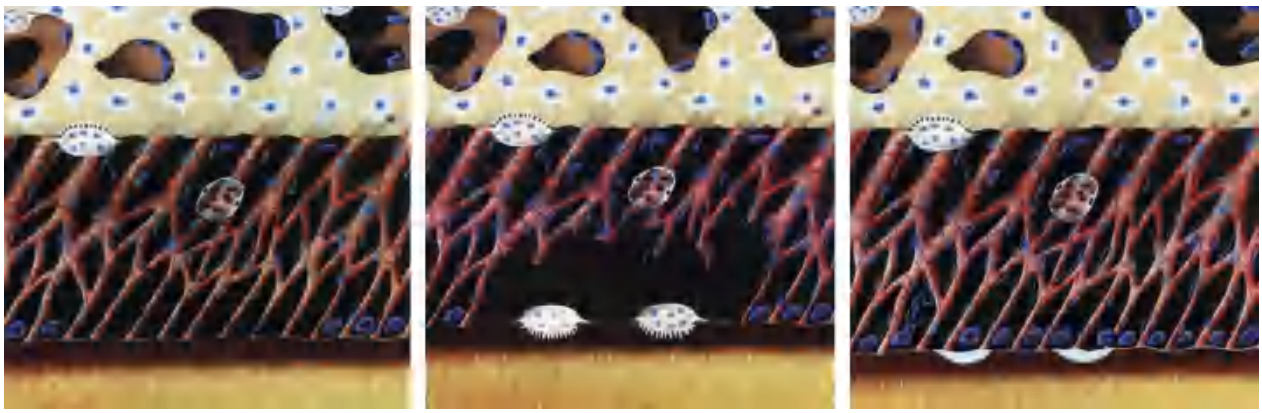


**Fig. 2.23** Repair-related resorption. Pathogenesis of repair-related resorption (surface resorption): healing with minor injury to the PDL ligament. The injury site is resorbed by macrophages and osteoclasts. The osteoclasts have exposed factors and other soluble molecules in dentin such as IGF-1, TGF-β, PDGF, BMPs, FGF-2 and A4 (amelogenin gene splice products). These molecules may serve as stimulators for cementoblasts (489). Subsequent repair takes place by the formation of new cementum and Sharpey's fibers.

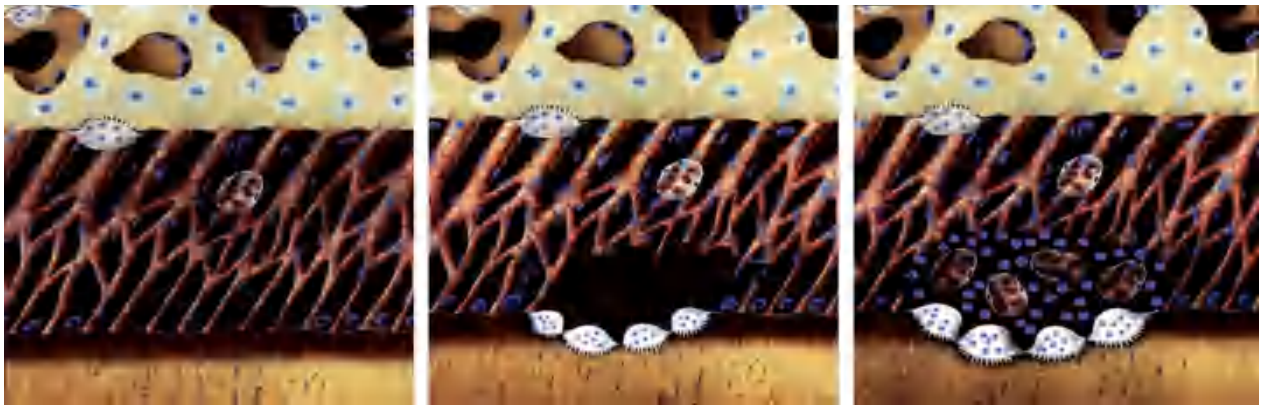


**Fig. 2.24** Infection-related resorption. Pathogenesis of infection-related resorption (inflammatory resorption): healing with moderate or extensive injury to the PDL and associated infection in the pulp and/or dentinal tubules. The initial injury to the root surface triggers a macrophage and osteoclast attack on the root surface. Osteoclasts have exposed toxins from bacteria located in the root canal and dentinal tubules such as LPS (lipopolysaccharide), MDP (muramyl dipeptide) and LTA (lipoteichoic acid). These toxins serve as direct activators for osteoclastic activity. The resorption process is accelerated and granulation tissue ultimately invades the root canal.

marrow-derived stem cells destined to form bone and PDL-derived cells which are programmed to form PDL fibers and cementum (224, 274, 275), resulting in *ankylosis resorption* (replacement resorption) (Fig. 2.25).

### *Response to infection*

Progression of gingival infection or spread of infection from the root canal through the apical foramen, accessory canals or dentinal tubules are the most common routes of infection to the PDL. Accordingly, the following discussion will emphasize PDL reactions due to infection in the root canal, based on several studies in humans and animals (278–285).

Cellular kinetics of the PDL due to pulpal infection have been examined in rats using tritiated thymidine. It was shown that after 1 hour there was increased labeling of fibroblasts, osteoblasts and cementoblasts, which reached a peak after 2–3 days. The increase in cellular activity continued in osteoblasts and fibroblasts for 30 days (the end of the experiment) while the cementoblast layer returned to its normal level after 25 days (276, 277).

Moderate or intense periapical inflammation has been found to lead to a breakdown of PDL fibers and resorption of the root apex and bone, resulting in an expansion of the PDL space or a periapical rarefaction (226–233) (Fig. 2.26). This resorption response has been found to be related to the presence of bacteria and their toxins in the root canal (286–295). This is supported by experiments in germ-free or conventional rats (617). The stimulus for osteoclastic activity affecting the root surface and alveolar bone appears to be a combination of a direct influence of bacteria and their toxins and an indirect influence on the osteoclast in response to inflammatory changes (224, 274, 296, 297) (Fig. 2.26). The cell population in the PDL, however, appears to be rather resistant to infection in the sense that, when infection has been eliminated, the PDL usually returns to normal. This phenomenon has been found in the marginal periodontium (298, 299), in the apical periodontium (300, 301) and in situations involving the entire PDL, such as after osteomyelitis (302). However, if the infection is not completely eradicated, chronic inflammation will develop and allow only minimal cementum and PDL repair (277, 303).

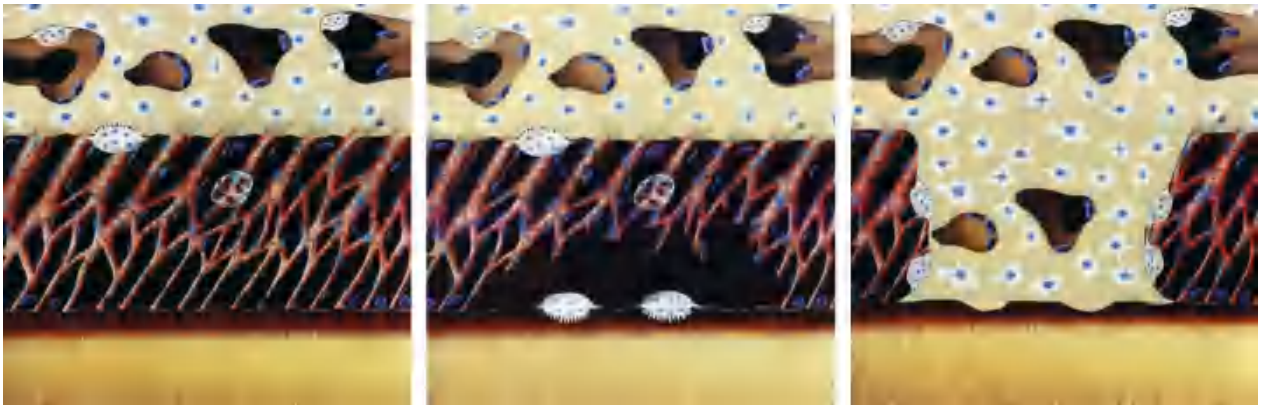
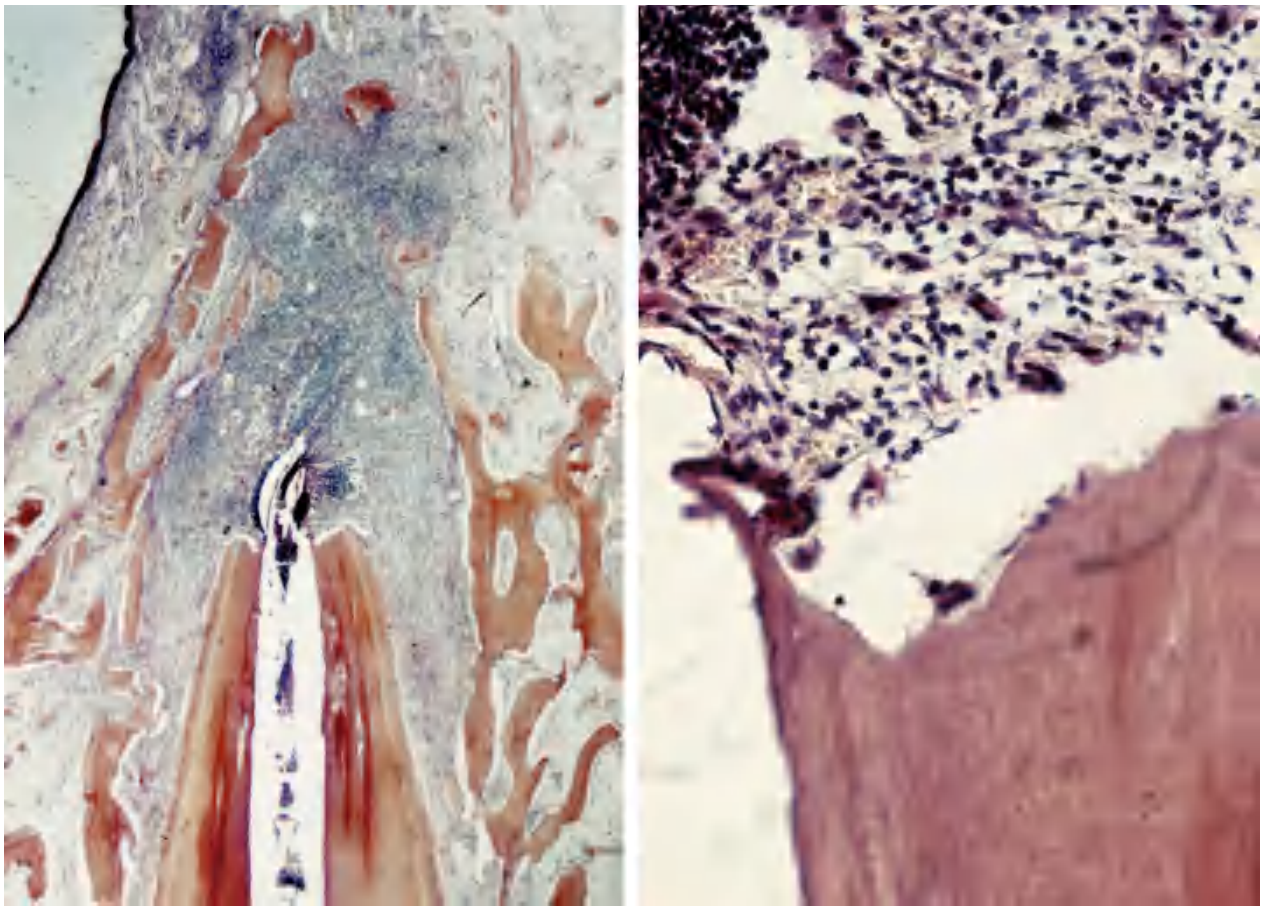


**Fig. 2.25** Ankylosis-related resorption. Pathogenesis of ankylosis-related resorption (replacement resorption): healing after extensive injury to the PDL. The osteoclasts have exposed factors and other soluble molecules in dentin such as IGF-1, TGF-β, PDGF, BMPs, FGF-2 and A4 (amelogenin gene splice products). These molecules may serve as stimulators for osteoclasts and/or bone formation (532). Ankylosis is formed because healing occurs almost exclusively by cells from the alveolar wall.



**A** **B**

**Fig. 2.26** Periapical inflammation and apical root resorption subsequent to pulp necrosis in a replanted permanent monkey incisor. A. ×10. B. ×100.

PDL healing after initial root resorption or surgical injury to the root surface is influenced by a number of factors, including age, size of lesion and functional stimuli, but usually leads to a new formation of cementum and inserting fibres (304). The process of healing is under the control of extracellular matrix, glycoproteins, growth factors and cytochines in a very complex process (for a survey see (303)).

The presence of bacteria in the root canal and dentinal tubules has been shown experimentally to interfere with the expected normal healing process with new deposition of cementum and reformation of PDL fibers. Instead a chronic inflammation occurs with osteoclastic activity, and cementum repair becomes restricted (306, 307). The presence of an intact cementum layer apparently prevents toxins and bacteria from interfering with healing.

With regard to periodontal apical healing, after endodontic treatment of an infected root canal a large study in monkeys has shown that the presence of bacteria in the canal after root filling results in significantly lower healing frequency (21% versus 72%) (618).

## Alveolar bone and marrow complex

The alveolar process may be defined as the tooth-supporting region of the mandible or maxilla (Fig. 2.27). It is made up of three components: (1) the *alveolar bone proper*, consisting of a thin plate of bone which provides attachment for either the dental follicle or the principal fibers of the PDL (308); (2) the *cortical bone plates* which form the outer and inner plates of the alveolar process; and (3) the *cancellous bone* and *bone marrow* which occupy the area between the cortical bone plates and the alveolar bone proper.

The *alveolar bone proper* consists of a thin, perforated bone plate, which appears radiographically as a radiopaque lining around the radiolucent PDL space (i.e. lamina dura). The perforations in the socket wall function as gateways for vascular channels that supply the periodontal ligament (309).

The *cortical bone plates* form the lateral borders of the alveolar process and are usually much thinner in the maxilla than in the mandible. In the incisor and canine regions, there is usually no cancellous bone or bone marrow to separate the lamina dura from the cortical plates, and this results in a fusion of these structures. For this reason, lateral luxation of maxillary incisors produces a combined fracture of the lamina dura and the cortical bone plate.

The *cancellous bone* consists of thin bone trabeculae which encircle the bone marrow. These trabeculae are lined with a delicate layer of connective tissue cells, the endosteum. The bone marrow consists of a reticulate tissue in which cells representing different stages of hematogenesis occupy the mesh (*red bone marrow*). If the hematopoietic cells disappear, the reticulate tissue is replaced either by adipose tissue (*yellow bone marrow*) or, as in some areas in the alveolar process, by fibrous tissue (*fibrous bone marrow*) (310). The exact topographic distribution of the various types of bone marrow in humans has not yet been documented.

As previously mentioned, the primary function of the *alveolar process* is to protect and support the dentition. More specifically, the alveolar bone proper protects the developing tooth and later provides an anchorage for the fibers of the PDL (311). Apart from its hemopoietic function, the *bone marrow* plays an important role in osteogenesis. Thus, the stromal cells of the bone marrow stroma can manifest osteogenic activity when stimulated by trauma (312). Furthermore, bone marrow plays an important role in the defense against infection (295).

### *Growth and maintenance*

Under normal physiologic conditions, osteoclast activity is regulated by a combination of direct and indirect osteoclast *activators* (e.g. parathyroid hormone (PTH), vitamin D metabolites, plasma calcium concentration, neurotransmitters, growth factors and cytokines) and osteoclast *inhibitors* (e.g. calcitonin and estrogen). The sum of osteoblast/osteoclast activity (i.e. net bone balance) can be seen in normal tooth eruption, growth and maintenance of the jaws, as well as in the homeostasis of plasma calcium and phosphate (Fig. 2.28).

### *Response to trauma*

The most common traumatic injury to the alveolar bone complex is extraction/avulsion or a luxation wound with displacement. Furthermore, the bone becomes involved after alveolar and bone fractures (see Chapter 21).

With respect to wound healing in hard tissues, the osteoclast can be considered analogous to the macrophage system

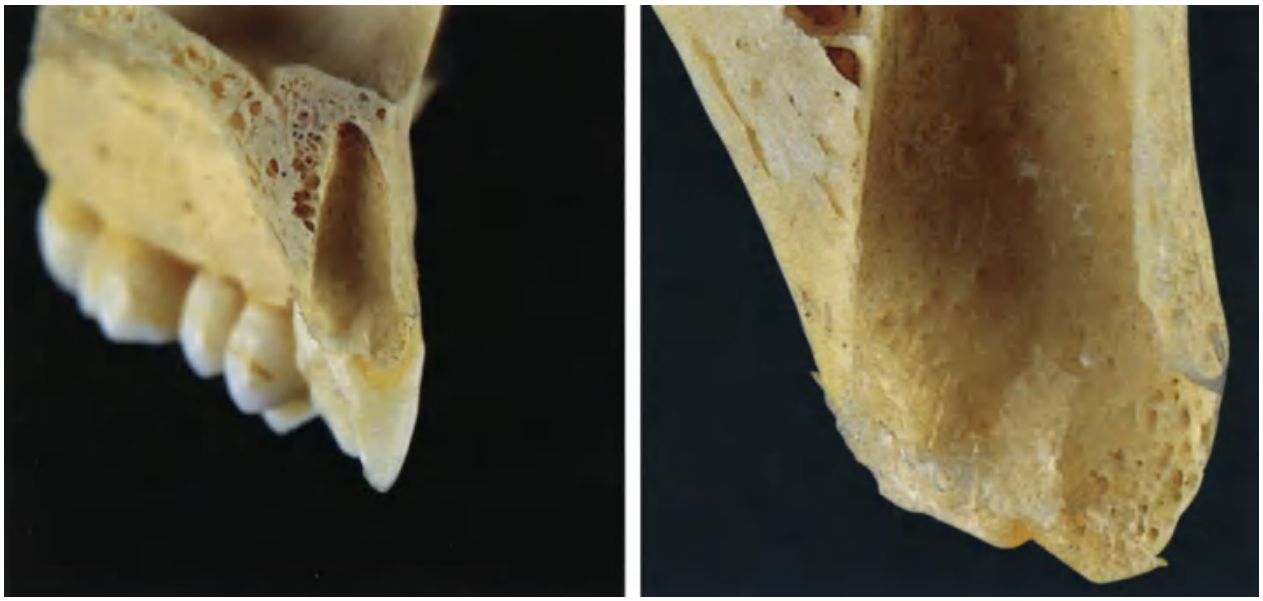


**Fig. 2.27** Anatomy of the alveolar bone. Section through the central incisor region in a human jaw. Note the fused alveolar bone proper and cortical bone plate labially, whereas both structures can be recognized palatally. Furthermore, multiple vascular canals perforate the lamina dura.

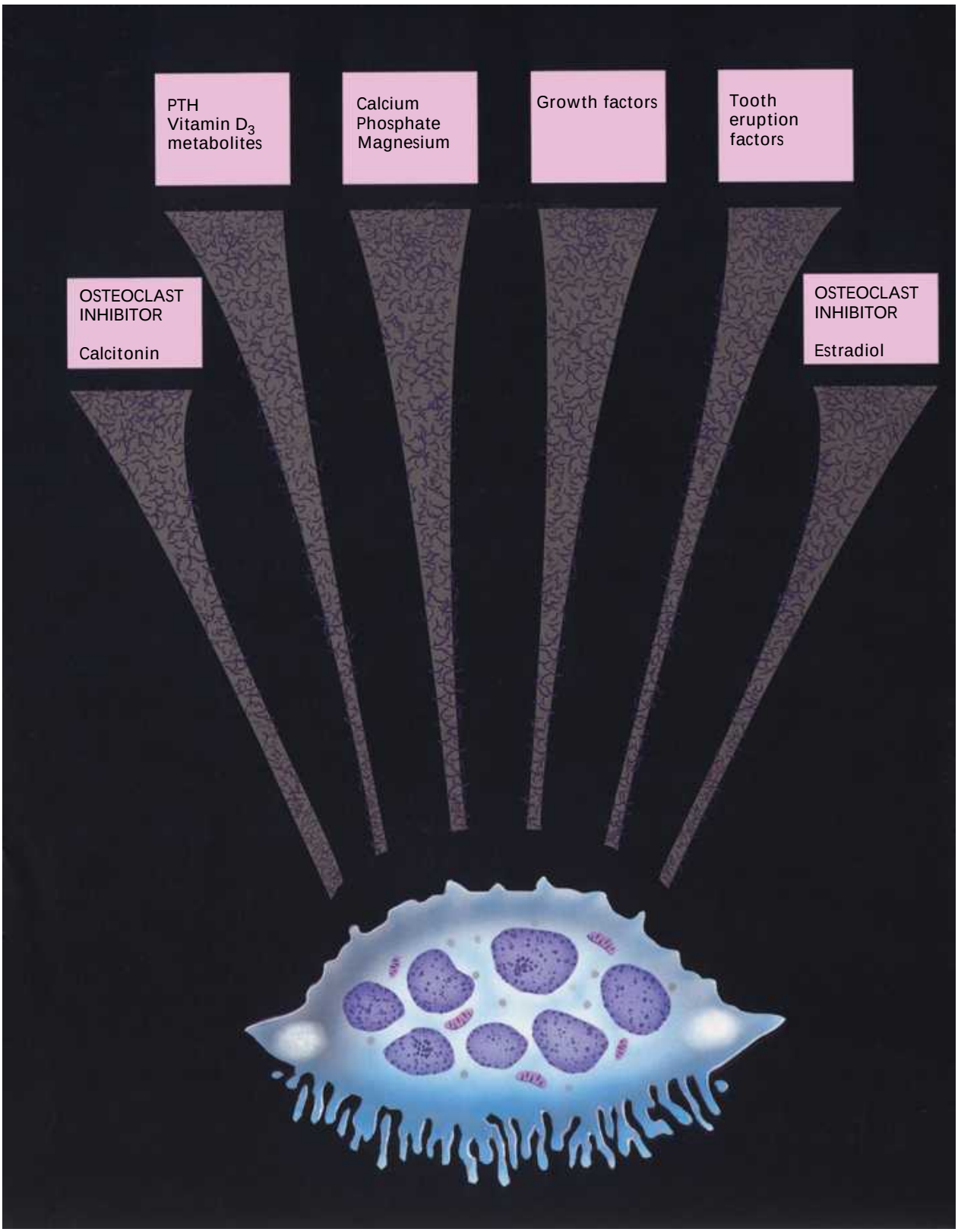



**Fig. 2.28** Activators and inhibitors regulating osteoclast activation during growth and maintenance of bone. PTH, parathyroid hormone.

operating in soft tissue wounds in its response to trauma and/or infection.

There are three hard tissues in the oral cavity that are at risk to resorption subsequent to a trauma: alveolar bone, dentin and cementum. Alveolar bone is itself highly vascular, while dentin and cementum are adjacent to the vascular tissues of the dental pulp and PDL, respectively. Thus, all three of these hard tissues are readily accessible to blood-derived inflammatory cells and serum proteins and can be resorbed as a result of inflammatory reactions.

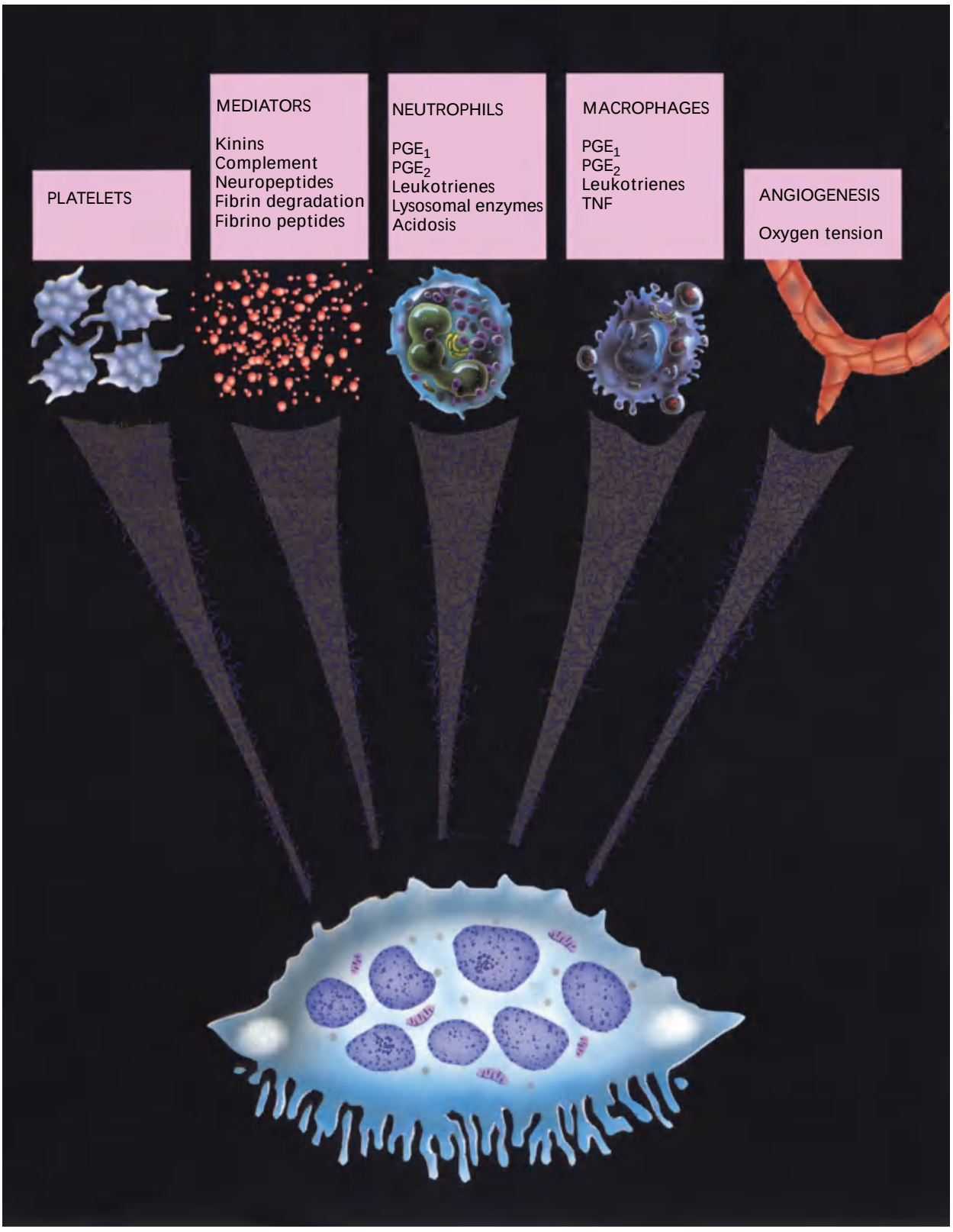



**Fig. 2.29** Osteoclast activators released during inflammatory response elicted by a traumatic dental injury. PGE, prostaglandin; TNF, tumor necrosis factor.

Hard tissues may be injured following trauma either directly (e.g. crushing injuries) or indirectly (e.g. ischemic injuries). In both instances, an inflammatory response is elicited that results in the liberation of cytokines and the promotion of hard tissue resorption (Fig. 2.29). The purpose of this response is to remove the damaged hard tissue prior to healing. In this regard, the integrity of the cementoblast and odontoblast layers that cover cementum and dentin, respectively, is of paramount importance. If the traumatic event results in an irreparable injury to these cell layers, the

hard tissue surface may succumb to resorption due to the intense osteoclastic activity that results from the liberation of a series of osteoclast-activating factors (Fig. 2.30).

Extensive studies have been conducted regarding socket healing after extraction whereas none has yet been performed relating to tooth luxation. The following is a synopsis of what is presently known about human extraction wounds.

#### Histologic evidence of socket healing

The following overlapping stages have been found histologically, based on biopsies from the healing of normal extraction wounds in human patients (313–326), and an excellent review has recently been published by Farina and Trombelli (629).

*Stage I*. A *coagulum* is formed once hemostasis has been established. It consists of erythrocytes and leukocytes, in the same ratio as in circulating blood, entrapped in a mesh of precipitated fibrin.

*Stage II*. *Granulation tissue* is formed along the socket walls 2–3 days postoperatively and is characterized by proliferating endothelial cells, capillaries and many leukocytes. PDL fibroblasts appear to immigrate into the coagulum and differentiate into osteoblasts (327). Within 7 days, granulation tissue has usually replaced the coagulum.

*Stage III*. *Connective tissue* formation begins peripherally and, within 20 days postoperatively, replaces granulation tissue. This newly formed connective tissue comprises cells, collagen and reticular fibers dispersed in a metachromatic ground substance.

*Stage IV*. *Bone development* begins 7 days postoperatively. It starts peripherally, at the base of the alveolus. The major contributors to alveolar healing appear to be cancellous bone and bone marrow while the remaining PDL apparently plays an insignificant role (327, 328). By 38 days, the socket is almost completely occupied by immature bone. Within 2–3 months, this bone is mature and forms trabeculae; after 3–4 months maturation is complete (324).

*Stage V*. *Epithelial repair* begins closing the wound 4 days after extraction and is usually complete after 24 days.

Finally, it should be mentioned that socket healing has been found to be significantly influenced by the age of the individual (315, 325). Thus, histologic alveolar socket repair is more active 10 days postoperatively in individuals in the second decade of life, while the same stage of activity is seen about 20 days postoperatively in individuals in the sixth decade or more. However, after 30 days, the healing sequence levels out and becomes identical in the two age groups (325).

Finally, a significant amount of bone remodeling, especially of the labial bone, will take place in the following months (329), a phenomenon which has a significant influence upon timing of implant insertion (see Chapters 34 and 35).

### *Response to infection*

The most common situations in which the alveolar bone becomes involved in infection are during marginal or periapical periodontitis (330, 331). The most frequent complication after replantation procedures and certain luxation types is infection of necrotic pulp tissue, thus emphasis will be placed on the reaction of the alveolar bone to infection of the root canal space.

In the case of bacterial contamination of root surface this response is a necessary step in the fight against invading bacteria (Fig. 2.30).

The events observed in the progression of necrosis and infection of the pulp tissue following pulp exposure due to crown fracture or caries are different than those observed as a consequence of luxation injury. In the latter, the severance of the vascular supply produces a necrotic and initially sterile pulp tissue. Bacteria reaching the pulp space through exposed dentin tubules, cracks or apical foramina will easily colonize the necrotic tissue because of the lack of inflammatory response (see later).

When necrosis and infection affect the root canal, some early inflammatory changes take place in the periapical tissue. These may be seen in radiographs as a widening of the PDL space, or as a frank periapical radiolucency. These observations do not lend credit to the widely held opinion that the entire pulp must be necrotic for an apical periodontitis lesion to form.

The result of root canal infection is inflammation in the periapical region, often referred to as *apical periodiontitis* (334–347). Clinically, it can be asymptomatic or symptomatic. Depending on the distribution of the various types of inflammatory cells and on the occurrence of epithelium, the periradicular lesions can be histologically classified into granulomas, abscesses and cysts (632–634, 658).

A *granuloma* is a chronic inflammation that consists of a granulomatous tissue infiltrated mainly by lymphocytes, plasma cells and macrophages (Fig. 2.31). These lesions can be *epithelialized* and *non-epithelialized*. All types of chronic inflammatory cells can be observed in varying numbers and distribution within the connective tissue of the granuloma: lymphocytes, plasma cells, macrophages, foam cells and multinucleated foreign body cells. In the capsule surrounding the periapical inflammatory zone, the dominant cell is the PDL fibroblast. Thus the capsule can be regarded as an extension of the PDL (344).

An *abscess* consists of a collection of pus in a cavity formed by tissue liquefaction. It is formed by an accumulation of polymorphonuclear neutrophils (PMNs), usually delimited by a granulomatous tissue composed mainly of lymphocytes and plasma cells. This is because, in many cases, the abscess is essentially a granuloma that underwent acute exacerbation, showing areas of acute inflammation of variable extent.

The *apical cysts* are further divided into 'true cysts' and 'pocket cysts'. The *true apical cyst* is a periradicular inflammatory lesion with a distinct pathologic cavity completely enclosed by an epithelial lining, without any communication with the root canal. The *pocket apical cyst* is a sack-shaped apical inflammatory lesion, with the epithelium-lined cystic lumen open to and continuous with the root canal. In both entities the cystic cavities may appear half empty or may contain clusters of cells in various stages of degradation, as well as necrotic material and in some cases cholesterol crystals. PMNs may be sometimes abundant in the cyst lumen. The cystic walls, composed of a stratified squamous

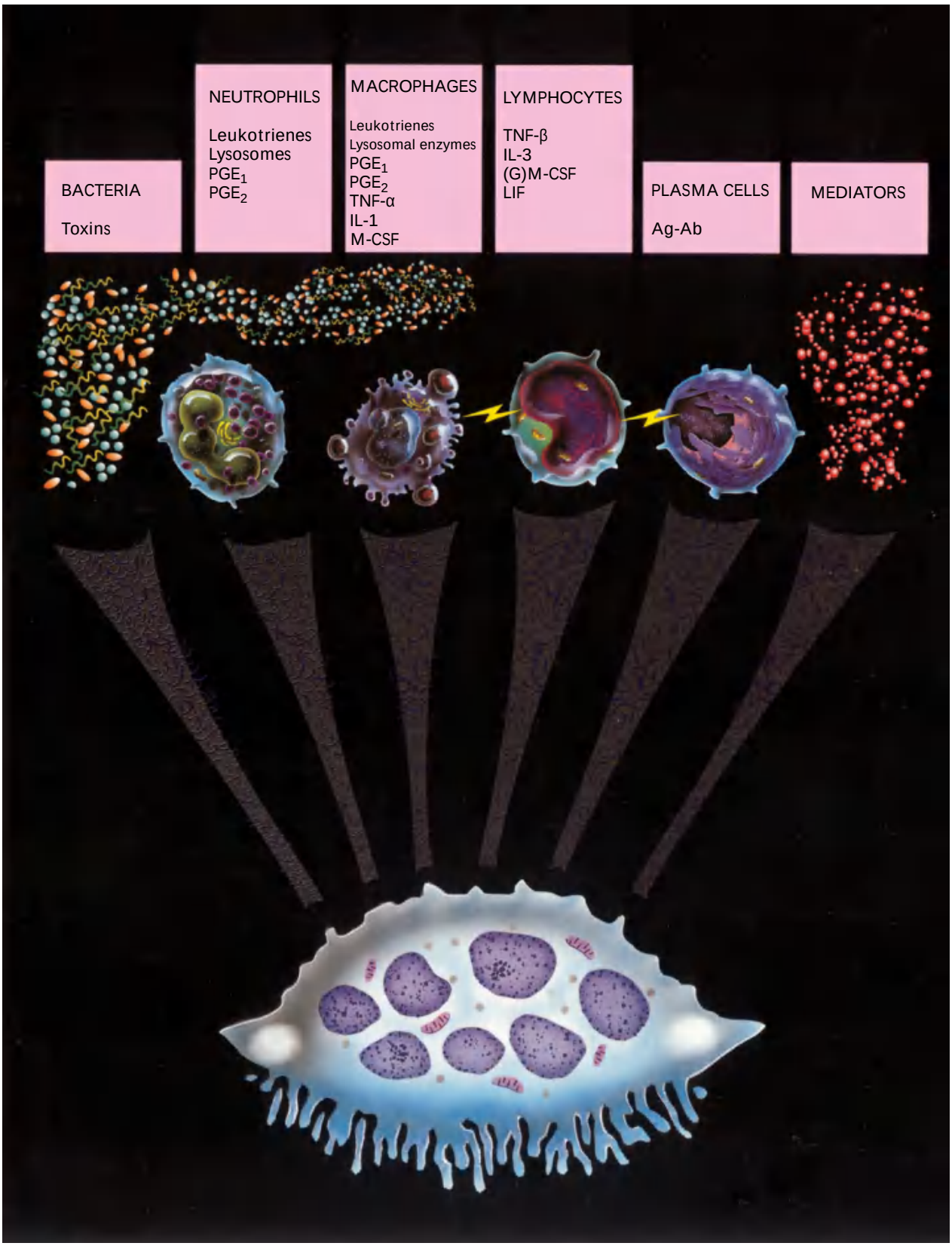



**Fig. 2.30** Osteoclast activators released by bacteria and the associated inflammatory response. Ag-Ab, antigen-antibody; (G)M-CSF, (granulocyte) macrophage colony-stimulating factor; IL, interleukin; LIF, leukemia inhibitory factor; PGE, prostaglandin; TNF, tumor necrosis factor.

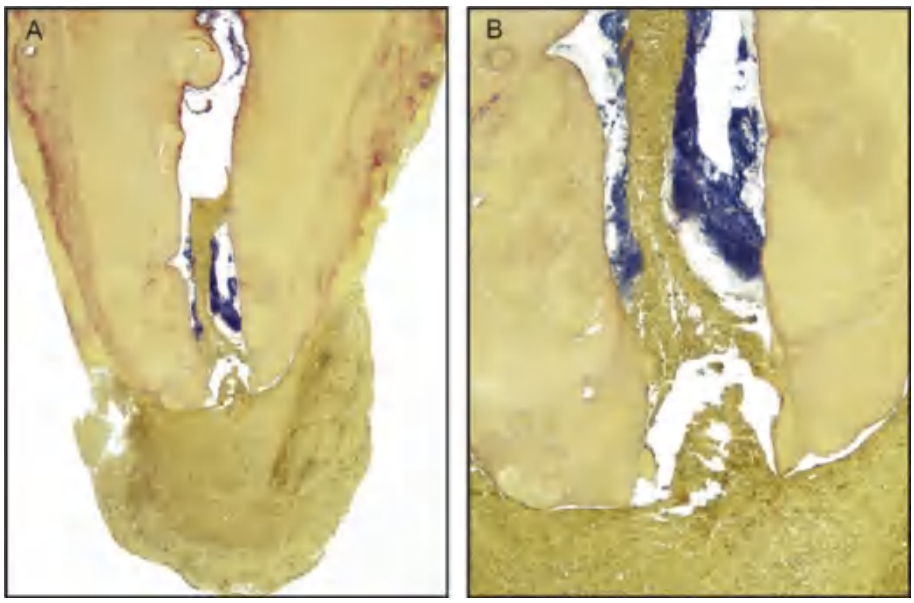



**Fig. 2.31** Mandibular canine with necrotic pulp and a periapical lesion. A. Section cut through main canal and foramen. A biofilm is present in the apical canal. Taylor's modified Brown & Brenn, original magnification ×16. B. Detail from the apical canal. The biofilm is enmeshed with inflammatory tissue. Original magnification ×50.

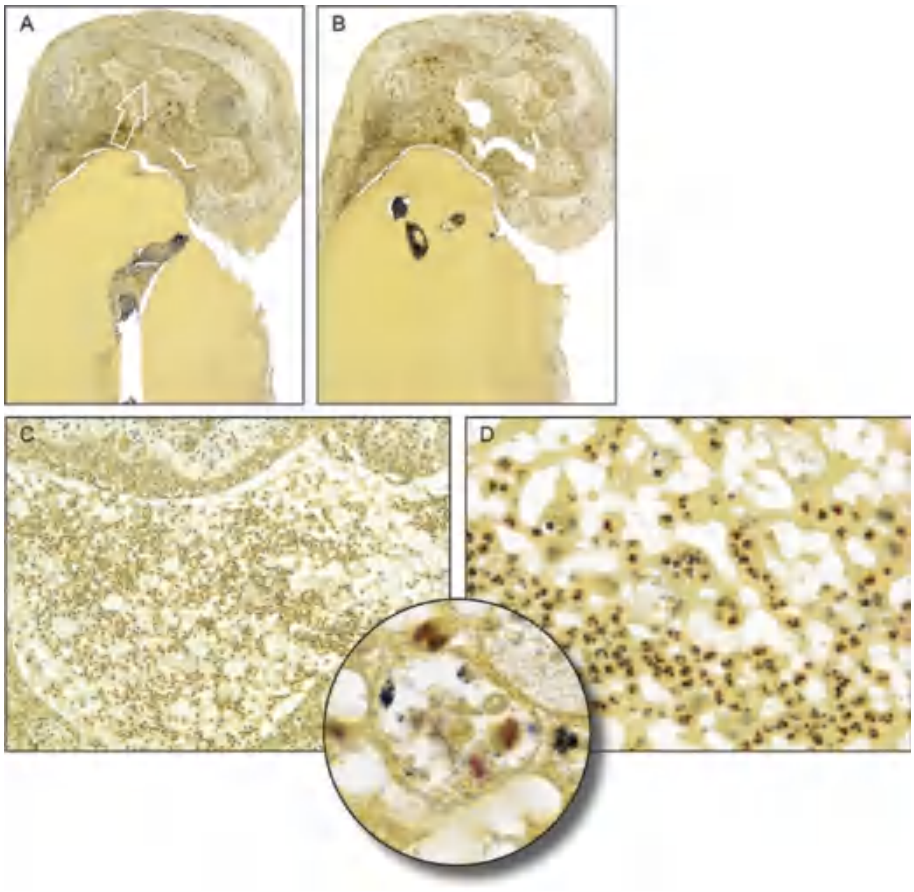



**Fig. 2.32** Distobuccal root of a maxillary first molar with apical periodontitis. A. Section encompassing the main canal and an anticipated foramen. The apical canal is clogged with a bacterial biofilm. A cyst cavity is present in the body of the lesion, apparently with no communication with the root canal space. Taylor's modified Brown & Brenn, original magnification ×16. B. Section cut 80 sections after that shown in (A). Apical ramifications harboring thick bacterial biofilms can be seen. Original magnification ×16. C and D. Progressive magnifications from the area of the cyst cavity indicated by the arrow in (A). Several microcavities and severe concentration of PMNs can be seen. Original magnification ×100 and ×400. Inset, high power view shows scattered bacterial cells in the microcavities. Original magnification ×1000.

epithelium, are surrounded by bundles of connective fibers organized to form a pseudo-capsular structure (Fig. 2.32).

The osteoclastic activity responsible for resorption of periapical bone and the subsequent expansion of the developing granuloma is most likely the result of the combined action of breakdown products of arachidonic acids (i.e. prostaglandins, thromboxanes, leukotrines and lipoxines) from phospholipids of cell membranes from leukocytes, macrophages and platelets (340, 348–359). Cytokines released by activated lymphocytes and plasma cells (352–362) include immune-complexes released by B lymphocytes (349) and bacterial toxins (e.g. lipopolysaccaride, muramyl dipeptide and lipoteichoic acid) (363–371) (Fig. 2.30). These events are described in detail in Chapter 7.

Periapically, the long-term effect of chronic infection is a change in the bone marrow from predominantly fatty to

predominantly fibrous (372, 373). An occasional finding is periapical osteosclerosis, whereby bone marrow is replaced by thickened spongiosa, resulting in a radiodense area (374, 375). The latter reaction is assumed to be the response to a low-grade irritant (infection) in the area. Supporting this hypothesis is the finding that osteosclerotic lesions often disappear when proper endodontic therapy is instituted (376, 377).

The clinical condition known as *acute apical abscess* is the response to invasion by virulent bacteria or immune complexes in the periapex (287, 332). The acute apical abscess is the most common form of extraradicular infection and represents a temporary condition. Even if treatments are not initiated, host defense mechanisms can usually control bacteria egressing into periapical tissues, and the extraradicular infection is terminated (630, 631, 658). Histologically, there is an accumulation periapically of polymorphonuclear leukocytes and disintegration of the PDL. Adequate bacterial staining show clusters of bacteria scattered in the periradicular inflammatory tissue (Fig. 2.32). A high power view may disclose bacteria or bacterial fragments phagocytosed in the cytoplasm of neutrophilic leukocytes (Fig. 2.32 inset). More peripherally, there is intense osteoclastic activity to remove periapical bone in order to provide space for the granulation tissue required to combat infection (333).

In jaw fractures, acute infection in the line of the fracture has been found to be related to a number of different factors, which are described in Chapter 21.

Mounting evidence indicates that apical periodontitis can be included in the set of the biofilm-induced diseases, like caries and periodontal disease (645–647). In a light microscopic study on human teeth with apical periodontitis lesions, the overall prevalence of biofilm was 77%. All root canals associated with very large lesions (>10 mm) were found to harbor intraradicular biofilms. The prevalence of biofilm was found to be high in epithelialized lesions (93%) and cysts (95%) (636).

## Pulp-dentin complex

The functions of the pulp-dentin complex are multiple. After tooth development is accomplished the pulp-dentin organ is able to preserve a vital and rigid structure with flexibility which resists the challenge of repeated mechanical forces. At the same time it maintains an important neural sensitivity, and it preserves the ability to produce reparative dentin against noxious stimuli, such as trauma, attrition, abrasion, the progression of caries and preparation.

Site-specific changes of the pulp-dentin complex are seen with age. The number of odontoblasts decreases, leading to uncovered gaps on the dentin wall. However, continued dentin formation indirectly causes a tendency of crowding and accumulation of collagen fibers (31). Dentin continues to be laid down in vital teeth as secondary dentin and tertiary dentin in response to physical impacts, with a consequent obliteration of the pulp chamber, especially in the crown, which proceeds apically.

### Dentin

Dentin consists of a mineralized organic matrix dominated by a collagen lattice skeleton traversed by dentinal tubules. Dentin contains numerous polypeptides and signaling molecules placed in a mineralized matrix (378–381). The exposure and release of these factors by a direct trauma to the tooth (e.g. intrusion or lateral luxation) or a root resorption process penetrating into the dentin may have a potential effect of influencing the activity of a number of cells (e.g. cementoblasts, periodontal fibroblasts and osteoblasts).

#### *Response to trauma and infection*

Any deviation in the composition of the organic structure of dentin may lead to fracture. Thus, teeth in patients suffering from dentinogenesis imperfecta, with its inherent defects in the collagen matrix, have a high risk of tooth fracture (382–384). Furthermore, exposure of dentinal tubules during trauma leads to bacterial invasion with a resultant permanent or transitory inflammatory reaction in the pulp (385, 386, 619). Finally, a weakening of the dentin under physical loads may occur due to early pulpectomy in immature teeth (387, 388). This may be intensified if inflammatory resorption weakens the root structure, especially in the cervical region (388).

The portal of entry of bacteria in the pulp space of teeth affected by trauma deserves particular attention. A series of studies performed in the early 1970s in anterior animal and human teeth affected by severe attrition, showed that bacteria colonize the cracks extending into dentin, causing pulp inflammation (637–639). Successively, in an *in vitro* study using extracted human teeth, it has been found that enamel-dentin cracks induced experimentally allowed bacteria propagation to the pulp cavity (389). Based on these findings it was suggested that the enamel should be covered with unfilled resin in a replantation situation. However, experimental studies in dogs did not show that this approach improved the chance of pulp revascularization (390). The histobacteriologic aspects of dentin cracks have been extensively investigated in a recent study performed in human teeth (640). Enamel and dentin cracks are always colonized by bacterial biofilms; as a consequence dentinal tubules are invaded by bacteria, especially when the crack extends perpendicularly into the dentin (Fig. 2.33). This study provided evidence that cracks are routes of bacterial entry to the pulp space and can predispose the pulp to severe inflammation and necrosis (640). Clinically undetected enamel and dentin cracks have to be regarded as the most common route for bacterial entry in teeth that undergo pulp necrosis following trauma that does not cause crown fracture and pulp exposure.

### Pulp

The pulp is a highly specialized, loose connective tissue with a specific response to traumatic injuries, as well as to bacterial insults (Figs 2.31 and 2.34). The predominant cells in the pulp are the fibroblasts, which appear as spindle-shaped or stellate cells with oval nuclei and are dispersed

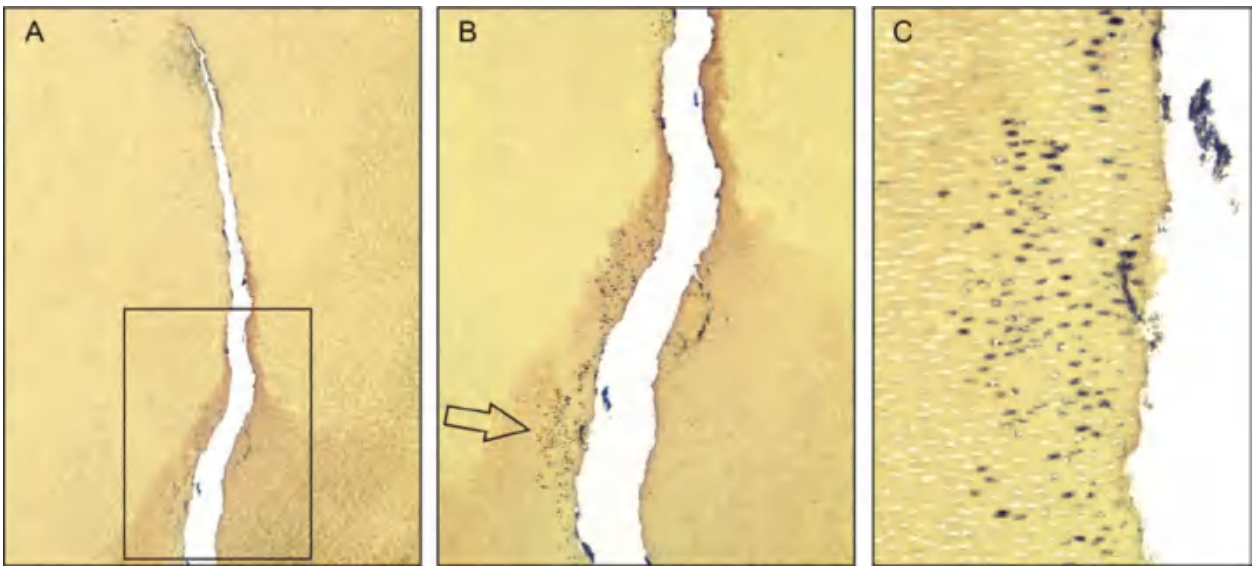



**Fig. 2.33** Crack in a maxillary premolar photographed at its termination in the dentin. A Taylor's modified Brown & Brenn, original magnification ×16. B. Magnification of the area demarcated by the rectangle in (A). Biofilms adhering to the cracked dentin. Original magnification ×100. C. High power view of the area indicated by the arrow in (B). Heavy bacterial colonization of dentinal tubules where the crack runs perpendicular to their course. Original magnification ×400.

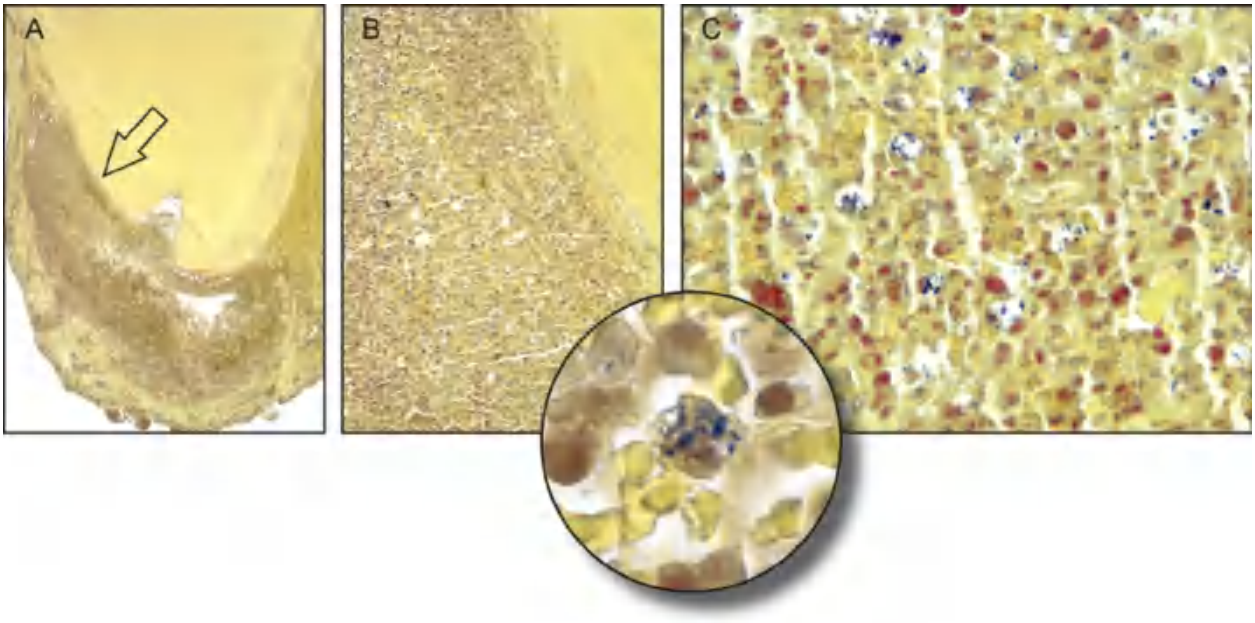



**Fig. 2.34** Distal root of a mandibular molar extracted while a clinical abscess was present. The periapical pathologic tissue remained attached to the root tip at extraction. A. Section passing through the apical foramen. Taylor's modified Brown & Brenn, original magnification ×16. B. Magnification of the area indicated by the arrow in (A). Microcavities appear in the inflammatory tissue. Original magnification ×100. C. High power view showing bacteria scattered into these cavities. Original magnification ×400. Inset, one inflammatory cell with the cytoplasm engulfed by bacteria or bacterial fragments. Original magnification ×1000.

uniformly throughout the pulp, except in the subodontoblastic layer. Undifferentiated mesenchymal cells are located paravascularly and can be recognized by their stunted or rounded form and their few, short processes. These cells probably play an important role in pulp repair (391), and they probably include resident and migrating pericytes (see Chapter 5) (Fig. 2.32).

Odontoblasts are elongated cells subjacent to the dentin. They have a polarized nucleus and processes that extend some distance into the dentinal tubules (392, 393) (Fig. 2.35). Their appearance varies from the coronal to the apical aspect of the pulp, being columnar coronally and flattened on the furcal wall and apically. Moreover, their appearance is also related to their functional stage (i.e. preodontoblasts, secretory, transitional or aged) (394). Odontoblasts have many adhesion molecules, for example for attachment, phenotype specification, gap junctions used for intercellular communication between

cells (395), and extracellular contacts with unmyelinated nerve fibers (396).

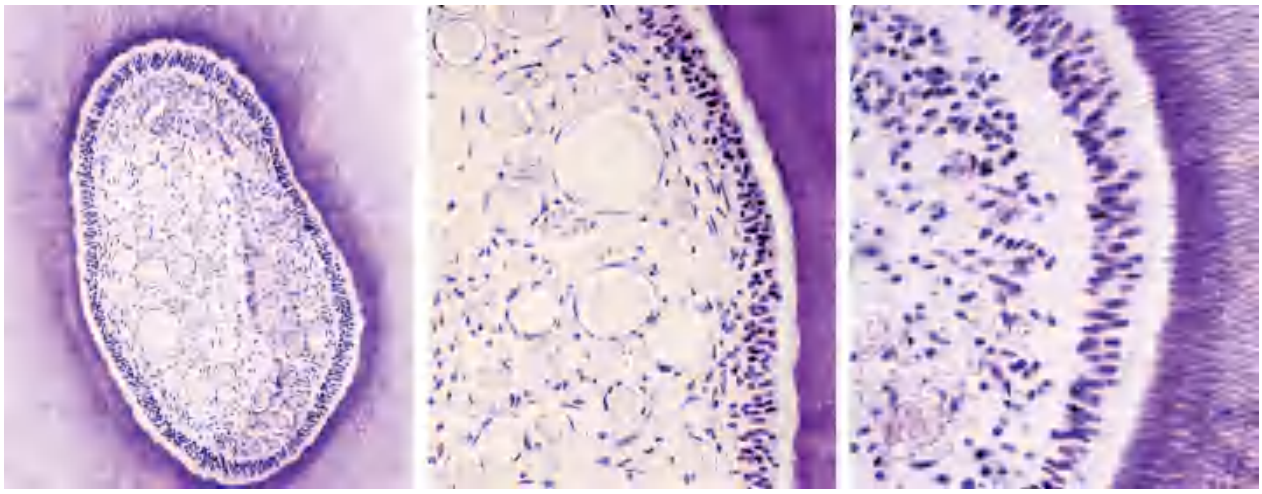
In the crown, a cell-free layer (i.e. the subodontoblastic layer or zone of Weil) exists that contains a network of nerve endings and vessels which, to a lesser degree, is also found in other parts of the pulp.

The cellular activities of odontoblasts are very complex. Beyond secretion of the organic matrix of predentin and control of calcium and phosphate ion transfer (395), the odontoblast also may release proteinases and degrade organic matrix. The average production of primary human dentin appears to be 3 μm per day during tooth formation and eruption (397). When eruption is complete, dentin formation decreases in the pulp chamber. In the root, production continues unchanged until root formation is complete, at which time dentin production is decreased to an extent that is not measurable over a 3-week period. Dentinogenesis, however, can be reactivated by external stimuli, such as dental caries, attrition and dentin fracture and luxation injuries. The production of new dentin can then rise to a level of that of primary dentin (397, 398).

### *Pulpal vasculature*

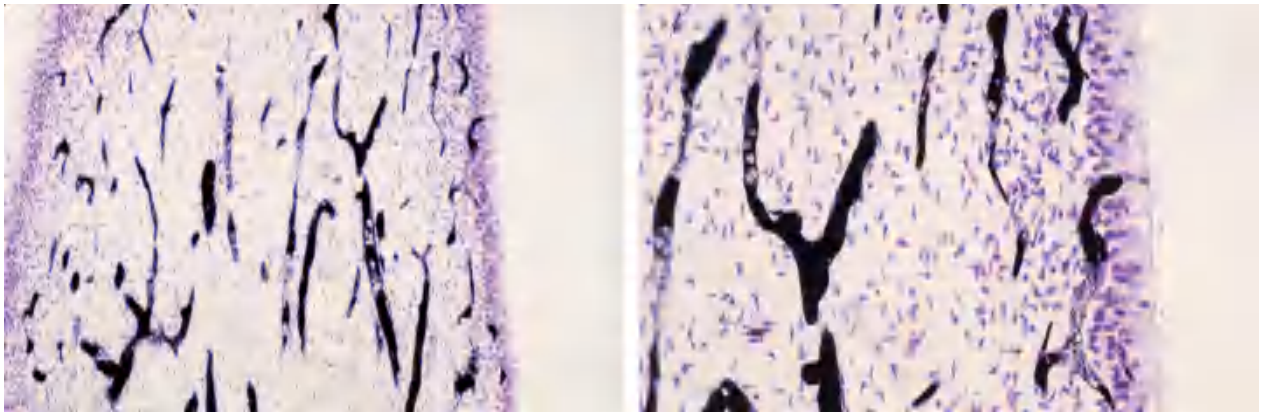
The vascular supply to the immature human dental pulp consists of multiple thin-walled arterioles, venules and veins passing through the apical foramen (397–401). In the mature tooth, vessels can also enter the tooth through lateral canals. The arterioles and venules run parallel to the canal walls up to the pulp horns (Fig. 2.36). En route, they give off branches to form a dense peripheral capillary plexus in the subodontoblastic layer. A few loops are also found between the odontoblasts (399–403) (Fig. 2.36).

Within the pulp, especially apically, there are many arteriovenous connections (shunts) which facilitate and regulate blood flow (404, 405). These shunts are important in the control of tissue pressure. When, during the initial inflammatory reaction, several vasoactive agents are released



A B C

**Fig. 2.35** Anatomy of the pulp. A. Horizontal section of a pulp from a maxillary permanent monkey incisor. Root development is complete. Note the distribution of the larger vessels and nerves. B and C. The odontoblast layer and the subodontoblastic cell free zone. ×40 and ×100.



A B

**Fig. 2.36** Pulp vascularity. A. Vascular pattern in the pulp of a permanent monkey incisor as revealed by India ink perfusion. B. Note the dense network of capillaries next to the odontoblastic layer. ×100.

(see Chapter 1), these agents cause edema and an increase in tissue pressure. When this pressure exceeds that of the venules, a decrease in blood flow results. The arteriovenous anastomoses in the apical part of the pulp dilate in this situation and carry the blood away from the injury zone (403).

The number of vessels entering the apical foramen appears to a certain degree to be related to the maturity of the tooth. However, the density of the vessels is not in direct proportion to the constriction of the pulpo-periodontal interphase (406).

### *Pulpal nerves*

The pulpal nerves generally follow the course of the blood vessels (Fig. 2.35). The nerve fibers enter the pulp in multiple bundles that contain both mylinated and unmyelinated axons (405). The majority of the axons (70–80%) are unmyelinated. Both types terminate as free nerve endings. These endings are receptors which respond to various external stimuli, environmental changes or inflammatory mediators under inflammatory conditions. They respond, for example, as nociceptors to mechanical forces and thermal changes. The number of myelinated fibers increases with tooth maturity and, at the same time, the threshold for electrometric pulp stimulation is lowered (407–411).

The innervation of the pulp–dentin border is extensive. A dense network of fine nerve filaments, known as the plexus of Raschkow, is formed close to the odontoblasts. A number of nerve terminals also enter the odontoblast layer and many of them extend into the dentinal tubules. A part of the axons also terminates in the deeper parts of the pulp, and they may have a significant role in sympathetic regulation of blood flow response to trauma, as well as in regulation of inflammation and repair (412, 413). Unmyelinated sympathetic nerves from the autonomic nervous system are associated with vasocontrol, and sympathetic impulses lead to constriction of arterioles (412). The absence of nerve fibers in feline teeth has been found to lead to accelerated secondary dentin formation (547).

### *Tertiary dentin: reactionary or reparative*

Extensive physical trauma may result in the formation of mineralized tissue. There is a strong relationship between traumas with their lesion environments, and different corresponding formations of *tertiary* dentin. After injury to the mature tooth, the fate of the odontoblast varies according to the intensity of the injury.

Milder injury can result in a stimulation of the involved odontoblastic cells leading to *reactionary* dentin formation. Upregulation of functional activity in primary surviving odontoblast cells leads to focal secretion of a reactionary dentin matrix in the dentinal tubules and predentin (414). Obturation of dentinal tubules are biologic responses that compensate for the loss of tissue.

Greater injury can lead to odontoblast cell death. Induction of differentiation of a new generation of odontoblast-like cells can then lead to *reparative* dentinogenesis. Secretion of matrix from a new generation of cells implies a discontinuity in tubular structure with subsequent reduction in dentin permeability. The initial repair response often represents a bone-like tissue (i.e. osteodentin) in the pulp chamber. The non-specific response leads to deposition of atubular dentinal matrix covered by cuboidal or polygonal preodontoblast-like cells, and inclusions of osteocyte-like cells are observed in a dense mineralizing matrix called osteodentin. Deep to the pulp injury, surviving post-mitotic odontoblasts respond with deposition of reactionary dentin along the walls. In such situations we observe a reactionary dentin matrix with less tubular density than in the primary dentin. The dentinal tubules in tertiary dentin often represent a mixture between reactionary and reparative dentinogenesis (413, 641, 655).

Odontoblast differentiation has been covered in an excellent review article by Ruch et al. in 1995 (656).

### *Response to trauma*

#### Repair after pulp exposure

The pulp-dentin organ must respond to a spectrum of traumatic events, such as exposed dentin due to fracture with subsequent bacterial invasion into the tubules (415). The pulp can be directly exposed to bacterial contamination from saliva following a complicated enamel-dentin fracture. Another, and usually sterile, exposure occurs during root fracture, when the pulp is exposed to the PDL via the fracture line. Finally, the pulp may become partly or totally severed and sometimes crushed at the apical foramen or at the level of a root fracture during luxation injuries. These different traumatic insults, all of which interfere with the neurovascular supply to the pulp, give rise to various healing and defense responses ranging from localized or generalized tertiary dentin formation to pulpal inflammation, internal resorption and bone metaplasia, as well as pulp necrosis with and without infection (416).

A general feature of the pulpal wound healing response is the replacement of damaged tissue with newly formed pulp tissue. This can occur along the pulp–dentin border if localized damage has been inflicted on the odontoblast layer (e.g. after dentin exposure), along an amputation zone in the coronal part of the pulp, or as a replacement of the major parts of the pulp if it has become necrotic because of ischemia (e.g. after a luxation injury). A common denominator of these different events is *replacement of pulp tissue* by the invasion of macrophages, new vessels and pulp progenitor cells in the injury zone, whereby the traumatized pulp tissue is gradually replaced by new pulp tissue. The exact nature of this process is only partly understood at present (416, 417).

The character of the pulpal wound healing response varies according to the origin of the progenitor cells involved (see Chapter 5). Thus, in the case of PDL-derived progenitor cells, PDL tissue will be formed with associated cementum deposition along the root canal walls. Moreover, if there is a patent apical foramen, periodontal stem cell progenitors may invade the root canal accompanied by bone formation and inserting Sharpey's fibers.

Formation of bone in the pulp subsequent to trauma is a known phenomenon (659–665). The characteristic appearance appears to be an extension of a cone of bone into the pulp cavity with a PDL-like separation line between bone and the canal wall (659, 668). Histologic examination of extracted teeth demonstrating this healing event has shown development of an 'internal periodontal ligament' separating the bone cone from the canal walls (666–668). Very little is presently known about the frequency and pathogenesis of this healing event. In two experimental studies using tooth extraction and subsequent replantation in monkeys it was demonstrated that removal or damage to the HERS resulted in invasion of bone and PDL in the canal (667). Furthermore, marked inadequate repositioning in cases of immature root formation could lead to bone and PDL invasion (666). Today, very little knowledge exists about the frequency and pathogenesis of this type of healing in humans. This fact is of concern, as the recent approach to pulp 'regeneration/revitalization' often implies that bone or bone-like tissue is becoming the new pulp canal tissue (669–672) (see Chapter 26). How that tissue will behave over time is presently under investigation (628).

It has been found that if an accidental exposure of the pulp is left untreated, even over a period of 1 week, inflammation is superficial and limited to a depth of 2 mm. It can be seen at that time that the pulp tissue has proliferated through the exposure site (see Chapter 25) (418, 419). In cases where the entrance to the exposure site is covered with a suitable capping material (i.e. which limits or prevents bacterial contamination), a hard tissue barrier is normally established (420, 421, 510). The cells responsible for the formation of this dentin bridge are apparently not odontoblasts, but most probably include mesenchymal cells located paravascularly, that subsequently differentiate into odontoblasts (392, 422–426). This has been shown very convincingly in a study where the odontoblast population was eliminated in rats by colchicine administration. After 3–5 days, revascularization and cell proliferation into the necrotic cell layer adjacent to the predentin were observed. New odontoblasts seemed to develop from paravascular cells and, after initial formation of a non-mineralized collagenous matrix, tubular dentin was formed (427).

The wound healing response to pulp capping has been discussed in several survey articles (429, 430). The ability to form a hard tissue bridge has been found to take place with a wide range of materials (428).

In the stimulation for a hard tissue bridge, the formation of a necrosis and degenerative zone by the capping material appears to be of importance (431–434). It has been speculated that this zone may mimic a basement membrane and serve as an attachment surface for components that induce odontoblasts and extracellular matrix production (e.g. fibronectin and transforming growth factor β1) (428, 435, 436).

#### Pulp healing after coronal exposure and capping

The events taking place after pulp wounding can be divided into the phases of hemostasis, inflammation, proliferation and remodeling. Wound healing is, however, a continuous process where the beginning and the end of each phase cannot be clearly determined and phases overlap (440). The observed sequence of initial pulp reactions is that which is expected when connective tissue is wounded.

Fibrinogen exudation takes place under the capping material in the pulp tissue for up to 4 days (441). After approximately 3–6 days the inflammatory infiltration is replaced by a migration of granulation tissue originating from central pulp sites. The granulation tissue is arranged along the wound surface and consists primarily of newly formed fibroblasts and capillary blood vessels which proliferate and grow into the damaged tissue. Layers of fibroblasts increase in thickness around the lesion. Synthesis of new collagen fibers along the tissue necrosis is detected from 4 days after application of pure calcium hydroxide. Cells surrounded by new matrix including calcifying nodules are found after 7 days (442). The initial precipitation of minerals is associated with the detection of matrix vesicles, indicating a close similarity to mineralization in bone (443). The minerals are found to originate from the blood supply (444). After 11 days the new matrix is associated with cuboidal cells, and some cells with odontoblast-like differentiation. After 14 days a clear odontoblast-like arrangement is observed (445). After 1 month, dentin bridges can be seen around the trauma, which represents a defensive interface between the potential necrotic remnants and the new odontoblast layer (446). Microscopic evaluation, however, revealed 89% of all dentin bridges contained tunnel defects (447).

The pulp healing response replaces injured tissue with newly formed tissue. This can occur along the pulp–dentin border if localized damage has been inflicted on the odontoblast layer (e.g. after dentin exposure), along the wound surface in the coronal part of the pulp, or as a replacement of the major parts of the pulp if it has become necrotic because of ischemia (e.g. after a luxation injury) (416). However, histologic studies in human teeth suggest that the pulp-dentin organ cannot be restored to its original structure either following direct pulp capping (641) or regenerative endodontic procedures (653–656).

## Biologic effects of calcium hydroxide

Calcium hydroxide-containing agents have been widely used for vital pulp capping (442, 448, 449) and the wound healing response to pulp capping has been described in several survey articles (428–430).

The effect of calcium hydroxide materials on exposed connective pulp tissue has for decades been studied in experimental animals (450–453) as well as in humans (442, 454–456). As experimental animals, the rat has been commonly used (453, 457–462), but dogs (458, 459, 463, 464), pigs (452, 458) and primates (450, 451, 457) have also been used. In comparative studies the conclusion has been that there is no significant difference in the reparative pulp response to calcium hydroxide between the different animal models (458–460). The ability to form hard tissue has been found to take place with a wide range of materials (428), irrespective of their pH (431).

The strong alkaline pH of $Ca(OH)_2$ contributes to its action. In the stimulation for a hard tissue bridge, the formation of a necrosis and degenerative zone by the capping material appears to be of importance (432–434). Pulp capping using calcium hydroxide also induces apoptosis in the underlying pulp. Apoptosis is a non-inflammatory, controlled cell death mechanism, whereas necrosis induces a pro-inflammatory response (466, 467). The balance of apoptosis and necrosis after pulp capping, therefore, may influence the subsequent inflammatory response.

The high pH of calcium hydroxide appears to cause local necrosis of the pulp tissue. $Ca(OH)_2$ may thereby, due to its pH effect, prevent bacterial infection and provide a bactericidal environment in which subsequent repair can occur. A few hours after application of calcium hydroxide on exposed pulp tissue an initial necrosis is created and inflammatory cells migrate towards the lesions, and the inflammation lasts for a few days (468).

The signaling processes responsible for $Ca(OH)_2$-induced odontoblast-like cell differentiation remains to be clarified (468). It has been suggested that $Ca(OH)_2$ is able to solubilize bioactive molecules with appropriate signaling functions from the dentin matrix (469).

There has been much speculation about which factors are of importance for a successful outcome of calcium hydroxide treatment of pulp tissue (470). Efforts have been made to find a formula that minimizes the proinflammatory actions, and at the same time stimulates dentin bridge formation (468, 471, 472).

Apparently, the rather abnormal appearance of the initial matrix of the dentin bridge, which is usually of the osteodentin type, is followed by formation of tubular dentin. During later bridge formation, the matrix structure often becomes more regular, resembling true tubular orthodentin (68).

It has been claimed that the size of the defect, the degree of inflammatory reaction, the control of bleeding following surgical treatment, the amount of clotting, the amount of dentinal chips formed during preparation, and the degree of immediate contact between tissue and agent, as well as the formulation of the calcium hydroxide material, may all play a key role (447, 472–474). Within the last decades several new capping materials have been introduced.

## Biologic effects of new pulp capping or pulpotomy materials

Calcium hydroxide compounds have throughout the years been the golden standard of capping materials, but several other materials have also been suggested (437).

*Resin adhesive materials* used for pulp capping have had supporters based on experimental results (439, 475, 476, 482). The aim of using adhesive materials has been to minimize microleakage and to thereby stimulate bridging. Recently, criticism has been raised concerning their use, based on an analysis of human teeth treated with various resin adhesive systems showing poor results (438, 477, 478) (Fig. 2.37). Studies *in vitro* suggest components of the resin may inhibit odontoblast differentiation (614).

Mineral trioxide aggregate (MTA; also called calcium silicates) used for pulp capping material produces a hard tissue barrier (480, 481). The composition of MTA is very similar to Portland cement and as such the material sets in a moist environment. During the setting reaction calcium hydroxide ions are released and the formation of apatite is observed in the exposed area. Histologic studies in animals have shown a high sealing ability and hard tissue-inducing capacity (479, 482–486). Proroot MTA used as a capping material was found to result in significantly less inflammation than calcium hydroxide (Dycal®). This material has furthermore been recommended for the repair of root perforations and as a retrofill material (487, 488). At present, calcium silicate appears to be a promising candidate as an alternative to calcium hydroxide. The clinical use of this material is described in Chapter 25.

Dentin contains numerous polypeptides and signaling molecules which may be released following a trauma. Among the growth factors isolated the following should be mentioned: insulin-like growth factor (IGF), transforming growth factor (TGF), platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), epidermal growth factor (EGF) and bone matrix protein (BMP) (489). This is of importance as all of these substances have also been found to be involved in bone and PDL healing processes in animals (see Chapter 1).

*Bioactive substances* such as calcium silicate appear to represent a new way to stimulate the formation of dentin bridges (489–497). Tissue injury leads to alterations in gene expression and the release of a range of cytokines including growth factors. Cytokines play a determinant role in regulation of cell proliferation, migration and differentiation during pulp healing. In particular, members of the TGF-β family have been implicated in dentin matrix formation (489–500). Nearly half of the TGF-β1 in dentin matrix has been reported to be present in an active form (506). However, TGF-β activity decreases with a short half-life (2–3 minutes) (503) due to the binding of active TGF-β to extracellular matrix (504).

A number of growth factors such as BMP, TGF-β1, IGF, FGF, PDGF and EGF have been tested in animal models for their capacity to stimulate dentin bridge formation (489–497, 501, 502). Among these, TGF-β enhances dentinal matrix formation (497, 504–506). BMP-7 has been found to stimulate reparative dentin formation in rats (507), ferrets (509), pigs (508) and monkeys (508). BMP-7 failed to work in pulps with induced inflammation (511). The mechanism whereby ongoing inflammation inhibits dentin bridging remains to be elucidated (451, 512). IGF-1 (493) and BMP (513) in rats, and PGDF-11 in dogs (514) have also been found to stimulate dentin bridge formation (513–515). A matter of concern in evaluating the many experimental studies performed in rats, dogs, pigs and monkeys is that animal models do not always correlate very well with human studies (430, 437, 438). This implies that material only tested in animals should be considered with a certain skepticism until clinical studies in humans have been performed.

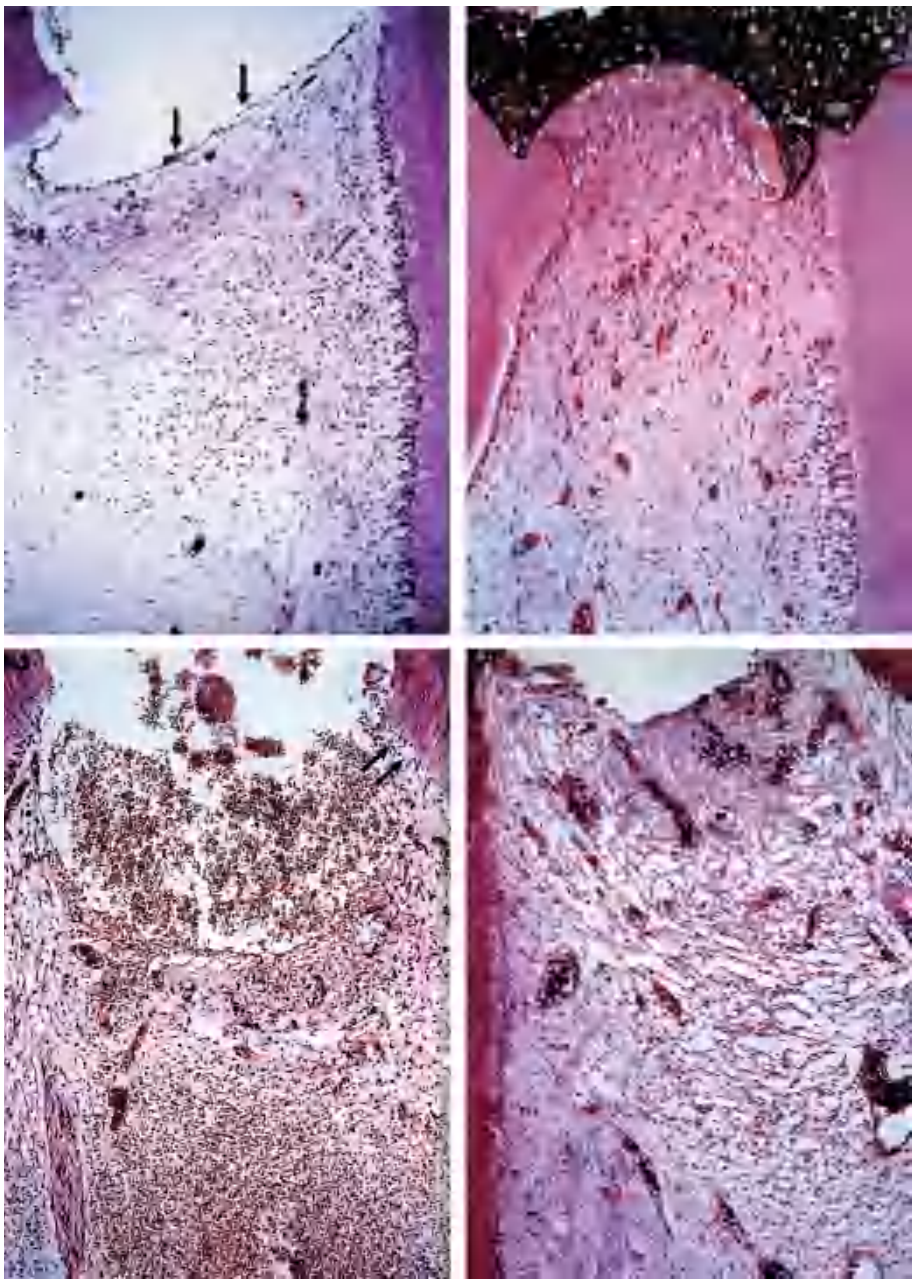



**Fig. 2.37** Pulp response to resin adhesive and calcium hydroxide. A. Human pulp capped with calcium hydroxide. Pulp response after 10 days (left) and 70 days (right). Note the calcified barrier and lack of inflammation (arrows). B. Pulp capping with a dentin bond adhesive. The border zone between resin and pulp is shown. No hard tissue barrier is formed and the pulp tissue is necrotic next to the adhesive (arrows). Reproduced from HÖRSTED-BINDSLEV et al. (477) with permission.

## Anatomy of dentin bridges

The cellular mechanisms in the formation of a dentinal bridge have been reported in several studies (518–521). Almost all dentin bridges formed after calcium hydroxide application show multiple tunnel defects (522–524), and these tunnels may lead to microleakage (522, 523). The tunnel defects have been found not to decrease or be obliterated over time (523). The consequence of this should be that a bacteria-tight seal is established over the bridge (see Chapter 25). Furthermore, one of the requirements for potential new capping materials should be that they create a fast and thick dentin bridge with a minimum of tunnel defects.

Exposure of the dental pulp at the fracture site through a tooth fracture requires formation of another type of dentin bridge. The formation of such a dentin bridge is usually favorable, provided that the pulp is not infected or inflamed.

The prognosis for healing of pulp capping is poor in locations with an inflamed pulp. Pulpotomy should be considered in order to create a wound in an uninflamed location (525).

## Extent of pulpal inflammation related to time of pulp exposure

It has often been cited that the time since exposure of the pulp should dictate the choice of treatment procedure, that is, pulp capping, pulpotomy or pulp extirpation. However, clinical studies in humans do not confirm these statements (526). This may be explained by the nature of the inflammatory process occurring after pulp exposure.

Thus it has been found in monkeys that there was only a moderate inflammatory invasion in the pulp after 1 week (525–528).

## Repair or regeneration of pulp due to luxation injuries or avulsion and replantation

Several clinical studies have shown that a pulp may heal after injuries where a complete severance of the neurovascular supply has taken place (see Chapters 13 and 16). The histologic events during revascularization are also described in these chapters.

Luxation injuries usually imply total or complete severance of the neurovascular supply. The ensuing ischemia affects all cells in the pulp. The healing processes begin apically, move coronally and are highly dependent upon the size of the pulpo-periodontal interface (i.e. stage of root development) (607, 608). The outcome of pulpal severance will be either total pulpal revascularization (Fig. 2.38) or the development of partial or total pulp necrosis, usually determined by the presence or absence of bacteria in the injury zone. Revascularization of the pulp appears to start after 4 days (531), but no experiments have been done to examine exactly when it starts (Fig. 2.38). In some cases of successful revascularization, an intact odontoblast layer will be found with an apparent continuity of the odontoblastic processes (Fig. 2.39). The mechanism of this is speculative, but end-to-end anastomoses in the ruptured apical vascular supply is a possibility (531, 532) (Fig. 2.40).

Vascular alterations and inflammatory cell infiltration are activated in order to eliminate the irritating molecules. Adhesion molecule interactions between blood leukocytes and endothelium enables transmigration of inflammatory cells from inside to outside the vessel wall (533). Failure to resolve inflammation after wounding leads to chronic non-healing wounds (534), and pulp tissue responds similarly with an absence of hard tissue healing. The mechanism whereby foregoing inflammation may inhibit tissue repair (512) and potentially influence gene activity necessary for stem cell recruitment remains to be elucidated.

In the case of an invasion of PDL-derived progenitor cells into the root canal, connective tissue will form in association with cementum deposition along the root canal walls. Dependent on a patent apical foramen, bone may invade the root canal accompanied by inserting Sharpey's fibers.

The source of the repopulating endothelial cells is variable (535). In most cases, osteodentin, bone or cementum-like tissue is formed on the canal walls as a response to the pulpal injury. The reasons for these varying responses have not yet been clarified.

Dentin formation after revascularization is usually very extensive and may soon lead to total pulp canal obliteration. In replanted or transplanted monkey incisors observed for 9 months, it was found that the average daily dentin production was 4 μm (25) (Fig. 2.41). The explanation for this accelerated dentin production is presumably a loss of autonomic and/or sensory nervous control of the pulp tissue (536–549).

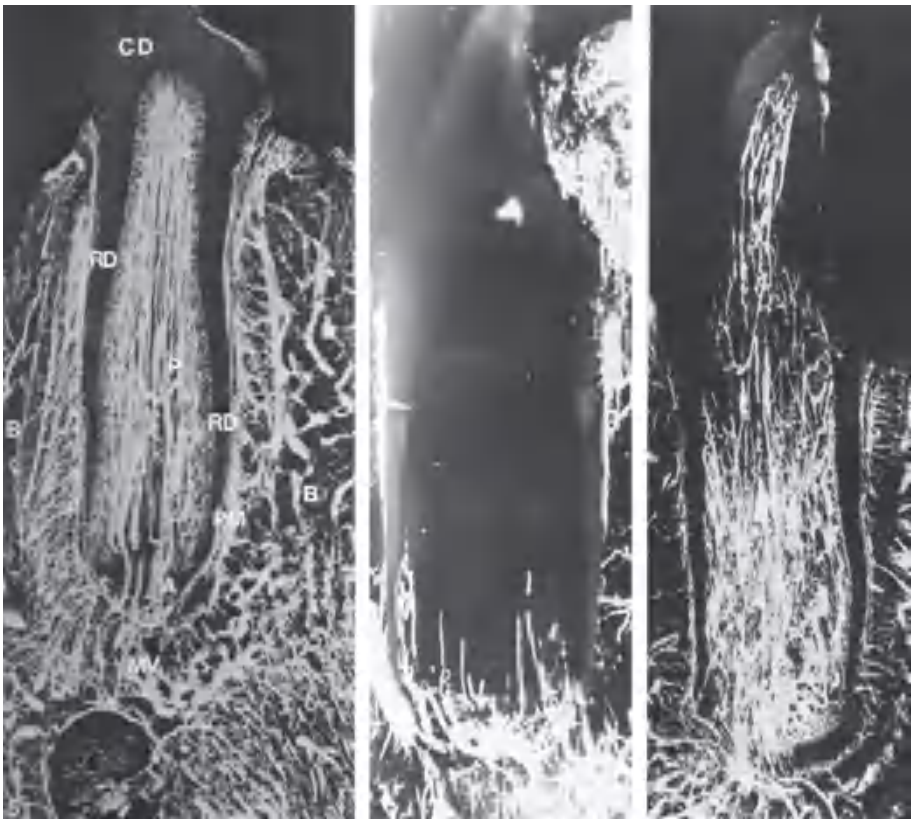
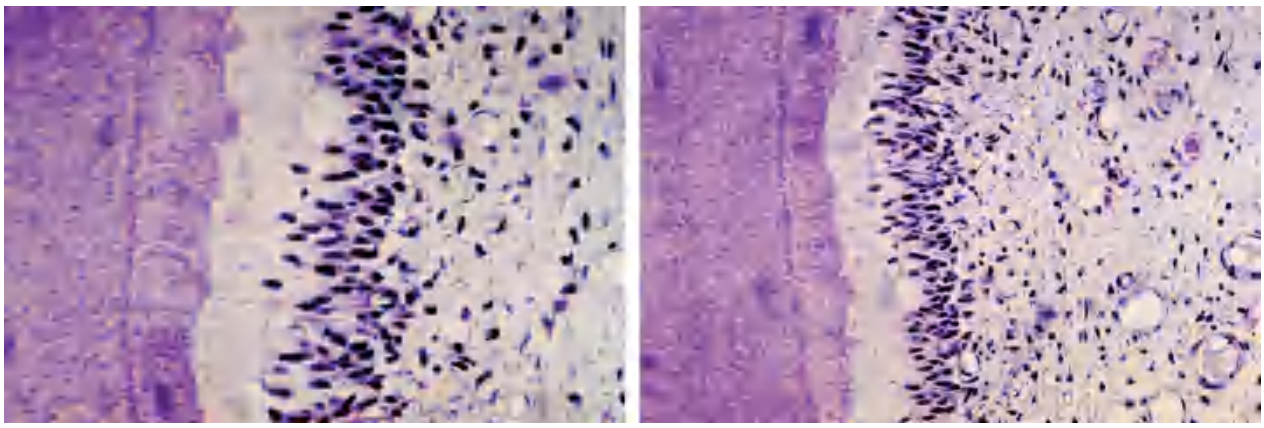
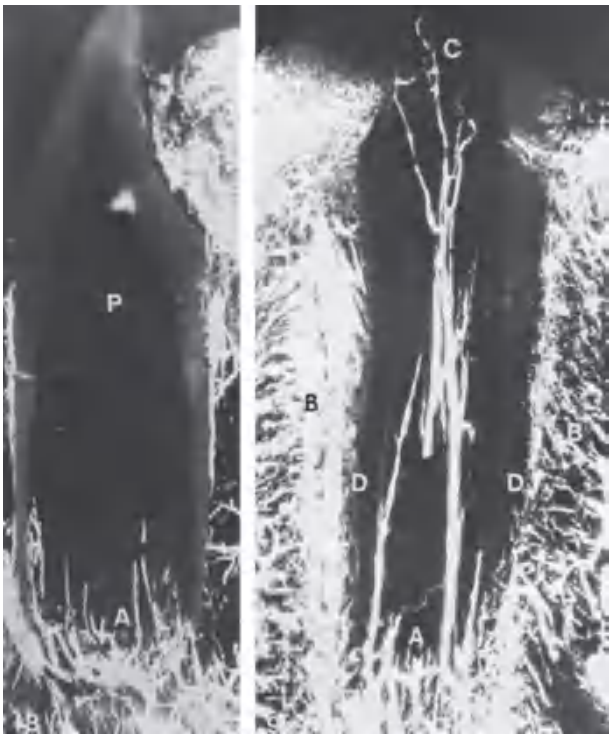



**Fig. 2.38** Revascularization of pulp. Ingrowth of vessels in the pulp after immediate replantation of an immature tooth in a dog demonstrated by a microangiographic technique. A. Control tooth. B. After 4 days, revascularization has begun in the apical portion of the pulp. C. After 3 weeks, revascularization is complete. From (529).



A B

**Fig. 2.39** Continued dentin formation in a replanted permanent maxillary central incisor in a monkey. Note continued dentinogenesis after replantation. Only a slight change in the direction of the dentinal tubules indicates the time of replantation. An average daily production of 2 µm of dentin was formed in the observation period of 8 weeks. A. ×25. B. ×50.




A B

**Fig. 2.40** End-to-end anastomosis. A. Microangiograph of a replanted mandibular third incisor in a dog, 4 days postoperatively. A few tiny vessels are seen in the apical region (A) of the pulp (P). B. Microangiograph of a replanted first maxillary premolar, 4 days postoperatively. Some slender vessels are visible in the apical region of the pulp, but, in addition, a few larger vessels are seen running all the way to the coronal pulp. A, apical region of the pulp; B, alveolar bone; C, crown portion of the pulp; D, dentin. From (529).

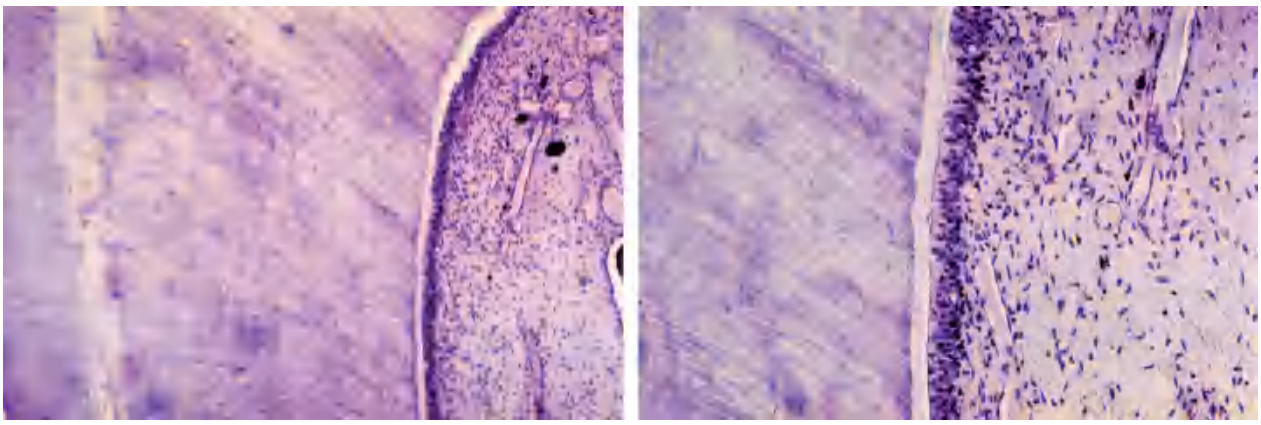
## Dentin response to infection

An untreated dentin fracture is a possible pathway for pulpal infection following bacterial invasion of the dentinal tubules. Pulpal reactions to crown fracture will be further described in Chapters 12 and 25. If the vascular supply to the pulp is intact and the bacterial insult moderate, the event will result in tertiary dentin formation and the pulp will survive (385, 386). However, if bacteria gain access to the pulp via dentin tubules, as in the case of dental caries or a crown fracture, and pulpal vascularity is in any way compromised (e.g. a luxation injury), pulpal healing will not take place (550, 551). This is probably due to impaired defense reactions and/or a loss of the pulpal hydrostatic pressure which presents a powerful barrier to bacterial invasion through the dental tubules (552, 553). As discussed earlier, a dentin crack is also a common portal of entry for bacteria (640) (Fig. 2.33). An alternative route is through the apical foramen via a severed PDL (550, 551) (Fig. 2.42). Finally, a hematogenous route (anachoresis) cannot be excluded (554–562). The explanation for this transmission of bacteria appears to be an increased vascular leakage in the tissue bordering the traumatized pulp tissue (563, 564).

An interesting clinical study in humans has shown that the speed of invasion of bacteria in exposed dental tubules is dependent upon the pulp status. Thus, in endodontically treated teeth, bacteria invasion of up to 2.1 mm was found. In vital teeth, bacteria invasion was significantly less pronounced (565). It was assumed that the dentinal fluid in a vital pulp or the pressure of intact odontoblastic processes may be the explanatory factor (565).

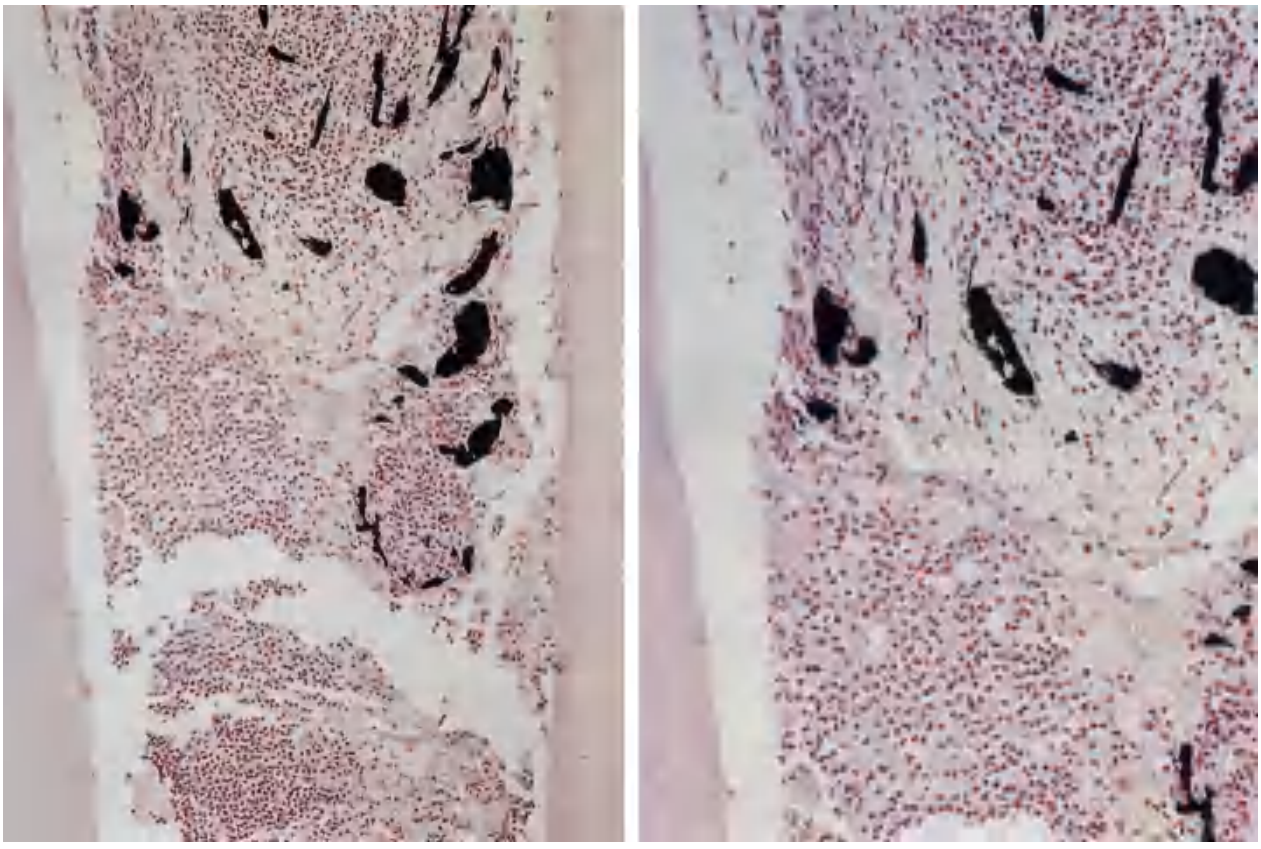
## Pulp response to infection

An interesting finding is that necrotic but sterile pulpal tissue can persist over a prolonged period (years) without becoming infected (332, 566–568). If the pulp becomes infected in its ischemic state, revascularization is apparently permanently arrested and a leukocyte zone is formed which separates infected necrotic tissue from the ingrowing apical connective tissue (82) (Fig. 2.42). In these cases, bacteria are found in the necrotic pulp as well as in the leukocyte zone, but seldom in the adjacent vital connective tissue.



A B

**Fig. 2.41** Rapid dentinogenesis after pulp revascularization. A. Extensive dentin production after autotransplantation of a permanent maxillary central incisor in a monkey. Observation period: 9 months. ×10. B. An intact odontoblast layer is seen forming tubular dentin. The dentin formed immediately after transplantation is acellular. The average dentin production after transplantation is 4 µm daily. ×40.



A B

**Fig. 2.42** Infection in the pulp has caused arrest of pulpal revascularization. Status of a replanted permanent lateral incisor in a monkey. A. Low power view. There is autolysis and infection in the coronal part of the pulp. ×40. B. A leukocyte zone separating the infected part of the pulp from the revascularized part. ×100.

The pulpal response to infection differs from the general response to infection by the end-organ character of the pulp tissue, implying limitations in the initial inflammatory response to infection (i.e. swelling) and a restricted access of the defense system to privileged sites for bacteria, such as the dentinal tubules, lateral canals and vascular inclusions in dentin. These anatomic obstructions usually make complete microbial kill impossible. The best compromise is usually the creation of a barrier against microorganisms, either by granulation tissue, hard tissue (such as secondary dentin or cementum) or proliferation of epithelium (282, 569–571, 642). Obviously, this type of defense system is not absolute

and with a change in the number of bacteria, their virulence or the level of resistance of the patient, an exacerbation of the infection may occur.

The organization of bacteria residing in the necrotic portions of the root canal is crucial for the understanding of the disease process. By histobacteriologic means bacteria may be observed as apparently floating in a fluid phase (planktonic state) or as aggregates (cells with the same morphology) or co-aggregates (cells with different morphologies). However, most often bacteria are observed to form complex structures adhering to the root canal walls (630). In recent years these structures have been referred to as biofilms (636, 644) (Fig. 2.32). A biofilm can be defined as 'a sessile multinuclear microbial community characterized by cells that are firmly attached to a surface and enmeshed in a self-produced matrix of extracellular polymeric substances' (643, 644). The biofilm community lifestyle provides microorganisms with a series of advantages and skills that are not observed for individual cells living in a planktonic state, including: establishment of a broader habitat range for growth; increased metabolic diversity and efficiency; protection against competing microorganisms, host defenses, antimicrobial agents and environmental stress; and enhanced pathogenicity (636, 652).

In histologic sections biofilms appear thick and composed of several layers of cells. Different morphotypes can be seen in the same biofilm: cocci, rods and filaments. The proportion of bacterial cells to extracellular matrix may vary considerably (Figs 2.31, 2.32 and 2.33). Concentrations of polymorphonuclear neutrophils and macrophages are often observed surrounding the biofilms. This observation confirms that bacteria enmeshed in a biofilm structure are protected against phagocytosis.

The clinical relevance of the biofilm structures in endodontic infections is evident. While bacteria present as planktonic cells in the main root canal may be easily accessed and eliminated by instruments and substances used during treatment, those organized in biofilms attached to the canal walls or located into isthmuses and ramifications are definitely more difficult to reach and eradicate (616). Even in the event antimicrobial agents reach the biofilm, this is no guarantee of successful antimicrobial activity, as bacteria arranged in biofilms exhibit increased resistance to antimicrobials (645).

Another important aspect is that many bacteria under the biofilm are seen invading dentinal tubules at varying depths, and this poses additional problems for successful disinfection.

The final stage in the progression of of pulp degeneration is reached when necrosis and infection reach the last portion of the apical tissue. Common features are the presence of a biofilm on the apical canal walls, and granulation tissue, with an abundance of acute and chronic inflammatory cells, proliferated into the apical canal (Fig. 2.31).

Bacterial biofilms have been described in immature teeth with pulp necrosis consequent to trauma. Lin et al. (651) reported on the failure of revascularization/revitalization treatment of an avulsed and replanted immature maxillary incisor. Histobacteriologic analysis of the extracted tooth showed that the majority of bacteria were observed in the apical portion of the canal and formed biofilm on the canal walls and had penetrated into the dentinal tubules (Fig. 2.43).

Biofilms may also be formed in apical ramifications, when present (Fig. 2.32). In cases with longstanding pulp necrosis, biofilm-like structures or calculus can be observed on the external apical surface. It has been reported that in cases that failed to heal after conventional endodontic treatment, histology of the resected root tip revealed bacteria held together by an extracellular matrix (648, 657). Similar bacterial structures were described on the external apical surface of untreated teeth with periapical lesions with scanning electron microscopy (649, 650). In a histobacteriologic study on human teeth with periapical lesions, Ricucci and Siqueira (626, 635, 636) reported that an apical extraradicular biofilm was present in 6% of teeth examined. They described the possible mechanisms by which extraradicular biofilm may mineralize. Saliva circulating from the oral cavity to the apical periodontitis lesion via a sinus tract can be the source of mineral salts. However, apical biofilms can calcify even in the absence of a sinus tract, and the source may be the inflammatory exudate and periradicular tissue fluids saturated with minerals from the solubilization of both bone and teeth (640).

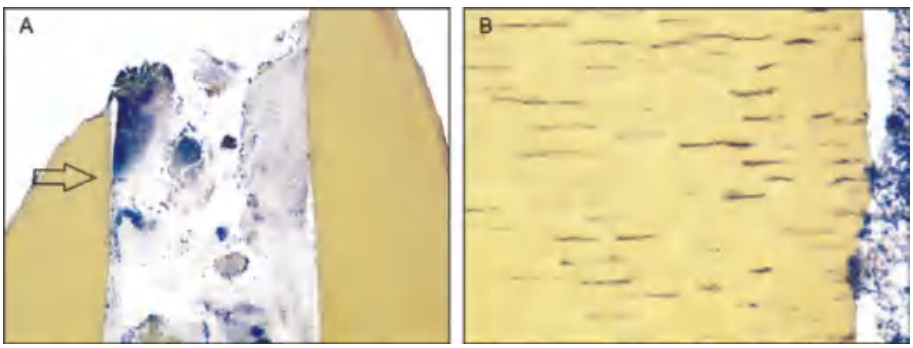
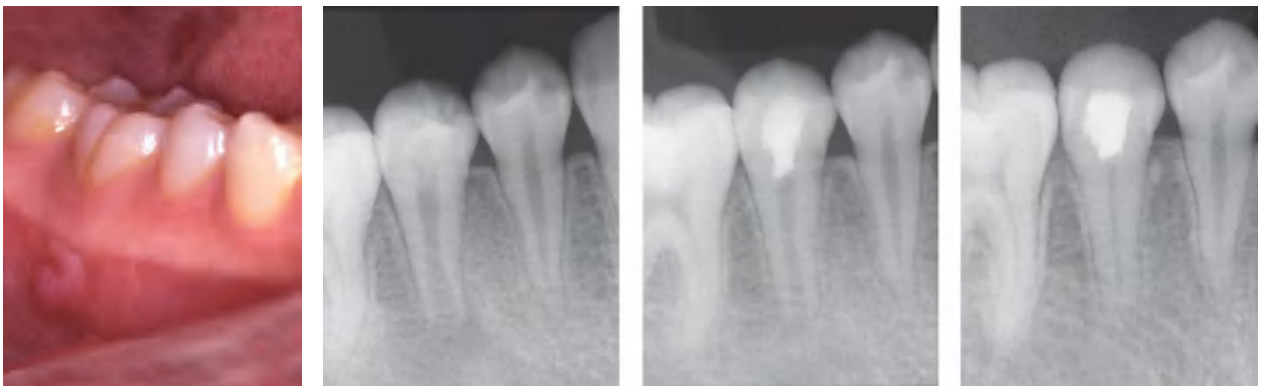



**Fig. 2.43** Avulsed and replanted immature maxillary incisor. (A) The tooth was extracted after regenerative endodontic procedures had failed. The section is cut approximately at the center of the wide canal. Necrotic debris and abundant biofilms occupy the apical canal. Taylor's modified Brown & Brenn, original magnification ×16. (B) High power view of the area indicated by the arrow in (A). Tubules of the dentin wall are extensively colonized by bacteria, which have penetrated to a considerable depth. Original magnification ×400.



A B C D

**Fig. 2.44** Revascularization of an immature premolar with apical periodontitis. A and B. Preoperative condition showing a fistula and an apical radiolucency. C. The root canal was opened and the superficial part of the canal cleaned. The tooth was left open. At the following visits the upper part of the root canal was irrigated using 5% sodium hypoclorite and 3% hydrogen peroxide. Antimicrobial agents (metronidazole and ciprofloxacin) were placed into the root canal. The root canal was not mechanically cleaned during the treatment period (B). At the fifth visit, the existence of vital tissue approximately 5 mm apical to the canal orifice was confirmed by visual inspection. A radiograph taken 5 months after permanent closure of the access cavity revealed the first signs of apical closure. A slight increase in thickness of the root canal wall was also observed (C). D. Thirty months after the intial treatment showing complete closure of the apex and thickening of the root wall due to a diminished pulp space. Reproduced from (574) with permission.

A specific response may be encountered during revascularization of an ischemic pulp if bacteria have invaded dentinal tubules, as with an untreated dentin fracture. Resorption may then occur in relation to infected dentinal tubules (see Chapter 16).

One factor found to arrest the revascularization process is infection where bacteria gain access to the ischemic part of the pulp. This has so far been considered a definitive step preventing further advancement of the revascularization process. In the late 1960s and the 1970s, attempts were made to examine whether removal of an ischemic pulp, sterilization of the pulp canal and inserting or provoking a blood clot in the root canal would result in pulp revascularization. However, these attempts were not successful, apparently due to contamination of the root canal with bacteria (572–574).

A very interesting clinical human case has been reported where an immature premolar with an apical granuloma was treated with pulp extirpation and treatment of the root canal with antimicrobial solutions. This tooth showed gradual revascularization up to the coronal level and subsequently included gradual canal obliteration (574) (Fig. 2.44). The results of this new technique are described in Chapter 26 (p. 72).

In immature teeth the response of HERS to infection is also a part of the pulpal response. This response has already been described.

## Internal root resorption

This is a rare phenomenon and a relation to trauma can often be made. For a review, readers are referred to recent reviw articles by Patel et al. (627) and Haapasalo and Endal (673). The etiology and treatment is further described in Chapter 25.

## Oral mucosa and skin

The lips comprise a complex unit of highly vascularized connective tissue, musculature, salivary glands and hair follicles (Fig. 2.45). Therefore, injury to this area can affect a number of different anatomic structures in which healing disturbances might arise.

### *Response to injury*

The main injuries affecting alveolar mucosa and lips are abrasions, incisions and lacerations. A combination of laceration and contusion is seen in the penetrating lip wound, where teeth have been forced through the tissues. In this type of lesion, the tissue is lacerated and contused and contaminated by foreign bodies and bacteria.

The wound healing response in both oral mucosa and skin has been researched after incision injury (577–580, 609, 610) or excision injury (581–595, 611). An excellent review on the healing events in oral mucoperiosteal wound healing has been published by Harrison in 1991 (594).

Within 4–24 hours after injury, epithelial cells begin their migration from the basal cell layer peripherally to the base of the coagulum. A thin epithelial cover is thereby able to close a simple wound within 24 hours (582, 583). This event is seen earlier in mucosa than in cutis and is possibly related to the moist environment found in the oral cavity (611). By mitotic activity this epithelium will eventually achieve normal thickness.

In the connective tissue, a number of wound healing processes are initiated. Thus after 2 days, a proliferation of endothelium can be observed. A wound healing module is created whereby macrophages, fibroblasts and capillaries, originating from existing venules, invade the injury zone (590). This process will close the wound with young

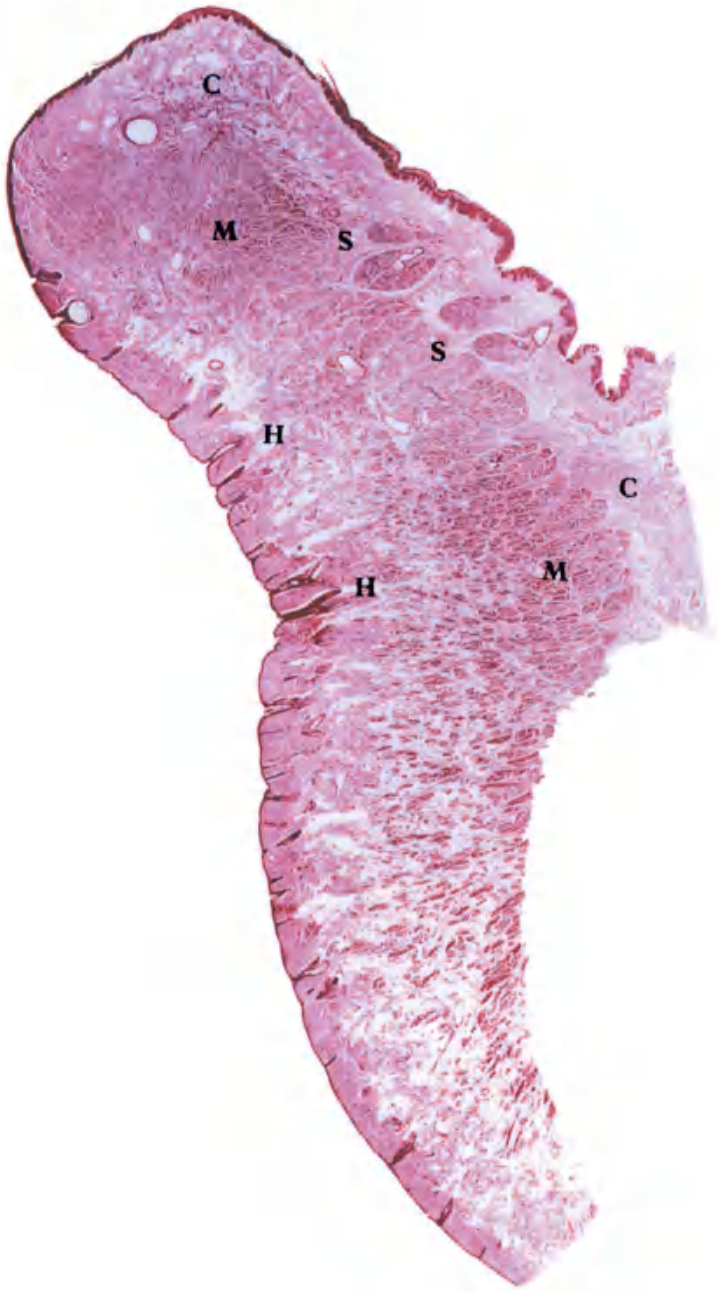



**Fig. 2.45** Anatomy of the human lip. C, connective tissue; H, hair follicles; M, musculature; S, salivary glands.

mature connective tissue within 5–10 days. These events are described in detail in Chapter 1.

In general, wound healing processes are facilitated by a very good vascular supply, as is seen in the lips; a fact which may explain the relatively rare occurrence of serious complications such as tissue necrosis in this region.

In an experimental study in monkeys, it was found that a penetration wound caused by an impact forcing the incisors through the lower lip resulted in crushing of musculature and salivary gland tissue, as well as occasional entrapment of foreign bodies (plaque, calculus, tooth fragments and remnants from the impacting object) (595). These events imply a complex pattern of simultaneous healing in the oral mucosa, salivary gland tissue, muscular tissue and skin. The regenerative potential of each of these tissue components, as well as their response to infection, varies significantly. For example, oral mucosa lesions – in contrast to skin lesions – generally heal without scarring, an observation which is presently based only on clinical documentation.

Healing of damaged salivary gland tissue is presently not very well researched. However, considering that these cells are highly differentiated, only limited or no regenerative potential can be expected.

Muscle can regenerate (596–604), but this process is often complicated by competition from connective tissue, frequently leading to formation of scar tissue in the lips.

### *Response to infection*

The incorporation of foreign bodies during wound healing implies a risk of infection by their mere presence, as well as possible bacterial contamination. Furthermore, even sterile

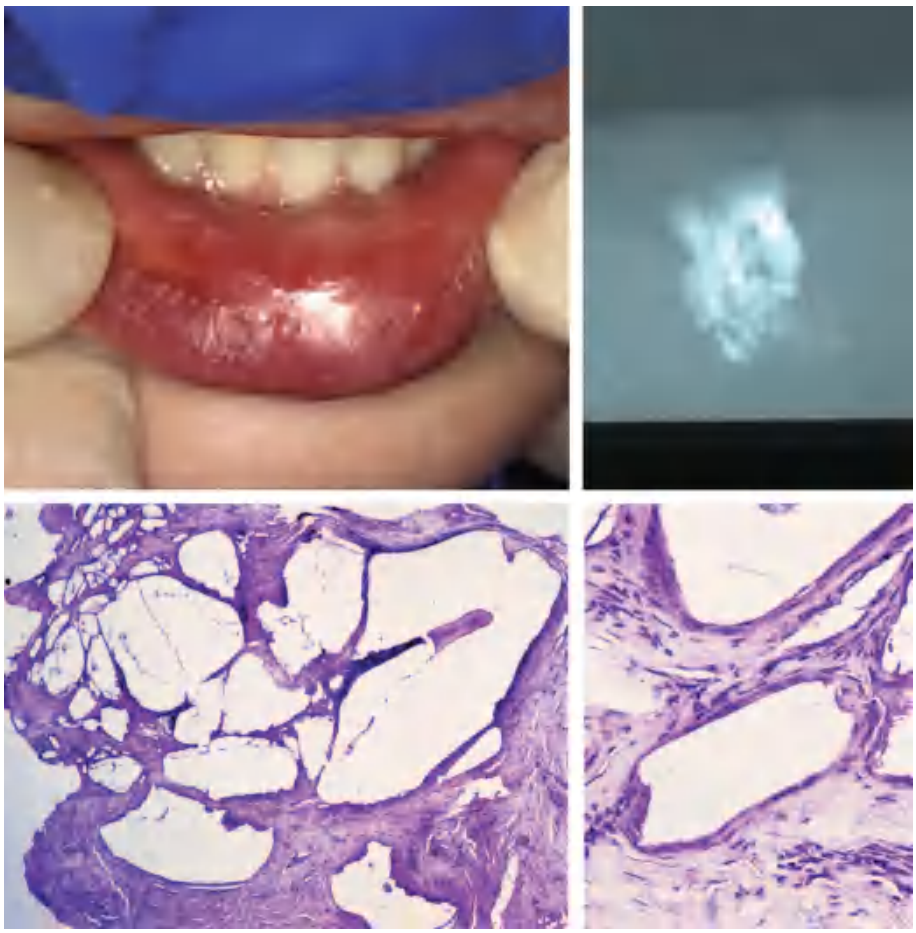


**Fig. 2.46 Healing complications after a penetrating lip lesion**
Scarring in a lower lip due to persistence of foreign bodies. The patient had suffered a penetrating lip lesion 6 months previously. Induration is felt in the lower lip and a radiographic examination shows foreign bodies.

**Removed tissue**
The indurated tissue was removed and a histologic examination showed that it consisted of fibrous tissue and multiple foreign bodies, most likely enamel and dentin fragments. ×10 and ×40.

foreign bodies usually invoke a foreign body reaction and formation of fibrous scar tissue (see Chapter 1). A long-standing chronic foreign body reaction and/or infection may lead to fibrosis which encapsulates the foreign body by a concentration of macrophages or giant cells (Fig. 2.46).

A common foreign body in the lips appears to be enamel fragments. The reaction to these appears to be fibrous encapsulation and a macrophage/giant cell response, while resorption of tooth fragments by osteoclasts (also called odontoclasts) is sometimes found.

## Essentials

### Dental follicle

The dental follicle has some regenerative potential. Severe damage leads to ankylosis and/or disturbances in tooth eruption.

### Cervical loop

This structure has a great regenerative potential after trauma whereby crown and later root formation can usually be completed.

### Ameloblasts

These cells are very sensitive to trauma and infection. Both events usually lead to partial or total arrest of enamel formation and secondary mineralization.

### Reduced enamel epithelium

This has limited, if any, regenerative potential. Trauma and infection may therefore lead to defects in secondary mineralization.

### Enamel organ

The enamel organ is sensitive to trauma and infection; injuries lead to disturbances in mineralization and enamel matrix formation. Short-term chronic inflammation (i.e. months) has very little or no effect whereas persisting inflammation (years) results in disturbances in enamel matrix formation and mineralization.

### Hertwig's epithelial root sheath

This structure can be damaged directly during a luxation injury or indirectly by delayed revascularization due to incomplete repositioning. It has a certain regenerative potential and, depending on the extent of injury, partial or

total arrest of root development may occur. It appears to be rather resistant to even prolonged pulpal infection.

## Gingiva and periosteal complex

Regeneration of gingival anatomy usually takes place 1 week after wounding.

## Cementum–periodontal ligament complex

Traumatic injury to the periodontal ligament can lead to various types of resorption (i.e. repair-related resorption, infection-related resorption and replacement resorption), depending upon the extent of the PDL injury, pulpal condition and age of the patient. The cementum–PDL complex is resistant to chronic infection. However, acute infection leads to degradation of PDL fibers. Resorption of the root surface can occur subsequent to chronic or acute infection.

## Alveolar bone and marrow complex

The response to fracture or contusion is resorption of necrotic bone and subsequent osteogenesis. The reaction of bone to chronic infection is bone resorption and formation of granulation tissue which delimits the focus of infection. In acute infection, bacteria spread to the bone and bone marrow where they elicit severe inflammatory changes.

## Pulp-dentin complex

The pulp exhibits a repair potential after traumatic injury as long as infection is avoided. This applies to accidental exposure of the coronal part of the pulp, where a hard tissue barrier usually walls off the exposure. In cases of severance of the vascular supply to the pulp due to luxation or root fracture, the width of the pulp–periodontal interface is decisive for successful revascularization. If infection occurs in the pulp in its ischemic phase after luxation or replantation, the revascularization process becomes permanently arrested.

## Oral mucosa and skin

The lips have, due to their vascularity, an excellent healing capacity. Scar tissue formation is frequent in skin but rare in mucosa. Acute or chronic infection usually results from penetrating lip wounds due to contamination with foreign bodies.

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# 3

# Response of Traumatized Dental Tissues to Various Treatment Procedures

J. O. Andreasen

This topic is very significant as a review of the world literature has shown that two-thirds of patients in different countries get inefficient or outright harmful treatment (1). This paradox can best be explained by the fact that many trauma treatments are traumatogenetic by their very nature, e.g. a forceful reposition may traumatize the root surface, periodontal ligament and alveolar bone. Likewise, the application of some splinting types may do the same (1). Furthermore, the quality of reporting in the literature is far from ideal (2–4). Finally, it is known that invasion of bacteria into traumatized dental tissue often prevents the natural occurring healing events. Therefore, this chapter represents a critical analysis of the role of the following treatment factors:

1. Repositioning
2. Splinting procedures
3. Antibiotic prophylaxis
4. Treatment, acute or delayed

## Role of repositioning

It is naturally assumed that correct repositioning of a displaced tooth may be a prerequisite to fast and optimal healing (Fig. 3.1). However, this assumption is challenged by the fact that teeth, usually premolars, can be transplanted to other alveolar regions with a very high likelihood of excellent pulp and periodontal healing (5, 6).

In dental traumatology only two experimental (7, 8) and three clinical (5, 9, 10) studies have analyzed this phenomenon, and the results of these studies will be presented.

### Experimental studies

In one study in monkeys, the healing events after accurate and non-accurate repositioning were analyzed for replanted maxillary incisors in relation to the speed of pulp healing. It appeared that slightly inaccurate repositioning (180° rotation of the replanted tooth in its socket) resulted in approximately 1 week's delay of revascularization (7).

In another experimental study using monkeys, less accurate repositioning (approximately 4 mm out) of teeth with very immature root formation resulted in some cases in the arrest of root formation and invasion of the pulp chamber with bone and PDL (8).

These two experimental findings seem to favor anatomically correct repositioning.

### Clinical studies

Three clinical studies have examined the value of optimal repositioning. In a study of 400 *root fractures* good repositioning resulted in more favorable healing, with more hard tissue healing and less pulp necrosis (9).

A study of *alveolar fractures* showed that optimal repositioning resulted in significantly fewer cases of pulp necrosis (5). Finally, a study of *mandibular fractures* showed that accurate repositioning resulted in less pulp necrosis and eliminated the loss of marginal bone (10).

## Role of splinting

### Periodontal healing

For various reasons this is a very controversial issue. Splinting of traumatized, loosened and displaced teeth has been performed for centuries, the objective being to prevent actual loss during mastication (Fig. 3.2). Another goal is to favor optimal pulpal and periodontal healing and this matter has been seriously questioned. The purpose of this chapter

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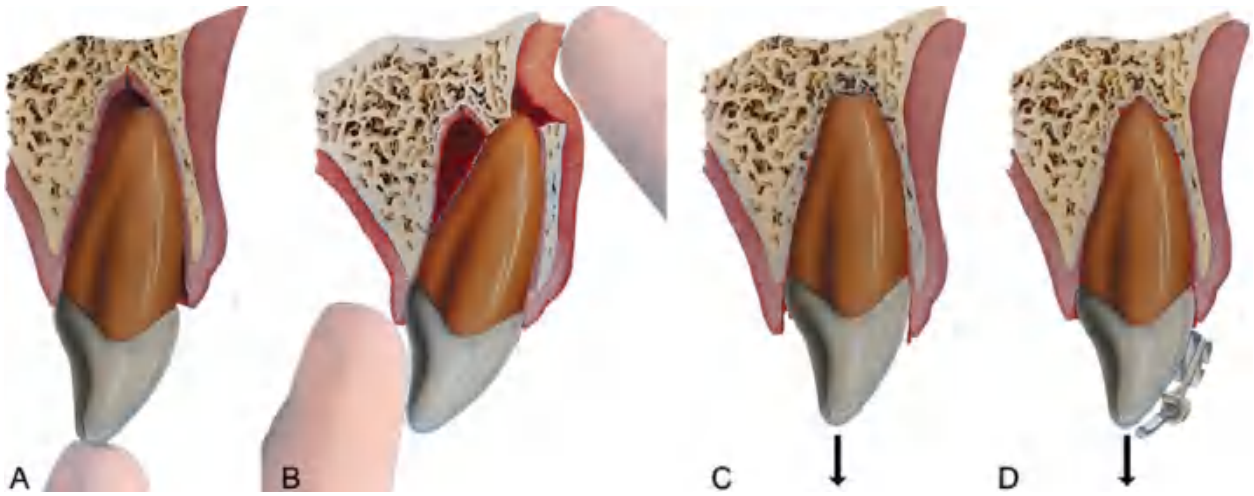


**Fig. 3.1** Effect of tooth repositioning: manual (A, B), spontaneous (C) and orthodontic (D).

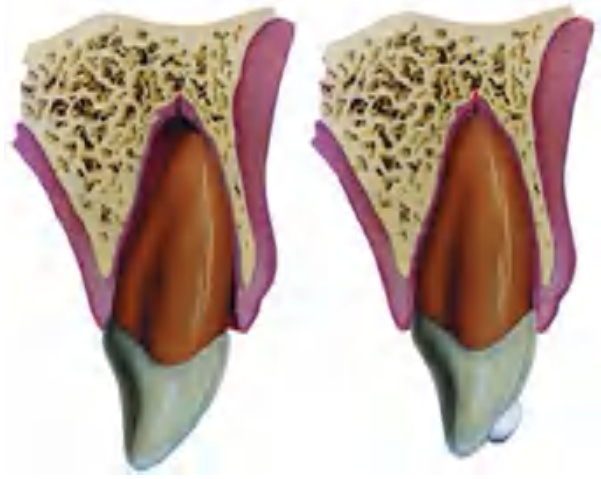

**Fig. 3.2** Effect of tooth splinting.

is to give a critical analysis of experimental and clinical experiences to date on this topic (11). Actual splinting procedures are presented in Chapter 39.

A trauma may affect many tissues such as the periodontal ligament, cementum, alveolar bone and pulp. Furthermore, the injury to these tissues may consist of a separation (rupture) injury or a crushing injury. In a case of avulsion, a drying injury may represent a third type of trauma. Each trauma type occurs with differing frequency.

Only a few studies have analyzed the effect of splinting upon various tissues after trauma, and the findings of these studies are presented here.

## *Periodontal ligament separation injuries (extrusion)*

Only a single experimental study has been performed where central incisors were extruded in monkeys. After 2 weeks there was no difference in the mechanical and histologic properties between the splinted and non-splinted teeth (12).

## *Periodontal ligament separation and crushing injury (avulsion and replantation)*

Several experimental studies on monkeys have analyzed the effect of ± *splinting* (13–15) and ± *rigid flexible splinting* (13, 15) and ± *short- or long-term splinting* (13, 16). Before the results of these studies are discussed, some facts about periodontal healing should be presented. Periodontal healing is a dynamic process and an overwhelming factor is the extent of initial damage to the periodontal ligament. Thus, for instance, a period of 1 hour drying results in almost total ankylosis; whether there is splinting or not, and the type of splinting, has no effect upon this event (17). Minor injuries to the periodontal ligament (i.e. 1–4 $mm^2$) before replantation resulted in transient ankylosis, whereas more extensive injuries (9–16 $mm^2$) resulted in an apparent permanent ankylosis that persisted for at least 8 weeks in monkeys (18). What happens after longer periods is presently unknown. In the following, the results of the above-mentioned studies will be presented.

### Effect of ± splinting

In two experiments splinting appeared to result in a slightly higher risk of ankylosis (13, 14).

### Effect of rigid versus non-rigid or no splinting

In one experiment with 8 weeks' observation, non-rigid splinting showed slightly less ankylosis than rigid splinting (13). In another experiment with 3 months to 1 year's observation no significant difference in the extent of ankylosis was found (15). Due to the dynamic nature of the extent of ankylosis over time, no definite conclusion can be made in relation to this parameter. The same finding has been found after autotransplantation of third molars (19).

### Effect of short- or long-term splinting in animal experiments

This parameter has been analyzed in two studies, and it appears from one study that splinting for only 7 days resulted in less ankylosis than in teeth splinted for 30 days (16). In

another monkey study, 2 and 6 weeks splinting were compared and no difference was found in relation to the extent of ankylosis (13). Thus, no definite conclusion about the effect of short- or long-term splinting can be made.

In studies using rats (20, 21) and monkeys (22) it was found that occlusal and/or masticatory function resulted in less ankylosis after replantation.

#### Human studies of avulsed and replanted teeth

In a clinical study of humans, the splinting effect was analyzed. A significantly better chance of optimal periodontal ligament healing was found for 0–20 days (27%) of splinting compared with longer splinting times (38%), whereas no significant effect was found for type of splinting used (rigid/non-rigid) (17). However, the results of this study should be considered with caution as splinting time was not applied in randomly.

For other trauma entities such as luxation injuries, root fractures and alveolar fractures, the effect of splinting will be described in individual chapters in this book.

### Pulp healing

Only one study in monkeys using autotransplanted incisors has been published (14). Rigid splinting was found to result in less optimal pulp healing, i.e. more extensive pulp necrosis.

### Conclusion

Based on experimental studies and one simple clinical study, the value of various aspects of splinting appear questionable such as whether or not to splint, as well as the duration and type of splinting. In essence, splinting should primarily be used for the comfort of the patient as there is not enough evidence to indicate that it favors healing of the pulp and the periodontium.

To support this conclusion the results of two studies should be considered (6, 23). A total of 370 human premolars were autotranplanted to various sites in the mandible and maxilla with excellent pulp and periodontal ligament healing, without splinting, and extrusion prevented by simply placing a suture over the occlusal surface of the premolar (6, 23).

In a similar study of autotransplanted molars, rigid splinting resulted in less favorable results for pulp and periodontal ligament healing (19).

## Role of antibiotics

It is known that some healing complications are strongly linked to the presence of bacteria in the healing tissues, resulting in acute inflammation and sometimes osteoclastic activity (24, 25). These events are especially seen after replantation of avulsed teeth and intrusive luxation. Root fractures may also suffer from infection in the fracture site.

The origin of a bacterial invasion in a trauma site is manifold and can basically be described as propagation of bacteria along a severed periodontal ligament or from the cervical area, or invasion through enamel infractions and dentinal tubules. In the case of replantation of an avulsed tooth, the root is bound to be contaminated with bacteria and possibly also foreign bodies. Finally, bacteria can be brought to the healing zone in the blood (anachoresis). In the following these four bacteria invasions will be described more precisely.

1. *Cervical invasion* consists of bacteria, often anaerobic, located in the gingival pocket and existing plaque. It has been shown that gingival bacteria can move in a blood coagulum. When bacteria invasion reaches a severe apical neurovascular supply, there is a risk that the bacteria will enter the apical area and later the whole pulp.
2. *Invasion through enamel cracks, exposed dentin tubules or an exposed pulp*. These invasion possibilities have been demonstrated *in vitro* (26–28); however, in an animal study this was not found to be an etiology (29).
3. *Root surface contamination* is an obvious risk when replanting an avulsed tooth. Before being replanted, a short cleansing of the root surface with tap water has shown a significant reduction in the extent of root surface resorption in a monkey experiment (30).
4. *Blood-borne bacteria* seeding in the healing area. A recent, large clinical study has shown that even people considered to be healthy have bacteria in their bloodstreams for many minutes every day, especially in relation to chewing and tooth brushing (31). The advancing healing edge of the wound area has leaking vessels whereby blood-borne bacteria can escape from the capillaries into the avascular tissue (32, 33).

A literature search for the effects of antibiotics in tooth and alveolar bone and jaw fractures was undertaken in 2006 (34, 35) (Fig. 3.3). Unfortunately it discovered that no

**Fig. 3.3** Antibiotics are used to limit bacterial infection in tooth trauma.

randomized controlled trials existed for traumatic dental injuries and only five studies were found for jaw fractures.

### Root fractures

In two clinical studies of root fractures, the administration of antibiotics had a negative effect. One study (36) found a borderline significance ($P = 0.10$), and, in the other study, a very significant negative influence of antibiotics was found in relation to healing $P = 0.006$) (37).

### Crown infractions and crown fractures with or without pulp exposure

The mechanical and chemical rinsing of exposed dentin or pulp with cleansing and/or disinfection media is standard in all dental filling procedures. The long-term effect of these procedures has not been established and the effect of systemic antibiotics has not been reported.

### Alveolar fractures

A clinical study of alveolar fractures did not find that systemic applied antibiotics (pencillin) favored a reduction of possible healing complications (5).

### Jaw fractures

In 2006, a review by Andreasen and co-authors (35) showed that antibiotic coverage was useful during the treatment of jaw fractures with communication of the fracture site to the oral cavity. Thus, five randomized clinical studies showed that antibiotic coverage during treatment reduced fracture infection from 39.5% in the non-antibiotic group to 7.3% in the antibiotic group (35).

### Luxation injuries

When all luxation types were considered as one group, a significant negative effect of systemic antibiotics was found for pulp necrosis, root resorption and marginal bone loss. However, when this material was broken down to individual luxation types, in lateral luxation antibiotics had a negative effect on root resorption ($P = 0.02$) (34). It should be stressed that systemic antibiotics were not administered in a random way.

### Avulsion and replantation

Several experimental studies have been carried out in dogs and monkeys to study the effect of topically and systemic administered antibiotics of various types on pulp and periodontal healing. A survey of all these studies has been presented in a recent survey article (34). However, no effect on pulpal and periodontal healing could be demonstrated in a large clinical study (17). It has therefore been suggested that prophylactic antibiotics should no longer be used.

### Topically administered antibiotics and periodontal healing

Topically applied minocycline on the root surface has been found in an animal experiment to reduce the extent of ankylosis (38–40). Unfortunately these findings could not be supported in a clinical study in humans (41) – a fact that illustrates that animal experimentation results cannot be translated directly to humans.

### Systemic administered antibiotics and periodontal healing

Systemic use of streptocillin and benzylpenecillin was initially attempted by the Hammerström group in Stockholm in 1980 in their monkey model (42, 43). A positive response was found in terms of the extent of infection-related resorption. In a similar study by Sae-Lim (44) using tetracycline and amoxillin, the same suppressing effect upon the extent of infection-related resorption was found. Unfortunately a large clinical study of 400 replanted teeth could not verify these findings (17).

### Systemic applied antibiotics and pulpal healing

Systemic administered doxycycline was not able to optimize the frequency of pulp healing in a monkey model (45).

## Role of treatment delay

For a long time it has been an axion that a treatment delay of several hours and days may result in compromised healing and/or maxillofacial injuries; however, very few studies have actually studied such a relationship. In 2002 a review article was published on this topic relating to traumatic dental injuries (46), and in 2008 a review article was published on the effect of early and delayed treatment of mandibular fractures (47). The latter divided treatment approaches into acute (i.e. within 3 hours), subacute (i.e. within 24 hours) and delayed (i.e. more than 24 hours). For obvious reasons no randomized studies were found in the two reviews.

### Uncomplicated crown fractures (enamel-dentin fractures)

In two studies of pulp capping and partial pulpotomy, no effect of a delayed treatment approach was found (48, 49).

### Complicated crown fractures (enamel-dentin fractures with pulp exposure)

In two studies of pulp necrosis and partial pulpotomy, no effect was found (48, 49).

### Crown-root fractures

No studies have been published on the effect of acute or delayed treatment in these fractures.

### Root fractures

Three studies were published and showed no effect of acute or delayed treatment (48–50).

### Fractures of the alveolar process

One recent study showed no effect of delayed treatment on pulp healing, root resorption and marginal bone loss (5).

### Mandibular fractures

Treatment delay was analyzed in six studies (47). Four studies showed no significant difference between immediate (i.e. treatment within 3 days) and delayed treatment. One study showed a presence of healing for cases treated within 3 days, whereas another study showed more optimal healing for cases treated 3–5 days after injury. There was a high risk of bias in all five studies.

### Luxation injuries

No effect of treatment delay could be detected in recent studies of various luxation types (46).

### Avulsion and replantation

In this type of injury a very significant relationship has been found with extraoral storage time, and a very complex relationship has been found between pulp and periodontal healing and time until replantation (51, 52). This relation is explained further in Chapter 17.

### Primary tooth injuries

No studies have been published where this parameter could be evaluated.

## Essentials

### Role of repositioning

From experimental evidence, in cases of *luxations*, the exactness of repositioning does not seem to be of much importance. Where repositioning inexactness is ≥4 mm, evidence has shown that this may lead to arrested root development and invasion in the pulp chamber of bone and periodontal ligament.

In cases of *root fractures*, exact repositioning may optimize pulp healing and fracture healing with hard tissue. In cases of *alveolar fractures*, exact repositioning favors pulp healing. In *jaw fractures*, exact repositioning eliminates or lessens the risk of pulp necrosis and the risk of loss of marginal bone support.

### Role of splinting

Based on animal experiments the role of splinting type (flexible/rigid and splinting time) cannot be determined. No clinical studies without a risk of bias have been published where the effect of splinting could be determined.

### Role of antibiotics

Several experimental replantation studies have been published where both systemic and topically applied antibiotics have been used and found efficient. However, clinical studies could not demonstrate such an effect. In regard to luxations and root and alveolar fractures, no clinical studies have shown a positive effect of antibiotics on healing. In mandibular jaw fractures, a meta-analysis showed that systemic antibiotics significantly reduced the risk of fracture line infection.

### Role of treatment delay

Apart from replantation of avulsed teeth, no significant negative effect of moderate treatment delay (i.e. hours, days) could be demonstrated.

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# 4

# Prediction and Timing of Healing Complications

J. O. Andreasen

## Introduction

Soon after a trauma patients will want to know if there will there be healing complications after the injury and when these can be diagnosed. To be able to answer these questions, long-term studies of well-documented trauma patients are necessary.

For various reasons, such studies have been very difficult or impossible to undertake in most countries. The fact that it takes 5–10 years before late complications can be diagnosed explains the rarity of such studies. In 1965, the Department of Oral and Maxillofacial Surgery at the University Hospital in Copenhagen started such studies and now, 50 years later, a large number of trauma patients have been followed for up to 20 years after trauma. All types of dental trauma patients can now be evaluated for their potential risk of developing healing complications. A series of follow-up studies showed that two factors have an overwhelming influence on the risk of complications, namely the type of injury – divided into fractures and luxation injuries, and combinations of these – as well as the stage of root development (1). Apart from that, the type of dentition, primary or permanent, also has a decisive influence.

In the following, the predictors for different healing complications in the permanent dentition will be described based on a series of long-term studies.

### Pulp healing/pulp of necrosis

The strongest healing predictor appears to be the *type of injury*, and the next strongest the *diameter of the apical foramen* as reflected by the stage of root development. Thus, the greater the diameter of the apical foramen, the better the chance of revascularization (2, 3). This factor has been shown to apply to luxations (2, 3) as well as avulsed and replanted teeth (4) and root fractures (5, 6). In cases of intrusion with immature root formation, crushing damage may occur to the apical part of the pulp, resulting in a higher risk of pulp necrosis when compared to the risk with an extrusive luxation (7). Another predictor appears to be the *length of the pulp* (3). This applies both to luxated teeth (3) as well as root fractures, where the length of the pulp is defined as the distance from the fracture to the tip of the pulp horns as this represents the length of revascularization from the fracture site (8). Age is possibly also a factor in determining the risk of pulp necrosis (2, 3) (Fig. 4.1).

*Bacterial invasion* through exposed dentinal tubules due to a crown fracture has been shown to lead to an increased risk of pulp necrosis in teeth with a luxation injury (9–11).

In cases of untreated crown fractures with pulp exposure, either a pulp polyp will be formed or a descending pulp necrosis (see Chapter 2). In cases of root fractures and alveolar process fractures, optimal repositioning appears to limit the risk of pulp necrosis and favors hard tissue formation between the fragments (12, 13).

### Periondontal ligament healing and root resorption

A series of clinical and experimental studies have shown that luxation injuries with displacement can lead to non-progressive root resorption (repair-related resorption) and progressive resorption (infection- and ankyloses-related resorption) (see Chapter 2). The most significant predictor for root resorption appears to be the type of luxation, especially types of luxation where compression of the periodontal ligament (PDL) has taken place (i.e. intrusion) (1, 14, 15).

In cases of avulsion, a further injury may damage the PDL, in particular the drying effect of the PDL, which after a few minutes may kill the vital PDL cells. Thus, a very strong association has been found between the length of the extraoral dry period and the extent of progressive root

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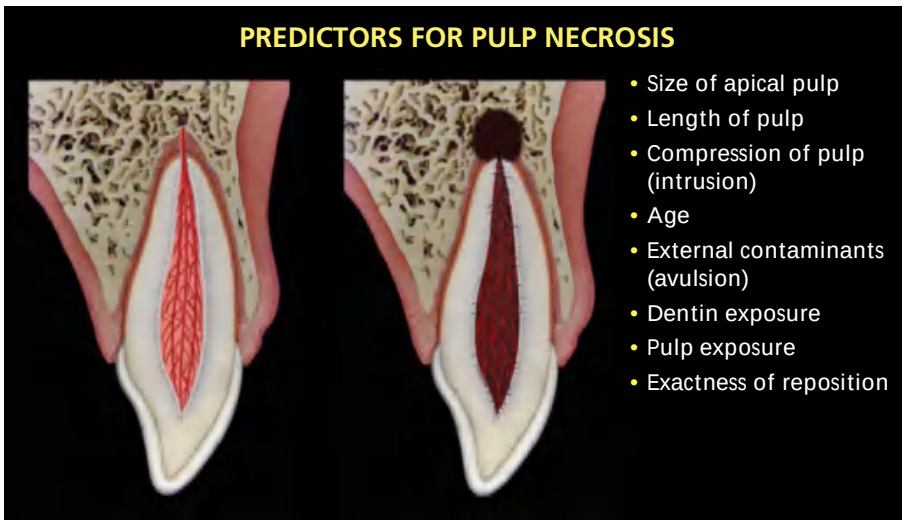


**Fig. 4.1** Predictors for pulp necrosis. From (1) with permission.

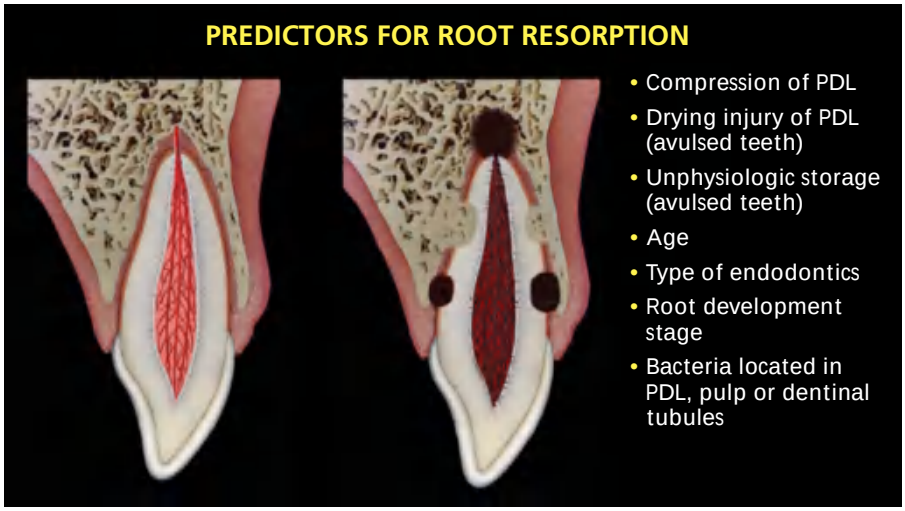


**Fig. 4.2** Predictors for root resorption. From (1) with permission.

resorption (4). Furthermore, a strong association has been found to exist between the stage of tooth development and the risk of progressive root resorption. Teeth with early stages of root formation are most severely affected (16). Finally, it has been found experimentally and clinically that bacteria contamination of the root surface of an extracted or avulsed tooth may augment the resorption attack on the root (21) (Fig. 4.2).

## Marginal bone loss

This complication is rare and primarily related to injury types such as lateral luxation, intrusive luxation and replantation of avulsed teeth; i.e. all injuries where marginal bone has suffered a crushing injury (1). Furthermore, this event may happen in cases of jaw fractures in sites exposed to the oral cavity (17). Transient marginal breakdown is also a healing phenomenon where injured marginal bone is resorbed whereafter new cervical bone is formed (see Chapter 16).

Predictors for marginal bone loss appear to be intrusion injuries, especially multiple intrusions, as well as older age(18). Lateral luxation may also result in marginal bone loss, especially lingually where compression of the bone appears to be at a maximum (19). Alveolar fractures and jaw fractures may develop marginal bone loss where the fracture site communicates with the oral cavity (Fig. 4.3).

## Avulsion injuries

This is the type of injury that most often leads to tooth loss, due to the high risk of progressive root resorption (20).

In young individuals, a failing and resorbing tooth can be partly saved by a decoronation procedure (see Chapter 32). In adults, it has been found that replanted teeth with ankylosis-related root resorption can survive for a long time (20).

## Intrusive luxation

This luxation type results in extensive injury to the PDL, and thus a high risk of progressive resorption, which may lead to tooth loss (7).

## Root fractures

Two very significant predictors indicate early tooth loss, namely a cervical location of the root fracture as well as pulp

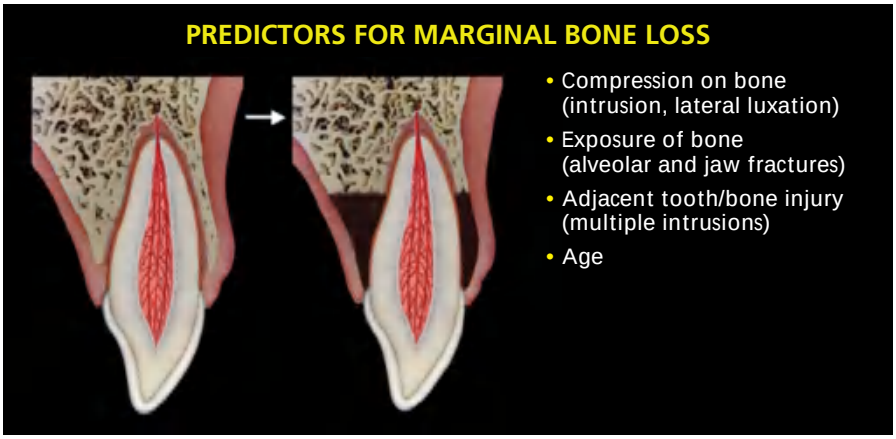


**Fig. 4.3** Predictors for marginal bone loss. From (1) with permission.

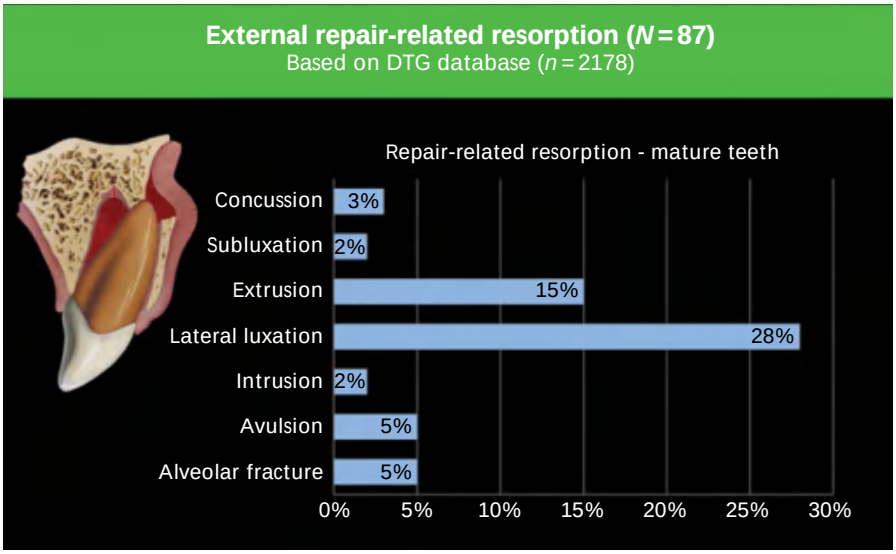


**Fig. 4.4** Repair-related resorption related to type of injury in mature teeth. From (23).

necrosis in the coronal part of the fractured tooth (21). A new trauma may lead to extrusion or avulsion of the coronal fragment (21).

### Crown-root fractures

Teeth with crown-root fractures are often extracted as it is not considered possible to preserve the teeth. Chapter 14 discusses several treatment possibilities that can be considered, such as fragment bonding, gingivectomy and/or osteoectomy. Surgical or orthodontic extrusion is also a possibility (22).

## Observation time until complications can be diagnosed

A recent large clinical and radiographic study of 2178 teeth has illustrated typical time frames when various complications can be diagnosed (23). However, the time of diagnosis will depend on when the patient is seen for a follow-up visit, and it is therefore not possible to decide the exact time of onset. The standard follow-up protocol in the Copenhagen study included examinations after 1 week, 3 weeks, 6–8 weeks, 6 months, 1 year, 5 years and 10 years (16).

### Repair-related resorption

This complication appeared in 87 teeth and most frequently affected teeth with extrusive or lateral luxation. After 3 months, 23% could be diagnosed and after 1 year, 86%. Repair-related resorption usually appeared as a blunting or shortening of the apex (14, 23, 24) (Figs 4.4 and 4.5).

### Infection-related resorption

This complication was registered in 174 cases and most frequently after intrusion or avulsion. After 3 months 68% were diagnosed, and 79% after 6 months. Finally, after 1 year 82% were diagnosed (Fig. 4.7).

### Ankylosis-related resorption

This complication was found in 305 cases and was almost exclusively related to intrusions and avulsions. After 1 year 78% were diagnosed, but ankylosis could first appear up to 10 years after the trauma. In general, ankylosis could be diagnosed earlier in children than adults (Figs 4.8 and 4.9).

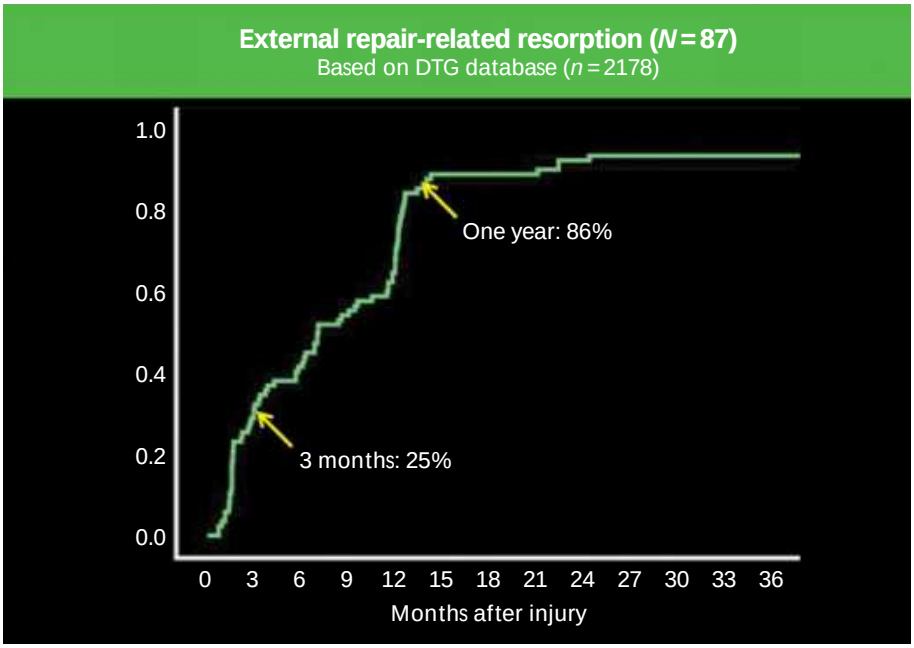


**Fig. 4.5** Observation time until appearance of repair-related resorption. From (16).

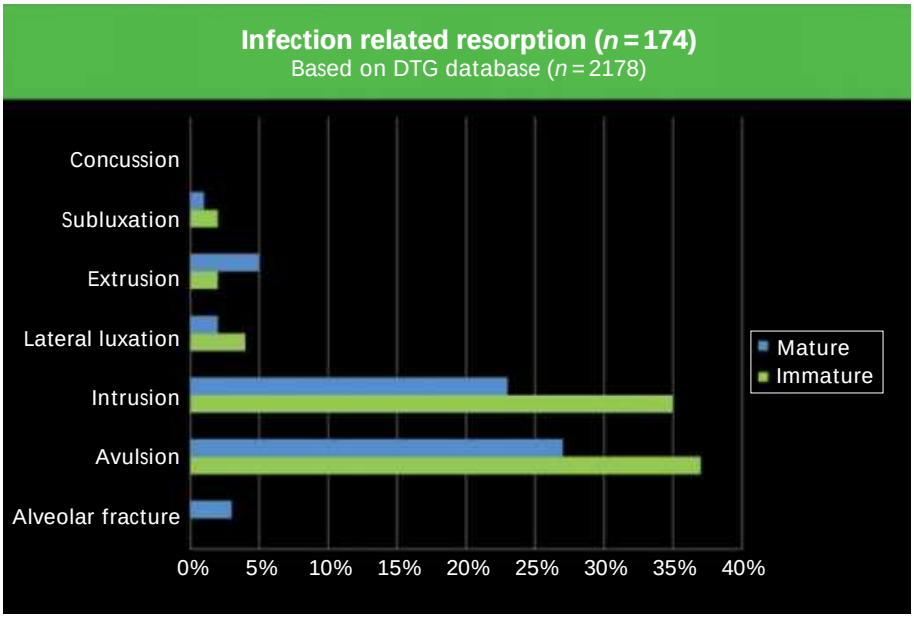


**Fig. 4.6** Infection-related resorption related to type of injury in mature and immature teeth. From (16).

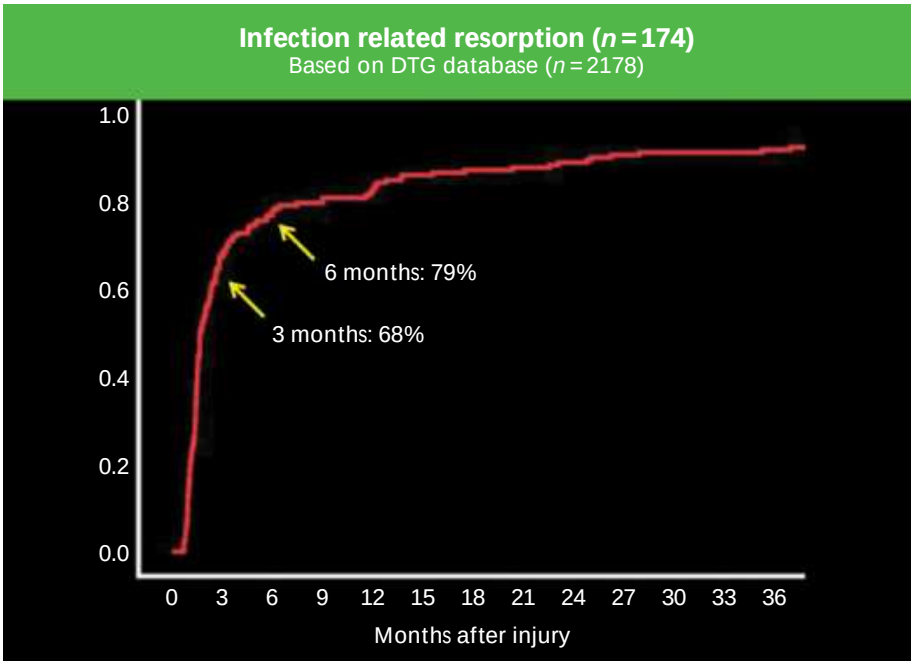


**Fig. 4.7** Observation time until appearance of infection-related resorption. From (16).

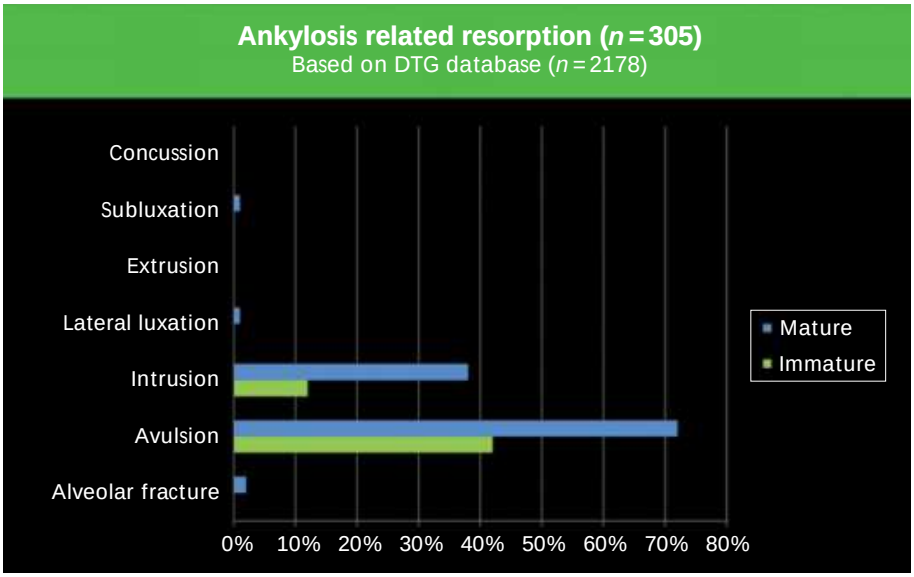


**Fig. 4.8** Ankylosis-related resorption related to type of injury in mature and immature teeth. From (16).

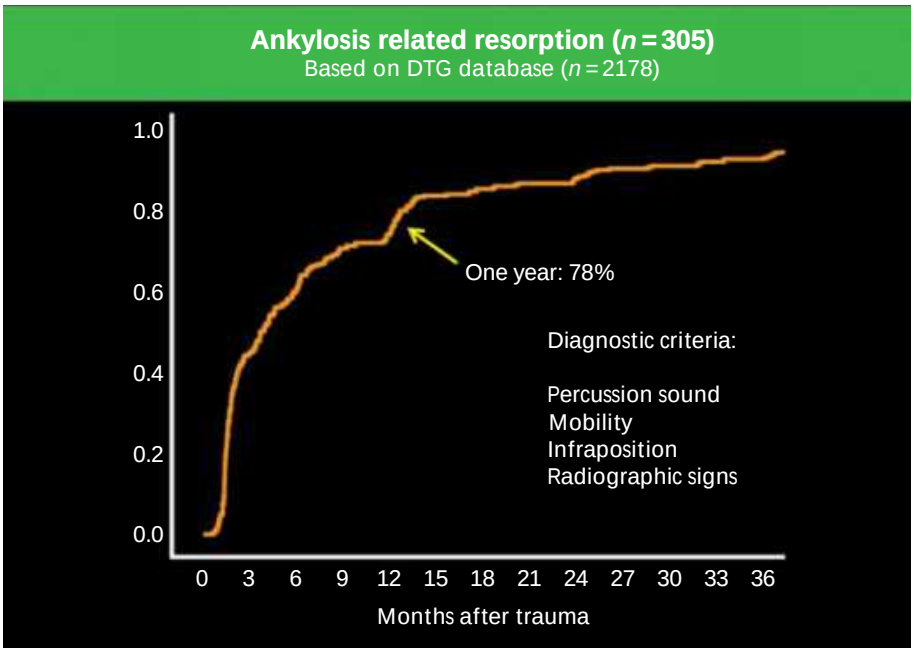


**Fig. 4.9** Observation time until appearance of ankylosis-related resorption. From (16).

## Tooth survival and tooth loss

Tooth loss after trauma can be immediate in cases of tooth avulsion where the tooth cannot be retrieved or in situations where replantation appears to be contraindicated (25). In most cases tooth loss appears at a later date, where a serious complication such as progressive root resorption has advanced to such an extent that extraction becomes the only possibility. Decoration should be considered in younger patients as the root may preserve the volume of the socket and thereby augment the chance for successful replacement with an implant (see Chapter 34).

A number of clinical studies have shown that the following types of injury are significantly related to early tooth loss: avulsions with subsequent replantation, intrusions, root fractures and crown-root fractures (Fig. 4.10).

## Use of predictors in an interactive dental trauma guide

In 2007 the University Hospital in Copenhagen launched an interactive Dental Trauma Guide (DTG) (www.dentaltraumaguide.org; accessed January 2018) that allows dentists to seek advice concerning the prognosis of a patient's actual trauma, if predictors relating to the trauma are transferred to the DTG (24). The program will then search in a database of approximately 4000 cases for a similar trauma type and similar tooth development stage and give up to 10 years' estimate of the risk of the complications described in this chapter. A detailed description of the function of the DTG is given in Chapter 43.

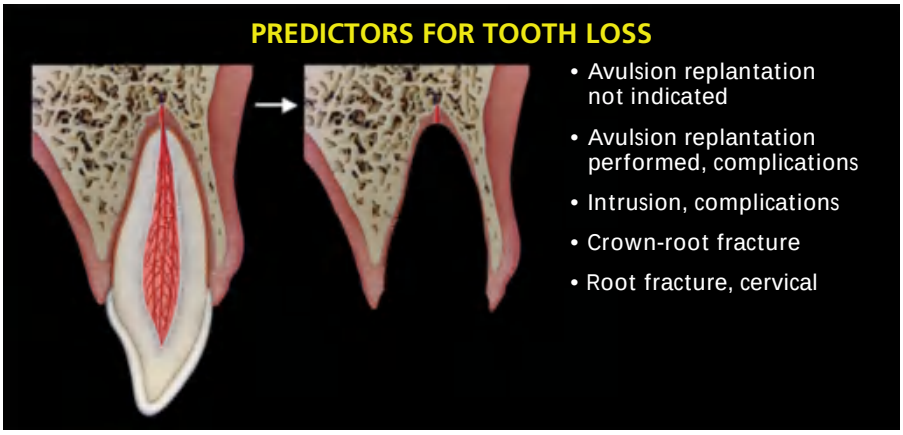


**Fig. 4.10** Predictors for tooth loss. From (1) with permission.

## Essentials

### Most significant predictors for pulp necrosis

- Type of traumatic injury
- Stage of root development
- Increasing age
- Combined crown fracture–luxation injuries

### Most significant predictors for progressive root resorption

- Type of luxation injury
- Non-physiologic storage including dry storage of avulsed teeth before replantation
- Stage of root development

### Most significant predictors for marginal bone loss

- Type of luxation injury, with intrusive luxation and avulsion with subsequent replantation being the most significant
- Alveolar process and jaw fractures
- Multiple intrusions

### Most significant predictors for tooth loss

- Avulsion, replantation not indicated
- Avulsion, replantation performed
- Intrusion
- Crown–root fractures
- Root fractures cervically located

Predictors can be used in prognosis estimation.

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# 5

# Characteristics of Oral Stem Cells and Their Role in Dental Tissue Regeneration

G. T.-J. Huang, P. Gauthier, M. Jamal & M. Al-Habib

## Introduction

Dental tissues have a limited capacity to repair after damage, especially after traumatic injuries. Pulp tissue may recover from minor trauma and can generate newly formed tertiary dentin including reparative and reactionary dentin. Cementum has more capacity to regenerate and repair as it can be remodeled in response to physiologic or pathologic conditions. One reason for this is that cementum is surrounded by peridontal ligament and alveolar bone where a good blood supply can be obtained. It is well known that when resorption occurs on root cementum, it can be regenerated by cementoblasts. Cementum can also be observed to grow into the root canal space if the pulp tissue is lost (for a review, see (1)). When a tooth root is cracked or fractured, new cementum may be formed to fill in the gap if infection is under control (2, 3). Similarly, the apical barrier formed at the apex of immature permanent teeth after apexification is mainly cementum (4). When a tooth is subluxated or avulsed, repositioning or replantation may be successful if the infection is controlled and the periodontal ligament can recover completely. If not, root replacement resorption by established bony ankylosis may ensue (5).

Various clinical protocols have been or are still being developed in the management of traumatic injury to teeth. In the case of avulsion, many temporary storage conditions of teeth and treatment methods have been studied and refined (6). However, there has been a lack of significant breakthroughs in terms of developing a treatment method that could salvage severely damaged teeth, for example from fracture or avulsion.

Recent advancements in cell-based tissue engineering and the discovery of dental stem cells have stimulated the thinking that these cells may be utilized for repairing traumatized teeth. In the field of medicine, regenerative therapies using cell-based approaches have made significant strides; for example, the regeneration of a whole urinary bladder and the main trachea in humans (7, 8). In dentistry, most cell-based regenerative work has been tested in small or large animal models, including whole tooth regeneration, pulp-dentin regeneration, periodontal regeneration or bone regeneration in large jaw bone defects (9–12). So far, very limited clinical studies on humans have been published using stem cells to regenerate pulp-dentin, to repair periodontal defects and to enhance jaw bone regeneration (13–16).

Since the discovery of dental pulp stem cells (DPSCs), reported by Gronthos et al. in 2000 (17), a number of dental/oral stem cells have been identified and characterized (for review, see (18, 19)). These include stem cells from apical papilla (SCAP), stem cells from human exfoliated deciduous teeth (SHED), periodontal ligament stem cells (PDLSCs), dental follicle stem cells (DFSCs) and gingival mesenchymal stem cells (GMSCs). In this chapter we briefly review the general characteristics of stem cells and then focus on oral stem cells, DPSCs, SCAP, PDLSCs, GMSCs and DFSCs with an emphasis on their tissue regeneration capacity evidenced by *in vivo* studies in small and large animals. Their potential use for repairing dental defects cause by trauma is also discussed. These stem cells are located in the teeth and associated tissues as depicted in Fig. 5.1.

## General concept of stem cells

### Stem cell definition and properties

A stem cell is defined as a cell capable of both self-renewal and giving rise to one or multiple lineages of differentiated and specialized cell of certain functional phenotypes (Fig. 5.2), thereby generating complex tissues and organs (20). Because of these properties, stem cells are considered

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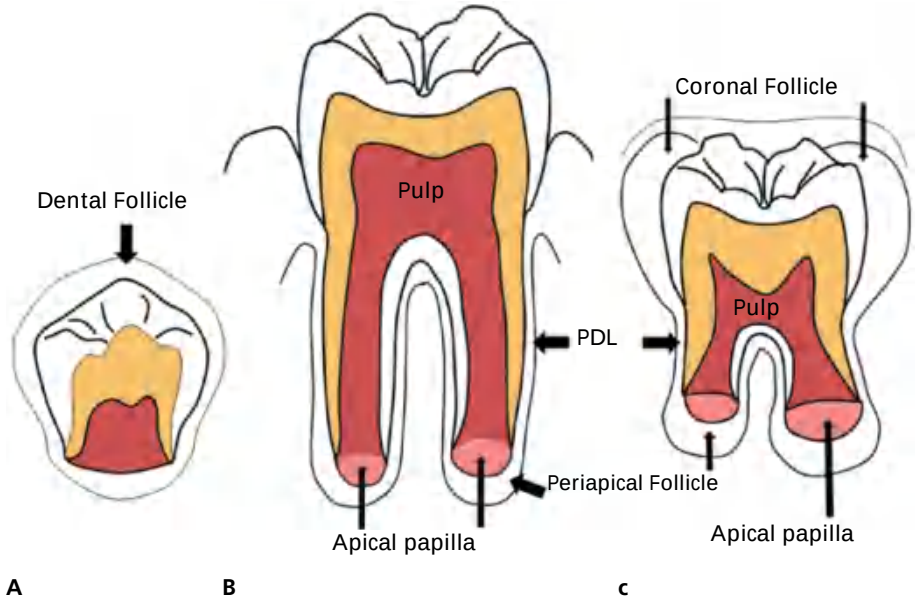



**Fig. 5.1** Dental tissues in permanent teeth that harbor stem cells. A. Unerupted tooth. B. Erupted tooth with immature apex. C. Erupting tooth before breaking into oral cavity.

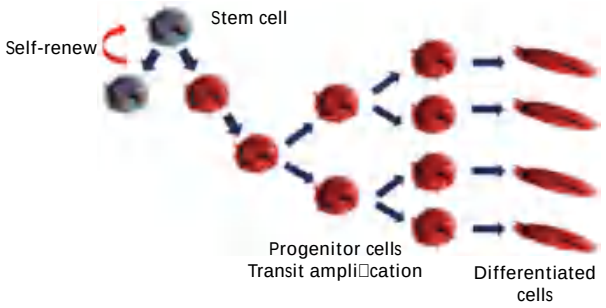



**Fig. 5.2** Schematic of the self-renewal and differentiation of a stem cell showing the asymmetric division of a stem cell into one stem cell and one progenitor cell, then undergoing amplification and differentiation.

one of the most promising tools for regenerative procedures and tissue engineering applications.

Depending on the potency of stem cells to differentiate into different cell lineages, thus forming various tissues, distinctive categories of stem cells have been established.

(1) Totipotent cells, such as the fertilized egg, are able to create an entire organism, a property retained by early progeny of the zygote at the four-cell stage. These cells can give rise to all the cell types of the body including the cells that make extraembryonic tissues such as the placenta.
(2) Pluripotent stem cells, such as embryonic stem cells (ESCs), can differentiate into all cell types of the three germ layers but they are not able to create the entire organism or give rise to the cells that make extraembryonic tissues.
(3) Multipotent stem cells, committed to more specific lineages, fetal stem cells and postnatal stem cells (adult stem cells) are considered multipotent. They are able to form more than one cell type, either those in or outside the tissue where they reside (21, 22).
(4) Unipotent stem cells, such as spermatogonal stem cells and oogonial stem cells, that only give rise to one cell type (23, 24).

ESCs are produced by culturing cells collected from the undifferentiated inner cell mass of the blastocyst, which represents an early stage of embryonic development after fertilization (25). Their self-renewal potential and unrestricted ability to generate all types of tissues and organs (pluripotency) make these cells an attractive cellular source for cell-based regenerative therapies (Fig. 5.3).

Fetal stem cells can be isolated from various fetal organs and tissues. Among these fetal stem cells, hematopoietic stem cells (HSCs) and mesenchymal stem cells (MSCs) have been well characterized and they have advantages over their adult counterparts for medical applications, for example their greater multipotentiality and lower immunogenicity (26).

Postnatal stem cells (adult stem cells) can be obtained from individuals at any stage in life. They are present in various tissues of endodermal, mesodermal or ectodermal lineages including bone marrow, blood, neural tissue, fat, liver, muscle, pancreas, skin, retina and dental tissues (20, 27–31). The concept of stem cell differentiation is established from a hierarchy tree where unspecialized stem cells divide to give rise to a large number of mature cellular phenotypes. A stem cell undergoes asymmetric division producing at least one daughter cell identical to the mother cell, which maintains contact with the stem cell niche and retains the ability to self-renew. The other daughter cell may differentiate into progenitors and precursor cells that are committed to lineage-specific differentiation and are only capable of symmetric division (32).

## Mesenchymal stem cells

The term mesenchymal stem cells (MSCs) was coined by Caplan to describe stem cells in the embryonic mesenchyme that are able to differentiate into specialized cell types to form and repair the mesenchymal tissues (33). Earlier, bone marrow was found to be a source of plastic adherent, fibroblast-like cells that develop colony forming units (CFU-Fs) when seeded in tissue culture plates. Their capability to

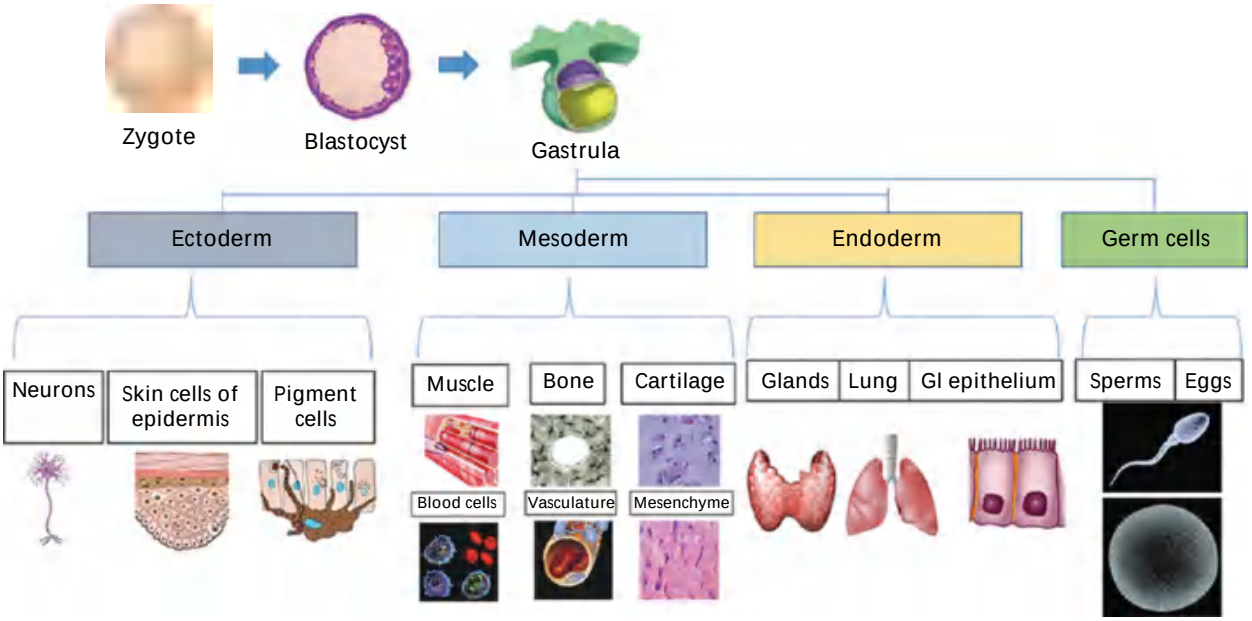



**Fig. 5.3** Derivation of differentiated tissues and organs from three germ layers. GI, gastrointestinal.

differentiate into various mesenchymal tissues gave rise to the concept of MSCs (27, 28). In 1999, Pittenger et al. characterized human MSCs from the bone marrow of the iliac crest as multipotent stem cells by demonstrating their isolation, expansion in culture and directed differentiation to osteogenic, adipogenic and chondrogenic lineages (34).

MSCs grown in culture are heterogeneous and no single marker has been identified to define MSCs, therefore it is difficult to isolate a pure population of MSCs from a tissue that can represent all MSCs (35). In fact, many markers have been associated with MSCs, thus the International Society for Cellular Therapy (ISCT) proposed minimal criteria to define human multipotent MSCs (36). The ISCT termed MSCs as mesenchymal stromal cells, which include stem cells, regardless of the tissue from which they are isolated. The key features of MSCs are the following: (1) cells are adherent to tissue culture-treated plastic when maintained in standard culture conditions; (2) cells express CD105, CD73 and CD90 and lack the expression of CD45, CD34, CD14 or CD11b, CD79α or CD19 and HLA-DR surface molecules; and (3) cells are able to differentiate to osteoblasts, adipocytes and chondroblasts *in vitro* (37). Since the announcement of this definition, more research on MSCs has been conducted and an updated definition to better define MSCs is awaited. Adult bone marrow contains rare multipotent stem/progenitor cells termed bone marrow-derived MSCs (BMMSCs). They have been the best characterized MSCs and have the potential to differentiate into various lineages such as osteogenic, chondrogenic, adipogenic, myogenic and neurogenic lineages (34, 38–40).

It was previously observed that the differentiation potential of MSCs was lineage restricted. This hypothesis is currently challenged as more evidence has evolved showing that MSCs have the potential to cross lineage boundaries, and are also able to differentiate into lineages that are not typical mesenchymal derivatives. MSCs have demonstrated their robust multipotency and even 'stem cell plasticity'. For instance, it has been confirmed that bone marrow stem cells have the ability to differentiate into neurons, osteoblasts, adipocytes and myoblasts (34, 41, 42). Stem cells that have been isolated from other differentiated tissues such as brain (29), muscle (31) and adipose tissue (30) also show similar multilineage potential. This plasticity property of MSCs increases their potential use in regenerative tissue therapy as they can regenerate into multiple tissues in different locations.

## Mesenchymal stem cell niches

Postnatal stem cells are located in particular local microenvironments known as niches. Schofield introduced the concept of a stem cell 'niche' in 1978 (43). Hallmarks of a niche include the stem cell itself, stromal supporting cells that interact directly with the stem cells via secreted factors, and cell surface molecules as well as the extracellular matrix that provides a structural support to the niche and allows the diffusion of mechanical and chemical signals. Systemic signals are implicated in the recruitment of inflammatory and circulating cells into the niche (44).

All together these elements regulate stem cell behavior. The stem cell niche is characterized by three essential properties: (1) it is an anatomic space where the number of stem cells is regulated to provide tissue homeostasis; (2) it is the place where stem cells are instructed to control maintenance, quiescence, self-renewal and recruitment in relation to differentiation, fate determination and long-term regenerative capacity; and (3) the niche will influence cell motility (45).

Stem cell niches are considered to be tissue specific, such as hematopoietic stem cells in the bone marrow and epithelial stem cells in the intestinal crypt; each has different local

microenvironments. MSCs have been reported to be associated with the vasculature (46, 47); they have a perivascular origin in multiple human organs and pericytes are considered to be MSCs because CD146$^+$ cells are perivascular cells (46). These CD146$^+$ cells co-localize with NG2$^+$ cells (a pericyte marker). Cultured perivascular cells show typical MSC markers including CD10, CD13, CD44, CD73, CD90, CD105 and CD140b (PDGF-Rb), while not expressing CD56, CD106, CD133, CD324 (E-cadherin), CD326 (Ep-CAM), CD344 (frizzled-4) and HLA-DR (46). These cells also are multipotent, having osteogenic, chondrogenic and adipogenic potential.

Caplan considered that pericytes are MSCs, however such a term may not entirely reflect the nature and function of MSCs. He coined the term 'medicinal signaling cells' as 'MSCs' because more recent studies have shown the role of MSCs that function to regulate immune responses, secrete paracrine factors to support a regenerative microenvironment, and produce antimicrobial peptides that kill bacteria (48).

## Dental pulp stem cells

### Localization of DPSCs *in vivo*

It has been long known that there are undifferentiated mesenchymal cells in the pulp tissue that can give rise to new odontoblasts forming tertiary dentin. Gronthos et al. reported the isolation of DPSCs and that they show stem cell characteristic in cultures as well as being capable of differentiating into odontoblast-like cells forming dentin-pulp complex *in vivo* (described in next section) (49). Subsequently, their putative locations in the pulp tissue were identified by several markers associated with endothelial cells, pericyte and/or smooth muscle cells such as STRO-1, 3G5, MUC-18 (CD146) and α-smooth muscle actin (50) (Table 5.1). They were mainly found to reside in a perivascular niche confirmed by immunohistochemical staining. *In vitro* magnetic bead sorting of STRO-1$^+$, CD146$^+$ or 3G5$^+$ subpopulations revealed that these cells possess more MSC characteristics than the original pool. There are also non-vascular DPSC niches as STRO-1 is expressed in the neurosheath layer as well. Other pluripotent stem cell markers such as OCT4 were also used to locate DPSCs in the pulp (51). For a review of DPSC niches, see Yu et al. (52). Some MSC markers such as STRO-1, CD90, CD105 and CD146 can be detected in human dental pulp and they are mostly associated with blood vessels as shown in Fig. 5.4A.

### Characterization of DPSCs *in vitro*

Heterogeneous populations of DPSCs can be isolated from human dental pulp using enzyme digestion and they form CFU-Fs after initial seeding into culture plates (20, 49). DPSCs have rapid proliferative rates and a capacity to form mineralized tissues both *in vitro* and *in vivo*. DPSCs share similar features to those of BMMSCs. For example, both cell types adhere to cell culture dishes and form clonogenic colonies (Fig. 5.4B). However, the majority of the individual colonies (clones) of DPSCs (67%) failed to proliferate beyond 20 population doublings in culture, suggesting that only a small portion of cells maintain high proliferation potential *in vitro* (49).

Dental stem cells are considered to be a type of MSC, therefore markers that have been used for identifying MSCs are also used, such as STRO-1, CD13, CD44, CD24, CD29, CD73, CD90, CD105, CD106, CD146 and β2 integrin. They are considered to be negative for CD14, CD34, CD45 and HLA-DR (53). Some pluripotent markers – OCT4, NANOG and SOX2 – have also been found to be positive in these cells, but are expressed at much lower levels than in ESCs (54). DPSCs can differentiate into multiple lineages *in vitro* under defined differentiation protocols including: osteogenic/odontogenic, chondrogenic, adipogenic, neurogenic and myogenic (20, 49, 55, 56). Considering their clonogenic nature, self-renewal capacity, multilineage differentiation and specific niche microenvironment, DPSCs satisfy the three criteria characteristic of human postnatal somatic stem cells.

DPSCs can also be isolated from inflamed pulp, termed DPSCs-IP (57). Similar to DPSCs from normal pulp (DPSCs-NP), DPSCs-IP may differentiate into odontoblast-like cells *in vitro* by expressing certain markers. However, they appear to have lower population doublings and less osteogenic/odontogenic capacities in cultures compared to DPSCs-NP (57). Their levels of STRO-1 expression, morphology, proliferation rate and CFU-F forming in cultures do not differ from DPSCs-NP (57, 58). DPSCs may show odontoblastogenic phenotype especially when DPSCs were seeded onto dentin as they convert into cells with a polarized cell body and a cell process extending into the existing dentinal tubules (56) (Fig. 5.5A).

DPSCs possess multiple physiologic functions that can be utilized for medical uses other than oral and dental use; for example, they promote the proliferation and differentiation of neural cells in the hippocampus of mice (59) and induce endogenous axon guidance (60) (see review (61)). A function that may be suited for oral use is their potential to form bone when combined with platelet-rich plasma or hydroxyapatite (62).

### Role of DPSCs in reparative dentin formation

One of the most important characteristics of DPSCs is their capability to form a dentin/pulp-like complex upon *in vivo* transplantation in conjunction with hydroxyapatite/tricalcium phosphate (HA/TCP) as a carrier vehicle (20, 49). Interestingly, the dentin-like material formed in the transplants had a globular appearance consistent with the structure of dentin *in situ* as revealed by electron microscopy (20). Examples of such pulp-dentin complexes are presented in Fig. 5.5B, showing the complexes formed from the transplanted DPSCs-NP and DPSCs-IP in immunocompromised mice.

**Table 5.1** Characteristics of dental stem cell types.

| Stem cells (reference) | Positive CD antigen expression | Negative CD antigen expression | Other representative markers | PD | *In vitro* differentiation capacity | *In vivo* tissue formation capacity |
|---|---|---|---|---|---|---|
| **DPSC** (18, 49, 70, 98, 132, 133) | CD9<br>CD10<br>CD13<br>CD29<br>CD44<br>CD49d<br>CD59<br>CD73<br>CD90<br>CD105<br>CD106<br>CD146<br>CD166 | CD14<br>CD31<br>CD34<br>CD45<br>CD117<br>CD133 | STRO-1<br>Nestin<br>OCT4 | >120 | Odontogenic<br>Osteogenic<br>Adipogenic<br>Chondrogenic<br>Myogenic<br>Neurogenic | Dentin<br>Pulp<br>Adipose<br>Muscle |
| **SCAP** (18, 70, 71) | CD49d<br>CD51/61<br>CD56<br>CD73<br>CD90<br>CD105<br>CD106<br>CD146<br>CD166 | CD14<br>CD18<br>CD34<br>CD45<br>CD117<br>CD150 | STRO-1<br>Nestin<br>Survivin | >70 | Odontogenic<br>Adipogenic<br>Neurogenic | Dentin<br>Pulp |
| **PDLSCs** (18, 86, 88, 97, 98, 134) | CD9<br>CD10<br>CD13<br>CD29<br>CD44<br>CD49d<br>CD59<br>CD73<br>CD90<br>CD105<br>CD106<br>CD146<br>CD166 | CD31<br>CD34<br>CD45 | STRO-1<br>Scleraxis | >50 | Cementogenic<br>Osteogenic<br>Adipogenic<br>Chondrogenic<br>Neurogenic | Cementum<br>Periodontal ligament<br>Alveolar bone |
| **GMSCs** (117, 118) | CD29<br>CD73<br>CD90<br>CD105<br>CD146<br>CD166 | CD34<br>CD45<br>CD117 | STRO-1<br>SSEA-4<br>OCT4<br>NANOG | ND | Osteogenic<br>Adipogenic<br>Chondrogenic<br>Neurogenic | Bone-like |
| **DFSCs** (18, 98, 135–137) | CD9<br>CD10<br>CD13<br>CD29<br>CD44<br>CD49d<br>CD59<br>CD73<br>CD90<br>CD105<br>CD106<br>CD166 | CD31<br>CD34<br>CD45<br>CD133 | STRO-1<br>HLA class 1 | ND | Odontogenic<br>Cementogenic<br>Osteogenic<br>Adipogenic<br>Chondrogenic<br>Neurogenic | Cementum<br>Periodontal ligament<br>Alveolar bone |

DFSCs, dental follicle stem cells; DPSCs, dental pulp stem cells; GMSCs, gingival mesenchymal stem cells; ND, not determined; PD, population doubling; PDLSCs, periodontal ligament stem cells; SCAP, stem cells from the apical papilla.

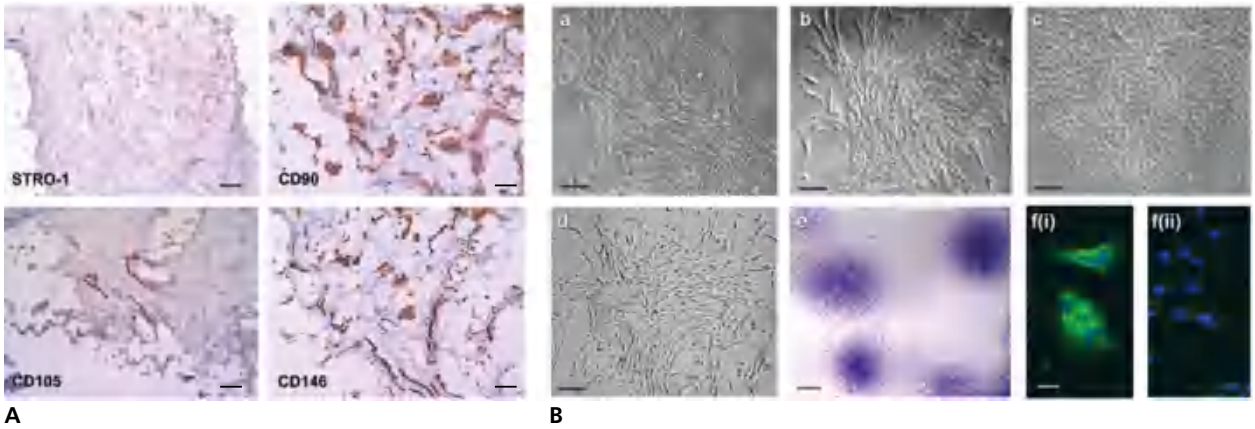



**Fig. 5.4** Marker expression by dental pulp stem cells (DPSCs). A. Immunohistochemical analysis of MSC markers STRO-1, CD90, CD105 and CD146 in normal pulps. Brown stains indicate the marker expression. Scale bars in all four images: 100 µm. B. Clonogenic DPSCs in cultures forming CFU-Fs at passages 0–4; (a) is from normal pulp and (b–e) from inflamed pulp of different donors. (e) Cell fixed and stained with toluidine blue. (f) (i) STRO-1 fluorescence staining (green) and DAPI (4′,6-diamidino-2-phenylindole) nuclear staining (blue) of seeded DPSCs from inflamed pulp. (ii) Non-immuned isotype control of the same cells in (i). Scale bars: (a–d) 200 µm; (e) 1 mm; (f) 20 µm. Adapted from (57) with permission with permission of Future Medicine Ltd.

Regeneration of pulp- and dentin-like tissues by DPSCs has been reported in an emptied canal space in both small and large animal models (9, 63). After being transplanted into the subcutaneous tissue of mice, DPSCs were capable of *de novo* synthesis of vascularized human pulp-like tissue with a layer of dentin-like tissue in an emptied human root canal space (6–7 mm long) with an enlarged root canal (2–3 mm in diameter) having one end sealed with mineral trioxide aggregate (MTA) cement (63). Furthermore, the transplantation of $CD105^+$ DPSCs in a dog root canal after pulpectomy resulted in complete pulp regeneration, including nerves and vasculature, followed by new dentin formation along the dentinal wall (9).

However, the nature of the newly deposited mineralized tissue remains to be determined in terms of its mechanical and chemical properties. The characteristics of the regenerated dentin-like tissue are quite different from natural dentin. First, tubular dentin is lacking except in some areas where convoluted tubules can be seen. Second, it is quite cellular; and third, the formation is not synchronous. During natural primary dentin formation, each dentinal tubule and the surrounding dentin material is gradually produced by one odontoblast. Therefore, each odontoblast occupies one dentinal tubule. However, during the regeneration process, dentinal tubules in the existing dentin are already occupied. The question is whether there are enough newly differentiated odontoblasts to occupy the dentinal tubules. There may be some dentinal tubules that are not occupied and some odontoblasts might fill more than one dentinal tubules; forming a highly organized new dentin is therefore unlikely (64). One important note regarding pulp capping procedures performed in the clinic or in animal studies is that it is still not certain whether the dentin bridge formed under the capping material is tubular or not (65–68). The dentin bridge formed in the regenerative process underneath the MTA appears to lacks tubules (64).

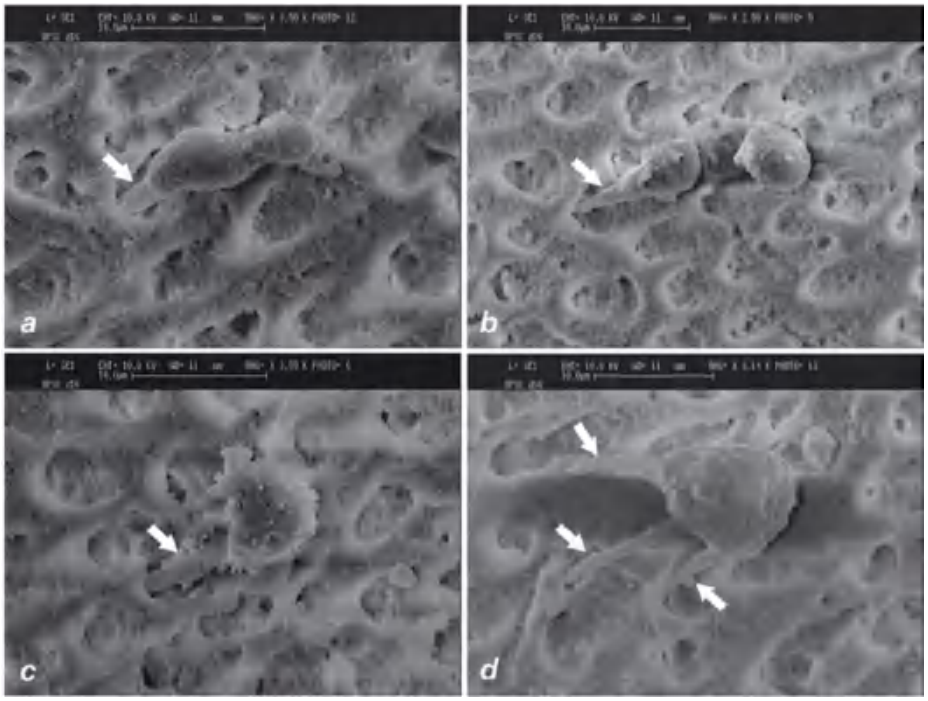
With respect to innervation, DPSCs have been shown to either produce neurotrophic factors or possess neural differentiation potential (20, 69). Regenerated pulp in dog models has shown reinnervation (9). It has been speculated, however, that if the newly regenerated dentin does not have organized dentinal tubules, it is unlikely to perform hydrodynamic activities even if the sensory nerve endings are able to innervate the new dentin. Therefore, normal dentin sensitivity may not occur (53).

In summary, vascularized and innervated pulp can be regenerated with new dentin-like deposition on existing dentin walls with a stem/progenitor cell-mediated tissue engineering approach. In the context of traumatic injury, one can utilize this technology for teeth that have lost pulp due to luxation or avulsion. The regeneration process for luxated or avulsed teeth is likely similar to that for regular teeth that have lost pulp. After repositioning or replantation, if the pulp underwent necrosis, a regeneration approach may be considered after a thorough prognostic evaluation. Nonetheless, further investigation is still needed to optimize various conditions related to this process before a reliable clinical protocol can be established for pulp-dentin regeneration.

## Stem cells from apical papilla

### Localization of SCAP *in vivo*

In 2006, Sonoyama et al. isolated a population of dental stem cells from the apical papilla, termed SCAP (70) (Fig. 5.6). The apical papilla is a connective tissue that is loosely attached to the developing root and contains less blood vessels and cellular components than the pulp tissue. Apical papilla is separated from pulp tissue by a cell-rich zone (70–72). Since its discovery, many studies have focused on the cellular and molecular aspects of the cells, however limited reports have shown their location *in situ*. STRO-1 was first used as a marker by Sonoyama et al. to identify SCAP in the apical

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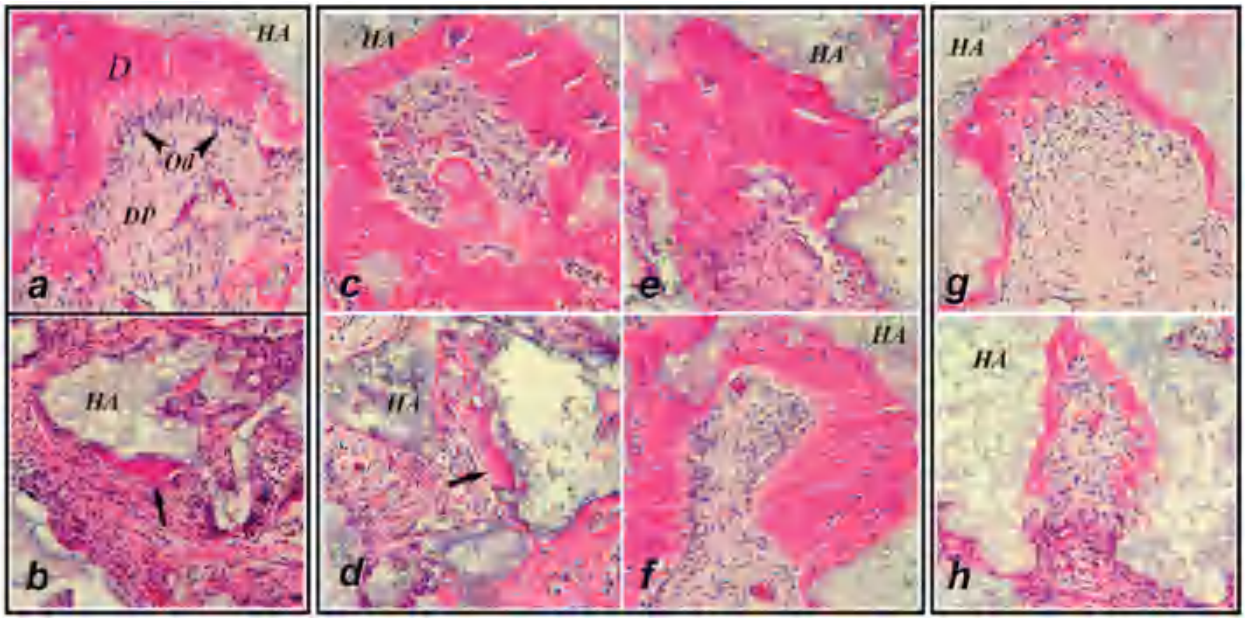



B

**Fig. 5.5** Dental pulp stem cell (DPSC) differentiation into an odontoblast-like phenotype. A. Scanning electron microscope analysis of odontoblast-like cells on a dentin surface *in vitro*. Cells were seeded onto the dentin surface (dentin disks) with open dentinal tubules. Odontoblast-like cells subsequently formed and showed cellular processes extending into the dentinal tubules. (a–c) Each odontoblast-like cell derived from human DPSCs extends one process (arrow) into one dentinal tubule. (d) One odontoblast-like cell derived from a human DPSC extends three observable cellular processes, and each process extends into one dentinal tubule (arrows). (a, b) Adapted from (56) with permission; (c,d) from (138) with permission. B. *In vivo* formation of pulp-dentin complexes by transplanted DPSCs. Cells mixed with HA/TCP were subcutaneously transplanted into Begie XIDIII nu/nu mice. After 8 weeks, the resected tissues were processed for H&E staining. (a) DPSCs from normal pulps as the control. DP: dental pulp-like. (b–h) DPSCs from inflamed pulps. Adapted from (57) with permission with permission of Future Medicine Ltd.

papilla and was found to be mainly perivascular with some scattered cells in the tissue (70) (Fig. 5.7A). Other MSC markers CD105, CD73 and CD146, as well as NOTCH3 and CXCR4, were also used to locate SCAP in apical papilla. Double staining with CD146 and NOTCH3 showed that they are mainly located perivascularly (73). Double staining with CXCR4 and vWF or CXCR4 and STRO-1 all showed these markers to be associated with blood vessels, suggesting the likelihood that SCAP are of perivascular origin (73–75) (Fig. 5.7).

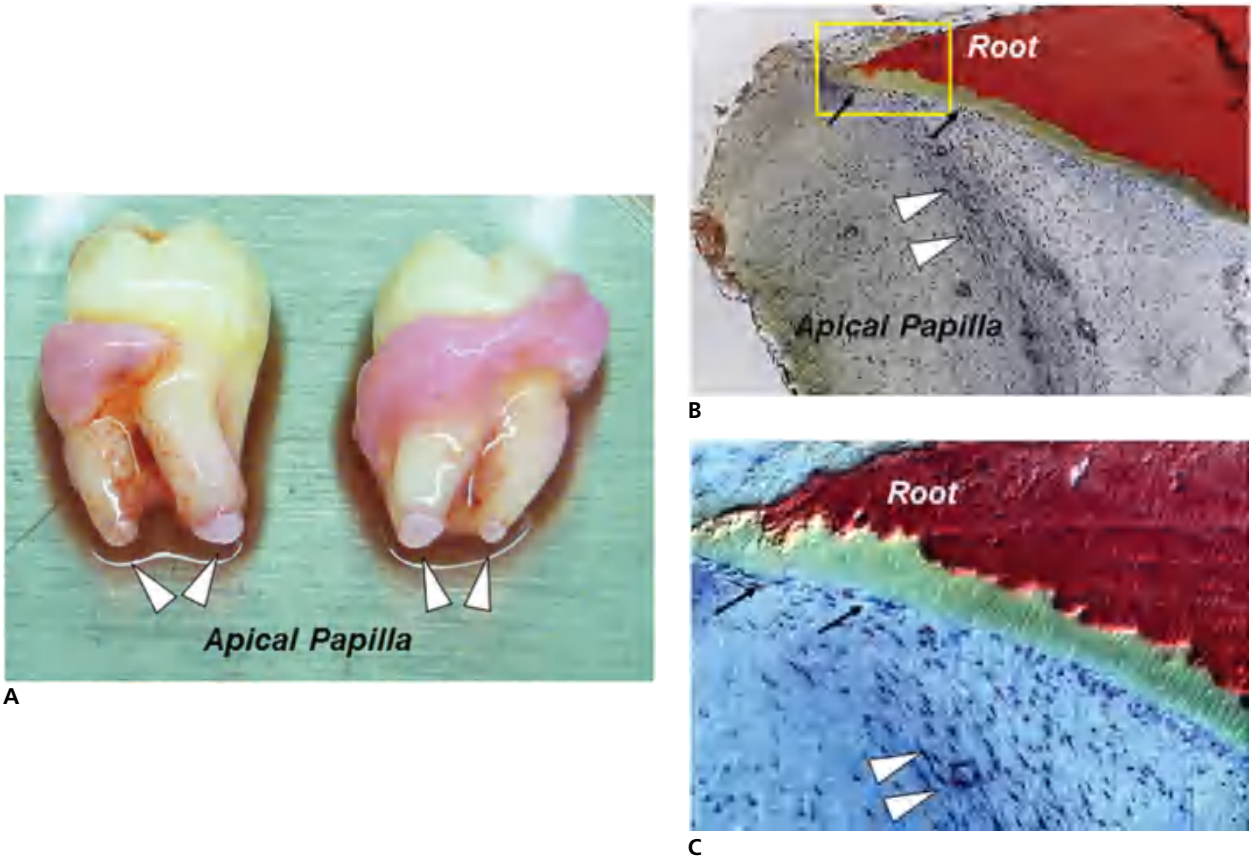



**Fig. 5.6** Apical papilla. A. Extracted human third molars depicting immature roots with apical papillae at the apices (arrowheads). B. A developing root tip with attached apical papilla was cultured *in vitro* for 3 days before being processed for H&E staining. Odontoblasts (black arrows), apical cell-rich zone (open arrowheads) and apical papilla tissue are indicated. C. Magnified view of the area indicated by the yellow rectangle. Adapted from (72) with permission of Elsevier.

## Characterization of SCAP *in vitro*

SCAP isolated from apical papilla are clonogenic fibroblast-like cells, having a higher proliferation rate than DPSCs and PDLSCs (70, 71, 76). SCAP express several mesenchymal surface markers such as CD24, CD105, CD73, CD90, STRO-1 and CD146. Other markers expressed by SCAP include NANOG, OCT3/4, SSEA-3, nestin, SOX9, P75, JAGGED-1 and NOTCH3 (70, 71, 73, 77, 78) (Table 5.1). Cultured SCAP can undergo osteogenic, adipogenic, chondrogenic and neurogenic differentiation when they are induced by respective differentiation mediums as evidenced by lineage-specific marker expression (18, 70). However, gene marker expression does not equate with functionality. In general, SCAP are strong in osteogenic/odontogenic or neurogenic differentiation, while weak on other lineages.

As with other MSCs, SCAP is a heterogeneous population of cells with different subpopulations. Jamal et al. reported the presence of $CD146^+NOTCH^+$, $CD146^+STRO\text{-}1^+$ and $CD146^+NOTCH3^+STRO\text{-}1^+$ subpopulations. Interestingly, SCAP lose their NOTCH3 expression after undergoing osteogenic differentiation (73). Bakopoulou et al. reported the presence of $STRO\text{-}1^-CD146^+$ and $STRO\text{-}1^+CD146^+$ subpopulations with higher CFU efficiency and $STRO\text{-}1^+CD146^+$ subpopulation with greater odontogenic potential (78).

## Implication of SCAP for regenerative endodontics

The identification of apical papilla and discovery of SCAP opened the field of regenerative endodontics (72, 79). Revitalization (revascularization) clinical protocols have been established and many case reports have shown that such protocols may achieve clinical success in terms of healing and maturation of the root (80, 81). However, histologic findings have shown that the generated hard and soft tissues in the canal were periodontal tissues including bone, cementum and periodontal ligament-like fibrous tissues (82, 83). It was hoped that if the apical papilla is still viable at the time of treatment, SCAP may be attracted into the canal and regenerate pulp and form new dentin using molecular approaches (64, 74) (Fig. 5.8). One idea was tested using the stem cell attractant stromal cell-derived factor-1α (SDF-1α) to chemoattract SCAP expressing the SDF-1α receptor CXCR4 (74). An *in vitro* device was utilized to demonstrate that SDF-1α was able to induce SCAP to migrate in a three-dimensional collagen gel (Fig. 5.9). This finding suggests that the hypothetical clinical strategy depicted in Fig. 5.8 may work.

As capable as DPSCs, SCAP form a dentin-pulp complex *in vivo* when mixed with HA/TCP and transplanted into immunocompromised mice (70). Using a tooth root

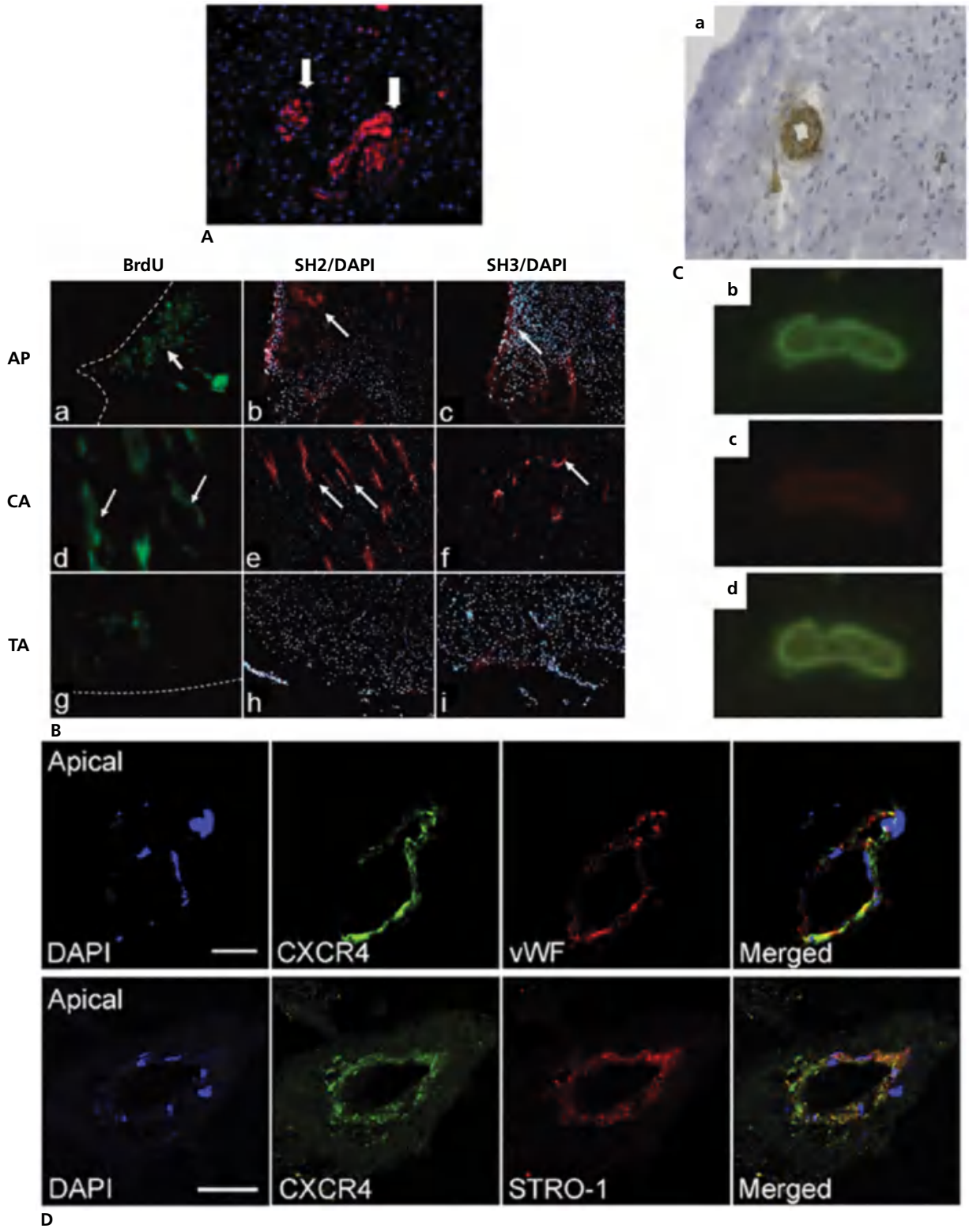



**Fig. 5.7** Mesenchymal stem cell (MSC) marker expression in apical papilla. A. Human apical papilla tissue was positive for STRO-1: red immunofluorescence (arrows). Adapted from (70). B. Immunohistofluorescence analysis of apical papilla of human immature tooth. (a–c) Apical papilla; (d–f) center of apical papilla; (g–i) tip of apical papilla. (a,d,g) BrdU-labeled cells; (b,e,h) MSC marker CD105; (c,f,i) expression of MSC marker CD73. (b,c,e,f,h,i) DAPI staining. (b,c,e,f,h,i), 100×; (a,d,g), 200×. Adapted from (139) with permission. C. Detection of NOTCH3 and CD146 in human apical papilla. (a) Immunohistochemical staining of the apical papilla, showing NOTCH3 staining is associated with blood vessels; (b–d) immunofluorescense staining of the apical papilla showing blood vessels stained for (b) NOTCH3 (green), (c) CD146 (red) and (d) co-localization of NOTCH3 and CD146. Adapted from (73) with permission. D. CXCR4 expression in apical papilla. (Top panel) Immunocytofluorescence double staining of the apical papilla showing CXCR4 (green) co-expressed with von Willebrand factor (vWF) (red). CXCR4 in the apical papilla was expressed in the paravascular region where vWF-positive endothelial cells were also present. (Lower panel) Immunofluorescence analysis of CXCR4 (green) and STRO-1 (red) in apical papilla. CXCR4-expressing cells in the apical papilla were also immunoreactive to anti–STRO-1 antibodies. Cell nuclei exhibited blue fluorescence by DAPI counterstaining. Scale bar: top, 20 µm; bottom, 50 µm. Adapted from (74) with permission.

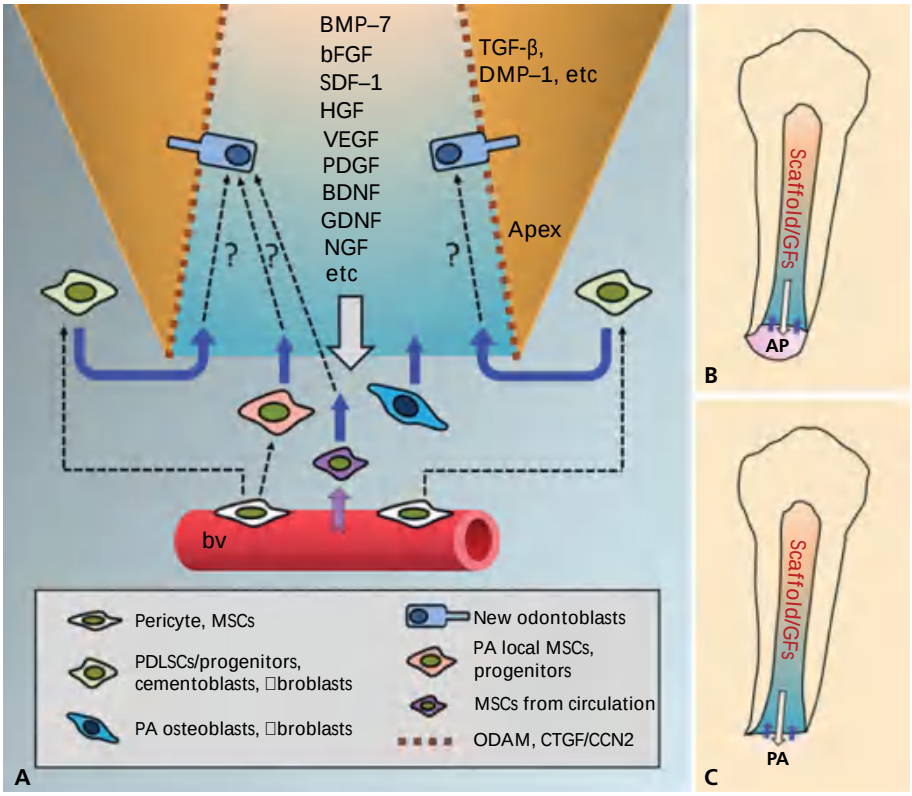
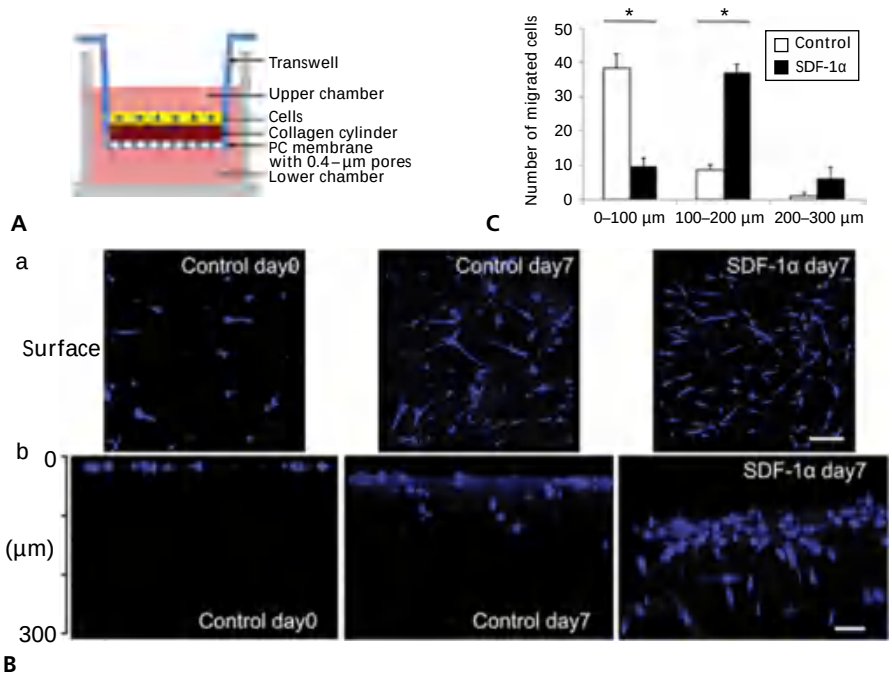



**Fig. 5.8** A conceptual theme of a non-cell-based revitalization/regeneration procedure. A. The root canal space is filled with a scaffolding material carrying growth factors (GFs), which are released into the adjacent tissues to induce stem/progenitor cell migration into the canal space and to guide the differentiation of the migrated cells into odontoblast lineages. The white arrow pointing down indicates the release of GFs into the apex. Purple and blue arrows pointing up are the cell migration direction into the canal. Dashed arrows indicate: (1) the perivascular pericytes at the local tissues, including periapical bone and periodontal ligament, giving rise to local MSCs and progenitors; and (2) cells migrating into the canal space and transdifferentiating into new odontoblasts. B. If the apical papilla (AP) is still present, cells in this tissue (e.g. stem cells in the AP) may be attracted into the canal space along with more distant cells from blood. C. If the AP is no longer present, periapical (PA) cells and distant cells from blood may be attracted into the canal space. Odontogenic ameloblast-associated protein and CTGF/CCN2 (connective tissue growth factor/CCN family 2) may be coated onto the dentin wall to facilitate odontoblast differentiation. TGF-β and DMP-1 embedded in dentin may be guiding odontoblast differentiation and new dentin generation. BDNF, brain-derived neurotrophic factor; bFGF, basic fibroblast growth factor; BMP, bone morphogenetic protein; bv, blood vessel; DMP-1, dentin matrix acidic phosphoprotein 1; GDNF, glia cell line-derived neurotrophic factor; HGF, hepatocyte growth factor; NGF, nerve growth factor; SDF-1, stromal cell-derived factor 1; TGF-β, transforming growth factor β. Reproduced from (64) with permission.

**Fig. 5.9** Chemotactic recruitment of stem cells from apical papilla (SCAP) into a 3D collagen gel Transwell model. A. Schematic diagram of the Transwell model (0.4 mm pore). PC, polycarbonate. B. (a) x–y axial planes of 3D reconstructed images. SCAP were sparsely seeded onto the 3D cylindrical collagen gel surface on day 0. By day 7, the cells had undergone significant proliferation. (b) Cell migration depth in collagen gel. More cells migrated into deeper levels in the SDF-1α group (100 ng/mL) than those in the control group (non-treated). Cell nuclei were counterstained by DAPI (blue fluorescence). Images are representative data of independent experiments with consistent results. Scale bar: (a) 100 μm; (b) 50 μm. C. Quantitative analysis of migration depth. The delivery of SDF-1α recruited significantly more cells into much deeper levels (mainly 100–200 μm) in collagen gel than the negative control (*, $P<0.05$ compared with negative control). Reproduced from (74) with permission.



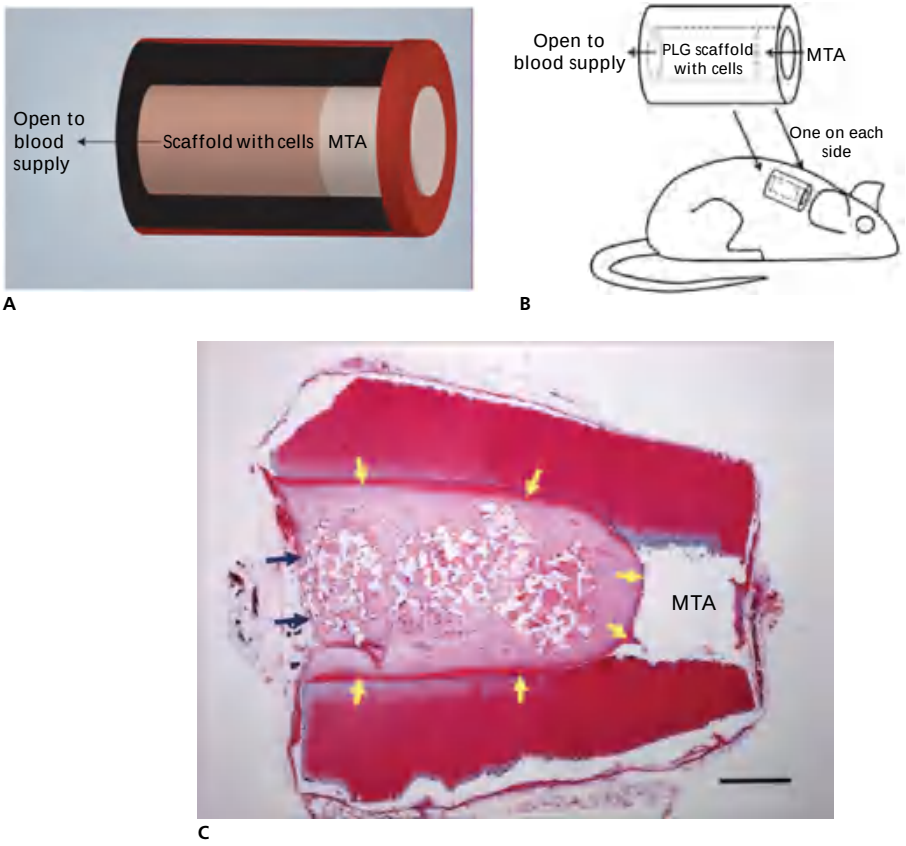



**Fig. 5.10** Proof of principle engineering of dental pulp tissue with human dental pulp stem cells using immunocompromised mice. A. A human tooth root fragment with enlarged canal space was generated. One end of the canal opening was sealed with mineral trioxide aggregate (MTA) cement and the canal filled with poly(D-L-lactide-co-glycolide) (PLG) scaffold seeded with stem cells. B. The construct was transplanted into the subcutaneous space of severe combined immunodeficiency (SCID) mice. The implanted tooth fragment containing cells/scaffold was removed after several months in the subcutaneous space of SCID mice and processed for analysis. C. A longitudinal section of the decalcified sample was stained with H&E, showing pulp-dentin regeneration using human stem cells from apical papilla seeded in PLG. Dark blue arrows indicate the demarcation between the subcutaneous tissue from the mouse and the regenerated pulp-like tissue in the canal. Yellow arrows indicate the newly deposited dentin-like mineral tissue onto the canal dentinal walls and MTA surface. Scale bar: 1 mm. Reproduced from (53, 63) with permission.

fragment model, a root canal space was filled with a scaffold seeded with SCAP. This resulted in the regeneration of new, vascularized, pulp-like tissue with the deposition of a new layer of dentin-like tissue, thus demonstrating the regenerative potential of SCAP (63) (Fig. 5.10).

## Periodontal ligament stem cells

### Localization of PDLSCs *in vivo*

Like most tissues of the body, the periodontal ligament (PDL) contains progenitor cells able to maintain the homeostasis and regeneration of the periodontium. It is well known that PDL contains MSCs with the potential to differentiate into fibroblasts, osteoblasts and cementoblasts (84, 85). Seo et al. were the first to identify the presence of STRO-1$^+$ cells in human PDL tissue and to isolate a population of clonogenic cells from the PDL of impacted third molars with characteristics of adult stem cells (86). Subsequently, Chen et al. located PDLSCs in human PDL using STRO-1, CD146 and CD44 markers; they also compared the positively stained cells in the PDL of healthy and periodontitis-affected teeth (87). All three markers were found to be expressed in the PDL, but only from some of the tooth samples. The markers were expressed perivascularly as well as away from the blood vessels in healthy PDL; while in diseased teeth they were found in the perivascular and extravascular areas and along or near the cementum surface (Fig. 5.11).

### Characterization of PDLSCs *in vitro*

After isolation from the tissue and being grown in cultures, PDLSCs form colonies in culture (86, 88) (Fig. 5.12). PDLSCs express many MSC-associated markers including STRO-1, CD29, CD44, CD73, CD90, CD105 and CD166 (86, 88–94) (Table 5.1). They express two endothelial cell markers, CD106/VCAM-1 and CD146/MUC18, which reflects their origin in the perivascular area; as well as a transcription factor, scleraxis, specific to tendon (86, 88, 95, 96). In addition, PDLSCs express embryonic stem cell markers OCT4, NANOG, SSEA-1 and SSEA-4 (91, 92). Like other MSCs, they possess multilineage differentiation potential. Under specific growth conditions they can undergo osteogenic, adipogenic, chondrogenic and neurogenic differentiation (88, 97–100) (Fig. 5.12). Their ability to differentiate can vary from other MSCs. In fact, the osteogenic differentiation potential of PDLSCs seems to be less than that of BMMSCs (89) and DPSCs (86).

In most studies, PDLSCs are easily grown and isolated from the healthy PDL of third molars or premolars extracted for orthodontic reasons. Although this seems straightforward, obtaining healthy PDL cells can be challenging in clinical situation especially if the PDL is damaged or unhealthy. PDLSCs may be isolated from other sources. The CD105-enriched

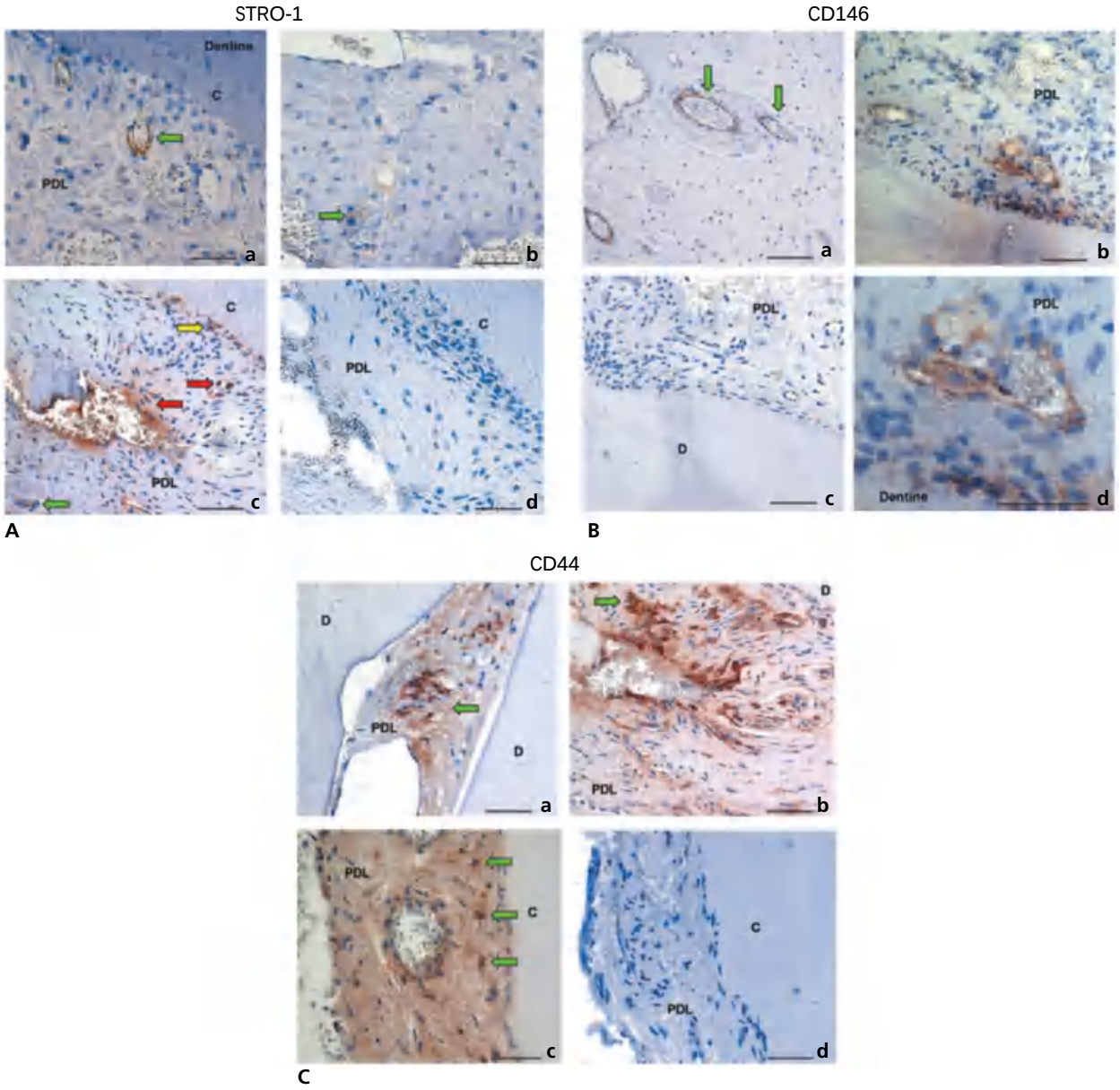



**Fig. 5.11** Mesenchymal stem cell (MSC) marker expression in human periodontal ligament (PDL). A. STRO-1 surface antigen in the PDL. (a,b) Staining occurred around the perivascular in healthy PDL. (c) STRO-1 surface antigen in the apical PDL of a periodontitis-affected tooth. STRO-1 surface antigen was widely distributed in the diseased sections compared with the healthy section. Apart from the positive staining in the perivascular area (red arrows), a small cluster of cells was also observed in the extravascular area (green arrow) and cementum (C; yellow arrow). Cells with different morphologies can be observed. (d) Control section of STRO-1. Scale bars: (a–d) 50 µm. B. CD146 antigen in the PDL. (a) Healthy tooth; CD146 antigens were mainly located in the perivascular area. Scale bar: 50 µm. (b) Diseased tooth; CD146 antigen can be observed in the perivascular space as well as in the area near the cementum. Scale bar: 50 µm. (c) Control section of a diseased tooth. Scale bar: 50 µm. (d) Diseased tooth. A higher magnification of (b) of the cells positive for CD146 staining highlighted their morphologies of small round nuclei with small amounts of cytoplasm. Scale bar: 25 µm. C. CD44 antigen in the PDL. (a) Healthy tooth. Scale bar: 50 µm. (b) Diseased tooth; the intense CD44 staining around the perivascular areas and the extravascular areas (green arrow) were consistent with STRO-1 staining. Scale bar: 50 µm. (c) Diseased tooth. Green arrows show cells with CD44 antigen along the region near the cementum. Scale bar: 25 µm. (d) Control section from healthy tooth. Scale bar: 50 µm. C, cementum; D, dentin. Reproduced from (87) with permission.

population of stem cells isolated from deciduous teeth is able to differentiate into osteogenic/cementogenic as well as adipogenic lineages and has a greater proliferation rate than their permanent teeth counterparts (94).

One source of PDLSCs is from the alveolar socket. After tooth extraction only part of the PDL is attached to the root surface, while the rest is still attached to the surface of the alveolar bone (101). The characteristics of the PDLSCs isolated from the alveolar portion of the PDL were compared to the PDLSCs isolated from the root surface (102). More stem cells possessing long-term proliferation potential were found on the alveolar bone side. The PDLSCs isolated from the

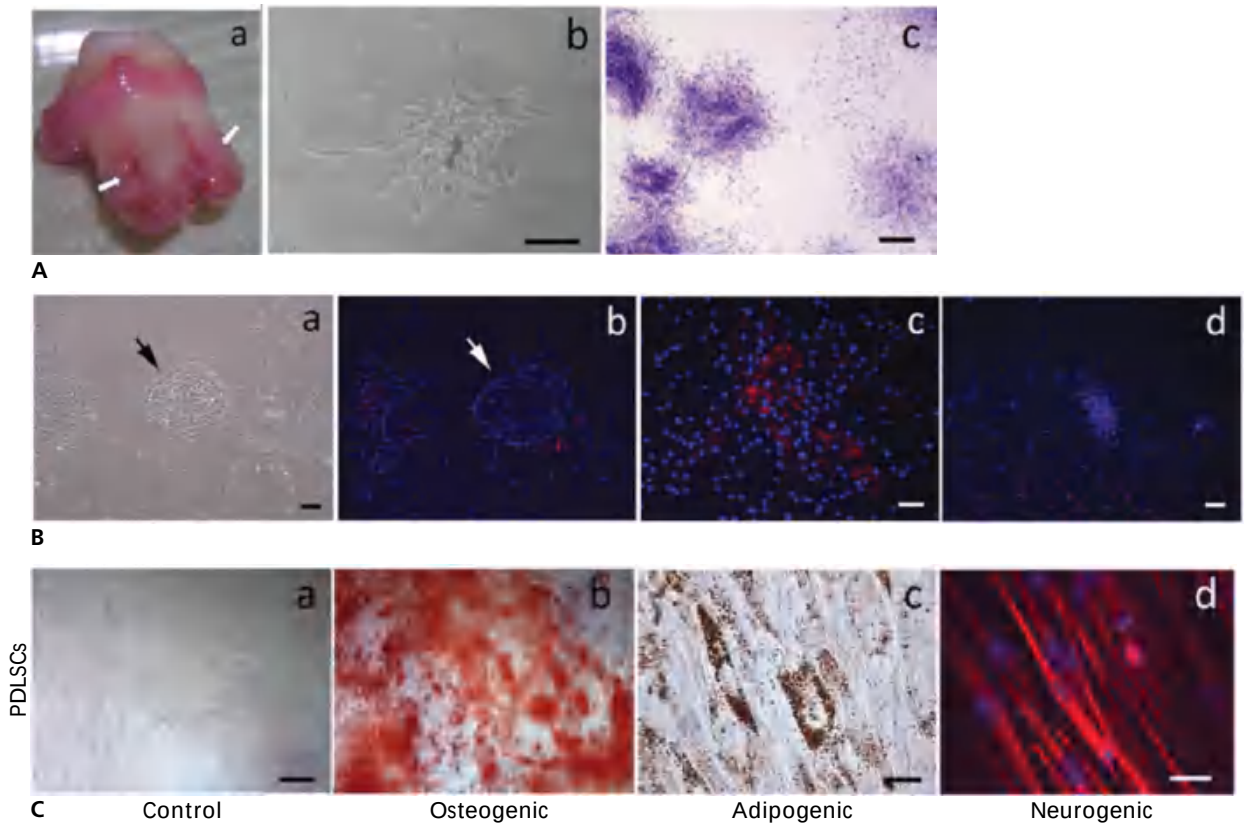



**Fig. 5.12** Isolation and characterization of heterogeneous human periodontal ligament stem cells (PDLSCs). A. (a) Image of a freshly extracted third molar. Arrows indicate PDL to be isolated. (b) Colony formation after initial seeding (passage 0). Scale bar: 100 µm. (c) PDLSC colonies formed at passage 1 fixed and stained with toluidine blue. Scale bar: 1 mm. B. (a) Initial seeding (passage 0) showing an island of epithelial cells (arrow), a CFU-F (on the left) and scattered single cells. Scale bar: 200 µm. (b) STRO-1 staining (red fluorescence) of the culture shown in (a). Notice the epithelia island is STRO-1 negative. The cell nuclei were stained with DAPI (blue). (c) Single STRO-1 positive PDLSC CFU-F (passage 0). Scale bar: 100 µm. (d) STRO-1 positive PDLSC CFU-F and in scattered cells (passage 2). Scale bar: 200 µm. C. Induced differentiation of PDLSCs at passages 2–3. (a) Controlled groups without stimulation; (b) osteogenic induction after ~6 weeks followed by Alizarin red stain showing mineral deposits; (c) adipogenic induction for ~6 weeks followed by oil-red stain showing oil droplet-filled adipocyte-like cells; (d) neurogenic induction for 4 weeks followed by staining of βIII tubulin (red fluorescence). Scale bars: (a, b) 200 µm; (c) 100 µm; (d) 20 µm. Adapted from (88) with permission.

alveolar socket express high levels of MSC markers and have greater differentiation potential than those isolated from the root side. These cells could represent a promising source of stem cells when a tooth extraction is needed.

Other studies have focused on the stem cells available in the inflamed human periodontal ligament (ihPDLSCs) (103). When the extraction of a tooth with healthy PDL is not possible, inflamed periodontal tissue could be another source of PDLSCs. This was shown by Chen et al., as mentioned earlier, who identified cells expressing MSC markers in the perivascular area and extravascular area of inflamed PDL (87). PDLSCs and ihPDLSCs express the same stem cell markers such as CD146 and STRO-1, which have multipotent cell differentiation potential. However, ihPDLSCs have a lower osteogenic and chondrogenic differentiation capacity. They also have impaired immunomodulation compared to PDLSCs from healthy PDL when grown in co-culture with peripheral blood mononuclear cells (104, 105).

Since the stem cells might not be available when needed, cryopreserving them beforehand until they are needed is a necessary step. It has been shown that 40% of PDLSCs could be isolated from frozen PDL tissue compared to fresh PDL (106, 107). The isolated cells maintained stem cell characteristics and an ability to form cementum and PDL-like tissue *in vivo*.

## PDLSCs in periodontal regeneration for avulsed teeth

*In vivo*, PDLSCs mixed with HA/TCP particles transplanted in immunocompromised mice form tissue with the characteristic of PDL (86, 106). In this type of experiment, a layer of cementum-like mineralized tissue is formed at the surface of the HA/TCP. The cementum is covered by a layer of cementoblast-like cells. Dense type 1 collagen-positive PDL-like tissue, with the presence of structures resembling Sharpey's fibers that connect with the newly formed cementum, can be observed (Fig. 5.13). Under these conditions, PDLSCs behave differently to BMMSCs and DPSCs when transplanted with HA/TCP. In comparison, BMMSCs produce mineralized tissue with the presence of bone, bone marrow, osteoblasts and osteocytes, while DPSCs produce

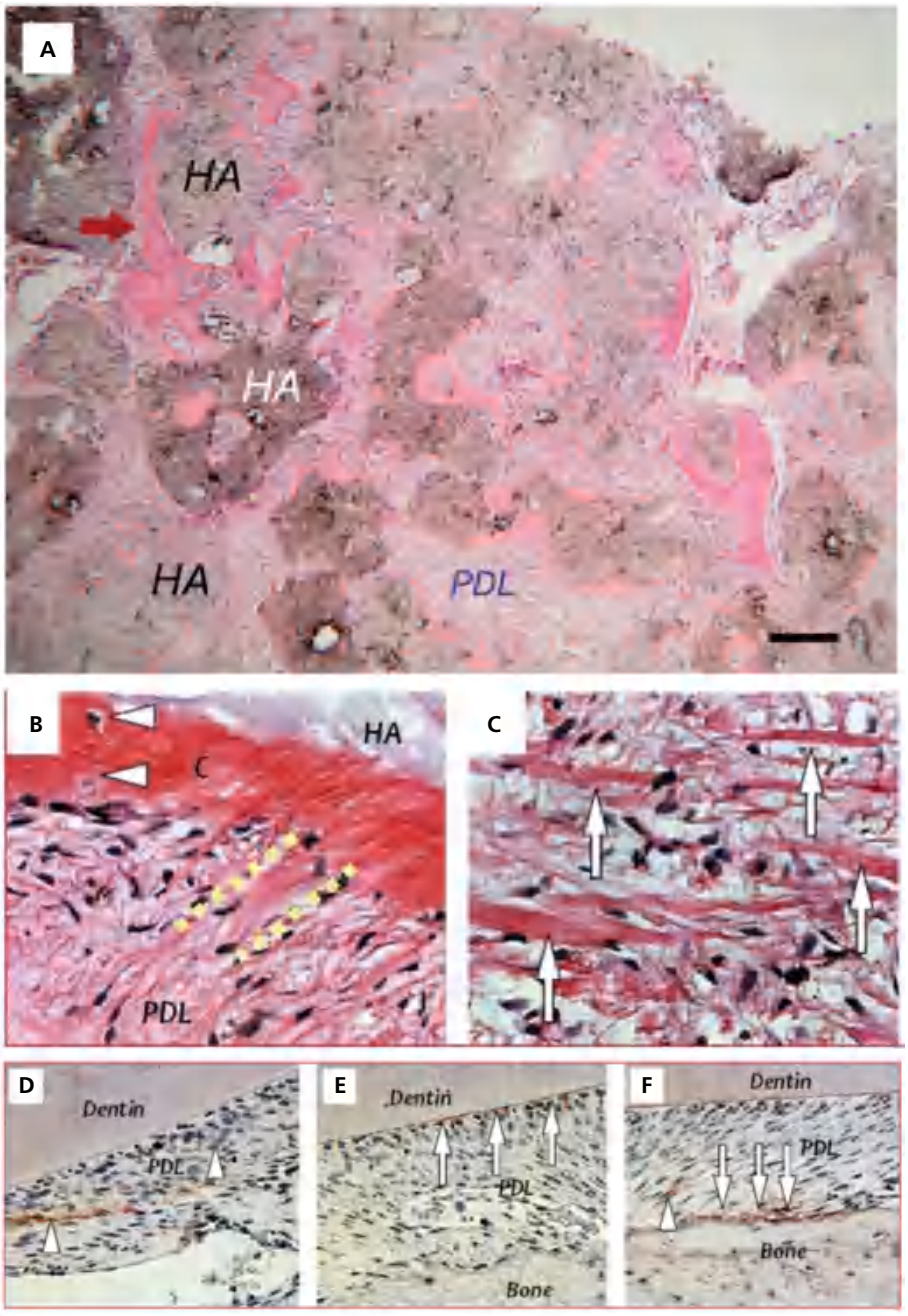



**Fig. 5.13** Formation of ectopic mineral tissues *in vivo* by human periodontal ligament stem cells (PDLSCs). A. Cells mixed with HA/TCP were transplanted into the subcutaneous space of SCID mice. After 3 months, the transplants were harvested for histologic analysis. Mineral tissue formation (red arrow) by the PDLSCs was found around the HA/TCP granules. Scale bar: 200 µm. B, C. Cementum-like structure and collagen fibers formed by PDLSCs in immunocompromised mice (yellow dashed lines in B and arrows in C), similar to the structure of Sharpey's fibers. D–F. PDLSCs in periodontal tissue repair in immunocompromised rats. Staining of human-specific antimitochondria antibody shows human PDLSCs located in the PDL compartment (arrowheads in D), involved in the attachment of PDL to the tooth surface (arrows in E), and participating in the repair of alveolar bone (arrows in F) and PDL (arrowhead in F). B–F adapted from (86).

pulp-like tissue with the presence of ondontoblast and dentin-like tissue.

There is a potential use of PDLSCs to help regenerate PDL in avulsed teeth. Cell-based therapy could theoretically prevent or lower the incidence of resorption following tooth replantation, especially when extended extraoral time has occurred. In a dog model using PDLSCs, cell sheets wrapped around the root before replantation may enhance the healing area and reduce ankylosis/resorption (108). Combining PDLSC cell sheet fragments and platelet-rich fibrin (PRF) granules also supports healing (109). PRF is known to release growth factors and can be used as a scaffold to introduce *ex vivo* cultured PDLSCs into the alveolar socket before replantation of the tooth. With this technique, an organized PDL tissue with well-oriented fibers and newly formed cementum on most parts of the roots was observed 8 weeks after surgery in dogs. The use of PDLSCs alone also had a good outcome but a combination with PRF seemed beneficial. Replacement resorption ankylosis and infection-related resorption were also significantly reduced in the PDLSCs/PRF group (Fig. 5.14). The majority of the cells that participated in the PDL formation were from the transplanted population, indicating that the transplanted cells actively participate in the regeneration of the tissue. Without using the cell sheet technique, a dog model showed that a tooth may also be transplanted

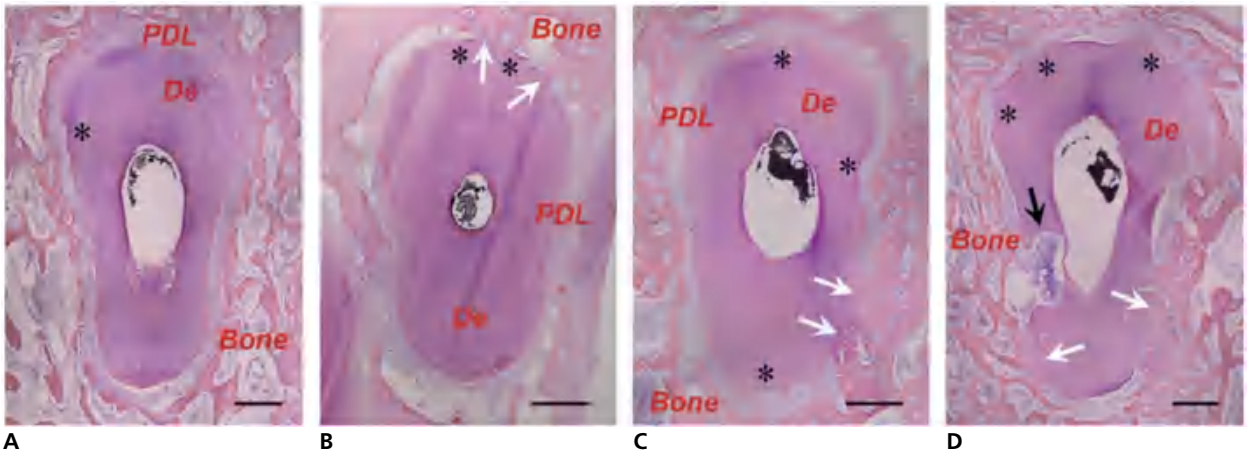



**Fig. 5.14** Representative images of periodontal healing in the different groups in dogs. A. Group I: the ideal regeneration of periodontium was identified that manifested through the formation of well-organized periodontal ligament-like tissues (PDL) with very few places of pathologic resorptions (*). B, C. Group II (B) and Group III (C): small areas of ankylosis (white arrows) and surface resorption (*) of the tooth root could be found. D. Group IV: the negative control group showed large areas of ankylosis (white arrows), surface resorption (*) of the tooth root and severe inflammatory resorption (black arrow). H&E staining. Scale bars: 1 mm. Group I: PDLSC cell sheet and platelet-rich fibrin (PRF); Group II: PDLSC cell sheet alone; Group III: PRF alone; Group IV: no adjuvant. Bone, alveolar bone; De, dentin. Reproduced from (109) with permission.

successfully by coating the root surface with a combination of calcium phosphate and fibronectin, followed by seeding the PDLSCs (110).

These cell-based techniques proved the feasibility of periodontal regeneration to treat avulsion using stem cells in the thin PDL space, which ranges in width between 0.15 and 0.38 mm (109). However, clinical translation could be difficult because of the complexity of the procedure, the time needed to culture the cells before they can be transplanted and the availability of the cells at the time of trauma. Culturing autologous cells in sufficient quantity to be used as a transplant after avulsion will take weeks and the healing of the socket might not allow replantation of the tooth by the time the cells are ready for transplantation. One way to overcome this problem would be using a cell-free approach by promoting endogenous cell homing to promote PDL regeneration. This way there is no need for cell culture because the cells from the host would be mobilized by the use of growth factors at the site where they are needed. A combination of PRF and treated dentin matrix tested in a dog model has shown that PRF may participate in recruitment of the cells and promote their proliferation, leading to PDL regeneration (111). However, PRF used alone without PDLSCs may not show the same adequate fiber orientation as when PDLSCs are used too (109). Moreover, only a very thin layer of cementum or no new cementum formation was present compared to the groups where PDLSCs were used. Therefore, a cell-free approach may not yet offer the same potential for regeneration as the cell-based therapy.

Another way to overcome the problems associated with culturing autologous cells would be using allogeneic PDLSCs which could be isolated, expanded and stored so they could be available when needed at the time of the trauma. Like other MSCs, PDLSCs possess low immunogenicity. Therefore, they do not tend to induce an immune response when used as allogeneic transplants. PDLSCs inhibit T-cell proliferation stimulated by mismatched histocompatibility complex molecules (112). The cells also possess marked immunosuppression of T cells through expression of prostaglandin E2 (PGE2). Allogeneic PDLSC cell sheet transplants in swine do not induce immunologic rejection and can repair bone defects in an experimental model of periodontitis (113). PDLSCs maintain their low immunogenicity and suppression of T cells after osteogenic differentiation (114). Therefore, an immunologic reaction from the host may possibly be prevented even after differentiation of the cells. It has been shown that allogeneic stem cell-based bio-root can be regenerated in a swine model without signs of immunologic rejection (115). These findings indicate that allogeneic PDLSCs may greatly facilitate clinical applications in future dental regeneration therapy.

In human systems, one report has shown that autologous periodontal ligament progenitor cells (PDLPs) may be used to treat periodontally involved teeth with deep intrabony defects (16). Three patients were involved in the study. The diseased roots were treated by transplanting a construct of PDLPs and bone graft directly into the bony defect. After a 72-month follow-up period, all patients showed improved periodontal health compared to presurgical conditions. PDL has also been shown to have regenerated between HA/TCP-coated titanium dental implants and alveolar bone cells (116). On the nine implants placed on humans, three were retained for more than 4 years. PDL and cementum formation was observed as well as increased bone formation in the area of the implant. These human studies, although limited, indicate their use is likely safe for the regeneration of PDL. With current results from large animal models, PDLSC-based therapy may finally provide a breakthrough to salvage avulsed teeth.

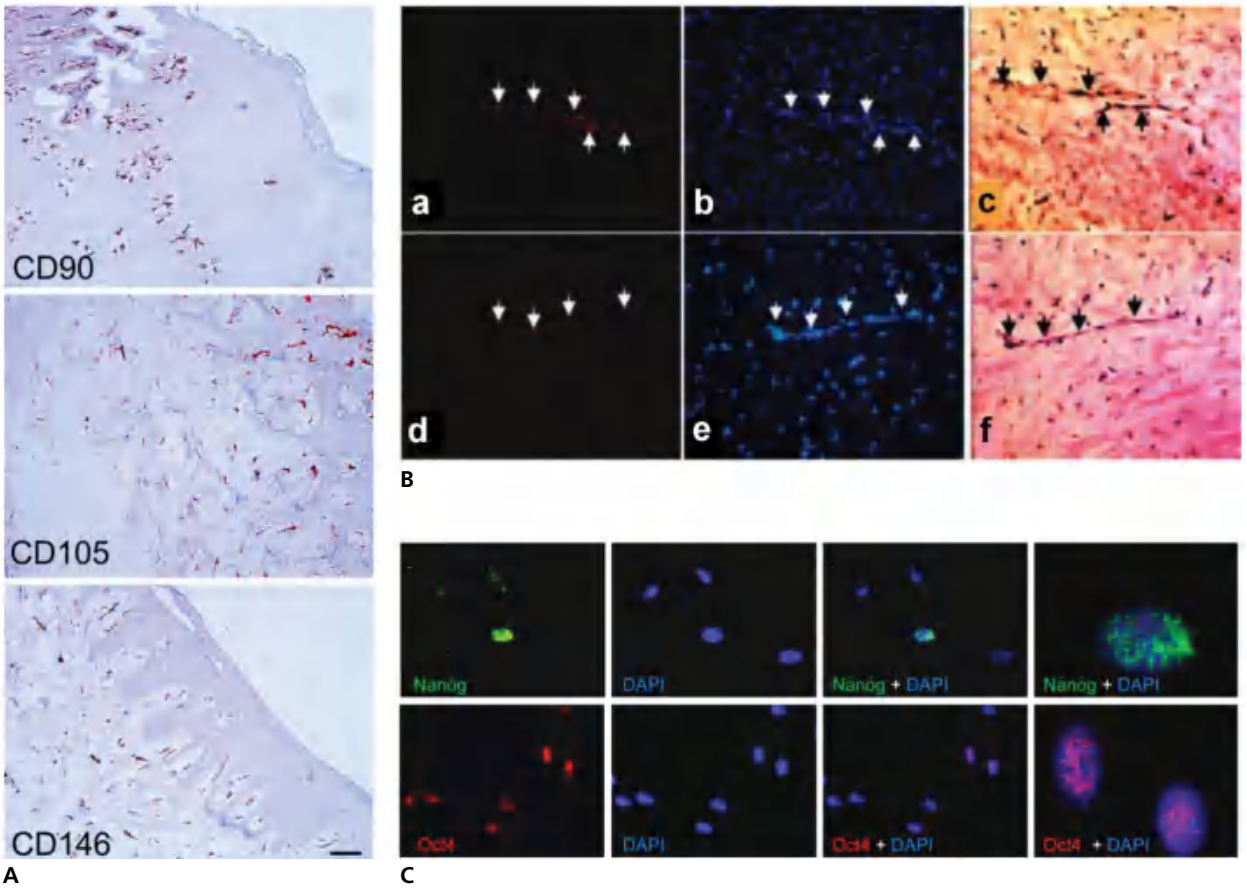



**Fig. 5.15** Mesenchymal stem cell (MSC) marker expression in human gingival tissues. A. Immunohistochemical analysis of CD90, CD105 and CD146 in normal gingiva (brown staining). Scale bar: 100 µm. B. *In vivo* expression of primitive stem cell markers. Immunostaining of oral mucosa lamina propria-derived sections. OCT4 (arrows in a–c) and SOX2 (arrows in d–f) are localized to cord-like structure of high-cellular density. (a,b,d,e) DAPI; (e.f) H&E. C. Immunofluorescent illustrations of the embryonic stem cell markers NANOG and OCT4 in a young donor-derived hOMSC culture 48 hours after plating in expansion medium (original photograph, ×400). Non-reduced photograph (two right panels) illustrates nuclear localization of OCT4 and NANOG (original photograph, ×400). DAPI, 4,6-diamidino-2-phenylindole. Adapted from (118) with permission from John Wiley and Sons.

## Gingival mesenchymal stem cells

### Localization of GMSCs *in vivo*

The location of GMSCs was identified in human gingiva submucosal tissues with the MSC markers CD90, CD105 and CD146 and stem cell markers OCT4, SOX2, SSEA-4 and STRO-1. Their expression appears to be, similar to other MSCs, associated with blood vessels (117, 118) (Fig. 5.15).

### Characteristics of GMSCs *in vitro*

GMSCs form colonies *in vitro* after isolation. Various percentages of GMSCs in culture express stem cell markers SSEA-4, OCT4, NANOG and STRO-1 as well as MSC markers CD29, CD73, CD90, CD105 and CD146 (117, 118) (Table 5.1, Fig. 5.15). GMSCs possess the capacity to differentiate into osteogenic, chondrogenic, adipogenic, endothelial and neural lineages (117, 118). In chondrogenesis in particular, oral MSCs are generally weak and cannot form chondrocyte-like cells in the pellet culture designed to form cartilage in cultures. GMSCs on the other hand appear to be stronger in forming chondrocyte-like cells *in vitro*, besides producing chondrogenic matrix such as sulfated proteoglycans and collagen (119). Human GMSCs appear to be equally osteogenic *in vivo* compared to BMMSCs while having a greater proliferation rate than BMMSCs, displaying stable phenotypes with normal karyotypes and telomerase activity in long-term cultures (120).

### Regenerative potential of GMSCs *in vivo*

As a cell source easy to access, GMSCs can be used to enhance bone regeneration in the context of traumatic injury treatment. *In vivo* orthotopic bone tissue regeneration using GMSCs has been demonstrated by repairing mandibular and critical-sized calvarial defects in Sprague Dawley rats (121). A tissue engineering approach using a silver lactate-containing Arg-Gly-Asp (RGD)-coupled alginate hydrogel scaffold as a GMSC delivery vehicle has shown the possibility for engineering bone with antimicrobial properties against bacteria *in vitro* (122), suggesting

its potential use for tooth replantation. Similar to other MSCs, GMSCs also possess immunomodulatory functions with the ability to suppress peripheral blood lymphocyte proliferation and to induce a wide range of immunosuppressive factors such as interleukin-10, Indoleamine 2,3-dioxygenase (IDO), inducible NO synthase (iNOS) and cyclooxygenase 2 (COX-2) in response to interferon-$\gamma$ (117). This suggests their potential allogeneic use without being rejected by the host.

## Dental follicle stem cells

The dental follicle of the developing tooth contains stem/progenitor cells. They were first termed dental follicle precursor cells (DFPCs) when being isolated from human dental follicles of impacted third molars (123). Later they were verified as having a trilineage differentiation potential as well as neurogenic potential *in vitro* similar to that of other MSCs (123–126). They express STRO-1, NOTCH-1, nestin, CD44, CD73, CD90 and fibroblast growth factor receptor (FGFR)-1-IIIC (123, 124, 127) (Table 5.1), as well as cementum attachment protein and cementum protein-23 if stimulated by enamel matrix derivatives or BMP-2 and BMP-7 (124). They also showed the potential for bone regeneration as evidenced in a rat calvarial defect model (128).

The radicular follicle is located at the apex of the developing tooth and is considered as the periapical follicle that harbors periapical follicle stem cells (PAFSCs) (129, 130). PAFSCs express STRO-1 and CD146, have osteogenic and adipogenic potential, and form cementum/PDL-like tissue *in vivo* (129), similar to the capacity of PDLSCs. The conditioned medium from PAFSCs has been shown to induce osteogenic/dentinogenic differentiation of SCAP in cultures suggesting their potential role in root development *in vivo* (131). Since the dental follicle is the precursor tissue of PDL, it is not surprising that DFSCs have similar characteristics to PDLSCs and their potential clinical use is likely similar.

## Conclusion

Severe traumatic dental injuries usually involve fracture, luxation or avulsion. Some of these conditions may be managed via traditional methods, which may yield optimal outcomes. However, certain conditions require more advanced technologies than are yet available. Stem cell-mediated or other non-cell-based regenerative approaches are needed to address these clinical issues. Various dental/oral stem cells described in this chapter shed light on the likely future of their use in the clinical setting. Improved understanding of DPSCs, SCAP, PDLSCs and GMSCs has been achieved from intense interest and vigorous research over the past decade. Continuous research is needed, especially the testing of clinical protocols in large animal models, and the establishment of a stem cell banking system. Large-scale clinical trials should also be prepared and initiated in order to pave the way for future cell- or regenerative-based management for dental traumatic injuries.

## Essentials

- **Stem cell properties.** A stem cell is capable of both self-renewal and giving rise to one or multiple lineages of differentiated and specialized cell of certain functional phenotypes thereby generating complex tissues and organs.
- **Stem cell differentiation potencies.** Depending on the potency of stem cells to differentiate into different cell linages thus forming various tissues, distinctive categories of stem cells are totipotent, pluripotent, multipotent and unipotent.
- **Main types of stem cells by developmental stage.** Depending on the stage during development that stem cells exist, there are embryonic stem cells, fetal stem cells and postnatal (adult) stem cells.
- **Mesenchymal stem cells (MSCs).** Stem cells in the mesenchyme that are able to differentiate into specialized cell types to form and repair the mesenchymal tissues.
- **Stem cell niche.** The stem cell niche has the following characteristics: (1) it is an anatomic space where stem cells are regulated to provide tissue homeostasis; (2) it is the place where stem cells are instructed to control maintenance, quiescence, self-renewal and recruitment in relation to differentiation, fate determination and long-term regenerative capacity; and (3) the niche will influence the cell motility.
- **Role of DPSCs in reparative dentin formation.** DPSCs exist in the pulp of permanent teeth. Isolated DPSCs have their capability to form dentin/pulp-like complex upon *in vivo* transplantation in conjunction with hydroxyapatite/tricalcium phosphate as a carrier vehicle. DPSCs in the pulp are considered to be responsible for giving rise to the replacement odontoblasts that form reparative dentin.
- **Apical papilla.** The apical papilla is a connective tissue that is loosely attached to the developing root and contains less blood vessels and cellular components than the pulp tissue. Apical papilla is separated from pulp tissue by a cell-rich zone.
- **Implication of SCAP for regenerative endodontics.** It is being investigated whether, if the apical papilla is still viable at the time of treatment, SCAP may be attracted into the canal and regenerate pulp and form new dentin using molecular approaches. One idea was tested using the stem cell attractant SDF-1α to chemoattract SCAP expressing the SDF-1α receptor CXCR4.
- **PDLSCs in periodontal regeneration for avulsed teeth.** There is a potential use of PDLSCs in helping regenerate the PDL in cases of avulsed teeth. Cell-based therapy

could theoretically prevent or lower the incidence of resorption following tooth replantation, especially when extended extraoral time has occurred. In a dog model, using PDLSC cell sheet wrapped around the root before replantation may enhance the healing area and reduce ankylosis/resorption.

- **GMSCs *in vivo* regenerative potential.** As a cell source easy to access, GMSCs can be used to enhance bone regeneration in the context of traumatic injury treatment. *In vivo* orthotopic bone tissue regeneration using GMSCs has been demonstrated by repairing mandibular and critical-sized calvarial defects in Sprague Dawley rats.

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# 6

# Development of a Bio-Root

S. Wang, F. Wei, Z. Gao & L. Hu

## Introduction

Tooth loss is caused by caries, dental trauma and periodontal disease, and has a serious impact on the quality of the patient's life. The restoration methods of tooth loss, such as a removable partial denture, fixed bridge, autotransplantation, allotransplantation and dental implants, have been employed for many years, but have never been totally satisfactory. With the great achievement of stem cell-based tissue engineering, it is possible that dental, non-dental, embryonic and adult stem cells could contribute to teeth formation in appropriate setting (1). With the increasing knowledge of stem cell biology, regenerative medicine and tissue engineering technology, tooth regeneration for the restoration of tooth loss is becoming a realistic possibility. Therefore, researchers are trying to explore the potential for regenerating living functional teeth. Stem cell-mediated root regeneration uses the regeneration of a bio-root and its associated periodontal tissues to restore tooth loss, thus offering an alternative, promising therapy for the restoration of tooth loss. Recent advances have shown the successful regeneration of a dental stem cell-based functional bio-root and its associated periodontal tissue in miniature pig models (2–4). This chapter introduces the exploration, current status and prospects of bio-root regeneration.

## Definition

The concept of a 'bio-root', which is generated through stem cell-based tissue engineering, was proposed in 2006 (2). The strategy is to implant preshaped root-like scaffolds combined with stem cells into the alveolar bone to form a functional bio-root, which is capable of supporting post crown prostheses. The criteria for judging success are: (1) to have a root-like structure with a computed tomography (CT) examination showing a complete root-shaped high density image; (2) to have similar biomechanical properties and elements to natural teeth; (3) to have a histology showing periodontal ligament (PDL) like tissue and a dentin-like matrix structure (3, 4) (Fig. 6.1).

## Development history

More than two decades ago, tissue engineering, based on a combination of cells, scaffolds and biofactors, was conceptualized as a means to functionally restore and regenerate diseased or damaged tissues and organs, such as the musculoskeletal system. More recently, a number of new concepts have been developed that promise to enhance the success of cell-based and biomaterial-based tissue engineering approaches (5). As regenerative medicine and tissue engineering technology develops, researchers are exploring the potential for regenerating living functional teeth. Evidence that stem cell populations are present in human teeth provides the opportunity to consider 'new for old' biological tooth replacement (6).

Recent bioengineering of complex tooth structures from tooth tissues of rats and humans suggests the potential for the regeneration of mammalian dental tissues. Based on a number of scientific studies, various precursors of specialized tooth-associated cell types have been identified, including their characteristics and properties in the postnatal organism (7, 8). It is known that in certain conditions, cultures of dental pulp cells derived from an early stage of developing dental root and pulp tissue can differentiate into odontoblast-like cells with the capacity to form mineralized nodules *in vitro* (7). Researchers have found that tooth tissue

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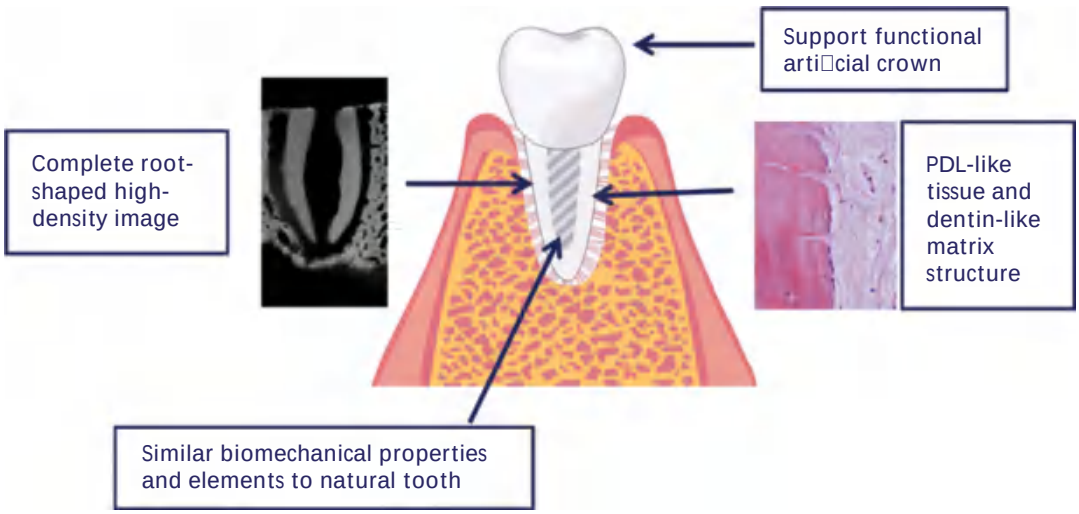



**Fig. 6.1** The concept and criteria of functional bio-root regeneration and restoration of tooth loss.

engineering methods can be used to generate both pig and rat tooth tissues by using cultured rat tooth bud cells (6). Recently, stem cell-mediated bio-root has been regenerated in miniature pig models, similar to that of human teeth in anatomy, development, physiology, pathophysiology and disease occurrence (2–4). Recent advances in bio-root regeneration are reviewed here.

## Components of stem cell-mediated bio-root regeneration

### Stem cells

#### *Dental stem cells*

Mesenchymal stem cells (MSCs) are multipotent cells that can differentiate into different types of cells such as osteoblasts, chondrocytes, myocytes and adipocytes. Since they were first isolated from bone marrow, it was thought that MSCs were located in almost all tissues, perhaps even in neural tissues. Thus, it was speculated that MSCs may be found in adult dental tissues, which also contain a population of multipotential stem cells (7, 9–12). Dental pulp stem cells (DPSCs)/stem cells from apical papilla (SCAP) and periodontal ligament stem cells (PDLSCs) are primarily used to generate dentin-like structures and cementum/PDL-like structures, respectively. Evidence shows that periapical follicle stem cells (PAFSCs) also have the potential to mediate bio-root regeneration (2, 7, 13, 14).

#### *Dental pulp stem cells*

Dental pulp stem cells can generate dentin/pulp-like structures when mixed with hydroxyapatite/tricalcium phosphate (HA/TCP) ceramic powder and transplanted into the dorsal surface of immunocompromised mice. The DPSC transplants are characterized by a well-defined layer of aligned odontoblast-like cells expressing the dentin-specific protein DSPP, with their processes oriented in the same direction and extending into tubular structures within newly generated dentin. The collagen matrix mimics the structure of primary dentin with ordered fibers perpendicular to the odontoblast layer (7).

#### *Stem cells from apical papilla*

Human apical papilla stem cells have been demonstrated to positively stain for mesenchymal stem cell surface molecule STRO-1. SCAP, isolated from the exterior of the root foramen area of extracted third molars, were capable of differentiating into odontoblasts/osteoblasts and other cell types such as adipocytes, which were analogous to DPSCs and bone marrow mesenchymal stem cells (BMMSCs). When transplanted into immunocompromised mice with HA/TCP, they would regenerate a typical dentin structure (2).

#### *Periodontal ligament stem cells*

Similar to BMMSCs and DPSCs, PDLSCs obtained from extracted human third molars have been isolated to adherent clonogenic cell clusters of fibroblast-like cells and have been identified as an outcome of stem cells and later differentiation. When transplanted into immunocompromised rodents, PDLSCs showed the capacity to generate a cementum/PDL-like structure, in which a thin layer of cementum-like tissue formed on the surface of the carrier, along with condensed collagen fibers with sparse cells that mimicked the physiologic attachment of Sharpey's fiber, which is needed to form a functional attachment of cementum/PDL structures. When transplanted into surgically created defects at the periodontal area of mandibular molars in immunocompromised rats, collagen fiber formation could be found within the transplants, which inferred that PDLSCs might have a potential functional role in periodontal tissue regeneration (8).

#### *Periapical follicle stem cells*

Periapical follicle stem cells (PAFSCs), isolated from the apical end of developing roots of human third molars at the

root-developing stage, showed crucial stem cell properties and a higher proliferation rate than PDLSCs *in vitro*. Meanwhile, PAFSCs also showed the tissue-regenerative capacity needed to produce a typical cementum/PDL-like complex *in vivo*. Developing root-derived PAFSCs are a distinctive cell population and might be a promising candidate for bio-root engineering (14).

### *Non-dental stem cells*

Non-dental stem cells also have potential odontogenic properties. Embryonic stem cells, neural stem cells and adult bone marrow-derived cells all have the potential of expressing odontogenic genes. Recombination between non-dental stem cell aggregations and embryonic oral epithelium transplanted into adult mice renal capsules, resulted in the development of tooth structures and associated bone. Moreover, transplantation of embryonic tooth primordia into the adult mouse jaw resulted in the development of tooth structures, showing that an embryonic primordium can develop continuously in an adult environment. These results thus provide significant evidence that artificial embryonic tooth primordia created from cultured cells can be used to replace missing teeth following transplantation into the adult jaw (15).

## Scaffolds

The scaffold is also an essential element in tissue engineering. Studies confirm the feasibility of using tissue engineering technology for successful tooth root regeneration. It is still unclear exactly what shape and size a bioengineered tooth root should be to produce the best retention and function. This is important since bioengineered organ/tissue shapes and sizes are routinely controlled by the scaffold fabrication. Therefore, exploring and optimizing the design of scaffolds has great significance on further research and clinical utilization of biological tooth root (bio-root) regeneration.

### *Spherical polyglycolide/poly-L-lactide (PGA-PLLA) scaffolds*

PGA-PLLA scaffolds are frequently used in tissue engineering and cell transplantation due to their fantastic biocompatibility and cell-retaining behavior. Briefly, PGA-PLLA scaffolds are fabricated through bonding PGA felt and PLLA in chloroform in a spherical polyvinylsiloxane negative mold (16).

### *Hydroxyapatite/tricalcium phosphate (HA/TCP) scaffolds*

Researchers have proved that human bones are generated after xenogeneic transplantation of bone marrow stromal cells with HA/TCP as a carrier vehicle. Consistent bone formation by human MSCs was achieved within HA/TCP ceramics in the form of blocks, powder and HA/TCP powder type I bovine fibrillar collagen strips; and bone was maintained for at least 19 weeks (17).

### *Treated dentin matrix (TDM) scaffold*

In order to validate the physiologic function of an optimal scaffold shape *in vivo*, researchers have seeded dental stem cells onto a TDM scaffold, which was transplanted into the alveolar bone of swine and later-installed porcelain crown. Tooth roots have not only been successfully regenerated histologically, but have also performed masticatory functions and remained stable for 3 months after crown restoration. A TDM scaffold 9.4 mm in length and 4.9/3.4 mm in upper/bottom diameter is a suitable biological scaffold for tooth root regeneration. These results also provide a recommendable design protocol for fabricating other scaffolds in tooth root reconstruction (18).

## Biofactors and cell sheets

In the past, tissue fabrication techniques in regenerative medicine have largely relied on scaffold-based approaches (19). Numerous challenges existed in fabricating functional tissue engineered organs. With the preservation of cellular junctions, endogenous extracellular matrix (ECM) and mimicking cellular microenvironments in terms of various mechanical, chemical and biologic properties, continuous cell sheets may be beneficial for cell transplantation. Cell sheet engineering has been developed as an alternative approach to tissue engineering in periodontal tissues (20). At present, several improvements have been made to harvest the living cell sheet more easily.

In a recent study, dexamethasone and ascorbic acid phosphate (vitamin C, Vc) were used to create cell sheets to enhance bone formation. When treated with 20 and 50 mg/mL Vc for 10–13 days in culture, human PDLSCs easily became confluent and wrapped at the dish edge; the entire cell sheets were detached smoothly using a crooked syringe needle. Vc-induced human PDLSC sheets preserved intercellular junctions with more extracellular matrix by activating telomerase and enhanced tissue regeneration in nude mice and miniature swine (21) (Fig. 6.2). Furthermore, it was found that prostaglandin E2 plays a crucial role in allogeneic PDLSC-mediated immunomodulation and periodontal tissue regeneration *in vitro* and *in vivo* (20).

# Stem cell-mediated root regeneration in miniature pig models

Compared with small animal models such as rodents, large animal models are superior in many aspects for the study of human diseases and preclinical therapies. Since the development of the Minnesota miniature pig in 1949 at the Hormel Institute (USA), miniature pigs have been used as a large animal model in medical studies for scientific, economic and ethical reasons (21).

The oral maxillofacial region of miniature pigs is similar to that of humans in anatomy, development, physiology, pathophysiology and disease occurrence. The miniature pig

A B

**Fig. 6.2** The whole view and structure of a PDLSC sheet. A. Whole view photograph of the harvested PDLSC sheet. B. Histologic staining of the harvested sheets with hematoxylin and eosin (H&E). Scale bar: 200 μm.

has both deciduous and permanent dentitions, which is important for investigating tooth development and regeneration. The initiation of tooth development and eruption in the miniature pig is quite similar to that in humans. Several stem cells, using single-colony selection from adult miniature pigs *in vitro*, have been cultured, including dental pulp stem cells, periodontal stem cells and bone marrow mesenchymal stem cells. These stem cells share characteristics with similar human cells (22, 23).

## Functional tooth regeneration

A long-term follow-up study of bio-roots based on functional tooth regeneration using autogenic and allogenic dental stem cells is now being performed in miniature pigs.

### *SCAP/PDLSC-mediated root/periodontal structure as an artificial crown support for the restoration of tooth function in swine*

#### Isolation and culture of SCAP

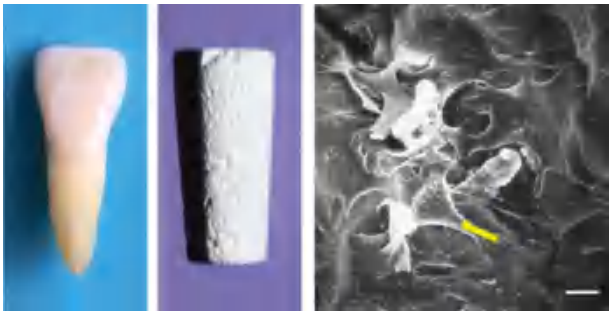
The root apical papilla contains SCAP that appear to have a greater capacity for dentin regeneration than DPSCs. Although matured dental pulp-derived DPSCs have dentin/pulp regenerative capacity, developing tissue-derived SCAP shows a superior tissue regeneration potential than that of DPSCs. Therefore, single colonies of swine SCAP are isolated and cultured to generate dentin.

#### Root-shaped HA/TCP carrier loaded with SCAP

To generate a root-shaped structure, HA/TCP scaffolds were selected as a carrier vehicle. Swine SCAP were added to a root-shaped HA/TCP block that contained an inner post channel space to allow the subsequent installation of a porcelain crown (Fig. 6.3).

#### Gelfoam scaffolds containing PDLSCs

Single colonies of swine PDLSCs were isolated and cultured to generate PDL. Gelfoam scaffolds containing PDLSCs were used to cover the HA/TCP carrier loaded with SCAP.



A B C

**Fig. 6.3** Procedure using a root-shaped scaffold and cell seeding. A. The extracted incisor. B. The root-shaped scaffold fabricated with HA/TCP. C. Scanning electron microscopy image of SCAP seeded on the HA/TCP scaffold; the yellow arrow indicates the SCAP. Scale bar: 20 μm.

#### Preparation of the lower incisor socket and implantation of a HA/SCAP-gelfoam/PDLSC structure

A lower incisor was extracted and the extraction socket was further cleaned with a surgical bur to remove remaining periodontal tissues. The HA/SCAP-gelfoam/PDLSC structure was inserted into the socket and sutured for 3 months. CT examination revealed an HA/SCAP-gelfoam/PDLSC structure growing inside the socket with mineralized root-like tissue formation and periodontal ligament space.

#### Insertion and cementation of a pre-made porcelain crown and functional evaluation

The surface of the implanted HA/SCAP-gelfoam/PDLSC structure was surgically reopened after 3 months post-implantation, and a prefabricated porcelain crown resembling a miniature pig incisor was inserted and cemented into the preformed post channel inside the HA/TCP block. After suture of the surgical opening, the porcelain crown was retained *in situ* and subjected to the process of tooth function for 4 weeks. CT and histologic analysis confirmed that the root/periodontal structures were regenerated. Moreover, newly formed bio-roots demonstrated a significantly

improved compressive strength compared to that of the original HA/TCP carriers after a 6-month implantation. Although newly formed bio-roots show a lower compressive strength than that of natural swine root dentin, they seemed capable of supporting porcelain crowns and resulted in normal functions. It might be possible to improve the compressive strength and hardness of the bio-roots by selecting the best bioengineered materials and optimizing the implanted stem cell numbers and quality.

### *Allogeneic DPSC/PDLSC-mediated root/periodontal structure as an artificial crown support for the restoration of tooth function in swine*

Previously, it was showed that autologous dental stem cells have the potential to form a bioengineered tooth root (bio-root) for temporally supporting artificial crowns in miniature pigs. However, most patients are aged, and sources of autologous dental stem cells are limited. Moreover, there are other barriers, including insufficient cell migration and retention into the scaffold, a mismatch between scaffold degradation and the cell proliferation rate, and the difficulty in generating functional tissues with complex architectural structure through scaffold-based tissue engineering methods.

Due to their low immunogenicity, allogeneic mesenchyme stem cells and cell sheet engineering have been developed as an alternative approach to tissue engineering. The research group regenerated a functional bio-root using allogeneic DPSCs and Vc-induced PDLSC cell sheets (3). A root-shaped HA/TCP scaffold containing DPSCs was covered by a Vc-induced PDLSC cell sheet and implanted into a newly generated jaw bone implant socket. After 6 months of implantation, a prefabricated porcelain crown was cemented to the implant and subjected to tooth function testing. Procedures in this study are similar to those in the above research (3). The regenerated bio-root exhibited characteristics of a normal tooth after 6 months of use, including dentin tubule-like and functional PDL-like structures. The regenerated bio-roots also demonstrated significantly improved compressive strength, with the direction of PDL-like tissue fibers moving from parallel to oblique and inserting into bone and regular dentin-like structures. The clinical features of the bio-root, including probing depth, gingival recession and attachment loss, were comparable with those of the natural teeth of miniature pigs. No immunologic response to the bio-roots was observed (3).

### *Comparison of allogeneic DPSC/PDLSC-mediated bio-root and implant-based restoration for the restoration of tooth function in swine*

Dental implants, which are also a substitute for the tooth root, are currently the most popular method in clinical use for restoration, with a relatively satisfactory level of tooth restoration. However, such implants require very particular therapeutic indications, such as sufficient alveolar bone (24). In addition, implant fractures caused by concentrated stress and peri-implantitis often lead to implant failure. Stem cell-mediated root regeneration offers the opportunity to regenerate a bio-root and its associated periodontal tissue, which is necessary for maintaining the physiologic function of the tooth. Thus, it is necessary to evaluate which is the best method for tooth restoration.

A recent study compared and evaluated both allogeneic DPSC/PDLSC-mediated engineered bio-roots and commercial dental implants in restoring tooth loss in a miniature pig model. This study showed that both regenerated bio-roots and dental implants could mediate tooth function (4). The biomechanical properties of the bio-root, such as compressive strength, elasticity modulus and torsional force, were very similar to those of the natural tooth root; however, significantly higher biomechanical properties were found for the dental implant.

Taken together, these results suggest that the bio-root is a promising alternative for the dental implants used clinically today, and that it is feasible to bank dental stem cells for use in dental clinical practices for future autologous and allogeneic use in tooth regeneration.

## Regeneration procedure of stem cell-mediated bio-roots

There are a number of steps in this procedure (Fig. 6.4):

- Isolation and identification of properties of stem cells
- Scaffold fabrication
- Stem cells seeded on the scaffold and harvested stem cell sheet
- Preparation of bio-root complex before implantation
- Preparation of implantation sockets
- Implantation of bio-root complex
- Regeneration of bio-root.

## Evaluation

### Stem cell-based tissue engineering and functional bio-root regeneration

A gross view and micro CT scan demonstrated that both high density tissues and implants stayed in the jaw bone for 3 months after transplantation. However, the HA/TCP/DPSC/PDLSC sheet implant formed a hard root structure, while pure osseointegration was only observed in the dental implant sample (25) (Fig. 6.5).

### Clinical assessment and evaluation of bio-roots and dental implants

Clinical features such as probing depth, gingival recession and peri-implantitis were compared between bio-roots and dental implants. No significant differences were found and

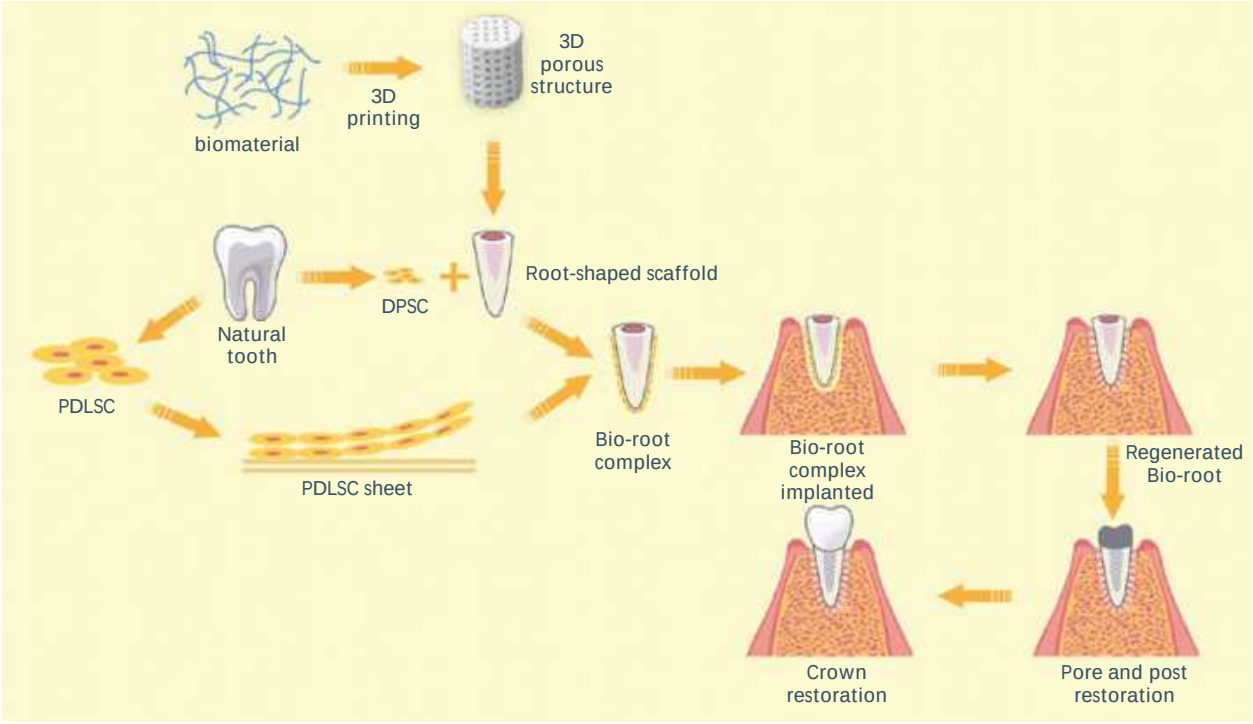
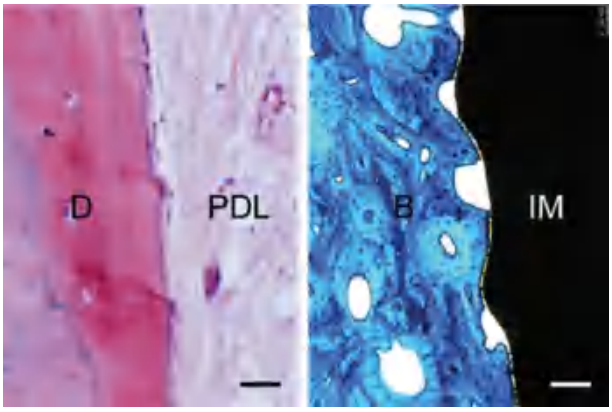



**Fig. 6.4** Schematic diagram for the use of bio-roots in the treatment of tooth loss. DPSC, dental pulp stem cells; PDLSC, periodontal ligament stem cells.




A B

**Fig. 6.5** Functional structure of a bio-root compared to a dental implant. A. H&E staining of the regenerated bio-root, showing PDL-like tissue and dentin-like tissue formation. B. Toluidine blue staining showed pure osseointegration in the dental implant. B, bone; D, dentin; IM, implant; PDL, periodontal ligament. Scale bars: 50 μm.

no gingivitis or peri-implantitis was found in either the bio-roots or the dental implants.

## Biomechanical properties and element analysis

In addition to the similar histologic characteristics described above, the regenerated bio-root displayed similar biomechanical properties to those of the natural root, with a compressive strength that was significantly lower than the implant and higher than the HA/TCP scaffolds. The compressive strength in the dental implants was more than 20-fold higher than that of normal teeth. The elastic modulus index in the bio-root was similar to that in normal teeth, while it was more than five-fold higher than normal teeth in the dental implants. The torsional force of the regenerated bio-roots was close to that of normal tooth roots, and was significantly lower than that of dental implants. In addition, element analysis showed that the bio-root had similar element content (mainly calcium, phosphorus and magnesium) to that of natural teeth, indicating that biologic changes had occurred during the bio-root regeneration.

## Bioengineered dentin-induced PDL/cementum-like tissue formation

Bioengineered dentin is able to induce cementogenesis and PDL formation, and condense PDL arranged perpendicularly on the dentin surface via a layer of cementum-like tissue. The results indicated that tissue engineered dentin could be induced using an inductive substrate and could be used as a further substrate for cementum and PDL tissue engineering (26).

# Challenges and prospects

It is clear that a tremendous amount of work is still needed to identify and maintain multipotential MSCs *in vitro*, and there are still some important hurdles that need to be overcome:

the identification of multipotential cells following *ex vivo* expansion; the establishment of growth and differentiation conditions that induce lineage-specific commitment; and the development of suitable carriers and inductive factors able to help implants integrate into the surrounding environment for the reconstruction of functional complex organ systems (27).

Since sources of autologous stem cells are limited, particularly for aged patients, the clinical application of this approach is, at the moment, restricted. Thus, it is critical to bank high quality dental stem cells for future autologous use. Although survival in freeze–thaw cycles and the immunogenicity of these cells require further exploration, allogeneic cells may be another source of stem cells for those older individuals who have already had their wisdom teeth extracted.

Bio-roots show a lower compressive strength than that of natural root dentin although they seem capable of supporting porcelain crowns, resulting in normal functions (2). However, it may be possible to improve the compressive strength and hardness of bio-roots by selecting better bio-engineered materials and optimizing implanted stem cell numbers and quality.

Stem cell-mediated root regeneration combined with clinical crown technology may be a promising approach to functional tooth restoration owing to the availability of high quality dental stem cells for allogenous and autologous transplantation as well as long-term experience with clinical dental implant procedures.

Stem cell-mediated root regeneration offers opportunities to regenerate a bio-root and its associated periodontal tissues, which are necessary for maintaining the physiologic function of teeth. All recent studies indicate that a regenerative approach is feasible using allogeneic MSCs to regenerate bio-roots for tooth loss in large animals (e.g. the miniature pig). This is the first step toward prospective preclinical trials. Results of ongoing preclinical and clinical trials will be needed to properly assess the therapeutic potential in clinical applications.

## Essentials

Stem cell-mediated root regeneration combined with clinical crown technology provides a promising approach to functional tooth restoration owing to the availability of high quality dental stem cells for allogenous and autologous transplantation as well as long-term experience with clinical dental implant procedures.

(1) Stem cell-mediated bio-root regeneration has been performed in miniature pigs based on a combination of stem cells (dental and non-dental stem cells), scaffolds (PGA-PLLA, HA/TCP, TDM scaffolds, etc.), biofactors (endogenous ECM, vitamin C, prostaglandin E2, etc.) and cell sheets.

(2) The steps in the production of stem cell-mediated bio-roots are: (i) isolation and identification properties of stem cells; (ii) scaffold fabrication; (iii) stem cells seeded on the scaffold and harvested stem cell sheet; (iv) preparation of the bio-root complex before implantation; (v) preparation of implantation sockets; (vi) implantation of the bio-root complex; and (vii) regeneration of the bio-root.

(3) The criteria for judging the success of a 'bio-root' are: (i) having a root-like structure and a CT examination showing a complete root-shaped high density image; (ii) similar biomechanical properties and elements to natural teeth; and (iii) histology showing PDL-like tissue and dentin-like matrix structure.

(4) The biomechanical properties of the bio-root such as compressive strength, elasticity modulus and torsional force are very similar to those of the natural tooth root; however, significantly higher biomechanical properties have been found for dental implants.

(5) There are still challenges for maintaining multipotential stem cells, banking high quality stem cells, improving the compressive strength and hardness of bio-roots, etc.

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# 7

# Bone-Resorbing Osteoclasts

U. H. Lerner

## Introduction

Osteoclasts are startlingly large cells, having a diameter of often more than 100 µm, which is considerably larger than most other cells with a diameter of 10–20 µm. The osteoclasts contain several, sometimes many, nuclei, and due to their size and multinuclearity osteoclasts are often easy to recognize in histologic sections. In certain pathologic conditions such as Paget´s disease, the osteoclasts can be huge, containing several hundreds of nuclei. Osteoclasts have similarities with multinuclear foreign body giant cells, but can be distinguished from these cells by their localization exclusively along bone surfaces and by using enzyme histochemical staining for the enzyme tartrate-resistant acid phosphatase (TRAP), or by immune histochemical staining for the cysteine proteinase cathepsin K (Fig. 7.1).

Osteoclasts (from the Greek words *osteon* (bone) and *klastos* (broken)) were first described by the Swiss physician Albert von Kölliker (1817–1905). He published in 1873, in Leipzig, the book *Die Normale Resorption des Knochengewebes* in which he beautifully describes osteoclasts and for the first time distinguishes these cells both morphologically and functionally from the multinuclear megakaryocytes also found in bone marrow (1). The drawings in the book clearly depict several morphologic characteristics such as large size, multinuclearity and even the ruffled border.The British surgeon and anatomist John Howship (1781–1841) had described grooves, nowadays called Howship's lacuna, in the skeleton in a treatise on the natural and pathologic anatomy of bone in 1820, but it was von Kölliker who 53 years later described that these grooves were created by bone-resorbing osteoclasts.

It was thought until the mid-1970s that osteoclasts originated from the fusion of several osteoblasts, which instead of making new bone as mononuclear cells resorbed bone as multinuclear osteoclasts. The idea was that this shift in functionality was the mechanism by which bone was renewed. Pioneering work by the Swedish orthopedic surgeon Gustav Göthlin and the pathologist Jan Ericsson at the Karolinska Institute showed that circulating cells from the bone marrow gave rise to osteoclasts (2). They used parabiosis experiments, in which bone marrow in one rat was destroyed by irradiation, and blood vessels from this rat were then joined to vessels from a normal rat. By labeling the cells in the bone marrow of the intact rat, it was found that osteoclasts that formed during fractures in the rat without bone marrow originated from the bone marrow of the other rat. Later, it was also demonstrated using chick and quail chimeras that osteoclasts were derived from circulating cells (3). We now know that osteoclasts are derived from myeloid stem cells in hematopoetic tissues and that they are very closely related to macrophages and dendritic cells in the immune system (4). The knowledge about how the stem cells are differentiated along the osteoclastic lineage has increased enormously during the last 15 years and has led to the development of new antiresorptive treatments (5).

## Osteoclastogenesis

When it was discovered during the late 1970s that osteoclasts are derived from hematopoetic cells in bone marrow, spleen or embryonic liver, it was found that receptors for hormones known to increase osteoclast formation, such as parathyroid hormone (PTH) and 1,25$(OH)_2$-vitamin D3, were not expressed by osteoclasts and that these hormones could not affect osteoclasts directly. Surprisingly, however, receptors for these hormones were found to be expressed by osteoblasts. It was then hypothesized by Rodan and Martin that osteoclast formation is indirectly regulated by some

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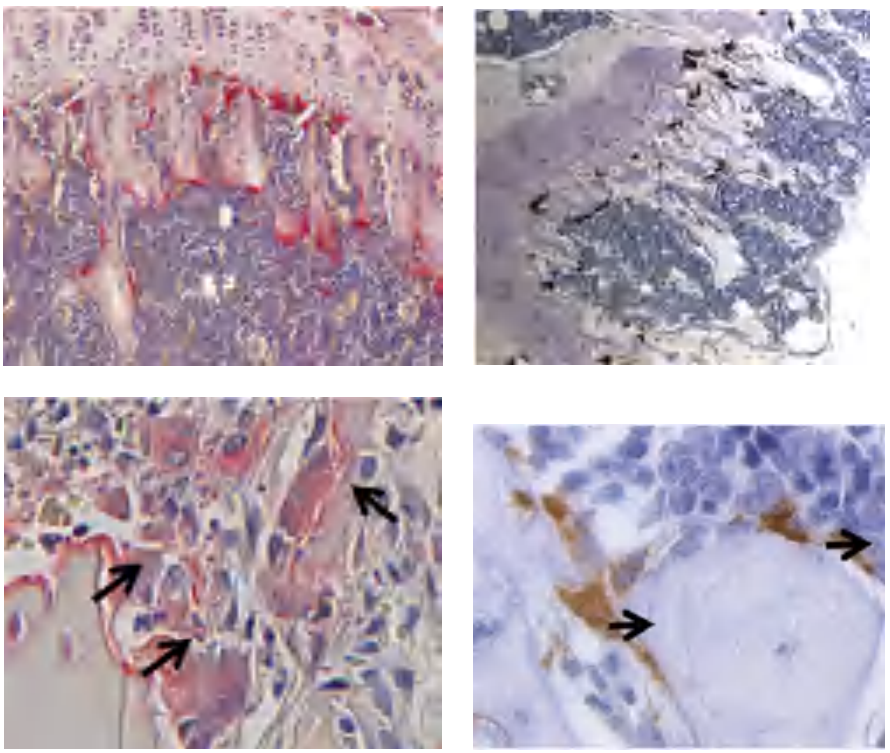



**Fig. 7.1** Numerous osteoclasts are present in metaphyseal bone. These cells can be visualized either by enzyme histochemical staining of tartrate-resistant acid phosphatase (A) or by immunohistochemical staining of cathepsin K (B). Dark arrows in the lower panels depict mature, multinucleated osteoclasts. The sections are from mouse tibiae.

paracrine signaling from osteoblasts (6). During the late 1980s, experimental data supported this view when it was found that activation of receptors recognizing PTH on osteoblasts was crucial for osteoclast activity (7). During the late 1990s it was eventually found that the osteoblast-derived factor stimulating osteoclast differentiation was a cytokine given the name receptor activator of nuclear factor κB ligand (RANKL) (8–10).

## M-CSF and RANKL

For osteoclastogenesis to occur, the receptor c-Fms (also called colony-stimulating factor 1 receptor, CSF1R) on mononuclear progenitor cells needs to be activated by macrophage colony-stimulating factor (M-CSF; also called colony-stimulating factor 1 (CSF1)) (Fig. 7.2A). The importance of M-CSF for osteoclast formation was initially shown by the demonstration that high bone mass in osteopetrotic (*op/op*) mice is due to a loss-of-function mutation in the gene encoding M-CSF, leading to decreased formation of osteoclasts (11, 12), and that the osteopetrotic phenotype of *op/op* mice can be cured by administration of M-CSF (13). Further demonstration of the importance of M-CSF is the observation that deletion of the *c-Fms*/*Csf1r* gene also results in osteopetrosis and decreased osteoclast formation (14). Recently, it has been found that interleukin-34 (IL-34), expressed by several cell types, including human gingival fibroblasts (15), can serve as a second ligand for c-Fms/CSF1R (16). Although IL-34 can recapitulate many functions of M-CSF, including during osteoclast formation *in vitro* (17), it also has effects different to those of M-CSF and might bind to distinct regions of c-Fms/CSF1R. The role of IL-34 and its relation to the role of M-CSF in bone physiology and pathology remains to be shown.

The receptor for M-CSF belongs to the receptor tyrosine kinase superfamily. Subsequent to autophosphorylation of the receptor at tyrosine residues, downstream intracellular signaling in osteoclast progenitor cells results in increased proliferation and enhanced survival due to induction of anti-apoptotic genes, but also in the induction of genes involved in macrophage differentiation (18).

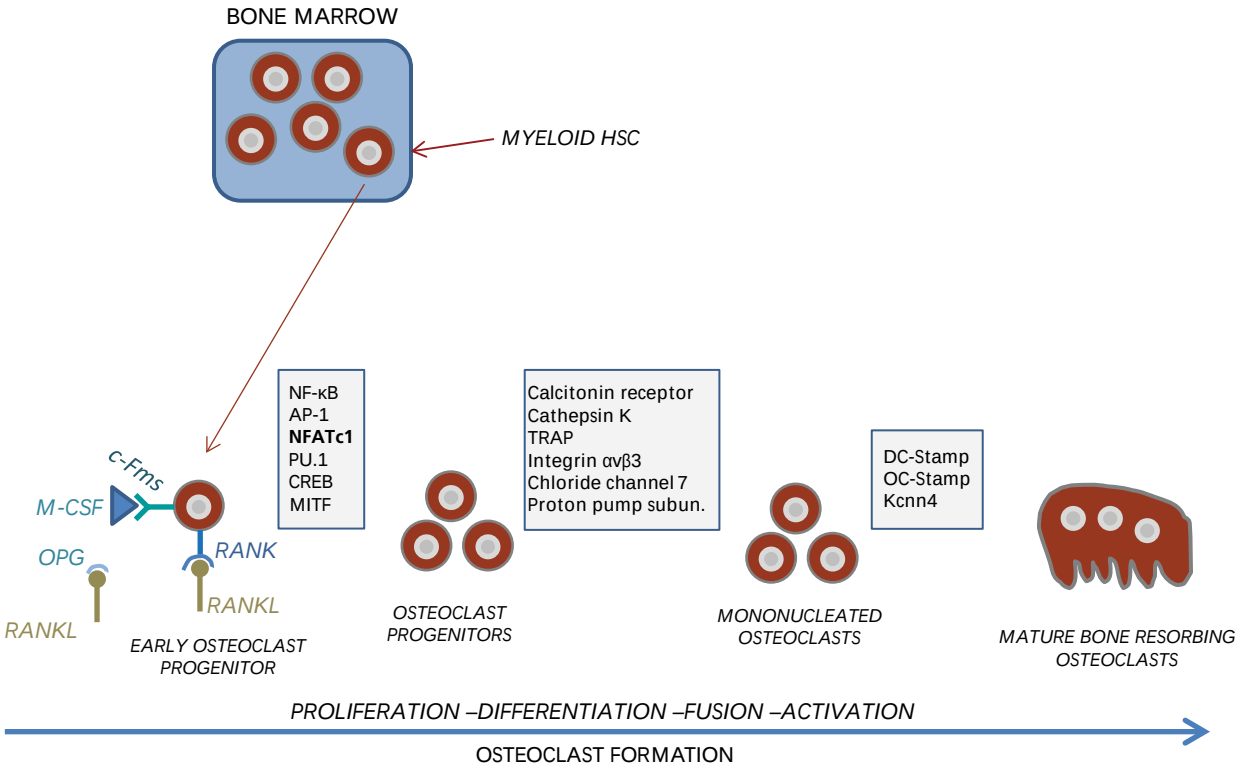
Differentiation along the osteoclastic lineage, rather than along the macrophage or dendritic cell lineages, is critically dependent on activation of the cell surface receptor RANK, expressed by the osteoclast progenitors, by its cognate ligand RANKL, expressed by osteoblasts (Fig. 7.2A). It was observed during the late 1980s, using co-cultures of osteoblasts and osteoclast progenitors from mice with deletion of either the receptor for PTH or that for $1,25(OH)_2$-vitamin D3 (the hormonal metabolite of vitamin D3), that the presence of PTH or vitamin D3 receptors on osteoclasts did not result in osteoclastogenesis. However, expression and activation of their receptors on osteoblasts resulted in osteoclast formation (19, 20). In these experiments, it was also observed that osteoclastogenesis only took place when osteoclast progenitors were in contact with islands of osteoblasts, indicating that osteoclastogenesis is dependent on cell to cell contact interactions between osteoblasts and osteoclast progenitors. Ten years later it was found that the osteoblast-derived factor is RANKL (21), which activates the receptor RANK on osteoclast progenitors (22), and that this interaction could be inhibited by the decoy receptor osteoprotegerin (OPG) (23). Stimulation of osteoblasts with PTH or $1,25(OH)_2$-vitamin D3 increased expression of RANKL and decreased that of

OPG. When the genes for either RANKL or RANK were deleted (21, 24), or the gene for OPG was overexpressed (23), no osteoclasts were formed and the skeleton was filled up with an excess of bone, leading to osteopetrosis. These observations, together with the finding that decreased expression of OPG causes early-onset osteoporosis (25), strongly suggested that RANKL-RANK signaling should be a potential target for drug development. This insight has led to the development of humanized antibodies neutralizing RANKL (denosumab) used for inhibition of excess osteoclast formation in osteoporosis and skeletal metastasis of malignant tumors (5).

RANK was initially cloned in dendritic cells by immunologists (26), who also found that RANKL (named TRANCE, or TNF-related activation-induced cytokine) was expressed by T cells and found to be important in adaptive immunity (27). Later, RANKL/TRANCE was shown to be involved in osteoclast formation in arthritis (28). Thus, RANKL-RANK signaling is not unique to bone, but is also involved in signaling between dendritic cells and lymphocytes. In line with these observations, mice with deletion of the genes for RANKL or RANK not only exhibited an osteopetrotic phenotype but also abnormal development of thymus and lymphoid tissues, which may be of concern in treatment with denosumab.

The importance of RANKL-RANK signaling in humans is demonstrated not only by the response to denosumab, but also by reports showing that loss-of-function mutations in the genes for RANKL and RANK are associated with high bone mass (29, 30), and that gain-of-function mutations have been linked to low bone mass (31, 32).

RANKL belongs to the tumor necrosis factor (TNF) superfamily, and RANK to the TNF receptor (TNFR) superfamily. Although signaling through RANK and TNFR shares many pathways, TNF cannot substitute for RANKL *in vivo*. The best evidence for this is that no osteoclasts are formed in




A

**Fig. 7.2** Formation of bone-resorbing osteoclasts involves proliferation, differentiation and fusion of mononuclear osteoclast progenitor cells to multinucleated, mature osteoclasts and activation of these cells to bone-resorbing osteoclasts. A. Differentiation of early osteoclast progenitor cells along the osteoclastic lineage requires stimulation of the receptor c-Fms by its ligand macrophage colony-stimulating factor (M-CSF) to increase proliferation and survival of the progenitor cells and of the receptor RANK by its ligand receptor activator of NF-κB ligand (RANKL) to induce expression of genes required for osteoclast differentiation. The amount of RANKL available for activation of RANK is balanced by the decoy receptor osteoprotegerin (OPG) which can bind to RANKL and inhibit the interaction with RANK. An initial down-stream signaling event includes activation of transcription factors such as NF-κB, AP-1, NFATc1, PU.1, CREB and MITF, which when translocated to the nucleus regulate a variety of genes necessary for osteoclast activity (calcitonin receptor, cathepsin K, TRAP, integrin αvβ3, chloride channel, protein pump subunits) and for fusion of mononuclear progenitors to mature osteoclasts (DC-Stamp, OC-Stamp, Kcnn4). HSC, hematopoietic stem cells. B. For osteoclastogenesis to occur, activation (by unknown ligands) of immunoglobulin-like receptors such as OSCAR and TREM-2 is also necessary. These receptors dimerize with ITAM-containing intracellular signaling co-receptors FcRγ and DAP12, respectively. The most important transcription factor in osteoclast differentiation is NFATc1, which is induced by the transcription factors NF-κB and AP-1. NFATc1 rapidly becomes phosphorylated and, thereby, inactivated. Signaling through FcRγ and DAP12 leads to increased levels of calcium in the cytoplasm and activation of the enzyme calcineurin. This enzyme dephosphorylates NFATc1, which then becomes activated, and once the amount of this transcription factor starts to increase it will induce its own expression by an autoamplification process. NFATc1, together with other transcription factors such as AP-1, MITF, CREB and PU.1, will turn on a variety of genes necessary for osteoclast differentiation, fusion and function.

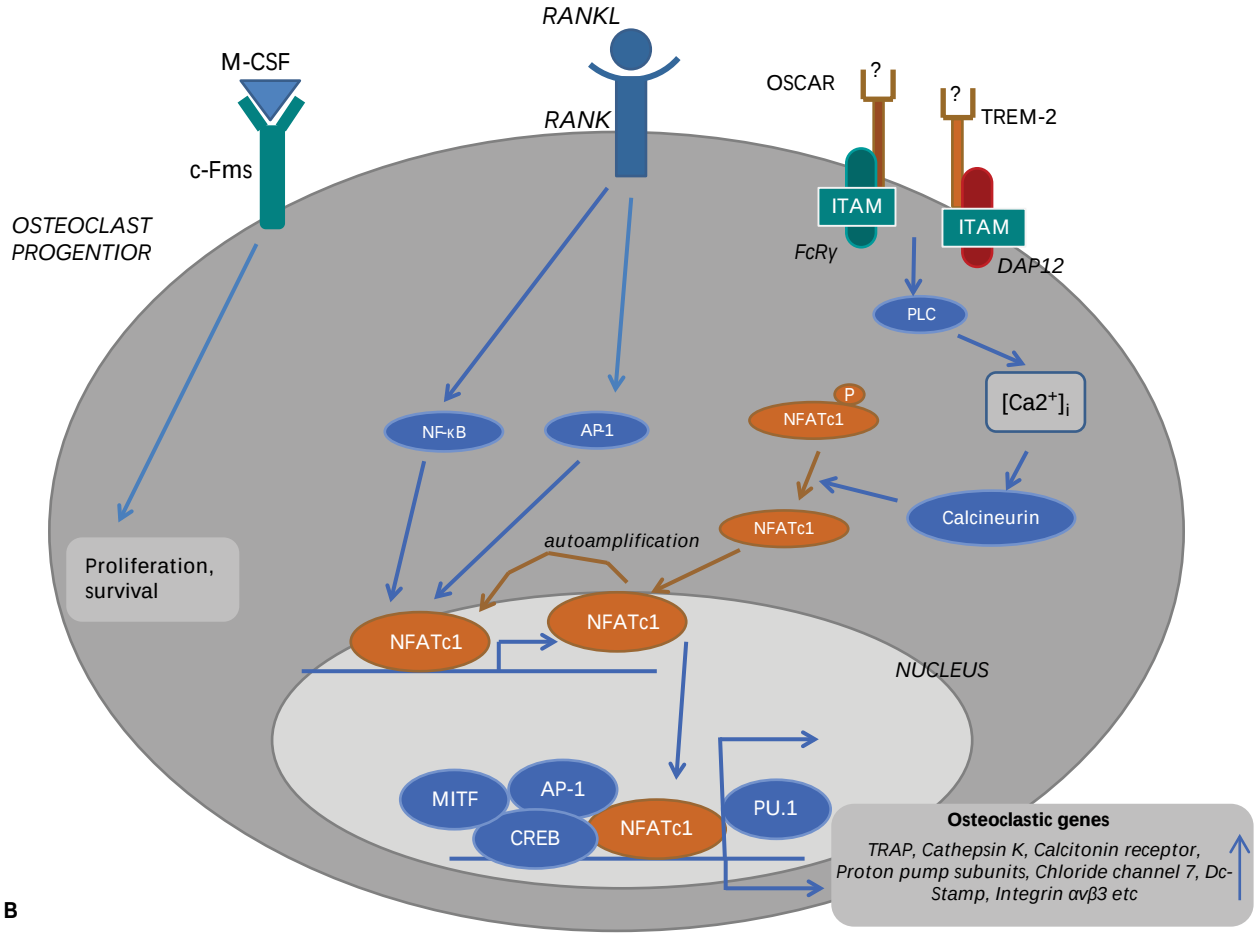



**Fig. 7.2** (*Continued*)

TNF-treated mice in which the gene for RANK is deleted, in contrast to wild type mice (33). *In vitro*, however, TNF can induce osteoclastogenesis independently of RANK, but the number of osteoclasts is considerably less than those induced by RANKL (34). Other members in the TNF superfamily include LIGHT, APRIL and BAFF. There are some reports indicating that these cytokines might also be able to induce osteoclast formation, although this is not firmly established (35). Ligation of RANKL to RANK induces the upregulation of a wide variety of osteoclastogenic genes needed for differentiation and fusion of progenitors to multinucleated osteoclasts (see later in this chapter). In parallel, several genes expressed by macrophages are downregulated.

Since RANKL is the crucial factor for osteoclastogenesis, the expression and regulation of this cytokine is critical for osteoclast formation in physiologically induced osteoclast formation, as well as in pathologic conditions with an excess of osteoclasts. Although osteoblasts and stromal cells from bone marrow are known to express RANKL, it has been shown by two independent research groups that osteocytes are the most important cells producing RANKL in physiologic bone remodeling (36, 37). When the vitamin D receptor was disrupted in osteocytes, however, RANKL formation in bones after administration of vitamin D was unaffected, as was the number of osteoclasts formed (38), indicating that cells in the osteoblastic lineage, rather than osteocytes, are important for vitamin D-induced osteoclastogenesis. Which cells express RANKL in pathologic conditions with an excess of osteoclasts is not clear. In one report it was shown that osteoclast formation in mice with experimentally induced arthritis is dependent on RANKL expression in synovial fibroblasts (39), whereas, in another report, osteoclast formation in experimentally induced periodontitis was shown to be dependent on RANKL expression by cells in the osteoblast/osteocyte lineage (40).

RANKL is most well known as a cell surface bound cytokine, which explains why osteoclast formation requires cell to cell contact between osteoclast progenitors and RANKL-expressing cells. RANKL also exists as a soluble cytokine, however, but the relative importance of cell bound and soluble RANKL for physiologic and pathologic osteoclast formation is not known.

## Intracellular signaling

The bone-resorbing activity of mature osteoclasts requires induction of several different genes making it possible for the cells to adhere to the bone surface (the integrin $\alpha_v\beta_3$), to secrete acid (proton pump proteins) and to release proteolytic enzymes (cathepsin K) dissolving organic and inorganic parts of the bone tissue (see later) (8, 41, 42). For the osteoclast progenitors to go through the genetic machinery

involved in transition of the progenitor cells to mature, polykaryonic osteoclasts, a variety of different genes need to be induced in the progenitors (Fig. 7.2A). Many of these genes encode transcription factors, i.e. intracellular proteins activated by cell surface receptors which are translocated to the nuclei where they bind to DNA and regulate gene transcription (18). These proteins are activated both at the transcriptional level (i.e. the gene expression is upregulated) and at the activation level (i.e. the proteins are translocated from the cytosol to the nuclei). Osteoclast formation requires sustained and coordinated activation of numerous transcription factors, some being NF-κB, AP-1, NFATc1, PU.1, CREB and MITF (42, 43). NFATc1 is regarded being the master regulator of osteoclast formation since overexpression of this transcription factor in the progenitor cells is sufficient for these cells to differentiate to mature osteoclasts (44). RANKL-induced osteoclastogenesis also involves downregulation of transcription factors regulating macrophage differentiation such as MafB, IRF-8 and Bcl-6 (18).

The final step in osteoclast differentiation involves fusion of the mononucleated progenitors to multinucleated mature osteoclasts (Fig. 7.2A). Although several genes have been suggested as being associated with the fusion, the molecular mechanisms involved are still elusive (45). One recent hypothesis is that *Kcnn4* (a gene encoding a calcium-activated potassium channel) plays an important role (46). Other molecules suggested as being involved in the fusion of mononucleated osteoclasts are dendritic cell-specific transmembrane protein (DC-stamp) and osteoclast stimulatory transmembrane protein (OC-stamp) (47, 48).

It is important to point out that although osteoclast progenitors are present in hematopoetic tissues such as bone marrow and spleen, as well as in the blood, the later steps in mature osteoclast formation only occur at the surfaces of bones. Therefore, mature osteoclasts are not present in bone marrow, spleen or blood. In addition, although mature osteoclast numbers are increased in the pathologic breakdown of bone, the number is not increased through the proliferation of osteoclasts; the cells that proliferate are the progenitor cells.

## Co-stimulatory immunoglobulin-like receptors in osteoclast formation

Osteoclast progenitor cells also express immunoglobulin-like receptors including OSCAR and Trem-2 (49). These receptors form complexes with the transmembrane proteins FcRγ and DAP12, respectively, which both lack extracellular ligand-binding domains but contain an ITAM domain in the cytoplasmic region needed for intracellular signaling. The ITAM motif causes calcium influx in the osteoclast progenitor cells, which is required for activation of the dephosphorylating enzyme calcineurin, which by removing phosphate from NFATc1 activates the master transcription factor of osteoclastogenesis, NFATc1 (50) (Fig. 7.2B). Genetic deletion of FcRγ and DAP12 in mice results in loss of osteoclast formation and osteopetrosis (51, 52), which demonstrates that not only is signaling through the receptors for M-CSF and RANKL needed for osteoclastogenesis, but also co-signaling through these receptor complexes. The ligands, however, activating these immunoglobulin-like receptors are essentially unknown, although OSCAR can bind to motifs in collagens type I, II and III (53). Loss-of-function mutations in the genes encoding DAP12 or TREM2 are causally related to Nasu-Hakola disease, a condition with several phenotypes, including skeletal abnormalities and defective osteoclast formation (54). Thus, M-CSF enhances the number of osteoclast progenitors and their survival, and RANKL, in collaboration with FcRγ and DAP12, induces the differentiation of osteoclasts. The differentiation process involves induction of numerous genes needed for the function of mature osteoclasts caused by several transcription factors such as NFATc1, NF-κB, CREB, MITF and PU.1 (Fig. 7.2B).

## M-CSF-, RANKL- and OPG-expressing cells

Since osteoclast formation needs coordinated signaling by co-activation of the receptors for M-CSF and RANKL, and since OPG inhibits RANKL, osteoclast formation is dependent on the synthesis and release of these three molecules. M-CSF is produced by a large variety of cell types, including stromal cells in the bone marrow, osteoblast, synovial and gingival fibroblasts, some epithelial cells and lymphocytes. The effect of M-CSF, however, is limited to the macrophage lineage due to the restricted expression of c-Fms.

Since osteoclasts are not formed unless RANK is activated, osteoclastogenesis is critically dependent on cells expressing RANKL. RANKL was initially found to be expressed in stromal cells in bone marrow and osteoblasts, and most, but not all, hormones and cytokines regulating osteoclast formation and activity do so indirectly by controlling the formation of RANKL in these cells. Recent studies have shown that physiologic osteoclast formation is regulated by RANKL expression in late osteoblasts/osteocytes rather than by bone-forming osteoblasts (36, 37). Table 7.1 lists the hormones and cytokines known to regulate RANKL in osteoblasts/osteocytes. RANKL has also been found to be expressed in T lymphocytes (27), B lymphocytes (55, 56), NK cells (57), periodontal (but not gingival) fibroblasts (58), dental pulp cells, odontoblasts, ameloblasts (59), dental follicle cells (60) and synovial fibroblasts (39). RANKL is also expressed in mammary epithelial cells, and pregnant RANKL-deficient mice do not develop lactating mammary glands, an effect dependent on stimulation of RANKL expression by progesterone (61). RANKL is also expressed in the brain and the RANKL-RANK-OPG axis has been found to have important effects in the central nervous system. Thus, RANKL and RANK are expressed in discrete areas of the hypothalamus and are found to be involved in thermoregulation (62). Interestingly, female mice lacking RANK expression in the hypothalamus have increased body temperature, and the rise of body temperature observed after ovariectomy was not seen in the RANK-deficient mice. The RANKL-RANK-OPG axis in the brain may have a protective role in ischemic stroke (63). Although RANKL was initially found to be involved in interactions between dendritic cells

and T cells, and between osteoblasts and osteoclast progenitors, the last decade has shown that RANK signaling is important also in several other biologic processes.

OPG was initially found in stromal cells and osteoblasts and to be decreased by hormones and cytokines stimulating osteoclast formation, but has since then been found to be ubiquitously expressed, including in gingival and periodontal fibroblasts. Surprisingly, however, the phenotype of mice deficient in OPG seems to be limited to the skeleton and blood vessels. Thus, mice with OPG gene knockout exhibit osteoporosis and calcification in the aorta and renal arteries (25), indicating that this soluble receptor may inhibit also cytokines other than RANKL. Interestingly, in view of the fact that RANKL-RANK signaling is important in the interaction between dendritic cells and T cells, overexpression of OPG in mice did not result in any obvious defects in the immune system (64).

## Osteoclastic resorption

Mature osteoclasts are the only cells in nature that can resorb bone, which is the reason why a skeleton can remain for hundreds of years in the ground. Although often referred to clinically as 'decalcification', an increased radiolucency on X-rays is never dependent only on decalcification of the skeleton, because when osteoclasts dissolve mineral crystals, the organic matrix is also degraded. This is not only a semantic issue since the use of the expression 'decalcified skeleton' sometimes has given rise to the misconception that such a skeleton needs calcium to be recalcified.

Since excess formation of osteoclasts is the reason for bone loss in several skeletal diseases, these cells are sometimes regarded as a threat for the skeleton in general. Osteoclasts, however, are important cells for physiologic remodeling of bone and a lack of these cells, or functional disturbances, leads to increased bone mass and the disorder osteopetrosis (65). It is, therefore, critical for a healthy skeleton that the amount of osteoclasts and their bone-resorbing activities are tightly regulated by a variety of different signaling molecules.

### Morphology

Mature osteoclasts are easily recognizable in histologic sections due to their large size and multinuclearity. They only appear on periosteal and endosteal surfaces of cortical bone, within Haversian canals, along trabecular bone surfaces, on root surfaces of teeth and occasionally on dentin surfaces in the pulp. A closely related cell, with similar morphology, can be observed on the surfaces of mineralized cartilage in the growth plate; these cells are often referred to as chondroclasts. Sometimes, osteoclasts resorbing teeth are called cementoclasts, dentinoclasts or odontoclasts.

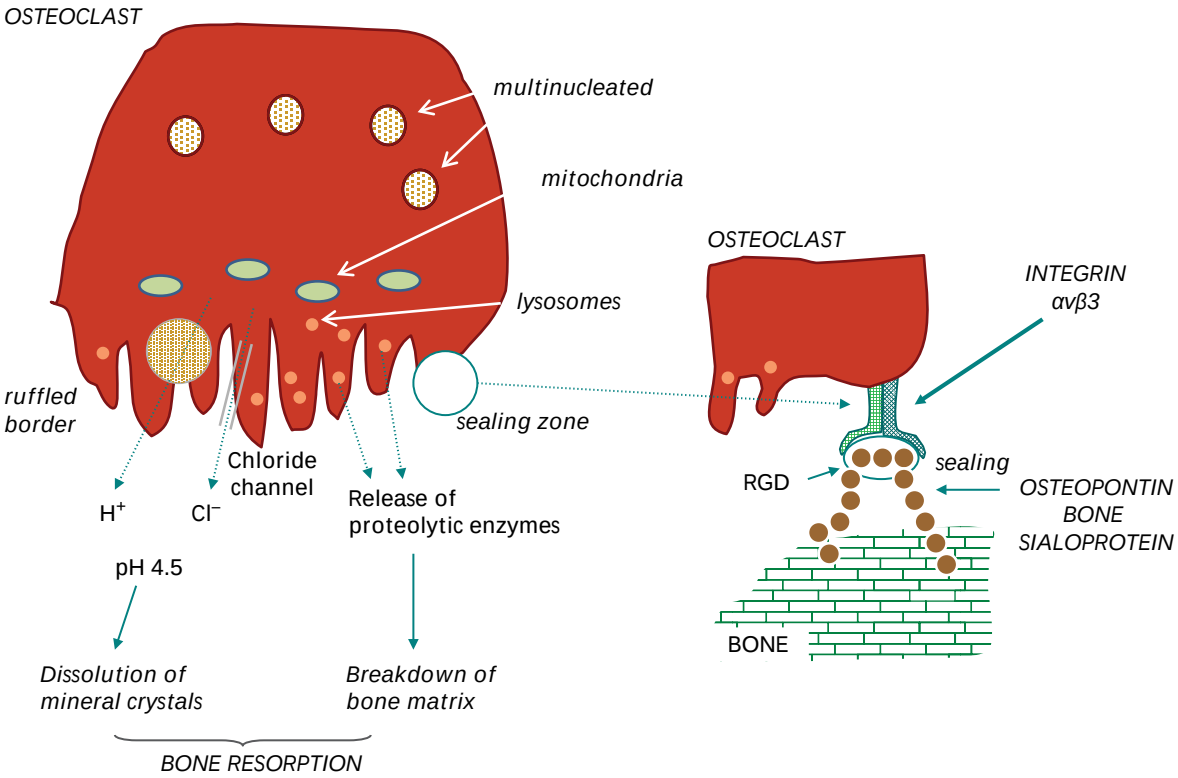
Morphologic characteristics of osteoclasts, besides large size and multinuclearity, are the ruffled border, sealing zone, high number of mitochondria and lysosomes accumulating in the ruffled border area (Fig. 7.3A).

#### *Sealing zone and ruffled border*

Since mineralized bone is covered by a layer of non-mineralized bone matrix, so-called osteoid, which in turn is covered by a layer of bone-forming osteoblasts, these two compartments need to be removed before osteoclasts can attach to bone. Therefore, the resorption process starts by osteoblasts removing the osteoid rich in collagen type I, using matrix metalloproteinases (MMPs). Subsequently, it has been considered that the osteoblasts retract from the area to be resorbed and, thereby, make the mineralized bone accessible for osteoclasts. Recently, it has been suggested that the osteoblasts do not retract but form a canopy under which resorption takes place.

The attachment of osteoclasts and the resorption process involves several cellular polarization events (66). Once the mononucleated osteoclast progenitors are fused to mature multinucleated osteoclasts on the surfaces of mineralized tissues, the cells will attach to bone by the use of an actin-rich sealing zone (Fig. 7.3B). The attachment is facilitated by interaction between the integrin αvβ3, highly expressed in the sealing zone, and sequences in bone matrix proteins containing the amino acids arginine (R), glycine (G) and aspartic acid (D) (Fig. 7.3A). Several proteins contain RGD sequences but it is generally regarded that the bone matrix proteins osteopontin and bone sialoproteins serve this function in bone (67). The interaction between the RGD motif and αvβ3 is a potential pharmacologic target for developing inhibitors of bone resorption. Several RGD-containing peptides have been synthesized and found to be able to inhibit experimentally induced bone resorption, but none has been taken to clinical trials so far.

The sealing zone encloses the Howship's lacuna in which the osteoclast cell membrane develops a characteristic, secretory ruffled border. The membrane is here crimped, tremendously increasing its resorption capacity (Fig. 7.3A). The attachment and formation of a ruffled border require intracellular signaling mediated to a large extent by Src, a tyrosine kinase (68). Genetic deletion of Src results in non-functional osteoclasts lacking a sealing zone and ruffled border (69). The ruffled border is rarely seen in light microscopy and electron microscopy is used for studies of its morphology (Fig. 7.3C). It is, therefore, most impressive that this characteristic feature of osteoclasts was described in the first report by Kölliker (1). Not much is known about how the ruffled border is formed, but mutations in the α3 subunit of the osteoclast proton pump, as well as in the genes encoding chloride channel-7 and its associated β-subunit OSTM1, which all cause osteopetrosis in humans, are associated with absence of a ruffled border. This observation indicates that these genes, most well known for participating in acidification, are also involved in lysosomal trafficking (65). The sorting nexin (SNX) family of proteins plays critical roles in protein sorting, vesicle trafficking and endosomal homeostasis. Mutations in *SNX-10* have been associated with osteopetrosis (65). Stattin et al. have recently described an osteopetrotic family with mutations in the *SNX-10* gene (292). Stimulation of $CD14^+$ blood monocytes isolated from these patients, with M-CSF and RANKL, results in

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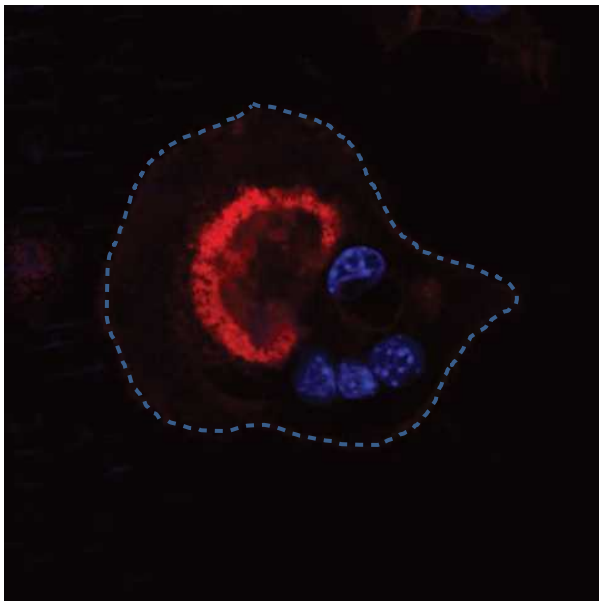

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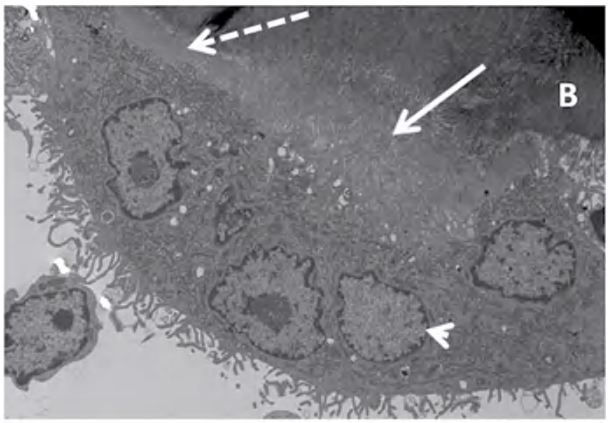
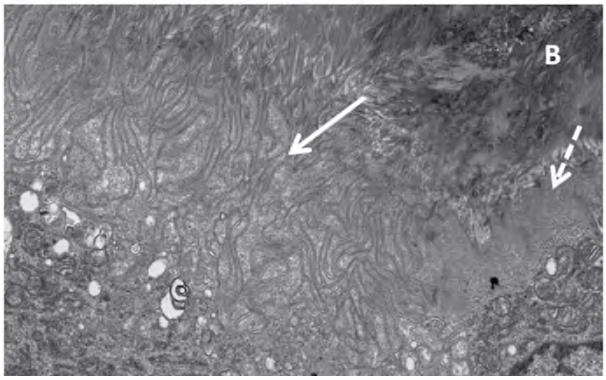



C

**Fig. 7.3** A. The mature osteoclasts are characterized by the presence of several nuclei, many mitochondria and an abundance of lysosomes in the ruffled border area. The ruffled border consists of an extension of the cell membrane in the area closest to the bone. In this area, a proton pump is expressed releasing protons into the extracellular space, which together with chloride ions released by a chloride channel creates an acid pH in the resorption lacunae leading to dissolution of the hydroxyapatite crystals in bone. This is followed by release of a cocktail of proteolytic enzymes, including cathepsin K, which degrade all the proteins in the decalcified extracellular matrix. The osteoclasts attach to bone through a sealing zone, which surrounds the ruffled border and resorption lacunae. The attachment is due to binding of the integrin αvβ3, expressed in the sealing zone, to three amino acids (RGD) present in osteopontin and bone sialoprotein in the extracellular matrix. B. The sealing zone in a mature osteoclast attached to bone is visualized by immunofluorescent staining of actin filament (red) which is an abundant protein in this zone. The nuclei in the osteoclasts are visualized in blue. The dashed line depicts the cell membrane (the whole area) of the osteoclast. Courtesy of Anh Nhi Tran, University of Aberdeen. C. An electron microscopic visualization of a mature osteoclast attached to bone (B) by an organelle-free sealing zone (dashed white arrow). The osteoclasts contain four nuclei (white arrowhead) and a ruffled border (white arrow) close to the resorption lacunae. The ruffled border is shown at higher magnification in the lower image. Courtesy of Emma McDermott, University of Aberdeen.

formation of multinucleated osteoclasts, but these cells are unable to resorb bone. The osteoclasts have sealing zones but defective ruffled borders, indicating that SNX-10 is involved in the formation of ruffled borders.

Osteoclasts resorb bone by secreting acid and proteolytic enzymes in the ruffled border (Fig. 7.3A). The resorption process, therefore, is primarily an extracellular process. Minerals and degraded matrix fragments can be taken up by osteoclasts for further degradation and transported through the cells and subsequently released through a secretory domain at the basolateral membrane (70, 71). Some of the collagen degradation fragments can be analyzed in serum or urine and used for assessment of bone resorption in patients (e.g. C-telopeptide of type I collagen, CTX). Due to the transendocytotic process, osteoclasts can stay attached to bone without accumulating degradation products in the resorption lacunae and make deep resorption tunnels while maintaining the sealing zone.

## Dissolution of minerals

The resorption process starts by active secretion of protons through a multi-subunit vacuolar type adenosine triphosphatase, which pumps protons into the ruffled border (Fig. 7.3A). To preserve intracellular electroneutrality, chloride ions are passively transported from the cytosol into the resorption area through a chloride channel (chloride channel-7). By these processes, hydrochloric acid is formed, pH is decreased to approximately 4.5 in the sealed resorption area, and the hydroxyapatite crystals are dissolved (72). The proton pumps, made up by several different proteins, are different in osteoclasts and parietal cells in the stomach, which is the reason why the gastric acid inhibitor omeprazol (e.g. Losec) does not inhibit bone resorption. The protons are generated intracellularly by the conversion of carbon dioxide and water to bicarbonate and protons, a reversible reaction catalyzed by the enzyme carbonic anhydrase-II, which is highly expressed in osteoclasts. Mice with genetic deletion of the proton pump α3 subunit (73), chloride channel-7 (74) or its β-subunit Ostm1 (75) exhibit non-functional osteoclasts and osteopetrosis. Loss-of-function mutations in these genes, or in carbonic anhydrase-II, are common reason for human osteopetrosis (65).

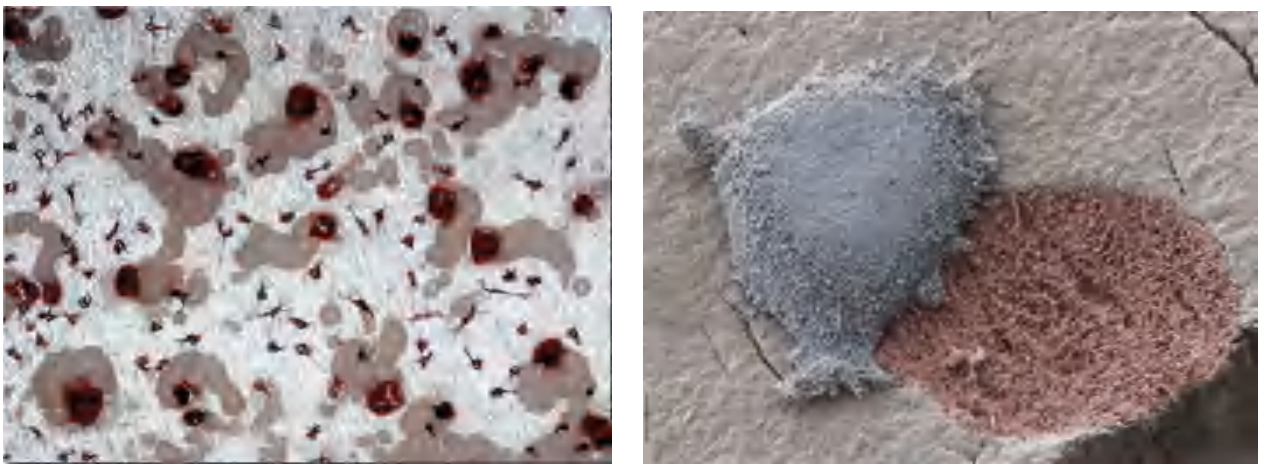
## Degradation of bone matrix

Once bone tissue is demineralized, the bone matrix proteins are accessible for proteolytic degradation. Approximately 90% of the proteins are made up of type I collagen fibers, but bone matrix also consists of a large variety of other proteins including type III collagen, thrombospondins, periostin, osteocalcin, bone sialoprotein, osteopontin, transforming growth factor-β (TGF-β), insulin-like growth factors (IGF-1, IGF-2) and small leucine-rich proteoglycans (biglycan, decorin) (76). It is not known in detail which proteolytic enzymes osteoclasts use to degrade all these proteins, but collagenolytic MMPs are not likely to be important since these enzymes are active mainly at neutral pH. In the resorption area of osteoclasts, lysosomes accumulate and fuse with the cell membrane, thus forming the ruffled border, which, therefore, is regarded as an extracellular lysosomal compartment. By this process, a cocktail of lysosomal enzymes are released into the resorption lacuna (Fig. 7.3A). Most attention has been paid to the secretion of lysosomal enzymes in the family of cysteine proteinases, which have an acid pH optimum. One of these family members is cathepsin K, which is highly expressed by mature osteoclasts (77), and which is able to degrade type I collagen molecules at several sites (78). Mice with genetic deletion of cathepsin K have increased bone mass and increased amounts of demineralized, but partially degraded, bone matrix in the resorption lacunae (79), demonstrating the crucial role this enzyme has in osteoclastic bone resorption. The cathepsin K knockout mice, however, although having increased bone mass, are not osteopetrotic, which shows that resorption is not totally blocked and that other enzymes also are involved. Congenital mutation in the cathepsin K gene is the cause of pycnodysostosis (also called Toulouse-Lautrec syndrome), a human disease with several phenotypes including short stature and dense bones (80).

Several low molecular weight inhibitors of cathepsin K have been developed and found to decrease experimentally induced bone resorption. One of these compounds, odanacatib, has been taken to clinical trials and a phase II study showed that bone mass was progressively increased in postmenopausal women (81). As expected, serum markers of bone resorption were decreased but, interestingly, this was not associated with decreased markers of bone formation, similar to what has been found for bisphosphonates or denosumab (see later in this chapter). The clinical trial has been extended to a phase III fracture trial, but its approval remains on hold due to an increased number of serious side effects such as atrial fibrillation, stroke, cerebrovascular events and overall mortality. These observations indicate that cathepsin K may be involved in more processes than degradation of type I collagen. Therefore, a new strategy is to develop exosite inhibitors of cathepsin K specifically blocking bone resorption (82).

Similar to macrophages and several other hematopoetic cells, mature osteoclasts are highly motile cells and, therefore, able to move along bone surfaces creating several resorption lacunae (Fig. 7.4A). Although MMPs expressed by osteoclasts (mainly MMP-9 and MMP-14) are most likely not participating in the breakdown of bone matrix in the resorption lacunae, these enzymes seem to be involved in the migration of osteoclasts from one lacuna to another to establish a new resorption site (83).

When osteoclasts have left the resorption lacunae, fragments of demineralized matrix proteins can still be observed in the lacunae (Fig. 7.4B). These remnants will then be cleaned up by mononuclear cells entering the lacunae. Previously, it was anticipated that these cells were monocytes, but during recent years it has been shown that these reversal cells are osteoblast-lineage cells and that they are involved in the coupling between bone resorption and bone formation (see later) (84).



A B

**Fig. 7.4** A. Osteoclasts (red cells) are highly motile cells which can create both resorption pits and resorption trenches when they move along a bone surface. Small red cells observed between the resorption areas are mononucleated osteoclast progenitors. B. Remnants of demineralized bone matrix can be observed when an osteoclast (grey cell) has left a resorption lacuna. Courtesy of Professor Tim Arnett, University College of London.

## Role of the enzyme tartrate resistant acid phosphatase

Staining for the enzyme TRAP is widely used as a marker for terminally differentiated mature osteoclasts (Fig. 7.1A), but TRAP is also expressed by mononuclear osteoclast progenitors at late stages. It is an enzyme which at acid pH can dephosphorylate a wide range of phosphorylated substrates (85). The physiologic role of this enzyme is, however, far from clear. One possible substrate for TRAP in osteoclasts might be phosphorylated osteopontin, and dephosphorylation of osteopontin by TRAP may be a mechanism to regulate osteoclast attachment and motility (86). TRAP exists in two isoforms; TRAP5b is mainly expressed in osteoclasts and TRAP5a is expressed in monocytes, but also in several other cells. Assessment of TRAP5b in serum is used as a parameter of bone resorption *in vivo*.

Loss-of-function mutation in the TRAP gene is the cause of spondyloenchondrodysplasia, a disease where patients exhibit 'cartilage within bone', but also neurologic pathologies with brain calcifications and autoimmune diseases (87, 88). In line with the observation that TRAP can serve as a substrate for osteopontin (86), patients with spondyloenchondrodysplasia have increased amounts of phosphorylated osteopontin in the serum. It is speculated that this might be the basis for the autoimmune diseases seen in patients with TRAP mutations, because osteopontin can regulate activities of dendritic cells as well as of T cells, and because serum levels of osteopontin are correlated with disease activities in certain autoimmune diseases.

## Regulation of RANKL-induced differentiation of mononuclear osteoclast progenitors

Mononuclear osteoclast progenitor cells express a variety of receptors that can affect signaling through the RANKL and M-CSF receptors, some of which are listed in Table 7.1. The ligands for these receptors are mostly derived from cells in the vicinity of the osteoclast progenitors, either from osteoblasts or immune cells, but can also be systemically derived. The relative importance of local or systemic factors affecting RANKL and M-CSF signaling in physiologic or pathologic osteoclastogenesis remains to be elucidated.

**Table 7.1** Effects of hormones and cytokines on the production of RANKL in osteoblasts.

| | | Stimulation | Inhibition |
|---|---|---|---|
| **Hormones** | Parathyroid hormone | + | |
| | $1,25(OH)_2$-vitamin D3 | + | |
| | 17β-oestradiol | | + |
| | Testosterone | | + |
| | Cortisone | + | |
| | Vitamin A (ATRA) | + | |
| **Cytokines** | Interleukin-1 | + | |
| | Interleukin-4 | | + |
| | Interleukin-6 | + | |
| | Interleukin-11 | + | |
| | Interleukin-13 | | + |
| | Interleukin-17 | + | |
| | Interleukin-20 | + | |
| | Tumor necrosis factor | + | |
| | Leukemia inhibitory factor | + | |
| | Oncostatin M | + | |
| | Cardiotrophin-1 | + | |

## Hormones

Sex steroids are well known to affect osteoclast formation. An increased number of osteoclasts is an important reason why bone mass is lost in sex steroid deficiency such as postmenopausal osteoporosis in women (89, 90). Osteoclast progenitors, as well as mature osteoclasts and osteoblasts,

express estrogen receptors, mainly estrogen receptor α. On important reason for an increased number of osteoclasts in estrogen deficiency is regarded to be due to the increased lifespan of mature osteoclasts (91, 92). Estrogen has also been shown to inhibit osteoclast formation by interfering with RANK signaling (93, 94). Estrogen receptors expressed by osteoblasts also seem to regulate osteoclast formation. It has been shown that formation by osteoblasts of osteoclastogenic cytokines (95), including RANKL (96), is decreased by estrogen and, therefore, may contribute to increased formation of osteoclasts during estrogen deficiency.

Calcitonin receptors are highly upregulated during osteoclast differentiation and activation of these receptors on mature osteoclasts by calcitonin rapidly inhibits their motility and bone-resorbing activity (see later) (97). The effect has been suggested to be particularly important when high calcium demand causes excessive bone resorption such as during lactation. Calcitonin might decrease bone resorption also by activation of calcitonin receptors expressed by osteoclast progenitor cells (98, 99) (Fig. 7.5). This effect is exerted at a late stage of differentiation since the expression of most osteoclastic genes is unaffected and it is therefore likely that calcitonin inhibits the fusion process.

Glucocorticoids are the largest risk factor for secondary osteoporosis (100). Osteoclast progenitors express glucocorticoid receptors that belong to the superfamily of nuclear receptors, similar to estrogen and retinoid receptors. When these receptors are ligated they bind to DNA and regulate gene transcription (101). Conaway et al. have recently observed that treatment of osteoclast progenitors from mouse bone marrow with glucocorticoids do not affect the number of RANKL-stimulated osteoclasts but increases their bone-resorbing activity (102) (Fig. 7.5), which might be one explanation why patients treated with high doses of glucocorticoids lose bone rapidly after onset of treatment.

Retinoids (vitamin A) are often regarded as being a potential risk factor for osteoporosis based upon epidemiologic data (103, 104). Observations of reduced cortical bone in rodents treated with very high doses of retinoids, and *in vitro* experiments showing that retinoids can stimulate bone resorption in organ cultured bones, further support the clinical data (105). There are, however, also studies in which dietary intake of vitamin A, or circulating levels of retinoids, have not been associated with decreased bone mass (105, 106). Surprisingly, activation of osteoclastic retinoid receptors on human and mouse osteoclast progenitors abolishes RANKL-induced osteoclast formation *in vitro* (107, 108) (Fig. 7.5). It remains to be clarified how these observations relate to the increased formation of periosteal osteoclasts in rodents treated with high concentrations of retinoids. More studies, in which rodents are treated with clinically relevant doses of retinoids, and in which osteoclast formation on periosteal, endosteal and trabecular bone is assessed, need to be performed to get better insight into what role retinoid receptors in osteoclast progenitors might have.

## Cytokines

Osteoclast progenitors express numerous different cytokine receptors, most of which inhibit RANKL-induced osteoclastogenesis, but some of which potentiate the action of RANKL (Fig. 7.5).

To the group of inhibitory cytokines belong IL-4, IL-10, IL-12, IL-18, IL-27, interferon-β (IFN-β), IFN-γ and GM-CSF (18, 109). These cytokines are produced mainly by immune cells and have been implicated as regulators of inflammation-induced osteoclast formation. IFN-β, however, is upregulated by the osteoclast progenitor cells themselves in response to RANKL and acts as an inhibitor in an autocrine, negative feedback manner by suppressing RANKL-induced expression of c-Fos, a subunit in the osteoclastogenic AP-1 transcription factor (110).

To the group of cytokines potentiating the effect of RANKL belong TNF-α, IL-20, IL-23 and TGF-β. Although TNF-α is

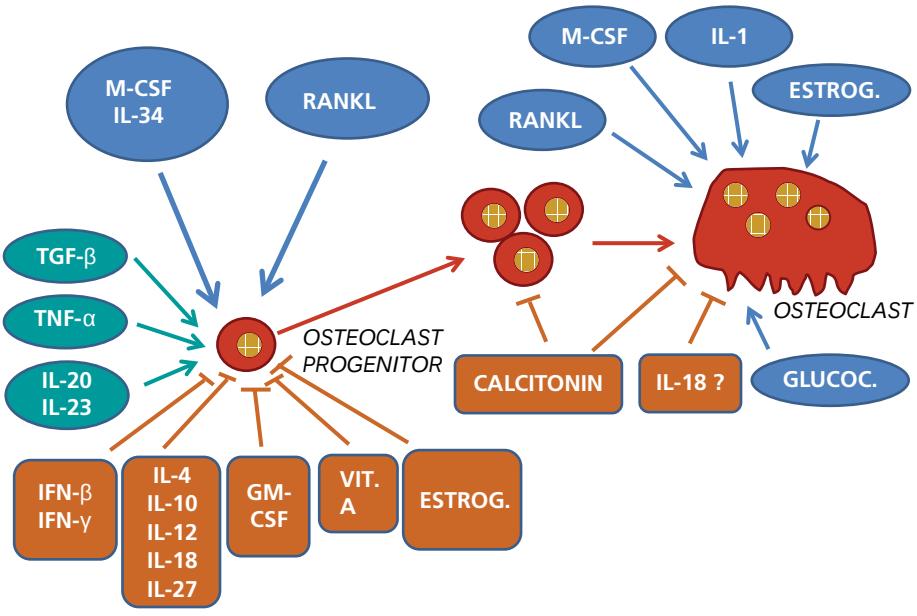



**Fig. 7.5** Formation and activity of mature osteoclasts are regulated by a variety of different hormones and cytokines. Some of these molecules act on early osteoclast progenitors, either as stimulators or inhibitors, one of the hormones acts on late osteoclast progenitors, and several on mature osteoclasts. Stimulatory molecules are shown in blue and inhibitory in brown.

unable to stimulate osteoclast formation in RANK null mice, this cytokine synergistically potentiates osteoclast stimulation by RANKL used at concentrations not affecting osteoclastogenesis by itself (4). The role of the potentiating cytokines is most likely to regulate osteoclastogenesis during inflammation, but it has been reported by Pacifici *et al.* that TNF-α expression in a subset of T cells might be involved in bone resorption observed in mice with estrogen deficiency (111).

## Wnts

Wnts consist of a large family of extracellular proteins which have a variety of effects on many different cell types. These signaling molecules are most well known for their effects on embryonic development and cell proliferation, but they have been implicated in many physiologic and pathologic processes, including bone formation and resorption (112, 113). In humans, 19 different Wnts have been found, of which several affect bone cells. Wnts bind to the non-signaling, cell membrane proteins lipoprotein receptor-related protein-5 (Lrp5) or Lrp6, which form complexes with seven-transmembrane, G-protein-coupled, signaling receptors called Frizzled (Fig. 7.6A). In humans, 10 different Frizzleds have been described, but it is not known, yet, which Frizzleds are most important in osteoblasts and osteoclasts. In addition, several inhibitors of Lrp/Wnt/Frizzled signaling exist, some of them acting by binding to the extracellular parts of Lrp and, thereby, interfering with its binding to Frizzled. To this family belongs sclerostin and Dickopfs (1–4). Other inhibitors act as scavengers neutralizing free Wnts. The important role of Lrp/Wnt/Frizzled signaling in bone was discovered when loss-of-function, as well as gain-of-function, mutations in the *LRP5* gene were found to cause decreased and increased bone mass in humans, respectively (114–116). The fact that loss-of-function mutations in the sclerostin gene was found to be the cause of high bone mass in patients with sclerostosis (117) and van Buchem's disease (118), further demonstrated the important role of this system for bone remodeling. The fact that sclerostin is a regulator of human bone mass, and is specifically expressed in osteocytes, has led to development of antibodies neutralizing sclerostin. These antibodies are now used in clinical trials for treatment of patients with postmenopausal osteoporosis in order to increase bone mass by enhancing the bone-forming activity of osteoblasts (119). The long-term effect, however, on bone mass and on fractures remains to be shown.

Initially, Lrp/Wnt/Frizzled signaling was found to be an important regulator of bone mass through receptors in osteoblasts but during recent years this signaling system has been found also to regulate osteoclasts (112, 113).

Canonical Lrp/Wnt/Frizzled signaling results in dephosphorylation of β-catenin, translocation of β-catenin to the

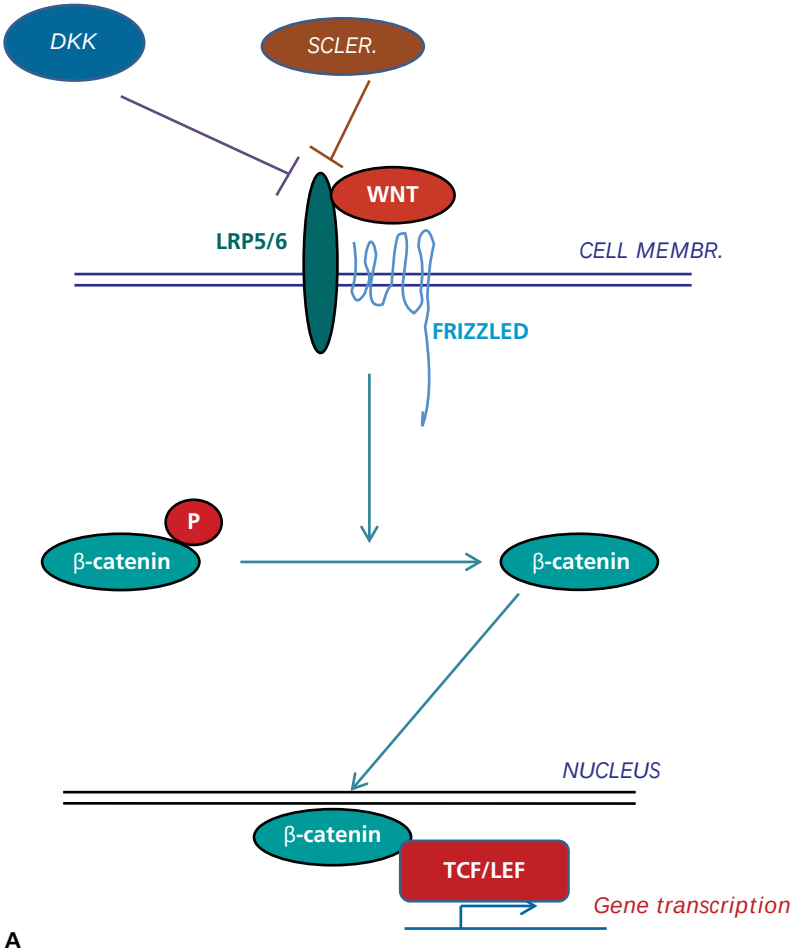



**Fig. 7.6** Wnt signaling is an important regulator of a wide variety of cellular activities, including bone formation and resorption. A. Binding of Wnt to the co-receptor Lrp5/6 results in activation of the signaling receptor Frizzled, which will activate β-catenin in the cytoplasm through preventing its phosphorylation. Active β-catenin then translocates to the nucleus and activates the transcription factor TCF/Lef, which then controls transcription of different genes. B. Some Wnts act on osteoclast progenitor cells and interact with M-CSF/RANKL-induced osteoclast differentiation. Wnt5a potentiates osteoclastogenesis, whereas Wnt3a, Wnt4 and Wnt16 inhibit it. C. Wnt16 is produced exclusively by osteoblasts in cortical bone and inhibits osteoclast differentiation by acting directly on osteoclast progenitors, and by acting in an autocrine manner to increase production of the osteoclastogenesis inhibitor osteoprotegerin (OPG).

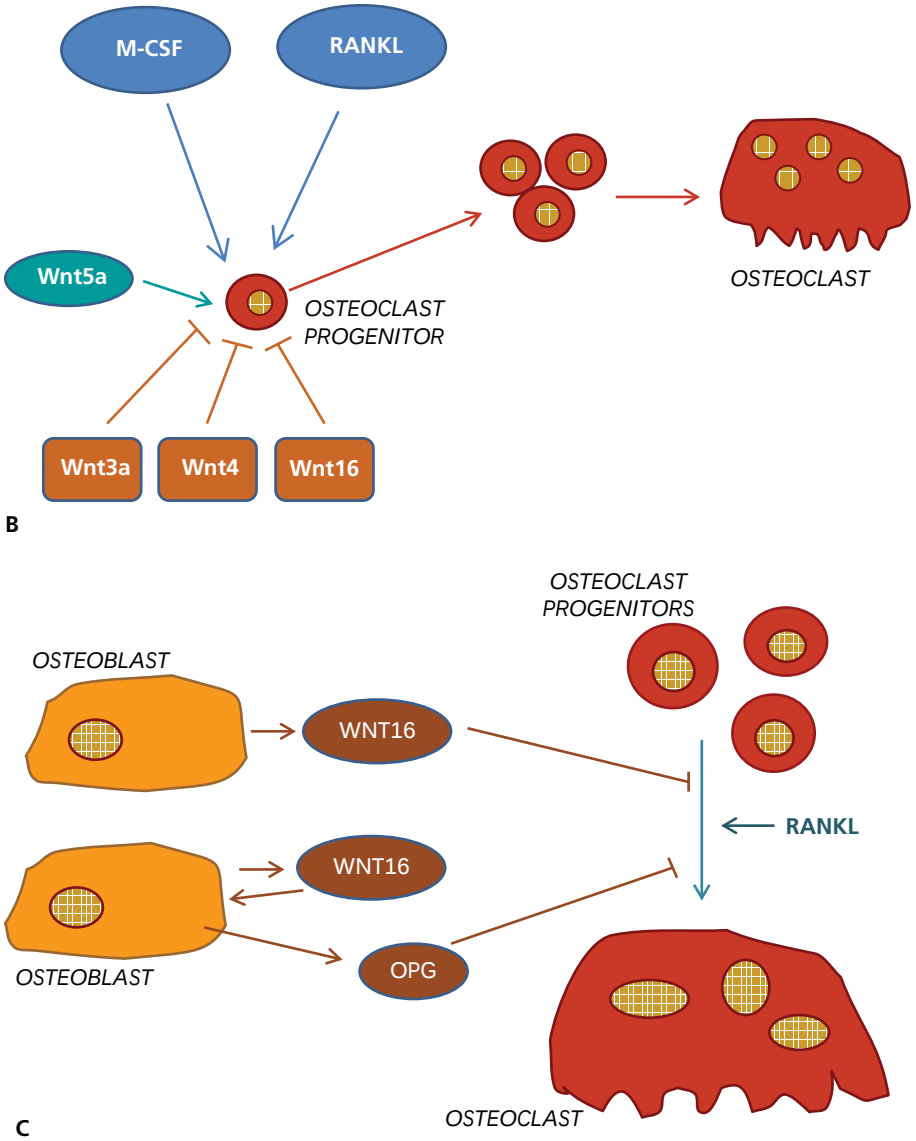



**Fig. 7.6** (*Continued*)

nucleus, and subsequent activation of the Tcf/Lef transcription factors regulating a variety of genes (Fig. 7.6A). Wnts can also regulate cellular activities through non-canonical signaling by activating Frizzled independently of Lrp (120).

Wnts regulating osteoclastogenesis include Wnt5a (Fig. 7.6B), which can be produced by osteoblasts and which can potentiate RANKL-induced osteoclast formation in mice both *in vivo* and *in vitro* by acting through Ror2-dependent non-canonical signaling (121). In contrast, Wnt3a, Wnt4 and Wnt16 inhibit osteoclast differentiation.

Using a translational approach, research teams have demonstrated the crucial role of Wnt16 for cortical bone. Based upon the observations that the *WNT16* gene in humans is associated with cortical, but not trabecular, bone mass, and with fracture risk of the distal radius (122, 123), it has been shown in mice that Wnt16 is produced by osteoblasts and acts on osteoclast progenitors to block RANK signaling (124) (Fig. 7.6C). Thus, global disruption of the *Wnt16* gene in mice resulted in decreased cortical bone mass with no effect on trabecular bone, an effect associated with an increased number of cortical osteoclasts and with no effect on bone formation. Cortical thickness was reduced to such an extent that mice experienced spontaneous fractures, a most rare phenotype in mice with gene deletions. Specific deletion of *Wnt16* in osteoblasts mimicked the phenotype of global deletion, but disruption of *Wnt16* in osteocytes did not result in any effect. These observations, together with the findings that Wnt16 protein is expressed by cortical osteoblasts and that Wnt16 is expressed in isolated osteoblasts and upregulated during differentiation, demonstrate that Wnt16 in osteoblasts is crucial for cortical thickness. In mechanistic studies, it was further demonstrated that Wnt16 inhibits RANKL-induced osteoclast formation both by an autocrine effect increasing OPG and by a paracrine effect targeting osteoclast progenitors. Besides showing the important role of Wnt16 for cortical thickness, the observations also demonstrate that cortical and trabecular bone mass are differentially regulated (125).

Knowing that Wnt16 is a specific regulator of cortical bone mass it would be possible to develop drugs specifically affecting Wnt16 expression, or Wnt16 signaling, to increase cortical bone mass. There is a need for these types of drugs since

currently used drugs for treatment of osteoporosis mainly affect trabecular bone and fractures in the spine. Recently, it has been reported that mice that overexpress *Wnt16* specifically in osteoblasts have increased bone mass (126).

### Semaphorin

Semaphorins are most well known as axon guidance molecules, but have been shown also to affect bone cells. Sema3B is a semaphorin expressed by osteoblasts and found to potentiate RANKL-induced osteoclast formation (127). In contrast, Sema3A increases bone mass due to increased formation with no effect on osteoclasts (128). Although semaphorins have been found to regulate both osteoclast and osteoblast activities it might be that the effects *in vivo* are mediated by effects on nerve development rather than by direct effects on bone cells (128).

### Ephrin

Ephrin ligands and their receptors (Ephs) are membrane-bound proteins that act through cell to cell interactions and regulate a variety of processes both in embryonic and adult life. An unusual feature is their capacity for bidirectional signaling, i.e. stimulation of Ephs by ephrin ligands results in tyrosine kinase activation and downstream signaling in the receptor-expressing cells, but also in reverse signaling in cells expressing the ligand.

Ephrin B and its cognate receptor EphB4 are both expressed on osteoblasts and osteoclasts. Activation of EphB4 on osteoclast progenitors by ephrinB2 inhibits RANKL-induced osteoclast formation (129). Reversed signaling through interactions between ephrinB2 on osteoclast progenitors and EphB4 on osteoblasts also inhibits RANKL-induced osteoclast formation (130). Although effects by this signaling system have been demonstrated both *in vivo* and *in vitro*, its roles in physiologic and pathologic osteoclastic bone resorption remain to be elucidated (131).

## Regulation of mature osteoclast activity

The amount of bone being resorbed is to a large extent dependent on mechanisms affecting differentiation of osteoclast progenitors and the number of osteoclasts, but mechanisms controlling the activity of mature osteoclasts are also important.

Studies on which receptors are expressed on mature osteoclasts and their role are less common compared to the role of receptor expression on osteoclast progenitors. To some extent, this is due to the difficulties of obtaining pure mature osteoclasts and in lifting such cells onto bone disks for assessing their activities, although some methods are in use.

Calcitonin receptors do not exist in osteoclast progenitors but are highly upregulated during osteoclast differentiation and are abundantly expressed in mature osteoclasts. Their expression is used as a hallmark of mature osteoclasts, together with positive staining for the enzyme TRAP, and their multinuclearity. Activation of calcitonin receptors on mature osteoclasts causes a rapid contraction, loss of ruffled border, ceased motility and inhibition of bone resorption (132) (Fig. 7.5). For this reason, calcitonin was one of the first drugs used to treat postmenopausal osteoporosis, once it was discovered that bone loss with increasing age was not a consequence of old age but caused by loss of estrogen. However, the inhibitory effect by calcitonin is lost within hours and, therefore, this hormone is not used any more for treatment of osteoporosis. It is believed that calcitonin, produced by thyroid C cells, acts to decrease bone resorption in situations with hypercalcemia, but the physiologic role is debated since neither excess, nor lack, of calcitonin seems to affect bone mass or serum calcium. Disruption of the calcitonin gene, or of the gene encoding calcitonin receptor, results in increased bone resorption, as expected, but does not result in bone loss (133–135). On the contrary, bone mass is increased, surprisingly due to increased bone formation. The effect is mediated by osteoclasts, which in the absence of signaling through calcitonin receptors, release sphingosine-1-phosphate acting through its receptor S1PR3 in osteoblasts stimulating their anabolic activities (136). This knowledge opens up new possibilities for development of osteo-anabolic drugs stimulating bone formation.

RANKL is not only required to stimulate formation of osteoclasts, but this cytokine is also necessary for keeping the mature osteoclast alive and active. Using highly purified mature osteoclasts, it has been shown that the stimulatory effect by RANKL on osteoclast activity is potentiated by M-CSF, independent of survival (137).

IL-1 exists in two forms, IL-1α and IL-1β, which are products of different genes. IL-1α and IL-1β act via the same receptor and can stimulate osteoclast formation through increased RANKL expression in osteoblasts, but have also been shown to increase the survival of mature osteoclasts (138) (Fig. 7.5).

IL-18 belongs to the IL-1 family of cytokines and is, similar to IL-1β, converted in the inflammasome from the 24 kDa precursor form to the 18 kDa mature form (139). In cell cultures, IL-18 inhibits both osteoclast formation and activity, through mechanisms dependent on IFN-γ and GM-CSF (140). It is, however, doubtful if this occurs *in vivo* since several studies report that IL-18 enhances bone resorption and osteoclast formation in collagen-induced arthritis in mice (141, 142).

## Osteoclast progenitor origin

It is well established that osteoclasts are derived from progenitor cells in the macrophage/monocyte lineage present in haematopoetic tissues in bone marrow, spleen or fetal liver. Osteoclast progenitors are also present in the monocytic fraction in blood and it has been estimated that 1–4% of these cells are osteoclast progenitors. How osteoclast

progenitors are recruited from haematopoetic tissues to surfaces of trabecular and cortical bone is not fully clear. The common view is that the progenitors are primarily recruited from blood to bone by some kind of homing process. That means that bone cells must be able to communicate specifically with the osteoclast progenitors present in the circulation.

Genetic and pharmacologic studies indicate that sphingosine-1-phosphate (S1P), produced by sphingosine-1-kinase, and acting through five different S1P receptors (S1PRs), may be important for the trafficking of osteoclast progenitors. Osteoclast progenitor cells have S1PR type 1 (S1PR1), but these receptors are downregulated during osteoclastogenesis and, therefore, less abundant on the mature osteoclasts (143). The vasculature produces a lot of S1P which might be a mechanism to keep the progenitors in blood. When the gene encoding S1PR1 is genetically deleted, the mice lose bone mass due an abundance of mature osteoclasts on bone surfaces, which demonstrates the importance of this receptor for the number of osteoclasts on bone surfaces (143). In agreement with this finding, ovariectomized mice lose much less bone if the interaction between S1P and S1PR is blocked by the compound FTY720, which blocks S1PR1 (143).

Another factor that may contribute to the recruitment of osteoclasts to bone surfaces is stromal cell-derived factor 1 (SDF-1), also called CXCL12 (chemokine (C-X-C) motif ligand 12), which is expressed on osteoblasts. This ligand binds to receptors called CXCR4 which are present on both osteoclast progenitors and mature osteoclasts. Since CXCL12 is highly chemotactic for osteoclast progenitors this ligand might contribute to homing of osteoclast progenitors to bone (144).

It seems as if two opposite mechanisms are involved in the trafficking of osteoclast progenitors from blood to bone: S1P keeping the cells circulating and CXCL12 trying to attract them to bone.

## Physiologic role of osteoclasts

One important function of osteoclasts is to resorb old or damaged bone. Bone tissue needs to be continuously renewed throughout life and a large number of osteoclasts at the surfaces of cortical bone, within cortical bone and at the surfaces of trabecular bone, are engaged in resorbing old bone. For bone mass to be preserved during physiologic circumstances, osteoblasts are recruited to the resorption lacunae and fill up these with new bone. This turnover of bone is called remodeling and needs to be tightly controlled by several local and systemic factors, acting in concert to regulate osteoclast differentiation and activity, and in interplay with factors controlling differentiation and function of osteoblasts. Activities by such factors also include a signaling network to control initiation and termination of osteoblast and osteoclast activities.

The sequential activities of osteoclasts and osteoblasts during remodeling were first described by Harald Frost and collaborators (145). The remodeling processes take place at restricted areas on bone surfaces and in cortical bone asynchronously at sites geographically and chronologically separated (131, 146), and these areas are called bone multicellular units (BMUs) (147, 148). It has been estimated that $1–2 \times 10^6$ BMUs are present throughout the skeleton and that 4–10% of the skeleton is remodeled each year, which results in a totally new skeleton every 10th year. Detailed histologic analyses in the human skeleton during recent years have shown that the remodeling in BMUs take place under a canopy of bone lining cells (149). The BMUs together with a network of capillaries covering the bone lining cells are now referred to as the bone remodeling compartment (BRC) (150).

### Remodeling

During the quiescent phase, bone is covered by inactive, flat osteoblasts called bone lining cells. Previously, it was thought that these cells retracted from bone during bone remodeling in order to make bone accessible for osteoclasts. It is now thought that remodeling takes place under a canopy of these lining cells. The current understanding is that bone remodeling starts by microfractures in old bone leading to osteocyte apoptosis and expression of RANKL (151) (Fig. 7.7). Osteoclast progenitors are recruited through the canopy and then become differentiated to mature osteoclasts which start resorbing old damaged bone. In human bone, it is estimated that the resorption phase goes on for 3 weeks (152). Subsequently, a reversal phase of approximately 5 weeks follows (152). During this phase, mononuclear cells enter the resorption lacunae. These cells clean up matrix remnants left by osteoclasts and then deposit a cement line at the bottom of the lacunae. This cement line serves as a marker to show where new bone has been formed in areas with previous resorption. It was previously believed that the reversal cells were of monocyte/macrophage origin, but recently it has been found likely that these cells are osteoblast progenitors which gradually differentiate to bone-forming osteoblasts (84). These two phases are followed by new bone formation in the lacunae which takes approximately 3–4 months until the resorption lacunae is filled up with new bone (153). Under physiologic conditions, the amount of bone formed is equal to that being resorbed and bone mass is preserved. Under pathologic conditions, however, the amount of bone formed is usually less than that resorbed and bone mass decreases, such as in postmenopausal osteoporosis caused by lack of estrogen.

Much effort has been devoted to investigating why resorption in the BRC is followed by bone formation. How are osteoblast progenitor cells recruited to the resorption lacunae and what stimulates their differentiation to mature bone-forming osteoblasts? It has been postulated that this is due to coupling factors released during resorption and subsequently acting on the osteoblast. Numerous factors have been suggested to serve as coupling factors between

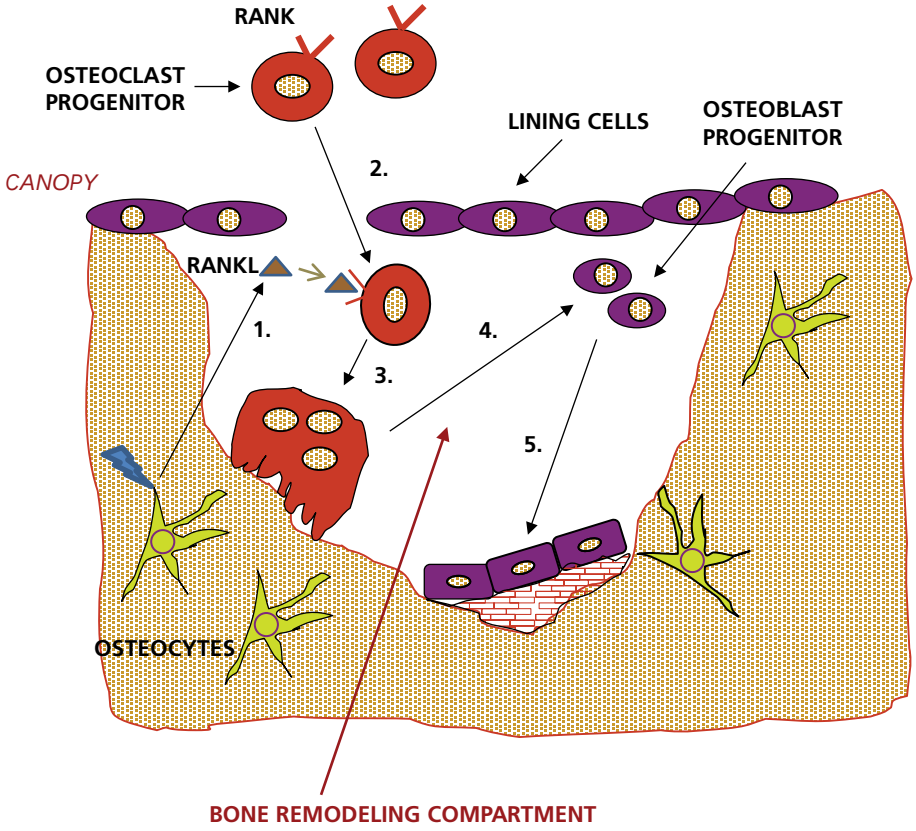



**Fig. 7.7** Bone remodeling is a process by which old bone is substituted for with new bone. This process starts by microcracks resulting in osteocyte apoptosis (blue jagged arrow) and release of RANKL (1). Osteoclast progenitors enter the area through the canopy of lining cells (2) which cover bone surfaces. Mature osteoclasts are formed that resorb the old, damaged bone (3). During this process, coupling factors are released that recruit and activate osteoblast progenitor cells (4), which then starts to produce new bone in the resorbed area (5). Under physiologic, balanced bone remodeling, osteoblasts will fill up the whole resorbed area with new bone and bone mass will be preserved.

resorption and formation (131, 154, 155). These can be divided in three categories: (1) factors released from the bone matrix during resorption; (2) factors released by the osteoclasts themselves; and (3) factors present on osteoclast surfaces.

Bone matrix constituents produced by osteoblasts and present in bone matrix do not only include collagen type I and other proteins making up a scaffold for mineralization, but also molecules with cell signaling capacities. To this group belong TGF-β, IGF-1, bone morphogenetic protein 2 (BMP-2) and platelet-derived growth factor (PDGF) which are released during resorption and which all can stimulate osteoblastic proliferation and differentiation and, therefore, potentially serve as coupling factors.

Osteoclasts also can produce and secrete factors that might be involved in the coupling process. To this group of coupling factors belongs BMP-6, Wnt10b, cardiotrophin-1 and sphingosine-1-phosphate. Osteoclast inhibitors such as bisphosphonates and denosumab not only reduce bone resorption, but also bone formation due to the fact that when fewerosteoclasts are present, fewer osteoblasts will be stimulated through coupling. Therefore, these drugs cause not only inhibition of bone resorption but also reduced bone formation, which decreases their effectiveness. The new antiresorptive drugs such as cathepsin K inhibitors do not affect osteoclast formation, but decrease their bone-resorbing activity and are not associated with decreased bone formation (156), most likely due to the observation that both active and inactive osteoclasts seem to produce coupling factors (157).

EphrinB2 and semaphorin belong to the group of osteoclast membrane-bound factors and have been discussed earlier in this chapter.

As pointed out by Sims and Martin (155), the matrix-derived, or osteoclast-secreted, factors may not remain in the BMUs during the reversal phase long enough to affect osteoblasts entering the lacunae during the formation phase. It might be that they affect the recruitment, proliferation or differentiation of osteoblast progenitors at earlier stages. It may also be questioned why these proteins, similar to other bone matrix proteins, are not degraded by the osteoclast proteolytic enzymes. As regards the membrane-bound factors, it may be argued that the osteoclasts do not make contact with bone-forming cells in the remodeling lacunae. However, the recent finding that osteoclasts are in close contact with osteoblastic cells in the reversal phase (84) suggests that such cell to cell contact may be involved in triggering osteoblastic differentiation in the resorption lacunae.

Although the search has been focused on trying to find an individual coupling factor, it is most likely that the coupling process is more complicated and that it will turn out that a series of factors are involved. It is also likely that cell types other than bone cells are involved. The latter possibility is indicated by a report showing that a subtype of macrophages present in the periosteum of mouse bones, so-called osteomacs, seems to be important for local bone formation (158). There are also reports suggesting that T cells may be involved in remodeling of bone (159), including in PTH-stimulated bone formation (160).

## Modeling

Whereas remodeling is a process where new bone formation is seen in areas of previous resorption, modeling stands for either new bone formation in areas without preceding bone resorption, or bone resorption not followed by new bone formation. Modeling mainly occurs on cortical bone and is a process which will change the shape and size of bones. Orthodontic tooth movement with resorption and formation on opposite surfaces of the alveolar bone surrounding the roots is one example of modeling. The process by which a canted fracture heals with formation of a straight bone is another example (Fig. 7.8).

## Calcium balance

Bone remodeling and osteoclasts are not only important in replacing old bone but also for calcium homeostasis. It is crucial for many cellular functions that calcium concentration in blood is kept at a constant level. Calcium homeostasis is regulated by bone resorption, kidney excretion and intestinal uptake (161). Decreasing calcium concentrations are sensed by the parathyroid cells which synthesize and secrete increasing amounts of PTH. This hormone stimulates osteoclast formation and bone resorption and, thereby, transfers calcium from bone to blood. In the kidneys, PTH increases the synthesis of $1,25(OH)_2$-vitamin D3 and the reabsorption of calcium from urine to blood. $1,25(OH)_2$-vitamin D3 also stimulates RANKL in osteoblasts and further increases osteoclast formation. In addition, $1,25(OH)_2$-vitamin D3 enhances intestinal calcium uptake.

Increasing calcium concentration is sensitized by parafollicular C cells in thyroid glands which increase production and secretion of calcitonin. Calcitonin rapidly inhibits the bone-resorbing activity of mature osteoclasts, but also the formation of new osteoclasts and, thereby, the release of calcium from bone to blood will decrease. Calcitonin also acts on kidneys to increase calcium excretion which further decreases serum calcium. In addition, calcitonin decreases formation of $1,25(OH)_2$-vitamin D3 in the kidneys, which will result in less intestinal uptake of calcium and decreased bone resorption.

The relative importance for calcium homeostasis of bone resorption, calcium excretion in kidneys and calcium uptake in the intestine is not fully known, but the intestinal uptake is probably the most important process for the long-term control of serum calcium. Calcium excretion in the kidneys is most likely important for the minute to minute regulation of serum calcium, whereas osteoclasts may play their main role during calcium deficiency. The role of regulations to control calcium balance for skeletal remodeling in jaw bones and for pathologic bone loss in these bones is not known.

**Fig. 7.8** Modeling is a process when bone formation and bone resorption take place independently of each other. This is mainly seen in cortical bone when the shape and the size of bones are changed. One such example is during fracture healing. A. Femur fracture seen in a newborn child and the callus observed after 10 days (B). C. Due to modeling, the fracture is completely healed with no dislocation after 3 months. Courtesy of Dr. Piotr Michno, University of Umeå.

## Glucose and fat metabolism

Recent observations in genetically modified mice indicate that the bone-specific bone matrix protein osteocalcin does not have a role in bone formation and mineralization, as expected, but seems to be an osteoblast-derived endocrine factor involved in the control of glucose homeostasis and fat metabolism. Thus, osteocalcin-deficient mice have only a mild skeletal phenotype with increased bone mass (162), but are fat and diabetic, similar to patients with metabolic syndrome (163). This phenotype was found to be due to the fact that osteocalcin regulates insulin secretion in pancreatic β cells and the proliferation of adipocytes (163, 164). Osteocalcin undergoes a K vitamin-dependent modification of certain amino acids after the protein has been synthesized and some of the glutamic acid residues become γ-carboxylated. Interestingly, the effects on β cells and adipocytes were obtained with non-carboxylated osteocalcin, not with γ-carboxylated osteocalcin. Thus, the diabetic phenotype of the osteocalcin-deficient mice was due to lack of signaling by non-carboxylated osteocalcin in pancreatic β cells and adipocytes (Fig. 7.9). The novel finding that osteoblasts seem to secrete a hormonally active substance is not unique for osteocalcin since it has also been found that osteocytes secrete fibroblast growth factor-23 which controls phosphate excretion in kidneys (165).

The osteocalcin knockout mice lack all of their osteocalcin, but what regulates the decarboxylation of γ-carboxylated to hormonally active osteocalcin in mice, secreting normal amounts of osteocalcin is not known, but may be acid produced by osteoclasts (Fig. 7.9). Two groups have found that activation of the insulin receptor on osteoblasts increases the circulating amounts of γ-carboxylated osteocalcin and the formation of the osteoclast inhibitor OPG (166, 167). In addition, it was observed that the low pH created by osteoclasts is sufficient to decarboxylate the γ-carboxylated form of osteocalcin. It was, therefore, postulated that when insulin decreases, less OPG is produced causing formation of more active osteoclasts. The more osteoclasts, the more will

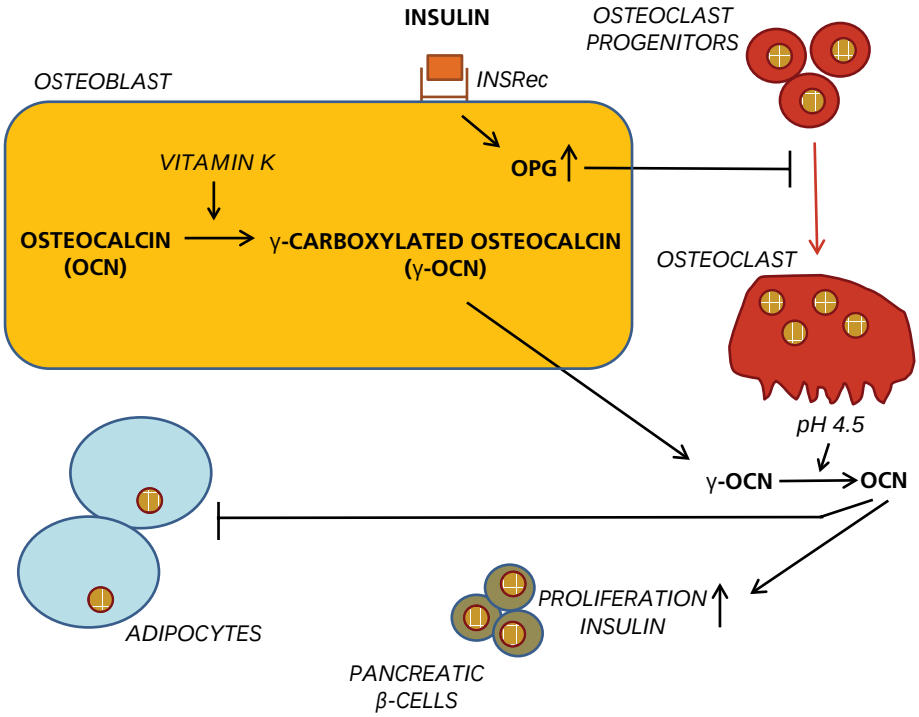



**Fig. 7.9** Osteocalcin (OCN) is one of the very few bone-specific proteins. It is produced in osteoblasts, which also transfer osteocalcin to γ-osteocalcin (γ-OCN) with the help of vitamin K. This protein has been shown to have a variety of hormonal-like activities on cells outside bone, including effects on adipocytes and pancreatic β cells. These effects require decarboxylation of γ-OCN to OCN, an effect that can be caused by acid secreted by active osteoclasts. Insulin can regulate the amount of osteoclasts by acting on insulin receptors (INSRec) on osteoblasts to increase the amount of the osteoclast inhibitor osteoprotegerin (OPG). Insulin deficiency will lead to less OPG and an increased number of osteoclasts converting γ-OCN to OCN, which then can act on β cells to increase insulin production.

osteocalcin be decarboxylated and activated, which then will increase insulin secretion in a feedback mechanism (168). Thus, osteoclasts seem to play a role also in glucose metabolism. These novel findings are made in genetically modified mice and need to be confirmed in humans.

## Role of osteoclasts in skeletal diseases

As discussed earlier, osteoclasts are important cells in physiologic remodeling of bone as well as during modeling processes. Osteoclasts also play important roles in pathologic processes associated with decreased bone mass, either locally or systemically, such as in osteoporosis, arthritis and skeletal metastasis of malignant tumors. The same cells also are involved in diseases with increased bone mass such as osteopetrosis.

### Osteoporosis

There are several forms of osteoporosis, but the most common form is primary osteoporosis in postmenopausal women due to decreasing amount of estrogen. Osteoporosis is a skeletal disorder characterized of decreased bone mass, leading to fragile bones, which are at increased risk for fracture. Half of all postmenopausal women will experience an osteoporotic fracture during their lifetime. Early during postmenopausal life, fractures in the forearm are most common. Later, vertebral fractures are more prevalent and, in elderly women, hip fractures are more frequent.

Osteoclast numbers are much higher in patients with postmenopausal osteoporosis because decreasing levels of estrogen result in enhanced expression of RANKL (89). Due to increased osteoclastogenesis, the number of BMUs is enhanced; i.e. there are more sites undergoing remodeling. Since estrogen deficiency also leads to decreased bone formation, osteoblasts will not be able to fill up all new resorption lacunae and bone mass decreases. The most common drugs used to treat patients with postmenopausal osteoporosis are bisphosphonates, which decrease the number of existing osteoclasts by inducing apoptosis, and denosumab, which decreases the formation of new osteoclasts (169). Thus, these drugs do not enhance bone mass primarily, but decrease further loss of bone.

Elderly men are also at risk of developing primary osteoporosis and the lifetime risk for experiencing an osteoporotic fracture is 25%. It is assumed that the decreasing amounts of testosterone increase osteoclastogenesis and enhance the number of BMUs, similar to observations in women with decreasing levels of estrogen. However, since estrogen is synthesized from testosterone in men, it might be that it is rather the decreasing levels of estrogen in testosterone-deficient osteoporotic men that are increasing the number of BMUs and the decrease of bone mass (170).

### Osteopetrosis

Osteoclasts play a crucial role in the genetic skeletal disease called osteopetrosis, previously also known as marble bone disease due to the excess of bone in the bone marrow compartments. The benign autosomal dominant form often has an adult onset, whereas the autosomal recessive form is more malignant and is usually diagnosed early after birth (171). The clinical hallmarks are enhanced bone mass, reduced bone marrow spaces and increased fracture risk. Due to the decrease of bone marrow, patients develop splenomegaly

and frequent infections. Narrowing of nerve channels often leads to deafness and blindness.

The enhanced bone mass is caused by decreased bone resorption, either due to decreased formation of osteoclasts or to defective capacity to resorb bone. Several mutations causing osteopetrosis have been revealed during recent years (65).

## Joint diseases

Patients with rheumatoid arthritis not only suffer from breakdown of articular cartilage, but also from local and systemic bone loss (172, 173). Formation of osteoclasts in the juxta-articular bone is due to increased RANKL in the synovium driven by the inflammatory process (174). Not only osteoblasts, but also T cells and synovial fibroblasts have been demonstrated to produce RANKL. and the relative importance in humans of these cell types for RANKL production is not known. In experimental arthritis in mice, RANKL production in synovial fibroblasts is crucial for joint osteoclast formation (39). Many inflammatory cytokines have been suggested to be important for RANKL and osteoclast formation in arthritis, but TNF-α seems to be of greatest interest since anti-TNF-α therapy is a successful treatment for many, but not for all, patients with rheumatoid arthritis (109, 175).

Epidemiologic studies have shown that periodontal disease and rheumatoid arthritis are associated (176). Patients with rheumatoid arthritis have more severe periodontitis than healthy controls (177, 178). Treatment of periodontal disease seems to improve rheumatoid arthritis, indicating a cause-and-effect relationship (179, 180). Anti-citrullinated protein/peptide antibodies (ACPAs) are present in serum from a majority of patients with rheumatoid arthritis, but not in other autoimmune diseases (181). Their presence can be detected years before the onset of clinically overt disease (182) and are associated with bone loss (183). Citrullination is a post-translation modification of proteins by which the amino acid arginine is converted to citrulline through the enzyme peptidyl-arginine deiminase (PAD). Interestingly, *Porphyromonas gingivalis* expresses PAD, which has generated the hypothesis that this bacterium is responsible for citrullination of certain proteins that ACPAs are directed towards, and maybe explaining the association between rheumatoid arthritis and periodontitis (184). The presence of ACPAs in patients with rheumatoid arthritis is associated with increased bone loss, even before onset of clinical disease (185). These observations have raised the question whether ACPAs can be involved in osteoclast formation. During osteoclastogenesis, the expression of the intermediate filament vimentin, as well as of PAD2, are increased, which leads to an increase of citrullinated vimentin during osteoclast formation (186). This is of particular interest since ACPAs against vimentin are highly specific for rheumatoid arthritis. When ACPAs against mutated vimentin, isolated from patients with rheumatoid arthritis, are added to RANKL-stimulated human osteoclast progenitors, ACPAs strongly potentiate the effect by RANKL on osteoclast formation (186). Injections of such ACPAs into mice resulted in an increased number of osteoclasts and decreased trabecular bone. These observations provide strong evidence for the binding of autoantibodies against citrullinated vimentin on osteoclast progenitors resulting in increased osteoclast formation and bone loss *in vitro* and *in vivo*. This is likely the explanation why patients with rheumatoid arthritis experience not only bone loss in their joints, but also systemic osteoporosis, even before clinical signs of onset of the disease. The effect seems to be mediated by TNF-α (186) and the chemokine IL-8/CXCL1 (187). Interestingly, ACPA positive patients with rheumatoid arthritis experience increased periodontitis as assessed by an enhanced number of sites with alveolar bone loss as compared to ACPA negative patients (188).

The fact that signs of bone loss in patients with rheumatoid arthritis can be observed before clinical signs of the disease indicates that osteoclast activity may not be a consequence of synovial inflammation but may rather precede the inflammatory process. These circumstances have generated the interesting hypothesis that bone resorption may play a role in the development of rheumatoid arthritis by priming the joint for susceptibility to chronic inflammation (189). This view does not imply that the inflammatory process does not have a role in driving osteoclast formation once the inflammatory process in the synovia is established.

## Malignancies

Osteoclasts are important cells in the spread of malignant tumor cells from the primary tumor to the skeleton in patients with metastatic bone disease. Many malignant tumors are capable of metastasizing to the skeleton, although their preference for this localization differs between different tumors. Interactions between tumor cells and osteoclasts (190), and the capacity of tumor cells to create an immunosuppressive niche in bone marrow (191), are the basis for a tumor-supporting microenvironment in the skeleton. In most cases, the metastases cause bone loss – so-called osteolytic metastases – but some tumors interact with osteoblasts and induce new bone formation – so-called sclerotic metastases. In the osteolytic metastases, the breakdown of bone is not caused by the tumor cells themselves, but is due to their capacity to induce RANKL formation and osteoclastogenesis. The osteoclast activity is not only important for the expansion of the tumors in the skeleton but also for tumor growth according to the seed-and-soil hypothesis (192), which was proposed by Paget more than 100 years ago (193). Based upon this view, it has been suggested that inhibition of osteoclast formation would not only decrease skeletal complications caused by the osteoclasts, but also tumor metastasis. High doses of bisphosphonates and denosumab are today used in the treatment of patients with advanced skeletal metastases, but the evidence that these compounds affect tumor cell expansion is limited.

Parathyroid hormone-related peptide (PTHrP) was discovered some 25 years ago independently by three groups

when investigating by which mechanisms lung cancer, renal cancer and breast cancer cells stimulated osteoclast formation (194). All three groups showed that the tumor cells released a peptide with large homology to PTH and, therefore, by acting on target cells via PTH receptors, was able to increase osteoclast formation (194). Initially, it was thought that PTHrP was a tumor-specific molecule but subsequent studies have shown that this peptide is produced by several non-malignant cell types and that PTHrP is involved in mammary gland development, calcium transport in the placenta, keratinocyte and hair follicle differentiation, bone and cartilage development and tooth eruption (194).

## Role of osteoclasts in the jaw bone

Similar to their role in the skeleton in general, osteoclasts are important for physiologic bone remodeling in the jaw bones and for pathologic intraosseous conditions affecting the mandibule and maxilla. Local activation of osteoclasts is a prerequisite also for tooth eruption and orthodontic tooth movement. During the last decade, a lot of interest has been focused on jaw bone osteoclasts due to the awareness of osteonecrosis of the jaw being a consequence of the use of osteoclast-inhibiting drugs for treatment of osteoporotic patients or skeletal metastases of malignant tumors.

### Remodeling of jaw bones

Most of the studies on bone remodeling in BMUs have been performed in long bones and vertebrae. It is, however, reasonable to assume that bones in the skull are also remodeled by similar mechanisms, although loading is not likely to be as important as in the long bones and spine. It has been suggested that jaw bones are remodeled more frequently than long bones and that this may be the reason that osteonecrosis is observed only in the jaws (195), but the evidence for this view is very limited. One important issue related to bone remodeling in jaw bones is if estrogen deficiency results in increased osteoclast formation and subsequent bone loss, similar to what happens in long bones and the spine. Several studies have tried to investigate if bone loss in periodontitis patients can be related to osteoporotic disease, and if osteoporotic treatment affects periodontal bone loss (196), but the information is inconclusive (197, 198) with both positive correlations (199–201) and absence of correlations (202–204) found.

There are no standardized techniques for analyzing bone mineral density in jaw bones similar to those used in long bones and vertebrae (where dual-energy X-ray absorptiometry (DEXA) is used), which limits the possibility of following bone loss in the jaws quantitatively over time. There is one study, using periapical radiographs, that reported that trabecular thickness, spacing and connectivity in jaw bones were decreased in osteoporotic patients compared to non-osteoporotic individuals, but the number of individuals studied was very few (205). Jonasson et al. have developed a semiquantitative technique using oral radiographs to assess trabecular patterns in the jaw bone. Using this method, it has been found in a prospective study that decreased trabecular pattern is a significant predictor of future osteoporotic fracture risk in a cohort of 731 women (206). In a subsequent study, it was reported that not only decreased trabecular pattern, but also cortical erosions, in the mandible is significantly associated with future fractures (207). It was estimated that dentists could identify 40–69% of women at risk for future fractures, depending on their age at examination. These studies suggest that osteoclasts in the jaw bones respond to estrogen deficiency in a similar way to those in long bones. It should be taken into account, however, that recent observations indicate that osteoclasts in different bones may vary and that there might be different physiologic and pathologic regulations of osteoclast differentiation and function in different parts of the skeleton (see 'Osteoclast heterogeneity' later in this chapter).

### Periodontitis

Alveolar bone loss is the hallmark of periodontal disease. The loss of bone is mainly due to excessive formation of osteoclasts in the periodontium (18). Support for this statement may be gained from observations showing that postmenopausal patients treated with bisphosphonates have a tendency to less tooth loss (208) and decreased loss of alveolar bone (209). Further support is the finding showing that treatment of rats with risedronate, an osteoclast inhibitory bisphosphonate, reduced periodontitis-induced alveolar bone loss (210). In addition, OPG-deficient mice develop spontaneous alveolar bone loss (211). Moreover, treatment of mice with OPG decreases osteoclast formation and alveolar bone loss caused by oral infection with *Porphyromonas gingivalis* (212).

Inflammatory processes within or in the vicinity of the skeleton are often associated with local bone loss. It is, therefore, reasonable to assume that signaling molecules produced in the inflammatory process cause excessive osteoclast formation and that these molecules might be similar in diseases such as marginal and apical periodontitis, rheumatoid arthritis or loosened tooth implants or joint prostheses. In that respect, cytokines stimulating RANKL production in osteoblasts such as IL-1, IL-6, IL-11, IL-17, leukemia inhibitory factor (LIF), oncostatin M (OSM) and TNF-α are likely candidates, all being found to be expressed in inflamed gingiva, periapical granuloma and synovium (18, 213, 214). Since other cytokines such as IL-4, IL-10, IL-12, IL-18, IFN-γ and IFN-β, which also are expressed by these tissues, can inhibit osteoclast formation, either by inhibiting RANKL formation or by acting directly on the osteoclast progenitors, it is the balance between RANKL stimulatory and inhibitory cytokines, and the expression of their receptors on osteoblasts and osteoclasts, that determines the amount of osteoclastogenesis and alveolar or periapical jaw bone being resorbed.

Although there are reasons to believe that inflammatory processes in different diseases cause osteoclast formation by

similar mechanisms, the possibility exists that osteoclastogenesis in marginal and apical periodontitis may differ from that in rheumatoid arthritis due to the close vicinity of bacterial plaque present on teeth or pulp cavities and bone surface. One important reason for making this distinction is the discovery of the Toll-like receptors (TLRs), which recognize ligands expressed by a large variety of bacteria present in the dental biofilm. Such receptors are present on most cells in the periodontium, including gingival fibroblasts, periodontal ligament cells, osteoblasts and osteoclasts. Activation of TLR4 by lipopolysaccharide from Gram negative bacteria, or of TLR2 by a lipoprotein from *Porphyromonas gingivalis*, can enhance osteoclast formation by stimulating RANKL production in osteoblasts both *in vitro* and *in vivo* (215, 216). Ligands for TLR2 or TLR4 can also act directly on osteoclast progenitors, either by inhibiting or stimulating their differentiation depending on if TLR4 is activated early or late during osteoclastogenesis (215, 217, 218). It is not known yet, however, to what extent the bacterial structures activating these receptors are present within the periodontium and the role they play in osteoclast activation during periodontal disease.

## Bone fracture

Healing of bone fractures requires initiation of several different pathways in a timely, organized manner including recapitulation of certain events in embryonic bone development (219). Osteoclast activity is important during several phases of fracture healing. One important initial step during bone healing is formation of a clot in the fracture site, followed by invasion of leukocytes and development of an inflammatory process. Formation of this inflammatory process is most crucial for the fracture to heal and includes expression of proinflammatory cytokines such as IL-1, TNF-α and IL-6. Similar to most inflammatory processes, several angiogenetic factors are released and an intricate network of new vessels is formed (220). Also similar to other inflammatory processes, osteoclasts are formed that resorb old, damaged bone fragments. In the central part of the clot, mesenchymal stem cells recruited from bone marrow are differentiated to chondrocytes and a cartilaginous callus is formed. In the periphery, mesenchymal stem cells in the periosteum differentiate to osteoblasts and intramembranous bone is formed as part of the callus. Eventually the cartilaginous callus is mineralized and, similar to endochondral ossification during bone development, osteoclasts will resorb the mineralized cartilage which then will be substituted by intramembranous bone. During the late phase of fracture healing, the callus consisting of woven bone will be resorbed by osteoclasts and substituted for by formation of lamellar bone.

These pathways in fracture healing are valid for healing of shaft fractures in cortical bone. Many fractures, however, including jaw bone fractures, also engage trabecular bone which heals through inter-trabecular bone formation with intramembranous bone without callus formation and devoid of osteoclast activity (221).

## Jaw tumors and cysts

Osteoclasts are important cells for the expansion of benign and malignant tumors in the jaws (Fig. 7.10A) and for the growth of developmental and inflammatory jaw cysts (Fig. 7.10B). In agreement with the view for other pathologic conditions associated with bone resorption, RANKL is considered to be crucial for osteoclastogenesis to occur in these conditions (222).

Similar to intraosseous tumors in the skeleton in general, odontogenic and non-odontogenic tumors in the jaws induce RANKL expression and stimulate local osteoclast formation. Thus, RANKL has been found in tumor cells of both epithelial and mesenchymal odontogenic tumors such as ameloblastoma (223), calcifying cystic odontogenic

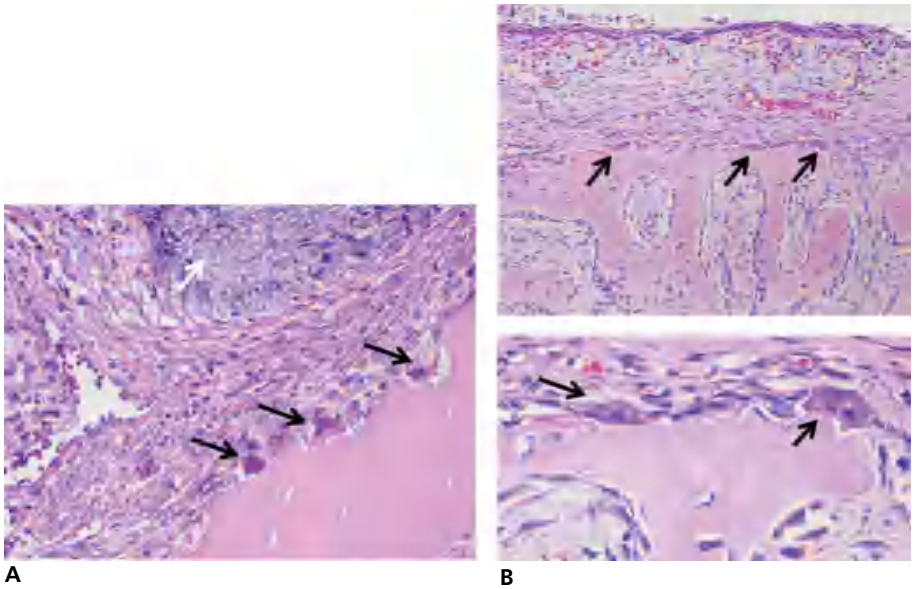



**Fig. 7.10** A. Malignant tumor cells (white arrow) in the vicinity of jaw bone tissue stimulate osteoclast formation (black arrows) and bone resorption. B. Cystic ameloblastoma in the jaw bone stimulates osteoclast formation (black arrows) and bone resorption. Courtesy of Professor Gunnar Warvinge, Malmö Dental School.

tumor, adenomatoid odontogenic tumor, calcifying epithelial odontogenic tumor, odontogenic myxoma and ameloblastic fibroma (224).

Several cell types in inflammatory radicular cysts, including cells in the fibrous capsule and the epithelial cells lining the cyst cavity, express RANKL (225–227). Cells expressing RANK and OPG have been observed in these lesions. RANKL expression has also been demonstrated in dentigerous cysts (228) and in odontogenic keratocysts (229). Interestingly, PTHrP expression has been found in the lining epithelium of jaw cysts, indicating that RANKL expression in these lesions may be induced by activation of PTH receptors (230). RANKL has also been detected in apical periodontitis (225, 226).

## Tooth eruption

For tooth eruption to occur, osteoclast formation and bone resorption are crucial events. Numerous numbers of osteoclasts are present in alveolar bone surrounding erupting teeth (Fig. 7.11). There are no other physiologic processes in the body where the number of osteoclasts is as large as in this situation. The critical role of osteoclasts for tooth eruption is demonstrated by observations showing that an absence of osteoclasts in osteopetrotic mice caused by lack of either M-CSF or RANKL is associated with lack of tooth eruption (21, 231). Inhibition of osteoclasts by treatment of mice with either a high dose of the bisphosphonate zoledronic acid, or by an antibody neutralizing mouse RANKL, results in irreversible block of tooth eruption in some teeth (incisors and first molars) or delayed eruption in other teeth (232, 233). On the other hand, overexpression of RANK in myeloid cells causes excessive osteoclast formation and early tooth eruption (234). Rescue of RANKL knockout mice with a RANKL transgene expressed in T and B lymphocytes results in partial rescue of the osteopetrotic phenotype, most evident in the diaphyseal regions exhibiting an ostoporotic appearance and abundance of osteoclasts (235). However, no osteoclasts were observed in the jaw bones and teeth did not erupt, indicating that RANKL needed for tooth eruption is expressed by other cells than those required for osteoclast formation in the diaphyseal bone. It has been elegantly demonstrated that the dental follicle (Fig. 7.11), a connective tissue sac that surrounds the unerupted teeth, is crucial for tooth eruption (236, 237), and that the coronal region of this follicle, in particular, regulates osteoclast formation (238). Interestingly, the expression of RANKL is greater in the coronal half of the follicle than in the basal part (239).

Studies in rats have shown that there is a burst of osteoclastogenesis at day 3 postnatally with many $TRAP^+$ mononuclear cells appearing in the dental follicle. At the same time, M-CSF, monocyte chemotactic protein 1 (MCP-1) and endothelial monocyte-activating polypeptide II (EMAP-II) are maximally expressed in the follicle and most likely involved in the recruitment of the osteoclast progenitor cells (240–242). At this point, osteoclastogenesis is induced by an increased RANKL/OPG ratio primarily caused by downregulation of OPG (243). There is another minor burst of osteoclastogenesis at day 10 postnatally in rats, associated with increased upregulation of RANKL.

These observations should be considered when using osteoclast inhibitors for the treatment of children with osteogenesis imperfecta, juvenile rheumatoid arthritis, juvenile idiopatic osteoporosis or malignant primary bone tumors.

## Tooth movement

Osteoclast formation and bone resorption on the pressure site is a fundamental event for tooth movement during orthodontic treatment. This is evidenced by case reports showing decreased response to orthodontic treatment in patients on bisphosphonates (244) and by the finding that these potent osteoclast inhibitors decrease experimentally induced tooth movement in mice (245). The osteoclasts are formed both on the periodontal ligament side and on the bone marrow side of cortical bone surrounding the roots, observations which give rise to the questions from where these osteoclast progenitors are derived and what causes their differentiation. It is likely the progenitors on the bone marrow side come from hematopoetic cells in the marrow, similar to osteoclasts on endocortical surfaces and on trabecular bone. This is, however, less likely for those on the

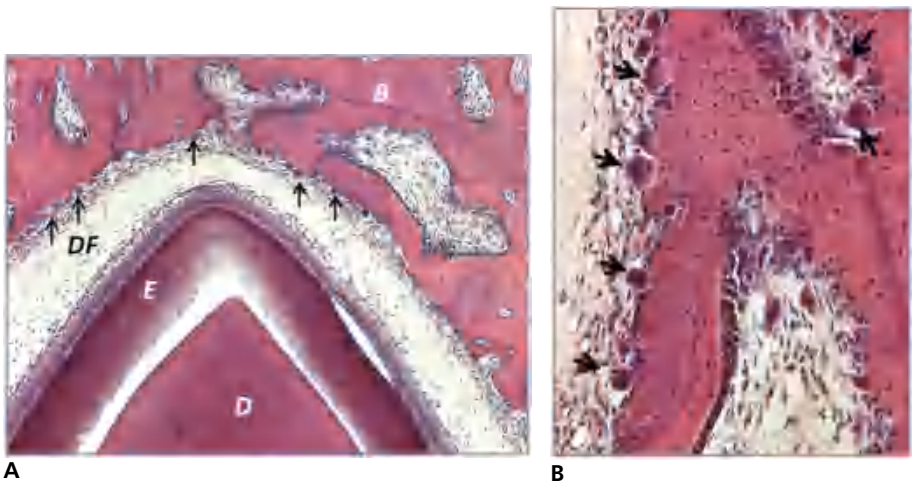



**Fig. 7.11** Tooth eruption requires extensive bone resorption around the crown. A. Numerous osteoclasts (black arrows) are seen on the surface of bone (B) close to the dental follicule (DF) surrounding the crown of a juvenile canine tooth bud. D, dentin; E, enamel. B. Higher magnification showing the abundance of osteoclasts (black arrows).

periodontal ligament side. These cells may either come from circulation or be present in the ligament, similar to those present in the periosteum of cortical bone.

The findings that the number of osteoclasts and rate of tooth movement are increased in mice deficient in OPG strongly indicate the importance of RANKL for osteoclast formation during orthodontic treatment (246). In line with this view, increased RANKL expression has been shown in the periodontal ligament during orthodontic treatment (247, 248). There are indications that both systemic and local factors are important for RANKL induction in the periodontal ligament. Thus, osteoclast formation and tooth movement is faster in mice during estrogen deficiency caused either by ovariectomy (249) or by deleting estrogen receptor alpha (250). Osteoclast formation and tooth movement are increased by induction of type 1 diabetes in rats (251). It is well known from studies in both patients and mice that inhibition of prostaglandin formation is associated with decreased tooth movement (252). Since prostaglandin $E_2$ is a potent stimulator of RANKL in periodontal ligament cells under mechanical loading (253, 254), these findings indicate that prostaglandin $E_2$ may be one candidate as stimulator of RANKL production during orthodontic treatment. Also, inhibition of 5-lipoxygenase metabolites results in decreased osteoclast formation induced by orthodontic treatment in mice (255). Another local factor that seems to be important is TNF-α. TNF-α is induced in periodontal ligament cells by orthodontic forces (247, 256) and lack of TNF receptor type 2 in mice results in decreased tooth movement (256). Since TNF-α is a potent stimulator of prostaglandin formation it might be that osteoclast formation during orthodontic tooth movement is induced by a cascade of TNF-α and $PGE_2$ formation. IL-1 seems to be important for orthodontic tooth movement since osteoclastogenesis and tooth movement are decreased by an IL-1 receptor antagonist, an effect associated with decreased levels of TNF-α in the periodontal tissue (257).

Since osteoclast progenitors need to be recruited during orthodontically induced osteoclastogenesis, it is not surprising that chemokine ligands such as CCL2 and CCL3 are upregulated during orthodontic treatment (258). The role of these cytokines is shown by findings demonstrating that mice deficient in CCL3, or the CCL3 receptor CCR1, or the CCL2 receptor CCR2, exhibit decreased osteoclast formation and tooth movement (259, 260). In contrast to these observations, CCR5 seem to be a negative regulator of osteoclast formation and orthodontic tooth movement (261).

Angiotensin II is the key molecule in the renin–angiotensin system acting on target cells through either angiotensin type I or type II receptors. Angiotensin II is formed from angiotensin I by the angiotensin-converting enzyme. This system is most well known for its control of blood pressure. Angiotensin II, however, has also been found to stimulate osteoclast formation *in vitro* and *in vivo* through stimulation of RANKL (262, 263). Patients treated with inhibitors of angiotensin-converting enzyme exhibit increased bone mineral density and reduced fracture risk (264). Interestingly, inhibition of angiotensin II receptors by losartan reduces osteoclast formation and orthodontic tooth movement (265).

Although several of these observations may explain osteoclast formation on the periodontal ligament side it is less obvious that such a mechanism explains osteoclast formation on the bone marrow side. One theoretical possibility might be that orthodontic forces cause microcracks in the cortical bone and that these cracks induce resorption on the bone marrow side, similar to that suggested for bone remodeling in BMUs (151). As important as induction of osteoclast formation is the cessation of osteoclast activity to avoid excessive bone resorption, but nothing is known about what regulates the decreased osteoclast formation and activity although decreased forces are likely to be involved.

## Osteoclasts and osteonecrosis of the jaws

Although osteonecrotic-like lesions ('phossy jaws') were observed during the 18th century in workers exposed to phosphorus vapors in match-making factories, the issue of osteonecrosis of the jaws (ONJ) started with a report in 2003 describing ONJ in patients treated with intravenous bisphosphonates for prevention of skeletal-related events in patients with malignant diseases (266). Since then, numerous reports describing ONJ in patients treated both intravenously and orally with different bisphosphonates have been published (267). ONJ is frequently found in patients with malignant diseases treated with high dose of bisphosphonates, but is also observed in patients with osteoporosis treated with lower doses (268). It is debated, however, how prevalent ONJ actually is, especially in osteoporotic patients. Although initially being regarded as an entity caused exclusively by bisphosphonates, it is now obvious that patients treated with denosumab are also at risk of developing ONJ (268, 269).

Recently, ONJ has been associated with angiogenesis inhibitors such as bevacizumab, a monoclonal antibody neutralizing vascular endothelial growth factor (VEGF), and sunitinib, an inhibitor of receptor-associated tyrosine kinase including VEGF-dependent tyrosine kinase (268, 270, 271). It is not clear if these drugs cause ONJ by inhibiting osteoclast formation or activity, but it is not unlikely given the role of VEGF as a regulator of osteoclast differentiation and bone remodeling (272, 273). ONJ has also been reported in patients treated with everolimus and temsirolimus, inhibitors of mammalian target of rapamycin (m-TOR), a serine/threonine kinase inhibitor (268, 274). These compounds also act as angiogenesis inhibitors, but have in addition been shown to decrease osteoclast formation *in vitro* and *in vivo* (275). Although not proven that patients treated with these compounds develop ONJ by decreasing jaw bone osteoclasts, it is not unlikely.

It is unknown why osteonecrosis seems to exclusively appear in jaw bones, although several hypotheses have been put forward. Thus it has been suggested that remodeling is faster in jaw bones than elsewhere in the skeleton; that ONJ is linked to infections or other pathologic processes only observed in the jaws; and that it is the only bone in the skeleton covered with a mucosal membrane. Arguing

against the opinion that osteonecrosis is a jaw bone-specific entity are reports showing that osteonecrosis can also be observed in the external auditory canal in some patients treated with bisphosphonates (276). Osteonecrosis is by far most commonly observed in the jaws but can also be observed in other bones in patients lacking bone-resorbing osteoclasts.

## Role of osteoclasts in tooth resorption

A striking difference between bone and tooth biology is the fact that teeth are not remodeled in the same way as bone, although their organic and inorganic constituents are almost identical. In apical and marginal periodontitis, osteoclasts appear on bone surfaces in the periodontium, but only very rarely on nearby root surfaces. Nonetheless, multinucleated, $TRAP^+$ cells can appear in the absence of periodontal inflammation in some patients on the external side of roots (external root resorption) or on the internal side in the pulp chamber (internal root resorption) (277, 278) (Fig. 7.12). These multinucleated cells are sometimes called odontoclasts or dentinoclasts, but it is not possible to distinguish them microscopically from osteoclasts (279, 280). Such cells are most frequent when roots of deciduous teeth are resorbed during eruption of permanent teeth (281, 282). They can be formed also on permanent teeth after use of excessive force, or during long-term orthodontic treatment (283). Idiopathic root resorption can be observed on single teeth, and is more rarely found in several teeth (284, 285). It is most frequent in the permanent dentition but can also be observed in primary teeth (286). Interestingly, familiar multiple idiopathic root resorption, with a late onset (40–60 years of age), has been reported, indicating that genetic factors may have a role (285).

If an external root resorption process reaches the dentinal tubules in a tooth with pulp infection, the resorption will be substantially enhanced until the infected pulp is reached.

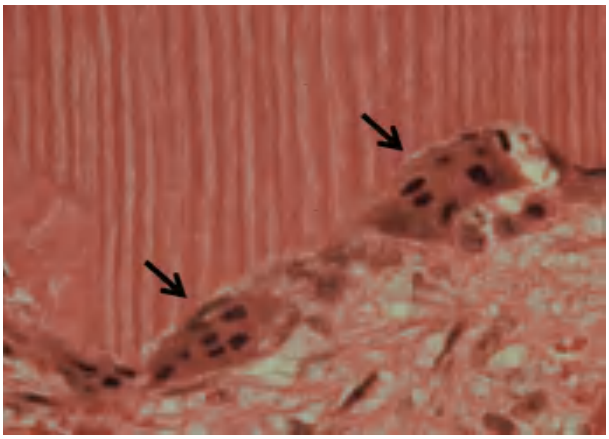


**Fig. 7.12** Idiopathic root resorption showing osteoclasts/dentinoclasts (black arrows) resorbing dentin in a human tooth.

Severe trauma to the teeth may lead to extensive root resorption, which is followed by new bone formation causing a bridge between alveolar bone and teeth, so-called dento-alveolar ankylosis.

An interesting issue is why teeth usually are protected from resorption and why osteoclasts developed in the periodontium only rarely attach to teeth, although being present sometimes in high numbers on nearby bone surfaces. If the driving event in physiologic bone remodeling is microfractures, it is not difficult to understand why such physiologic remodeling of teeth does not occur. It is also reasonable to assume that teeth do not need to be remodeled in a way similar to bone tissue. One possibility why teeth are protected from resorption might be that teeth, in contrast to bones, are covered by a cementum layer which may contain an inhibitor of osteoclastogenesis – a view that may gain some support by the finding that teeth, similar to cartilage, contain anti-invasive protease inhibitors (287). It is also possible that cementoblasts may be different to osteoblasts and not sensitive to endocrine or paracrine regulators of RANKL and OPG.

Recently, it was shown that Wnt signaling may have a role in root resorption (288). It was observed that mice lacking Wntless (Wls) in osteocalcin-expressing cells (i.e. osteoblasts but also presumably odontoblasts and cementoblasts) exhibit extensive, spontaneous external root resorption. Wls is a chaperone protein that is crucial for transferring Wnt proteins from the Golgi apparatus to the cell surface, which means that Wls-deficient mice lack signaling from all Wnt proteins. The excessive root resorption observed in the Wls knockout mice was associated with enhanced formation of osteoclasts on root surfaces, but also on bone surfaces in the periodontium. It was suggested that enhanced osteoclastogenesis might be due to an increased RANKL/OPG ratio, which is reasonable given the fact that canonical Wnt signaling is associated with increased OPG expression (112, 113), and that increased RANKL has been found in the periodontium during orthodontic treatment (289). The question still remains, however, why excessive osteoclast formation in the periodontium in general is not necessarily associated with root resorption. In the Wls-deficient mice it was noted that the cementum was thinner than in wild type controls, which might be an explanation why osteoclasts frequently resorbed the roots.

## Osteoclast heterogeneity

Although osteoclasts in textbooks and review articles are described as one and the same type of cell in different parts of the body, there is evidence indicating that osteoclasts in different skeletal sites may exhibit a heterogeneity not so often recognized. Thus, skull bone and long bone osteoclasts express different relative amounts of MMPs and cysteine proteinases. Osteoclasts in trabecular bone are more active than in cortical bone. Osteoclasts in different sites respond differently to certain hormones and drugs. Some gene

deletions affect long bone osteoclasts more than skull bone osteoclasts. As discussed elsewhere in more detail (290, 291), there are reasons to take this heterogeneity into account when discussing osteoclast biology and considering the role of hormones, cytokines and other signaling molecules able to affect these cells. Thus, there is a risk for being misled when interpreting results obtained by studying osteoclasts in general when discussing osteoclastogenesis and bone resorption in jaw bones.

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## Essentials

- Osteoclastogenesis
  - M-CSF and RANKL
  - Intracellular signaling
  - Co-stimulatory immunoglobulin-like receptors
- Osteoclastic resorption
- Regulation of RANKL-induced differentiation of mononuclear osteoclast progenitors
- Osteoclast progenitor origin
- Physiological role of osteoclasts
  - Bone remodeling
  - Calcium balance
  - Glucose and fat metabolism
- Role of osteoclasts in the jaw
  - Remodeling of jaw bones
  - Tooth eruption
  - Tooth movement as a response to traumatic dental injuries
- Role of osteoclasts in tooth resorption

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# 8

# Physical and Chemical Methods to Optimize Pulpal and Periodontal Healing After Traumatic Injuries

M. Trope

## Introduction

When a tooth suffers a traumatic injury, a variable degree of damage will occur to the periodontal structures as well as the neurovascular bundle at the root apex (1). Under all circumstances the first reaction to a trauma will be an inflammatory process. If this process is minimal, healing of the periodontal ligament (PDL) will take place without resorption (*normal healing*), which is considered to be *favorable healing*.

If the root surface damage and subsequent inflammation are of sufficient intensity, root resorption will result through osteoclastic activity (2) (Fig. 8.1). Under most circumstances, this resorption will be transient with spontaneous healing resulting in *surface resorption with cemental repair* (*repair-related resorption*); this healing is also considered favorable.

If a continual stimulus is present (e.g. if the pulp canal becomes infected or sulcular infection corresponds to an affected root surface), no healing takes place and the active inflammatory resorption will continue until the entire root is lost. This will be referred to as *infection-related resorption* (see Chapter 2) (Fig. 8.2).

The type of healing that occurs after the initial inflammation subsides is also (apart from the pulp condition) dependent on the surface area of root that is damaged. If the surface area of damaged root is small, the surviving cementoblasts that surround the area will produce new cementum and PDL to cover the denuded area and will result in surface resorption with cemental repair. This type of healing is favorable or cemental healing (see Chapter 2) (2) (Fig. 8.3).

If, on the other hand, the damage to the root covers a large surface area, bone-producing cells will attach directly onto the root before the area can be healed by new cementum. When the bone attaches directly to the root it undergoes physiologic turnover comprising resorption and apposition. The osteoclasts have little difficulty resorbing dentin; however in the apposition stage, bone (rather than dentin) is formed and thus the root is slowly replaced by bone. This osseous replacement is obviously an *unfavorable healing* outcome and has been termed *osseous replacement resorption* in Chapter 2 (2) (Fig. 8.4).

Therefore, in order to optimize healing after a traumatic injury, it is essential to ensure that the surface area of root damage is as small as possible. It is not possible to control the physical damage to the root surface due to the trauma itself. However, if the initial inflammatory response after the injury can be minimized, additional 'secondary' root damage will be limited, thus promoting cemental (favorable) healing. Also, it is essential to ensure that the pulp canal does not become infected and act as a continuous stimulus for inflammatory root resorption. Thus the treatment objective after a traumatic injury is to limit active inflammation immediately after the injury and to prevent or treat pulpal infection (2, 3). In addition, attempts have been made to stimulate cemental healing so as to aid in favorable healing (4, 5). In this chapter a series of experiments will be discussed demonstrating the effect of physical and chemical methods to optimize healing using the division of healing types mentioned earlier. In a few studies an alternative classification has been used, dividing healing into two groups:

(1) Favorable healing:
   - normal healing (without root resorption);
   - suface resorption with cemental repair (repair-related resorption).

(2) Unfavorable healing:
   - osseous replacement resorption (ankylosis);
   - infection-related resorption.

When experimental results are evaluated, the proportion of the root surface showing favorable healing will naturally be of major interest. Osseous replacement resorption can be an acceptable healing in adults, especially if the extent of the

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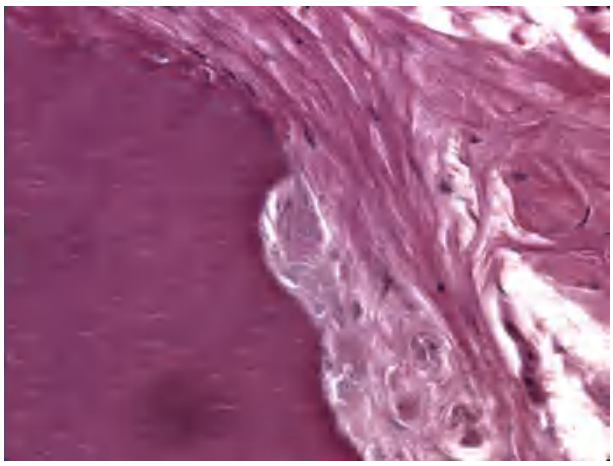


**Fig. 8.1** Multinucleated osteoclasts are formed in reaction to the damage to the root caused by the traumatic injury and are now resorbing the root.

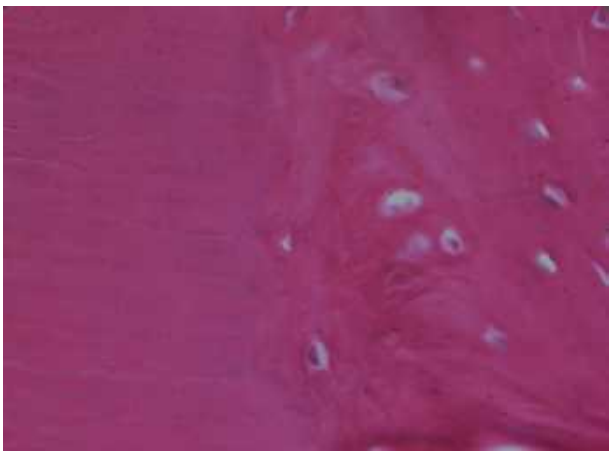


**Fig. 8.4** Unfavorable healing with osseous replacement. Bone has attached directly to the root surface. In time the osteoclasts will resorb the dentin and the osteoblasts will lay down new bone. In this way the root dentin will be replaced by bone.

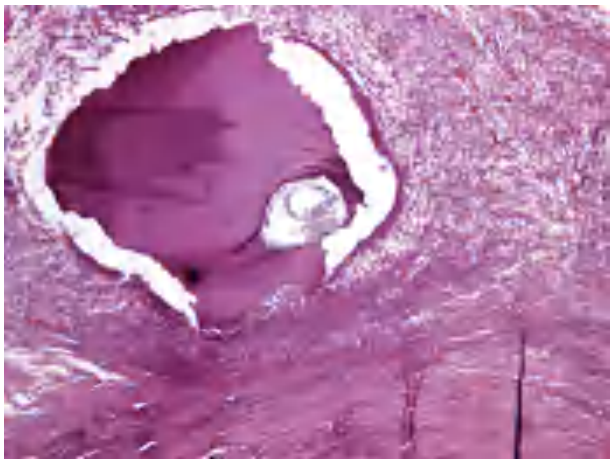


**Fig. 8.2** Infection-related inflammatory root resorption that has destroyed almost the entire root.

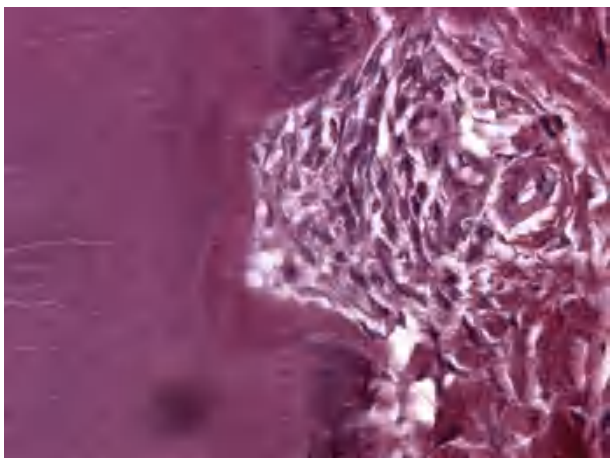


**Fig. 8.3** Favorable healing. The inflammatory root resorption has stopped and the denuded root surface is covered with cementoblasts and new cementum.

ankylosis is limited. Inflammatory root resorption cannot be regarded as a healing process but a pathologic infection-related condition which will transfer either into favorable or unfavorable healing when the infection is treated.

## Treatment strategies

### Accident site

The best healing results are obtained if the traumatized tooth is returned to its original position as soon as possible. The sooner this is achieved, the better the prognosis (6–8). Most luxated teeth require repositioning. This should be performed as gently as possible so as to limit additional trauma.

Avulsed teeth pose a unique challenge. The damage to the cemental layer/periodontal ligament when a tooth is avulsed is usually not extensive since in most cases the PDL tears, leaving viable cells on the root surface that are capable of healing (Fig. 8.5).

In fact, the damaged root surface usually covers a small surface area with a good potential for favorable healing. The potential danger for unfavorable healing in an avulsed tooth is if the PDL cells on the root surface dry out and die before replantation (2, 6, 8, 9). The dead cells will then act as potent inflammatory stimulators on replantation. Therefore dry time must be kept to a minimum.

Strategies at the accident site to limit the inflammatory potential of the root PDL are:

- **Physical:** Gently wash the root surface of gross debris with tap water (Fig. 8.6) or sterile saline if the tooth is to be replanted immediately (2, 10).
- **Chemical:** If the tooth cannot be replanted immediately it should be placed in an appropriate storage medium until it can be replanted. Dry time must be avoided.

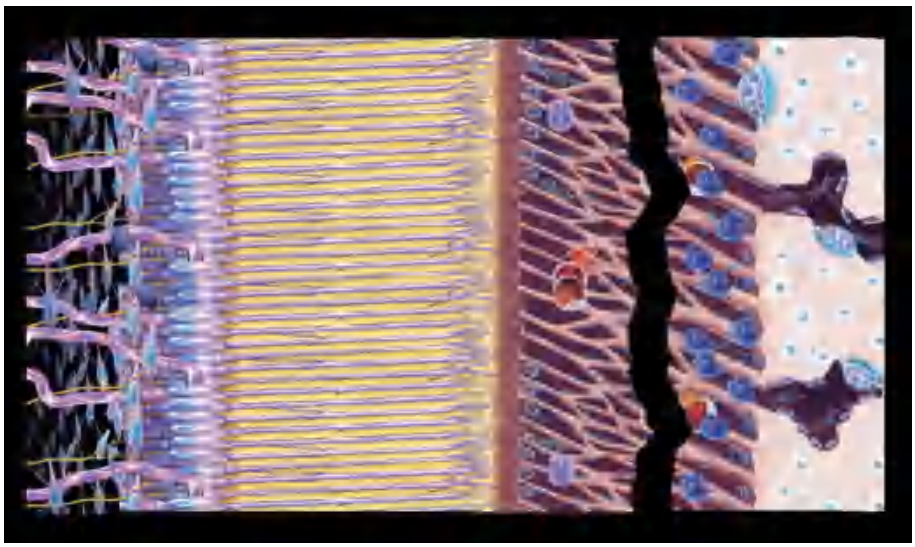


**Fig. 8.5** After an avulsion injury most of the external root surface is covered with periodontal ligament fibers since the avulsion occurs due to tearing of the periodontal ligament.

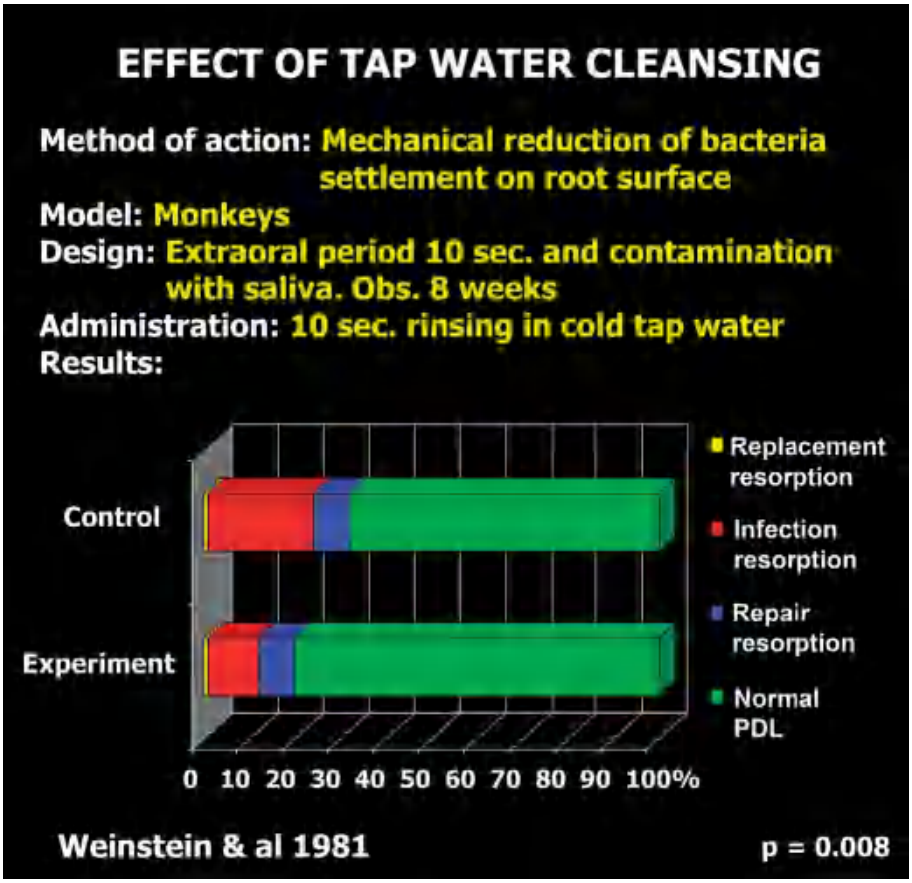



**Fig. 8.6** Effect of saline cleansing on PDL healing after replantation in monkeys. Reproduced from (10) with permission.

Acceptable media include physiologic saline, milk or the patient's saliva (in cheek, under tongue or in a cup with patient's spit). These media can keep the PDL cells viable for healing for about 2 hours (6, 11, 12). Hanks balanced salt solution (HBSS), if available at the accident site, may extend the extraoral period to up to 8 hours (13, 14). In special circumstances, such as possible neurologic damage or a life-threatening accident, storage media (e.g. Viaspan®) can be used to retain a PDL viable for healing for longer periods (see later) (14). Since in the majority of cases accidents occur near the home or school, milk appears to be the most appropriate and popular storage medium.

## Emergency dental visit

At this visit the focus is on minimizing the external inflammatory root resorption due to the traumatic injury and promoting cemental regeneration. Pulp space infection is a slower process and is the focus of later visits.

## General strategies

### Physical methods

If the tooth is in the socket (luxation injury or replanted at the accident site), its positioning should be checked. If repositioning is required it should be performed as atraumatically as possible.

In an intruded tooth, the practitioner may chose to allow for spontaneous re-eruption depending on the stage of root development (see Chapter 9) (15).

If the tooth has been avulsed, the root should be soaked in physiologic saline or HBSS while the history and examination are performed so as to flush debris and dead cells off the root surface (14, 16).

The tooth is retained in position using a physiologic splint. A physiologic rather than rigid splint has been shown to favor cemental healing (15, 17). The movement of the tooth is thought to break up ankylotic areas on the root surface which are then replaced by cementum and periodontal ligament.

### Chemical methods

There has been extensive research done on how to chemically prepare the root to promote favorable healing, or, in situations where favorable healing is not possible, to slow down osseous replacement of the root. The strategies employed are dependent upon the extent of the injury to the root surface and/or the potential for repair at the time of replantation and/or repositioning. Obviously a tooth that has undergone a luxation injury without avulsion cannot have the root surface treated. Therefore most of the strategies described will be relevant only for the avulsed tooth.

After an avulsion injury, deleterious effects occur to the PDL very quickly. Recent clinical research has shown that after approximately 20 minutes dry time some degree of unfavorable healing is expected (7, 8). However, most of the research into the beneficial effects of anti-inflammatory or antiresorptive procedures has been performed on roots that have been dry for 60 minutes or more. It is assumed (although not proven) that medicaments that are beneficial after 60 minutes dry time will be beneficial after shorter dry periods as well.

#### *Mature tooth – extraoral dry time <20 minutes*

After the tooth has been soaked in physiologic saline or HBSS, while the history and examination was taking place, these teeth should be replanted and splinted as soon as possible.

#### *Mature tooth – extraoral dry time 20–60 minutes*

A number of medicaments have been studied in order to minimize the initial inflammation after replantation by soaking the root surface.

**Tetracycline**

Tetracyclines are antibacterial and have proven antiresorptive properties. It has been theorized that by soaking the tooth in tetracycline antibiotic the reduction in bacteria on the root surface would decrease the potential for inflammation; in addition, the antiresorptive actions would limit the area of root surface damaged thus promoting favorable healing by the process already described (Fig. 8.7). Tetracyclines exert their antimicrobial activity by inhibiting protein synthesis, and are broad-spectrum antibiotics against anaerobes

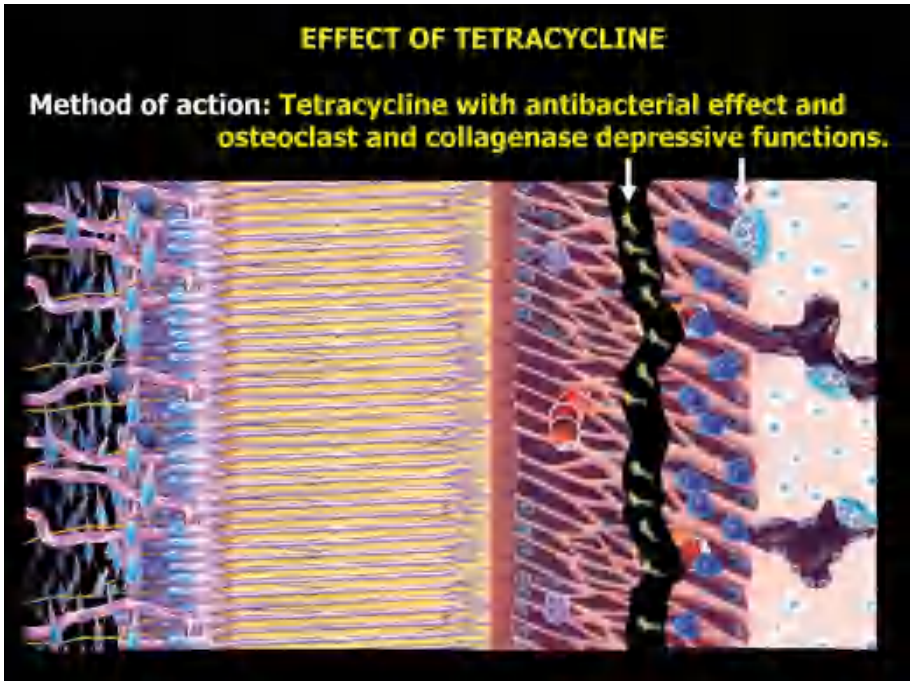



**Fig. 8.7** Possible ways that tetracycline may optimize PDL healing.

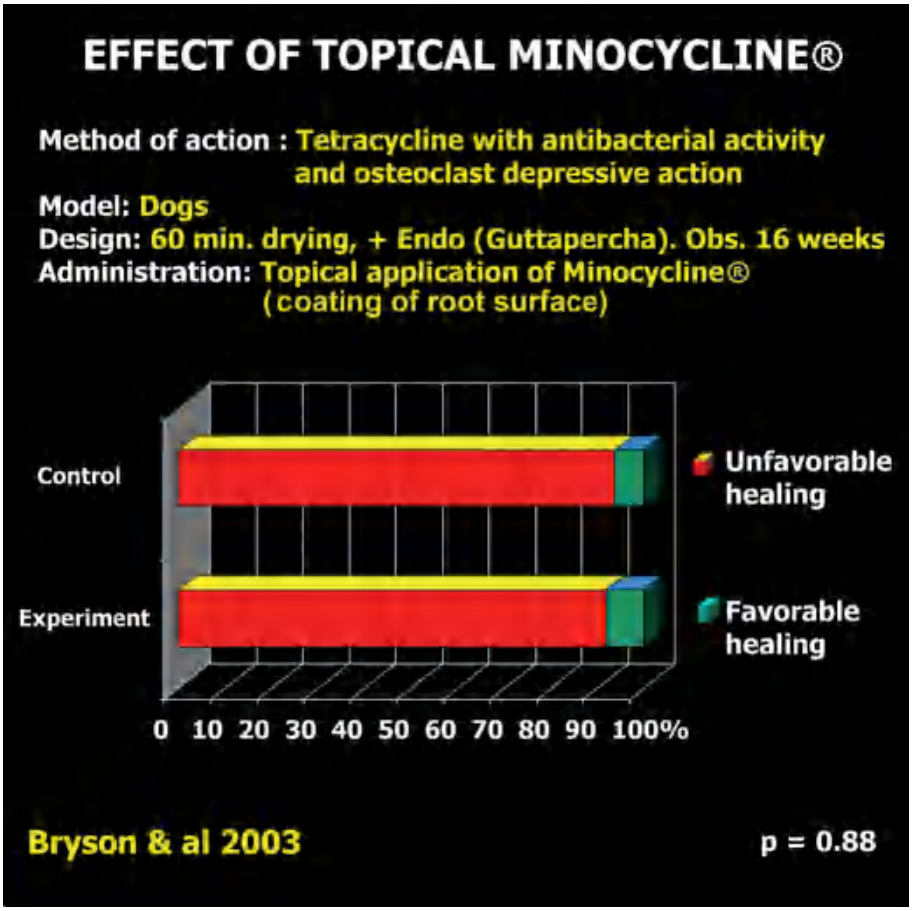



**Fig. 8.8** Effect of minocycline® on PDL healing after replantation in dogs. Reproduced from (22) with permission.

and facultative organisms. They provide bactericidal action at the high concentrations achieved with local delivery of the drug (18).

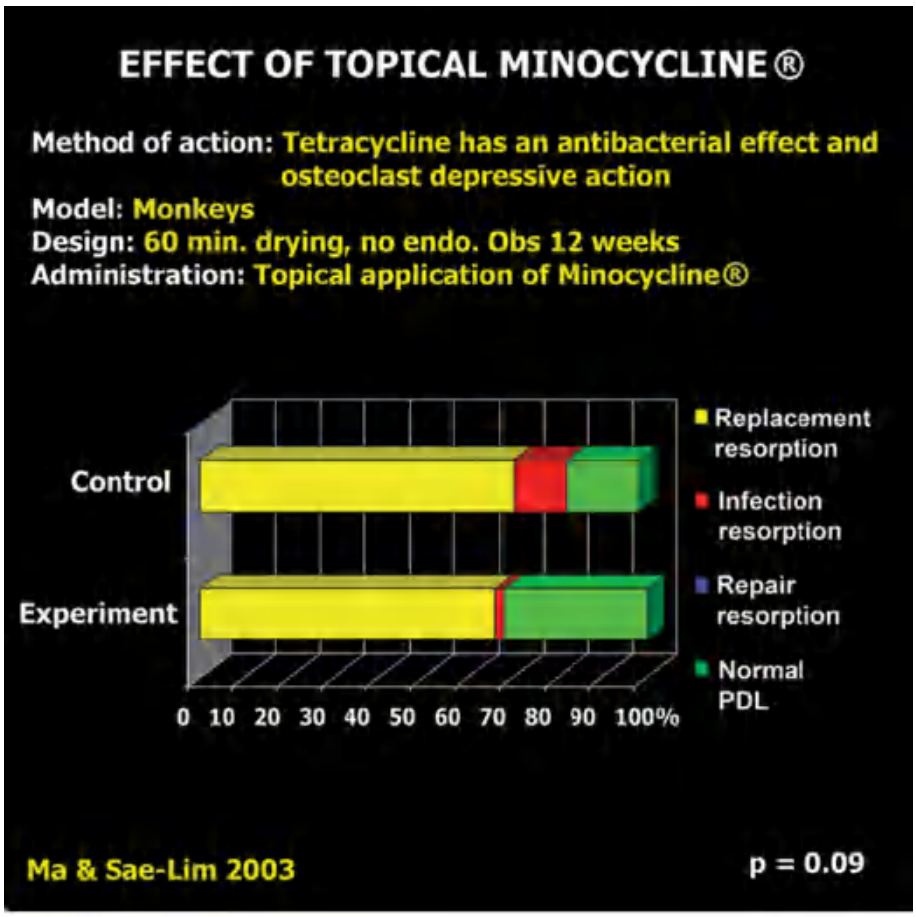
Tetracyclines are substantive due to their hard tissue binding capability and also have an innate antiresorptive action. They have the ability to bind to the tooth surface and then be slowly released in an active form (19). Tetracyclines also promote fibroblast and connective tissue attachment, enhancing regeneration of periodontal attachment lost to pathologic processes (20). Tetracyclines inhibit collagenase activity and osteoclast function (21), which could be beneficial to a replanted tooth. Their effects on the osteoclasts include diminished acid production, decreased ruffled border area and decreased adhesive properties, all of which inhibit bone resorption. Despite these effects, Bryson et al. were not able to show a beneficial effect of soaking the tooth in minocycline for 60 minutes in endodontically treated dogs' teeth dried for 60 minutes (22) (Fig. 8.8).

Ma and Sae-Lim, in monkey teeth dried for 60 minutes, showed an increase in normal periodontal healing after soaking the roots in minocycline (23) (Fig. 8.9A). However, there was no decrease in osseous replacement in the experimental teeth. Thus, favorable healing occurred by decreasing the inflammatory root resorption. Since the teeth were not endodontically treated, as in the Bryson study, it can be assumed that the effect was due to an inhibition of bacteria from the root canal space. In the clinical situation these teeth would be root treated, so it is assumed that the effect would be similar to that in the Bryson study, where root treatment was performed on the teeth. Here, too, little effect of enhancing favorable healing was seen.

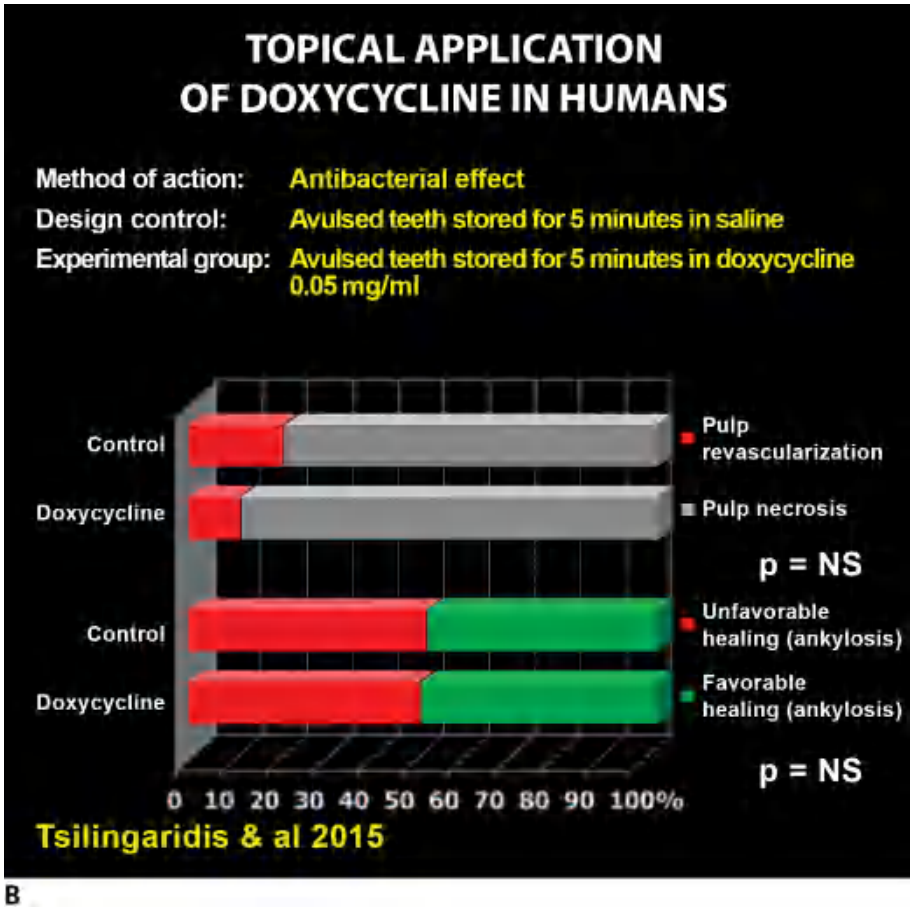
Finally, in a clinical study in humans, topical storage of avulsed teeth for 5 minutes showed no difference in pulp or periodontal healing (72) (Fig. 8.9B). *Thus, these studies were not able to validate the use of this method after an avulsion injury*. Further research needs to be performed in roots where the dry time is less than 60 minutes.

### Corticosteroids

Glucocorticosteroids have been widely used to reduce the deleterious effects of inflammatory responses. They have been reported to reduce and block the expression of macrophage activation, thus affecting interleukin 1 (IL-1), tumor necrosis factor α (TNFα), IL-6 and prostaglandin production (24). These effects have been attributed to inhibition at the transcriptional level of processes leading to the complex events of macrophage activation (24). Long-term systemic administration of dexamethasone is known to reduce skeletal bone mass, whilst its local administration has been shown to reduce osteoclastic bone resorption. Several mechanisms have been reported for this effect, including reduction of the number of osteoclasts by direct receptor-mediated and specific toxicity to these cells (25, 26), and enhancement of calcitonin receptors on these cells, making them more responsive to the existing systemic concentration of hormone whose main function is to reduce bone resorptive




A




B

**Fig. 8.9** A. Effect of minocycline® on PDL healing after replantation in monkeys. Reproduced from (23) with permission. B. Effect of topical application of doxycycline upon pulp and PDL healing in humans after replantation. Reproduced from (72) with permission.

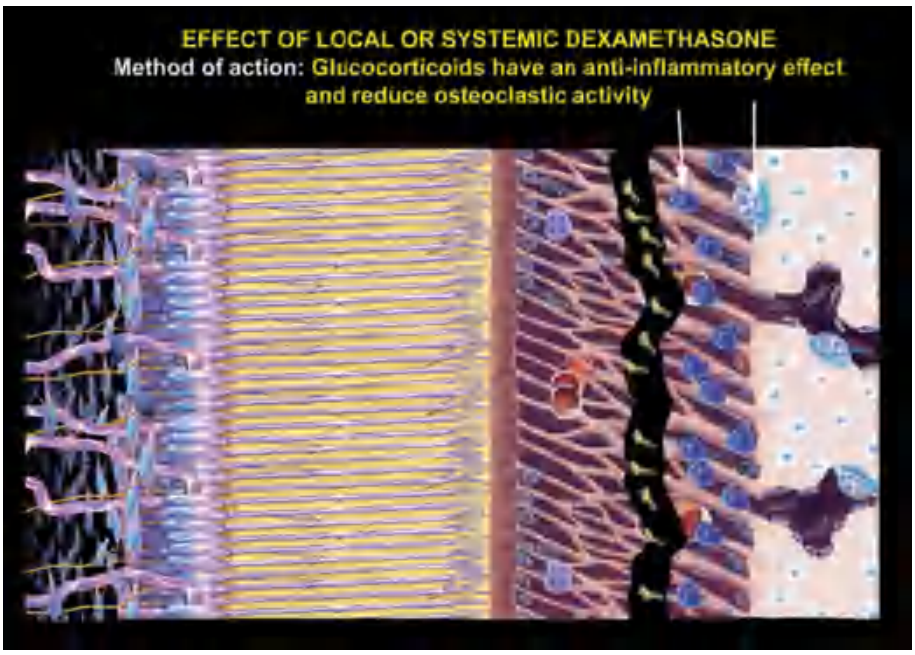



**Fig. 8.10** Possible effect of local or systemic dexamethasone.

activity (Fig. 8.10). This upregulation is also steroid receptor mediated and is effective at the transcriptional level (27, 28).

Sae-Lim et al. found that in dogs the topical (and not systemic) application of corticosteroids had a beneficial effect on healing (29). In this experiment the teeth had been stored in a wet medium for 48 hours (Fig. 8.11). The beneficial effect of the corticosteroid was presumably due to its ability to locally shut down the inflammatory response, thus limiting the damage to the root surface. The reason that dexamethasone was ineffective systemically is subject to speculation. It may not have been adequately absorbed in time or may not have reached a concentration in the periodontal space that was adequate to shut down the inflammation.

### Alendronate

Alendronate (ALN) is currently being used to inhibit pathologic osteoclast-mediated hard tissue resorption in disease states such as osteoporosis, Paget's disease and osteolytic malignancies of bone (57). The affinity of ALN for calcium phosphate and its tenacious binding to hydroxyapatite leads to a rapid uptake into the skeleton (30). Once incorporated into the skeleton, the terminal half-life of ALN activity has been determined to be as high as 1000 days in dogs (31). The mechanism of osteoclast inhibition has been attributed to: (i) a decrease in osteoclast activity with minimal effects on recruitment (58); (ii) the interference of receptors on the osteoclasts for specific bone matrix proteins (32), promoting the production of an osteoclast inhibitor by osteoblasts that reduce the lifespan and/or the number of differentiated osteoclasts (59); and (iii) the obstruction of resorption by interfering with the ruffled border of the osteoclast (57) (Fig. 8.12). Most importantly, the inhibitory effect of ALN on osteoclasts is widely recognized and is currently used therapeutically in the osteoclast-mediated disease states previously mentioned (57). Dog roots soaked in HBSS followed by ALN had statistically significant improved healing than roots soaked in HBSS without ALN (30% vs. 11%) (33). This was true for roots dried for 40 or 60 minutes (Fig. 8.13). Soaking in ALN also resulted in significantly less loss in root mass due to resorption compared to those teeth soaked in HBSS without ALN.

### Calcitonin

Calcitonin is a hormone secreted by the parafollicular cells of the thyroid gland and is known to cause contraction of osteoclasts and inhibit their activity (see Chapter 7) (Fig. 8.14). Topical use of calcitonin (placed in the root canal) has been found to be effective in controlling inflammation related to root resorption (70) (Fig. 8.15).

### Emdogain® (enamel matrix protein)

Emdogain® has been shown to promote PDL proliferation. Enamel matrix molecules induce acellular cementum formation on traumatized root surfaces. The progenitor cell pool for repopulating the root surface is thought to be the remaining vital cells on the root surface, undifferentiated cells from the marrow and progenitor cells from the socket. Trope et al. have shown that the socket environment plays an important role in the healing of replanted teeth (34), suggesting that the PDL cells on the socket wall may be important progenitor cells for Emdogain® (Fig. 8.16).

Once the PDL has been destroyed due to avulsion, the cell surface can be repopulated by cementum-derived cells or with osteogenic cells from the marrow. It is best to repopulate the surface with PDL or cementum-derived cells. Guided tissue regeneration techniques are designed to facilitate cell-specific repopulation. Barrier techniques can inhibit competition between marrow-derived cells and cementum-derived cells to favor the latter. Thus, a lower number of seeding cells would be required to repopulate the root surface. The prognosis may, therefore, be improved for teeth with longer extraoral times.

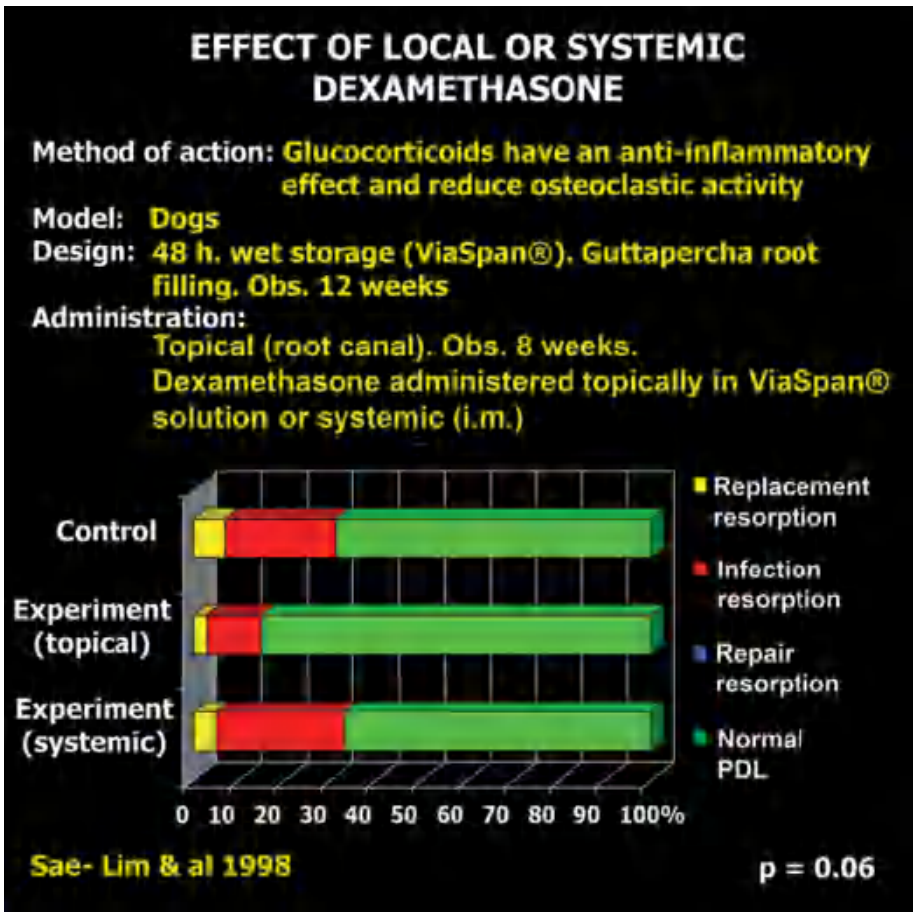



**Fig. 8.11** Effect of topical and systemic dexamethosone on PDL healing after replantation. Reproduced from (29) with permission.

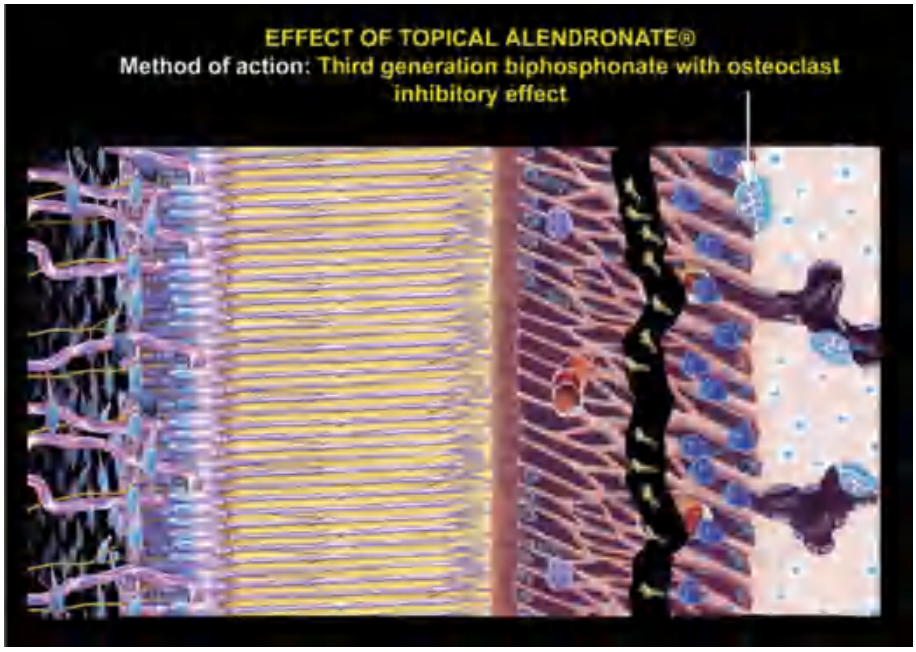



**Fig. 8.12** Possible ways that a biphosphonate may optimize PDL healing.

Emdogain® was shown in a study by Iqbal and Bamaas to almost double the favorable healing in dogs' teeth dried for 60 minutes (5) (Fig. 8.17). However, Araujo et al. failed to show a benefit in healing after soaking roots in Emdogain® (35) (Fig. 8.18).

There have been numerous case reports in which Emdogain® was used after extended dry times. It is obvious from these reports that while the use of Emdogain® slowed down the osseous replacement it was not able to reconstitute the PDL completely (35). Like the other medicaments

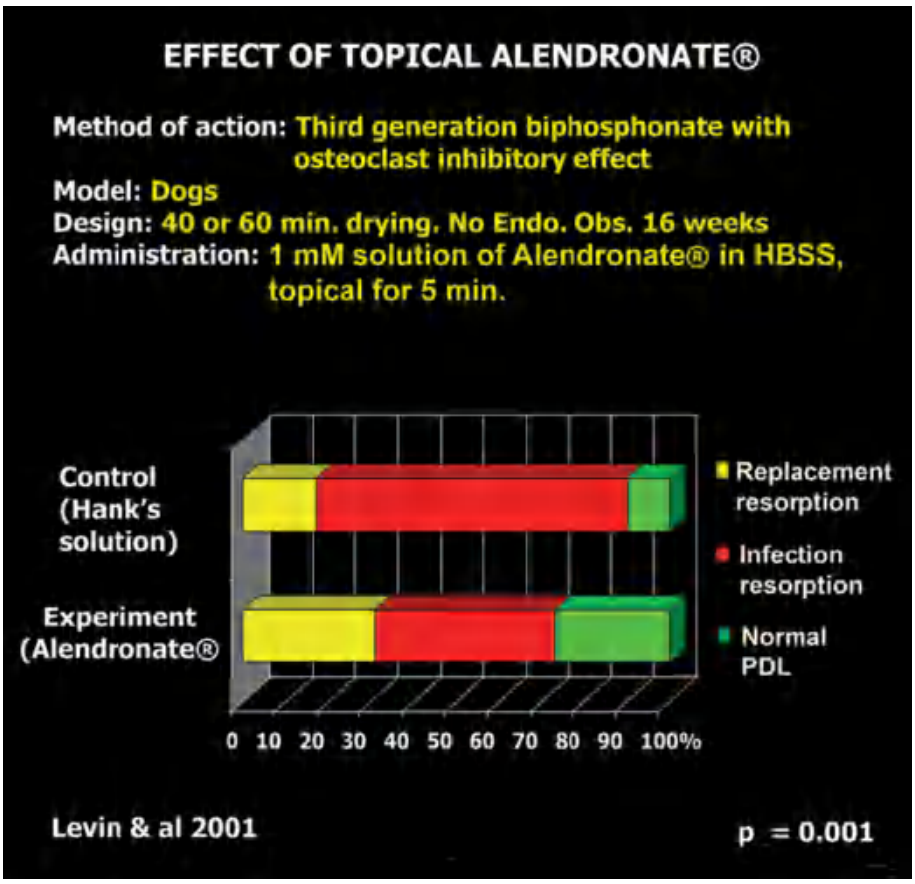



**Fig. 8.13** Effect of topical biphosphonate on PDL healing after replantation in dogs. Reproduced from (33) with permission.

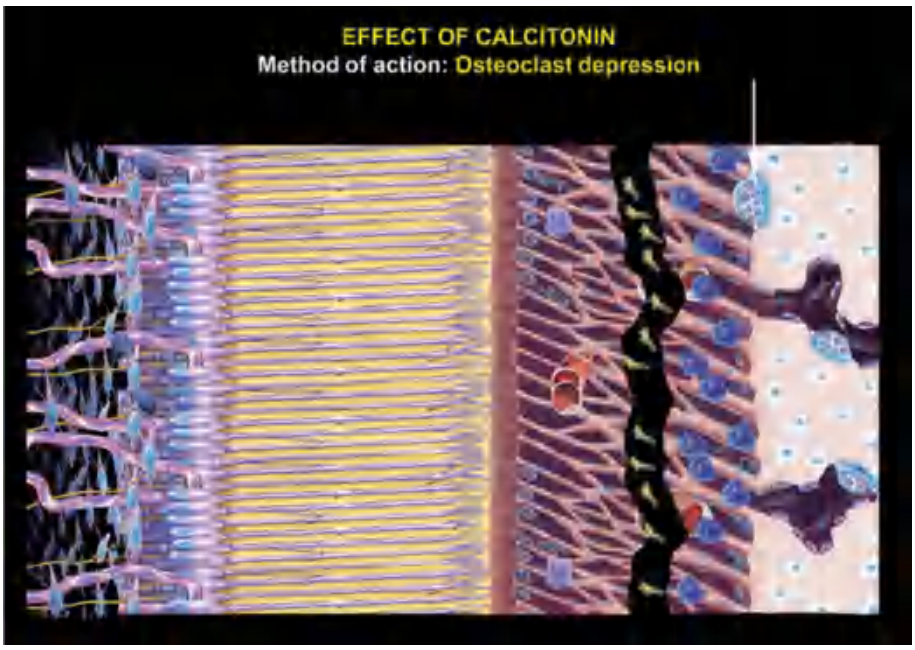



**Fig. 8.14** Possible ways calcitonin may optimize PDL healing.

discussed in this chapter, most research has been performed on roots that have been left dry for 60 minutes. These medicaments should be tested at shorter dry times where there is more likelihood of progenitor cells remaining on the root surface.

**Specialized tissue storage medium**

ViaSpan® is a storage medium used when transporting livers for transplantation (63) and this medium has been found also to be an excellent storage media for teeth (62) (Fig. 8.19). In a study on dog teeth extraction with

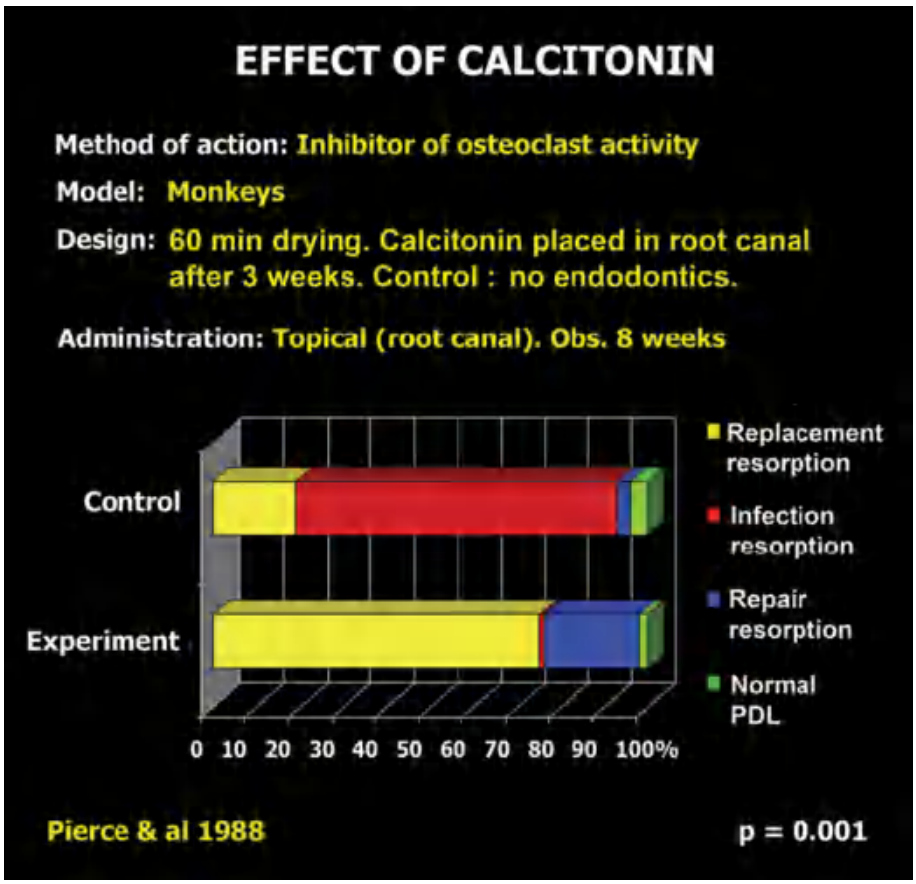



**Fig. 8.15** Effect of topically placed calcitonin (in the root canal) upon PDL healing. Reproduced from (70) with permission.

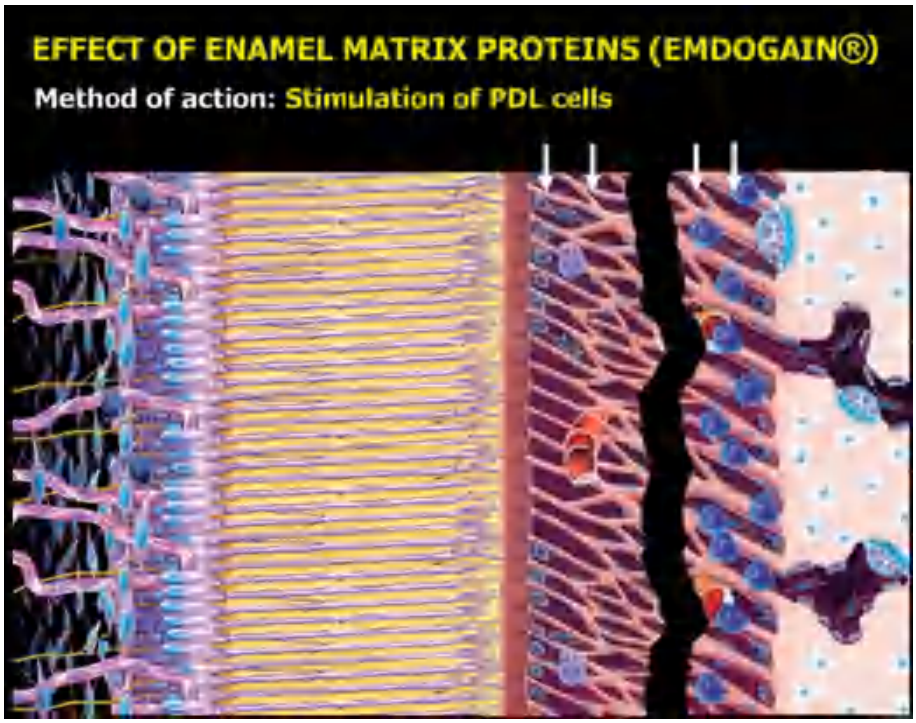



**Fig. 8.16** Possible ways that enamel matrix proteins may optimize PDL healing.

varying drying periods, it was found that teeth dried for 45–60 minutes showed an improvement in periodontal healing after 30 minutes soaking in ViaSpan® compared to dried replantation after 45–60 minutes of dry storage (64) (Fig. 8.20).

**Slow release anti-inflammatory drugs using the root canal as a reservoir**

Exciting experimental results have been obtained recently by placing anti-inflammatory agents inside the root canal so as to achieve a slow and continuous release of the

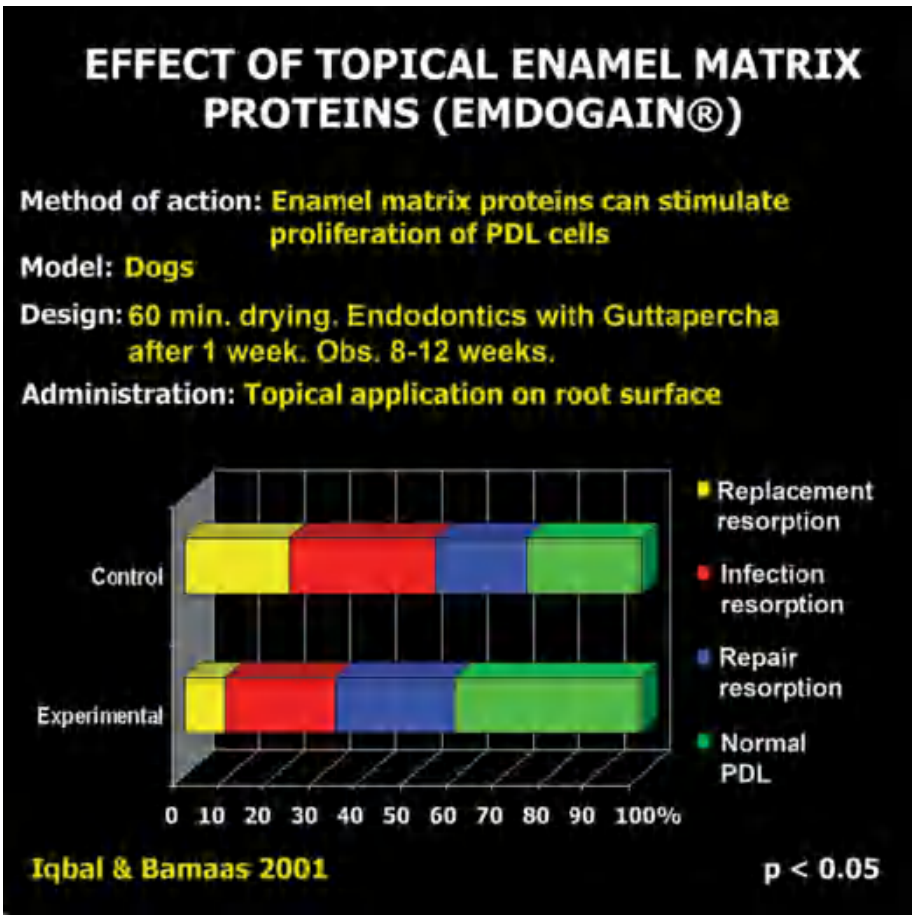



**Fig. 8.17** Effect of Emdogain® on PDL healing after replantation in dogs. Reproduced from (5) with permission.

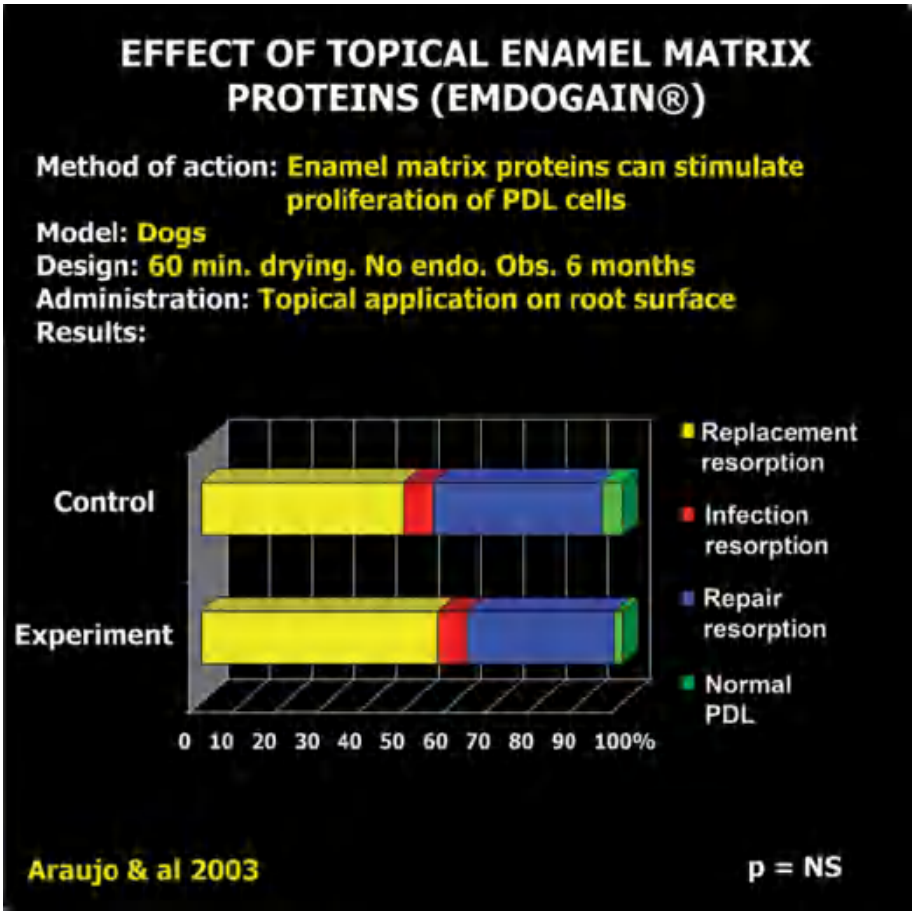



**Fig. 8.18** Effect of Emdogain® on PDL healing after replantation in dogs. Reproduced from (35) with permission.

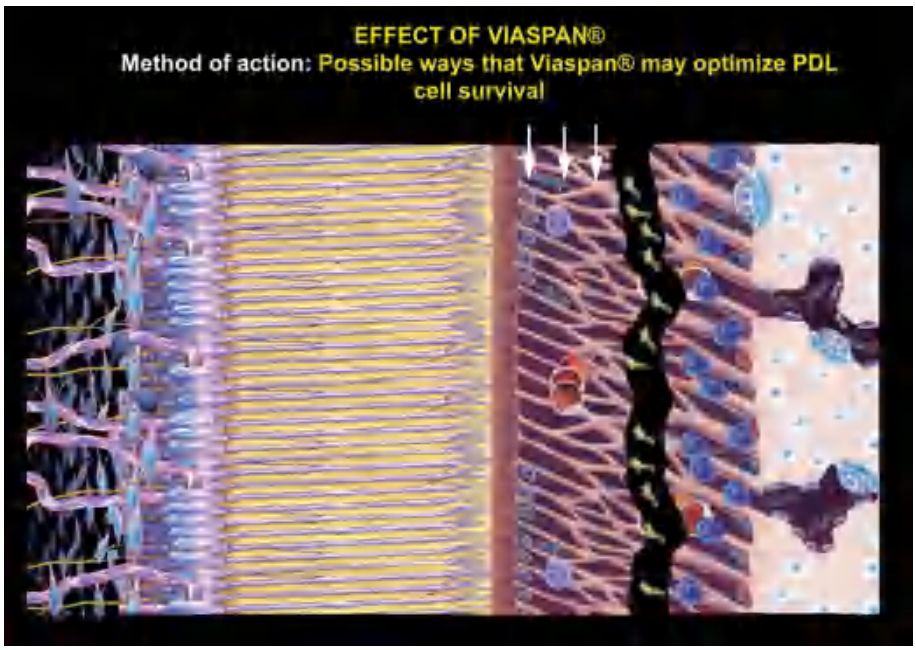



**Fig. 8.19** Possible ways that ViaSpan® may optimize PDL healing.

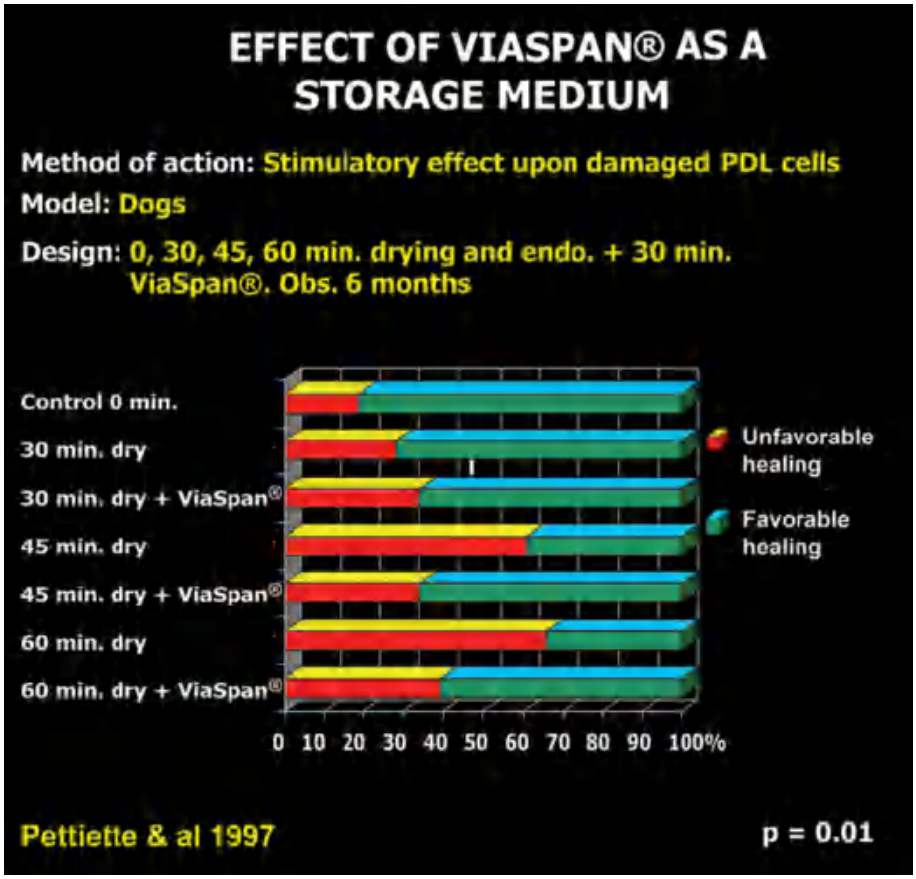



**Fig. 8.20** Effect of ViaSpan® on PDL healing after replantation in dogs. Reproduced from (64) with permission.

medicaments through the dentinal tubules to the site of osteoclastic activity.

Ledermix® is a water-soluble paste containing 1% triamcinolone and 3% demeclocycline. Triamcinolone is a highly active steroid providing potent anti-inflammatory action and demeclocycline is a broad-spectrum antibiotic effective against a large range of Gram-positive and Gram-negative bacteria. Studies have shown that the active agents in Ledermix® have a rapid initial release followed by a slow steady release. Ledermix® is able to diffuse through dentin, and levels of triamcinolone sufficient for anti-inflammatory action have been recorded in the periradicular area. In addition, triamcinolone has been shown to directly inhibit the spreading of dentinoclasts *in vitro* (36–38). Ledermix® has

been previously proposed as a medicament to control pulp infection. This procedure is usually delayed until the second visit. The hypothesis in this experiment was that the Ledermix® should be used at the emergency visit so as to use its anti-inflammatory action when the initial active inflammation occurs (Fig. 8.21).

Bryson et al. tested the effect of the immediate placement of Ledermix® compared to calcium hydroxide in the pulp canal of dogs' teeth that had been dried for 60 minutes (39). The Ledermix®-treated roots showed 59% favorable healing compared to 14% for the calcium hydroxide group, which was not different from the control group (Fig. 8.22).

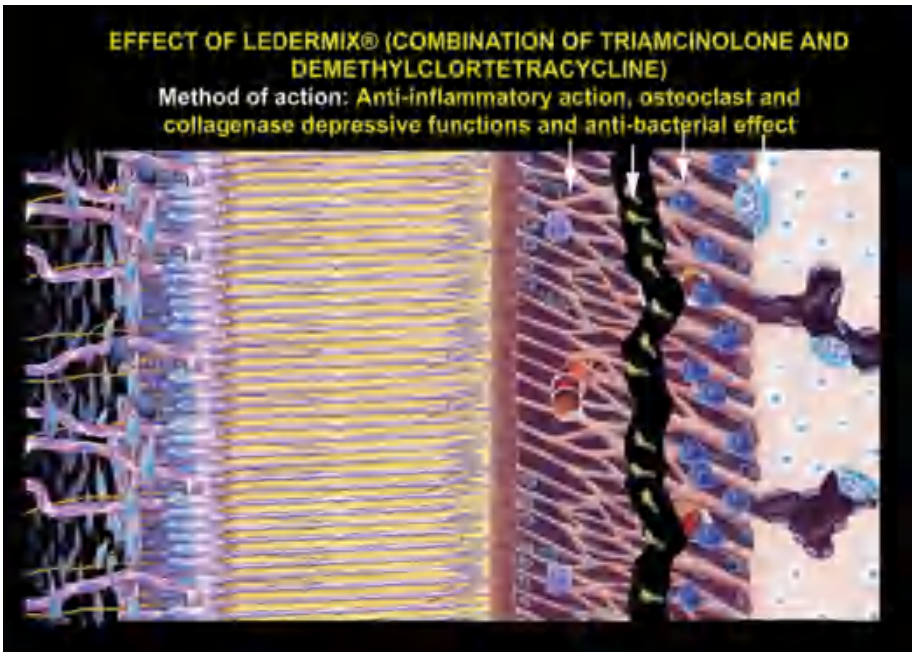



**Fig. 8.21** Possible ways that Ledermix® may optimize PDL healing.

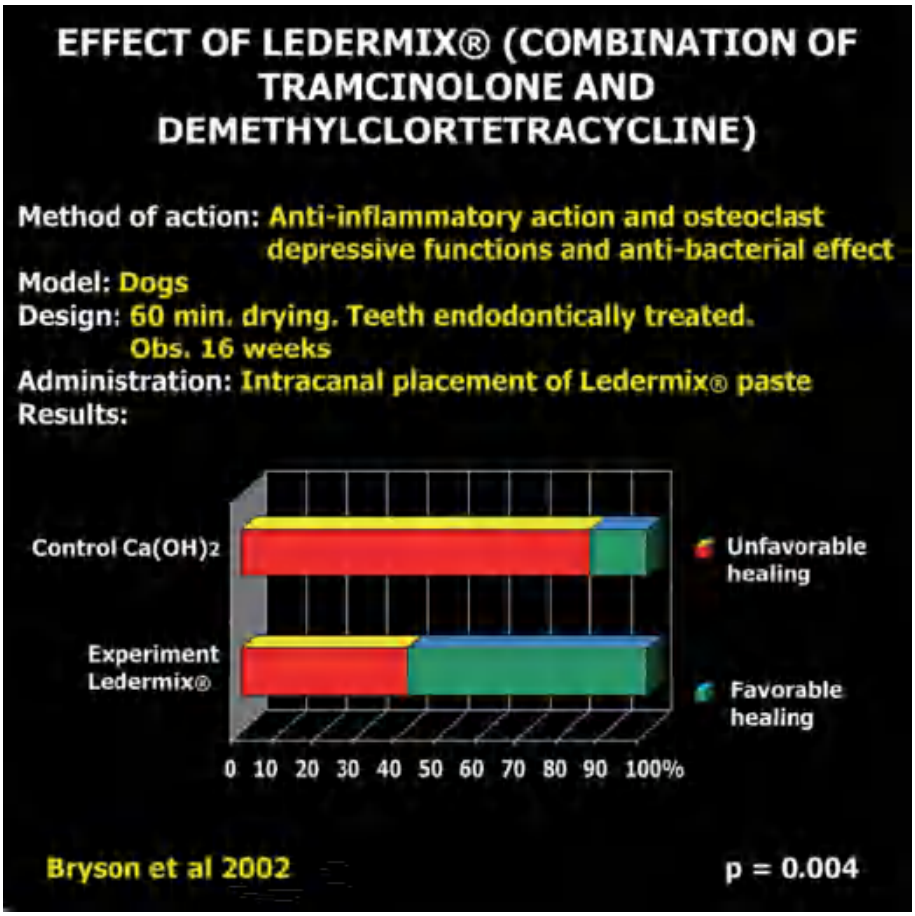



**Fig. 8.22** Effect of Ledermix® on PDL healing after replantation in dogs. Reproduced from (39) with permission.

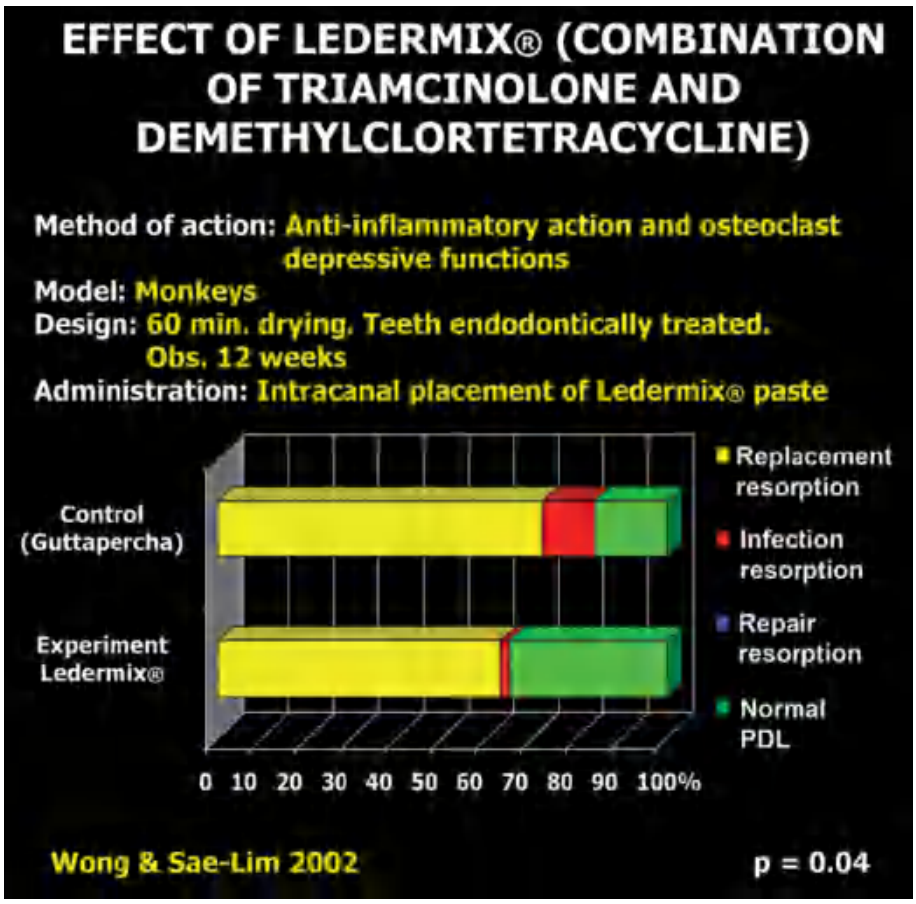



**Fig. 8.23** Effect of Ledermix® on periodontal healing after replantation in monkeys. Reproduced from (40) with permission.

In addition, the remaining root structure was significantly greater in the Ledermix®-treated teeth.

In monkey teeth, Wong and Sae-Lim also found a threefold favorable healing compared to the control teeth (40) (Fig. 8.23). This high incidence of favorable healing under the conditions of these studies is extremely promising and may constitute a major step forward in the treatment of these types of traumatic injuries.

Pierce and Lindskog had previously used Ledermix® in the root canals of monkey teeth and did not find a beneficial effect in relation to normal PDL (41) (Fig. 8.24). However, they placed the medicament 3 weeks after the experimental injury when presumably the acute inflammation had subsided and had already produced its deleterious effect.

The use of an anti-inflammatory medicament at the emergency visit is a change from previous practice where the root canal was not involved until the second visit. Unfortunately, the tetracycline medicament in Ledermix® has the potential to discolor the tooth and to address this, Chen et al. (61) examined the potential for favorable healing when tetracycline and the corticosteroid were placed alone compared to being placed in combination in the Ledermix® medicament. The favorable effect of Ledermix® was confirmed in this experiment, but, the corticosteroid was shown to have an equally positive effect (Fig. 8.25A). Thus it appears that the corticosteroid may be used instead of Ledermix®, thus negating the discoloration problem. Finally, it should be mentioned that one randomized study in humans comparing Ledermix® and calcium hydroxide (Ultracal XS®) as a root canal dressing for 6 months showed no significant difference in PDL healing (73) (Fig. 8.25B). However, in this study only 27 teeth in both groups qualified for evaluation, making statistically significant differences an unreasonable expectation.

## Systemic methods to limit inflammation

As previously discussed, a traumatic injury will predictably cause inflammation due to the physical damage to the root surface. In addition, if bacteria are able to colonize the root surface an additional inflammatory stimulus is present. The most likely source of bacteria is the external environment at the site of the injury or bacteria from the mouth that migrate down the blood clot that forms in the periodontal ligament.

Systemic antibiotics have been tested in order to remove socket bacteria that may act as inflammatory stimuli. These were started immediately at the time of repositioning at the emergency visit when the bacterial count and thus the resultant inflammatory response should be highest (Fig. 8.26).

*Penicillin.* Penicillin is the traditional antibiotic used in oral infections and thus logically would appear to be the drug of choice to counteract bacteria within the periodontal space after a traumatic injury.

Hammarström et al. tested penicillin under two experimental conditions. In the first experiment the root canals

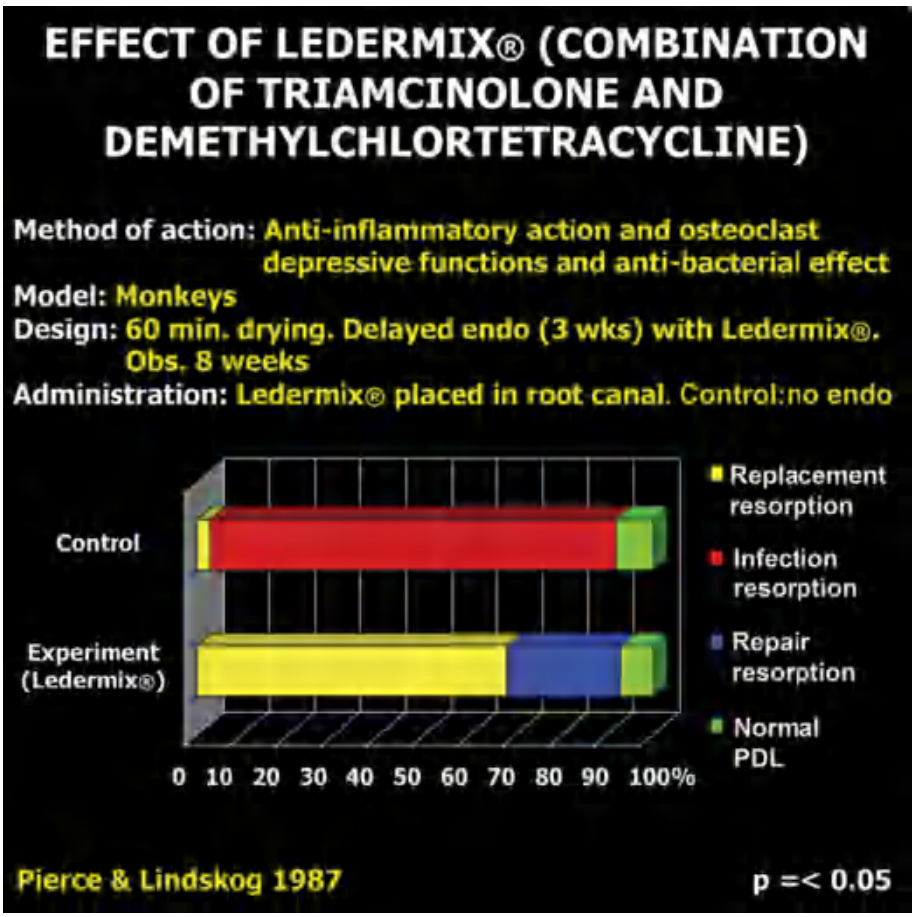



**Fig. 8.24** Effect of Ledermix® on periodontal healing after replantation in monkeys. Reproduced from (41) with permission.

were infected in addition to an extraoral dry time of 60 minutes. In this experiment the use of systemic antibiotic had a marked beneficial effect on healing by eliminating active inflammatory root resorption (42) (Fig. 8.27).

In the second experiment root canal therapy was performed and thus root canal bacteria did not play a role after the 60-minute dry time. Under these circumstances the antibiotic did not have an appreciable effect on the healing results (43) (Fig. 8.28).

Since at the emergency visit periodontal inflammation is unlikely to be present due to pulp space infection, the experiment where endodontic treatment was performed before replantation appears to be more relevant to the emergency visit. The use of systemic penicillin was not shown to be beneficial, a finding supported by a clinical study of avulsed and replanted teeth (7).

*Tetracycline.* As already described, tetracyclines have antiresorptive properties in addition to their antibacterial properties. Therefore they may be ideal as a drug to act systemically against bacteria on the root surface and also act against the resorbing osteoclasts.

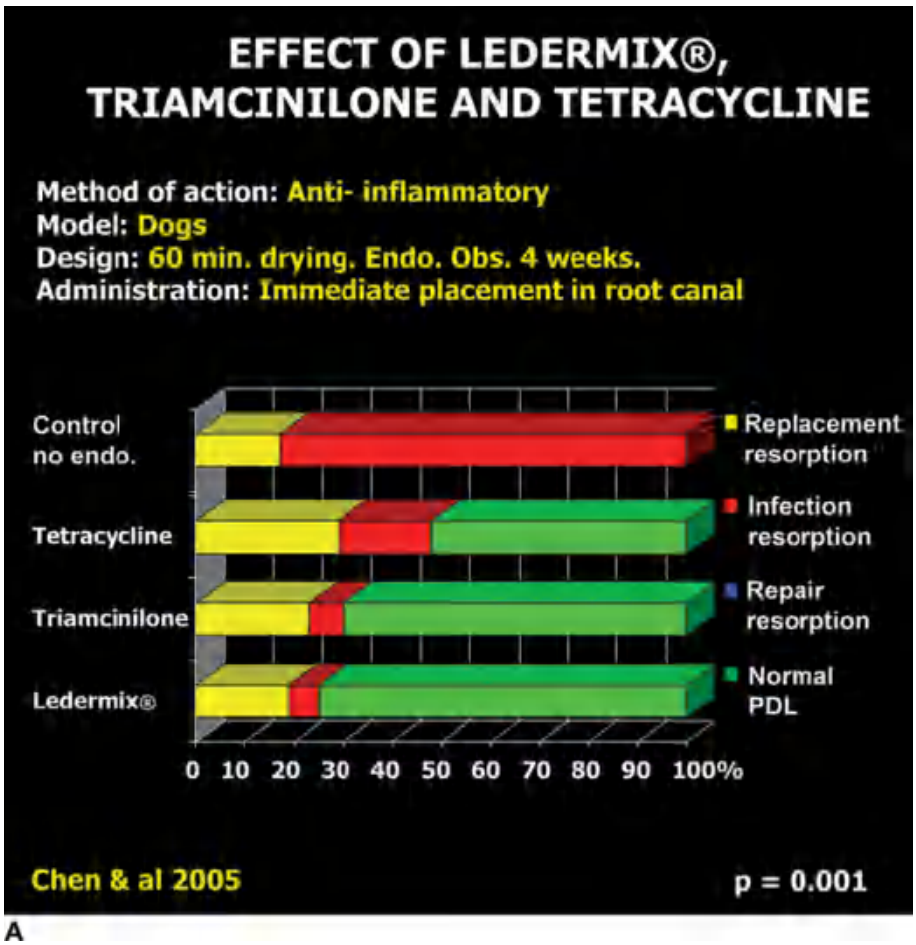
Sae-Lim et al. (44, 45) tested systemic doxycycline in similar models to the previous studies performed by Hammarström et al. with penicillin. When the root canals were infected in roots dried for 60 minutes both doxycycline and amoxicillin showed improved healing compared to the control. The healing with doxycycline was equal to that shown with the amoxicillin (Fig. 8.29). Thus it appears that the antibacterial effect of the tetracycline was similar to that of the penicillin drugs used by the Hammarström group (42, 45).

However, when the tetracycline antibiotics were tested in the model where dog teeth dried for 60 minutes were endodontically treated before reimplantation, an almost significant improvement in normal healing was seen compared to the controls (44) (Fig. 8.30). This was presumably due to the antiresorptive effect of the tetracycline which is not present for the penicillin drugs.

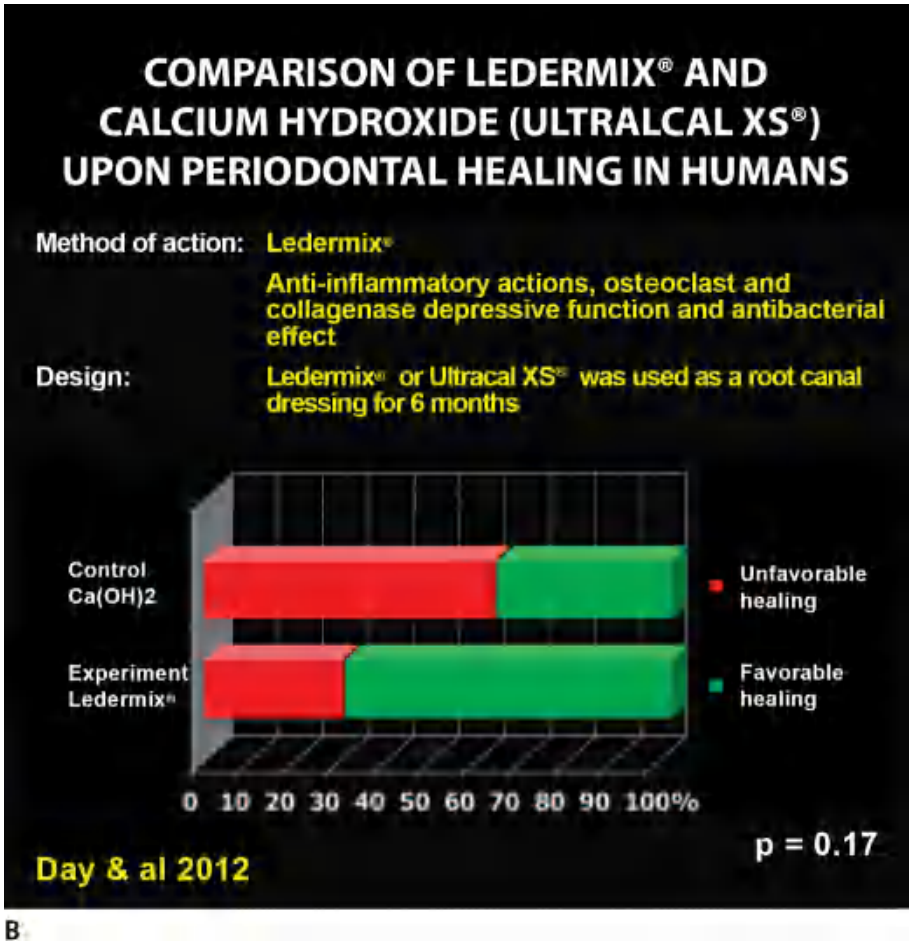
Thus it appears that systemic tetracycline rather than penicillin is the appropriate antibiotic for severe avulsion injuries and should be started at the emergency visit.

### *Mature tooth – extraoral dry time >60 minutes*

If a root has been dry for more than 60 minutes the PDL is not expected to be viable [46]. Thus eventual osseous replacement of the root is inevitable. However, with traumatic injuries, particularly in young patients, a slow replacement of the tooth root can be more successful compared to a root that is replaced quickly. Therefore, when the practitioner deems that it is worthwhile to keep the tooth, the aim of treatment is to prepare the tooth for as slow a replacement process as possible [74].




A




B

**Fig. 8.25** A. Effect of Ledermix®, triamcinilone and tetracycline on PDL healing after replantation. Reproduced from (61) with permission. B. Comparison of Ledermix® and calcium hydroxide on periodontal healing in humans. Reproduced from (73) with permission.

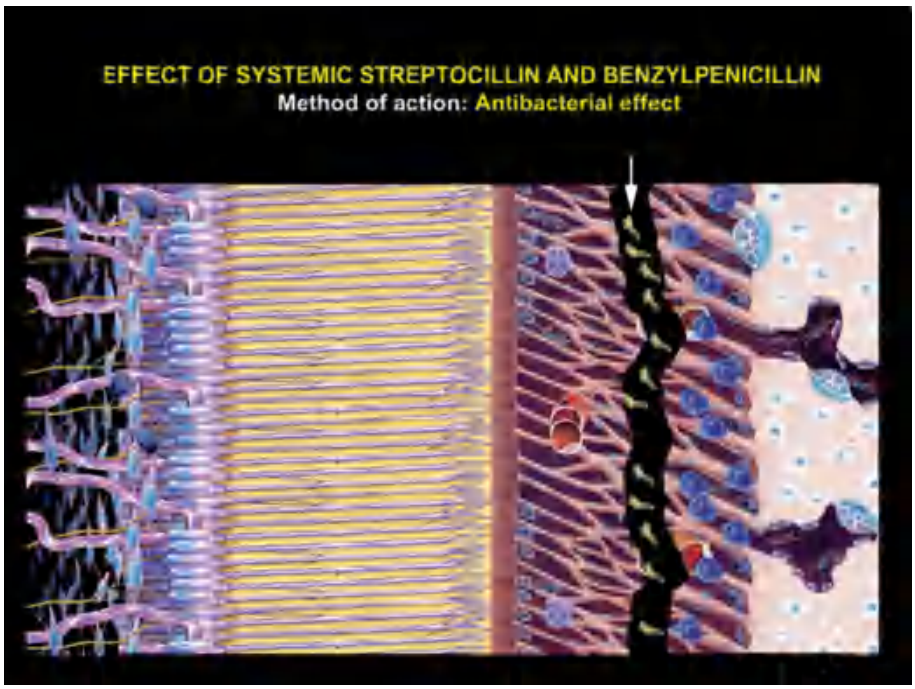



**Fig. 8.26** Effect of antibiotics on PDL healing after replantation.

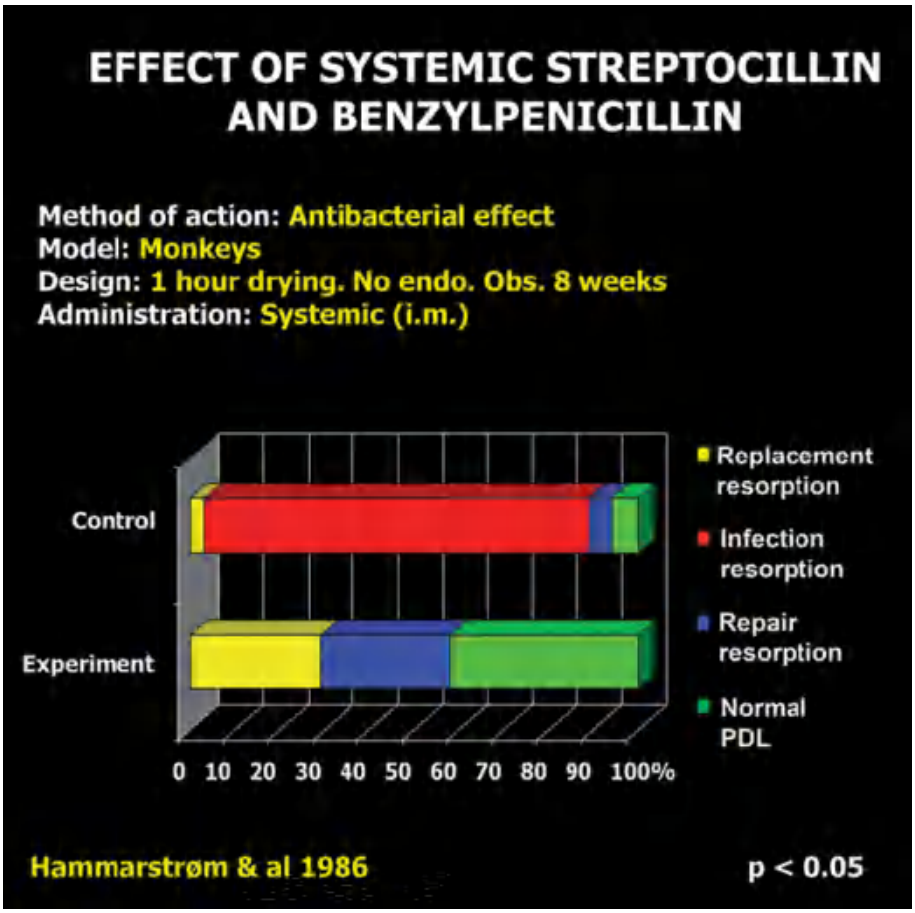



**Fig. 8.27** Effect of systemic streptocillin and benzylpenicillin on PDL healing after replantation and root canal infection in monkeys. Reproduced from (42) with permission.

## Fluoride pretreatment of the root

Since 1968 it has been known that sodium fluoride treatment of the root surface before replantation may decrease the rate of osseous replacement in experimental replanted teeth in monkeys (65) (Fig. 8.31). A study in humans likewise demonstrated a 50% reduction in progression of root resorption after replantation (66). In later experimental studies it was found that 1% stannous fluoride supplemented

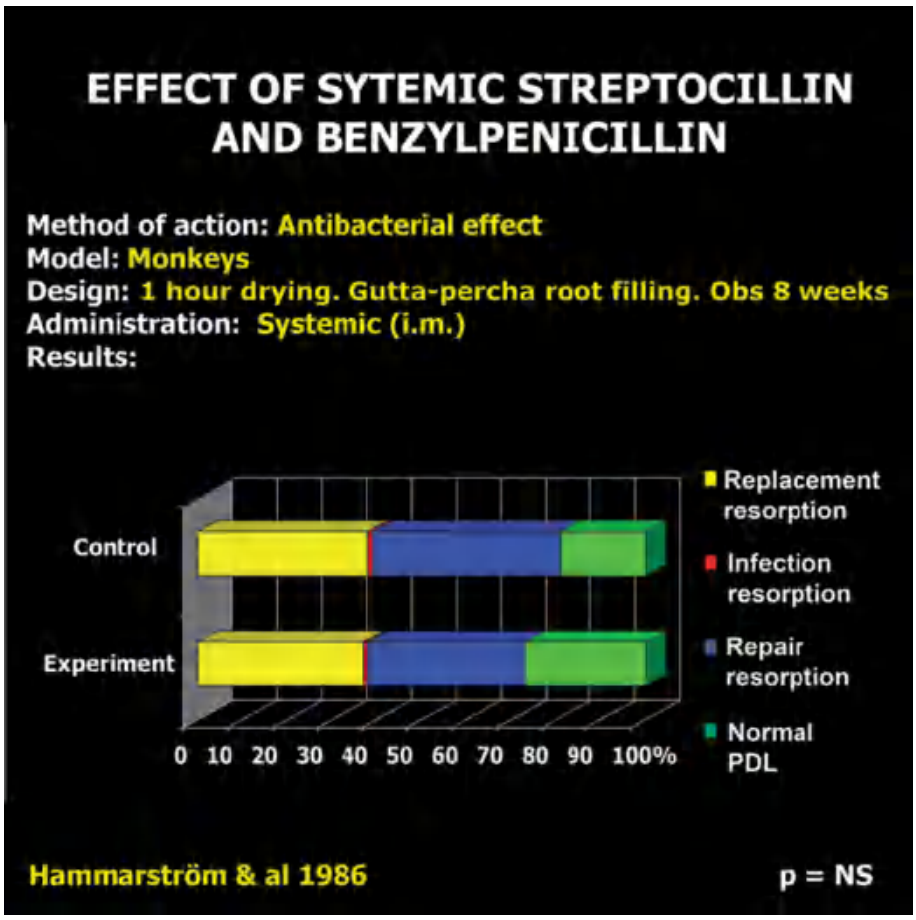



**Fig. 8.28** Effect of systemic streptocillin and benzylpenicillin on PDL healing after replantation in monkeys. Reproduced from (42) with permission.

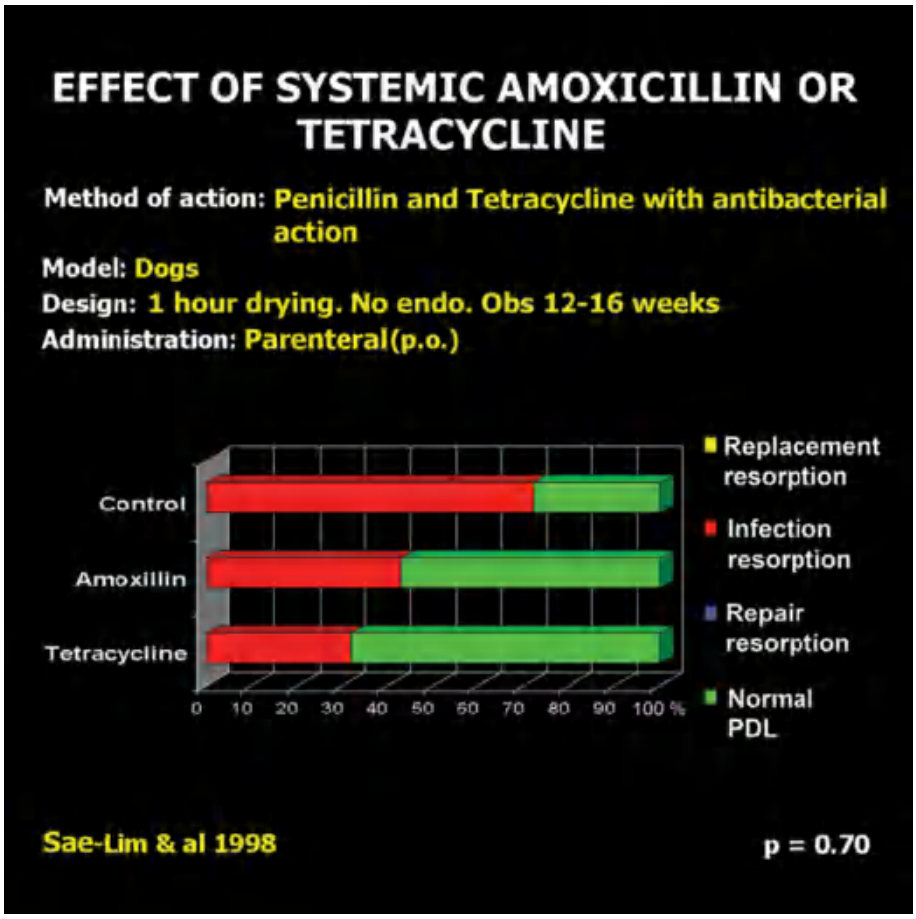



**Fig. 8.29** Effect of amoxicillin or tetracycline on PDL healing after replantation in dogs. Reproduced from (45) with permission.

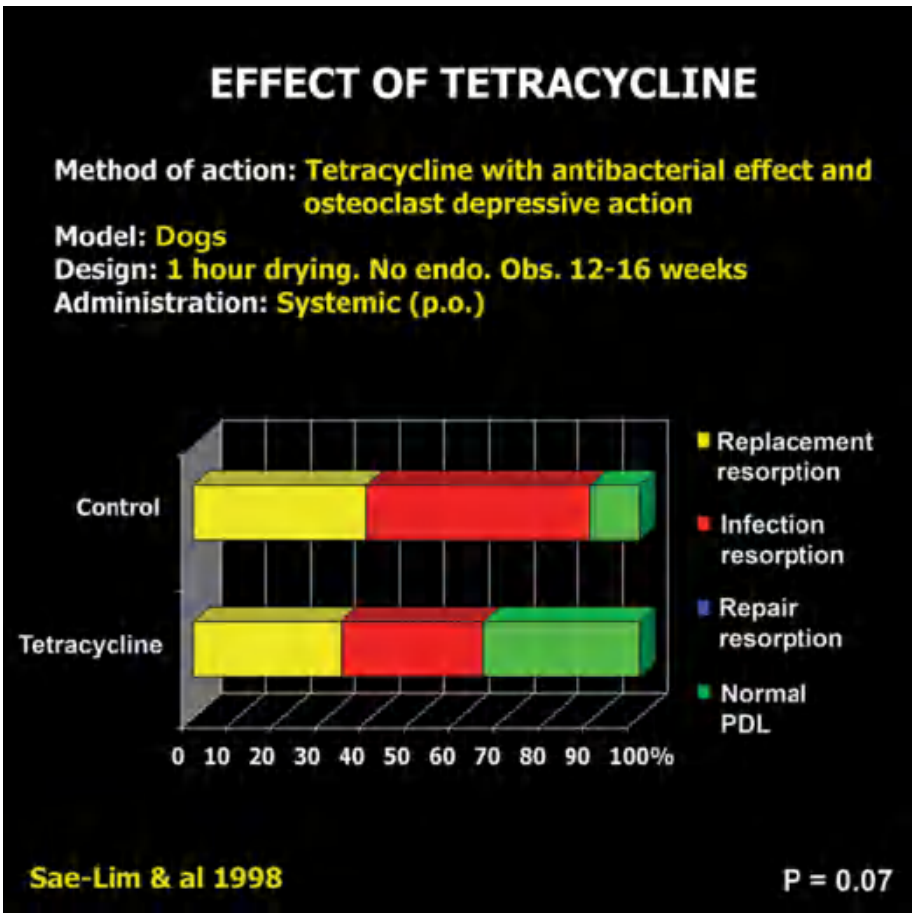



**Fig. 8.30** Effect of tetracycline on PDL healing after replantation in dogs. Reproduced from (44) with permission.

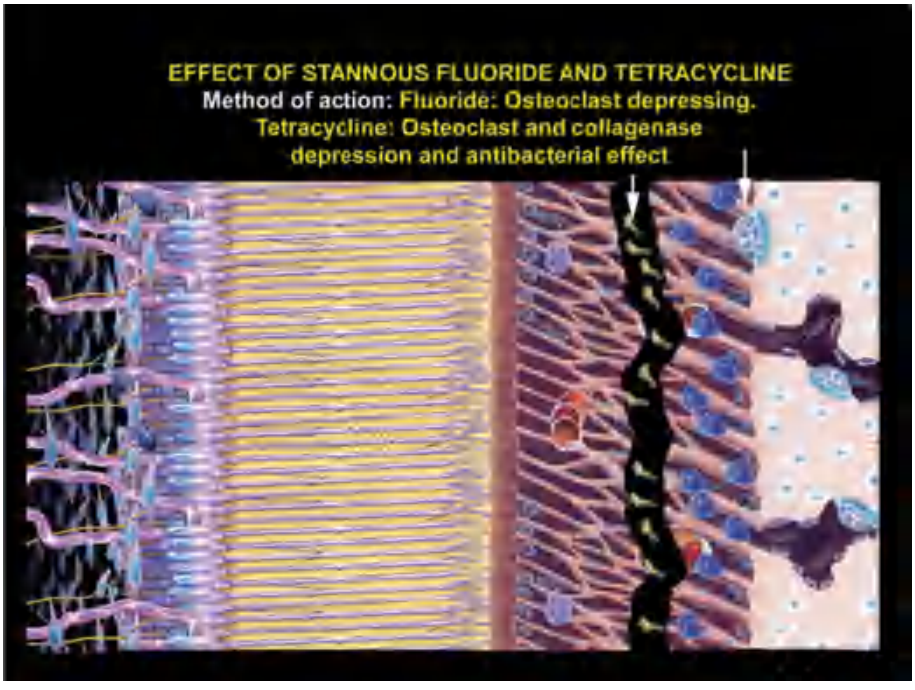



**Fig. 8.31** Effect of stannous fluoride and tetracycline on PDL healing after replantation. Reproduced from (60) with permission.

with tetracycline had an ankylosis-depressing effect; however a longstanding inflammation reaction in the PDL was found (67, 68). In a subsequent study, the concentration of stannous fluoride was lowered from 1% to 0.1% which resulted in a significant reduction of inflammation in the PDL (Fig. 8.32) with a minimal amount of ankylosis. Although this study seems promising it should be considered that the observation period in this experiment was only 4 weeks, an observation period which in similar experiments has been found much too short to exclude later osseous

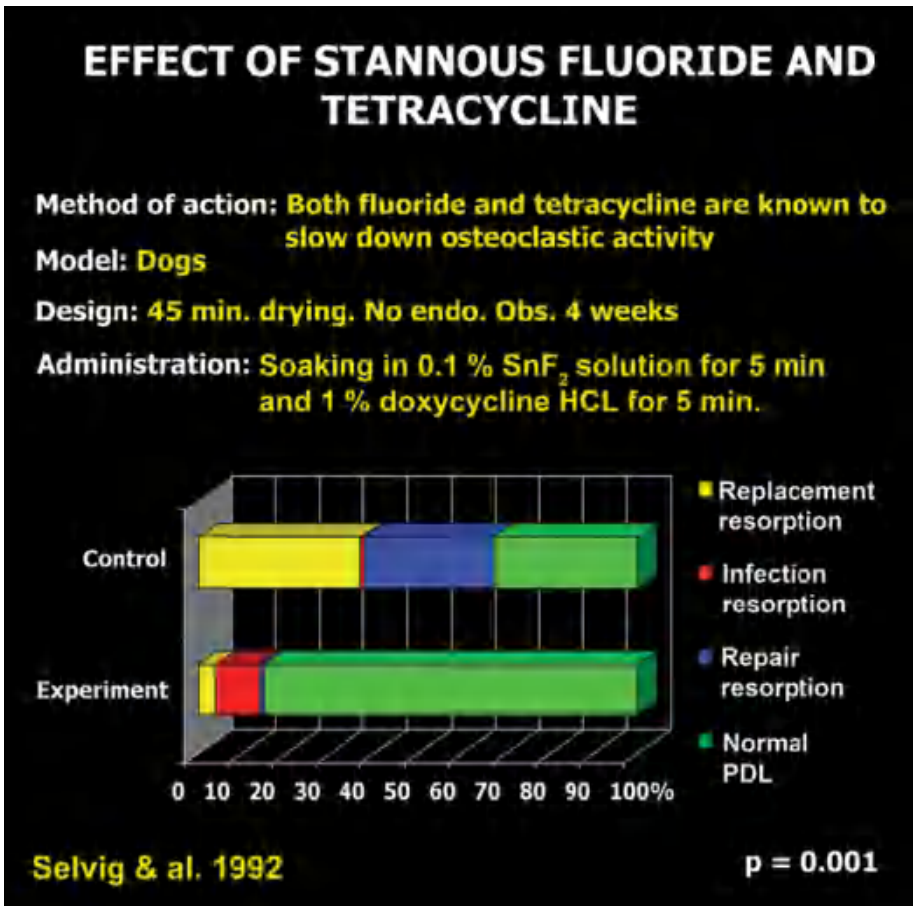



**Fig. 8.32** Effect of stannous fluoride and doxycycline on PDL healing after replantation in dogs. Reproduced from (60) with permission.

replacement (66). This implies that the use of sodium fluoride solution (2.4%) is the only tested and useful method that can be recommended (66).

## Physical methods

Probably the most important action that should be taken to slow down osseous replacement is to remove the dead PDL cells on the root surface (2, 8, 16). If these cells remain after replantation they will act as potent inflammatory stimulators. In order to remove the remaining cells the root can be soaked in sodium hypochlorite or a weak acid. Some authors have proposed using a periodontal curette to remove the cells. Logically this may not be as good since scraping off the cemental layer would probably allow the resorption to progress at a more rapid rate.

## Chemical methods

### *Immature tooth – extraoral dry time <60 minutes*

The luxated or avulsed immature tooth is different from the mature tooth in that the pulp that is necrotic due to the traumatic injury has a possibility of revascularization. Revascularization of an immature tooth is of tremendous benefit as the root canal walls will continue to develop and strengthen and the apex will close, making future endodontic treatment, if needed, a more predictable procedure. The less developed the root at the time of injury, the greater the potential for revascularization (47–49).

It appears that bacteria on the root surface, particularly at the apical foramen, are the reason for failure of revascularization (47). Thus it is essential to limit bacteria from the accident site and those tracking down the blood clot in the socket.

Doxycycline, as discussed, has antibacterial and antiresorptive properties. Cvek et al. tested the effect of soaking extracted immature monkey teeth in doxycycline before replantation (47). They found that the revascularization rate doubled (18–41%) after the doxycycline soak (Fig. 8.33). However, they could not reproduce this effect with systemic doxycycline (71) (Fig. 8.34).

Yanpiset and Trope repeated the Cvek study in dogs and were able to replicate the doubling of healing rates with the use of the doxycycline (30–60%) compared to saline controls (48) (Fig. 8.35).

More recently, Ritter et al. were able to further increase the revascularization rate in dogs with the use of minocycline (50) (Fig. 8.36). Minocycline is a slow release tetracycline that has been shown to decrease periodontal bone loss when administered topically. Its slow release action may keep bacteria from entering the pulp space long enough to allow a higher incidence of revascularization than with doxycycline, which has a shorter period of action. A number of studies have shown that the local administration of a 2% minocycline gel is effective in the treatment of periodontitis (51–53).

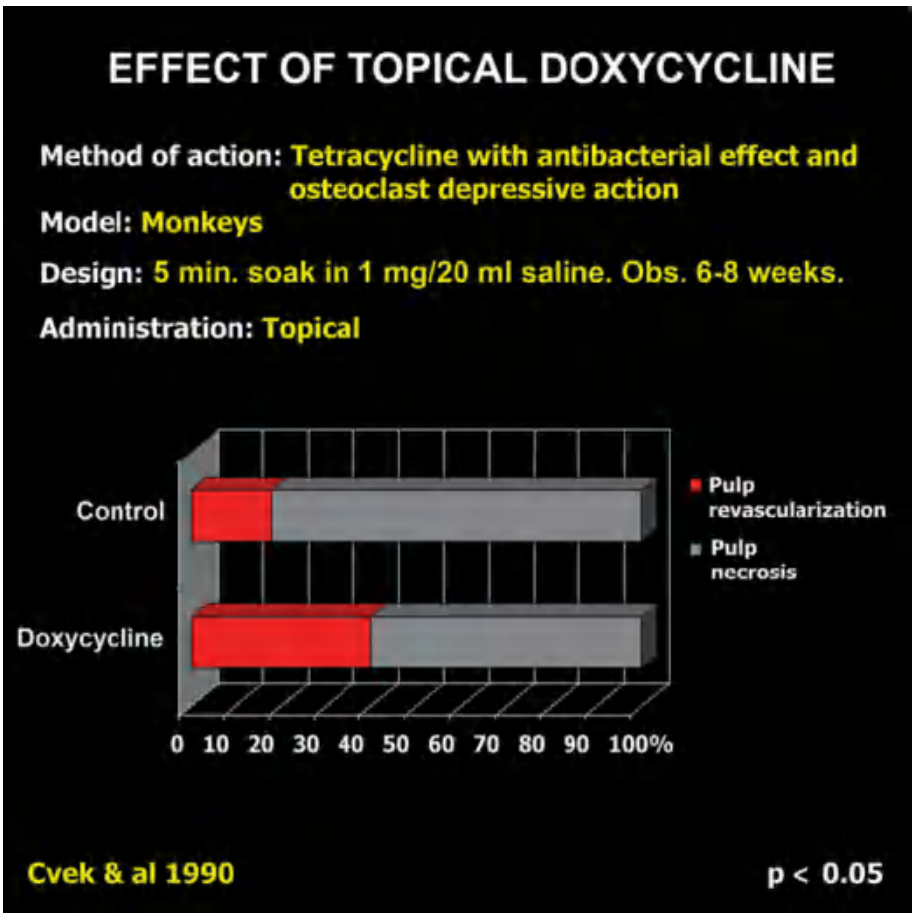



**Fig. 8.33** Effect of topical doxycycline on pulp healing after replantation in monkeys. Reproduced from (47) with permission.

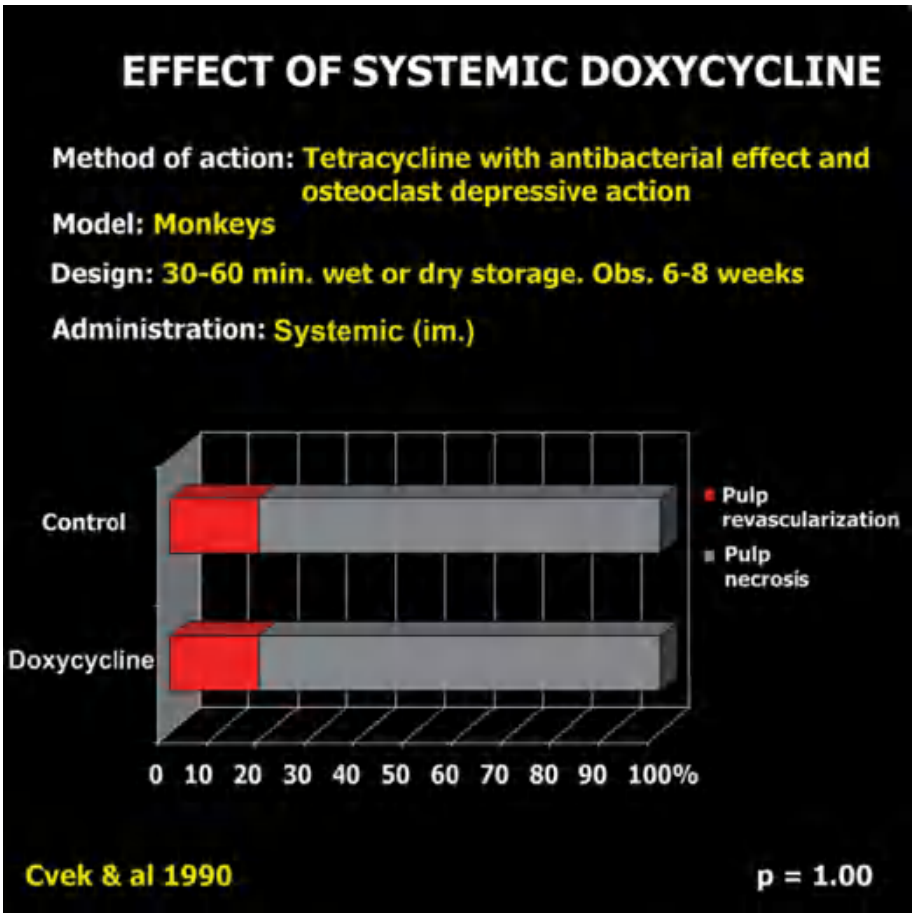



**Fig. 8.34** Effect of systemic doxycycline on pulp healing after replantation in monkeys. Reproduced from (71) with permission.

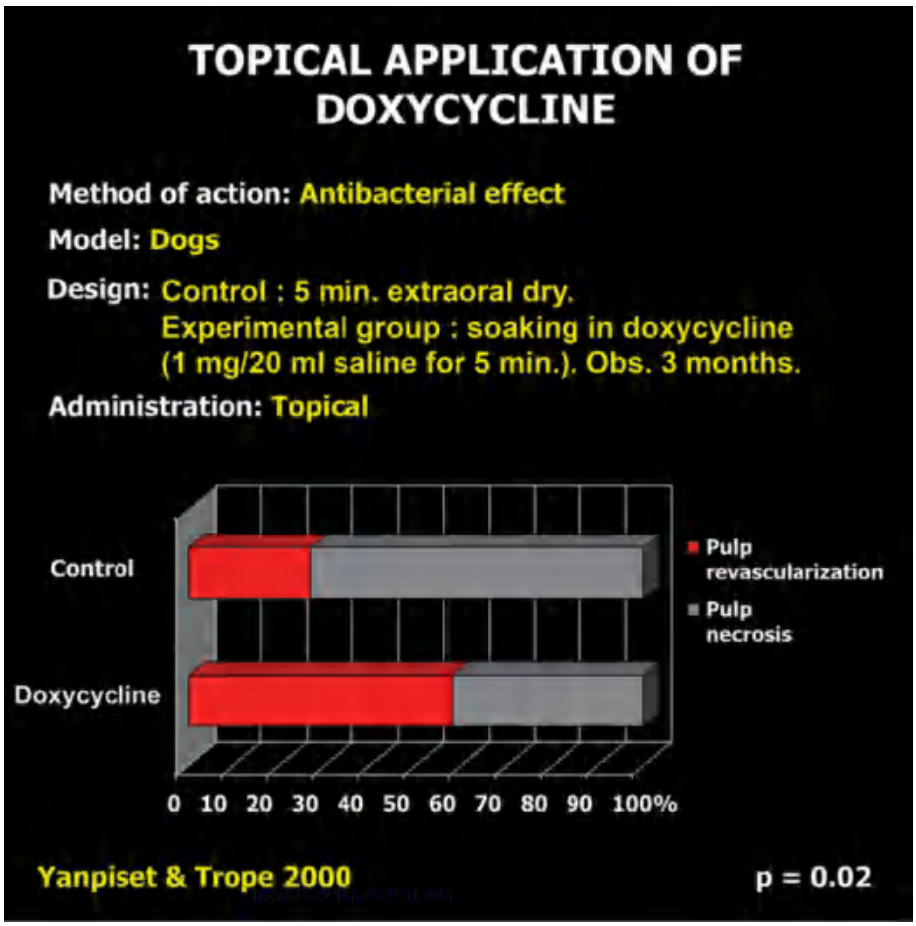



**Fig. 8.35** Effect of topical application of doxycycline on pulp healing after replantation in dogs. Reproduced from (48) with permission.

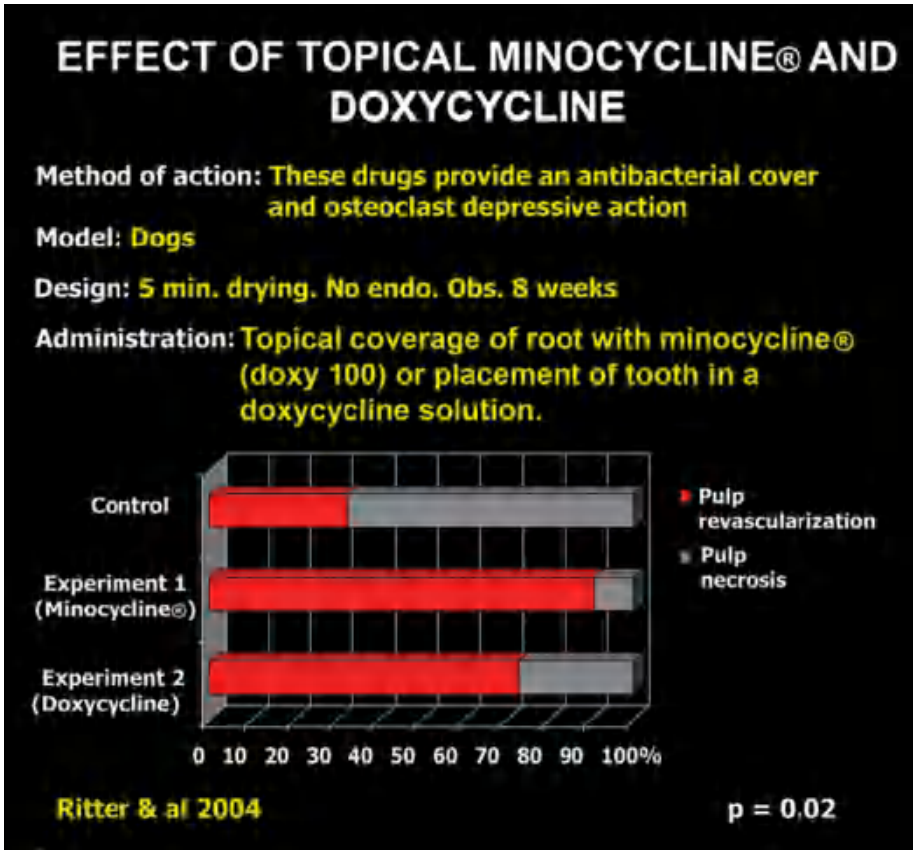



**Fig. 8.36** Effect of topical application of minocycline and tetracycline on pulp healing in dogs. Reproduced from (50) with permission.

Minocycline is available in the form of Arestin™ Microspheres (OraPharma, Inc.), which is a sustained-release product containing minocycline hydrochloride in a bioabsorbable polymer (polyglycolide-co-DL lactide). The resulting microspheres combine the minocycline and bioabsorbable polymer in a powder form. Immediately upon contact with moisture, the polymer hydrolyzes, releasing minocycline. Concentrations of 340 µg of minocycline per milliliter have been measured in human crevicular fluid after 14 days, exceeding the minimum inhibitory concentrations of many pathogens (54).

In a recent clinical retrospective case–control study, 5 minutes' doxycycline storage of avulsed teeth compared to saline storage affects periodontal ligament and pulp healing. At the follow-up no significant differences were found between doxycycline and saline storage in regard to both periodontal and pulp healing (72).

### *Immature tooth – extraoral dry time >60 minutes*

The benefit of replanting an immature tooth that will definitely be lost due to osseous replacement has been debated; and some authors have concluded that such teeth should not be replanted (see Chapter 20).

In this author's opinion, such teeth can provide a benefit to the young patient while waiting for facial growth to be completed. If the tooth is replanted with the root conditioned in a manner similar to that of the adult tooth, with an extended dry time and then submerged, it can help to maintain the height and width of the socket for an extended time period (see Chapter 32). In some cases this will negate the need for complicated prosthetic reconstruction when facial growth is completed.

## Second visit

The attachment damage due to the traumatic injury and minimizing subsequent inflammation is the focus of the emergency visit. The second visit should be scheduled 10–14 days after the emergency visit. At this visit the focus is on preventing pulp space infection which sustains active inflammatory root resorption and results in loss of the root structure in a very short period of time (2, 7).

Root canal disinfection removes the stimulus to periradicular inflammation and the active resorption will stop (55, 56). In most cases a new attachment will form; but if a large area of root is affected, osseous replacement can result by the mechanism already described. Again, treatment principles include prevention of pulp space infection or elimination of the bacteria if they are present in the pulp space. Details of how to achieve these aims are described in detail in Chapters 25 and 26.

## Essentials

A series of physical and chemical methods have been used experimentally to optimize periodontal and/or pulpal healing after replantation of avulsed teeth. Most are experimental methods that have been validated in animals only and need to be verified in future clinical studies. For treatment guidelines of avulsion see also Chapter 20.

## Summary of experimental findings

### *PDL healing*

- Rinsing the root surface with tap water for 10 seconds before replantation appears to favor PDL healing
- Minimizing dry time and appropriate storage is critical
- 30 minutes extra storage in a tissue storage medium may improve PDL healing after 45–60 minutes' dry storage
- A combination of triamcinolone and tetracycline or triamcinolone alone placed in the root canal at time of replantation appears to favor PDL healing
- Tetracycline hydrochloride systemically administered appears to favor PDL healing
- Amoxicillin systematically administered for 7 days (22 mg/kg) appears to favor PDL healing
- Sodium fluoride 2.4% used topically for 20 minutes appears to slow down progression of replacement resorption but should only be used in cases with extensive damage to the PDL

### *Pulpal healing*

#### Immature tooth

Doxycycline (1 mg/20 mL saline) soaked for 5 minutes or minocycline topically appear to greatly improve the chance of pulp revascularization.

#### Mature tooth

Pulp healing is not possible. Endodontic treatment to prevent pulp space infection is critical.

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# 9

# Psychosocial Aspects of Traumatic Dental Injuries

J. Traebert, U. Rydå & A. Robertson

## Introduction

Facial esthetics play an important role in self-identification, self-image, self-presentation and interpersonal confidence (1). Furthermore, they affect social behavior (2). In most cultures, the face is regarded as the most salient characteristic of one's identity (1). The face is an important part of human development and facial expression is the most important element in non-verbal communication. The significance of the teeth in a smile should not be underestimated (3).

Deviation from the 'norm' can be as simple as a dental anomaly or as complex as a craniofacial deformity (4). A traumatic dental injury (TDI), be it a fracture, discoloration of teeth or avulsion of a tooth, will alter facial appearance (5) (Fig. 9.1). What is more, the effects of TDIs on self-esteem and self-awareness are important because the majority of TDIs occur in the early years of life and adolescence (6, 7), which are periods of important psychological development when the growing person is particularly sensitive (8–10) (Fig. 9.2). This may lead to long-term psychological effects, in particular if the TDI is associated with a physical as well as a psychological traumatic experience.

Understanding the complexity of factors that make up esthetic standards is important in treatment planning (11). Personal appearance is important for self-esteem. The lack of positive references can lead to a loss of self-confidence, an unwillingness to perform daily activities and even isolation (12). A study conducted in Brazil (13) evaluated the need for orthodontic treatment using both normative criteria and the adolescents' self-perception of their dental appearance and mastication function. The authors concluded that the adolescents' feelings of satisfaction or dissatisfaction with their dental appearance should be taken into consideration to define the need for treatment.

Psychologists define a traumatic experience as an intense and sudden event that overwhelms the person's capacity to cope with the memories and feelings that are triggered by it. Such traumatic experiences may lead to psychological symptoms, such as depression and anxiety (14).

Quality of life measurements include a wide range of health-related factors. There is empirical and theoretical justification to consider the relationship between health status and quality of life as complementary concepts. Social responses conditioned by facial or dental esthetics and functionality can seriously affect the overall well-being of an individual and their dental problem should not be trivialized (15).

This chapter will address the social and psychological impact of TDI on the quality of life and point out procedures that can be incorporated into dental practice to reduce psychosocial outcomes.

## Pyschosocial impacts of traumatic dental injuries

Clinical or normative indicators measure the biologic consequences of oral health injuries, but do not express the distress and limitations experienced by individuals. The emphasis on clinical indicators masks the social dimensions of oral health (16). Therefore, clinical diagnosis should not be the only determining factor for any intervention or treatment; the subjective perception of the individual should also be taken into account.

The impact of oral conditions on an individual's physical and psychosocial well-being can be assessed through subjective indicators called Oral Health-Related Quality of Life (OHRQoL) measures. The aim is to assess major changes in behavior that upset social function and consequently alter people's quality of life (16).

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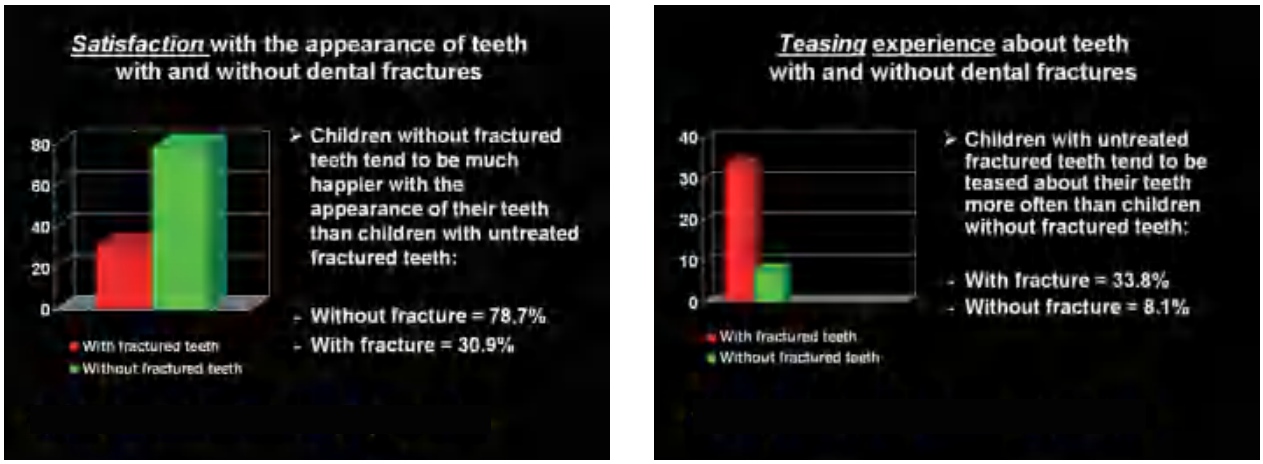
**Fig. 9.1** The fracture or loss of incisors may dramatically alter facial appearance. The photographs on the right simulate various types of dental trauma.




A B

**Fig. 9.2** Patient satisfaction and teasing experience related to trauma experience. From (5).

Studies on oral health and quality of life should address four areas: (1) pain and discomfort; (2) functional aspects related to the ability to chew and swallow food without difficulty and speak and pronounce words correctly; (3) psychological aspects related to physical appearance and self-esteem; and (4) social aspects reflecting social interaction and communication with people (17, 18). These dimensions were described by Locker (19), based on *The International Classification of Functioning, Disability and Health* developed by the World Health Organization (WHO) (20). According to Locker (19), mouth injuries and diseases cause damage that can directly lead to disabilities or indicate the following symptoms: pain and discomfort, functional limitation and dissatisfaction with one's own appearance. These symptoms can trigger physical, psychological or social limitations, and consequently, disability. In turn, pain and discomfort can lead straight to disability.

Oral health problems have been increasingly recognized as having an important negative impact on quality of life for individuals and populations. Different oral health status levels have distinct impacts on the daily lives of people. It is vital that social and psychosocial dimensions are considered simultaneously with the clinical condition in the study of people's oral health needs (5). The WHO (21) recognizes that oral diseases and conditions cause pain, suffering, psychological constraints and social deprivation at both individual and population levels.

The appearance and position of the anterior teeth have a particular potential to affect pyschosocial aspects of quality of life (22). Given that TDIs occur most commonly in the

anterior sextants of the dental arches (23), this is especially important. The psychosocial problem may occur due to the incident that caused the TDI or because of esthetic impairment by a broken or blackened tooth (24), an increase in dentin sensitivity attributed to the exposure by fracture (25) or another functional problem (24).

Consequently, TDIs that result in pain and discomfort, dyschromia and other esthetic changes or partial or total tooth loss can make the person avoid smiling or speaking clearly, thus affecting social relationships (26). Also, a study conducted in the UK highlighted the high cost of reparative and restorative TDI treatments, in addition to school absenteeism from post-trauma conditions (27).

A number of recent studies have pointed to the negative impacts associated with TDIs (22, 25, 26, 28–38). Indeed, the nature of occurrence invariably includes some kind of either voluntary or involuntary violence (39). The magnitude of the psychological impact depends on the type of event or associated violence. For example, when TDI is the result of aggression, physical assault or abuse, the potential to generate psychological stress is much higher than when resulting from sports activities or an accidental fall. These conditions can cause serious esthetic, psychological and social damage, besides producing significant costs for the TDI victim and their family (40).

Back in the early 2000s, a study conducted in Brazil showed that children with fractured teeth had a 20-fold increased chance of suffering a negative impact on quality of life as compared to children without TDI when they performed daily tasks such as eating, tooth cleansing and smiling, as well as a greater problem in maintaining a normal emotional state (5). Another study investigating the impact of treatment for enamel-dentin fracture on the daily activities of adolescents, pointed to a 3.3-fold higher chance of reporting impacts related to daily activities among those with treated fractures, as compared to those without fractures (41). These two studies, however, used the Oral Impact on Daily Performance (OIDP) measure (42), which is not an OHRQoL measure designed specifically for children.

In Canada, Fakhruddin et al. (43) used a specific indicator to measure OHRQoL in children, the Child Perception Questionnaire for 11–14-year-old children ($CPQ_{11-14}$) (44), in a case–control study. They found that children with untreated TDIs had more difficulty in chewing and avoided smiling more often than their counterparts. It was concluded that TDI-related problems affect children's psychological and social well-being, diminishing their quality of life.

However, a more recently published study (26) showed no significant difference in quality of life between children with treated TDIs as compared to those with an untreated TDI, using the total scores of the $CPQ_{11-14}$ indicator (44). Nevertheless, this study pointed out that children with an untreated TDI were 1.4-fold more likely to report an impact on the item 'avoided smiling/laughing' than those without TDIs, while children with a treated TDI were two-fold more likely to report an impact on the item 'other children asked questions' than those without TDIs.

Aiming to study the impact of TDI on the quality of life of 11–14-year-old schoolchildren using $CPQ_{11-14}$ (44), Traebert et al. (24) demonstrated a statistically significant and independent association between poorer perception of quality of life and the presence of a TDI. Children with TDIs showed a 62% higher prevalence of poor quality of life as compared to those without TDIs, regardless of gender, level of education of their mothers, present or past history of caries and presence of malocclusion.

Nonetheless, it is important to highlight that different studies have not achieved similar results, which indicates the need for further research to find out which variables are determining or interfering with such inconclusive results. Piovesan et al. (45) showed no significant differences in the reporting of impact on quality of life among 12-year-old schoolchildren with crown fractures and without fractures. Others studies have shown similar results (46, 47). On the other hand, non-population-based studies (36, 48, 49) have found significant differences in the reported impact on quality of life only in the most severe cases of TDI, involving pulp tissue, dislocations and other more severe conditions, between children with and without TDIs. Minor injuries such as cracks and fractures involving only enamel are less important in in having a psychosocial impact (50). Freire-Maia et al. (28) confirmed an association between the impact report on the social and emotional domains of $CPQ_{8-10}$ (44) and the occurrence of severe TDI cases.

It can be concluded that even using different measurement tools, severe TDI is associated with a negative impact on quality of life in children, especially in the psychosocial dimensions. Although there are an increasing number of studies on the subject, there is still much to be researched to generate sound scientific evidence to establish both clinical protocols that include the impact gauged from the perception of patients and their families, and for the generation of appropriate public policies to address the problem. Subjective health perception cannot exclude clinical assessment, but they should be combined in order to propose a rational treatment including functional, social and psychological aspects.

## Stages of psychological development

An understanding of the different stages of psychological development is crucial to the success of an intervention, thus reducing the impact of TDIs and associated events.

### Toddlers

Toddlers aged about 1.5 to 3 years are developing a sense of doing things on their own. The child has a very strong drive to investigate the boundaries set by adults, to try out his or her own power and abilities, and to experience new, thrilling situations (51). Sometimes, toddlers exceed the limits of their capacity and expose themselves to a trauma. A traumatic experience such as intense pain puts limits on the

child's expanding world (52). Furthermore, the experience of pain is imprinted on the mind of young children (53, 54).

### Later preschool childhood (3–5 years)

During later childhood, aged 3–5 years, children develop a clear concept of themselves as 'me', knowing they are a separate person and no longer dependent on their parents in familiar situations. Development brings the child into a world of magic, oscillating between reality and fantasy. Intellectual growth is dominated by the increasing use of symbols, the most important of which is language. Words become loaded with feelings, ideas and associations and help the child to create an understanding of the world that is not strictly logical, but holds a deep meaning for the child (55). The child internalizes the parent's guidelines for right and wrong and, during this process, the child gradually develops a conscience and an inner representation of the values and rules that parents represent (8). Having a conscience means feeling guilt when doing the 'wrong thing'. In addition, children may feel guilt even if they are not responsible for the TDI event. For example, a child may imagine he or she caused the painful incident by being angry with a parent that morning. A feeling of guilt may intensify the psychological impact. Furthermore, at this age, children perceive the body as a bag containing feelings, tears, food, a heart and blood; it is frightening if there is a hole in the bag so it has to be mended as soon as possible. This is the 'age of adhesive plaster'.

### School age (6–12 years)

School age (6–12 years) is a period of life characterized by intense development in social skills and cognitive growth. Intellectual development in the period has been described by Piaget as concrete, operational thinking, meaning that the child develops the ability to 'make up things in its head', starting from his/her own knowledge and experience (55). This is, however, limited and might lead the child to unrealistic conclusions. Logical ability increases and, therefore, accuracy is needed when explaining something to the school-age child. A very strong sense of justice is accompanied by a strong wish to punish the wrongdoer. This punishment could also be directed to the self, if he/she is the one blamed for the TDI event. Around 9 years of age, the child reaches an adult conception of life and death, namely, a full understanding that everyone who lives will also die, including oneself. This leads to a deeper understanding of the transient nature of life and might cause an easily evoked fear of death and illness (56). Such a fear could be triggered by a TDI event associated with intense pain.

### Adolescence (13–20 years)

Adolescence (13–20 years) is the final period of the child's development into adulthood. Intellectually, the young person is capable of formal operations (55). This means, for example, using double symbols (as in mathematics) and handling hypothetical facts. Thus, an adolescent has adult intellectual concepts, but less experience than an adult. Teenagers often experience mood swings, as they are trying to achieve a stable inner identity and self-esteem. This is a process that goes on for years and takes great energy. Emotionally, the teenager will pass through different periods, the first of which is dominated by regression, as the wish to remain an innocent child clashes with the need to grow up. The next period is typically dominated by easily provoked aggression, when all earlier self-evident values and norms have to be questioned. The last period also represents the final emotional separation from parents and the security of childhood, in which there is loss and often some degree of depression until the young person finds a new sense of belonging (10). A traumatic experience with intense pain during this period of development may exaggerate any expression of regression, aggression or depression.

## Implications for treatment

The majority of dental injuries occur in children, with approximately 80% of all TDIs taking place before the age of 19 years (6, 57–59). Most injuries are minor, but problems ensue in a limited number of cases with the patient seeking care due to complications with bleeding, pain and esthetics (5).

Accidents involve stress and anxiety for both children, their parents and other adults. It is therefore important that the dental staff are well prepared. The psychological impact of a TDI event and the pain associated with the injury may be even more serious than the physical damage, with the treatment and prognosis of a TDI being an additional source of psychological stress. The assessment of treatment needs and the treatment plan must reflect psychological factors in order to prevent undesirable treatment outcomes. In a 15-year follow-up study, 25% of the patients remembered the demeanor of the dental team, and most of the patients did not remember having received any information about the prognosis for the traumatized teeth (60).

TDIs can have a negative impact on quality of life due to pain, difficulty chewing and appearance, which may have an effect in social situations. The goal of all trauma therapy should be to restore function and esthetics as far as possible so that the patient obtains a good quality of life. Studies have shown that treatment of TDIs in emergency care services is often inadequate, with the unsuccessful emergency care affecting the outcome (61, 62). Thus, there is an urgent need to improve the quality of care of TDIs.

## Treatment assessment

Traumatic dental injury is relatively common, especially in children, and is a stressful experience in its physical, emotional and psychological aspects, resulting in a negative impact on quality of life (28). The fracture of a front tooth can affect children's behavior (32) and school performance (24),

and affects also the families (30). The demand for treatment of TDIs is growing in dental offices, clinics and hospitals (28) and involves a larger number of children and adolescents than adults, because their etiology involves exposure to recreation activities, games and contact sports (23).

Epidemiologic studies indicate that the most common types of injury are fractures of enamel and enamel and dentin (63, 64). It is important to remember that such lesions may need treatment or a follow-up protocol to monitor possible future effects on the teeth, bones and soft tissues. In the case of TDIs, unlike in other parts of the body, the healing processes can last several years, and the sequelae can appear several years after the injury incident (64).

Proportions of treatment need as high as 66.7% have been reported both in developed countries (65, 66) and in developing countries (50, 64, 67), indicating a great neglect in the treatment of TDIs. The reasons for such neglect are unclear. One could suggest that, in developing countries, the majority of the population cannot afford private dental treatment, and the public services are unable to offer more complex treatments. Nevertheless, high levels of untreated traumatized teeth were found in developed countries as well, such as the UK (65), where quality public dental services are accessible to the majority of the population. One factor that could result in low rates of treatment is related to the fact that a TDI is not a disease, and parents might not pay it sufficient attention. This could be related to the severity of the TDI. As the majority of TDIs affect only the enamel and enamel and dentin (63, 64), this could have a lower negative impact on children and their parents. Another factor could be the dentist's lack of knowledge regarding the treatment of TDIs (68, 69). The great variability of methodologies used for its classification, and a lack of standard treatment protocols and follow-up to assess TDI sequelae, could also contribute.

On the other hand, a systematic review of outcome measures used in clinical trials of treatment interventions following TDI has shown that outcomes reported were predominantly the injury's related activities and physical consequences (70). The authors concluded that there is a clear need to establish a more consistent and wider range of outcomes, including patient-based outcomes, adverse effects, family quality of life outcomes and health resource utilization (70). It is also important to remember that children are not independent beings, and family and friends play important roles in children's relationships and feelings, markedly affecting children's perception of quality of life.

These findings corroborate those by Cohen and Jago (71), who were the pioneers in identifying the psychosocial impacts caused by oral health problems. In 1976, they identified that oral health conditions can interfere in people's daily lives and their well-being.

Therefore, in determining the need for treatment due to TDI, OHRQoL measures, in addition to normative clinical indicators, are fundamental to assess treatment needs. As TDIs affect mainly children, indicators such as the Child Perception Questionnaire (CPQ) (44), Child Oral Impact on Daily Performance (C-OIDP) (72) and Early Childhood Oral Health Impact Scale (ECOHIS) (73) are good examples.

## Treatment plan

### Injury event

Everyone involved at the scene of an accident should act in a professional manner. Too many people surrounding the injured person should be avoided and the person situated closest should manage communication. If the injured person is irritated or stressed, it is important for the helper to stay calm, to avoid inducing more stress. Major emergencies requiring ambulance services have well-trained staff for the situation, while minor accidents often receive the aid of the public, who generally are less knowledgeable and experienced.

### Emergency treatment

Waiting in an emergency room can often be stressful and cause anxiety, especially if exposed to the frightening sight of other emergency patients. Many accidents occur on evenings and weekends with emergency waiting rooms full of distressed people. Optimally, the environment of the waiting room should be friendly and adapted for different ages; a quiet, peaceful place with age-appropriate furniture and books or magazines for distraction (Fig. 9.3).

Trauma treatment by dental staff is often short and intense, with the environment and situation new and overwhelming for the patient. The emergency treatment room is also important and should be a suitable, quiet place with possibilities for distraction (Fig. 9.4).

First and foremost, informing the patient (and parents) that help is forthcoming is vital. Treatment is aimed at giving the best possible care during procedures, which patients may perceive as painful, unpleasant or even frightening. Pain is one of the symptoms that patients fear most and trauma often means acute, intense pain. It is important to inform the patient and parents of alternative ways to help reduce pain before, during and after procedures. All treatment of trauma-injured teeth should be as pain-free as possible and the dental team should strive to diminish discomfort during treatment. Mental factors such as stress can cause physical symptoms and, therefore, providing a warm and empathetic environment may reduce the pain experience (74).

### The child patient

#### *Age of the patient*

Children as patients need to be cared for according to their age and maturity. Based on individual circumstances and abilities, children should be introduced to the new elements and instruments as required for the treatment. The child's cooperation and participation is best achieved by communicating at the relevant level for that child's age and maturity.

#### *Parents*

Children generally experience less stress and anxiety if a parent is present, but the parent's behavior also affects the

**Fig. 9.3** The waiting room should be a quiet, peaceful place with age-appropriate furniture and books or magazines for distraction.

**Fig. 9.4** A quiet place with possibilities for distraction after sedation or treatment.

child's experience (75). Well-functioning interaction within the dental team will provide an extra sense of security. After being thoroughly informed, children and parents should participate in decisions concerning treatment. The parent's view on how the child reacts in stressful situations should be considered by the dental team (76–78).

Parents often perceive the situation of TDIs as stressful and may need information on how they can best support their child. Although initially worried, parents can usually sooth their child if they themselves are given support and information. In rare cases, the child may instead accept support from a person in the dental team.

### *The dental team*

Establishing a good relationship between the dental team and the patient before treatment includes giving the child a feeling of control. There is a strong link between a child's experience of pain and anxiety, its ability to handle stress during procedures and the behavior of the parents and the dental team (79). The well-being of the child is important in the trauma situation. It is imperative that the dental team shows empathy and commitment, and provides tailored treatment. Offering a warm, empathetic environment may reduce the patient's pain experience. If the dental team takes the time to talk to the child in a friendly and age-appropriate way, anxiety can be greatly alleviated (80). McMurtry et al. (81) showed that, in particular, facial expression (afraid or happy), but also voice mode (up or down), affects the child's level of stress and fear.

## Preparation of the patient

One feature common to all patients with acute trauma is the fact that they come to the dental clinic unexpectedly. It is important to handle the acute situation in a way that makes the patient feel secure and protected so as to avoid or limit the psychological secondary effects of the physical trauma.

To prepare the child and parents psychologically, treatment should be preceded by age-appropriate information and the child and parents should be given time to process it. Fear and anxiety are common in all age groups and it is important to discover why the child is fearful. As far as possible, introduce the child to the techniques and instruments that the emergency situation requires, explaining through cooperation and participation. TDIs do not always allow time for adapting to the circumstances, but it is important that information is given honestly and empathetically, allowing the child to take part in decisions when possible. Honest explanations allow for a sense of security, enabling the patient to gain confidence in the dental team. Making the child feel involved gives him or her a sense of control and security. Children may grow with the experience for future treatments if they feel they handled the situation well (77, 78, 82–84). Encouragement should always be given, even if the treatment does not go as planned.

## Talking to children

The dental team needs to be aware of and control their emotions in relation to the patient. Even if affected by the situation, the dental team must not express these feelings in front of the child or the parents. As far as verbal expression is concerned, most professionals are trained in self-control. However, it is important to remember that body language expresses just as much as words, and for the youngest children it is even more important than words.

### *The infant*

To communicate with an infant means to mainly 'talk with the body', and the young child 'talks' back in the same way. Words by themselves mean nothing. What counts are eye contact, mimicry and voice pitch. The child answers by smiling, crying, yelling, moving away or showing interest, or by imitating (85). The young child perceives all this, whereas words will be directed to and understood by the parents.

### *The toddler*

The toddler is still very dependent upon body language in their communication and, in particular, if words are contradictory, they will rely upon body signals rather than words to understand the message. Words become more important as children get older and act as an important support for the child's memory (55). During this period of life, children love to imitate, for example, animals, and to identify themselves with familiar persons. This offers a way for the dental team to make contact. Children want to feel security and continuity. They want things to be repeated and done the same way every time. Parents should be asked about the child's specific rituals, which animals or people or cartoon figures they prefer to imitate or with which they identify. This might be helpful in establishing contact and making the child cooperate. It is important to remember that the child is still very dependent upon the parents to understand and handle the environment around him/her.

### *Later preschool childhood*

In later preschool childhood, there is still a great dependency upon parents, but the child has reached a concept of self as 'me' (9). Their verbal capacity has increased, which adds to the child's self-assurance in new situations and with unfamiliar people. It is important to remember that a child in crisis and pain will not be able to handle the situation as under normal circumstances (this holds true for all ages). The child likes to use new adult words, but does not always understand the exact meaning. Thus, it is crucial to ask the child: 'What do you mean exactly?' Praise the child for whatever he/she achieves. Four-year-olds will be happy to try to follow instructions as long as they feel the efforts are appreciated.

### *School age*

During school age, children gradually achieve an adult way of handling language; they talk correctly and are logical. Still, there is a lack of experience and a certain rigidity in their thinking, which might lead to false conclusions (55). Therefore, it is preferable to ask questions to find out exactly what they are thinking, what ideas they have about the outcome of treatment and the procedures involved. Always

answer their questions honestly. It is important to make sure that the school age child has a correct explanation for anything they may ask. If the dentist is not honest, the child's confidence in them will probably be lost for ever, or at least for a very long time. Listen carefully to understand the child's real worries, and attempt to respond to their thoughts. Sometimes one might need to use adult technical terms; if so, take care to explain them precisely. Making a 'contract' might be a way to solve a critical situation during treatment. This will usually be made between three parties: the child, the parent and the professional (52). To make sure the child has fully understood the information given, it would be wise to ask the child to talk about it. Instead of just asking the child to repeat the information given, the dentist can say, for example: 'Your best friend will surely want to know all about what happened and what will come next. How can you explain this?'

### *The teenager*

During adolescence, the teenager expects to be treated like an adult and also has the intellectual capacity to discuss and think like one (55), although the dental team must remember that mood swings are common and there is a lack of experience that might make things seem and feel worse. Thus, the dentist has to be patient and show understanding of the teenager's thoughts and feelings using the same techniques as when talking to adults. Arguing is typical for this age group and understanding should be shown, but what can and cannot be done needs to be clarified.

## Treatments for procedural pain

Treating pain and suffering should be a priority for all clinicians. TDIs can be frightening and painful with consequences for the child's future pain management. In a 15-year follow-up study, 21% of DTI patients remembered pain during the trauma treatment. The importance of painless emergency treatment of children with DTIs, and proficiency in traumatology, was stressed in the study performed by Robertson and Norén (60). The International Association for the Study of Pain (IASP) defines pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (86). Pain is always subjective and should never be questioned. This implies that only the patient can decide whether a clinical procedure is painful or not. There is evidence showing that fearful children report a higher intensity of pain during dental procedures than children not feeling dental fear, i.e. fear lowers the pain threshold (87, 88).

If the child patient is not properly prepared and informed about the upcoming procedure, at an individual cognitive level, or if he or she expects pain, the child may refuse to cooperate during further care (89–91). Many studies have shown that pain in children is underestimated and undertreated (92–94). The child with previous experiences of pain from health care and dental care may react with a flight reaction in a new dental situation even without pain occurring (95).

There are practical as well as *humanitarian reasons* for treating pain. Children of all ages feel pain and require analgesics during painful procedures (96). The assumption that small children do not perceive or remember a pain experience is incorrect (97); it has often been argued that small children cannot experience, perceive or remember pain like adults since their nervous system is immature (98). The last 30 years of pain research in children has come to the conclusion that these beliefs are completely untrue (99). It has also been argued that pain in small children has no adverse long-term effects, which has been proven to be incorrect.

Anticipation is the key to managing pain from dental procedures. Effective pain management involves a combination of pharmacologic, psychological, cognitive behavioral and physical treatments. Pain adds significantly to both physiologic and psychological stress. The treatment offered during procedural pain varies from non-pharmacologic (child-centered approaches) – such as distraction, muscle relaxation, paced breathing, modeling and cognitive behavioral methods – to pharmacologic (disease-centered approaches) – such as analgesics in various forms of administration, conscious sedation, inhaled nitrous oxide and local and general anesthesia (52).

There is consensus that psychological interventions are generally beneficial in medical procedures in children (100). Psychological care concerns minimizing stress and discomfort while strengthening and providing methods for the child's own stress management. The selection of methods should be based on clinical judgment of how vulnerable the child may be, and must be done in collaboration with the child and the parents (52).

Distraction can be used after the patient is aware of what will happen and how the treatment will be carried out. Distraction is also an effective method in reducing procedure-related pain in children and young people during less painful procedures, meaning that attention is focused on something other than pain. Distraction techniques can be quite simple, such as reading to the child, watching TV, listening to music or talking about anything other than the procedure.

The need for disease-centered approaches must be estimated based on both the nature and extent of tissue damage, and the vulnerability of the child. When fear, worry or anxiety is expected, premedication and/or nitrous oxide should be offered to the patient, especially when treatment steps are demanding or stressful. Conscious sedation is a useful method for managing uncooperative or fearful children who need dental treatment and can aid carrying out a procedure, but can never replace proper care or sufficient pain relief. The use of sedatives alone during painful procedures does not provide analgesia, but makes a child less prone to being distressed. In all sedation, safety is paramount due to the sedative effect, the impact on respiratory function and the interaction between drugs that may involve potential side effects, such as respiratory depression (52, 101–103). General anesthesia should be considered if the procedure is invasive and the patient is not expected to cooperate during treatment.

It is important that pain is adequately treated for its duration to avoid unnecessary suffering. Immediate and competent pain control using paracetamol and/or non-steroidal, anti-inflammatory drugs (NSAIDs) should be

offered from early on at the emergency ward to give the patient relief (104). At the dental clinic, there should be clear procedures regarding general analgesics.

Treatment should be carried out with a minimum of pain and discomfort, which requires adequate local analgesia. However, it is not possible to guarantee completely pain-free treatments. Intraoral topical analgesics and local analgesic solutions should always be used in invasive procedures in the primary and permanent teeth and the mucous membranes. Topical analgesic agents are extremely important for reducing pain during intraoral injections and should be routinely used. They are available as sprays, gels, ointments or solutions (52). There is evidence of topical analgesics being beneficial in reducing pain during needle puncture (105, 106). The use of local analgesics is the most important method in achieving pain relief. Proven techniques that inflict the least amount of pain to the patient should be used, e.g. a slow injection and transpapillary injection should always precede the palatal/oral injection (52). A lack of pain management may also have immediate adverse medical implications due to uncontrolled pain causing the release of local and general stress hormones, which increases tissue damage and prolongs the healing process (107).

Measuring pain is an important factor in improving the care of children. Pain evaluation is complicated since the experience of pain depends on many variables consisting of both physiologic and psychological factors. Therefore, self-reporting is the gold standard according to McGrath et al. (108, 109). The aim is to provide acceptable pain levels for the child undergoing dental treatment. Pain analysis entails a methodical approach to identifying and analyzing the elements of that experience. The assessment should assume that pain is subjective and therefore should be assessed on the basis of each individual. When a child is communicating about pain, it may have difficulty distinguishing between intensity and discomfort and, therefore, pain cannot be measured unambiguously. Measuring pain should be based on the following components: verbal reports, direct painful expressions, physiologic expressions and parental involvement (110).

Part of the assessment is to measure the pain intensity with a validated pain measurement instrument. Several different instruments for the assessment of children's pain have been published. Children do not have the same understanding of graded scales as adults do. Self-assessment for children depends on the child's age, development, and cognitive and verbal maturity. To use a self-rating scale, the child should be able to grade and estimate the pain and match it with a graded scale (111). Given below are proposed instruments that are essentially one-dimensional and are clinically useful. These provide opportunities to detect pain, document pain and optimize pain management.

### *Verbal reports*

The following are used in the assessment of pain in children >6 years of age.

- *Horizontal scale*. School children and teenagers can use the one-dimensional, horizontal Visual Analog Scale (VAS), graded from 1 to 10. Number 1 represents free from pain, and number 10 the worst pain imaginable. VAS has one side for the patient and the other side for the staff, graded to simplify reading. The patient points to or moves a marker of the scale and the staff reads the scale on the back, where a value between 0 and 10 is obtained. This scale is well known and validated (Fig. 9.5).
- *Vertical scale*. A vertical VAS, the Colored Analog Scale (CAS), has been developed for children aged 5–16 years. On this scale, height, width and red color degree indicate the intensity of pain. Studies have shown that younger children find it easier to handle this compared with the VAS scale (108) (Fig. 9.6).
- *Verbal descriptive scale*. Pain is estimated by the patient using an adjective to describe the pain intensity. A verbal scale should be displayed so that the patient can read the different options. The staff then grades the pain from 0 to 5 as below:

| *Self-assessment* | *Grading* |
|---|---|
| No pain | 0 |
| Mild | 1 |
| Unpleasant | 2 |
| Difficult | 3 |
| Terrible | 4 |
| Intolerable | 5 |

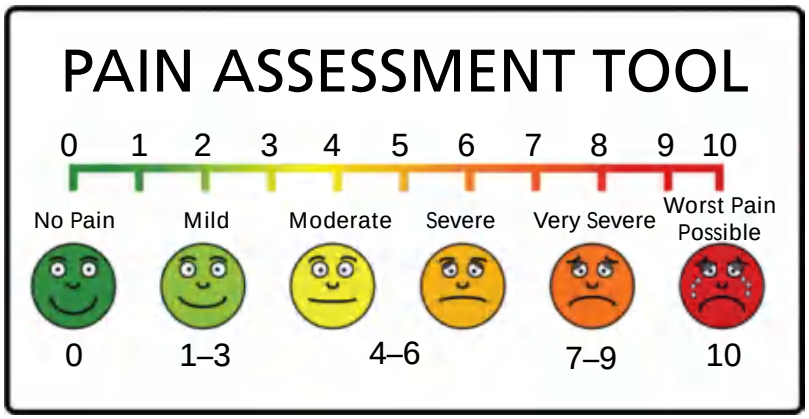



**Fig. 9.5** Horizontal scale. School children and teenagers can use the one-dimensional, horizontal Visual Analog Scale (VAS), graded from 1 to 10. Number 1 represents free from pain, and number 10 the worst pain imaginable. A variation on the visual analog scale is the Faces Pain Scale, which is considered a reliable facial scale for children >4–5 years of age.

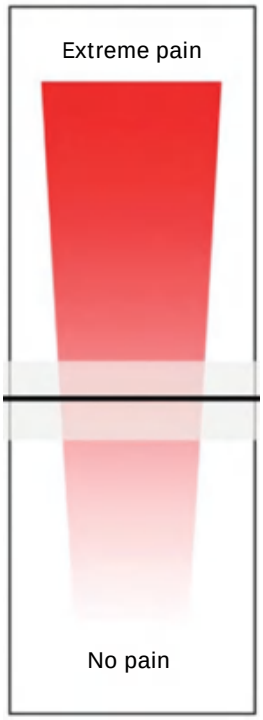



**Fig. 9.6** Vertical scale. A vertical VAS, the Colored Analog Scale (CAS), has been developed for children aged 5–16 years. On this scale, the height, width and degree of red color indicate the intensity of pain.

One of the limitations is associated with the use of self-report measures with young children, particularly those under 5 years of age. Children younger than 3 years of age are unable to quantify pain using self-report measures. A variation on the VAS is the Faces Pain Scale (Fig. 9.5), which is considered a reliable facial scale for children >4–5 years of age. A weakness of facial scales is that they appeal to the child's emotional state of mind. The younger the child is, the harder the child has to distinguish pain from uneasiness and fear. Therefore, it is important to present the scale so that the child understands that it is pain that is being assessed (112).

The child needs to be introduced to the pain assessment scale and to understand how self-assessment is done. Pain assessment instruments should always be shown before they are used. Children and parents should be informed that a self-assessment scale will be used to find out if the child has experienced pain to help with pain relief after treatment. It is important to form an opinion of the whole situation surrounding the child. Are there factors in the dental environment affecting the child's experience of pain? Is the child sad or worried? The assessment should not begin by asking whether the child is in pain, but rather by asking if the child is feeling anything from the injury or the treatment. If the child confirms pain, questions should be asked regarding how much it hurts, where it hurts and what hurts. Pain assessment should be done with the help of an instrument that is tailored to the child's level of development, with the assessment completed by the parents' interpretation and assessment of the child's pain.

Evaluation and documentation should be included in the medical file to facilitate any next treatment (113).

For more complicated situations regarding pain, the one-dimensional measuring instrument is often insufficient. More complex instruments will be needed to assess pain quantity and quality.

General analgesics should be prescribed for the next few days as the child is likely to feel pain post treatment (52). The treatment of acute postoperative pain changes over time and the goal is to make the patient as comfortable as possible. Depending on the intensity of pain and its quality, administering general analgesics should be based on a step-down approach with stronger pain medicine given immediately after treatment, since pain from dental trauma injuries can be very intense at the beginning. As recovery progresses, a step down to weaker pain relief medicine should be followed.

## The adult patient

In many ways, adult patients are treated in the same way as the child patient, but taking age into account becomes less important. It is, however, important to be attentive to patient preferences and previous dental care experiences, with signs of dental fear seriously considered. Allowing the patient the opportunity to participate in the planning and treatment, and tailoring information to the patient's ability to absorb the information, as well as continuously informing what transpires during treatment, shows consideration and respect. Pain relief and treatment procedures are the same as for the child patient. Since good communication varies among caregivers, some basic principles can be defined such as integrity and flexibility in language. Furthermore, the patient's mood and personality should be taken into consideration in order for communications to be understood by the patient.

## Discussion post treatment

Post trauma treatment information is an essential part of the treatment plan and will decrease anxiety significantly if properly given. Post procedure discussions allow the patient to ask questions about what transpired and to clarify things that might have been misunderstood in the acute situation. If the patient is a child, it is important to give credence to the child's thoughts and to answer honestly. Some children will need active encouragement from the dental team to start talking. This post procedure dialogue could be done as play with the youngest children. Information should be given in a concise and positive manner, verbally as well as written, emphasizing the likelihood of recovery and explaining post treatment pain as:

- Common after dental trauma injuries
- Important to treat
- Subjective and variable during the day
- Subsiding gradually

## Follow-up treatments

The same principles as those mentioned earlier regarding the emergency situation are relevant during follow-up treatment. Information given in a stressful situation may not have been

fully understood by the patient (60), therefore it is necessary to repeat the information and to check that it has been fully understood. Written information and opportunities for the patient or parents to ask questions should be given more than once. The follow-up treatments should be carried out with appropriate use of conscious sedation and local anesthetics and other substances for reducing pain and tension. This is especially important in young children who do not fully understand the reason or need for dental treatment. The patient should be adequately informed about the reasons for follow-ups (identifying healing complications) and the necessity to perform certain procedures (e.g. endodontics, extractions).

### Economic consequences

The patient should be informed about means of reducing a possible economic impact of the TDI (i.e. support from public or insurance companies).

## Essentials

- Dentofacial disfigurement may have social and psychological implications. In most cultures, the face is regarded as the most precious characteristic of human identity and therefore it enjoys a privileged status in relation to the rest of the body.
- Any deviation from the 'norm', such as a dentofacial disfigurement, may stigmatize a person and make that person less acceptable socially. Deviation from the 'norm' can be as simple as a dental anomaly or as complex as a craniofacial deformity.
- A smile is an essential feature both for children and adults. A traumatic dental injury may affect the appearance due to a fracture, discoloration of teeth or avulsion of teeth.
- Oral health influences how people look, speak, chew, taste and enjoy food. Thus it influences how people socialize, their self-esteem, self-image and their feelings of social well-being.
- The magnitude of the psychological impact will depend on the type and severity of the injury, type of event associated with the injury, level of pain and fear, and quality of treatment provided.
- The trauma event in itself might have serious psychological effects. Keep in mind that even the youngest child will remember. Negative effects could be reduced by means of good emergency care. The dental team must be aware of and understand the anxiety of both children and parents. Awaiting treatment is a stressful experience for children, especially if they are also exposed to the frightening sight of other emergency patients and are in unfriendly surroundings.
- Give information in words adjusted to the child's level of development. This will relieve worries and feelings of guilt and increase cooperation. Listen to the child and try to find out how he or she thinks and feels about the accident, the treatment and the outcomes. The dental team must have the knowledge and skills to make an accurate diagnosis and perform appropriate and prompt emergency treatment.
- Pain adds significantly to both physiologic and psychological stress. This means that immediate and skillful pain control should be given very early at the emergency ward or if possible at the place of injury.
- Lack of postoperative information might add to emotional stress during emergency treatment as the child anticipates future problems. Give information in as concise and positive a manner as possible, emphasizing the likelihood of recovery and also give written information.
- Post-traumatic follow-up gives an excellent opportunity to talk the whole treatment through; from the moment of the injury until the expected end result. This will reduce negative feelings about dental care.

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# 10

# Child Physical Abuse and Neglect

R. R. Welbury & A. M. Cairns

## Definitions

A child is considered to be abused if he or she is treated in a way that is unacceptable in a given society at a given time; this is underpinned by the legislation applicable to that country. This definition takes into account how our tolerances for certain behaviors have changed over time but also helps us to realize that due to cultural diversity not only are children treated differently in different countries, but also within different societies of the same country.

Hopefully now in the 21st century we are starting to move away from the constraints of cultural and geographic variations in what may be perceived as a need for child protection, a change which began with the United Nations Convention on the Rights of the Child in 1989 (1). Since then many countries have legislated on matters of child protection and in many countries the role of the dentist in child protection has been given more clarity.

There are several categories in the child abuse spectrum, namely: physical abuse, sexual abuse, emotional abuse and neglect which may be physical and/or emotional. It will be obvious to the reader that most maltreated children suffer under multiple categories; for example, it is impossible to see how a child who is physically or sexually abused would not also suffer emotional abuse.

With regard to the role of the dentist in child protection it is clear that within daily practice all forms of abuse may be detected and action to prevent further harm should be taken. Although the subject of this textbook is dental trauma, this chapter will discuss in detail the physical signs of abuse. It will also provide information on dental neglect as part of physical neglect as the role of the dentist in this field is rapidly expanding.

Physical abuse is a form of abuse which may involve hitting, shaking, throwing, poisoning, burning or scalding, drowning, suffocating or otherwise causing physical harm to a child. Physical harm may also be caused when a parent or carer fabricates the symptoms of, or deliberately induces, illness in a child.

Neglect can be defined as the persistent failure to meet a child's basic physical and/or psychological needs, likely to result in the serious impairment of the child's health or development (2).

## Historical aspects

Violence towards children has been noted since early civilization and infanticide has been documented in almost every culture (3). Ritualistic killing, maiming and severe punishment of children in an attempt to educate them, exploit them or rid them of evil spirits has been reported since early biblical times and ritualistic surgery or mutilation of children as part of religious and ethnic traditions continues today (4).

The industrial revolution in the 18th century saw an increase in the economic value of child labor. Harsh punishment of children was accepted as normal in courts, schools and in the home (5).

In the 19th century, Western societies became more protective towards children, and their lives gradually improved. However, the mortality rate remained high, and the isolated death of a child was unlikely to arouse suspicion. Lord Shaftesbury, who campaigned to create better conditions in Britain for children at work, recognized the problem of child abuse at home, but was powerless to intervene.

As more effective health care became available in developed countries, more children survived poor living and working conditions. But in the middle of the 20th century reports appeared in the USA of unexplained skeletal injury to children (6, 7). These reports described unexpected skeletal

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trauma, sometimes associated with subdural hematoma. The possibility that these injuries could have been inflicted by a parent was recognized but there was a general reluctance to accept that parents could deliberately harm their own children (8). However, when Kempe et al. published their paper 'The battered-child syndrome' in 1962 (9), the full impact of the physical maltreatment of children was brought to the attention of the medical community and the general public. The battered-child syndrome or non-accidental injury (NAI) is now usually referred to as child physical abuse. Its recognition had such a profound effect upon the professions and the public that within a few years the majority of states in the USA had introduced laws which made it mandatory for physicians, dentists and other health-related professionals to report suspected cases. The primary aim of all professionals involved in the child protection process is to ensure the safety and well-being of the child. The secondary aim is to provide support and counselling for the parents or caregivers in order to eliminate the maltreatment.

## Prevalence

Physical abuse is as an international issue and has been reported in many countries (10–20). Despite this global recognition, prevalence rates in most countries are still not available. An estimated 679 000 children in the USA were documented victims of maltreatment in 2013, and approximately 1520 of these children died from abuse or neglect (21); this is a major threat to children's mental health (22, 23). Over a 5-year period in Britain most social service and child welfare departments noticed a 40% increase in reporting of suspected child abuse (24).

It is now a widely held belief that what is reported in respect to child maltreatment may just be the tip of the iceberg, with huge numbers of suffering children remaining undetected. In Britain, at least one child per 1000 under 4 years of age per year suffers severe physical abuse, for example fractures, brain hemorrhage, severe internal injuries or mutilation (25). In the USA more than 95% of serious intracranial injuries during the first year of life are the result of abuse (26). Studies analyzing the attendance of children in accident and emergency departments of hospitals have shown that in the USA 10% of children younger than 5 years (27), and in Denmark 1.3 children per 1000 per year (28), will have injuries that were willfully inflicted. Each year as many as 1520 children in the USA (29) and 67 children in Britain (24) will die as a result of abuse or neglect. In Scandinavia, the estimated number of deaths from physical abuse is much lower, at about 10 annually, giving an estimated frequency of 0.5 child deaths per million inhabitants per year (30). A common finding in all countries is that the workers concerned with child abuse believe that many cases remain undetected and the real mortality figures are considerably higher.

Children of all ages are subject to physical abuse, but the majority of cases occur in younger children (31). This is partly because they are more vulnerable and partly because they cannot seek help elsewhere. Children under 2 years of age are most at risk from severe physical abuse. Death from abuse is rare after the age of 1 year. The number of boys subjected to violence slightly exceeds that of girls; and first-born children are more often affected. Within a family, it is not uncommon for just one of the children to be abused and the others to be free from such abuse.

## Etiology

The etiology of physical abuse is based on the interaction between the personality traits of the parents or abusing adult, the child's characteristics and environmental conditions (32). Due to the wide variation in behavioral characteristics, personality traits and psychiatric symptoms among abusive adults, a specific abusive personality does not exist. However, certain commonly recurring traits can be recognized (10, 25, 32–34).

Physical abuse and neglect encompasses all social classes; but more cases have been identified in the poorer socioeconomic groups. Many cases of physical abuse or neglect are by the child's parents or by persons known to the child (31). Often the mother of the affected child may be divorced or single. It is also common for a co-habitant who is living in the home, but who is not related to the child, to be the perpetrator. Young parents, often of low intelligence, are more likely to be perpetrators of abuse or neglect. This is especially true if they have been exposed to such behavior during their own childhood (35). Indeed, abuse is thought to be 20 times more likely if one of the parents was abused as a child (25). A significant proportion of the perpetrators have a criminal record of some kind; and although they usually do not have an identified mental illness, they may exhibit personality traits predisposing to violent behavior. Contributing factors to abuse by the adults involved include alcohol and other drug abuse, poverty, unemployment and marital problems. Children already at risk may add to the stress by continually crying, throwing tantrums or soiling their clothes. In addition, the child may be handicapped, be the result of an unwanted pregnancy or may fail to attain the expectations of the parents. These factors can provoke frustration in the most stable parent. But in association with other stresses, they may lead to physical neglect or injury.

## Diagnosis

It is one of the roles of the dentist to recognize the possibility that an injury in a child may have been caused on purpose. The diagnosis is a difficult intellectual and emotional exercise, and is one of the most consuming tasks for a pediatrician, requiring time, experience and emotional energy. Failure to spot the signs and make the diagnosis can vitally influence a child's future life. At worst, it is a matter of life or

death for the child. Short of death, there may still be possible brain damage or handicap. Whilst it is not the role of dentist to ultimately diagnose and investigate child abuse, it is essential that they pass on any suspicions.

Physical abuse is not a full diagnosis; it is merely a symptom of disordered parenting. The aim of intervention is to diagnose and cure (if possible) the disordered parenting and abnormal family dynamics. It is not the intention to take children away from their natural parents unless there is serious risk of physical injury. In practice only about 1–4% of all children who are the subject of referrals are taken out of the home to a place of safety. In the 1970s it was estimated that in the USA 5% of abused children who were returned to the home environment without some form of intervention died following further trauma (36) and 35–50% sustained serious re-injury (36, 37). However, by 1986 it was recognized that probably as many as 50% of severely abused children returned to the abuser would die of recurrent abuse if proper therapeutic measures were not introduced (38). In some cases, the occurrence of physical abuse may provide an opportunity for intervention. If this opportunity is missed, there may be no further opportunity for many years.

There are no hard-and-fast rules to make the diagnosis of physical abuse or neglect easier (39). The following list constitutes seven classic indicators to the diagnosis. None of them is pathognomonic on its own; neither does the absence of any of them preclude the diagnosis of physical abuse (40).

(1) There is a delay in seeking medical help (or medical help is not sought at all).
(2) The story of the 'accident' is vague, is lacking in detail and may vary with each telling and from person to person.
(3) The account of the accident is not compatible with the injury observed.
(4) The parents' mood is abnormal. Normal parents focus their attention on the child and what is to be done to help them. Abusive or neglectful parents may be preoccupied with their own problems and issues and do not focus on the child.
(5) The parents' behavior gives cause for concern – for example they may become hostile and rebut accusations that have not been made.
(6) The child's appearance and the interaction with the parents are abnormal. The child may look sad, withdrawn or frightened.
(7) The child may say something concerning the injury which is in contradiction to what the parents have told you.

## Types of orofacial injuries in child physical abuse

At least 50% of cases diagnosed as child physical abuse have orofacial trauma, which may or may not be associated with injury elsewhere (10, 41–46). Although the face often seems to be the focus of impulsive violence, facial fractures are not frequent. The most detailed studies were those reported by Becker et al. (1978) (43), Da Fonseca et al. (1992) (44), Jessee (1995) (45) and Cairns et al. (2004) (46). In Becker's study the medical records of 260 cases of child abuse admitted to The Children's Hospital in Boston between 1970 and 1975 were reviewed. Of these patients, 128 (49%) had facial and/or intraoral trauma. An additional 16% of the children had injuries to the head, such as skull fractures, subdural hematomas, contusions and lacerations of the scalp. Of the 236 injuries sustained by the orofacial structures, 61% involved the face (66% contusions and ecchymoses, 28% abrasions and lacerations, 4% burns and bites, 2% fractures) and 6% involved the intraoral structures (43% contusions and ecchymoses, 28% abrasions and lacerations, 28% dental trauma). In Da Fonseca's study the number of cases was higher (1248) but the spectrum of injuries was similar. The incidence of orofacial signs in physical abuse was 78%, sexual abuse 13%, and neglect 24%. In all types of abuse the average incidence of orofacial signs was 34%. Jessee's study, undertaken in Texas, found that out of 266 patients with physical abuse some 65% had orofacial injuries, and Cairns et al. in Scotland found a figure of 59% with orofacial injuries in a similar population of 390 children. In all the studies it was found that soft tissue injuries, most frequently bruises, were the most common injury sustained to the orofacial structures in physical abuse.

It is extremely important to state that there are no injuries which are pathognomonic of child abuse. Any text that suggests so is incorrect.

### Bruising

Accidental falls rarely cause bruises to the soft tissues of the cheek, but instead involve the skin overlying bony prominences such as the forehead or cheekbone. Inflicted bruises occur at typical sites and/or fit recognizable patterns (28) (Table 10.1). The clinical dating of bruises according to color is inaccurate (47, 48). However, bruises of different vintage indicate more than one episode of abuse and, together with extensive bruising and a history of minimal trauma, are very suggestive of physical abuse (Fig. 10.1).

Bruises on the ear are commonly due to the child being pinched or pulled by the ear (Fig. 10.1), and there will usually be a matching bruise on the posterior surface of the ear.

Bruises or cuts on the neck are almost always due to being choked or strangled by a human hand, cord or some sort of collar. Accidents to the neck are extremely rare and should be

**Table 10.1** Typical sites for inflicted bruises.

| |
|---|
| Buttocks and lower back (paddling) |
| Genitals and inner thigh |
| Cheek (slap marks) |
| Earlobe (pinch marks) |
| Upper lip and frenum (forced feeding) |
| Neck (choke marks) |

A B

**Fig. 10.1** Three-year-old boy suffering from physical abuse. Note bruising of different vintages involving the skin overlying the bony prominences of the cheekbones and the soft tissue areas of the cheek and circumoral region (A). This history was inconsistent with the amount, degree and vintage of the bruising. The 'pinch' type bruises on the superior surface of the right ear (B) was a result of the ear being held between the fingers and thumb of the abuser and then pulled.

looked upon with suspicion. 'Resuscitation' attempts do not leave bruises on the face or neck. Bruising and laceration of the upper labial frenum of a young child can be produced by forcible bottle feeding and is another injury of physical abuse, but one which may remain hidden unless the lip is carefully everted (Fig. 10.2). The same injury can also be caused by gagging or gripping and violent rubbing of the face; and may be accompanied by facial bruising/abrasions. A frenum tear is not uncommon in the young child who accidentally falls while learning to walk (generally between 8 and 18 months). However, a frenum tear in a very young non-ambulatory patient (less than 1 year) should arouse one's suspicion as to the possibility of this injury being non-accidental (49).

**Fig. 10.2** A torn labial frenum and bruising of the buccal sulcus, sustained during forceful feeding.

## Human hand marks

The human hand can leave various types of pressure bruises: grab marks or fingertip bruises, linear marks or finger-edge bruises, hand prints, slap marks and pinch marks. The most common types are grab marks or squeeze marks which leave oval-shaped bruises that resemble fingerprints. Grab mark

**Fig. 10.3** A slap mark bruise. Three parallel linear bruises at finger-width spacing can be seen to run through a more diffuse bruise.

**Fig. 10.4** Tattoo bruising in the right forehead region of the child shown in Fig. 10.1. The exact object causing this injury was not known.

bruises can occur on the cheeks if an adult squeezes a child's face in an attempt to get food or medicine into his or her mouth. This action leaves a thumb mark bruise on one cheek and 2–4 finger mark bruises on the other cheek. Linear marks are caused by pressure from the entire finger. In slap marks to the cheek, parallel linear bruises at finger-width spacing will be seen to run through a more diffuse bruise (Fig. 10.3). These linear bruises are due to the capillaries rupturing at the edge of the injury (between the striking fingers), as a result of being stretched and receiving a sudden influx of blood.

## Bizarre bruises

Bizarre-shaped bruises with sharp borders are nearly always deliberately inflicted. If there is a pattern on the inflicting implement, this may be duplicated in the bruise – so-called tattoo bruising (Fig. 10.4).

## Abrasions and lacerations

Penetrating injuries to the palate, vestibule and floor of the mouth can occur during forceful feeding of young infants; these are usually caused by the feeding utensil. Abrasions and lacerations on the face may be caused by a variety of objects, but are most commonly due to rings or fingernails on the inflicting hand and injuries are rarely confined to the orofacial structures (Figs 10.1 and 10.5).

## Burns

Approximately 10% of physical abuse cases involve burns (50). Burns of the oral mucosa can be the result of forced ingestion of hot or caustic fluids in young children. Burns from hot solid objects applied to the face are usually without blister formation and the shape of the burn often resembles its agent. Cigarette burns give circular, punched out lesions of uniform size (Figs 10.5 and 10.9). Lesions produced by cigarette burns may resemble bullous impetigo, hence it is critical to reiterate the point made earlier that no single type of lesion is pathognomonic of child abuse.

## Bite marks

Human bite marks are identified by their shape and size (Fig. 10.6). When necessary, serological techniques are available and may assist in identification (51). The nature and location of the bite is likely to change with increasing age of the child. In infants, bite marks tend to be punitive and are often a response to soiling or crying. As a result, bite marks may appear anywhere, but they tend to be concentrated on the cheek, arm, shoulders, buttocks or genitalia. In childhood, bite marks tend to be less punitive and more a function of assault or defense. Sexually oriented bite marks occur more frequently in adolescents and adults (52). However, a bite mark at any age should raise the suspicion of child sexual abuse.

A B

**Fig. 10.5** A. Multiple superficial lacerations, extensive facial bruising of different vintages, a cigarette burn of the forehead and a 'pinch' injury of the left ear in a child subjected to repeated physical abuse. B. There are numerous pinch mark bruises over the chest and abdomen, extensive bruising over the suprapubic region and a number of small abrasions and lacerations.

**Fig. 10.6** A human bite mark injury on the right cheek.

The duration of a bite mark is dependent on the force applied and the extent of tissue damage. Teeth marks that do not break the skin are visible for up to 24 hours. In those cases where the skin is broken, the borders or edges will be apparent for several days depending on the thickness of the tissue. Thinner tissues retain the marks longer (53).

## Dental trauma

Trauma either to the primary or permanent dentition in physical abuse can be due to blunt trauma (Fig. 10.7). A similar range of injuries to those found in accidental trauma is seen.

## Eye injuries

Most periorbital bruises caused by child physical abuse involve both sides of the face. Ocular damage in child physical abuse includes acute hyphema, dislocated lens, traumatic cataract and detached retina (54). More than half of these injuries result in permanent impairment of vision affecting one or both eyes.

## Bone fractures

Fractures are among the most serious injuries sustained in physical abuse. They may occur in almost any bone and may be single or multiple, clinically obvious, or occult and detectable only by radiography. Most fractures in physically

**Fig. 10.7** Luxation of four incisors and extensive bruising in the upper buccal sulcus sustained as a result of a blow to the mouth.

abused children occur under the age of 3 years (55). In contrast, accidental fractures occur more commonly in children of school age.

Facial fractures are relatively uncommon in children. They can, however, occur during physical assault, with nasal fractures occurring most frequently (45%), followed by mandibular fractures (32%) and zygomatic maxillary complex and orbit fractures (20%) (56).

The presence of a fracture of the facial skeleton in a case of child physical abuse is an indication for a full skeletal radiographic survey (Fig. 10.8). Generally, the force required to produce a facial fracture in a child is greater than that required to produce metaphyseal, epiphyseal, spiral, oblique and transverse fractures in long bones. A skeletal survey of a child who has suffered child physical abuse may show evidence of multiple fractures at different stages of healing.

In some instances, a number of the varied features mentioned may be present at any one time and the diagnosis of child physical abuse will be clear. However, there are occasions when clinical evidence is inconclusive and the diagnosis merely suspected.

## Differential diagnosis

Although dental practitioners should be suspicious of all injuries to children, they should never consider the diagnosis of child physical abuse on the basis of any one sign, as various diseases can be mistaken for child physical abuse. Impetiginous lesions may look similar to cigarette burns (Figs 10.9 and 10.10), birthmarks can be mistaken for bruising, and conjunctivitis can be mistaken for trauma. All children who are said to bruise easily and extensively should have a full blood count, platelet estimation and blood coagulation studies to eliminate a blood dyscrasia (e.g. leukemia), a platelet deficiency (e.g. thrombocytopenia) or other hemorrhagic disorders (e.g. hemophilia, von Willebrand's disease).

**Fig. 10.8** A fracture of the nasoethmoidal complex caused by a heavy blow to the midface.

**Fig. 10.9** A discrete well-demarcated lesion, typical of the appearance of a cigarette burn.

## The dentist's role in the management of child physical abuse

The dental practitioner may be the first professional to suspect physical abuse as a result of injuries involving the orofacial structures (57–66). The primary aim of all professionals involved is to ensure the safety of the child. The secondary aim is to provide help and counseling for the parents or care givers so that the abuse stops. The method

**Fig. 10.10** An impetiginous lesion, which can resemble the appearance of a cigarette burn.

of liaison and referral between dental practitioners and the other health care professionals involved in child abuse cases will vary, not only from country to country, but also between cities and different regions within one country. It is now law in a number of countries that regional child welfare departments issue procedural guidelines for dental practitioners in suspected child abuse cases. A small number of studies have investigated general dental practitioners' views on their role within child protection or on possible barriers specifically related to child protection which militate against their undertaking such a role (67–75). They felt some reluctance to become involved because of their lack of knowledge both of the signs of child abuse and the workings of the other agencies involved in child protection. They also reported significant concerns about how notifying possible incidents might affect the child, family unit and practice. This suggests a lack of knowledge about the aim of the child protection agencies that are not only to protect children from further abuse but also to strengthen the family unit. It seems highly likely that unless this training issue is addressed, children will present to dentists with indicators of potential abuse and will receive an inadequate and/or inconsistent response to their need for protection. This is particularly disturbing as the consequences of such inadequate responses from health and social care professionals have recently been widely publicized in landmark cases (76).

A child with a severe injury should be referred immediately to a hospital-based consultant pediatrician. Where suspicions are aroused in other cases, the dentist should speak to the designated person in the local guidelines who will advise on the appropriate course of action.

Dental practitioners should ensure that their clinical records are completed immediately with illustrations of the size, position and type of injuries. Photographic documentation would be beneficial in this respect. These records may be referred to in any subsequent case conference or legal proceedings.

In law the needs of a child are paramount. Dental practitioners should not feel any guilt about referring a child in need to the child protection services as they are not accusing either parent; they are simply asking for help and a second opinion on an important and difficult diagnosis. It is neither in the interest of the child nor the parents for child abuse to be covered up. To do so leaves the parents at greater risk of inflicting more severe injuries next time, being imprisoned for causing more severe injuries, and long-term loss of custody of their children. Early intervention may help to prevent these events. Failure to follow up suspicions is a form of professional negligence. Dental practitioners should be aware that in the United States a doctor who fails to report suspected child abuse is guilty of a federal offence that is punishable by imprisonment.

This being said, it must be stressed that the diagnosis of child physical abuse is a difficult one to make with certainty. Because of the high frequency of intraoral and facial injuries, dental professionals comprise a very important part of the team necessary for identifying and reporting child abuse and neglect (44, 77). However, the team approach is absolutely necessary, whereby members of multiple specialties collaborate to confirm the diagnosis of child physical abuse. Successful child protection involves the sharing of albeit small pieces of information between the caring agencies to obtain a whole picture of that child and their environment. Without adequate training, dental staff will not feel empowered to take responsibility for referring a child to the protection services. This mismatch between dental practitioners' attitudes to and knowledge of the child protection process and the reality of child abuse poses of itself a significant risk to children.

In conclusion, the dental practitioner's contribution to the management of child physical abuse should be to do the following (34):

- Recognize the possibility of physical abuse.
- Provide essential emergency dental treatment and arrange further treatment if required.
- Inform the appropriate authorities of his or her suspicions.

## The dentist's role in the management of dental neglect

It is important that dental practitioners recognize dental neglect as part of overall physical neglect. Dental neglect can be defined as 'the persistent failure to meet a child's basic oral health needs, likely to result in the serious impairment of a child's oral or general health or development' (78).

Studies have concluded that children with a welfare concern have a larger proportion of dental caries (79), with much of this caries being untreated. Untreated dental disease can lead to significant pain and sepsis; it has also been shown

to contribute to poor overall growth and diminished quality of life (80).

In most developed countries it is not unreasonable to expect that families are aware that their children's teeth need to be brushed twice daily with a fluoride-containing toothpaste and that sweet foods and beverages should be limited to mealtimes. However, it is often difficult to establish that adequate oral care advice has been given to the carer and the presence of caries cannot automatically be considered as being negligent. However, once professional advice has been provided and there is then failure to return for appropriate oral care for a child (with social support if necessary), this should be considered negligent and should be dealt with according to local guidelines. It is unacceptable for carers only to seek treatment when their child is in pain and fail to return with the child for follow-up treatment as prescribed by a dentist.

Dental caries is entirely preventable and this may signify missed potential, it is also entirely treatable once established and failure to access treatment is an act of omission. The dental team needs to engage with other health professionals to identify those families that require additional help and support to enable them to attend appointments thus safeguarding the child and helping them achieve their full potential.

## Essentials

### Terminology

- Non-accidental injury (NAI) or child physical abuse

### Frequency

- Prevalence in children ranges from 0.1% to 10% in various countries

### Clinical findings

- Delay in seeking treatment
- Explanation for injury does not fit the clinical findings
- Explanation for injury may differ with each telling and from person to person
- Abnormal parental reaction and behavior
- Type of relationship between parents and child
- The child's reaction to other people
- The child's story may differ from the parents' one
- 50% of child physical abuse cases have orofacial findings
- Usually more than one sign is present
- Hematomas of different vintage suggest repeated trauma

### Treatment

- Recognize the possibility of abuse or neglect
- Provide emergency dental treatment
- Refer the patient for a medical examination
- Share suspicions with relevant agencies

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# 11

# Classification, Epidemiology and Etiology

L. Andersson, S. Petti, P. Day, K. Kenny, U. Glendor & J. O. Andreasen

## Classification

An accurate definition of the disease or condition under investigation is a prerequisite for the study and treatment of a disease or condition. Traumatic dental injuries (TDIs) have been classified according to a variety of factors, such as etiology, anatomy, pathology, therapeutic considerations and degree of severity (1–7).

### Classification in the clinic

The most used classification at the moment is considered the gold standard and was developed by Andreasen based on a system adopted by the World Health Organization (WHO) in its *Application of the International Classification of Diseases to Dentistry and Stomatology* (7). For the sake of completeness, it was felt necessary to define and classify certain trauma entities not included in the WHO system. The following classification includes injuries to the teeth, supporting structures, gingiva and oral mucosa and is based on anatomic, therapeutic and prognostic considerations and can be applied to both the primary and the permanent dentitions (Table 11.1, Figs 11.1, 11.2, 11.3 and 11.4). The code number refers to the International Classification of Diseases (1992) (7). This classification is appropriate to use in a dental practice with diagnostic aids such as pulp sensibility tests, transillumination and radiographic examination. In this text, this classification will be used to describe the various trauma types. This should always be the system of first choice when using classification in the clinic and is today the most suitable system for comparing results from different studies. When registering data it is important to be aware of that many teeth also have combination injuries with more than one diagnosis for each tooth (8).

### Classification for field screening

There may be situations when diagnostic equipment and tools are not available (e.g. when doing field screening for epidemiological studies) where some of the important methods used in clinics, for example radiographic examination, cannot be used. A solution to this is to use a classification that is a compilation based on the most common classifications currently used in epidemiological surveys. It is very similar to the one proposed by Ellis and Davey in 1970 (1). However, one must be aware of that this way of field screening has many limitations and weaknesses and should never be the first choice when the Andreasen system (Table 11.1) is applicable. This field screening classification includes six categories: no TDI, treated TDI, enamel fracture only, enamel and dentin fracture, pulp injury and missing tooth due to TDI (Table 11.2). It is important to note that many signs and symptoms assessed in a dental surgery at the time of the injury (Table 11.2) cannot be identified in a screening survey. For example, injuries to the supporting structures are not included in any screening because they do not leave a visible permanent marker, such as tooth fractures. Similarly, other signs are excluded because surveys do not use diagnostic aids. Hospital- or surgery-based studies present a similar challenge as these do not record injuries in those who did not visit a dentist about the injury.

### Why do we need to improve trauma registration?

Traumatic dental injuries are common and can occur throughout life. Numerous treatment options and interventions are available, depending on the specific TDI sustained. Evidence-based comparisons of treatments and interventions is challenging for a number of different reasons.

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**Table 11.1** Clinical classification of traumatic dental injuries (TDIs) according to Andreasen including codes of the WHO International Classification of Diseases to Dentistry and Stomatology.

| **Injuries to the hard dental tissues and the pulp (Fig. 11.1)** | | |
|---|---|---|
| **Code** | **Injury** | **Criteria** |
| N 502.50 | Enamel infraction | An incomplete fracture (crack) of the enamel without loss of tooth substance (Fig. 11.1A) |
| N 502.50 | Enamel fracture (uncomplicated crown fracture) | A fracture with loss of tooth substance confined to the enamel (N 502.50) (Fig. 11.1A) |
| N 502.51 | Enamel-dentin fracture (uncomplicated crown fracture) | A fracture with loss of tooth substance confined to enamel and dentin, but not involving the pulp (Fig. 11.1B) |
| N 502.52 | Complicated crown fracture | A fracture involving enamel and dentin, and exposing the pulp (Fig. 11.1C) |
| N 502.54 | Uncomplicated crown-root fracture | A fracture involving enamel, dentin and cementum, but not exposing the pulp (Fig. 11.1D) |
| N 502.54 | Complicated crown-root fracture | A fracture involving enamel, dentin and cementum, and exposing the pulp (Fig. 11.1E) |
| N 502.53 | Root fracture | A fracture involving dentin, cementum and the pulp (Fig. 11.1F). Root fractures can be further classified according to displacement of the coronal fragment (see under Luxation injuries below) |
| **Injuries to the periodontal tissues (Fig. 11.2)** | | |
| **Code** | **Injury** | **Criteria** |
| N 503.20 | Concussion | An injury to the tooth-supporting structures without abnormal loosening or displacement of the tooth, but with marked reaction to percussion (Fig. 11.2A) |
| N 503.20 | Subluxation (loosening) | An injury to the tooth-supporting structures with abnormal loosening, but without displacement of the tooth (Fig. 11.2B) |
| N 503.20 | Extrusive luxation (peripheral dislocation, partial avulsion) | Partial displacement of the tooth out of its socket (Fig. 11.2C) |
| N 503.20 | Lateral luxation | Displacement of the tooth in a direction other than axially. This is accompanied by comminution or fracture of the alveolar socket (Fig. 11.2D) |
| N 503.21 | Intrusive luxation (central dislocation) | Displacement of the tooth into the alveolar bone. This injury is accompanied by comminution or fracture of the alveolar socket (Fig. 11.2E) |
| N 503.22 | Avulsion (exarticulation) | Complete displacement of the tooth out of its socket (Fig. 11.2F) |
| **Injuries to the supporting bone (Fig. 11.3)** | | |
| **Code** | **Injury** | **Criteria** |
| N 502.40 | Comminution of the maxillary alveolar socket | Crushing and compression of the alveolar socket. This condition is found concomitantly with intrusive and lateral luxations (Fig. 11.3A) |
| N 502.60 | Comminution of the mandibular alveolar socket | |
| N 502.40 | Fracture of the maxillary alveolar socket wall | A fracture confined to the facial or oral socket wall (Fig. 11.3B) |
| N 502.60 | Fracture of the mandibular alveolar socket wall | |
| N 502.40 | Fracture of the maxillary alveolar process | A fracture of the alveolar process which may or may not involve the alveolar socket (Figs 11.3C, D) |
| N 502.60 | Fracture of the mandibular alveolar process | |
| N 502.42 | Fracture of the maxilla | A fracture involving the base of the maxilla or mandible and often the alveolar process (jaw fracture). The fracture may or may not involve the alveolar socket (Figs 11.3E, F) |
| N 502.61 | Fracture of the mandible | |
| **Injuries to gingiva or oral mucosa (Fig. 11.4)** | | |
| **Code** | **Injury** | **Criteria** |
| S 01.50 | Laceration of gingiva or oral mucosa | A shallow or deep wound in the mucosa resulting from a tear, and usually produced by a sharp object (Fig. 11.4A) |
| S 00.50 | Contusion of gingiva or oral mucosa | A bruise usually produced by impact with a blunt object and not accompanied by a break in the mucosa, usually causing submucosal hemorrhage (Fig. 11.4B) |
| S 00.50 | Abrasion of gingiva or oral mucosal | A superficial wound produced by rubbing or scraping of the mucosa leaving a raw, bleeding surface (Fig. 11.4C) |

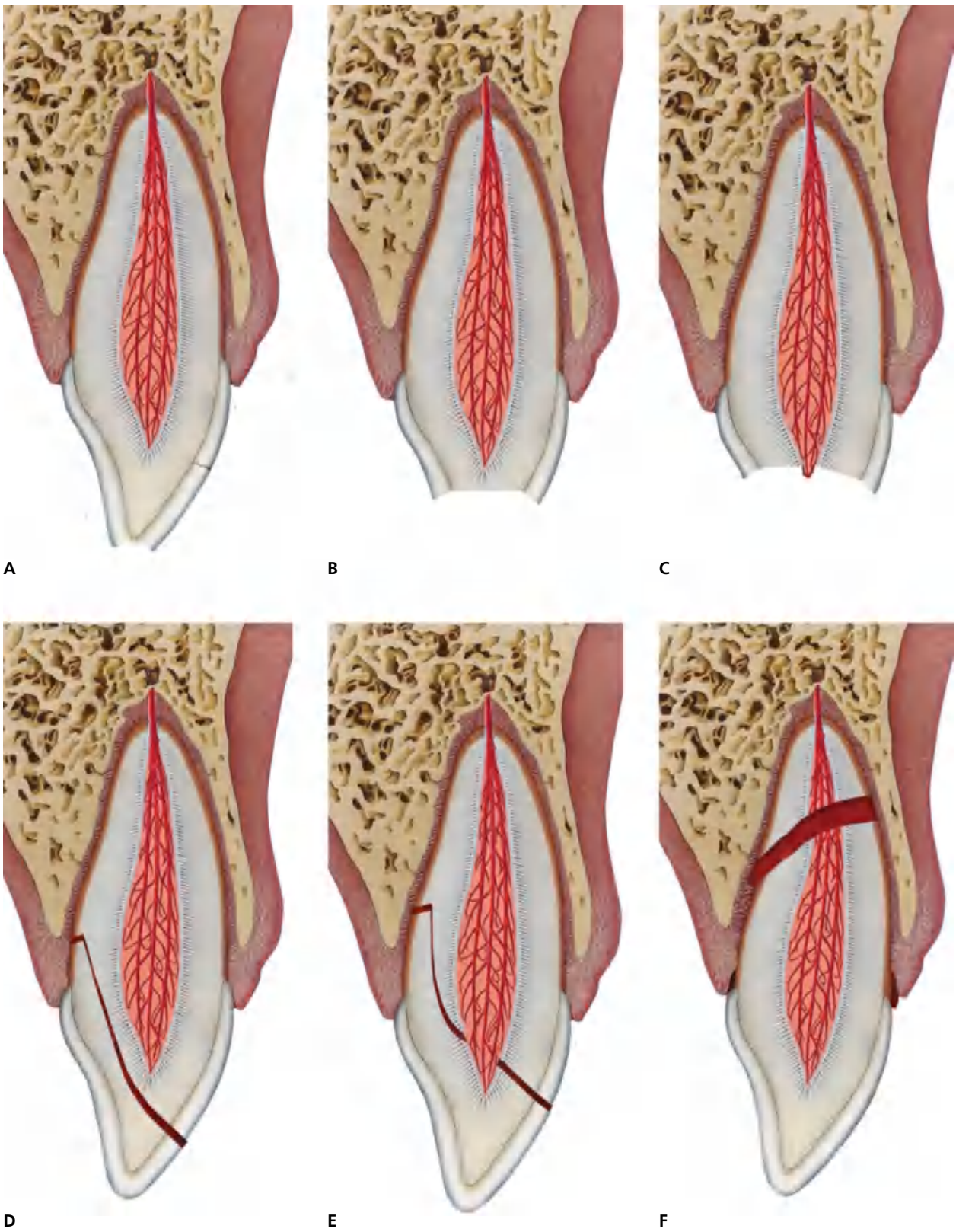



**Fig. 11.1** Injuries to the hard dental tissues and pulp. A. Crown infraction and uncomplicated fracture without involvement of dentin. B. Uncomplicated crown fracture with involvement of dentin. C. Complicated crown fracture. D. Uncomplicated crown-root fracture. E. Complicated crown-root fracture. F. Root fracture.

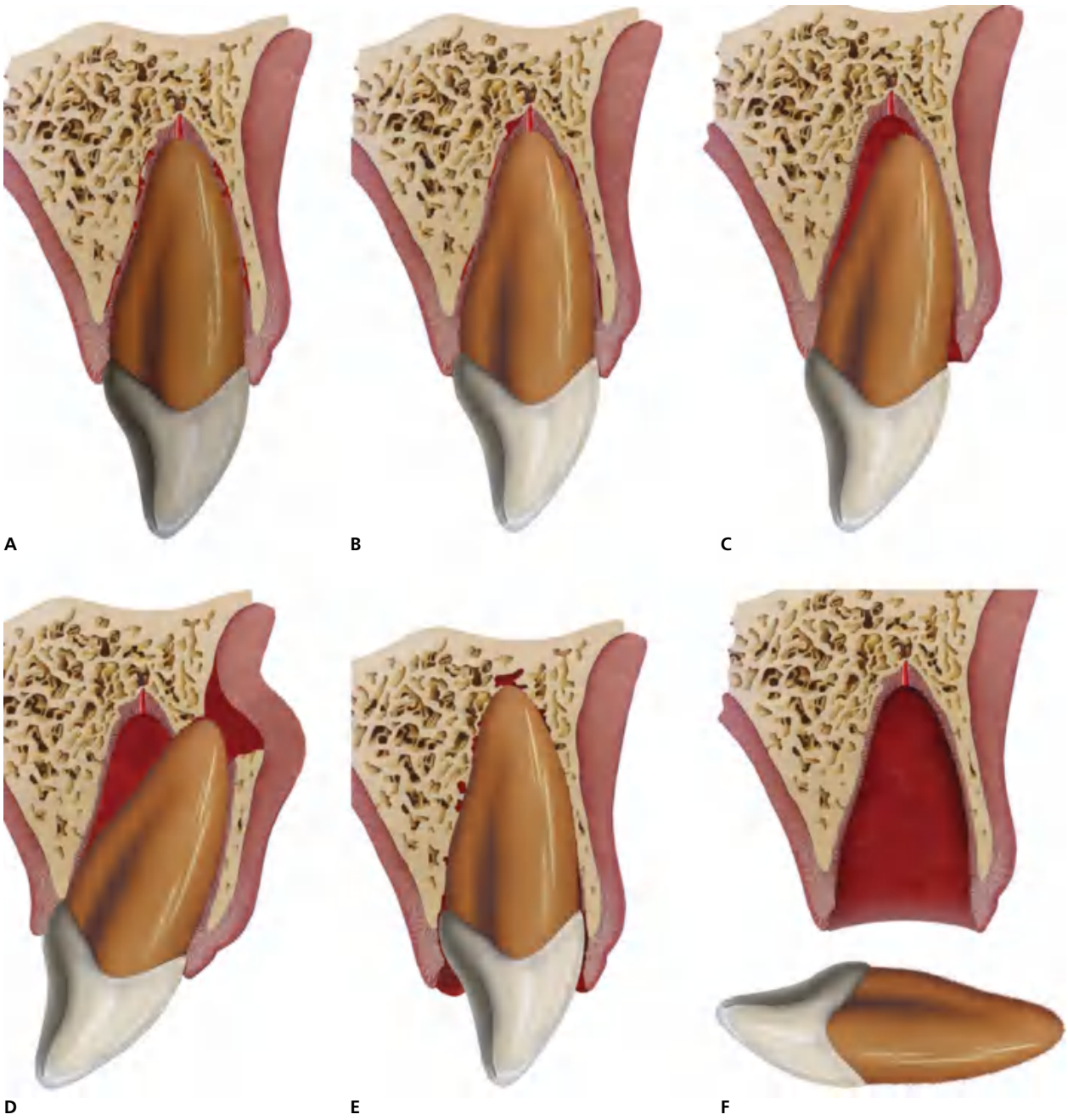



**Fig. 11.2** Injuries to the periodontal tissues. A. Concussion. B. Subluxation. C. Extrusive luxation. D. Lateral luxation. E. Intrusive luxation. F. Avulsion.

*Reporting bias.* When the worldwide trauma literature is reviewed, it is dominated by one center in Copenhagen. The lifetime's work of Dr Andreasen and his research group is remarkable in both its longevity and the prolific publication of their results (9). One of the key fundamentals of scientific research is replication, where results found in one center with one group of patients are consistently seen across other patient groups as well. For example in the avulsion literature, over a third of teeth suffering an avulsion injury were seen by the Copenhagen group (see Chapter 20). While we can only commend the Copenhagen group, it is vital that the results from other centers are published even when they confirm the findings from earlier studies. By increasing the number of studies available for clinicians and researchers to analyze, our ability to compare, contrast and combine studies as appropriate is enhanced.

*Heterogeneity of outcomes.* Even when clinical outcomes studies are published, a further problem arises in the diversity of outcomes reported. A recent systematic review identified that there was significant heterogeneity in outcomes reported for TDIs in the literature (10). Only 10 studies, confined to two types of TDI, were eligible for inclusion in this review. The outcomes reported predominantly concentrated on injury activity and the physical consequences of the injury. There was little consistency between studies for length of

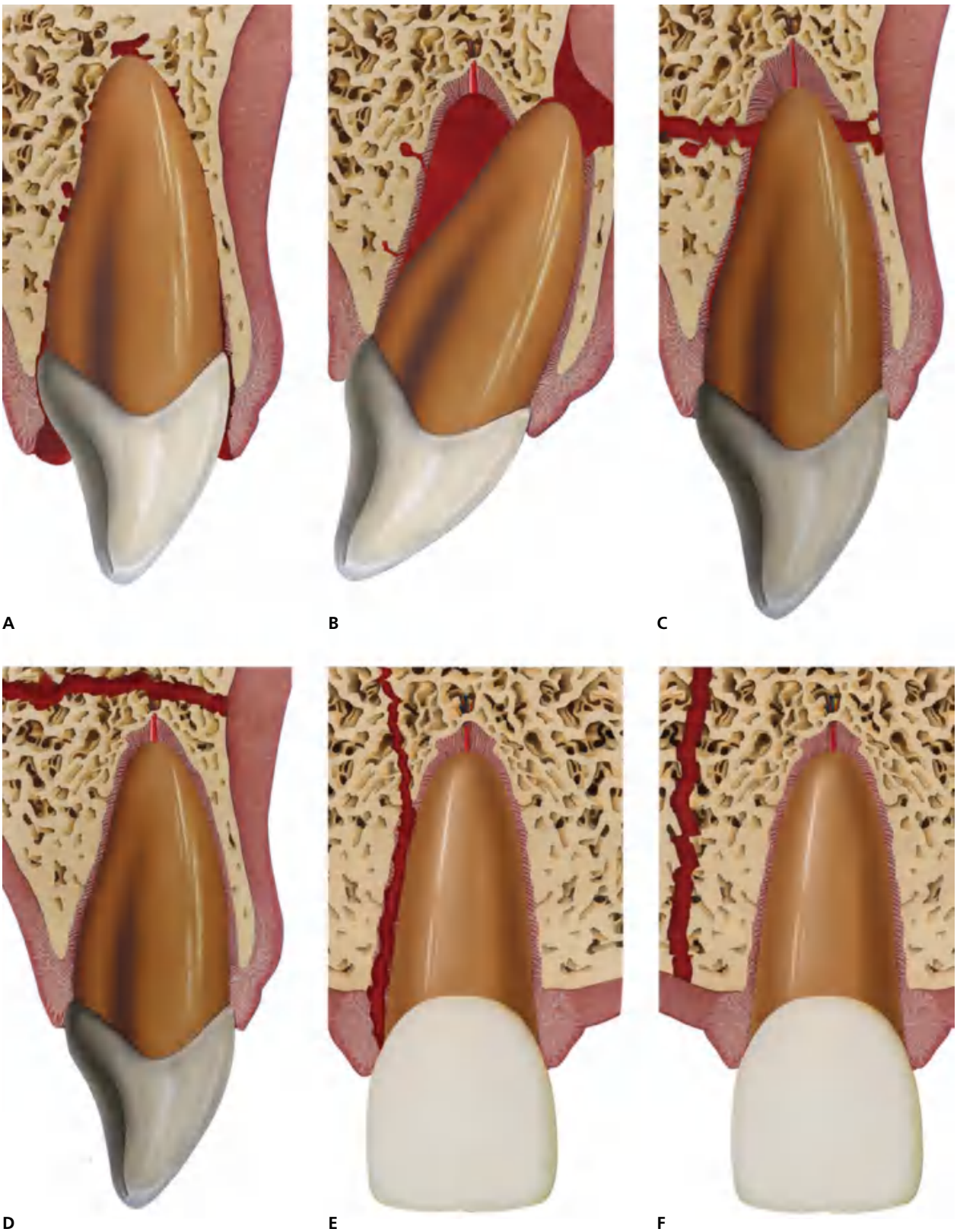



**Fig. 11.3** Injuries to the supporting bone. A. Comminution of alveolar socket. B. Fractures of facial or lingual alveolar socket wall. C and D. Fractures of alveolar process with and without involvement of the tooth socket. E and F. Fractures of mandible or maxilla with and without involvement of the tooth socket.

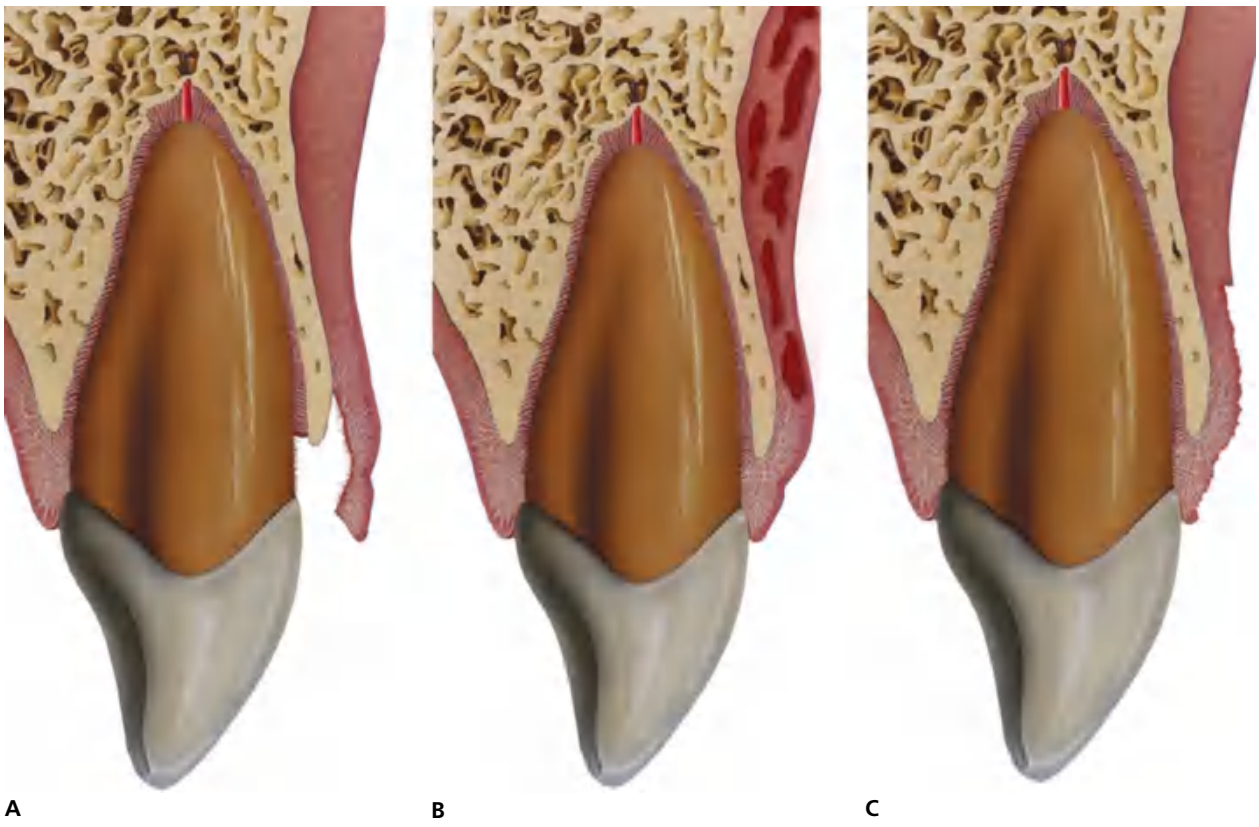



**Fig. 11.4** Injuries to the gingiva or oral mucosa. A. Laceration of gingiva. B. Contusion of gingiva. C. Abrasion of gingiva.

**Table 11.2** Field screening for epidemiological classification of traumatic dental injuries (TDIs) including codes of the WHO International Classification of Diseases to Dentistry and Stomatology.

| Code | Injury | Criteria |
|---|---|---|
| Code 0 | No injury | No evidence of treated or untreated dental injury |
| Code 1 | Treated dental injury | Composite restoration, bonding of the tooth fragment, crown, denture or bridge pontics replacing missing teeth due to TDI, restoration located in the palatal/lingual surface of the crown suggesting endodontic treatment and no evidence of decay, or any other treatment provided due to TDI<br>Note: Composite restorations may be difficult to recognize |
| Code 2<br>(N 502.50) | Enamel fracture only<br>(Fig. 11.1A) | Loss of a small portion of the crown, including only the enamel |
| Code 3<br>(N 502.51) | Enamel/dentin fracture<br>(Fig. 11.1B) | Loss of a portion of the crown, including enamel and dentin without pulp exposure |
| Code 4<br>(N 502.52)<br>(N 502.53)<br>(N 502.54)<br>(N 503.20)<br>(N 503.21) | Pulp injury<br>(Fig. 11.1C)<br>(Fig. 11.1F)<br>(Fig. 11.1E)<br>(Fig. 11.2C, D)<br>(Fig. 11.2E) | Signs or symptoms of pulp involvement due to dental injury. It includes fractures with pulp exposure, dislocation of the tooth, presence of sinus tract and/or swelling in the labial or lingual vestibule without evidence of caries and discoloration of the crown. The examiner must check if pulp involvement was due to caries (presence of treated or untreated caries lesion), and ask the subject whether they have a history of a harmful incident involving the front teeth/mouth |
| Code 5<br>(N 503.22) | Missing tooth due to trauma<br>(Fig. 11.2F) | Absence of the tooth due to a complete avulsion. Code 5 should be used only for teeth judged to be missing due to trauma. A positive history of trauma is needed to record missing due to trauma and the examiner must ask the subject if the avulsion was due to a harmful incident involving the front teeth/mouth or if teeth have been extracted due to caries |
| Code 9 | Excluded tooth | Signs of traumatic injury cannot be assessed, i.e. presence of appliances or all permanent incisors missing due to caries |

follow-up, the time points at which outcomes were evaluated, or the methods used to report them (10). It was also noted that patient-related outcomes were particularly poorly represented, with no outcomes reported for quality of life and family outcomes. The authors concluded that the findings demonstrated a clear indication for the development of a core outcome set for TDIs.

Therefore, there is an urgent need to identify what clinical information and outcomes should be recorded, how these should be recorded and when these should be recorded. This would act as a core or minimum dataset with clinicians and researchers encouraged to collect additional information and outcomes depending on their specific research interests.

## How do we improve trauma registration?

### *Developing a core outcome set for traumatic dental injuries*

A core outcome set (COS) is an agreed and standardized collection of outcomes for use as a minimum in clinical studies and trials. An outcome is a clinical measure used to make judgments about the efficacy of treatment. However, outcome measures also represent the suffering or loss of health experienced by an individual as a result of the process of disease (11). Outcomes can be thought of as *what* is measured in the trial and outcome measures as *how* that is measured (12). Outcomes need to be relevant to patients, clinicians and policy makers if the findings of research are to influence both clinical practice and future research. Core outcome sets do not imply that outcomes in a particular trial should be restricted to those in the COS. Rather, there is an expectation that the core outcomes will always be collected and reported, and that researchers will continue to explore other outcomes (13).

One of the earliest attempts to standardize outcomes was undertaken by the WHO in the 1970s. More than 30 representatives from cancer trial groups collaborated and published the WHO handbook of guidelines recommending the minimum requirements for data collection in cancer trials. Another notable example is OMERACT (Outcome Measures in Rheumatology) – an independent initiative of international health professionals interested in outcome measures in rheumatology. The OMERACT group are credited with developing the first ever COS during their inaugural conference in the Netherlands in 1992 (11). As a result of this first COS, the number of studies reporting all these outcomes doubled from 35% to 70% (14). There are now more than 50 other groups working on COS across all areas of medicine.

The COMET (Core Outcome Measures in Effectiveness Trials) initiative brings together researchers interested in the development, application and promotion of COS. Data on relevant individual studies, both published and ongoing, are being included in a free, publically available internet-based resource (http://www.comet-initiative.org; accessed January 2018). To date, there are 10 COS being developed in the dentistry and oral health subject field of the database, though only one has been completed and published (15).

The COMET group have been instrumental in providing guidance on how to develop a COS (http://www.comet-initiative.org/resources). Key issues to consider in the development of a COS include its scope, the stakeholder groups to involve, choice of consensus method and achievement of a consensus.

Following the publication of the systematic review (10), which advocated the development of a COS for TDIs, the International Association of Dental Traumatology (IADT) agreed to endorse this project. A protocol was developed by the research team and published on the COMET database (16, 17). An expert working group (EWG) was established. This is novel and innovative and involved a broad range of experienced and motivated experts from a variety of dental specialties from across the world. The majority of the participants are prominent in their field and have been involved in international guideline development.

A number of outcomes were identified as recurring throughout the different injury types. These outcomes were then included as 'generic', i.e. relevant to all TDI (Table 11.3). The remaining or 'injury-specific' outcomes, i.e. related only to one or more particular TDIs, were included in a separate table (Table 11.4). For further methodological details see Kenny et al. (18).

The EWG decided unanimously to measure the COS at routine recall intervals as recommended by the IADT guidelines for different TDIs (19–21) (see Appendix 1 in this book). Where possible, dental traumatology gold standard outcome measurements were used, but if not available, best evidence-based outcome measurement tools were chosen. For some outcomes, no suitable outcome measurement yet exists. The finalized outcome matrices for each outcome can be found at the IADT webpage (http://iadt-dentaltrauma.org; accessed January 2018) and as supplementary materials

**Table 11.3** Core outcome set – generic outcomes.

| Domain | Generic outcomes |
|---|---|
| Injury activity | **Periodontal healing**<br>Bone loss<br>Gingival recession<br>Mobility<br>Ankylosis/resorption |
| | **Pulpal healing**<br>Infection |
| Physical consequences of injury | **Pain** |
| | **Discoloration** |
| Functional status | **Tooth loss**<br>Premature loss primary teeth |
| Quality of life and social outcomes | **Quality of life**<br>Days off work<br>Days off school<br>Days off sport |
| Side effects of therapy | **Trauma-related dental anxiety** |
| Health resource utilization | **Number of clinic visits** |

in the Kenny et al. publication (18). An example of an outcome matrix is shown in Table 11.5.

It is important to re-emphasize here that the set does not imply that outcomes in a particular trial should be restricted to those in the COS. Rather, there is an expectation that the core outcomes will always be collected and reported, and that researchers will continue to explore other outcomes. If a COS is not implemented in a trial, the researchers should explain their decision in the trial protocol and subsequent report. Similarly, if the primary outcome for a particular trial is not within the COS, then the relevance and importance of the chosen outcome should be explained. Journal editors, funders and review boards will have an important role encouraging and/or mandating the use of COS in research. In this way, a development towards a global standard in data collection can be encouraged.

Further work is required to ensure the COS is fit for purpose. This will include engaging with patients (and their parents where applicable) to ensure the COS is relevant to them, and to identify any outcomes of importance to them that have not been identified. This may be different from country to country and is likely to be related to how health care is provided in different jurisdictions. The practicalities of day to day use of the set – whether it will be paper or technology based – is of utmost importance and will require dissemination and implementation science research before full roll-out and uptake can be expected.

This leads to the conclusion that the COS is a dynamic entity requiring ongoing work and research. It has the potential to revolutionize data collection for TDIs, which will ultimately lead to more effective clinical care for our patients.

**Table 11.4** Core outcome set – injury-specific outcomes.

| Injury | Injury-specific outcomes |
|---|---|
| Uncomplicated crown fracture, Complicated crown fracure | Quality of restoration<br>Loss of restoration |
| Crown-root fracture | Mobility<br>Loss of restoration |
| Root fracture | Root number site repair<br>Mobility |
| Extrusion, Lateral luxation, Alveolar fracture | Infraocclusion |
| Intrusion/avulsion | Realignment<br>Infraocclusion |
| Immature non-vital and infected permanent teeth | Late stage crown fracture<br>Root length<br>Root width |
| Primary teeth | Realignment<br>Impact on permanent successor |

## *Future trauma registration*

Dental trauma registration has been considered for many years. Many individual centers have used their own trauma registration data to allow them to review specific injuries or patients that are of interest to them. As is shown in the literature, collecting a large cohort of patients with a specific injury, especially when it is relatively rare, can take a significant amount of time; for example, the 400 avulsions collected in the seminal paper published by Andreasen et al. (22) took 23 years. Therefore, authors in dental traumatology (23–25) have on a regular basis called for a more collaborative approach with multiple centers working together to permit large cohorts of patients with TDIs to be collected over shorter periods. However, this requires collaborating centers to agree on what information they will collect as well as the treatment protocols they plan to use.

In essence a trauma recorded in the patient's clinical records is split into five different components.

(1) Trauma history and initial consultation (see Appendix 1). The literature has clearly identified that the use of a structured history at initial consultation led to a significant improvement in the quality of the trauma records (26–28). Earlier work identified variations in what was recorded at initial presentation across the UK and Ireland (29).

**Table 11.5** Example of a COS matrix: trauma-related dental anxiety. For more details, see (18) and the IADT core outcome set (http://iadt-dentaltrauma.org). Reproduced from (18) with permission.

| Domain | Generic outcome | Measurement and rationale | Time points for assessment of outcomes |
|---|---|---|---|
| Side effects of therapy | Trauma-related dental anxiety | **What to record:** Levels of dental anxiety immediately after the accident and at various time points throughout the treatment. A traumatic dental injury in childhood has been shown to lead to increased levels of dental anxiety in some patients<br>**Definition:** Anxiety is a feeling of apprehension characterized by fear, tension, nervousness or restlessness. A traumatic dental injury in childhood has been shown to lead to increased levels of dental anxiety in some patients<br>**How:**<br>Children aged 5–8 years: Facial Image Scale<br>Children aged 8–12 years: Faces version of the Modified Child Dental Anxiety Scale ($MCDAS_f$)<br>Age 12 and over: Kleinknecht's DFS Dental Fear Survey and/or Corah's DAS Dental Anxiety Scale<br>**By whom:** by a dentist *(or a member of the wider clinical team with appropriate training)* | **When:**<br>At the time of the injury<br>3 months<br>1 year |

(2) Prognostic factors relating to the injury and subsequent treatment which may influence outcomes (30). These prognostic factors need to be carefully collected at both initial consultation and at follow-up clinical visits. This is most likely to be achieved using the structured histories described in Appendices 1 and 2.
(3) Diagnosis of each and every injured tooth, as well as other associated hard and soft tissue injuries. The different trauma diagnoses are described earlier in this chapter.
(4) Treatment provided.
(5) Outcomes. As a minimum these should include the COS (Tables 11.3 and 11.4).

In its simplest design, trauma registration software extracts the relevant information from the clinical records, thereby minimizing the impact on the busy clinician (31). However, this requires each clinician in each center to record their clinical records in a similar fashion (e.g. use Appendices 1 and 2 in this book for their trauma history and follow-up review appointments) and in a comprehensive manner. Researchers and clinicians may be interested in many different areas and therefore great thought has to be given as to how specific information is collected (see earlier section on core outcome sets) and what specific research questions they propose to answer.

An approach taken by the Copenhagen and Newcastle/Belfast groups was to use consistent data collection protocols and to simply record the diagnosis of the TDI on a database. At this point, if a researcher wanted to examine a particular injury, the database then provided them with a list of relevant patients. The researcher would then examine the clinical records of each patient with this injury and manually extract and enter the data into statistical software to permit analysis.

While a number of electronic trauma registration systems have been developed, none has progressed to widespread use. Moving forward, there are a number of key barriers which any comprehensive system needs to address.

- Preventing duplication. Asking busy clinicians write up their clinical records as well as recording data on a separate data capture system is unlikely to work and therefore data collection needs to be integrated with the clinical record keeping.
- Research (26, 32) has identified that if there is an important question that needs answering, then a specific question should be asked. Relying on the clinician to remember to ask it and then record it is unrealistic.
- Drop down lists. For important questions, provide the clinician with a drop list of answers. This ensures that the question is answered in the same way each time, simplifying data analysis and minimizing any potential errors with a researcher trying to interpret what the clinician meant from their free text entry (32).
- Computerizing clinical records. The only realistic method for efficient data collection is via electronic records and therefore the original patient record should be recorded on a computerized clinical record. This, however, induces significant complexity for data extraction, especially when different computerized clinical record software is used.
- Information governance and data security. This is a major logistical headache even when different centers are united by working for one health provider, for example the National Health Service in the UK. Firstly, the patient must consent to data sharing, especially if patient identifiable data are used. In the field of TDI this is almost inevitable where the records from one clinical visit need to be linked with subsequent visits. Secondly, secure and encrypted data transfer is mandatory and must meet the standards of both the local employers and wider national standards. Finally, if data are transferred between countries or outside blocks of countries (e.g. the European Union), further safeguards are required.

Realistically, these barriers are unlikely to be overcome in the near future. Therefore, a more simple solution is required. At its most basic, a trauma registration system would record the injury diagnosis (for both hard tissue and periodontal ligament) for each injured tooth, the stage of root formation and the core outcomes outlined earlier in the chapter.

In conclusion, when planning a study and before commencing it, the investigators should always make sure that the trauma registration system is in accordance with what is suggested in this chapter and Appendices 1 and 2, as well as the IADT COS (18). In the future, this may be a prerequisite for a manuscript to be accepted in scientific journals.

## Epidemiology

Around the world, trauma is responsible for more than 5 million deaths per year and is the leading cause of death for individuals up to the age of 45 years; trauma causes half of all deaths in the age range 10–24 years (33–35). Several hundred million people are injured by trauma worldwide. A large proportion of people surviving their injuries incur temporary or permanent disabilities.

Hands and feet are the parts of the body most often subjected to injuries caused by trauma (36) (Fig. 11.5). Although the oral region comprises as small an area as 1% of the total body area, a population-based 1-year longitudinal prospective Swedish survey has shown that oral injuries account for

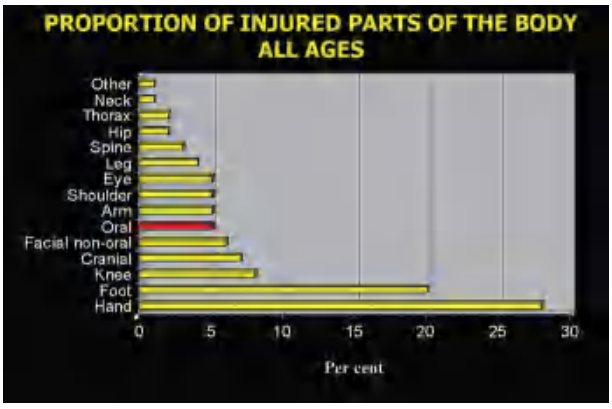



**Fig. 11.5** Proportion of injured parts of the body (all ages). From (36).

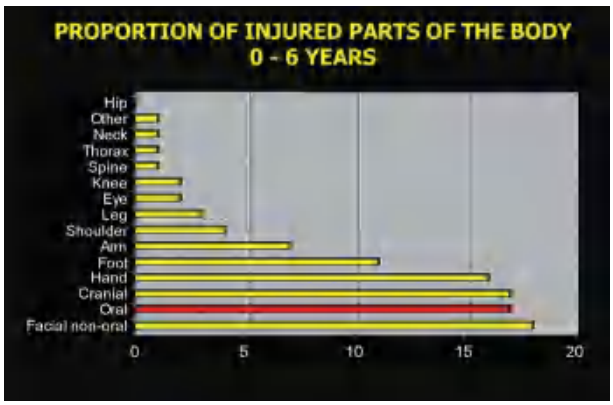



**Fig. 11.6** Proportion of injured parts of the body (ages 0–6 years). From (36).

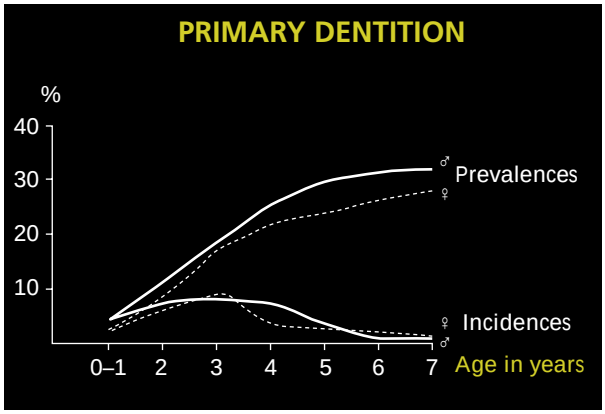



**Fig. 11.7** Prevalence and incidence of traumatic dental injuries to primary teeth among children in Copenhagen. Prevalence indicates frequency (in percentage) of children who have sustained dental injuries at the various ages examined. Incidence indicates the number of new dental injuries (in percentage) arising per year at the various ages examined. Reproduced from (41) with permission.

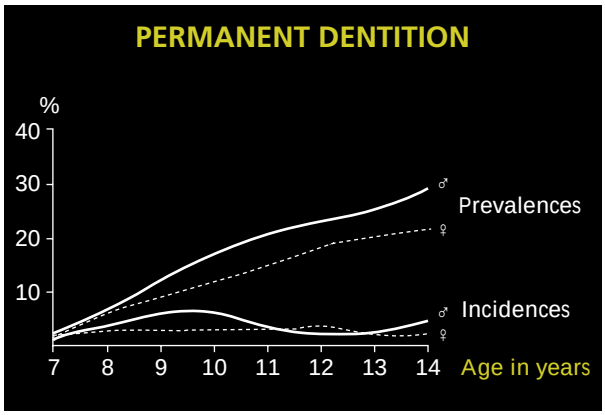



**Fig. 11.8** Prevalence and incidence of traumatic dental injuries to permanent teeth among children in Copenhagen. Prevalence indicates frequency (in percentage) of children who have sustained dental injuries at the various ages examined. Incidence indicates the number of new dental injuries (in percentage) arising per year at the various ages examined. Reproduced from (41) with permission.

5% of all bodily injuries in all ages (36) (Fig. 11.5). In preschool children aged 0–6 years in Sweden, traumatic oral injuries may make up as much as 17% of all bodily injuries, with injuries to the head being the most common (36) (Fig. 11.6). The oral region is at higher risk in children and adolescents, but more rarely seen in adults and elderly in contrast to bodily injuries (36).

The proportion of maxillofacial trauma in relation to all types of trauma reported from hospital accident and emergency departments has been reported as being 33% (37).

In a population-based longitudinal prospective study it was found that, among patients seeking consultation or treatment for injuries to the oral region, 92% present with TDI as compared to 28% for soft tissue injuries and 6% with bone fractures (36). Combinations are often seen, especially TDI and soft tissue injuries (36).

## Prevalence

The prevalence of TDI in the literature is high worldwide and varies considerably. This variation reflects not only socio-economic, behavioral and cultural diversity, but also the lack of standardization of methods and classifications observed in the literature. The use of standardized epidemiological protocols would facilitate comparison between countries (38). For example, differences in age and sex distribution between population-based studies examined have significantly contributed to the large variation observed in the literature. One way to eliminate the influence of age is to study the prevalence of TDI at given stages of development, such as for the primary dentition at the age of 5 (i.e. prior to the mixed dentition period) and at the age of 12 (i.e. after the mixed dentition period and the period of high trauma incidence).

### *Prevalence of traumatic dental injury to permanent teeth*

A large national survey in the USA in 6–50-year-olds showed that approximately one in four adults had evidence of TDI (39). In the UK, one in five children have experienced TDI to their permanent anterior teeth before leaving school (40).

The findings of a prospective study, where all dental injuries occurring from birth to the age of 14 years were carefully registered, demonstrated that 30% of the children had sustained injuries to the primary dentition and 22% to the permanent dentition (Figs 11.7 and 11.8). Altogether, every second child had sustained a TDI by the age of 14 years (41).

In a literature review article, Glendor concluded that in the primary dentition one-third of all children has sustained a TDI. In the permanent dentition one-fourth of all school children and one-third of all adults have sustained TDIs (42).

According to a recent global analysis by the chapter authors, based on 95 recently published studies (43–137) in the past two decades and involving more than 250 000 individuals

from all over the world, there are globally 15.5% adolescents and young adults aged 7–20 years with at least one TDI affecting permanent teeth (Table 11.6). This means that there are between 223 and 303 million children and adolescents around the world affected with TDI to the permanent teeth.

### *Prevalence of traumatic dental injury to primary teeth*

The analysis of global data based on 45 recent studies (138–182) of primary teeth involving almost 60 000 children shows that TDI to primary teeth is more frequent than TDI to permanent teeth. It affects as many as 23% individuals (Table 11.7), corresponding to 155–263 million children affected by TDI to the primary teeth.

### *Prevalence outside the literature*

The prevalence of TDI in the real world is likely to be much higher than the figures presented in the literature. Apart from a few longitudinal studies, most reports are cross-sectional, which means that the registration of previous injuries is to a certain extent dependent upon information from either the child or parents, which can entail a significant error. A clinical study demonstrated that parents in about half the cases of previously recorded injuries to primary teeth in a pre-school dental clinic, denied in a questionnaire that such injuries had occurred (41). Furthermore, the lack of diagnostic aids in cross-sectional surveys implies missing several relevant signs of TDI, such as luxation injuries (concussion, subluxation, intrusion, extrusion), pulp injuries, root fractures and resorptions, periapical lesions, replanted teeth and injuries to the supporting bone, gingiva and oral mucosa.

It is worth noticing that although TDIs show a high prevalence with a significant impact on individuals and society, and the presence of a sound body of knowledge about its causative factors and treatment, TDI is still a neglected oral condition at both the individual and public health level. Indeed, despite its high frequency, TDI is not included in the list of the 300 most important acute and chronic diseases and injuries provided by the Global Burden of Disease Study (183), while accurate cohort studies that supervise selected individuals for several years, show that the number of TDIs that actually occur is roughly double the number of referred and reported TDIs.

Globally, between 378 and 566 million children and adolescents aged 20 years or younger show at least one TDI (Table 11.8). This condition ranks, therefore, as being between the second and fourth most common oral conditions, and could be the 13th most frequent among all bodily diseases and injuries, being even more frequent than other important diseases such as diabetes, anxiety and asthma, according to the Global Burden of Disease Study (183).

## Incidence

There are very few studies of sufficiently high quality that have assessed the incidence of TDI (Table 11.9). Such studies

**Table 11.6** Prevalence of traumatic dental injuries (TDIs) to permanent teeth in the WHO regions and global world prevalence estimates.*

| WHO region | Mean age | Number of subjects | Prevalence | 95% confidence interval |
|---|---|---|---|---|
| European Region | 13.6 years | 55 996 | 14.9% | 10.0–20.5% |
| Region of the Americas | 14.6 years | 53 080 | 19.1% | 16.4–21.9% |
| Western Pacific Region | 12.1 years | 79 177 | 9.9% | 1.9–23.2% |
| South-East Asia Region | 15.0 years | 40 322 | 12.4% | 9.4–15.7% |
| Eastern Mediterranean Region | 12.3 years | 17 392 | 16.0% | 11.1–21.7% |
| African Region | 12.5 years | 5 895 | 10.6% | 8.0–13.5% |
| World | 14.0 years | 251 862 | 15.5% | 13.2–17.9% |

* Analysis based on 95 studies from all over the world published in the period 1996–2016 (43–137).

**Table 11.7** Prevalence of traumatic dental injuries (TDIs) to primary teeth

| WHO region | Mean age | Number of subjects | Prevalence | 95% confidence interval |
|---|---|---|---|---|
| European Region | 3.6 years | 14 180 | 14.2% | 6.0–25.0% |
| Region of the Americas | 3.1 years | 27 138 | 26.6% | 21.3–32.2% |
| Western Pacific Region | Unavailable data | | | |
| South-East Asia Region | 4.5 years | 2 626 | 37.3% | 1.8–98.5% |
| Eastern Mediterranean Region | 4.7 years | 2 052 | 22.7% | 12.0–35.6% |
| African Region | 3.3 years | 3 467 | 22.7% | 13.3–33.7% |
| World | 3.4 years | 57 152 | 23.0% | 17.3–29.3% |

*Analysis based on 45 studies from all over the world published in the period 1996–2016 (138–182).

**Table 11.8** Global prevalence of oral conditions and ranking including traumatic dental injuries (TDIs) in the list of the most important diseases considered by the Global Burden of Disease Study.

| Condition name | Prevalence* | 95% confidence interval* | Global rank | 95% uncertainty interval |
|---|---|---|---|---|
| Asymptomatic permanent caries | 2390 | 2350–2429 | 1 | 1 |
| Periodontal diseases | 504 | 497–511 | 13 | 13–15 |
| Asymptomatic deciduous caries | 475 | 472–478 | 16 | 14–17 |
| TDI (overall) | 468 | 378–566 | 18 | 13–19 |
| Edentulism and severe tooth loss | 251 | 248–253 | 31 | 27–32 |
| TDI to permanent teeth | 262 | 223–303 | 29 | 25–38 |
| TDI to deciduous teeth | 206 | 155–263 | 38 | 27–41 |

* Number of cases × 1 000 000.

**Table 11.9** Reported incidence of traumatic dental injuries in longitudinal high-quality surveys during at least 1 year.

| Study | Year | Country | Age | Per 100 |
|---|---|---|---|---|
| Ravn & Rossen (230) | 1969 | Denmark | 7–16 | 3 |
| Andreasen & Ravn (227) | 1972 | Denmark | 0–14 | 4 |
| Hedegård & Stålhane (210) | 1973 | Sweden | 7–15 | 1.5 |
| Ravn (209) | 1974 | Denmark | 7–16 | 3 |
| Hansen & Lothe (229) | 1982 | Norway | 7–18 | 2.5 |
| Stockwell (125) | 1988 | Australia | 6–12 | 1.7 |
| Glendor et al. (184) | 1996 | Sweden | 0–19 | 1.3 |
| | | | 0–6 | 1.5 |
| | | | 7–19 | 1.3 |
| Borssén & Holm (55)* | 1997 | Sweden | 1–16 | 2.8 |
| Eilert-Petersson et al. (36)† | 1997 | Sweden | All ages | 0.4 |
| | | | 0–12 | 1.8 |
| Hamilton et al. (203) | 1997 | UK | 11–14 | 3.4 |
| Cecconello & Traebert (187) | 2007 | Brazil | Adolescents | 4.4 |
| Skaare & Jacobsen (196) | 2003 | Norway | 7–18 | 1.8 |
| Ramos-Jorge et al. (186) | 2008 | Brazil | 11–13 | 1.2–5.7 |
| Oldin et al. (169) | 2015 | Sweden | 0–17 | 2.8 |
| Basha et al. (189) | 2015 | India | 13 | 3 |
| Lexomboom et al. (185) | 2016 | Sweden | 8–10 | 2.9 |

* The yearly incidence of 16-year-olds born in 1975 and followed retrospectively until 1991.
† Oral injuries including dental injuries, injuries to the mandible or maxilla and injuries of the oral soft tissue.

**Table 11.10** Global annual incidence rate of traumatic dental injuries (TDIs) in children and adolescents (0–19 years of age) and difference between cohort studies and population-based studies.*

| Studies | Number of subjects | Incidence rate† | 95% confidence interval |
|---|---|---|---|
| World | 233 480 | 3.0 | 2.4–3.6% |
| Cohort studies | 4 046 | 5.2 | 2.8–8.3% |
| Population-based studies | 229 434 | 2.4 | 1.9–3.1% |

* Analysis based on 12 studies from all over the world published in the period 1996–2016 (186–197).
† TDI development per 100 persons per year.

are hard to carry out because it is difficult to relate findings in a representative sample of the society. Typically, most studies are from Scandinavia where almost all the dental care of children and adolescents used to be carried out in the public sector, which enabled collaboration and consistency in data collection and can for this reason be regarded as being highly accurate (Figs 11.7 and 11.8).

In a Swedish prospective study, the mean incidence for boys was 1.6 and for girls 1.0 per 100 individuals per year in the age interval 0–19 years (184). In a longitudinal multicenter study from Sweden an incidence rate of 2.8 per 100 individuals were reported. Similar findings were reported in another Swedish study extended over several years, reporting increasing standardized incidence rates from 1.9 to 2.9 per 100 individuals (185). Another Swedish study reported incidence rates of 2.8% among 0–17-year-old children (169). In a prospective study carried out in Australia, an incidence of 2.0 cases of TDI per hundred per year in children aged 6–12 years was reported (125). With an incidence rate among children and adolescents of three events per 100 subjects per year, in terms of risk, this means that every children/adolescent has a 3% probability of developing a TDI each year and, cumulatively, from 0 to 19 years, the probability of incurring a TDI is higher than 50% (Table 11.10).

The analysis of cohort studies, where a few subjects are followed and regularly examined, provides a higher rate (5.2 events per 100) than the analysis of population-based studies where the events are reported by public and private dental practitioners (2.4 per 100) (Table 11.10). This difference is probably due to the fact that several patients who suffer a mild TDI do not seek care, therefore incidence is artificially lower in population-based studies which refer to dental health care settings. Conversely, in cohort studies all TDIs are registered during follow-up examinations, therefore higher and more reliable incidence rates are found (Table 11.10). Although difficult to carry out, such studies should be considered the gold standard.

## Trends

The trend in TDI is not as clear and well documented as in, for example, trends in dental caries. Very few comparable studies exist that allow one to identify a trend in TDIs.

Data from the last 20–30 years suggest that there has not been a significant change in TDI rates in children and adolescents in Scandinavian countries (196). Similarly, data from the USA suggested small changes: 16–18.4% between 1996 and 2004 among 6–20-year-old individuals (39, 122). Data from England showed a small reduction, but not a clear trend (Fig. 11.9). Variations may be explained by variations in measuring TDIs. Also, the data from 1973 to 1993 refer to England and Wales, while in 2003 to only England. It is of interest to compare trends in dental caries and TDI. It seems that the prevalence of dental caries is falling more rapidly than TDIs. If this trend continues TDIs may became more prevalent than dental caries in the future (198–202). This is a special challenge because TDI affects mainly the front teeth, thus esthetic and facial attraction, while dental caries in children with low levels of the disease present only small occlusal caries in molars. Furthermore, most treatments needed for TDI are more complex and expensive than treatment of occlusal caries.

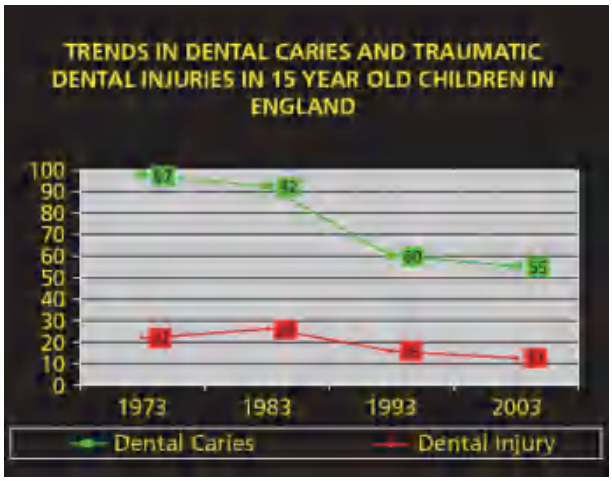



**Fig. 11.9** Trends (%) in dental caries and traumatic dental injuries in 15-year-old children in England. From (198–200, 202).

## Repeated trauma episodes

Dental trauma affects children and adolescents unequally. Some individuals are not affected at all, or just once, while others suffer from repeated trauma episodes (39, 203–206) (Fig. 11.10), with *no gender differences* (55, 205, 207) and with *repeated trauma episodes to the same teeth* (207, 212). Frequencies of repeated trauma episodes have been reported to range from 4% to 49% (4, 39, 159, 203, 207–211, 213), while repeated trauma episodes *to the same teeth* have been reported to range from 8% to 45% (198, 207, 211, 213). The risk of sustaining a second TDI episode has been found to be eight-fold for patients with their first trauma episode at 9 years of age compared with 12 years of age (207). Due to the increased risk for some individuals of sustaining multiple trauma episodes during life, prevention and/or orthodontic treatment should be performed at an early stage in life (42, 207, 212, 214). Prevention should be based on behavioral and environmental changes. Orthodontic treatment should be provided if it is cost-effective.

## Distribution by sex, age, ethnicity and socio-economic status

Boys sustain TDIs more often than girls in most studies. The general finding that boys have almost twice the number of TDIs in the permanent dentition than girls seems to be related to their more active participation in contact games and sports (4, 8, 196, 215–217); but recent studies have shown a reduction in this difference, which may reflect a change in girls' behavior in playing sports traditionally regarded as boys' games such as ice hockey and soccer (101, 218–220).

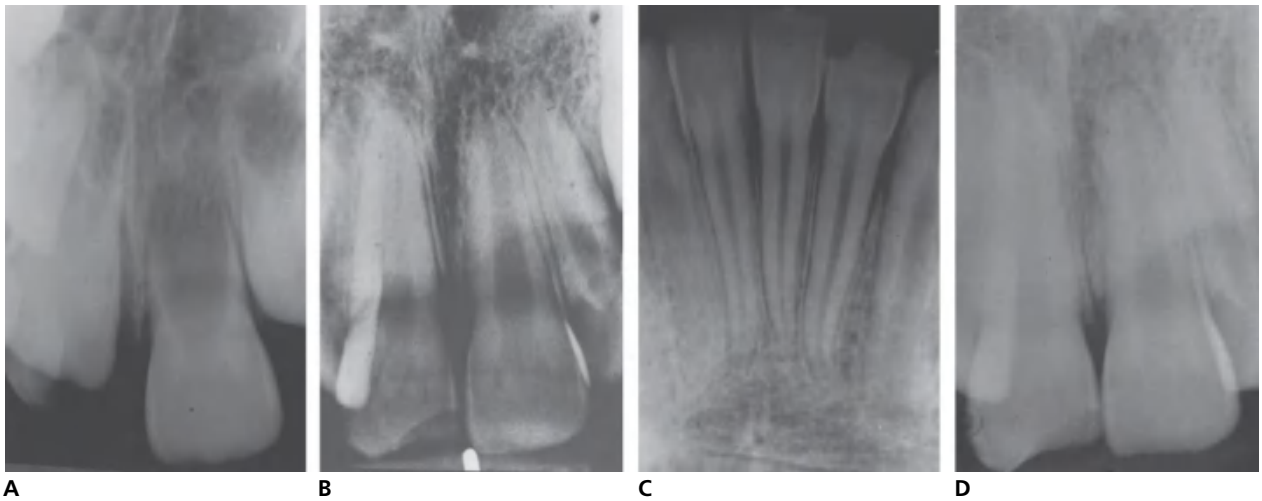



**Fig. 11.10** Individual with repeated trauma episodes. A. Avulsion of primary right maxillary central incisor at the age of 7 years. B. Crown fracture at the age of 9. C. Luxation of the right mandibular central incisor at the age of 10. D. New trauma to the right central incisor at the age of 12.

The relationship between TDI and ethnicity is not clear. Ethnic minorities tend to experience more financial adversities and live in more deprived areas. Therefore, it is difficult to disentangle the effects of these factors. Furthermore, few studies have recorded race or ethnicity of participants. Data from the USA showed a similar prevalence of TDI in different race/ethnicity categories (39).

In a Swedish study oral injuries were most frequent during the first 10 years of life, decreasing gradually with age and were very rare after the age of 30, whereas non-oral injuries were seen most frequently in adolescents and were present throughout life (36) (Fig. 11.11).

Data from several studies demonstrated that the majority of TDIs occur at childhood and adolescence. It is estimated that 71–92% of all TDIs sustained in a lifetime occur before the age of 19 years (184, 221–223). Holland et al. (224) reported a decrease of traumatic tooth injuries after the age of 24, and Shulman and Peterson (225) after the age of 30 years. Gassner et al. (226) also reported that 81.2% of all TDIs occur before 30 years of age; of that, almost 50% before 10 years of age. The *prevalence* and *incidence* of TDI, according to age and sex in a group of children from Copenhagen, followed continuously from the age of 2 to 14 years, is shown in Figs 11.7 and 11.8 (22). It can be seen that the first peak appears at 2–4 years of age. By the age of 7 years, 28% of the girls and 32% of the boys have suffered a TDI to the primary dentition. In the permanent dentition, a marked increase in the incidence of TDIs is seen in boys aged 8–10 years; while the incidence is rather stable for girls (227–229). This peak incidence in boys is probably related to the more vigorous play characteristic of this age group compared to girls (208, 209, 230–233). An increase in the rate and absolute number of oral and maxillofacial injuries was reported among persons aged 65 or more in New Zealand (234). This may be because an increasing proportion of older people are keeping their own natural teeth for life, and the risk of falling is high in old adults, which may lead to an increase in TDI. The scale of this problem is currently unknown as research reports on prevalence of TDI in older people are scarce.

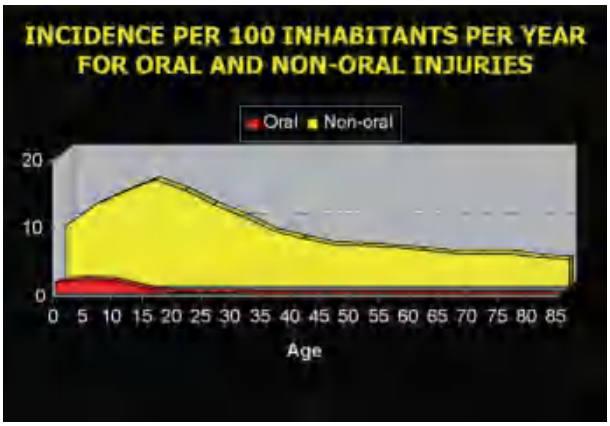



**Fig. 11.11** Incidence per 100 inhabitants per year for oral and non-oral injuries. From (36).

Very few reports on TDI have included socio-economic indicators and the results have been conflicting. Intriguingly, some studies reported a higher prevalence of TDI among adolescents from higher compared to those from lower socio-economic groups. The higher risk of dental injuries among children from higher socio-economic backgrounds may be related to greater ownership of bicycles, access to skiing, skateboards, roller-skating, horse-riding and swimming pools than those from low socio-economic groups (235). Low parenteral education has been reported to increase the risk of TDI and active and social children seems to be more prone to TDI (236, 237). Further research is needed to elucidate this relationship.

## Predisposing factors

Increased overjet with protrusion of upper incisors and insufficient lip closure are significant predisposing factors to TDI (211, 212, 225, 238–244). Studies have shown that TDIs are approximately twice as frequent among children with protruding incisors as in children with normal occlusion (245, 246), and that the greatest number of injured teeth in the individual patient is associated with protrusive occlusion (247) (Fig. 11.12). In a large meta-analysis of 54 studies, Petti concluded that the fraction of global TDIs attributable to

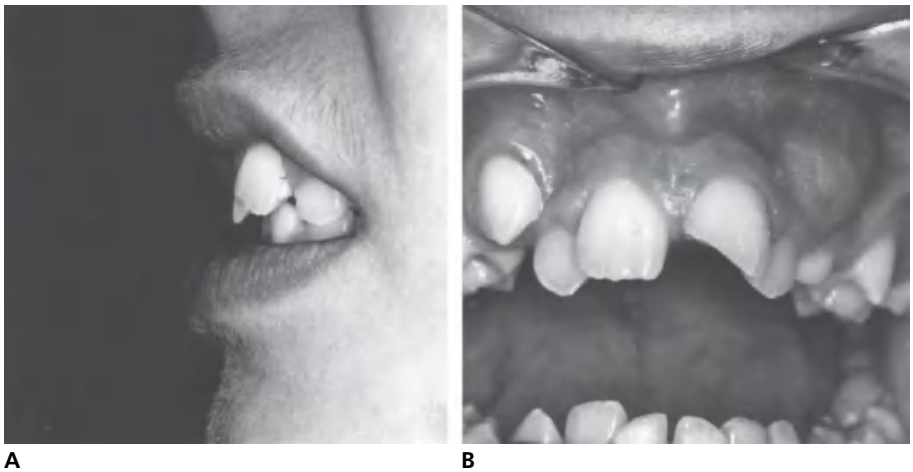



**Fig. 11.12** A. Protruding central incisors with incomplete lip coverage. B. Crown fracture of a left central incisor.

large overjet is 21.8% and that large overjet is co-responsible for 235 million global TDI cases (244).

Inadequate lip coverage has been shown to result in a three-fold risk for TDIs to the maxillary incisors compared to adequate lip coverage (87, 219), while other studies show no significant relation between inadequate lip coverage and TDIs (248, 249). The study showing a significant association (87) did not account for other risk factors. The other study carried out by Marcenes et al. (248) showed that the association between lip coverage and TDI was no longer significant after adjusting results for levels of deprivation experienced. Therefore, the conflicting findings may be due to the interaction between predisposing factors (size of the overjet and lip coverage) and environmental (playground design) and/or behavioral factors (risk taking). Unfortunately, very few studies have accounted for all these factors together. For orthodontic evaluation of these patients, the reader is referred to Chapter 28.

Other important factors such as risk-taking children, children being bullied, emotionally stressful conditions, obesity and attention-deficit hyperactivity disorder have been pointed out by Glendor (214). Some studies have shown that overweight and obese children run a higher risk of sustaining TDIs (52, 135, 250, 251).

### Teeth involved

Irrespective of type of study, the majority of dental injuries involve the anterior teeth, especially the maxillary central incisors; while the mandibular central incisors and maxillary lateral incisors are less frequently involved (1, 4, 8, 122, 196, 209–211, 233, 252–255). This preference for location also applies to the primary dentition (256–258).

Dental injuries usually also affect only a single tooth (39, 196, 228, 230, 252, 254, 255). However, certain trauma events, such as sports and automobile accidents, tend to result in multiple tooth injuries (4, 210, 259), especially among teenagers (205, 259).

Concomitant injuries to teeth and oral tissues are also common in patients presenting with oral trauma due to violence and traffic accidents.

### Types of injuries

The most common type of traumatic injury to permanent teeth is enamel fracture, followed by enamel and dentin fracture (8, 39, 122, 195, 208, 210, 227, 228, 233, 260). Other types of TDIs are less common. However, pulpal and periodontal tissue lesions were not assessed in some cross-sectional studies. In a prospective Swedish study, dental injuries were recorded in 92% of the patients, whereas soft tissue injuries were seen in 28%, and fractures involving the jaw bone in only 6% of all patients presenting with oral injuries (36). Findings from hospital- or surgery-based studies presented a different picture to studies in schools (261). This is because many people who sustain a small fracture do not seek treatment, and soft tissue injuries and the most serious injuries end up in hospital or dental surgeries for treatment. Hospital data tend to show facial bone fractures (37%), dentoalveolar injuries (50%) and soft tissue injuries (62%) most frequently (262). The number of injuries often exceeds the number of teeth due to multiple injuries for some teeth. Combination injuries are a common finding (8, 263–266). Concomitant crown fractures have been found more often with concussion, subluxation or intrusion. It is important to raise awareness of combination injuries since such injuries increase the risk of pulp necrosis and later complications (8, 263–266).

It appears that injuries to the primary dentition are usually confined to the supporting structures, i.e. luxation and exarticulation (4, 55, 227, 240, 267, 268). In some studies, however, the primary dentition was dominated by enamel fractures instead of injuries to the supporting structures (140, 147, 159, 195, 267, 269). This is probably due to the periodic visits of these patients to the emergency clinic, a fact that favors the recording of enamel fractures which might normally go undetected (269).

### Place of injury

The great majority of population-based studies reviewed showed that most TDIs occur at home, followed by at school and in the street or other public places (36, 134, 270, 271). It is disturbing when performing epidemiological studies that in most studies a high proportion reported that they could not remember where the injury event had occurred.

The place of injury varies in different countries according to local customs. In studies examining dental trauma in Iraq, India, Australia, Norway and England, it was found that the majority of injuries occurred outside school grounds (221, 229, 271, 272). Furthermore, a greater number of severe injuries occurred after school hours (36, 273).

### Seasonal variations

A relationship seems to exist between the time of year and occurrence of TDI. This seasonal variation in TDI is dependent upon local customs. While several studies have shown that the frequency of TDI increases during the winter months (36, 208, 228, 247, 262, 274), other reports indicate an increase in the summer months (257, 259, 260, 275–279). This may be related to the climate, where winter climate increases the risk for injuries caused by for example slipping on ice, or may be related to the geographic area of sampling and in either being a vacation area or an area that people leave for vacation, which may give contrasting results.

## Etiology

Clearly, TDIs are caused by a collision that can generate enough mechanical energy to produce the injury. Any object, animate or inanimate, in motion has energy that depends on its mass and speed. An increase in mass and/or speed increases energy. Thus, it is relevant to understand the vehicles and circumstances that generate mechanical energy,

**Table 11.11** World frequency distribution of traumatic dental injuries for permanent and primary teeth according to causes. The variables presented follow WHO nomenclature (for each category, the sum of the frequencies is not equal to 100% because the studies included in this analysis did not adopt the same criteria for the classification of causes). Based on studies 287–332.

| Cause | Number of subjects | Prevalence | 95% confidence interval | Number of studies used for the analysis |
|---|---|---|---|---|
| **No distinction between permanent and primary teeth** | | | | |
| Physical leisure activity | 10 481 | 19.4% | 12.6–27.3% | 15 |
| Collisions | 11 532 | 12.1% | 8.1–16.7% | 19 |
| Falls | 11 722 | 45.2% | 38.2–52.4% | 25 |
| Sports | 13 534 | 12.5% | 8.2–17.7% | 21 |
| Traffic accidents | 13 940 | 7.6% | 5.3–10.2% | 22 |
| Violence | 13 120 | 7.8% | 5.7–10.1% | 22 |
| Inappropriate use of teeth | 1 494 | 3.0% | 2.2–4.0% | 3 |
| **Permanent teeth** | | | | |
| Physical leisure activity | 2 948 | 20.8% | 14.0–28.6% | 8 |
| Collisions | 3 799 | 13.5% | 8.3–19.7% | 12 |
| Falls | 4 899 | 45.0% | 36.6–53.5% | 14 |
| Sports | 4 811 | 12.9% | 8.3–18.3% | 14 |
| Traffic accidents | 4 811 | 9.6% | 5.6–14.6% | 14 |
| Violence | 4 458 | 8.2% | 5.7–11.1% | 12 |
| Inappropriate use of teeth | 163 | 10.4% | 6.2–16.2% | 1 |
| **Primary teeth** | | | | |
| Physical leisure activity | 1 755 | 11.6% | 2.8–25.4% | 9 |
| Collisions | 3 647 | 15.2% | 10.4–20.4% | 13 |
| Falls | 6 392 | 57.9% | 49.7–66.0% | 17 |
| Sports | 1 281 | 5.8% | 3.2–9.2% | 6 |
| Traffic accidents | 3 453 | 2.9% | 1.4–4.8% | 10 |
| Violence | 895 | 2.7% | 0.8–5.9% | 5 |
| Inappropriate use of teeth | 1 474 | 0.5% | 0.2–1.0% | 1 |

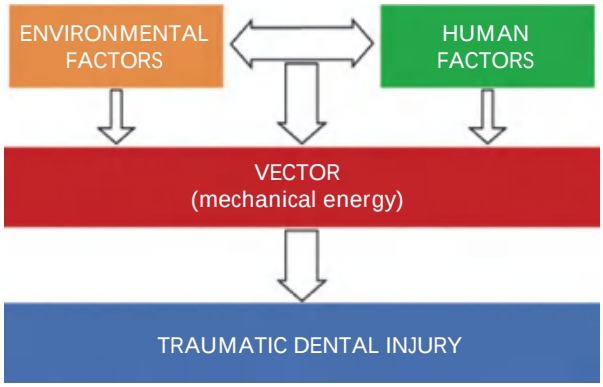



**Fig. 11.13** Environmental and behavioral causes of traumatic dental injuries.

which in turn cause a TDI. Violence, sports, traffic incidents and falls are commonly cited in the literature as causes of TDI (Table 11.11). In addition, it is important to learn about the 'causes of the causes' of TDIs. What makes people fall or have a collision? What makes people have an injury when driving, cycling or walking on the street? What makes people have an injury when playing sports? What makes people fight? The answers to these questions lead to the environmental and behavioral causes of TDIs (Fig. 11.13).

A major *environmental determinant* of TDI is deprivation. Data from the UK showed a higher prevalence of TDIs in deprived areas compared with the overall prevalence recorded in England (40). In addition, even within a deprived area, more deprived children have more TDIs than their less deprived counterparts. Overcrowding was the major environmental factor related to TDI (40, 89, 280). This seems logical, as deprived areas have less safe playgrounds, sport facilities, streets, schools and houses. This unsafe environment facilitates falls and collisions, which in turn lead to TDIs.

*Human behavior* also plays an important role in the occurrence of TDI. As expected, risk-taking children tend to have more TDIs than their less risk-taking counterparts. Other problem behaviors have also been related to TDIs. Odoi et al. (281) showed that children with peer relationship problems (e.g. being picked on or bullied by other children) have significantly more TDIs than other children. Interestingly, the same study reported a tendency for children who show prosocial behaviour to have less dental injuries than their counterparts. Oldin reported a weak influence of temperamental factors and that active and social children experienced more TDIs (101, 236, 237). Lalloo (282) reported that hyperactive children have significantly more TDIs than non-hyperactive children. Conversely, Odoi et al. (281) failed to demonstrate a significant relationship between TDI and hyperactivity. This conflicting result is easy to understand and suggests that environment plays a stronger role in determining TDI than human behavior. A hyperactive child can express this without risk if the environment is safe.

Emotionally stressful states measured using biologic markers or questionnaires have also been reported to be associated with TDI (206, 283, 284). Nicolau et al. (285) applied the life course approach to further elucidate the causes of TDI and concluded that adolescents who experienced adverse psychosocial environments had more traumatic dental injuries than their counterparts who experienced more favorable environments. This included living in a non-nuclear family and experiencing high levels of paternal punishment (285).

Broad categories like those mentioned may mask the actual causes of TDI, because they do not assess intention. For example, a fall from tripping (unintentional) differs from falls from pushing (intentional). The latter should be considered as a minor form of violence. Similarly, it is common to record collision when a child is pushed against another child or object, in particular the school water fountain. If human intent had been assessed in previous studies, these events would have been recorded as bullying, or minor violence, rather than collision. Thus, it is possible that the role of violence in the occurrence of TDI has been considerably underestimated (134). Of further concern is the high proportion of children who tend to report 'unknown cause' in most studies. Victims tend to report unknown cause when an injury is due to violence. The overrepresentation of accidents compared to violence at home may result from a difficulty in finding the actual rate for injuries associated with child, spouse and elderly abuse.

Worldwide, physical leisure activities, violent incidents and traffic accidents accounted for most TDIs among adolescents. Rough playing with others, biting hard items and inappropriate use of teeth also contribute to having a TDI, but to a lesser extent. Falls and collisions tend to be the most prevalent events associated with TDIs reported in the literature. These very broad TDI groups may be classified based on etiology, taking into account the TDI event or the intent as proposed by WHO in the document *Dictionary for Minimum Data Sets on Injuries* (286). A TDI event is defined as the activity related to the occurrence of the TDI. It includes falls, collisions, physical leisure activities, traffic accidents, rough playing with others, violence, inappropriate use of teeth and biting hard items. Less common events are recorded as 'other events'. The last two TDI events were added for the sake of completeness. They are relatively common TDI events, but not applicable to general injuries. Intent represents the human involvement in the occurrence of the TDI event; it includes unintentional TDI, intentional TDI (self-harm events and violent events) and iatrogenic (doctor's mistreatment) TDI.

Falls are the main cause of TDIs, being responsible for approximately half of all TDIs among adolescents and adults, and even more among children worldwide (Table 11.11). Physical leisure activities, particularly playing, bicycling and skateboarding, are the second most frequent TDI cause among adolescents and young adults, being responsible for 20% of all traumatic events. Collisions against objects and people rank second among children and represent 15% of all traumatic events. Sport playing also is frequently responsible for TDI to permanent teeth, accounting for 13% of all injuries.

A fraction ranging between 5% and 10% of all TDIs among adolescents and young adults is due to violence, such as assaults, fighting and even physical abuse. One should be aware of that untreated traumatic dental injuries may, indeed, be considered one of the typical signs of physical abuse (333), however their frequency without coexisting signs is relatively modest and other injuries of the soft tissues of the head and face, such as hematoma, ecchymosis, laceration, oedema, burn, and scratches, are often detectable. The latter lesions tend to prevail, being present in 50–65% of physically abused children and adolescents. Thus, if there are untreated TDIs and there are no injuries of the soft tissues of the head and face, physical abuse could be ruled out (334), while neglect could be suspected (335).

## Falls and collisions

Falls are common among children and old adults. Primary teeth TDIs increase substantially with the child's first efforts to move about. Due to lack of experience and coordination, frequency increases as the child begins to walk and tries to run. A prospective survey of injuries to pre-school children in a home setting showed that oral and head injuries were sustained following falls on or from stairs or pieces of furniture (336). They were incurred on stairs, in garages or on/from verandas, and pre-school children aged 1 year were more often involved than expected by chance. The incidence of TDIs reaches its peak just before school age and consists mainly of injuries due to falls and collisions (4, 36, 227, 252, 254). Home is the most common place of injury for pre-school (36, 205) and school age children. Falls or the child being struck by an object tend to be reported as the most common causes of injury (36, 220, 271, 293, 337). This is not surprising as one cannot imagine any other way to sustain a dental injury. However, the event that caused the fall or collision is the actual cause of the TDI, and should have been recorded.

A refined assessment of the causes of TDIs would be expected to identify a smaller proportion of TDIs due to falls. The result may be a higher proportion of TDIs due to violence. Victims of abuse tend to give a vague history, such as a fall, when asked about the cause of the injury. Furthermore, falls among young children are mainly related to neglect of the child and can often be prevented by better supervision from parents and safe house design.

## Physical leisure activities

Injuries during the teenage years are often due to sports (36, 210, 211, 228, 338–345). Federation Dentaire International (FDI) has organized sport in two categories due to risk for TDI: *high risk sports* that include American football, hockey, ice hockey, lacrosse, martial sports, rugby and skating; and *medium risk sports* including basketball, diving, squash, gymnastics, parachuting and waterpolo (346). Studies confirmed that contact sports, such as ice hockey, soccer, baseball, American football, basketball, rugby, wrestling and handball (83, 232, 347–349) are the major TDI events (Fig. 11.14). As many as one-third of all dental injuries and

**Fig. 11.14** The unprotected ice hockey player runs a high risk of injuries due to collision with other players or being hit by a puck or a hockey stick.

**Fig. 11.15** Horseback riding-related injuries. In the jump, the rider's face can collide with the horse's neck, usually resulting in luxation injuries.

**Fig. 11.16** A kick by a horse in the face normally results in serious injuries, e.g. exarticulation, intrusion and sometimes jaw fractures.

up to 19% of injuries to the head and face are sports related (350–355). Horseback riding, a popular sport in many countries, is a significant source of injury. In a single season, 23% of all riders sustained injuries of various types, including dental and maxillofacial injuries (356, 357). Figures 11.15 and 11.16 illustrate the typical trauma situations related to horseback riding. There is little doubt that special precautions, such as the use of sturdy helmets, can reduce the number and severity of these accidents (356). Similarly, the provision of a well-fitting mouthguard may possibly reduce the occurrence of TDI. However, there is still insufficient evidence that planned intervention is effective in reducing the prevalence or incidence of sports-related injuries to the mouth and face; and much remains to be elucidated regarding attitude and effective use of protective equipment (358). Sports injuries, as well as measures for their prevention, are described in Chapter 37. Physical leisure activities at playgrounds also account for a significant proportion of TDIs (205, 228, 230, 271, 275). Most of the resultant injuries are characterized by a high frequency of crown fractures (4,

**Fig. 11.17** Automobile accidents in which the front seat passenger hits the dashboard result in soft tissue injuries and damage to the supporting bone.

**Fig. 11.18** Bicycle accidents usually result in multiple crown fractures and soft tissue injuries.

210). This type of TDI can be dramatically reduced through environmental changes, including better playground design and construction.

## Traffic accidents

Traffic accidents include pedestrian-, bicycle- and car-related injuries. Facial and dental injuries resulting from automobile accidents are seen frequently (Fig. 11.17). The front seat passenger is particularly prone to facial injuries if not using a seat belt. These traumas are caused by high energy often resulting in multiple dental injuries, injuries to the supporting bone, and soft tissue injuries to the lower lip and chin (4, 359–361). This pattern of injury is seen when the front seat passenger or the driver hits the steering wheel or dashboard (362). A study in road traffic accidents from Nigeria has shown that occupants of commercial vehicles were the ones most likely to receive maxillofacial injuries, especially the rear seat occupants (363). Children seated on, or standing on, the front seat are in a very dangerous position, as dental injuries often occur as a result of being thrown against the dashboard during sudden stops. Children with dental injuries and involved in traffic accidents have a 2.4-fold risk for bone fractures compared to accidents during play, sport and assaults (262). Enforcement of speed limits for cars and the use of seat belts, airbags and special car seats for smaller children in motor vehicles have reduced severe injuries, but new types of facial trauma have been reported and are attributable to airbag deployment in cars (364, 365).

Bicycle-related injuries have been widely reported. These injuries usually result in severe trauma to both the hard and soft tissues due to the high velocity at the time of impact (366) (Fig. 11.18). Bicycle-related oral injuries seem to be more common up to the age of 14 years, compared to non-oral injuries (367). The use of cycle helmets may reduce the risk of serious facial injury to the upper and middle face regions by approximately 65% compared to non-users, but no protection is provided for the lower face and jaw (368). Patients sustaining this type of trauma frequently experience severe facial and dentoalveolar trauma in addition to injuries to the upper lip and chin (366, 369) (Fig. 11.18). To reduce dentoalveolar trauma there is a need to extend the area of helmet coverage (369).

## Inappropriate use of teeth

Few studies have included this category. However, those that have found that many individuals sustain a TDI when using the teeth as a tool. Nicolau et al. (98) reported that 6% of

TDIs were due to inappropriate use of teeth and Tapias (370) reported 8.5%, while Traebert et al. (134) found a smaller proportion of 3.3%. The most common inappropriate uses of teeth reported in the literature are biting a pen, opening hair clips, opening packets of savory snacks, trying to fix electronic equipment or changing batteries, cutting or holding objects and opening screw top bottles.

Another event related to the occurrence of TDIs is biting hard items. TDIs have been shown among patients wearing piercing jewels. Patients and health care professionals should be informed of procedures and risks associated with tongue and oral piercing (371–373). Limited information has shown that piercings can result in chipping and fracturing of teeth and restorations, pulpal damage, cracked tooth syndrome and tooth abrasion (374). Information around dental/oral and systemic complications from tongue piercing has to date primarily been presented as case reports. Only one prevalence study has been published. Campbell et al. (373) showed a chipped teeth prevalence of 19.2% among individuals wearing tongue piercings, especially to molars and premolars, and to individuals wearing short stem barbells. They also showed an increased risk with years of wear.

## Presence of illness, physical limitations or learning difficulties

Attack of illness is an uncommon cause of TDI in the general population, but common in individuals suffering from epilepsy, cerebral palsy, anemia and dizziness. Dental trauma has been reported with cerebral palsy (375). Epileptic patients present special risks and problems with regard to dental injuries (376, 377). A study of 437 such patients in an institution showed that 52% had suffered TDIs, many of which were of a repetitive nature. In one-third of the cases, the injuries could be directly related to falls during epileptic seizures (378) (Fig. 11.19). Another study showed that 10% of patients with epilepsy, who during the past year had suffered from seizures resulting in injury, had suffered a TDI (376). Epileptic seizures have been shown to be the third most common medical incident in dental surgeries (379, 380). There is also a risk for the dental personnel to be infected from human bites of a patient suffering an epileptic seizure (381–383).

A very high frequency of dental injuries has been found among patients with learning difficulties (338), a phenomenon probably related to various factors such as lack of motor coordination, crowded conditions in institutions or concomitant epilepsy. O'Donnell (384) indicated that totally blind children and young adults in Hong Kong were at greater risk of sustaining anterior tooth injury than were sighted and partially sighted populations. Hearing impaired children, compared to visually impaired children, have been found to have significantly more TDIs compared to children as a whole (385). This difference is probably due to greater opportunities to play and move around for the hearing impaired children compared to the visually impaired children.

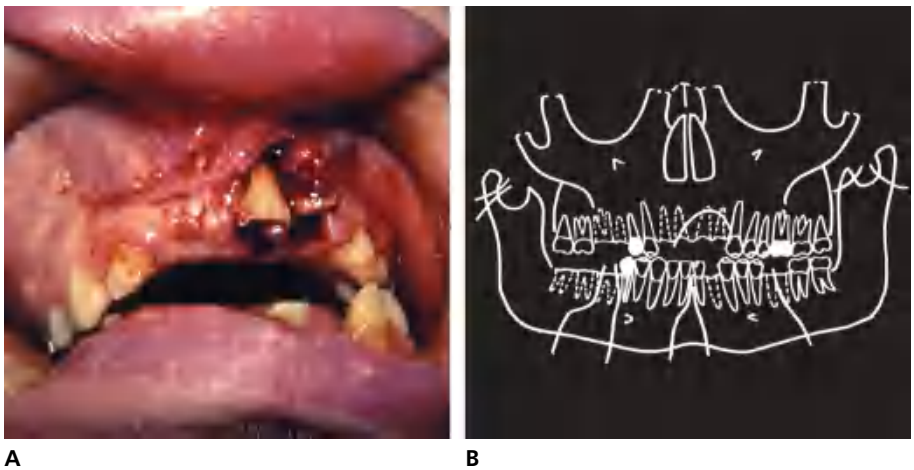
An unusual type of injury is root fracture without apparent reason affecting individuals with dentinogenesis imperfecta (386, 387). The explanation for this phenomenon could be the decreased microhardness of dentin (388) and abnormal tapering of the roots (Fig. 11.20).

## Physical abuse

A tragic cause of oral injuries in small children is manifested in the battered child syndrome (or non-accidental injuries (NAIs)), a clinical condition in infants who have suffered serious *physical abuse* (435) (Fig 11.21). The etiologic and diagnostic aspects of child abuse are further described in Chapter 10.

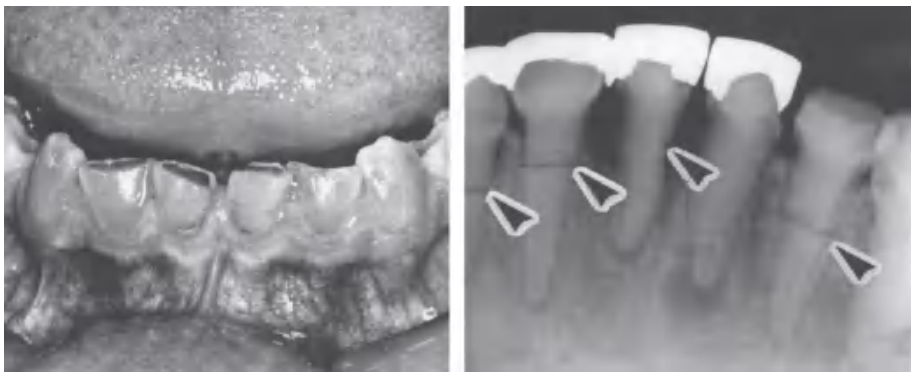
*Assaults*, together with road accidents, comprised 71% of maxillofacial injuries in a study in the UK, as reported by Dimitroulis and Eyre (389). Injuries to the face represented 62% of all injuries due to assault (390). Injuries from fights are prominent in older age groups and are closely related to alcohol abuse (36). This type of trauma usually results in a particular injury pattern characterized by luxation and avulsion of teeth as well as fractures of roots and/or supporting bone (4) (Fig. 11.22).

Domestic violence is a universal problem resulting in trauma to the facial region (Fig. 11.23). Establishing the victim as a battered person is complicated by the fact that these



A B

**Fig. 11.19** A 32-year-old male who has sustained five trauma episodes within 7 years due to epilepsy. These episodes involved multiple fractures of the teeth and mandible, as well as an alveolar fracture of the maxilla. A. Intruded maxillary incisors. B. Diagram with indication of fracture lines as well as missing teeth due to trauma. Reproduced from (378) with permission.



A B

**Fig. 11.20** Spontaneous root fractures in a 15-year-old patient with osteogenesis and dentinogenesis imperfecta. A. Clinical view of incisors at the age of 14 years. The color of the teeth is bluish-brown. Note the marked attrition. The incisal edges of the incisors were later restored with cast inlays. B. Root fractures affecting three incisors and the left canine at the age of 15 years (arrowheads). There was no evidence of trauma in the history. The right central incisor was later extracted. Reproduced from (386) with permission.

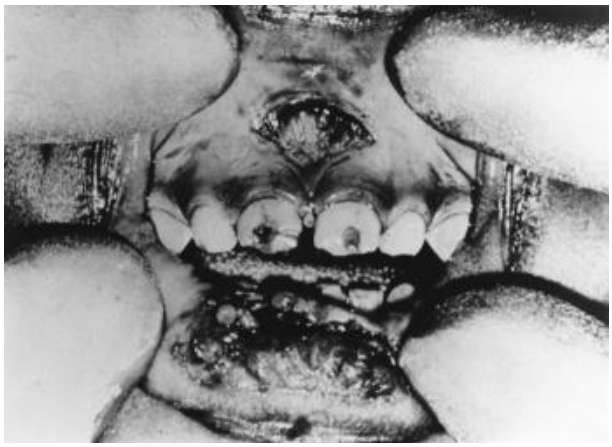


**Fig. 11.21** Battered child syndrome in a 2.5-year-old girl. The mother suffered from depression and shook the child violently against a fireplace. The child died and was found to have contusion of the brain with intracranial hemorrhage. The child also had partly healed fractures at the lower end of each radius, which had been treated by an orthopedic consultant 1 month previously; but battering was not suspected at that time. The oral injuries consisted of laceration of the mucosa of the lower lip and tearing of the frenum. Reproduced from (435) with permission.

victims will seldom volunteer that information. Social agencies for the care and counselling of these victims exist in many countries and should be used (390).

A disgraceful and apparently increasing type of injury is represented by trauma to the oral and facial regions of *tortured* prisoners. Investigations have shown that the majority of these victims, apart from other atrocities inflicted upon their persons, have suffered from torture involving the oral region (391) (Fig. 11.24). A study has described the findings from examinations of 34 former prisoners from six countries who had all been subjected to torture involving the facial region (391). The most common type of torture was beating, which resulted in loosening, avulsion or fracture of teeth and soft tissue laceration. Deliberate tooth fractures with forceps were also seen. Furthermore, electrical torture was described in which electrodes were attached to the teeth, lips, tongue and soft tissue over the temporomandibular joint (Fig. 11.24). In the latter instance, very forceful occlusion resulted due to muscle spasm. The result was loosening and fracture of teeth as well as severe pain in the muscles and the temporomandibular joint.

Under the auspices of Amnesty International, working groups of dentists and physicians have been formed in several countries with the aim of gathering evidence to document the torture of prisoners.

## Iatrogenic procedures

*Prolonged intubation* is a procedure which is used in the care of prematurely born infants. The prolonged pressure of tubes against the maxillary alveolar process is an iatrogenic procedure that may lead to a high frequency of developmental enamel defects in the primary dentition (392, 393) (see Chapter 23) as well as injuries to the tooth germs of the first and second dentition and deformation of the maxillary skeleton (394).

The incidence of *perianesthetic dental injury* varies from 0.04% to 12% (395), and is considered to be the most frequent anesthesia-related cause for claims in the UK, representing approximately one-third of all confirmed claims (396). A retrospective study in France showed a mean incidence of 9.5 accidents per 100 anesthetists per year (397), while other studies have shown a yearly incidence ranging from 1:150 to 1:1000 cases (398, 399). The maxillary incisors are the most commonly affected (398–403), especially on the left side (397, 398, 403). Tooth injuries range from microfractures of the natural tooth substance to actual avulsion (397, 398, 403, 404), and include pulpal necrosis (403) and damage to crowns or bridges (397, 398, 403). Skeie and Schwartz (405) have shown a spectrum of fractured teeth in 47%, dislocated or mobile teeth in 41%, and avulsed teeth in 10% of patients with perianesthetic dental injuries. Givol et al. (406) showed that 72% of the patients were 50–70 years of age; and the most common injury was to upper (87%) and lower incisors (12.5%). Avulsion of teeth was the most common type of injury (48%), followed by crown fracture (22%); and 65% of the teeth could not be restored. The major risk factor for dental injury seems to be pre-existing poor dentition (406). The risk of injuries to the teeth can be reduced by a presurgical inspection of the oral cavity and evaluation of the individual anatomic conditions in the head and neck region, which might interfere with endotracheal intubation (393, 407). Using a toothguard has been shown to be a simple way of preventing dental trauma during general anesthesia and to have a low effect on the difficulty of intubation (408).

**Fig. 11.22** Kicks to the face during assaults result in severe damage to teeth and supporting structures.

**Fig. 11.23** A 19-year-old woman battered by her husband. The patient was beaten around the face and suffered lacerations of the lower lip and an alveolar fracture confined to the mandibular incisor region.

A B

**Fig. 11.24** A 35-year-old political refugee who was tortured by having live electrodes attached to the oral mucosa. Furthermore, the lower incisors were avulsed during 'interrogation'. The patient still suffers profound pain in the masticatory muscles, 9 years after being tortured. B. A 35-year-old male political refugee who had been tortured during his imprisonment. The man was beaten by security police. During torture, he fell and fractured and luxated two central incisors. No treatment was given. The radiograph is indicative of his condition 1 year after torture. It was taken at an examination by the medical board of Amnesty International. Courtesy of Drs P. Marstrand, B. Jerlang and P. Jerlang, Amnesty International, Copenhagen.

However, such recommendations would have to be subject to controlled cost–benefit analysis prior to their widespread application (394), especially since Skeie and Schwartz (405) have found that 0.062% of patients in general anesthesia suffered dental injury despite the use of a toothguard, compared to 0.063% not wearing a toothguard. At present, the use of a toothguard for each intubation does not seem reasonable; but tooth fragility must be researched (409). The cost of restoring a perianesthetic dental injury has been estimated to be approximately $2000 per patient in Israel (400).

**Fig. 11.25** Direct tooth trauma without soft tissue injury. The impact was absorbed by the protruding central incisors leading to fractures. From (4).

**Fig. 11.26** Induced tooth trauma. The impact was transferred via the chin to the dental arches and inflicted crown-root fractures in both right premolars by the forceful occlusion. From (4).

## Mechanisms of traumatic dental injuries

The exact mechanisms that produce the mechanical energy leading to dental injuries are, for the most part, unknown and without experimental evidence. A few experimental attempts have been made to construct *in vitro* models for fracture resistance of extracted human teeth (410, 411). Injuries can be the result of either direct or indirect trauma (2). *Direct trauma* occurs when the tooth itself is struck, e.g. against playground equipment, a table or chair (Fig. 11.25). *Indirect trauma* is seen when the lower dental arch is forcefully closed against the upper, as by a blow to the chin in a fight or a fall. While direct trauma usually implies injuries to the anterior region, indirect trauma favors crown or crown-root fractures in the premolar and molar regions (Fig. 11.26), as well as the possibility of jaw fractures in the condylar regions and symphysis. Another effect of a blow to the chin can be a forceful whiplash effect to the head and neck, occasionally leading to cerebral involvement. This condition can be revealed during the clinical examination (see later in this chapter).

The following factors characterize the impact and determine the extent of injury (412).

(1) *Energy of impact*. This factor includes both mass and velocity. Examples of these combinations are a force of high velocity and low mass (gunshot) (Fig. 11.27) or of high mass and minimal velocity (striking the tooth against the ground). Experience has shown that low velocity blows cause the greatest damage to the supporting structures, whereas tooth fractures are less pronounced. In contrast, in high velocity impacts the resulting crown fractures are usually not associated with damage to the supporting structures. In these cases, the energy of the impact is apparently expended in creating the fracture and is seldom transmitted to any great extent to the root or supporting structures of the tooth (4).

(2) *Resilience of the impacting object*. If a tooth is struck with a resilient or cushioned object, such as an elbow during play, or if the lip absorbs and distributes the impact, the chance of crown fracture is reduced while the risk of luxation and alveolar fracture is increased (4) (Figs 11.28 and 11.29).

(3) *Shape of the impacting object*. Impact with a sharp object favors clean crown fractures with a minimum of displacement of the tooth, as the energy is spread rapidly over a limited area. On the other hand, with a blunt object, impact increases the area of resistance to the force in the crown region and allows the energy to be transmitted to the apical region, causing luxation or root fracture.

(4) *Direction of the impacting force*. The impact can meet the tooth at different angles, most often hitting the tooth facially and perpendicular to the long axis of the root. In

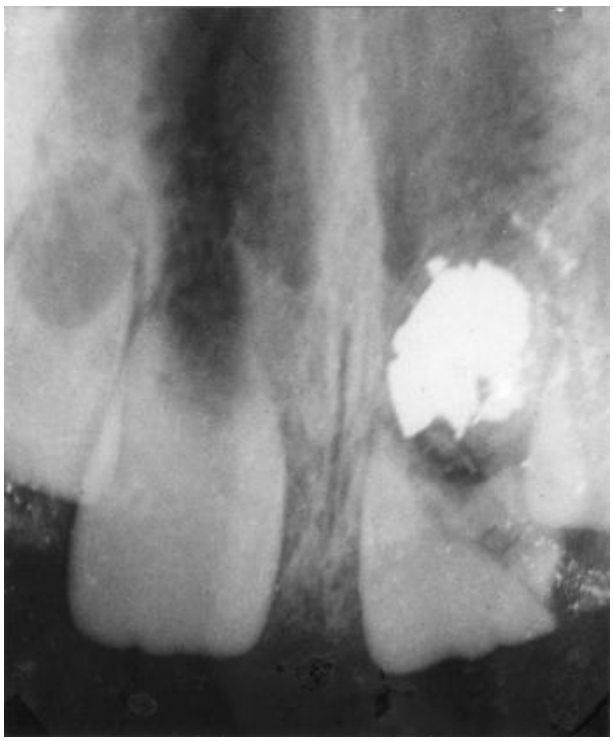


**Fig. 11.27** Multiple dental fractures caused by a gunshot.

this situation, typical cleavage lines will be encountered, as demonstrated in Fig. 11.30. With other angles of impact, other fracture lines will arise.

When considering the direction and position of fracture lines caused by frontal impacts, the fractures fall easily into four categories (Fig. 11.31).

(1) Horizontal crown fractures.
(2) Horizontal fractures at the neck of the tooth.
(3) Oblique crown-root fractures.
(4) Oblique root fractures.

Engineering principles can provide a description of the forces involved in injuries caused by frontal impacts. Frontal impacts to anterior teeth generate forces that tend to displace the coronal portion orally. Under certain conditions, such as blunt impacts and high resilience of tooth-supporting structures in young individuals, a tooth is more likely to be displaced orally without fracturing, as the energy of the impact is absorbed by the supporting structures during displacement (Fig. 11.32).

A different situation arises if the bone and periodontal ligament resist displacement (Fig. 11.33A). The root surface is forced against the bone marginally and apically (a and b), creating high compressive forces. As the tensile and shearing

**Fig. 11.28** Direct trauma to the upper lip. The impact was transmitted through the lip, resulting in extrusive luxation of right incisors and laceration of gingiva. The upper surface of the lip shows minor lacerations in the area which was in contact with the tooth surfaces during transmission of the trauma. From (4).

A B

**Fig. 11.29** Direct trauma to the lower lip. A. Force of a frontal blow to the lower lip was transmitted through the lip to the lower incisor region, resulting in a fracture of the alveolar process. B. The oral mucosa is lacerated in areas where the incisal edges contacted the labial mucosa during impact (arrows). From (4).

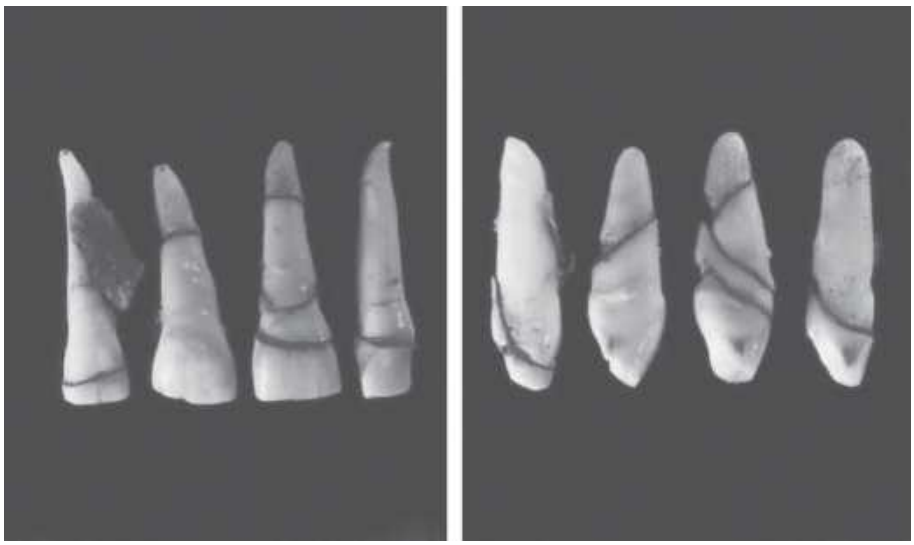



**Fig. 11.30** Fracture lines in four extracted maxillary incisors inflicted by a frontal impact from an ice-hockey puck. A. Labial aspect. B. Lateral aspect; note that the courses of the crown-root fractures and the root fractures are approximately perpendicular to each other.

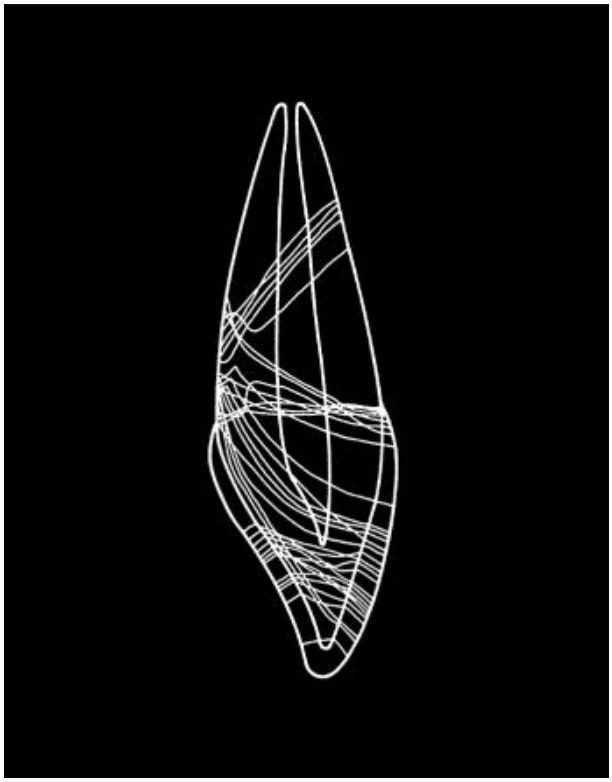


**Fig. 11.31** Facio-lingual orientation of 33 fracture lines.

strength of the brittle dental tissues are much lower than the compressive strength, shearing strains develop between the two zones of the opposing forces, and the root is fractured along the plane joining the two compression areas (413) (Fig. 11.33B).

Figure 11.34 illustrates a condition possibly leading to horizontal fractures at the level of the gingival margin. The tooth is firmly locked in its socket so that stresses in the shearing zone will not be as high as those shown in Fig. 11.33. The impact will, therefore, induce a pure bending fracture at the site of maximum bending stress, i.e. where the tooth emerges from its supporting structures (Fig. 11.34B). Tensile strains on the facial surface of the crown usually result in horizontal infraction lines in the cervical enamel (Fig. 11.35). Horizontal fractures at the cervical area usually affect the maxillary lateral incisors, probably due to their firm, deep anchorage in alveolar bone.

In Fig. 11.36, the stresses possibly causing oblique crown-root fractures are illustrated. The oblique fracture line follows a course along the inclined tensile stress lines developed between the compressive areas a and c. This line of fracture is supported by experiments using brittle materials, such as concrete (Fig. 11.37).

In Fig. 11.38, the same compressive forces and tensile strains as in Fig. 11.36 are present, but are not great enough to cause an oblique fracture, so that the cleavage line follows the shortest possible route, resulting in a horizontal fracture of the crown. The area of contact between the impacting object and the enamel may show a shallow notch surrounded by radiating infraction lines (see Chapter 13). The orientation of the enamel prisms determines the course of the fracture line in enamel, while the direction of the fracture in dentin is primarily perpendicular to the dentinal tubules (Fig. 11.39). The theoretical explanation for this has been sought in experimental procedures testing the fracture properties of enamel and dentin (414, 415). It has been found that enamel is weakest parallel to the enamel rods and that dentin is most easily fractured perpendicularly to the dentinal tubules (415).

Penetrating lip lesions occur when the direction of impact is parallel to the axis of the mandibular or maxillary incisors (Figs 11.40 and 11.41). In these cases, foreign bodies such as gravel and tooth fragments are frequently found in the lip lesion. The management of this type of wound is described in Chapter 18.

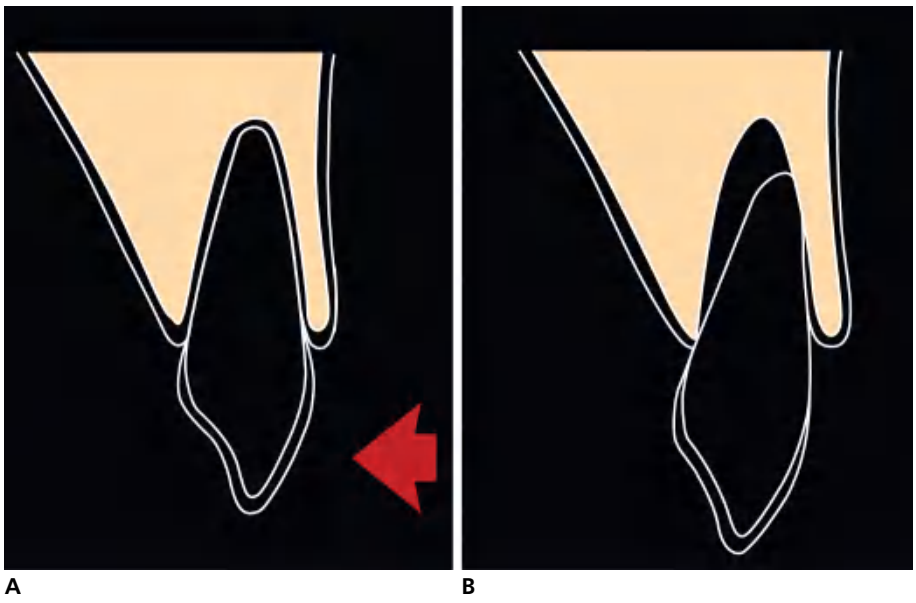



**Fig. 11.32** Pathogenesis of extrusive luxation. A. Frontal impact to the facial surface of an anterior tooth tends to displace the tooth lingually. B. If the energy is absorbed by the tooth-supporting structures, the tooth is displaced.

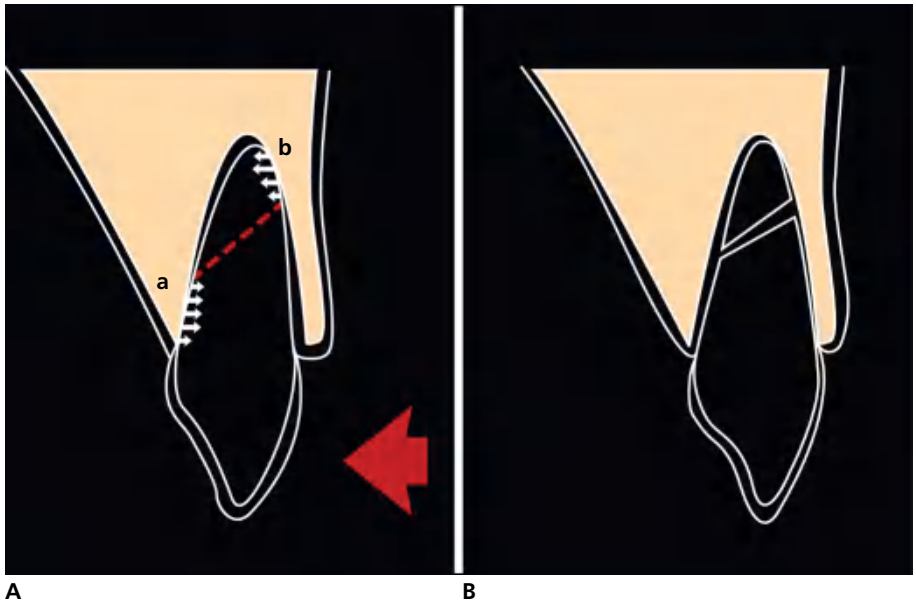



**Fig. 11.33** Pathogenesis of a midroot fracture. A. Bone and periodontal ligament resist displacement from a frontal impact. Compressive forces are exerted upon the root surface at a and b and tensile strains develop across the line connecting a and b. B. A root fracture occurs in the shearing zone, at the area of tensile strain.

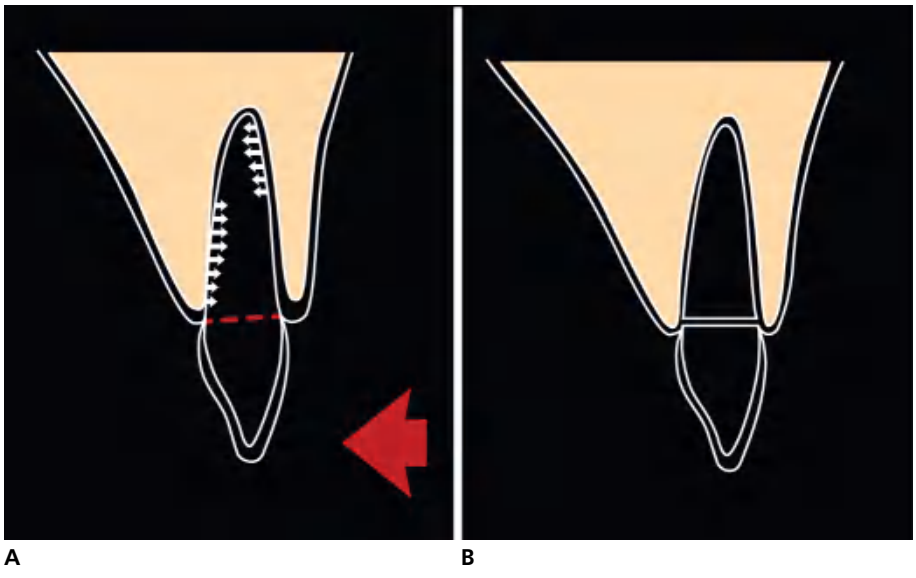



**Fig. 11.34** Pathogenesis of a cervical root fracture. A. The tooth is firmly locked in its socket. B. A pure bending fracture occurs at the site of maximum bending stress.

## Treatment need

Epidemiological studies have demonstrated that the treatment needs of TDIs are not, in general, properly met. This applies to both poor developing countries and rich developed countries.

In Britain only 10–15% (199, 416) and in Finland only 25% of children who sustained TDIs had received treatment (238). Onetto et al. (205) reported a tendency for neglecting treatment of serious TDI in very young patients. In a study from Tanzania, 21% of the children had at least one type of untreated TDI, where the highest percentage (26%) was observed among children with high socio-economic status (417). However, almost all (94%) of the teeth showing untreated TDIs were enamel or enamel-dentin fractures. The low rates of treatment observed worldwide may be because TDI is not perceived as a disease. Another aspect that could enhance treatment neglect is the dentists' lack of knowledge regarding the treatment of TDIs (418). In addition, dental school curricula and health authorities tend to focus resources on other oral health conditions but not on treatment of TDIs.

One may argue that these studies did not assess normative treatment need and that most injuries may be too small to need treatment. Alternatively, it may be argued that epidemiological studies too often include only visual assessment and therefore tend to underestimate the need for treatment. Marcenes and Murray (89) evaluated the need for treatment in addition to assessing untreated damage, and showed that not all untreated dental injuries needed treatment because some injuries were minor. Conversely, the study also showed that not all treated injuries were satisfactory and some needed retreatment. A more accurate estimation of treatment need used in these studies confirmed that the treatment of TDIs was neglected, as 56% of incisors that sustained damage were assessed as needing treatment. Another study, using

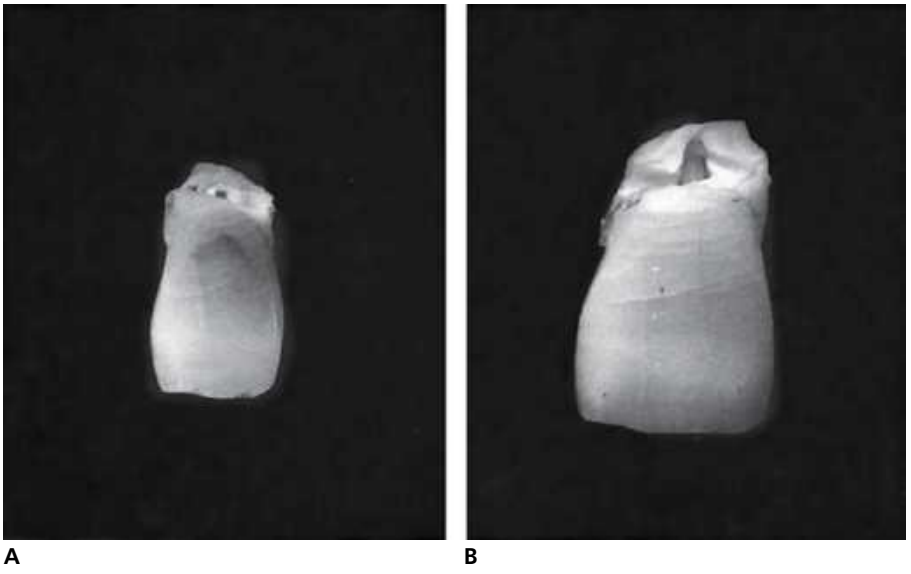



**Fig. 11.35** A. Horizontal infraction lines in the cervical area of a fractured maxillary incisor. B. High magnification of the cervical area.

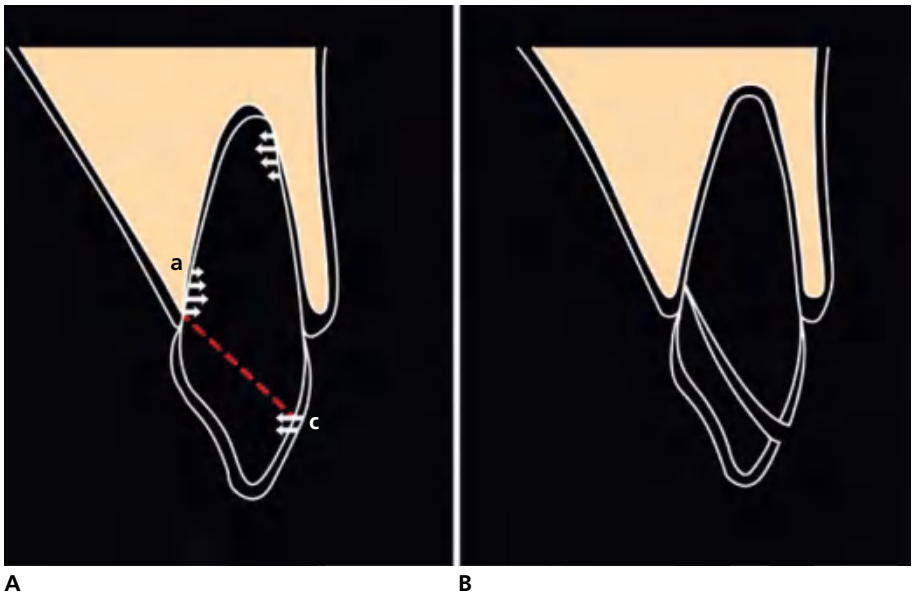



**Fig. 11.36** Pathogenesis of a crown-root fracture. A. Tensile strain develops across the line between the compressive areas a and c. B. An oblique fracture occurs in the area of tensile strain.

radiographs to assess the treatment provided, found that only 47% of damaged teeth had received treatment, of which 59% was inadequate (418). A study carried out in a developing country (Jordan) has shown that only 3% of the injured teeth were treated, while 64% of the teeth needed treatment (419).

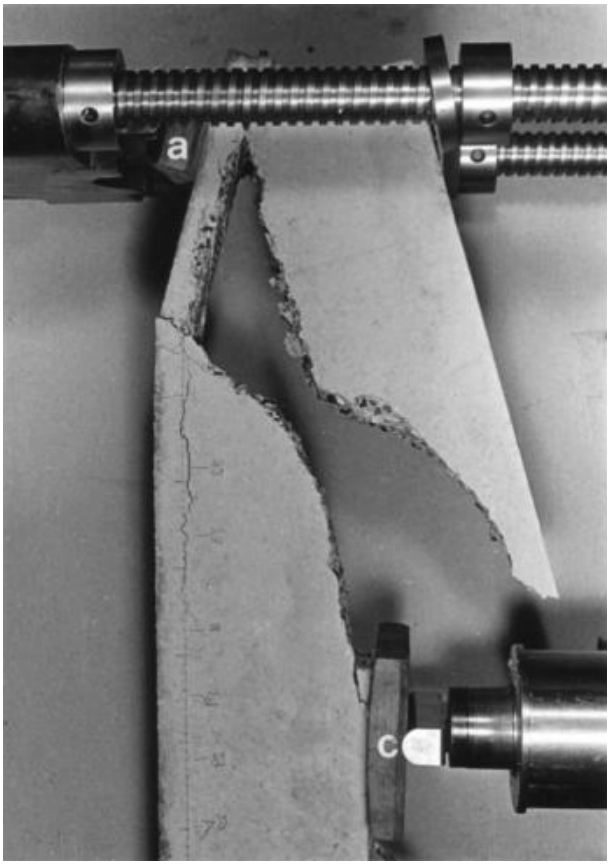



**Fig. 11.37** Experimental model in concrete showing an oblique fracture connecting the area of compression c and supporting area a. Courtesy of Dr. H. H. Krenchel, Structural Research Laboratory, Technical University, Copenhagen.

Several studies confirmed that treatment of TDI often is neglected. Marcenes et al. (201, 235) have shown in studies in Brazil and Syria that a majority of the traumatized teeth were not treated. In another study it was reported that nearly half of the sample who had experienced injuries to the permanent anterior teeth were not taken to the dentist for evaluation or treatment (248). A study from central Taiwan also showed that a minor number of the subjects had sought dental or medical help (220).

## Organization of emergency care

Traumatic dental injuries account for a large proportion of oral-related emergency treatment. Analysis of after-hours emergency dental service data during one calendar year at the Royal Belfast Hospital for Sick Children revealed that traumatic injuries to teeth, supporting structures, gingiva and oral mucosa represent the second most frequent complaint. While 39% visited the service due to dental injuries, 49% visited it due to all types of toothache including abscess (420). Similar proportions are reported in other studies. Oral injuries tend to occur in leisure time (421), in the middle of the week and in the afternoon (0–15 years) and during week-ends and late evening (16–30 years) (87). Emergency room reports also indicated a greater frequency of TDIs in the summer months, whereas reports from Scandinavian public dental health services indicated a drop in the same period (36, 193, 230, 343). This discrepancy could be related to the fact that most people tend to leave the bigger cities for summer vacations and if a TDI occurs, they visit a local dental surgery.

For this reason it is important that a dental emergency service is organized in each geographic region. Ideally, such a service should be provided on a 24-hour basis. During office hours, dental clinics can provide emergency service. However, because the majority of dental injuries occur outside office hours, other solutions must also be provided. An

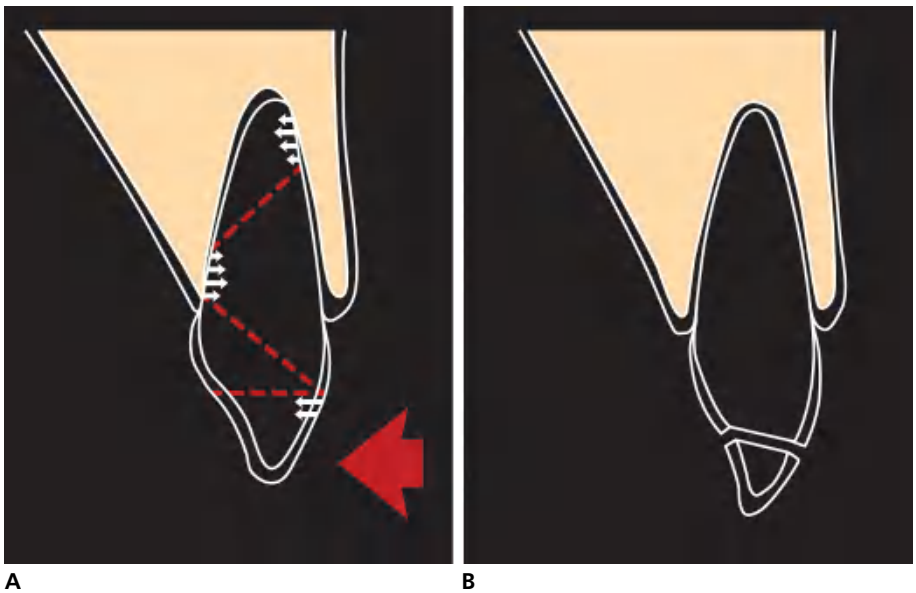



**Fig. 11.38** Pathogenesis of a crown fracture. A, B. The cleavage line takes the shortest route, resulting in a pure horizontal shear fracture of the crown.

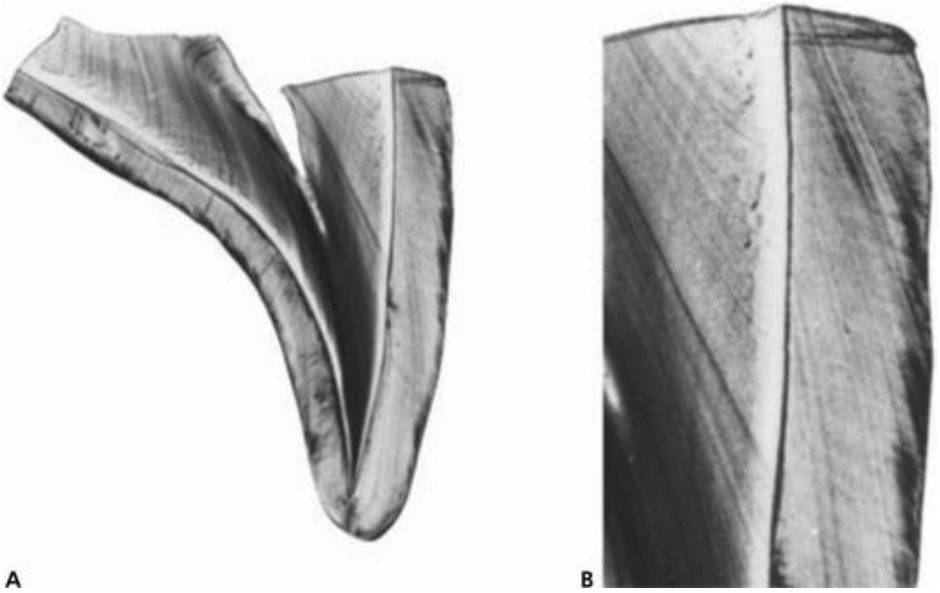



**Fig. 11.39** Fracture line orientation in enamel and dentin. A. The orientation of the enamel prisms and, to a certain degree, the dentinal tubules determine the course of the fracture line. B. Ground section of a fractured maxillary central incisor.

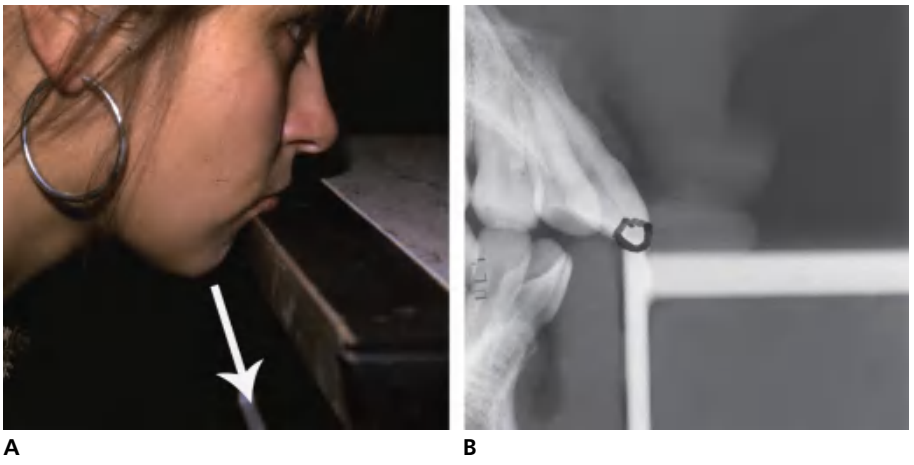



**Fig. 11.40** Etiology of a penetrating lip lesion. A. Impact direction parallel to the axis of the maxillary incisors. B. The lower lip has been interposed between the incisal edges and the staircase.

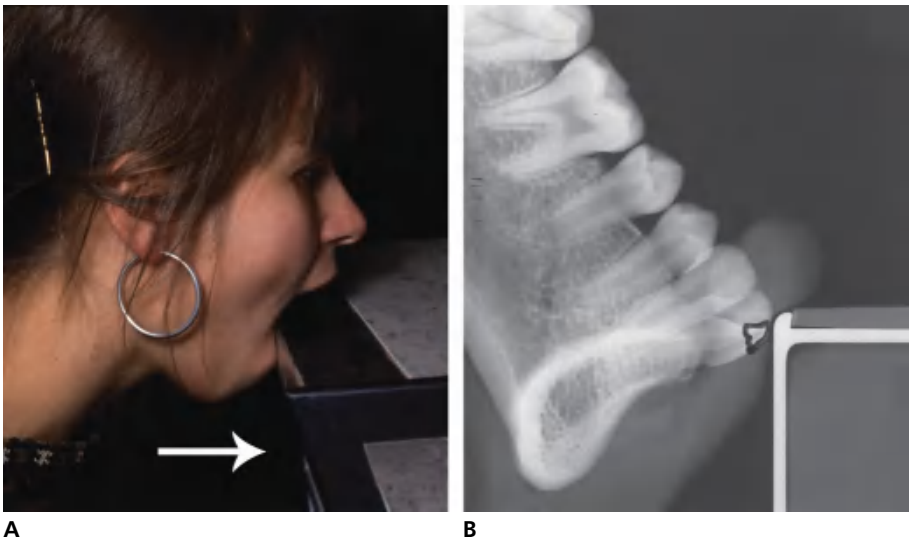



**Fig. 11.41** Etiology of a penetrating lip lesion. A. Impact direction is parallel to the axis of the mandibular incisors. B. The lower lip has been interposed between incisal edges and the staircase.

emergency service should be provided via a central dental emergency clinic or hospital.

Studies have shown that treatment of dental trauma in emergency care services is often inadequate (201, 418, 422–425), and that patients are not always satisfied with the care provided (418, 426). For this reason it is important that emergency dental staff have experience in dental trauma treatment and preferably be regularly exposed to such situations (421, 425).

Manuals and guidelines (19–21) or web pages for emergency treatment (e.g. www.dentaltraumaguide.org, accessed January 2018; for more information see Chapter 42) are valuable tools and will facilitate the treatment of dental trauma for dentists serving in the emergency service.

Prevention of TDIs is also largely neglected. This may be because injuries have traditionally been regarded as random events, unavoidable 'accidents'. This assumption is incorrect, as a body of knowledge now exists on the etiology of TDIs. For example, it is well established that human factors, such as increased incisal overjet, and environmental factors, such as playgrounds, coupled with a vector, such as tough sports, are significant factors predisposing to TDI. Therefore, TDI has causal factors and can often be prevented. Nevertheless, few health professionals provide appropriate information to the public on how to prevent and manage a TDI event. A public survey conducted in Australia reported that, while most parents would seek emergency dental care if necessary, only 10% were informed on the correct emergency procedures if a permanent tooth was avulsed (416). For more information on how to inform the public see Chapter 42.

## Future models

While learning how to care for a patient is a continuously ongoing process, a goal for the future would be to increase lay knowledge in managing a TDI, and to increase the accessibility and the quality of emergency care. A model to follow would be the *WHO Healthy Cities Programme*, where a healthy city is defined as one that continually creates and improves the physical and social environment and expands community resources for enabling mutual support among population groups for living (427). The WHO *Health Promoting Schools Programme* offers a broad solution for dental trauma as a public health problem, where a Health Promoting School constantly strengthens its capacity as a healthy setting for living, learning and working (428). A wide range of actions and policies are possible, including personal and social education aimed at developing life skills, school policies against bullying and violence, physical environment, school health policy, alcohol policy, provision of mouth guards and links with health services (429). Moysés et al. showed that 10% fewer children in Brazil had TDIs in Health Promoting Schools that demonstrated a commitment towards health and society (430).

## Essentials

### Injuries to the hard dental tissues and the pulp (Fig. 11.1)

- Enamel infraction
- Enamel fracture
- Enamel-dentin fracture
- Complicated crown fracture (i.e. with pulp exposure)
- Uncomplicated crown-root fracture
- Complicated crown-root fracture (i.e. with pulp exposure)
- Root fracture

### Injuries to the periodontal tissues (Fig. 11.2)

- Concussion
- Subluxation (loosening)
- Extrusive luxation (peripheral dislocation, partial avulsion)
- Lateral luxation
- Intrusive luxation (central dislocation)
- Avulsion (exarticulation)

### Injuries to the supporting bone (Fig. 11.3)

- Comminution of alveolar socket
- Fracture of alveolar socket wall
- Fracture of alveolar process
- Fracture of mandible or maxilla (jaw fracture)

### Injuries to the gingiva or oral mucosa (Fig. 11.4)

- Laceration of gingiva or oral mucosa
- Contusion of gingiva or oral mucosa
- Abrasion of gingiva or oral mucosa

### Standardized registration

- To be able to compare different studies and see trends in the future, the registration of TDIs must be improved in the future and be globally standardized

### Core outcome set

- The International Association of Dental Traumatology has established a core outcome set which fulfils the minimum requirement of registration in future studies. Anyone planning a study should consult these recommendations (www.iadt-dentaltrauma.org; accessed January 2018) to make sure these minimum criteria for registration are used

### Prevalence and incidence

#### *Global burden of TDI (Table 11.8)*

- Fourth (between second and fourth) most frequent oral disease
- 18th (between 13th and 19th) most frequent among all bodily diseases and injuries

#### *World TDI prevalence in primary dentition (at 5 years of age) (Table 11.7)*

- Mean age 3.4 years; prevalence 23% (17.3–29%)

#### *World TDI prevalence in permanent dentition (at 12 years of age) (Table 11.6)*

- Mean age 14.0 years; prevalence 15.5% (13.2–17.9%)
- Prevalence of dental injuries decreases significantly after 30 years of age (Fig. 11.11)

### *Incidence (Tables 11.9 and 11.10)*

- In population-based studies: 24.3 (18.6–30.6) per 1000 persons per year
- In cohort studies: 51.9 (27.6–83.3)

### *Trends*

- Dental trauma may become more prevalent than dental caries in some countries in the future

### *Repeated episodes*

- If a trauma episode occurs at an early age (9 years) there is an eight-fold increase in risk for new trauma
- Increased risk for trauma to the same teeth

## Sex and age

- Boys are affected almost twice as often as girls
- Peak incidence of dental injuries is at 2–4 and 8–10 years of age

## Predisposing factors

- Increased overjet
- Protrusion of upper incisors
- Insufficient lip closure

## Types of dental injuries

- Primary dentition: most often luxations
- Maxillary central incisors are most commonly involved
- Permanent dentition: most often uncomplicated crown fractures

## Place of injury

- At home
- In the street or in public places

## Seasonal variations

- Dependent on local customs

## Etiology

- Human behavior:
  - Risk-taking
  - Peer relationship problems
  - Hyperactivity
  - Stress behavior
- Environmental factors:
  - Deprivation
  - Overcrowding
- Unintentional injuries:
  - Falls and collisions
  - Physical leisure activities (sports)
  - Traffic accidents
  - Inappropriate use of teeth
  - Biting hard itims
  - Presence of illness, physical limitations or learning difficulties
- Intentional injuries:
  - Physical abuse
  - Iatrogenic procedures

## Mechanism of dental injuries

### *Trauma type*

- Direct trauma
- Indirect trauma

### *Factors characterizing an impact to the teeth*

- Energy of impact
- Resilience of the impacting object
- Shape of the impacting object
- Angle of direction of the impacting force

## Treatment need

- Treatment needs are insufficiently met in most countries

## Organization of emergency care

- Dental emergency service should be provided on a 24-hour basis by emergency dental staff regularly exposed to such situations, and backed up by specialists

## Future models

- Increase professional and lay knowledge in managing dental trauma by including the WHO Health Promoting Schools Programme and WHO Healthy Cities Programme

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# 12

# Examination and Diagnosis of Dental Injuries

F. M. Andreasen, J. O. Andreasen, M. Tsukiboshi & N. Cohenca

## Introduction

A dental injury should be considered an emergency and should ideally be treated immediately to relieve pain, facilitate reduction of displaced teeth and for some injuries also improve prognosis. However, minor injuries can very well be treated with some delay. This issue is further discussed in Chapter 42.

Rational therapy depends upon a correct diagnosis, which can be achieved with the help of various examination techniques. While a dental injury can often present a complex picture, most injuries can be broken down into several smaller components. Information gained from the various examination procedures will assist the clinician in defining these trauma components and determining treatment priorities (1–18, 56–64, 96–106). It must be understood that an incomplete examination can lead to inaccurate diagnosis and less successful treatment (101). Surveys of examination procedures in children have been published by Hall in 1986 (107), Newton in 1992 (142) and Bakland and Andreasen in 2004 (141).

An adequate history is essential to the examination and should include answers to the questions listed in the next section. In order to save time, standardized charts are recommended (see Appendices 1–4, pp. 1020–5) (143) (Fig. 12.1). The recorded information is of value in insurance claims and for other medico-legal considerations.

Despite the importance of a systemic approach, which begins with an adequate medical and dental history, acute bleeding or respiratory problems and replantation of avulsed teeth will change this sequence.

The *pattern of injury* observed depends primarily upon factors such as: (1) the energy of impact; (2) the direction and location of impact; and (3) the resilience of the periodontal structures.

Resilience of the periodontal structures appears to be the most significant factor in determining the *extent of injury*. Thus, impact in the very resilient skeleton supporting the primary dentition usually results in tooth displacement rather than fracture of hard tissues.

The opposite is true of injuries in the permanent dentition. Thus, with impact against the ground in an adult, either a *crown*, *crown-root* or *root fracture* may result, as the energy of impact will tend to produce a combination of zones of compression and tensile stress.

In the event that the lips intercept the initial blow, the energy delivered may be distributed over several teeth, resulting in *concussions*, *subluxations*, *lateral luxations* or *intrusions*.

If the course of impact is steeper and there is direct impact against the teeth, *crown fractures* can result. An indirect impact with the lips intervening may result in *extrusive luxation* with or without a *penetrating lip wound*.

In case of an axially directed impact against the chin, energy may be absorbed by the mandibular condyles or symphysis, and the premolars or molars of both dental arches. Such an impact can result in *fracture* or *luxation* of the mandible or *temporomandibular joint*, as well as *crown* or *crown-root fractures* of the involved teeth. In the event of this type of impact, it should also be borne in mind that the axis of rotation of the head can be at the base of the skull. In these cases, *cerebral involvement* should also be considered (see also under 'History').

The extent of the above-mentioned injuries will naturally be altered by the relative binding strength of the teeth to their supporting structures.

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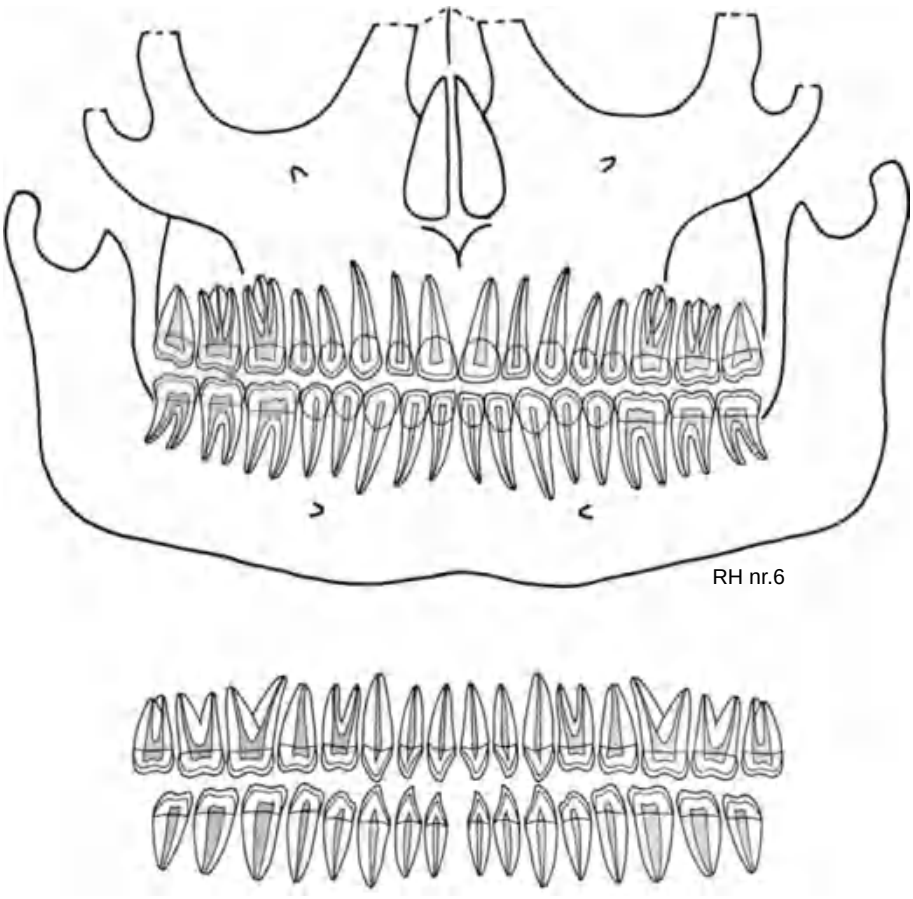



**Fig. 12.1** Diagram used for recording dental and maxillo-facial injuries.

In conclusion, when examining a dental trauma, consider the following features with respect to determining the pattern of injury and the subsequent extent of injury:

(1) The direction of impact (its relationship to the occlusal plane)
(2) Possible lip/soft tissue involvement
(3) Resilience of the periodontal structures; in this regard, the patient's history is valuable

## History

(1) Patient's name, age, sex, address, and telephone number
(2) When did injury occur?
(3) Where did injury occur?
(4) How did injury occur?
(5) Treatment elsewhere
(6) History of previous dental injuries
(7) General health

The implications of the answers to these questions are discussed separately.

**Item 1.** Apart from the obvious necessity of such information, the ability of the patient to provide the desired information might also provide clues to possible cerebral involvement or general mental status (e.g. inebriation) (see also Item 8).

**Item 2.** The time interval between the injury and treatment significantly influences the result of replantation of avulsed teeth (137, 138). Furthermore, the result of treatment of luxated teeth (19), crown fractures with and without pulp exposures (20–22), as well as bone fractures, may be influenced by delay in treatment (23, 24).

**Item 3.** The place of the accident may indicate a need for of tetanus prophylaxis.

**Item 4.** As already indicated, the nature of the accident can yield valuable information on the type of injury to be expected, e.g. a blow to the chin will often cause fracture at the mandibular symphysis or condylar region as well as crown-root fractures in the premolar and molar regions (Fig. 12.2). Accidents in which a child has fallen with an

object in its mouth (e.g. a pacifier or toy), tend to cause dislocation of teeth in a labial direction.

In young children and adults presenting multiple soft tissue injuries at different stages of healing, and where there is a marked discrepancy between the clinical findings and the past history, battered child or domestic violence syndromes should be considered (see Chapter 10). In such cases the patient should be referred for a medical examination.

**Item 5.** Previous treatment, such as immobilization, reduction or replantation of teeth, should be considered before further treatment is instituted. It is also important to ascertain how the avulsed tooth was stored, e.g. tap water, sterilizing solutions or dry.

**Item 6.** A number of patients may have experienced repeated injuries to their teeth. This can influence pulpal sensibility tests and the recuperative capacity of the pulp and/or periodontium (Figs 12.3 and 12.4).

**Item 7.** A short medical history is essential for providing information about a number of disorders such as allergic reactions, epilepsy or bleeding disorders such as hemophilia (65–67). These conditions can influence emergency as well as later treatment (Fig. 12.5).

**Fig. 12.2** A soft tissue bruise under the chin can imply a complicated injury pattern, often involving fractures of premolars and molars, the temporomandibular joint and symphysis, as well as a risk of cerebral involvement. In this case, a blow to the chin has resulted in multiple crown-root fractures of the premolars (arrow).

**Fig. 12.3** Previous injury, primary dentition. This 4-year-old girl has just suffered subluxation of her right central incisor. At the age of 1 year there was an injury affecting both central incisors resulting in pulp necrosis of the right central incisor and pulp canal obliteration of the left incisor.

**Fig. 12.4** Previous injury, permanent dentition. The patient has just suffered enamel fractures of the incisors. However, a dental injury 4 years earlier resulted in pulp necrosis of both central incisors and pulp canal obliteration, arrested root formation and secondary pulp necrosis of the left lateral incisor.

**Fig. 12.5** An 8-year-old boy with known hemophilia experiencing prolonged bleeding from the periodontal ligament around the left central incisor. The patient had suffered a subluxation injury 22 hours earlier. From (66).

The subjective complaints can provide the examiner with a clue to the injury. The following questions should also be addressed.

(8) Did the trauma cause amnesia, unconsciousness, drowsiness, vomiting or headache?
(9) Is there spontaneous pain from the teeth?
(10) Do the teeth react to thermal changes, sweet or sour foods?
(11) Are the teeth tender to touch, or during eating?
(12) Is there any disturbance in the bite?

**Item 8.** Episodes of amnesia, unconsciousness, drowsiness, vomiting or headache indicate cerebral involvement. Amnesia can be disclosed by the patient's response to questions (e.g. Item 1), repetition of questions (e.g. 'Where am I?', 'What's happened?') and inability to recall events immediately before or after the accident. In such cases, the patient should immediately be referred for medical examination to establish priorities for further treatment (108). It should be noted, however, that most acute dental treatment (e.g. radiographic examination, repositioning of displaced teeth, application of fixation) can be performed at the patient's bedside. As previously mentioned, this early treatment will help to improve the long-term prognosis of dental injury.

Another type of trauma may occur in patients under general anesthesia who are slowly regaining consciousness. At a certain stage of recovery, strong masticatory muscle activity can take place with resultant clenching and biting which can lead to injury of the tongue, lips and teeth (107, 109).

**Item 9.** Spontaneous pain can indicate damage to the tooth-supporting structures, e.g. hyperemia or extravasation of blood into the periodontal ligament. Damage to the pulp due to crown or crown-root fractures can also give rise to spontaneous pain.

**Item 10.** Reaction to thermal or other stimuli can indicate exposed dentin or pulp. This symptom is to some degree proportional to the area of exposure.

**Items 11 and 12.** If the tooth is painful during mastication or if the occlusion is disturbed, injuries such as extrusive or lateral luxation, alveolar or jaw fractures, or crown-root fractures should be suspected.

## Clinical examination

An adequate clinical examination implies a thorough examination of the entire injured area and the use of special examination techniques. The use of standardized examination charts can aid data registration (see Appendices 1–4, pp. 1020–5). In general, the direction of examination should progress from the facial surface into the oral cavity in order to pinpoint treatment needs. In contrast, treatment should begin in the oral cavity and progress outwards, to ensure optimal access to areas needing treatment. These diagnostic procedures can be summarized as follows.

(13) Recording of extraoral wounds and palpation of the facial skeleton
(14) Recording of injuries to the oral mucosa or gingiva
(15) Examination of the crowns of teeth for the presence and extent of fractures, pulp exposures or changes in color
(16) Recording of displacement of teeth (i.e. intrusion, extrusion, lateral displacement or avulsion)
(17) Disturbances in occlusion
(18) Abnormal mobility of teeth or alveolar fragments
(19) Palpation of the alveolar process
(20) Tenderness of teeth to percussion and change in percussion (ankylosis) tone
(21) Reaction of teeth to pulpal sensibility testing

**Item 13.** Extraoral wounds are usually present in cases resulting from traffic accidents. The location of these wounds can indicate where and when dental injuries are to be suspected, e.g. a wound located under the chin suggests dental injuries in the premolar and molar regions and/or concomitant fracture of the mandibular condyle and/or symphysis. Palpation of the facial skeleton can disclose jaw fractures. Subcutaneous hematomas can also be an indication of fracture of the facial skeleton.

**Item 14.** Injuries of the oral mucosa or gingiva should be noted. Wounds penetrating the entire thickness of the lip can frequently be observed, often demarcated by two parallel wounds on the inner and/or outer labial surfaces (Fig. 12.6). If present, the possibility of tooth fragments buried between the lacerations should be considered (25, 26, 68–71). Such

embedded fragments can cause acute or chronic infection and disfiguring fibrosis. The probable mechanism for these injuries is that the tooth, having penetrated the full thickness of the lip, is fractured as it emerges from the skin and strikes a hard object. The tooth fragment is retained within the soft tissue, which then envelops it at the moment of impact (25). These fragments can seldom be palpated, irrespective of size. Careful radiographic examination of the involved soft tissues is therefore necessary to disclose these fragments (Fig. 12.6). Along with tooth fragments, other foreign bodies can often be found within the soft tissue.

Gingival lacerations are often associated with displaced teeth. Bleeding from non-lacerated marginal gingiva indicates damage to the periodontal ligament.

Submucosal hematomas sublingually, in the vestibular region or in the palate can be indicative of a jaw fracture. Thorough radiographic examination, including examination of the border of the mandible and mobility of jaw segments en bloc, must accompany this finding, as a jaw fracture could otherwise be overlooked (Fig. 12.7).

Finally, it is essential that blood covering the alveolar process be removed as this can sometimes reveal displacement of the mucoperiosteum into the buccal sulcus. Typically these patients demonstrate severe edema of the upper lip and sharp pain upon palpation of the exposed periosteal surface (107).

**Item 15.** Before examining traumatized teeth, the crowns should be cleaned of blood and debris.

Infraction lines in the enamel can be visualized by directing a light beam parallel to the long axis of the tooth or by shadowing the light beam with a finger or mouth mirror (see Chapter 13). When examining crown fractures, it is important to note whether the fracture is confined to the enamel or includes dentin. The fracture surface should be carefully examined for pulp exposures; if present, the size and location should be recorded. In some cases, the dentin layer may be so thin that the outline of the pulp can be seen as a pinkish tinge beneath the dentin. One should take care not to perforate the thin dentinal layer during the examination.

Crown-root fractures in the molar and premolar regions should be expected in the case of indirect trauma (see Chapter 14). It is important to remember that crown-root fractures in one quadrant are very often accompanied by similar fractures on the same side of the opposing jaw. It is therefore necessary to examine occlusal fissures of all

**Fig. 12.6** Penetrating lip lesion. Two parallel lesions, either in the mucosa and skin or mucosa only, are an indication that teeth have penetrated the tissue and that tooth fragments and other foreign bodies can be expected deep within the wound. A radiographic film is placed between the lips and the dental arch. The exposure time is 25% of the normal for a dental exposure.

A B C

**Fig. 12.7** A 10-month-old child referred for a luxated left primary central incisor. A. The clinical examination revealed slight loosening of the incisor and a sublingual hematoma (arrow). B. Radiographic examination of the involved incisors revealed no sign of jaw fracture. C. Because of the finding of a sublingual hematoma, a new radiograph was taken that included the border of the mandible. This radiograph clearly demonstrated fracture of the symphysis.

molars and premolars to confirm the presence or absence of possible fractures.

Depending on the stage of eruption, fractures below the gingival margin can involve the crown alone or the cervical third of the root.

Color of the traumatized tooth should be noted, as changes can occur in the post-injury period. These color changes are most often prominent on the oral aspect of the crown at the cingulum. Moreover, examination with transillumination can reveal changes in translucency (see Chapter 16). With respect to *crown color*, color change at the time of injury or at follow-up visits can often only be detected from the oral aspect of the tooth, using a mirror and transillumination, where changes can be limited to the cingulum. Andreasen (129) demonstrated that reversible color change can occur at the time of injury or later in the healing period of luxated teeth and is thus not pathognomonic for pulp necrosis. It was not until 2012 that Malmgren and Hübel (199) reported transient coronal discoloration in root-fractured teeth where the discoloration disappeared from 4 weeks to 6 months after injury in eight of the nine teeth reported. The sensibility lost at the time of injury followed the changes in discoloration; and all teeth regained normal sensibility when the discoloration had disappeared. Only one tooth that showed a grayish hue developed pulpal necrosis (199).

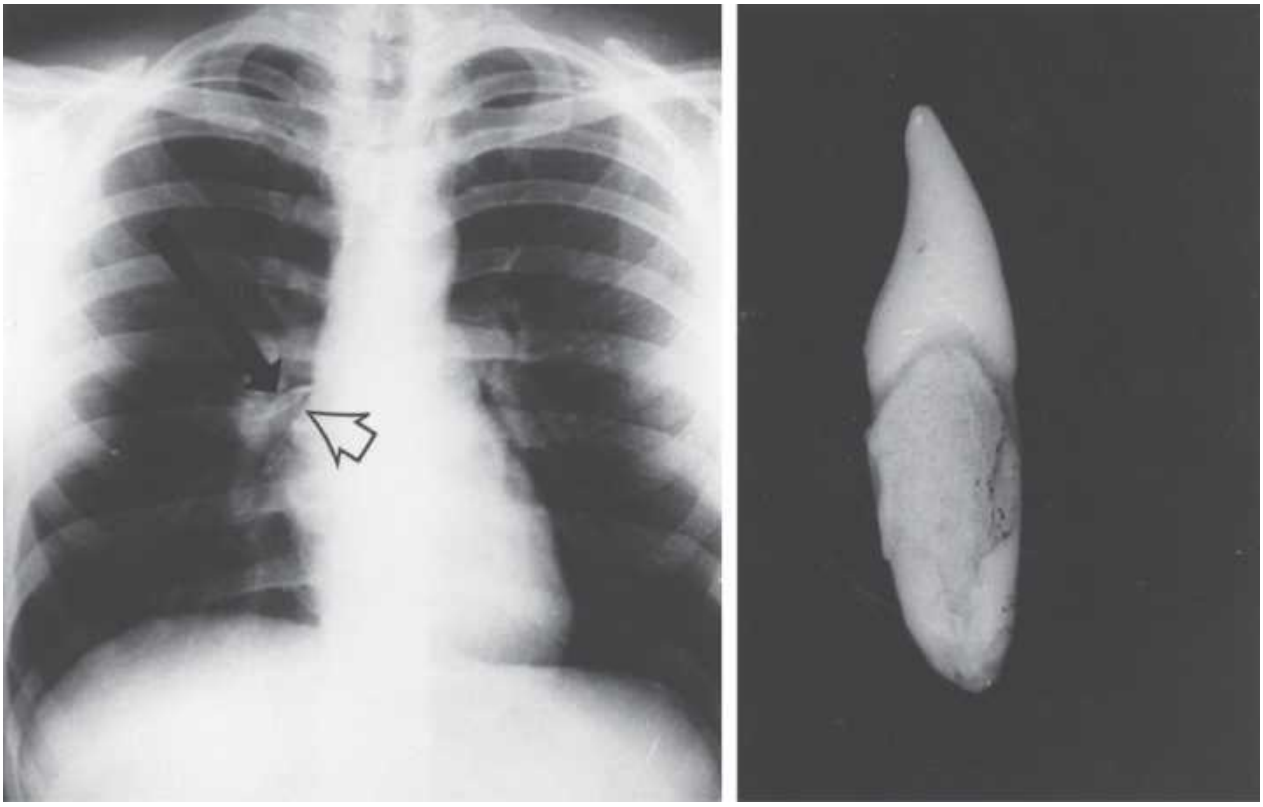
**Item 16.** Displacement of teeth is usually evident by visual examination; however, minor abnormalities can often be difficult to detect. In such cases, it is helpful to examine the occlusion as well as radiographs taken at various angulations.

The possibility of inhaling or swallowing teeth at the time of injury should always be considered when teeth or prosthetic appliances are missing and their presence elsewhere cannot be established (72–75).

Although inhalation of foreign bodies in connection with traumatic injuries is normally associated with a loss of protective reflexes in an unconscious patient, it may also occur in a conscious patient without producing symptoms (75). Consequently, if there is reason to suspect inhalation or swallowing of a tooth or dental appliance, it is important that radiographs of the chest and the abdomen be taken as soon as possible (Fig. 12.8).

In case of tooth luxation, the direction of the dislocation as well as extent (in millimeters) should be recorded. In the primary dentition, it is of utmost importance to diagnose oral dislocation of the apex of a displaced primary tooth, as it can impinge upon the permanent successor.

It is important to remember that, apart from displacement and interference with occlusion, laterally luxated and intruded teeth present very few clinical symptoms. Moreover, these teeth are normally firmly locked in their displaced position and do not usually demonstrate tenderness to percussion. While radiographs can be of assistance, diagnosis is confirmed by the percussion tone (101, 204).



A B

**Fig. 12.8** A. Posterior/anterior radiograph of the chest showing a foreign body in the right middle lobe intermediate bronchus after maxillofacial injury (arrow). B. Tooth recovered from the right lung via bronchoscopy. From (75).

**Item 17.** Abnormalities in occlusion can indicate fractures of the jaw or alveolar process. In the case of jaw fractures, abnormal mobility of the jaw fragments can be demonstrated.

**Item 18.** All teeth should be tested for abnormal mobility, both horizontally and axially. With respect to *mobility testing*, this is recorded on a scale of 0 to 3, where 0 = no abnormal looseness, 1 = faciolingual and/or mesiodistal movement of not more than 1 mm, 2 = faciolingual and/or mesiodistal movement of more than 1 mm, and 3 = faciolingual and/mesiodistal movement as well as axial movement. A shortcoming with this measurement is the nil value. Not only might it imply no abnormal looseness, but is could also mean 'locked into bone' either at the time of injury (e.g. intrusive or lateral luxation) or at follow-up visits (i.e. replacement resorption/ankylosis). However, when this is coupled with information from the *percussion sound*, its meaning becomes clear (101, 204). Disruption of the vascular supply to the pulp should be expected in case of axial mobility.

It should be remembered that erupting teeth and primary teeth undergoing physiologic root resorption always exhibit some mobility.

The typical sign of alveolar fracture is movement of adjacent teeth when the mobility of a single tooth is tested.

In case of root fracture, location of the fracture determines the degree of tooth mobility. However, without radiographic examination, it is usually not possible to distinguish between luxation injuries and root fractures.

**Item 19.** Uneven contours of the alveolar process usually indicate a bony fracture. Moreover, the direction of the dislocation can sometimes be determined by palpation.

**Item 20.** Reaction to percussion is indicative of damage to the periodontal ligament (76). The test may be performed by tapping the tooth lightly with the handle of a mouth mirror, in a vertical as well as horizontal direction. Injuries to the periodontal ligament will result in pain. As with all examination techniques used at the time of injury, the percussion test should be begun on a non-injured tooth to assure a reliable patient response. In smaller children, the use of a fingertip can be a gentler diagnostic tool. In infants, this is neither possible nor reliable.

The sound elicited by percussion in a horizontal direction is also of diagnostic value. Thus, a hard, metallic ring elicited by percussion in a horizontal direction indicates that the tooth is locked into bone; while a dull sound indicates subluxation or extrusive luxation. However, it should be noted that teeth with apical and marginal periodontal lesions can also give a dull percussion sound (76). Dental trauma has also been found to be associated with increased touch thresholds in permanent teeth with recovery towards healthy control values after 3–12 months (144, 204).

A calibrated percussion instrument has been introduced, Periotest®, which is an electronic device that measures the dampening characteristics of the periodontium (110, 111, 145, 146, 171). However, the force imparted by such an instrument might contribute a new trauma, possibly leading to damage to the neurovascular supply of the pulp. In the follow-up period, however, this instrument could be of value in the diagnosis of post-traumatic sequelae, such as ankylosis (147).

A dental trauma implies a number of examination procedures, whose purpose is to determine the extent of injury and the status of healing processes or healing complications in the follow-up period. Each of these procedures has a certain *sensitivity* (i.e. the capacity to diagnose the extent of injury (e.g. type of luxations) or healing complications (e.g. pulp necrosis)). Thus, a sensitivity value of 1.0 implies 100% effectiveness that all cases of a certain of luxation were diagnosed correctly and that all cases of pulp necrosis were identified. *Specificity* is the capacity to correctly diagnose no traumatic changes or a healthy healing status. A specificity value of 1.0 implies 100% effectiveness. An ideal testing method should, therefore, have 100% sensitivity (i.e. no false positives) and 100% specificity (i.e. no false negatives), a utopian situation which presently does not exist (148). This concept will be applied in the description of the various examination procedures.

In selecting examination procedures, it is important to consider the consequences of the value of specificity. If a method is chosen which favors high sensitivity, this will imply that a certain number of teeth with vital or healing pulps will be subjected to endodontic treatment if precision is not reasonably high.

If all attention is paid to the precision (i.e. avoiding endodontic treatment of vital or healing teeth), a certain number of teeth with pulp necrosis will not be detected or treated. In case of pulp-related root resorption, this is an unfortunate situation; so in this situation, a high specificity of a test procedure should be used.

**Item 21.** Pulp testing following traumatic injuries is a controversial issue. These procedures require cooperation and a relaxed patient in order to avoid false reactions. However, this is often not possible during initial treatment of injured patients, especially children.

Pulpal sensibility testing at the time of injury is important for establishing a point of reference for evaluating pulpal status at later follow-up examinations. A number of tests have been proposed. However, the value of these has been questioned (27–29, 112, 113, 204). The principle of these tests involves transmitting stimuli to the sensory receptors of the dental pulp and registering the reaction; while others register the vascular component in the pulp canal.

## Pulp testing principles

In the evaluation of various pulp testing procedures, it should be considered that most procedures (e.g. thermal tests, electrometric pulp testing (EPT)) assess the nerve supply to the pulp (which is naturally dependent upon an intact vascular supply); whereas laser Doppler flowmetry (LDF) and pulse oximetry (PO) assess the presence of a functioning vascular supply.

A disturbing fact then comes to mind, that neural regeneration in a traumatized pulp is slower than vascular regeneration and sometimes is even lacking (153–156). Thus, a

priori vascular detecting systems (e.g. LDF and PO) are more sensitive than EPT and thermal testing devices, which are specifically related to nerve regeneration.

In a recent, very detailed study on the sensitivity and specificity of various pulp testing methods, LDF was compared to other pulp testing methods in vital teeth and teeth with known pulp necrosis. LDF was found to be far better than EPT and ethyl chloride (i.e. higher sensitivity and specificity) (148, 161) (Fig. 12.9). However, several problems must be solved before LDF can become a routine instrument in the dental trauma armamentarium (see later).

The assessment of pulp vitality following dental trauma is an important diagnostic challenge, as often temporary loss of sensibility may occur which is also coupled with the limitations of conventional pulpal sensibility testing. After acute trauma, the dental pulp blood supply might be torn (e.g. concussion, subluxation) or severed/ruptured (extrusive or lateral luxation). This could lead to pulpal edema, which could result in a negative pulpal 'response' just after injury. In this situation, a period of approximately 10–14 days could elapse before a positive pulpal response returns. In the case of tooth dislocation and subsequent rupture of the neurovascular supply at the apical foramen, it might take up to 3 months in immature teeth or several years in teeth with closed apices for pulpal sensibility to return (199, 200). Thus, negative sensibility at the time of injury is not a sign of pulp necrosis, but a sign of pulpal damage, which has prognostic significance in the follow-up period with respect to the development of pulp canal obliteration after luxations (201, 221), interposition of hard or connective tissue after root fracture (202, 218) and pulp necrosis in combined crown fracture/luxation injuries (203–206). Moreover, Öhman (30) could demonstrate histologically that although some teeth did not respond to EPT, revascularized pulpal tissue had indeed proliferated into the root canal, short of the pulp horn. This finding was corroborated by Andreasen (222) in pulps extirpated after acute injury.

Pulp testing is often erroneously referred to as 'vitality testing'. However, it merely reflects the capacity of pulpal nerves to respond to stimuli, whether electrical or thermal. True vitality testing requires equipment capable of monitoring pulpal blood flow, that is, LDF (207, 208) or pulp oximetry. Mesaros and Trope reported a case following severe luxation of both maxillary central incisors in an 8-year-old where at 76 days after repositioning only one incisor was weakly responsive to $CO_2$ ice. Yet LDF indicated revascularization was occurring in both teeth from a much earlier time (161).

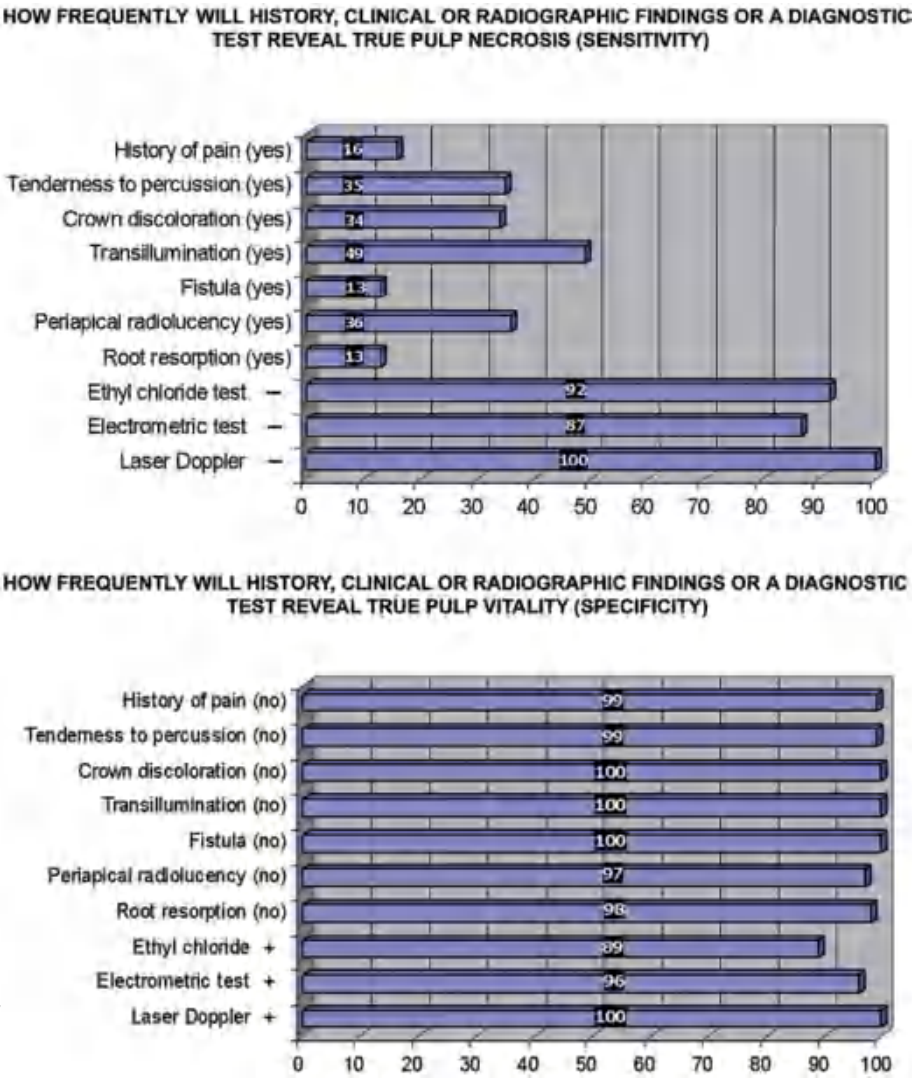



**Fig. 12.9** Sensitivity and specificity of various pulp testing procedures. Reproduced from (148) with permission.

Thus, unwarranted endodontic intervention was avoided. Evans et al. (148) found LDF to be a reliable method of assessing pulpal status of traumatized teeth, although it was a technique sensitive and time-consuming to use. Moreover, in the case of coronal discoloration, Heithersay and Hirsch found that bleeding into the injured pulp resulted in negative laser Doppler readings (139). To date, however, no equipment has yet proven itself amenable to routine clinical use.

Concerning *electrometric pulp testing*, it was found that the lowest threshold value was achieved if the electrode was placed on the incisal edge (77, 78). The numeric value of the EPT response should be recorded for each tooth. Teeth with open apices will generally react to a higher threshold value than teeth with closed apices. A 'positive' value for a tooth with a closed apex might disguise a false positive response, where the actual value is at a level corresponding to an immature tooth and would imply damage to pulpal nerves.

*Thermal (i.e. cold) tests* are generally conducted with either frozen sticks of $CO_2$ snow (–78 °C) or a cotton pellet sprayed with difluordichlormethane (–50 °C) (211). While interpretation of pulp sensibility testing can be variable in young patients and in traumatized teeth, $CO_2$ and to a lesser extent difluorchlormethane, are more reliable than electric pulp testing for immature teeth (31, 77, 211).

Bastos et al. (201) in a study of 121 permanent incisor teeth found that the temporary loss of sensibility was a frequent finding during post-trauma healing, especially after luxation injuries. The accuracy of each sensibility test at the initial and final appointments was, respectively, 55.1% and 67.8% for the heat test, 55.9% and 77.9% for the cold test, and 57.6% and 89% for the electrical test. That is, these authors found electric pulp testing most reliable in the long-term follow-up for pulpal diagnosis. However, cold testing is recommended over electric pulp testing in young individuals (i.e. open apical foramen) (211).

## Mechanical stimulation

In crown fractures with exposed dentin, pulpal sensibility can be tested by scraping with a dental probe. Some authors have proposed drilling a test cavity in the tooth in order to register the pain reaction when the bur advances into the dentin. However, in a study on sensibility reactions of replanted teeth, it was found that a pain reaction was not elicited until the dentin–pulp border was reached (30). Moreover, it has been found that there are intermediary stations in pulpal response (i.e. transient apical breakdown) (129, 130, 223) which mimic the cardinal signs of pulp necrosis, and which could be reversible and lead to pulpal healing (see Chapter 16) (197, 198). Test cavities would expose otherwise intact dentin and involve the risk of bacterial contamination of the pulp. Such tests are, therefore, contraindicated.

In the case of crown fractures with exposed pulp tissue, the reaction of the pulp to mechanical stimuli can be tested by applying a cotton pellet soaked in saline. Exploration with a dental probe must not be attempted as it may provoke severe pain and inflict additional injury to the pulp.

## Thermal tests

Thermal stimulation of teeth has been used for many years and various methods have been advocated. Among these, the most frequently used are heated gutta-percha, ethyl chloride, ice, carbon dioxide snow and dichlor-difluormethane.

Thermal pulp testing results are not reproducible in terms of graded intensity, and normal pulp tissue may yield a negative response (28, 77, 78). A positive reaction usually indicates a vital pulp, but it may also occur in a non-vital pulp, especially in cases of gangrene when heat produces thermal expansion of fluids in the pulp space, which in turn presumably exerts pressure on inflamed periodontal tissues (37).

### Heated gutta-percha

The following standardization has been advocated by Mumford (28). A stick of gutta-percha is heated by holding about 5 mm of its length in a flame for 2 seconds, whereupon it is applied to the tooth on the middle third of the facial surface (Fig. 12.10). The value of this test has been questioned, as the intensity of sensation reported by the patient is not reproducible, and even non-injured teeth may fail to respond (28, 78). The sensitivity of this test has been found to be 0.86 and specificity to be 0.41 (149).

### Ice

This method involves the application of a cone of ice to the facial surface of the tooth (Fig. 12.11). Reaction depends on the duration of application; a period of 5–8 seconds increases the sensitivity of this test (32). The reliability of this procedure has also been questioned as non-injured teeth may not respond (31, 32, 77).

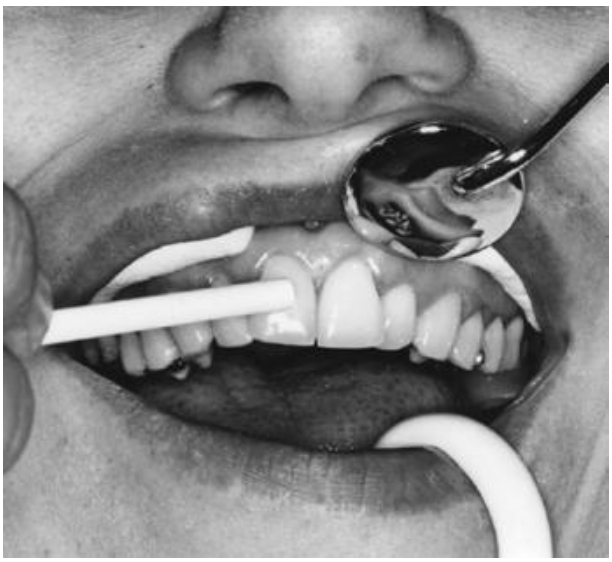


**Fig. 12.10** Pulp testing with heated gutta-percha. The stick of gutta-percha is applied to the middle third of the facial surface.

## Ethyl chloride

Ethyl chloride can be applied by soaking a cotton pledget and then placing it on the facial surface of the tooth to be tested (Fig. 12.12) (Caution! this is flammable). The limitations described for heated gutta-percha also apply to this method, although ethyl chloride gives more consistent results (28, 79, 80). The sensitivity for this test has been found to be 0.83 and specificity 0.93 (149).

## Carbon dioxide snow

Because of its low temperature (–78 °C, –108 °F), carbon dioxide snow gives very consistent and reliable results, even in immature teeth (33–36, 77, 78, 81, 82) (Fig. 12.13). This method also allows pulp testing in cases where an injured tooth is completely covered with a temporary crown or splint (78) (Fig. 12.11). However, a serious drawback to this procedure is that the very low temperature of the carbon dioxide snow can result in new infraction lines in the enamel (83, 84) (Fig. 12.14), although this finding has not been substantiated in later studies (113, 114). Furthermore, no changes could be found in the pulp in animal experiments (115). Only prolonged exposure to very low temperatures (i.e. –80 °C for 1–3 minutes) has been shown to elicit transient pulpal changes (secondary dentin formation) (116).

## Dichlor-difluormethane

This is another cold test (e.g. Frigen®, Provotest®, EndoIce®) in which an aerosol is released at a temperature of –28 °C (–18 °F) onto the enamel surface (84–86). Like carbon dioxide snow, it elicits a very reliable and consistent response from both mature and immature teeth (87). However, the same drawback has been recorded with this test as with carbon dioxide snow, although to a lesser degree; namely, infraction lines in the enamel caused by the thermal shock (84).

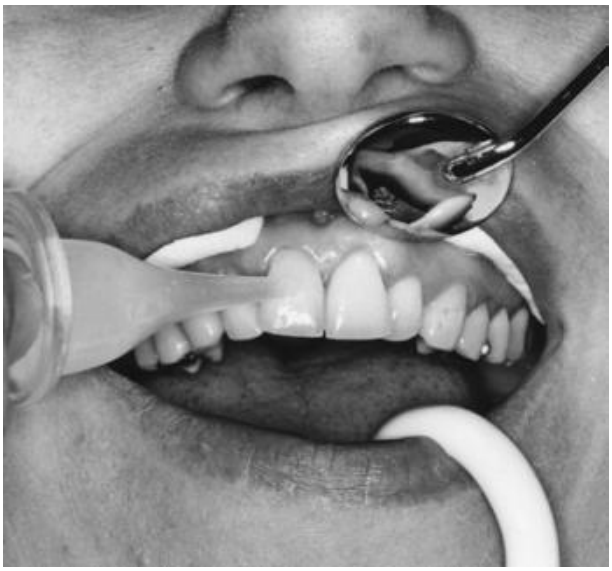


**Fig. 12.11** Pulp testing with ice. A cone of ice is applied to the middle third of the facial surface of the tooth: 5–8 seconds may be required to elicit a reaction.

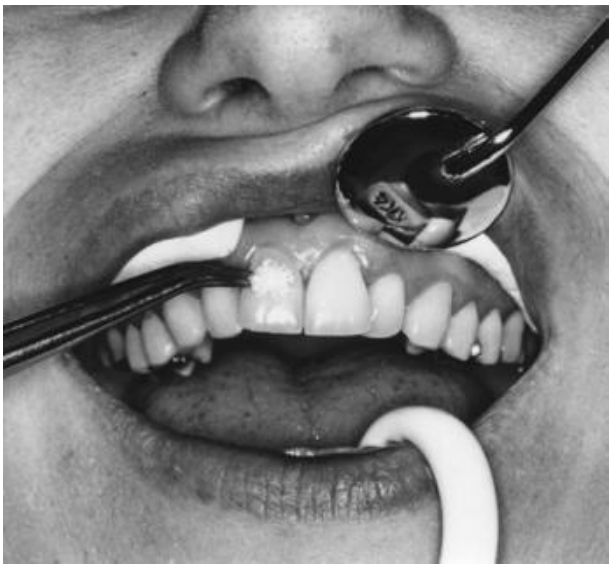
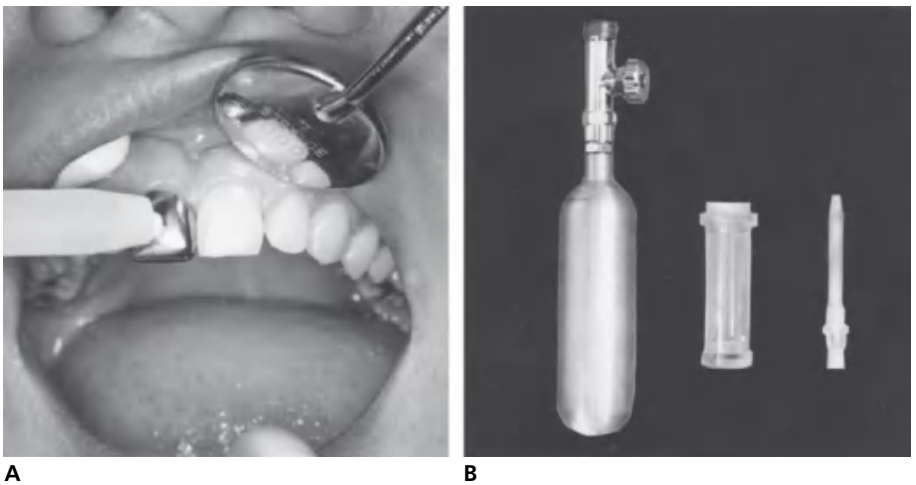


**Fig. 12.12** Pulp testing with ethyl chloride. A pledget of cotton is saturated with ethyl chloride and applied to the middle third of the facial surface.



A B

**Fig. 12.13** Pulp testing with carbon dioxide snow. A. Pulp testing with carbon dioxide snow of a fractured incisor covered with a stainless steel crown. B. Disassembled model of a carbon dioxide snow pulp tester (Odontotest®, Fricar A.G., Zürich 1, Switzerland).

## Electrometric pulp testing

EPT should employ a current measurement instrument that allows control of the mode, duration, frequency and direction of the stimulus (37, 117). Voltage measurement is not satisfactory because a given voltage produces varying currents as a result of differences in the electrical resistance of the tissues, especially enamel. Such variations can result from fissures, caries and restorations (38). Experimental studies have shown that the current is probably carried ionically through the electrolytes of the tooth (39, 40). Moreover, immediate changes in the vascular flow after a luxation injury are reflected in loss of EPT response, which with time can return to normal (129, 157).

The stimulus should be clearly defined, as it significantly affects nerve excitation (41). Furthermore, the electrode area should be as large as the tooth shape will permit, allowing maximum stimulation (38, 42, 118). Stimulus duration of 10 milliseconds or more has been advocated (42). Recently, a digital pulp tester has been introduced which appears to produce very reliable readings (119).

Electrical sensibility testing is usually carried out in the following way (Fig. 12.15).

(1) The patient is informed as to the purpose and nature of the test and is instructed to indicate when a sensation is first experienced.

(2) The tooth surface is isolated with cotton rolls and air-dried. Saliva on the tooth surface can divert the current to the gingiva and periodontal tissue, giving false readings. However, the tooth should not be desiccated for long periods, as enamel can lose moisture with a resultant increase in electrical resistance (43, 44). Several media such as saline and toothpaste can be used as a conductor between the electrode and tooth surface (140).

(3) The electrode is placed as far from the gingiva as possible, preferably on a fracture area or the incisal edge, where the strongest response can be obtained (120, 121). The neutral electrode can be held by the patient. A modification of this system involves the examiner completing the circuit by touching the patient's mouth with a finger or a mouth mirror. A metal dental instrument (e.g. dental explorer) can also serve as an electric conductor to the tooth (122). The use of rubber gloves presents a clinical problem, as the correct use of the pulp tester requires that the dentist completes the electrical circuit by direct physical contact with the patient. One study has indicated that this problem can be overcome if the patient grasps the end of the pulp tester after it is positioned on the tooth (123); however, other studies suggest that such a procedure can produce unreliable results (124, 125). Recently, a clip to the lip has been developed which permits the dentist to conduct a test wearing rubber gloves (126).

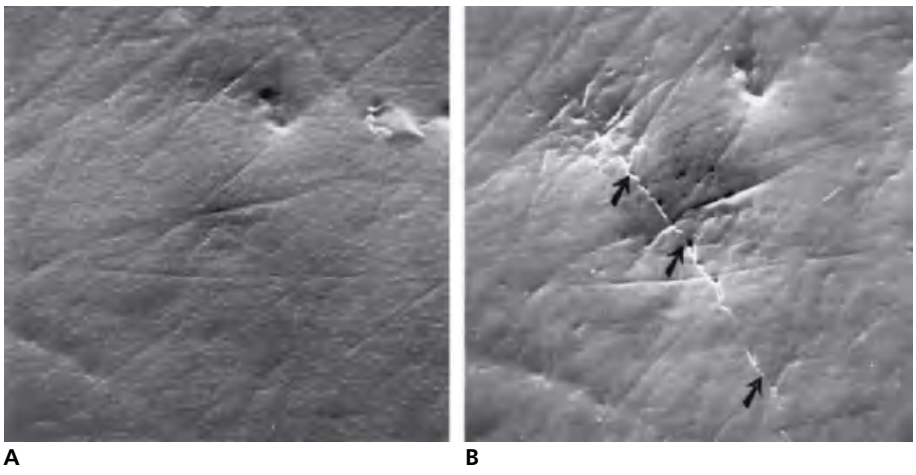



**Fig. 12.14** Scanning electron microscope view of the enamel surface before (A) and after (B) pulp testing with carbon dioxide snow (×1200). It is apparent that an infraction line has developed after testing (arrows). From (84).

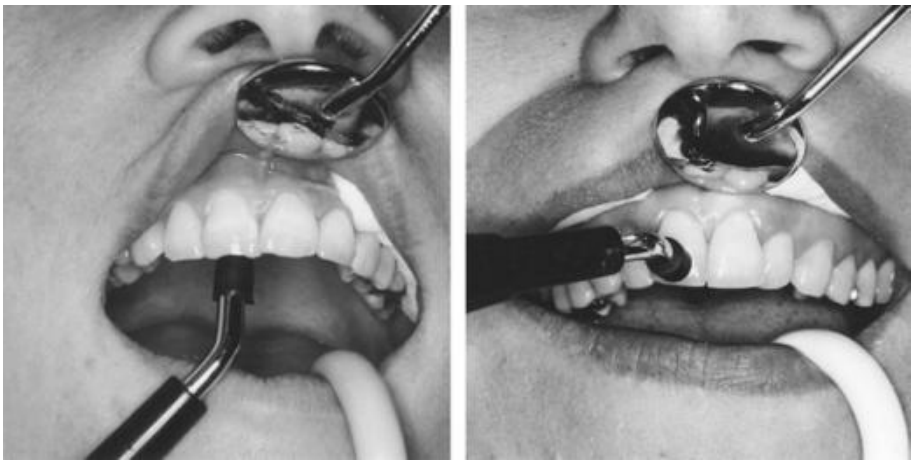


**Fig. 12.15** Electrometric pulp testing. The tooth is air-dried and isolated with cotton rolls. The electrode is placed on the incisal edge or on the incisal third of the facial surface of the crown.

(4) The rheostat of the tester is advanced continuously until the patient reacts. If the current is then maintained at the existing level, adaptation occurs and the patient feels that the pain has disappeared, so that a further increase of current gives a higher threshold value. This phenomenon implies that the pain threshold cannot be regarded as constant (42). The threshold value should therefore be determined by a quick rather than a slow increase in current. However, the current should not be increased so quickly that it is painful. The value of the pain threshold of the tooth should be recorded for later comparison.
(5) Splints and temporary crowns used in the treatment of traumatic dental injuries can alter the response to both thermal and electrometric tests (78). Thus, contact between the gingiva and a stainless steel crown, a metal cap splint or arch bar significantly increases the pain threshold, as the current bypasses the tooth and is conducted to the gingiva or adjacent teeth (78). In order to obtain a reliable sensibility response to EPT, the electrode must be placed upon enamel and the tooth isolated from adjacent vital teeth (Fig. 12.15). A distance of at least 1 mm between the electrode and metal is recommended. If the temporary crown or splint cannot be altered in the above-mentioned manner, thermal pulp testing with carbon dioxide snow is a reliable alternative (78) (Fig. 12.16).

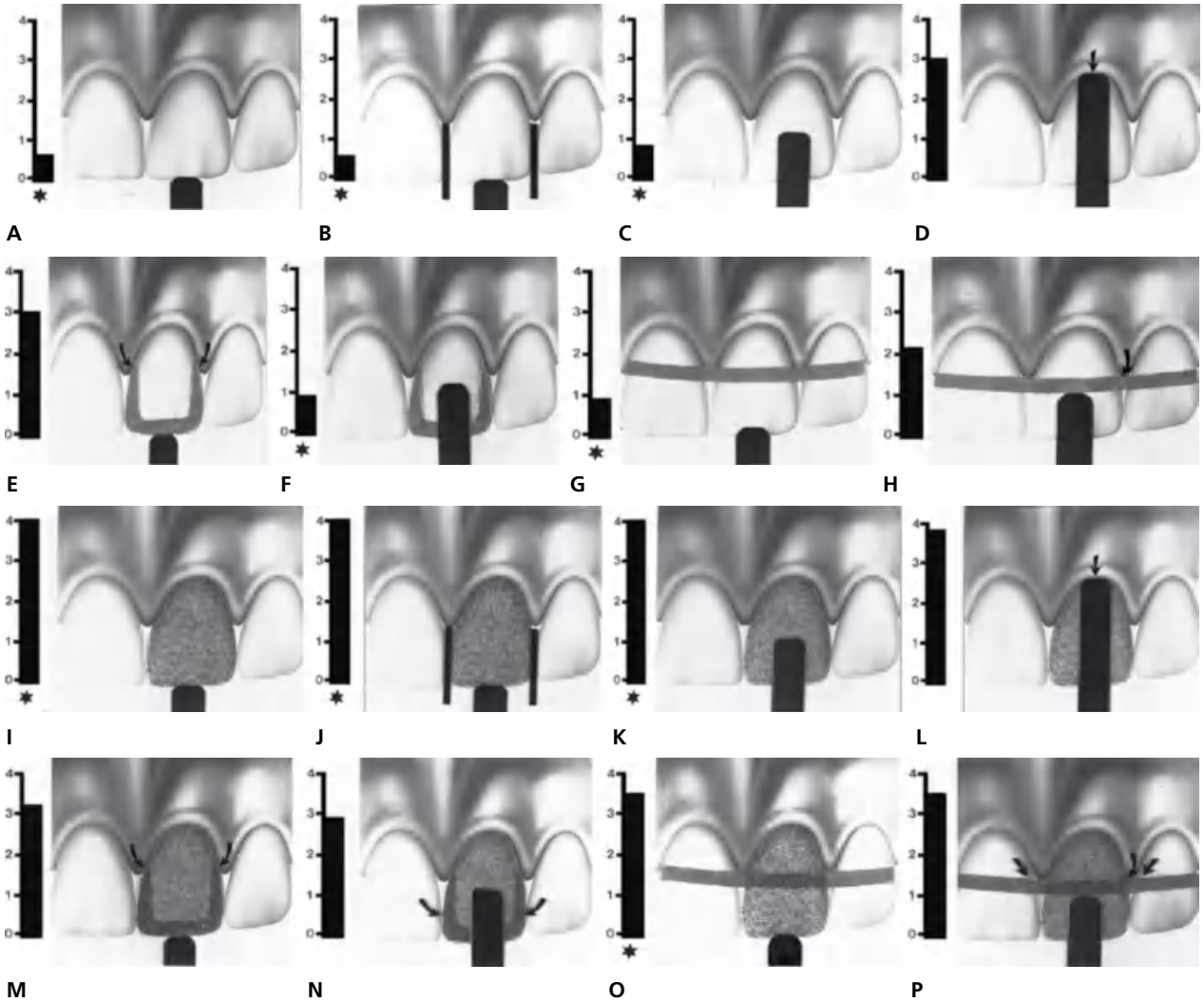



**Fig. 12.16** Influence of splints and temporary crowns upon electric pulp testing procedures. The schematic drawings illustrate the reactions recorded with a Siemens pulp tester (Sirotest II®) which has a scale from 0 to 4. The columns next to the drawing of the teeth indicate the average reaction to the specific testing circumstances. A to H illustrate testing of a vital tooth, and I to P illustrate testing of a tooth with pulp necrosis. A. Electrode placed upon the incisal edge. B. Tooth isolated with an acetate strip. C. Electrode placed upon the middle third of the labial surface. D. Electrode contacting gingiva. E. Electrode contacting an open-faced steel crown. F. Electrode placed upon enamel on a tooth with an open-faced steel crown. G. Electrode placed upon the incisal edge of a tooth splinted with a metal arch bar. H. Electrode placed upon the metal arch bar. I. Electrode placed upon the incisal edge of a tooth with pulp necrosis. J. Tooth isolated with acetate strips. K. Electrode placed upon the middle third of the labial surface. L. Electrode contacting gingiva. M. Electrode contacting an open-faced steel crown, the current is passed on to the gingiva, giving a weak response. N. Electrode placed upon enamel on a tooth with an open-faced steel crown. The current is passed on to the adjacent teeth. O. Electrode placed upon the incisal edge of a tooth splinted with a metal arch bar. The current is passed on to neighboring vital teeth. P. Electrode placed upon the metal arch bar. The current is passed on to neighboring vital teeth. *Note*: all testing situations revealing a reliable response are indicated with an asterisk. Reproduced from (78) with permission.

The value and reliability of EPT have been evaluated by comparison of the pain threshold with the histologic condition of the pulp (29, 45, 88–91, 222). Apparently, there is not always a direct relationship; thus teeth which fail to respond to stimulation with maximum current can show a histologically normal pulp, while inflamed or even necrotic pulps can respond electrometrically within the normal range. A common belief has been that low readings indicate hyperemia or acute pulpitis while high readings indicate chronic pulpitis or degenerative changes. However, recent investigations do not support this view (29, 46).

The interpretation of pulpal sensibility tests performed immediately after traumatic injuries is complicated by the fact that sensitivity responses can be temporarily or permanently decreased, especially after luxation injuries (127–130, 223). However, repeated testing has shown that normal reactions can return after a few weeks or months (1, 31, 47–50, 80, 92, 128–130, 223). Moreover, teeth which have been loosened can elicit pain responses merely from pressure of the pulp testing instrument. It is therefore important to reposition and immobilize, for example, root-fractured or extruded incisors prior to pulp testing. If local anesthetics are to be administered for various treatment procedures, pulp testing should be performed prior to doing this.

Another factor to consider is the stage of eruption. Teeth react differently at various stages, sometimes showing no reaction at all when root formation is not complete (51–53, 77, 93, 94). However, the excitation threshold is gradually lowered to the normal range as maturation proceeds, although it increases again in adulthood when the pulp canal becomes partly obliterated (131, 132) (Fig. 12.17). One explanation could be incomplete communication between the odontoblastic processes and nerve fibers in immature teeth (54). However, this has been questioned in a recent study where numerous myelinated nerve fibers were found in the coronal odontoblast layer irrespective of stage of development (133). Moreover, it is often difficult to isolate partially erupted teeth and the current may circumvent the tooth, passing directly to the gingiva (55).

Finally, teeth undergoing orthodontic movement display higher excitation thresholds (95).

The sensitivity of EPT has been found in the range of 0.72 and specificity 0.93 (148–152).

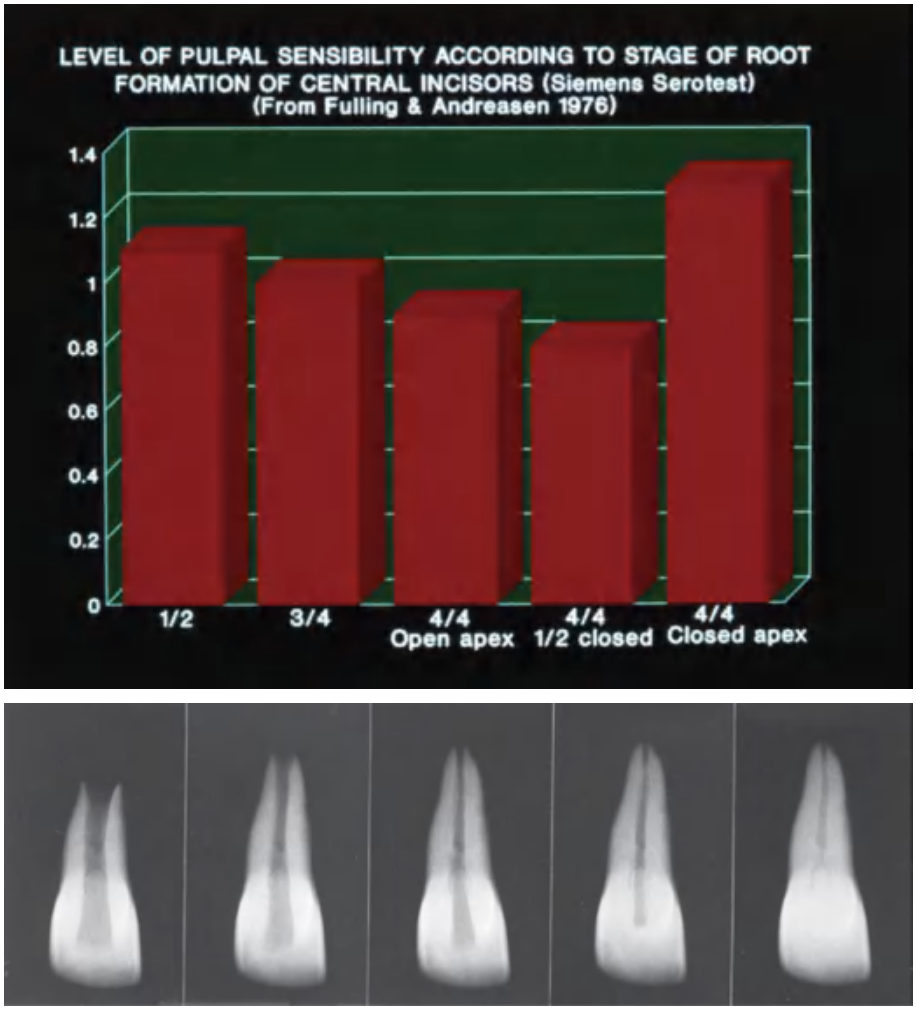



**Fig. 12.17** Variation in sensibility level according to stage of root development of permanent incisors. The diagram illustrates the reactions recorded with a Siemens pulp tester (Sirotest 77® which has a scale from 0 to 4). It appears that early and late stages of root development are related to higher sensibility levels. Reproduced from (77) with permission.

## Laser Doppler flowmetry

A method has been developed whereby a laser beam can be directed at the coronal aspect of the pulp. The reflected light scattered by moving blood cells undergoes a Doppler frequency shift. The fraction of light scattered back from the pulp is detected and processed to yield a signal (Fig. 12.18). The value of this method has been demonstrated in several clinical studies where it was possible to diagnose the state of pulp revascularization approximately 3–4 months before EPT (113, 134, 135, 158–165) (Figs 12.19 and 12.20). The design of the probes (163, 164), wave length, band width and laser power have been found to influence the sensitivity and specificity of LDF (166–168). Furthermore, it has been found that the optimal position of the probe should be 2–3 mm from the facial gingiva (167). Experimental studies have also demonstrated the superiority of this procedure (160).

In a detailed clinical study it was found that in traumatized incisors that showed no electrometric sensibility reactions and where LDF indicated no pulp vitality, the reliability of LDF was 97%. Conversely, in electrometric non-sensible teeth, where LDF showed the presence of blood perfusion, the accuracy of LDF in regard to pulp vitality was 100% (158, 159).

### Improved diagnosis of pulp necrosis by the use of LDF compared to other methods

In several studies, it has been found that LDF was able to advance detection of the revascularization process of a damaged pulp due to a luxation injury compared to other examination procedures (e.g. $CO_2$ and EPT) – from 6 months to 3 months after injury (134, 135, 158–160, 162). In a recent and very comprehensive study, the sensitivity and specificity of LDF was tested in 80 patients with luxated maxillary incisors (169, 170). In this study, the control teeth demonstrated

**Fig. 12.18** Pulp testing using a laser Doppler flowmeter. The probe is placed on the labial surface of the tooth.

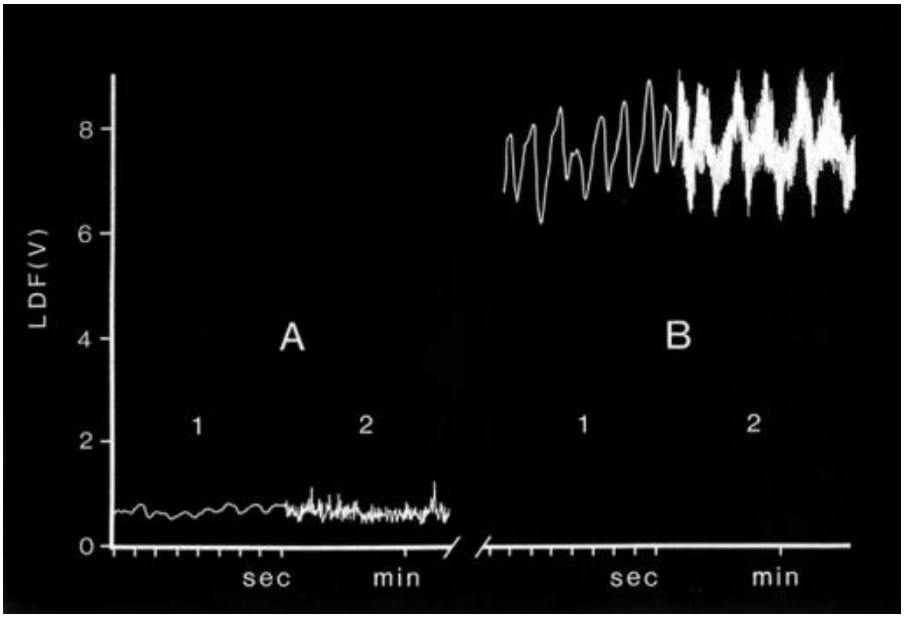



**Fig. 12.19** Laser Doppler flowmetry (LDF) recording from a non-vital tooth (A) and adjacent normal (control) tooth (B). Chart speed 600 mm/min. Reproduced from (134) with permission.

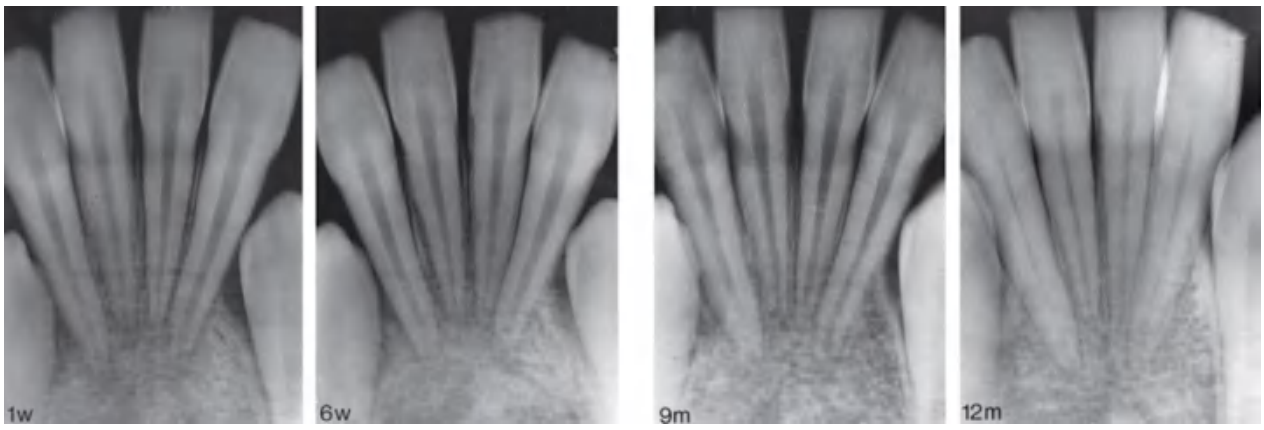



**Fig. 12.20** Follow-up of four luxated incisors at 1 and 6 weeks, 9 and 12 months. LDF represents laser Doppler flow values and electrometric settings (Analytic Technology® pulp tester) at which sensory responses were obtained in each tooth. – indicates an absence of sensory response at the highest stimulus level. Reproduced from (134) with permission.

pulpal blood flow values of 9.9 at the time of injury. The final status 6 months after trauma showed that the cases which at that time could be classified as having obvious signs of pulp necrosis (type 4, loss of sensitivity and periapical radiolucency; and type 5, loss of sensitivity, periapical radiolucency and grey discoloration) had the maximum predictive values (high sensitivity (70%) and specificity (93%)) if a PU value of 2.9 was chosen (169, 170).

However, several problems must be solved before this method can be of general use. First, it has been found that blood pigment within a discolored tooth crown can interfere with laser light transmission (139). Second, the equipment necessary for this procedure needs further refinement before it can be of general clinical value. Thus, the time required to test a single tooth must be reduced significantly (presently it is 10–15 minutes). Third, the price of the equipment must be considerable lower than it is at present.

## Pulse oximetry

Pulse oximetry measures actual blood flow rather than the neural response (sensibility tests), and has been shown to be a reliable and accurate way of assessing the vitality of the pulp (178–180). Pulse oximetry is a non-invasive means of assessing the vitality of a dental pulp by measuring the percentage of hemoglobin in the arterial blood that is saturated with oxygen. Oxygenated blood is bright red while deoxygenated blood is of a darker, red-blue shade. The pulse oximeter, which uses a light-emitting diode (LED) to transmit light through the vascular bed, measures the color difference to determine the oxygen saturation of arterial blood (Fig. 12.21). To ensure that it is measuring arterial blood and not venous oxygen saturation, the pulse oximeter is designed to detect only pulsating blood vessels.

In 2007, Gopikrishna et al. (178, 179) compared the efficacy of a custom-made pulse oximeter dental probe with electric pulp testing and thermal testing for measuring the pulp vitality status of 17 recently traumatized permanent teeth. The results demonstrated that pulp oximetry was a reliable and accurate test. Specifically, positive responsiveness to thermal/electric pulp tests increased from no response on day 0 to 29.4% of teeth on the 28th day, 82.35% of teeth at 2 months, and 94.11% of teeth at 6 months. More recently, Bargrizan et al. conducted a clinical study on 329 maxillary central and lateral incisors in children (180). Mean oxygen values recorded in the patient's finger were 97%, and mean oxygen values in the maxillary central and lateral incisors were 87% and 84%, respectively. The authors reported no significant correlation between blood oxygen levels in the finger and in the teeth. There was a significant negative correlation between the stage of root development and the blood oxygen levels in the patients' teeth. Mean oxygen values in teeth with an open apex were significantly higher than in the teeth with a closed apex.

Recently, new and smaller sensors have been developed matching the dental dimensions (Fig. 12.21). Thus, the need to customize digital sensors is no longer needed.

## Radiographic examination

### Intraoral techniques

All injured teeth should be examined radiographically. This examination serves two purposes: (1) it reveals the stage of root formation; and (2) it discloses injuries affecting the root portion of the tooth and the periodontal structures. Most root fractures are disclosed by radiographic examination, as the fracture line usually runs parallel to the central beam (101, 136) (see Chapters 11 and 15).

The clinical diagnosis of tooth displacement is corroborated radiographically. There is a widening of the periodontal space in lateral and extrusive luxations, whereas intruded

**Fig. 12.21** Pulse oximeter. A. Photoelectric diode and receptor. Receptor applied to a central incisor. B. Diagram of the light-emitting diode (LED) and its relation to the pulp and receiver.

teeth often demonstrate a blurred periodontal space. However, determination of dislocation on the basis of radiographs is highly dependent on the angle of the central beam (19, 101, 203). Radiographic demonstration of dislocation of permanent teeth normally requires the use of more than one exposure at differing angulations. Swedish studies demonstrated that by varying the angulation of the central X-ray beam of the same periapical area, the likelihood of disclosing pathosis could be increased and that changes apparent in one angulation might not be apparent in others (205–207). Danish studies from the 1980s demonstrated similar findings with respect to diagnosing tooth displacement, root fractures and fractures of the supporting bone (208, 209). The ideal method is the use of three different angulations for each traumatized tooth, using a standardized projection technique (101, 203). Thus, a traumatized anterior region is covered by one occlusal film and three periapical exposures, where the central beam is directed between the lateral and central incisors and the two central incisors. This procedure ensures diagnosis of even minor dislocations in cases of luxations as well as root fractures (Fig. 12.22) and these are the current recommendations of the International Association of Dental Traumatology (IADT) (216).

Figure 12.22 illustrates how multiple radiographs at differing angulation can disclose lateral luxation. Many authors advocate the use of multiple films at different angulations to best identify intra-alveolar root fractures (101, 203, 223), and indeed, this is the current IADT recommendation. Molina et al. (219) advised multiple films for teeth with crown fractures as these were twice as likely to have root fractures as those without crown fractures.

The use of film holders is very important. It has been found that tooth dimension measurements from one radiographic examination to another can vary by a factor of 3 if film holders are not used (101).

In recent years, digital intraoral radiographic systems have become increasingly common and are replacing conventional dental X-ray films for the diagnosis of dental diseases. Specifically in cases of traumatized teeth, no significant differences between the two techniques were detected in the diagnosis of root fractures or external root resorption (203, 211–213). However, an inherent problem with both techniques is that the radiographic image is a two-dimensional shadow of a three-dimensional image. As a consequence, a number of studies have demonstrated a reduced sensitivity for diagnosing traumatized teeth with two-dimensional imaging (214–217).

Children under 2 years of age are often difficult to examine radiographically because of fear or lack of cooperation. With the parents' help and the use of film holders, it is usually possible to obtain a radiograph of the traumatized area (Fig. 12.23). It should also be noted that exposure time can be reduced by 30% for each 10 kilovoltage peak (KVP) increase (Fig. 12.24). In this way, radiographs of diagnostic quality can be taken even with uncooperative patients.

**Fig. 12.22** A, B. Clinical examination reveals displacement of a left central incisor. C–F. The radiographic examination of the anterior region consists of one occlusal film (C) and three periapical exposures, where the central beam is directed between the lateral (D) and central (F) incisors and between the central incisors (E). Note that the displacement of the left central incisor is clearly shown on the occlusal exposure (C), whereas an apical exposure (E) shows hardly any displacement.

**Fig. 12.23** Radiographic examination of infants. A special film holder is used in combination with a standard dental film (3.8 × 5.1 cm) (Twix film holder®, AIB Svenska Dental Instrument, Stockholm, Sweden). The film holder is held between two fingers and the film placed between the dental arches. The mother holds the infant in position with her left arm while steadying the child's head with her right hand. The projection angle should be more vertical than normal in order not to have the index finger projected onto the film. Note that both mother and child are protected by lead aprons.

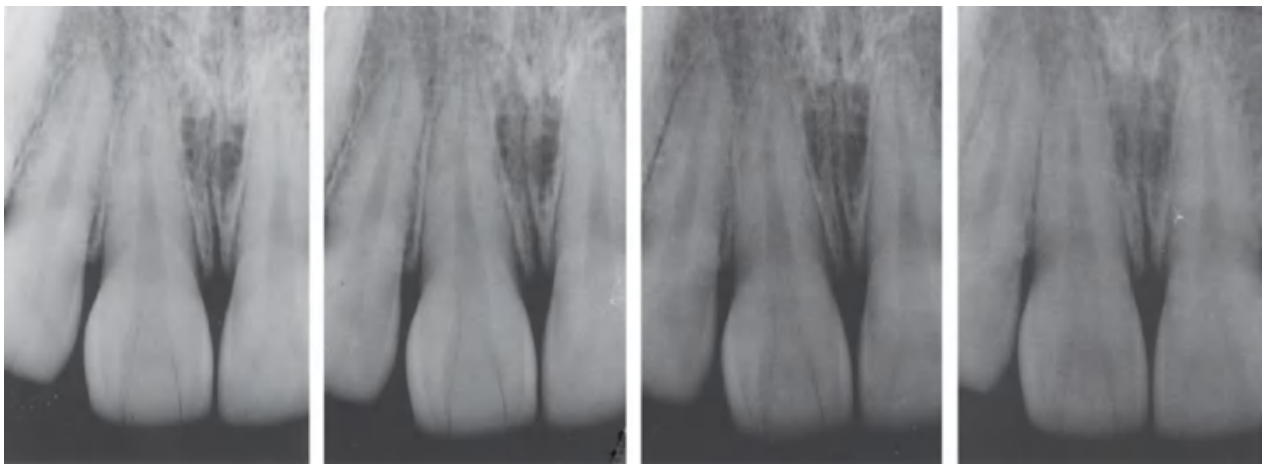



**Fig. 12.24** An increase in kilovoltage peak (KVP) slightly reduces the contrast. The right central incisor in a skull was radiographed using ranges from 55 to 85 KVP. The infraction lines in the crown which are clearly shown at 55 KVP are not so prominent at 85 KVP.

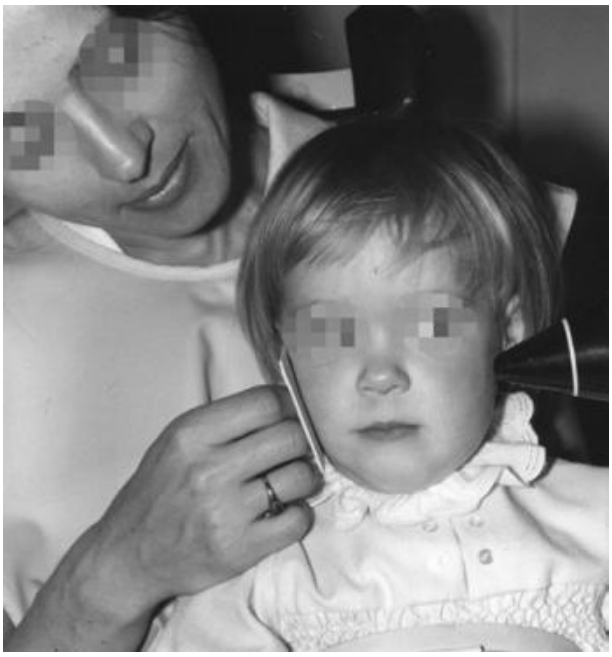


**Fig. 12.25** Extraoral lateral projection used to determine the position of displaced anterior teeth. The central beam is directed against the apical area and is parallel to the occlusal plane. A 6 × 7.5 cm film is held against the cheek perpendicular to the central beam.

Extraoral radiographs are of value in determining the direction of dislocation of intruded primary incisors (see Chapter 22) (Fig. 12.25). Bone fractures are usually discernible on intraoral radiographs unless the fracture is confined to the facial or oral bone plate.

Dislocated tooth fragments within a lip laceration can be demonstrated radiographically by the use of an ordinary film placed between the dental arches and the lips. A short exposure time (i.e. one-quarter to one-half normal exposure time) or the use of a low kilovoltage is advocated for these exposures.

## Extraoral techniques

### *Panoramic technique*

This procedure is always indicated in cases where a jaw fracture is suspected or a temporomandibular joint (TMJ) problem is found. Sensitivity and specificity values of this technique have not yet been studied in relation to dental trauma.

### *Cone beam computed tomography*

The first clinical and radiographic examination of the traumatized patient is crucial for determining the initial diagnosis, severity of injury and treatment plan and for establishing a baseline for follow-up. However, traumatized teeth present a clinical challenge with regard to their diagnosis, treatment plan and prognosis. Unfortunately, film-based intraoral radiographs provide poor sensitivity in the detection of minimal tooth displacements and root and alveolar fractures (181). This limitation is due to the projection geometry, superimposition of anatomic structures and processing errors. Cone beam computed tomography (CBCT) has significantly improved the ability to accurately diagnose traumatic injuries with the potential of overcoming most of the technical limitations of the plane film projection, along with the capability of providing a 3D representation of the maxillofacial tissues in a cost- and dose-efficient manner (182–185).

CBCT, also called digital volume tomography (DVT), produces 3D digital imaging at a reduced cost and with less radiation for the patient when compared with traditional spiral CT scans (183). It delivers a faster and easier image acquisition. Additionally, since no film and/or material are introduced inside the patient's mouth, the technique might be suitable even for patients presenting with different levels of fear and anxiety or behavioral problems.

Cone beam technology uses a cone-shaped beam of radiation to acquire a volume in a single 360° rotation similar to a panoramic radiograph. The various systems differ in four

main aspects: the scan time, the voxel (or resolution), the dosimeter (radiation) and the field of view (FOV).

The field of view could be defined as the area of interest to be covered during the scanning procedure. The dimensions of the FOV, or scan volume, are primarily dependent on the detector size and shape, beam projection geometry and the ability to collimate the beam. Collimation of the primary X-ray beam limits X-radiation exposure to the region of interest. Field size limitation therefore ensures that an optimal FOV can be selected for each patient based on disease presentation and the region designated to be scanned. In general, the smaller the scan volume, the higher the resolution of the image and the lower the effective radiation dose to the patient. CBCT systems can be categorized according to the available FOV or selected scan volume height as large, medium and small or limited FOV.

Scanning of the patient takes between 10 and 70 seconds. One major advantage of most CBCT units in comparison with CT is the ability to reduce the FOV to the region of interest.

Taking into consideration that most traumatic injuries occur in children, minimizing radiation is extremely important. More specifically, imaging of the maxillo-mandibular region with CBCT results in an absorbed dose of radiation of approximately 57 µSv (184), which is 96–98% less than that with traditional medical CTs (185). For purposes of comparison within the dental field, a panoramic radiograph results in an absorbed dose of radiation of 6 µSv and a full mouth series ranges between 33 and 100 µSv (186, 187) depending on variables such as film speed, technique, kilovoltage peak and collimation.

**Fig. 12.26** Micro CT (J. Morita Mfg. Corporation).

**Micro CT scanning**

Conventional dental and panorama images have limited diagnostic ability due to their two-dimensional character. Recently, a micro CT scanner has been developed and the clinical efficacy has been evaluated in a variety of clinical situations (171, 172).

A micro CT scanner (3DX: J. Morita Mfg. Corp.) is small in size, which means that it can also be used in smaller offices. The cone-shaped X-ray beam is received by a high sensitivity and high resolution light-intensifying tube (Fig. 12.26). As the beam passes through only a very limited region of the body, the result is a 3D image with extremely high resolution, but requiring only a very small radiation dose (173–177) (Fig. 12.27). The tube is usually set at 12–30 µSv, which is the equivalent of approximately four dental film exposures or a single panorama exposure, and represents only 1/50 of the dosage for a conventional CT. The resolution is much higher at 2 line pairs/mm, minimum, which is eight times greater than a conventional CT (Figs 12.27 and 12.28).

The image-processing software is able to reconstruct a 3D image in only 1 or 2 minutes. The *x*, *y* and *z* axes of the 3D image can be observed at 0.25–1 mm slice intervals (Fig. 12.28). While the 3DX has a wide range of applications, the examples presented here have been limited to trauma cases. Figure 12.29 shows the root fractures of two central incisors. A fracture line was observed only on the left incisor at the first visit using conventional radiographic techniques. Four months later, a second fracture line became obvious in the right central incisor. The micro CT scan revealed very clear images of the fracture lines of both teeth. Oblique fracture lines are difficult to detect with conventional methods, but micro CT appears useful in this regard.

While there is no debate about the benefits of CBCT in dental traumatology, all clinicians must always consider the principle of ALARA (as low as reasonably achievable) when using CBCT and any other imaging modalities. In order to minimize radiation exposure while maximizing diagnostic information, clinicians should consider the use of CBCT only when the need for imaging cannot be adequately met by lower dose conventional dental radiographs or alternate imaging modalities. Therefore, it is important to consider the effective dose of various imaging modalities and their resolution in relation to the information they provide to the clinician. In a recent conference organized by the Swiss Society of Dentomaxillofacial Radiology on the use of CBCT in pediatric dentistry, endodontics and periodontology it was concluded that CBCT should be used restrictively since radiation protection reasons do not allow its routine use. CBCT should therefore be reserved for complex cases where its application can be expected to provide further information that is relevant to the *choice of therapy* (202).

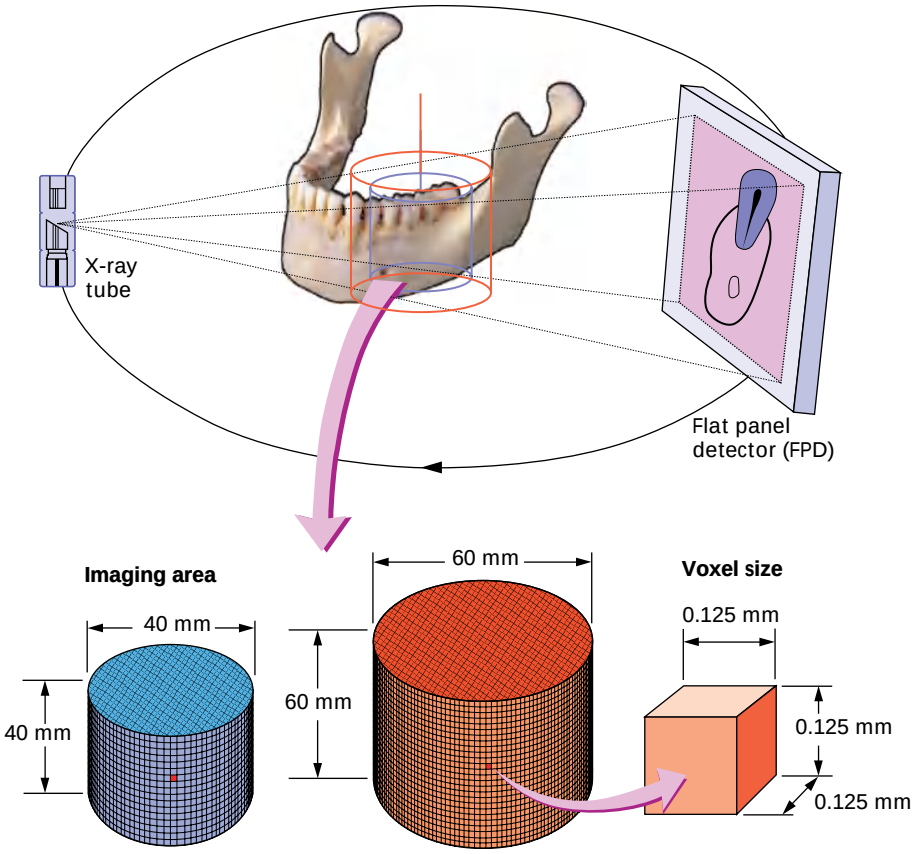



**Fig. 12.27** Exposure principle for the 3DX Multi Image Micro CT (J. Morita Mfg. Corporation). The smaller boxel size makes it possible to create a three-dimensional image that is eight time clearer than one obtained by conventional medical CT.

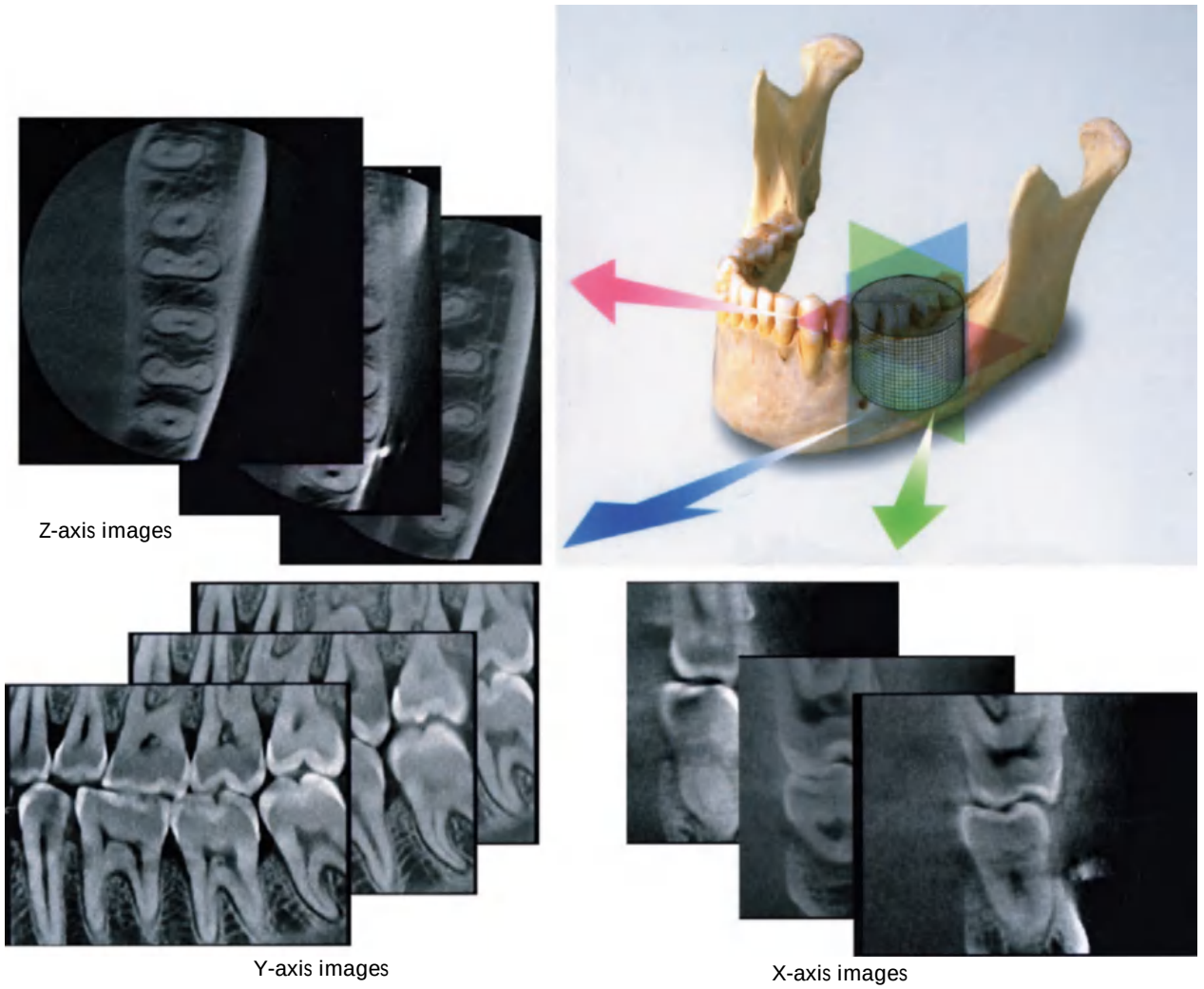



**Fig. 12.28** Cross-section images of the cylindrical imaging area. Images of a tooth model demonstrate the 3DX's 3D feature. The imaging area is a cylinder 40 mm in diameter and 30 mm high. Within this area, slices at 1 mm intervals can be viewed for all three dimensions: the *x* axis (right–left or buccal lingual), the *y* axis (back–front or mesial–distal) and the *z* axis (vertical).

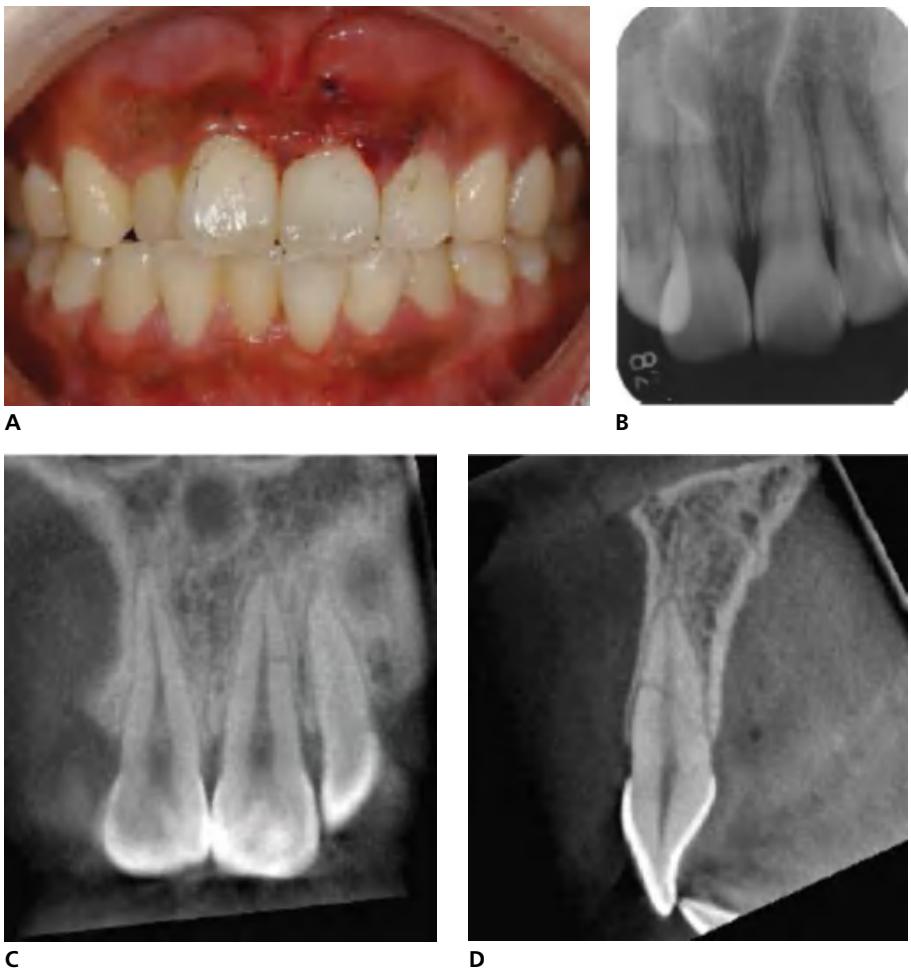


**Fig. 12.29** Cone beam computed tomography of a root fracture. A. A 26-year-old woman was referred 2 hours after the trauma. The electrometric pulp test of all her anterior teeth showed a positive response. B. A conventional radiograph at the first examination. There is no sign of a root fracture. C. A coronal CBCT image at the first examination. A mid-root fracture line is diagnosed on the left central incisor. D. A sagittal CBCT image of the left central incisor. The actual fracture line is oblique and located close to the gingival.

The potential use of CBCT in diagnosis and treatment planning in dental trauma was first discussed in 2007 by Cohenca et al. (196, 214). These reviews described the advantages and disadvantages of 3D imaging of dento-alveolar trauma and various healing complications.

### Crown-root, root and alveolar fractures

With the recent advent of CBCT, a new tool is available to accurately diagnose root and alveolar fractures regarding their presence or absence as well as the exact location, extent and direction of the fracture (184) (Figs 12.29 and 12.30). Limited FOV CBCT should be considered for diagnosis, after considering the strengths and limitations of intraoral radiography. The usefulness of CBCT may be questioned in cases in which a single periapical film has definitively shown a fracture in the apical or coronal third of a root. However, clinical outcomes depend on the exact location of the fracture, extent of displacement and potential connection of the fracture to the oral cavity (188). In 2004, Andreasen et al. reported the healing pattern of root fractures in correlation to the diagnosis and treatment provided (189, 190). Therefore, in a case with an uncertain assessment using conventional radiographs, CBCT might be considered (Fig. 12.29).

More recently, May et al. concluded that CBCT is most useful in cases in which conventional radiography yields inconclusive results or shows a fracture in the middle third of a root (191). In such cases CBCT may rule out false negatives, for example, a suspected root fracture not visualized with conventional radiography. For a root fracture in the middle third, CBCT may rule out or confirm an oblique course of fracture involving the cervical third in the labio-lingual dimension. When intraoral radiography shows a fracture in the middle third or the absence of fracture in a case when root fracture is suspected, CBCT is also indicated (Fig. 12.29). In the case of the former, CBCT is useful because it can reveal the course of the fracture in the sagittal plane (Fig. 12.29). Current therapeutic guidelines published by the IADT and the American Association of Endodontists (AAE) recommend considering the use of CBCT in cases with uncertain diagnosis (192, 210). In the case of an alveolar fracture, CBCT may give valuable information of the labio-lingual course of the fracture (184) (Fig. 12.31).

### Injuries to the periodontium

Periodontium injuries including lateral, extrusive and intrusive luxations and avulsions can frequently lead to concomitant injuries such as alveolar fracture. Taking into consideration that the impact is always in a bucco-lingual direction, lateral luxations are usually characterized by the displacement of the crown lingually and the root buccally, followed by a fracture of the labial bone plate. Accurate diagnosis of these injuries can be accomplished by reviewing the sagittal plane of 3D CBCT scans.

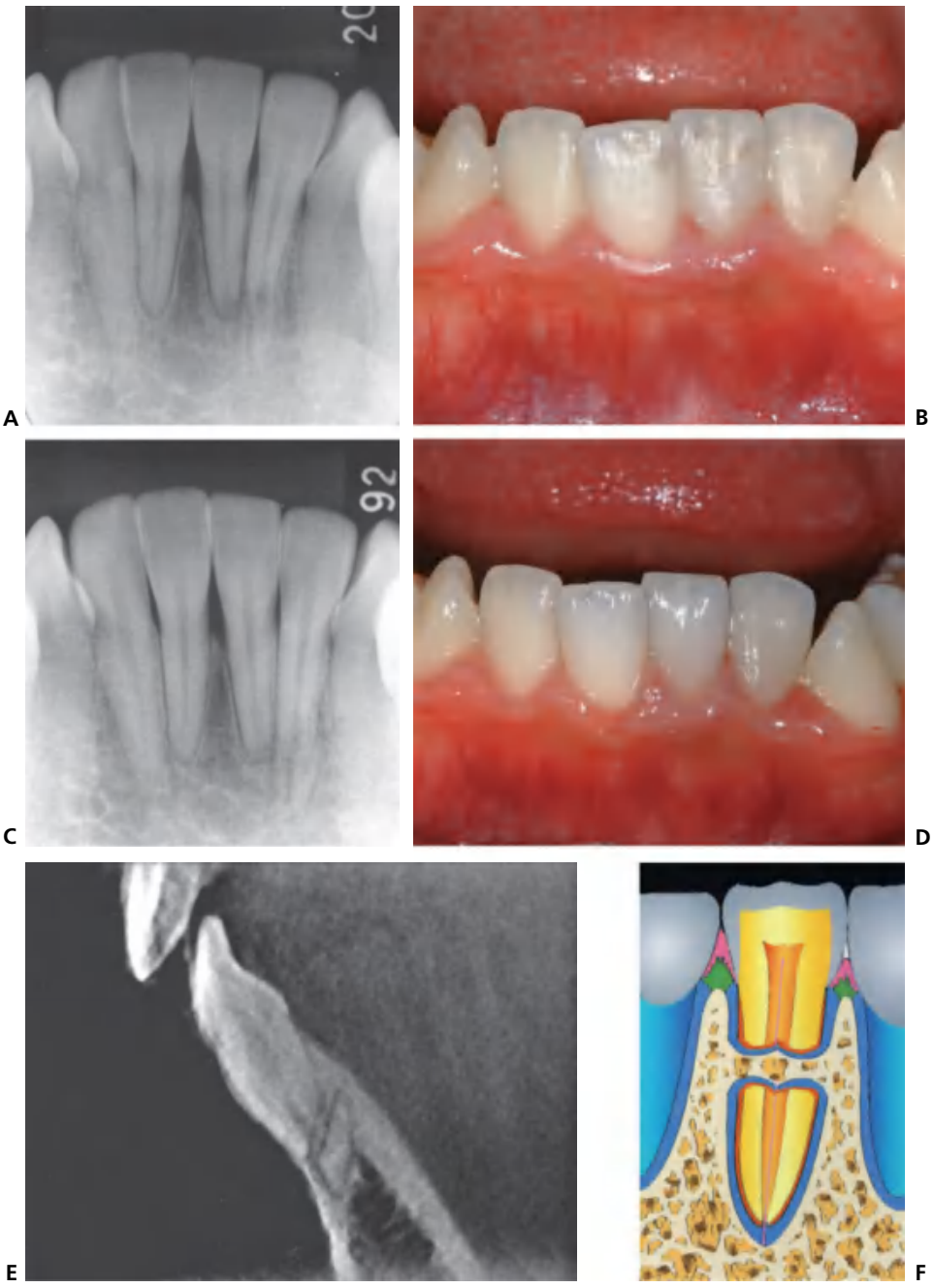



**Fig. 12.30** Root fracture. A, B. Conventional radiograph and clinical photograph taken at the initial examination of a 17-year-old male referred 2 months after injury. Although there were no symptoms, the patient's history disclosed that the right central incisor had suffered a subluxation injury. Examination revealed a root fracture at the apical third of the right lateral incisor. The crowns of the teeth were very slightly discolored. Both teeth showed negative EPT. C, D. Six months later, both teeth showed positive EPT. E. One year later, a 3DX image showed root fracture healing with interposition of the periodontal ligament and bone. F. Schematic illustration of this healing type.

### Pulpal and periapical healing

A part of pulp healing after luxation injuries can be transient apical breakdown which is usually combined with a root resorption process in the apical part of the root canal (129) (see Chapter 16). To diagnose the nature of this process a CBTC may play a role (Fig. 12.32).

A recent study showed that CBCT reduces the risk of false diagnosis and optimizes the chance of diagnosing periapical pathology (198).

### Root resorption

Common complications of dento-alveolar trauma are pulp necrosis, pulp canal obliteration, periapical pathosis and root resorption. The traumatic effect to the periodontium depends on the type and severity of the injury and can be related to different types of resorptions. These have been identified as repair related, infection related, ankyloses related or extra-radicular invasive cervical resorption (196). Eventually, the resorptive process may lead to tooth loss. Treatment of root resorption is often complex, time consuming, expensive and unpredictable. This is because with a plain 2D radiograph, the extent of the resorption and portals of entry cannot be visualized. In most cases the treatment requires a multidisciplinary approach of specialists from the following areas: endodontics, pediatric dentistry, periodontics, oral surgery, implantology, prosthodontics and orthodontics.

The diagnosis and 3D imaging assessment of the resorption is important in order to determine the treatment complexity and expected outcome based on the location and

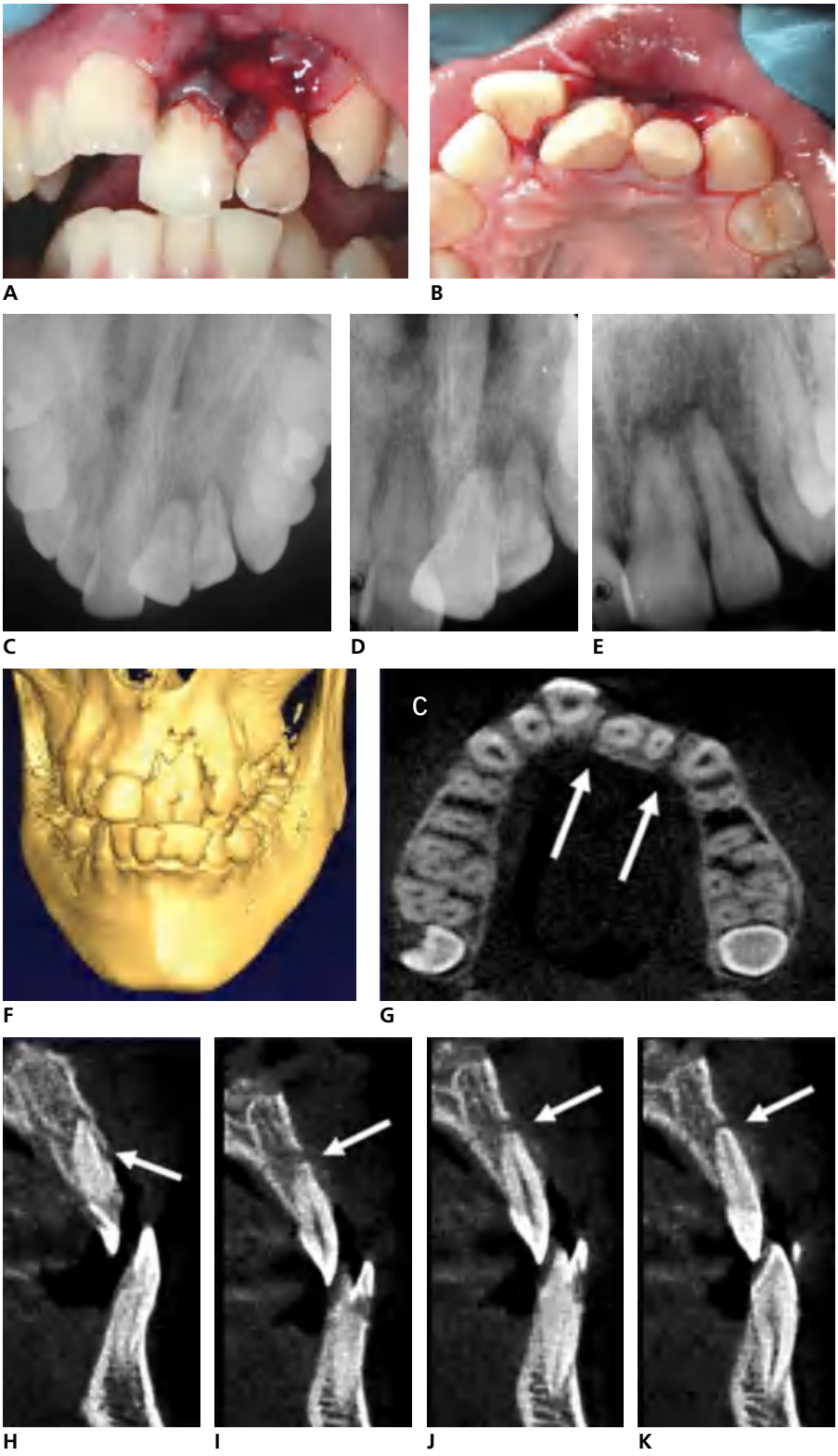



**Fig. 12.31** Cone beam computed tomography of an alveolar fracture. A, B. Preoperative clinical photographs immediately after admission at the emergency room. Intraoral examination showed a severe displacement of an alveolar fracture of both the maxillary left incisors and injury of adjacent soft tissue. C. Occlusal radiograph of the pre-maxilla. D, E. Periapical radiographs demonstrating periradicular radiolucencies associated with the traumatic displacement of the maxillary left incisors. F, G. Frontal reconstruction with the NewTom showing the severity of the impact and subsequent trauma to the left premaxilla. H–K. Different axial cuts revealing a severe palatal displacement of the maxillary left central incisor and alveolar fracture of the palatal bone. This series of cross-sectional 0.2 mm cuts showed lateral luxation of the teeth with concomitant alveolar fracture of the buccal cortical plate (see arrows). Reproduced from (184) with permission.

extension of the root defect. In addition, the proximity to anatomic structures is visualized for a surgical approach. Several studies (193–195) reported the geometric accuracy of CBCT, particularly regarding linear measurements. Lascala et al. (195) found that the measurements made from the CBCT images were similar to real *in vivo* distances between skull sites and it is therefore a reliable method for linear measurement between anatomic sites. Additionally, CBCT offers the advantage of a 3D visualization of the defect and facilitates determining the location, portal of entry/exit and the extension of the resorption (Fig. 12.33). Better preoperative assessment and understanding of the pathology can help the operator to find and repair the affected tissues, and identify treatable cases. 3D visualization should be used with discretion. It may enhance visibility of post-traumatic cellular processes, but might lead to over-diagnosis and hence over-treatment (203).

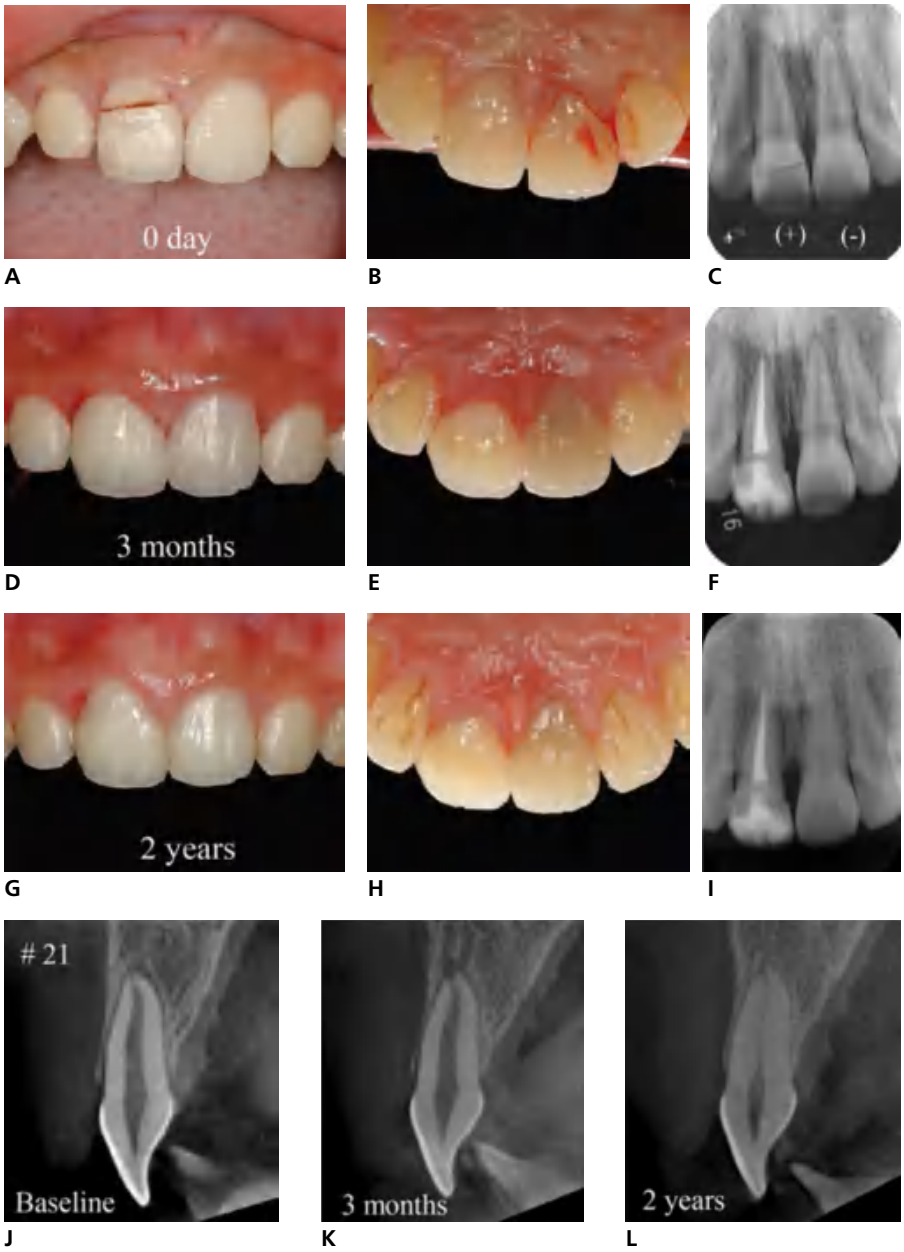



**Fig. 12.32** Cone beam computed tomography of a subluxated left central incisor with a transient apical breakdown. A–C. Clinical views and a radiograph of a 12-year-old boy who was referred 1 day after trauma. The right central incisor shows a crown-root fracture with positive EPT and the left central incisor is subluxated with negative EPT. D–F. Three months later. The right central incisor has been restored with composite after surgical extrusion and endodontic treatment. Note the discoloration of the left central incisor; the EPT is still negative. G–I. A clinical view and radiograph after 2 years. The color of the crown of the left central incisor has returned to normal and pulp canal obliteration has progressed. The EPT is now positive. J. A CBCT image of the left central incisor at the first visit. Note that the tooth is mature and the apex is almost closed. K. Three months later. Note the small area of apical bone resorption and root resorption on the inner surface of the apical part of the canal – typical features of transient apical breakdown. L. Two years later. Note that the pulp canal obliteration has progressed.

### *Magnetic resonance scanning*

Recently, magnetic resonance scanning with administration of a contrast medium was found to be able to demonstrate signs of revascularization of transplanted teeth earlier than a $CO_2$ test (172). However, the complexity of this procedure appears prohibitive as a suitable method for monitoring the healing status of traumatized teeth.

All radiographs should be stored carefully as they provide a record for comparison at future controls.

### *Photographic documentation*

The importance of a photographic examination as part of a complete examination was outlined in 1985 (101, 203). While photographic documentation is not specifically included in the current IADT guidelines, the clinician is referred to specific sources. The Dental Trauma Guide states 'photographic registration of the trauma is recommended, as it offers an exact documentation of the extent of injury and can be used later in treatment planning, legal claims, or clinical research' (220). Note that patient consent can be required.

## Effect of treatment delay

Few studies have examined possible relationships between treatment delay and pulpal and periodontal ligament healing complications. It has been commonly accepted that all injuries should be treated on an emergency basis, for the comfort of the patient and to reduce wound healing complications. For practical and especially economic reasons, various

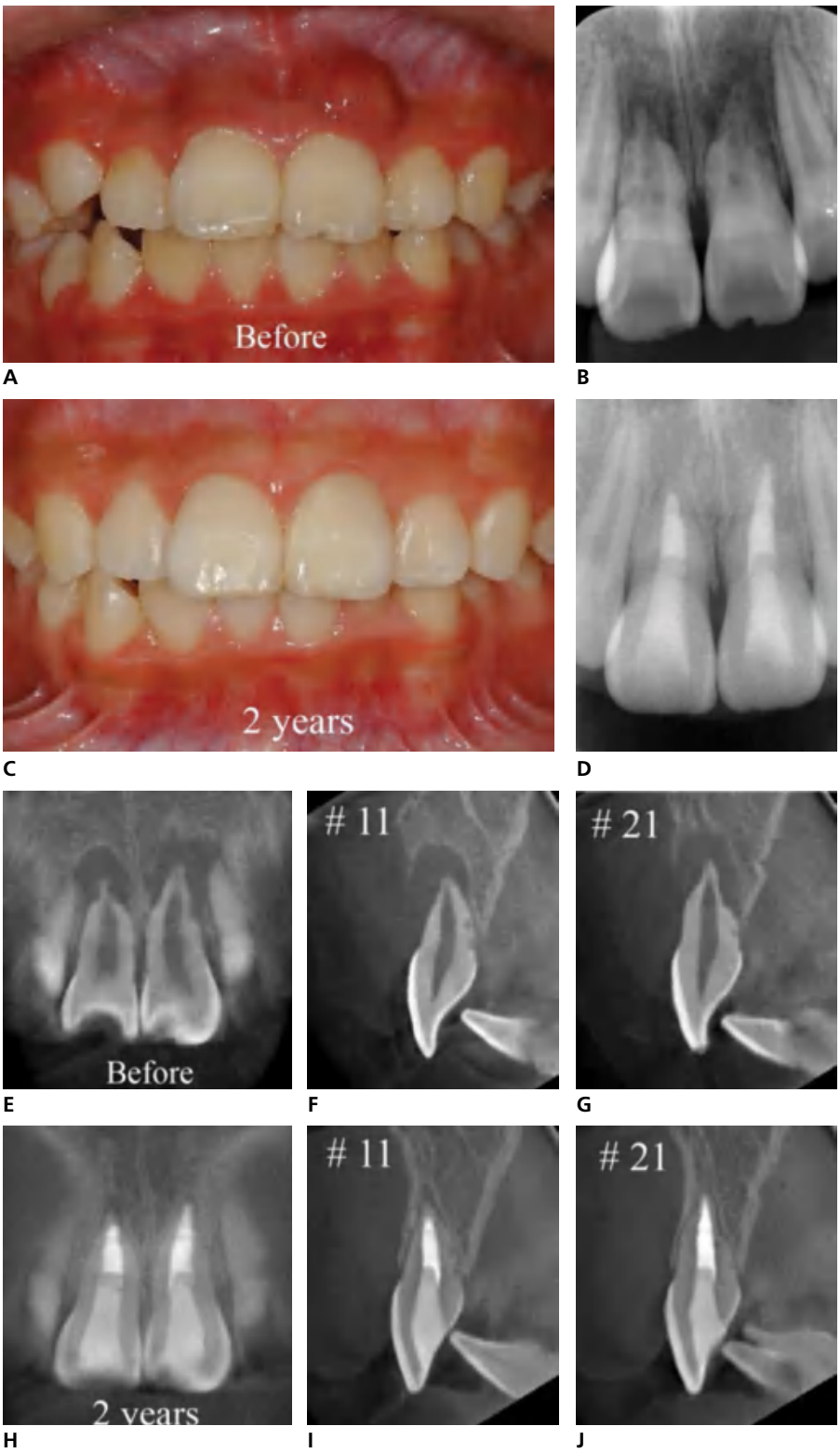



**Fig. 12.33** Cone beam computed tomography of the extent and location of infection-related root resorption as revealed by CBCT. A. A clinical view of an 11.5-year-old girl who was referred 3.5 months after a trauma. Both central incisors were avulsed and replanted after more than 1 hour. The teeth were left untreated thereafter. An abscess is observed on the buccal aspect of the left central incisor. B. A periapical radiograph taken before treatment. A large rarefaction and severe root resorption is observed affecting both incisors. C, D. A clinical view and a periapical radiograph taken after 2 years. Tooth mobility and gingival probing depth appear normal. No infra-occlusion is observed. Both teeth were endodontically treated and restored with composite. E–G. CBCT images before treatment. The CBCT images reveal the precise morphology of the bone and root resorption. Note that the buccal bone has been completely resorbed. Endodontic treatment was performed to eliminate bacteria in the canals. H–J. CBCT images after 2 years. Note that root resorption has been arrested and periodontal ligament healing is apparent. The buccal bone has reformed.

approaches can be selected to fulfill such a demand, such as *acute treatment* (i.e. within a few hours), *subacute treatment* (i.e. within the first 24 hours) and *delayed treatment* (i.e. after the first 24 hours). In a recent study, the consequences of treatment delay on pulpal and periodontal healing have been analyzed for various dental trauma groups (173). Applying such a treatment approach to various types of injuries, the following guidelines can be recommended, based on our present, rather limited knowledge of the effect of treatment delay upon wound healing:

- *Crown and crown-root fractures*: subacute or delayed approach
- *Root fractures*: acute or subacute approach
- *Alveolar fractures*: acute approach (evidence, however, is questionable)

- *Concussion and subluxation*: subacute approach
- *Extrusion and lateral luxation*: acute approach (evidence, however, is questionable)
- *Intrusion*: subacute approach (evidence, however, is questionable)
- *Avulsion*: if the tooth is not replanted at the time of injury, use an acute approach; otherwise use a subacute approach
- *Primary tooth injury*: subacute approach, unless the primary tooth is displaced into the follicle of the permanent tooth or occlusal problems are present; in the latter instances, an acute approach should be chosen

These treatment guidelines are based on very limited evidence from the literature and should be revised as soon as more evidence about the effect of treatment delay becomes available.

### Follow-up examinations

Standardized reporting of traumatic injuries is essential to validate any pathologic change or failure in continued maturogenesis of the root in subsequent follow-up examinations. The timing of follow-up appointments is outlined in the IADT and AAE guidelines.

## Essentials

### History

- Patient's name, age, sex, address and telephone number
- When did the accident occur?
- Where did the accident occur?
- How did the accident occur?
- Treatment elsewhere
- History of previous dental injuries and general health
- Did the trauma cause amnesia, unconsciousness, drowsiness, vomiting or headache?
- Is there spontaneous pain from the teeth?
- Do the teeth react to thermal changes, sweet or sour foods?
- Are the teeth painful to touch or during eating?
- Is there any disturbance in the bite?

### Clinical examination

- Recording of extraoral wounds and palpation of the facial skeleton
- Recording of injuries to the oral mucosa or gingiva
- Examination of the tooth crowns for the presence and extent of fractures, pulp exposures or changes in color
- Recording of displacement of teeth (i.e. intrusion, extrusion, lateral displacement or avulsion)
- Abnormalities in occlusion
- Abnormal mobility of teeth or alveolar fragments
- Palpation of the alveolar process
- Reaction and sound of teeth to percussion

### Reaction of teeth to sensibility tests

- Mechanical stimulation
- Heated gutta-percha (Fig. 12.10)
- Ice (Fig. 12.11)
- Ethyl chloride (Fig. 12.12)
- Carbon dioxide snow (Fig. 12.13)
- Dichlor-difluormethane
- Electric pulp testers (Figs 12.15 and 12.16)
- Laser Doppler flowmetry (Fig. 12.18)
- Pulse oximetry (Fig. 12.21)

### Radiographic examination

- Intraoral radiographs (Figs 12.22 and 12.23)
- Extraoral radiographs (Fig. 12.25)
- Panoramic radiographs
- CBCT scanning
- Micro CT scanning (Figs 12.26 and 12.27)
- Magnetic resonance scanning

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# 13

# Crown Fractures

F. M. Andreasen, E. Lauridsen & J. O. Andreasen

## Terminology, frequency and etiology

The following classification of crown fractures is based upon anatomic, therapeutic and prognostic considerations (Fig. 13.1).

(1) *Enamel infraction*: an incomplete fracture (crack) of the enamel without loss of tooth substance.
(2) *Enamel fracture*: a fracture with loss of tooth substance confined to enamel (uncomplicated crown fracture).
(3) *Enamel-dentin fracture*, a fracture with loss of tooth substance confined to enamel and dentin, but not involving the pulp (uncomplicated crown fracture).
(4) *Enamel-dentin fracture involving the pulp* (complicated crown fracture).

In the permanent dentition, crown fractures comprise 26–76% of dental injuries (1–4, 94–96, 265). The most common etiologic factors of crown and crown-root fractures in the permanent dentition are injuries caused by falls, contact sports, automobile accidents or foreign bodies striking the teeth (1, 95).

Enamel fractures comprise a very common occupational hazard among glassblowers, presumably due to impact by the blowpipe (153).

## Clinical findings

*Enamel infractions* are very common but often overlooked. These fractures appear as crazing within the enamel substance which stop at the dentino-enamel junction and may appear with or without loss of tooth substance (i.e. uncomplicated or complicated crown fractures) (5, 6, 97, 155). Infractions are caused by direct impact to the enamel, e.g. traffic accidents and falls, which explains their frequent occurrence on the labial surface of upper incisors (155). Various patterns of infraction lines can be seen depending on the direction and location of the trauma, i.e. horizontal, vertical or diverging. Infractions are often overlooked if direct illumination is used, but are easily visualized when the light beam is directed perpendicular to the long axis of the tooth from the incisal edge (Fig. 13.2). Fiberoptic light sources are also very useful in detecting infractions. By modifying the intensity of the light beam, many infractions become readily visible (154). Infractions are often the only evidence of trauma, but can be associated with other types of injury. Thus the presence of infraction lines should draw attention to the possible presence of associated injuries, especially to the supporting structures.

*Enamel fractures* are very frequent and only involve enamel. As these fractures do not involve dentin they appear to have a different prognosis in relation to pulp necrosis (291).

*Enamel-dentin fractures* without pulpal involvement occur more often than complicated crown fractures in both the permanent and primary dentitions (3, 7–9, 94, 98, 99). They are often confined to a single tooth, usually the maxillary central incisors (7, 10, 98), especially the mesial or distal corners (7) (Fig. 13.3). Fractures can be horizontal, extending mesiodistally. Occasionally only the central lobe of the incisal edge is involved (Fig. 13.4). In rare cases, the fracture can involve the entire facial or oral enamel surface.

Although not frequently found in combination with extrusive and lateral luxation injuries (156, 157), crown fractures are frequently seen in teeth with concussion, subluxation and intrusion (1, 158, 159). This combination of luxation injury and crown fracture is of prognostic importance. A very unusual finding is that of crown fractures of non-erupted permanent teeth due to trauma transmitted from impact to the primary dentition (100, 101, 262).

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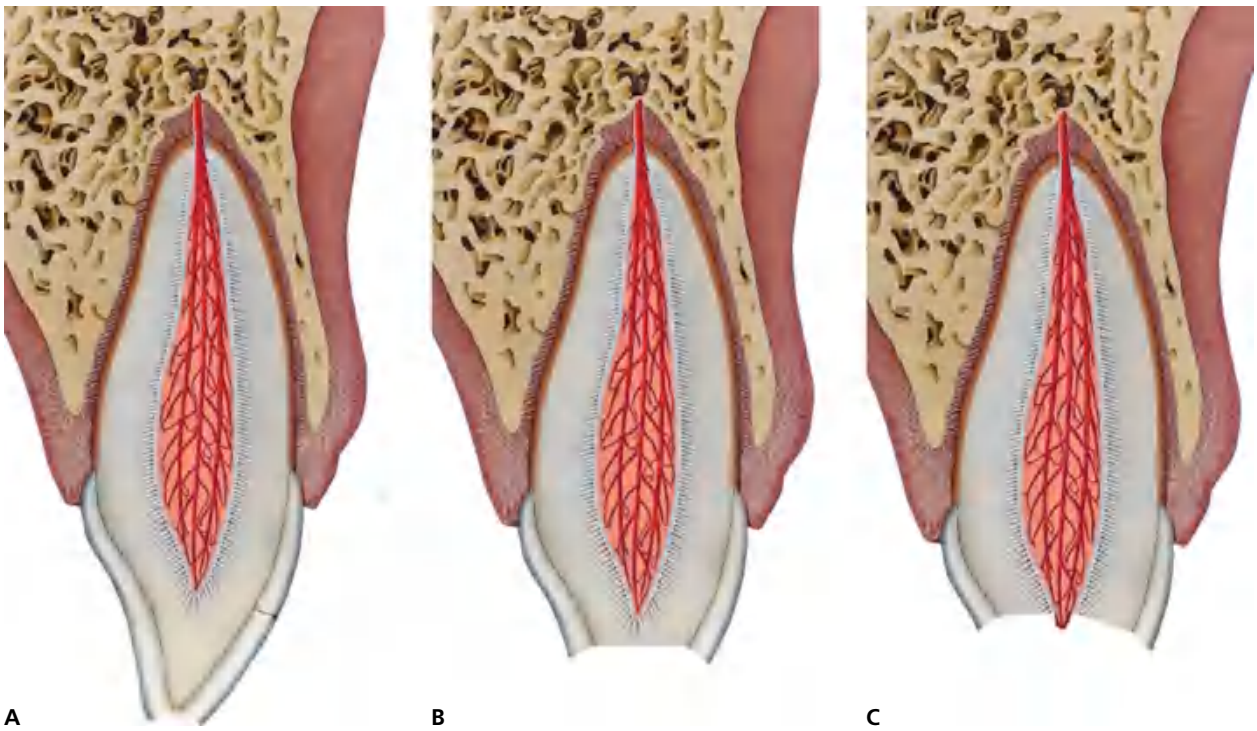



**Fig. 13.1** Schematic drawings illustrating different types of crown fractures. A. Crown infraction and uncomplicated crown fracture without involvement of dentin. B. Uncomplicated crown fracture with involvement of dentin. C. Complicated crown fracture.

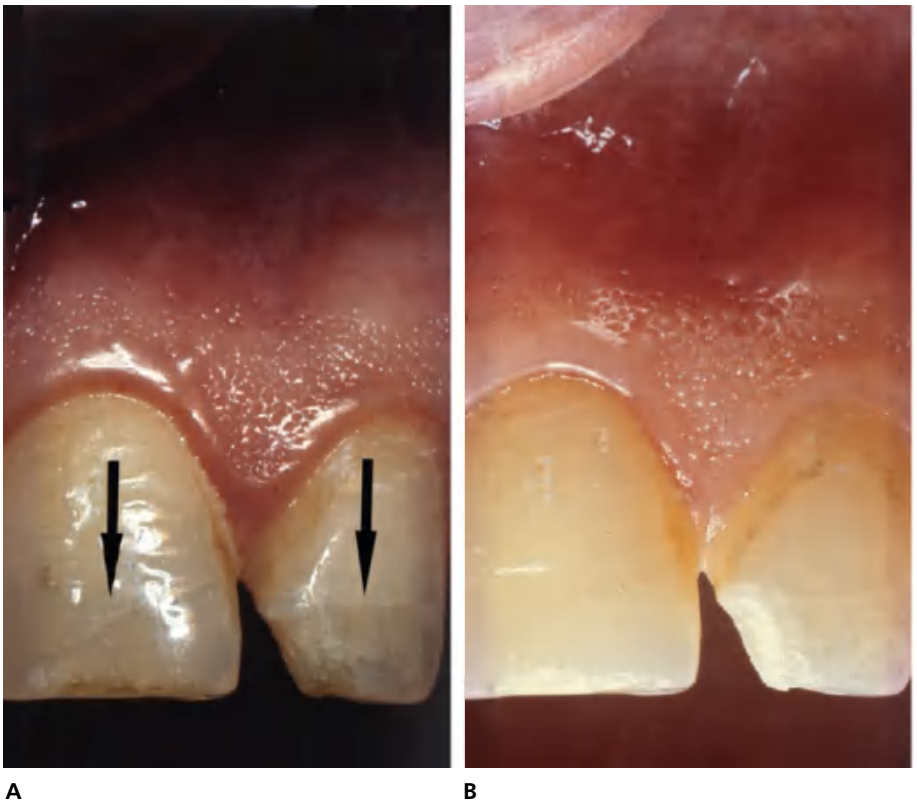



**Fig. 13.2** Infraction lines involving the right central and lateral incisors. The use of indirect illumination reveals the infraction lines (A) although they are barely visible by direct illumination (B).

Examination of fractured teeth should be preceded by thorough cleansing of the injured teeth with a water spray. This is followed by an assessment of the extent of exposed dentin as well as a careful search for minute pulp exposures.

Dentin exposed after a crown fracture usually gives rise to symptoms such as sensitivity to thermal changes and mastication, which are to some degree proportional to the area of dentin exposed and the maturity of the tooth (160).

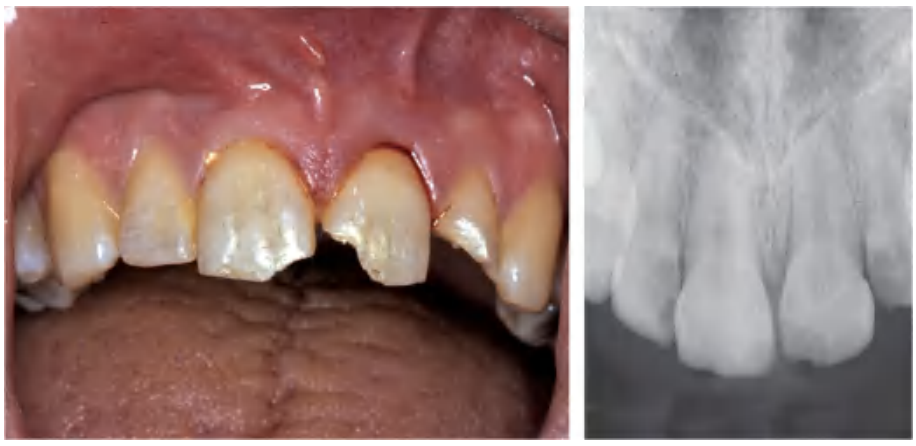


**Fig. 13.3** Central and lateral incisors with typical uncomplicated crown fractures involving the mesial corners.

**Fig. 13.4** Uncomplicated crown fracture involving the central lobe of the incisal edge.

The layer of dentin covering the pulp may be so thin that the outline of the pulp is seen as a pinkish tinge. Clinical examination should also include sensibility testing as a point of reference for later evaluation of pulpal status (see Chapter 9) (102).

*Complicated crown fractures* usually present with a slight hemorrhage from the exposed part of the pulp (Fig. 13.5). Proliferation of pulp tissue (i.e. pulp polyp) can occur when treatment in young teeth is delayed for days or weeks (Fig. 13.6). Pulp exposure is usually followed by symptoms, such as sensitivity to thermal changes.

## Radiographic findings

The radiographic examination adds important information to the clinical evaluation which can influence future treatment, such as the size of the pulp and the stage of root development (161–163). Moreover, the radiograph serves as a record for comparison at later visits. This is especially true in the verification of a hard tissue barrier over an exposed pulp when clinical verification is not possible. However, it should be borne in mind that a radiograph can only provide an estimate of pulpal dimensions; and that the pulp cavity is usually larger and the distance between the pulp horns and the incisal edge is usually smaller than that shown radiographically (161). In rare cases, displacement of primary teeth may lead to a crown fracture of a permanent successor, a finding which can be shown radiographically (Fig. 13.7).

## Healing and pathology

*Enamel infractions* can be seen in ground sections, where they appear as dark lines running parallel to the enamel rods and terminate at the dentino-enamel junction (11) (Fig. 13.8).

*Enamel-dentin crown fractures* expose a large number of dentinal tubules. It has been estimated that the exposure of 1 $mm^2$ of dentin exposes 20 000 to 45 000 dentinal tubules (12, 103). Dentinal tubules constitute a pathway for bacteria and thermal and chemical irritants which can provoke pulpal inflammation, for which reason dentin-covering procedures described later in this chapter and in Chapter 22 are necessary. The speed of bacterial penetration into prepared dentin left exposed to saliva and plaque formation *in vivo* was found by Lundy and Stanley (164) to be 0.03–0.36 mm 6–11 days after preparation and 0.52 mm after approximately 84 days. No studies have so far studied the progress of bacteria after fracture exposure of dentin (166). In one experimental study in monkeys, bacteria were formed in dentinal tubules after 3 months in both treated (composite and fragment bonding) and non-treated teeth. The presence of bacteria in tubules was related to significant hard tissue formation in the coronal part of the pulp (264).

These findings are supported by a study in monkeys, where induced fractures were either restored with Dycal® and resin composite or by reattachment of the crown fragment with the

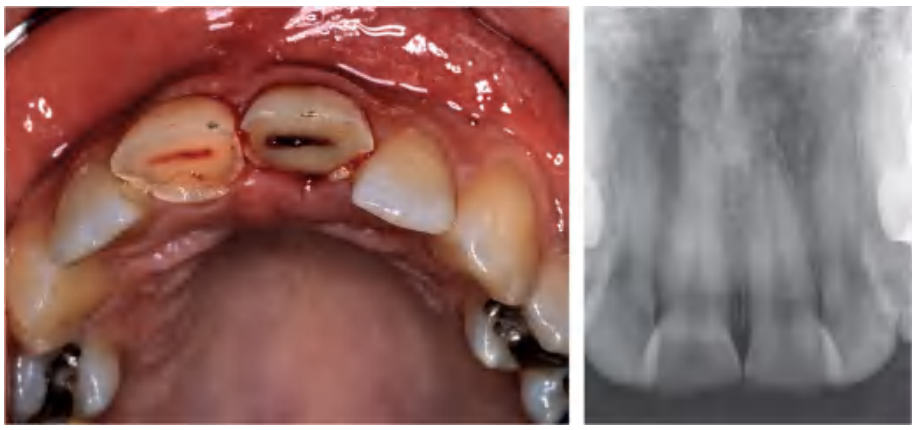
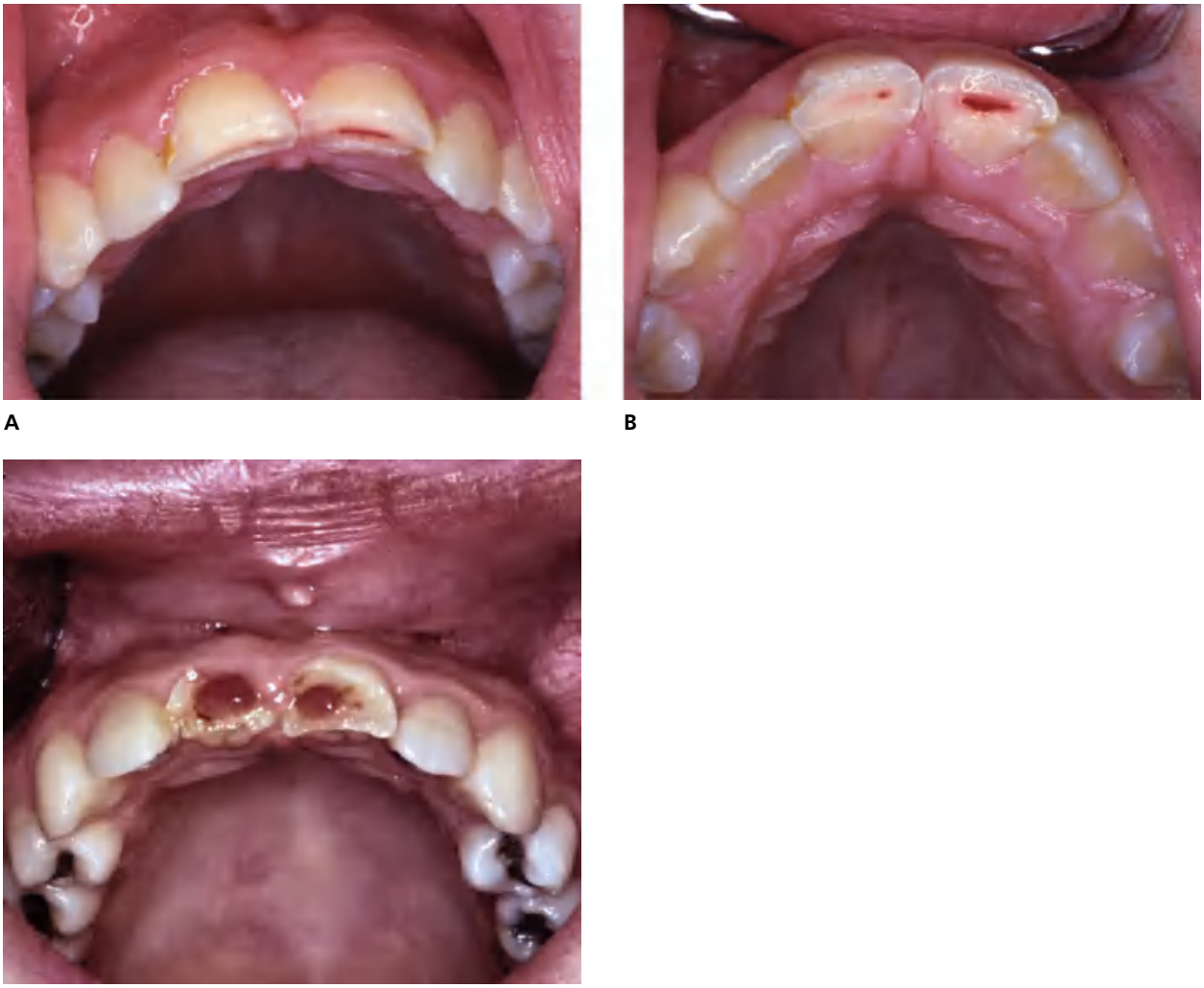


**Fig. 13.5** Complicated crown fractures showing difference in pulp circulation illustrated by variations in color of the exposed pulp.



**A** **B** **C**

**Fig. 13.6** Complicated crown fractures of permanent incisors. A and B. Small pulp exposures of central incisors. C. Pulp proliferation in a case of complicated crown fractures left untreated for 21 days.

GLUMA® dentin bonding system. After 3 months, the general appearance was hard tissue deposition in the coronal portion of the pulp, and inflammatory infiltrates were seen in only a few teeth and then as clusters of mononuclear leukocytes. More extensive hard tissue formation correlated with the presence of bacteria in dentinal tubules (263).

The ingrowth of bacteria is to a certain extent inhibited by an outward flow of dentinal fluid within the tubules due to a

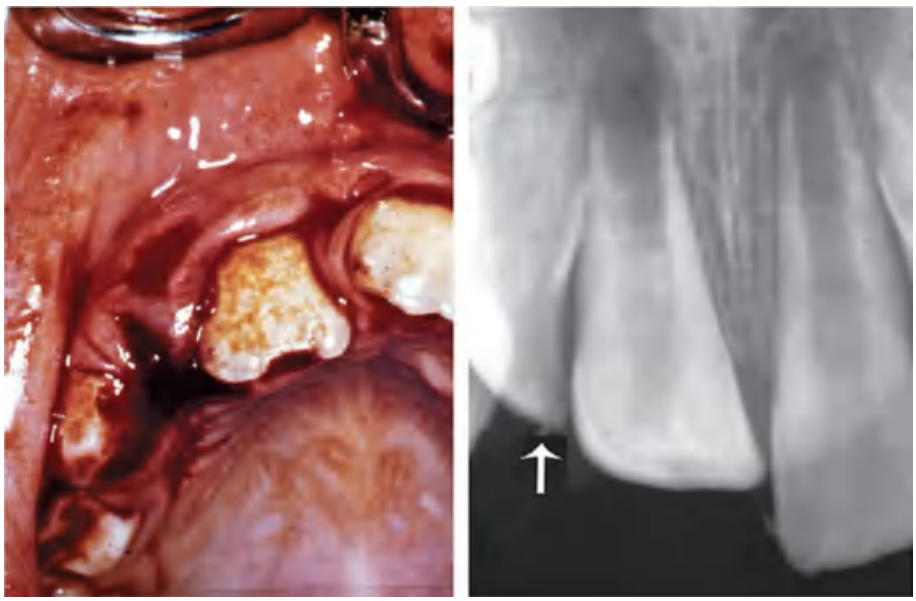


**Fig. 13.7** A primary tooth injury has resulted in an enamel fracture of the non-erupted, lateral incisor (arrow).

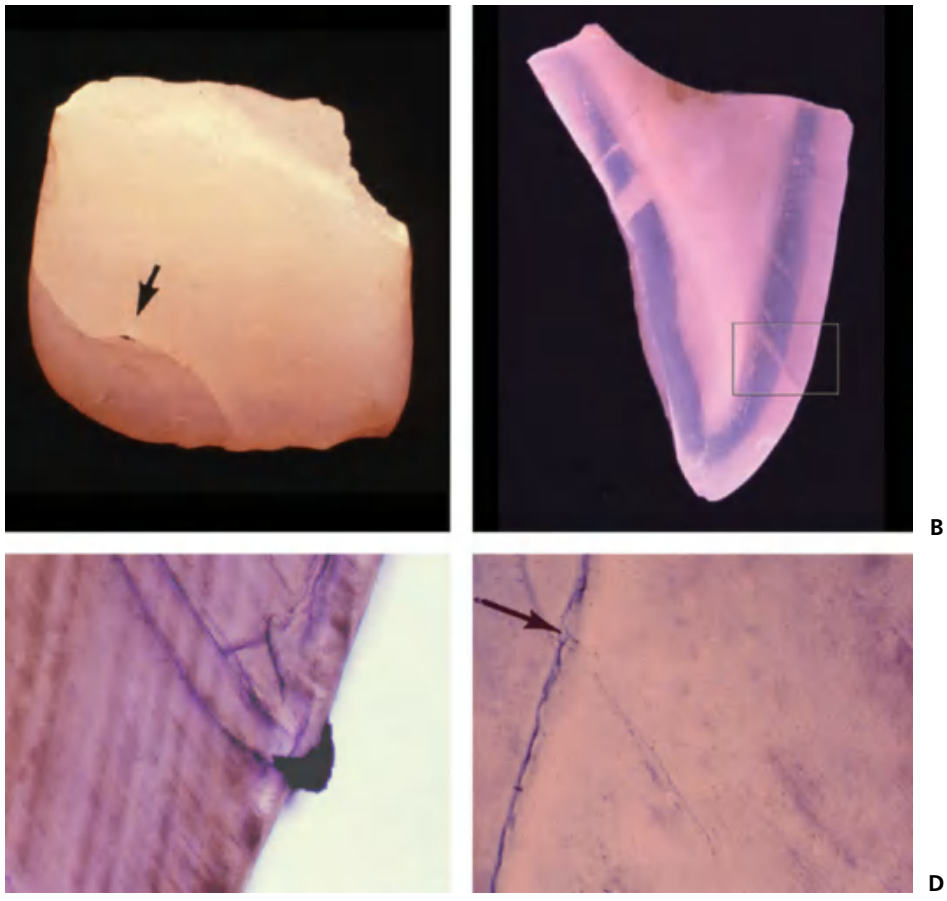
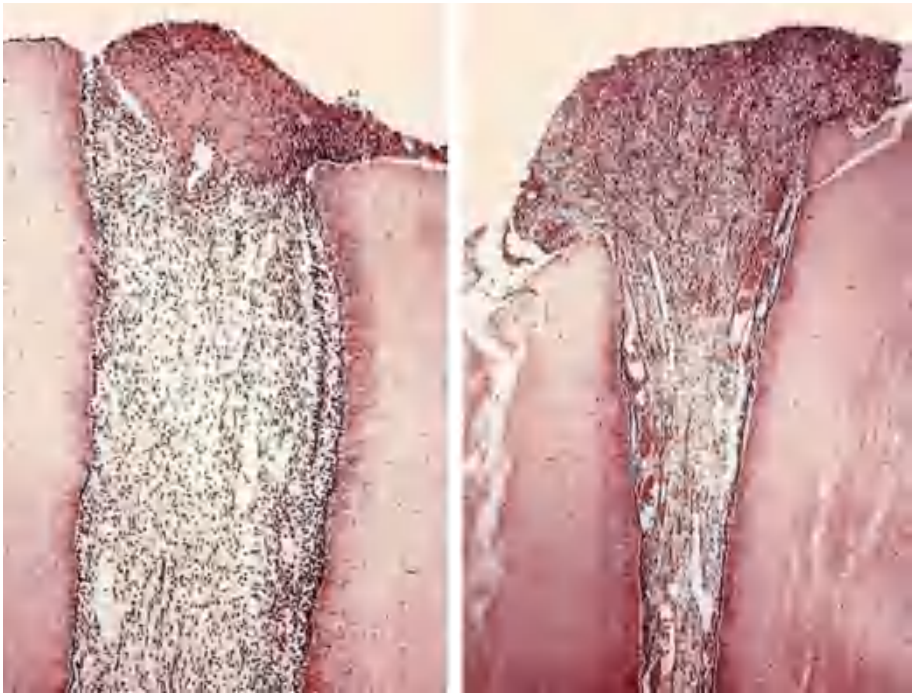



**Fig. 13.8** Histologic features of a permanent central incisor showing crown infractions. A. Gross specimen. Note the point of impact (arrow) with irradiating infraction lines. B. Low power view of ground section through the impact area. ×8. C. Facial aspect of the crown exhibiting an infraction line. ×30. D. Higher magnification of (C) reveals that the line follows the direction of the enamel prisms. ×195.

positive pulpal pressure (264, 281, 282). In contrast, bacterial penetration is more rapid where impeding hydrostatic pressure from an outward pulpal fluid flow is minimal or non-existent, as after concomitant luxation injuries where there is a compromised pulpal blood supply (166–171, 292–294). Experimental studies *in vivo* in cats have thus demonstrated an increased fluid flow from exposed dentinal tubules with an intact pulpal blood supply, presumably due to a chain of events involved in neurogenic inflammation arising from dentinal irritation following exposure and subsequent stimulation of, for example, the inferior alveolar nerve (167–171). This fluid flow might mechanically



A B

**Fig. 13.9** Immediate and histologic response to a crown fracture in a monkey. A. The inflammation is very superficial after 24 hours. B. After 1 week the pulp shows proliferation and still very limited inflammation.

inhibit bacterial ingress through patent dentinal tubules, and also distribute antibodies (166).

This increase in fluid flow after dentin exposure might also have clinical implications with respect to moisture control in the use of dentin bonding agents. Thus, it might seem advisable to administer a local anesthetic prior to such procedures to reverse dentinal fluid flow due to neurogenic stimulation (172). However, further investigation is necessary to test this theory before it can be advocated universally.

Little information exists about pulpal changes after *enamel-dentin crown fractures*. Experimental studies have demonstrated inflammatory changes when artificially exposed dentin was left uncovered for 1 week (13, 14). However, recent research suggests that the inflammatory changes are of a transient nature if the pulpal vascular supply remains intact and bacterial invasion is prevented (see later in this chapter) (173).

Histologically, exposed pulp tissue in *complicated crown fractures* is quickly covered by a layer of fibrin. Eventually, the superficial part of the pulp shows capillary budding, numerous leukocytes and proliferation of histiocytes. This inflammation spreads apically with increasing observation periods. However, experimental studies in permanent teeth of monkeys have shown that the inflammatory process does not usually penetrate more than approximately 2 mm in an apical direction (104, 174), a finding which is of clinical significance in treatment planning (see Chapter 22).

Complicated crown fractures that are left untreated for longer periods of time normally show extensive proliferation of granulation tissue at the exposure site (Fig. 13.9). However, rare cases of spontaneous closure of the perforation with hard tissue have been reported (15, 105).

## Treatment

Many authors have discussed treatment principles for crown fractures in the *permanent dentition* (16–59). For reviews, the reader is referred to excellent reports published by Rauschenberger and Hovland (1995) (265), Blatz (2001) (266) and Olsburgh et al. (2002) (267). Therapeutic considerations comprise *pulpal* response to injury and treatment as well as the *prosthetic* considerations arising at the time of injury and final, definitive treatment. The reader is referred to Chapter 25 regarding pulpal considerations following crown fracture. The emphasis in the present chapter will be placed upon emergency treatment procedures. Final restorative treatment using composite resin will be described in Chapter 29.

### Enamel infractions

While infractions in enamel, dentin and cementum in the *posterior* region are often implicated in the 'cracked tooth syndrome', enamel infractions in *anterior* teeth following acute trauma do not appear to imply the same risk to tissue integrity due to the fact that these infractions are usually limited to enamel and stop at the dentino-enamel junction (Fig. 13.8). However, due to frequently associated injuries to periodontal structures, sensibility tests should be carried out in order to disclose possible damage to the pulp. In a recent study analyzing combination injuries, a significantly higher risk of pulp necrosis was reported in teeth with subluxation and concomitant enamel infraction (293). Moreover, in a series of retrospective investigations into the long-term prognosis of luxation injuries in the permanent dentition, an

A B C

**Fig. 13.10** Grinding procedure used in the treatment of an uncomplicated crown fracture confined to enamel. A. Before treatment. B. After grinding of injured tooth. C. Grinding of the non-injured incisor in order to restore symmetry.

exclusion criterion was the presence of concomitant crown fractures. However, enamel infractions were admitted. In these studies, all extruded, laterally luxated and intruded incisors with infractions developed pulp necrosis, albeit their number were small (295). These findings lead to the suspicion that an enamel infraction can be a port of entry for bacteria if the blood supply to the pulp is compromised due to a concomitant luxation injury.

As a rule, enamel infractions do not require treatment. However, in case of multiple infraction lines or in the case of concomitant luxation injury, the indication might be to seal the enamel surface with an unfilled resin and acid-etch technique.

## Enamel and enamel-dentin crown fractures

Immediate treatment of crown fractures *confined to enamel* can be limited to smoothing of sharp enamel edges to prevent laceration of the tongue or lips. Selective reduction can be undertaken at the same time or at a later visit with good esthetic results, especially in imitating an accentuated rounding of a distal corner (47, 60) (Fig. 13.10). However, because of esthetic demands for midline symmetry, a fractured mesial corner cannot usually be corrected in the same way. Selective reduction can in some cases be combined with orthodontic extrusion of the fractured tooth in order to restore incisal height. However, cervical symmetry must also be considered (see Chapter 36) (61, 175).

When the shape or extent of the fracture precludes recontouring, a restoration is necessary.

Whatever treatment is decided, it is essential that the crown's anatomy and occlusion be restored immediately in order to prevent labial protrusion of the fractured tooth, drifting or tilting of adjacent teeth into the fracture site (6, 34, 63, 106) or overeruption of opposing incisors (60, 64) (Fig. 13.11).

An isolated fracture of enamel does not appear to represent any hazard to the pulp. However, in crown fractures with *exposed dentin*, therapeutic measures should be directed towards dentin coverage in order to avoid bacterial ingress, thereby permitting the pulp to recover and elicit repair.

**Fig. 13.11** Untreated crown fracture. Note drifting and tilting of adjacent teeth into the fracture area. Courtesy of Dr. J. J. Ravn, Department of Pedodontics, Royal Dental College, Copenhagen, Denmark.

Immediate reattachment of the original fragment or restoration with a composite resin (see Chapter 29) is normally to be preferred over a temporary filling or temporary crown. The most significant disadvantage of temporary restorations is the potential risk of leakage which permits access of bacteria to the exposed dentin and thereby represents a significant threat to the recovery of the pulp.

It was in the 1970s that the impact on pulpal healing of bacterial invasion and colonization in the gap between the tooth and the restorative material after exposure of dentin was first appreciated (165, 176–201, 268, 269).

Thus, when bacteria are excluded from the tooth–restoration interface experimentally by placement of a superficial layer of zinc oxide-eugenol cement over the restorations or liners, healing with formation of a hard tissue bridge can be achieved even after direct pulp capping with silicate cement, zinc-phosphate cement, amalgam and light-cured composite resin (197, 198, 202–205). It would therefore seem that the initial cytotoxic effect of these materials placed on or

near the human pulp is not greater than that which is seen after capping with calcium hydroxide (160).

Experimentally it has been found that in teeth with vital pulp tissue, dentin can provide considerable resistance to bacterial invasion and that dentin which has been exposed to the oral environment for longer periods of time appears to be less permeable than fresh dentinal wounds (201). Furthermore, bacterial irritation via an area of exposed dentin causes limited pulpal inflammation with little permanent damage (206).

As support for these findings, long-term clinical studies have shown very little pulpal response (e.g. pulp necrosis or pulp canal obliteration) to either crown fracture with or without pulp exposure or subsequent restorative procedures as long as there was not a concomitant periodontal injury (194, 207–214, 292–294).

#### *Treatment strategy*

Review articles (265–267) have addressed this problem and should be consulted. In light of current knowledge concerning the role of bacterial influences associated with marginal leakage, one must conclude that the primary objective of restorative procedures following crown fractures which expose dentin is to prevent injury to the pulp. Such pulpal injury is initiated by bacteria and bacterial components in the event that the restoration does not provide an adequate marginal seal (211).

### Use of dental bonding agents: should exposed dentin be lined?

Until recently, strategy for the treatment of enamel-dentin fractures without pulp exposure has been dentin coverage with a hard-setting calcium hydroxide-containing liner. Clinical experience, however, indicates that hard-setting calcium hydroxide cements disintegrate beneath dental restorations with time. This finding has been confirmed both experimentally *in vitro* (215, 216) and *in vivo* (191, 217). Moreover, cultivable and stainable bacteria have been found within the calcium hydroxide liners used in both exposed and pulp-capped and non-exposed control teeth and these are thus unable to provide a permanent barrier against microleakage (191, 217). Finally, it has been found *in vitro* that calcium hydroxide liners can have the same softening effect on composite resins as zinc oxide-eugenol liners (218). The long-term benefit of their use is therefore questionable (219).

What appears to be critical to pulpal healing is how effectively the dentin is sealed from bacterial irritants. If deeply exposed dentin is adequately sealed, the non-exposed pulp will form reparative dentin even without calcium hydroxide (see Chapter 2).

Microleakage around composite resin restorations is one of the major causes of restoration failure (220). Microleakage can be counteracted in part by a strong micromechanical bond arising between a composite resin and acid-etched *enamel* (221–223). If *dentin* bonding is also employed, it has been shown experimentally that the bonding strength of reattached crown fragments is approximately three times greater than if acid-etched enamel is the only source of retention (224).

The goal of a dentin bonding agent is, therefore, to supplement enamel acid etching by mediating a bond between an adhesive resin and the dentin surface. This would imply a hermetic seal against the oral flora and optimize pulpal healing after fracture.

As the bond strength of any dentin bonding system is proportional to the surface area of available unlined dentin for bonding, it is important that any calcium hydroxide liner does not cover more dentin than is absolutely necessary. In Chapter 29 various bonding systems and restorative material are described in detail.

### Enamel-dentin crown fractures with pulp exposure

The principles for the restoration of crown fractures with pulpal involvement differ from those of uncomplicated crown fractures only with respect to treatment of the exposed dental pulp. This implies pulp capping, partial pulpotomy or pulpal extirpation, which are described in Chapter 25.

## Provisional treatment of crown fractures

### Uncomplicated fractures

Several approaches exist, depending on the trauma setting (i.e. whether private practice or an emergency room) and the need for an immediate esthetic solution. All approaches rest on the premise of creating a hermetic seal against bacterial invasion into dentinal tubules.

### Glass ionomer cement 'bandage'

This is a very simple procedure, whereby light- or chemically activated glass ionomer cement is used to cover the exposed dentin and adjacent enamel (Fig. 13.12). The advantage of using glass ionomer instead of composite in this situation is that no etching is required and that the relatively low bond strength of glass ionomer to enamel and dentin facilitates its removal.

Before application, the tooth surfaces are rinsed with a water spray or saline. Increments of glass ionomer cement can be used to build up the 'bandage' esthetically. This procedure is especially useful in the case of multiple fractures and limited resources for definitive restorative procedures (e.g. emergency room settings).

All of these findings indicate that glass ionomer may be an ideal material for temporary coverage (bandage) or as a liner for deep fractures before restoration with composite restoration.

**Fig. 13.12 Provisional coverage of an enamel-dentin fracture with glass ionomer cement**
A glove-covered finger can be used to mold the palatal surface of the glass ionomer.

Application of glass ionomer.

Molding and curing the glass ionomer.

The finished glass ionomer bandage.

### Resin or celluloid crowns

When esthetic demands are foremost, a temporary acrylic crown should be considered. Various types of prefabricated temporary crowns are available. Resin or celluloid crown forms have too little strength for this purpose and should only be used as a mold for the crown. After placement of a calcium hydroxide liner or glass ionomer over the fracture surface, a suitable crown form is selected and contoured to fit the fractured tooth. A hole is made with a sharp explorer through the mesial and/or distal incisal corner to permit escape of excess crown material during placement. The fitted form is then filled with a composite resin or resin material, seated, and excess resin removed. When polymerized, the crown is removed, finished and cemented (60, 150). The crown is finished short of the gingival margin in order to permit optimal gingival health and prevent the restorative material from being forced into the injured periodontal ligament.

### Splints as dentin/pulpal coverage

In case of concomitant injuries to the periodontium, dentin and pulp protection must be incorporated into the splint. In these instances, it is a good idea for ease of splint removal and later restoration to cover the exposed enamel and dentin with a calcium hydroxide liner or glass ionomer before application of an acid-etch/resin splint. Enamel coverage with a liner is especially important in the case of later reattachment of the fractured crown fragment to ensure an intact enamel margin to which the fragment can be bonded (see later). In the case of profound crown fracture and simultaneous need for splinting, a fiber-reinforced splint can be the solution for satisfactory retention until splint removal and final restoration (225, 226).

## Definitive treatment of crown fractures

Because of recent improvements in dental materials available, the boundary between provisional and definitive treatment has become less distinct.

Various cast semipermanent restorations (e.g. basket crowns, gold-acrylic open-faced crowns and pinledge inlays) have been designed in the past to meet the esthetic demands of a young traumatized permanent dentition until such time as definitive treatment was considered appropriate (60, 64, 65, 67–80, 82–93, 152). However, with the advent of composite resin materials and the acid-etch technique (107–149, 221–223, 227, 228), the indications for these restorations has been eliminated. Moreover, in many situations, considering the life expectancy of these new materials, other definitive treatments of the past might not be necessary or might actually be overtreatment.

Definitive restoration of crown fractures presently consists of composite restorations (see Chapter 29) (291), reattachment of the original crown fragment (see later), full crown coverage (see Chapter 31) (65, 69, 73, 81–93), partial crown coverage or laminate veneers (see Chapter 30) (229, 230).

## Reattachment of the original crown fragment

Reattachment of the fragment provides several advantages over other forms of dental restoration following crown fracture (Fig. 13.13). It results in exact restoration of crown and surface morphology in a material that abrades at the same rate as adjacent teeth. Chair time for the completion of the restoration is minimal, normally requiring less time than that needed for completion of a temporary restoration.

Prior to the advent of dentin bonding agents, the only available method of fragment reattachment was by the use of enamel acid etching. Preparation of the tooth and avulsed fragment included either an internal bevel of enamel on both fracture surfaces (231) or simply hollowing out of the dentin of the fragment fracture surface to accommodate a thickness of hard-setting calcium hydroxide liner on the fracture surface of the tooth. This was then followed by enamel etching and adhesion of fragments using a creamy mixture of unfilled and filled composite resin on both fracture surfaces (231–244). Reports of these procedures have been anecdotal and with mixed long-term success. The concept of fragment reattachment was, therefore, re-evaluated with the development of new dental bonding agents in the mid-1980s (245–252, 270).

Recently, it has been shown in *in vitro* testings that beveling along the fracture line may increase the fracture resistance (279, 280, 283).

In animal studies, the bonding system has been proved safe for pulpal tissues as long as a 0.2 mm thickness of dentin remains between the pulp and the bonding surface. However, the adverse pulpal response seen after 8 days when the remaining dentin thickness was less than 0.2 mm was not evident at 90 days (253).

At the time the GLUMA® dentin bonding system was introduced and crown fracture bonding initiated, it was one of the most promising of the dentin bonding agents available. Since then, significant advances have been made in the area of dentin bonding agents and in bond strength to dentin (255–259, 271–278, 284, 285). Fracture strengths have been achieved that are not significantly different from those of intact teeth (272), although present laboratory tests cannot simulate a traumatic injury (273). But, at least in the case of reattachment of crown fragments, recent experiments have shown that successful fragment bonding can be achieved irrespective of the dentin bonding agent used as long as the manufacturer's directions are followed closely (224, 260). The choice of bonding agents is further discussed in Chapter 29.

### Indications

It goes without saying that successful fragment reattachment is dependent upon fragment retrieval at the time of injury. As in the correct treatment of tooth avulsions, fragment retrieval requires an informed public. In Scandinavia, public

**Fig. 13.13 Reattaching a crown fragment with a dentin bonding agent and reinforcement of the bonding site with composite resin**

This 10-year-old boy has fractured his central incisor after a fall from his skateboard. The fracture is very close to the mesial pulp horn.

**Testing pulpal sensibility**

Pulpal response to sensibility testing is normal. The radiographic examination shows no displacement or root fracture.

**Testing the fit of the fragment**

The fragment fits exactly. The enamel surface is intact, with no apparent defect at the enamel margins.

**Temporary dentin coverage with calcium hydroxide**

Due to the close proximity of the fracture surface to the pulp, a temporary glass ionomer lining is placed on the exposed dentin and enamel prior to placement of the temporary restoration.

**Etching the enamel**
A 2 mm wide zone of enamel around the fracture surface is etched.

**Covering the fracture surface**
The tooth is temporarily restored with a temporary crown and bridge material. To provide greater stability, the restoration may be extended to adjacent teeth.

**Storage of the crown fragment**
The tooth is stored in physiologic saline for 1 month. The patient is given the fragment and is instructed to change the solution once a week to reduce contamination.

**Bonding of the fragment after 1 month**
The temporary cover is removed and the fragment is fastened to a piece of sticky wax for ease of handling.

**Preparing for bonding**
Pulpal sensibility is monitored and the fracture surfaces (of the tooth and crown fragment) are cleansed with a pumice-water slurry and a rubber cup.

**Etching enamel**
Enamel on both fracture surfaces as well as a 2 mm wide collar of enamel cervical and incisal to the fracture are etched for 30 seconds with 35% phosphoric acid being sure that the etchant does not come in contact with dentin.

**Removal of the etchant**
The fracture surfaces are rinsed thoroughly with a copious flow of water for 20 seconds.

**Drying the fracture surfaces**
The fracture surfaces are air-dried for 10 seconds. NOTE: To avoid entrapment of air in the dentinal tubules and a subsequent chalky mat discoloration of the fragment, the air stream must be directed parallel with the fracture surface and not perpendicular to it.

**Conditioning the dentin with EDTA and GLUMA®**
The fracture surfaces are conditioned with EDTA for 20 seconds, followed by 10 seconds water rinse and 10 seconds air drying. Thereafter 20 seconds GLUMA® and 10 seconds air drying.

**Bonding the fragment**
The fracture surfaces are covered with a creamy mixture of a filled composite and its unfilled resin. After repositioning of the fragment, the composite is light polymerized.

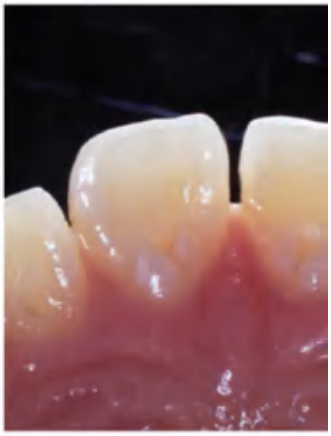
**Light polymerization**
The composite is light polymerized 60 seconds facially and 60 seconds orally.

**Removal of surplus composite**
With a straight scalpel blade or composite finishing knives surplus composite is removed from the fracture site. The interproximal contacts are finished with finishing strips.

**Reinforcing the labial aspect of the fracture site**
A round diamond bur is used to create a 'double chamfer' margin 1 mm coronally and apically to the fracture line. To achieve optimal esthetics, the chamfer follows an undulating path along the fracture line.

**Finishing the labial surface**
After restoring the labial aspect with composite, the restoration is contoured using abrasive disks.

**Reinforcing the palatal aspect of the fracture**
The palatal aspect of the fracture is reinforced using the same procedure. Due to its position, esthetic consideration is less. The preparation can, therefore, follow the fracture line exactly.



**Final restoration**
The condition 1 month after reattachment of the crown fragment.

**Fig. 13.14** Esthetic problems following crown fragment reattachment due to discoloration of the composite bonding material at the fracture line. A. Appearance at the time of fragment bonding of the maxillary left central incisor. B. Appearance 1 year later. There is discoloration of the bonding material.

information campaigns in the form of television spots and posters have proved successful in informing patients of their role in treatment success.

An intact enamel-dentin fragment is the sole indication for reattachment. That is, the majority of the enamel margin should be present so that the fragment can rest firmly against the fracture surface when it is tried against the fractured tooth. Small defects, however, can be restored with composite resin at the time of, or following, the bonding procedure. Moreover, if the fragment is in two pieces, these fragments can be bonded together prior to bonding the final fragment (Fig. 13.13).

## Treatment strategy

In 1994, at the time of publication of the third edition of this book, treatment strategy for fragment reattachment was defined according to eventual pulpal involvement and thickness of the remaining dentin in the case of no pulpal involvement. However, since then, a clinical study has been published which indicates that enamel-dentin tooth fragments can be reattached without a hazard to pulpal vitality at the time of injury (213). Immediate fragment reattachment has several advantages: short treatment time (often compared to traditional temporary/emergency measures), an immediate hermetic seal of dentinal tubules, and immediate restoration of function and esthetics. The bonding procedure is illustrated in Fig. 13.13.

However, there can be situations that are not conducive to this approach, for instance with an uncompliant patient, where some time should be left between the traumatic event and and definitive treatment, or with a concomitant luxation injury, where there may be difficulties in maintaining a dry operating field.

If provisional treatment is the chosen strategy and there is an uncomplicated crown fracture, the entire fracture surface (enamel and dentin) can be covered with calcium hydroxide liner and glass ionomer cement. These are placed for approximately 1 month to permit hard tissue deposition in the pulp horn. Provisional restorations can be incorporated in the necessary splints, and bonding can be performed at the time of splint removal. In the interim period, the crown fragment is kept moist, for example in tap water or physiologic saline, which is changed weekly. If the coronal fragment has been allowed to desiccate prior to bonding, the fragment will whiten; *in vitro* tests have shown a decreased bonding strength of such a fragment (275, 276). In these cases, wet storage for at least 24 hours can normalize the situation (274).

The treatment strategy for crown fractures with pulpal involvement includes pulp capping or partial pulpotomy (see Chapter 25). Direct bonding of the fragment can be performed after pulpotomy.

## Clinical results of fragment reattachment

With respect to the long-term success of fragment reattachment, factors such as pulpal response, esthetics and retention of the fragment should be considered.

## Pulpal response to fragment reattachment

The reattachment of enamel-dentin crown fragments has not been found to lead to pulpal complications in a large long-term study (211). The few cases of pulp necrosis and pulp canal obliteration were all found in relation to concomitant luxation injuries. This low complication rate is more likely a response to the injury itself than to the treatment procedure (259).

## Esthetics following fragment reattachment

Approximately half of the teeth bonded demonstrate acceptable esthetics at long-term follow-up (211). Esthetic problems at the follow-up include discoloration or degradation of the composite bonding material at the fracture line (Fig. 13.14) or discoloration of the incisal fragment with time. The problem of discoloration of the fracture line is most pronounced in earlier cases of bonding, due to discoloration of the catalyst system of the chemically cured resins used. This problem has been solved in part by the use of light-cured composite materials. Esthetics has also been enhanced by the use of a double chamfer preparation along the fracture line after fragment bonding and restoration with a composite resin (Fig. 13.13).

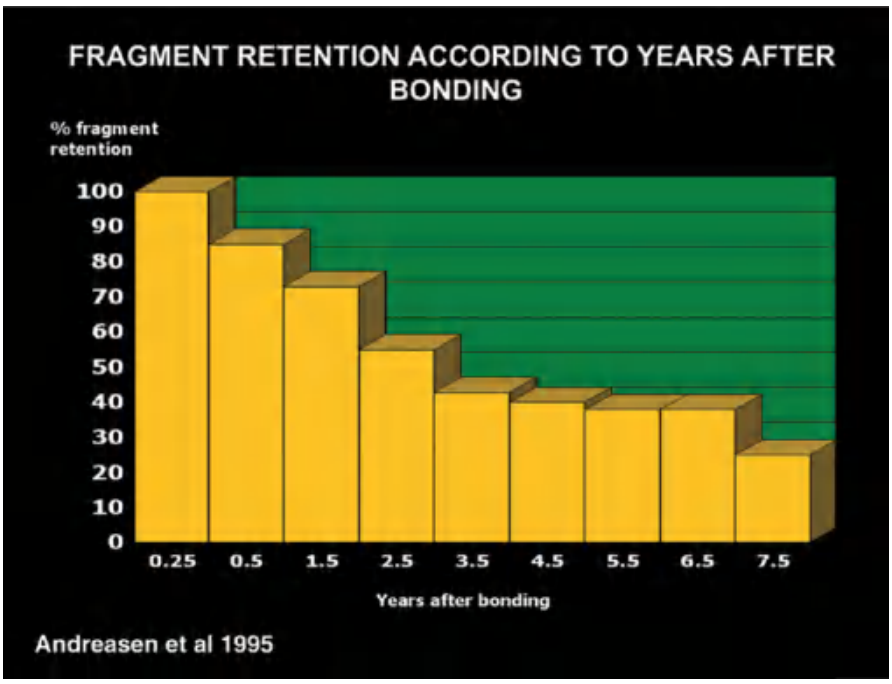



**Fig. 13.15** Survival of fragment bonding. From (213).

The problem of fragment discoloration, usually to a mat white color, is presumably due to dehydration of the underlying dentin (211). This is more difficult to manage clinically, as direct composite facings tend not to mask the color disharmony between the tooth and bonded fragment.

### Fragment retention

Results from a large Scandinavian study indicate that 60% of the bonded fragments were lost after 5 years due primarily to a new trauma or non-physiologic use of the restored teeth (213) (Fig. 13.15). Fragment debonding occurred irrespective of the use of enamel acid etch used alone or supplemented with dentin bonding agent; debonding was also not related to the use of composite resin along the fracture line for esthetic revision and reinforcement of the restoration. While fragment debonding represents a practical inconvenience, it has no impact on pulpal vitality since debonding occurs as a cohesive failure within the bonding resin and not in the dentin. Thus, the fracture surfaces of teeth with debonded fragments are glossy from retained resin plugs within the treated dentin. For this reason, the dentin must be freed of bonded resin (e.g. with a slurry of pure pumice and water) prior to rebonding of fragments.

Several *in vitro* studies have analyzed different techniques and their influence on bonding strength, including enamel beveling, a V-shaped enamel groove, internal dentin groove, external chamfer, overcontour and simple rebonding (296). However, there are presently no controlled clinical studies comparing these techniques.

It would seem that at present reattachment of the original incisal fragment can only be considered as an acceptable semipermanent anterior restoration if gross and surface anatomy are restored perfectly with a material that abrades at a rate identical to adjacent teeth and which does not threaten pulpal vitality (212, 260, 261).

Fragment reattachment or restoration with a composite build-up is a realistic treatment alternative in the young (preteen and teenage) patient groups. These treatments serve to postpone the time of definitive treatment until an age when the gingival marginal contours are relatively stable (i.e. 20 years of age).

## Prognosis of crown fractures

### Permanent teeth – follow-up after trauma

Pulpal sensibility reactions in crown-fractured teeth are often lowered immediately after injury (7, 66, 151, 156). Usually 1–8 weeks need to elapse before a normal pulpal response can be elicited. However, longer observation periods may be required. Pulp testing can usually be carried out during this observation period without removing the temporary restoration (see Chapter 9).

In the following, the risk of pulp necrosis is presented for the various types of fracture. It appears that the most important factor is an associated luxation injury (Figs 13.16 and 13.17).

### Enamel infractions

The prognosis of isolated infractions with respect to risk of pulp necrosis appears to be very good (Table 13.1). Thus, three studies have shown risks of between 0% and 3.5%, which is about the same as for 'non-injured control teeth' (154, 156–158). This implies that the few cases with pulp necrosis possibly reflect overlooked concussion or subluxation injuries, or a pathway for bacterial invasion following extrusion, lateral luxation or intrusive luxation (158, 293).

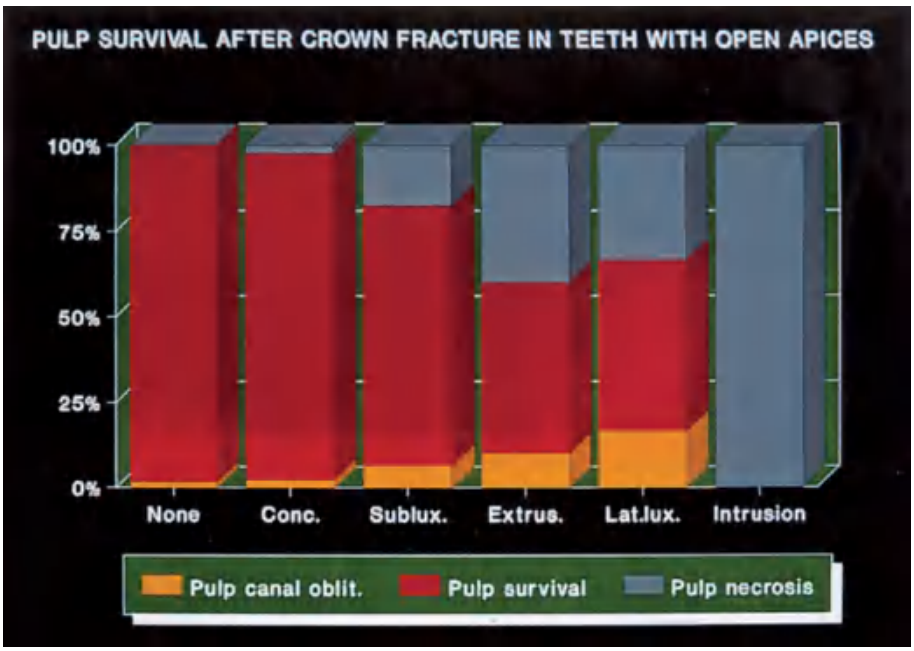



**Fig. 13.16** Pulp survival following crown fracture in the permanent dentition with immature root formation (open apices) in relation to concomitant luxation injuries (290, 292–294, 297).

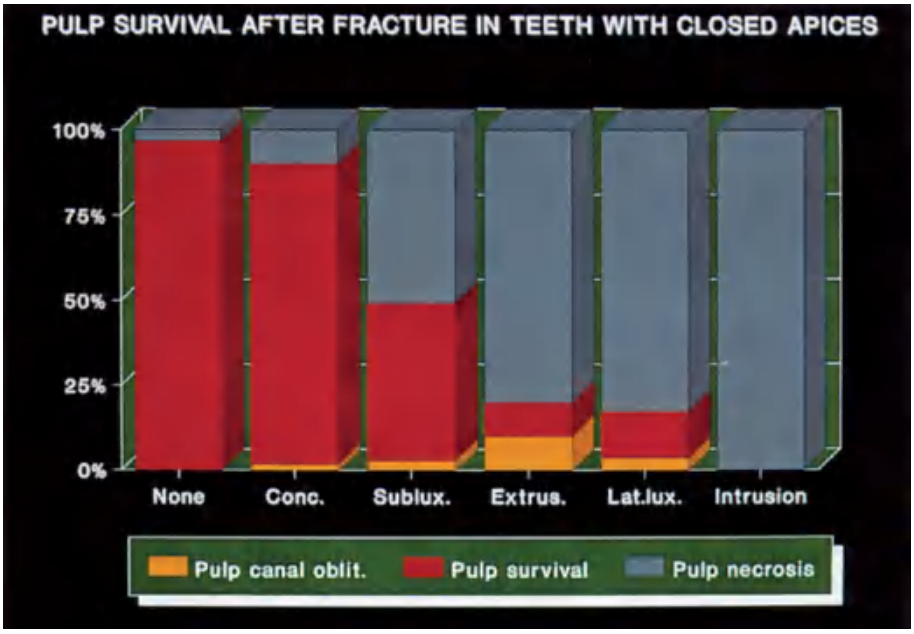



**Fig. 13.17** Pulp survival following crown fracture in the permanent dentition with mature root formation (closed apices) in relation to concomitant luxation injuries (290, 292–294, 297).

**Table 13.1** Prevalence of pulp necrosis after crown infraction.

| Study | No. of teeth | Pulp necrosis |
|---|---|---|
| Stålhane & Hedegård 1975 (99) | 656 | 23 (3.5%) |
| Ravn 1981 (155) | 174 | 0 (0%) |
| Robertson 1998 (286) | 16 | 0 (0%) |

**Table 13.2** Prevalence of pulp necrosis after enamel fracture.

| Study | No. of teeth | Pulp necrosis |
|---|---|---|
| Fialova & Jurecek 1968 (62) | 31 | 0 (0%) |
| Stålhane & Hedegård 1975 (99) | 876 | 2 (0.2%) |
| Ravn 1981 (156) | 2862 | 29 (1%) |
| Robertson 1998 (286) | 46 | 0 (0%) |

## Enamel fractures

In two sets of studies, the risk of pulp necrosis was found to range from 0.2% to 1.0% (Table 13.2, Figs 13.16 and 13.17). Using the above-mentioned rationale, these figures would imply virtually no risk to the pulp.

## Enamel-dentin fractures without pulpal involvement

The overall risk of pulp necrosis irrespective of the extent of fracture and type of treatment ranges from 1% to 6%

**Table 13.3** Prevalence of pulp necrosis after crown fracture with exposed dentin.

| Study | No. of teeth | Pulp necrosis |
|---|---|---|
| Stålhane & Hedegård 1975 (99) | 413 | 3 (1%) |
| Zadik et al. 1979 (102) | 123 | 7 (6%) |
| Ravn 1981 (157) | 3144 | 250 (6%) |
| Robertson 1998 (286) | 60 | 2 (2%) |
| Robertson et al. 2000 (287) | 106 | 0 (0%) |

**Table 13.4** Relation between associated luxation injuries and enamel-dentin fractures on the frequency of pulp necrosis. Reproduced from (157) with permission.

| | No. of teeth | Pulp necrosis |
|---|---|---|
| Enamel-dentin fracture | 3144 | 100 (3%) |
| Enamel-dentin fracture + concussion | 327 | 19 (6%) |
| Enamel-dentin fracture + subluxation | 423 | 106 (25%) |

**Table 13.5** Risk of pulp necrosis estimated after 3 years of observation (Kaplan Meier method) (292–294, 297).

| Immature root development | No of teeth | Risk of pulp necrosis, *n* and % |
|---|---|---|
| Concussion + crown fracture without pulp exposure | 107 | 1 (0.9%) |
| Subluxation + crown fracture without pulp exposure | 75 | 15 (21.4%) |
| Extrusion + crown fracture without pulp exposure | 8 | 0 (0%) |
| Lateral luxation + crown fracture without pulp exposure | 7 | 3 (42.9%) |
| Intrusion + crown fracture without pulp exposure | 34 | 24 (70.9%) |
| **Mature root development** | **No of teeth** | **Risk of pulp necrosis, *n* and %** |
| Concussion + crown fracture without pulp exposure | 137 | 15 (11%) |
| Subluxation + crown fracture without pulp exposure | 62 | 24 (42.3%) |
| Extrusion + crown fracture without pulp exposure | 17 | 13 (76.5%) |
| Lateral luxation + crown fracture without pulp exposure | 28 | 26 (92.9%) |
| Intrusion + crown fracture without pulp exposure | 68 | 66 (98.5%) |

(Table 13.3, Figs 13.16 and 13.17). It should be noted that factors such as associated luxation injuries, stage of root development, type of treatment and extent of fracture exert significant influence upon the risk of pulp necrosis. A detailed study on the combined effects of these factors has been reported by Ravn (157).

**Table 13.6** Relation between extent of enamel-dentin fracture and pulp necrosis. Reproduced from (157) with permission.

| | No. of teeth | No. with pulp necrosis |
|---|---|---|
| Small mesial fracture | 1034 | 26 (0.2%) |
| Comprehensive mesial fracture | 435 | 30 (7%) |
| Fracture involving the entire incisal edge | 1019 | 38 (0.4%) |

**Table 13.7** Relation between treatment and the frequency of pulp necrosis in teeth with extensive mesial or distal enamel-dentin fractures. Reproduced from (157) with permission.

| | No. of teeth | No. with pulp necrosis |
|---|---|---|
| No treatment | 24 | 13 (54%) |
| Dentin coverage | 620 | 30 (8%) |

With respect to the effect of *stage of root development* on the risk of pulp necrosis after uncomplicated crown fracture with or without concomitant luxation injury, it has been found that teeth with constricted apices have a significantly greater risk of pulp necrosis than do teeth with open apices (261, 287, 290). Furthermore that the extent of periodontal ligament injury as revealed by the *luxation diagnosis* is significantly related to pulp survival after injury (287, 292–295, 297) (Tables 13.4 and 13.5, Figs 13.16 and 13.17).

Another factor to be considered is the *extent* and *location* of the fracture. Thus, horizontal and proximal superficial (corner) fractures demonstrate a very low frequency of pulp necrosis; whereas deep proximal fractures show increased risk (Table 13.6).

Only a single study has examined the *effect of treatment* (i.e. untreated versus treated crown fractures). A treatment effect was only found for deep proximal fractures (Table 13.7). In this connection, *no treatment* was found to be associated with 54% pulp necrosis; whereas *dentin coverage* resulted in a decrease to 8% (157).

The effect of time interval between injury and dentin coverage and subsequent risk of pulp necrosis has yet to be examined (288). A certain relationship has been claimed in one study but associated luxation injuries were not monitored (157).

In Fig. 13.18 the predictors for pulp necrosis are shown.

## Pulp canal obliteration

In one study, pulp canal obliteration (PCO) was analyzed subsequent to crown fractures without associated luxation injury (99) (Table 13.8). The paucity of PCO cases possibly implies non-diagnosed luxation injuries. It seems safe to assume that a crown fracture per se does not elicit PCO.

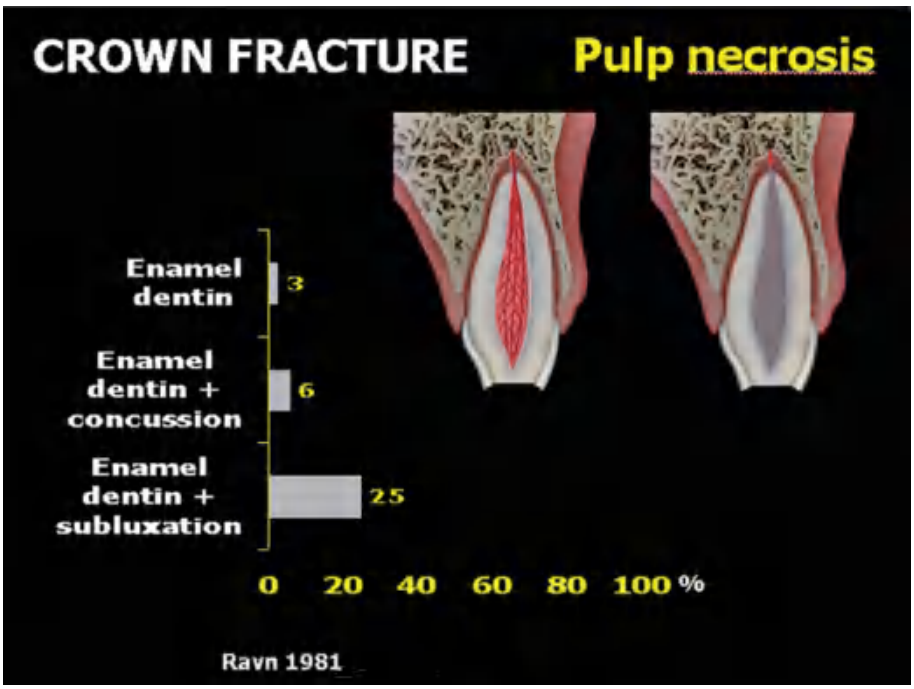



**Fig. 13.18** Predictors for pulp necrosis after crown fracture. From (155–157).

**Table 13.8** Prevalence of pulp canal obliteration and root resorption after crown fracture. From (99).

| | No. of teeth | No. with pulp canal obliteration | No. with external root resorption |
|---|---|---|---|
| Enamel infraction | 174 | 0 (0%) | 0 (0%) |
| Enamel fracture | 876 | 4 (0.5%) | 2 (0.2%) |
| Enamel and dentin fracture | 413 | 2 0.5% | 1 (0.2%) |

### Root resorption

In one study, where only crown fractures without associated luxation injuries were examined, root resorption was a rare finding (99) (Table 13.8). Thus, like PCO, root resorption is probably not related to crown fractures per se.

### Reattachment of the crown fragment

With respect to pulp survival, fragment retention and esthetics, preliminary results from a long-term clinical study (212, 213) indicate that pulp survival without radiographic change was the predominant finding. Only one out of 198 bonded incisors (0.5%) developed pulp necrosis and four developed PCO (2%). All five teeth had suffered concomitant luxation injuries (212, 213).

## Essentials

### Terminology (Fig. 13.1)

- Enamel infraction
- Enamel fracture
- Enamel-dentin fracture
- Enamel-dentin fracture with pulp exposure

### Frequency

- Permanent dentition: 26–76% of dental injuries

### Etiology

- Usually falls with direct impact on the crown

### History

- Symptoms

### Clinical examination

- Extent of fracture
- Pulp exposure
- Dislocation of tooth
- Reaction to sensibility tests

### Radiographic examination

- Size of pulp cavity
- Stage of root development
- Concomitant root fracture or luxation

## Pathology

- If the pulp is exposed, pulp inflammation is normally confined close to the exposure site

## Enamel infraction

### *Treatment*

- Pulpal sensibility control after 6–8 weeks
- Eventual sealing of infraction lines with resin

### *Prognosis*

- Pulp necrosis (0–3.5%)

## Enamel fracture (no dentin exposed)

### *Treatment*

- Removal of sharp enamel edges
- Corrective grinding (Fig. 13.10) or restoration with a composite resin
- Radiographic and sensibility controls

### *Prognosis*

- Pulp necrosis (0–1.0%)

## Enamel-dentin fracture, no pulp exposure

### *Immediate (provisional) treatment (Fig. 13.12)*

- Place glass ionomer cement over the exposed dentin and enamel to permit optimal bonding of the restorative material at the time of definitive therapy (Fig. 13.12)
- Alternatively adapt a temporary crown (e.g. acrylic crown). Crown cemented with a non-eugenol containing luting agent
- Check the occlusion
- Control the tooth radiographically and with sensibility tests after 6–8 weeks

### *Permanent treatment*

- Reattachment of the crown fragment (Fig. 13.13)
- Restoration with laminate veneer (see Chapter 30)
- Restoration with composite resin (see Chapter 29)
- Restoration with full crown coverage

### *Prognosis*

- Pulp necrosis (0–6%)

## Enamel-dentin fracture with pulp exposure

### *Immediate treatment*

- Exposed pulp tissue is treated by pulp capping, pulpotomy or pulpectomy (see Chapter 25)

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# 14

# Crown-Root Fractures

J. O. Andreasen, F. M. Andreasen, M. Tsukiboshi & F. Eichelsbacher

## Terminology, frequency and etiology

A crown-root fracture is defined as a fracture involving enamel, dentin and cementum. The fractures may be grouped according to pulpal involvement into *uncomplicated* and *complicated*.

In some studies this type of injury is not recognized as a special entity and is, therefore, classified as either a crown or a root fracture. In the authors' material, however, crown-root fractures comprised 5% of injuries affecting the permanent dentition and 2% in the primary dentition (1).

The most common etiologic factors are injuries caused by falls, bicycle and automobile accidents and foreign bodies striking the teeth (1).

Crown-root fractures in the anterior region are usually caused by *direct trauma* (see Chapter 11). The direction of the impacting force determines the type of fracture. A frontal blow results in the typical fracture line shown in Fig. 14.1. In the posterior regions, fractures of the buccal or oral cusps of premolars and molars may occur (2, 12). These fractures extend below the gingival attachment, often without pulp exposure (uncomplicated) (Fig. 14.2). The causes of such injuries are often *indirect trauma* (see Chapter 11).

Although not caused by accidental trauma, for the sake of completeness, it should be mentioned that crown-root fractures can also have an iatrogenic etiology, such as longitudinal crown-root fractures, especially in the premolar and molar regions, caused by lateral pressure during root filling procedures, cementation of posts, corrosion of posts or improperly designed restorations (13–24). The clinical and radiographic signs of these fractures have been previously described (41).

## Clinical findings

Most commonly, the fracture line begins a few millimeters incisal to the marginal gingiva facially and follows an oblique course below the gingival crevice orally. The fragments are usually only slightly displaced, the coronal fragment being kept in position by fibers of the periodontal ligament orally and/or the dental pulp (Fig. 14.3). Displacement of the coronal fragment is often minimal, which explains why these fractures are frequently overlooked, particularly in the posterior regions (Fig. 14.3).

In rare cases, a crown-root fracture may occur prior to eruption of the permanent tooth due to a trauma transmitted by displacement of a primary incisor (42).

The fracture line is usually *single*, but *multiple* fractures can occasionally be seen (Fig. 14.4A). A rare type of injury is a vertical fracture running along the long axis of the tooth (25) (Fig. 14.4B), or deviating in a mesial or distal direction.

Crown-root fractures of anterior teeth often expose the pulp in fully erupted teeth, while fractures of teeth in earlier stages of eruption can be uncomplicated.

Even with pulp exposure, symptoms are normally few, and are usually limited to slight pain due to mobility of the crown fragment during function.

## Radiographic findings

Radiographic examination of crown-root fractures following the usual course seldom contributes to the clinical diagnosis, as the oblique fracture line is almost perpendicular to the central beam (Fig. 14.5). Radiographic determination of the oral limit of the fracture is frequently unsuccessful due to

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A B

**Fig. 14.1** Complicated crown-root fracture of a right central incisor due to direct trauma. A. Clinical condition. B. Lateral view of crown portion after removal. Note extension below gingival crevice palatally.

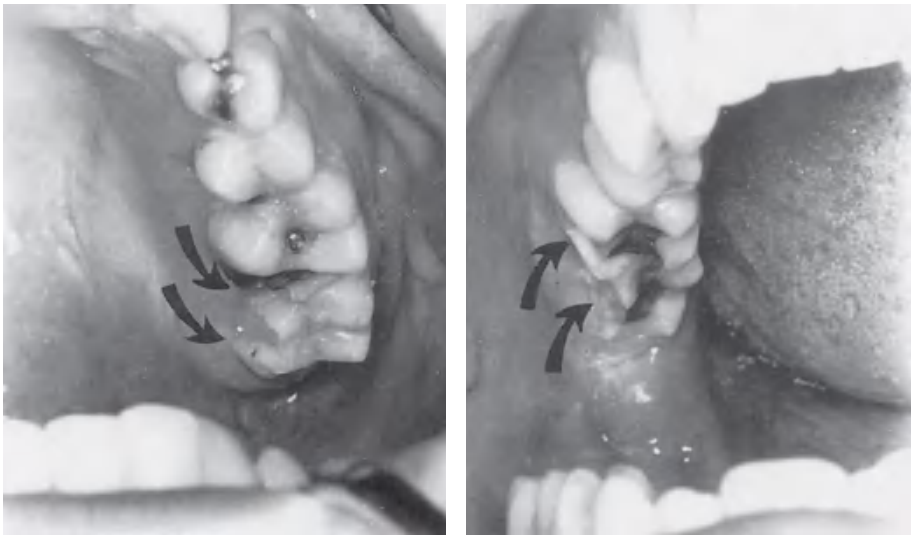
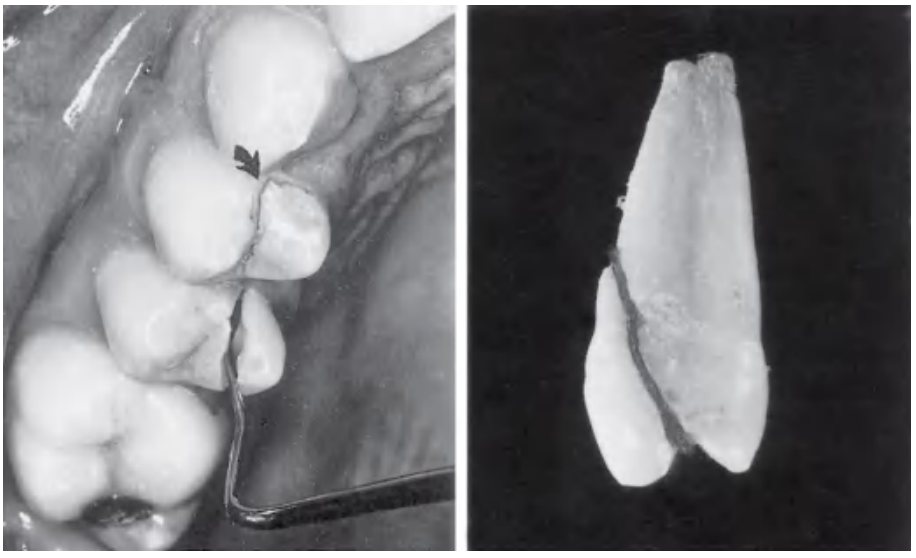


**Fig. 14.2** Uncomplicated crown-root fractures involving palatal cusps in the maxilla and buccal cusps in the mandible (arrows) due to forceful occlusion from a blow to the chin. Note that the fractures of the lingual cusps in the maxilla correspond to the fractures of the buccal cusps in the mandible.



A B

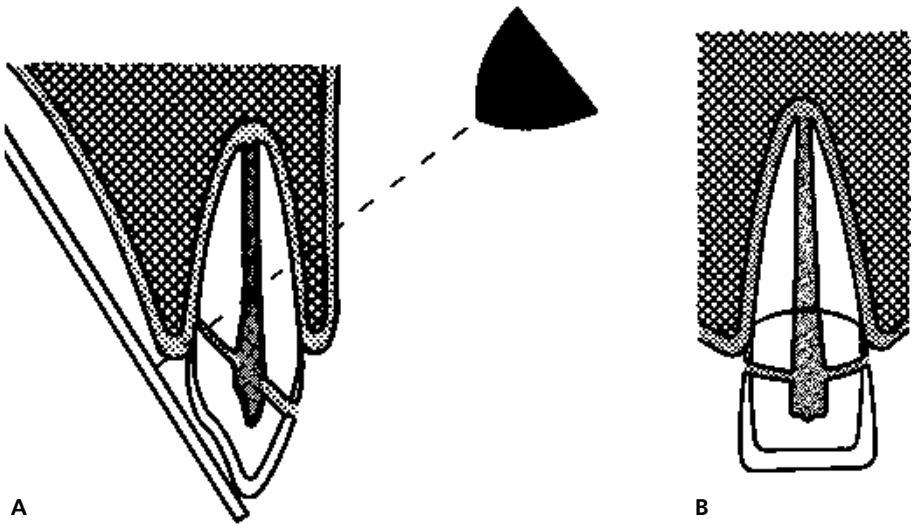
**Fig. 14.3** Complicated crown-root fractures of the right first and second premolars (arrow) following indirect trauma (blow to the chin). A. Minimal displacement of fragments in the first premolar. B. Lateral view of the extracted tooth.

the close proximity of fragments at this level. On the other hand, the facial limit is always visible (Fig. 14.6).

Vertical fractures are easily demonstrated if oriented in a facio-oral direction. This also applies to superficial vertical fractures deviating in a mesial or distal direction (chisel fractures) (Fig. 14.7). However, vertical root fractures running in a mesiodistal direction can seldom be seen radiographically (see Fig. 14.10, below).

A B

**Fig. 14.4** A. Multiple complicated crown-root fractures of a left central incisor. B. Complicated crown-root fracture of a right central incisor running along the long axis of the tooth.

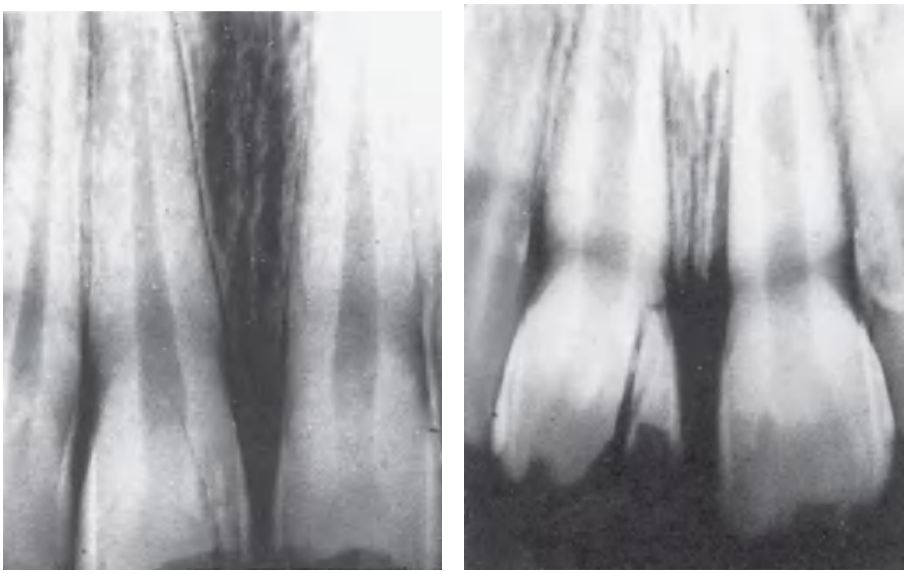


A B

**Fig. 14.5** Schematic drawings of radiographic orientation in a complicated crown-root fracture. A. The normal projection angle is almost perpendicular to the fracture surface. B. Radiographic determination of the lingual part of the fracture is obscured due to the perpendicular relationship between the fracture line and central X-ray beam.

A B

**Fig. 14.6** A. Crown-root fractures involving both central incisors. B. In contrast to the lingual aspect of the fractures, the facial aspect is clearly visible.



A B

**Fig. 14.7** Radiographic demonstration of longitudinal crown-root fractures. A. Vertical complicated longitudinal crown-root fracture affecting a right central incisor. B. Uncomplicated crown-root fracture located in the mesial part of a right central incisor (chisel fracture).

## Healing and pathology

Communication with the oral cavity to the pulp and periodontal ligament in these fractures permits bacterial invasion and subsequent inflammation (3–5). For this reason fracture healing cannot be expected in crown-root fractures, in contrast to root fractures where the fracture is located entirely within the alvelous.

Early histologic changes consist of acute pulpal inflammation located close to the fracture caused by invasion of bacteria (56) (Fig. 14.8). Later, proliferation of marginal gingival epithelium into the pulpal chamber can be seen (Figs 14.9 and 14.10). Repair of the fracture by deposition of osteodentin along the fracture line is exceptionally rare (5–7) (Fig. 14.11) and should not govern a treatment decision. Instead the coronal fragment is usually removed and the treatment should be focused on the possibility of using the remaining fragment.

In a recent histologic study in dogs, the healing events were compared between surgical and orthodontic extrusion. It appeared that surgical extrusion inflicted more repair-related resorption than orthodontic extrusion; however, after 6 months the resorption cavities showed healing (69).

## Treatment

Treatment of crown-root-fractured teeth is a very controversial issue even among specialists in restorative dentistry (70).

In the *emergency treatment* of crown-root fractures in the *anterior region* it is possible to stabilize of the coronal fragment with an acid-etch/resin splint to adjacent teeth (Fig. 14.12) as a temporary measure. Despite contamination from saliva along the fracture line to the pulp, the tooth will generally remain without symptoms. However, it is essential that definitive treatment is initiated within a few days after injury. In cases of multiple, uncomplicated crown-root fractures in the *premolar and molar region*, immediate provisional treatment can include removal of loose fragments and coverage of exposed supragingival dentin with a glass ionomer cement.

*Vertical crown-root fractures* must generally be extracted. However, it should be mentioned that cases have been reported where bonding of the coronal fragment has led to consolidation of the intra-alveolar part of the fracture (44, 45). The tissue involved in this type of healing has not yet been established.

In cases where conservative treatment is not considered possible because the fracture extends too deeply below the gingival margin, or there is pulpal pathology, it is worth considering the option of keeping the apical fragment. This may prevent the alveolar process from resorbing (77) (see also Chapter 26)

Finally, it should be mentioned that in the case of vertical fractures of *immature permanent incisors*, the fractures are usually incomplete, stopping at or slightly apical to the level of the alveolar crest. These fractures are amenable to orthodontic extrusion, whereby the level of the fracture is brought to a level where pulp capping and restoration are possible (see Chapter 28).

Definitive conservative therapy in the *permanent dentition* comprises one of four treatment alternatives. The choice is primarily determined by exact information on the site and type of fracture, but cost and complexity of treatment can also be deciding factors (Table 14.1). In the following, conservative treatment of various types of crown-root fractures will be described.

## Bonding of coronal fragment

### Treatment principle

The principle is to remove and reattach the fragment with a bonding technique.

This new technique has been documented in case reports (71, 72) and in a larger cohort study (73). Due to the development of reliable resin composites and bonding systems,

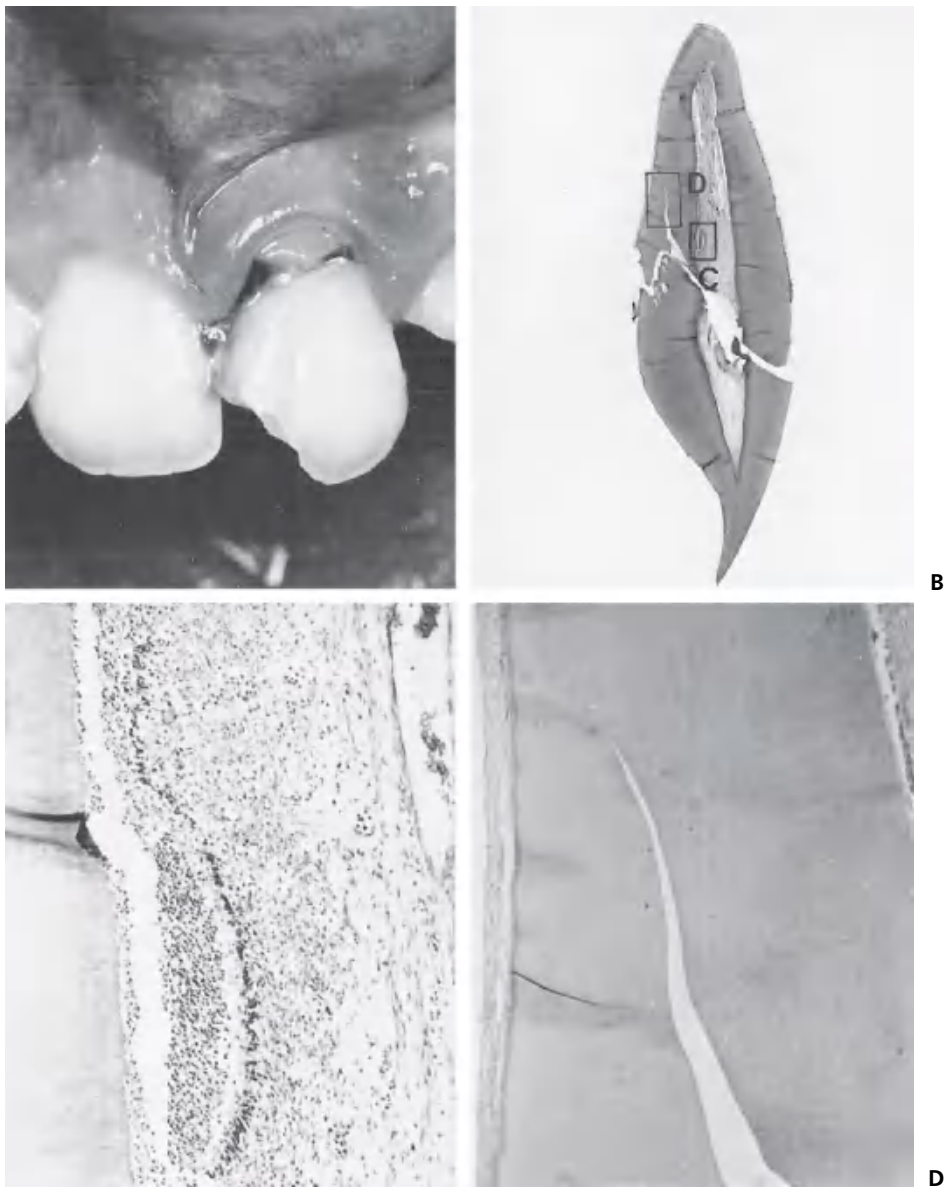



**Fig. 14.8** Early histologic reactions following a complicated crown-root fracture of a left central incisor. A. Clinical condition. The tooth was extracted 4 days after injury. B. Low power view of sectioned incisor. Interruption of the pulp tissue at the fracture site is an artifact. ×3. C. A marked acute inflammation is present close to the fracture site, while only slight inflammation is found more apically. ×75. D. Incomplete fracture extending halfway through the dentin. ×20.

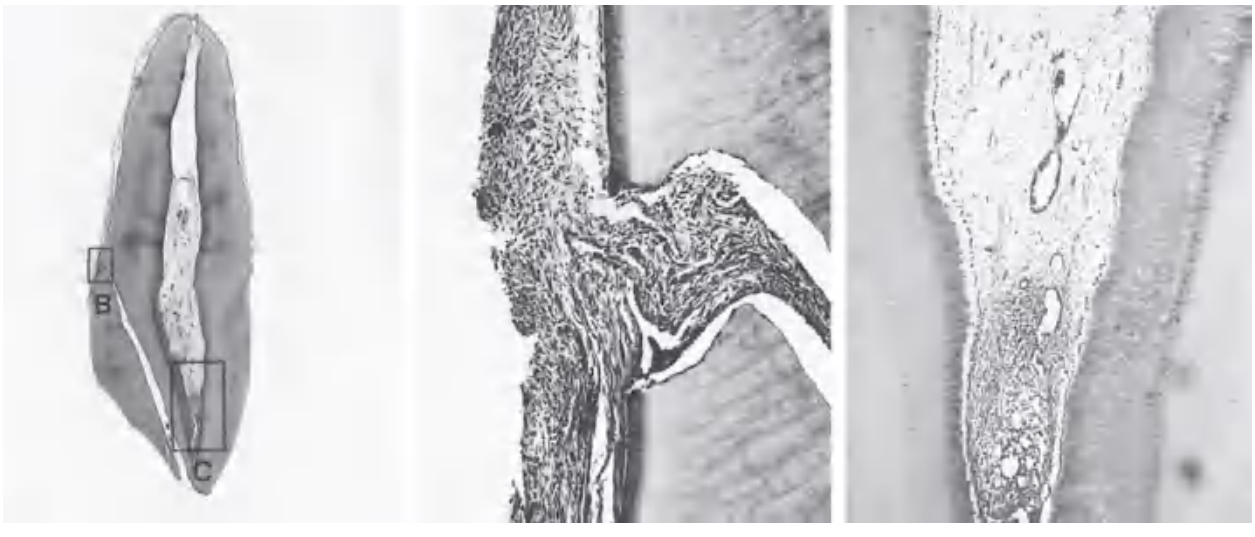



**Fig. 14.9** Late histologic reactions following an uncomplicated crown-root fracture of a right central incisor. The tooth was left untreated and extracted 3 weeks after injury. A. Low power view of sectioned incisor. ×3. B. Proliferation of connective tissue into the fracture line. ×75. C. Marked inflammation in the coronal part of the pulp. ×30.

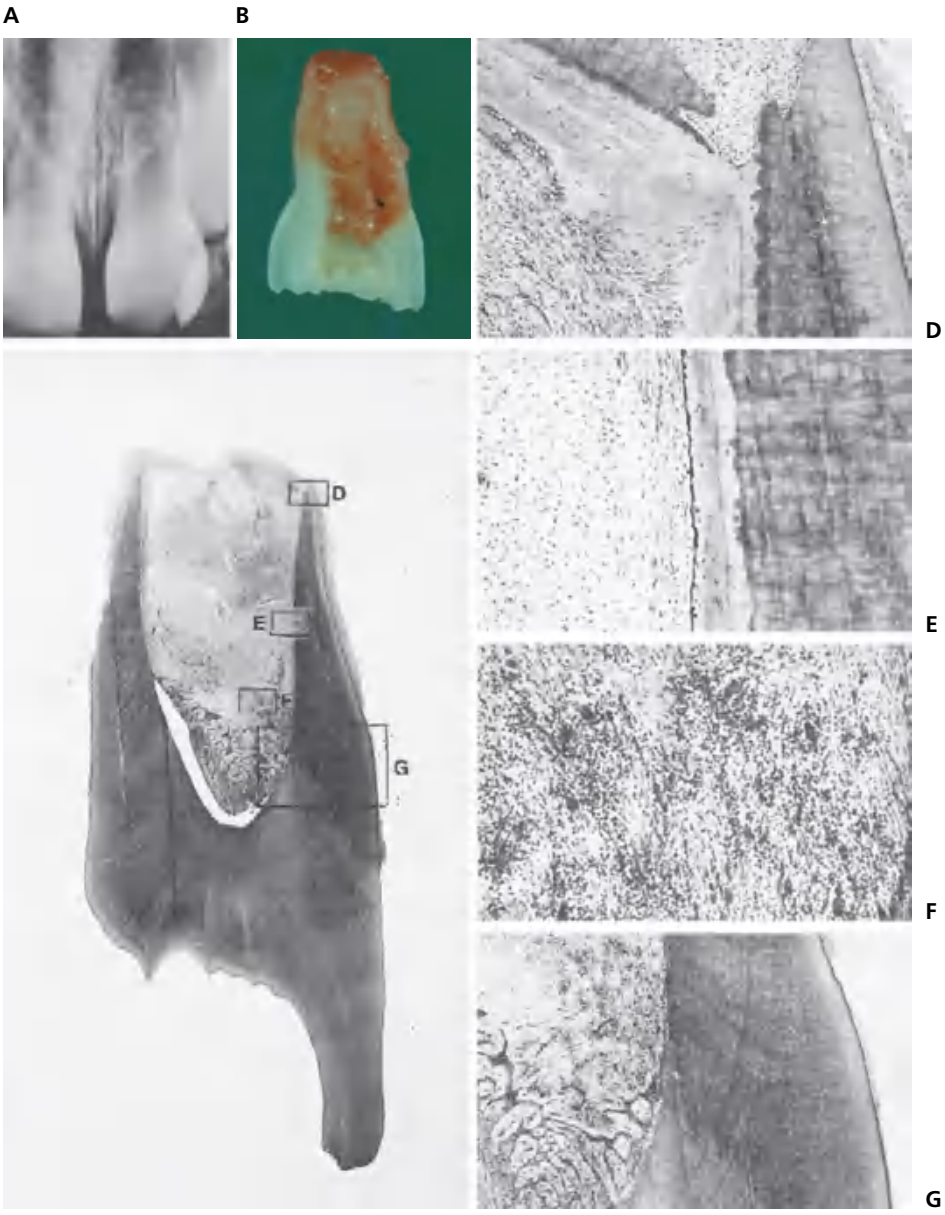



**Fig. 14.10** Late histologic reactions following a complicated crown-root fracture of a left central incisor. The tooth was traumatized 2 months earlier. A. Radiograph taken at the time of extraction. The fracture line is not seen on the radiograph due its mesiodistal orientation. B. Lingual fragment. C. Low power view of sectioned tooth. ×8. D. Apex of the root is fractured and forced into the pulp. Note pulpal repair with new dentin. ×75. E. Deposition of cellular dentin. ×75. F. Inflammatory changes in the coronal part of the pulp. ×75. G. Proliferation from the cuff epithelium. ×30. From (4).

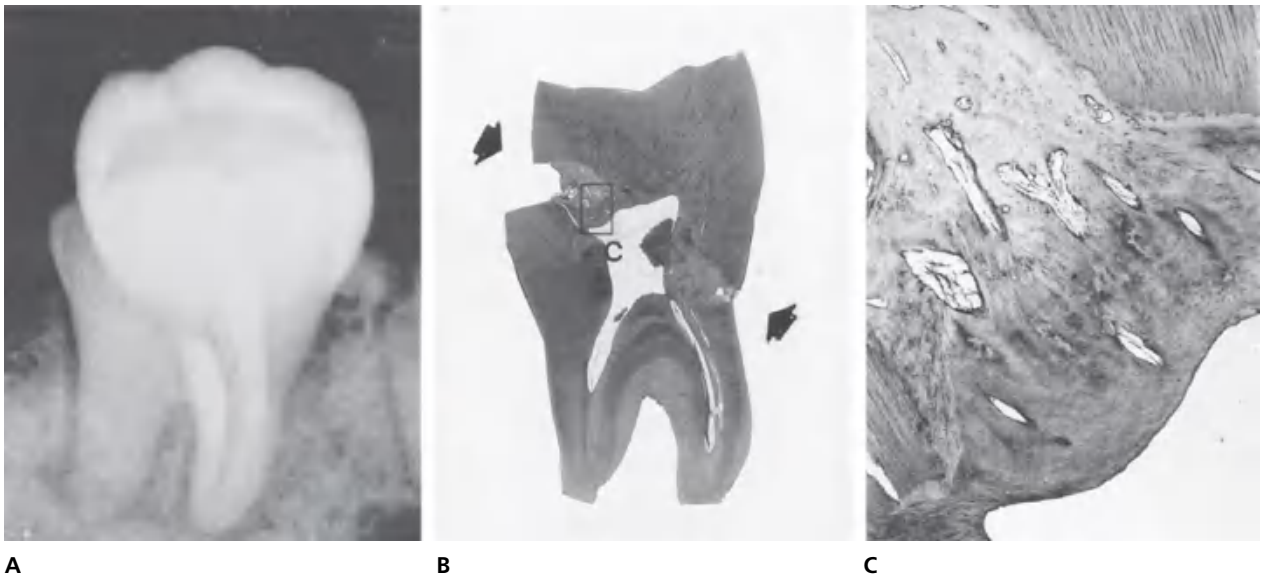



**Fig. 14.11** Healing of a crown-root fracture with calcified tissue. A. Radiograph of a right second molar taken 10 months after the crown-root fracture. The tooth was immobilized after the injury. Fourteen months after injury, the tooth was extracted for prosthetic reasons. B and C. Note that the fracture communicating with the oral cavity is united by new dentin with vascular inclusions. C ×30. Reproduced from (7) with permission.

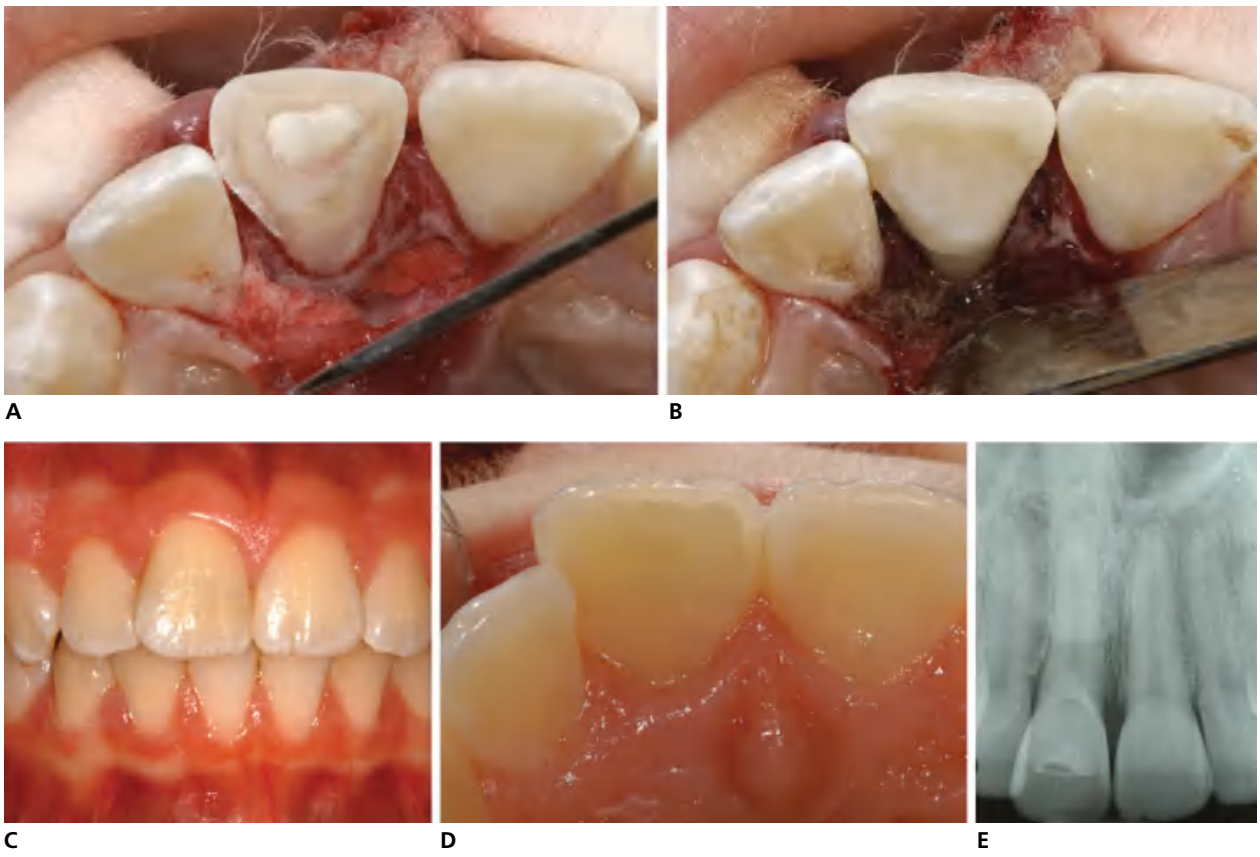



**Fig. 14.12** Bonding of a coronal fragment. A and B. The fragment is removed and a palatal flap is raised. Hemostasis is secured by cotton pellets impregnated with adrenalin. After etching with phosphoric acid, the coronal fragment is bonded with light-cure total etch adhesive and a light-cure flowable composite. B. Status after bonding. C–E. Clinical and radiographic condition after 2.5 years.

**Table 14.1** Comparison between various treatment modalities of crown-root fractures.

| Procedure | Indications | Advantages | Disadvantages |
|---|---|---|---|
| Bonding of coronal fragment | Superficial fractures | Relatively easy procedure | Only a single cohort study (73) |
| Fragment removal and gingivectomy (sometimes ostectomy) | Fractures where denudation of the fracture site does not compromise esthetics (i.e. fractures with palatal extension) | Relatively easy procedure. Restoration can be completed soon after injury | Restored tooth may migrate labially due to accumulation of granulation tissue in the deep palatal pocket (34) |
| Orthodontic extrusion of apical fragment | All types of fractures, assuming that reasonable root length can be achieved | Stable position of the restored tooth. Optimal gingival health. Can be performed in complicated crown-root fractures | Technically time-consuming procedure, with late completion of final treatment* |
| Surgical extrusion of apical fragment | All types of fractures (except uncomplicated crown-root fractures where vitality should be preserved) assuming that reasonable root length can be achieved | Rapid procedure. Stable position of the tooth. The method allows inspection of the root for additional fractures | Limited risk of root resorption and marginal breakdown of periodontium. Cannot be performed in uncomplicated crown-root fractures where vitality should be preserved |

* Note that cervical gingival fibers must be incised once the tooth is at its desired position to prevent return of the tooth to its original position.

tooth fractures limited to enamel and dentin can now be restored by adhesive reattachment of the coronal fragment mostly without the necessity of additional restorative interventions. A case series with a total of 20 treated teeth revealed that 2 years after therapy the periodontal conditions were healthy in 90% of the subjects, whereas in 10% of the subjects the teeth had suffered fragment loosening again due to a new trauma (73).

### Initial measures

Prior to the definitive reattachment, fragments should remain *in situ* or be splinted with a provisional ligature splint with an interfacing layer of calcium hydroxide between the fragment and the root stub. If provisional splinting of the fragment is not feasible, the surface of the root surface is sealed with a calcium hydroxide cement and subsequently covered with a temporary resin. Coronal fragments that cannot be kept *in situ* should be stored in saline until fragment reattachment.

### Fragment reattachment

The fracture site is exposed under local anesthesia by raising a surgical flap (Fig. 14.12). If the crown fragment can be reattached to the root without bleeding or mechanical obstacles, no ostectomy is needed. Hemostasis is secured by sterile cotton pellets impregnated with adrenalin.

After cleaning the fracture site on the root and the crown fragment, enamel surfaces adjacent to the fracture line are etched using a phosphoric acid gel. Subsequently, the crown fragment is reattached to the root using a filled, light-cure total-etch adhesive and a light-cure flowable composite (Fig. 14.12). Curing is performed by a LED curing light held at the fracture line at different angles. After removing any excess resin using a scalpel blade and oscillating files, the flap is repositioned using non-resorbable sutures. Subsequently, occlusion is checked using occlusion foil and eventual occlusal interferences removed by a fine diamond bur. The patient should refrain from tooth brushing at the surgical site and a chlorhexidine mouth rinse is used. After 10 days sutures are removed.

## Removal of coronal fragment and supragingival restoration

### Treatment principle

This is to allow gingival healing (presumably with formation of a long junctional epithelium), whereafter the coronal portion can be restored (8–11, 26–31, 43) (Fig. 14.13). With respect to restoration, the following procedures can be used: bonding the original tooth fragment, where the subgingival portion of the fragment has been removed, composite build-up using dentin and enamel bonding agents; or full crown coverage (Fig. 14.14).

Pulpal considerations are identical to those that apply to the treatment of crown fractures (see Chapter 13).

## Surgical exposure of fracture surface

### Treatment principle

This is to convert the subgingival fracture to a supragingival fracture with the help of gingivectomy and osteotomy (8–11, 26–31, 43) (Fig. 14.15).

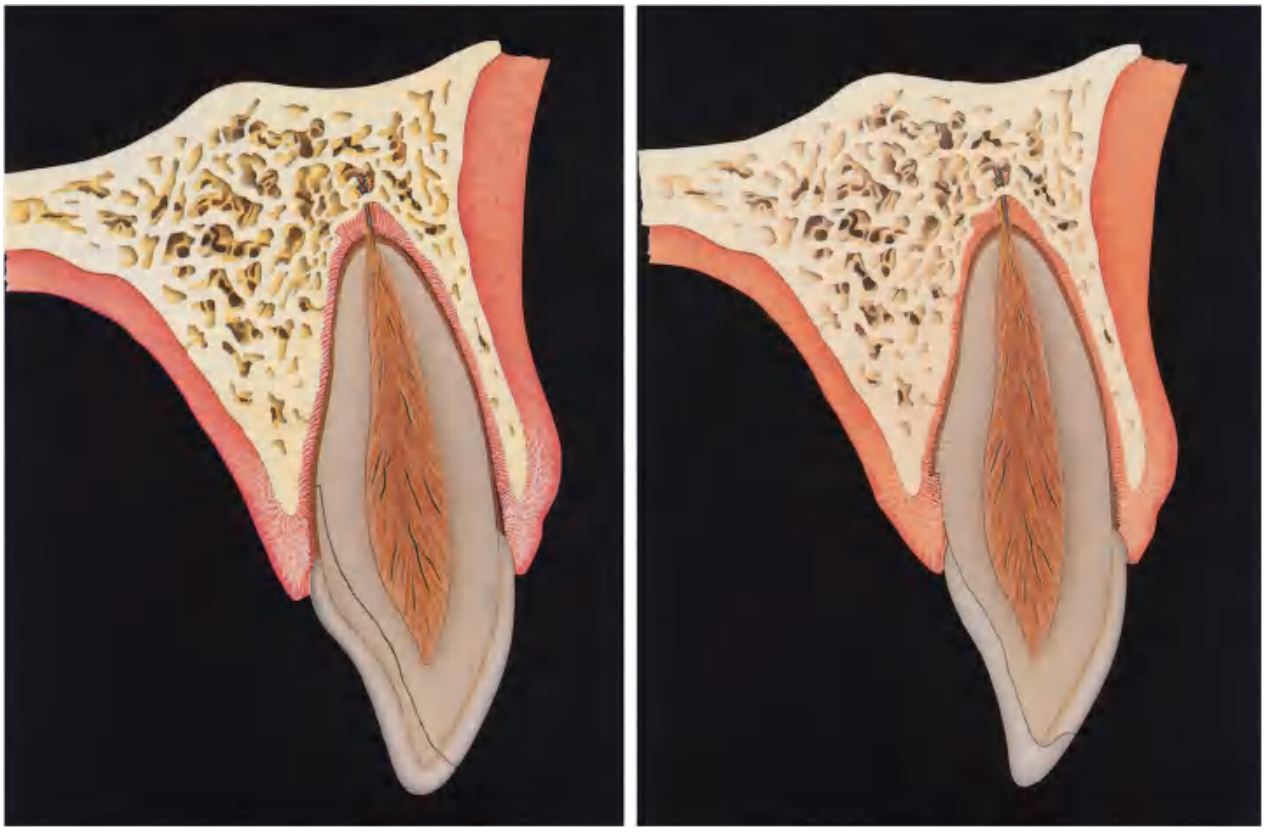


**Fig. 14.13** Removal of the coronal fragment and supragingival restoration.

**Fig. 14.14 Removal of the coronal fragment and supragingival restoration**
This 16-year-old girl has suffered a crown-root fracture which has exposed the palatal root surface. The clinical condition is shown after fragment removal.

**Condition 1 week later**
The palatal fragment has been removed and the exposed root dentin smoothed with a chisel (left); the exposed dentin was covered with calcium hydroxide and a temporary crown. Two weeks later (right) creeping reattachment is seen and the palatal aspect of the crown has been restored with a dentin bonded composite resin restoration.

**Follow-up**
Clinical and radiographic condition 4 years after treatment. (Courtesy of Dr. B. Malmgren, Eastman Institute, Stockholm, Sweden.)

## Indication

This should only be used where the surgical technique does not compromise the esthetic result, i.e. only the palatal aspect of the fracture must be exposed by this procedure.

## Treatment procedure

After administration of a local anesthetic, the coronal fragment is removed and the fracture surface carefully examined. It is important to remember that most crown-root fractures contain a lingual step (Fig. 14.15). In some cases, this step is a part of an incomplete or complete fracture extending more apically (Fig. 14.8). It is therefore essential to determine whether the lingual step in the root is part of a secondary fracture. This can be done during the gingivectomy by placing a sharp explorer or similar instrument at the base of the step and, with a gentle palatal movement, check whether abnormal mobility can be detected. Axial fracture lines running from the pulp chamber to the root surface should also be carefully explored. If these fractures are overlooked, an inflammatory reaction in the periodontium will develop after completion of the restoration (32, 34). The use of a conventional cast core and separate crown instead of a single unit restoration has the advantage that future changes in the position of the gingiva and subsequent loss of esthetics can easily be corrected.

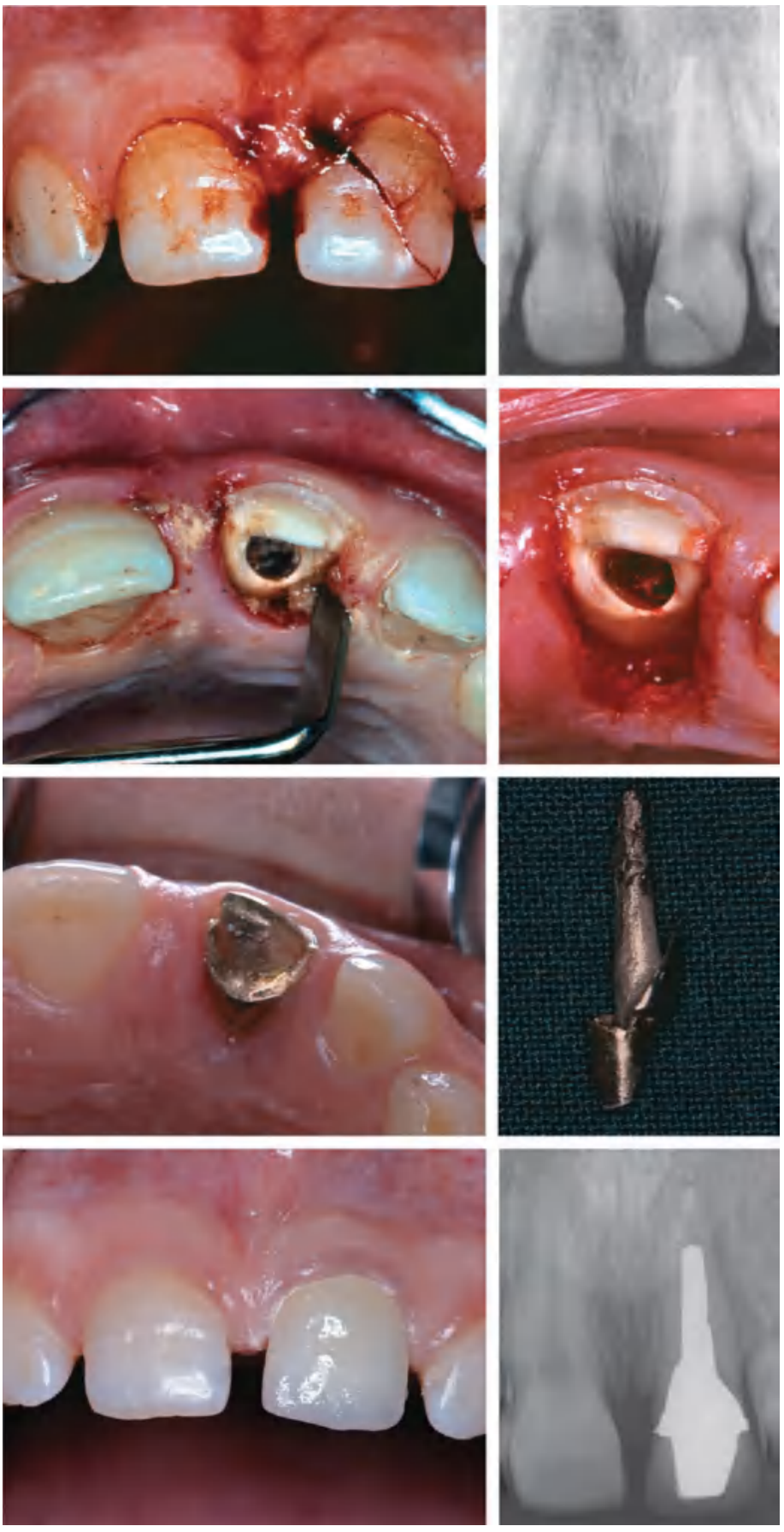


**Fig. 14.15 Removal of the coronal fragment and surgical exposure of the fracture**
Clinical and radiographic appearance of a complicated crown-root fracture.

**Exposing the fracture site**
The coronal fragment is removed. A combined gingivectomy and osteotomy expose the fracture surface.

**Constructing a post-retained crown**
After taking an impression, a post-retained full crown is fabricated.

**The finished restoration**
The clinical and radiographic condition 2 months after insertion of the crown.

### Cost–benefit

Treatment time is short. The long-term prognosis of these restorations has been evaluated and the following results obtained (34). After gingivectomy, and despite good marginal adaptation, regrowth of the gingiva often takes place, leading to development of a pathologic pocket palatally and inflammation of the surrounding gingiva. After some years, this can result in labial migration of the restored teeth. Migration has been found to be approximately 0.8 mm over a 5-year period (34) (Fig. 14.16).

## Orthodontic extrusion of apical fragment

### Treatment principle

The treatment principle is to move the fracture to a supragingival position orthodontically (Fig. 14.17) and was introduced in 1973 by Heithersay (33). Since then several clinical studies have supported its value (31, 35–39, 46–53).

A B

**Fig. 14.16** Labial movement of a crown-root-fractured tooth, restored after surgical exposure of the fracture surface. A. Condition immediately after restoration of the right lateral incisor. B. Condition after 6 years. From (34).

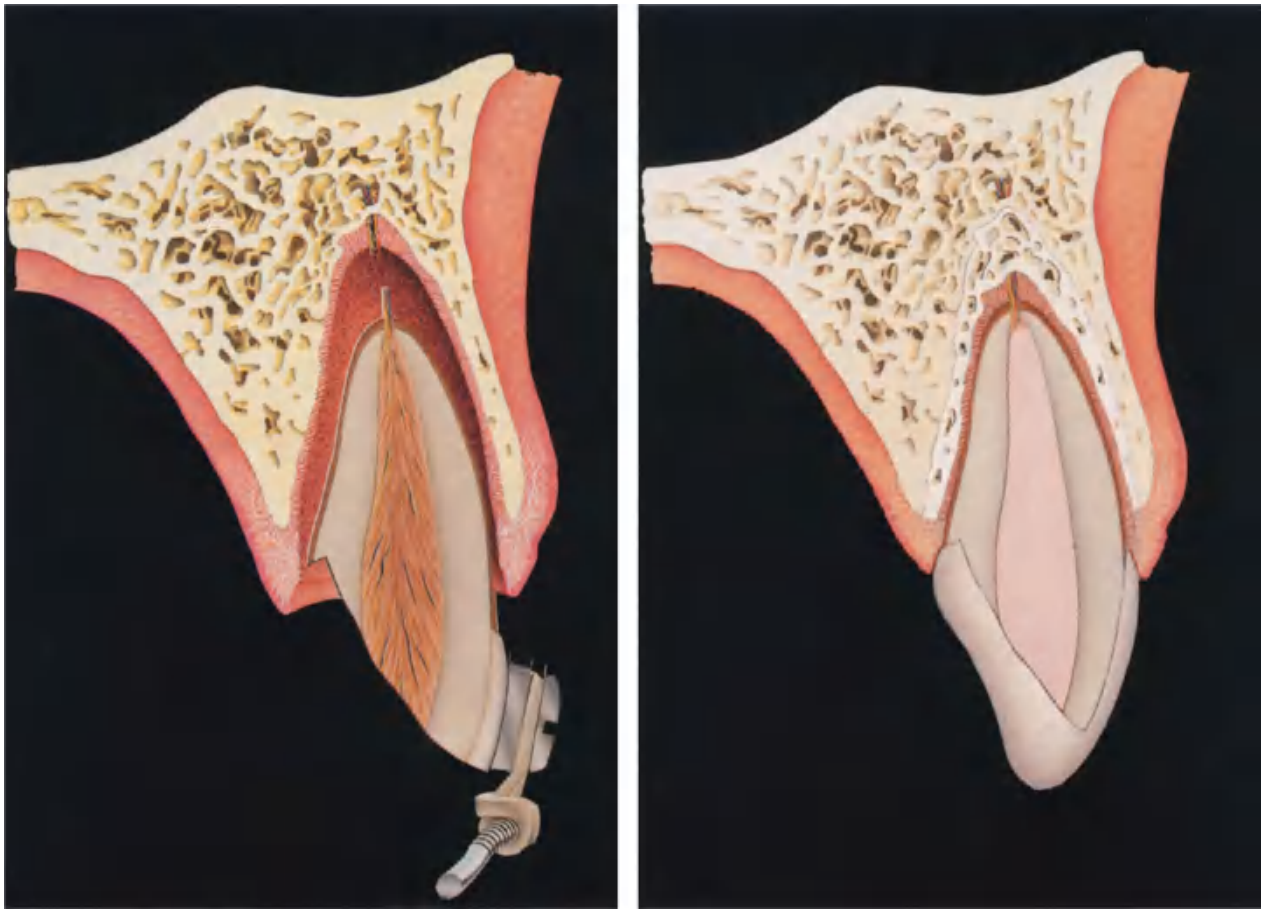


**Fig. 14.17** Removal of the coronal fragment and orthodontic extrusion of the root.

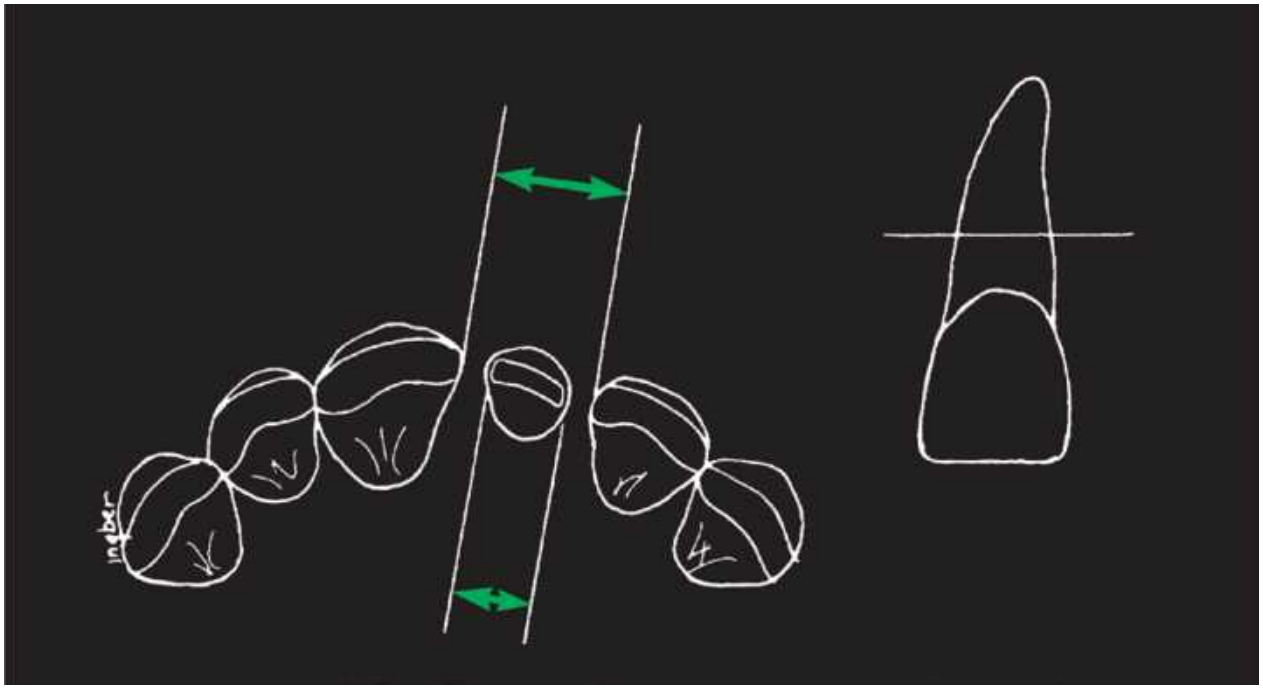
## Indication

This is the only method that can be used for uncomplicated crown-root fractures if pulp vitality is to be preserved. It can also be used for complicated crown-root fractures but is more time-consuming than surgical extrusion. In cases where it is desirable to reconstruct osseous and/or gingival defects, slow orthodontic extrusion can be used to guide downgrowth of these tissues. The cervical diameter after extrusion is essential and should be analyzed before extrusion (Fig. 14.18).

## Treatment procedure

In teeth with *completed root formation* and a crown-root fracture, endodontic therapy can be performed prior to removal of the coronal fragment (i.e. while the coronal fragment is splinted to the adjacent teeth). Fragment retention

A



B

**Fig. 14.18** Cervical root dimensions after extrusion. A. Tooth preparation at the level of the cemento-enamel junction is directly related to the fixed amount of space between the teeth. B. Tooth preparation after eruption of the apical root fragment; the diameter of the preparation is small relative to the fixed amount of space between the teeth. Restoration will thus require greater attention. From (36).

**Fig. 14.19** Orthodontic extrusion of a crown-root-fractured incisor. A. Before treatment. The patient is a 16-year-old female who suffered a dental trauma 7 years ago. The two central incisors were fractured and were restored with composite. However, the restoration of the right central incisor failed due to secondary caries. B. After treatment. The right extruded central incisor was restored with a porcelain jacket crown after orthodontic extrusion and the left incisor was restored with new composite. C and D. Incisal and palatal aspect at the first examination. E. Radiograph at the first examination. Caries has invaded to the crest of the alveolar bone. Buccal aspect after endodontic treatment and before orthodontic extrusion. There seems to be no sound tooth structure preserved above the alveolar crest. F. Condition after endodontic treatment. G. The occlusal relation; enough space is found to accommodate an orthodontic appliance. H. After placing an orthodontic appliance, a hook is placed in the canal. Elastic is placed between the wire and the hook, and the distance between them is 4 mm. I. Radiograph before extrusion. J and K. Palatal and buccal aspect at the start of extrusion. A laminate is bonded to neighboring teeth. Note there is enough space between the gingival margin and the cervical margin of the temporary crown. L. Radiograph taken at the start of extrusion.

can facilitate the endodontic procedure, whereby placement of a rubber dam and tissue control of the operative field is improved (in contrast to fragment removal). Thereafter orthodontic extrusion is performed (66) (Fig. 14.19). Recent esthetic improvements have been achieved whereby the extrusion devices have been masked by using either the hollowed out crown portion of the tooth or a pontic fastened to adjacent teeth with wire (62). The orthodontic procedure is described in Chapter 28.

In teeth with *incomplete root formation*, pulp capping or pulpotomy may be performed. Thereafter, orthodontic traction is initiated; this technique is further described in Chapter 28. Recently, a new method of orthodontic extrusion with magnets has been described. One or two neodymium-iron-boron magnets were attached to the remaining root and a second, larger magnet was incorporated in a removable appliance. The roots were extruded over a period of 9–11 weeks; two patients were successfully treated using this method (63).

In cases of *incomplete vertical fracture of immature incisors* (see later), it can be difficult to gain access to the pulp and to control moisture and hemorrhage in order to perform a pulpotomy. It could then be an advantage to seal the fracture lines facially and orally with an acid-etch technique and an unfilled resin while the tooth is being extruded. Once the fracture is at a level where access is possible, the fragment can be removed, and pulp capping or pulpotomy performed (see Chapter 28) and the crown restored.

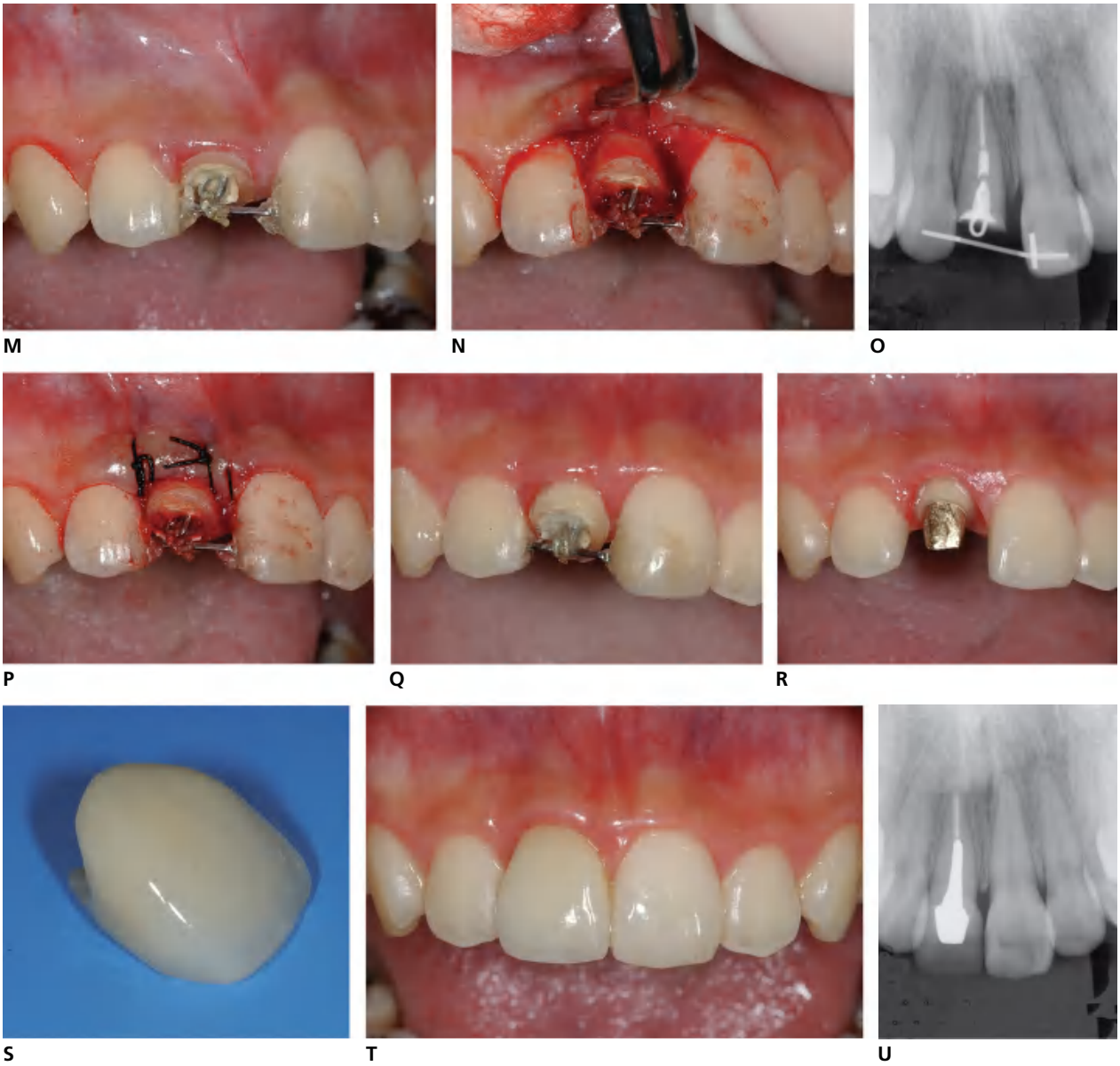



**Fig. 14.19** (*Continued*) M. Labial aspect just after extrusion. Note that gingival tissue has grown along with the extruded tooth resulting in an unacceptable gingival topography. N. Apically repositioned flap. A gingival flap was raised and positioned apically after osseous resection of excessive bone around the extruded tooth. O. Radiograph taken 6 weeks later at the end of the extrusion. A slight amount of bone seems to have regenerated along with extrusion. P. Immediately after surgery. Q. Three months after extrusion. R. Four months after extrusion. S. A porcelain jacket crown to be seated. T. Buccal view after crown placement. U. Radiograph after the restoration.

An important question is: how much can a tooth be extruded and still maintain reasonable periodontal support? This can best be answered by considering the crown-root ratio. If the goal of extrusion is a crown-root ratio of approximately 1:1, a central incisor can be extruded 2–4 mm, while a lateral incisor can be extruded 4–6 mm. However, it should be noted that, as yet, no study has confirmed that a crown-root ratio of 1:1 cannot be exceeded while maintaining stable periodontal support. Clinical experience with long-term observations of root fractures showing healing with interposition of connective tissue (i.e. a shortened root; see Chapter 28) indicates that a relatively large deviation from a crown-root ratio of 1:1 can be accepted without resulting in excessive mobility or breakdown of the periodontium.

After extrusion and stabilization, restorative procedures can be carried out as described under the surgical approach. However, special restorative problems arise when dealing with a root which has been moved coronally. Thus, it should be realized that the extruded root will normally present a smaller cervical diameter for restoration than a root in its normal position (36, 51) (Fig. 14.18). If conventional restorative techniques are employed, the final restoration will have greater divergence from the gingival margin incisally. However, experience gained by the restoration of single tooth implants has shown that conventional porcelain techniques can be used to restore anterior symmetry.

Concerning the prognosis of teeth treated by forced eruption, nothing definite can be said at the present time. However, experience to date has shown that this procedure can result in stable periodontal conditions (34).

### Cost–benefit

The procedure is slow and cumbersome (Table 14.1). However, it can provide excellent esthetic results and gingival health appears to be optimal. Moreover, pulpal vitality can in some cases be maintained where indicated.

## Surgical extrusion of apical fragment

### Treatment principle

The treatment principle is to surgically move the fracture to a supragingival position (Fig. 14.19). This procedure was introduced by Tegsjö et al. in 1978 (40, 54), and the method further developed by Bühler (55, 56) and Kahnberg (57–61).

### Indication

This approach should only be used where there is completed root development and the apical fragment is long enough to accommodate a post-retained crown. Surgical extrusion results in loss of pulp vitality if performed in uncomplicated crown-root fractures. In such cases orthodontic extrusion may be a better alternative if there is a desire to keep the pulp vital.

### Treatment

Clinical experience has shown that the time factor for removal of the coronal fragment is not critical for success of treatment. Thus, fragment removal can be instituted days or weeks after injury. The pulp can be extirpated and the tooth root filled at this time. However, clinical studies have shown that postponement of endodontic therapy for 3–4 weeks gives better results (61). If the latter approach is chosen, the root canal is sealed with zinc oxide-eugenol cement. Moreover, experience has shown that postponement of the surgical procedure for 2–3 weeks facilitates atraumatic extraction due to inflammatory processes arising in the periodontal ligament.

The apical fragment is luxated with a thin periosteal elevator (Fig. 14.20). The extracted root is then inspected for incomplete fractures that will contraindicate repositioning of the root (Fig. 14.21). The root is moved into a more coronal position and stabilized in the new position with interproximal sutures and/or a splint. In case of palatally inclined fractures, 180° rotation can often imply that only slight extrusion is necessary to accommodate crown preparation due to the difference in position of the cemento-enamel junction labially and palatally. The exposed pulp is covered with a zinc oxide-eugenol cement. After 3–4 weeks, the tooth can be treated endodontically. After another 1–2 months, the tooth can be restored with a post-retained crown.

### Prognosis

In the reported series of patients treated, with up to 5-year observation periods, either no or slight root resorption (repair-related resorption) was seen (54, 59) (Table 14.2 and Figs 14.22, 14.23 and 14.24). In a recent 10-year follow-up study of 19 surgically extruded teeth, Kahnberg showed that all teeth survived, and only one showed cervical resorption (65).

### Cost–benefit

Several clinical studies and two systematic reviews have indicated that this generally is a safe and rapid method for the treatment of crown-root fractures (75, 76). However, pulp vitality must be sacrificed (Table 14.1).

## Vital root submergence

In cases not considered restorable in young individuals, it might be indicated to keep the root portion in place in order to maintain the alveolar width and height (67, 68). This procedure is described in Chapter 32.

## Extraction

This alternative is relevant when none of the previously mentioned treatment procedures are indicated. With respect to subsequent tooth replacement, it should be

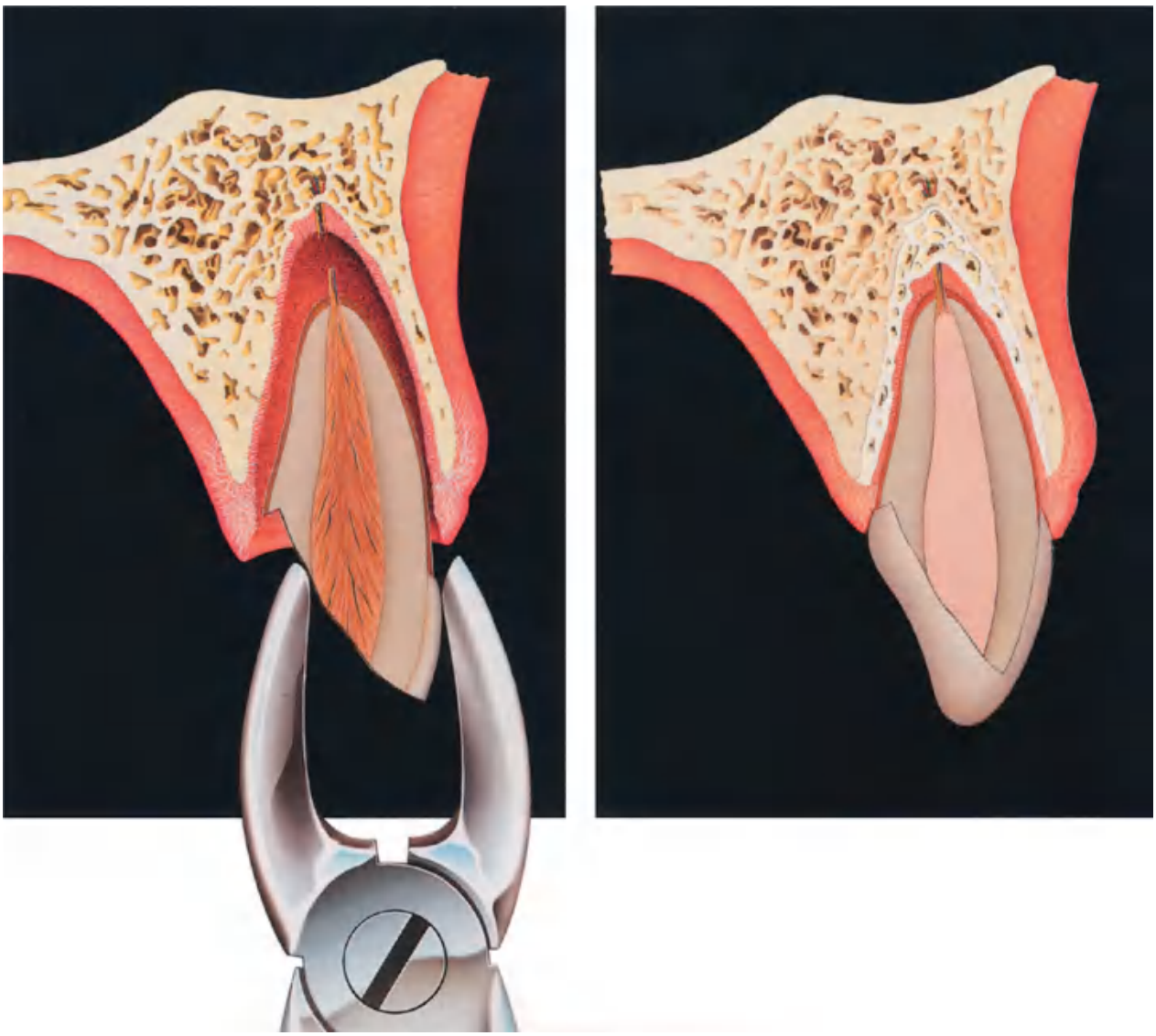


**Fig. 14.20** Removal of the coronal fragment and surgical extrusion of the root.

**Table 14.2** Long-term results of surgical extrusion of crown-root-fractured anterior teeth.

| Study | Observation period, years (mean) | Age of patient, years (mean) | No. of teeth | Tooth survival (%) | PDL healing* (%) | Apical healing (%) | Gingival healing (%) |
|---|---|---|---|---|---|---|---|
| Tegsjö et al. 1987 (54) | 4 (4.0) | 9–33 (15.0) | 56 | 91 | 88 | 98 | |
| Kahnberg 1988 (59) | 5.5 (2.4) | 13–75 (31.0) | 17† | 100 | 65 | 94 | 100 |
| | | 13–75 (31.0) | 41‡ | 100 | 74 | 95 | 100 |
| Caliskan et al. 1999 (64) | 1.2 | 10–45 (21.5) | 20 | 100 | 90 | 100 | 95 |
| Khayat & Fatehi 2006 (74) | 1.0 | 10–40 (26.0) | 21 | 100 | 95 | | |

* Non-healing represents cases with surface resorption that results in a slight shortening of the root.
† Transplant performed via apical exposure.
‡ Transplant performed via coronal approach.
PDL, periodontal ligament.

remembered that the supporting bone is very rapidly resorbed (see Chapter 32). For this reason the apical root fragment may be submerged to maintain the volume of the alveolar process. Provided growth is complete, implant treatment may be a good alternative (see Chapter 35). In still-growing patients orthodontic space closure (see Chapter 28) or autotransplantation (see Chapter 33) are better alternatives.

**Fig. 14.21 Removal of the coronal fragment and surgical extrusion of the root**
A complicated crown-root fracture in a 13-year-old boy. The loose fragment is stabilized immediately after injury with a temporary crown and bridge material using the acid-etch technique.

**Incision of the PDL**
After a local anesthesia the PDL is incised using a specially contoured surgical blade. The PDL is incised as far apically as possible.

**Luxation of the root**
The root is then luxated with a narrow elevator which is placed at the mesiopalatal and distopalatal corners respectively.

**Extracting the root**
The root is extracted and inspected for additional fractures.

**Replanting the apical fragment**
The root is tried in different positions in order to establish where the fracture is optimally exposed, yet with minimal extrusion. In this instance, optimal repositioning was achieved by rotating the root 45°.

**Stabilization of the apical fragment during healing**
The root is splinted to adjacent teeth. The pulp is extirpated and the access cavity to the root canal closed.

**Root filling**
Two weeks after initial treatment, endodontic therapy can be continued, in the form of an interim dressing with calcium hydroxide. The root canal is obturated with gutta-percha and sealer as far apically as possible 1 month after surgical extrusion.

**Completion of the restoration**
Two months after surgical extrusion, healing has occurred and it is possible to complete the restoration. Reproduced from (58) with permission.

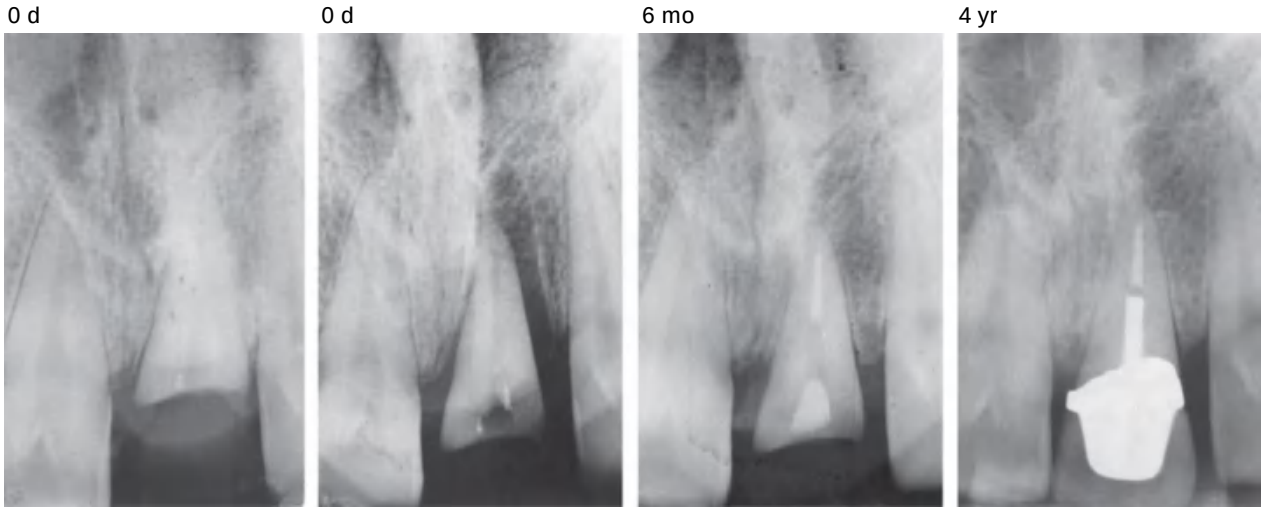



**Fig. 14.22** Intra-alveolar transplantation of a crown-root-fractured central incisor. No sign of root resorption is seen 4 years after transplantation. Reproduced from (58) with permission.

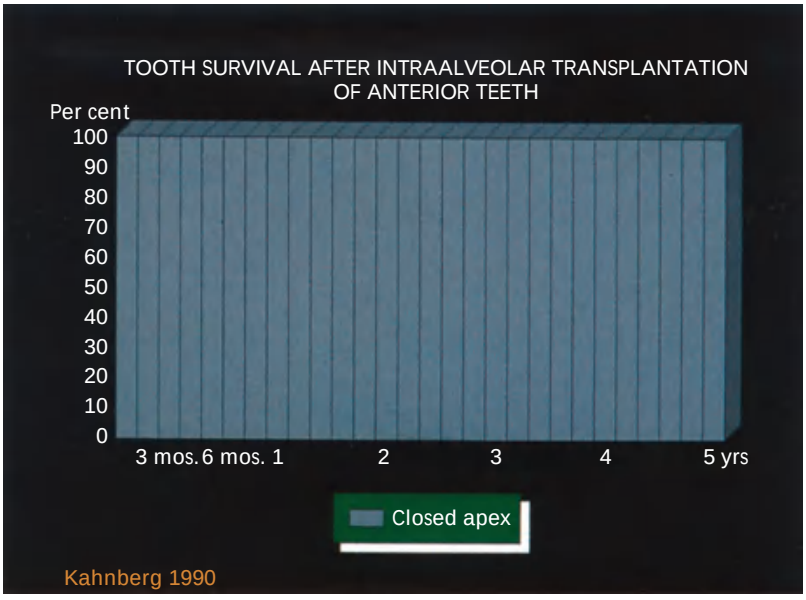



**Fig. 14.23** Tooth survival of 68 intra-alveolar transplanted anterior teeth with completed root formation at time of surgery. From (60).

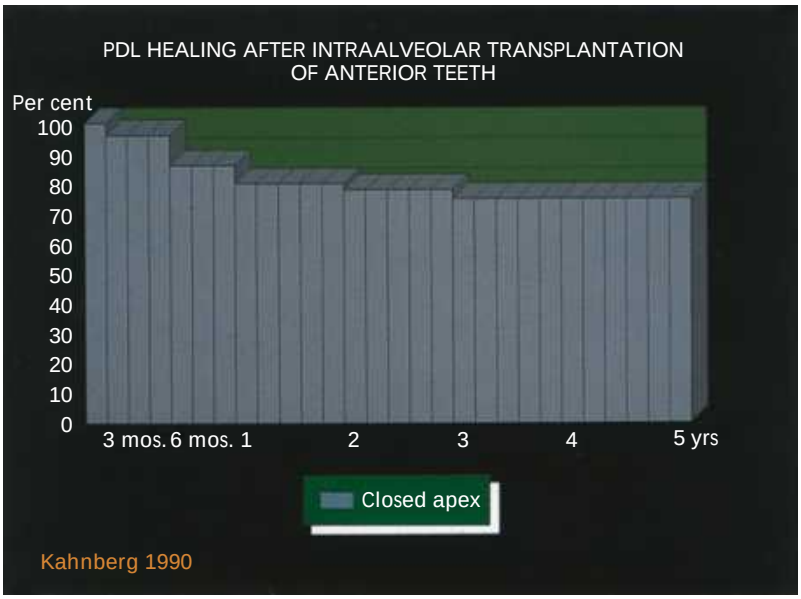



**Fig. 14.24** Periodontal ligament (PDL) healing of 68 intra-alveolar transplanted anterior teeth with completed root formation at time of surgery. Complications involved surface resorption of the repositioned tooth. From (60).

## Essentials

### Permanent teeth (primary teeth)

See also Chapter 22.

#### *Emergency procedures*

Fragments of crown-root-fractured teeth can be temporarily splinted to alleviate pain from mastication (Fig. 14.12). Definitive treatment should be provided within a few days.

#### *Definitive treatment*

The level of the fracture determines the type of therapy (i.e. extraction of the root, bonding of fragment, surgical exposure of the fracture surface or orthodontic or surgical extrusion of the root).

### Extraction of the tooth

Indicated in teeth where the coronal fragment comprises more than one-third of the clinical root and in the case of fractures following the long axis of the tooth.

### Bonding of coronal fragment

Indicated for superficial crown-root fractures that do not extend below the alveolar crest.

- Administer local anesthesia
- Raise lingual flap
- Remove the fragment and ensure that there are no extra fractures
- Secure hemostasis with sterile cotton pellets soaked in adrenalin
- Clean the fracture surface
- Etch the fracture surface with 35% phosphoric acid gel
- Bond and cure the fragment and remove excess bone material
- Suture the lingual flaps

### Removal of coronal fragment and supragingival restoration

Indicated in superficial fractures that do not involve the pulp (Figs 14.13 and 14.14).

- Administer local anesthesia
- Remove loose fragments
- Smooth rough subgingival fracture surface with a chisel
- Cover supragingival exposed dentin
- When gingival healing has occurred a supragingival restoration is made using bonded composite or the original fragment where the subgingival portion has been removed

### Surgical exposure of fracture surface

Indicated in teeth where the coronal fragment comprises one-third or less of the clinical root (Fig. 14.15).

- Administer local anesthesia
- Remove loose fragments
- Perform a pulpectomy and obturate the root canal with gutta-percha and a sealer
- Expose the fracture surface with a gingivectomy and ostectomy
- Restore the tooth with a post-retained porcelain jacket crown

### Orthodontic extrusion of apical fragment

Indicated in teeth where the coronal fragment comprises one-third or less of the clinical root (Figs 14.17 and 14.19).

- Administer local anesthesia
- Remove loose fragments
- In teeth with *mature root formation*, perform pulpectomy and obturate the root canal with gutta-percha and a sealer. In teeth with *immature root formation*, perform a cervical pulpotomy (see Chapter 25)
- Expose the fracture surface via orthodontic extrusion of the root (see Chapter 28)
- When the root is extruded, perform a gingivectomy and ostectomy, if needed, to restore symmetry of the gingival contour
- Restore the tooth temporarily and splint to adjacent teeth for a retention period of 6 months
- After the retention period, restore definitively

### Surgical extrusion of apical fragment

Indicated in teeth where the coronal fragment comprises less than half the root length (Figs 14.19 and 14.21).

- Administer antibiotics and local anesthesia
- The pulp can be extirpated and the root canal filled with gutta-percha and a sealer prior to intra-alveolar transplantation; or endodontics can be postponed and the root canal entrance sealed with a zinc oxide-eugenol cement
- The periodontal ligament is incised, the tooth luxated with an elevator and the tooth extracted with forceps
- The root surface is inspected for incomplete root fractures, which would contraindicate transplantation
- The root is repositioned at a level 1 mm coronal to the alveolar crest. If desirable, the root can be rotated to achieve a maximum periodontal surface area within the socket
- The tooth is stabilized using interproximal sutures
- Take a postoperative radiograph
- After 2–3 weeks, the transplant is usually firm. If the root canal has not been filled, calcium hydroxide can be used as an interim dressing which will ensure apical hard tissue closure. A temporary restoration can now be fabricated

- After some months, a permanent root filling as well as a definitive crown restoration can be completed
- If a gutta-percha root filling has been made prior to transplantation, the tooth can be restored after 2 months

### Vital root submergence

Indicated in young individuals where the earlier-mentioned treatment alternatives cannot be carried out in order to maintain the dimensions of the alveolar process.

- Administer a local anesthetic
- A flap is raised
- The supra-alveolar fragments of the tooth are removed
- The flap is closed over the exposed root including the pulp
- Insert a space maintainer

### Extraction

Indicated in cases where none of the other treatments can be performed.

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# 15

# Root Fractures

F. M. Andreasen, J. O. Andreasen & G. Tsilingaridis

## Terminology, frequency and etiology

Root fractures, defined as fractures involving dentin, cementum and pulp, are relatively uncommon among dental traumas, comprising 0.5–7% of the injuries affecting the permanent dentition (1–8, 71, 72, 111, 140, 141) and 2–4% in the primary dentition (1, 4, 139, 140). Frequent causes of root fractures in the permanent dentition are fights and foreign bodies striking the teeth (1).

The mechanism of root fractures is usually a frontal impact which creates compression zones labially and lingually. The resulting shearing stress zone then dictates the plane of fracture (see Chapter 11). The result on a histologic level is a periodontal ligament injury (rupture and/or compression) confined to the coronal fragment and stretching or lacerating the pulp at the level of fracture (Figs 15.1 and 15.2).

**Fig. 15.1** Palatal and incisal displacement of left central incisor due to root fracture.

## Clinical findings

Root fractures involving the *permanent dentition* predominantly affect the maxillary central incisor region in the age group of 11–20 years (1, 9–12, 115). In younger individuals, with the permanent incisors in various stages of eruption and with incomplete root development, root fractures are unusual (13, 115, 147). This finding is possibly related to the elasticity of the alveolar socket which renders such teeth more susceptible to luxation injuries than to fractures (73). However, careful scrutiny of radiographs following luxation injuries in this age group can sometimes reveal incomplete (partial) root fractures (see later). In the *primary dentition*, root fractures are uncommon, but most frequent at the age of 3–4 years where physiologic root resorption has begun, thereby weakening the apical part of the root (74).

Root fractures can often be associated with other types of injuries; among these, concomitant fracture of the alveolar process, especially in the mandibular incisor region, is a common finding (1).

Clinical examination of teeth with root fractures usually reveals a slightly extruded tooth, frequently displaced in an oral direction (Fig. 15.1). While the site of the fracture determines the degree of tooth mobility, it is usually not possible to distinguish clinically between displacement due to a root fracture and a luxation injury. Diagnosis is entirely dependent upon radiographic examination.

## Radiographic findings

Radiographic demonstration of root fractures is facilitated by the fact that the fracture line is most often oblique and at an optimal angle for radiographic disclosure (13)

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(Figs 15.3 and 15.4). In this context, it should be remembered that a root fracture will normally be visible only if the central beam is directed within a maximum range of 15–20° of the fracture plane (112, 115). Thus, if an ellipsoid radiolucent line is seen on a radiograph, two additional periapical radiographs should be taken – one with an increased angulation of 15° to the original and the second at a negative angulation of 15° to the original (75) (Fig. 15.3). However, the direction of the fracture can vary considerably. Thus, the typical apical or mid-root fracture follows a steep course facio-orally in an incisal direction, while fractures of the cervical third tend to be more horizontal. This change in direction dictates a radiographic technique that involves multiple exposures, including a steep occlusal exposure, which is optimal for detecting fractures of the apical third of the root (115, 174). There is no significant difference in the sensitivity of digital radiography and conventional radiography in their capacity to disclose these fractures (142, 174).

In several studies it has been shown that multiple radiographic projections augment the chance of detection of root fractures (115, 168).

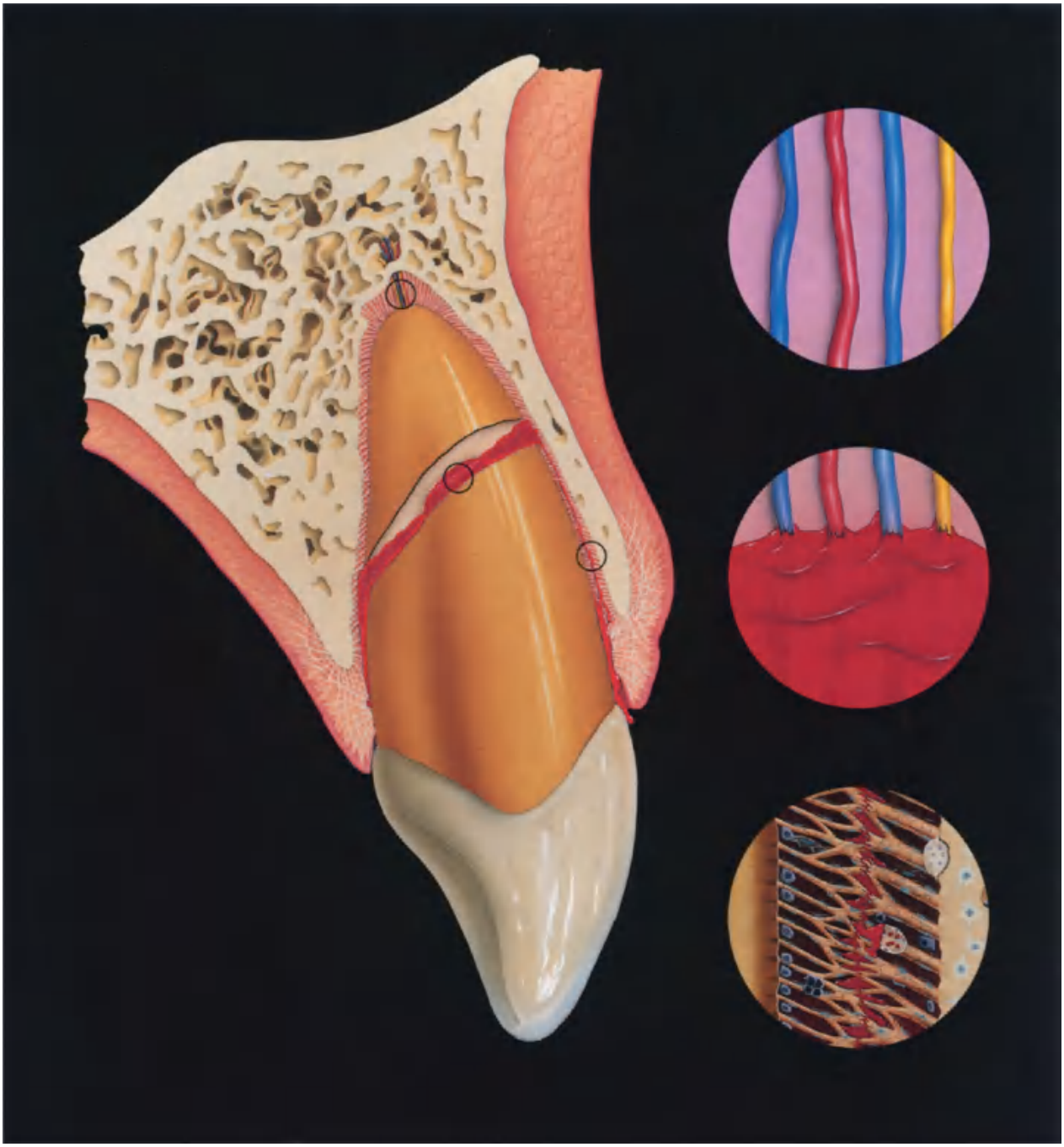


**Fig. 15.2** Pathogenesis of a root fracture. A frontal impact displaces the tooth orally and results in a root fracture and displacement of the coronal fragment. This results in both pulpal and PDL damage coronally. The pulp in the apical part of the root usually remains vital.

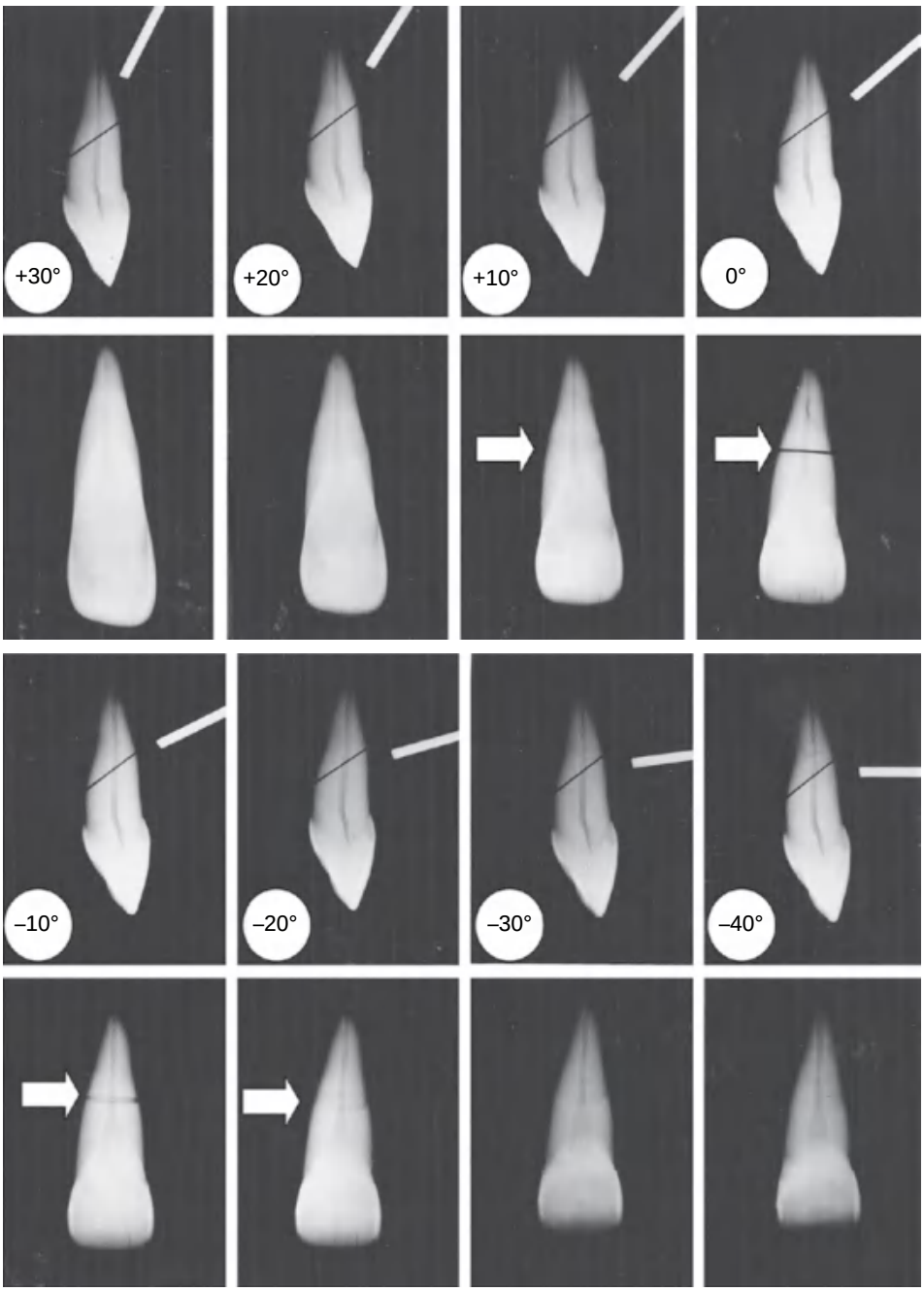



**Fig. 15.3** Radiographs taken of a human tooth with an artificial root fracture. The top row of radiographs indicates the direction of the central beam. The 0° beam corresponds to the direction of the central beam used in ordinary occlusal exposures. It appears from the bottom row that the fracture line was only disclosed with an angulation of the central beam from +10° to −20° in relation to the occlusal angulation of 0° (arrows). It also appears that deviations of the central beam from the fracture plane tend to depict the fracture as an ellipsoid structure mimicking an intermediary fragment.

Root fractures occasionally escape detection immediately after injury, even though they are clearly visible in later radiographs (4, 15–17, 76, 77) (Fig. 15.5). This can be due to the development of either hemorrhage or granulation tissue between the fragments, which displaces the coronal fragment incisally or due to resorption at the fracture line, which is a part of the healing process (see later) (113, 175, 176).

In previous clinical studies, root fractures were seen to occur most often in the apical or middle third of the root, and only rarely in the coronal one-third (11–13, 76, 77). However, in more recent clinical studies, fractures of the middle third of the root were the most frequent, while fractures of the apical and cervical thirds occurred with equal frequency (114, 115). A single transverse fracture is the usual finding; however, oblique or multiple fractures can occur (Fig. 15.6).

Root fractures of teeth with incomplete root formation can demonstrate a partial root fracture, a possible analog to 'green stick' fractures of long bones (4, 18). They are usually seen as a unilateral break in the continuity of the thin root canal wall/root surface of the immature root (Fig. 15.7). In a large clinical study, these fractures were subsequently seen to heal with hard tissue formation (115).

## Cone beam computed tomography in radiographic diagnosis of root fractures

The value of cone beam computed tomography (CBCT) in trauma-related horizontal root fractures will now be discussed (160–164, 166–168, 174). Not included in this discussion are endodontically related vertical root fractures, which present

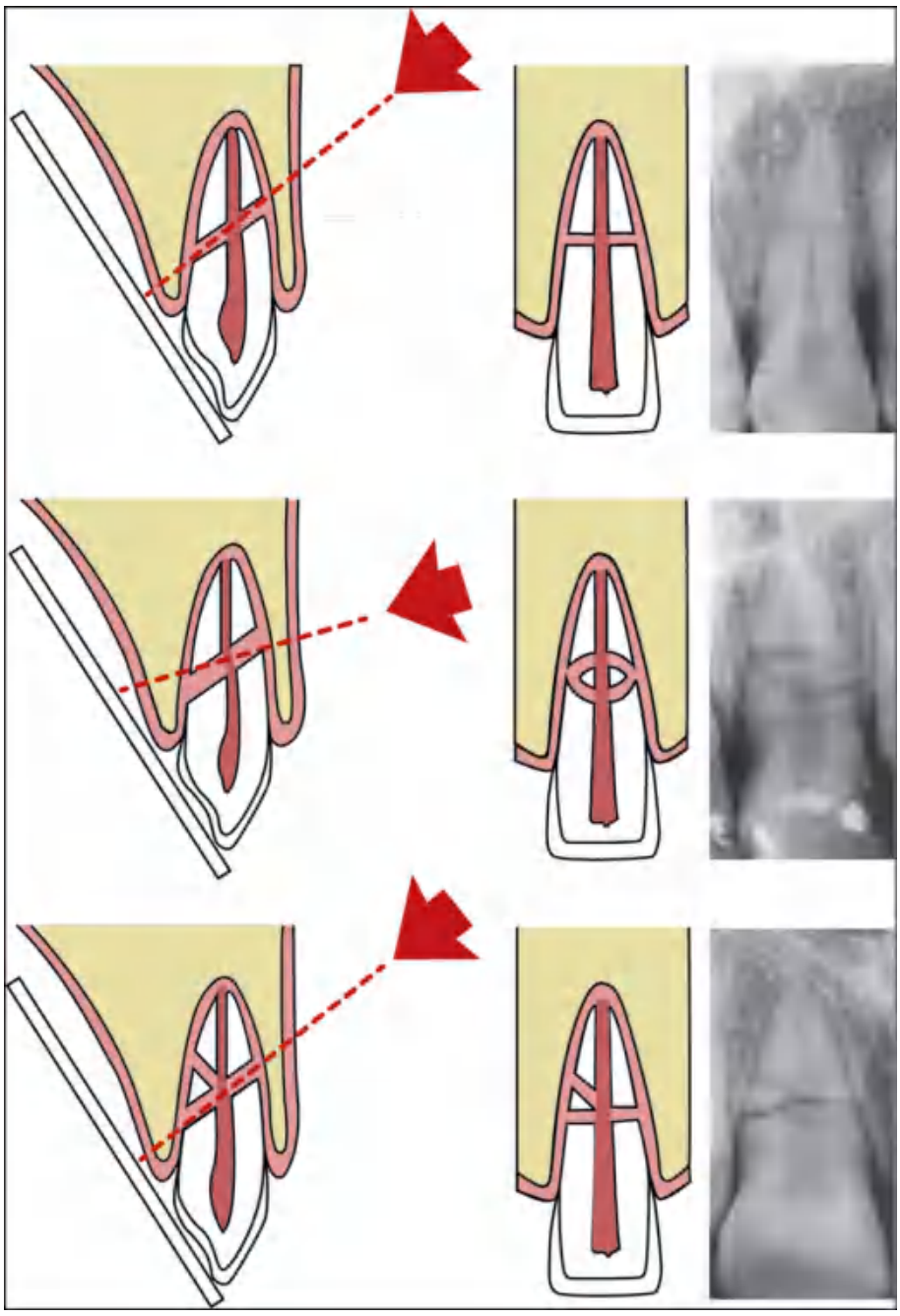


**Fig. 15.4** Radiographic demonstration of root fractures. The normal projection angle is parallel to the fracture surface, resulting in a single transverse line on the radiograph. A decrease or increase of the projection angle results in an ellipsoid fracture line on the radiograph. The fracture line in multiple root fractures shows an irregular shape on the radiograph. From (13).

special diagnostic problems as they often run in a mesiodistal plane, making radiographic detection extremely difficult. A significant meta-analysis of the value of CBCT has been published on this topic (162).

An interesting study published by Bornstein et al. in 2009 (161) compared recommended standard radiographic examination with CBCT examination of the same 44 root-fractured teeth. This study showed that the usual classification of root fractures into apical, mid-root and cervical fractures based on a two-dimensional radiographic technique was different when CBCT was used. Thus the palatal part of the fracture line in mid-root fractures often took an oblique direction cervically. Such a relation was previously demonstrated in an analysis of 33 fracture lines by Andreasen in 1972 (165) (see Chapter 11, Fig. 11.31) and in histologic studies of root-fractured teeth (see Fig. 15.17 below).

With this new information from CBCT, the authors of the Bornstein et al. article have suggested that recommendations for acute treatment be changed, which is hardly realistic. The basic treatment is repositioning and splinting, and it is known that mid-root fractures have a high pulpal healing rate of 79%, not significantly different from cervical fractures (85%) (158). In the publication by Bornstein et al. (161), the figures from Cvek et al. (159) were examined with respect to the fate of 534 root-fractured teeth. Forty-two (0.8%), usually cervical root fractures, were extracted at the time of injury. It was argued that CBCT could have helped diagnose those. However, this statement ignores the fact that

**Fig. 15.5** Late appearance of a root fracture. In the radiograph taken immediately after injury the root fracture of the right central incisor is barely discernible. Four weeks later a fracture line is clearly visible.

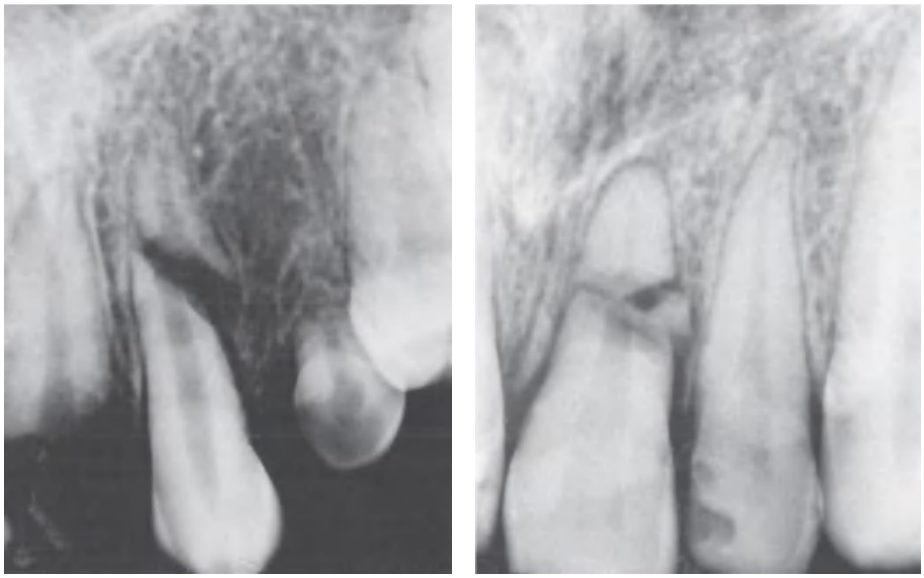


**Fig. 15.6** Oblique root fracture of a left cuspid and multiple root fracture of a left central incisor.

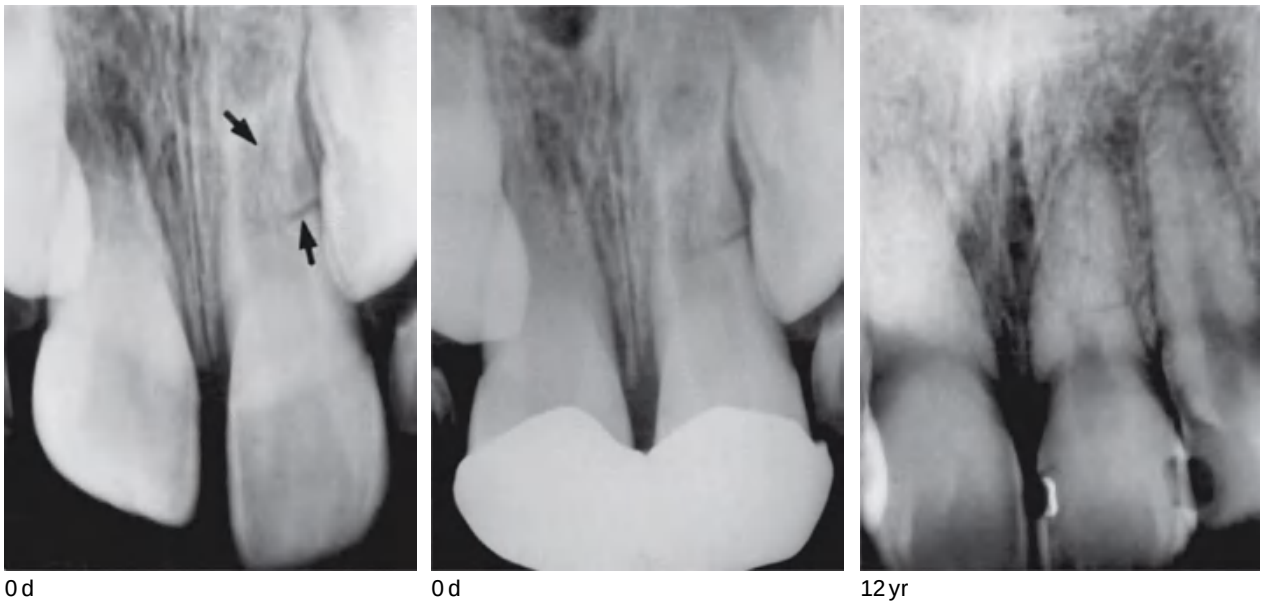



**Fig. 15.7** Incomplete root fracture of a left central incisor. Twelve years later the fracture line is hardly visible.

a periapical radiograph is usually as informative as CBCT, as these fractures usually have a strictly horizontal position. Moreover, there is the risk of overdiagnosis and hence overtreatment, as the work by Cvek et al. (159) demonstrated good healing in many cases despite delayed or no treatment when fractures had not been diagnosed until a later date.

In essence, CBCT presently has little to offer based on current knowledge of root fracture healing in diagnosis and treatment planning. Further studies are needed to prove whether the use of CBCT would result in a changed treatment strategy, which is one of the requirements needed for the use of this new diagnostic tool in root fracture diagnosis (167, 168, 174).

## Healing and pathology

### Root fracture healing

Healing events following root fracture are initiated at the pulpal and periodontal aspects of the fracture line and lead to two types of wound healing response (19–21, 78, 113–118) (Fig. 15.8). These processes apparently occur independently of each other and are sometimes even competitive in their endeavor to close the injury site with either pulpal or periodontally derived tissue.

On the *pulpal side* of the fracture, two healing events might occur, depending upon the integrity of the pulp at the level of fracture. Thus, if the pulp is intact at the fracture site (i.e. no displacement of the coronal fragment), it will react in a manner analogous to a coronal pulp exposure under optimal conditions (i.e. with an intact vascular supply and absence of infection). Odontoblast progenitor cells will be recruited and create a small, hard tissue dentin bridge that will unite the apical and coronal fragments after 2 weeks in dogs (20)). This bridge forms the initial callus and could stabilize the fracture (Figs 15.8 and 15.9). Callus formation is followed by deposition of cementum derived by ingrowth of tissue from the periodontal ligament at the fracture line, first centrally and then gradually obliterating the fracture site (20). Hard tissue union of the fractured root fragments cannot be diagnosed radiographically earlier than 3 months after injury and may take several years to be completed (113–115, 175).

In the event that the pulp is severed or severely stretched at the level of the fracture (i.e. displacement of the coronal fragment), a revascularization process in the coronal aspect of the pulp is initiated (144). In the absence of bacteria, this process will result in obliteration of the coronal pulp canal. While this revascularization process is under way, periodontally derived cells can dominate root fracture healing, resulting in 'union' of the coronal and apical root

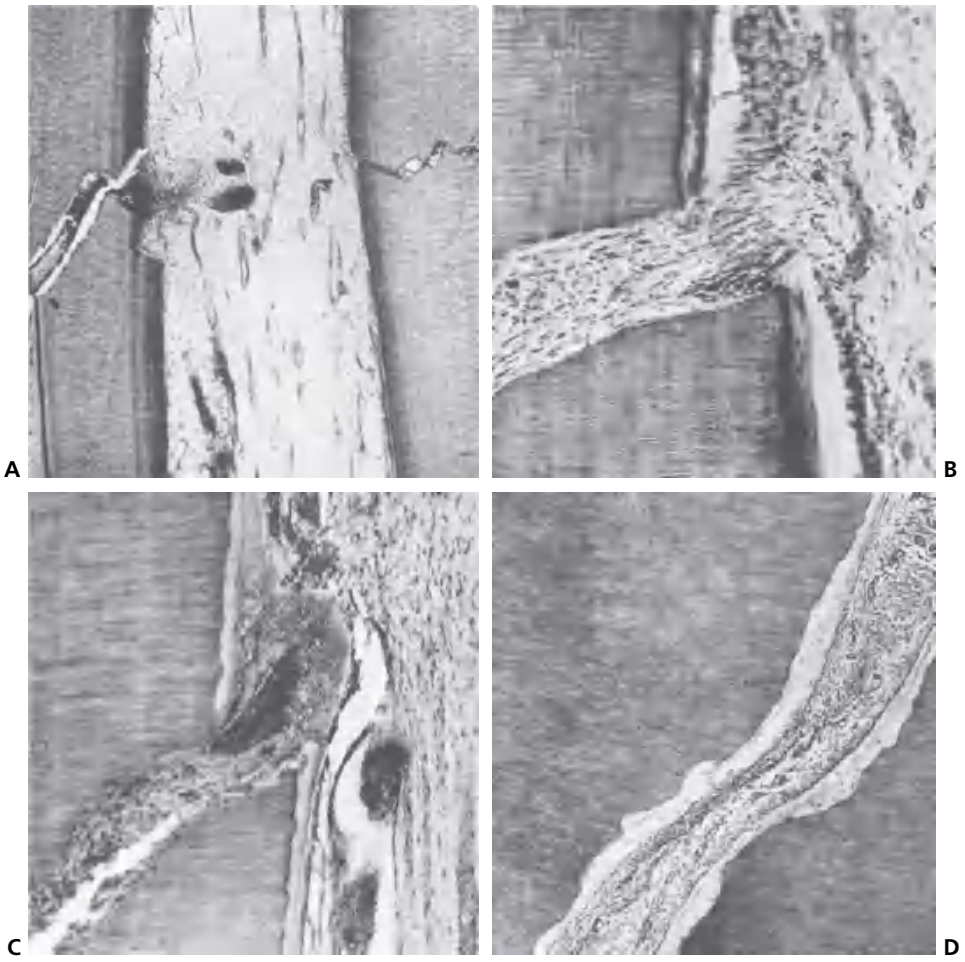



**Fig. 15.8** Healing sequence after experimental root fractures in dogs. A. Eight days postoperatively, proliferation of odontoblasts and pulpal cells. B and C. After 2 weeks, a dentinal callus is formed, uniting the fragments. D. Nine months after experimental fracture, connective tissue separates the fragments in the peripheral part of the fracture. From (20).

fragments by interposition of connective tissue (114, 115, 144, 175) (Fig. 15.10).

Finally, if bacteria gain access – usually to the coronal pulp – infected pulp necrosis results, with accumulation of inflamed granulation tissue between the two root fragments (Fig. 15.11). The source of these bacteria is still subject to debate, but three points of entry are considered likely: (1) through a tear in the coronal aspect of the periodontal ligament; (2) through exposed dentinal tubules (e.g. an associated crown fracture, or denuded neck of the tooth); or (3) via the invading neovasculature (anachoresis) (177).

During the initial stages of wound healing, traumatized pulpal and hard dental tissues can stimulate an inflammatory response and thereby trigger the release of a series of osteoclast-activating factors (see Chapters 2 and 17) (117).

Thus, root resorption processes beginning either at the periphery of the fracture line adjacent to the periodontal ligament or centrally at the border of the root canal were observed in 60% of clinical material of root-fractured permanent incisors (113). These processes could usually be detected within the 1st year after injury and preceded fracture healing and obliteration of the coronal aspect of the root canal. It should be noted that the apical aspect of the root canal almost always became obliterated. The changes observed represented three resorption entities:

(1) *External repair-related resorption* (i.e. rounding of proximal fracture edges at the periodontal side of the fracture) (Fig. 15.12).
(2) *Internal repair-related resorption* (i.e. rounding of fracture edges centrally, at the pulpal side of the fracture) (Figs 15.12 and 15.13).
(3) *Internal tunneling resorption* (i.e. resorption which burrows behind the predentin layer and along the root canal walls of the coronal fragment) (Figs 15.13 and 15.14).

It has been speculated that the various root resorption entities represent osteoclastic activity connected with ingrowth of new, highly vascularized connective tissue into the fracture site or coronal aspect of the root canal, as revascularization processes are known to elicit transient osteoclast activity (see Chapter 2) (113).

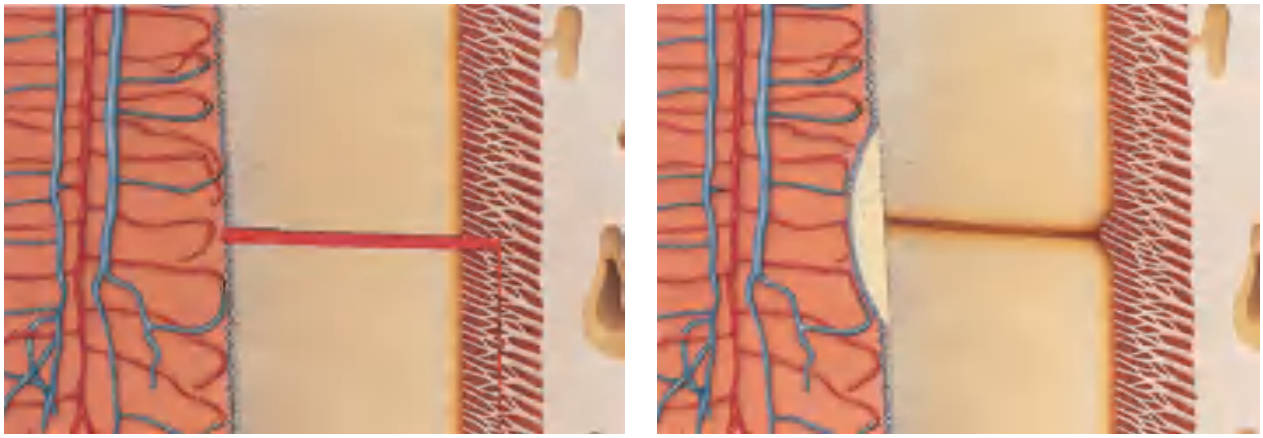


**Fig. 15.9** Hard tissue healing after root fracture. Due to minimal displacement of the coronal fragment, the pulp is probably only slightly stretched at the level of the fracture. Fracture healing with ingrowth of cells originating from the apical half of the pulp ensures hard tissue union of the fracture.

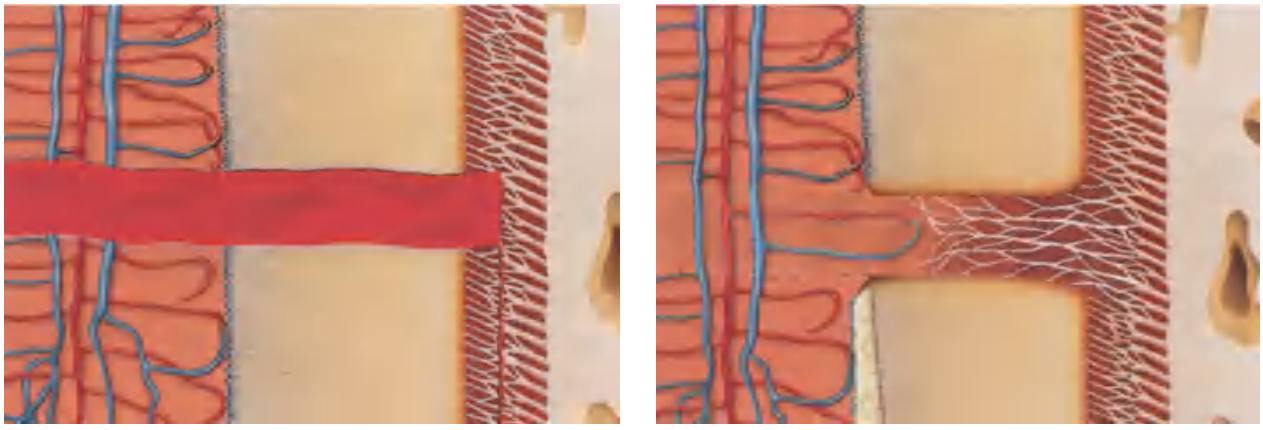


**Fig. 15.10** Connective tissue healing after root fracture. The pulp is ruptured or severely stretched at the level of the fracture following displacement of the coronal fragment. Healing is dominated by ingrowth of cells originating from the periodontal ligament and results in interposition of connective tissue between the two fragments.

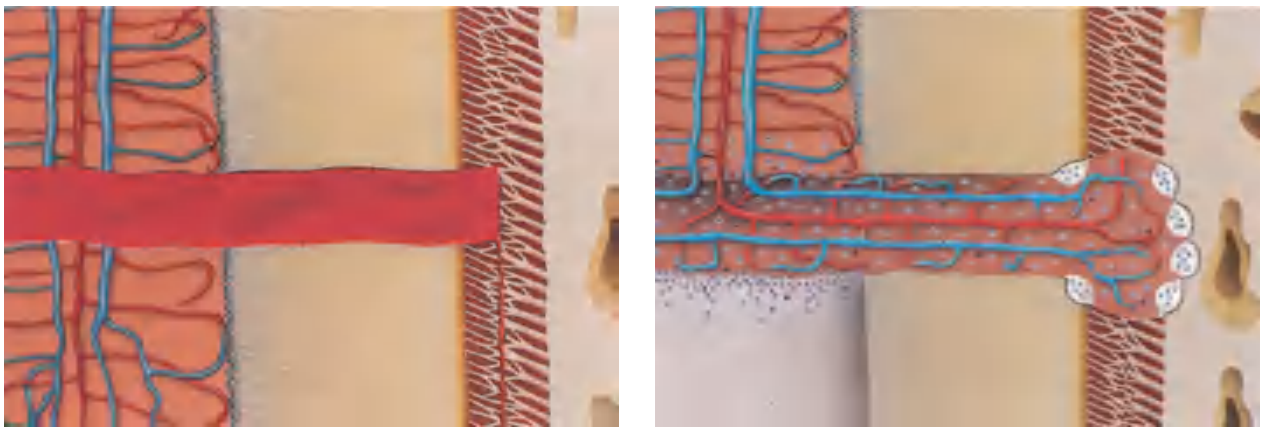


**Fig. 15.11** Non-healing due to infection in the line of fracture. Infection occurs in the avascular coronal aspect of the pulp. Granulation tissue is soon formed which originates from the apical pulp and periodontal ligament. Accumulation of granulation tissue between the two fragments causes separation of the fragments and loosening of the coronal fragment.

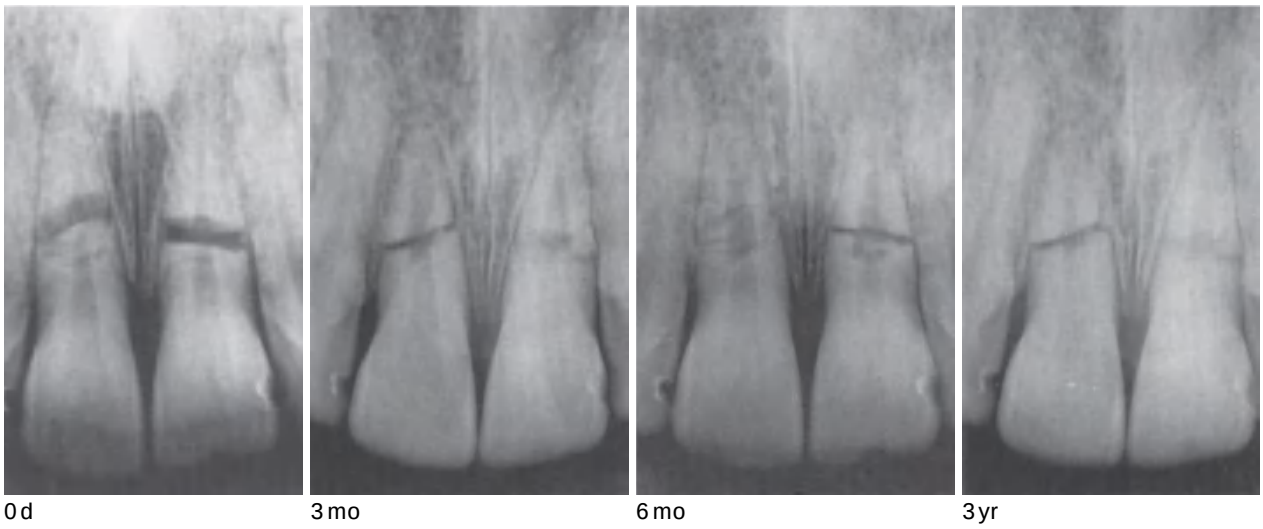



**Fig. 15.12** Root fracture of the right and left maxillary central incisors of a 22-year-old woman, with extrusion of the fragments after 3 and 6 months. There is evidence of internal surface resorption (ISR) at the junction of the pulp canal and fracture line of both teeth and external surface resorption (ESR) of the right central incisor. After 3 years, there is resolution of ISR and pulp canal obliteration of the coronal canal can be seen. Healing is by interposition of connective tissue. Reproduced from (113) with permission.

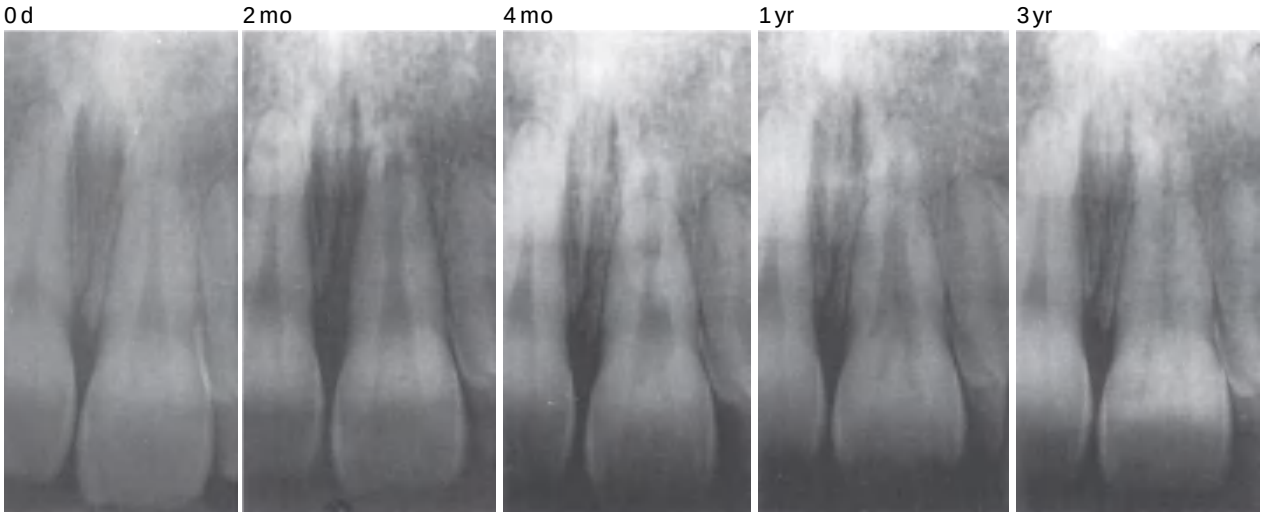



**Fig. 15.13** Root fracture of the left maxillary central incisor in a 19-year-old man with extrusion of the coronal fragment. Two months after injury, there is evidence of resorption of the pulp canal walls (internal surface resorption (ISR)). Four months after injury, simultaneous resorption and deposition of hard tissue can be seen. Internal tunneling resorption (ITR) can be clearly seen after 1 year. After 2 years, arrest of the resorption process and obliteration of the apical and coronal pulp canals can be seen. Healing is by interposition of connective tissue. Reproduced from (113) with permission.

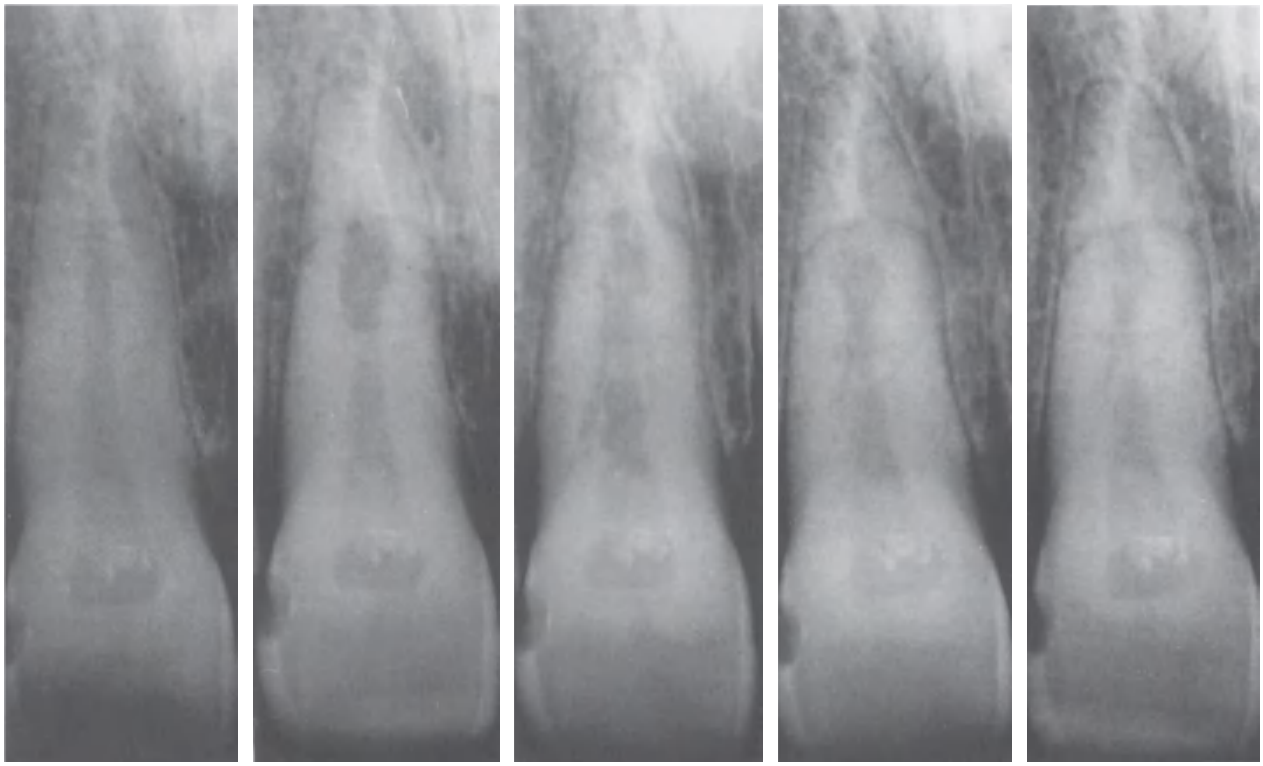



**Fig. 15.14** Root fracture of the right maxillary central incisor in a 32-year-old man with extrusion of the coronal fragment. Four months after injury, there is evidence of internal surface resorption. Just over 1 year after injury, internal tunneling resorption can be seen. After 2 years, arrest of the resorption process and obliteration of the apical and coronal pulp canals can be seen. Healing is by interposition of connective tissue. Reproduced from (113) with permission.

The resorption processes were all self-limiting, usually resolving within the first 1–2 years after injury, and therefore required no interceptive treatment. The pattern of resorption and pulp canal obliteration appeared to be decisive for fracture healing. Thus, all resorption entities collectively were significantly related to healing by interposition of connective tissue between fragments; however, when seen alone, internal surface resorption was significantly related to hard tissue union. Pulp canal obliteration in both aspects of the root canal indicated interposition of connective tissue; when seen only in the apical aspect, healing was by hard tissue union (113, 114, 175).

It should be emphasized that the pathogenesis of root fracture healing, while suggested by retrospective clinical studies (13, 76, 113), is only weakly supported by experimental research. Revision of this hypothesis could therefore be expected when future studies examine the specific roles of the pulp and periodontium in root fracture healing.

So far, radiographic and histologic observations in *human subjects* have revealed that the final outcome after root fracture can be divided into the events listed below (13, 22, 113, 114, 175) (Fig. 15.15).

## Healing with calcified tissue

A uniting callus of hard tissue has been demonstrated histologically in a number of cases (9, 13, 18, 23–32, 79–82) (Figs 15.16 and 15.17). Varying opinions exist on the nature of the hard tissue found uniting the fragments. Dentin (32), osteodentin (23) or cementum (24, 25, 29, 30) have all been found at the repair site. In most cases, the innermost layer of repair seems to be dentin (Fig. 15.17 F), while the more peripheral part of the fracture is incompletely repaired with cementum (9, 13, 26–28, 32) (Fig. 15.17E, F). The first layer of dentin is often cellular and atubular, later followed by normal tubular dentin (27, 32). Cementum deposition in the fracture line is often preceded by resorptive processes both centrally and peripherally (Fig. 15.17E). Most often, cementum will not completely bridge the gap between the fracture surfaces, but is interspersed with connective tissue originating from the periodontal ligament (Fig. 15.17G). This, combined with the greater radiodensity of cementum as compared with dentin, could explain why a fracture line is often discernible radiographically although the fragments are in close apposition and the fracture is completely consolidated (Fig. 15.18).

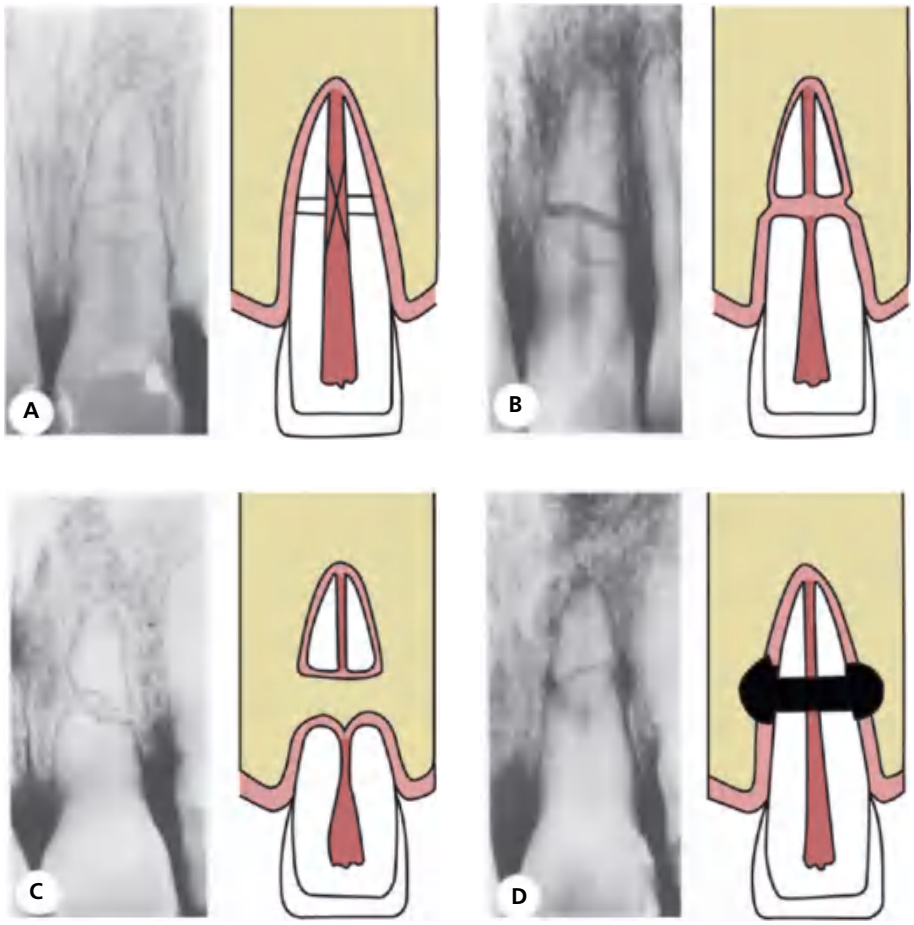



**Fig. 15.15** Radiographs and diagrams illustrating various modalities of healing after root fracture. A. Healing with calcified tissue. B. Interposition of connective tissue. C. Interposition of bone and connective tissue. D. Interposition of granulation tissue. From (13).

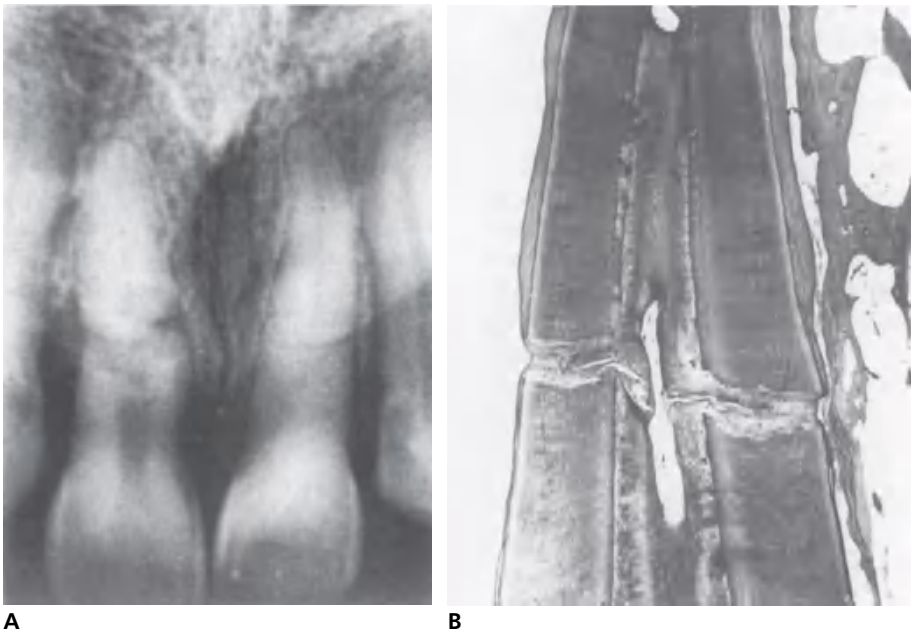



**Fig. 15.16** Complete healing with calcified tissue after root fracture. A. Radiographic condition 8 years after root fracture of both central incisors. Fracture lines are still visible. B. Histologic examination of the left central incisor reveals a complete hard tissue callus. From (32).

Occasionally, a slight widening of the root canal close to the fracture site is seen (i.e. *internal repair-related resorption* (113)), followed by hard tissue formation (83, 84) (Fig. 15.18). Moreover, it is characteristic that there is limited peripheral rounding of the fracture edges (*external surface resorption* (113)) (Fig. 15.18).

*Partial pulp canal obliteration*, confined to the apical fragment, is a frequent finding. Clinical examination of teeth

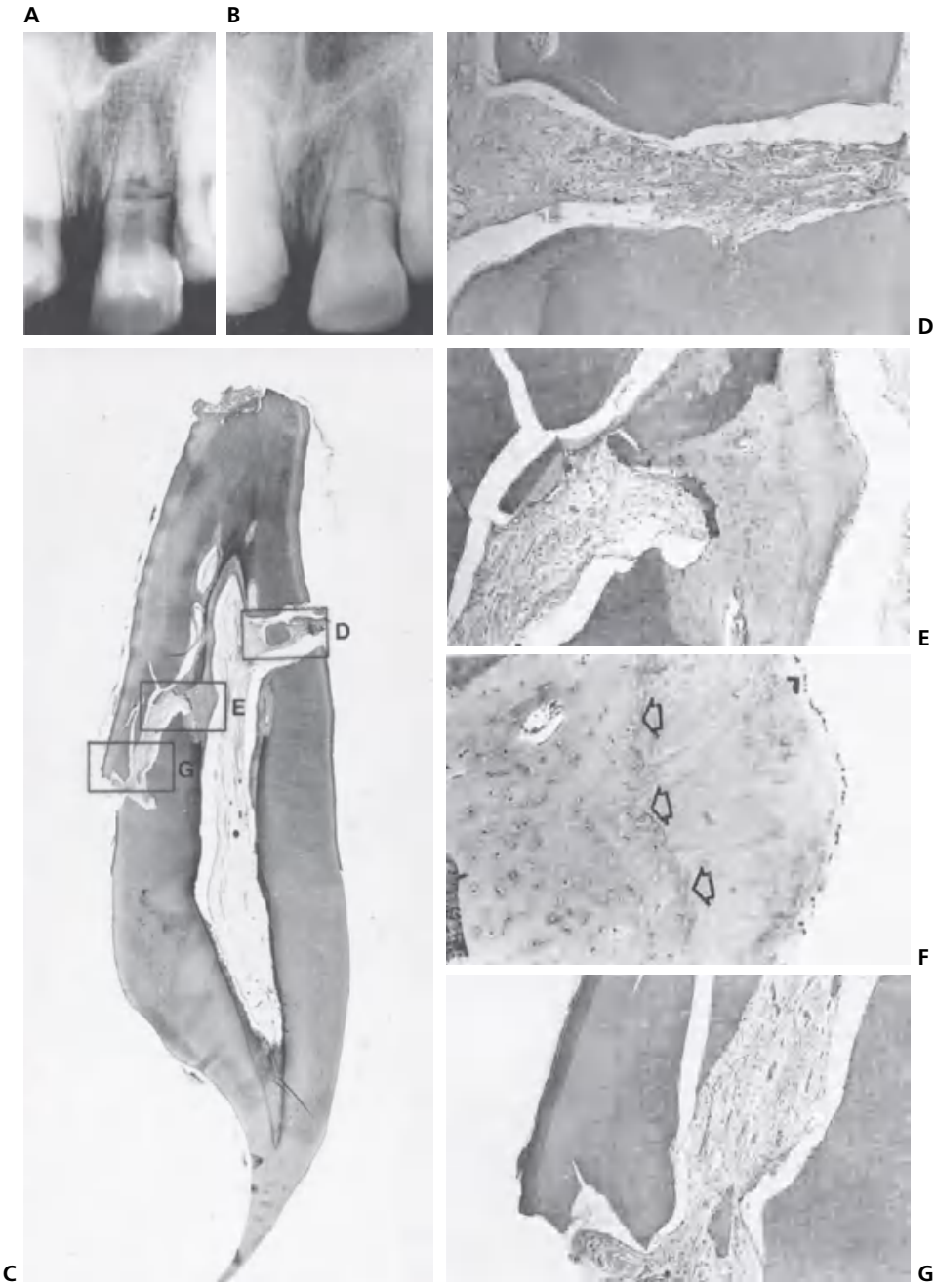



**Fig. 15.17** Incomplete healing with calcified tissue after root fracture of a left central incisor. A and B. Radiographs taken 3 and 10 months after injury. C. Low power view of sectioned incisor removed 1 year after trauma. The lingual part of the fracture line shows repair with calcified tissue. ×6. D. Interposition of connective tissue. ×30. E. Uniting callus of hard tissue. ×30. F. Higher magnification of E. There is a distinct demarcation between dentin and cementum (arrows). A few tubules are found in the dentin. The cementum is probably formed after resorption of root fragments. ×75. G. Barely visible hard tissue formation at the peripheral part of fracture. ×30. From (13).

within this healing group reveals normal mobility, as compared with non-injured adjacent teeth. Moreover, there is a normal reaction to percussion and a normal or slightly decreased response to pulpal sensibility testing (13, 85, 113).

This type of healing is dependent upon an intact pulp and is seen primarily in cases with little or no dislocation (i.e. concussion or subluxation) of the coronal fragment and most often in teeth with immature root formation (13, 73, 85, 113) (Fig. 15.19).

## Interposition of connective tissue

This type of healing is apparently related to a moderate pulpal injury (i.e. extrusion or lateral luxation of the coronal fragment), whereby pulpal revascularization and/or reinnervation must be completed prior to pulpal participation in fracture healing. In the interim, periodontal ligament cells are able to dominate the healing process (13, 115). Histologically, this is characterized by the presence of connective tissue between the fragments (13, 33, 40) (Fig. 15.20). The fracture surfaces are covered by cementum, often deposited after initial resorption (34, 35, 37, 81, 145), with connective tissue fibers running parallel to the fracture surface or from one fragment to the other. By means of secondary dentin formation, a new 'apical foramen' is created at the level of the fracture (28, 38, 113, 114, 146). A common finding is peripheral rounding of the fracture edges (*external repair-related resorption* (113)), sometimes with slight ingrowth of

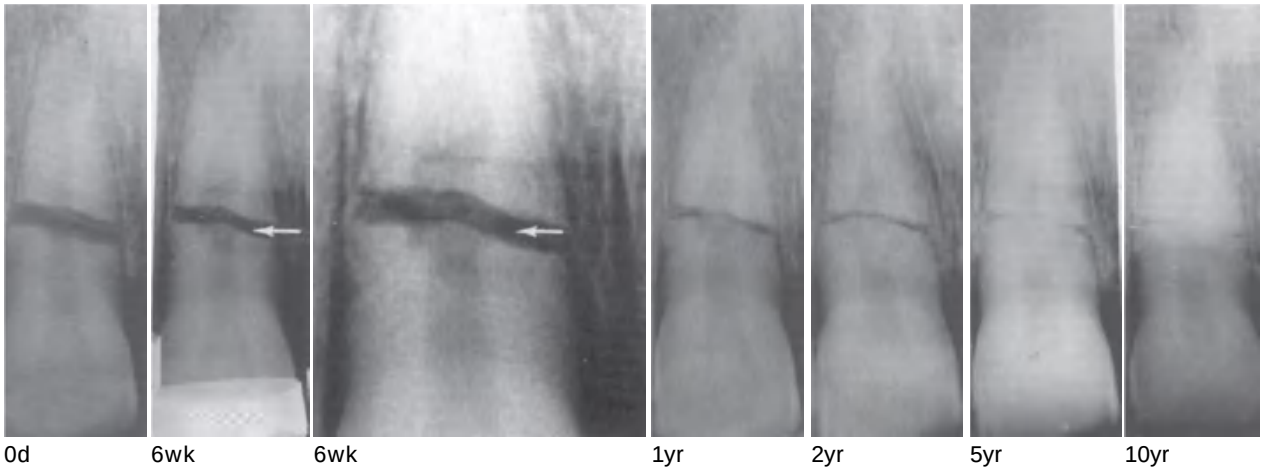
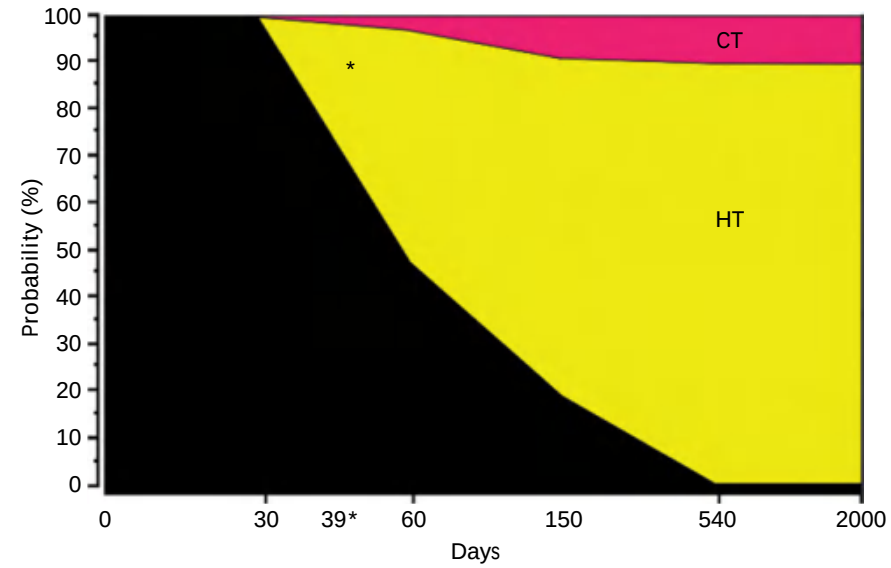



**Fig. 15.18** Healing with calcified tissue of a fracture located at the gingival third of the root of a left lateral incisor. At the 6-week control, evidence of widening of the root canal close to the fracture site (arrow) is apparent. At controls 1 and 2 years later, the resorption cavity in the pulp canal as well as the fracture site show repair with calcified tissue. After 5 and 10 years, the fracture site is almost occluded with hard tissue. Reproduced from (115) with permission.



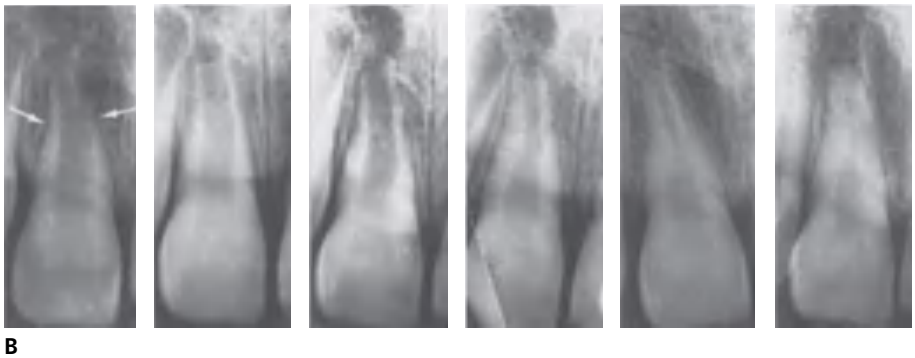




**Fig. 15.19** A. Probability of the various healing modalities after root fracture of the maxillary right central incisor (arrows in B) in an 8-year-old boy based on the following information from the time of injury: the coronal fragment was subluxated, with first degree loosening; the diameter of the apical foramen was 2.8 mm; antibiotics were administered due to avulsion and replantation of the maxillary left central incisor. Splinting was with an acid-etch splint. The times on the horizontal axis of the probability curves are the intervals defined in the statistical analysis. The times given on the radiographs are the exact observation periods, which correspond to the defined intervals. CT, connective tissue; HT, hard tissue. B. Radiographic appearances at the various observation periods. At 39 days, the diagnosis of hard tissue union was made (asterisk). Reproduced from (115) with permission.

bone into the fracture area from the lateral side (13, 23) (Fig. 15.18). The width of the periodontal space around the fragments reflects the functional activity of the two fragments. The periodontal space surrounding the apical fragment is narrow, with fibers oriented parallel to the root surface, while the space around the coronal fragment is wide, with normal fiber arrangement (33, 36).

The radiographic features in this type of healing consist of peripheral rounding of the fracture edges and a radiolucent line separating the fragments (Fig. 15.15B). Initially, *external*

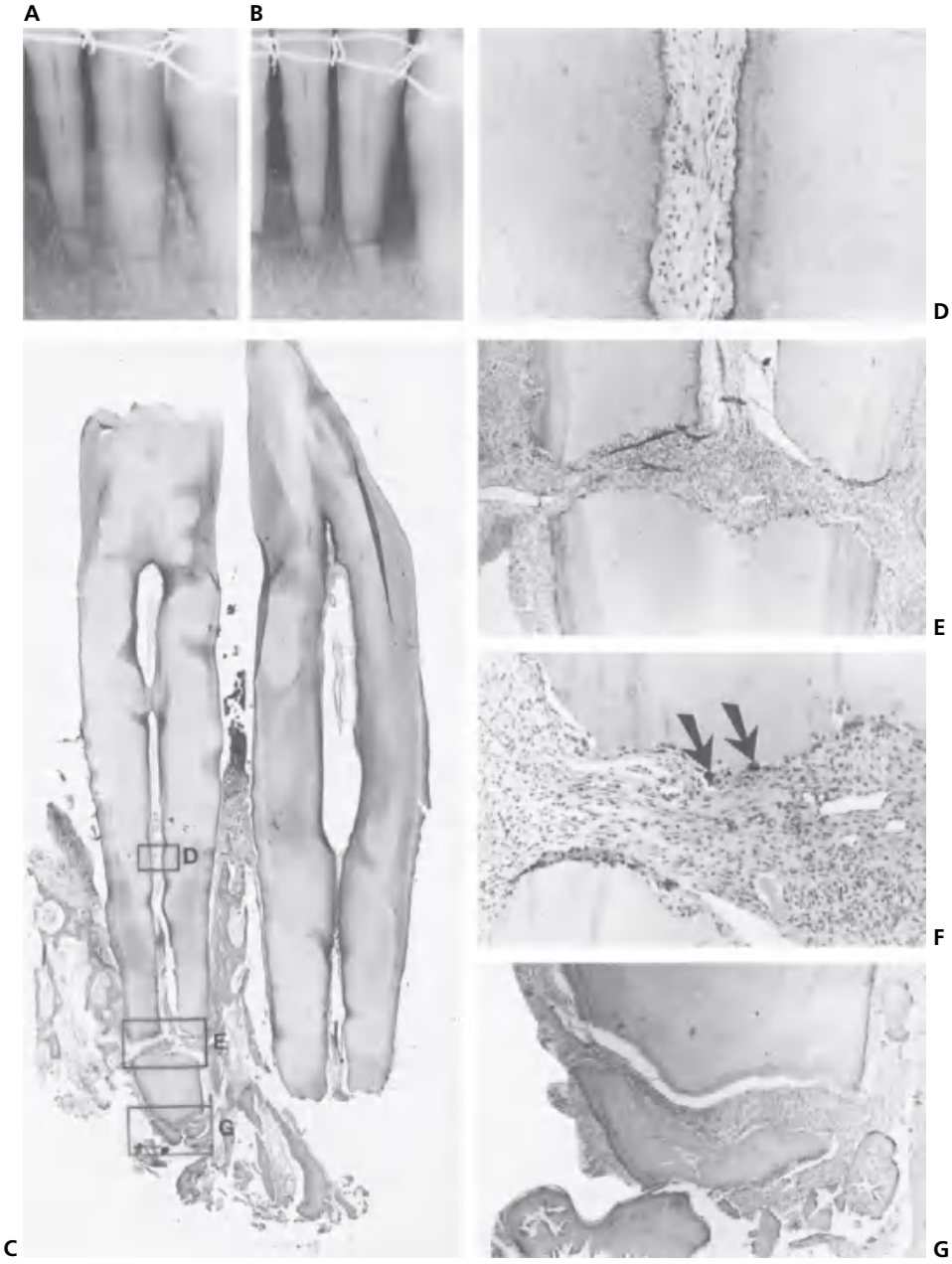



**Fig. 15.20** Interposition of connective tissue after root fracture of left central and lateral incisors. A. One week after injury. B. Radiograph at time of removal 6 weeks after injury. C. Low power view of sectioned central incisor shows interposition of connective tissue. ×30. D. Vital pulp tissue in crown portion. ×75. E. Connective tissue in fracture line. ×30. F. Higher magnification of E. Note active resorption on surface of fragments (arrows). ×75. G. Comminuted fracture in apical region. Note formation of cementum on fragments. ×30. From (13).

and *internal repair-related resorption* are often seen (113), as well as *pulp canal obliteration* of both the apical and coronal aspects of the root canal (115).

Clinically, the teeth are normally firm or slightly mobile and with a weak pain response to percussion. The response to sensibility testing is usually within the normal range (13, 76, 113, 114). Although no interceptive therapy is required at the appearance of the above-mentioned resorption processes, it should be mentioned that a few cases have been observed whereby the post-resorptive mineralization process resulted in *ankylosis*. Thus, longer observation periods as well as more frequent follow-up examinations during the 1st year after injury might be advisable when these resorption processes are diagnosed (see Appendix 3, p. 1024).

## Interposition of bone and connective tissue

Histologically, teeth in this healing group demonstrate interposition of a bony bridge and connective tissue between the apical and coronal fragments, with a normal periodontal ligament surrounding both fragments (23, 41–45). In some instances, bone can be seen extending into the root canals (41–43) (Fig. 15.21).

This mode of healing is apparently a result of trauma prior to completed growth of the alveolar process, thus the coronal fragment continues to erupt, while the apical fragment remains stationary in the jaw (13) (Figs 15.22 and 15.23).

Radiographically, a bony bridge is seen separating the fragments, with a periodontal space around both fragments.

Total *pulp canal obliteration* of the root canals in both fragments is a common finding (Fig. 15.15C).

Clinically, the teeth are firm and react normally to pulp tests.

It should be noted that the same radiographic picture could be seen after orthodontic movement of a root-fractured tooth which has healed by interposition of connective tissue, as only the coronal fragment would be moved in contrast to movement of the entire root after healing by calcified tissue.

## Interposition of granulation tissue

Histologic examination of teeth in this group reveals inflamed granulation tissue between the fragments (13, 33, 45–49, 119) (Figs 15.24 and 15.25). The coronal portion of the pulp is necrotic and infected, while the apical fragment usually contains vital pulp tissue (13) (Fig 15.24D, G). The necrotic and infected pulp tissue is responsible for the inflammatory changes along the fracture line (13). In some cases, however, communication between the fracture line and the gingival crevice is the source of inflammation (13, 39, 49) (Fig. 15.25).

Radiographically, widening of the fracture line, loss of lamina dura and rarefaction of the alveolar bone

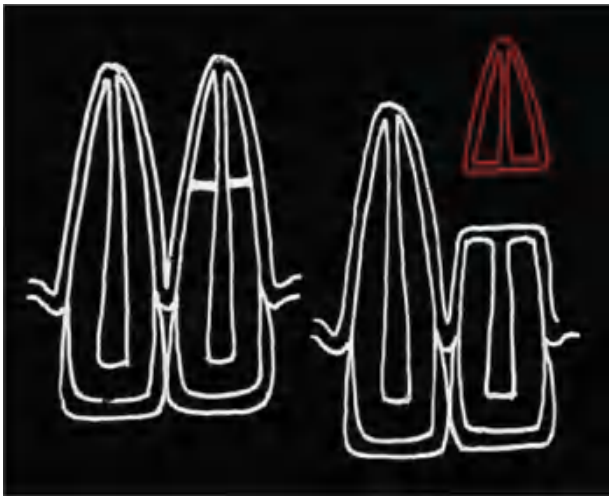


**Fig. 15.21** Growth of the alveolar process leaves the apical fragment behind in its original position.

**Fig. 15.22** Interposition of bone and connective tissue between fragments of a permanent incisor removed 6 months after root fracture. Note extension of bone into the pulp canal. ×7. Reproduced from (41) with permission.

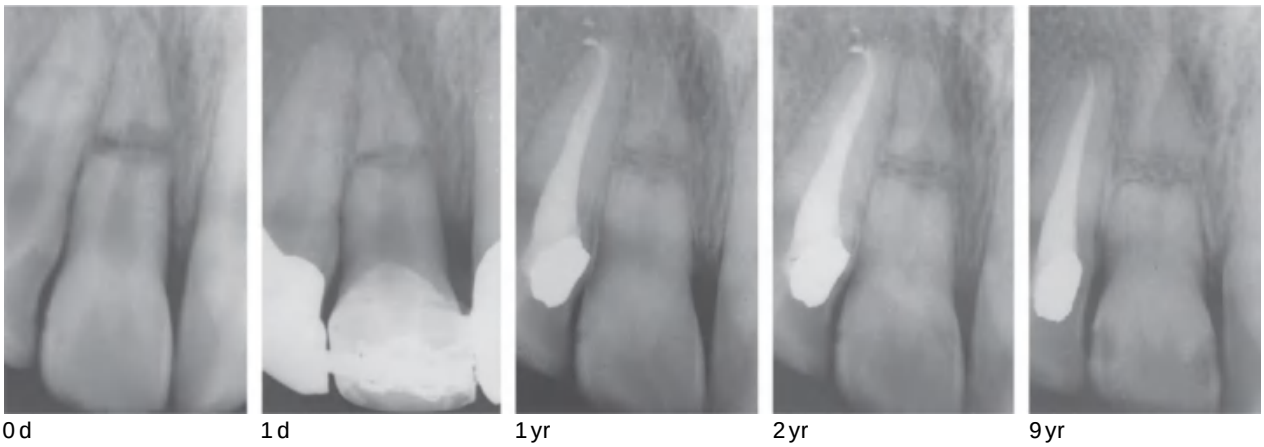



**Fig. 15.23** Healing of fracture with interposition of bone and periodontal ligament. On the day of injury, there is a fracture with diastasis of about 1.8 mm and less than optimal splinting. At controls after 1, 2 and 9 years, respectively, there is ingrowth of bone between the two fragments, further eruption of the coronal fragment and pulp canal obliteration in both fragments. Reproduced from (154) with permission.

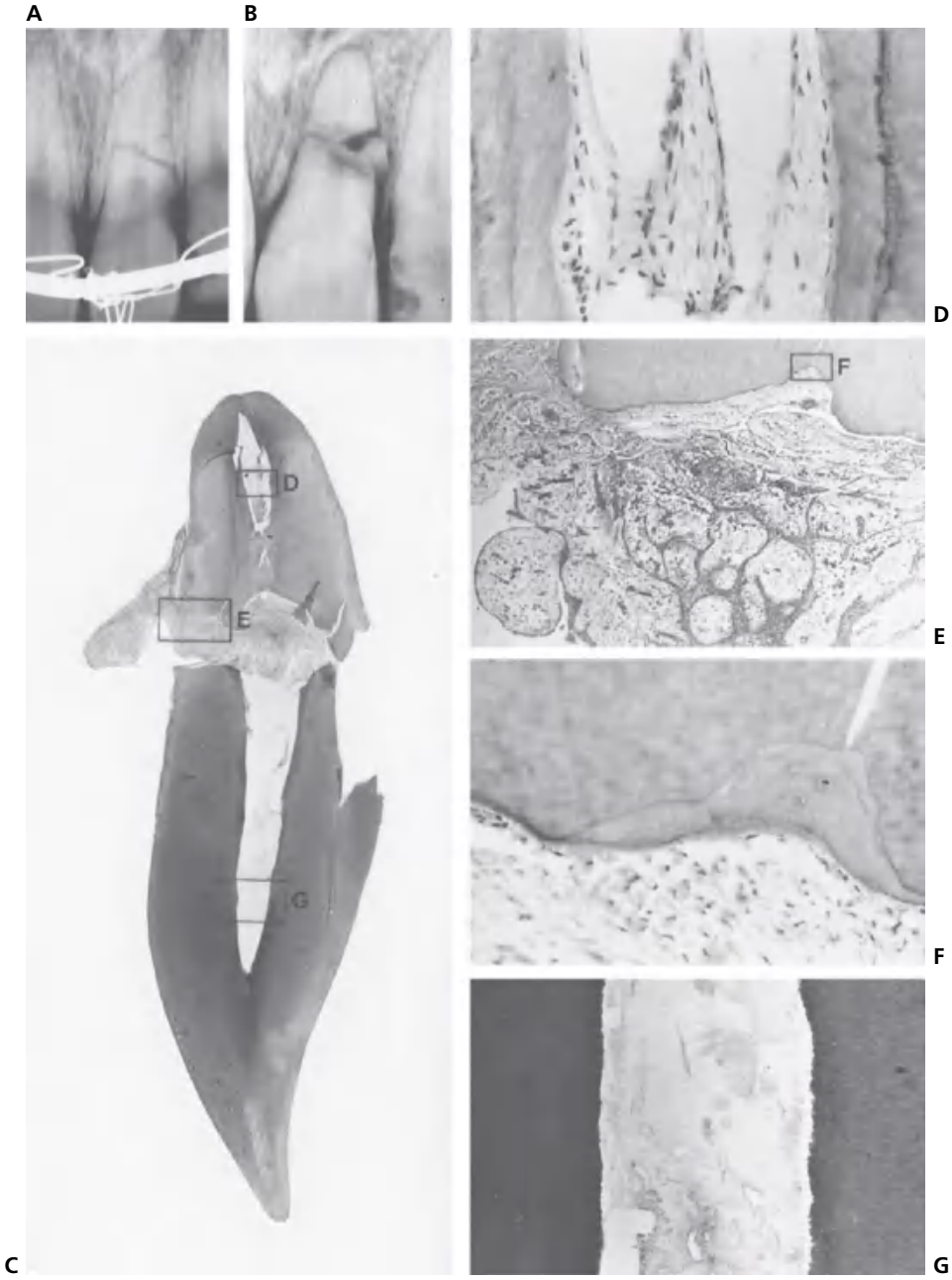



**Fig. 15.24** Non-healing with interposition of granulation tissue between fragments of a left central incisor. A. Immediately after injury. B. Radiographs at the time of removal 6 months after injury. C. Low power view of sectioned incisor. ×6. D. Vital pulp tissue in apical fragment. ×75. E. Granulation tissue with epithelial proliferation and chronic inflammation. ×30. F. Higher magnification of E. Area with newly formed cementum. ×195. G. Necrotic pulp tissue in coronal fragment. ×30. From (13).

corresponding to the fracture line are typical findings (120) (Fig. 15.15D). If the tooth is not splinted, the coronal fragment is loose, slightly extruded and sensitive to percussion. If splinted, the apical fragment becomes displaced in an apical direction. Fistulae at a level on the buccal mucosa corresponding to the fracture line are an occasional finding (13).

It was found that negative pulpal sensibility at the time of injury was significantly related to later pulp necrosis, presumably a reflection of severe pulpal injury. However, a positive pulpal response was not able to predict healing by hard tissue union or interposition of connective tissue (115). Moreover, splinting by the forceful application of orthodontic bands (a splinting technique employed prior to the advent of the passively applied acid-etch splints) (115) or cap splints (147) played an important role in the development of coronal pulp necrosis following root fracture. This adverse effect was presumably due to an additional trauma, including possible dislocation of the coronal fragment, during splint application (147).

## Treatment

The principles of treating *permanent teeth* are reduction of displaced coronal fragments and immobilization (Fig. 15.26). Surveys of treatment principles have been published (148, 149). If treatment is instituted immediately after injury, repositioning of the fragment by digital manipulation is easily

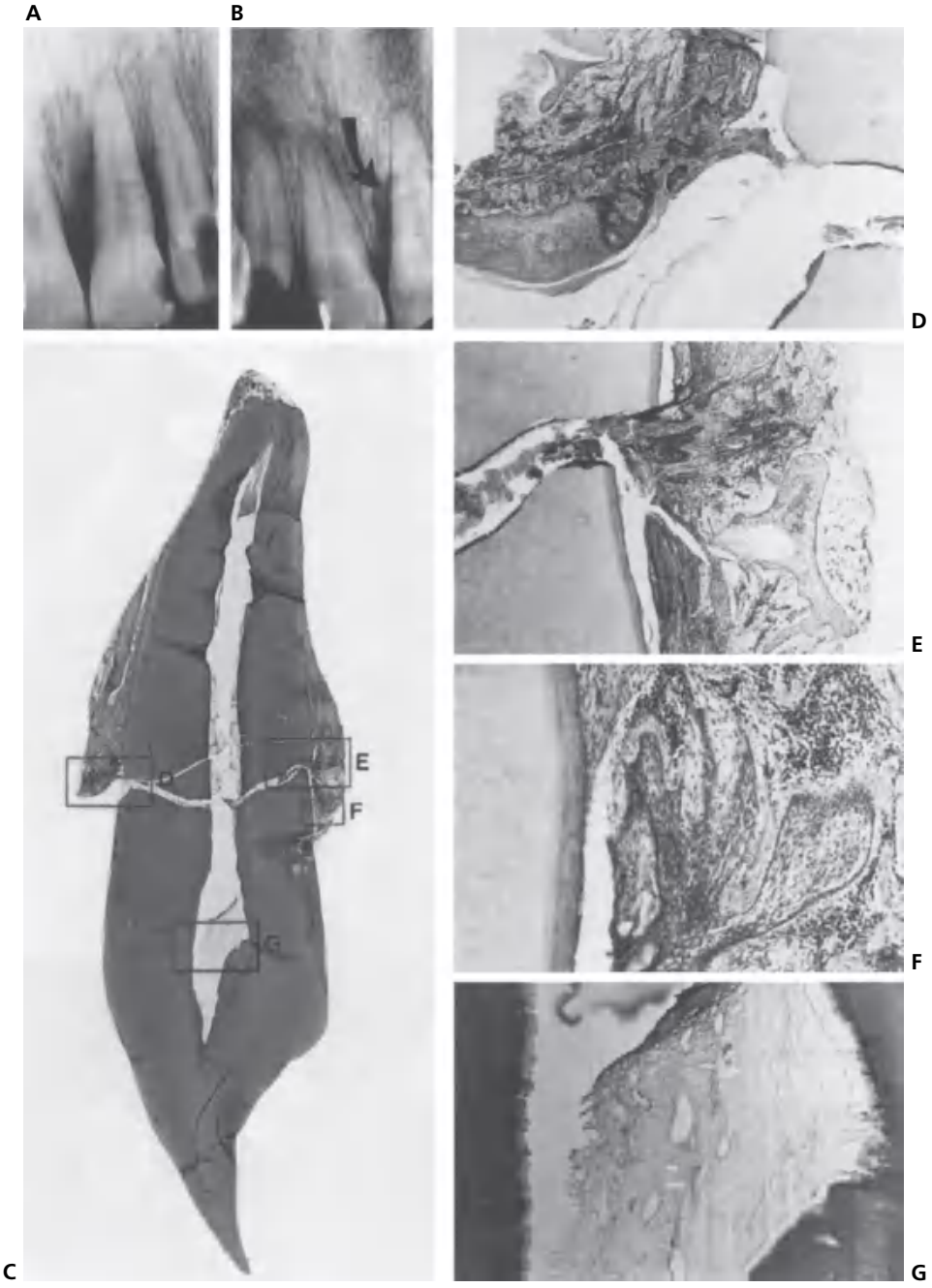



**Fig. 15.25** Non-healing with interposition of granulation tissue between fragments of a left central incisor. A and B. Radiographs taken 2 months after trauma. Note the periradicular radiolucency adjacent to the fracture line (arrow). C. Low power view of sectioned incisor. The tooth was removed 2 months after injury. ×6. D. Downward proliferation of epithelium from gingival crevice. ×75. E and F. Palatal fracture with epithelial proliferation. ×30 and ×75. G. Necrotic pulp tissue in coronal fragment. ×30. From (13).

achieved (Fig. 15.27). If resistance is felt upon repositioning, it is most likely due to fracture of the labial socket wall. In this case, repositioning of the fractured bone is necessary before further attempts are made to reduce the root fracture (Fig. 15.28). After reduction, the position should be checked radiographically.

Immobilization of teeth with root fractures is achieved with semi-rigid fixation, e.g. an acid-etch/resin splint (see Chapter 39) (147). As previously mentioned, forceful application of, for example, orthodontic bands for the purpose of immobilizing the coronal fragment is contraindicated due to its traumatic influence on the already traumatized pulp, which could result in pulp necrosis. A passively applied splint (e.g. using the acid-etch technique) is advised. The fixation period should be about 1 month to ensure sufficient hard tissue consolidation; longer splinting periods have not been found to be of value (147).

However, it should be noted that a new trauma to a tooth with a successfully healed root fracture will heal less optimally the second time around. Therefore, in active individuals, a palatally bonded retention wire might be an advantage.

It should be noted that immature teeth with incomplete root fractures require no fixation and will heal by hard tissue union (115). However, these teeth may be included in a splint if multiple tooth injuries require this. See Appendix 4 (p. 1025) for a suggested schedule for recall examinations.

If the fracture is located at the cervical third of the root and below the alveolar crest, various studies have shown that healing is possible and a conservative approach justified (13, 22, 50, 154) (Figs 15.29 and 15.30). In cases where oral hygiene is optimal, treatment can be permanent fixation of the coronal fragment to adjacent non-injured teeth with a lingual orthodontic retention wire (Fig. 15.30). However, very

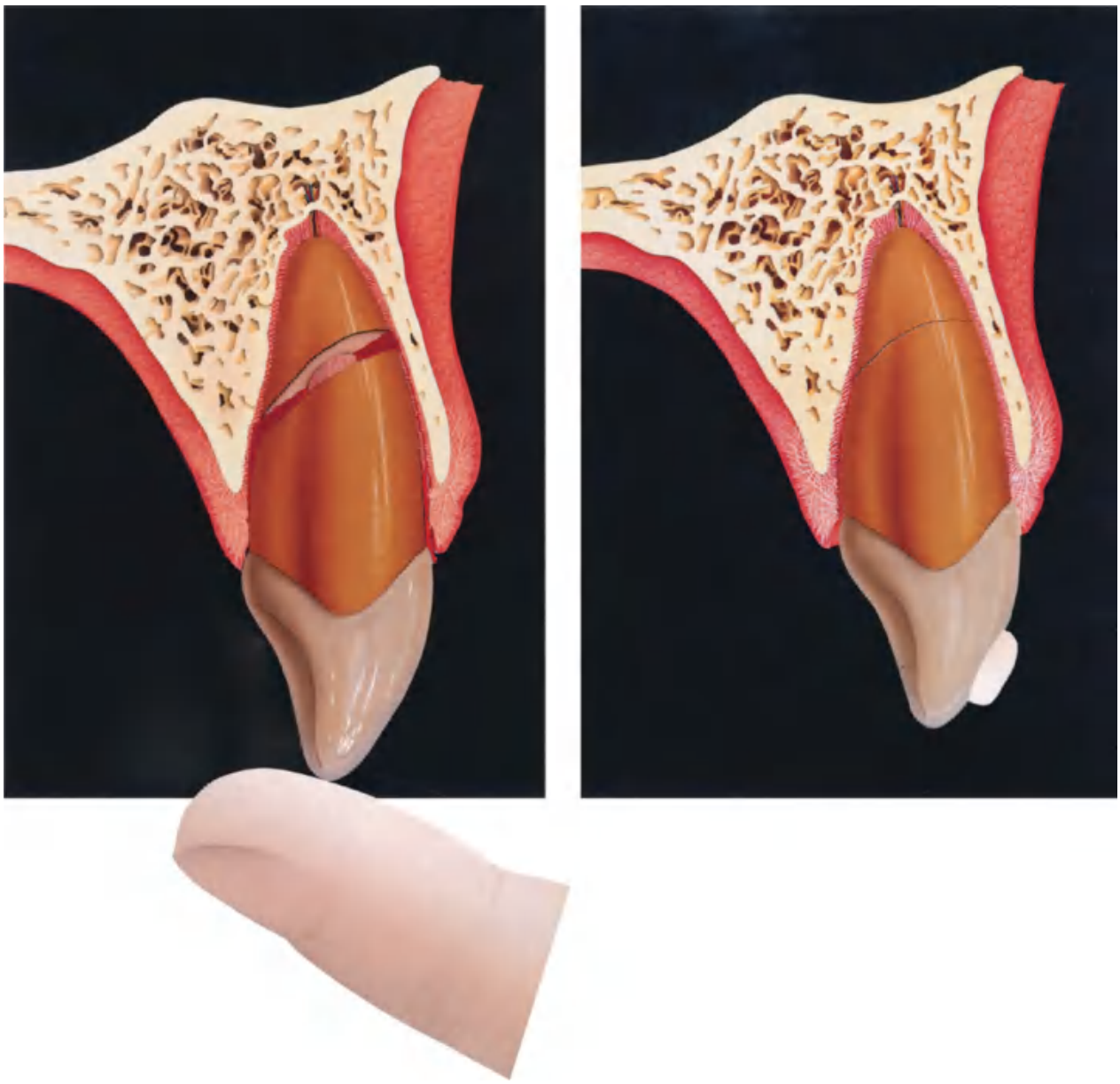


**Fig. 15.26** Principles for treatment of root fractures. Treatment of root fractures consists of complete repositioning and firm splinting, preferably with a passively applied splint until a hard tissue callus is formed, usually after 3 months.

close proximity of the root fracture to the gingival crevice may dictate alternative treatment, as the chance of healing with calcified tissue is poorest when a cervical fracture line is close to or communicating with the gingival crevice. A treatment option which could then be considered is the removal of the coronal fragment and subsequent orthodontic (87) or surgical extrusion of the remaining apical fragment. These techniques are described in more detail in relation to treatment of complicated crown-root fractures (see Chapters 11 and 24).

In cases where it is not possible to treat the fractured tooth conservatively and the tooth must be extracted, it should be remembered that careless extraction procedures will result in extensive damage to the alveolar process and subsequent severe atrophy, especially in the labio-lingual direction, ultimately resulting in a compromised esthetic restorative treatment. This problem can be prevented to a certain degree by careful removal of the apical fragment with no or minimal sacrifice of labial bone. Thus, if removal of the apical fragment is indicated, but not possible via the socket, surgical removal should be undertaken by raising a flap and making an osteotomy over the apical area, thereby pushing the apex out of its socket. Under no circumstance should the marginal socket wall be removed, as this can lead to labio-lingual collapse of the alveolar process (Fig. 15.31). In growing individuals it may be sensible to keep the apical fragment *in situ* as a decoronation procedure (see Chapter 32).

A preferred alternative to the above situation is the preservation of the apical fragment, which normally contains vital pulp tissue. Experimental evidence seems to indicate that intentionally submerged root fragments with vital pulps prevent or retard resorption of the alveolar process. These roots are usually covered along the fractured surface with a new layer of cementum as well as a thin layer of new bone. Moreover, the

**Fig. 15.27 Treatment of a laterally luxated root fracture**
This 13-year-old boy received a horizontal blow to the left maxillary central incisor.

**Examining the tooth**
The tooth reacts to *sensitivity testing*, indicating an intact vascular supply. There is a high metallic sound elicited by the *percussion test*, indicating lateral luxation of the coronal fragment. The tooth is locked firmly in its displaced position, confirming the lateral luxation diagnosis.

**Repositioning**
Because of the force necessary to reposition a laterally luxated tooth, local anesthesia is administered prior to repositioning. Firm pressure is applied to the facial bone plate at the fracture level in order to displace the coronal fragment out of its alveolar 'lock'. This is followed by horizontal (forward) pressure at the palatal aspect of the incisal edge, which repositions the coronal fragment into its original position.

**Verifying repositioning**
The correct position of the coronal fragment is confirmed radiographically.

**Splinting procedure**
The acid-etch technique and a temporary crown and bridge material are used. Phosphoric acid gel is applied for 30 seconds to the facial surfaces of the injured and adjacent non-injured teeth. The tooth surfaces are then rinsed thoroughly with a stream of water for 20–30 seconds and blown dry. A mat white enamel surface indicates adequate etching.

**Applying the splinting material**
Temporary crown and bridge material (Luxatemp®) is applied to the etched enamel surfaces. The bulk of the material should be placed on the mesial and distal corners of the traumatized and adjacent, non-injured incisors. After setting, any irregularity in the surface is removed with abrasive disks or a scalpel blade.

**Removing the splint**
The healing events are monitored radiographically. If the tooth reacts to sensibility testing and there is no sign of infection in the bone at the level of the fracture, the splint may be removed.

**One year after injury**
The tooth responds normally to sensibility testing and the radiograph shows increasing radiopacity of the fracture line.

**Fig. 15.28 Treatment of a severely extruded root fracture**
This 20-year-old woman has suffered a frontal blow to the left central incisor, resulting in extreme displacement of the coronal fragment.

**Examining the displaced coronal fragment**
After cleansing the exposed root surface with saline, it can be seen that the coronal fragment has been forced past the cervical margin of the labial bone plate. The stretched and displaced pulp is seen within the socket area.

**Repositioning**
After local anesthetic infiltration, the coronal fragment is repositioned. To guide the root fragment into place, an amalgam carver is inserted beneath the cervical bone margin and used like a shoe horn.

**Splinting**
The acid-etch technique is employed once optimal repositioning has been verified radiographically. The labial surfaces are etched and a flexible splinting material is applied.

**Fig. 15.29** Hard tissue healing of a root fracture located at the marginal third of the root of a left incisor.

**Fig. 15.30** Permanent splinting of the coronal fragment of a root-fractured lateral incisor healed with interposition of connective tissue. Filled composite resin is placed at the contact areas proximally. Radiograph shows 10-year follow-up.

**Fig. 15.31** Atrophy of the alveolar process after surgical removal of a root-fractured tooth. A. Condition after trauma. The right central incisor shows a root fracture close to the gingival crevice. The tooth was surgically removed. During this procedure the labial bone plate was also removed. B and C. Marked atrophy of the alveolar process is evident 1 year later.

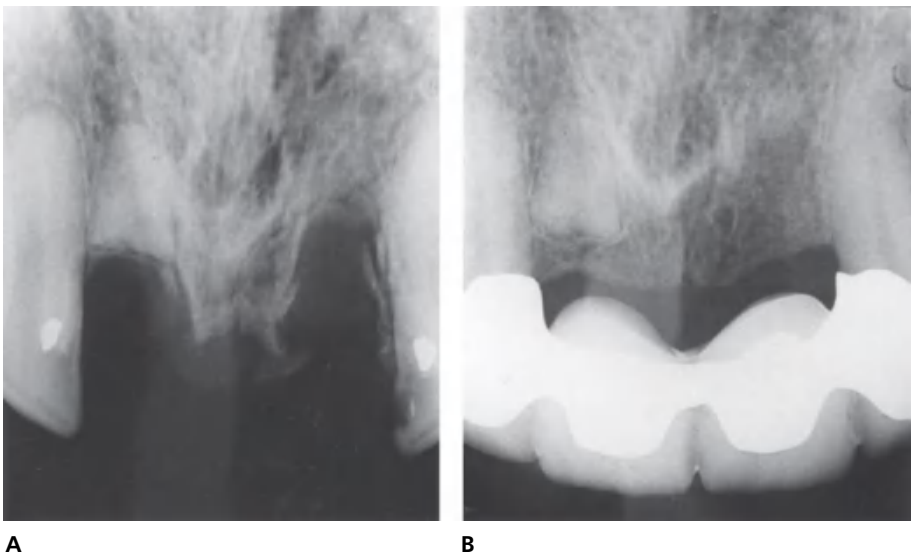


**Fig. 15.32** Apical fragment left *in situ* after root fracture. The two central incisors were avulsed. A. Condition at the time of injury. B. At a follow-up 5 years after injury, the pulp canal in the apical fragment shows complete obliteration and bone covers the fractured surface of the root.

pulp retains its vitality (88–90). Preliminary clinical experience seems to indicate that an intentionally buried apical fragment resulting from root fracture acts similarly (Fig. 15.32). This is presumably analogous to the submergence of the apical fragment which is seen after healing by interposition of bone and connective tissue. Whether this treatment procedure is reliable must await the results of long-term clinical studies.

## Prognosis

Several clinical reports have demonstrated successful treatment of root fractures (13, 22, 33, 51–63, 73–75, 82, 85, 91, 92, 112, 115, 121–123, 143, 147). However, follow-up examinations can disclose deviations in pulpal and periodontal healing. In this context, radiographic findings, such as *pulp canal obliteration* or *external and internal repair-related resorption*, have been found to be related to specific healing modalities (113, 114, 175).

## Follow-up

A standard follow-up strategy using clinical information based on sensibility testing should be used (see Chapter 9). In this regard, a laser Doppler study has shown that this approach could be advantageous in demonstrating early revascularization of the coronal pulp (151). Furthermore, radiographic examination should be included using standardized exposures (see Chapter 12).

### Pulpal healing

Clinical experience has shown that the pulp is more likely to survive after root fracture than after luxation with no fracture of the root (36, 53, 57, 64–66, 114) (Fig. 15.33). An explanation could be that the fate of the injured pulp depends in part upon revascularization from the periodontal ligament. Such revascularization in luxation injuries is limited to the periapical tissues through the apical foramen, whereas a fractured root offers broader communication from the pulp canal to the periodontal tissues, facilitating re-establishment of the blood supply. Moreover, if one assumes that only the coronal aspect of the pulp has been damaged, the extent of tissue requiring revascularization is considerably reduced following root fracture than after luxation injuries. Another important factor could be the development of pulpal edema, which can escape through the fracture, minimizing pressure upon the delicate pulpal vessels. Furthermore, the root fracture itself could prevent transmission of the impact to the apical area, thus reducing damage to the vulnerable area at the constricted apical foramen (57).

An interesting finding has been that a transient discoloration ranging from gray to brown-rose may occur in about 20% of root-fractured teeth. This discoloration persisted from 4 to 8 weeks and was generally followed by healing with interposition of connective tissue or connective tissue and bone (171).

Close clinical and radiographic follow-ups are necessary to disclose pulp necrosis (120). Extrusion of the coronal fragment, tenderness to percussion and, most significantly, radiographic signs of pulp necrosis, such as the development of radiolucencies within bone adjacent to the fracture line (Figs 15.34 and 15.35), can usually be detected within the first 2 months after injury.

A negative sensibility response immediately after injury does not necessarily indicate pulp necrosis, as a slow return to normal sensibility is often observed (85). However, it has been found that teeth that did not respond to pulp testing immediately following root fracture demonstrated a significantly greater risk of later pulp necrosis than those that responded positively at the time of injury (115, 143, 147). The diagnosis of pulp necrosis should therefore always be based upon a combined clinical and radiographic evaluation.

Table 15.1 lists various reports on the prevalence of pulp necrosis after root fracture. Factors related to healing events (hard tissue, connective tissue and non-healing) can be divided into *pre-injury* and *injury factors* and *treatment-dependent factors* (143, 147).

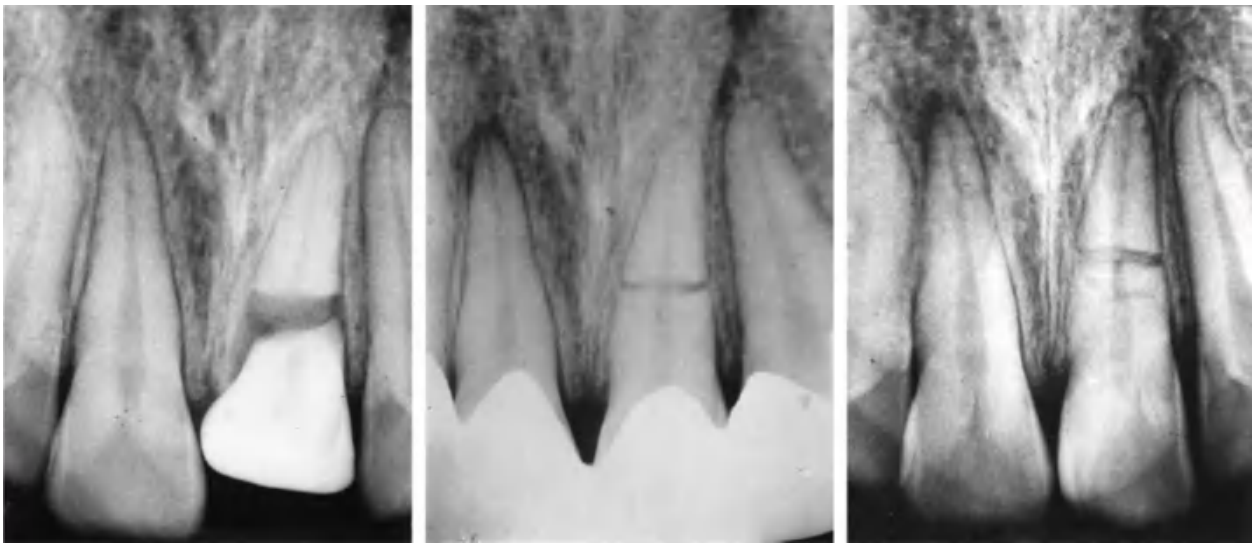


**Fig. 15.33** Pulp necrosis and periapical involvement after subluxation and pulp survival after root fracture. The left central incisor showed root fracture and dislocation while the right central incisor was subluxated. The subluxated tooth has developed pulp necrosis but the tooth with the root fracture has remained vital.

**Fig. 15.34** Radiographic demonstration of pulp necrosis after root fracture. The enlargement of the periodontal space adjacent to the fracture line indicates pulp necrosis (arrows).

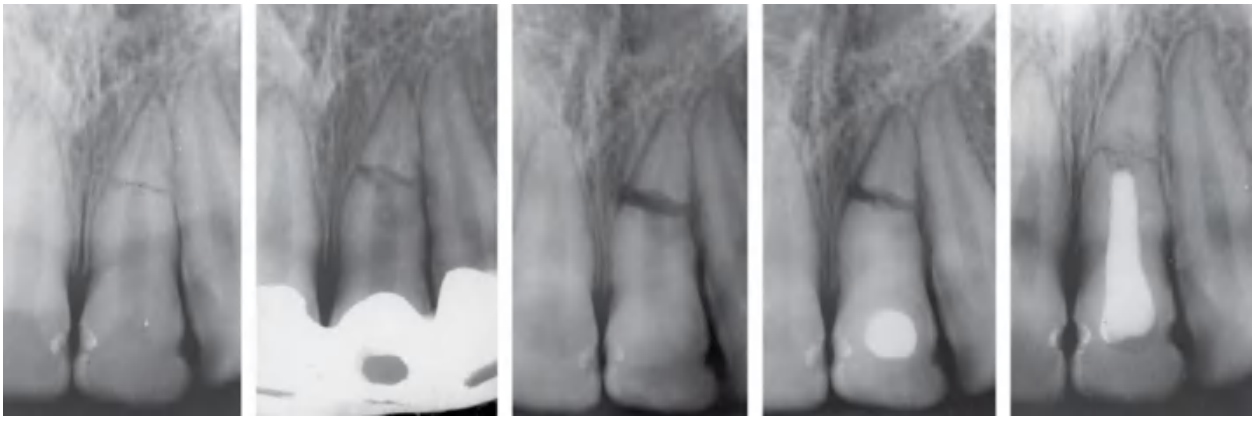



**Fig. 15.35** Non-healing after root fracture. The root fracture was splinted on the day of injury. At control 3 months after injury a periradicular radiolucency is seen adjacent to the fracture, and there is no reaction to sensibility testing and there is discoloration of the crown. Treatment of the coronal fragment is instituted with calcium hydroxide. Control after 6 years shows periodontal healing after treatment and filling of the root canal in the coronal fragment with gutta-percha. Reproduced from (154) with permission.

### *Pre-injury and injury factors*

The pre-injury or injury factors which had the greatest influence upon healing (i.e. whether hard tissue fusion or pulp necrosis) were *age* (143), *stage of root development* (i.e. the size of the pulpal lumen at the fracture site) (73, 76, 115, 143, 147, 154), *mobility of the coronal fragment* (143), *dislocation of the coronal fragment* (13, 76, 115, 143) and *diastasis between fragments* (i.e. rupture or stretching of the pulp at the fracture site) (143) (Fig. 15.36).

It has previously been supposed that root fractures located at the marginal one-third of the root had a very poor long-term prognosis, thus indicating extraction therapy. However, this assumption has not been supported by studies where no relationship could be demonstrated between the frequency of pulp necrosis and the position of the fracture line (13, 73, 76, 77, 85, 154, 155).

**Table 15.1** Frequency of pulp necrosis after root fracture of permanent teeth.

| Study | No. of teeth | Pulp necrosis |
|---|---|---|
| Austin (12) 1930 | 40 | 8 (20%) |
| Doniau & Werelds (10) 1955 | 24 | 5 (21%) |
| Lindahl (11) 1958 | 25 | 6 (24%) |
| Andreasen & Hjörting-Hansen (13) 1967 | 48 | 21 (44%) |
| Stålhane & Hedegård (92) 1975 | 18 | 4 (18%) |
| Zachrisson & Jacobsen (76) 1975 | 64 | 13 (20%) |
| Ravn (77) 1976 | 50 | 11 (22%) |
| Krenkel et al. (150) 1985 | 26 | 1 (4%) |
| Andreasen et al. (115) 1989 | 95 | 25 (26%) |
| Yates (141) 1992 | 22 | 5 (23%) |
| Caliskan & Pehlivan (152) 1996 | 56 | 21 (37%) |
| Majorana et al. (139) 2002 | 31 | 13 (40%) |
| Welbury et al. (153) 2002 | 84 | 46 (55%) |
| Feely et al. (65) 2003 | 34 | 7 (21%) |
| Andreasen et al. (143, 147) 2004 | 400 | 88 (22%) |

In this regard, it should be considered that these teeth are at risk of a new trauma which easily could lead to loss of the coronal fragment, or excessive mobility being an indication for extraction (153, 154) (Fig. 15.37). In such situations it is important to consider whether the fracture is strictly horizontal or oblique and thus involving both the cervical or middle thirds of the root (Fig. 15.38). In a more recent long-term study of 94 cervical root fractures, 44% were lost. In comparison, only 8% horizontal and oblique fractures were lost when located mid-root (154).

Other factors that have been found to be predictive of the type of fracture healing have included the presence of *restorations* at the time of injury as well as the presence of *marginal periodontitis* (115). Thus, if either of these factors were present, there was a significantly greater risk of healing by interposition of connective tissue than healing by hard tissue union. It was impossible to determine whether this finding reflected an age phenomenon or reduced pulpal defense due to disease or operative procedures (115).

### *Treatment factors*

Concerning treatment factors, the following had a negative influence upon healing: *forceful application of splints* (115) and *type of splint*, rigid splints giving the poorest results (147). The length of the splinting period appeared to be of no significance; and 4 weeks appeared to be acceptable (147). Furthermore, *treatment delay* of a few days did not have a negative effect on healing (147). Finally, *antibiotics* did not improve healing; in fact an unexplained inverse relation to healing was found (115, 147).

Many types of treatment have been proposed for the management of pulp necrosis in root-fractured teeth (22, 156) (Fig. 15.39). The most important feature to consider is that the apical fragment normally contains vital pulp tissue (13). This is the basis for treatment where only the coronal fragment is

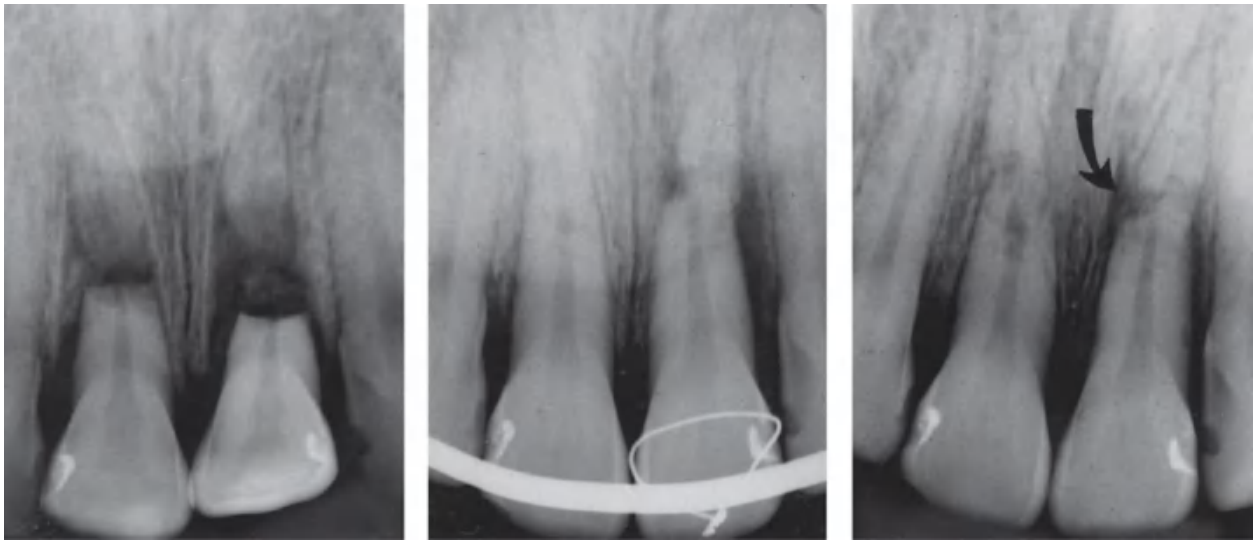


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**Fig. 15.36** Dislocation of a root fracture leading to pulp necrosis. Note that the left maxillary incisor is dislocated more than the right incisor. Three months later, the right central incisor was found to be vital whereas pulp necrosis and periapical rarefaction was diagnosed in the left central incisor (arrow).

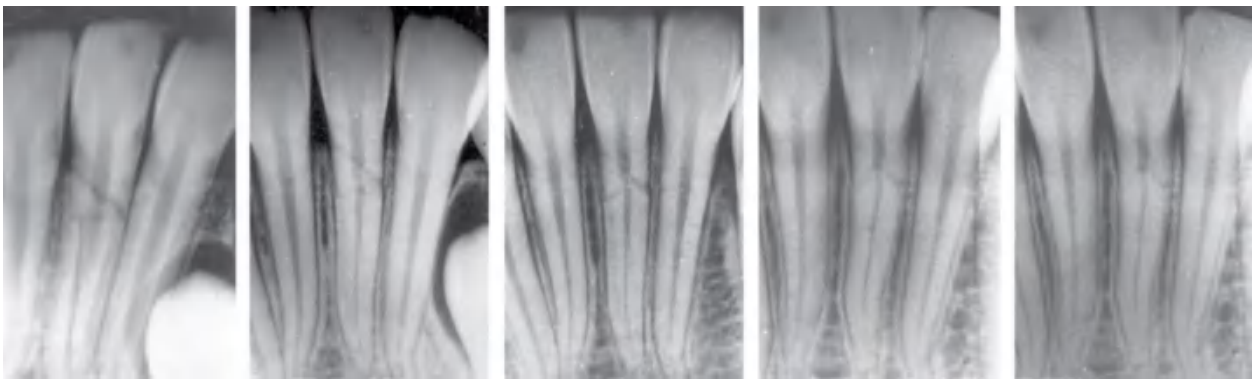


**Fig. 15.37** Healing of a cervical fracture with hard tissue. A transverse oblique and complicated fracture of a mandibular incisor involving the cervical part of the root healed by the formation of hard tissue between the fragments. The tooth was splinted with a cap splint for 49 days. Control radiographs show slowly progressing healing with the formation of hard tissue; note formation of hard tissue at the fracture site in the pulpal lumen. Reproduced from (154) with permission.

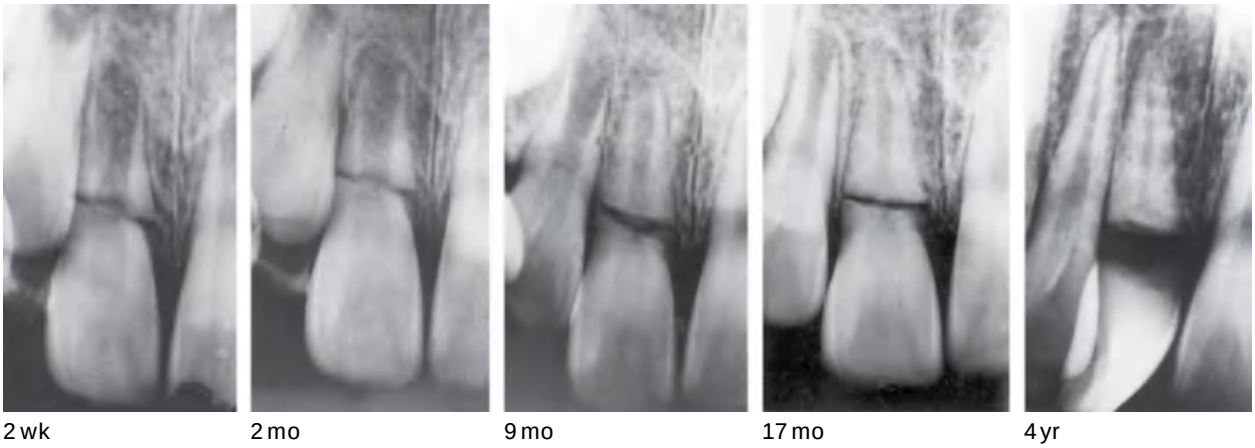


**Fig. 15.38** Healing of a cervical fracture with connective tissue. An immature incisor with a transverse root fracture in the cervical one-third, healed with interposition of soft tissue between the fragments, followed by a secondary injury and tooth extraction. Treatment was delayed by the patient's fear of treatment. The tooth was immobilized with a cap splint for 2 months. Control radiographs taken 2, 9 and 17 months after injury show healing with interposition of soft tissue and continued root development. After 4 years, the tooth suffered a new luxation injury and had to be extracted. Reproduced from (154) with permission.

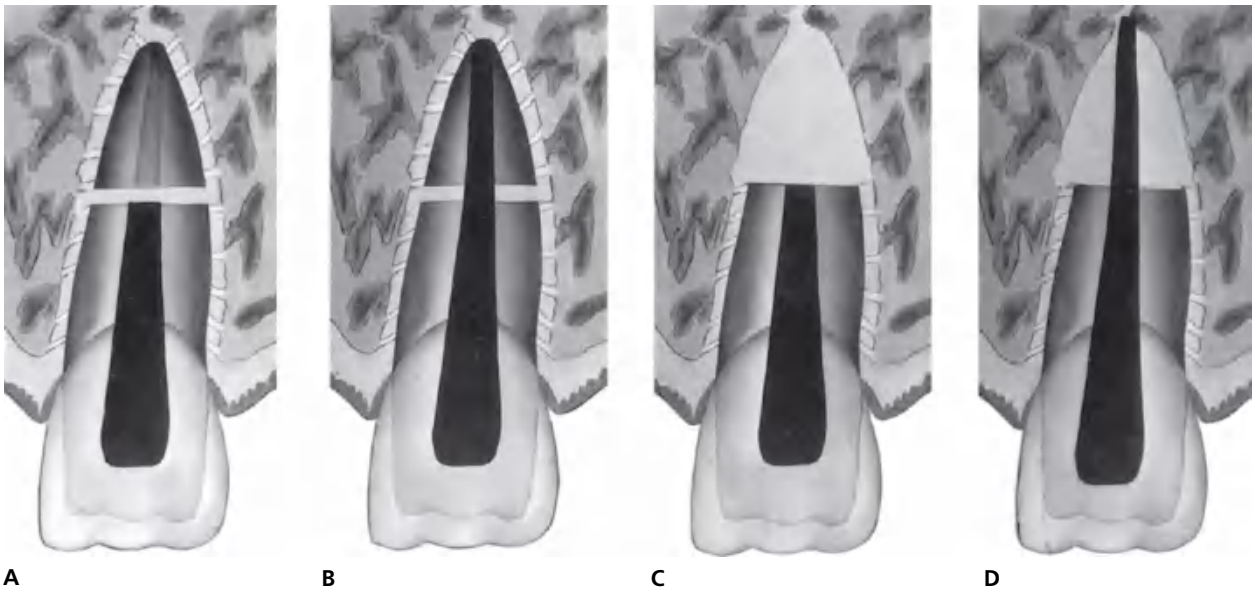


**Fig. 15.39** Schematic drawings illustrating various types of treatments of pulp necrosis after root fracture. A. Root canal filling of coronal fragment. B. Intraradicular splinting with a metal pin used as root canal filling. C. Surgical removal of apical fragment together with root canal filling of coronal fragment. D. Metal implant replacing the apical fragment and acting as root canal filling. Reproduced from (70) with permission.

root-filled, a treatment form which has been shown to result in a high rate of healing (22, 67, 93, 120, 156). This form of therapy is described in detail in Chapter 25.

If the fracture line is situated in the cervical third of the root and the pulp is necrotic, the coronal fragment will become quite mobile. Intraradicular splinting, with a metal pin uniting the fragments and serving as a root canal filling, has been tried (92, 94–97, 124–126). So far there are only limited clinical data concerning long-term prognosis of this form of therapy. However, the failure rate appears to be as high as half of all treated cases (125, 126).

Another suggested method of stabilizing the coronal fragment involves using metal endodontic implants to replace the apical fragment (66, 68–70, 86, 98–105, 127–135). Prefabricated implants are normally used in association with standardized endodontic intracanal instruments (68) (Fig. 15.40). The purpose of the implant is to shift the fulcrum of transverse movements to a more apical position. Clinically, this shift is evident in the stability of the fractured tooth immediately after implantation. However, successful long-term prognosis for these implants is doubtful. Thus, in the authors' experience, 12 implants that originally were considered to have a good prognosis failed at later follow-ups (70) (Fig. 15.41). Moreover, increasing numbers of experimental studies have demonstrated evidence of corrosion of the pins as well as severe inflammation in the soft tissues adjacent to these implants (106–109, 136). Only a single experimental study found implants in teeth with no initial periapical inflammation to be accepted with no evidence of inflammation after 6 months (136).

Another treatment procedure has been advocated whereby the coronal fragment is extracted and the apical fragment removed. The root canal in the coronal fragment is enlarged and a combined root filling/root elongation is performed using an aluminum oxide or titanium implant which extends outside the apical foramen (see Chapter 20). However, to date the long-term results of this procedure do not suggest that this procedure surpasses endodontic treatment of the coronal fragment only (14, 93, 137, 156). In this connection it should be borne in mind that root-fractured incisors which heal after endodontic treatment of the coronal fragment tend to show decreasing mobility because of the physiologic decrease in tooth mobility with age (110, 138).

## Pulp canal obliteration

Partial or complete obliteration of the pulp canal is a common finding after root fracture (111, 113–115, 143, 152) (Fig. 15.42). Thus, in clinical studies of root-fractured permanent incisors, pulp canal obliteration was found in 69–73% of the teeth (76, 85, 115). *Partial pulp canal obliteration* is seen most often in the fracture region and the apical fragment. In addition, partial obliteration extends 1–2 mm into the coronal fragment. *Complete pulp canal obliteration* is seen as an even decrease in the size of the entire pulp cavity, leading to total obliteration. Both obliteration types progress at the same rate and are normally well advanced after 9–12 months and approach full density 1–2 years later (85). Obliteration of the apical root canal alone is commonly seen in cases of healing with hard tissue, while obliteration of the

A B C

**Fig. 15.40** Surgical removal of apical fragment and insertion of a metal implant in a central incisor. A. Prefabricated cobalt-chromium implants and standardized endodontic instruments (Endodontic implant Starter Kit-B3, Star Dental Mfg. Co., Inc., Philadelphia, USA). B. A mucoperiosteal flap has been raised and the apical fragment removed. The entire root canal of the coronal fragment is enlarged to the level of the fracture. C. A cobalt-chromium implant of proper size is inserted into the root canal and the course taken by the implant noted. Subsequently, a cavity is created in the alveolar bone, ensuring that the length of the implant exceeds the original length of the root by 2–4 mm. The length of the implant is adjusted to the bone cavity and, in addition, to the level of the lingual surface of the crown. Finally, the implant is cemented into the root canal with a root canal sealer and special care is taken to remove excess cement.

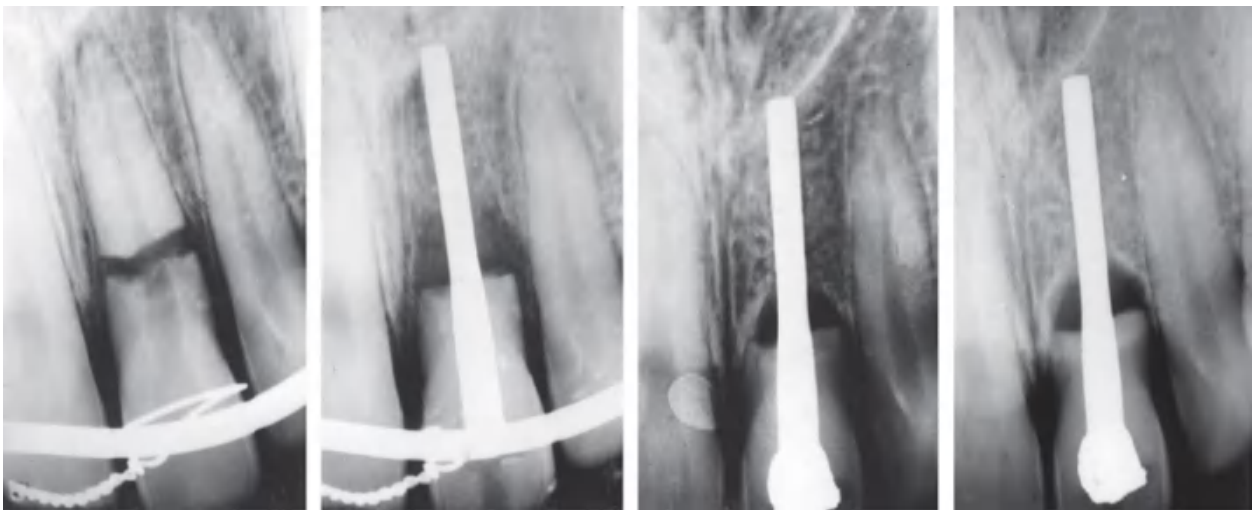


**Fig. 15.41** Treatment of root fracture by removal of apical fragment and insertion of a metal implant. At a follow-up 12 years later, inflammation around the implant is evident and a fistula is present.

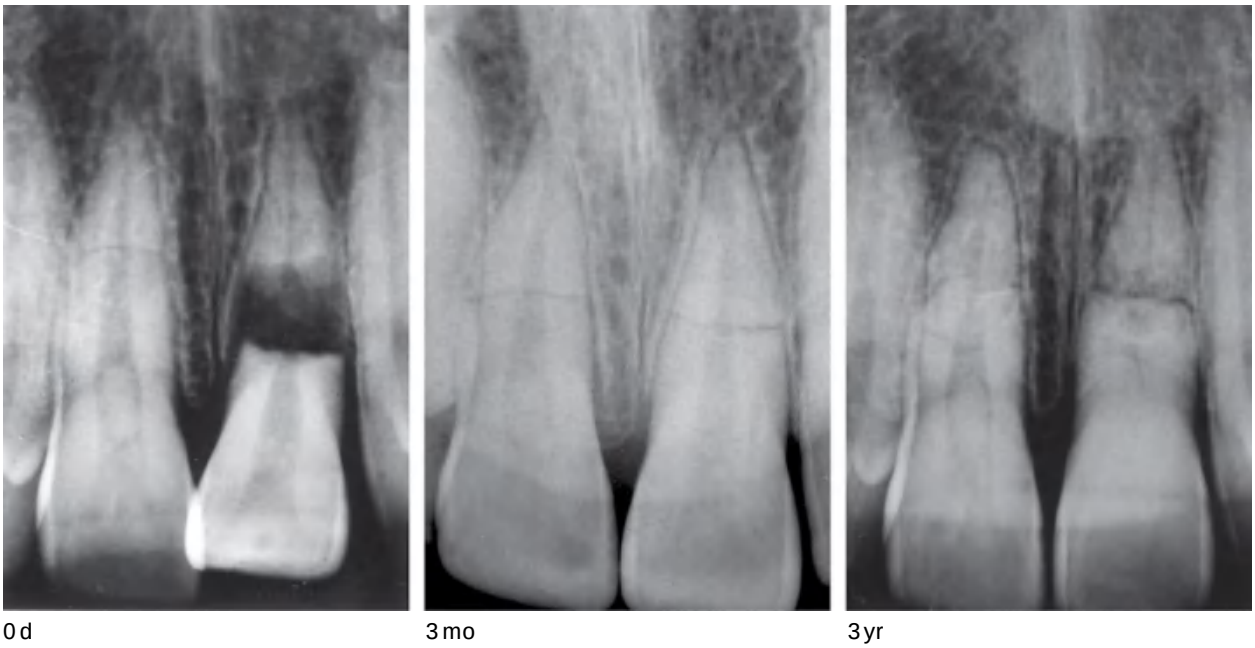


**Fig. 15.42** Effect of displacement of the coronal fragment on root fracture healing. The teeth were repositioned and splinted for 3 months. At the control appointment 3 years later, the right central incisor showed healing, with calcified tissue and partial pulp canal obliteration confined to the apical fragment and at the fracture site whereas the left central incisor demonstrated healing with interposition of connective tissue and complete pulp canal obliteration. Reproduced from (115) with permission.

apical and coronal aspects of the root canal is often seen in cases with interposition of connective tissue, as well as in teeth with interposition of connective tissue and bone (85, 113). In this context, however, one should be aware that in cases of pulp necrosis, while the coronal root canal is usually the only part of the root canal affected, the apical root canal can be completely obliterated. Thus, one must use other criteria (e.g. resorption of bone and loss of lamina dura at the level of the fracture, loss of sensibility, loosening of the coronal fragment) for the determination of pulpal status. Clinically, a slight yellowish discoloration of the crown is sometimes seen in cases of coronal pulp canal obliteration. Pulpal sensibility testing is in most cases normal, but a negative response can be registered. Secondary pulp necrosis is a rare finding (85).

## Root resorption

Root resorption has been found to occur in approximately 60% of root-fractured permanent incisors and can usually be detected within 1 year after injury (113). These

processes often precede fracture healing and obliteration of the coronal and/or apical aspects of the root canal and should be distinguished from resorption of bone at the level of the root fracture, which is indicative of coronal pulp necrosis. Root resorption appears in the following types.

(1) *External repair-related resorption* characterized by the rounding of the fracture edges mesially and/or distally (Fig. 15.12).
(2) *External infection-related resorption* (Fig. 15.43).
(3) *External ankylosis-related resorption* (Fig. 15.44).
(4) *Internal repair-related resorption* manifested as rounding of the fracture edges centrally, in the apical and coronal root canals, at the intersection between the root canal and fracture line (Figs 15.12, 15.13 and 15.14).
(5) *Internal tunneling resorption*, going behind the predentin layer and burrowing along the root canal walls of the coronal fragment (Figs 15.13 and 15.14).

Only external inflammatory resorption needs treatment (pulp extirpation and root-filling). External replacement resorption (ankylosis) cannot be treated. The other types of resorption just require observation.

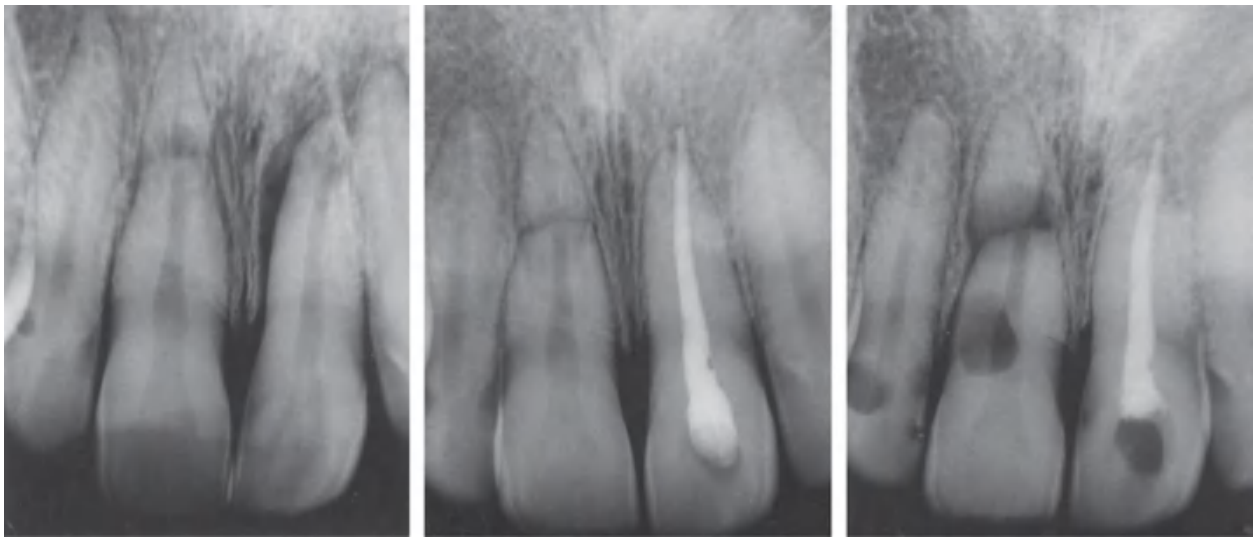


**Fig. 15.43** External infection-related resorption following root fracture of a right central incisor. External resorption is evident at re-examination 2 years after injury. Mesial and distal radiographic projections reveal the external nature of this resorption process. Pulp necrosis was diagnosed at the clinical examination.

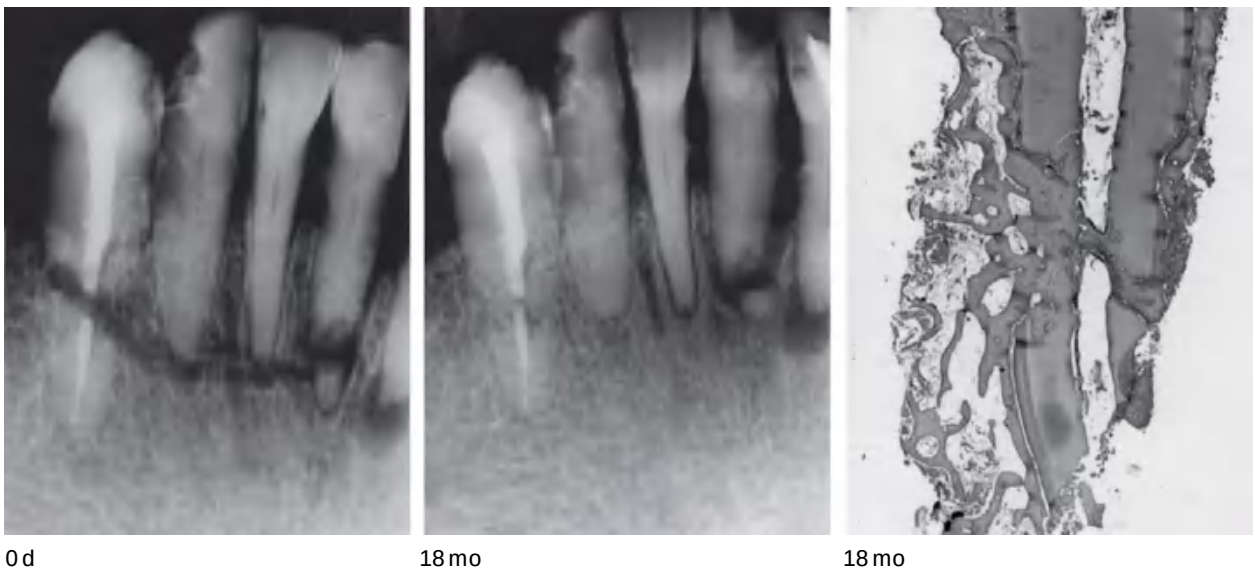


**Fig. 15.44** External replacement resorption following root fracture of a right canine as well as fracture of the alveolar process. At re-examination 18 months later, external replacement resorption is evident. Histologic examination of the extracted canine demonstrates marked replacement resorption. ×6.

## Tooth survival

In 1989, the first long-term study was published by Andreasen and co-workers based on a follow-up of up to 10 years of 95 permanent teeth (157). In 2012, a long-term study of the fate of root fractures up to 20 years after trauma was published (172). The most significant finding was that root fracture location is a very significant predictor of tooth survival (172). Thus root fractures located in the apical third had a 10-year survival of 89%, whereas root fractures located mid-root had a 10-year survival of 78%. A disturbing finding was that root fractures located in the cervical area had only a survival rate of 33% (Fig. 15.45). A more detailed analysis of these figures showed that tooth loss was related to endodontic complications where pulp necrosis in the coronal part of the fracture was not considered amenable to treatment. Two other complications leading to premature tooth loss were excessive loosening and loss due to a new trauma. In this study (172), it was suggested that stability of the cervical root fracture should be increased through the use of an orthodontic retention wire once the type of fracture healing was established (Fig. 15.46). Whether such a procedure will decrease the failure rate is still to be tested and proved.

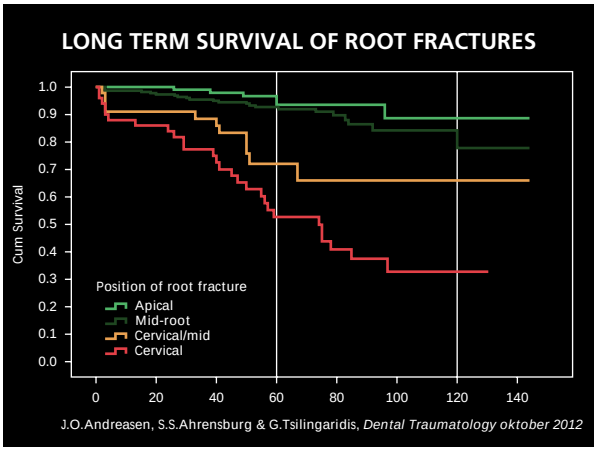


**Fig. 15.45** Tooth survival related to fracture location on the root, showing point estimates after 10 years with 95% confidence intervals.

**Fig. 15.46** Orthodontic palatal retainer.

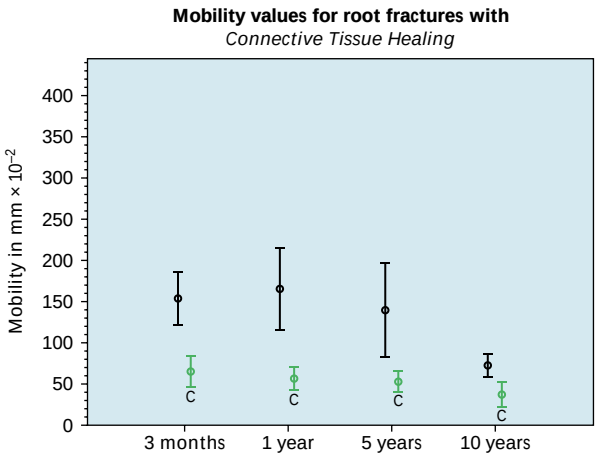


**Fig. 15.47** Mobility values for root-fractured teeth with connective tissue healing and control teeth (C) within a 10-year observation period.

## Tooth mobility

Abnormal loosening is a complication that affects almost all root-fractured teeth. In 2012 a longitudinal study was published on tooth mobility changes over a 10-year period (173). It appeared that abnormal mobility values were significantly reduced, and approached normal values, in cases with connective tissue healing (Fig. 15.47).

## Orthodontic treatment of root-fractured teeth

This problem is discussed in detail in Chapter 28.

## Predictors of healing

In this respect, the following three events are important: development of *pulp necrosis* and *healing by hard or connective tissue*. Predictors of healing are summarized in Fig. 15.48.

Basic information about these topics has been disclosed by two larger clinical studies (143, 147). As it appears from Fig. 15.49, the healing events are generally controlled by the *extent of injury* (± rupture or stretching of the pulp at the level of fracture) and the *anatomy of the pulp* (size and vascularity). Concerning treatment effects, *optimal repositioning* and the use of *flexible* (versus rigid) *splinting* have been found to favor healing.

In Fig. 15.50, the relative influence of the various factors is presented, based on a recent study of 400 root-fractured incisors (143, 147). By entering information from the time of injury, the likelihood of different healing events can be ascertained.

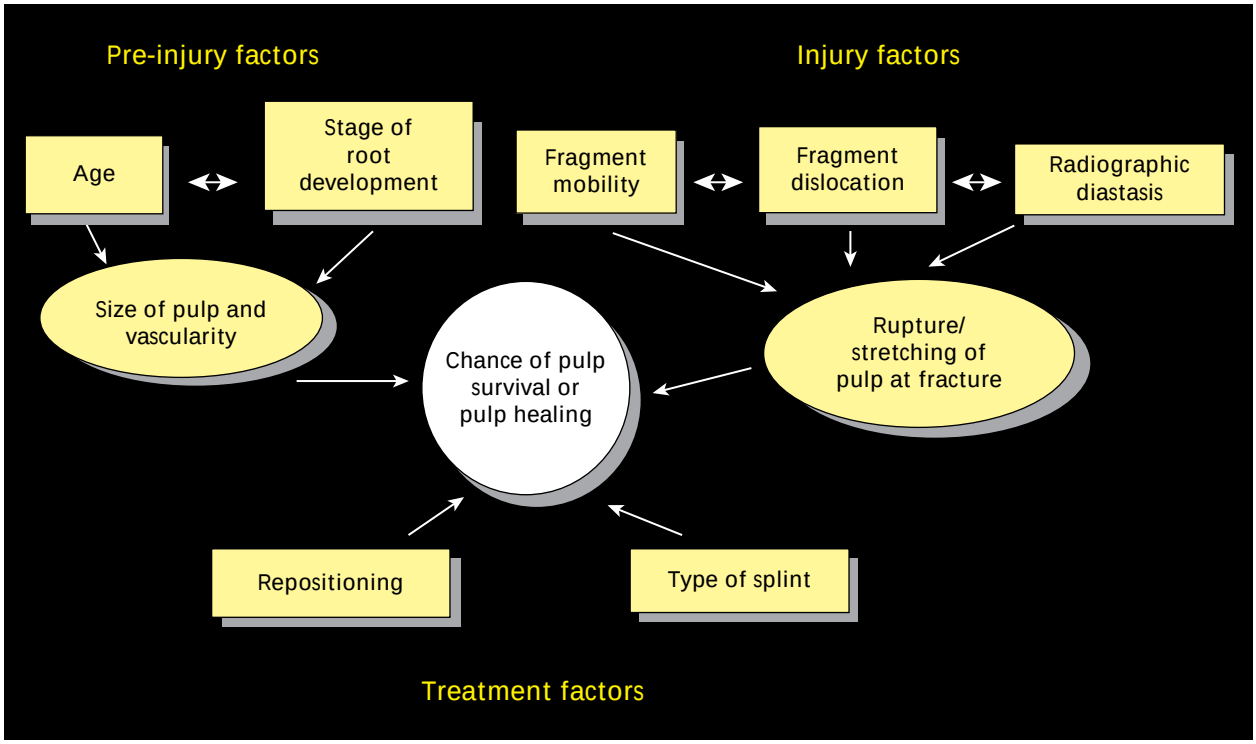


**Fig. 15.48** Summary of predictors for healing.

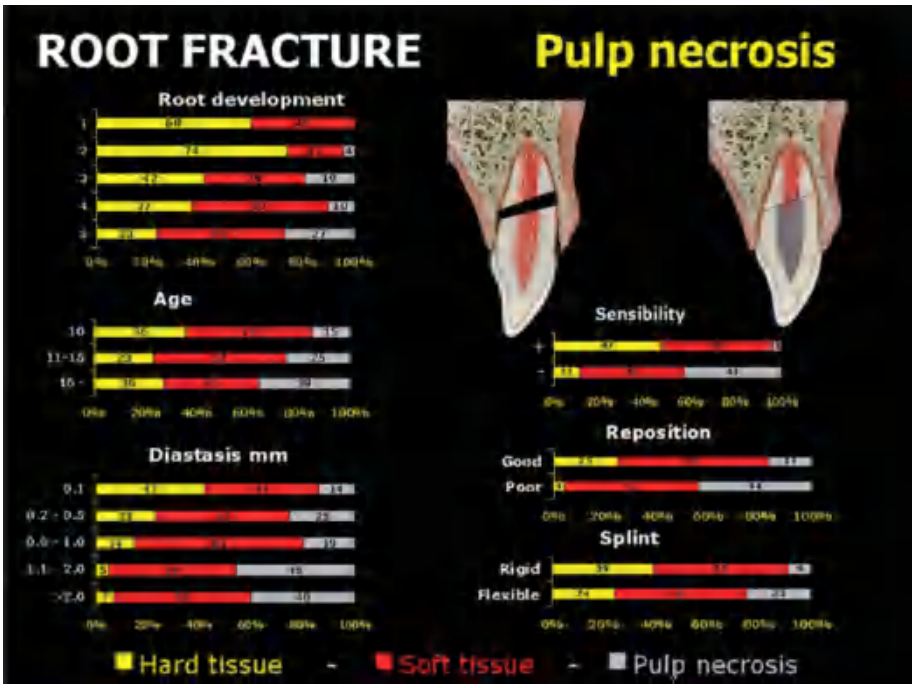


**Fig. 15.49** Predictors for healing.

## Type of healing

The following *pre-injury and injury factors* had a positive influence on healing (i.e. hard tissue versus non-healing/pulp necrosis): immature root formation, lower age and less displacement of the coronal fragment. The *treatment factors* which had a positive influence upon healing were optimal repositioning and flexible splinting (Fig. 15.50).

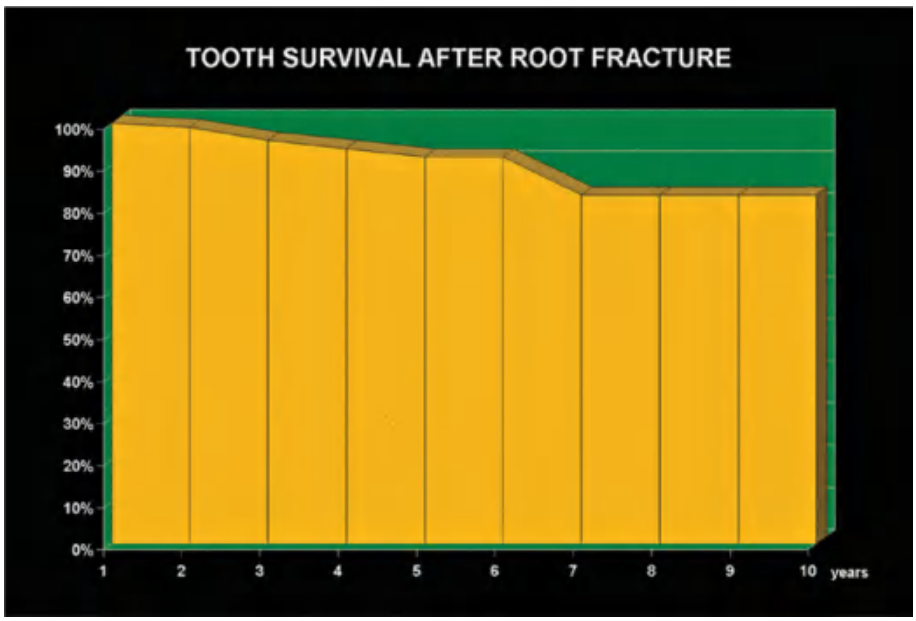


**Fig. 15.50** Long-term survival of root fractures. Reproduced from (157) with permission.

## Essentials

### Terminology

- Root fracture

### Frequency

- Primary dentition: 2–4% of dental injuries
- Permanent dentition: 0.5–7% of dental injuries

### Etiology

- Fight injuries
- Foreign bodies striking the teeth

### History

- Symptoms
- Tenderness from occlusion

### Clinical examination

- Mobility
- Dislocation
- Reaction to sensibility tests
- Stage of root development
- Fracture site
- Dislocation
- Single, comminuted fracture

### Radiographic examination

- Occlusal exposure plus orthoradial periopical exposure and one disto- and mesioradial exposure
- CBCT is not indicated in the acute examination

### Healing and pathology

- Healing with calcified tissue
- Interposition of periodontal ligament
- Interposition of bone and periodontal ligament
- Non-healing with interposition of granulation tissue

### Treatment

- Reposition the coronal fragment, if displaced (Figs 15.27 and 15.28)
- Check position of the coronal fragment radiographically
- Immobilize the tooth with a semi-rigid splint (e.g. acid-etch/resin splint)
- Control the tooth radiographically and with sensibility tests
- Maintain the splint for 4 weeks
- Follow-up period minimum of 1 year
- If the fracture is located very close to the gingiva, extraction of the coronal fragment may be indicated, followed by orthodontic or surgical extrusion of the apical fragment (see Chapter 24). However, in the case of optimal oral hygiene, a conservative approach comprising permanent proximal fixation may be attempted

### Prognosis

- Pulp necrosis: 20–40% (age dependent)
  - Treatment of pulp necrosis
  - Root canal treatment of coronal fragment (see Chapter 25)
- Pulp canal obliteration: 69–73%
- Root resorption: 60%
  - Surface resorption (internal and/or external) is considered to be a link in fracture healing and requires no treatment
  - External inflammatory resorption and ankylosis (replacement resorption) are very rare

### Tooth survival

- Cervically located root fractures may suffer premature loss caused by excessive loosening or new trauma. A lingual orthodontic retainer is indicated to reduce the risk

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# 16

# Luxation Injuries of Permanent Teeth: General Findings

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## Terminology, frequency and etiology

Depending on the direction of impact, a variety of luxation injuries may occur. From a therapeutic, anatomic and prognostic point of view, five different types of luxation injuries can be recognized (Fig. 16.1).

(1) *Concussion*: an injury to the tooth-supporting structures without abnormal loosening or displacement but with marked reaction to percussion (Fig. 16.1A).
(2) *Subluxation (loosening)*: an injury to the tooth-supporting structures with abnormal loosening but without clinically or radiographically demonstrable displacement of the tooth (Fig. 16.1B).
(3) *Extrusive luxation (peripheral displacement, partial avulsion)*: partial displacement of the tooth following the axis of the tooth out of its socket but without leaving the socket (Fig. 16.1C). Radiographic examination always reveals an increased width of the periodontal ligament space.
(4) *Lateral luxation*: eccentric displacement (other than axial) of the tooth. This is accompanied by comminution or fracture of the alveolar socket (Fig. 16.1D, E). Depending on the angulation of the central beam, radiographic examination may or may not demonstrate an increased width of the periodontal ligament space (82).
(5) *Intrusive luxation (central dislocation)*: displacement of the tooth deeper into the alveolar bone. This injury is accompanied by comminution or fracture of the alveolar socket (Fig. 16.1F). The direction of dislocation follows the axis of the tooth. Radiographic examination reveals dislocation of the tooth and sometimes a missing or diminished periodontal space. In the adult dentition, an apical shift of the cemento-enamel junction of the involved tooth can be seen.

The most important clinical difference between intrusive and extrusive luxation is that in the latter the apex is displaced out of its socket and not through the alveolar bone socket as in intrusive luxation. Moreover, extrusive luxation can imply complete rupture or stretching/tearing of the neurovascular supply to the pulp at the apical foramen, and the periodontal ligament fibers are to a great extent severed, whereas the supporting bone is not affected.

The factors that determine the type of luxation injury appear to be the force and direction of impact (Fig. 16.2).

Luxation injuries comprise 15–61% of dental traumas to permanent teeth (1–5).

## Clinical findings

In both the primary and permanent dentitions, tooth luxations primarily involve the maxillary central incisor region and are seldom seen in the mandible (1–5).

With increasing age, the frequency and pattern of injury change. In the primary dentition, intrusions and lateral luxations comprise the majority of all injuries, a finding which is possibly related to the high resilience of the alveolar bone at this age. In contrast, in the permanent dentition the number of intrusive luxation injuries is considerably reduced and usually seen in younger individuals (5).

Most frequently, two or more teeth are luxated simultaneously and a number of luxations show concomitant crown or root fractures (1, 2, 170).

Clinical and radiographic findings for different luxation injuries are summarized in Table 16.1. While the diagnosis of luxation injuries is based on a combination of

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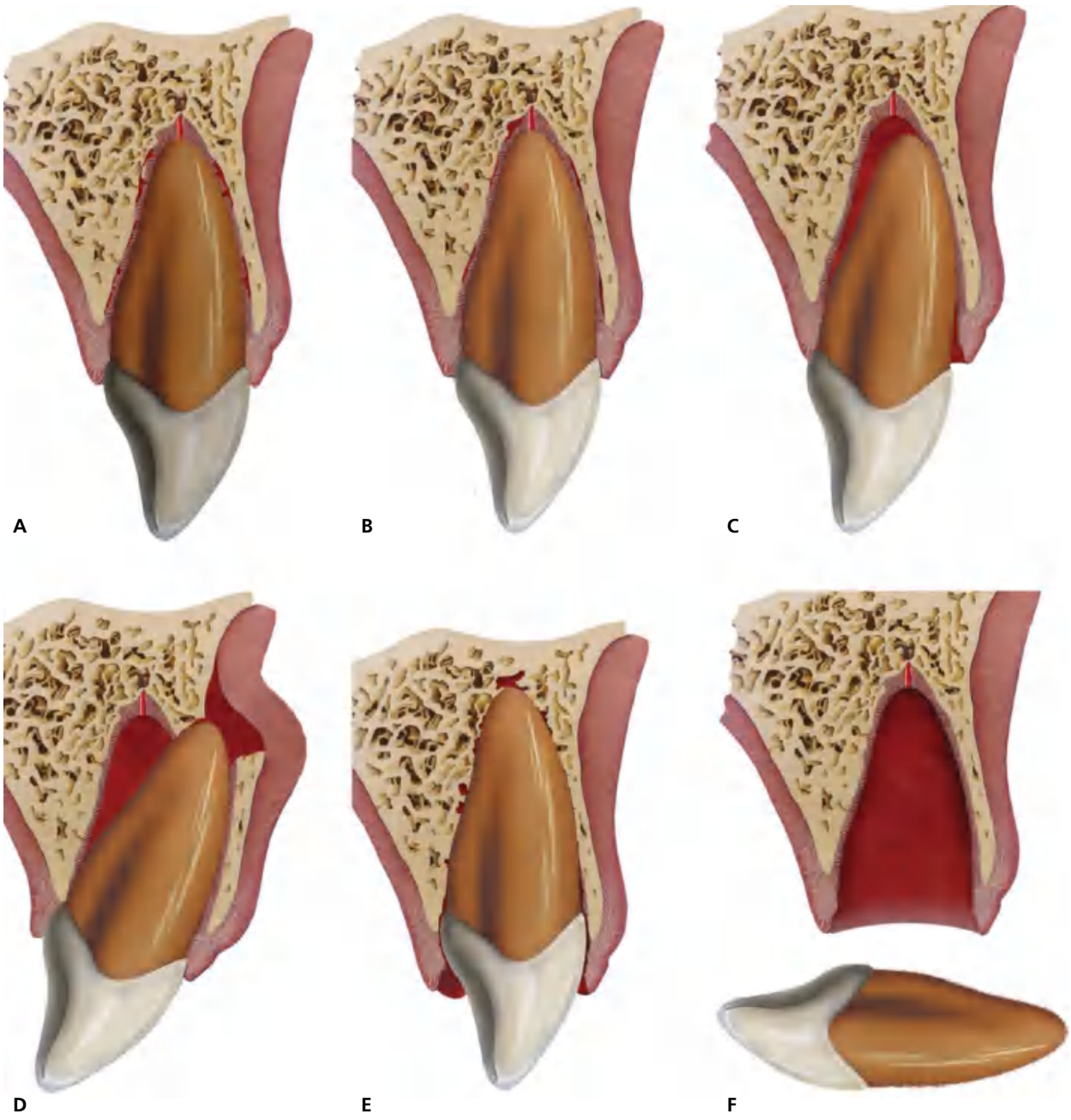



**Fig. 16.1** Injuries to the periodontal tissues. A. Concussion. B. Subluxation. C. Extrusive luxation. D and E. Lateral luxation. F. Intrusive luxation.

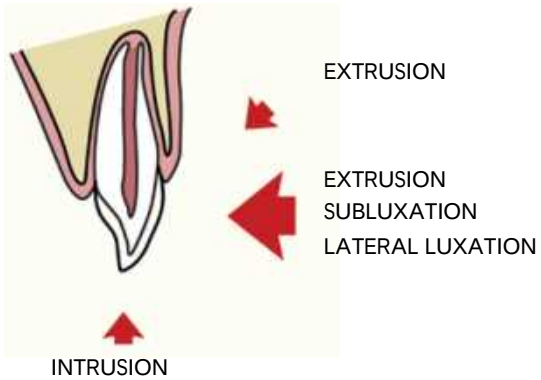



**Fig. 16.2** The energy and direction of the impact determine the type of luxation injury.

radiographic and clinical findings including sensibility testing (Table 16.1), diagnostic accuracy increases with multiple radiographic exposures, where angulation of the central beam is altered (82).

## Radiographic findings

Radiographic examination is an important adjunct to the clinical examination as it can disclose minor dislocations. It has been shown experimentally that error in the radiographic recording of the distance of dislocation is minimal if the bisecting angle technique is used (82). Moreover, in

**Table 16.1** Typical clinical and radiographic findings with different types of luxation injuries.

| | Type of luxation injury | | | | |
|---|---|---|---|---|---|
| | **Concussion** | **Subluxation** | **Extrusion** | **Lateral luxation** | **Intrusion** |
| Abnormal mobility | – | + | + | –(+)* | –(+) |
| Tenderness to percussion | + | +(–) | +(–) | – | – |
| Percussion sound | Normal† | Dull | Dull | Metallic | Metallic |
| Positive response to sensibility testing | +/– | +/– | –(+) | –(+) | – |
| Radiographic dislocation | – | – | + | + | + |

* A sign in parentheses indicates a finding of rare occurrence.
† Teeth with incomplete root formation and teeth with marginal or periapical inflammatory lesions will elicit a dull percussion sound.

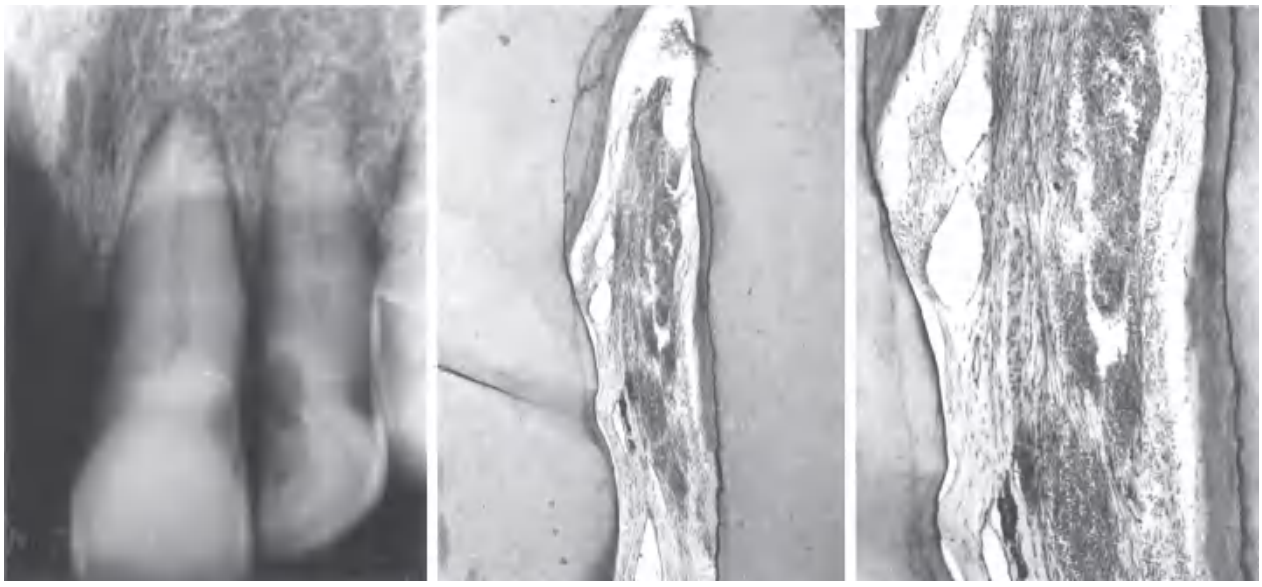



**Fig. 16.3** Early histologic changes following subluxation of a permanent central incisor. A. Radiograph of a left central incisor following subluxation. The tooth was extracted 6 hours after injury. B. Perivascular bleeding is evident in the apical part of the pulp. ×30. C. Higher magnification of B. ×75.

order to obtain reproducible radiographs, film holders should be employed to standardize exposure technique. Thus, an average of 6.9% error in measurement of tooth length was encountered if film holders were not used compared to a 2.0% average error if they were (82). The probability of diagnosing displacement (irrespective of direction) is increased if multiple radiographic exposures with varying vertical and horizontal angulations of the central beam are taken of the injured region (82).

## Healing and pathology

Most luxations represent a combined injury to the pulp and periodontium. However, the pathology of luxation injuries has received little attention. At present, only a few specific changes can be ascribed to the periodontal ligament after luxation injury (83–88, 132, 133), whereas pulpal changes have been studied to a certain extent (6, 54, 57–60, 87–90, 132–134).

### Pulp

Changes seen in the pulp soon after injury are edema and disorganization of the odontoblast layer as well as nuclear pyknosis of pulp cells (6, 134). Perivascular hemorrhage can be demonstrated within a few hours in human subjects (Fig. 16.3). Such bleeding could be the result of a partially severed vascular supply. However, in most cases a complete arrest of pulpal circulation occurs, which leads to ischemia, breakdown of vessel walls, escape of erythrocytes and the eventual conversion of hemoglobin to red granular debris which permeates the pulp tissue and gives it a scarlet red color (60). Ultrastructurally as well, blood vessels with disrupted or absent endothelium and erythrocyte remnants can be seen (89).

Histologic evidence of pulp necrosis, including nuclear pyknosis, disappearance of odontoblasts and stromal elements, can be seen 6 days after injury (Fig. 16.3). This response is related to either partial or total rupture of the pulpal neurovascular supply. The later events of pulpal healing after luxation injury are presently unknown. However, based on experimental findings after extraction and replantation of teeth, it might be expected that a gradual revascularization and reinnervation of the pulp occurs if the size of the apical foramen is adequate to permit vascular ingrowth and that intervening infection of the ischemic pulp does not occur. This process is further described in Chapter 2.

If the pulp survives or becomes revascularized, a number of regressive pulp changes can occur, such as hyalinization and deposition of amorphous, diffuse calcifications (6, 9, 10). Furthermore, the injury usually interferes with normal dentin formation, which appears to be related to a number of clinical factors, among which the stage of root formation and the type of luxation injury seem to be decisive (62, 63, 92, 93).

In teeth with incomplete root development, a distinct incremental line usually indicates arrest of the formation of normal tubular dentin at the time of injury. Most of the dentinal tubules stop at this line, while the original predentin layer is often preserved.

After a certain period, apposition of new hard tissue is resumed, but without the normal tubular structure. This tissue often contains cell inclusions which maintain their tubular connections with dentin formed before the injury (11, 15), as well as vascular inclusions. The junction between old and new dentin is rather weak, explaining the separation which may occur in this area during extraction (12–14). This hard tissue formation often continues to the point of total obliteration of the root canal (15, 17–25, 64).

Although the calcified cellular tissue that is formed in response to injury may resemble bone and cementum, it lacks the cellular organization characteristic of these tissues. Due to its tendency to convert to tubular dentin, the repair tissue has been termed cellular dentin (16). The tendency to return to tubular dentin could be related to differentiation of new odontoblasts from the primitive mesenchymal cells of the pulp, a response which is especially marked in the apical portion of the root canal, possibly due to the rapid re-establishment of blood supply in this area after injury (16).

The rate of hard tissue formation is accelerated after injury, resulting in large amounts of newly formed hard tissue, especially coronally.

In the case of luxation of young permanent teeth, bone can be deposited within the pulp after trauma and is usually connected to the root canal walls by a collagenous fiber arrangement imitating a periodontal ligament (see Chapter 2) (135). This form of healing has been found to be related to damage or destruction of Hertwig's epithelial root sheath (HERS). Such damage can arise from either incomplete repositioning, whereby HERS suffers injury due to ischemia, or by forceful repositioning, whereby all or parts of HERS are crushed (94).

In mature teeth, the disturbance of the odontoblast layer can be more severe, with resorption sometimes preceding deposition of new hard tissue (16–18, 95, 119, 131, 172, 173). Presumably, the time required for re-establishing vascularization after injury is longer in a tooth with complete root formation, thus increasing the damage to the pulp. The cellular hard tissue formed after the injury rarely assumes a tubular appearance; this applies especially to the coronal portion.

Temporary resorption of bone surrounding the root apex and of dentin within the apical foramen of extruded and laterally luxated mature incisors has been described (transient apical breakdown (TAB)). Mineralization of these resorption processes could be associated with normalization of coronal discoloration and/or restoration of pulpal sensibility and was significantly related to later pulp canal obliteration (see Chapter 2) (95) (Figs 16.4 and 16.5).

Teeth with incomplete root formation at the time of injury sometimes show pulp necrosis confined to the coronal part of the pulp, while the apical portion apparently survives for some time, ensuring occlusion of the wide apex with calcified tissue (see Chapter 2) (8, 61).

## Periodontal ligament

The histologic response in the periodontal ligament (PDL) has only been sparsely studied experimentally. *Concussion* and *subluxation* have been induced in rat molars (83, 87, 88, 171) and in dog incisors (85). Furthermore, *extrusive luxations* have been studied in monkeys (86) and rats (87, 88, 134), whereas *intrusions* have been analyzed in dogs (84, 132, 133). A single study has examined the effect of alendronate on the healing response in rats (156). These studies have revealed the following PDL changes after trauma (see Chapter 2).

### *Concussion/subluxation*

The general features are edema, bleeding and sometimes laceration of PDL fibers. The neurovascular supply to the pulp may or may not be intact. The PDL changes that occurred 1 hour after trauma were characterized by hemorrhage, stretched, torn or compressed PDL fibers, cell destruction and edema (88). After 1 day, cell-free zones could be seen in the PDL, bordered by a zone of inflammation. In the bony socket, 1 day after trauma, osteoclastic activity had begun. After 1 week, this had reached the root surface. After 10 days, the resorption activity had ceased, leaving healed surface resorption cavities along the root surface (88).

### *Extrusive luxation*

Immediate changes are characterized by a complete rupture of the PDL fibers and the neurovascular supply to the pulp. After 3 days, in monkeys, the split in the PDL, which is usually seen midway between the bone and root surface, is filled in with a tissue dominated by endothelial cells and young fibroblasts. In some areas, cell-free zones indicate infarcted tissue. After 2 weeks, newly formed collagen fibers are seen. After 3 weeks, the PDL appears normal (86).

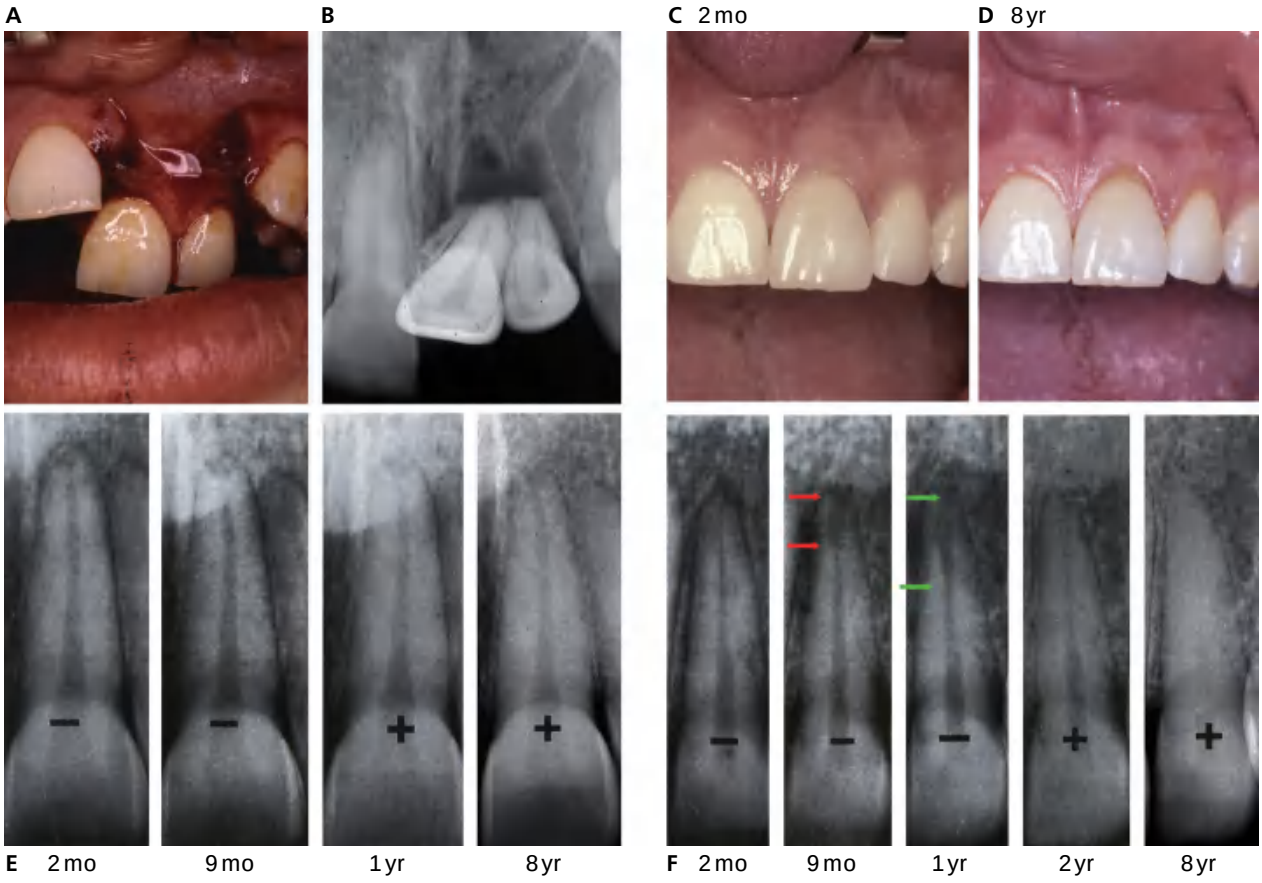



**Fig. 16.4** Root canal repair-related resorption of the apical foramen after fracture of the alveolar process and extrusion of the left maxillary central and lateral incisors in a 21-year-old woman. A and B. Clinical and radiographic appearance at the time of injury. C. Clinical appearance 2 months after injury. The crown of the left central incisor has become gray. D. Clinical appearance at 8 years. E and F. Orthoradial exposures of the central and lateral incisors respectively at 2 months, 9 months, 1 year, 2 years and 8 years after injury. Plus and minus signs on the radiographs indicate positive or negative responses to pulp testing. Red arrows indicate the start of root canal repair-related resorption and green arrows an arrest of resorption. At the 8-year control, the color of the left central incisor is almost normal. Reproduced from (98, 172) with permission.

### *Lateral luxation*

This comprises a complex injury involving rupture or compression of PDL fibers, severance of the neurovascular supply to the pulp and fracture of the alveolar socket wall.

### *Intrusive luxation*

The immediate response to intrusion in monkeys appears to be an extensive crushing injury to the PDL and alveolar socket and rupture of the neurovascular supply to the pulp (91). After an observation period of 3 months, some intruded teeth in dogs showed extensive ankylosis, whereas others showed areas with surface resorption and otherwise normal PDL (84, 132, 133).

## Treatment

Therapeutic measures vary greatly from the primary to the permanent dentitions and according to the type of injury to the tooth-supporting structures. In this context, it should be borne in mind that repositioning and sometimes even splinting procedures can elicit further trauma. Before repositioning of a displaced tooth, it is therefore worth considering whether the repositioning procedure achieves any of the following goals:

(1) Facilitates pulpal healing?
(2) Facilitates periodontal ligament healing?
(3) Eliminates occlusal interferences?
(4) Improves esthetics?

At least one or more of these objectives should be achieved by the repositioning procedure, especially if this procedure, as in the case of lateral luxation, results in further damage at the site of injury. The use of and effect of treatment procedures are described in the following chapters on the various types of luxation injury.

## Prognosis

The follow-up period can disclose a number of complications, such as pulp canal obliteration, pulp necrosis, root

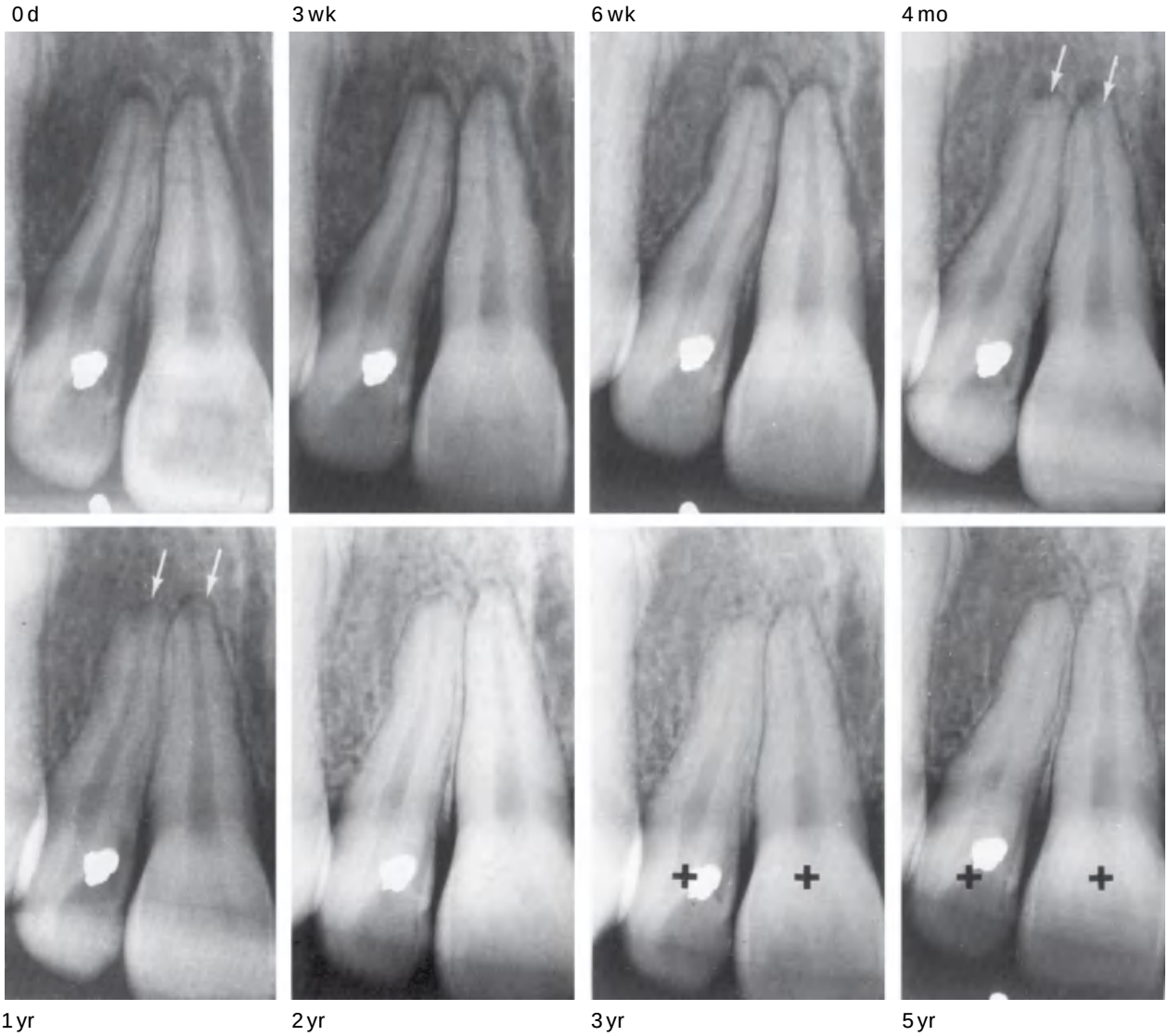



**Fig. 16.5** Transient apical breakdown after extrusive luxation of the maxillary right central and lateral incisors in a 19-year-old man. There is coronal discoloration of both teeth at the 1- and 2-year controls. Note internal repair-related resorption of both teeth at the apical foramina (arrows) 4 months and 1 year after injury, which is later followed by apical blunting (external repair-related resorption) and pulp canal obliteration of the lateral incisor. Plus signs on radiographs indicate when the incisors regained normal pulpal sensibility. Reproduced from (95) with permission.

resorption and loss of marginal bone support. Good surveys of these complications have been published by Dumsha (138) and Andreasen and Vestergaard Pedersen (92). A follow-up schedule whereby healing complications can be diagnosed and interceptively treated is presented in Appendix 3 (p. 1024). However, in order to utilize the suggested recall schedule to full advantage, certain information must be gathered at the time of injury in order to identify those cases in which healing complications can be anticipated. The information needed for such clinical decisions will be discussed in the following.

'Tooth luxation' as a clinical diagnosis covers a broad spectrum of injury from a therapeutic, anatomic and prognostic point of view. A multivariate analysis of clinical and radiographic data registered at the time of injury confirmed the relevance of the current classification system, in that each type of injury could be described by a unique survival curve with respect to the development of pulp necrosis following injury (see later in this chapter) (92). The same classification system was found useful in describing the luxation injury to the coronal fragment of a horizontal root fracture (see Chapter 12) (98, 99).

## Pulp necrosis

The frequency of pulp necrosis after luxation injuries in the permanent dentition has been found to range from 15% to 59% (Table 16.2).

Three factors have been found to be significantly related to the development of pulp necrosis: the *type of luxation injury*, the *stage of root development* and the *presence of a*

**Table 16.2** Prevalence of pulp necrosis after luxation of permanent teeth irrespective type of luxation injury.

| Study | No. of teeth | Pulp necrosis |
|---|---|---|
| Skieller (7) 1960 | 107 | 44 (41%) |
| Weiskopf et al. (38) 1961 | 121 | 72 (59%) |
| Anehill et al. (39) 1969 | 76 | 18 (24%) |
| Andreasen (3) 1970 | 189 | 98 (52%) |
| Stålhane & Hedegård (62) 1975 | 1116 | 172 (15%) |
| Rock et al. (56) 1974 | 200 | 75 (38%) |
| Rock & Grundy (97) 1981 | 517 | 192 (37%) |
| Wepner & Bukel (125) 1981 | 142 | 30 (21%) |
| Andreasen & Vestergaard Pedersen (92) 1985 | 637 | 156 (24%) |
| Oikarinen et al. (100) 1987 | 147 | 74 (50%) |
| Herforth (101) 1990 | 319 | 81 (25%) |
| Crona-Larsson et al. (136) 1991 | 104 | 24 (23%) |
| Robertson (137) 1997 | 196 | 34 (17%) |

**Table 16.3** Prevalence of pulp necrosis according to type of luxation of permanent teeth with immature root development estimated after 10 years of observation. Reproduced from (168) with permission.

| Type of luxation | No. of teeth | Pulp necrosis |
|---|---|---|
| Concussion | 62 | 0% |
| Subluxation | 155 | 0% |
| Extrusive luxation | 34 | 5.9% (95% CI: 0–13.6) |
| Lateral luxation | 43 | 4.7% (95% CI: 0–10.8) |
| Intrusive luxation | 18 | 67.6% (95% CI: 49–85.9) |

**Table 16.4** Prevalence of pulp necrosis according to type of luxation of permanent teeth with mature root development estimated after 10 years of observation. Reproduced from (168) with permission.

| Type of luxation | No. of teeth | Pulp necrosis |
|---|---|---|
| Concussion | 115 | 3.5% (95% CI: 0.2–6.8) |
| Subluxation | 112 | 12.5% (95% CI: 6.4–18.6) |
| Extrusive luxation | 23 | 56.5% (95% CI: 37.7–75.4) |
| Lateral luxation | 103 | 75.3% (95% CI: 64.8–85.7) |
| Intrusive luxation | – | – |

*concomitant crown fracture* (3, 92, 100, 101, 138, 150–155, 157–166) (Tables 16.3 and 16.4). In these investigations it was not possible to demonstrate any effect of treatment upon the development of pulp necrosis (e.g. repositioning, fixation, antibiotic therapy).

## Type of luxation injury

The greatest frequency of pulp necrosis is encountered among intrusive luxation followed by lateral luxation and extrusion (Fig. 16.6), while the least frequent occurrence of pulp necrosis was after concussion and subluxation (3, 7, 62, 73, 92, 101) (Tables 16.3 and 16.4).

## Stage of root development

Pulp necrosis occurs more frequently in teeth with fully developed roots (3, 7, 62, 73, 92) (Tables 16.3 and 16.4, Fig. 16.6). In teeth with immature root development, slight movement of the apex can presumably occur without disruption of the blood vessels passing through the apical foramen. Moreover, the process of revascularization is more easily achieved in teeth with a wide apical foramen, thus favoring pulp survival.

Figure 16.6 shows pulp survival following luxation injury according to type of injury and stage of root development (defined by quarters of anticipated root growth and patency of the apical foramen). It can be seen that teeth with a constricted apical foramen which have been displaced run a greater risk of pulp necrosis than do teeth with patent root apices, where there is a greater possibility of pulpal revascularization.

If the stage of root development is expressed as the mesiodistal diameter of the apical foramen at the time of injury, it is also possible to demonstrate an improved revascularization potential following extrusion and lateral luxation with an increase in apical diameter, presumably reflecting the area of the pulpo-periodontal interface from which new vessels could proliferate (102).

Regarding the diagnosis of pulp necrosis following luxations, different chronologic patterns are followed for the various luxation types. Thus, pulp necrosis can often be diagnosed within the first 6 months after concussion and subluxation; and up to 2 years after injury following extrusion, lateral luxation and intrusion (92), presumably due to difficulties in radiographic diagnosis related to the extent of damage to the periodontal structures.

## Presence of a concomitant crown fracture

A recent epidemiologic study based on 10 166 traumatized permanent teeth reported that 31% of the injured teeth were diagnosed with a luxation injury and a concomitant crown fracture (170). Concomitant crown fracture occurred most frequently in teeth with concussion, subluxation and intrusion (170). Moreover, laterally luxated teeth with infractions had a high rate of pulp necrosis (98, 172). The risk of pulp necrosis is increased in teeth with combination injuries, presumably due to increased bacterial penetrating of the exposed dentin when the blood and nerve supply to the pulp is compromised (see Chapter 13) (150–152, 163–166).

## Diagnosis of pulp necrosis

In the permanent dentition, the development of pulp necrosis can be associated with symptoms such as spontaneous pain or tenderness to percussion or occlusion. In clinical material, where it was possible to compare pulpal histology with clinical and radiographic parameters, it was found that *tenderness to percussion* was the only sign that was significantly related to infected pulp necrosis following tooth luxation (90, 98). However, pulp necrosis following luxation injuries and root fractures is normally asymptomatic and the diagnosis must therefore be based on clinical and

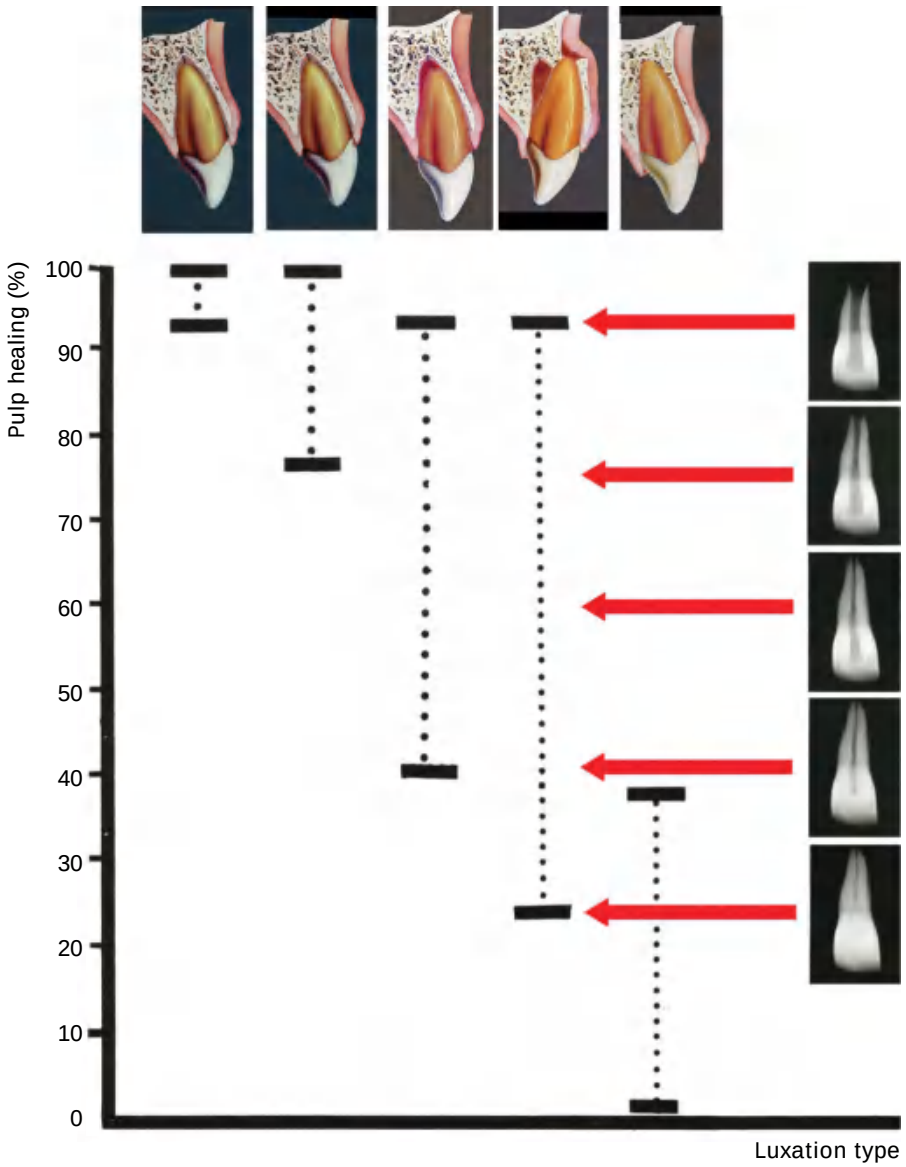



**Fig. 16.6** Relationship between luxation diagnosis, stage of root development and pulpal healing after trauma. The sequence of luxations is concussion, subluxation, extrusion, lateral luxation and intrusion. The upper lines indicate chance of pulpal healing for teeth with immature root formation, and the lower lines the chance for teeth with complete root formation. Reproduced from (92, 98) with permission.

radiographic parameters alone. Transillumination might reveal *decreased translucency* (Fig. 16.7). *Gray color change* in the crown can be seen, which is especially prominent on the oral surface (Fig. 16.7). This may be accompanied by a *periapical radiolucency* which can be observed as early as 2–3 weeks after injury, but many cases show no sign of periodontal involvement radiographically. In these cases, a sterile necrosis might be suspected. Teeth with periapical rarefaction almost always represent pulpal infection, which is dominated by anaerobic microorganisms (70–72). The diagnosis of pulp necrosis should be further confirmed by *pulpal sensibility tests*. However, these findings can be difficult to interpret or can even be misleading. If a pulpal response changes from positive to negative, pulp necrosis should be strongly suspected.

From previous studies, there appears to be general agreement that lack of pulpal sensibility alone (7, 103–106) or coronal discoloration alone (103, 105) are not enough to justify a diagnosis of pulp necrosis. The development of periapical radiolucency (103, 105, 106) has so far been considered the only 'safe' sign of pulp necrosis. However, more recent investigations have cast doubt on the validity of the presently accepted diagnostic criteria (95, 172). Even the concomitant presence of all three classic signs of pulp necrosis (coronal discoloration, loss of pulpal sensibility and periapical radiolucency) could still, in rare cases, be followed by pulpal repair (95) (Figs 16.4 and 16.5). Thus, it has been shown that there are intermediary stations in pulpal response (i.e. TAB) which mimic the cardinal signs of pulp necrosis and which could be reversible and lead to pulpal healing. Even the concomitant presence of all three classic signs of pulp necrosis (i.e. coronal discoloration, loss of pulpal sensibility and periapical radiolucency) could still be followed by pulpal repair (95, 172) (Figs 16.4 and 16.7).

For practical reasons, guidelines for endodontic interventions after luxation injuries are necessary. In the following, the validity of various examination procedures will be described.

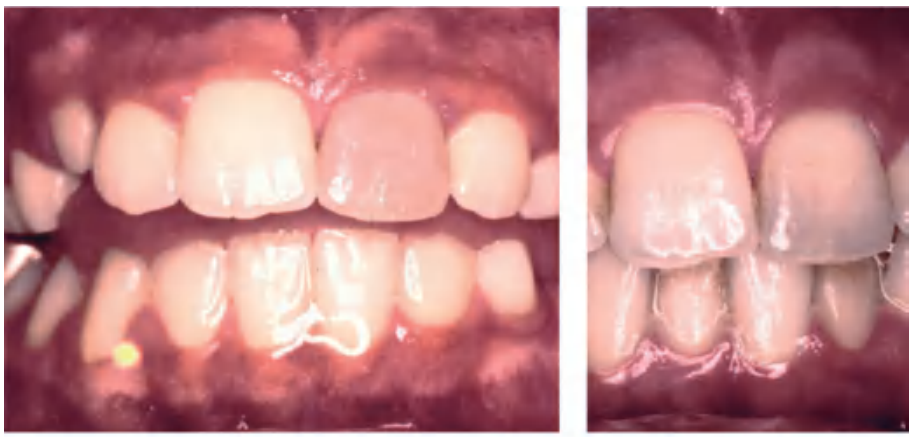
**Fig. 16.7 Color changes following luxation injuries in the permanent dentition**
Gray discoloration 3 months after lateral luxation of a right central incisor.

**Influence of the perpendicular direction of the light beam on the appearance of coronal discoloration**
The color of the left central incisor is almost normal when the beam of light is perpendicular to the long axis of the tooth. Only the palatal surface is discolored, whereas the labial surface of the tooth appears normal.

**Influence of the axial direction of the light beam**
A shift in color to gray appears when the light beam is parallel to the long axis of the same tooth as shown above. Furthermore, a change in translucency is evident.

**Reversibility of gray discoloration**
This laterally luxated left central incisor shows marked gray discoloration which becomes normal at a later follow-up examination.

**Reversibility of red discoloration**
The clinical condition is shown 11 days after injury and at the 5-year followup.



**Yellow discoloration due to pulp canal obliteration**
Marked yellow coronal discoloration seen 10 years after luxation.

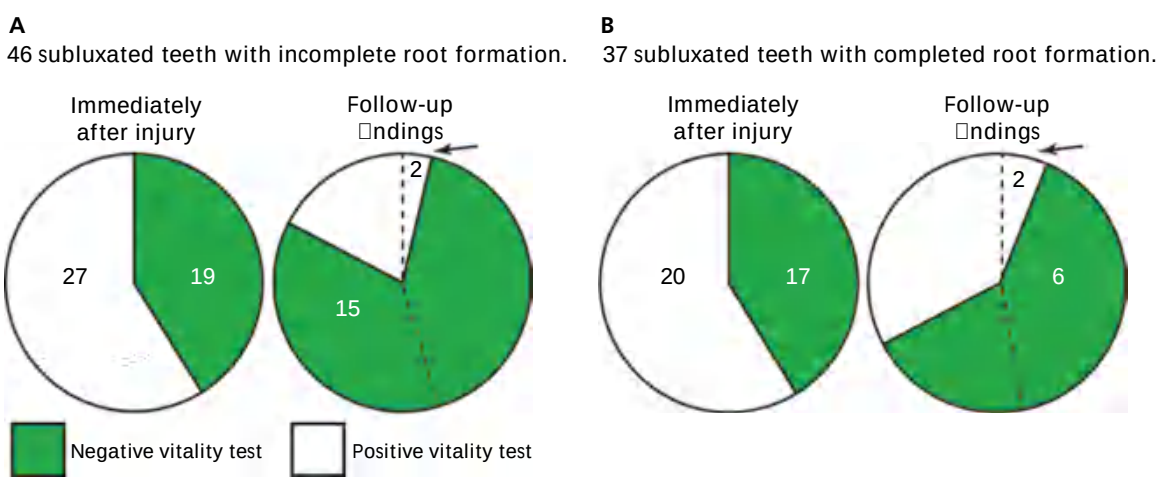



**Fig. 16.8** Diagrams illustrating electrometric sensibility reactions following subluxation of permanent teeth. Reproduced from (7) with permission.

## Electrometric pulp testing

Pulpal sensibility tests have many limitations in diagnosing pulp necrosis after traumatic dental injuries. Immediately following trauma approximately half of the teeth with luxation injuries do not respond to sensibility tests (7, 68, 92, 93) (Fig. 16.8). In a recent clinical study, it was found that a negative response to pulp testing at the time of injury was closely related to the type of pulpal healing achieved. Thus, there were significantly more teeth not reacting to electrical stimulation at the time of injury whose root canals ultimately became obliterated than teeth that survived injury with no radiographic change (93, 98).

An explanation for the temporary loss of normal excitability could be pressure or tension on the nerve fibers in the apical area. If complete rupture of nerve fibers has occurred, a period of at least 36 days is required before a positive response can be expected in immature permanent teeth (31, 68). In mature incisors, the observation period can be much longer (i.e. 1 year or more) prior to a return of a positive sensibility response (7, 30, 56, 69, 95, 98) (Fig. 16.8).

At later follow-up examinations, a previously negative reaction can return to positive, usually within the first 2 months after injury (68); but a period of at least 1 year can elapse before pulp excitability returns (7, 30, 95, 172). While this change in reaction is much more common in teeth with incomplete root formation, longer observation periods can demonstrate restoration of pulpal sensibility in many mature permanent incisors (7, 56, 69, 172) (Fig. 16.8).

In rare cases, a previously positive response can become negative, a phenomenon usually evident within 2 months. However, cases have been reported in which a year or more elapsed before an avital response was recorded (7, 68).

## Pulp testing using laser Doppler flowmetry

Lately, a new method has been developed whereby a laser beam can be directed against the coronal aspect of the pulp. The reflected light scattered by moving erythrocytes undergoes a Doppler frequency shift. The fraction of light scattered back from the pulp is detected and processed to yield a signal. The value of this method in determining pulp vitality has been demonstrated clinically (see Chapter 12) (107–111). However, the equipment necessary for this procedure is quite costly and needs further refinement before it can be of general clinical value. Moreover, it has demonstrated false negative responses in the case of discolored vital teeth (see Chapter 9).

Clearly, the diagnosis of pulp necrosis cannot be based upon sensibility tests alone, but should include additional findings such as progressive gray discoloration of the crown, reaction to percussion, periapical radiolucency or cessation of root development.

## Color changes in the crown

Post-traumatic color change is a well-known phenomenon (9, 31, 32). These changes can range from a lack of translucency to pink, bluish or gray discoloration (Fig. 16.7). Fish (35) offered an explanation for the pathogenesis of some of these changes, suggesting that an injury that was not strong enough to rupture the arteries passing through the apical foramen could occlude or sever the thin-walled veins. Hence, blood continues to be pumped into the canal, causing hemorrhage in the pulp and later diffusion into the hard dental tissues. Another explanation has been that occlusion or rupture of the apical vessels due to trauma leads to ischemia with breakdown of capillaries and subsequent escape of erythrocytes into the pulpal tissue (60, 95, 172).

When the injury displaces the tooth (e.g. intrusive or extrusive luxation), apical vessels are instantly severed with no extravasation of blood into the pulpal tissue and, thus, no immediate discoloration (37). In the case of moderate injury, where disruption of the vascular supply is less than total, ischemia can lead to increased vascular permeability. However, the same phenomenon could occur in the case of healing, as the immature vessels invading the avascular, traumatized pulp lack a basement membrane and are thereby incompetent until vessels mature (112–118). In this case, the ingrowing vessels can still carry blood to pulpal tissues, but blood and blood products could spill out into the stroma (95, 173). In one clinical study, temporary gray coronal discoloration following luxation of mature permanent incisors was described (172). These changes and their normalization could, in some cases, be associated with concomitant loss and restoration of pulpal sensibility, appearance and disappearance of apical radiolucencies and ultimately with pulp canal obliteration. It was suggested that, in the absence of infection, these transient changes could be links in a chain of events leading to pulpal healing. However, if bacteria gained access to the injured avascular pulp, permanent coronal discoloration might then be attributed to autolysis of the necrotic pulp and extravasation of these products into dentin.

Experimental findings indicate that hemoglobin breakdown products enter dentinal tubules (36). This penetration initially alters the crown color to a pinkish hue (Fig. 16.7). As the hemocomponents disintegrate, the color turns bluish which, when seen through the gray enamel, gives a grayish blue tinge. This shift from pink to grayish blue takes approximately 2 weeks in the permanent dentition (9). A certain fading of the gray-blue tint can occur, or an opaque gray hue can persist (9). If the pulp survives, the stain can disappear (34).

Late color changes can occur if the pulp canal becomes obliterated. In these cases, the color of the crown shifts to a yellow hue (3) (Fig. 16.7).

## Radiographic changes

### *Apical radiolucency*

The typical change reflecting a necrotic and infected pulp is a widened periodontal ligament space or an apical radiolucency. These changes may develop as early as 2–3 weeks after injury and are caused by infection-induced release of a number of osteoclast-activating factors (see Chapter 4) (119).

### *Transient apical breakdown*

In recent years, temporary apical radiographic changes in the region of the apical foramen following acute dental trauma and in connection with healing have been described and termed transient apical breakdown (TAB) (Figs 16.4 and 16.5) (119, 139, 140, 172).

It would seem that as a result of displacement of the root after luxation (or of the coronal fragment of a root fracture), the vascular supply at the apical foramen (or 'fracture foramen' in a root fracture) could be partially or totally severed, leading to ischemic changes in the pulp. This would in turn lead to wound healing response and release of osteoclast-activating factors (see Chapter 7). Subsequent resorption of hard tissue (dentin, cementum and alveolar bone), presumably at the interface between the vital and necrotic tissues, could then result in either (1) an increase in the periodontal ligament space immediately adjacent to the

injured apical foramen; or (2) an increase in the diameter of the apical foramen in the case of extrusion or lateral luxation of teeth with completed root formation or at the level of fracture in the coronal pulp canal following horizontal root fracture. This event might be seen as a way to gain space to accommodate vascular ingrowth into the root canal. Presumably once repair has reached the remodeling phase, the resorption processes resolve. This can be seen as a normalization of the radiographic condition within the root canal (i.e. hard tissue deposition in the previous resorption process and occasionally pulp canal obliteration) and restoration of a normal apical periodontal ligament space (93, 95, 119, 139, 140, 172, 173) (Figs 16.4 and 16.5).

It might be speculated whether delay in treatment after the appearance of an apical radiolucent zone would worsen the prognosis of future endodontic therapy. However, it has been found that the success rate of endodontic therapy of teeth demonstrating periapical radiolucencies can be equal to the success rate of teeth treated before the development of manifest radiographic change, at least when the former are initially treated with calcium hydroxide prior to final gutta-percha root filling (versus immediate gutta-percha root filling) (120). Thus, it appears that there would be limited risk of healing complications in the event of late endodontic therapy (i.e. up to 1 year after injury), delayed in anticipation of pulpal repair in selected patient categories (i.e. extrusive and lateral luxation of mature incisors). A possible risk is the development of progressive root resorption in the observation period. However, this risk would appear to be minimal, as TAB was not seen in teeth with immature root formation, probably because with an open apex there is space enough for ingrowth of new vessels and nerves (95).

### Guidelines for the diagnosis of irreversible pulp necrosis

The diagnosis of pulp necrosis should be based primarily on two or more of the following signs: crown discoloration, negative sensibility testing (electrometric and/or laser Doppler) and periapical radiolucency. In this regard, the importance of transient negative sensibility testing should be considered (i.e. a minimum of 2–3 months' observation). If these criteria are used, a certain percentage of mistakes will be made where pulps under revascularization are extirpated. The risk of this phenomenon is possibly about one in 10 cases (90) (Fig. 16.9).

Further details concerning when endodontic intervention is appropriate are presented in Chapter 18.

### Internal bone formation (pulp bone)

In cases with severe injury to both the pulp and the HERS, invasion of bone and periodontal ligament into the pulpal cavity may be seen (see Chapter 2) (135).

### Guidelines for observation without endodontic intervention

In selected cases, a conservative approach might be indicated, whereby active treatment can be avoided (95, 172).

As long as the following conditions are met, a tooth with one or more signs of pulp necrosis can be observed for pulpal repair over a longer period of time (up to approximately 1 year):

(1) The patient is at low risk of inflammatory resorption (95, 121, 172).
(2) The injury type and stage of root development (extrusion and lateral luxation of teeth with completed root development) imply that transient apical changes might be anticipated (95).
(3) Neither prosthetic treatment nor orthodontic therapy is planned to immediately involve the injured tooth. As the clinical and radiographic signs previously described are assumed to reflect healing processes within the pulp, presumably because of a temporary disruption of the neurovascular supply, the additional trauma of crown preparation or orthodontic tooth movement might be anticipated to shift the balance of pulpal survival unfavorably.
(4) As it is not presently possible to adequately evaluate pulpal status following dental luxations, the possibility of *asymptomatic* sterile pulp necrosis must not be ignored. It is therefore of utmost importance that the patient be thoroughly informed regarding the diagnostic problems involved and the consequent need for extra follow-up examinations – and is willing and able to cooperate. If there is any doubt as to the possibility of recalling the patient, endodontic therapy rather than 'observation therapy' should be the treatment of choice (95, 172).
(5) Consider the patient's individual needs. People who are routinely exposed to extreme changes in air pressure (e.g. airline personnel, professional divers) are at risk of barodontalgia, which could induce severe dental pain in teeth with pulpal change. Teeth demonstrating the above-mentioned changes in such individuals should be treated endodontically and not merely monitored.

### Treatment of pulp necrosis

This subject is discussed in detail in Chapter 25.

## Pulp canal obliteration

Pulp canal obliteration is related to the process of pulpal revascularization after tooth luxation. In that respect, the speed of obliteration appears to be associated with the degree of dislocation at the time of injury (Fig. 16.10).

Pulp canal obliteration can be regarded as a response to a severe injury to the neurovascular supply to the pulp which, after healing, leads to accelerated dentin deposition and is frequently encountered after luxation injuries of permanent teeth (3, 20, 27, 28, 40, 41, 62–64, 75–77, 93, 97, 100,

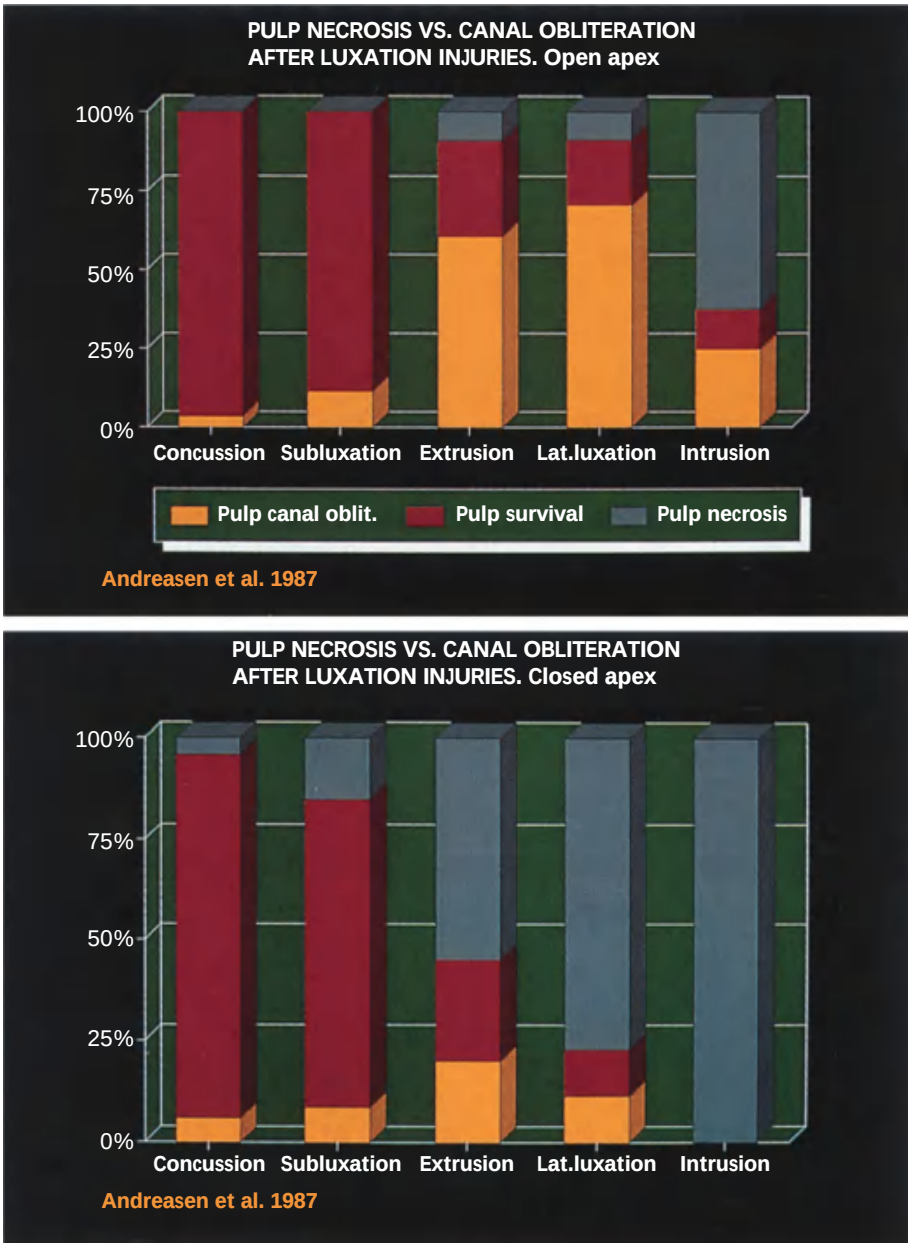



**Fig. 16.9** Relationship between pulp canal obliteration, pulp survival with no radiographic change and pulp necrosis in teeth with incomplete and complete root formation (open or closed apex). Reproduced from (93) with permission.

122–124) (Table 16.5). Pulp canal obliteration is strongly related to the severity of the luxation injury, being especially common in severely mobile or dislocated teeth (62, 93, 97). It would appear to be a phenomenon that is closely related to the loss and re-establishment of pulpal neural supply (98), as it is rarely seen when there is no tooth displacement and thus little damage to the pulpal neurovascular supply (i.e. concussion and subluxation). Moreover, this complication mainly involves teeth injured before completion of root formation (3, 93) (Fig. 16.9).

A clinical manifestation of pulp canal obliteration is a yellow discoloration of the crown (Fig. 16.7). Response to thermal sensibility tests has been reported to be lowered or absent (42, 68); and response to electrical stimulation is also reported as decreased and often absent (42, 63, 75). However, one clinical investigation could find no difference in sensibility threshold between paired incisors with and without pulp canal obliteration up to 5 years after injury (93). Nevertheless, with longer observation periods there is a tendency to higher thresholds or absence of reaction in pulp canal obliterated teeth.

The first radiographic sign of obliteration is reduction in the size of the coronal pulp chamber, followed by gradual narrowing of the entire root canal, occasionally leading to partial or complete obliteration (Fig. 16.10). However, histologic examination of these teeth always shows a persisting narrow root canal. Normalization of temporary apical radiolucencies, as described earlier, has been found to be significantly related to pulp canal obliteration in mature permanent incisors. This apparently reflects the wound healing processes involved in revascularization and reinnervation of the injured pulp (95) (Fig. 16.4).

Pulp canal obliteration usually appears between 3 and 12 months after injury (Fig. 16.10). It has been shown that two

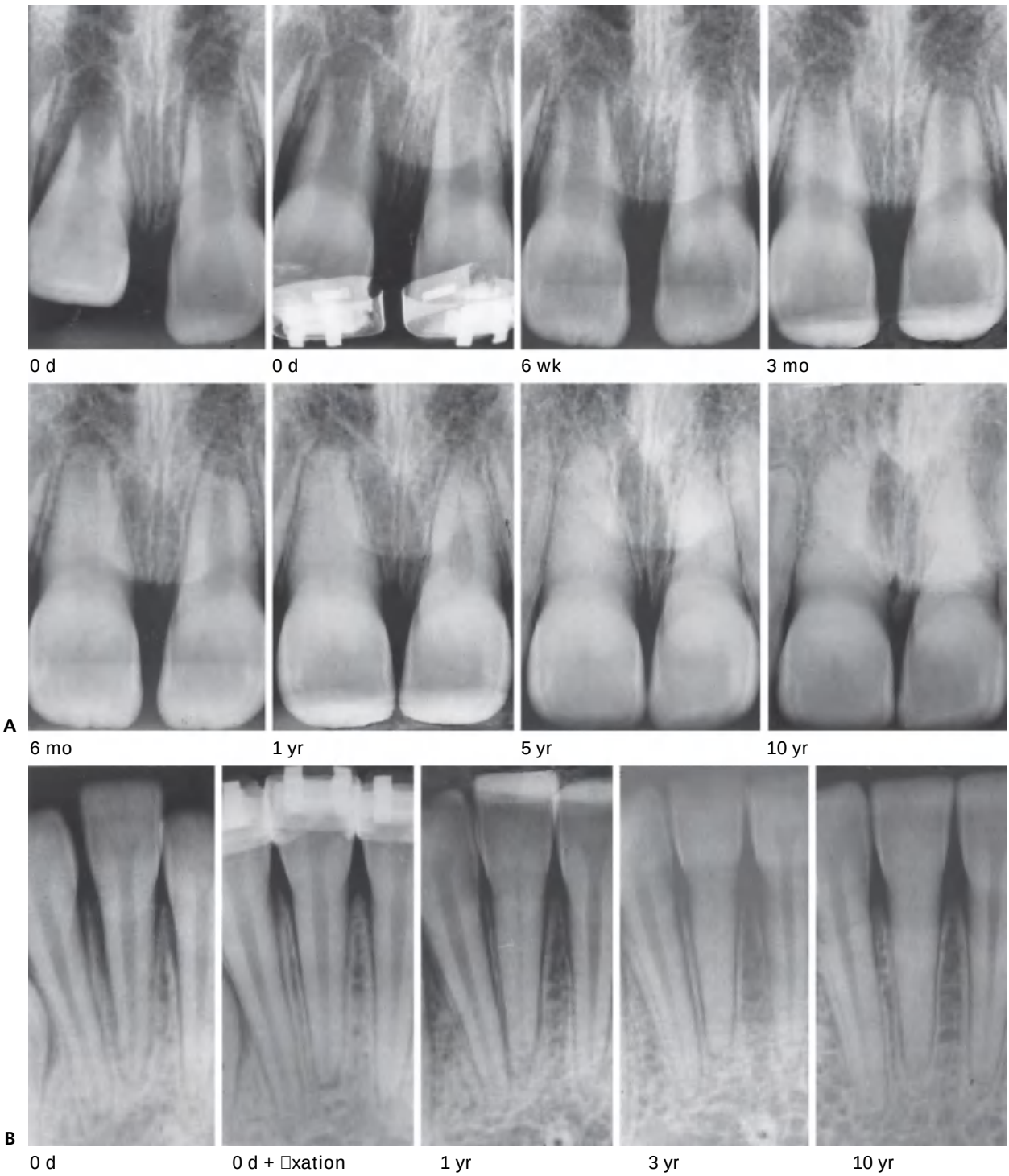



**Fig. 16.10** A. Radiographic illustration of the various stages in pulp canal obliteration (PCO). Extrusive luxation of the maxillary right central incisor and avulsion of the maxillary left central incisor in a 6-year-old boy. Onset of PCO is seen 6 months after injury. Total PCO of the right incisor and partial PCO of the left incisor is found 1 year after injury. B. Radiographic demonstration of the various stages in PCO. Lateral luxation of a mandibular right central incisor in a 10-year-old boy. Partial PCO is seen 1 year after injury and total PCO after 3 and 10 years. Reproduced from (93) with permission.

types of pulp canal obliteration exist. In *partial canal obliteration*, the coronal part of the pulp chamber is not discernible, while the apical part is markedly narrowed but still discernible (Figs 16.10 and 16.11). In *total canal obliteration*, the pulp chamber and root canal are hardly (or not at all) discernible (63).

As with pulp necrosis, pulp canal obliteration is determined by the *type of luxation injury* and stage *of root development* at the time of injury (93, 141) (Fig. 13.9). Pulp canal obliteration appears to be more frequent after luxation injuries with displacement and in teeth with incomplete root formation (93, 141) (Fig. 13.9).

In contrast to pulp necrosis, where no treatment effect at the time of injury could be established, pulp canal obliteration was found to be significantly related to the type of fixation

used (93). Prior to the use of acid-etched splinting techniques, orthodontic bands were cemented onto the traumatized and adjacent teeth and united with self-curing acrylic (Fig. 16.12). There was a significant relationship between the use of this type of splint and this healing complication, presumably reflecting additional injury to the already traumatized periodontium due to forceful placement of the bands. In some cases, displacement of initially non-displaced traumatized incisors could be demonstrated after orthodontic band/acrylic fixation (Fig. 16.12).

A late complication following pulp canal obliteration is the development of pulp necrosis and the occurrence of periapical changes, and this has been found to occur in 7–16% of cases (3, 42, 43, 63, 75, 141) (Table 16.6, Figs 16.13 and 16.14). The pathogenesis of this complication is still obscure (141). Minor injuries are probably able to sever the vulnerable vascular supply at the constricted apical foramen, or the pulp vessels are progressively occluded due to hard tissue formation. Caries and restorative treatment have been suspected to lead to pulp necrosis; but no certain link could be demonstrated in a study done in 1996 (141) (Table 16.6). Several clinical investigations have recorded secondary pulp necrosis following pulp canal obliteration in 7–16% of cases. However, no distinction was made between new trauma, secondary caries and/or crown preparation. A clinical investigation with an average observation period of up to 10 years reported a frequency of 9% (141). No clear relationship could be made to factors, such as dental caries, crown preparation, orthodontics or new trauma. However, a worldwide general annual trauma incidence of approximately 1% might suggest that new traumas do present a hazard for patients with a compromised dental pulp. It has also been reported that secondary pulp necrosis was only found in teeth with total canal obliteration and especially in those teeth which showed rapid obliteration after injury (i.e. total canal obliteration within 2 years after injury). Moreover, complicating pulp necrosis was common in teeth with completed root formation and with severe periodontal injury at the time of trauma (63).

While early prophylactic pulp extirpation and endodontic intervention can prevent periapical lesions, the rather low frequency of this complication does not support such early intervention measures (93, 126–129). The endodontic considerations of pulp canal obliteration are further discussed in Chapter 25.

**Table 16.5** Frequency of pulp canal obliteration after luxation injuries irrespective type of luxation.

| Study | No. of teeth | No. with pulp canal obliteration |
|---|---|---|
| Andreasen (3) 1970 | 189 | 42 (22%) |
| Gröndahl et al. (74) 1974 | 320 | 77 (24%) |
| Herforth (68) 1976 | 161 | 57 (35%) |
| Stålhane & Hedegård (62) 1975 | 1116 | 67 (6%) |
| Rock & Grundy (97) 1981 | 517 | 83 (16%) |
| Wepner & Bukel (125) 1981 | 142 | 12 (8%) |
| Lawnik & Tetsch (122) 1983 | 246 | 20 (8%) |
| Posukidist & Lehmann (123) 1983 | 162 | 25 (15%) |
| Andreasen et al. (93) 1987 | 637 | 96 (15%) |
| Oikarinen et al. (100) 1987 | 147 | 40 (27%) |
| Herforth (101) 1990 | 319 | 106 (33%) |
| Crona-Larsson et al. (136) 1991 | 103 | 3 (3%) |
| Robertson (137) 1997 | 196 | 36 (18%) |

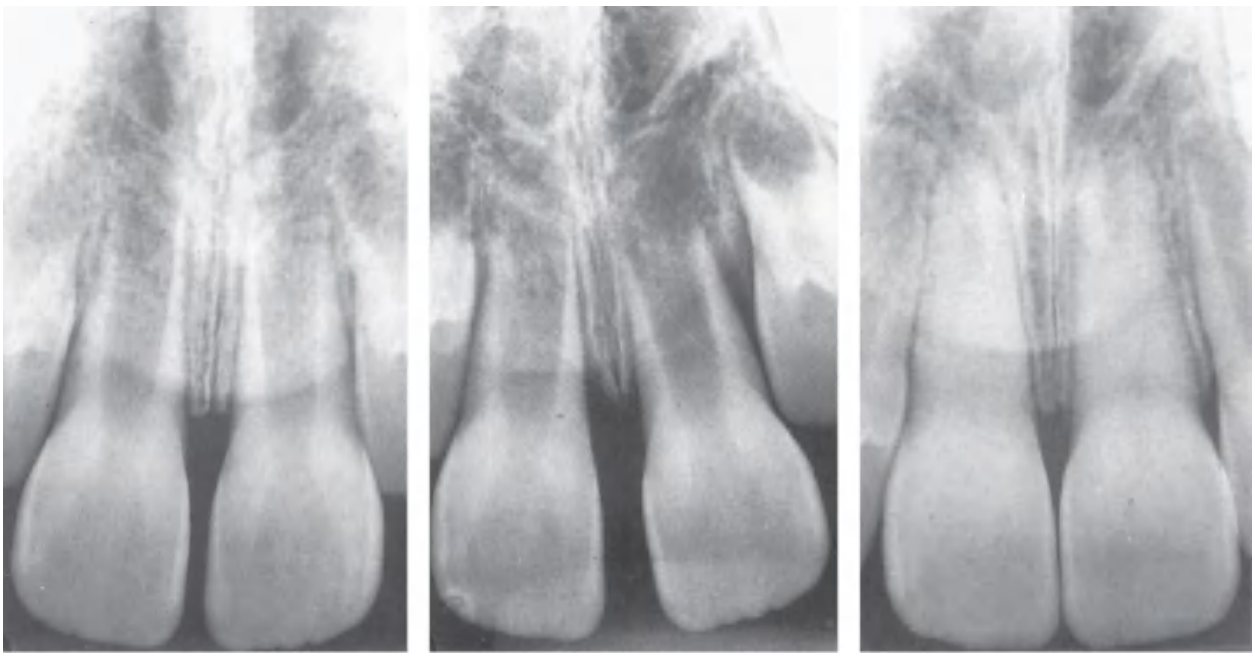



**Fig. 16.11** Partial pulp canal obliteration after extrusion of two central incisors. Partial canal obliteration is evident after 1 year.

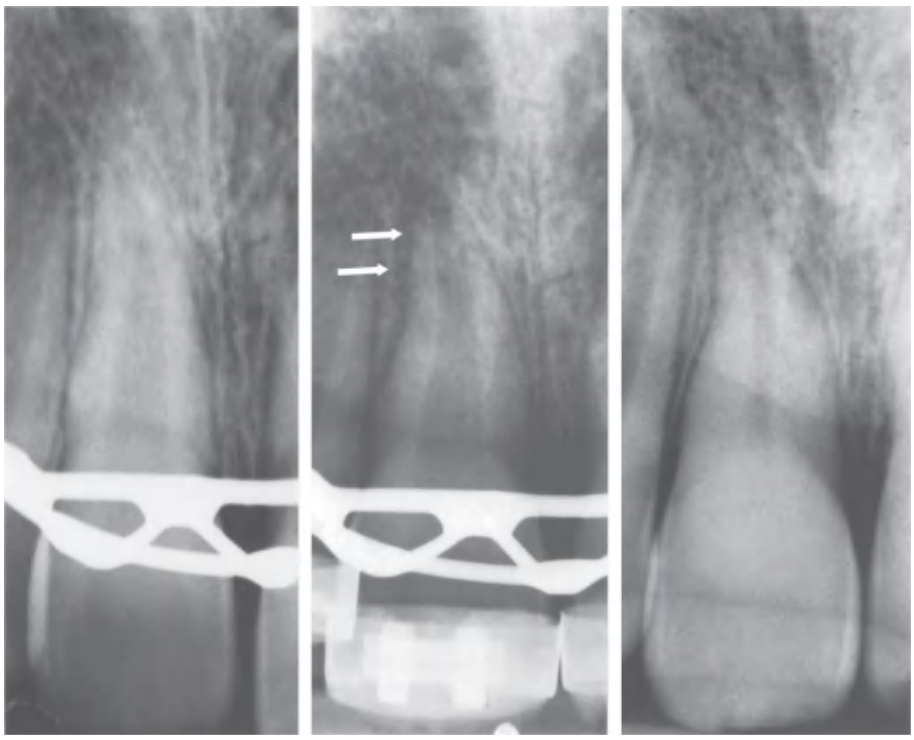



**Fig. 16.12** Canal obliteration as a probable effect of orthodontic splinting, showing a subluxated maxillary right central incisor in a 9-year-old girl. After orthodontic band fixation, it can be seen that the root apex is displaced. There is pulp canal obliteration 1 year after injury. Reproduced from (93) with permission.

**Table 16.6** Frequency of necrosis secondary to pulp canal obliteration in permanent teeth irrespective of type of luxation injury.

| Study | Observation period, years (mean) | No. of teeth | Pulp necrosis |
|---|---|---|---|
| Holcomb & Gregory (42) 1967 | 4 | 41 | 3 (7%) |
| Andreasen (3) 1970 | 1–12 (3.4) | 42 | 3 (7%) |
| Stålhane (75) 1971 | 13–21 | 76 | 12 (16%) |
| Jacobsen & Kerekes (63) 1977 | 10–23 (16.0) | 122 | 16 (13%) |
| Robertson et al. (141) 1996 | 7–22 (16) | 82 | 7 (9%) |

### Internal bone formation (pulp bone)

In cases with severe injury to both the pulp and the HERS, invasion of bone and periodontal ligament into the pulpal cavity may be seen (see Chapter 2) (135).

### Treatment delay

Delay in treatment seems to play a questionable role in the frequency of healing complications. In cases of alveolar fractures, treatment delay appears to be related to pulp necrosis and root resorption (6). Concerning jaw fractures, four studies showed no significant difference between immediate and delayed treatment (66, 84–86). One study showed a preference for healing for cases treated within 3 days (83), whereas another study indicated that treatment times between 3 and 5 days were optimal, with the lowest rate of complications (56). In conclusion, there is presently no strong evidence for either acute or delayed treatment of mandibular fractures in order to minimize healing complications; new studies including a number of cases treated on an acute basis are needed (87).

## Root resorption

A late complication following luxation injuries in the permanent dentition is root resorption (26, 29, 34, 44, 55, 62, 92, 96, 119, 130) (Table 16.7). The diagnosis of root resorption is entirely dependent upon a radiographic examination. *In vitro* studies have shown that radiographic demonstration of defects on the root surface is dependent upon the size and position of the defect, with 0.6 mm being the critical size (149). Subtraction radiography does not seem to provide additional information (142, 143).

Root resorption can be classified into various types such as root surface resorption and root canal resorption (Figs 16.15 and 16.16).

### Root surface resorption (external root resorption)

The damage inflicted to the periodontal structures and the pulp by luxation injuries can result in various types of *root surface resorption*. The etiology and pathogenesis of these complications seem to be identical to root resorption following replantation of avulsed teeth (see Chapter 17). Three types of external root resorption can be recognized (Fig. 16.15).

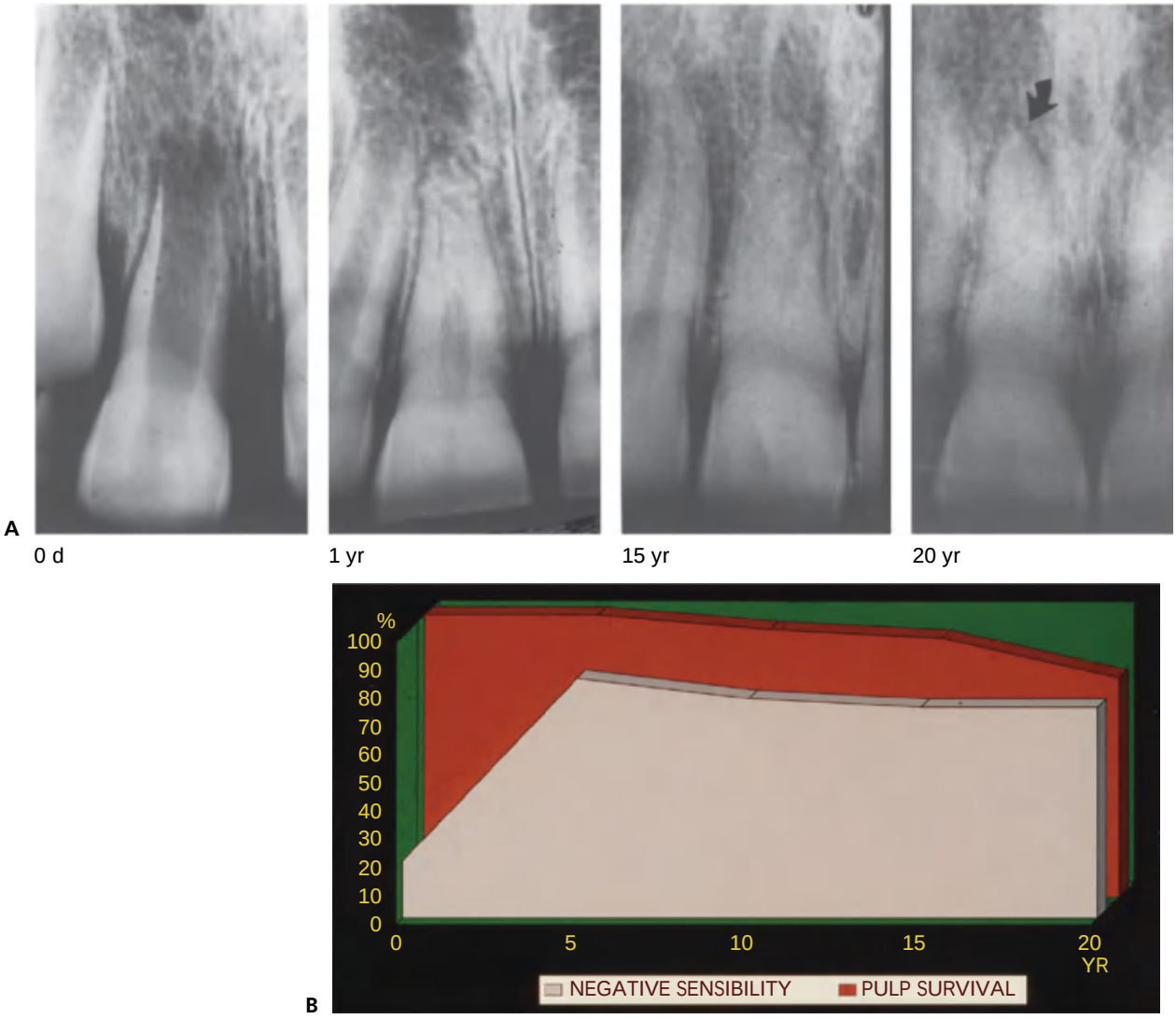



**Fig. 16.13** A. Pulp canal obliteration (PCO) followed by secondary pulp necrosis. The extruded central incisor is seen to develop PCO. At the 20-year follow-up examination, pulp necrosis and a periapical rarefaction have developed. B. Pulp survival in relation to canal obliteration. Reproduced from (141) with permission.

In summary, *external root resorption* is most commonly seen after intrusive luxation. However, this can also be found following severe lateral luxations (92). Concussion and subluxation show the lowest frequency of resorption, primarily in the form of surface resorption, and intrusion the highest frequency and with frequent occurrence of inflammatory and replacement resorption (3, 7, 55, 62, 92, 96) (Table 16.8). This reflects a correlation between the degree of injury to the periodontal structures and root resorption (see Chapter 2).

For treatment of external root resorption, see Chapter 25.

### *Repair-related resorption (surface resorption)*

The root surface shows superficial resorption lacunae repaired with new cementum. These lacunae have been termed surface resorption. It has been suggested that they occur as a response to a localized injury to the periodontal ligament or cementum (3, 45). In contrast to other types of resorption, surface resorption is not progressive, but is self-limiting and shows spontaneous repair. Surface resorptions are not usually seen on radiographs due to their small size; however, it is sometimes possible to recognize small excavations of the root surface delineated by a normal lamina dura (3) (Fig. 16.17). When visible, these resorption cavities are usually confined to the lateral surfaces of the root, but may also be found apically, resulting in a slight shortening of the root.

### *Infection-related resorption (inflammatory resorption)*

Histologically, bowl-shaped areas of resorption of both cementum and dentin are seen together with inflammation of adjacent periodontal tissue. The inflammation and resorption activity are apparently related to the presence of infected, necrotic pulp tissue in the root canal (Fig. 16.18). Radiographically, root resorption with an adjacent radiolucency in bone is the typical finding (3) (Fig. 16.18).

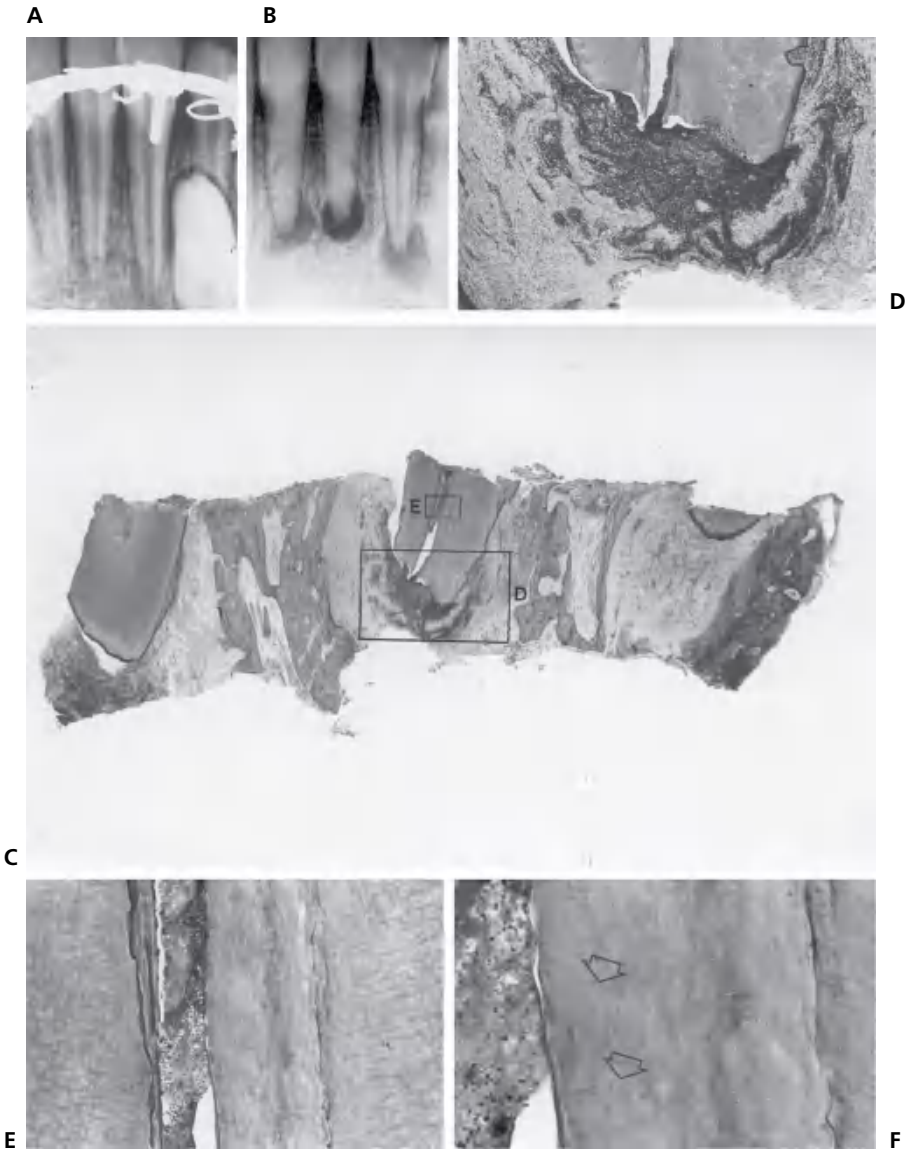



**Fig. 16.14** Extrusive luxation of permanent incisors leading to pulp obliteration and complicating pulp necrosis. A. Condition immediately after reduction and fixation. B. At follow-up 12 years later pulp canal obliteration and periapical inflammation are evident. C. Histologic specimen including apex of left central incisor. ×10. D. Periapical inflammation. ×30. E. Extensive hard tissue formation following injury. The pulp tissue is necrotic. ×75. F. Higher magnification of E. A few dentinal tubules in the post-traumatic hard tissue can be recognized (arrows). ×195.

**Table 16.7** Frequency of progressive root resorption after luxation of permanent teeth irrespective type of luxation.

| Study | No. of teeth | Progressive root resorption (ankylosis- and infection-related resorption) |
|---|---|---|
| Skieller (7) 1960 | 107 | 6 (6%) |
| Andreasen (3) 1970 | 189 | 21 (11%) |
| Stålhane & Hedegård (62) 1975 | 1116 | 16 (1%) |
| Rock & Grundy (97) 1981 | 517 | 37 (8%) |
| Andreasen & Vestergaard Pedersen (92) 1985 | 637 | 47 (7%) |
| Oikarinen et al. (100) 1987 | 147 | 27 (18%) |
| Crona-Larsson et al. (136) 1991 | 104 | 15 (14%) |

### *Ankylosis-related resorption (replacement resorption)*

Ankylosis is a rare finding after luxation injuries except for intrusive luxations (Table 16.8). A direct union between bone and root substance is seen, with the root substance gradually being replaced by bone (Figs 16.19 and 16.20). Disappearance of the periodontal space and progressive root resorption are typical radiographic findings (3) (Fig. 16.19).

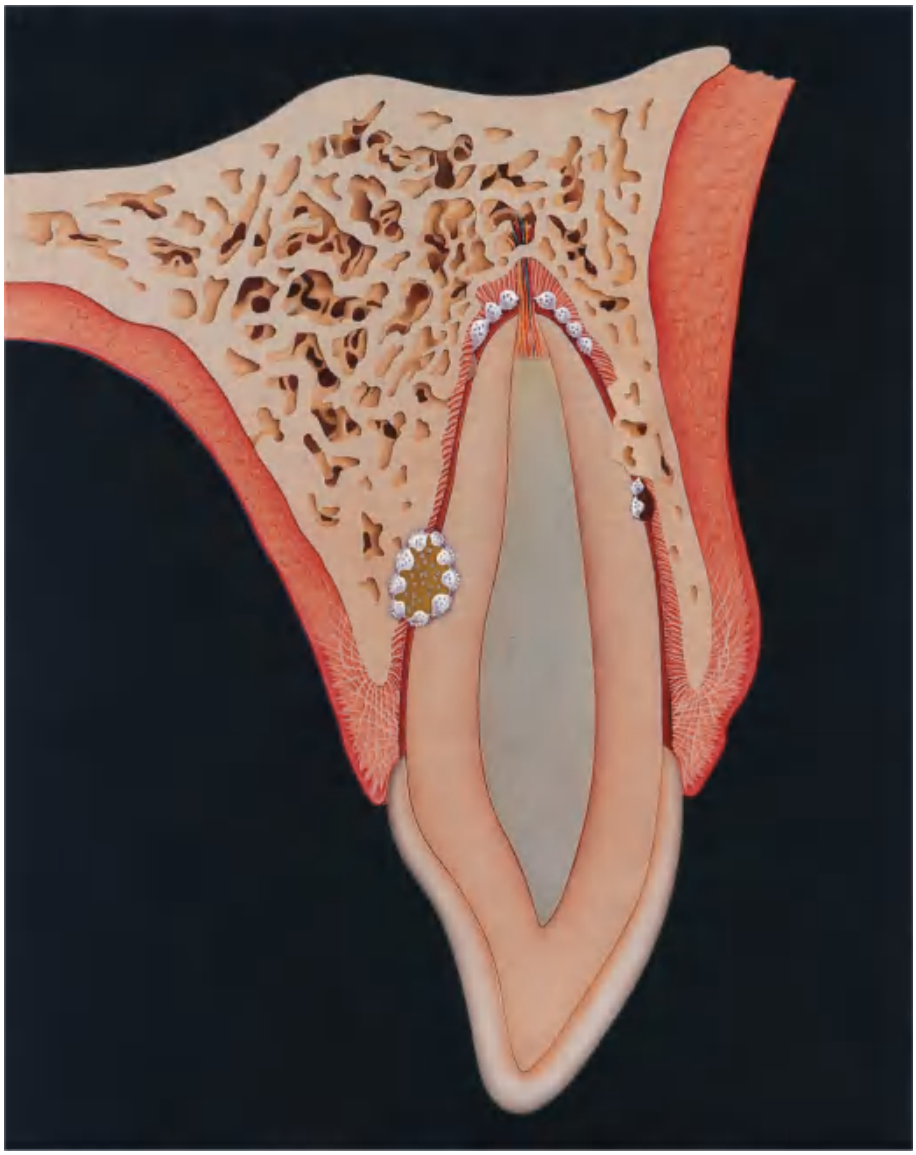


**Fig. 16.15** External root repair-related resorption. Three types of root surface (external) resorption may develop subsequent to trauma: repair-related resorption, infection-related resorption and ankylosis. From (119).

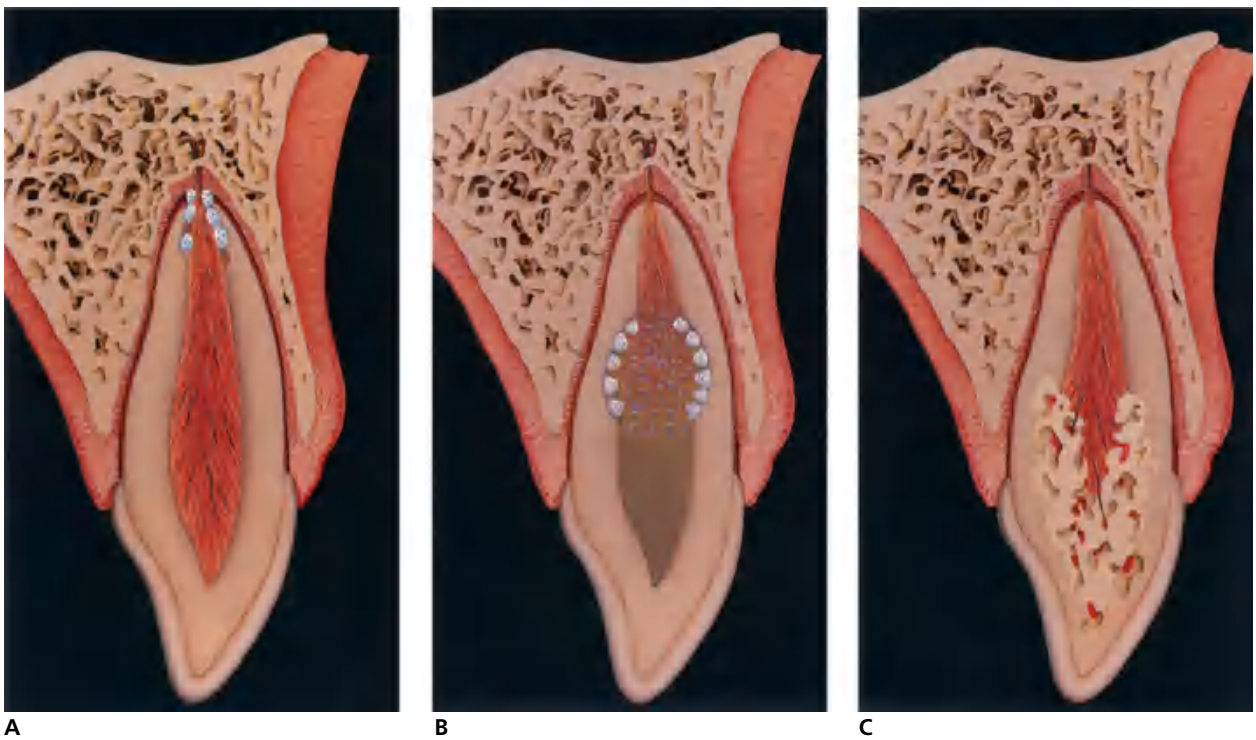



**Fig. 16.16** Root canal resorption. Three types of root canal (internal) resorption may develop subsequent to trauma: internal repair-related resorption (A), internal infection-related resorption (B) and internal ankylosis (C). From (119).

**Table 16.8A** Risk of external root resorption related to luxation diagnosis for permanent teeth with *immature* root development estimated after 10 years. Reproduced from (168) with permission.

| | No. of teeth | Repair-related resorption | Infection-related resorption | Ankylosis-related resorption |
|---|---|---|---|---|
| Concussion | 62 | 0% | 0% | 0% |
| Subluxation | 155 | 0% | 0% | 0% |
| Extrusive luxation | 34 | 0% | 2.9% (95% CI: 0–8.5) | 0% |
| Lateral luxation | 43 | 2.3% (95% CI: 0–6.7) | 0% | 0% |
| Intrusive luxation | 18 | 15 (24%) | 33% (95% CI: 13–53.8) | 5.6% (95% CI: 0–8.5) |

**Table 16.8B** Prevalence of external root resorption related to luxation diagnosis for permanent teeth with *mature* root development. Reproduced from (168) with permission.

| | No. of teeth | Repair-related resorption | Infection-related resorption | Ankylosis resorption |
|---|---|---|---|---|
| Concussion | 115 | 0% | 0% | 0% |
| Subluxation | 112 | 2.7% (95% CI: 0–5.7) | 0% | 0% |
| Extrusive luxation | 23 | 27% (95% CI: 9.5–44.5) | 0% | 0% |
| Lateral luxation | 103 | 33.6% (95% CI: 23–44.2) | 3.3 (95% CI: 0–8.2) | 1% (95% CI: 0–2.9) |
| Intrusive luxation | 21 | 5% (95% CI: 0–14.5) | 4.8% (95% CI: 0–13.9) | 37.5 (95% CI: 11–64) |

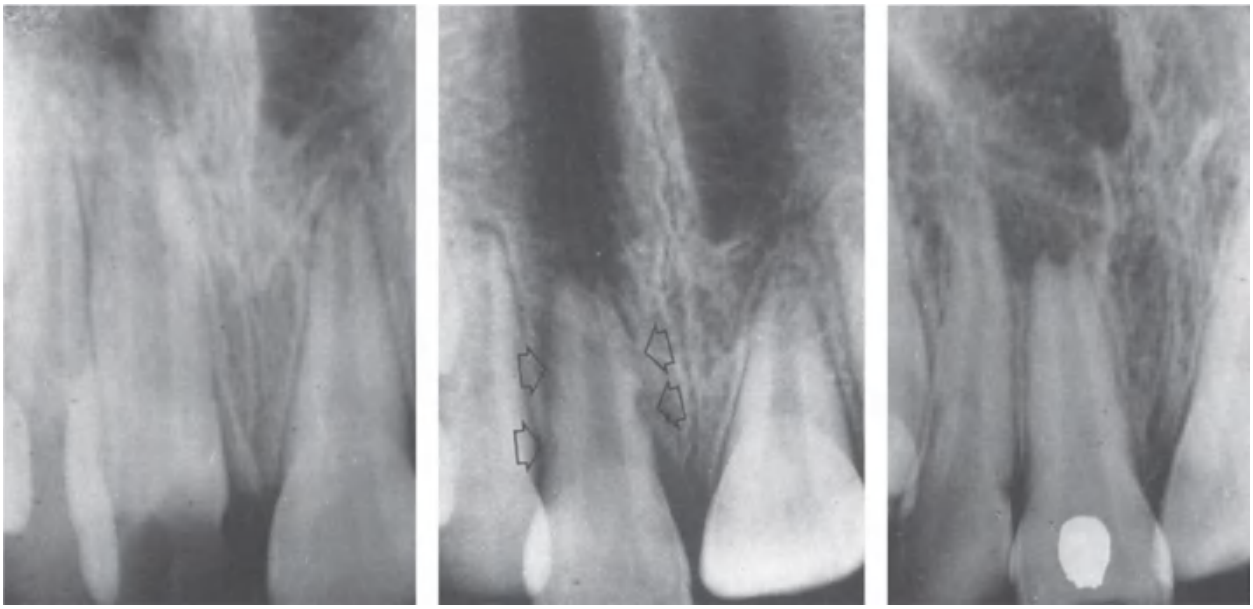



**Fig. 16.17** Repair-related resorption following intrusive luxation of a right central incisor. Six weeks after injury multiple small resorption cavities are evident along the root surface (arrows). One year after injury repair is evident with re-establishment of the periodontal space adjacent to the resorption areas. Reproduced from (3) with permission.

## Cervical invasive resorption

This resorption entity can have many etiologies, with trauma being one of the most frequent, as well as orthodontic treatment (144, 145). Bleaching has also been found to lead to this complication (see Chapter 25) (144, 145).

## Root canal resorption (internal root resorption)

*Root canal resorption* is a rather unusual finding (28, 46, 47, 49, 119, 142) and has been recorded in only 2% of re-examined luxated permanent teeth (3). This resorption type is also seen in the primary dentition (78). In the radiographic diagnosis of internal root resorption, one must consider that root surface resorption located labially or lingually on the root can mimic internal resorption as it is superimposed over the root canal (79–81). It is therefore necessary to take supplementary mesial and distal eccentric radiographs of the root. If the resorption cavity does not change position, it is located centrally in the tooth indicating root canal resorption. If the outline of the root canal is intact, then it is external resorption.

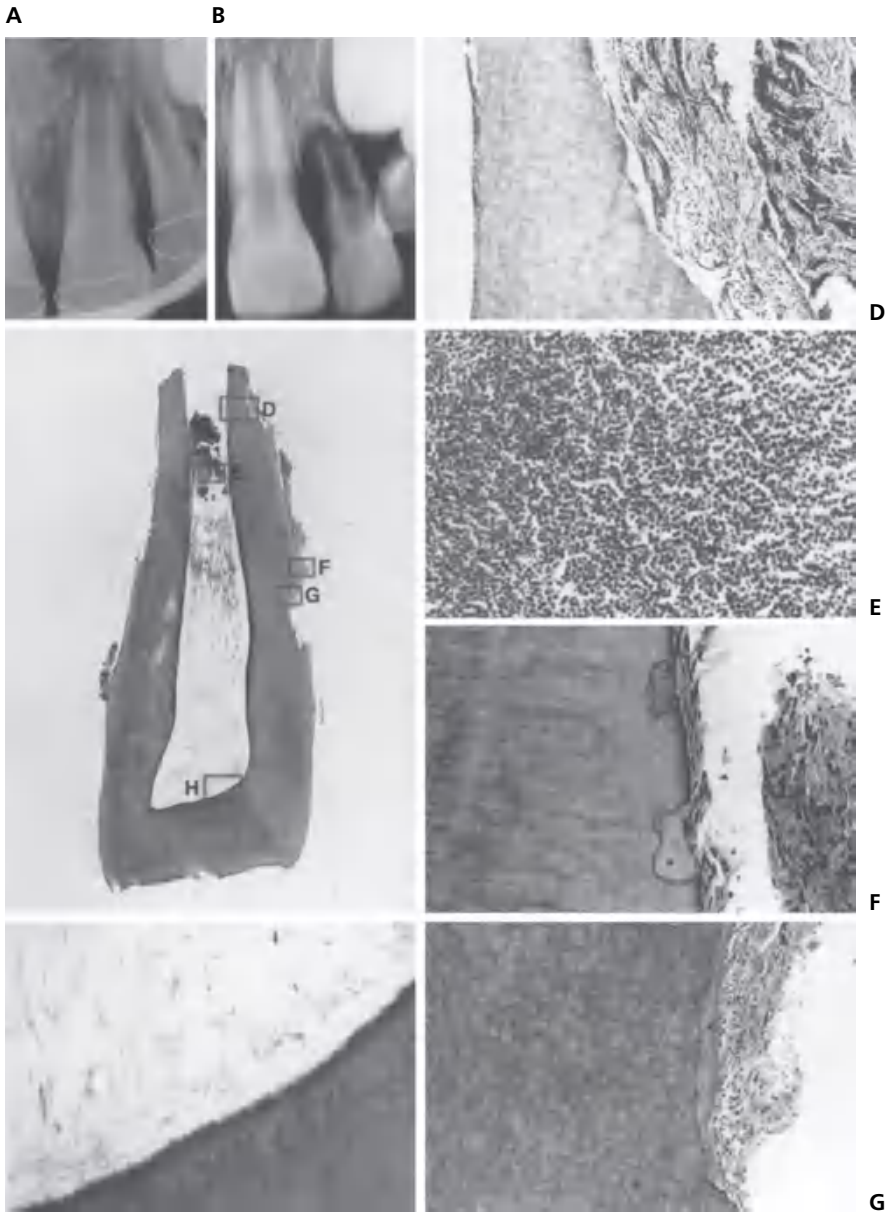



**Fig. 16.18** Infection-related resorption following extrusive luxation of a left lateral incisor. A. Condition immediately after reduction and splinting. B. Six months later. Note periradicular radiolucency as well as root resorption. C. Low power view of sectioned incisor removed 6 months after injury. ×7. D. Resorption area with inflammation in the connective tissue and epithelial proliferation. ×75. E. Abscess bordering coronal necrotic pulp tissue. ×195. F and G. Repair-related resorption repaired with new cementum. ×195. H. Complete autolysis of pulp tissue. ×75.

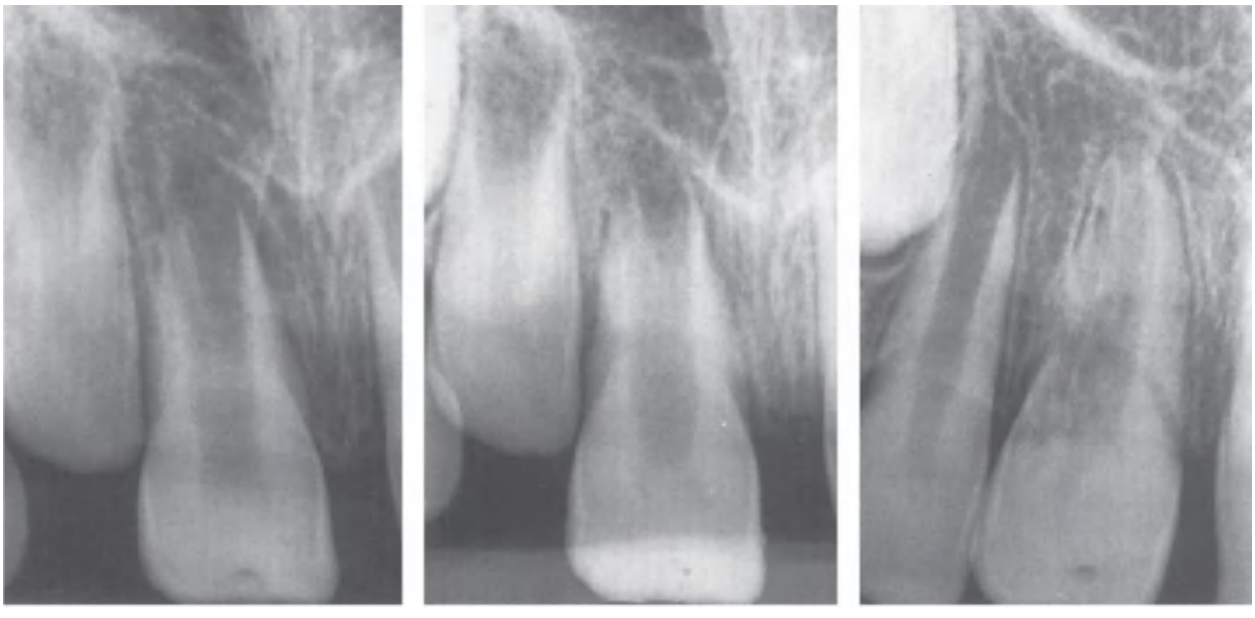



**Fig. 16.19** External ankylosis-related resorption as a sequel to lateral luxation. At the time of removal of the splint 1 month later there is no evidence of root resorption. At the 1 year follow-up, ankylosis is evident distally.

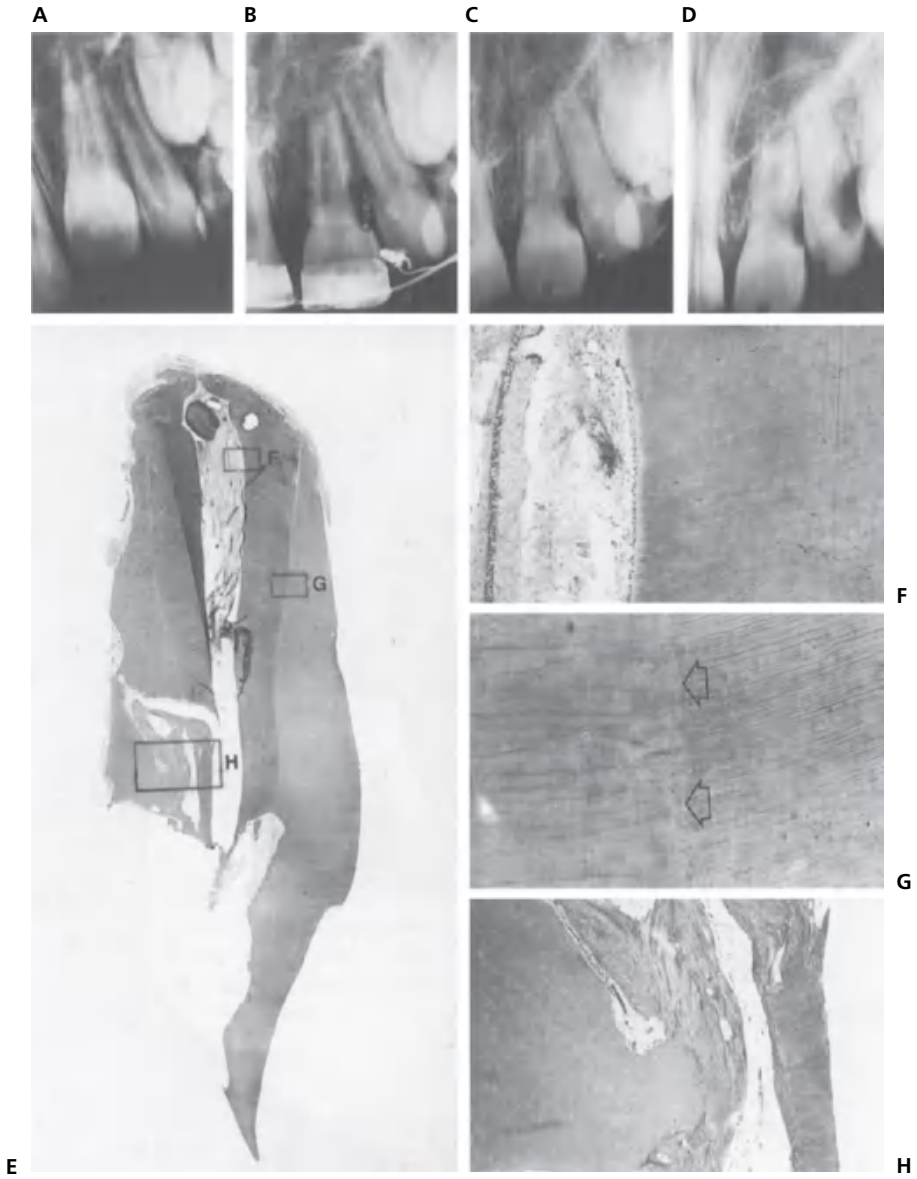



**Fig. 16.20** Ankylosis-related resorption following intrusive luxation of the left permanent incisors. A to D. Progression of ankylosis-related resorption is seen over a 2.5-year period. At this time both incisors were extracted. E. Low power view of sectioned lateral incisor. ×8. F. Vital pulp tissue. ×75. G. Marked post-traumatic dentin formation containing few dentinal tubules (arrows). ×75. H. Ankylosis area with apposition of bone. ×30.

Root canal resorption can be classified as follows (Fig. 16.16).

### *Root canal ankylosis (replacement resorption)*

This resorption type is characterized radiographically by an irregular enlargement of the pulp chamber (48, 49) (Fig. 16.21). A variant has been termed *internal tunneling resorption*. This resorption is usually found in the coronal fragment of root fractures (131), but may also occur after luxation (Fig. 16.22). The typical feature of this resorption is a tunneling resorption process next to the root canal. After some time the resorption process becomes arrested and complete pulp canal obliteration takes place.

Histologically, the teeth show metaplasia of the normal pulp tissue into cancellous bone. The continuous rebuilding of bone at the expense of dentin is responsible for the gradual enlargement of the pulp chamber (15, 50). This resorption type should just be observed as they tend not to progress (Fig. 16.22).

### *Root canal infection-related resorption*

This resorption type is radiographically characterized by an oval-shaped enlargement within the pulp chamber (51–54, 65, 67, 169) (Fig. 16.23). This type of resorption is usually found in the cervical aspect of the pulp. If initial resorption is located in the apical aspect, it is often a sign of active revascularization (internal surface resorption) and not inflammatory resorption (95, 98, 131).

Histologically, a transformation is seen of normal pulp tissue into granulation tissue with giant cells resorbing the dentinal walls of the root canal, advancing from the dentinal surface towards the periphery (Fig. 16.23). A zone of necrotic pulp tissue is usually found coronal to the resorbing tissue. This necrotic zone, or dentinal tubules containing bacteria,

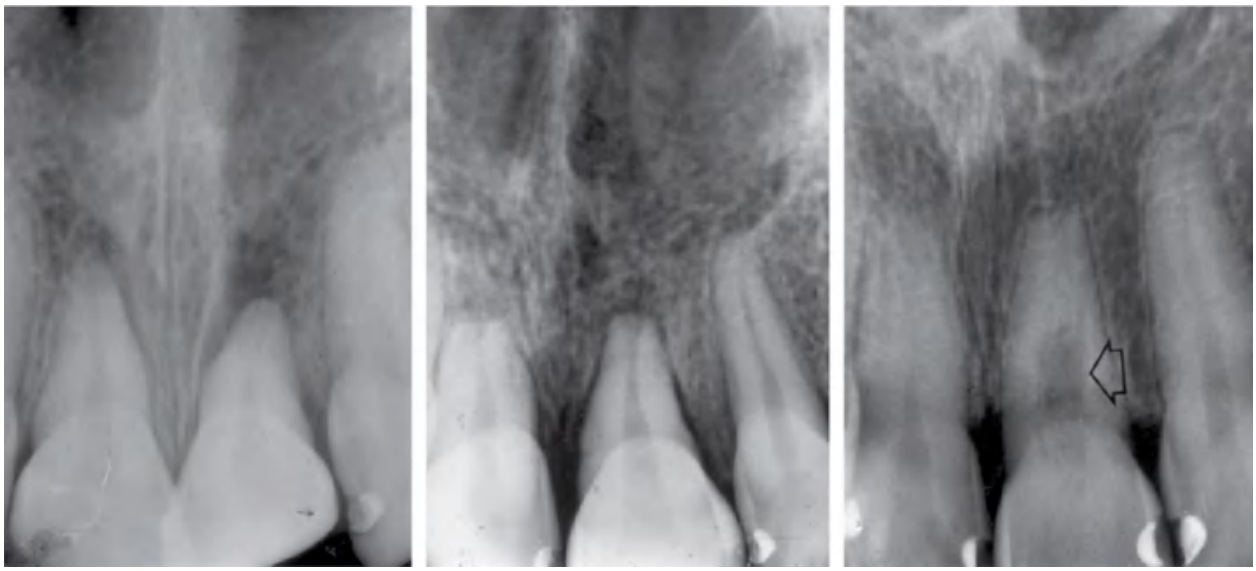



**Fig. 16.21** Internal ankylosis-related resorption as a sequel to extrusive luxation. At re-examination 10 years later internal ankylosis-related resorption is evident (arrow). Reproduced from (3) with permission.

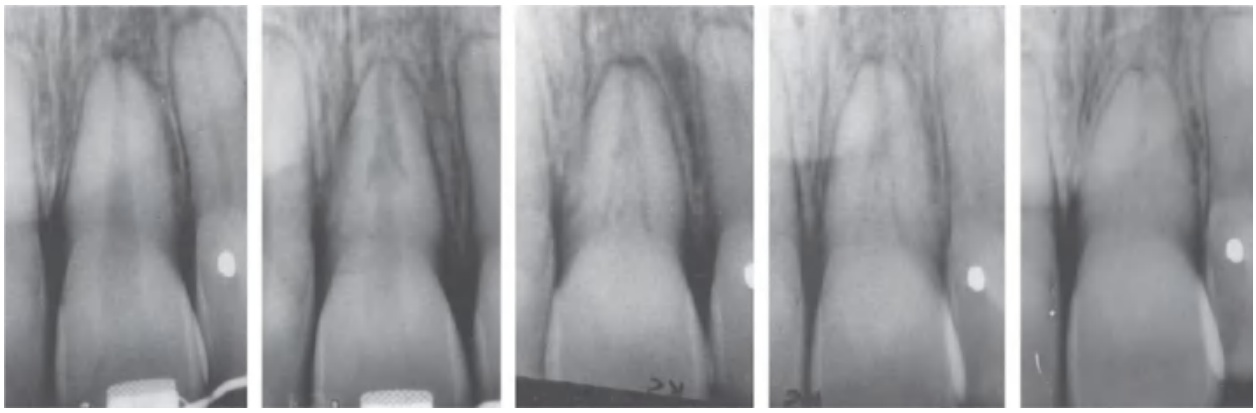



**Fig. 16.22** Internal ankylosis-related resorption (tunneling resorption variant) developed in an extruded and repositioned left central incisor. Tunneling resorption is evident after 6 months, but gradually disappears. Courtesy of Dr. B. Malmgren, Eastman Institute, Stockholm.

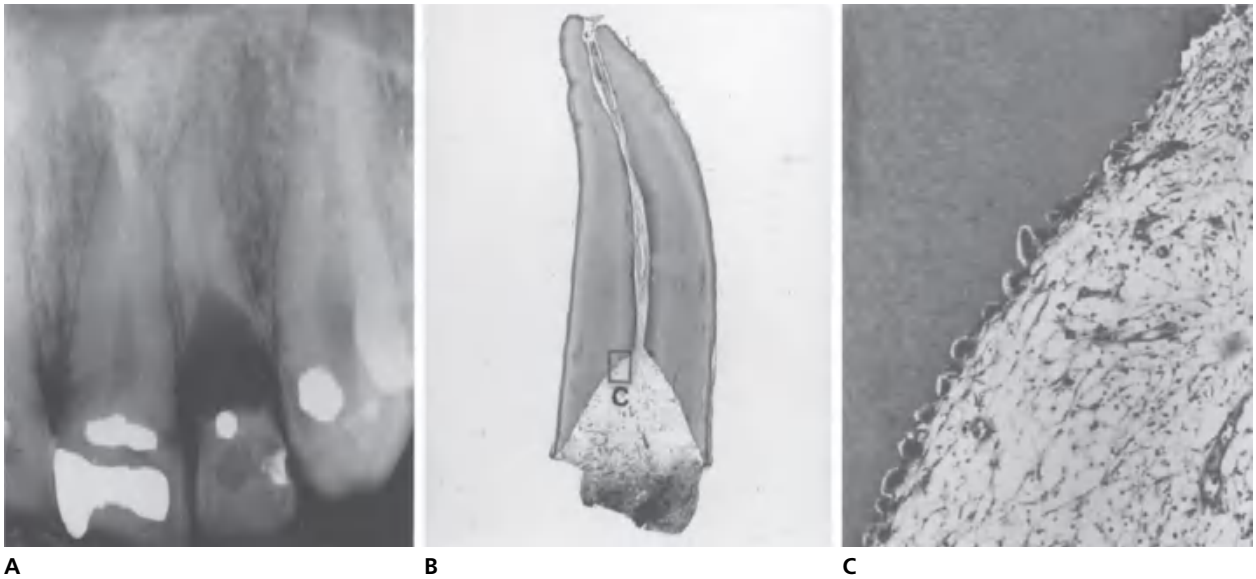



**Fig. 16.23** Internal inflammatory root resorption in a lateral incisor possibly caused by a luxation injury sustained 18 years previously. A. Radiograph showing marked internal infection-related resorption. B. Histologic section of removed incisor. ×6. C. Active resorption of dentin by multinucleated cells. ×195.

is apparently responsible for maintaining the resorptive process.

It should be emphasized that progression of root canal resorption depends upon the interaction between necrotic and vital pulp tissues at their interface. More recently, it has been demonstrated that progressive, invasive resorption is also due to bacterial infection (167). Consequently, root canal treatment should be instituted as soon as possible after root canal resorption has been diagnosed unless the resorption cavity is located in the vicinity of the apical foramen and suspected of being related to pulpal revascularization (see Chapter 25).

## Loss of marginal bone support

The post-traumatic course following intrusive or lateral luxation is often complicated by temporary or permanent changes in the marginal periodontium. A frequency of 5–24% was found among luxated permanent teeth (92, 100) (Table 16.9). If broken down according to specific luxation categories, 7% of laterally luxated and 31% of intruded permanent incisors demonstrated this complication (92) (Table 16.10). It has been found that the repositioning procedure following intrusive luxation plays an important role in periodontal healing. Thus, immediate surgical repositioning (i.e. complete repositioning at the time of injury) of the intruded mature incisors results in a slightly greater loss of marginal bone and a higher frequency of ankylosis compared to orthodontic extrusion. The best results have been found with respect to marginal bone healing in cases of spontaneous eruption compared to surgical or orthodontic repositioning (146–148).

**Table 16.9** Frequency of loss of marginal bone support after luxation of permanent teeth irrespective type of luxation injury.

| Study | No. of teeth | Loss of marginal bone support |
|---|---|---|
| Andreasen & Vestergaard Pedersen (92) 1985 | 637 | 34 (5%) |
| Oikarinen et al. (100) 1987 | 147 | 35 (24%) |

### Permanent or transient marginal breakdown

Loss of marginal support is a common healing complication following lateral luxation. It becomes clinically evident with the appearance of granulation tissue in the gingival crevice and sometimes secretion of pus from the pocket. Probing the pocket reveals a loss of attachment. Radiographically, rarefaction and loss of supporting bone is seen. This clinical situation might represent the first phase of the healing process in a traumatized periodontium, namely the resorption of traumatized bone. After 6–8 weeks, repair of the periodontium takes place with reattachment of new periodontal fibers (Fig. 16.24). This condition has been termed *transient marginal breakdown* (TMB) (146–148). If signs of marginal

**Table 16.10** Frequency of loss of marginal bone support related to type of luxation injury. Reproduced from (92) with permission.

| | No. of teeth | Loss of marginal bone support |
|---|---|---|
| Concussion | 178 | 0 (0%) |
| Subluxation | 223 | 3 (1%) |
| Extrusive luxation | 53 | 3 (6%) |
| Lateral luxation | 122 | 9 (7%) |
| Intrusive luxation | 61 | 19 (31%) |

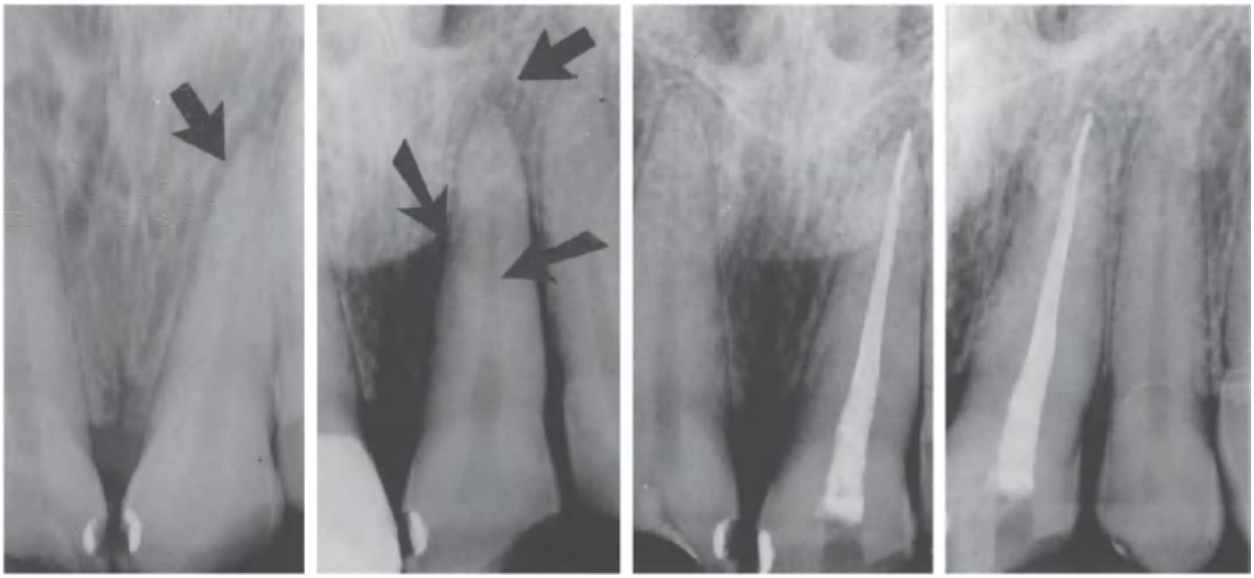



**Fig. 16.24** Transient destruction of marginal bone around a left central incisor after lateral luxation. At the time of injury there is displacement of the tooth in its socket (arrow). Six weeks later, extensive marginal and apical bone destruction is evident (arrows). Clinically, a pathologic deepening of the gingival crevice of 10 mm is found lingually. At follow-up one year later, marginal bone formation can be seen accompanied by normal pocket depth.

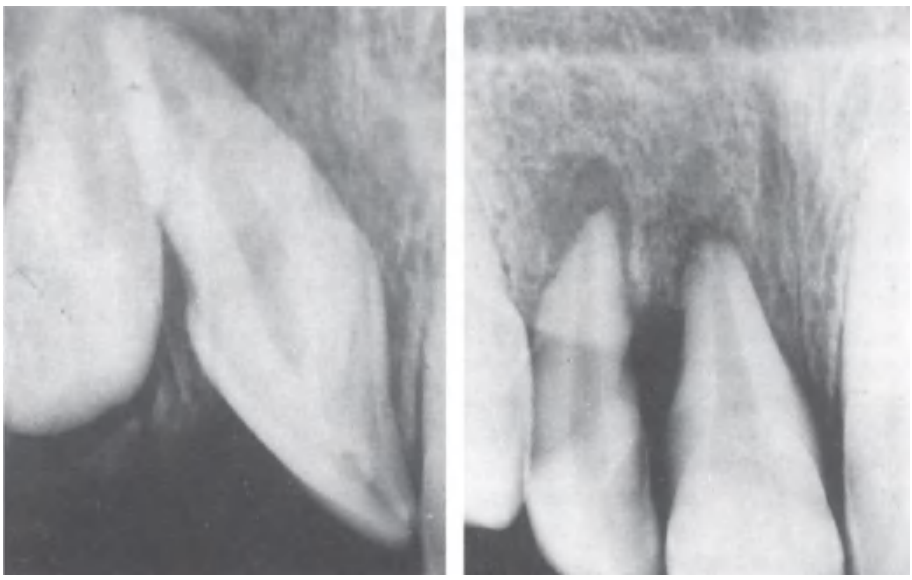



**Fig. 16.25** Loss of marginal supporting bone as a result of intrusive luxation. A. Radiograph at the time of injury reveals intrusive luxation of both right incisors. The teeth were repositioned and splinted. B. Three months later a marked loss of supporting marginal bone has occurred. Reproduced from (3) with permission.

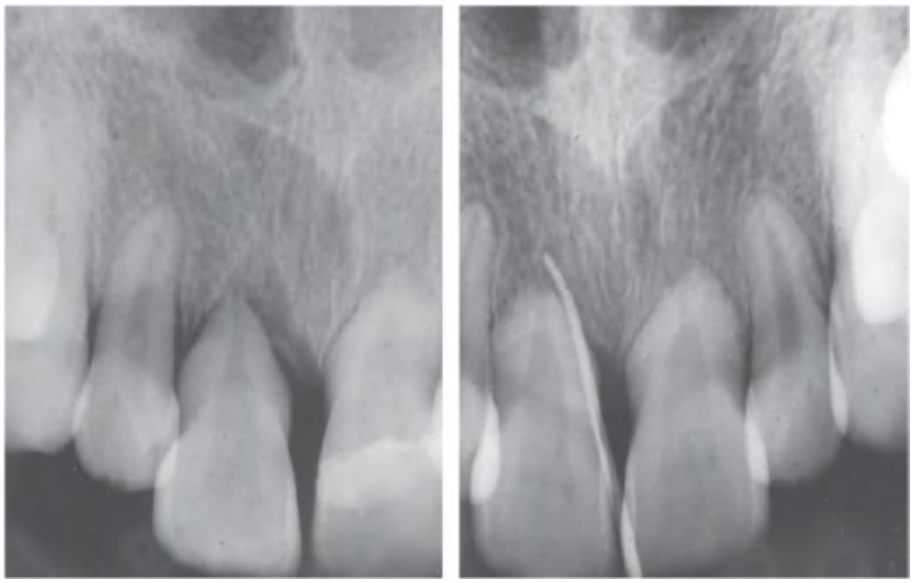



**Fig. 16.26** Loss of supporting bone as a result of delayed reduction of an extruded central incisor. A. Condition at time of admission. The tooth had been luxated 7 days previously. B. Two months later a periodontal pocket extends to the apex. A gutta-percha point is inserted in the periodontal pocket lingually.

bone loss are evident, the fixation period following lateral luxation should be extended until the situation has resolved (Fig. 16.25).

If there has been injury to the marginal bone, it is essential that optimal oral hygiene be maintained throughout the healing period. If there is secretion of pus, the pocket should be rinsed with saline. Moreover, oral rinsing with chlorhexidine should be instituted. Where sequestration of bone occurs during the healing period, permanent loss of marginal bone can be expected.

Delayed reduction of luxated teeth seems to increase the risk of damage to the supporting structures (3) (Fig. 16.26). Further, it has been found that loss of marginal bone support increases with increasing age of the patient, duration of the splinting period and the extent of displacement (74, 100, 147).

## Essentials

### Terminology (Fig. 16.1)

- Concussion
- Subluxation (loosening)
- Intrusive luxation (central dislocation)
- Extrusive luxation (peripheral dislocation, partial avulsion)
- Lateral luxation

### Frequency

- Permanent dentition: 15–40% of dental injuries
- Etiology
- Fall injuries

- Fight injuries
- History
- Symptoms
- Pain with occlusion

## Clinical examination (Table 16.1)

- Direction of dislocation
- Mobility testing
- Response to percussion
- Response to sensibility tests

## Radiographic examination

- Several projections necessary

## Pathology

- Periodontal ligament (PDL) changes varying from bleeding, PDL rupture to compression necrosis
- PDL changes, depending upon interference with the neurovascular supply (i.e. partial or total ischemia), leading to:
  - Pulpal healing
  - Pulpal revascularization
  - Partial pulp necrosis
  - Total pulp necrosis

## Root surface (external) resorption

- Surface resorption (repair-related resorption)
- Inflammatory resorption (infection-related resorption)
- Replacement resorption (ankylosis)

## Root canal (internal) resorption

- Internal replacement resorption
- Internal inflammatory resorption

## Prognosis

- Pulp necrosis: 15–52%
  - Risk groups: dislocated mature teeth
  - Treatment of pulp necrosis: see Chapter 25
- Pulp canal obliteration: 3–35%
  - Risk groups: extremely mobile or dislocated teeth with immature root formation
- Root surface resorption: 1–18%
  - Risk groups: intrusions
  - Treatment of external root resorption: see Chapter 25
- Root canal resorption: 2%
  - Treatment of internal root resorption: see Chapter 25
- Loss of supporting bone: 5%
  - Risk groups: intrusion and lateral luxation

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# 17

# Concussion and Subluxation

F. M. Andreasen, E. Lauridsen & J. O. Andreasen

## Definitions

### Concussion

Concussion is an injury to the tooth-supporting structures without abnormal loosening or displacement of the tooth, but with marked reaction to percussion (Fig. 17.1).

### Subluxation (loosening)

Subluxation is an injury to the tooth-supporting structures with abnormal loosening, but without displacement (Fig. 17.2).

## Frequency

The reported frequency of concussion and subluxation injuries is fairly high. In a recent study analyzing a population of Norwegian children aged 7–18 years, concussion injuries comprised 32% of all traumatic dental injuries, whereas subluxation injuries accounted for 17% of the injuries (1). Similar results have been reported from a large trauma center in Denmark where frequencies of 23% and 21% were found for concussion and subluxation, respectively (2).

## Pathology

### Concussion

In luxation injuries there is only minor damage in the periodontium. In the case of *concussion*, the impact results in bleeding and edema, and the patient will complain that the tooth is tender to touch. Clinical examination reveals a marked reaction to percussion in a horizontal or vertical direction (Fig. 17.3). However, the tooth shows no abnormal mobility. There is no bleeding from the gingival sulcus and the tooth usually responds positively to sensibility tests.

### Subluxation

In the case of *subluxation*, some damage (rupture) to the ligament fibers occurs. Subluxated teeth retain their normal position in the dental arch but the tooth is mobile in a horizontal direction, and sometimes also a bit in a vertical direction, and sensitive to percussion and occlusal forces. Hemorrhage from the gingival crevice is usually present, indicating damage to the periodontal tissue (Fig. 17.3). The tooth usually responds to sensibility tests.

## Radiographic findings

Usually no changes can be seen in the configuration of the periodontal ligament (PDL) space. However, in cases with marked mobility (grade 3, including vertical mobility), a slight widening of the PDL space may be seen.

## Treatment

Treatment for concussion and subluxation injuries may be confined to occlusal grinding of the opposing teeth and by repeated pulp tests during the follow-up period (Fig. 17.4). While splinting is not needed following

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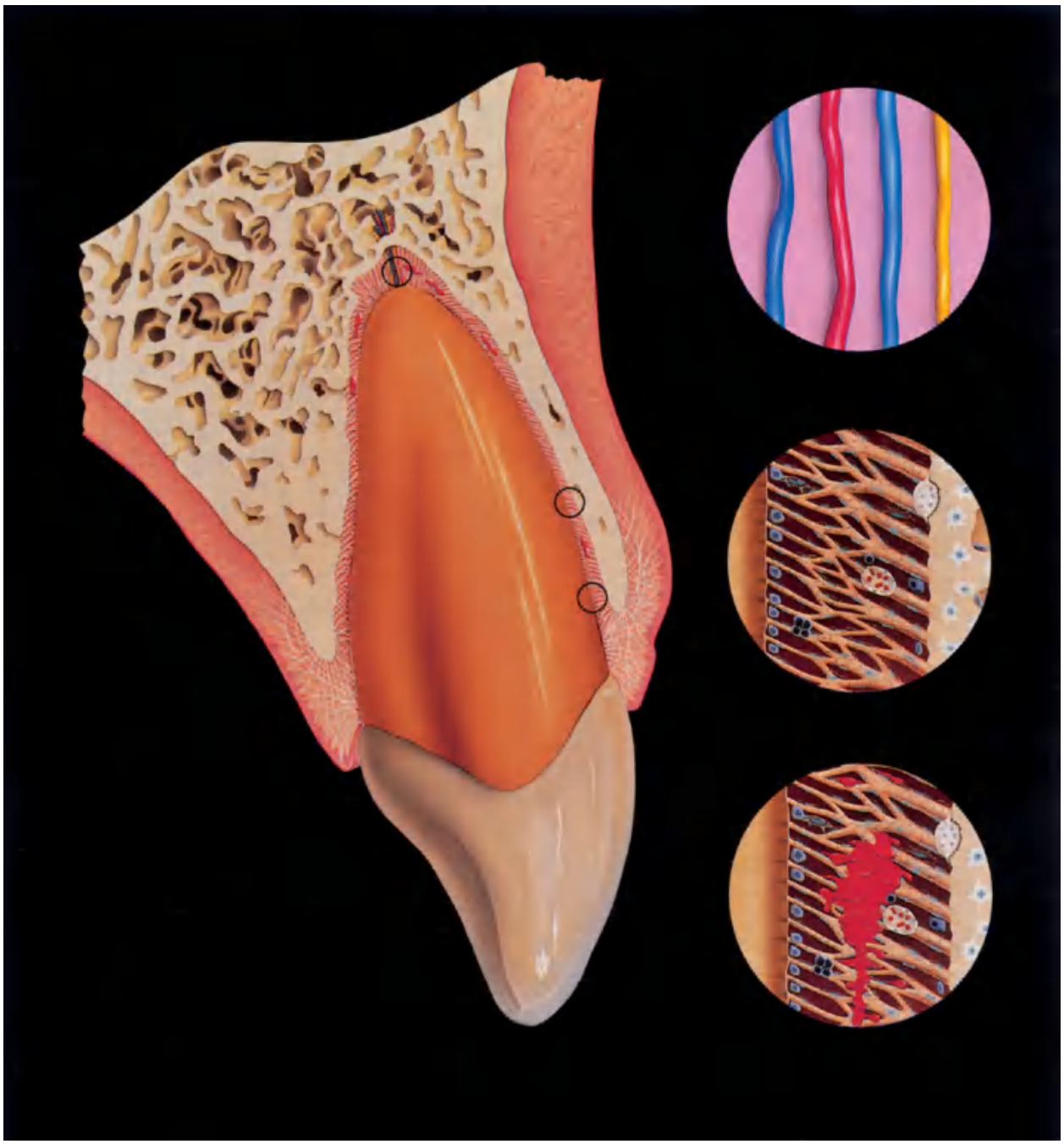

**Fig. 17.1** Mechanism and pathology of a concussion injury. A frontal impact leads to hemorrhage and edema in the periodontal ligament.

minor injuries, in the case of multiple tooth injuries the teeth may be included in the splint with no risk of damage to the PDL by the splinting procedure or splinting period (3).

## Follow-up

Follow-up after 4 weeks, 6–8 weeks and 1 year is recommended for teeth with a concussion injury (3). For teeth with a subluxation injury, follow-up after 2 weeks, 4 weeks, 6–8 weeks and 1 year is recommended (3). However, if clinical findings (including sensibility tests) and radiographic examination show no abnormal findings after 6–8 weeks, controls can be terminated.

## Complications

### Pulp necrosis

This event is rare (4–9) (Figs 17.5 and 17.6).

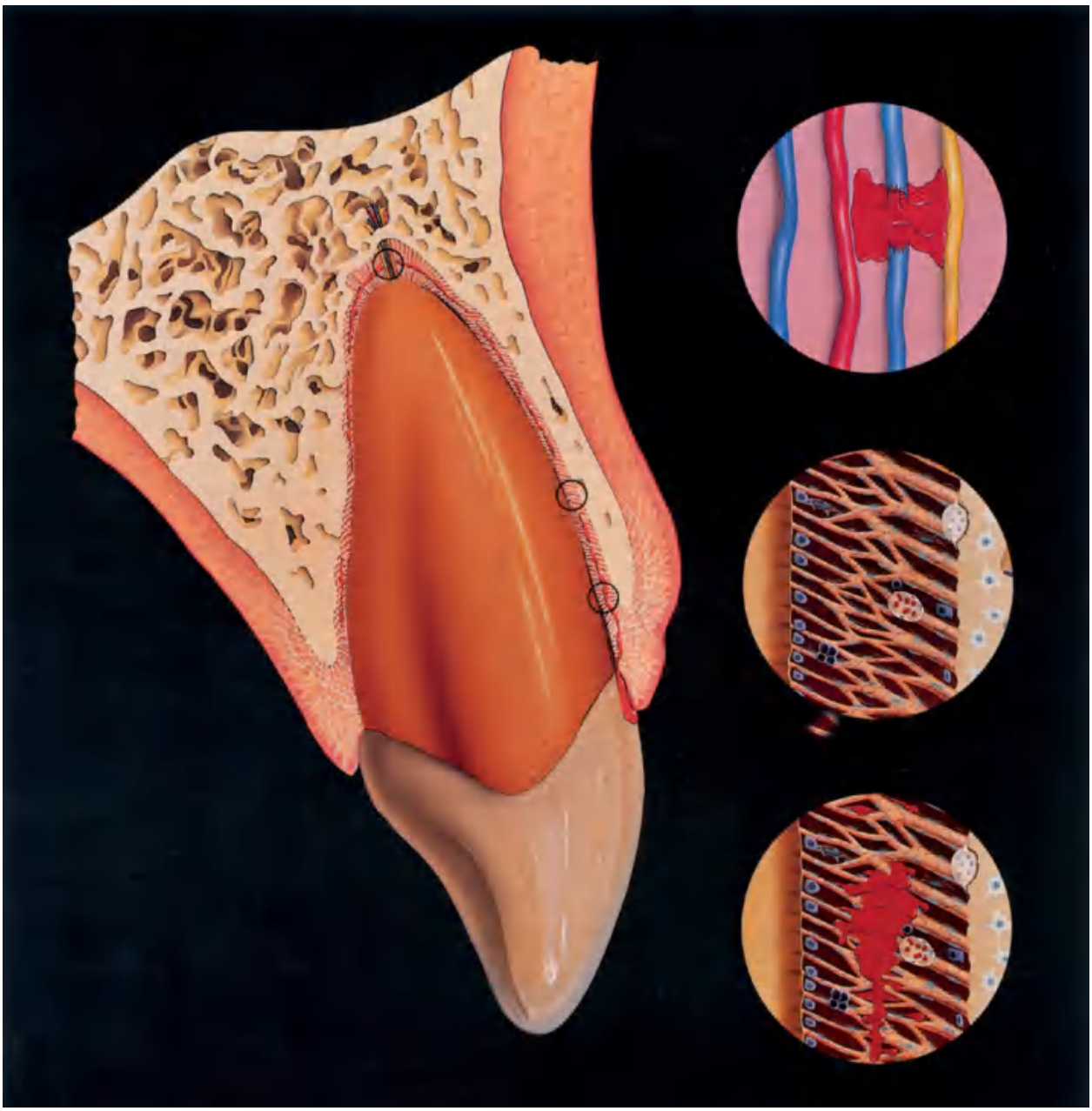

**Fig. 17.2** Mechanism and pathology of a subluxation injury. If the impact has greater force, periodontal ligament fibers may be torn, resulting in loosening of the injured tooth.

### Root resorption

Only a rare occurrence of surface resorption has been found in these trauma entities (4–9) (Figs 17.7 and 17.8).

## Predictors for healing and prognosis

The stage of root development and associated crown fracture have been found to be significantly related to the development of pulp necrosis following injury (4–10, 13).

A number of predictors for pulp and periodontal healing have been identified (4–10) (Figs 17.9 and 17.10).

### Associated crown fracture

In a detailed study of healing complications in teeth with combination injuries it was reported that the presence of a concomitant enamel-dentin fracture significantly increased the risk of pulp necrosis in mature teeth with concussion and subluxation, with a reported frequency of 11% and 55%, respectively. Even the presence of a concomitant trauma-induced enamel infraction increased the risk of pulp necrosis

**Fig. 17.3** Clinical and radiographic features of concussion and subluxation. The right and left maxillary central incisors have received a blow and are tender to percussion. The right central incisor is firm in its socket (concussion), while the left central incisor is loose, with hemorrhage from the gingival sulcus (subluxation).

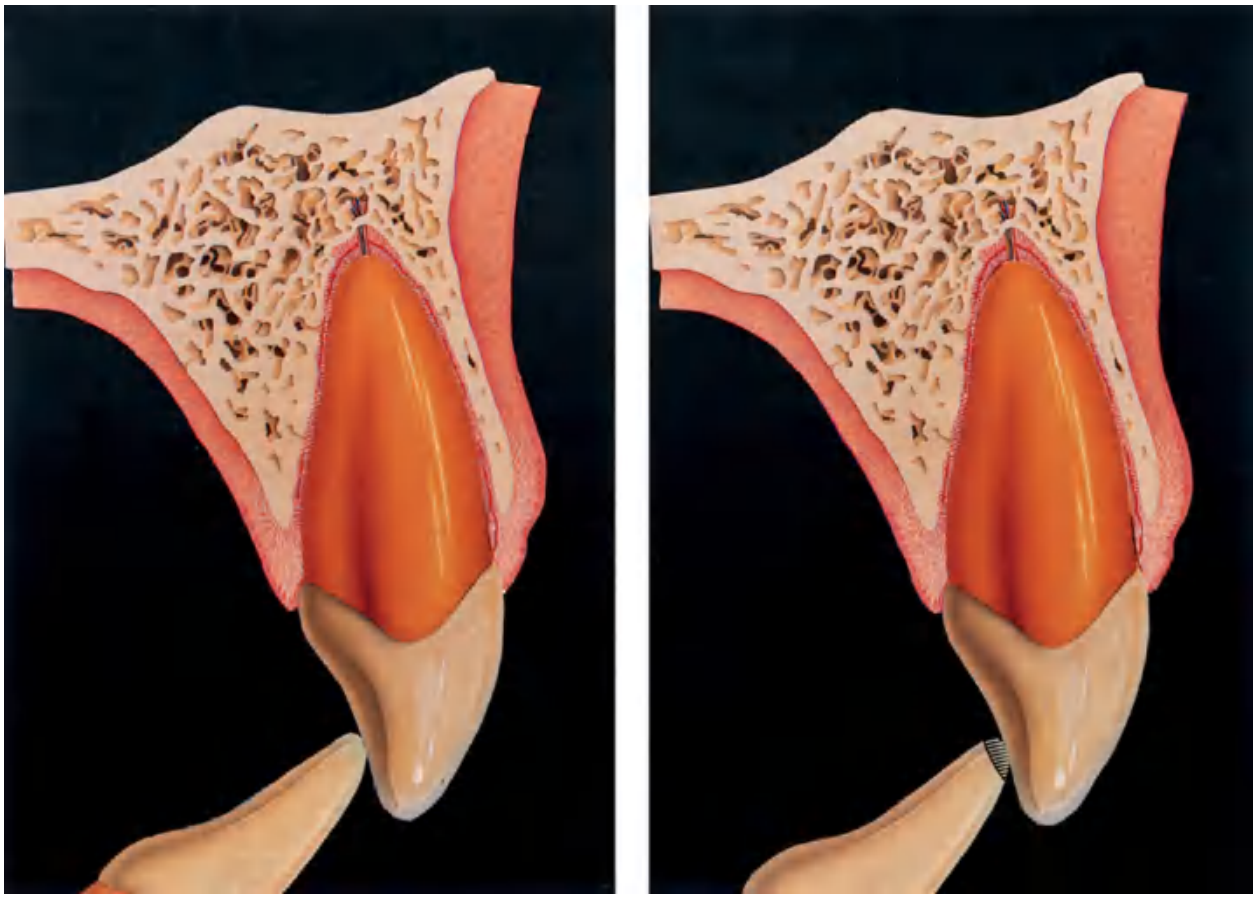

**Fig. 17.4** Treatment principles for concussion and subluxation. Relief of occlusal interference by selective grinding of opposing teeth may be necessary. In the case of severe loosening and/or multiple tooth injuries, teeth may be splinted. Otherwise, a soft diet is recommended for 14 days.

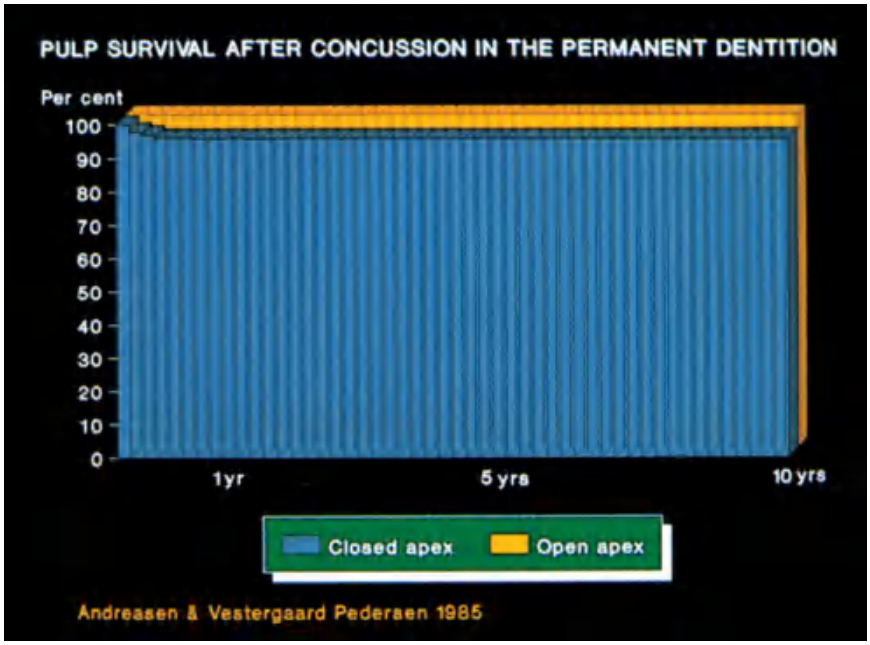


**Fig. 17.5** Pulp survival after concussion. Reproduced from (4) with permission.

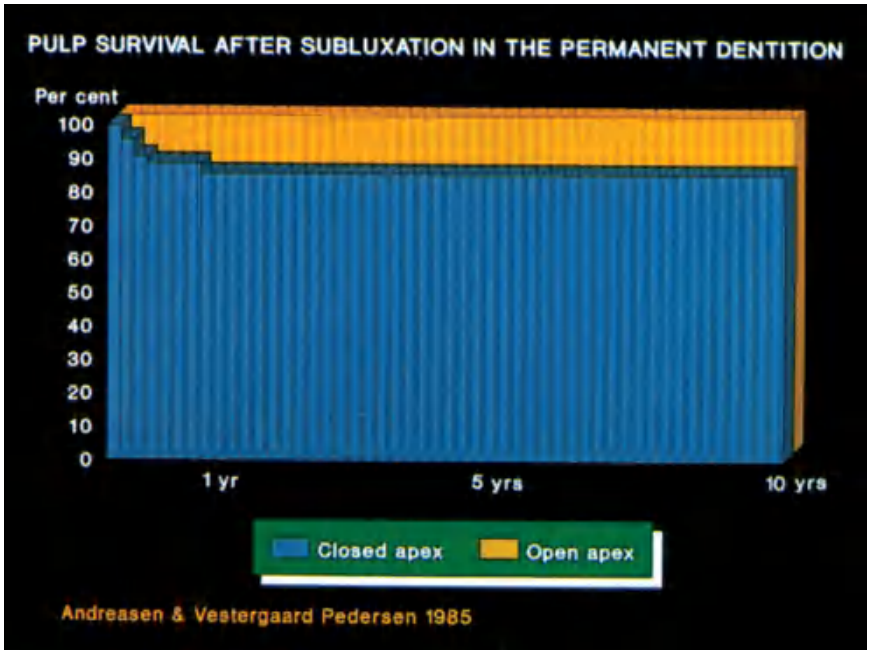


**Fig. 17.6** Pulp healing after subluxation. Reproduced from (4) with permission.

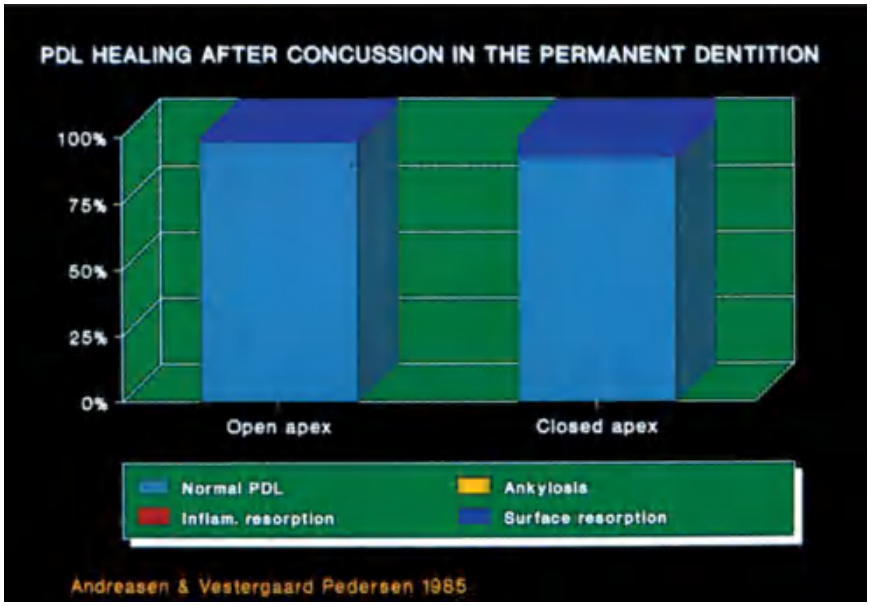


**Fig. 17.7** Periodontal healing after concussion. Reproduced from (4) with permission.

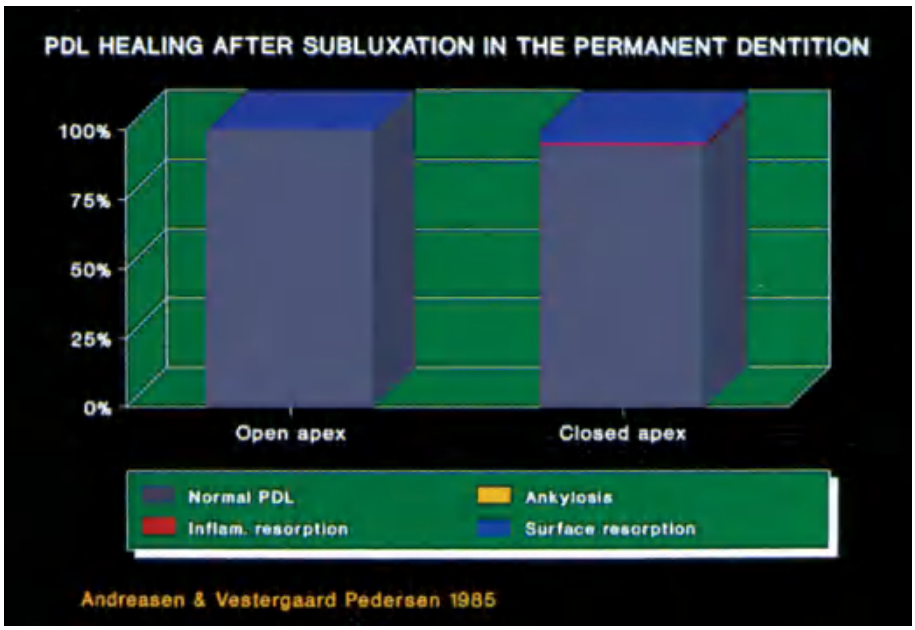


**Fig. 17.8** Periodontal healing after subluxation. Reproduced from (4) with permission.

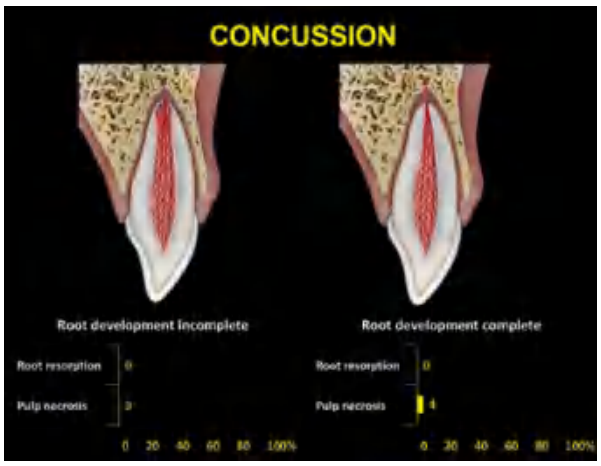


**Fig. 17.9** Predictors for healing after concussion.

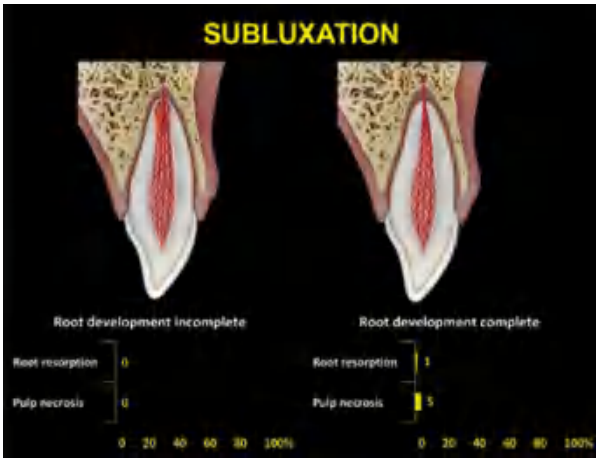


**Fig. 17.10** Predictors for healing after subluxation.

in teeth with subluxation injury (5, 10, 14). Teeth with concussion/subluxation and a concomitant crown fracture exposing the pulp showed no significant increase in the risk of pulp necrosis. This may be due to the low number of teeth examined in this group.

These results were supported by a later study from China on the risk of pulp necrosis after concussion and subluxation with uncomplicated crown fractures. However, these authors reported a somewhat higher frequency of pulp necrosis (70–80%) (13). This may be due to variation in diagnosis and treatment procedures.

## Pulp necrosis

The reported frequency of pulp necrosis is low, especially in teeth with immature root development (4–10, 14) (Table 17.1).

## Root resorption

There is a very limited risk of surface resorption and infection-related resorption (4, 11, 12) (Table 17.2).

## Essentials

- Relief of the occlusion on injured teeth and/or immobilization may be indicated, especially in the case of marked loosening. Otherwise, recommend a soft diet for 7 days
- Control the tooth radiographically and with sensibility testing
- The risk of pulp necrosis and progressive root resorption is low

**Table 17.1** Risk of pulp canal obliteration and pulp necrosis in teeth with concussion and subluxation (9, 10).

| | No. of teeth | Pulp canal obliteration *N* (%) | Pulp necrosis *N* (%) |
|---|---|---|---|
| Concussion, immature root development | 169 | 2 (1.6) | 0 (0) |
| Concussion, mature root development | 300 | 5 (4.4) | 4 (3.5) |
| Subluxation, immature root development | 230 | 20 (13) | 0 (0) |
| Subluxation, mature root development | 174 | 10 (9) | 14 (12.5) |

**Table 17.2** Risk of external root resorption in teeth with concussion and subluxation (11, 12).

| | No. of teeth | Repair-related resorption *N* (%) | Infection-related resorption *N* (%) | Ankylosis-related resorption *N* (%) |
|---|---|---|---|---|
| Concussion, immature root development | 169 | 0 (0) | 0 (0) | 0 (0) |
| Concussion, mature root development | 300 | 4 (3.2) | 0 (0) | 0 (0) |
| Subluxation, immature root development | 230 | 0 (0) | 4 (1.7) | 0 (0) |
| Subluxation, mature root development | 174 | 3 (3.6) | 1 (0.6) | 1 (0.6) |

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# 18

# Extrusive Luxation and Lateral Luxation

F. M. Andreasen, E. Lauridsen & J. O. Andreasen

## Definitions

### Extrusive luxation (axial dislocation, partial avulsion)

Extrusive luxation is partial displacement of the tooth out of its socket (Fig. 18.1).

### Lateral luxation (peripheral/eccentric dislocation)

Lateral luxation is displacement of the tooth in a direction other than axially. This is accompanied by comminution or fracture of the alveolar socket (Fig. 18.2).

## Frequency

The frequency of extrusive and lateral luxation has been found to be 7% and 11%, respectively, among traumatized permanent teeth examined at a major trauma center (1). In a more recent study analyzing a population of Norwegian children aged 7–18 years, these types of injury occurred less frequently (1.3% and 2.1%, respectively) (2).

## Healing and pathology

In extrusive luxation there is a complete rupture of the neurovascular supply to the pulp and severance of periodontal ligament fibers leading to extrusion. In cases of lateral luxation the periodontal injury is accompanied by a fracture of the labial bone plate as well as contusion of the palatocervical periodontal ligament.

In a study in rats the pulp response after extrusion was found to be separation of the odontoblast layer, especially in the coronal part of the pulp; furthermore interstitial bleeding was found. After 4 and 8 weeks irregular dentin formation took place (3).

## Clinical findings

### Extrusion

Extruded teeth appear elongated, usually with palatal deviation of the crown, as the tooth is suspended only by the palatal gingiva (Fig. 18.1). There is always bleeding from the periodontal ligament. The tooth shows increased mobility. The percussion sound is dull.

### Lateral luxation

The crowns of laterally luxated teeth are in most cases displaced palatally. The injury is usually associated with fracture of the vestibular part of the socket wall (Fig. 18.2). Displacement of teeth after lateral luxation is normally evident by visual inspection. Palpation of the vestibule at the level corresponding to the displaced apex will reveal the point of penetration through the labial bone plate. However, in cases of marked inclination of the maxillary teeth, it can be difficult to decide whether the trauma has caused a minor change in tooth position. In such cases, occlusion should be checked. Due to the frequently locked position of the tooth in the alveolus, clinical findings revealed by percussion and mobility tests are identical with those found in intruded teeth (see Chapter 19, Table 19.1).

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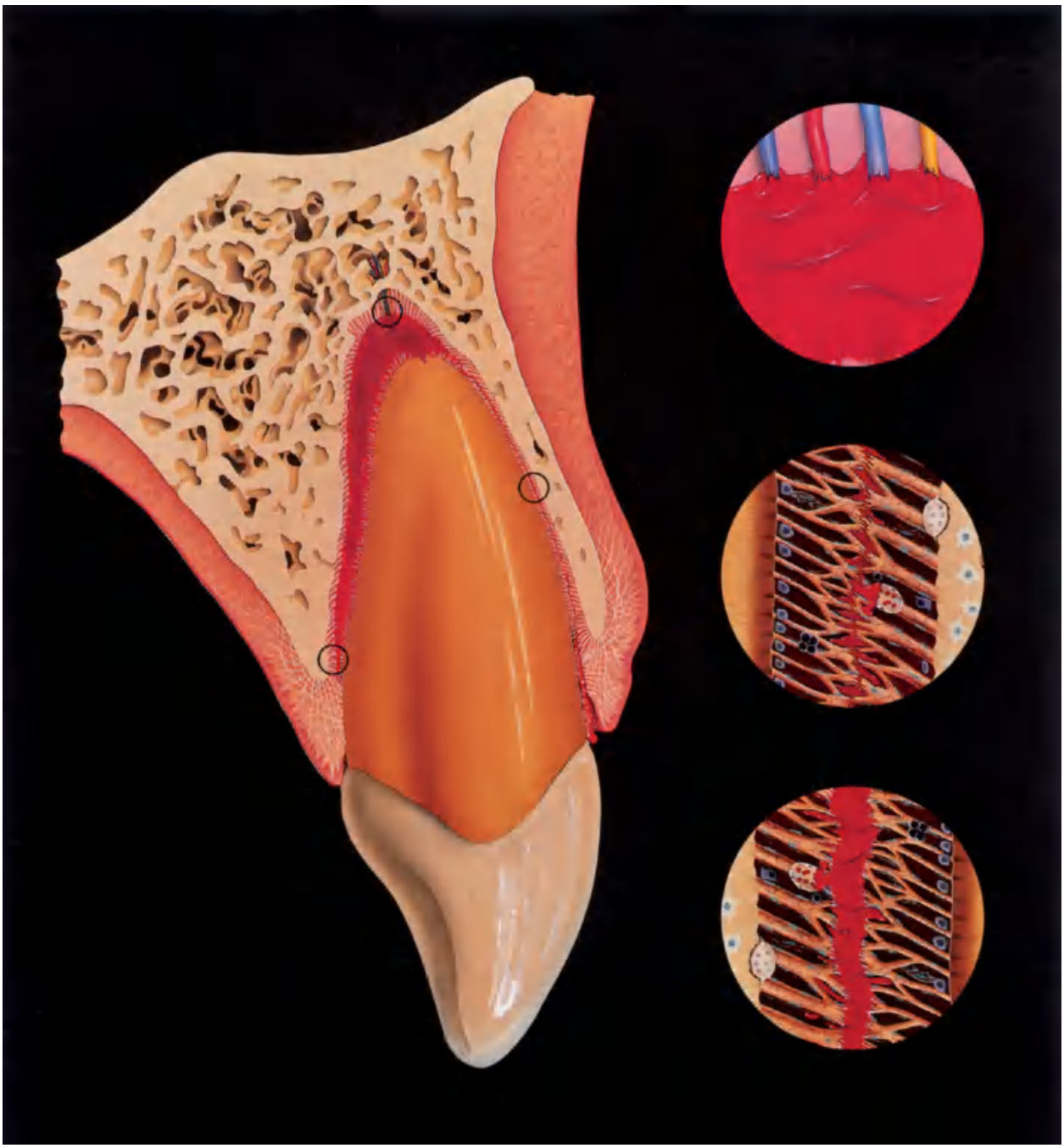


**Fig. 18.1** Pathogenesis of extrusive luxation. Oblique forces displace the tooth out of its socket. Only the palatal gingival fibers prevent the tooth from being avulsed. Both the periodontal ligament and the neurovascular supply to the pulp are ruptured.

## Radiographic findings

*Extruded teeth* show an expanded periodontal space, especially apically (Fig. 18.3). This will be evident in both occlusal and orthoradial exposures (Fig. 18.3).

Likewise, a *laterally luxated tooth* shows an increased periodontal space apically when the apex is displaced labially. However, this will usually be seen only in an occlusal or eccentric exposure. An orthoradial exposure will give little or no evidence of displacement. The radiographic picture, which imitates extrusive luxation, is explained by the relation between the dislocation and direction of the central beam (4) (Fig. 18.4).

## Treatment

The treatment of *extruded* permanent teeth seen soon after injury consists of careful repositioning, whereby the coagulum formed between the displaced root and socket wall will be slowly pressed out along the gingival crevice (Fig. 18.5). Administration of local anesthetic is generally

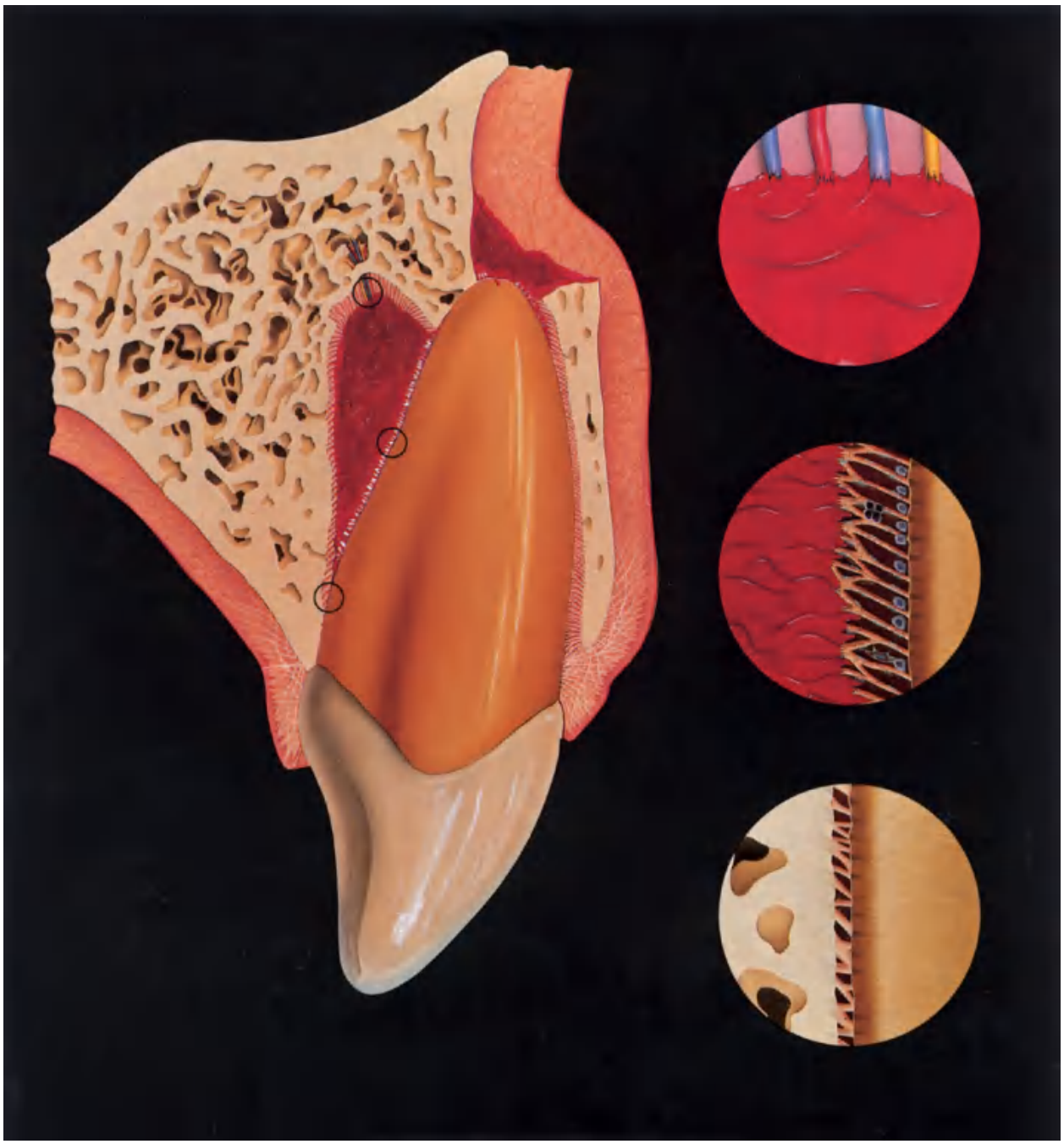


**Fig. 18.2** Pathogenesis of lateral luxation. Horizontal forces displace the crown palatally and the root apex facially. Apart from rupture of the periodontal ligament (PDL) and the pulpal neurovascular supply, compression of the PDL is seen on the cervico-palatal aspect of the root.

not necessary. As the repositioned tooth often has a tendency to migrate incisally, a flexible splint should be applied for 2 weeks (5) (Fig. 18.6).

In the case of *lateral luxation*, repositioning is usually a very forceful and, therefore, a traumatogenic procedure (Figs 18.7 and 18.8). Prior to this procedure, it is necessary to anesthetize the area. An infraorbital regional block anesthesia on the appropriate side of the maxilla is the most effective. As indicated, this type of luxation is characterized by forceful displacement of the root tip through the facial alveolar wall, which complicates the repositioning procedure. In order to dislodge the root tip from its bony lock, firm digital pressure in an incisal direction must first be applied immediately over the displaced root, which can be localized by palpating the corresponding bulge in the sulcular fold. Once the tooth is dislodged, it can be maneuvered apically into its correct position.

If manual repositioning is not possible, a forceps can be applied, whereby the tooth is first slightly extruded past the bony lock and then directed back into its correct position.

Once the tooth is repositioned, the labial and palatal bone plates should also be compressed to ensure complete repositioning and to facilitate periodontal healing. Lacerated gingiva should then be readapted to the neck of the tooth and sutured.

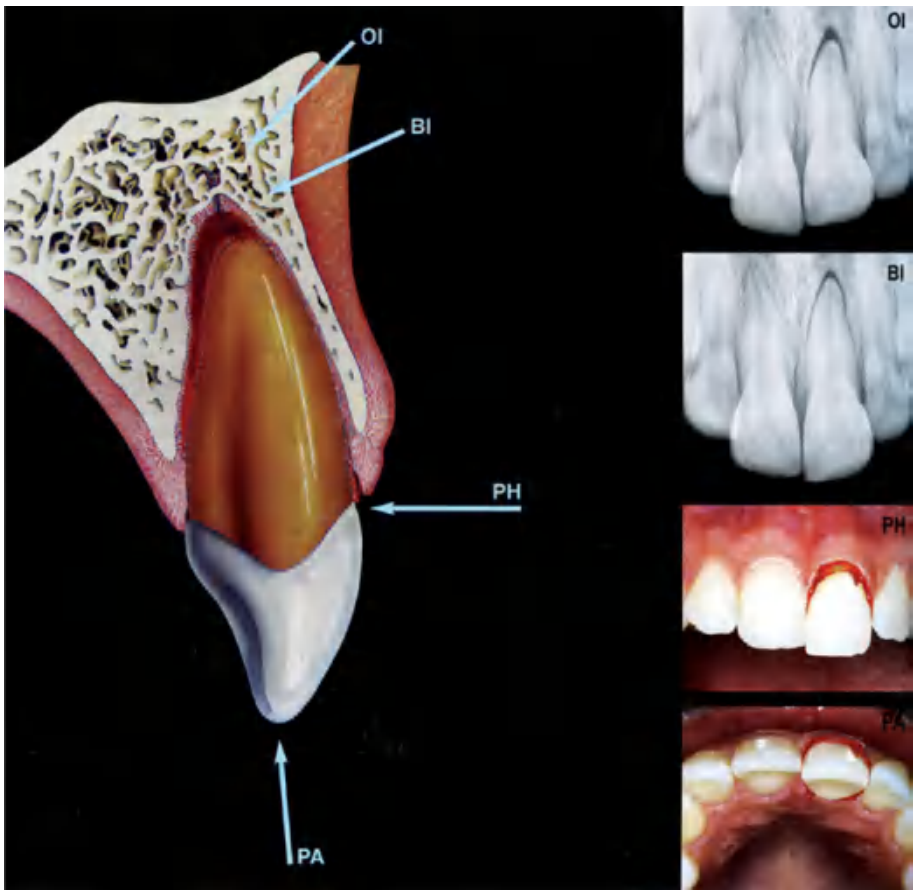



**Fig. 18.3** Clinical and radiographic features of extrusive luxation. The standard bisecting angle periapical radiographic technique is more useful than a steep occlusal exposure in revealing axial displacement. BI, bisecting angle identical radiograph; OI, occlusal identical radiograph; PA, photograph in axial plane; PH, photograph in horizontal plane. Reproduced from (4) with permission.

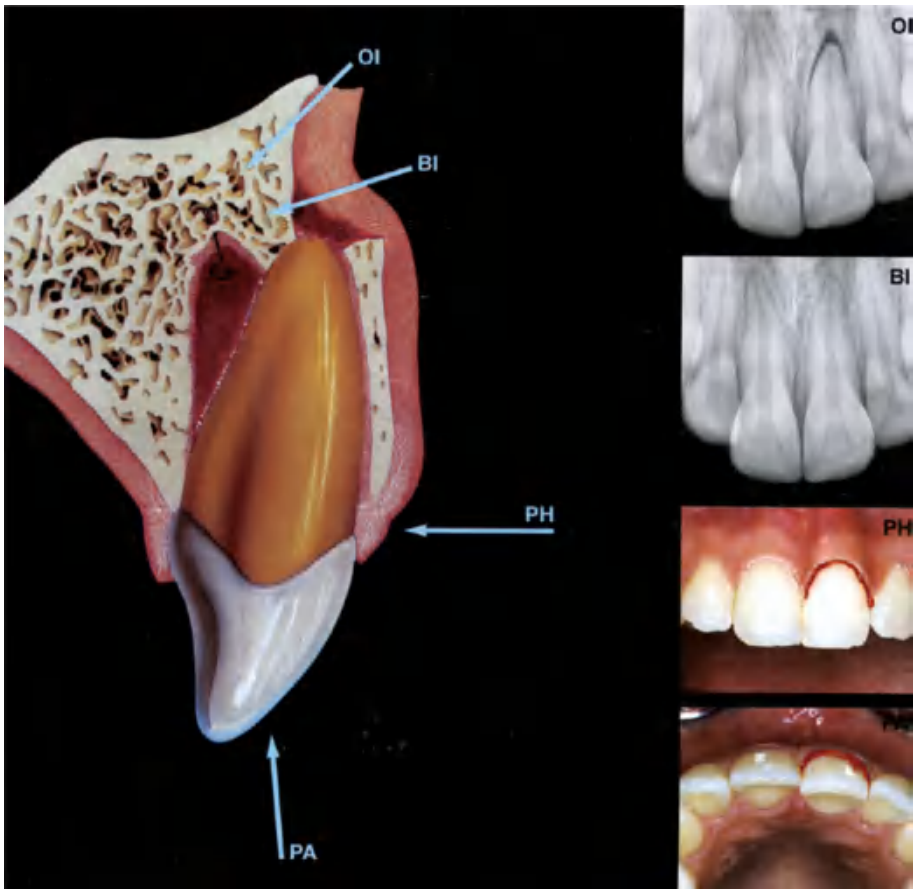



**Fig. 18.4** Clinical and radiographic features of lateral luxation. The steep occlusal radiographic exposure or an eccentric periapical bisecting angle exposure are more useful than an orthoradial bisecting technique in revealing lateral displacement. For abbreviations, see Fig. 18.3. Reproduced from (4) with permission.

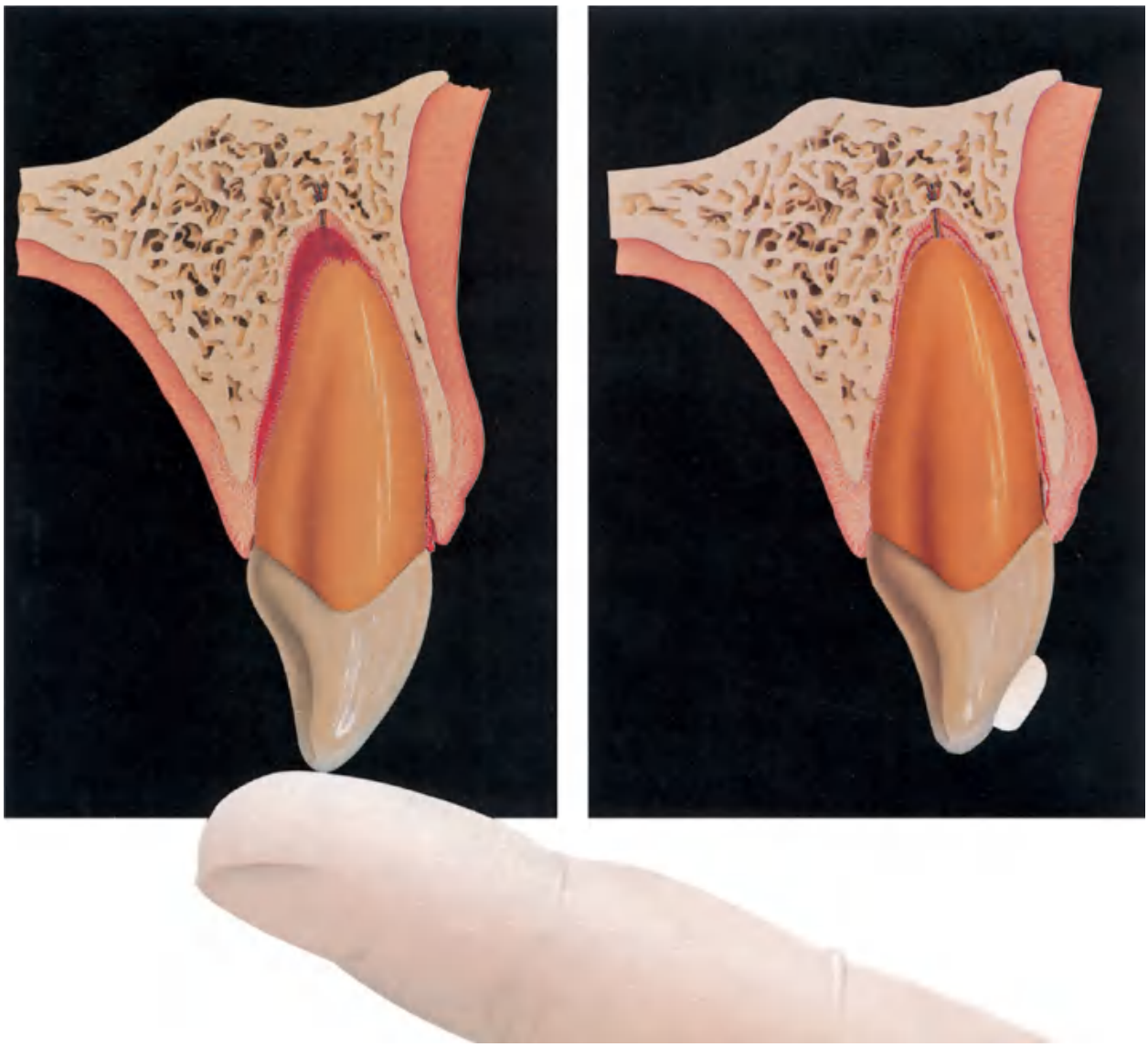


**Fig. 18.5** Treatment of extrusive luxation. The extruded tooth should be gently repositioned using axial finger pressure on the incisal edge.

The tooth should be splinted for 4 weeks in its normal position (5). A radiograph is then taken to verify repositioning and to register the level of the alveolar bone for later comparison. This is recommended in order to monitor eventual loss of marginal bone support in the follow-up period (see later).

If treatment of a laterally luxated or extruded permanent tooth is delayed (i.e. more than 3–4 days), it is usually found that the tooth is difficult to reposition. In young children, where the laterally luxated tooth does not interfere with occlusion, it might be appropriate to await spontaneous repositioning (Fig. 18.9).

## Prognosis

Very few studies have been performed on the prognosis of extrusive and lateral luxation. In Tables 15.1 and 15.2 and Figs 18.10, 18.11, 18.12 and 18.13 the frequencies of complications such as pulp necrosis, pulp canal obliteration, root resorption and marginal breakdown are given. In the studies reported by the Copenhagen group (6–9) and the Toronto group (10,11) it should be noted that the latter group was confined to a pediatric population (children and adolescents).

The prognosis has been shown for all luxation injuries to be significantly related to associated crown fractures (12, 18).

## Pulp necrosis

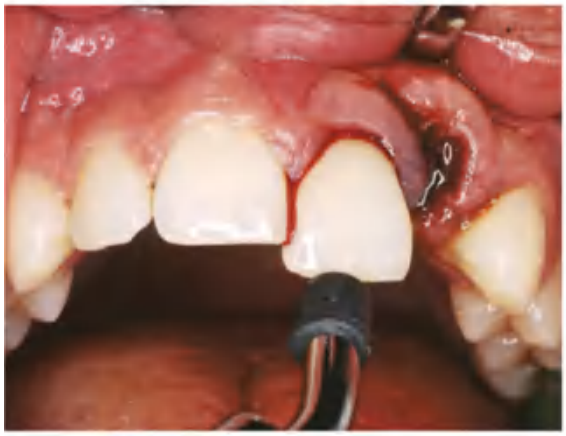
### Extrusion

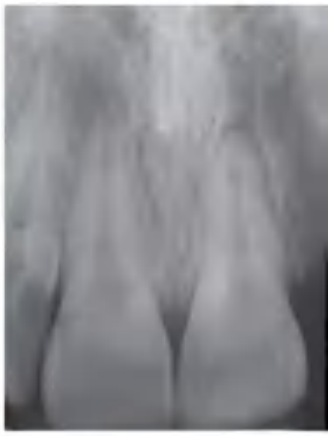
This complication was very dependent upon root development in the Copenhagen study, where immature root development had a superior healing potential compared to that of teeth with more mature root development (4–9) (Table 18.1, Fig. 18.10).

**Fig. 18.6 Diagnosis and treatment of extrusive luxation**
This 17-year-old has extruded the left central incisor and avulsed the lateral incisor, which could not be retrieved.

**Mobility and percussion test**
The tooth is very mobile and can be moved in horizontal and axial directions. The percussion test reveals slight tenderness and there is a dull percussion sound.

**Sensibility testing and radiographic examination**
The tooth does not respond to sensibility testing. The radiograph reveals coronal displacement of the tooth.





**Repositioning**
The tooth is gently pushed back into it socket. Thereafter the labial surfaces of both central incisors are etched in preparation for the splinting material.

**Applying splinting material**
After rinsing the labial surfaces with a water spray and drying with compressed air, the splinting material (Protemp®, Espe Corp.) is applied.

**Smoothing the splint**
The surface of the splint is smoothed with abrasive disks and contact with gingiva removed with a straight scalpel blade.

**The finished splint**
Note that the splint allows optimal oral hygiene in the gingival region, which is the most likely port of entry for bacteria that may complicate periodontal and pulpal healing.

**Suturing the gingival wound**
The gingival wound is closed with interrupted silk sutures. The final radiograph confirms optimal repositioning of the tooth.

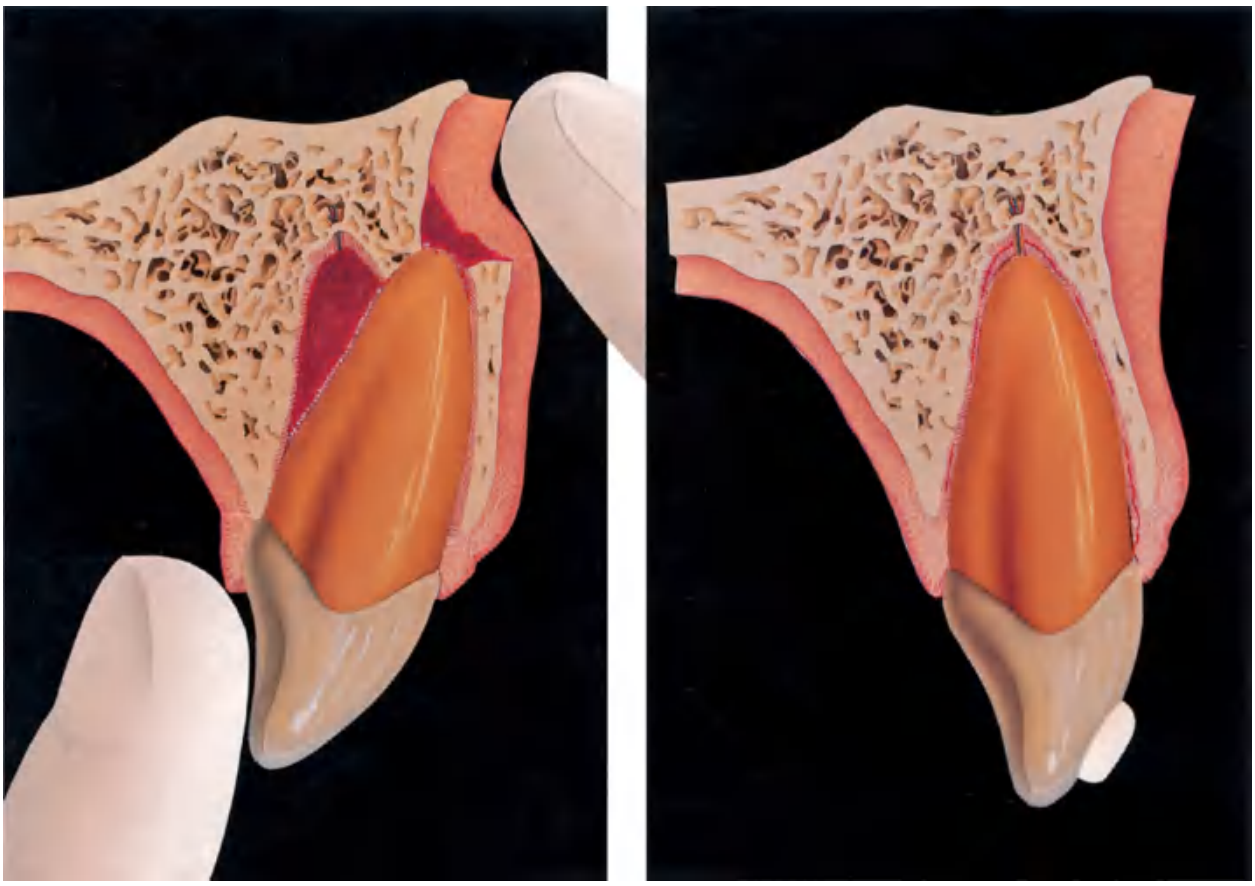


**Fig. 18.7** Treatment principles for lateral luxation: repositioning and splinting.

### Lateral luxation

The same conclusions were made as for extrusion (4–9) (Table 18.2, Fig. 18.11).

## Pulp canal obliteration

### Extrusion

In cases of pulpal revascularization, pulp canal obliteration was almost always a standard sequel to injury.

### Lateral luxation

The same conclusions were made as for extrusion.

## Root resorption (external)

### Extrusion

Surface resorption was quite common, and infection-related resorption rare. Ankylosis was not seen (6, 8) (Fig. 18.12).

### Lateral luxation

The resorption pattern was quite similar to that for extrusions (Fig. 18.13). Infection-related resorption and ankylosis-related resorption were rare. Ankylosis was in these instances located in the cervical region, i.e. where the compression zones are located (6, 8) (Fig. 18.14).

Moreover, the frequent shortening of the root tips (i.e. surface resorption) appears to be related to the apical compression zone.

## Root resorption (internal)

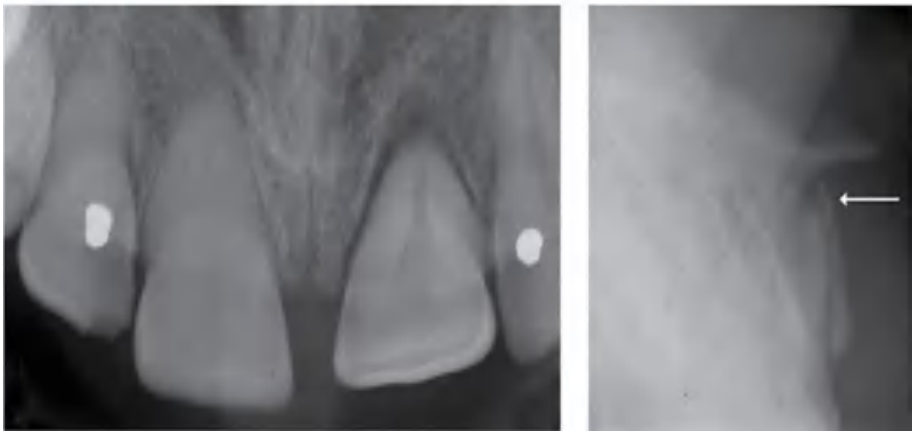
In approximately 10% of extrusions and lateral luxations studied by Andreasen (7, 13–15) *internal surface resorption* was seen at the internal aspect of apices of teeth with completed root development within the 1st year after injury. This type of resorption (transient apical breakdown (TAB)) was associated with transient discoloration of the clinical crown and temporary loss of pulpal sensibility. It was interpreted as a link in pulpal healing, whereby the process increased the diameter of the narrow apical foramen and permitted ingrowth of new blood vessels and nerves into the

**Fig. 18.8 Diagnosis and treatment of lateral luxation**
This 23-year-old man suffered a lateral luxation of the left central incisor.

**Percussion test**
Left, percussion of the labial surface of the injured tooth reveals a high metallic sound. Right, axial percussion discloses no tenderness.

**Mobility and sensibility testing**
Mobility testing, using either digital pressure or alternating pressure of two instrument handles facially and orally, discloses no mobility of the injured tooth. There is no response to pulpal sensibility testing.

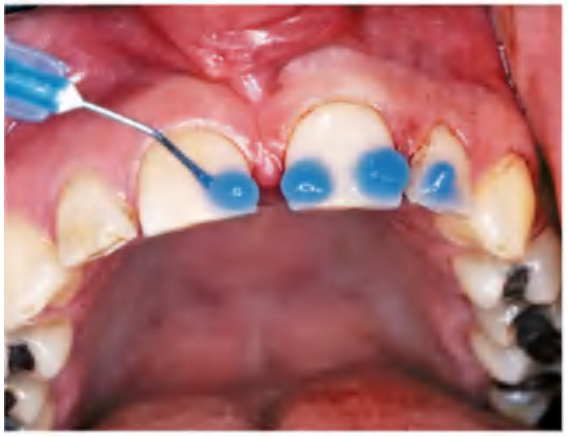
**Radiographic examination**
A steep occlusal radiographic exposure revealed, as expected, more displacement than the bisecting angle technique. A lateral radiograph reveals the associated fracture of the labial bone plate (arrow).



**Anesthesia**
An infraorbital reginal block is placed and supplemented with anesthesia of the nasopalatinal nerve.

**Repositioning**
The tooth is repositioned initially by forcing the displaced apex past the labial bone lock and thereby disengaging the root. Thereafter, axial pressure apically will bring the tooth back to its original position. It should be remembered that the palatal aspect of the marginal bone has also been displaced at the time of impact. This, too, must be repositioned with digital pressure to ensure optimal periodontal healing.

**Verifying repositioning and splinting with the acid-etch technique**
Occlusion is checked and a radiograph taken to verify adequate repositioning. The incisal one-third of the labial aspect of the injured and adjacent teeth is acid etched (30 seconds) with phosphoric acid gel.



**Preparing the splinting material**
The etchant is removed with a 20-second water spray. The labial enamel is dried with compressed air, revealing the matte, etched surface.

**Applying the splinting material**
A temporary crown and bridge material (e.g. Protemp®) is then applied. Surplus material can be removed after polymerization using a straight scalpel blade or abrasive disks.

**Four weeks after injury**
At this examination, a radiograph is taken to evaluate periodontal and pulpal healing. There has been neither periapical radiolucency nor breakdown of supporting marginal bone, as compared to the radiographs taken after repositioning.

**Splint removal**
The splint is removed using fissure burs, by reducing the splinting material interproximally, and thereafter thinning the splint uniformly across its total span. Once thinned, the splint can be removed using a sharp explorer.

**Six months after injury**
After 6 months, there is a slight sensibility reaction and normal radiographic conditions.

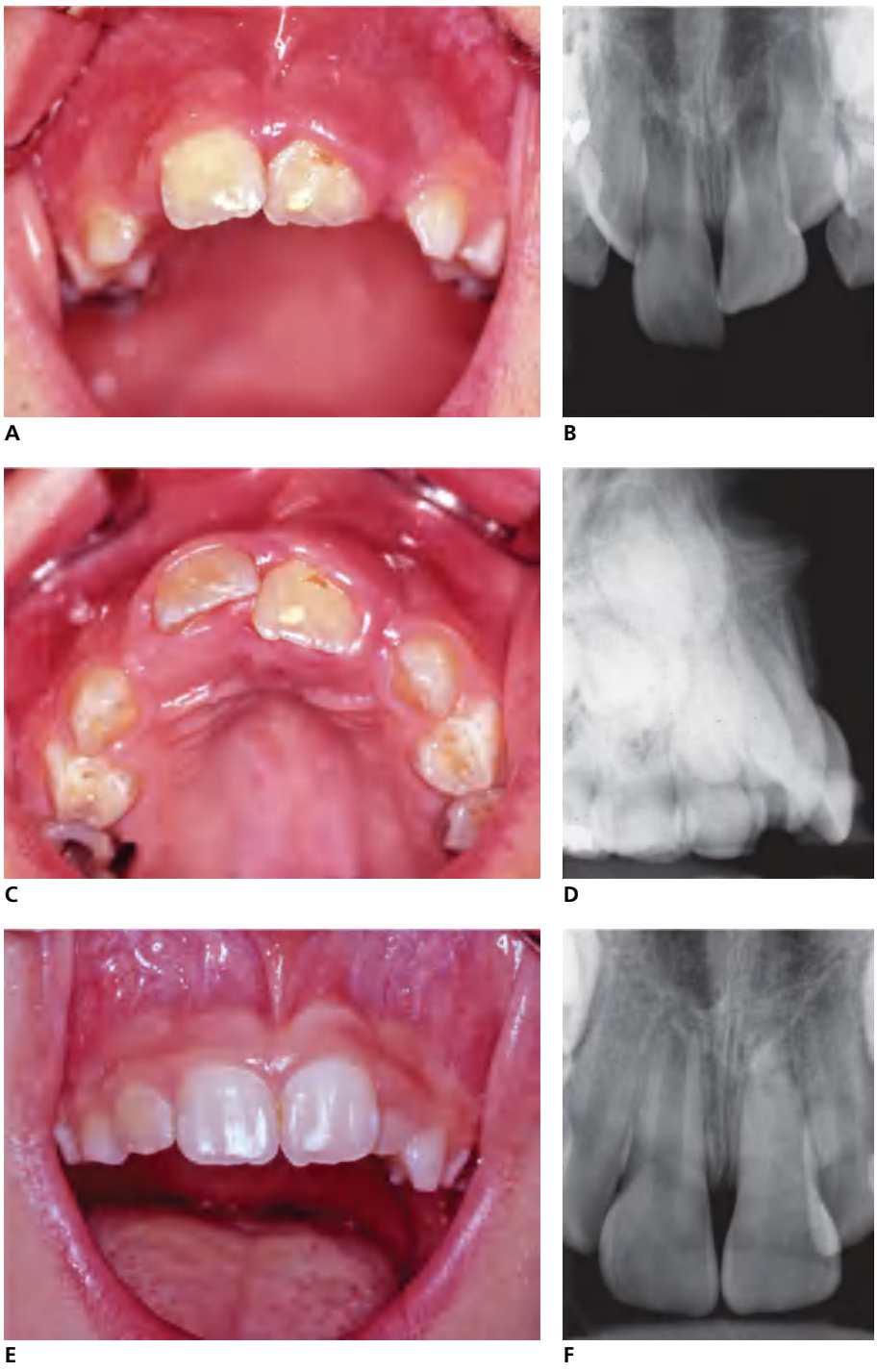



**Fig. 18.9** Non-repositioning of a laterally luxated left central incisor which did not interfere with occlusion, with spontaneous repositioning. A–D. Time of injury. Note that the labially displaced apex. E and F. One year later, with the tooth in its normal position. There is pulp canal obliteration.

traumatized pulp. The situation reverted to normal, occasionally accompanied by pulp canal obliteration, when coronal color and pulpal sensibility also returned.

Similar resorption processes have been described following root fracture (see Chapter 12) (16).

## When to do endodontics?

The end points in pulpal response to acute dental trauma can generally be classified as pulp necrosis, pulp canal obliteration and pulp survival (i.e. pulp survival with no radiographic change). However, clinical changes associated with TAB imply that there can be intermediate phases in the pulpal healing sequence that could result in diagnostic confusion. General guidelines are presented in Chapter 16. The following might also be considered:

(1) *Diagnosis of arrested root development.* If the root canal walls of the affected tooth have not increased in dimension (thickness), as compared to non-affected

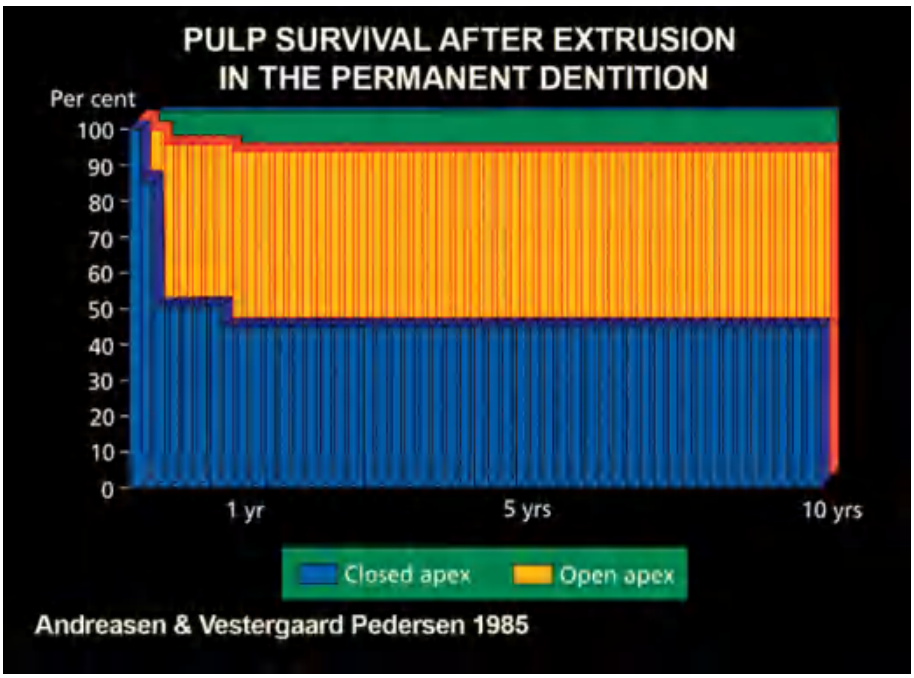



**Fig. 18.10** Pulpal healing after extrusive luxation (2,17). Reproduced from (6) with permission.

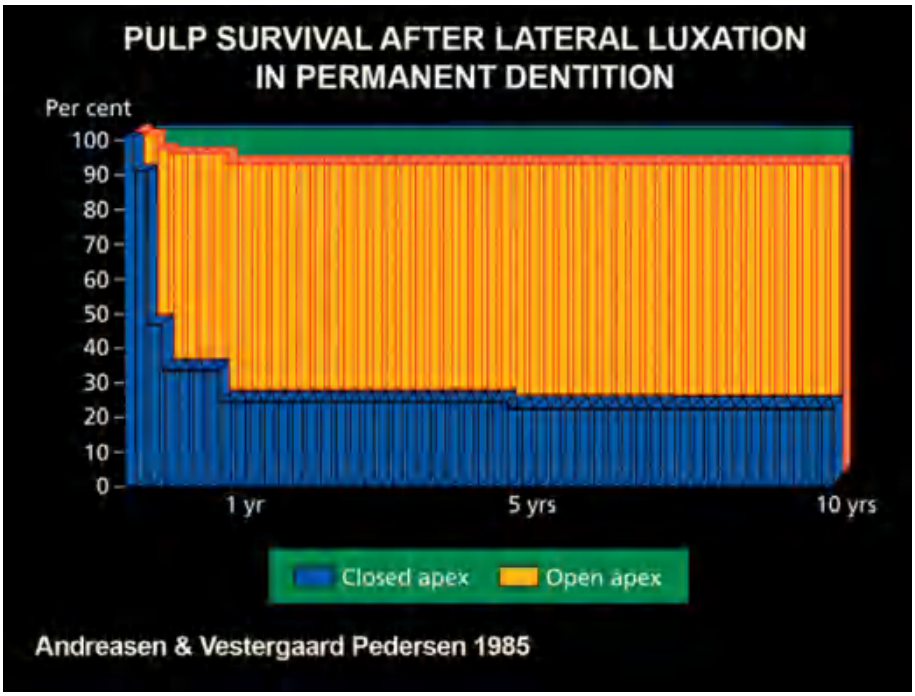



**Fig. 18.11** Pulpal healing after lateral luxation (2,17). Reproduced from (6) with permission.

teeth, since the time of injury, then pulpal function has ceased.

(2) *Progressive loosening.* This is a sign of infection/inflammation originating from a necrotic pulp.

(3) *Missing hard tissue barrier* following a crown fracture with pulp exposure.

(4) *Absent periodontal healing 6–12 months after injury.* In the absence of other signs of non-healing, *defects in the lamina dura* surrounding the root indicate infection.

(5) *Persistent tenderness to percussion.* Although tenderness to percussion at the time of injury implies damage to the periodontal ligament and/or other supporting

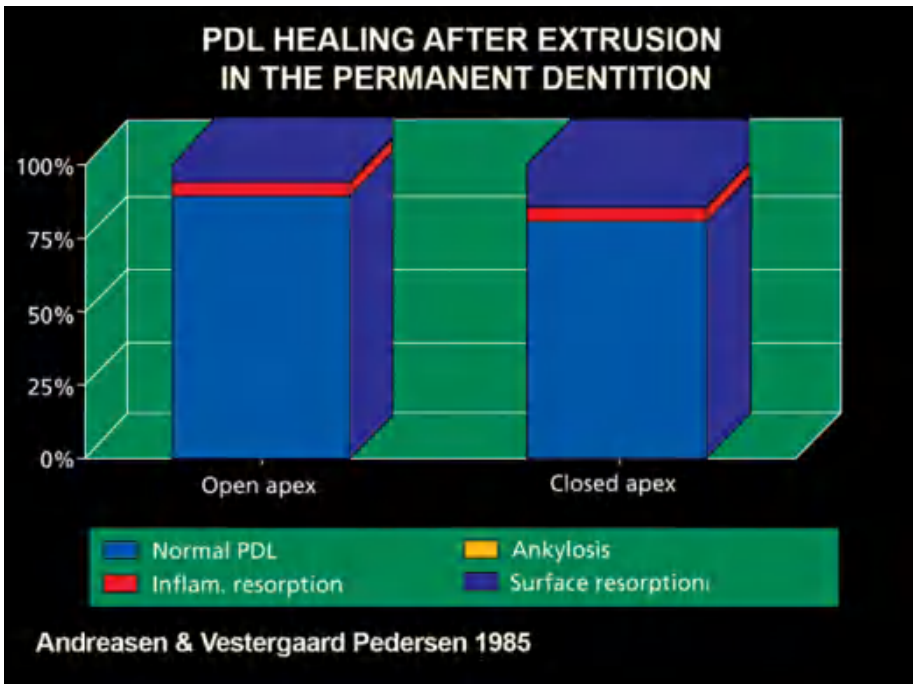



**Fig. 18.12** Periodontal ligament (PDL) healing after extrusive luxation (2,17). Reproduced from (6) with permission.

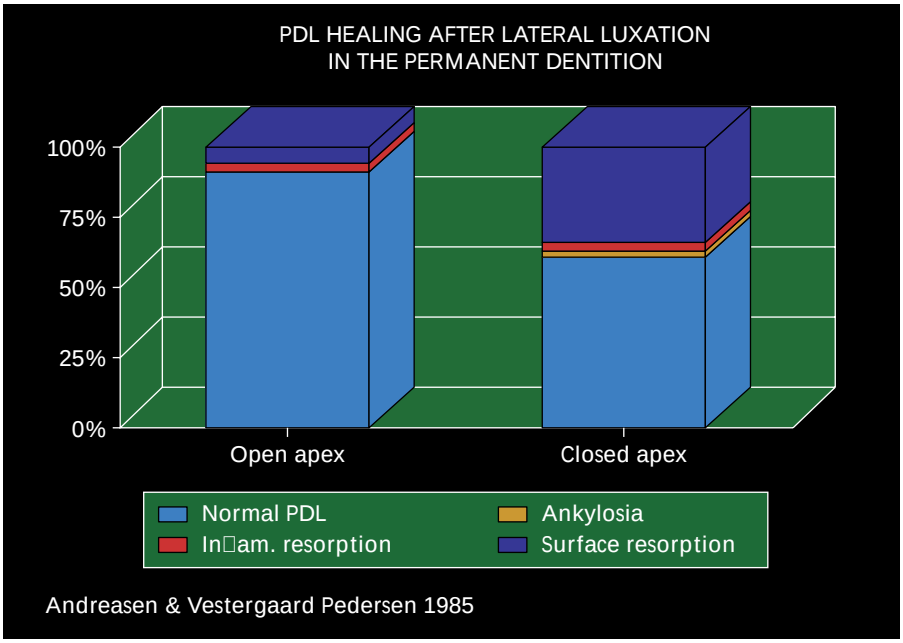



**Fig. 18.13** Periodontal ligament (PDL) healing after lateral luxation (2,17). Reproduced from (6) with permission.

**Table 18.1** Pulp and periodontal healing complications following extrusive luxation.

| Study | Stage of root development | No. of teeth | Pulp necrosis | Pulp canal obliteration | Pulp survival | Root resorption | Marginal breakdown |
|---|---|---|---|---|---|---|---|
| Andreasen & Vestergaard Pedersen (6) 1985 | Open apex | 34 | 3 (9%) | 20 (61%) | 10 (30%) | 4 (12%) | 3 (6%)† |
| | Closed apex | 20 | 11 (55%) | 4 (20%) | 15 (25%) | 4 (20%) | |
| Lee et al. (11)* 2003 | Open + closed apex | 54 | 23 (42%) | 19 (35%) | 12 (23%) | 3 (6%) | ? |

* Only children and adolescents.
† Open and closed apices.

**Table 18.2** Pulp and periodontal healing complications following lateral luxation.

| Study | Stage of root development | No. of teeth | Pulp necrosis | Pulp canal obliteration | Pulp survival | Root resorption | Marginal breakdown |
|---|---|---|---|---|---|---|---|
| Andreasen & Vestergaard Pedersen (6) 1985 | Open apex | 34 | 3 (9%) | 24 (71%) | 7 (20%) | 3 (9%) | 9 (7%)† |
| | Closed apex | 88 | 68 (77%) | 10 (11%) | 10 (11%) | 34 (39%) | |
| Nikoui et al. (10)* 2003 | Open + closed apex | 58 | 23 (40%) | 23 (40%) | 12 (20%) | ? | ? |

* Only children and adolescents.
† Open and closed apices.

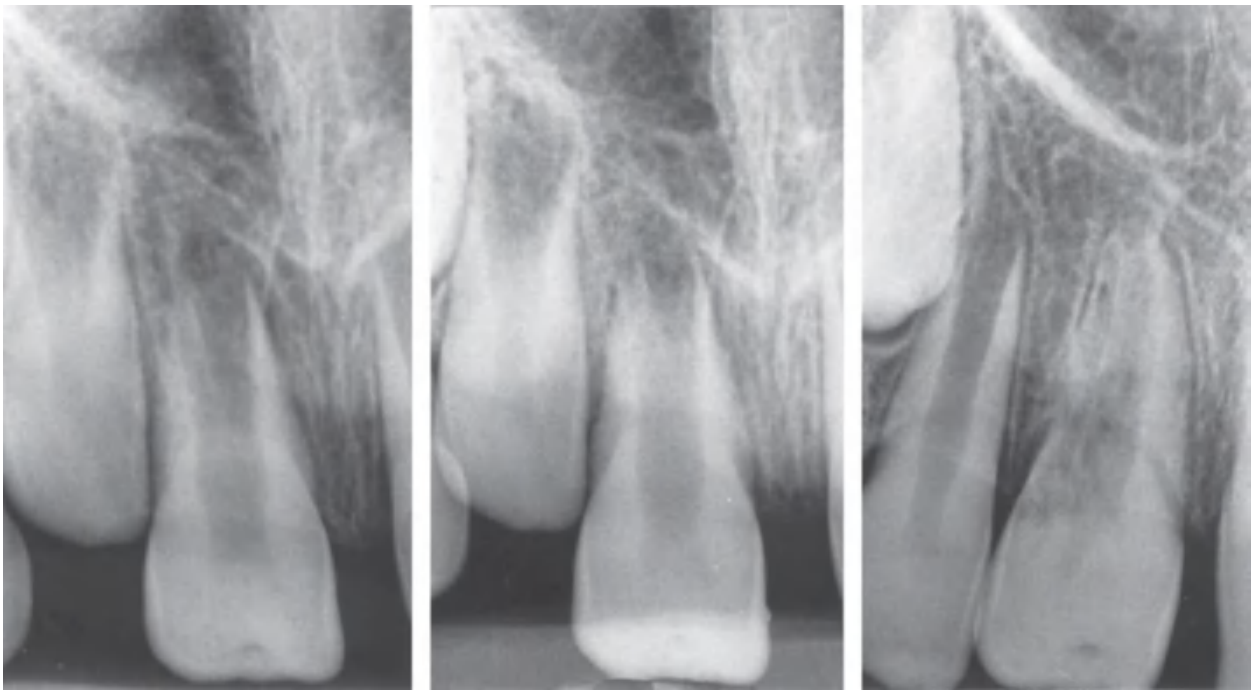



**Fig. 18.14** External replacement resorption of a central incisor following lateral luxation. At the time of splint removal, 1 month after injury, there is no evidence of root resorption. At the 1-year follow-up, ankylosis is evident distally.

structures, persistent tenderness to percussion in the follow-up period was found to be significantly related to infected pulp necrosis (15).

A comprehensive review of pulpal response following acute dental injury has been recently published (17).

## Marginal bone loss

### Extrusion

As expected, this trauma group showed limited (6) or no loss of marginal bone (8) due to the limited nature of the injury (i.e. rupture of the periodontal ligament) (Table 18.1).

### Lateral luxation

In this trauma group, loss of marginal bone was seen, which is explained by the compressive nature of the injury in the cervical region (6, 8) (Table 18.2, Fig. 18.15).

## Predictors of healing

In a multivariate analysis the strongest predictor appeared to be the stage of root development (6). The relation to root development was further refined by measuring the apical diameter (13). Moreover, it was found that increased age after completed root development was significantly related to pulp necrosis (6).

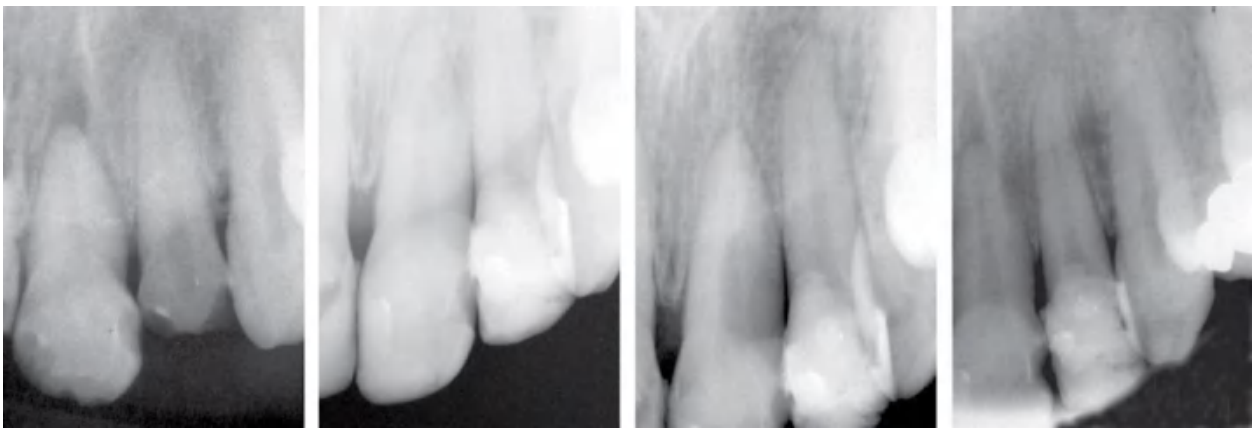



**Fig. 18.15** Transient marginal breakdown in a 46-year-old male who suffered lateral luxation of the right central incisor. Three months after repositioning, there is no sign of pathology. Four months after repositioning, there is marked breakdown of bone between the central and the lateral incisors. Four months later the interdental bone has been reformed. Courtesy of Dr. H. Lund, Kolding, Denmark.

**Table 18.3** Risk of pulp necrosis in cases of extrusion with or without crown fractures.

| Study | | Immature root development | | Mature root development | |
|---|---|---|---|---|---|
| | | No. of teeth | Risk of pulp necrosis* | No. of teeth | Risk of pulp necrosis* |
| Lauridsen et al. (12) 2012 | No crown fracture | 34 | 5.9% | 23 | 56.5% |
| | Associated crown fracture | 8 | 0% | 17 | 76.5% |

* The risk estimate at the 1-year control.

**Table 18.4** Risk of pulp necrosis in cases of lateral luxation with or without associated crown fractures.

| Study | | Immature root development | | Mature root development | |
|---|---|---|---|---|---|
| | | No. of teeth | Risk of pulp necrosis* | No. of teeth | Risk of pulp necrosis* |
| Lauridsen et al. (12) 2012 | No crown fracture | 43 | 4.7% | 103 | 65.1% |
| | Associated crown fracture | 5 | 40.0% | 28 | 93.0% |

* The risk estimate at the 1-year control.

Another very strong predictor for pulp necrosis appears to an associated crown fracture, which significantly increased the risk of pulp necrosis (12, 18) (Tables 18.3 and 18.4).

In a similar study from China, these two types of luxation had too few cases to arrive at any conclusion (16). Figures 18.16 and 18.17 present the healing predictors for *extrusion* and *lateral luxation*.

## Tooth survival

It appears from clinical studies that excellent long-term tooth survival can be expected for extrusions and lateral luxations (8,10,11).

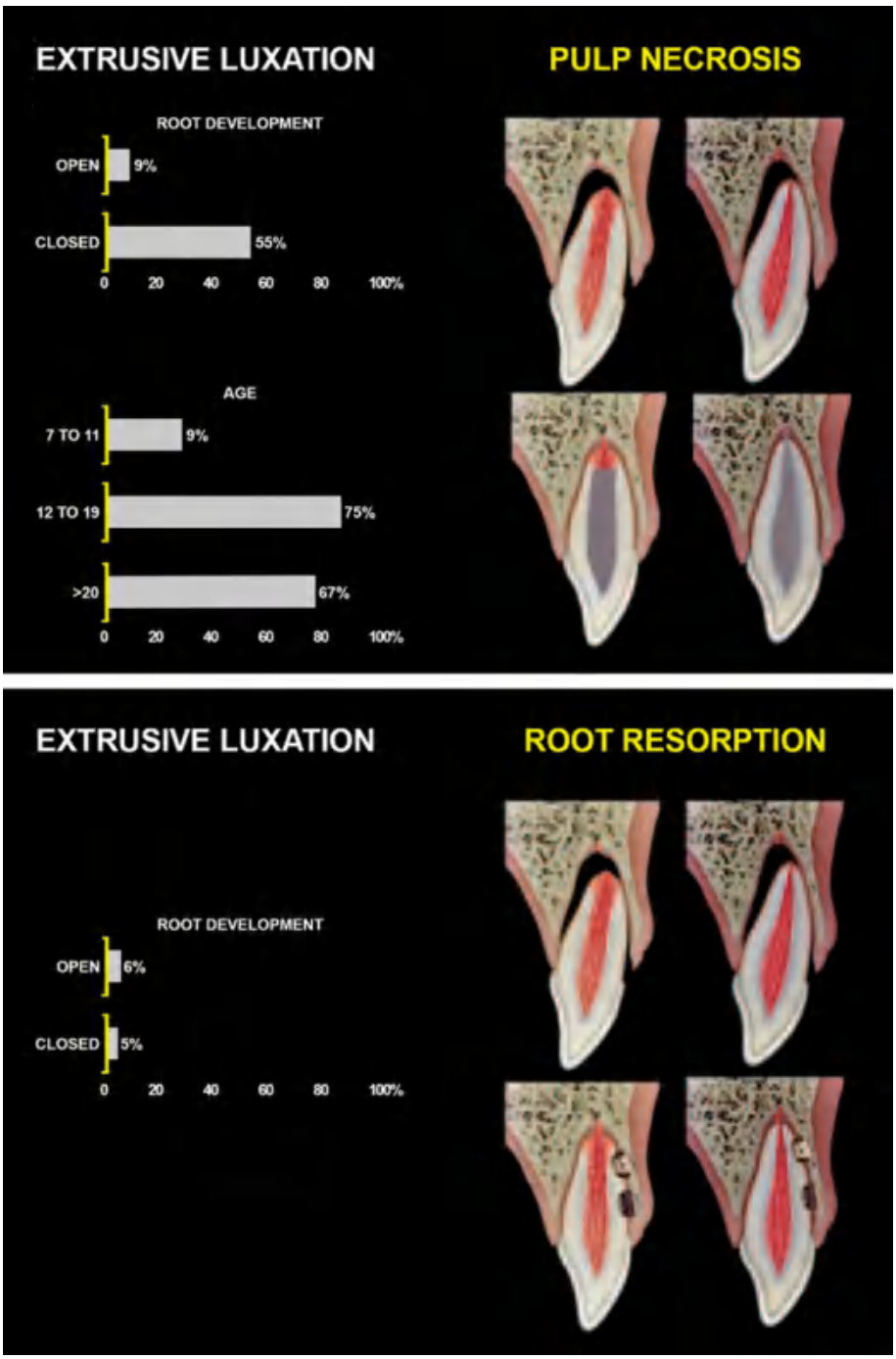



**Fig. 18.16** Predictors for healing outcome after extrusive luxation.

## Essentials

- Administer local anesthesia if forceful repositioning is anticipated (i.e. lateral luxation)
- Reposition the tooth into normal position (Figs 18.6 and 18.8). In cases of delayed treatment, the teeth should be allowed to realign spontaneously into a normal position or be moved orthodontically
- Splint the tooth with an acid-etch/resin splint
- Monitor the tooth radiographically.
- Splinting period:
  a. Extrusion: 2 weeks
  b. Lateral luxation: 4 weeks. In cases of marginal bone breakdown, extend the fixation period to 6–8 weeks
- Follow-up period: minimum 1 year

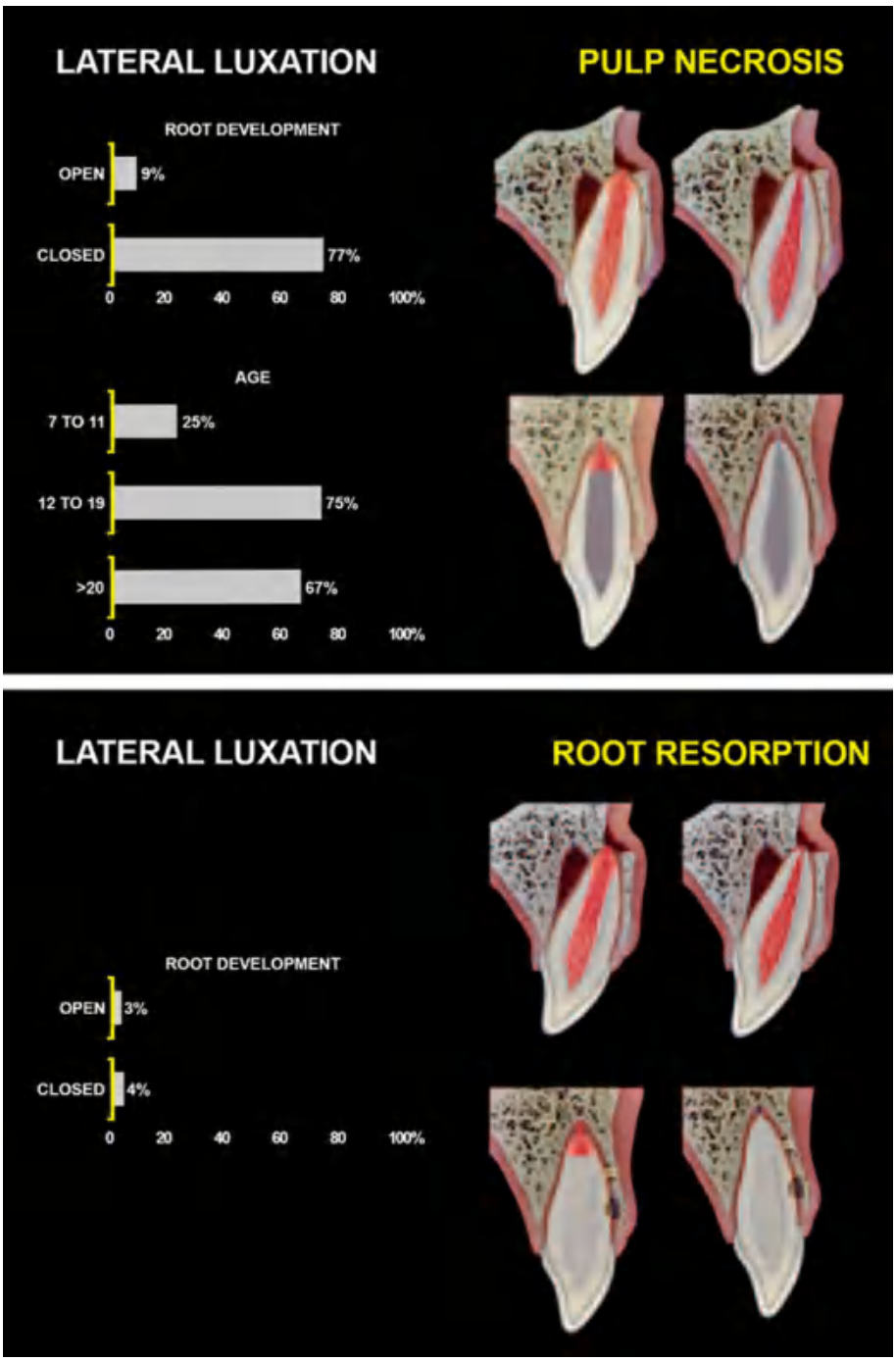



**Fig. 18.17** Predictors for healing outcome after lateral luxation.

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# 19

# Intrusive Luxation

J. O. Andreasen, F. M. Andreasen & G. Tsilingaridis

## Definition

Intrusive luxation (intrusion) is displacement of the tooth into the alveolar bone along the axis of the tooth and is accompanied by comminution or fracture of the alveolar socket (Figs 19.1 and 19.2).

## Frequency

Intrusive luxations have been found to comprise 0.3–2.4% of the traumas affecting permanent teeth (1–5, 25).

## Healing and pathology

An intrusion represents the most severe injury to the dentition through damage to the gingival attachment, contusion of the periodontal ligament and bone and damage to the Hertwig's epithelial root sheath (in the case of immature teeth). Moreover, the intrusive displacement of the tooth will force bacteria from the bacterial plaque covering the dental crown into the compromised wound site (Fig. 19.1). All of these events individually and combined have the potential of eliciting healing complications (see Chapter 2). It is, therefore, no wonder that intruded permanent teeth have a very grave long-term prognosis (20).

With regard to the histopathology changes after intrusion, only a few experimental studies exist, undertaken with rats (6, 7), dogs (8–10, 26) and pigs (27). These are not very comparable to human situations, due to significant differences in tooth anatomy. In one study with dogs, surgical repositioning was compared with spontaneous eruption and it was claimed that surgical repositioning showed more optimal healing; however, no histometric analysis was presented (10).

In a study in humans, intruded permanent teeth were extracted and a histologic examination showed that half of the root surface was denuded of periodontal ligament (28).

## Clinical findings

Due to their locked position in bone, most intruded teeth are not sensitive to percussion and are very firm in their socket. The extent of intrusion may vary from 1 mm to complete burial of the displaced tooth (5). The percussion test often elicits a high-pitched metallic sound, similar to that of an ankylosed tooth. This test is of great importance in determining whether erupting teeth are indeed intruded (5) (Fig. 19.1). A tooth which is completely buried in the alveolar process might be erroneously considered to be avulsed until a radiograph discloses the intruded position. Palpation of the alveolar process can often reveal the position of the displaced tooth.

If a permanent central incisor is completely intruded, it should be remembered that the root apex has most likely been forced into the nasal cavity, resulting in bleeding from the nose. Examination of the floor of the nostril will reveal the protruding apex (Fig. 19.3).

## Radiographic findings

Following intrusive luxation, the periodontal ligament space will be partially or totally obliterated. However, it should be noted that in some cases of obvious displacement, a periodontal space of normal width can still be seen

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**Fig. 19.1** Intrusion of a tooth with incomplete root formation. The semi-erupted position of the tooth leaves doubt as to whether the tooth is undererupted or intruded from a more coronal position. A high percussion (ankylosis) tone, however, reveals the intrusion of the right central incisor.

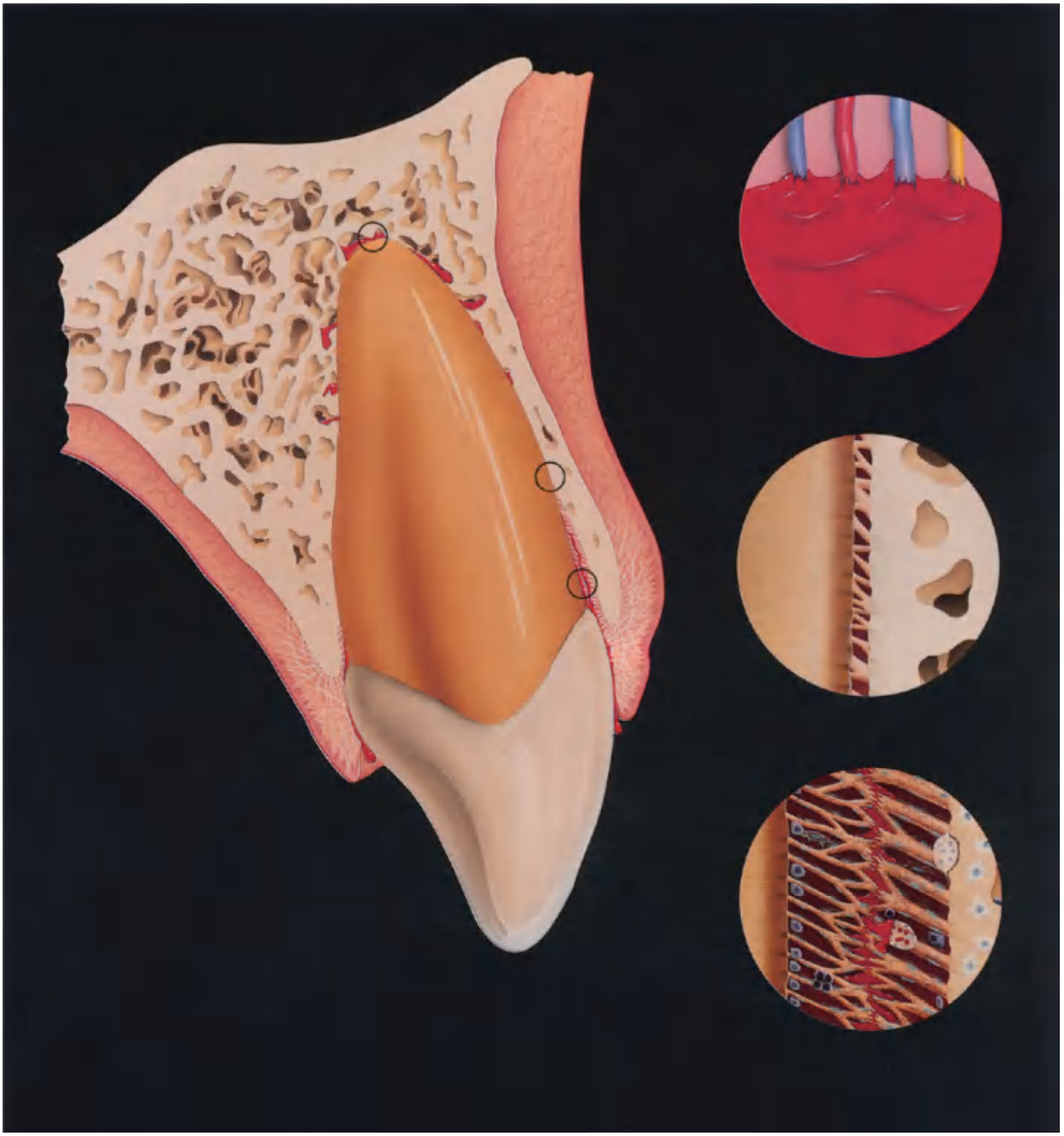
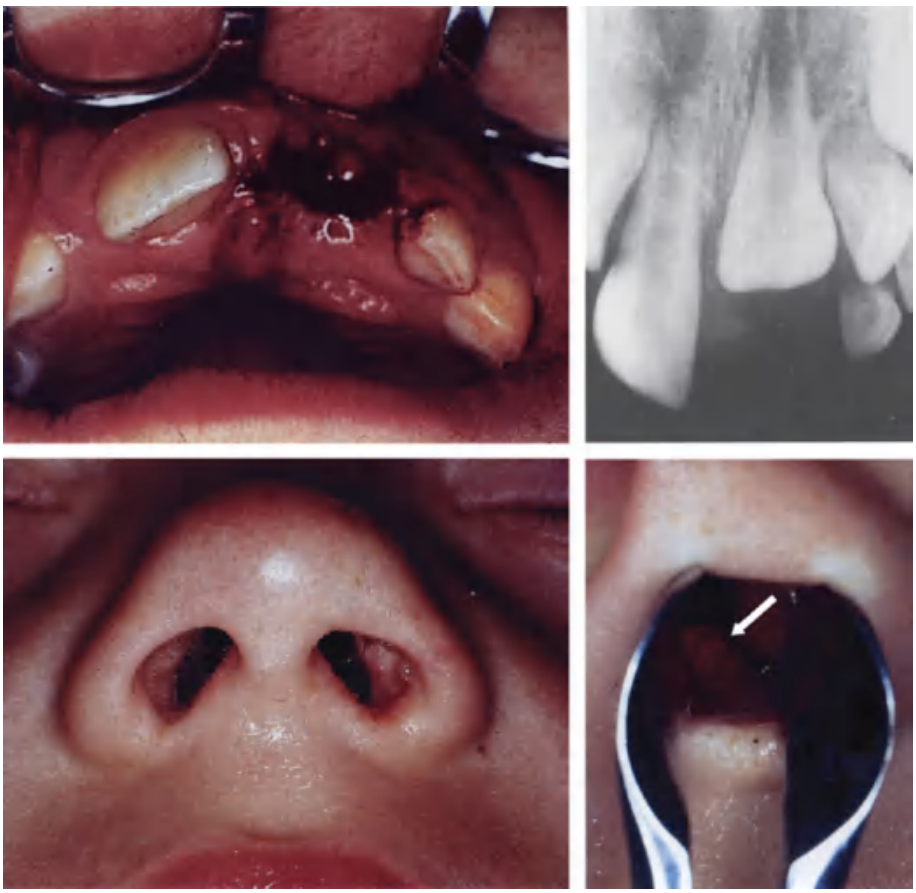
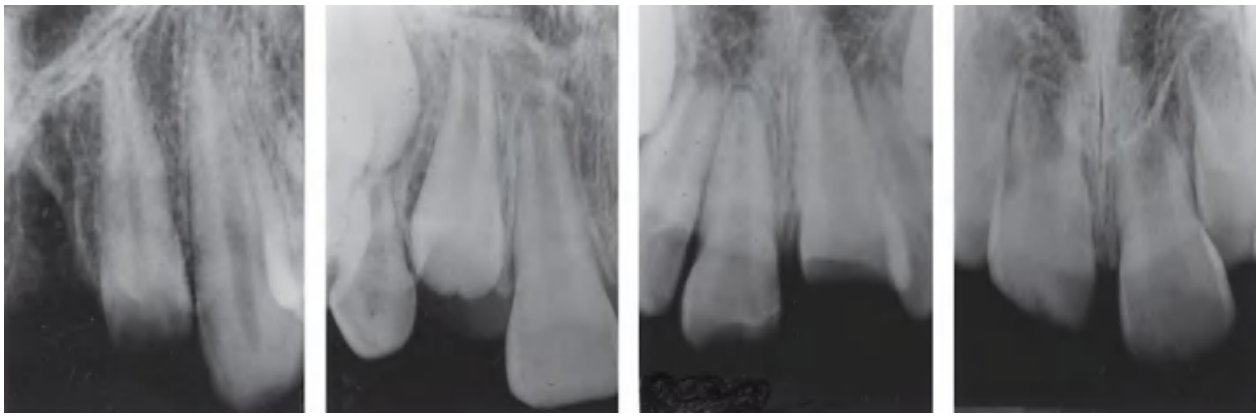


**Fig. 19.2** Pathogenesis of intrusion. Axial impact leads to extensive injury to the pulp and periodontium.

**Fig. 19.3 Displacement of an intruded central incisor into the nasal cavity**
Clinical and radiographic condition. The tooth is completely intruded.

**Nasal inspection**
Inspection of the right nostril with a nasal speculum shows protrusion of the apex through the floor of the nose (arrow).





A B C D

**Fig. 19.4** Radiographic appearance of intrusions. A. Intrusive luxation of a lateral incisor. Note disappearance of the periodontal ligament space around the displaced tooth. B. Intruded lateral incisor. Note that a periodontal space is present along most of the root surface. C and D. Intrusion of teeth with incomplete and complete root formation. Note the difference in position of the cemento-enamel junction compared with the adjacent tooth.

radiographically (5) (Fig. 19.4). In general, when the apex has been forced through the labial bone plate, the intruded incisor will appear foreshortened when compared to the non-injured antimere (Fig. 19.5). Conversely an elongated tooth implies intrusion in the palatal direction (5).

A steep occlusal exposure gives a less reliable impression of the depth of intrusion compared to a conventional periapical (bisecting angle) exposure (Fig. 19.6). Another reliable finding in periapical exposures is the position of the cemento-enamel junction, which in erupted teeth is placed approximately 1 mm incisal to the bony crest (Fig. 19.7).

In the presence of an associated penetrating lip lesion, it is important to take a soft tissue radiograph of the lip in order to diagnose embedded tooth fragments or other foreign bodies (see Chapter 21) (Fig. 19.8).

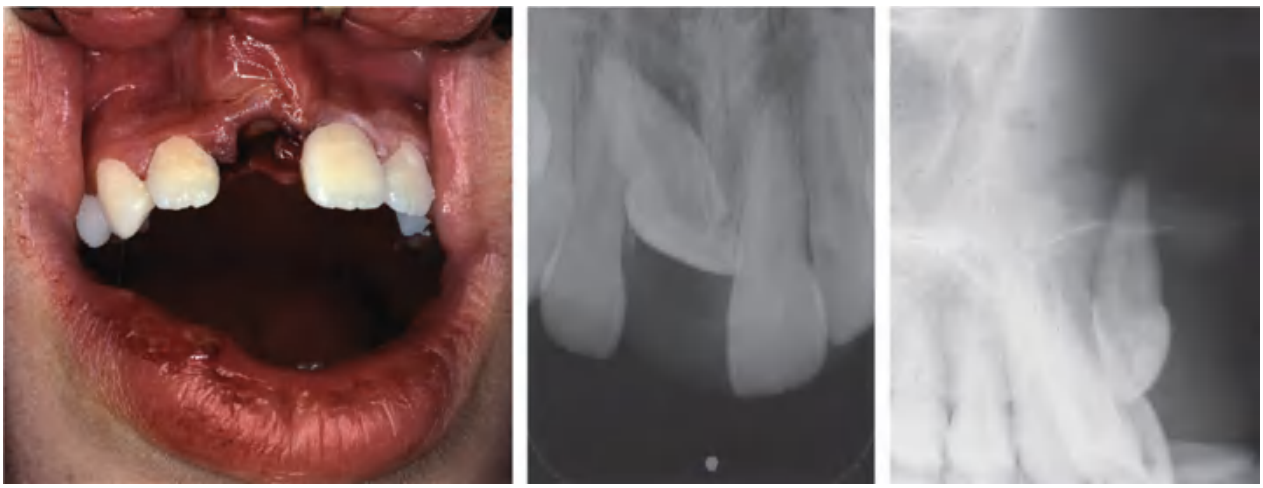


**Fig. 19.5** Radiographically foreshortened intruded central incisor appears on a lateral exposure to be displaced through the buccal bone plate.

**Fig. 19.6** Radiographic appearance related to projection angle. A more steep exposure (i.e. an occlusal exposure) does not yield a reliable picture of the depth of intrusion compared to a periapical bisecting angle exposure.

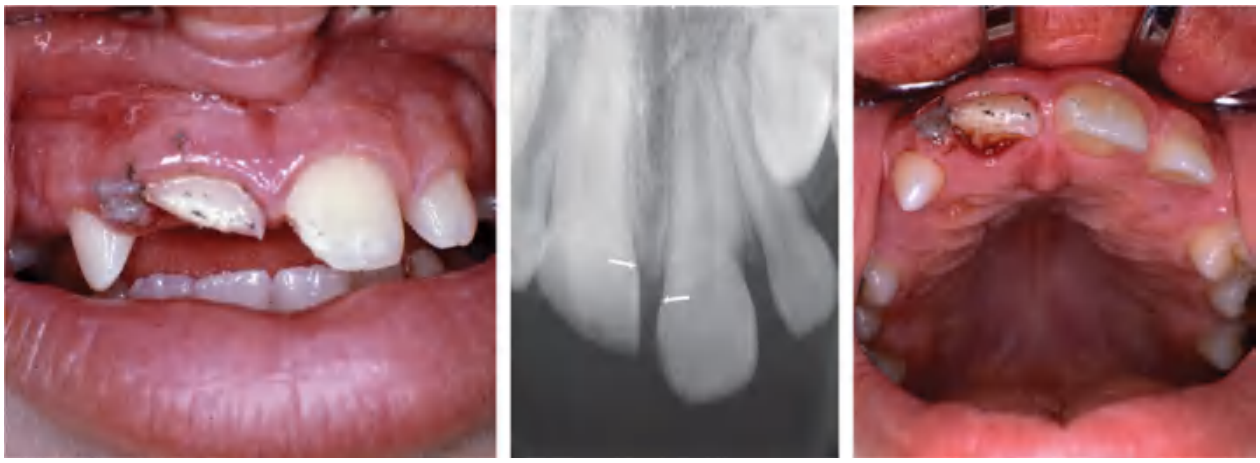


**Fig. 19.7** Slightly intruded permanent right central incisor. Note that the cemento-enamel border is positioned more apically than the non-injured antimere.

**Fig. 19.8** Diagnosis of foreign bodies in the lower lip in a case with combined intrusion injury and penetrating lip injury. Frontal and lateral radiographs disclose multiple foreign bodies in the lower lip.

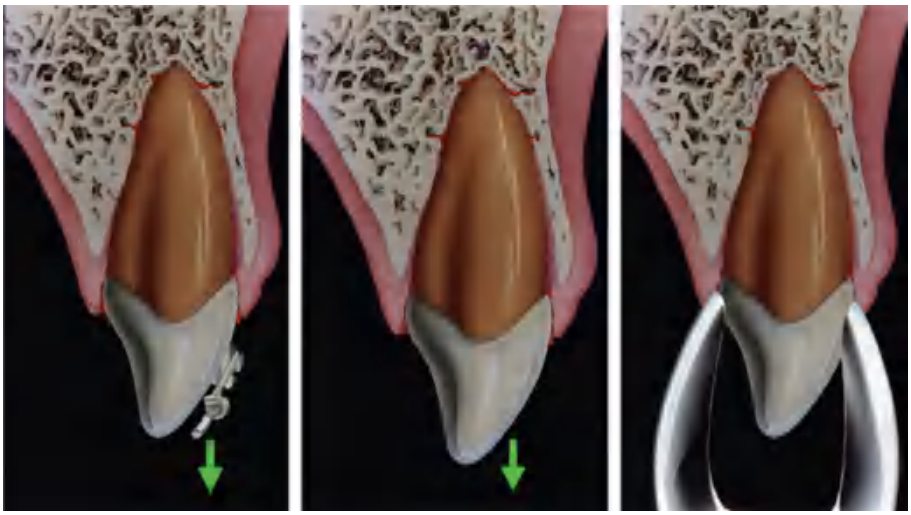



**Fig. 19.9** Treatment principles for intrusion: orthodontic extrusion (A), spontaneous eruption (B) or surgical extrusion (C).

## Treatment

In the past, significant controversy has existed concerning the optimal treatment for intruded permanent teeth (5, 11, 22, 24). Three treatment modalities are currently in use for the treatment of permanent teeth with intrusive luxation: spontaneous re-eruption, orthodontic repositioning and surgical repositioning (33) (Fig. 19.9).

It has been demonstrated that intruded teeth with immature root formation may re-erupt spontaneously (11–14, 20, 21, 31, 32); this has also been seen in some cases with completed root formation (20–23, 31, 32). The advantage of this laissez-faire treatment philosophy is that no additional damage from active repositioning procedures is inflicted upon the already injured periodontium, a factor which should optimize marginal bone healing. This procedure has usually been confined to patients with immature root formation.

There are situations and cases where spontaneous re-eruption can be unreliable, so the second treatment philosophy has been to stimulate spontaneous re-eruption by orthodontically guided eruption (17).

Finally, surgical repositioning has been suggested, whereby the intruded tooth is brought back to its original position and splinted (11).

A major obstacle in the analysis of the value of these treatment alternatives has been the lack of high quality studies, including ones with sufficiently large numbers of patients, as well as the difficulties in conducting a clinical randomized study (29). However, in a large, retrospective, Scandinavian multicenter study of 230 intruded teeth, the result of the three treatment models were compared with respect to pulp necrosis and replacement resorption (34). This study indicated that root development, and degree of intrusion, are important for the development of pulp necrosis as well as replacement resorption, whereas choice of treatment only seems to influence the development of replacement resorption in intruded permanent teeth. Awaiting re-eruption resulted in fewer teeth developing pulp necrosis and replacement resorption (34).

## Spontaneous re-eruption

This appears to be the treatment of choice for teeth with incomplete root formation (32). To facilitate the re-eruption process, the intruded tooth may be slightly loosened with forceps at the first visit (to release the mechanical grip of the bony walls on the root surface). Thereafter, re-eruption is awaited, which usually takes on average 6 months for completion (range 1–14 months) (21, 31, 32) (Fig. 19.10).

If clinical and radiographic examinations show no signs of re-eruption after 1 month, and, in particular, the percussion test shows signs of ankylosis, the tooth must be loosened with forceps and brought to occlusal height orthodontically. Furthermore, spontaneous re-eruption has been found to take place also in teeth with mature root development and only mild to moderate intrusion (34) (Fig. 19.11). However, spontaneous re-eruption may take several months, and the risk for pulp necrosis and infection-related resorption is high. Therefore, if spontaneous re-eruption is the treatment of choice, it is important, throughout the eruption process, that these teeth are monitored very carefully so endodontic treatment can be initiated if pulp necrosis and infection-related resorption is diagnosed. A very unusual case has been reported where an intruded incisor showed spontaneous eruption after 15 years (30). Suggested observation periods are listed in Appendix 3.

## Orthodontic extrusion

The general indication for orthodontic extrusion will be in cases with completed root formation. This strategy appears to result in slightly better bone healing. Ideally, orthodontic extrusion should be performed at a pace that keeps up with the repair of marginal bone. Furthermore, it is important that the tooth is sufficiently repositioned within 2–3 weeks to ensure access to the pulp chamber should endodontic treatment be necessary. In some cases a surgical exposure of the palatal crown surface may enable access to the pulp. This is important as pulp necrosis and infection-related resorption may be initiated by this time, and the only way to arrest this process is by endodontic therapy (see Chapter 25). This is also the reason for not awaiting spontaneous eruption in cases with severe intrusion and completed root formation.

Orthodontic movement of intruded permanent teeth can begin at the initial examination at the time of injury or some days later, when swelling has subsided (Fig. 19.12). The orthodontic appliances used for extrusion are similar to those used for extrusion of crown-root-fractured teeth (see Chapter 28).

If the tooth has been completely intruded, it is essential that it is partially repositioned with a forceps after administration of local anesthesia so that half of the crown is exposed. This will hasten the final re-eruption and facilitate application of an orthodontic bracket on the labial surface.

**Fig. 19.10** Spontaneous eruption of two intruded incisors. A. Clinical condition in a 7-year-old girl after an axial impact. B. Condition 6 weeks later, after onset of eruption. C. Follow-up 1 year after injury. Eruption is complete.

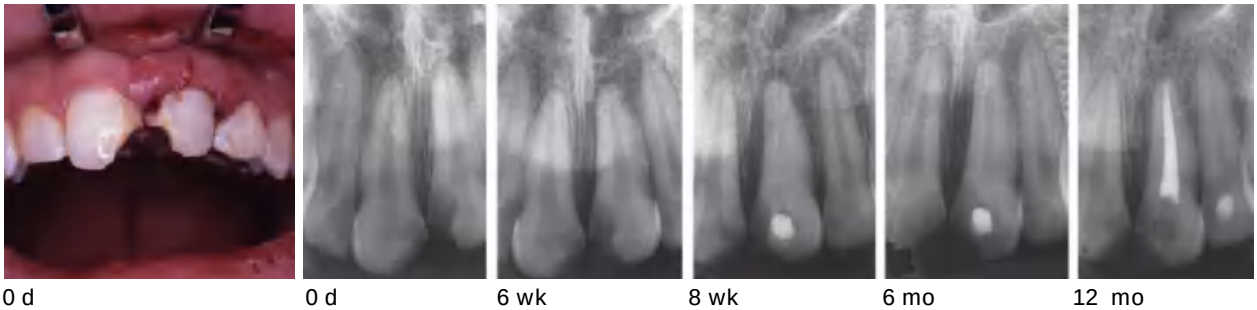



**Fig. 19.11** Spontaneous eruption of an intruded left central incisor with mature root formation, and condition after 6 and 8 weeks and 6 and 12 months.

**Fig. 19.12 Orthodontic extrusion of an intruded incisor**
Clinical and radiographic condition in a 22-year-old woman after an axial impact. Before application of orthodontic appliances the tooth may be slightly loosened with forceps.

**Covering exposed dentin**
The exposed dentin of both central incisors is covered with a hard-setting calcium hydroxide cement (e.g. Dycal®) and glass ionomer cement.

**Applying orthodontic traction**
A 0.5 mm thick semi-rigid orthodontic wire is adapted to follow the curvature of the dental arch, including two adjacent teeth on either side of the intruded incisor. The orthodontic wire is bonded to the adjacent teeth using an acid-etch technique. In the area where elastic traction is exerted, a wire coil (e.g. 0.228 × 0.901 Elgiloy®) is placed in order to prevent slippage of the elastic.

**Placing the bracket**
The fractured incisal edge is covered with a temporary crown and bridge material. A bracket is placed on the labial surface.

**Orthodontic traction**
Elastic traction of 70–100 g is activated. The direction of traction should extrude the tooth out of its socket in a purely axial direction. Elastic traction should wait for 4–6 days if surgical loosening has been performed.

**Extrusion initiated**
After approximately 10 days, osteoclastic activity around the intruded tooth has usually resulted in loosening; and extrusion can then take place. If extrusion has not yet begun after 10 days, a local anesthetic is administered and the tooth is luxated slightly with a forceps. After 2–3 weeks, a rubber dam is applied, the pulp extirpated and the root canal filled with calcium hydroxide paste.

**Extrusion complete**
After 4 weeks, the intruded tooth is extruded to its original position and retained in this position for 2–4 weeks. Thereafter, the orthodontic appliance can be removed.

**Crown restoration**
The fractured crowns are restored with composite resin.

In some cases, it can be an advantage to luxate the tooth slightly prior to orthodontic extrusion. If this alternative is chosen, orthodontic extrusion should be postponed for a few days to avoid avulsion of the tooth upon activation of the appliance (21).

### Surgical extrusion

This procedure implies immediate repositioning of the tooth into its normal position. After administration of local anesthesia, the tooth is grasped with a forceps (preferably proximally) and brought down into its normal position. Thereafter, the displaced labial and palatal bone is repositioned by finger pressure and the gingival lacerations sutured (Fig. 19.13). A splint is applied and maintained for 6–8 weeks.

This procedure is primarily indicated in teeth with severe intrusion and mature root formation (34) (Fig. 19.14). With multiple intrusions that have been surgically repositioned, there may be difficulty in reaching stability after repositioning. This must be taken into consideration when fabricating a splint, which has to be extended bilaterally to involve stable teeth.

### Treatment guidelines according to trauma scenario

In order to provide evidence-based treatment, certain pre-trauma and trauma factors must be considered in order to choose the correct treatment alternative (i.e. spontaneous repositioning, orthodontic extrusion or surgical repositioning) (21).

The stage of root development (complete or incomplete) appears to be one of the strongest factors. In the case of incomplete root formation, the extent of intrusion becomes decisive. There is a cut-off at the ages of 12 years and >17 years with respect to choice of treatment (Fig. 19.15). Whether there is one intruded tooth or multiple intrusions also needs to be taken into account. The treatment guidelines shown in Fig. 19.15 are only partially supported by a clinical study (21).

## Prognosis

A number of studies have been carried out on the long-term prognosis after intrusive luxation. All of these studies indicate that severe complications dominate the healing pattern (Table 19.1). One of the most common complications is the occurrence of pulp necrosis in teeth with immature root formation. Furthermore, ankylosis and loss of marginal bone are of concern as these complications may lead to tooth loss (17, 18, 20, 21, 34).

## Diagnosis of healing complications

### Pulp necrosis

It appears from Fig. 19.16 that pulp necrosis (PN) is significantly related to the stage of root development and can in most cases be diagnosed within 2 months after injury (34). However, time from trauma to diagnosis of pulp necrosis is longer in very immature and mildly intruded teeth since there is a higher chance of revascularization (34). Furthermore, in some cases a late diagnosis of PN is made due to the effect of partial pulp necrosis, whereby Hertwig's epithelial root sheath closes the root apex with hard tissue irrespective of necrotic root canal contents (see Chapter 25) (Fig. 19.17).

### Root resorption

The most frequent type of root resorption (RR) appears to be infection-related resorption, followed by ankylosis (replacement resorption) and repair-related resorption (20, 34).

In cases of infection-related root resorption, the resorption process can be arrested in the majority of cases by endodontic therapy (see Chapter 25).

In approximately one-fourth of all cases with ankylosis, the resorptive process appears to be transient.

From Fig. 19.18 it appears that the diagnosis of RR is related to the stage of root development, where teeth with a mature root development are at higher risk at developing replacement resorption. Most cases can be diagnosed within the first year (34). While a relatively stable plateau of healing takes place up to 5 years after injury of immature teeth, new RR activity can be found as late as 10 years after injury (20,21).

### Marginal bone loss

Marginal bone loss is a frequent complication and is related to stage of root development (Fig. 19.19).

Marginal bone loss may be transient in nature (Fig. 19.20). This phenomenon is further described in Chapter 16. Furthermore, bone loss is more often found on 'proximal' surfaces compared to distal in cases of multiple intrusions (20, 21) (Fig. 19.21).

The majority of marginal bone loss takes place in cases of incomplete root development within the first 4 years after injury; whereas teeth with completed root formation show a constant amount of bone loss throughout the observation period.

### Survival of intruded teeth

Two studies provide data that enable survival curves to be constructed. In both studies, long-term survival of intruded teeth appears far from optimal (16, 18, 21).

It appears from Fig. 19.22 that there is a fairly constant loss of teeth throughout the observation period for teeth

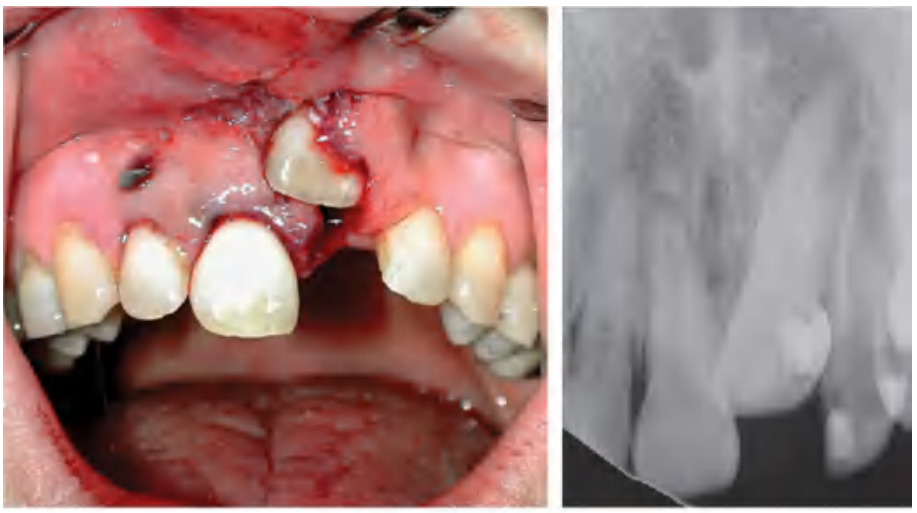
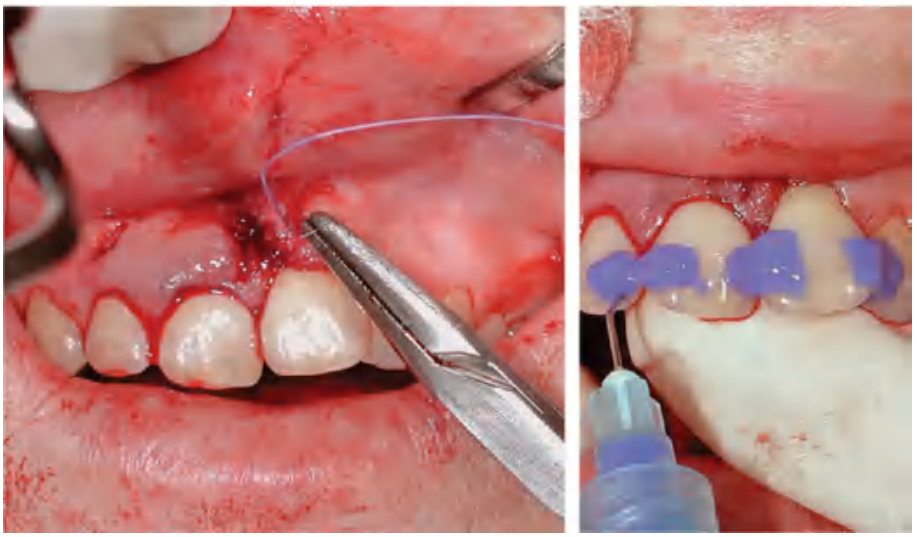


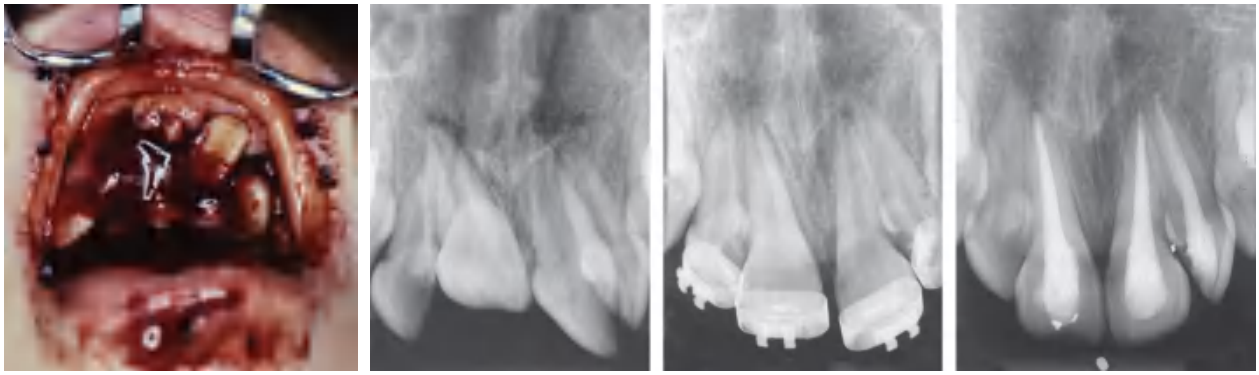
**Fig. 19.13 Surgical repositioning of an intruded left central incisor and repositioning and splinting of an extruded and root-fractured right central incisor**
Clincial and radiographic appearance.

**Repositioning**
After local anesthesia, the right central incisor is repositioned. The left central incisor is grasped proximally with forceps and repositioned. With finger pressure, the tooth is repositioned, as well as the displaced labial bone.

**Suturing and splinting**
The lacerated gingiva is sutured. The incisal third of the labial enamel of all incisors is acid etched and splinting material applied.



**Finishing the splint**
A Luxatemp® splint has been applied.



A B C D

**Fig. 19.14** Treatment of multiple severely displaced and intruded incisors. A–C. Multiple intrusions (three incisors) in a 10-year-old girl treated by surgical repositioning and splinting. D. Condition 6 months later. There is no evidence of root resorption.

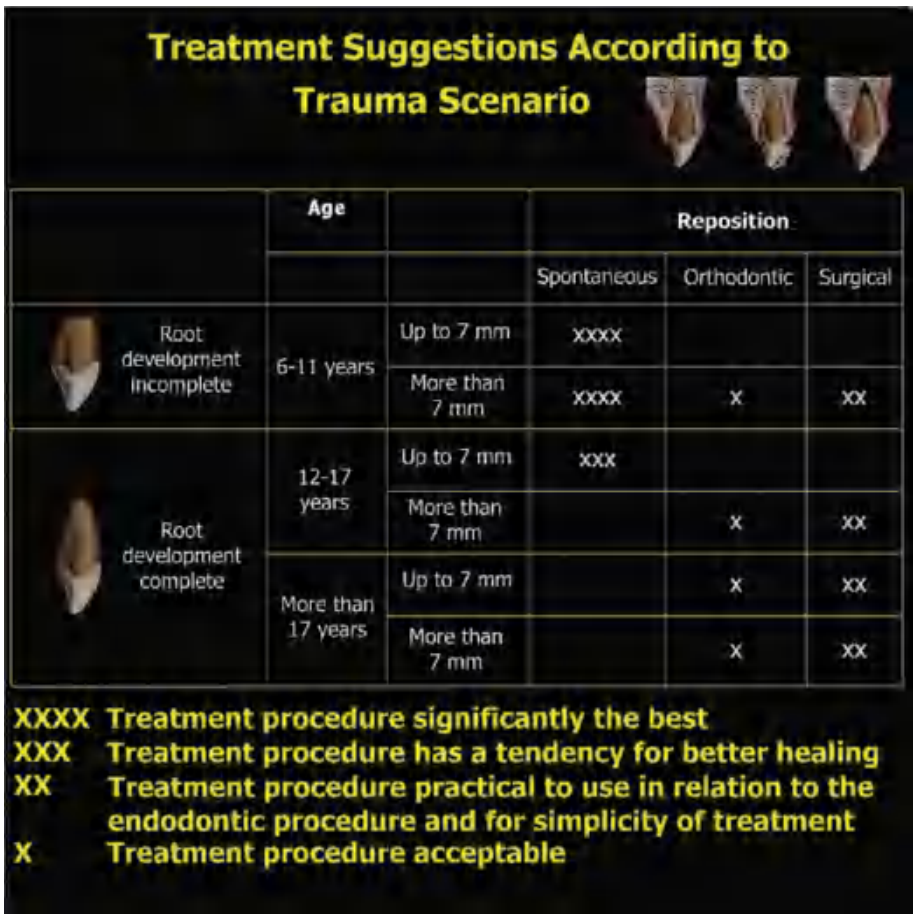



**Fig. 19.15** Treatment related to the trauma scenario.

**Table 19.1** Clinical studies on the prognosis of intrusion of permanent teeth.

| Study | No. of teeth | Age mean, years (range) | Tooth survival | Pulp necrosis | Root resorption | Loss of marginal bone |
|---|---|---|---|---|---|---|
| Andreasen (11) 1970 | 23 | | ? | 22 (96%) | 12 (52%) | 11 (48%) |
| Andreasen & Vestergaard Pedersen (12) 1985 | 61 | (6–67) | ? | 52 (85%) | 40 (66%) | 19 (31%) |
| Jacobsen (13,14) 1983, 1991 | 40 | 8.0 (6–16) | 36 (90%) | 25 (63%) | ? | ? |
| Kinirons & Sutcliffe (15) 1991 | 29 | 9.5 (7–12) | 20 (69%) | ? | 11 (38%) | 7 (24%) |
| Ebeseleder et al. (16) 2000 | 58 | 11.1 (6–16) | 55 (95%) | 36 (64%) | 18 (31%) | 20 (34%) |
| Al-Badri et al. (17) 2002 | 61 | 9.3 (7.1–14) | 48 (79%) | ? | 36 (59%) | ? |
| Humphrey et al. (18) 2003 | 31 | 9.3 (6–18) | 26 (83%) | 14 (45%) | 25 (80%) | 12 (39%) |
| Chaushu et al. (19) 2004* | 31 | 8–11 | 28 (90%) | 26 (83%) | 13 (41%) | 2 (6%) |
| Wigen et al. (31) 2008 | 51 | ? (6–18) | 40 (57%) | 39 (57%) | (38%) | ? |
| Tsilingaridis et al. (32) 2012 | 60 | 9.4 (6–16) | 49 (81%) | 43 (41%) | 31 (72%) | ? |
| Andreasen et al. (20) 2006 | 140 | 15.6 (6–67) | 112 (80%) | 124 (88%) | 67 (48%) | 45 (32%) |

* Including 22 case reports from the literature, all treated by orthodontic extrusion.

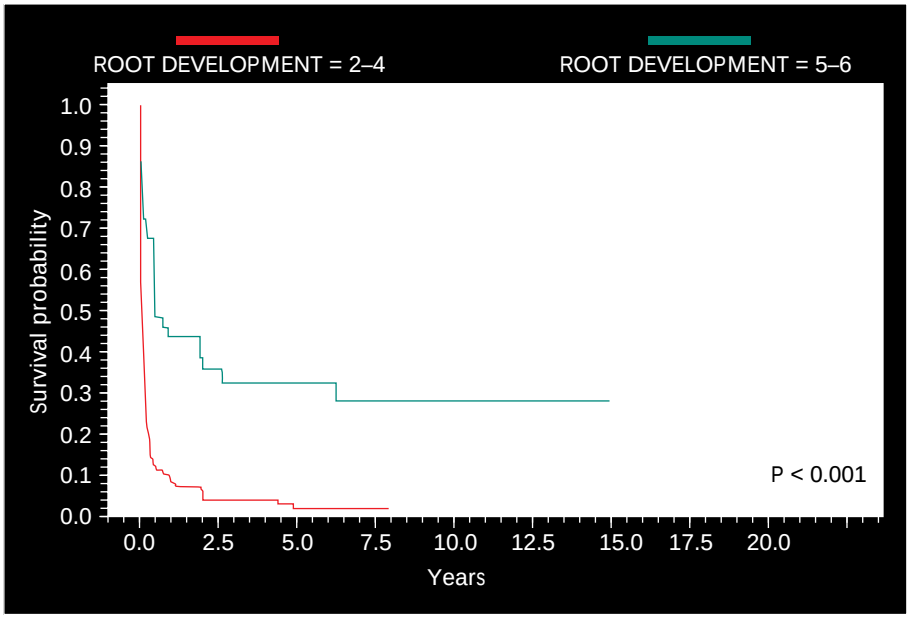



**Fig. 19.16** Pulp necrosis related to root development. From (20).

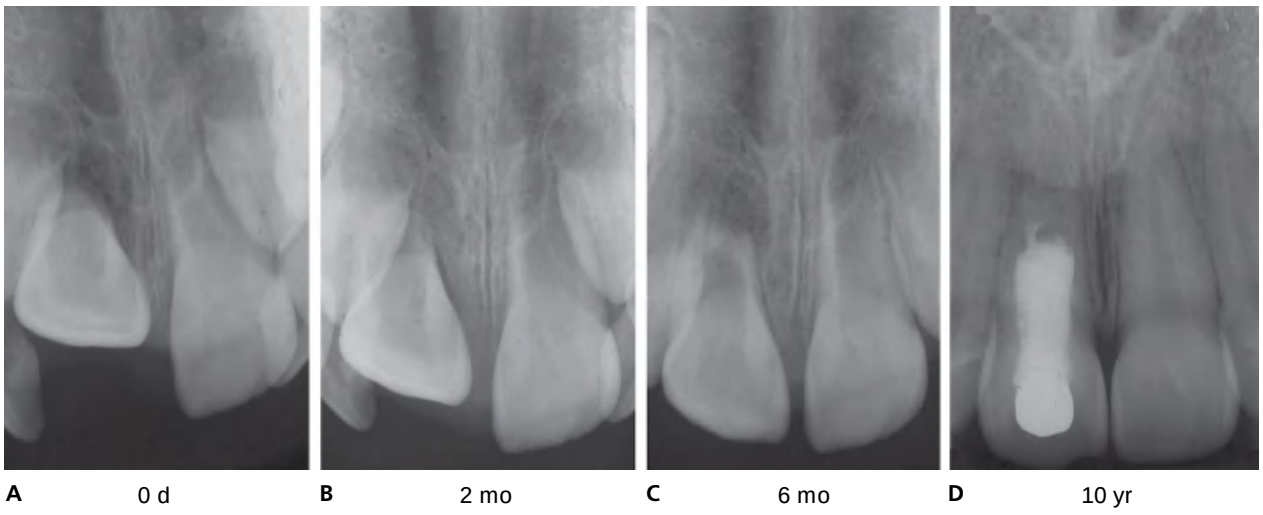



**Fig. 19.17** Partial necrosis of a right central incisor. A. Time of injury. B. Status after 2 months: spontaneous eruption is taking place. C. 6 months: full eruption and apical closure. The coronal part of the pulp has developed pulp. D. Status after 10 years.

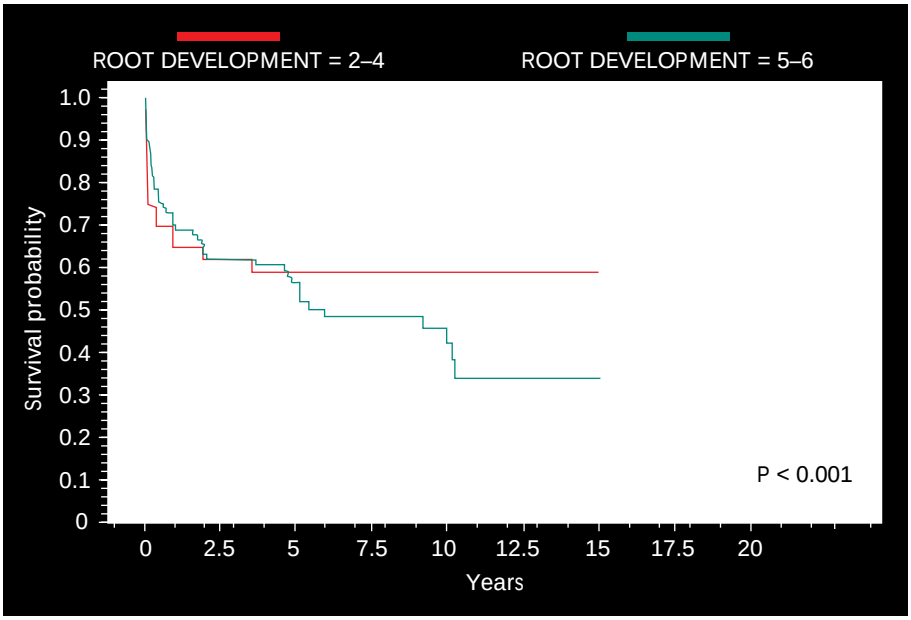



**Fig. 19.18** Root resorption related to root development.

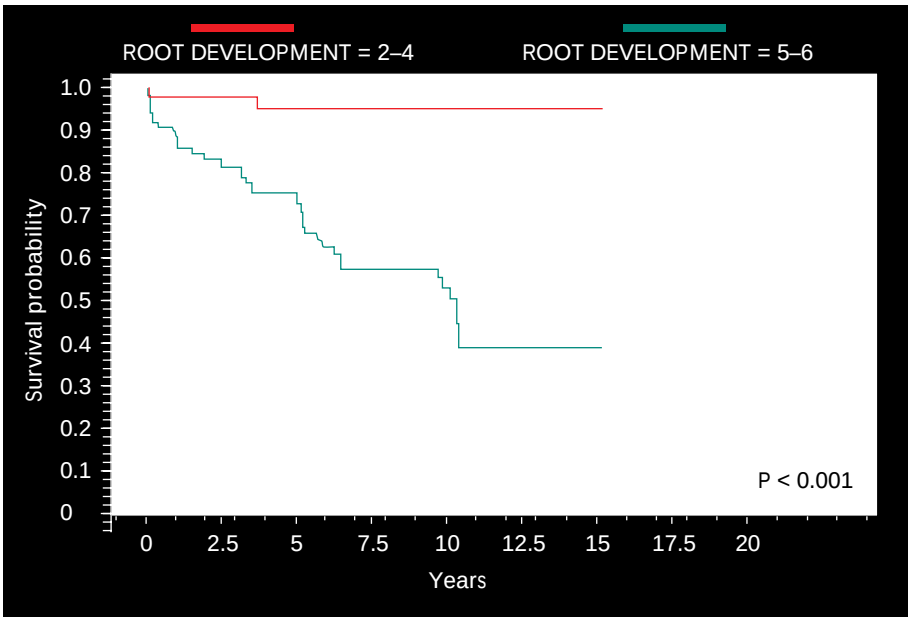



**Fig. 19.19** Marginal bone loss related to root development.

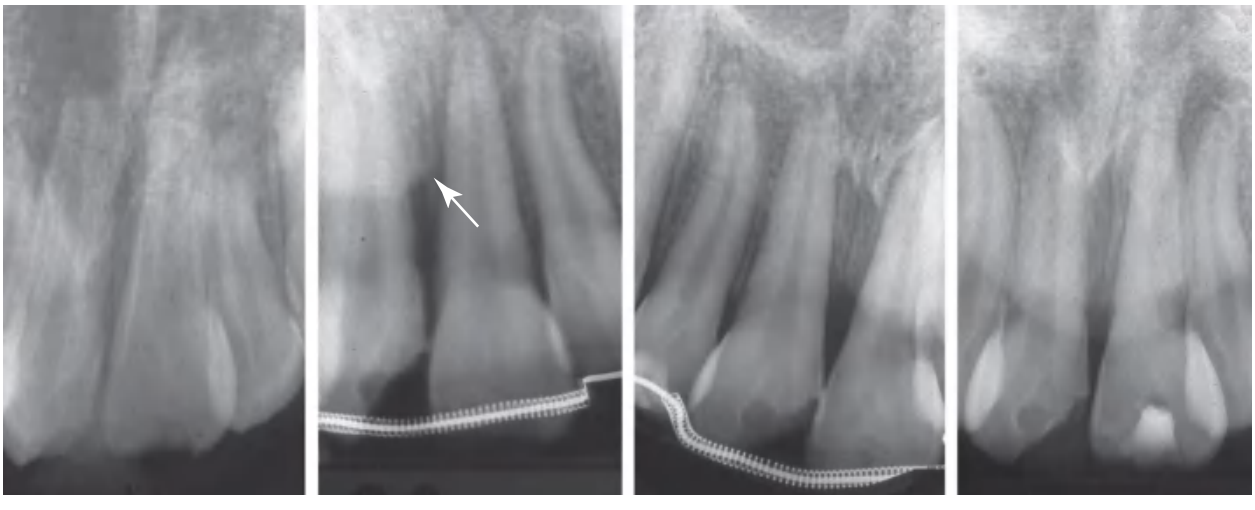



**Fig. 19.20** Transient marginal breakdown around an intruded left central incisor (arrow).

**Fig. 19.21** Multiple intrusions in a 16-year-old boy. More marginal bone loss is found 'proximally'.

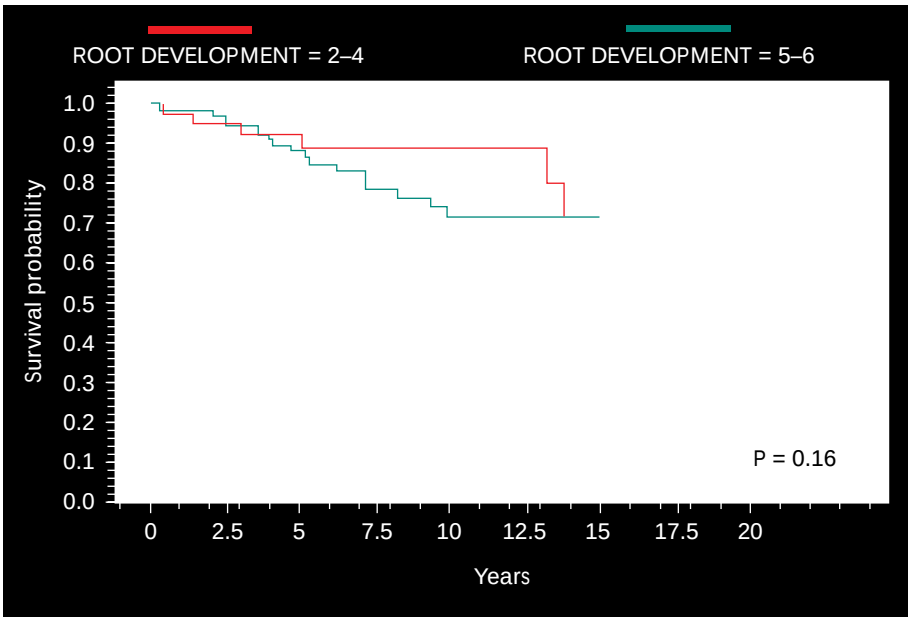



**Fig. 19.22** Tooth loss related to root development.

with incomplete and completed root formation. The choice of treatment procedure seems to favor spontaneous eruption and orthodontic repositioning (Fig. 19.23). Furthermore, approximately 30% of the intruded teeth are lost after 15 years irrespective of root development.

## Predictors for healing

Two recent studies have isolated a number of predictors for pulp necrosis, root surface resorption and loss of marginal bone support (20, 32). These predictors are shown in Figs 19.24 and 19.25. It appears that *stage of root development*, *extent of intrusion* and *method of repositioning* are most informative about the risk of complications.

## Treatment guidelines

The International Association for Dental Traumatology (IADT) has developed guidelines for the treatment of intruded permanent teeth (33). Teeth with immature root development with an intrusion depth <7 mm are recommended to left to spontaneously re-erupt. In teeth with immature root development with an intrusion depth >7 mm the guidelines recommend surgical or orthodontic repositioning. Furthermore, the guidelines recommend spontaneous re-eruption for mature teeth with an intrusion depth <3 mm. For mature teeth intruded >7 mm, orthodontic or surgical repositioning is recommended. For teeth intruded 3–7 mm no treatment recommendations are given in the guidelines. These recommendations are partially supported by a few clinical studies (20, 21, 31, 34).

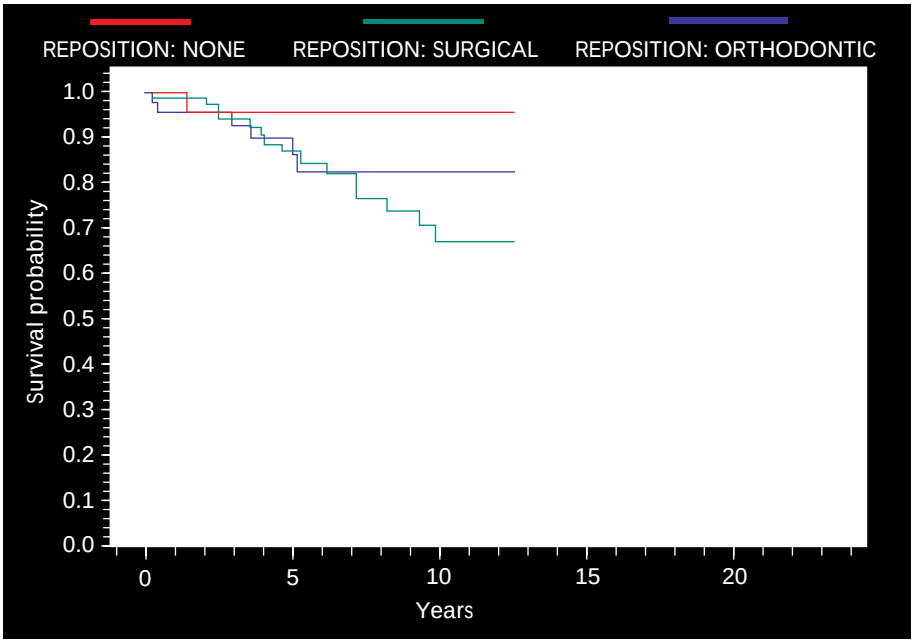



**Fig. 19.23** Tooth loss related to treatment procedure.

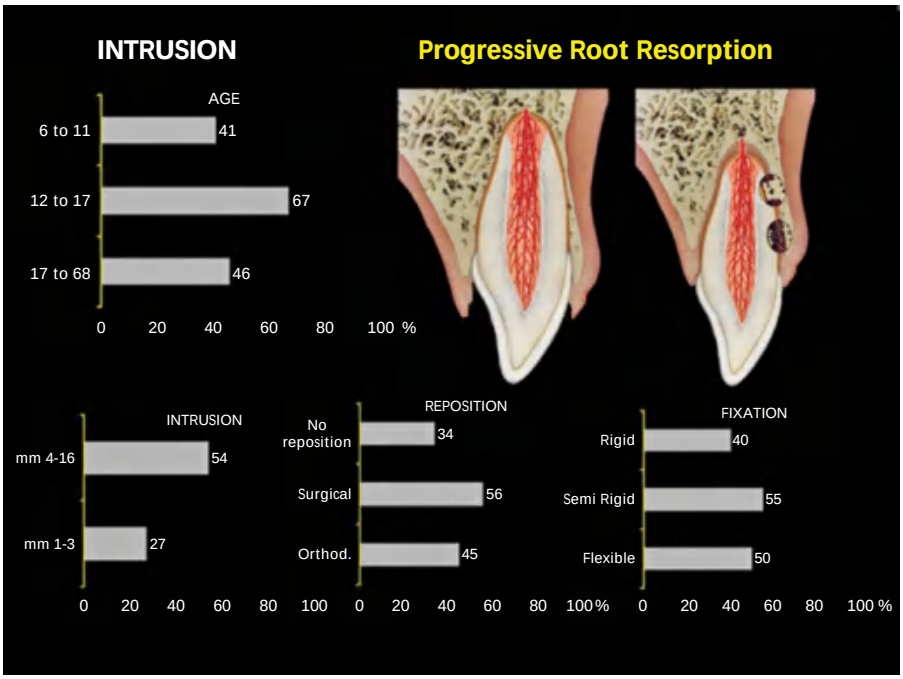



**Fig. 19.24** Predictors for progressive root resorption.

In the systematic review by Al-Khalifa et al. (29), one of the implications for future research was that high quality observational studies should be conducted with larger numbers of patients. In Denmark, Sweden and Norway the same protocol for examination and follow-up of permanent teeth with intrusive luxation has been used for several years, providing an opportunity to conduct a restrospective multicenter study (32). This multicenter study in part supports the IADT guidelines.

Regarding teeth with immature root development with an intrusion depth <7 mm, the DiAngelis et al. study supports the guidelines and recommends spontaneous re-eruption (33). In immature teeth with an intrusion depth of 7 mm the study indicates that awaiting re-eruption may be a favorable treatment, as well as orthodontic and surgically repositioning.

In mature teeth with an intrusion depth <3 mm the study supports the IADT recommendation of spontaneous re-eruption. For mature teeth with an intrusion depth of 3–7 mm, awaiting re-eruption resulted in the fewest complications. If the choice of treatment is awaiting re-eruption it is important that these teeth are monitored very carefully during re-eruption so endodontic treatment can be initiated if pulp necrosis is diagnosed.

In mature teeth with an intrusion depth >7 mm, orthodontic or surgical repositioning is recommended. Although,

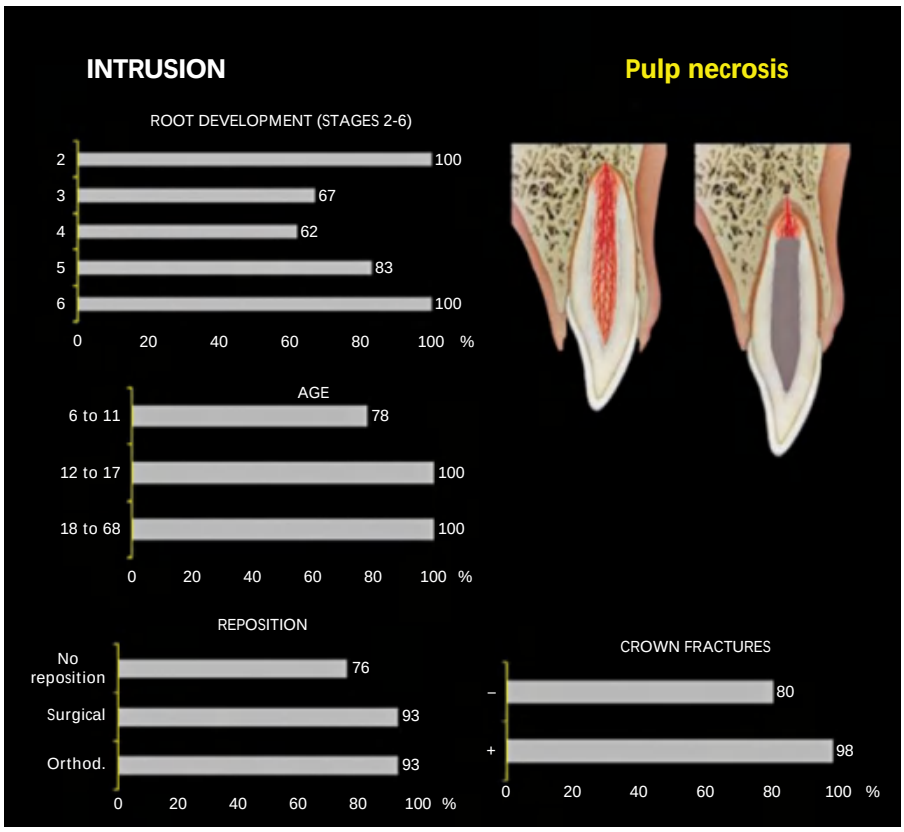



**Fig. 19.25** Predictors for pulp healing.

there is no significant difference between orthodontic and surgical repositioning, and even though orthodontic repositioning seems to have a slightly better healing outcome, this small difference is of no importance compared with the practicality of endodontic access, financial issues, number of visits and quality of life for the patient. Therefore, surgical repositioning is the preferred treatment.

## Essentials

### Treatment alternatives

- *Spontaneous repositioning* should be anticipated in all teeth with incomplete root formation.
- *Surgical repositioning* is the treatment of choice in cases of mature teeth and severe intrusion (i.e. >7 mm).
- Surgical repositioning is preferred over orthodontical extrusion in teeth with mature root development and severe intrusion because of endodontic practicality.
- Gingival lacerations should be *sutured*.
- Conduct *radiographic controls* during the re-eruption phase.
- If signs of periapical radiolucency and/or inflammatory resorption are seen, *endodontic treatment* is indicated. In cases of completed root formation, prophylactic pulp extirpation is indicated with gutta-percha root-filling and sealer.

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# 20
# Avulsions

J. O. Andreasen, F. M. Andreasen & G. Tsilingaridis

## Terminology, frequency and etiology

*Tooth avulsion* (exarticulation, total luxation) implies total displacement of the tooth out of its socket.

Various statistics have shown that avulsion of teeth following traumatic injuries is relatively infrequent, ranging from 0.5 to 16% of traumatic injuries in the permanent dentition (1–5, 125, 126). The main etiologic factors in the *permanent dentition* are fights and sports injuries (1, 127).

## Clinical findings

The maxillary central incisors are the most frequently avulsed teeth, while the lower jaw is seldom affected (6–8, 128, 129, 199–203).

Avulsion of teeth occurs most often in children from 7 to 9 years of age, when the permanent incisors are erupting (6–8, 199–203) (Fig. 20.1). At this age, the loosely structured periodontal ligament and low mineralized bone surrounding erupting teeth provide only minimal resistance to an extrusive force.

Most frequently, avulsion involves a single tooth; but multiple avulsions are occasionally encountered (6–8, 199–202). Other types of injuries are often associated with avulsions; among these, fractures of the alveolar socket wall and injuries to the lips are the most common (1). The level of the attachment apparatus of the avulse tooth is essential when considering whether or not to replant the tooth. The presence of a ring of calculus or discoloration of the root surface is a good indicator (Fig. 20.2). The circumstances related to extraoral storage of the avulsed tooth should be noted such as extra-oral time and storage conditions; especially whether the tooth has been kept dry or under physiological conditions (Fig. 20.3). Also, the contamination of the root surface should be registered, as this might influence healing.

## Radiographic findings

In cases where the avulsed tooth has been found, radiographs should only be taken if the clinical examination arouses suspicion of bone fracture (Figs 20.4 and 20.5). Moreover, in cases where the "avulsed" tooth is not found there is an indication for radiographic examination since the tooth can be severely intruded and also a fractured root may be left in the alveolus, swallowed or inhaled.

## Healing and pathology

The pathology following *tooth replantation* can be divided into pulpal and periodontal reactions. Both the pulp and the periodontal ligament suffer extensive damage during an extra-alveolar period with healing reactions almost entirely dependent upon the storage media and length of extra-alveolar period and extra-alveolar handling (Fig. 20.6).

The healing reactions after replantation of teeth have been the subject of numerous experimental studies using mice (35, 315), hamsters (36–42), rats (167–169, 309, 312, 329), guinea pigs (170), rabbits (27), cats (204–208), dogs (9–29, 130–137, 209–212, 306, 327, 337) and monkeys (17, 24, 31–34, 138–166, 213–226).

### Pulpal reactions

Studies on *pulpal reactions* have mainly been performed in animals such as cats (204, 205), dogs (136, 137, 209–212) and monkeys (225, 226). Experimental studies have

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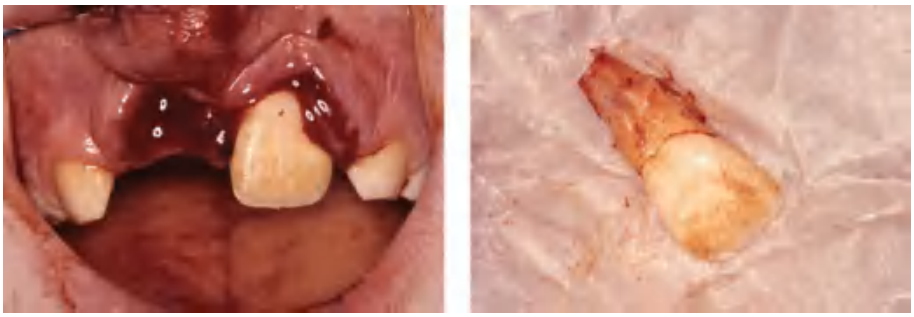


**Fig. 20.1** Avulsed central incisor in a 7-year-old boy. The tooth has been wrapped in paper for 60 minutes.

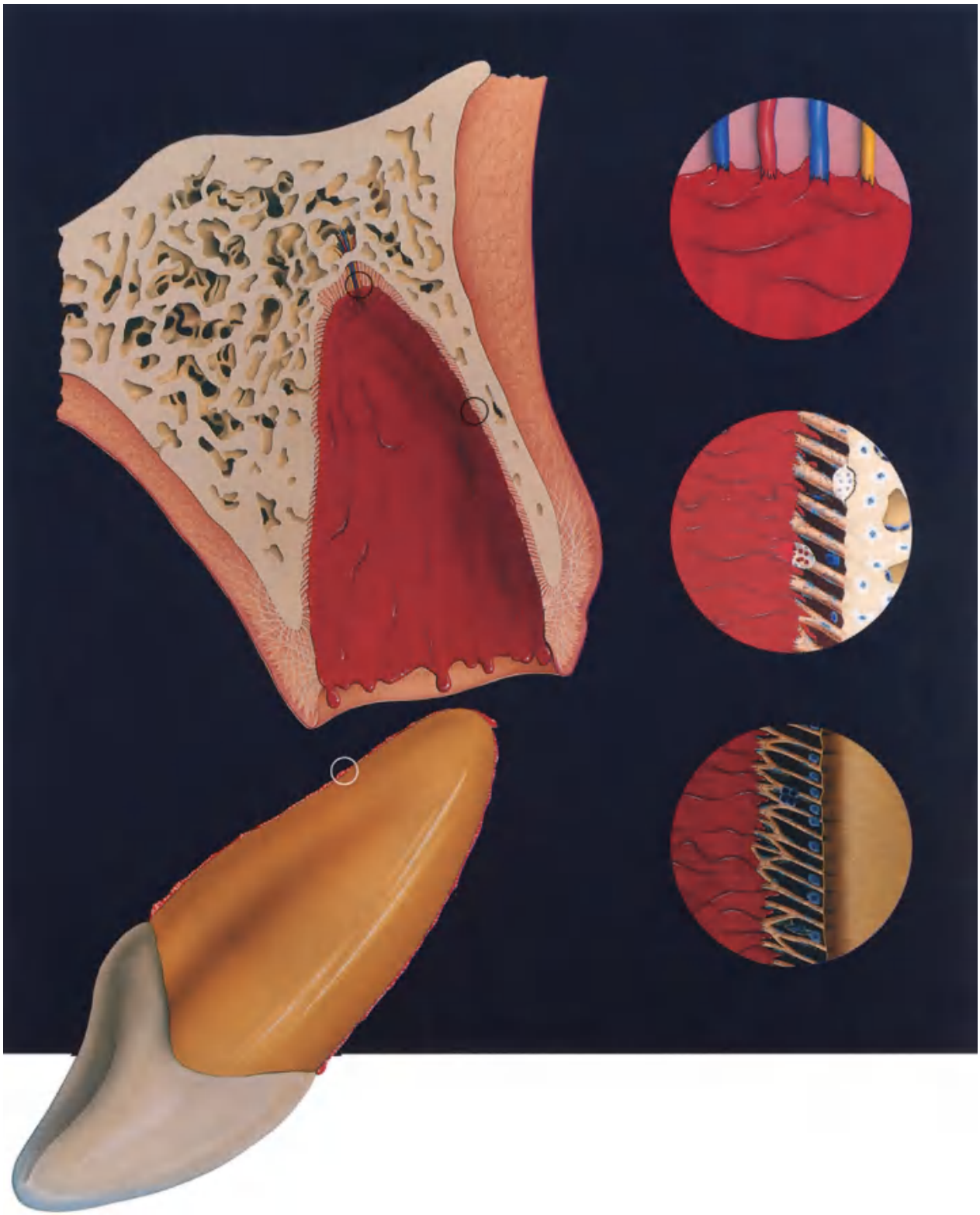


**Fig. 20.2** Mechanism of avulsion. Frontal impacts lead to avulsion with subsequent damage to both the pulp and periodontal ligament. The extra-oral time and environment determines the fate of the PDL and pulp after replantation.

**Fig. 20.3** A ring of calculus on the root surface indicates the level of PDL attachment.

**Fig. 20.4** An avulsed central incisor is kept moist in saliva in the buccal vestibule.

**Fig. 20.5** Contusion and fracture of the socket of two central incisors.

disclosed various distinct pulpo-dentinal responses which can occur after immediate replantation and have been classified as follows (29) (Fig. 20.7):

(1) Regular tubular reparative dentin.
(2) Irregular reparative dentin with diminished tubular structures.
(3) Irregular reparative dentin with encapsulated cells (osteodentin).
(4) Irregular immature bone.
(5) Regular lamellated bone or cementum.
(6) Internal resorption.
(7) Pulp necrosis.

It is uncertain whether all of these reactions are encountered in humans; however, pulpal reactions observed in human patients after intentional replantation seem to support at least part of this classification.

Several comprehensive studies of pulp reactions of extracted and immediately replanted permanent premolars in *human* patients have been published (43, 227–229). Extensive pulpal changes could be observed as early as 3 days after replantation. The most severe damage was usually observed in the coronal part of the pulp. Signs of healing were seen within 2 weeks after replantation. Damaged coronal pulp tissue was gradually replaced by proliferating mesenchymal cells and capillaries (Figs 20.8 and 20.9). In the

border zone between vital and necrotic tissue, neutrophils and round cells were present in some cases.

In the majority of cases with long observation periods, more advanced healing was found. This healing process led to the formation of a new cell layer along the dentinal wall in regions where the odontoblasts had been destroyed. The mesenchymal cells along the dentinal wall usually did not have processes extending into the dentinal tubules (Fig. 20.9). New hard tissue formation along the dentinal walls was noted after 17 days; but in most cases matrix formation started somewhat later. In the early stages of healing, a tissue was formed without dentinal tubules but with occasional cell inclusions (Fig. 20.10). Gradually, the cells along the pulp walls began to show similarities to odontoblasts with cytoplasmic processes within the newly formed matrix. This apparently corresponded to the degree of differentiation; however, in areas where new hard tissue formation indicated total primary destruction of the original odontoblasts, completely normal conditions were never found.

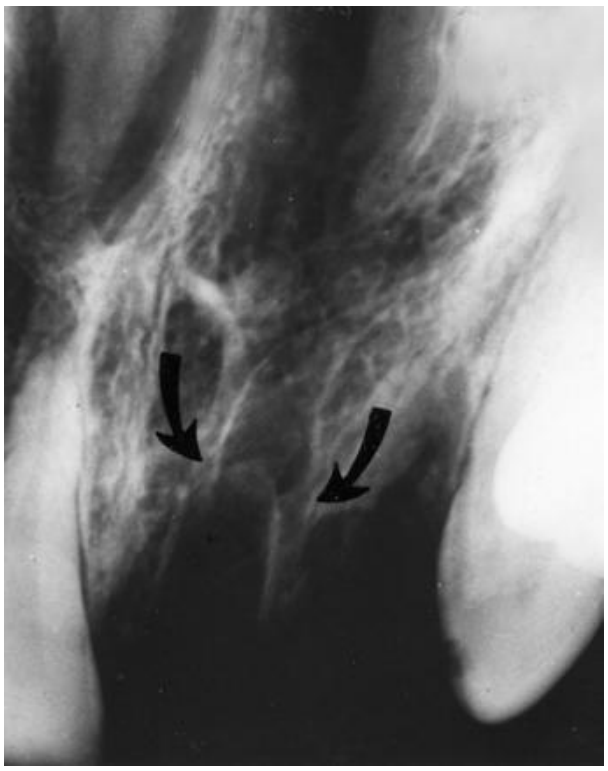


**Fig. 20.6** Radiograph illustrating an alveolar fracture (arrows) following avulsion of both central incisors.

Severe primary pulpal damage was more often found in teeth with completed root formation than in those with an open apex, where pulpal repair also seemed to be more rapid. Mitoses were seen in bands of Schwann cells 14 days after replantation (Fig. 20.10). Regenerating nerve fibers were observed after 1 month. In teeth with irregular hard tissue formation in the pulp chamber, bundles of nerve fibers passed between the trabeculae of hard tissue; separate fibers could be followed to the newly formed layer of irregular odontoblasts. However, neither the number nor the caliber of the nerve fibers reached normal levels. In microangiographic studies of the revascularization process after replantation of teeth in dogs, it was demonstrated that ingrowth of new vessels could be seen 4 days after replantation. After 10 days, vessels were seen in the apical half of the pulp and, after 30 days, in the entire pulp (136, 137).

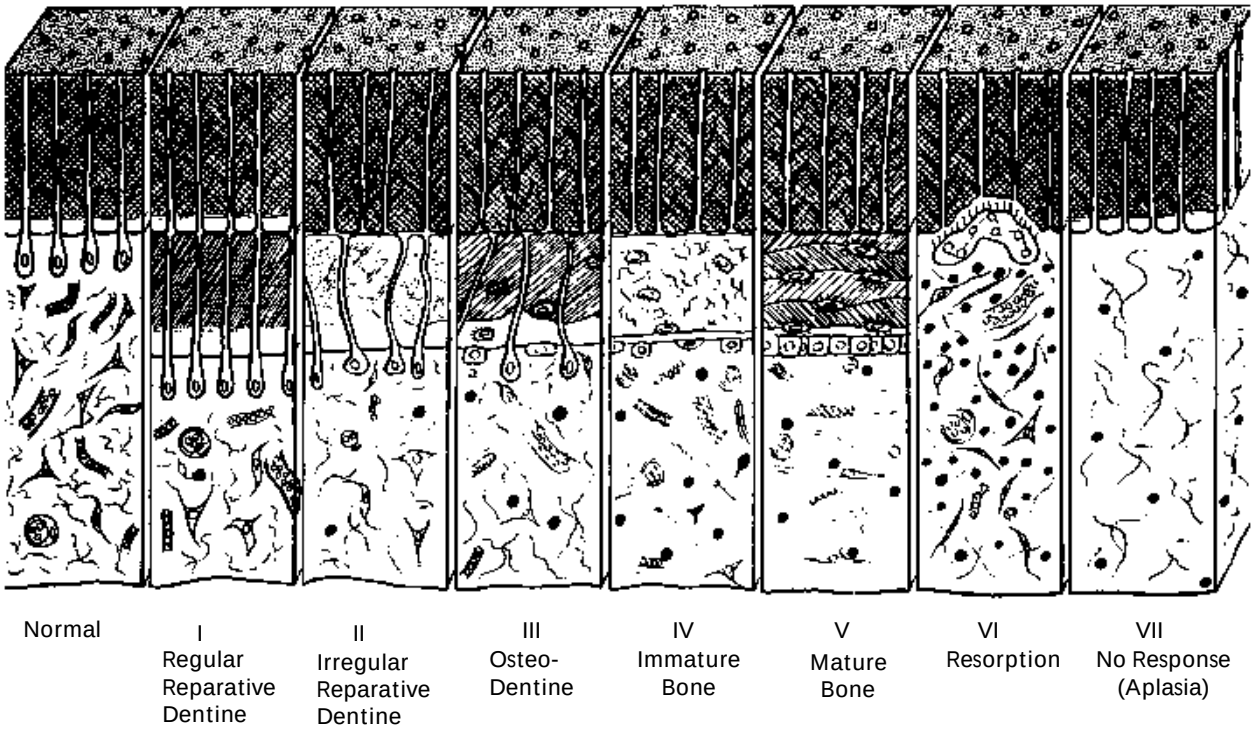



**Fig. 20.7** Schematic drawing illustrating the various pulp reactions following experimental replantation of incisors in dogs. Reproduced from (29) with permission.

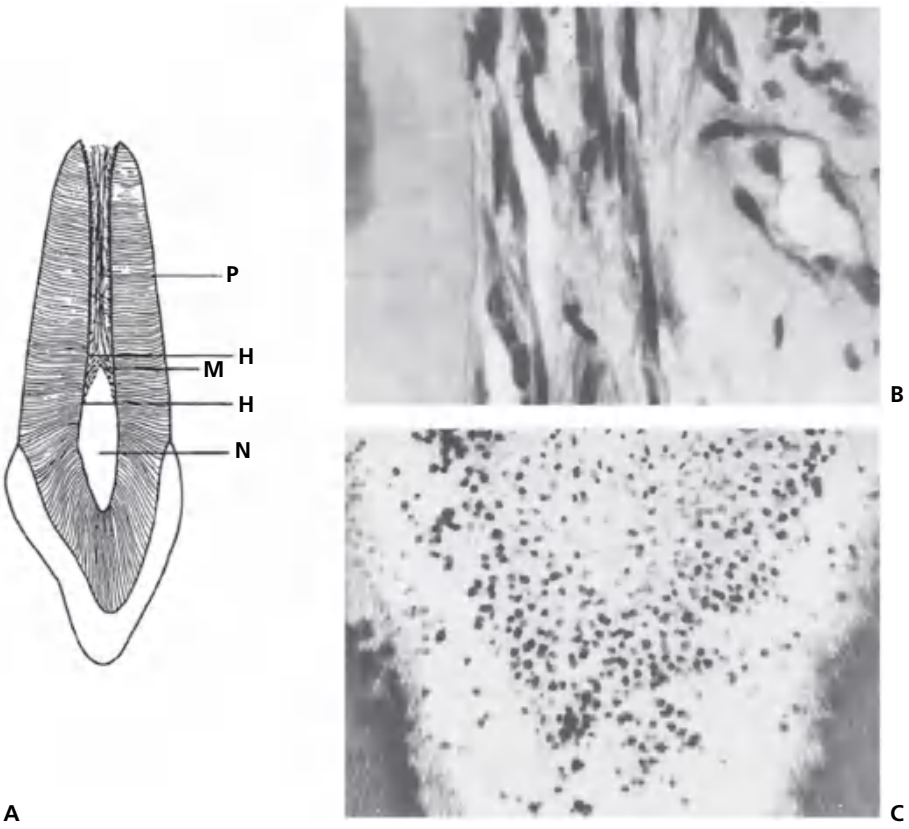



**Fig. 20.8** A. Diagram illustrating initial pulp changes after replantation. N = necrotic pulp tissue, M = mesenchymal cells, H = healing zone, P = preserved vital pulp tissue. B. Proliferating mesenchymal cells along the root canal wall at the level of necrotic and vital pulp tissue 10 days after replantation. × 410. C. Karyorrhexis and pyknosis in the coronal pan of the pulp 3 days after replantation. × 200. Reproduced from (43) with permission.

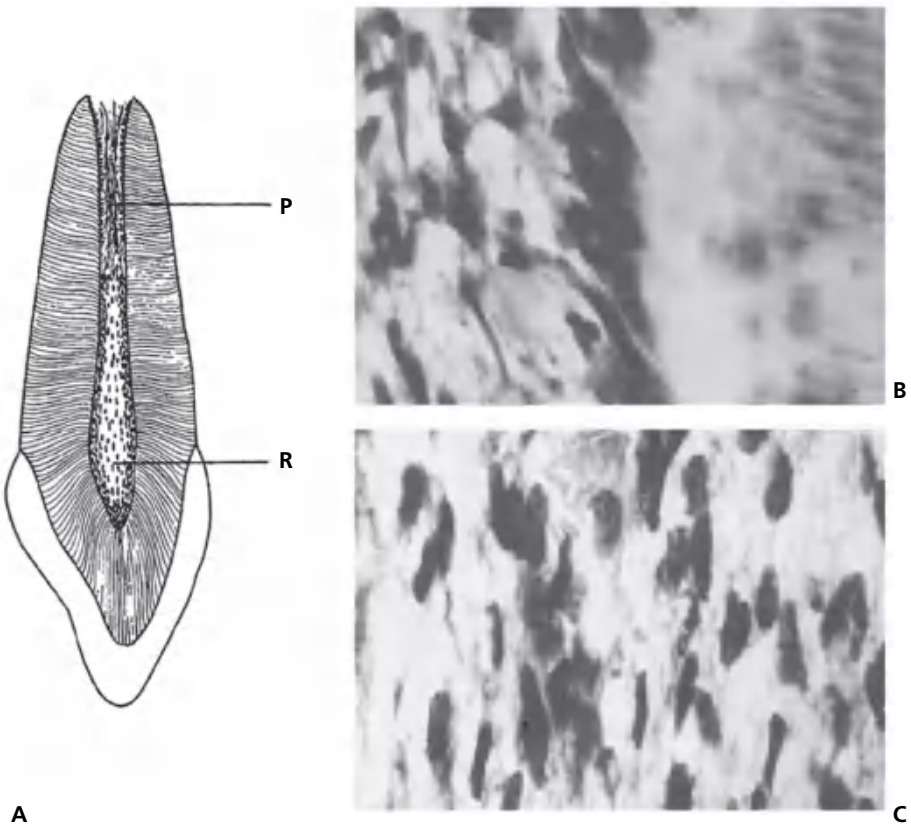



**Fig. 20.9** A. Diagram illustrating intermediate pulp changes after replantation. New mesenchymal cells proliferate into the injured zones during the first months of the healing period. R = regenerated pulp tissue, P = preserved vital pulp tissue. B. The cells adjacent to the predentin in the apical part of the pulp appear to send no processes into the dentinal tubules (observation period = 17 days). × 430. C. Regenerated pulp cells are aligned nearly parallel with the long axis of the tooth. × 520. Reproduced from (43) with permission.

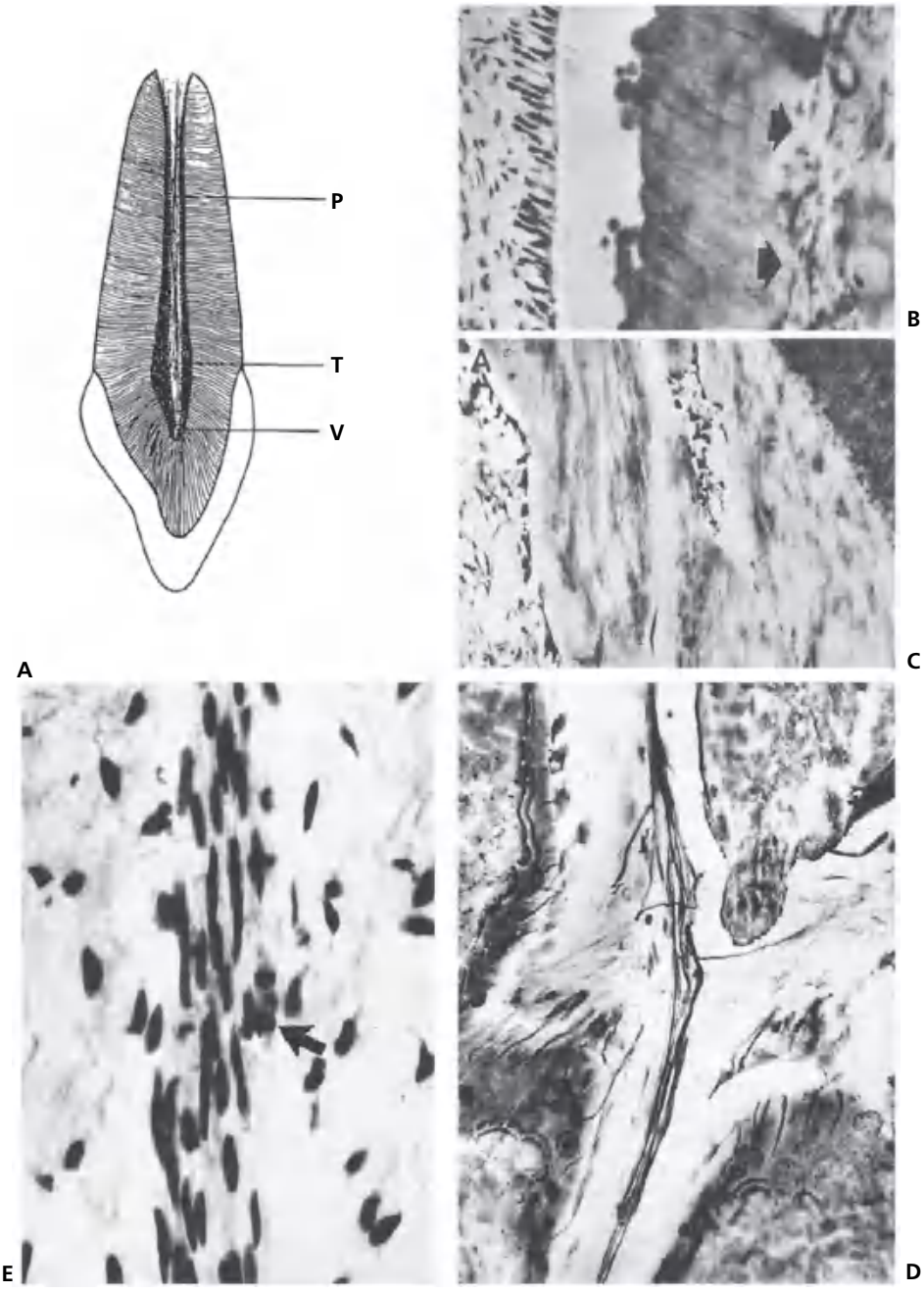



**Fig. 20.10** A. Diagram illustrating late pulp changes after replantation. Extensive obliteration of the pulp chamber with hard tissue and strands of vital pulp tissue extending into the hard tissue. V = vital regenerated pulp tissue, T = newly formed hard tissue, P = preserved vital pulp tissue. B. Two phases of repair. First, irregular atubular matrix (arrows), second atubular dentin (observation period = 101 days). × 180. C. Irregular new hard tissue formation in an area with severe pulp damage (observation period = 158 days). × 125. D. Nerve fibers coursing through newly formed hard tissue in the pulp chamber (observation period = 360 days). Palmgren's silver stain. × 275. E. Nerve fibers with mitosis of Schwann cells (arrow) (observation period = 14 days). × 450. Reproduced from (43) with permission.

## Periodontal healing reactions

A series of avulsed human teeth, analyzed by histology, showed that almost half of the periodontal ligament was lost, indicating that serious healing complications may occur in the form of root resorption (317) (see Chapter 2). The following healing sequence in the periodontal structures has been demonstrated after experimental immediate replantation in dogs (24, 306), monkeys (138, 162), and humans (230).

Immediately after replantation, a coagulum is found between the two parts of the severed periodontal ligament (Fig. 20.11A). The line of separation is most often situated in the middle of the periodontal ligament, although separation can occur at the insertion of Sharpey's fibers into cementum or alveolar bone. Proliferation of connective tissue cells soon occurs and, after 3 to 4 days, the gap in the periodontal ligament is obliterated by immature connective tissue (Fig. 20.11B). After 1 week, the epithelium is reattached at the cementoenamel junction (138). This is of clinical importance because it may imply a reduced risk of gingival infection and/or reduced risk of bacterial invasion of either the root canal or periodontal ligament via the gingival pocket. Gingival collagen fibers are usually spliced, while the infrabony fibers are united in only a few areas at this time (138). The first superficial osteoclast attack can now be seen along the root surface (140).

After 2 weeks, the split line in the periodontal ligament is healed and collagen fibers are seen extending from the

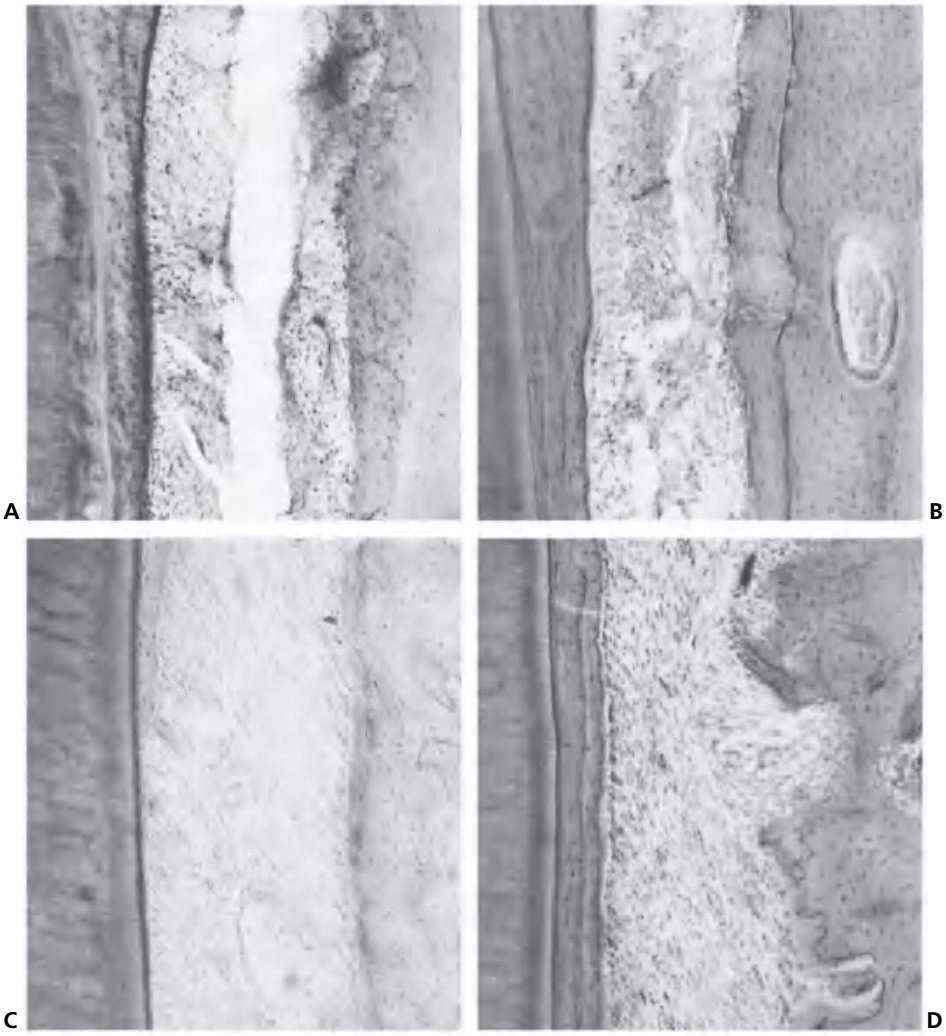



**Fig. 20.11** Healing sequence in the periodontal structures following experimental tooth replantations in dogs. A. Condition immediately after re plantation. The line of separation is situated in the middle of the periodontal ligament. × 75. B. Three days later proliferating connective tissue cells invade the coagulum of the severed periodontal ligament. × 75. C. Two weeks later new collagenous fibers have bridged the periodontal space. × 75. D. Normal periodontal conditions 8 months after replantation. × 75.

cementum surface to the alveolar bone (Fig. 20.11C). Resorption activity can now be recognized along the root surface (140).

Histologic examination of replanted human and animal teeth has revealed four different healing modalities in the periodontal ligament (44, 45, 147, 221, 222, 231).

### *Healing with a normal periodontal ligament*

*Histologically*, this is characterized by complete regeneration of the periodontal ligament (10, 24, 36, 44, 47, 48), which usually takes about 4 weeks to complete including the nerve supply (233, 306) (Fig. 20.12). This type of healing will only occur if the innermost cell layers along the root surface are vital (142, 215).

*Radiographically*, there is a normal periodontal ligament space without signs of root resorption (Fig. 20.13).

*Clinically*, the tooth is in a normal position with normal mobility and a normal percussion tone can be elicited.

This type of healing will probably never take place under clinical conditions (i.e. after tooth avulsion), as trauma will result in at least minimal injury to the innermost layer of the periodontal ligament and lead to surface resorption (see Chapter 2).

### *Healing with repair-related resorption (surface resorption)*

*Histologically*, this type of healing is characterized by localized areas along the root surface which show superficial resorption lacunae repaired by new cementum. This condition has initially been termed surface resorption, presumably representing localized areas of damage to the periodontal ligament or cementum which have been healed by periodontal ligament-derived cells (44, 140, 142, 146, 222, 231). In contrast to other types of resorption, surface resorption is not progressive, i.e. self-limiting and shows repair with new cementum (Fig. 20.14). Most resorption lacunae are superficial and confined to the cementum. In cases of deeper resorption cavities, however, healing occurs, but without restoration of the original outline of the root. It should be noted that resorption lacunae with similar morphology and location have been reported on non-traumatized root surfaces with a frequency as high as 90% of all teeth examined (49).

Due to their small size, surface resorptions are usually not disclosed *radiographically*. However, with ideal angulation of the central beam it is sometimes possible to recognize small excavations of the root surface with an adjacent periodontal ligament space of normal width (Fig. 20.15).

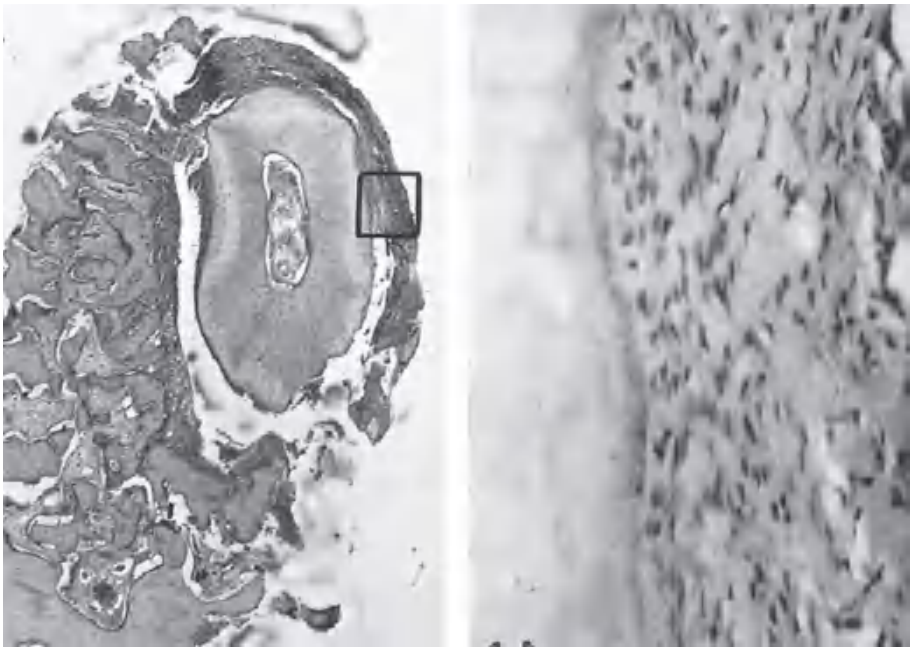



**Fig. 20.12** Healing with a normal periodontal ligament following replantation of a left lateral incisor. A. Apex removed 3112 months after replantation. B. Normal periodontal ligament and cementum. × 12 and × 195. Reproduced from (44) with permission.

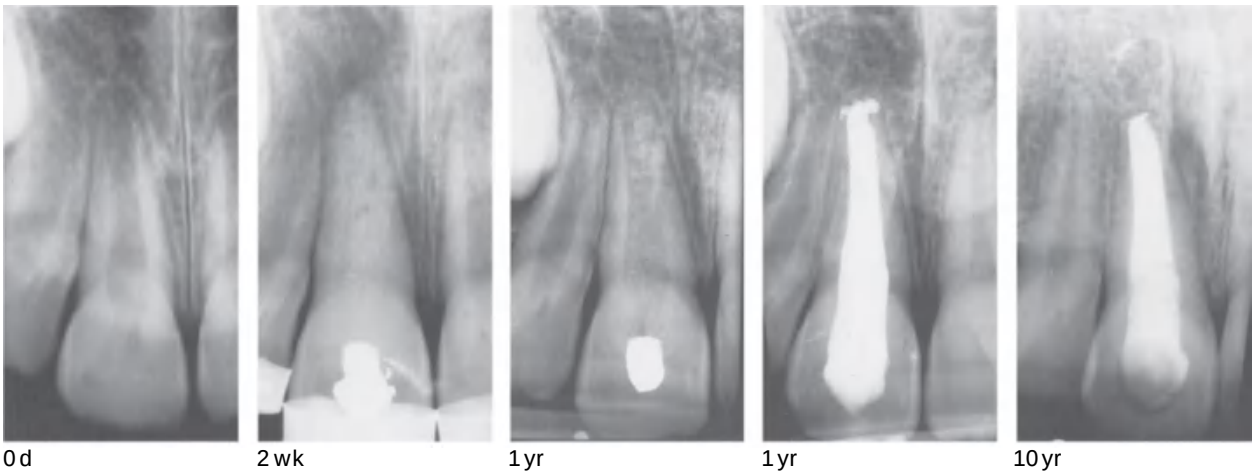



**Fig. 20.13** Periodontal ligament healing after replantation of right central incisor. Reproduced from (202) with permission.

*Clinically*, the tooth is in a normal position and a normal percussion tone can be elicited.

*Pulpal status,* the pulp is usually vital.

### *Healing with ankylosis-related resorption (replacement resorption)*

*Histologically*, ankylosis represents a fusion of the alveolar bone and the root surface and can be demonstrated 2 weeks after replantation (138). The etiology of replacement resorption appears to be related to the absence of a vital periodontal ligament cover on the root surface (141–143, 216, 221, 231, 234). Ankylosis develops in two different directions, depending upon the extent of damage to the periodontal ligament cover of the root: either *progressive* resorption, which gradually resorbs the entire root, or *transient* resorption, in which a once-established ankylosis later disappears (144, 171) (Figs 20.16 and 20.17). Progressive ankylosis is always the result when the entire periodontal ligament is removed before replantation (143) or after extensive drying of the tooth before replantation (143, 146). It is assumed that the damaged periodontal ligament is repopulated from adjacent bone marrow cells, which have osteogenic potential and will consequently form an ankylosis (172). Transient ankylosis is possibly related to areas of minor damage to the root surface. In these cases, the ankylosis is formed initially and later resorbed by adjacent areas of vital periodontal ligament. This theory is supported experimentally by research in which the effect of limited drying or limited removal of the periodontal ligament upon periodontal healing after replantation was studied (144).

Figure 20.18 demonstrates an initial phase of ankylosis-related resorption. The ankylosed root becomes part of the normal bone remodeling system and is gradually replaced by

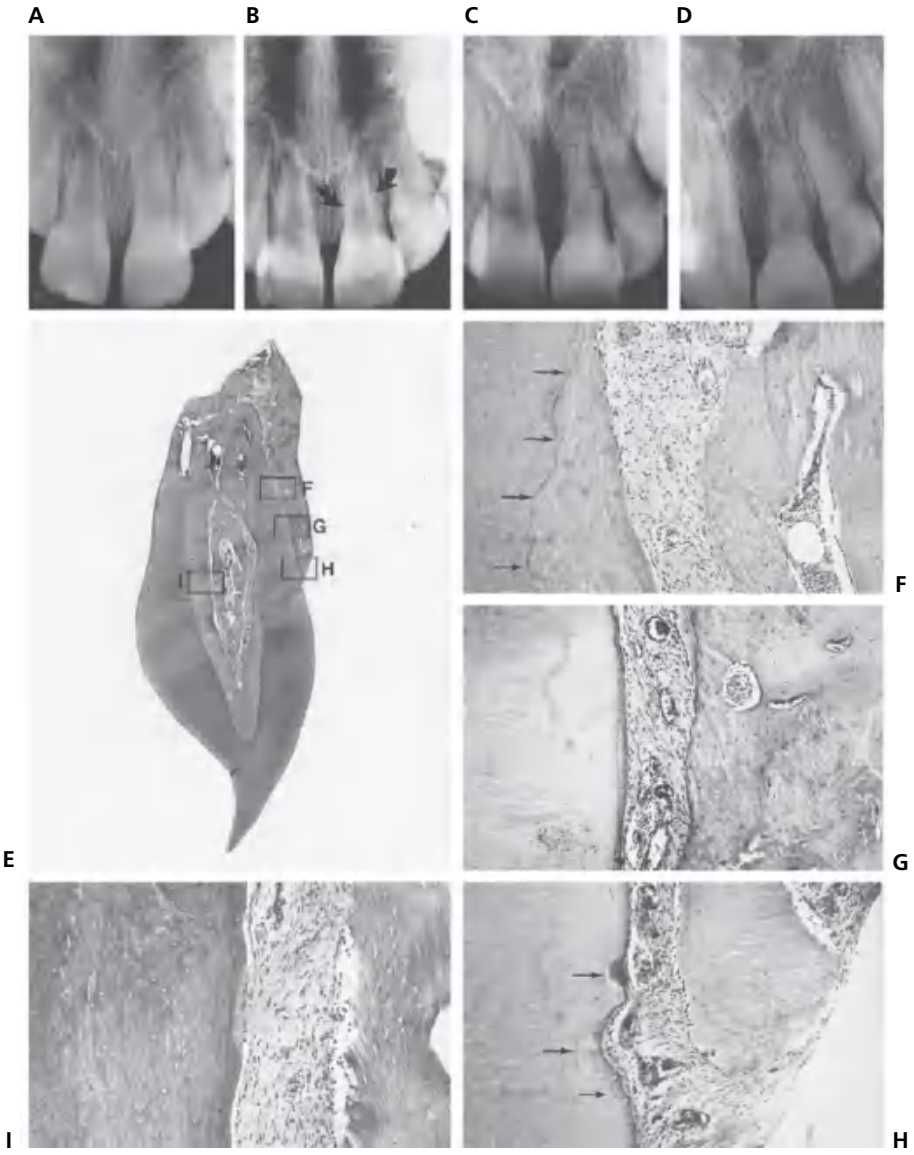



**Fig. 20.14** Healing with a normal periodontal ligament and marked repair related resorption after replantation of a left central incisor. Extra-alveolar period 95 minutes. A. Immediately after replantation. B. Five months later. Repair related resorption of the root is evident (arrows). C. and D. Condition 2 and 4 years later. No progression of repair related resorption has occurred whereas the pulp canal has become obliterated. For orthodontic reasons the tooth was later extracted. E. Low power view of sectioned incisor. × 4. F. Large repair related resorption defect repaired with new cementum (arrows). The periodontal ligament is normally structured. × 75. G. Area without repair related resorption. × 75. H. Minor repair related resorption areas repaired with cementum. × 75. L Bone formation in the central part of the pulp. An internal periodontal ligament connects this structure with the hard tissue deposited on the canal walls. × 75.

**Fig. 20.15** Repair related resorption (arrow) of a replanted right central incisor. Note the superficial appearance on the root and sparse involvement on the lamina dura. The resorption cavity is stationary during the entire observation period. Reproduced from (202) with permission.

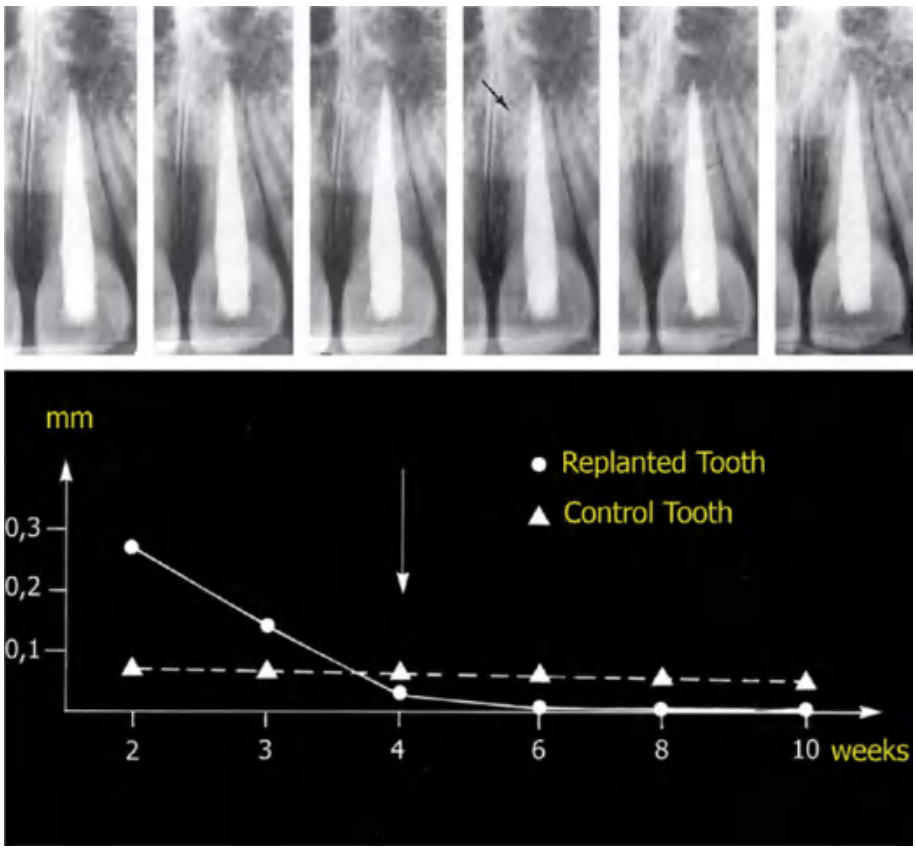



**Fig. 20.16** Permanent ankylosis related resorption. Mobility values indicated that an ankylosis was established 4 weeks after replantation. Ankylosis related resorption was first radiographically demonstrable 6 weeks after replantation (arrow). Reproduced from (171) with permission.

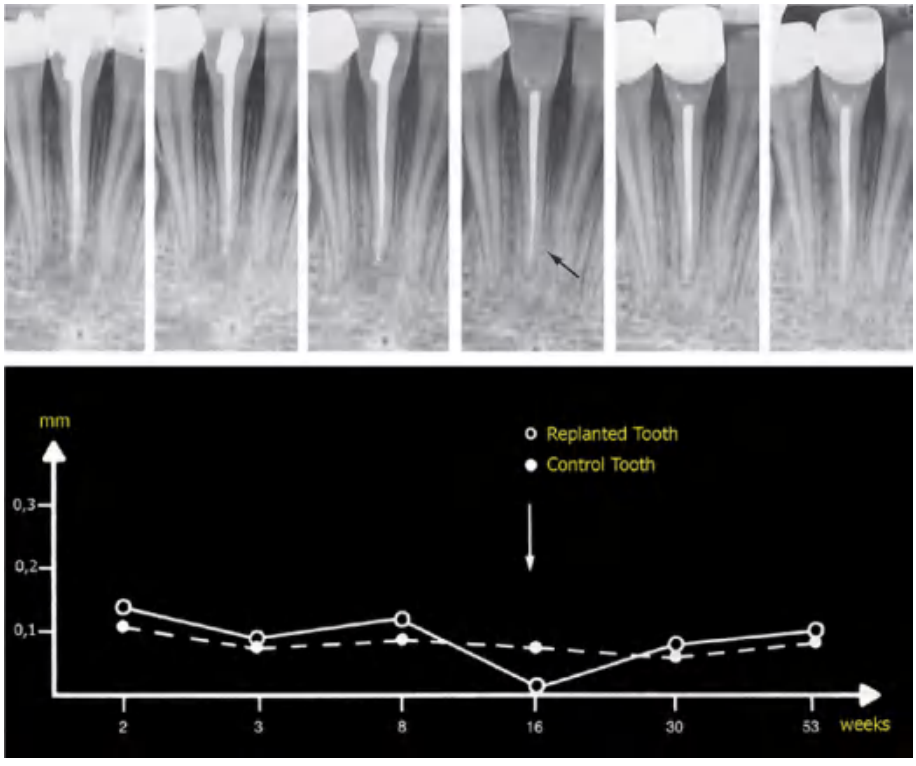



**Fig. 20.17** Transient ankylosis related resorption. Mobility testing indicated an ankylosis 16 weeks after replantation. Ankylosis also demonstrable radiographically (arrow). A normal periodontal space was restored at later controls. Reproduced from (171) with permission.

bone. After some time, little of the tooth substance remains (Fig. 20.19). At this stage, the resorptive processes are usually intensified along the surface of the root canal filling, a phenomenon known as internal tunneling resorption (Fig. 20.19). (See also Chapter 16.)

*Radiographically*, ankylosis is characterized by loss of the normal periodontal space and continuous replacement of root substance with bone (Fig. 20.20, A to D).

Ankylosis can usually first be recognized radiographically 2 months after replantation; however, in most cases

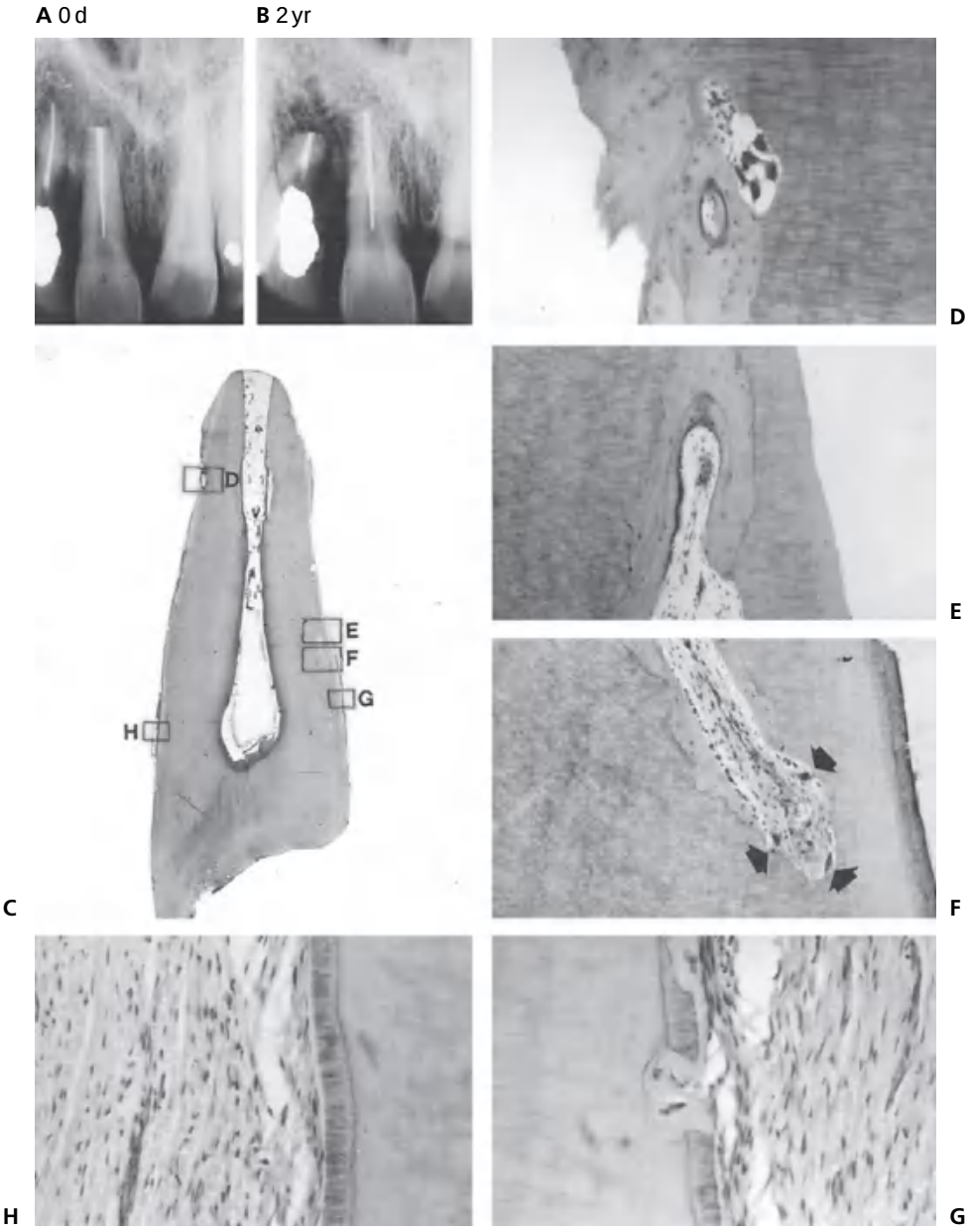



**Fig. 20.18** Ankylosis (ankylosis related resorption) after replantation of a right central incisor. A. Radiograph taken immediately after replantation. B. Condition at the time of extraction 2 years after injury. C. Low-power view of sectioned incisor. × 5. D. Bony union between root surface and surrounding alveolar bone. Note resorption lacunae with osteoclasts. × 75. E. and F Ankylosis related resorption with apposition of bone and active resorption (arrows). × 75. G. Repair related resorption repaired with cellular cementum. × 195. H. Area with normal periodontal ligament. × 195. Reproduced from (44) with permission.

ankyloses is diagnosed radiographically 6 months or up to 1 year after the replantation (202) (Fig. 20.21).

Most cases can be diagnosed within 2 years; but it has been found that up to10 years may elapse before radiographic diagnosis can be made (202) (Fig. 20.22). This problem of diagnosing ankylosis has also been verified in experimental studies, where histologic findings were compared with radiography (235, 236).

*Clinically*, the ankylosed tooth is immobile and in growing individuals usually in infraposition. The percussion tone is high, differing distinctly from adjacent non-injured teeth. The percussion test can often reveal replacement resorption in its initial phases before it can be diagnosed radiographically (171, 202, 235).

Cases of transient ankylosis can sometimes be demonstrated radiographically as small areas where the periodontal ligament space has disappeared (Fig. 20.17). Most often this type of ankylosis is demonstrated by its high percussion tone. Disappearance of the ankylosis, which always happens within the first year, is followed by the return of a normal percussion tone (171).

## Infection-related resorption (healing with inflammatory resorption)

*Histologically*, inflammatory resorption is characterized by bowl-shaped resorption cavities in cementum and dentin associated with inflammatory changes in the adjacent

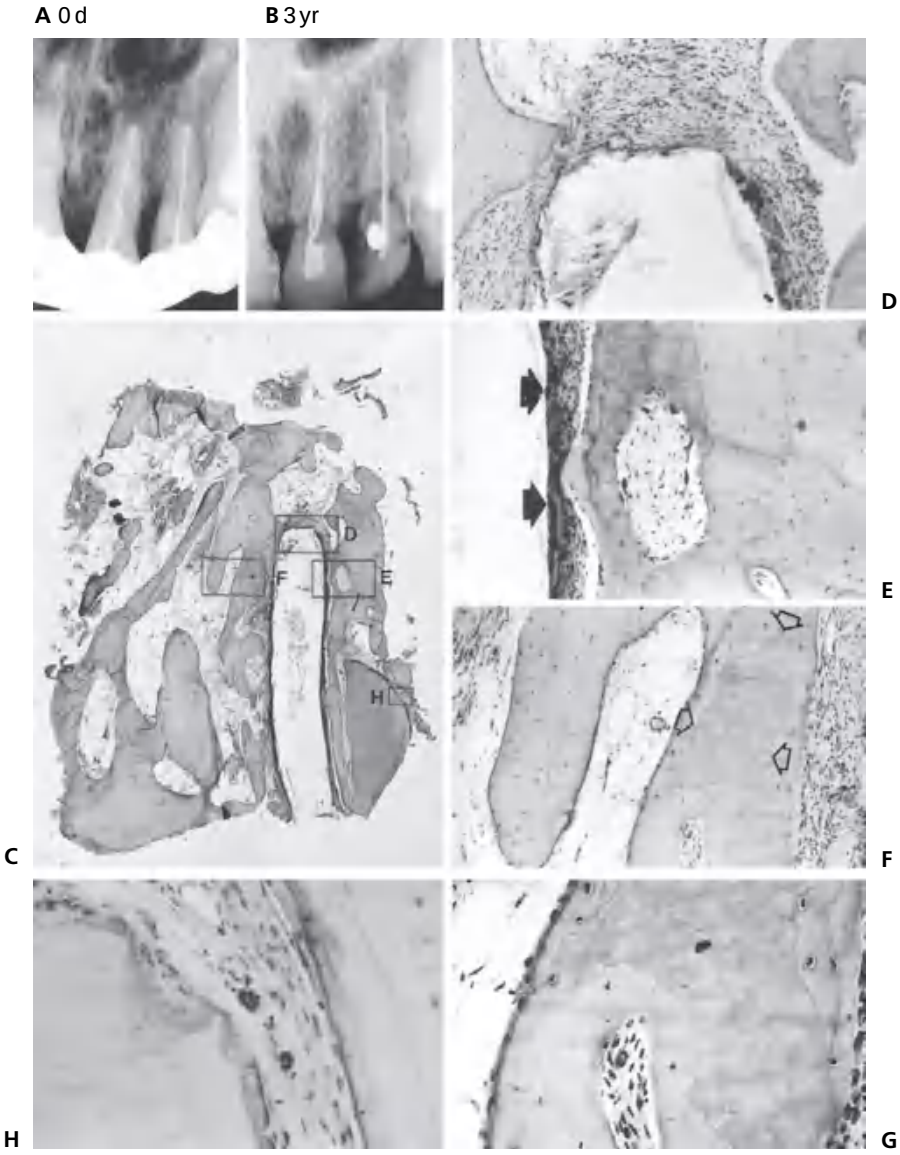



**Fig. 20.19** Advanced ankylosis (ankylosis related resorption) after replantation of upper left central and lateral incisors. A. Radiograph immediately after replantation. B. Condition at time of removal of the central incisor 3 years after replantation. C. Low power view of sectioned incisor. × 12. D. Apical part of root canal filling surrounded by bone and connective tissue. × 30. E. Haversian canal under remodeling. The walls consist of both dentin and bone. Note tunneling resorption along the root canal filling (arrows). × 30. F. Fragment of dentin incorporated in bone (arrows). × 30. G. Higher magnification of F. × 195. H. Area with normal cementum and periodontal tissue including epithelial rests of Mallassez. × 195. Reproduced from (44) with permission.

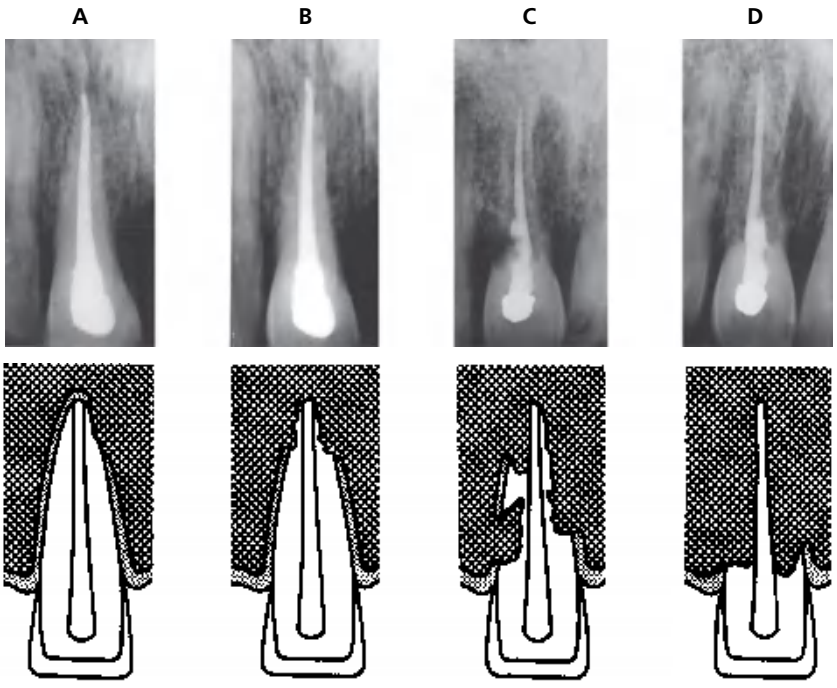



**Fig. 20.20** Schematic and radiographic appearance of ankylosis and infection related resorption. A to D. Progression of ankylosis related resorption. E to H. Progression of infection related resorption. Reproduced from (6) with permission.

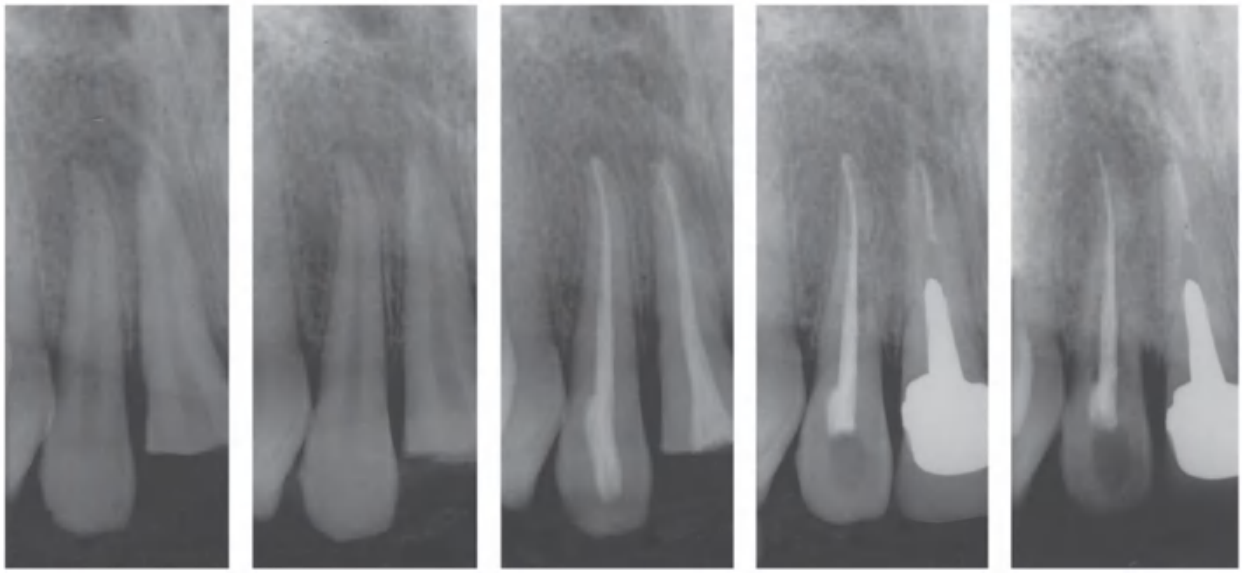



**Fig. 20.21** Progression of ankylosis related resorption is followed by tunneling infection related resorption along the root filling. Reproduced from (202) with permission.

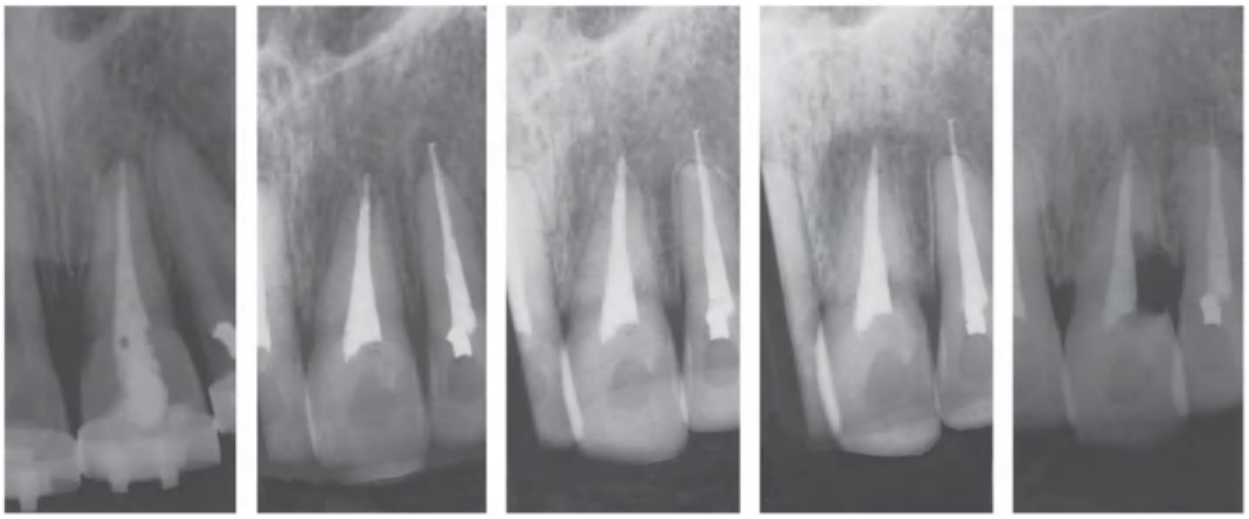



**Fig. 20.22** Late appearance of ankylosis. Resorption activity is suspected after 3 and 5 years and becomes manifest after 10 years. Reproduced from (202) with permission.

periodontal tissue and bone (44, 53–57, 139, 147, 237) (Fig. 20.23). The inflammatory reaction in the periodontium consists of granulation tissue with numerous lymphocytes, plasma cells, and polymorphonuclear leukocytes. Adjacent to these areas, the root surface undergoes intense resorption with numerous Howship's lacunae and osteoclasts.

The pathogenesis of infection-related resorption can be described as follows (139): Minor injuries to the periodontal ligament and/or cementum due to trauma or contamination with bacteria induce small resorption cavities on the root surface, presumably in the same manner as in repair-related resorption. If these resorption cavities expose dentinal tubules and the root canal contains infected necrotic tissue, toxins from these areas will penetrate along the dentinal tubules to the lateral periodontal tissues and provoke an inflammatory response. This in turn will intensify the resorption process which advances towards the root canal (44, 139). The resorption process can progress very rapidly, i.e. within a few months the entire root can be resorbed.

Infection-related resorption is especially frequent and aggressive after replantation in patients from 6 to 10 years of age. The explanation for this is probably a combination of wide dentinal tubules and/or a thin protective cementum cover (6, 139). In older age groups, the resorption process follows a more protracted course.

It should be noted that replanted teeth can show simultaneous infected and ankylosis-related resorption (Fig. 20.24). Moreover, if the resorption process is allowed to progress

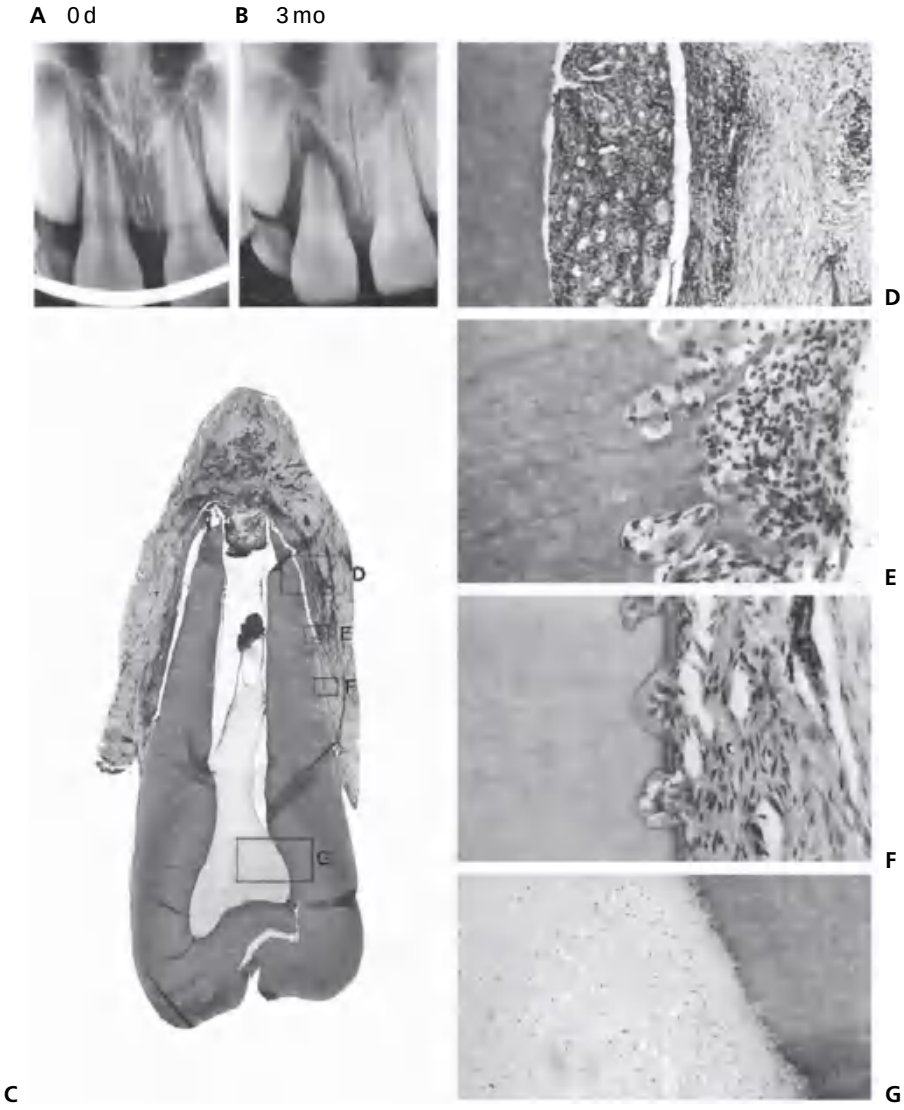



**Fig. 20.23** Infection related resorption after replantation of a right central incisor. A. Radiograph taken immediately after replantation. B. Condition at time of removal 3 months later. C. Low power view of sectioned incisor. The pulp shows complete necrosis with autolysis. An intense inflammatory reaction is seen around the apical half of the root. × 7. D. Granulation tissue in relation to resorbed root surface. × 30. E. Area with active resorption. × 195. F. Repair related resorption partly repaired with new cementum. × 195. G. Necrotic pulp tissue. × 30. Reproduced from (44) with permission.

and involve large areas of the root surface, *ankylosis resorption* can take over once infection-related resorption has been arrested by endodontic therapy.

*Radiographically*, infection-related resorption is characterized by radiolucent bowl shaped cavitations along the root surface with corresponding excavations in the adjacent bone (Fig. 20.25). The first radiographic sign of infection-related resorption can be demonstrated as early as 2 weeks after replantation and is usually first recognized at the cervical third of the root (6) (Fig. 20.25). As in the case of ankylosis, this resorption type is usually evident within the first 2 years after replantation.

*Clinically*, the replanted tooth is loose and extruded. Moreover, the tooth is sensitive to percussion and the percussion tone is dull (vs. ankylosis).

## Treatment of the avulsed tooth

This issue has been the topic for several committees' attempts to create evidence-based guidelines. With respect to avulsed teeth, this task is very difficult due to the paucity of clinically documented studies. The reader is referred to two published reviews by Barrett & Kenny (1997) (279) and Trope (1995) (304). The present guidelines are primarily based on an analysis of a series of experimental studies and a few clinical studies where enough data support the given procedures.

### At the site of accident

In some instances, a telephone call at the time of injury will precede the office visit. In these cases, the patient or an available adult should be instructed to replant the tooth immediately. In this way, the extra-alveolar period is decreased, and the prognosis improved significantly. Replantation can be accomplished in the following manner.

If dirty, the tooth can be cleaned by simply rinsing it under running, cold tap water for 10 seconds and placing it immediately in its socket. This procedure has been found to decrease the extent of root resorption experimentally (280). If the replantation procedure cannot be performed at this time, the tooth can be stored in the patient's buccal vestibule. Animal experiments have shown that storage in milk or

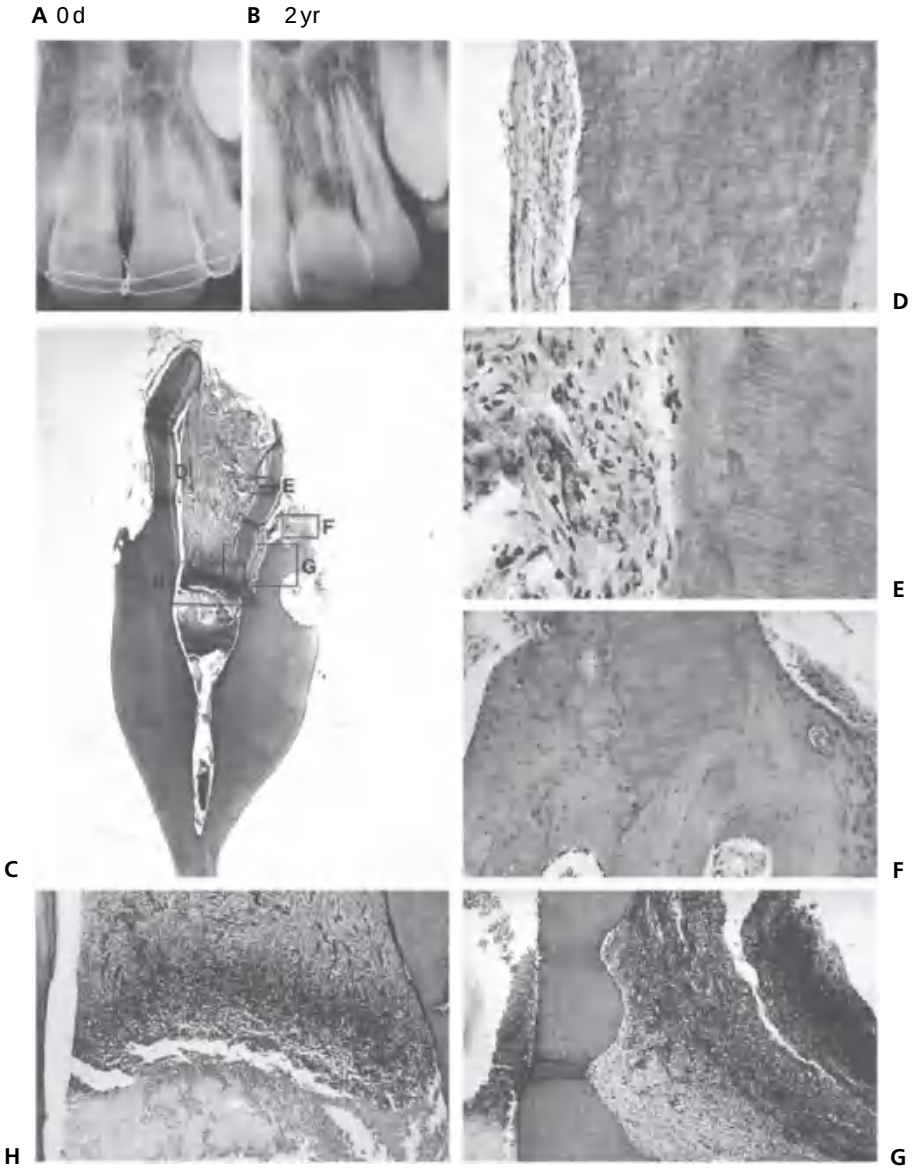



**Fig. 20.24** Ankylosis and infection related resorption after replantation of a left central incisor. A. Radiograph taken immediately after replantation. B. Condition at time of removal 2 years later. C. Low-power view of sectioned incisor. Note partial pulp necrosis and root resorption. × 5. D. Arrested external root resorption. × 195. E. Acellular hard tissue (presumably cementum) deposited on the canal walls. × 195. F. Area with ankylosis related resorption. × 75. G. Area with active infection related resorption. × 30. H. Border zone between vital and necrotic pulp tissue. × 30. Reproduced from (44) with permission.

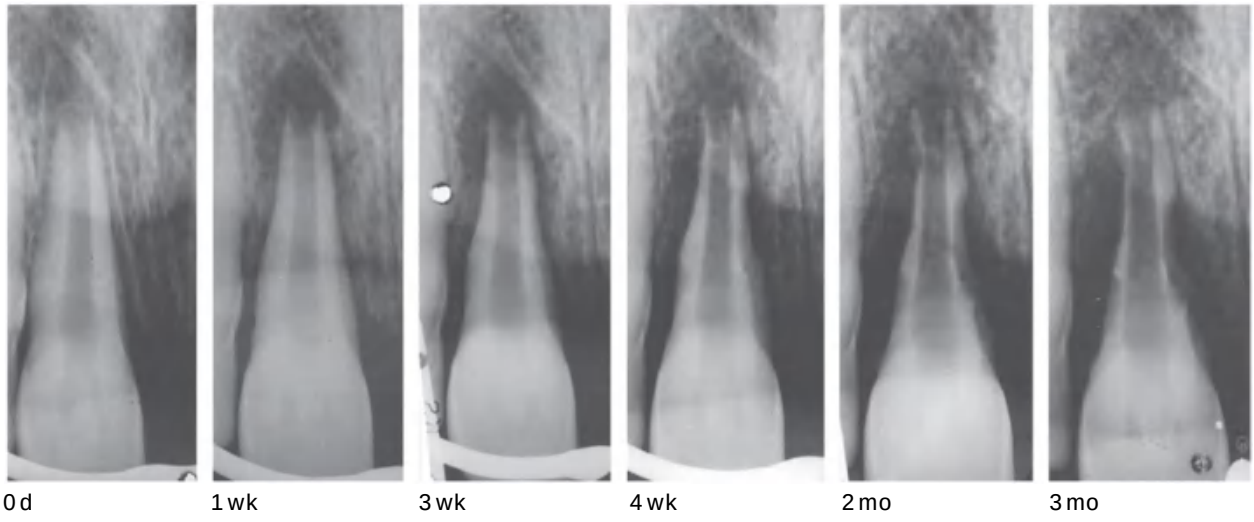



**Fig. 20.25** Infection related resorption of a replanted right central incisor. Note the excavating nature of the resorption process on the root and a corresponding marked resorption of the lamina dura. Reproduced from (6) with permission.

saliva has almost the same effect as storage in saline. However, long-term storage in tap water (i.e. more than 20 minutes) has an adverse effect on periodontal healing (146, 202). After replantation and en route to the dental office, the patient should be instructed to keep the tooth in place with either finger pressure or by biting on a handkerchief.

Avulsed teeth should always be replanted as soon as possible and if this is not possible a storage medium such as saliva, saline or milk should be used to store the replanted tooth until the dentist is visited. The patient should visit a dental emergency service as soon as possible. A detailed review of possible storage medias has recently been published (324).

In this regard a commercial tissue culture medium (Viaspan®) could be used for extraoral storage. *In vitro* experiments have shown it to be an effective storage medium even for several days (238, 324). In conditioned media it has been shown experimentally that teeth can be kept in storage for up to 96 hours and still lead to optimal healing (308). Another medium that has shown good effect is propolis® (280) However, *in vivo* studies are still lacking that demonstrate its effectiveness. Unfortunately, commercial storage media are seldom available at the site of accident.

## At the clinic

The case history should include exact information on the time interval between injury and replantation as well as the conditions under which the tooth has been stored (e.g. saline, saliva, milk, tap water, or dry).

If replantation is performed in a clinical environment, the avulsed tooth is examined for obvious contamination. The alveolus is also examined. As prognosis is significantly related to the length of the extra-alveolar period, pre-treatment radiographs can serve to extend this period and should, therefore, be taken prior to treatment only if there is suspicion of comminution or fracture of the socket wall.

Careful planning is of the utmost importance for the success of replantation of avulsed teeth.

The following conditions should be considered before replanting a permanent tooth:

(1) The avulsed tooth should be without advanced periodontal disease.
(2) The alveolar socket should be reasonably intact in order to provide a seat for the avulsed tooth.
(3) The extra-alveolar period should be considered, i.e. dry extraoral periods exceeding 1 hour are usually associated with marked root resorption.

While certain conditions, such as those listed above, might appear to contraindicate replantation of an avulsed incisor, it should be borne in mind that the time of injury is usually not the time to make such decisions. Definitive treatment planning (e.g. orthodontic evaluation of a crowded dentition) can seldom be made in the heat of acute treatment. Furthermore, an extended extraoral period — even dry storage — is not an absolute contraindication to replantation, as the root surface can be treated chemically to protract the resorption process in mature teeth (see later). In most situations, replantation of the avulsed tooth, even with a dubious prognosis, can be performed and the tooth considered as a temporary restoration until such time as definitive treatment planning, often following specialist consultation, can be carried out.

If replantation is decided upon, the following procedures are recommended (Fig. 20.26). The tooth is placed in saline. If visibly contaminated, the root surface is rinsed with a stream of saline from a syringe until visible contaminants have been washed away — including around the apical foramen. The alveolus is also rinsed with a flow of saline to remove the contaminated coagulum. No effort should be made to sterilize or mechanically cleanse the root surface, as such procedures will damage or destroy vital periodontal tissue and cementum (202).

The socket is then examined. If there is evidence of fracture, the fractured bone should be repositioned by inserting an instrument in the socket and then modelling the bone. Local anesthesia is usually not necessary unless gingival lacerations require suturing, or the alveolar socket requires remodelling. The tooth is replanted using light digital pressure. It is important that only light pressure is used during the replantation procedure, as this will permit detection of resistance from displaced alveolar bone fragments that impede replantation. If resistance is met, the tooth should again be placed in saline while the alveolus is re-examined, and any displaced bone fragments repositioned. Repositioning can then be completed. The replanted incisor should fit loosely in the alveolus in order to prevent further damage to the root surface.

Studies have shown that rigid splinting of replanted mature and autotransplanted immature teeth increases the extent of root resorption (147, 148, 173, 239–241, 303). Replanted teeth should, therefore, only be splinted for a minimal period of time. One week to 10 days is normally sufficient to ensure adequate periodontal support, as gingival fibers in the cervical region are already healed by this time.

Proper repositioning can now be evaluated by the occlusion and the tooth splinted to adjacent incisors. An acid etch/resin splint is usually the method of choice. Finally, a radiograph is taken once the splint has been applied, to verify that the normal position of the tooth has been achieved.

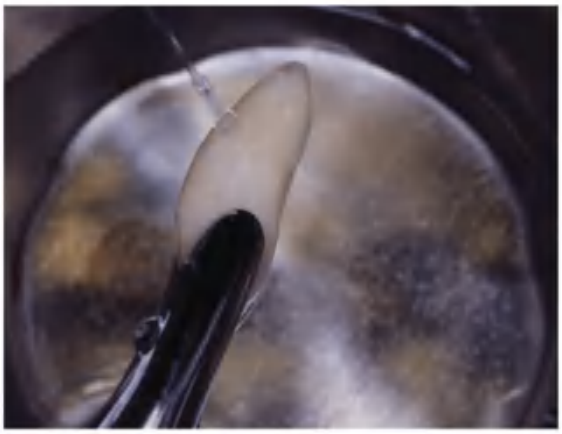
When the splint is to be removed, it is important to remember that the replanted tooth is still rather loose. It is therefore important to remove the splinting material carefully, with finger support on the replanted tooth. Furthermore, if endodontic treatment is indicated, it should be carried out prior to splint removal (see suggestions in Chapter 25).

Tetanus prophylaxis is important, as most teeth have been in contact with soil, or the wound itself is soil-contaminated.

The current Trauma guidelines recommend systemic antibiotic therapy for patients with avulsed permanent teeth (335). The purpose of the antibiotic treatment is to prevent a microbial infection. The recommended antibiotic is tetracycline in the appropriate dose for the patients age and weight, the first week after replantation. Since there is a great risk of

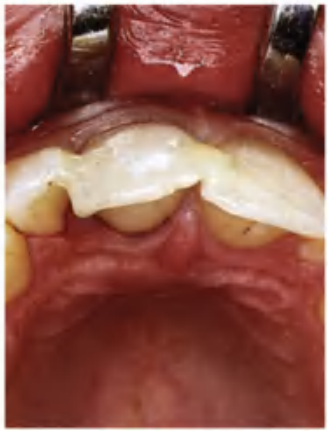
**Fig. 20.26** Replantation of a tooth with completed root formation Replantation of an avulsed maxillary right central incisor in a 19-year-old man. Radiographic examination shows no sign of fracture or contusion of the alveolar socket. The tooth was retrieved immediately after injury and kept moist in the oral cavity. Upon admission to the emergency service, the avulsed incisor was placed in physiologic saline.

**Rinsing the tooth**
The tooth is examined for fractures, position of the level of periodontal attachment and signs of contamination. The tooth is then rinsed with a stream of saline until all visible signs of contamination have been removed. If this is not effective, dirt is carefully removed using a gauze sponge soaked in saline. The coagulum in the alveolar socket is flushed out using a stream of saline.



**Replanting the tooth**
The tooth is grasped by the crown with forceps and partially replanted in its socket. Replantation is completed using gentle finger pressure. If any resistance is met, the tooth should be removed, placed again in saline and the socket inspected. A straight elevator is then inserted in the socket and an index finger is placed labially. Using lateral pressure, counterbalanced by the finger pressure, the socket wall is repositioned. Replantation can then proceed as described.

**Splinting**
An acid-etch retained splint is applied. As soon after injury as possible, antibiotic therapy should be instituted. Suggested dosage: penicillin 1 million units immediately, thereafter 2–4 million units daily for 4 days. Good oral hygiene is absolutely necessary in the healing period. This includes brushing with a soft tooth brush and a 0.1 % chlorhexidine mouth rinse.



discoloration of the permanent teeth in young growing patients, tetracycline is not recommended, instead the use of penicillin V or amoxycillin is to be preferred (335).

The use of antibiotics is further discussed in Chapter 3.

Topical antibiotics may be more beneficial to treatment outcome than systemic antibiotic. Cvek et al 1990 demonstrated in avulsed immature permanent monkey teeth that topical treatment with doxycycline for five minutes prior to replantation significantly reduced infection-related resorption as well as increased pulp survival. However, a recently published retrospective case-control study on 66 avulsed human permanent teeth (30 teeth treated topically with doxycycline and 36 teeth treated with saline) found no significant difference in treatment outcome regarding pulp survival and periodontal healing. This was regardless of root development, extra oral time and storage medium (336). One reason for the contradictory results could be that the study conditions in the animal studies cannot be replicated in humans.

One has also to consider that topical antibiotics can be considered impractical and are not generally available at dental clinics.

Also, when regarding treating avulsed teeth with topical antibiotics, the clinical evidence is week and there is need of more randomized clinical studies evaluating its effect on treatment outcome.

Finally, awaiting future research, it is recommended that dentists follow the guidelines of IADT prescribing systemic antibiotics when treating avulsed permanent teeth (325).

In the case of a closed apical foramen, where pulp necrosis can be anticipated, endodontic treatment should always be performed prophylactically. A long-debated question has been whether root canal treatment of incisors with mature root development (i.e. with the diameter of the apical foramen of less than 1.0 mm) should be performed before or after replantation, if survival of the pulp cannot be expected. Experimental studies in monkeys have shown that extraoral root filling procedures as well as the root filling materials themselves apparently injure the periodontal ligament. This could be a result of seepage through the apical foramen or mechanical preparation of the root canal, which results in increased ankylosis apically when compared to non-endodontically treated teeth (149, 152). Thus, endodontic treatment should be delayed for 1 week after replantation in order to prevent development of ankylosis and inflammatory resorption, as well as to allow splicing of periodontal ligament fibers which limits seepage of potentially harmful root filling materials into the traumatized periodontal ligament.

Where the apical foramen is wide open, and replantation has been performed within 3 hours after injury, it is justifiable to await revascularization of the pulp (200, 243). Radiographic controls should be made 2 and 3 weeks after replantation, as the first evidence of root resorption and periapical osteitis can usually be seen at this time. If this occurs, endodontic therapy should be initiated immediately, and calcium hydroxide introduced into the root canal to eliminate periapical inflammation and arrest root resorption (6, 45, 174, 244) (see Chapter 25). The timing of the endodontic procedure when pulpal revascularization is absent is critical, as root resorption can proceed very rapidly in teeth with incomplete root formation (i.e. with a speed of up to 0.1 mm loss of root substance per day) (Fig. 20.26).

In teeth with prolonged extra-alveolar periods, where the periodontal ligament can be assumed to be necrotic, it has been suggested that the root surface be treated with various substances, such as sodium fluoride (175), tetracycline, stannous fluoride (245, 246), citric acid (247, 248), hypochloric acid (161), calcium hydroxide (178), formalin (179), alcohol (180), diphosphonates (181), and indomethacin (249) in order to inhibit root resorption (Fig. 20.27). However, apart from sodium fluoride, long-term inhibition of root resorption has not been demonstrated (see Chapter 8).

Several attempts have been made to overcome the problem of ankylosis by placing different materials between the tooth and the socket, such as silicone grease and methyl methacrylate (methyl2-cyanoacrylate) (178, 182, 183), absorbable surgical sponge (Gelfoam®, The Upjohn Co., Kalamazoo, USA) (184), venous tissue (160), fascia and cutaneous connective tissue (156). The general outcome of these experiments was that root resorption was either not prevented or that the teeth were exfoliated.

To overcome the problem of resorption, a number of procedures have been developed, e.g. replacement of the apical part of the root with a cast vitallium implant (68, 73, 78, 190). However, the results of these procedures have not been convincing. An attempt has been made to prolong the life of replanted teeth by a replacement of the root tip with a ceramic implant (dense sintered aluminum oxide) or titanium (251–262) (Fig. 20.28). Before replantation, the apical half of the root is resected; the root canal enlarged with special burs, whereafter a corresponding ceramic implant is cemented in place. Preliminary studies indicate that this procedure, as expected, does not prevent root resorption, but does tend to prolong survival time of the replant (261, 262, 305).

### Follow-up procedures

Successive radiographic controls should be performed in the follow-up period in order to disclose root resorption (199) (see Appendix 3, p. 1024). If not present within the first 2 years after injury, the risk of root resorption is significantly reduced, but can still occur (202) (Fig. 20.29).

The onset of resorption appears to be earlier in cases with crown damage, visible contamination and dry extra-alveolar periods of time exceeding 15 minutes.

## Prognosis

Replantation of teeth has been considered a temporary measure as many teeth succumb to root resorption. However, a number of cases have been reported where replanted teeth have been in service for 20 to 40 years with a normal periodontium, as revealed clinically (positive pulpal sensibility) and radiographically (6, 63–68, 186) (Fig. 20.30 and

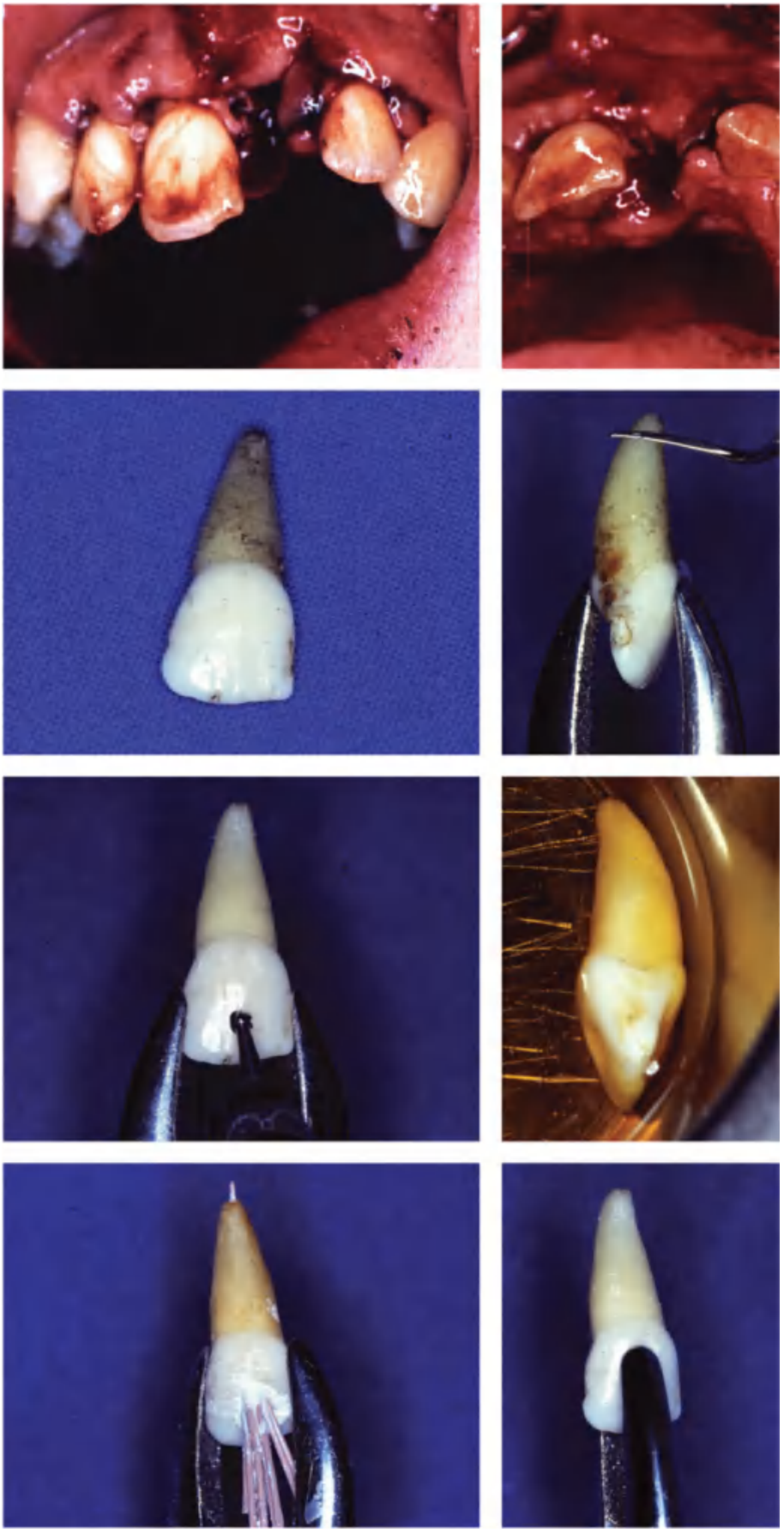


**Fig. 20.27** Replanting a tooth with an avital periodontal ligament In this 21-year-old man, the tooth has been kept dry for 24 hours. Total and irreversible damage to the PDL and pulp can be expected. Furthermore, there is severe contusion of the alveolar socket. In this situation, delayed replantation (to allow healing of the socket), treatment of the root surface (to make it resistant to ankylosis) and endodontic therapy (to prevent infection related resorption) is the treatment of choice.

**Treatment of the root surface**
The avulsed tooth in this case was kept dry in a refrigerator until healing of the contused socket has taken place. Prior to sodium fluoride treatment, the root surface is rinsed and scraped clean of the dead PDL and the pulp extirpated. The goal of therapy is to incorporate fluoride ions into the dentin and cementum in order to protract the resorption process.

**Fluoride treatment of cementum and dentin**
The pulp is extirpated and the root canal enlarged to provide access for the fluoride solution along the entire root canal. The tooth is then placed in a 2.4% solution of sodium fluoride (acidulated to pH = 5. 5) for 20 min.

**Endodontic treatment**
After rinsing in saline, the root canal is obturated with gutta percha and a sealer.

**Condition of the socket**
After 3 weeks, the socket area and the contused gingiva are healed.

**Replanting the tooth**
The socket is evacuated with excavators and a surgical bur. The tooth is replanted after cleansing with saline to remove excess fluoride solution.

**Splinting**
The tooth is splinted for 6 weeks in order to create a solid ankylosis. In these cases, where no periodontal ligament exists, ankylosis is the only possible healing modality.

**Follow-up**
Radiographic follow-up over a 3-year period shows no progression of the ankylosis process.

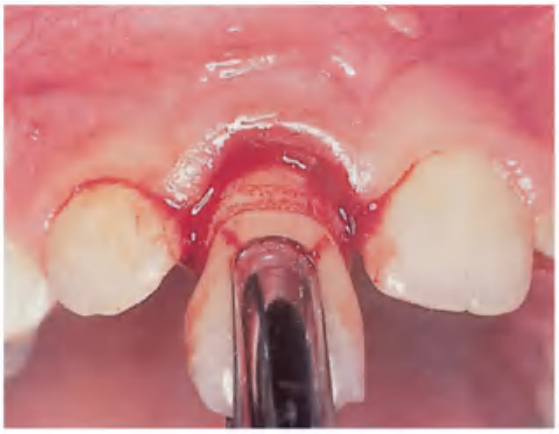
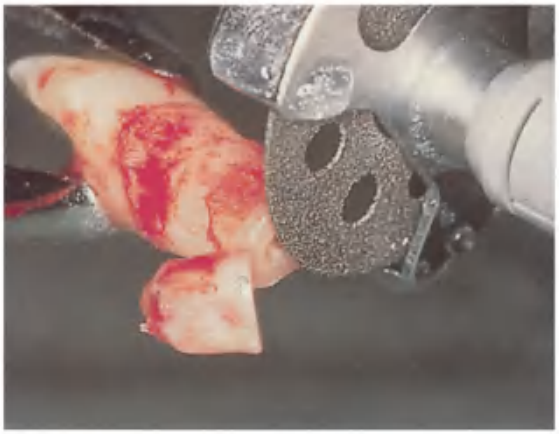
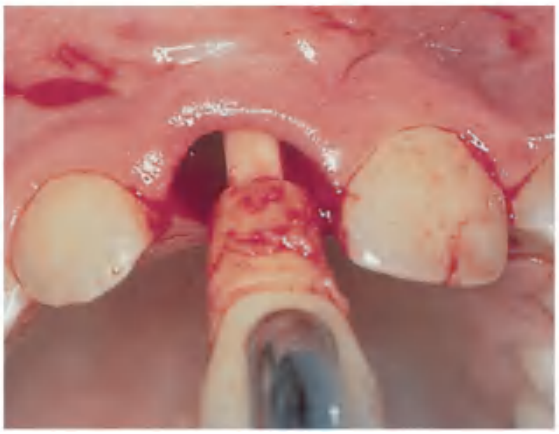


**Fig. 20.28** Apical resection and root extension with a ceramic implant. Pulp necrosis and arrested root formation in a 10-year-old child subsequent to trauma. The tooth is extracted with forceps having diamond grips. It is important that the forceps do not touch cementum after incision of the gingival fibers with a scalpel. Reproduced from (253) with permission.



**Preparing the root for the implant**
The apex is resected with a diamond disc to a level where there is no obvious resorption. The root canal is enlarged with a bur which matches the ceramic implant. The root canal is enlarged with a bur with internal cooling. During all tooth preparation the root is kept moist with saline.

**Cementing the implant**
After drying the cavity with a sterile pipe cleaner, the aluminium oxide implant (Biolox®) which matches the dimension of the drill is cemented with Diaket®.



**Replanting the tooth**
The base of the alveolus is enlarged with a drill to accommodate the root extension. After replantation, the tooth is splinted for 2 to 4 weeks. Subsequently, the crown should be opened palatally and filled to the level of the implant. The radiographic condition is shown 2 years after implantation.

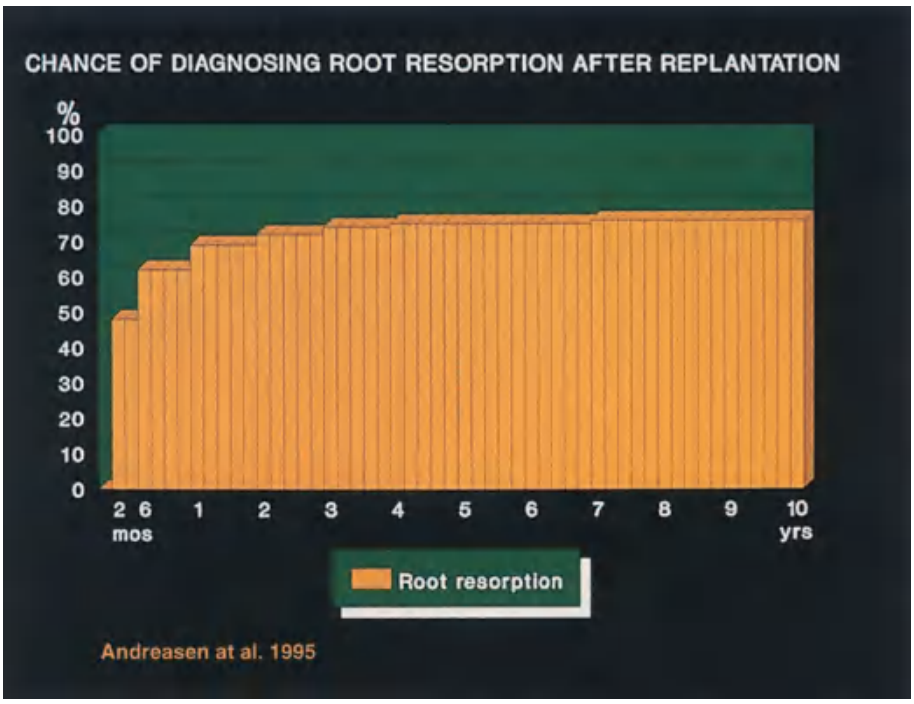



**Fig. 20.29** Chance of detection of root resorption with increasing observation period. Reproduced from (202) with permission.

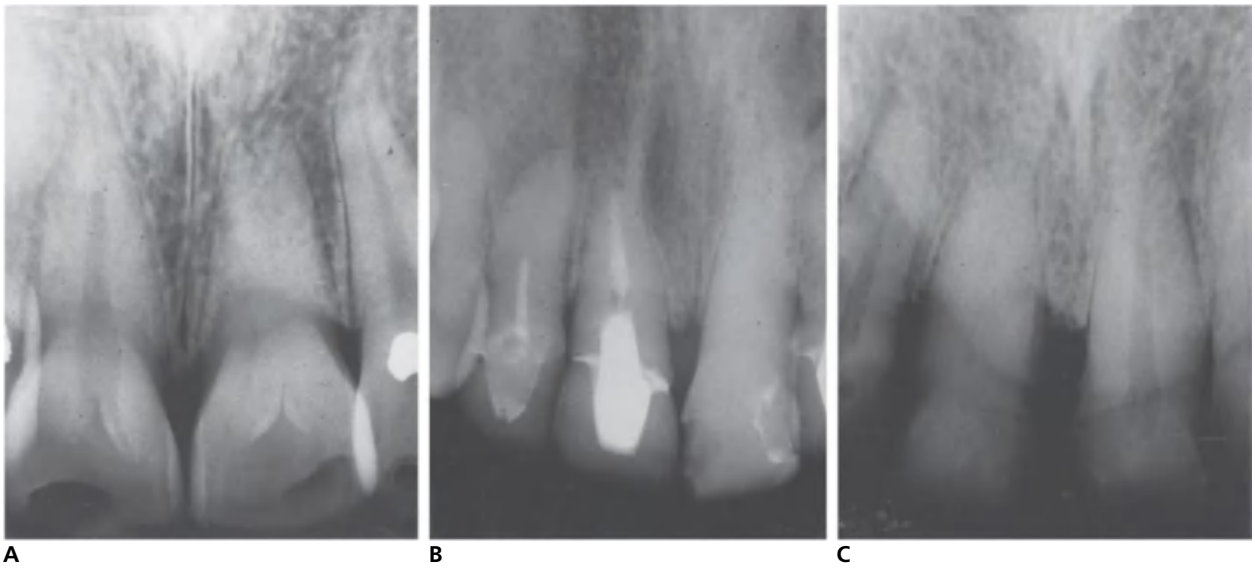



**Fig. 20.30** Cases demonstrating periodontal healing after replantation. A. Left central incisor replanted 13 years previously. B. Right central incisor replanted 37 years previously. C. Two central incisors replanted 40 years previously. Reproduced from (6) with permission.

Table 20.1). Such reports demonstrate that replanted teeth, under certain conditions, can maintain their integrity and function.

## Tooth survival and tooth loss

Several studies have shown that teeth can function for 20 years or more after replantation (263, 267).

Several studies have shown that tooth survival was significantly related to the stage of root development at the time of injury, being more favorable with increasing developmental maturity (199, 287) (Table 20.1 and Fig. 20.27).

## Pulpal healing and pulp necrosis

In rare cases, revascularization of the pulp will occur in replanted teeth with *completed* root formation, provided that replantation is carried out immediately (6, 43). Pulps

**Table 20.1** Long-term results of replantation of avulsed permanent teeth.

| Examiner | Observation period in years (mean) | Age of patients (mean) | No. of teeth | Tooth survival % | PDL healing % | Pulp healing % | Gingival healing % |
|---|---|---|---|---|---|---|---|
| Lenstrup & Skieller (7, 8) 1957, 1959 | 0.2–5 | 5–18 | 47 | 57 | 4 | 9 | |
| Andreasen & Hjörting-Hansen (6) 1966 | 0.2–15 | 6–24 | 110 | 54 | 4 | 20 | 95 |
| Gröndahl et al. (187) 1974 | 2–5 | | 45 | 69 | 7 | 23 | 67 |
| Cvek et al. (188) 1974 | 2–6.5 | 6–17(11) | 38 | | | 50 | |
| Hörster et al. (128) 1976 | | | 38 | | | 26 | |
| Kling et al. (243) 1976 | | 6–18 | 72* | | | 18 | |
| Kemp et al. (127) 1977 | 0.1–10 | 10–15 | 71 | 61 | | 30 | |
| Kock & Ullbro (263) 1982 | 1–9 | 7–17 | 55 | 65 | 4 | 27 | |
| Herforth (264) 1982 | 1–8(4.5) | 7–15 | 79 | 51 | 4 | 11 | |
| Jacobsen (265) 1986 | 1–14(5) | | 59 | 39 | 15 | 29 | |
| Mackie & Worthington (267) 1992 | 1–7 | 6–14(9) | 46 | 89 | | 46 | |
| Andreasen et al. (199–203) 1994, 1995 | 0.2–20(5.1) | 5–52(13.4) | 400 | 70 | 8 | 36 | 93 |
| Schatz et al. (285) 1995 | (2.9) | 6–17 | 33 | | 27 | 25 | |
| Ebeseleder et al. (283) 1998 | (2.5) | | 103 | 77 | 26 | 19 | 90 |
| Boyd et al. (286) 2000 | 2.0–7.1(4.8) | 6–16(9.5) | 50 | | 18 | | |
| Chappuis & von Arx (333) 2005 | (1.0) | 6–48(21) | 45 | 96 | 64 | 7 | |
| Werder et al. (321) 2011 | 1–4(2.8) | 6–63(16.3) | 42 | 84 | 50 | | |
| Roskamp et al. (319, 320) 2009, 2011 | | | 88 | | 28 | | |

*Only teeth with incomplete root formation.

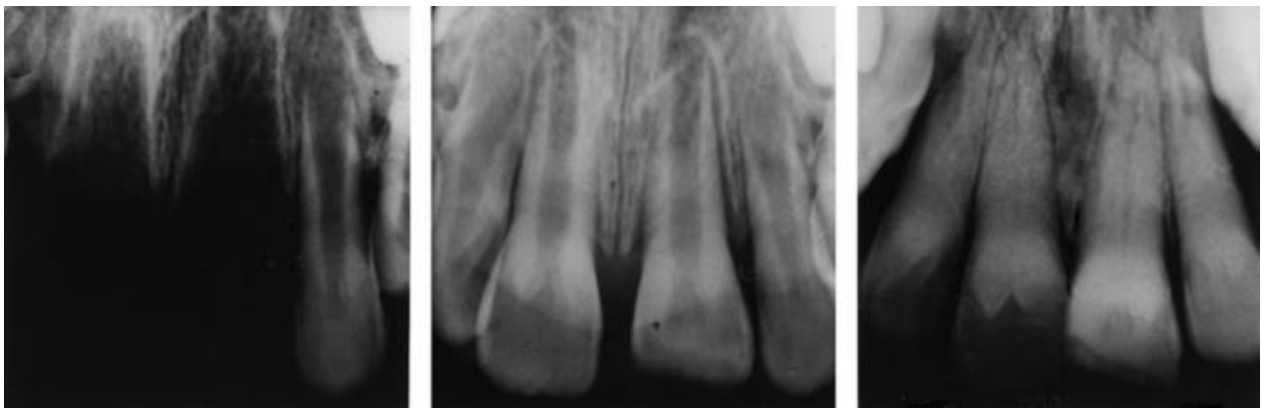


**Fig. 20.31** Pulpal healing in three replanted incisors after an extra-alveolar period of 2 minutes dry and 88 minutes in saline in an 8-year-old boy. Reproduced from (69) with permission.

of teeth with *incomplete* root formation can become revascularized if replantation is carried out within 3 hours (6, 8, 9, 43, 63, 66, 79–85, 191–196, 200, 243, 268–270, 282, 330) (Fig. 20.31). Pulpal sensibility tests are unreliable immediately after replantation. Functional repair of pulpal nerve fibers in human teeth is established, earliest in immature teeth, approximately 1–2 months after replantation. At this time electrical stimuli can elicit sensibility responses. With longer observation periods, an increasing number of teeth will respond (43, 200). In the absence of a reaction to electrical stimulation, it should be borne in mind that a decrease in the size of the coronal part of the pulp chamber or root canal (pulp canal obliteration) on the radiograph is a more reliable sign of vital pulp tissue than thermal or electrical pulp testing (43) (Fig. 20.32).

The most significant predictors of pulpal healing appear to be the width and length of the root canal as well as the duration and type of extra-alveolar storage (200).

Pulp necrosis can usually be diagnosed after 2–4 weeks and normally appears as a combined apical radiolucency and infection-related resorption located to midportion or cervical part of the root (200, 202) (Figs 20.33 and 20.34).

In Table 20.1 studies are listed which fulfill the following criteria: Study group of more than 30 teeth in order to allow reliable statistics. Furthermore, the clear criteria of pulp and periodontal healing based on a radiographic analysis has been used. Concerning PDL healing most studies showed healing rates from 20 to 40%. Only two studies from the same trauma clinic in Bern (321, 335) showed remarkably higher healing rates (50–64%). A closer examination of these

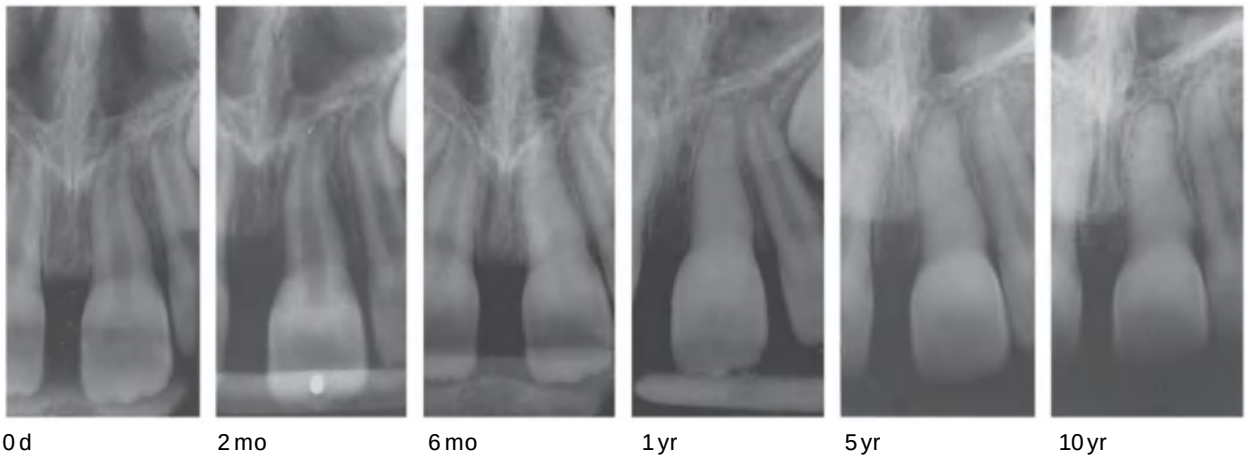



**Fig. 20.32** Pulp canal obliteration after replantation of a left central incisor. The canal obliteration becomes apparent after 6 months. Reproduced from (200) with permission.

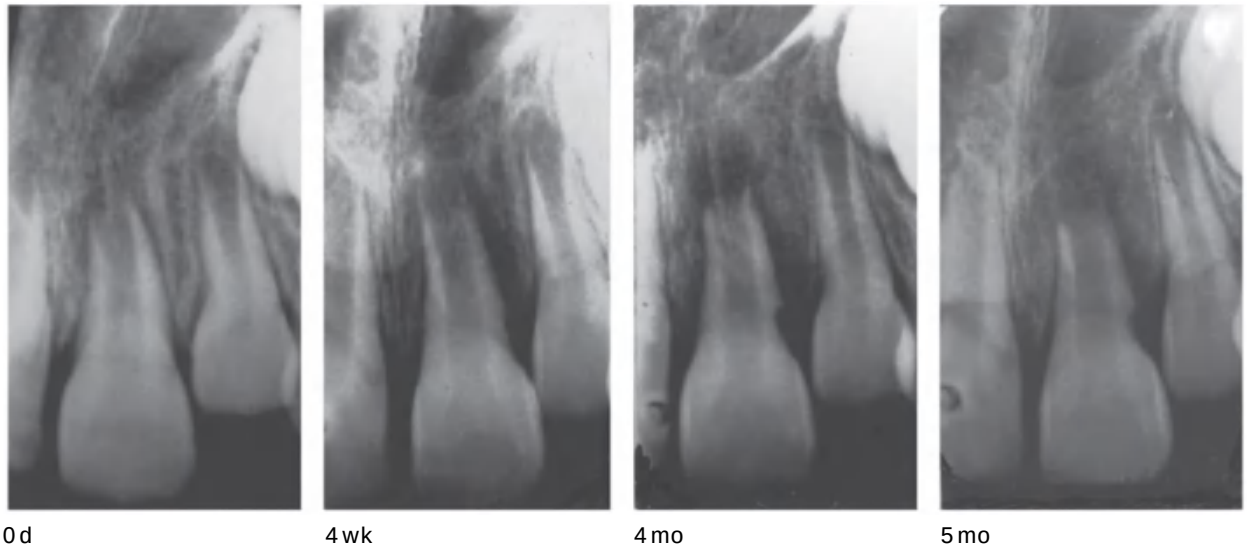



**Fig. 20.33** Infection related resorption of a left central incisor.

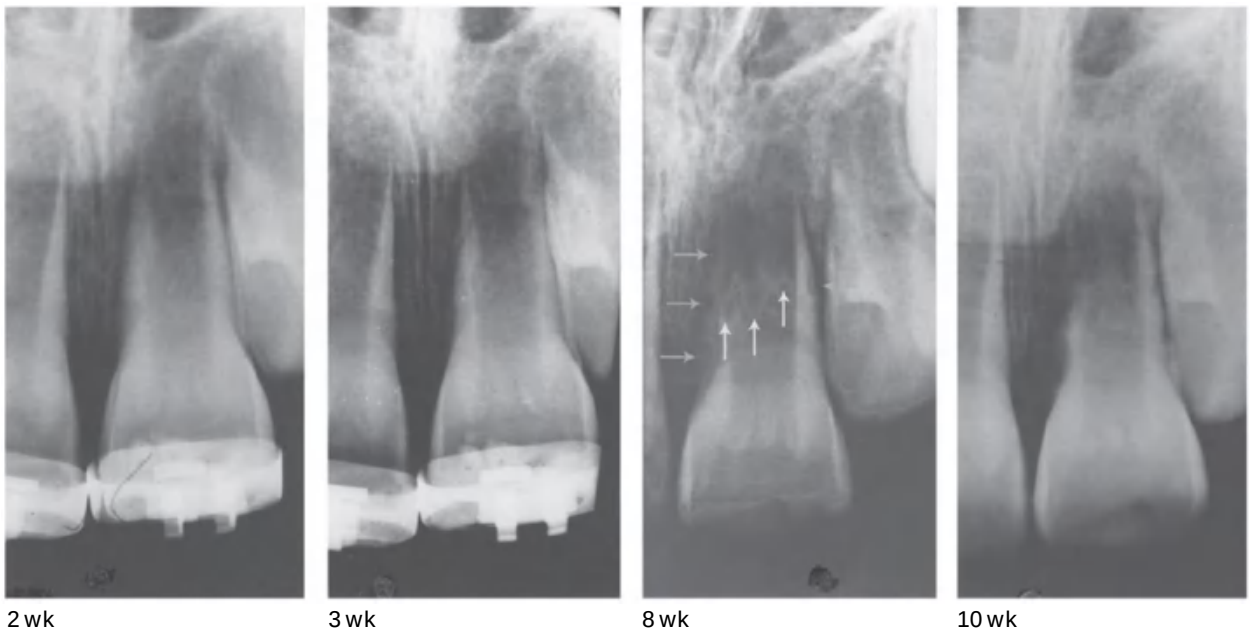



**Fig. 20.34** Infection related resorption in a replanted central incisor. Resorption is evident after 3 weeks. The resorption cavities appear as radiolucent areas within the root structure (white arrows). The mesial and distal part of the root also shows a resorption attack (gray arrows).

studies showed that great a proportion has short extra alveolar dry periods, and a strict endodontic procedure was used. The majority of the avulsed teeth were stored in physiologic media. Furthermore, pulp extirpation was performed either immediately or after a few days whereafter Ledermix® (demeclocyclononum, calcium, triamcinolone acetonidum) was installed in the root canal followed by a calcium hydroxide dressing.

### *The width and length of the root canal*

The relationship between the diameter of the apical foramen and the chance of pulpal revascularization is apparently an expression of the size of the contact area at the pulpo-periodontal interface (Fig. 20.35), whereas the length of the root canal probably reflects the time necessary to repopulate the ischemic pulp (200) (Fig. 20.36). With a favorable ratio (i.e. broad apical foramen and short root canal versus a narrow apical foramen and long root canal) the odds for an intervening pulpal infection are reduced. A limiting factor in pulpal revascularization after replantation appears to be an apical diameter of less than 1.0 mm (200, 243) (Fig. 20.35). This size, however, is to a certain degree arbitrary, as pulps in teeth with constricted apical foramina are usually extirpated prophylactically.

### *Storage period and storage media*

Another significant relationship which could be demonstrated in several clinical studies was the strong dependence between storage period and media and pulpal healing (200, 267). This is possibly due to the detrimental effect of cellular dehydration during dry storage on the apical portion of the pulp or by damage incurred by non-physiologic storage (e.g. prolonged tap water storage, chloramine, chlorhexidine and alcohol). The net result appears to be the following:

With non-physiologic storage, the chances of pulpal revascularization are minimal. With storage in physiological media (e.g. saline, saliva or milk), there is only a weak relationship between the duration of storage and chances of pulpal revascularization (200, 267) (Fig. 20.37). In contrast, dry storage yields a constant and, with time, increasingly harmful effect on pulpal healing (Fig. 20.38). However, when compared to periodontal healing, this effect is less noticeable (200, 202).

In some cases where the HERS has not survived, bony invasion of the root canal can take place; and which in rare cases might lead to internal ankylosis (201, 331) (Fig. 20.14).

## Periodontal healing and root resorption

Most replanted teeth demonstrate root resorption after a certain period of time. In the literature, the frequency of periodontal healing is usually around 20% (Table 20.1). However, a recent study following a series of public information campaigns showed a success rate of up to 36% after replantation (199).

A number of clinical factors have been shown to be associated with root resorption after replantation. Among these, the length and type of extra-alveolar storage (201), stage of root development and contamination of the root surface

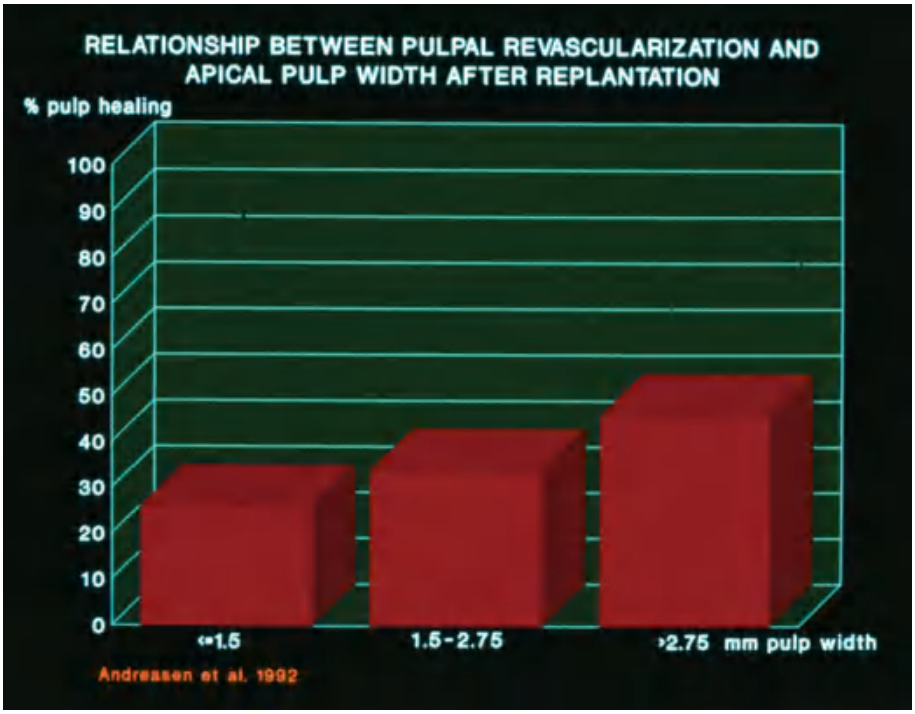



**Fig. 20.35** Relationship between size of foramen and pulp revascularization after replantation. Reproduced from (200) with permission.

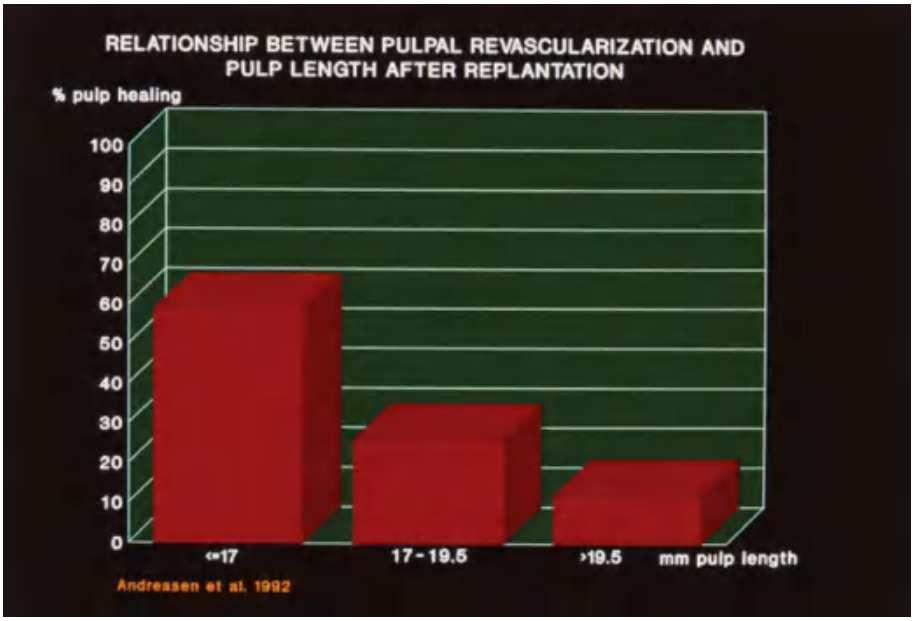



**Fig. 20.36** Relationship between length of the pulp canal and pulp revascularization after replantation. Reproduced from (200) with permission.

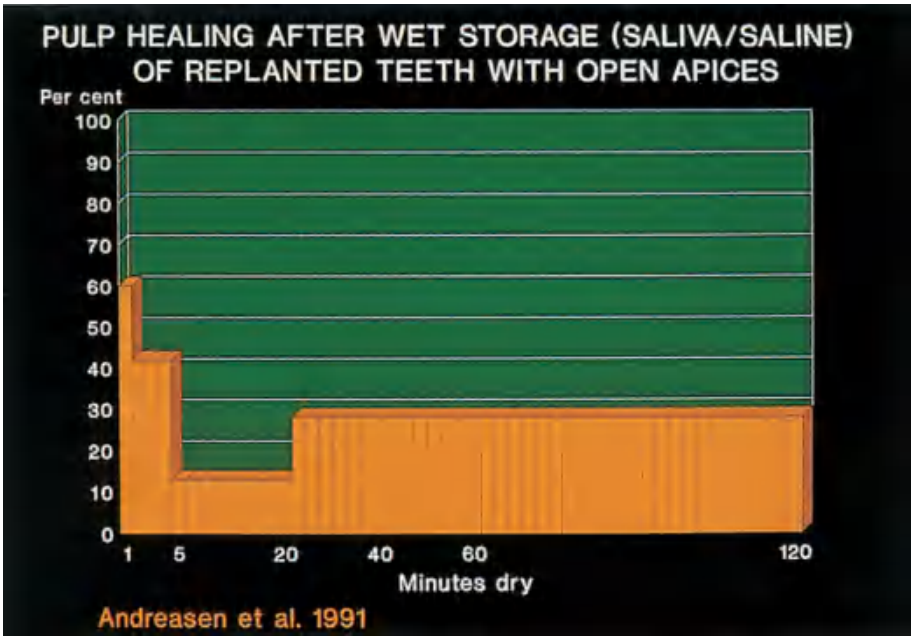



**Fig. 20.37** Relationship between pulpal healing of teeth with incomplete root formation and length of extra-alveolar wet storage. Reproduced from (200) with permission.

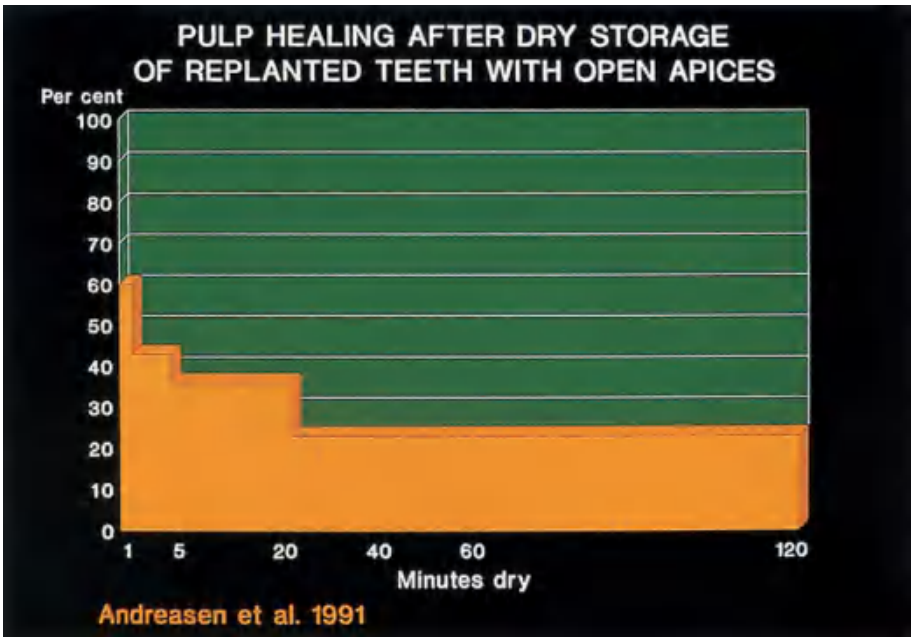



**Fig. 20.38** Relationship between pulpal healing of teeth with incomplete root formation and type and length of extra-alveolar dry storage. Reproduced from (200) with permission.

(201, 284). The type and length of storage has been found to be related to both the extent and progression of root resorption (203). Among these, the length of the dry extra-alveolar period seems to be the most crucial (6, 128, 171, 188, 189, 202, 203, 271–274, 285, 286) (Fig. 20.39). In a follow-up study of 400 teeth replanted after traumatic injury, 73% of the teeth replanted within 5 min demonstrated PDL healing; in contrast, PDL healing occurred in only 18% when the teeth were stored prior to replantation (202). These findings are in agreement with animal experiments (16, 28, 33, 70, 130, 146) assessing PDL vitality of extracted teeth allowed to dry out for varying periods (275–277).

## Storage media

Dry storage leads to cell necrosis and compromised healing (138, 146, 326). Teeth prevented from drying will heal with a normal periodontal ligament (301) In most clinical cases, avulsed teeth have been stored either in the oral cavity or in other media, such as physiologic saline or tap water, before replantation. Tap water is detrimental to the cell viability (146, 188) due to the low osmolality of tap water, resulting in rapid cell death (291, 292). Experimental studies have indicated that the storage media more than the length of the extra-alveolar period determine prognosis. Milk and saline have been documented as good storage media in a number of experimental studies (238, 288–300). The positive effect of saliva for shorter storage periods has also been documented (146, 188, 289, 292).

Storage in saline or saliva did not significantly impair periodontal healing, while even short dry storage periods had an adverse effect (146, 272). Although the above-mentioned experimental studies seem to stress the significance of the extra-alveolar period with these media, a less pronounced effect has been found in a larger clinical study (202, 203) (Figs 20.40 and 20.41). It should also be mentioned that successful cases have been reported even after extra-alveolar periods of several hours (71, 72, 202).

## Stage of root development

In a clinical avulsion situation, the layer of PDL on the root can vary in thickness from a single cell layer to the full thickness of a periodontal ligament. Thus, the more mature the root formation the thinner is the PDL tissue layer. This could possibly explain the influence of root formation upon development of root resorption found in a recent clinical study (202). Thus, a thick periodontal ligament, which supposedly can tolerate a certain dry period before evaporation has killed the critical cell layers next to the cementum, showed less dependence upon dry storage (222).

Among other factors influencing root resorption, experimental and human data have shown that the removal of the periodontal ligament prior to replantation is followed by extensive replacement resorption (25, 33, 48, 143). Consequently, this procedure, unless performed in relation to sodium fluoride treatment of the root surface is contraindicated.

## Splinting

Several clinical and experimental studies have shown that prolonged splinting (i.e. more than 10 days) leads to an increase in ankylosis-related resorption (286). Moreover, it has been found that physiologic movement during the splinting period promotes healing with less ankylosis (171, 303).

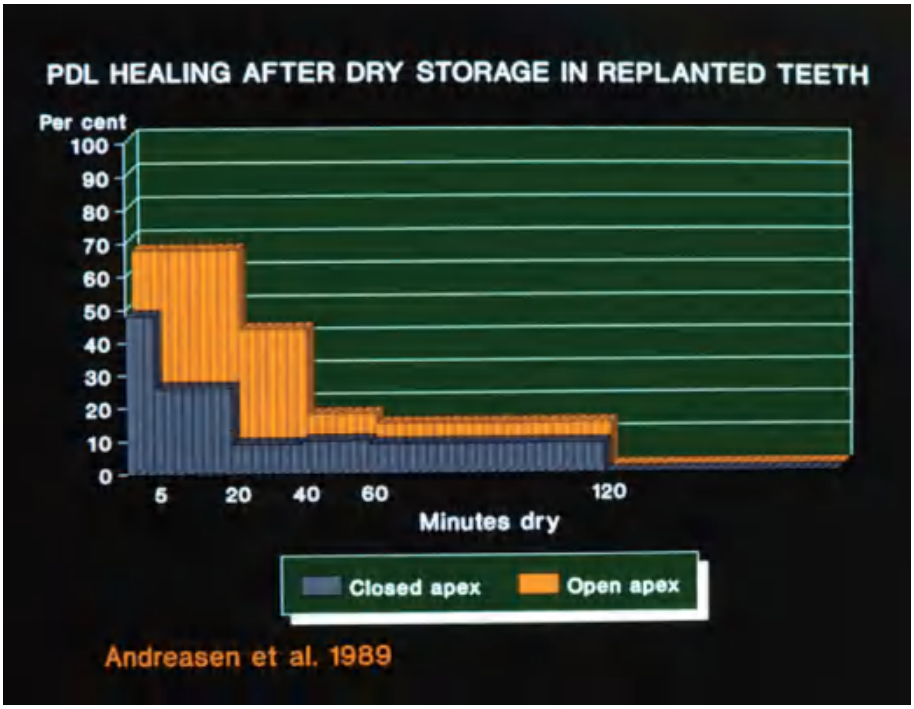



**Fig. 20.39** Relationship between PDL healing, length of extra-alveolar storage and stage of root development. Reproduced from (202) with permission.

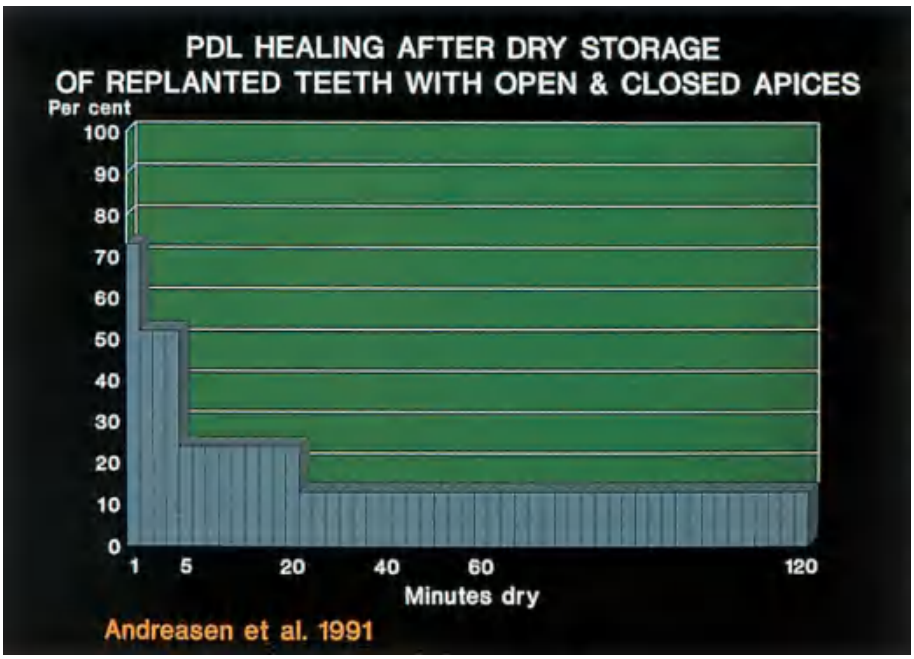



**Fig. 20.40** Relationship between PDL healing and length of extra-alveolar dry storage. Reproduced from (202) with permission.

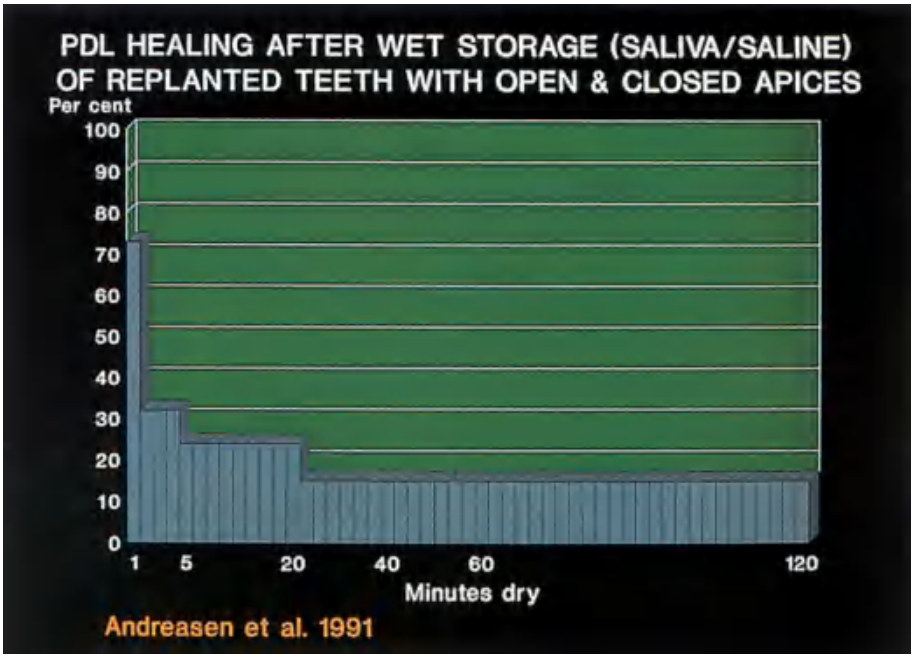



**Fig. 20.41** Relationship between PDL healing and length of extra-alveolar wet storage. Reproduced from (202) with permission.

## Time of pulp extirpation

In a recent clinical study, it was found that delayed pulp extirpation (i.e. after 20 days) resulted in acceleration of inflammatory resorption (286). In a recent systematic analysis of 4 clinical studies it was found that pulp extirpation within 10–14 days after replantation seems to reduce the risk of infection-related resorption (334).

## Contamination of the root surface

An interesting study has recently been published where the extent of contamination of the root surface prior to replantation and the cleansing procedure was found to be highly significant for subsequent PDL healing events (Tables 20.2 to 20.4). Using the results from two clinical studies (202, 284) and one experimental study (280) it seems reasonable to conclude that a short rinsing with tap water or saline is recommended.

## Ankylosis-related resorption (replacement resorption)

Ankylosis can usually be diagnosed clinically by a percussion test after 4–8 weeks, whereas radiographic evidence of root resorption usually requires a year (202). Recently, a mechanical device (Periotest®) which registers tooth mobility has also been shown to provide an early diagnosis of ankylosis (278).

Fracture of the alveolar process has been found to increase the risk of ankylosis (202) (Fig. 20.43).

A marked difference in the rate of progression of ankylosis is often seen in cases with similar extra-alveolar periods. This phenomenon might be related to differences in the initial damage to the root surface (6, 202, 203, 273), as well as the age of the patient and the type of endodontic treatment performed (203) (Fig. 20.42). Thus, if a tooth is replanted shortly after avulsion, the periodontal ligament is either re-established completely, or a few areas of ankylosis can arise (274). In the latter case, a long period will elapse before the process of replacement resorption results in total resorption of the root. Conversely, in teeth replanted with a long extra-alveolar period, an extensive ankylosis is formed which leads to rapid resorption of the root.

Alveolar fracture has been found to increase the risk of ankylosis (202) (Fig. 20.43).

If an ankylosis is established, its progression is also dependent upon the age of the patient, or rather the turnover rate of bone. Thus, replacement resorption is usually very aggressive in young individuals and runs a very protracted course in older patients (202, 203, 273).

A factor to be considered in young patients is that ankylosis can anchor the tooth in its position and thus disturb normal growth of the alveolar process. The result is a marked infra-occlusion of the replanted tooth with migration and malocclusion of adjacent teeth (6, 55, 69, 129, 281) (Fig. 20.44). The treatment of choice in these cases is either decoronation or luxation with subsequent orthodontic extrusion (see Chapter 32). Otherwise, later restorative procedures may be unnecessarily complicated by tooth migration and decreased height of the alveolar process (69). For review of treatment alternatives in cases with ankylosis and their relation to the growth of the young patient see Andersson & Malmgren 1999 (302).

In planning the decoronation of an ankylosed incisor, it is important to consider that ankylosed roots will ultimately be transformed to bone during the remodeling process. It is, therefore, not indicated to remove the root, as this will lead to a marked reduction in the height of the alveolar process. The treatment of choice is to remove the crown from the ankylosed and resorbed root and then remove the root filling. It should be mentioned that although alveolar height is indeed maintained by this procedure, it has been shown that

**Table 20.2** Relationship between visible contamination of the root surface and cleansing procedures and subsequent root resorption. After Kinirons et al. (284) 2000.

| Contamination | n | PDL resorption |
|---|---|---|
| None | 70 | 40 (57%) |
| Washed clean | 44 | 33 (75%) |
| Rubbed clean | 8 | 7 (88%) |
| Replanted but not clean $P=0.01$ | 6 | 6 (100%) |

**Table 20.3** Relation between visible contamination and subsequent root resorption. After Andreasen et al. (202) 1995.

| Contamination | n | PDL resorption |
|---|---|---|
| No | 115 | 76 (64%) |
| Yes | 56 | 11 (81%) |
| $P=0.05$ | | |

**Table 20.4** Effect of cleansing of the root surface before replantation. After Andreasen et al. (202) 1995.

| Cleansing | n | PDL resorption |
|---|---|---|
| No | 115 | 52% |
| Yes | 185 | 80% |
| $P=0.001$ | | |

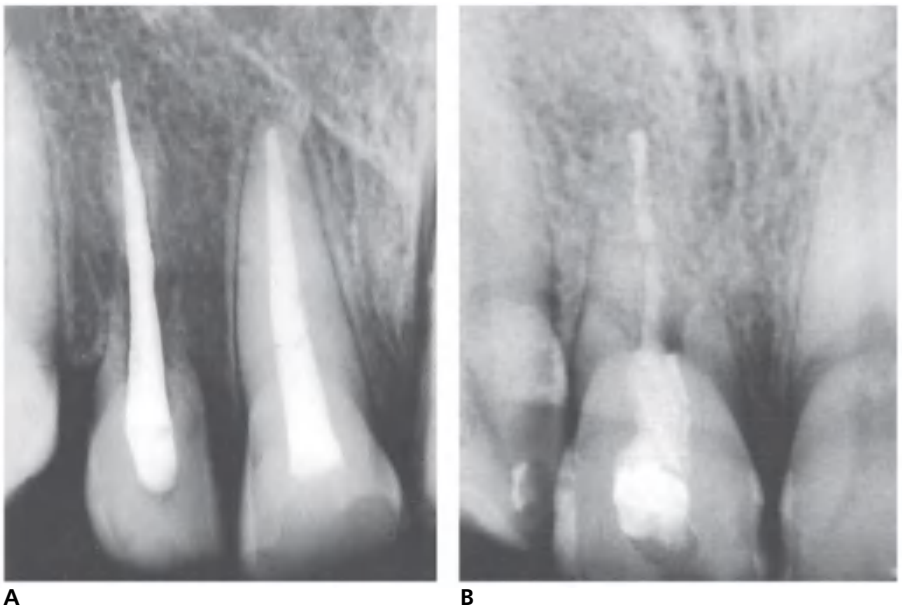


**Fig. 20.42** Difference in progression of ankylosis related resorption after replantation. A. Marked ankylosis related resorption of replanted right lateral incisor after 1½ years. B. Same degree of ankylosis related resorption 9½ years after replantation of right central incisor. Reproduced from (6) with permission.

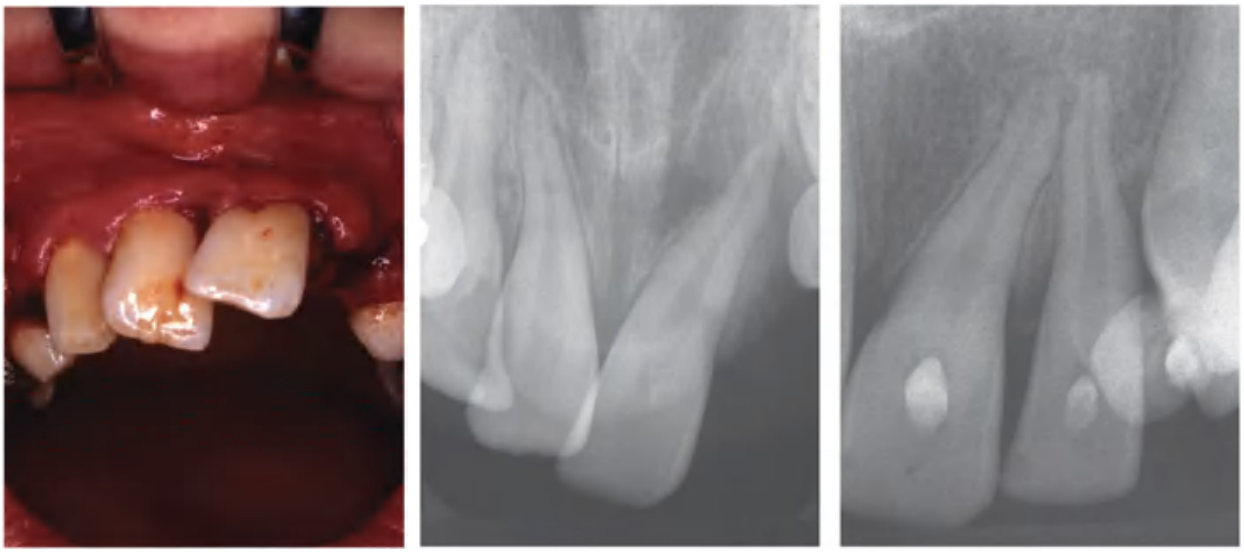



**Fig. 20.43** The socket of the lateral incisor is involved in an alveolar fracture, leading to subsequent ankylosis related resorption of the replanted incisor.

**Fig. 20.44** Progressive infraocclusion of an ankylosed replanted right central incisor. The replantation was carried out at the age of 7.

there may be a loss of alveolar bulk (332). This technique is described in Chapter 32.

In older patients an ankylosed tooth can be retained; the life-span of such a tooth can vary from 10 to 20 years, due to the slow remodelling rate of bone in older age groups.

## INFECTION-RELATED ROOT RESORPTION

Unless treated, inflammatory resorption can result in rapid loss of the replanted tooth, even as early as 3 months after replantation (6) (Fig. 20.21). This type of resorption is, as mentioned before, related to the presence of an infected pulp. Thus, human and experimental data indicate that arrest of the resorptive processes can be achieved by appropriate endodontic therapy (6, 45, 149, 188, 202) (see Chapter 25).

Replanted teeth can demonstrate simultaneous inflammatory and replacement resorption, a phenomenon possibly explained by superimposition of inflammatory resorption when replacement resorption exposes infected dentinal tubules or tubules leading to an infected necrotic pulp (Fig. 20.45).

### Resorption by erupting teeth

A special resorption phenomenon is encountered when a replanted tooth comes into contact with an erupting tooth, as when a lateral incisor lies close to the path of an erupting canine. Apparently, the pressure exerted by the follicle of the erupting tooth initiates or accelerates root resorption (202) (Fig. 20.46). A method to minimize the risk of resorption from the erupting tooth could be early removal of the primary predecessor in order to facilitate eruption, optimally in a direction away from the replanted tooth.

# Root development and disturbances in root growth

### Root growth

Continued root development can occur, especially if the pulp has become totally revascularized (Fig. 20.47). However, root development can continue despite pulp necrosis (201)

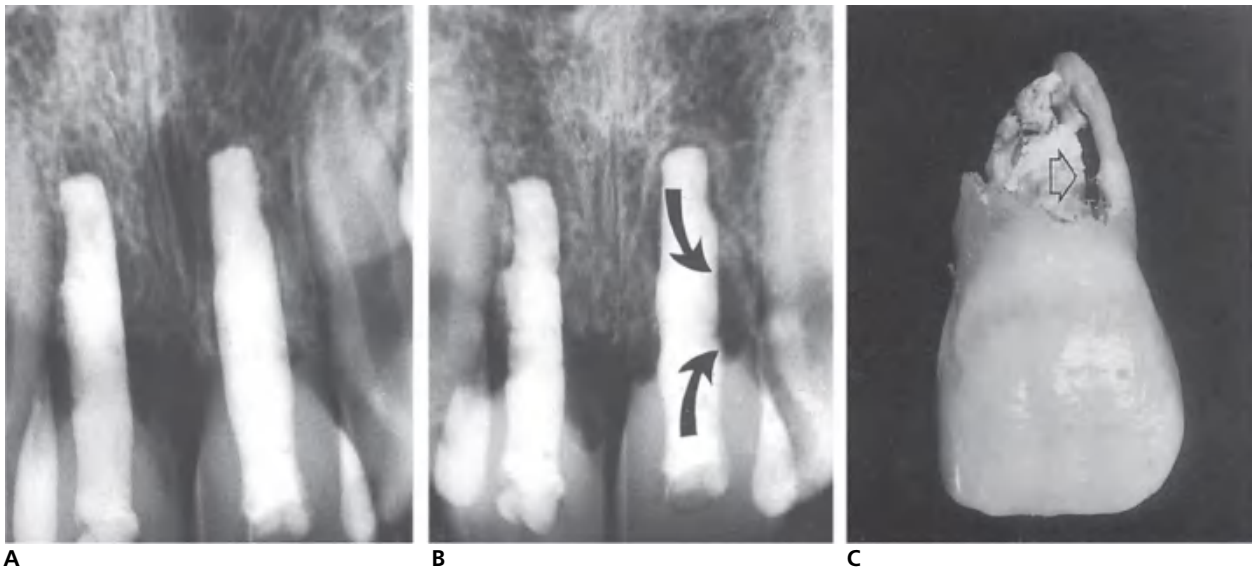



**Fig. 20.45** Left central incisor with simultaneous ankylosis and infection related resorption. A. Condition 2 years after replantation. Marked ankylosis related resorption is evident. B. Three years after replantation. Ankylosis related resorption has advanced to the root canal filling and infection related resorption is superimposed (arrows). C. The extracted tooth shows tunnelling resorption along root canal filling (arrow). Reproduced from (201) with permission.

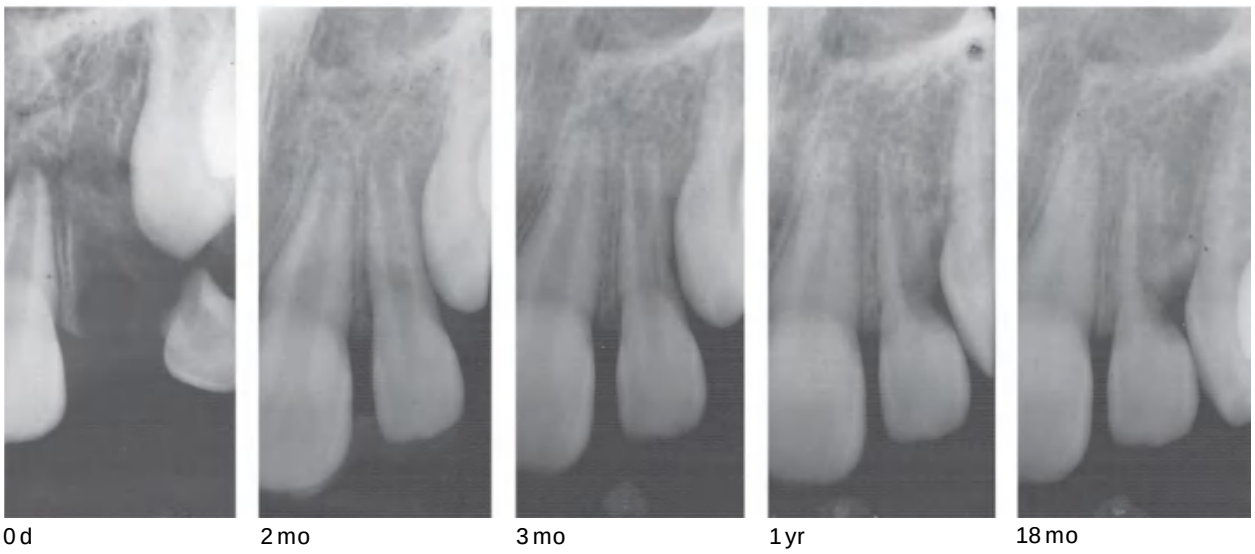



**Fig. 20.46** Replantation of a left lateral incisor after extra-alveolar period of 90 minutes. A. Radiograph immediately after replantation. B and C. Condition after 10 months and 1½ years, respectively. Note marked root resorption apparently provoked by the erupting canine. The obliteration of the root canal, especially evident in B, shows that the pulp has survived replantation. Reproduced from (201) with permission.

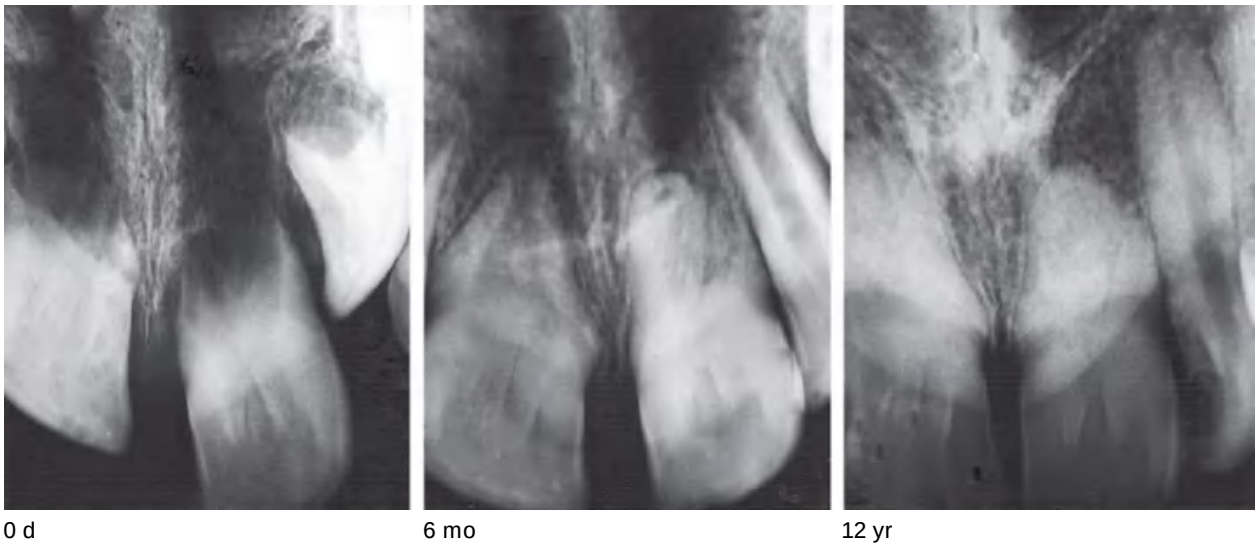



**Fig. 20.47** Continued root formation after partial pulp necrosis in a replanted left central incisor. Reproduced from (201) with permission.

(Fig. 20.48). Most often, however, root development is partially or completely arrested and the root canal becomes obliterated (Fig. 20.49) or bone and PDL can invade the pulp chamber which in some cases can lead to an ankylosis (331) (Fig. 20.50) (see also Chapter 25).

### Phantom roots

A rare complication to avulsion of immature permanent teeth is the formation of an abnormal root structure at the site of tooth loss (118–120, 194, 197, 198). The explanation for this appears to be that pulp tissue and Hertwig's epithelial root sheath remain in the alveolar socket after avulsion. These tissues resume their formative function after injury. New dentin is formed by the odontoblasts, and the Hertwig's epithelial root sheath initiates root development (118) (Fig. 20.51). A parallel to this is the tooth-like structures occasionally formed when natal or neonatal teeth are extracted and the dental papilla is left *in situ* (121, 122).

## Gingival healing and loss of marginal attachment

Gingival healing is a common finding after replantation, irrespective of storage conditions (202). In cases where the trauma has elicited extensive alveolar damage, loss of marginal bone support may occur.

## Complications due to early loss of teeth

### Malformation in the developing dentition

If it is decided not to replant an avulsed permanent tooth, problems arise with regard to further treatment. The same applies to cases where extraction of the replanted tooth is necessary due to root resorption. If no treatment is instituted, a marked degree of

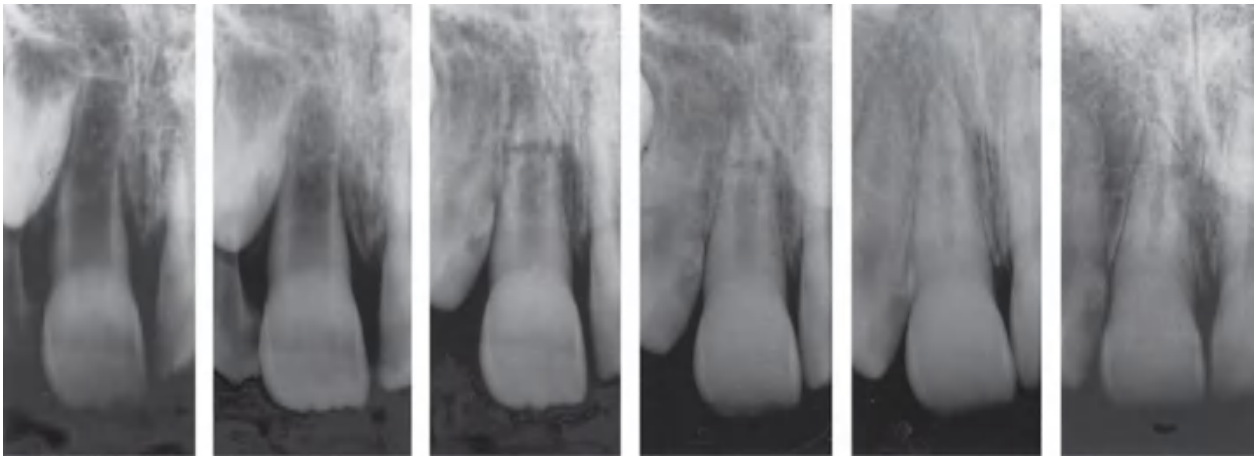



**Fig. 20.48** Continued root formation after replantation of a left central incisor. Reproduced from (201) with permission.

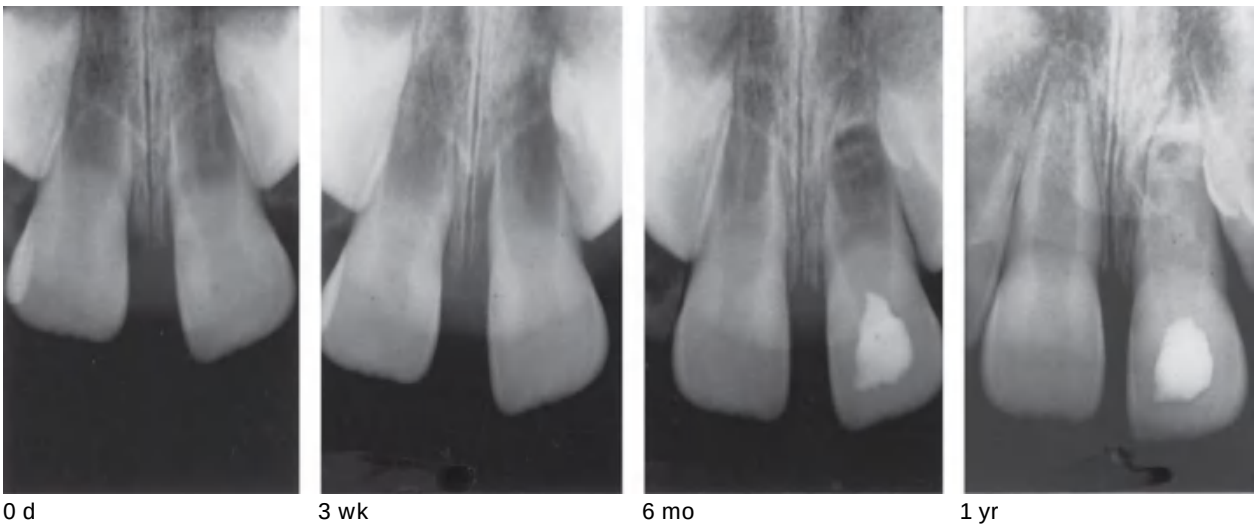



**Fig. 20.49** Arrested root formation. Bone invasion in the root canal and formation of an internal PDL in a replanted right central incisor. After 12 years infraocclusion was found apparently due to an internal ankylosis process. Reproduced from (201) with permission.

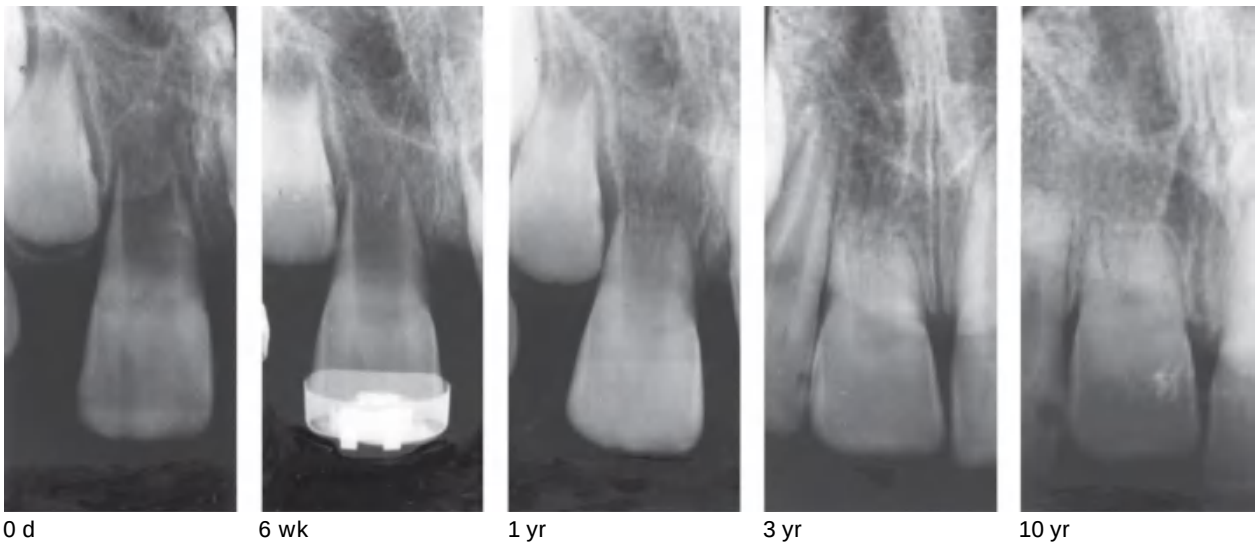



**Fig. 20.50** Pulpal healing of a replanted maxillary right central incisor with full root and pulp canal obliteration after replantation. Reproduced from (201) with permission.

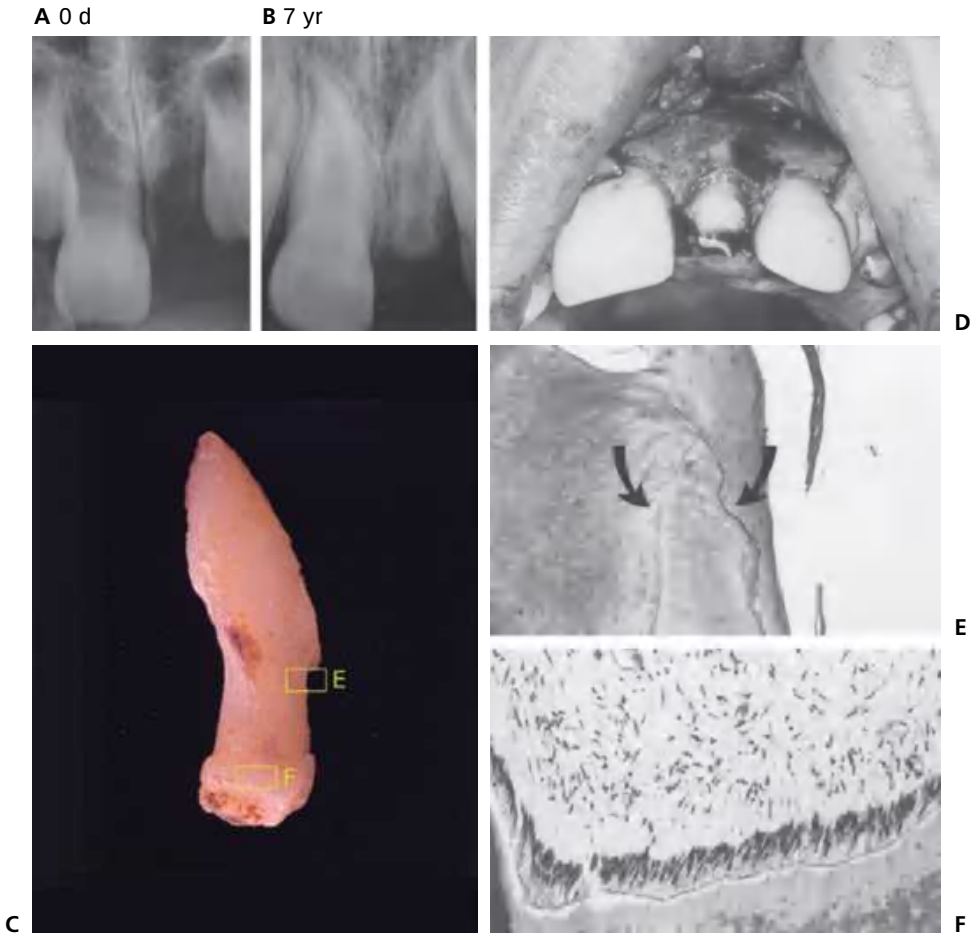



**Fig. 20.51** Phantom root formed after avulsion of a permanent central incisor at the age of 7 years. A. Radiographic condition immediately after injury. B. At follow-up 3½ years later a phantom root is found at the site of the avulsed tooth. C. The root is surgically removed. D. Low power view of sectioned incisor. × 5. E. Fragment of dentin (arrows) representing the apical area at time of injury which together with pulpal tissue and the Hertwig's epithelial root sheath has been left in the tooth socket after avulsion. × 75. F. Normal dentin and odontoblastic layer in the coronal part of the root structure. × 195. Reproduced from (118) with permission.

spontaneous tooth migration is often seen (90–92, 129). Unfortunately, this drifting is often esthetically undesirable due to midline deviation. Therapy should therefore consist of either orthodontic space closure, prosthetic tooth replacement or autotransplantation or implantation to the site. These treatment solutions are described in Chapters 31–36.

## Socioeconomic burden of tooth replantation

Due to the frequent development of healing complications after replantation of an avulsed tooth, this topic is very relevant (310, 311, 313, 314, 316). This issue will be addressed in length in Chapter 41.

## Essentials

### Terminology

Complete avulsion (exarticulation)

### Frequency

Permanent dentition: 0.5 to 16% of dental injuries

### Etiology

- Fall injuries
- Sports injuries
- Fight injuries

### Advice for the site of accident (e.g. telephone advice)

- Replant the tooth (primary teeth should however not be replanted).
- If contaminated rinse in tap water for 10 seconds.
- If not possible to replant store in patient's mouth (saliva), milk or saline.
- Visit a dental clinic as soon as possible.

### History

- Extra-alveolar period
- Condition under which the tooth has been preserved

### Clinical examination

- Condition of the avulsed tooth
- Condition of the alveolus

### Radiographic examination

Associated bone fractures.

### Healing and pathology

#### *Repair of pulpal and periodontal structures*

Root resorption, external:

- Repair-related resorption, related to minor areas of damage to the periodontal ligament upon the avulsed tooth.
- Ankylosis-related resorption, permanent or transient depending upon the initial extent of damage to the periodontal ligament upon the avulsed tooth.
- Infection-related resorption, related to the presence of an infected root canal and associated damage to the root surface.

### Treatment of permanent teeth

#### *Indications for replantation*

- The avulsed tooth should be without advanced periodontal disease.
- The alveolar socket should be reasonably intact in order to provide a seat for the avulsed tooth.

#### *Procedure for replantation (Fig. 20.22)*

#### *Short extra-alveolar storage*

- Immediate replantation by the patient should be encouraged. Otherwise the tooth should be stored in saline, saliva or milk.
- If obviously contaminated, cleanse the root surface and the apical foramen with a stream of saline (from a syringe). No attempt should be made to sterilize the root surface.
- Flush the coagulum from the socket with a flow of saline. Examine the alveolar socket. If there is a fracture of the socket wall, reposition the fracture with an instrument.
- Replant the tooth in its socket using light digital pressure.
- Suture gingival lacerations.
- Apply a splint and maintain it for 7–10 days.
- Verify normal position of the replanted tooth radiographically.
- Provide tetanus prophylaxis and antibiotic therapy.
- In the case of mature teeth with a narrow apical foramen, endodontic therapy should be instituted 7–10 days after replantation and prior to splint removal (see Chapter 25).
- When the apical foramen is wide open, and the tooth replanted within 3 hours, revascularization of the pulp is possible.
- Control the tooth radiographically. If signs of infection-related resorption appear, institute root canal treatment immediately (see Chapter 25).
- Follow-up: minimum 1 year.

#### *Long extra-alveolar period*

In cases with an extra-oral dry period of 60 min. or more, treatment of the tooth with sodium fluoride should be considered.

- Remove the PDL and extirpate the pulp.
- Root fill the tooth extra-orally.
- Remove the coagulum from the socket.
- Replant the tooth.
- Splint the tooth for 6 weeks.
- Follow-up: minimum 1 year.

### Treatment of root resorption

#### *Repair-related resorption*

No treatment indicated.

#### *Ankylosis-related resorption*

- Decoronation in cases with progressive infraocclusion of the ankylosed tooth and where residual alveolar growth is anticipated.
- Otherwise, preservation of the tooth in the interim before final treatment.

### *Infection-related resorption*

Institute root canal therapy (see Chapter 25).

## Prognosis

- Tooth survival: 51 to 89%
- PDL healing: 9 to 50%
- Pulp healing: 4 to 27%

## Complications after premature loss of permanent teeth

- Close the space orthodontically (see Chapter 28).
- Maintain the space by means of autotransplantation, bridge/implant prosthetics or orthodontic closure (see Chapters 31, 33 and 35).

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# 21

# Injuries to the Supporting Bone

J. O. Andreasen & E. Lauridsen

## Terminology, frequency and etiology

Injuries to the supporting bone can be divided into the following types (Fig. 21.1).

1. *Comminution of the alveolar socket:* Crushing of the alveolar socket, associated with intrusive or lateral luxation.
2. *Fracture of the alveolar socket wall:* A fracture confined to the facial or lingual socket wall.
3. *Fracture of the alveolar process:* A fracture of the alveolar process which may or may not involve alveolar sockets.
4. *Fracture of mandible or maxilla (jaw fracture)* A fracture involving the base of the mandible or maxilla and usually also the alveolar process. The fracture may or may not involve alveolar sockets.

The primary etiologic factors are fights and automobile accidents (1, 2, 159). Thus, after automobile accidents, alveolar fractures result from direct impact against the steering wheel or other interior structures (2, 3, 76, 159).

In infants, where only the incisors have erupted, lack of support in the lateral regions can imply that forceful occlusion resulting from impact to the chin can result in fracture of the anterior portion of the alveolar process (Fig. 21.2).

## Clinical findings

Clinical features and treatment of comminution of the alveolar socket have already been described in connection with luxation injuries (see Chapter 16).

Fractures of the *alveolar socket wall* are primarily seen in the upper incisor region, where the fracture usually involves several teeth (1). Among associated dental injuries, luxation with dislocation and avulsion are the most common (Fig. 21.3). Palpation usually discloses the fracture site. Abnormal mobility of the socket wall is demonstrated when the mobility of involved teeth is tested.

Fractures of the *alveolar process* usually affect older age groups. A common location is the anterior region, but the canine and premolar regions can also be involved (159). The fracture line may be positioned beyond the apices, but in most cases involves the alveolar socket. In these cases concomitant dental injuries, such as extrusive or lateral luxations as well as root fractures, are common findings (1–6, 159, 160).

Fracture of the alveolar process is usually easy to diagnose due to displacement and the mobility of the fragment (Fig. 21.4). Typically, when mobility of a single tooth is tested, adjacent teeth move with it. Furthermore, the percussion tone of the teeth in the fragment differs clearly from adjacent teeth in that the former yield a dull sound.

Approximately one-half of all jaw fractures involve teeth in the fracture line (Fig. 21.5), and most of these are found in the mandible (8) (see p. 535).

The location of *jaw fractures* is significantly related to the state of the dentition (11). Of the tooth-bearing areas, the lower third molar region is most often involved; the mandibular canine, incisor and premolar regions follow with decreasing frequency (12, 13). The presence of a marginal periodontal bone defect also appears to be related to the location of a fracture line (85). In children, developing permanent teeth in the line of fracture are usually seen in the mandibular canine and incisor regions (14, 15).

Clinically, there is displacement of fragments and disturbance of occlusion (Fig. 21.5). Palpation with a finger placed over the alveolar process can disclose a step in the bony contours. In the absence of dislocation, bimanual manipulation of the jaws will usually reveal mobility between the fragments, often with accompanying crepitus. Pain provoked by movement of the mandible or maxilla, or upon palpation, is also a positive sign of fracture.

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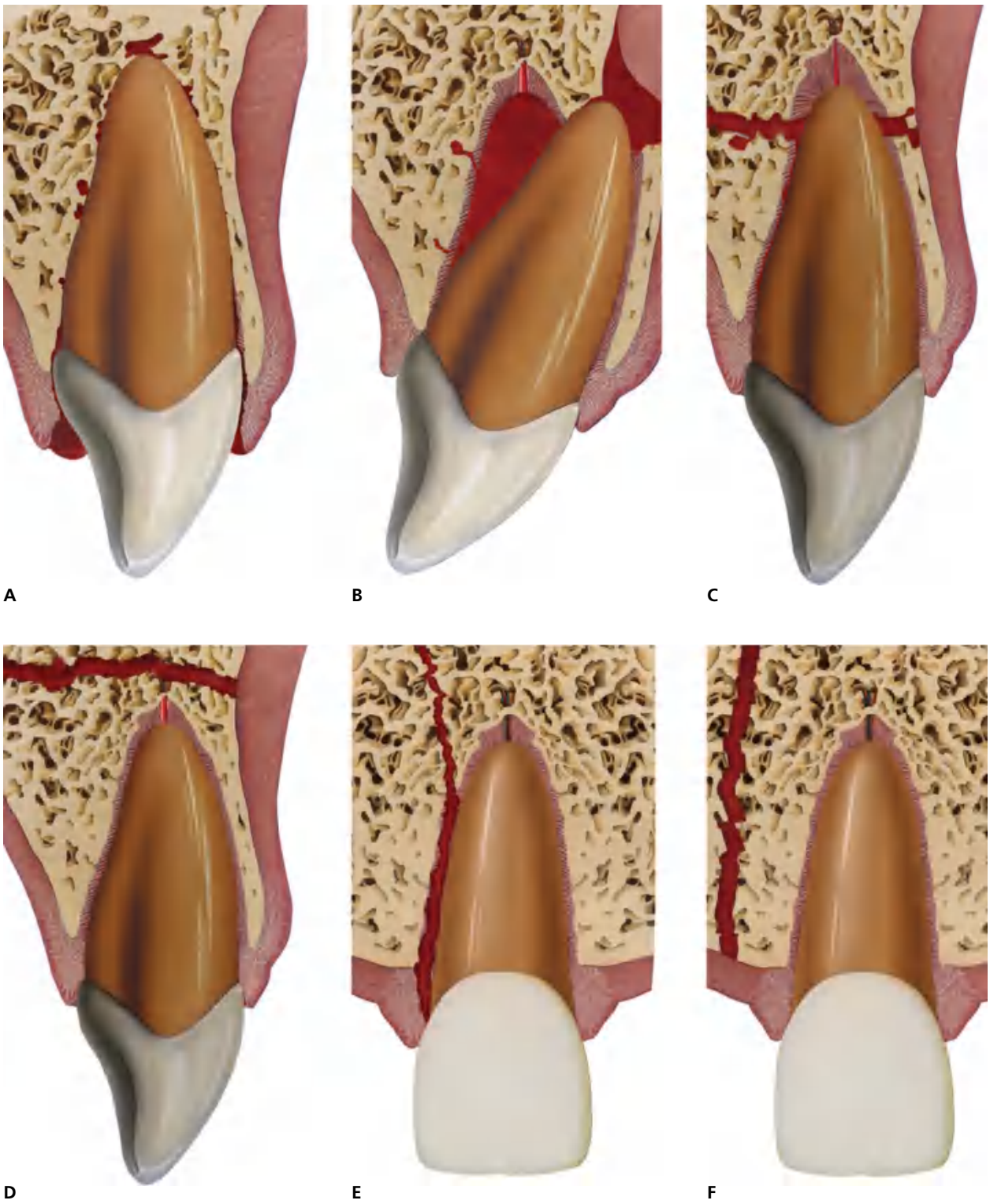



**Fig. 21.1** Injuries to the supporting bone. A. Comminution of alveolar socket. B. Fractures of the facial or lingual alveolar socket wall. C and D. Fractures of the alveolar process with (C) and without (D) involvement of the socket. E and F. Fractures of the mandible or maxilla (jaw fractures) with and without involvement of the socket.

## Radiographic findings

Intraoral radiographs of the socket wall seldom reveal the line of fracture; but a laterally exposed extraoral radiograph will usually disclose the fracture's location (Fig. 21.6). In fractures involving the alveolar process, a distinct fracture line is usually visible in intraoral as well as extraoral radiographs (Figs 21.6–21.8). Fracture lines may be positioned at all levels, from the marginal bone to the root apex (Fig. 21.8).

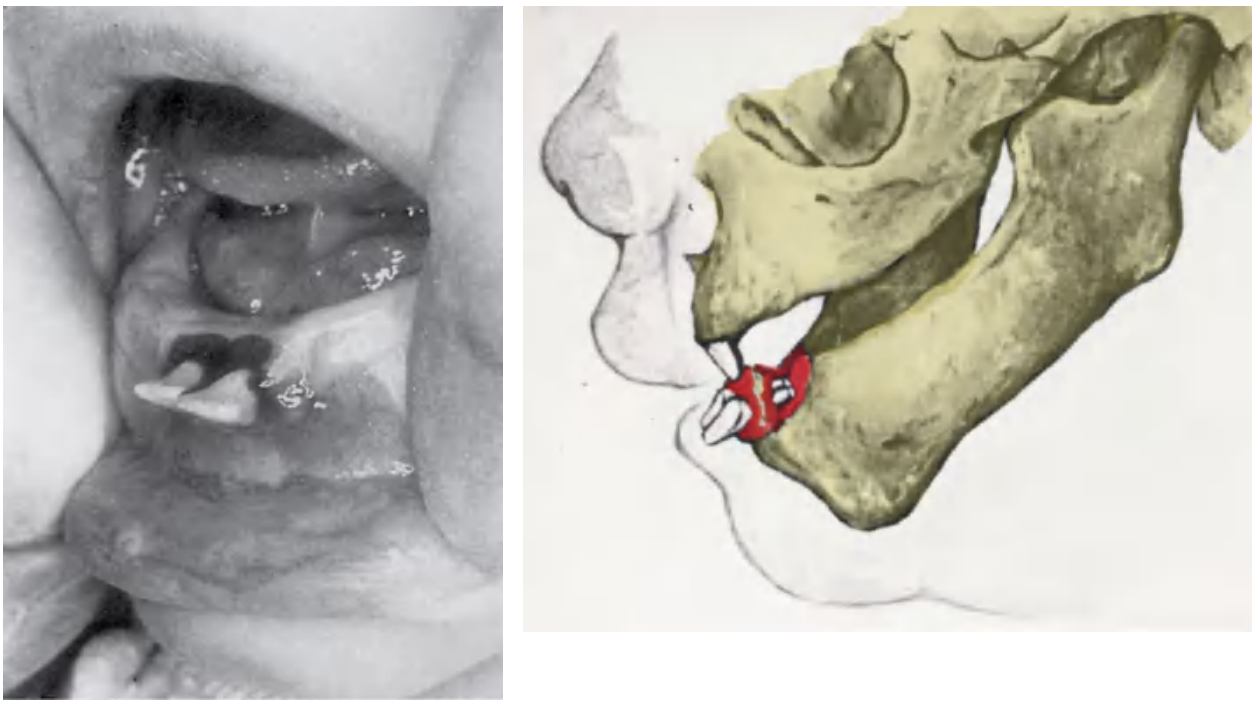


**Fig. 21.2** Pathogenesis of fractures of the mandibular alveolar process in infants. The energy from impact to the chin area is transmitted exclusively to the incisor region due to the lack of lateral tooth support. Reproduced from (4) with permission.

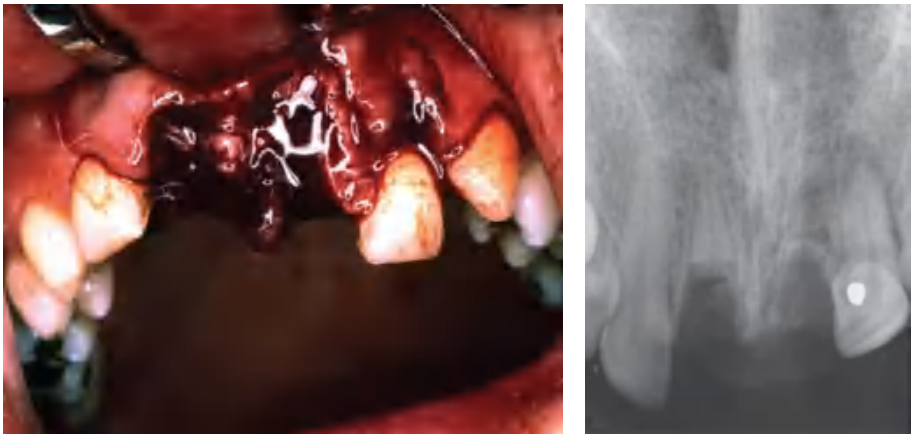
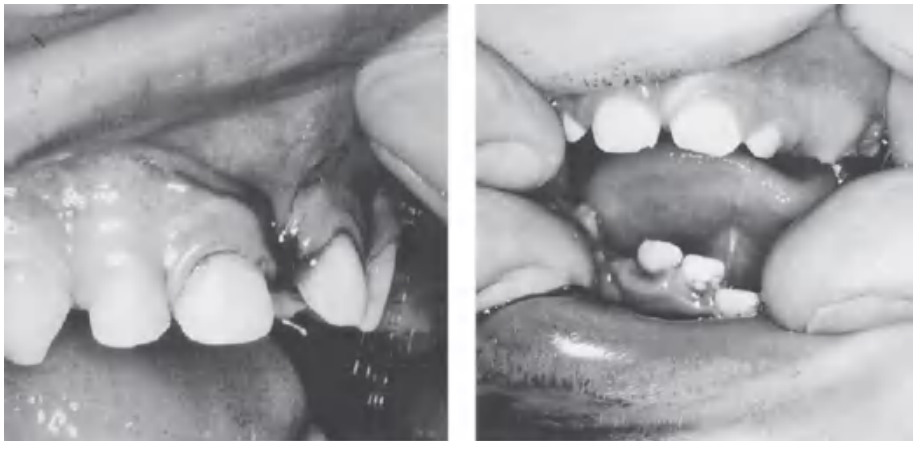
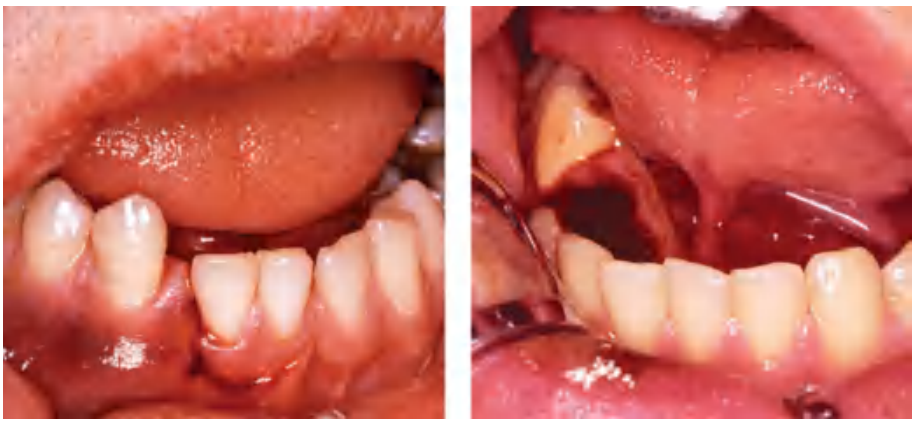


**Fig. 21.3** Contusion and gingival laceration of the facial socket wall following avulsion of both central incisors.



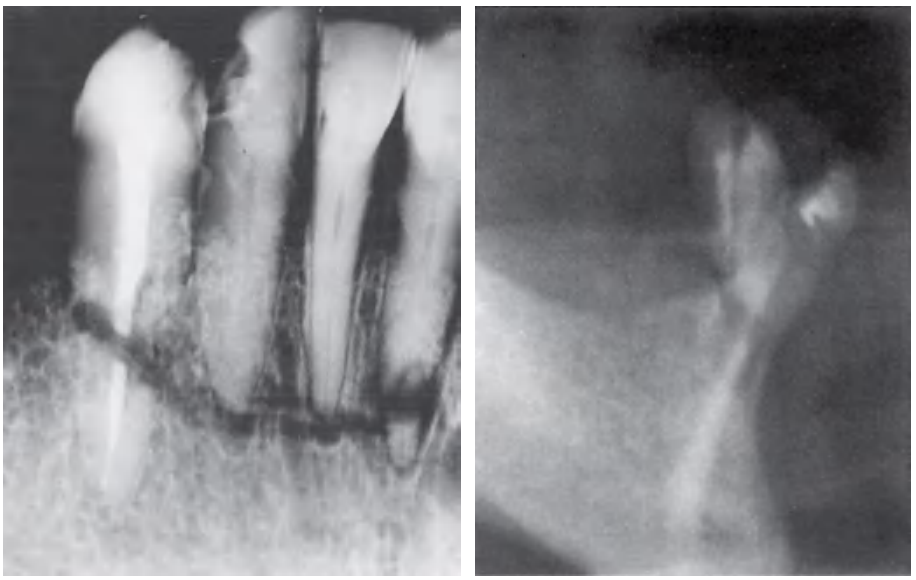
**A** **B**

**Fig. 21.4** A. Facial displacement of alveolar fracture affecting left incisors in a 2-year-old boy. B. Lingual displacement of alveolar fracture, involving right incisors in a $1^1/_2$-year-old child.



A B

**Fig. 21.5** A. Marked displacement of fragments in a patient with jaw fracture in the right molar region. B. Jaw, fracture in right canine region. Note gingival laceration and slight displacement of fragments.



A B

**Fig. 21.6** Lateral luxation of a maxillary central incisor with associated fracture of the facial socket wall. A. No fracture visible on an intraoral radiograph. B. A fracture of the facial socket wall is evident on a lateral extraoral radiograph.

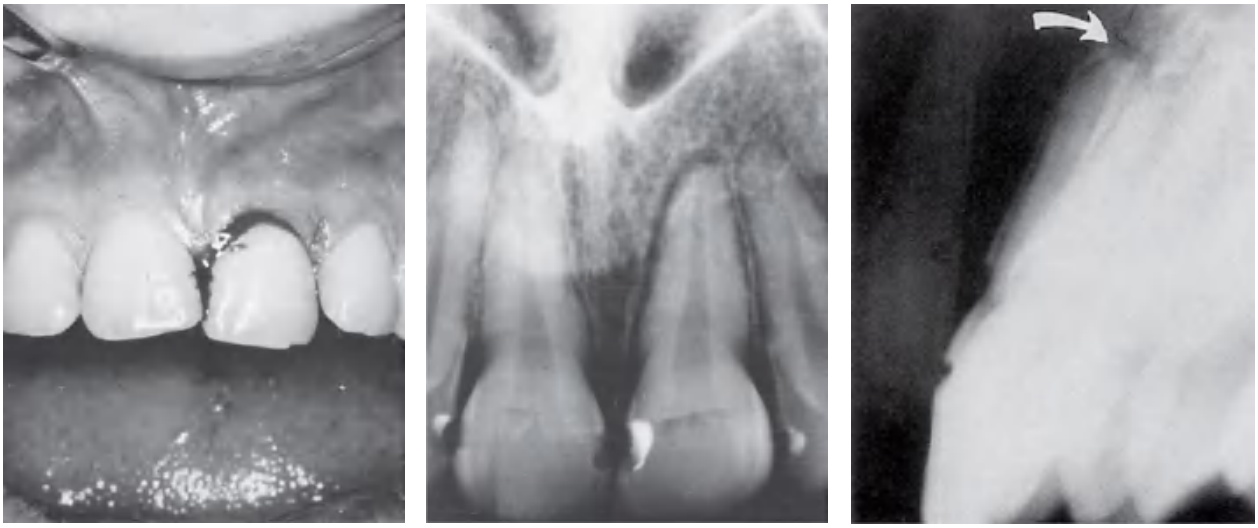


**Fig. 21.7** Dislocation of mandibular alveolar fragment clearly demonstrated by the extraoral lateral radiographic technique (arrow).

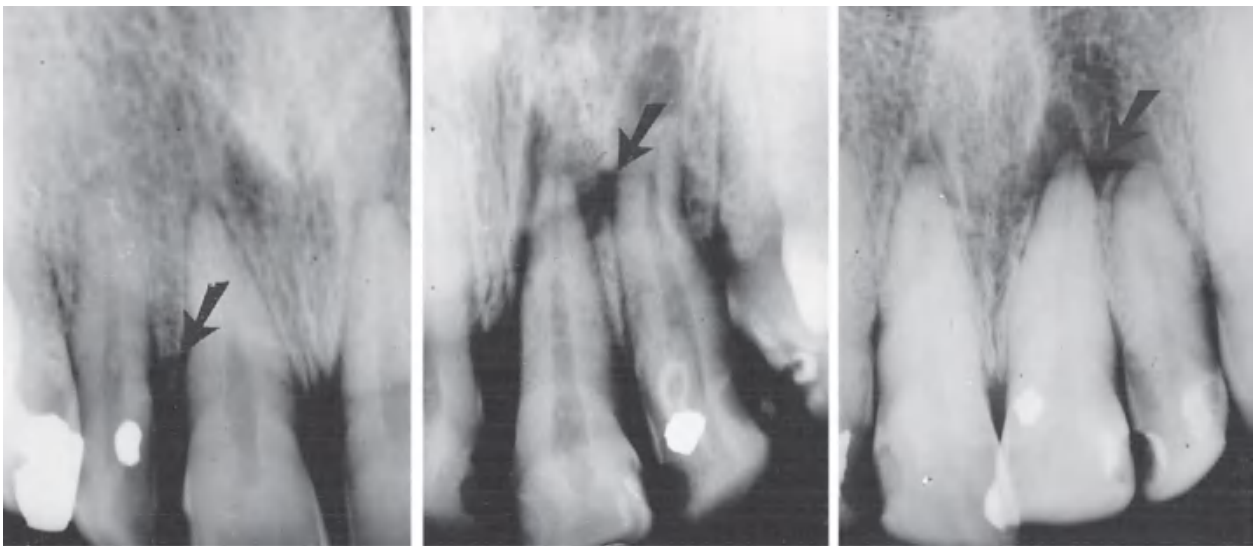



**Fig. 21.8** Various locations of alveolar fractures. A. Fracture line passing through the marginal part of the interdental septum (arrow). B. Alveolar fracture traversing the septal bone close to the midportion of the root (arrow). Note the marked extrusive luxation of involved teeth. C. Alveolar fracture located close to the apices (arrow).

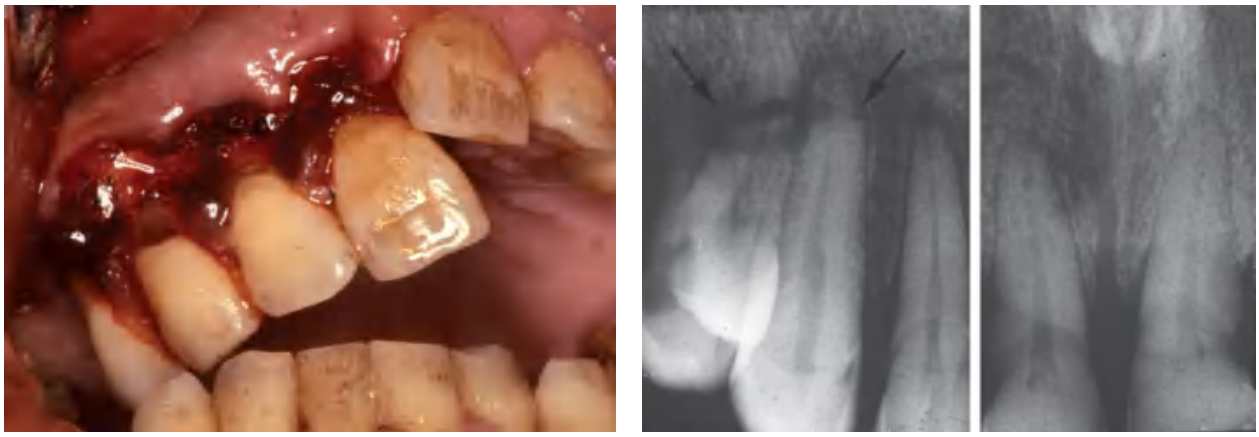


**Fig. 21.9** Fracture of the right maxillary alveolar process. Radiograph taken after reduction reveals the fracture line. Note the associated root fracture of upper first premolar. The alveolar fracture mimics a root fracture in the canine region.

Despite strong clinical evidence of a fracture, they are often very difficult to visualize radiographically (Figs 21.9 and 21.10). Cone beam computed topography (CBCT) may give a good 3D visualization of the whole fracture site (160).

When fracture lines traverse interdental septa, extrusive luxation and root fractures are often concomitant findings. Fractures involving the most apical portion of the root are quite common, but are often overlooked, especially in the lower anterior region. On the other hand, alveolar fracture lines transversing the apices may simulate root fractures. Careful examination of the radiographs (e.g. continuity of root surfaces and root canals) will usually reveal a superimposed fracture line and intact teeth (Fig. 21.9). Moreover, superimposed alveolar fracture lines will change position in relation to height along the root surface when the angulation of the central beam is altered (Fig. 21.11).

Radiographic examination of mandibular or maxillary fractures with tooth involvement should include extraoral as well as intraoral exposures. Generally, extraoral radiographs, especially panoramic exposures, are valuable in determining the course and position of fracture lines (84, 85) (Fig. 21.12), while intraoral radiographs can reveal the relationship between the fracture line and involved teeth. This is especially important in cases of maxillary fractures, which can be difficult to diagnose from extraoral radiographs due to superimposition of many anatomical structures (Fig. 21.13). Again, here CBCT may give precise information about displacement of fractures (160).

The fracture usually follows the midline of the septum or the contours of the alveolar socket; but a combination of these routes can sometimes be seen (Fig. 21.14). Fractures of the body of the mandible do not always run parallel with the long axis of the teeth, but obliquely downwards and posterior towards the base of the mandible (13, 16) (Fig. 21.15).

Fractures generally follow the path of least resistance (Figs 21.14 and 21.16). Thus, at the angle of the mandible,

**Fig. 21.10** Alveolar fracture confined to the central incisor region. Although clinically obvious at the time of injury, there is no clear radiographic evidence of bone fracture.

**A** **B**

**Fig. 21.11** Alveolar fracture in the canine and premolar regions. A. In the canine region the fracture line simulates a root fracture. B. By altering angulation of the central beam, the superimposed fracture line changes position, thus ruling out a root fracture.

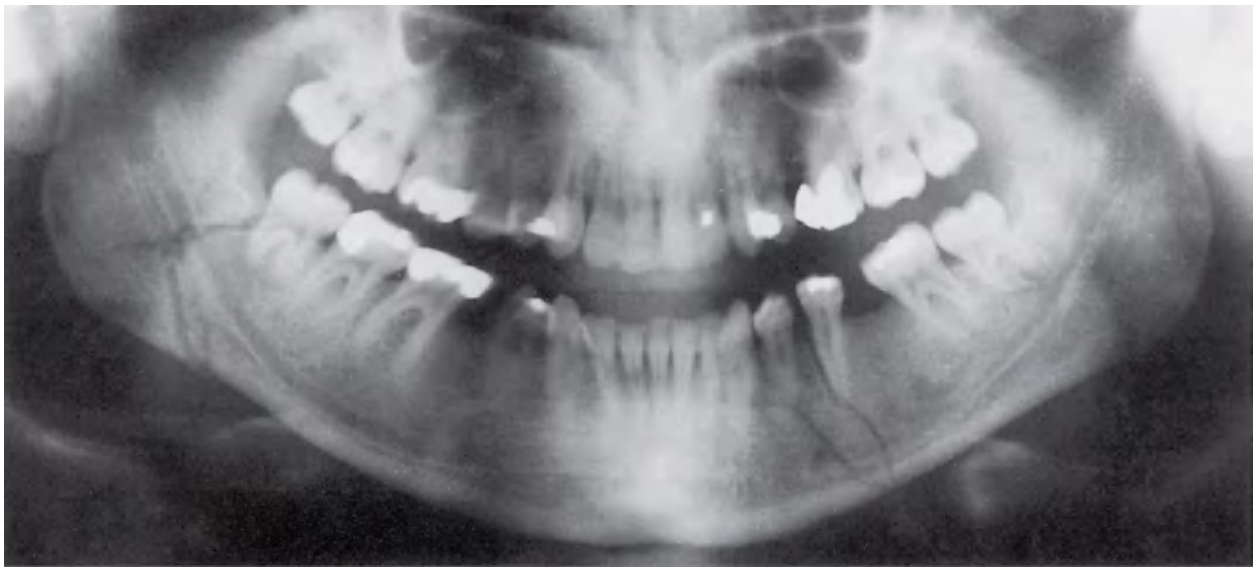
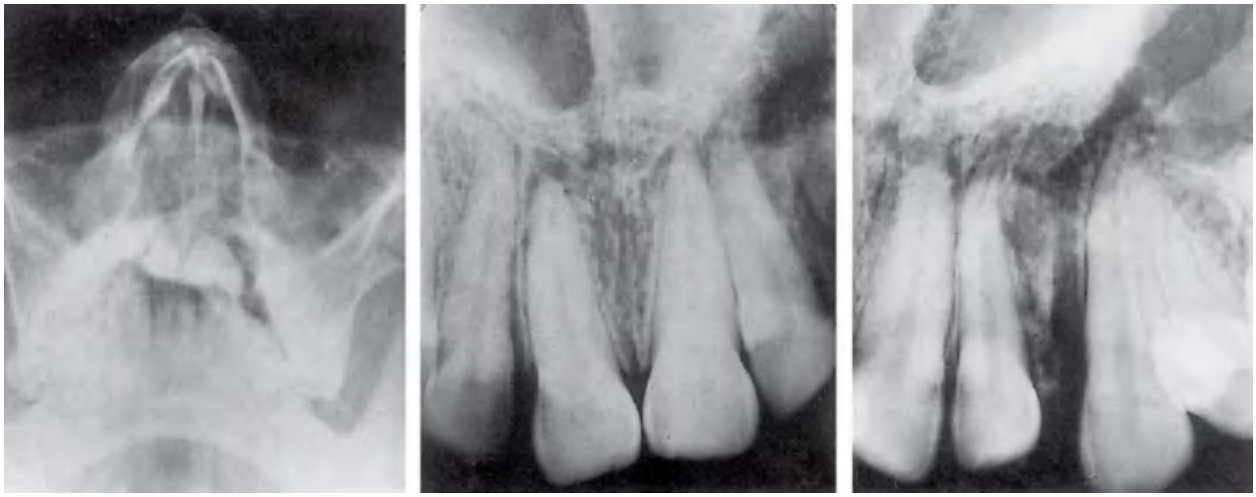
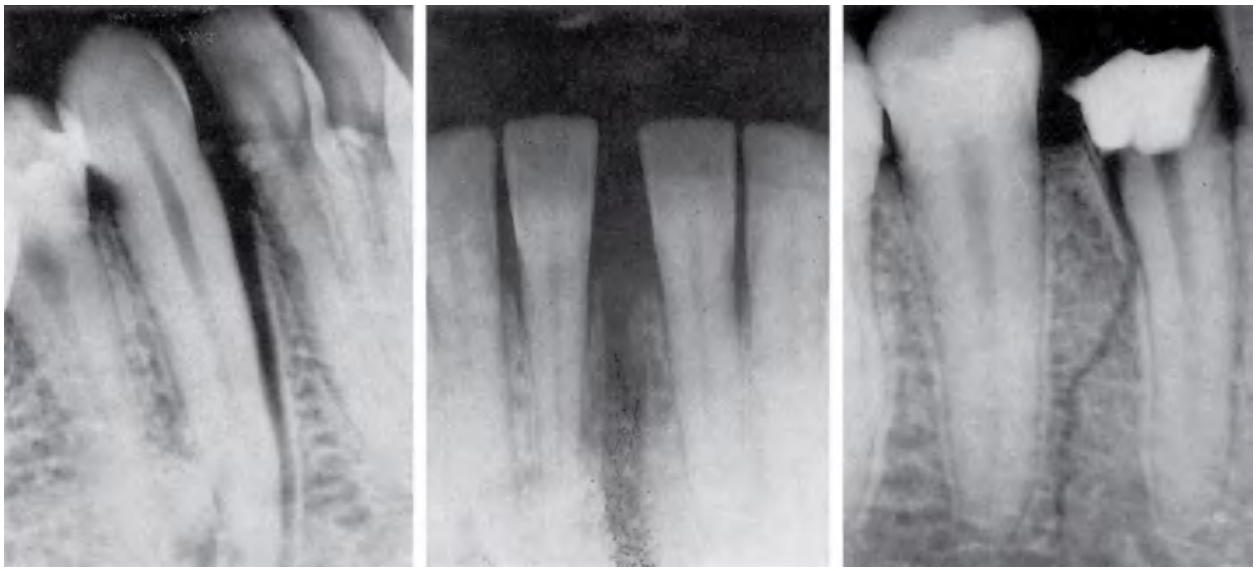


**Fig. 21.12** Fracture lines in the premolar and third molar regions as revealed by panoramic technique.



A B C

**Fig. 21.13** Fracture of the maxilla involving incisors, premolars, and molars. A. Only few details are depicted on extraoral radiograph. B. and C. Intraoral radiographs reveal the relationship between involved teeth and fracture lines.



A B C

**Fig. 21.14** Different courses of jaw fracture line following the periodontal ligament space. A. Jaw fracture passing through the periodontal ligament. B. Jaw fracture passing through the middle of the interdental septum. C. Combination of periodontal and septal involvement.

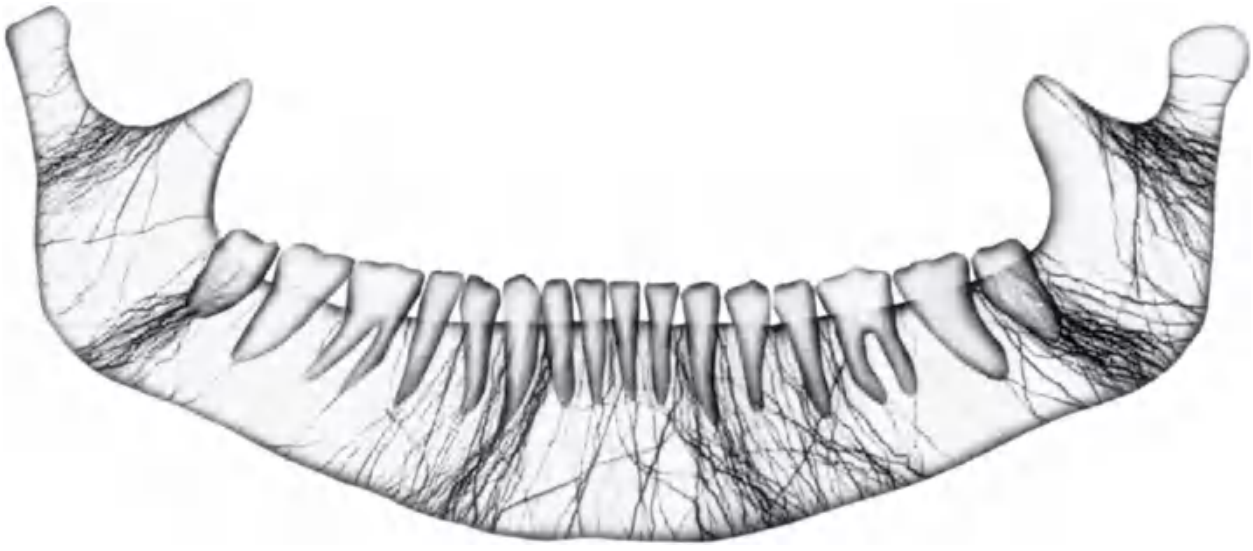


**Fig. 21.15** Location and course of fracture lines in 225 patients with mandibular fractures due to fights. The course of the fracture lines was determined from panoramic radiographs. Note that the majority of the fracture lines are situated in the so-called weak areas of the mandible, the subcondylar, third molar, and canine regions. Fractures of the body of the mandible usually run obliquely downwards and backwards to the base of the mandible. Reproduced from (13) with permission.

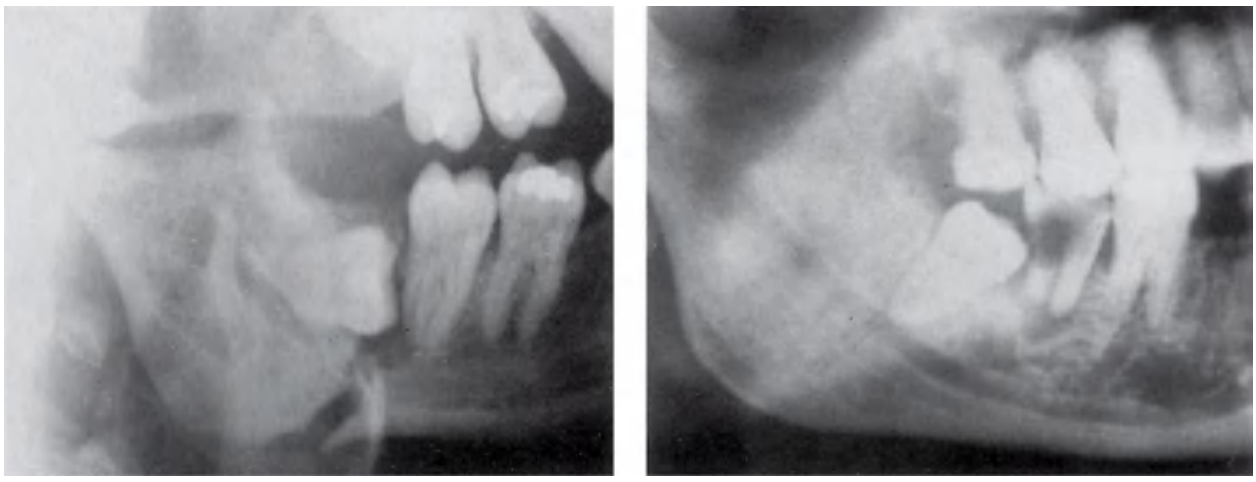


**Fig. 21.16** Fracture lines in the lower third molar region following the line of least resistance as determined by impacted third molars.

the position of the lower third molar generally determines the course of the fracture line (13, 114).

It should be remembered that two lines will be seen radiographically if the central beam is not parallel to the plane of fracture, as the fracture lines of both outer and inner cortical bone plates will be depicted (114) (Figs 21.17 and 21.18).

## Pathology

Most fractures of the supporting bone represent a complex injury where there is damage not only to supporting bone but also to the pulp, periodontal ligament and the gingiva (Fig. 21.19). Due to this multiplicity of involved tissues, various types of healing complications can occur, such as loss of supporting bone, root resorption and pulp necrosis.

Adequate knowledge of bony repair after fracture is necessary in order to appreciate the role teeth play in these events. As relatively little information exists on the healing events after jaw fractures, most of our present knowledge is derived from clinical and experimental findings from fractures of the shaft of long bones (18–44) and a few studies in dogs (22–25). The following description of fracture healing is therefore based upon the latter material. Special implications for bone healing in the jaws will be discussed later in this chapter.

The events immediately following fracture include extravasation and clotting of blood exuding from injured vessels. The normal vascular supply to the fracture site is compromised and necrosis of osteocytes is seen in adjacent areas. Organization of the blood clot by formation of granulation tissue begins within the first 24 hours after injury. Its primary function is removal of necrotic or damaged tissue components. The granulation tissue becomes dense connective tissue in which cartilage and fibrocartilage develop, forming a cuff of fibrocartilaginous callus around the fracture site and thereby closes the gap between the fracture edges.

New bone originating from the deeper layers of the periosteum and endosteum is formed some distance from the fracture site. Immature bone then invades the fibrocartilaginous callus and ultimately unites the two fragments, whereupon mineralization of the callus takes place. At the same time, resorptive and remodeling processes make the bone structure on either side of the fracture less dense, a change often observed in follow-up radiographs (26). Subsequently, reorganization of the bony callus occurs, and the immature fibrillar bone is replaced by mature lamellated bone. Eventually, functional reconstruction takes place, i.e. internal reconstruction and resorption of excess bone.

It is presumed that the above-mentioned healing processes also apply to jaw fractures. The presence of a cartilaginous callus has, however, been questioned. In a study of experimental jaw fractures in dogs, no evidence of cartilage formation was found (22), whereas other investigators have found occasional islands of cartilage in animal experiments (23–25, 33, 35) and in human material (26). Finally, it has been shown that mobility of the jaw fragments influences the rate of bony callus formation. When fragments are mobile, more time elapses before bony bridging is seen as compared to immobilized fractures (24).

The influence of the presence of teeth upon fracture healing has been elucidated primarily by animal experiments (22, 28–31). Experimental jaw fractures in dogs, with teeth present along the fracture line, have revealed formation of granulation tissue which resorbs the interdental bone and adjacent root surfaces (22). Examination of human teeth located along the fracture line, however, has shown evidence of repair of resorption areas (45, 46).

Figs 21.20 and 21.21 show the early histologic healing events following fracture of the vestibular socket wall and alveolar process.

## Treatment

### Fractures of the socket wall

Fractures of the socket wall are usually associated with dislocation of teeth. Thus, the first step after administration of local anesthesia is to reposition the displaced teeth.

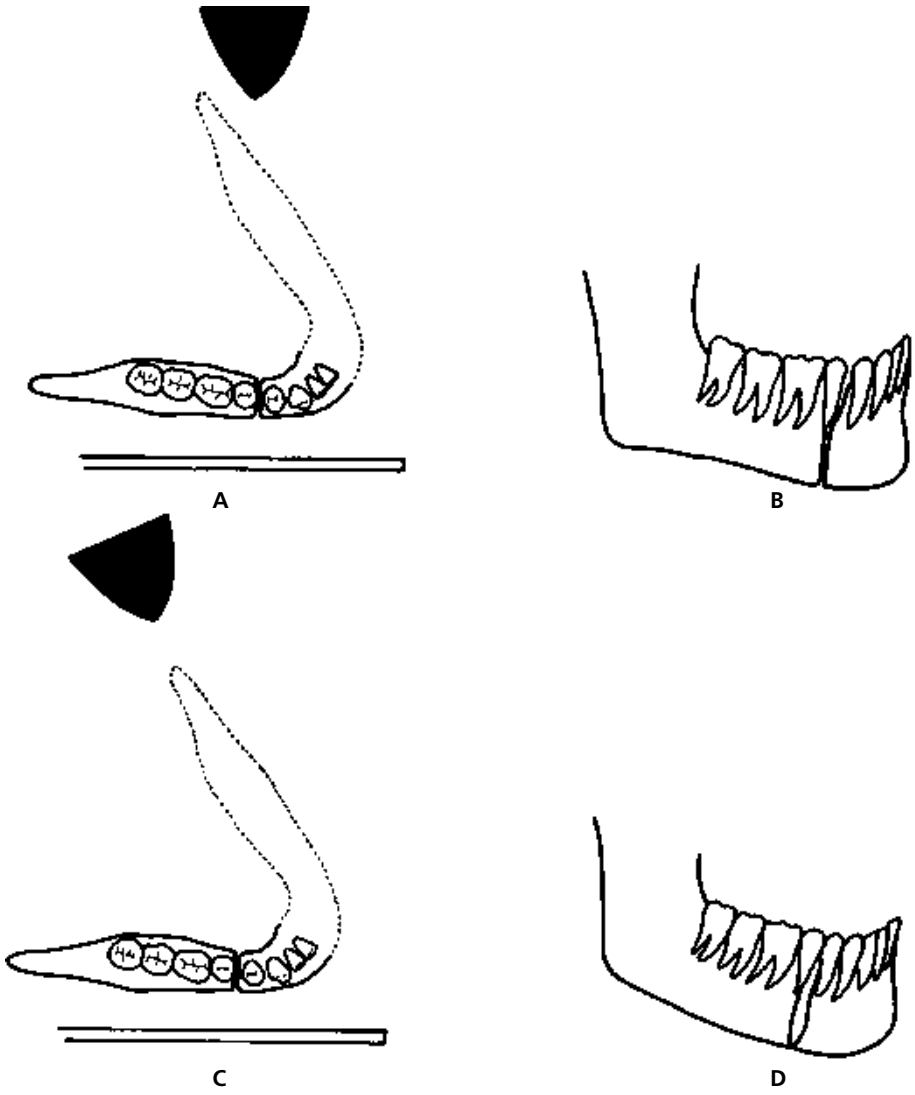



**Fig. 21.17** Diagrams illustrating the difference in the radiographic appearance of a single fracture line depending on the angle of the central beam. A and B. Central beam parallel to the fracture plane results in a single fracture line. C and D. A more posterior exposure results in projection of the fracture of the outer and inner cortical bone plate as separate lines.

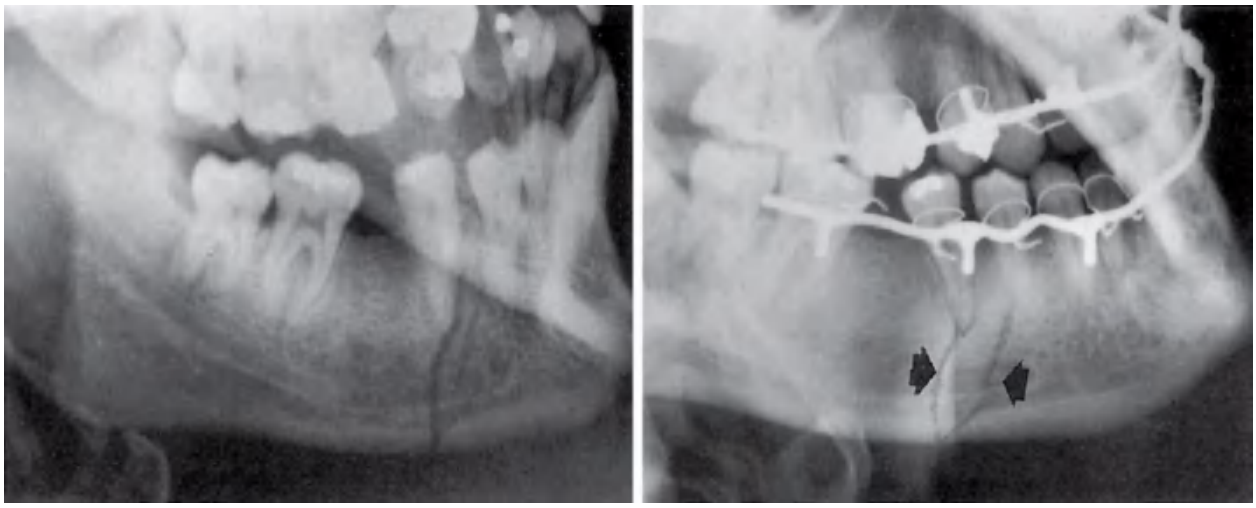


**Fig. 21.18** Radiographs illustrating change in position of the fracture lines when the angle of the central beam is altered. A. Central ray almost parallel to the fracture plane. B. A change in the projection angle results in two distinct fracture lines (arrows).

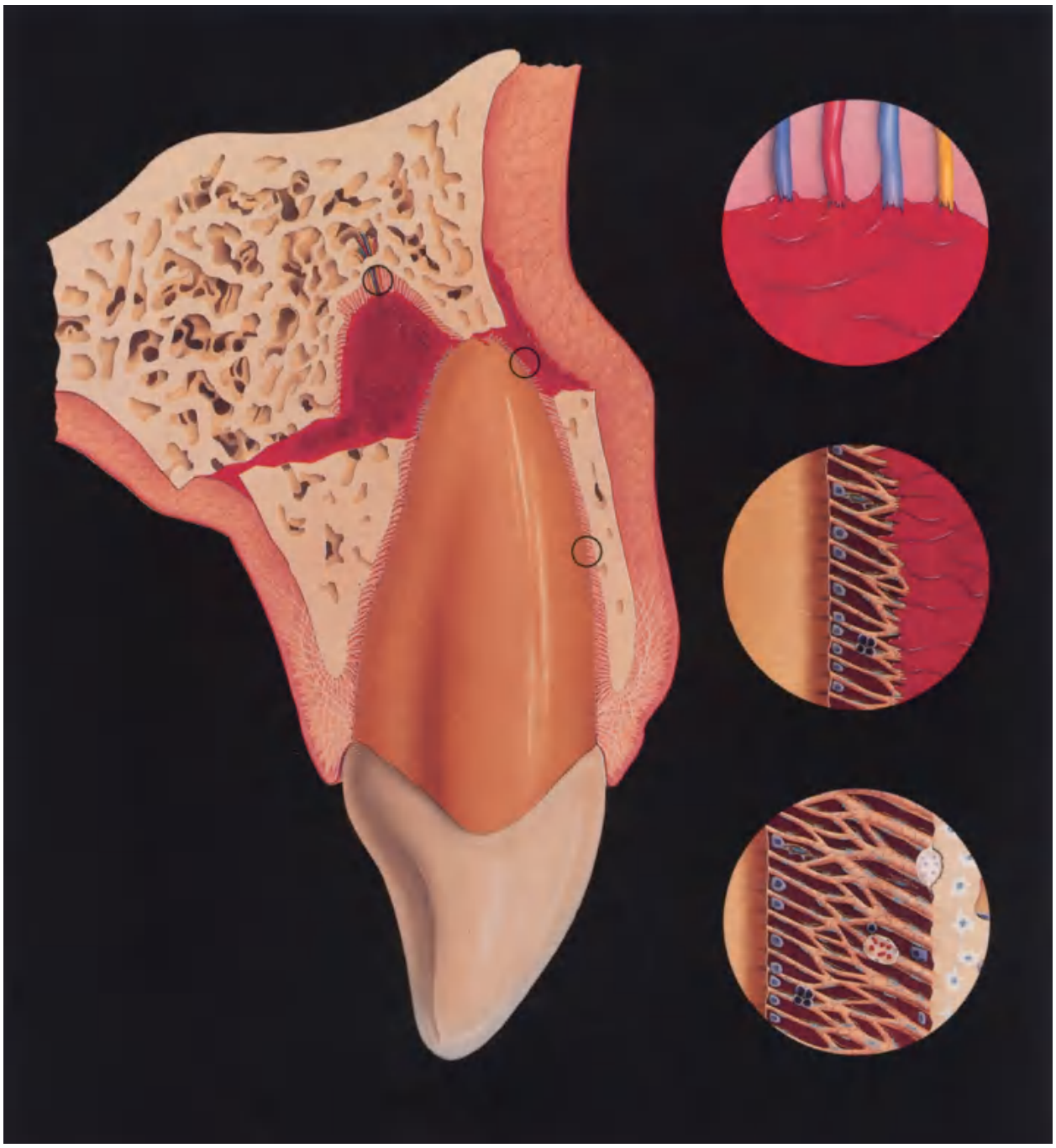


**Fig. 21.19** Fracture of the alveolar process. Both the PDL and the neurovascular supply to the pulp are severed.

Reduction in these cases is similar to that after lateral luxation (see Chapter 18); that is, by disengaging the apices from the vestibular bone plate. This is done by simultaneous digital pressure in an incisal direction over the apical area and in a facial direction at the lingual aspect of the crown. This maneuver will usually free the apices and permit repositioning of the fragment. The socket wall is repositioned at the same time. In case of open comminuted fractures, it may be necessary to remove loose fragments which are not adherent to the periosteum. Clinical experience has shown that, despite removal of the entire vestibular bone plate, there is still enough structural support to ensure adequate stability of the teeth. After reduction of displaced teeth and bone fractures, lacerations of soft tissues should be sutured. Although it is tempting to suture immediately, suturing of soft tissue wounds should be left to last, as suturing early in the treatment procedure limits access for repositioning procedures. Splinting is carried out according to the principles outlined in Chapter 39.

Due to rapid bony healing in children, most fractures of the alveolar socket wall involving the primary dentition do not require splinting. In these cases, the parents should be instructed to restrict nourishment to a soft diet during the first 2 weeks after injury.

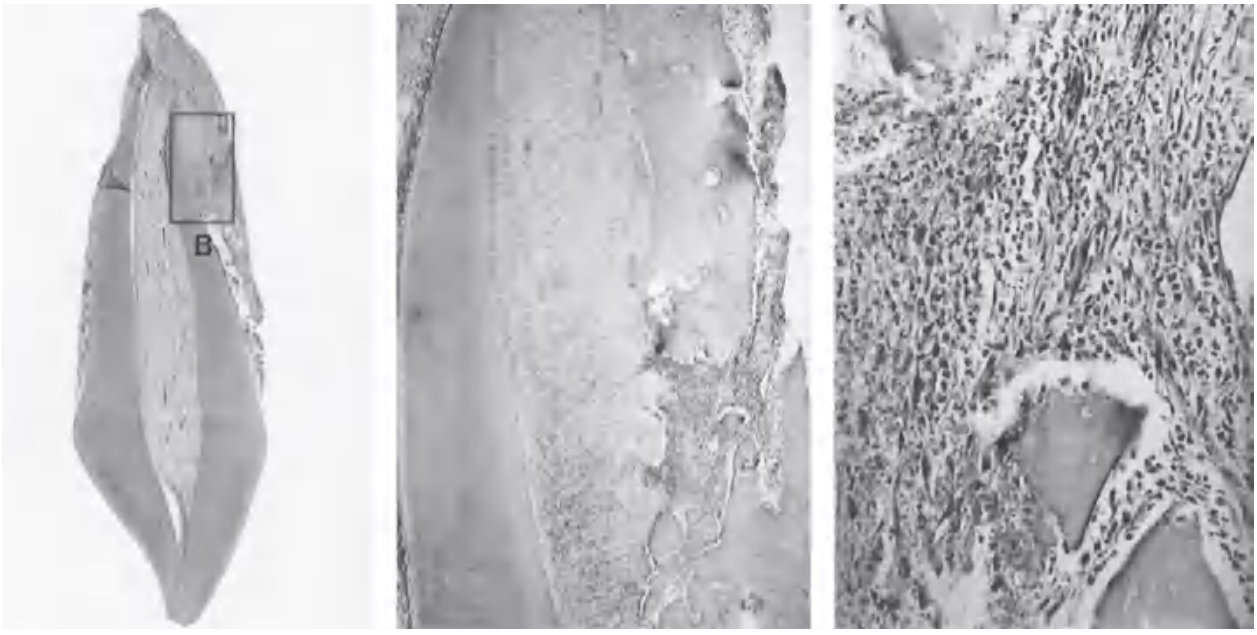



**Fig. 21.20** Early histologic changes following fracture of the facial socket wall in a $1^{1}/_{2}$-year-old child. A. Low-power view of sectioned incisor, removed 5 days after injury. ×5. B and C. Higher magnification of the fracture area. The blood clot has been organized by granulation tissue. ×30 and ×195.

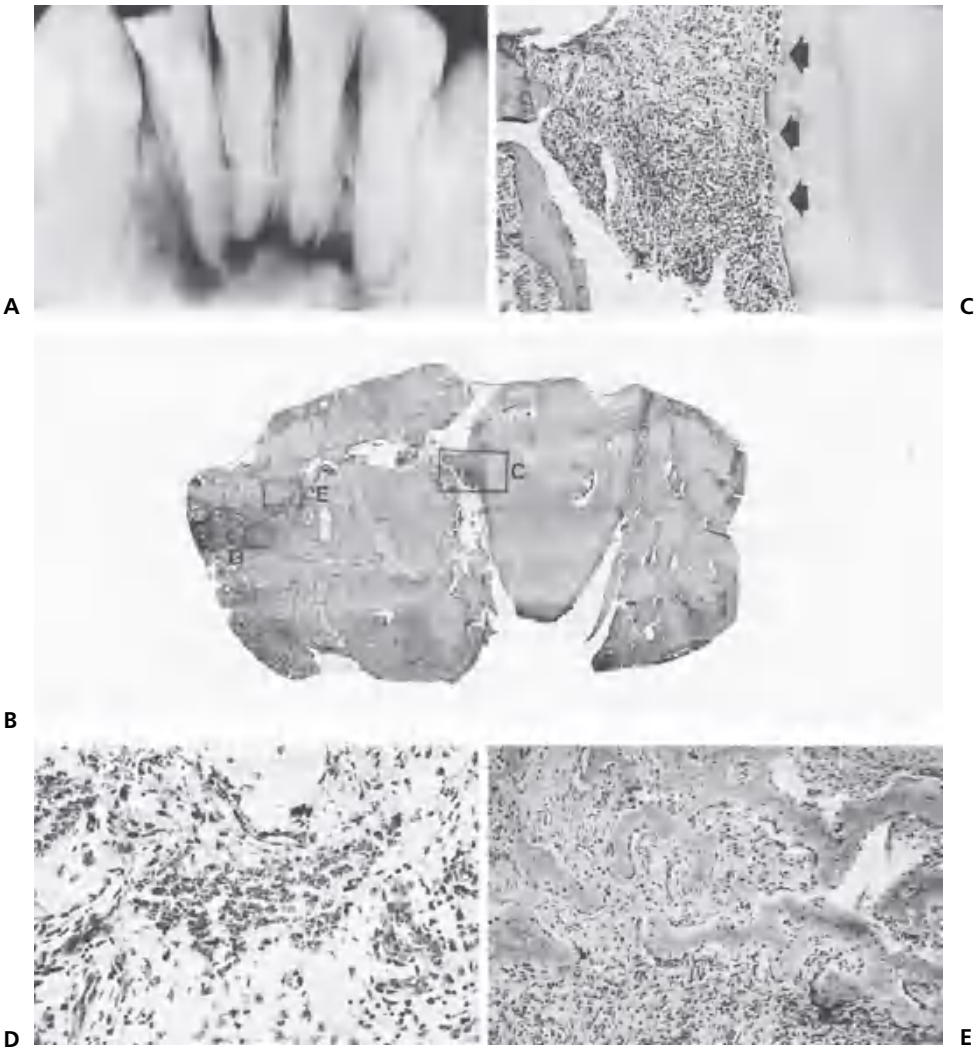



**Fig. 21.21** Healing events following a fracture of the alveolar process in the mandibular incisor region. A. Radiograph taken at time of injury. B. Surgical specimen including apex of left lateral incisor removed 18 days after trauma. ×10. C. Superficial resorption of the mesial aspect of the root (arrows). ×30. D. Remnants of coagulum. ×195. E. Developing young immature bone bridging the fracture line. ×75.

## Fractures of the alveolar process

Treatment of fractures of the alveolar process includes reduction and immobilization (4, 6, 47–50, 76, 77, 159, 165) (Fig. 21.22). After administration of local anesthesia, the alveolar fragment is repositioned with digital pressure. In this type of fracture, apices of involved teeth can often be locked in position by the vestibular bone plate. Reduction in these cases follows the principles mentioned under fractures of the alveolar socket wall.

Splinting of alveolar fractures can be achieved by means of an acid-etch/resin splint or arch bars. Intermaxillary fixation is not required provided that a stable splint is used (Fig. 21.22). A fixation period of 4 weeks is usually recommended. However, due to more rapid healing in children, this period can be reduced to 3 weeks.

Teeth in a loose alveolar fragment might be doomed to extraction due to marginal or periapical inflammation. However, these extractions should generally be postponed until bony healing has stabilized the fragment (Fig. 21.23), otherwise the entire alveolar fragment might be inadvertently removed with the teeth. Again, regarding treatment sequence, associated soft tissue lacerations should be sutured last in order to allow access for intraoral manipulation.

Treatment of alveolar fractures in children offers special problems due to lack of sufficient teeth for splinting procedures. In most cases where the fragment can be reduced into a stable position, one can omit the splinting procedure. In such cases, nourishment should be restricted to soft or liquid foods. Alternatively, an acrylic splint fastened with permandibular wires can be a solution (see Chapter 39).

A recent multivariate analysis of the outcome of 223 teeth involved in alveolar fractures showed a number of significant relations. Completed root development and age >30 years showed a significant higher risk of pulp necrosis. Fracture site in relation to apices also showed a higher frequency of pulp necrosis of the affected teeth (Fig. 21.26). Horizontal displacement of more than 2 mm resulted in an increased risk of pulp necrosis. Finally, incomplete reposition also resulted in an increasing risk of pulp necrosis (165) (Fig. 21.27).

# Fractures of the mandible or maxilla

Jaw fracture and especially mandibular fracture are often combined with dental injuries (118).

The management of fractures of the mandible or maxilla involves many procedures beyond the scope of this book. In this regard, only teeth involved in the fracture area will be considered. The reader is otherwise referred to standard textbooks on this subject (53, 86, 87).

Treatment of jaw fractures in children with developing teeth in the line of fracture follows the given general principles, i.e. exact repositioning and usually intermaxillary fixation. A review survey article has been published by Hard and von Arx (119). It is important that developing permanent teeth in the fracture line be preserved. The only exception would be in cases of infection along the fracture line being maintained by infected tooth germs (54, 55, 120). Moreover, in case of open reduction, screws should be place at a distance from developing teeth (120–122) (see Chapter 23).

Treatment of jaw fractures in adults with teeth in the fracture line is a controversial issue (68) and has been the subject of a review article (88). Particularly prior to the antibiotic era, it was customary to extract all teeth in the fracture line. However, studies have shown that this approach does not reduce the frequency of complications, which has gradually led to more conservative treatment.

## Teeth in the line of fracture

In the past, a number of clinical studies have appeared concerning the effect of various factors related to healing complications when teeth were involved in the line of fracture (56–63, 68–74, 78–82, 123, 150). This topic has recently been debated in a review article (162). However, all of these studies are retrospective, implying a possible bias in the evaluation of extraction therapy and the use of antibiotics. The resultant conclusions should therefore be regarded with extreme caution. In the following, a survey of factors related to fracture healing complications is presented.

The presence of teeth in the fracture line apparently represents an increased risk for infection compared to a fracture where no teeth are involved (68, 89).

### *Type of teeth involved*

The type of tooth involved also appears to be of importance. Thus, multirooted teeth appear to enhance the risk of complications compared to single-rooted teeth (68, 70) (Table 21.1).

### *Eruption status*

The most frequent tooth involved in jaw fractures appears to be the third molar. The eruption status of this tooth (especially semi-erupted) appears to be significantly related to the occurrence of healing complications. Thus, complications are usually only seen with semi-erupted mandibular third molars; whereas fully erupted or impacted third molars rarely develop complications along the line of fracture (91) (Fig. 21.24).

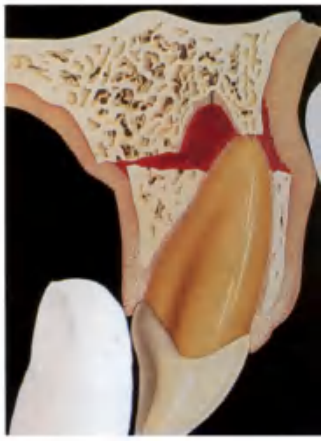
### *Extraction of teeth*

In general, no beneficial effect has been demonstrated following the extraction of teeth in the line of fracture. In fact, a few studies have shown an increased morbidity (57–59, 79, 124) but most studies no effect of extraction (56, 77, 78, 89, 90, 92, 113, 125, 126, 150). However, none of the cited studies were designed as randomized investigations with respect to extraction of teeth in the line of fracture. Only one randomized study has been published dealing with the effect of extracting the third molar. In this study it was found that only removal of semiimpacted molars had en advantage in avoiding fracture healing complications (91) (Fig. 21.24).

**Fig. 21.22 Treatment of an alveolar fracture**
This 21-year-old woman suffered a fracture of the maxillary alveolar process including the lateral and central incisors.

**Anesthetizing the traumatized region**
An infraorbital block and infiltration to the incisive canal are necessary prior to repositioning.

**Repositioning**
With firm finger pressure to the apical region, the apices are disengaged. If this is not possible, the fragment must be moved in a coronal and palatal direction with the help of a forceps.



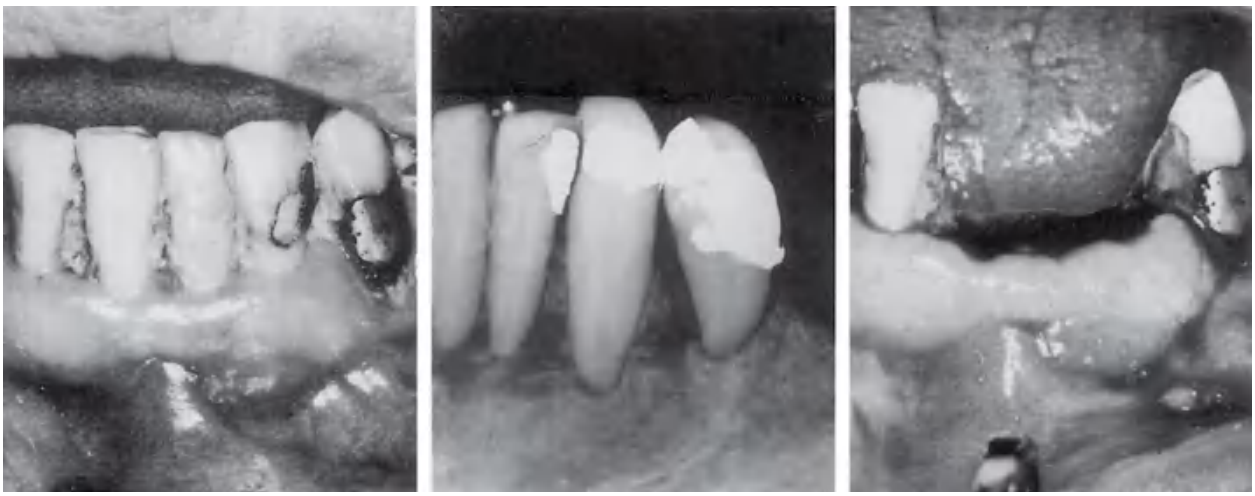
**Verifying repositioning**
Occlusion is checked, and a radiograph taken to verify adequate repositioning.

**Splinting**
The incisal one-third of the labial aspect of injured and adjacent teeth are acid-etched (30 seconds) with phosphoric acid gel. The etchant is removed with a 20-second water spray. The labial enamel is dried with compressed air, revealing the mat, etched surface, whereafter the splinting material is applied. During polymerization of the splinting material, the patient occludes in order to ensure correct repositioning of the alveolar segment.

**Finished splint**
The splint does not contact the gingiva so that optimal oral hygiene can be maintained.

**Removing the splint**
After 4 weeks the splint is removed with a scaler or fissure bur.

**Controlling pulpal sensitivity**
Pulpal revascularization is monitored after 3 months. Due to a lack of response to pulp testing as well as a slight apical radiolucency, pulp necrosis is suspected.



A B C

**Fig. 21.23** A and B. Alveolar fracture in left incisor and canine region associated with marginal periodontitis. It was decided to splint the fracture and postpone extraction of lower incisors for 6 weeks. C. Lower incisors extracted without damage to the alveolar process.

**Table 21.1** Frequency of complicated fracture healing in adults with teeth preserved along the fracture line.

| Examiner | No. of cases | No. of complications (infection) |
|---|---|---|
| Götte (57) 1959 | 178** | 30 (17%) |
| Andrä & Sonnenburg (56) 1966 | 264** | 40 (15%) |
| Andrä & Sonnenburg (56) 1966 | 139*+ | 7 (5%) |
| Müller (68) 1968 | 118++ | 23 (19%) |
| Neal et al. (79) 1978 | 132* | 39 (29%) |
| Rink & Stoehr (91) 1978 | 99* | 13 (13%) |
| Kahnberg & Ridell (81) 1979 | 132* | 10 (8%) |
| Choung et al. (89) 1983 | 152** | 4 (3%) |
| de Amaratunga (92) 1987 | 124** | 6 (5%) |
| Gerbino et al. (124) 1997 | 78 | 8 (10%) |
| Krenkel & Grunert (152) 1986 | 36 | 0 (0%) |
| Marker et al. (123) 1994 | 57*++ | 2 (4%) |
| Rubin et al. (125) 1990 | 69*++ | 16 (23%) |
| Ellis (150) 2002 | 258 | (19.5)% |

*Antibiotics given in all cases.
**Antibiotics given in some cases.
+Single-rooted teeth.
++Multi-rooted teeth.

Furthermore, there was no difference in the complication rate of completely impacted teeth, whether open or closed repositioning (114).

### *Effect of antibiotics*

In cases of conservatively or surgically treated mandibular fractures, prospective randomized studies have shown that antibiotic treatment given from 30 min before surgery and up to 1 day afterwards significantly decreased the frequency of infection (93–95, 126, 127). However, an increase in antibiotic coverage for more than 1 day did not decrease the rate of infection (94, 95, 127, 128).

The effect of antibiotics on infection in the line of fracture in dentulous parts of the jaws is presently a subject of debate. If teeth are present in the line of fracture, there is an uncertain (56, 57) or no value of antibiotic therapy (91), whereas one study showed a positive effect (60).

If teeth were extracted, the rate of infection was unaffected by antibiotic therapy in one investigation (91) and found to be reduced in another (56). In one study, it was found that a positive effect was only related to multirooted teeth (68).

### *Delay in treatment*

Delay in treatment seems to play a questionable role in the frequency of healing complications. In case of alveolar fractures, treatment delay appears to be related to pulp necrosis and root resorption (6). Concerning jaw fractures, four studies showed no significant difference between immediate and delayed treatment (126, 154–156). One study showed a preference for healing for cases treated within 3 days (153), whereas another study indicated that treatment between 3 and 5 days after injury was optimal, with the lowest rate of complications (97). In conclusion, there is presently no strong evidence for either acute or delayed treatment of mandibular fractures in order to minimize healing complications; new studies, including a number of cases treated on an acute basis, are needed (157).

### *Type of splinting*

An increase in the rate of infection was found following rigid internal fixation (i.e. surgical repositioning and osteosynthesis using wire or plates) in fractures with teeth in the fracture line compared to similar fractures treated by closed reduction and conventional intermaxillary fixation (98–100).

Another factor which influences fracture healing is rigid arch bar fixation compared to a flexible wire splint.

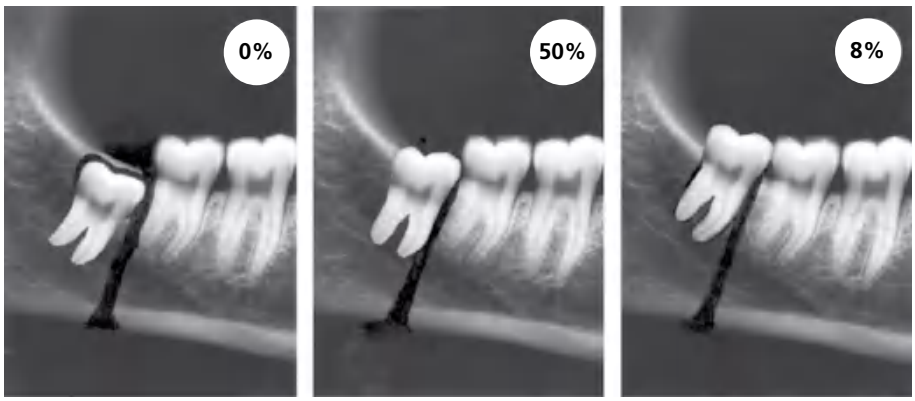



**Fig. 21.24** The infection risk in the fracture bone is strongly related to the eruption status of the third molar. The frequency is indicated. Reproduced from (91) with permission.

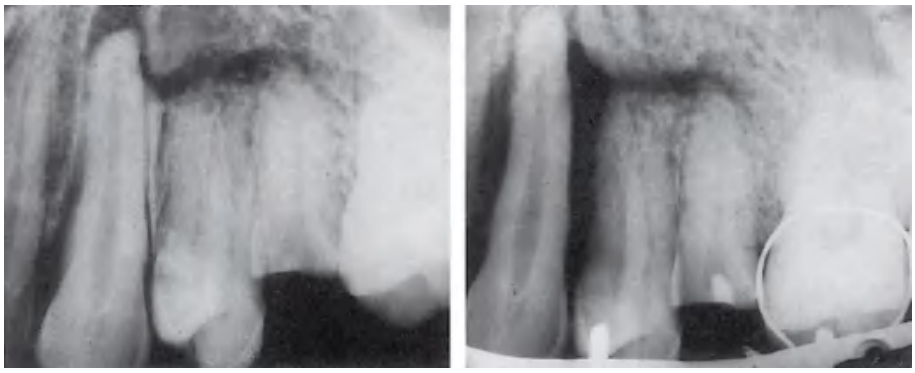



**Fig. 21.25** Persistent infection in the fracture line of an alveolar fracture involving canine and premolars. A. Condition immediately after injury. B. Radiograph 10 weeks later reveals marked bone destruction in the fracture area.

By eliminating movement of the fragments, seepage of saliva along the fracture line is prevented, which thus decreases the possibility of secondary infection (57). Finally, interdental wiring in jaw fractures does not appear to lead to adverse effects in the periodontium (138).

In conclusion, teeth present in the line of fracture should generally be preserved, as they can later serve as functional units. Moreover, their removal may further traumatize the fracture site and lead to dislocation of the fragment (59, 101). Exceptions to this approach would be teeth with crown-root fractures, semi erupted third molars and teeth with severe marginal or apical breakdown whose fate is already compromised. Teeth with pulp necrosis and apical breakdown can possibly be saved by endodontic therapy. This also applies to teeth with complicated crown and root fractures. Treatment of these injuries follows principles outlined in previous chapters. However, root canal treatment must necessarily be postponed until the intermaxillary fixation has been removed. When pulp exposures due to crown fractures are present, pulp extirpation should be carried out and the root canal provisionally sealed until removal of the intermaxillary fixation allows completion of therapy. Root fractured teeth are stabilized with splints.

If inflammation occurs, the treatment of choice is antibiotic therapy, possibly together with extraction of teeth involved in the inflammatory process, or endodontic therapy if pulp necrosis has developed.

### *Open or closed reduction*

A systematic review revealed that open reduction in most studies showed an increase in the risk of fracture line infection (165).

## Prognosis

### Fractures of the socket wall

The immediate course of healing after fracture of the socket wall is usually uneventful; however, later follow-ups can reveal resorption of roots of involved teeth. These events are described for lateral luxated teeth (see Chapters 16 and 18).

### Fractures of the alveolar process

The healing events of alveolar fractures in the permanent dentition are in most cases uneventful; however, in rare cases, sequestration of bone and/or involved teeth can occur (Fig. 21.25). Careful follow-up is mandatory in order to register later pulp necrosis and periapical inflammation (5, 6).

**Table 21.2** Frequency of late dental complications following fractures of the alveolar process.

| | No. of cases | No. of involved teeth | Frequency of pulp necrosis | Frequency of pulp canal obliteration | Frequency of progressive root resorption | Frequency of loss of marginal supporting bone |
|---|---|---|---|---|---|---|
| Andreasen (6) 1970 | 29 | 71 | 75% | 15% | 11% | 13% |
| Krenkel & Grunert (152) 1986 | 23 | 73 | 63% | 11% | 6% | 6% |
| Lauridsen et al. 2015 (167) | 9 | 223 | 56%* | 13% | 5% | 3% |

*Estimated risk after 10 years

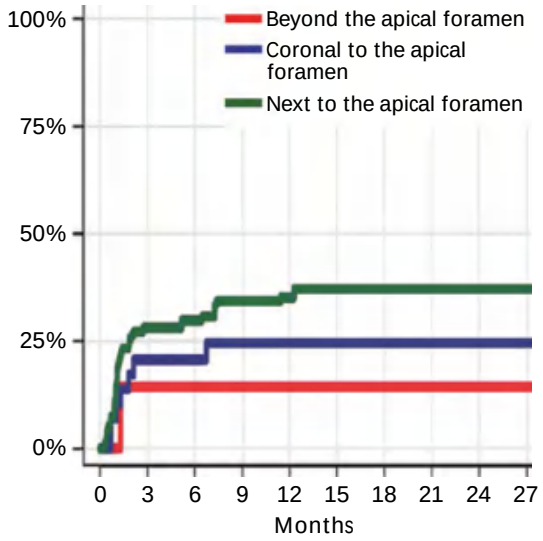



**Fig. 21.26** The risk of pulp necrosis in relation to the location of the horizontal part of the fracture for mature teeth with complete repositioning Reproduced from (160) with permission.

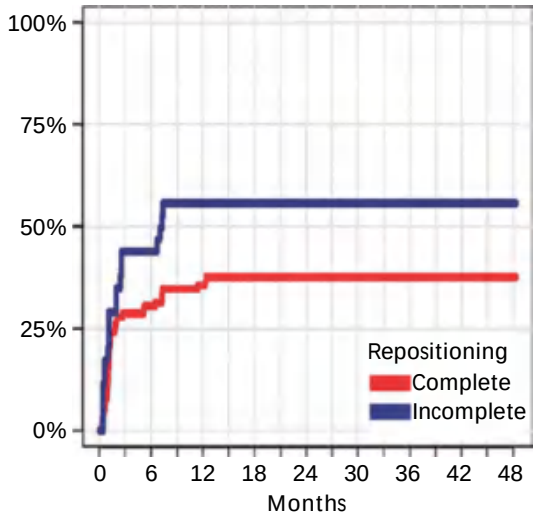



**Fig. 21.27** The risk of pulp necrosis in relation to the degree of repositioning for mature teeth where the fracture was located in relation to the apex of the tooth. Reproduced from (160) with permission.

Such complications are rather frequent and apparently not related to the time interval between injury and fixation (160).

Apart from pulp necrosis, pulp canal obliteration, root resorption (Table 21.2) and loss of supporting bone may occur (160).

The risk of pulp necrosis appears to be related to the location of the horizontal part of the fracture in relation to the apical foramen and how optimal the reposition has been (16) (Figs 21.26 and 21.27).

Apart from pulp necrosis also root resorption and loss of marginal bone may occur (Figs 21.28 and 21.29).

Concerning the prognosis of alveolar fractures in the primary dentition, it has been found that root development of preserved primary teeth is often arrested (51).

## Fractures of the mandible or maxilla in children

### Inflammation at the line of fracture

Fractures of the mandible or maxilla in children with developing teeth along the fracture line are seldom complicated by inflammation. It appears from the studies listed in Table 21.3 that frequencies of complications were found to range from 10 to 18%. The clinical picture of inflammation in the fracture area is characterized by swelling and abscess formation. Draining fistulae may also develop, as well as immediate or protracted sequestration of involved tooth germs (65).

When inflammation does occur, antibiotic therapy is the treatment of choice. Surgical removal of involved teeth may be indicated if radiographs reveal infected tooth germs in the fracture area (64–67). In such cases, osteolytic changes will be found in the fracture area, with disappearance of distinct outlines of the dental crypts of the involved teeth. It is conjectured from animal experiments (27) and human data (65) that infected tooth germs in these cases are responsible for protracted inflammation.

### Disturbances in the developing dentition

Another problem which should be considered when treating jaw fractures in children is odontogenic disturbances in involved developing teeth (see Chapter 20).

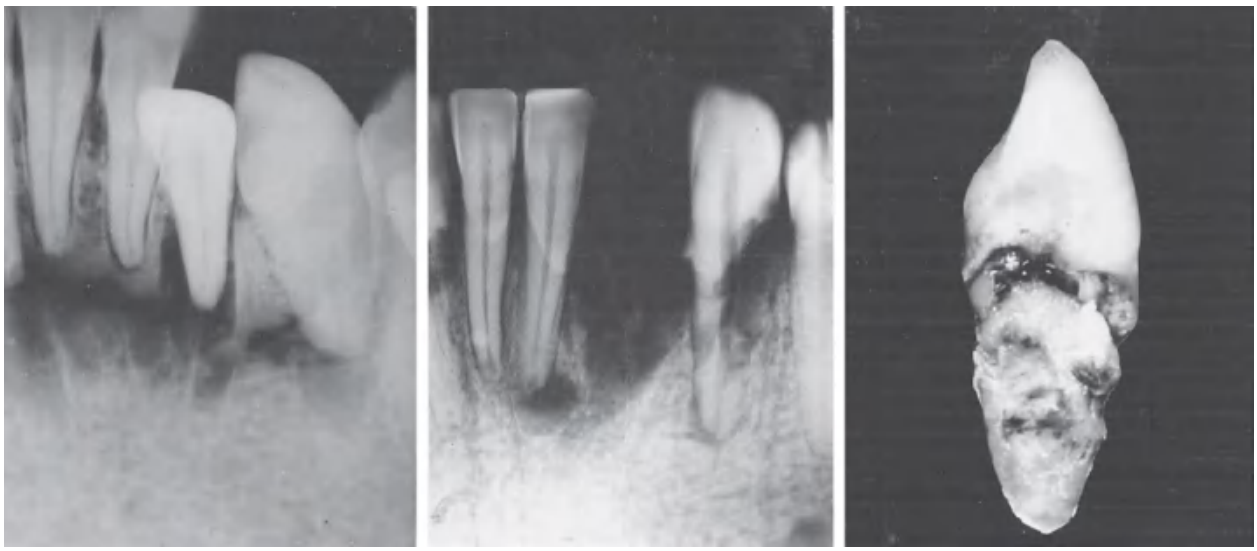



**Fig. 21.28** Root resorption and periapical inflammation after an alveolar fracture involving incisors and left canine. A. Condition after injury. B. Re-examination 3 years after injury reveals periapical inflammation as well as infection-related root resorption. C. Clinical view of extracted canine. Marked resorption areas present on root surface. Reproduced from (6) with permission.

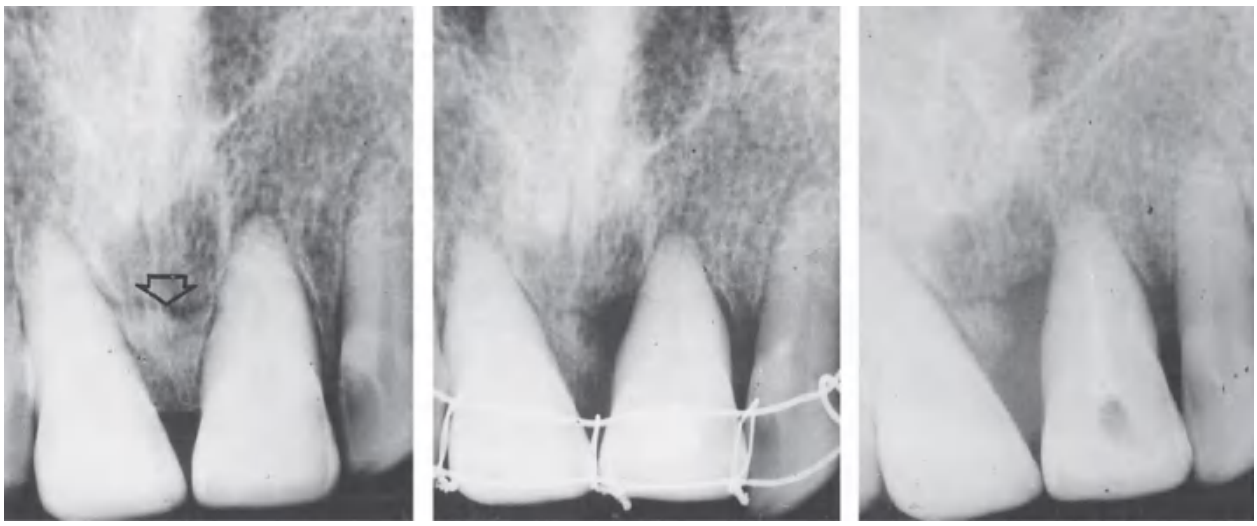



**Fig. 21.29** Loss of marginal supporting bone following alveolar fracture in the central incisor region. Horizontal fracture line is evident on radiograph taken at time of injury (arrow). At follow-up, loss of marginal supporting bone is evident around left central incisor. Reproduced from (6) with permission.

**Table 21.3** Frequency of complicated fracture healing in children with developing permanent teeth involved in the fracture line.

| Examiner | No. of cases | No. of complications (inflammation) |
|---|---|---|
| Lenstrup (14) 1955 | 22 | 4 (18%) |
| Taatz (64) 1962 | 29 | 3 (10%) |
| Koenig et al. (139) 1994 | 30 | 0 (0%) |
| Suei et al. (162) 2006 | 28 | 1 (3%) |

## Fractures of the mandible or maxilla in adults

### Fibrous union

In a recent study of 714 mandibular fractures, fibrous union was found in 27 patients (3.8%); and was apparently related to the presence of teeth in the fracture line. However, a more detailed analysis of this material revealed no

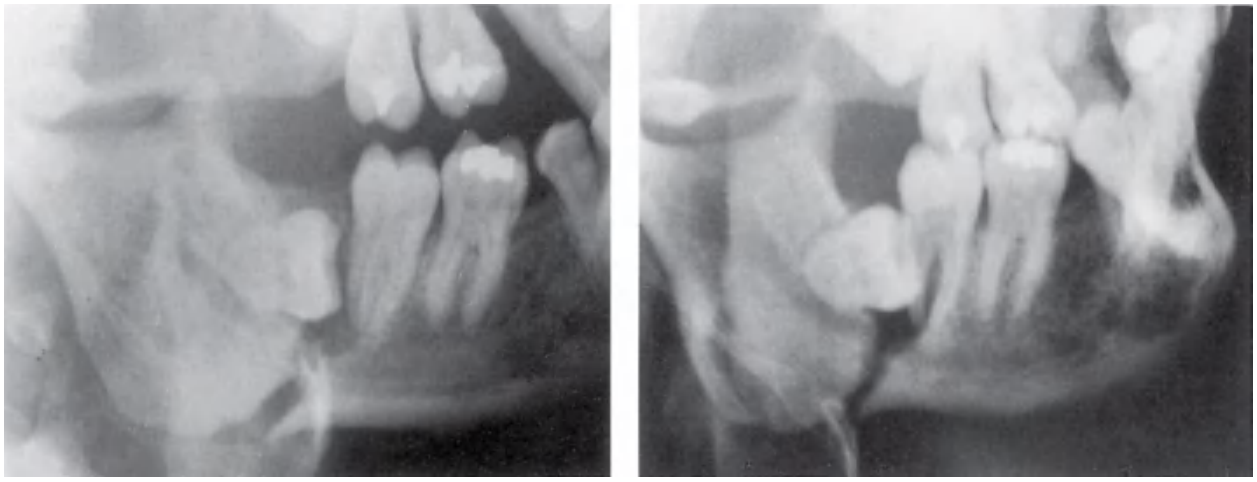



**Fig. 21.30** Radiograph illustrating delayed fracture healing due to inflammation maintained by an impacted third molar. Six months later, bone healing has not occurred.

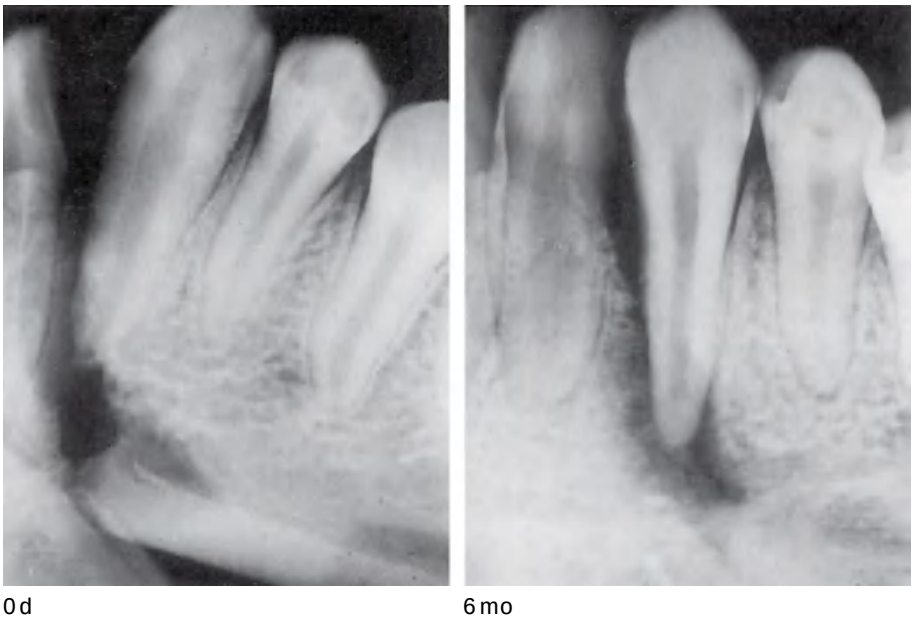



**Fig. 21.31** Pulp necrosis and incomplete healing of marginal periodontium following jaw fracture in the canine region. Note that the fracture line involves the apex of the canine. Follow-up examination after 6 months revealed incomplete healing of the marginal periodontium as well as pulp necrosis with periapical involvement. Reproduced from (69) with permission.

relation to fracture sites where teeth had been removed or maintained (145).

## Inflammation at the line of fracture

Studies of jaw fractures in adults have shown that the frequency of inflammatory complications along the line of fracture when teeth are preserved ranges from 3 to 29% (Table 21.1, Fig. 21.30). A tooth positioned along the line of fracture can be the source of inflammation due to pulp necrosis from compromised circulation to the pulpal tissues or already established infection in the pulp which can spread to the fracture site (Fig. 21.31). This finding is supported by the finding that pulp necrosis in involved teeth is common when a fracture directly involves the apical region (69, 82). Infection can also occur along the denuded root surface when a fracture directly involves the alveolar socket (26).

## Pulpal healing and pulp necrosis

Teeth preserved along a fracture line should be carefully followed in order to detect later pulp necrosis (58, 69, 74, 81, 102, 140, 142) (Table 21.4). In interpreting pulpal sensibility of involved teeth, one should remember that reactions may be temporarily decreased and later return to normal (75, 81, 103). It has been found that pulp necrosis is primarily associated with the relationship of the apices of involved teeth to the fracture line (69, 82, 83, 102). Thus, if the apex is exposed by the fracture line, the risk of pulp necrosis is increased (81, 141, 146) (Fig. 21.31). Moreover, fractures treated more than 48 hours after injury show an increased incidence of later pulp necrosis (69, 82, 102). Finally, the use of open repositioning and plates (compared to closed repositioning and intermaxillary fixation) resulted in significantly more pulp necrosis, also in adjacent teeth (147).

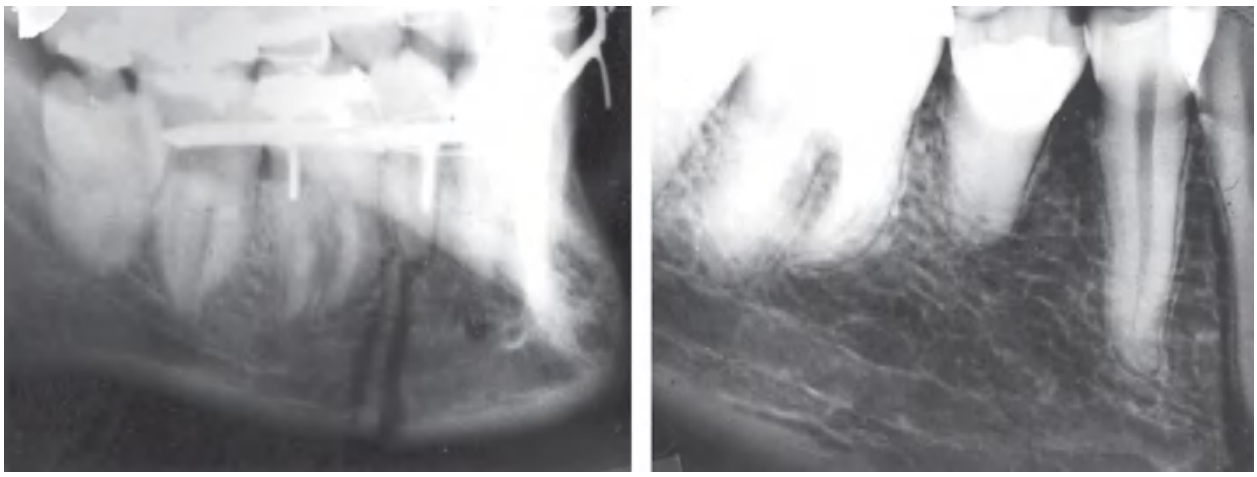



**Fig. 21.32** Obliteration of pulp canal in a second premolar following jaw fracture. The fracture line involves the apical area. At follow-up a marked pulp canal obliteration is evident. Reproduced from (69) with permission.

**Table 21.4** Frequency of late dental complications following jaw fractures.

| Examiner | No. of cases | No. of involved teeth | Frequency of pulp necrosis | Frequency of pulp canal obliteration | Frequency of progressive root resorption | Frequency of loss of marginal supporting bone |
|---|---|---|---|---|---|---|
| Roed-Petersen & Andreasen (69) 1970 | 68 | 110 | 25% | 5% | 0% | 12% |
| Ridell & Astrand (82) 1971 | 84 | 142 | 5% | 2% | 1% | 11% |
| Kahnberg & Ridell (81) 1979 | 132 | 185 | 14% | – | 3% | 12% |
| Krenkel & Grunert (140) 1987 | 21 | 50 | 23% | 2% | 2% | 2% |
| Oikarinen et al. (141) 1990 | 45 | 47 | 38% | 24% | 4% | 18% |
| Berg & Pape (142) 1992 | 41 | 59 | 22% | | 5% | 12% |
| Thaller & Mabourakh (143) 1994 | 142 | | 9% | | | |

In the follow-up period, obliteration of the root canal is often seen in teeth involved in the fracture, and especially in individuals below 20 years of age (102) (Figs 21.32 and 21.33).

## PDL healing and root resorption

Root resorption following jaw fractures is a rare finding and has been found to affect up to 4% of the teeth involved in the fracture line (69, 81, 87, 140–142) (Table 21.4).

No difference was found in mobility of teeth involved in jaw fractures, whether with conservative or surgical repositioning (148).

## Gingival healing and loss of marginal attachment

Generally speaking periodontal healing is the usual event after a jaw fracture involving interdental bone (149).

In case of arch bar fixation (i.e. Sauer's, Schuchardt's or Erich's arch bar), no significant difference in periodontal health after splinting has been found in a clinical situation (105). Experimentally, a mild periodontitis has been observed during the splinting period (106–108). However, these inflammatory changes appear to be reversed following splint removal (109, 110, 138).

The dental septum and gingiva involved in jaw fractures appear to possess a good healing potential. Complications, such as loss of interdental bone and pocketing, are rare (111).

Loss of marginal bone can be recorded at later follow-up examinations (69, 82, 102). This is found especially among involved canines. Incomplete reduction of displaced fragments appears to be the main cause of loss of marginal bone support (Fig. 21.34), while optimal repositioning will ensure complete periodontal repair (69, 81) (Fig. 21.35). However, a slight long-term increase in mobility of teeth along the line of fracture has been reported (102, 112).

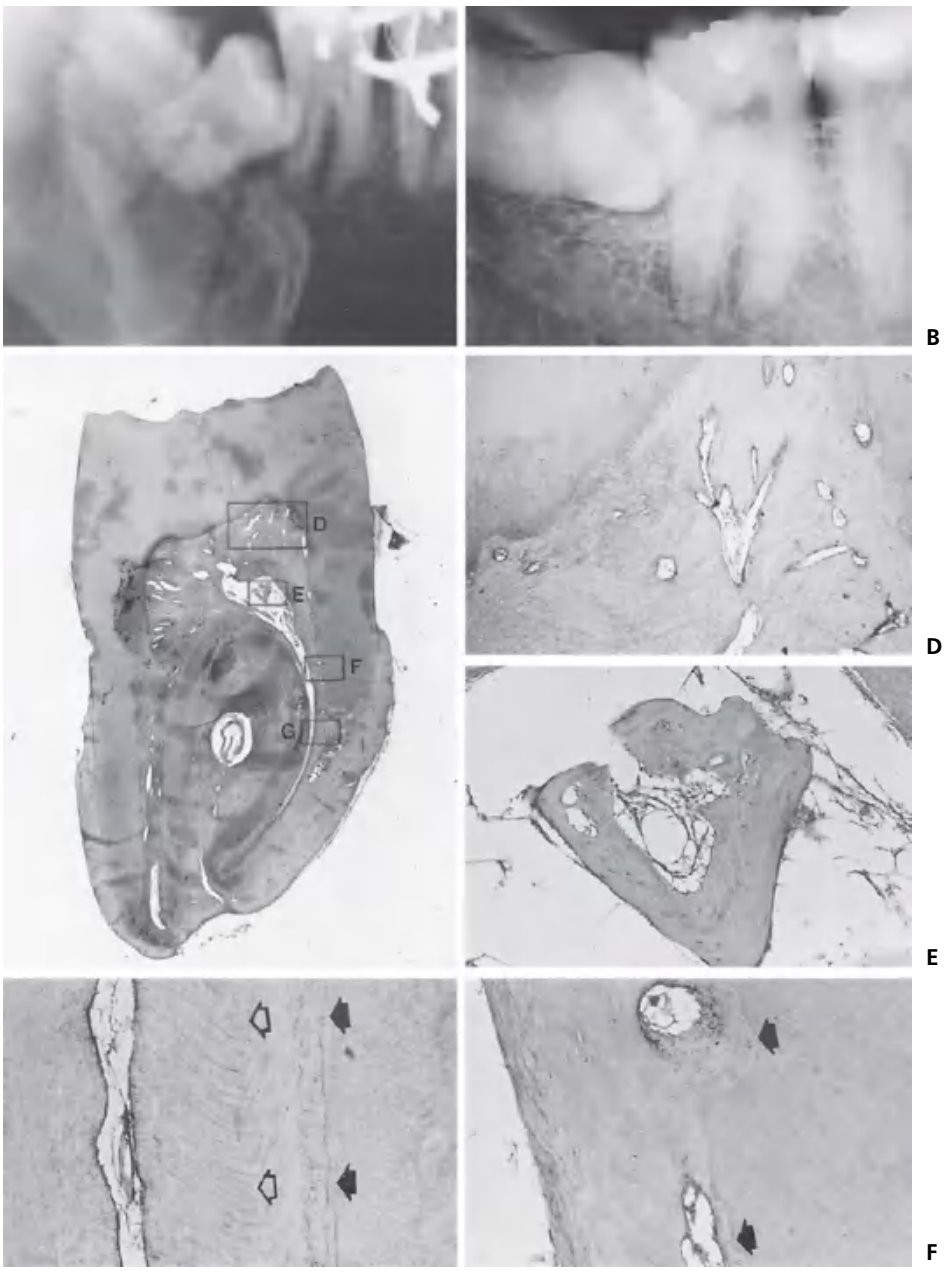



**Fig. 21.33** Obliteration of the pulp canal secondary to a jaw fracture. A. Time of injury – the jaw fracture involves the third molar region. B. Six years later, marked pulp canal obliteration of the third molar is evident. C. Low power view of sectioned molar. ×3. D. Coronal part of the pulp completely occluded with hard tissue. ×30. E. Islands of bone in the middle of the pulp. ×75. F. Apposition of hard tissue following initial resorption of the canal walls (arrows). ×75. G. Normal dentin deposited after formation of atubular dentin immediately after injury (arrows). ×75.

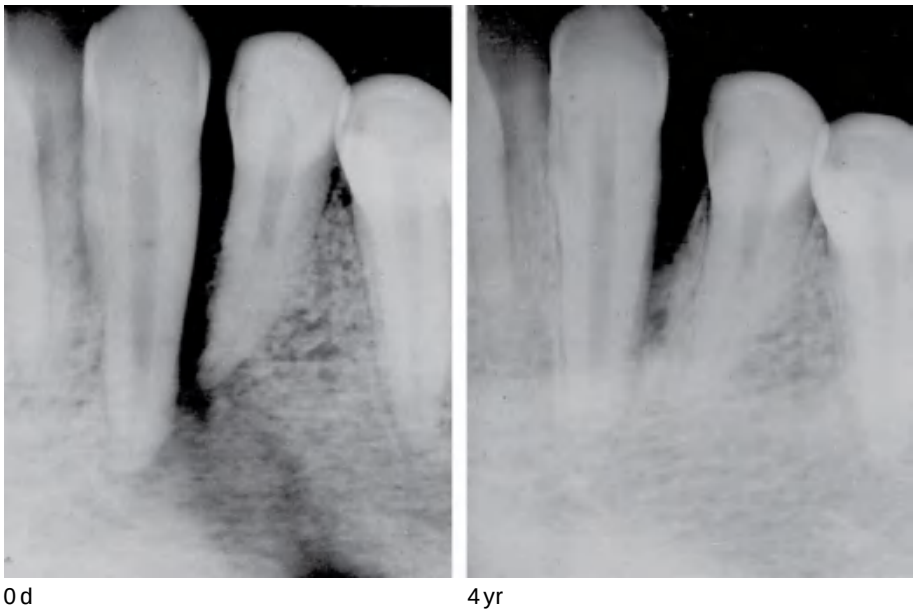



**Fig. 21.34** Incomplete healing of marginal periodontium following jaw fracture in the canine region. 4 years later – deep pocket formation and loss of supporting bone is found. Reproduced from (69) with permission.

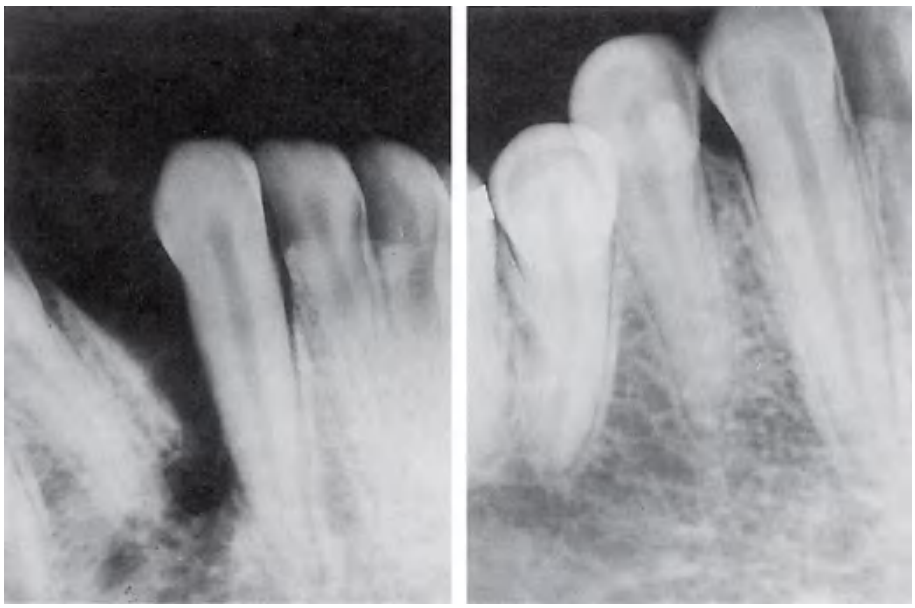



**Fig. 21.35** Complete healing of the periodontium despite extensive dislocation of fragments and exposed cementum of a lower right canine. Reproduced from (69) with permission.

## Essentials

### Terminology (Fig. 21.1)

- Comminution of alveolar socket
- Fracture of alveolar socket wall
- Fracture of alveolar process
- Fracture of mandible or maxilla (jaw fracture)

### Frequency

- Permanent dentition: 16% of dental injuries
- Primary dentition: 7% of dental injuries.

### Etiology

- Fight injuries
- Automobile accidents.

### History

- Symptoms

### Clinical examination

- Palpation of facial skeleton
- Mobility of fragments
- Dislocation of fragments
- Reaction to sensibility tests

### Radiographic examination

- Dislocation of fragments
- Relation between bone fracture and marginal and apical periodontium

### Pathology

- Fracture healing
- Superficial root resorption

### Treatment (Fig. 21.22)

- Administer local or general anesthesia.
- Reposition displaced fragments.
- Teeth in jaw fracture lines should generally be preserved unless severe inflammatory changes of marginal or apical origin are present. Teeth with periapical inflammation can be maintained if antibacterial medication is placed in the root canal. Semierupted third molars should, as a rule, be removed.
- Apply fixation (acid-etch/resin splint). In the primary dentition, a direct splinting method with acrylic may be used. In cases where the fragment can be reduced into a stable position, one may refrain from splinting. In such cases, the patient should be instructed to restrict nourishment to soft food.
- Check reduction radiographically.
- Antibiotic therapy may be indicated in the treatment of jaw fractures.
- Control the involved teeth radiographically and with sensibility tests.
- Immobilize for 4 weeks.
- Follow-up period minimum one year.

### Prognosis for permanent teeth involved in fractures of the alveolar process

- Pulp necrosis: 56–75%
- Pulp canal obliteration: 13–15%
- Progressive root resorption: 5–11%
- Loss of marginal bone support: 3–13%

## Predictors for pulp necrosis in mature teeth

- Fracture in relation to apex
- Displacement in the horizontal part of the fracture >2 mm
- Incomplete repositioning
- Age >30 years

## Prognosis for permanent teeth involved in fractures of the maxilla or the mandible

- Infection in the fracture line: 5–29%
- Pulp necrosis: 5–38%
- Pulp canal obliteration: 2–24%
- Progressive root resorption: 0–4%
- Loss of marginal bone support: 11–18%

## Treatment of pulp necrosis

See Chapter 25.

## Treatment of root resorption

See Chapter 25.

## Treatment of inflammation in the fracture line

- Antibiotic therapy; and
- Extraction or endodontic therapy of involved permanent teeth if they sustain inflammation.

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# 22

# Injuries to the Primary Dentition

M. T. Flores, G. Holan, J. O. Andreasen & E. Lauridsen

## Introduction

This chapter describes the specific characteristics of injuries to the primary dentition, the exclusive reaction patterns of primary teeth to trauma and explains the rationale of the approach to treatment and how it is different from that for permanent teeth.

In the past 15 years the number of articles on traumatic injuries to the primary teeth has increased (13.1%) (1). However, there is still lack of scientific data and suggested treatment protocols are supported by empirical treatment and anecdotal case reports rather than well-controlled research studies.

Four factors have a major influence on the selection of treatment for injured primary teeth:

- The relatively short-period primary teeth are in function in the child's mouth. The maxillary primary central incisors, which are involved more than any other tooth in traumatic injuries, usually serve the child no longer than 6 years.
- The close proximity of the root of the primary tooth to its developing permanent successor (Figs 22.1 and 22.2). This implies that damage to the permanent tooth may be inflicted not only when the primary teeth are injured, but also later as a result of the treatment rendered. This must be considered as an important factor when weighing the cost and benefit of various treatment options for injured primary teeth. The option that has the least likelihood to have a deleterious effect on the permanent tooth should be considered as the treatment of choice. If no damage is expected to the permanent tooth, a conservative approach should be adopted, and attempts made to save injured primary teeth. In some cases, however, extraction is the only reasonable option leaving the child with a missing tooth for several years. Several consequences of early loss of primary incisors are mentioned in the dental literature. However, most lack scientific support.
- The difficulty of gaining the child's compliance. Traumatic injuries to the primary teeth occur more often in very young children before they have mastered walking skills and later running skills. These children are too young to understand what is required, and most of them cannot cooperate during examination and implementation of treatment. The situation is distressing for both, the child and the parents. Fortunately, most luxation injuries affecting the primary dentition heal spontaneously and there is no need for treatment other than recommending good oral hygiene. However, if a therapeutic procedure is going to be performed, consider whether the intervention is immediately necessary, and if the available analgesia in addition to non-pharmacological methods are likely to be sufficient for the child. When sedation is indicated, it should be only administered by a skilled provider or emergency department physicians who are aware of the possible adverse consequences and have the necessary skills to manage such adverse events (3, 4). Thus, lack of child cooperation should not be considered an argument against conservative treatment of injured primary teeth. Moreover, the need to cope with the child's behavior cannot be ignored even if extraction is the treatment of choice.
- The clinician's skills and experience with pediatric patients is of outmost importance for managing the patient's and the parent's behavior in the emergency situation (5). After an accurate diagnosis, the clinician's obligation is, therefore, to describe to the parents all treatment options including the advantages and disadvantages of each option. The final decision on which treatment will be provided to the child should always consider his or her own best interest and is made together by the clinician, -preferably a trained pediatric dentist- and the parents (6).

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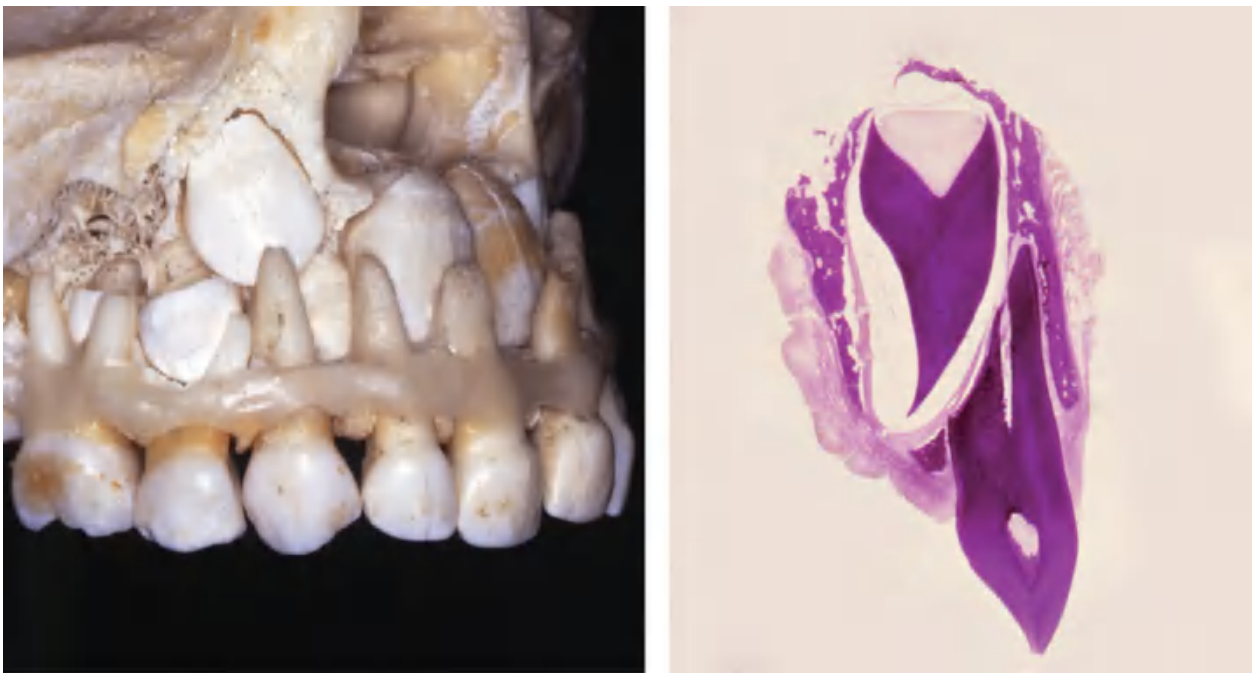


A B

**Fig. 22.1** Anatomic relationship between the two dentitions. A. The maxilla in the skull of a 3-year-old child. B. The intimate facial-oral relationship is shown between the primary central incisor and the permanent successor in a histologic section. Source: Reproduced from (2) with permission.

Consequently, the objectives of trauma management in the primary dentition are:

- To comfort the child and parents in the acute state.
- To avoid inducing dental fear and anxiety in young children who may be experiencing their first dental problem. This implies being very conservative in the choice of treatment, partly by ensuring sufficient pain control and in some cases proper premedication with a tranquilizing drug before necessary intervention.
- To minimize the risk of further damage to the permanent teeth:
  - by limiting the number of acute extractions (which may lead to loss of facial alveolar bone in the area as well as damage to the gubernacular cord (see Chapter 2)).
  - by following the traumatized dentition with appropriate control intervals to detect and treat any symptoms of infection in the area as early as possible.

There are many ways in which an injured primary tooth may transmit damage to the permanent successor.

The direct displacement of a primary incisor toward the developing permanent tooth germ may result in a significant injury (Fig. 22.3). An ensuing infection caused by invasion of bacteria into the injury site may be another threat to the developing successor (Fig. 22.4). Finally, both extraction and attempt to reposition a displaced primary tooth may compromise development of the permanent successor, depending upon the technique used.

Disturbances in permanent tooth germs are latent complications that may be seen following all types of primary tooth injuries especially intrusion, avulsion and alveolar bone fracture. This topic is presented in Chapter 23.

A series of animal experiments has been carried out to estimate the risk of the above-mentioned factors (2, 7–11). The general treatment concepts are primarily based on the results of these studies as well as a few clinical studies in humans (12–20).

## Epidemiology and etiology

Injuries to the primary dentition are common and occur with a significantly higher annual incidence than in the permanent dentition (21–23). In particular, between 18 to 30 months children are very prone to accidents (Fig. 22.5) (21–32). In a Norwegian study the dental trauma incidence was 1.3% with 3.5-year old being the most accident-prone (33). Poor motor coordination and the child's inability to evaluate potential risks are relevant conditions to be considered for a mandatory close supervision at this early age. Lack of supervision, child-related factors and familial factors play as predictors of injuries and have been associated with injuries in young children (34, 35). As falls are the predominant etiology for dental injuries in young age groups, the trauma incidence is usually equally high in both genders (21–23, 36–39) (Fig. 22.5). More boys than girls affected with injuries to the primary dentition, have also been observed (40, 41).

As found in permanent teeth, increased overjet and incompetent lips are predisposing factor that increases the risk of dental injuries of the primary teeth (42, 43)

Children with anterior open bite (41, 44, 45), inadequate lip coverage (40), increased over jet (42), greater than 5 mm (45) or 6 mm (41), experienced more dental

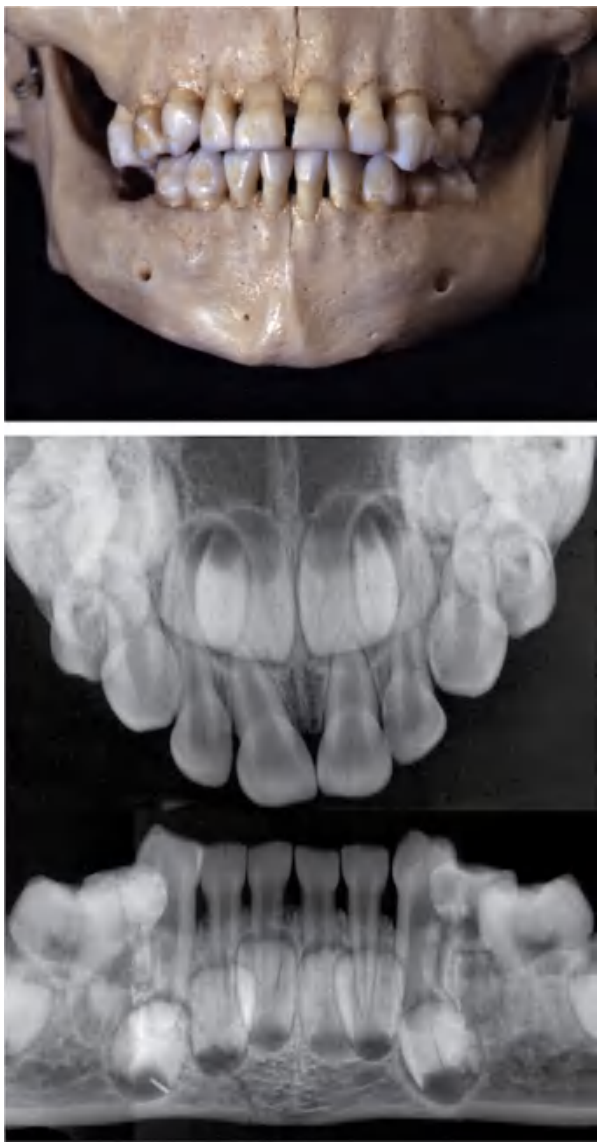


**Fig. 22.2** Skull of 3-year-old individual with a full set of primary teeth. The radiographs illustrate the vertical closeness of the two dentitions.

injuries compared to those with normal occlusion. In a study from Ireland in 839 children, non-nutritive sucking habits were associated with anterior open bite and increased overjet. Both clinical findings were related with an increased prevalence of dental injuries in the primary dentition (41). Besides, a recent study from Brazil involving 1316 children aged 0–5 years, found a strong association between the use of a pacifier and the occurrence of traumatic dental injuries (46).

Several studies have shown that one out of three children has suffered a traumatic dental injury by age 5 (21–23, 47–50). A study from Scotland has shown that the incidence of dental injury in five-year-old children had considerably decreased from 1993 to 2007 (39). For further information concerning the epidemiology and etiology of dental trauma, the reader is referred to Chapter 11.

In some countries, traumatic dental injuries are often not presented as an emergency because parents usually do not seek treatment for the child immediately (51–55), and especially those without soft tissue involvement were presented at a later time after injury (56). In a US study of 487 dental trauma visits seen at children's hospital during a 3-year period, 69% occurred after regular office hours. The emergency visit was the first contact with a dentist for 80% of children 3.5 years old and younger (30), which is similar to the results found in a later study from the same institution (57). This naturally may present a psychological impact upon the child, a condition that is discussed in Chapter 9.

## Self-inflicted oro-dental injuries

Although rare, case reports of self-mutilation injuries in children have been published, some of them related to Leigh disease and Lesch-Nyhan syndrome. In these cases, lacerations of oral and perioral areas are associated with early eruption of primary teeth (58–68). The baby self-inflicts wounds by biting the surrounding soft tissues such as the

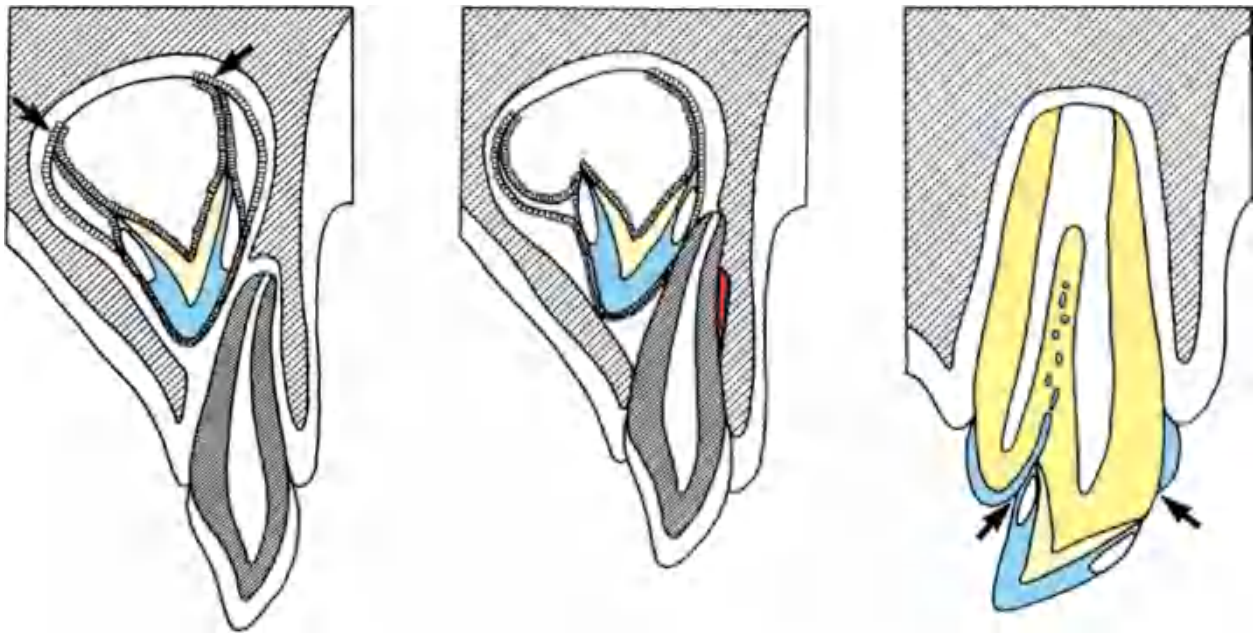


**Fig. 22.3** Intrusion of a primary tooth into the follicle of the permanent successor. All odontogenic tissue becomes involved and a crown dilaceration is the result.

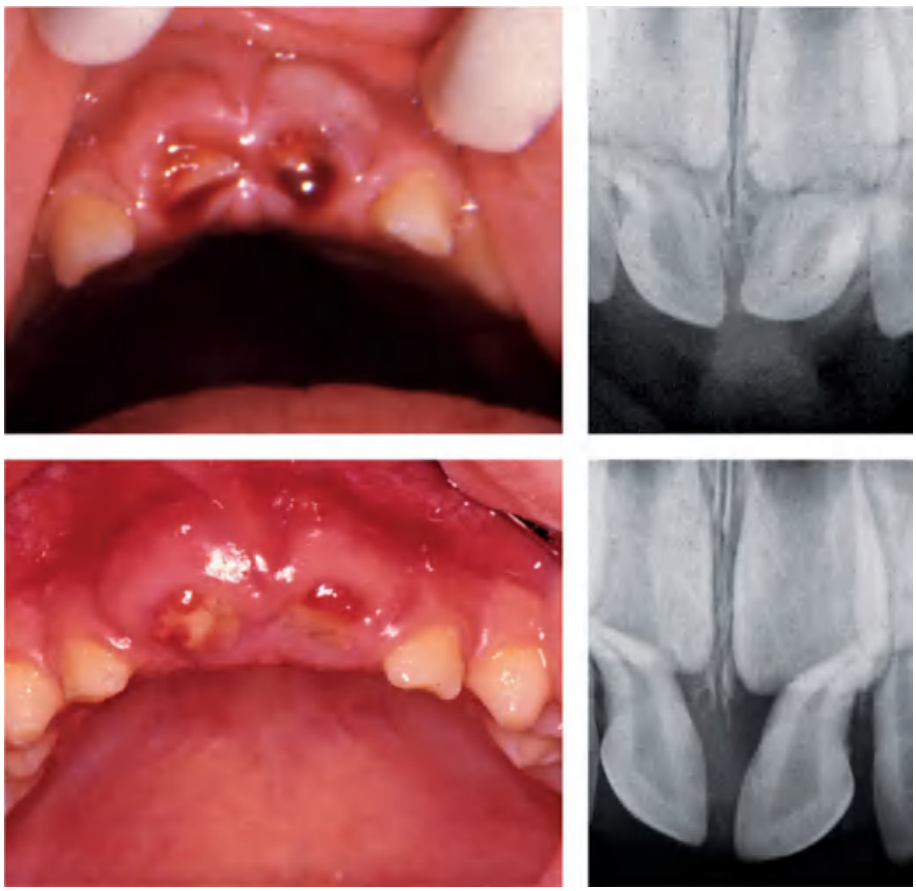


**Fig. 22.4 Acute infection following intrusion**
This 3-year-old boy suffered intrusion of two central incisors. As the root tips were displaced away from the developing tooth germs, spontaneous eruption was anticipated.

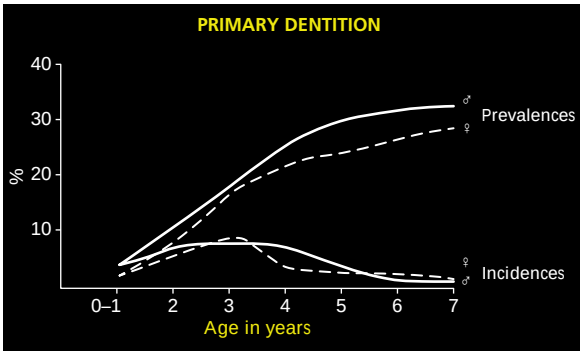



**Fig. 22.5** Trauma incidence in the primary dentition. Among annual trauma incidences (i.e. the number of new injuries suffered during a year), peak incidences in the primary dentition are found at 2–3 years of age, when motor coordination is developing and the children start moving around on their own. Source: Reproduced from (21) with permission.

**Fig. 22.6** Crown malformation of left maxillary incisors due to intubation of a preterm infant.

tongue, cheeks and lips. It seems to be a painless condition and appears with increasing severity when the primary dentition becomes complete.

## Iatrogenic injury to the primary incisors

Endotracheal intubation in preterm neonates

The maxillary primary incisors may be injured during endotracheal intubation in preterm infants (69–73). The laryngoscope blade inserted into the newborn's mouth to identify the entrance of trachea is pressed against the alveolar ridge. The pressure is transferred to the un-erupted developing primary incisors and inflicts damage to the teeth. After eruption, damaged incisors present dilacerations or hypoplasia of the crown (Fig. 22.6). The left maxillary incisors are the most affected teeth because of the shape of the blade and because the laryngoscope is hold in the left- hand side using the right-hand side to insert the endotracheal tube (74–76).

## Battered child syndrome

The physical abuse which may lead to injuries of the teeth and soft tissues is described in Chapter 10.

## Pathogenesis

One difference between injuries in the primary and permanent dentition appears to be the difference in the mechanical properties of the supporting periodontium. In primary dentition, the surrounding bone is less dense and less mineralized, which implies that a tooth hit by a traumatic impact can easily be displaced instead of fractured (19). In a follow-up study from Denmark, up to 98% of the injured primary teeth suffered a type of luxation injury, whereas only 10% showed crown or crown-root fractures (77). This difference in the trauma pattern favoring luxations rather than fractures has been found to be typical for the primary dentition (22, 23). Like in the permanent dentition, there appears to be a strong link between an increased risk of overjet and the risk of traumatic injuries in the primary dentition (41, 45).

Acute trauma results in severe injuries to the crown as well as in luxated teeth. It is important to differentiate acute trauma due to a severe impact on the teeth from trauma resulting in minor crown fractures. Several studies, mainly from Brazil (36, 40, 44, 78) have reported enamel and enamel-dentin crown fractures as the most frequently injuries affecting the primary dentition (37, 41, 45, 79). These studies were carried out during vaccination days or presenting at a dental screening day. By visual inspection, enamel and enamel-dentin fractures as well as missing teeth following trauma and tooth discoloration were diagnosed.

Due to the direction of impact in falls against the ground, floor or furniture, frontal trauma is quite frequent, resulting in lateral luxations with oral displacement of the crown. Consequently, the apical part of the root will almost inevitably penetrate (or fracture) the thin labial bone plate (Fig. 22.7). In falls in which the impact has an axial component, the teeth will be intruded due to the labial curvature of the root; the intrusion will usually result in an axial and labial displacement in which the apex penetrates the labial bone plate (Fig. 22.7). In some cases, the impact direction may have a strong oral component, typically when the child falls with an object in the mouth (e.g. pacifier or toy). In these cases, the apex of an injured tooth may be forced into the follicle of the permanent successor, sometimes resulting in a severe injury to the developing permanent tooth germ (see Chapter 23). However, it is important to emphasize that direct involvement of the permanent tooth germ in the injury is not restricted to cases of oral displacement of the apical part of the primary incisor into the follicle. If the displacement is large enough, the coronal part of an intruded or laterally luxated primary incisor can also be forced against the underlying tooth germ, causing displacement of the germ and squeezing of the epithelial root sheath with the latter resulting in malformation of the crown or arrested root formation of the successor (2, 80).

## Healing and pathology

Most luxations represent a combined injury to the pulp and periodontium. However, the pathology of luxation injuries of primary teeth has received little attention. At present, no specific changes can be ascribed to the periodontal ligament after luxation injury, whereas pulp changes have been reported in a few studies.

**Fig. 22.7** Intrusion of a primary incisor. It has penetrated the labial bone plate.

## Pulp

### *Crown fractures with pulp exposure*

Histologically, exposed pulp tissue in complicated crown fractures is quickly covered by a layer of fibrin. Eventually the superficial part of the pulp shows capillary budding, numerous leukocytes and proliferation of histiocytes (Fig. 22.8A–C). This inflammation spreads apically with increasing observation periods (Fig. 22.8D–F). Pulp exposure, mainly in cases of crown-root fracture, may result in the development of a pulp polyp (81).

## Luxation injuries

Changes seen in the pulp soon after injury include edema and disorganization of the odontoblast layer as well as nuclear pyknosis of pulp cells (Fig. 22.9).

Histologic evidence of pulp necrosis, including nuclear pyknosis and disappearance of odontoblasts and stromal elements can be seen by 6 days after injury (Fig. 22.9). This response is related to either partial or total rupture of the pulpal neurovascular supply. The later events of pulpal healing after luxation injury are presently unknown.

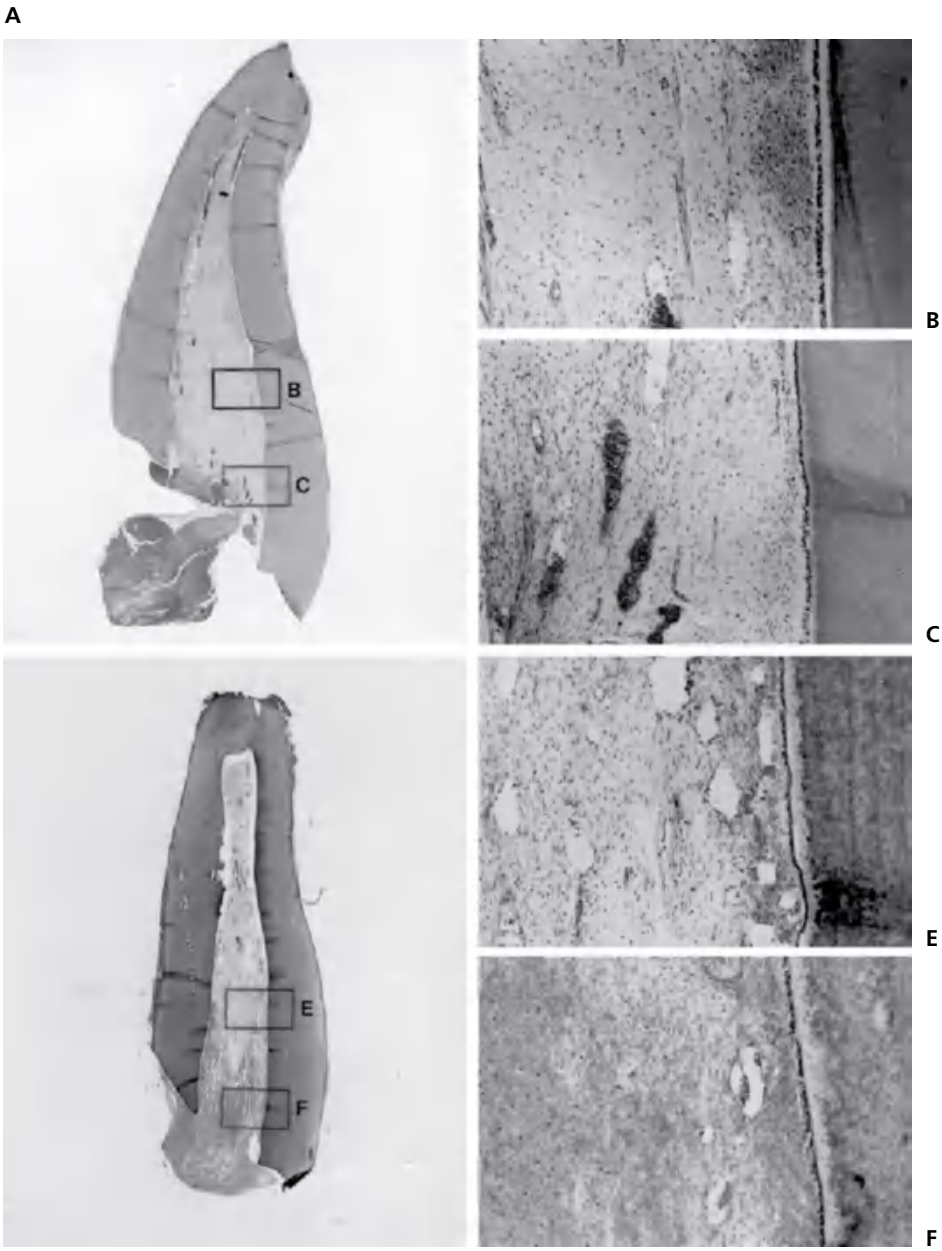



**Fig. 22.8** Early pulp reactions following complicated crown fractures in maxillary primary incisors. A. Low power view of incisor removed 19 hours after injury. Moderate inflammatory changes are present in the coronal part of the pulp while the apical part shows only minor changes, such as hyperemia and perivascular bleeding. ×9. B. Hyperemia and perivascular bleeding. ×75. C. Moderate inflammatory changes. ×75. D. Low power view of incisor removed 41 hours after injury. Marked inflammatory changes are present in the coronal part of the pulp and spread of the inflammation apically is evident. ×9. E. Moderate inflammation. ×75. F. Marked inflammatory changes. ×75.

If the pulp survives or becomes revascularized, a number of regressive pulp changes can occur, such as hyalinization and deposition of amorphous, diffuse calcifications. Furthermore, the injury usually interferes with normal dentin formation. This interference is apparently related to a number of clinical factors, among which the stage of root formation and the type of luxation injury seem to be decisive (82, 83).

In teeth with incomplete root development, a distinct incremental line usually indicates arrest of normal tubular dentin formation at the time of injury. Most of the dentinal tubules stop at this line, while the original predentin layer is often preserved.

After a certain period, apposition of new hard tissue is resumed, but without the normal tubular structure (Fig. 22.10). This tissue often contains cell inclusions which maintain their tubular connections with dentin formed before the injury, as well as vascular inclusions. This hard tissue formation often continues to the point of total obliteration of the root canal (Fig. 22.11).

Although the mineralized tissue formed in response to the injury may resemble bone and cementum, it lacks the cellular organization characteristic of these tissues. Due to its tendency to convert to tubular dentin, the repair tissue has been termed cellular dentin.

Furthermore, tube-like mineralization in the dental pulp of traumatized primary incisors has been described (Fig. 22.12) (84, 85). This phenomenon is possibly analogous to the ingrowth of bone into the pulp cavity of injured permanent teeth (see Chapters 2 and 16).

## PDL healing

So far there are no studies analyzing periodontal ligament (PDL) healing events.

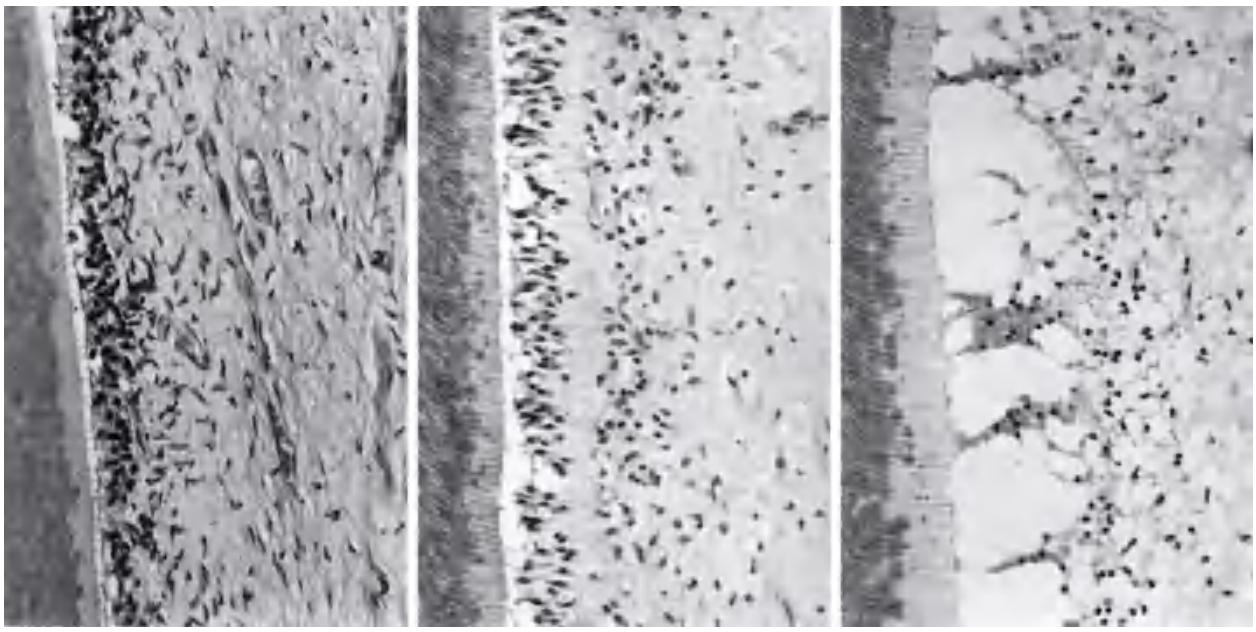



**Fig. 22.9** Early pulp changes at various time intervals following intrusive luxation of primary maxillary incisors. A. Two hours after injury, pulpal structures – including the odontoblasts – are without significant change, apart from edema. ×195. B. After 13 hours nuclear pyknosis and disintegration of odontoblast layer is evident. ×195. C. Six days after injury, only pyknotic nuclei are seen within a stroma of autolyzed pulp tissue. ×195.

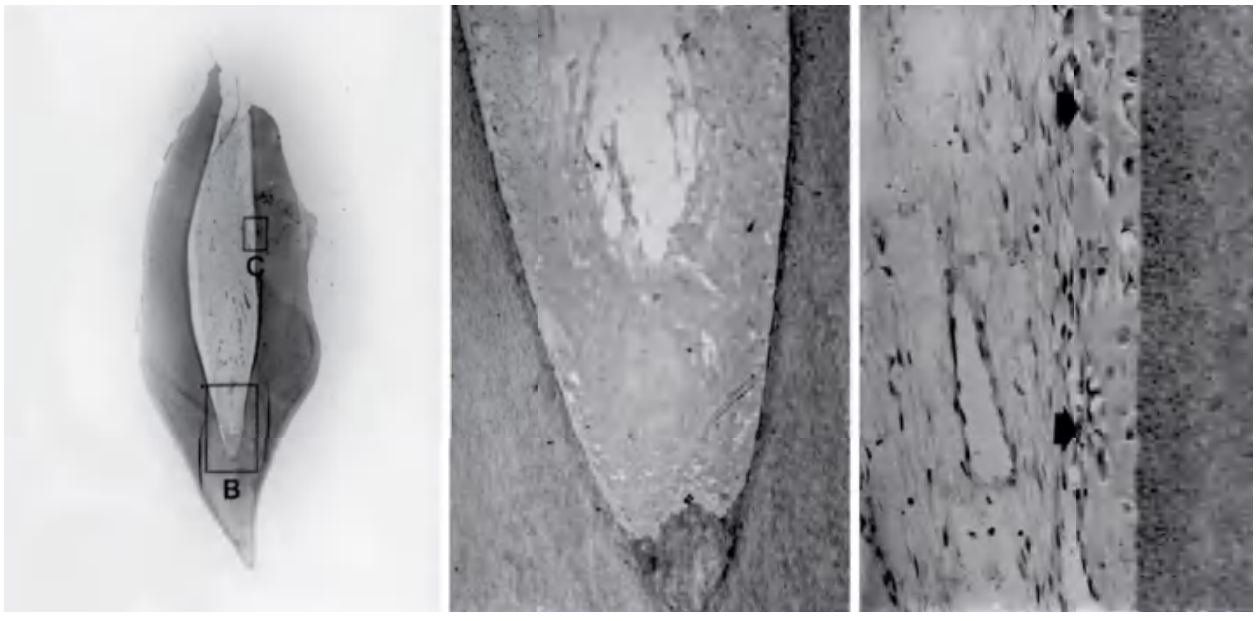



**Fig. 22.10** Early histologic changes following extrusive luxation of a primary right central incisor. A. Low-power view of sectioned incisor. The tooth was extracted 4 weeks after injury. ×6. B. Complete necrosis of the coronal part of the pulp. ×30. C. More apically, formation of new hard tissue with numerous cell inclusions (arrows). ×195.

## Clinical and radiographic findings and treatment of various injury types

### Examination

Before clinical examination begins, a complete anamnesis should be obtained including general health condition and dental history. The examination of the injured tissues should always follow general examination of all facial and oral organs and tissues to avoid overlooking pathologic finding that are not related to the injury (171, 172).

#### *Extraoral examination*

The child must be checked for facial asymmetry (indicating jaw fractures), swelling of the lips, skin lacerations and cuts, and scars (indicating previous injuries). Bleeding from the

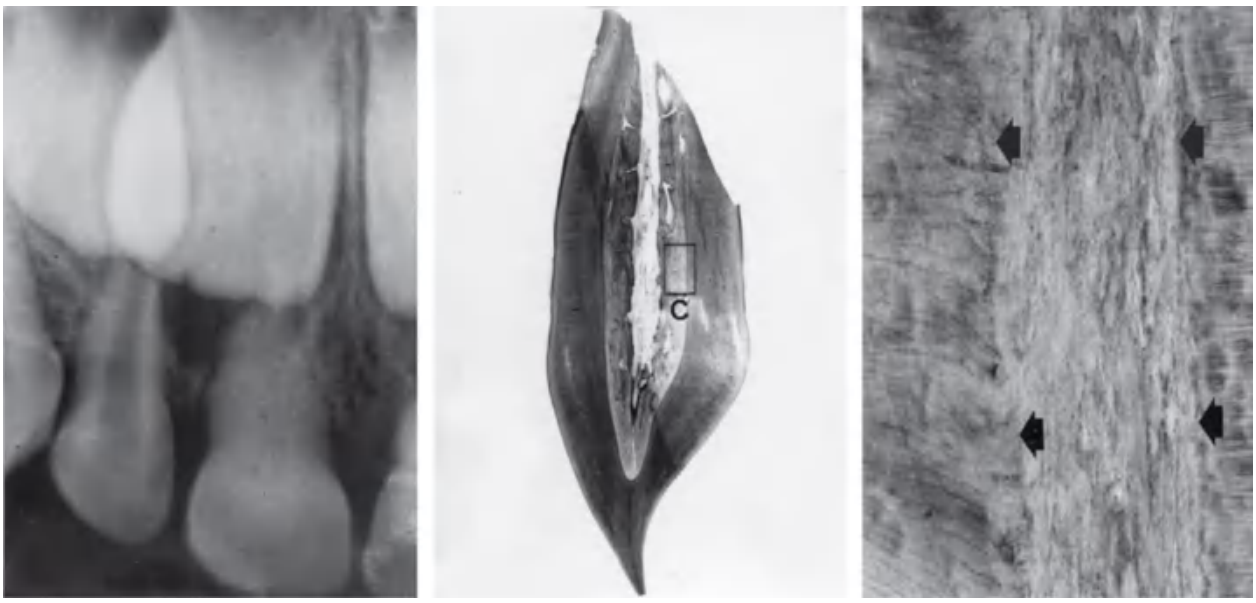



**Fig. 22.11** Pulp canal obliteration in a primary central incisor following luxation injury. A. Radiographic condition at time of extraction. No pulp cavity is discernible. B. Low power view of sectioned specimen. Obliteration of the pulp canal is evident. ×10. C. Note atubular dentin deposited immediately after trauma followed by a return to tubular dentin (arrows). ×75.

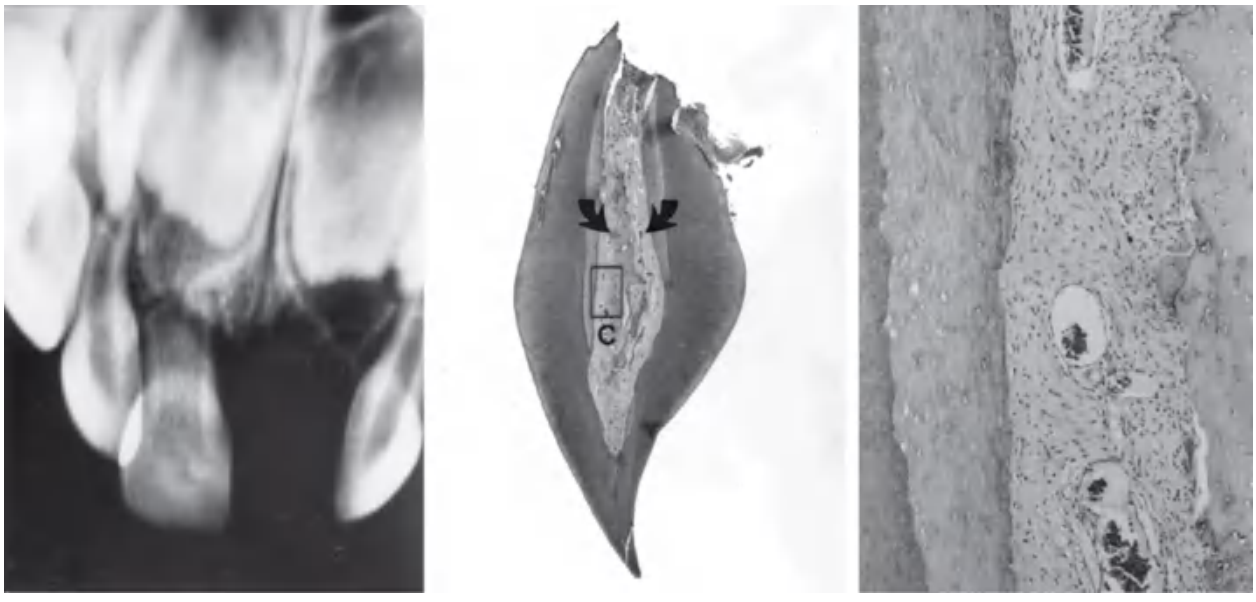



**Fig. 22.12** Bone invasion in the pulp after trauma in a primary incisor. A. Radiographic exposure. Note the two longitudinal radiopaque stripes in the root canal. B. Histopathologic section showing incomplete ring of calcified tissue in the pulp canal surrounded by non-inflamed pulp tissue. C. Note that odontoblasts are not seen along the root dentin and the irregular secondary dentin.

nostrils and subcutaneous hemorrhage near the nostrils may indicate fracture of the alveolar bone. Oro-facial and maxillofacial injuries need especial attention and follow-up for early detection of later possible complications. Displacement of primary teeth into the nasal cavity has been reported (86–88) and also a long-term chronic facial pain due to a traumatic dislocation of a primary tooth into the nasal concha (89). Limited ability of mouth opening may indicate damage to the temporomandibular joint. Sensitivity of facial bones when palpated may point to underlying jaw fractures. For reviews the reader is referred to articles published by Harding, 1995 (90); Wilson, 1995 (91); Flores, 2002 (92) and McTigue, 2009 (93).

A laceration or hematoma located at the chin should draw attention to a possible fracture of the mandible at the symphysis or condyle (94). Fractures of molar teeth resulting from sudden impact of the mandibular teeth against their maxillary antagonists should also be looked for after traumatic injuries to the chin (95–97).

### *Intraoral examination*

Intraoral tissues must be carefully examined. The surrounding soft tissues (lips, oral mucosa, attached and free gingivae and frenum) should be checked for lacerations and hematomas caused by recent injuries or fistulae, which may be the result of old injuries. The lips should be inspected looking for possible embedded tooth fragments. It has been reported a total displacement of a primary incisor into the lip tissue in a child patient who initially had inadequate diagnosis and treatment (98). Submucosal hemorrhage in the upper lip may result from a direct impact or fracture of the labial bone plate. Similarly, submucosal hemorrhage under the tongue should raise the suspicion of fracture of the mandible. Midline fracture in the mental suture area appears in children under one year of age.

Signs of bleeding from the sulcus surrounding the injured tooth indicate damage to the periodontal ligament.

Palpation of the gingivae and the vestibule may reveal a fluctuant hematoma above a displaced tooth and sometimes a displaced root can be felt.

### *Tooth mobility*

An injured tooth can present increased mobility in the horizontal as well as vertical plane. Increased mobility is a sign of injury to the periodontal ligament. It appears in subluxation, root fracture, extrusive luxations and associated infection. Sometimes the patient seeks treatment several days after the trauma due to tooth mobility caused by an infection. In cases of crown-root fractures the coronal fragments will present increased mobility. If mobility of a segment of teeth is observed, fracture of the alveolar bone should be suspected (see Chapter 21).

### *Tooth alignment*

Changes in tooth alignment can be seen following luxation injuries. In case of doubt, parents should be asked about the original tooth alignment or present a recent picture taken before the accident, in which the child's teeth are seen. Extrusion, lateral luxation or root fracture may result in occlusal interference.

### *Pulp vitality*

Pulp vitality testing by electric or thermal methods has not been proven to be valid in the primary dentition.

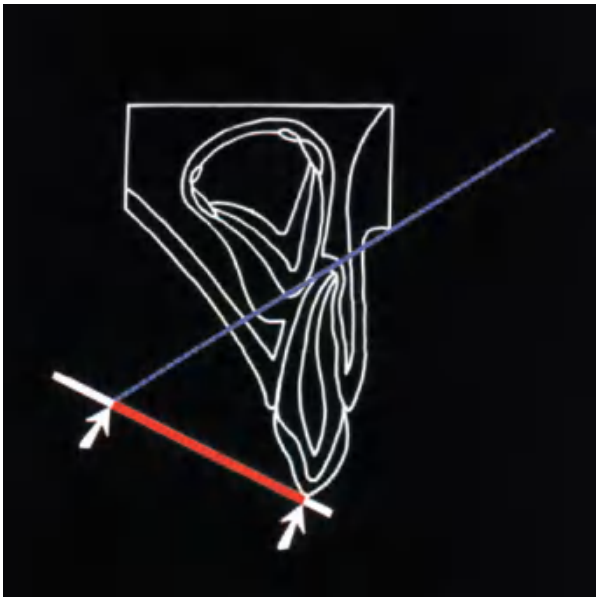
## Radiographic examination

The general features of radiographic examination of primary tooth injuries have been described in Chapter 12 Radiographs are an important adjunct to the clinical examination, providing valuable information that may affect the decision regarding treatment of the injured primary tooth. It shows the degree of development of the primary tooth and its permanent successor and the relationship between the two. Furthermore, physiologic and pathologic root resorption and the position of displaced primary teeth can be seen. An acceptable radiograph appears to be the occlusal exposure due to the fact that neither the child nor relatives can position the film in an optimal position (periapical exposure) (Fig. 22.13). When there are soft tissue injuries associated, a radiograph of the lip is necessary to look for possible embedded tooth fragments (98). In the radiographic examination it is important to consider the relation between the injured primary tooth and the permanent successor. In this regard a very simple rule exists: if the tooth appears foreshortened compared with its non-injured antimere, this indicates a labial displacement of the root with a minimal risk for the permanent successor. On the other hand, if a displaced primary tooth radiographically appears elongated, this tooth has most likely been intruded into the follicle of the permanent tooth (Fig. 22.14). Another important feature is to evaluate the position of the mineralized part of the permanent tooth in the follicle. In case of displacement an asymmetry will be seen compared to a non-injured antimere (Fig. 22.15).

These guidelines can only be applied when the central beam is oriented exactly at the midline between the two incisors to be compared. Apart from this, lateral projections can be of assistance in determining the direction of dislocation of the displaced tooth as well as its relationship to the nasal floor and labial alveolar bone (Fig. 22.16; see also Chapter 12). If there is doubt about the position of

**Fig. 22.13** Radiographic examination of a young child. A parent can assist in stabilizing the child during the examination. The parent and child are furnished with lead aprons. One arm is used to hold the child, while the other holds the film-holder and stabilizes the child's head against the parent's chest.

A



B

**Fig. 22.14** Schematic illustration of the geometric relationship between an intruded primary incisor and the developing tooth germ and the resultant radiographic image. A. If the primary tooth is *away* from the developing tooth germ, the radiographic image will be *foreshortened*. B. If the primary tooth is intruded *into* the developing tooth germ, the radiographic image will be *elongated*.

a displaced primary incisor in relation to the nasal floor, a lateral exposure can be of value (Fig. 22.16).

## Treatment regimens

The overall principle of treatment is pain management, to avoid additional suffering to the child and not to take any risk with regard to the permanent successor which usually implies a very conservative approach (92, 99).

Acute treatment can often be restricted to dealing with conditions causing marked symptoms, such as crown fractures with pulp exposure and in few cases, repositioning or removing displaced teeth with occlusal interference (100).

As most dental injuries occur during early childhood, when the child is not able to communicate or express pain intensity, the use of topical anesthetics, local anesthesia and sedation should be considered. It has been demonstrated that from birth the child can experience pain which may cause prolonged behavioral consequences if not alleviated immediately (101). In more complicated tooth injuries, such as those affecting the alveolar bone as intrusion, lateral luxation and alveolar fractures, prescription of paracetamol or non-steroidal anti-inflammatory drugs could be indicated if the child is in pain before further examination and treatment (102, 103) (see also Chapter 9).

In addition, use of analgesics may improve the quality of care when pain is anticipated, such as application of a local anesthesia. The administration of a single dose of analgesic one hour before injection and the use of topical anesthetic will reduce the discomfort of a local anesthetic (101).

Apart from advising the parents to administer adequate pain control, adequate hygiene procedures and a soft diet to the child's liking must be reviewed.

In the following pages, each type of trauma will be described as it presents clinically and radiographically, along with currently recommended treatment (104). However, most of the recommended treatment procedures have minimal evidence supporting their value. Therefore, treatment decision making at the time of acute injury should always consider the child's best interest (which goes far beyond the scientific evidence), like minimal intervention, a painless procedure and a safe environment. This is extremely important especially for children younger than 3 years old (92).

The following treatment recommendations are in agreement with the Guidelines for the management of traumatic injuries to the primary dentition published for the first time by the International Association of Dental Traumatology (IADT) in 2001 (105) and updated twice since then (104, 106).

## Crown fractures

Crown fractures cause relatively few symptoms when they do not involve the pulp. Exposure of the pulp is a complicated injury that deserves immediate care in order to avoid pain to the child during meal time. For review, the reader is referred to an article published by Güngör (107).

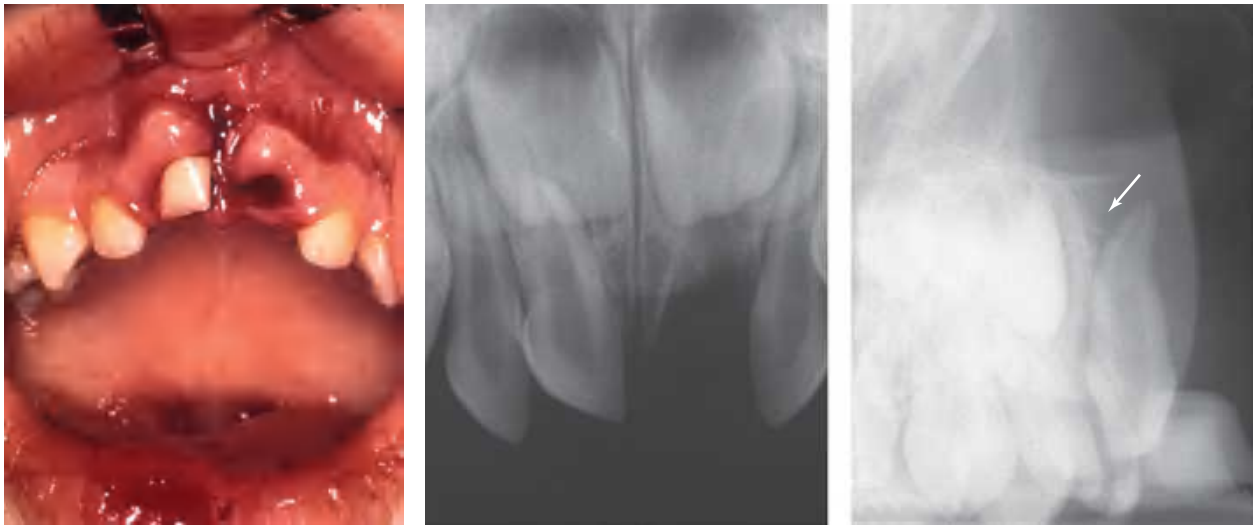
### *Radiography*

This will normally show the relation between the fracture and the pulp chamber.

### *Treatment*

In cases of uncomplicated fractures with sharp fracture edges, an abrasive disc or bur can be used to smooth the fractures. If the parents demand an esthetic solution, and the

**Fig. 22.15** Radiographic demonstration of displacement of the developing permanent tooth germ following intrusion of the primary incisor. The distance between the incisal edge and mineralization front of the involved developing tooth germ is shorter than the same distance in the non-involved tooth germ, implying displacement of the involved tooth germ. Although the displaced primary tooth was removed, a slight dilaceration of the permanent crown developed.



A B C

**Fig. 22.16** Radiographic demonstration of the direction of displacement in a case of intrusive luxation of a primary central incisor. A. Clinical condition. B. Right central incisor appears shorter than right lateral incisor due to displacement in apico-labial direction of the former. C. Lateral radiograph reveals the relationship between the apex of the displaced tooth and the permanent tooth germ as well as the direction of dislocation. Note that the apex of the primary tooth has been forced through the facial bone plate (arrow).

patient can comply, the crown can be repaired with composite resin.

Complicated crown fracture is a difficult emergency to deal with if there is lack of cooperation from the child and because the treatment (pulpotomy) is technique-sensitive. The treatment options are partial pulpotomy with calcium hydroxide (104, 106) or pulpotomy with formocresol and zinc oxide eugenol paste (105).

It seems that the outcome is as good between the choices available, supporting the indication for a conservative approach to treat these injuries. In one clinical study, the success rate of pulpotomy was 76% (108). In a histological study on exposed human pulp due to trauma to primary incisors, it was found few inflammatory cells in the root canal. Therefore, cervical pulpotomy has been recommended for large traumatic pulp exposures (109). In another clinical study, pulpotomies (using formocresol) and pulpectomy (using zinc oxide eugenol) were compared and found to have success rates of 86% and 78% respectively (110). A disturbing finding concerning pulpectomy was, however, that the majority of cases showed incomplete resorption of the zinc oxide eugenol dressing, a fact which may leave zinc oxide particles in the gingival area (110).

### *Prognosis*

In a study by Mendoza-Mendoza et al. (155) 58 enamel and enamel-dentin fractures were analyzed. Among 28 enamel-dentin fractures, nine teeth were diagnosed with pulp necrosis. No other complications have been reported.

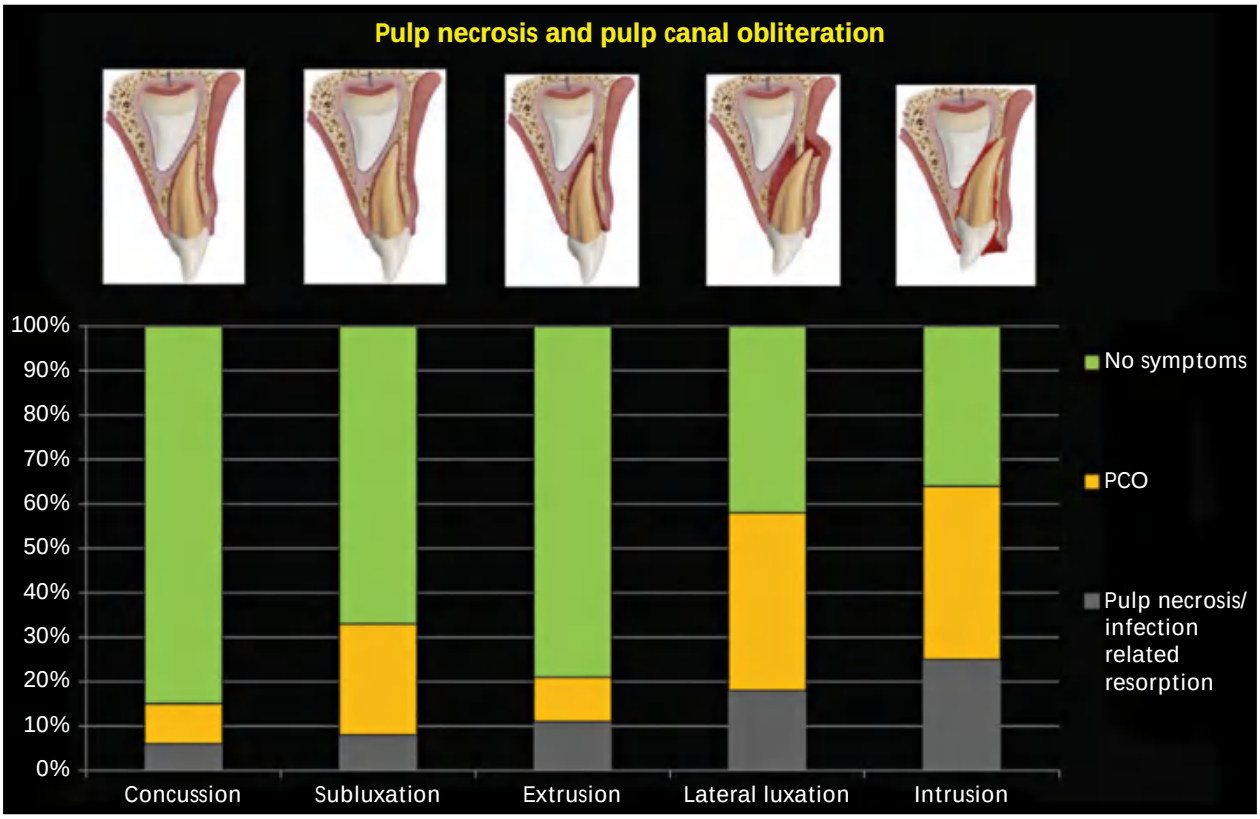



**Fig. 22.17** The risk of pulp healing complications in primary teeth with luxation injuries. Source: Reproduced from (174–176) with permission.

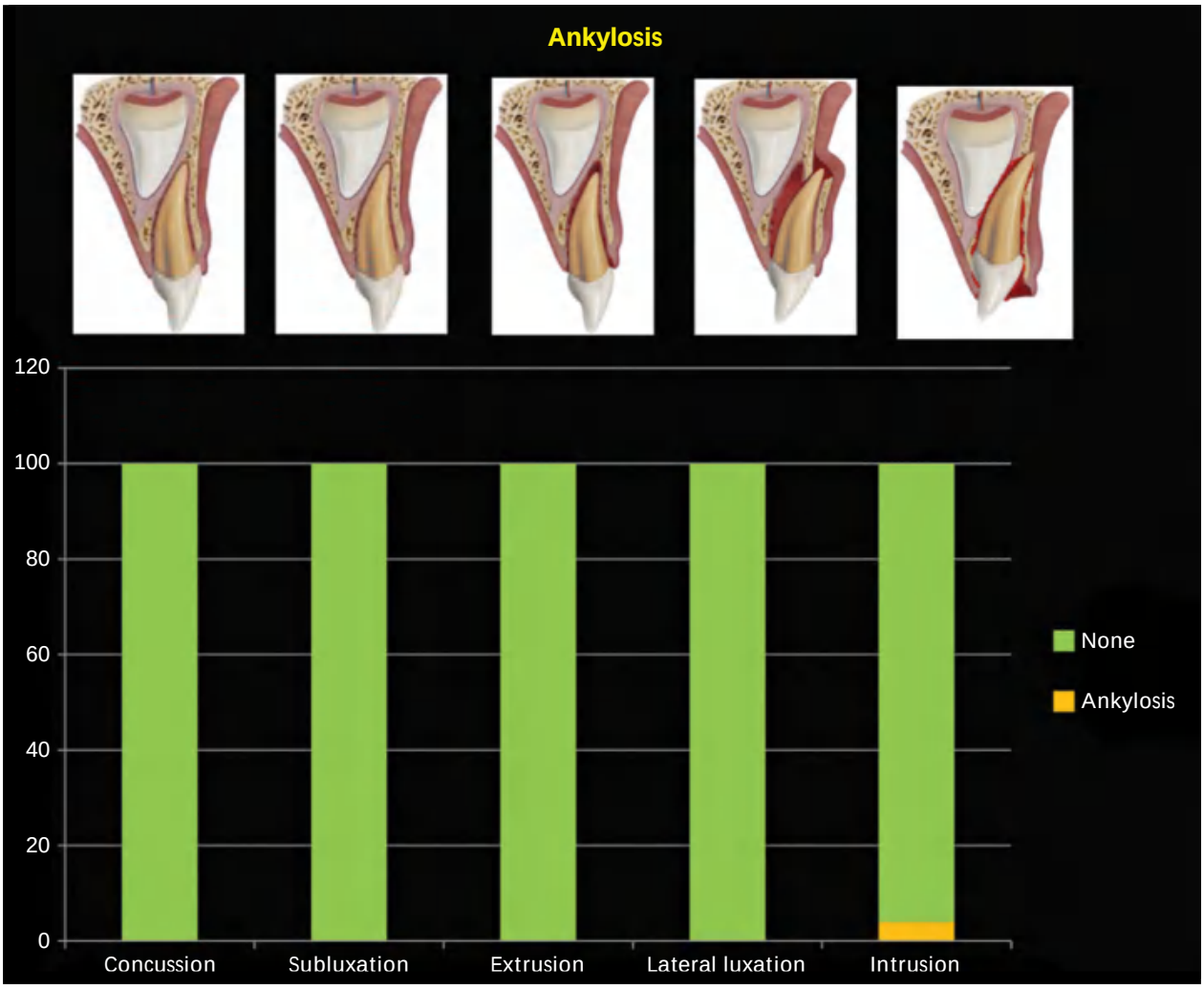



**Fig. 22.18** The risk of ankylosis in primary teeth after luxation injuries. Source: Reproduced from (174–176) with permission.

**Fig. 22.19** Uncomplicated crown fracture of a right central incisor and subluxation of both central incisors in a 2-year-old girl.

**Fig. 22.20** Mesial crown-root fracture of a right central incisor and an uncomplicated (enamel) fracture of the left central incisor.

## Crown-root fractures

Crown-root fractures are rare, and the crown fragment is usually kept *in situ* by the gingival and periodontal fibers. Because of mobility during mastication there might be transitory pain (Fig. 22.20).

### *Radiography*

In laterally positioned fractures the extent in relation to the gingival margin can be seen. One exposure is necessary to disclose multiple fragments (104).

### *Treatment*

Depending on the clinical findings, two treatment scenarios may be considered: Fragment removal only if the fracture involves a small part of the root and the stable fragment is large enough to allow coronal restoration (104). In children with immature, still developing roots, after removal of the loose segment, it is advantageous to preserve pulp vitality by pulp capping or partial pulpotomy. Calcium hydroxide is a suitable material for such procedures (106).

Extraction is usually the treatment of choice.

### *Prognosis*

In a follow-up study of 17 crown-root fractured teeth which were preserved, it was indicated that 16 were re-examined and none of these showed any complications (155).

## Root fractures

The coronal fragment may be mobile and may be displaced (104).

### *Radiography*

The fracture is usually located mid-root or in the apical third.

### *Treatment*

Root fractures with minimal displacement of the coronal fragment can be left untreated and will resorb at the expected time (Fig. 22.21). When the coronal fragment is very loose the extruded coronal fragment should be extracted to prevent the child inhaling it. The apical fragment should be left for physiological resorption

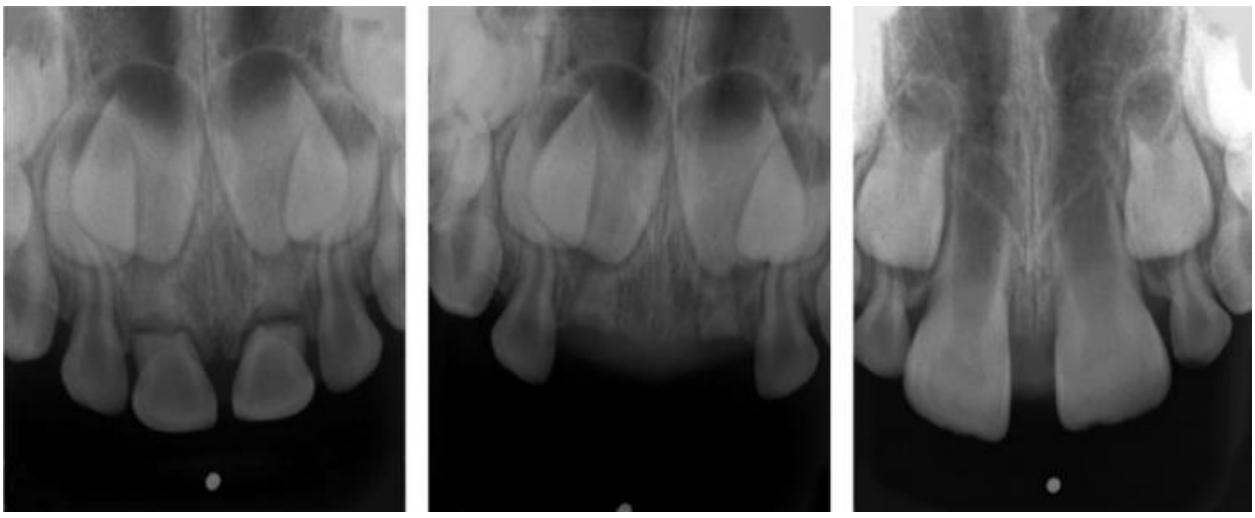


**Fig. 22.21** Normal physiologic resorption after root fracture of both primary central incisors in a 4-year-old child. Note apparently normal physiologic resorption of the traumatized incisors.

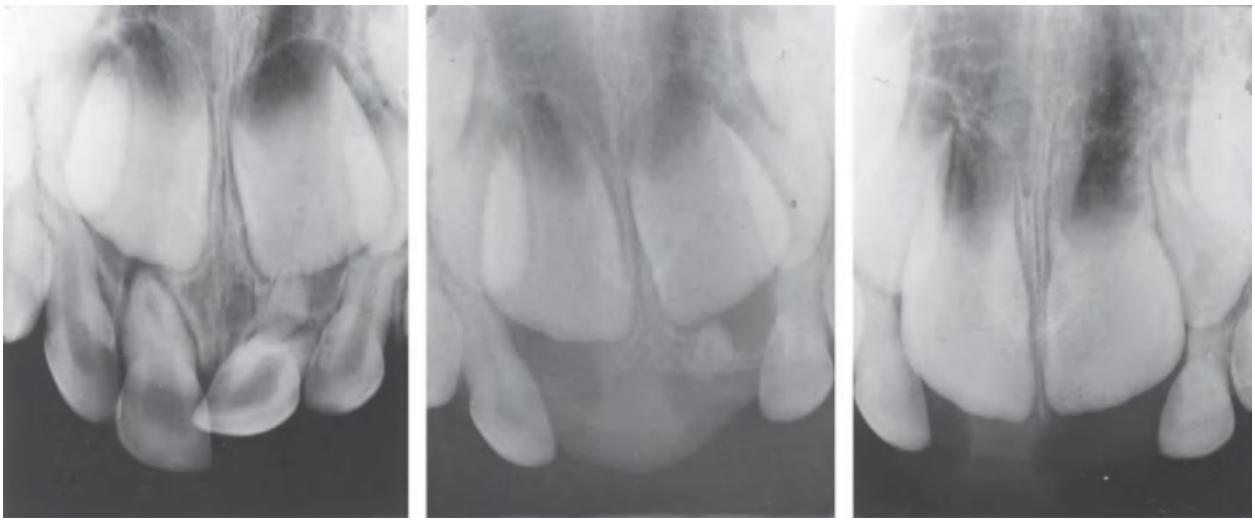



**Fig. 22.22** Root fracture of left central incisor and lateral luxation of a right central incisor in a 5-year-old child. The right incisor was extracted, as was the coronal portion of the left central incisor. Condition after 1 year. After 2 years, both permanent central incisors are erupting and the apex of the left primary incisor has been resorbed.

(Fig. 22.22) (90, 105). Attempts to remove the apical fragment may damage the developing permanent successor. If the child is able to cope and the coronal fragment is a little displaced, a wire-composite splint has been suggested for 3 weeks. A study on the use of splint to stabilize 90 teeth affected by lateral luxation, extrusion and root fractures, showed good prognosis when a splint was used for root fractured teeth (182).

### *Prognosis*

Only one case series has been published reporting the prognosis for primary teeth with root fracture (156). The study included 17 teeth. Within the first week the coronal fragment was extracted in six teeth due to excessive mobility. Among the 11 teeth which were left without further treatment, one showed subsequent pulp necrosis and the remaining teeth showed successful healing and normal physiological resorption of the apical and coronal fragment.

## Fractures of the alveolar process

### *Clinical findings*

In this situation an entire fragment of the jaw is involved. This happens typically when a severe frontal impact hits the mandible or the maxilla. Displacement may vary according to direction of the impact, usually being frontal in the maxilla and sometimes the result of a fall with an object in mouth in the mandible. In these cases, the segment tilts in labial direction (Fig. 22.23).

**Fig. 22.23** Alveolar fracture comprising all primary incisors in a 5-year-old boy.

### *Radiography*

This will reveal the topography of the horizontal fracture line to the apices of the primary teeth and their permanent successors. A lateral radiograph may also give valuable information about the relation between the two dentitions (Fig. 22.23).

### *Treatment*

Reposition is essential to normalize occlusion. General anesthesia is often indicated. The treatment of this trauma entity is described in Chapter 21.

### *Prognosis*

One case report has shown successful outcome after following the IADT Guidelines recommendation (157).

## Fractures secondary to chin trauma

When there has been an impact to the chin, multiple crown and crown-root fractures may occur in the molar regions (95–97). The treatment depends on the extent and severity of the fractures. While minor enamel fractures can be left without treatment, composite restoration should be considered for fractures with dentin exposure to avoid pain to thermal stimulus. Stainless steel crowns should be used to cover primary molars with large fractures extending slightly below the gingival margins. Pulpotomy can be performed if the fracture involves the pulp. Extraction is the treatment of choice for other more severe fractures in which the tooth is not restorable. In these cases, space maintenance must be considered.

## Concussion and subluxation

### *Clinical findings*

In concussion the tooth is tender to touch (performed with a fingertip). In subluxation the tooth appears loosened but has not been displaced and bleeding from the gingival crevice can be observed if the child is examined shortly after the injury. Diagnose of subluxation, if examination is performed several days after the injury, is based on tooth mobility and parents' report on bleeding around the tooth immediately after the traumatic event.

### *Radiography*

No radiographic abnormalities. Normal periodontal space (104).

### *Treatment*

These injuries require no acute treatment apart from instructing the parents to maintain good oral hygiene to prevent bacterial contamination through the ruptures gingival and periodontal attachment. Brushing with a soft tooth brush and topical application with cotton swabs of alcohol-free 0.12% chlorhexidine to the tooth-gingival junction twice a day for 7 days is recommended. Soft diet for the first few days after the injury may also be recommended.

### *Prognosis*

The concussion diagnosis is based on the patient's response to percussion test. Therefore, it is difficult to assess in minor children. Hence, there is very little documentation of the risk of healing complications following this type of injury in the primary dentition. However, it appears that the risk of pulp and periodontal complication is low (Figs 22.17 and 22.18) (77, 174). A few studies have reported the complication rates after subluxation (52, 77, 155, 158, 174).

Some of these studies have included patients which showed up for treatment weeks or months after the injury had occurred (52, 158). With such delay in treatment the subluxation diagnosis is questionable. In a study from Spain, where children were regularly examined for follow-up controls, out of 96 subluxated teeth, 13 teeth developed pulp necrosis and 11 teeth, root resorption until time of tooth loss (155). Figs 22.17 and 22.18 show the risk of healing complications from a study based on 241 teeth where all cases were diagnosed up to 2 days after trauma (174). The risk of pulpal and periodontal complications was low.

## Extrusion

### *Clinical findings*

The tooth appears elongated and there is usually a marked loosening of the tooth. The crown is generally displaced in lingual direction which may cause occlusal interference or cross bite with the lower incisor, unless the child has an open bite due to sucking habits.

### *Radiography*

A marked apical periodontal ligament space is seen.

### *Treatment*

Extruded primary teeth can be repositioned and stabilized for a short period if the child is seen soon after the injury. If a blood clot has already become organized in the alveolar socket and repositioning is no longer applicable, the tooth can be left for spontaneous alignment or extracted depending on the degree of extrusion and mobility and if there is interference with the opponent teeth.

### *Prognosis*

Teeth with extrusive luxation often get extracted at the initial visit. However, few case series have documented the prognosis for extruded primary teeth which were left untreated and observed (155) or were observed after repositioning (159, 175). Figs 22.17 and 22.18 show the risk of healing complications based on 26 teeth (175).

## Lateral luxation

### *Clinical findings*

The crown is usually tilted in the oral direction and the tooth firm in its displaced position (Figs 22.24 and 22.25). Most of these luxations result from a frontal impact that causes a tilting of the tooth with the crown displaced in lingual direction and the apex with a labial bone plate displaced in labial direction. In rare cases, in which the child has fallen with an object in the mouth, the opposite displacement direction can be found. These cases may represent a danger to the permanent successor as the apex of the primary tooth may have invaded the follicle. These teeth should either be repositioned or extracted.

### *Radiography*

A marked apical periodontal ligament space is best seen on the occlusal exposure. The same exposure can sometimes also show the position of the displaced tooth and its relation to the permanent successor (104).

### *Treatment*

Because of the common anterior open bite in young children most lateral luxated primary teeth have no occlusal interference and can be left without treatment, and spontaneous repositioning influenced by physiologic forces of the tongue can usually be expected within 3 months (Fig. 22.24). However, in a follow-up study, 5% of laterally luxated primary teeth were not fully repositioned after 1 year (77). In case of minor occlusal interference, slight grinding is indicated (104).

In some cases of lateral luxation, when there is more severe occlusal interference or cross bite with the opponent teeth, after the use of local anesthesia, the tooth can be gently repositioned by combined labial and palatal pressure (Fig. 22.25) (104, 111). If necessary and possible, a splint can be applied for 2–3 weeks. A recent study from Korea has shown that a splint may be the treatment of choice in selected cases, for tooth stabilization, when repositioning of a lateral luxated tooth is indicated (182).

### *Prognosis*

Only a few studies have reported the frequency of healing complications after this luxation type (77, 155, 159, 175). Figs 22.17 and 22.18 show the risk of pulp and periodontal complications based on 331 laterally luxated teeth which were left without further treatment. Generally, there was a fairly low risk of pulp necrosis and infection and subsequent tooth loss. Soporowski et al. reported a somewhat higher frequency of pulp necrosis (50%) for 28 teeth which were repositioned compared to 52 teeth (17.3%) which were left untreated and observed in the long-term study (159).

## Intrusion

### *Clinical findings*

In the primary dentition, the apices of intruded teeth will usually be driven through the thin vestibular bone, the direction probably being determined by the direction of the impact and the labial curvature of the root. This usually results in marked swelling of the upper lip due to hematoma.

A B C D

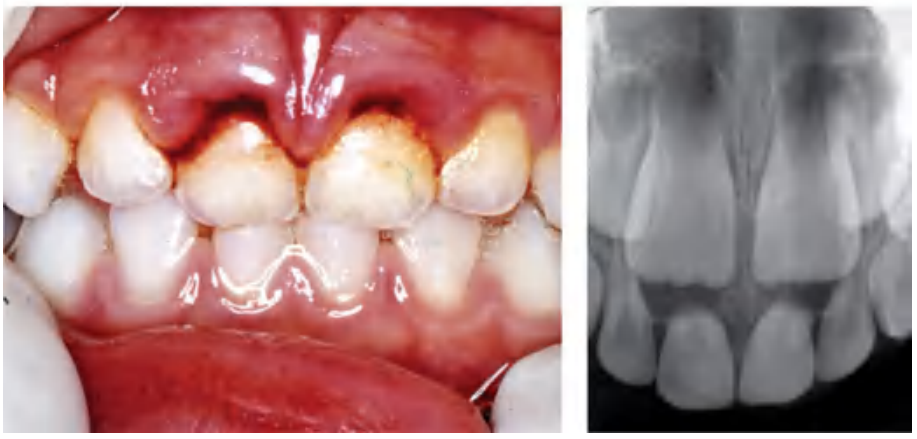
**Fig. 22.24** Spontaneous reposition of a lateral luxated primary incisor in a 1.4-year-old boy. A. Lateral luxation of a left central incisor. B. The tooth appears foreshortened in the radiographic examination compared with the contralateral. Spontaneous repositioning is therefore anticipated. C and D. At follow-up, 2 months after injury there is spontaneous realignment.

**Fig. 22.25 Repositioning of a laterally luxated primary central incisor**
This 4-year-old boy suffered a lateral luxation of the left maxillary primary central incisor. Repositioning is indicated due to occlusal interference.

**Treatment evaluation**
Due to interference with occlusion and the distance between the apex of the primary tooth and the permanent tooth germ, repositioning is indicated.

**Repositioning**
After application of a local anesthetic, the tooth is repositioned with combined labial and palatal pressure.

**Postoperative condition**
The tooth has been adequately repositioned, as revealed both clinically and radiographically.



The degree of intrusion cannot always be assessed by measuring the length of the clinical crown as the gingiva may be enlarged due to swelling. The degree of intrusion and re-eruption are determined by the distance between the incisal edge of the intruded tooth and the horizontal line connecting the incisal edges of two adjacent non-injured teeth.

For later comparison, the degree of dislocation should be recorded in millimeters (i.e. the distance between the incisal edge of the intruded incisor and the non-intruded adjacent tooth), with the direction of dislocation indicated as well. Especially in the primary dentition, it is of great importance to determine whether the apex is dislocated facially or orally because in the latter case, the permanent successors can be directly involved (see Figs 22.15 and 22.16).

In some cases, the tooth has disappeared completely into the socket and can often be palpated labially.

### *Radiography*

An occlusal radiograph is indicated and will normally show the position of the displaced tooth and its relation to the permanent successor. If there is doubt about this question a lateral exposure can be of value (see Figs 22.15 and 22.16). A lateral extraoral exposure has traditionally been recommended to assess the relations between the intruded primary incisor and its permanent successor. A study evaluating the diagnostic value of lateral extraoral radiograph in determining the relative position between the root apex of the intruded primary tooth and the labial surface of its permanent successor (112) found that in the majority of cases the contribution of the lateral exposure was limited. The diagnostic value was low when more than one incisor was intruded, when lateral incisors were intruded, and often due to overlap of anatomical structures. The relations are obvious when the root of the intruded primary incisor has been pushed labially accompanied by fracture of the labial bone plate. In these cases, however, the lateral extra-oral radiograph is unnecessary as clinical signs and a periapical radiograph are sufficient to determine the relationship (112)

### *Treatment*

The treatment of intruded teeth is still being debated (113). The essential problem is the prevention of injuries transferred to the developing permanent teeth. In experimental studies in monkeys, in which primary incisors were intentionally intruded toward the permanent successor, it appeared histologically that extraction of the intruded primary incisor resulted in minor damage to the reduced enamel epithelium of the permanent successor compared to when the intruded primary incisor was preserved (2). However, in a similar macroscopic study, it was found that the frequency and extent of the macroscopic enamel defects were almost identical in the two groups (7).

In general, spontaneous repositioning should be awaited, which normally takes place within 3 months (77) (Fig. 22.26). However, in the same study, one out of four intruded teeth were not fully repositioned one year after trauma and no decisive factor could be identified to explain the lack of realignment (77). It has been demonstrated that most intrusion injuries are repositioned spontaneously (116–118, 176).

During the "re-eruption" phase of intruded primary teeth, there is a risk of infection around the displaced tooth. This is manifested clinically as swelling and hyperemia of the gingiva, sometimes with abscess formation and oozing of pus from the gingival crevice (Fig. 22.4). There is a rise in temperature and complaint of pain from the traumatized region. In these cases, immediate extraction and antibiotic therapy are essential to prevent the spread of infection to the permanent tooth germ. To minimize the risk of such infection, the parents or caregivers should be provided with strict instruction to keep the teeth clean and use of alcohol-free 0.1% chlorhexidine gluconate topically on the affected area with cotton swabs twice a day for 1 week. A soft diet for 10 days and restriction in the use of an intra-oral pacifier are also recommended (104). When these recommendations are observed and the child is brought to the clinic for follow-ups controls, healing of the gingiva and the absence of plaque contribute to a successful spontaneous repositioning (176).

In the few cases where the intruded primary incisor has been displaced into the follicle as shown radiographically, surgical removal is indicated. In this context, it is important to consider that a proper surgical technique be employed to avoid further injury to the developing dentition. Elevators should never be used due to the risk of their entering the follicular space. Moreover, it is necessary that the intruded incisor be grasped proximally with narrow forceps and removed with the root pointing in a labial direction. These precautions are necessary to avoid collision with the developing tooth germ. Finally, once the tooth has been removed, the palatal and facial bone plates should be repositioned with slight digital pressure. The open wound should then be sutured (Fig. 22.27).

In rare cases an intruded primary incisor becomes ankylosed in an intruded position. In most cases this will only imply a slight delay in shedding of the tooth.

In conclusion the optimal therapy for most intruded primary teeth appears to be to wait for spontaneous repositioning.

## Prognosis

A good review of treatment and healing complications has been published by Diab & Elbadrawy (160). In a study from Brazil on 221 intrusion injuries, children aged 1 to 4 years were the most affected. Pulp necrosis, premature tooth loss and color changes, were the most common sequelae observed (161).

In a study from Turkey, it was observed that most intrusion injuries occurred between the ages of 13 and 36 months and complete spontaneous reposition happened in 66 teeth (83%) in this age group. This study did not find any correlation between the child's age at the time of intrusion and the frequency of subsequent developmental disturbance (116). In a study from Copenhagen, most intruded teeth (83.7%)

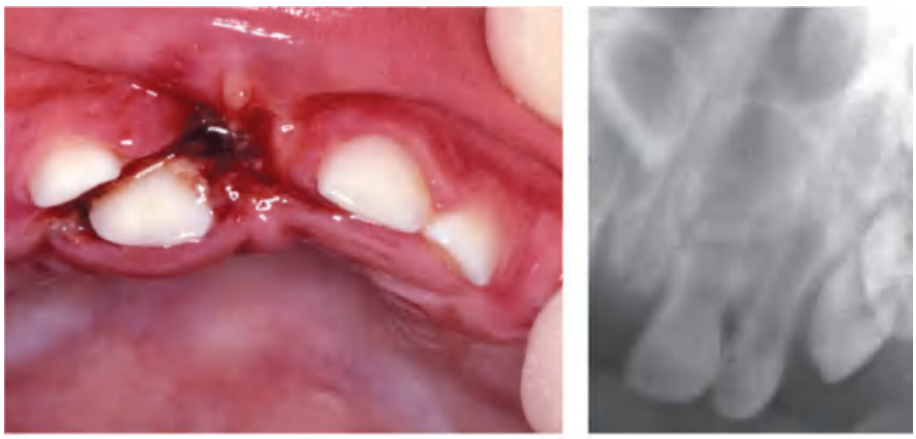
**Fig. 22.26 Spontaneous re-eruption of an intruded primary incisor**
This 2-year-old boy suffered an intrusion of the right central incisor. The foreshortened appearance of the intruded tooth implies labial displacement. Spontaneous re-eruption is therefore anticipated.

**Follow-up, 2 months after injury**
The tooth has erupted approximately 2 mm coronally.

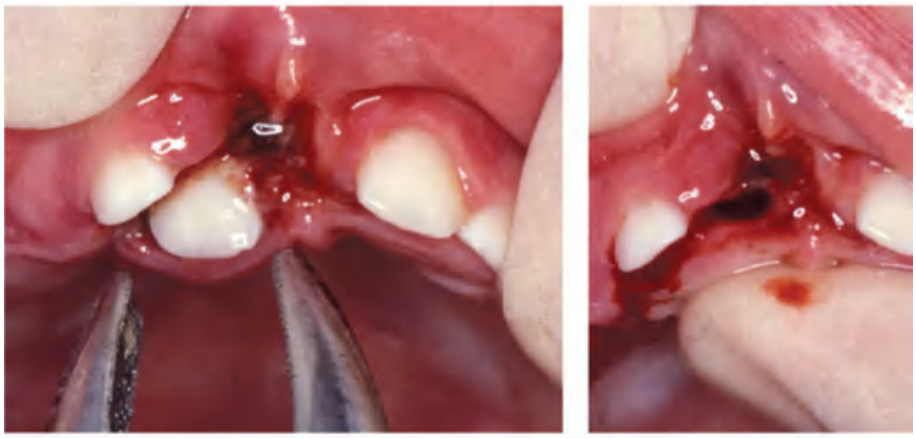
**Follow-up, 3 months after injury**
The tooth lacks 1 mm for complete eruption.

**Follow-up, 1 year after injury**
The tooth is in normal position. Crown color is normal, and the radiographs show no sign of pathology.

**Fig. 22.27 Intrusion of a primary right central incisor**
This 1-year-old boy sustained an axial impact, resulting in intrusion. Note the severe displacement of the permanent tooth germ. To limit the damage to the permanent tooth it was decided to remove the primary incisor.



**Removing the displaced tooth**
Using sedation and local anesthesia, the tooth is grasped proximally with forceps and removed in a labial direction. The fractured and displaced palatal bone is repositioned with digital pressure, and a suture placed to close the entrance to the socket.



**Follow-up**
At examination 1 week later, a certain change in the position of the tooth germ is seen.

**Disturbance in eruption**
At the age of 6 years, it is evident that a crown dilaceration has developed.

spontaneously re-erupted within the first year as well as complications were diagnosed during this period. The risk of pulp necrosis was lowest in patients less than 2 years of age (176).

## Avulsion

### *Clinical findings*

The tooth appears missing in the dental arch. The clinical appearance resembles that of complete intrusion or root fracture with loss of the coronal fragment. Before making the diagnosis of a primary tooth avulsion, alternative trauma scenarios should be explored:

- Was the primary tooth actually avulsed; has it been found?
- Is there a possibility that the tooth is deeply intruded? (Take a radiograph)
- Has the tooth been inhaled? Have there been any coughing or breathing problems after the injury? (Refer for chest X-ray)
- Even if there is no intention to replant the avulsed primary incisor, the tooth must be found to ensure that it has not been aspirated (119, 120) or pushed into the nostrils (86–88). If the tooth is not found, the child should be referred to an emergency department for further examination.

### *Radiography*

This should be performed to verify an empty socket and rule out that the missing tooth is totally intruded.

### *Treatment*

Though several reports have been published on replantation of avulsed teeth (114, 115, 121–127), this practice cannot be recommended for the best interest of the child (6). The guidelines for the treatment of avulsed primary teeth of the International Association of Dental Traumatology (104), endorsed by the American Academy of Pediatric Dentistry (128), recommended avoiding replantation of avulsed primary teeth. Their main claim was that insertion of the avulsed tooth back to its socket has a potential risk to damage to the developing permanent tooth germ because replantation of a primary tooth may displace a coagulum into the follicle of the permanent incisor. Furthermore, a periapical inflammation after pulp necrosis in the replanted primary tooth may cause mineralization disturbances in the permanent dentition. Whereas a published critical review of the literature, challenged the traditional arguments that recommend to avoid replantation (129), a systematic review on replantation of avulsed primary teeth, discusses the outcomes of 17 case reports that were published in the literature since 1925 (130). This study included 41 replanted primary teeth, and 12 of these showed post replantation infection or other complications. All reports except one (127), lack of relevant information about the child's management and how the procedure was carried out. In recent years, studies have shown that unintentional injuries lead to a significant number of preschool children suffering from long-lasting post-traumatic stress disorder (PTSD) (131, 132). Replantation of an avulsed primary tooth in early childhood is a severe traumatic event for a child and parents.

**Fig. 22.28** Lost primary incisor replacement with a prosthetic device anchored to the primary molars.

Because medical procedures significantly affect a child's PTSD symptoms (133) and the parents' as well (134), replantation of a primary tooth is a traumatic dental event that should be avoided to prevent the immediate and long-term effects of childhood trauma associated to pain or injury in young children (132).

The space created by loss of a maxillary primary incisor can be restored for esthetic purposes by fixed appliances (173) (Fig. 22.28). However, special attention should in these cases be paid to a possible interference with the physiologic expansion of the maxilla. A published review of the literature showed that premature loss of primary incisors because of trauma has neglected effect on space loss, speech impairment, biting and mastication or acquired habits (135). The only effects found are changes in the eruption patterns of the permanent successors that are known to common complication after traumatic injuries to the primary teeth and an esthetic problem in the eyes of the parents.

# Complications in the primary dentition

As with the permanent dentition, a series of pulp and periodontal complications may occur especially in relation to the more severe luxation injuries. In regard to diagnosis of healing the chosen observation periods appears to be of significance (77, 136, 174–176). Most complications can be diagnosed within the first year.

## Pulp healing complications

The status of the pulp is mainly evaluated from color changes of the crown and radiographic changes as electrometric pulp testing is not reliable in young children. In the following section, the validity of these findings will be discussed.

### *Color changes of the crown*

Color changes are frequent following luxation injuries. In one study of 545 injured primary incisors, more than half of the teeth present at follow-up showed discoloration during the follow-up period (77). Discoloration was a frequent finding after examination of 836 (41) and 1500 children (137).

Discoloration can be transient or permanent, and the latter occurs in about half of the cases (20, 77, 138, 139). Such discoloration may fade, and the tooth may regain its original shade (77, 140). Transient discoloration is often followed by permanent yellow discoloration (Fig. 22.29) which is often associated radiographically with pulp canal obliteration. Teeth with persisting dark discoloration may remain asymptomatic clinically and radiographically or they may develop apical periodontitis (20, 141). There is an association between crown discoloration and pulp necrosis in traumatized primary teeth (20, 142, 143)

However, crown discoloration alone is not enough to establish this diagnosis, which requires additional clinical findings (fistula, abscess).

The question naturally occurs about the histological status of teeth in which discoloration is the only sequela after trauma. In two clinical and histologic studies, it was found that it was not possible to relate color changes to the histological status of the pulp (83, 144) whereas the opposite was found in another study (141).

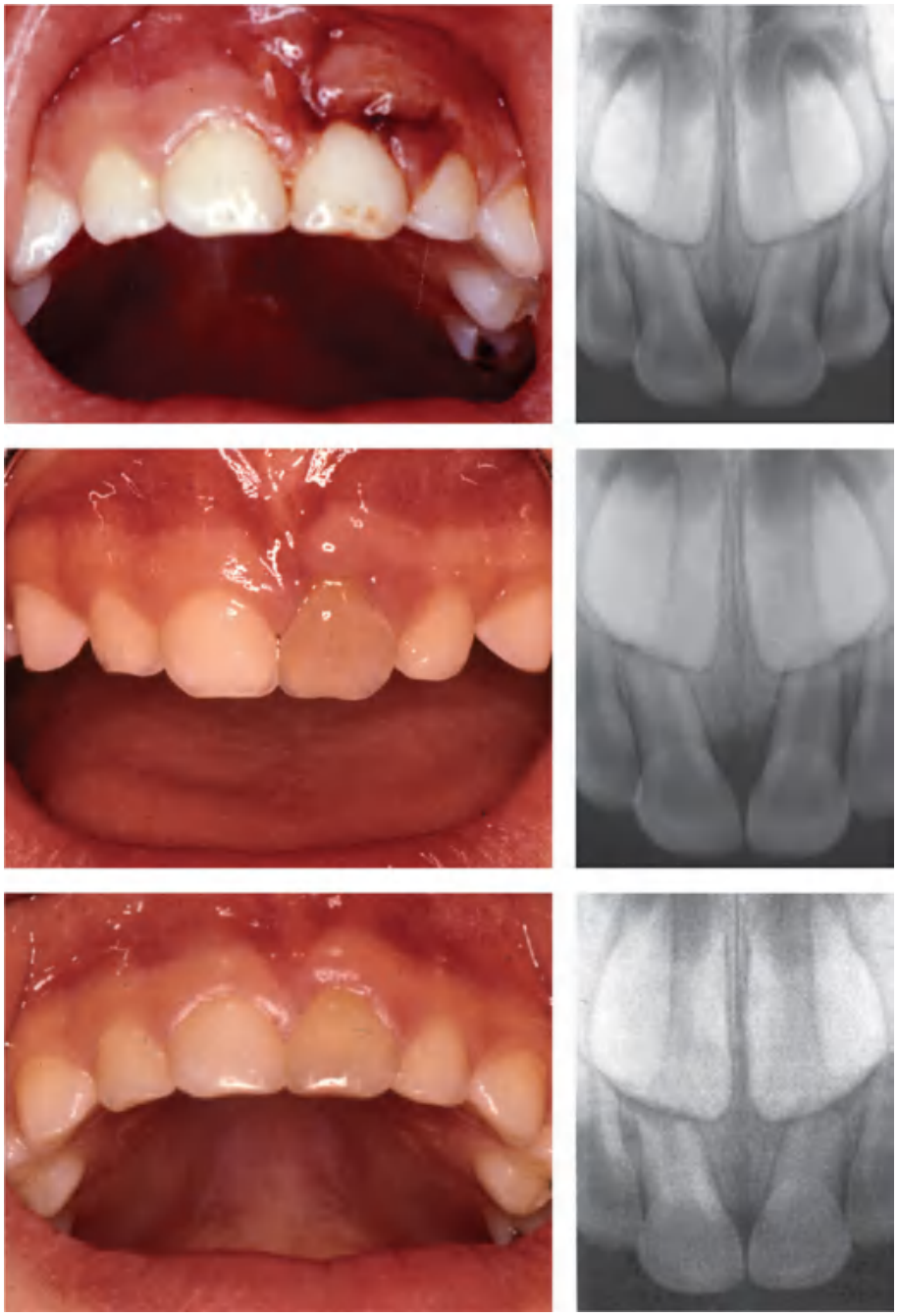


**Fig. 22.29 Clinical and radiographic follow-up after primary tooth injury**
Reversal of coronal discoloration of a subluxated primary incisor.

**Follow-up, 3 weeks after injury**
Intense reddish-brown discoloration is seen.

**Follow-up, 1 year after injury**
The color has changed to yellow, and the radiographs demonstrate pulp canal obliteration.

In clinical studies, it has been shown that most discolored primary teeth do not develop radiographic or clinical signs of infection and are exfoliated at the expected time (20, 139, 140, 142, 145). Thus, the presence of discoloration alone is not an indication for treatment, but an adequate follow-up schedule should be maintained to detect any additional symptoms. Unless associated infection exists, root canal treatment is not indicated (139).

In rare occasions the injured tooth becomes pink. This, if observed shortly after the injury, may be transient and reflect intra-pulpal hemorrhage (138). Pink discoloration that appears long after the injury is the result of internal resorption. Resorption of the dentin from the pulpal side becomes possible when odontoclasts are formed in a chronic-inflammatory-environment and the odontoblasts fail to produce predentin, which normally protects the dentin against odontoclastic activity (181).

### *Radiographic findings*

Periapical radiolucency may develop. In most cases periapical radiolucency is seen within the first year after the injury (174–176). Another radiographic sign is arrested root development and lack of physiologic continuous dentin apposition (i.e. the width of the root canal does not diminish after injury). In the evaluation of radiographs, it should be considered that during the initial phases of eruption of the permanent successor, an enlargement of the follicle of the permanent successor takes place that may imitate a periapical radiolucency related to a pulp necrosis of the primary incisor (Fig. 22.30). In one study this phenomenon appeared in 72% of follow-up cases of primary teeth showing dark discoloration. In the 26% control teeth the same feature was seen. At present there is no valid explanation for this phenomenon (20). Also, the presence of a sinus tract is significantly related to infection related resorption and increased radiolucency of the dental sac (146).

### *Other findings*

A fistula or an abscess almost always indicates pulp infection.

## Pulp necrosis

Pulp necrosis is seen less frequently in the primary dentition compared with the permanent dentition after similar types of luxation (77, 142, 174–176) and can in most cases be diagnosed within the first year after trauma (174–176) (Fig. 22.31). In a multivariate analysis, the decisive factors in the primary dentition were the *age* of the patient (with the lowest frequency of necrosis in children less than two years (176), the *extent of displacement* as well as the *extent of loosening* of the luxated tooth, and finally – as in the permanent dentition – the presence of a *crown fracture* (77, 174–176).

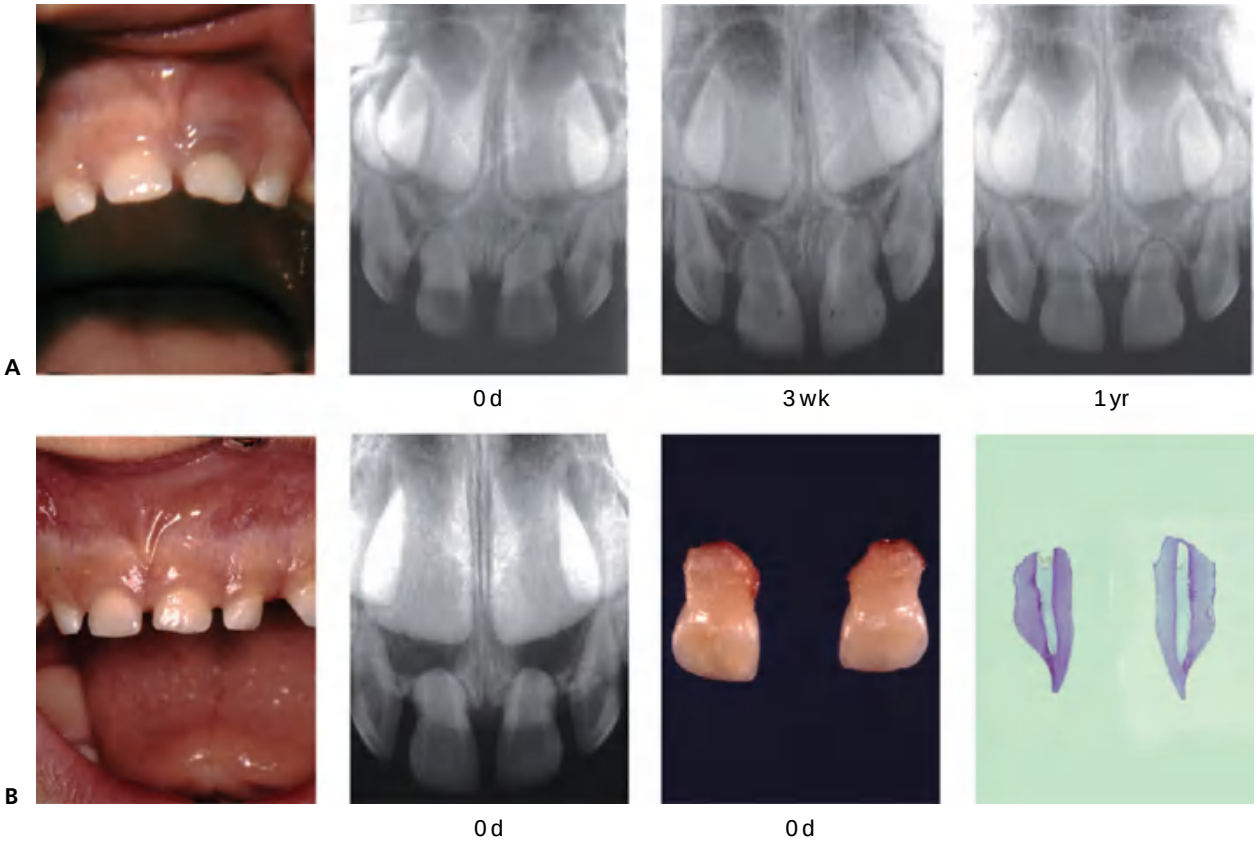



**Fig. 22.30** An expanding follicle simulates apical pathology of both primary central incisors subsequent to a luxation injury. A. The patient suffered a subluxation injury of the left central incisor at the age of 4. At the one-year control, apical rarefication/expansion of both follicles was found in the central incisor region. B. A month after this control, the patient suffered a new injury where both incisors became subluxated. At that time both incisors were extracted but showed normal histologic pulp conditions.

**Fig. 22.31** Apical radiolucency of a left central incisor developed one year after lateral luxation. Radiographic and clinical conditions are shown at time of injury and after 6 and 12 months.

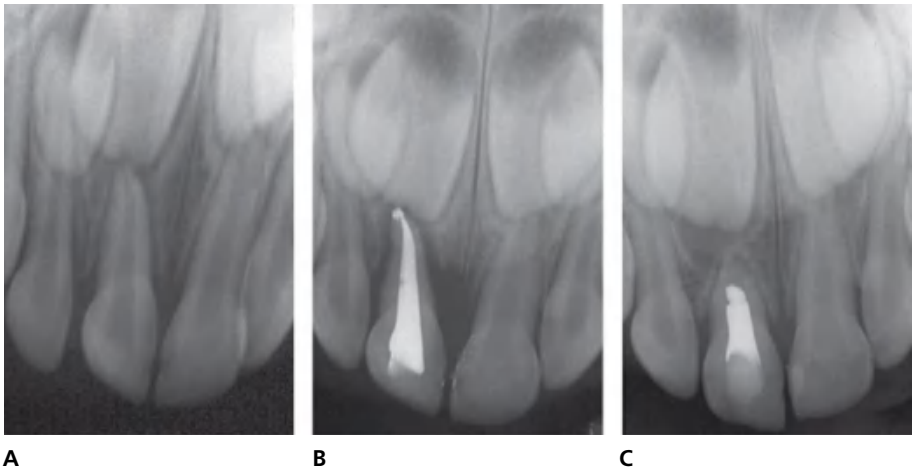



**Fig. 22.32** Endodontic treatment of primary central incisor with pulp necrosis. A. Radiograph taken two days after luxation of the maxillary right central incisor in a 4-year-old girl. B. Radiograph taken immediately after completion of endodontic treatment 4 weeks after the injury. Note the external root resorption and the expansion of the periodontal ligament space. C. Radiograph taken 12 months after the endodontic treatment. Note the resorption of the filling material from the root canal, the accelerated resorption of the root canal and the expansion of the dental follicle of the permanent successor. Clinically the tooth is asymptomatic.

Complication of pulp necrosis (pulp infection) is evident when conclusive clinical symptoms are present, including development of a fistula or acute inflammation with swelling and abscess formation. In the absence of these signs, sterile pulp necrosis may be present.

The diagnosis of pulp necrosis of primary teeth is based primarily on gray color changes of the crown and radiographic evidence of a periapical rarefaction and/or lack of physiologic continuous dentin apposition of the root. A therapeutic problem is whether, during the observation period, pulp infection may inflict damage to the permanent tooth germ. Admittedly, there is a long-term effect of pulp infection associated to a sinus tract and chronic periapical inflammation upon the developing permanent dental follicles (146) (see Chapters 2 and 25). However, an experimental study in monkeys has shown that pulp necrosis and periapical inflammation of primary incisors of only 6 weeks' duration do not lead to developmental disturbance of the permanent successors (8). Thus, a short observation period appears to be justified, thereby facilitating a diagnosis of pulp necrosis. In most cases root canal treatment is indicated. However, extraction of a primary tooth with periapical infection including abscess formation or fistula may also be considered.

### *Procedure for root canal treatment of traumatized primary incisors*

The tooth is isolated with a rubber dam without a clamp, the pulp chamber is exposed from the palatal aspect of the crown using a #330 tungsten bur on a high-speed hand piece with water spray. Local anesthesia is not used if the pulp is necrotic. If the patient complains of pain during insertion of the broach, local anesthetic is injected directly into the root canal. The pulp is removed, root canal cleaned, washed with 0.5% sodium hypochloride and saline and dried with paper points (147). A resorbable paste is used to fill the root canal with a spiral lentulo on a slow-speed hand piece, and the tooth is restored with a composite resin using the acid-etch technique. A postoperative radiograph is taken to assess the extent of root filling (Fig. 22.32).

### *Results of treatment*

In a study of endodontic treatment of traumatized primary incisors, a combination of an initial calcium hydroxide dressing and later obliteration with zinc oxide eugenol was used (149, 150). A success rate of only 64% was found, considering that all teeth had some kind of resorption and half of them were vital at the time of endodontic access, a finding that can hardly support the general use of this treatment procedure, especially considering the resources used in teeth that are going to be shed in the foreseeable future. Root canal treatment of primary incisors using zinc oxide eugenol as filling material, has shown success rates of 93% and 68% with a follow-up period of 18 months and 2 years, respectively (147, 148). In the latter study, failure of root canal treatment was associated with pre-intervention periapical lesions and pathological root resorption) (148).

In a study on the survival analysis of endodontically treated traumatized primary teeth, it was concluded that endodontic treatment enables the maintenance of the traumatized tooth up to its physiological resorption, and that trauma recurrence is a factor that leads to treatment failure (149). An important issue is whether endodontic treatment *per se* may induce developmental disturbances in the permanent dentition(110, 150, 151). Considering the effect of endodontic treatment on permanent successors, one clinical study found that root filling of primary teeth with zinc oxide eugenol resulted in 60% disturbance in enamel formation in the permanent successors compared with 21% in a non-treated group. However, the enamel disturbances were usually minimal in both groups (151).

Finally, a study has shown no difference in the long-term outcome for both the primary teeth and their permanent successors, whether mere observation therapy or root canal treatment of dark-gray discolored asymptomatic primary incisors was performed (139).

### *Predictors for pulp necrosis*

In a multivariate analysis on the long-term prognosis of injuries to primary teeth it was found that older age, presence of *dislocation* and *mobility* were predictors for pulp necrosis (77). It is important to keep in mind that pulp necrosis refers to a sterile pulp and its complication is usually associated to a sinus tract or fistula, mobility and periapical lesion.

Finally, it should be considered that re-eruption of luxated teeth occurs irrespective of whether pulp necrosis develops. Thus, a study on the fate of re-erupted intruded primary teeth showed that almost half of the cases erupted with a necrotic pulp (77).

### *Pulp canal obliteration*

Pulp canal obliteration (PCO) is a frequent finding, especially after luxation injuries in the younger age groups before physiologic root resorption is radiographically evident, usually around the age of 4–5 years (77, 82, 136, 140, 152). In a study from Brazil on clinical and radiographic records of 1675 traumatized primary teeth, PCO was found in 15% of all injured teeth. PCO appeared within the first 12 months, more frequent after repeated trauma and luxation injuries (153).

Even though there is a close relationship with yellow discoloration of the crown, radiographic evidence of PCO can often be seen in teeth with normal color and even in initially discolored teeth (Figs 22.29 and 22.33). In some cases, PCO was the only evidence of a previous trauma when seen on radiographs in coincidence with acute examination of a new injury.

Secondary pulp necrosis can occur in teeth with PCO; and frequencies of 0–13% have been reported (77, 140, 142). It is reasonable to assume that when a secondary necrosis occurs, it is related to a new trauma as an obliterated pulp has less healing potential due to the narrow pulp canal.

## External root resorption

This phenomenon is divided into physiologic root resorption and pathologic root resorption (26, 77).

### *Pathologic root resorption*

## External root resorption

This entity can be divided into *repair-related resorption* (surface resorption), *infection related resorption* (inflammatory resorption) and *replacement resorption* (ankylosis). In a long-term study of 387 traumatized primary incisors

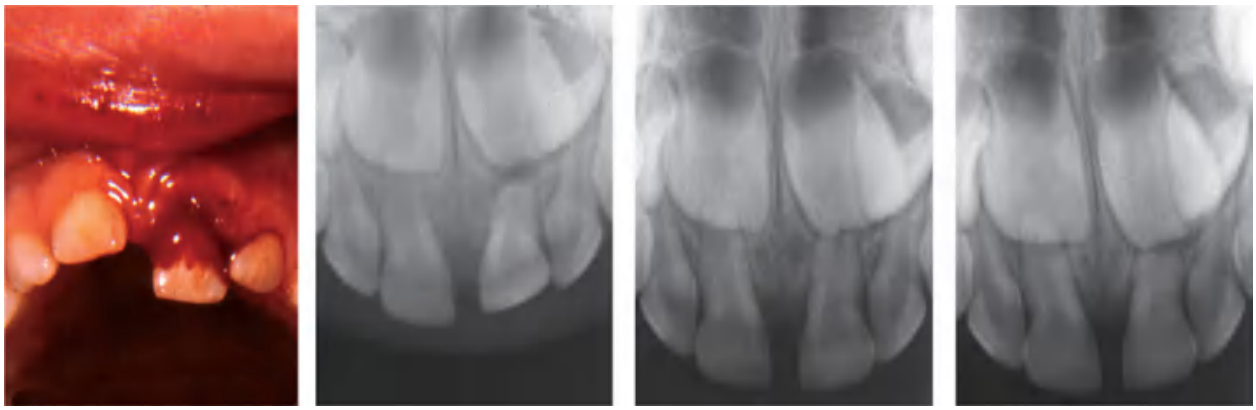


**Fig. 22.33** Pulp canal obliteration subsequent to lateral luxation of a primary central incisor.

repair-related resorption was found to be extremely rare (1%). Inflammatory resorption was found in 10% and was frequently related to intrusion and lateral luxation (77). In a study of discolored primary teeth after trauma, the frequency of inflammatory resorption was found in 11% of the cases (20). Another type of external root resorption in primary incisors, termed 'atypical root resorption', was found to be associated with traumatic dental injuries (154). It is defined as narrowing of the apical part of the root up to a certain level from where the coronal part retains its original width, and both parts are surrounded by periodontal ligament and alveolar bone (Fig. 22.35).

**Fig. 22.34** Clinical and radiographic illustration of internal root resorption.

**Fig. 22.35** 'Atypical root resorption' is defined as narrowing of the apical part of the root up to a certain level from where the coronal part retains its original width and both parts are surrounded by periodontal ligament and alveolar bone.

Finally, ankylosis was very rare, only affecting 0.8–4% of the luxated teeth, with a preference for intruded teeth (77, 175, 176). In case of ankylosis, spontaneous resorption by the erupting permanent successor was found to take place in most cases (Fig. 22.36).

In conclusion, external root resorption patterns seem to follow that of permanent teeth, although at a much lower rate (see Chapter 16). This can possibly be explained by the softer bone structure, which allows tooth displacement with fewer traumas to the root surface.

### Internal root resorption

This phenomenon is extremely rare; only two studies found internal root resorption in 2% of the cases (20, 77) (Figs 22.34 and 22.37).

In conclusion external or internal root resorption does not appear to be a significant problem in the management of primary tooth injuries. In case of inflammatory resorption either endodontic treatment or extraction should be performed. In case of replacement resorption, spontaneous shedding, although delayed, can be expected.

## Space loss after premature tooth loss

The need for space maintenance following early extraction of primary molars has been discussed in many publications (135, 162, 163). However, the risk of space loss due to premature loos of primary incisors gained very little attention in the dental literature, and when mentioned, it is based on personal experience and belief rather than evidence. Some authors advocated space maintenance if the primary incisor had been extracted before the age of 4 (164). Others thought that the risk of space loss exists only if the primary incisor has been removed or avulsed before the eruption of the canines. During eruption of the primary canines the remaining incisors are pushed mesially to close the space (135). Regardless if this is true or not, it is impractical to maintain the space at this age. Hargreaves (165) wrote that space loss in the anterior maxillary region occurs in crowded dentition only. Fig. 22.38 shows a case of space loss in a crowded anterior segment following premature loss of a maxillary primary incisor. It seems that the canines did not move mesially and remained in their original alignment. If there is at all space loss following premature loss of a primary incisor it seems to be negligible (77).

## Speech problems due to early loss of primary incisors

The importance of the dentition to speech production is well known (166) and especially that of the maxillary incisors to the /f/, /v/ and /s/ sounds (167). It is, therefore, reasonable to believe that early loss of maxillary incisors will result in speech impairment. Such speech problems were identified after extraction of primary incisors due to early childhood

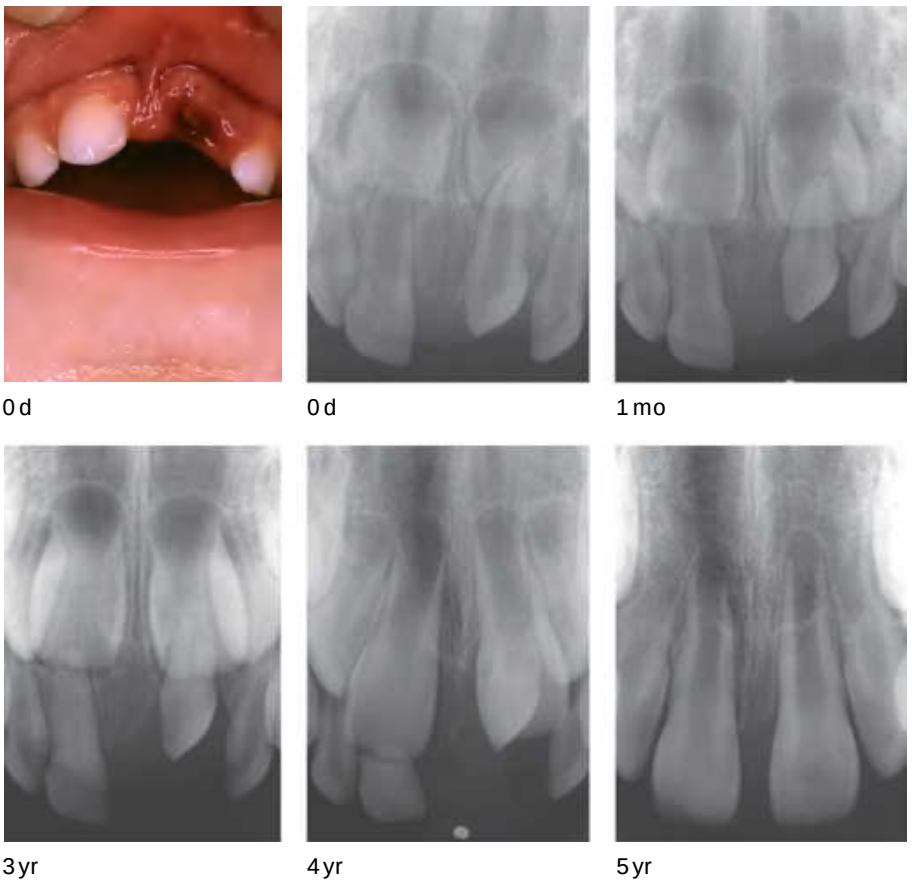



**Fig. 22.36** Ankylosis subsequent to intrusion of a central incisor in a 2-year-old girl. The intruded incisor became ankylosed, but the permanent incisor was able to shed the primary tooth.

**Fig. 22.37** Internal root canal resorption subsequent to a luxation injury.

caries (168). Articulation disorders were found both in children with premature extraction and in children with normal exfoliation of the primary incisors before the eruption of the permanent teeth (169). Such articulation disorders were found to decrease with age and disappear with the eruption of the permanent incisors (169, 170).

**Fig. 22.38** Space loss following premature loss of a central primary incisor in a dentition with crowding in the anterior region.

Parents who are concerned about possible speech impairment as a result of early loss of primary incisors can be assured that if such problem does develop it will be transient and dissolve after the eruption of the permanent successors.

## Essentials

### Epidemiology

Children at the age of 6 worldwide have a trauma experience to the primary dentition of 20–40%.

## Pathogenesis

Because of anatomical conditions and the common direction of traumatic impacts, primary teeth are usually displaced away from the permanent successor.

## Examination procedure

By clinical and radiographic means an exact position of the displaced primary incisor in the jaw in relation to the permanent successor should be made.

## Treatment principles

The important factor is to avoid inducing dental fear and anxiety in children and to safeguard the development of the permanent successor

## Enamel and enamel-dentin fractures

These fractures can be treated by grinding sharp edges or restoring with composite.

## Complicated crown fracture

If the child can cooperate, partial pulpotomy can be performed with due consideration to the degree of physiological resorption of the involved tooth. Otherwise, extraction must be performed.

## Crown-root fracture

Fragment removal only or extraction of the tooth are usually the treatments of choice.

## Root fracture

If the coronal fragment is not displaced the tooth can be left *in situ*. If the coronal fragment is very mobile and displaced, the coronal fragment should be extracted to prevent the child from swallowing or inhaling the loose fragment. The apical fragment is left for physiological resorption.

## Alveolar fracture

Reposition the displaced segment and splint it to adjacent teeth for up to 4 weeks. General anesthesia is often necessary. Follow-up minimum 1 year.

## Concussion and subluxation

No active treatment indicated. Careful oral hygiene is required, with a follow-up after 8 weeks.

## Extrusion

Depending on the extent of injury, the tooth may be repositioned and if possible splinted for 3 weeks, or leaving the tooth for spontaneous alignment.

If the tooth has suffered a severe extrusion, extraction is usually indicated to prevent the child from swallowing or inhaling the tooth. Follow-up: minimum 1 year.

## Lateral luxation

- Lateral luxated teeth should generally be allowed to realign spontaneously. However, if a lateral luxated tooth interferes with occlusion, repositioning should be attempted. After the use of local anesthesia, the tooth is repositioned and can be splinted.
- Clinical and radiographic follow-up after 1 and 2 months.
- Follow-up: minimum 1 year.

## Intrusive luxation

- Intruded primary teeth should generally be allowed to realign spontaneously. If a radiographic examination reveals that an intruded tooth has been forced into the follicle of the permanent tooth germ and has caused significant displacement of the tooth germ, careful decision making and expert opinion are mandatory before recommending the parents extraction of the primary tooth.
- If a conservative approach is chosen, the intruded tooth should be re-examined clinically and radiographically after 1 and 6 months to monitor healing.
- Follow-up: minimum 1 year.
- If the influence of trauma on the permanent successor must be evaluated, an additional follow-up examination is indicated at the age of 6.

## Avulsion

- A radiographic examination is essential to ensure that the missing tooth is not intruded.
- Avulsed primary teeth should not be replanted.

## Fractures secondary to chin injuries

If crown and crown-root fractures occur in the molar region, treatment will depend on the extent and severity of injuries.

## Complications in the primary dentition

There is no need for root canal treatment in dark discolored teeth when no other clinical or radiographic findings are present.

## Instructions to parents

- The affected area should be kept clean by brushing the child's teeth carefully after each meal with a soft toothbrush.
- Chlorhexidine should be applied topically twice a day for one week.
- The use of pacifiers and nursing bottles should be restricted.
- Dental care should be sought if new symptoms occur (e.g. fever, swelling, fistula, prolonged loosening or pain).
- The child must be brought for recall examination when invited.

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# 23

# Injuries to Developing Teeth

J. O. Andreasen, M. T. Flores & E. Lauridsen

## Terminology, frequency and etiology

Traumatic injuries to developing teeth can influence their further growth and maturation, usually leaving a child with a permanent and often readily visible deformity. Especially when the injury occurs during initial stages of development, enamel formation can be seriously disturbed due to interference with a number of stages in ameloblastic development, i.e., the morphogenetic, organizing, formative and maturation stages (1, 2).

The close relationship between the apices of primary teeth and the developing permanent successors explains why injuries to primary teeth are easily transmitted to the permanent dentition (Figs 23.1 and 23.2). Likewise, bone fractures located in areas containing developing tooth germs can interfere with further odontogenesis (3, 4). Also, the force of the impact may cause an underlying crown fracture of the unerupted permanent incisor (5)

The nature of these injuries has been studied clinically in humans (1, 2, 6–17) and experimentally in animals (18–33). Thus, anatomic and histological deviations due to injuries to developing teeth can be classified as follows (1):

- White or yellow-brown discoloration of enamel
- White or yellow-brown discoloration of enamel with circular enamel hypoplasia
- Crown dilaceration
- Odontoma-like malformation
- Root duplication
- Vestibular root angulation
- Lateral root angulation or dilaceration
- Partial or complete arrest of root formation
- Sequestration of permanent tooth germs
- Disturbance in eruption

In this classification, the term *dilaceration* describes an abrupt deviation of the long axis of the crown or root portion of the tooth. This deviation originates from a traumatic non-axial displacement of already formed hard tissue in relation to the developing soft tissue (1).

The term *angulation* denotes a curvature of the root resulting from a gradual change in the direction of root development without evidence of abrupt displacement of the tooth germ during odontogenesis (1).

In 2015 an excellent systematic review was published by Lenzi and coworkers, and a series of studies were identified where reliable data was presented (34). The prevalence of such disturbances, secondary to dental injuries in the primary dentition, ranges from 8 to 69%, according to the studies listed in Table 23.1. Considering the frequency of traumatic injuries to primary teeth (see Chapter 11), it is apparent that enamel hypoplasia of traumatic origin must be rather common (35). In a clinical study, it was estimated that 10% of all enamel hypoplasias affecting anterior teeth in schoolchildren in Copenhagen were related to trauma in the primary dentition (7). The type of *dental trauma* sustained apparently determines the type and degree of developmental disturbance. Avulsion and intrusive luxation represent injuries with very high frequencies of developmental disturbance, while subluxation and extrusion represent low-risk groups (Table 23.2) (2, 3, 9–11, 13, 17, 36–46). Furthermore, the age at the time of injury is of major importance; thus, fewer complications are seen in individuals over 4 years of age than in individuals in the younger age groups (2, 40, 42, 45) (Table 23.3). Furthermore, intrusion of rat molars has shown severe malformation in root development (47).

According to the studies listed in Table 23.4, the frequency of developmental disturbances due to *jaw fractures* ranges from 19 to 68%. Furthermore, the frequency of developmental

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**Fig. 23.1** Skull of a 5-year-old child revealing the relationship between primary and permanent teeth.

**A** **B**

**Fig. 23.2** A. Anatomic relationship between the primary incisor and its permanent successor in a human skull. Note that both central and lateral primary incisors are in close relation to the permanent central incisor. B. Histologic relationship between a primary incisor and its permanent successor. Note that most of the resorbing primary root is separated from the permanent tooth germ by only a thin layer of soft tissue. Source: Reproduced from (31) with permission.

**Table 23.1** Frequency of disturbances in development of permanent teeth after traumatic injuries to the primary dentition.

| Examiner | No. of teeth involved | No. of teeth with developmental disturbances |
|---|---|---|
| Zelnner (48) 1956 | 26 | 3 (12%) |
| Schreiber (49) 1959 | 42 | 8 (19%) |
| Taatz (50) 1962 | 33 | 7 (21%) |
| Ravn (51) 1968 | 40 | 23 (57%) |
| Lind et al. (6) 1970 | 72 | 36 (50%) |
| Selliseth (36) 1970 | 128 | 88 (69%) |
| Andreasen & Ravn (2) 1971 | 213 | 88 (41%) |
| Andreasen & Ravn (7) 1973 | 147 | 85 (58%) |
| Mehnert (8) 1974 | 28 | 10 (36%) |
| Watzek & Skoda (9) 1976 | 77 | 39 (51%) |
| Ravn (10) 1976 | 100* | 54 (54%) |
| Brin et al. (12) 1984 | 56 | 72 (47%) |
| Morgantini et al. (15) 1986 | 60 | 15 (25%) |
| Ben Bassat et al. (14) 1985 | 414 | 190 (46%) |
| Ishikawa et al. (52) 1990 | 126 | 72 (57%) |
| von Arx (17) 1991 | 144 | 33 (23%) |
| von Arx (53) 1993 | 255 | 59 (23%) |
| Soporowski et al. (39) 1994 | 169 | 7 (8%) |
| Sleiter & von Arx (37) 2002 | 74 | 24 (32%) |
| Christophersen et al (40) 2005 | 44* | 10 (30%) |
| Sennhenn-Kirchner et al (41) 2006 | 81 | 20 (25%) |
| Spinas et al. (54) 2006 | 130* | 32 (25%) |
| Da Silva Assuncao et al (42) 2009 | 620 | 126 (20%) |
| Calvalho et al. (55) 2010 | 221 | 84 (69%) |
| Altun et al (43) 2009 | 138 | 74 (54%) |
| do Espirito Santo Jacomo et al (44) 2009 | 174 | 89 (51%) |
| Scerri et al (56) 2010 | 67 | 35 (52%) |
| de Amorim et al (45) 2011 | 241 | 54 (22%) |
| Skaare et al (57, 58) 2015 | 300 | 66 (22%) |
| Andreasen (46) 2018 | 421 | 184 (43%) |

*Only intrusions.

**Table 23.2** Frequency of disturbances in development of permanent teeth according to type of injury to the primary dentition.

| Type of injury | No. of teeth | No. of teeth with developmental disturbances |
|---|---|---|
| Subluxation | 94 | 32 (34%) |
| Extrusive luxation | 23 | 9 (39%) |
| Lateral luxation | 106 | 49 (46%) |
| Avulsion | 48 | 31 (65%) |
| Intrusive luxation | 68 | 46 (65%) |

Source: From Andreasen et al. (46) 2018.

**Table 23.3** Frequency of developmental disturbances of permanent teeth related to the age at which injury to the primary dentition occurred.

| Age (years) | No. of teeth | No. of teeth with developmental disturbances |
|---|---|---|
| 1 | 43 | 13 (30%) |
| 2 | 119 | 24 (20%) |
| 3 | 57 | 15 (26%) |
| 4 | 79 | 14 (18%) |
| 5 | 81 | 22 (27%) |
| 6 | 37 | 9 (24%) |
| 7 | 7 | 1 (14%) |

Source: From Andreasen et al. (46) 2018.

**Table 23.4** Frequency of developmental disturbances among permanent teeth involved in jaw fractures.

| Examiner | No. of teeth involved | No. of teeth with developmental disturbances |
|---|---|---|
| Fischer (62) 1951 | 12 | 7 (58%) |
| Lenstrup (63) 1955 | 22 | 15 (68%) |
| Taatz (50) 1962 | 29 | 14 (48%) |
| Bencze (64, 65) 1964 | 10 | 5 (50%) |
| Ideberg & Persson (59) 1971 | 100 | 19 (19%) |
| Ridell & Åstrand (66) 1971 | 170 | 6 (35%) |
| Ranta & Ylipaavalniemi (67) 1973 | 37 | 19 (51%) |
| Perko & Pepersack (61) 1975 | 35 | 16 (46%) |

**Fig. 23.3** A 14-year-old patient with an impacted mandibular permanent canine due to osteosynthesis at the age of 5. Source: Reproduced from (61).

disturbances has been found to be related to fragment displacement at the time of injury (59, 60). Treatment of jaw fractures by osteosynthesis has also been shown to increase damage to developing teeth (61) (Fig. 23.3).

Oral surgical procedures can also induce dental malformations. Thus, patients operated for cleft palate show a very high frequency of enamel defects in the primary as well as the permanent dentitions. Histologic findings in these cases indicate that the surgical trauma could be a contributing factor (68–70). Exodontia has also been recorded among surgical etiologic factors. Due to the close relationship between the developing crowns of the permanent premolars and the roots of their primary predecessors, developing premolars are especially prone to disturbances in enamel and dentin formation resulting from extraction of primary

molars (71) (Fig. 23.4). Also, root canal infection can influence enamel formation of the permanent successor (72–78) (see Chapter 2).

Evaluation of the full extent of complications following injuries sustained in early childhood must await complete eruption of all involved permanent teeth, a problem which should be considered in the case of legal action or insurance claims. However, most serious sequelae (i.e. disturbances in tooth morphology) can usually be diagnosed radiographically within the first year after trauma (2).

Injuries which occur very early can also interfere with the development of the primary dentition (79). Thus, a study of prematurely born and intubated infants revealed enamel hypoplasias affecting the primary dentition in 18–80% of the children (79–87), usually on the left side of the maxilla corresponding to the customary placement of the tube (79, 81, 82) (Fig. 23.5). A later autopsy study has supported the theory that compression of the alveolar process during endotracheal intubation in neonates is the causative factor which can elicit these disturbances in primary tooth development (88, 89) (Fig. 23.6).

Finally, it should be borne in mind that a number of other pathological conditions may result in enamel hypoplasia (e.g. fluorine, rickets, hypoparathyroidism, exanthematous fevers, severe infections and metabolic disturbances such as celiac disease and acidosis). However, these are unfortunately not entirely pathognomonic, but often overlapping in their expression (90, 91). A descriptive classification system for developmental defects of enamel (DDE index) (92) and a modified Index of DDE were developed (93) which, to a certain degree, incorporate some of the pathological features for various developmental disturbances and both are the most commonly used in multifactorial analysis of enamel defects (58, 93–99). Besides, an index for scoring all forms of tooth discoloration and surface defects has been developed (100).

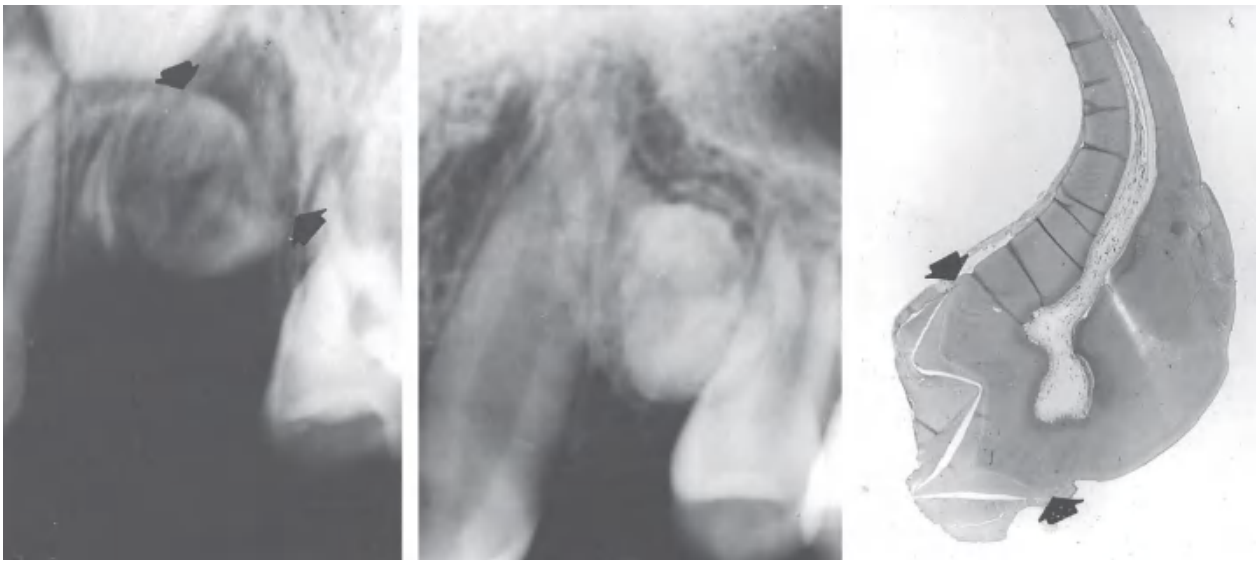
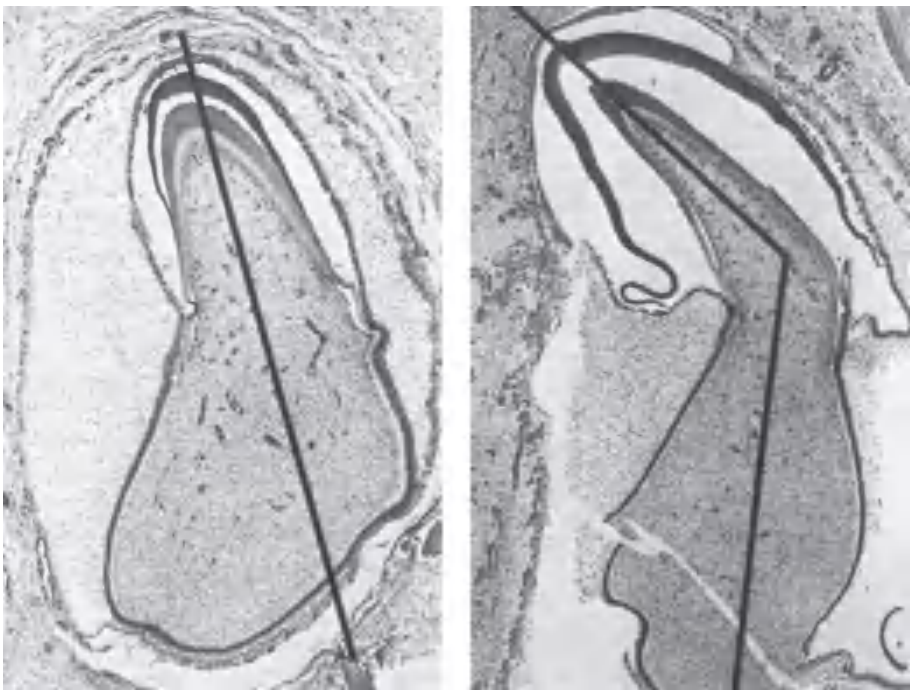



**Fig. 23.4** Crown dilaceration and impaction of a first premolar following extraction of the first primary molar. A. Radiograph taken immediately after extraction of the primary molar at the age of 4 years. The permanent tooth germ appears to be dislocated (arrows). B. Radiograph at the age of 14 years. As the first premolar was impacted, it was decided the tooth be removed. C. Low power view of sectioned premolar. Slight crown dilaceration is seen (arrows). ×5.

**Fig. 23.5** Enamel hypoplasias caused by a laryngoscopy and/or prolonged endotracheal intubation. The enamel defects are usually located on the left side of the maxilla due to the selective pressure in this area by the laryngoscope. Source: Reproduced from (81).



A B

**Fig. 23.6** Dilaceration of primary tooth germ due to endotracheal intubation. This 3-day-old child was intubated postpartum due to respiratory distress but died after 3 days. A. At necropsy the unaffected part of the alveolar process shows a normal primary incisor tooth germ. B. Sections from the deformed alveolar ridge where the tube has rested shows disruption of the tooth germ with deviation of the long axis and a resultant cystic space around the enamel matrix. Source: Reproduced from (88).

## Clinical, radiographic and pathologic findings

Pathologic changes in permanent tooth germs have been studied experimentally in intrusions of primary teeth in monkeys (31) and in rat molars (47). Immediate changes consisted of contusion and displacement of the reduced enamel epithelium and slight displacement of the hard dental tissue in relation to the Hertwig's epithelial root sheath (see Chapter 2). After 6 weeks, metaplasia of the reduced enamel epithelium into a thin stratified squamous epithelium took place. In most cases, changes in morphology of the dentin and/or enamel matrices were seen (see Chapter 2).

Developmental disturbances of human teeth caused by trauma have been studied in humans (1) and can be divided into the following types:

### White or yellow-brown discolorations of enamel

These lesions appear as sharply demarcated, stained enamel opacities, most often located on the facial surface of the crown; their extent varies from small spots to large fields (1, 2, 7, 14, 46, 48, 49, 51, 57, 101–115) (Fig. 23.7A–C). These color changes are usually not associated with clinically detectable defects in the enamel surface; however, some cases can present such defects (1) (Fig. 23.7D). In this context, it should be mentioned that white enamel discolorations with a diameter of 3 mm are frequent in teeth without a history of trauma (7, 85, 94, 98, 99, 116).

The frequency of these lesions has been reported to be 23% following injuries to the primary dentition (2), commonly affecting maxillary incisors, with the age of patients at the time of injury ranging from 1 to 3.5 years (42, 44, 45, 56, 58). The stage of development of the permanent tooth germ at the time of injury may vary (14). No specific type of injury is especially related to this group of lesions.

Similar disturbances in enamel formation may be seen in developing teeth involved in jaw fractures (63, 64) (Fig. 23.8). For the sake of completeness, it should be mentioned that color changes with or without a defect in the enamel surface may occur as a sequel to periapical inflammation of primary teeth, (50, 73–78, 117–121) giving rise to so-called Turner teeth.

The nature of white enamel discolorations has been studied by means of microradiography and polarized light microscopy, as well as transmission and scanning electron microscopy (Figs 23.9, 23.10 and 23.11). Findings from these investigations indicate that the trauma interferes with enamel mineralization, while matrix formation is apparently not involved (1, 33) (Figs 23.10 and 23.11). An experimental study in monkeys indicated that these areas develop corresponding to areas where trauma has altered the reduced enamel epithelium into a flattened stratified squamous epithelium (see Chapter 2) (31).

Radiographic examination prior to tooth eruption will usually not reveal defective mineralization. Consequently, these disturbances can only be diagnosed clinically after complete eruption (2).

### White or yellow-brown discoloration of enamel with circular enamel hypoplasia

These lesions are a more severe manifestation of trauma sustained during the formative stages of the permanent tooth germ (1, 2, 7, 32, 45, 46, 52, 105, 122–131). The typical

**Fig. 23.7 Discoloration and morphologic changes secondary to injuries to the primary dentition**

A. Slight white enamel discolorations involving both central incisors. B. Slight white and yellow-brown staining of the enamel of both central incisors. Source: Reproduced from (1).

**Marked color changes and enamel hypoplasia**

C. Marked color changes of permanent left central and lateral incisors associated with distal inclination of the crown of the central incisor. D. Yellow-brown enamel changes of central incisor associated with a cavity in the enamel surface.

**Yellow-brown discoloration and circular enamel hypoplasia**

E. Slight circular enamel hypoplasia apical to a yellow-brown discoloration of enamel with an external cavity in a central incisor (arrow). F. Same changes as in E; note the same positional relationship between hypoplasia and area of discoloration (arrows).

**Severe malformation**

G, H. Severe disturbances in development of three permanent incisors. Right central incisor is partly impacted due to malformation, left central incisor shows marked color changes. A horizontal enamel hypoplasia is especially evident in H (see also Fig. 23.14 which shows the condition after orthodontic realignment of the right central incisor).

finding in this group, which distinguishes these lesions from those in the first group, is a narrow horizontal groove which encircles the crown cervical to the discolored areas (Fig. 23.7E–H). In some cases, an external defect is found centrally in the coronally placed white or yellow-brown lesions (Fig. 23.7E).

The frequency of this type of change has been reported to be 12% following injuries to the primary dentition (2). Maxillary central incisors are usually involved; the age at the time of injury is usually 2 years. The stage of development of the permanent tooth germ varies from half to complete crown formation at the time of injury. As a rule, the injury to the primary tooth is either avulsion, extrusive or intrusive luxation (1, 43, 128–132).

Radiographic examination of these teeth reveals a transverse radiolucent line at the level of indentation and usually a radiolucent area corresponding to the coronally placed enamel defect (Fig. 23.12). This type of developmental disturbance can usually be diagnosed before eruption (2).

It should be noted that the enamel changes are confined to the site of coronal mineralization at the time of injury (Fig. 23.13). Although the pathogenesis of color changes in

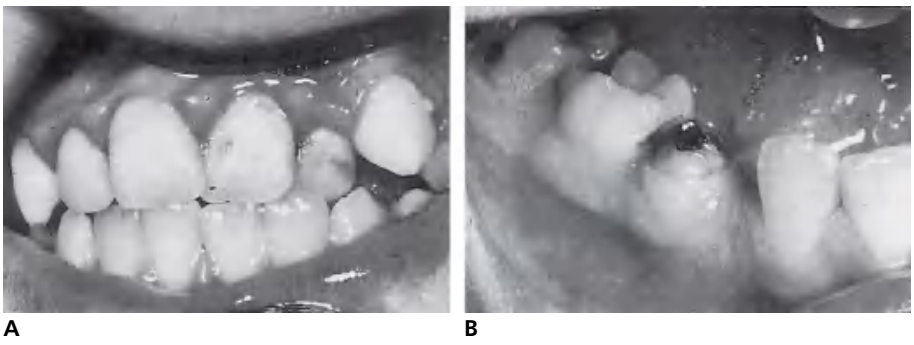



**Fig. 23.8** A. Enamel discolorations affecting left central and lateral incisors following a fracture of the maxilla at the age of 4 years. B. Disturbed enamel formation following a jaw fracture at the age of 3 years. Defective enamel covering the occlusal surface of the lower first premolar. Source: Reproduced from (63) with permission.

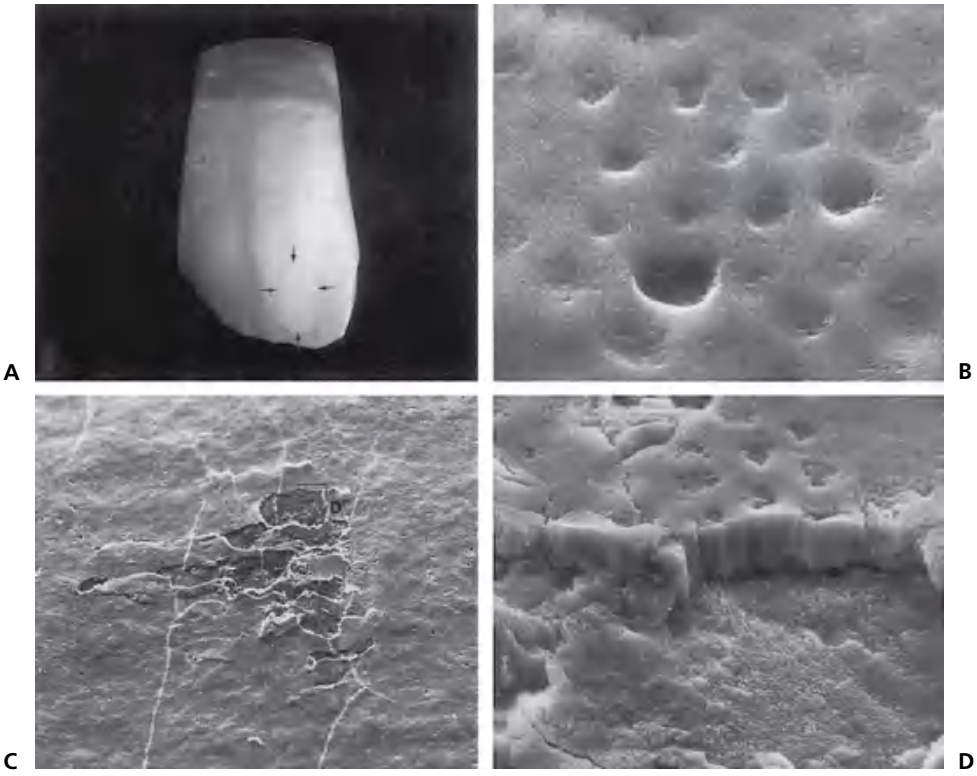



**Fig. 23.9** A. Maxillary right permanent central monkey incisor 6 weeks after traumatic intrusion of the primary incisor. Note the circumscribed white discoloration of the enamel (arrows). B. Scanning electron micrograph of the adjacent non-affected enamel. The pits indicate arrest of matrix formation. ×3300. C. General view of white enamel discoloration showing rupture of the enamel surface. ×110. The enclosed area is shown at a higher magnification in D. ×2400. Source: Reproduced from (33) with permission.

enamel has not yet been completely clarified, it is assumed that the displaced primary tooth traumatizes tissue adjacent to the permanent tooth germ and possibly the odontogenic epithelium, thereby interfering with final mineralization of enamel. The configuration of the resulting hypomineralized area closely coincides with the outline of normal progressive 'secondary' mineralization. The lesions are usually white; however, blood breakdown products in the traumatized area can seep into areas of mineralization during further enamel formation. This could explain why yellow-brown areas are located exclusively apical to the white lesions (1) (Fig. 23.7 B). Surface defects in the enamel most probably reflect direct injury to the enamel matrix before mineralization has been completed (Figs 23.13 and 23.14).

Experiments in monkeys have shown that circular enamel hypoplasia represents localized damage to the ameloblasts in their formative stages due to traumatic displacement of already formed hard tissue in relation to the developing soft tissues (31) (Figs 23.15 and 23.16).

## Crown dilaceration

These malformations are due to traumatic non-axial displacement of already formed hard tissue in relation to the developing soft tissues (1, 2, 17, 46, 51, 103, 107, 133–167). Three per cent of the injuries to primary teeth result in this type of malformation (2).

Due to their close contact to the primary incisors, crown-dilacerated teeth are usually maxillary or mandibular central incisors (1, 168–174). Approximately half of these teeth become impacted, whereas the remaining erupt normally or in facio- or linguo-version. Injury to the primary dentition usually occurs at the age of 2 years, with a range of from less than 1 year to 5 years. Most often the injury occurs at a time when up to half the crown has been formed, a finding possibly related to the anatomy of the developing tooth germ which allows tilting of the tooth germ within its socket (175). The trauma to the primary dentition which can result in crown dilaceration is usually avulsion or intrusion (1).

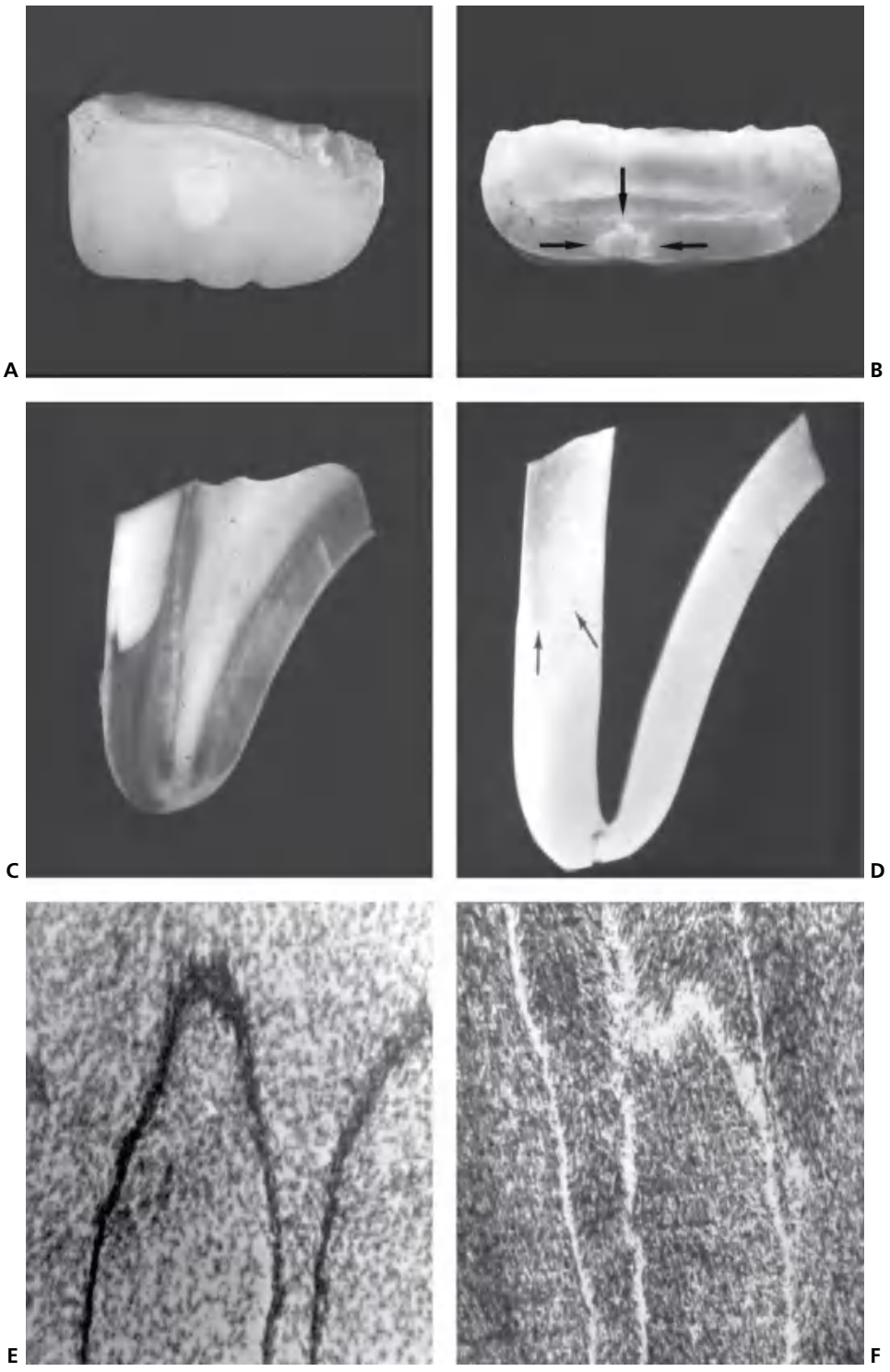



**Fig. 23.10** White enamel discoloration affecting the left central permanent incisor secondary to a dental injury at the age of $1^1/_2$ years. A. Macroscopic frontal view of specimen consisting of the incisal part of the crown. B. Axial view of the fracture surface; note the extension of the white lesion (arrows). C. Ground section of the specimen photographed in incident light. ×15. D. A microradiograph of the same section reveals that the clinical white area is less mineralized (arrows) than surrounding non-involved enamel. ×15. E. Electron micrographs of decalcified non-involved enamel. The black areas represent organic material. ×8,000. F. Electron micrograph of clinical white area; note that this area contains an increased amount of organic stroma. ×8,000. Source; Reproduced from (1) with permission.

The pathology of crown-dilacerated teeth supports the theory of displacement of the enamel epithelium and the mineralized portion of the tooth in relation to the dental papilla and cervical loops (1, 157–161) (Figs 23.17 to 23.19). This results in the loss of enamel on a part of the facial surface of the crown. On the lingual aspect, a cone of hard tissue is formed which projects into the root canal, while the lingual cervical loop forms an enamel-covered cusp (1). A pathogenesis of displacement of the non-mineralized portion of the tooth in the socket is supported by radiographic findings immediately after injury where a tilting of the tooth germ can be seen (see Chapter 22).

The deviation of the coronal portion varies according to the tooth's location. Maxillary incisors usually show lingual deviation, while lower incisors are usually inclined facially (1).

Radiographically, unerupted crown-dilacerated teeth are seen as foreshortened coronally.

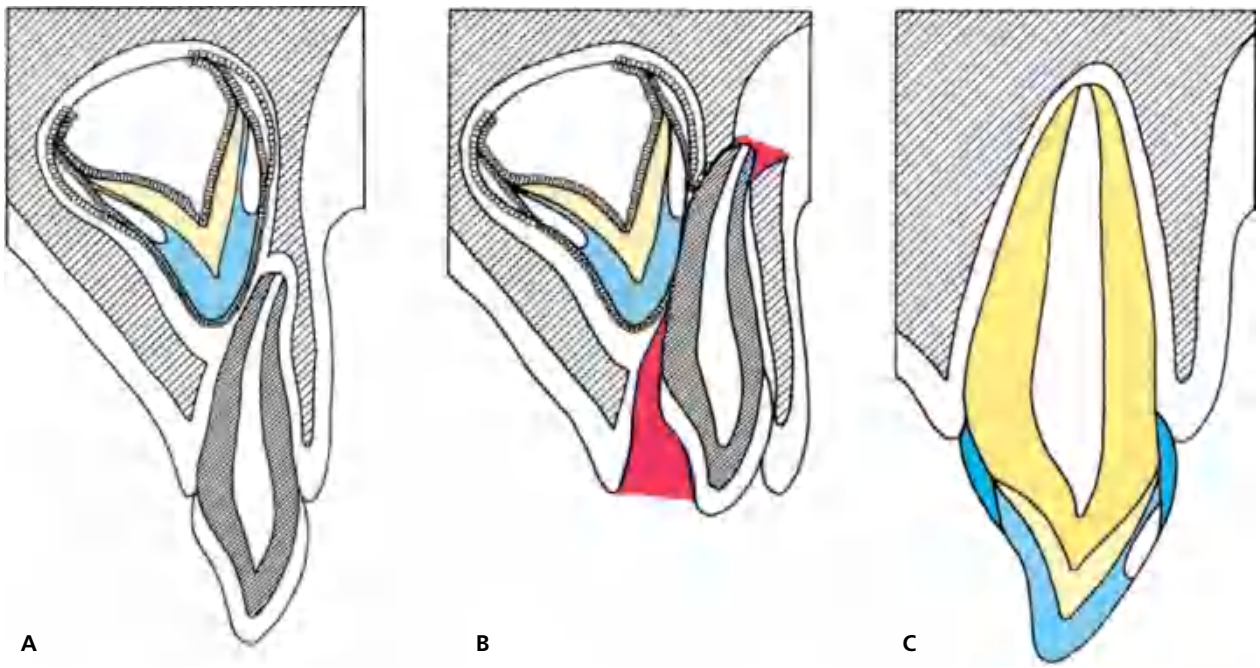



**Fig. 23.11** Schematic drawing illustrating the mechanism of white or yellow-brown discolorations of enamel. A. Condition before injury. B. Primary incisor intruded through the labial socket wall resulting in moderate damage to the reduced enamel epithelium of the permanent successor. C. As a result, enamel maturation is arrested in the area with affected enamel epithelium, creating a white or yellow-brown enamel discoloration.

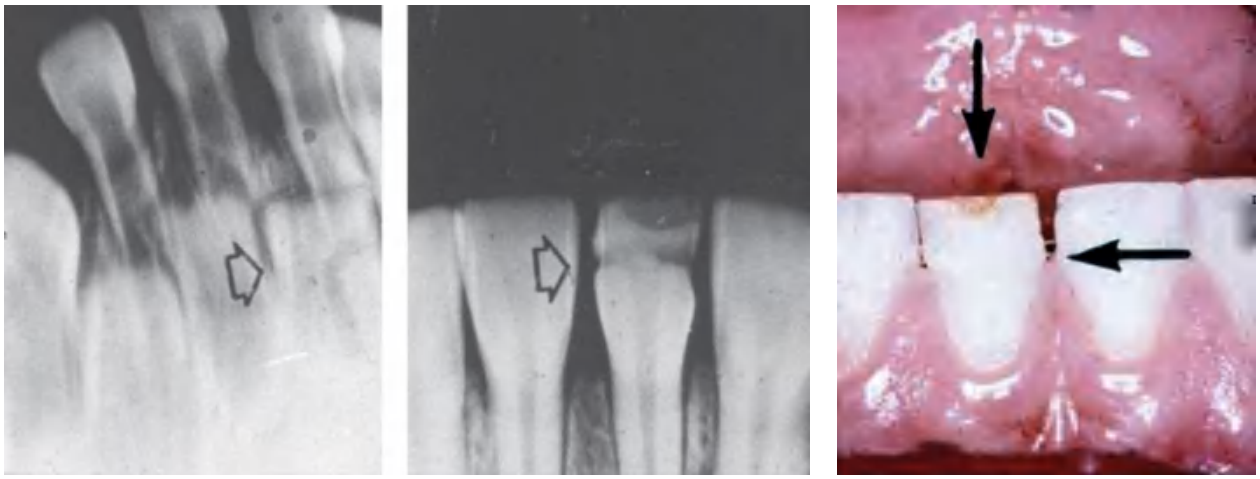



**Fig. 23.12** Discoloration and circular enamel hypoplasia of a permanent incisor following injury to the primary dentition. A. Slight intrusive luxation of left central primary incisor at the age of 1 year. Note the degree of mineralization of the permanent incisor (arrow). B. and C. Radiographic and clinical condition at the age of 12 years. Note mineralization disturbances of the incisal edge as well as the circular hypoplasia related to the part of the tooth formed at time of injury (arrow). Source: Reproduced from (1) with permission.

## Odontoma-like malformations

These malformations are rare sequelae to injuries in the primary dentition. Reported cases are confined primarily to maxillary incisors (1, 16, 17, 35, 105, 111, 131, 135, 139, 152, 176–184). The age at the time of injury ranges from less than 1 year to 3 years (131, 181, 183, 184). The type of injury affecting the primary dentition appears to be intrusive luxation or avulsion (1) (Fig. 23.20). The histology and radiology of these cases show a conglomerate of hard tissue, having the morphology of a complex odontoma or separate tooth elements (Figs 23.21 to 23.23). Experimental evidence supports the theory that these malformations occur during early phases of odontogenesis and affect the morphogenetic stages of ameloblastic development (19, 20, 25, 185) (Figs 23.22 and 23.23). The traumatic origin of these malformations is further supported by the observation that similar changes have been reported after ritual extractions of primary canines in peoples in Africa (186) (Fig. 23.24) as well as sequelae to extraction of primary molars due to pulpal complications (71).

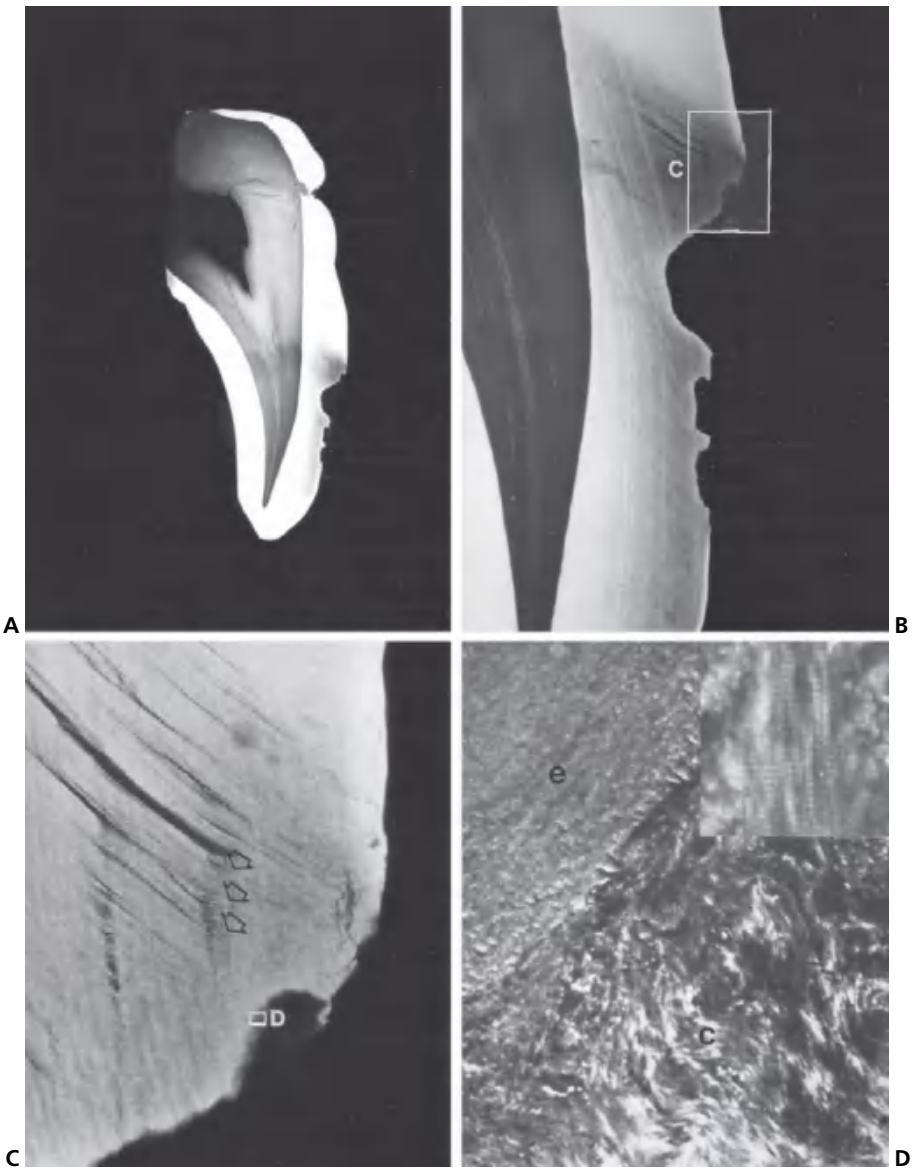



**Fig. 23.13** Microradiographic and electron microscopic findings in a permanent central incisor with crown dilaceration and a yellow-brown enamel discoloration. The patient sustained intrusion of the primary incisor at the age of 2 years. A. Microradiograph of ground section. B. Higher magnification of the enamel defect. Note the radiolucency in this area, indicating a decrease in mineral content. C. Note angulation of enamel prisms (arrows), possibly the result of a direct trauma sustained during matrix formation. The scalloped external surface possibly indicates resorption before deposition of new hard tissue. D. Electron micrograph showing transition between the injured enamel in the cavity (e) and cementum-like tissue (c) deposited in the defect. ×10000. Insert, in upper right corner, a higher magnification of the cementum-like tissue showing cross-banded fibers (collagen). ×20000. Source: Reproduced from (1) with permission.

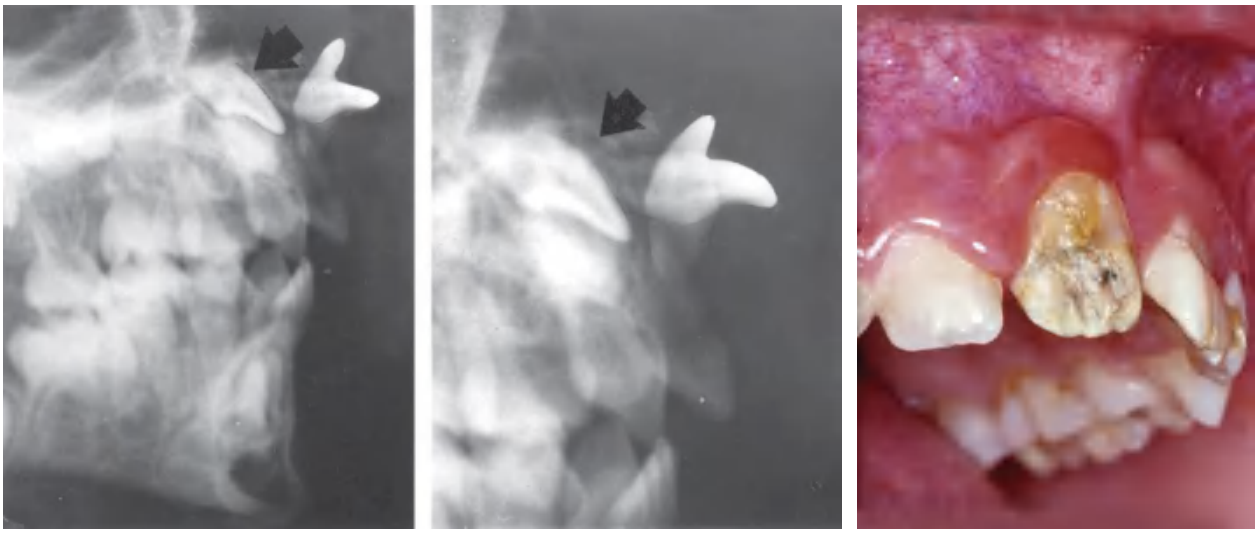



**Fig. 23.14** Severe disturbances in tooth development caused by intrusive luxation of both central incisors at the age of 3 years. A. Lateral projection showing displaced primary incisors (arrow). B. Enlarged view of A. Note that one of the displaced teeth (the right central incisor) faces the facial surface of the permanent tooth germ (arrow). C. Clinical view after surgical exposure and orthodontic realignment of right permanent central incisor. Note the hypoplastic enamel corresponding to previous trauma region (see also Fig. 23.7, G and H). Source: Reproduced from (1) with permission.

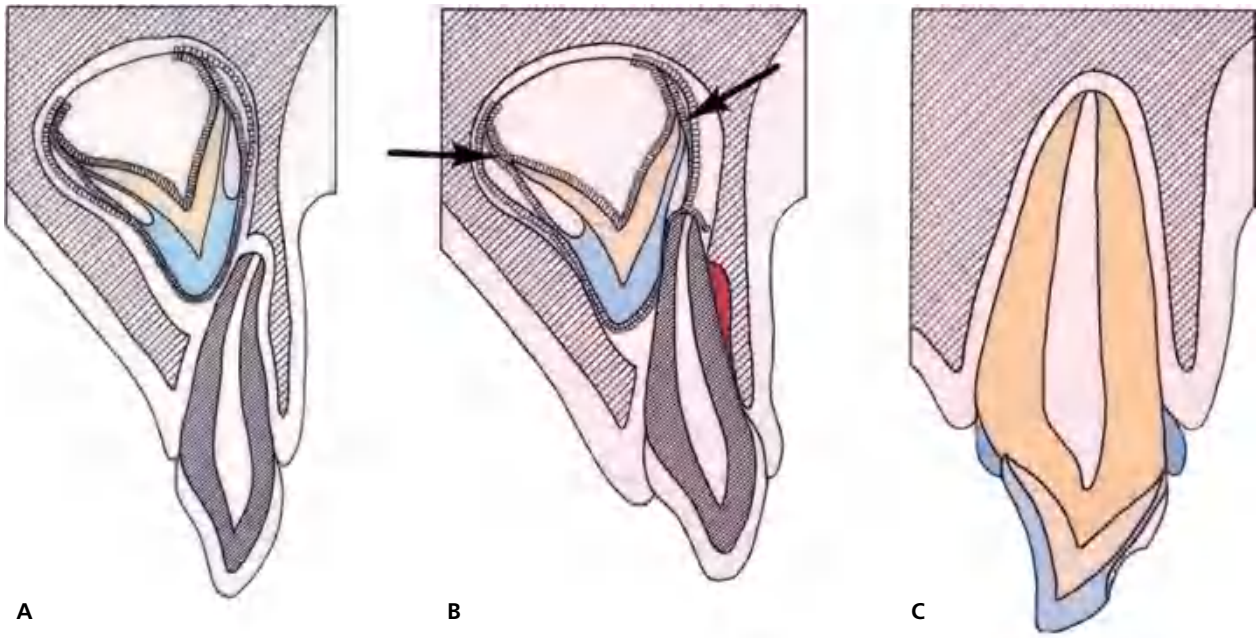



**Fig. 23.15** Schematic drawing illustrating the possible mechanism of enamel discoloration and circular enamel hypoplasia. A. Condition before injury. B. Slight axial dislocation of the already formed part of the tooth in relation to the remaining tooth germ. C. While enamel formation is resumed apical to the site of dislocation from the intact cervical loops, no further enamel formation occurs coronal to this level. Thus, a horizontal indentation is created between enamel formed before and after injury. Source: Reproduced from (1) with permission.

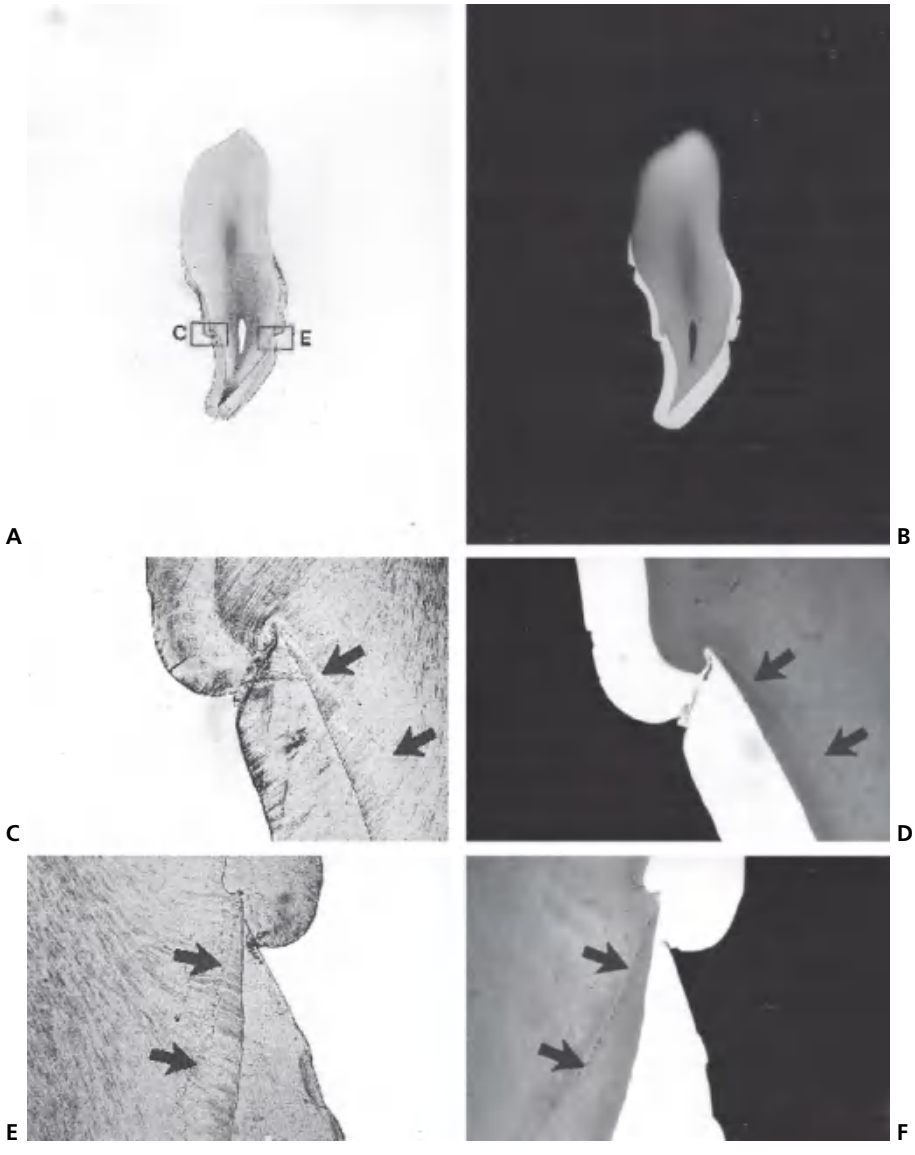



**Fig. 23.16** Yellow-brown discoloration of enamel and circular enamel hypoplasia of a maxillary lateral incisor as a result of avulsion of the primary predecessor at the age of 3 years. A. Ground section of extracted incisor. ×3. B. Microradiograph of the same section. ×3. C. and D. Higher magnification of lingual surface of the crown. Note a slight axial displacement and the calcio-traumatic line in the dentin revealing disturbance in dentin formation immediately after injury (arrows). ×30. E. and F. Higher magnification of facial surface of the crown. The same findings are present in this area as described above. ×30. Source: Reproduced from (1) with permission.

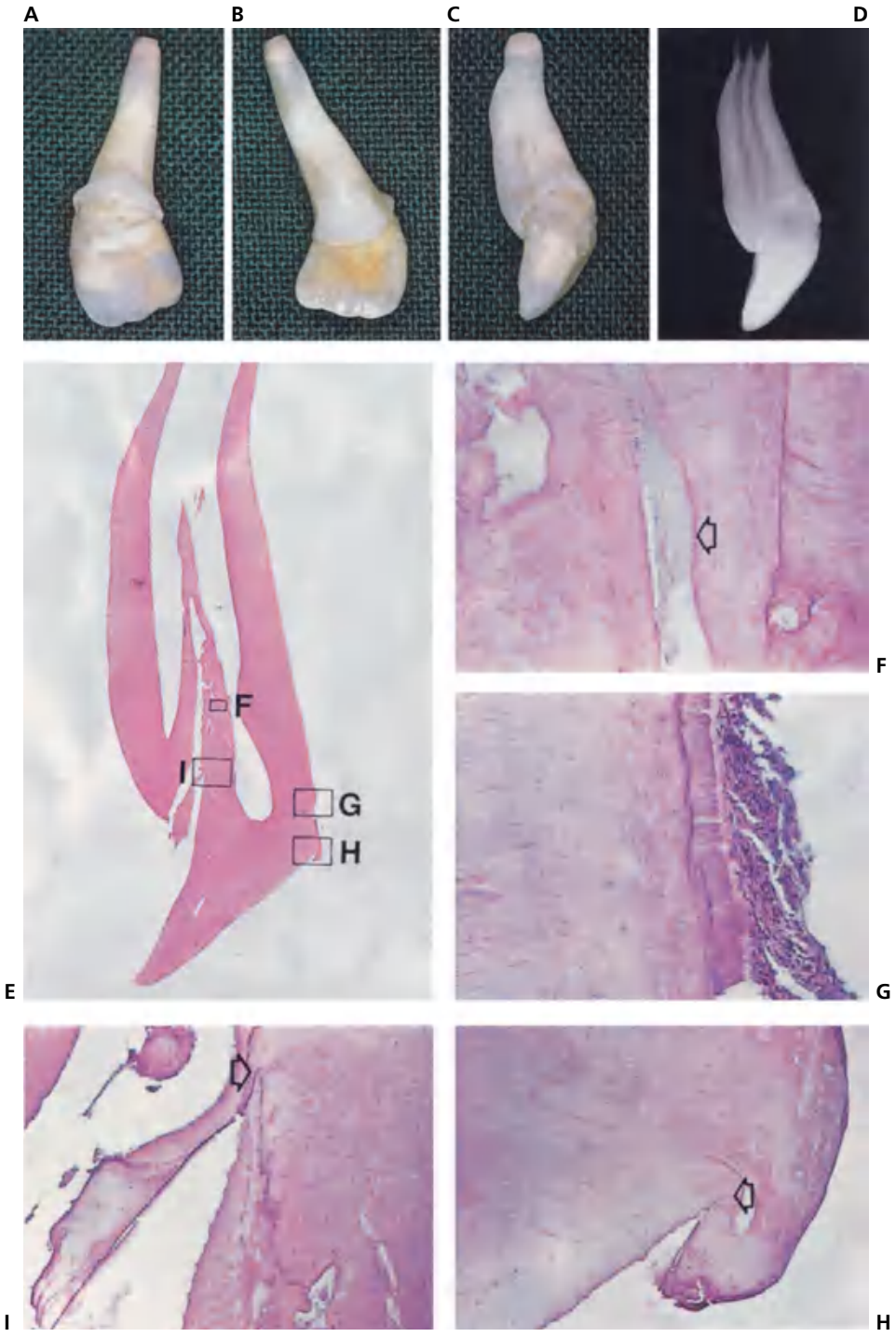



**Fig. 23.17** Clinical, radiographic, and histologic features of a crown dilacerated maxillary central incisor. The patient sustained intrusive luxation of the corresponding primary incisor at the age of $2^{1}/_{2}$ years. A to C. Clinical appearance of extracted incisor. Note area on the facial surface of the crown without enamel and the white and yellow-brown discoloration of the enamel. D. A radiograph discloses an internal cone of hard tissue. E. Low power view of sectioned incisor. ×4. F. Internal hard tissue cone consisting of enamel matrix (arrow) and dentin. ×195. G. Enamel-free crown area covered with cementum. ×195. H and I. Dilaceration area facially and lingually. Arrows indicate the margins of hard tissue formed at the time of injury. ×75. Source: Reproduced from (1) with permission.

Radiographically there is a radio-opaque mass with little resemblance to a tooth germ. Sometimes, however, a relatively normal root may be seen (Fig. 23.22). Fig. 23.25 illustrates the possible mechanism of formation.

## Root duplication

This is a rare occurrence, seen following intrusive luxation of primary teeth (1, 130, 131, 168, 187–190). This complication is usually the result of an injury at the time when half or less than half of the crown is formed (1) (Fig. 23.26). The pathology of these cases indicates that a traumatic division of the cervical loop occurs at the time of injury, resulting in the formation of two separate roots (187) (Fig. 23.27).

Radiographically, a mesial and distal root can be demonstrated which extends from a partially formed crown (Figs 23.26 and 23.27).

**Fig. 23.18** Microradiographic features of crown-dilacerated tooth shown in Fig. 12.17. A. Ground section. ×5. B. Microradiograph. ×5. C. Enamel covers the lingual cusp (arrows). ×30. D. Irregular dentin formed immediately after injury (arrows). ×30. Source: Reproduced from (1) with permission.

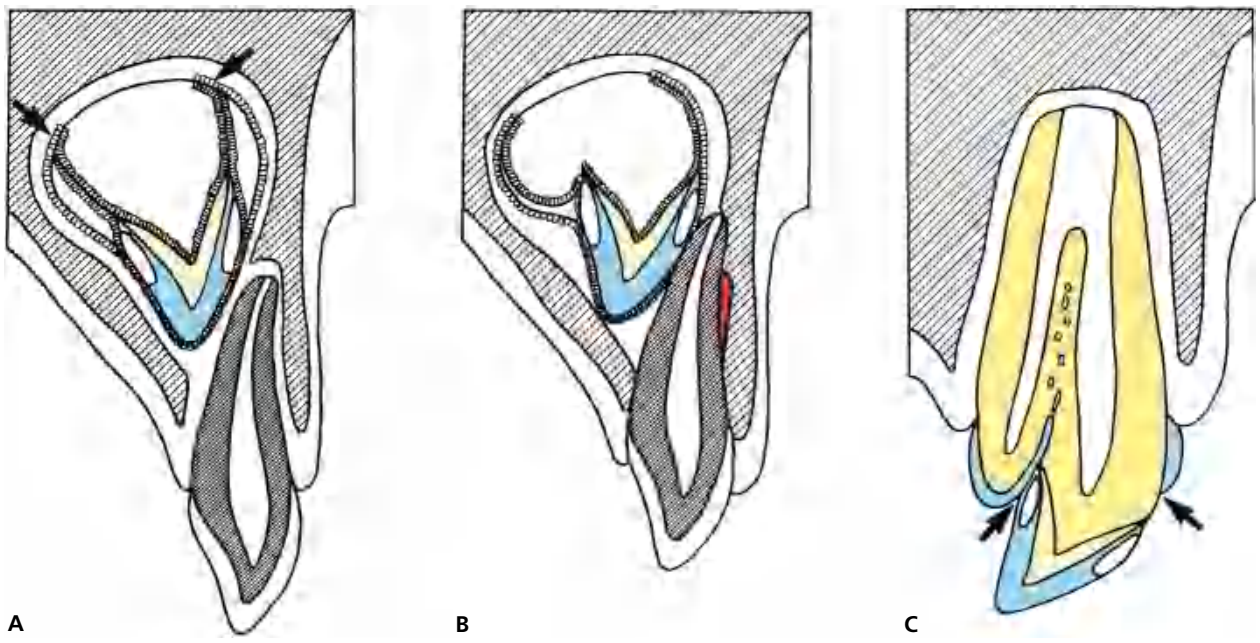



**Fig. 23.19** Schematic drawing illustrating the possible mechanism of crown dilaceration. A. Condition before injury (arrows indicate the cervical loops). B and C. Non-axial dislocation of the already formed part of the tooth in relation to the dental papilla, inner and outer dental epithelium and the cervical loops. Facially, the stretched inner enamel epithelium is able to induce differentiation of new odontoblasts, but its enamel forming capacity is not expressed. Consequently, a horizontal band of dentin will be left without enamel facially. The facial cervical loop has either not been injured or has been completely regenerated, thus explaining normal amelogenesis apical to the trauma site (arrow). On the lingual aspect of the crown, the displaced inner enamel epithelium and ameloblasts form a cone of hard tissue projecting into the pulp canal. Presumably the ameloblasts stripped from their matrix proliferate and induce dentin as well as enamel matrix formation. The presumably intact lingual cervical loop forms an enamel-covered cusp (arrow). Source: Reproduced from (1) with permission.

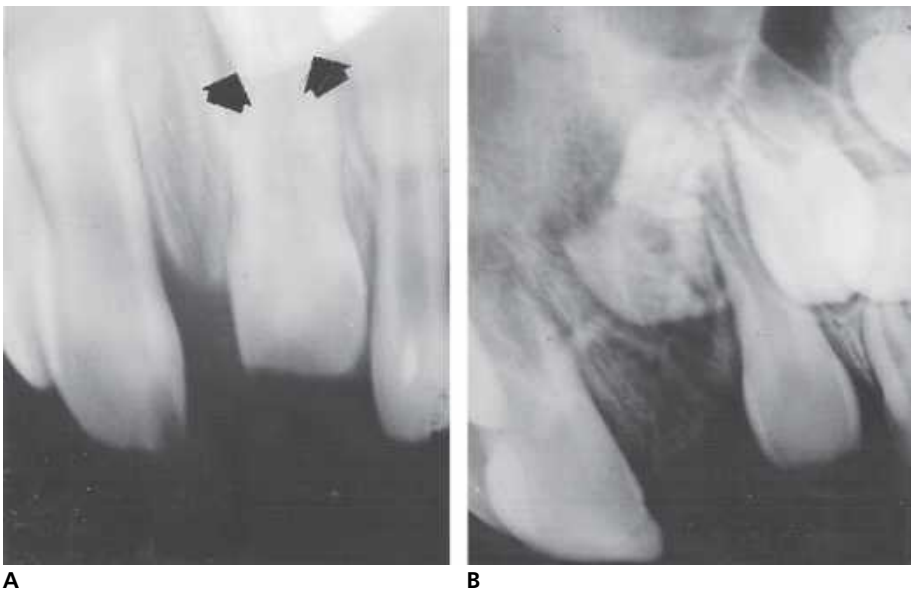



**Fig. 23.20** Odontoma-like malformation of permanent tooth germ due to intrusive luxation of primary left central incisor at the age of 2 years. A. Condition at time of injury. Note the outline of the calcified portion of the permanent tooth germ (arrows). B. Radiograph taken at the age of 8 years. An odontoma-like malformation is found at the site of the central incisor. Source: Reproduced from (1) with permission.

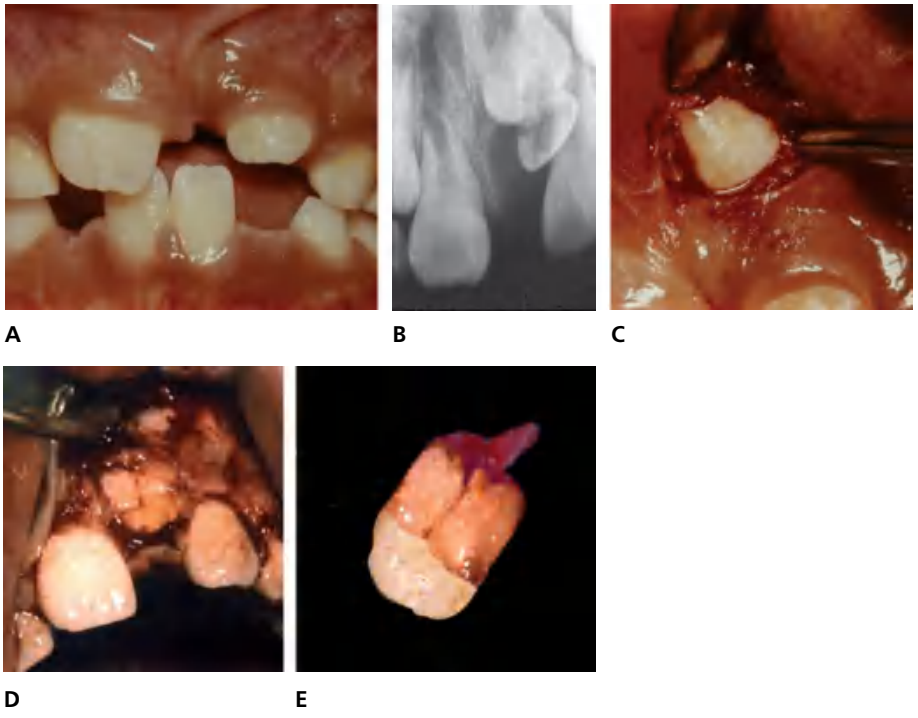



**Fig. 23.21** Odontoma-like malformation of a permanent tooth germ due to intrusive luxation of primary left central incisor at the age of 8 months. A. Clinical condition at the age of 8 years. The left central incisor has not erupted. B. The radiograph reveals intrusion and ankylosis of the primary incisor. An odontoma-like malformation is found at the site of the central incisor. C. Surgical removal of the ankylosed primary tooth. D. and E. Surgical removal of the permanent incisor which shows severe malformation. Frontal view of removed odontoma.

## Vestibular root angulation

This developmental disturbance appears as a marked curvature confined to the root as a result of an injury sustained at the age of 2 to 5 years (1, 101, 106, 107, 155, 159, 191–203). The malformed tooth is usually impacted and the crown palpable in the labial sulcus (202, 203). The only teeth demonstrating this malformation are maxillary central incisors. The injuries to the primary dentition consist of intrusive luxation and avulsion (1).

The histopathologic findings in these cases consist of a thickening of cementum in the area of angulation, but with no sign of acute traumatic changes in the hard tissue formed (1, 159, 192) (Fig. 23.28). The loss of a primary incisor can present an obstacle in the eruption of the developing tooth, forcing it to change its path of eruption in a labial direction (Figs 23.28, 23.29). Presumably Hertwig's epithelial root sheath remains in position despite the impact and thereby creates a curvature of the root (1). It should be mentioned, however, that the traumatic origin of this malformation has been questioned. Thus, in a study of 29 teeth, Stewart (166) found no history of trauma. Moreover, this type of malformation was six times more frequent in girls than in boys. According to Stewart, the most likely explanation for facial root angulation was ectopic development of the tooth germ (166).

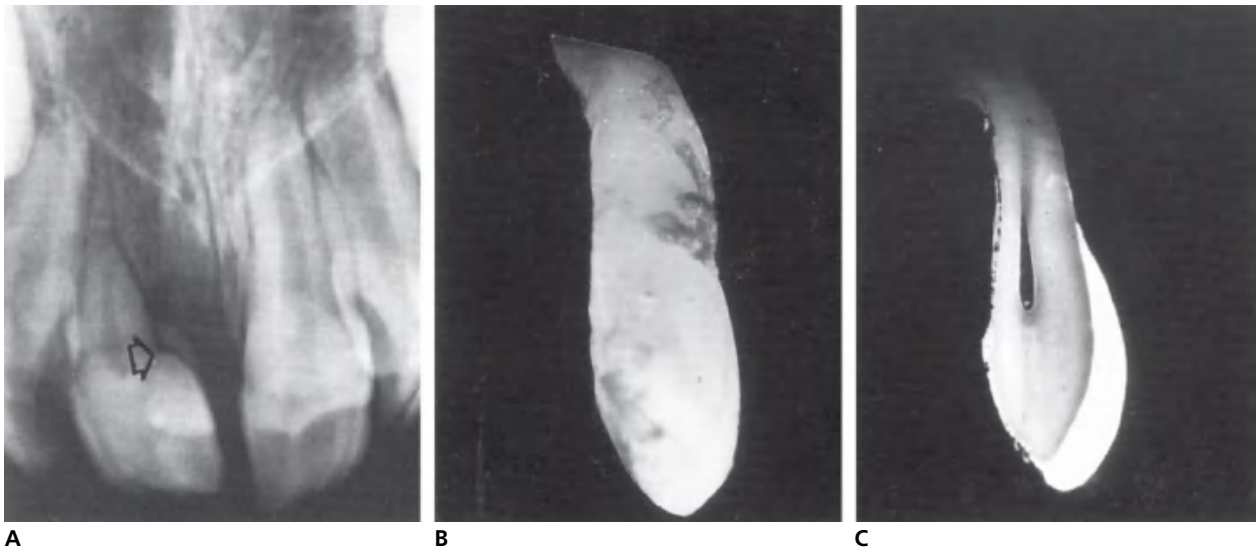



**Fig. 23.22** Development of a separate tooth element following intrusive luxation of primary incisors at the age of 3 years. A. Radiographic condition at the age of 8 years. Note the small tooth element in the central incisor region (arrow). B. Lateral view of removed odontoma. C. Microradiograph reveals an enamel layer covering part of the tooth element. Source: Reproduced from (1) with permission.

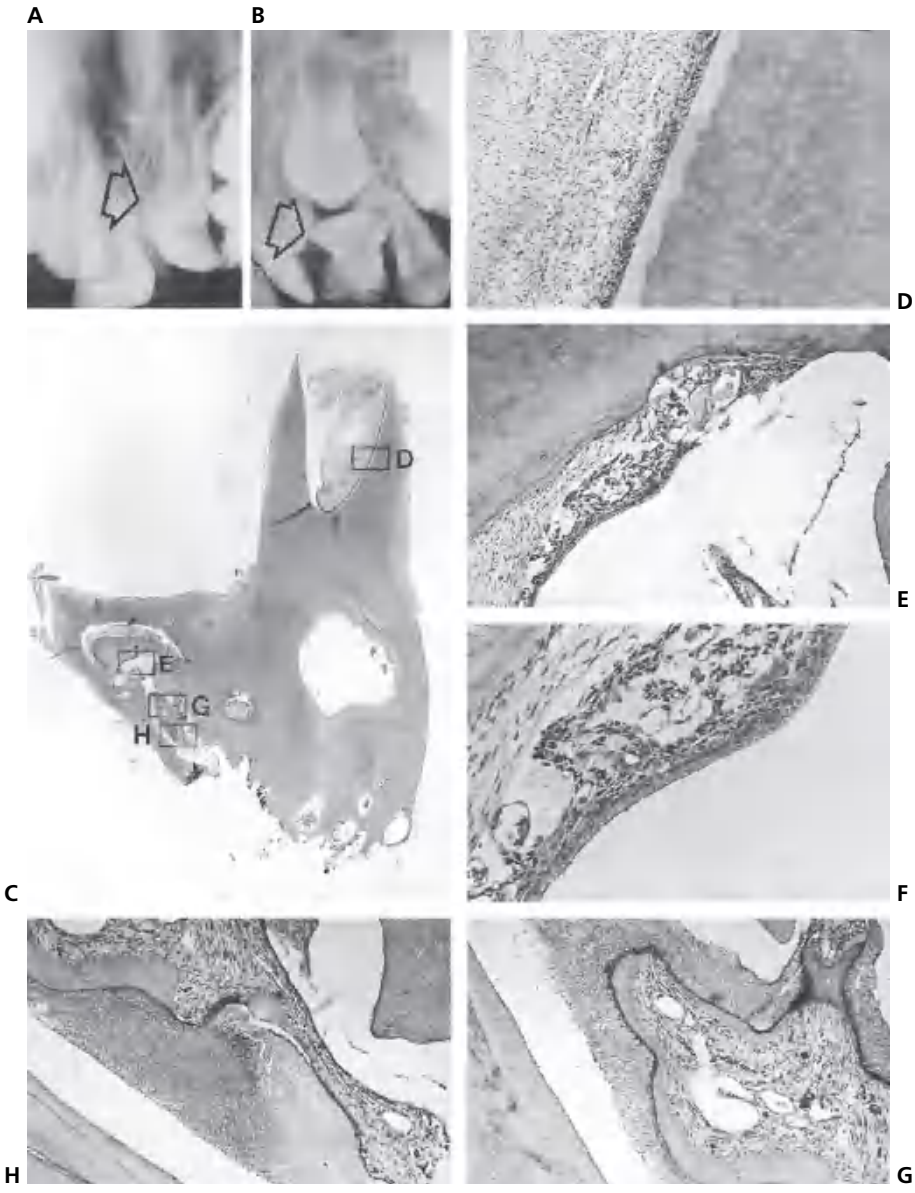



**Fig. 23.23** Odontoma-like malformation of a permanent lateral incisor following avulsion of the primary lateral incisor at the age of 2 years. A. Left central incisor shows a marked crown dilaceration (arrow). B. Radiograph revealing odontoma-like malformation of left lateral incisor (arrow). C. Low power view of sectioned lateral incisor. ×7. D. Apical part of the odontoma bears similarities to a normal root. Note intact odontoblast layer. ×75. E. Odontoma-like enamel inclusions – most of the enamel was lost during demineralization. ×75. F. Reduced enamel epithelium. ×195. G and H. Preserved enamel matrix. ×75. Source: Reproduced from (1) with permission.

**Fig. 23.24** Malformation of permanent teeth following ritual extractions of lower primary canines in an African population. A. In place of the left canine, two apparently separate tooth elements have erupted. B. Severe crown malformation of lower right permanent canine. Source: Reproduced from (186) with permission.

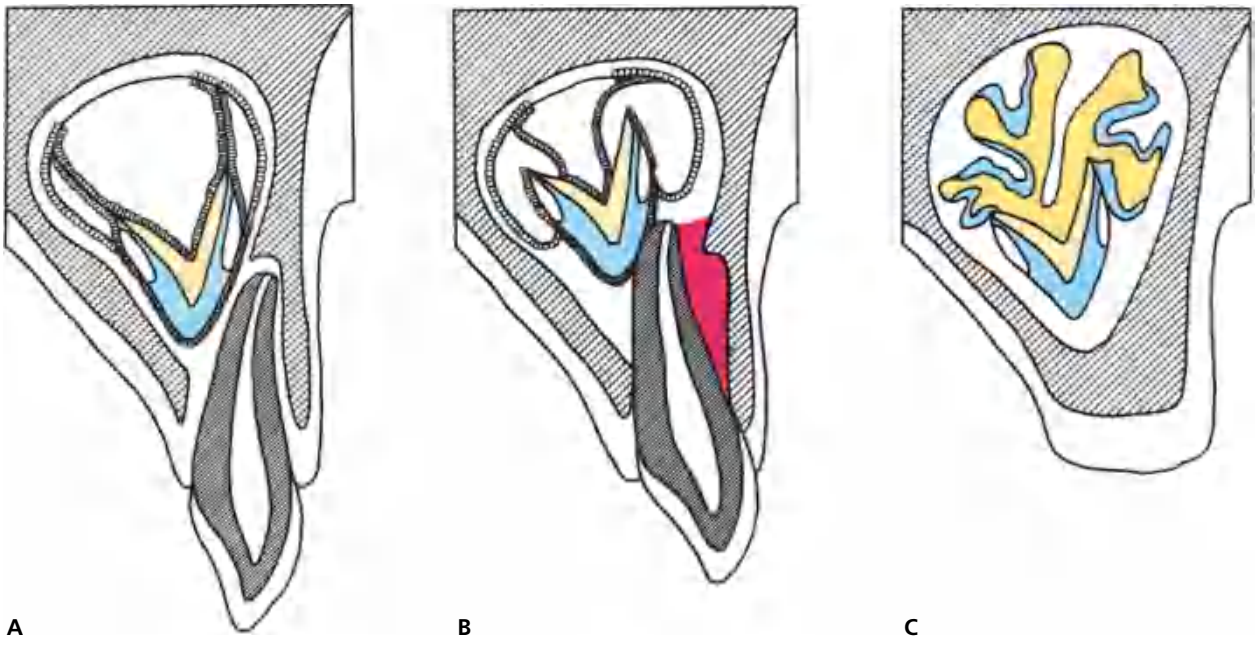



**Fig. 23.25** Schematic drawing illustrating possible mechanism for odontoma-like malformation. A. Condition before injury. B. Axial dislocation of the primary incisor with extensive damage to the permanent tooth germ. C. Formation of an odontoma-like malformation.

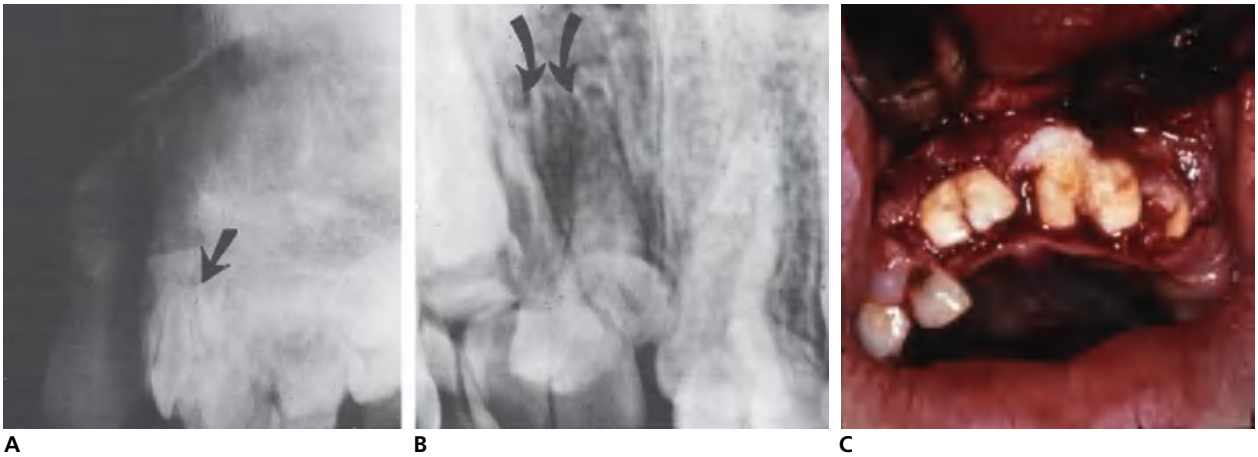



**Fig. 23.26** Root duplication of 2 central incisors following intrusive luxation of both primary incisors at the age of 2 years. A. Lateral radiograph at time of injury. Note the displaced primary incisors (arrow). B. Radiograph taken at the age of 9 years. The root is divided into a mesial and distal portion in both incisors (arrows). C. After surgical exposure, abnormal root development is evident as well as vertical fractures of the crown of both central incisors (arrows), presumably a result of the previous injury.

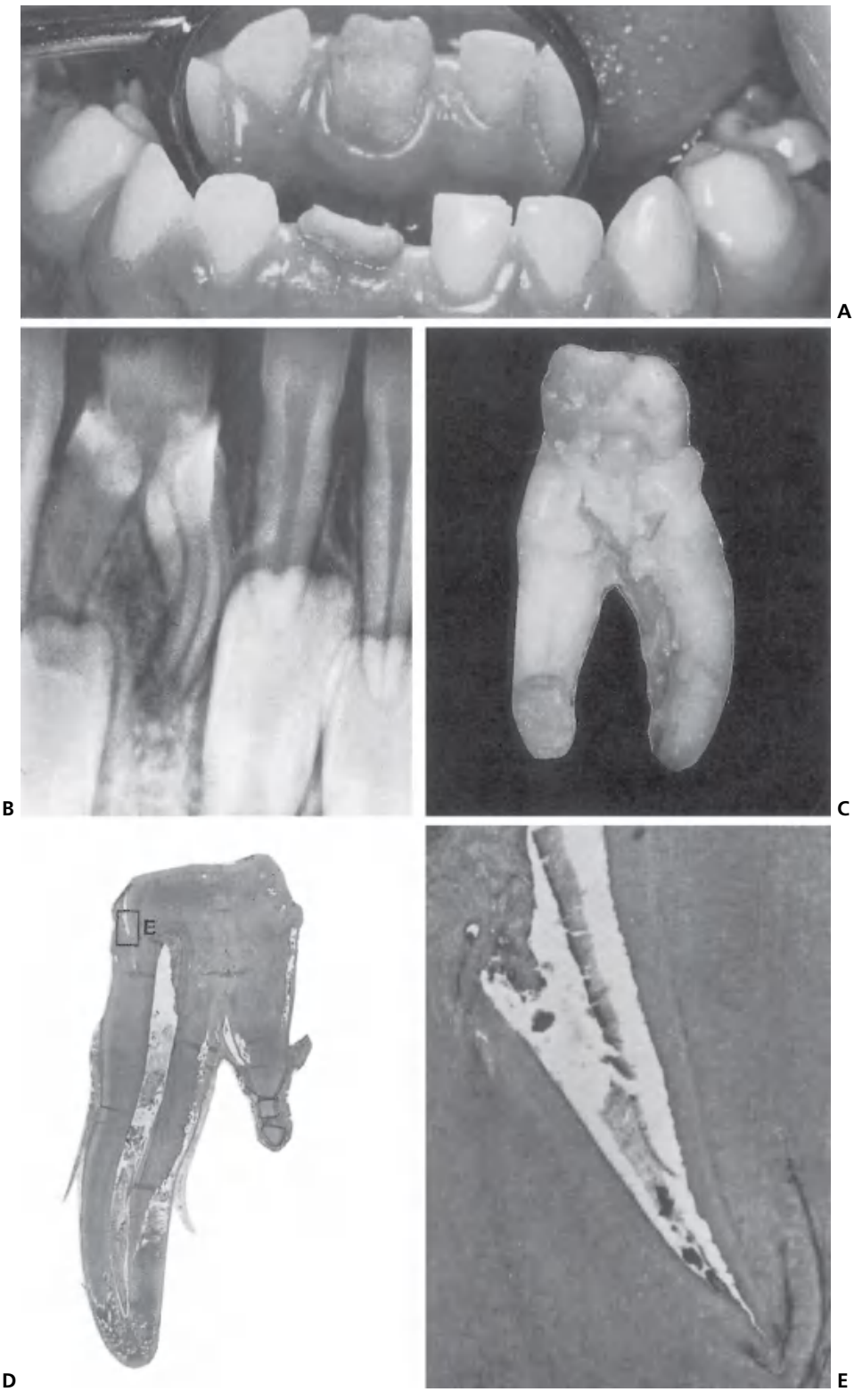



**Fig. 23.27** Histologic features of root duplication of a right permanent central incisor due to an injury sustained at the age of 6 months. A. Clinical appearance. B. Radiograph showing root division. C. Frontal aspect of the extracted tooth. D. Low power view of sectioned incisor. E. Higher magnification of D indicates that the incisal part of the crown has been intruded. The cleft is lined with enamel. The dentinal tubules are compressed at the bottom of the cleft. Source: Reproduced from (187).

Radiographically a root-angulated tooth appears foreshortened. Lateral projections can more precisely localize the position of the tooth within the jaw (Fig. 23.28).

## Lateral root angulation or dilaceration

These changes appear as a mesial or distal bending confined to the root of the tooth (16, 35, 105, 106, 136, 139, 155, 161, 176, 191, 199, 204–207) (Fig. 23.30). They are seen in 1% of cases with injury to the primary dentition, usually avulsion. The injury usually occurs at age 2 to 7 years and usually affects the maxillary incisors. In contrast to vestibular angulation, most teeth with lateral root angulation or dilaceration erupt spontaneously (1). Similar malformations have been seen in developing teeth involved in jaw fractures (50, 62–65, 208–210) (Fig. 23.31).

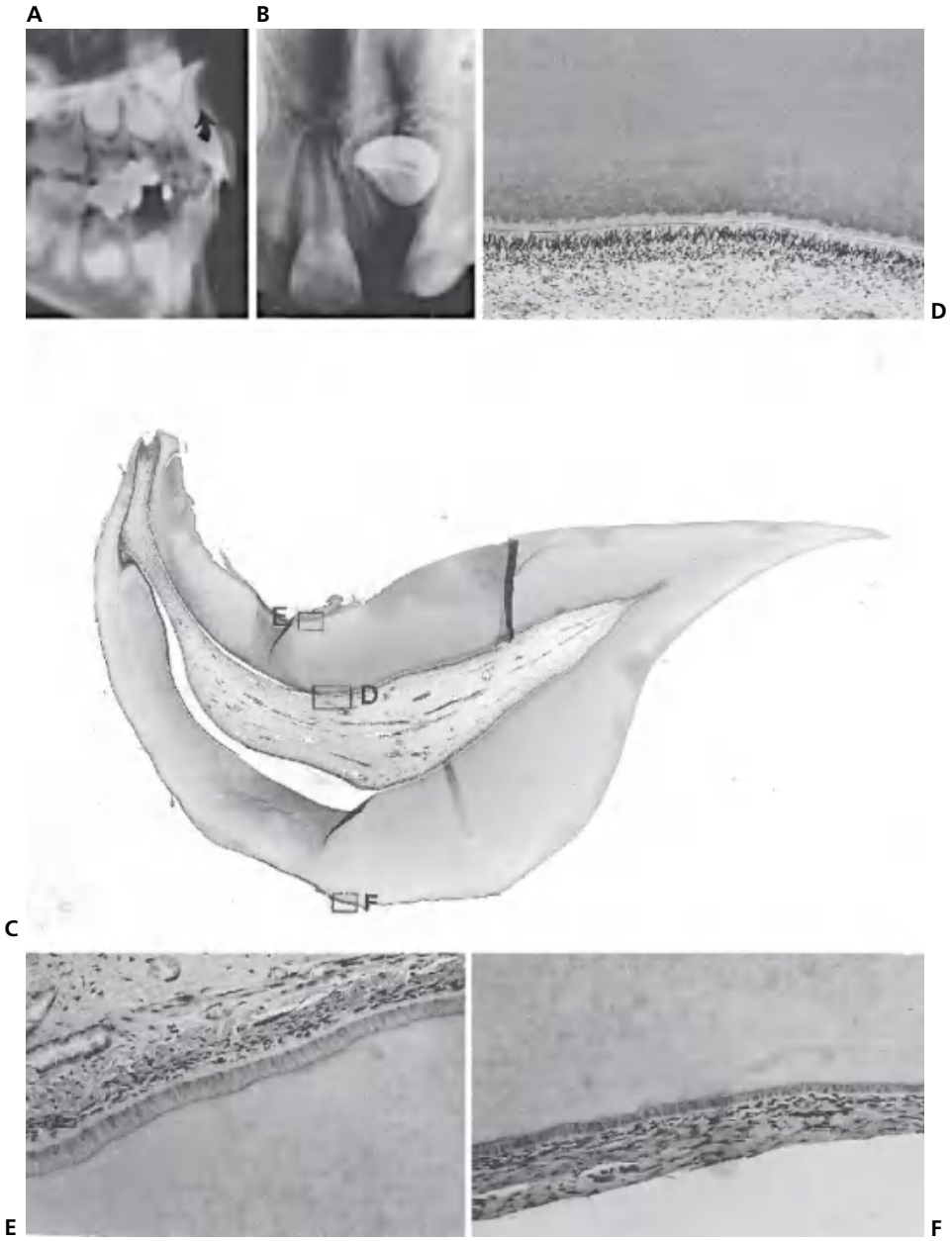



**Fig. 23.28** Vestibular root angulation of a central incisor secondary to extrusive luxation of a primary central incisor at the age of 4 years. A. Lateral radiograph showing facial orientation of the crown (arrow). B. Intraoral radiograph of malformed incisor. C. Low power view of sectioned incisor. ×9. D. The normally structured dentin indicates that no acute dental trauma has been inflicted on the tooth. ×30. E and F. Slight increase in thickness of the facial cementum compared to the lingual. ×75. Source: Reproduced from (1) with permission.

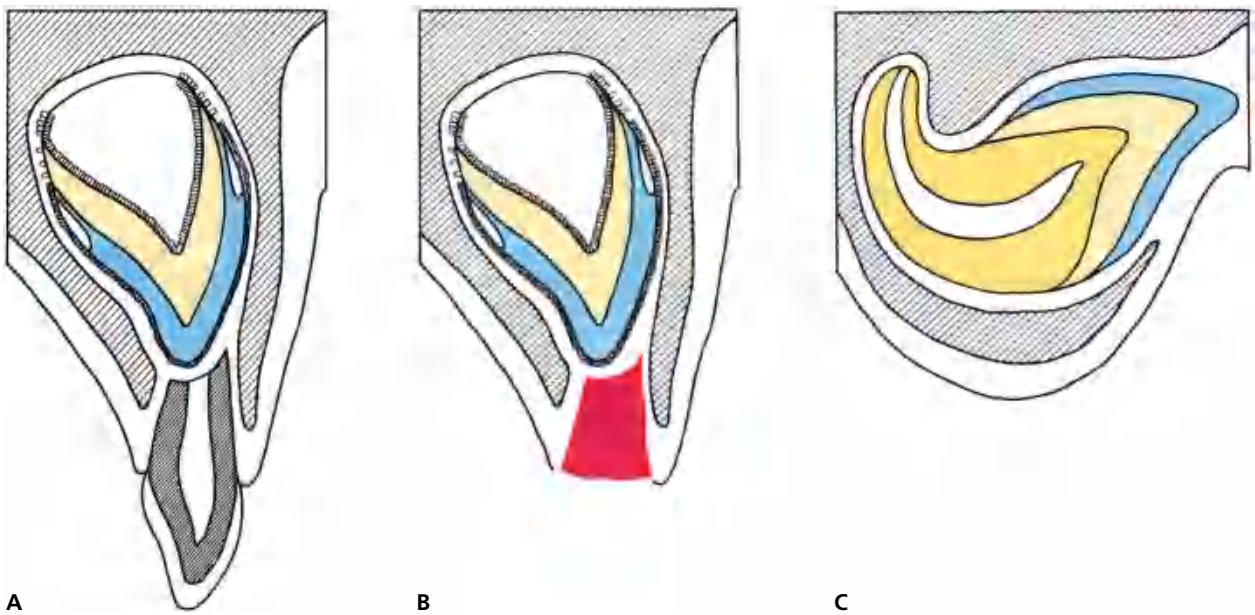
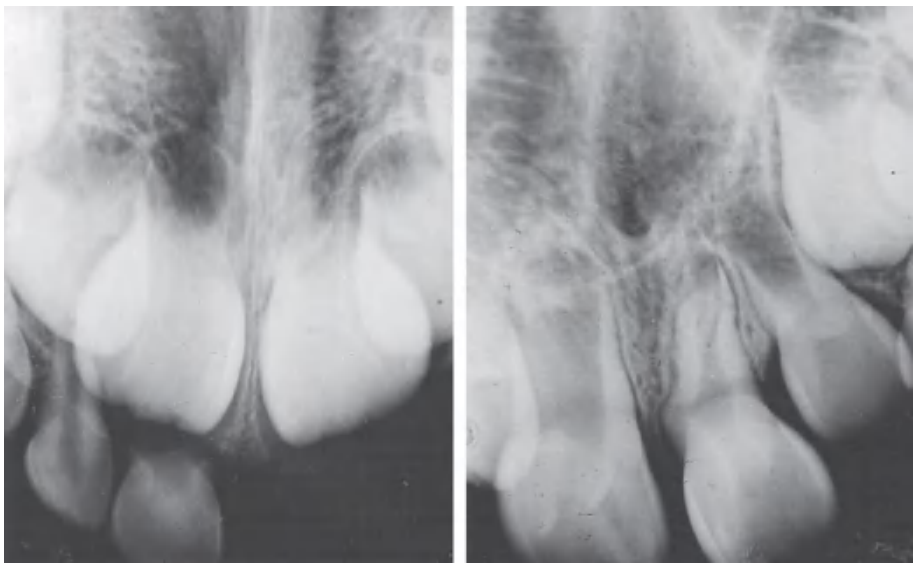
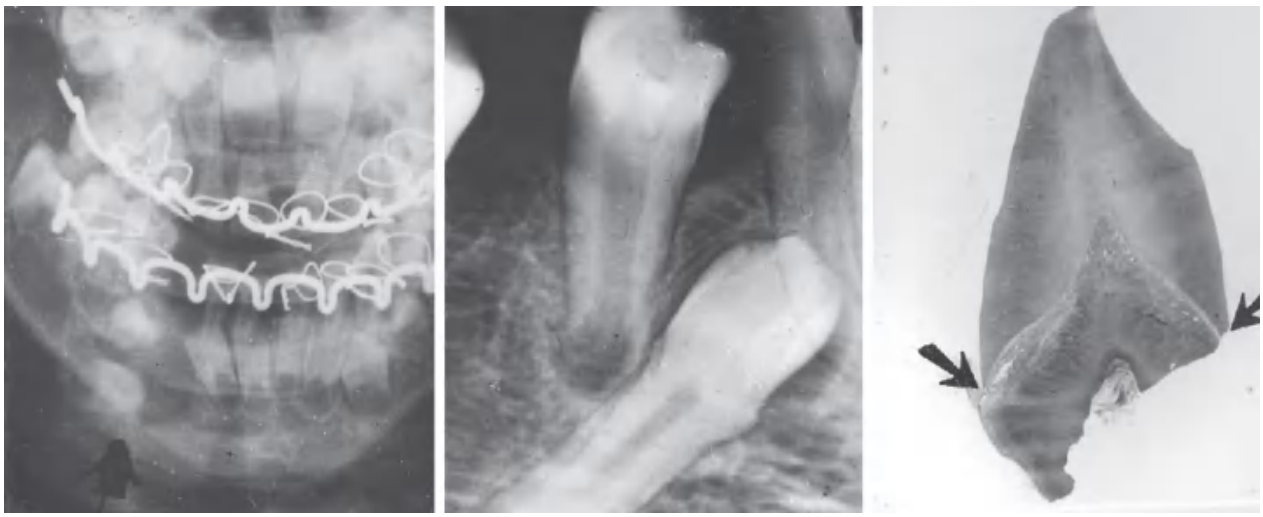



**Fig. 23.29** Schematic drawing illustrating possible mechanism of vestibular root angulation. A. Condition before injury. B. Primary incisor lost at an early age. C. Possibly due to formation of scar tissue along the path of eruption, the developing tooth changes its position facially.



A B

**Fig. 23.30** Lateral root angulation as a result of avulsion of left primary lateral and central incisors at the age of 5 years. A. Condition after injury. Note that root development of the central incisors has just started. B. Radiograph taken at the age of 7 years reveals angulation between the root and crown portion. Source: Reproduced from (1) with permission.



A B C

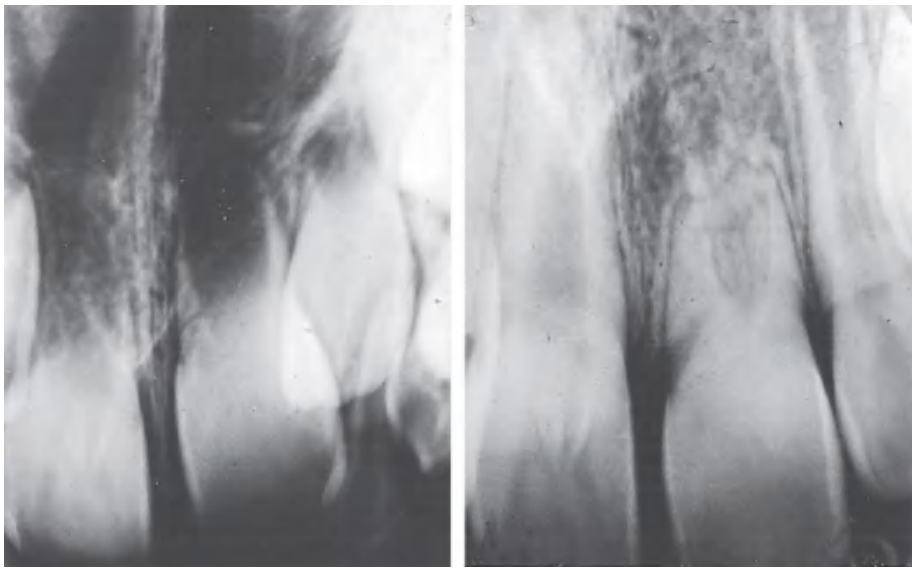
**Fig. 23.31** Slight root dilaceration and impaction of a right canine after jaw fracture sustained at the age of 5 years. A. Radiograph taken 5 days after the injury. Note displacement of the developing canine (arrow). B. Condition 9 years later. Note that the impacted and dislocated canine shows slight root dilaceration. C. Histologic examination of the coronal part of the canine. The dilaceration is clearly visible (arrows). Source: Reproduced from (63) with permission.

The pathogenesis of these lesions is not fully understood, but histologic studies have shown that displacement apparently occurs between the mineralized root portion and the developing soft tissues (63, 161) (Fig. 23.31).

## Partial or complete arrest of root formation and root angulation

This is a rare complication among injuries in the primary dentition, affecting 2% of involved permanent teeth (1, 2, 16, 17, 105, 130, 153, 155, 191, 206, 211–219) (Figs 23.32–23.37). The injury to the primary dentition usually occurs between 5 and 7 years of age and normally affects maxillary incisors. The injury sustained is usually avulsion of the primary incisors (1). A number of teeth with this type of root malformation remain impacted, while others erupt precociously and are often exfoliated due to inadequate periodontal support (220). Similar root abnormalities have been found in developing teeth involved in jaw fractures (50, 62, 63, 208, 221–224) or after replantation of an avulsed immature incisor with vital periodontal ligament (225).

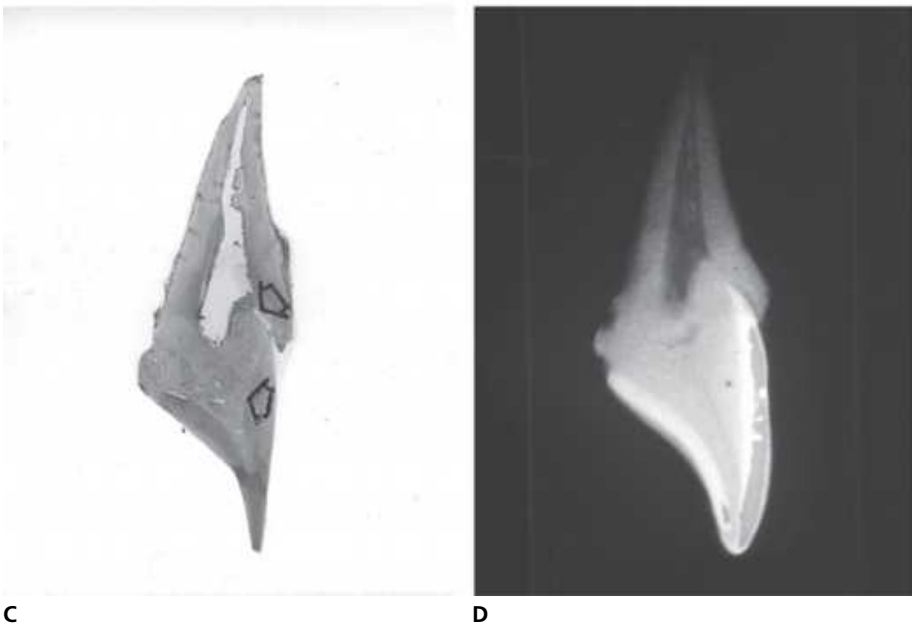
The histopathology of root malformation varies. Some cases show diminutive root development without evidence of a previous acute traumatic episode during hard tissue deposition (1, 216, 217) (Fig. 23.33). Scar tissue developing after premature loss of the primary predecessor has been thought to prevent normal eruption and also to interfere with root



A B

**Fig. 23.32** Complete arrest of root formation following avulsion of a left primary central incisor at the age of 5 years. A. Radiograph at time of injury. B. Condition at the age of 7 years. Note complete arrest of root development, invasion of bone into the root canal and development of an internal periodontal ligament. Source: Reproduced from (1) with permission.

A B



C D

**Fig. 23.33** Partial arrest of root formation of an upper left central incisor. The patient sustained an injury to the primary dentition at the age of 5 years. A. Radiographic examination at the age of 8 years shows a secondary pulp necrosis and a periapical radiolucency. Pulp necrosis was possibly induced by bacterial invasion through the poorly mineralized tissue formed immediately following injury. B. Mesial view of extracted incisor. C. Low power view of sectioned incisor. A slight dilaceration and a marked calcio-traumatic line with poorly mineralized tissue (arrows) indicate that an acute injury has occurred during odontogenesis. ×3. D. Microradiograph of the sectioned tooth. Source: Reproduced from (1) with permission.

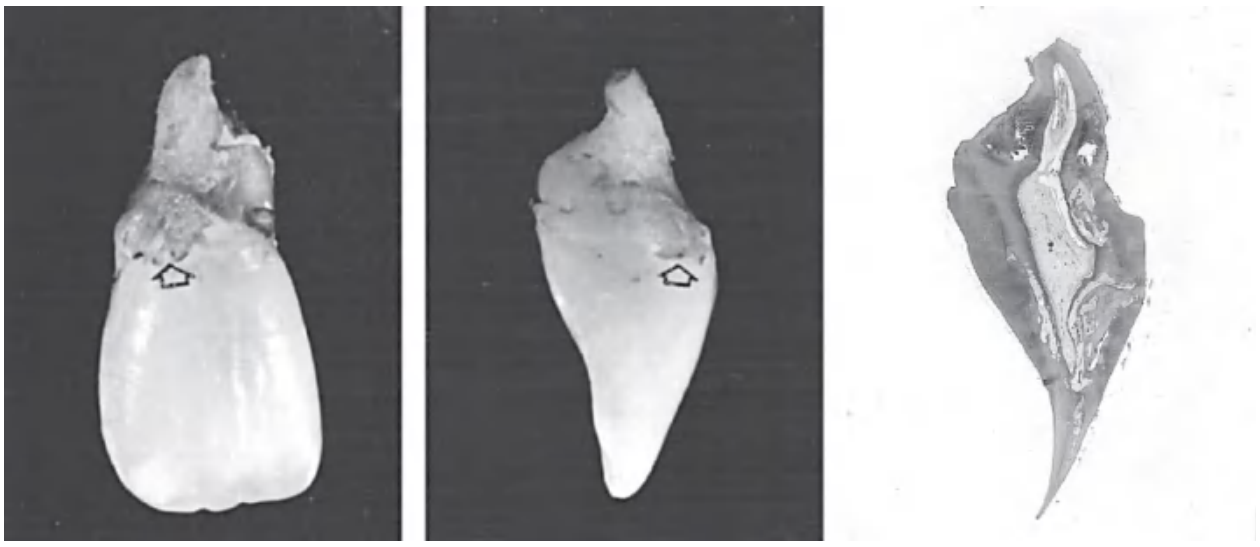



**Fig. 23.34** Partial arrest of root formation of a right central incisor. The patient sustained luxation injury of both primary central incisors at the age of 5 years. A and B. Frontal and lateral views of extracted incisor. A small resorption cavity is evident cervically (arrows) possibly inflicted by previous surgical exposure of the impacted tooth. C. Low-power view of sectioned incisor. Apart from peripheral root resorption there is no evidence of acute injury. ×5. Source: Reproduced from (1) with permission.

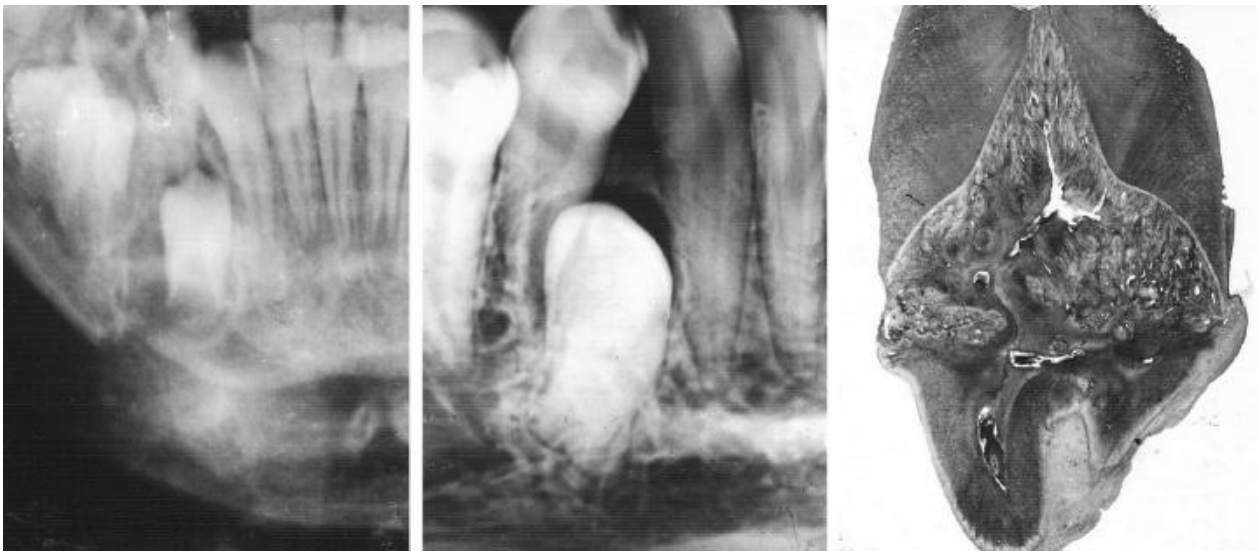


**Fig. 23.35** Partial arrest of root development after jaw fracture affecting the right side of the mandible in an 8-year-old patient. A. Condition immediately after injury. B. Six years later. The canine is impacted with a partial arrest of root development. C. Histologic examination of the removed canine. Note sharp demarcation between pre- and post-traumatic hard tissue formation. ×8. Source: Reproduced from (63) with permission.

formation (1). In other instances, a typical calciotraumatic line, separating hard tissue formed before and after injury, is seen (1, 63, 213, 226) (Figs 23.34 and 23.35). In these cases, the trauma has apparently directly injured Hertwig's epithelial root sheath, thus compromising normal root development (Fig. 23.36).

Radiographic examination reveals typical foreshortening of the root. Root resorption may also be seen with this type of root abnormality (63, 221, 224) (Fig. 23.37).

## Sequestration of permanent tooth germs – dentigerous cyst

This is exceedingly rare after injuries to the primary dentition (16, 155, 180, 197, 227) (Figs 23.38 and 23.39). Infection can complicate healing of jaw fractures. In these instances, swelling, suppuration and fistula formation are typical clinical features, sometimes leading to spontaneous sequestration of involved tooth germs (62, 209, 224, 228) (Fig. 23.39). Clinical

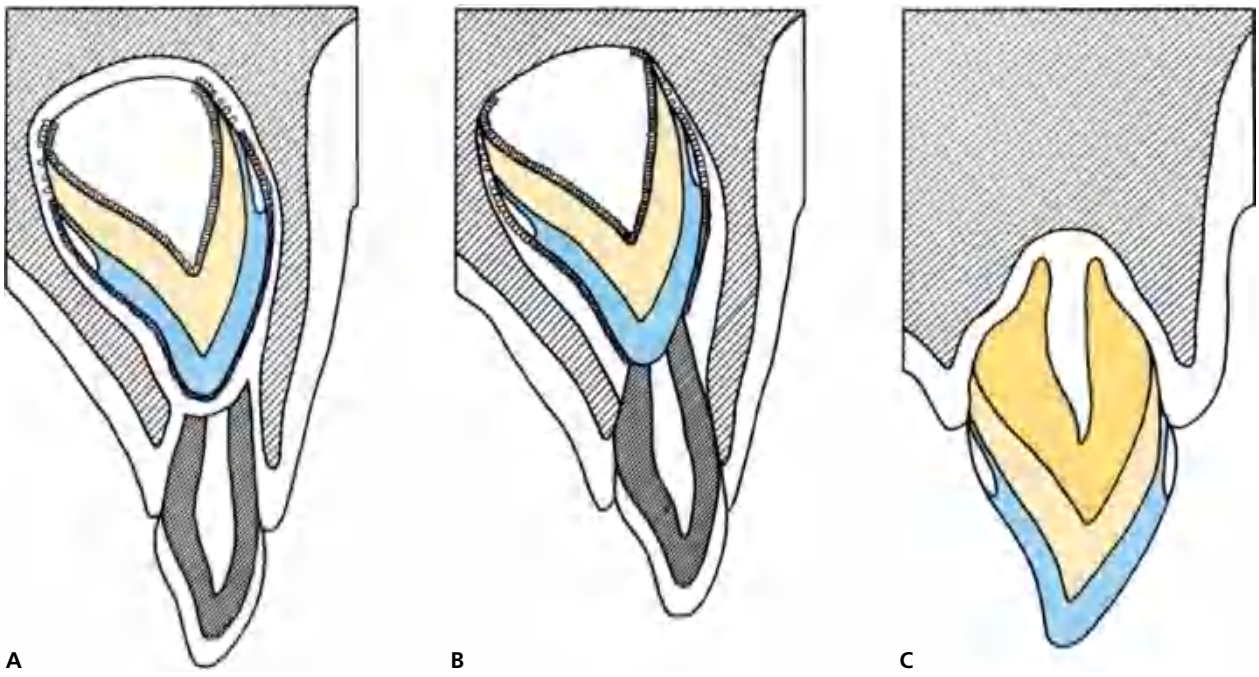



**Fig. 23.36** Schematic drawing illustrating mechanism of partial arrest of root formation. A. Condition before injury. B. The impact from the intruded primary incisor is transmitted to the permanent incisor with subsequent damage to the Hertwig's epithelial root sheath. C. Partial arrest of root formation.

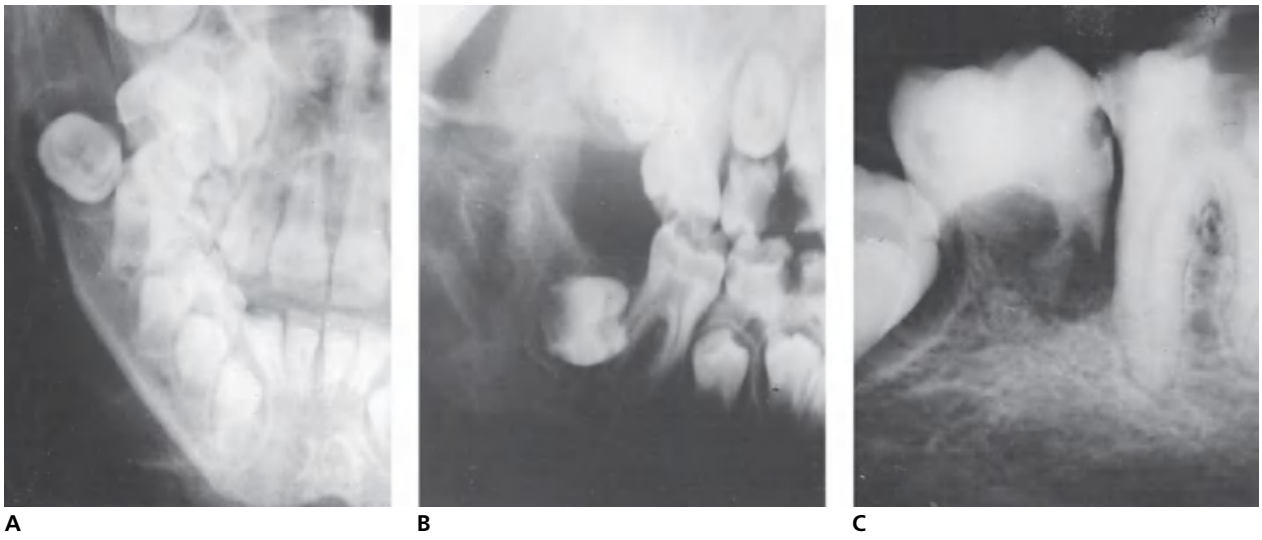



**Fig. 23.37** Complete arrest of root development and root resorption after jaw fracture in the second molar region of a 9-year-old child. A and B. Condition immediately after trauma. The developing second molar is displaced. C. Condition 7 years later. The second molar has erupted; however, there is a complete arrest of root development and root resorption. Source: Reproduced from (63) with permission.

cases have reported swelling of the maxilla, retained darkened primary incisors and absence of the permanent incisor. It has been hypothesized that chronic periapical inflammation of injured primary incisors may stimulate proliferation of the reduced enamel epithelium of the adjacent developing tooth germ, leading to a follicular cyst (229–231).

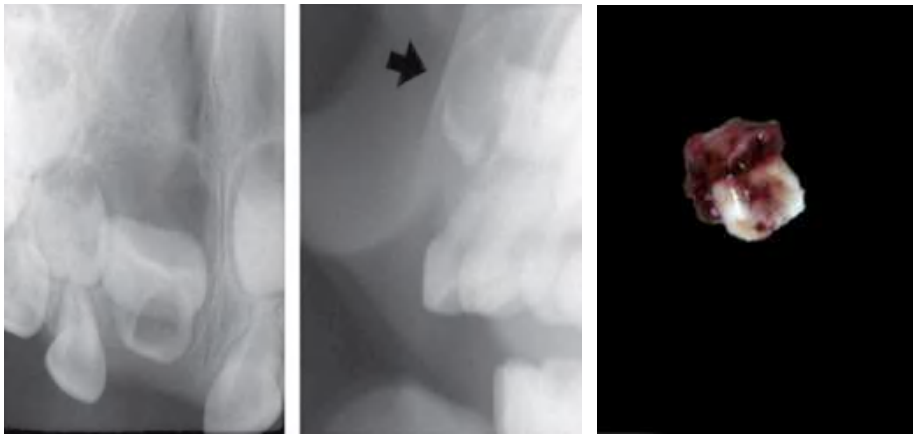
Radiographic examination discloses osteolytic changes around the tooth germ, including disappearance of the outline of the dental crypt and expanded cortical alveolar bone (Figs 23.38 and 23.39).

## Eruption disturbances

Disturbances in permanent tooth eruption may occur after trauma to the primary dentition and it is suggested that this is related to abnormal changes in the connective tissue overlying the tooth germ (232). The eruption of succeeding permanent incisors is generally delayed for about 1 year after premature loss of primary incisors (233, 234), whereas premature eruption of permanent successors is rare (153, 155). Early loss of primary incisors (avulsion or

**Fig. 23.38** Sequestration of a permanent central incisor tooth germ in a $1^1/_2$-year-old child. A. Radiographic examination at the time of injury reveals intrusion of the right primary central incisor. The primary incisor was removed 2 weeks later due to acute inflammation and a labial abscess. B. A chronic fistula appeared in the region 2 months later. Antibiotic therapy was instituted, and the fistula disappeared. However, upon withdrawal of antibiotics, the fistula returned. C. Radiographic condition after 6 months. Note the osteolytic changes around the permanent tooth germ. At this time, the tooth germ was removed. Courtesy of Dr. H. Echerbom, Luleå, Sweden.

**Fig. 23.39** Sequestration of a permanent cental incisor tooth germ in a 4-year-old child. The girl fell from her cradle at the age of 6 month and expirienced another fall at the age of 2 years. A. Radiographic examination at the time she sought treatment (4 years old) reveals osteolytic changes around the permanent tooth germ. B. The lateral radiograph discloses an expansion of the cortical alveolar bone and a large radiolucency area surrounding the tooth grem. Note the expanded bone causing upper lip deformity. C. The central incisor was removed under general anesthesia. The removed tooth was included in a dentigerous cyst.

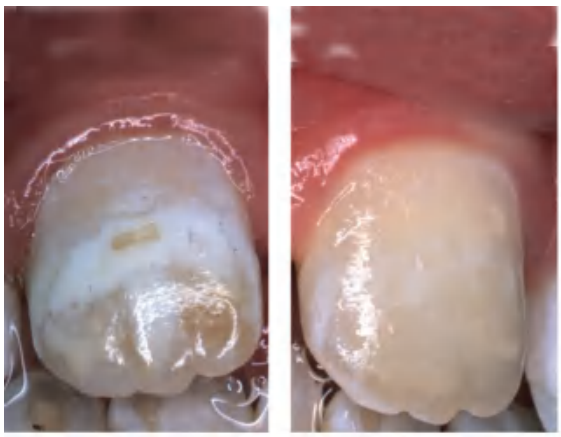



extraction) leads to space loss only in rare instances (13, 17, 235). However, ectopic eruption of permanent successors has been seen, possibly due to lack of eruption guidance otherwise offered by the primary dentition (13). These teeth often erupt labially (17). Impaction is very common among teeth with malformations confined to either the crown or root (1, 183, 206, 236). When the permanent tooth does erupt, it is often in facio- or linguoversion (202, 203, 233, 234, 237, 238). It has been reported a rare case of an ectopic eruption of a permanent tooth germ in the nasal cavity of a 6-year-old child, one year after maxillofacial trauma (239).

## Treatment

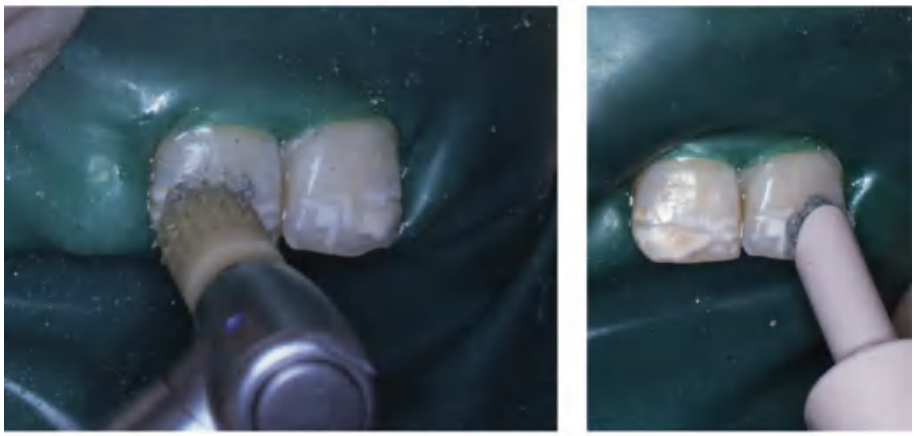
Minor white or yellow-brown discolorations of enamel seldom require treatment. However, if the enamel changes are esthetically disturbing, techniques for enamel microabrasion have been suggested (240–244) (Fig. 23.40). This implies application of an 18% hydrochloric acid-pumice paste to the isolated enamel surfaces for a series of, for example, 6–8 sequential 5-sec rubbing applications with 10-sec interim water rinsings. This treatment is followed by a 4-min application of 2% sodium fluoride gel and polishing with abrasives when necessary.

**Fig. 23.40 Microabrasion of a tooth with superficial enamel discoloration**
A central incisor with multicolored superficial enamel defects is shown on the left. Immediately after enamel microabrasion (right), the discoloration is eliminated. Courtesy of Dr. T. P. Croll, Doyletown, USA.



**Microabrasion of trauma-induced enamel discolorations**
The enamel changes consist of opaque white and yellow color changes.

**Enamel abrasion**
Both central incisors are isolated with a rubber dam and enamel abrasion carried out with hydrochloric acid pumice mixture, a mandrel tip and a hand applicator.



**Condition after enamel abrasion**
The enamel changes are markedly accentuated compared to the status before enamel abrasion.

**Fig. 23.41** Restoration of a right central incisor with yellow-brown discolored enamel with composite resin. A. Clinical condition. B. Cleansing the enamel with pumice followed by a bur. C. Etching the enamel. D and E. A celluloid crown form is used as a matrix for the composite resin. F. The finished restoration.

It has been found experimentally on extracted teeth with intact enamel surfaces, that such a procedure removes 12 µm enamel initially and 26 µm with successive applications (243), but that the amount of enamel which was lost was clinically undetectable (241). Unfortunately, most enamel discolorations due to trauma penetrate the entire enamel thickness (1) (Figs 23.10, 23.13 and 23.19). This fact explains why this technique does not work satisfactorily in trauma-related enamel discoloration and may sometimes even accentuate these discolorations (Fig. 23.40).

Treatment of white or yellow-brown discoloration of enamel with circular enamel hypoplasia implies removal of the discolored enamel with a bur, etching with an acid conditioner and restoration with a composite resin (43, 131, 132, 173, 245–250) (Fig. 23.41).

When discoloration and enamel defects occupy most of the labial surface, a porcelain jacket crown or laminate veneer can be indicated (251, 252).

Crown-dilacerated teeth often erupt spontaneously into normal position. In some cases, surgical exposure of the crown is necessary (Fig. 23.42). Because of the severity of the malposition, surgical exposure sometimes must be supplemented with orthodontic realignment (167, 175, 182, 203, 253, 254) (see Chapter 28).

When the tooth has erupted to the level such that the dilacerated area is free of the gingiva, restorative therapy

**Fig. 23.42 Surgical and restorative treatment of a crown-dilacerated incisor**

The patient sustained an intrusion injury of both right primary incisors at the age of 1 year. These teeth were later removed due to pulp necrosis. The radiographic examination shows crown dilaceration of the right central incisor and enamel hypoplasia of the right lateral incisor.

**Clinical and radiographic condition at the age of 7 years**

Eruption of the right central incisor has not taken place. Surgical exposure was therefore planned.

**Surgical exposure of the malformed incisors**

After administration of a local anesthesia both incisors were surgically exposed.

**Condition after exposure**

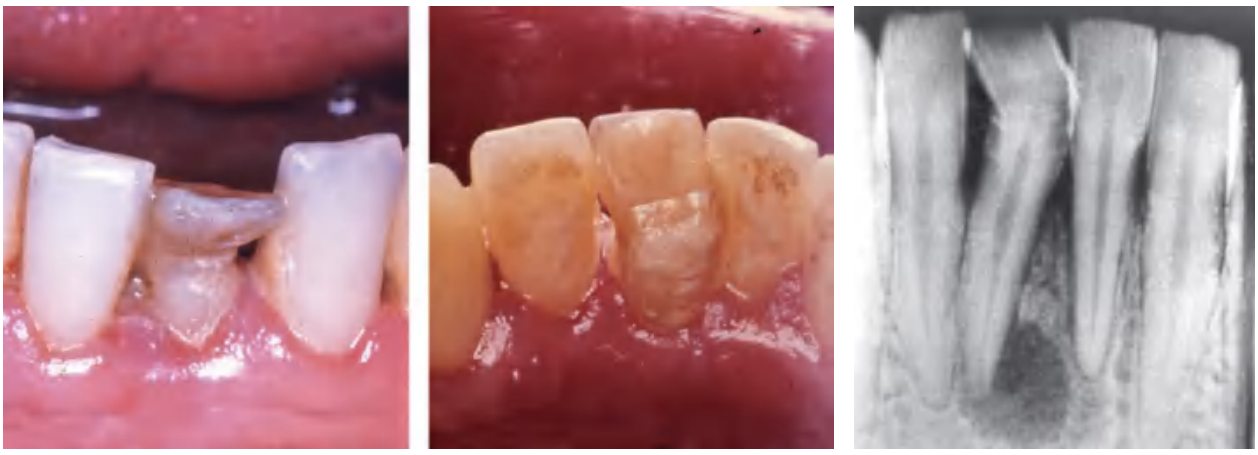
Two weeks after surgical exposure a new gingival collar has been formed.

**Condition 4 months later**
The incisors have further erupted and are now ready for restoration.

**Removing the dilacerated crown portion**
After a local anesthesia the dilacerated crown portion is removed and the exposed dentin is covered with a hard-setting calcium hydroxide cement.

**Temporary restoration**
The incisor is temporarily restored. Due to problems with moisture control the tooth was initially restored with a steel crown and later with a temporary crown and bridge material.

**Restoration with composite resin**
As an interim restoration both incisors were restored with composite resin.



A B C

**Fig. 23.43** Pulp necrosis and periapical inflammation following crown dilaceration of a central incisor. A and B. Frontal and lingual view of involved incisor. C. Radiographic demonstration of pulp necrosis and periapical inflammation.

should be instituted, as the central lumen of the 'internal root' constitutes a pathway for bacteria into the pulp. Thus, a number of these teeth have been found to develop pulp necrosis and periapical inflammation after eruption without any evidence of decay (1, 115, 158, 160) (Fig. 23.43). It is therefore important to remove the dilacerated part of the crown as soon as possible. A temporary crown can then be placed until eruption is complete, when an acid-etch/composite resin restoration, a veneer or a cast restoration can be made (Fig. 23.42).

The use of CBCT may be justifiable in the assessment of selected cases of developmental disturbances to permanent teeth, when conventional radiographs provide inadequate information for treatment planning (87, 207).

Malformed impacted teeth, e.g., odontoma-like malformations, teeth with root duplication, root dilaceration or angulation should generally be removed. However, future treatment needs (i.e. dental implants) might indicate their preservation in order to maintain the alveolar ridge at an acceptable height. Successful conservative treatments have been reported in growing patients (131, 190, 202, 203, 255). A possible exception is vestibular root angulation. Provided there is adequate space, such teeth can be realigned by surgical exposure followed by orthodontic intervention (105, 115, 191, 196, 256) (see Chapter 28). An alternative therapy can be extraction of the malformed tooth, resection of the angulated root portion and insertion of an apical implant (257).

Although rare, chronic periapical inflammation of injured primary teeth may lead to dentigerous cysts and spontaneous sequestration of the tooth germ. If not, they should be removed surgically (117). A successful conservative treatment of a dentigerous cyst has been reported, where extraction of the lower primary incisors, followed by removal of the granulation tissue, was carried out. The lower permanent incisors erupted in normal position, eighteen months later (258).

In some cases, premature loss of primary teeth can lead to disturbance in eruption. Apparently the tooth germ is not able to penetrate the mucosa covering the alveolar process. In these cases, excision of the tissue overlying the incisal edge will result in rapid eruption of the impacted tooth (232, 259) (Fig. 23.44). If the teeth are impacted with their crowns inclined facially and above the mucogingival junction, it is important to know that a wide incision of non-functional mucosa can give rise to retraction of the gingiva. Furthermore, the gingiva is non-keratinized and prone to periodontal disease (232) (Fig. 23.45). While this finding has been questioned in a case report (259), it is recommended that a smaller incision be made, preferably only in the functional gingiva. When the permanent tooth erupts it is often in facio- or linguoversion (Figs 23.45 and 23.46).

## Essentials

### Terminology

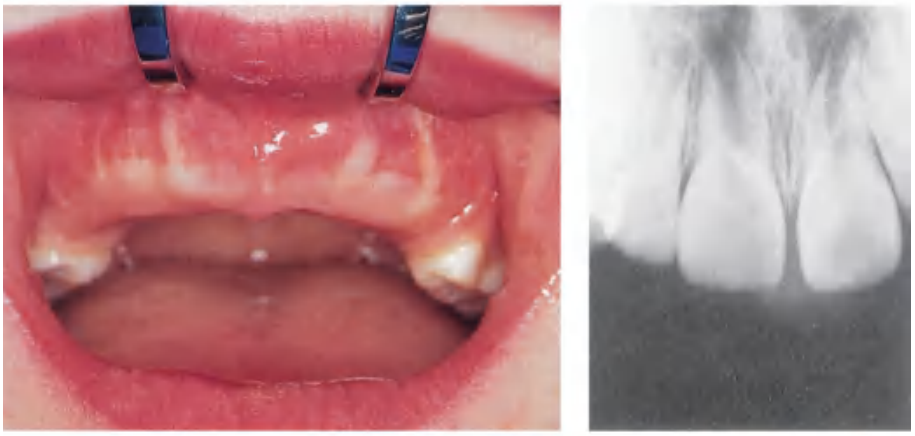
- White or yellow-brown discoloration of enamel
- White or yellow-brown discoloration of enamel and circular enamel hypoplasia
- Crown dilaceration
- Odontoma-like malformation
- Root duplication
- Vestibular root angulation
- Lateral root angulation or dilaceration
- Partial or complete arrest of root formation
- Sequestration of permanent tooth germ
- Disturbance in eruption

### Frequency

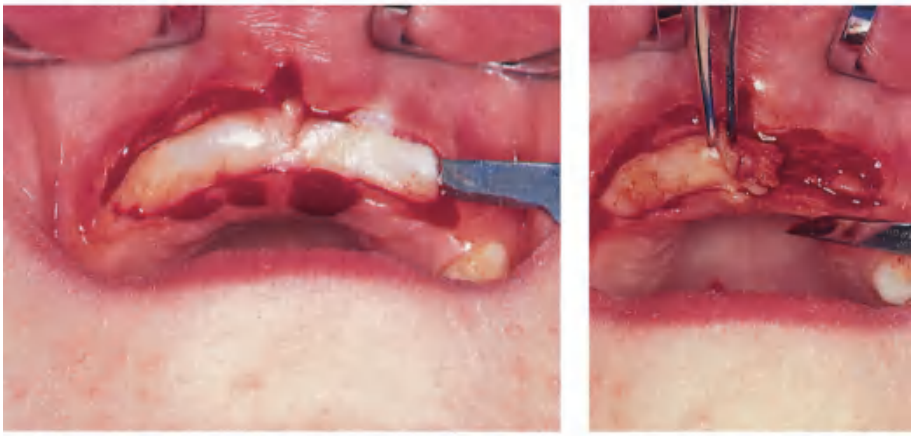
- Injuries to primary teeth: 12–69% of involved permanent teeth
- Jaw fractures: 19–68% of involved permanent teeth

**Fig. 23.44 Surgical exposure of impacted permanent incisors**
This 1-year-old girl suffered avulsion of all primary incisors due to a fall.

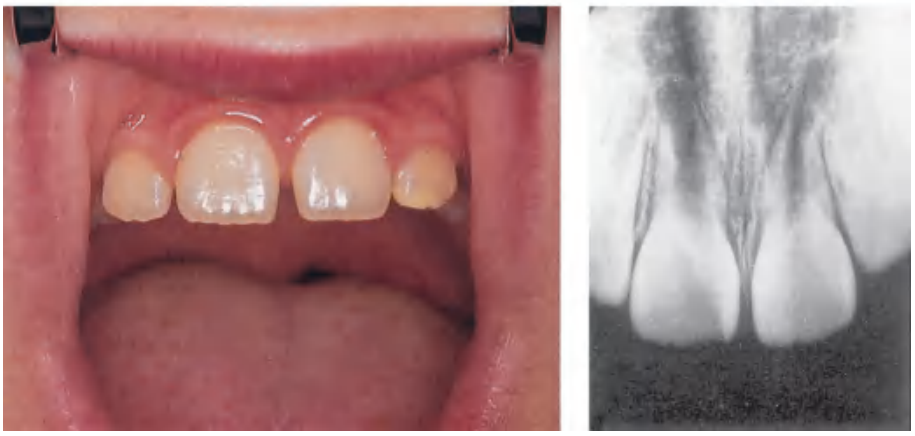
**Condition at the age of 10**
There is no sign of eruption in spite of advanced root development. The incisal edges of the impacted incisors can be palpated.



**Exposing the incisors**
After administration of a local anesthesia the mucosa overlaying the incisal edges is removed with a surgical blade.



**Condition 6 months after exposure**
The incisors are now fully erupted.



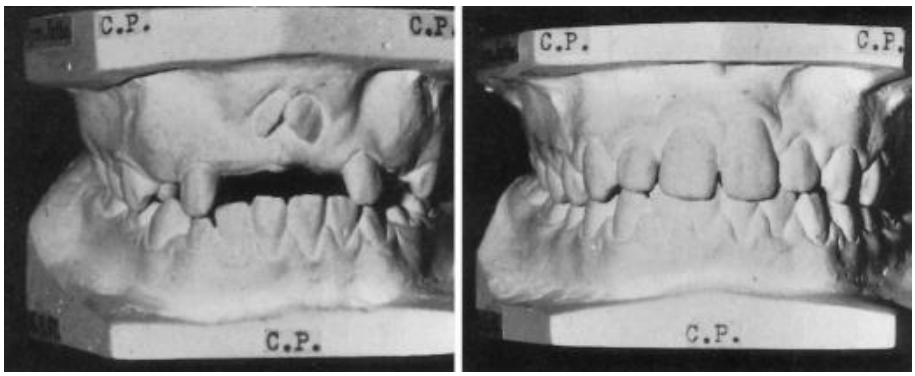



**Fig. 23.45** Gingival retraction and enlarged gingival cuffs in a patient where both central incisors were exposed above the muco-gingival junction. From DIBIASE (232) 1971.

**Fig. 23.46** Permanent lateral incisor erupted in linguoversion.

## Pathology

- Disturbances in mineralization and morphology

## Treatment

### *Yellow-brown discoloration of enamel with or without enamel hypoplasia*

- Enamel microabrasion (Fig. 23.40)
- Composite resin restoration (Fig. 23.41)
- Porcelain laminate
- Porcelain jacket crown

### *Crown dilaceration (Fig. 23.42)*

- Surgical exposure and possibly orthodontic realignment
- Removal of dilacerated part of the crown
- Temporary crown until root formation is completed
- Semi- or permanent restoration

### *Vestibular root angulation*

- Combined surgical and orthodontic realignment

### *Other malformations*

- Extraction is usually the treatment of choice. Whenever possible, conservative treatment in growing patients is the best option.

### *Eruption disturbances (Fig. 23.44)*

- Surgical exposure

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# 24
# Soft Tissue Injuries

L. Andersson & J. O. Andreasen

## Terminology, frequency and etiology

A large number of dental traumas are associated with injuries to the lip, gingiva and oral mucosa. One third of all patients treated for oral injuries in dental emergency settings and more than half of all patients treated in a hospital emergency setting are associated with soft tissue injury (1–4).

The dentition is shielded by the lips covering the teeth in case of impact to the lips (Fig. 24.1). Trauma energy will be absorbed in the soft tissue resulting in less severe tooth injuries. However, this will result in various types of soft tissue trauma such as contusion, abrasion, laceration and tissue avulsion depending on the direction of force, shape and size of object, and energy.

Moreover, when a patient is subjected to trauma, the teeth may also cause injury to the surrounding soft tissue, most commonly penetrating into the lips but sometimes also the cheeks and tongue. When teeth are dislocated the gingiva will sometimes be lacerated.

Incorrect treatment in the emergency situation, such as failure to remove foreign bodies, may result in unesthetic scarring (Fig. 24.2).

## Types of soft tissue trauma

Soft tissue injuries are usually classified into the following four groups and their characteristics are described below. They can be seen extraorally (skin) as well as intraorally (gingiva and oral mucosa).

### Abrasion

An abrasion is a superficial wound produced by rubbing and scraping of the skin or mucosa leaving a raw, bleeding surface (Fig. 24.3). This injury is usually seen on knees and elbows in children and in the oral region the lips, chin, cheek or tip of the nose are frequently affected. The friction between the object and the surface of the soft tissue removes the epithelial layer and papillary layer of the dermis, and the reticular layer of the dermis is exposed. Superficial abrasions can be quite painful because terminal nerve endings are exposed.

### Contusion

A contusion is a bruise usually produced by impact with a blunt object and not accompanied by a break in the skin or mucosa but usually causing subcutaneous or submucosal hemorrhage in the tissue (Figs 24.4 and 24.5). Contusions may also be caused by the disrupting effect of fractured bone in maxillofacial injuries. Contusions may therefore indicate an underlying bone fracture.

### Laceration

A laceration is a shallow or deep wound in the skin or mucosa resulting from a tear and is usually produced by a sharp object or by teeth penetrating into the soft tissue (Figs 24.6 and 24.7). Laceration involves epithelial and subepithelial tissues and if deeper may disrupt blood vessels, nerves, muscles and involve salivary glands. The most frequent lacerations in the oral region caused by trauma are seen in lips, oral mucosa and gingiva. More seldom the tongue is involved.

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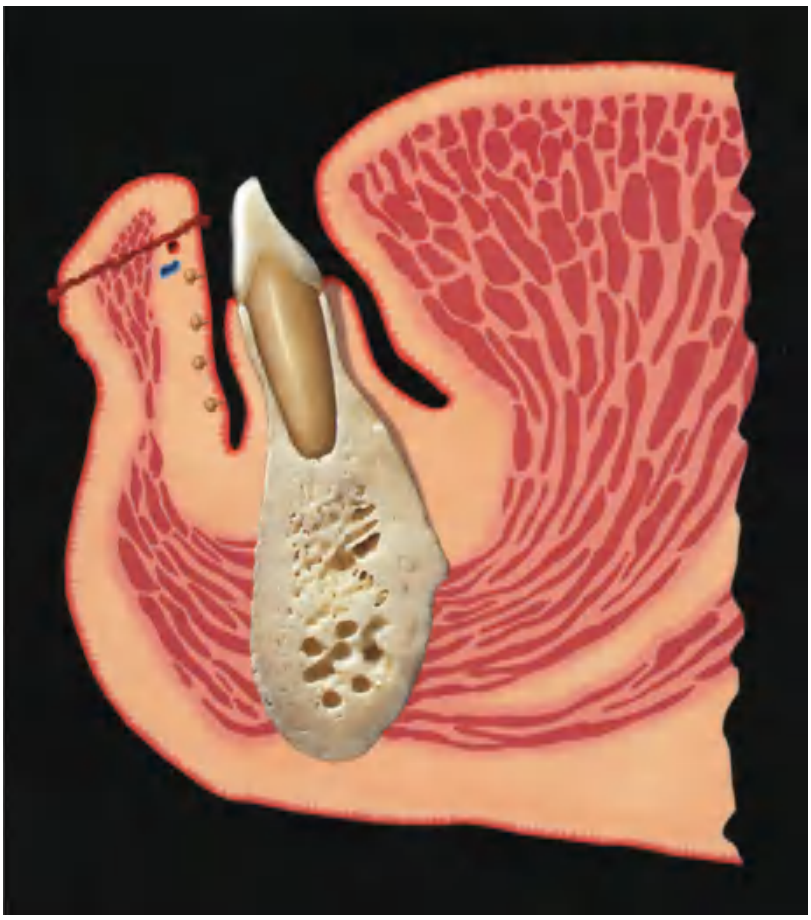


**Fig. 24.1** Section through a human lower lip in the midline. Note the composite structure of the lip, with musculature, salivary glands and hair follicles. Most of the musculature is oriented parallel to the vermilion border.

### Tissue avulsion

Avulsion (tissue loss) injuries are rare but seen with bite injuries or deep abrasions (Fig. 24.8). These are complex injuries from a treatment point of view in the emergency phase because a decision has to be made whether to excise and primary close the defect with flaps or grafts (large defects) or wait for spontaneous healing (small defects).

## Emergency management

After the diagnosis has been established, the extent and contamination of the lesion is examined (95, 96). Ascertainment of the extent of tissue damage demands thorough exploration after administration of local anesthesia. Soft tissue injuries are often seen together with dental injuries and bone fractures. A systematic approach is recommended. If the soft tissue injuries are sutured first, before intraoral treatment of teeth and bone fractures, the sutures will most likely make the tissue rupture when later intraoral manipulation is taking place. This will also result in the tissue margins being more difficult to close in a second suturing. For this reason, it is important to plan the emergency treatment so that intraoral treatment is performed first, and extraoral suturing of lips after the intraoral treatment. This is in contrast with the examination procedure where we start with extraoral examination before we perform intraoral examination. A golden rule: '*Examine from outside towards inside – treat from inside towards outside*' may help to remember the systematic examination and treatment sequences.

Local anesthetics should be used to allow manipulation of the tissue without pain. Topical anesthetics are preferable, and recent reports have indicated that those containing a combination of prilocaine/lidocaine are effective in reducing pain from needle stick so that it may even be possible to close minor lacerations without needle stick injection but only topical anesthesia (70–76). However, topical anesthetics used directly in lacerations should be used with caution in small children and infants because of the risk of methemoglobinemia (75, 76).

There are four major steps in the emergency management of soft tissue injuries: *cleansing*, *debridement*, *hemostasis* and *closure* (5). One of the aims of wound cleansing is to remove or neutralize microorganisms, which contaminate the wound surface, in order to prevent infection where time after injury plays an important role (77). Wound detergents reduce the bioburden (6). However, the fact remains that almost all common wound disinfectants have been shown to have a detrimental effect upon wound healing (7). Physiologic saline or Ringers solution appear to be without a harmful effect upon cells in the wound. Even water can be used for rinsing. The volume of fluid seems to be more important than if saline or water is used and copious irrigation is recommended as soon as possible after the injury (8, 78).

**Fig. 24.2 Scarring subsequent to a penetrating wound in the lower lip**
A 3-cm wide laceration of the cutaneous and mucosal aspects of the lower lip in a 9-year-old girl. Suturing of the cutaneous aspect of the wound was the only treatment provided.

**Condition after 1 week**
Note saliva seepage and non-closure of the wound.

**Follow-up**
Healing has resulted in extreme scarring of the skin, whereas the wound on the mucosa is hardly noticeable.

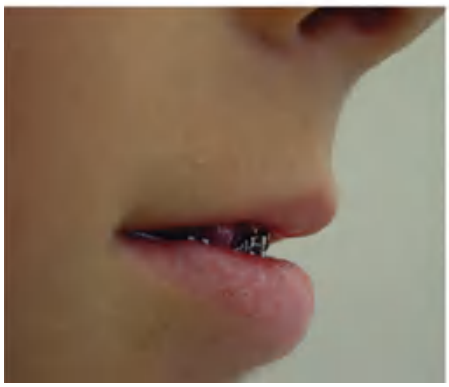
**Fig. 24.3** Abrasion of the upper and lower lip.

**Fig. 24.4** Periorbital hematoma indicating contusion injury after road traffic accident. Clinical and radiographical examination revealed underlying bone fracture.

**Fig. 24.5** Hematoma of soft tissue in right premolar region. Perimandibular hematoma is strongly indicative of mandibular fracture and radiographic examination revealed a mandibular fracture.

**Fig. 24.6** (Left) Laceration of lip after road traffic accident.

**Fig. 24.7** (Right) Intraoral laceration of the upper lip. The blood around the anterior teeth originated from this laceration and not from any dental injuries.



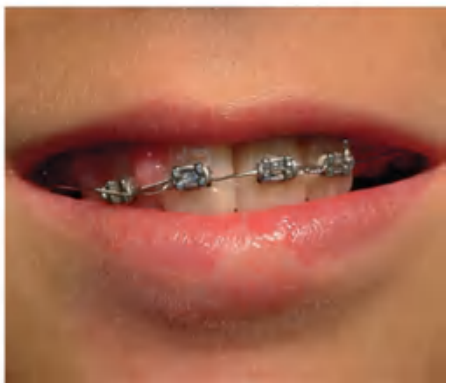


**Fig. 24.8** Avulsion injury of lower lip. Note the loss of soft tissue. Nevertheless, this was left for spontaneous healing without flaps or grafts. Note the final result one year after trauma. A normal contour of the lip is seen. Scar tissue is seen centrally in a circular area at the vermilion border with a 9mm diameter.

The presence of foreign bodies in the wound significantly increases the risk of infection (9, 97, 98) and retards healing, even in wounds free of infection (10) (see Chapter 1). Foreign bodies also contribute to extensive scarring and tattooing of tissues. This finding emphasizes the importance of adequate removal of all foreign particles prior to suturing.

## Abrasion

It is important to remove all dirt, gravel, asphalt and other foreign bodies to avoid future permanent tattoo and scarring in the skin (Fig. 24.9). This is very time consuming but extremely important (Fig. 24.10). There is only one chance to clean the abrasion properly and that is during the emergency phase. After administration of local anesthesia, the wound and surroundings are thoroughly rinsed and washed with saline. A scrub brush, gauze swabs or even a soft toothbrush may be used. If the contamination is severe a mild soapy solution may be used (Fig. 24.9). Thereafter, all foreign bodies are removed with a small excavator or a surgical blade which is placed perpendicular to the cutaneous surface in order to prevent it from cutting into the tissue. Finally, irrigation with saline should be performed. The wound is usually left open without any applications but may be covered with a bandage. The patient should avoid excessive sunlight during the first 6 months to decrease risk of permanent hyperpigmentation. Intraoral abrasions do not have to be treated but limited to removal of any foreign bodies.

**Fig. 24.9 Cleansing of a skin wound containing asphalt particles**

A, B. In order to adequately cleanse the abrasions, a topical anesthetic is necessary. In this case, a lidocain spray was used. Note that the nostrils are held closed to reduce discomfort from the spray entering the nose. C. Washing the wounds. The lips are washed with surgical sponges or gauze swabs soaked in a wound detergent.

A

B

C

**Removing asphalt particles**

D and E. The impacted foreign bodies cannot be adequately removed by scrubbing or washing; but should be removed with a small excavator or a surgical blade held perpendicular to the direction of the abrasions. Petroleum jelly (Vaseline) can be used to dissolve asphalt

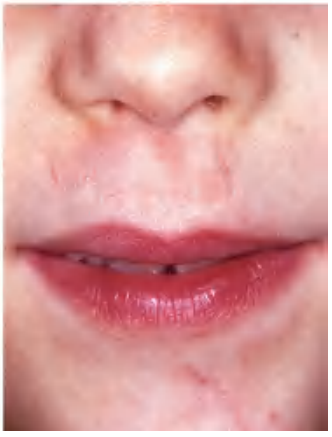
D

E

**Cleaned wound and follow-up**

F and G. Two weeks after injury, the soft tissue wounds have healed without scarring.

F



G

**Fig. 24.10** Extensive asphalt tattoo. The patient suffered an injury, years earlier. Inadequate wound debridement resulted in extensive asphalt tattooing. Courtesy of Dr. S. BOLUND, University Hospital, Copenhagen.

## Contusion

Swelling and bruising may indicate a deeper injury; usually only bleeding but sometimes bone fractures are the causes of contusions (Figs 24.4 and 24.5). Radiographic examination is therefore indicated. Care should be taken to extend the clinical examination also to indirect trauma related areas such as palpation and radiographic examination of the condyles when a bruise is seen in the chin region. Make sure that there is no ongoing bleeding if the swelling is located in a sensitive area for the airways such as the floor of the mouth or the tongue. No treatment is necessary for contusions when the injury is limited to soft tissue injury.

## Laceration

The possibility of contamination of the wound requires inspection for foreign bodies or tooth fragments. In deeper wounds, clinical inspection should be supplemented with a radiographic examination which can reveal at least some of the contaminating foreign bodies.

The removal of foreign bodies in facial and oral tissues after laceration is known to be a difficult and time-consuming procedure (11). A syringe with saline under high pressure, a scrub brush or gauze swabs soaked in saline can be used to remove foreign bodies. If this is not effective, a surgical scalpel blade or a small spoon excavator may be used. Complete removal of all foreign bodies is important, to prevent infection and also to prevent disfiguring scarring or tattooing in the skin.

Devitalized soft tissue serves to enhance infection by at least three mechanisms: first, as a culture medium that can promote bacterial growth; second, by inhibiting leukocyte migration, phagocytosis and subsequent bacterial kill; and third, by limiting leukocyte function due to the anaerobic environment within devitalized tissue (i.e. low oxygen tension impairs killing of bacteria) (9, 12, 13) (see Chapter 1). However, the maxillofacial region has a rich blood supply. For this reason, debridement should be kept to a minimum. However, severely contused and ischemic tissue should be removed in order to facilitate healing (14, 15).

## Gingival and vestibular lacerations

After administration of a local anesthetic, the wound is cleansed with saline, and foreign bodies removed. The lacerated gingiva is brought back into normal position, implying that displaced teeth have been repositioned (Figs 24.11 and 24.12). After repositioning of the gingiva, the necessary numbers of thin sutures are placed to prevent displacement of tissue. A minimum number of sutures should be used. The patient is then placed on an oral hygiene regimen using 0.1% chlorhexidine for 4–5 days, where after the sutures are removed. In cases of loss of gingival tissue, a gingivoplasty should be performed whereby flaps are elongated by placement of periosteal incision (Fig. 24.13). If tissue loss has occurred in the region of erupting teeth, it is important to consider whether tissue loss has exposed the cemento-enamel junction. If this is not the case, further eruption and physiologic gingival retraction will normalize the clinical appearance with time (Fig. 24.14). With minor displacements, gingival regeneration amounting to approximately 1 mm will usually occur (68) (Fig. 24.15).

A trauma force parallel to the front of the maxilla or the mandible will result in complete displacement of the labial mucosa into the sulcus area (67) (Fig. 24.16). Vestibular laceration with denuding of soft tissue from the bone is sometimes accompanied by severe contamination (Fig. 24.17). Wound cleansing and removal of all foreign bodies is very important before closure with sutures. Wound closure and primary healing is the main objective in these injuries and should aim at covering of bone. Later wound dehiscence with exposure of sequestered bone is sometimes seen and secondary healing with the loss of a thin superficial layer of the exposed bone will take place over a long time (Fig. 24.18). In that case daily rinsing is necessary to keep the area clean and support healing. The scarring from this secondary healing can be accepted because it is intraorally located and does not have any esthetic consequences for the patient.

## Lip lacerations

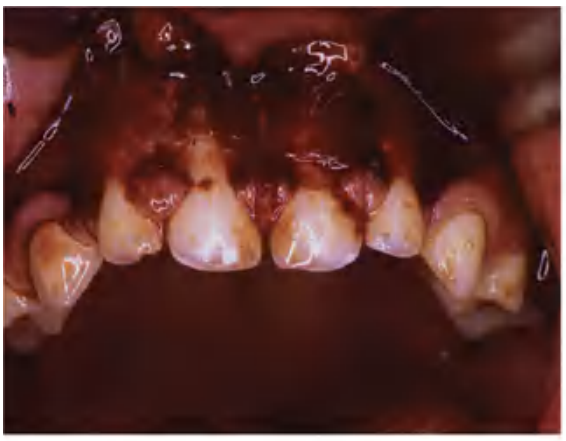
In cases of a frontal impact, the labial surfaces of protruding incisors may act as a bayonet, resulting in a sagittal split of the lip (Fig. 24.19). Because of the circumferential orientation of the orbicularis oris muscles, these wounds will usually gape, with initially intense arterial bleeding due to the rich vasculature in the region. Hemorrhage is usually soon spontaneously arrested due to vasoconstriction and coagulation but with manipulation of tissue bleeding may start again. Sometimes electrocoagulation may be necessary, but it should be borne in mind that extensive use of electrocoagulation in the lip should be avoided.

If the direction of impact is more vertical, parallel to the axis of either the maxillary or mandibular incisors, the incisal edges may penetrate the entire thickness of the lip. When the incisal edges hit the impacting object, fracture of the crown usually occurs.

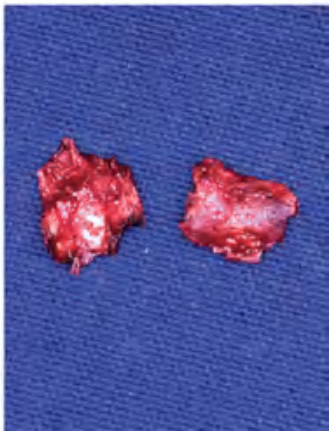
**Fig. 24.11 Treatment of horizontal gingival laceration with tissue displacement in the permanent dentition**
Due to an impact the gingiva has been lacerated and displaced whereas the central incisors are left intact.

**The displaced attached gingiva is repositioned and sutured**
Postoperatively a scan is seen mid-root in the left incisor region

**Fig. 24.12 Treatment of a gingival laceration with exposure of bone in the primary dentition**
Due to a fall against an object, the gingiva has been displaced into the labial sulcus and bone is exposed whereas the incisors are left intact.



**Repositioning and suturing of the gingiva**
The condition immediately after gingival repositioning and suturing. Loose labial bone fragments have been removed.



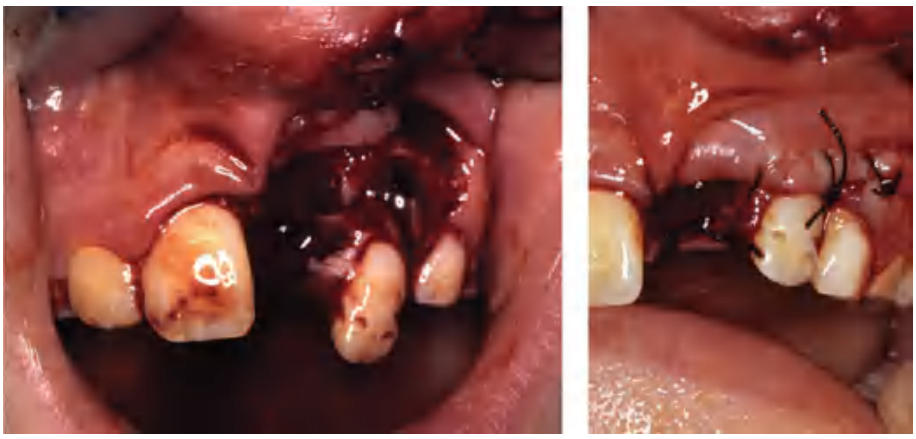


**Fig. 24.13** Gingival tissue loss. The central incisor has been avulsed and lost. There is tissue loss with exposure of labial bone. A flap is raised, and the periosteum incised, whereby it is possible to cover the denuded bone.

**Fig. 24.14** Gingival loss in an erupting permanent central incisor. In erupting teeth, gingival displacement or tissue loss should be assessed in relation to the position of the cemento-enamel junction. In this case of a 12-year-old boy, trauma resulted in tissue loss not extending beyond the cemento-enamel junction of the left central incisor. At a follow-up examination 5 years later, there is almost complete gingival symmetry.

**Fig. 24.15** Gingival laceration with spontaneous regeneration. The marginal gingiva in this 9-year-old girl was displaced apically and not repositioned. At the 1-month follow-up examination, normal gingival relations could be observed.

To find foreign bodies a thorough exploration of the laceration is important. Radiographic examination adds to the information and may show a variety of typical foreign bodies, such as tooth fragments, calculus, gravel, glass and fragments of paint (Fig. 24.20). However, other typical foreign bodies such as cloth and wood cannot be seen (69).

The radiographic technique consists of placing a dental film between the lip and the alveolar process. In cases of a wide lesion, orientation of images is facilitated by placing a small metal indicator (e.g. a piece of lead foil) in the midline of the vermilion border in order to locate possible foreign bodies. The exposure should be made at a low kilo voltage (to increase contrast), and the exposure time should be kept at the lowest value to be able to reveal particles with low radiographic contrast. If the intraoral film discloses foreign bodies, a lateral exposure may verify their position in a sagittal plane (Fig. 24.21).

In the case of narrow penetrating wounds, a special technique of opening up the wound, as illustrated in Fig. 24.22, is recommended. Management of a broad penetrating lip lesion is seen in Fig. 24.23. Treatment starts by cleansing the wound and surrounding tissue. The wound edges are elevated, and foreign bodies are found and retrieved. It is essential to consider that foreign bodies are usually contained within a small sac within the wound. When all fragments that have been registered on radiographs have been retrieved,

**Fig. 24.16** Vestibular laceration in the mandibular front region. This laceration was caused by a bicycle accident with degloving of the lower lip and vestibular tissue from the alveolar bone.

**Fig. 24.17** Severely contaminated vestibular laceration. This was caused by a football player falling on the chin in the grass resulting in degloving of the soft tissue from the bone. All soil and foreign bodies have to be removed before closure can take place.

**Fig. 24.18** Vestibular laceration with exposed bone. In spite of meticulous cleaning and closure and suturing in the emergency phase this laceration opened up and bone was exposed 2 weeks after trauma. This exposure was treated by daily rinsing by the patient and weekly follow up visits with irrigations at the clinic. A superficial sequestration was seen after 3 weeks. Completed healing is seen 7 weeks after trauma.

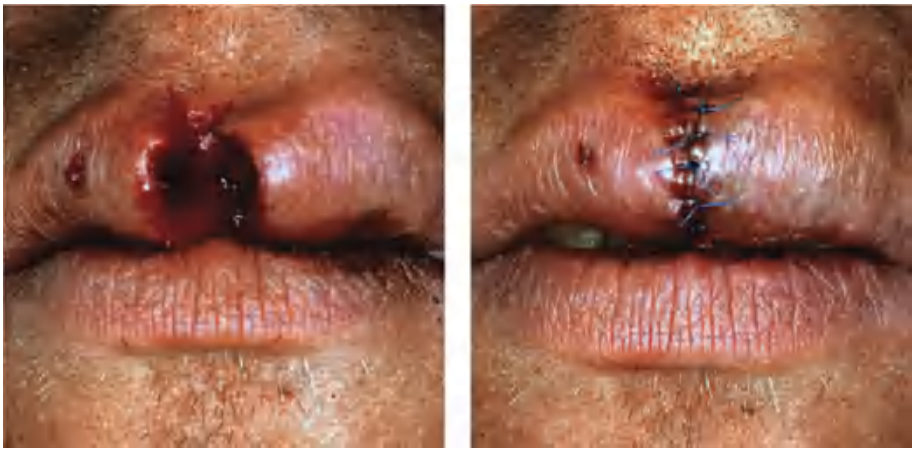


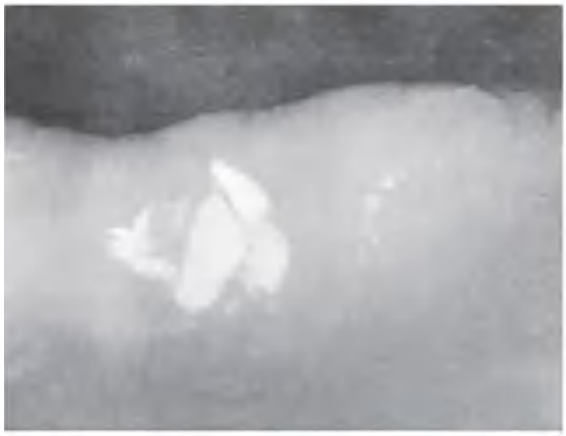
**Fig. 24.19** Split lip due to a frontal impact. This patient was hit in the face with a bottle, resulting in a split lip and lateral luxation of the right central incisor. The vermilion border is sutured first, whereafter the rest of the laceration is closed with interrupted sutures (e.g. Prolene® 6.0).

**Fig. 24.20** Radiographic appearance of typical foreign bodies. From left to right, the following types of foreign body are seen: tooth fragment, composite resin filling material, gravel, glass and paint. The different objects vary in radio-opacity.

**Fig. 24.21 Penetrating lip lesion with embedded foreign body**
This 8-year-old boy fell against a staircase, whereby the maxillary incisors penetrated the prolabium of the lower lip. Parallel lesions are found corresponding to each penetrating incisor.

**Radiographic investigation**
A dental film was placed between the lip and the dental arch. Exposure time is 1/4 of that for conventional dental radiographs. A large occlusal film is placed on the cheek and a lateral exposure taken using half the normal exposure time.

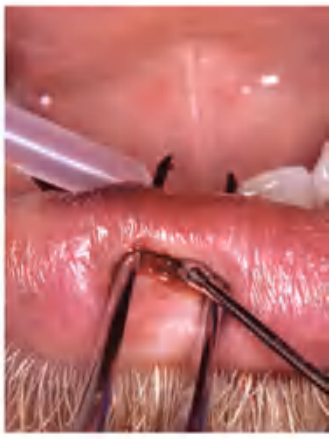
**Radiographic demonstration of multiple foreign bodies in the lower lip**
Orthoradial and lateral exposures show multiple fragments in lower lip. The lateral exposure could demonstrate that the fragments are equally distributed from the cutaneous to the mucosal aspect of the lip.



**Retrieved dental fragments**
The lip lesion is sutured after removal of tooth fragments, foreign bodies such as tooth fragments, plaque, calculus, and fragments from the impacting object usually become trapped within the lip.

**Fig. 24.22 Treatment of narrow penetrating lip lesion**
This 27-year-old man fell, causing the right central incisor to penetrate the lower lip and fracture. A radiograph shows that multiple tooth fragments are buried in the lip.

**Removing foreign bodies from the lip**
After administration of local anesthesia, the narrow penetrating wound is opened using a pincette. When the pincette is open, a rectangular wound is formed whereby two sides of the wound become clearly visible. Foreign bodies are removed, and the wound cleansed with saline.

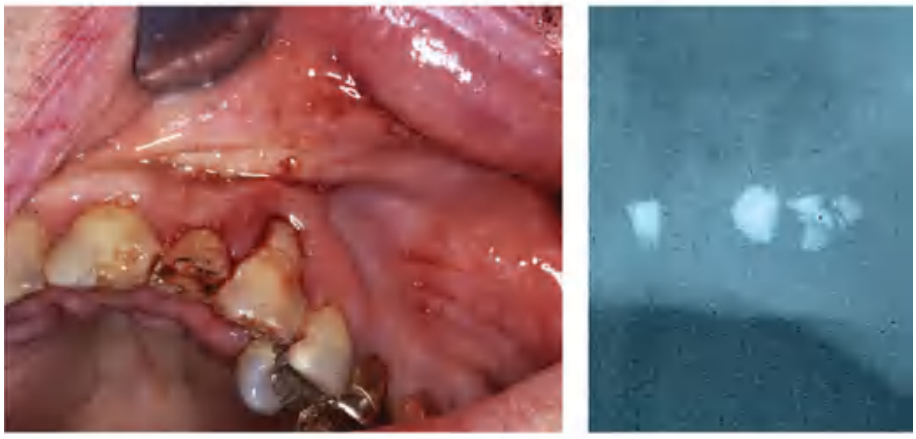


**Repeating the cleansing procedure**
The pincette is turned 90° and the procedure is repeated.

**Suturing the wound**
The wound is sutured with interrupted 6.0 silk sutures. A radiograph shows that all fragments have been removed.

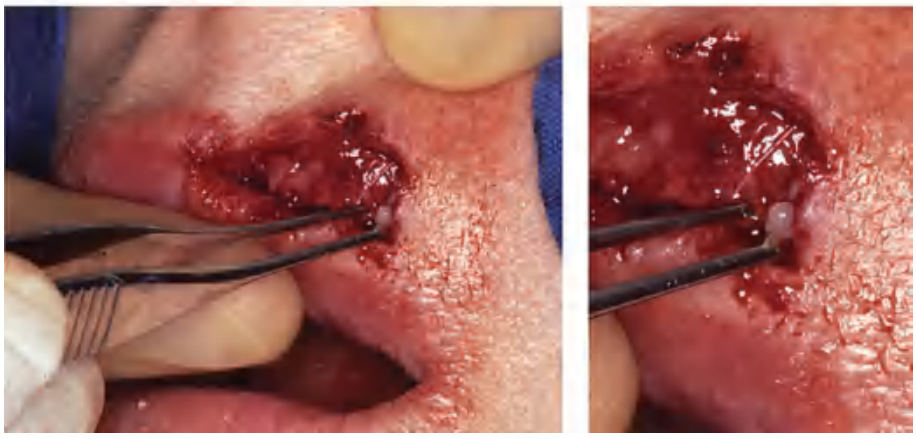
**Fig. 24.23 Treatment of a broad penetrating lip lesion**
Penetrating lesion of the upper lip in a 66-year-old woman due to a fall. Clinical appearance 4 hours after injury.

**Dental injuries**
The crown of the left central and lateral incisors and canine have been fractured while the lateral incisor and canine have been intruded. Soft tissue radiographs demonstrate multiple foreign bodies in the upper lip.

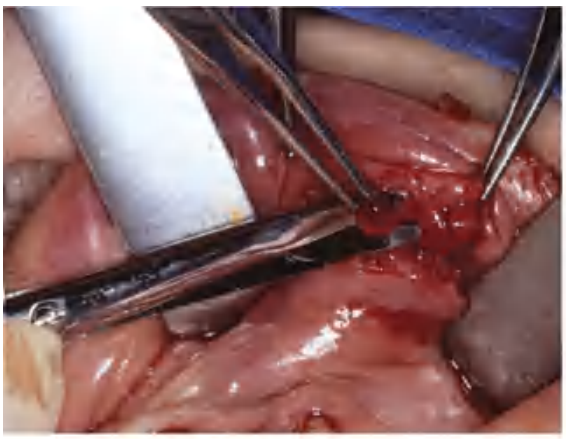


**Wound cleansing**
The cutaneous and mucosal aspects of the wounds are cleansed with a surgical detergent followed by saline.

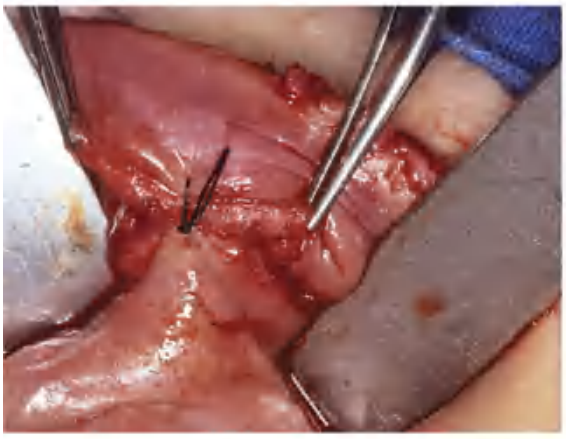
**Removal of foreign bodies**
All radiographically demonstrated tooth fragments are located and removed.



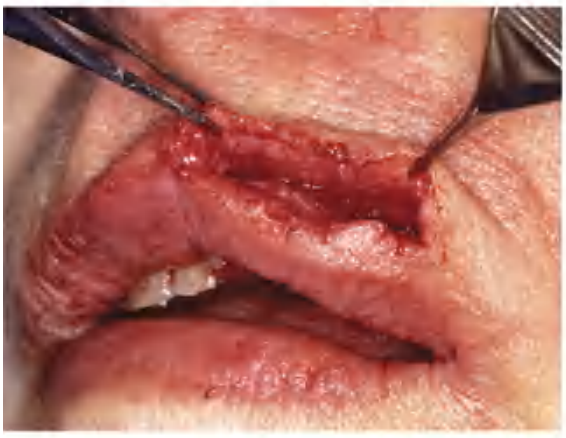
**Wound debridement**
Traumatized salivary glands are excised in order to promote rapid healing.



**Wound closure**
The oral wound is closed with interrupted 4.0 silk sutures.



**Repeated cleansing of the cutaneous wound**
The cutaneous aspect of the wound is cleansed with saline to minimize contamination from closure of the mucosal wound.



**Buried sutures**
As a matter of principle, buried sutures should be kept to a minimum number; and, when indicated, be resorbed over a short period of time (e.g. Vicryl® sutures). The point of entry of the needle should be remote from the oral and cutaneous wound surfaces in order to place the knot (i.e. the most infection-prone part of the suture) far from the wound edges; that is, at approximately one-half the total thickness of the lip.

**Assessing cutaneous wound closure**
After closing the muscular tissue, the cutaneous part of the wound is evaluated. It is important that after muscular approximation, the wound edges can be approximated without tension. If this is not possible, approximation of the muscular part of the wound must be revised.

**Suturing the cutaneous wound**
Wound closure is principally begun at the vermilion border. In cases where the wound is parallel to the vermilion border, the first suture is placed at a site where irregularities of the wound edge ensure an anatomically correct closure. The wound is closed with fine, monofilament interrupted sutures (Prolene® 6.0), under magnification (i.e. using spectacles with a 2 or 4 × magnification).

**Suturing completed**
The wound is now fully closed, and antibiotics administered (i.e. penicillin, × 4, for 2 days in doses according to weight).

**Healing 6 weeks after injury**
There is a minimum of scarring.

the wound is debrided for contused muscle and salivary gland tissue. The anatomy of the wound should be respected. Never excise wounds to make long straight scars which invariably are more visible. Thereafter the wound is carefully rinsed with saline; and a check is made to ensure that bleeding has been arrested.

When suturing the lip special attention must be paid to carefully approximate the transition of skin to mucosa (vermilion border) as any inaccuracy in wound closure will be very esthetically apparent. A minimum of sutures should be used, as deep sutures in contaminated wounds have been shown to increase the risk of infection (9). In penetrating lesions, the mucosal side of the wound is first sutured so that no saliva can enter the wound (Fig. 24.23). A few resorbable 5.0 sutures are placed in the musculature to reduce tension on the cutaneous sutures. Thereafter, the cutaneous part of the wound is closed with 6.0 sutures. Magnifying lenses, e.g. ordinary spectacles with 4×loupes can be used to ensure meticulous suturing. Intracutaneous sutures may be used for cutaneous closure in esthetically-sensitive areas (Fig. 24.24). Adhesive tape/strips may be used in addition to relieve tension (Fig. 24.25).

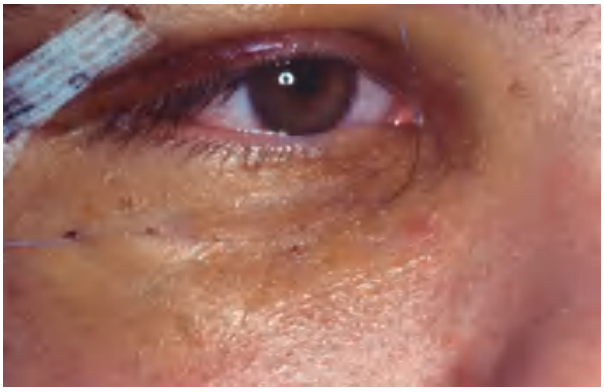


**Fig. 24.24** Intracutaneous suture of an infraorbital soft tissue wound. Note the excellent adaptation of tissue without stitch marks 5 days after suturing.

## Tongue lacerations

Other than in patients suffering from epilepsy, tongue lesions due to trauma are rare. In the former instance, bite lesions may occur along the lateral part of the tongue during seizures. Furthermore, following an impact to the chin with the tongue protruding, a wound may be caused by incisor penetration through the apex of the tongue.

A wound located on the dorsal surface of the tongue should always be examined for a ventral counterpart (Fig. 24.26). If there are concomitant crown fractures, fragments may be located within the wound. These fragments can be revealed by a radiographic examination (see above).

Treatment principles include cleansing of the wound, removal of foreign bodies and suturing of the dorsal and ventral aspects of the lesion. After administration of anesthesia (local, regional or general), foreign bodies are retrieved, the wound cleansed with saline, and the wound entrances sutured tightly (Fig. 24.26). A clinical study has shown that suturing is not critical (85). Deep lacerations in the tongue will increase the risk of postoperative bleeding and some of these patients should be observed postoperatively so that continuing deep bleeding in the tongue and floor of the mouth with life threatening occlusion of airways can be prevented.

## Avulsion of tissue

Tissue loss may be seen intra- and extra orally. In case of loss of gingival tissue, a gingivoplasty should be performed whereby flaps are elongated by the placement of periosteal incisions (Fig. 24.13).

Tissue losses on the skin side are complex injuries from a treatment point of view in the emergency phase. There is a high risk for extensive scar contraction resulting in esthetic failure if not properly handled by an experienced specialist. Minor injuries in young individuals may be left for secondary healing and possible later reconstruction (Fig. 24.8). With larger avulsion injuries secondary healing should be avoided due to excessive scar formation and local flaps or

**Fig. 24.25** Laceration of the lower lip. This laceration was closed by intracutaneous suturing. To relieve tension two supplementary separate sutures were placed at the angles of the Z shaped laceration. To cover the sutured wound and to further relieve tension a tape was finally placed.

**Fig. 24.26 Treatment of a penetrating tongue lesion**
This 10-year-old girl suffered a penetrating tongue lesion. Note the parallel lacerations on the dorsal and ventral surfaces of the tongue.

**Radiographic examination**
A dental film is placed under the extended tongue and exposed at 1/4 the normal exposure time. The exposure demonstrated several tooth fragments embedded within the tongue.

**Removal of tooth fragments**
After administration of a regional anesthetic all tooth fragments were retrieved. It is important that all radiographically demonstrated fragments are also retrieved in order to prevent infection and/or scar formation.

**Suturing the tongue wound**
After all foreign bodies have been removed, the wound is cleansed and sutured on the dorsal and ventral surfaces.

skin grafts should be used in the early treatment of these cases (16–18).

Tissue losses are sometimes seen with animal bites to the face and are usually caused by dogs (19–21, 87–89, 99). Because of contamination from the dog's saliva the main concern is infection. Copious irrigation by saline and debridement should be performed. Antibiotics should always be administered regardless of duration. Rabies vaccine should be considered depending on the status of the dog. Ideally the animal should be caught and observed. Antibody test should be performed to see if the animal has rabies. The incubation period of rabies is 2–8 weeks in humans and the patient can be treated within this period with a rabies vaccination protocol.

## Wound closure materials and principles

The management of facial soft tissue wounds is not a well-documented area and wound healing often leads to scarring (69). Sutures and tapes/strips are the traditional methods for closure of wounds in the skin of the face. Strips can be used together with sutures to relieve tension (Fig. 24.25). Strips can also be used alone to close small superficial wounds. Tissue adhesives e.g. cyanoacrylate or fibrine glue, have been used in emergency rooms (22–25, 91, 92, 94, 96). The advantage is that the closure procedure can be performed in less time and with less pain as compared to suturing. There are some reports comparing tissue adhesive and sutures in pediatric facial lacerations (23, 26, 27). The results showed comparable esthetic results between tissue adhesives and suturing. However, it should be borne in mind that lip lacerations were excluded from these studies. There may also be problems associated with this location, such as the child licking or biting off the glue. Moreover, a statistically significant increase in wound dehiscence has been found with tissue adhesives (28). Further studies are needed to learn more about the indications for tissue adhesives in lip injuries before they are generally applied (29, 30).

A general principle in wound treatment is the approximation of wound edges in order to reduce the distance for the wound healing module and thereby increase the speed of healing. This principle has, unfortunately, not been supported by animal experiments in which sutures were used to approximate wound edges (27, 30–35). The explanation for these findings could be that, as well as approximating the wound edges, suturing also induces ischemia of wound edges and acts as a wick, leading bacteria into the wound. Thus, the placement of just a single suture, however, significantly decreases the number of bacteria needed to cause wound infection (32) (see Chapter 1).

The tissue response to various types of sutures in the oral mucosa has been studied extensively in animals (36–42) and in humans (43, 44), and a vigorous inflammatory reaction around the sutures could be demonstrated. The general findings in these experimental studies have been that silk and catgut sutures (plain or chromic) elicited a very intense clinical and histological inflammatory reaction after 3, 5 and 7 days; whereas polyglycolic acid showed considerably less clinical and histological reaction (42).

The greatest part of the inflammatory reaction is probably related to the presence of bacteria within the interstices of multifilament suture materials (34, 37–40). Thus, the impregnation of multifilament sutures with antibiotics has been shown to decrease the resultant tissue response (40). A further finding has been that monofilament sutures (e.g. nylon) display a significant reduction of adverse reactions compared to multifilament sutures, a finding possibly related to a reduced wick effect of monofilament sutures (41–45).

Monofilament suture material has been developed from a very inert and tissue compatible biomaterial made of polytetrafluoroethylene (PTFE). A comparison between the tissue response in oral mucosa between this material and silk sutures shows definitively less tissue response in the former (46).

Finally, it should be borne in mind that it has been shown experimentally that there is an increased risk of infection with both increased suture diameter and submucosal/subcutaneous suture length (9).

The essential lesson of all these experiments in suturing and subsequent wound healing is therefore that suturing is still the best overall method to use intra- and extraorally, using a minimal number of sutures with a small diameter, preferably monofilament and, finally, instituting early suture removal (i.e. after 3–4 days in oral tissues), eventually in two stages, i.e. 3 and 6 days.

The choice of sutures varies with the surgeon (86). There is an ongoing development to use synthetic absorbable sutures in the mucosa such as polyglycolic acid (Dexon®) polyglactic acid (Vicryl®) or polydioxanone (PDS®). Although absorbable sutures can also be used for skin closure, many surgeons prefer non-absorbable sutures such as nylon (Dermalon®, Ethilon®), polypropylene (Prolene®) or Polybutester (Novofil®) for skin closure. Tape/strips may be used for skin closure in addition to sutures to relieve tension or used as an alternative to sutures for closing shallow, small wounds. The use of tissue adhesives/glue have been reported as an alternative to suturing (81). However, in the perioral region, which is an area of high tensions, is not yet sufficiently evidence-based and show a higher rate of dehiscence (81, 82).

## Antibiotic prophylaxis

It is surprising that the benefit of antibiotic therapy in oral soft tissue injuries has only been sparsely documented (90, 93). Due to their pathogenesis, almost all of these injuries can be considered contaminated with microorganisms and sometimes with foreign bodies. Based on this assumption, it has been proposed that the surgical treatment of mucosal and cutaneous wounds should always be supported by prophylactic antibiotic coverage (47). However, clinical evidence for the benefit of this treatment is presently tenuous

(48–52) (Table 24.1). Thus, no effect of antibiotic therapy could be shown on the rate of infection in cutaneous and/or mucosal wounds (86). In penetrating wounds to the oral cavity, conflicting results have been reported (48, 49) (Table 24.2). Antibiotics shall be used restrictively but can be of supportive value in certain situations (77–79).

Based on these findings it seems reasonable – until further studies are presented – to restrict the prophylactic use of antibiotics in soft tissue wounds to the following situations:

- When the wound is heavily contaminated and wound debridement is not optimal (e.g. impacted foreign bodies or otherwise compromised wound cleansing);
- When wound debridement has been delayed (i.e. more than 24 hours) (41);
- Penetrating lesions through the full substance of the lip (5);
- When open reduction of jaw fractures is part of the treatment. In these situations, the benefit of short-term antibiotic coverage preceding and following osteosynthesis has been documented (47, 53–55) (see also Chapter 21). This relationship contrasts with the lack of effect of antibiotic treatment in relation to 'clean' orthognathic surgery (56–59).
- When the general defense system of the patient is compromised (8) (e.g. insulin-dependent diabetes, alcohol abuse, immunocompromized patients, patients with risk for developing infective endocarditis (82, 83)); and
- Human or animal bite wounds (60–62).

If prophylactic antibiotic coverage is decided upon, early institution is very important. Thus, experimental studies have shown that the first 3 hours after trauma are critical; i.e. to obtain an optimal effect from the antibiotics, they should be administered within this period (see Chapter 1). If delayed, contaminating bacteria may multiply and invade the wound (12–14). Antibiotics should therefore be administered before surgery and maintained for 24 hours (63). Prolonged administration does not optimize healing (64–66), and has a serious effect upon the ecology of the oral microflora. The antibiotic of first choice is penicillin (phenoxymethyl penicillin). The dosage (adults) should be: 2 million units (=1.2 g) orally at once followed by 2 million units (1.2 g) 3 times for 1 day. For children the dosage is given in relation to body weight. If the patient is allergic to penicillin, clindamycin should be administered. The dosage (adults) should be: 600 mg orally at once followed by 300 mg 3 times for 1 day. In children the dosage is 15 mg/kg body weight given 3 times for 1 day.

## Tetanus prophylaxis

Tetanus prophylaxis should always be considered in the case of contaminated wounds. In a previously immunized patient (i.e. longer than 10 years prior to injury), a dose of 0.5 ml tetanus toxoid should be given (booster injection). In unimmunized patients, passive immunization should be provided.

**Table 24.1** Effect of systematic antibiotic treatment subsequent to various combinations of cutaneous and oral mucosal wounds.

| Type of lesion | Antibiotic treatment | No. of cases | Frequency of infection | Probability level* |
|---|---|---|---|---|
| Mucosal wounds | – | 32 | 4 (13%) | 0.66 |
| | + | 17 | 2 (12%) | |
| Mucosal and penetrating wounds (penetrating lesions) | – | 33 | 11 (33%) | 0.16 |
| | + | 24 | 4 (17%) | |

*Probability level based on Fisher's exact test.
Source: After GOLDBERG (48) 1965.

**Table 24.2** Effect of systemic antibiotic treatment subsequent to various combinations of facial cutaneous and oral mucosal wounds.

| Type of lesion | Antibiotic treatment | No. of cases | Frequency of infection | Probability level* |
|---|---|---|---|---|
| Mucosal wounds | – | 23 | 0 (0%) | 1.0 |
| | + | 12 | 0 (0%) | |
| Cutaneous wounds | – | 85 | 2 (2%) | 0.06 |
| | + | 51 | 6 (12%) | |
| Mucosal and cutaneous wounds | – | 21 | 0 (0%) | 1.0 |
| | + | 23 | 0 (0%) | |
| Mucosal and penetrating wounds (penetrating lesions) | – | 24 | 3 (13%) | 0.08 |
| | + | 48 | 15 (31%) | |

*Probability level based on Fisher's exact test.
Source: After PATERSON et al. (49) 1970.

## Essentials

### Type of soft tissue lesion

- Abrasion
- Laceration
- Contusion (including hematoma)
- Tissue loss (avulsion)

### General treatment principles

- Contusions need not be treated but may indicate an underlying bone fracture.
- Abrasions and lacerations should be thoroughly cleansed, and all foreign bodies removed.
- Larger avulsion injuries should be treated by specialists.

### Gingival lacerations (Figs 24.11–24.14)

- Rinse the wound and surroundings with a wound detergent.
- Reposition displaced gingiva.
- Place a few fine sutures (4.0 or 5.0 Vicryl®, Dexon® or PDS®).
- Instruct in good oral hygiene including daily mouth rinse with 0.1% chlorhexidine.
- Remove sutures after 4–5 days.

### Lip lacerations

Establish whether the injury is a penetrating wound of the lip or a laceration of the vermilion border (split-lip lesion).

#### *Penetrating lip wounds (Figs 24.22 and 24.23)*

- Administer antibiotics if indicated (see Antibiotic prophylaxis).
- Take a radiograph of the lip with decreased exposure time.
- Use regional anesthesia.
- Rinse the wound and surroundings with a wound detergent.
- Remove foreign bodies and contused muscle and salivary gland tissue.
- Suture the labial mucosa (4.0 or 5.0 Vicryl®, Dexon®, PDS®).
- Rinse the wound again with saline.
- Suture the cutaneous wound with fine sutures (6.0 nylon or Prolene®). Take special consideration of the vermilion border.
- Remove sutures after 4–5 days.

#### *Split-lip wounds (Fig. 24.19)*

Use the same procedure as for penetrating lip lesions. However, in this case a few buried resorbable sutures are indicated (e.g. Dexon® 4.0/5.0).

### Tongue lacerations

Examine whether the injury is a penetrating wound or a lesion of the lateral border.

#### *Penetrating tongue wounds (Fig. 24.26)*

- Administer antibiotics if indicated (see Antibiotic prophylaxis).
- Take a radiograph of the tongue with decreased exposure time.
- Use a regional or general anesthesia.
- Rinse the wound with saline.
- Remove foreign bodies.
- Rinse the wound again with saline.
- Suture the mucosal wounds.
- Remove sutures after 4–5 days.

#### *Lateral tongue border wounds*

After administration of a regional anesthetic the wound is rinsed and sutured. Buried resorbable sutures are sometimes indicated in order to approximate the wound edges and relieve tension on the mucosal sutures.

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# 25

# Management of Trauma-Related Pulp Disease and Tooth Resorption

M. Cvek, P. V. Abbott, L. K. Bakland & G. S. Heithersay

## Introduction – Tribute to Miomir Cvek, DDS, Odont Dr. 1927–2008

It was his concern for preserving traumatized immature teeth that inspired Miomir Cvek to devote much of his professional life to the application of calcium hydroxide in the treatment of such teeth. As a result, Dr. Cvek's remarkable career became intimately linked to pediatric dentistry, endodontics and dental traumatology.

Early in his career at the Eastman Institute in Stockholm, Sweden Dr. Cvek became interested in endodontic problems associated with traumatized teeth with open apices. He was instrumental in discovering how calcium hydroxide could be used to promote apical healing by inducing an apical hard tissue barrier. He also recognized the value of preserving pulp health in young traumatized teeth and developed new techniques including the one that is still often referred to as the 'Cvek-type pulpotomy.' The clinical techniques were based on evidence from research on pulp responses to materials such as calcium hydroxide. The span of his involvement in dental traumatology further included research on revascularization role of antibiotics in replanted teeth.

What made his clinical recommendations significant as treatment guidelines was his insistence on meticulous clinical procedures and careful outcomes evaluations, combined with in-depth laboratory and clinical research. He earned well deserved recognition for his work, including being presented with the 2003 Ralph Sommer Award from the American Association of Endodontists for his publications on vital pulp therapy. Dr. Cvek's involvement with this textbook included writing chapters on endodontics and the use of calcium hydroxide in traumatized teeth in both its 3rd and 4th editions. In additions, his interest in, and knowledge about dental trauma made him a much-valued consultant and advisor on content issues. The loss of this dear friend and colleague in 2008 was sad; his memory will live on through the legacy of all his contribution to dental trauma management in young accident victims.

This chapter is dedicated to the memory of Professor Miomir Cvek, who always searched for sound biological explanations for complicated clinical phenomena.

## Trauma-related pulp disease

The majority of traumatic dental injuries affect children and adolescents when root development is still in progress. Management of such injuries requires that all efforts be made to preserve the pulp for continued root maturation. Historically, calcium hydroxide has been the main dental material used in treatment of teeth needing pulp and apical repair. While new materials in the bioceramic category (i.e. mineral trioxide aggregate) have to a large extent replaced calcium hydroxide, it is still used and will probably have a place in dental trauma treatment for some time.

## Crown and crown-root fractures without pulp exposure

To the extent that a tooth with crown-root fracture is considered savable and the pulp is not exposed, the treatment is similar to that of a tooth with crown fracture.

### Healing and pathology

Crown fractures involving enamel and dentin result in exposure of dentinal tubules to the oral environment. The description of crown fractures is covered extensively in

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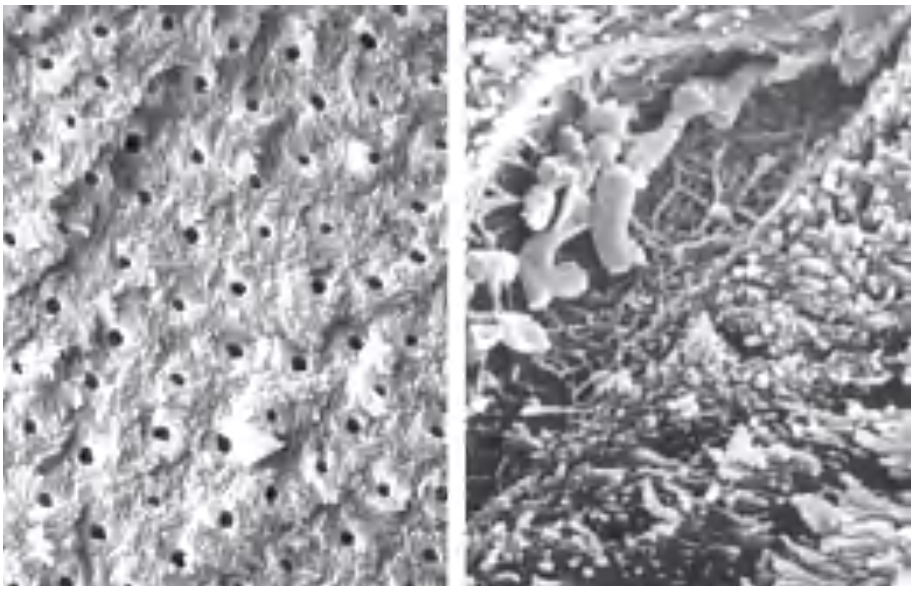
Chapter 13. For the purpose of this chapter, it must be stressed that protecting the pulp from bacterial toxins and bacterial invasion is of utmost importance, especially in cases of concomitant luxation injuries (1). Absent injury to the apical blood supply from tooth luxation, shallow exposure of dentin causes only minor changes in the pulp that may resolve, and the exposed dentinal tubules will be sealed off by formation of tertiary reactive dentin (2, 164). Deeply exposed dentin, if left unprotected, may allow bacteria and their components in dental plaque on the fracture surface to penetrate the tubules and cause inflammation in the underlying pulp (3–6) (Figs 25.1 and 25.2). Reparative or degenerative changes depend on elapsed time since the injury and the distance between the fracture surface and the pulp (164).

In young permanent teeth, wide dentinal tubules may be an additional factor (Fig. 25.3). If irritation is eliminated by treatment of the exposed dentin, localized inflammation in the pulp may resolve, with damaged tissue being replaced by tertiary dentin (7–9, 165). Left unprotected, pulps may undergo infection-related necrosis and the teeth may require endodontic treatment (see later).

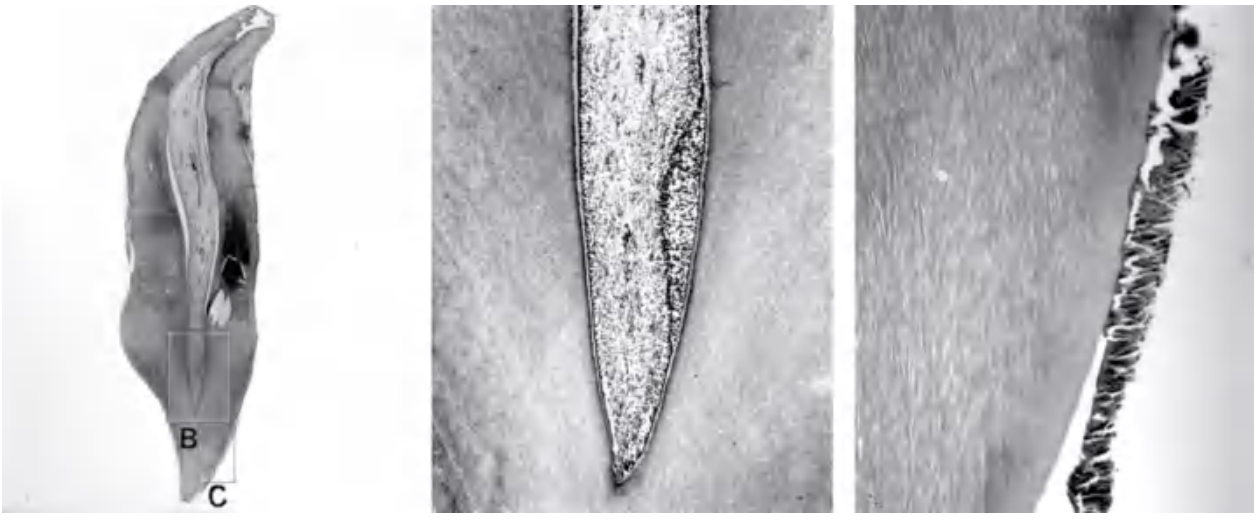
## Treatment

Crown fractures without pulp exposure can be restored quite efficiently either with resin composite materials or by re-bonding the fractured crown fragment, if available. The restorative treatment procedures are described in Chapter 29.



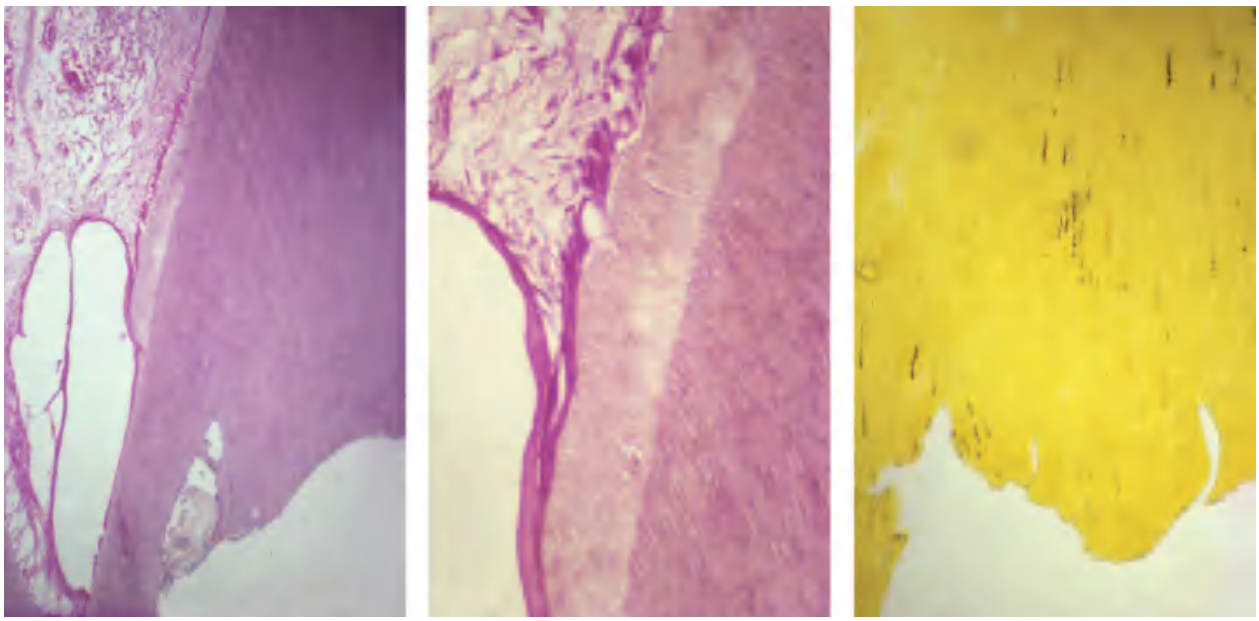
A B

**Fig. 25.1** Surface of fractured dentin. A. There are 36 000 exposed tubules per $mm^2$. ×1200. Source: Reproduced from (2) with permission. B. Invasion of bacteria into the tubules of acid etched dentin, after exposure for 1 week. ×400. Source: Reproduced from (3) with permission.




A B C

**Fig. 25.2** Early changes in the pulp of a primary incisor 3 days after an uncomplicated crown fracture. A. Low power view. ×3. B. Displacement of odontoblasts and slight infiltration of leukocytes corresponding to the fracture surface. ×30. C. Bacterial accumulation along the fracture surface. ×75.



A B C

**Fig. 25.3** Late pulpal changes in an untreated uncomplicated fracture. A primary incisor 4 months after injury. A. Necrosis of the pulp tissue subjacent to the fracture surface. ×32. B. Formation of reactive dentin at a distance from the fracture surface. ×60. C. Penetration of bacteria through the dentin, from fracture surface towards the pulp, Brown & Brenn bacterial stain. ×200.

Bonding systems have been reported to increase the bonding strength of composite restorations, reduce microleakage, decrease permeability of dentin and possess an antibacterial effect (10–25). Microleakage due to poor bonding or loss of bonding may however, result in pulp inflammation. It has been suggested that in deep carious lesions and deeply exposed crown fractures, a calcium hydroxide liner should be placed under the composite restoration (18, 23). In such teeth, treatment with calcium hydroxide may remove bacteria from the fracture surface and give a potentially inflamed pulp a chance to recover and seal off the exposed dentinal tubules by deposition of tertiary dentin (26–28, 164, 165). Moreover, calcium hydroxide significantly decreases the permeability of dentin to penetration by bacterial components into the pulp, which may provide additional pulp protection under a subsequent restoration (26–28).

### Prognosis

Restoration of a fractured crown with resin composites and re-bonding fractured crown segments can be regarded as effective tooth repair. However, it should be noted that bacterial leakage has been demonstrated in the gaps between composites and dentinal walls in experimental cervical cavities (28, 29). Fortunately, most uncomplicated fractures do not involve the cervical area; thus the risk of later pulp damage seems to be minimal (30–32) (see Chapter 13).

## Crown and crown-root fractures with pulp exposure

A crown fracture through the pulp chamber causes laceration and exposure of the pulp to the oral environment. Healing does not occur spontaneously, and untreated exposures ultimately lead to pulp necrosis, a process in which bacteria are the dominant factor (33). The early changes in the pulp are hemorrhage and local inflammation, caused by breakdown products from lacerated tissue and bacterial toxins (34, 35). The fibrin clot that forms over the wound surface resolves after a couple of days. The subsequent changes can be proliferative, such as a pulp polyp, or destructive, such as pulp necrosis and abscess formation. The most common early response is formation of granulation tissue at the exposure site, (Fig. 25.4). In studies of patients aged 7 to 16 years with crown-fractured incisors, seen 12 hours or more after injury, hyperplasia was a typical pulp reaction, regardless of the extent of the exposure (36, 37). Similar observations have been made in multi-rooted human teeth when the pulp was exposed by removal of the crown and left untreated for 2 weeks (38). These findings have also been supported by experimental studies in which pulps of monkey incisors were exposed by either crown fracture or grinding. In these experiments, pulp changes were characterized by a proliferative response, invariably associated with only superficial inflammation, extending less than 2 mm in to the pulp from the exposure site (39, 40) (Fig. 25.4).

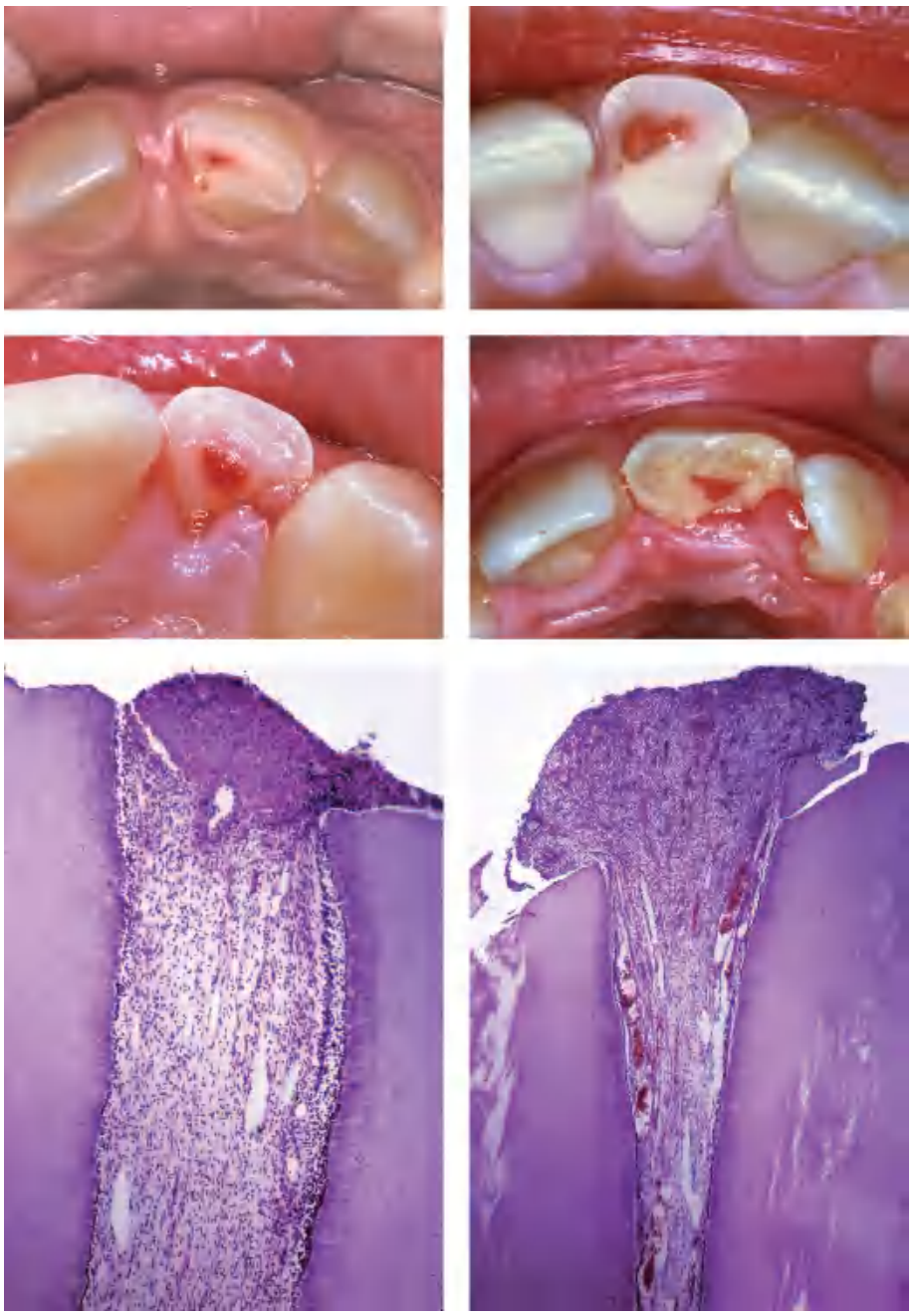


**Fig. 25.4 Clinical appearance of pulp exposures at various observation periods**
Four hours after injury, hemorrhage is the predominant finding. After 2 days (right), granulation tissue is formed at the exposure site.

**Clinical appearance**
One week after injury (left): hyperplasia of the pulp. Three months after injury (right): a polyp has formed and is covered with plaque.

**Histologic appearance**
Pulp reactions to experimental exposure in monkey incisors. After 2 days (left), the exposed surface is covered with fibrin, under which a limited pulp proliferation can be seen with moderate infiltration of inflammatory cells to a depth of 1.4mm. ×90. After 1 week (right), there is proliferation of the pulp through the exposure and moderate inflammation in the proliferated tissue and a mild infiltration of inflammatory cells in the pulp to a depth of 0.4mm. ×32. Source: Reproduced from (13).

A protective proliferative response of the pulp is probably favored by an exposure that permits salivary rinsing and prevents impaction of contaminated debris, as occurs in caries or experimentally induced cavities (39, 41).

Early necrosis of an exposed pulp is a rare occurrence. A common etiological factor seems to be plaque and contaminated debris that are permitted to accumulate over the exposure site; this may allow bacteria to settle into the injured pulp tissue and lead to necrosis (Fig. 25.5). Thus, in monkey incisors in which the coronal cavities were left open for 7 days after pulpotomy, impaction of contaminated food debris caused deep abscesses in the underlying pulp tissue (39).

## Treatment

The treatment goal for teeth with exposed pulps is to preserve vital, non-inflamed pulp tissue that can be walled off by a continuous hard tissue barrier. Pulp capping or shallow pulpotomy can achieve this goal (42, 164–166). If these procedures cannot be performed, or if they fail, conventional root canal therapy can be the next treatment option (see later). Factors to be considered in choosing treatment options include maturity of the tooth, concomitant luxation injury, and age of the patient.

The maturity of the traumatized tooth often dictates the ranking of treatment options. In young patients with immature teeth and incomplete root formation, pulp

A B

**Fig. 25.5 Necrosis of the pulp at the exposure site**
A. Necrosis of the pulp tissue in a tooth in which the exposure was only covered with a surgical packing for 2 weeks. B. Necrosis of the pulp at the exposure site 4 weeks after injury. The tooth was initially restored with a temporary crown, seated with temporary cement which was lost after 3 weeks.

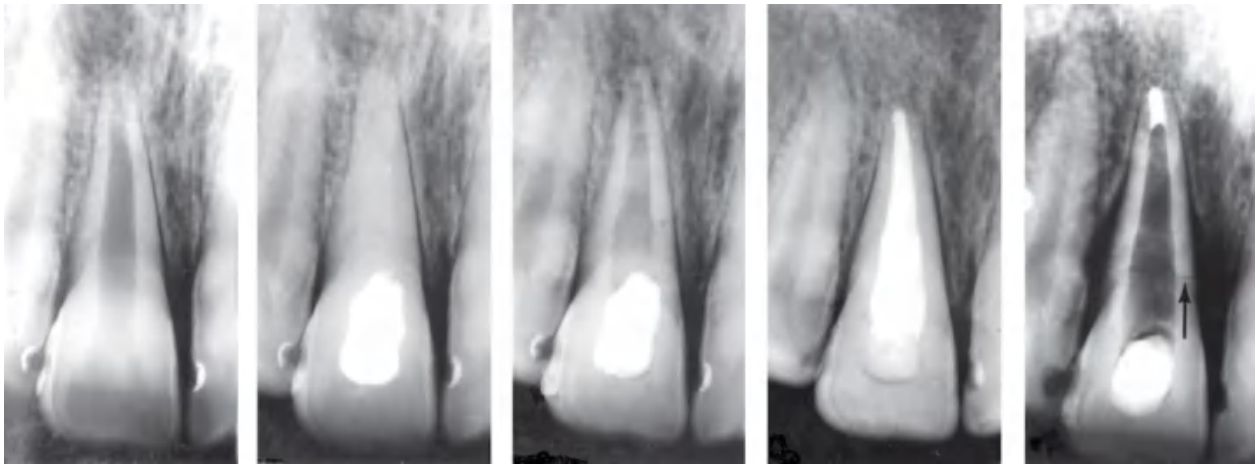



**Fig. 25.6** Spontaneous root fracture due to thin dentinal walls after pulp necrosis at an early stage of root development and excessive canal instrumentation. Permanent incisor treated with calcium hydroxide and root filled with gutta-percha. Cervical fracture of the thin dentinal walls (arrow) occurred during chewing 6 years later.

preservation is essential for continued proper growth and development of the tooth and the supporting alveolar structures. In patients with fully developed teeth, conventional root canal procedures can be recommended; it should be noted however, that even in fully developed teeth the procedures described for pulp protection in immature teeth may also be an option. It should not be overlooked that teeth in young patients where the apical openings are small, the roots may still need continued development, favoring pulp preservation techniques to allow strengthening of the roots (43–45, 167) (Fig. 25.6).

Lauridsen (1) has provided extensive data to support the observation that concomitant luxation injuries are significant factors in the outcomes of pulp survival after crown fractures. Luxation injuries affect the blood supply to the pulp and disruption of blood flow reduces the ability of the pulp to defend itself against bacterial invasion. Pulp survival is possible absent bacterial invasion so coronal protection is necessary for successful revascularization of a pulp deprived of its blood supply. Successful revascularization may allow further root development (46, 47) (Fig. 25.7).

The effect of age on teeth with pulp exposures is probably limited to the availability of a good blood supply to the traumatized pulp. Experimentally, an inferior response to injury and treatment has been observed in pulps of old rats compared to young ones (34, 35). Yet, successful treatment of pulp exposure due to either trauma or caries in older patients has repeatedly been reported (34, 48). For various reasons, degenerative changes in the pulp undoubtedly increase with age. One reason may be due to autophagy in an effort to preserve cell function (164). Thus, removal of the pulp could be a more successful procedure, although no age limit can be set for either pulp preservation or removal. However, outcomes of conservative treatment, i.e. capping or shallow pulpotomy, may be reduced in teeth with degenerative or inflammatory changes, e.g. in teeth with reduced pulp lumen due to trauma or age, or in periodontally involved teeth in adults (49–52).

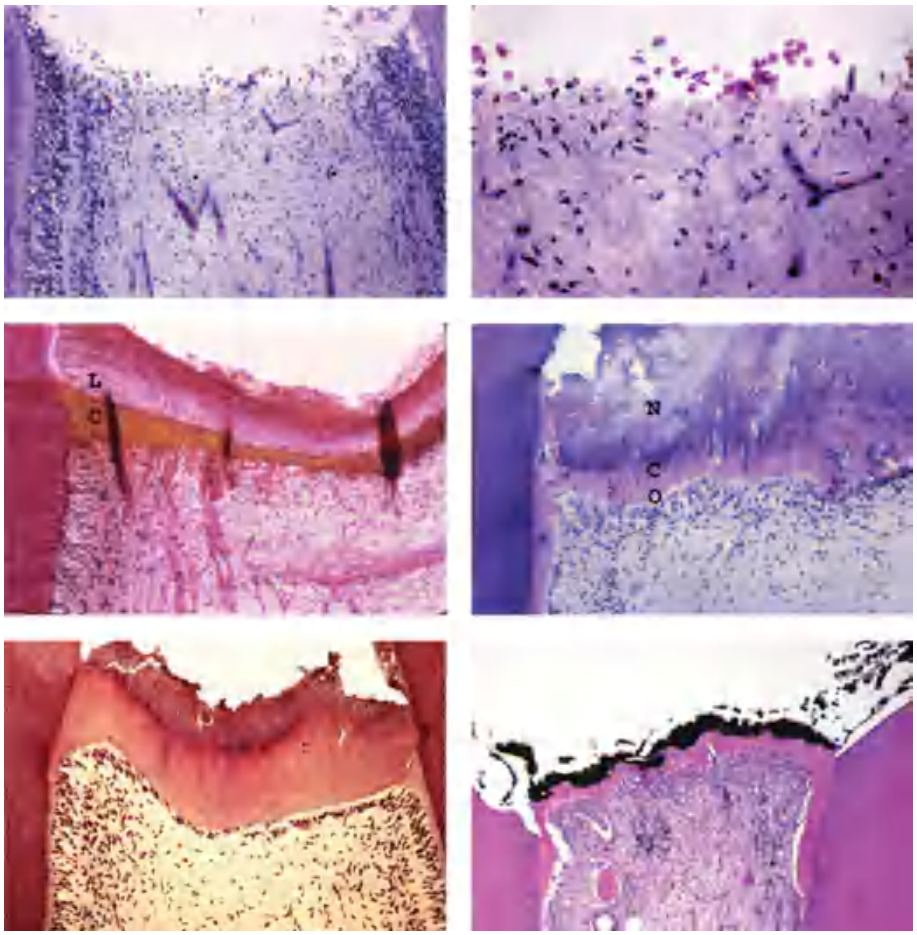
Surgical pulp procedures invariably cause further injury to the remaining pulp and should be kept to a minimum. Various instruments have been recommended for pulp amputation, such as spoon excavators, slowly rotating

**Fig. 25.7** Treatment of pulp exposure in a slightly dislocated immature incisor. Formation of a hard tissue barrier in the pulp, continued root development and diminishing of the pulp subsequent to partial pulpotomy.

**Fig. 25.8 Effect of pulp amputation technique on the pulp**
The effect of cutting with an abrasive diamond instrument in a high speed contra-angle hand piece; no tissue damage below the wound surface. ×25 and 100. Source: Reproduced from (31).

**Effect of calcium hydroxide on the pulp**
Left: Formation of liquefaction necrosis (L) close to calcium hydroxide. Coagulation necrosis (C) close to the vital pulp is colored yellow. ×75. Right: formation of hard tissue 17 days after capping; layer of necrotic tissue (N) and dark stained band of necrotic and later calcified tissue (C) and osteoid-like tissue (O) with lining cells. ×75. Source: Reproduced from (35).

**Formation of a hard tissue barrier**
A completely formed hard tissue barrier after capping with calcium hydroxide paste (Calasept®). ×25. Right: formation of hard tissue barrier without visible intermediate layer of necrosis after capping with hard setting calcium hydroxide compound (Dycal®). ×25. Source: Reproduced from (39).




round burs and high-speed abrasive diamonds. Of these, the spoon excavator, successfully used in molars, has proven unsuitable in young incisors (53). Slowly rotating instruments are known to inflict significant injury to the remaining pulp, limiting the chance of survival (54–56). However, it has been shown that injury to the underlying tissue is minimal when abrasive diamond is used at high speed to remove part of the pulp, provided that the bur and tissues are adequately cooled (57) (Fig. 25.8). If effective cooling is not possible, e.g. when the amputation site is deep in the root canal, a round bur at low speed should be used in order to avoid overheating the pulp.

A commonly used wound dressing has been calcium hydroxide. This material has been widely used for the treatment of accidentally exposed pulps since the first histological studies appeared decades ago (58–60). Calcium hydroxide

has been the subject of extensive experimental research and several explanations have been offered for its effect on the vital pulp (61, 164–166). When placed on vital pulp tissue, calcium hydroxide causes a superficial tissue necrosis, approximately 1–1.5 mm in depth (62). This necrotic tissue consists of several layers, including a layer of firm coagulation necrosis in close contact with vital pulp tissue, which seems not to be appreciably affected (63). Based on observations in experiments with sound human teeth, it has been suggested that it is neither calcium hydroxide nor its components, but the low-grade irritation from coagulation necrosis that induces defensive reactions in the pulp, resulting in formation of a demarcating hard-tissue barrier (63–65). The underlying tissue seems to react to this irritation by producing collagen that is subsequently mineralized (Fig. 25.8). Accordingly, formation of the hard tissue barrier itself can be regarded as a defensive, probably stereotypic, reaction of the pulp to a non-specific low-grade irritant (168). This hypothesis of a low-grade irritation that is not strong enough to destroy the pulp tissue but is sufficient to elicit defensive reactions was strongly supported by findings of the formation of hard tissue barriers after only 10 minutes' treatment with calcium hydroxide or capping with cyanoacrylate, a material possessing none of the calcium or hydroxyl ions that are thought to be responsible for induction of hard tissue (65) (Fig. 25.9). Even though in experiments, cyanoacrylate appears to stimulate hard tissue formation in pulp tissue it is not recommended for treatment of exposed pulps (66, 165).

Various materials have been tested for pulp dressing (67, 68). Indeed, in the absence of bacteria, healing of the pulp with or without formation of hard tissue has been observed after capping with various materials, such as cyanoacrylate, zinc-phosphate, tricalcium phosphate or silicate cements and various composite materials. The results, obtained mostly in experimental animals such as monkeys, dogs and rats, were not always unanimous (42, 69–73) and it is not recommended for use in humans (168).

Calcium hydroxide has been and continues to be a suitable and well-tested pulp dressing which repeatedly gives predictable results in the form of a non-inflamed pulp under a well-formed hard-tissue barrier. It does not appear however, to be beneficial when treating a chronically inflamed pulp (74). Internal dentin resorption and dystrophic calcifications, reported to occur after dressing of the pulp with calcium hydroxide, seem to be related to the presence of an extra-pulpal blood clot or to the damage caused by operative procedures (55, 75–80). Thus, when an extra-pulpal blood clot was not allowed to form, and a gentle amputation technique was used for amputation, these changes were not observed clinically or histologically (35, 37, 64, 81–83).

An improvement in pulp dressing materials occurred when the first of the bioceramic materials was developed by Torabinejad and colleagues at Loma Linda University in California, USA in the early 1990s. The new material – mineral trioxide aggregate (MTA) – has now been tested and applied to numerous dental situations by researchers and clinicians worldwide (169–171).

When first developed, MTA was a gray powder, which has since been modified to a white color for esthetic reasons, without any apparent physical or therapeutic changes (84–87). This change has been made possible by the reduction of ferrite ($Fe_3O_3$) in the making of white MTA (84). MTA has a pH range of 10.2–12.5 that is time related during the first 3 hours of setting time, after which it remains constant. While MTA sets in about 3 hours, its compressive strength continues to increase over a period of 3 weeks (88).

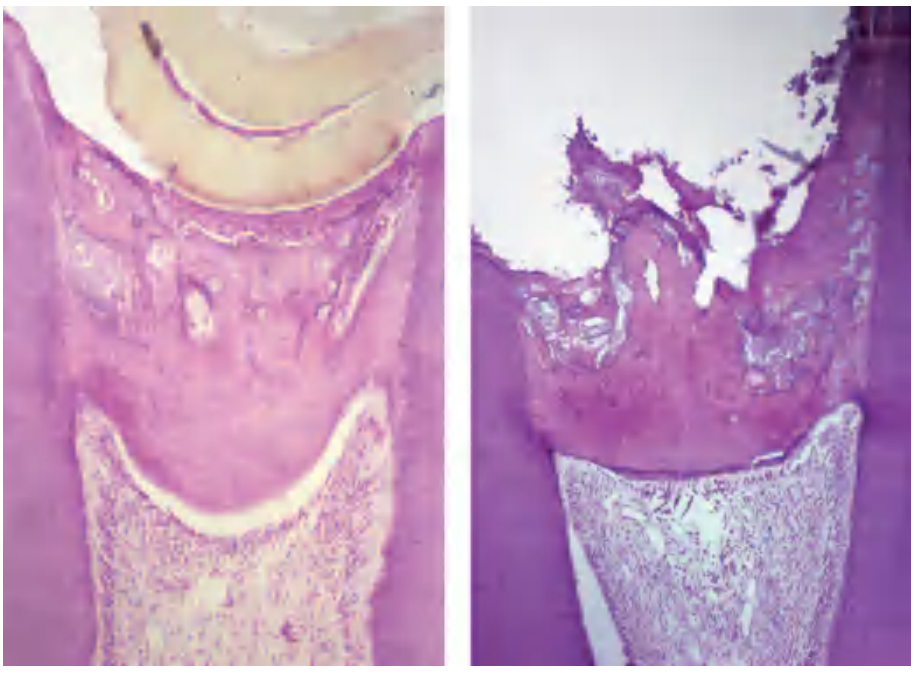


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**Fig. 25.9** Response of monkey incisor to capping with cyanoacrylate or 10 minutes dressing with calcium hydroxide. A. Formation of hard tissue barrier 3 months after the pulp was treated with calcium hydroxide for 10 minutes, then washed away, and the pulpal wound covered with several sterile Teflon discs sealed in place with IRM®. ×50. B. Formation of hard tissue barrier 3 months after the pulp was covered with cyanoacrylate. ×40. Note that hard tissue barriers are not continuous and include vascular tunnels (arrow). Source: Reproduced from (160) with permission.

Mineral trioxide aggregate consists of *calcium silicate* ($CaSiO_4$), *bismuth oxide* ($Bi_2O_3$), *calcium carbonate* ($CaCO_3$), *calcium sulfate* ($CaSO_4$), and *calcium aluminate* ($CaAl_2O_4$). MTA is mixed (3:1, MTA:$H_2O$) with water or other fluids such as saline, to form an amorphous structure of calcium crystals consisting of 33% calcium, 49% phosphate, 2% carbon, 3% chloride, and 6% silica (26). MTA is a hydrophilic material that sets in the presence of any moisture, including blood (89).

One of the major advantages of MTA is its demonstrated biocompatibility and non-mutagenicity. Numerous studies have shown that it is well tolerated by tissue cells both in the pulp and periradicular areas (90–97), and it appears to have a mechanism of action that encourages hard tissue deposition similar to that of calcium hydroxide (85).

Since bacterial microleakage is a major concern with any dental material, it is noteworthy that MTA has been shown to resist bacterial penetration quite favorably compared to other materials (98–100). Recently, Murray et al. (101) demonstrated that the pulp's reparative activity occurs more readily beneath capping materials that prevent bacterial microleakage, a feature favoring the use of MTA.

The reason for MTA's resistance to bacterial penetration is said to be related to its adaptation to adjacent dentin, a tight physical adaptation that includes penetration of MTA into dentinal tubules. The result is a low tendency to microleakage (102–104).

Mineral trioxide aggregate is a biological active material which induces hard tissue formation through mechanisms that are still being investigated (105). Koh et al. (106) noted that it stimulates interleukin formation and provides a substrate for osteoblasts, a finding also noted by Perez et al. (107), though the latter observed that osteoblasts did not survive as long on white MTA as on gray MTA. The bioactivity of MTA may be similar to that of calcium hydroxide; Friedland and Rosado (108, 109) described the results of exposing set MTA to water and found that calcium hydroxide is released from MTA (for at least up to 3 months), which can explain the stimulation of hard tissue formation against the material. Holland et al. (110) found that calcite crystals formed, similarly to that found with calcium hydroxide, when MTA comes in contact with water.

Another recognized advantage of MTA is its prevention of leakage of bacteria into a healing tissue wound, whether it is pulp or periradicular tissue (98–100). It is certainly true that the dental pulp's reparative activity can occur beneath capping materials in the absence of bacterial microleakage and that success is increased with materials that prevent bacterial leakage (101); the same can probably be said for apexification and repair of root fractures.

In spite of the similarity to calcium hydroxide (108, 109), MTA does not appear to have a detrimental effect on dentin (111), a problem noted with calcium hydroxide (112–115). This makes MTA especially well-suited for treatment involving pulp necrosis in immature, developing teeth (e.g. teeth needing apexification and teeth with root fractures in which the coronal pulp tissue deteriorates and becomes infected).

The bioactive property of MTA has resulted in superior (when compared to calcium hydroxide and other materials) dentin bridges after pulp capping and pulpotomies (105, 116–124) and apexifications (125, 126), although Fransson et al. (166) in a recent in a recent systematic review found no scientific evidence that MTA promoted hard tissue formation more frequently than calcium hydroxide. An advantage in the use of MTA is that it provides an excellent protection against bacterial leakage (101) and possesses a biocompatible quality that allows tissue repair (104, 106, 116, 125–129) along with providing stimulation for hard tissue deposition (110).

One of the concerns that have been raised with the use of MTA is the apparent staining that appears subsequently. While such stained crowns can be bleached successfully after the dentin bridge has developed (172), newer bioceramics appear to be less prone to staining (173).

Since the introduction of MTA, many new materials - collectively referred to as bioceramics – have been added to the list with some improvements in handling characteristics (130). One can expect continued developments in this area of dental materials. For the sake of convenience, the term MTA will be used throughout the chapter when referring to bioceramic materials.

## Pulp capping

Capping with calcium hydroxide is indicated when a small exposure of the pulp can be treated shortly after injury which, according to experimental studies, seems to mean within 24 hours (131–133).

It is thought that, in small exposures treated soon after injury, the mechanical damage and inflammation in the pulp cannot be deeper than the necrotizing effect of calcium hydroxide. Thus, the effect is on healthy pulp tissue, and bacteria on the dentin fracture and the wound surfaces are eliminated by the action of calcium hydroxide. Accordingly, the primary contamination of the pulp may not be critical for healing. However, pulp healing may be threatened by later contamination due to microleakage of defective restorations, since all calcium hydroxide compounds gradually lose their antibacterial property. Furthermore, the hard tissue barriers may include structural defects such as 'tunnel' formations that allow subsequent bacterial contamination of the underlying pulp tissue (65, 69, 134–139, 164) (Fig. 25.9).

### *Treatment procedure*

The fracture surface and pulp wound can be disinfected with either sodium hypochlorite or chlorhexidine. Cover the exposed pulp, after bleeding has stopped, with a soft- or a hard-setting calcium hydroxide. If the pulp is covered with a soft calcium hydroxide compound, it should be protected with glass ionomer cement or a hard-setting calcium hydroxide liner before the crown is restored (Fig. 25.10). If the definitive restoration of the crown must be postponed, a temporary crown restoration should be placed with a material that does not allow microleakage, e.g. zinc oxide eugenol or polycarboxylate cement (140, 141).

**Fig. 25.10** Treatment of a complicated crown fracture with pulp capping. A. Fractured incisor with a small pulp exposure 4 hours after injury. Note the good vascularity of the exposed pulp. B and C. After isolation with a dental dam, the exposed pulp is covered with a soft calcium hydroxide compound (Calasept®) which together with the remaining dentin is covered with hard-setting calcium hydroxide cement. D. The tooth is restored with a composite.

If MTA is chosen for pulp capping it is best to do a small pulpotomy (see below) to allow the material to be secured; it is difficult to keep it in place if it placed on a flat surface.

## Pulpotomy

Pulpotomy consists of removing damaged and inflamed tissue to the level of clinically healthy pulp tissue, followed by placement of a dressing, such calcium hydroxide. Depending on the size of the exposure and time elapsed since the injury, different levels of pulp amputation have been recommended, i.e. partial or deep pulpotomy (36, 142). It has been shown, however, that neither exposure size nor time interval between injury and treatment are critical for healing when only superficial layers of the pulp are removed (36, 37, 81). Thus, in teeth with vital and/or hyperplasic pulp tissue at the exposure site, only superficial layers of the pulp and surrounding dentin need to be removed; i.e. a shallow pulpotomy can be performed in immature as well as mature teeth.

### *Treatment procedure using calcium hydroxide*

As pointed out above, a diamond bur in the high-speed handpiece is best suited for surgical removal of pulp tissue (Fig. 25.11). Effective cooling is essential. To avoid injury to the pulp due to insufficient cooling, the tooth and cutting instrument should be flushed continuously with water or saline by means of a syringe or the turbine spray. Furthermore, cutting should be performed intermittently for brief periods and without unnecessary pressure.

The level of amputation should be about 2 mm below the exposure site. This level is deep enough to remove inflamed tissue and provide an adequate cavity for both the dressing and the sealing material. The pulp wound is then rinsed with saline until bleeding has ceased, or a cotton pellet soaked in sodium hypochlorite can be placed in the wound until bleeding stops (174). The wound surface is covered with calcium hydroxide and adapted with cotton pellets using light pressure, whereby the water from the paste is also removed. The surplus of calcium hydroxide is then easily removed with an excavator. The coronal cavity is closed with a tight-sealing material, e.g. glass ionomer cement.

The advantage of a shallow pulpotomy lies in the minimal injury to the pulp and undisturbed physiologic apposition of dentin, especially in the critical cervical area of the tooth. The limited loss of crown substance offers continued opportunity for sensitivity testing and, in most cases, a post in the root canal will not be required for crown restoration. Compared with pulp capping, this procedure provides better wound control and, by sealing off the cavity with a material which does not allow microleakage, provides effective protection for the pulp. Most probably, this is one of the reasons for the high rate of healing reported after this type of treatment (36, 37, 73, 81, 143) (Figs 25.12 and 25.13). It may also be related to the lesser incidence of severely inflamed pulps in traumatically exposed pulps compared to pulps subjected to carious insults (166). The frequency of failures is low and, judged from clinical observations, depends on factors, such as misjudged pulp diagnosis, overheating pulp at the time of treatment and poor restoration of the crown, which allows microleakage and penetration of the bacteria into the pulp (Fig. 25.14). It has been suggested that, after capping of pulp exposures close to the cervical area of anterior teeth, formation of hard tissue may compromise the blood supply to the coronal part of the pulp tissue, causing its degeneration (49). However, this phenomenon appears to be rare (144). Thus, in a comprehensive clinical study, no adverse effects could be

**Fig. 25.11 Treatment of a complicated crown fracture with partial pulpotomy**
A complicated crown fracture of an immature incisor 2 days after injury.

**Clinical condition**
After the crown fragments were removed a large pulp exposure is found.

**Isolation with a dental dam**
After administration of a local anesthesia, the tooth is isolated with a dental dam and washed with saline. A diamond bur is chosen for the pulpotomy.

**Pulpotomy**
The pulpotomy is carried out to a depth of 1.5–2.0 mm. The cutting of the pulp and surrounding dentin is performed using a high-speed turbine. This is done intermittently, with brief cutting periods and continuous gushing with the water spray from the turbine, eventually supplemented with extra saline from a syringe.

**Cavity preparation**
The cavity should be boxlike, with a slight undercut in dentin.

**Applying the dressing material**
Hemostasis is awaited, and usually takes place within a couple of minutes. Otherwise a slight pressure can be applied with a cotton pellet soaked in saline or an anesthetic solution with vasoconstrictor or soaked in sodium hypochlorite. After complete arrest of bleeding, calcium hydroxide paste (e.g. Calasept©) is placed over the pulpal wound.

**Compressing the dressing material**
The material is compressed slightly with a dry cotton pellet to adapt the material to the wound surface. After surplus dressing material is removed, the amputation cavity is sealed with zinc-eugenol cement or glass ionomer cement. In cases where the tooth is immediately restored, the cavity is sealed with a hard-setting calcium hydroxide compound, or zinc phosphate cement can be used.

**Restoration**
The tooth has been restored during the same appointment with composite resin and a dentin bonding agent.

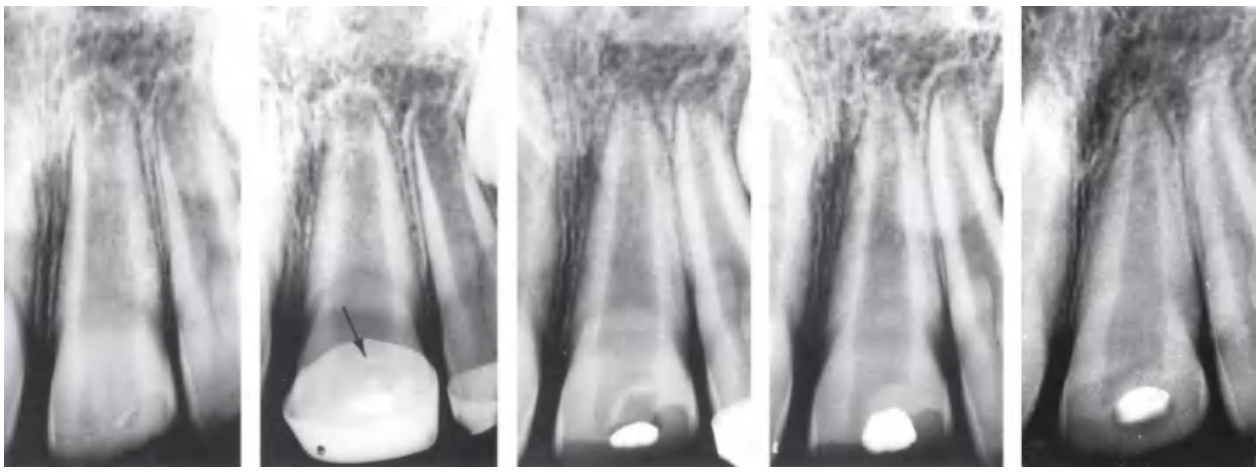



**Fig. 25.12** Radiographic demonstration of healing after partial pulpotomy in an incisor with immature root. The tooth was treated 3 hours after injury. Note initial formation of hard tissue after 3 weeks (arrow) and completed barrier after 6 months. A control after 5 years shows healing and completed root development.

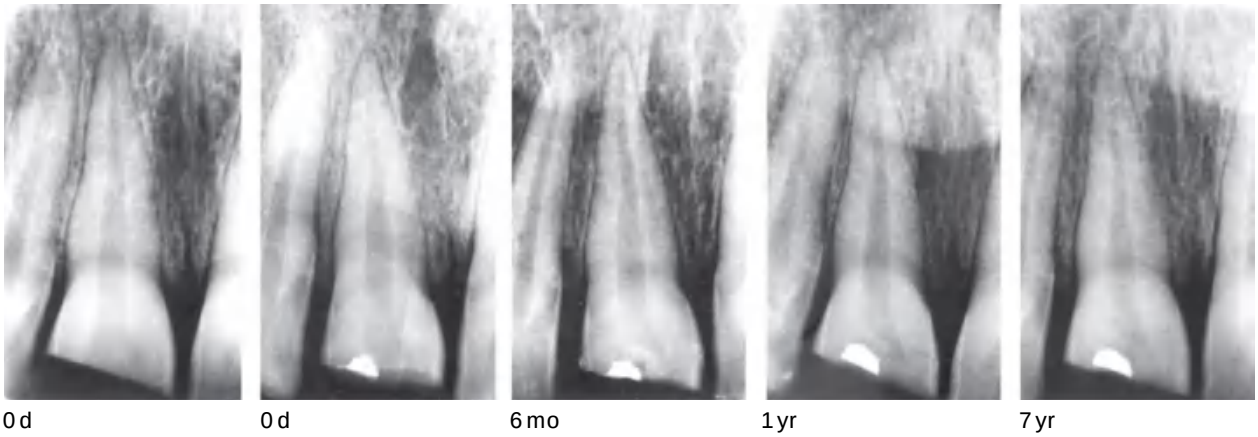



**Fig. 25.13** Radiographic demonstration of healing after partial pulpotomy in an incisor with completed root formation. The tooth was treated 12 hours after injury. Formation of a hard tissue barrier is present after 6 months and healing is evident 7 years after treatment.

observed after partial pulpotomy treatment of proximal exposures in crown-root fractured teeth (37) (Fig. 25.15).

When necrotic tissue or obviously impaired vascularity is present at the exposure site of immature teeth, the pulp should be amputated to a level at which fresh bleeding tissue is found, i.e. a cervical or deep pulpotomy should be performed (Fig. 25.16) (174). Due to problems with adequate cooling of the diamond at high speed at that level, a round carbide bur at low speed should be used. In mature teeth, a pulpectomy is usually the treatment of choice.

### *Treatment procedure using bioceramics (MTA)*

The bioceramic materials, such as mineral trioxide aggregate (MTA), offer many advantages for use in pulpotomies. They are biocompatible (104, 106, 116, 125–129, 170), provide excellent resistance to microleakage (98–100, 170), allow opportunity for dentin bridging at the site of pulp exposure (105, 116–118, 120, 121, 166) (Fig. 25.17), and appear to be associated with very positive clinical outcomes (119, 122, 123, 176) (Fig. 25.18). And since they don't deteriorate over time, it is not necessary to re-enter the site of material placement at a later time to replace the material.

The action of MTA on exposed pulp tissue is similar to that of calcium hydroxide (105). A thin layer of necrotic pulp tissue superficial to the bridge suggests a similar action (105). Initially a superficial zone of extracellular matrix forms, which is followed by hard tissue depositions, under which formative cells produce reparative dentin as a sign that the pulp is back to normal function (121). The main difference between MTA and calcium hydroxide may be that MTA provides a good protective barrier against microleakage (98–100) and does not break down requiring replacement as is the case with calcium hydroxide (36, 145). If microleakage occurs in the case of calcium hydroxide, it allows bacteria to reach the dentin bridge which usually has numerous tunnel defects (131, 164), a factor not of significance when MTA is used (121).

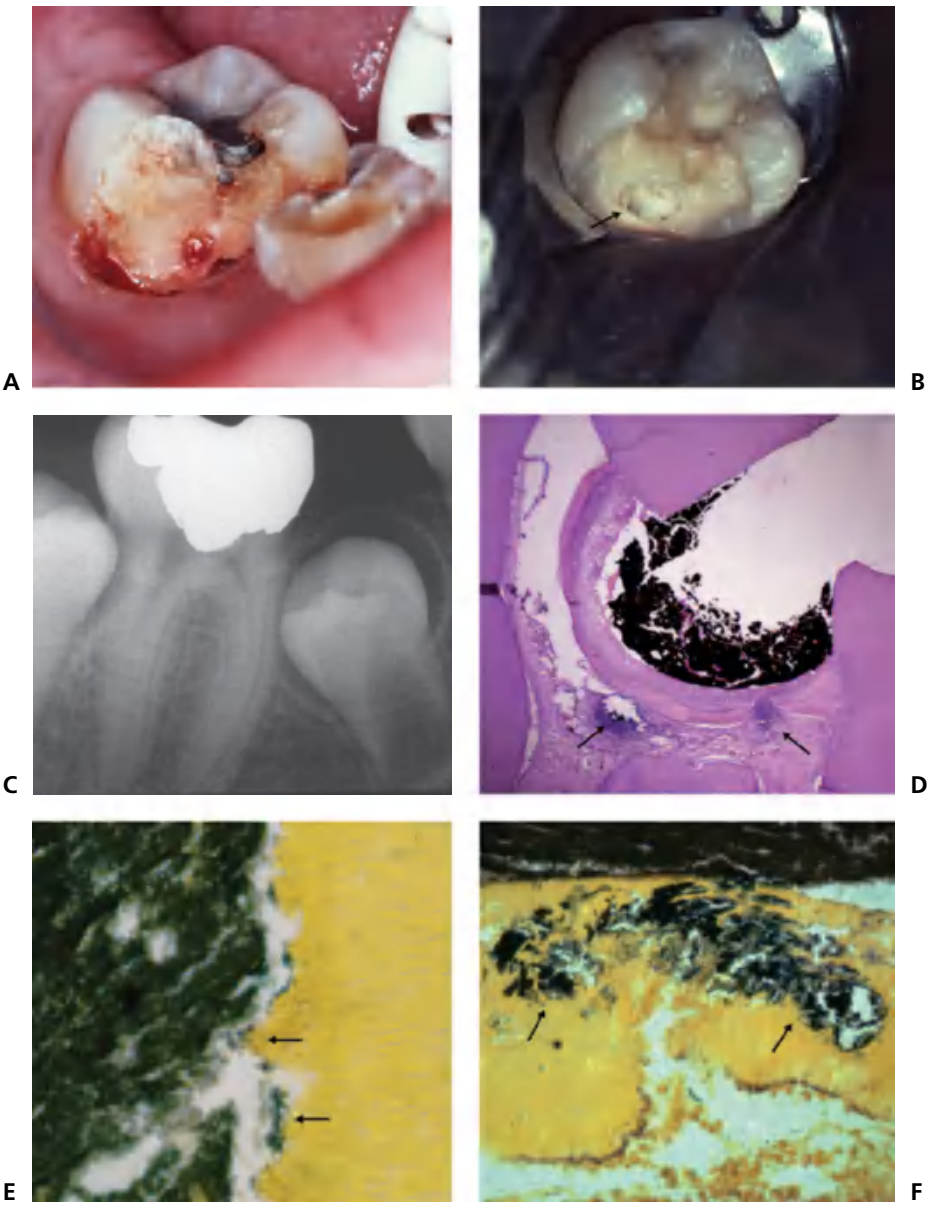



**Fig. 25.14** Penetration of bacteria to the pulp via marginal microleakage. A. The pulp exposed by a crown-root fracture was treated by partial pulpotomy in a first mandibular permanent molar. B. Three months later, a continuous hard-tissue barrier was covered with a hard-setting calcium hydroxide compound and the crown restored with amalgam. C. Four months later, the tooth showed no clinical or radiographic symptoms but was extracted for orthodontic reasons; note extension of amalgam restoration below the marginal level. D. Histologic sections show a well formed hard-tissue barrier, covered with dark stained lining compound and below it, two small abscesses in the pulp (arrows); the empty space to the left is an artefact. ×10. E and F. Stain for bacteria reveals dark-bluish bacteria on dentinal walls and in the gap between dentin and the dressing compound as well as in the hard tissue barrier. ×60.

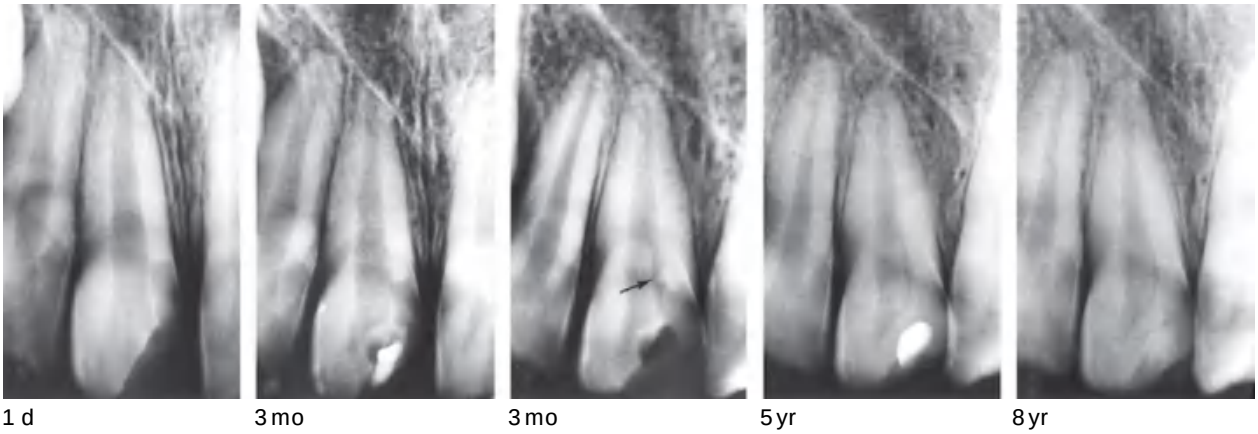



**Fig. 25.15** A crown-root fractured incisor with a proximal exposure of the pulp, treated 24 hours after injury with a partial pulpotomy. A loose fragment was removed 3 months later (arrow). There is formation of a hard tissue barrier but no further constriction of the coronal pulp.

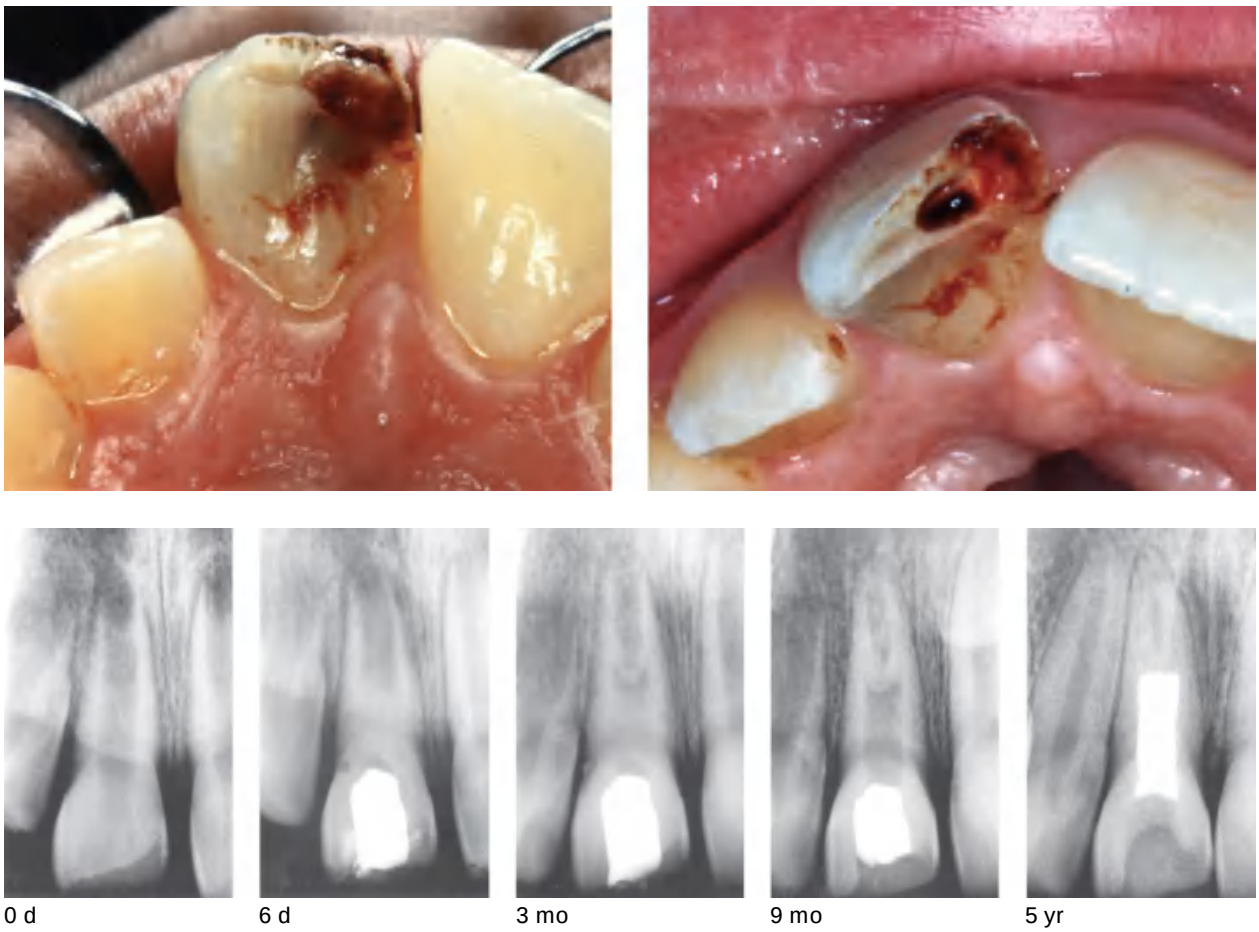



**Fig. 25.16** Deep pulpotomy in an incisor with crown fracture and impaired pulpal vascularity. Vital and bleeding pulp tissue was found in the middle of the root canal. Three and 9 months after a deep pulpotomy, formation of a hard-tissue barrier can be seen deep in the root canal. A 5-year control of the subsequent root canal filling with gutta-percha shows completed root development.

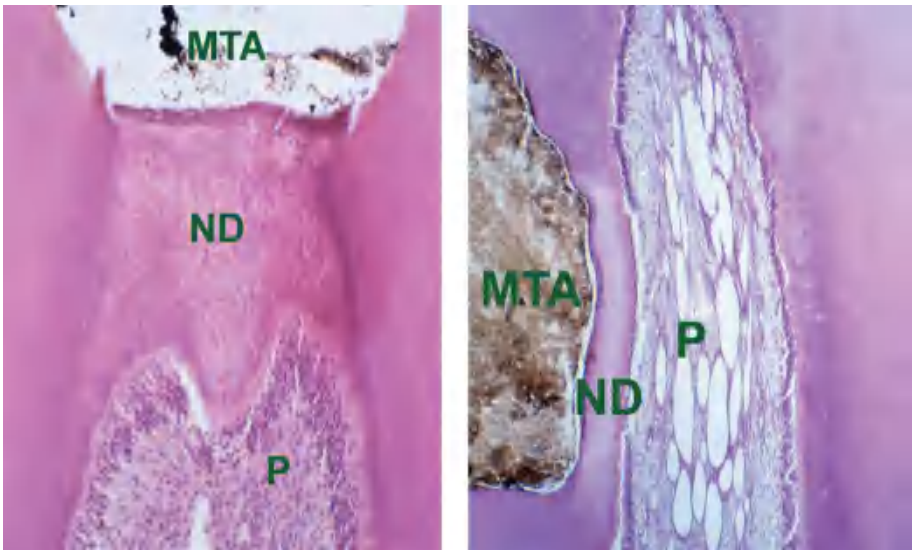



**Fig. 25.17** Histologic response to shallow pulpotomy and pulp capping. A. Shallow pulpotomy on a dog's tooth shows healthy, normal appearing new dentin (MTA – position of MTA during treatment; ND – new dentin formed under MTA; P – pulp). B. Pulp capping on a monkey's tooth. Bridge formation at site of mechanical pulp exposure.

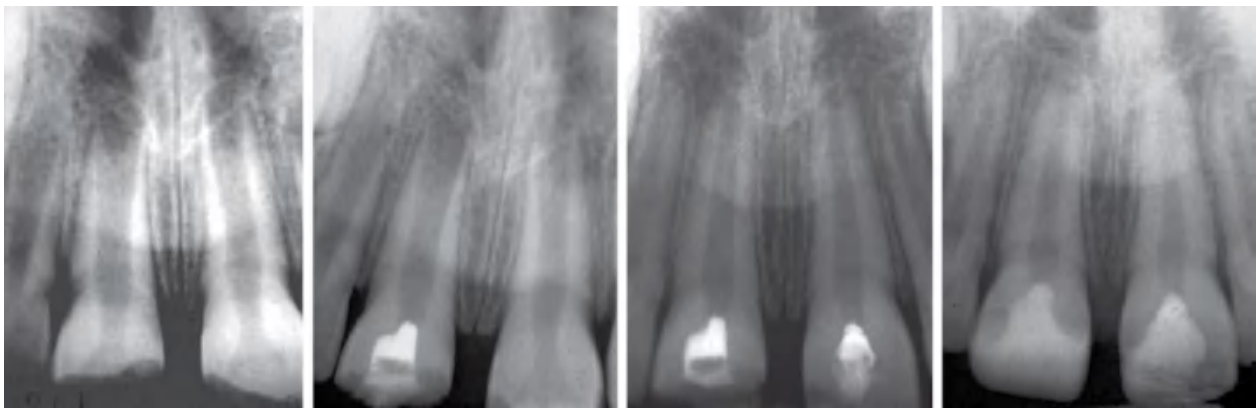



**Fig. 25.18** Healing after shallow pulpotomy. A. Two central incisors fractured in a boy age 8. B. First the right central incisor received an MTA pulpotomy, following which the left central incisor was also treated in the same manner. C. The teeth one year postoperatively. D. The MTA was changed from gray to white for cosmetic reasons after 3 years. Note continued root development.

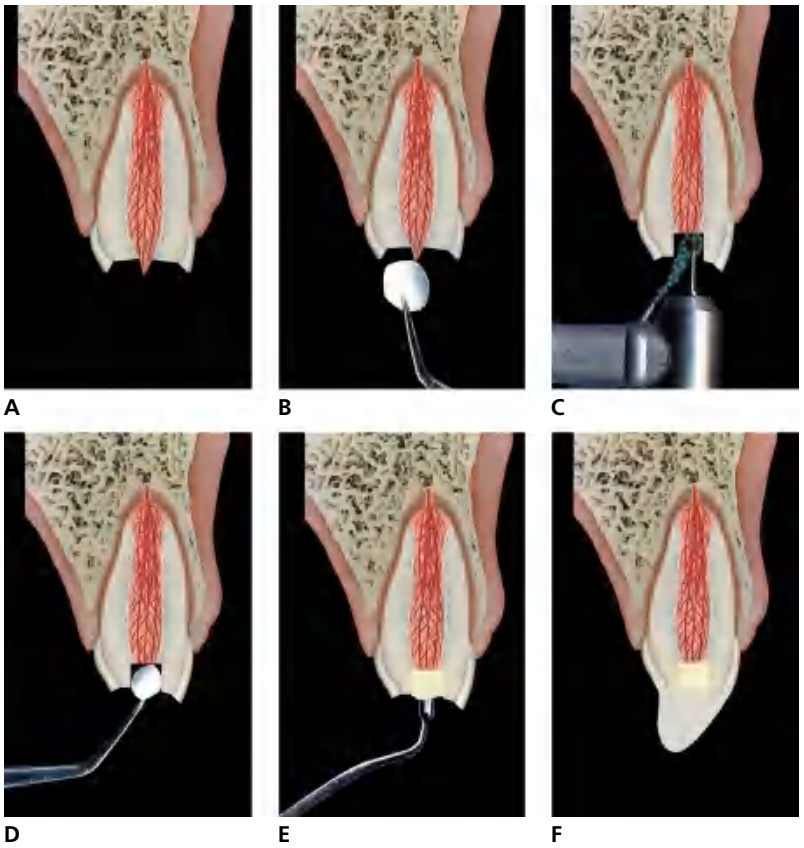



**Fig. 25.19 Shallow (partial) pulpotomy using MTA.**

A. Isolate the tooth with a dental dam after administering local anesthesia.
B. Disinfect the exposed dentin and pulp with either sodium hypochlorite or chlorhexidine.
C. Remove pulp and surrounding dentin to a depth of ~2 mm from the site of exposure, using a diamond bur with irrigation.
D. Place a cotton pellet moistened with sodium hypochlorite (NaOCl) on the pulpal wound to stop the bleeding; 2–3 minutes is usually sufficient time.
E. Mix the MTA with water and place it directly on to the pulp wound in the prepared cavity, filling the entire cavity. Remove excess moisture with a cotton pellet.
F. Restoration of the fractured crown can be done after the MTA has cured, or immediately by covering the fresh MTA with a glass ionomer cement to prevent contact of the MTA with the acid used for etching of the surrounding tooth structure.

Finally, several studies have shown MTA to be superior to other alternative capping agents (e.g. resins) (123, 124). While there are promising possibilities with the use of resins for protecting the exposed pulp, the available evidence raises questions about their safety (146–149, 168).

The technique for using MTA is in many ways similar to the shallow pulpotomies in which calcium hydroxide is used. These are the recommended steps for MTA pulpotomies in teeth with reversible pulpitis (Fig. 25.19):

- After anesthetizing the tooth, isolate it with a dental dam and disinfect the operative site. Sodium hypochlorite or chlorhexidine are excellent agents for disinfection.
- Using a round diamond (about the size of a #4 round bur) in the high-speed handpiece and with a water coolant spray, gradually remove pulp tissue from the site of the exposure to a depth of about 2 mm into the pulp proper. Use a cotton pellet soaked in sodium hypochlorite to stop the bleeding; it usually takes less than 2–3 minutes for the bleeding to stop (175).

- Mix the MTA to manufacturer's recommendations (3:1, MTA:$H_2O$); it will have the consistency of wet sand. Place the material directly against the pulp tissue and gently tap it in place using a cotton pellet. The MTA can be covered before it has cured with glass ionomer cement following which the crown can be restored with bonded composite resin, or the broken crown fragment can be re-bonded to the remaining tooth structure.

### Clinical evaluation of pulp healing

Histological evaluation is the gold standard in assessing healing outcomes following pulp treatment and in research projects that is possible. Clinical evaluation can however be made using the following criteria (150):

(1) No clinical symptoms
(2) No radiographically demonstrable intra-radicular or peri-radicular pathological changes
(3) Continued root development in immature teeth
(4) Radiographically observed (and eventually clinically verified) continuous hard tissue barrier
(5) Positive sensitivity to electrical stimulation
(6) Follow-up for at least 3 years.

These criteria apply both to pulp capping and pulpotomy, with the exception of sensitivity testing which usually is not possible if the amputation site is in the root canal. In the pulps of teeth treated with shallow pulpotomy and judged to be healed according to these criteria, no or only insignificant pathologic changes have been found in the histologically examined pulps (82, 83).

A hard tissue bridge that appears solid in radiographs may have numerous 'tunnels' when examined histologically. The pulps however, in such teeth frequently are non-inflamed (56, 82, 151). It is important to consider that a continuous hard tissue barrier and positive sensitivity do not exclude chronic pulp inflammation, which may persist in an asymptomatic tooth (Fig. 25.14). Long term monitoring is recommended since it may take a year or more to identify treatment failures. The teeth that were judged as healed at the 3-year control remained healed 10–15 years thereafter, indicating that a 3-year follow-up should be considered adequate (37).

Pulp canal obliteration and pulp necrosis are rather frequent long-term complications after a cervical or deep pulpotomy. This procedure is therefore regarded as a temporary treatment to be followed by pulpectomy and root canal filling once root formation is complete (68).

### Prognosis

The frequency of healing after pulp capping, partial or cervical pulpotomy treatments varies from 72–96% (36, 37, 81, 143, 151–158, 176). It is also apparent that teeth with mechanical or traumatic pulp exposures have higher success rates than teeth with cariously exposed pulps (166). The reported frequency of success after pulpectomy and filling of the root canal with various materials varies from 80–96% (159–163).

## Root fractures

### Healing and pathosis of root fractures

When a root fracture occurs, there may or may not be concurrent displacement (luxation) of the fragment of tooth coronal to the fracture line. If the coronal fragment has been displaced, then the healing response and long-term prognosis will be affected more than in a tooth where there has not been any displacement. Early repositioning and stabilization of the displaced fragment are therefore essential to provide the optimum conditions for healing.

As the forces that fractured the root will be concentrated at the fracture site, there is usually little, if any, effect on the apical fragment of teeth with transverse root fractures. Hence the apical fragment is not displaced, and the pulp's blood supply is not normally severed or even reduced at the apical foramen. However, the pulp blood vessels may be affected at the level of the root fracture, particularly if the coronal fragment has been displaced. The amount and direction of displacement of the coronal fragment will determine whether the pulp is unaffected, stretched, partially severed or completely severed. In increasing order of severity, the coronal fragment may be laterally luxated, extruded or avulsed with the severity of pulp injury similarly increasing with each of these concurrent injuries.

Fortunately, most teeth with root fractures survive and can remain functional for many years without the need for treatment apart from regular follow-up (Fig. 25.20). Pulp necrosis and infection with subsequent peri-radicular inflammation is not common following root fractures with only about 27% of cases developing these problems (see Chapter 15). This is likely related to most root fractures having relatively minor displacement of the coronal fragment. When pulp necrosis does occur, it is usually only in the coronal fragment and the resultant peri-radicular inflammation occurs at the fracture line (that is, between the two fragments and in the tissues surrounding the fracture) rather than periapically (Figs 25.21–25.26).

As the apical blood supply is not usually affected by a root fracture, the pulp in the apical fragment remains clinically normal (177–187). It is rare for the pulp in this fragment to necrose and become infected although this is a possibility if the necrotic and infected coronal pulp is not treated in a timely manner. However, in the vast majority of cases, the pulp in the apical fragment survives and over time it will show radiographic signs of pulp canal obliteration (Figs 25.20, 25.22, 25.24 and 25.27). There will also be a normal periodontal ligament space and lamina dura, indicating clinically normal periapical tissues. Hence, in these cases, the pulp in the apical fragment does not require any treatment even if the pulp in the coronal fragment has become necrotic and infected with peri-radicular inflammation adjacent to the fracture.

Pulp necrosis of the coronal fragment does not usually occur immediately, or at least will not be evident immediately. Since pulp necrosis is not common, root canal treatment

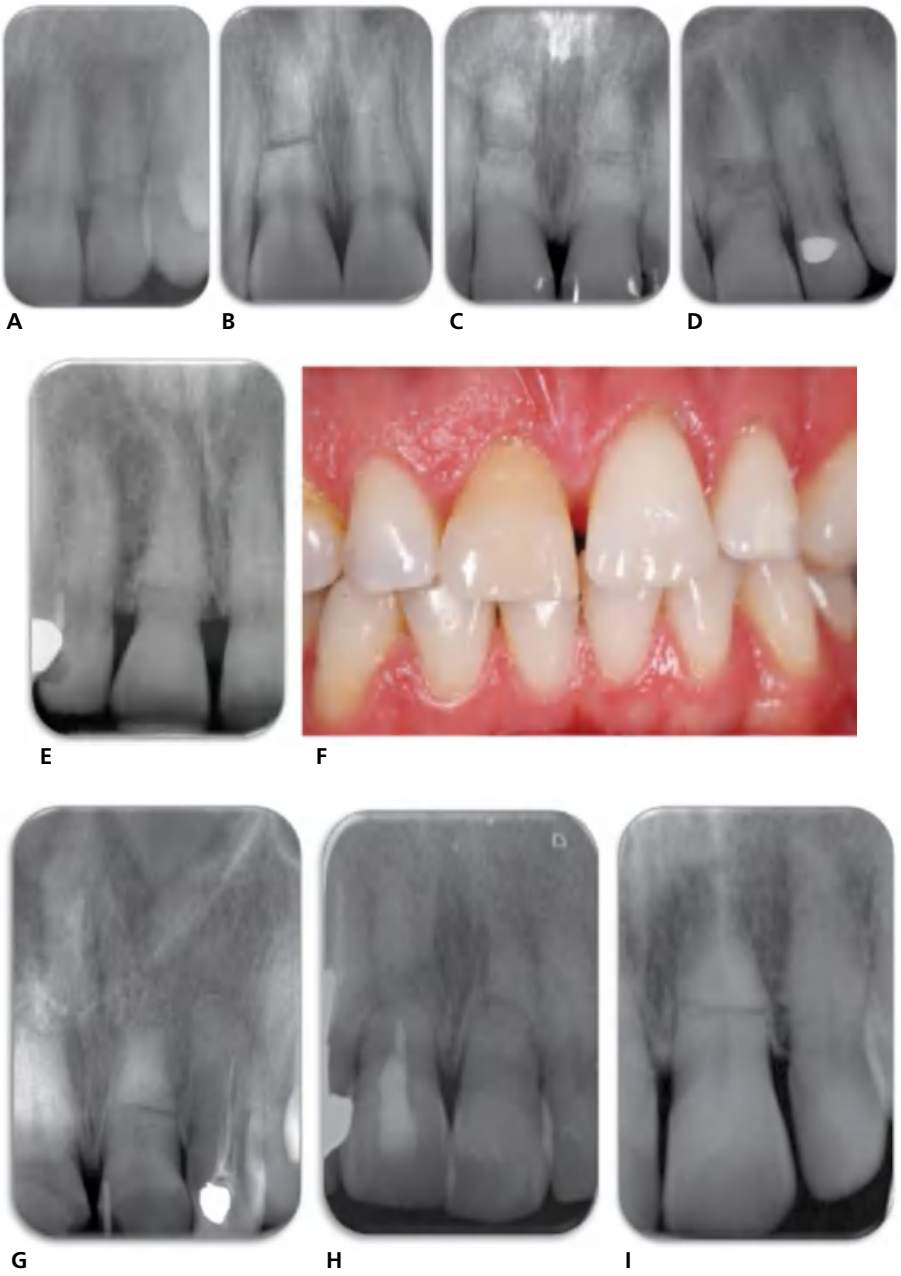



**Fig. 25.20** Examples of long-term survival of various teeth with root fractures due to trauma. The fractures occurred at varying levels of the root. The histories provided by the patients indicate that no treatment was provided other than stabilization and follow-up. A. 4 years after injury. B. 6 years after injury. C. 12 years after injury. D. 15 years after injury. E and F. 25 years after injury. Note some slight yellow discoloration of the crown as a result of pulp canal obliteration. G. 40 years after injury. H. 45 years after injury. The right central incisor had root canal treatment 25 years after the injury following further trauma. The left central incisor has required no treatment. I. 50 years after injury. Source: C, D, G, I courtesy of Dr. Tony Sandler, Perth, Western Australia, Australia.

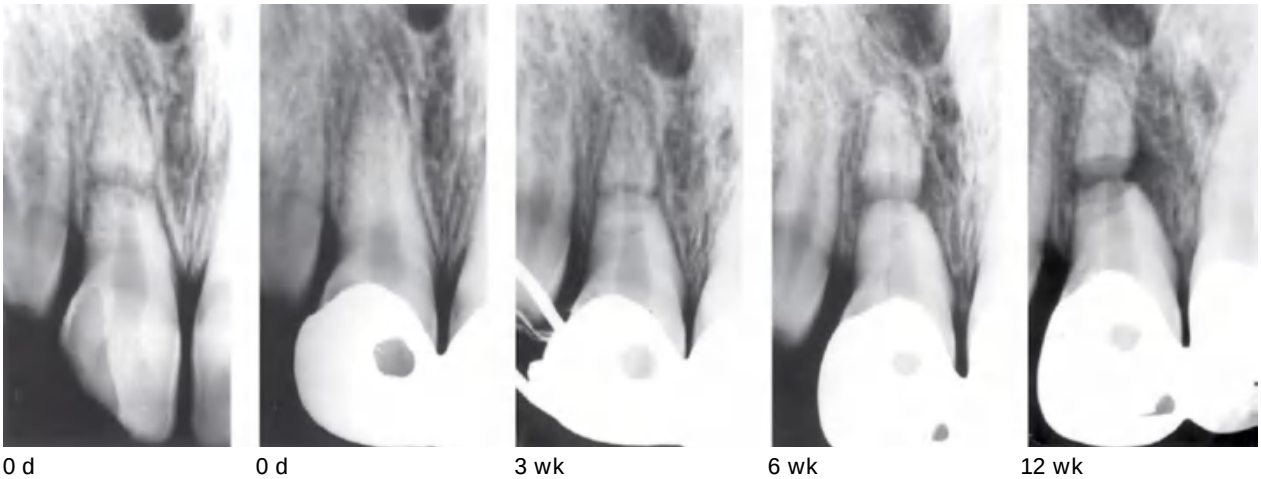



**Fig. 25.21** Radiographic signs of pulp necrosis and infection of the coronal fragment after root fracture. Note progressive widening of the space between fragments and the appearance of radiolucency in the adjacent alveolar bone.

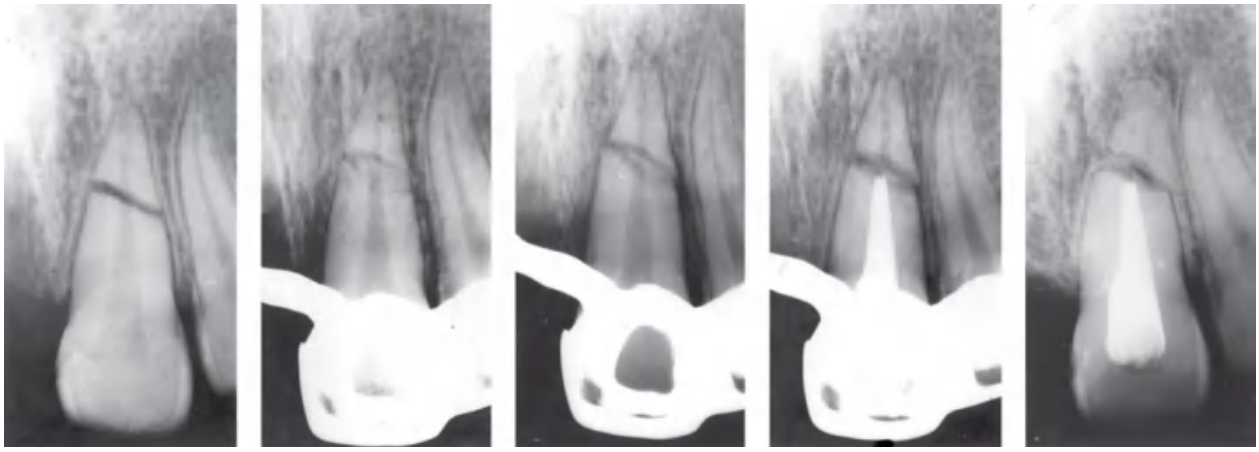



**Fig. 25.22** Root canal treatment of the coronal fragment of a root fractured incisor with gutta-percha and cement. The tooth is shown before and after repositioning of the coronal fragment at the time of injury. After 6 weeks, a slightly widened space between the fragments and a radiolucency in the adjacent alveolar bone indicate pulp necrosis and infection of the coronal fragment. The tapered root canal in the fragment permitted an adequate filling with gutta-percha and cement.

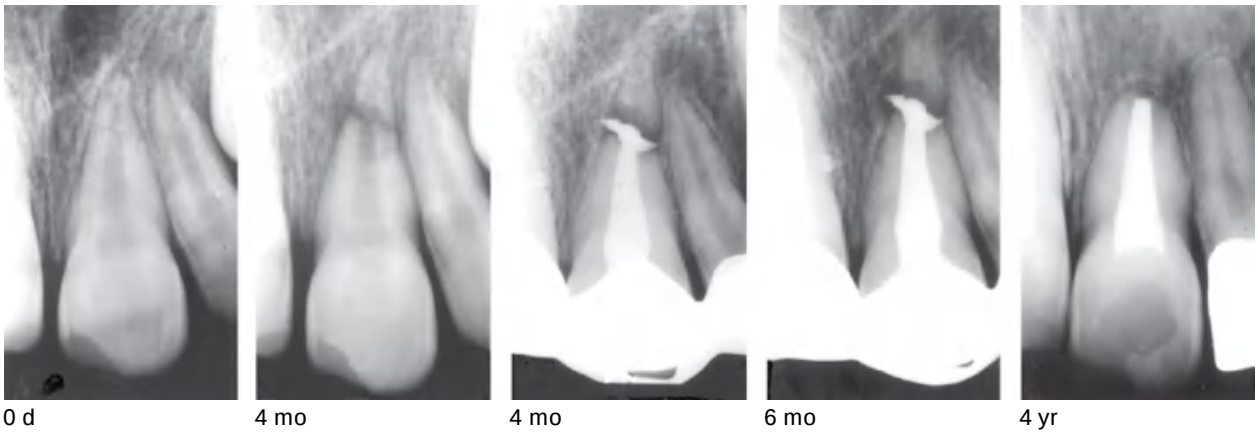



**Fig. 25.23** Difficulties in root canal filling of the coronal fragment after root fracture. After 4 months there is a widening of the space between the fragments and a radiolucency adjacent to the fracture. After antibacterial treatment, the coronal root canal was filled but gutta-percha was extruded into the fracture site. After six months, the space between the fragments as well as the adjacent radiolucency increased in size. Subsequently, the apical fragment was surgically removed and at the 4-year review appointment, periapical healing is evident.

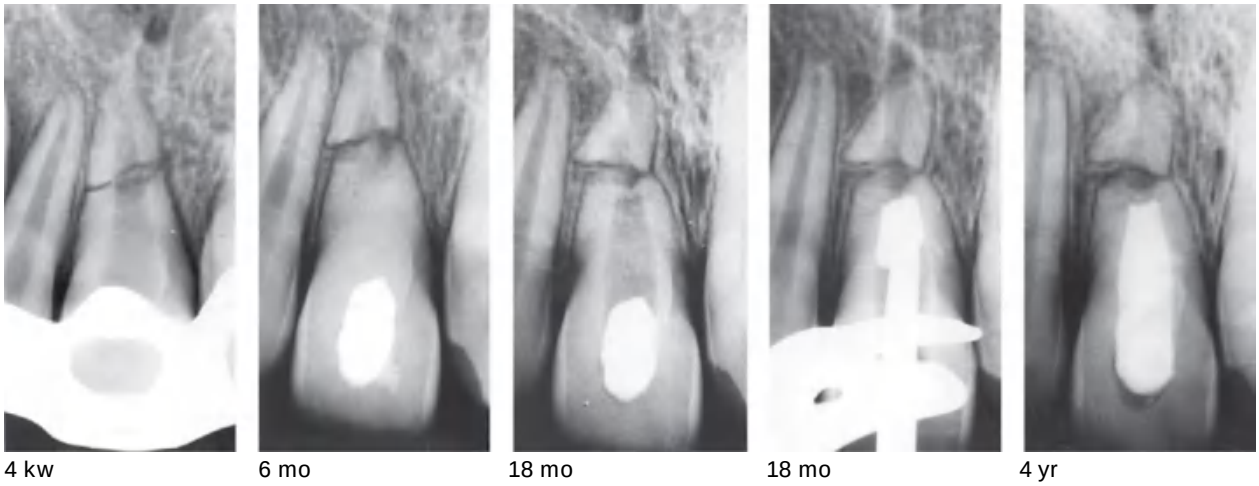



**Fig. 25.24** Treatment of the coronal fragment of a root fractured incisor with calcium hydroxide as there was a wide root canal. After 18 months, periradicular healing has taken place and a hard tissue barrier has formed apically in the coronal fragment, against which an adequate root filling with gutta-percha and cement could be placed.

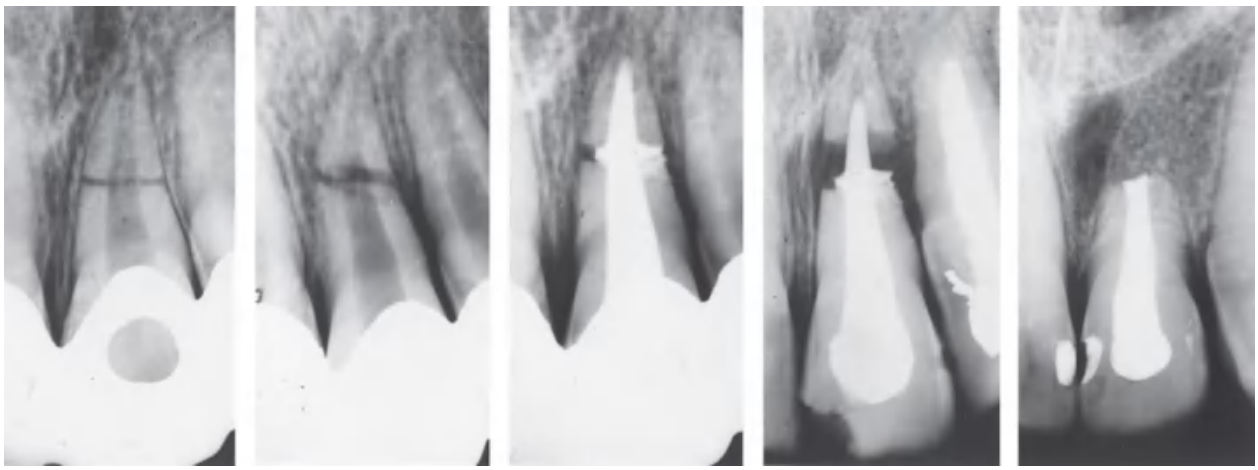



**Fig. 25.25** Root canal treatment of both fragments of a root fractured incisor. Note the widening of the space between fragments and the adjacent radiolucency 4 months after injury. The root canal in both fragments was filled with gutta-percha and cement, some of which was extruded into the space between the two fragments. After 8 months, the space between the fragments and adjacent radiolucency increased. The apical fragment appears to have also been displaced. Therefore, the apical fragment was surgically removed. Peri-radicular healing can be seen 4 years later.

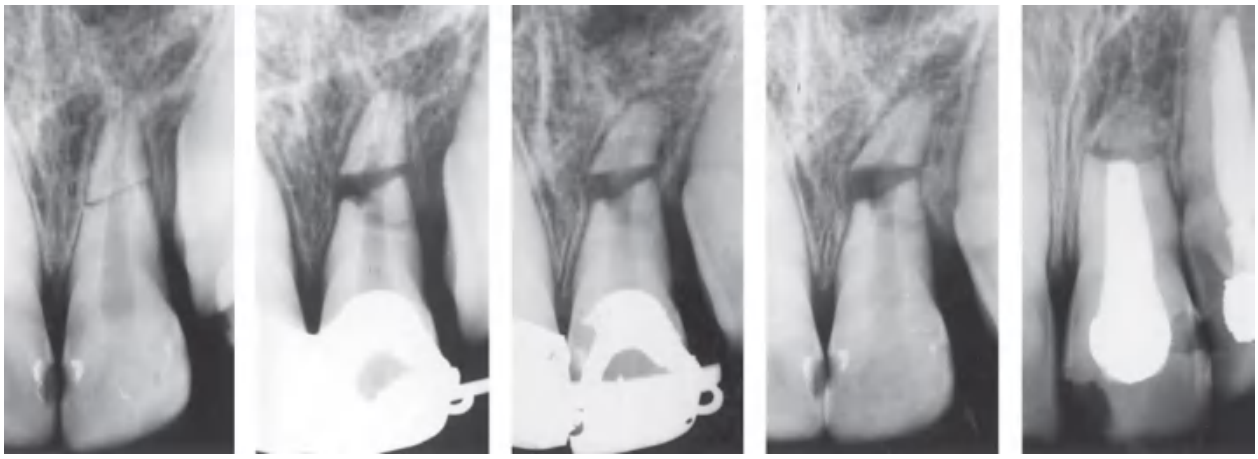



**Fig. 25.26** Root canal treatment of the coronal fragment and simultaneous surgical removal of the apical fragment. Displacement of the apical fragment made root canal treatment of both fragments impossible.

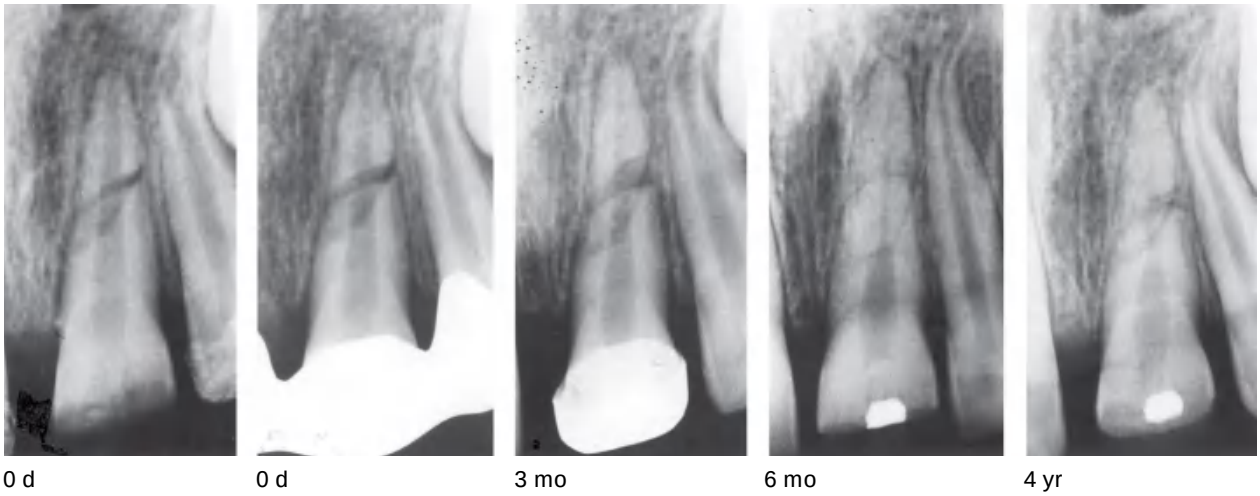



**Fig. 25.27** Treatment of an incisor with concurrent complicated crown and root fractures. A partial pulpotomy was performed and the tooth was splinted. At the final review appointment after 4 years, a hard tissue barrier is seen in the coronal fragment as well as connective tissue healing of the root fracture.

should not be instigated as part of the emergency management of root-fractured teeth. Instead, these teeth should be stabilized and then reviewed regularly to assess whether the pulp recovers or necroses. If pulp necrosis occurs, it is usually not evident for about three months after the injury (180, 181) which is when a radiolucency may be seen in the peri-radicular tissues adjacent to the fracture line.

## Diagnosis of pulp necrosis and infection in root-fractured teeth

The diagnosis of pulp necrosis and infection in the coronal fragment is based on the usual symptoms and signs of this same condition in any tooth. A thorough examination must be carried out and this must include pulp sensibility tests, percussion, palpation, mobility, periodontal probing, and periapical radiographs. In most cases, more than one radiograph is required in order to assess the extent and position of the root fracture unless it has been identified previously and the patient has been attending for regular reviews of the injury.

Some patients may complain of discomfort associated with a root-fractured tooth and some teeth may show increased mobility of the coronal fragment as a result of peri-radicular inflammation and bone loss around the coronal fragment. These teeth may initially have been stabilized with no, or minimal, mobility and no obvious bone loss in the early healing phases, but then these problems have developed as the pulp has necrosed and become infected with peri-radicular inflammation occurring.

Clinically, some color change may be evident in the crown of the tooth although this is not always the case. The tooth will not respond to pulp sensibility tests such as a cold or electric test. There may be some tenderness to percussion or the tooth may feel different on percussion, depending on whether the patient presents with acute of chronic inflammation of the peri-radicular tissues at the fracture line.

Radiographically, in the early stages of the infection and inflammatory processes, there will be a widened periodontal ligament space between the apical and coronal fragments (Figs 25.21–25.26). As the inflammatory response progresses in the peri-radicular tissues, there will be bone resorption resulting in a radiolucent area surrounding the apical end of the coronal fragment and it usually appears to be spreading to the mesial and distal aspects of the fracture line.

Some patients may complain of a draining sinus on the nearby mucosa, indicating that a chronic abscess has developed. If a draining sinus is present, a gutta percha point should be placed in the sinus tract to trace its origins and to confirm that it is originating from the radiolucency associated with the coronal fragment of the root-fractured tooth and not from another tooth.

Radiographs should be carefully assessed to distinguish the development of radiolucencies and resorption indicating a disease process from that of the remodeling processes that occur at the angles of either or both the apical and coronal fragments (Figs 25.20 and 25.27). The remodeling essentially results in rounding of the sharp corners and involves some loss of tooth substance (i.e. surface resorption) before the repair process with new cementum. Some cases may appear to have internal surface resorption or even a tunneling resorptive process into the root canal of the coronal fragment – but these are a temporary phenomenon which may be the tooth's natural way of opening the canal space to allow for pulp revascularization of the coronal fragment. These conditions do not require root canal treatment (181, 182) (see Chapter 15).

In contrast, any evidence of external inflammatory resorption of the tooth root is a very definite indication that the pulp has become necrotic and infected (188). These cases require immediate commencement of root canal treatment. External inflammatory resorption is more likely to occur in root-fractured teeth where the coronal fragment has also been luxated as this concurrent injury may have caused damage to the cementum of that fragment, plus the pulp is less likely to survive and could become infected – hence, the ideal conditions for external inflammatory resorption (i.e. an infected root canal system with cementum damage) are present. In such cases, where external inflammatory resorption is very likely to occur, it is wise to consider immediate commencement of root canal treatment (see below regarding luxation and avulsion injuries).

## Treatment of root-fractured teeth

In general, if pulp necrosis and infection has been diagnosed in a root-fractured tooth, then root canal treatment of only the coronal fragment is indicated – that is, the apical fragment does not require treatment and all efforts should be made to avoid instruments and materials going beyond the fracture line (311) (Fig. 25.28). However, the first decision that must be made is whether the tooth is suitable for such treatment and restoration afterwards.

If the root fracture occurs at the cervical level, and especially if it is located coronal to the crestal bone level, then the tooth has a poor prognosis (186) and needs to be assessed to determine whether it will be feasible to restore the tooth with a prosthetic crown (typically retained by a post-core). In these cases, the coronal fragment should be removed so the root can be visually inspected for any further fractures within the root. If the remaining root is suitable for a prosthesis, then root canal treatment can be performed immediately, and the tooth can be restored as required. Some of these cases may need orthodontic extrusion to facilitate restoration (see Chapter 28). Teeth with such fractures will generally require this treatment as part of the emergency management of the injury as it is not usually feasible to stabilize a fracture at this level of the root. It is highly likely that the pulp will initially develop pulpitis and then undergo necrosis rapidly since it may be affected by bacteria from dental plaque as the patient will not be able to clean this area effectively.

When root fractures occur in the coronal third of the root (but within bone), root canal treatment should only be commenced if the pulp undergoes necrosis and becomes

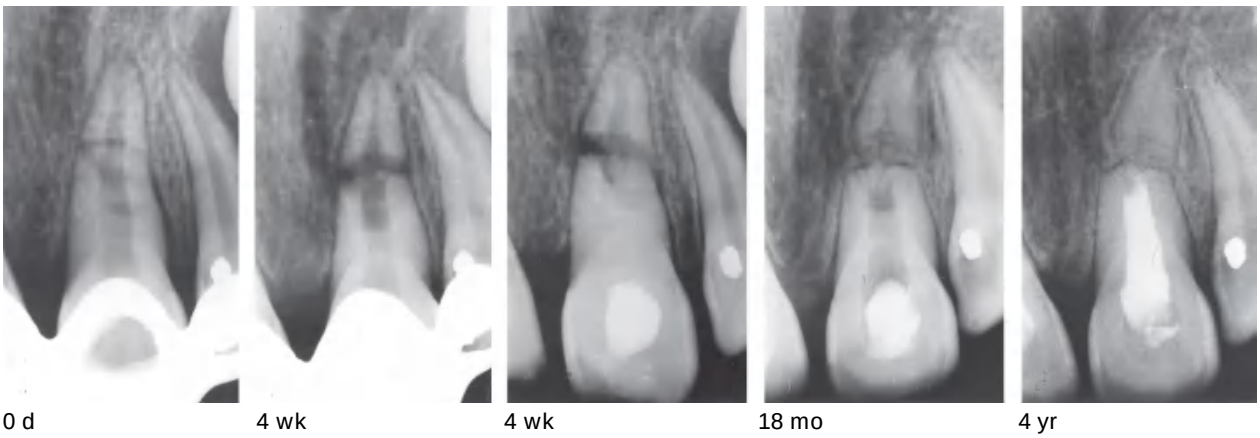



**Fig. 25.28** Root fracture and treatment of the infected coronal fragment with calcium hydroxide. Four weeks after injury there is widening of the space between fragments and adjacent radiolucency. The coronal fragment was treated with calcium hydroxide and 1½ years later, periradicular healing and apical closure with hard tissue of the coronal fragment was observed. Four years after filling with gutta-percha, periradicular healing is evident. Source: Reproduced from (65) with permission.

infected, as outlined above. Likewise, root canal treatment should only be done for teeth with root fractures in the middle or apical third of the root when pulp necrosis and infection has been diagnosed. In these cases, the root canal treatment is only performed to the fracture line. The exact nature of the root canal treatment depends on the condition of the tooth at the time that the treatment is commenced – in particular, it depends on how wide the root canal is, especially at the opening to the fracture line. Whilst this is not the 'apical foramen' of the tooth, it is effectively the 'new apical foramen' of the coronal fragment. If this is wide open, then there are several choices for treatment - see below. If the canal and this foramen are narrow, then routine root canal treatment can be done, and the root canal filling can be placed once the root canal system has been adequately disinfected (Figs 25.20 H and 25.22). Ideally, this involves the use of appropriate intracanal medicaments such as calcium hydroxide to ensure all of the root dentin has been disinfected as well as possible in order to increase the predictability of the treatment (189). If there are symptoms of acute inflammation of the peri-radicular tissues at the time of presentation, then some clinicians may choose to use other medicaments such as a corticosteroid-antibiotic compound to manage the initial pain before then using calcium hydroxide to ensure maximum anti-bacterial action has been obtained (190).

If the foramen of the coronal fragment is wide open, then it can be difficult to complete the root canal filling without any of the materials being extruded into the peri-radicular tissues. The potential problem with such extrusion is a foreign body reaction which may delay or prevent healing (Fig. 25.23). Hence, the root canal treatment needs to be modified to overcome this potential problem. One possible approach is to take extra care to keep all materials and instruments within the coronal fragment, but this is not always possible, especially because cements (unset when placed) and soft materials are used, and they require compaction or condensation to place them adequately. Two main approaches have been advocated by various authors and clinicians to provide more predictable root canal fillings in these cases – either the use of long-term calcium hydroxide dressings (similar to apexification for an open apical foramen) to encourage a natural hard tissue barrier to form (191) (Fig. 25.24), or the placement of a specific hard-setting bioceramic material (such as mineral trioxide aggregate) at the open end of the canal to create an artificial barrier (Figs 25.29 and 25.30).

In rare cases, the apical fragment may have pulp necrosis and infection of the root canal system. This would be evidenced by the presence of a periapical radiolucency. Fortunately, these cases are rare. They can be managed by treating the entire canal but there are potential problems associated with impacting necrotic and infected debris between the fragments (i.e. in the fracture line), as well as the root filling materials (gutta-percha and cement) which may delay or prevent healing (Fig. 25.25). This type of treatment generally has a poor prognosis if the problem develops in a young tooth soon after the fracture. However, if the necrosis and infection occur as a late problem after the root canal has narrowed and the fracture has healed with hard tissue, then the prognosis is better (Fig. 25.31).

Surgical removal of the apical fragment may occasionally be necessary. This must be accompanied by root canal treatment of the coronal fragment. Surgical removal of the apical fragment should only be contemplated if the apical fragment is infected and cannot be treated with conventional root canal treatment, or it has not responded to this treatment. There may also be the occasional case where the apical and coronal fragments are quite separate and therefore access to the apical fragment cannot be gained through the coronal fragment (Fig. 25.26). In some cases, surgery may be required where there has been extrusion of material through the foramen of the coronal fragment and this is affecting the healing response – in these cases, the apical fragment usually requires removal in order to gain adequate access to curette around the end of the coronal fragment (Figs 25.23 and 25.25).

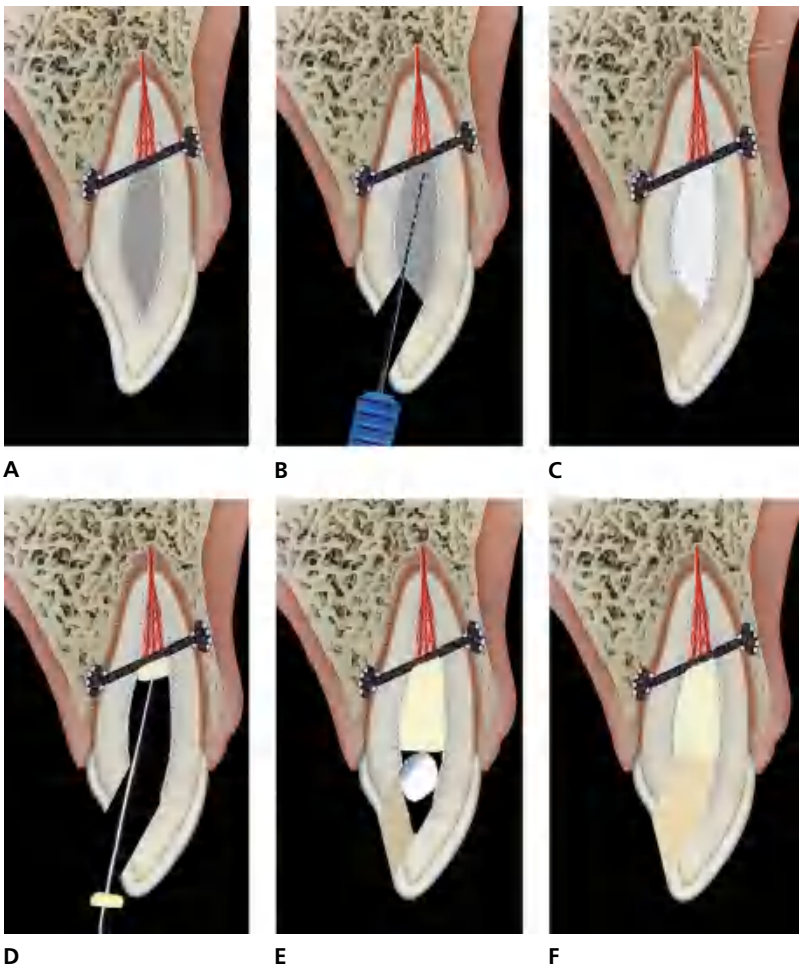



**Fig. 25.29** MTA used in the treatment of a root fracture with pulp necrosis and infection of the coronal fragment. A. The tooth a circum-radicular radiolucency associated with the fracture site is anesthetized if needed and isolated with a rubber dam. B and C. A standard access cavity to the root canal system is prepared. The necrotic tissue to the level of the root fracture is removed; the canal is biomechanically prepared to that level and irrigated with sodium hypochlorite. The canal (to the fracture level only) can be filled with calcium hydroxide as an interim anti-bacterial medication between appointments. D, E and F. The clinician must decide whether to use a 'plug' of MTA at the 'apex' of the canal in the coronal fragment (i.e. at the fracture line) or alternatively to fill the entire coronal fragment with the MTA. In either case, the MTA should be left to set before either adding gutta-percha to the remainder of the coronal canal followed by the access cavity filling or placing the access cavity filling directly on top of the MTA if the MTA was placed to the cervical level.

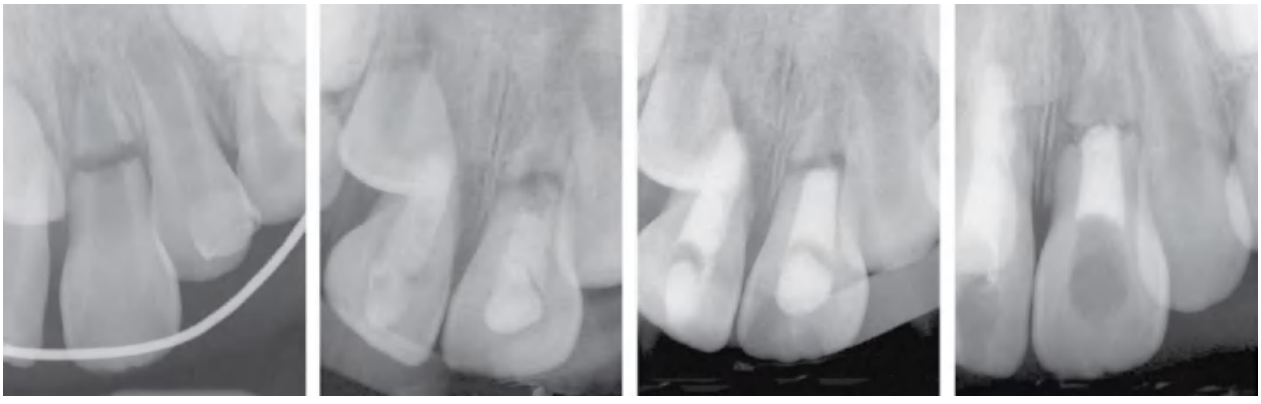



**Fig. 25.30** MTA used in root fracture treatment. A. A 9-year-old boy was in an accident in which the right central incisor was avulsed. and the coronal segment of the root fractured left central incisor was also avulsed. Both teeth were replanted within a few minutes. B. Three weeks after replantation, calcium hydroxide was placed in the canals of both teeth. C. Two weeks later, MTA was placed in the canals, but only to the fracture line in the left incisor. D. Nine-month follow-up.

## Prognosis for root-fractured teeth

Several studies have investigated the prognosis of root-fractured teeth (179, 180, 189, 192–196). The overall prognosis of root-fractured teeth is good, as described above, with only about 27% requiring root canal treatment. Healing of the peri-radicular tissues following root canal treatment of root-fractured teeth is generally quite predictable. However, the responses do vary according to the type of treatment, the position of the fracture along the length of the root, the timing of the treatment (soon after or a long time after the injury), and whether any material was extruded into the peri-radicular tissues between the fragments.

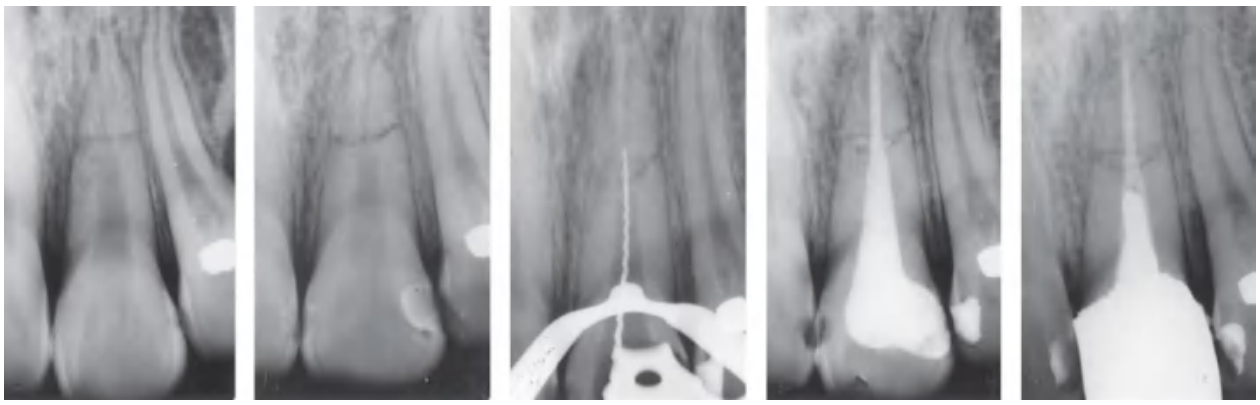



**Fig. 25.31** Treatment of a tooth with a healed root fracture and subsequent pulpitis due to a carious lesion. Three years after injury the root fracture appears to have healed. Two years later, a deep carious lesion was diagnosed and filled temporarily. However, symptoms of pulpitis continued and therefore root canal treatment was instituted. Note that no gutta-percha or cement was extruded between the fragments at the time of filling, which confirms initial internal repair of the root fracture. There were also no peri-radicular changes evident 4 years later (i.e. 9 years after injury).

In a review of 98 teeth with middle-third and apical-third root fractures, no healing occurred when both fragments were root-filled (187). The poor outcomes were reported to be due to extrusion of the root filling materials and/or necrotic debris which resulted in persistent radiolucencies in the adjacent tissues. Most of the fractures were in the apical third where the root canal is narrow and could be easily filled without any extrusion. If only the coronal fragment was filled with gutta-percha and cement, then healing occurred in 76% of cases. If the apical fragment had been removed surgically, healing was evident in 68% of the cases.

Periodontal healing was found in 86% of teeth that were treated with calcium hydroxide to form a hard tissue barrier in the coronal fragment when reviewed four years after treatment. Furthermore, in most teeth where the pulp in the apical fragment was not involved at the time of treatment, the root canal usually showed pulp canal obliteration when reviewed over time (180, 189) (Figs 25.20, 25.22, 25.24 and 25.27).

## Luxation and avulsion injuries

When considering the endodontic management of luxated and avulsed teeth, the main tissues to consider are the dental pulp and the periodontal ligament. The pulp is obviously the main concern with pulp necrosis and infection of the root canal system being the particular problem that leads to the need for root canal treatment. However, the periodontal ligament is also important as its healing responses can be affected by events within the pulp and root canal system. In particular, if the root canal system becomes infected and there has been damage to the periodontal ligament, then the conditions required for external infection-related resorption (also called inflammatory resorption) to occur will be present. Hence the following discussion will include some comments regarding this type of resorption and its management in conjunction with management of the pulp and root canal system.

### Pulp responses to trauma

When a tooth is displaced, the pulp blood vessels and nerves at the apical foramen can be compressed, stretched, lacerated or severed. The pulp response to such injuries depends on the type and degree of displacement, the time until repositioning of the tooth, the stage of root development and whether there is any bacterial contamination of the affected tissues. In general, the risk of pulp necrosis and infection increases with increasing stages of root development and severity of the luxation injury (197–204). Concussion and subluxation have the best prognosis as they do not result in displacement of the tooth whereas (in increasing order of severity) extrusion, lateral luxation, intrusion and avulsion are more severe injuries with increasingly greater risk of pulp necrosis and infection.

A sudden, complete severance of the pulp blood supply leads to infarction and coagulation necrosis of the pulp. In mature teeth and in the absence of bacteria, this necrotic pulp tissue may remain in the root canal without any clinical symptoms or obvious radiographic change for long periods of time. The infarcted tissue can also revascularize with ingrowth of mesenchymal tissues. This will usually be followed by deposition of hard tissue along the canal walls and, sometimes there may also be continued root development (Fig. 25.32) (see Chapter 2). However, the necrotic pulp most often becomes contaminated by micro-organisms from the oral cavity with the subsequent development of periapical inflammation (204–209). The micro-organisms have several potential pathways which have been demonstrated for penetration into the pulp or root canal system. One pathway is via the presence of blood clots harboring bacteria in the apical area of the root canal of avulsed and replanted teeth – this area was probably contaminated during the extra-alveolar period (Fig. 25.33). Another source of contamination is via any mechanically damaged cervical root surfaces through which bacteria from dental plaque could penetrate into the root canal system to infect the necrotic pulp tissue (Fig. 25.34). The root surface in this

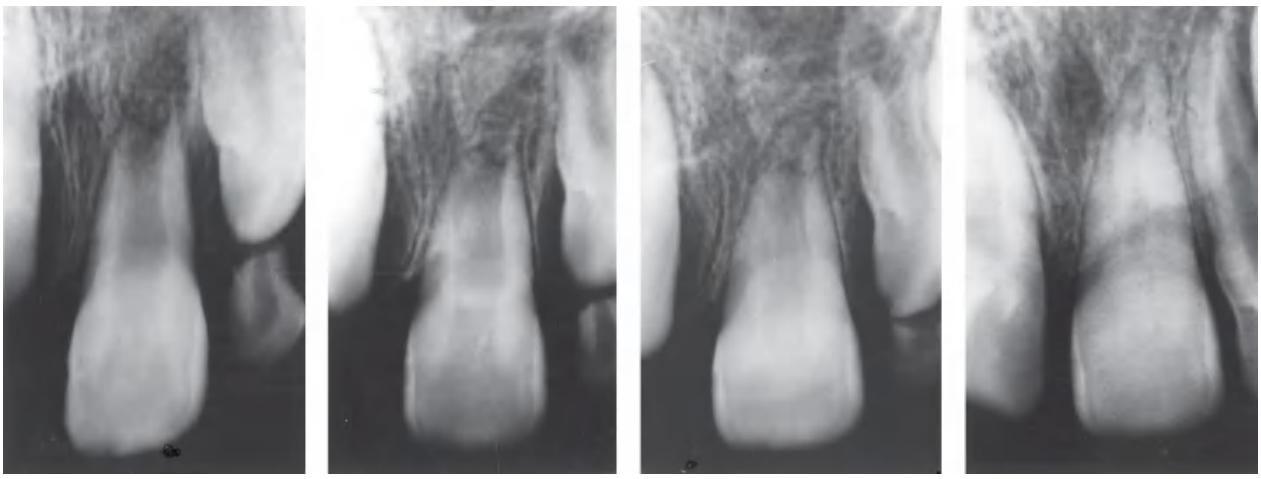



**Fig. 25.32** Continued root development after avulsion and replantation of an immature incisor. The tooth was replanted after 30 minutes of dry storage. Source: Reproduced from (214) with permission.

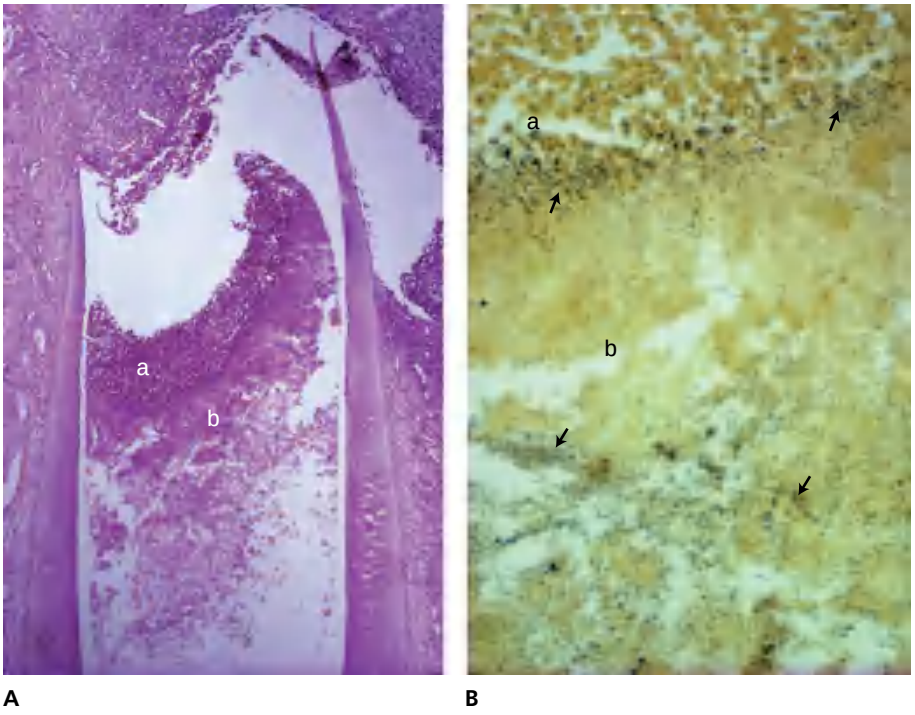



**Fig. 25.33** Presence of a contaminated blood clot in the root canal of an extracted and replanted immature monkey incisor. A. Apical area of the tooth; abscess formation at the apical foramen (a) and beneath it a blood clot (b). ×15. B. Enlarged view of the area between dense accumulation of leukocytes (a) and blood clot (b) shows heavy contamination of the blood clot with micro-organisms (arrows), Brown & Brenn bacterial stain. ×140. Source: Reproduced from (212) with permission.

region may be damaged during luxation injuries as a result of the fulcrum effect of the marginal bone when the tooth is displaced (212) (Fig. 25.35). It is also conceivable that bacteria may originate from a contaminated blood clot along the root surface. If a luxated or avulsed tooth also has a concurrent crown fracture, then the possibility of pulp necrosis and infection becomes higher – this is a result of the crown fracture allowing bacteria to enter the tooth either directly via a pulp exposure (if there has been a complicated crown fracture) or via the dentin tubules (if there has been an uncomplicated crown fracture).

Immature teeth have a greater chance of pulp recovery by revascularization through the open apical foramen than mature teeth with complete root development. If revascularization does occur, the pulp may then be able to resist the bacterial invasion with no long-term consequences apart from the need for regular monitoring. The chances of revascularization occurring depends on many factors such as the degree of displacement of the tooth, the degree of root development, the treatment provided, etc. Clinical and experimental studies of immature replanted teeth have reported that the frequency of complete pulp revascularization was relatively low (204, 211–214).

If revascularization does not occur, then a periapical radiolucency and/or infection-related root resorption is likely to develop – these changes are related to the presence of microorganisms in the root canal system. In experimental studies designed to imitate clinical conditions, the presence

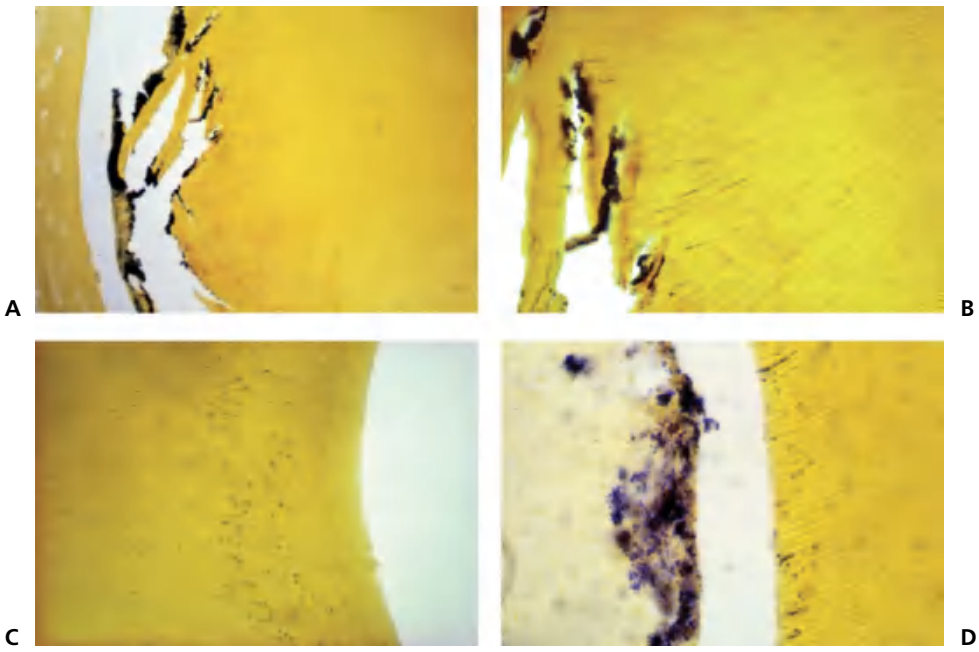



**Fig. 25.34** Pathway of entry of micro-organisms via cervical root damage to the root canal. A and B. Crushed cementum and dentin in the cervical area of the tooth, formation of plaque and penetration of micro-organisms into dentinal tubules, which could be followed in serial sections through the dentinal wall into the root canal. ×30 and 140. C and D. Micro-organisms in the dentinal wall close to the root canal and formation of colonies in the necrotic pulp. Source: Reproduced from (212) with permission.

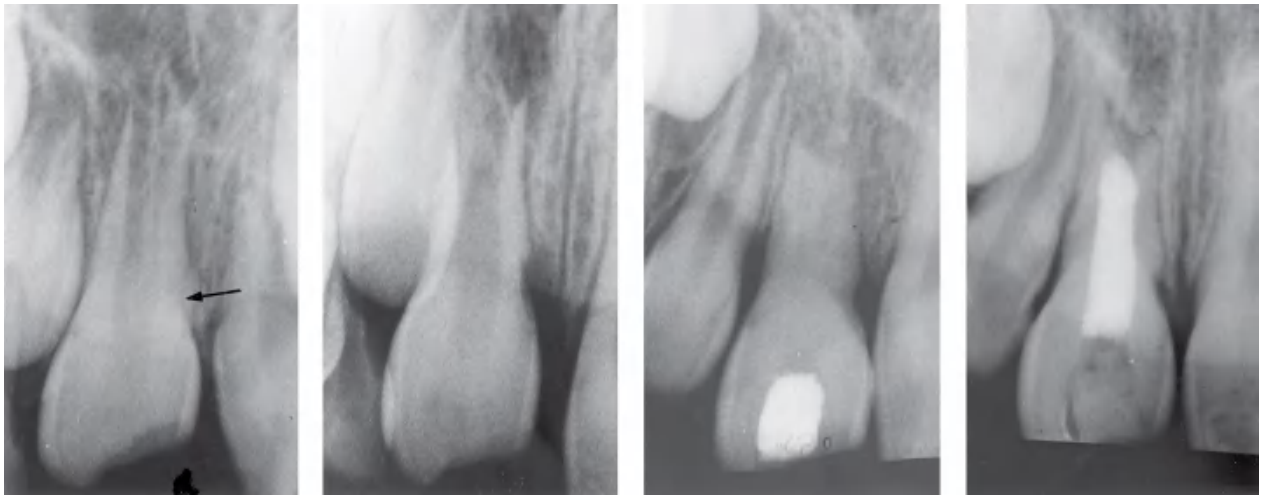



**Fig. 25.35** Effect of the impact of marginal bone on the root surface of an intruded incisor. The arrow points to the area of impact. Six weeks later there is resorption of the root surface and a periapical radiolucency. After calcium hydroxide treatment and filling of the root canal with gutta-percha and cement, the root resorption has been arrested and periapical healing is evident 5 years after the injury.

of micro-organisms was the predominant reason for the absence of complete revascularization (204).

In most experimentally replanted immature teeth with necrotic and contaminated pulps, some viable pulp tissue and sometimes formation of new hard tissues can be seen in the apical portion of the root canal, despite adjacent inflammation and abscess formation either in the root canal or periapically (204, 212) (Fig. 25.36). In some teeth, new dentin can even be seen, indicating that part of the original pulp survived, probably due to the diffusion of nutrients from the periapical tissues (215) (Fig. 25.37). The presence of viable pulp tissue in the apical part of the root canal is important from a therapeutic point of view, as this may help a more rapid formation of a hard-tissue barrier and sometimes continued root development after treatment of infected immature teeth with calcium hydroxide (216–218) (see below for further details).

### *Concussion and subluxation*

Concussion and subluxation are relatively minor injuries that mainly affect the periodontal ligament and periradicular tissues rather than the pulp. The neuro-vascular bundle at the apical foramen is usually not damaged to any extent since the tooth is not displaced with these injuries, and especially with concussion. Whilst there is loosening with subluxation, the natural elasticity of the pulp nerves and blood vessels allows some stretching and

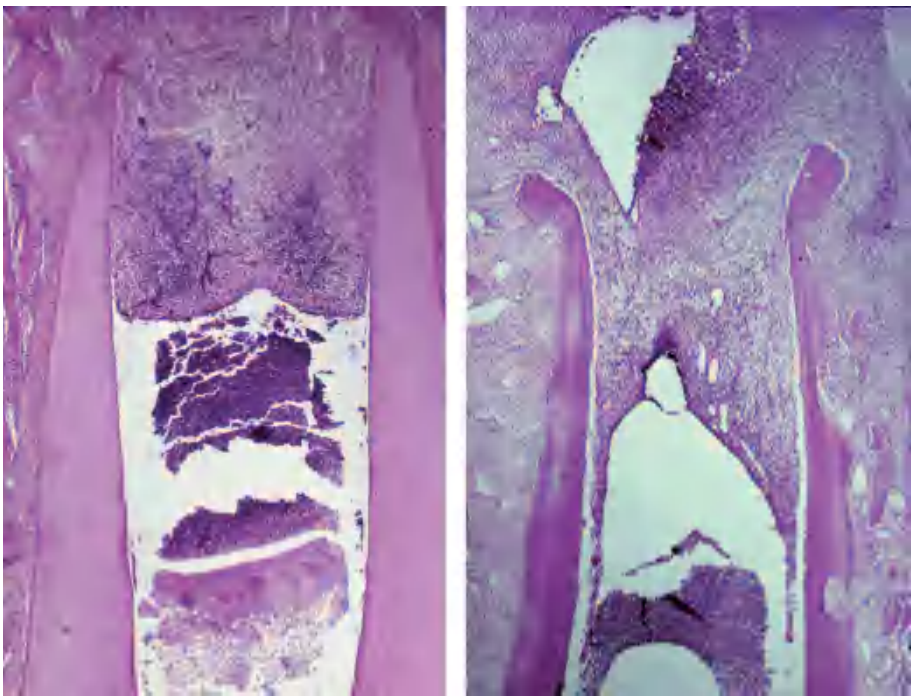
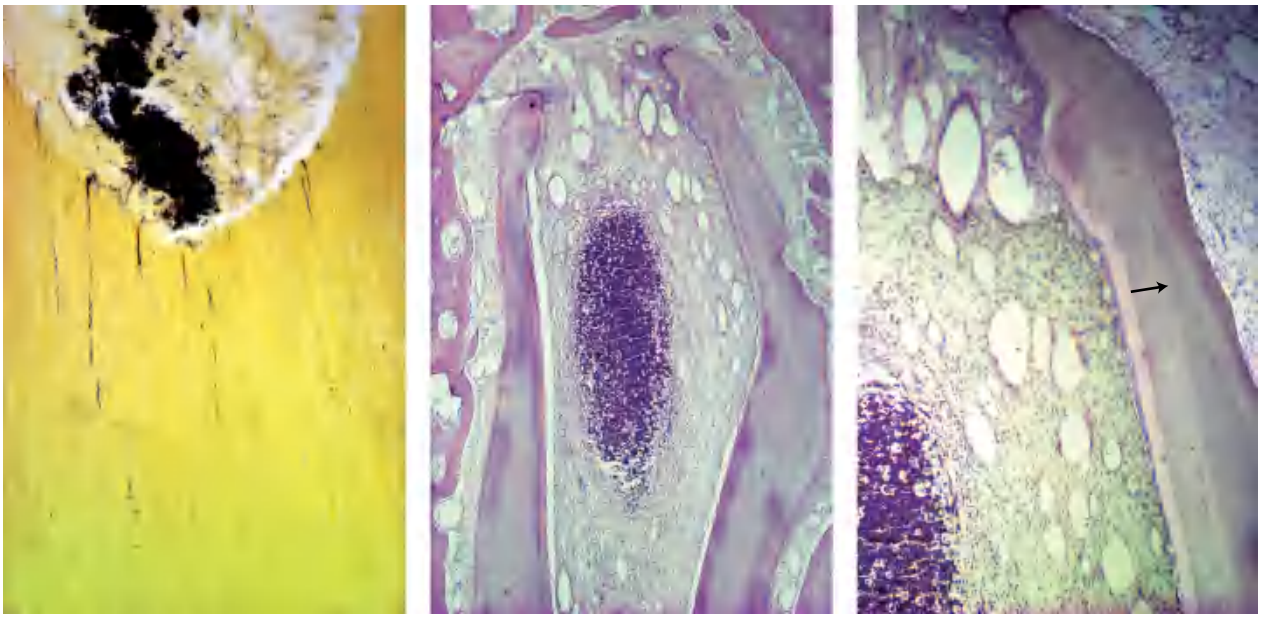


**Fig. 25.36** Presence of viable tissue in the apical portion of the root canal in replanted immature incisors, despite the presence of abscesses in the canal and periapically. ×20.



A B C

**Fig. 25.37** Penetration of micro-organisms into the necrotic pulp. A. Immature monkey incisor replanted after 30min storage in saliva showing penetration of micro-organisms through dentinal tubules of coronal dentin into the root canal; bacterial colonies are seen in the necrotized pulp, Brown & Brenn bacterial stain. ×100. B. Abscess formation in apical area. ×15. C. Formation of dentin is seen. The arrow indicates the line between pre- and post-traumatically formed dentin. ×33. Source: Reproduced from (212) with permission.

therefore it is unlikely that the nerve fibers or blood vessels will be severed. However, with both concussion and subluxation injuries, there may be some crushing of the neuro-vascular bundle at the apex and this may compromise the nerve and blood supply which may result in longer-term consequences. The presence of a concurrent crown fracture increases the risk of pulp necrosis and infection, as discussed above.

The periodontal ligament will be damaged slightly with concussion and subluxation injuries. Typically, there will be bleeding, swelling and rupture of some ligament fibers. These usually heal without complications although if the pulp necroses and becomes infected, then there is a possibility of external infection-related resorption developing. This type of resorption also needs damage to the cementum which is not a

typical feature of these injuries; hence, infection-related resorption is not common.

The overall prognosis for teeth that have had concussion or subluxation is generally quite good. Pulp necrosis and infection is relatively rare, especially in the absence of a concurrent crown fracture, and ranges from 3% for concussion to 6% following subluxation (200). The periodontal ligament usually heals without complication. External root resorption is also rare – in one large study, only 4% and 2% of concussed and subluxated teeth, respectively, developed external repair-related resorption, only 1% of subluxated teeth had external infection-related resorption and no cases had external ankylosis-related resorption or ankylosis (200). The stage of root development and concurrent crown fractures are significant factors related to the development of pulp necrosis and infection following these injuries (219) (see Chapter 17). The low rate of problems following these injuries is related to the relatively minor, and reversible, damage that occurs.

Healing following root canal treatment if pulp necrosis and infection occurs is usually uneventful and quite predictable.

### *Extrusive luxation*

Extrusive luxation is a more severe injury than concussion and subluxation since there is likely to be more damage to the periodontal ligament, plus there will be stretching of the neuro-vascular bundle at the apex of the tooth root – that is, the pulp's blood and nerve supply. This will affect the pulp immediately after the injury as well as potentially affecting its ability to recover. The natural elasticity of the pulp nerves and blood vessels will allow some stretching with a chance of recovery, but the extent of extrusion will determine whether the nerves and blood vessels will be severed or not – that is, the further the extrusion, the more likely they will be severed. Damage can also occur during repositioning of extruded teeth so careful manipulation is required.

If an extruded tooth also has a concurrent crown fracture, then the possibility of pulp necrosis and infection becomes higher, as discussed above. If the pulp's blood supply has not been affected, then the pulp may be healthy enough to resist the bacterial invasion with no long-term consequences as long as the tooth is restored adequately and in a timely manner. However, if the pulp's blood supply has been compromised (e.g. by being severed at the apical foramen), then the pulp may not be able to resist the bacterial invasion and pulp necrosis and infection may result. This will not be apparent immediately after the trauma and therefore the tooth must be closely monitored with clinical and radiographic examinations. Pulp sensibility tests and radiographs are essential as part of this follow-up process.

There will be at least some damage to the periodontal ligament with extrusion injuries. Typically, there will be bleeding, swelling and rupture of the ligament fibers. These usually heal without complications, especially if the pulp also recovers. However, if the pulp necroses and becomes infected, then there is a possibility of external infection-related resorption developing.

The overall prognosis for teeth that have been extruded is generally quite good. Pulp necrosis and infection in the absence of a concurrent crown fracture occurred in 26% of extruded teeth (200) and the periodontal ligament usually heals without complication. External root resorption is also rare – only 6% of extruded teeth had external repair-related resorption, only 6% had external infection-related resorption and no cases had external ankylosis-related resorption or ankylosis (200). The stage of root development and concurrent crown fractures are significant factors related to the development of pulp necrosis and infection following this injury (219) (see Chapter 18). The low rate of problems following extrusion is related to the minor damage that occurs.

Healing following root canal treatment if pulp necrosis and infection occurs is usually uneventful and quite predictable.

### *Lateral luxation*

Lateral luxation is a more severe injury than concussion, subluxation and extrusion since there is more damage to the periodontal ligament plus the cementum may also be damaged in the apical portion of the tooth root. In addition, there will be damage to the neuro-vascular bundle at the apex of the root. This will therefore affect the pulp immediately after the injury as well as potentially affecting its ability to recover. When a tooth is laterally luxated, the pulp blood vessels and nerves will always be severed due to the nature of the injury and the amount of displacement that occurs. The apex of the tooth root will very likely have some physical damage as the tooth root moves across the 'lip' or 'ledge' of bone that is created when the alveolar bone fractures – typically, this occurs on the labial aspect of the socket wall. As the root apex scrapes across the bone, the neuro-vascular bundle will most likely be severed. Damage can also occur during repositioning of laterally luxated teeth so careful manipulation is required.

In some cases, the pulp's blood supply may be re-established (revascularization) once the tooth has been repositioned and stabilized. Then the pulp may be able to resist the bacterial invasion with no long-term consequences apart from the need for regular monitoring. However, if a tooth has been laterally luxated in conjunction with a crown fracture, then the possibility of pulp necrosis and infection becomes much higher with very few, if any, pulps surviving (219). This may not be apparent immediately after the trauma and therefore the tooth must be closely monitored with clinical and radiographic examinations. Pulp sensibility tests and radiographs are essential as part of this follow-up process.

The periodontal ligament will be damaged with lateral luxation injuries. Typically, there will be bleeding, swelling and rupture of the periodontal ligament fibers. These usually heal without complications if the pulp also recovers. However, if the pulp necroses and becomes infected, then

there is a high possibility of external infection-related resorption developing. This type of resorption is more likely after lateral luxation because of the physical damage to the cementum that likely occurs in the apical part of the root due to the root scraping across the bone lip or ledge, as described above.

The overall prognosis for teeth that have been laterally luxated is generally quite good although some intervention may be necessary (e.g. root canal treatment) for more than half the teeth as pulp necrosis and infection in the absence of a concurrent crown fracture has been reported to occur in 58% of laterally luxated teeth (200). The periodontal ligament usually heals but external root resorption may occur – 26% of laterally luxated teeth had external repair-related resorption, although only 3% had external infection-related resorption and 1% had external ankylosis-related resorption or ankylosis in one study (200). The stage of root development and concurrent crown fractures are significant factors related to the development of pulp necrosis and infection following this injury (219) (see Chapter 18). The higher rate of problems following lateral luxation compared to concussion, subluxation and extrusion is related to the increased damage that occurs during the injury and possibly also whilst being repositioned.

Healing following root canal treatment if pulp necrosis and infection occurs is usually uneventful and quite predictable.

### *Intrusion*

Intrusive luxation is potentially a very severe injury since there is far more damage to the periodontal ligament plus the cementum may also be damaged along the entire length of the tooth root. In addition, all intrusion injuries will cause damage to the neuro-vascular bundle at the apex of the root – this damage will typically be in the form of crushing. The amount of damage will depend on the degree of intrusion that occurs with minor intrusions possibly having little effect whereas intrusions beyond a few millimeters are potentially very damaging. This damage will affect the pulp immediately after the injury as well as potentially affecting its ability to recover.

In some teeth with immature root formation, the pulp's blood supply may be re-established (revascularization) once the tooth has been repositioned and stabilized. The pulp may then be able to resist the bacterial invasion with no long-term consequences apart from the need for regular monitoring. However, if the tooth is fully developed or if there has also been a crown fracture, then the possibility of pulp necrosis and infection becomes much higher with very few, if any, pulps surviving. As the pulp's blood supply has been compromised by being crushed and/or severed at the apical foramen, the pulp is not able to resist the bacterial invasion so pulp necrosis and infection result (308).

The periodontal ligament will be damaged with intrusion injuries. Typically, there will be bleeding, swelling and rupture of the ligament fibers. These may heal without complications if the pulp also recovers. However, if the pulp necroses and becomes infected, then there is a high possibility of external infection-related resorption developing. This type of resorption is more likely after intrusive luxation because of the physical damage to the cementum. External ankylosis-related resorption is also more likely following intrusion because of this damage to the cementum and periodontal ligament (309).

The overall prognosis for teeth that have been laterally luxated is not as favorable as the other luxation injuries discussed above. Root canal treatment is likely to be required in the majority of intruded teeth as a result of pulp necrosis and infection - this has been reported to have occurred in 85% of intruded teeth in the absence of a concurrent crown fracture and close to 100% in fully developed teeth with a concurrent crown fracture (200). Periodontal ligament healing is also less predictable and more likely to be associated with external root resorption – 24% of intruded teeth had external repair-related resorption, another 38% had external infection-related resorption and 24% had external ankylosis-related resorption or ankylosis in one study (200). The stage of root development and concurrent crown fractures are significant factors related to the development of pulp necrosis and infection following this injury (219) (see Chapter 19). The higher rate of problems following intrusion compared to the other luxation injuries is directly related to the increased damage that occurs during this injury.

Healing following root canal treatment if pulp necrosis and infection occurs is usually uneventful and quite predictable.

### *Avulsion*

Avulsion injuries are a severe injury that affect the entire periodontal ligament and root surface, as well as having complete severance of the pulp's blood and nerve supply. Hence, recovery is less predictable following avulsion. However, the healing events are dependent on the actual amount of damage that occurs along with many other factors such as stage of root development, extra-oral time, storage medium, etc.

In some teeth with immature root formation, the pulp's blood supply may be re-established (revascularization) once the tooth has been replanted and stabilized. The pulp may then be able to resist the bacterial invasion with no long-term consequences apart from the need for regular monitoring. However, if the tooth is fully developed or if there has also been a crown fracture, then pulp necrosis and infection is a certainty. Fully developed roots have a small apical foramen and hence there is little space for blood vessels to grow back into the root canal to revascularize the pulp. A crown fracture potentially allows bacteria to enter the tooth either via a pulp exposure (if there has been a complicated crown fracture) or via the dentin tubules (if there has been an uncomplicated crown fracture). As the pulp has lost its blood supply by being severed at the apical foramen, it is not able to resist the bacterial invasion so infection of the root canal system will rapidly result.

The periodontal ligament will be damaged (severed) with avulsion injuries. Typically, there will be bleeding, swelling and rupture of the ligament fibers. These may heal without complications if the pulp also recovers. However, if the pulp necroses and becomes infected, then there is a high possibility of external infection-related resorption developing. This type of resorption is also more likely after avulsion because of the physical damage that may occur to the cementum during the injury, whilst the tooth is out of the mouth and during replantation. External ankylosis-related resorption is also more likely following avulsion because of this damage to the cementum and periodontal ligament.

The overall prognosis for teeth that have been avulsed is not as favorable in the long term as the luxation injuries discussed above. There are many factors that affect the prognosis of these teeth – see Chapter 20 for a full discussion. In summary, it is unlikely that the pulp in a fully developed tooth will survive and hence pulp necrosis and infection of the root canal system is almost a certainty in these teeth. The pulps in some teeth with incomplete root formation may survive and revascularize – up to 50% have been reported (220). Periodontal ligament healing is also less predictable and ranges from 9–50% for the various types of resorption (see Chapter 20). Of particular relevance to this discussion is external infection-related resorption which is related to infection of the root canal system and can be prevented (or arrested if already present) by performing root canal treatment (see below).

## Diagnosis of pulp necrosis and infection

Diagnosis of pulp necrosis following concussion, subluxation and luxation injuries is not possible immediately after trauma. In most cases, the pulp necrosis can only be confirmed by the development of symptoms and/or clinical signs as a result of the root canal system becoming infected – that is, via the presence of periapical changes evident radiographically, or via clinical signs of infection (swelling, tenderness to percussion, etc.). In addition, the tooth will not respond to pulp sensibility tests. Hence, rather than waiting for acute symptoms to occur, it is important to regularly monitor these traumatized teeth in order to identify pulp necrosis and infection at an earlier stage.

When assessing teeth for pulp necrosis and infection following trauma, there is no difference between the various types of injuries, except for severely intruded or avulsed and replanted teeth that are fully developed. For teeth with these latter injuries, no healing of the pulp can be expected and there is very likely to be significant damage to the periodontal ligament and cementum – that is, the conditions that lead to the development of external infection-related root resorption are present. Hence, root canal treatment should be commenced immediately following repositioning/replantation and stabilization of the tooth, irrespective of the clinical findings, in order to prevent external infection-related root resorption (see further details below). Pulp healing is also very unlikely following lateral luxation with a concurrent crown fracture of a mature tooth, extrusion with a concurrent crown fracture of a mature tooth, avulsion with a concurrent crown fracture of an immature tooth and intrusion with a concurrent crown fracture of an immature tooth (221) (Table 25.1). These injuries are also likely to have damaged the cementum and periodontal ligament. Hence, teeth with these injuries should also have root canal treatment commenced immediately to avoid external infection-related root resorption. All other teeth should be stabilized and then reviewed over time to determine whether the pulp recovers. and periodontal ligament healing occurs. It is also possible to find internal infection-related resorption occurring in rare cases (Fig. 25.38).

**Table 25.1** Injuries that are likely to result in pulp necrosis and infection of the root canal system, according to the stage of root development at the time of injury. Immediate root canal treatment following repositioning/replantation and splinting should be considered for teeth with these injuries in order to prevent the development of external infection-related (inflammatory) root resorption.

| Incompletely Developed Teeth | Fully Developed Teeth |
|---|---|
| • Intrusion with crown fracture<br>• Avulsion with crown fracture | • Avulsion<br>• Intrusion<br>• Lateral Luxation with crown fracture<br>• Extrusion with crown fracture |

Source: Reproduced from (221) with permission.

Both cold and electric pulp sensibility testing should ideally be performed at the emergency appointment following trauma – that is, when the patient first presents with the injuries. However, it is important to recognize traumatized teeth may not respond in a reliable manner on initial presentation. Hence, the results of this initial series of pulp tests should not be used to determine whether the pulp has necrosed or not. However, these tests provide good prognostic information as studies have shown a better prognosis for teeth that do respond immediately after trauma compared to those that do not respond. For example, approximately 50% of teeth that did not respond immediately after subluxation developed pulp necrosis whereas only about 3% of those that did respond became necrotic (222). Hence, the initial pulp tests are a good prognostic indicator for dentists and patients.

The initial pulp tests results, also provide baseline information that can be used for comparison with future tests at review appointments in order to determine whether the responses are changing. For example, if a tooth initially does not respond and subsequently it responds at later reviews, then this firstly alerts the clinician to the fact that the tooth has had trauma which may not be otherwise obvious, and secondly, the return to normal responses indicates that the pulp has recovered. On the other hand, if the tooth responded initially but not at later review appointments, then this is an indicator that the pulp has probably necrosed and so other signs of infection should also be assessed (e.g. pain, percussion, apical periodontitis, periapical radiolucency, infection-related resorption, etc.).

Pulp sensibility test results alone should not be used to determine pulp necrosis or recovery. They should be interpreted carefully in conjunction with all of the other clinical and radiographic signs obtained during the examination of

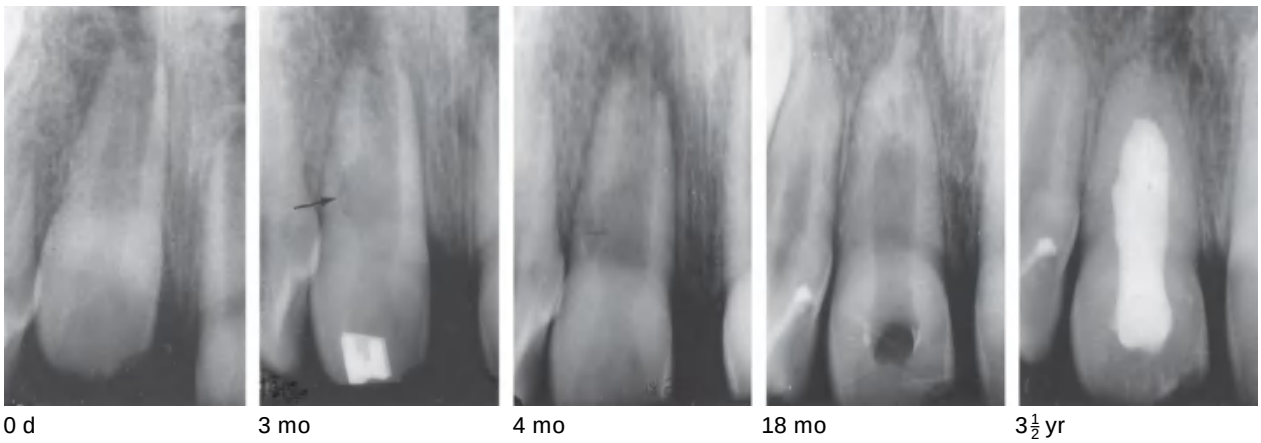



**Fig. 25.38** Internal inflammatory root canal resorption after intrusive luxation of a central incisor. Three and 4 months after injury, an inflammatory internal resorption with communication to the periodontium has developed cervically (arrow). After 18 months' treatment with calcium hydroxide, there is apical closure with hard tissue and communication with periodontium was no longer found. A control 3½ years after filling of the root canal with gutta-percha shows periradicular healing

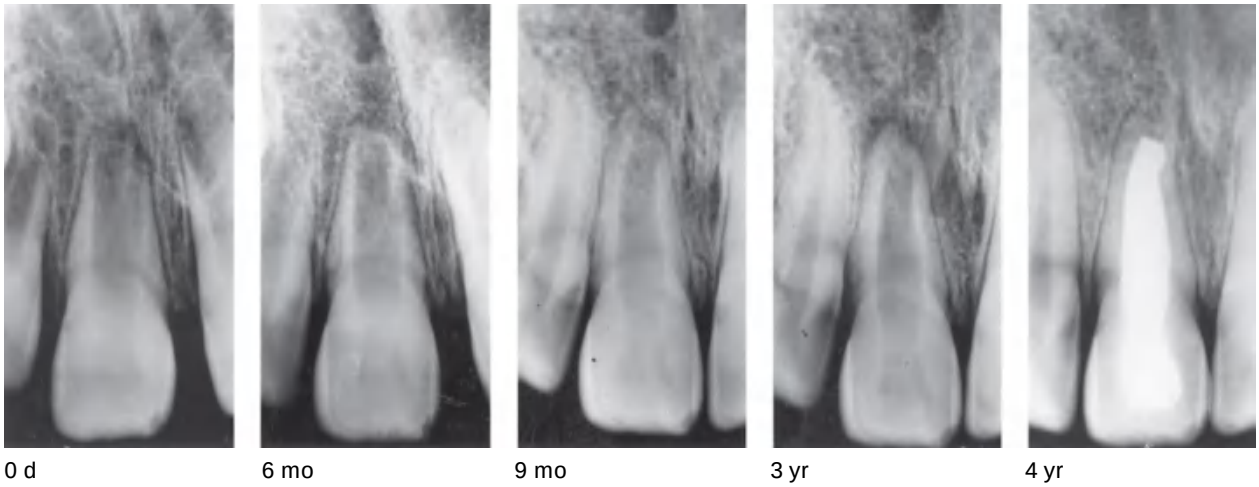



**Fig. 25.39** Arrested root development after a luxation injury. There is formation of hard tissue apically but none in the rest of the root canal, indicating pulp necrosis. A periapical radiolucency was seen 3 years after injury. Periapical healing is evident 4 years after treatment with calcium hydroxide and following root canal filling with gutta percha and cement.

the patient. Immature teeth may not respond as expected and some teeth can take months or even years to return to normal pulp test responses. Teeth with pulp canal obliteration are unlikely to respond to thermal testing although they may (but not always) respond to an electric pulp test. However, notwithstanding these limitations, positive responses to pulp tests are a good indicator of pulp innervation which then usually implies that the pulp has a viable blood supply.

At review appointments, patients should be questioned about whether they have noticed any changes or have any symptoms. A thorough clinical and radiographic examination should also be performed at each review appointment. Apart from pulp sensibility tests, other particular signs of pulp necrosis and infection should be checked – such as color change of the crown, mobility, palpation and percussion.

Radiographically, in the early stages of the infection and inflammatory processes, there will be a widened periodontal ligament space around the apex of the tooth root which can normally be seen within 2–8 weeks after the injury. As the inflammatory response progresses in the peri-radicular tissues, there will be bone resorption resulting in a radiolucent area surrounding the root apex. Hence the periapical radiolucency may appear later (225) (Figs 25.38–25.41).

The tooth should also be carefully assessed for any radiographic signs of resorption. However, care must be taken to avoid confusing transient apical breakdown with external infection-related resorption (see Chapter 16).

Some patients may complain of a draining sinus tract on the nearby mucosa, indicating that a chronic abscess has developed. If a draining sinus tract is present, a gutta-percha point should be placed in the sinus tract to trace its origins and to confirm that it is originating from the radiolucency associated with the injured tooth and not from another tooth.

The periodontal ligament generally heals without complications although compete healing takes up to about three

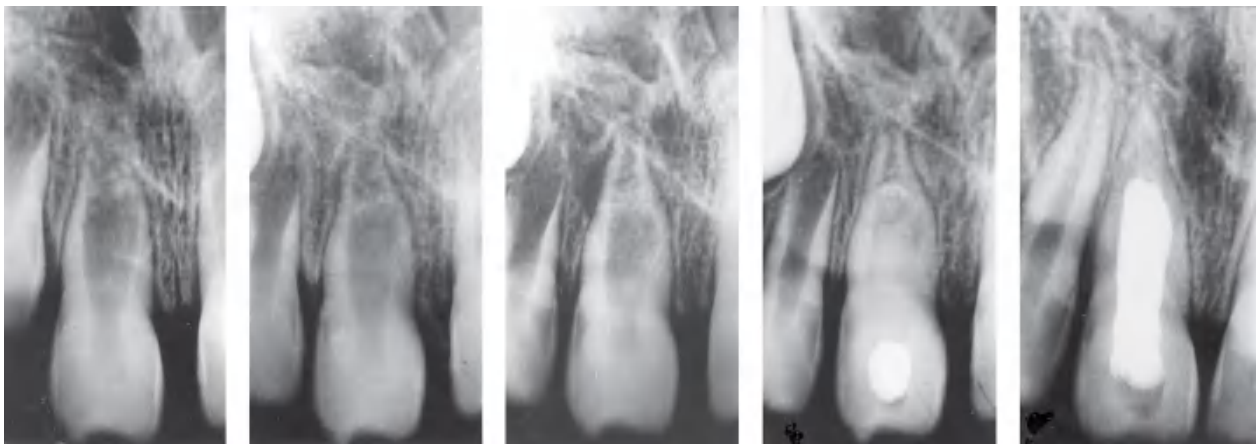



**Fig. 25.40** Minor disturbance in root development of a luxated incisor. There is formation of irregular hard tissue in the apical area, followed by further root development. Note the absence of hard tissue formation in the rest of root canal, indicating pulp necrosis. A periapical radiolucency was noted 1 year after injury. Peri-radicular healing is evident 5 years after root canal treatment with calcium hydroxide and subsequent filling with gutta percha and cement.

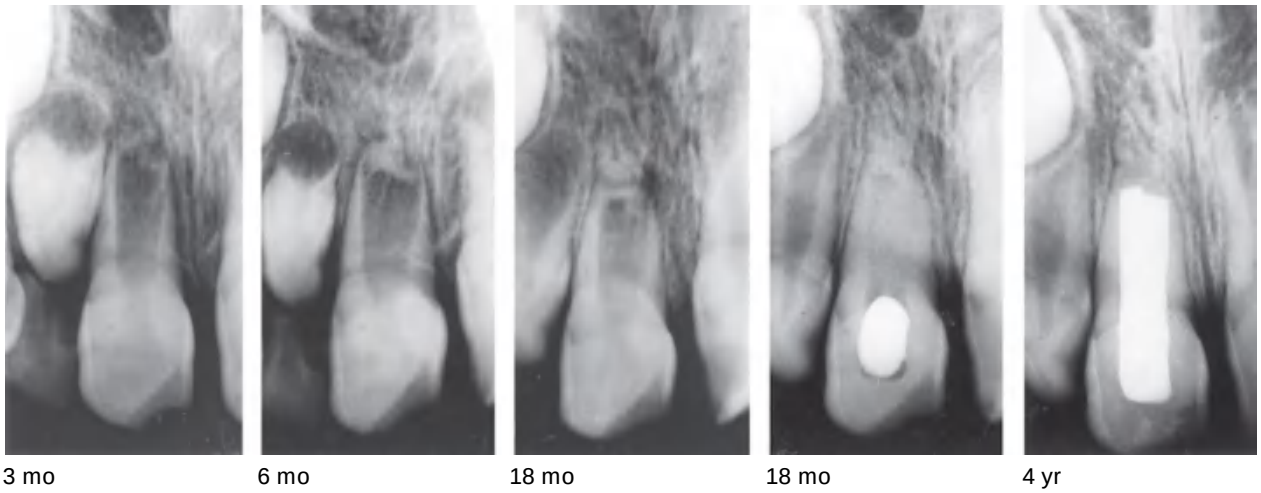



**Fig. 25.41** Disturbance in root development and formation of a root tip. There is formation of hard tissue apically but none in the rest of the root canal, indicating necrosis of the remaining pulp. Formation of separate root tip as well as a peri-radicular radiolucency were noted 18 months after injury. Healing is evident 4 years after after root canal treatment with calcium hydroxide and subsequent filling with gutta-percha and cement.

months. Hence, orthodontic or other irreversible treatment should be deferred until this healing has taken place.

Discoloration of the crown subsequent to trauma is not necessarily a sign of pulp necrosis and hence needs careful consideration. Discoloration may be pink, yellow, brown, grey or a combination of these colors (198). A pink or reddish discoloration within a few days of the injury indicates bleeding within the pulp but this may resolve, and the crown can regain its natural color several weeks later or even after several months (198, 219, 223–225). Persistent discoloration, especially with a shift to grey, may indicate necrosis and infection of the pulp (219, 226).

Disturbed or arrested root development is frequently seen after luxation injuries to immature teeth and it is usually followed by pulp canal obliteration, indicating recovery and the presence of viable pulp tissue (Figs 25.42 and 25.43). Barrier-like formation of hard tissue apically, with or without continued root development, may also occur when the coronal pulp is necrotic. However, sooner or later it becomes bacterially contaminated with acute or chronic periapical inflammation as a result. Thus, absence of any hard tissue formation in the rest of the root canal in these teeth, indicating no cell activity, is a strong indication of a coronal pulp necrosis (Figs 25.39–25.41). In these teeth, root canal treatment should therefore be considered before the appearance of radiographic periapical changes.

In general, the decision to perform root canal treatment should be based on an evaluation of clinical and radiographic findings as well as the history and the information gained from comparing radiographs taken immediately after the injury with those taken at subsequent review appointments.

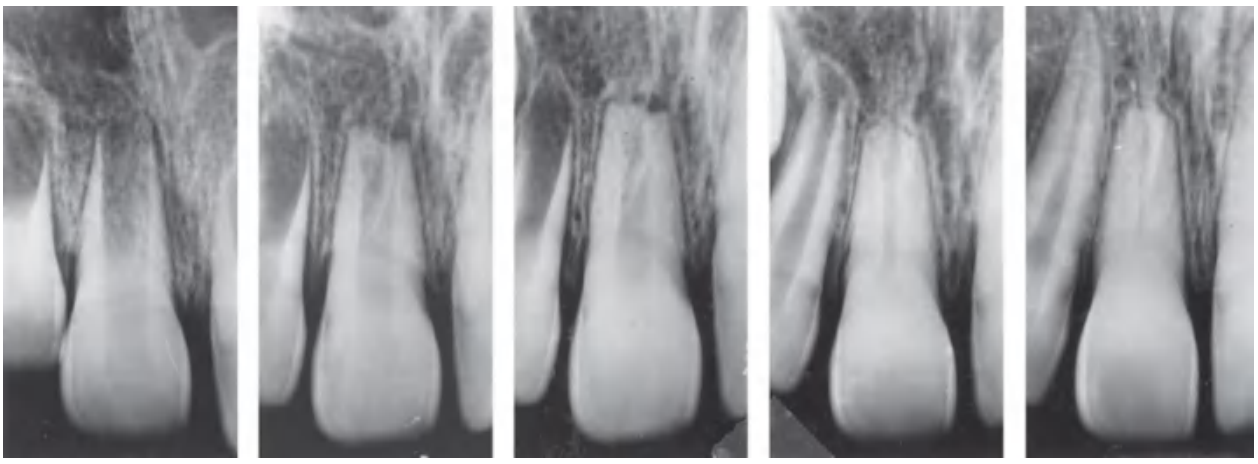



**Fig. 25.42** Arrested root development after a luxation injury. The root canal space has successively narrowed by formation of hard tissue, indicating presence of viable pulp tissue.

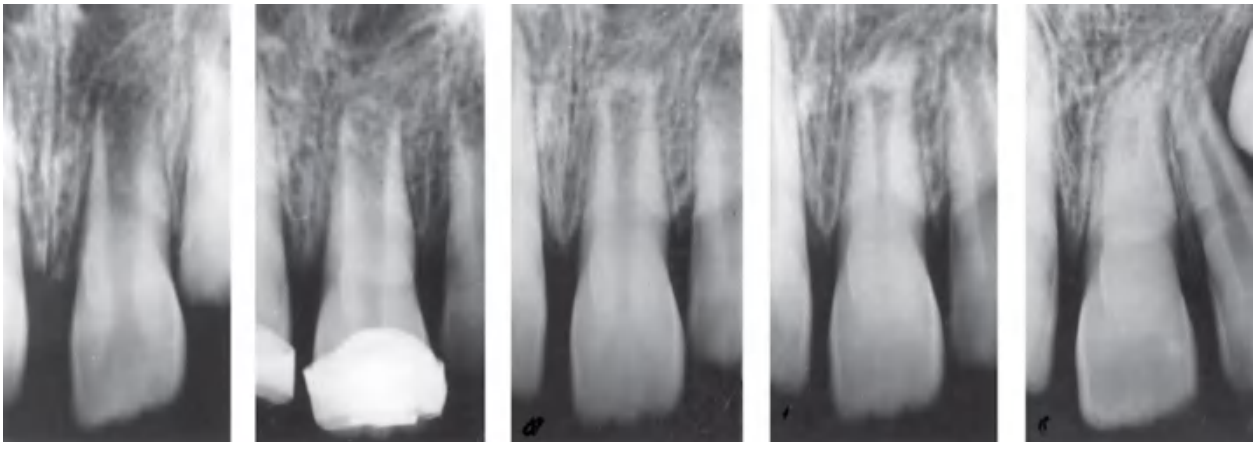



**Fig. 25.43** Disturbance in root development of an immature incisor after a luxation injury. Note successive narrowing of the root canal space by formation of hard tissue, indicating viable pulp tissue was present.

## Management of pulp necrosis and infection

Once a definitive diagnosis of pulp necrosis and infection of the root canal system has been made, root canal treatment should be commenced as soon as possible. The exact nature of the root canal treatment depends on the condition of the tooth at the time that the treatment is commenced – in particular, it depends on the width of the root canal and especially the width of the apical foramen – that is, the stage of root development. If the canal and the apical foramen are narrow, then routine root canal treatment can be done, and the root canal filling can be placed once the root canal system has been adequately disinfected. Ideally, this involves the use of appropriate intracanal medicaments such as calcium hydroxide to ensure all of the root dentin has been disinfected as well as possible in order to increase the predictability of the treatment (189). If there are symptoms of acute inflammation of the peri-radicular tissues at the time of presentation, or if external infection-related resorption is present or likely to occur, then some clinicians may choose to use other medicaments such as a corticosteroid-antibiotic compound to manage the initial pain, inflammation and resorption before then using calcium hydroxide to ensure maximum anti-bacterial action has been obtained (190).

### *Endodontic management of incompletely developed teeth*

If the apical foramen is wide open, then there are several choices for treatment – namely, apexification, placing an artificial barrier or performing a regenerative endodontic procedure. All three approaches can lead to healing and retention of the tooth in the long-term, and there are some advantages and disadvantages with each. A comprehensive review by Harlamb (227) has discussed this topic and the following treatment options in depth.

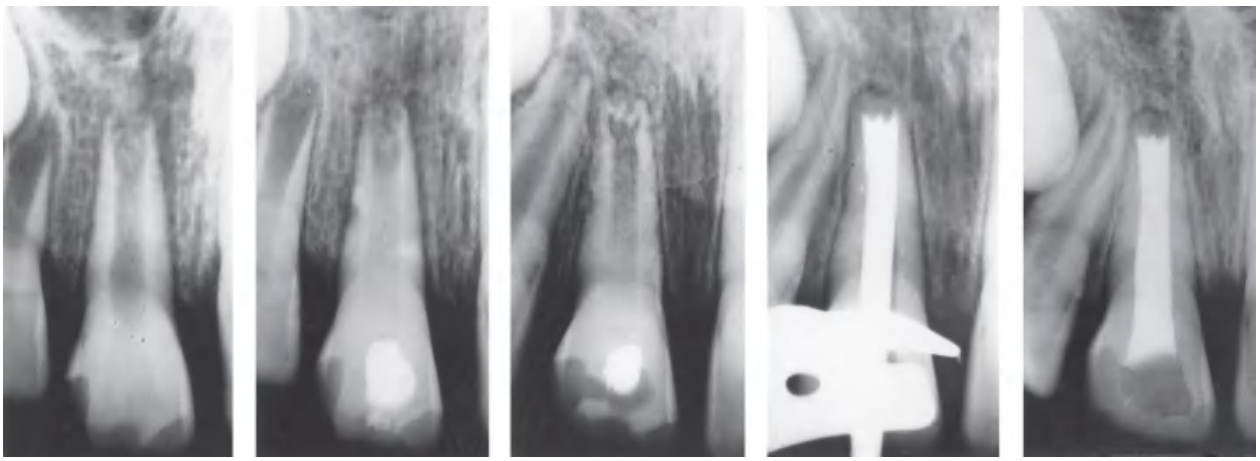



**Fig. 25.44** Treatment of an immature tooth with calcium hydroxide as a result of pulp necrosis and infection. Periapical healing and apical closure with hard tissue was evident after 18 months and the root canal was filled with gutta-percha and cement. A custom-fitted master gutta-percha point was fitted by heat-softening the tip and inserting it against the hard tissue barrier. Thereafter, the root canal was filled with additional gutta- percha points, using the lateral condensation technique.

### Apexification

Apexification is the process by which a natural hard tissue barrier is induced prior to placing a conventional root canal filling (Fig. 25.44). Rafter has published a comprehensive review of this technique (228). It involves the long-term application of a material such as calcium hydroxide paste which works by irritating the surrounding tissues leading to a calcification process (Fig. 25.45). Whilst the exact mechanism is not well understood, it is a highly predictable procedure that results in a hard tissue barrier closing the open foramen of the canal – typically this barrier consists of cementum and/or bone. The location of the barrier depends on the level at which the calcium hydroxide has been brought into contact with viable tissue (Fig. 25.46) and some cases may even show further root development, probably due to survival of Hertwig's epithelial root sheath (216–218) (Fig. 25.47).

The calcium hydroxide paste needs to be replaced several times, typically at intervals of about 2–3 months (191, 229). The more frequent change of calcium hydroxide dressing has been shown to result in significantly more rapid formation of the apical hard tissue barrier (229). Once the barrier is felt, then the root canal filling can be completed with minimal chance of material extrusion into the surrounding tissues (Fig. 25.48). The root canal filling technique requires the use of either a heat-softened customized gutta-percha point with lateral condensation (Fig. 25.49) or some other form of thermoplasticized gutta-percha technique (e.g. injection or vertical compaction) (Fig. 25.50).

The apical hard tissue barrier that forms can also facilitate root canal re-treatment if it becomes necessary in the future (i.e. if the root canal system becomes infected again) by confining the instruments, irrigants, medicaments and other materials within the canal (Fig. 25.50).

The only slight potential disadvantage of this technique is the time taken which averages about 12 months (191) although it is dependent on the pre-operative width and shape of the apical part of the root canal – one study has reported a mean of 8 months for convergent and parallel canal walls up to a mean of 11 months for divergent walls although two cases out of a total of 44 took about 17 months (229).

In recent years, there has been some concern about the effects of calcium hydroxide on weakening of the tooth structure. Cvek (230) first mentioned this problem when he reported that young children's teeth were susceptible to cervical root fractures following apexification procedures. (Figs 25.51 and 25.52). In that study, the least developed teeth were the most likely to fracture whilst fully developed teeth had few fractures. Cvek's results suggest that the amount of remaining dentin is the most critical factor that can predispose a tooth to fracture (Fig. 25.53). Subsequent studies (231–233) have shown some effects on dentin from the application of calcium hydroxide although these studies do not replicate the in vivo situation, especially when further trauma occurs. A downfall of most of those studies is the way loads have been applied in a continuous manner using a universal testing machine on root segments rather than by using an impact device on the whole tooth. Unfortunately, the alternative treatments suggested below have yet to overcome this problem as neither produce dentin in the critical cervical part of the tooth and hence these fractures may still occur, especially if there is a subsequent traumatic incident involving that same tooth. In addition, the use of mineral trioxide aggregate materials (MTA) is in effect a delivery system for calcium hydroxide as MTA produces calcium hydroxide (234). Also, the root canal system needs to be disinfected prior to application of the MTA or prior to regenerative endodontic procedures and calcium hydroxide is commonly used for this, although for shorter periods of time. Hence, the potential for cervical root fractures still exists and this problem requires further research.

**Fig. 25.45 Filling of the root canal with calcium hydroxide**
A palatal opening is made wide enough to allow adequate mechanical cleansing of the root canal.

**Filling the root canal**
After completed cleansing, the cannula is introduced in the root canal close to the apical area and then slowly withdrawn while pressing calcium hydroxide out of the syringe.

**Compressing calcium hydroxide**
The paste is then pressed lightly towards the apex with a dry cotton pellet to ensure apical contact with vital tissue. Filling and condensing of calcium hydroxide is repeated until the root canal has the same radiodensity as the surrounding dentin.

**Sealing the access cavity**
After the excess of the paste is removed, the coronal cavity is sealed of with zinc oxide-eugenol cement. One year after commencing root canal treatment, periapical healing and formation of the apical hard-tissue barrier is seen

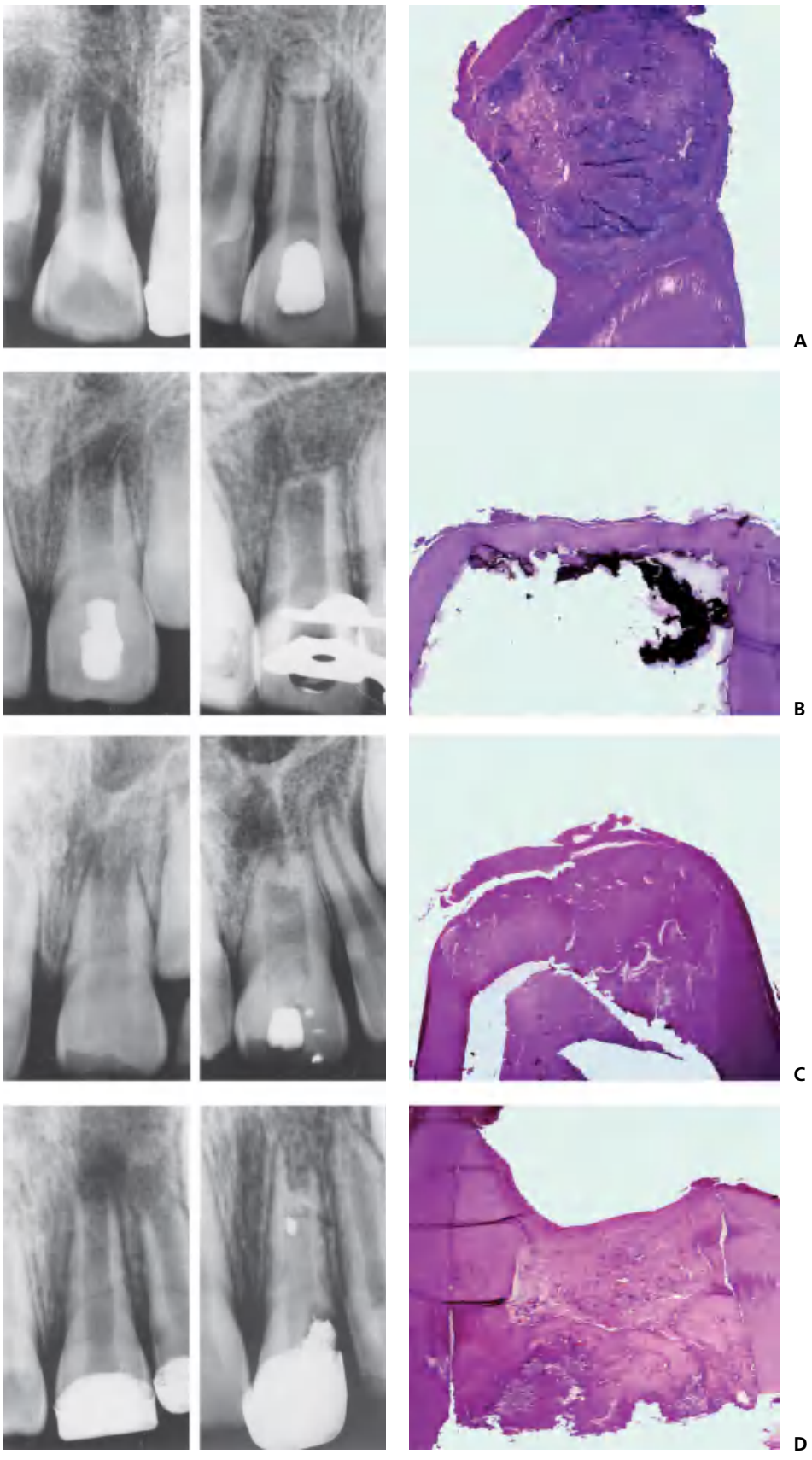



**Fig. 25.46** Different levels of formation of the hard tissue barrier after treatment with calcium hydroxide. Radiographs were taken before treatment and after completed apical closure. The teeth were extracted because of cervical root fractures. A. Formation of hard tissue in the periapical area as a result of overfilling with calcium hydroxide. ×10. B and C. Barriers formed across the apical foramen. ×20. D. Formation of hard tissue barrier within the root canal. ×20. In all cases, newly formed hard tissue was found to be cementum-like.

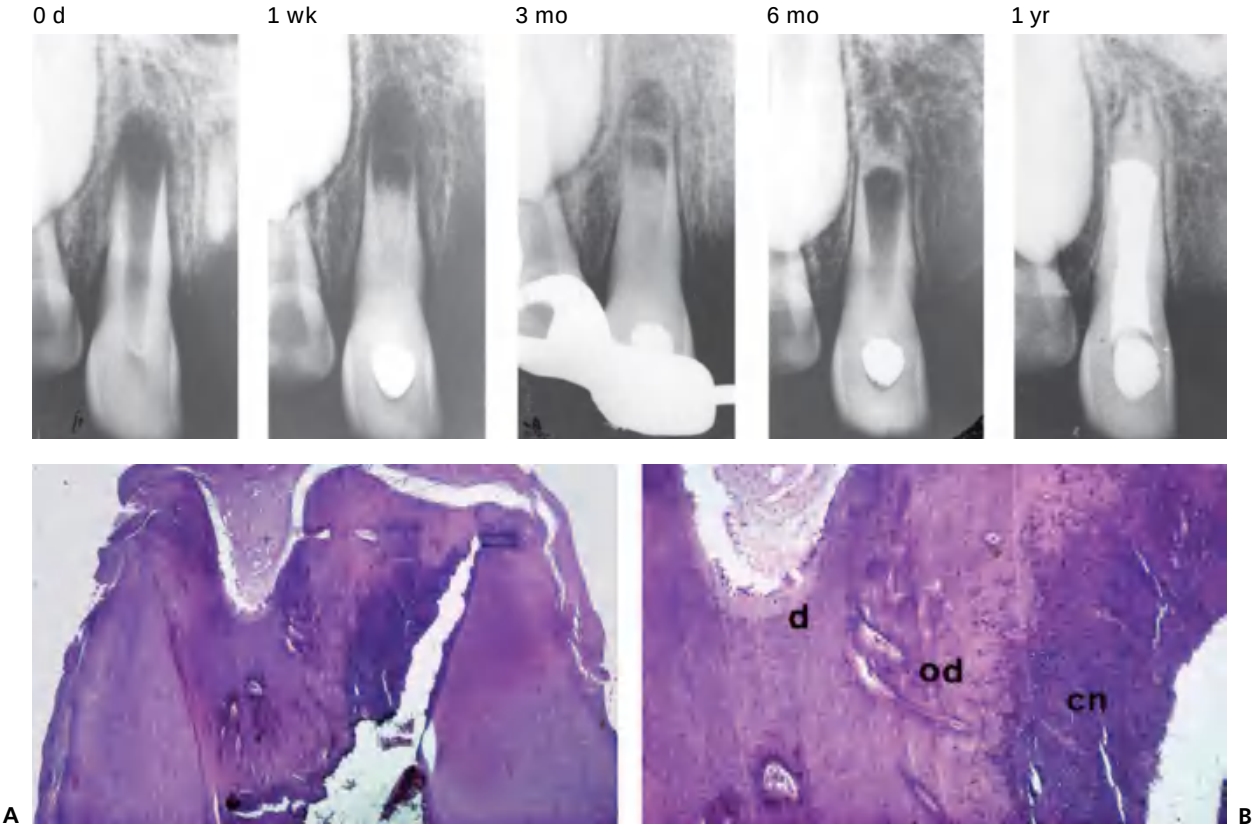



**Fig. 25.47** Continued root development after treatment of an incisor with partial pulp necrosis. Note the level of calcium hydroxide in the root canal, indicating the presence of some viable tissue in the apical portion. Formation of a hard-tissue barrier and continued root development can be seen at review appointments 6 and 12 months after initial treatment. A and B. Histologic section of a similarly treated tooth, extracted due to cervical root fracture. Close to the calcium hydroxide is a darkly stained area of necrotic and later calcified tissue (cn), followed by a layer of bone-like tissue (od), and above it dentin and predentin with lining odontoblasts (d). ×20 and 40.

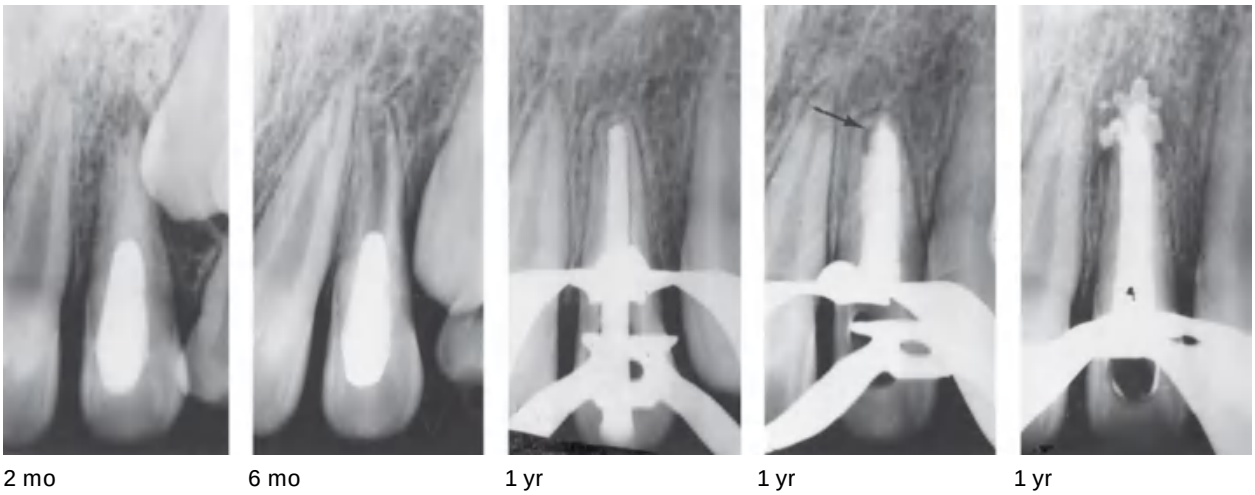



**Fig. 25.48** Disruption of the apical hard tissue barrier due to forceful obturation of the root canal. During filling of the canal with gutta-percha, an attempt was made to fill a void in the root canal (arrow). The pressure applied resulted in the rupture of the thin hard tissue barrier and overfilling.

### Artificial barrier

An artificial barrier can be placed by using a bioceramic material (such as mineral trioxide aggregate) once the root canal system has been adequately disinfected (310) (Fig. 25.54). This technique has been shown to promote periapical repair (234) as well as being a barrier against which the root canal filling can be placed once the MTA has set (Fig. 25.55). This is a technically difficult procedure (Fig. 25.56) and ideally should only be done by experienced clinicians or specialist endodontists with magnification to assist in accurate placement as the risk of material extruding beyond the root is quite high. The material then needs to be

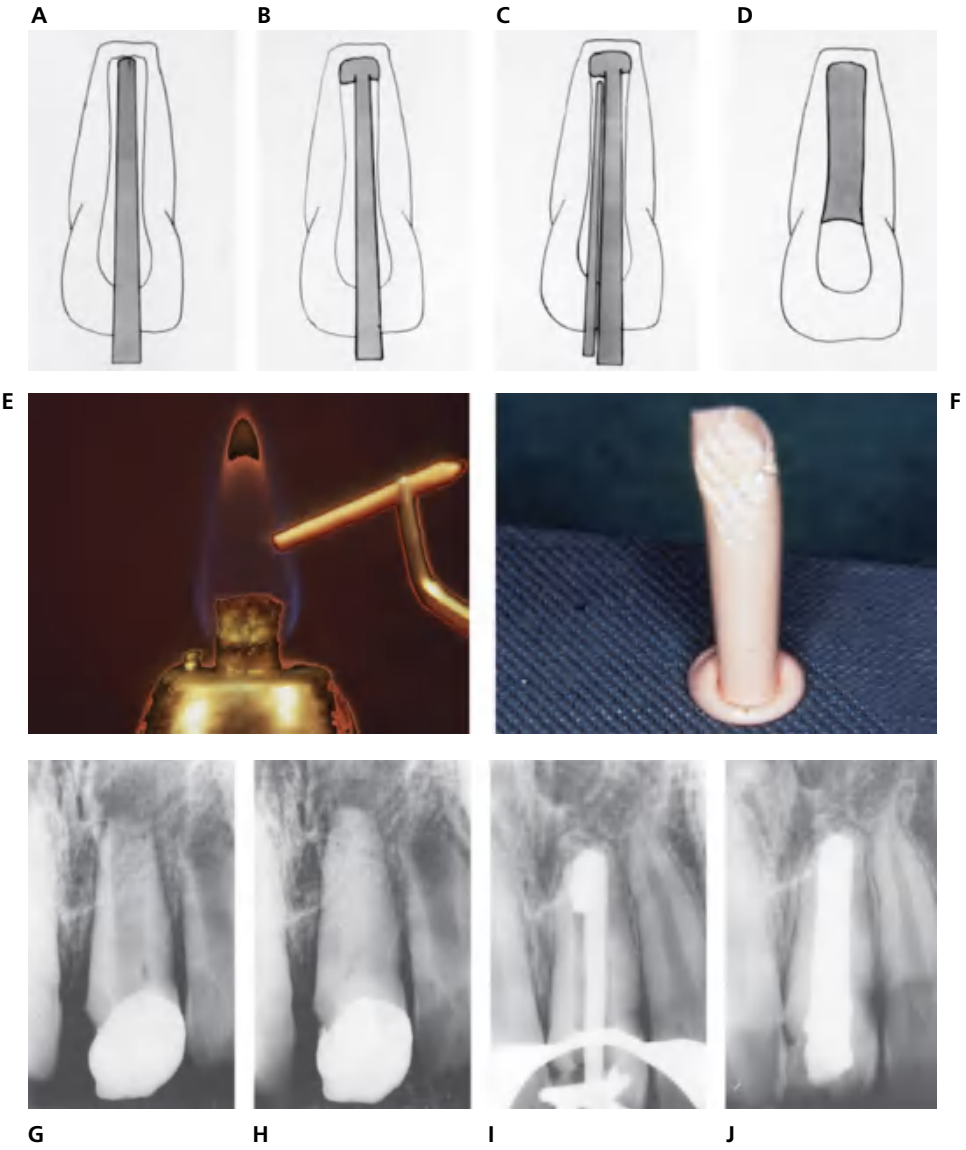



**Fig. 25.49** Clinical procedures for filling the canal with partially heated and softened gutta-percha. A. Selection of a suitable master point. B. The tip of the point is softened by passing it once through ethanol flame and then introduced into the root canal and pressed against the apical barrier. C. Subsequent filling using lateral condensation. D. Completed filling. E. The tip of the point is softened by passing it once through ethanol flame. Only the tip of the point should be softened, and the rest should remain stiff if the desired effect is to be achieved. F. The effect of pressing a partially softened point against a firm surface. G–J. Filling of the root canal in an immature incisor after treatment with calcium hydroxide. The softened tip of a master point was pressed against the hard tissue barrier and the root canal completely filled with additional points using lateral condensation.

left to set hard before the remaining canal can be filled in the routine manner with gutta-percha and cement, usually at another appointment since the setting time of most MTA materials is several hours. This procedure can be completed within a shorter time period with less appointments than apexification, but its main disadvantage arises if root canal re-treatment becomes necessary in the future as it will be very difficult to remove the barrier material which may affect the ability to disinfect the entire root canal system and achieve periapical healing. As mentioned above, this technique does not overcome the problem of thin dentin in immature teeth at the cervical level – hence, subsequent fracture is still a potential problem.

### Endodontic regenerative procedures

Endodontic regenerative procedures are discussed in more detail in Chapter 26. This type of procedure was first reported by Nygaard-Østby in 1961 (235). He tested whether the presence of a blood clot within a root canal system would promote healing. The treated teeth were extracted after various time periods ranging from 17 days to 3.5 years and then they were examined histologically. He reported resolution of clinical signs and symptoms plus radiographic evidence of apical closure. Histological analysis revealed ingrowth of connective tissue into the root canal and varying amounts of mineralized tissue along the walls plus 'islands' of mineralized tissue embedded within the newly formed tissue. There were inclusions of cementoblasts and osteoblasts but there was a lack of odontoblasts and there was no true regeneration of the dental pulp. In a further study in 1971, Nygaard-Østby and Hjortdal (236) found whilst the apical periodontitis healed, there was no evidence of pulp regeneration in the root canal when the teeth had been infected.

Interest in regenerative procedures was reignited in 2001 when Iwaya et al. (237) used a combination of two antibiotics as a root canal medicament in a tooth that had an infected root canal system and a chronic apical abscess. They then induced bleeding into the canal and subsequently reported that follow-up radiographs suggested there had been thickening of the root canal walls and narrowing of the apical

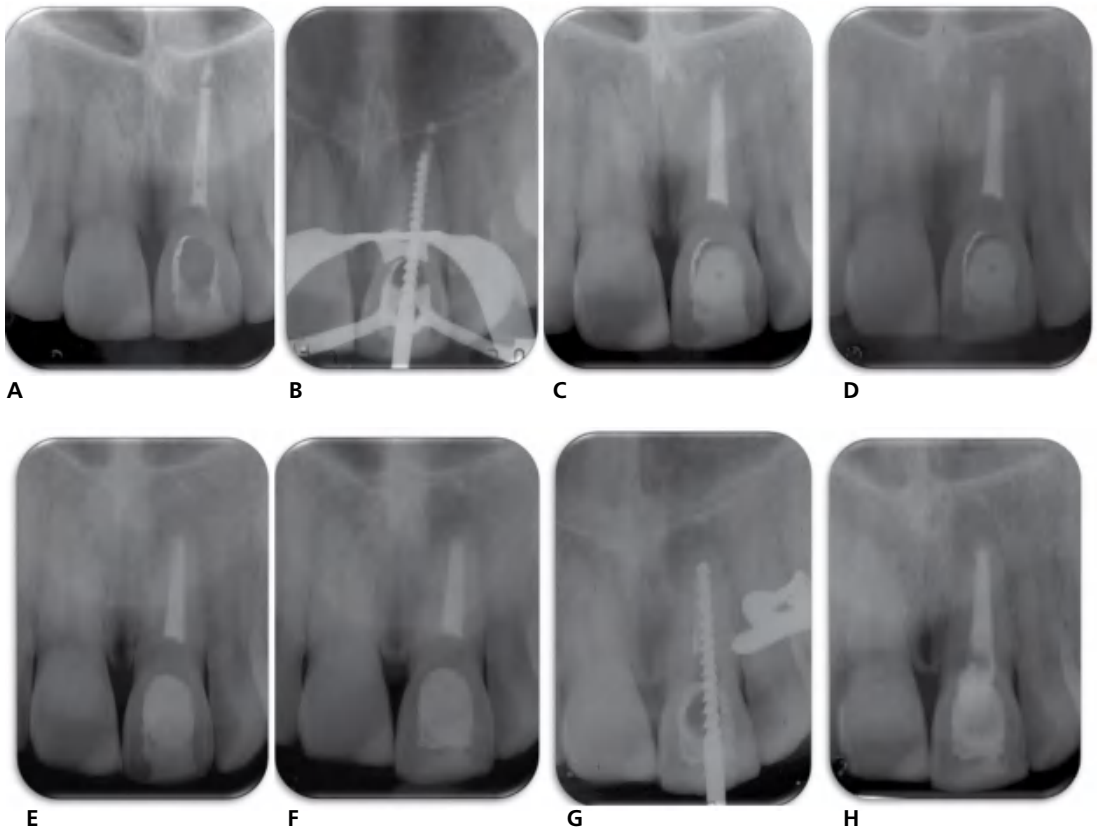



**Fig. 25.50** Root canal re-treatment of a tooth that had previously had apexification with apical hard tissue barrier formation but subsequently became infected again 10 years later. The tooth originally had a crown fracture that led to pulp necrosis and infection approximately two years before again becoming infected. A. Radiograph taken approximately two years following trauma and previous root canal treatment. The patient had symptoms consistent with an infected root canal system and chronic apical periodontitis. Note extrusion of the root filling materials through the open apical foramen. B. Root canal re-treatment was commenced, and calcium hydroxide was used at three-monthly intervals. C. Root canal filling after 15 months when an apical hard tissue barrier was confirmed. Note gradual resorption of the previously extruded root filling material. D. Six-month review radiograph shows good periapical healing. E. A 7-year review suggests that a periapical radiolucency may be developing. F. The patient returned after a further 5 years (i.e. 13 years after apexification and root filling) with acute apical periodontitis. G. Root canal re-treatment was commenced, and the apical hard tissue barrier was still intact. H. After two further dressings with calcium hydroxide, a new root canal filling was placed. Periapical healing is evident radiographically and the apical hard tissue barrier is still intact.

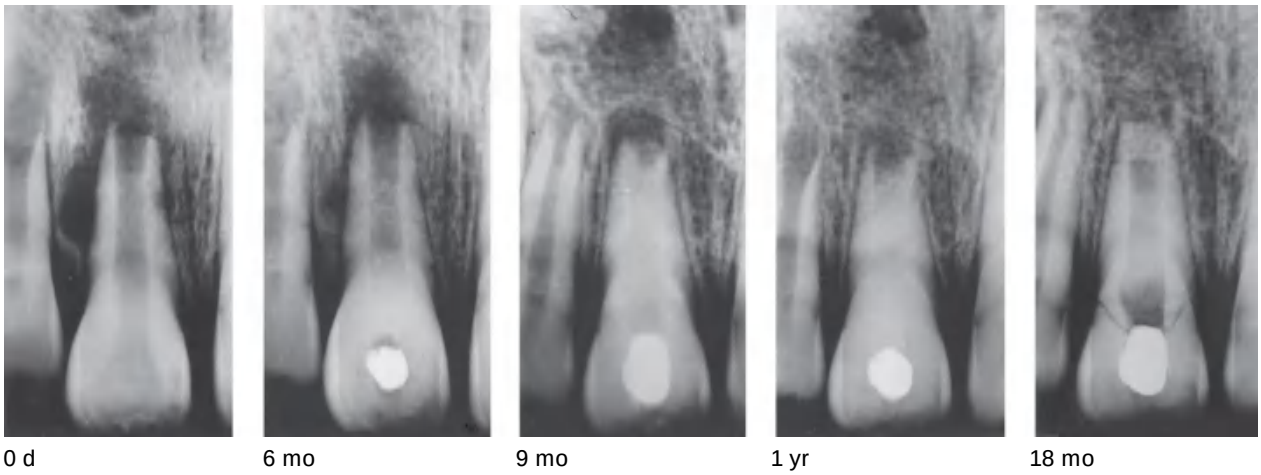
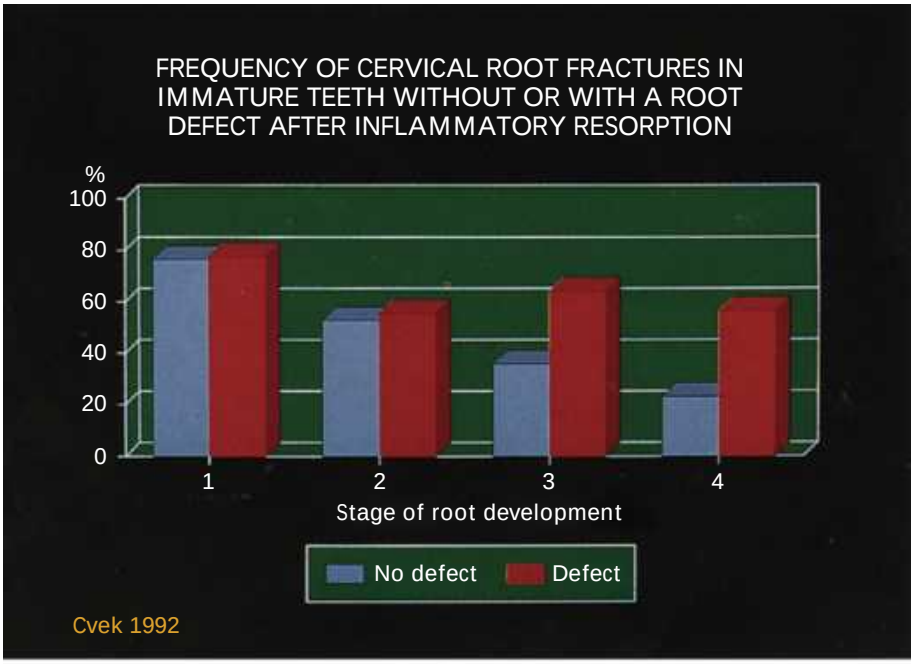



**Fig. 25.51** Cervical root fracture after successful endodontic treatment of an incisor with immature root development. A minor trauma caused the cervical root fracture 18 months after initial treatment.

foramen. The tooth also responded to pulp sensibility tests at the 30-month recall appointment. Banchs and Trope then published a case report in 2004 (238) regarding a mandibular premolar that had an infected root canal system and a chronic apical abscess as a result of a dens evaginatus that had fractured and exposed the pulp. They also reported increased dentin thickness and narrowing of the apical foramen, based on radiographic appearance. Since then, there



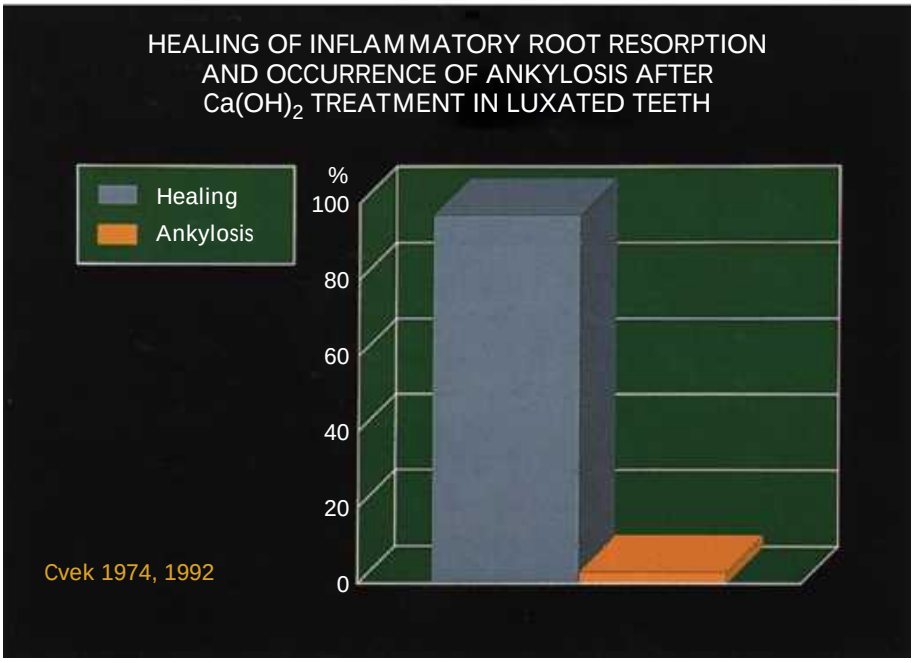




**Fig. 25.52** A. Relationship between cervical root fractures and presence of defects and inflammatory root resorption. Fractures in 397 immature incisors, following calcium hydroxide treatment and an observation period of 4 years after filling with gutta percha. The material is distributed according to the presence of resorption defects in the cervical area of the root as well as the stage of root development. B. Healing of inflammatory root resorption after luxation injuries. The frequency of root resorption arrest and healing as well as the occurrence of ankylosis in 187 luxated permanent incisors, treated with calcium hydroxide. Source: Reproduced from (221) with permission.

have been numerous reports in the literature (239) of similar management of immature teeth with infected root canals – some as a result of dens evaginatus and some following trauma. Whilst this technique shows great promise, the ideal procedure and materials have yet to be determined. Some authors have used a mixture of three antibiotics, some have used two antibiotics, and some have used a single antibiotic as the disinfecting agent. Others have used calcium hydroxide. Timing of appointments also varies as do the indications for such procedures. The key points are to disinfect the canal and to induce bleeding into the canal to create a scaffold for ingrowth of tissue. Preservation of the stem cells that may be present within the periapical tissues is also essential and this may be a limiting factor following trauma to the tooth since the periapical tissues are likely to be damaged – in some injuries, this will be inevitable (e.g. intrusion) whilst in others it may occur in some cases, depending on the severity of the injury (e.g. subluxation, lateral luxation, avulsion). Clinically, it is not possible to determine the extent of damage for any particular tooth and therefore the predictability of these regenerative procedures following trauma is uncertain. Another key factor is the need to place an adequate coronal restoration in order to prevent any further ingress of bacteria. This may be difficult in teeth with concurrent crown fractures or crown-root fractures. In addition, the long-term consequences of breakdown of the coronal restoration have yet to be determined with respect to whether re-treatment is feasible. The use of MTA as a barrier material in the root canal at the cervical level may preclude the ability to, or at least it may make it difficult to, retreat the root canal system. The depth at which the MTA has been placed and the thickness of the barrier

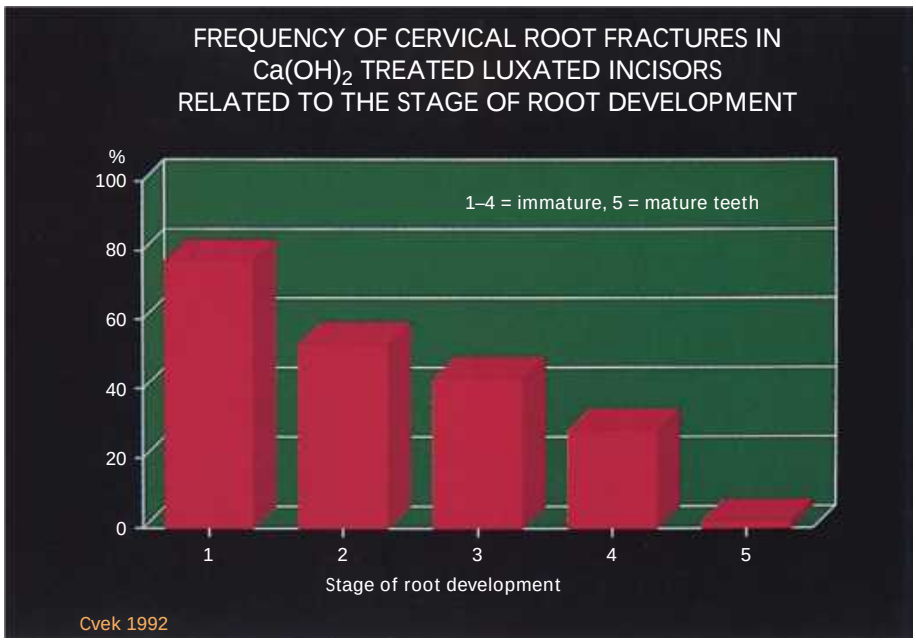



**Fig. 25.53** Frequency of cervical root fractures. Frequencies of cervical fractures in 759 luxated maxillary incisors, after calcium hydroxide treatment and 4 years observation following gutta-percha filling, distributed according to the stage of root development. Source: Reproduced from (230) with permission.

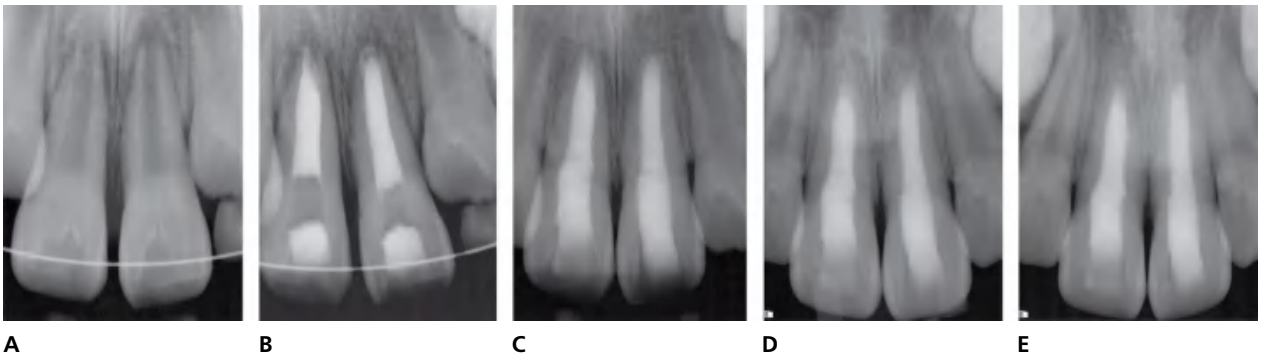



**Fig. 25.54** A. Luxation injury to both maxillary central incisors resulted in pulp necrosis and infection. The necrotic pulps were removed, and calcium hydroxide was placed in the canals. B. One month later, the canals were filled with MTA, covered with wet cotton pellets and a temporary restoration. A few days later, the coronal access cavities were filled with composite resin. C. Six-month follow-up. D. Two-year follow-up. E. Three-year follow-up shows good periapical repair.

are likely to be factors that affect whether it can be removed or not. These aspects have yet to be investigated and reported in the literature.

### Comparison of techniques

In general, the results of regenerative procedures have been quite mixed. Radiographically, many case reports suggest further root development occurs with hard tissue that resembles dentin. However, a downfall of the many case reports is the lack of standard angulations for the radiographic images which makes it impossible to accurately assess further root development. Histological studies of treated teeth that have subsequently been extracted for various reasons (e.g. fracture) have shown that the hard tissue is typically cementum and bone. In addition, there is little histological evidence of true pulp tissue with the soft tissue that forms in the canal being mainly fibrous connective tissue and it often also contains bone (240). One histological study has also reported bone growth into the root canal with ankylosis (i.e. fusion between the bone and the canal wall), probably due to the absence of the protective qualities of the periodontal ligament (241). Hence, ankylosis and internal ankylosis-related resorption may be a long-term outcome of such regenerative procedures.

None of the above three techniques for managing immature teeth with infected root canal systems has been reported to be better than the other techniques with respect to treatment outcome or retention of the tooth as shown by Alobaid et al (242) who investigated 31 teeth over an average of 17 months. All but one tooth from the regeneration group survived and all but four teeth from this same group had a successful outcome. The tooth that did not survive had a further traumatic incident and required extraction as a result. All of the calcium hydroxide apexification group and all of the MTA artificial barrier (apical plug) group survived and were successful. There were more adverse events in the regeneration group (8 of 19 teeth, 42%) compared to the apexification group (1 of 12 teeth, 8%). Interestingly, there

**Fig. 25.55** Apexification using MTA as a root-filling material. The apexification was performed on a dog's tooth and it shows apical cementum (C) developing below the initial osteodentin (OD) which formed under the MTA (MTA – space where the MTA was placed). Masson's trichrome stain. Source: Courtesy of Dr. Shahrokh Shabahang, Loma Linda University, California, USA.

were also no statistical differences between the apexification group and the regeneration group in terms of radiographic changes in width of root wall and the length of the roots. The majority of teeth in both groups had changes in width and length that were less than 20% which the authors assumed to be the threshold for a clinically meaningful change in each entity – overall, only 4 (of 15) of the regeneration cases and 1 (of 15) apexification case met this threshold. Although the number of teeth studied was low, the authors concluded that regeneration was not superior to other apexification techniques which were equivalent to each other.

Another study of the clinical and radiographic outcomes of regenerative procedures in 20 teeth following trauma reported that 90% of the teeth had radiographic evidence of closure of the apical foramen after 12 months. The in-case change of root length averaged only 5% while the in-case change of root thickness averaged 21% (243). None of the teeth responded to cold, heat and electric pulp sensibility tests after 12 months. The authors stated that many of the radiographic changes were not be discernible by visual examination and they were only detected and quantified by using computer software to measure the radiographic changes. When the data presented in the graphs in Figure 2 of this paper is further analyzed, the median change in root length was only about 1.0 mm, the change in median root wall width was about 0.54 mm and the original apical opening was only about 1.2 mm. Hence, the actual changes are very minor and it would seem unlikely that these will increase the strength of the tooth roots.

As discussed above, none of these three approaches to managing immature teeth with infected root canal systems addresses the problem of the lack of dentin in the cervical part of the tooth where fractures have been reported. Hence, whilst the possibility of being able to truly regenerate pulp tissue is exciting, and some further root growth may occur, there is still much research to be done to determine whether pulp can truly be regenerated as well as the best and most predictable way to achieve this. In addition, a most important aim should be to develop a treatment modality that actually addresses the problem of susceptibility to cervical fracture. No studies have shown any clinical advantage from obtaining a small increase in root length or a small increase in the thickness of the root wall in the apical part of the tooth root. It is unlikely that the new hard tissue (i.e. bone and cementum) that forms in the apical part of the root could contribute to the strength of the cervical part of the tooth to render it more resistant to the impact forces that occur during subsequent trauma. However, research is required to determine whether any such effects occur.

### *Endodontic management of fully developed teeth*

Root canal treatment of fully developed teeth with pulp necrosis and infection as a result of trauma is the same as for teeth that develop pulp necrosis and infection of the root canal system as a result of other causes (e.g. caries, cracks, restorations breaking down, etc.). Commonly accepted endodontic instruments, procedures, medications and root filling materials can be used (244, 245). High frequencies of healing have been reported if the root canals have been dressed with calcium hydroxide prior to filling with gutta-percha and cement (230, 246–249). Medicating the root canal system with calcium hydroxide before completing the root canal filling is therefore recommended for routine use in teeth with infected root canal systems and apical periodontitis following trauma (Fig. 25.57).

The main purpose of the calcium hydroxide is to disinfect the root canal system which occurs through its anti-bacterial properties as a result of its high pH of 12.5 (250–266). An in vitro study (259) reported that 99.9% of bacteria commonly isolated from root canal flora were killed within a few minutes upon direct contact with calcium hydroxide with *Enterococcus faecalis* being the only resistant strain. However, in clinical cases, *E. faecalis* is not a typical commensal organism in teeth with primary root canal infections, such as would occur following trauma to a tooth. *E. faecalis* may be present in some teeth that have been root-filled previously, which suggests that it is more likely to be a contaminant or opportunistic organism.

The use of calcium hydroxide leads to predictable healing of the peri-radicular tissues, including arrest of infection-related root resorption and formation of a hard tissue barrier apically, against which an adequate root filling can be placed (as discussed above). In clinical and experimental studies, this has been shown to occur with high predictability (Fig. 25.58). Infection-related resorption can however in some cases change to ankylosis-related resorption (Fig. 25.59).

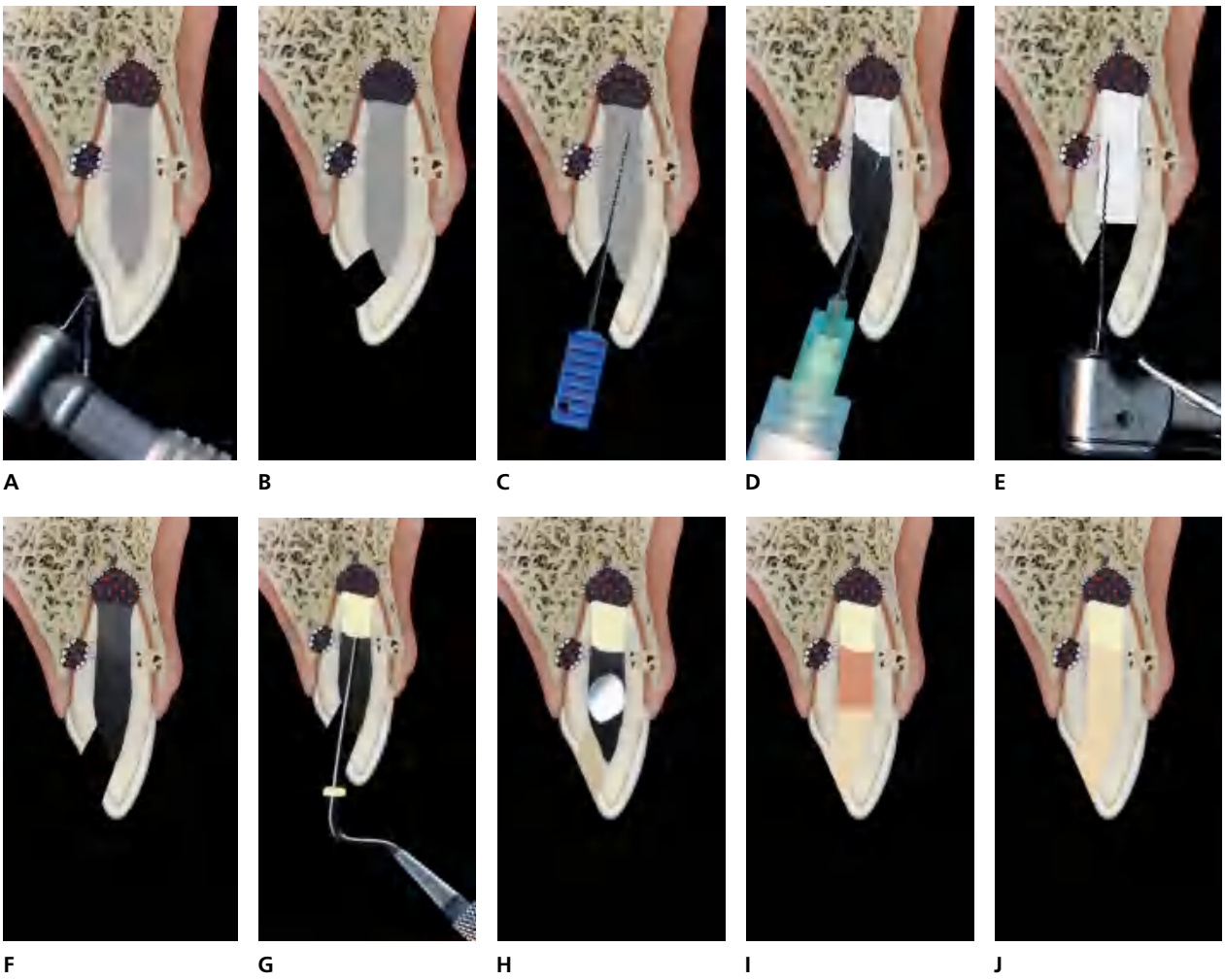
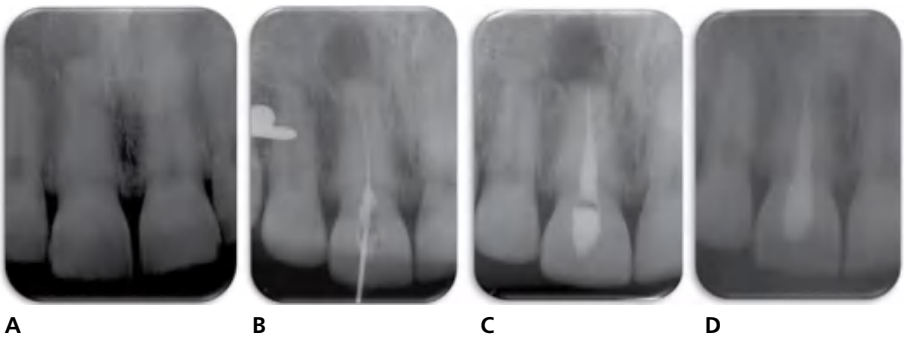



**Fig. 25.56** Artificial barrier technique with MTA. A and B. The tooth is anesthetized (if needed) and isolated with a rubber dam. Access is obtained to the root canal space in the usual manner. C. Carefully remove necrotic tissue to the level at which bleeding is first encountered (but not to exceed the length of the root canal). D. Limit the amount of filing within the canal because of the thin walls but thoroughly irrigate the canal with sodium hypochlorite. E. Medication with calcium hydroxide and then place a cotton pellet and a temporary restoration F. At the next visit, isolate with rubber dam and re-enter the root canal. Gently remove the calcium hydroxide by irrigating the canal and then dry it. G. Place a small amount of the mixed MTA into the coronal chamber and use pluggers considerably smaller than the root canal diameter to carefully push the material towards the apical foramen. An ultrasonic device can be used against the plugger to assist in compacting the MTA into the apical location. H. Prior to placing the MTA, a decision must be made about how much of the canal is to be filled with MTA. One can place an artificial barrier ('apical plug') of MTA (at least 4 mm) and fill the rest of the canal with gutta-percha (after the MTA has set), or fill the entire canal with MTA. With both approaches, the MTA must be left to set (a minimum of 4–6 hours) before completing the treatment. During the setting period, a moist cotton pellet should be placed coronal to the MTA and the access cavity should be closed with a temporary filling. I. After the MTA has set, the coronal part of the root canal can be filled with gutta-percha and cement (if it was not completely filled with MTA). J. The coronal access cavity can be restored with composite resin and review appointments should be arranged.

**Fig. 25.57** Root canal treatment of a fully developed tooth as a result of pulp necrosis and infection with an acute apical abscess that developed several years after lateral luxation. Routine treatment was provided with calcium hydroxide as an intracanal medicament before placing the root canal filling with gutta-percha and cement. A. Pre-operative radiograph. B. Working length radiograph. C. Immediate post-operative radiograph. D. The radiograph taken 18-months after treatment shows good periapical healing.



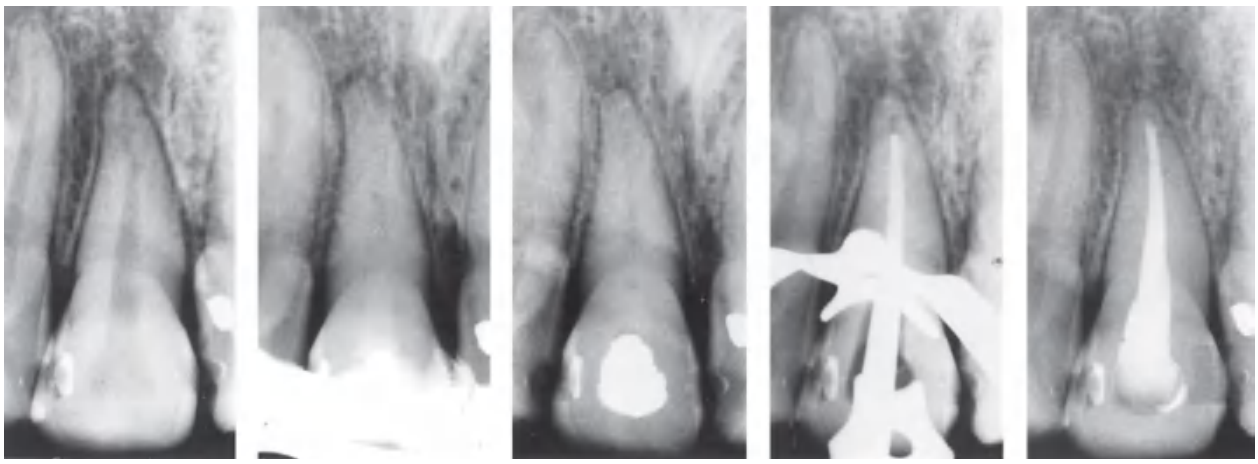



**Fig. 25.58** Treatment of a mature permanent incisor with calcium hydroxide. Periapical healing and closure apically with hard tissue was found after 6 months and the root canal was filled with gutta-percha. Periapical healing is seen at a 4-year control.

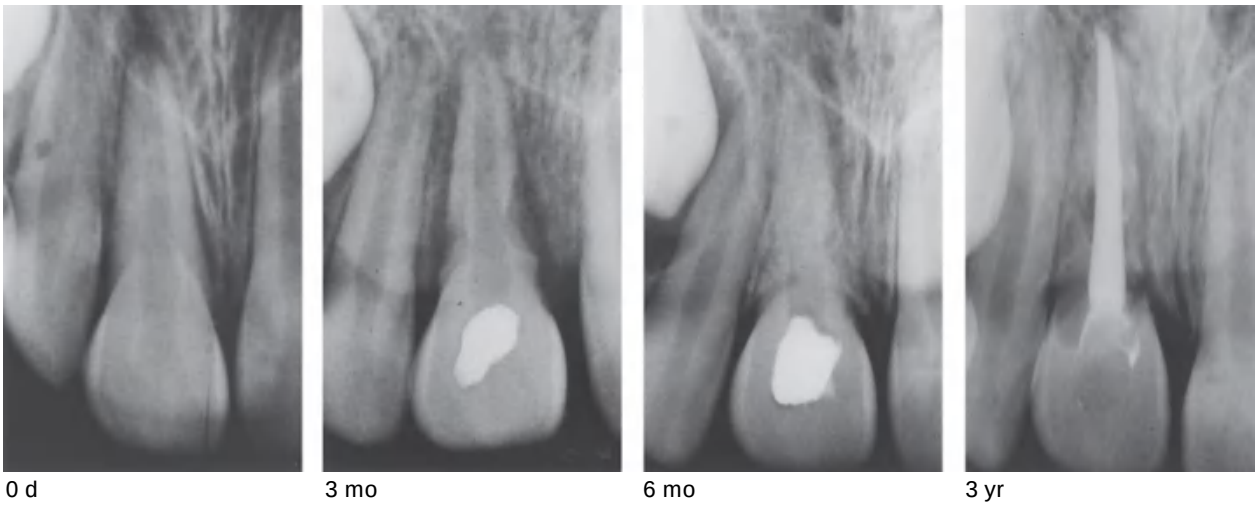



**Fig. 25.59** Change of inflammatory root resorption into ankylosis after treatment with calcium hydroxide. The tooth was replanted after 60min dry storage. Three months after replantation there is inflammatory root resorption with associated radiolucency in the adjacent alveolar bone and the tooth was treated with calcium hydroxide. Six months after treatment there is periradicular healing with development of replacement resorption, i.e. dentoalveolar ankylosis.

### *Management of external infection-related root resorption*

One of the unfavorable responses to trauma is the development of external infection-related root resorption, also known as inflammatory resorption (Figs 25.60 and 25.61). This condition requires the root canal system to be infected as well as damage to the cementum and periodontal ligament, as discussed above. There are two approaches to managing external infection-related resorption, namely a preventive approach and an interceptive approach – these have been described in detail by Abbott (221).

The preventive approach involves the commencement of root canal treatment immediately after the involved tooth has been repositioned/replanted and stabilised – that is, commence treatment on the same day as the trauma (Fig. 25.62). This approach applies to teeth that have a high degree of susceptibility to developing external infection-related resorption because their pulps are highly unlikely to survive, bacterial contamination of the root canal system is likely and there has been damage to the root surface and periodontal ligament (Table 25.1). The principle behind this approach is to remove the infarcted pulp tissue, clean the root canal and place anti-inflammatory and anti-bacterial agents into the canal in the form of a corticosteroid-antibiotic medicament (see Chapter 8). To date, studies have shown that the use of a tetracycline in the combination medicament contributes to the prevention of resorption through the tetracycline's ability to inhibit clastic cells. Commencing this treatment immediately following stabilization of the tooth is recommended in an attempt to reduce the intensity of the inflammatory response that will inevitably

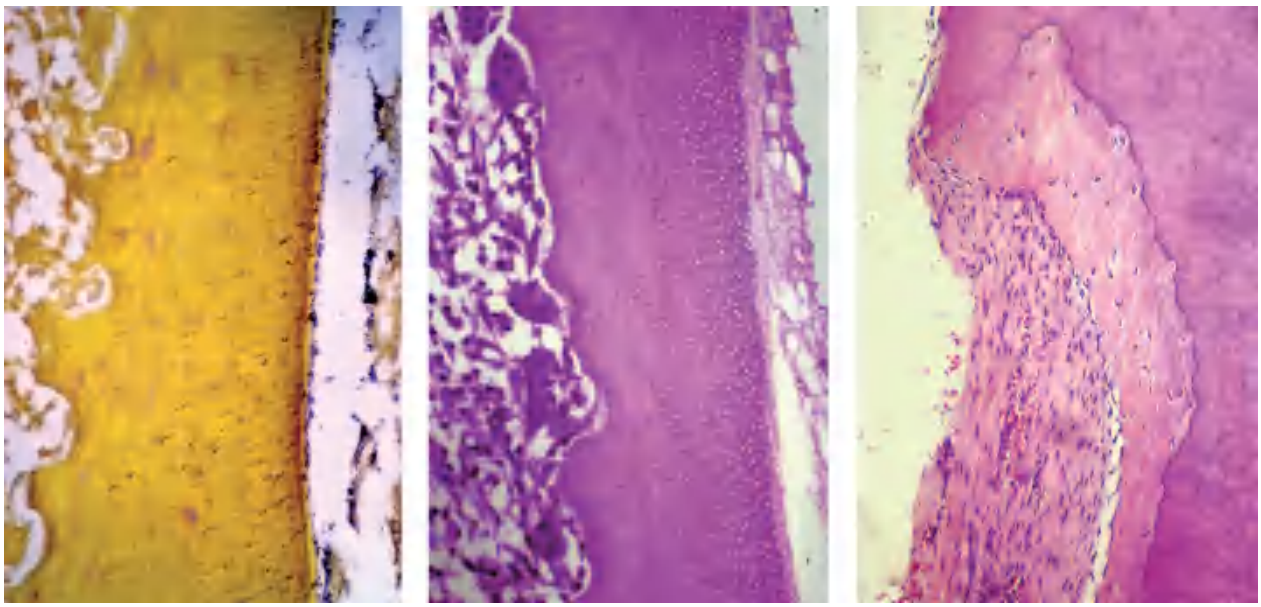



**Fig. 25.60** Histological appearance of external inflammatory root resorption (also known as infection-related resorption). A. Penetration of bacteria through dentinal tubules, from the infected root canal towards the periodontium, Brown & Brenn bacterial stain. ×100. B. Bacteria and their toxins cause accumulation of leukocytes. As the leukocytes cannot reach the bacteria in the tubules, osteoclasts are stimulated to resorb dentin. ×100. C. If bacteria are removed from the root canal, resorption lesions can be repaired by cementum-like tissue. ×80.

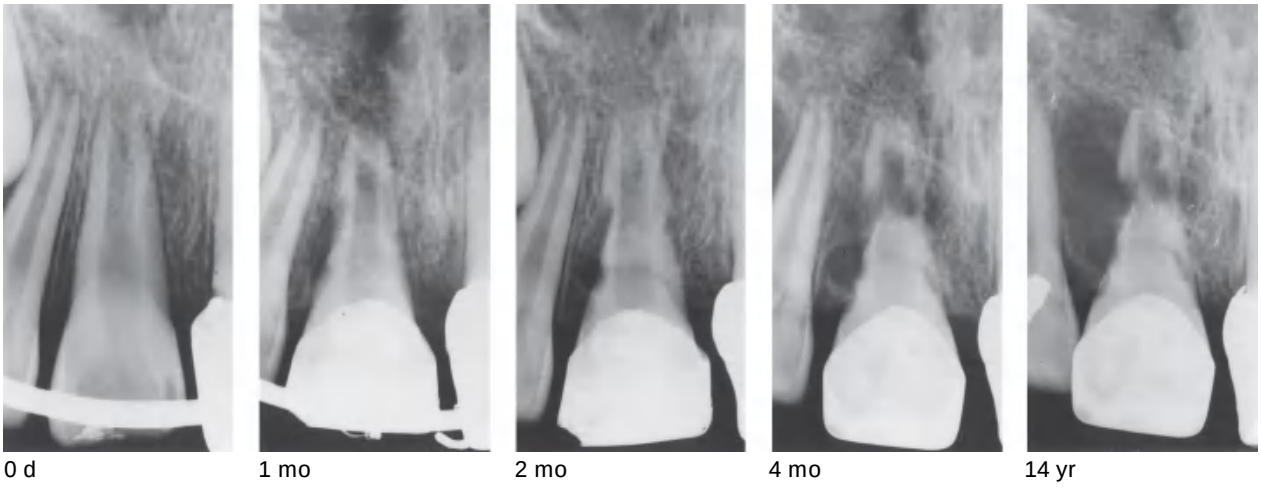



**Fig. 25.61** Radiographic appearance of external inflammatory root resorption. Since the tooth has not been endodontically-treated, there has been progressive loss of root substance associated with an increasing number and size of the radiolucencies in the adjacent alveolar bone.

occur in the peri-radicular tissues as a result of the trauma itself plus the repositioning/replantation procedure. If the inflammatory response is minimized, then resorption is less likely to occur. Various studies in dogs have shown this approach to be very effective and it may even slow the development of ankylosis-related resorption to some extent (267). Systemic antibiotics are also recommended to help prevent the development of external infection-related resorption (268), especially tetracyclines as they have some anti-resorptive activity (269), although penicillin can also be effective.

The second approach for external infection-related resorption is the interceptive approach which applies to teeth where this type of resorption has already commenced (Figs 25.63–25.67). The principles are very similar to the preventive approach although systemic antibiotics are not required, and long-term use of calcium hydroxide is usually necessary to encourage hard tissue repair of the resorptive defect. Abbott (221) has reported a very high success rate using these two approaches – 100% of the teeth managed with the preventive approach were successfully treated and the resorption was arrested with hard tissue repair following in 97% of teeth managed with the interceptive approach.

The aims and procedures for the above management approaches are summarized in Tables 25.2–25.5.

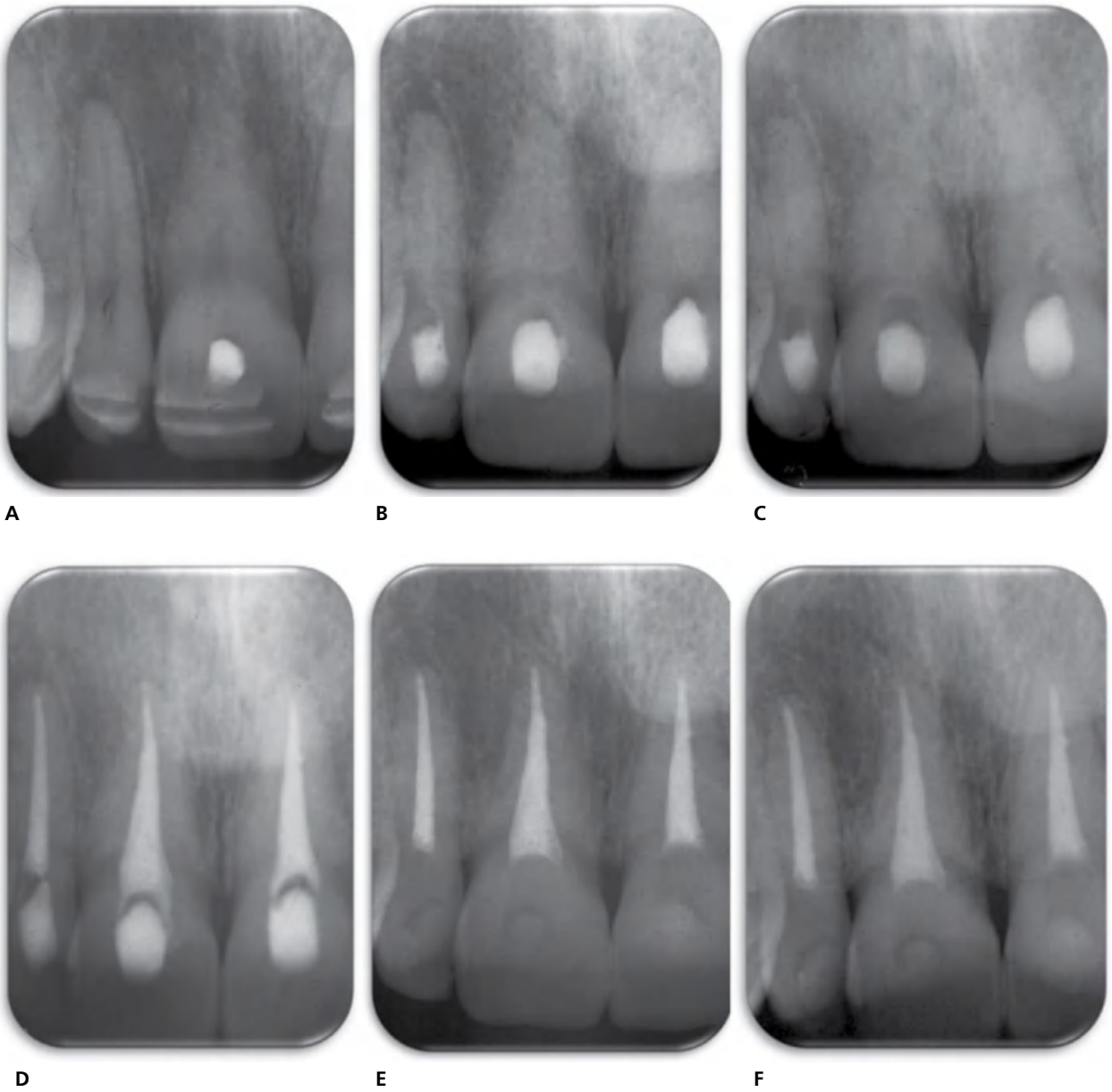



**Fig. 25.62** Preventive management of an avulsed tooth to avoid external infection related resorption. The upper right central incisor was avulsed and replanted within 15 minutes by the patient. The patient was seen by a dentist soon after – the dentist splinted the teeth and commenced root canal treatment immediately. Ledermix® paste was placed in the root canal system. A. Periapical radiograph one week after the accident. B. Radiograph taken three months after the accident and after two dressings of Ledermix® paste for 6 weeks each – no resorption is evident. Note: the adjacent teeth have had root canal treatment commenced because of pulp necrosis and infection and acute apical periodontitis. C. Calcium hydroxide was then used in the canal. Radiograph taken 6 months after the accident. D. The root canal filling was completed eight months after the accident. E. Two and a half years after the accident, no resorption is evident. F. Five years following the accident, there is no evidence of external infection related resorption. Some ankylosis is evident on the distal surface of the coronal third of the root. Source: Reproduced from (221) with permission.

### *Management of teeth with pulp canal obliteration*

As discussed above, pulp canal obliteration may develop in some teeth that have had trauma. Pulp canal obliteration is relatively common after luxation injuries and root fractures. The obliteration is an indication that the pulp survived the accident, or in some cases it indicates that the pulp revascularized as part of its recovery process. The obliteration process is a progressive one which can be seen radiographically as a narrowing of the pulp chamber and root canal. Clinically, there may be some yellow discoloration of the crown (Fig. 25.20 F) but the patient typically has no symptoms. If symptoms do occur, they are not a result of the obliteration process but they occur because the pulp has become inflamed or it has necrosed and become infected. Such events are usually a result of bacterial ingress via caries, cracks or restorations breaking down and typically occur a considerable time after the original trauma.

The hard tissue that forms in the root canal varies histologically depending on the origin and differentiation of cells

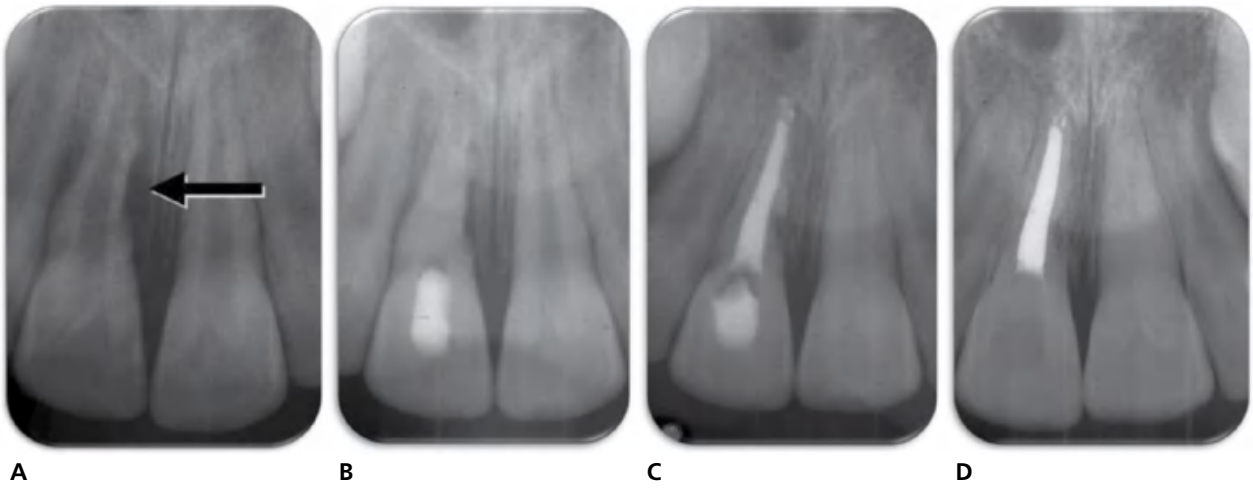



**Fig. 25.63** Interceptive management of a tooth with external infection related resorption. A. Radiograph showing external lateral infection related resorption on the mesial surface (indicated by the arrow) of the root of the upper right central incisor – this was evident six months following avulsion and replantation of the tooth. B. Periapical radiograph taken after Ledermix paste had been used as a root canal medicament for two periods of six weeks each (i.e. three months total). The resorption has ceased and repair of the PDL and adjacent bone is evident. A 50:50 mixture of Ledermix paste and calcium hydroxide was used for two months, followed by 3 dressings with calcium hydroxide alone. C. Periapical radiograph taken following completion of the root canal filling after 14 months of intracanal dressings. There is no sign of any further resorption and there has been good PDL and bone repair. D. A four-year follow-up radiograph shows no ongoing resorption. Source: Reproduced from (221) with permission.

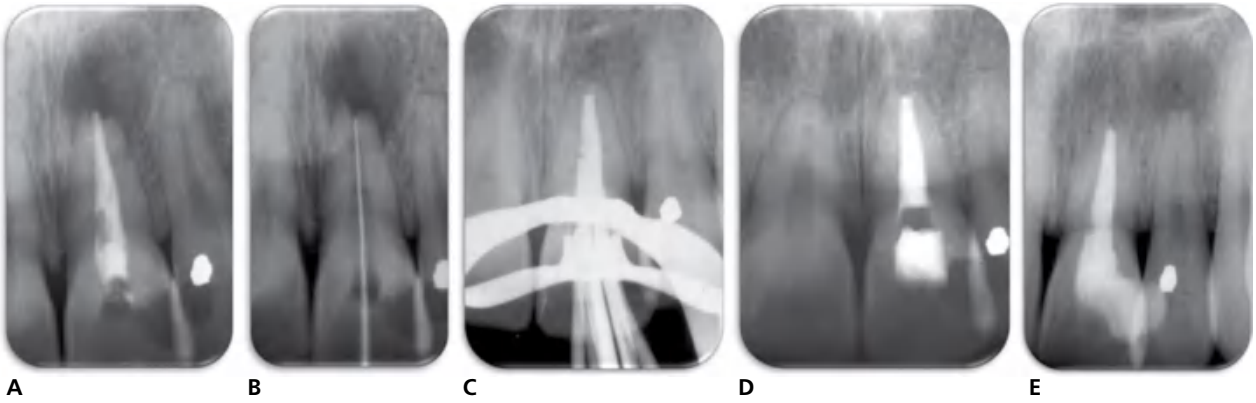



**Fig. 25.64** Interceptive management of established external infection related resorption of the upper left central incisor. This tooth had been traumatised when the patient was 12 years old. Root canal treatment and a composite restoration were done soon after that accident. Ten years later the restoration had broken down leading to an infected root canal system with chronic apical periodontitis and external apical infection related resorption. A. Pre-operative periapical radiograph. B. 'Working length' radiograph showing the extent of the resorption. C. Radiograph taken after filling the apical third of the root canal with a customised gutta-percha point and cement – after 15 months of intracanal medication according to the protocol outlined in Table 25.5. The apical hard tissue barrier is clearly evident. D. Post-operative radiograph on completion of the root canal filling. E. Review radiograph taken 4 years after completing the root canal filling. Note: the left lateral incisor now has a pulpless, infected root canal system with chronic apical periodontitis and apical infection related resorption as a result of breakdown of the composite restoration – this tooth now requires the same treatment as the central incisor has had. Source: Reproduced from (221) with permission.

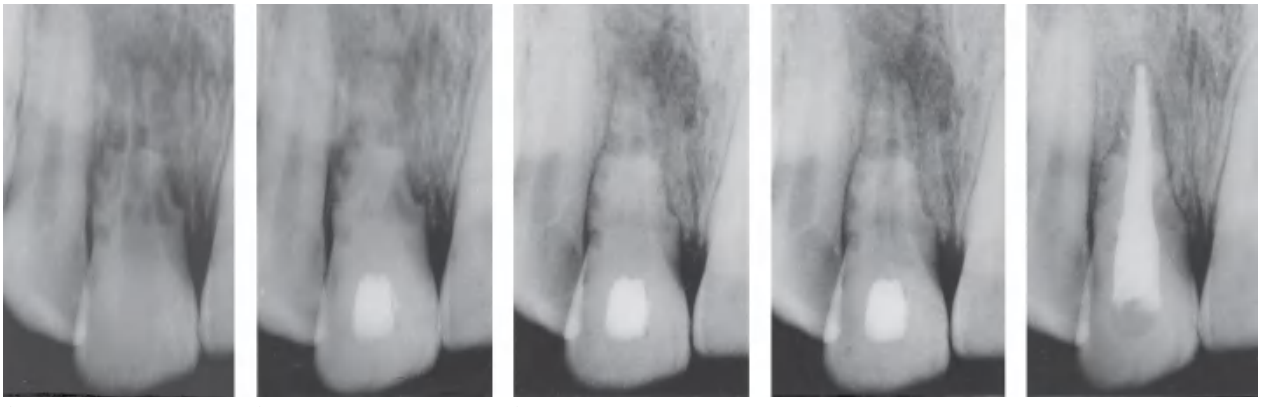



**Fig. 25.65** Treatment of infection related root resorption. Six months after luxation extensive infection related resorption cavities are seen and, in some places, only very thin dentinal walls seem to have remained. The root canal was filled with calcium hydroxide. After 9 months, there is arrest of resorption and periodontal healing. At the 1-year control of the gutta percha filling, deposition of hard tissue into the resorption cavities is evident.

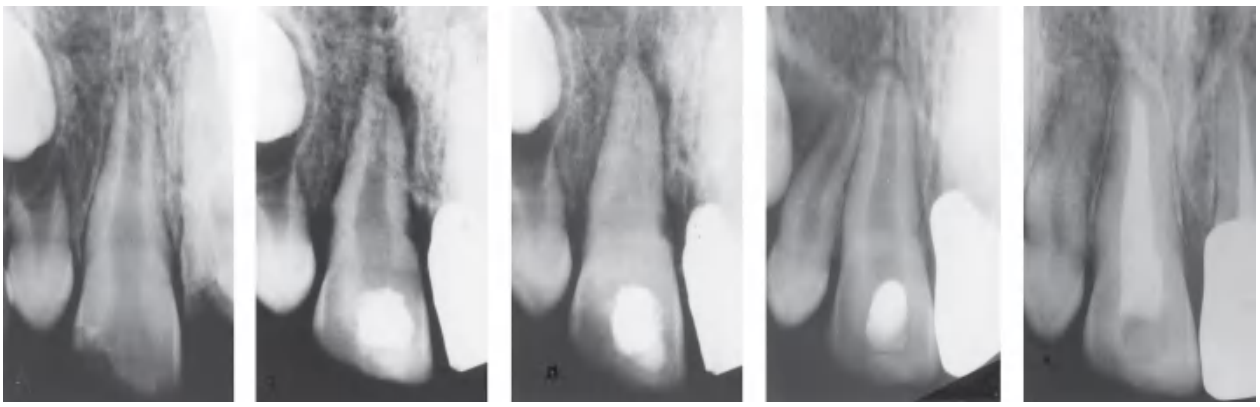



**Fig. 25.66** Treatment of infection related root resorption apparent 6 weeks after replantation. Two years after filling with calcium hydroxide, there is periradicular healing and arrest of root resorption. At a 4-year control, there is repair of resorption lesions.

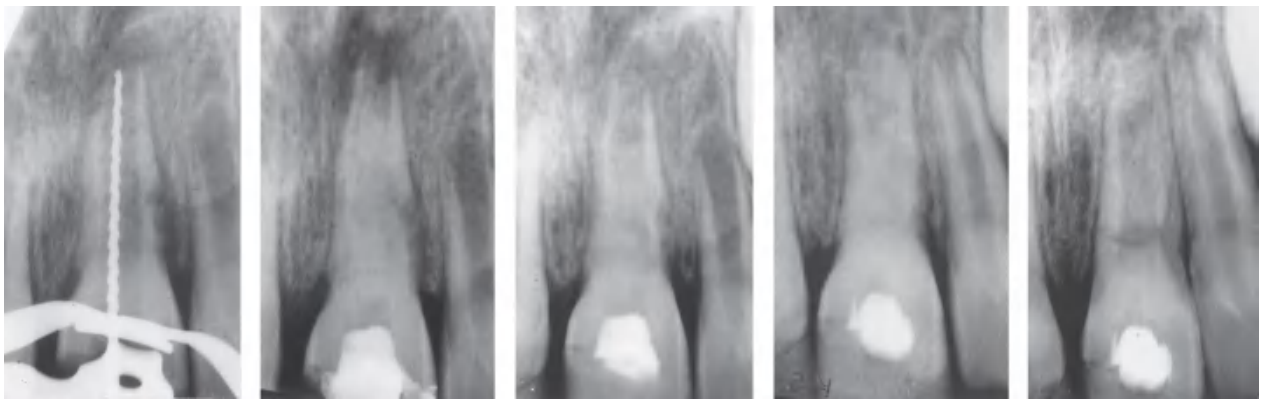



**Fig. 25.67** Arrest of infection related root resorption after calcium hydroxide treatment. After 18 months there is periapical healing and arrest of root resorption. Six months later, a cervical root fracture has occurred through the resorption defect. Source: Reproduced from (221) with permission.

**Table 25.2** Summary of the indications and aims of the preventive and interceptive approaches to managing external inflammatory root resorption.

| Preventive management | Interceptive management |
|---|---|
| **Indications:**<br>• Immediately following the injuries listed in Table 25.1 – to reduce the chances of external inflammatory resorption developing | **Indications:**<br>• Teeth where external inflammatory resorption is already occurring |
| **Aims** – to:<br>• Reduce PDL inflammation<br>• Inhibit clastic cells<br>• Stop bacteria entering the root canal system<br>• Kill any bacteria that have entered the root canal system during the injury/repositioning | **Aims** – to:<br>• Reduce PDL inflammation<br>• Inhibit clastic cells<br>• Kill all bacteria in the root canal system<br>• Encourage healing of PDL and hard tissue repair of the peri-radicular tissues |

Source: Reproduced from (221) with permission.

involved in its production (see Chapter 2). There may be ingrowth of bone and periodontal tissue into the root canal, usually after pulp revascularization in replanted or luxated teeth (Fig. 25.68) or the root canal may be diminished by excessive, irregular hard tissue formation (Fig. 25.69). However, these changes are only seen in young teeth with incomplete root formation, resulting in cessation of further root development. In these teeth neither prophylactic nor therapeutic root canal treatment are recommended because of mechanical problems as the irregular arrangement of hard tissue prevents effective canal cleansing and disinfection.

In most teeth with pulp canal obliteration, the new hard tissue is deposited regularly along the root canal walls and the canal space diminishes gradually until only a very narrow root canal remains – the resultant canal may or may not be seen radiographically (Fig. 25.70). In such cases of

**Table 25.3** – Recommended preventive treatment protocol for incompletely developed teeth to prevent external inflammatory root resorption after suffering the injuries listed in Table 25.1.

| **Incompletely developed teeth – preventive management** |
|---|
| • Systemic antibiotics – start IMMEDIATELY<br>– Check the patient's age, weight, allergies etc. to determine appropriate systemic antibiotic and doses<br>– Tetracycline preferred - e.g. doxycycline 100 mg – 2 tablets on the 1st day, and then 1 tablet daily for 1 week<br>– Alternatively use phenoxymethyl penicillin 1000 mg immediately and then 500 mg every 6 hours (1 hour before meals) for 1 week<br>– Or use amoxicillin 1000 mg immediately, then 500 mg every 8 hours for 1 week<br>• IMMEDIATELY after replantation/repositioning and stabilisation with a splint<br>– Remove the pulp, clean the root canal – if time: measure, file, irrigate, dry<br>– Place a CS-AB paste intracanal dressing, e.g. Ledermix paste<br>• After 4 weeks – complete canal preparation, place a new CS-AB paste intracanal dressing<br>• After another 4 weeks – place a new CS-AB paste intracanal dressing<br>• After another 4 weeks – take a periapical radiograph<br>– If no inflammatory resorption evident – place a new intracanal dressing using a 50:50 mixture of CS-AB & $Ca(OH)_2$<br>• After 2–3 months - take a periapical radiograph<br>– If no inflammatory resorption evident – place a $Ca(OH)_2$ intracanal dressing to induce formation of an apical hard tissue barrier<br>• Change the $Ca(OH)_2$ intracanal dressing every 3 months until hard tissue repair (e.g. apexification) is evident. Periapical radiograph every 6–9 months to ensure healing<br>• Place the root canal filling using gutta percha and cement<br>• Perform internal bleaching if required and then restore the access cavity<br>• Arrange to review after 6 months and then annually for at least 5 years. |

NOTES: 1) The root canal treatment must be commenced as soon as the tooth has been replanted/repositioned and stabilized with a splint. 2) CS-AB = corticosteroid/antibiotic. 3) Working length and canal preparation can be deferred until the second appointment if insufficient time available at the emergency appointment. 4) Rubber dam must be used for all endodontic procedures.
Source: Reproduced from (221) with permission.

**Table 25.4** Recommended preventive treatment protocol for fully developed teeth to prevent external inflammatory root resorption after suffering the injuries listed in Table 25.1.

| **Fully developed teeth – preventive management** |
|---|
| • Systemic antibiotics – start IMMEDIATELY<br>– Check the patient's age, weight, allergies etc. to determine appropriate systemic antibiotic and doses<br>– Tetracycline preferred - e.g. doxycycline 100 mg – 2 tablets on the 1st day, and then 1 tablet daily for 1 week<br>– If allergy to tetracycline, use phenoxymethyl penicillin 1000 mg immediately and then 500 mg every 6 hours (1 hour before meals) for 1 week<br>– Another alternative if allergy to tetracycline: use amoxicillin 1000 mg immediately, then 500 mg every 8 hours for 1 week<br>• IMMEDIATELY after replantation/repositioning and stabilisation with a splint<br>– Remove the pulp, clean the root canal – if time: measure, file, irrigate, dry<br>– Place a CS-AB paste intracanal dressing, e.g. Ledermix paste<br>• After 6 weeks – complete canal preparation, place a new CS-AB paste intracanal dressing<br>• After another 6 weeks – take a periapical radiograph<br>– If no inflammatory resorption evident – place an intracanal dressing using a 50:50 mixture of CS-AB & $Ca(OH)_2$<br>• After 2–3 months – take a periapical radiograph<br>– If no inflammatory resorption evident – place the root canal filling using gutta percha and cement<br>• Perform internal bleaching if required and then restore the access cavity<br>• Arrange to review after 6 months and then annually for at least 5 years. |

NOTES: 1) The root canal treatment must be commenced as soon as the tooth has been replanted/repositioned and stabilized with a splint. 2) CS-AB = corticosteroid/antibiotic. 3) Working length and canal preparation can be deferred until the second appointment if insufficient time available at the emergency appointment. 4) Rubber dam must be used for all endodontic procedures.
Source: Reproduced from (221) with permission.

**Table 25.5** – Recommended interceptive treatment protocol for teeth where external inflammatory root resorption is occurring. This protocol applies to both permanent and primary teeth. From ABBOTT (221) 2016.

| **Interceptive management** |
|---|
| • Remove all restorations, caries and cracks from the tooth; ensure the tooth is suitable for root canal treatment and restoration.<br>– If unsuitable for restoration, extract the tooth<br>• If suitable for treatment, gain access to the root canal system<br>• Negotiate the root canal, measure working length and chemo-mechanically prepare the root canal; irrigate thoroughly and dry the canal<br>• Place a CS-AB paste intracanal dressing, e.g. Ledermix paste<br>• After 6 weeks – place a new CS-AB paste intracanal dressing<br>• After another 6 weeks – take a periapical radiograph<br>– If the inflammatory resorption has not progressed – place a new intracanal dressing using a 50:50 mixture of CS-AB & $Ca(OH)_2$<br>• After 2–3 months – take a periapical radiograph<br>– If the inflammatory resorption has not progressed – place a $Ca(OH)_2$ intracanal dressing – to induce hard tissue repair<br>• Change the $Ca(OH)_2$ intracanal dressing every 3 months until hard tissue repair of the resorptive lesion is evident. Take a periapical radiograph every 6–9 months to monitor healing and to determine when the root canal filling can be completed<br>• Once hard tissue repair is evident, place the root canal filling using gutta percha and cement<br>• Perform internal bleaching if required and then restore the access cavity<br>• Arrange to review after 6 months and then annually for at least 5 years. |

NOTES: 1) CS-AB = corticosteroid/antibiotic. 2) Rubber dam must be used for all endodontic procedures. 3) Systemic antibiotics are NOT indicated, unless the patient has systemic signs (i.e. malaise, increased body temperature, lymph node involvement, rapid spreading infection, etc.) of a systemic infection associated with the tooth (e.g. secondary acute apical abscess or facial cellulitis).

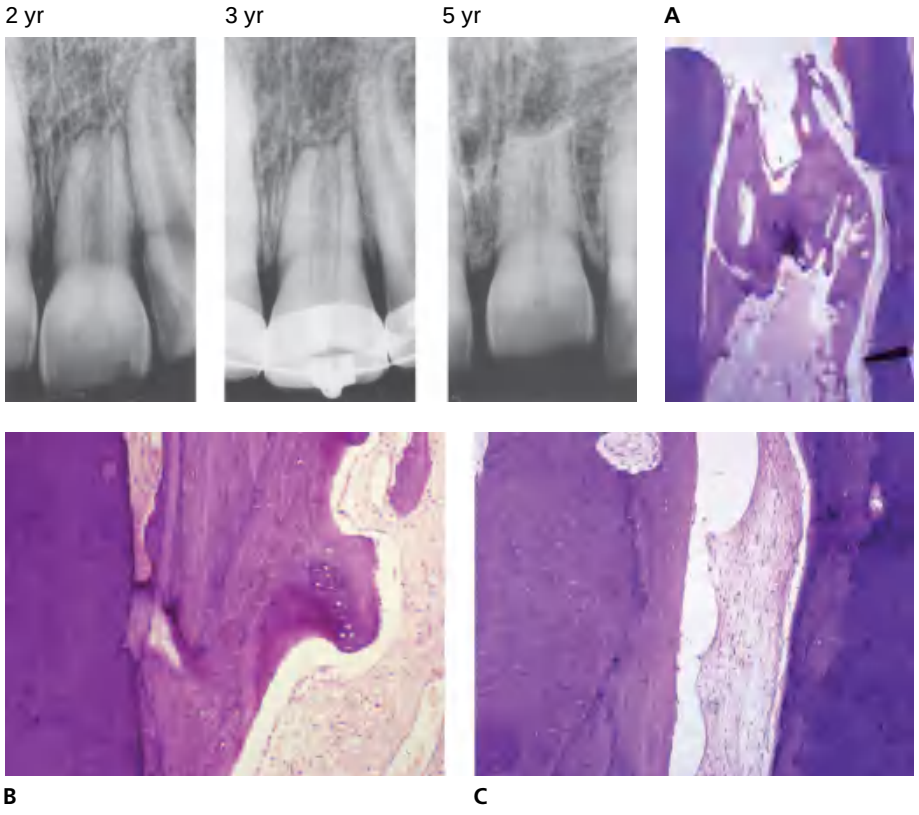



**Fig. 25.68** Radiographic and histological appearance of bony ingrowth into the root canal. There is increasing infraposition of the tooth at the 2-years and 5-years review appointments. Histologic appearance of bone ingrowth into the root canal of an intruded and subsequently infrapositioned incisor, 3 years after injury. A. Low power view of the extracted tooth. Note ingrowth of the bone into the root canal. ×3. B. Internal ankylosis. ×40. C. Internal periodontal ligament. ×40.

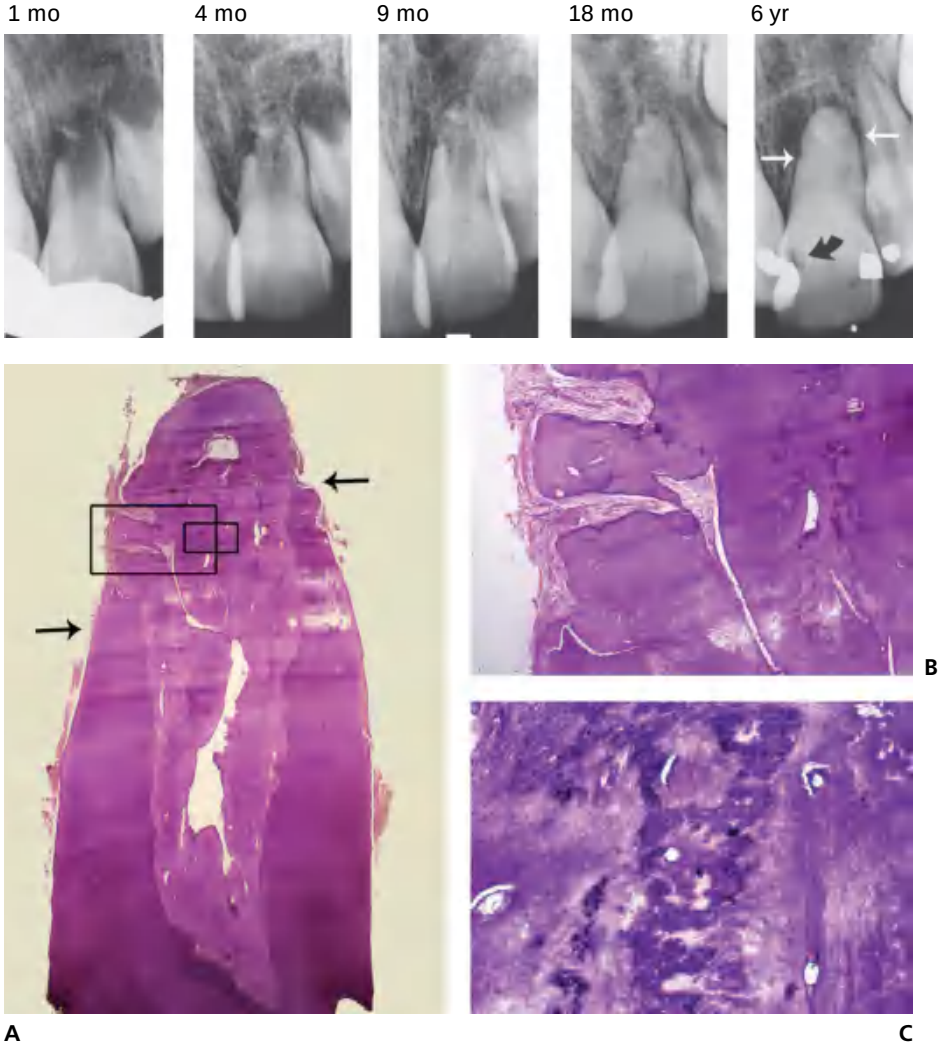



**Fig. 25.69** Irregular hard tissue formation in the root canal of a luxated immature incisor with subsequent pulp necrosis and infection. Six years after injury, a deep carious lesion (black arrow) and a periapical radiolucency is seen. A. Low power view of the extracted tooth. Note arrested root development, according to the extent of root development at the time of injury (arrows), and irregular formation of hard tissue in the root canal containing islands of soft, inflamed and necrotic tissue. ×7. B. Connection between the necrotic and infected pulp and the chronically inflamed periodontal space. ×10. C. Areas of dystrophically calcified tissue are surrounded by the irregularly formed dentin. ×50.

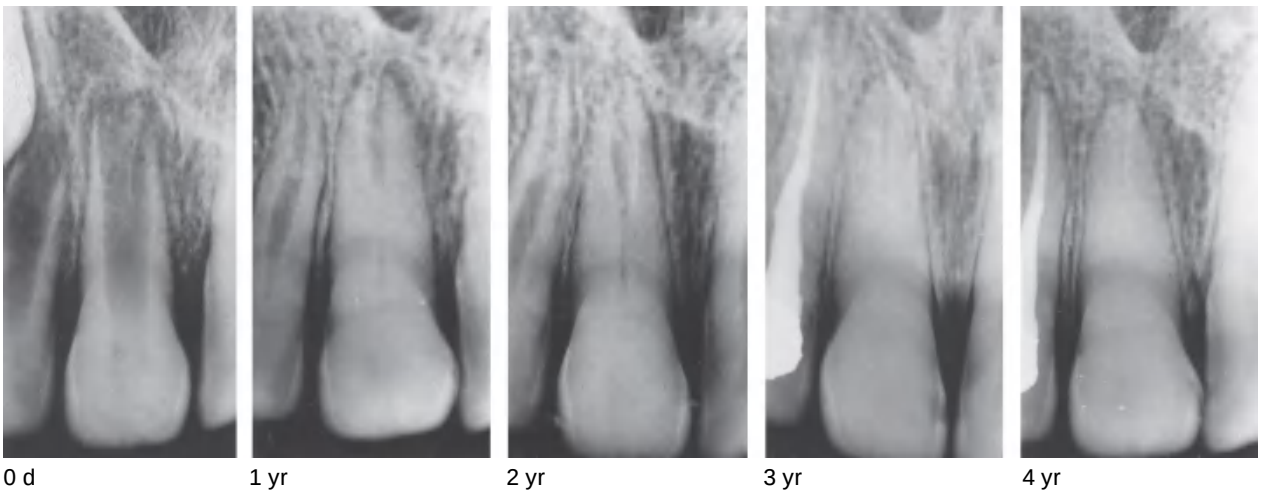



**Fig. 25.70** Radiographic appearance of the progressive narrowing of the root canal by apposition of hard tissue on the dentin walls. A luxated incisor at the time of injury and 1, 2, 3 and 4 years afterwards.

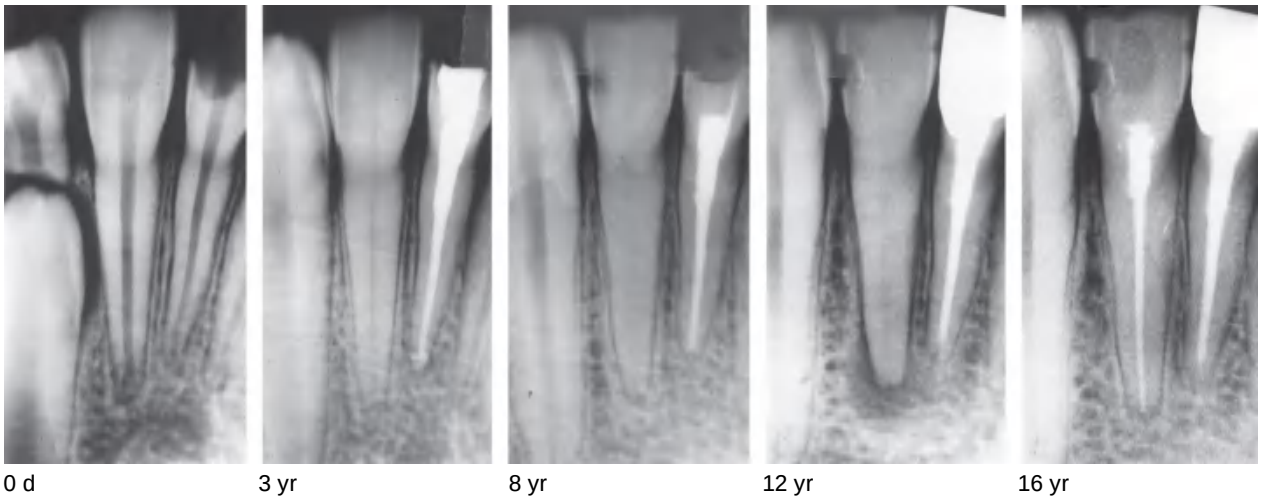



**Fig. 25.71** An incisor with pulp canal obliteration and then subsequent pulp necrosis and infection due to a deep carious lesion. Periapical healing can be seen 4 years after root canal treatment.

traumatically-induced pulp canal obliteration, a periapical radiolucency, indicating an infected root canal system, has been reported to occur in 13–16% of teeth over observation periods of up to 20 years (272, 273). In most cases, the occurrence of a periapical radiolucency is related to caries, cracks, breakdown of coronal restorations or new trauma (Figs 25.71 and 25.72). In teeth with apparently intact crowns, the pathogenesis can be difficult to determine but it is likely to be due to the presence of minor, undetected enamel craze lines or cracks extending to the dentin and allowing bacterial ingress to the pulp. An additional minor injury may cause ischemic necrosis of a pulp due to rupture of the few existing blood vessels. However, in this latter situation, the access of bacteria to the pulp remains to be explained.

The progressive diminishing of the root canal by formation of hard tissue represents a challenge for root canal treatment (272, 273). In the past, opinions varied as to whether or not so-called 'prophylactic endodontic treatment' should be instituted as soon as the onset of pulp canal obliteration had been diagnosed (272, 274–277). However, when the pulps from prophylactically treated teeth with pulp canal obliteration were examined histologically, changes were characterized by an increase of collagen and a varying decrease in the number of cells – that is, changes which do not warrant endodontic intervention (278).

One reason given by proponents for 'prophylactic endodontic treatment' was that ongoing obliteration would make the root canal inaccessible for later root canal treatment. However, in a study of 54 incisors with pulp canal obliteration that were treated because of the presence of infected root canals and apical periodontitis, the root canal was found and negotiated in all teeth but one, even though no or only a 'hairline' canal was visible in pre-operative radiographs (279). That particular study was done when modern techniques and devices such as transillumination, magnification, operating microscopes and enhanced

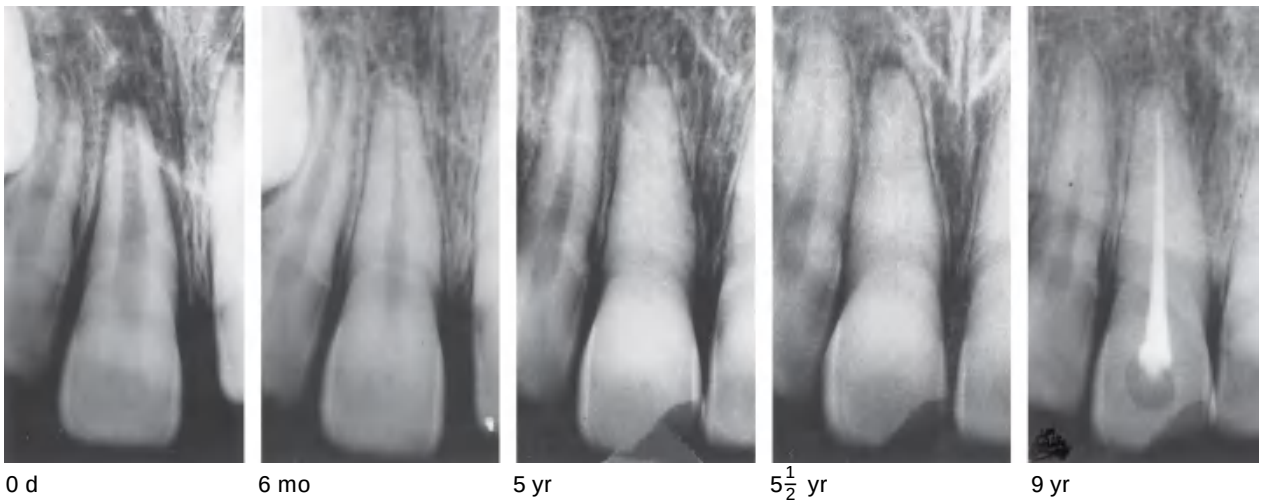



**Fig. 25.72** Root canal treatment of an incisor with pulp canal obliteration and subsequent pulp necrosis and infection due to another traumatic incident. The tooth suffered a crown fracture during the further trauma 5 years after the initial injury. Six months later, a periapical radiolucency has developed so root canal treatment was performed at this time. At the 4-year review after root canal treatment (9 years after the initial injury), periapical healing is evident.

lighting were not available or only in their infancy. These devices and techniques are now routinely used in modern endodontic practices which makes the location of obliterated canals much easier and more reliable. Thus, considering the low frequency of late pulp necrosis and infection with apical periodontitis, and the ability of clinicians to provide routine root canal treatment for teeth with obliterated canals, the need for 'prophylactic' endodontic intervention in teeth with early or ongoing obliteration of the root canal is not justified unless there is a specific indication such as the need for a post to retain a restoration in a tooth that has fractured.

If root canal treatment does become necessary in teeth with pulp canal obliteration, then a careful approach is required and adequate time should be allowed. Under rubber dam isolation, an access cavity should be prepared in the normal manner and to the typical depth of the pulp chamber for that particular tooth type. After irrigating the cavity with a solution such as ethylene diamine acetic acid and/or sodium hypochlorite, careful visual inspection may reveal a small area of dentin with a slightly different color to the surrounding dentin (Fig. 25.73). This is likely to be the canal orifice and it should be explored with a very small root canal file. Transillumination with a powerful light source can help reveal the canal orifice, as can magnification with loupes or an operating microscope. Radiographs with a file in the tooth to check the direction of exploration can also be helpful (Fig. 25.74). Extra radiographs including 'tube shift' views may be needed to assess the orientation and direction of progress. If the canal cannot be located at the base of the initial access cavity, then careful extension of the cavity to a slightly deeper level may help. This can be done progressively with regular monitoring of progress and direction, along with thorough visual and tactile searching for the canal orifice. Once the canal has been located, it should be negotiated to its full length, so the root canal treatment can continue in a normal manner (Fig. 25.74).

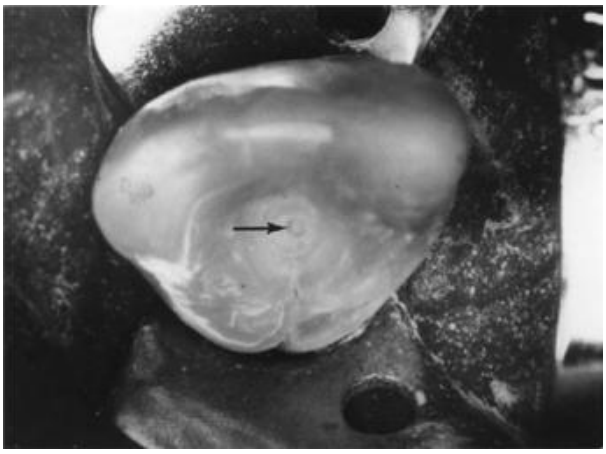


**Fig. 25.73** Access cavity for root canal treatment of an incisor with a pulp necrosis and infection plus pulp canal obliteration. An area of discolored dentin (arrow) indicates the location of the orifice to the obliterated root canal.

## Prognosis

Periapical healing after root canal treatment and filling of the root canal with various materials has been reported in clinical studies to range from 76 to 91% (245, 249, 280, 281). Periapical healing, after medication with calcium hydroxide and subsequent filling with gutta-percha and cement has been reported to be successful in 92–96% of the treated teeth (230, 246–248). The prognosis for successful root canal treatment of incisors with pulp canal obliteration, pulp

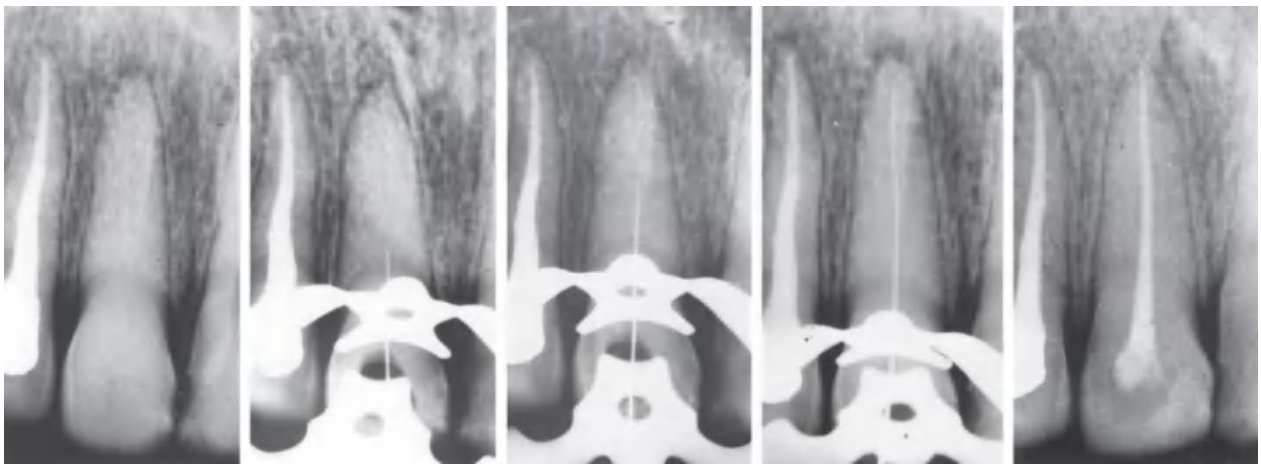



**Fig. 25.74** Root canal treatment of a central incisor with what appears radiographically to be 'complete' pulp canal obliteration by hard tissue after a luxation injury. A periapical radiolucency developed 9 years after injury as a result of pulp necrosis and infection. The root canal was located and carefully negotiated using small files. Periapical healing is evident 4 years after root canal filling with gutta-percha and cement.

necrosis and apical periodontitis has been reported to be 80% (279) but is likely to be higher with modern methods being used.

## External invasive tooth resorption following trauma

The late Miomir Cvek, previous author of this chapter (282), described this relatively infrequent type of resorption as 'late external resorption' which in many ways was an apt title as the clinical manifestations of this insidious type of tooth resorption often become apparent after a lag phase from the initial trauma which can vary from months to years. Over past decades this type of resorption has been accorded more varied terminologies than probably any other pathologic entity in dentistry (283–296) and this has led to some confusion.

The terms *invasive coronal resorption, invasive cervical resorption* and *invasive radicular resorption* will be used throughout this chapter each being descriptive of the nature of the resorption and its location on the external surface of the tooth. The term invasive implies a tendency to spread and while this has generally been applied in medicine to neoplasms, it is also apt for this group of tooth resorptions. The most common type of external invasive resorption is invasive cervical resorption. Although the aetiology of these resorptions is unknown, potential predisposing factors have been proposed for invasive cervical resorption and these have been investigated in a group of 222 patients with 257 teeth displaying varying degrees of this type of resorption (297, 298). This study confirmed dental trauma as a major potential predisposing factor either alone or in combination other factors such as intra-coronal bleaching or orthodontics (25.2% patients, 25.7% teeth). The dental injuries incurred were either luxations or avulsions. The study also showed that orthodontics alone or in combination with trauma and/or intra-coronal bleaching constituted the largest potential predisposing factor (26.1% patients, 28.4% teeth).

These resorptions are insidious in nature and present therapeutic challenges particularly in more advanced cases where the resorptive tissue invades tooth structure in a destructive and apparently uncontrolled fashion akin to the nature of some fibro-osseous lesions such as fibrous dysplasia (284). The resorptive tissue is thought be derived from ecto-mesenchymal precursor cells, within the reduced enamel epithelium in erupting teeth or from precursor cells within the periodontal ligament in fully erupted teeth. These precursor cells presumably are stimulated into resorptive activity by local factors elaborated following tooth damage. In the case of invasive coronal resorption, a developmental defect in the enamel is a likely pre-requisite while damage to the normally protective cementoid/cementum layer may predispose to the development of invasive cervical resorption (299).

Affected teeth are generally asymptomatic and often are diagnosed as a result of a pink discoloration in the cervical region of the tooth which is due to highly vascular tissue becoming visible through resorbed dentin and enamel. While rare examples of internal replacement resorption may show a pink coronal discoloration, the majority of teeth with pink discoloration are due to external invasive resorptions. Misconceptions in diagnosis can be traced from late 19$^{th}$ and early 20$^{th}$ century descriptions of teeth with 'pink spots' being labelled as internal resorption (300, 301).

Despite an absence of clinical signs or symptoms, diagnosis may also result from chance radiographic findings following routine examinations or follow up evaluations following dental trauma. The progression of external invasive resorptions is generally slow, but more aggressive forms do occur.

## Invasive coronal resorption

Invasive coronal resorption is a rare but destructive form of external root resorption which has been associated with areas of tooth hypomineralization in erupting teeth (302). Coronal enamel defects can also be the result of dental trauma in the form of an intrusive luxation of a primary tooth onto the crown of the developing permanent successor. Such an enamel defect may predispose to invasive coronal resorption as illustrated in Fig. 25.75 where a hypomineralized defect can be observed near the labio-cervical region. A pink discoloration and a probeable labial defect are indicated by a yellow arrow. On the palatal surface a crescent shaped pink area can be seen as indicated by the yellow arrows in Fig. 25.75. The radiographic appearance shows an irregular radiolucency extending into the coronal tooth structure and mirroring the clinical appearance (Fig. 25.75).

## Pathogenesis

It is likely that the resorptive tissue is derived from ecto-mesenchymal precursor cells in the reduced enamel epithelium in an erupting tooth and is fibro-vascular in nature as with invasive cervical resorption.

## Management

The basic aim of treatment is to remove or inactivate the resorptive tissue and to restore the resorptive defects with a suitable material. The example of invasive coronal resorption shown in Fig. 25.75 illustrates management. After protection of adjacent soft tissues with glycerol, the placement of a glycerol impregnated cotton roll into the labial sulcus and rubber dam application using the cuff technique (303) the active resorptive tissue was accessed both from the palatal and labial surfaces and treated by the topical application of 90% aqueous trichloracetic acid and curettage. The resorptive defects on the labial and palatal surfaces were then restored with a glass ionomer cement. Due to proximity of the resorptive process to the dental pulp, an adverse response in the form of apical periodontitis due to an infected root canal system became radiographically evident two years later and was managed by endodontic treatment. The clinical appearance

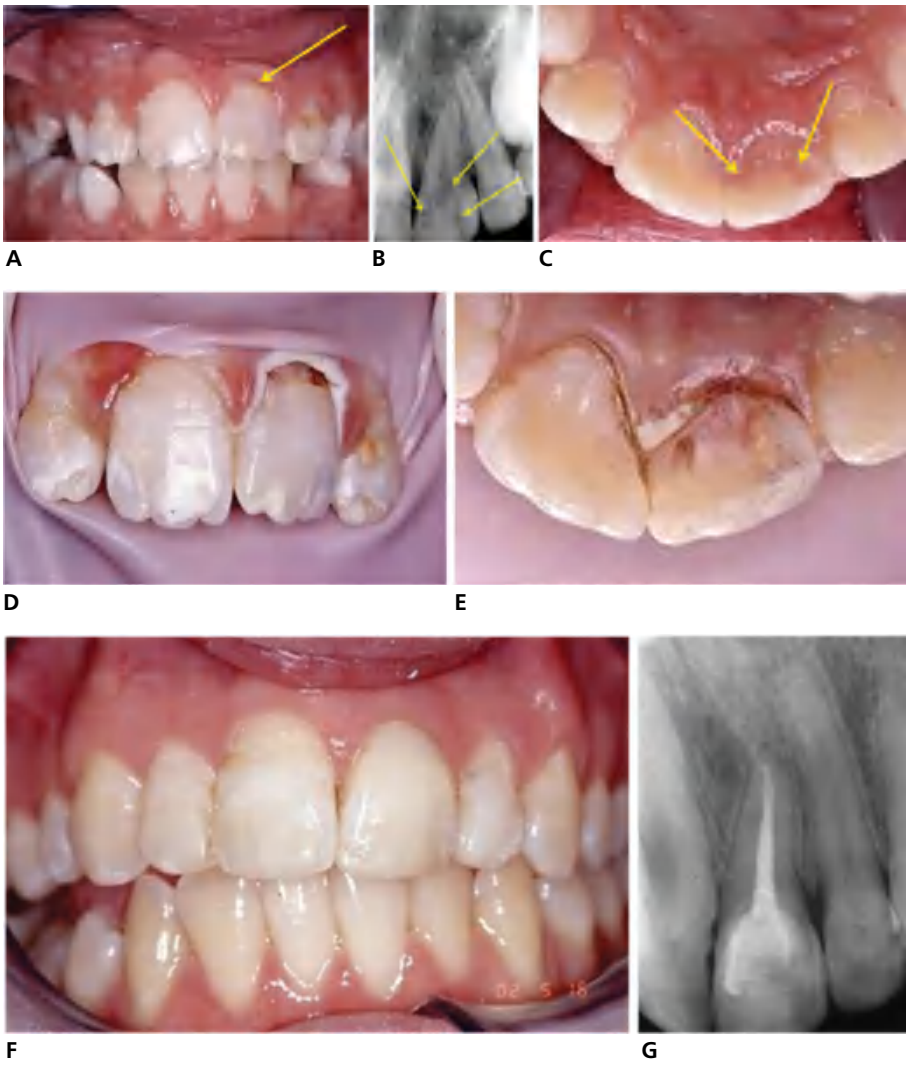



**Fig. 25.75** A. Labial view of a maxillary left central incisor showing a pink discoloration near the hypomineralised cervical aspect of the tooth. Some enamel cavitation which was probeable is indicated by the yellow arrow. B. A radiograph of the maxillary left central incisor shows an irregular radiolucency extending from the cervical region into the crown of the tooth, mirroring the shape of the pink discoloration. C. -Palatal view showing a crescental shaped pink discoloration extending from the gingival aspect of the tooth to the mesio and disto-incisal region as indicated by the yellow arrows. D. Labial view of the resorptive defect after the topical application of 90% aqueous trichloracetic acid, and curettage, prior to restoration with glass-ionomer cement. E. Palatal view after removal of the resorptive tissue as above, prior to restoration with glass ionomer cement. F. Appearance of the maxillary incisors 10 years after treatment. G. Radiograph of the maxillary left central incisor at the 10-year follow-up examination shows no evidence of pathosis.

**Fig. 25.76** A. The maxillary right central incisor of a 21-year old female shows a pink discolouration on the labial aspect of the crown. The tooth had been traumatised nine years earlier. B. A radiograph of the maxillary right central incisor reveals an irregular radiolucency overlying the root canal with no obvious extensions into the root canal. Source: Reproduced from (303) with permission.

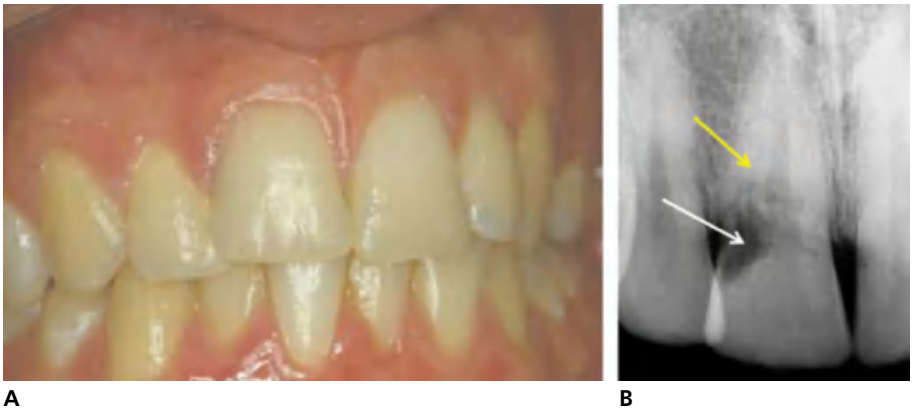


**Fig. 25.77** A. The maxillary right central incisor of a 24-year-old male shows a pinkish discoloration and slight enamel cavitation near the disto-gingival margin. B. The radiographic appearance of the maxillary right central incisor shows an irregular 'moth eaten' radiolucency extending to the outline of the root canal as indicated by the white arrow and into the radicular tooth structure for 3–4 mm as indicated by the yellow arrow. Source: Reproduced from (312) with permission.

of the tooth 10 years after the initial treatment and the corresponding radiograph are shown in Fig. 25.75.

## Invasive cervical resorption

Invasive cervical resorption is the most common form of external invasive resorptions which may often occur as a late complication of dental trauma, particularly where it involves damage to cementum and supporting tissues. As with invasive coronal resorption, the lesion may be clinically evident initially as a pink coronal discoloration but later in the process there may be enamel cavitation. An example of a maxillary right central incisor of a 21-year old female which had been traumatized nine years earlier is shown in Fig. 25.76. Pink discoloration of the crown is evident and there is a probeable defect at the labial cervical margin. The radiograph shows an irregular radiolucency overlying the image of the root canal.

An example of a more extensive invasive cervical resorption can be seen in Fig. 25.77 which show the clinical and radiographic images of a 24-year old male who received a blow to his right central incisor with a cricket ball, nine years earlier. A slight pink discoloration is evident on the disto-labial surface near the gingival margin, while the radiographic appearance of the incisor shows a characteristic irregular, 'mottled' radiolucency extending to the root canal outline and intra-radicularly for 3–4 mm as indicated by the yellow arrows.

## Pathogenesis

Invasive cervical resorptions are characterised by the ingrowth into coronal or radicular tooth structure by resorptive tissue which initially is fibro-vascular in nature and progressively resorbs dentine, enamel and cementum (Fig. 25.78) (298, 304). The resorptive tissue is thought to be derived from ecto-mesenchymal precursor cells within the periodontal ligament. These precursor cells presumably are stimulated into resorptive activity by local mediators elaborated following tooth trauma. In invasive cervical and radicular resorptions, a defect in the normally protective cementoid/cementum layer is a likely pre-requisite often being the result of dental trauma (299).

As the resorption progresses, the nature of the resorptive tissue become more fibro-osseous in nature with bone-like deposits observable both within the bulk of the invading tissue and also evident at an interface with dentin (Fig. 25.79). Although the latter histologically has similarities to replacement resorption, it differs in that there are infiltrating channels in dentine which can interconnect more apically with the periodontal ligament.

The pulp remains isolated from the resorptive tissue until late in the process, protected by an apparent anti-invasion factor in the predentin/dentin (305). A micro-CT scan of a tooth with extensive invasive cervical resorption shows the root canal of a tooth encircled by the image of invading resorptive fibro-osseous tissue (Fig. 25.80).

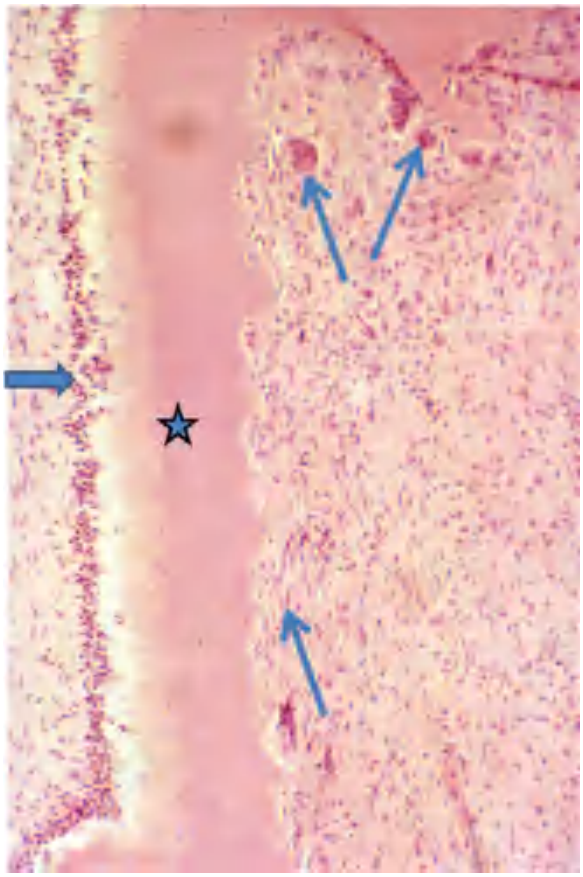


**Fig. 25.78** Histologic appearance of an incisor tooth with an early but established invasive cervical resorption. An intact layer of dentine and predentin on the pulpal aspect (*) separates the pulp (large arrow) from the resorbing tissue. The resorption cavity is filled with a mass of fibrovascular tissue with active mononucleated and multi-nucleated clastic cells lining resorption lacunae (arrows). (Haematoxylin-eosin stain; original magnification ×40). Source: Reproduced from (312) with permission.

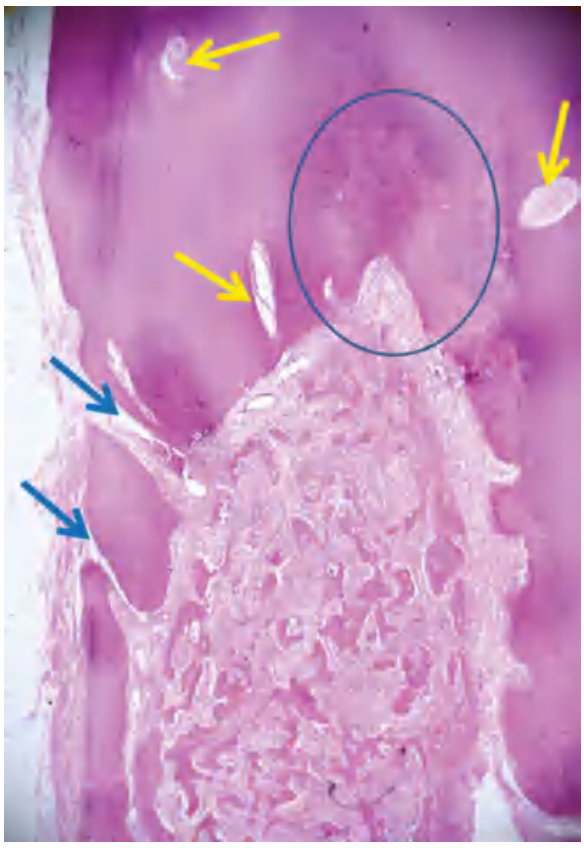


**Fig. 25.79** Histologic appearance of an extensive invasive cervical resorption with radicular extensions. Masses of ectopic calcific tissues are evident both within the fibrovascular tissue occupying the resorption cavity and on resorbed dentine surfaces (blue oval). In addition, communicating channels can be seen connecting with the periodontal ligament (blue arrows). Other channels can be seen within the radicular dentine (yellow arrows). (Haematoxylin-eosin stain; original magnification x30.) Source: Reproduced from (303) with permission.

Unless secondarily invaded with micro-organisms, the resorptive tissue is devoid of inflammatory cells which indicates a non-bacterial etiology. If secondary bacterial invasion occurs clinical signs and/or symptoms of pulp or periodontal infection can develop (298, 304).

A diagrammatic classification was developed as a guide to clinicians when making decisions regarding diagnosis and possible treatment (297, 298). The classification which was based on the periapical radiographic appearance is shown in Fig. 25.81.

The classification consists of 4 classes:

- **Class 1:** A small invasive resorptive lesion near the cervical area with shallow penetration into dentin.
- **Class 2:** A well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into radicular dentin.
- **Class 3:** A deeper invasion of dentin by resorbing tissue not only involving the coronal dentin but also extending into the cervical third of the root.
- **Class 4:** A large invasive resorptive process that has extended beyond the coronal third of the root canal and may involve almost the entire root.

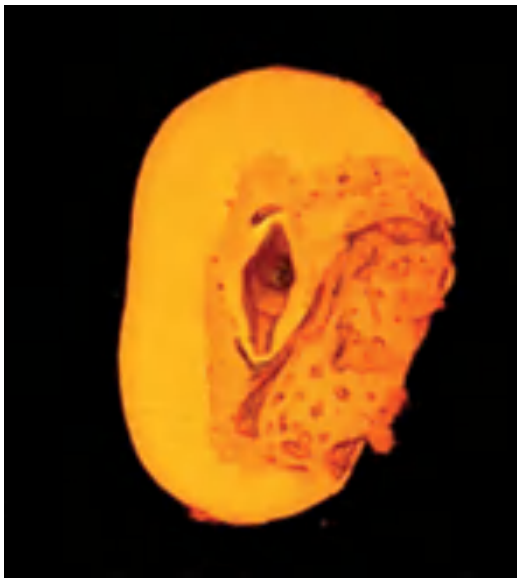


**Fig. 25.80** A cross-sectional view from a micro-CT scan of a maxillary canine tooth which exhibited an extensive invasive cervical resorption showing the intact root canal surrounded by image of resorptive tissue.

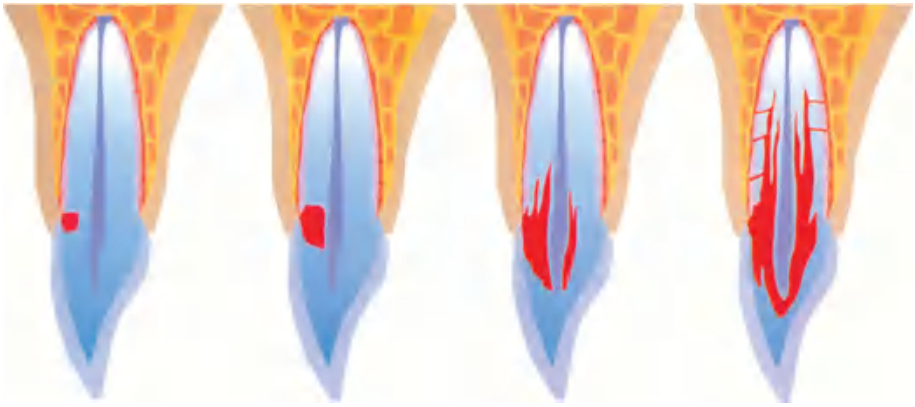



**Fig. 25.81** Clinical classification of invasive cervical resorption. Source: Reproduced from (297) with permission.

Since the advent of cone beam computerised tomography, additional useful information has been provided which can aid in diagnosis and treatment (306). Nevertheless the classification remains relevant as a simple guide for clinical decisions regarding management of teeth with invasive cervical resorption, Wherever possible such decisions should be based on published success rates following the treatment of each class of invasive cervical resorption.

## Management

Unlike some other resorptions which can follow dental trauma where the removal of the underlying cause eg micro-organisms, can control the progression of the resorptive process, the management of external invasive resorptions involves the total removal or inactivation of resorptive tissue to avoid the risk of recurrence or concurrence. Concurrence indicates the incomplete removal or inactivation of the active resorptive tissue while recurrence indicates the re-establishment of resorptive process.

While this aim is readily achievable with Class 1 and 2 lesions due to the localised nature of the resorptive process, the more advanced Class 3 and 4 lesions pose greater challenges, due to the complexity of the fibro-osseous resorptive network. In fact treatment of these more advanced resorptions is only recommended for class 3 lesions (302, 303).

In *Class 1 and 2* lesions, two principal treatment options have been described. The first involves the topical application of 90% aqueous trichloracetic acid to cause a coagulation necrosis of the active tissue followed by curettage and restoration (298, 302, 303). The second method involves periodontal flap refection, removal of the resorptive tissue by curettage, followed by restoration. The advantage of the first approach is that hemorrhage is avoided as the resorptive tissue is rendered avascular by the chemical agent and there is clinical evidence that overall success rates are extremely high and recurrence is avoided (296, 303). Precautions must be observed when using trichloracetic acid for the treatment of invasive resorptions. Care must be exercised in the dispensing of small quantities of the chemical agent by dental assistants. Glycerol which inactivates trichloracetic acid, should be applied to adjacent tissues and a glycerol impregnated cotton roll should be placed in the labial or buccal sulcus prior to the placement of a rubber dam cuff. Trichloracetic acid is applied on a small cotton pellet (size 00 divided in half) which has been dipped into the solution and then excess removed by dampening on a guaze square

While the periodontal flap and curettage method is more widely employed, studies as to success rates have yet to be reported. Some periodontists supplement the periodontal approach with the careful application of trichloracetic acid to the resorption cavity if there is any suspicion of infitrating channels within the dentin base.

An example of the treatment of a *Class 2* resorption shown in Fig. 25.76 is documented in Fig. 25.82A–G. Treatment involved the topical application of trichloracetic acid, curettage and restoration. An example of the periodontal approach is shown in Fig. 25.83A–F.

Treatment of *Class 3* lesions is challenging due to the nature of the fibro-osseous resorptive process. As for the Class 1 and 2 lesions, two principal treatment modalities can be employed. The first which utilises the topical application of 90% trichloracetic acid, curettage and restoration can be applied by an external or internal approach or a combination of both appraches. Alternatively the lesion may be treated with periodontal flap refection, curettage and repair of the resorptive defect with a suitable restorative material such as a glass ionomer cement, Geristore or MTA. Prophylactic endodontic treatment in the form of pulpectomy is desirable as it then allows enlargement of the root canal using Gates-Glidden burs to access encircling resorptive tissue and remove it mechanically or with the aid of the careful topical application of trichloracetic acid using a mini-applicator or paper point and ultrasonics. A radiograph of a Class 3 lesion is shown in Fig. 25.84 and the appearance of the tooth after pulpectomy, canal enlargement and chemical treatment using trichloracetic acid is shown in Fig. 25.84B. The root canal was then obturated and the defect restored with a glass ionomer cement. A 10-year radiograph follow-up is shown in Fig. 25.84C and clinical photographs in Figs 25.84D and 25.84E.

An entirely internal approach to the treatment of some of these externally derived resorptions is preferably executed using the operating microscope or with enhanced vision

A

B

C

D

E

F

G

**Fig. 25.82 Treatment of a class 2 resorption**
A. After a protective application of glycerol to adjacent soft tissue, supplemented with a glycerol impregnated cotton roll placed in the labial sulcus, a rubber dam 'cuff' has been placed for protection and isolation. B. Trichloracetic acid on a small cotton pellet is applied to the resorptive defect with slowly increasing pressure, so that the resorptive tissue within the cavity undergoes coagulation necrosis. C. The appearance of the devitalised tissue within the resorptive defect following the application of trichloracetic acid. The adjacent whitened gingival tissues indicate a limited zone of coagulation necrosis. D. Following curettage of the avascular tissue from the resorption cavity, the glistening dentinal base of the cavity is revealed. The incisal margin of the cavity has been smoothed with a high-speed bur under water spray. E. A glass-ionomer restoration has been placed in the cavity, and its surface has been protected with a light-activated unfilled bonded resin. F. Clinical appearance of the tooth five years post-operatively. The original glass-ionomer cement has been faced with a resin composite restoration. G. A 5-year follow-up radiograph of the maxillary right central incisor shows no evidence of periapical pathosis or recurrence of the treated resorptive lesion. Source: Reproduced from (302) with permission.

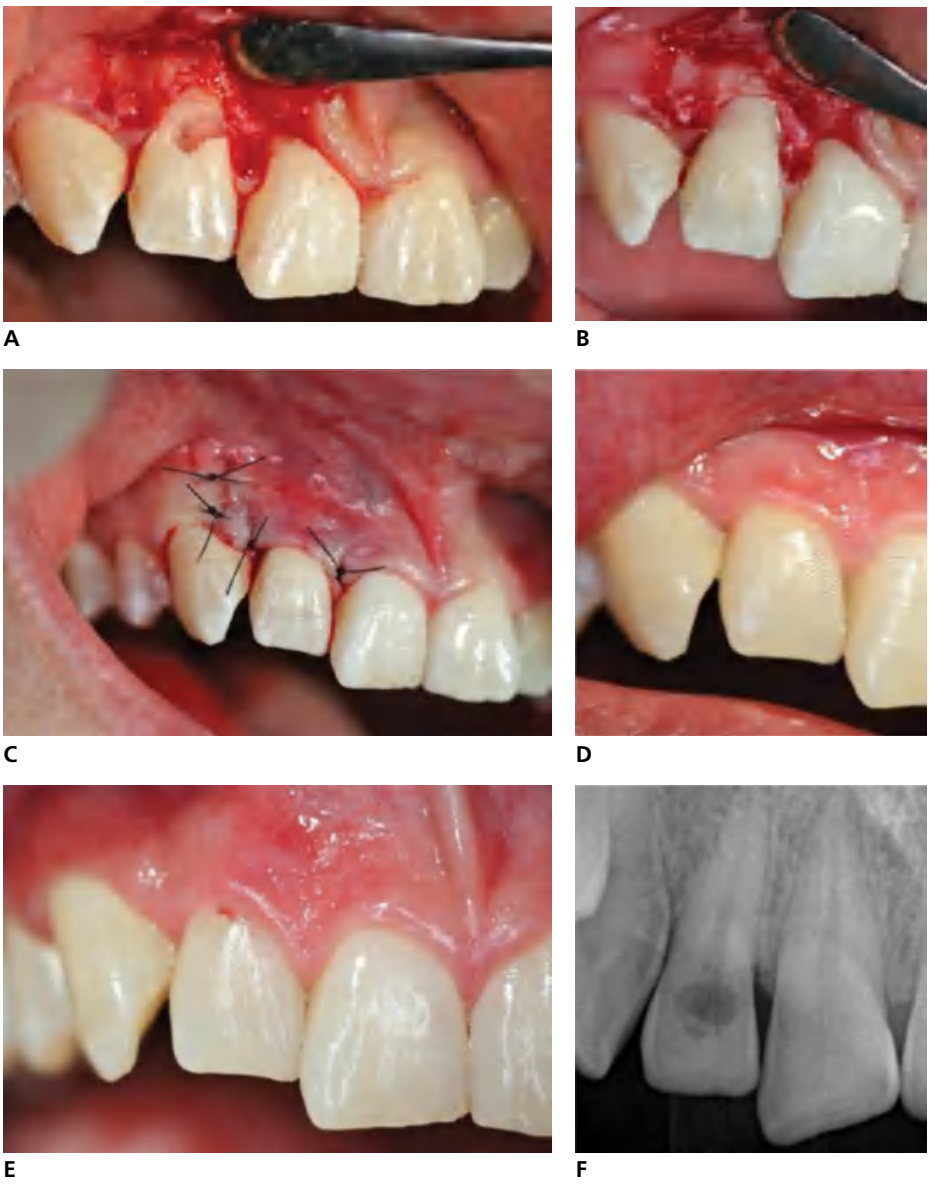



**Fig. 25.83** A. Labial view of a maxillary right lateral incisor showing a pink coronal discoloration near the gingival margin. B. A radiograph of the maxillary right lateral incisor shows an irregular shaped radiolucency overlying the outline of the root canal. C. Clinical photograph taken after periodontal flap reflection and curettage. D. Restoration of the resorptive defect with resin composite. E. Flap repositioning and suturing. F. Excellent soft tissue healing is evident post-operatively. Source: panels B–F courtesy Dr Steve Soukoulis Specialist Periodontist, Adelaide. Source: Reproduced from (302) with permission.

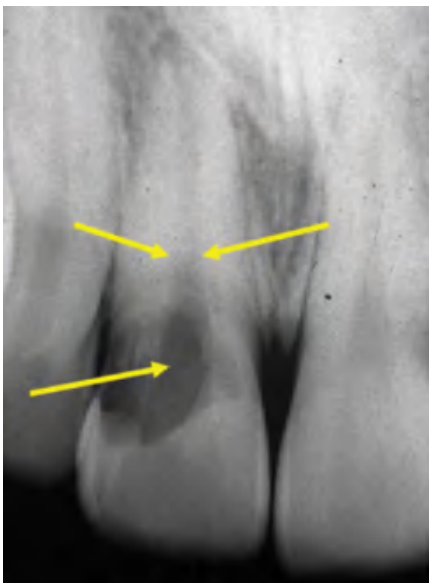
with a headlight so that the 'portal of entry' (288) can be visualised. The careful application of trichloracetic acid aids in the inactivation and removal of the resorptive tissue to the external site/s of entry,which can be facilitated with the use of a tungsten carbide ultra-sonic tip. When there are no clinical signs of active resorptive tissue, evidenced by avascularity, the defect can be sealed either with a glass ionomer cement or MTA followed by a conventional root canal filling. Orthodontic extrusion can be used to advantage in some Class 3 resorptions by improving access to the base of the resorption cavity and providing a supra-gingival margin for a subsequent restoration (302).

In a study of the treatment of 63 teeth with Class 3 invasive cervical resorptions (303) using the topical application of trichloracetic acid, pulpectomy and root canal widening, curettage, irrigation, root filling and restoration, 96.8% showed resorption control and 92.2% had tooth retention over the review period which varied from 3 to 12 years (mean 5.5 years), When all other factors, which included extraction due to root fracture, angular bone loss and periapical changes were taken into consideration, the overall success rate for Class 3 invasive cervical resorptions was 77.8%.

In the same study (303), while the retention rate of teeth with Class 4 lesions was 50% over the control period, the overall success rate, judged by the inclusion of other factors as above, was 12.5%. The complexity of Class 4 lesions and the poor results obtained with attempted treatment indicate that either the affected tooth be left *in situ* to progressively resorb or alternative therapy of extraction and prosthodontic replacement be instituted.

## Invasive radicular resorption

Invasive radicular resorption denotes the invasive process being located on radicular tooth structure as illustrated in Fig. 25.85. Another example is shown in Fig. 25.86 taken from a 78-year old patient who received a lateral luxation injury while playing football 47 years earlier. This late external invasive radicular resorption has been superimposed with an infection induced localised periodontitis with



A

B

C

D

E

**Fig. 25.84** A. A radiograph of the maxillary right central incisor shows a large, irregular radiolucency extending both coronally and into the radicular tooth structure as indicated by arrows. This invasive cervical resorptive lesion is classified as Class 3. B. The appearance of the tooth after the topical application of trichloracetic acid, curettage, elective pulpectomy, and canal enlargement with Gates-Glidden drills to allow access to encircling resorptive tissue for inactivation and removal. The extensive resorptive defect has well defined margins and no signs of further resorptive activity. C. A 10-year follow-up radiograph shows no evidence of further resorption or peri-radicular pathosis. D. The labial appearance 10 years after treatment shows a satisfactory esthetic result. E. After 10 years, the palatal gingival tissues appear healthy. The original glass-ionomer restoration has been refaced because of some surface crazing. Source: Reproduced from (302) with permission.

associated bone loss. Treatment of the first patient would involve an internal approach as detailed above, while for the second the defect could be accessed externally, curetted and sealed with a suitable restorative material.

The insidious nature of invasive cervical or radicular resorptions which can develop often without signs or symptoms years after an initial traumatic insult (307) emphasises the importance of long term clinical and radiological follow-up examinations of luxated or avulsed teeth, as early diagnosis and treatment of these invasive resorptions can result in an excellent prognosis for tooth retention.

**Fig. 25.85** Radiograph of a mandibular premolar showing radiographic evidence of invasive radicular resorption.

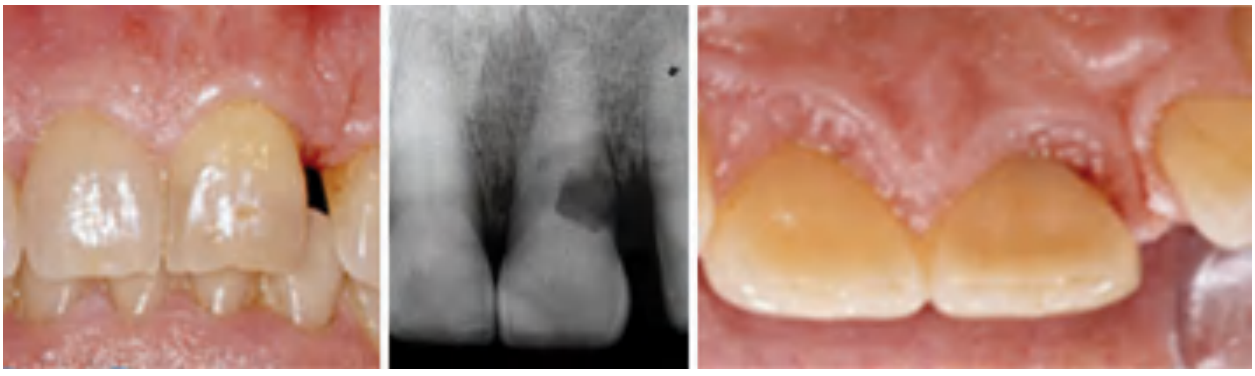
## Essentials

### Crown and crown-root fractures without pulp exposure

In crown fractures without direct pulp exposures bacteria can still cause problems for the pulp, especially if there is a concomitant luxation injury to the tooth. Compromised blood supply to the pulp reduces its ability to react to bacterial invasion through open dentinal tubules. Protecting the pulp can be accomplished by covering exposed dentin with a glass ionomer cement, followed by restoration of the fractured crown.

### Crown and crown-root fractures with pulp exposure

- Exposure of the pulp from traumatic crown fracture subjects the pulp to possible direct invasion by bacteria.
- Provided the blood supply to the pulp has not been compromised, the pulp is capable of protecting itself similarly to other bodily areas: a blood clot followed by granulation tissue covering that can keep the pulp protected for a considerable amount of time. This permits direct pulp treatment procedures such as pulp capping and pulpotomies to be accomplished even days after the traumatic dental injury.
- Direct pulpal protection procedures may be performed using either calcium hydroxide (CH) or one of the bioceramic materials available (such as MTA). The procedures for both dental materials are similar and can be summarized thus:
  (1) Anesthetize, isolate, and disinfect the tooth.
  (2) With a diamond bur under water coolant spray, remove about 2 mm pulp tissue below the site of pulp exposure.



A B C

**Fig. 25.86** A. The appearance of the anterior segment of a 78-year old male who had suffered a lateral luxation injury 47 years earlier. Note the disto-labial drifting of the lateral incisor. B. Palatal view of the maxillary right central incisor shows signs of an inflammatory response within the periodontium on the disto-palatal aspect of the tooth tissues. C. A radiograph reveals and extensive invasive radicular resorption on the disto- palatal aspect of the central incisor and a radiolucency in the adjacent bone indicative of chronic periodontitis.

(3) Place a cotton pellet moistened with sodium hypochlorite (NaOCl) directly on the pulp wound and leave until bleeding has stopped.
(4) Place the dental material (CH or MTA) directly on to the pulp wound followed by a layer of protective glass ionomer cement.
(5) The tooth can then be restored.

- Prognosis is considered good.

## Root fractured teeth

- The majority of the pulps survive root fractures.
- Only about 27% develop pulp necrosis and infection – this is usually confined to the coronal fragment.
- The pulp in the apical fragment usually remains viable and undergoes pulp canal obliteration.
- Initial management should be repositioning (if the coronal fragment is displaced) and stabilization.
- Review is essential
- If pulp necrosis and infection develop, then only treat the coronal fragment with root canal treatment.
- Either use calcium hydroxide dressings to encourage closure of the foramen at the fracture line, or use an 'apical barrier' technique before finishing the root canal filling.
- Rarely, the apical fragment may need root canal treatment or surgical removal.
- Prognosis – is good for root-fractured teeth.

## Luxated or avulsed teeth

- The pulp may be unaffected, compressed stretched, partially severed, or completely severed – depends on the type of luxation injury and its severity.
- Many pulps will survive minor luxation injures.
- The more sever the injury and displacement, the greater the possibility of pulp necrosis and infection. Avulsion and intrusion are the most severe injuries.
- A concurrent crown fracture with a luxation injury increases the possibility of pulp necrosis and infection.

### *Immature permanent teeth*

- Pulp survival is higher as revascularization is more likely – but concurrent crown fracture and more sever luxations have lower possibility of pulp survival.
- Initially stabilize the tooth and monitor for signs of pulp necrosis and infection unless these are inevitable (see below infection-related resorption).
- If pulp necrosis and infection do occur, then decide whether to do apexification treatment with long-term calcium hydroxide dressings, place an artificial barrier (e.g. an MTA 'plug'), or attempt a pulp regeneration procedure.
- Prognosis for healing is good.
- Long-term follow-up is essential.

### *Fully developed permanent teeth*

- Pulp necrosis is more likely due to the narrow apical foramen and restricted ability to revascularize.
- Some teeth will almost always have pulp necrosis and infection – i.e. avulsed teeth, intruded teeth, teeth that have been laterally luxated with a concurrent crown fracture, and teeth that have been extruded with a concurrent crown fracture. These teeth should have root canal treatment commenced immediately to prevent external infection-related root resorption. Preventive strategies should be used – such as corticosteroid-antibiotic dressings followed by calcium hydroxide dressings prior to root canal filling.
- Teeth with other injuries should be repositioned and stabilized, and then reviewed to determine whether pulp necrosis and infection occurs. Root canal treatment should only be done once this problem is evident. Routine root canal treatment has a good prognosis, especially if calcium hydroxide dressings are used to ensure disinfection of the root canal system.
- Prognosis for healing is good.
- Long-term follow-up is essential.

### *External infection-related root resorption*

- This resorption requires an infected root canal system plus damage to the root surface and/or periodontal ligament.
- As above, some injuries are more likely to be predisposed to this resorption – hence, preventive strategies should be adopted for these injuries.
- Prognosis for healing is good if early intervention.
- Long-term follow-up is essential.

### *Pulp canal obliteration*

- Commonly occurs after luxation injuries and root fractures.
- Pulp canal obliteration is an indication of pulp survival and does not require treatment.
- Root canal treatment is only required if the pulp necroses and becomes infected, causing apical periodontitis.
- Root canal treatment can be performed with careful operative procedures. The use of transillumination, magnification and enhanced lighting aids the location and negotiation of the canals.
- Once the canal has been located, the root canal treatment is routine and ideally should include the use of calcium hydroxide dressings to ensure disinfection of the root canal system.
- Prognosis for healing is good.
- Long-term follow-up is essential.

### *External Invasive resorptions*

- Trauma is a major predisposing factor for the development of external invasive resorptions.
- Invasive resorptions are defined according to their location as Invasive coronal, Invasive cervical, or invasive radicular resorptions.

- Invasive resorptions may develop within a few weeks or years following the original injury
- A pink discoloration may be evident in the tooth crown.
- Radiolucencies are evident in the crown and/or root dependent on the stage of progression of the resorptive process.
- Invasive coronal resorption can develop at the site of enamel hypomineralization in erupting permanent teeth. The enamel defect can result from an intrusive luxation of a primary tooth impinging on a permanent successor.
- Invasive cervical resorption is characterised by the invasion into tooth structure by fibro-vascular/fibro-osseous resorptive tissue.
- The site of invasion is the cervical region due to a defect in the normally protective cementum/cementoid layer.
- A clinical classification used as a guide for treatment planning defines 4 classes based on the radiographic evidence of the extent of the resorption.
  Class 1: A small invasive resorptive lesion near the cervical area with shallow penetration into dentin.
  Class 2: A well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into radicular dentin.
  Class 3: A deeper invasion of dentin by resorbing tissue not only involving the coronal dentin but also extending into the cervical third of the root.
  Class 4: A large invasive resorptive process that has extended beyond the coronal third of the root canal and may involve almost the entire root.
- Management of external invasive resorptions involves the total inactivation of resorptive tissue by either of two documented regimes:
  (1) Carefully controlled topical application of 90% trichloracetic acid, curettage of the devitalised resorptive tissue, root canal treatment if required, and restoration of the defect with a suitable restorative material.
  (2) Surgery with flap refection, curettage and restoration.
- Prognosis: Excellent for Class 1 and 2 ICRs, acceptable for Class 3s and poor for Class 4s where treatment is not recommended.
- It is important to do short and long term follow-up examinations of luxated or replanted teeth to provide early diagnosis and treatment of ICRs.

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# 26
# Regenerative Endodontics

B. Kahler & L. M. Lin

## Overview

Regenerative endodontics is an exciting and developing field in the treatment of immature teeth with infected root canals that has been described as a "paradigm shift" in the management of these teeth and can result in continued root maturation and apical closure (1–3). The excitement and potential of regenerative endodontics has been the subject of many reviews (4–10), with further developments expected in the use of stem cells, scaffolds, growth factors and bioengineering protocols (6).

## Diagnosis of traumatic dental injuries and revascularization of teeth with an open apex

Traumatic dental injuries primarily affect maxillary central and lateral incisors with a prevalence of 20–30% in young patients (11). Studies have also shown that the majority of these injuries occur between the ages of 7–12 years when a tooth may have incomplete root development (12). Not all is lost as numerous studies that show that teeth with open apices have a greater potential for revascularization with healing of the pulp and continued root formation. Revascularization of a necrotic pulp following avulsion in an immature tooth has been demonstrated (13–15), also after root fractures (16, 17) and after luxation injuries (17). The healing mechanism demonstrated in transient apical breakdown further showed that repair of teeth with completed root maturation could occur (18) illustrating the enormous reparative capacity of the pulp for repair without endodontic interventions. Therefore, it is of the utmost importance that a correct diagnosis is determined for injured immature teeth prior to any endodontic intervention (19, 20). Furthermore, maintaining pulp vitality wherever possible in the developing dentition is paramount and certainly dependent on a definitive diagnosis. This is particularly true for trauma cases where loss of pulp sensibility may make diagnosis difficult. However, a diagnosis of pulp necrosis can be associated with dental trauma (21).

### The consequences of pulp necrosis for the immature tooth

Pulp necrosis as a consequence of trauma in the developing dentition results in cessation of root maturation leaving the roots of the teeth prone to root fracture and tooth loss. Cvek (22) in a study of 885 luxated, non-vital incisors demonstrated that the frequency of cervical root fractures was dependent on the stage of root development and ranged from 77% in teeth with the least to 28% in teeth with the most developed roots. Traditionally these traumatized teeth were treated with calcium hydroxide ($Ca(OH)_2$) apexificaton techniques involving multiple treatment visits with an extended treatment time (23–25). While this treatment technique has been shown to be successful (26), long term placement of $Ca(OH)_2$ in root canals may increase the risk of root fracture (27). With the development of mineral trioxide aggregate (MTA) it was proposed that this material could be used as a barrier material for immature teeth with open apices (28). The use of MTA has been shown to eliminate the need for multiple visits and extended treatment times with one-visit barrier placement (29, 30). However, neither technique results in further substantive root maturation, leaving the roots weak and possibly prone to fracture.

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## Regenerative endodontics

Regenerative endodontics has been defined as "biologically based procedures designed to replace damaged structures, including dentin and root structures, as well as cells of the pulp-dentin complex" (4). The clinical considerations for regenerative endodontic protocols are (1) disinfection of the root canal system, (2) provision of a scaffold which often involves laceration of the periapical tissue to induce a blood clot and introduce stem cell activity within the root canal, and (3) an adequate coronal seal to prevent reinfection (4–6, 8–10).

The assumption is that regenerative endodontic protocols which result in continued root maturation mean the teeth and roots are not as inherently weak and susceptible to fracture as the traditional techniques of $Ca(OH)_2$ apexification and MTA barrier placements. A recent study that compared the traditional and regenerative protocols showed revascularization was associated with significantly greater increases in root length and thickness compared with calcium hydroxide apexification and MTA barrier placement as well as higher overall survival rates (31).

## Regeneration or repair

Tissue injury caused by infection or trauma after appropriate treatment will result in healing by either regeneration or repair (32). Regeneration is defined as reconstitution of damaged tissue by tissue similar to original tissue and restoration of biological functions (32, 33). Repair is replacement of damaged tissue by tissue different from original tissue and loss of biological functions (32, 33). Regeneration and repair depend on several factors, such as intensity and duration of tissue injury, and the ability of the tissue to regenerate. Most tissues have certain potential of regeneration after injury. Although stem cells exist in all living tissues and organs, some tissues appear to have minimal or no potential of regeneration such as heart and central nerve system. Dental pulp has a limited potential of regeneration (34).

Aging is also an important factor in tissue regeneration due to decline in stem cell functionality (35), which can be caused by disregulation in intrinsic regulatory mechanisms of cells such as epigenetic modifications and DNA damage, as well as diminution in extrinsic local and systemic microenvironment cues (36–39). It has been shown that dental pulp tissue regeneration is related to aging (40). Therefore, dentin-pulp tissue regeneration of immature permanent teeth with necrotic pulps may not be easily achieved after regenerative endodontic procedures (REPs) in adult patients (41).

Regeneration is unlikely to occur de novo if living tissue/organ including parenchymal cells and extracellular matrix is completely damaged in situ. Therefore, in such cases tissue/organ transplant is a common practice in medicine. When pulp tissue in a tooth is completely destroyed by trauma or infection, in situ pulp tissue regeneration or transplantation is not possible; therefore root canal treatment is routinely performed. However, tissue engineering or regenerative medicine has made some miracle advances in medicine but not in dentistry to replace tissues/organs damaged by infection, trauma, or other disease via implanting bioengineered tissue/organ constructs into the patients, for example, artificial skin graft for severe burn patients and artificial bone marrow transplant for leukemic patients.

Wound healing requires recruitment of stem/progenitor cells to damaged tissue/organ to differentiate into tissue-specialized somatic cells (42–44), because tissue somatic cells have limited regenerative potential (45, 46). Differentiation of stem/progenitor cells into tissue-specialized somatic cells is tightly controlled by micro-environmental cues and bioactive signaling molecules (growth/differentiation factors) (42–44). In REPs, many growth factors embedded in the dentin matrix are released into the canal space after EDTA treatment (47). These growth factors have been shown to be able to signal pulp stem cells to differentiate into odontoblast-like cells and produce reparative dentin (48–51). However, the mesenchymal stem cells (52) introduced into the canal space during REPs do not appear to be able to differentiate into odontoblast-like cells and produce the dentin-pulp complex in many animal and human studies. Therefore, tissue engineering may be a promising approach using ex vivo expanded dental pulp stem cells seeded on bioactive scaffold and implanted into the disinfected canal space to regenerate the dentin-pulp complex of immature permanent teeth with necrotic pulps (53).

## Histologic characterization of tissues formed in the canal space

As aforementioned, many clinical case reports and case series have showed that immature permanent teeth with necrotic pulps after REPs could result in radiographic thickening of the canal walls and/or continued root maturation or apical closure. In addition, the triad of tissue engineering, blood clot, mesenchymal stem cells and bioactive growth factors, were brought into the canal space during induction of periapical bleeding in REPs. Therefore, it was believed clinically that the dentin-pulp complex could be regenerated in immature permanent teeth with necrotic pulps after REPs. However, the nature of tissues formed in the canal space of immature permanent teeth with necrotic pulps after REPs can only be determined by histological examination. Many histological studies of REPs of immature teeth with necrotic pulps and apical periodontitis have been investigated in animal models and humans. It was revealed that the tissues formed in the canal space were mineralized tissue similar to cementum and bone, and fibrous connective tissue resembling periodontal ligament (Figs 26.1 and 26.2) (54–60). Thickening of the canal walls and/or apical closure were due to apposition of cementum-like or bone-like tissue and not dentin. In some cases, the apical canal space was almost completely filled with mineralized tissue (61). While in other cases, there was minimal thickening of the canal walls and the canal space contained fibrous connective tissue (59). Ingrowth of apical alveolar bone into the apical canal space was discernible in some cases (Fig. 26.3) (61, 62).

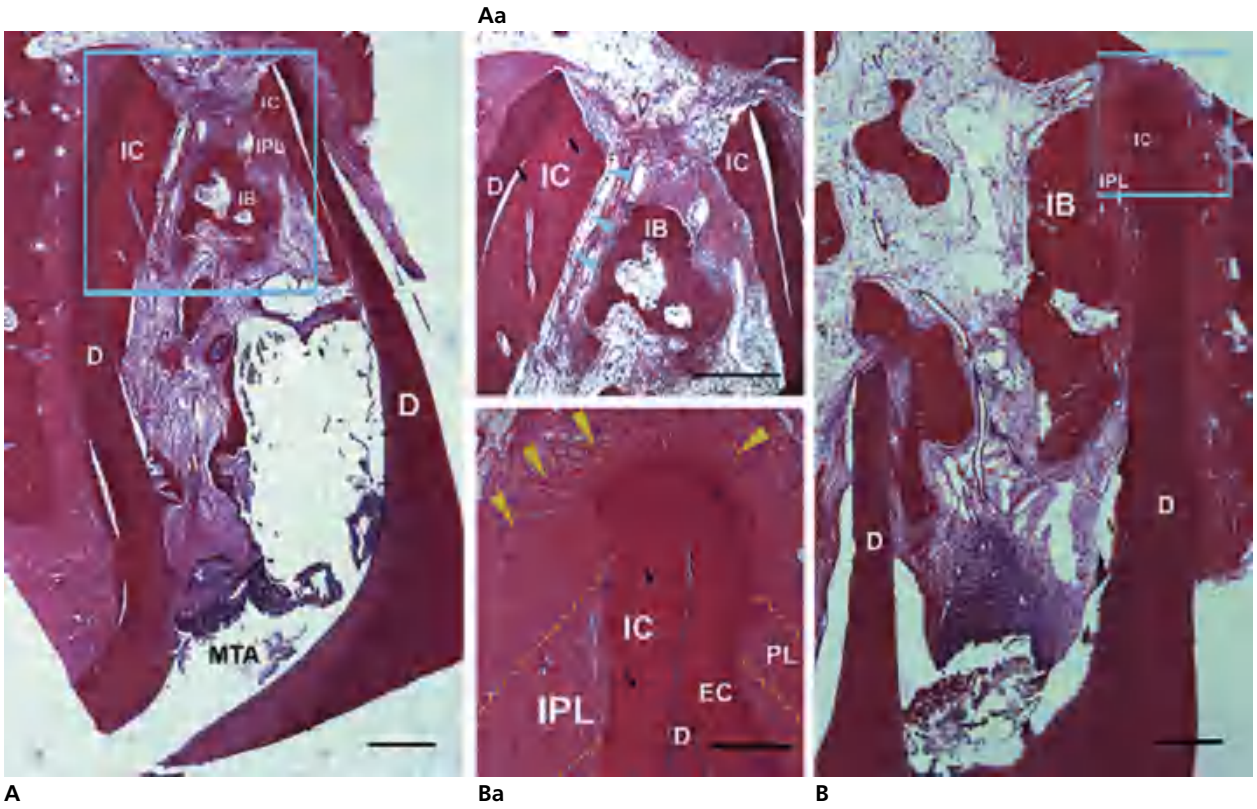


**Fig. 26.1** Revascularized dog immature tooth. A. The canal space filled with intracanal cementum (IC), intracanal bone (IB), fibrous connective tissue, and deposition of cementum on the canal dentin walls. Aa. High magnification of square in A. The canal walls covered by intracanal cementum (IC). Ba. High magnification of square in B. Intracanal periodontal ligament inserted into intracanal cementum (IC). B. Ingrowth of alveolar bone (IB) and cementum (IC) into the apical canal space. D, dentin;IC, intracanal cementum;IB, intracanal bone;IPL, intracanal periodontal ligament;EC, external cementum;PL, periodontal ligament. Source: Reproduced from (54) with permission.

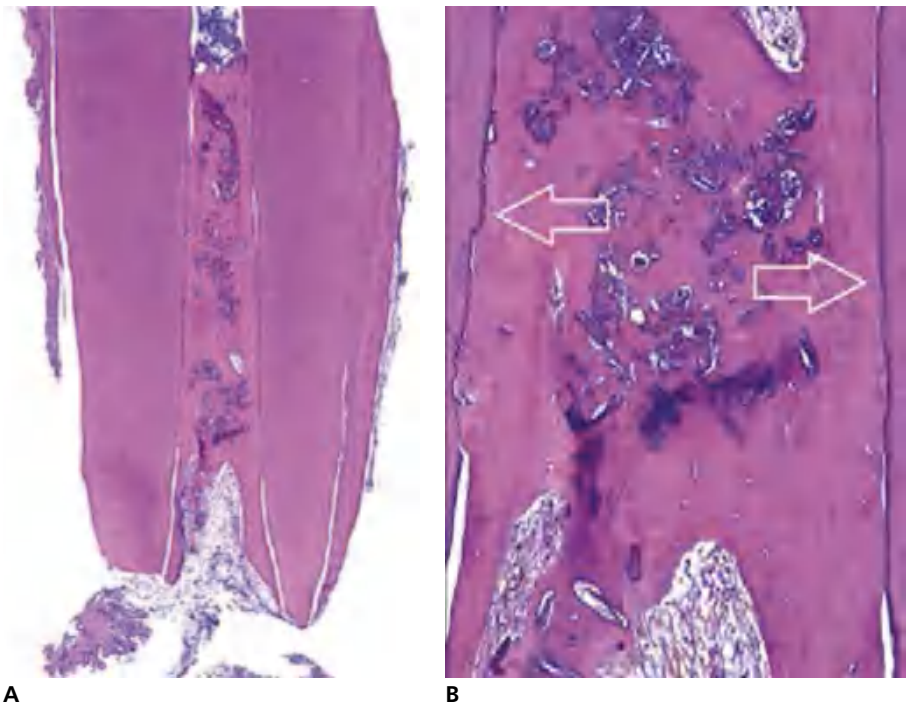


**Fig. 26.2** Revascularized human immature permanent tooth. A. The canal space filled with mineralized and fibrous connective tissue. B. High magnification of A. Deposition of cellular cementum-like tissue on the canal dentin walls (arrows). Source: Reproduced from (57) wirh permission.

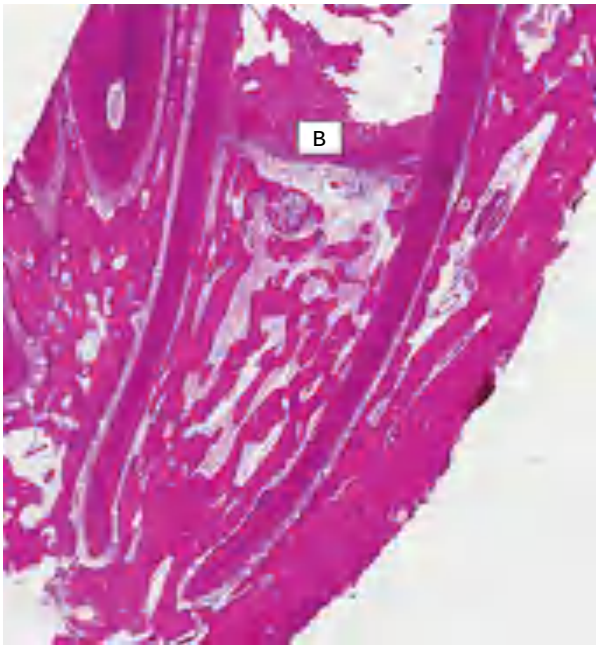


**Fig. 26.3** Revascularized ferret immature tooth. Ingrowth of bonelike tissue into the root canal to the level of the MTA where a hard tissue bridge (B) had formed in a specimen treated with PRP as scaffold. Source: Reproduced from (62)with permission.

Clinically, this may result in ankylosis of the tooth and the apical alveolar bone.

Interestingly, studies with uninfected and healthy immature teeth treated with REPs showed that the tissues formed in the canal spaces were fibrous connective tissue, bone-, and cementum-like tissue and no dentin-pulp-like tissue in animals and humans (62, 63). Therefore, the tissues formed in the canal space of immature permanent teeth with infected necrotic pulps and with healthy pulps are similar after REPs. The stem cells introduced into the canal space of teeth with necrotic pulps during REPs are most likely from bone marrow and periodontal ligament, because these stem cells are capable of differentiating into osteoblasts and cementoblasts, respectively, upon receiving appropriate signaling molecules (64). Based on these studies, it appears that only cells from the dental pulp tissue or pulp tissue precursor (apical papilla) are capable of regenerating the dentin-pulp complex after REPs. The cellular and molecular mechanism of cementum-like or bone-like tissue formed in the canal space after REPs of immature teeth with necrotic pulps in human and animal studies is largely unknown.

Several studies have showed that pulp-like tissue could be regenerated by cell homing in REPs in animals (65, 66). However, if bona fide odontoblasts, a highly specialized and multi-functional cell capable of producing primary and secondary dentin (67), recognizing foreign microbial products by Toll-like receptors (68), and perceiving external stimulus through transient receptor potential (TRP) channels (69), are absent in regenerated pulp-like tissue, the regenerated tissue is not different from fibrous connective in other parts of the body and cannot be considered as a genuine pulp tissue.

Histologically, REPs of human immature permanent teeth with necrotic pulp are considered a reparative and not a regenerative process (70, 71). Radiographic thickening of the canal walls, and continued root maturation of immature permanent teeth with necrotic pulps after REPs should not be regarded as a regeneration of dentin-pulp complex without histological confirmation. If the primary goal of REPs is elimination of clinical symptom/sign and resolution of apical periodontitis (72), then repair, although not an ideal wound healing, is not a treatment failure (70, 71). However, the long-term outcome of immature permanent teeth with necrotic pulps after REPs has been questioned because of ectopic cementum- and bone-like tissues formed in the canal space (73).

## Terminology

A number of terms have been adopted in the literature with regenerative endodontics, revascularization and revitalization being the most commonly used.

The term 'revascularization' is well established in the endodontic literature and relates to the reestablishment of vascularity in the pulp space after traumatic injuries that sever the blood supply to the pulp of immature teeth (74, 75). As earlier reports on this new technique showed renewed root maturation in infected immature teeth and described the introduction of a blood clot into the root canal, it is not surprising that the term "revascularization" was adopted (76, 77).

Regenerative endodontics implies that further root maturation results in reestablishment of the pulp dentine complex. Many studies show that this is not the case with a variety of tissues such as dentine, cementum, periodontal ligament, bone, osteoid and possibly pulp being the tissue found in treated teeth with regenerative endodontic protocols, suggesting 'repair' rather than 'regeneration' (54, 57, 59).

'Revitalization' has been suggested as it describes nonspecific vital tissue rather than just blood vessels as implied by the term 'revascularization' (78).

For the purpose of this chapter, the term REPs will be used to include procedures which have been called revascularization, revitalization and regenerative endodontics discussed above. Diogenes et al. argue that this term is more inclusive and encompasses all procedures that aim to attain organized repair of the dental pulp and include future therapies yet to evolve in the field of regenerative endodontics (6). Known areas of emerging research include root canal revascularization via blood clotting, post-natal stem cell therapy, pulp implantation, scaffold implantation, injectable scaffold delivery, three-dimensional cell printing and gene delivery (4).

## History

The promise and potential of regenerative endodontic therapies in necrotic teeth was first explored by Nygaard-Östby (79) in 1961 with limited success. The protocol involved

disinfection of the root canal space with sulfathiazole and 4% formaldehyde. Clinical and histological studies of that time investigated the potential for repair when bleeding was induced by over instrumentation beyond the apex prior to partial root filling of the canal with Kloropercka and gutta percha (80). Teeth with vital pulps resulted in repair with formation of fibrous connective tissue and concomitant deposition of cellular cementum. However, no repair occurred in teeth with necrotic pulps.

Forty years later, in 2001 Iwaya et al. (76) reported a case using a procedure termed 'revascularization', on an infected necrotic immature premolar tooth that showed continued root maturation and thickening of root canal walls with mineralized tissue. Subsequent case reports also demonstrated the efficacy of this technique primarily in premolar teeth when using a triple-antibiotic paste (77, 81, 82) or calcium hydroxide (83, 84). Further reports demonstrated successful outcomes in traumatized central incisor teeth (78, 85–87). A number of authors have described regenerative endodontic techniques as a "paradigm shift" in the treatment of immature teeth with necrotic pulps (1–3).

## Clinical considerations for regenerative endodontic procedures

### *Young patient*

Whilst REPs have been utilised on mature teeth (88–90), the vast majority of reported cases are undertaken on young patients with immature infected teeth where cessation of root maturation has occurred as a consequence of pulp necrosis. The American Association of Endodontists (AAE) clinical considerations for REPs define success by three measures (72):

- Primary goal (essential): the elimination of symptoms and the evidence of bony healing
- Secondary goal (desirable): increased root wall thickness and/or increased root length
- Tertiary goal: positive response to vitality testing (91)

Whilst the primary goal is an objective for all endodontic treatments, it is the secondary goal of increased width and/or length that is pertinent in the immature tooth. The assumption is that further root maturation often assessed in conjunction with apical closure of the immature root apex may minimise the incidence of root fracture. Another assumption is in the tertiary goal that a return of neural capacity may indicate a more organized vital pulp tissue (72). In 2013, a review reported many cases demonstrated a positive response to pulp sensibility testing (6). Whilst the presence of some vital tissue prior to commencement of REPs has been reported (81), approximately 40% of treated cases were diagnosed with apical periodontitis and another approximately 35% of cases presented with an apical abscess (6). In some case series, the primary goal of elimination of symptoms and evidence of bony healing has not always been attained (86, 87).

### *Minimal or no instrumentation of the dentinal walls*

REPs advocate minimal or no filing of the canal (92). The AAE guidelines have no mention of mechanical instrumentation (59). A review in 2015 reported canal walls were not mechanically instrumented in 68% of clinical articles (9). However, in a histologic and histobacteriologic study of a failed REP treatment most bacteria were noted in the apical and not the coronal portion of the canal where a biofilm had formed on the canal walls and penetrated into the dentinal tubules. The authors concluded that on the basis of the histobacteriologic findings that some degree of mechanical debridement may also be required to disrupt biofilm on the canal walls for continued root maturation to occur (92).

### *Disinfection of the root canal system*

Disinfection of the root canal system is thought to be critical to the success of REPs as infection prevents regeneration, repair and stem cell activity (92, 94). Chemical disinfection of the root canal system is not solely dependent on bacteriocidal/ bacteriostatic properties of the agents as these irrigants/medicaments should not damage the survival and proliferative capacity of the patient's stem cells (6). As few case reports have been performed in a single visit (95–97), the protocol describes a two-visit approach with the use of irrigants and an intra-canal medicament (to be discussed in the next subsection).

The guidelines suggest copious irrigation with 20 ml sodium hypochlorite (NaOCl) using an irrigation system that minimizes the possibility of extrusion of irrigants into the periapical space (e.g., needle with closed end and side-vents, or EndoVac™). Lower concentrations of NaOCl are advised (1.5% sodium hypochlorite (20 mL/canal, 5 min) and then irrigated with saline or EDTA (20 mL/canal, 5 min) with the irrigating needle positioned about 1 mm from the root end to minimize cytotoxicity to stem cells in the apical tissues (72).

A review in 2015 found that 97% of clinical studies used sodium hypochlorite as either the only irrigant or in combination with other irrigants. Sodium hypochlorite, chlorhexidine and EDTA were used in the final irrigation protocol in 75%, 4% and 13% of the clinical studies respectively (9). In 2013, 63% of all REP cases were irrigated with 3% NaOCl, 36% of cases irrigated with 5–6% NaOCl and 1% with 1% NaOCl (6).

Growth factors contained in the dentine matrix are released by endodontic irrigants (98–100). Lower concentrations of NaOCl are now recommended as 6% concentration has been shown to significantly decrease the survival of stem cells of the apical papilla (SCAP) (101, 102). A concentration of 1.5% NaOCl had minimal destructive effects on SCAP (102). Furthermore the use of 17% EDTA resulted in increased SCAP survival expression as well as partially reversing the deleterious effects of NaOCl (102). EDTA acts to demineralise the dentine and expose the dentine matrix which acts to release growth factors from the dentine matrix

(56, 99, 100). In cases where minimal filing has been undertaken, the use of EDTA and removal of the smear layer may expose binding sites for attachment of newly formed tissue to the canal walls (83, 86) as well as stem cell differentiation (56, 98). EDTA conditioning of dentin promoted the adhesion, migration and differentiation of dental pulp stem cells towards or onto dentin (99). Exposure of the dentin matrix by EDTA also appeared to increase the adherence of newly formed mineralized tissue to the root walls (56). Therefore, a final rinse with EDTA before creation of a blood clot is advised (99, 100).

Whilst chlorhexidine has been used as the final irrigant in REPs, 2% chlorhexidine has been reported to be cytotoxic effects on stem cells (101) and it interferes with cell adhesion onto the dentin surface (10, 103). Therefore the use of chlorhexidine should be avoided.

### *Placement of an intra-canal medicament*

In 2013 approximately 53% of all published regenerative endodontic cases were treated with an antibiotic paste as an intracanal medicament (6). By 2015, the number of published cases using an antibiotic paste had increased to 80% (9). The first report of revascularization in an infected immature tooth in 2001 used a double antibiotic paste of metronidazole and ciprofloxacin (76). The next report used a combination of metronidazole, ciprofloxacin and minocycline often referred to as '3mix' or 'triple antibiotic paste' (TAP) (6). The rationale for the use of TAP was a number of studies of endodontic infections in deciduous teeth where a combination of metronidazole, ciprofloxacin and minocycline at a concentration of 100 μg/mL of each drug completely eliminated cultivatable bacteria from infected root canals *in vitro* and *in vivo* (104, 105). Triple antibiotic pastes using this combination of antibiotics have been shown to reliably disinfect dentine in infected root canals with diffusion of the drugs throughout the entire root dentine (106, 107). The use of cefaclor instead of minocycline has also been shown to reliably eliminate bacteria in vitro and in situ (104). This alternative combination of TAP has been used in successful regenerative endodontic cases (108, 109). Other reports used amoxicillin instead of minocycline which showed continued root maturation (82, 87).

In early reports the combination of antibiotics were mixed with water, saline, or propylene glycol to make a thick creamy paste, achieving a required physical consistency rather than a specific concentration of the drugs. The concentration of these paste mixtures is about 1000 mg/mL (110). Studies have shown pastes at these concentrations are toxic and lethal to stem cells and papilla cells when in direct contact (111–114). The toxicity of the drug concentration had a concentration-dependent effect (113). The AAE protocol recommends TAPs at concentrations no greater than 0.1 mg/mL (72). At this concentration, TAP is conducive with stem cell survival and proliferation (57) and also effective in eliminating micro-organisms within the root canal (104, 105).

AAE guidelines also recommended calcium hydroxide (72). Calcium hydroxide has been shown to be conducive to SCAP survival and proliferation (111). Furthermore a study has shown that SCAPs had the highest survival when cultured on dentine exposed to calcium hydroxide when compared to dentine exposed to concentrations of 1 mg/mL or higher TAP (113). Clinically, an analysis of case reports that used calcium hydroxide as an intracanal medicament resulted in further root maturation (115).

Thus the concentrations of both irrigants and medicaments are important to find the balance between disinfection of the canal, alteration of the dentine matrix for the release of growth factors and survival and proliferation of SCAP. It is of note, that the group from University of Texas at San Antonio responsible for much of the aforementioned research use calcium hydroxide as their first choice of intracanal medicament for REPs (6). A recent study has shown that the attachment of human apical papilla cells to root dentin is higher when treated with calcium hydroxide rather than TAP (116).

### *Creation of a blood clot or protein scaffold in canal*

After disinfection of the canal and resolution of symptoms, REPs usually involve lacerating the periapical tissues to initiate bleeding or the use of platelet-rich plasma (PRP) or platelet-rich fibrin (PRF) (121). A review in 2015 reported that 87% of published studies utilised these approaches to provide a structure for the delivery of stem cells (9). Furthermore, these authors noted an increasing trend for not leaving the canal empty. REPs recommend that after irrigation of the intracanal medicament with 17% EDTA (30 mL for 10 minutes) bleeding be induced by rotating a pre-curved K-file size #25 at 2 mm past the apical foramen with the objective of filling the canal with blood to the level of the cemento-enamel junction (110). An important study demonstrated that the evoked-bleeding step in regenerative procedures triggers a significant accumulation of undifferentiated stem cells into the canal space (52). Contemporary regenerative endodontics follows principles of bioengineering with the foundation dependent on the interaction of stem cells, scaffolds and growth factors (110).

A number of studies reported difficulties in initiating a blood clot within the canal (108, 117, 118). In these cases, conventional apexification procedures provided a traditional treatment modality. However, a number of studies have used PRP/PRF autologous fibrin matrix (AFM) as they contain growth factors that have the potential of promoting differentiation of stem cells into the canal space (109, 119, 121). Unfortunately, a number of studies have failed to show that REPs utilising natural or synthetic scaffolds were able to regenerate pulp within the canals (57, 122). The tissues formed in the canals were mineralized tissue and some fibrous connective tissue with no pulp-like tissue, which would be characterized by the presence of odontoblast-like cells observed lining the dentine-like mineralized tissue (57). In a study comparing REP with blood clot and REP with blood clot + injectable scaffold found both groups allowed the continued development of roots in teeth with necrotic pulps.

However, the use of artificial hydrogel scaffold and basic fibroblast growth factor was not essential for repair (122).

### *Effective coronal seal*

Once a blood clot or scaffold is in place within the canal, a coronal barrier is placed to prevent coronal leakage of microorganisms. In 2015, 85% of studies used mineral trioxide aggregate (MTA) for this purpose (9). Current protocols recommend that when a blood clot is formed, a premeasured piece of Collaplug (Zimmer Dental Inc, Warsaw, IN) should be carefully placed on top of the blood clot to serve as an internal matrix for the placement of approximately 3 mm of white MTA (Dentsply, Tulsa, OK) followed by a 3–4 mm layer of glass ionomer layer (e.g., Fuji IX;GC America, Alsip, IL, or other) is placed over the MTA. A bonded reinforced composite resin restoration (e. g., Z-100;3 M, St Paul, MN, or other) is then placed over the glass ionomer (72, 110). MTA is a biocompatible material with bioactive properties that resists bacterial contamination (123). Glass ionomer cements have also been used as a coronal barrier (76, 90).

## Outcome assessment

In 2016, there were 80 publications of clinical studies that employed regenerative endodontic protocols with 379 treated teeth. There were 4 high-level cohort studies (LOE 2), 14 case series (LOE 4) and 62 case reports (LOE 5) (124). In 2015, there were 51 publications of clinical studies that employed regenerative endodontic protocols. There were 2 high-level cohort studies (LOE 2), 8 case series (LOE 4) and 41 case reports (LOE 5) (125). This increase in number of published papers in just one year illustrates the substantial research endeavor in the field of regenerative endodontics.

Case reports were instrumental in highlighting a clinical approach which was amenable to continued root maturation in the treatment of infected immature teeth with regenerative endodontic protocols. Unfortunately, case reports are low level evidence, with a bias towards over reporting of successful cases. It is also known that there have been a variety of REPs undertaken in clinical studies which have not employed standardized REPs (9). There is much variability in the use of irrigants, medicaments, instrumentation, type of material used as an intra-canal barrier and follow-up review. There are also differences in clinical outcomes as illustrated in Figs 26.4–26.13. However, all of these studies assess small sample sizes and therefore no reasonable statistical analysis of the varying protocols can be undertaken. Furthermore, contradictory and conflicting data exists between the studies. Further studies with larger cohorts and longer follow-up are required. As the 5 published cohort studies are the highest level of evidence to date their findings will be discussed next particularly as there are conflicting results on the efficacy of this technique when compared to placement of an apical barrier of MTA (Tables 26.1–26.6).

### *Cohort studies (LOE 2)*

Bose et al. (115) analyzed 54 REP treated teeth, and 40 control teeth (20 apexification and 20 non-surgical root canal treatments) retrospectively and used a geometrical imaging program NIH ImageJ with TurboReg plug-in to minimize differences in angulations between the preoperative and recall images to quantitatively calculate the continued development of root length and dentine wall thickness. The results showed regenerative endodontic treatment with triple antibiotic paste (TAP) and calcium hydroxide produced significantly greater increases in root length than either the MTA apexification or nonsurgical root canal therapy control groups. The triple antibiotic paste produced significantly greater differences in dentin root wall thickness than either the calcium hydroxide or formocresol groups. This study is

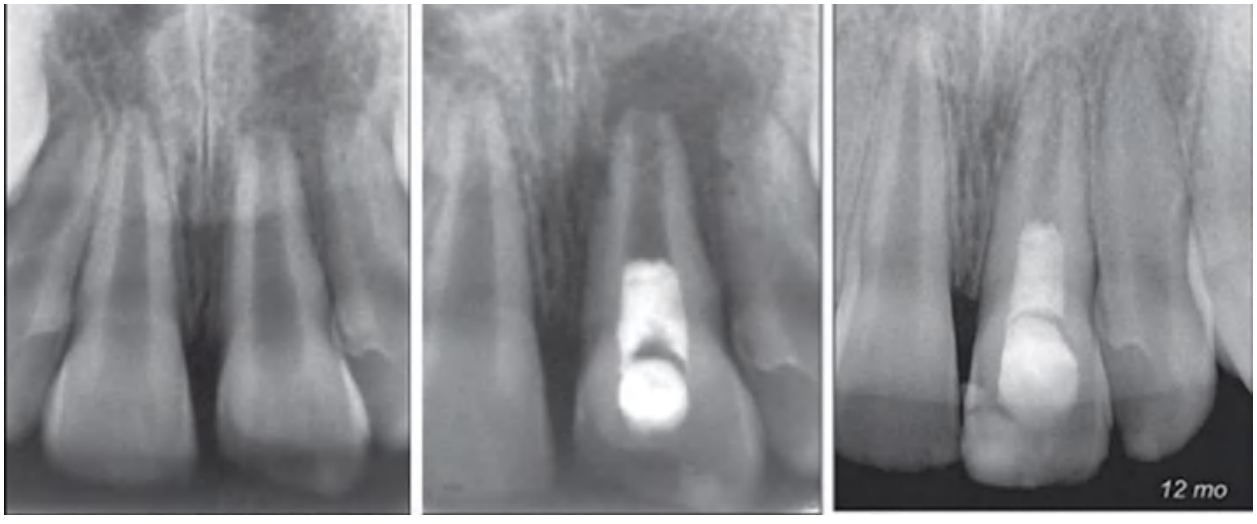


**Fig. 26.4** Traumatic injury causing uncomplicated crown fracture and pulp necrosis associated with apical periodontitis treated with REPs. A) Preoperative radiograph. B) Post-operative radiograph. C) At 12–month follow-up resolution of apical periodontitis, thickening of the canal walls and apical closure is evident as compared to the preoperative radiograph A. From Shimizu et al. (58) 2013. A quantitative analysis of this case with NIH ImageJ and TurboReg plug-in software showed that there had been a 16% increase in dentin root thickness but no change in root length.

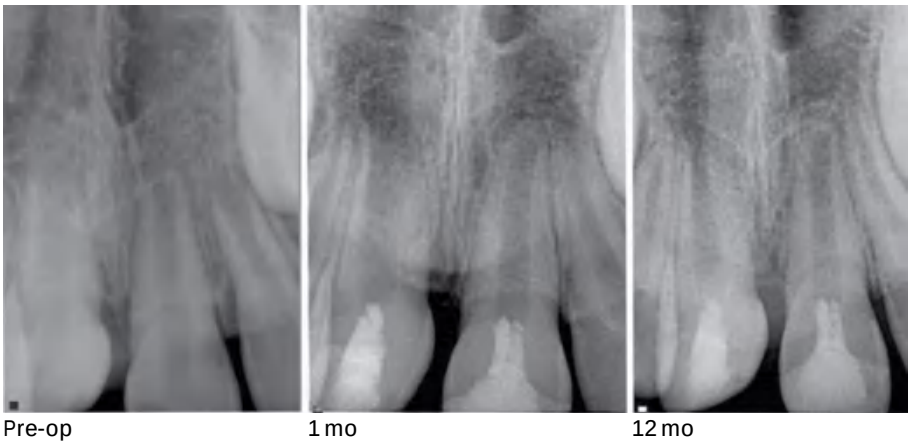


**Fig. 26.5** Traumatic injury to the maxillary central incisors resulting in pulp necrosis treated with REPs. A) Preoperative radiograph. B) Postoperative radiograph. C) at 12 months, the left central incisor showed continued root development with closed blunt apex consistent with Type 2 radiographic response. The right central incisor showed continued root development with open apex consistent with Type 3 radiographic response as compared to the preoperative radiograph.

**Fig. 26.6** Traumatic injuries to the maxillary central incisors resulting in a chronic periapical abscess to the right central incisor and apical periodontitis to the left central incisor treated with REPs. Severe calcification (obliteration) of the canal is observed in the left central incisor consistent with Type 4 radiographic response. The right central incisor radiographic response is consistent with Type 3 response with continued root maturation and an open apex as compared to the preoperative radiograph. A) Preoperative radiograph, B) Review at 18 months; C) Review at 3 years; D) Review at 5 years. Source: Reproduced from (87) with permission.

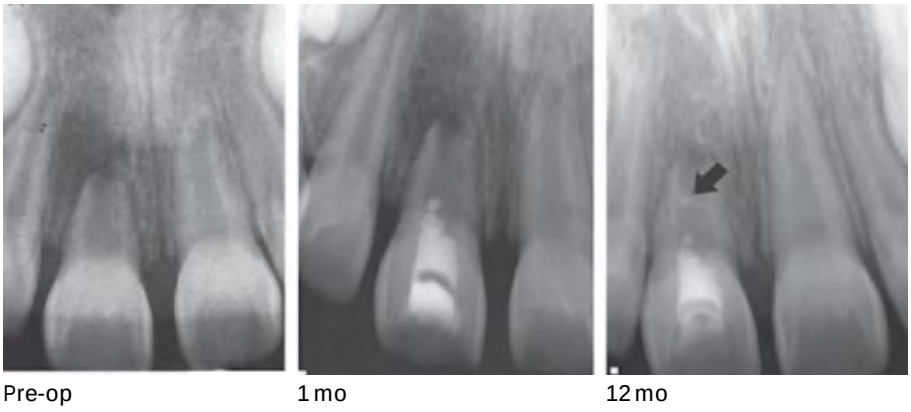


**Fig. 26.7** A maxillary right central incisor had a traumatic injury resulting in pulp necrosis and apical periodontitis treated with REPs. A) Preoperative radiograph. B), Postoperative radiograph. C) At 12-month review, resolution of apical periodontitis and a hard tissue barrier formation in the canal space between apex and coronal MTA plug is evident (arrow) consistent with Type 5 radiographic response. There was no obvious thickening of the canal walls or apical closure after REPs as compared to the preoperative radiograph.

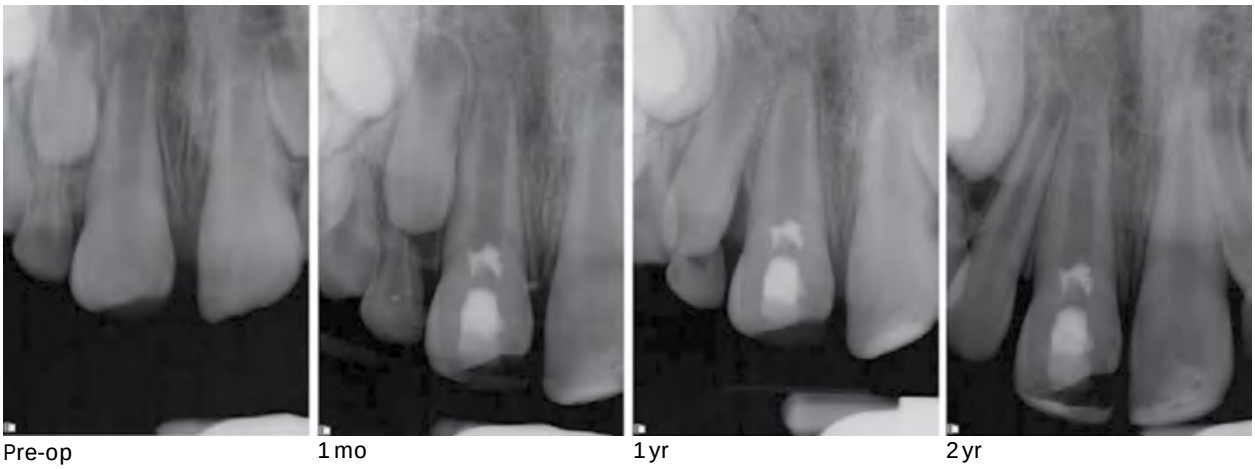


**Fig. 26.8** A maxillary right central incisor that sustained a complicated crown fracture and developed pulp necrosis treated with REPs. A) Preoperative radiograph. B) Postoperative radiograph. C) Radiograph at 1-year review. D) Progressive ingrowth of bone and a calcific bridge in the apical third of the root occurred over a 2-year review as compared to the preoperative radiograph.

**Fig. 26.9** A right maxillary central incisor that sustained an uncomplicated crown fracture developed asymptomatic apical periodontitis and noted at time of orthodontic assessment and treated with REPs. A) Preoperative radiograph. B) Postoperative radiograph. C) After 6-year follow-up some further root development is evident with apical closure as compared to the preoperative radiograph and some calcific material in the apical third of the root. The tooth was discolored and was bleached.

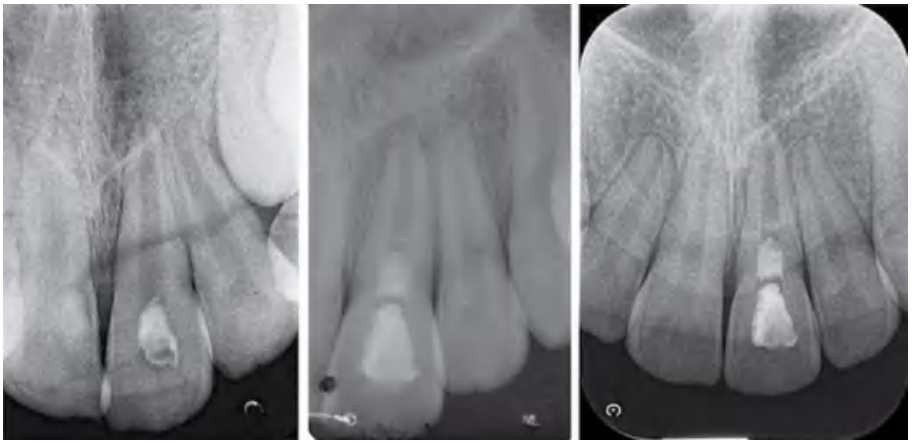


**Fig. 26.10** A maxillary left central incisor that sustained an uncomplicated crown fracture developed asymptomatic apical periodontitis treated with REPs. A) Preoperative radiograph. B) Postoperative radiograph at 18 months; C) Apical closure without substantial changes in root length or width is seen at 5 year review as compared to the preoperative radiograph. Source: Reproduced from (87) with permission.

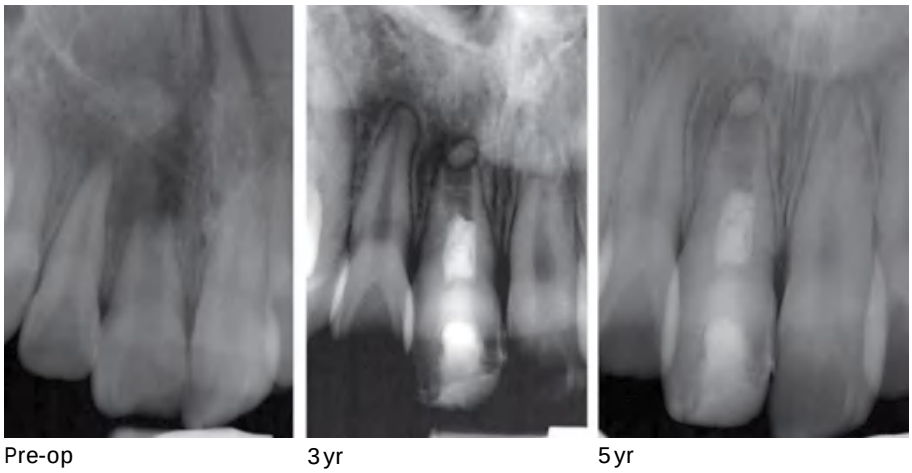


**Fig. 26.11** A maxillary left central incisor that sustained an uncomplicated crown fracture and then developed symptomatic apical periodontitis treated with REPs. A) Preoperative radiograph. B) Postoperative radiograph. C) No substantial change in root length or width as compared to the preoperative radiograph, though a calcific opacity noted at the apex that most likely is proliferation of Hertwig's epithelial root sheath to form some root development evident at 3 and 5-year review.

important as it showed quantitatively that increases in root length and dentine thickness occurred in teeth treated with REPs which did not occur in teeth treated with MTA apexification barrier placements.

Jeeruphan et al. (31) conducted a retrospective analysis to evaluate radiographic and clinical outcomes of immature infected teeth treated with calcium hydroxide apexification (22 teeth), MTA apexification (19 teeth) or revascularization (20 teeth). This study is often cited as REP treated teeth have a higher survival rate and greater changes in root length and width. However, there were different follow-up times for the 3 groups. While the survival rate for revascularization cases was 100%, only 80% satisfied a strict criterion for healed, with 20% determined as healing despite a follow-up of $21.15 \pm 11.70$ months (31). Therefore, the results may be overstated.

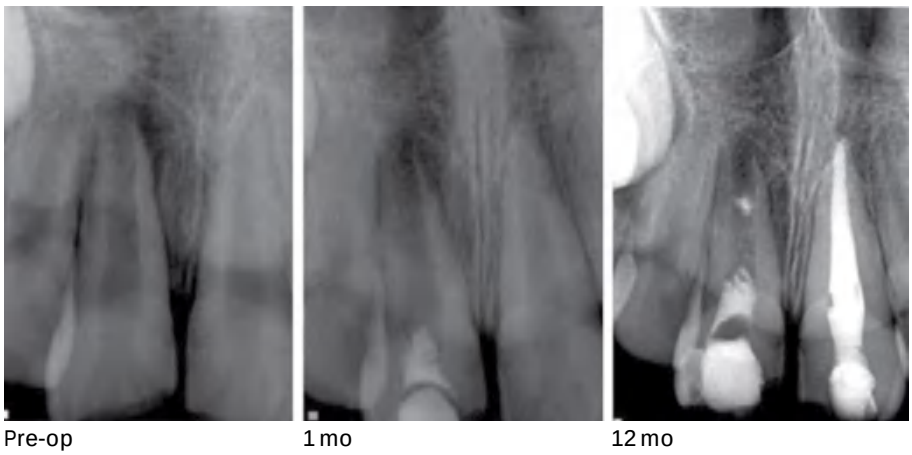


**Fig. 26.12** The maxillary right central incisor had a history of trauma causing uncomplicated crown fracture. Subsequently, the tooth developed apical periodontitis and treated with REPs. A) Preoperative radiograph. B) Postoperative radiograph. C) Healing of periapical lesion without apical closure and thickening of the canal walls at 12-month follow-up as compared to the preoperative radiograph. MTA is seen in the apical third of the canal illustrating the difficulty in placing MTA in the canal.

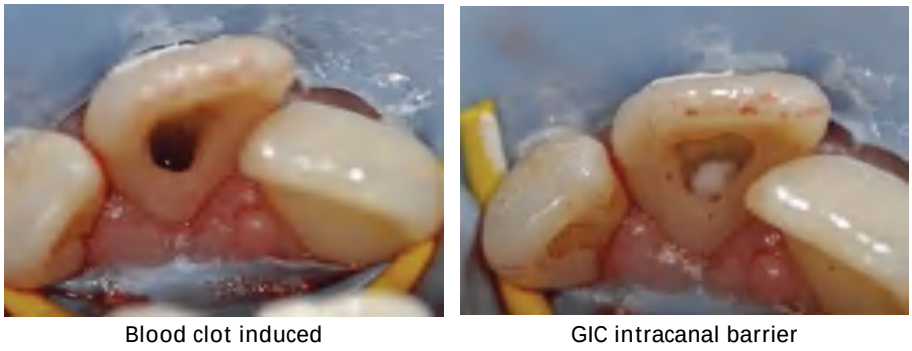

**Fig. 26.13** A maxillary right central incisor that developed pulpal necrosis and asymptomatic apical periodontitis following trauma treated with a REP of disinfection and development of A) a blood clot is induced. B) glass ionomer cement used as an intracanal barrier. C) Preoperative radiograph. D) No further root development was noted at a 3-year review as compared to the preoperative radiograph.

In a retrospective study, Alobaid et al. (126) also compared clinical and radiographic outcomes of endodontic treatment undertaken on immature nonvital permanent teeth by apexification and revascularization. In this study revascularization was not superior to other apexification techniques. This study differs from the 100% survival success of the Jeeruphan et al. study (31) and highlights the importance of case selection on clinical outcomes especially in a trauma cohort.

In a prospective study, Nagy et al. (122) compared clinical outcomes on 36 maxillary central permanent immature incisors with pulp necrosis and treated with (1) MTA apical plug, (2) REP with the blood clot as a scaffold and (3) REP with a blood clot and an injectable scaffold impregnated with basic fibroblast growth factor (FGF). After a follow-up of 18 months, the success rates for MTA, REP and FGF groups were 100%, 90% and 80%, respectively (122).

**Table 26.1** Type of teeth, REPs, follow-up and clinical outcome for LOE 2 cohort studies.

| Study | Teeth | Irrigants | Paste | Canal barrier | Follow-up | Clinical outcomes |
|---|---|---|---|---|---|---|
| Bose et al. | 94 teeth | Varying [] of NaOCl 3% $H_2O_2$ | TAP CH Formocresol | Not stated | 6- > 36 months | Landmark study that validated quantitative assessment of further root maturation. Teeth treated with REPs showed greater increases in further root maturation than MTA apexification. |
| Jeeruphan et al. | 36 incisors 25 premolars | 2.5% NaOCl | TAP | Not stated | 21 months ±11.7 | Teeth treated with REPs showed significantly greater changes in root length and width than MTA and CH apexification groups. The survival rate for teeth treated with REPs was higher. |
| Nagy et al. | 36 incisors | 2.6% | TAP | Not stated | 18 months | Teeth treated with REPs showed further root maturation when compared to MTA apexification. |
| Alobaid et al. | 31 incisors | Varying [] of NaOCl | TAP BAP CH | MTA | Average 17 months | In this study teeth treated with REPs were not superior to apexification techniques. Although more teeth treated with REPs showed increased root area. |
| Saoud et al. | 20 incisors | 2.5% NaOCl | TAP | MTA | 12 months | Teeth treated with REPs showed increased root width, length and decrease in apical diameter although in many cases changes were quite small and unlikely to be clinically significant. |

TAP based on Hoshino; BAP minocycline excluded; CH calcium hydroxide.

It is noteworthy that the cohort studies by Alobaid et al. (126) and Nagy et al. (122) where trauma was the etiology of pulp necrosis reported apexification with placement of an apical barrier of MTA showed better outcomes than REPs when compared to Jeeruphan et al. (31) where the majority of teeth were premolars with fracture of the occlusal tubercle and evaginatus as the etiology of pulp necrosis. There is a difference in the progression of pulp necrosis with trauma related necrosis being the result of bacterial contamination from a lacerated periodontal ligament into an avascular pulp space whereas pulp necrosis in a premolar tooth with a fractured evaginatus is coronal contamination of bacteria into a vital pulp. It is unknown whether this affects the potential of stem cell of the apical papilla.

Saoud et al. (127) treated 20 incisor teeth with a history of trauma and pulp necrosis of which 80% of the teeth had sustained complicated crown fractures. Teeth showed a statistically significant increase in radiographic root length and width and decrease in apical diameter, although the changes in many cases were quite small that the clinical significance was questionable. However, apical closure occurred in 55%, increase in root length 5% and increase in root width of 21% at 12 months (127).

In the studies of REPs for immature teeth with necrotic pulps, the criteria used to evaluate periapical healing and measure the increased thickness of the canal walls and continued root development vary considerably among all studies. The periapical index (PAI) (128) should be used to evaluate periapical healing as some studies have claimed healing is occurring despite the presence of significant persistent radiolucencies being present at 1 year reviews (129), and ImageJ software program and TurboReg plugin (115) be employed to measure the changes in thickness of the canal walls and root length of teeth after regenerative procedures at different follow-up times to standardize the observation.

## Are outcomes different for teeth that have sustained trauma?

As discussed earlier, the etiology of pulp necrosis is different. For premolars with a dens evaginatus, pulp necrosis is due to bacterial invasion through fractured tubercle into pulp cavity and likely to be more long standing than in teeth with an etiology of trauma. Pulp necrosis in trauma cases is usually the result of bacterial invasion from a lacerated periodontal ligament into an avascular coronal part of the pulp. Conversely, pulp necrosis in a premolar tooth with a fractured evaginatus is a consequence of coronal contamination of bacteria into a vital pulp. While some case series (129–137) have shown incisor teeth with an etiology of pulp necrosis all showed continued root maturation and thus have the potential for continued maturation following REPs, other case series showed variable results (86, 87) and even no further root maturation (71). Trauma to immature teeth might cause additional impact damage to Hertwig's epithelial root sheath, which regulates the root maturation. So the potential for further root maturation is inconsistent (118) and patients and their parents should be advised that root maturation might not occur.

## Regenerative endodontic procedures for other pathological conditions associated with traumatized teeth

Besides pulp necrosis, traumatic injuries to immature and mature permanent teeth also can cause other types of pathological conditions, such as external inflammatory root resorption, horizontal root fracture and complete avulsion. These pathological conditions are usually treated with control of root canal infection using chemo-mechanical debridement and root filling (10, 73). Recently, REPs have been employed to manage traumatized teeth with horizontal root fracture, external infection related root resorption after avulsion and replantation.

Traumatized teeth with external infection related root resorption were shown to be managed with REPs, resulting in resolution of apical periodontitis and arrest of external root resorption (138). Traumatized tooth with horizontal root fracture was also successfully treated with the concept of REPs, achieving healing of root fracture by hard tissue formation (139). An avulsed permanent mature incisor with more than 8 hours extra-oral dry time was replanted into the alveolar socket after complete chemo-mechanical debridement of the canal space and enlargement of the apical foramen to 1.5–2 mm. The tooth was then treated with regenerative endodontic therapy, using platelet-rich plasma as scaffold. At 12-month follow-up, the tooth showed arrest of internal and external infection related resorption (140). In another case, following intrusion of a mandibular incisor treated in a single visit showed healing of the severe infection related external resorption and continuous root development with dentin wall thickening in the apical third of the root at 24 month review (97). A recent report of three cases utilized REPs for the treatment of mature teeth with a horizontal root fracture and another tooth with perforating resorption. The horizontal root fracture showed hard tissue healing after the blood clot was induced from the apical fragment. There was complete healing of the periapical lesion for the tooth with perforating root resorption. A third case involved an avulsed immature tooth that subsequently root fractured after a secondary trauma and showed connective tissue healing between the fractured fragments (141). REPs should be explored more in the management of other pathological conditions associated with traumatized teeth.

## Is revascularization an effective and reproducible technique?

A recent systematic review reported that 97 of 101 teeth (96%) treated with REPs between 2001 and 2014 were successfully treated as measured by some degree of apexogenesis including increased root length, increased root thickness and apical closure. Only 4 teeth (4%) failed to show no further root maturation. Apical closure was detected in 55.4% of teeth with increased root length (76.2%) and increased root width (79.2%) reported more frequently. These outcomes were independent of clinical variables such as operator and material selection or differences in protocols (142). The finding of increased root length of 79.2% is at odds with other LOE 2 cohort studies that reported 5% (126), 15% (31) and less than 20% (127) changes in root length. Saoud et al. (127) suggested that severe trauma has the potential to produce damage to the Hertwig epithelial root sheath and/or the apical papilla and that teeth subjected to trauma may be less likely to achieve clinically meaningful continued root development than treated immature teeth with pulpal necrosis caused by caries or dens evaginatus. This review had strict inclusion criteria and only 33 case reports were included in the analysis (142). Many of the cohort (31, 122, 126, 127) and case series (87, 136, 137) listed above were not included. Kontakiotis et al. (125) reported 51 publications over a lesser time frame so many reports were excluded and may have affected the findings.

The review (142) also did not report any instances of persistent infection yet many of the higher evidence studies reported that clinical symptoms persisted (86, 87, 117, 122). The presence of infection is thought to preclude any possibility of continued root maturation (93, 143). A tooth treated with REPs and extracted for orthodontic management and then histologically examined showed the presence of inflammatory cells in the coronal and middle third of the canal of a tooth that at the 2 year review showed only narrowing of the root apex without root lengthening and only minimal thickening of the canal walls (59). Another histologic and histobacteriologic observations of failed cases that utilized REPs but after 16 months pain and periapical swelling developed. Bacteria were observed in the root canal space primarily in the apical portion of the root as well as biofilms on the root wall that penetrated into the dentinal tubules. In this report, any tissue within the canal was completely destroyed (79). It was suggested that mechanical debridement might be required to disrupt biofilms to improve REP outcomes. Also current disinfectants employed in REPs may not create the appropriate microenvironment required for dentin and pulp regeneration (143).

Many reports elaborate the difficulty in introducing a blood clot within the canal (78, 87, 108, 126, 132, 136). The use of an anesthetic without a vasoconstrictor is advised to facilitate bleeding within the canal. Bending the file to lacerate the apical tissue is also recommended. Lenzi and Trope postulated an incisor tooth that failed to show continued root maturation might have been due to the disintegration of the blood clot leaving an empty space in the canal and subsequently no scaffold to support regeneration or repair within the canal space (78). However, a radiopaque hard tissue barrier at the apex was evident and was described as auto-apexification. Other authors have described auto-apexification in a tooth not treated with REPS and attributed the calcific barrier to be due to the viability of the Hertwig's epithelial root sheath (144). It is unknown why some teeth show no further root maturation but a disruption of the blood clot is a reasonable hypothesis. The use of cell-free scaffolds that employ growth factors and chemoattractants described earlier may improve the outcomes of REPs in the future.

The ingrowth of bone into the canal as shown in Figs 26.8 and 26.9 could also result in ankylosis and an

adverse outcome (73). The long term prognosis for repair with the ingrowth of cementum, PDL and bone repair is not known (73).

The placement of an intracanal barrier of MTA has been reported to be technically challenging (87, 136) and illustrated in Fig. 26.12 where MTA is seen deep into the canal. Therefore, the use of Collatape is recommended so placement of MTA is limited to the cervical third of the root (72, 108, 132).

## Radiographic responses of teeth treated with REPs

Chen et al. (134) described 5 types of responses to the REPs

- Type 1: Increased thickening of the canal walls and continued root maturation (Fig. 26.4)
- Type 2: No significant continuation of root development with the root apex becoming blunt and closed (Fig. 26.5)
- Type 3: Continued root development with the apical foramen remaining open (Fig. 26.5)
- Type 4: Severe calcification (obliteration) of the canal space (Fig. 26.6)
- Type 5: A hard tissue barrier formed in the canal between the coronal MTA plug and the root apex (134) (Fig. 26.7)

Other types of radiographic responses are also illustrated.

- Ingrowth of bone and adjacent calcific barrier in the apical third of the root (Fig. 26.8)
- Ingrowth of bone and some increase in dentine width without apical closure (Fig. 26.9)
- Apical closure evident though no substantial changes in root length or width (Fig. 26.10)
- No substantial change in root length or width though a calcific opacity noted at the apex that most likely is proliferation of Hertwig's epithelial root sheath to form some root development (Fig. 26.11)
- Healing of periapical lesion without apical closure and thickening of the canal walls or any change in root length (Fig. 26.12)

## Discoloration

Many studies have shown that discoloration is a significant problem following regenerative endodontic treatment (124). This is of particular concern for traumatized anterior teeth as appearance and pleasing esthetics are patient centered outcomes. Discoloration is more often associated with TAP that includes minocycline though discoloration has also been reported with calcium hydroxide. Kahler et al. (87) noted discoloration in 10 out of 16 cases (62.5%) in a prospective study that treated 16 teeth, of which 13 were incisors, with a TAP that substituted minocycline for amoxicillin. McTigue et al. (136) noted discoloration in 14 of 32 teeth (44%), of which 29 were incisors.

Dabbagh et al. (108) replaced minocycline with cefaclor, which resolved the issue of discoloration. It would also appear that discoloration is less likely when calcium hydroxide is used as the intracanal medicament, as Nagata et al. (137) reported only 3 of 11 teeth discolored with calcium hydroxide and chlorhexidine as opposed to 10 out of 12 with TAP that included minocycline. Berkhoff et al. (145) evaluated the removal of TAP using different irrigation procedures and found that approximately 88% of TAP was retained in the root canal system regardless of the irrigation technique used, and TAP was present circumferentially up to 350 μm within the dentin. Conversely, up to 98% of calcium hydroxide was removed and was only present in 50 μm of dentin (145). In a spectrophotometric analysis of crown discoloration induced by various antibiotic pastes used in revascularization, TAP had a significant association with discoloration. However, calcium hydroxide showed no color changes exceeding the perceptibility threshold (146). MTA which is the most commonly used material as an intacanal barrier has also been shown to discolor teeth (147).

Kirchoff et al. (148) in a study of extracted teeth, reported that teeth treated with TAP which included minocycline can be bleached successfully with sodium perborate paste. However, McTigue et al. (136) reported only 11 of the 14 discolored teeth they treated could be bleached satisfactorily. Kim et al. (149) also reported unsuccessful attempts to bleach discolored teeth treated with TAP that included minocycline. Therefore patients should be advised that discoloration of the tooth is often associated with REPs as stated in the AAE guidelines (72).

## Follow-up

Most studies have undertaken 6-month radiographic reviews to assess further root maturation. Bose et al. (115) showed that radiographic changes could be detected as early as 6 months though greater percentage changes occurred between 12 and 30 months. A study utilizing quantitative analysis showed progressive maturation of 2 teeth at 1 month and 36 month reviews (87). Long term follow-up of teeth treated with REPs are illustrated in Figs 26.6, 26.9 and 26.13.

## Clinical REP technique

As aforementioned, REPs for immature permanent teeth with non-infected and infected necrotic pulps in humans varies considerably in terms of concentrations of sodium hypochlorite irrigant and triple antibiotic paste in all published studies. There is no standard protocol of REP. The American Association of Endodontists (AAE) suggests a Clinical Considerations for a Regenerative Procedure for its members (Tables 26.2–26.6) (72).

**Table 26.2** Case selection.

| Case selection |
|---|
| Tooth with necrotic pulp and an immature apex |
| Pulp space not needed for post/core, final restoration |
| Compliant patient/parent |
| Parents not allergic to medicaments and antibiotics necessary to complete procedure |

**Table 26.3** Informed consent.

| Informed consent |
|---|
| Two (or more) appointments |
| Use of antimicrobial(s) |
| Possible adverse effects: staining of crown/root, lack of response to treatment, pain/infection |
| Alternatives: MTA apexification, no treatment, extraction (when deemed non-salvageable) |

**Table 26.4** Clinical procedure.

| First appointment |
|---|
| Local anesthesia, dental dam isolation and access |
| Copious, gentle irrigation with 20 mL NaOCl using an irrigation system that minimizes the possibility of extrusion of irrigants into the periapical space (e.g., needle with closed end and side-vents, or EndoVac™. Lower concentrations of NaOCl are advised [1.5% NaOCl (20 mL/canal, 5 min) and then irrigated with saline or EDTA (20 mL/canal, 5 min), with irrigating needle positioned about 1 mm from root end, to minimize cytotoxicity to stem cells in the apical tissues. |
| Dry canals with paper points |
| Place Calcium Hydroxide $Ca(OH)_2$ or low concentration of triple antibiotic paste. If the triple antibiotic paste is used: 1) consider sealing pulp chamber with a dentin bonding agent [to minimize risk of staining] and 2) mix 1:1:1 ciprofloxacin: metronidazole: minocycline to a final concentration of 0.1 mg/mL. |
| Deliver into canal system via syringe |
| If triple antibiotic is used, ensure that it remains below CEJ (minimize crown staining) |
| Seal with 3–4 mm of temporary restorative material such as Cavit™, IRM™, glass ionomer or another temporary material. |
| Dismiss patient in 1–4 weeks |
| **Second appointment (1–4 weeks after first visit)** |
| Assess response to initial treatment. If there is signs/symptoms if persistent infection, consider additional treatment time with antimicrobial, or alternative antimicrobial |
| Anesthesia with 3% mepivacaine without vasoconstrictor, dental dam isolation. |
| Copious, gentle irrigation with 20 mL of 17% EDTA |
| Dry paper points |
| Create bleeding into canal system by over-instrumenting (endo file, endo explorer) (induce by rotating a pre-curved K-file at 2 mm past the apical foramen with the goal of having the entire canal filled with blood to the level of the cemento-enamel junction.) An alternative to creating of a blood clot is the use of platelet-rich plasma (PRP), platelet rich fibrin (PRF) or autologous fibrin matrix (AFM). |
| Stop bleeding at a level that allows for 3–4 mm of restorative material |
| Place resorbable matrix such as CollaPlug™, Collacotre™, CollaTape™ or other material over the blood clot if necessary and whit MTA as capping material. |
| A 3–4 mm layer of glass ionomer (e.g. Fuji IX™, GC America, Alsip, IL) is flowed gently over the capping material and light-cured for 40 s. MTA has been associated with discoloration. Alternatives to MTA (such as resin-modified glass ionomer [RMGI] or bioceramics [e.g., Biodentine®]) should be considered in teeth where there is an aesthetic concern. |
| **Anterior and premolar teeth –** Consider the use of Collatape/ Collaplug and restoring with 3 mm of a non-staining restorative material followed by bonding a filled composite to the bevelled enamel margin. |
| **Molar teeth or teeth with PFM crown** – Consider use of CollaTape/CollaPlug and restoring with 3 mm of MTA, followed by RMGI, composite or alloy. |

**Table 26.5** Follow-up.

| Follow-up |
|---|
| Clinical and Radiographic exam |
| No pain, soft tissue swelling or sinus tract (often observed between first and second appointments). |
| Resolution of apical radiolucency (often observed 6–12 months after treatment). |
| Increased width of root walls (this is generally observed before apparent increase in root length and often occurs 12–24 months after treatment). |
| Increased root length. |
| Positive Pulp vitality test response |

**Table 26.6** The degree of success of REPs is largely measured by the extent to which it is possible to attain primary, secondary and tertiary goals.

| Follow up goals |
|---|
| Primary Goals |
| The elimination of symptoms and the evidence of bone healing |
| Secondary Goals |
| Increased root wall thickness and/or increased root length (desirable, but perhaps not essential) |
| Tertiary Goals |
| Positive response to vitality testing |

## Are teeth treated with REPs stronger?

A major limitation of REPs is that the placement of an intracanal barrier of 3 mm of MTA in the coronal third of the canal prevents thickening of the dentine in the coronal third of the root (71, 87). Furthermore teeth subjected to trauma may well have sustained crown fractures and/or crazing that initiate a fracture in the crown that could subsequently be propagated by occlusal forces or further trauma.

As discussed earlier, it is assumed that further root maturation will decrease the risk of fracture and tooth loss. However, no studies have examined this assumption. Furthermore, outcome studies show mixed results on whether MTA apexification or teeth treated with REPs have a more successful outcome (31, 122, 126). In the cases, where further root maturation does not occur (Fig. 26.12) the teeth presumably remain weak (71, 134). Indeed, incidences of root fracture following REPs have been reported (58, 87). It is also likely that cementum repair of immature teeth with necrotic pulps after REPs is not as structurally strong as true regeneration of the pulp dentine complex.

Reinforcement of the coronal third of the root canal following apexification has been recommended as it has been shown to increase the fracture resistance of teeth (150, 151). Alternatively, the use of fiber reinforced composite posts may strengthen the teeth (152). However, it is not known which protocol would be most likely to reduce the risk of root fracture in immature teeth.

## Publication bias and assessment of outcomes

There must be a publication bias in the reports of regenerative outcomes as it is likely that successful cases are being submitted for publication. Prospective studies show that

clinical symptoms can persist with ongoing infection within the root canal system (86, 87, 117). Indeed in a number of studies, teeth originally treated with REPs subsequently were treated with apexification barrier placements due to persistent symptoms (117, 122). It is important that future studies accurately assess the success and failure of REPs that have been undertaken in the endodontic literature for conventional endodontic procedures (153). As outlined in the AAE guidelines, the primary goal of REPs is to eliminate symptoms and observe evidence of bony healing. It is hoped that the secondary goal of increases in root wall thickness and root length can also become more consistently achieved as REPs utilize bioengineering techniques. Indeed, REPs based on the blood clot and wound healing protocol have been described as a paradigm lost (154). A new paradigm in regenerative endodontics is emerging based on concepts of tissue engineering where clinical protocols do not damage stem cells and scaffolds imbued with growth factors and chemoattractants. In addition to traditional calcium hydroxide or MTA apexification, REP provides another feasible treatment option for immature teeth having necrotic pulps with the potential of thickening of the canal walls and/or continued root development (125). However as discoloration following REPs is a particular concern in anterior teeth and studies have shown that further root maturogenesis is not consistently achieved, the clinician should make patients aware of these uncertainties associated with REPs before advocating treating teeth with REPs over the more traditional apexification and barrier techniques. While REPs may promote further root maturogenesis, key factors such as the type of tissue formed in the repair process and the long term prognosis are not fully understood. Although at the present time there are no randomized clinical studies into the long-term usefulness of REP, the promise and potential of this approach deserves serious consideration;but cannot fully replace the established MTA procedure for the treatment of immature teeth following trauma.

## What does the future hold?

Currently, there are two approaches to pulp tissue regeneration in regenerative endodontics, namely cell-free and cell-based. Both approaches are based on the concept of tissue engineering applying stem cells, bioactive growth/differentiation factors and biomimetic scaffold. The cell-free approach involves homing of endogenous stem cells, transplantation of scaffold and growth factors or chemoattractants in the canal space (65, 66). Clinical REPs can be considered a cell-free approach. The cell-based approach employs transplantation of exogenous stem cells by ex vivo expansion and scaffold in the canal space (155, 156). Technically, cell-free is simpler than cell-based approach because the former does not have to be concerned about stem cell source, and stem cell isolation. However, in the cell-free approach the endogenous stem cells are not pulp tissue specific. They can be from periapical tissues or bone marrow. The cell-based approach employs pulp tissue specific stem cells, such as dental pulp stem cells (157) stem cells from exfoliated deciduous teeth (158) and stem cells from apical papilla (162). These stem cells have been shown to be capable of differentiating into odontoblasts and produce dentin (156–159). Nevertheless, several problems are associated with cell-based approach, such as limitation of stem cell source, isolation of stem cells, ex vivo expansion of stem cells, contamination, mutation, stem cell bank, cost, regulatory issues and clinician's ability to perform stem cell transplantation (160, 161). Basically, both cell-free and cell-based approaches of pulp tissue regeneration are still in the preclinical stage of experiments (157). However, from future prospect pulp tissue regeneration appears to be an attainable goal, based on the concept of stem cell-based pulp tissue engineering (162).

## Essentials

### Aims

Regenerative endodontic protocols have been described as a "paradigm shift" in the treatment of immature teeth with pulp necrosis and an infected root canal system due to the potential for further root maturation. It was clinically hypothesized that REPs could regenerate new pulp tissue in the disinfected root canal space of immature permanent teeth with necrotic pulps. Many different irrigants, medicaments and materials have been used. There is a lack of standardization of the protocols employed in published studies; therefore, meaningful analysis of the best technique is not possible. In April 2015 the AAE released a suggested protocol, which allows for more consistency in further studies on the efficacy of this technique.

### Technique

The REP is primarily for immature permanent teeth with necrotic pulps in young patients. The protocol generally requires disinfection of the canal with 1.5% NaOCl, either triple antibiotic paste or calcium hydroxide to be used as a medicament. At the second appointment a blood clot is induced to bring scaffold and stem cells into the canal by provoking periapical tissues that allow for formation of new tissues in the root canal space and further root maturation. MTA is placed as an intracanal barrier and the tooth restored with composite resin.

### Outcomes

- Primary goal (essential): the elimination of symptoms and the evidence of bony healing
- Secondary goal (desirable): increased root wall thickness and/or increased root length
- Tertiary goal: positive response to vitality testing

The primary goal of REPs is often achieved with the elimination of symptoms and evidence of bony healing. However, clinical studies show that the secondary goal of further

root maturation is not consistently achieved with variable changes in root maturation possible. An increase in root wall thickness appears to be more routinely achieved than an increase in root length. This may be a consequence of laceration of Hertwig's epithelial root sheath when inducing a blood clot by provoking periapical tissues into the root canal space, and perhaps long-standing chronic apical periodontitis. No high-level of evidence of studies have shown that REPs have better outcomes than traditional MTA barrier plug of immature permanent teeth with necrotic pulps in terms of elimination of clinical symptoms/signs and resolution of apical periodontitis as well as cervical root fractures.

## Difficulties with the technique

- Discoloration of the crown is often associated with this technique and patients and their parents should be advised of this risk. Discoloration is more likely if the TAP contains minocycline.
- If calcium hydroxide is used as an intracanal medicament it should only be placed in the coronal half of the root canal space
- At the second appointment an anesthetic without a vasoconstrictor should be used as it can be difficult to induce bleeding into the canal space
- The use of Collatap®is recommended when placing MTA as an intracanal coronal barrier to control the placement of this material to just apical of the cemento-enamel junction
- Persistent infection is possible and may be related to minimal or no filing advocated with this technique. Patients should be advised that the tooth may have to be treated with apical barrier techniques
- Tissue repair rather than true regeneration of the pulp is achieved with current REPs
- It is unknown whether REPs strengthen teeth as placing MTA in the coronal third of the root prevents further root maturation of this part of the root where cracks in the coronal tooth structure as a result of trauma may propagate
- A lack of randomized, prospective clinical trials and long term follow-up studies are required to fully understand the efficacy of REPs for the treatment of infected immature teeth

Therefore clinicians should consider carefully whether to undertake technically challenging REPs or the traditional MTA as an apical barrier.

## The potential and promise of the technique

It is hoped that emerging protocols, which employ scaffolds, growth factors, chemo-attractants, stem cells and bioengineering protocols will result in consistent root maturation. Many issues have to be resolved before tissue-engineering technology can be applied in clinical practice. However, like in medicine this is an exciting and emerging field of research where future pulp tissue regeneration appears to be an attainable goal based on the concept of stem cell-based pulp tissue engineering.

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# 27

# Reinforcing Endodontically Treated Teeth

A. Kishen

## Introduction

Fractures in endodontically treated teeth are not a rare occurrence in clinical practice. Reinforcing endodontically treated teeth is a significant objective that involves several challenges and is not considered to be a straightforward step. Many physiological, pathological and iatrogenic factors have been discovered to be responsible for compromising the mechanical integrity of restored endodontically treated teeth. Understanding the ultrastructure of dentin, the major hard tissue in teeth, and the response of tooth structure to chewing forces would provide us with a foundation, based on which (a) perplexing incidents can be avoided in clinical practice by judiciously recognizing risk factors for fracture and (b) essential clinical steps could be implemented that would decrease the susceptibility of endodontically treated teeth to fracture.

## Structural consideration in endodontically treated teeth

Dentin is a mineralized tissue that occupies the main bulk of a tooth. It is regarded as a biological composite made up of an organic fraction (30 wt. %), an inter-penetrant inorganic fraction (60 wt. %) and water (10 wt. %) (1–4). The inorganic phase in dentin is composed of poorly crystalline hydroxyapatite with needle and/or plate like morphology (10 nm × 50 nm), which is present both within the collagen fibrils (intra-fibrillar) and between fibers (inter-fibrillar) on a nanometric scale. Ninety percent of the organic phase is collagen, which is exclusively Type I collagen (5, 6). Type I collagen is a strong, three-dimensional fibrous biopolymer that is typically found in an aqueous biological environment. It often is associated with proteoglycan molecules that contain large amounts of bound water (7). The water content of dentin varies with location. One type, which is *tightly bound* in nature, is associated with the apatite crystal of the inorganic phase, and collagenous/non-collagenous matrix proteins of the organic phase. The second type of water is the *free or unbound water*. The free water fills the dentinal tubules and other porosities in dentin and transports inorganic ions within the dentin matrix (8). Water at an optimum level of hydration, will act as a plasticizer molecule, maintaining the soft and pliable nature of the dentin matrix (9). Time-dependent properties (ex: *viscoelasticity*), which are characteristic of dentin matrix are contributed by the free water in dentin (10). The water in dentin matrix also contributes distinctly to the fracture toughening mechanisms in dentin. The inorganic fraction in dentin contributes to the elastic modulus and compressive strength, while the organic fraction of the dentin contributes to the toughness and tensile strength of dentin (11). Thus, all major constituents in the dentin are known to contribute to its mechanical integrity (Fig. 27.1).

## Biomechanical considerations in endodontically treated teeth

Intact natural teeth display a distinct manner of distributing chewing forces. Tooth structure generally experiences flexing or bending stresses during chewing. The structural adaptations of dentin allow uniform transfer of stress and minimize stress increase or concentrations within a tooth. The maximum degree of these stresses is transferred at the cervical region of the tooth (Fig. 27.2). The bending nature of stress distribution reduces markedly towards the root apex (12). Typically, when a tooth functions, there is more

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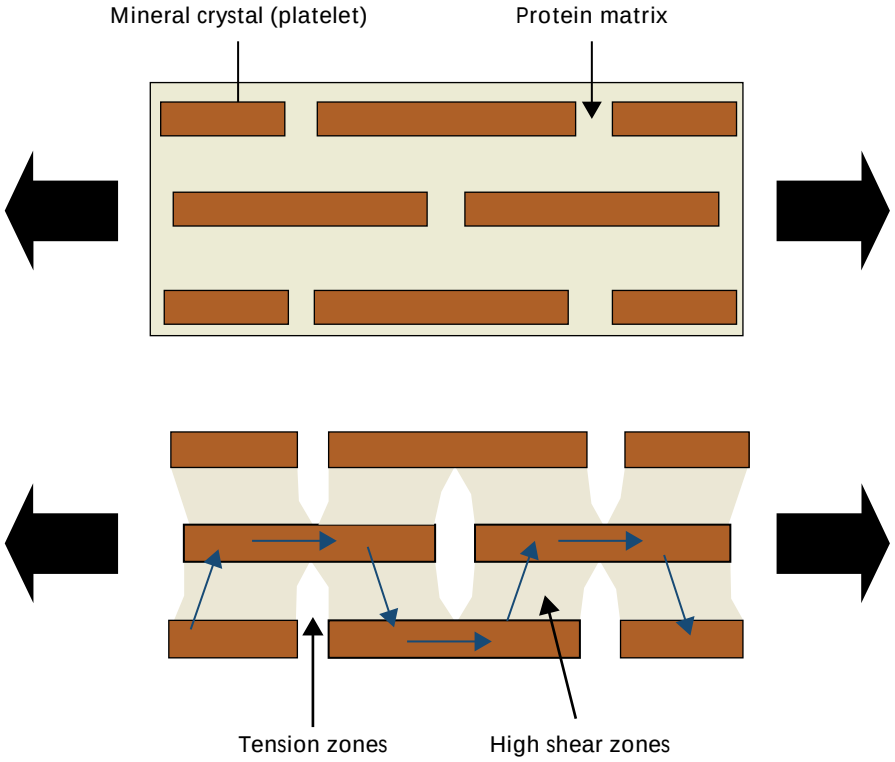


**Fig. 27.1** Schematic diagram showing a model of a natural biocomposite. (A) The arrangement of staggered mineral crystals embedded in a protein matrix. (B) A basic model showing the load-carrying structure of the mineral–protein composites. The load is chiefly carried by the mineral platelets whereas the protein transfers load via the high shear zones between mineral platelets. Source: Reproduced from (50) with permission.

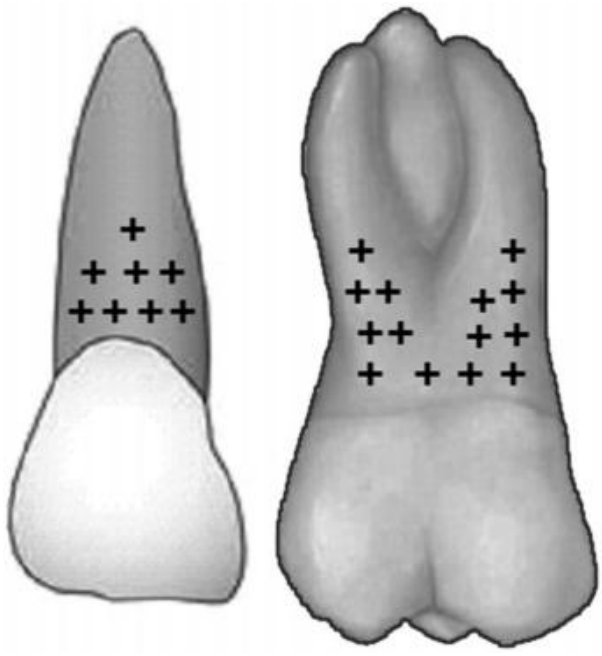

**Fig. 27.2** Shows that the cervical root dentin distributes maximum degree of bending stresses in teeth. The magnitude of bending stress distribution reduces markedly towards the root apex.

generalized distribution of compressive stresses in the root structure. The nature of stress distribution in a tooth is influenced by the coronal tooth structure, direction of occlusal forces (functional or parafunctional), the bulk of the cervical dentin, shape of the root and the relationship between root and surrounding alveolar bone (11).

## Fractures in endodontically treated teeth

Fractures in endodontically treated teeth are typically fatigue failures, which is a complex process that involves nucleation and growth of micro-cracks within dentin. Knowledge of how cracks are formed and how they propagate in dentin is important to comprehend the causes of fractures in endodontically treated teeth. In a fatigue failure, microscopic cracks tend to grow with time, eventually resulting in fracture. Fracture originates from a location of *stress concentration*, which can be a micro-defect, or notch with sharp corner or hole. The tensile strength of dentin is much lower than its compressive strength, thus any *tensile stress* with magnitudes high enough to produce microscopic plastic deformation at the crack tip may serve as the driving force for crack propagation (13). Still, it is not essential to apply tensile stress for crack propagation. Crack propagation may be governed by the residual stresses formed in dentin due to loss of free water or due to the loss of tooth structure or due to elastic modulus mismatch between restorations/devices and dentin. So, any variation in the mechanical characteristics of dentin, alongside any discrepancy in the biomechanical response of the tooth would predispose restored endodontically treated teeth to fracture (Fig. 27.3).

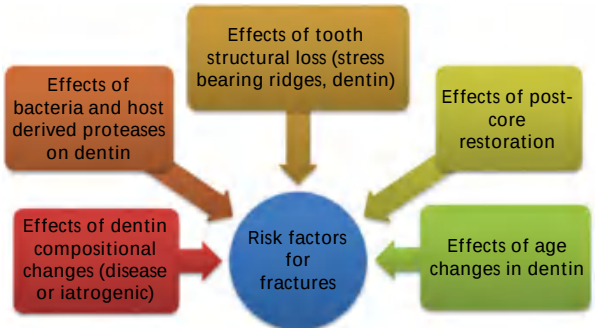


**Fig. 27.3** Risk factors that predispose root-filled teeth to fracture.

**Fig. 27.4** Shows a mesio-distal crack in a maxillary first molar tooth, progression towards the root (cracked tooth).

**Fig. 27.5** Shows a vertical root fracture in a maxillary premolar tooth. The fracture occurred in the bucco-lingual plane of the root, progression towards the crown.

Generally, it is believed that the causes of crown fracture are located in the coronal structure of the tooth. For instance, endodontic access cavity preparation and/or increased removal of coronal dentin result in greater degree of coronal flexure in the bucco-lingual direction, and mesio-distal crack progression from the crown to the root (*cracked tooth*) (Fig. 27.4). On the other hand, the causes of root fracture would generally be located in the root structure. For instance, conspicuous loss of root canal dentin and iatrogenic micro-defects in root dentin results in crack extension through the greatest bulk of the root in the bucco-lingual direction and progression from the root towards the crown (*vertical root fracture*) (Fig. 27.5) (14).

## Strengthening of coronal structure

Preservation of dentin is considered to be a crucial step in maintaining the mechanical integrity of restored endodontically treated teeth. Initially, an access cavity, by itself compromises the mechanical integrity offered by the roof of the pulp chamber, which results in greater cuspal flexure in the bucco-lingual direction during chewing (14). The impact of an access cavity on cuspal flexure is appreciably more than its impact on the stiffness of posterior teeth (15, 16). The bulk of dentin supporting the cusps and the integrity of the marginal ridges influences the degree of cuspal flexure. Hence loss of tooth structure ensuing from extensive caries and previous restorative procedures, particularly those involving the marginal ridges compromises the mechanical integrity of the crown. The presence of a large access cavity in a tooth with unsupported cusps or without marginal ridge support predisposes the tooth to structural failures (14, 16–18). Unfortunately, fracture resistance is only partially recovered when Mesio-Occlusal-Distal preparations associated with an access cavity are restored with composite resin (17, 18). The repeated flexure of cusps associated with chewing cycles (fatigue) results in mesio-distal crack propagation from the crown towards the root of the tooth (cracked tooth) (19). Accordingly, clinical steps aimed to strengthen coronal structure should ideally focus on minimizing cuspal flexure (Figs 27.6 and 27.7).

## Steps to strengthen coronal tooth structure

- Preoperative assessments
- Interim and temporary restorations
- Endodontic access cavity design
- Cuspal coverage with crowns

### *Preoperative assessments*

Endodontic treatment fundamentally aims to eliminate all the portals of entry of bacteria that lead to apical periodontitis. This necessitates the inspection of restorations or tooth structure for defects (Fig. 27.8). These defects have to be eliminated prior to the commencement of root canal therapy since these are not only potential pathways of reinfection, but also focal points for crack propagation during or after endodontic treatment. Conventional clinical and radiographic examinations of a tooth prior to endodontic treatment may not reveal with certainty the presence of caries, crack or breakdown of restoration (20). It is also difficult to assess if there is sufficient remaining tooth structure for adequate restoration of the tooth following endodontic treatment (20). Removal of restorations and caries during preoperative assessment will aid in assessing the restorative requirements for the tooth prior to the root canal therapy (21). This step is also crucial to consider the need of an interim restoration while the endodontic treatment is carried out; since root

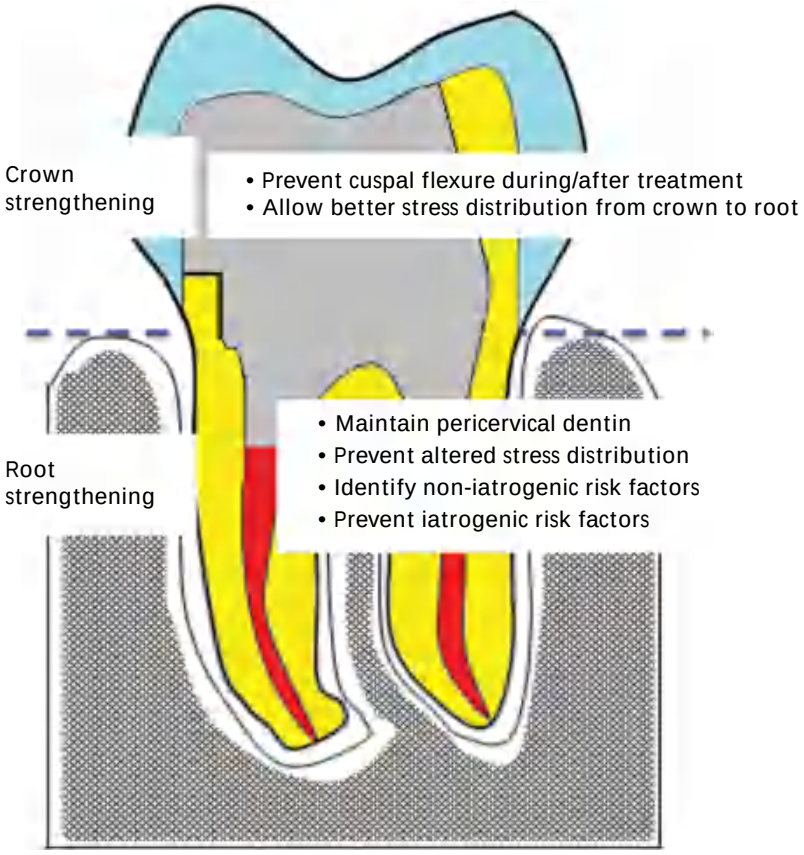


**Fig. 27.6** Shows the main factors associated with crown and root strengthening in endodontically treated posterior teeth.

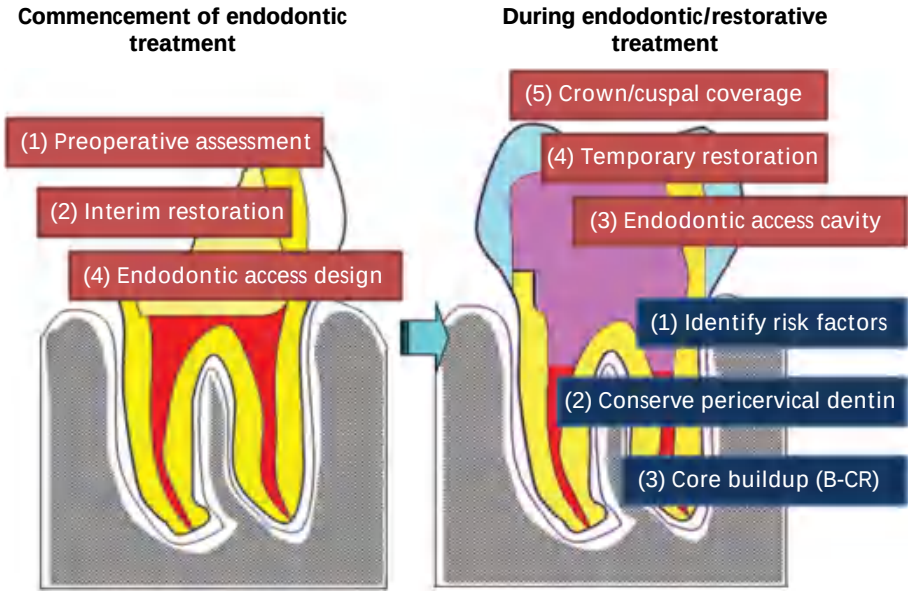


**Fig. 27.7** Schematic diagram showing steps taken prior, during and post endodontic treatment to reinforce structurally compromised endodontically treated molar teeth.

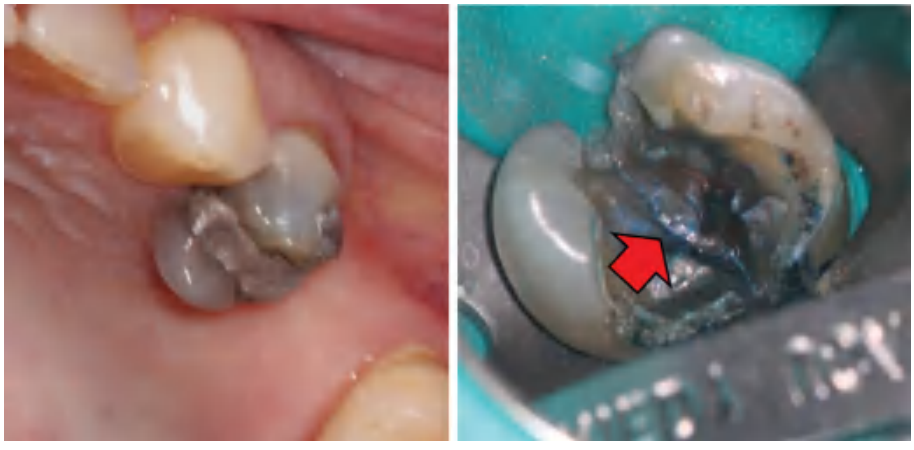

**Fig. 27.8** Shows a mesio-distal crack under a large amalgam restoration in a maxillary premolar planned for endodontic treatment. Source: Reproduced with permission of Dr. A. Shrestha.

canal treatment is usually completed over multiple appointments (20). Even if the endodontic treatment is carried out in a single appointment, an interim restoration of the tooth is still required unless a definitive coronal restoration is completed at the same appointment. A study that evaluated the survival rate of root-filled cracked teeth over a 2-year period concluded that preoperative features such as multiple cracks, terminal teeth and pocketing were significant prognostic factors for the survival of root-filled cracked teeth (22).

### *Interim and temporary restorations*

The risk of tooth fracture increases from the commencement of endodontic therapy, so effective interim and temporary restorations are imperative to buttress the remaining tooth structure during root canal therapy. Interim restoration is a restoration that is placed in a tooth after the previous restoration and/or caries have been removed at the commencement of root canal therapy. The interim restoration is expected to remain in the tooth while the root canal therapy is being performed and until the definitive coronal restoration is placed. It is different to a temporary restoration, which is a restoration placed within an endodontic access cavity in between appointments. The requirements of each of these restorations are different, and different materials are used. The requirements of interim restorations are to prevent bacterial ingress, identifiable from adjacent tooth structure, easy manipulation/handling characteristics, adhesion with tooth structure, reinforce remaining tooth structure and ultimately allow dentin conservation. The temporary restorations allow the closure of the access cavity immediately after root canal treatment, circumventing issues associated with coronal bacterial leakage. The selection of a temporary restoration depends on the extent of the cavity, anticipated period of function and requirements (21, 23).

These endodontic restorations should have good dimensional stability and adequate tensile strength in minimal bulk allowing tooth to continue functioning. The material should have satisfactory appearance in areas of esthetics and reproduce tooth contours to maintain oral hygiene. They should present only a moderate degree of moisture sensitivity when placed subgingivally. Currently there is no ideal material available for interim restoration that meets all the above criteria, though clinical results are still positive (21). Some of the commonly used materials for interim restoration are: glass ionomer cement, resin modified glass ionomer cement, reinforced ionomer cement, composite resins, amalgam, zinc oxide based reinforced materials such as IRM (Dentsply, Caulk, Milford, USA), TERM® (Temporary Endo Restorative Material, Dentsply Maillefer, Switzerland), and calcium sulphate based filling material (Cavit-3 M ESPE, See Feld, Germany) (Fig. 27.9). The latter two materials are frequently used as temporary restorations either alone or in combination such as double seal (Cavit (inner seal) + IRM or GIC (outer seal)) (21, 24).

Stainless steel and orthodontic bands have been suggested during endodontic treatment of posterior teeth (25). Cementation of stainless steel bands in combination with interim restorations is recommended in teeth with large cavities when there are missing cusps and when cusps are susceptible to fracture during the course of endodontic treatment (Fig. 27.10). The main purpose of the band is to help retain and strengthen the interim restoration, particularly when an access cavity is later prepared through it (21). Pane et al. (26) examined the effect of stainless steel bands on cusp flexure of maxillary premolar teeth. They concluded that stainless steel bands used during endodontic treatment decreased cuspal flexure by half, and increased fracture resistance of tooth placed under load. Although marginal adaptation of stainless bands and gingival irritation was suggested to be a concern, this issue may not be very significant due to the following reasons: (a) the short-term use of these bands, (b) if the patient is undertaking adequate oral hygiene measures, and (c) if additional crown-lengthening procedure is required. However, there is no long-term study

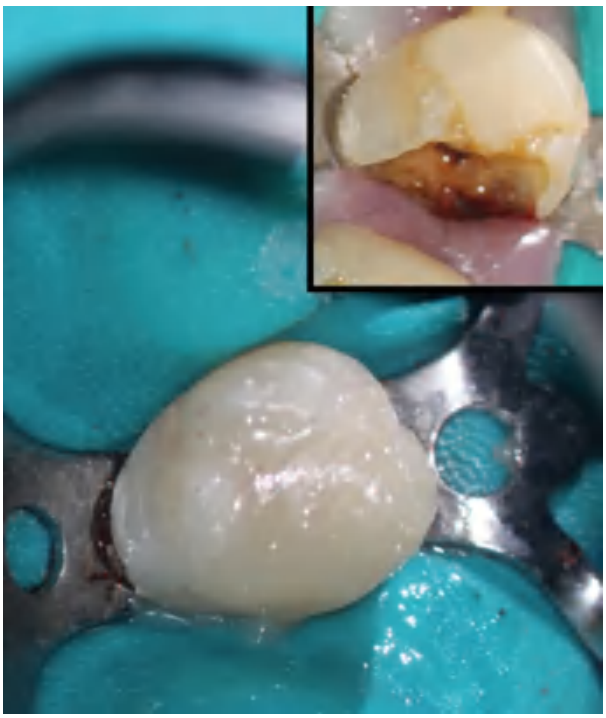

**Fig. 27.9** Shows an interim restoration in place in a maxillary premolar using bonded composite resin. The inset shows the preoperative condition of the tooth.

**Fig. 27.10** Shows an interim restoration in place in a mandibular molar using stainless steel band.

linking the use of stainless steel bands during endodontic treatment in adult patients and their impact on periodontal tissues (21).

### *Endodontic access cavity designs*

An adequately prepared endodontic access cavity is crucial for effective instrumentation and optimum delivery of irrigants into the root canal systems during endodontic therapy. The primary purpose of an endodontic access cavity is to render the succeeding steps in root canal therapy easier, effective and safer (28). An endodontic access cavity that has been inadequately prepared will make locating, negotiating, debriding, disinfecting and filling of the root canal system a challenging task (28, 29). Additionally, an inadequate endodontic access cavity may cause iatrogenic complications during endodontic treatment procedures. All the above mishaps, would hamper the prognosis of endodontic therapy (28, 29).

*Traditional endodontic access cavities* are geometric predesigned shapes. The outline of an endodontic cavity is determined by the degree of dentin removed at specific locations so as to achieve a straight-line access to the root canal orifices (27). In addition, endodontic cavity preparation involves the removal of dentin to explore for expected features in the root canal anatomy. The summation of all the steps in endodontic cavity preparation involves the removal of crucial dentin that distinctly alters the biomechanical response of a tooth to chewing forces. Thus, it is desirable to preserve the essential coronal dentin structure so as to (a) retain the mechanical integrity of the coronal structure and (b) accomplish ample foundation for the restorative procedures that follow endodontic therapy (30–33).

The recent developments in dental imaging, magnification, ultra-flexible instruments and irrigation devices have considerably advanced clinical practice in dentistry. Leveraging on these developments, new designs of endodontic access cavities called *contracted endodontic cavities* have been promoted by certain clinicians. The contracted endodontic cavity aims to transform the outline of endodontic cavity from a traditional *operator centric design* to a scheme that focuses more on dentin preservation and restorative considerations (30, 34, 35) (Fig. 27.11). This approach discounts the traditional requirement of a straight-line access and complete unroofing of the pulp chamber, while emphasizing the importance of preserving the dentin, especially at the cervical region of the tooth (36), and some portion of the roof around the entire coronal aspect of the pulp chamber (*dentin roof strut or soffit)* (34) (Fig. 27.12). The cingulum dentin (*pericingulum dentin*) in incisors may also be conserved to improve the mechanical integrity (37). It is presumed that contracted cavity would provide some effect of structural bracing, which in turn would minimize cuspal flexure of posterior teeth during chewing. Yet the long-term reinforcing attributes of dentin preservation in contracted endodontic cavity design is not clearly established at this time, Furthermore, it is key to judiciously assess the impact of contracted endodontic cavities on the effectiveness of subsequent steps such as cleaning and shaping before accepting them more routinely in clinical practice (38).

### *Cuspal coverage with crowns*

Clinical studies have demonstrated improved long-term prognosis for root-filled posterior teeth that received full cuspal coverage restoration (39–42). Since the axial margin of the crown is an important location for high stresses, the margin of the final preparation should extend well onto the sound tooth structure to allow better distribution of functional stresses and minimize stress concentrations (43). To achieve this end, occasionally a surgical crown-lengthening procedure may be necessary prior to crown preparation. When different core materials were tested on extracted teeth without final crowns, there was a considerable difference in their resistance to fracture. However, when the test was repeated with a final crown in place and their margins extended 2 mm onto the sound tooth structure (ferrule), there was little difference in the resistance to fracture between the groups (44).

A retrospective clinical study covering a 20-year period of root-filled posterior teeth restored with silver amalgam or composite resin showed that at the 5 and 10-year points, there were fewer tooth fractures in the composite resin group than in the amalgam group. At the 10-year period, fracture occurred in teeth without cuspal coverage in 16% of teeth restored with silver amalgam and 13% of teeth restored with composite resin. At the 20-year period, the incidence of fracture was over 25% for both groups, and there was no difference between the two materials. It was concluded that the incidence of tooth fracture was so high that all posterior root-filled teeth should be restored with cuspal coverage to minimize the possibility of fracture (45). Another clinical study suggested that four conditions were significant to improve the survival of root-filled teeth. In descending order of influence, they were: (1) tooth with crown restoration after root canal treatment; (2) tooth with both mesial and distal proximal contacts with adjacent teeth; (3) tooth not functioning as an abutment for removable or fixed prosthesis; and (4) tooth type or specifically non-molar teeth (46). These studies emphasized that timely placement of crown/ cuspal coverage, and uniform circumferential stress distribution from the crown to the bulk dentin was critical for the long-term survival of root-filled teeth (46).

## Strengthening of root structure

Steps to strengthen root structure should ideally aim to enhance the resistance of the root to vertical root fracture. Vertical root fracture is described as the longitudinally oriented fracture of a tooth that originates from the apical region of the root and propagates towards the crown. It is recognized that factors that alter the stress distribution pattern in the root and presence of microdefects (iatrogenic or non-iatrogenic) in root dentin can increase the susceptibility of endodontically treated teeth to vertical root fracture. Biomechanical studies demonstrated that chewing forces resulted in stresses, which is chiefly distributed in the pericervical dentin, which is described as the dentin located 4 mm above and 4 mm below the crestal bone (35). Since the pericervical region experiences

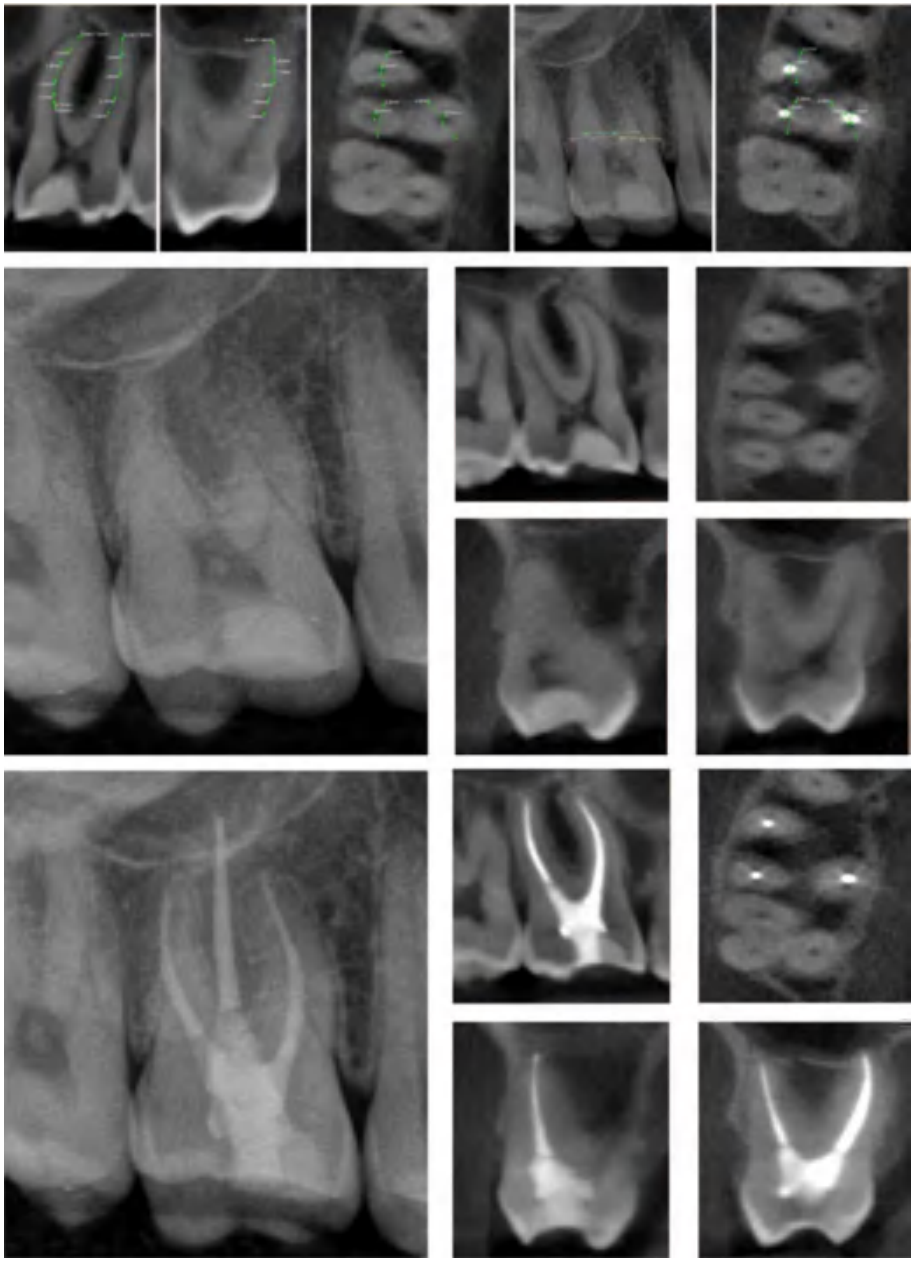

**Fig. 27.11** Individualized determination of shaping taper based on the pericervical horizontal measurements of each root and canal on the axial CBCT slices. Preoperative CBCT slices can be used to determine preoperative horizontal dimensions of each root and canal at the pericervical area. Determination of the initial dimensions allows predetermination of the maximum structural removal and the taper of the instruments needed to reach this size. Source: with permission of Dr Boveda.

bending during chewing, the pericervical root cross-sectional area markedly influences the flexural resistance of the root. Thus, a large, thick-walled dentin at the pericervical region is more resistant to fracture merely because it distributes stresses over a larger surface area. Also, any non-uniform removal of dentin around the centroid of the root canal would reduce the resistance of the root to flexure. With increasing loss of root canal dentin, the root experiences greater flexure, which in the presence of microdefects, would favor fatigue crack propagation and vertical root fractures (47). The cracks that lead to vertical root fracture, mostly originates in proximity to the root canal wall (48). Hence in order to maintain the mechanical integrity of the root, it is crucial not only to preserve the bulk of pericervical dentin, but also preserve the original root canal geometry (49, 50) (Figs 27.6 and 27.7).

## Steps to strengthening the root structure

- Identifying risk factors
- Conserving pericervical dentin
- Root canal fillings and core build-ups

### *Identifying risk factors*

There are both iatrogenic and non-iatrogenic risk factors that increase the susceptibility for fractures in endodontically treated teeth (Fig. 27.3). Occlusal forces from functional and parafunctional habits are important factors to consider in the restorative planning of endodontically involved teeth. Endodontically treated teeth when used as abutments for precision attachment and distal-extension

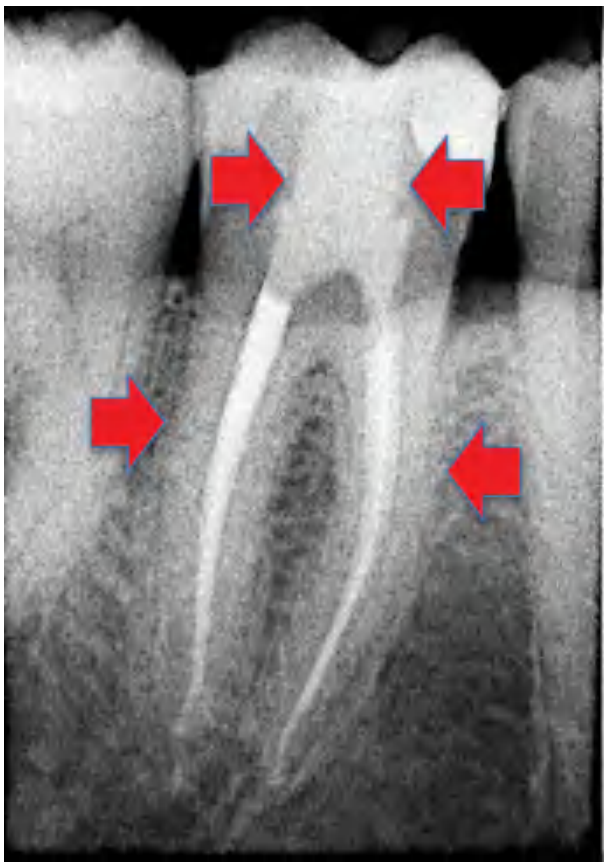

**Fig. 27.12** Shows an endodontically treated mandibulary molar treated through a contracted access cavity and limited shaping. Source: Reproduced from (38) with permission.

base Removable Partial Dentures, and posterior cantilevered Fixed Partial Dentures are particularly at higher risk for fracture than vital teeth (46). In mature teeth with complete root formation, decreased amounts of remaining sound tooth structure before or after root canal treatment or coronal restoration is indicative of compromised mechanical integrity. Further, prolonged application of high concentrations of commonly used endodontic irrigants significantly compromises the mechanical properties of dentin, while the application of high compaction forces during obturation might initiate micro-cracks leading to vertical root fracture with time (50, 51). Therefore, application of lower concentration of sodium hypochlorite (1.5–2.5%), especially as a final irrigant would aid in curtailing significant changes to dentin ultrastructure. Along similar lines, it is recommended to restrain from applying large amount of wedging forces during root canal filling.

Anatomical variations in the root canal geometry influenced changes during root canal preparation (example: number of untouched walls) more than the preparation technique itself (52, 53). Consequently oval-shaped root canals in mandibular molars left a variable portion of root canal surface unprepared regardless of the instrumentation technique (54). So, the influence of root canal anatomy on the degree of root dentin removed should be anticipated. Yet preflaring of the root canal at the cervical region is usually recommended to facilitate cleaning, shaping and filling. The taper of endodontic instruments have changed from the traditional size of 0.02 to bigger and even variable designs in recent times (51). Cervical preflaring of the root canals and root canal instrumentation with instruments with large tapers would increase the amount of pericervical dentin removed.

The influences of root morphology and dentinal age changes on fracture susceptibility are other considerations that should be borne in mind. It has been suggested that maxillary second premolars, mesiobuccal roots of maxillary molars; mesial roots of mandibular molars and mandibular premolars are most prone to vertical root fracture (VRF) (55, 56). The reason for the propensity of VRF in posterior teeth may be attributed to the root anatomy (narrow mesio-distal width) and the higher masticatory loads on these teeth. Higher propensity to VRF in older patients has also been reported. One study reported higher fracture susceptibility in patients over 40 years (57), while another study reported higher fracture susceptibility in patients over 60 years (58). The reason for the increased predilection of fracture in older patients could be attributed to the age related physical effects in dentin such as reduced fracture toughness (59) and tensile strength (60).

### *Conservation of pericervical dentin*

The goals to reinforce endodontically treated root should value the pericervical dentin. Intact pericervical dentin allows better transfer of functional forces to the radicular portion of the tooth. In young patients, this goal can be achieved by maintaining the natural configuration of the root canals. This means that the horizontal dimensions of the root canal at the pericervical region is minimally altered. In calcified teeth, attempts to mechanically recreate the intended shape / taper in a meticulous manner by staying away from the furcal area would be of benefit. It is recommended to adjust the instruments and their taper based on the dimensions of the horizontal configuration of each root canal and root (38). Even the apical preparation should aim to remove minimum tooth structure, while the apical foramen is kept as small as possible (Fig. 27.12).

Dentin experiences an age-induced alteration from a normal dentin to a transparent or sclerotic dentin. The dentinal tubules in the transparent dentin are gradually filled up with minerals, beginning at the apical region of the root and progressing towards the cervical region. The fracture toughness of transparent dentin is 20% lower than the normal dentin (brittle behavior). Bang & Ramm 1970 (61) showed a linear increase in the width of the transparent root tip dentin from the age of 20 (approximately 2 mm) to the age on 70 (approximately 10 mm). These observations necessitate caution while performing endodontic treatment in older patients, especially at the apical portion of the root.

### *Root fillings and core build-ups*

Stable adhesion between the obturating material and root canal dentin plus similarity in the elastic modulus of the core filling material and root dentin are key features for improving resistance to fracture in root-filled teeth (51) (Fig. 27.13). Gutta-percha has been used as a core filling material for root canal obturation since the 1800s. From the biomechanical

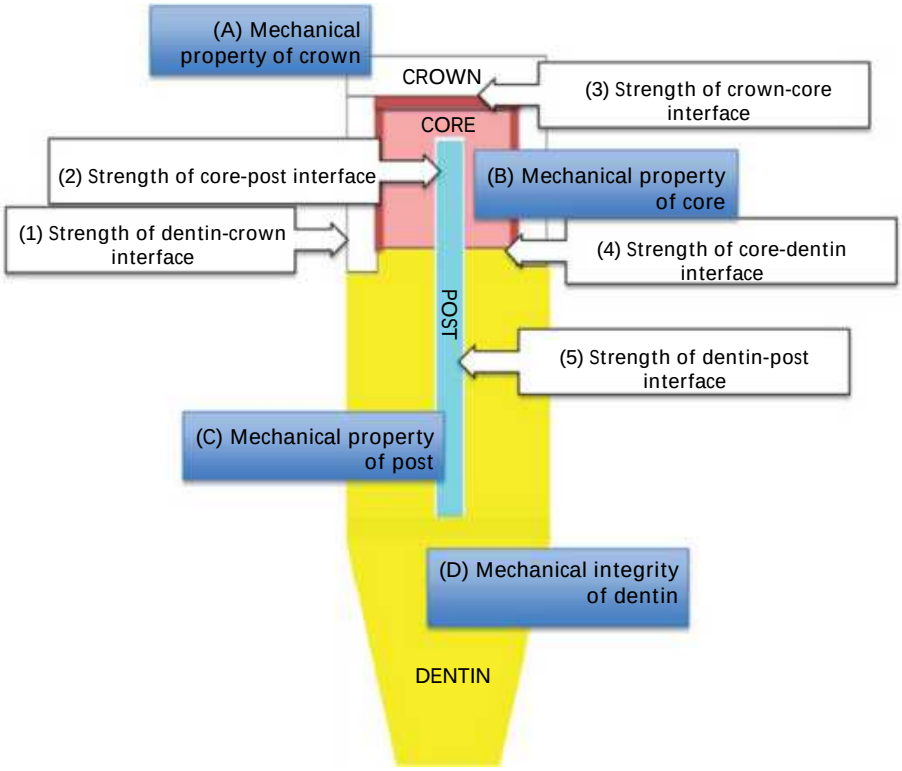


**Fig. 27.13** Schematic diagram showing interactions between different factors that influences mechanical stress distribution in restored endodontically treated teeth (1-5 are the strength of different interfaces and A-D are mechanical characteristics of different parts of restorations / remaining dentin).

perspectives, the specific limitations of gutta-percha are (a) it does not adhere to the root canal wall and (b) its elastic modulus is markedly less than that of dentin. Resilon™ is a synthetic resin-based polymer, used along with Epiphany, a resin sealer, which is expected to form an adhesive bond at the interface of the resin polymer core and the canal wall (62). This resin based obturation system was developed as a gutta-percha substitute. Though they formed a chemical bond with dentin, their bond strength and elastic modulus were very low. Highly filled resin composites have a similar elastic modulus to dentin and are bonded to the root dentin walls, though they are not ideal root canal obturation materials. Furthermore, *in vitro* studies have suggested inconsistent and insignificant advantage of any particular obturation material (bonded / non-bonded resin or gutta-percha) to improve fracture strength in root-filled teeth (63–65).

The primary purpose of a core buildup is to replace the lost coronal tooth substance and serve as a foundation that increases the height of coronal tooth structure to retain the coronal restoration. In most cases, during crown or abutment preparation, the outer walls of the remaining tooth structure will be reduced or removed completely, and the retention of the core buildup must be obtained from the tooth structure that remains after the final preparation. Modern dentin adhesives are able to retain composite resin in cavities without any additional retentive features. Nevertheless, adequate mechanical retention is important to supplement dentin bonding. The options to achieve mechanical retention of a core buildup are different in single-and multi-rooted teeth. The large size of the pulp chamber (width and depth) in multi-rooted teeth is of considerable advantage for achieving mechanical retention. Undercuts can provide excellent mechanical retention for the core buildup. They are a natural feature of multi-rooted teeth with divergent root canals. In anterior teeth, the functional forces act in a more horizontal direction and the tooth cross-sectional area is smaller than in posterior tooth. This will result in an unfavorable lever-arm relationship. Whenever the remaining tooth structure and pulpal space can support sufficient retention of a core buildup, a post should be avoided since the risks with the use of a post do not exceed the advantages in most cases (66).

Composite resin is the material of choice for core build-ups and to strengthen remains tooth structure (41). Its modulus of elasticity is equal to or higher than that of dentin. When used in combination with a dentin adhesive, it offers superior bond strength with the tooth structure, leading to improved reinforcement. The mechanical properties of a composite resin make it suitable for restoring even more than half of the coronal tooth structure. Since it is a tooth colored restorative material, it has the esthetic advantage when used in combination with ceramic crowns (41). Cements with high compressive strength such as metal reinforced glass ionomer cements are not suitable for core buildup. The fracture strength and shear strength of a glass ionomer cement core is significantly less than composite resins or silver amalgam (67). Resin modified glass ionomer cements and compomers achieve fracture strengths similar to that of composite resins, but they experienced a slow expansion due to water sorption. Slow expansion of core build-ups is likely to exert tensile stresses and subsequent cracking of other restorations and remaining tooth structure (68).

Immature permanent teeth with incomplete root formation requiring endodontic treatment are extremely weakened due to their wide, flared canal spaces and thin dentin walls. Root reinforcement following apexification procedures is essential to enhance the mechanical integrity of these teeth (51). An effective reinforcement is a major feature to be

considered for the long-term success of such teeth. In this case, an effective reinforcement can be achieved by building the post-bearing portion of the root with light-cure composite resin using a transparent light-conducting post. Subsequently, the transparent light-conducting post is removed, and a prefabricated metal post is cemented. This procedure will help in improving the overall fracture resistance of the treated tooth (69). By using a light-conducting post, a curing depth of up to 11 mm can be achieved depending on the diameter of the post (70). The strengthening effect of such internal composite resin build-up for up to 3 mm apical to the cemento-enamel junction has been confirmed (71). However, clinical evidence on the longevity of this "strengthening effect" is lacking.

### *Post systems*

A post is used to retain the coronal restoration when insufficient tooth structure remains. They maintain the marginal integrity of the final restoration by providing sufficient rigidity under functional loads (11). The selection of a post depends upon the amount of remaining tooth structure, anatomical position of the tooth and the functional and esthetic requirements of the root-filled tooth. Considerable controversy exists regarding the ideal choice of material and design of post and core to improve fracture resistance in post-core restored teeth. Different studies have highlighted the dangers of using tapered posts (72–77) Although it is believed that parallel-sided posts could distribute functional loads passively to the remaining tooth structure (78, 79), some studies have observed only minimal advantages with parallel-sided posts when compared to tapered posts (80, 81). The most important factor for preventing fracture however is not the post design but the final crown. This was confirmed by a retrospective clinical study that showed that the presence of a post had little effect on the fracture rate of a crowned tooth (39). It was also shown that bonded posts resulted in less stress within dentin than non-bonded posts. It is important to realize that a post and core restoration does not reinforce remaining radicular dentin or permit uniform transfer of stresses within the remaining dentin structure (82). Tooth deformation is said to increase progressively after root canal preparation and obturation and particularly after tapered and parallel post space preparation (49). In addition, post core restorations direct occlusal stress, apically and subsequently alter the nature of stress distribution in the remaining tooth structure. The factors responsible for this altered stress distribution pattern in a post core restored tooth are (a) the greater stiffness of the intracanal post, (b) the angulation of the post and (c) increased flexure of the remaining tooth structure. This increased tooth flexure and microdefects created in root dentin during post preparation would further predispose such a tooth to vertical root fracture (83).

It is generally believed that carbon and glass fiber posts have a transverse elastic modulus that is close to that of dentin and therefore are less damaging to the remaining dentin (84, 85). In the case of posts with mechanical properties similar to those of dentin, the remaining dentin, cement interface and post will all deform together in function. Thus, the failure mostly appears at the weakest point, which would be the adhesive joints at the core-dentin and post-cement-dentin interfaces. Hence the mode of failure will be loss of marginal seal, core fracture, post fracture or loss of retention (86). The greater the loss of coronal tooth structure, the greater will be the stress at the adhesive interface. Yet, the reinforcement effect after cementation of a complete crown with ferrule effect makes the difference between stiff and elastic posts less obvious (80). It is also suggested that a stiff post placed in a tooth with minimal coronal dentin would withstand greater biting stresses. Fatigue-induced failure in such post-core systems would occur at higher stress levels and after a considerably longer time compared to a low-modulus post, but the risk of an irreparable vertical root fracture would be high (87–89).

## Conclusions

There are different iatrogenic and non-iatrogenic risk factors that could initiate or propagate cracks leading to fractures in endodontically treated teeth. Increased cuspal flexure in endodontically treated posterior teeth has been the prime feature that results in mesio-distal fractures and cracked teeth. The loss of pericervical root dentin as well as other iatrogenic and non-iatrogenic alterations in root dentin contributes to vertical root fractures. The basis of reinforcing endodontically treated teeth revolves around conserving dentin to maintain the structural integrity so as to satisfy the functional requirements of teeth. Thus, clinical procedures should aim at conserving tooth structure and avoiding potential risk factors for fractures in root-filled teeth. These concepts critically hinge on advanced technologies, such as magnification and imaging, as well as skill and knowledge of the clinicians.

Recent years have witnessed great advances in the materials and methods available for restoring root-filled teeth. Many of these advances are related to adhesive core materials, luting cements and dentine bonding agents. Yet, current studies do not show a consistent and significant advantage of using any particular obturating material to improve fracture resistance in endodontically treated teeth. There is also an improved understanding of the behavior of root-filled teeth with and without post-core restorations. It should be understood that the risk of tooth fracture increases from the beginning of endodontic therapy, so effective interim, temporary and final restorations are vital to protect the tooth during function. Other important aspects to be considered while restoring root-filled teeth are to keep the flexural stresses within the remaining tooth structure to a minimum. From a restorative perspective, this includes providing cuspal coverage for posterior teeth, use of posts with adequate strength in thin diameter and maximizing resistance form by including an adequate ferrule. Intracanal posts should be placed only when they are essential for the retention of cores and permanent restorations. Thus, there is no single step to reinforce endodontically treated teeth but it may be achieved by being mindful of both non-iatrogenic and iatrogenic risk factors and by carefully executed steps during endodontic and restorative treatments.

## Essentials

- After endodontic treatment several changes occur in the material and bulk structure of a tooth.
- The maximum chewing stresses are distributed at the cervical region of the tooth.
- Fracture in an endodontically treated tooth is typically a fatigue process related to structural and material changes in dentin.
- Strengthening of the coronal structure should be aimed to strengthen the coronal structure.
- Timely placement of the cuspal coverage/crown is recommended.
- Conservation of pericervical dentin is important to reinforce endodontically treated teeth.
- Core buildup and stable adhesion between the obturating material and root canal dentin are key factors to resist fracture in endodontically treated teeth.
- Post systems: carbon and glass fiber posts are recommended, although the risk of fracture should be determined by the amount of remaining dentin.

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# 28

# Orthodontic Management of the Traumatized Dentition

O. Malmgren & B. Malmgren

## Introduction

Treatment planning for patients with traumatized teeth involves a detailed evaluation of both the prognosis for the injured teeth and treatment of an eventual malocclusion. A coordinated treatment plan, incorporating clinical and radiographic findings of healing and of complications must be established before orthodontic treatment is initiated. The incidence of trauma has its peak when the dentition is developing, and growth of the face is intense. Furthermore, many children have various types of malocclusions. Therefore, many factors shall be considered after a severe trauma. What will be the long-term outcome of the traumatized teeth, which are the possible treatment options for reconstruction of a malocclusion with traumatized teeth, what will be the outcome of an orthodontic treatment if a complication occurs due to the trauma and which are the treatment options if a tooth is lost? All these factors vary individually, and a careful planning of treatment is important. The plan shall involve various dental specialties (i.e. pedodontics, endodontics, oral surgery, orthodontics and general dentistry) and be based on an evaluation of orthodontic treatment possibilities and optimal treatment of the traumatized teeth.

### Preventive orthodontics

Dental injuries in mixed and permanent dentitions are most frequent in children from 8 to 9 years of age and most injuries involve the upper incisors. Boys are injured twice as often as girls. Patients with an increased overjet are at significantly greater risk of dental injury (1–7). An increased overjet from 0–3 mm to 3–6 mm doubles the extent of traumatic dental injuries (3), and the severity is tripled with an overjet exceeding 6 mm (3, 8). An additional trauma factor is insufficient lip closure, which often leaves the upper incisors unprotected (6, 8–11). Many authors claim that this is the most important factor (12). Patients treated for dental injuries often sustain repeated trauma to the teeth (7), particularly if the first trauma occurs at an early age (13, 14). Considering the frequency of traumatic dental injuries in school-age children, it is apparent that the majority of children with increased maxillary overjet will have sustained a traumatic dental injury prior to school-leaving age (3, 7, 15–17). The treatment of increased maxillary overjet should therefore begin early, as a possible precaution against traumatic dental injuries (18, 19). If this is not possible, the child should be provided with a mouth guard during contact sports. Such a mouth guard can even be combined with a fixed orthodontic appliance (see Chapter 37).

### Orthodontic treatment after trauma in the primary dentition

Injuries to the primary incisors are common. Its prevalence ranges from 11 to 47% in various studies. Most injuries occur at the age of two to three years of age when the child is learning to walk and exploring the surroundings but is liable to fall (14, 15, 20, 21) (see Chapter 11).

Early avulsion or extraction of primary incisors do not usually lead to loss of space and drifting of adjacent teeth and consequently do not require space maintenance but may lead to disturbances in eruption of the permanent successors (22–24). Early loss sometimes leads to delayed eruption of the permanent successors often in a more labial position. This is most likely due to additional resistance of more fibrotic tissue after the loss of the primary teeth (24). Surgical exposure, sometimes completed with orthodontic extrusion might be necessary (Fig. 28.1). Root malformation of a permanent successor is not frequent but occurs when the root is developing and the epithelial root sheath of Hertwig is disturbed. This may lead to a short root and delayed tooth eruption. Conversely, loss of primary teeth in a later stage of

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**Fig. 28.1 9.5-year-old boy with delayed eruption of a central incisor** A. (Left) The left primary central incisor was lost when the boy was 2.5 years old. (Right) The left lateral primary incisor near exfoliation. B. 6 months later a surgical exposure was performed, and enamel hypoplasia was found labially. C. Status 8 months after exposure. The tooth had erupted spontaneously, and a restoration was performed.

**Fig. 28.2 6-year-old boy with a premature eruption of a first primary incisor**
The first left primary incisor was lost when the boy was 5 years old, and the permanent successor erupted 4 months later.

development can lead to premature eruption of permanent teeth (25) (Fig. 28.2) (see Chapters 22 and 23).

Crown dilacerations of permanent successors can occur after a more severe trauma in the age interval 1 to 3 years when the newly formed hard tissue of the crown deviates in relation to the long axis of the tooth. In severe cases a complete palatal deviation of the crown with additional enamel hypoplasia is seen (26). These lesions have no negative effect on the occlusion. Damaged permanent incisors erupt spontaneously as a rule, but surgical exposure and/or orthodontic extrusion might be necessary (Fig. 28.3).

Root malformations can in rare cases be lateral or vestibular root angulation. These disturbances occur mostly when the child is 4 to 5 years of age (27) (Fig. 28.4). Odontoma-like teeth can be the result after a severe trauma to primary teeth at a very early age 0 to 1 year. The permanent tooth germ is severely damaged and on radiographs the malformed tooth resembles a complex odontoma (27–31). Careful treatment planning is important after such serious injuries (32). In general, orthodontic treatment should be initiated in the mixed dentition.

## Orthodontic treatment planning

Before orthodontic treatment is initiated after a traumatic injury, a number of factors should be evaluated. Orthodontic treatment might be necessary already after injuries in the primary dentition (Fig. 28.5). In some cases, orthodontic movement of teeth with less favorable prognosis can be justified to preserve the alveolar crest during growth of the jaws. Even an uncomplicated extraction of a tooth leads to loss of alveolar bone especially in the labio-lingual direction which makes insertion of future implants difficult (Fig. 28.6) (see Chapter 35).

An early start of orthodontic treatment in patients with dental injuries and proclined upper incisors can reduce a large overjet (33). Headgear treatment, bite jumping devices or appliances with mini plate anchorage may be used. Follow-up treatment with a second phase later is however often necessary. The risk of root resorption of the traumatized teeth during the second phase might thereby be reduced (34). This is important both to avoid heavy forces to the teeth during the second phase and for the risk of further injuries. The expected cost of the total treatment also seems to be smaller even with the additional cost of a two-phase orthodontic intervention (33) (Fig. 28.7).

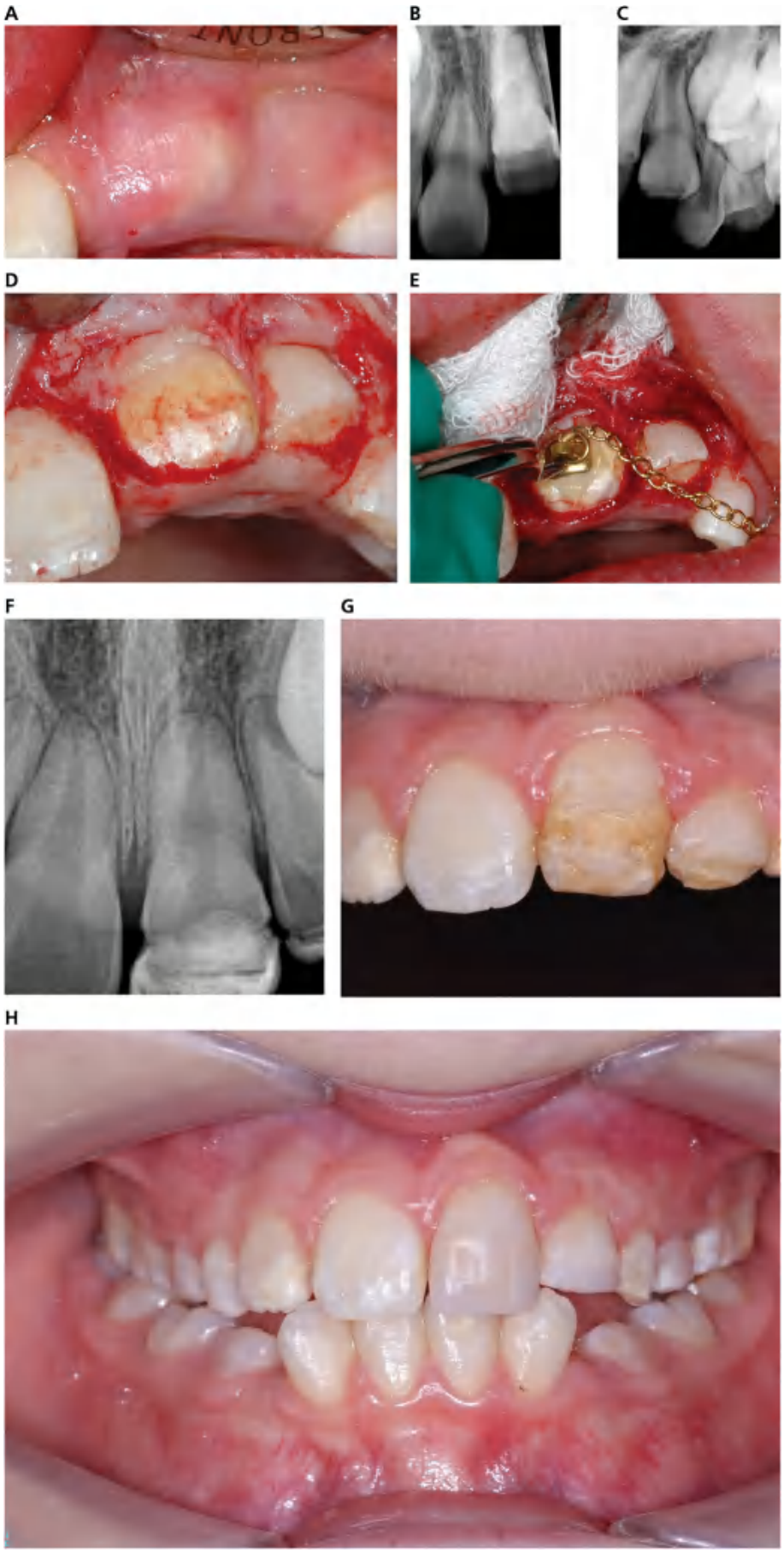



**Fig. 28.3 Crown dilacerations after an injury to the primary dentition**
A. An 8-year-old girl with an unerupted central and a lateral incisor. Both teeth had crown dilacerations due to an early trauma to the primary dentition when she was 4 years old. B. X-ray of the left maxillary central incisor. C. X- ray of the left lateral incisor. D. Treatment started with a raising of a muco-periostal flap for exposure of the two teeth and bonding of eyelets to the crowns. E. A fixed appliance bonded to the temporary teeth with a spring to the eyelets was used for the extrusion. The flap was sutured over the crowns. F. The dilacerated crowns were carefully checked when the teeth started to erupt through the gingiva. No pulp communication was observed but the palatal part was covered with composite. G. The crowns were restored with composite when the teeth were fully erupted. H. The developing occlusion was followed-up and future aligning of the dentition was necessary.

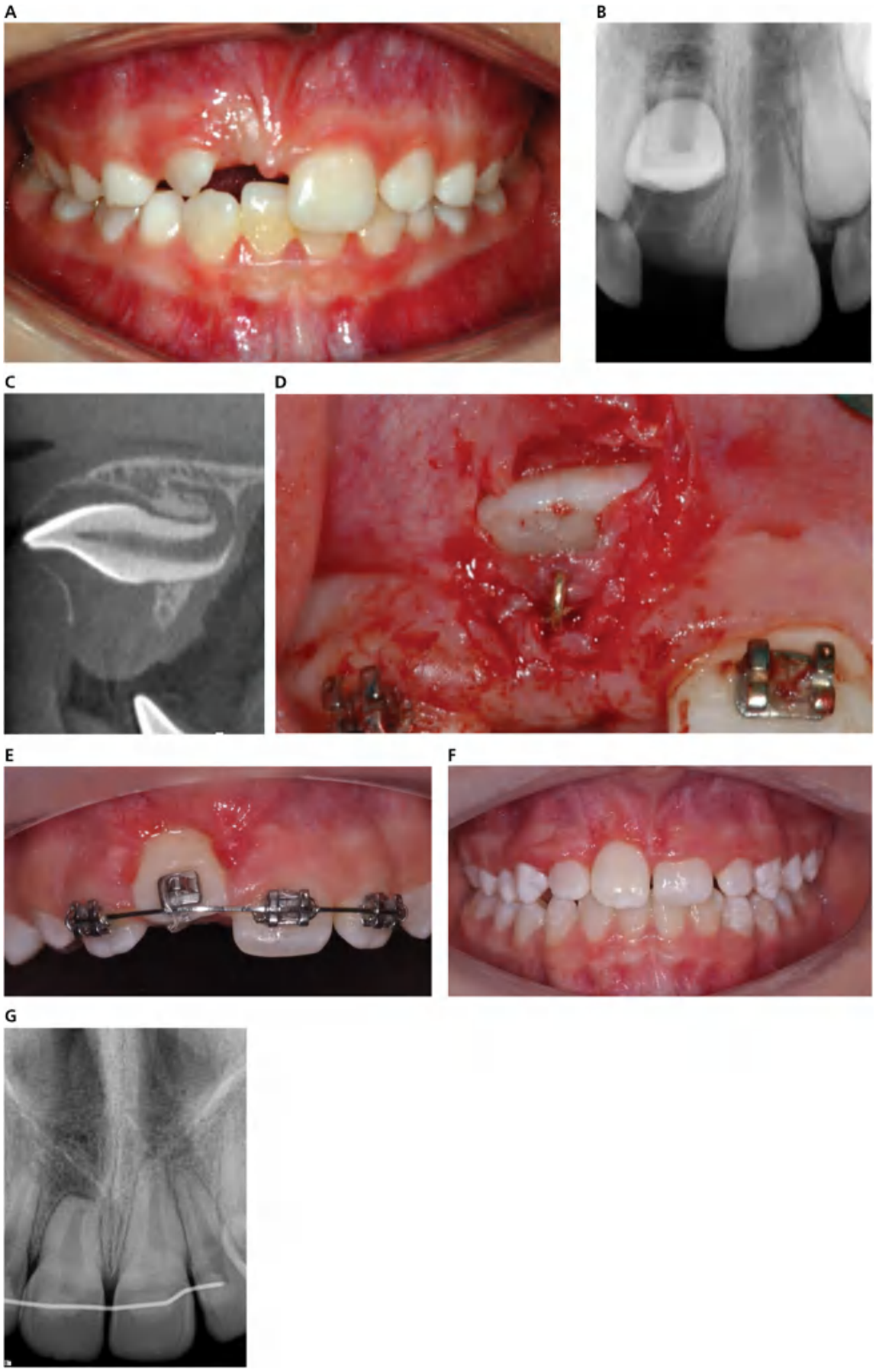



**Fig. 28.4 Vestibular root angulation after an injury to the primary dentition**
A. A 9-year old girl with a serious trauma to the primary teeth and no right upper central incisors visible. B. The right central incisor was impacted and had a horizontal position. C. A root angulation with the root bent in the apical part was seen on a CBCT scan. The malformed tooth was surgically exposed and extruded below the gingiva. D. A second exposure was performed when the tooth had almost erupted through the gingiva. E. The extrusion was completed after one year. F. Retention was performed with a palatal bonded twist-flex arch. G. X-ray after the extrusion. The patient was followed until complete development of the occlusion.

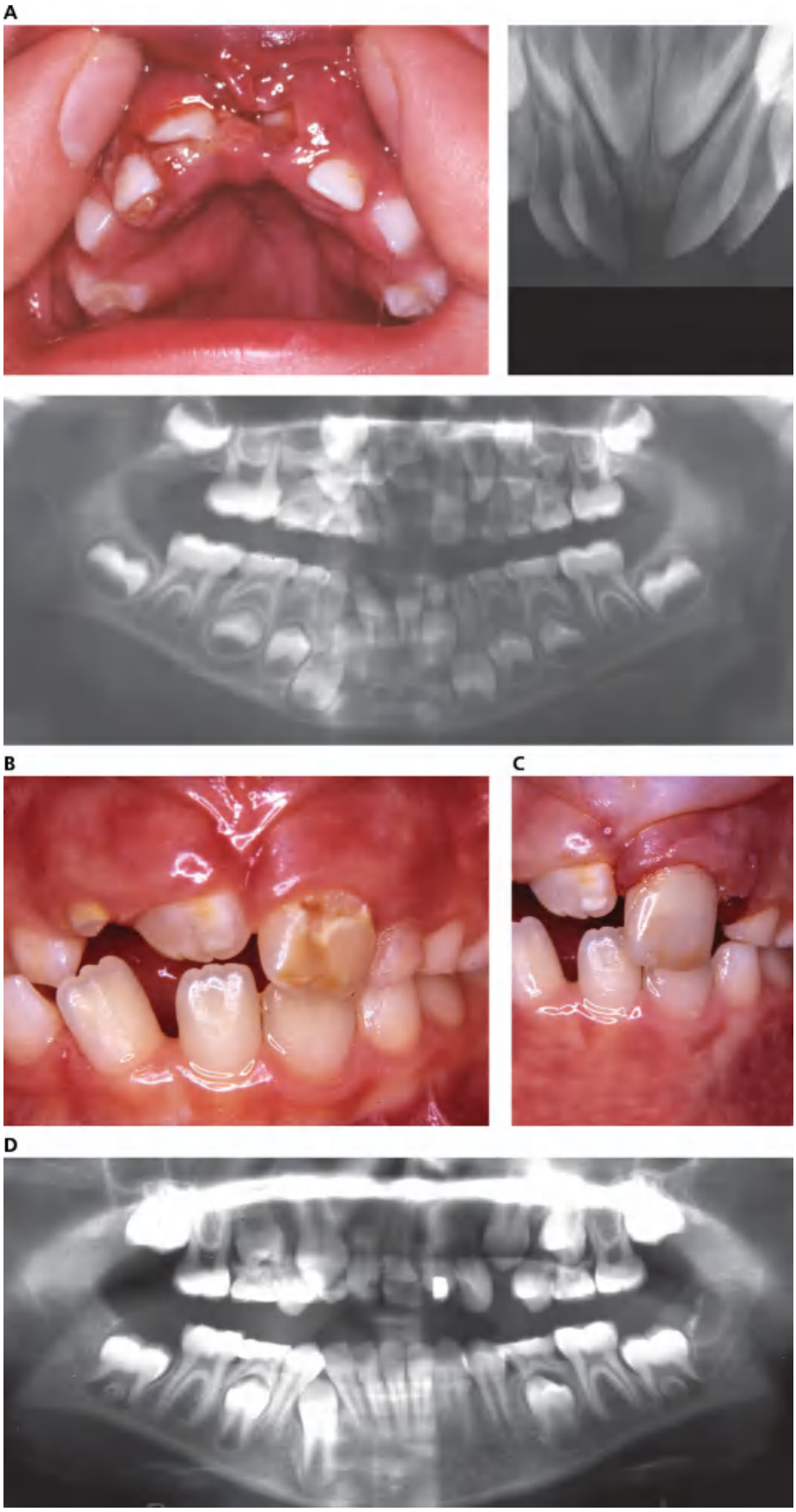



**Fig. 28.5 Complications after intrusion of central incisors**
A. A 2-year old boy with both central incisors intruded. B. Follow-up at 6 years of age. C. Clinical appearance at 8 years of age. Both upper incisors and the right lateral are dilacerated and the right lower incisor is tipped distally. D. Panoramic radiograph at 11 years of age. Due to a second traumatic injury, the left upper incisor has been treated endontically. Crowding in the right sides in both jaws are seen. Autotransplantation of the second right lower premolar is decided. Note difference in root development of the premolars.

**Extraction of the right upper incisor**
Note crown dilaceration.

E

**Extraction of the right lower first premolar**

F

**Right lower first premolar transplanted into right central incisor position**
Radiograph two months after autotransplantation. Note damage to the lateral.

G

**Right upper second premolar transplanted into left central incisor position**
The first transplant restored with composite as well as the hypomineralized lateral. Radiograph 1 year after the first autotransplantation.

H

**Condition 3 years after first autotransplantation**
No orthodontic treatment performed.

I

**Fig. 28.6 Reduced volume of the alveolar bone in buccal/palatal direction after loss of permanent teeth**
Reduced volume of the alveolar bone in buccal/palatal direction after loss of permanent teeth. A. An 18-year-old boy, whose left upper incisor was extracted conventionally at the age of 12 years. B. A 10-year-old girl, whose left upper incisor was avulsed two years earlier.

**Fig. 28.7 10-year-old girl with a severe postnormal occlusion and a large overjet and severely damaged upper incisors**
A. Condition before and after injury. At the time of injury. The right upper central incisor intruded and the left avulsed and immediately replanted. B. Two months after trauma a headgear treatment was started. Both incisors endodontically treated. C. Fixed appliance therapy with Class II treatment was initiated 1 year after trauma. D. Final occlusion 3 years after the trauma.

## Treatment principles for various trauma types

It is essential that tests for sensibility and a radiographic examination be made before the start of orthodontic treatment even in teeth with slight injuries such as uncomplicated crown fractures. When in doubt as to the clinical condition of the pulp, a 3-month observation period with repeated sensibility tests is recommended before initiating orthodontic treatment.

### Crown and crown-root fractures

Crown fractures and crown-root fractures without pulpal involvement have a good prognosis if properly treated. An observation period of about 3 months prior to initiation of treatment is sufficient. A crown and crown-root fracture with pulpal involvement can be treated orthodontically when a hard tissue barrier has been established after partial pulpectomy. As a rule, such a barrier can be diagnosed radiographically 3 months after treatment (35) (see Chapters 13 and 14).

A complicated crown fracture implies involvement of enamel, dentin and the pulp. A complicated crown-root fracture also involves the root cement. Capping of the pulp or partial pulpotomy on proper indications followed by a calcium hydroxide cover and a tight dressing of mineral trioxide aggregate (MTA) will in most cases preserve the vitality of the pulp (see Chapter 25). It is recommended that orthodontic movement of the injured immature teeth be postponed until root development is complete. Clinical and radiographic controls should be carried out after 6 months, 1 year and 2 years.

### Cervical root fractures

To complete a crown restoration of a tooth with a crown-root or a cervical root fracture, it is often necessary to extrude the fractured root. Two types of therapy are available, orthodontic or surgical extrusion (36–43 44, 45). Different types of fractures amenable to orthodontic extrusion are illustrated in Fig. 28.8. Orthodontic extrusion can be performed with a variety of orthodontic appliances. A rapid extrusion technique to save such teeth was introduced by Heithersay in 1973 (37) and further developed by Ingber in 1976 (38). Endodontic treatment of the root portion is usually performed before the orthodontic phase of treatment. Orthodontic extrusion normally leads to a coronal shift of the marginal gingiva caused by growth of the attached gingiva and not by coronal displacement of the muco-gingival junction (Fig. 28.9). The increase of gingival tissue may partially mask the extent of root extrusion (47, 48). It can thus be achieved without a coronal shift of marginal bone, thereby facilitating the coronal restoration, as there is no need to reshape bone.

Relapse may follow orthodontic extrusion, the prime reason being the stretched state of marginal periodontal fibers (39). To avoid this, fibrotomy should be performed before the retention period, which should last at least 3–4 weeks (42, 43) (Fig. 28.10). A non-vital tooth can be extruded, 3–5 mm during 3–4 weeks. Rapid movement is possible because stretching and readjusting the fibers and not by bone remodelling. Rapid extrusion of teeth, in comparison to conventional orthodontic extrusion, may only in rare cases elicit root resorption (49). Both histological and clinical studies of extruded teeth indicate that root resorption after extrusion is very rare (36–41, 49). For a tooth with a complicated crown-root or cervical root fracture below the marginal bone palatally, surgical extrusion combined with rotation of the root 180° might be a favorable treatment as less extrusion is needed which gives more bone support to the root (Fig. 28.11) (see Chapter 14). In teeth with thin root canal walls, i.e. teeth treated endodontically at an early stage of root formation, orthodontic extrusion seems to be less traumatic.

### Intra-alveolar root fractures

An intra-alveolar root fracture is a relatively rare injury. It has a good prognosis and the pulp will remain vital in most cases (see Chapter 15). While an observation period of 2 years has been recommended before orthodontic movement of root-fractured teeth (50), clinical experience indicates that most complications (e.g. pulp necrosis) occur during the 1st year after the trauma (35, 51, 52). Thus the observation period may be shortened if no complications occur (see Chapter 12).

Orthodontic management of root-fractured teeth depends on the type of healing and location of the fracture (50, 51, 53, 54). Radiographic and histological observations have shown different types of healing, with calcified tissue and with interposition of connective tissue, sometimes combined with in-growth of bone. Healing with *calcified tissue* means that the fracture is healed with dentin and cementum. The bridging of the fracture may not be complete, but the fracture is consolidated. Tooth mobility is normal, and the pulp is sensible to testing. Orthodontic movement of a root-fractured tooth, which has healed with calcified tissue, can be performed without breaking up the fracture (Figs 28.12 and 28.13). Healing with *interposition of connective tissue* means that the fracture edges are covered with cementum and periodontal ligament (PDL). Orthodontic movement of such a tooth where the fragments are separated by connective tissue leads to further separation of the fragments (Fig. 28.14). A common finding after treatment is then rounding of the fracture edges. In planning orthodontic therapy, it must therefore be realized that a fractured root with interposition of connective tissue should be looked upon as a tooth with a short root and the tooth must be evaluated with respect to the length of the coronal fragment. This means that teeth with fractures in the apical third of the root generally have enough periodontal support to allow orthodontic movement. Teeth with fractures located in the middle third of the root represent a hazard to tooth integrity because of the

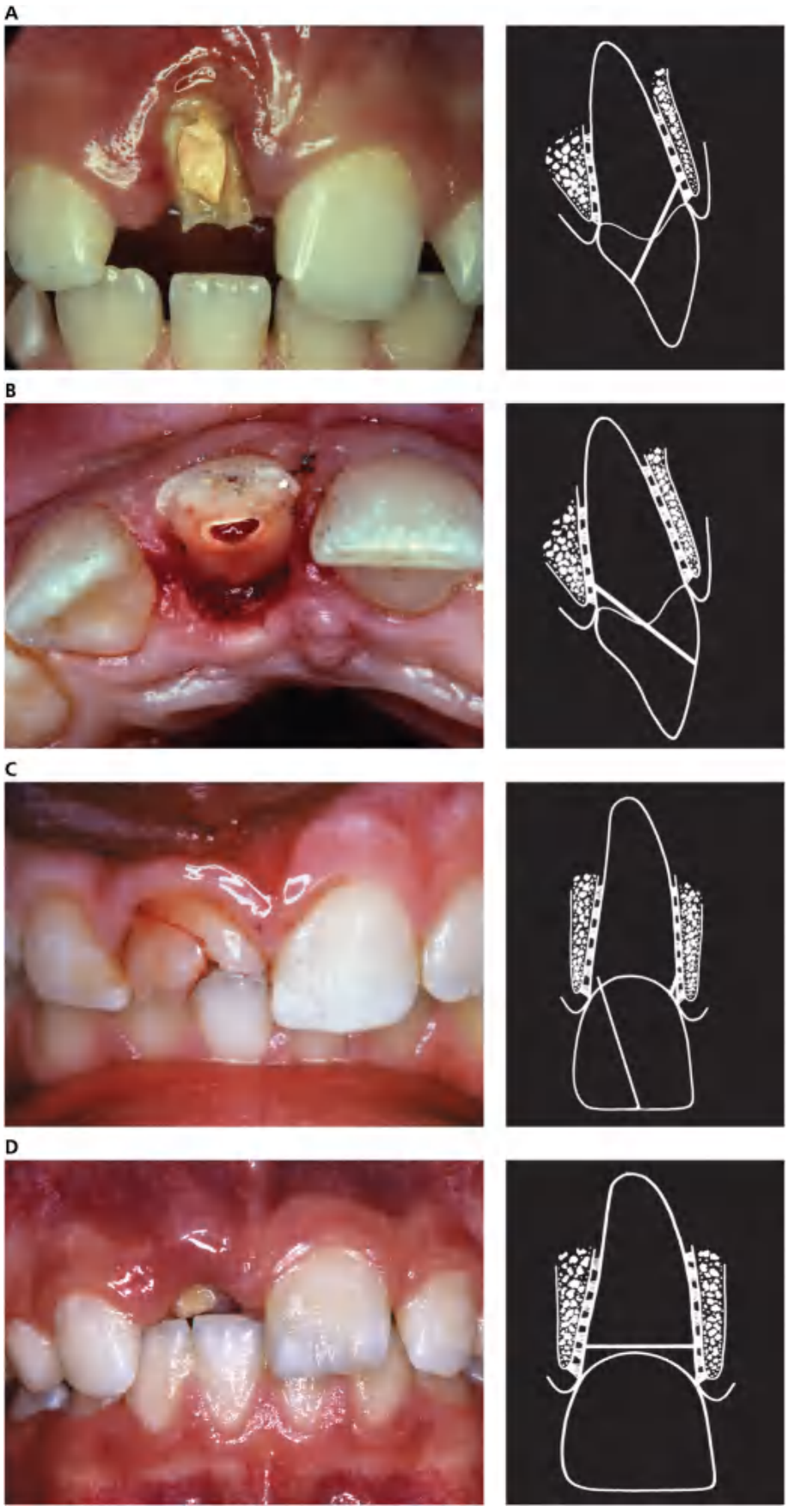



**Fig. 28.8 Orthodontic extrusion of teeth with crown-root fractures reaching below the marginal bone level**
A. Oblique crown-root fracture below the marginal bone buccally. B. Oblique crown-root fracture below the marginal bone palatally. C. Crown-root fracture extending below bone proximally. D. Transverse root fracture.

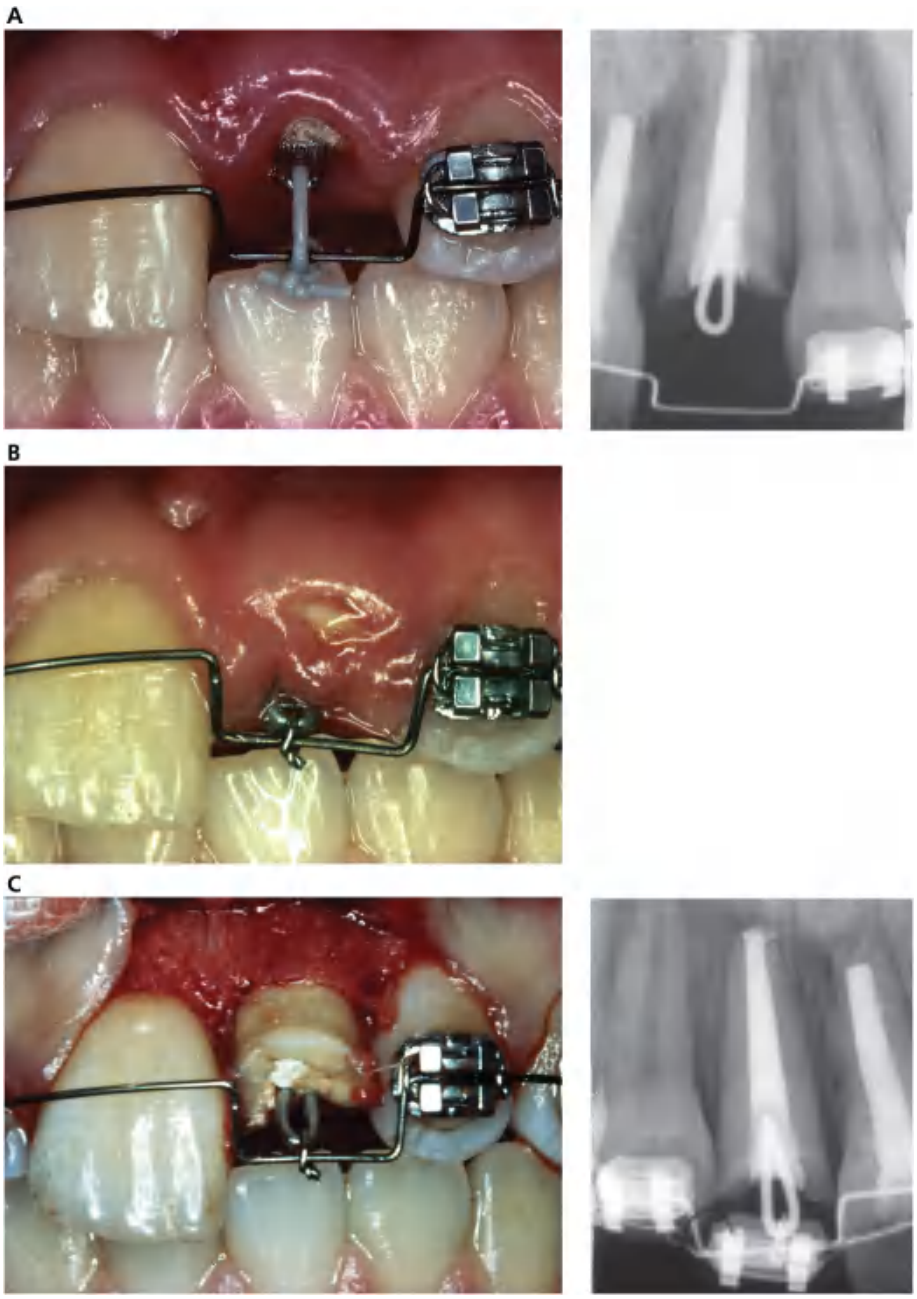



**Fig. 28.9 Rapid extrusion of a crown-root fractured central incisor**
A. A steel hook is cemented in the root canal. Traction with an elastic thread is applied axially. B. Condition after 4 weeks. The root is fully extruded and retained with a stainless-steel ligature wire. C. Condition after raising a mucoperiosteal flap. No coronal shift of the bone margin is found. Note the rapid remodelling of bone surrounding the apex

risk of further shortening of the very short coronal fragment. Orthodontic movement of such a tooth may result in a root with very little periodontal support. If the apical fragment is close to the marginal border it might be extruded with a rapid extrusion technique or by surgical means (Fig. 28.15). If a root-fractured tooth is endontically-treated and has an adequate root filling it may be orthodontically moved with the same prognosis as if the coronal fragment is vital.

## Luxations

After luxation of teeth, a number of complications (e.g. pulp necrosis, root resorption and loss of marginal bone) may occur. Clinical experience indicates an observation period of at least 3 months after a mild injury (e.g. concussion and subluxation). Pulp canal obliteration as a consequence of trauma indicates repair of the traumatized pulp. However, a repeated trauma or a heavy orthodontic force may cause further damage to the pulp (55, 56). After a moderate or severe luxation injury, endodontic treatment is most often needed. Then orthodontic movement should be postponed until there is radiographic evidence of healing. A follow-up period with clinical and radiographic controls of the endodontic treatment during 3 to 4 years has been advocated. If orthodontic treatment is indicated, however, it is not reasonable to wait such a long time before start or continuing treatment in most cases. But it is advisable from clinical experience to delay start of the orthodontic movement until a permanent endodontic treatment has been performed.

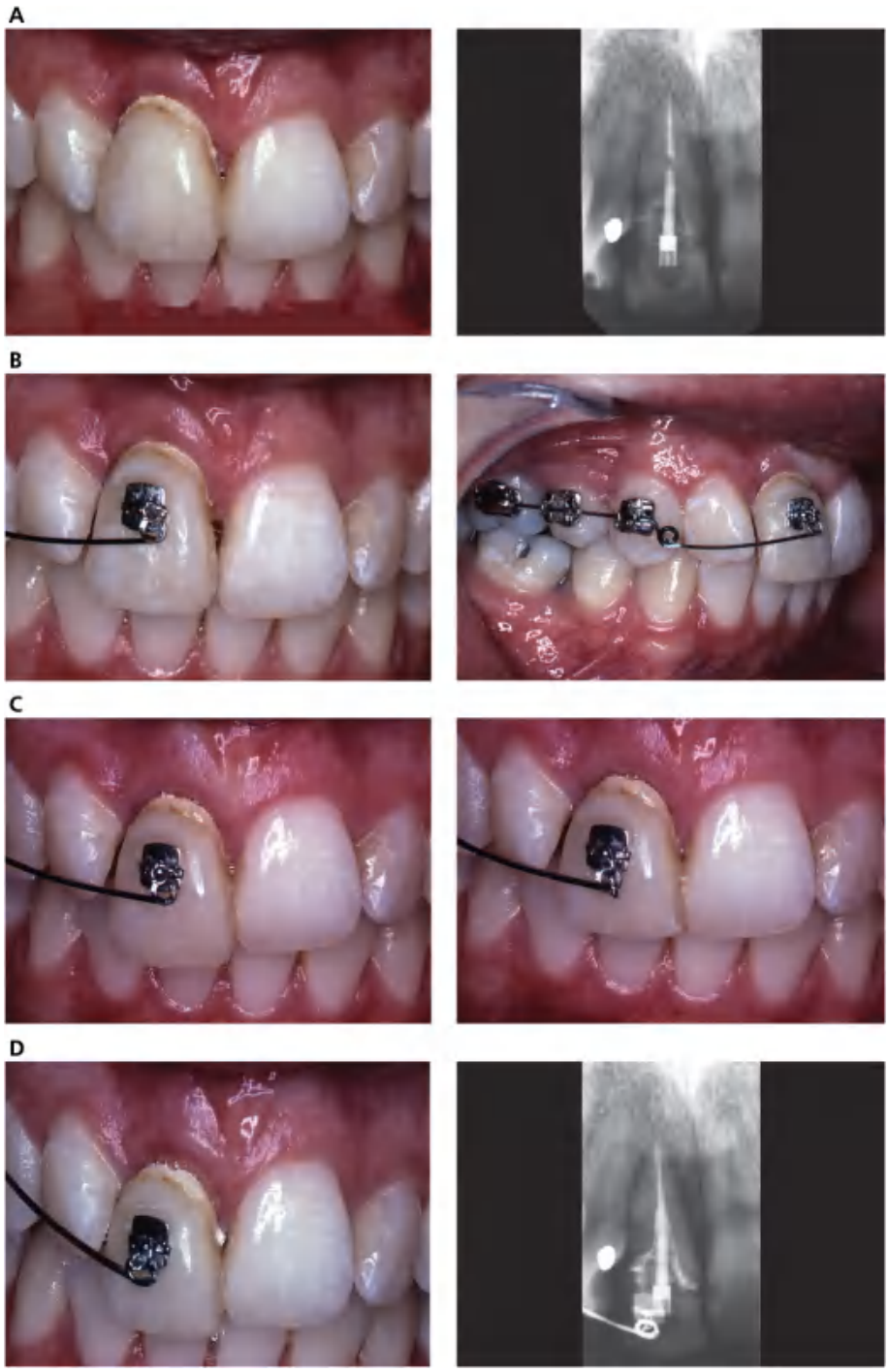



**Fig. 28.10 Orthodontic extrusion of a crown-root fractured central incisor**
A. The coronal fragment has been used as a temporary crown, cemented with a screw post in the root canal. B. Orthodontic appliance. A heat-treated spring of Elgiloy® wire (0.016 × 0.016 inches) with a force of approximately 60 to 70 p is used for the extrusion. C. Condition after 1 week. The tooth has been extruded 1.5 mm. The crown is shortened to enable further extrusion. D. Condition after 2 weeks. The tooth has been extruded another 1.5 mm and the root is now available for preparation.

## Intrusions

Intrusion of a permanent tooth into the alveolar bone is a severe traumatic injury. Pulp necrosis, infection related resorption and replacement resorption and marginal bone loss are complications that can be expected. The IADT guidelines for treatment of intruded permanent teeth distinguish between immature and mature root development (57). In teeth with very immature and immature root development, awaiting re-eruption seems to result in less healing complications. These teeth must be monitored carefully during re-eruption so endodontic treatment can be initiated if pulp necrosis is diagnosed. For mature teeth, the IADT guidelines recommend orthodontic or surgical repositioning in teeth with more than 3 mm intrusion depth. There seems to be no difference in the prognosis for orthodontic and surgical repositioning. The practicality of endodontic access, financial issues, number of visits and quality of life for the patient, makes surgical repositioning the preferred treatment (58–64).

The risks of pulpal damage and ankylosis are important considerations in orthodontic treatment planning of intruded teeth (58, 59, 61, 62, 64). Movement of a tooth with

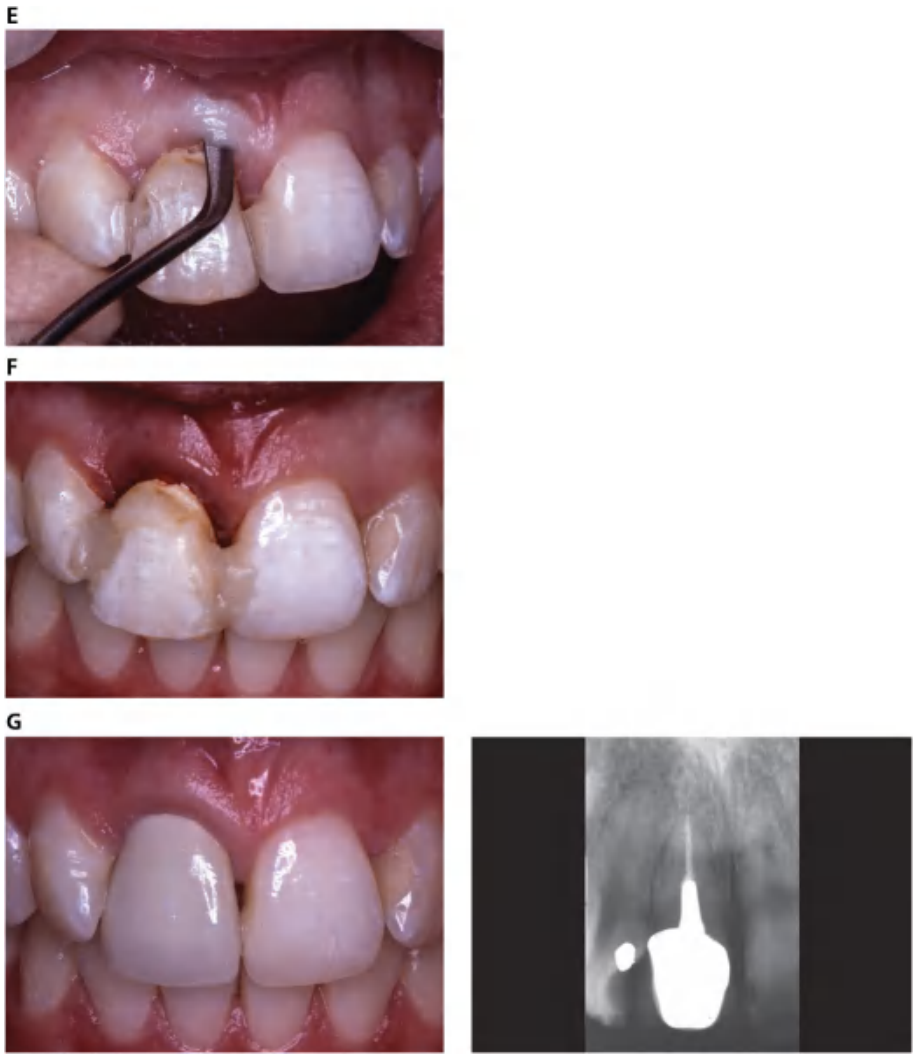



**Fig. 28.10** (*Continued*) E. Fibrotomy and retention. A fibrotomy extending below the level of marginal bone is performed prior to the retention period. The extruded tooth is splinted to adjacent teeth. F. Final restoration. G. A post-retained crown is fabricated. Radiographically there is no sign of resorption after 2 years.

small areas of replacement resorptions may be moved initially. Many years after treatment it will be obvious that replacement resorption further developed. Pulp canal obliteration as a consequence of intrusive luxation can be seen in immature teeth (65). Particular care should be taken during orthodontic treatment of such teeth. The risk of pulp necrosis is obvious (56). In experimental studies of intruded mature premolars in dogs, Turely et al. (66, 67) conclude that when an intrusive injury to the teeth was severe, orthodontic extrusion had little effect on repositioning of teeth but resulted in undesirable effects on anchorage teeth. When the injury was less severe the orthodontic forces could favorably reposition the affected teeth. A literature review by Chaushu et al. (68, 69) and AlKhalifa and AlAzemi (63) concludes that treatment of intruded teeth is based largely on empirical experience rather than on scientific data.

## Immature teeth

Intruded immature teeth may be left to erupt spontaneously (70, 71), or slightly loosened with finger pressure if the intrusion is not too severe. Orthodontic extrusion is indicated if the intrusion is severe or if the tooth has not begun erupting within 2–4 weeks. A plate with an extrusion spring is recommended for the extrusion in order to avoid pressure on neighbouring teeth (Figs 28.16 and 28.17). Intrusion always leads to pulpal damage. Sometimes the pulp may heal with subsequent pulp canal obliteration. Providing ankylosis does not develop, these teeth may have a good prognosis, as might teeth with pulp necrosis after adequate endodontic treatment.

## Mature teeth

Intrusion of mature teeth often leads to pulp necrosis particularly if the intrusion is severe, more than 3 to 4 mm (58–62). Prophylactic endodontic treatment is therefore recommended, as a necrotic and infected pulp can lead to rapidly progressive infection related root resorption. The critical period for the onset of external root resorption is 2 to 3 weeks. Therefore, in order to facilitate endodontic therapy, the intruded tooth must be at least partly repositioned within the first 3 weeks after injury. This can be accomplished either by orthodontic extrusion or surgical repositioning (66, 67, 72–74). If the intrusion is severe, there is a risk that orthodontic extrusion will have little effect. The tooth might be tightly locked in bone and orthodontic forces may not be able to overcome this mechanical barrier. A slight luxation of the tooth can facilitate the extrusion.

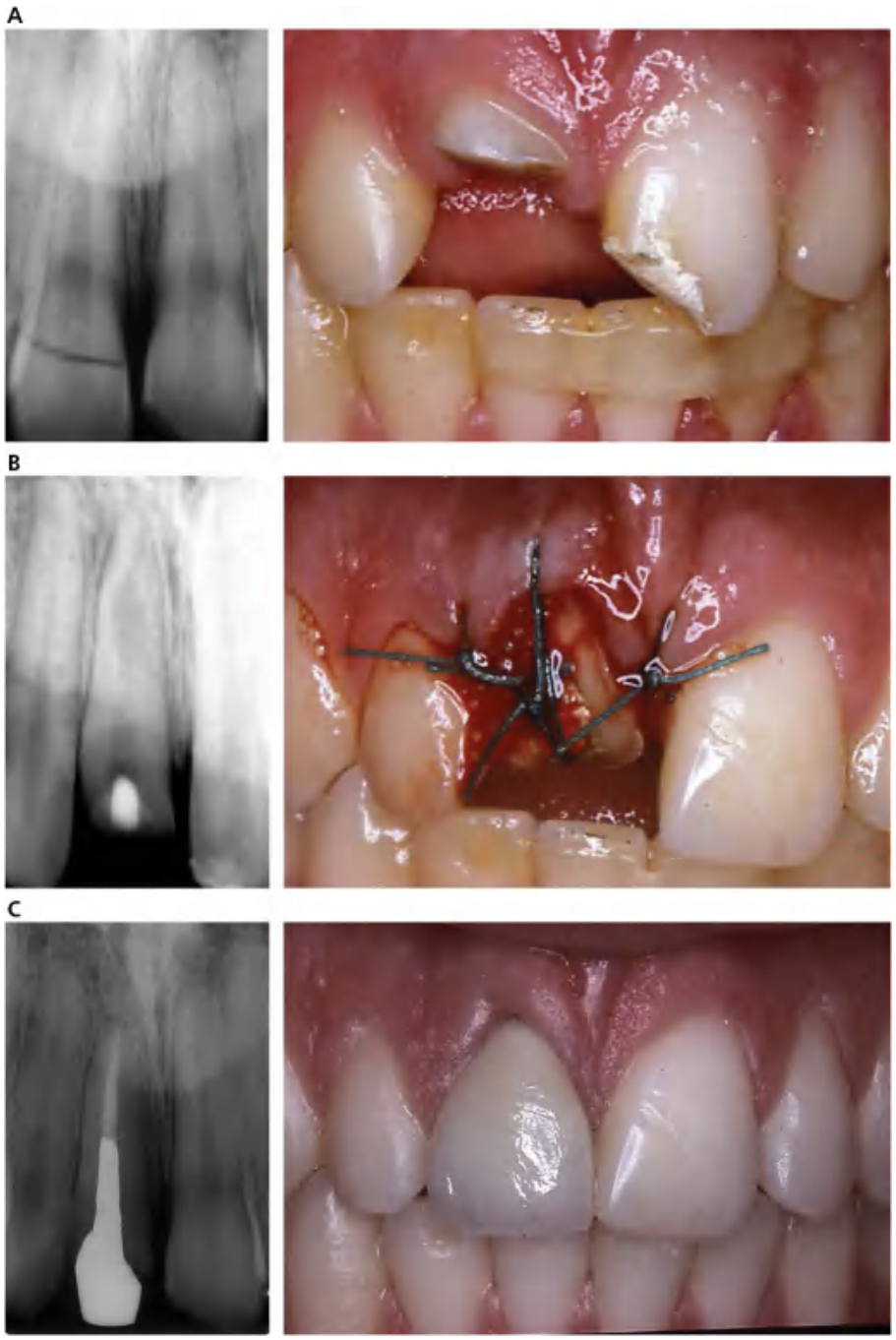



**Fig. 28.11 A 17-year-old boy with complicated crown-root fracture below marginal bone palatally of right upper incisor and an uncomplicated fracture of the left upper incisor**
A. The right upper incisor had been covered with a calasept dressing and zinc-oxide eugenol at the time of referral. B. The tooth was endodontically treated and surgically rotated 180° and fixed with single sutures. The left upper incisor was restored. C. A permanent root filling was performed after six weeks.

## Avulsions

### Space closure

Space closure is of prime importance in patients with maxillary arch length deficiency in Class I and II malocclusions. Treatment should start early or at least be planned in the mixed dentition. It is then possible by extraction or interproximal reduction of deciduous molars to guide molars, premolars and canines mesially. An intact dental arch can then be obtained with minor or no appliance therapy. Space closure for a missing maxillary lateral incisor usually implies mesial positioning of the maxillary cuspid in the lateral incisor region. Periodontal status has been reported to be better in patients with closed lateral incisor spaces than in patients with prosthetic lateral incisors (75). Treatment of missing maxillary lateral incisors in patients with Class I malocclusion is schematically illustrated in (Fig. 28.18) (76). The same principles can be applied in cases with loss of central incisors. Mesial movement of a lateral with fixed appliance to replace a missing upper central incisor is complex. There is a risk of space reopening, increased load on the root to support a larger crown and unfavorable fit of the prosthetic crown in the cervical area. Czochrowska et al. (77), however, found in a follow-up study of 20 consecutively treated

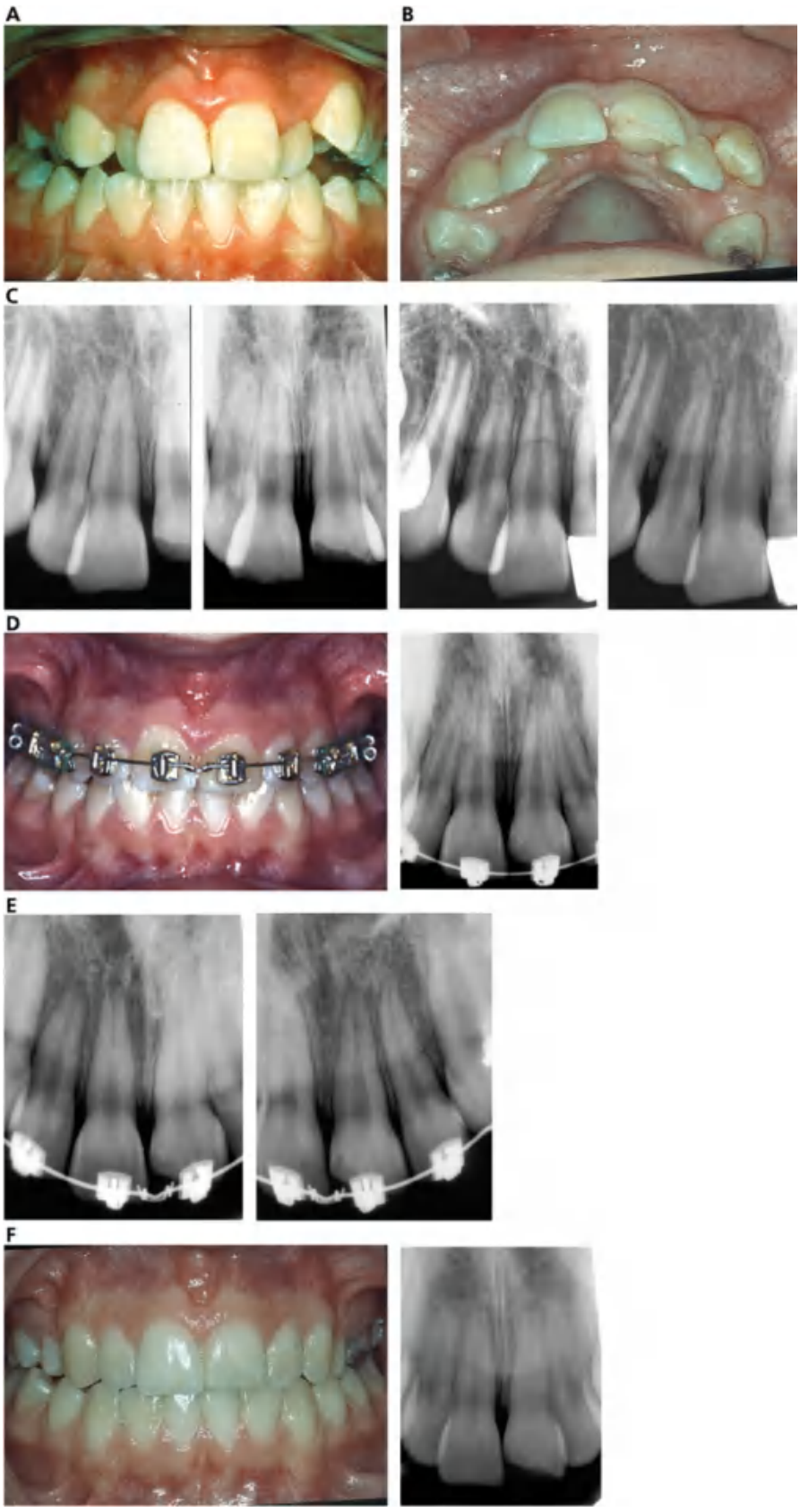



**Fig. 28.12 10-year-old girl with an intra-alveolar root fracture and a complicated crown fracture**
A. Status before injury. Orthodontic treatment was planned with extraction of four premolars. B. At the time of injury. A complicated crown fracture on the left upper incisor with a small pulpal lesion. A pulp capping with calasept was performed and covered with zinc-oxide eugenol and a temporary steel- crown. C. At control 3 weeks and 3 months later. An intra-alveolar root fracture is clearly visible on the right incisor. D. The orthodontic treatment was started 1.5 years after injury after extraction of four premolars. E. 2.5 years after injury the left upper incisor was subluxated due to second trauma, and it was desired to finish the orthodontic treatment. F. 4 years after the first injury. Note hard tissue healing on the root-fractured incisor and pulp canal obliteration of the subluxated left incisor.

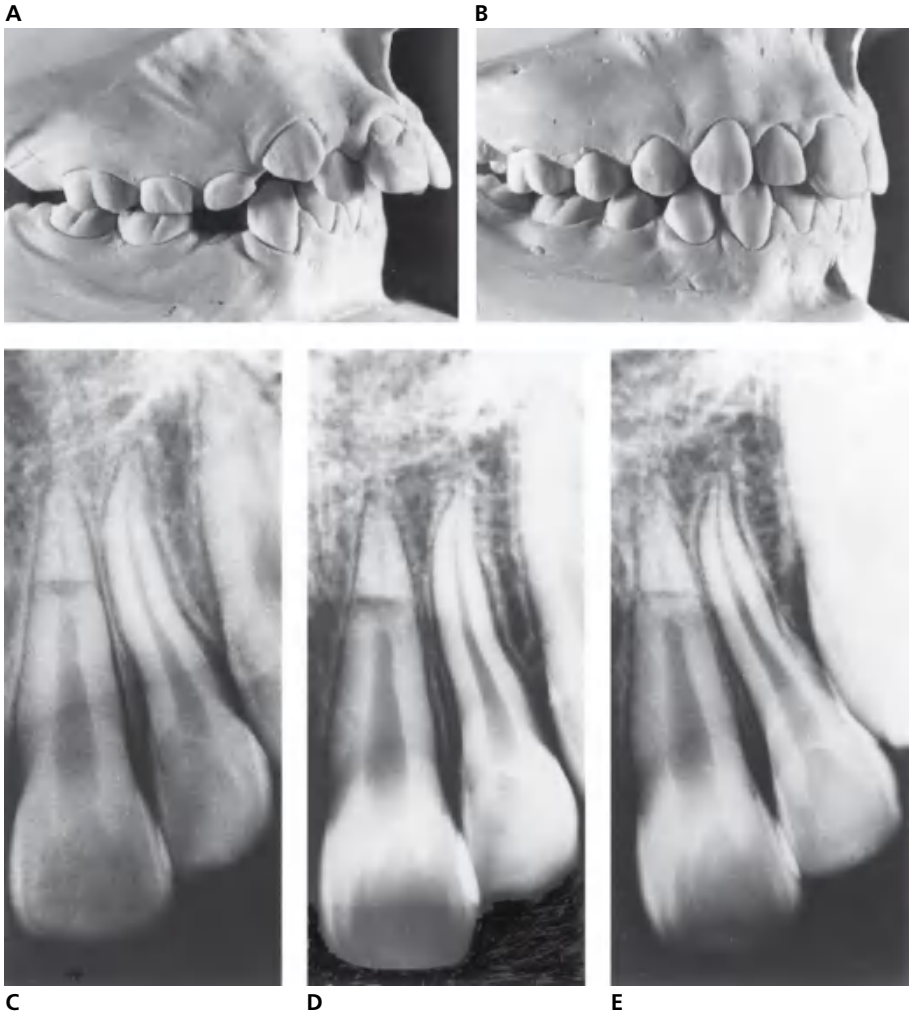



**Fig. 28.13 Orthodontic movement of a root-fractured tooth healed with a hard tissue callus**
A. Models of a Class II div. 1 malocclusion with deep bite before and after treatment. B. The patient was treated with extraoral traction and an activator for 2 years. C. The root-fractured incisor before treatment. Note internal surface resorption at the level of the root fracture, which is typical for hard tissue healing. D. Immediately after active treatment. There is obliteration of only the apical part of the root canal, also an indication of continued hard tissue fracture healing. E. Condition 4 years after active treatment. Note that tooth movement was achieved without separating the fragments.

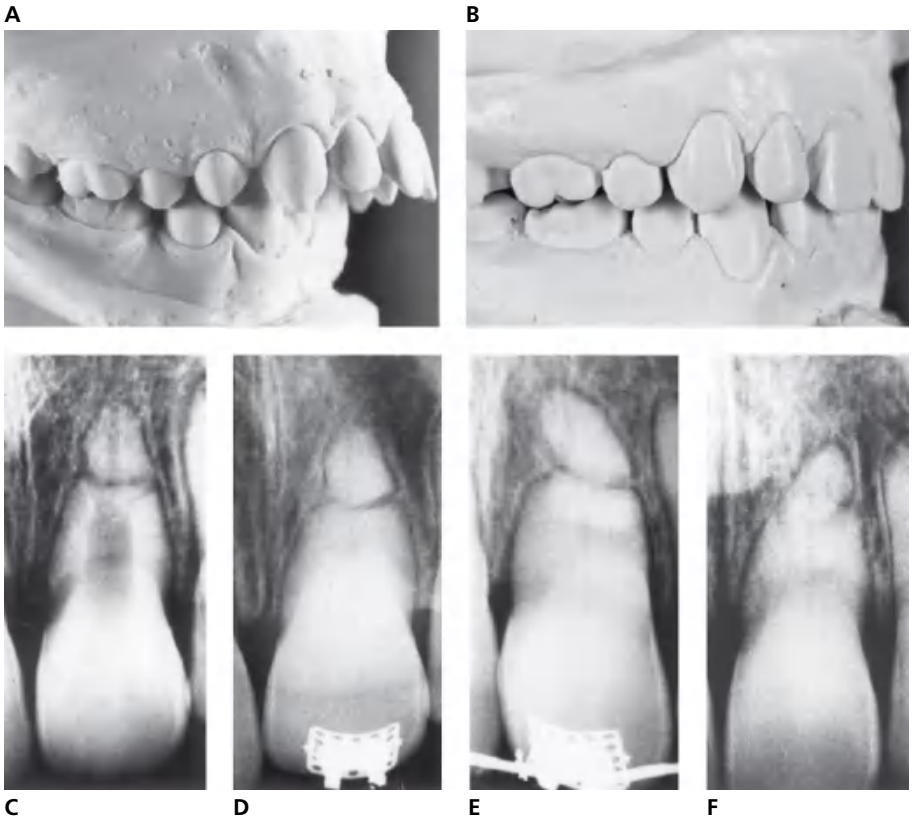



**Fig. 28.14 Orthodontic movement of a root-fractured tooth with healing by interposition of connective tissue**
A. Models of a Class II div. 1 malocclusion with deep bite. B. Models after treatment. The patient was treated with a fixed appliance for 19 months. C. The fractured incisor 3 years prior to treatment. Note initial obliteration of the apical and coronal aspects of the root canal. D. Condition at the start of orthodontic treatment. There is rounding of the fracture edges and obliteration of the entire root canal, indications of interposition of connective tissue between root fragments. E. Four months after the start of orthodontic treatment. F. Condition 2 years after orthodontic treatment.

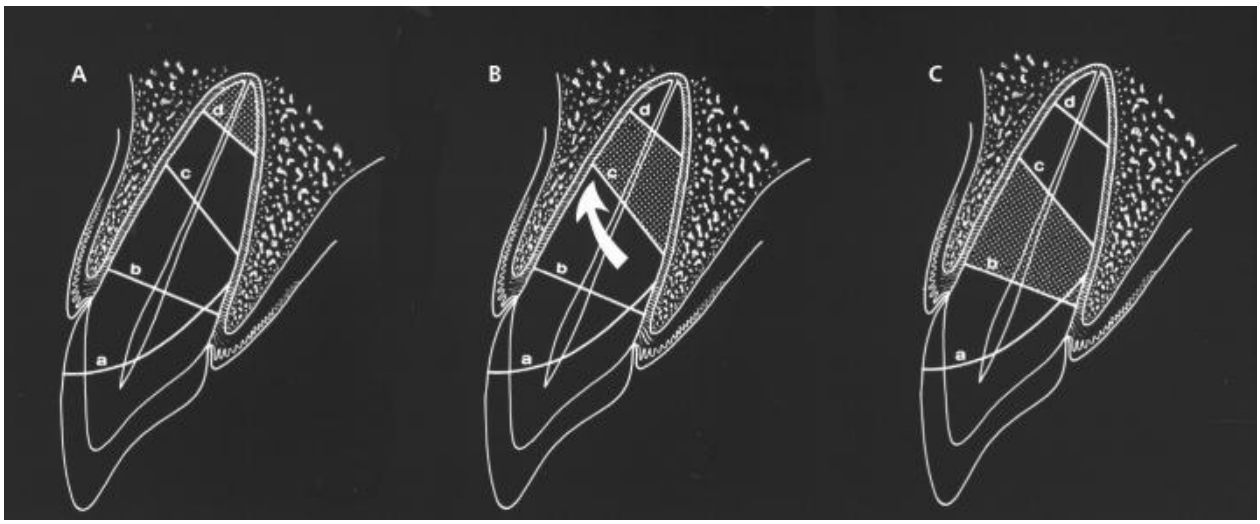



**Fig. 28.15 Evaluation of the prognosis for orthodontic treatment of root-fractured teeth healed with interposition of connective tissue**
A. Root fractures apically of lined. The coronal part of the root is so long that movement of the root is possible without impairment of the prognosis. B. Root fractures between d and c. The coronal portion of the root is long enough to withstand careful orthodontic movement. Note the thin labial part of the root (arrow) which can easily resorb during orthodontic movement and which will lead to a significant shortening of the root length. C. Root fractures between c and b. The coronal part of the root is usually too short to assure enough periodontal support and orthodontic treatment is therefore contraindicated. If the fracture line is too close to b, extrusion of the apical fragment is an alternative.

patients that most patients (85%) were satisfied regarding the esthetical result, but only 50% of the professionals. Another study showed that unilateral space closure often resulted in a high degree of patient dissatisfaction (see also Chapter 36). Crown form and inclination of the lateral incisors and canines must be favorable. The lateral must be properly up righted both in mesial-distal and labio-lingual direction (Figs 28.19–28.21). Crown form, dental arch symmetry and lip seal are important factors for an acceptable end result. Altering the crown forms with composite material in the mixed dentition and later on with porcelain veneers in the permanent dentition and grinding of canines, sometimes supplemented with gingivectomy to adjust differences in clinical crown height can contribute to an esthetical pleasing result (78–81) (Fig. 28.22). In rare cases with severe crowding in the upper jaw were two central incisors are lost, mesial movement of both laterals might be an alternative (Fig. 28.23). In cases where there is doubt about the functional or esthetical result of space closure, after incisor loss, it is advisable to wait until growth of the jaws is finished (77). Meanwhile, the space can be maintained prosthetically (82, 83). When treatment is planned, arch length and form, crown form and inclination of the anterior teeth should be studied carefully on casts and radiographs. Different alternatives of tooth position and compensatory extractions can be studied on diagnostic set-up models. The set-up model also facilitates discussion with the patient, parents and colleagues (Fig. 28.24).

## Space maintenance

When space closure is contraindicated, a space maintainer can be constructed. This situation arises if several incisors are lost in the same arch. It is also true in patients with good alignment of teeth and normal occlusion, and in patients with spaces between teeth. If a tooth is lost in the upper jaw in a Class II division 2 or a Class III malocclusion, a space maintainer is often the treatment of choice. For esthetic reasons, space maintenance is indicated if there is great discrepancy in crown form between central and lateral incisors. The crown form of cuspids sometimes also contraindicates space closure. Asymmetry of the upper incisors after space closure can be very obvious in patients with incomplete lip seal. A space maintainer followed by autotransplantation, a fixed bridge or an implant is then to be preferred. A fixed space maintainer followed by bridgework is sometimes the only possible treatment in an uncooperative patient who does not want appliance therapy. Various space maintainers can be used (see Chapter 34).

Autotransplantation of bicuspids to maintain the alveolar arch width and length in a growing child is illustrated in Figs 28.25 and 28.26. The anatomy of the transplant is subsequently altered by a composite resin restoration. Orthodontic treatment planning is important, to make transplantation possible at an optimal stage of root development of the bicuspids and to minimize movement of the transplant. Insertion of implants is important to consider but can only be used when growth of the jaws and alveolar crest is complete (see Chapters 34 and 35). If space maintenance is indicated, pre-prosthetic orthodontic therapy is often necessary. Malocclusions due to skeletal deviations (primarily open and deep bites, cross bites and Class II malocclusions) should be treated in the early permanent dentition. Remaining growth can thereby facilitate orthodontic therapy. After this treatment, semi-permanent prosthetic therapy can be provided. In other malocclusions,

**Fig. 28.16 Orthodontic extrusion of an intruded incisor**
A. An 8-year-old boy suffered intrusion of an immature maxillary right incisor. Over a period of 2 months there has been no sign of re-eruption. B. A removable appliance with an extrusion spring acting on a bracket bonded to the injured tooth. A light force was used to avoid damage to the pulp. C. The extrusion was complete within 2 months. Pulp necrosis and inflammatory resorption necessitated endodontic treatment. D. The clinical and radiographic condition is shown 4 years after trauma.

**Fig. 28.17 Loosening of two severely intruded central incisors combined with orthodontic extrusion**
A. Preinjury condition: An 8-year-old boy suffered intrusion of both central incisors 1 week after this photo was taken. B. The patient suffered intrusion of both central incisors. C. Both incisors were loosened with finger pressure. Note the infection related resorption on the central incisor. The radiograph is taken after orthodontic extrusion.

**Fig. 28.17** (*Continued*) D. Status 2 months after injury. There was no spontaneous re-eruption. Orthodontic extrusion was therefore initiated. Endodontic treatment was performed in the left incisor due to pulp necrosis. E. Status after 1 and 4 years. Clinical condition after 1 year and radiographic condition after 4 years. Note the partial pulp canal obliteration in the canal part of the pulp chamber of the right incisor.

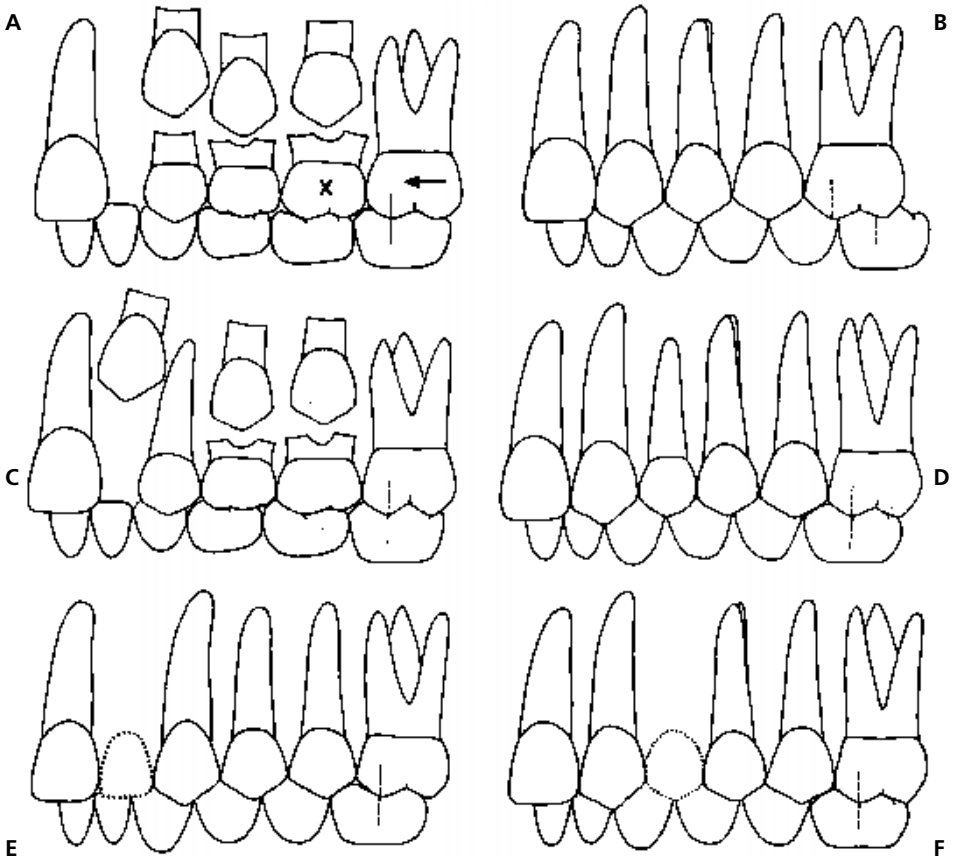



**Fig. 28.18 Treatment alternatives when a lateral incisor is avulsed in a Class I malocclusion arch length deficiency** A. Early extraction of the primary second molar facilitates mesial movement of the first permanent molar, arch length in the upper jaw is thereby reduced. B. The canine and premolars have moved mesially one tooth width and a Class II occlusion without spacing has been achieved. No arch length deficiency. C. The canine is positioned mesially. The root of the primary canine demonstrates no extensive resorption. D. The canine replaces the avulsed lateral incisor and the primary canine is used as a space maintainer as long as possible. E. A space maintainer is constructed while awaiting permanent therapy. F. Canine is used as a lateral incisor and a space is maintained between the canine and the first premolar.

**Fig. 28.19 Closure of space resulting from avulsion of a permanent central incisor**
A. The lateral incisor has moved mesially and has a fairly good position, which would have been better if the tooth had been up-righted. Note the mesial drift of the central incisor. A base plate or a lingual arch wire with a stop could have prevented this. B. The lateral incisor has been re-contoured by selective grinding and the crown form reshaped with composite resin.

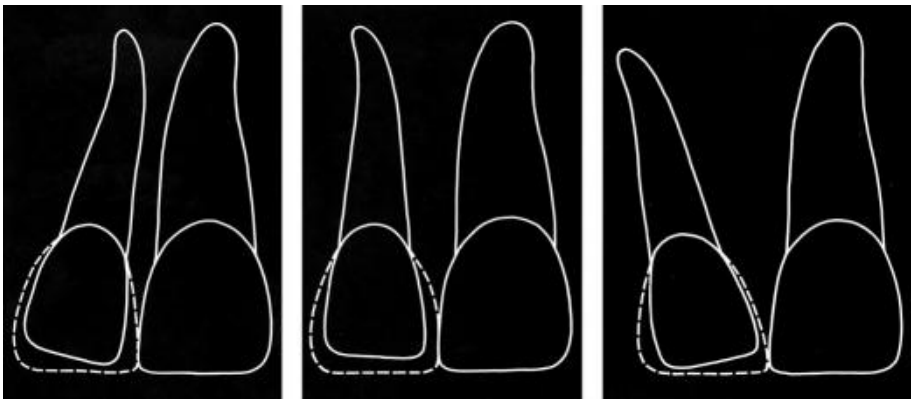


**Fig. 28.20 Schematic illustration of the need for mesio-distal uprighting of a lateral incisor** which has been moved mesially to replace a missing maxillary central incisor.

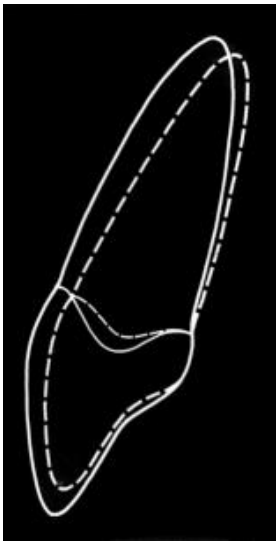


**Fig. 28.21 Schematic illustration of buccal root torque** of a lateral incisor which is moved mesially to replace a missing maxillary central incisor. The solid line illustrates the inclination of a central incisor and the dotted line a lateral incisor.

treatment can be performed in close association with the definitive therapy. This facilitates both orthodontic and prosthetic treatment planning and shortens the retention period.

## Replanted teeth

In cases where an avulsed tooth has been replanted, it may later be necessary to move the replanted tooth. The problem then arises whether orthodontic movement of the replanted tooth is possible without a significant risk of root resorption. Preliminary clinical studies (84–86) as well as an experimental study in monkeys indicate that severe root resorption can be elicited. Most root resorptions after replantation are seen during the 1st year after injury (see Chapter 4). Thus, if no such complication is seen in this period, movement of the replanted tooth is possible (Fig. 28.27). However, it must be considered that both replanted and intruded teeth may show progressive root resorption 5 or 10 years after trauma despite more optimistic evaluation earlier in the course of healing (see Chapter 4). Such teeth are often candidates for single implants after the growth period.

## Root resorption during orthodontic treatment

Orthodontic treatment planning involves evaluation of the type and extent of tooth movement needed, duration of treatment and if extraction is necessary. In patients where several factors indicate a risk of root resorption a short treatment time, less forces and a limited goal are possible alternatives.

High frequencies of orthodontically-induced apical root resorption have been reported in histological studies, and the

tissue reactions are well documented. In clinical radiographic studies the reported frequency varies. Phillips (87) found apical resorption exceeding one quarter of the original root length in 1.5% of maxillary central incisors and in 2.2% of the laterals. Linge and Linge (88) discovered apical root resorption of 3 mm or more in 4% of the investigated maxillary incisors. Individual variations have also been reported between patients and between different teeth in the same person (89–91). Substantial root resorption might lead to tooth mobility and cause reduction of supporting bone (92).

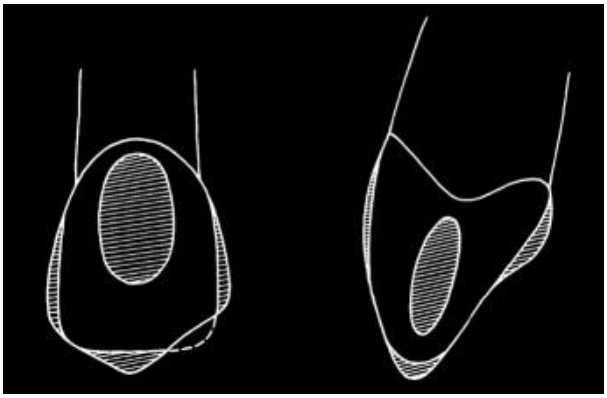


**Fig. 28.22 Reduction of a canine to simulate a lateral or central incisor.** The shaded areas illustrate the spots of grinding. The dotted line indicates where a corner can be enhanced by composite resin build-up.

Several studies have shown that small areas of resorption can be found in almost all teeth even if they have not undergone orthodontic treatment. Simultaneously a repair of these areas can be seen. The findings indicate that the cementum layer covering the root is a tissue that continuously changes. The periodontium consists of a mixture of mineralized and non-mineralized tissues that forms a unit with a complex mechanism reacting to the orthodontic forces. The reasons why severe root resorption sometimes occurs is not fully understood. The biological response to orthodontic forces is formation of compressed areas of the PDL which undergo hyalinization. This hyaline zone is described as a cell-free area which initiates cellular reactions in the adjacent alveolar bone and root surface. According to Reitan (46) the alveolar bone adjacent to the hyaline zone is removed by indirect resorption by osteoclasts working from adjacent marrow spaces on the surface of the alveolar bone.

**Fig. 28.23 An 11-year-old boy with a lost left upper incisor and an intruded repositioned right upper incisor**
A. At the time of injury. B. The canines start to erupt after a couple of months. Infraocclusion of the right central incisor can be seen indicating ankyloses. C. The right incisor was extracted 1 year after injury. D. Orthodontic mesial movement of both upper laterals to mimic central incisors.

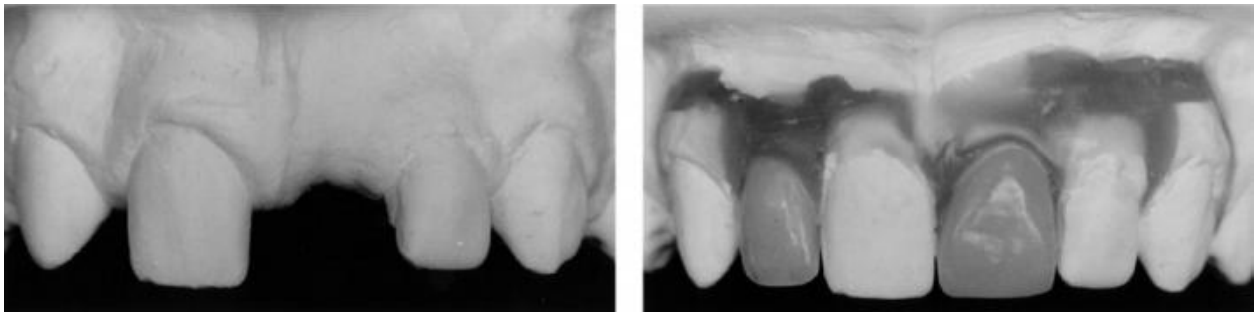


**Fig. 28.24 Set-up model as a guide in orthodontic treatment planning**
A model set-up for a patient with agenesis of the maxillary right lateral incisor and traumatic loss of the left central incisor. Treatment planning involves mesial movement of the maxillary right central incisor, distal movement of left canine and alignment of the maxillary teeth. The acrylic tooth was later used as space maintainers during the orthodontic treatment and retention.

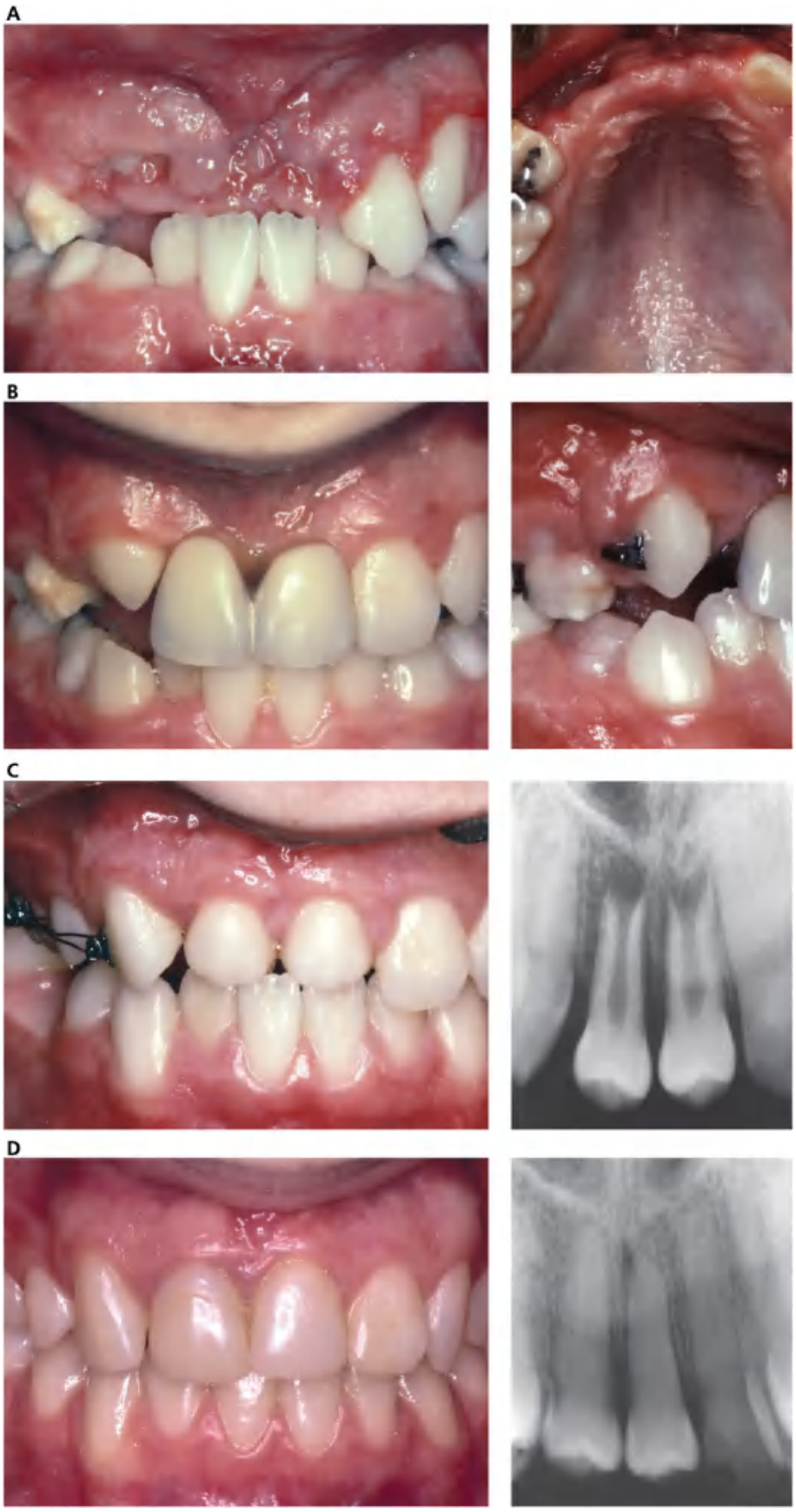



**Fig. 28.25 Combined orthodontic treatment and premolar autotransplantation after anterior tooth loss**
A. Three maxillary incisors have been avulsed in a 10-year-old girl. Note the extensive loss of alveolar bone buccally. Radiographic examination revealed that the mandibular second premolars were suitable as grafts. B. A temporary plate with 2 incisors is used as a space maintainer. After 5 months the upper right cuspid has erupted in a mesial position. The cuspid was therefore moved distally with elastics to make room for the 2 transplants. C. Two years after the trauma, the roots of the premolars have developed to 3/4 of anticipated root length. The premolars are then transplanted to the central incisor region. The teeth are aligned with a fixed appliance, 6 months after transplantation. D. At the end of treatment the premolars and the cuspid are recontoured with composite resin. The clinical and radiographic situation is shown 6 years after trauma and 4 years after transplantation.

**Fig. 28.26 Orthodontic movement of autotransplanted teeth**
A. A boy 12.5 years old with severe crowding in both jaws. Both upper incisors had a poor prognosis due to ankylosis after a traumatic injury. B. Both upper second permanent premolars were transplanted to incisor position. Note position of the lingual cusps. C. Orthodontic treatment and composite restorations, with radiograph 3 years after orthodontic treatment.

A similar resorption occurs on the dental root. No resorption of the root seams to occur immediately under the hyaline zone but appears during and after its eliminating. There is also a healing going on parallel to the resorption due to activity of the cementum cells. Under optimal conditions these simultaneous reactions interact so that the cementum seams unaffected at least on x-rays.

The interaction between biological and mechanical factors during treatment must be taken into account in assessment of the risk of root resorption, and should be included in the treatment plan although there is no definitive evidence in the literature of their importance (93, 94).

## Hereditary and gender

Familial difference in susceptibility for root resorption during orthodontic treatment was found in a study of a large number of full siblings indicating a heritable component (95). A correlation between gender and apical root resorption has been reported (96–98). Linge and Linge (88) reported that females are more susceptible than males. Other studies (99–101) failed to verify this finding.

### *Systemic factors*

Davidovitch et al. (102) have proposed that individuals with preexisting inflammatory conditions, such as periodontitis, diabetes and allergy are at high risk for orthodontic root resorption. Furthermore, Kjær (103) demonstrated a relationship between some dental anomalies, particularly ectopic eruption and agenesis of teeth, and tendency to root resorption during orthodontic treatment. An investigation designed to analyze the risk of root resorption in patients with agenesis verified this observation (104). In patients with multiple agenesis, the risk of root resorption must be considered carefully, because the teeth are often intended to

**Fig. 28.27 8-year old boy with an avulsed and replanted immature central incisor**
A. Status at the time of injury. The left upper central incisor was replanted and splinted. B. Revascularization was awaited, but 3 weeks after injury obvious signs of apical periodontitis was found and the tooth was endodontically treated and filled with calcium hydroxide and after healing with gutta-percha. C. 2.5 years after injury the upper canines were surgically exposed, and after another 2.5 years orthodontic treatment started. D. Status 9 years after injury.

serve as prosthetic abutments. The variables: treatment with rectangular archwires and intermaxillary elastics as well as duration of treatment was significantly related to the severity of root resorption. This may reflect the difficulty of controlling the force applied, when fewer teeth are available for anchorage (104).

### *Local factors*

Some morphological characteristics of the root anatomy can be diagnosed in pre- treatment intra-oral radiographs. Oppenheim (105) alleged that incisors with deviating root forms are especially at risk and similar observations were

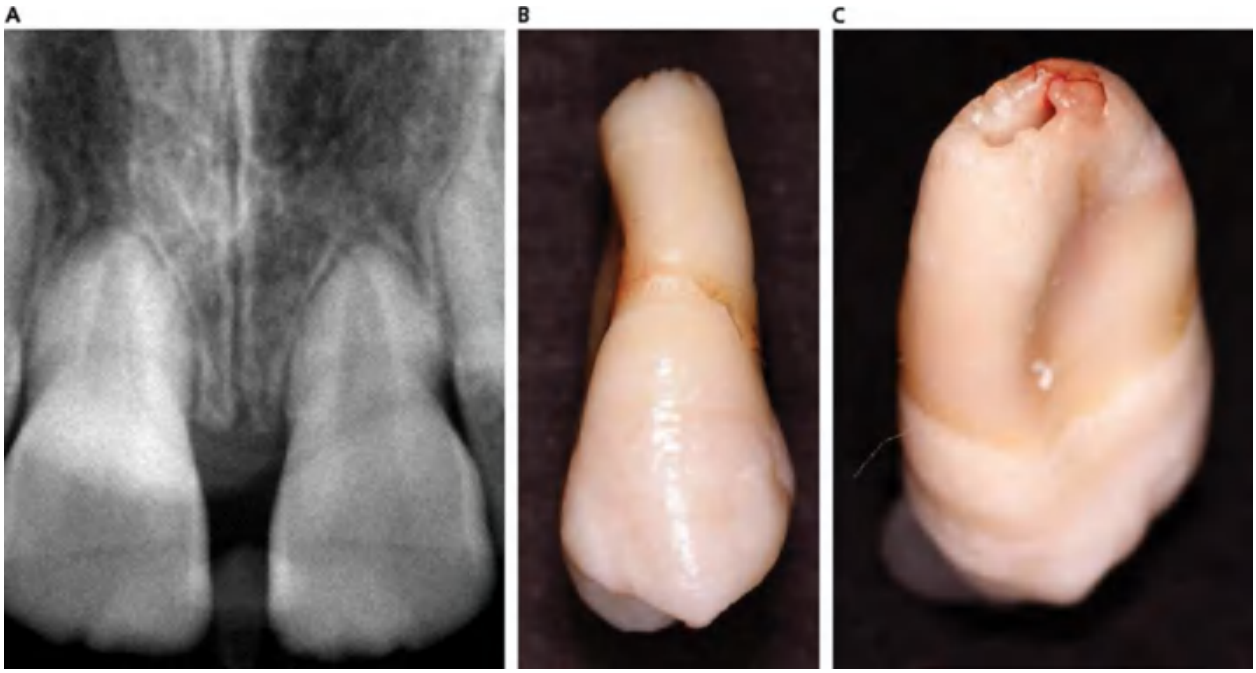



**Fig. 28.28 Short root anomaly is a development disorder and means that the root apex closes before the root is fully developed**
A. Two upper central incisors with short root anomaly. B. An extracted premolar with short root anomaly. C. The apical part of the tooth has a plumb shape.

made by Levander et al. (106). Lind (107) and Newman (96) claimed that the condition "short root anomaly" predisposes to root resorption during orthodontic treatment. Short root anomaly means that the roots are already abnormal at an early stage in development and the final root shape is not due to resorption. The anomaly always affects both central incisors almost symmetrically (Fig. 28.28). As a rule these roots are characteristically plump. Other teeth are more rarely involved, and when they are, it is usually premolars and canines (Fig. 28.28). No clear evidence of the risk for root resorption in these teeth has been shown. However, even a small resorption might jeopardize the long-term prognosis for the teeth.

It is also necessary to carefully study the anatomy of the roots (Fig. 28.29). An assessment of the radiographic outline of the apex provides useful information regarding the risk of root resorption during orthodontic treatment (106) (Fig. 28.30). Roots with pipette shaped, blunt or bent apices are more liable to root resorption than teeth with normal root forms (Figs 28.31 and 28.32). A root resorption index permits quantitative assessment of root conditions prior to treatment and can be used for evaluation of further resorption (106) (Fig. 28.33).

It has been shown that movement into labial or lingual cortical bone can initiate extensive root resorption, it is therefore important to establish the borders of the cortical bone, based on profile radiographs, prior to initiation of orthodontic therapy (108–110) (Fig. 28.34). Particularly if the alveolar crest is narrow, resorption can easily occur. During retraction of the maxillary incisors, it is necessary to avoid juxtaposition with the palatal or buccal cortical plates. In many situations, the incisors are protrusive and require palatal root torque during retraction. It is then advisable to perform this root movement in the roomier cancellous bone than in the compact cortical bone of the maxillary alveolus. It is therefore desirable to use a treatment approach that can intrude the anterior segment at the onset of orthodontic therapy (111).

### *Trauma*

In a study, by Malmgren et al. (112), root resorption after orthodontic treatment of traumatized teeth, 55 luxated incisors and their uninjured antimere were analyzed. All teeth had been carefully examined at the time of injury by experienced pedodontists according to standardized procedures. Signs of root resorption before and after treatment were registered with scores from 0 to 4. After treatment, 9% showed an irregular root contour, 32% minor resorption, 15 % moderate and only 4% (2 teeth) a severe resorption. The extent of resorption was the same in the traumatized as in the contralateral uninjured control teeth. However, a few teeth that exhibited resorption before orthodontic treatment were further resorbed during treatment.

### *Forces*

The importance of forces has been discussed for years. Most authors consider only heavy forces to be responsible for root resorption, but intensity and duration of forces are also of great importance. The conclusions in the literature are however conflicting. Kvam (113, 114), Harry and Sims (115) and Vardimon et al. (116) found an association between the amount of resorption and the magnitude of force, but Owman Moll (89) concluded that root resorption did not seem to be very force sensitive. It is often stated that forces

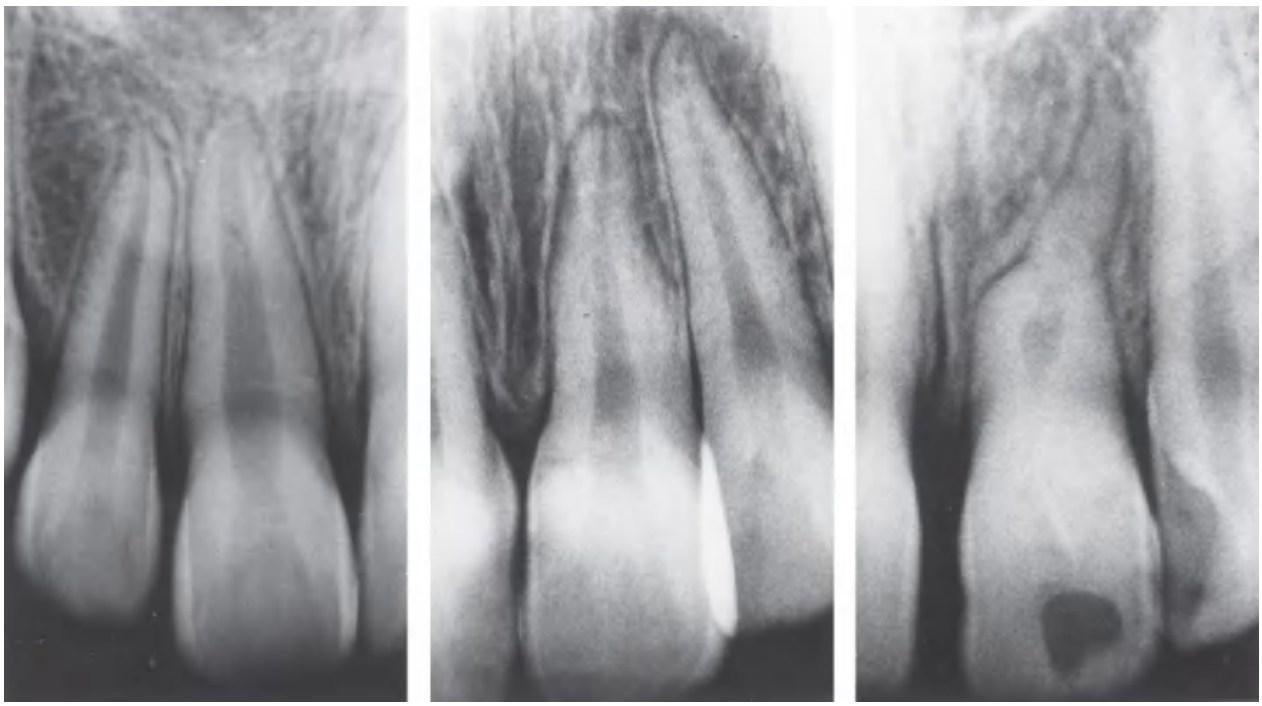



**Fig. 28.29 Root anatomy differing from normal. These roots are probably prone to resorption. Special care must be taken in these cases to avoid excessive forces during tooth movement**
A. Incisors with slightly irregular apical root contour. B. Concavity along the distal surface of the root (surface resorption). C. Root malformation.

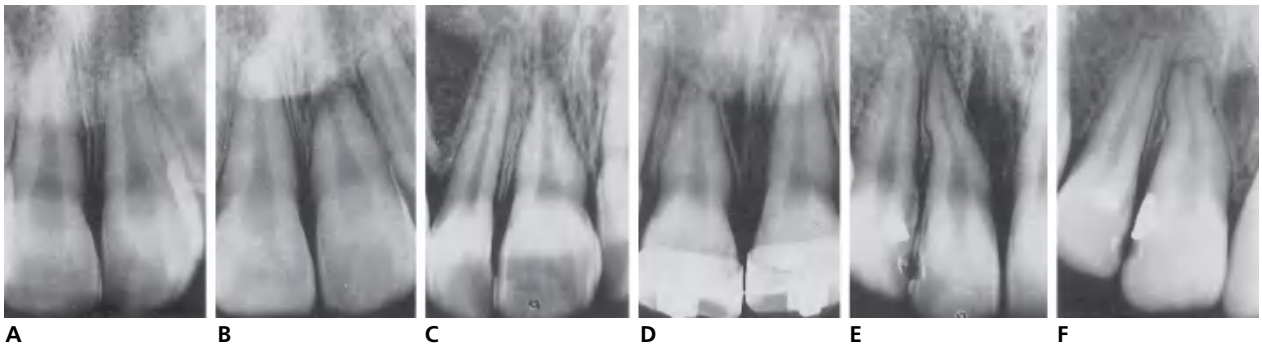



**Fig. 28.30 Root resorption developing during orthodontic treatment in teeth with abnormal root anatomy**
A. Slightly irregular apical contour of a left central incisor. B. Marked root resorption after treatment. C. Right central incisor with a concavity along the distal root surface. D. Root resorption seen after treatment. E. Malformed root of a right central incisor. F. Marked root resorption is evident after treatment. All patients were treated with fixed appliances for 18–21 months.

causing jiggling of teeth, intrusion and/or torquing of roots increase the risk of root resorption (88, 99). A correlation between resorption and the duration of active treatment has been shown in some studies (117, 118), but not in others (119, 120). Significantly more root resorption of central incisors was found in patients treated with standard edgewise compared to patients treated with straight wire edgewise (121). In another study less resorption was found when a technique with heat-activated and super elastic wires was used compared to standard and straight wire edgewise technique (122). It may be concluded that both treatment duration and mechanical factors and individual variations are of importance.

## Observation periods prior to orthodontic treatment

### *Endodontically-treated teeth*

The risk of root resorption during movement of endodontically-treated teeth has been debated for years. A slightly greater frequency of root resorption after orthodontic treatment has been reported in endodontically-treated compared to vital teeth by Wickwire et al. (123). Remington et al. (124) found that root-filled teeth sometimes resorb less than their vital antimeres (Fig. 28.35). These differences were statistically significant but minimal (0.7 mm). Similar observations have been reported by Spurrier et al. (125). Mirabella and

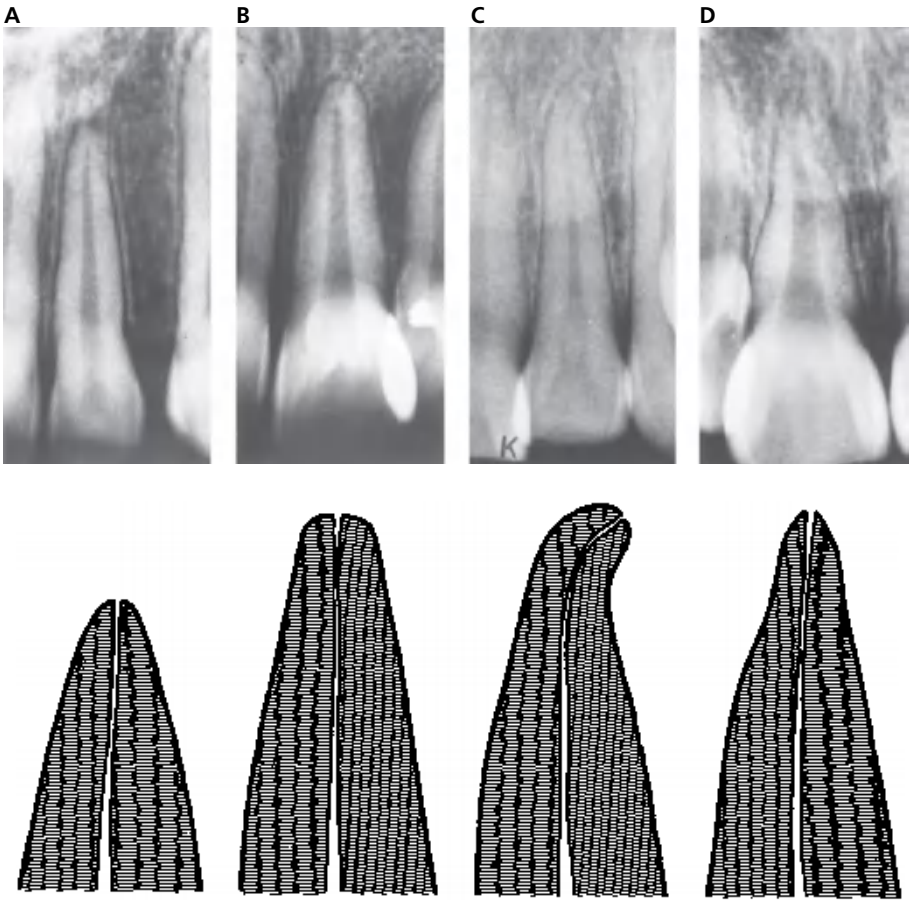



**Fig. 28.31 Deviating root forms**
A. Short root. B. Blunt root. C. Root with apical bend. D. Root with apical pipette shape.

Artun (126, 127) claim that endodontic treatment is a preventive factor. A few root filled teeth, however, resorb for unknown reasons (Fig. 28.36). An experimental study by Hunter et al. (10) showed no difference in the frequency of root resorption after orthodontic movement when comparing vital and endodontically-treated teeth. In a comprehensive literature review Hamilton and Gutman (128) conclude that minimal resorptive/remodelling changes occur apically in teeth that are being moved orthodontically well endodontically obturated. These conclusions coincide with the authors´ observation that root-filled teeth without signs of already existing root resorption can normally be moved without extensive root resorption (112).

### *Pulp canal obliterated teeth*

Pulp canal obliteration is common sequelae after luxation of immature teeth. It has been debated whether these teeth can be moved without losing pulp vitality or severe apical resorptions. Bauss et al. found an increased risk of pulp necrosis in a study of 59 teeth with severe periodontal injuries during orthodontic therapy (56). The risk of root resorption has been examined in a clinical study of 9 teeth by Malmgren et al. (112), but no apical resorptions or radiolucency were seen. Obliteration is also always seen after autotransplantation of immature teeth and even those teeth can be moved if root development continues and no complication occurs with only a limited risk of negative effects (129). However it is essential to closely monitor obliterated teeth during orthodontic movement.

### *Teeth with root resorptions*

Damage to the periodontal ligament after a trauma can result in three types of external root resorption: repair related resorption, infection related resorption and ankylosis related resorption. Repair related resorption implies a self-limiting resorption process, which is repaired with new cementum. Teeth with minor surface resorptions can be moved orthodontically with a prognosis similar to that of uninjured teeth. Infection related resorption implies a rapid resorption of both cementum and dentin with inflammation of adjacent periodontal tissue. This type of resorption is related to an infected and necrotic pulp and can usually be seen 3 to 6 weeks after injury. It is essential that proper endodontic treatment be initiated immediately. Arrest of the infection related resorption can be seen in about 96% of cases130. Orthodontic treatment should be postponed until radiographic healing is seen and at least one year should elapse before orthodontic treatment is instituted. Teeth with evidence of root resorption appear to be more liable to further resorption during orthodontic movement (112, 123, 130–135) (Fig. 28.37). This does not necessarily

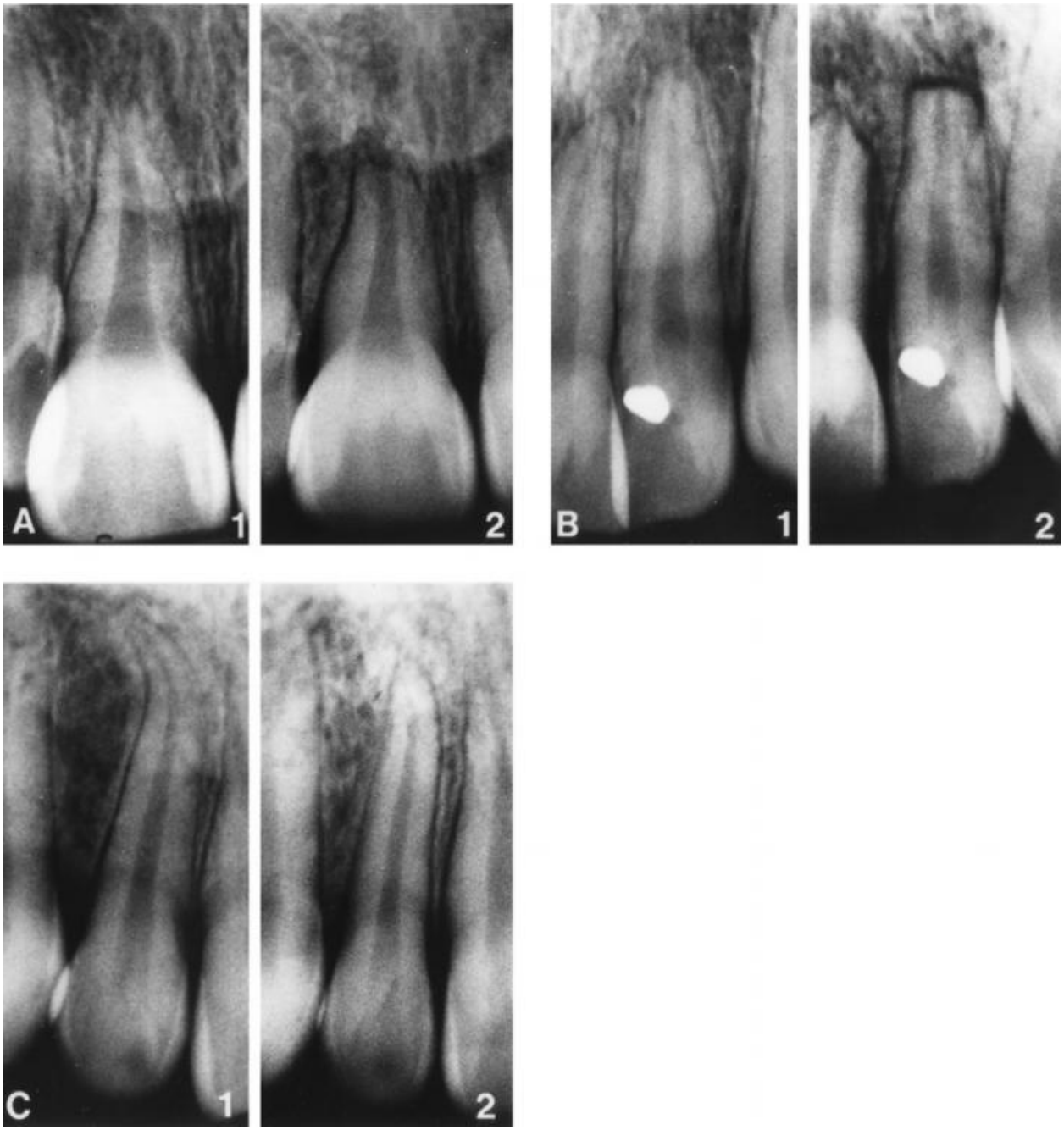



**Fig. 28.32 Typical effect of root resorption**
A. A pipette-shaped root (1) before and (2) after treatment. B. A blunt root (1) before and (2) after treatment. C. A root with an apical bend (1) before and (2) after treatment.

contraindicate orthodontic treatment; but special care should be taken to avoid excessive pressure during the tooth movement (Fig. 28.38).

Ankylosis related resorption implies fusion of the alveolar bone and root substance and disappearance of the periodontal ligament space. This type of resorption is progressive, eventually involving the entire root. Ankylosed teeth do not respond to orthodontic movement. A special extraction technique is then needed to avoid excessive loss of alveolar bone (136, 137) (see Chapter 32).

## Treatment principles

A clinical examination, including standardized periapical radiographs taken with a film holder should be performed in order to assess the potential contribution of local factors. Periodontal conditions should be registered. Based on the radiographic findings the following factors should first of all be evaluated: Earlier root resorption, including resorptions due to eruption disturbances (138, 139), apical root form

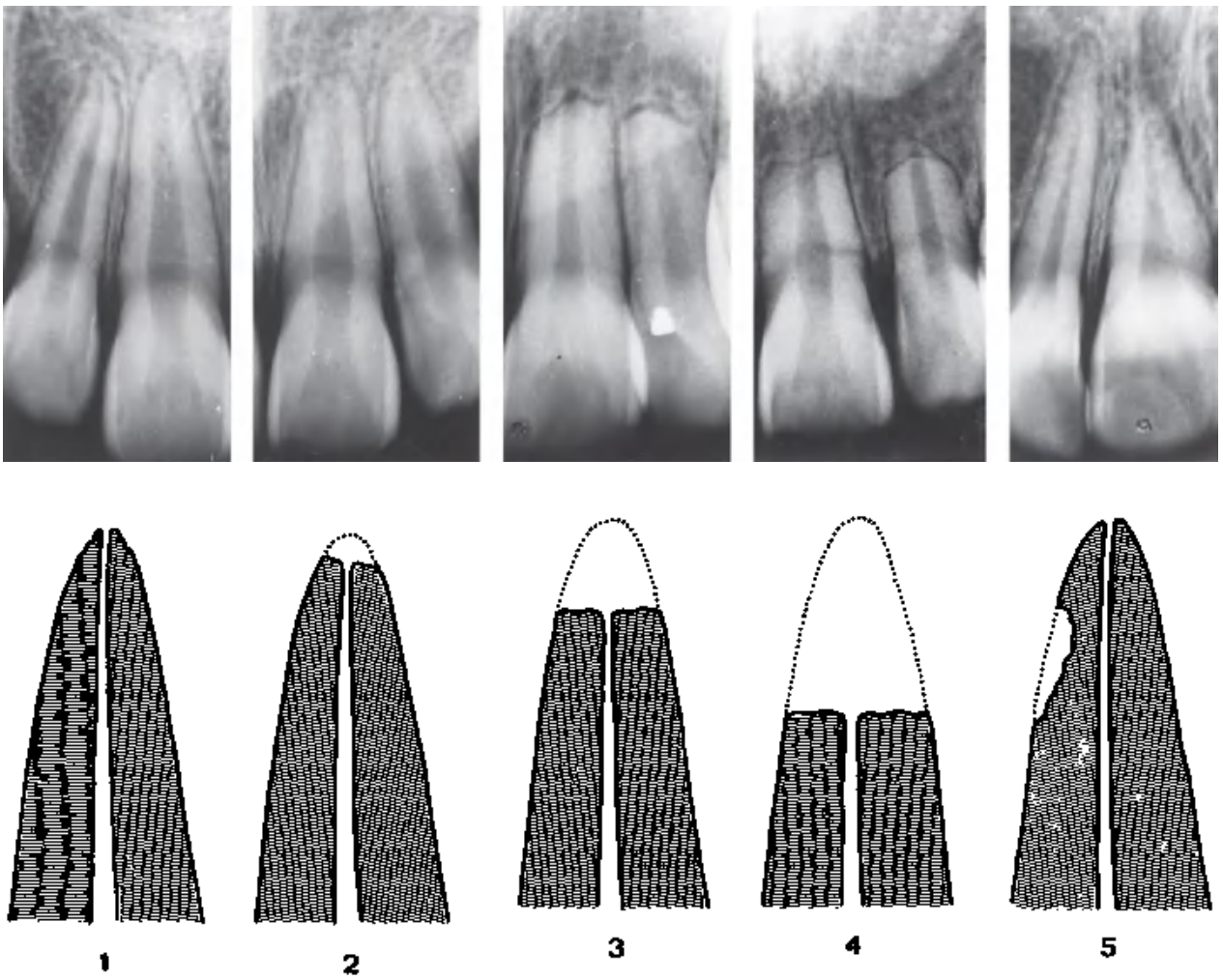



**Fig. 28.33 Root resorption index used for quantitative assessment of root resorption**
Score description: 1. Irregular root contour; 2. Root resorption apically amounting to less than 2 mm of the original root length; 3. Root resorption apically amounting to from 2 mm to one-third of the original root length; 4. Root resorption exceeding one-third of the original root length; 5. Lateral root resorption.

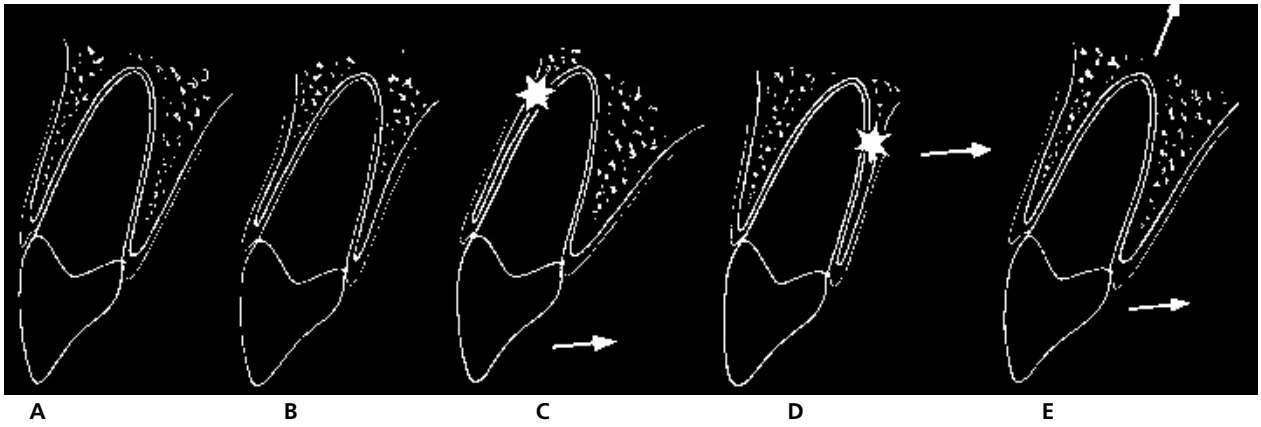



**Fig. 28.34 Schematic drawing illustrating the importance of examining the relation between the position of the root and cortical bone prior to orthodontic treatment**
A. An alveolar crest with a wide cancellous bone area. B. An alveolar crest with a narrow cancellous bone area. C. Lingual tipping of the crown moves the root into juxtaposition with the buccal cortical plate, which can initiate root resorption. D. Lingual root torque during retraction can move the root into juxtaposition with the palatal cortical bone, which can initiate root resorption. E. Intrusion of an incisor into the roomier cancellous part of the bone area is probably the best procedure.

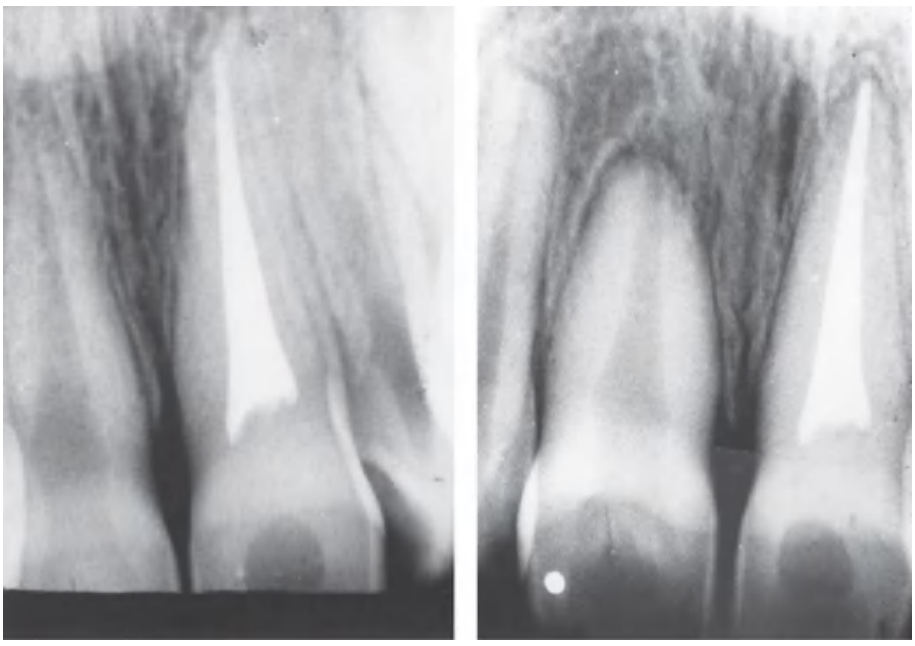


**Fig. 28.35 Response of a vital and a root filled tooth to orthodontic movement. The patient had a Class II div. 1 malocclusion and was treated with fixed appliances for 18 months**
A. Condition before orthodontic treatment. Both central incisors sustained traumatic dental injury. As a result, the left central incisor was treated endodontically due to pulp necrosis. B. After orthodontic treatment, the left central incisor demonstrates slight apical root resorption and the right central incisor, with a vital pulp, severe root resorption. Both teeth were moved orthodontically in the same manner.

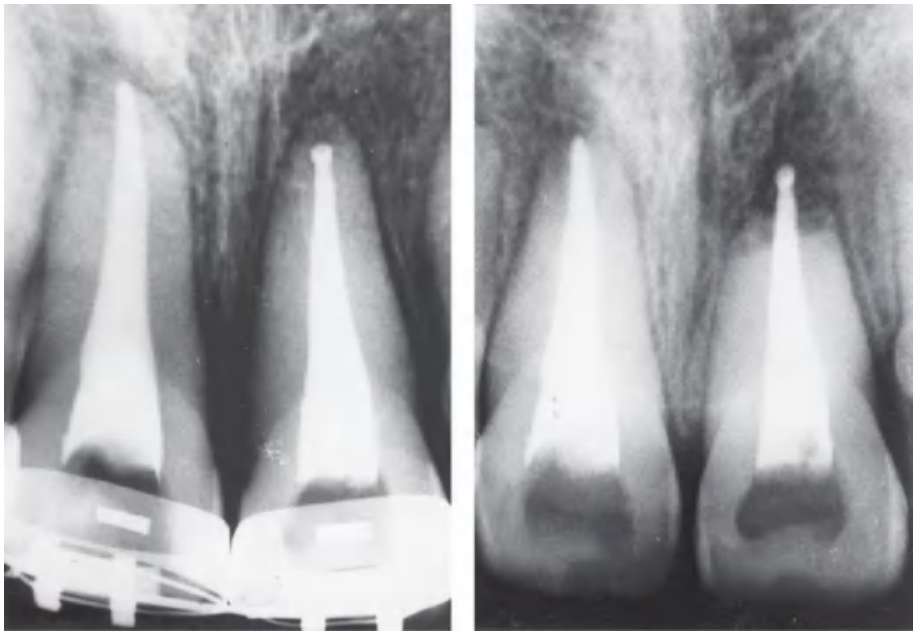


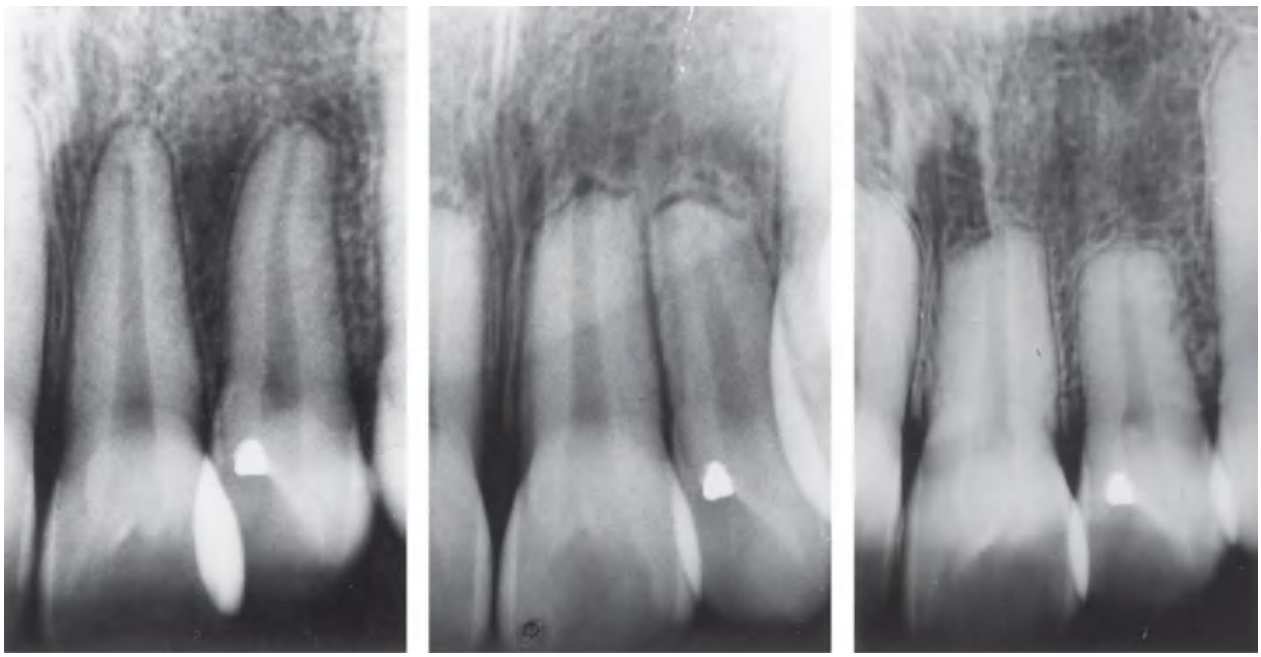
**Fig. 28.36 Different responses to orthodontic movement of two endodontically treated teeth. Both teeth were moved in the same manner. The patient had a Class II div. 1 malocclusion and was treated with fixed appliances for 19 months**
A. Before orthodontic treatment. Both central incisors were traumatically injured, and root filled. B. After orthodontic treatment, the right central incisor demonstrates slight root resorption, while the left central incisor demonstrates unusually extensive root resorption.

(126, 140), invagination (103), agenesis (104) and short root anomalies (96, 107, 141).

During treatment it is recommended that the first follow-up should be made after six to nine months (106). The follow-up should include periapical radiographs of all upper and lower incisors as these teeth are most susceptible to root resorption (122). It is important that the root apices are seen on at least two radiographs taken from different directions. In order to standardize the assessment of root resorption an index might be used (106) (Fig. 28.33).

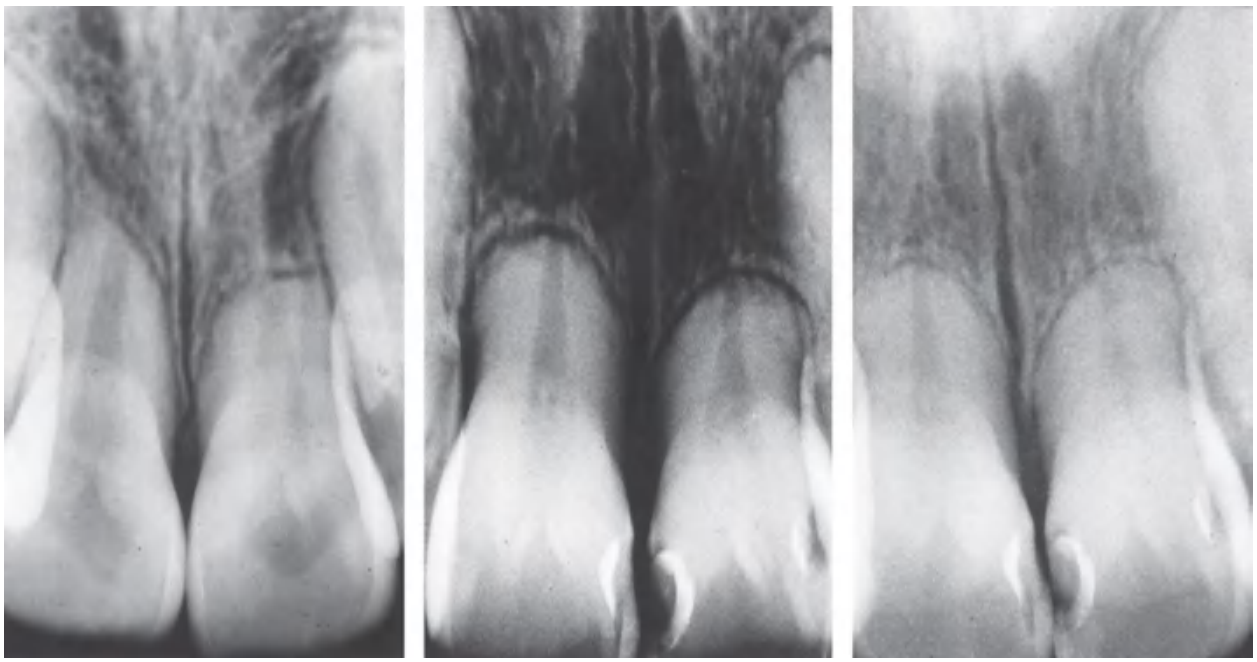
If no radiographic sign of root resorption is seen at the control there is a minimal risk of severe resorption at the end of treatment (Fig. 28.39 A). Resorption at this control indicates a risk of further resorption as treatment continues (Fig. 28.39 B) (106). The risk of severe resorption may be reduced by temporary suspension of active treatment for two to three months (Fig. 28.40) (93). In teeth where progress of resorption is seen, further radiographic follow-up every three months is recommended.

After treatment a radiographic examination is mandatory and the patient and referring dentist should be informed. If minor or moderate resorptions are seen, no further action is necessary. If the orthodontic treatment has led to a severe resorption, leaving a remaining root length corresponding



A B C

**Fig. 28.37 Vital teeth with signs of root resorption before orthodontic treatment**
A. The lateral incisor shows an irregular root contour due to root resorption prior to initiation of orthodontic treatment. B. Conditions after orthodontic treatment with fixed appliances for 20 months. Extensive root resorption of the incisors has taken place. C. At a 10-year follow-up, slight rounding of the resorbed root surface can be seen. Otherwise, there has been no progression of the root resorption.



A B C

**Fig. 28.38 Patient with Class II div. 1 malocclusion and severe root resorption of both central incisors due to trauma. Orthodontic treatment is possible if it is performed carefully**
A. Condition before orthodontic treatment. Activator treatment was carried out for 2 years. B. Seven years later, slight rounding of the root surfaces is seen. Secondary crowding developed later, and 4 first premolars were extracted. Fixed appliances were then used for 18 months. C. Control after 20 years. There has been no progression of the resorptive process.

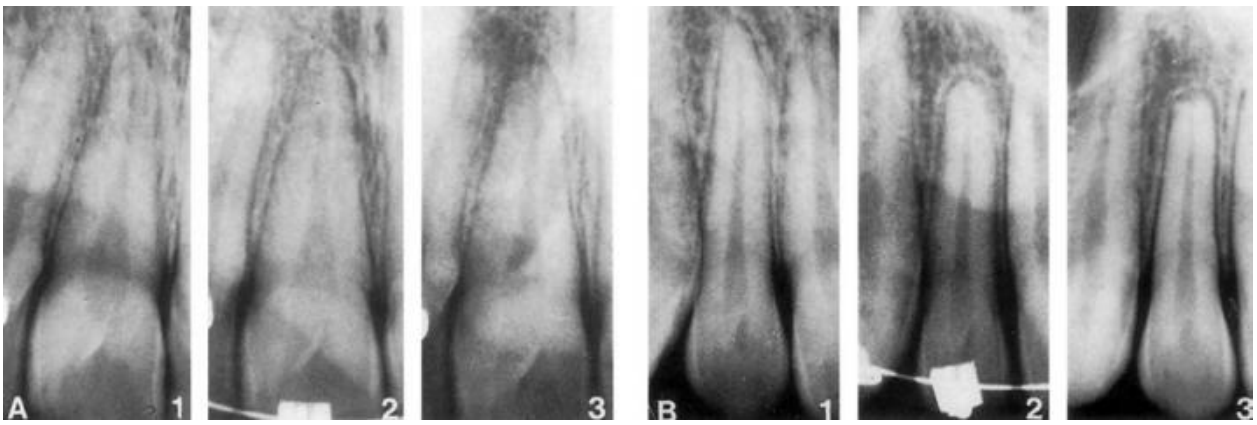



**Fig. 28.39 Root resorption after completion of treatment in relation to initial resorption status**
A. Status before treatment. (2) After 6 months there is no resorption. (3) At the end of treatment, minor resorption and blunting of the apex is seen. B. Before treatment. (2) After 6 months, minor resorption is seen. (3) At the end of treatment, severe resorption is seen.

to the crown of the tooth, there is a risk of tooth mobility (92). In such a case further follow-up and instructions to the patient are necessary.

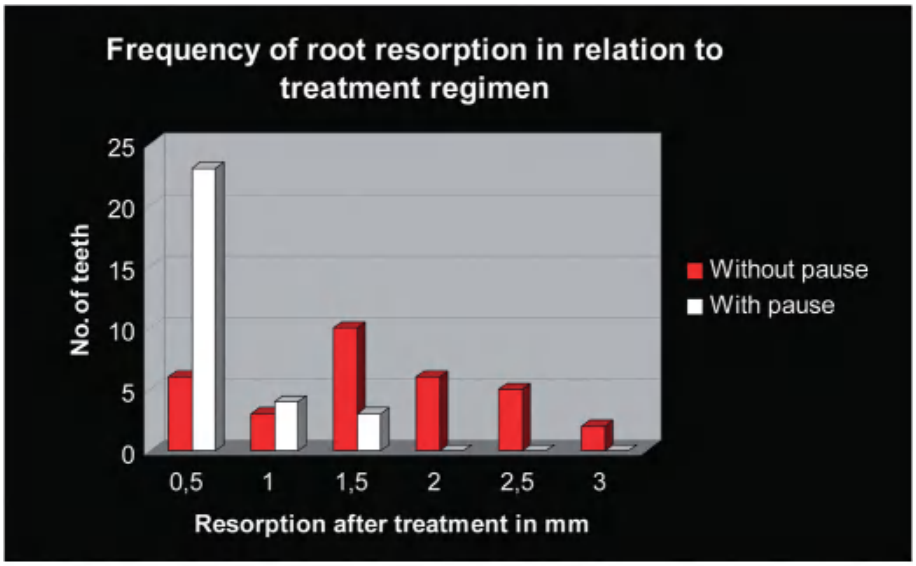
Cone beam computer technique (CBCT) is a new technique for follow-up of root resorption changes during orthodontic treatment. It provides high measurement accuracy and a precision that is superior to conventional radiographs. It shall however only be used in selected cases due to a relative high radiation dose and cost (142).

An important question is the management of apical root resorption detected early during active orthodontic treatment. Reitan (143) recommended a period of suspension of treatment in cases showing a strong tendency to root resorption. Rygh (144) goes even further by proposing the scheduling of periods of treatment suspension throughout the course of orthodontic therapy. There is consensus that root resorption usually ceases after orthodontic treatment, when applied forces are discontinued (119, 124, 145). Histological studies have shown that repair of resorption cavities will take place after force removal. Brudvik and Rygh (146, 147) studied the reparative process in animal experiments in rats and observed new mineralized cementum on the resorbed root surface 21 days after the forces were discontinued. In human experiments, Owman Moll (89) found various degrees of repair after 8 weeks, and Odenrick et al. (148) reported deposition of hard tissue in resorption lacunae after 53–90 days. In a study where active treatment of initially resorbed teeth had been suspended for 2–3 months, there was significantly less post-treatment resorption than in cases where treatment proceeded without interruption (149).

### *Long-term prognosis*

Little is known of the long-term prognosis of teeth with severely or extremely resorbed roots. In agreement with an earlier study by VonderAhe (119), Remington et al. (145) reported that even teeth with severe orthodontically-induced resorption seemed to function reasonably well clinically, several years post-treatment. In a case report of a patient with extremely shortened maxillary central incisor roots, examined 33 years after treatment, radiographic, visual and tactile examination revealed that the resorbed teeth functioned well (150). It has been claimed that root resorption up to one third of the original root length does not result in decreased stability (151), because the highest percentage of periodontal attachment is in the crestal two thirds of the root according to Henry (152). If there is a considerable decrease in the original root, the survival of the tooth might be compromised. Such a tooth might not resist normal functional loads. A further risk is that in the event of crestal alveolar bone loss, a critical stage for the residual periodontal attachment may be reached prematurely in teeth with pronounced apical root resorption (151).

The clinical sequelae to severe root resorption are of great interest. Wainwright (153) concluded that resorption could lead to mobility and even to exfoliation of the tooth. Of 57 teeth with post-orthodontic root resorption, VonderAhe (119) reported only one mobile tooth. Sharpe et al. (118) investigated orthodontic relapse and reported a higher prevalence of root resorption in the relapse group than in the control group. In a long-term follow-up study of 139 teeth with root resorptions 10 to 25 years after orthodontic treatment by Jonsson, Levander and Malmgren (92, 154), an increased mobility was found in incisors with a total root length <9-mm (Fig. 28.41). There was no difference between central and lateral incisors. Loss of root length moves the centre of resistance coronal so that the same amount of force will have a greater impact than on an intact root. The results of the study indicate that if the root is shorter than the crown the risk of tooth mobility enhances.



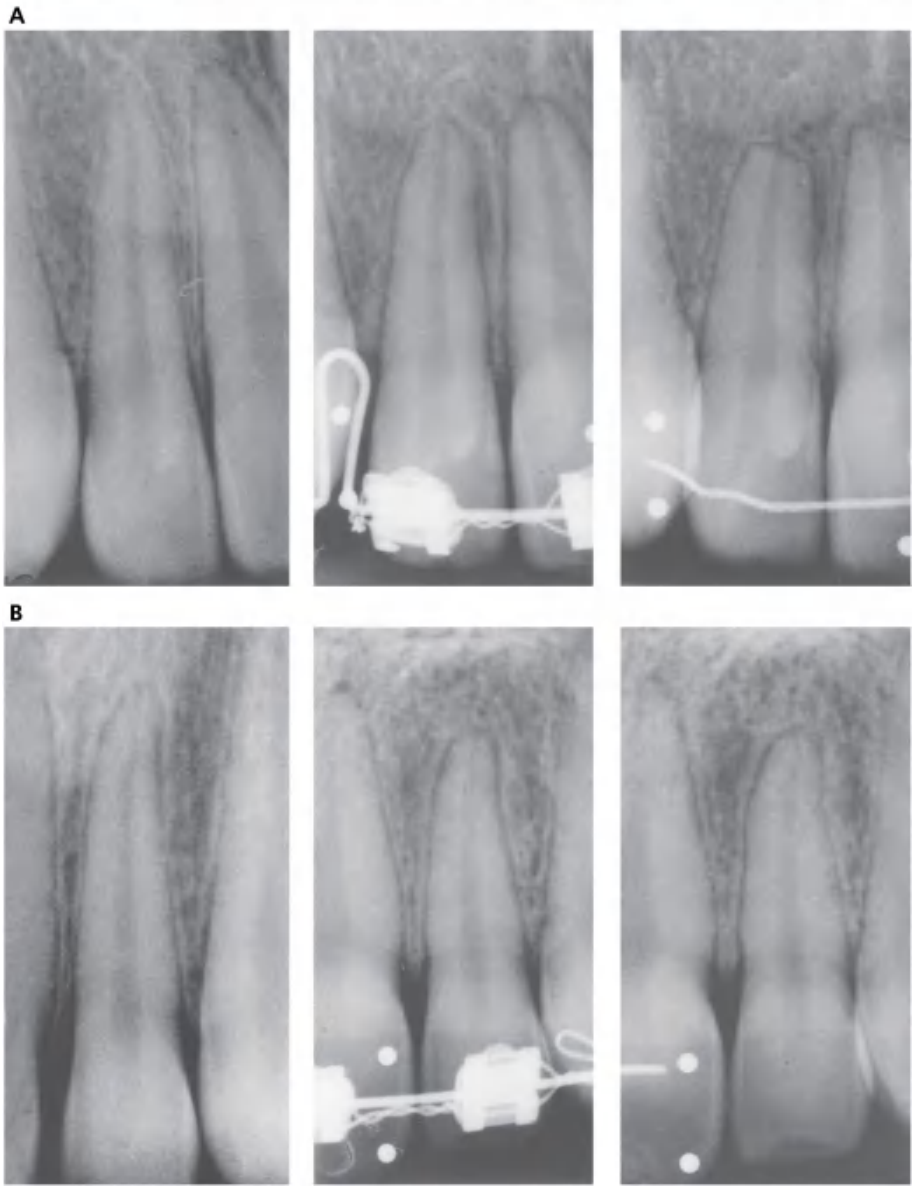




**Fig. 28.40 Root resorption in relation to two orthodontic treatment regimens**
A. Continuous treatment. An upper incisor before treatment; after 6 months when a minor resorption is seen; and at the end of treatment, when no further resorption is observed. B. Treatment was performed without pause. An upper incisor before treatment; after 6 months when a minor resorption is seen; and at the end of treatment, when no further resorption is observed.

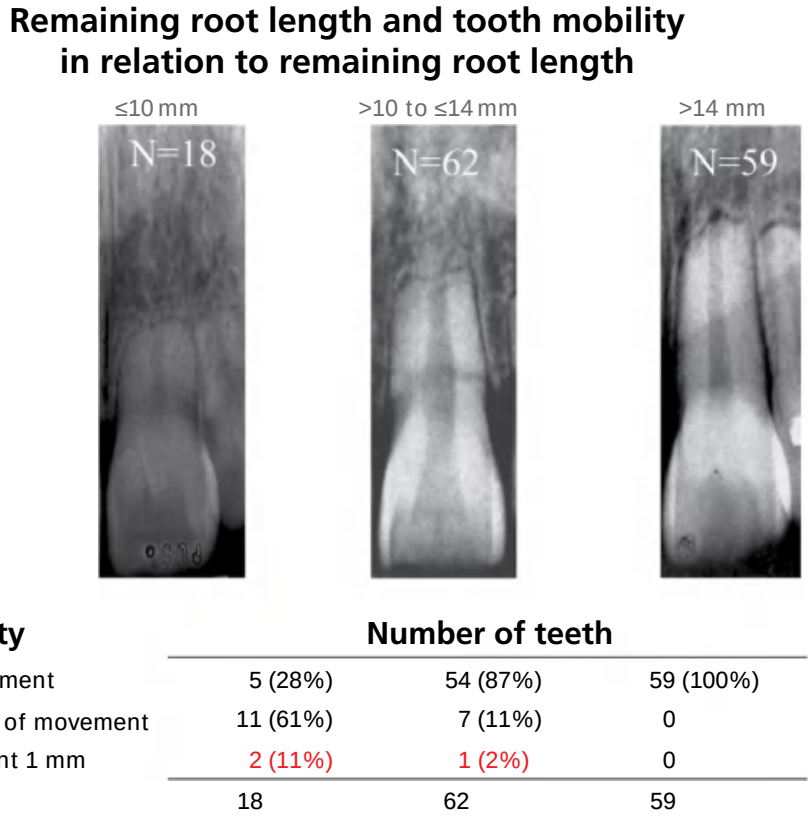



**Fig. 28.41 Tooth mobility in relation to remaining root length**
Tooth mobility in relation to remaining root length in a long-term follow-up study of 139 upper central and lateral incisors with root resorptions 10 to 25 years after orthodontic treatment. Tooth mobility was registered using Miller's index and the periotest method. Increasing mobility was seen in teeth with extremely resorbed roots, ≤ 10 mm. Teeth with a root length > or =10 mm and a healthy periodontium remained stable.

## Essentials

### Mixed dentition

Many factors shall be considered after a severe trauma. What will be the long-term outcome of the traumatized teeth, which are the possible treatment options for reconstruction of a malocclusion with traumatized teeth, what will be the outcome of an orthodontic treatment if a complication occurs due to the trauma and which are the treatment options if a tooth is lost? All these factors vary individually, and a careful treatment planning is important.

#### *Risk factors*

- Increased overjet and proclined upper incisors
- Insufficient lip closure

### Preventive orthodontics

- Children with an increased overjet shall be provided with a mouth guard during sport activities.
- Early orthodontic treatment might minimize risk factors in patients with increased maxillary overjet.

### Primary dentition

Intrusion and avulsion of primary teeth may cause delayed eruption of permanent teeth, sometimes combined with crown or root malformations. Surgical exposure in combination with orthodontic extrusion is an option to save such teeth. Treatment is best performed in the early mixed dentition.

### Treatment planning

Early start of orthodontic treatment in patients with dental injuries and proclined upper incisors can reduce a large overjet. Headgear treatment, bite jumping devices or appliances with mini plate anchorage may be used. Follow-up treatment with a second phase later is often necessary. The risk of root resorption of the traumatized teeth during the second phase might be reduced.

Teeth with poor prognosis can sometimes be part of the orthodontic treatment to avoid alveolar bone loss during development of the jaws.

### Observation period prior to orthodontic treatment

- Mild injury (e.g. concussion, subluxation): 3 months
- Moderate or severe injury (e.g. extrusion, intrusion, lateral extrusion, replantation): 6 months
- Root-fractured teeth: 1 year
- Endodontically-treated teeth: Orthodontic treatment should be postponed until evidence of radiographic healing is seen

### Principles for orthodontic movement of traumatized teeth

Orthodontic treatment represents an extra hazard to the periodontium and pulp.

### *Endodontically-treated teeth*

There is no evidence of a greater risk of root resorption of root filled teeth than vital teeth.

### *Pulp canal obliteration*

There is no evidence of a greater risk of root resorption in teeth with pulp canal obliteration than vital teeth. There is however a risk of pulp necrosis.

## Root resorption during orthodontic treatment

Regular radiographic controls are necessary in order to check if any root resorption occurs or increases during treatment.

- The first control should be performed after 6 to 9 months after initiation of treatment
- Signs of root resorption indicates controls every 2 to 3 months
- A suspension of treatment for about 3 months might reduce the risk of further resorption if signs of resorption are seen.

### *Avulsion*

#### Space closure

Space closure is of prime importance in patients with maxillary arch length deficiency. Mesial movement of a lateral to substitute a missing central incisor is complex. Proper uprighting of the incisors, grinding and composite buildups are necessary. Crown form and inclination of the lateral incisors and canines must be favorable. The treatment seldom leads to optimal esthetic results. In severe trauma cases however even space closure after avulsion of two central incisors can be an option.

#### Space maintenance

The space can be maintained with removable or fixed appliances in growing children until prosthetic treatment is possible. The appliance should be constructed to avoid interference with eruption of permanent teeth.

Autotransplantation of premolars when the roots are developed to ¾ of full root length is an option.

## Fractures

### *Crown-root fractures*

A non-vital tooth can be extruded over a period of 3–4 weeks

- Surgical fibrotomy and gingival contouring are important to avoid relapse.
- After a retention period of 3 to 4 weeks, the tooth can be restored.

### *Root fractures*

Orthodontic movement of fractured teeth is related to the type of healing and location of the fracture.

- Root-fractured teeth healed with a calcified tissue can be moved without breaking the callus.
- Root-fractured teeth healed by interposition of connective tissue must be looked upon as teeth with short roots. Further shortening can be expected during orthodontic treatment.
- Teeth with fractures located in the apical third can normally be moved with good prognosis whereas teeth with fractures located in the middle third should be treated with extra care.

## Luxations

- Teeth with pulp necrosis and/or infection related root resorption should be treated endodontically prior to orthodontic treatment.
- Orthodontic movement of luxated teeth with initial signs of resorption should be carefully checked for further resorption. The first observation should be made after 6 to 9 months and thereafter at 3-month intervals.

## Intruded teeth

### *Immature teeth*

Intruded immature teeth may be left to erupt spontaneously if the intrusion is not too severe or slightly loosened with finger pressure.

- Orthodontic extrusion is indicated if the intrusion is severe or if the tooth has not begun erupting within two to three weeks.
- Impacted teeth with crown or root dilacerations can be moved into a normal position after surgical exposition if the damages are not too severe. Treatment is best performed in the mixed dentition stage.
- Crown and root malformed teeth can be used as space maintainers during growth and development of the jaws.

### *Mature teeth*

- The critical period for onset of external root resorption is 2 to 3 weeks.
- Intruded teeth must be repositioned within the first 3 weeks after injury.
- Repositioning can be accomplished either orthodontically or surgically.
- If the tooth is tightly locked in bone it shall be slightly luxated before orthodontic extrusion.

## Replanted teeth

### *Normal periodontal ligament*

Replanted teeth with a normal periodontal ligament can be moved with the same prognosis as uninjured teeth.

### *Ankylosis*

There is no treatment known for ankylosis.

- A treatment plan for the patient is of importance when the diagnosis has been established.
- Development of the infraocclusion is important to carefully follow-up.

### *Composite build-up of incisal level*

If the alveolar growth is finished or almost finished, only a minor infraocclusion can be expected and a composite build-up of the incisal edge is an option. A slight increase of the infraocclusion can however be expected.

### *Extraction*

Providing no adverse changes are seen, an ankylosed tooth can be retained until the crown breaks off, or is removed with forceps when most of the root substance has been replaced by bone.

### *Decoronation*

A technique called decoronation is recommended for extraction of ankylosed teeth in infraposition to avoid severe alveolar bone loss. The crown of the tooth is removed and the ankylosed root is left in the alveolus to be substituted by bone. In growing children, new marginal bone will then be formed coronal to the resorbing root.

## Retention

Retention follows the same principles as for non-traumatized teeth.

## Prognosis

Teeth with an intact periodontal ligament prior to orthodontic treatment can be moved with a prognosis comparable to that of non-traumatized teeth.

Teeth with shortened roots and a final root length less than the crown height (<9 mm) might have a tendency to increased tooth mobility.

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# 29

# Restoration of Traumatized Teeth with Resin Composites

U. Pallesen & J. W. V. van Dijken

The primary choice for initial restoration of a crown-fractured front tooth has long been resin composite material. The restoration can in most cases be performed immediately after injury if there is no sign of periodontal injury. The method's adhesive character is conservative to tooth structure and with minimal risk of pulpal complication. In addition, it offers an esthetic solution to the patient immediately after an injury, which may bring a little comfort in a sad situation.

The resin composite build-up is often changed or repaired a couple of times, before the tooth is restored with a porcelain or porcelain fused to metal crown, at a time when the pulp is out of danger for a more invasive preparation. In some cases, an endodontic treatment is still necessary. After crown therapy a gingival inflammation may occur due to the usual sub-gingival preparations. After some years in situ, the crowns may present an esthetic problem due to exposure of un-esthetic crown-margins. The invasive permanent crown restorations are therefore often not successful on a long-term scale. On the other hand, a conservative direct restoration of an extensively fractured incisor crown with resin composite may be an exceedingly demanding procedure, involving esthetic acceptability, function and biological aspects and require significant skills, which may influence durability.

When are we ready to consider the non-invasive resin composite crown build-up as a permanent restoration? Has its durability improved? Do today's materials and techniques result in a better prognosis? Where are the problems? In this chapter these matters will be discussed with focus on factors in clinical procedures, which can influence the longevity of the restoration.

## Long-term prognosis of resin composite restorations

Traumatic injuries to anterior teeth in children and adolescents are a common problem. About 50% of children are exposed to dental trauma before 15 years of age and the majority of these injuries imply crown fractures (1). Follow-up studies are very scarce. One of the earliest studies of Class IV restorations was a 5-year follow-up of children with crown-fractured permanent incisors reported by Ulvestad in 1978 (2). Three conventional resin composites were used following enamel etching. Among the 253 composite restorations, fracture of the composites was observed in 9–14% and fracture of enamel in 2–3%. A high frequency of marginal discoloration was reported (17–19%), but no case of secondary caries. Abrasion was seen in 52–79%. Robertson et al. 1997 (3) evaluated crown fractures restored with resin composite materials, which were carried out during a 15-year period. In this study all restorations had been replaced at least once and 19% of the restorations had been replaced 10 times or more, in fact most of the restorations had been in service for only 2–4 years. In a retrospective cohort study published by Borssén and Holm 2000 (4), 44% of the 16-year-old patients where crown fractures were restored with composite, had had the resin composite revised one or more times.

In 2004, Spinas reported a longitudinally clinical study, using newer resin composites to restore traumatically injured teeth (5). Here 70 restorations were followed during 7 years with modified USPHS criteria. A total-etch technique and third or fourth generation bonding systems were used. In the

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period between 3–5 years, all the restorations belonging to all the injury types, needed a complete replacement. The number of replaced restorations during the follow-up were at 1 year: 8.5%, 2 years: 26%, 3 years; 47%, 4 years: 71%, 5 years: 87%, 6 years: 96% and after 7 years 100%. The main reasons for replacement were: loss of the restoration, loss of marginal integrity, discoloration caused by the loss of pulp vitality. At the end of the study 48% of the replaced restorations had been replaced at least one more time.

Since only few publications on longevity of crown build-ups are available, the long-term prognosis may also be based on the experience of caries lesions in anterior teeth or replacement of Class III restorations because of incisal fracture, restored with Class IV restorations. The class of anterior cavity preparation substantially affects the resin composite survival (6–8). Unfortunately, most anterior resin studies do not distinguish between cavity classes and therefore limited clinical evidence has been published concerning the longevity of the more difficult Class IV resin composite restoration. In 1990 Tyas (9) restored 102 Class IV cavities with microfilled and hybrid composite materials. A cumulative failure rate of 26% (5–42%) was observed after 3 years. The 2 microfilled resin composite materials showed the highest failure rates (40% and 42%) compared to the hybrid materials (5% and 9%). Smales (10) reported the survival of a conventional anterior resin composite with a low viscosity enamel bonding resin. The median survival time for Class III restorations was 9.8 years and 4.1 years for Class IV. He attributed the losses to increased wear and occlusal stresses. Smales & Hawthorne (11) examined in a retrospective study the survival and cost-effectiveness of restorations in various classes of cavity preparation in permanent teeth placed before 1980. For Class III resin composite restorations 72% survived for 10 years and for Class IV 48% survived 15 years. Browning & Dennison (12) investigated the modes by which Class IV restorations failed. The main reason for replacement of Class IV was secondary caries (19%), marginal discoloration (11%) fracture of tooth (10%) and fracture of composite (47%) and 50% had failed by 5 years. The high failure rate of Class IV restorations was also shown by Miller et al. (13), however, based on very few restorations. Burke et al. (14) examined Class IV resin composite restorations and reported a mean longevity of 3.9 years.

Adhesive techniques and materials may influence longevity. Recently, results were reported from a 14-year follow-up study of 85 Class IV hybrid resin composite (RC) (43: Pekafil), polyacid modified resin composites (PMRC) (24: D Dyract, H Hytac) and resin-modified glass ionomer cements (RMGIC) (18: F Fuji II LC, P Photac Fil) restorations placed in ongoing longitudinal follow-ups in 45 patients (mean age 54.5 yrs) (15). The restorations were evaluated during the 14 years by slightly modified USPHS criteria at annual recalls especially for their fracture behaviour. For all restorations, 36.5% fractured, with a Kaplan-Meier estimate of 8.8 yrs. 11 RC (25.6%; K-M 9.9 yrs), 13 PMRC (54.2%; D 66.6%; H 50.0%; K M 7.5 yrs) and 7 RMGIC (36.5%; F 22.2%, P 71.4%; K-M 6.9 yrs). Significant differences were seen between RC and PMRC ($p = 0.043$). A significant higher fracture rate was observed in the maxillary lateral incisors compared to central incisors. No significant differences were observed between male and female patients. Restorations in bruxing patients showed significantly higher frequency of fractures than in non-bruxing patients. Longevity of the replaced failed Class IV restorations was for RC: mean 4.5 yrs, PMRC: mean 4.3 yrs, and RMGIC: mean 3.3 yrs.

A recent meta-analysis of direct anterior restorations included 21 clinical studies. However, most of the studies were carried out between 1980 and 2000 and there were only 6 studies for Class IV restorations and diastema closure (16). The median replacement frequency of resin composites was twice for Class IV than for Class III after 10 years (10% versus 5%). Class IV restorations with microfilled resin composites showed significantly more chippings and fractures than those with hybrid and macrofilled resin composites. The reasons for replacement of Class IV restorations were predominantly bulk fractures while the frequency of secondary caries was low. This was confirmed in a recent review indicating that anterior restorations are likely more prone to replacement due to trauma fractures, esthetic demands and loss of retention (17). This review did not separate results for Class IV restorations.

In conclusion, findings of the few present studies on longevity of direct crown build-ups of fractured teeth have led to the concept that older resin composites were considered as semi-permanent restorations. However, a few studies of newer resin composite systems have shown a potential for improved longevity with Class IV restorations (Fig. 29.1 A–D).

The most frequent reasons for failures appear to be fractures of the resin composite, and for older resin composite systems also marginal leakage and bulk discoloration (Fig. 29.2 A–D), which presents the profession with a great challenge. Many of the old studies used old materials and non-optimal dentin bonding systems. Over the years, resin composites with different formulations have been introduced. However, resin composites should not be considered as a single product, but as part of a complete system. It is reasonable to believe that further improvement in the retention and esthetics of Class IV restorations can be achieved by improvements in preparation, bonding and light curing techniques as well as in filling, finishing and polishing procedures (see below).

## Material considerations

### Bond to enamel

In most cases traditional mechanical methods of retaining dental restorative materials required removal of sound tissue. These techniques have now been replaced largely with adhesive techniques. Buonocore's pioneering work, noticing the industrial use of phosphoric acid to improve adhesion of paints on cars, led to major changes in operative dentistry.

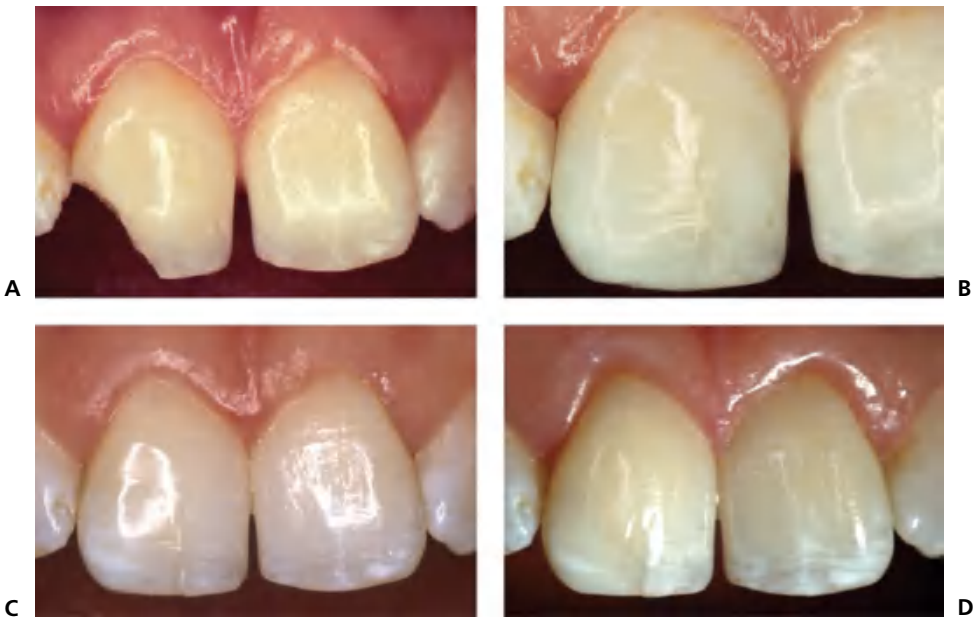


**Fig. 29.1** Initial result and durability of a resin composite crown build-up. A. The fractured incisor before restoration. B. The restored tooth at baseline. Morphology and white spots of the synergist tooth has been imitated. Horizontal lines in the surface structure mask the preparation line. C. Acceptable result after 5 years. D. Acceptable result after 10 years with no re-treatment between baseline and 10 years.

**Fig. 29.2 Frequent reasons for failures of resin composite crown build ups of fractured teeth**
A. Marginal discolouration, lack of colour-match and insufficient morphology. B. Marginal leakage has led to secondary caries. C. Incorrect morphology of restorations in both central incisors is the reason for an unacceptable esthetic result. D. After shaping the old restorations in C to a better morphology replacement could be postponed.

Large preparations and extension for prevention have been gradually replaced by minimally invasive techniques. For resin-based materials the basic mechanism of bonding to enamel is a micromechanical bond. It is essentially a replacement of minerals by resin monomers that after setting becomes interlocked in the irregularities and porosities created by conditioning. Adhesive restorations are tooth-substance preserving and prevent microleakage along the cavity walls, which may improve the longevity of the restoration. The strength and durability of the adhesive bond depends on many factors such as: the physico-chemical properties of the adhesive and adherent adhesive technique applied, polymerization stresses developed during curing and the continuous stress in the oral environment.

Adhesive dentistry began with the first commercial systems in the mid-1960s, followed in the early 1970s with the introduction of the acid-etch technique in the clinic. Since then there has been a continuous development and improvement in bonding agents. Effective bonding to enamel was achieved early and was proved to be durable and reliable in the clinic. Enamel etching transforms the smooth enamel surface into an irregular surface with a surface energy twice that of unetched enamel (about 72 dynes/cm). An unfilled, low viscosity resin wets the high-energy surface and is drawn into the micro porosities by capillary attraction. Acid etching with phosphoric acid removes about 10 μm from the surface and creates irregularities from 5–50 μm deep. Different enamel etching patterns can be observed after etching

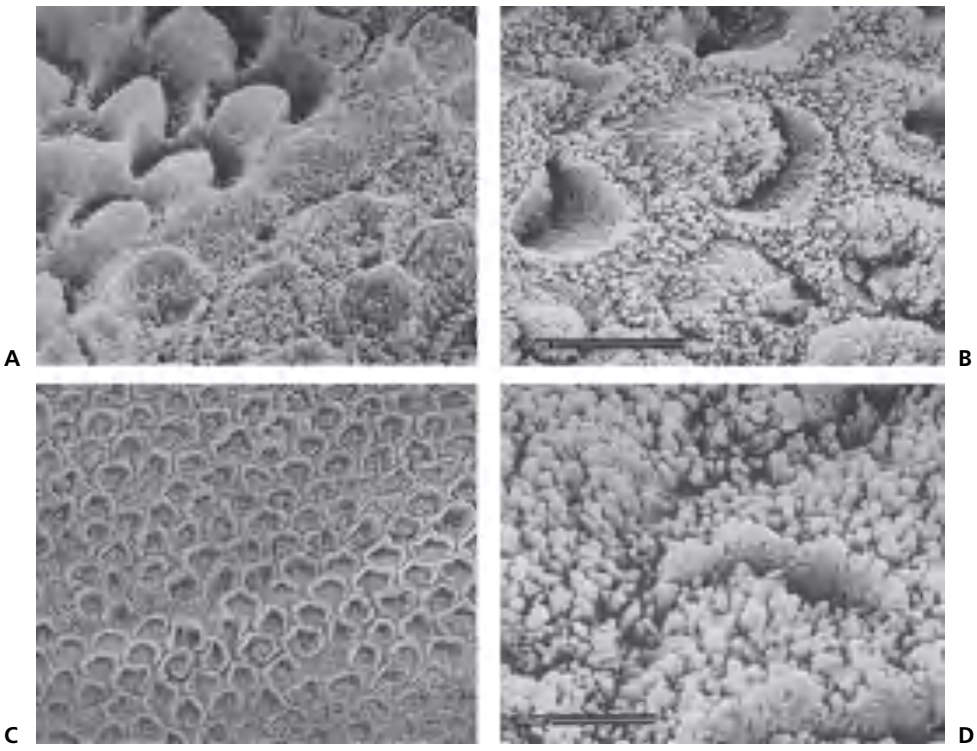


**Fig. 29.3** Un-prepared (non-ground) buccal surface of a premolar acid etched with 37% phosphoric acid for 15s. Different etch patterns can be observed. A. Two etch patterns with different degrees of demineralization between rod and interrod enamel. Original magnification 3600x. B. Etch pattern with high dissolution between rod and interrod enamel. Observe increase of surface area. Original magnification 7200x. C. Etch pattern with highest demineralisation in the rod enamel. Original magnification 1200x. D. Higher magnification of C showing the various degrees of peripheral dissolution of the apatite crystals. Original magnification 12000x.

(Fig. 29.3 A–D). Generally, a 32–40% phosphoric acid etching time of 10–15 seconds for instrumented enamel is used. Rinsing times of 10–20 seconds have been recommended when the acid-etch technique was introduced in order to achieve the most receptive enamel surface for bonding, though without clinical evidence. Recent long-term studies of posterior resin composite restorations employing rather short rinsing times pointed out that rinsing times can be shortened (15, 18–20).

## Bond to dentin

Adhesion to dentin has been more difficult to obtain compared to bonding to enamel. Amphiphilic adhesive systems have produced bond strength to dentin approaching that of enamel bonding and have achieved higher levels of clinical success. The early selective enamel etching was replaced in the 1990s by the total-etch technique with simultaneous conditioning and priming of the enamel and dentin. Dentin bonding systems have been classified chronologically in generations. In the first and second generations enamel was acid etched separately, while the dentin adhesive mechanism to dentin involved an ionic interaction to the positively charged Ca-ions. Modest bond strength and high clinical failure rates were observed. In the early-to-mid 1990s many dentin adhesives in the third and fourth generations were based on the dentin etching technique. Most of these 3-step adhesive systems required a first step of dentin conditioning to decalcify the intertubular and peritubular dentin. In the second step a primer with amphiphilic monomers, displaces the dentinal liquid and infiltrates the collagen network of the demineralized dentin. The third step with adhesive monomers enhances the bond with the hydrophobic resin composite. The production of a high micromechanical bond to the dentin is ensured by formation of the so-called hybrid layer. The degree of monomer penetration is enhanced by water chasing solvents in the primer step and the collagen fibrils have to be coated optimally by the primer monomers in order to obtain a high bond. The multiple-step procedure is technique sensitive and the clinical success may depend on the handling of the system (21).

In adhesive dentistry developments include the simplification of the multi-step systems, to reduce their technique sensitivity during clinical handling in the fifth and six generation systems. Several simplified, one-bottle and self-etching primer systems have recently been introduced to decrease the technique sensitivity and allergic potential of the 3-step systems. In the one-bottle or two-step systems, primer and adhesive are combined in one step. Commonly, this step was applied twice, but in recently marketed systems a single application is recommended. In the self-etching primer systems unsaturated, potentially polymerizable organic acids or acidic monomers are incorporated and etching and priming of the dentin occur simultaneously. They are based on infiltration and modification of the smear layer by acidic monomers or by dissolving of the smear layer and demineralizing of the underlying outer layer of the dentin followed by infiltrating the smear layer covered dentin with acidic monomers of the primer (22). In one type of self-etching primer systems an adhesive is applied on top, while in other systems, the "all-in-one" systems, all 3 steps are applied simultaneously. Recent developments are selective bonding and universal adhesive systems as described above. Only short term clinical evidence is available for these techniques.

In conclusion, different approaches are marketed today: 1) etching with a phosphoric acid of enamel/dentin is

followed by application of an amphiphilic primer with or without adhesive, 2) self-etch or self-adhesive bonding systems are applied without prior phosphoric acid etch of the tooth tissues, 3) selective bonding systems, combining a phosphoric acid etch of enamel followed by application of self-etch priming and/or adhesive in the whole cavity and 4) universal bonding systems, which can be used according to the total-etch or self-etch approach. All use recommended light curing procedures with appropriate curing device. After these steps the final restoration can be performed with a resin composite.

## Glass ionomer bond

Parallel to the progress made with resin-based adhesives, glass ionomer cement technology has been modified and improved. Glass ionomer-based materials have a chemical bond to tooth tissues based on their specific chemical formula. Developments during the 1990s combined glass ionomer and resin composite to new adhesive materials with mixed characteristics. Improved modifications, the resin-modified glass ionomer cements (RMGIC) were introduced in 1988. Addition of light curing resin components led to a higher resistance to early moisture contact and desiccation and higher mechanical characteristics. RMGIC were used first as bases or restoratives and in 1995 a modern RMGIC was developed as dentin-enamel adhesive. After pretreatment of the cavity with a weak polyalkenoic acid, self-adhesion of the adhesive is obtained by both a micromechanical interlocking by a submicron hybrid layer (0.5–1μm) and a chemical bond through ionic bonds between the carboxyl groups of the glass ionomer and calcium of hydroxyapatite that remains around the collagen. Good clinical durability has been shown in a long-term evaluation (23). Kemp-Scholte & Davidsson (24) showed that a material with enhanced flow and reduced elastic modulus may function as a stress-absorbing layer and improve marginal sealing. Resin composites have a higher Youngs modulus of elasticity which result in a relative high remaining contraction stress compared to polyacid modified resin composites (PMRC) and RMGIC. Recently, it was shown that glass ionomer derivates and compomers showed high fracture rates in Class IV cavities (15). Unfortunately, glass ionomer materials do not have the mechanical strength to withstand the sheer forces in Class IV cavities (15). Therefore, glass ionomer restorative materials are not commonly used in anterior restorations when esthetics is a major consideration. They are indicated in cavities with deep cervical located margins or high caries risk patients.

## Resin composite materials

Historically, restoration of fractured anterior teeth has evolved from the use of stainless steel crowns to the current procedures of bonded esthetic resin-based materials. The commercial introduction of resin composites in the late 1960s led to their widespread use in the restoration of Class IV cavities. Retention of the resin restorations relied for the most part on the effectiveness of the bonding systems utilized. Recommended preparation techniques have varied over time such as butt joint margins, feathered edge margins, bevels, threaded pin and retention slot preparations, long bevels and chamfer preparation. Retentive pins improve the retention of Class IV restorations only to a small extent (10%) but faces risks such as perforation of the pulp chamber, inflammatory responses of the pulp, dentin cracks, perforation of the root surface and increase its fracture susceptibility (25, 26). Semi-permanent restoration of an incisor with a resin composite material, following acid etching of the enamel became a frequently used method during the seventies (2). Resin composites used at that time in anterior build-ups lacked color stability, polishability and physical properties. These limitations could not compete with the esthetics of the laboratory produced porcelain or porcelain-fused-to-metal crowns. Patients demand superior esthetics from anterior restorations that have to simulate the natural tooth in color, texture and translucency. The materials must have adequate strength, good wear characteristics, good marginal adaptation and sealing, color stability, insolubility and biocompatibility. Resin composites are currently the direct restorative materials that best fulfil these requirements.

A resin composite contains four components: a polymer matrix, filler particles, a silan coupling agent and an initiator system. Newer formulations of hybrid resin composites with improved particle size distribution have imparted additional strength and polishability, allowing the materials to be used in rather high stress bearing areas. Varying degrees of opacity required for the different areas of the tooth are available. Undesirable characteristics of resin composites are their volumetric shrinkage and coefficient of thermal expansion. Shrinkage stress during the post-gel phase may cause crack and marginal gap formation between the resin composite and the walls of the preparation with the weakest bonds. Gap formation may result in postoperative sensitivity, marginal leakage, marginal discoloration and secondary caries. The size of shrinkage stress formation on the bonding surface may in part depend on the configuration of the cavity. The higher the number of bonded surfaces in the cavity, the higher the stress formation on the bonded margins. Occlusal Class I cavities show the highest configuration-factor (27), while the Class IV cavity with more unbounded surfaces has a more favourable configuration. However, the high failure frequency expected in Class I cavities due to high polymerization stress was not observed in long-term evaluations (18). It was concluded that factors other than the C-factor of the cavity are probably more important for the longevity of the restoration.

The majority of resin composites marketed during the last 25 years have been hybrid resin composites. The first-generation hybrid materials contained a blend of submicron (0.04 μm microfiller agglomerates) and small filler particles (1–4 μm), allowing a higher level of filler loading resulting in improved physical properties compared to the earlier traditional resin composites. Hybrid resin composites were for many years the materials of choice for the whole restoration,

but were also used as the bulk/dentin part of Class III and IV restorations. Microfilled resin composites containing submicron inorganic fillers in low proportions, and therefore not as strong as the hybrid composites, could be used as the enamel veneering part of these anterior restorations because of their better polishability and translucency. Their abrasion resistance was however unfortunately low.

Many resin composites today belong to the nano-composites, which contain very small filler particles in the range of 0.1–100 nm. Nano-fillers are now included in almost all new resin composites materials and have replaced conventional hybrid materials. The term "nano-composite" includes two types of materials nano-hybrid and nano-filled resin composites. The newer nano-composite materials show increased filler load and improved mechanical properties compared to earlier resin composites. Claimed advantages are: increased polishability, good lasting surface characteristics, improved translucency, less polymerization shrinkage and higher wear resistance. In several randomized short and long-term studies acceptable performance has been observed for several nano-hybrid and nano-filler posterior resin composite restorations, which had been placed with recent functional adhesive systems. However, no significant differences in durability were observed between nano-composites and conventional hybrid resin composites (19, 28–32). No Class IV clinical evidence has been reported for these modern resin composite materials that have shown improved esthetic properties but not improved fracture toughness.

## Curing of materials

Autocured resin composites were the first resin composite materials used for treatment of anterior cavities. A major drawback was their bulk discoloration by time. Light cured resin composites therefore became increasingly popular since their introduction in the mid-1970s, making dental restorations more conservative and esthetic. The degree of polymerization in cross linked resin-based systems plays a potentially significant role in determining the ultimate physical and mechanical properties of the material. Inadequate polymerization results in inferior biological and physico-mechanical properties, such as poor resistance to wear, increased rates of water absorption and poor color stability (33–36). The degree of cure is affected by a lot of factors such as: the power density of the curing unit, the exposure time, the resin shade and the filler size (37). As light passes through the bulk of the restoration, its intensity decreases greatly, thus decreasing the curing efficacy and limiting the depth of cure (38). Power density is fundamental for an adequate depth of cure.

The use of high intensity light sources to improve resin composite properties was introduced in early 2000. These lights may provide higher degrees of conversion and superior mechanical and physical properties, but on the other hand also produce higher contraction strain rates during the polymerization of resin composites (39). The most widely used curing units during the 1990s and early 2000 were quartz tungsten halogen (QTH) units. When connected to an electric current a tungsten filament glows and produces a very powerful light (40). White light is produced, which is then filtered to the range of blue light (400–500 nm). However, only part of the emitted spectrum of light is effective for activating the photo initiators. A QTH curing unit should deliver a power density of 350–400 mW/cm$^2$ in order to adequately cure. Heat generation is a major disadvantage of QTH units and increases with increasing radiation time (36, 41, 42). Other drawbacks are limited lifetime of the bulb and degradation of the reflector and filter over time. It was generally accepted that 2 mm increments of traditional hybrid and microfilled resin composite should be placed combined with a 40–60 s light cure (43, 44). Most newer curing units are more powerful and may offer higher depth of cure and/or reduced exposure time. Not least with the recently introduced bulk-fill resin composites where a curing depth of 4–5 mm can be obtained. Curing features like the soft-start programs, e.g. two-step curing, and pulse-delay curing were recommended during the early 2000. The aim was to improve marginal adaptation by prolonging the phase during which the resin composite may flow and thus compensate for polymerization shrinkage and stress. However, no clinical evidence of improved longevity has been shown for these curing techniques compared to conventional continuous curing (39, 45–47).

During the last years, light emitting diode (LED) technology has been replacing QTH curing in operative dentistry (48). In these devices, junctions of doped gallium nitride semiconductors (p-n junctions) are subject to an electric current and generate blue light. LED's curing units have a narrower wavelength spectrum compared to QTH curing units and require no filters. In contrast to QTH units, 100% of the light emitted by the first generations blue LEDs lay within the spectrum that can be used to cure most resin composites (450–490 nm), i.e., resin composites initiated by camphorquinone (42). However, several recently introduced units have additional wave length for other catalyst/activator systems. It can be concluded that LED curing units have significant advantages compared with the QTH curing units. The diodes have a lifetime of more than 10.000 hours compared with the 40–100 hrs effective lifetime of QTH bulbs. There is no degrading of bulb, reflector or filter over time resulting in reduction of curing effectiveness. They produce relatively little heat and for most units no fan is needed. However, whereas the first LED units were characterized by relatively low power density, more recently marketed LED units display higher power density and/or wider wavelength spectrum resulting in improved curing efficiency. Furthermore, a fan has become necessary for some LED curing units (49, 50).

# Clinical implications

The need for a high level of esthetics is obvious when an anterior fractured tooth is restored. This, together with previously mentioned problems of too early replacements of

Class IV resin composite restorations, present the profession with a great challenge. Quality of restoration at baseline highly influences longevity. The correct choice of material, bonding system and curing method has already been mentioned as important factors, but also other aspects in the clinical procedure such as awareness of tooth characteristics, preparation techniques, shade selection, filling and finish/polish techniques must be optimized. The better longevity, the closer to the goal, where this non-invasive restoration technique can be considered as a permanent restoration (Fig. 29.1 A–D).

## Critical points in the restorative procedure

### Identifying tooth characteristics

If a fractured tooth needs functional or esthetic restoration, it is necessary to identify characteristics of the remaining tooth structure and of the other teeth (51). Morphology, color and surface structure can often be directly copied from the synergist tooth.

*Young teeth* have intact enamel (no wear) with remaining surface texture (many reflexes) and often the enamel has small spots of white (hypo-mineralized areas, opaque/white). Dentin is unaltered (open dentinal tubules) and the organic substance in the young dentin reflects light and gives a more opaque (white) appearance than the older dentin. Developmental lobes at the surface as well as mamelons in the incisal area are intact.

*Adult teeth* may have beginning wear of the enamel and less surface structure (less reflexes). The white enamel spots are disappearing (less opacity, less white) and the incisal edge may start to wear (less pronounced mamelons). The developmental lobe tops may be exposed in the incisal edge area. Dentin has continued mineralization, which leads to less opacity (less white).

*Old teeth* may have improved wear of enamel with no remaining surface structure (few reflexes) and dentin may be exposed (discoloration). Developmental lobes have usually disappeared and wear of the enamel in the incisal area have removed the translucent area (mamelons disappear). The dentin becomes sclerotic (more transparent and dark) due to the formation of tertiary dentin. Thin enamel and increased mineral content in enamel and especially in dentin lead to a higher translucency and make teeth look darker than in adult or young teeth.

*Endodontic treated teeth* may have grey, blue or yellow discoloration and therefore need internal bleaching before restoration (see Chapter 40). After bleaching the resin composite crown build-up, procedure should be postponed 1–2 weeks, due to left oxygen in the dentin, which can inhibit optimal polymerization of resin materials. For the pulpal cavity part of the crown build-up a high-contrast white resin material is recommended to facilitate later access to the inner part of the tooth in case of need for retreatment of the internal bleaching (52).

### Preparation techniques

To prevent loss of the restoration, marginal chipping and marginal discoloration, preparation technique should be carefully considered. Also, the esthetic appearance is influenced by the preparation technique. First unsupported enamel must be removed, and a margin defined. In the interproximal and palatal areas where esthetic is not an option, the best cavity angle for the resin material is 90 degrees. This may be provided either by leaving the fracture facet or preparing a flat margin. In the vestibular area a bevel or a chamfer of at least 1½ mm width half way through the enamel is applied. A bevel compared to a chamfer is conservative with regards to removal of tooth structure and makes transition from composite to natural enamel less visible. On the other hand, the fragile margins of the restoring material frequently deteriorate over the years and may lead to marginal chipping and discoloration. A chamfer is more invasive and provides more bulk to the restoring material at the margins but may fail to blend in the restoration with the color of the tooth. To overcome this, the stair-step or wave preparation technique has been recommended in which the preparation follows the anatomical contour of the human incisors (Fig. 29.4). Another advantage of the chamfer compared to the bevel is the increased enamel surface area providing a better bond to the tooth surface (26). Most preparation types will provide sufficient retention for even large crown build-ups. To prevent small fractures of unsupported enamel prisms during contraction of the resin composite material, the margins are smoothened using silicone points under water-cooling. This may prevent later marginal discolorations.

### Shade selection

In contrast to old resin composite materials, the new generations have shown acceptable color stability and smoother surface characteristics. Shade selection plays an important role for the esthetic outcome of an anterior restoration and requires knowledge of colors and careful analysis of the remaining tooth structure and of the adjacent teeth. The color-layering technique used by the dental technician for indirect esthetic restorations may be applied. Resin composites with different hues, chroma and values have been used for a long time, but properties such as high/low translucencies, opalescence and fluorescence – known from ceramic materials – have lately been added. This chapter will not include information of shade selection at a high level, and only a few definitions and recommendations will be mentioned (53–55).

*Hue* is the basic shade of the tooth and can be assigned to the dentin. Hue is often referred to as A, B, C or D in the Vita shade-guide.

*Chroma* is a further refinement of the hue that defines the quality of color, strong or weak, or saturation/intensity of the color. Chroma is often referred to as 1, 2, 3 or 4 in the Vita shade-guide. Hue and chroma may be evaluated in the cervical area of a canine where the enamel is thin and the dentin thick. The hue is usually uniform in a person's teeth, whereas chroma may vary from tooth to tooth.

*Value* is the degree of brightness or darkness in the tooth or the amount of white reflected from the tooth. Value is assigned to the enamel, and although the enamel itself is colorless it projects the underlying color in the dentin. Value is evaluated at the incisal third of the tooth.

*Translucency/opacity* in a tooth expresses how much light is able to pass through the tooth-substance. Part of the light is absorbed, part is diffused, and part is transmitted.

*Opalescence* is the enamel's capacity to distinguish wavelength in the light. The short wavelengths are reflected (appear blue) and the long wavelengths pass through the enamel and appear orange in transmitted light. When translucent enough, modern enamel composite materials exhibit opalescence-like effects, which are essential in order to be able to reproduce the bluish reflections of the incisal third of a young tooth.

*Fluorescence* in natural teeth will reflect blue light, when exposed to incident UV-light. The natural fluorescence of dental tissues was recognized early as critical for the development of highly esthetic ceramics and is now also considered an important issue for composite restorations. Fluorescence in dentin resin materials gives the restoration "vitality" and contributes to a correct value. When a non-fluorescent material is used, it tends to have a grayish appearance due to the not reflected small amounts of UV-light included daylight. If seen in UV-light e.g. in a night club, a restoration without fluorescence will appear as a "black hole".

*Refractive index* also characterizes an optical property of a material. This property can be made visible at the boundary between two materials where light travels through one material to another. If a resin composite material does not have the same (or close to) refractive index as enamel, light will be refracted differently from the two materials, which may influence the esthetic outcome at the margins of the restoration.

*Identifying tooth color* is of significant importance, when decision of shades for a crown build-up is made (51–55). Natural tooth architecture affects color by the different layers, by its composition and by the quality of dentin and enamel as mentioned previously. Dentin is more opaque than enamel, and enamel translucency is high. At the incisal edge and in the periphery of a crown the enamel is thick and without underlying dentin, which makes these areas translucent (appearing gray or blue). In the cervical area enamel is thin and supported by dentin, which makes the area more opaque (appearing yellow/brown). Distinct kinds of mineralization disturbances in enamel may give special characteristics to the tooth. Exposure of dentin may lead to discoloration due to penetration of dyes in the dentinal tubules. Each tooth has its own characteristics to be mimicked in the crown build-up.

*Shade guides* for resin composite systems are often used to decide color of the restorative materials. However, commercial shade guides for high esthetic crown build-ups are often of limited value. Most shade guides are made of another material than the composite resin material. Tabs are of unrealistic specimen thickness and only few allow the overlap of enamel and dentin tabs (56, 57). Therefore, it can be recommended to make a diagnostic mock-up (without acid etching), where the different selected colors and translucencies are applied in the right thickness and place and with the natural background (the dark oral cavity or the tooth structure). Evaluating colors of the wet mock-up after light curing will also give an impression of the color change from before and after curing of the material.

Decision of colors and form should always be performed before isolation with rubber dam or cotton rolls, when the tooth is still moistened with saliva and in its natural surroundings. During treatment the neighbor teeth often dry out and will appear too white after treatment compared to the restored tooth. This should be informed to the patient before treatment.

## Choice of resin composite material

For the esthetic crown build-ups, enamel materials of different translucencies/values, dentin materials of different hue/chromas and characterizer materials (white, yellow etc.) are needed (58). They may be of different viscosities which are often related to the practitioner's preferences. In order to reduce the number of different colors some manufacturers only provide A hues in several chromas, whereas others provide A, B, C and D hues in only few chromas. The esthetic outcome of composite resin materials is not only influenced by hue, chroma and value. The optical properties are also influenced by the nature and size of the fillers, the material's thickness, the fluorescence of the dentin materials and the degree of translucency, opalescence and refractive index of the enamel materials (56). Knowledge of the optical properties of the resin composite used is necessary for the correct application of the different masses available in each system in order to achieve natural looking restorations. Consequently, continual practice with the material of choice, constructing various samples and applying different layering techniques is the path to success.

## Anatomical layering technique

In addition to the mentioned knowledge of optimal properties of the material in use, information of tooth morphology, colors, translucencies and special characteristics from the remaining tooth structure and the adjacent teeth is also important when a fractured front tooth is restored with a direct resin composite build-up.

The anatomical layering technique may be preferred in order to control these different elements (Fig. 29.4 A–N) (59–61). By this method the restoration is built up with 2–3 layers of dentin material (opaque) of the same hue, but with different chroma (highest saturation of the first palatal layer) and a facial layer of enamel material (translucent). The preparation line may be masked by partly covering the bevel/champher with the last dentin layer, but without extending it to the prepared margin. Special effects in the restoration such as mottled enamel and white or yellow spots can be added by characterizers (tints) between the last dentin and

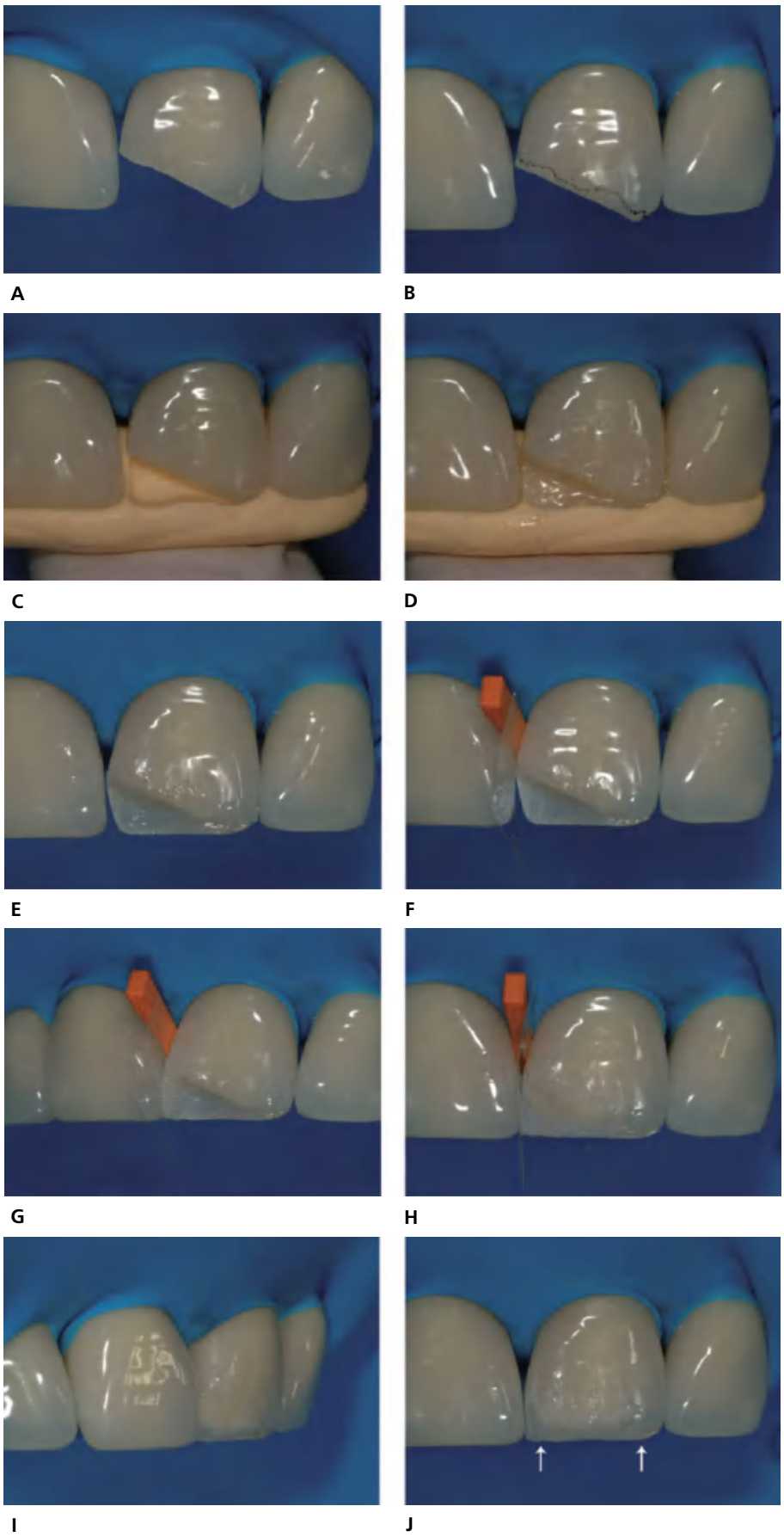


**Fig. 29.4 Anatomical layering technique for restoration of an incisor with incisal translucency**
A. The crown-fractured incisor. B. A 1–1.5 mm wide wave-shaped bevel preparation has been made using a flame shaped diamond bur. Here visualized by a pencil line. C. An impression of the old crown build-up or of an indirect wax-up/direct composite mock-up can provide a mould for the lingual and incisal areas. Control of the layering technique is hereby made easier. D. After bonding procedures the palatinal part is build up of a thin layer of enamel-material (transparent). The silicone mould is used to form the palatinal surface in the right place. Care should be taken in adapting the material to the enamel in the palatinal part of the tooth. Colour of the enamel has been selected before treatment by using a shade guide in the incisal area of the synergist tooth. E. The first enamel layer in place. F. A matrix fixed by wedge helps to control form in the mesial area. G. The mesial enamel is built up of enamel-material. H. The first layer of dentin-material (opaque) is placed. The colour of this layer should have the same hue but a higher chroma than the second dentin layer. Here an A3 has been used. I. The second dentin layer should be conformed with the facial surface and leave space for the enamel layer. The mamelons are shaped, and for masking purpose the bevel may be partly covered. Selection of colour for this second/last dentin layer has been performed in the gingival area of an upper canine, where the enamel is thin and the dentin saturated. Here an A1 has been used. J. To imitate translucency incisally in the mesial and distal areas and below/between the mamelons a translucent blue material is added here (arrows). K. The incisal halo effect is made by amber/yellow coloured material closest to the incisal edge (arrow). L. Finally the whole facial surface is covered with a thin layer of enamel-material. The facial morphology should only be slightly overextended, in order to facilitate finishing. M. Incisal aspect before finish. The silicone mould has helped placing the incisal edge in the right position. N. Final result after polish. Diamonds burs and abrasive disks have been used for shaping the morphology and fine disks and rubber polishers for the polish. The finial gloss is obtained by polishing paste applied with a brush or a wheel. Notice the important reflexes placed as in the neighbour teeth. The halo and the mesial and distal translucencies are visible in the incisal area.

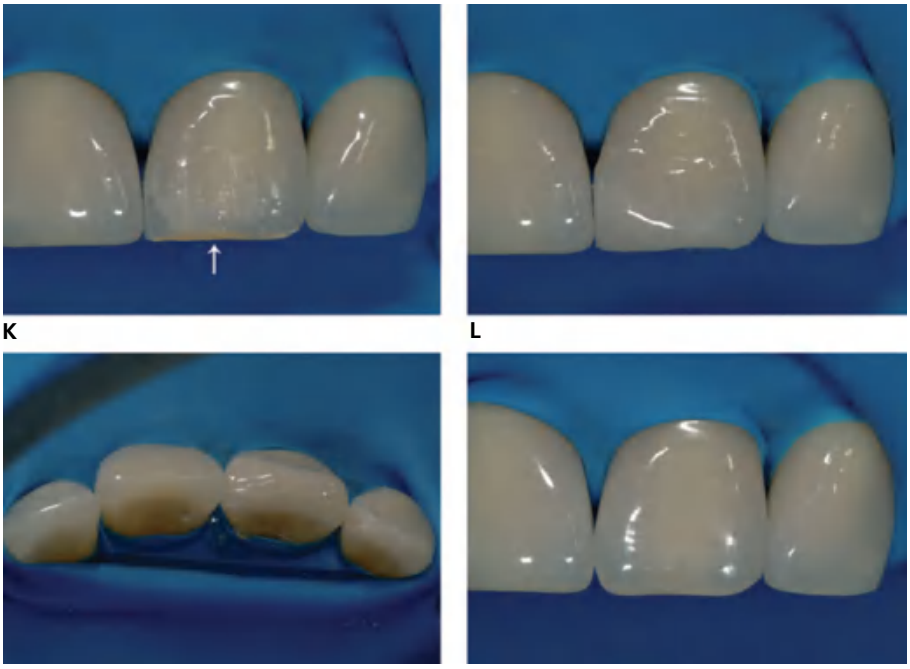


**Fig. 29.4** (*Continued*)

the enamel layer. Mamelons in the incisal area are built by the last dentin color with transparent/opalescent enamel material in between. Halo effect can be added with white or yellow characterizer at the incisal edge before the final surface enamel layer. If high translucency is needed in the incisal and approximal areas – typically for young people – these areas may be built up of only enamel material.

Each resin composite system has its own color-system with different intensities of hue, chroma, value, translucency etc. influencing the layering thickness and the characterization of the restoration and it is necessary to learn the possibilities with the system in use.

Different insertion techniques can be used to obtain the correct morphology and to control the layering technique (60–63):

- An impression of an old crown build-up to be replaced or of an indirect wax-up/direct composite mock-up can provide an excellent matrix for the lingual, proximal and incisal areas. Control of the layering technique is hereby made possible (Fig. 29.4 C, D).
- A celluloid crown matrix (Odus Pella® Crown) with a facial window can be an alternative to the impression technique, when a fractured tooth needs restoration.
- A finger-hold matrix band may also give support to the first lingual layer. However, the exact position of the lingual surface may be difficult to control by this method. Due to its allergic potential the uncured resin material should never be touched by fingers. Monomers from resin materials also penetrate gloves within a short time (64–67).
- A crown matrix (Odus Pella® Crown or CoForm Angulus®) may also provide possibilities for the layering technique (Fig. 29.5 A–L). The facial part of the matrix can be filled with enamel material followed by 1–2 dentin colors. This method, however, makes control of the layers more difficult.

## Finish and polish

Every single step in direct restorations is important and clinicians often devote too little time to consider morphology and surface of the restoration. An optimal finish and polish of the restoration improves fracture resistance, prevents periodontal problems and improves esthetics. If morphology of the restoration is not in accordance with tooth anatomy after placement, finish and polish may be time-consuming. Time spent during the modeling procedure before light curing will be valuable during the finishing procedure.

### Occlusion/articulation control

As previously mentioned, clinical studies have shown that fracture is one of the main reasons for early failure of Class IV restorations. This may be caused by insufficient morphology (too thin) and adjustment of occlusion and articulation. Although current resin composite materials have improved mechanical properties, inadvertently left supracontacts during eccentric movements may lead to cohesive fracture of the composite material. Therefore, care should be taken to provide light group contact in centric, protrusive and protrusive lateral positions if contact is necessary.

### Finishing in general

Diamonds, carbide burs, stones, disks, strips, knives etc. may all be used to remove excess material and shaping in order to correct the macro-morphology. The transition line between tooth and resin material can be masked by letting structures in the natural tooth surface such as curves, grooves or crests cross the margins and continue in the surface of the restoration. It may be difficult to distinguish between resin composite material and tooth structure, and care should be taken not to remove sound tooth structure. Overheating is another process that harms the pulp and deteriorates composite

**Fig. 29.5** Restoring technique using a crown matrix A. A fractured central incisor. B. Preparation of a facial bevel with a flame shaped diamond. C. Preparation of a palatal champher using a round stone or a rounded conic diamond. D. If sufficient enamel in the gingival area is left, a bevel can be made using a metal polishing strip. E. The final preparation. F. Shade selection for the enamel is made in the incisal area of the neighbour tooth, whereas dentin colour can be selected in the gingival area of the canine. G. A corner matrix is adjusted to cover only 1–2 mm outside the preparation. The right wedge is selected. H. After acid etching and bonding the dentin part of the restoration is build up by hand using dentin-material (opaque) followed by light-curing. I. For restoration of the enamel part the corner matrix is used. Only the facial and palatal part of the matrix is filled with enamel-material (translucent). This can be done before the bonding procedure starts, as long as the filled matrix is kept protected from light. After placement surplus of material is removed before light curing. J. The restoration is finished and polished to mimic the synergist tooth by morphology, surface structure and reflexes. K. The lingual aspect of the final restoration.

material creating visible white lines along the margins. Therefore, water-cooling should be applied in all finishing procedures.

Surface gloss and texture may be as important as the color for the final esthetic result of a restoration. Technicians spend a long time polishing indirect restoration before sending them to dental offices. In direct restorations this must be done by the dentist. Fine polishing disks or rubber polishers can be used for surface polish, but care should be taken not to sacrifice the morphology of the finished surface. Finally, a highly reflective surface gloss may be obtained using abrasive polishing paste applied with a brush. In teeth of young people natural surface texture (micro-morphology) reflects light in many different directions. This makes them look shiny and lively and may give the illusion of a tooth with a higher value (Fig. 29.6 A). In older people wear or erosion has often removed surface structure and the reflection of light will be from only one or two reflexes (Fig. 29.6 B).

**Fig. 29.5** (*Continued*)

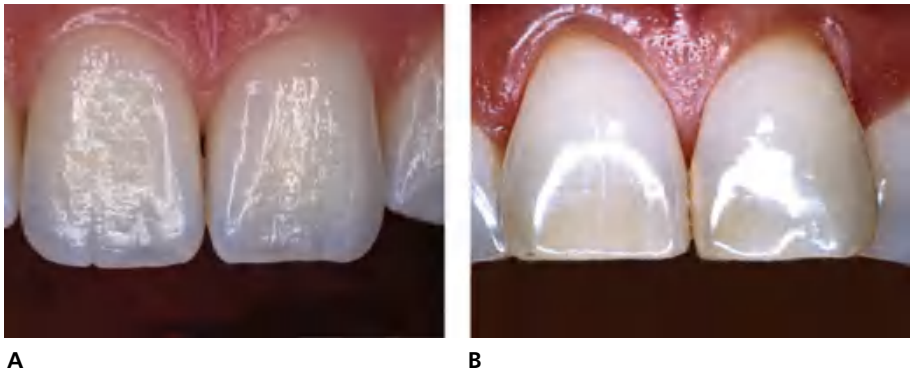


**Fig. 29.6 Awareness of surface structure is important. A composite crown build up should have the same surface texture as the adjacent natural teeth.**
A. In teeth of young people natural surface texture reflects light in many different directions, which makes them look shiny and lively. B. In older people wear or erosion has often removed surface structure and the reflection of light will be from only one or two reflexes.

A composite crown build-up should have the same surface texture as the adjacent natural teeth. Texture of a restoration can be made with a sharp stone or a diamond after polish, but before the final polishing paste. Creating surface texture when needed will often give the finishing touch to an esthetic direct crown build-up.

## Indications

The initial treatment of a fractured front tooth will most often be a resin composite crown build-up. Concerning the final treatment, the pros and cons for the direct resin composite restoration or porcelain laminates (see Chapter 30) or full crown (porcelain/porcelain-fused-to-metal) (see Chapter 31) should be discussed in all cases. Age of the patient, size of the fracture, bite function/occlusion, parafunctions, esthetic need and economy and not least the skills of the dentist – and the technician – should be included in the decision.

A disadvantage with resin composite crown build-ups is the lack of information on durability. However, the durability of Class IV restorations with newer materials and bonding systems has improved – as already mentioned – and although longevity of ceramic restorations still is better, the biological consequences by the invasive treatment and the high initial cost for the patient should be included in the treatment decision.

Full coverage should be avoided in the young adolescent or the early adult dentition in order to prevent a negative pulp response of the combined irritants of trauma, extensive preparation, temporization and cementation. Young anterior teeth may not have reached the state of full eruption and the gingival response to placement of a crown margin beneath the free gingival crest is often less than positive (68). Fracture resistance with modern resin composites and bonding materials is acceptable, and in case of occurrence of a fracture, it is easily and reliably repaired (69). Chipping at the margins and marginal discoloration

appearing over time may be removed by finishing the margins. Therefore, direct composite resin crown build-ups will often be able to cover the demand for esthetics, biological acceptability and resistance to abrasive wear, where the size of the fracture is small to medium and should be first choice of permanent treatment in such cases both in young and older patients (70). For larger crown fractures the choice of treatment may more often be a ceramic restoration, which also depends on both the skills of the dentist and the economic resources of the patient. If in any doubt, the least invasive treatment should always be preferred. If it does not meet the expectation of the patient or the dentist, the non-invasive treatment may always be changed to a more invasive treatment later, whereas the opposite is not possible.

## Essentials

### Indications

Resin composites offer a conservative restoration of crown-fractured teeth with a minimal risk of pulpal and periodontal complications in both children and adults. The restoration can be performed immediately after injury if there is no associated periodontal or supporting bone injury. Hybrid resin composites and amphiphilic bonding systems are especially useful.

### Durability

Class IV resin restorations have a shorter longevity than full-ceramic and porcelain fused to metal crowns. The durability of restorations with newer materials and bonding systems has improved. Compared to ceramic restorations a resin composite restoration may be easily and durably repaired.

### Biology and economy

A composite build-up of a fractured tooth is more conservative to the hard tooth-structures, the pulp and the surrounding soft tissue than a crown. Considering that the resin composite build-up may need replacement 2–3 times in order to equal the durability of a crown, the economic consequences for the patients over time may be the same for both treatments. However, crowns are also replaced or repaired over the years due to caries, fracture of ceramic, lack of retention or for esthetic reasons.

### Skills of the dentist

Clinicians often look for a miracle material, but the final result is linked to the clinician's manual skills and knowledge. All steps in the direct restorative procedure are mandatory for a lasting and esthetic restoration.

### Treatment

(1) Shade selection and preparation of matrix system.
(2) Select resin composites for layering technique.
(3) Prepare a 1–1½ mm wide chamfer or bevel. To mask the transition line between tooth and resin material a facial bevel may be shaped like a stair-step or a wave.
(4) A pulp protective agent should be applied only on locations in dentin with very close relation to the pulp.
(5) Enamel and dentin bonding.
(6) Insert the resin composite material in opaque (dentin) and translucent (enamel) layers of max. 2 mm. Characterization materials may be added below the last enamel layer or may be part of the enamel layer, depending on the resin system.
(7) Each layer is formed to the right morphology and polymerized for at least 20 sec. each.
(8) Remove matrix and major excess of material.
(9) Trim margins and finish to correct morphology with diamonds, burs or discs. Polish with rubber polisher, abrasive discs and paste.
(10) Surface texture can be made using a sharp stone or a diamond bur before final polish.

### Survival of resin composites

Survival of crown build-ups are dependent on:

- Size of restoration
- Skills of the dentist
- Bruxism/clenching
- Oral hygiene
- Material properties
- Enamel and dentin bonding systems
- The patient's esthetic needs

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# 30

# Restorations Using Porcelain Laminate Veneers

D. M. Layton & T. R. Walton

## Introduction

According to the 2017 Glossary of Prosthodontic Terms (1), a porcelain laminate veneer is a "*thin bonded ceramic restoration that restores the facial, incisal and part of the proximal surfaces of teeth*". The prosthesis is also known as a porcelain veneer. These veneers, by definition, are thin and conservative, encasing less tooth structure than onlays and three-quarter crowns.

Dental veneers were first described in 1937 by Pincus (2) to temporarily improve the appearance of teeth. The technique gained mainstream acceptance when the introduction of acid etching by Buonocore (3) and composite resins by Bowen (4) improved the predictability of the bond, as outlined by research in the 1980s by Simonsen and Calamia (5) and Horn (6). Dental veneers are now an accepted and regularly-used restoration.

The incidence of veneer treatments has been reported infrequently. In the General Dental Services of England and Wales porcelain veneers accounted for 0.5% of treatments (2562 out of 503 965 over 11 years (7, 8)). In a prosthodontic practice in Australia, porcelain veneers accounted for nearly 2% of treatments (304 out of 5712 over 16 years (9)).

Recent years have seen an increase in ceramic materials apparently suitable for veneers, with outcomes supported by literature of varying degrees of scientific rigor. These apparently new-and-improved materials are heavily marketed to both dentists and the general public. In a commentary in 2009, Friedman (10) lamented, stating "*the economic incentives associated with the porcelain veneer "industry" have spawned disturbing trends, misleading information, and an unprecedented level of overtreatment in our profession*." An improved understanding of the indications, clinical procedures, risk factors and related outcomes for these restorations would clearly be of benefit.

The veneering technique allows tooth discoloration, malalignments and unesthetic contours to be disguised and teeth that have lost structure through trauma, caries, erosion, abrasion and parafunction to be restored. However, it is technique sensitive. For veneers to survive over the long term, it is important that careful attention is paid to each step of the process – from the initial assessment through the tooth preparation and impression, to bonding and maintenance. The following chapter will discuss and illustrate each of the clinical aspects of this process.

## Patient assessment

In addition to a general assessment encompassing patient wishes, extraoral and intraoral assessments should focus on aspects that may specifically impact porcelain veneer design and longevity.

Veneers can be placed on permanent teeth from post-adolescent years. Ongoing passive eruption is not a significant contraindication, as ideal veneer margins should be placed in enamel equi- or supra-gingivally. Preparations are minimal, making this rehabilitative treatment appropriate for teeth with large and immature pulp chambers, and for teeth with developing roots.

Veneers can restore damaged tooth structure, alter orofacial appearance, or both. Veneers can also be used to overlay and reinforce teeth where fractured fragments have been reattached following trauma (11).

When used to alter orofacial appearance, such as tooth color, contour and alignment, the expected outcome should be clearly discussed before embarking on treatment. This is especially important when multiple veneers are planned, and changes to the smile line, lip line, buccal positioning and intra-arch distribution are contemplated. Mock-ups

*Textbook and Color Atlas of Traumatic Injuries to the Teeth*, Fifth Edition. Edited by Jens O. Andreasen, Frances M. Andreasen and Lars Andersson.
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(Fig. 30.1), photographs, study casts, diagnostic wax-ups and digital work-ups are of use, and arguably essential. Psychosocial assessments to identify patients with unrealistic expectations and potential body dysmorphic disorders should be considered.

Veneers can improve tooth color through careful choice of porcelain shade, porcelain thickness and bonding resin value. However, the potential for color change is limited, and cannot be trialed to allow patient assessment prior to fabrication. Pre-treatment vital and non-vital tooth bleaching can be considered, but this cannot alter the shade of discolored cementum and roots. Examples of discolored teeth suitable, and not suitable for veneering are demonstrated in Fig. 30.2.

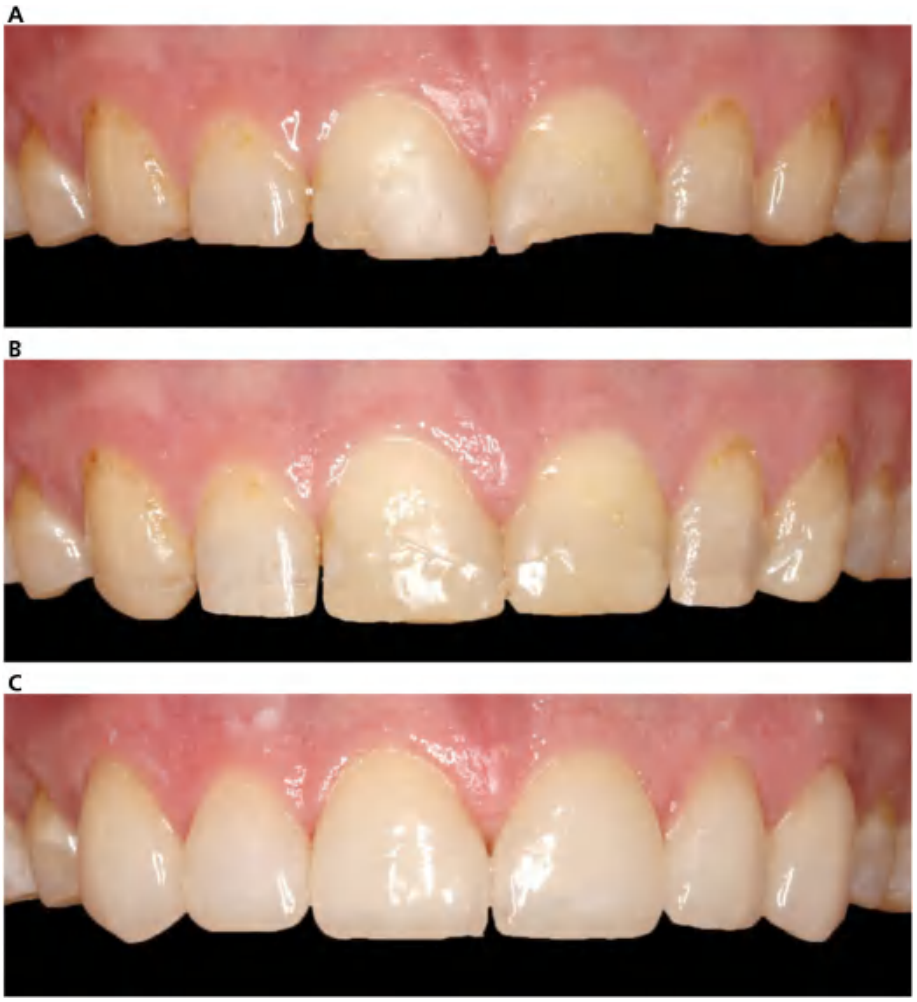



**Fig. 30.1** Diagnostic intraoral composite mock-up to assess the length for veneers planed to restore maxillary incisors and canines. A. Before treatment. B. Composite intraoral mock up, with changes made to incisors and canines. C. The final feldspathic porcelain veneers. The veneer length of the canines was increased slightly to allow future rehabilitation of the contour of the premolar teeth.

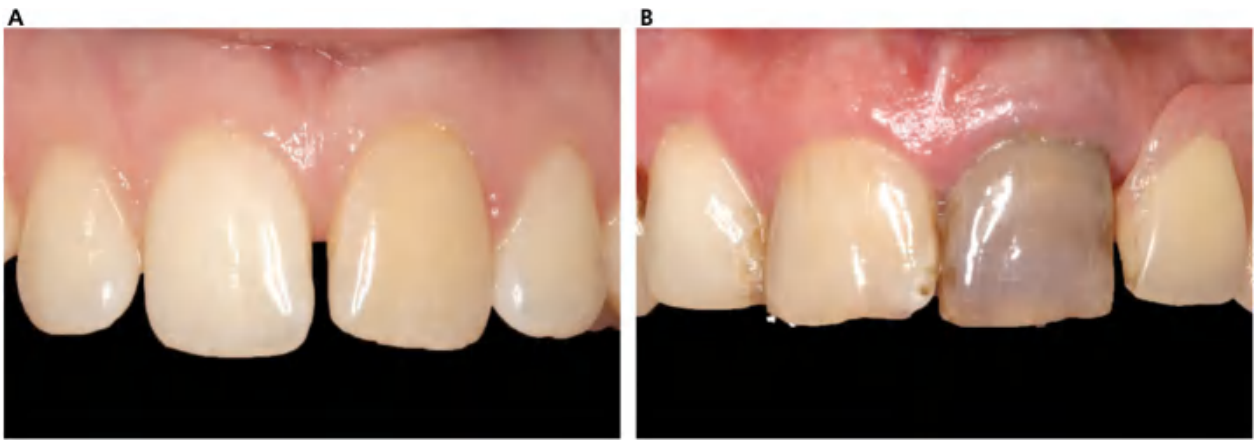



**Fig. 30.2** Examples of discolored teeth suitable (A), and not suitable (B) for veneering. Both teeth are resistant to tooth bleaching. A. The maxillary left central incisor, 10 years after acute injury from a bicycle accident. The tooth is in infraocclusion, but the gingival margins remain at an acceptable level. B. The maxillary left central incisor, over 15 years after an acute ice hockey injury. The remaining tooth structure in both cases is sufficient to facilitate veneering; but the porcelain veneer would not adequately disguise the discolored tooth in B. The maxillary left central incisor in A is suitable for a porcelain veneer, while the maxillary left central incisor in B is not.

When used to restore damaged teeth, such as those affected by trauma, caries, erosion, abrasion and parafunction, the amount of remaining tooth structure should be carefully assessed. It is important that sufficient enamel is present to support final veneer bonding, and further discussions relating to this criterion are undertaken later.

Veneers should not be placed immediately following traumatic injuries. Definitive management of such teeth should be delayed until initial monitoring stages have been completed. This will allow intruded teeth to re-erupt, suspected fractures to be reviewed and vitality to be monitored. Teeth where trauma results in pulpal changes may change color. Such teeth that undergo root canal treatment may respond to non-vital bleaching prior to veneering, and other teeth that do not require root canal treatment can have the shade discrepancies disguised by different porcelain materials, careful shade selection and final bonding agent choice. Ongoing changes to the color of the underlying tooth structure may adversely impact the appearance of the translucent porcelain veneers over time.

Often the damaged teeth have been previously restored, and the extent of the restorations can be difficult to judge clinically. Radiographic assessments can help indicate their size. It may not be appropriate to proceed with veneering if the restorations are large, such as those that span more than 1/3 of the mesio-distal width of the tooth.

Assessment of tooth vitality with pulp sensibility testing and radiographs should be undertaken. Teeth requiring root canal therapy can still be suitable for veneering, if the marginal ridges remain intact. However, if one or both marginal ridges have been undermined, it is likely that use of a full coverage crown would be a more appropriate restoration of choice.

Assessment of gingival health is also specifically important to porcelain veneers. The veneers are adhesively bonded, and this process is undermined when the gingival health is suboptimal. In addition, long-term plaque accumulation impacts bonding resin integrity and contributes to marginal staining, bonding degradation and caries.

Official diagnosis of parafunction can only be undertaken with formal sleep studies, but clinical signs and reported symptoms can subjectively indicate that habits are present. Attrition and bruxofacets indicate that parafunctional loads have been present in the past, and restoration of this damage often leads patients to enquire about porcelain veneers. In addition, soft tissue changes including linea alba, tongue scalloping and lip indentations indicate that the habits persist. Parafunctional habits create higher compressive and tensile loads, both in magnitude and duration, than those produced during normal function, thereby increasing the risk of fracture.

Although the presence of one or more of these risk factors complicates veneer treatments, they are not direct contraindications. However, evidence shows that veneers in such environments can be expected to have reduced longevity, and patients should be advised accordingly prior to commencing treatment. Provision of occlusal splints may enhance the veneer longevity where significant parafunction is suspected.

It is not always clear prior to commencing treatment whether a tooth is suitable to be veneered. Initial steps involve restoring small carious lesions, replacing failing restorations, and placing composite in small areas that would have undercuts (such as a small interproximal defect). These steps are important to allow the remaining tooth structure to be directly assessed prior to commencing veneer preparation. Teeth that have been traumatized or are being treated for esthetic reasons, have often been restored with composite resin restorations for the short or medium term. It is then recommended to remove these restorations before confirming whether it is appropriate to continue with a porcelain veneer or to proceed with a more extensive treatment.

Research has shown that the longevity of porcelain veneers is associated with the amount of prepared enamel available for bonding (12–14). The results support the following general rules. If the majority of the periphery and 80% or more of the bonding surface of the tooth substrate is prepared enamel, then it is appropriate to continue with a porcelain veneer. If the prepared enamel covers between 50% and 80% of the surface, then it is appropriate to discuss with the patient the risks of leakage and failure, and clearly include them in the decision of whether to proceed or not. If the prepared enamel covers less than 50% of the surface, then it is recommended that the clinician consider abandoning the veneer procedure in favour of three-quarter or full crown coverage.

Following completion of veneers, patients should be recalled for review. The frequency of the recall is related to their individual risk factors.

## Choosing the material

Today's porcelain veneers are made from two main ceramics – feldspathic porcelain and lithium disilicate. In addition, each material can be manufactured with two different techniques. Feldspathic porcelains (Durecem, Mirage, Fortress, Vita 900) can be formed using a refractory die or platinum foil handling technique. Lithium disilicate is either pressed (IPS e.max press) or, less commonly milled from presintered blocks in a CAD/CAM process (IPS e.max CAD). Both materials are etchable, an essential characteristic for effective enamel bonding of the porcelain veneers.

Other ceramics have been used in the past including castable ceramics (Dicor, CerePearl), pressed lithium disilicate (Empress 2), CAD/CAM Alumina Procera and pressed feldspathic with leucite (Cerinate, Empress 1, IPS Empress Esthetic).

Ideal material characteristics required for veneers relate to the appearance, handling, bonding potential and strength. Veneers should be translucent enough to maximize light transmission, but opaque enough to mask discolorations. The surface finish, luster and shade should be durable over time. The material should facilitate conservative tooth preparations to maximize the enamel substrate for bonding. Handling should be straightforward during fabrication and clinical procedures. Micro-mechanically, they should be

etchable to facilitate retention given the limited resistance form inherent with veneer preparations. When bonded, they should be strong in tension and compression to maintain their marginal seal without distortion, and decrease risk of fracture propagation.

Feldspathic porcelain is a predominantly glassy ceramic, whilst lithium disilicate is a modified feldspathic glass (15). Each material presents with its own advantages and disadvantages for use in porcelain veneer construction. Feldspathic porcelain has been used for porcelain veneers since the early 1980s. It is based on feldspar, which is a naturally occurring glass containing silicon oxide, aluminum oxide, boric oxide, potassium oxide and sodium oxide. The material is etchable, translucent and can be made to be as thin as 0.3 mm. Thus less tooth structure needs to be removed during preparation, thereby conserving the important enamel bonding substrate. The restorations can also appear very life-like, with the transition between material and tooth well-disguised and light reflection and refraction properties advantageous. However, these properties are a disadvantage when the tooth substrate is darkened and requires masking, as can occur as a result of trauma, endodontic medicaments, tetracycline staining and intrinsic color changes.

Fired feldspathic porcelain is weak prior to bonding, but once adhered to the supporting enamel substructure it achieves mechanical characteristics similar to that of porcelain bonded to high noble alloy (such as a ceramo-metal crown). Unfortunately, the egg-shell thin material prior to bonding creates veneers that are awkward to handle both in the laboratory and clinic. Limitations with color masking and handling has meant that manufacturers and clinicians have sought alternatives.

IPS e.max, a feldspathic porcelain with 70% volume lithium disilicate has been in use since 2005, and has no equivalent competitor on the market. The material is thicker than feldspathic porcelain, making it less fragile and easier to handle prior to bonding. The material is also translucent, but e.max is less translucent than feldspathic porcelain. The thicker and denser material contributes to its reduced translucency, and means that it is more effective at masking dark underlying tooth substrate. This thicker material though necessitates a deeper veneer preparation (0.6 mm to 0.8 mm), more removal of the enamel and greater reliance on less predictable bonding to dentin, which can contribute to reduced longevity. Both materials have much higher resistance to compressive compared to tensile forces.

Fig. 30.3 shows two patient cases where two sets of veneers were made. The first case shows feldspathic porcelain veneers where one has more characterization than the other.

The second case shows a different case where the same teeth were restored with e.max press lithium disilicate veneers and feldspathic porcelain veneers. The former looks more solid, and the latter more translucent with characteristic swirls.

## Tooth preparation and veneer design

Tooth preparation and veneer design involves assessment of the remaining enamel; choosing the apical and interproximal margin locations, incisal edge reduction, palatal finish line; and the overall labial reduction required. The authors recommend tooth preparation where 80% of the bonding

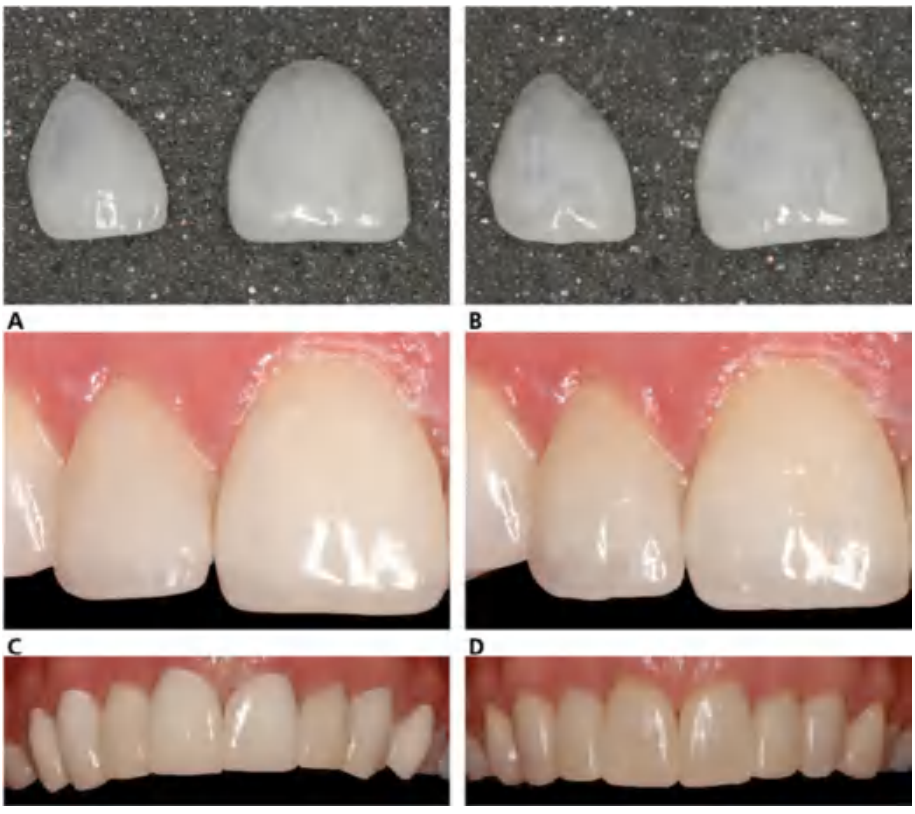



**Fig. 30.3** Two patient cases where two sets of veneers were made. A–B. Feldspathic porcelain veneers on maxillary lateral and central incisors, A with more characterization than B, seated with value zero tri-in Variolink® Veneer paste. C. E.max press lithium disilicate veneers on right maxillary first premolar to left maxillary first premolar. D. The same teeth with feldspathic porcelain veneers.

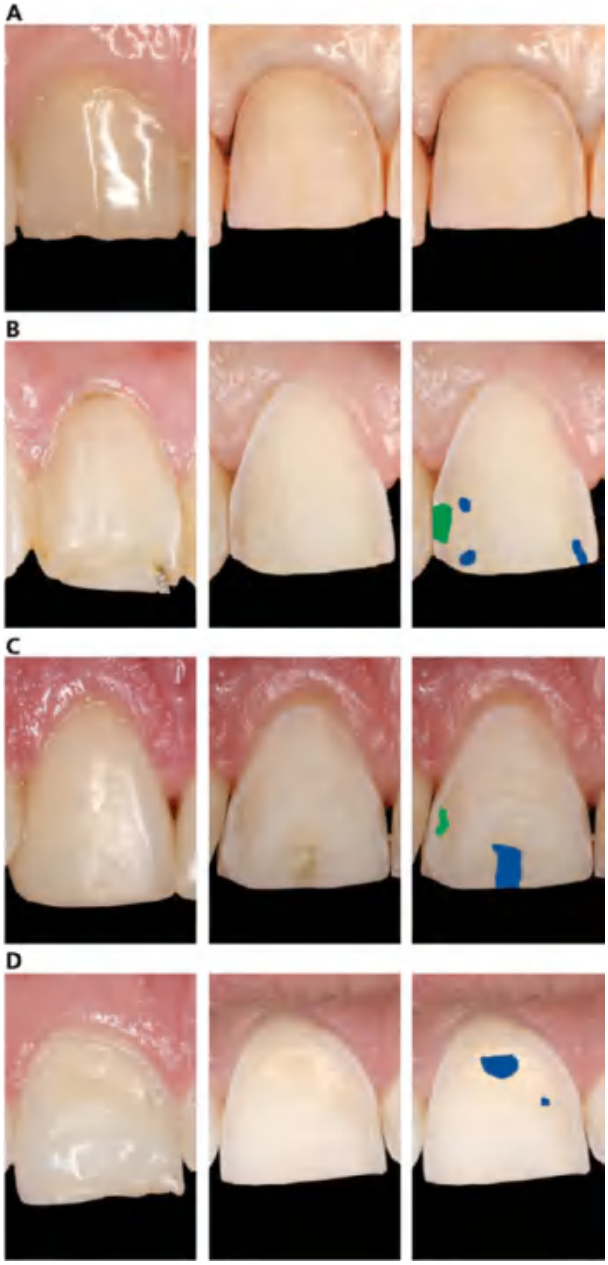



**Fig. 30.4** Examples of teeth before and during tooth preparation. The exposed dentin and the retained restorations have been colored blue and green respectively. A. The maxillary right central incisor has approximately 100% of the prepared surface in enamel. B–D. Enamel preparations between 85% and 95% of the prepared surface.

surface is prepared in enamel, with the apical margin finished above the cemento-enamel junction. Chamfer margins, interproximal contacts reduced on the facial aspects only, incisal reduction of 1 mm to 2 mm, a palatal overlap and a labial reduction of 0.5 to 0.7 mm are preferred. This preparation design was evaluated over a 21-year period, where an estimated cumulative survival of 96% (CI 92% to 100%) was observed (16).

There is no evidential consensus regarding what type of veneer preparation will result in the most optimal outcome. Unprepared enamel is a poor substrate to support porcelain veneers, as its aprismatic structure results in an inferior bond. Studies show that there is a trend towards an increased failure rate when the restorations are not bonded to prepared enamel (17–21), and it is therefore important that clinicians avoid "no-prep" veneer designs. Evidence to guide the choice of margin design (shoulder, chamfer, knife) and incisal finishing design (feather-edged, overlapped) remains lacking. Finite element analysis has been used to assess different preparation geometries (22–24), but clinical studies fail to show a significant difference in outcome (25).

Retraction cord is placed on the labial aspect and interproximal regions of the teeth prior to preparation to minimize the risk of soft tissue trauma. Initial enamel reduction is completed under water spray with a high speed diamond bur and magnification. If desired, the final preparations can be refined with a tapered diamond bur without water spray.

Figure 30.4 shows examples of teeth before and during tooth preparation. The exposed dentin and retained restorations have been colored blue and green respectively. The tooth in Fig. 30.4A has approximately 100% of the prepared surface in enamel, with the traumatic fracture lines clearly visible; the teeth in Fig. 30.4B-D have between 85 and 95% of the prepared surface in enamel, with Fig. 30.4B and C also showing small, retained composite resin restorations.

Thin enamel can be differentiated from dentin by its characteristic frosty appearance following etching. This is demonstrated in Fig. 30.5, where the prepared tooth surface before and after acid etching with 37% phosphoric acid is illustrated. The figures show that it can be difficult to estimate how much enamel will be available as an effective bonding substrate during the initial assessment, and that it is therefore important to re-evaluate the tooth during preparation to reduce the risks of failure.

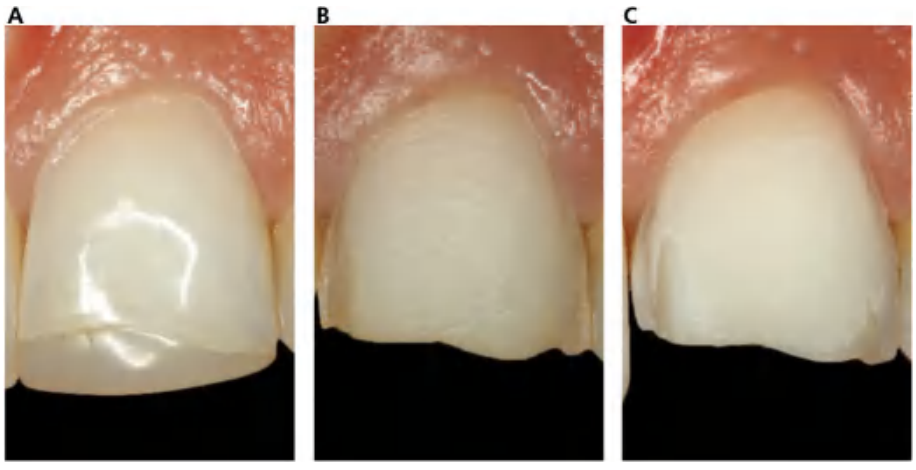



**Fig. 30.5** The left maxillary central incisor A. Before treatment. B. After preparation and before acid etching C. After acid etching with 37% phosphoric acid. The etch pattern assists differentiation between thin enamel, retained restorations and dentin.

Choice of the apical extension for the preparation should be considered carefully. Each example in Fig. 30.4 shows that the gingival margin ceases within enamel, and does not extend below the cemento-enamel junction. This is most evident in Fig. 30.4C. The contour of the gingival margin usually resembles a shallow chamfer. As the tooth extends apically, the enamel becomes thinner and it is more difficult to maintain the preparation within the enamel substructure. Without overcontouring the emergence profile of the final veneer, it is easier to achieve an enamel gingival margin if using feldspathic porcelain when compared with lithium disilicate. Feldspathic porcelain can be fabricated as thin as 0.3 mm, but it is difficult to fabricate lithium disilicate thinner than 0.6 mm. Examples of chamfer gingival margins can be seen in Fig. 30.6. The marginal depth varies between teeth, with that of the prepared tooth in Fig. 30.6B the deepest, being designed to encompass a gingival buccal abrasion lesion. It is likely that in some instances, the preparation will extend onto the exposed root surface. Both the clinician and patient should be aware that this might result in marginal staining in the medium to long term.

By definition, veneers restore the facial and part of the interproximal surfaces of teeth. This means that the interproximal contacts are not intentionally removed during the preparation, and are reduced on the facial aspects only. Such intact interproximal margins are visible in the preparations shown in Figs 30.4A through 30.4D, including the mesial margin on Fig. 30.4B. The palatal portion of its mesial marginal ridge remains intact, only appearing to be reduced because the adjacent tooth had been prepared for a crown. This definition is often forgotten, with three-quarter crowns being misclassified as "veneers". Examples include teeth with significant loss of interproximal structure, teeth that are severely microdontic (Fig. 30.7), or teeth that require significant reshaping to resemble a different tooth (such as transplanted teeth, or canines disguised as lateral incisors). It is important to recognize the difference between these two types of restorations, as the bulk of the restoration would influence the material choice, failure risks and survival rates.

Viewing the preparation from the palatal angle (Fig. 30.8) shows the classic double curve appearance, where the midlabial face is positioned more buccally, and the chamfer margins extend interproximally to the contact points creating a curved appearance at the mesial and distal margins.

## Incisal edge reduction

There are differences of opinion in the literature regarding whether to reduce and overlap the incisal edge during preparation or maintain the current incisal edge location regardless of whether the final plan involves tooth lengthening. A systematic review assessing the outcomes of veneers with different preparation geometry did not find a difference in outcome (25), but this could be due to the low numbers of studies available for assessment. The authors prefer a palatal overlap design.

Esthetic porcelains such as feldspathic and lithium disilicate can support their own structure up to 1.2 mm in length, and possibly beyond. Reducing the incisal edge (or planning to increase the length) provides the technician with room to control light transmission in the region and enhance the esthetic contour. It also simplifies the clinical steps of blending the palatal overlap into the outline form and ensuring the veneer is definitively seated during adhesive luting.

Example preparations can be seen in Fig. 30.9. The incisal edges in Fig. 30.9A were not reduced, because lengthening was planned, but the finishing line overlapped onto the palatal. However, comparison between adjacent teeth on Fig. 30.9B clearly show the reduction provided, and the length restored.

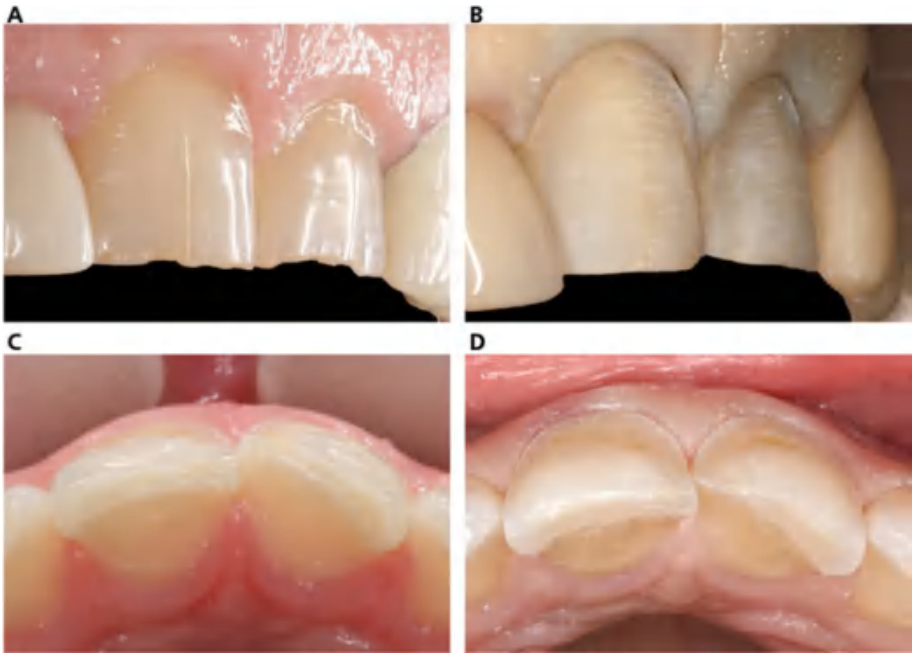



**Fig. 30.6** Examples of porcelain veneer preparations with chamfer gingival margins. A–B. Facial views of the first case before and after preparation of both maxillary central incisors. C–D. Axial views of the second case before and after preparation of both maxillary central incisors.

**Fig. 30.7** Treatment of microdontia. The maxillary canine and lateral incisor teeth required significant reshaping to improve the esthetic contour and orofacial harmony. Despite the minimal preparations and bonding to enamel, the bulk of porcelain implies that these restorations are three-quarter crowns, rather than veneers. The restorations placed on the maxillary central incisors are minimal and are classified as porcelain veneers.

**Fig. 30.8** The chamfer margins extend interproximally to the contact points creating a curved appearance at the mesial and distal margins.

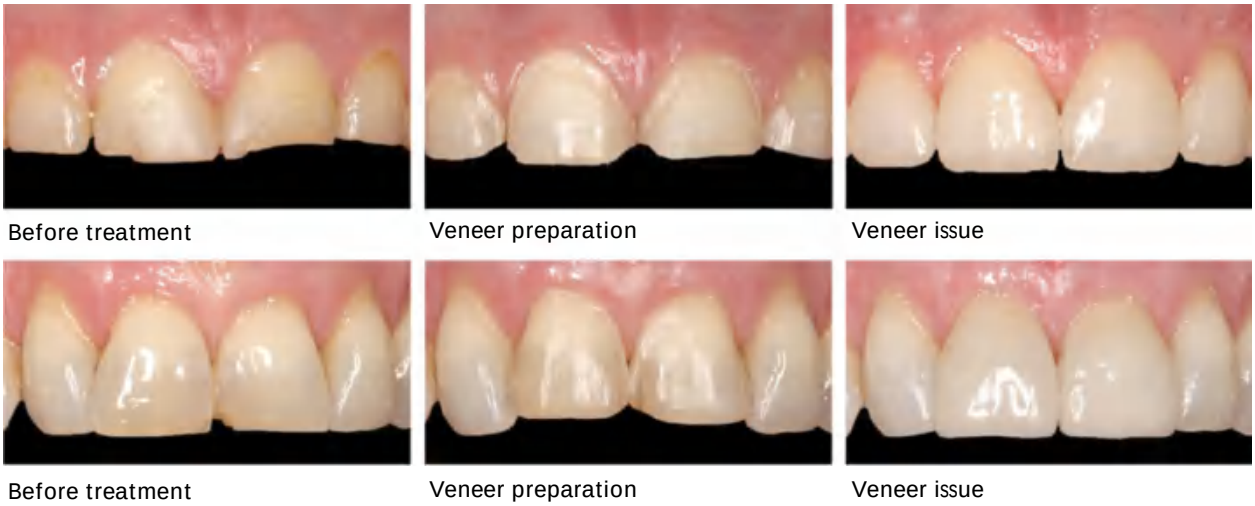



**Fig. 30.9** Preparations of teeth for porcelain veneers. A. No reduction of the incisal edges of the central and lateral incisors during preparation, with the length of these teeth increased with the veneers. B. Reduction of the incisal edges of both central incisors, as seen compared to adjacent teeth; which were then restored to the original length with the veneers.

### Palatal finishing line

When the veneer preparation finishes at the incisal edge, retention of the prosthesis is solely derived from the luting adhesive. However, when the preparation is overlapped onto the palatal surface, resistance form is provided in one direction, resisting movement of the prosthesis away from the tooth, thereby contributing to retention. If the veneer is not overlapped to the palatal surface, there is a risk of loss of incisal edge tooth structure through attrition or erosion, resulting in the formation of a ledge between the tooth and the veneer. This can provide a source of tensile stress directly applied to the incisal edge of the veneer resulting in fracture. Therefore, in addition to improved preparation geometry, overlapping the incisal edge protects the integrity and form of the underlying tooth structure.

When overlapping the palatal surface, the finishing line should be free of any contact with the opposing arch in maximum intercuspation. Preferably, the occluding pattern is maintained on natural tooth structure. However, if this is not possible, the palatal overlap can be continued for at least 1 mm past the occlusal contact point, with the contact pattern in maximum intercuspation now resting on supported porcelain.

### Overall bulk reduction

Veneer preparations can remove up to approximately 0.8 mm of tooth structure, but as outlined above, care must be taken to remain within enamel where possible. As the labial reduction extends towards the gingival margin, the enamel becomes thinner and the preparation should remove less tooth structure in an effort to keep the periphery rimmed by enamel. Preparation depth should be sufficient to achieve the desired final outcome within the limits of the porcelain material. These outcomes include matching the original tooth form, harmonizing with the adjacent tooth form, or creating a new tooth form to disguise contour and alignment concerns.

Following assessment, some treatments plan to thicken teeth with porcelain veneers. This has led to marketing companies espousing the "no-prep" veneers. Although enamel may not need to be removed in every case to create sufficient room for the emergence profile and final porcelain thickness, research has clearly shown that unprepared enamel is a poor bonding substrate with outcomes of veneers bonded to such structure only achieving 58% survival over a 6.5 year period (19). Some minor preparation of the surface is required to remove the aprismatic layer, even when thickening the tooth labially.

### Dentin sealing

The procedure described in this chapter does not include immediate dentin sealing. This is because it is recommended that the maximum amount of dentin exposed be maintained below 20%. Excellent longevity of greater than 96% over a 21-year period was achieved with this traditional veneer procedure (16), whilst a decreased survival of 65% was observed over a 10-year period in a different study when the amount of bonding enamel was reduced (18, 26). If continuing with porcelain veneers where more dentin is exposed, employing a dentin sealing procedure following preparation and prior to the impression should be considered (27).

## Impressions

Veneer impressions are taken with addition polyvinyl siloxane such as President® (Coltene), Imprint™ 3 or 4 (3 M ESPE) and Take 1® Advanced (Kerr). Porcelain veneer preparations finish equi- or supra-gingivally. Therefore, they can be completed with a one or two cord technique. The first cord is placed before tooth preparation to protect the soft tissues rather than provide retraction, and it is preferably left in-situ during impression procedures. A second cord can be placed if desired, and removed prior to the impression. This cord may be placed to span the length of all the veneer preparations, and the wetted cord can then be removed from the entire region with a single movement of a pair of tweezers, simplifying the impression procedure (Fig. 30.10). If there are substantial interproximal spaces, there is a risk that the impression material will squeeze underneath intact contact points during the impression procedure, thus bridging between the labial/buccal and palatal/lingual regions. The material must tear, possibly across the margin, when the impression is removed. To reduce this risk, soft orthodontic wax can be placed across the gingivo-palatal surface, preventing this bridging (Fig. 30.11).

## Bonding

Veneers are translucent, and their color is influenced by the shade of the underlying substrate. When choosing the veneer shade, the shade of the prepared, but hydrated tooth surface is also taken and used to guide the porcelain build up in the laboratory. Trial of the appearance of the veneers intraorally is important prior to final cementation.

Some teeth are veneered because patients are unhappy with the color of the teeth. This can occur with an internal hemorrhage following trauma, staining from endodontic medicaments, excipient inclusion during tooth formation and intrinsic color defects. It is preferable that any masking be attempted during formation of the veneer, but if required, minor tooth darkening can be disguised and lightened during the bonding procedure by selecting a high-value bonding agent. However, masking discoloration at this stage can be risky as the amount of change in color cannot be guaranteed, and the clinician and patient may be unhappy with the final result.

The veneers are fiddly to handle and brittle, but can rest on the tooth by placing a drop of water, glycerin or tri-in paste

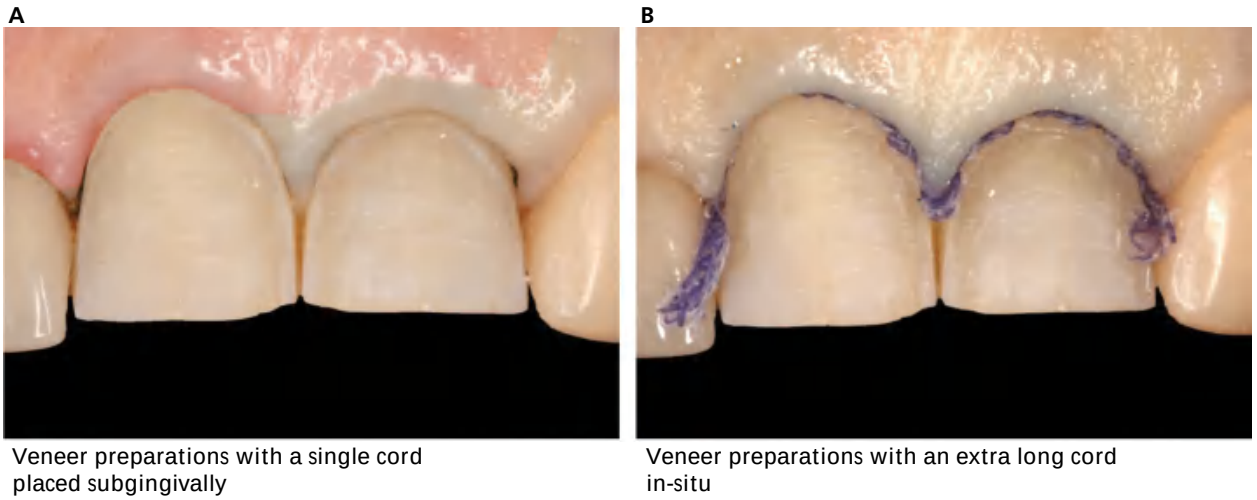



**Fig. 30.10** Placement of two gingival cords. A. The first cord is placed subgingivally. B. A second long cord can be placed spanning the length of both teeth after the preparations are completed. The second cord can be moistened and removed from both prepared teeth using a pair of tweezers with a single movement, simplifying the impression procedure.

**Fig. 30.11** Soft orthodontic wax is placed in the bucco-palatal communication between teeth to reduce the risk of tearing of the impression material at the margin during removal.

under the fitting surface. Bonding kits, such a Variolink® Veneer (Ivoclar Vivadent) provide bonding resins and matching tri-in pastes with a range of different "shades" designed to mimic or alter the apparent value of the veneer in-situ. There is a neutral shade (value = 0), three shades that consecutively darken the value and three shades that consecutively lighten the value. The impact of the shade of the bonding resin on the appearance of the veneers is illustrated in Fig. 30.12

Retraction cords are not recommended during bonding. The cord pushes the gingiva apically and exposes cementum, increasing the risk of reversible pulpitis and associated patient discomfort secondary to acid exposure during bonding.

Once assessed by both the clinician and patient for fit, color and contour, the veneers are washed with water, prepared with hydrofluoric acid, and silianated. Care must be taken to effectively and safely apply hydrofluoric acid to remove saliva and material contaminants, and prepare the surface for bonding. Ineffective preparation results in an inadequate bonding strength.

The interproximal regions are abraded with a metal or sandpaper strip to facilitate placement of celluloid strips during the bonding procedure. The incisal edge is lightly planed in order to remove minor irregularities that may have been abraded on the model during fabrication. This ensures accurate adaptation of the veneer to the tooth surface and minimizes the risk of creating localized tensile stresses during bonding. The tooth substrate is cleaned with pumice and water, etched with 37% phosphoric acid, and the bonding system applied. Care should be taken to follow the instructions for individual bonding systems. Each veneer should be bonded and finished individually, and can be isolated from the surrounding dentition with celluloid strips or teflon tape. Fig. 30.13 outlines the steps involved in veneer bonding.

## Review and maintenance

All patients should be placed on a tailored maintenance program, with recall timing determined by individual patient-based factors. These include the patients' susceptibility to disease; changes in the patients' physical and mental condition; the complexity of the treatment; the patients' willingness and effectiveness in carrying out hygiene procedures; and the perceived biological, physiological and mechanical stability of the oral and associated extraoral tissues over time. In addition to a general assessment, traumatized teeth are reviewed, the occlusion may be adjusted, minor porcelain chips that are not impacting esthetics or function may be smoothed, the marginal integrity reviewed and the patient's satisfaction with the treatment be assessed. Although veneers have an excellent long-term prognosis, concerns arising from this assessment should be reviewed closely to evaluate whether complications or failures have occurred.

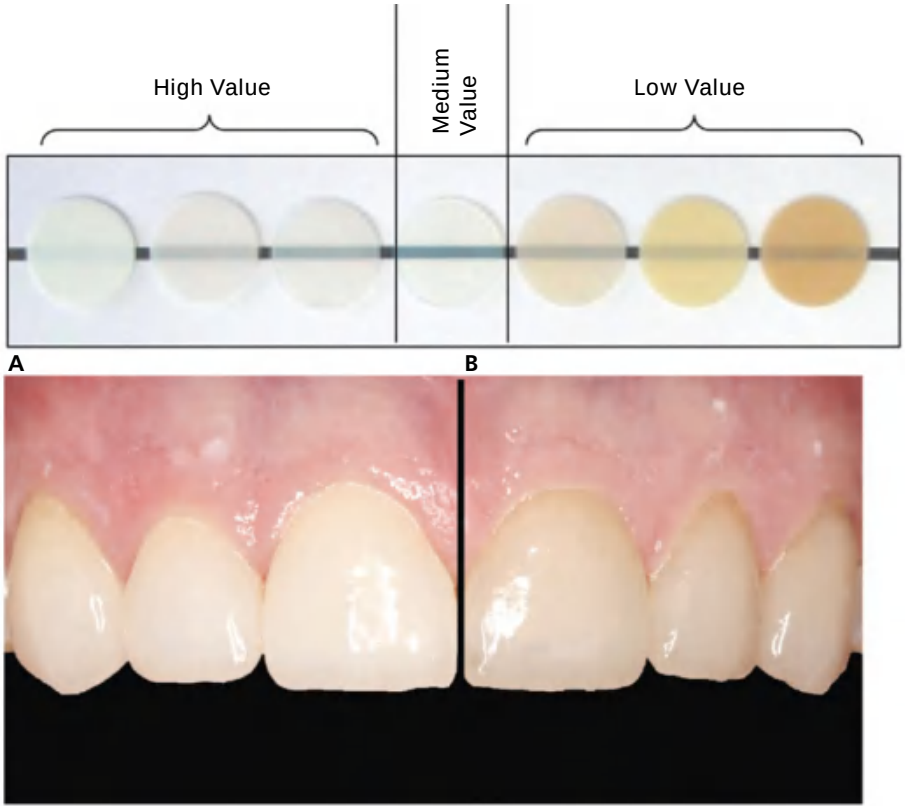



**Fig. 30.12** Illustration of the impact of the shade of the luting cement on the appearance of the veneers. The color chart has been reproduced with the permission of Ivoclar Vivodent®. A. Porcelain veneer with value + 3 tri-in paste. B. Porcelain veneer with value - 3 tri-in paste.

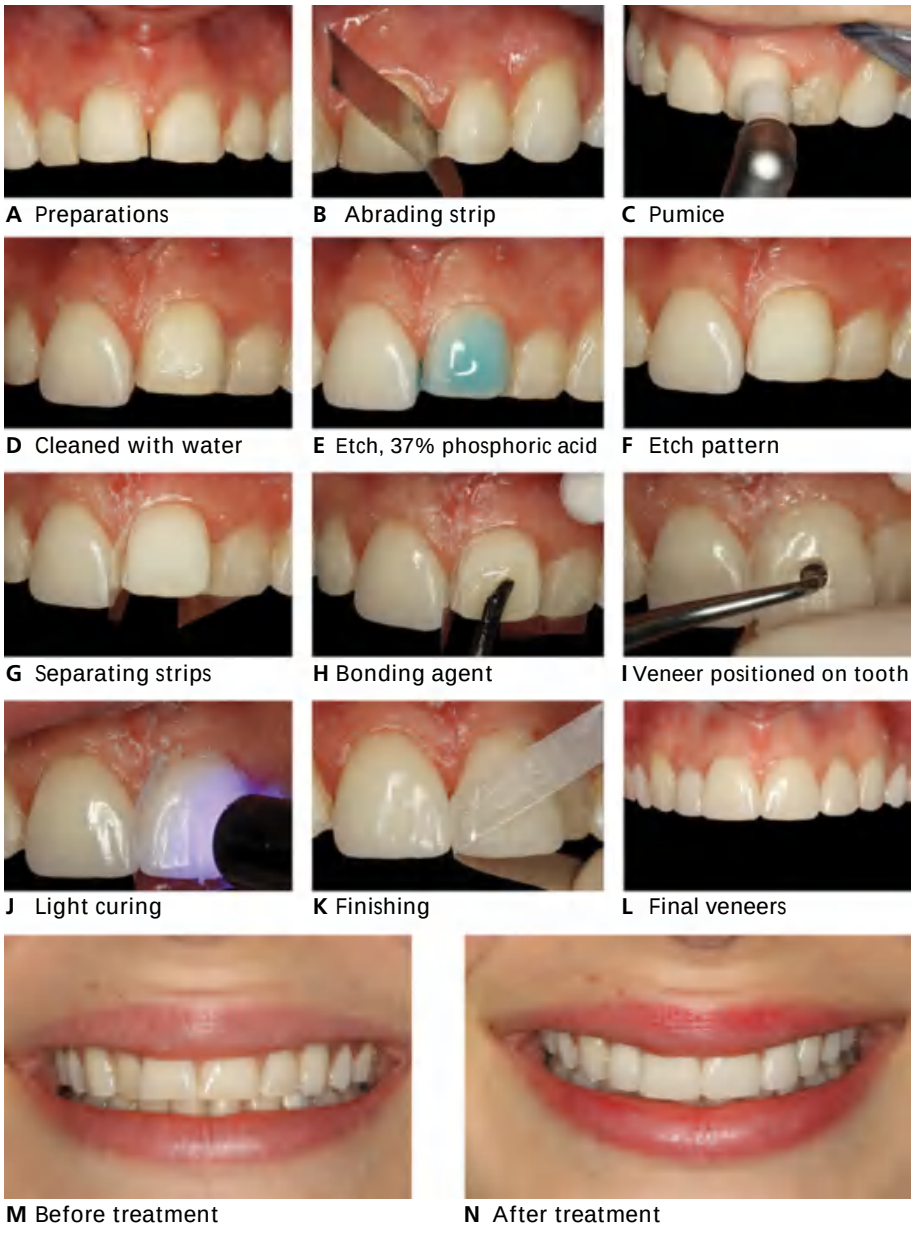



**Fig. 30.13** Outline of the steps involved in veneer bonding. A. Tooth preparations. B. Abrasive strips are used to free interproximal contacts. C–F. The prepared tooth is cleaned with pumice and water and etched with 37% phosphoric acid. G. Interproximal celluloid strips are placed. H. The unfilled bonding resin is applied. I. A thin layer of selected filled resin is carefully applied to the etched fitting surface of the veneer and positioned on the tooth after preparing with hydrofluoric acid and silane. Labial and incisal pressure is applied to fully seat the veneer. J. The resin is spot cured at the margins. Excess resin is removed and the remaining resin fully cured. K–L. The interproximal and palatal margins are then trimmed and polished. M. Clinical view before treatment. N. Clinical view after treatment.

## Longevity and risk factors

Many materials have been advocated for the fabrication of porcelain laminate veneers over the past 30 years, with feldspathic porcelain and e.max lithium disilicate now regularly used in daily practice. Use of leucite reinforced feldspathic, such as IPS Empress Esthetic also continues. Evidence for the survival of feldspathic veneers is stronger than that for leucite reinforced feldspathic, which in turn has more evidence than that for lithium disilicate. This is primarily because the former has been available since the early 1980s, and the latter since the mid-2000s.

Two recent systematic reviews (13, 14) reported the estimated cumulative survival (ECS) at 5 years was 96% for feldspathic and 92% for Empress veneers. The data was less consistent across 10 years, but indicated that the ECS was 97% for feldspathic, but inconclusive for Empress veneers. In other research, the survival was 66% at 2 years for lithium disilicate e.max press porcelain veneers, where the high failure was attributed by the authors to operator inexperience (28).

Outcomes of different types of porcelain veneers across studies of different durations are outlined in Fig. 30.14.

Outcomes of different veneers in different studies ranged dramatically, and were apparently contradictory. This makes it difficult to interpret results. The studies differed by setting, operator, direction of inquiry (prospective versus retrospective) and inclusion criteria. In some studies, patient and restoration numbers were small (17, 21, 26, 29); follow-up times were short (17, 19, 26, 29–31); failure criteria did not include esthetic failures (32); lost to follow-up were unaccounted (32); and inexperienced clinicians skewed results (19, 28). Veneer outcomes may also be affected by environmental

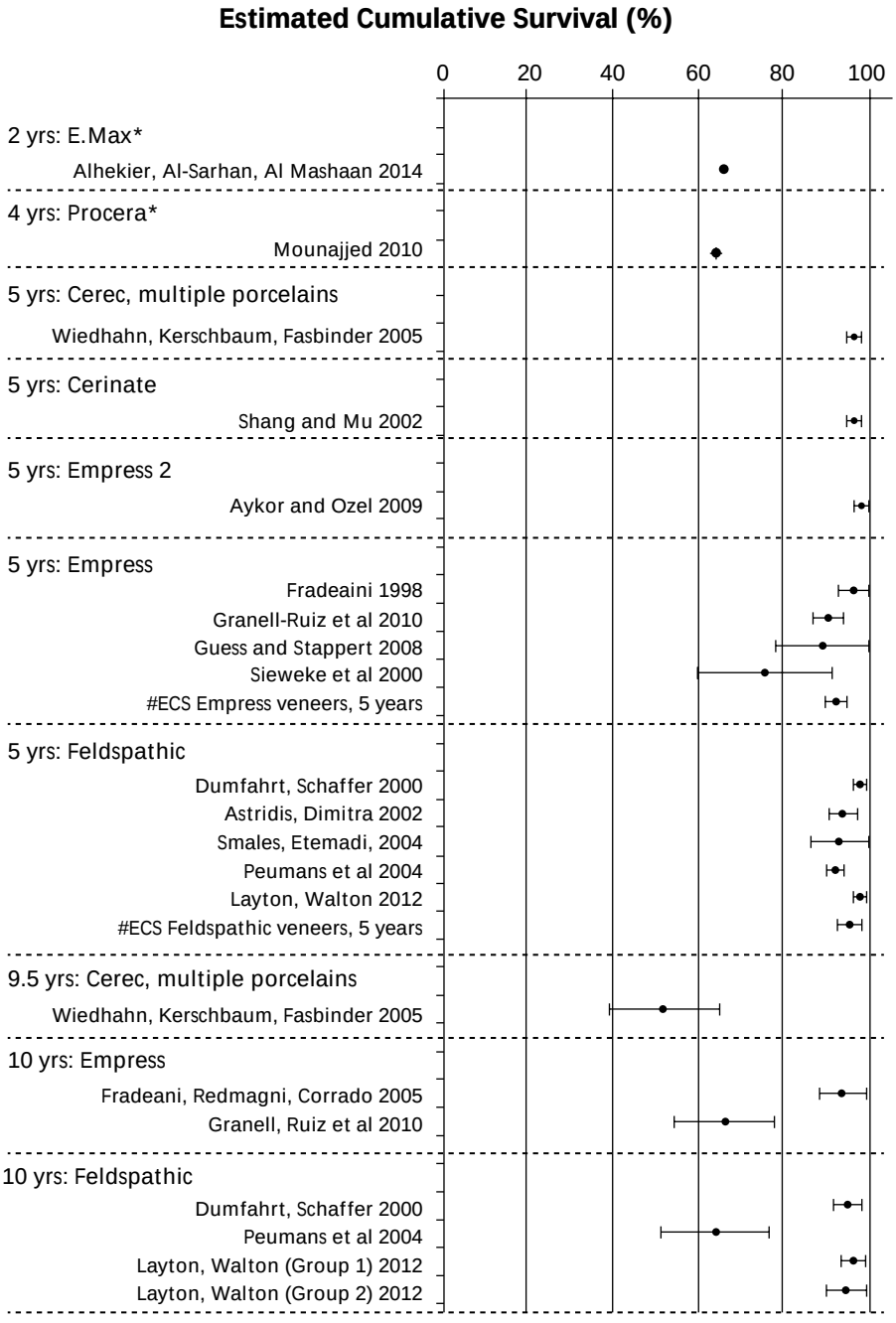



**Fig. 30.14** Survival of different types of porcelain veneers across studies of different durations.

* 95% confidence interval not reported.
# Data combined with meta-analysis (23, 24).

factors such as the venue and clinician, as well as local and systemic patient factors.

Patterns in failures were noted across these studies. Failure rates increased when the quality of the bonding substrate was compromised (7, 17, 19, 21, 26, 29, 33), loads were high (17, 29, 33, 34), intraoral acidity may have been present (17, 29), operators were inexperienced (19, 28, 35, 36) and patient motivation may have been lower (7, 33). Across these studies, 5 and 10 year ECS was below 75%, and could be as low as 47% (19).

On the other hand, studies where the tooth preparation was substantially confined to enamel, and where other risk factors were controlled, had much lower failure rates (9, 20, 37–43). In these studies, 5 year and 10 year ECS were above 90%, and one study reported a 20 year ECS of 96% (16) (Fig. 30.15).

Importantly, in these studies reporting high outcomes over time, the authors described at least one exclusion criterion that related to a perceived inadequacy in enamel bonding substrate. Enamel has an improved micromechanical bond in comparison with dentin and cementum (44), and its high modulus of elasticity (45, 46) imparts natural support to the brittle porcelain. A lack of enamel can be related to patient factors, where patients are unwilling to proceed with a full coverage crown rather than veneer; or operator factors, where remaining enamel is erroneously considered sufficient.

The higher failure rates when bonding substrates were compromised is of particular topical interest. There is a trend towards more aggressive preparations and less emphasis on the importance of enamel as a bonding substrate. Therefore, it is conceivable that such "modern veneers", which in effect are three-quarter crowns, will have reduced survivals when compared with the outcomes of traditional enamel-conserving protocols. It has to be recognized that these types of "veneers" are not veneers at all, and that the excellent survival rates that have been achieved cannot be extrapolated to these new types of restorations.

Additionally, when patients receive more than one veneer, individual characteristics of that one patient will adversely or favorably affect outcomes. The outcome of a single veneer in these patients cannot be considered to be independent from other veneers in the same patient. Thus the outcomes become clustered, and this should be recognized when the studies are designed, reported and read.

The results from all these studies are important for our clinical decisions. It should not be concluded that any one study is better than another, just because the outcomes appeared to be greater. The results are actually telling us that whilst porcelain veneers are possible restorative options in many situations, exposure to high risk situations will result in higher failure rates. Overall, with good clinical technique and low risk populations, feldspathic porcelain veneers can be expected to have a 10-year ECS over 90%. Also, 5-year ECS for etchable non-feldspathic porcelain veneers may be over 90%, but this result is supported by limited evidence, and use of these materials for veneers currently remains experimental. Clinical photographs of patients who have had feldspathic porcelain veneers surviving over relatively long-term periods can be seen in Fig. 30.16.

## Modes of failure

Porcelain veneers may fail for a range of reasons, and these can be related to esthetics, biology, mechanics, supporting structure or patient and operator factors. Veneer procedures are technique sensitive and it is imperative that attention to detail is maintained. Expert technical back-up is also required. Modes of failure are summarized in Table 30.1, and demonstrated in Fig. 30.17.

Outcomes reported in an up to 21-year prospective study shows that the incidence of failure for that patient population was low. Seventeen veneers out of 499 failed across the 21-year study period, equating to 0.46 per 100 prosthesis years. The most common reason for failure related to esthetics, accounting for 10 of the 17 failures (Fig. 30.18) (39).

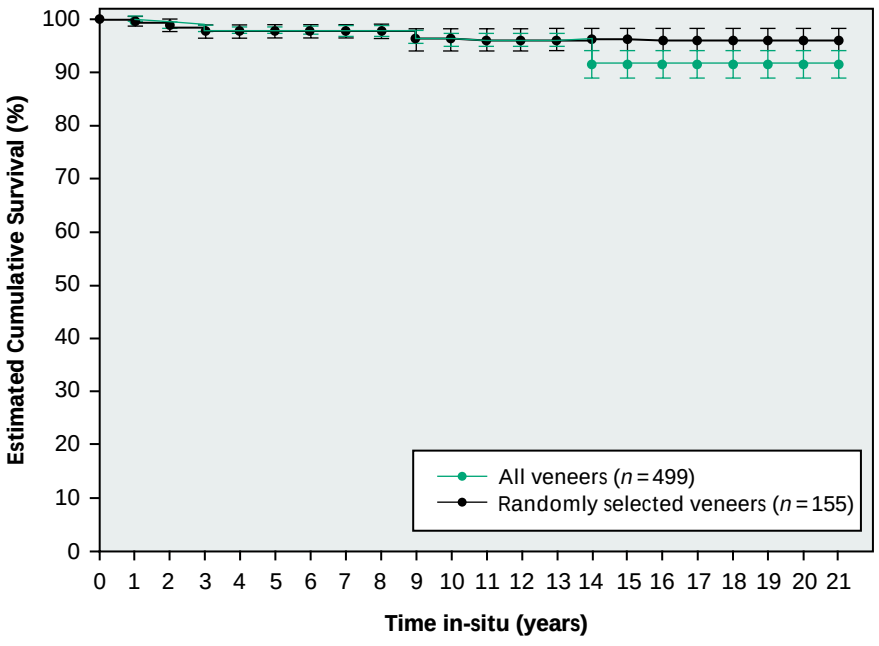
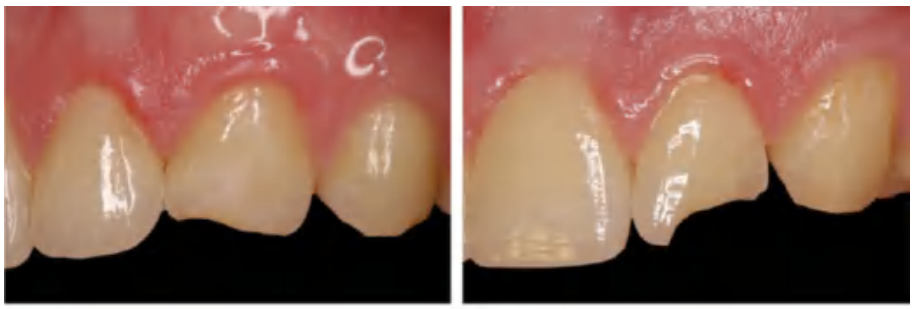



**Fig. 30.15** 21-year Kaplan Meier survival curve of feldspathic porcelain veneers of 96 ± 2% (*n*=499); and of 91 ± 2% (*n*=155), when one veneer was randomly selected in each patient to account for clustering.

**Fig. 30.16** Clinical application of porcelain veneers. A, B. Microdontia, 4 years post-op. C, D. Erosion, 10 years post-op. E, F. Replacement of composite restorations, 12 years post-op. G, H. Hypomineralization, 18 years post-op.

**Table 30.1** Modes of porcelain failure, and reasons failure may occur.

| Mode of failure | Reason failure may occur |
|---|---|
| Esthetic failure | Marginal staining<br>Veneer shade discrepancy with changing shade of adjacent teeth over time<br>Gingival recession |
| Biologic changes | Caries |
| Mechanical failure | Porcelain fracture secondary to occlusal loads or extrinsic trauma |
| Compromised supporting structure resulting in inadequate bonding substrate | Porcelain fracture<br>Loss of retention<br>Marginal staining<br>Caries |
| Patient associated factors | Dissatisfaction |
| Operator associated factors | Inadequate clinical technique directly contributing to failure |



**A** Mechanical failure



**B** Mechanical failure

**C** Esthetic failure, before veneer removal

**D** Esthetic failure, after veneer removal

**E** Biologic failure

**F** Operator assisted failure, before cement removal

**G** Operator assisted failure, after cement removal

**Fig. 30.17** Examples of failed veneers, and reasons for failure. A. Mechanical failure: Porcelain fracture secondary to parafunction, 3 years post-op. B. Mechanical failure: Porcelain fracture secondary to acute trauma, 5 years post-op. C, D. Esthetic failure: Discoloration secondary to marginal leakage and staining of retained glass ionomer restorations, before and after veneer removal respectively, 10 years post op. E. Biologic failure: Caries subsequent to poor veneer adaption to the underlying teeth, 8 years post-op. F, G. Operator assisted failure: Significant marginal overhangs secondary to an inadequate bonding technique, before and after cement removal respectively, 2 years post op.

## Essentials

Overall, porcelain veneers provide an excellent restorative solution for teeth that have been traumatized, damaged or affected by caries; and for patients wishing to disguise areas of concerns. They are conservative, esthetic and with attention to clinical detail they can provide excellent service over time with appropriate case selection.

Porcelain veneers restore labial, incisal and part of interproximal tooth surfaces.

They allow tooth discoloration, malalignments and unesthetic contours to be disguised. They rehabilitate teeth that have lost structure through trauma, caries, erosion, abrasion and parafunction.

Veneers can be placed on permanent teeth from post-adolescent years. Veneers should not be placed immediately following trauma.

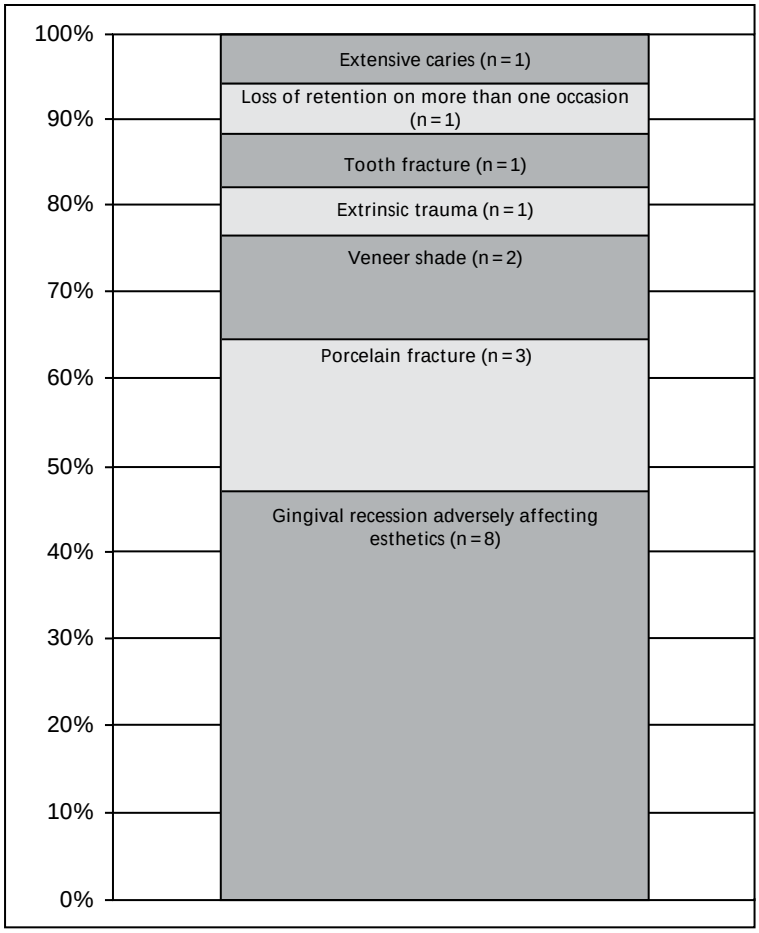



**Fig. 30.18** Reasons feldspathic porcelain veneers failed. Seventeen failed veneers out of 499 veneers completed, in an up to 21-year prospective cohort study.

Many ceramics are apparently suitable for veneers, but not all have proven outcomes. Feldspathic porcelain and e.max lithium disilicate are commonly used materials. Veneer materials must be etchable to facilitate retention given the limited resistance form inherent with veneer preparations.

Veneer procedures are technique sensitive, requiring attention to detail. It is recommended that at least 80% of the prepared tooth surface be in enamel. Outcomes are directly related to the amount of remaining prepared enamel. Unprepared aprismatic enamel is a poor bonding substrate. Veneer margins have a chamfer design. Incisal reduction may be 1 mm to 2 mm. Palatal overlap to improve resistance form is preferred. Labial reduction is minimal, of 0.5 mm to 0.7 mm. Porcelain veneer preparations finish equi- or supra-gingivally.

Addition polyvinyl siloxane impression materials are preferred. Impression procedures may involve a single or double cord technique.

Veneers are translucent, and their color is influenced by the shade of the underlying substrate. The bonding cement can affect the color of the final veneers. Retraction cords are not recommended during bonding.

Veneers should be prepared with hydrofluoric acid prior to bonding. Tooth structure should be prepared with an acid etch technique prior to bonding. Ineffective preparation results in an inadequate bonding strength.

Outcomes of feldspathic porcelain veneers over 21 years can be over 95%. Outcomes of e.max lithium disilicate veneers remain unknown, with outcome data limited. Reported outcomes of different veneers vary, and can appear to be contradictory. Failure rates increase when less enamel is present, loads are high and the intraoral environment is acidic.

In higher risk patients and environments, ECSs over 5 years can be lower than 75%. In lower risk patients and environments, ECSs over 10 years can be higher than 90%.

If insufficient enamel is present, options such as 3/4 or full crowns should be considered.

Patients should have a tailored maintenance program.

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# 31

# Resin-Bonded Bridges and Conventional Bridges in the Anterior Region

K. Gotfredsen & B. Pjetursson

## Indications

When a tooth or teeth are lost due to trauma and an implant-supported reconstruction is for one reason or another not indicated, a tooth supported fixed dental prosthesis (FDP) is a good option for patients with suitable abutment teeth.

Regarding tooth supported FDPs, resin-bonded fixed dental prostheses (RBFDPs), synonymous with resin-bonded bridges (RBBs), constitute a minimal invasive approach in the traumatized dentition compared to conventional tooth supported FDPs (Table 31.1). RBBs can be used either as an interim or a definitive treatment, which can be useful following dental trauma. Moreover, it has several advantages compared with conventional tooth supported FDPs. The amount of healthy tooth substance needed to be removed in order to create space for the material and retention is significantly less, and so is the risk of having pulp necrosis and apical pathology caused by preparation. Furthermore, the RBBs would normally cost significantly less than tooth supported conventional FDPs and implant-supported restorations (1). Patient satisfaction has been reported to be high for RBBs (2), and the same oral health related quality of life can be expected when replacing single tooth space with RBB compared with implant-supported restoration (3).

Thus, RBBs are indicated in the following situations:

- Implant-supported crowns are contraindicated due to cost, anatomical situations, patient related factors or other issues.
- Suitable abutment teeth are available (not-mobile, sufficient enamel for adequate bonding).
- Interim treatment is desired due to the patients age or other issues.
- The occlusal forces and moments can be controlled or are estimated to be limited.

For trauma in the anterior region where the remaining potential abutment teeth have crown fractures, root fractures or are discolored teeth, RBBs are not the treatment of choice. RBBs are also contraindicated if the abutment teeth are in a need for a crown due to large restorations or insufficient enamel for adequate bonding. Furthermore, mobile abutment teeth or teeth under excessive load (e.g. bruxism and parafunctions) are also risk factors for RBBs.

## Different types of RBBs

When teeth are lost due to trauma, application of a direct resin-bonded solution at the first appointment may be necessary for the patient. Direct fixed RBBs in acute situations can be made by using the natural crown of the lost tooth as a pontic or by using a custom acrylic tooth or an all-composite tooth bonded directly to the neighbouring teeth. The reconstruction can also be strengthened by using metal wires or glass fibers.

Although several designs, metals and surface treatments have been proposed for metal-based RBBs, it is reasonable to distinct between the early Rochette bridges (Fig. 31.1), characterized with non-preparation of abutment teeth and perforations in the metal frame and Maryland bridges, characterized with preparation of abutment teeth and non-perforated, acid-etched metal substructure (Fig. 31.2a,b). Both metal-based RBBs were usually fabricated in a Co-Cr-alloy with acrylic or ceramic veneering and cemented with a chemically active resin-cement.

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**Table 31.1** Advantages and disadvantages of resin-bonded fixed dental prostheses (RBFDPs).

| RBFDP | Advantages | Disadvantages |
|---|---|---|
| Metal-based | Long-term clinical data<br>Increased retention features<br>Cantilever design<br>Less extension of connection<br>Good access for oral hygiene<br>Thin abutment wing<br>Simple repair/re-bonding | Metal discolorations<br>Metal unpopular<br>De-bonding |
| All-ceramic | Esthetics<br>Biocompatibility | Few long-term clinical data<br>Bulky<br>Retentive features restricted<br>Increased risk of fractures<br>De-bonding<br>Zirconia adhesion to resin-cement |
| Fiber-reinforced | Adhesion to resin-cement<br>Repair possibilities | Few long-term clinical data<br>Increased risk of fractures<br>De-bonding<br>Bulky |

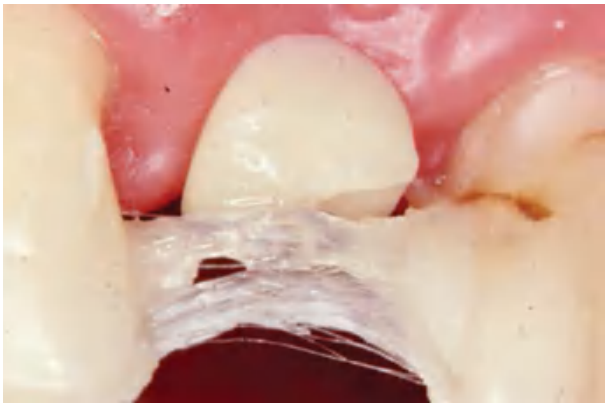

**Fig. 31.1** A direct constructed resin-bonded replacement for a lost central incisor by using all-composite reinforced with everStick® fibers.

The all-ceramic RBBs have been fabricated with framework out of glass-infiltrated ceramic, glass reinforced ceramic and densely sintered – zirconia (Figs 31.3 and 31.4 and Table 31.1).

When RBBs are used as a temporary restoration only minimal preparation of abutment teeth is performed. The main reason for the preparation is to create space and to increase retention. Several studies have found a positive effect of abutment tooth preparation with different preparation features (4). Thus, the extension of the preparation should be assessed against the need for space and retention including the time-period the RBB is supposed to serve, before a definitive treatment will be performed. It has to be taken into account that no treatment is definitive, when young adults are treated after dental trauma. Thus, the young age of the patient along with prospective considerations have to be included in the treatment plan.

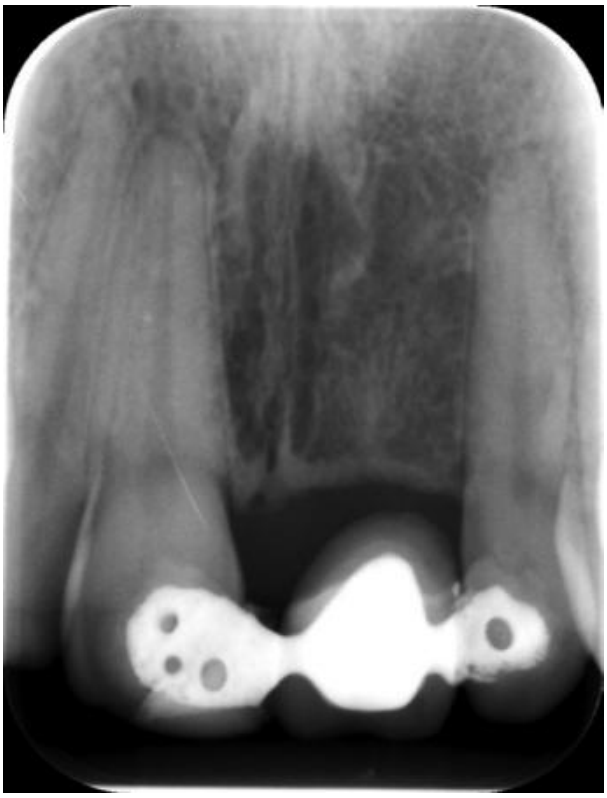

**Fig. 31.2** Radiograph of a temporary Rochette bridge.

Beside the preparation technique, the number of missing teeth to be replaced and their position in the oral cavity has a great influence on the prognoses of the RBB (5). Thus, if more than one tooth must be replaced in the anterior region of the upper jaw, conventional tooth supported FDPs or implant-supported reconstructions could be preferred. Although, very few clinical reports exists with

RBBs with four and more units, there is limited data indicating that in the anterior area of the lower jaw two or even three teeth can be replaced with RBBs. When only one tooth is missing, the area in the oral cavity as well as the number of retainers seems to be of great importance for the outcome (6, 7). Number of studies, have demonstrated that for replacements of lateral incisors, one abutment retainer is sufficient (8, 9). A recent systematic review on RBBs concluded that the survival rate was higher for RBBs inserted in the anterior area of the oral cavity compared with posterior RBBs and that one retainer RBBs had a significantly higher survival rate and a lower de-bonding rate compared with RBBs retained by two or more retainers (10).

A RBB can be constructed as a metal-based reconstruction (Fig. 31.2A,B) or a ceramic-based reconstruction (Figs 31.3 and 31. 4). In both situations conventional, CAD-CAM or combination of the two techniques can be used. The 1-retainer cantilever design has several advantages. It simplifies the oral hygiene compared with 2-retainers pontic design RBB and does not require special oral cleaning equipment. Normal dental floss can be used additional to tooth brushing. Less tooth substance is removed compared with RBBs with more retainer teeth and the "gray-out" caused by the "shine-through" of the lingual or palatal metal retainer can in many cases be avoided on anterior teeth with high translucency (Fig. 31.3). For 3-unit RBB a greater tensile force will be present at the interface between cemented retainers at the second retainer tooth, when the first retainer tooth is loaded. This tensile force will be eliminated by designing a RBB with only one retainer.

Today most RBBs are constructed by the use of CAD-CAM techniques, although these techniques imply more difficulties in including well-fitting retention elements as groves (Fig. 31.5) and small boxes, especially if the reconstruction is milled based on a *Surface Tessellation Language* (STL) data set. The preparations are performed without macro-mechanical retention elements and only indentations and rounded irregularities can be accepted (Fig. 31.6). Thus, the principles already known from adhesive dentistry, based on micro-mechanical anchorage, are normally used. A digital impression will be taken and the preparation can be evaluated directly at the computer screen and correction performed (Fig. 31.7). A SLA-model can be created (Fig. 31.8) for further analyses, but is not needed to design an all-ceramic RBB (Fig. 31.9–31.11).

## Dental ceramic for RBBs and tooth supported FDPs

A simple, frequently used clinical classification of dental ceramics is in feldspathic porcelain, glass ceramic and oxide ceramic (11). When the indication area is restricted to FDPs in the anterior region, only glass ceramic and oxide ceramic-based systems can be recommended and porcelain is only

**Fig. 31.3A** A 3-unit Maryland bridge replacing a right central incisor.

**Fig. 31.3B** A 2-unit Maryland bridge replacing a right lateral incisor.

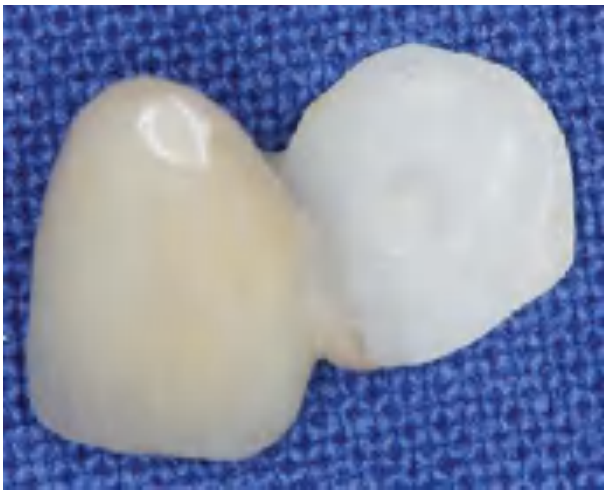

**Fig. 31.4A** A 2-unit zirconia RBFDP veneered with porcelain.

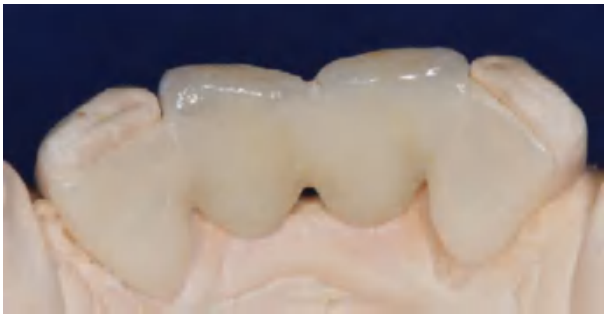

**Fig. 31.4B** A 4-unit zirconia RBFDP veneered with porcelain.

**Fig. 31.5** Extension of the preparation (light blue) in an abutment tooth for a metal-based RBFDP with retention elements (redish).

**Fig. 31.6** Preparation for an all-ceramic RBFDP without macromechanical retention elements beside the rounded indentation in the cingulum area.

**Fig. 31.7** A picture created of a digital impression of an abutment tooth (a right central incisor) for a 2-unit all ceramic RBFDP.

**Fig. 31.8** A SLA model created based on a digital scan.

**Fig. 31.9** Two 2-unit zirconia-based RBFDPs constructed based on digital impression. The inside of the framework is coated with porcelain to improve the chemical bonding to the resin-cement.

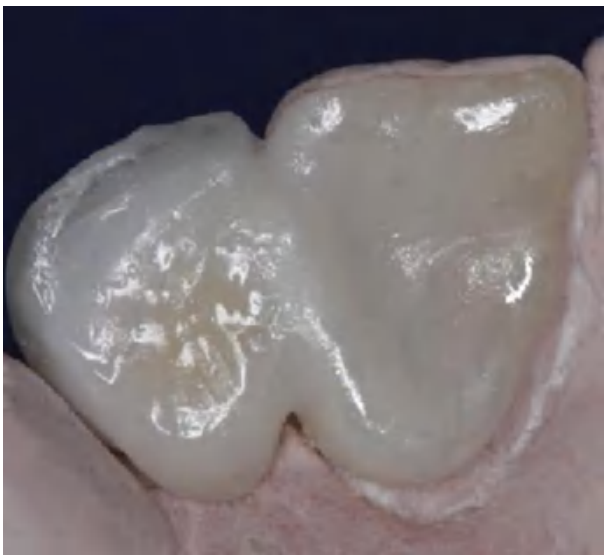

**Fig. 31.10** The two 2-unit zirconia-based RBFDPs seen from the palatinal aspect placed at a stone cast model.

**Fig. 31.11** The 2-unit zirconia-based RBFDP in situ.

**Fig. 31.12** A zirconia FDP veneered with porcelain.

used for veneering in the layering technique to obtain good esthetic results (11).

The flexural strength and toughness are significantly higher for glass ceramic compared to porcelain, and can be built up into thicker layers than porcelain. The optical feasibility of glass ceramic offers almost the same good light properties as porcelain, but it is not built up in layers and the special effects in the ceramic has to be built in, using a staining technique or by using cut-back technique, where a thin layer of porcelain is fired on the glass ceramic to replace the reduction. As the glass-ceramic can be bonded with adhesive technique, the cement and the handling of the cement is important for the outcome. Thus, dry conditions must be ensured for optimal bonding. A number of publications have demonstrated that monolithic glass ceramics, based on lithium-disilicate, can be used for tooth supported FDPs up to 3 units in the anterior and premolar region (12). Also 2-unit FDPs have demonstrated a good prognosis in a recent study (9). However, the scientific evidence for using lithium-disilicate FDPs is still limited.

Within the oxide ceramics, ytria-stabilized, tetragonal zirconia polycrystal (Y-TCP) has become more dominant recently. The flexural strength has proven to be better as most other ceramics and the toughness of the zirconia result in crack growth inhibiting properties. Thus, the risk of fracture for tooth supported FDPs produced by monolithic zirconia is reduced compared with other commercial available ceramics. Clinical studies have demonstrated quite good results for tooth supported FDPs in zirconia over a short period of time, but it has been questioned if the support of the veneering porcelain, when the normal layering technique is used, is sufficient (Figs 31.11 and 31.12) (13). Thus, a high frequency of fractures or chipping of the veneering materials have been reported for FDP using zirconia framework veneered with porcelain (14). Furthermore, different techniques can be used for pressing oxide ceramics, and this may influence the quality of the zirconia. Further developments with coloured Y-TZP in order to produce a more tooth coloured substructure than the original white zirconia has been accomplished, but the colouring makes the material more opaque than the uncoloured ceramic. The opaque appearance of the compressed ceramic granules has been an esthetic limitation. Recently more translucent zirconia has been fabricated, but until now only limited reports is available. However, it must be kept in mind that the tensile strength of the more translucent zirconia is less than the tensile strength of the original opaque zirconia.

Another challenge with the zirconia-based FDPs is the bond of the adhesive cement to the oxide ceramic (13). Different methods as grit-blasting, heat treatment, micro layering with porcelain and treatment with glass additives have been used in various combinations to treat the inner part of the zirconia but without any reported clinical success (14). This implies that zirconia might not be the ideal material for RBFDPs. Studies where glass additives have been used to increase the bonding strength to the adhesive cement have shown that the fit of the RBB is compromised (Fig. 31.10). However, a recent systematic review analyzing the survival and de-bonding rate of RBBs concluded that the highest 5-year survival rate was seen for zirconia-based RBBs, but the de-bonding rate was rather high (15).

## Disadvantages with tooth supported FDPs

Conventional tooth supported FDP will rarely be the first treatment choice after trauma in the anterior region because it is very difficult to obtain successful results from an esthetic and biological point of view. The esthetical challenges are great when e.g. a single central incisor has to be replaced (Figs 31.13 and 31.14). Beside separate factors such as colour, surface texture, incisal edges, contact placement, position, rotation and gingival contours, the symmetry and proportion is essential in the dento-facial composition. It is difficult to obtain the symmetry and especially the colour differences. The translucency is also frequently a problem (Fig. 31.14). Furthermore, enough space for the framework and the veneering material is needed to obtain sufficient esthetic results and this may compromise the biology. In young patients from 10-19 years old, the combined thickness of the dentin and enamel facial on a central incisor is around 1.8 mm compared with an average thickness of 3.1 mm at age of 60 years. Furthermore, another study concluded that if the thickness of the remaining dentin goes below 1 mm after tooth preparation a pulp trauma can be demonstrated histologically (16). Thus, loss of pulp vitality is a frequently observed problem, especially in young patients, when

**Fig. 31.13** A conventional FDP replacing 11 after a trauma. The symmetry is difficult to obtain and a soft tissue graft could be performed in region 11 to improve the esthetic outcome.

**Fig. 31.14** An apical infection after preparation and cementation of a conventional FDP for replacing a right central incisor.

healthy intact abutment teeth are prepared for conventional tooth supported FDPs (Fig. 31.15). The risk of devitalization is reported with different frequency in the literature (Table 31.2) (17, 18). For cantilever tooth supported FDPs, the prevalence of abutment pulp vitality loss, has been reported higher than for FDPs without cantilevers. Furthermore, it can be hypothesized that this is especially true for young people with teeth without fillings, no or little tertiary dentin formation or calcification formed and with an extended size of the pulp. Thus, the distance to the pulp and the risk of loss of pulp vitality is increased compared to older patients with earlier prepared teeth and reduced area of the pulp caused of reactionary dentin formation by odontoblasts.

## Prognosis of tooth supported FDPs, RBBs and implant-supported single crowns (ISC)

No randomized controlled trials (RCTs) have been performed comparing conventional tooth supported FDPs with RBBs, conventional tooth supported FDPs with ISC or RBBs with implant-supported single crowns (SCs), but a number

**Table 31.2** Studies of the prevalence of endodontic treatment of abutment teeth for FDPs.

| Authors | Year | Follow-up time | Endodontic treatment |
|---|---|---|---|
| Bergenholtz & Nyman (17) | 1984 | 5 years | 8.2% |
| Karlsson (15) | 1986 | 10 years | 10% |
| Palmquist & Schwartz (16) | 1993 | 18–23 years | 15% |
| Pjetursson (meta-analyses) (18) | 2007 | 5 years | 6.1% |

**Fig. 31.15** An implant-supported crown for replacement of a right central incisor.

of meta-analyses have examined the three treatment options (20–22). A series of systematic reviews utilizing the same inclusion criteria and methodology evaluating the outcomes of different types of reconstructions has been published in the recent years. The objective of those papers has been to obtain the long-term survival rate and to evaluate the incidence of technical, biological and esthetic complications over an observation period of 5 and 10 years.

For RRBs, 29 studies reporting on 27 patient cohort fulfilled the relatively simple inclusion criteria's of having a mean follow-up time of at least 5 years and that the patients had been clinically examined. Hence, the results were not based exclusively on patient records, interviews or insurance databases. Meta-analysis revealed, that of the originally 2300 RBBs placed, 251 were known to be totally lost or had de-bonded more than once and therefore were classified as a failure in the publication. The included studies were divided according to the mean observation time. A group of 18 studies reported on 1755 RBBs with a mean follow-up time around 5 years and a group of five studies reported on 545 RBBs with a follow-up time around 10 years. The estimated 5-year survival rate for RBBs was 91.4% (95% C.I.: 86.7–94.4%) (Table 31.3) and the 10-year survival rate was estimated to be 82.9% (95% C.I.: 73.2–89.3%) (Table 31.4). The studies were also divided according to the material utilized. For metal-ceramic RBBs, based on eight studies with 977 reconstructions, the estimated 5-year survival rate was 91.3%, for metal-acrylic RBBs based on one study with 203 reconstructions the estimated 5-year survival rate was 88.9%, for RBBs with glass-infiltrated ceramic as framework material based on one study with 38 reconstructions the estimated 5-year survival rate was 93.4% (95% C.I.: 85.3–97.8%), for RBBs with reinforced glass ceramic framework based also on one study with 49 reconstructions the estimated 5-year survival rate was 95.3% (95% C.I.: 84.4–99.4%), three studies with 68 RBBs with zirconia as framework material had an estimated a 5-year survival rate of 100% (95% C.I.: 94.5–100%) and finally three studies reporting on 267 RBBs with fiber-reinforced composite as framework material estimated the 5-year survival rate to be 92.8% (95% C.I.: 47.9%-99.2%). For fiber-reinforced composite RBBs, the results from the three included studies varied significantly. Two of the studies reported a 5-year survival rate of 99.4% and 100%, respectively. The third study, however, reported a 5-year survival rate of only 72.9% (Table 31.3). The survival rate of RBBs was also analyzed by the number of retainers utilized. A group of 350 RBBs with one retainer was compared with a group of 1376 RBBs with two or more retainers. The 1-retainer group had significantly higher survival rate compared with the 2-retainers group. The survival rate of RBBs was also analyzed regarding the RBB location in the oral cavity. The position of the pontic (missing tooth) was used for the classification. The survival failure rate of RBBs inserted in the anterior area was higher, compared with the survival rate of RBBs placed in the posterior part of the mouth.

For *conventional tooth supported FDPs*, fifteen studies meeting the inclusion criteria's provided data on the survival of the reconstructions. A group of seven studies with a total of 2088 FDPs and a mean follow-up time around 5 years and a group of eight studies with a total of 1218 FDPs and a mean follow-up time exceeding 10 years. In the former group, the estimated 5-year survival rate was 93.8% (95% C.I.: 87.9–96.9%) (Table 31.3) and for the latter group the estimated 10-year survival rate was 89.2% (95% C.I.: 76.1–95.3%) (Table 31.4). A recent systematic review and meta-analysis based on 40 publications compared the prognoses of 1796 metal-ceramic and 1110 all-ceramic tooth supported FDPs (23). The analysis evaluated the survival rate of tooth supported FDPs made out of different materials. The included studies indicated an estimated 5-year survival rate of metal-ceramic FDPs of 94.4% (95% C.I.: 91.2–96.5%), an estimated survival rate of reinforced glass ceramic FDPs of 89.1% (95% C.I.: 80.4–94.0%), an estimated survival rate of glass-infiltrated alumina FDPs of 86.2% (95% C.I.: 69.3–94.2%) and an estimated survival rate of densely sintered zirconia FDPs of 90.4% (95% C.I.: 84.8–94.0%) (Table 31.5). Even though the survival rate of all-ceramic FDPs was lower than for

**Table 31.3** Survival rate of different types of reconstructions after an observation period of 5 years.

| Type of reconstruction | Number of publications | Total number of reconstruction | 5 year survival rate (95% C.I.) |
|---|---|---|---|
| Implant-supported single crowns | 20 | 1385 | 96.3% (94.2%–97.6%) |
| Conventional tooth supported FDPs | 7 | 2088 | 93.8% (87.9%–96.9%) |
| Resin-bonded bridges | 18 | 1755 | 91.4% (86.7%–94.4%) |

**Table 31.4** Survival rate of different types of reconstructions after an observation period of 10 years.

| Type of reconstruction | Number of publications | Total number of reconstruction | 10 year survival rate (95% C.I.) |
|---|---|---|---|
| Implant-supported single crowns | 7 | 268 | 89.4% (82.8%–93.6%) |
| Conventional tooth supported FDPs | 5 | 874 | 89.2% (76.1%–95.3%) |
| Resin-bonded bridges | 5 | 545 | 82.9% (73.2%–89.3%) |

Pjetursson et al. 2012 (25), Jung et al. 2012 (21), Thoma et al. 2016 (in press).

**Table 31.5** Survival rate of metal-based and all-ceramic FDPs.

| | 5-years% | 95%CI |
|---|---|---|
| Metal-ceramic FDPs | 94.4 | 91.2-96.5 |
| Reinforced glass-ceramic FDPs | 89.1 | 80.4-94.0 |
| Glass-infiltrated alumina FDPs | 86.2 | 69.3-94.2 |
| Densely sintered zirconia FDPs | 90.4 | 84.8-94.0 |

Reprinted with permission from (24) and Thoma et al. 2016 (in press).

metal-ceramic FDPs, the differences did only reach statistical significance for the glass-infiltrated alumina FDPs (23). The reinforced glass ceramics had a higher incidence of framework fractures and the zirconia-based FDPs had a significantly higher incidence of ceramic fractures, loss of retention and secondary caries (23).

For *implant-supported single crowns (SCs)*, 30 of the studies that met the inclusion criteria's provided information on the survival of the crowns. A group of twenty studies with a total of 1385 ISC (SCs) estimated a 5-year survival rate of 96.3% (95% C.I.: 94.2–97.6%) (Table 31.3). Twenty-eight SCs were lost because the implants were lost, while in 25 cases only the reconstructions failed. Furthermore, a group of seven studies reporting on 268 implant-supported SCs with a mean follow-up around 10 years estimated a 10-year survival rate of 89.4% (95% C.I.: 82.8–93.6%) (Table 31.4). Regarding survival rates there was neither significant difference between cemented and screw-retained crowns nor metal-ceramic and all-ceramic crowns.

Based on the meta-analyses, it can be concluded that the survival rate of RBBs is inferior to the survival rate of implant-supported SCs after 5 years (Table 31.3). On the other hand, the meta-analysis and single studies indicate that RBBs have survival rate that are comparable with survival rates for conventional tooth supported FDPs (24). When the outcome variable is cost-effectiveness, RBBs was reported to be even better than tooth supported FDPs (1). When the oral health related quality of life (OHRQoL) was the primary outcome parameter, a case-control study by Lam and co-workers (3) demonstrated no difference between cantilevered RBBs and implant-supported SCs. In that particular study, the Oral Health Impact Profile (OHIP-49) was used to measure the OHRQoL. Although 6 out of the 49 questions are related to the esthetics, the OHIP-49 is not the preferred way to evaluate esthetic outcome. Thus, no studies have yet compared the esthetic outcome for the three discussed treatment modalities, RBBs, tooth supported FDPs and implant-supported SCs. Theoretically, an optimally placed implant would give the best opportunities for a good esthetic tooth-replacement, when the neighbouring teeth are healthy (Fig. 31.15). However, according to the meta-analysis of implant-supported SCs, 7.1% of the evaluated crowns demonstrated unacceptable esthetic outcomes. For RBBs esthetic evaluation was not reported, but only 0.3% of the reconstructions had to be re-done due to esthetic failures.

## Complications of FDPs, RBBs and ISC

The most frequent complication by RBBs was de-bonding of the reconstructions. The annual de-bonding rate was 3.3%, translating into a 5-year de-bonding rate of 15% (Table 31.6). The incidence of de-bonding was significantly dependent on the framework material utilized. For metal-ceramic RBBs, the annual de-bonding rate was 2.9%, for metal-acrylic RBBs it was 4.2%, for fiber-reinforced composite RBBs it was 1.7% and for zirconia framework RBBs it was 1.4%. However, for RBBs with glass-infiltrated and glass reinforced ceramic frameworks no de-bonding occurred. The de-bonding rate was also influenced by the position in the mouth. RBBs inserted in the posterior area of the mouth demonstrated a higher de-bonding rate than anterior positioned RBBs. Other technical complications reported for RBBs were material fractures and ceramic chipping. Over a 5 years observation period, 1.7% of the RBBs were lost due to material fractures (Table 31.6). This incidence was also dependent on the material. None of the RBBs made of metal-ceramic, zirconia and reinforced glass ceramic were lost due to material fractures. Studies on RBBs made of metal-resin, glass-infiltrated ceramic and fiber-reinforced composite, however, reported that significantly more RBBs were lost due to material fractures. Ceramic chipping was reported for 4.1% of the inserted RBBs over a 5 years observation period. Biological complications were relatively rare for RBBs. Hence, 0.8% of the reconstructions were lost due to periodontal disease, 0.2% was lost due to retainer tooth fracture and 1.7% of the retainers developed secondary caries over a 5 years observation period (Table 31.6).

The most frequent complication by conventional tooth supported FDPs were loss of abutment tooth vitality (pulp necrosis), occurring in 6.1% of the originally vital abutments. The second most common complication was dental caries, detected at the crown margins by 4.8% of the abutment teeth. However, only 1.6% of the tooth supported FDPs were lost due to secondary caries and 0.4% were lost due to recurrent periodontal diseases. The most frequent technical complications were loss of retention and fracture of the veneering material (Table 31.6).

For implant-supported SCs, technical complications were more frequent than biological complications. The most frequent complication was abutment or screw loosening, followed by loss of retention and ceramic fractures. Soft tissue complications were reported for 8.5% of the implants and for 5.2% of the implants a significant bone loss of more than 2 mm was reported (Table 31.6).

## Essentials

Resin-bonded bridges (RBBs), constitute a minimal invasive approach in the traumatized dentition compared to conventional tooth-supported FDPs. RBBs can be fabricated with a framework of metal or all-ceramic and can be supported by

**Table 31.6** Incidence of biological and technical complications by different types of reconstructions over an observation period of 5 years.

| Complication | Tooth supported FDPs | Resin-bonded bridges | Implant-supported single crowns |
|---|---|---|---|
| Caries on abutments | 4.8% | 1.7% | n.a. |
| Reconstruction lost due to caries | 1.6% | n.r. | n.a. |
| Reconstruction lost due to periodontal diseases | 0.4% | 0.8% | n.r. |
| Loss of retention | 3.3% | 15.0% | 4.1% |
| Loss of abutment tooth vitality | 6.1% | n.r. | n.a. |
| Reconstruction lost due to abutment tooth fracture | 1.0% | 0.2% | n.a. |
| Soft tissue complication | n.r. | n.r. | 7.1% |
| Marginal bone loss > 2 mm | n.r. | n.r. | 5.2% |
| Reconstruction remade due to esthetic failures | n.r. | 0.3% | n.r. |
| Compromised esthetic outcome | n.r. | n.r. | 7.1% |
| Implant fracture | n.a. | n.a. | 0.2% |
| Fracture of abutment or occlusal screw | n.a. | n.a. | 0.4% |
| Loosening of abutment or occlusal screw | n.a. | n.a. | 8.8% |
| Loss of the access hole filling by screw-retained crowns | n.a. | n.a. | 0% |
| Fracture or chipping of ceramic | 1.6% | 4.1% | 3.5% |
| Reconstruction lost due to framework fracture | n.r. | 1.7% | 1.3% |

n.r. = not reported, n.a. = not applicable.

one, two or more abutment teeth. Conventional tooth supported FDPs will rarely be the first treatment choice after trauma in the anterior region, because it is very difficult to obtain successful results from an esthetic and biological point of view. Thus, the risk of abutment tooth devitalization for conventional FDPs is reported with frequencies between 6 and 15% over a 5-year period, whereas the risk is very low for RBBs, where the preparation is mainly done in the enamel.

A series of systematic reviews utilizing the same inclusion criteria's and methodology have evaluated the outcome of RBBs and FDPs. The estimated 5-year survival rate for RBBs was 91.4% and the 10-year survival rate was estimated to be 82.9%. The similar frequencies for FDPS were 93.8% and 89.2%, respectively. This was comparable with ISC (SCs) with an estimated 5-year survival rate of 96.3% and an estimated 10-year survival rate of 89.4%. When the outcome variable was changed from survival rate to cost-effectiveness, RBBs was reported to be even better than tooth supported FDPs. When an OHRQoL variable was the primary outcome parameter no difference between cantilevered RBBs and implant-supported SCs was reported in a case-control study. In that particular study, the Oral Health Impact Profile (OHIP-49) was used to measure the OHRQoL. Although 6 out of the 49 questions are related to the esthetics, the OHIP-49 is not the preferred way to evaluate esthetic outcome. Thus, no studies have yet compared the esthetic outcome for the three discussed treatment modalities, RBBs, tooth supported FDPs and implant-supported SCs.

The most frequent complication by RBBs was de-bonding of the reconstructions. The most frequent complication by conventional tooth supported FDPs were loss of abutment tooth vitality (pulp necrosis), whereas for implant-supported SCs it was abutment or screw loosening, followed by loss of retention ceramic fractures and soft tissue complications.

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# 32

# Dentoalveolar Ankylosis, Decoronation and Alveolar Bone Preservation

B. Malmgren, O. Malmgren & L. Andersson

## Introduction

Dentoalveolar ankylosis is seen when the periodontal ligament has been severely damaged (1–11). Such severe damage is seen mainly after tooth avulsion, seen in 0.5–3% of all traumatic dental injuries (TDI), and intrusive luxations, seen in 0.5–1.9% (12–17). The consequence of these injuries is a fusion of the alveolar bone and root. As a result of this fusion an ankylosed root is continuously resorbed and gradually replaced by bone, until the entire root is resorbed. The condition is progressive, and the rate of this resorption seems to vary with age. To date there is no ways of arresting or reversing the process. Providing no other changes supervene, the ankylosed tooth can be retained until the crown falls off or is removed by forceps. However, ankylosis in a young growing patient is associated with inhibition of growth of the alveolar process. In recent decades, clinical experience and research has given us increased insight and provided us with methods to manage such situations. Paradigm shifts from saving the tooth to preserving the alveolar bone is seen. This chapter is summarizing the most important insight and today's knowledge on ankylosis and how to manage such situations clinically.

## The normal and damaged periodontal ligament

The normal periodontal ligament (PDL) is not only attaching the root to its socket by fibers. It is also an effective barrier which protects the PDL from bony ingrowth and ankylosis of the root under normal conditions.

Regarding the periodontal space there is a fundamental difference between the tooth side and the bone side of the alveolar socket. On the tooth side, the cementoblasts are covering the root and on the bone side the osteoblasts are covering the bone. Osteoblasts and osteoclasts can react to variations in calcium level in the blood and hormones such as parathormone. A low calcium level may give signals to osteoclasts to start resorbing the bone resulting in release of calcium to the blood. Keeping the calcium level in the blood constant is a prerequisite for life and is given highest priority. Bone may also be resorbed and remodelled due to loading. So, the bone side of the alveolar socket, like the other bone tissue in the body is undergoing constant bone remodelling. In contrast, the tooth side of the socket is not being influenced by variations in the calcium level or hormones in the blood. This is the reason why teeth under normal conditions are not resorbed or remodelled in contrast to the bone. A normal healthy PDL is acting as a protective barrier.

Moreover, in the normal physiologic situation it seems as if bone is kept on distance from the root (18). Hence, it may be appropriate to regard the PDL as a physiologic membrane protecting the root from the remodelling bone. The epithelial cells in the PDL have been suggested to play an important role in keeping the bone on distance (18–21). It seems as if epithelium and bone are not compatible tissues. Nowhere in the body is epithelium in close connection with the bone, unless there is resorption of bone taking place e.g. eruption of tooth where the follicular sac of the eruption tooth is in close contact with bone, or an epithelial cyst in contact with bone, in both cases the bone is yielding way to the epithelium. In the same way, the epithelium in the normal non-damaged periodontal ligament is probably contributing to keep the bone on distance, preventing bony ingrowth in the periodontal ligament under normal conditions. If this was not the case, the bony ingrowth would be seen in the PDL and teeth would be fused with bone and remodelled and gradually be replaced by bone. This is exactly what happens

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when the PDL is damaged by an avulsed tooth replanted after long dry extra oral storage time when the PDL cells are necrotic. Similar bony fusion is seen in other parts of the body, when other protective tissues in the body are damaged due to trauma or infection e.g. in temporomandibular joint ankylosis.

## Development of dentoalveolar ankylosis

After severe trauma to the PDL, e.g., drying during extra oral period, the bony ingrowth in the PDL results in a fusion between the tooth and the bone within a week after trauma (22). This area of ankylosis will increase with increasing time after replantation, gradually resorbing cementum and dentin and eventually resulting in the PDL will be replaced by bone (22) (Fig. 32.1). This new bone is covered by osteoblasts in continuity with the endosteal cells outlining the marrow spaces of the alveolar bone (22). It has been shown that root resorption associated with dentoalveolar ankylosis is a bone remodelling process initiated by endosteal osteoblasts and is thus a hormonally regulated process (10, 22).

This osseous replacement is progressive and goes with a higher rate in young individuals than old individuals like all bone remodelling in the body (11) (Fig. 32.2). Replacement resorption is not associated or driven by infection but instead by the normal bone remodelling process.

## Clinical diagnosis of ankylosis

An ankylosed tooth changes its percussion sound to a bone-like, metallic, high pitch percussion sound. This can be heard when tapping the tooth with e.g. handle of a dental instrument and comparing the normal percussion sound of the adjacent non-damaged tooth. Ankylosis is diagnosed by percussion test and mobility test when more than 20% of its root surface is affected by ankylosis (8). In the early stages, radiographic diagnosis for ankylosis is of limited value. However later, replacement resorption by bone can be seen in radiographs (1, 8, 11, 23). Replacement resorption can be seen on radiographs as resorption cavities with ingrowth of bone (Fig. 32.2B).

## Methods to prevent ankylosis

Functional stimulation cannot prevent ankylosis in replanted teeth subjected to drying before replantation (7, 9). Interpositioning of various tissues in the PDL have been tried but all failed to prevent ankylosis (24, 25). Topical treatment of various agents prior to replantation has not been successful to avoid ankylosis (for review see Chapter 8).

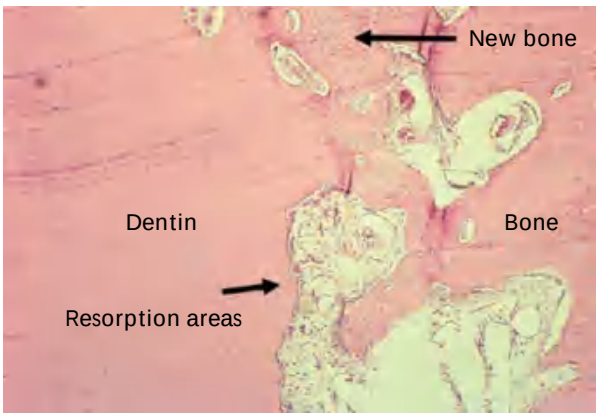



**Fig. 32.1 Histological picture of ankylosis and replacement resorption**
The replanted monkey incisor was examined 4 months after replantation. In the left side of the picture remaining root dentin can be seen, and in the right side of the picture the alveolar bone. The periodontal ligament has been almost totally replaced by bone in the upper part of the picture. In the lower part of the picture ongoing resorption by osteoclasts can be seen in the dentin. New bone formation is seen in the former periodontal space.

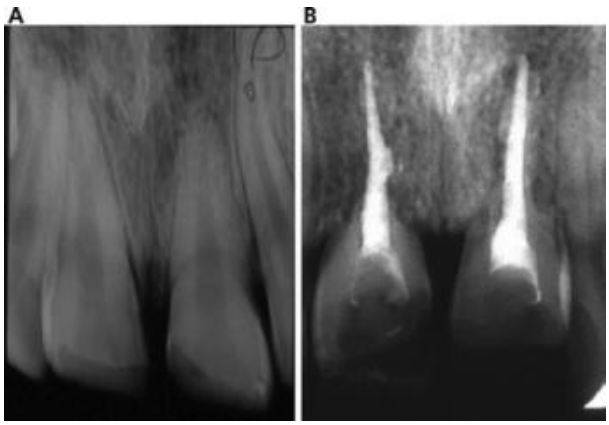



**Fig. 32.2 Progression of ankylosis**
Replanted upper central incisors. A. Immediately after replantation at 12 years of age. B. After two years.

## Progression of ankylosis

The rate of replacement resorption is age related (11, 68). In young individuals, in whom the bone remodelling process is high, an ankylosed tooth after delayed replantation may be completely resorbed within 4–5 years (Fig. 32.2), while in an old individual, where the bone remodelling is slow, the tooth can remain functional for a very long period, possibly throughout life (11). The replacement resorption is a non-infective osseous replacement driven by bone remodelling (11, 22).

## Ankylosis in adult patients

There is no treatment to stop the ankylotic tooth to be progressively replaced by bone. In an adult patient, where growth of the alveolar bone is completed the ankylosed

tooth can be left in place to be replaced by bone. Keeping an ankylosed tooth is a very good way to prevent the alveolar process to be resorbed, which would be the case if the tooth was extracted. For this reason, ankylotic roots should be left in the bone today for osseous replacement. And when the tooth has been completely resorbed by bone, other treatment e.g. installation of an implant can be performed.

## Ankylosis in young, still growing, patient

Most of the dental traumas occur to a large extent in the anterior region of young growing patients. This results in special complex problems, treatment considerations and timing (26). An ankylosed tooth, fused with the alveolar bone will inhibit vertical growth of the alveolar process in that site, while the surrounding teeth continue to grow vertically. This inhibition of growth results in a lagging behind of the ankylosed tooth ending up in a more superior position. This is called infraposition (infraocclusion) of the ankylotic tooth (Fig. 32.3). This is not only affecting the tooth but also other tissues in the area such as the alveolar bone and gingiva resulting in negative esthetic consequences.

## Infraposition (infraocclusion)

Dentoalveolar ankylosis is accompanied by an increasing infraposition (infraocclusion) of the affected tooth in children and adolescents, and to a less degree also in some adults (27, 28).

Orthodontic extrusion is not possible. An ankylosed tooth can be compared with an osseointegrated implant and cannot be moved. Such an attempt will only result in intrusion of the adjacent teeth (Fig. 32.4).

## Treatment options of infraposition

Six options have been suggested to deal with infraposition, some of them less suitable than others.

(1) *Composite build-up* is not recommended in growing individuals (Fig. 32.5) but may be a treatment option if the patient has passed his pubertal spurt and the infraposition is minimal. In adolescents when the alveolar growth is finished or almost finished, only a minor further infraocclusion can be expected. Then a build-up of the incisal edge is a more permanent option (Fig. 32.6).

(2) *Surgical repositioning* has been suggested when the ankylosed area is minimal. However, the long-term prognosis is poor, since the reduced marginal bone level is unchanged, and the root has a poor bone support. Furthermore, new ankylosis and replacement resorption is also expected after such an attempt (Fig. 32.7).

**Fig. 32.3 Infraposition**
A 13-year-old boy who had intruded his left upper incisor two years earlier. The incisor is ankylosed and is infrapositioned.

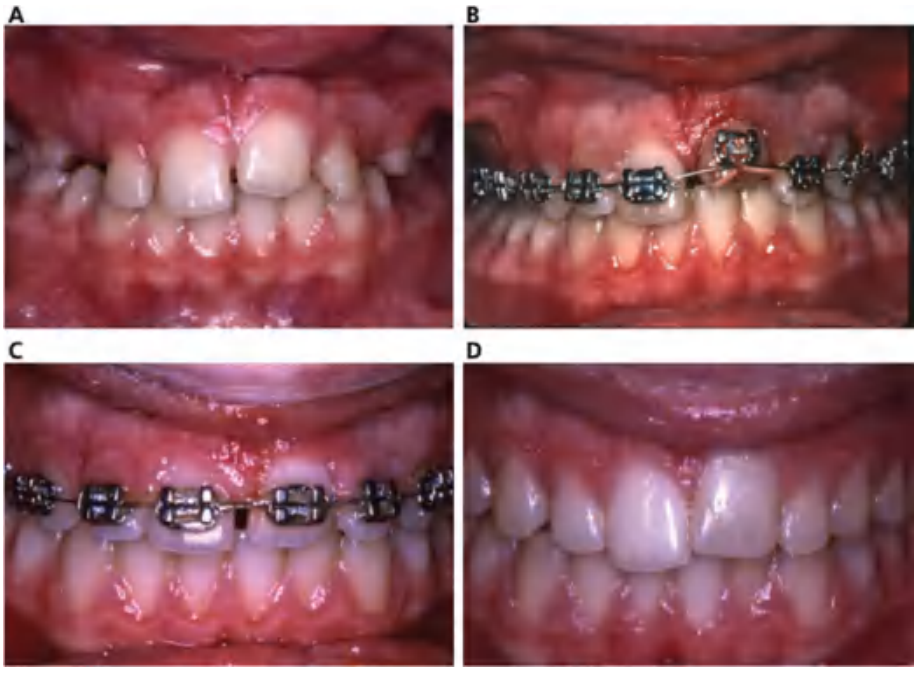



**Fig. 32.4 Unsuccessful attempt to extrude an ankylosed incisor**
A. An 11-year-old girl with an ankylosed left upper incisor in infraposition. B. An orthodontic appliance with an extrusion force was applied. C. 6 months later, there is intrusion of all adjacent teeth. D. Relapse after treatment. Despite composite build-up the outcome is a failure.

3. *Bone distraction* by dento-osseous osteotomy of a segment with an ankylosed tooth and surrounding bone has been reported. Different types of appliances for extrusion of the segment have been suggested (29–33). This treatment must be postponed until growth of the jaws is completed. It is important to keep in mind that there is still a fusion between the root and the adjacent bone. If bone distraction by dento-osseous osteotomy of the segment is performed in a growing individual an infraposition of the damaged tooth will be seen again. Moreover, this method cannot stop the progressive resorption of the root (Fig. 32.8).
4. *Extraction* of an ankylosed tooth may involve loss of attached bone; particularly the thin buccal plate of the

**Fig. 32.5 Incisal build-up of an ankylosed infrapositioned incisor**
A. This is a 13-year-old girl. An incisal build-up was performed in the left upper ankylosed infrapositioned incisor. B. 3 years later the infraposition has increased and the esthetic effect is unsatisfactory. Observe that the level of infraposition is best evaluated at the gingival margin.

**Fig. 32.6 Minimal infraposition**
A. 19-year-old man one month after replantation of avulsion of his right upper incisor. B. Six years after injury only a slight infraposition can be noticed.

**Fig. 32.7 Ankylosed incisor surgically repositioned**
A and B. Before repositioning. C and D. After repositioning. Note the unchanged marginal bone-level.

maxilla and the socket healing becomes jeopardized, i.e. with a defect both in the horizontal and vertical dimension and is for this reason regarded a non-suitable method today (Fig. 32.9).

5. *Autotransplantation* of a premolar to substitute an ankylosed incisor might be an alternative. In order to achieve revascularization of the pulp and successful periodontal healing the ideal root development should be ¾ of the full root length (see Chapter 33). The whole root of the ankylosed must be extracted. Attached bone will not be problem, since the transplant with its sound periodontal ligament will induce new bone formation (Fig. 32.10).
6. *Decoronation* of the infrapositioned tooth. This is the recommended alternative today and will for this reason be presented more in detail (Fig. 32.11).

## Decoronation

It was shown experimentally already in the 1970s that new marginal bone might form over the coronal surface of submerged roots covered with a mucoperiosteal flap (34–36). Very few inflammatory changes were seen when vital roots had been submerged, whereas this was a consistent finding, both periapically and pericoronally in submerged endodontically filled roots. Based on these findings a technique to remove crowns of ankylosed teeth was developed in 1984 by Malmgren and co-workers (37). Since then many case reports have been published and decoronation is today a well-documented method (38–47). The technique is now known as decoronation. The crown of the tooth is removed, and the ankylosed root is left in the alveolus to be substituted by bone (Fig. 32.11).

The initial study using this method study comprised a material of 24 ankylosed permanent incisors. An increase of marginal bone was demonstrated, particularly in patients treated before the pubertal growth. A more, comprehensive and detailed study by Malmgren et al 2015 described a follow-up of 103 decoronations (48). The study group consisted of 95 patients with 103 decoronated permanent incisors. The age at trauma ranged from 6.8 to 17.8 years (mean 10.7; median 10.4) and the age at decoronation from 9.3 to 22 years (mean 14.9; median 15.0). The average follow-up period was 4.6 years (range 1 to 19.3 years). The final group for statistical evaluation was based on 75 patients, 56 boys and 19 girls with only one tooth decoronated.

A considerable increase in the alveolar bone level was found in boys with a mean age of 14.6 years and in girls with a mean age of 13.0 years. The age of the patients at decoronation has thus a significant effect on the alveolar bone development which indicates that it is favorable to perform decoronation when the patients are young, particularly girls. In general, decoronation shall ideally be performed before the pubertal growth spurt, which starts about two years earlier in girls than in boys. In the recent study (48), estimations of changes of bone level were evaluated in relation to the cementum-enamel junction of the homolog on the most reliable radiographs (Fig. 32.12 and Table 32.1).

In four patients where two central maxillary incisors were decoronated, no increase of bone at the suture was found. Instead a resorption and rounding of the suture edges could be seen, and a resorption of the sharp edges of the bone at the suture had taken place and no vertical bone growth had occurred, whereas there was a successive formation of new marginal bone at the mesial surfaces of the laterals. A possible explanation may be a split of the uniting periosteum layer covering the suture. The periosteum consists of two layers, one outer fibrous layer containing blood vessels and one inner cellular osteogenic layer. When decoronation of two central incisors is made, the fibers of the outer layer are cut off and extend into the suture and no uniting periosteum layer is formed. The stimulation for new bone formation is lost (Fig. 32.13). When a central and a lateral are decoronated new marginal bone is formed and bone level increases (Fig. 32.14).

Implants had been inserted in 18 of the 29 patients that had reached the age of 20 years. In six patients root remnants were still present at the time of insertion. In four of these the implants had been in inserted in contact with the roots. They made no obstacle for insertion of neither the implants nor the healing process. 14 of the 18 implant patients did not receive ridge augmentation.

## Alveolar bone development after decoronation of ankylosed teeth

The marginal collagen fiber system and periodontal fibers formed during eruption of teeth play an important role for this development.

When two adjacent teeth are erupting, bone apposition occurs on top of the interdental septum via forces from both the periodontal and gingival fibers. The forces from these marginal fibers in this way form an active alveolar periosteum in growing individuals (Fig. 32.15).

When a tooth becomes ankylosed the periodontal fibers are partly or totally replaced by bone in the ankylosed area and tooth eruption is completely arrested.

The interdental fibers still link the ankylosed tooth to the adjacent teeth and thus partly prevent their eruption. Instead, the linked fibers cause tipping of the teeth, as they continue to erupt. No marginal bone development can be seen (Fig. 32.16).

After decoronation the blood clot is organized from the surrounding tissues and a thin bone layer apposition takes place over the resorbing root during follow-up (Figs 32.17, 32.18 and 32.19). This indicates that the interdental fibers between adjacent teeth and the dentoperiosteal fibers linked to these teeth have formed a new periosteum on top of the alveolar crest. The ongoing eruption of the teeth induces bone apposition through traction from the fibers to the periosteum (49).

**Fig. 32.8 Treatment of an ankylosed permanent incisor and alveolar bone with alveolar distraction**
Condition 4 years after trauma. Ankylosis of right central incisor with severe vertical infraocclusion and horizontal malposition. See Chapter 35 for the surgical technique.

**Surgical procedure**
The transport segment is mobilized.

**Distraction device**
An extrusion device (Martin® Track 1) is formed and applied. The vector of distraction is adjusted.

**The use of a screwdriver 3 times a day for distraction**
0.3 mm for each turn of the screwdriver, leading to 0.9 mm distraction per day for 8 days.

**Final treatment result**
Replacement resorption will, within several years, lead to replacement of the root with bone, ideally formed for replacement by an implant without bone grafting when the crown is eventually lost. In the meantime, the ankylosed tooth has been esthetically and functionally satisfactory for years.

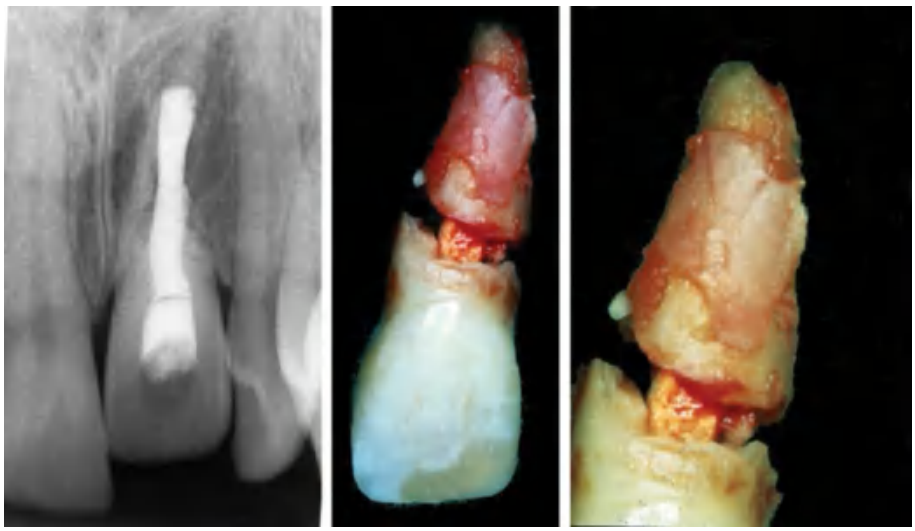


**Fig. 32.9 Complicated extraction**
Extraction of an ankylosed incisor where the bone plate was simultaneously removed.

**Fig. 32.10 Autotransplantation of a premolar in treatment of resorbing incisors**
A. 9-year-old girl with severe luxation of two central incisors and avulsion and replantation of the left lateral incisor after avulsion. B. Replacement resorption of the left central incisor seven months after injury. During eruption of the left canine total resorption of the lateral incisor occurred. C–D. A substantial alveolar bone loss was found after the extraction. E–F. Autotransplantation of a premolar from the lower jaw to the left central incisor region nine months after injury and orthodontic treatment started in the upper jaw 4 years after injury. G–H. Follow-up 15 years after trauma and 13 years after autotransplantation.

**Fig. 32.11 Surgical treatment of an ankylosed and infrapositioned incisor**
Moderate infraposition of a right upper central incisor due to ankylosis in a 14-year-old boy.

**Sectioning the crown from the root (decoronation)**
A mucoperiosteal flap is raised. The crown is removed with a diamond bur under a continuous flow of saline.

**Removing of the root filling**
The crown has been removed and the root filling removed with an endodontic file.

**Preparation of the root**
The coronal part of the root surface is reduced to a level of 2.0 mm below the marginal bone. The empty root canal is thoroughly rinsed with saline and thereafter allowed to fill with blood.

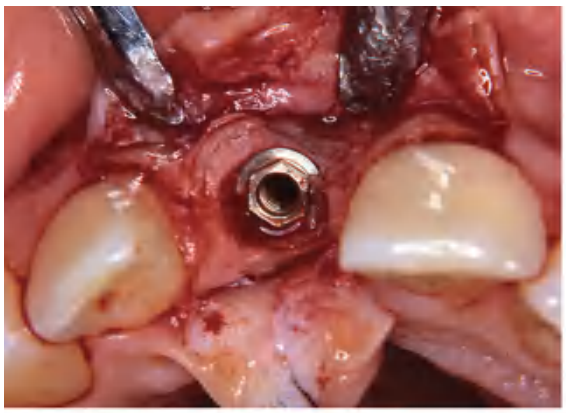
**Suturing the flap**
The mucoperiosteal flap is pulled over the alveolus and sutured with single sutures. A blood clot is formed in the gap between the labial and palatal mucosa.

**Temporary restoration**
The removed crown is shaped as a pontic with composite material and splinted to the adjacent teeth with acid-etch technique and an enamel composite bond.

**Follow-up 1 year after treatment**
A resin-retained bridge has been inserted. Note coronal growth of bone.

**10 years after decoronation**
The alveolar ridge has a favorable width for inserting an implant.

**Insertion of implant**
A root remnant maintains in the alveolar ridge. It makes no obstacle for the insertion and integration of the implant.



**Five-year follow-up after insertion of implant**

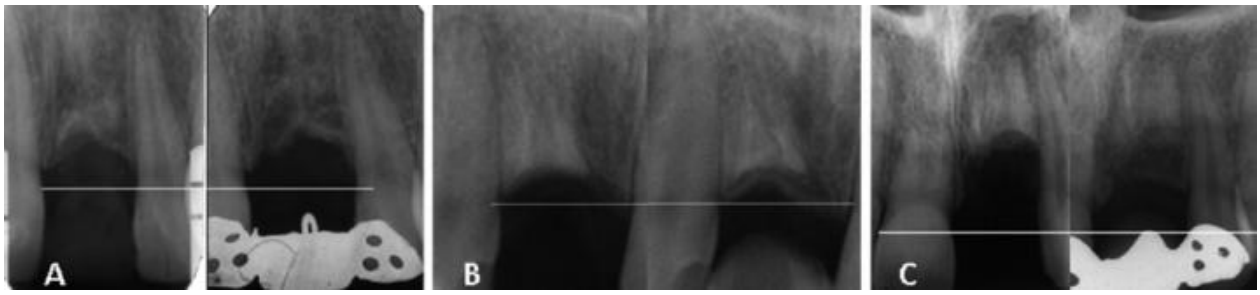



**Fig. 32.12 Estimation of change of bone level in vertical direction after decoronation**
A. Score 1: Unchanged or reduced alveolar bone level. B. Score 2: A moderate increase of the alveolar bone level. C. Score 3: A considerable increase of the alveolar bone level. Reproduced from (48) with permission.

**Table 32.1** Alveolar bone level after decoronation of 75 teeth in 56 boys and 19 girls in relation to age at operation (48).

| | Alveolar bone level | Number of teeth | Age at decoronation | |
|---|---|---|---|---|
| | | | Mean | SD |
| Boys | 1. Unchanged or reduced | 16 | 16.8 | 2.2 |
| *N* = 56 | 2. Moderate | 20 | 15.1 | 2.3 |
| | 3. Considerable | 20 | 14.6 | 2.6 |
| Girls | 1. Unchanged or reduced | 3 | 17.3 | 4.6 |
| *N* = 19 | 2. Moderate | 7 | 14.2 | 1.8 |
| | 3. Considerable | 9 | 13.0 | 1.2 |

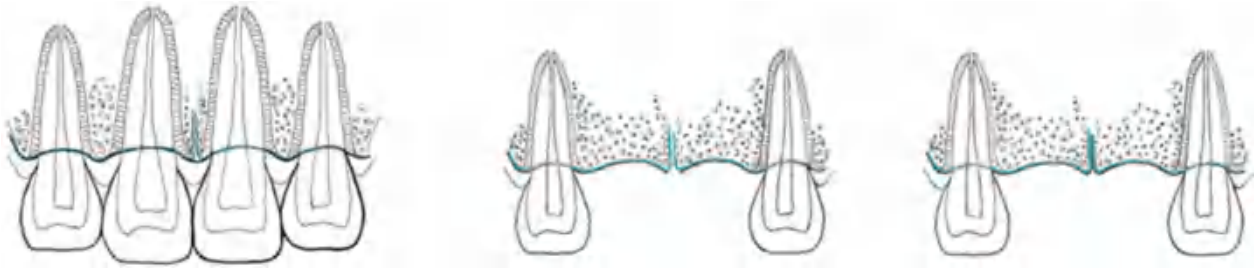
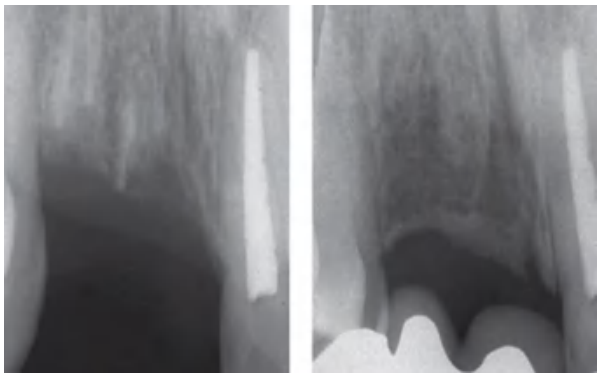


**Fig. 32.13 Schematic drawing showing the two layers of the periosteum**
A. The suture is bridged over by the outer united layer. B. The outer layer is cut off at the decoronation procedure. C. The two parts extend from both sides down into the suture. Reproduced from (48) with permission.



A B

**Fig. 32.14 Decoronation of right upper lateral and central incisors**
A. A 16-year-old boy immediately after decoronation. B. Result after three years. Note increase of bone in vertical direction and formation of lamina dura. Reproduced from (48) with permission.

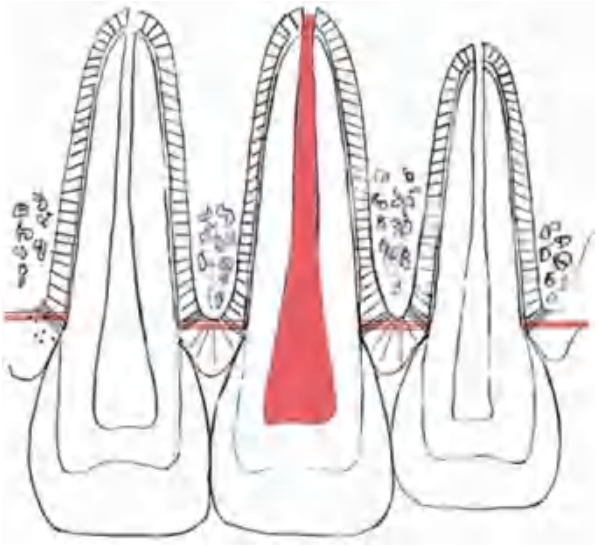


**Fig. 32.15 The marginal collagen fiber system and periodontal fibers formed during eruption of teeth play an important role**
When two adjacent teeth are erupting, bone apposition occurs on top of the interdental septum by forces from the marginal fibers. In this way an active alveolar periosteum is formed in growing individuals. Reproduced from (49) with permission.

**Fig. 32.16 A 17-year-old boy who had his left upper incisor avulsed and replanted 4 years earlier**
A. The replanted incisor is ankylosed and tilting of the neighbouring teeth is seen. B. Implication of ankylosis in a dentition with active growth. The ankylosis prevents further eruption of tooth, and the interdental ligaments tilt the adjacent teeth against the ankylosed tooth. Reproduced from (49) with permission.

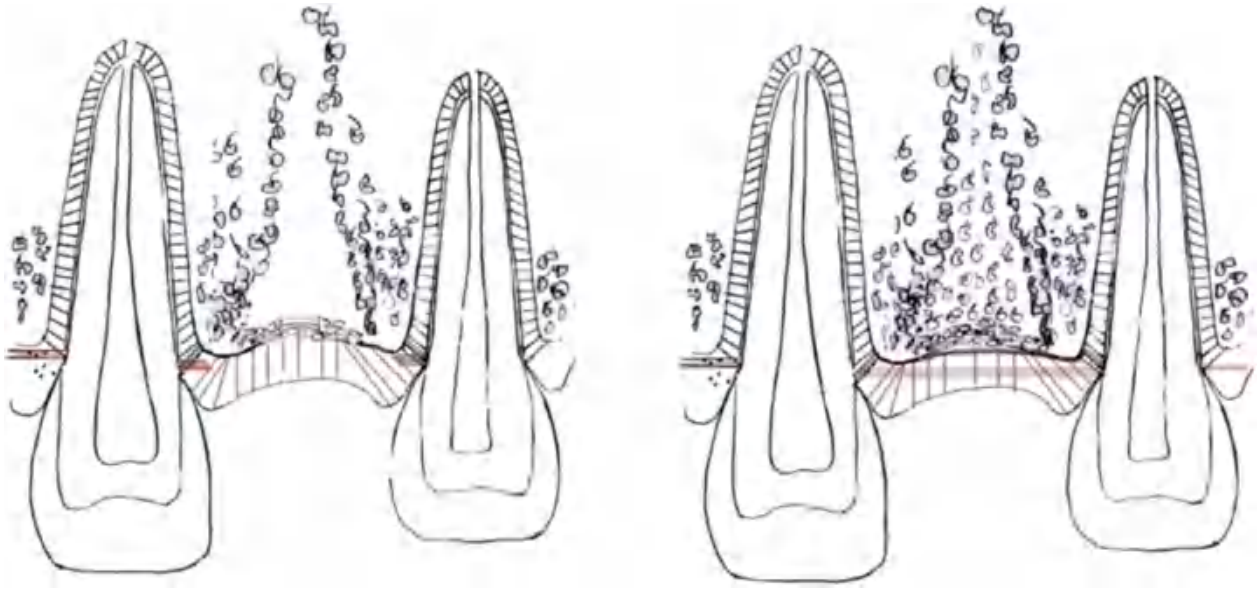


**Fig. 32.17 Bone apposition after decoronation**
A. A thin layer of bone apposition takes place over the resorbing root. B. A new periosteum on top of the alveolar crest is formed. The ongoing eruption of the teeth induces bone apposition through traction from the fibers on the periosteum.

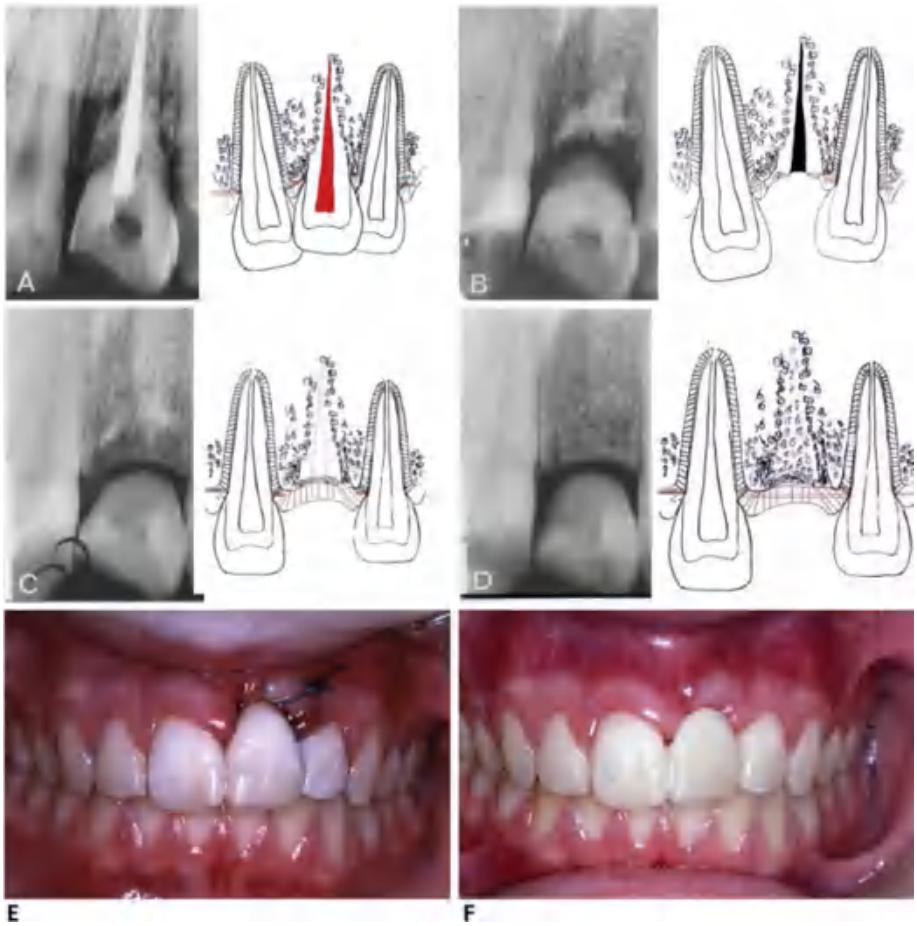



**Fig. 32.18 Decoronation of an ankylosed upper central incisor in a 13.5-year-old boy**. A. A and B. Before and immediately after treatment. Note bone level after decoronation. Arrows indicate cementum-enamel level of adjacent tooth. C. 6 months after treatment. Note shortened pontic and formation of new marginal bone coronal to root remnants. D. 12 months after treatment no remnants of the root can be seen. E. Clinical appearance at the time of decoronation. F. Due to continuous vertical growth of marginal bone the pontic had to be rebonded in vertical direction. Reproduced from (49) with permission.

This explanation is in accordance with known bone inducing effects during normal dental eruption. Similar findings have been observed after autotransplantation of teeth. Marginal alveolar bone deposition was seen when the transplanted teeth were put into an infraoccluded position and afterwards erupted into normal position.

Remodeling and growth of the alveolar bone in children and adolescents is intimately connected to skeletal jaw growth and tooth eruption. The collagen fiber system with free gingival, circular, transeptal and periodontal fibers formed during eruption of teeth play an important role for this development (50). Alveolar growth is diminished if the fiber system around

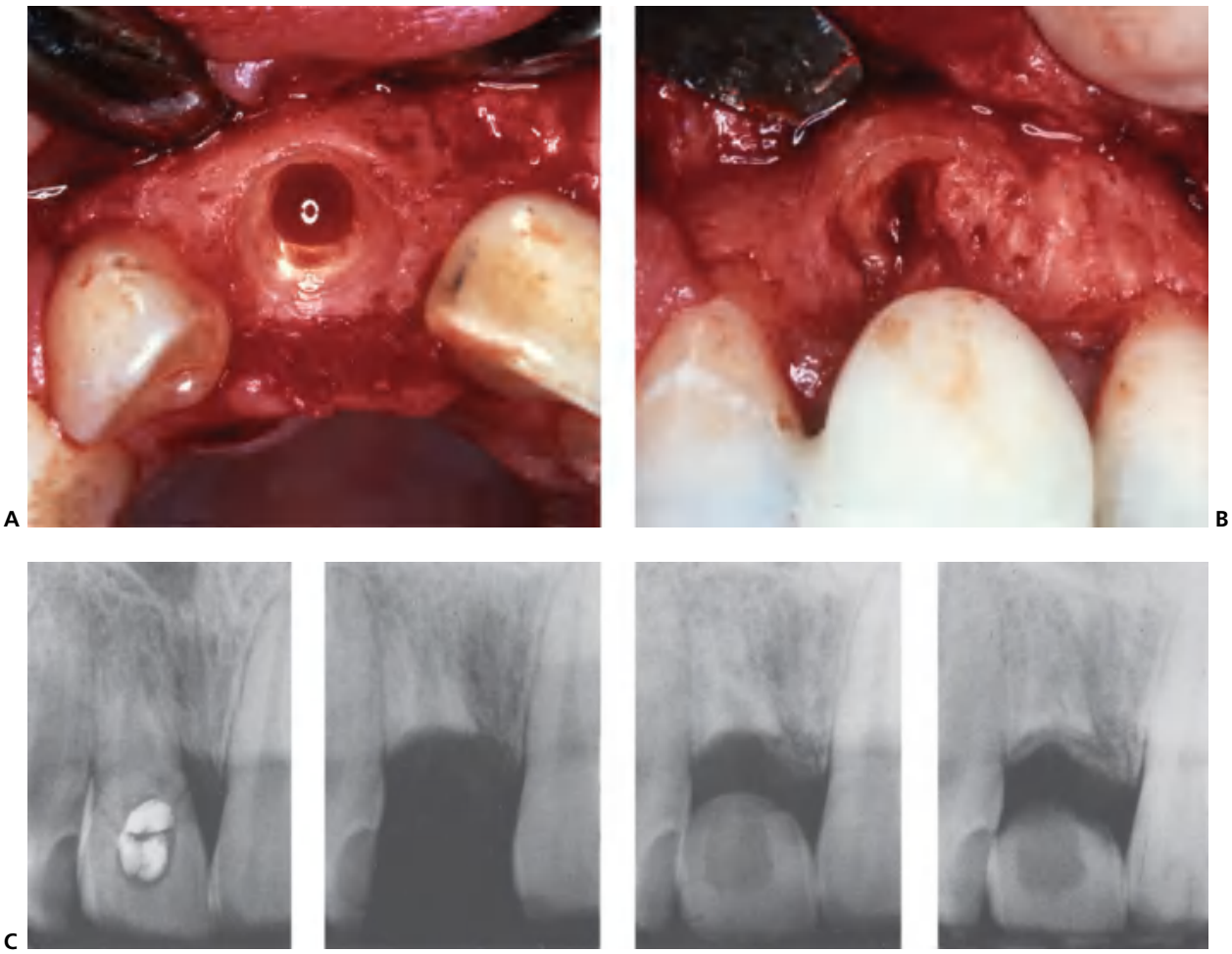



**Fig. 32.19 Formation of bone after decoronation**
A. Condition at the time of surgery. B. A flap was raised 10 months later. New bone is seen replacing the resorbed root remnants. C. Radiographs immediately before and after surgery and at 6 and 18 months follow-ups. Note regeneration of normal bone.

teeth is lacking as in cases of missing teeth. The marginal fiber anatomy in the interdental area is therefore of fundamental importance for the alveolar bone development if ankylosis and infraocclusion occurs after a trauma. They are also of importance for the explanations of the favorable bone development after decoronation of such teeth.

## Rate of infraposition

In young patients there are advantages in retaining the tooth for as long as possible as a space maintainer. Thus, the timing of extraction is very important.

Most avulsions occur at the age 8–12 years when growth of the jaws and alveolar processes are intense. The growth varies individually above all with the general skeletal growth, age and gender. In a study from 2002 (28), the relationship between the rate of infraposition and age at the time of injury, growth intensity and facial growth was analyzed. Development of the infraposition was analyzed by measurements on study casts with a digital caliper. In most cases the infraposition could be measured at the incisal part of the crown. Correction of the incisal edge of infrapositioned and/or reference tooth had been made in many cases and made it difficult or impossible to measure the changes in the incisal region. When the distance between the cervical/margin line of the reference crown and the infrapositioned tooth was measured it was found that this measurement corresponded very well with the measurement at the incisal region. The patients` body height curves were established and analyzed.

It was found that the rate of infraposition related to the age at the time of injury. In children younger than 10 years at the injury the increase of infraposition was faster and more severe than if the trauma had occurred later. In patients 10–12 years the infraposition did not increase as fast as in the youngest group, and in patients older than 12 the rate of infraposition varied.

The infraposition was in most cases also related to the skeletal growth (Figs 32.20 and 32.21). However, there was no absolute correlation between infraposition and total growth. There was a difference between horizontal and vertical growers (Fig. 32.22). Therefore, it is important to keep in mind that progression of infraposition varies individually. Patients older than ten years when ankylosis is diagnosed must also be monitored regularly. There is a great risk of severe infraocclusion

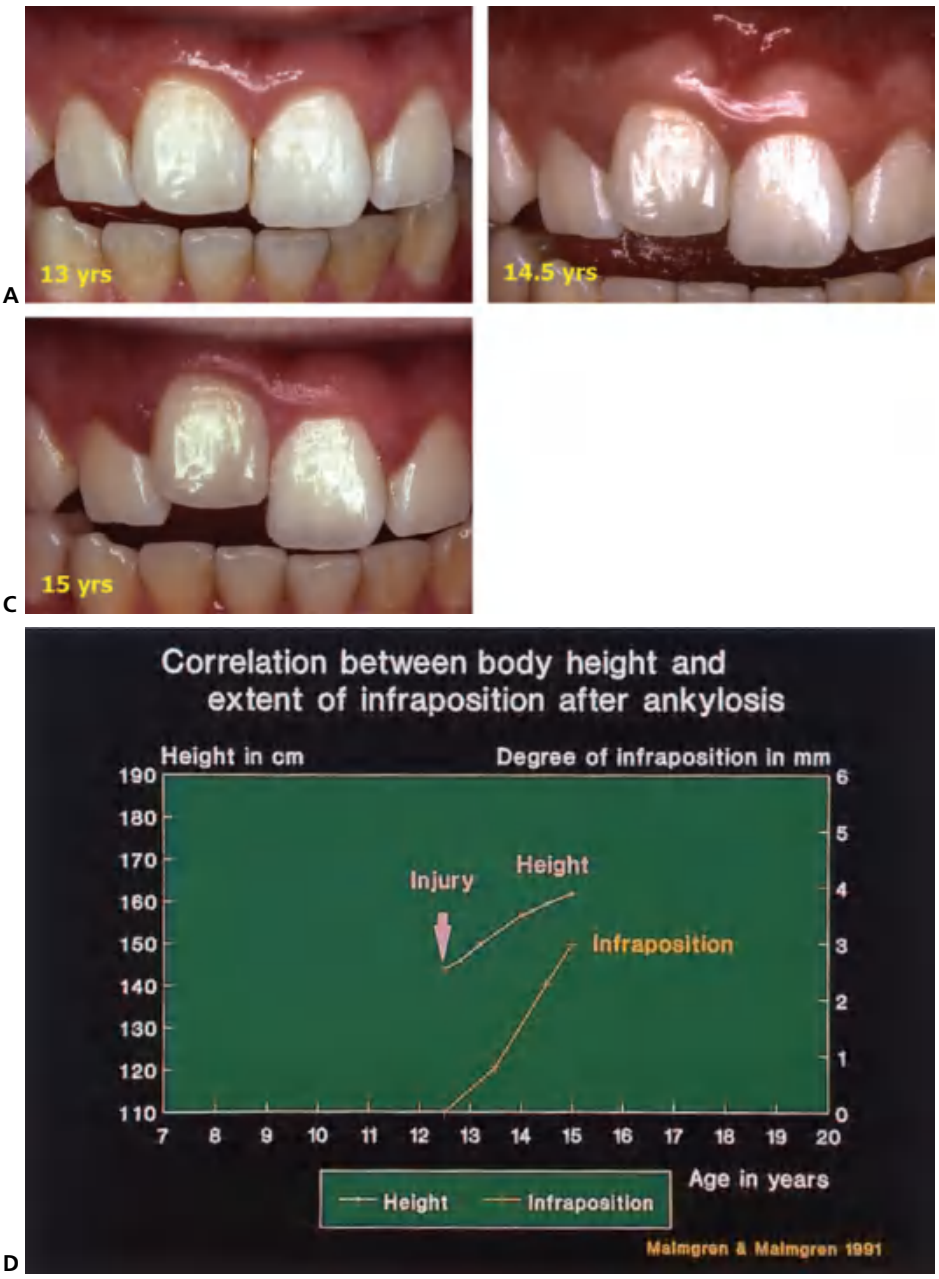



**Fig. 32.20 Relationship between general growth and infraposition of an ankylosed central incisor**
A. The right central incisor was replanted in a boy at 14 years of age. B. Slight infraposition can be seen after 1 year. C. At 5-year control, there is a slight increase in infraposition. D. Annual body height measurements were registered for the patient and showed almost completed growth 2 years after the injury. The degree of infraposition was measured in mm.

during the growth spurt. The time for the growth spurt varies individually. Annual body height measurement to evaluate the patient's individual growth intensity, and study casts or photos are aids to evaluate the rate of infraocclusion.

## Preservation of the alveolar process

Decoronation preserves the horizontal alveolar width and rebuilds lost vertical bone of the alveolar ridge in growing individuals. The biological explanation is that the decoronated root serves as a matrix for new bone development during resorption of the root and that the lost vertical alveolar bone is rebuilt during eruption of adjacent teeth. First a new periosteum is formed over the decoronated root, allowing vertical alveolar growth. Then the interdental fibers that have been severed by the decoronation procedure are reorganized between adjacent teeth. The continued eruption of these teeth mediates marginal bone apposition via the dental-periosteal fiber complex. The erupting teeth are linked with the periosteum covering the top of the alveolar socket and indirectly via the alveolar gingival fibers, which are inserted in the alveolar crest and in the lamina propria of the interdental papilla. Both structures can generate a traction force resulting in bone apposition on top of the alveolar crest. This theoretical biological explanation is based on known anatomical features, known eruption processes and clinical observations. A considerable marginal bone development when decoronation was performed on average at 14.6 years of age in boys and at 13 years of age in girls, which corresponds well with studies of the craniofacial complex. An age

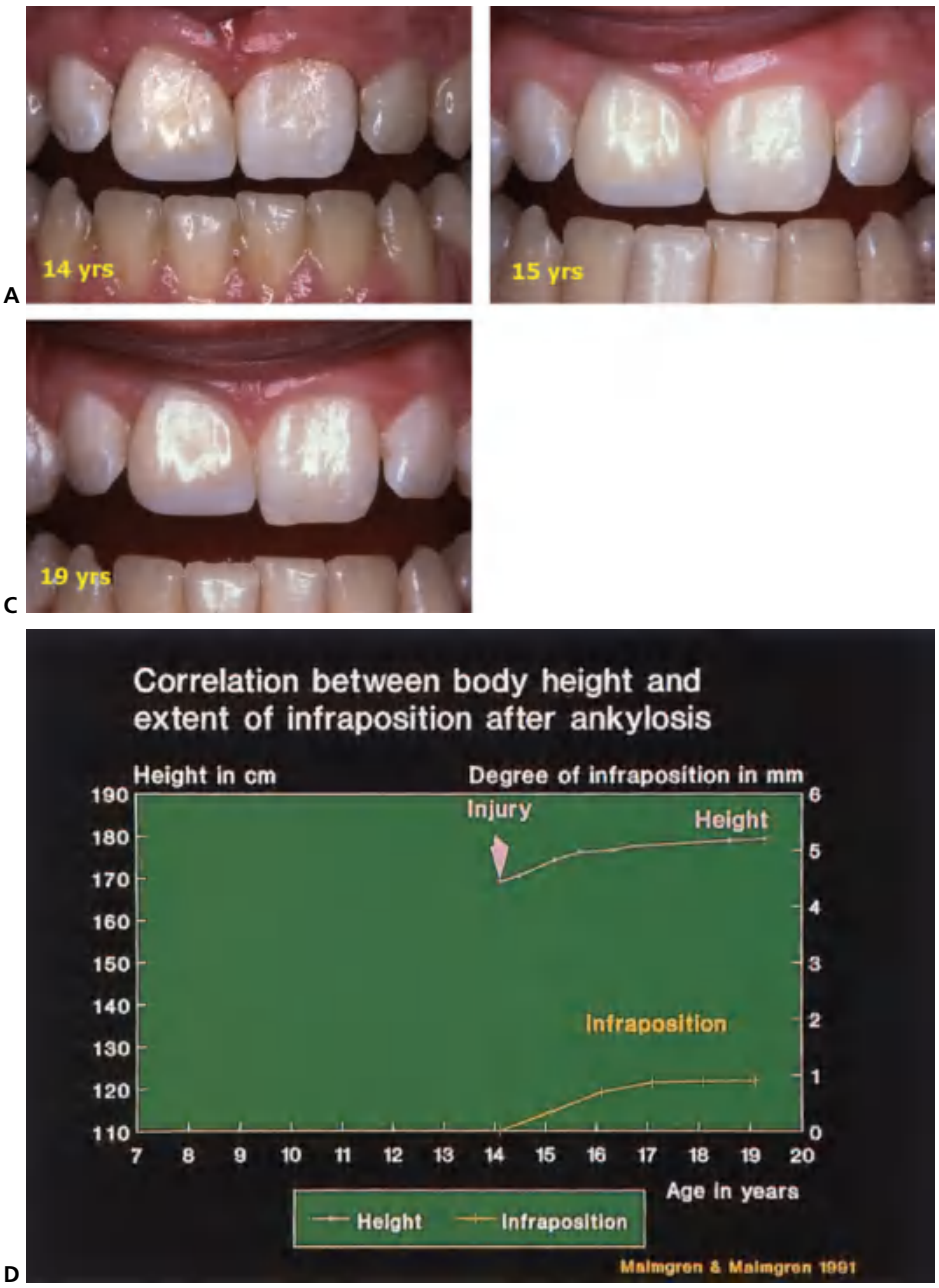



**Fig. 32.21 Relation between general growth and infraposition of an ankylosed central incisor**
A. The right central incisor was replanted in a boy at 13 years of age. Slight infraposition can be seen after 1 year. B and C. At 2- and 21/2-years after injury there is a marked increase in infraposition. D. There is a marked growth spurt in this patient which correlates with the marked infraposition.

difference of two years between boys and girls in this age interval has been described by Thilander 1995 (51) and corresponds with the growth velocity curves presented by Taranger and Hägg 1980 (52).

It is well known that the resorption of the alveolar process after tooth extraction in both jaws is significantly greater on the buccal aspect than on the lingual or palatal (53–57). With the decoronation technique the bone loss is minimized, and the ankylosed root is gradually resorbed and replaced with bone (Fig. 32.23). Lin et al. 2013 (40) measured the bucco-palatal alveolar dimensions of the alveolar ridge at the mid-mesio-distal distance after decoronation on study casts. They found a reduction of only 1 mm in the bucco-palatal dimension at the mid-decoronation area compared with the contralateral homologous tooth. This reduction is significantly less than the findings by Lam 1960 (58) who measured the alveolar change in the same way after merely extractions and found a reduction between 3–5.5 mm. In a recent case study, it was shown that some buccal bone was resorbed also after decoronation in teeth which had been root canal treated and decoronated without removing the root filling material (59). In the original method (37), an important part of the treatment is that the root filling material is removed, and the root canal is filled with a blood clot. The root acts as a matrix for new bone formation (Fig. 32.19), and the blood clot will organize from the surrounding bone tissue and bone is expected to be created in the canal. By not removing the root filling material can possibly explain the findings of this study (59). Further long-term studies are needed to elucidate the long-term effects of the alveolar process but today the decoronation seems to be the best alternative to preserve the crest. After avulsion when no replantation is performed, vertical

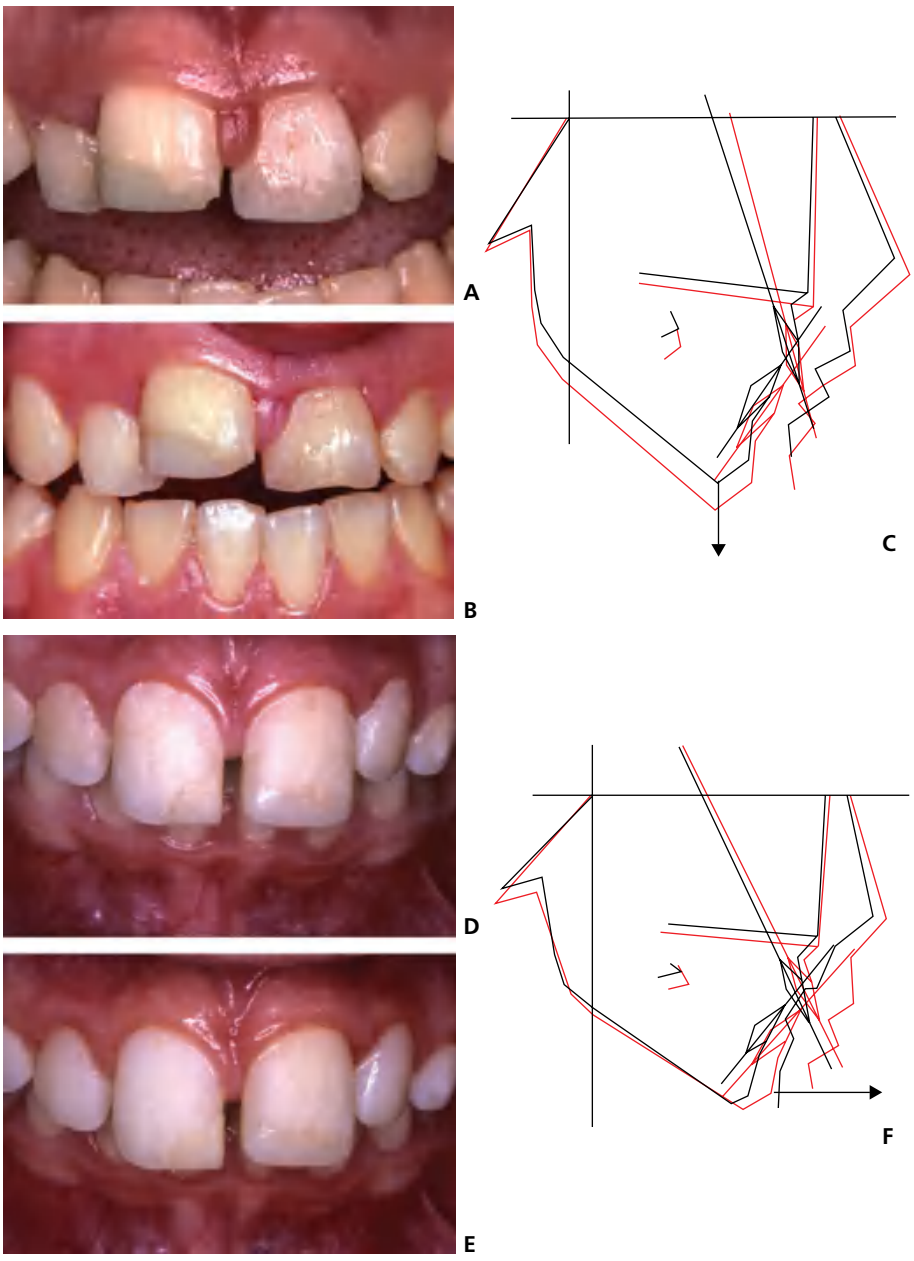



**Fig. 32.22 Relation between vertical growth direction of the face and infraposition of an ankylosed central incisor in a 14-year-old boy**
A. At the age of 14 years there was a minimal infraposition of the maxillary right incisor. The body height was 170 cm. B. At age 16 the infraposition of the ankylosed incisor had increased markedly. Note incisal attrition of the maxillary left central incisor. The right lateral incisor has tilted mesially. The body height was 176 cm. C. Schematic illustration of the cephalometric changes of the face shows vertical growth of the face.

**Relation between a horizontal growth direction of the face and infraposition of an ankylosed central incisor in an 8-year-old boy**
D. At the age of 8.2 years, 3 months after replantation, body height was 129 cm. E. At 9.6 years only a minimal infraposition can be seen. The body height was 137.5 cm. F. Schematic illustration of the cephalometric changes of the face shows horizontal growth of the mandible.

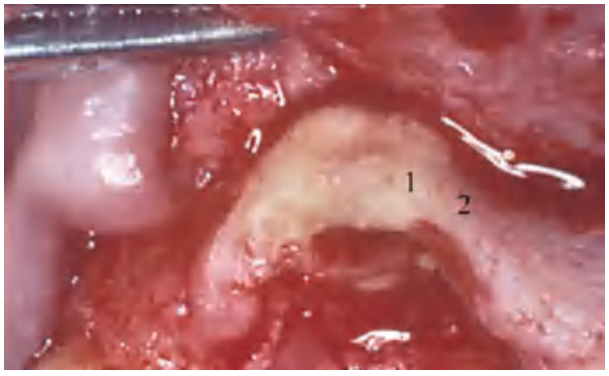



**Fig. 32.23** An upper incisor avulsed and replanted when the patient was 12 years old. Four years later a decoronation was performed. The buccal part of the root (1) is gradually replaced by bone (2) and no collapse of the buccal bone has occurred.

and horizontal bone atrophy will occur. Implant insertion requires a combined horizontal and vertical bone augmentation that can only be done surgically by either bone autotransplantation or vertical distraction osteogenesis. Consequently, avulsed teeth in young children should be replanted irrespective of the extra-alveolar condition and a compromised prognosis since decoronation has been shown to be a good treatment alternative (60).

## Implants and root remnants

Decoronation is used to preserve the crest for possibly later implant installation, and the question arises what happens to implants installed in bone containing remnants of dentin.

In the authors material, implants had been inserted in four cases in contact with the remaining root remnants. Dentin made neither any obstacle for insertion of the implants nor affected the healing process. Implant integration in bone crests containing root substances without ridge augmentation has been presented earlier (61–63). In recent studies on dentin it was found that dentin is osteoconductive and will be subjected to fusion with the bone, osseous replacement resorption and formation of new bone when implanted in bone (64–67). Malmgren et al. demonstrated that 14 of 18 implant patients where decoronation had been carried out, did not receive ridge augmentation indicating a preserved bone volume suitable for dental implants. This finding is supported by Lin et al. 2013 (40) who followed-up avulsed teeth, with CBCT, that received implants after decoronation without ridge augmentation. For more details regarding treatment planning for implants and alternative methods in the growing patient see Chapter 34.

## Essentials

Damage to the periodontal membrane or drying during an extra oral period and replantation causes bony ingrowth with fusion between tooth and bone. This area will increase with increasing time, gradually resorbing cementum and dentin and eventually the root will be replaced by bone.

### Clinical signs of ankylosis

(1) An ankylosed tooth changes its percussion sound to a bone-like, metallic, high pitch percussion sound. When more than 20% of its root surface is affected by ankylosis a decreased mobility is found.
(2) Later, ankylosis related resorption of the root can be seen in radiographs.
(3) Dentoalveolar ankylosis is accompanied by an increasing infraposition (infraocclusion) of the affected tooth in children and adolescents, and to a less degree also in some adults.
(4) An ankylosed tooth can be compared with an osseointegrated implant and cannot be moved. Thus, orthodontic extrusion is not possible. Such an attempt will only result in intrusion of adjacent teeth.

### Treatment options

(1) *Composite build-up*. This is not recommended in growing individuals, but may be a treatment option if the patient has passed his pubertal spurt and the infraposition is minimal.
(2) *Surgical repositioning*. This has been suggested when the ankylosed area is minimal. However, the long-term prognosis is poor, since the reduced marginal bone level is unchanged, and the root has a poor bone support.
(3) *Bone distraction by dento-osseous osteotomy* of a segment with an ankylosed tooth and surrounding bone. This treatment must be postponed until growth of the jaws is completed. This is a temporary solution and cannot stop the progressive resorption of the root.
(4) *Extraction of the ankylosed tooth*. This may involve loss of attached bone; particularly the thin buccal plate of the maxilla and the socket healing becomes jeopardized, i.e. with a bone defect both in the horizontal and vertical dimension. This option should be avoided.
(5) *Autotransplantation* of a premolar to substitute an ankylosed incisor might be an alternative (see Chapter 33).
(6) *Decoronation*. This is the first option.

### Decoronation technique

(1) A mucoperiosteal flap is raised and the crown of the tooth is removed with a diamond bur under a continuous flow of saline.
(2) The root filling is removed.
(3) The coronal part of the root surface is reduced to a level of 2.0 mm below the marginal bone level.
(4) The empty root canal is thoroughly rinsed with saline and thereafter allowed to fill with blood.
(5) The ankylosed root is left in the alveolus to be substituted by bone.
(6) The mucoperiosteal flap is pulled over the alveolus and sutured with single sutures. No stretching or lengthening of the flap should be made. A blood clot shall be formed in the gap between the labial and palatal mucosa.

### Timing of decoronation

- Bone development has the best prognosis if decoronation is performed when the infraocclusion is less than 1/3 of a homolog tooth.
- The rate of infraposition is related to the dental development, growth of the face and general skeletal growth.
- Decoronation performed before the pubertal growth spurt has a better prognosis than if it is performed later. The growth spurt starts about two years earlier in girls than in boys.
- The time for the growth spurt varies individually. Annual body height measurement to evaluate the patient's individual growth intensity, and study casts or photos are aids to evaluate the rate of infraocclusion and timing of decoronation.

### Implants insertion after decoronation

When growth is completed, implants can be inserted. Should there be remaining dentin in the bone, implants can still be installed in the region, even when implants are in contact with remaining root remnants. Dentin makes neither any obstacle for insertion of the implants nor affects the healing process.

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# 33

# Autotransplantation of Teeth to the Anterior Region

J. O. Andreasen, L. Andersson, M. Tsukiboshi & E. M. Czochrowska

Autotransplantation of teeth is today a well documented treatment method and is well suited for treatment of anterior tooth loss (1–12, 17–20, 31–36, 49–56, 58–60, 71–74). Due to the unique osteogenic capacity of the graft, this procedure offers a treatment alternative, where both the lost tooth and the atrophied alveolar process can be replaced (21, 37) (Fig. 33.1). Studies have demonstrated good long-term survival of these transplants, thus providing a realistic treatment alternative for tooth replacement in young individuals (see Appendix 4, p. 1025).

Furthermore, this treatment procedure has its optimum of success in early childhood, a period where implants are definitively contraindicated (see Chapter 34).

The key to successful tooth transplantation has been shown clinically and experimentally to be proper selection of grafts with adequate root development as well as the design of surgical techniques for non-traumatic graft removal and graft insertion. These techniques are described in detail in published textbooks on tooth replantation and transplantation (21, 37) and survey articles (34, 38).

## Treatment planning

When a tooth has been lost after trauma there are many factors that have to be taken into consideration when a long-term treatment plan is made. A multidisciplinary approach is recommended with many specialists involved. When anterior tooth loss occurs before completion of skeletal growth a panoramic radiograph should be taken, in which potential grafts, such as canines, premolars and in some cases diminutive third molars, can be identified. When donor teeth are available, the cost–benefit of autotransplantation should be weighed against other treatment solutions (for overview see Chapter 34), such as orthodontic space closure (see Chapter 28), fixed prosthetics (see Chapter 31) and implants (see Chapter 35). As optimal prognosis depends upon teeth with incomplete root formation, the patient group best suited for this procedure is from 10 to 13 years old (at this age, alveolar growth is not yet complete, thereby contraindicating implants and fixed prosthetics except for resin-retained bridges). An important question is whether to close the space of the lost tooth or keep the space open for replacement with a transplant. In some patients it may be preferable to close the space by orthodontic space closure. The choice between autotransplantation and orthodontic space closure depends upon an orthodontic analysis of growth and occlusion (see Chapters 28, 34 and 36).

## Surgical considerations

### Autotransplants versus implants

Most traumatic tooth loss occurs in young, growing patients. Implants are contraindicated in growing patients because they interfere with the growth of the alveolar process resulting in an infraocclusion (see Chapters 32, 34 and 35). Implant treatment must therefore often be postponed until growth is finished. During this waiting period there is a risk of atrophy of the alveolar crest: when it is time for implant installation this cannot be done without bone grafting prior to implant treatment to achieve an esthetically optimal situation.

Autotransplantation of teeth to the region of tooth loss is a method that can be applied regardless of whether the patient has finished growth or not. In most cases, autotransplantation

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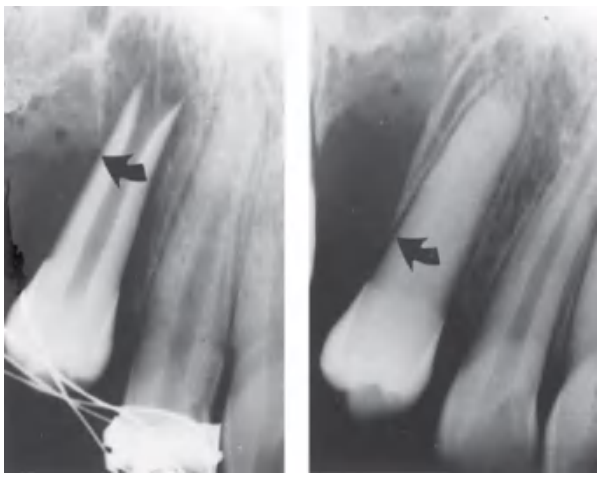

A B

**Fig. 33.1** Alveolar bone induction by a premolar transplant to the canine region. A. Due to a severe trauma considerable bone has been lost in the region (arrow). B. The autotransplanted premolar has induced formation of alveolar bone distal to the transplant (arrow). Source: Reproduced from (10) with permission.

is a permanent choice, but can also be applied temporarily in young patients to preserve the alveolar bone volume until growth is finished. The ultimate goal of autotransplants in growing individuals is the potential of not only maintaining bone but also creating a new alveolar bone by periodontal ligament induction and the eruption process (Fig. 33.1) (see Chapter 34).

Implant treatment can be a good choice for replacing a lost tooth in adult patients, who have finished growth. The completion of growth must be confirmed by height measurement or a hand-wrist radiograph before implant treatment can start (see Chapter 34).

A comparison between autotransplant and implant reveals almost identical long-term prognosis and good esthetics can be achieved with both methods.

There is, however, a significant difference in the cost efficiency (see Chapter 41) as autotransplantation does not carry the high cost of implant treatment. This is an especially important consideration for those individuals throughout the world who cannot afford implant treatment.

## Graft selection

The primary goals in graft selection are to ensure optimal periodontal healing and pulp survival, which in turn ensures optimal root development after transplantation. In the selection of donor teeth, crown and root anatomy should be considered.

With respect to coronal anatomy, reduction of tooth structure should generally be limited to enamel. Dentin exposure should be limited due to the risk of exposure of soft tissue inclusions which are found in post-transplantation dentin and which can imply a risk of subsequent pulp necrosis (see later). However, in cases with compatible root anatomy but less than optimal crown anatomy, crown anatomy following transplantation can be modified significantly by the use of porcelain crowns or veneers following interceptive endodontic treatment (see the section on restoration of transplanted teeth).

Concerning root anatomy, the graft should fit the recipient site loosely, avoiding contact with adjacent bone and providing at least 1 mm to the adjacent roots. Many potential grafts will exceed the existing labio-palatal dimension, especially if atrophy of the alveolar process has taken place. However, special surgical techniques, such as intentional fracture of the labial bone plate or a composite tooth-labial bone plate graft can solve this problem. An important issue is the dimension at the cervical aspect of the graft. This must be in reasonable harmony with its antimere. Such harmony can often be obtained if the graft is rotated 45° or 90°. Moreover, if possible the donor tooth should be harvested from the opposite side of the arch to the recipient site.

With these limitations in mind, the following grafts (in order of preference) appear to be suitable for the *maxillary central incisor* region: second mandibular premolars, canines, first mandibular premolars, second maxillary premolars and diminutive third molars (Figs 33.2–33.4). Maxillary first premolars should generally not be used because of their bifid roots, which present surgical obstacles to non-traumatic graft removal.

In the *lateral incisor* region, the mandibular first premolar is, because of its size, the only suitable candidate for transplantation (Figs 33.5 and 33.6). In the *canine* region, all premolars, except the maxillary first, can be used (Figs 33.7 and 33.8). In general, grafts should be selected with three quarter to full root formation with a wide-open apex. At this stage of development, the graft is easy to remove and both periodontal and pulpal healing are very predictable.

## Analysis of the recipient site

A radiographic examination is necessary to analyze the vertical bone level and space available between adjacent teeth. As a rule, the crown of the potential graft should fit into the space available mesiodistally above bone level. Furthermore, dimensions of the root of the graft should ensure at least 1 mm of bone between itself and the roots of adjacent teeth.

The labio-palatal dimension of the alveolar process can be estimated by inspection clinically. In case of doubt as to whether a potential graft can fit into the recipient site, an open surgical procedure should be chosen.

# Surgical procedure

In this context only, certain principles of graft removal and recipient site preparation will be presented. Readers interested in detail about the surgical procedures are referred to recently published textbooks on this subject (21, 37).

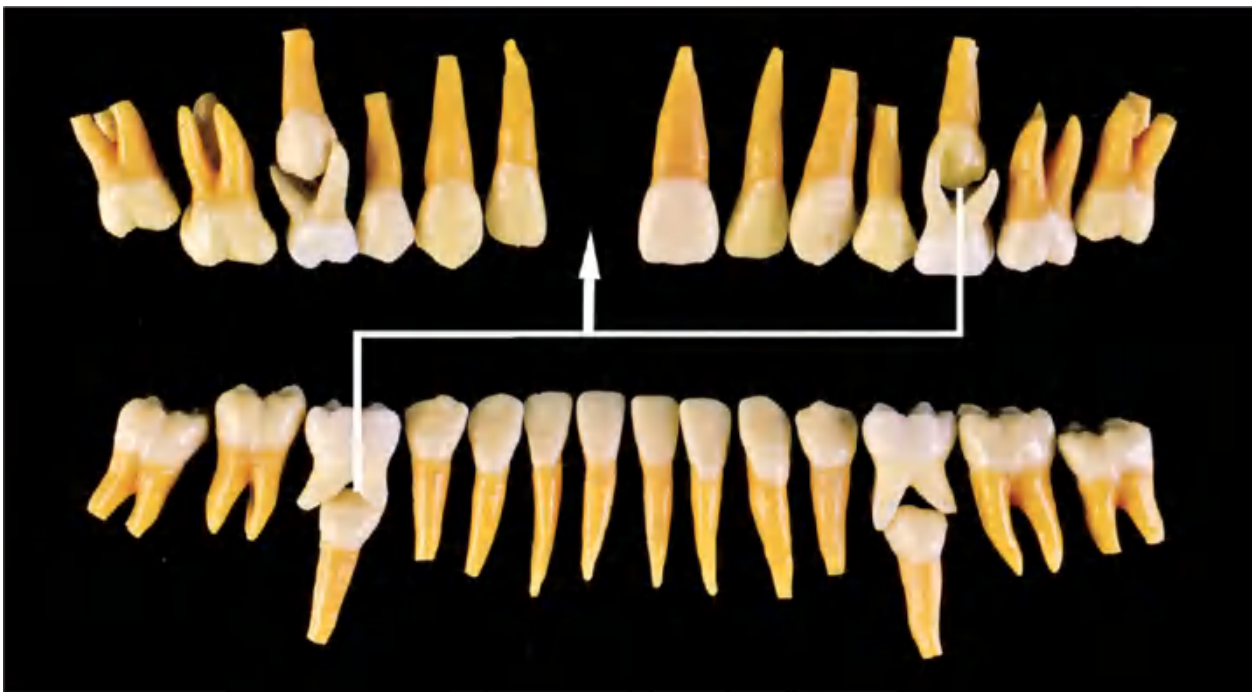

**Fig. 33.2** Central incisor loss, possible donor teeth. Except for mandibular first premolars, all premolars can serve as donor teeth. In rare cases, diminutive maxillary third molars can also be used.

**Fig. 33.3** Central incisor region, transplantation of a mandibular second premolar. The double exposure illustrates where crown substance should be added or reduced.

## Preparation of the recipient site

Based on the radiographic and clinical examination and depending on the fit of the graft in the existing alveolar process, a *closed* (without raising a flap) or *open* (with flap exposing the buccal alveolar bone area) procedure is selected. As a rule, the recipient site is prepared prior to graft removal in order to shorten the extra-alveolar period for the graft. Before the surgical procedure, antibiotic coverage is achieved (e.g. phenoxymethylpenicillin 2 g) 1 hour before surgery and maintained for a period of 4 days (at doses of 1 g twice daily). (The doses mentioned are for a 70 kg man of normal weight and should be reduced accordingly for persons with a lower body weight such as children and adolescents.)

If a tooth is present at the recipient site, it is extracted, and the socket enlarged so that the future graft will be surrounded by a 1–2 mm coagulum (Fig. 33.9). This will optimize periodontal and pulpal healing (13). The socket preparation should be performed non-traumatically using a bur with internal saline cooling (15). Thereafter the entrance to the socket is covered with a gauze sponge while awaiting the graft.

With the help of Cone Beam technology and three-dimensional printing the fabrication of accurate dummies can today be used in the preparation of the site, so the dummy is used and not the graft itself until the prepared site is ready for grafting (57). Surgical templates can also be used (58, 59).

## Open procedure

A trapezoid incision is made in the attached labial gingival mucosa. The affected tooth is extracted (Fig. 33.10) and an osteotomy cut is made with a thin bur at the site of the future alveolus. The labial bone plate is then removed with a chisel

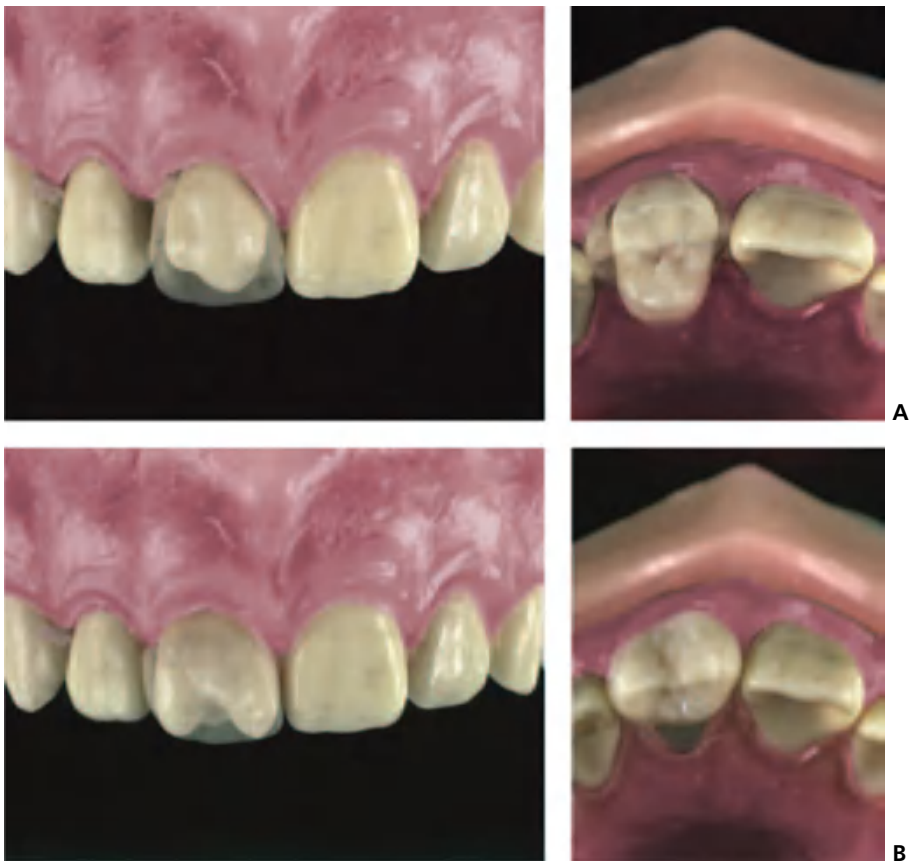


**Fig. 33.4** Central incisor region, transplantation of a maxillary second premolar. The double exposure illustrates where crown substance should be added or reduced when the transplant is positioned without (A) or with (B) rotation.

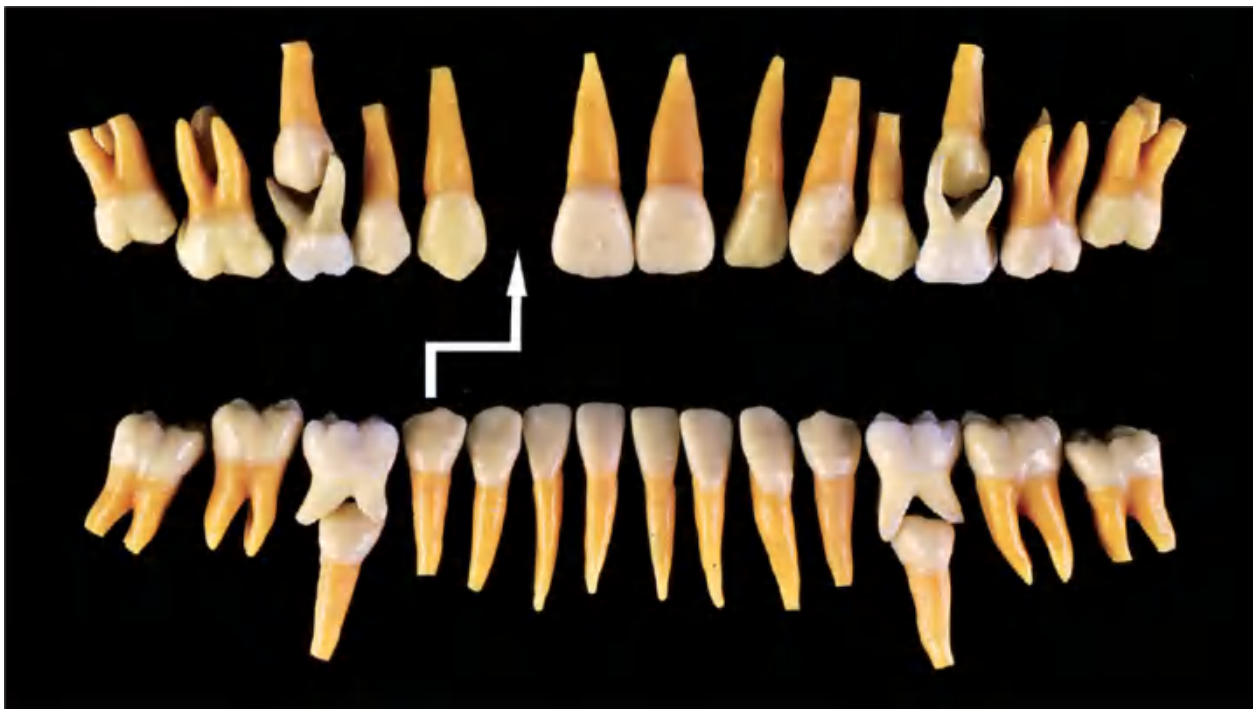

**Fig. 33.5** Lateral incisor loss, possible donor teeth. Mandibular first premolars are optimal donor teeth.

and stored in saline. Thereafter the socket is prepared with a bur with internal saline cooling. Finally, the socket is covered with a gauze sponge in order to prevent saliva contamination during graft removal.

## Graft removal and insertion

The donor tooth is removed non-traumatically, usually implying a flap procedure and removal of marginal bone labially in the case of mandibular premolars or palatally in

**Fig. 33.6** Lateral incisor region, transplantation of a mandibular first premolar. The double exposure illustrates where crown substance should be added or reduced.

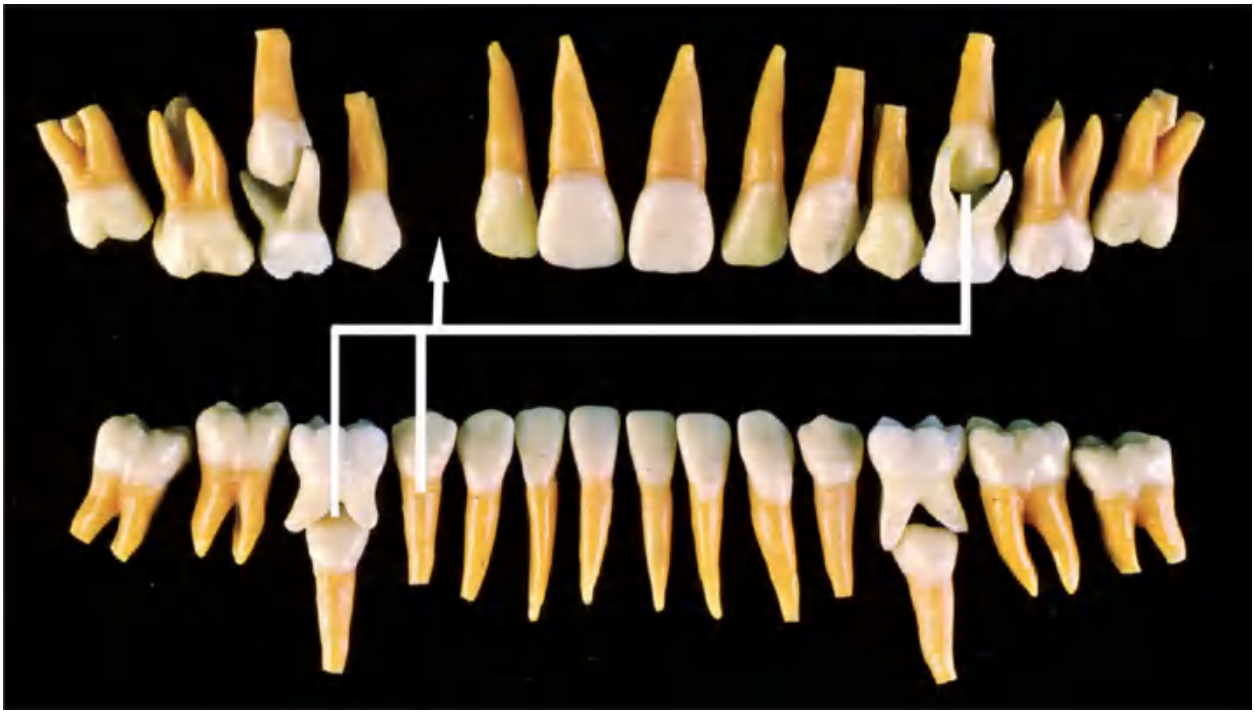

**Fig. 33.7** Canine loss, possible donor teeth. Mandibular premolars are optimal donor teeth.

**Fig. 33.8** Canine region, transplantation of a mandibular first or second premolar. The double exposure illustrates where crown substance should be added or reduced.

**Fig. 33.9 Autotransplantation of a premolar to the central incisor region using a closed procedure**
The treatment plan is to transplant a maxillary second premolar to the central incisor region. The incisor is to be removed due to root resorption subsequent to replantation.

**Extracting the incisor**
The incisor is extracted. Note the extensive root resorption.

**Preparing the socket**
The socket is enlarged with a surgical bur with internal saline cooling. The socket is expanded palatally. The socket is then rinsed with saline.

**Testing the size of the socket**
Using a porcelain replica of a premolar, the size of the socket is tested. Thereafter the socket is washed with saline.

**Removing the maxillary second premolar**
After making a gingival incision and incising the cervical part of the PDL, the tooth is extracted using gentle luxation movements.

**Repositioning the graft**
The graft is placed in a 45° rotated position in order to achieve sufficient cervical width.

**Splinting the graft**
The tooth is splinted with an 0.2 mm stainless steel wire placed around the necks of the adjacent teeth. The position for the wire is ensured by etching the labial enamel of the adjacent teeth and incorporating the wire in composite.

the case of maxillary premolars. The graft is placed in a semi-erupted position in the new socket and stabilized with a suture cervically.

In an open procedure, the dissected labial bone plate is cut lengthwise with a heavy scissors into 2–3 pieces. These grafts are placed so that they cover the labial part of the root with cortical bone facing the root. Thereafter the labial flap is elongated using periosteal incisions and then sutured tightly around the neck of the transplant. As experimental studies have shown that rigid splinting is detrimental to both pulpal and periodontal healing, this procedure should be avoided (13, 39). To prevent vertical displacement during healing, a suture is placed over the occlusal surface of the graft. Alternatively, a figure-of-eight stainless steel wire (diameter 0.2 mm) inserted between adjacent teeth may serve the same purpose. The stabilizing sutures or wires can be removed after 1 week.

## Follow-up period

Clinical and radiographic controls should be made 4 and 8 weeks after grafting, where healing complications, such as

**Fig. 33.10 Autotransplantation of a premolar to the central incisor region using an open procedure**

The treatment plan is to transplant a right maxillary premolar to the central incisor region where there is marked vertical and horizontal bone atrophy.

**Flap procedure and socket preparation**

A trapezoidal incision is made, and the atrophied alveolar ridge exposed. Osteotomy cuts through the labial bone plates and subsequent removal of bone plate with a chisel will expose the future socket. The socket is prepared with a bur with internal saline cooling.

**Transplanting the premolar**

The premolar is seated in the socket and fixed in its new position with a suture placed cervically. Thereafter, the labial bone plate is cut lengthwise in 2-mm wide strips with heavy scissors and placed over the transplant, with the concave cortical surface against the periodontal ligament (arrow).

**Flap repositioning and splinting**

The flap is lengthened via periosteal incisions at its base and then sutured. A thin (0.2 mm) stainless steel wire anchored between the right lateral incisor and the left central incisor stabilizes the tooth during initial healing.

pulp necrosis or external root resorption (i.e. inflammatory or replacement resorption) can be diagnosed (13). After 4 months, the graft can be restored (see later). Further clinical and radiographic controls should be performed at the following intervals: 6 months, 1 year, 2 years and 5 years after grafting in order to assess pulpal and periodontal healing (Fig. 33.11).

## Pulpal healing

Three to 6 months after transplantation, pulpal healing can be diagnosed clinically by a positive sensibility response as well as radiographically by pulp canal obliteration (Fig. 33.12), which is a sign of reinnervation and revascularization of the traumatized pulp (14). Pulp necrosis due to

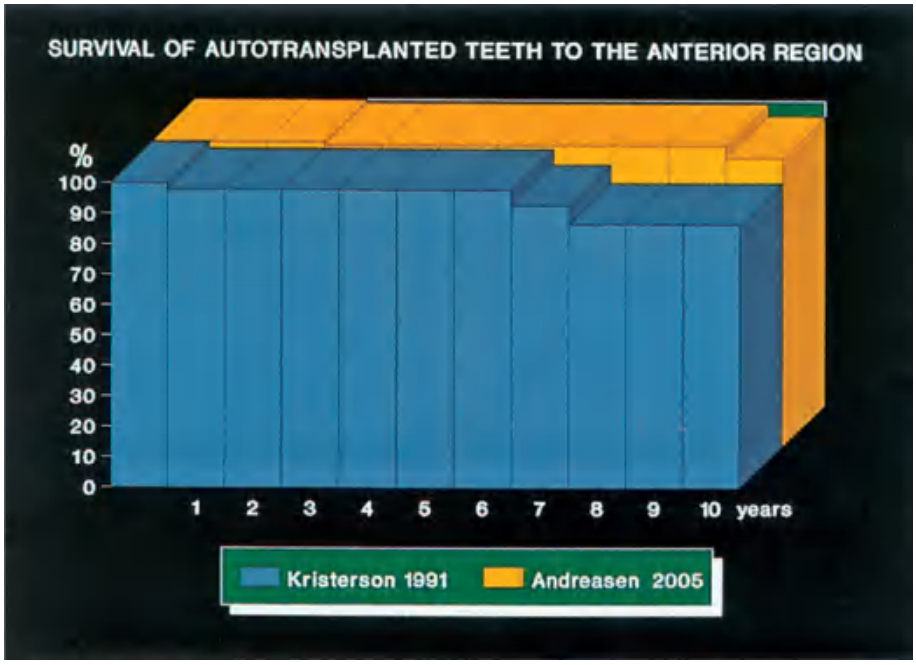


**Fig. 33.11** Tooth survival of autotransplanted teeth used in the treatment of anterior tooth loss. A life table analysis of two clinical studies representing 50 and 130 transplants to the anterior region. Source: Reproduced from (20) and (22) with permission.

**Fig. 33.12 Long-term survival of a grafted premolar**
A maxillary second premolar transplanted to the lateral incisor region.

**10-year control**
Stable periodontal and pulpal healing is evident 10 years after transplantation.

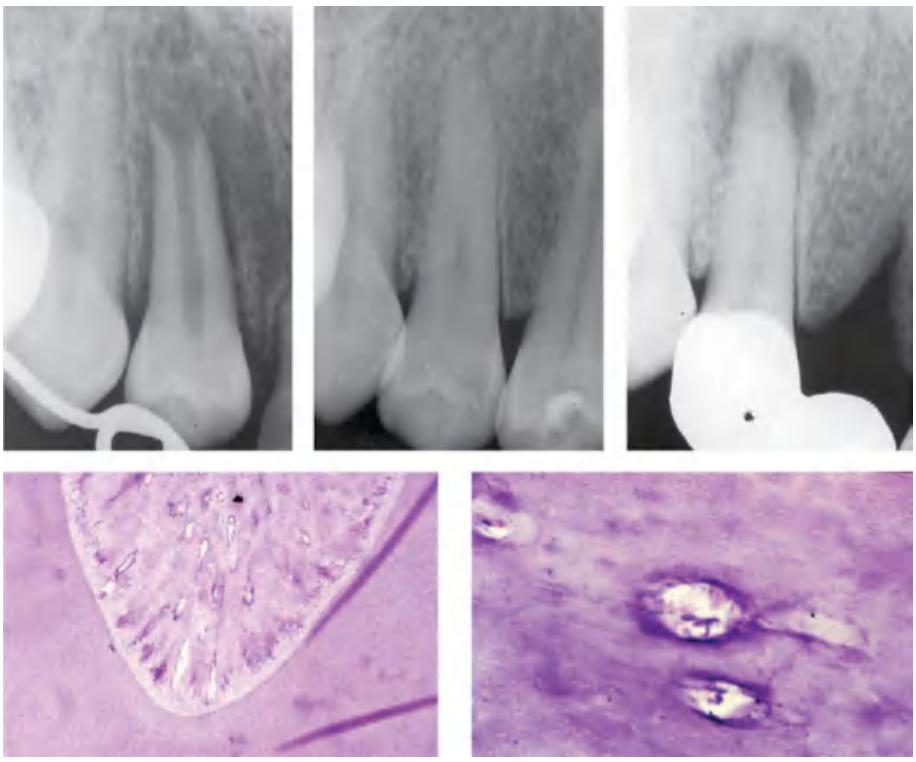

**Fig. 33.13** Pulpal complications subsequent to crown preparation. The histologic slides show extreme canal obliteration with vascular inclusions. One year after crown preparation, a periapical radiolucency is seen. Source: Reproduced from (10) with permission.

infection can usually be diagnosed after 4 to 8 weeks. Treatment of pulp necrosis is outlined in Chapter 25. Secondary pulp necrosis may occur following pulp canal obliteration and is suspected to be related to a new trauma or dentin exposure during crown preparation (10) (Fig. 33.13).

### Periodontal healing

The earliest radiographic signs of healing (i.e. reformation of the periodontal ligament space) can be seen after 4 weeks and are usually manifest after 8 weeks. Inflammatory and replacement resorption are usually evident after 4–8 weeks. These complications were found with a frequency of 7% in the authors' material (22). The treatment of both resorption types follows the principles outlined for avulsed and replanted teeth (see Chapter 25).

### Root development

When transplanting teeth with an open apex a continuing root development is seen (Fig. 33.12).

## Orthodontic considerations

### Orthodontic considerations in selection of a donor tooth

The main indication for a donor selection is prognosis for a successful healing. Cone-beam computer tomography (CBCT) is an important diagnostic tool and very useful in order to obtain a best match between the donor and the recipient site as it enables detailed and accurate assessment of the donor tooth morphology, root development and recipient site anatomy (CBCT). If more space is required to accommodate the available donor, pre-surgical orthodontic treatment can include space opening procedures at the recipient site to allow for root parallelism. For optimal transplant accommodation at the recipient site 3D donor replicas can be obtained from a CBCT examination and can serve as a guiding tool during surgical preparation of the recipient site (63–65).

Patients with traumatic loss of maxillary incisors may display different clinical scenarios before transplantation such as single or multiple tooth loss which may be combined with alveolar bone loss in severe and/or older traumas. Alveolar bone loss makes the matching between the donor tooth and the recipient site often more difficult in patients with traumatic injuries to the anterior maxilla than in patients with congenitally missing premolars and preserved deciduous molars. It is essential during surgery that the donor tooth is placed at the most optimal position regarding tooth healing; the transplant position can be later adjusted orthodontically for restorative purposes. Often teeth transplanted to the anterior maxilla are placed superiorly and labially to account for the deficient alveolar process and to avoid occlusal interferences during healing.

Post-surgical orthodontic treatment can include: 1. closure of the donor space aiming to establish normal dentoalveolar relations, 2. alignment of the donor tooth in the dental arch

aiming at gingival levelling and matching of the mesiodistal width to a contralateral central incisor (single-tooth loss) or neighboring teeth (multiple tooth loss). The commencement of the orthodontic levelling procedures of a transplanted developing premolar is determined by the stage of its root development at the time of transplantation and should coincide with the final stages of root development. Donors transplanted at earlier stages of root development will require longer healing periods to allow for completion of root development which preferably should not be disturbed by orthodontic forces. Usually, orthodontic levelling of a transplanted developing premolar should not be initiated before 3–6 months after surgery (34, 66). The start of orthodontic treatment after transplantation depends on the existing malocclusion and the individuals' skeletal and dental development.

## Overjet

There is an association between the frequency of traumatic injuries to maxillary incisors and increased incisal protrusion (68). Recent meta-analysis on the association between large overjet and dental injuries to the anterior teeth (70) confirmed that large overjet may double or even triple the risk for trauma to anterior permanent teeth. Järvinen (69) reported that injuries were twice more common in children with overjet > 3 mm and 2.7 times more common in children with overjet > 6 mm than in children with normal overjet. Both the severity of the injury and the number of injured teeth increased with increasing overjet. Insufficient lip closure which presumably leaves the upper incisors unprotected was reported as predisposing factor for increasing the risk of traumatic injury (63). The current evidence suggests that providing early orthodontic treatment for children with prominent upper front teeth is an effective approach in reducing the incidence of incisal trauma (67).

Increased overjet is associated with the presence of Class II malocclusion. Extractions of premolars can be considered a camouflage treatment for moderate skeletal malocclusions aiming to establish normal dental relationships and to correct the overjet. In patients with Class II malocclusion extraction of upper premolars is therefore justified from an orthodontic treatment perspective. The extracted teeth can be considered as donors in case there is traumatic loss of central incisors. The extraction spaces will allow to retract the protruding anterior teeth including the transplanted premolars and to establish a normal overjet.

## Space conditions

In crowded cases, where tooth extractions are indicated replacing traumatically lost anterior teeth with developing premolars otherwise scheduled for extraction is well justified. However, often in a crowded dentition orthodontic space opening to accommodate transplanted premolars must be performed before surgery. The length of pre-surgical orthodontic treatment must be included in the treatment planning in relation to the optimal root development of the premolar.

Spacing is generally a contraindication for tooth transplantation, because removal of a donor premolar creates additional space which must be closed. It is still reasonable to consider autotransplantation of a developing premolar to replace missing maxillary incisors even when orthodontic indications are unfavorable, because in young patients' prosthodontic replacements including dental implants are not possible. Natural teeth adapt well to the growth and development of the jaws, have a good long-term prognosis (42) and usually provide better esthetics than dental implants (71) which is important in the anterior maxilla. It is usually less demanding to obtain a satisfactory tooth replacement in posterior segments because their gingival parts are not that visible as in the anterior maxilla.

If a traumatic loss of a maxillary anterior tooth or teeth occurs in a patient with a normal occlusion, then autotransplantation of a developing premolar creates an additional space at the posterior segment, which must be managed. It is usually easier to mesialize upper than lower molars with orthodontic forces, therefore, it is preferable to consider as donors maxillary second premolars (Figs 33.2 and 33.3). Application of skeletal anchorage (TADs, temporary anchorage devices) or Class II mechanics may be utilized to assist orthodontic space closure in those patients. Also, compensatory extractions of the contralateral, non-transplanted premolars can be applied to control the upper midline in cases with unilateral loss of a maxillary central incisor.

## Premolar transplantation versus orthodontic space closure

In patients with a unilateral loss of a central incisor, it is better, if available, to select a donor tooth from the opposite side to avoid loss of two teeth in the same quadrant. If a failure occurs, then orthodontic space closure including mesialization of a lateral incisor to the position of a central incisor followed by mesialization of all other teeth in the segment is a viable option (73). Space remaining after removal of a donor premolar can be utilized to control the midline during orthodontic space closure. The main advantage of the orthodontic space closure approach over tooth transplantation is its predictability, since there is always a risk of failures including different types of resorption after transplantation surgery.

Similarly, in patients with a traumatic loss of both central incisors, it is prudent to select lower premolars as donors, since unsuccessful healing after transplantation will not increase the number of missing maxillary teeth and orthodontic space closure will still be the option. From an orthodontic perspective extraction of lower premolars in Class II patients is undesirable, but in young patients with missing maxillary incisors it may be still justified, since treatment options are limited. The occlusion

should be then corrected orthodontically to establish normal dental relation.

From the restorative perspective premolar morphology is usually closer to the morphology of a maxillary central incisor than to the morphology of a maxillary lateral incisor. Czochrowska et al. evaluated the esthetic outcome after transplantation of developing premolars and after orthodontic space closure by lateral mesialization to replace traumatically lost central incisors (40, 41). Autotransplanted premolars and mesialized lateral incisors were reshaped to incisor morphology with composite build-ups or porcelain veneers. Gingival width was reported to be of a critical importance, since major changes in tooth morphology, including grinding or enlargement are not biologically possible.

Professional assessors showed a small preference to the esthetic results obtained from autotransplanted premolars compared to patients who were more satisfied after orthodontic mesialization of lateral incisors. A midline shift was present in the majority of patients with orthodontic space closure by lateral incisor mesialization and the average length of the orthodontic treatment was relatively long (2 years 10 months). Lateral incisor mesialization and orthodontic space closure to replace a missing central incisor often includes canine reshaping; thus more restorative treatment is usually needed compared to premolar transplantation.

### Orthodontic treatment of transplanted teeth

In many trauma cases, where autotransplantation to the anterior region has been performed, it is often necessary to reduce a maxillary overjet orthodontically. Clinical studies indicate that orthodontic treatment is possible 3–6 months after autotransplantation without a significant risk of progressive resorption (15, 20, 22).

## Restorative considerations

The success of tooth transplantation in the anterior maxilla cannot be evaluated solely from a biological perspective and successful healing (41). In the smile zone satisfactory esthetics of the final restoration are very important and suboptimal results are easily recognized by patients (40). During treatment planning careful evaluation of the donor and neighboring incisors morphology, especially in cases with unilateral loss of a central incisor, is important to obtain optimal tooth match. During surgery, a transplanted tooth must be positioned in the alveolar bone to allow optimal healing, however, this position may not be ideal from a restorative perspective. Often a transplant must be placed superiorly in a deficient alveolar process in relation to natural teeth for better accommodation and/or to avoid occlusal interferences. In those cases, post-surgical orthodontics are necessary, which should be included in the overall treatment plan and discussed with the patient and the parents. Orthodontic extrusion of transplanted teeth helps to regenerate the alveolar process offering an important advantage over osteointegrated dental implants which remain ankylosed in bone.

### Tooth morphology and color match

Premolar transplantation in patients with bilateral loss of both central maxillary incisors may be more demanding from a surgical perspective, since it involves removal of two donor teeth and sometimes transplant accommodation in a deficient alveolar process in patients with or without space deficiency problems. Orthodontic space opening before surgery may also be more difficult in those cases. However, from a restorative perspective it is easier to obtain a good match between two transplanted premolars at the position of central incisors, than to match a natural, central incisor with a premolar. Premolar teeth are usually narrower than central incisors and have more rounded labial surfaces than maxillary central incisors (Fig. 33.22, below). CBCT is a great tool for donor morphology evaluation and optimal donor selection.

Czochrowska et al. (40) evaluated esthetics of restored premolars autotransplanted to the position of maxillary central incisors and reported that a common finding was a mismatch in color and gingival width between the transplant and the natural, central incisor. In this study, most of the transplanted premolars were reshaped with direct composite veneers, which are considered esthetically inferior to ceramic veneers. Orthodontic repositioning of a transplanted premolar was found to significantly improve esthetics of the final restoration and therefore the need of pre-transplantation orthodontics should be thoroughly discussed with a patient and parents before treatment.

Stenvik & Zachrisson (34) suggested that the best match between a premolar and a central incisor morphology can be obtained by rotating lower first premolars up to 90 degrees; upper premolars are too narrow and rotated 90 degrees are usually too wide cervically. However, orthodontic indications for extraction of a lower first premolar are rather uncommon (Class III malocclusion, severe crowding) and therefore orthodontic space closure after lower premolar extraction(s) for transplantation purposes can be complicated, especially, if the position of the lower incisors must be preserved. A possible solution, if a lower first premolar is chosen as a donor to replace a missing central incisor is to autotransplant at the same time an upper second premolar in the position of a lower first premolar (double transplantation). This option will redistribute the space after a donor removal to a posterior maxilla, where is usually easier to perform orthodontic mesialization. However, a second transplantation doubles the number of operations for a young patient and the possibility of transplant failures and therefore this option should probably be the last resort.

Usually, premolars transplanted to the anterior maxilla are reshaped to the incisors' morphology after the completion

of the orthodontic treatment. However, if post-surgical orthodontic treatment is planned, then it may be sensible from a restorative perspective to make a composite build-up before bonding an orthodontic bracket. The restoration should follow the long axis of the transplanted premolar and its height should correspond to the contralateral central incisor (if preserved) or to the height of the neighboring teeth. A calibrated periodontal probe can be used to measure pocket depth at the transplanted and neighboring teeth so that the restoration can be adjusted accordingly. If the true long axis of the donor tooth is used as the reference during reshaping, then the orthodontic treatment achieves root parallelism of the transplant with the neighboring teeth. When the donor tooth is restored with the aim to obtain the best match with the neighboring teeth, it may camouflage the true position and the long axis of the transplant. In this case, orthodontic repositioning will not result in root parallelism between the transplant and the neighboring teeth. This can pose a problem in the future, because of the amount of grinding necessary in order to camouflage the premolar morphology and the position of the transplant.

The general principles for restoration of transplanted teeth should imply a minimum interference with the health of the pulp and the gingiva. In a tooth transplanted with an incompletely formed root, revascularization of the pulp normally occurs, but as a rule with pulp canal obliteration to follow. Pulp canal obliteration can usually be diagnosed 3–6 months after transplantation. This newly formed dentin differs from normal dentin by virtue of its cellular content and many vascular inclusions (21). Thus, any exposure of dentin, as during crown preparation or caries, may permit progressive bacterial invasion. As an adequate secondary dentin response in the pulp is not possible due to the constricted root canal, an infected pulp necrosis may occur (Fig. 33.13). For this reason, it is suggested that restorative procedures are performed prior to total canal obliteration, i.e. 3–9 months after transplantation.

## Dental papilla

It has been documented that the interproximal papillae next to the transplanted premolars in the anterior maxilla to be generally well preserved or slightly hyperplastic (41). Cardaropoli et al. (79) reported, that following implant placement remodeling of the labial bone plate takes place resulting in horizontal and vertical bone reduction. This bone reduction can have a negative effect on the esthetic outcome. Recession of the midfacial mucosa was reported to be a risk factor after immediate or early implant placement (82). It has been reported that soft tissue recession including the interproximal papillae are expected following implant placement (74, 83). This is especially important, if two or more maxillary incisors are missing, because preservation of the interproximal papillae between adjacent artificial teeth supported on dental implants may be difficult (76, 84). Mucosal discolorations adjacent to the reconstruction and even visibility of metal parts have been reported in studies on dental implants with long-term follow-up periods (80). Therefore, premolar transplantation to replace missing maxillary anterior teeth provides long-term normal soft periodontal tissue appearance (Fig. 33.4) which was recently described in a case report comparing periodontal tissues following an implant and a transplant placement in anterior maxilla in the same patient (72).

Before restoration, the contour of the gingival margin should also be considered. If a gingivectomy is to be performed, coronal regrowth of approximately 1 mm in the anterior region should be expected (24).

Safe restoration of transplanted teeth implies the following considerations:

(1) Pulpal status should be determined prior to restoration, i.e. a positive sensibility response should be elicited.
(2) The restoration should be made so that no or only a minimum of dentin is exposed. At least no post-transplantation dentin must be exposed.3. The restorative procedure used should prevent or limit microleakage, which leads to bacterial invasion, hyper-sensibility and discoloration of the restoration. The use of the new dentin-bonding agents may help to eliminate these problems related to bacteria invasion following tooth reduction.

If the tooth has been endodontically treated, the first two considerations can usually be ignored.

The restorative procedures are presently confined to two types: *composite resin restoration* or *ceramic laminate veneers*, both employed after adequate enamel reduction.

## Composite restoration

These restorations can be performed very soon after transplantation. After necessary enamel reduction, the crowns are built up according to the principles described for the use of composite resin in the treatment of crown fractures (Figs 33.14–33.17).

An important aspect in the restoration of the traumatized dentition is the restoration of symmetry in the dental arch. A decisive factor is the cervical dimension of the graft, which can be adjusted during the surgical phase of treatment by graft rotation at the recipient site. However, in the case of anterior asymmetry, either due to deviation of graft dimension with its antimere, or where adjacent teeth have migrated into the site of tooth loss or infraocclusion in the case of protracted ankylosis, and where for various reasons orthodontic realignment is not possible, the problem arises as to how to re-establish lost symmetry.

Slight reduction of the proximal surface of the adjacent incisor may compensate for some space loss. There should be maximal length of the restored transplanted tooth to enhance the illusion of symmetry. The facial surface should be flattened, with maximum separation between mamelons, and acute proximal angles to allude to a broader tooth. Finally, the mesial and distal corners of the incisal edge should be squared (21).

**Fig. 33.14 Maxillary central incisor replacement using auto-transplantation**
Right central incisor replaced with a left mandibular second premolar. The tooth has been restored with composite.

**Right central incisor replacement**
The incisor was replaced with a left mandibular second premolar. The tooth has been restored with composite.

**Right central incisor replacement**
The incisor was replaced with a right maxillary second premolar. The tooth has been restored with composite.

**Left central incisor replacement**
The incisor was replaced with a left maxillary third molar. The tooth has been restored with composite.

**Fig. 33.15 Autotransplantion of mandibular premolar to the central incisor areas with a closed technique**
Transplantated premolar sutured into position. The postoperative radiograph shows incomplete root development.

**Follow-up**
A radiograph shows that full root development has been achieved and the usual obliteration is seen.

Another situation can arise when the mesiodistal dimension of the transplant is greater than its antimere, as when a maxillary premolar has been transplanted and rotated 90°. In this situation, the opposite optical effect can be used to achieve optimal symmetry (21). The transplant should be restored to maximum length, with the facial surface as well as the mesial and distal corners rounded and minimal distance between mamelons. This technique will deflect light and suggest smaller dimensions. Moreover, the size of the contralateral can also be enhanced to improve symmetry (21).

Finally, in teeth with diminutive cervical dimensions, cervical build-up in ceramic, similar to that done in the case of single-tooth implants can provide another solution to anterior asymmetry (25). In case of replacement of lateral incisors very minimal composite restoration is necessary.

Composite build-ups are inferior to porcelain veneers in terms of bacterial accumulation, since they attract more easily dental plaque (72). Composite build-ups can in general be later replaced by veneers, for improved matching in shape and color and optimal esthetics, especially, in the presence of the natural contralateral incisor (see Chapters 30 and 36).

## Ceramic laminate veneers

Porcelain veneers provide, especially at the gingival part, better contour match to the natural teeth during laboratory fabrication than direct composite restorations. Ultra-thin feldspathic porcelain veneers (0.1–0.2 mm) require minimum enamel preparation (minimally invasive prosthodontics). They provide excellent esthetics and are durable with excellent survival characteristics. It has been documented that survival of ultra-thin feldspathic porcelain veneers is comparable or superior to that of other prosthodontics restorations (70, 77, 78). The superior properties of feldspathic porcelain veneers make it feasible to successfully restore transplanted teeth in the anterior maxilla of young adults (Fig. 33.18). The main disadvantage of feldspathic porcelain veneers are the high cost and the need for skilled clinicians and laboratory technicians.

The conservative preparation necessary for veneer restoration makes this treatment procedure suitable for restoring the esthetics to the anterior region following premolar autotransplantation while preserving pulpal vitality following post-transplantation canal obliteration (25) (Fig. 33.22, below). Although the crown height in younger individuals is very likely to undergo changes, the gingival margin of ceramic veneers cemented with a composite luting agent is esthetically acceptable despite its eventual visibility. Patient satisfaction with these restorations, including the prospect of revision at a later date due to maxillary skeletal growth, is more than adequate.

Special problems arise with respect to preparation of premolars for anterior restorations. Often orthodontic

**Fig. 33.16 Autotransplantation of a supernumerary premolar to the anterior region**
A. Frontal view of a 21-year-old man at first examination. His chief complaint was the replacement of an anterior bridge with an implant. The left central incisor was avulsed due to a traumatic injury and replaced by a Maryland bridge. B. A supernumerary tooth was found between the right second premolar and the first molar. The extracted donor tooth shows a root form optimal for transplantation. When the flap was raised, it was realized that the alveolar ridge was too thin to accommodate the donor tooth. C. The buccal bone plate was removed first and stored extra-orally in saline during socket preparation and replaced upon the transplant. The flap was sutured tightly around the transplant, and the transplant was splinted to adjacent tooth with wire and resin. A root canal treatment of transplants was started two weeks after surgery and finished with gutta-percha three months later. D. Buccal and palatal view six years after transplantation. Tooth mobility and gingival probing depth is normal. On the radiograph a normal PDL space is found around the root and no sign of progressive root resorption.

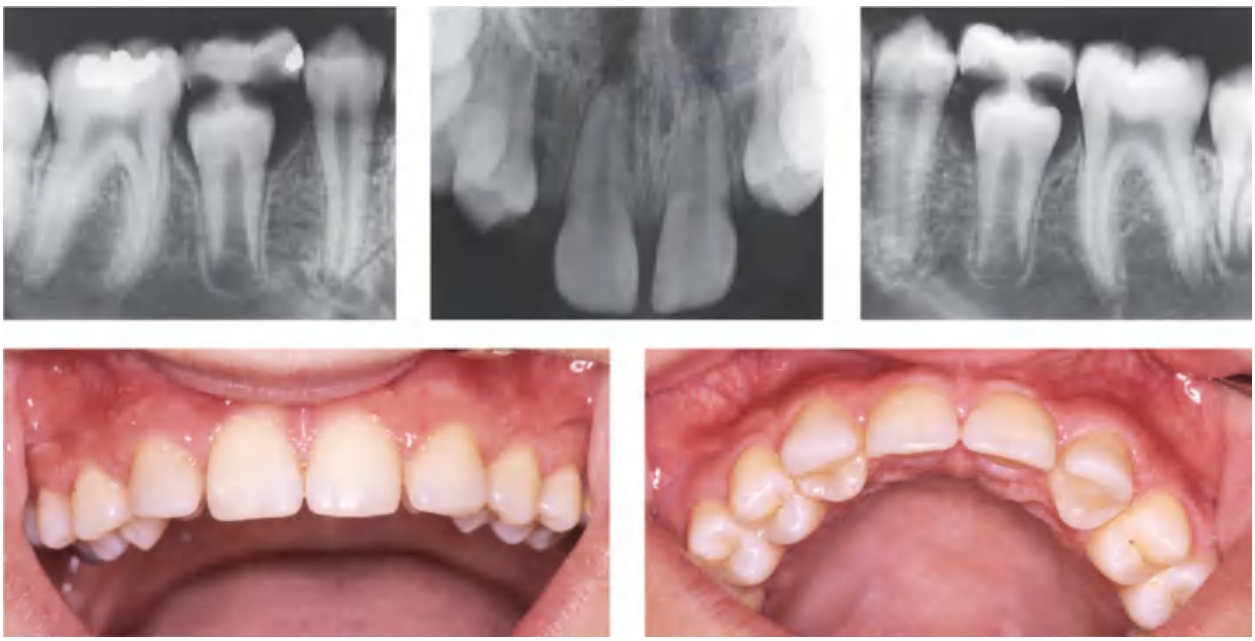

**Fig. 33.17** Maxillary lateral incisors replaced by mandibular second premolar transplants. This patient suffered lateral incisor and canine aplasia. Orthodontic evaluation showed that mandibular second premolar transplantation to the lateral incisor position, together with anterior orthodontic movement of the distally placed teeth was indicated in order to compensate for the missing teeth. The two mandibular premolars are at an optimal stage of root development, with three-quarters of the anticipated root length. The clinical condition is shown 5 years after transplantation and after completion of orthodontic treatment.

**Fig. 33.18 Autotransplanted premolars restored with a porcelain laminate veneer**
Two restored central incisors were replaced with 2 second maxillary premolars. The transplants were later restored with porcelain laminate veneers.

**Final result**
The transplants, after grinding were restored with porcelain laminates.

adjustment of the transplant is necessary prior to definitive restoration. In such cases, the period of retention must be complete before preparation for veneers. Otherwise, there is a risk of tooth migration. Alternatively, a retention wire should be applied palatally to assure transplant orientation even after restoration.

Other problems that also present in these cases concern the proximal extension of the preparation to ensure adequate

ceramic bulk to withstand occlusal loading during function, as well as the extent of reduction of the palatal cusp to ensure occlusal stability. It is still a matter of debate whether a bulky palatal cusp invites lingual pressure that could force the transplant facially or whether occlusion in the central fossa stabilizes the tooth. Esthetic demands might well dictate cusp reduction. Whatever decision is made with regard to tooth preparation, caution must be exercised if dentin is exposed during tissue reduction. In such cases, it is advisable that the exposed dentin be treated with a dentin-bonding agent and a thin layer of composite resin immediately after preparation and prior to impression taking in order to seal dentinal tubules until veneer cementation.

The following procedures have been suggested for detecting exposed dentin: search under magnification or with an explorer where dentin feels and looks smooth compared to a roughened, prepared enamel, or by a 5-second etch where enamel looks frosty and dentin dull. Provisional restoration would seem appropriate in such situations; also, in the case of palatal-occlusal reduction (i.e. a reverse 3/4 crown) in order to maintain interdental and intermaxillary relationships. When provisional restorations are indicated, they can be retained either as a resin splint by spot-etching the prepared and adjacent teeth or as a provisional veneer with a eugenol-free luting agent (26–28).

In unpublished clinical material, porcelain veneers were used to restore autotransplanted premolars. The conservative approach which was necessary in order to avoid exposure of post-transplantation dentin often demanded an unfortunate compromise in porcelain construction. It is, therefore, suggested that if crown anatomy demands such compromises, the graft be endodontically treated when adequate root development is achieved.

## Combined orthodontic closure and autotransplantation

In some cases, it might be an advantage in the case of loss of two incisors to close one of the sites and autotransplant the second (Fig. 33.21).

## Autotransplantation of teeth with completed root formation

Recently a technique for autotransplantation of teeth with completed root formation has been used in the initial or late treatment of acute dental trauma (29, 30) (Figs 33.20 and 33.21).

In this technique, the socket is prepared non-traumatically with surgical burs to a size which is slightly larger than the donor tooth. The graft tooth is luxated and extracted after incision of the periodontal ligament with a thin scalpel blade and immediately transferred to the new socket and placed slightly out of occlusion. The graft is splinted for 8 weeks with a flexible wire splint and an acid-etch/composite technique.

**Fig. 33.19 Autotransplantation of two premolars with complete root formation to the central incisor region**
This 14-year-old girl had lost her two central incisors due to progressive root resorption subsequent to replantation after avulsion. In conjunction with orthodontic reduction of a maxillary overjet, two second mandibular premolars were extracted and transplanted to cental incisor region.

**10-year follow-up**
The clinical and radiographic condition is shown 10 years after transplantation. The transplanted teeth have been treated endodontically and later restored vith post-retained crowns.

**Fig. 33.20 Autotransplantation of an intruded central incisor with completed root formation**
Radiographic and clinical condition at the time of injury in a 32-year-old woman. There is extrusion of the right maxillary lateral incisor with associated gingival laceration. The right central and left lateral incisors have been avulsed and the left central incisor intruded with severe crushing of the associated alveolar process.

**Treatment plan**
Treatment includes autotransplantation of the left central incisor to a site with least bony injury in order to avoid later bone loss (i.e. the right central incisor socket) and repositioning of the extruded right lateral incisor.

**Clinical and radiographic condition after treatment**
The teeth are repositioned, and the gingiva sutured. The teeth are splinted with flexible wire and an acidetch/composite resin technique for 3 months. The root canals were initially treated with a calcium hydroxide dressing for 3 months prior to final root filling with gutta percha and sealer.

**Follow-up**
The clinical condition is shown 4 years after completed prosthetic treatment. Radiographic condition 6 years after completed treatment shows slight apical repair related resorption of the transplanted central incisor.

**Fig. 33.21** A. A maxillary right second premolar was transplanted to replace the missing maxillary left central incisor. B. The tooth was rotated during surgery 180 degrees and positioned superiorly in relation to other incisors in order to be accommodated at the recipient site. Nine months after surgery the transplant was reshaped to central incisor's morphology using a direct composite build-up. C–D. After orthodontic treatment, which also included alignment of the transplanted premolar in the maxillary arch, the composite build-up was replaced with a porcelain veneer. E. Excellent esthetics were obtained after orthodontic and prosthodontics treatment following transplantation.

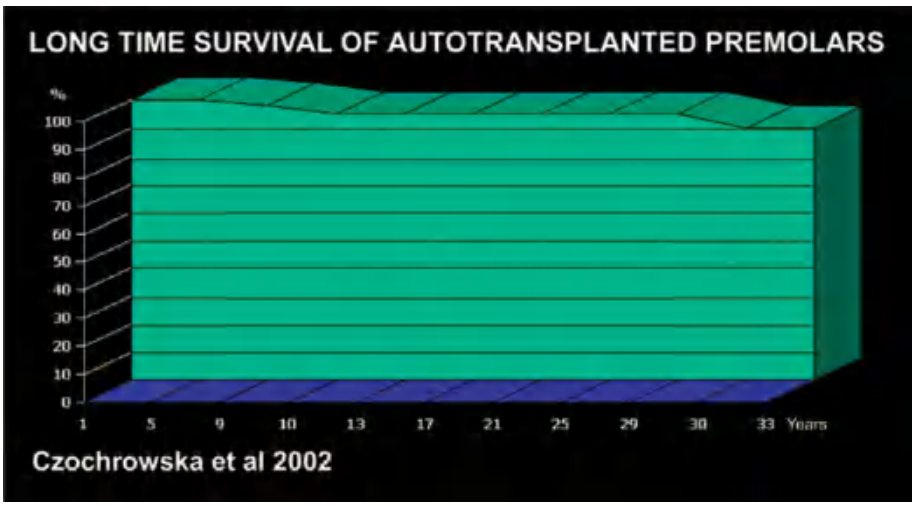


**Fig. 33.22** Long-term prognosis of autotransplanted premolars. Source: Reproduced from (42) with permission.

After 3 to 4 weeks, endodontic treatment can be performed using an interim dressing of calcium hydroxide followed by a gutta-percha root filling (see Chapter 25).

In cases of acute trauma, this technique can also be used to place the tooth in a site of less injury.

In a recent study, the gingival and periodontal condition of transplanted premolars to the anterior region by Czochrowska et al. was compared to adjacent natural incisors (41). Identical gingival and periodontal conditions were found, a finding which is rare with regard to implants. Furthermore, the esthetic outcome of autotransplanted premolars was analyzed. In a study of 22 transplants, a satisfactory or acceptable esthetic outcome was found in 86% of the cases (40).

## Prognosis

In several studies a success rate (i.e. without progressive root resorption) of 87–93% was found for transplants and a tooth

**Fig. 33.23** Predictors for successful autotransplantation of premolar.

survival rate of 90–98% (Table 33.1) (13–16, 20, 40–46, 71, 81). With respect to pulpal and periodontal healing, it should be, however, mentioned that 10–20% of cases may show complications at a later date, such as primary or secondary pulp necrosis as well as external (cervical) root resorption, which may require additional treatment. Another factor to consider is arrest of root development which may influence the prognosis if teeth are transplanted in early root development stages (16).

In long-term studies of autotransplanted teeth with immature root development, where data allow *life table analysis* of the prognosis, there appears to be almost identical survival: *5-year survival* ranges of 98–99%; and *10-year survival* of 87–95% and 80% (20–23). The longest study of autotransplanted premolars showed 90% survival after 39 years, a record that neither prosthetics nor implantology can surpass (42) (Fig. 33.2). This survival rate thus appears to be equal to

**Table 33.1** Success rate of transplanted premolars to the anterior region.

| | No. of teeth | Success rate (%) | Survival rate (%) |
|---|---|---|---|
| Andreasen et al. (13–16) 1990 | **33** | **88** | **98** |
| Kristerson & Lagerström (20) 1991 | **23** | **87** | **96** |
| Kugelberg et al. (43) 1994 | **31** | **87** | **90** |
| Czochrowska et al. (40, 41) 2000 | **45** | **93** | **93** |
| Kvint et al. (65) 2010 | **24** | **100** | **100** |

alternative treatment approaches, such as certain fixed prosthetic appliances and implants (see Chapters 31 and 35).

With respect to the restorative procedures presently available, little is known of long-term success when applied to autotransplanted teeth.

In conclusion, autotransplantation of premolars (and sometimes diminutive molars) when indicated due to problems of crowding offers a unique treatment possibility of restoring the bone, gingiva and the tooth in a trauma region. Such a treatment solution should therefore be considered in all cases of early loss of anterior teeth and when found indicated performed with due regards to the prognosis related factor.

## Predictors for healing

The strongest predictor for optimal healing appears to be the *stage of root development* and *apical diameter* at the time of grafting (14–16, 37, 38) (Fig. 33.23). This factor appears to influence both pulp and periodontal healing and to a less degree also graft survival (Figs 33.24 and 33.25).

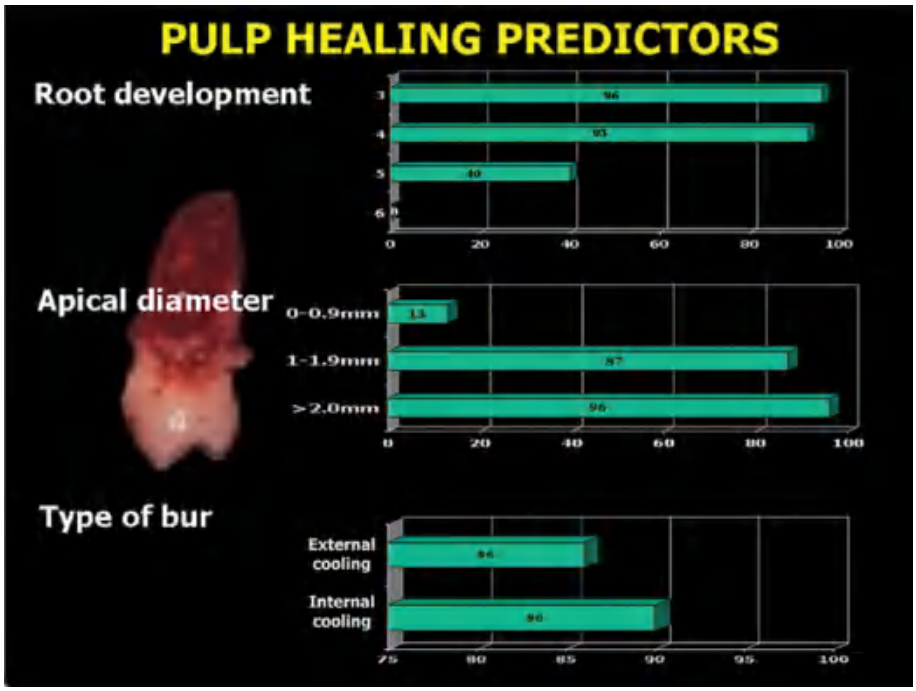


**Fig. 33.24** Predictors for pulp healing.

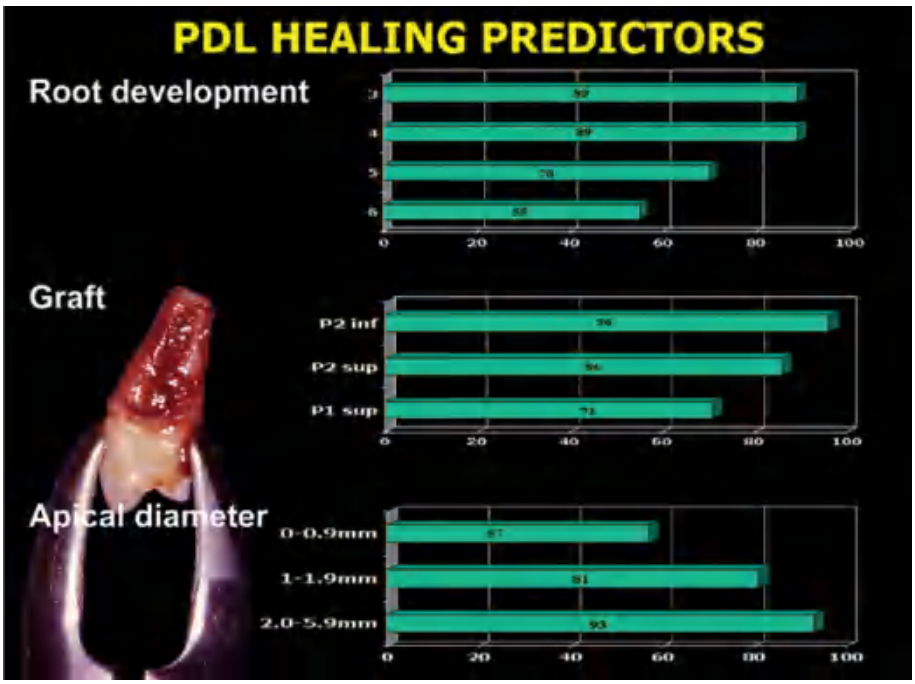


**Fig. 33.25** Predictors for PDL healing.

A very significant factor appears to be the *experience of the oral surgeon*: individuals who had performed more than 25 autotransplants had a significantly higher success rate (47, 48).

Another parameter shown to influence graft survival was a structured *follow-up regimen* to monitor periodontal and pulpal complications. Thus, it was found to be able to increase the survival rate of autotransplants in 30% (47, 48).

Finally, the type of graft is of importance, with maxillary first premolars having significantly less healing potential (14, 15).

## Essentials

### Indications

- All cases where an early loss of permanent incisors could be anticipated – in that case a panoramic radiograph should be taken in order to detect possible tooth grafts.
- The advantage of tooth autotransplantation in comparison to alternative treatment procedures is the establishment of a functional tooth unit, which allows tooth eruption and development of the alveolar process.

### Graft selection

#### Central maxillary incisor region

- All premolars except maxillary first premolars, sometimes canines or maxillary third molars of size small

#### Lateral maxillary incisor region

- Only mandibular first premolars

#### Canine region

- All premolars except maxillary first premolars

### Stage of graft removal

- $^{3}/_{4}$ or full root development with wide open apex is optimal; however, full root development with subsequent endodontic therapy is also an option.

### Surgical procedures

- Non-traumatic surgery and experience is the key to success.
- Closed procedure in cases with adequate bone support (Fig. 33.9).
- Open procedure in cases with composite bone-tooth grafting (Fig. 33.10).
- Non-traumatic graft removal and graft insertion is essential for successful long-term results.

### Orthodontic treatment

- If orthodontic treatment is necessary, it can be performed when revascularization of the pulp has been completed.

### Prosthetic restoration

- Autotransplanted teeth can be restored by a composite technique after 3 months and at a later age with a laminate veneer with a porcelain laminate technique.
- Avoid preparation if possible.

### Prognosis

- 5-year graft survival: 98–99%
- 10-year graft survival: 87–95%

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# 34

# Strategies for Treatment of Tooth Loss after Trauma to the Anterior Region of Young Patients

L. Andersson, B. Malmgren, O. Malmgren & S. Storgård Jensen

## Introduction

Traumatic dental injuries (TDI) most often affect the anterior region of the maxilla of young growing individuals (1–3). Tooth losses in this region in young growing patients present special problems often with esthetic consequences. Leaving the space without treatment may result in atrophy of the alveolar process and disturbance of the growth and development of the alveolar bone in that region. Implants are contraindicated until growth is completed and fixed partial dentures may interfere with growth in children. Instead an active approach aiming at promoting preservation, growth and development of all tissues in the affected region is recommended. A multidisciplinary approach in planning is important and many considerations have to be taken. Timing in relation to the growth status of the patient is important.

When facing a tooth loss in the anterior region of the maxilla in a growing patient, first of all a decision must be made to either close the space or keep the space and replacing the lost tooth, which is often a difficult multidisciplinary decision involving many specialists such as orthodontists, pediatric dentists, surgeons and prosthetic dentist. Different treatment alternatives must be considered such as prosthetic treatment, orthodontic space closure, autotransplantation of another tooth to the anterior region or if growth is in the final stages, if and when implant treatment shall be considered.

## Growth of the anterior maxilla

It is well known that the craniofacial growth pattern follows the general body height measurements from 5 to 19 years of age with a marked increase during puberty (4). The development is however very complex, and the individual variation is considerable. The increase in height of the maxilla is due to sutural growth and re-modelling of the maxillary bone with resorption on the nasal side and apposition on the oral side (4). The increase in growth velocity during puberty may fall within the age of 13 years in most individuals, while in others close to 16 years or even later. The craniofacial growth acceleration in males seems to coincide more closely to that of the body height than in females where the craniofacial growth spurt occurs somewhat later than that of the body height (5). On average it appears two years earlier in girls than in in boys. The anterior maxilla is mainly growing horizontally by bone apposition buccally and tvertically by the eruption of teeth. When teeth are lost there is loss of vertical and horizontal growth of the alveolar process. Moreover, rapid resorption of the alveolar crest is seen if the space is left untreated (6, 7). The bone resorption is especially rapid in the horizontal dimension and more in the buccal bone than in the palatal (6, 7) (Fig. 34.1). When anterior teeth are lost, the vertical growth potential of the alveolar process is reduced. The result might be severe inhibition of the development of the maxillary alveolar process, especially after multiple tooth losses (Fig. 34.2).

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**Fig. 34.1 Horizontal atrophy of the alveolar process after tooth loss**
Early loss of central incisor which has resulted in atrophy of the alveolar process over time.

For this reason, every effort should be made to save teeth to enable growth and development of the alveolar process. Even teeth with dubious long-term prognosis after trauma should be saved to minimize resorption of the alveolar bone crest (Figs 34.3–34.5) and root remnants can be left to preserve the alveolar process. Even ankylotic teeth undergoing replacement resorption can contribute to the preservation of the alveolar process during the osseous resorption. In young growing patients with infraocclusion decoronation must be carried out to prevent infraocclusion (see Chapter 32) (8, 9).

## Prosthetic alternatives in the early mixed dentition

The majority of traumas to the permanent dentition affect children between 8 and 10 years of age, which is in the mixed dentition. Replacement after loss of permanent incisors in this period is complicated. Erupting teeth change the position of the neighboring teeth as the alveolar process is developing and the growth of the jaws is intense (Fig. 34.6).

Therefore, fixed bridges are not suitable. They may interfere with growth of the jaws and development of the dentition. In order for the canines to erupt, the erupting canine will have to push the root of the lateral incisor mesially during its eruption. If the lateral incisor is included in a fixed bridge the canine cannot erupt and may be impacted. For this reason, fixed bridges should not be used until the canine is fully erupted. Instead removable plates are the most common appliances. They can be used until the canines are fully erupted (Fig. 34.7). Due to the risk for tipping of the adjacent teeth if a central or later maxillary incisor is lost, stops should be applied mesial and distal to the lost tooth. Removable appliances are however difficult to wear for the young child before the first permanent molars are fully erupted and when the primary teeth are shedding.

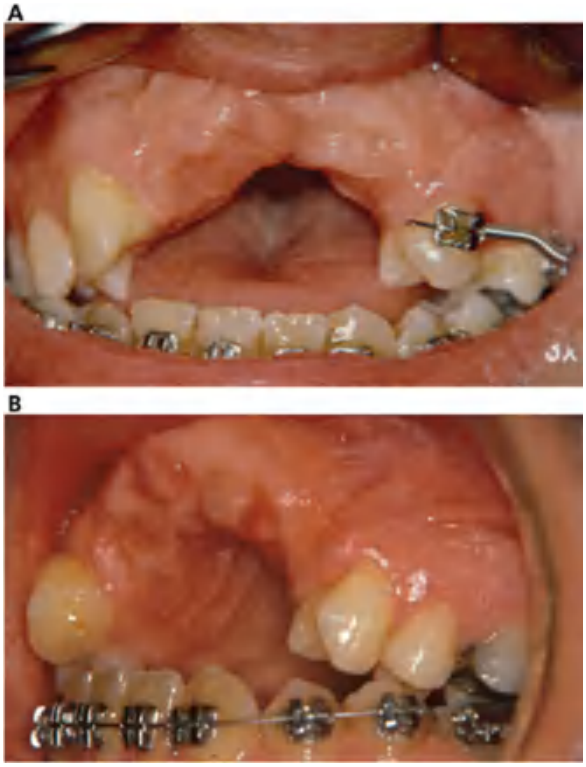



**Fig. 34.2 Vertical loss of growth of the alveolar process after tooth loss**
Anterior maxillary region of an 18 year old girl who lost five anterior teeth due to trauma when she was 7. She was treated only with several removable partial dentures during eleven years. Note the loss of vertical growth of the alveolar process caused by the loss of incisors. A. Anterior view B. Lateral view.

An alternative therapy is a lingual arch wire soldered to bands on the second primary molars, with a prosthetic tooth fixed to the arch wire (Fig. 34.8). To use bands on the second primary molars is a way to avoid interference with permanent molar eruption. Stops should be placed on both sides of the space to avoid drifting of teeth. Occlusal stops are necessary to avoid pressure by the prosthetic tooth on the alveolar ridge (Fig. 34.8). Later when the first molars are fully erupted a similar appliance fixed to molar bands can be used.

When the canines are fully erupted or when there is no risk of interference between the lateral incisor and the canine, an acid etch bridge can be bonded to the adjacent teeth. Later conventional bridges or insertion of an implant can be performed. (See Chapters 31 and 35.)

## Orthodontic space closure

Early loss of upper permanent central incisors is a serious clinical problem. The treatment plan in each situation depends on many factors such as the number of missing teeth, occlusion, space conditions, age, facial morphology, growth pattern, tooth morphology, and the need for orthodontic treatment in general. The choice of treatment is especially challenging in the early mixed dentition. It is therefore

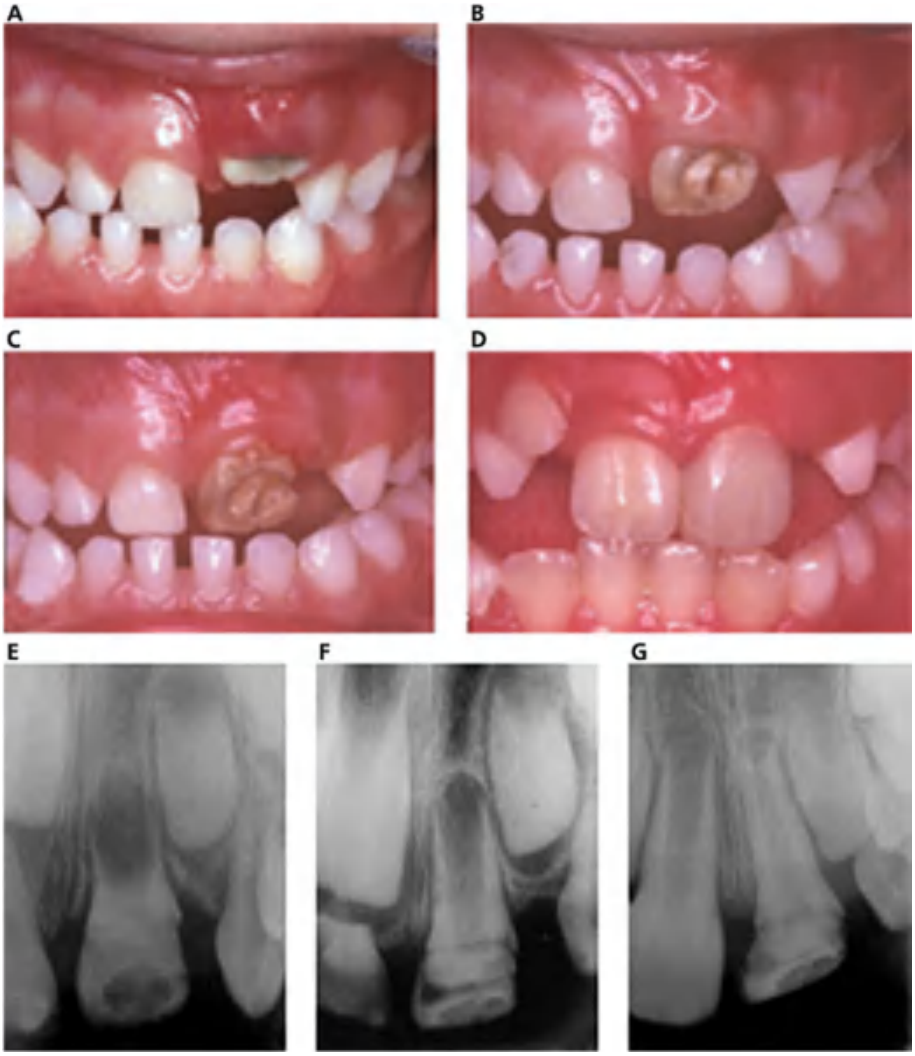



**Fig. 34.3 Premature eruption of a hypomineralized crown of a permanent incisor**
A. 3.5-year-old girl who intruded her primary first left incisor at the age of 18 months. There is premature eruption. B. The hypomineralized crown was restored with composite later to prevent infection of the pulp through damaged hard tissues. C, D. Situation at the age of 5 years before new restoration and at the age of seven years after new restoration. E–G Radiographs at 4, 5 and 7 years of age. Source: Reproduced from (33).

easier to postpone definitive choice of treatment to the late mixed or permanent dentition stage is reached. The problem is to avoid side effects during the development of the dentition. The permanent maxillary lateral incisors are during development located behind the central incisors. When a central incisor is lost, and the lateral incisors erupt, the result can be a rapid mesial tipping of the lateral and the uninjured central incisor and a severe midline shift, which can jeopardize later careful treatment planning (Fig. 34.9). There is also a risk of unfavorable tipping of uninjured lateral or central maxillary incisors and midline shift during eruption of the upper canines. Therefore, space maintenance is important during development of the dentition.

Space closure by moving a lateral incisor mesially to mimic a central incisor is often questioned due to the treatment complexity, the risk of space reopening, the increased loading on the root of the lateral incisor when it is supporting a larger crown, and the quality of the esthetic result. The form of the lateral crown, the dental arch symmetry and the lip seal are important factors for an acceptable result. The form of the lateral and cuspid has to be build up with composite in combination with grinding. The gingival margin has to be recontoured to harmonize with the dentition. The advantage is that treatment can start early and that a new alveolar process with attached gingiva and an intact interdental papilla will be formed. The teeth move along with the bone and surrounding tissues during growth of the dentofacial complex. The final result is achieved after the orthodontic treatment and malocclusions can be treated simultaneously (Figs 34.10 and 34.11).

Space closure is of prime importance in patients with maxillary arch length deficiency in Class I and II malocclusions and crowding. Treatment should be planned in the late mixed dentition. It is then possible by extraction or interproximal reduction of deciduous molars to guide molars, premolars and canines mesially. An intact dental arch can then be obtained with minor or no appliance therapy. Space closure for a missing maxillary central incisor also implies mesial positioning of the maxillary cuspid in the lateral incisor region. Periodontal status has been reported to be better in patients with closed lateral incisor spaces than in patients

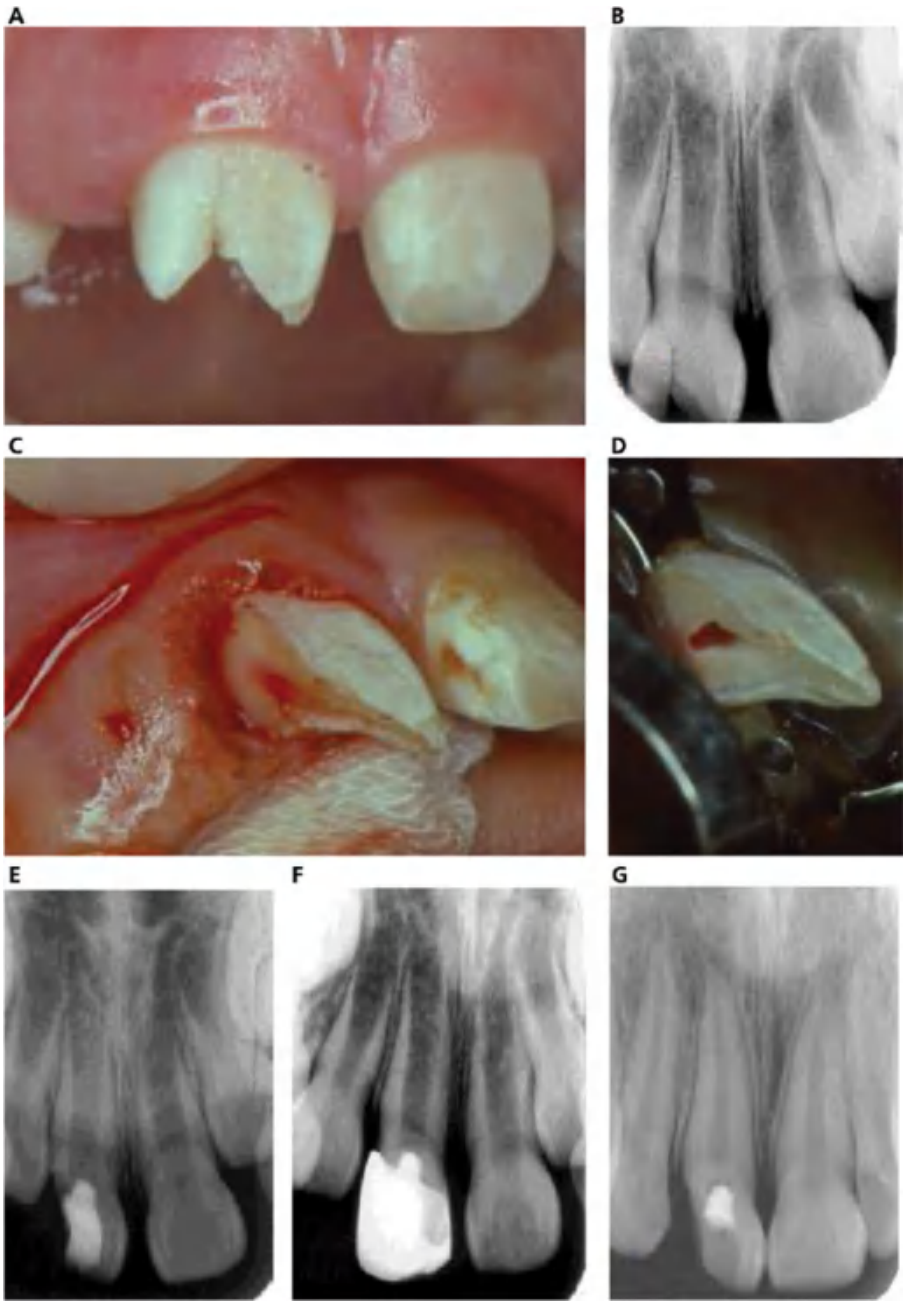



**Fig. 34.4 An immature tooth with a complicated crown-root fracture**
A–D. A 7-year-old girl with a complicated crown-root fracture. Partial pulpectomy was performed. E. Radiograph immediately after partial pulpectomy. F–G. Radiographs at 2 months after injury and a follow-up 5 years later. Source: Reproduced from (33).

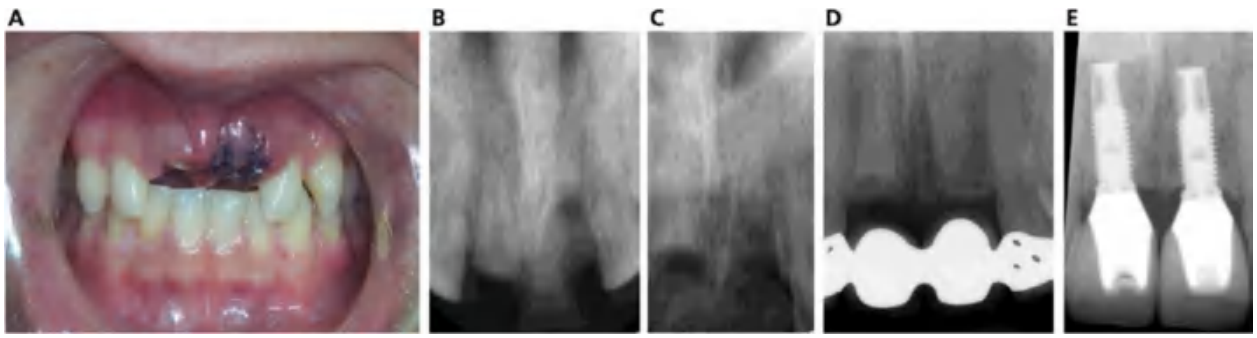



**Fig. 34.5 Decoronation after crown-root fractures of two central incisors**
A and B. Time of injury. C. The root of the right incisor was lowered under the bone level 4 days later. D. 2.5 years after injury. New bone formation coronal of the submerged roots. E. Insertion of implants. Source: Courtesy of Dr. Maria Andersson.

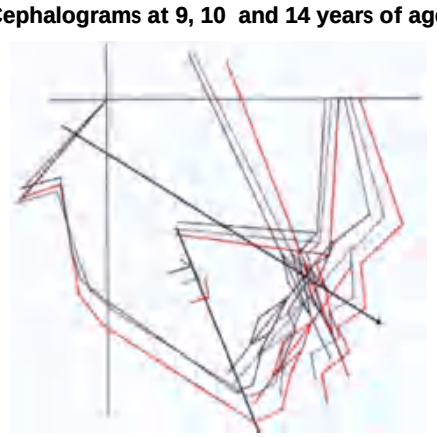



**Fig. 34.6 Growth of the face**
Computer drawings showing the normal forward downward growth of the face. The cephalograms were taken at 9, 10 and 14 years of age.

**Fig. 34.7 Space maintainer**
A. A removable space maintainer for a lost central incisor in the mixed dentition. Note the occlusal stops and the mesial and distal stops in the incisor region. B. A fixed space maintainer with bands on the second deciduous molars with a soldered lingual arch is an option.

with prosthetic lateral incisors (10). Mesial movement of a lateral with fixed appliance to replace a missing upper central incisor is complex (11). In a follow-up study of 20 consecutively treated patients it was found that most patients (85%) were satisfied regarding the esthetic result, but only 50% of the professionals (12). Crown form and inclination of the lateral incisors and canines must be favorable.

**Fig. 34.8 Space maintainer**
After eruption of the first bicuspids, and during eruption of the canines a lingual arch can be soldered to molar bands.

However, when two central incisors are lost, orthodontic space closure may be a suitable symmetric result, given that prerequisites mentioned above with regard to Angle class, crown shape and symmetry are acceptable.

## Important consideration when planning for later implant treatment

Although implant treatment is contraindicated in still growing patients it is important to have knowledge when growth is completed, and implant treatment possibly can be started and how to assess this and the risks of installing implants too early in a young person. Osseointegration of dental implants has been shown to be a fusion between a titanium implant and the bone (13) and also described as functional ankylosis (14) and experimental studies have demonstrated that placement of implants in a developing alveolar process will have similar implications as ankylosed teeth, namely local arrest of the alveolar growth, infraposition of the implant, tilting of neighboring teeth, and deep periodontal and peri-implant pockets (15–18). Consequently, there is consensus not to place dental implants in growing individuals (19). Based on this, an intense search is ongoing for the ideal measure to indicate arrested growth of the alveolar process and, thereby,

**Fig. 34.9 Tipping of central incisor**
Mesial tipping of a left maxillary central incisor after severe damage to the right central incisor in an 81/2 year-old boy. The right maxillary central incisor was severely damaged due to an early trauma in the primary dentition and was later extracted. Both the right lateral and the left central incisor tipped towards the space for the lost incisor. Note the right erupting cuspid pushing the left lateral in a mesial direction.

**Fig. 34.10 Space closure**
Closure of space resulting from avulsion of the permanent left central incisor. A. Before orthodontic treatment. B. After orthodontic treatment. The lateral has been moved mesially and reshaped with composite.

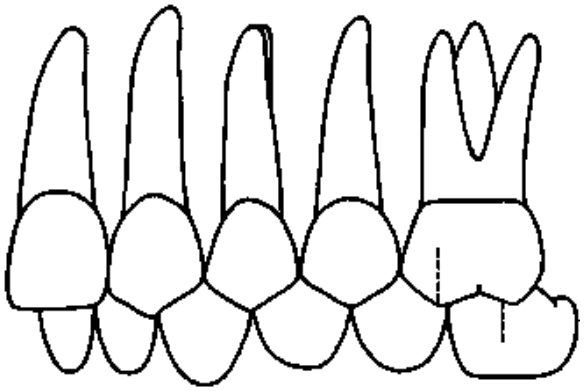


**Fig. 34.11** Schematic drawing of a postnormal occlusion and space closure with a lateral incisor substituting a central incisor.

for placement of dental implants. Clinical determinants like chronological age, completed skeletal growth as measured by stability in body height, and completed eruption of permanent teeth, as well as radiographic measures like hand wrist x-ray, and superimposed lateral cephalograms have been proposed (Figs 34.12 and 34.13).

Chronological age is easy to measure and communicate to the patient, and the average degree of implant infraposition has been shown to decrease with increasing age (20). However, even mature adults (>40 years of age) suffer the risk of implant infraposition of >1.5 mm when evaluated 5 to 18 years after implant placement (21). Chronological age is therefore considered a poor predictor for the risk of implant infraposition.

The degree of infraposition of dental implants has been directly correlated to remaining increase in body height (23). However, even patients showing no signs of ongoing skeletal growth may develop an infraposition of up to 2.2 mm (22, 23). Therefore, arrest in skeletal growth cannot be used as the sole measure to exclude the risk of implant infraposition.

Completed eruption of permanent teeth except for the third molars has also been investigated as a clinical sign of arrested craniofacial growth and thereby and indicator for the possibility of placing dental implants. However, significant growth of the alveolar process and progressing infraposition of implants have been observed in this patient population too (24).

Hand and wrist radiographs are used to estimate patient's skeletal age and especially to predict the adolescent growth spurt (25, 26). However, they are not ideal to determine cessation of craniofacial growth as the sole method.

An explanation for late infraposition of implants placed in adult patients with closed epi- and diaphyses as documented by hand and wrist radiograph and with arrested skeletal growth as documented by repeated body height measurements

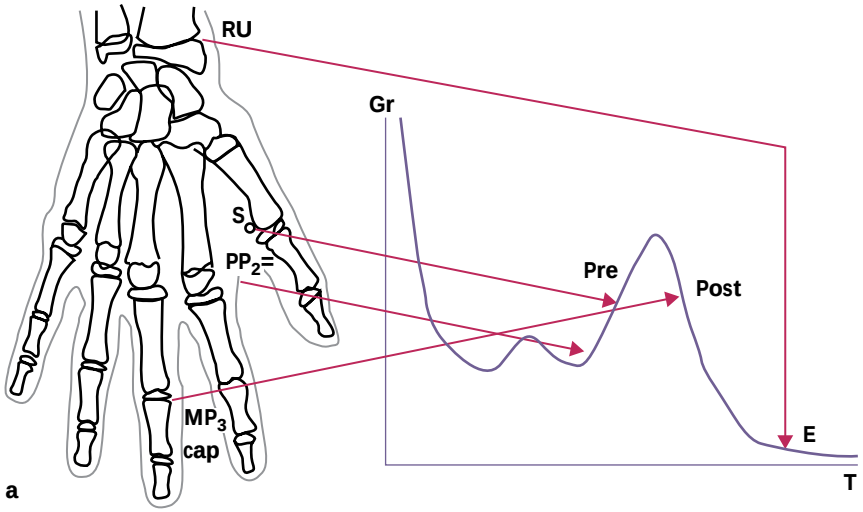



**Fig. 34.12 Growth curves**
A. Curve showing growth (Gr) over time (T) with different radiographic signs related to growth curve. E is indicating end of growth. Ossification of the bones of the hand and wrist is related to the skeletal growth curve and may be evaluated using a hand-wrist radiograph. When epi- and diaphysis of the radius and ulna have fused (Ru), remaining skeletal and cranio-facial growth is limited. B. Radiograph showing fusion not fully completed of epi-diaphyse of radius and ulna, indicating there will still be growth expected.

**Fig. 34.13 Cephalometric used in growth analysis**
Lateral cephalometric radiographs taken at least one year apart can be superimposed to evaluate signs of continuous growth of the anterior alveolar process and/or eruption of the incisors. This radiograph indicated good support between upper and lower incisors. However, the actual contact should be confirmed clinically.

is most likely the phenomenon of slight continuous eruption. This takes place in the dentition even in adults. Continuous eruption has been analyzed in long-term studies of normal patients, which revealed an annual vertical eruption of 0.07–0.1 mm of incisors in adults aged 20–40 years (27–30) (Fig. 34.14).

Superimposed lateral cephalometric radiographs taken with intervals of a minimum of 1 year have been shown to be a good predictor of cessation of skeletal growth and continuous eruption (31). To reduce the risk of continuous eruption of neighboring teeth it is highly recommended that any orthodontic treatment prior to implant therapy in young individuals should aim at establishing ideal contacts between upper and lower incisors.

Based on the available evidence, no specific age, clinical or radiographic measure can guarantee the clinician or patient against the risk of infraposition of dental implants – especially not in the upper anterior region. To reduce this risk, the following is suggested:

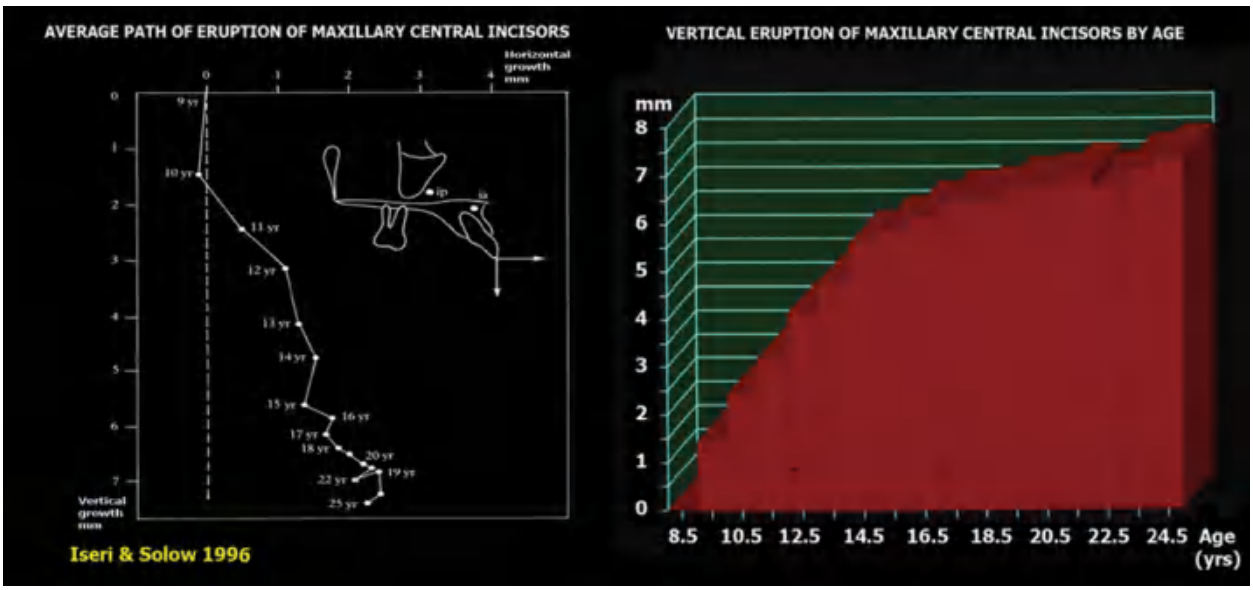
- Repeated lateral cephalographs demonstrating arrest of craniofacial and alveolar growth; or
- Terminated growth in height (as demonstrated by two repeated measurements at least one year apart); combined with
- Ideal occlusal contacts between upper and lower incisors.

It may be speculated that cases where the entire maxillary front has been lost due to TDI safely can be treated with dental implants at any phase of skeletal maturity, since continuous growth of the alveolar process of the anterior maxilla is mainly related to the eruption of teeth. This may be the case. However, it is so far scientifically poorly documented. Implant treatment procedures are described more in detail in Chapter 35.

## Autotransplantation

Autotransplantation (transplantation within the same individual) of another tooth, usually a premolar, to the space is a biologically appropriate alternative to support the development of normal tissues in the region subjected to tooth loss promoting normal growth and development of the alveolar process. Not only the development of alveolar bone is promoted but also the soft tissues such as the keratinized gingiva and papillas, which is important for esthetics, is promoted by transplantation of a tooth to the area of tooth loss and good long-term results has been reported after autotransplantation to the anterior region after trauma (Fig. 34.15) (see Chapters 34 and 36).

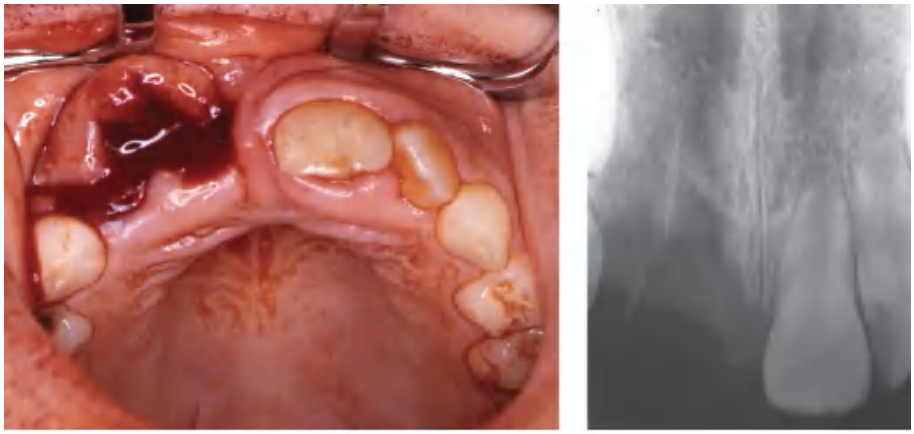
When a tooth is transplanted the vascular supply is immediately lost. Studies of transplanted teeth have shown that young teeth with ongoing root development and open apex can revascularize. Successful revascularization and healing is related to the stage of root development. In order to optimize conditions for revascularization, transplantation should ideally be carried out before the root reaches full root length and when the root apex is still open. Teeth with fully developed roots and a closed apex cannot be expected to




A B

**Fig. 34.14 Estimate of expected infraposition**
Estimate of expected infraposition for anterior dental implants inserted in children and young adults. A. Based on data from a longitudinal growth study of 14 girls from 8–25 years, the expected residual vertical and horizontal component of continued eruption of the upper central incisors can be estimated in relation to age for girls. This measure is also an estimate of the amount of infraposition to be expected in the adult for a dental implant inserted at the age in question. The graph is constructed according to the subject's age, and the expected infraposition can be determined from the vertical scale. Somewhat larger values can be expected for boys. B. Graphic illustration of vertical eruption (i.e. expected infraposition) related to age. Source: Modified from (16).

**Fig. 34.15 Maxillary lateral and central incisor replacement with combined orthodontic space closure and premolar transplantation**
Two incisors have been avulsed in a 9-year-old girl. The treatment plan is to orthodontically move the canine into the lateral incisor position, the first premolar into the canine position, and finally to transplant the mandibular second premolar into the central incisor position.



**Restoration of the anterior segment**
The premolar has been moved into the canine position, rotated 45° and the oral cusp reduced. The canine has been moved into the lateral incisor position and its cusp and proximal surfaces reduced. The transplanted premolar has been restored with composite.

revascularize but can still be transplanted provided root canal treatment is performed. Autotransplantation should therefore ideally be planned in a multidisciplinary approach with, in addition to the surgeon, orthodontist, pediatric dentist and endodontist. The orthodontist has a central role to advise on a suitable transplant and perform preoperative and postoperative orthodontic treatment when required. It is important to have a thorough preoperative radiographic examination of both the donor and recipient areas to estimate root anatomy, root development and space conditions. Estimation of the degree of root development is the key factor for correctly timing the transplantation. A root development of 70–80% seems to be optimal. It is important that the surgeon has a full understanding of the highly sensitive tissues (PDL and pulp) involved. These are cases which should be referred to surgeons who are specially trained in this methodology. In recent decades, multidisciplinary teams have been formed and there seems to be an increased interest for this treatment option in young growing trauma patients. Transplantation procedures are covered in more detail in Chapter 33.

## Treatment should be related to the growth status of the patient

As said from the above, the final choice of treatment after tooth loss in the growing patient is depending on many different factors and conditions. Treatment should always be carefully related to where the patient is in his/her growth status. Girls in general, are usually developing earlier than boys but the variation between individuals is large. For this reason, there must always be assessment for each individual undergoing treatment and related to the individual's growth curve. In summary, Fig. 34.14 may serve as a guide in treatment planning for the multidisciplinary team discussions:

In the *pre-peak period*, transplantation a good alternative on many occasions. Orthodontic space closure can be considered but only in suitable cases. Removable partial dentures (RPD) can be used as a temporary alternative. However, the alveolar process will not develop optimally when RPD is chosen over a long period of time.

In the *peak period* transplantation is still a good alternative. Acid-etch bonded bridges can be considered given the canine is fully erupted, but this choice will not develop the alvelolar process, which may atrophy. Orthodontic space closure can be considered in suitable cases.

In the *post-peak period* implant treatment and acid-etch bonded bridges can be considered. Fixed bridge constructions can be performed but is seldom chosen today in young patients.

## Essentials

Tooth losses after trauma presents special problems in the anterior region of the young still growing patient:

- Teeth contribute to the growth and development of the alveolar process of the anterior region.
- Extraction of teeth and of root remnants result in atrophy of the alveolar process and inhibited growth and development.
- Given not infected, keep teeth and root remnants, even if the prognosis for the individual tooth is poor, to preserve the alveolar process until growth is completed.
- Try to establish the growth status of the patient and plan the treatment according to this.
- Work in a multi disciplinary team of specialist to decide the most suitable treatment in each individual case.

## Treatment options

### Prosthetic treatment

- Avoid any fixed constructions until canine is fully erupted.

### Orthodontic space closure

- Take maxillary arch length into consideration
- Suitable cases are Class II and Class I cases with crowding
- Similarities in crown shape and size between central and lteral incisors
- Unilateral closure may result in esthetical compromises.

### Autotransplantation

- Selection of suitable tooth to be transplanted and timing of root development stage is important.

### Implant treatment (after growth is completed)

- Growth should be compeleted before implant treatment is considered

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# 35

# Implant Therapy in the Esthetic Zone

S. Storgård Jensen, V. Chappuis & D. Buser

## Indications for dental implants in trauma patients

Implant therapy is often indicated as permanent replacement of teeth lost due to traumatic dental injury. Over the last 40 years, implant dentistry has evolved to become a predictable standard of care, since the first description of osseointegration as the intimate contact between viable bone and a titanium implant surface (1, 2). However, before implant therapy is considered, it is mandatory to evaluate whether the traumatized tooth can be restored predictably as described in Chapters 29–31. If the traumatized tooth was avulsed and never replanted, if later removed due to complications, or if considered irrational to treat, implant therapy may be considered as one of the options for tooth replacement. The most frequent dental traumas leading to tooth loss are described in the previous chapters.

The present chapter will focus on implant therapy in the anterior region only, and the reader is referred to dedicated textbooks for a complete description of the field of implant dentistry.

Tooth replacement in the anterior maxilla is challenging, since poor judgment during preoperative analysis, the selection of an inappropriate treatment approach, or the inappropriate execution of clinical procedures can lead to esthetic short-comings, esthetic failures, or even esthetic disasters. Esthetic complications with implant-borne restorations are either iatrogenic or anatomic in nature (3). Iatrogenic causes include implant placement in a malposition, the selection of an oversized implant, such as wide-platform implants, or the selection of an inappropriate treatment approach overstressing the biologic healing capacity of the oral tissues. Anatomic causes include bone deficiencies in the horizontal or vertical direction, combined bone and soft tissue defects, or implant sites with multiple missing teeth leading to the placement of adjacent implants.

These considerable challenges make implant placement in the anterior region one of the most demanding procedures in implant dentistry (3, 4).

To successfully meet the challenges of esthetic implant dentistry in daily practice, a team approach is advantageous and highly recommended. The team includes an implant surgeon, a restorative dentist and a dental technician who preferably all have advanced knowledge and clinical experience. A successful implant surgeon working in the esthetic zone must have a thorough understanding of tissue biology, a good surgical education, and the skills to perform precise and low-trauma surgical procedures, and a large patient pool providing sufficient surgical experience of esthetic implant placement. In addition, especially for young patients with missing anterior teeth, a good collaboration with an orthodontist with a profound understanding of esthetic dentistry is indispensable (5, 6).

## Treatment planning

A thorough and systematic patient assessment is mandatory before any implant treatment is planned. The assessment includes:

(1) Anamnesis
   a. Patient history incl. patient expectations
   Initiation of therapy starts with an understanding of the patient's desires. In most cases, the patient's primary demand is an esthetically pleasing tooth replacement offering a nice smile. Furthermore, the patient usually expects a lifelong treatment result with a minimum of

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maintenance. Finally, patients are usually concerned by the cost of the treatment. For the clinician, the reestablishment of esthetics and function requires knowledge of all treatment options. Conventional fixed partial dentures and implant-supported restorations should be objectively evaluated for their potential to provide long-term function and stability (see Chapter 31). Today, implant-supported restorations often represent the best solution since intact tooth structure and supporting tissues of neighboring teeth can be preserved (7) (see Chapter 31).

b. Social factors
c. Medical factors
d. Dental factors

Anamnestic information concerning social, medical and dental factors should reveal any absolute or relative contraindications to implant therapy. Patients suffering traumatic dental injuries are most often young and healthy individuals. However, a complete registration of health status, medication, allergies, smoking habits and alcohol consumption is indispensable. In addition, the patient should be aware of the importance of ideal oral hygiene and a good collaboration with the patients' general practitioner to ensure a good long-term prognosis of the treatment result.

(2) Clinical examination
   a. Extraoral
   b. Intraoral

   A general extra- and intraoral examination will assist the clinician in evaluating the potential need for adjunctive procedures prior to implant therapy such as scar tissue corrections, periodontal therapy, orthodontic treatment etc.

   c. Site specific
      – Hard and soft tissue volume
      – Restorative space

   The site-specific evaluation should reveal soft and hard tissue deficits at the implant site that may warrant adjunctive augmentation procedures. In addition, the status of the neighboring teeth should be thoroughly evaluated, since traumatic dental injury often affects the prognosis of neighboring teeth as well. Especially, the restorative, periodontal and endodontic status of the neighboring teeth should be examined. Finally, the restorative space for the future tooth replacement should be evaluated.

(3) Radiographic assessment (see Chapters 31 and 41)

   Periapical radiographs are indispensable for evaluating the bone height at the future implant site, and for the marginal bone level and endodontic status of neighboring teeth. Often supplemental CBCT will provide useful information about crest width of the alveolar ridge and will allow the clinician to evaluate whether a staged or simultaneous approach should be planned.

A useful tool and checklist for the preoperative evaluation can be obtained by using the SAC classification categorizing implant treatments into *straight forward*, *advanced*, or *complex* according to defined surgical and restorative parameters (8), and by performing the Esthetic Risk Assessment – a tool that can assist the clinician in identifying risk factors when planning implant placements in the esthetic zone (8).

## Timing of implant placement in relation to growth

Dental implants should only be placed in patients where craniofacial growth and, most importantly, growth of the alveolar process has stopped to reduce the risk of later infraposition of the implant (see Chapter 34). Infraposition of dental implants is of specific esthetic concern when implants are placed unilaterally and especially in the central incisor region where comparison with the incisal edge of the contralateral tooth makes any incongruence obvious. Methods to determine the safe time for implant placement are presented in Chapter 34. If implant placement has to be postponed due to remaining growth, it is important that the ideal space and stable occlusion is retained.

## Timing of implant placement in relation to dental trauma

A "simple avulsion" without damage to the alveolar socket and surrounding soft tissues may be considered regular flapless tooth extraction and the implant placement may be carried out according to the considerations below. In case of fractures of the alveolar ridge with or without concomitant mucosal lacerations, a healing period of minimum 3 months is recommended to be able to evaluate the need for adjunctive hard and or soft tissue augmentations and whether these most predictably should be performed staged or simultaneous with the implant placement.

## Timing of implant placement in relation to tooth extraction

When a traumatized tooth is deemed hopeless and scheduled for extraction, and the treatment plan foresees replacement with a dental implant, four different implant placement protocols may be applied (9, 10). The clinician has to make a decision with respect to the best time for placing the implant following removal of the tooth. Each placement protocol has its advantages and disadvantages. Immediate implant placement (Type 1) will reduce the number of surgical interventions and the overall treatment time. However, immediate implant placement is associated with greater variability in outcome compared to early implant placement (11). Especially, an increased risk of recession of the facial mucosa following immediate implant placement is of concern in the esthetic zone. Patients with a thin and/or traumatized facial bone wall and a thin gingival phenotype are of specific risk of developing facial mucosal recession after immediate implant placement (11). Therefore immediate implant placement should only be considered in patients with a thick

gingival phenotype and an intact facial bone wall with a thickness of preferably >1 mm (12). These conditions may be expected to be present in less than 10% of the cases (13, 14). Early implant placement after 6 to 8 weeks of soft tissue healing (Type 2) will not provide clinically significant bone healing compared to immediately after extraction. However, healed soft tissues will increase the long-term predictability of guided bone regeneration (GBR) procedures performed simultaneous with the implant placement (11, 15, 16). In addition, in sites with a thin or damaged facial bone wall, a spontaneous soft tissue thickening can be expected, which offers advantages for the subsequent implant surgery (17).

Implant placement after 12 to 16 weeks (Type 3) offers partial bone healing of the extraction socket. Increased resorption of the facial bone wall and treatment delay, however, makes this approach most relevant in cases where primary implant stability cannot be achieved immediately or after 6–8 weeks of healing. Late implant placement (Type 4) with at least 6 months of healing may be indicated in cases where ongoing growth of the alveolar process contraindicates implant placement, in cases where concomitant traumatic dental injuries to neighboring teeth warrants prolonged observation before a final prognosis of the teeth can be determined, and in cases with prolonged healing due to extensive trauma to the area. In addition, late implant placement can be indicated for site-specific reasons, such as an ankylosed tooth in an apical malposition without a sufficient bone volume apical to the root, which is required for primary implant stability. In such rare indications for late implant placement, a socket grafting is strongly advised to limit the post-extraction ridge alterations or even ridge atrophy (18, 19).

## Biologic aspects in esthetic implant sites

It is important for the clinician to recognize that ridge anatomy includes the soft tissues and the supporting bone in all dimensions, and that the soft tissue contours around implants are heavily influenced by bone anatomy. In the last 20 years, numerous studies revealed that the concept of the *biologic width*, is also valid for osseointegrated implants as well as for teeth, since soft tissues also demonstrate relatively constant dimensions around implants (20–22) (Figs 35.1 and 35.2). These studies have demonstrated a thickness of the peri-implant soft tissues of approximately 3 mm on the facial aspect, and of approximately 4 to 5 mm at interproximal areas. The size of this soft tissue cuff dictates the position of the bone level. In addition, there are differences in soft tissue thickness among different gingival biotypes (23). A thin biotype with a highly scalloped gingival architecture has a reduced soft tissue thickness when compared with a thick biotype featuring a blunted contour of papillae (24).

Keeping these relatively constant dimensions of peri-implant soft tissues in mind, the underlying bone structure plays the key role in the establishment of esthetic soft tissue contours in the anterior maxilla. Two anatomic structures are of importance: the bone height of the alveolar crest at interproximal areas, and the height and thickness of the facial bone wall. The interproximal crest height plays a role in the presence or absence of peri-implant papillae. Today, it is well accepted that a distance of more than 5 mm from the alveolar crest to the contact point reduces the probability of intact papillae not only at teeth, but also at implants (25, 26).

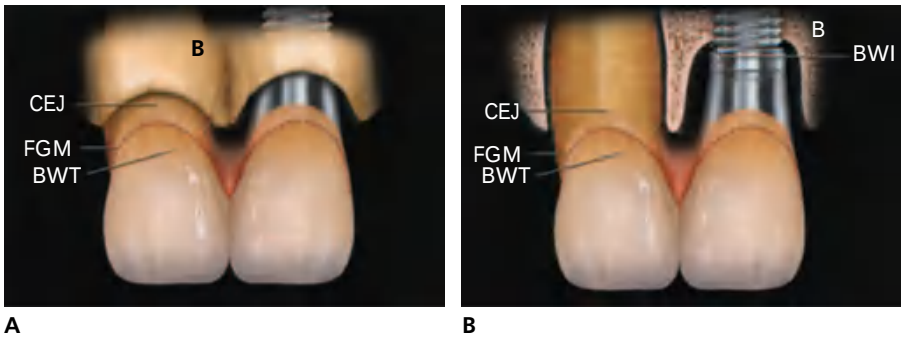



**Fig. 35.1** Placement of one implant next to a natural tooth. A. Implant placement adjacent to healthy dentition does not affect the interdental papilla due to the supracrestal position of the biologic width (BWT). B. Removal of buccal bone to visualize the interproximal bone level midway between buccal and palatal corticals. Note position of biologic width around implant (BWI) in comparison to the tooth (BWT). Source: Reproduced from (22) with permission.

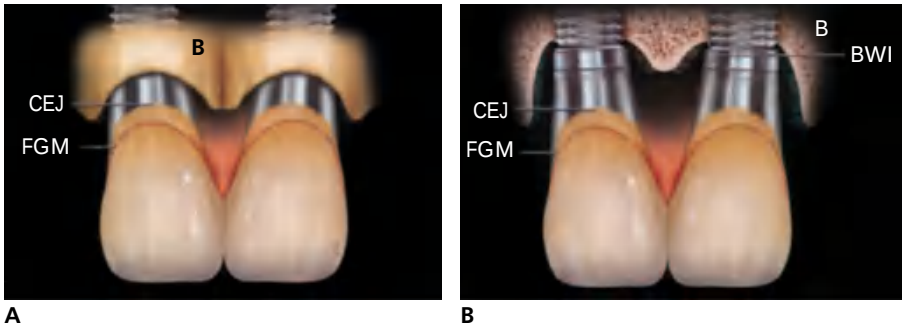



**Fig. 35.2** Placement of two adjacent implants. A. The placement of two adjacent implants results in the loss of the supracrestal biologic width. This means the soft tissue papilla will be higher. B. Buccal cortical plate removal to visualize the interproximal bone level midway between the buccal and palatal corticals. Note the position of the biologic width around the implant in relation to the contact point. Source: Reproduced from (22) with permission.

It has also been shown that the height of peri-implant papillae in single-tooth gaps is *independent* of the proximal bone level next to the implant, but depends upon the interproximal bone height at adjacent teeth (23). Clinical situations with reduced vertical bone on adjacent teeth are challenging, since there are still not any surgical techniques available to predictably regain lost crest height at teeth.

Having a facial bone wall of sufficient height and thickness is important for the long-term stability of a harmonious mucosal margin on the facial aspect without changes in tissue height. Patients with traumatic tooth loss frequently present with a bone wall that is missing or is insufficient in height and/or thickness. Thus, various surgical techniques have been proposed in the past 25 years to correct such bone defects at the facial aspect of potential implant sites. The best documented technique is *guided bone regeneration* (GBR) using barrier membranes, which can be carried out using a simultaneous or a staged approach (27). Clinical studies and experience demonstrate that horizontal bone augmentation can be predictably obtained with the GBR technique, whereas vertical bone augmentation is much more difficult to accomplish (28).

## Ideal implant position

Placement of implants in the correct three-dimensional (3D) position is the key for the esthetic outcome in implant patients, regardless of the implant system used (3, 29). The relationship between the position of the implant and the proposed restoration should be based upon the position of the implant shoulder. The implant shoulder position can be viewed in three dimensions: *oro-facial*, *mesio-distal* and *corono-apical*.

In the *oro-facial* direction, an implant shoulder placed too far facially will result in the risk of soft tissue recession, since the thickness of the facial bone wall is clearly reduced by a facial malposition of an implant (30). In addition, potential prosthetic complications could result in restoration–implant axis problems, making the implant difficult to restore. Implants positioned too far palatally can result in emergence problems, as seen with ridge-lap restorations.

Improper *mesio-distal* positioning of implants can have a substantial effect on the generation of interproximal papillary support in addition to the osseous crest on the adjacent natural tooth. Placing the implant too close to the adjacent root can cause resorption of the interproximal aveolar crest to the level of that on the implant. With this loss of the interproximal crest height comes a reduction in papillary height. The loss of crest height at adjacent teeth is caused by bone saucerization, routinely found around osseointegrated implants with a non-platform-switching design. This saucerization has a horizontal and vertical component (Fig. 35.3). Radiographs demonstrate that the horizontal component of proximal bone saucerization measures about 1.0 to 1.5 mm from the implant surface (31). This minimal distance needs to be respected at implant placement to prevent vertical bone loss on adjacent teeth. Too close a position results in loss of proximal bone (31, 32) and thereby the height of the interdental papilla (33).

The vertical component of saucerization amounts to approximately 2 mm at interproximal areas, measured from the implant shoulder. This radiographic observation routinely seen in patients (31, 34, 35) has been confirmed by an experimental study (36).

With regard to the corono-apical position of the implant shoulder, the same study (36) demonstrated that the position of the implant/abutment interface, often called the microgap, has an important influence on the hard and soft tissue

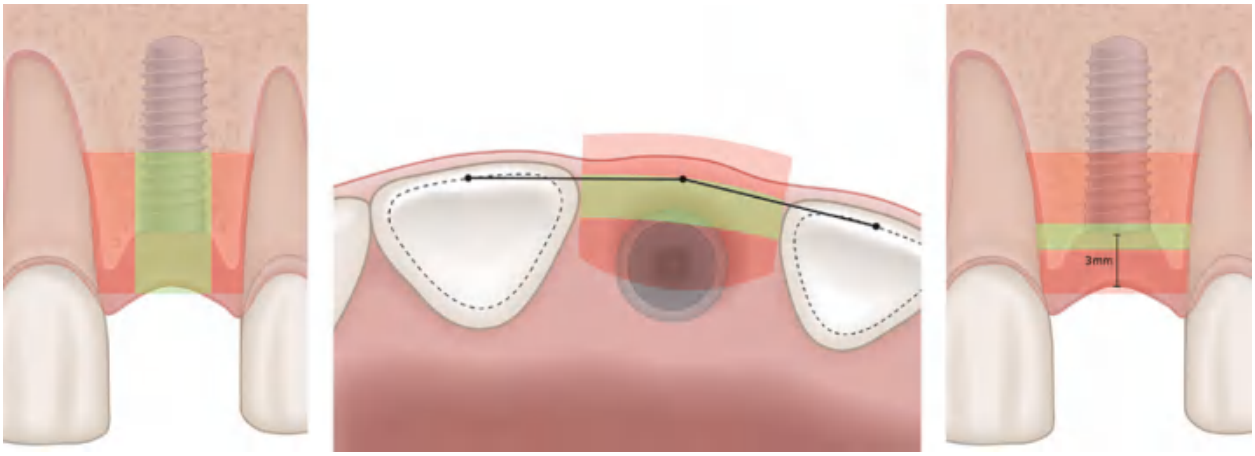



**Fig. 35.3** Comfort and risk zones in implant insertion. A. Correct implant position in the mesiodistal dimension. The implant shoulder should be positioned within the comfort zone, avoiding the danger zones, which are located close to adjacent root surfaces. The danger zone is about 1.0–1.5 mm wide. B. Correct implant position in the orofacial dimension. The implant shoulder is positioned about 1 mm palatal to the point of emergence at adjacent teeth. The danger zone is clearly entered when the implant is placed too far facially; this can cause resorption of the facial bone wall with subsequent recession. C. Correct implant position in the apicocoronal dimension. The implant shoulder is positioned about 2–3 mm apical to the CEJ of the contralateral tooth in patients without gingival recession. The danger zone is entered when the implant is placed too far apically using excessive countersinking, or too far coronally, which results in implant shoulder exposure at the mucosa

reactions around osseointegrated implants. The further apically the microgap is located, the more bone resorption is observed. Clinically, if an implant is inserted with an excessive countersinking procedure, an unnecessary amount of bone loss will occur. Since this resorption will take place circumferentially (Fig. 35.3), it will affect not only the proximal bone structure, but also the height of the facial bone wall, and can lead to undesired soft tissue recession (3). This phenomenon is also important in sites with two adjacent implants since the inter-implant bone will be resorbed, leading to a shortened inter-implant papilla (Fig. 35.3). Recent evidence suggests that the concept of platform-switching – a horizontal offset of the implant abutment connection – may reduce the marginal bone loss around implants especially on the facial aspect of the implants (37, 38). However, systematic reviews are inconclusive regarding the predictability and potential benefit of platform-switching in preserving marginal bone around implants (39, 40).

In addition, oro-facial implant angulation influences the esthetic outcome as well. A correctly planned implant angulation will help to optimize the emergence profile and facilitate the restorative phase. An implant angulation too much directed to the facial, will lead to a facial hard and soft tissue loss. The correct implant axis should be located roughly 1 mm palatal to the future incisal edge allowing a transocclusal screw retention of the implant restoration in the cingulum area. In the esthetic zone, a screwed retained restoration is clearly desired and offers different clinical advantages.

Esthetic failures can also be caused by improper implant selection, mainly due to the use of oversized implants. The use of 'tooth analogous' implant diameters based solely upon the mesio-distal dimension of the tooth to be replaced should be avoided. With such wide-platform or wide-neck implants, the implant shoulder could be placed too close to adjacent teeth and too far facially, leading to the complications mentioned above (3). On the other hand, reduced diameter implants may lead to challenges ensuring the ideal emergence profile of the implant-supported restoration (41, 42).

## The concept of comfort and danger zones

When planning for an ideal 3D implant position, a distinction is made between 'comfort' and 'danger' zones in each dimension (mesio-distal, oro-facial, and corono-apical) (3) (Fig. 35.3). Selection and placement of the dental implant should be based on the planned restoration in these zones (3, 22, 29). If the implant shoulder is positioned within the danger zones, one of the above-mentioned complications could occur, potentially resulting in esthetic short-comings.

Respecting the comfort zones in three dimensions results in an implant shoulder located in an ideal position, allowing for an esthetic implant restoration with stable, long-term peri-implant tissue support.

### Mesio-distal dimension

In the mesio-distal dimension, the *danger zones* are located next to adjacent teeth. It is recommended that the implant shoulder and the adjacent root surface be at least 1–1.5 mm apart (Fig. 35.3). The critical distance between two implants has been found to be 3 mm. Too close a position results in loss of interproximal bone from saucerization around the implant shoulders and thereby the height of the interdental papilla (22, 26, 33).

### Oro-facial dimension

With regard to the oro-facial dimension, the *facial danger zone* is located anywhere facially to the imaginary line highlighted from the point of emergence of the adjacent teeth and/or planned restoration (Fig. 35.3). The *palatal danger zone* starts about 2 mm from this point of emergence and leads to an increased risk of a ridge-lap restoration. Placement of the implant oro-facially in the comfort zone, which is located anywhere in between these areas, will allow for a restoration with a proper emergence profile to maintain the harmonious scalloping of the gingival margins (3).

### Coronal-apical positions

Tooth loss will, as a rule, result in not only loss of the oro-facial dimension but also the vertical height. This phenomenon becomes excessive if tooth loss is accompanied by severe trauma to the alveolar bone, occurs early, or is followed by gradual infraposition of an ankylosed tooth (43, 44). In these cases the installation of implants will result in a significant esthetic problem due to gingival asymmetry.

Another disturbing phenomenon is that the position of the gingiva will be below the interdental crest, and this may lead to subsequent loss of interdental bone (32). Furthermore, if implants are placed too deep the saucerization will interfere with labial bone, leading to retraction of the facial mucosal margin and the papillae (3).

The corono-apical positioning of the implant shoulder follows the philosophy 'as shallow as possible, as deep as necessary', being a compromise between esthetic and biologic principles. The position of the implant shoulder should be approximately 2–3 mm apical to the mid-facial gingival margin of the planned restoration (45). This can be accomplished through the use of surgical templates highlighting the gingival margin of the planned restoration. In patients without vertical tissue deficiencies, the use of periodontal probes leveled on the adjacent cemento-enamel junction (CEJ) in single-tooth gaps has proven to be a valid alternative. Implant placement within the *apical danger zone* (located anywhere 3 mm or greater apically from the proposed gingival margin) can result in undesired facial bone resorption and subsequent recession of the facial mucosal margin. The *coronal danger zone* is invaded with a supragingival shoulder position leading to a visible metal margin and poor emergence profile (Fig. 35.3).

## To create and maintain a harmonious soft tissue

Soft tissue appearance is related to the level, color and texture of the peri-implant mucosa. Scar tissue may dominate the area caused by the trauma itself or by later surgical procedures to preserve the affected teeth (Fig. 35.4).

### Level and appearance of the peri-implant mucosa

#### *Facial and oral*

In addition to scar tissue related to the previous trauma, repeated surgical incisions in the implant area (e.g. to insert bone transplant and/or membranes) may create scar tissues. If repeated surgical interventions have to be made, incisions should be made along previous incision lines.

The final location of the facial mucosal margin is primarily determined by the vertical position of the implant. However, the oro-facial position and the implant angulation also has an important impact on the restorative dentists possibilities predictably to place the mucosal margin in accordance with that of neighboring teeth (11). According to the abutment plateau the level of the labial and palatal mucosa will be approximately 2.5 mm coronally (Fig. 35.3). This means a 5–7 mm deep interproximal position of the implant shoulder due to the gingival scallop (46). Such a deep position may prevent satisfactory cementation and call for screw retention of the restoration (29, 47).

A stable facial mucosal margin may be predictably achieved long-term, provided the presence of a thick gingival phenotype and an intact or augmented facial bone wall of 1.5 mm or more (11, 15, 48). However, esthetically compromised recessions of the facial mucosal margins have been reported (49). The risk of recession of the mucosal margin increases with an immediate implant placement protocol, thin gingival biotype, a thin facial bone wall, and implant placement beyond the facial and vertical comfort zone (11, 50, 51).

**Fig. 35.4** 18-female patient with scar tissue after a previous lateral luxation of the upper left central incisor at the age of 9. Later endodontic surgery was performed before the tooth was removed due to progressive infraposition.

#### *Papillae*

A tooth has gingival collagen fibers inserted in cementum, which support and maintain the interdental papilla. In contrast, dental implants are characterized by circumferential collagen fibers which do not have the same capacity (22).

The creation of a papilla next to a dental implant with adequate fill of the interproximal space is related to a number of factors:

- The original soft tissue level at the implant site;
- The marginal bone level at the implant site;
- The presence of a neighboring natural tooth or implant;
- Position of the contact point to neighboring tooth/crown;
- Surgical techniques (see later);
- The use of platform-switching vs. matching abutment-implant connection;
- Time after implant placement; and
- Whether or not a customized temporary restoration is used.

Since Tarnow's classical studies on the relation between the position of the interdental septa in natural teeth and the position of the interdental papilla (25), a similar relation has been found in regard to implants next to natural teeth (26, 33). Thus when the distance from the interproximal contact point to the top of the bone crest was 3–4 mm, the chance of a gingival papilla filling the interdental space was found to be 100%, whereas the chance was reduced to 50–60% when the distance was 6 mm (26, 52).

The strong relation between an adjacent neighboring tooth with normal periodontal conditions implies that the natural tooth is the essential factor for an intact papilla and will determine the position and height of the papilla (23). In certain cases, this principle may be applied and where a papilla is lacking, orthodontic extrusion of a neighboring tooth may be used to create a more favorable position of the papilla (53, 54).

A different problem arises if two implants are placed next to each other (Fig. 35.2). In this case the height of the papilla will be approximately 3 mm above bone level (22, 52, 55), a situation that usually implies that a 1–2 mm reduced soft tissue "papilla" will be found between the two implants (22, 52, 55).

Another factor that determines the presence of a papilla is the distance of the cervical level between implant to adjacent teeth or implant-to-implant. The chance of a 100% papilla fill is found when this distance is less than 2.5 mm (52).

The volume and position of the interdental papilla has been found to increase over time (56–59). Furthermore, customized intermediate restorations that are slightly oversized may induce growth of the interdental papilla (58). However, it appears that the inherent spontaneous growth of the papilla over a two-year period of time will give the same result (58, 59).

### *Gingival pocket depth*

This feature appears to be quite stable over the years, thus in a 1–9 year follow-up study no significant deepening took place (59, 60).

## To create and maintain a harmonious alveolar process

In almost all trauma cases alveolar bone has been lost and needs to be recreated. A variety of methods are available today, with varying success rates (see below).

### Proximal bone

The maintenance of proximal alveolar bone is of interest as loss of this bone may endanger an adjacent natural tooth. In a 10-year study in young adolescents a loss ranging from 0.5–1.5 mm took place (32). Such an amount may be of concern if this rate of bone loss continues. In adults, however, the bone loss appeared to be more confounded and leveled out after some years (23, 61). A new concept, scalloped implant design, could possibly lead to less proximal bone loss (62, 63). However, this has not yet been confirmed clinically (64).

### Bone augmentation

Bone loss and resorption of the partially edentulous alveolar ridge due to trauma frequently compromises dental implant placement in a prosthetically ideal position. Therefore, augmentation of an insufficient bone volume is often indicated prior to or in conjunction with implant placement to obtain a predictable long-term functioning and esthetic treatment outcome (3). A variety of techniques exist to augment localized defects in the alveolar ridge (28). The preoperative analysis should reveal the morphology and extent of the bone deficiency to evaluate whether bone augmentation may be performed simultaneous with the implant placement or using a staged approach. When implants can be placed with primary stability in the ideal 3D position for the later prosthetic restoration leaving a bone defect in the horizontal dimension, simultaneous ridge augmentation is predictable and should usually be the treatment of choice (28). Bone defects exposing the marginal part of the implant are termed dehiscence-type defects (Fig. 35.5), whereas bone defects exposing the apical part of the implant are termed fenestration-type defects (Fig. 35.6).

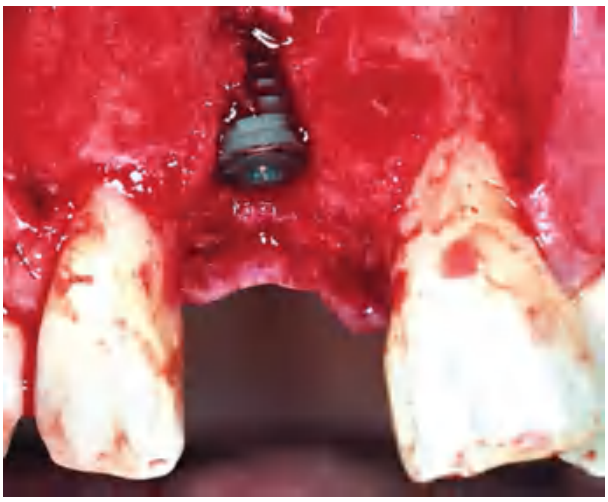


**Fig. 35.5** Dehiscence-type bone defect related to an implant inserted to replace an upper right central incisor lost due to a previous traumatic dental injury.

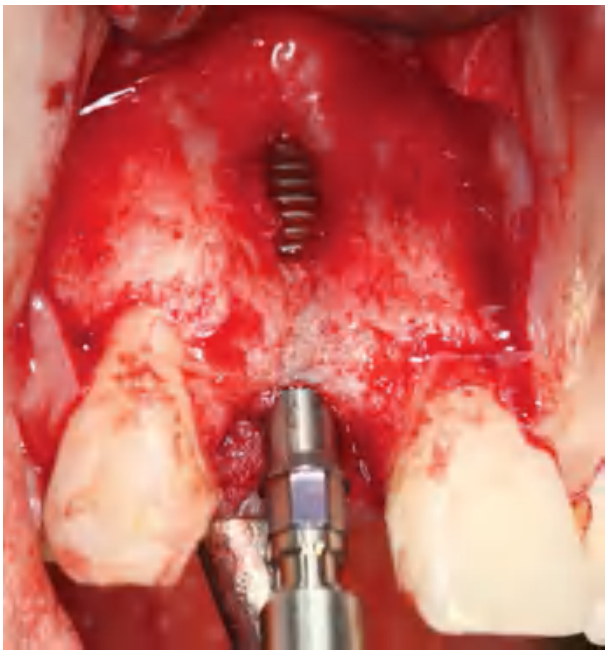


**Fig. 35.6** Fenestration-type bone defect related to an implant inserted to replace a congenital missing upper right lateral incisor.

## Horizontal augmentation techniques

In the last four decades, a number of horizontal augmentation techniques have been described, including:

- Use of bone block grafts;
- Use of particulate autogenous bone grafts or bone substitute materials;
- Use of barrier membranes with or without block grafts or particulate grafting materials – GBR;
- Use of titanium mesh with autografts; and
- Bone splitting.

With respect to time sequence, ridge augmentation is either performed simultaneously with implant placement, or as a preparatory step in a staged procedure before implant placement.

### Onlay grafts

Autogenous block grafts have been applied for lateral ridge augmentation as an onlay or veneer graft. Block grafts should

be fixed to the residual bone using fixation screws ensuring optimal stability and absence of micro-movements that allows for osseous integration of the bone blocks (65). However, considerable surface resorption of so-called non-protected bone blocks has been reported (66, 67). Therefore over-augmentation has been recommended, although the magnitude of the expected surface resorption is reported to be unpredictable (66).

### Guided bone regeneration (GBR)

The use of barrier membranes has become widely accepted in periodontology and implant dentistry. Although in the past clinicians have advocated the use of barrier membranes for space secluding, it is now recognized that membranes collapse into the defect resulting in limited success of the treatment. Therefore, it is now unequivocally recommended that a block or particulate material be used for membrane support (27).

The first clinical and experimental studies on GBR reported the use of a non-resorbable expanded polytetrafluoroethylene (ePTFE) membrane (68–73). Although ePTFE membranes showed excellent biocompatible properties they had an increased risk of wound dehiscence with possible site infection and/or reduced volume of augmented bone (69, 74). Additionally, the hydrophobic ePTFE-material showed to have poor handling and adaptation properties and required fixation to the underlying bone. Another disadvantage was the necessity of a second flap procedure for membrane removal. To improve their mechanical stability, ePTFE-membranes were reinforced with titanium, but this did not help to reduce their significant drawbacks.

In the past 20 years, ePTFE membranes have gradually lost their dominance for GBR procedures, and resorbable barrier membranes, in particular collagen membranes, were used more and more in daily practice (27). The hydrophilic properties of collagen membranes made the surgical management easier and faster. Furthermore, no additional flap surgery is required for membrane removal. However, resorbable membranes need to be supported by bone fillers to compensate for their tendency to collapse (75). For polyester membranes, inflammatory tissue responses have also been reported following membrane degradation (75–78).

Several clinical and experimental studies have proven that bone regenerated with the GBR technique reacts to implant placement like pristine bone, and this bone is load bearing over the evaluated periods (79–82).

### Use of titanium mesh

Titanium mesh has been used for ridge augmentation procedures because of its enhanced mechanical properties (83–85). However, titanium mesh has no barrier effect and neighboring soft tissues grow through it. Therefore, some authors have combined titanium mesh with barrier membranes (86). Because the mesh is rigid it must be perfectly adapted to the bone contour and must be secured with small fixation screws to prevent mucosal perforations. A second surgical procedure is also necessary for mesh removal. Since handling and application of a titanium mesh is a rather technique-sensitive procedure, it has not gained wide acceptance. However, recent clinical studies have indicated that titanium mesh may be indicated for some cases of vertical alveolar ridge augmentation (87–89), although complications may hamper the augmentation result (85).

### Bone splitting

The crest of an edentulous ridge is expanded by splitting the two cortical plates. This technique is most often combined with immediate implant placement, which reduces treatment time (90). Since no block grafts are used the need for a second surgical site is eliminated. Prerequisites for successful bone splitting include:

- Intact vertical height of the alveolar ridge;
- Presence of a cancellous compartment of the alveolar ridge in the entire height of ridge, which means that a minimum ridge width of 3 mm should be present; and
- A favorable angulation of the alveolar ridge where the facial cortical plate will not be displaced apically after splitting reducing the support of the facial mucosal margin of the implant.

In addition, bone splitting usually should be combined with the GBR technique to avoid resorption of the thin facial bone wall (91). Since these criteria are rarely met in the anterior maxilla, the technique most often indicated in the posterior mandible

## GBR technique: simultaneous approach (Fig. 35.7)

Provided the clinical and radiographic site analyses have shown that a simultaneous approach is feasible, the patient is scheduled for implant placement with concomitant contour augmentation. Following local anesthesia a crestal incision (in esthetic sites, the incision is moved to the palatal aspect of the crest) is made with intrasulcular incisons on facial and lingual aspects of the adjacent teeth. One or, if necessary, two vestibular divergent release incisons are placed at the line angle of adjacent teeth. Full mucoperiosteal flaps are raised on the facial and lingual aspects and are retracted with retraction sutures. All remaining soft tissues are thoroughly cleaned off and the crest may be slightly scalloped with a large round bur.

The implant bed is prepared according to the implant surgery protocol. A correct 3D implant positioning is most important for prosthetic (and esthetic) tooth restoration (3). In esthetic sites, the implant shoulder should be placed 2–3 mm below the CEJ of the adjacent teeth with respect to vertical implant position. In the horizontal direction, the implant shoulder should be located slightly inside the CEJ of the adjacent teeth. Both measures are

important to ensure an optimal emergence profile of the implant restoration. A 2 mm healing cap is inserted into the implant.

The bone adjacent to the exposed implant surface is perforated with a small round bur for bone marrow penetration. The exposed implant surface is covered with autogenous bone chips locally harvested from nearby cortical bone surfaces using bone scrapers or bone chisels. A layer of bone substitute material with a low substitution rate is applied above the autogenous bone chips for overcontouring the local ridge anatomy. A collagen membrane is trimmed to the appropriate size and is placed to fully cover the augmentation material. The membrane is soaked in blood for tight adaptation but it is not secured with screws or tacks. A second strip of membrane is positioned to form a double layer to enhance barrier function.

Following a periosteal release incision, the flap is repositioned in a coronal direction and tension-free primary wound closure is accomplished with single interrupted sutures. No tension should be exerted on the wound margins. In case of minor dehiscence-type defects, fenestration-type defects, or non-esthetic sites transmucosal healing is predictable (92, 93). Any removable provisional prosthesis must be adapted to avoid pressure to the augmented tissues. Sutures are removed within 7–10 days. For the same period, chemical plaque control is instituted with chlorhexidine-digluconate 0.1–0.2%. Perioperative, short-term antibiotic prophylaxis is recommended for up to 3 days post-closure.

After a healing period of 8 weeks the submerged implant is uncovered with a small semilunar incision, and a longer healing cap is placed for peri-implant soft tissue healing. The provisional or definitive restoration is fabricated within 1–3 weeks.

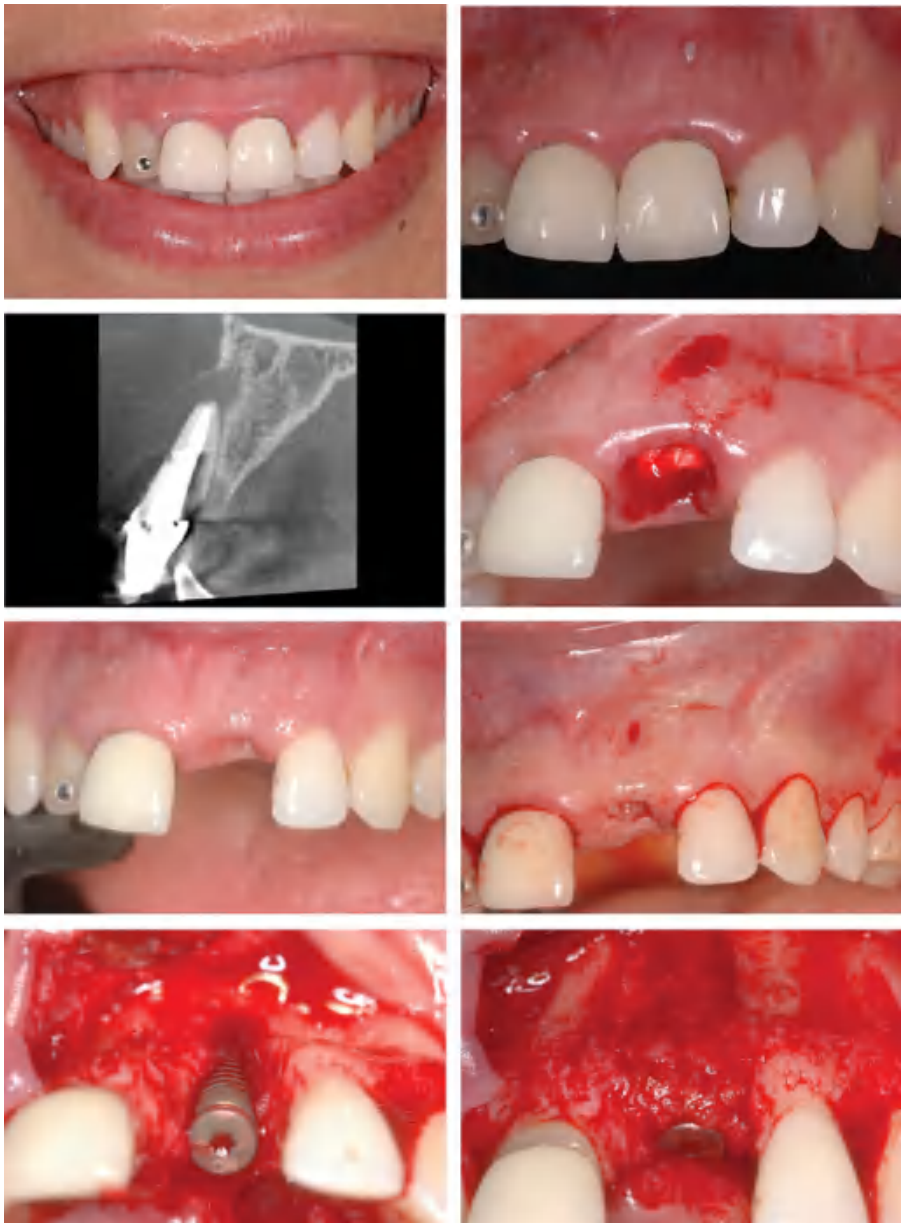


**Fig. 35.7 Treatment of failed endodontically treated traumatized incisor**

Clinical status of a 26 year female patient showing a high smile line situation exposing a significant portion of the maxillary alveolar process. Detail view of the two central incisors, which have been crowned for several years. The left central incisor presents with a fistula and probing depths of ≥6 mm.

**Radiographic condition**

Sagital CBCT clearly depicts the problem, since the incisor has no visible facial bone wall and a large periapical lesion. The condition of the tooth is hopeless, and it needs to be removed. Clinical status following flapless extraction, debridement, and application of a collagen plug to stabilize the blood clot.

**Postoperative healing**

The post extraction healing period was uneventful. The fistula closed, and a typical flattening is observed in the mid-facial area of the extraction socket. 8 weeks post extraction, implant surgery is performed using a typical triangular flap design with a releasing incision remote at the first premolar.

**Implant insertion including bone grafting**

The implant is inserted in a correct 3D position resulting in a large dehiscence-type bone defect on the facial aspect. The wide defect, however, has 2-wall morphology, which is a prerequisite for a simultaneous GBR procedure. The large bone defect is completely filled with locally harvested autogenous bone chips to accelerate new bone formation in the defect area. The augmentation is done to the rim of the applied 2 mm healing cap.

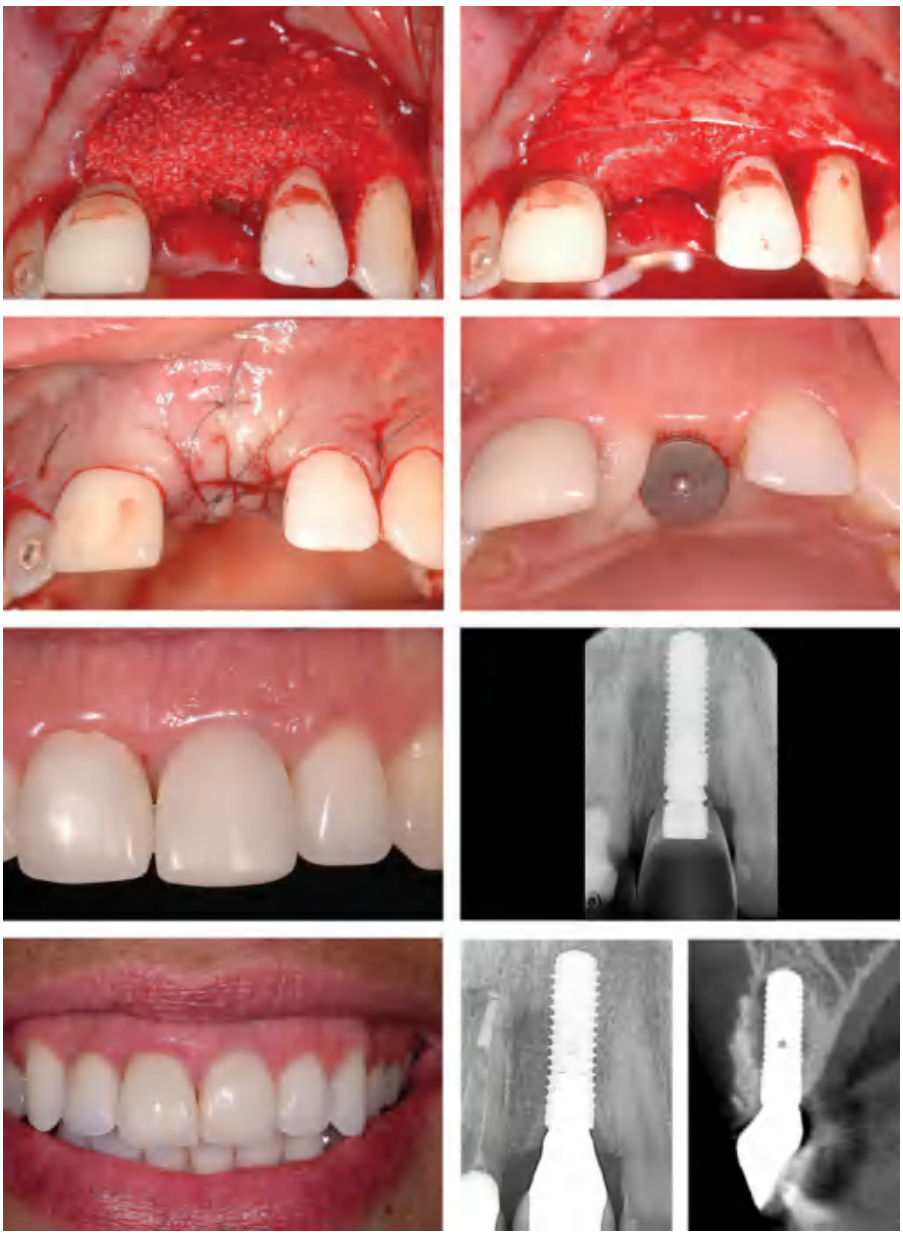
**DBBM particle insertion plus collagen membrane**
A second layer with DBBM particles is applied on top of the autografts to achieve a contour augmentation in the defect area. This bovine bone filler has a low-substitution rate and offers long-term volume stability. A native collagen membrane is applied to serve as a temporary barrier to protect the augmentation area against the ingrowth of soft tissue cells during early wound healing.

**Wound closure and implant exposure**
The surgery is completed with a tension-free primary wound closure to protect the applied biomaterials. After 8 weeks of healing, the site is reopened with a punch technique, and the 2 mm healing cap is exchanged by a longer and larger one.

**Acrylic temporary restoration**
Clinical status following restoration with acrylic provisional crowns to optimize the soft tissue condition. The periapical radiograph shows excellent crestal bone stability 6 months post implant placement.

**Clinical condition after restoration**
Final restoration with two all ceramic crowns at both central incisors. Clinical status 5 years post extraction. The overall esthetic result is very pleasing, although the ginvigal tissue at natural tooth 11 is irritated. The peri-implant soft tissues at implant 21 is excellent without clinical signs of inflammation. The position of the mid-facial mucosa is just right.

**Radiographic condition after restoration**
The corresponding peri-implant radiograph shows excellent stability of the crestal bone. The bone seems well integrated without signs of crestal bone loss. A sagittal CBCT taken 3 years post placement shows a thick und dense buccal bone wall which is important for esthetic soft tissue support.



## GBR technique: staged approach (Fig. 35.8)

In sites with severe bone deficiencies, where implant placement and implant stability are compromised, a staged approach should be chosen. A thorough clinical and radiographic examination (cone-beam CT) is highly recommended to analyze both the augmentation and possible harvest sites. Since staged horizontal augmentation requires larger volumes of bone grafts, donor sites for bone blocks must be evaluated before surgery. Potential intraoral donor sites for block grafts include the mandibular body or ramus area and the symphysis (94–98).

This surgical technique often involves a second surgical site with prolonged surgical time. Therefore, patients are normally given a sedative premedication, but surgeries may still be performed under local anesthesia. The site to be augmented is prepared with similar incisions as those utilized in the simultaneous procedure. Full mucoperiosteal flaps are raised again on both aspects of the deficient alveolar ridge. The bony surfaces are cleaned with a large round bur in counter clockwise rotation. The cortex is perforated with a small drill for early blood supply from the bone marrow. It has been indicated that this procedure may enhance fusion and integration of the block grafts (99). The dimensions of the defect are measured with a caliper or probe and a corresponding block graft is harvested. The block graft is adapted to the recipient site for horizontal augmentation, and it is fixated with one or two fixation screws (diameter 1.5 mm) to the residual bone wall. The screws should glide in the block graft and only engage the host bone (lag screw principle).

Following block graft fixation, any sharp corners or edges are smoothed. Voids around the block graft are filled with

additional bone chips. Bone substitute particles with a low substitution rate are placed in a thin layer over the block graft to prevent surface resorption. In order to avoid displacement of the particles, a collagen membrane is adapted to cover the entire augmentation site. A second layer of collagen membrane is positioned to enhance barrier function and membrane stability. Membranes are not secured *per se* with screws or tacks, but soaking with blood or saline is recommended for membrane adaptation. Following a periosteal release incision ensuring full flap mobilization, the flap is coronally repositioned and the incision is reapproximated with single interrupted sutures.

A provisional prosthesis is carefully adapted to avoid pressure by the pontic or flange onto the augmentation site. Sutures are removed after 10–14 days, and patients are instructed to rinse with chlorhexidine-digluconate 0.1–0.2%

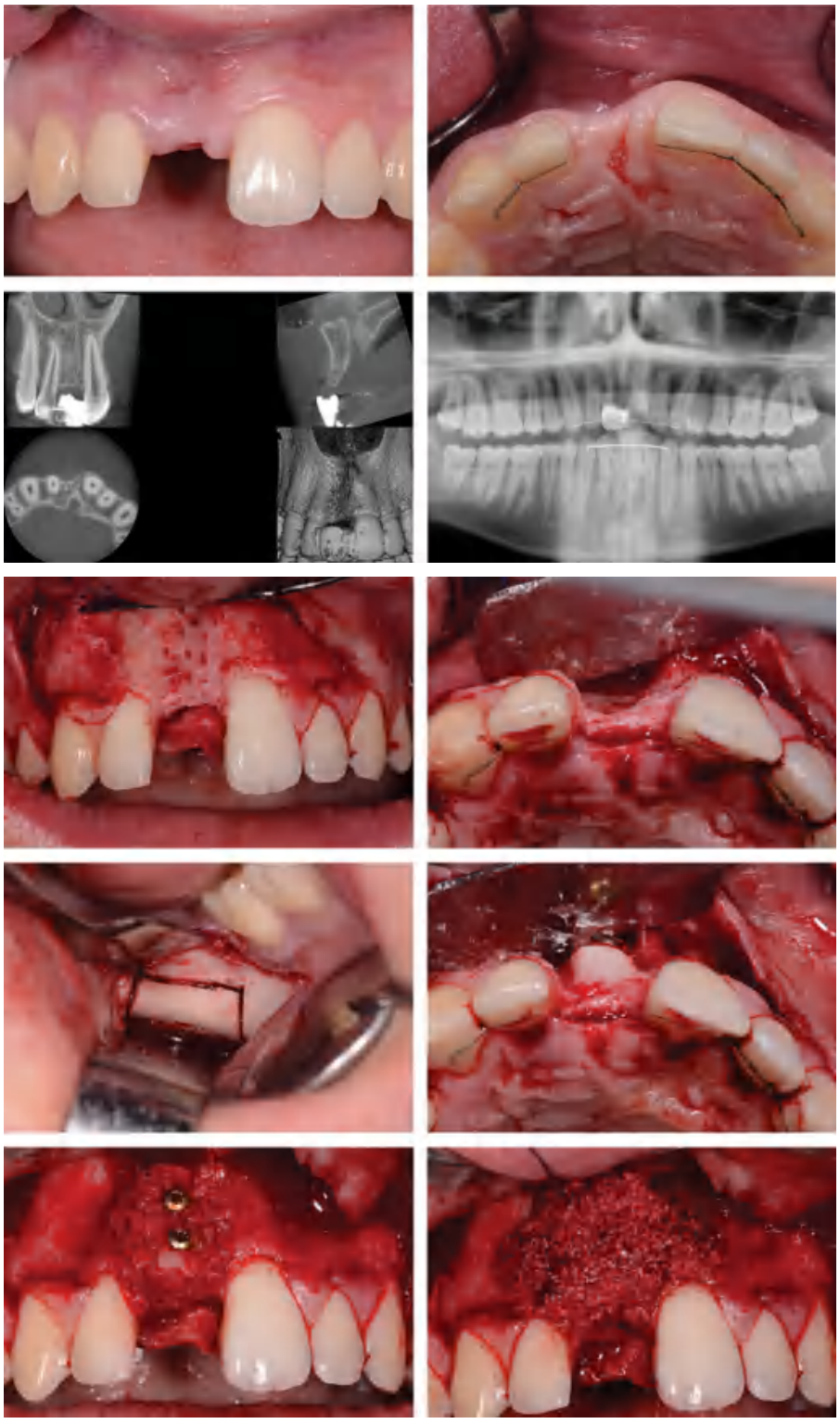


**Fig. 35.8 Implant, insertion and bone transplantation after tooth loss**
A male patient presented with subsequent loss of right central incisor due to trauma at the age of 9. In the clinical examination by the age of 20, the patient revealed healthy gingival tissues. A significant horizontal defect was visible 11 years following tooth loss. Due do the neighboring virgin teeth an implant supported single crown was planned.

**Radiographic condition**
A CBCT revealed a sufficient bone height. However the crest width was insufficient with only 3.4 mm and with the nasopalatine canal in close proximity. Therefore a staged ridge augmentation using autogenous bone block and guided bone regeneration was planned. The retromolar area was chosen as block harvesting site.

**Preparing the graft site**
Following mucoperiosteal flap elevation using a papilla-base incision technique with one releasing incision distal to right canine mandilas, the cortical bone plate was perforated to ease vascularization and new bone formation from the bone marrow space.

**Harvesting bone graft and subsequent transplantation**
An autogenous block graft was harvested from the retromolar area using piezosurgery.The block was adapted to the recipient site and stabilized using two fixation screws

**Transplantation of bone chips**
The voids around the block were filled with autogenous bone chips harvested locally and from the donor site.

**Covering the graft area with a collagen and wound closure**

The autogenous bone block was covered with DBBM followed by a double layer of a native collagen membrane to stabilize the grafted site. Such protected autogenous block grafts have shown a minimal resorption rate of 7.7% 10 years following staged ridge augmentation (108). Primary wound closure was carried out to protect the augmented site by non-resorbable polyamide sutures.The patient went abroad for 1 year. After 15 months of healing, the site showed sufficient bone and soft tissue volume.

**Clinical and radiographic condition after 15 months**

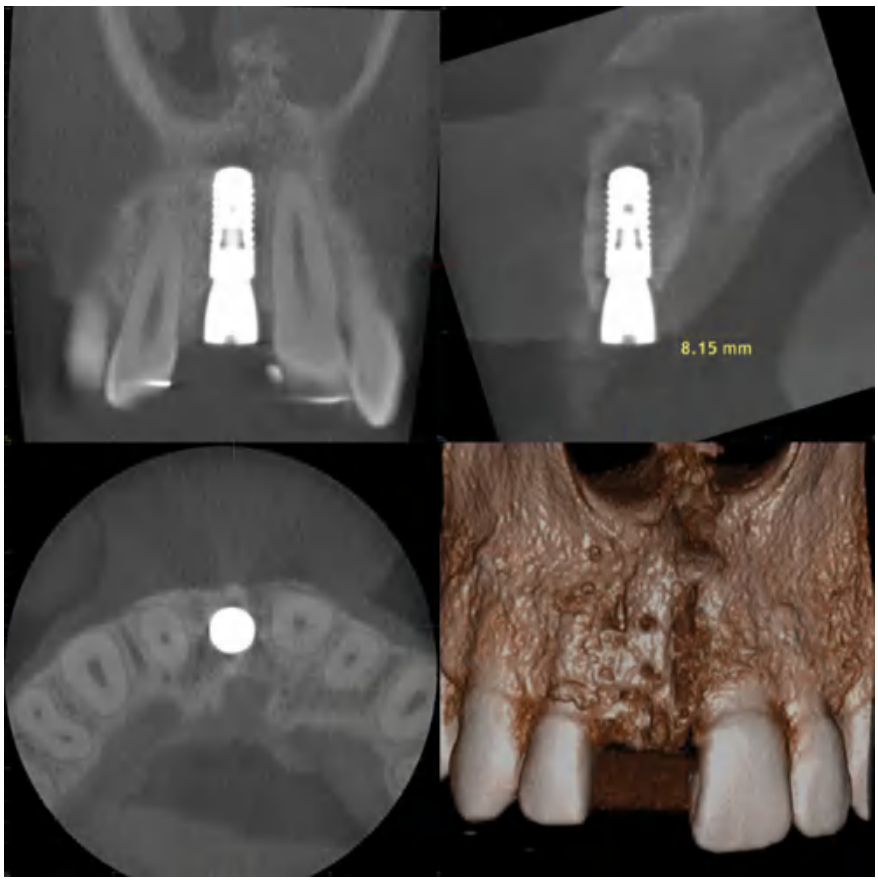
The CBCT revealed sufficient bone height and width of 18.6 mm respectively 7.5 mm.

**Exposing the implant bed**

The ridge showed newly regenerated bone with minimal signs of resorption. The fixation screws were removed and the implant bed was prepared.

**Postoperative healing**

After one week the sutures were removed. After 10 weeks the site healed uneventfully. The CBCT revealed a sufficient crest width of 8.2 mm with a thick facial bone wall. Bone is forming above the shoulder level up to the healing cap.
A larger healing cap was inserted showing a mild blenching of the soft tissues. The patient was for provisional restoration.



**Impression was taken**
A screw retained provisional with a titanium abutment was individually modified to allow optimal soft tissue conditioning (Prosthodontist: Dr. R. Buser, Dental technician: P. Müller)
The provisional was applied showing some blenching of the soft tissue.
Two weeks following the soft adapted well and the papillae are starting to form.

twice daily. Perioperative antibiotic coverage should be initiated one hour preoperatively and is recommended to last for 3–6 days after the procedure (100).

After a healing period of 4–6 months the augmentation site is reentered using a similar incision and flap technique. The bone block fixation screws are located and removed. Implant placement is now performed according to the standard implant surgical protocol.

## Rationale for the described techniques

Both presented simultaneous and staged augmentation techniques use three distinctive surgical steps, and materials respectively: placement of autogenous bone, application of bone substitute particles with a low substitution rate, and use of a collagen membrane.

Autogenous bone is considered the gold standard for ridge augmentation in implant dentistry (101). However, non-protected bone grafts can undergo severe surface resorption with a compromised treatment outcome (67, 102). Experimental and clinical studies have shown, that protection of grafted bone with a bone substitute material with a low substitution rate in combination with a barrier membrane ensures graft integration and volume stability (80, 103–109). In particular in the esthetic zone, contour preservation is of highest importance to warrant the long-term esthetic outcome following implant placement (15, 16, 48).

With the emergence of non-resorbable barrier membranes, which are liable to degradation or resorption within weeks (110–112), the protection of grafted bone is only assured for a limited period. Thereafter, the bone grafts may undergo surface resorption comparable to non-protected grafts. One option to circumvent this problem is to cover the bone graft with a slow or non-resorbable material, such as hydroxyapatite. Deproteinized bovine bone mineral (DBBM) has been shown to fulfill these criteria. Once inserted in host tissue, this bone substitute is not resorbed following bony integration (74, 109, 113–115). When

located at the periphery of bone defects, DBBM particles are embedded in fibrous tissue with very slow resorption if at all (74, 116). Therefore, the rationale of covering bone grafts with DBBM particles in augmentation sites is twofold: 1) to maintain the contour of the augmented site and 2) to protect the bone grafts from surface resorption.

The use of a collagen membrane in a double layer technique assures that the applied filler particles are not dislodged until they have become encapsulated with fibrous connective tissue.

## Vertical augmentation techniques

In addition to horizontal bone loss, tooth loss due to traumatic dental injury often leads to alveolar resorption of the alveolar ridge in the vertical dimension. However, vertical augmentation of hard and soft tissues prior to implant therapy is considerably less predictable than in the horizontal dimension (28, 117).

Vertical bone augmentation may be performed by:

- Onlay block grafts;
- GBR using titanium reinforced non-resorbable membranes with or without bone grafts;
- Titanium mesh in combination with particulated autografts;
- Distraction osteogenesis; and
- Segment osteotomies with interpositional bone grafts.

As for staged horizontal ridge augmentations, autogenous bone block grafting is the standard against which all techniques of osseous reconstruction of the maxillofacial skeleton must be judged. Usually, bone block grafts harvested from the mandible are preferred before blocks from extraoral donor sites due to less resorption, complications, and morbidity (118–120) (see below). Characteristics of autografts from intra- and extraoral donor sites are listed in Table 35.1.

### Staged vertical bone augmentation

Onlay block grafts for vertical ridge augmentation have been attempted with simultaneous implant placement and using a staged approach. Resorption of autogenous onlay bone grafts used for augmentation of severely atrophied alveolar ridges has been a frequent problem in preprosthetic surgery, with resorption rates up to 100% within the first 3 years being reported (121). Early studies indicated that simultaneous implant placement could limit the resorption of the autogenous bone blocks (122–124). However, more recent clinical studies have documented staged vertical bone augmentation to be more predictable both short- and long-term due to increased graft maturation and osseointegration, reduced crestal bone loss, and importantly a better chance to place the implants in the correct 3D position for the later prosthetic rehabilitation (27, 125). However, vertical ridge augmentation using onlay grafting is still less predictable and is accompanied by significantly more complications than augmentation in the horizontal dimension (28).

In contrast to horizontal ridge augmentations using autogenous block grafts, vertical augmentations are preferably performed through vestibular incisions 5–10 mm above the muco-gingival junction to prevent postoperative dehiscence. Otherwise, the surgical technique follows the same biologic principles as for horizontal ridge augmentation.

### Alveolar distraction osteogenesis

Reconstruction of the traumatized atrophic alveolar is often challenged by the concomitant lack of hard and soft tissue in the vertical dimension. Bone augmentation by gradual distraction of the hard and soft tissues is a method that aims at reducing these problems.

The principle of osseodistraction was first applied in orthopedic surgery by Ilizarov (126) followed by mandibular lengthening in maxillofacial surgery by McCarthy and coworkers (127), and was later used for reconstruction of the alveolar process (128).

The alveolar defect must be assessed according to the vertical and transversal deficiency. In cases with a very thin alveolar ridge, horizontal block grafting according the technique described above may be indicated in a separate surgical procedure before or after distraction.

When the initial planning has been done the choice of distraction device can be made. There are presently numerous

**Table 35.1** Donor sites for autografts.

| | Region | Geometry | Volume*** | Resorption | Complications |
|---|---|---|---|---|---|
| Intraoral | Chin | Cortico-cancellous* | ++ | ++ | ++ |
| | Mandibular body/ramus | Cortical | ++ | + | + |
| | Nasal spine | Cortico-cancellous* | + | +++ | + |
| | Maxillary tuberosity | Cortico-cancellous** | + | +++ | + |
| | Zygomatic body | Cortico-cancellous** | + | +++ | + |
| Extraoral | Illiac crest anterior/posterior | Cortico-cancellous** | +++ | ++ | ++ |
| | Tibial condyle | Cancellous | ++ | +++ | + |
| | Calvaria | Cortical | ++ | + | + |
| | Fibula (vascularized) | Cortico-cancellous** | +++ | + | ++ |

* More cortical than cancellous bone.
** More cancellous than cortical bone.
*** +sufficient bone for augmenting 1 tooth gap, ++ sufficient bone for up to 2 sinus augmentations, +++ sufficient for major inlay and onlay augmentations and reconstruction of continuity defects.

commercial devices for alveolar distraction on the market and many custom-made devices have been described. The choice includes extraosseous or intraosseos; unidirectional or adjustable, titanium or resorbable materials (128).

Only a limited number of long-term follow-up studies on alveolar distraction have been published. In general, implant survival rates are high (90–100%) (129), but esthetic results are not always optimal (130) and complications are frequent (131).

The technique may be used to move an ankylosed infrapositioned incisor into a favorable occlusal position to serve as a long-term provisional solution preserving bone and keratinized mucosa in growing individuals or adults (132) (Fig. 35.9). However, most frequently it is used in skeletally mature patients where a whole alveolar segment is moved in an occlusal direction.

For the placement of an extraosseous distraction device (commercial or custom-made) a high vestibular incision is made, and the buccal side of the alveolus is exposed. Periosteum on the top and lingual aspect of the alveolus is left attached to bone. The distraction device is placed into the correct position and the level of osteotomy is marked. The device is removed and an osteotomy of the transport segment is made with thin burs, saw and osteotomes. Free mobility of the transport segment is secured and the distraction device is repositioned and activated 3–5 mm to ensure it functions. Before wound closure the device is deactivated. Following a latency period of 5–7 days the patient is instructed to activate the distraction device 0.5–1.0 mm per day. The patient is followed closely to ensure problem-free distraction and for inspection of the wound. The distraction is complete when the desired level has been obtained (including 3–4 mm over-correction to compensate for any relapse). After this the device is left passive for 2 months. Implant installation can usually be done when the distraction device is removed and, if there has been an over-correction, contouring of the bone may be needed.

## Segment osteotomy with interpositional graft

A segment osteotomy with interpositional bone graft may be indicated in cases with only limited vertical atrophy of the alveolar ridge (133). The surgical approach is similar to the alveolar distraction osteogenesis. Only, no distraction device is placed. Instead, an "instant distraction" is performed, whereby the transport segment is mobilized in an incisal direction to the correct vertical level. Thereafter, an interpositional graft is placed. An autogenous bone block may be utilized or alternatively a bone substitute block with good mechanical properties (134, 135) (Fig. 35.10). Often the press-fit of the block in combination with the stability of the palatal mucosa will ensure rigid fixation. Otherwise, an osteosynthesis miniplate can be used to fixate the transport segment.

## Bone harvesting

Most autografts used in oral and maxillofacial osseous reconstruction are free grafts. Free autografts may be utilized as blocks or in particulate form and can be harvested intraorally or extraorally according to clinical indication and the amount and type of graft needed (136, 137). The character and quality of bone and thereby also of harvested autografts shows large inter- and intra-individual differences in terms of geometry, cortical-to-cancellous bone ratio, osteoconductivity, osteogenicity and osteoinductivity depending on age, gender, systemic disease, medication, donor site, and harvesting method (138–141). In general, cortical bone has the highest concentration of growth factors, whereas cancellous bone contains the most viable cells (142).

The most frequently used extraoral donor sites for harvesting bone blocks for oral and maxillofacial reconstruction include the iliac crest (anterior or posterior), the tibia, and the calvaria (136). Bone from the iliac crest has a thin outer cortical layer and a large cancellous component, whereas calvarial and mandibular bone grafts are mainly cortical. Correspondingly, grafts from the iliac crest have shown faster revascularization (143) but also more initial resorption clinically than calvarial grafts (144, 145). Usually, extraoral bone harvesting requires general anesthesia, whereas intraoral harvesting procedures most often can be accomplished using local anesthesia with or without sedation (136). In addition, extraoral harvesting procedures are accompanied by more frequent and more severe complications and increased morbidity as compared to intraoral bone harvesting (94, 146–148).

The most frequent intraoral donor sites for bone block harvesting are the mandibular body/ramus and the chin. The architecture of bone blocks from the mandibular body/ramus is mainly cortical, whereas cortico-cancellous blocks may be harvested from the mandibular chin. This is probably the reason for a faster revascularization of chin blocks than mandibular body blocks (149, 150). The morbidity related to bone harvesting from the mandibular body/ramus is low compared to the mandibular chin area (94, 96, 97, 136, 148). Therefore, most authors consider the mandibular ramus as the donor site of choice for intraoral bone harvesting.

### Harvesting methods

All autografts are resorbable. However, their resorbability depends on the donor, the donor site, and the harvesting method (136). Characteristics of autografts harvested using different techniques are listed in Table 35.2.

Different harvesting methods result in very different morphology of autograft particles (151–154). In general, the content of growth factors and viable cells in autografts decreases the more the bone is processed or manipulated during and/or after the harvesting procedure (152, 154, 155). More viable bone cells are found in unprocessed cancellous bone chips than in cortical chips (155). This is also reflected in the finding that more cells can be cultured from cancellous bone in the maxilla than from the more compact bone in the mandible (156).

**Fig. 35.9 Alveolar bone** distraction of an ankylosed permanent incisor. Condition 4 years after trauma. Ankylosis of the right central incisor with severe vertical infraocclusion and horizontal malposition. A radiograph shows sign of ankylosis.

**Surgical procedure**
The transport segment is surgically mobilized.

**Distraction device**
An extraosseous anchored device (Martin® Track 1) is formed and applied. The vector of distraction is adjusted. The use of a screwdriver 3 times a day for distraction 0.3 mm for each turn of the screwdriver, leading to 0.9 mm distraction per day for 8 days.

**Final treatment result**
Ankylosis will, within several years, lead to replacement of the root with bone, ideally formed for replacement by an implant without bone grafting when the crown is eventually lost. In the meantime, the ankylosed tooth will be esthetically and functionally satisfactory for years.

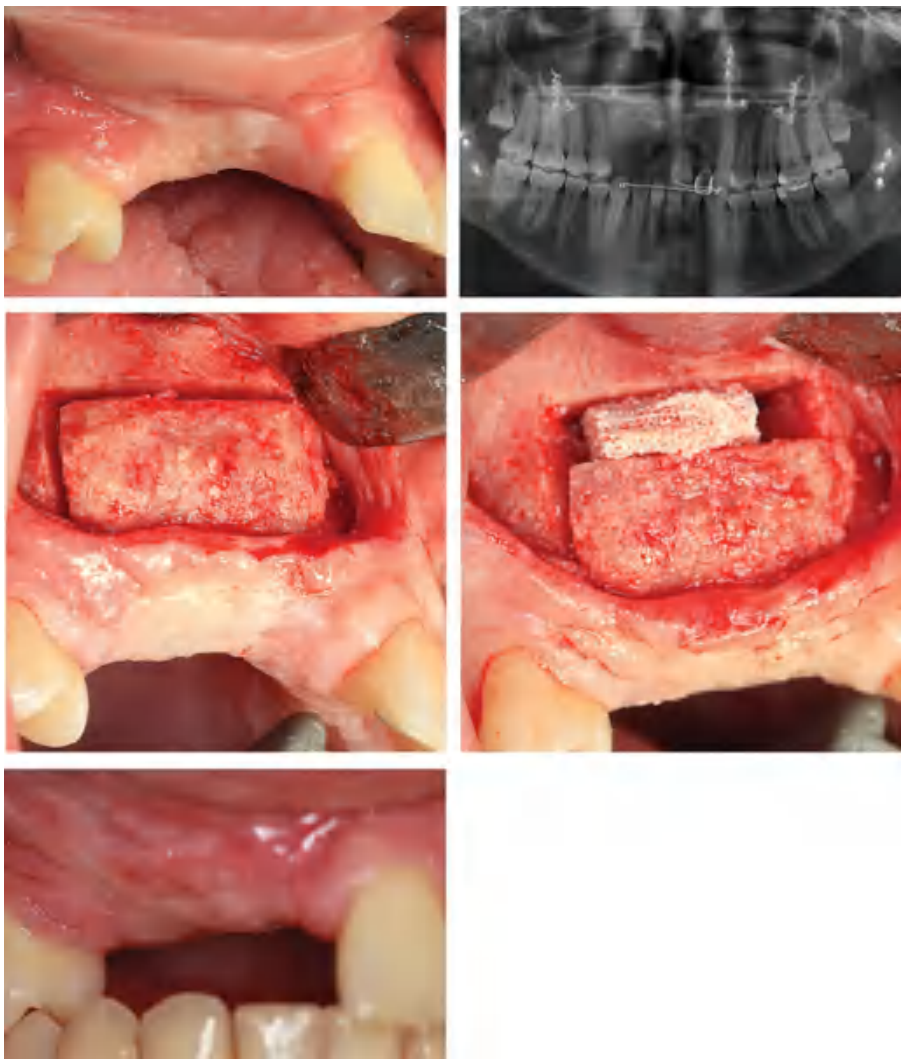


**Fig. 35.10 Vertical atrophy of the alveolar ridge treated with segment osteotomy**

21-year old female patient with vertical atrophy of the upper right maxillary alveolar process. Panoramic x-ray confirming the vertical alveolar atrophy and showing intact marginal bone on first maxillary premolar and left central incisor.

**Transport segment stabilized with xenogenic bone substitute block**

After a vestibular incision, a segment osteotomy is prepared using piezosurgery. The transport segment is mobilized and thereafter stabilized using a coral-derived hydroxyapatite block as interpositional block graft.

**Final treatment result**

Clinical situation 6 months after vertical augmentation just prior to implant placement.

**Table 35.2** Characteristics of autografts and their potential indications. Reproduced with permission from (27).

| Graft type | Osteogeinc cells | Growth factors | Mechanical stability | Resorption | Potential indication |
|---|---|---|---|---|---|
| Cortical block | ++ | ++++ | ++++ | + | Lateral and vertical ridge augmentation in a staged approach |
| Cortico-cancellous block | ++++ | +++ | +++ | ++ | Lateral and vertical ridge augmentation in a staged approach |
| Cortical particles | + | +++ | ++ | +++ | Minor lateral augmentations, peri-implant defects, fenestrations and dehiscence-type defects simultaneous with implant placements<br>Sinus augmentation procedures<br>Packing around blocks<br>Mixing with bone substitute materials |
| Cancellous particles | +++ | ++ | + | ++++ | Minor lateral augmentations, peri-implant defects, fenestrations and dehiscence-type defects simultaneous with implant placements<br>Sinus augmentation procedures<br>Packing around blocks<br>Mixing with bone substitute materials |
| Bone from bone scraper | + | ++ | + | ++++ | Minor lateral augmentations, peri-implant defects, fenestrations and dehiscence-type defects simultaneous with implant placements<br>Minor sinus augmentation procedures<br>Mixing with bone substitute materials |
| Bone from suction device | + | + | - | +++++ | May be mixed with bone substitute materials |

Osteogenic cells and growth factors: + = few, ++ = moderate number, +++ = many, ++++ = abundant.
Mechanical stability and resorption: + = minimal, ++ = limited, +++ = moderate, ++++ = substantial, +++++ = pronounced.

### Particulate from a bone mill

Bone blocks may be particulated using a bone mill. The character of the resulting autograft particles depends on the geometry of the original block and on the bone mill utilized (157). Small autograft particles have a larger surface area, which increases the presentation of growth factors and BMP's as compared to larger particles. As a result, the osteoconduction and osteoinduction are increased (158). However, osteoclasts too have increased access to the enlarged surface area, leading to a higher resorption rate (158). Preparation of a bone block in a bone mill reduces the amount of viable cells in the graft (155). However, the rate of revascularization of the augmented area is significantly higher with particles than with blocks (159).

### Particulate harvested with bone scrapers or bone chisels

Bone scrapers are single- or multi-use devices that collect small bone particles from the bone surface. By scraping on the surface, mainly cortical bone chips are harvested. The size of the particles is smaller than those prepared by bone milling and larger than the ones obtained with piezosurgery and bone filters (160). Despite their cortical origin, *in vitro* studies have documented viable osteogenic cells to be present in grafts harvested with bone scrapers (152, 154, 161). Experimentally, the ability of bone mill- and bone scraper-derived autografts to support osteoblastic differentiation and production of mineralized tissue was significantly increased compared to those harvested with a piezoelectric device and with a bone filter (152). In addition, the concentration of growth factors known to be involved in bone formation, like BMP-2 and VEGF, has been demonstrated to be higher in autografts prepared by bone mill and bone scraper than with piezosurgery and bone filters (160).

### Particulate harvested with piezoelectric devices

Piezoelectric devices work through modulated ultrasound microvibrations and are increasingly used in osseous surgery because there is no damage to neighboring soft tissues, in contrast to traditional rotating instruments. It has been hypothesized that the protective effect on soft tissues may be extended to the osteogenic cells in autogenous bone grafts as well (162). *In vitro* studies have failed to document any beneficial effect of piezosurgery on bone cell viability when compared to other harvesting techniques (152, 160, 162). In contrast, autografts prepared by bone milling or harvested with a bone scraper have shown higher osteogenic potential in terms of cell viability and concentration of bone growth factors than grafts harvested with the piezo technique (160, 162).

### Particulate harvested with bone filters

Viable osteocytes and osteoblasts may also be found in bone particles collected through a filter connected to the suction device (155, 162–164). However, the cell counts, the cell growth on the autografts, and the concentration of bone growth factors is significantly lower than those observed on bone harvested with scrapers, chisels, or processed in a bone mill (152, 154–156, 160). In addition, despite preoperative mouth rinsing with chlorhexidine gluconate and a stringent suction protocol where the device connected to the bone filter is exclusively used for suctioning at the site of implant bed preparation, several studies have documented these grafts to be contaminated with bacteria from the oral micro flora (165–169). Therefore, in general, bone dust from bone filters is not recommended for augmenting the alveolar ridge.

## Long-term prognosis

When osseointegrated implants were introduced to replace missing teeth the most frequent indication was edentulous patients with lack of retention of their full removable dentures. Since then the range of indications have shifted towards a younger population with few or single missing teeth. In general, also patients suffering from traumatic dental injury are young (see Chapter 11). Therefore, there is an increasing demand for valid long-term data on survival and success of implant-retained prosthetic solutions. Few prospective studies have been published including data of implant survival and success after 10 years of loading or more (170–175). In addition, some of the studies present data on implants that are no longer on the market (170, 173). Emerging evidence suggest that the survival rate and success rate of dental implants after 10 years of loading is above 95%.

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# 36

# Esthetic Considerations in Restoring the Traumatized Dentition: a Biologic Approach

B. U. Zachrisson & S. Toreskog

## Tribute to Sverker Toreskog 1936–2013

Sverker Toreskog passed away on February 13, 2013, after a prolonged illness, at age 76. Although he was a prosthodontist, Sverker was well known in the orthodontic community. He was the key member in our Scandinavian interdisciplinary dentistry team. Whereas traditional restorative dentistry depends on destructive tooth preparation to restore teeth, Sverker's minimally invasive and esthetic bonded porcelain techniques opened up a whole new panorama of possibilities.

## Introduction

One of the most challenging problems in dental traumatology is the choice of treatment for replacement of one or more maxillary incisors that have been lost as a result of traumatic injuries. The task is to restore optimal function and esthetics.

In order to produce an optimal treatment result, it is frequently necessary to utilize the combined efforts of a multidisciplinary team of experts, representing orthodontics, prosthodontics, periodontics, oral surgery and dental technology. By using the skills of such a team, the beauty and function of the natural dentition can sometimes be recreated, a fact that can have a significant psychological impact upon a previous trauma victim (see Chapter 9).

The problem of recreating esthetics and function varies significantly according to the *age* of the patient, the trauma *location*, and the *extent* of the traumatic injury. These three factors will be considered in this chapter. Furthermore, a minimally invasive porcelain laminate veneer and onlay technique will be presented, with the main emphasis on restoring teeth after crown fractures.

## Replacement of a missing maxillary central incisor

Several options are available for the treatment of accidentally lost maxillary central incisors in children and adults. This is the most challenging treatment situation in dental traumatology. In the maxillary anterior region there is a maximum demand for an esthetically satisfactory solution that mimics the contralateral non-injured incisor. Any flaws in the appearance of the replacement can easily be seen by a comparison of the anatomy and color of the two adjacent teeth.

The situation generally calls for cooperation among orthodontist, restorative dentist, endodontist and oral surgeon. The armamentarium used by these specialists may include the following techniques:

- Individualized orthodontic extrusion and intrusion to create optimal alveolar bone and gingival contours (1–4)
- Localized simple minor surgeries (5)
- Use of ultra-thin porcelain veneers and new hybrid resins for build-ups on mesially repositioned teeth or transplanted premolars (1–3)
- Vital bleaching of selected teeth (e.g. for canines that have been moved mesially to the position of the lateral incisors)
- Incisal edge contouring
- Reshaping of mesially repositioned canines by grinding (5–7)
- Mesio-distal stripping to solve problems with crowding, interdental gingival recession ('black triangles'), and tooth size discrepancies between the maxillary and mandibular dentition (9, 10).

Basically, there are 4 ways of replacing lost central incisors:

- Orthodontic space closure
- Premolar autotransplantation

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- Placement of a single-tooth implant
- Conventional or cantilever bridge-work.

Each of these options will be briefly discussed below.

## Orthodontic space closure treatment

The movement of the entire lateral segment mesially with the lateral incisor replacing the missing central incisor is indicated when there is a concomitant malocclusion that has to be corrected by orthodontic means. The result of orthodontic space closure is rarely ideal, but the use of several techniques adopted from esthetic dentistry, coupled with careful orthodontic treatment, may provide an outcome that approximates that of a natural dentition (Fig. 36.1) (9–11). This is a biologic approach with good long-term prognosis (10).

The primary concern in order to obtain a satisfactory esthetic result with space closure is a good alignment of the long axis of the lateral incisor (11), and to secure an adequate width along the gingival margin of the replacement porcelain laminate veneer bonded onto the lateral incisor (Figs 36.1 and 36.2) (10). It is also emphasized that the

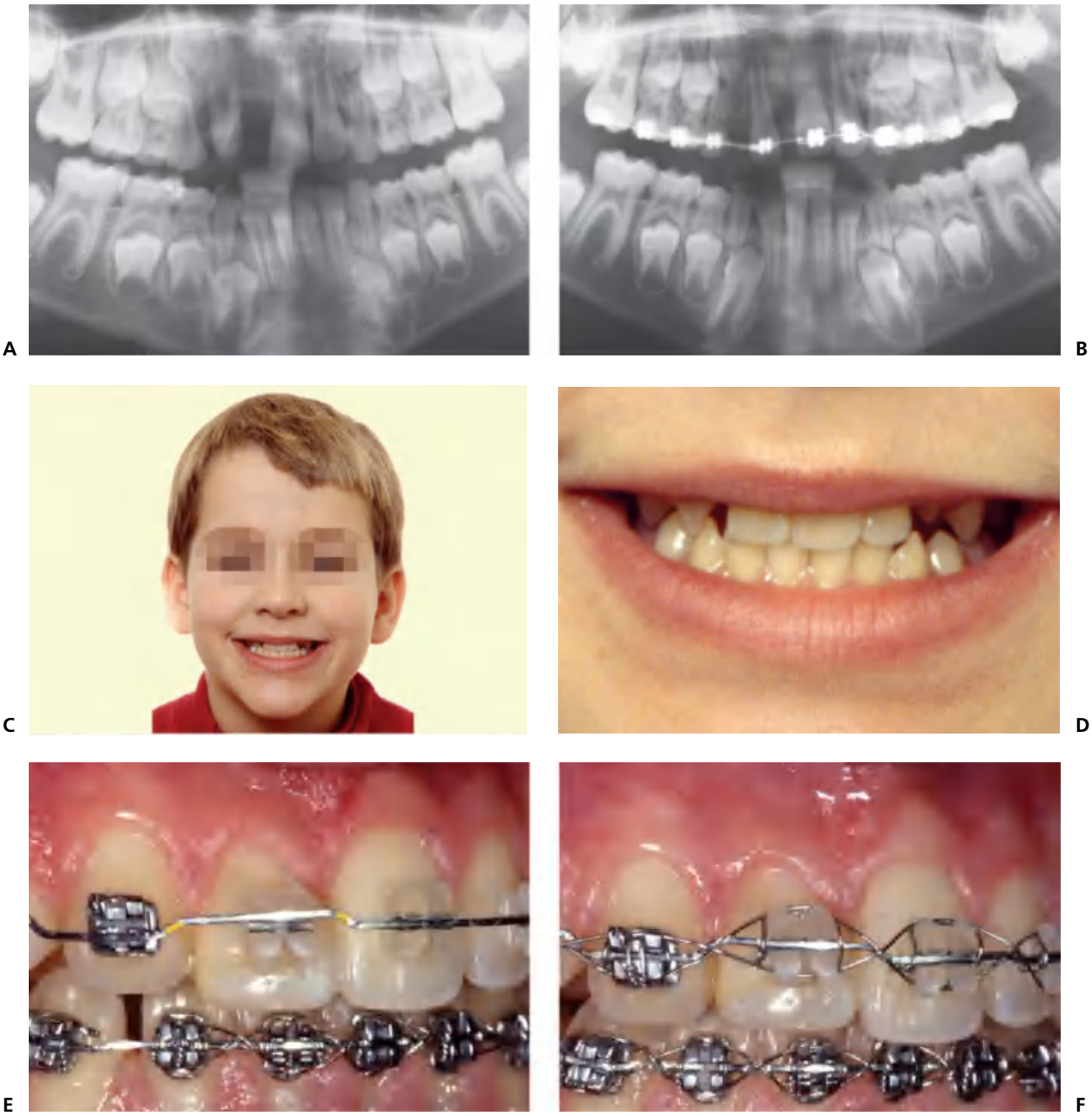


**Fig. 36.1** Orthodontic space closure combined with minimally invasive esthetic dentistry to replace maxillary right central incisor after traumatic accident in 8-year-old boy. Radiographic and clinical appearance is given in A–D. The lateral incisor was moved mesially to the midline with push-coil. Marginal gingival leveling was made by intrusion of lateral incisor, extrusion of the canine, and intrusion of the first premolar, respectively (E–H). The lateral incisor was restored to central incisor shape by a porcelain laminate veneer (I, J). The canine was restored to lateral incisor morphology by grinding and a hybrid composite 'corner' mesially. The first premolar was intruded orthodontically and built-up incisally with hybrid composite (J). Note naturally looking dentition with good occlusal interdigitation and good axial inclinations of all teeth after treatment (K, L).

**Fig. 36.1** (*Continued*)

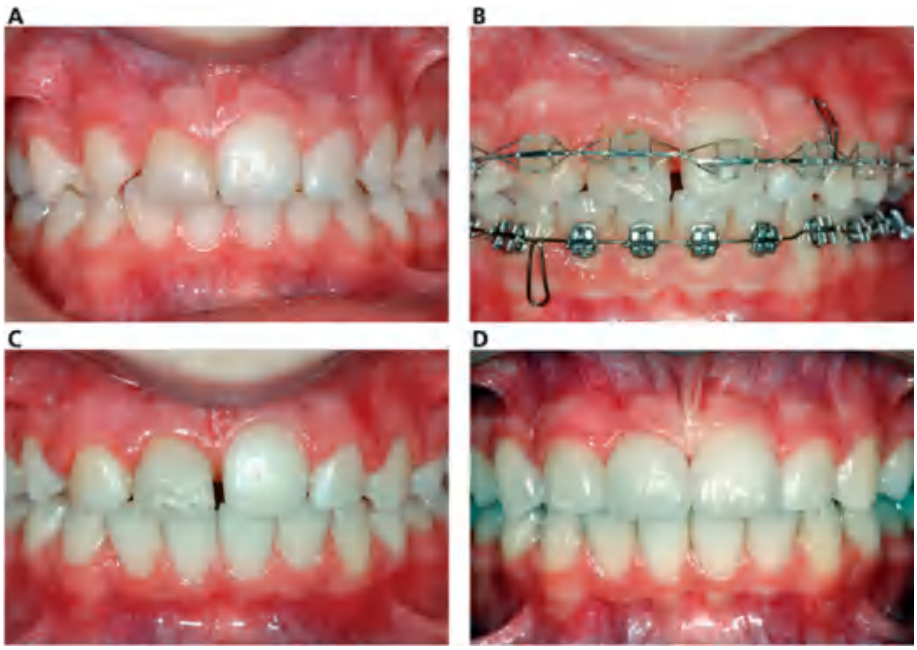


**Fig. 36.2** Young girl with traumatically lost maxillary right central incisor and mesial drift of the teeth (A). Orthodontic space closure was chosen with the purpose to move the entire maxillary right quadrant one tooth width mesially. The midline was corrected (B) and gingival leveling was achieved by intrusion of the first premolar (replacing the canine) and lateral incisor (to replace the central incisor), and extrusion plus grinding of the canine, which was to substitute for the lateral incisor (B). The orthodontic treatment result (C), and the interdisciplinary result with 5 porcelain veneers (D).

canine, now placed in the lateral incisor location, and the first premolar in the canine position, normally need correction of their anatomy and axial inclination (torque), and frequently adjustment of the color. By extruding the canine during the orthodontic treatment, the gingival margin is moved in an incisal direction, and the incisal portion can be ground with diamond burs to lateral incisor shape (7, 8). Sometimes a composite resin mesial 'corner' is useful to obtain better lateral incisor morphology. The first premolar should be intruded to move the gingival margin in an apical direction and be built up incisally with a hybrid composite resin (1–4) (Figs 36.1 and 36.2).

Color differences between canines and incisors may vary. The canines are usually more yellowish and/or darker than the incisors, accentuating the contrast between the maxillary central incisors and the 'new' lateral incisors (1–4). The color difference can be handled by in-office or at-home vital bleaching of single teeth (see below).

The crown torque difference between canines and lateral incisors, and the marked individual variation in crown torque of maxillary canines, must be taken into account and corrected. In a follow-up study, 10 years after orthodontic treatment of space closure cases, where the canines had been moved mesially to replace the lateral incisors, the most common mistake was inadequate crown torque of the mesially relocated canines (10). The lateral incisors normally have labial crown inclination, whereas the canines most often have a lingual crown inclination. If the crown torque of a canine that has been moved mesially is not corrected, the 'new' lateral incisor will not look natural (9, 10).

There is usually a marked tendency for spaces to reopen in the maxillary anterior region after orthodontic space closure and conventional retention with removable plates or splints. For this reason, the retention of space closure cases must not be taken lightly. We recommend long-term (10 years or more) or even permanent retention with lingually bonded multi-stranded wire retainers over 6 teeth, combined with a removable plate to be used continuously for the first 6 months and then at night (1–4, 12).

## *Keys to esthetically optimal treatment*

### Set-ups

A diagnostic set-up on plaster models can identify the tooth-size problems and the amount of crown reshaping that will be needed. It is also advisable to make a pictorial set-up with the patient smiling and/or speaking (1, 13). This will allow the clinician to focus on the crucial aspects of the tooth display – the relationship between teeth, gingiva and lips – and make it age- and sex-related (13–15). The 'visual set-up' procedure is helpful in planning any intrusion and extrusion of different teeth that is needed to obtain normal relationships for the marginal gingival contours. The central incisors and the 'new' canine (i.e. first premolar) should have the gingival margins at the same level, with the 'lateral incisor' (canine) at a more incisal level (Figs 36.1 and 36.2), to provide a natural exposure of the gingiva on smiling. It usually implies that the canines have to be extruded and the first premolars be intruded during the orthodontic treatment (4) (Figs 36.1 and 36.2). This is achieved either by bracket positioning at the start of treatment, or via vertical steps in the archwires. Because the canines are thicker buccolingually than the lateral incisors, their extrusion should be coupled with moving them labially, increasing their lingual root torque, and grinding their incisal and lingual surfaces.

### Cosmetic contouring of canines to simulate lateral incisors

As demonstrated in 1970 by Tuverson (6), it is possible to recontour a canine to almost an ideal lateral incisor shape by grinding with diamond instruments. Iatrogenic effects of grinding, such as increased sensitivity to hot and cold, and other pulp and dentin reactions, can be prevented with careful attention to adequate cooling (7) with abundant water and air spray, and preparation of smooth and self-cleansing surfaces (7, 8). When this technique is used, no harmful pulp or dentin reactions have been observed, even with extensive grinding and incisal dentin exposure. Gross recontouring can be done in one session at the start of orthodontic treatment, with the fine tuning delayed until the end of treatment, if necessary. Since the labial enamel of canines may be thin in the gingival parts, it may be safer to avoid flattening of the labial surfaces of canines and accept a somewhat more rounded labial shape than what is seen on natural lateral incisors. The mesiodistal dimension can also be reduced, particularly on the the distal surface, which may be too convex compared to a lateral incisor. The mesial margins can be corrected with composite resin 'corners' (1). Mesial and incisal build-ups are relatively easy to make with a hybrid of resin restorative materials. They will effectively conceal the difference in morphology between first premolars, canines, and lateral incisors (Figs 36.1–36.3).

With the use of esthetic porcelain laminate veneers, it is possible to recontour mesially relocated premolars and canines into, respectively, 'normal' canine and lateral incisor shapes (Figs 36.1–36.3). Since the porcelain veneers are bonded to enamel, and not cemented, it is possible to increase their cervical width along the gingival margin, without risk for cervical leakage or gingival irritation. By this extension, teeth restored with porcelain veneers can get the proper width along the gingival margin to resemble an optimal central incisor, and the "triangular" appearance of composite build-ups can be avoided (compare composite build-up in Fig. 36.3 C–D with veneer in Fig. 36.3 F–H). Porcelain veneers on the first premolars and canines are more expensive than grinding and resin build-ups, but compare favorably with the cost of restorations on single-tooth implants or conventional composite restorations with their short life-span (see Chapter 29).

### Surgical crown lengthening

When orthodontic space closure is performed in young patients, the marginal gingiva around the mesially moved canine may sometimes become hyperplastic, which can significantly reduce its crown length. A simple localized gingivectomy to the bottom of the clinical pocket will increase the crown length. The same procedure can be used on the first premolar moved into the canine position. As shown in humans, nearly 50% of the excised tissue will regenerate and

**Fig. 36.3** Autotransplantation of mandibular right second premolar to the maxillary right quadrant. Both the lateral and central incisors were lost in traumatic injury (A, B). The premolar crown was restored initially with composite resin (C). Orthodontic treatment was performed to close all spaces, upright the posterior teeth, and level the gingival margins (D). Clinical result after orthodontic treatment and three porcelain laminate veneers shows an almost natural dentition (E–H). Note the change in axial inclinations of the canines and premolars (compare C and F) to broaden the smile. During treatment, the canine to replace the lateral incisor was extruded, and the first premolar to replace the canine was intruded, in order to level the gingival margins before the ultrathin porcelain veneers were made.

become indistinguishable from normal gingiva (16). This means, for example, that if the labial probing pocket depth on the canine is 4 mm, a permanent gain of 2 mm in crown length can be anticipated. Electrosurgery and diode soft tissue laser can be used but are no more effective than using a scalpel. Even if the excision is extended into the alveolar mucosa, the coronal part of the regenerated gingiva will still become keratinized (16). Careful oral hygiene procedures, using single-tufted brushes, are required for 2 months after the gingivectomy so that the regenerated gingiva will appear entirely normal (5).

If a greater crown length increases than 50% of the probing depth is wanted, a surgical flap must be raised, and bone recontouring should be made to about 3 mm away from the desired future gingival margin (17).

**Whitening (vital bleaching of the translocated canine)**

The problem of a relocated canine being more yellowish than the intact lateral incisor can be solved reasonably easy and predictably with either at-home or in-office vital bleaching procedures (18–20) (see Chapter 40). It is generally agreed in esthetic dentistry that yellow discolorations are the easiest to improve. Nocturnal use of 10% hydrogen peroxide gel in a tray is a convenient way of bleaching teeth (18). If only one canine is to be whitened, the bleaching gel is injected only into the canine reservoir. In-office bleaching with stronger gel concentrations may sometimes be preferable for single teeth (19). Recent evidence indicates that the clinical use of light and/or heat during in-office bleaching does not make teeth lighter than with bleach alone, once the teeth have rehydrated for 2 to 5 days following treatment (20).

### Conclusion

Orthodontic space closure with the lateral incisor replacing the missing central incisor, and mesial movement of the entire lateral segment, may be a good option for treatment of patients who have traumatically lost a maxillary central incisor. This treatment alternative is particularly indicated when there is a concomitant malocclusion that must be corrected by orthodontic means. The use of several techniques adopted from esthetic dentistry, together with careful orthodontic treatment, may provide an outcome that approximates the looks of a natural dentition (Figs 36.1–36.3). This biologic approach has a good long-term prognosis, when bonded lingual retainers are used to maintain the treatment outcome (12).

### Autotransplantation of premolars

Over 40 years ago, Slagsvold and Bjercke (21) developed a method of transplanting teeth with partly formed roots. After transplantation, root growth continues, and the teeth maintain their capacity for functional adaptation, and endodontic treatment is usually not necessary. Since then, a series of investigations have supported these findings (see Chapter 33). With regard to graft selection, mandibular premolars appear to be optimal for transplantation to the maxillary incisor region (see Chapter 33). The optimal time for autotransplantation of premolars to the maxillary anterior region is when the root development has reached two-thirds to three-quarters of the final root length. The prognosis for complete periodontal healing at this stage of root development is better than 90% (21, 22). Patients suited for autotransplantation of premolars to the maxillary anterior region are about 9 to 12 years of age, which corresponds with the period when most serious traumatic injuries occur in children. It is remarkable that tooth transplants have inherent potential for bone induction (9, 21) and reestablishment of a normal alveolar process (Figs 36.3 and 36.6).

### Long-term survival and success rates

The long-term outcome of tooth transplantation, including gingival and periodontal conditions, was examined in a study from the University of Oslo (23–25). Patient's attitude about treatment and outcome was also evaluated. The follow-up period for 33 transplanted premolars ranged from 17 to 41 years, with a mean of 26.4 years. Both the survival (teeth still present at the examination) and success (teeth fulfilling defined success criteria) rates were high – 90% and 79%, respectively. The patients generally responded favorably regarding their perception of the treatment. The study showed that autotransplantation of teeth with partly formed roots compares favorably in a long-term perspective with other treatment modalities for substituting missing teeth.

### Orthodontic and restorative treatment for autotransplanted premolars

Because the root of an autotransplanted premolar continues to develop and a normal periodontal ligament is established, such teeth can be moved orthodontically like any other tooth that has erupted into occlusion (24–26). It is generally recommended to wait for an observation period of 3–4 months before orthodontic treatment is started. Premolar crowns can be reshaped to resemble incisor morphology (Figs 36.2 and 36.3). A direct composite resin build-up can be performed and later replaced with a porcelain laminate veneer (PLV) (Fig. 36.3). There are some drawbacks with the composite resin build-ups because it is difficult to establish normal incisor width along the gingival margin, and the build-ups tend to get a triangular crown form (Fig. 36.3C, D). Furthermore, some composite resins tend to discolor with time. Both problems can be solved by using thin PLVs instead (Fig. 36.3E–H). The 2 main reasons to avoid cemented crowns in children and adolescents – that the large pulp chambers limit preparation and that the gingival retraction over time could lead to unesthetic root display – are not valid for PLVs. The reflection of light will not be stopped by an enamel-bonded PLV, and any later root exposure will display a normal color and no darkening of the gingiva (Figs 36.6 and 36.7).

In a recent study, 45 premolars autotransplanted to the maxillary incisor region in 40 adolescent patients were evaluated, after restoration, with a mean observation period of 4 years (24). Mean age at surgery was 11 years. Clinical criteria assessed tooth mobility, plaque and gingival conditions, probing pocket depths, and reaction to percussion. The interproximal gingival papilla fill was assessed according to an index. Pathosis, pulp obliteration, root length, and crown-root ratios were studied on standardized radiographs. The result showed that the clinical variables for transplants did not differ from those of the natural incisors, except for some increased mobility and more plaque in a few transplanted premolars (24, 25). The interproximal gingival papillas adjacent to all transplanted teeth were normal or slightly hyperplastic, and no interdental gingival recesssion (black triangles) were seen.

### Esthetic outcome and patient satisfaction

A comprehensive study comparing the esthetics of 22 autotransplanted premolars reshaped to incisor morphology with their natural, intact, contralateral incisor was made (25). Features considered important for esthetics (color, soft tissue appearance, tooth morphology and position) were compared. Most of the transplanted teeth matched the contralateral incisor, and most patients were satisfied with the appearance of the transplant. The distribution in set categories assessed professionally and by the patient was not significantly different. However, the color and the gingival width of the transplanted tooth were scored as different from the natural incisor when the restorations were made with composite resin. Thus, the findings demonstrated that interdisciplinary treatment planning is important for successful esthetic results and that porcelain laminate veneers rather than composite build-ups should be used for restoration of the crowns (Figs 36.3–36.7).

### Conclusion

Premolar autotransplantation represents an attractive, and maybe the best, solution to difficult treatment planning problems when a maxillary incisor is traumatically lost in young patients (Fig. 36.3). The transplanted tooth represents a normal root with normal periodontal membrane, and it can be moved orthodontically like any other natural tooth.

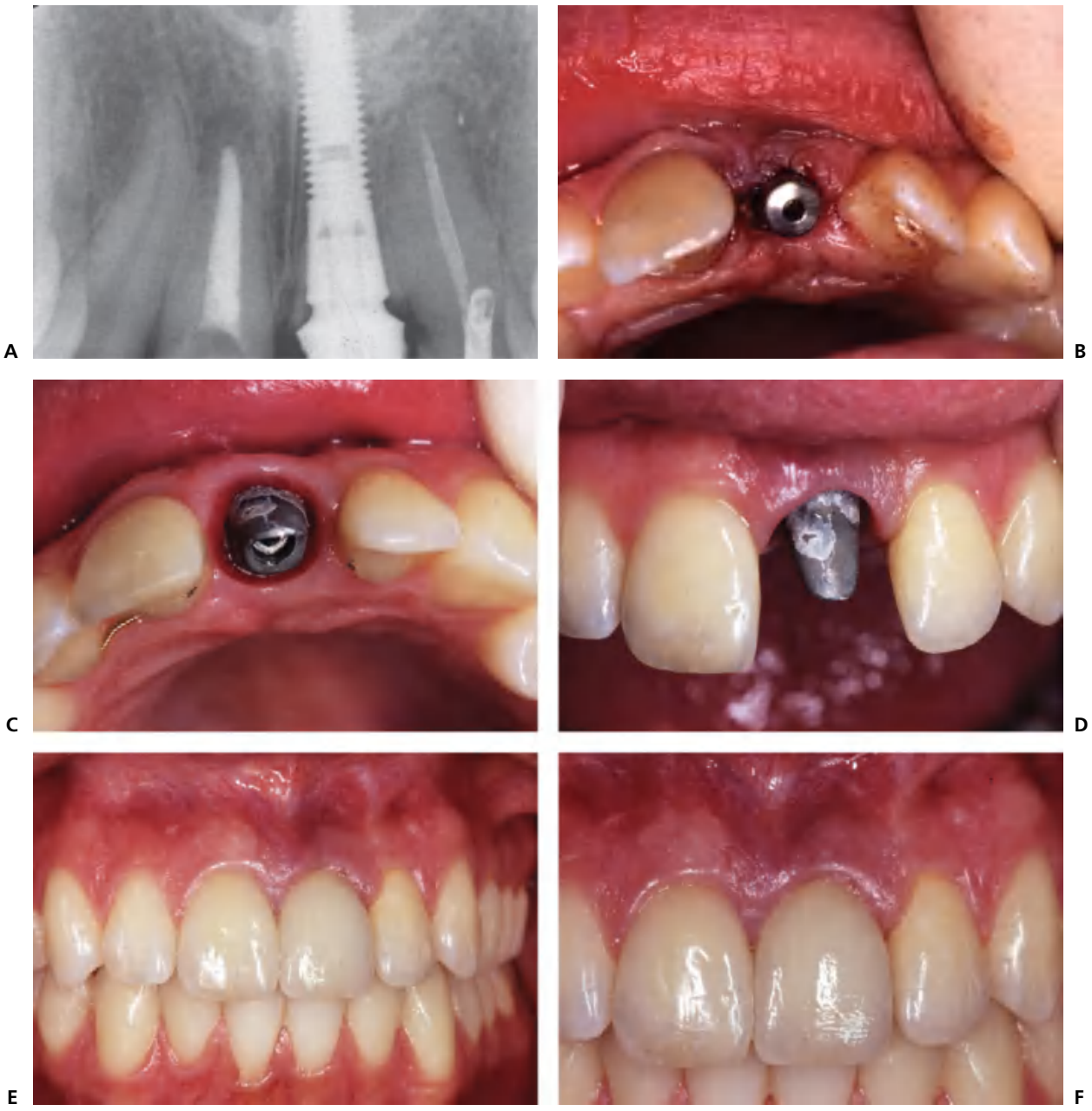


**Fig. 36.4** Implant-supported porcelain crown used to replace a central incisor. A 24-year-old female patient has, due to trauma, lost a left maxillary central incisor. A, B. Condition immediately after implant insertion. C,D. Sculpting the gingiva. By the use of a temporary crown the labial and interdental gingivae have been optimally reshaped. E,F. A good esthetic result has been achieved; however, there is a slight reduction in the height of the distal papilla.

The 'abnormal' crown should be restored with a porcelain veneer. Transplantation represents a biologic approach in which the transplanted tooth germ retains the potential to induce alveolar bone growth.

## Single-tooth implants

The use of osseointegrated implants to replace missing incisors has become a common treatment solution for patients 20 years of age and older (see Chapter 35). Experience gained to date with single-tooth implants is favorable, with survival rates of about 90% after 10 years in multicenter studies. However, filling an anterior space with an implant-supported porcelain crown is a major challenge from both the esthetic and functional aspects (Chapter 35). Clinical success depends not only on persisting osseointegration but also on harmonious integration of the crown in the dental arch (Fig. 36.4).

Recent studies indicate that the esthetic result for single implants in the maxillary anterior region is sometimes suboptimal (27–30). There are several potential esthetic problems associated with the long-term use of implant-supported crowns.

### *Age changes in tooth position*

An implant is, by definition, ankylosed and cannot change position, in contrast to the neighboring natural teeth. Occlusion over time is a result of developmental and

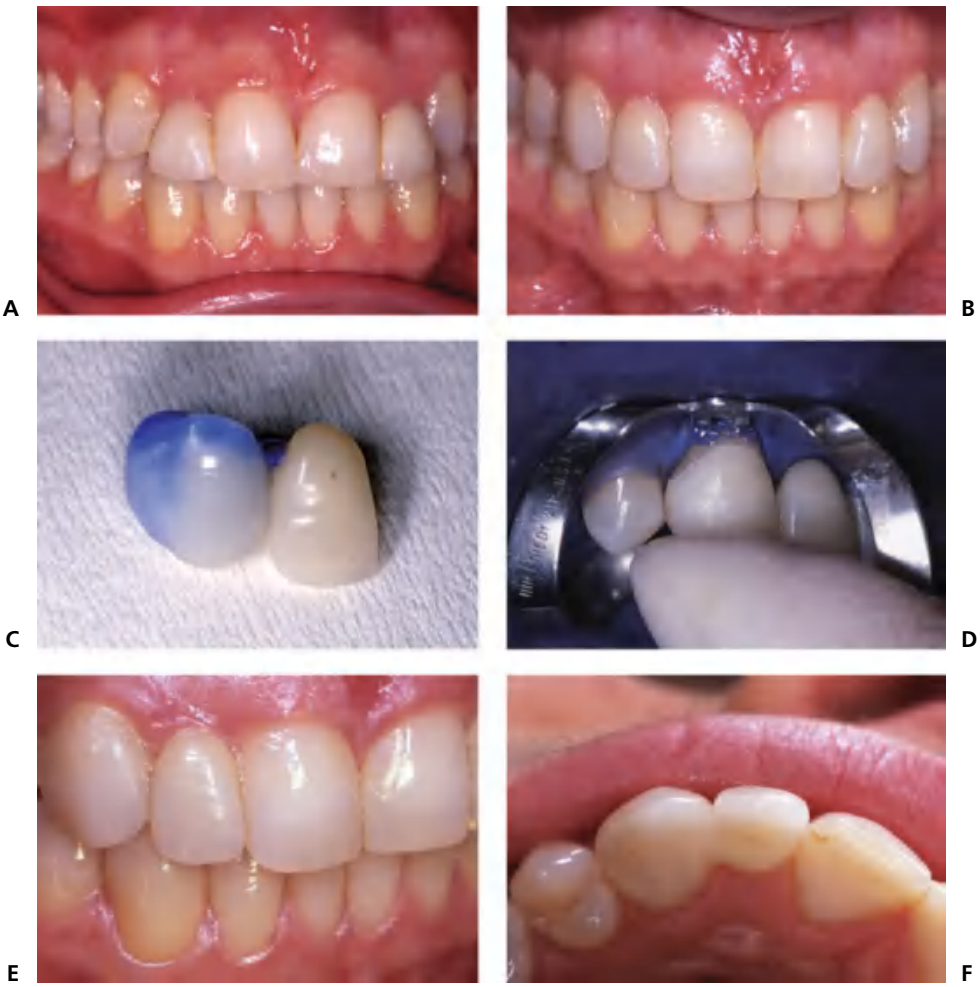


**Fig. 36.5** Cantilever fiber reinforced composite bridge. Patient with 2 existing unesthetic 3-unit Maryland bridges replacing congenitally missing upper laterals (A) was substituted with two 2-unit cantilevered fiber reinforced (Stich Tech) bonded composite bridges (B). The shallow cervical preparation is illustrated by the translucency of the composite (C) as the blue cleaning aid is kept on the inside of the crown. The rubber dam placement exposes the bridge site plus the contact points of the adjacent teeth (D). Note the cervical fit on the prepared canine. Close-ups at cementation (E) and 5 years later (F).

adaptive processes in which facial growth, dynamic interrelationships between aging facial structures, dental eruption, function, tooth wear and orthodontic relapse can contribute (27). These processes show much individual variation throughout life (26, 27, 30–32). Even small tooth movements after implant placement can cause esthetic problems (29, 30, 32). Complications like tooth-implant contact displacements after completed orthodontic treatment can also occur, for example as a consequence of unwanted tipping of neigh boring teeth after retention (27). A recent study from the University of Geneva documented clinical infraocclusion of implant-borne restorations not only in a group of young adults (15–21 years) but also in most of the patients in a group of mature adults between 40 and 55 years of age (30).

Obvious dentofacial changes take place over the years in adult patients (31, 32). Increasing degrees of infraocclusion may occur even after completion of skeletal growth (27). Long-term studies more than 10 years after implant placement may therefore show slight vertical eruption of the natural upper anterior teeth combined with retroclination (31, 32). This implies that implant crowns placed between the teeth may in time show infraocclusion and proclination (Fig. 36.10).

### *Gingival recession and dark margins along porcelain crowns*

An implant-supported crown placed in a young adult should be expected to have a long life. During this time, which might span 60 or more years, the marginal and interdental gingival tissue surrounding the implant crown is unlikely to remain unchanged. Gingival recession might occur due to overzealous toothbrushing or to periodontal disease in adult and elderly patients. Gingival recession may result in a darkening effect along the exposed implant crown (see Fig. 36.3).

### *Lack of gingival papilla fill, probing depths, and bleeding*

The difficulties in obtaining natural marginal gingival contours around implant-supported porcelain crowns is partly a result of the relationship between implants and the bone and gingiva surrounding them. The reduction in osseous scallop from facial to interproximal areas and lack of difference in gingival heights above bone from facial to interproximal, compared with those of natural teeth, may lead to a flat gingival form (33). In a study comparing the soft tissue characteristics of implant-supported single-tooth crowns and their

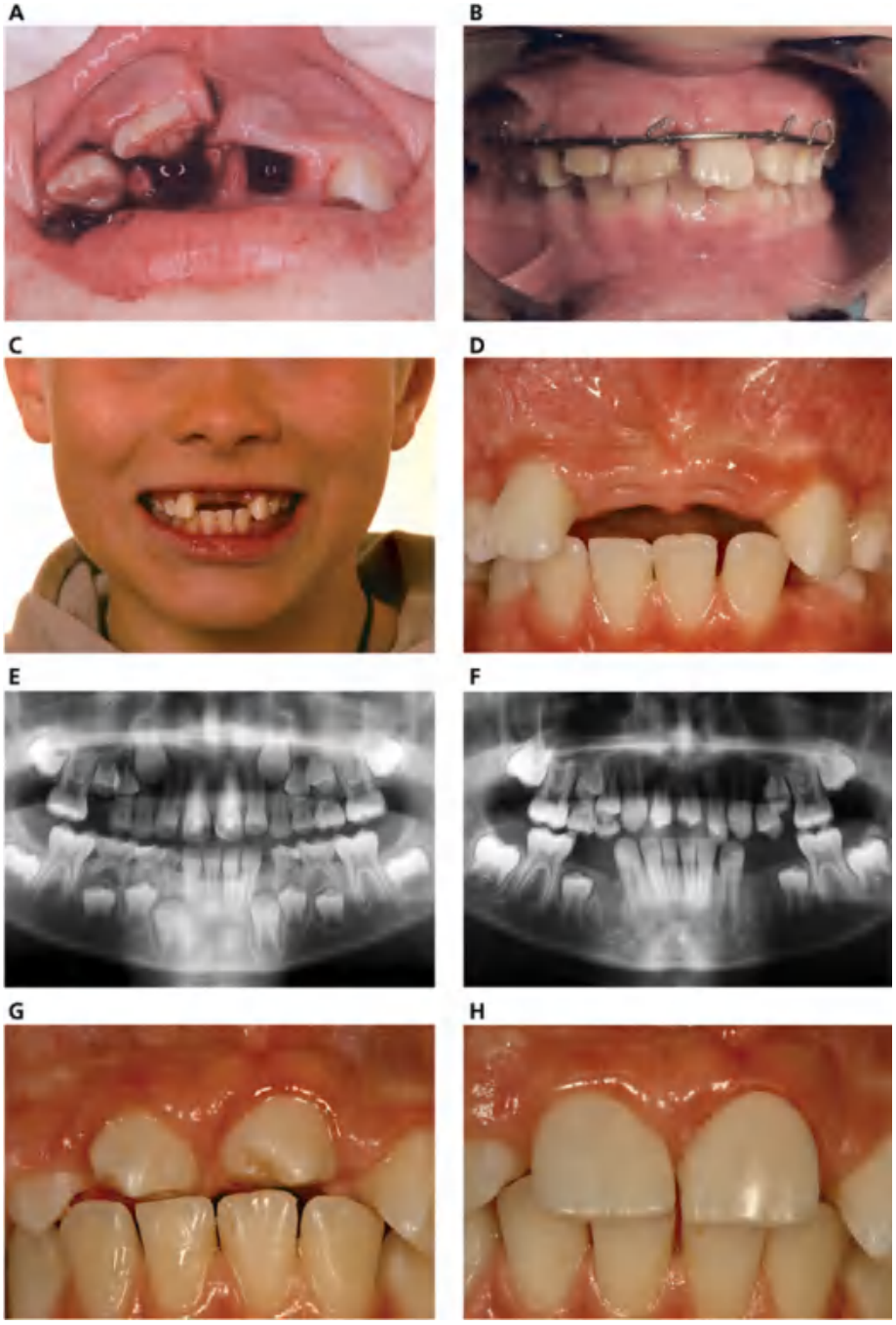


**Fig. 36.6** Replacement of 3 maxillary incisors lost in severe traumatic injury (A, B). The right lateral incisor was ankylosed and extracted. Two mandibular first premolars were autotransplanted to the wound region (E–G) and provided with temporary composite resin build-ups (H). I–K shows typical continued root growth (3, 10 and 16 mos. after transplantation) and progressive pulp canal obliteration. Orthodontic space closure (L,M). Orthodontic treatment result (N) and interdisciplinary treatment result with 4 porcelain veneers (O). P–U shows the clinical result at 9 years follow-up.

contralateral natural teeth in the esthetic zone of the maxilla (28), it was demonstrated that most implant crowns displayed some lack of interdental papilla fill, particularly on the distal papilla (Fig. 36.4). Such 'dark triangles' between teeth are rarely, if ever, observed with orthodontic space closure treatment (10) or after premolar autotransplantation (23–26). In addition, implant crowns have more mucositis/gingivitis, increased probing depths, and more bleeding on probing (28) than natural teeth.

### *Buccal alveolar bone loss and gingival discoloration*

In an award-winning 10-year follow-up study by Thilander et al. (27) of implant-supported crowns replacing maxillary incisors, some patients showed progressive loss of marginal bone support at the buccal aspects of the implant. The ongoing buccal bone resorption resulted in discolored soft tissue, gingival retraction, or a denuded implant due to vigorous toothbrushing. In a recent study, blue-coloring of the labial

**Fig. 36.6** (*Continued*)

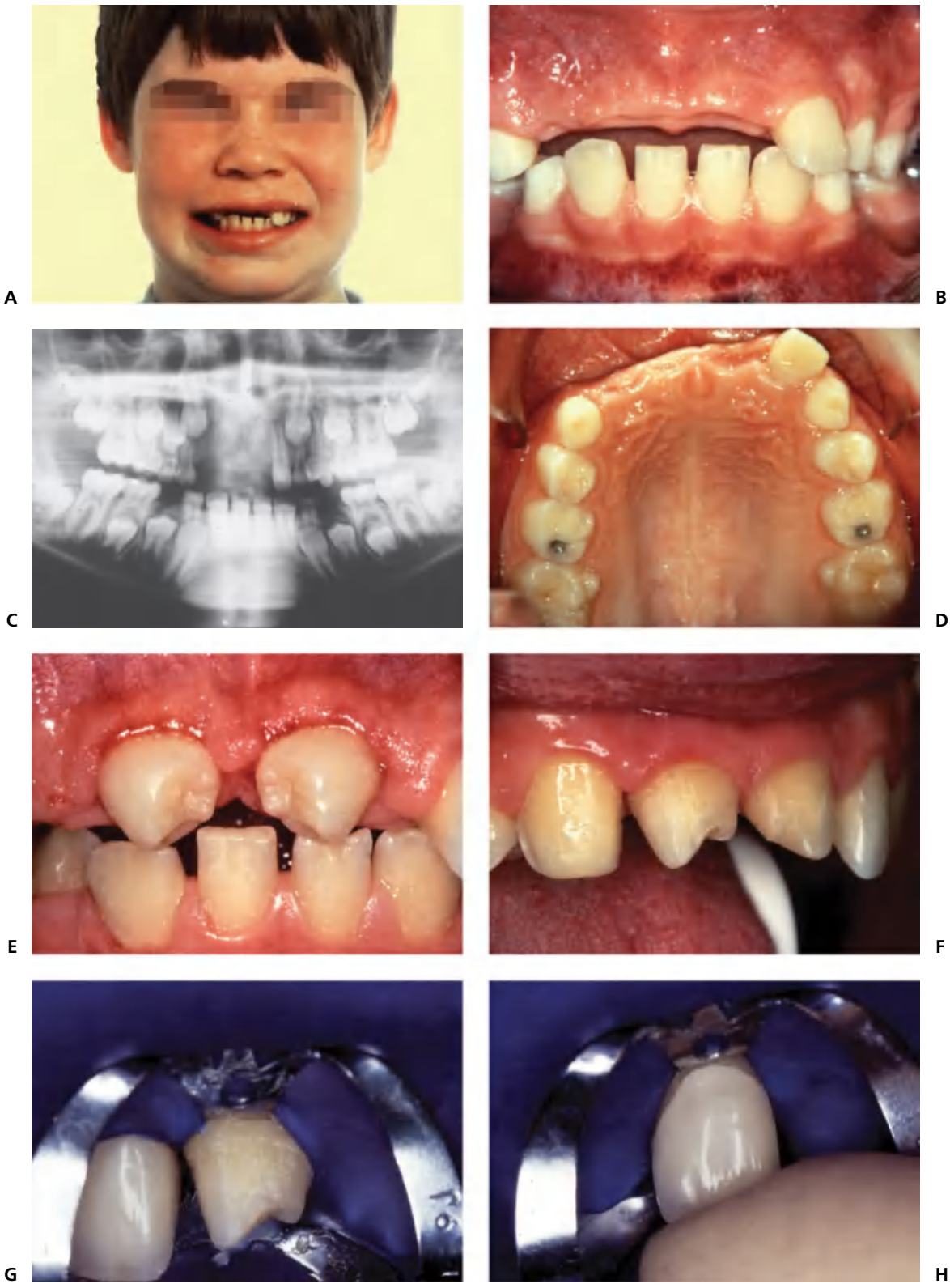


**Fig. 36.7** Replacement of three lost incisors with two autotransplanted premolars and space closure in the canine region in a 10-year-old boy due to trauma. Note the loss of horizontal bone in the trauma region (B–D). E–H. Premolar transplantation and restoration. Two mandibular premolars were transplanted to the central incisor region and later restored with porcelain laminate veneer crowns. The canine was orthodontically moved into the lateral incisor region and restored in the same way. I–L. Clinical result after 12 years. Note optimal shape of alveolar bone and dental arch form (J, K) (compare with the clinical situation before restoration, B, D). M, N. Clinical result after 14 years. The gingival recessions that occurred show a natural appearance, since the thin veneers reflect light like intact teeth. There is no darkening effect along the exposed root surfaces.

**Fig. 36.7** (*Continued*)

gingiva or visible implant crown margin occurred in 57 % of the patients already at the 4-year follow-up (35). The buccal bone plate in the incisor region is often very thin. Sometimes resorption can occur even when the implant had sufficient alveolar bone support at time of placement (Fig. 36.10 compare A with E).

### *Conclusion*

The single-tooth implant may be a useful treatment option in adults when a maxillary central incisor has been accidentally lost. Implants should normally not be placed until the skeletal growth is completed. Careful procedures, including gingival sculpting with the provisional crown are needed to obtain adequate soft tissue thickness interdentally and labially (Fig. 36.4E, F). Independent recent studies indicate that the clinical long-term result for single implants replacing maxillary incisors is sometimes esthetically suboptimal (34, 35). Extended long-term evaluations are needed.

## Fixed prosthetics

Although this treatment appears to have reasonably good long-term success (36), it has several shortcomings related to the common alveolar process atrophy in the trauma region. Furthermore, conventional abutment preparation may compromise the long-term esthetics (see Chapter 31). Fixed prosthetics appears today not to be an attractive treatment option in young patients.

## Orthodontic implant site development

Orthodontically guided regeneration in adults is a nonsurgical means of regenerating alveolar hard and soft tissues associated with implant placement after tooth extraction (36, 37). The major advantage of the orthodontic implant site development (OISD) is the possibility to rebuild (and/or overbuild) the hard and soft tissues (Fig. 36.8) to compensate for expected postextraction remodeling, which

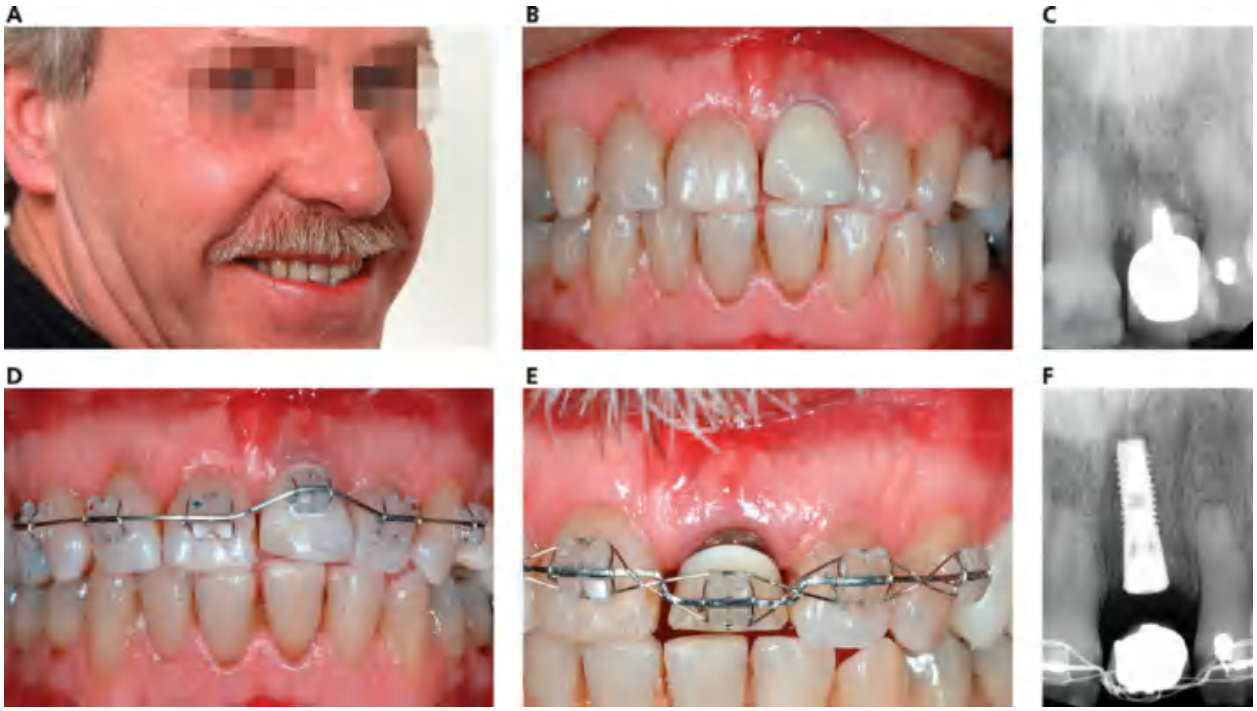


**Fig. 36.8** Orthodontic implant site development in adult male patient. The maxillary left central incisor had poor prognosis and more apical and thinner gingival margin than on the adjacent central incisor (A–C). Forced extrusion of the left central incisor, with bracket bonded apically, and initial leveling with superelastic wire (D). Interrupted continuous forces using step-bends were used to gradually extrude the incisor. The incisal edge was progressive ground out of occlusion with opposing arch. Increased root exposure (E) confirms that gingival tissue does not completely follow the extruded tooth. Note increase of labial gingival thickness. Both labial soft tissue and alveolar bone followed the extruded incisor enough to allow placement of implant-supported crown.

otherwise may provide discrepancies in gingival margin levels between an implant crown and the contralateral natural tooth. The surgical experience for the patient is significantly reduced by the avoidance of complex subepithelial connective tissue grafting procedures simultaneous with immediate implant placement (38), which may be painful, and complicated with unpredictable healing. Hence preservation takes place rather than reconstruction or replacement (36). It negates the risk of resorption and gradual loss of graft volume seen over time with most surgical grafting techniques (38). The soft tissue result is as good as or better than that achieved with any surgical reconstruction (Fig. 36.8). The main indication for OISD is for adult patients in need of conventional orthodontic treatment and who need to have one or more teeth extracted in the anterior maxilla for future implant placement (Figs 36.8 and 36.9). The technique is particularly indicated to avoid recessions in persons with thin gingival biotypes.

The creation of alveolar bone and gingival tissues by means of selective extrusion of single teeth with fixed orthodontic appliances utilizes the normal tissue reaction to tooth movement (39). When a tooth is extruded, both the alveolar bone and the soft tissues will follow the tooth. The bone deposition is a result of the tension exerted by the stretched fibers, transferring mechanical strain to cells in the bone. A series of mechano-transduction mediators (Indian hedgehog) are expressed and lead to bone formation. The periodontal fibers elongate, and new bone will be deposited in areas of the alveolar crest along the stretched fiber bundles. The ability to affect the environment is maintained along the entire attachment apparatus of the root. This offers a unique possibility to use teeth designated as "hopeless" for regenerative purposes. Salama and Salama (40) recommended that, when feasible or necessary, the designated hopeless tooth should be extruded almost to extraction to achieve maximal benefits. The term "orthodontic extraction" was suggested, because its purpose is to extract the tooth with all the augmentative benefits inherent to the orthodontic extrusive process. During the extrusion, the crown should be ground with burs at each visit (1 or 2 mm each time) to avoid jiggling with the opposing teeth, which would probably be detrimental to the "new" bone. When tooth sensitivity appears as incisors are shortened, provisional endodontic treatment with calcium hydroxide or simple pulpectomy may be sufficient. Animal and human studies (41–45) have demonstrated that the gingival margin will move coronally about 70–80% of the distance a tooth is extruded (Fig. 36.8). The orthodontic extrusion only relocates the existing attachment in a coronal direction. It does not create a new attachment.

A slow extrusion rate apparently is most appropriate to preserve the integrity of the residual periodontal attachment. In the absence of scientific data about the optimal force system to be used (37, 42, 44), we recommend using interrupted continuous forces which allow rest periods (for consolidation of the extruded tissues) between monthly activations. Placing small vertical step-bends on light wires will provide about 1 mm of extrusion per month (37, 43, 44). The

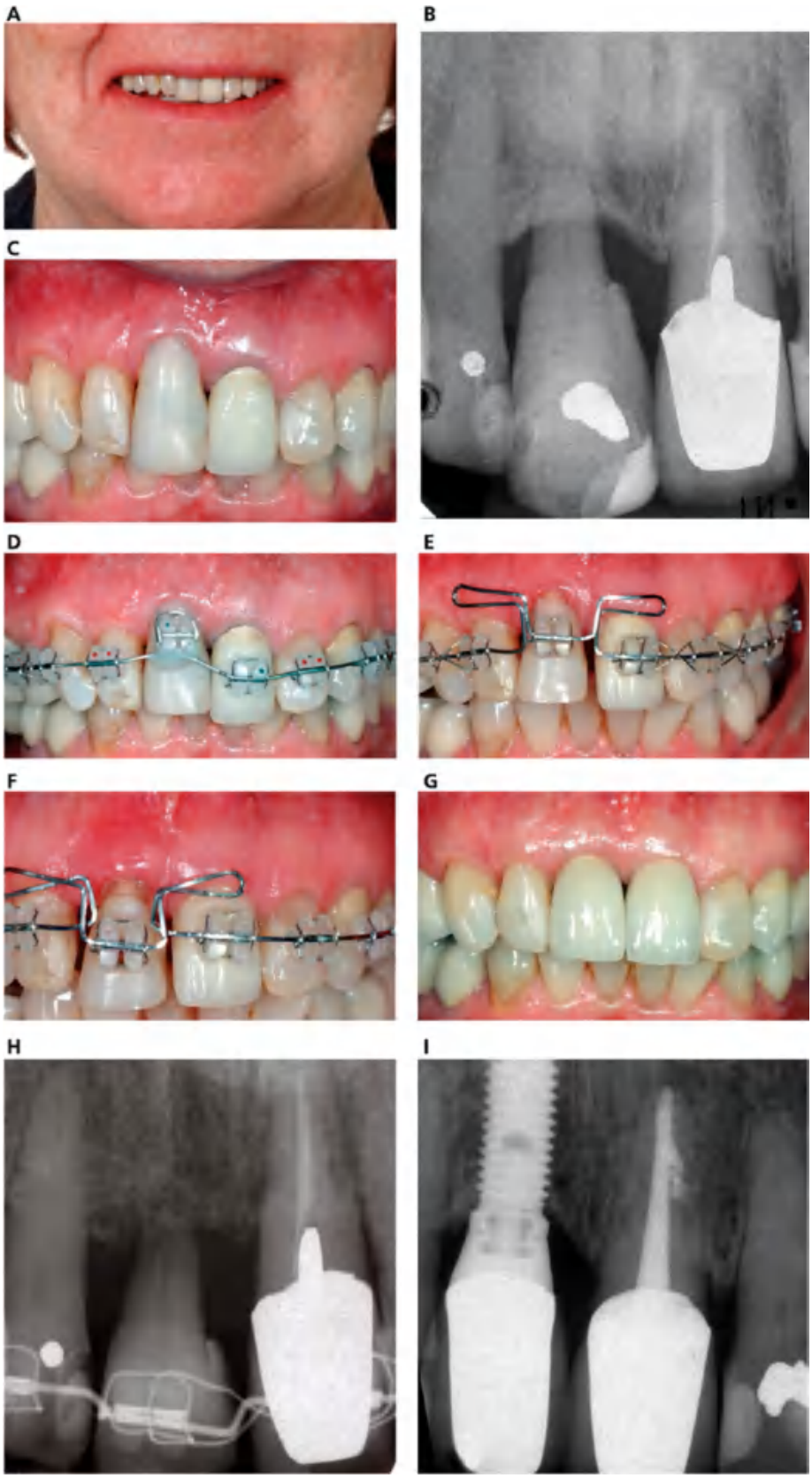


**Fig. 36.9** Orthodontic implant site development in adult female patient with "hopeless" right central incisor (A–C). Careful gradual extrusion of the very long incisor (D–F) and incisal grinding to avoid jiggling. The vertical bone defect (C) was converted to "horizontal" pattern (G) with considerable gain of alveolar bone and soft tissue. The esthetic improvement was noteworthy after placement of a porcelain crown on an implant (H,I).

decision when to end extrusion is based on the observation of the new bone level. Generally, the goal is to exceed by at least 2 mm the bone level of the contralateral tooth, which may act as reference (Fig. 36.8). After the site is well developed, a period of 3–6 months may be applied to allow the newly formed matrix to mature into more stable bone. The stable type of bone may accept an implant without showing relapse (Figs 36.8 and 36.9).

The esthetic significance of the orthodontic bone and soft tissue relocation is obvious. The reduced regenerative

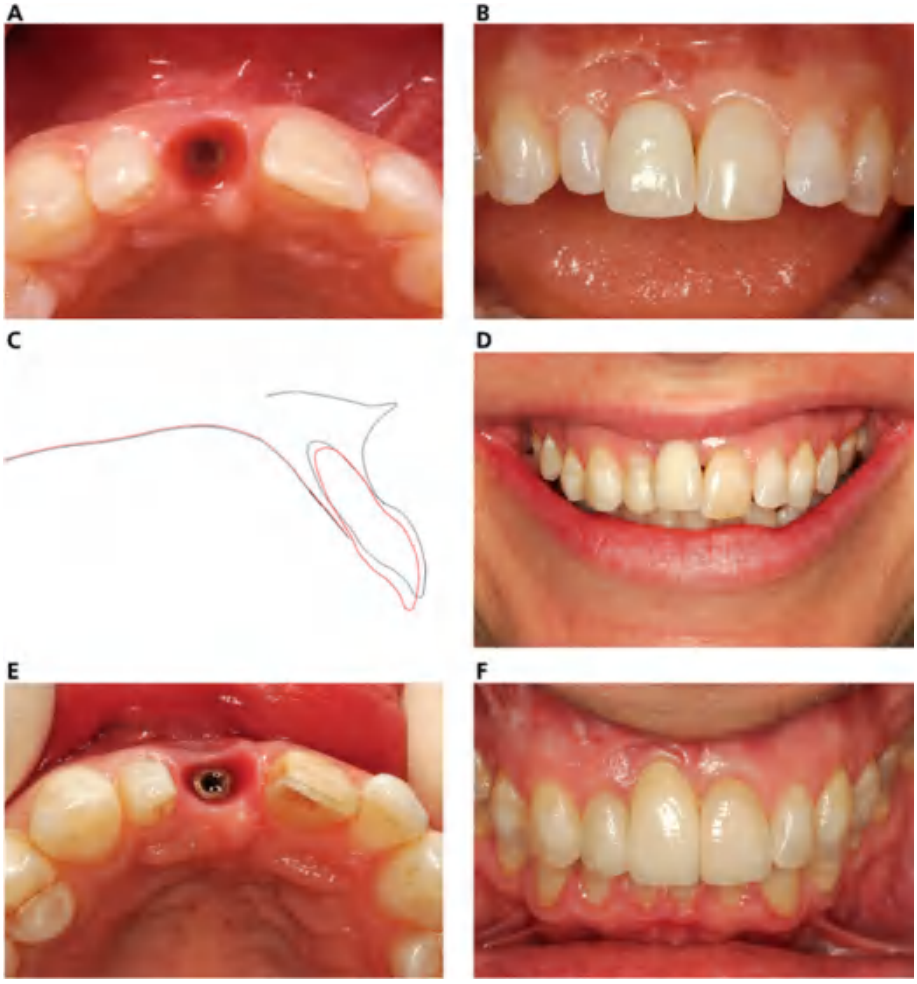


**Fig. 36.10** Undesirable long-term outcome of implant crown in the anterior maxilla of adult female patient. At time of crown placement when the patient was 24 years old, labial bone and soft tissue coverage appeared normal (A,B). 12 years later, the crown had progressively infra-occluded and was also in labial position relative to her natural teeth (C,D). The crown had gradually become too short incisally and too long gingivally (D). When implant crown was removed for reconstruction, collapse of labial soft tissue was evident (E), caused by gradual loss of labial bone coverage. Tracing of implant porcelain crown (black) and natural teeth (red) 12 years after implant placement showed that the natural teeth had continued to extrude and upright while osseointegrated implant crown maintained its position (C). New implant crown was made by Dr. Toreskog, along with porcelain veneers on remaining incisors (F); despite all efforts, the final smile was unsatisfactory.

potential of a significant defect environment may otherwise force a more apical and less ideal placement of the implant, and lead to unesthetic compromises in the form of longer restorations or uneven gingival margins.

Orthodontic implant site development can also be made in the horizontal plane. If an implant cannot be placed because of reduced bucco-lingual ridge thickness after a previous extraction, a tooth (generally a premolar) can be moved into the edentulous space. By slow bodily movement of the tooth into the edentulous site, the periosteum on the buccal and lingual surfaces of the alveolus will form alveolar cortical bone. The bucco-lingual volume of the new bone on the tension side will be remarkably increased, and an implant can then be placed in the position previously occupied by the tooth that has been moved (27, 43, 44, 46, 47). But if the tooth is moved too rapidly, a dehiscence may occur (47).

## Replacement of a missing maxillary lateral incisor

The cause for absence of the maxillary lateral incisor may be traumatic loss of the tooth or congenital agenesis. This is a relatively frequent problem. Basically, there are four ways of replacing missing lateral incisors, namely *orthodontic space closure*, insertion of a *single implant* (directly, or after orthodontic space opening), *autotransplantation* of a developing premolar, or restoration with conventional or cantilever resin-bonded *bridge-work*. The longevity of these treatment solutions is described in Chapter 31. In the following, esthetic advantages or shortcomings will be described for these treatments.

### Orthodontic closure

When a maxillary canine is moved mesially to substitute for a missing lateral incisor, and the first premolar is used in place of the canine, the same esthetic problems occur as those discussed above for replacement of a missing central incisor. When careful orthodontic treatment procedures are combined with clinical techniques adapted from esthetic dentistry, the outcome with space closure can be very satisfying, and almost indistinguishable from a natural dentition (1–4). Long-term periodontal and occlusal studies on congenitally missing lateral incisors have shown that space closure with premolar substitution for canines may lead to an acceptable functional relationship, with modified group function on the working side (4, 48, 49).

In a recent study, the long-term result (mean 7 years after treatment) of prosthetic replacement of maxillary lateral incisors were compared with orthodontic space closure (49).

The space closure patients were more satisfied with the treatment results than the prosthesis patients; there was no difference between the two groups in prevalence of signs and symptoms of temporomandibular joint (TMJ) dysfunction; and patients with prosthetic replacement had impaired periodontal health with accumulation of plaque and gingivitis. It was concluded that orthodontic space closure produced results that were well accepted by patients, did not impair TMJ function, and encouraged periodontal health in comparison with prosthetic replacement (49).

Similar findings were made in a recent retrospective long-term study (10 years after treatment) by Rosa et al. (4) of 26 consecutive adolescent and adult patients with agenesis of maxillary lateral incisors, after orthodontic space closure with first premolar intrusion and canine extrusion. The control group included 32 orthodontic patients with no missing teeth and no need for extractions. The long-term periodontal tissue health and the incidence of dysfunction or TMD signs were similar in the space-closure agenesis group and in the control group of non-extraction patients.

### Implants

Although this treatment solution is at present a popular option, it represents a considerable challenge from an esthetic point of view, particularly in the long-term perspective. An implant-supported crown placed in a young adult should have an expected life span of 60 or more years. It is difficult for dental technicians to make an implant-supported crown with optimal shade and translucency that blends perfectly with the neighboring teeth (52). In our experience, the color of a canine that has been moved mesially and vital-bleached (and/or restored with a thin translucent porcelain laminate veneer) frequently comes closer to that of adjacent teeth than the color of porcelain crowns on implants. Even in adults, the esthetic outcome of implant-supported crowns in the maxillary anterior region may sometimes lead to esthetic results that are less optimal than desired (27–32, 50–52).

In a recent study from the Czech Republic on 26 patients (aged between 18 and 54 years) with unilateral agenesis of a maxillary lateral incisor, dental implant restorations were compared with their contralateral natural lateral incisor (52). Mean time after implant insertion was 6.5 years (range 1–12 years). All patients received orthodontic pretreatment prior to implantation. Professional assessments (shape, size, color, gingival margin position, and overall appearance) were registered, and patient satisfaction was analyzed by means of questionnaire. The results showed that none of the implant crowns satisfied all professional parameters, and the majority of implant crowns worsen with time. Still, 96 % of the patients were satisfied or rather satisfied with the outcome.

### Autotransplants

Whereas premolar autotransplantation may represent the best treatment option for a lost maxillary central incisor in a young patient, it does not represent a common treatment choice for replacement of a missing lateral incisor. One of the reasons is that most premolars would normally be too wide mesiodistally, an exception being the mandibular first premolars. When selected, therefore, the transplantation generally is associated with some form of orthodontic treatment.

### Resin-bonded cantilever prosthetics

Since most maxillary lateral incisors are small teeth that take less load than the central incisors in occlusion and function, the use of fiber-reinforced resin-bonded cantilever prosthetics (53) represents an interesting esthetic treatment alternative for replacement of missing lateral incisors. Clinical experience with this technique in the esthetic zone over the past few years is promising. Most commonly, we have used the canine as the abutment tooth, with a cantilever lateral incisor (Fig. 36.5). Since there are presently no translucent ceramic materials with adequate strength, we currently use a fiber-reinforced (Stick Tech®) indirect laboratory composite resin (Sinfony®, 3 M ESPE Company) for the 2-unit bridge. Further clinical studies are needed to evaluate if such cantilever bridges can be used as a more permanent replacement for missing lateral incisors, compared with single tooth implants.

## Replacement of a canine

Because of the anatomy of the canines (massive crown and a long root), this tooth is rarely lost due to trauma (see Chapter 11). As for lost central and lateral incisors, the same 4 treatment solutions exist. In the following the esthetic considerations will be described for canine loss.

### Orthodontic closure

In these cases, a first premolar is moved mesially to replace the missing canine. Because of the shorter clinical crown length of the first premolar in comparison with the canine to be replaced (Fig. 36.3 M), it is advisable to intrude the premolar and build it up incisally with hybrid composite resin (Fig. 36.1 J) or a porcelain laminate veneer after treatment (Fig. 36.2E–H). For further details, see Zachrisson (55).

### Autotransplants

Mandibular premolars are very good candidates for transportation to replace maxillary canines, and due to their anatomy need little crown alteration. Necessary changes can be performed with either hybrid composite or porcelain laminates. Concerning the esthetic advantage of using transplants (see Chapter 33).

### Implants

The esthetic problem using implants in the canine region is the same as for lateral incisors. However, the atrophy of the labial bone with related bluish gingival discoloration due to the implant is possibly even more marked in the canine location, due to the thin gingiva and the frequent gingival

recessions often related to mechanical trauma (vigorous tooth brushing) (31, 32).

### Fixed prosthetics

The use of fixed prosthodontics to replace missing canines presents not only esthetic problems due to the common alveolar bone atrophy in the region (see Chapter 31), but also functional problems related to the canine location which imply a significant stress upon the tooth during articulation.

## Replacement after loss of multiple maxillary anterior teeth

Multiple losses of anterior teeth represent a significant increase in the number of esthetic problems. This is primarily due to the associated alveolar bone loss which may require both horizontal and vertical bone augmentation (see Chapter 31). Fortunately, in children the autotransplantation of developing premolars may induce alveolar bone growth and thus make it possible to restore the dental arch form using a combination of autotransplantation and orthodontic space closure after severe accidents with loss of several maxillary incisors. Figs 36.6 and 36.7 show cases where 3 lost anterior teeth (2 central incisors and 1 lateral incisor) were reduced to two by orthodontic treatment, and where the 2 missing central incisors were replaced with autotransplanted mandibular premolars that received bonded porcelain veneer crowns. At a follow-up at 14 years after treatment, satisfactory esthetic results are apparent. Despite considerable retraction of the marginal gingiva, there is no shadowing effect, and the exposed roots have a light and normal color.

In adults, a significant esthetic problem will occur when 2 implants are placed next to each other. In these instances, it is generally not possible to create a satisfactory gingival 31 between the two implants (33, 47), and an esthetic compromise has to be accepted (see Chapter 35). One treatment principle to reduce this problem may be to combine orthodontic space closure and a surgical solution (e.g. implants).

## Restoring fractured teeth

Replacement of the tooth structure lost due to trauma provides both a practical and an ethical problem. How much more tooth substance can justifiably be removed in order to optimally restore the anatomy of the injured tooth? By tradition, it has been accepted in dentistry that removal of large amounts of healthy tissue is acceptable to restore an injured tooth esthetically and functionally. The main reason for this acceptance has been that the strength of various types of available restorations until recently have depended on the strength of the restoration as such. Consequently, it was necessary to design the remaining tooth substance in such a way that it favored the strength of the restoration rather than the strength of the tooth.

The conventional jacket crown preparation is a typical example, where the concept is to optimize the strength and survival of the porcelain crown without giving consideration to the biological drawbacks. Consequently, even after a minor injury, a fractured tooth would end up with a subgingivally located, circumferentially prepared 1.2 mm shoulder and a jacket crown as the permanent restoration. Still, even with these maximally invasive preparations, the fracture rate of the jacket crowns remained high (54).

In the late 1950s, the metal-ceramic concept was introduced and allowed fabrication of a crown that was both strong and esthetic. The metal-ceramic crown replaced the jacket crown as the permanent restoration for fractured incisors. However, the invasiveness of the preparation was not reduced. Therefore, the introduction of acrylic materials and, later, composite resins that could be bonded to enamel was welcome, since they made removal of healthy tooth substance obsolete. Acid etching of enamel became the retentive tool for tooth colored restorative materials, allowing a truly minimally invasive restorative technique. However, the composite build-ups did not represent a permanent restoration (see Chapter 29). The initial composite resin materials were weak, prone to wear, and they discolored extensively with time.

In 1983, Calamia and Simonsen (56) produced the idea of bonding thin pieces of porcelain to the labial part of a tooth – thus the porcelain laminate veneer concept was invented. By etching enamel with phosphoric acid and the inside of the porcelain veneer with hydrofluoric acid gel, it became possible to get enough micromechanical retention for a diluted composite material to bond to both sides, thus producing a strong veneered tooth. Later, the diluted composite resin was replaced with light cured composite cements (57, 58). This idea was effective, since only minimal reduction of tooth substance was necessary, and the preparation could thus be kept in enamel. Intact enamel provides the most reliable substrate for etched porcelain veneer restorations (57, 58). The porcelain cervically was very thin, so the supragingivally placed finishing line became virtually invisible.

Today, the original concept has been altered in accordance with knowledge obtained during past years (57, 58), and the *Toreskog/Myrin concept* to be described below represents ultrathin porcelain laminate veneers/crowns based upon a minimally invasive procedure, in which proper bonding to existing enamel is a basis (Figs 36.11 and 36.12) (58). Unfortunately, in some areas of the world the preparations for bonded veneers/crowns are much too extensive, and are carried far into dentin (59–62). This will produce problems with retention of the restoration and potential pulp problems. Leading experts in the USA claim that preserving existing enamel as much as possible is a worthy goal (59–62), and therefore, it is wise to always consider orthodontic treatment prior to tooth preparation in cosmetic dentistry (61). Even minor movement and derotation of anterior teeth can have a profound impact on enamel preservation, and the longevity of the porcelain veneer

**Fig. 36.11** Supragingival laminate veneer preparations on central incisors after fracture of both incisal edges (A). The apparent opacity of the veneer during try-in disappears when a drop of water enters between the tooth and the veneer (D), making it blend with the colour of the tooth. It is important to use a translucent composite cement for this to occur. The veneers are bonded one by one under rubber dam. The dam should be applied so that contact points are exposed for perfect seating of the veneer (E). F shows the veneer just after bonding.

**Fig. 36.12** Veneer used to restore a fractured incisor. Patient with old unesthetic composite restoration on fractured upper right central (A). The supragingival shallow preparation, all in enamel (except for the exposed dentine at the fracture site) has been carried into the approximal areas (B). Note the fit and the emergence profile of the veneer as it is tried on the original, uncut mastermodel (C). The restored tooth at the one year follow up demonstrating a good blend between porcelain and tooth at the transition zone (D).

restorations (61). It is not acceptable treatment to casually prepare teeth in malocclusion for traditional crown restorations and refer to them as 'veneer restorations' with no regard whatsoever for enamel preservation (59–62).

## The Toreskog/Myrin concept

### Don't cut through enamel just out of habit

By staying in enamel we will secure a long-term predictable bond to etched enamel. Even though dentine bonding works and should be used as soon as any part of the dentine is exposed, dentine bonding is still more technique sensitive and not as predictable in a long-term perspective. Furthermore, by staying in enamel we minimize the risk of pulp irritation during the entire procedure.

### Place cervical finishing line supragingivally

This will increase the chance to stay in enamel which has been shown to be of a positive long-term advantage. There are contraindications for the supragingival preparation, e.g. when caries or existing fillings are subgingivally placed. Furthermore, whenever changes of the cervical anatomy are planned or when the background color is very dark and should be brightened, the gingival finishing line must be in the gingival area or sometimes even placed subgingivally, e.g. when a lateral has been placed in the central's position and should be restored to look like a central.

### Make a very shallow cervical preparation (0.2–0.4 mm in depth)

This will again decrease the risk of exposing dentine during preparation and thus increase the positive prognosis of the veneer/crown. It also gives the best possibility of having an invisible finishing line that does not have to be hidden subgingivally.

The invisibility of the cervical margin comes from the fact that fairly translucent porcelain and translucent cement is used in this area letting the background color of the tooth shine through. As soon as the porcelain becomes thicker it is impossible to use as much translucency since the porcelain then will appear gray.

### Let the preparation be carried past existing proximal fillings

The quality of the bond to existing old composite fillings is not as good as that to enamel and dentine and the long-term survival of the veneer/crown will be endangered when the finishing line is in a filling or when too much of the prepared tooth constitutes the prepared bonding surface.

### Let porcelain cover the incisal edge

This is important for two reasons: to optimize the strength of the incisal part of the crown and to secure a definite seating of the restoration during the cementation procedure. Normally the incisal edge should be shortened 1–1.5 mm during the preparation. However, if the teeth are going to be lengthened, which is a very common situation, no reduction of the incisal edge has to be done.

### Use rubber dam for maximum control and expose one preparation at a time plus the immediate contact point(s); cement one restoration at a time

The bonding and cementation procedure is technique sensitive and all efforts should be taken to optimize the circumstances during bonding. If a less than optimal bond is achieved the risk of fracture of the brittle restoration is critical.

### Use the all-etch technique with 35% phosphoric acid, dentin bonding and a translucent cement

It is virtually impossible to see if dentine is exposed so all surfaces have to be etched and subsequently dentine bonding has to be used all the time and on all surfaces.

## Essentials

Tooth loss requires a combination of esthetics and function. Methodology depends upon age, location and extent of traumatic injury.

### Replacement of a central incisor

- Orthodontic space closure.
- Premolar transplantation.
- Implant.
- Correctional or cantilever bridge work.

### Orthodontic implant site development

- Orthodontic extrusion

### Replacement of a lateral incisor

- Orthodontic closure.
- Premolar transplantation.
- Implant.
- Resin bonded cantilever prosthetics.

### Replacement of a canine

- Orthodontic closure.
- Autotransplants.
- Implants.
- Fixed prosthetics.

### Replacement after loss of multiple anterior teeth

- Combination of orthodontic closure and autotransplants.

## Restoring fractured teeth

- Porcelain laminate veneers using Toreskog/Myrin concept of preparation.
- Don't cut through enamel.
- Place cervical finishing line supragingivally.
- Make a very shallow cervical preparation.
- Let the preparation be carried past existing proximal fillings.
- Let porcelain cover the incisal edge.
- Use rubber dam, expose on preparation at a time plus the immediate contact point(s).
- Cement one restoration at a time.
- Use all-etch technique with 35% phosphoric acid, dentin bonding and a translucent cement.

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# 37

# Prevention of Dental and Oral Injuries

A. Sigurdsson & N. Cohenca

## Introduction

Trauma is still one the leading cause of morbidity and mortality with an average of 1.5 million injuries, 500 000 hospitalizations, 20 000 deaths and 120 000 disabled children each year (1). From this staggering numbers, 4.6–5.3% were maxillofacial and dental injuries identified among 277 008 pediatric trauma patient admissions (1, 2).

Traumatic dental and maxillofacial injuries are very common and appear worldwide to affect approximately 20–30% of permanent dentition and with often serious psychological, economic, functional and esthetic consequences for the individual (see Chapters 9, 10 and 11) (3, 4). With such a high frequency of injuries, prevention becomes a primary goal. This must take its origin in an identification of common causes for these injuries and whether such causes can be avoided or if preventive measures can be used to reduce the impact of such injuries on teeth.

A number of epidemiological studies of traumatic injuries have examined the etiology of injury (see Chapter 11). Overjet, incompetent lip closure and history of previous dental trauma are predisposing factors linked to dental trauma (5–7). Sgan-Cohen et al reported the odds of children with overjet of 6mm or more being almost 4 times more (8) (Fig. 37.1). Current literature found new predisposing factors including obesity and attention-deficit/hyperactivity disorder (ADHD) (9–11). Basha et al randomly selected 785 school children (13-year-old) and found that those with obesity and overweight had 2.78 times greater chance of suffering traumatic dental injuries (TDI) (9). Additionally, it has been shown in a respectable sample of over 200 adolescents with previous history of dental trauma that they had over two years observation time 4.8 times greater odds ratio for sustaining another dental injury compared with equal number of adolescents that did not have history of trauma and were age and gender matched (7). This number has even been reported higher in certain situations or close to 8.5 time higher (see later) (12).

Few studies have offered a detailed analysis regarding preventive measures and its relation to the cause of injuries (13–15). In Fig. 37.2 a detailed analysis is shown for etiology of traumatic dental injuries. The various causes of injuries are related according to the possibility of using preventive measures to reduce the consequences of such injuries. It also appears that sports injuries for those older than 7 years of age represent about 20% of the trauma population (red columns) and it could be speculated that this is the most obvious activity where preventive measures would be possible and effective in reducing the rate of oral trauma. Traffic accidents appear to be the next frequent cause (blue columns) and also call for preventive measures to prevent or reduce the consequences of these usually severe impacts.

A significant number of oral and dental injuries result from participation in contact sports such as American football, basketball, rugby, soccer, boxing, wrestling or 'stick sports' (Fig. 37.3). Cohenca et al published a 10-year retrospective study of injuries among college athletes. Only injuries that were severe enough under the criteria established by the National Collegiate Athletic Association (NCAA) Injury Surveillance System (ISS) were reported in the study. Their result indicated five times more incidence rate among basketball than football players (16). Malanga and Chimes reported the incidence of dental injuries among basketball athletes to be up to 36.4%, which was 13 times higher than in American football (17). Another study looked recently at "weekend warrior soccer players" where older group of public employees from 78 public institutions, including ministry offices, directorates-general, universities, and government offices in Turkey (18). The study revealed few alarming statistics; 9.8% of nearly 1000 participants had experienced orofacial trauma, but only 19.6% were aware of

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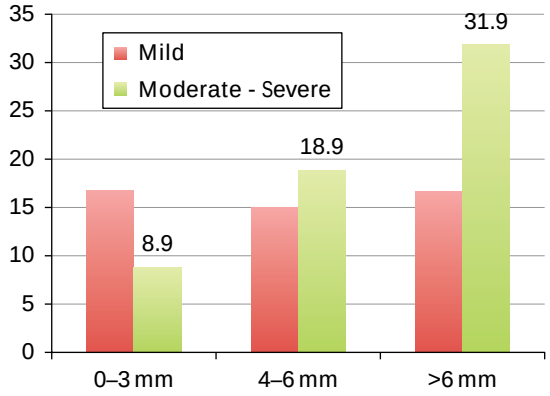



**Fig. 37.1** Trauma prevalence in relation to overjet. Source: Reproduced from (8) with permission.

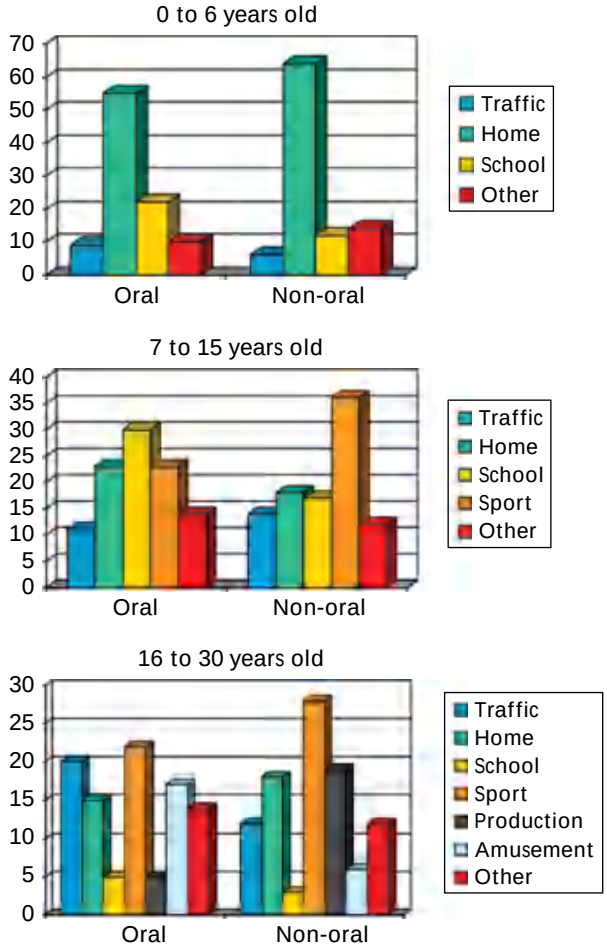



**Fig. 37.2** Injury environment for oral and non-oral injuries at various ages. An epidemiologic study on injuries during 1 year in a Swedish county. Source: Reproduced from (13) with permission.

emergency treatment for dental trauma indicating that education is sorely lacking (see later) (18).

It is though important to note that oral and dental injuries occur as much if not more often during children's play or leisure activities (13, 14) (see Chapter 11). For example, in a study by Skaare and Jacobsen from Norway, in 2003 (14) nearly half (48%) of the 1,275 injured individuals that were reported on in the study were injured at school even though many of those injuries could have been classified as "sport" activity they occurred during playground play than organized sports activities.

Similar numbers were reported by Andreasen in 2001 from a sample of 3,655 dental casualty insurance claims from a major Danish insurance company (15). In this, 7% of the dental trauma claims were due to organized sports, while 93% were from various, unpreventable causes. In both studies, the authors felt that it is neither easy to prevent dental injuries nor to make guidelines on prevention. Based on this it should be clear that promoting mouthguards and facial masks is not the only preventive measure that needs to be taken.

A closer look at where and why accidents occurred, particularly in 10–12 year-old children, the patterns are distinctive (Fig. 37.4) (8). Traumatic injuries within school were equally distributed between falls, sport and violence. At home most accidents are due to falls and sharp objects. Falls, sports, bicycle and games characterized accidents at playgrounds, street and parks.

The best approach to prevent dental and oral injuries lays on education, early orthodontics in children with severe overjet, and protection. Education shall focus on how to avoid injuries as well as how to manage them at the site of the injury when they occur. Accordingly, the education should include children, teenagers, as well as those individuals who are present at sites of injuries, e.g. parents, school officials, coaches, trainers, paramedics, nurses and youth leaders. It should be the aim of every dentist to discuss risk factors that could lead to dental or oral injury during routine dental visits (see also Chapter 42). It is important to remember that this education should be aimed at both sexes equally. Even though some of the older epidemiological data indicated that boys were more prone to sustain oral and dental injury, with increased participation of girls in the more traditionally male sports, e.g. basketball and soccer, as well as in leisure activities, e.g. roller-blading and skate-boarding, that gap seems to be narrowing, at least in some areas (19).

Special emphasis should be placed on those who are at high risk, such as those with severe maxillary overjet (20, 21). It has even been suggested that a preventive orthodontic treatment should be initiated for these individuals and completed before the age of 11, i.e. in the early to middle mixed dentition in an attempt to reduce their risk of trauma (22).

Another group that should receive special counseling are those who have already sustained an oral injury, for those seem to be much more likely to sustain another injury than those who have not (7, 12, 23). It is of interest that the risk of sustaining multiple injuries has been reported as 8.4 times higher when the first trauma episode occurred at 9 years of age, compared with those occurring at age 12 (12). Therefore, young children should receive special attention. Their activities, games and sporting involvement should be carefully assessed; and any risk behavior should be discussed with them as well as with their parents or those who care for them. A typical example is the recent popularity of basketball

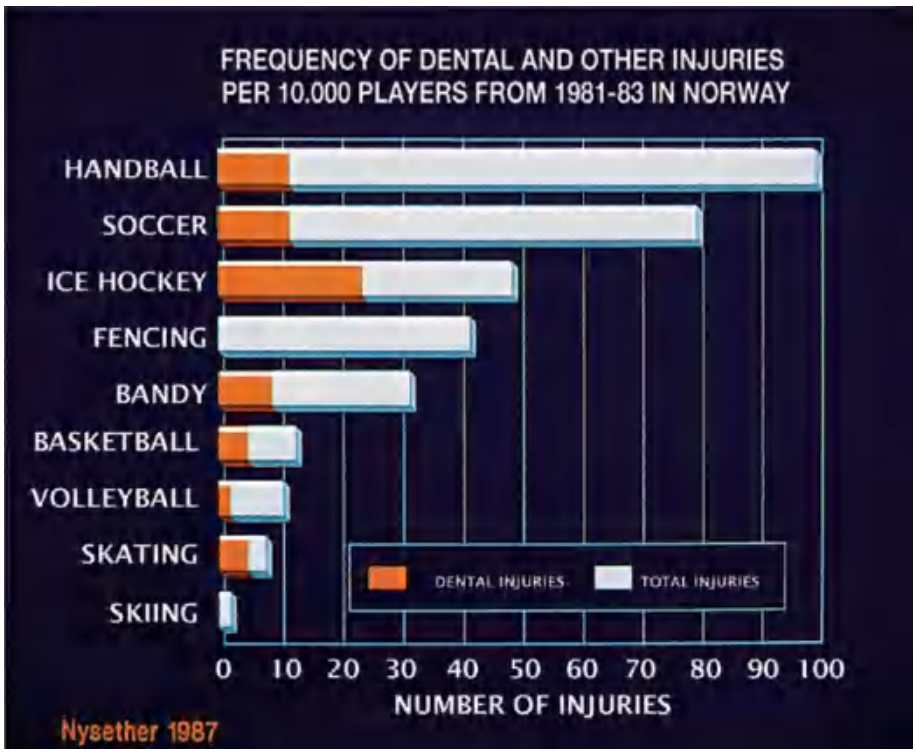



**Fig. 37.3** Dental injuries related to various sports based on insurance records which are precise documentation on dental and other injuri8es in Norway. Source: Reproduced from (79) with permission.

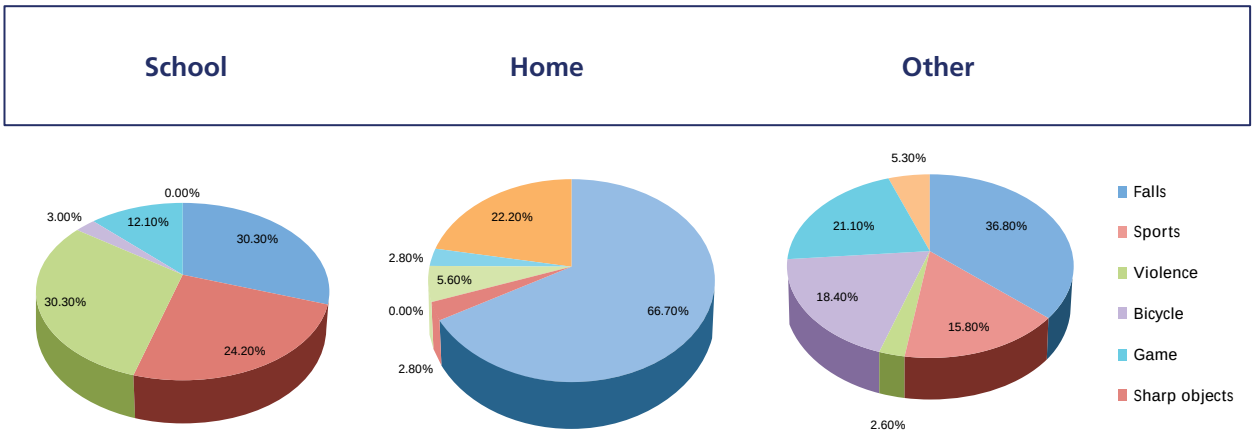



**Fig. 37.4** Cause of dental trauma injuries related to place where it occurs. Source: Reproduced from (13) with permission.

hoops that can be lowered from standard height, so that 10- to 12-year-olds can 'slam-dunk' or place the ball in the net by jumping up and hanging on the net ring that holds the net after letting go of the ball. Because their arms are short at that age, their maxillary incisors are often at the level with the basket net. This scenario has now been reported to result in an increase in severe dental trauma, where multiple teeth are frequently avulsed (24).

All children should also be made aware of correct first aid when an injury does occur. Things like replanting avulsed teeth immediately if at all possible or alternatively storing them in milk or saliva, looking for all fragments of a broken tooth crown before running home or after help, should be discussed with them in clear, simple language. Posters like the one that the International Association of Dental Traumatology (IADT) and others have sponsored (Fig. 37.5) are likely to gain the attention of young individuals; and if widely displayed they should also reinforce their knowledge (see also Chapter 42).

Not only should young ones be educated about dental trauma but also those who care for them, especially those who are responsible for their safety during school and organized activities. A simple instruction sheet, like that in Table 37.1, is in many cases more than sufficient to ensure that those who are injured receive proper emergency care at the site of the injury.

When educating non-dentally trained persons it is important to remember to avoid complicated language. Table 37.1 shows a suggested communication form that is based on the recommendations of the IADT. Its language is clear; and it explains simply what to do and not to do immediately, as well as how urgent dental consultation is.

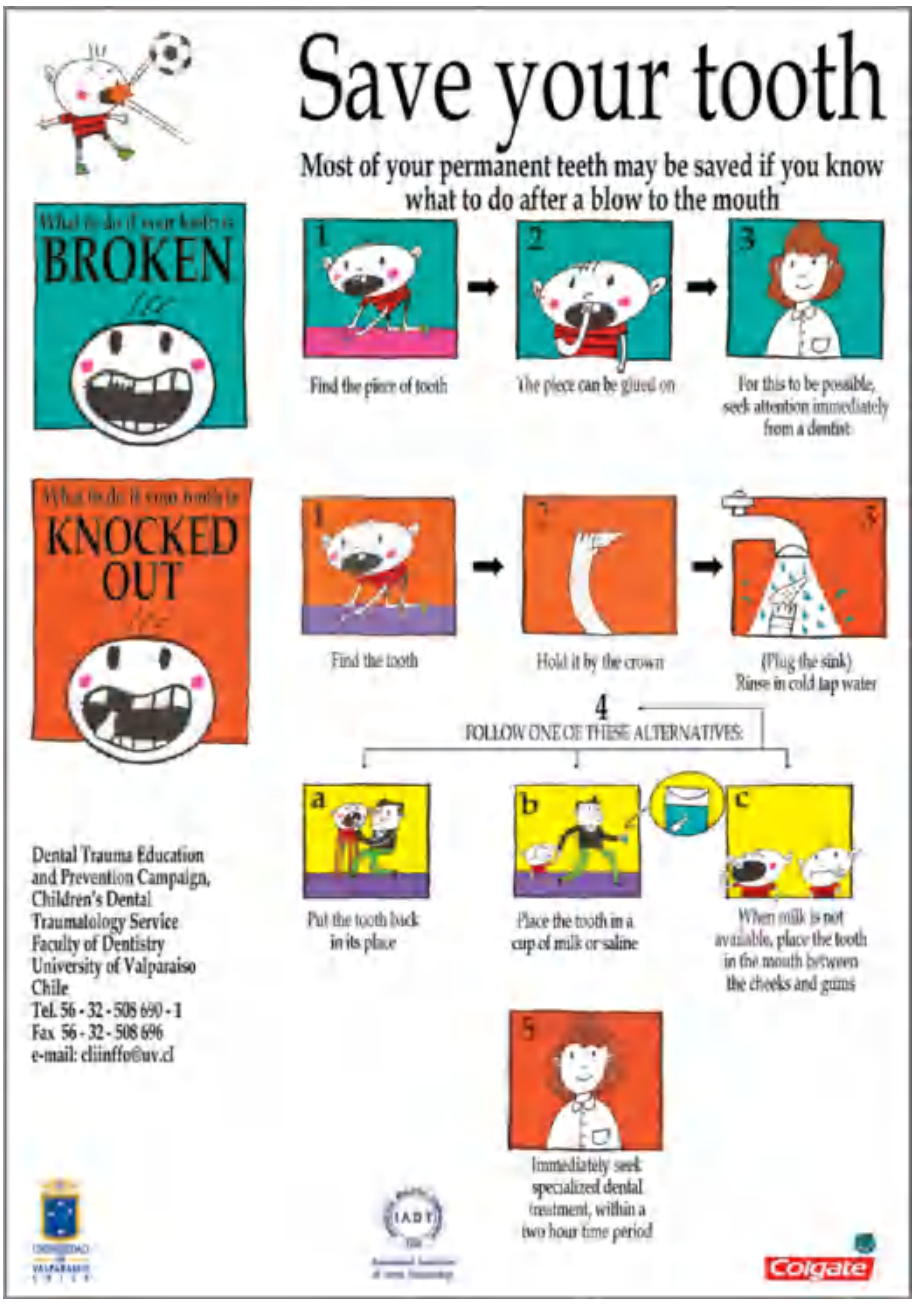



**Fig. 37.5** Poster co-sponsored by several national dental organizations and the International Association of Dental Traumatology.

## Means to prevent dental maxillofacial injuries

The nature of a trauma implies acute delivery energy released upon soft and hard tissues and resulting in laceration, contusion or ablation of tissue. A protective device to reduce the consequences of such traumas can be devices that prevent the impact from getting in contact with the oral region or a cushioning device that absorbs and/or distributes the impact forces (mouthguards) (Fig. 37.6). Finally, restraining devices such as safety belts may absorb a significant part of the energy from the impact and thereby reduce the chance of the impact becoming delivered to the oral and maxillofacial region.

Not appearing in Fig. 37.2 is traumatic dental injuries occurring during oral endoscope and oro-tracheal intubations. By using preventive measures these injuries can be significantly reduced. In the following a description will be given of possible ways to prevent dental and maxillofacial injuries in relation to various sports and traffic activities.

## Appliances to prevent dental injuries

During sports and other activities, where there is a risk of falling or being hit by an object, wearing a faceguard and/or mouthguard still seems to be the only way to prevent or at

**Table 37.1** Suggested review chart for athletic trainers or those who are responsible for children and teenagers in play.

| Term | Type of injury | Immediate treatment | Refer to a dentist |
|---|---|---|---|
| Uncomplicated crown fracture | A portion of the tooth broken, no bleeding from the fracture | None | Refer as soon as possible, within 48 hours, especially if the tooth is sensitive to cold air |
| Complicated crown fracture | A portion of the tooth broken and there is bleeding from the fracture | None, do not place any medication on the bleeding pulp, stop the bleeding with moist sterile cotton gauge | As soon as logistically possible within 24 to 48 hrs |
| Root fracture | Tooth might appear in normal position but bleeding from the gum around the tooth. Alternatively, the crown of the tooth might be pushed back or loose | None or reposition the tooth in its normal location, but only if it moves easily | As soon as possible |
| Concussion and subluxation | Tooth still in its normal place and firm or slightly loose | None | As soon as possible, within 48 hours, usually for evaluation only |
| Luxation | Tooth very loose and/or has moved from its normal position | None or reposition the tooth in its normal location, but only if it moves easily | As soon as possible, especially if it is not possible to reposition the tooth to its normal position |
| Intrusion | The tooth is pushed into the gum/jaw and is firm or slightly loose | None | As soon as possible |
| Avulsion | Tooth completely out of the mouth | Replace the tooth in its hole. If it is contaminated by surface debris, first rinse the tooth for a few seconds under running water. Do not scrape or wipe it clean. If it is not possible then store the tooth in milk, saliva or saline | Immediately; it is extremely important for prognosis of the tooth to be treated immediately |

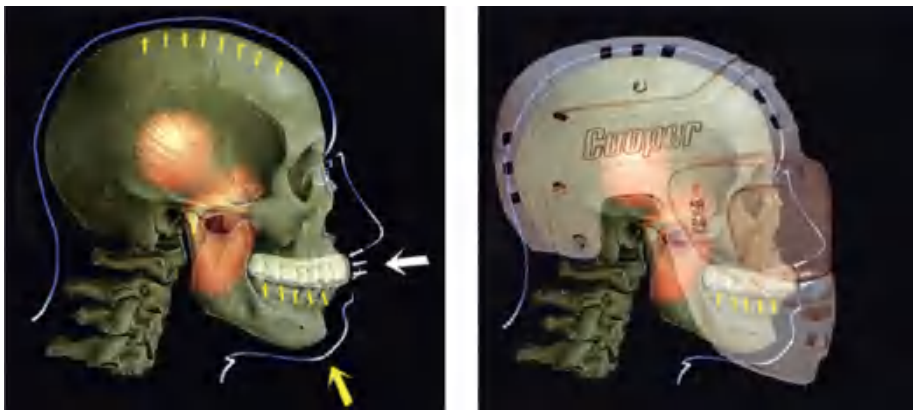



**Fig. 37.6** Mechanisms of mouthguard and facemask protection. The protection varies depending on the energy and direction of the impact. The full facemask protects the face and teeth from direct hits and the mouthguard protects the teeth from frontal impacts by distributing the forces over a greater area and from an impact on the base of the mandible by the cushioning effect between the maxilla and mandible.

least significantly reduce the seriousness of dental injuries. It has been reported that prior to the mandate of wearing face- and mouthguards in US high school football, facial and oral injuries constituted up to 50% of all reported football injuries (25, 26). Subsequent to that mandate a significant decrease was noted in reported injuries, down to a small percentage (25, 27).

Another common mechanism of oral injury is traffic accidents. Wearing seatbelts in a car, and when riding a motorcycle using a helmet with a chin arch, is in many places mandatory. The most commonly used bicycling helmets do provide very good protection against head injuries as shown in Victoria, Australia, when one year after mandatory helmet protection was introduced there was a 48% reduction of reported head injuries for bicyclists (28). Unfortunately, these same helmets do not offer mouth or dental protection.

## Faceguards

Faceguards are usually a prefabricated cage of metal or composite that is attached to a helmet or a head strap (Fig. 37.7). More recently guards made out of clear polycarbonate plastic have become available, either as prefabricated or custom-made. It would appear that these faceguards provide good protection to the face and the teeth, but are not applicable to all activities; and in many cases, do not protect the teeth if the individual is hit under the chin.

A B C

**Fig. 37.7** A. A typical cage-like face-mask as work by American football players. It provided good protection to the face and mouth but even with this extended type there is a minimal protection from a blow under the chin (note the athlete is not wearing the mandatory chin strap for illustrations purposes). B Full mace masks are used in the junior level of ice hockey and they have been found effective in protecting he players form orofacial injuries C. With a face-mask, an injury like this could easily be avoided.

Very few large-scale studies have been conducted on the actual benefits of wearing faceguards in games or practice; but it is clear that the introduction of mandatory helmet and facial protection has been effective in virtually eliminating ocular, facial and dental injuries in juvenile hockey (29). However, an unforeseen problem has been reported for the same groups of youths: while the number of head injuries has been reduced, an increase in catastrophic spinal injuries has been noted. It has been speculated that players get a false sense of security when donning the equipment, leading them to take excessive and unwarranted risks due to the protection they are supposedly afforded (29).

One of the few intervention studies on the effectiveness of faceguards was performed by Danis et al. (30) on a group of youth league baseball teams in the USA. Approximately one-half of the teams were supplied with guard helmets (intervention); all others used this protection at their discretion (comparison). They found that the intervention teams reported a significant reduction in the incidence of oculofacial injuries compared to comparison team respondents ($p = 0.04$).

Half facemasks have been popular in hockey, as it has been speculated that a full face shield may increase the risk of concussions and neck injuries, offsetting the benefits of protection from dental, facial and ocular injuries. This does not seem to be the case as a study found that the use of full face shields is associated with a significantly reduced risk of sustaining facial and dental injuries without an increase in the risk of neck injuries, concussions or other injuries (31).

With the emergence of many new fiber-composites, custom fabricated faceguards will become more available and affordable. Currently only few high-level college (US NCAA Division 1) athletes do wear faceguards, and most of those only during recovery time after having sustained a facial injury (32). The majority of those are wearing a custom-made ones but the programs cite disadvantages like high cost and slow manufacture turnaround time for lack of more widespread use. The study concludes that only a handful of over the counter face masks exist for U.S. consumers, but none of them have been reviewed or classified by product design, sport application, price, and collegiate consumer use (32). These studies confirming their benefits must be conducted.

## Mouthguards

Mouthguards have existed for well over a century. According to Reed (33) they were originally designed and fabricated in the late 19th century by a London dentist, Woolf Krause. They were initially made from gutta-percha and their main purpose was to protect boxers from lip lacerations and other soft tissue injuries. Philip Krause, son of W. Krause, a dentist as well as an amateur boxer, further developed the 'gum shield' to something that approaches what is known today. He used vella rubber rather than the relatively hard gutta-percha to create his mouthguards (33, 34).

The use of mouthguards in contact sports has been reported in the past to reduce the occurrence of dental injuries to over 90% (35–38). Since rules regarding the use of headgear and mouthguards in high school football were established in the 1960s, facial and dental injuries sustained on the field have dropped by approximately 48% (27).

Few studies have specifically investigated, prospectively or in real time, whether athletes who wear mouthguards sustain significantly fewer dental injuries than those who do not (39, 40). The first study to do so involved a sample population of 272 high school rugby players. The athletes received a pre-season clinical examination by a team of dentists and completed a questionnaire. Mouthguards were fitted one week later, and the players were instructed in their use. At the end of the season, a follow-up questionnaire was completed. There was a significant difference in the number of tooth fractures between mouthguard wearers and non-mouthguard wearers (39). The second study, which evaluated US male college basketball players (age 18–22), collected 'real time' data (40). Trainers reported information about their teams on a weekly basis using an interactive website. The results of this study are likely to be significant, as it captured 70 936 athlete exposures (an athletic exposure is a one athlete participating in a game or practice, whether it was

**Table 37.2** Dental injury rate difference between mouthguard wearers and non-wearers for male college basketball players in a large real-time study.

| Injury rate per 100 players | Brain concussion | Mouth injuries | Dental injuries (fract/lux/avuls) | Dental referrals |
|---|---|---|---|---|
| Mouthguard | 6.2% | 13.0% | 0.6% | 1.6% |
| No mouthguard | 6.1% | 14.2% | 2.0% | 5.8% |
| | NS | NS | $P<0.03$ | $P<0.02$ |

(fract/lux/avuls) = fractures, luxations, avulsions.
Source: From LaBELLA et al. 2002 (40).

**Table 37.3** Athletic exposures and brain concussions reported by 87 USA NCAA Division I college football teams for the 2001 season.

| | Total athletic exposures (contact practices and games; additional 168 800 exposures in non-contact practices excluded) | Brain concussions |
|---|---|---|
| Custom mouthguard | 141 165 | 166 (0.1%) |
| Boil-and-bite mouthguard | 196 332 | 199 (0.1%) |
| | | NS |

Source: From LaBELLA et al. 2002 (40).

one play, one quarter, one half or the entire game). The study found that mouthguard users had significantly lower rates of dental injuries and dentist referrals than non-users. However, there was no significant difference between mouthguard users and non-users in the rate of soft tissue injuries. Of note, this study reported significantly more oral/dental injuries than that reported by the NCAA for the same season (40) (Tables 37.2 and 37.3).

Not all studies have demonstrated a beneficial effect of mouthguards. In a cross-sectional study, a sample of 321 university rugby players participating on 555 player occasions was examined (41). The results of that study indicated no statistically significant association between oral, dental and lip injuries sustained during rugby playing with the use or non-use of mouthguards. This study, like most mouthguard studies, irrespective of whether they demonstrated a beneficial effect or not, is relatively small and therefore it is likely that it did not have enough statistical power. More large-scale studies are therefore needed.

There are still many questions unanswered regarding to mouthguards and how they are best constructed to afford the best protection. Recently a study investigated the use and quality of mouthguards used in Swiss boxing (42). Of the 215 mouthguards examined, 193 (89.8%) were insufficient yet despite the observed deficiencies, only a few dental injuries occurred during boxing (42).

## *Suggested function of mouthguards*

The four main suggested functions of a mouthguard are (43):

(1) Preventing tooth injuries by absorbing and deflecting blows to the teeth
(2) Shielding the lips, tongue and gingival tissues from laceration
(3) Preventing opposing teeth from coming into violent contact
(4) Providing the mandible with resilient support, which absorbs an impact that might fracture the unsupported angle or condyle of the mandible.

Various materials and methods have been used to achieve these basic functions, as many test protocols and devices tried to investigate them and confirm the effectiveness. The main problem with testing these suggested functions are that there is no *in vivo* model ethically possible and *in vitro* models are at best crude approximations. To further complicate matters, large prospective cohort studies are very difficult to conduct, and to some degree are unethical as well, because of the need for crossover or control groups. One very interesting attempt to circumvent some of these problems was a study by Johnston and Messer (44), where they used mandibular sheep segments with incisors at four developmental stages. Customized pressure formed mouthguards were made for each mandibular segment and then trauma was inflicted with servo-hydraulic materials testing machine. Injuries were examined clinically, radiographically and by dissection. The magnitude of lateral luxation measurements of individual teeth was reduced significantly by mouthguard protection in both deciduous and permanent dentitions. Fourteen- to 24-fold greater forces were required to cause an injury in the sheep mandibular dentition covered with a custom-made mouthguard compared with dentition without mouthguard coverage. Of interest was their finding that the mouthguard tended to increase the mobility of the teeth it encompassed and, in some instances, promoted dentoalveolar injury of adjacent teeth.

Other studies have used the upper central incisors in a dry human skull that were excited by an electrodynamic shaker (45), or a simulated maxilla, constructed of polymethylmethacrylate, containing replaceable resin teeth where a steel ram was dropped on the maxillary incisor region. Changes in voltage, which were induced by a strain gauge at the back of the upper left incisor, were the measure of protection (46), or measuring the cushioning effect of the material directly, as by deflection caused by impact forces induced by a pendulum ram impact testing machine (47). The main concern with all these approaches it that it is not clear if they translate into true protection of the dentition and alveolus in humans.

Japanese group attempted to investigate real objects (steel balls, baseball, softball, ice hockey puck, cricket ball, wooden baseball bat) that could hit mouthguard and teeth during an athletic event rather than just a steel drop-ball that has been frequently used in studies on mouthguards (48). Their studies indicated that transmitted forces were less when a standard single layer mouthguard was used compared to no mouthguard, but as importantly they demonstrated that the object type that hit the teeth significantly influenced the effect. The steel ball showed the biggest (62.1%) absorption ability while the wooden ball showed the second biggest (38.3%). Other objects, baseball, field hockey ball and hockey puck, showed 0.6–6.0% absorbency. These results show that it is important to test the effectiveness of mouthguards on specific types of sports equipment rather than using some standard experimental equipment that might not give a realistic outcome. This group also investigated different sensors in a follow-up paper (49) and found that the sensor plays an important role in the measurement values reported for absorbency of mouthguard materials; they concluded that a standard sensor should be used for all experiments. It is therefore clear that further studies are warranted in standardizing this kind of test so that effectiveness of mouthguards can be compared with confidence.

How the mouthguard is able to achieve these suggested protective functions is not quite clear. Many studies have placed an emphasis on investigating the energy absorption of different materials with the idea in mind that the higher the absorption, the better protection the mouthguard provides. It has been shown that the physical and mechanical properties vary with their chemical composition and this itself varies with different brands of the same material (see later in this chapter). The resilience of PVAc-PE materials appears to vary inversely with the magnitude of the impact energy at which they are tested. A number of factors may be responsible for this variation including the degree of crosslinking between polymer chains, the proportion of plasticizer present and the volume of filler particles. It has been suggested that laminated thermoplastic mouthguards are dimensionally more stable in use (50). Again, the clinical significance of these variations has not been determined; but it has been suggested that high energy absorption does not necessarily indicate that the material will give maximum protection, since some of the absorbed energy may be transmitted directly to the underlying dental structures (51). Research elucidating if this is the case is very much needed.

A fifth suggested function of the mouthguard has been that it prevents neck and cerebral brain injuries. Through the years there has been a great deal of discussion in the literature that the mouthguard prevents cerebral concussion in athletes (52–54). However, most of these papers have only been case reports or opinions, not based on controlled scientific studies. The two papers usually cited as a foundation of this presumed effect were written by Stenger et al. in 1964 (55) and Hickey et al. in 1967 (56).

The first article reported on only five football players who had a history of head and/or neck injuries. Three players gave a history of 'being concussion prone' and one had previous neck injuries. The fifth athlete experienced definite pain with crepitation on the left side when the temporomandibular joint was palpitated and cervical pain extending halfway down his shoulder. Each athlete wore a custom fitted mouthguard when playing football in conjunction with daily inter-maxillary acrylic splint therapy. Each player's symptoms were either eliminated or diminished. The author concluded that the custom-fitted mouthguard provided protection and relief for patients.

The second paper was a report on an *in vitro* study, where series of impact blows were applied to a single cadaver using an intracranial pressure transducer to measure pressure changes associated with the blows. The authors of that study concluded that 'there was a decided reduction in the amplitude of the intracranial pressure wave when the mouth protector was in place. Bone deformation was also decreased moderately when the mouth protectors were in place (56). However, the correlation between those factors and cerebral concussion is not at all clear (57). Recently, with more careful scrutiny of scientific evidence in the literature, this claim of protectiveness of the mouthguard has been called into question (34).

Two very large studies using interactive websites to collect weekly information on the incidence of cerebral brain concussion in athletes have failed to show any benefits (40, 58) (Tables 37.2 and 37.3).

In the former, a comparison was made between those who wore a mouthguard and those who did not in US college men's basketball. Almost 71 000 athletic exposures were reported; there was no significant difference in the rate of brain concussion between the groups (40). The second study compared 'boil and bite' versus custom-made mouthguards worn by American college football players (58). Over 500 000 athletic exposures were recorded; again, there was no significant difference in trauma risk between the two groups (40, 58). It is important to note that these two studies did not assess the mechanism of injury, for example blow under the chin versus fall to the ground. However, and unfortunately, these two studies do not indicate that wearing a mouthguard helps reduce cerebral brain concussion in any significant way.

### *Types of mouthguard*

Mouthguards can be divided in to three basic types based on how they are manufactured and used:

- Stock – prefabricated
- Mouth formed
- Custom-made.

Some of these basic types have now several sub-groups, especially the custom-made ones.

*Stock* mouthguards may be made either from rubber or one of the plastic materials (Fig. 37.8). They are generally available in two or three sizes, and are supposed to have a universal fit, sometimes aided by flanges in the molar area. Modification is limited to trimming the margins to relieve the frenula. The loose fit means that the wearer must occlude

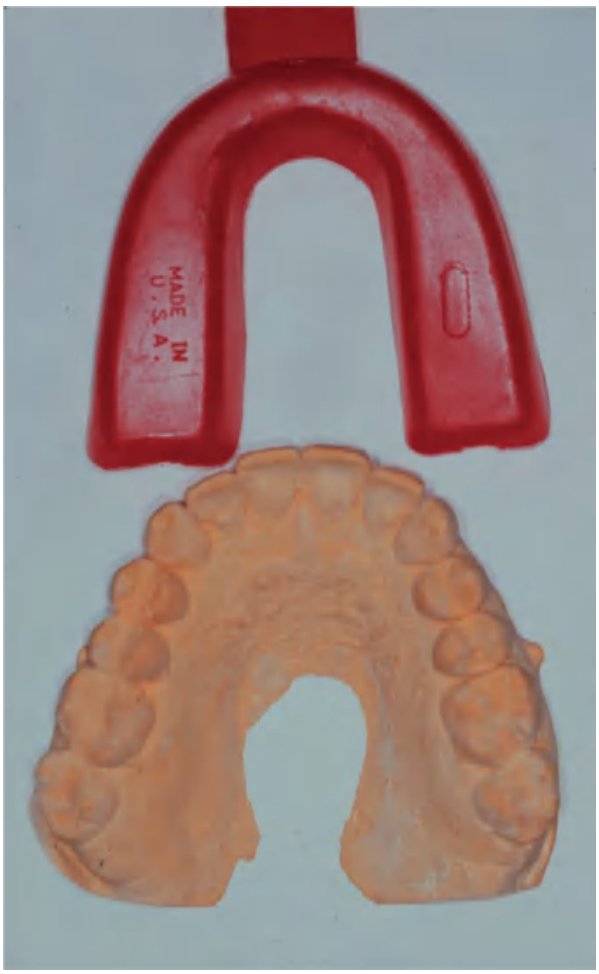



**Fig. 37.8** Stock mouthguard that is too small for the arch and therefore will not stay easily in place as it is not comfortable.

to prevent the guard from being displaced. The main advantage of this type of mouthguard is that they are inexpensive and may be purchased by the public in sports shops. Also, because they do not require any preparation, a replacement is readily available at any time. Most reports agree (59, 60) that these types of mouthguard provide the least protection of all available types due to their poor fit, though admittedly there is no conclusive scientific data confirming this opinion. However, it is unquestioned that these mouthguards are uncomfortable for the wearers: they tend to obstruct speech and breathing because the wearer must keep them in place by either clenching or supporting with the tongue (61) and therefore are less likely to be worn, and when needed could be blown out of the mouth prior to impact with the ground or other obstacles.

There are two types of *mouth-formed mouthguards*. The first consists of a hard and fairly rigid outer shell that provides a smooth, durable surface and a soft, resilient lining that is adapted to the teeth (Fig. 37.9). The outer shell of vinyl chloride may be lined with a layer of self-curing methyl-methacrylate or silicone rubber. The outer shell is fitted and trimmed, if necessary, around the sulci and frenal attachments. It is filled with the soft lining and seated in the mouth. Care must be taken to ensure that it is centrally placed. The lining is allowed to polymerize for 3–5 minutes. Excess material is trimmed with a sharp knife and the margins smoothed with dental stones. This type of mouthguard tends to be bulky and the margins of the outer shell may be sharp unless protected by an adequate thickness of the lining material. The most commonly used type of mouth-formed protector is constructed from a preformed thermoplastic shell of PVAc-PE copolymer or PVC (59) that is softened in warm water and then molded in the mouth by the user (Fig. 37.9). These mouthguards have several distinct advantages over the stock mouthguard. If carefully adapted, they give a closer fit and are more easily retained than stock protectors. Care must be taken during the molding process so that the mouthguard fits accurately. The temperature necessary to allow adequate adaptation to the teeth is fairly high, so additional care must be taken to prevent burning the gingiva. Similar to the stock mouthguard, this type of mouthguard is relatively inexpensive, readily available to the general public and can be formed into a decent appliance with some care.

*Custom mouthguards* are individually made in a laboratory, on plaster of Paris models poured from impressions of

A B

**Fig. 37.9** Boil-and-bite being formed in the mouth. A. The stock mouthguard is heated in boiling water for a few minutes and then inserted in to the mouth with care. B. The index fingers should be used to adapt the mouthguard as well as possible to the facial sides of the molars and the thumbs at the same time press on the palatal side.

the player's mouth. Many studies have shown that these mouthguards are more acceptable and comfortable to athletes than the other types (62, 63). There is no evidence, however, that custom-made protectors are more effective in preventing injuries.

Historically, three groups of materials were used to fabricate custom-made mouth protectors: *molded velum rubber* (62), *latex rubber* (64) and *resilient acrylic resins*. Currently by far the most common material used for custom-made mouthguards is ethylene vinyl acetate (EVA) copolymers. Previously polyvinylchloride, soft acrylic resins and polyurethane were also used; but the superiority of the EVA copolymers has practically eliminated the others. Its popularity is mainly due to its elastomeric softness and flexibility so that it can be relatively easily processed. Also, the material has good clarity and gloss, barrier properties, low-temperature toughness, stress-crack resistance and little or no odor (59, 65). Many prefabricated plates of EVA are now commercially available; but it is important to know that the percentage of vinyl acetate (often indicated as a percentage) can vary between manufactures and therefore show differences in properties. The more vinyl acetate is copolymerised (higher % marking), the more flexible, stretchable, softer and tougher is the material (65). This property will also lower the softening temperature, allowing manipulation of the material within a comfortable temperature. The most common material for mouthguards contains 28% vinyl acetate. It should be remembered that the actual performance of a mouthguard is just as dependent upon the design, thickness and manner of impact on the mouth as the base material used (65).

The EVA mouthguard material can now be bought in varying color, thickness and hardness. There has been some discussion about whether there should be different stiffness or hardness suggested for different sports. Thus, while the low-stiffness guards absorb shock during hard-object collisions (e.g. baseballs), they may not protect the tooth-bone during soft-object collisions (e.g. boxing gloves) (66). To date, clinical studies are lacking to substantiate such recommendations.

In 1985 Chaconas et al. (50) described a laminated thermoplastic mouthguard that showed significantly less dimensional change than other materials tested (i.e. poly vinyl acetate-ethylene) copolymer clear thermoplastic and polyurethane). This was the start of several different subcategories within custom fabricated mouthguards.

A few years ago, layered EVA stock plates were introduced (Fig. 37.8). The idea was to further strengthen the mouthguard without losing its protective capacity. When a stock plate of EVA is fabricated, it is drawn out in one direction so that the polymer chains are more or less parallel, like the grain in wood. This can theoretically make a difference in properties whether the 'grains' are running facio-palatally or mesio-distally on the crown of the tooth. To eliminate this and increase the stiffness without adding bulk, manufacturers have started to market 2- or 3-layered stock EVA, where the layers have been added perpendicular to each other. Some manufactures have even added a low percentage EVA plate between layers, which is designed to further stiffen the mouthguard palatally behind the anterior incisors (Figs 37.10 and 37.11). There is not much, if any, scientific proof that this can increase the protectiveness of the mouthguard and again is a question of whether a too stiff mouthguard could not cause other damage to the tooth or alveolus. Some disturbing findings were reported in a recent study (67). It was found that a hard insert resulted in reduced energy absorption when compared with a control sheet of the same material and approximate thickness but without the hard inserts (67). The same research group has, however, shown improved impact characteristics of the EVA mouthguard material with regulated air inclusions (68). But as yet, there is no clinical data available to support this, and durability of air-included mouthguards is unknown.

Another version of this layered concept is the fusion of two plates of different stiffness. It is important to know that these only seem to improve the mouthguard when the softer material is next to the teeth. A study, using a finite element model, by Kim and Mathieu (51) showed that a soft outer layer covering a hard core had no significant difference from a monolayer in stress distribution and impact force. However,

**Fig. 37.10** Prefabricated double layered mouthguard stock plate. Both layers are of the same vinyl acetate percentage but the top layer is clear and the bottom composed of three different colors, for esthetic reasons, all fused into one mass.

**Fig. 37.11** Mouthguard made of prefabricated double-layered stock plate with a further added low percentage EVA plate between layers that is designed to stiffen the mouthguard on the palatal site of the anterior incisors. When making a mouthguard with these plates care must be taken to align the insert with the center and on the palatal site of the teeth.

a soft core was found to have a significant effect on stress distribution. Controlling ratios of modulus and volume fractions of the core and outer layer could increase this effect.

The main question regarding these various types of mouthguard is whether there is any actual protective difference between them. Very few studies have investigated the efficacy of the different types in preventing dental injuries with large enough samples to have significant statistical power.

In one study on 98 professional rugby players, custom-made mouthguards did not significantly reduce the amount of dental injuries sustained as compared to mouth-formed mouthguards (69). At a follow-up clinical examination, there was no damage to teeth when either custom-made or boil-and-bite mouthguards were worn.

Stokes et al. compared mouth-formed and custom-made mouthguards (62). They showed that although there were no dental injuries in either group, the users preferred laboratory-formed mouthguards because of comfort.

In a very large study on college football players, where their trainers reported every week for the entire season using an interactive website the number of players, mouthguard use and dental and oral injuries, there was no apparent difference between boil-and-bite or custom-made mouthguards (70). The sample consisted of 87 (76%) out of a possible 114 Division I teams, with a total of 506 297 athletic exposures recorded (Table 37.4).

Most of those teams had a mixed use of custom and boil-and-bite mouthguards and thereby there was a possibility that these teams would obscure the benefits of one particular mouthguard. Therefore, the data was further analyzed where a sub-selection of fourteen teams was selected. Seven of these used exclusively custom-made mouthguards and the other seven exclusively boil-and-bite. The results were the same: no statistically significant benefits of type of one mouthguard over another (70). It is important to emphasize that in this study there was no attempt to inspect the quality of the mouthguard used or the comfort of one type over the other.

Wear and tear will affect all mouthguards; it has been suggested that they should be replaced regularly, not only due to lack of fit, but also because of reduction in protective properties. Recently it has been shown in a simulated aging study on different types of custom-made mouthguards, that aging induced various dimensional changes. Most of the dimensional change for all mouthguards occurred at the central incisor region. Pressure-laminated mouthguard specimens showed the lowest range of changes at the central incisor region, suggesting potentially improved fit, comfort and protection (71).

**Table 37.4** Athletic exposures and dental injuries reported by 87 USA NCAA Division I college football teams for the 2001 season.

| | Total athletic exposures (contact practices and games; additional 168 800 exposures in non-contact practices excluded) | Dental injuries (tooth fractures, luxations and avulsions) |
|---|---|---|
| Custom mouthguard | 141 165 | 13 (0.1%) |
| Boil-and-bite mouthguard | 196 332 | 21 (0.1%) |
| | | NS |

Source: From Wisniewski (70).

## Fabrication of custom mouthguards

Organizations, such as the Federation Dentaire International (FDI) have created and published recommended criteria on the construction of an effective mouthguard (72). Most of these recommendations state the same criteria:

(1) The mouthguard should be made of a resilient material, which can be easily washed, cleaned and readily disinfected.
(2) It should have adequate retention to remain in position during sporting activity and should allow for a normal occlusal relationship to give maximum protection.
(3) It should absorb and disperse the energy of a shock by:
   - covering the maxillary dental arch;
   - excluding interferences;
   - reproducing the occlusal relationship;
   - allowing mouth breathing; and
   - protecting the soft tissues.

Furthermore, the FDI also recommends that mouthguards be made by dentists from an impression of the athlete's teeth.

## Mouth-formed mouthguards

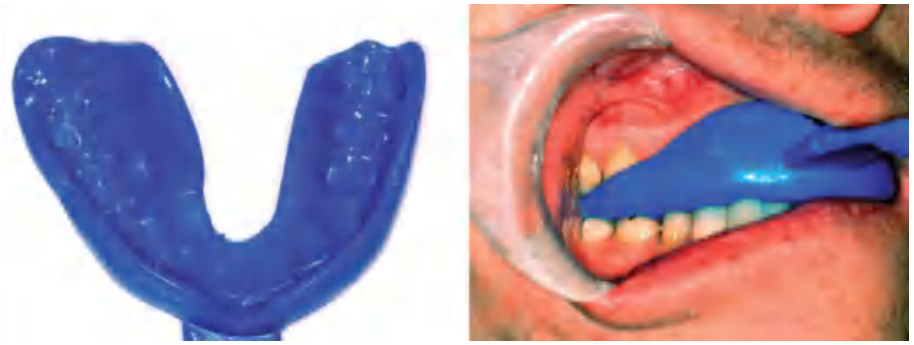
Regardless of type, the key for functionality is selection of a stock that fits the arch. If too small, it is likely that the molars will not be properly covered, thereby reducing retention and fit (Fig. 37.12). Once the proper size is found and fitted, the mouthguard should be made strictly following the manufacture's guidelines or recommendations.

### Common pitfalls in making mouth-formed mouthguards

- *Size*: Care must be taken to select the appropriate size that fits over the teeth in the dental arch. The mouthguard must cover at least one molar tooth on each side.
- *Adaptation to the teeth and surrounding tissue*: Care must be taken for optimal adaptation to the teeth and gums.
- *Trimming of the mouthguard*: After forming the mouthguard it should be carefully trimmed so that unnecessary excess is removed, without compromising the retention or strength.

## Custom mouthguards

The best way to construct a custom mouthguard is to take alginate impressions of both arches together with a wax bite taken with the patient's mandible in a physiological rest position.



A B

**Fig. 37.12** A. When making a boil and bite mouthguard, it is important to select a stock that fits the arch that it is going to be adapted to. B. If too small it is likely that the molar arch will not be properly covered and the retention and fit will be greatly reduced (B).

**Fig. 37.13** It is important to gain a good impression of the alveolus above all the teeth, even to the point that the vestibule is over-extended. The cast should reflect this with as much impression of the alveolar structure above the teeth as possible, especially in the anterior region. This will allow good adaptation of the mouthguard material to the soft tissue area and which in turn will ensure better retention as well as comfort.

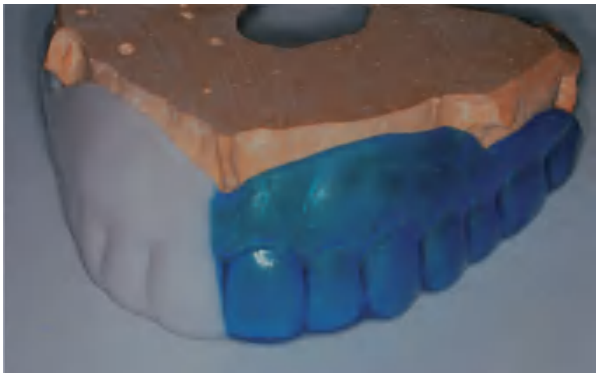


**Fig. 37.14** The cast should be trimmed so that the vestibule is almost removed. This will ensure that there will be a good adaptation of the mouthguard material to the cast when it is sucked over the cast.

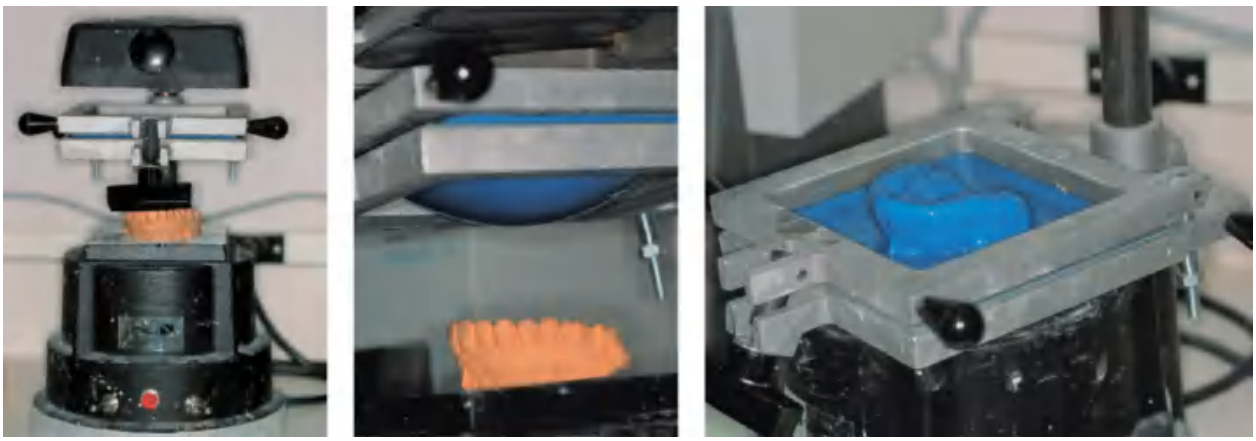
However, when time and cost is a major issue, impression of the maxilla can be considered sufficient. It is very important to get a good impression of the alveolus over all teeth, even to the point that the vestibule is overextended (Fig. 37.13). This will allow good adaptation of the mouthguard material to the soft tissue area and will in turn ensure better retention and comfort.

In a crossover study of different mouthguard extensions, McClelland et al. (73) found that comfort of wear was likely to be increased if the mouthguard was extended labially to within 2 mm of the vestibular fold, adjusted to allow even occlusal contact, rounded at the buccal peripheries, and tapered at the palatal edges. To further ensure good adaptation of the mouthguard material to the cast, the cast should be carefully trimmed so the vestibule is almost removed (Fig. 37.14). It has also been shown that residual moisture in the working cast is the most critical factor in determining the fit of the mouthguard made by vacuum-forming machines. The best fit was achieved when the working cast was thoroughly dried, and its surface temperature was elevated (74).

There are two basic methods of fabricating a custom mouthguard: the first is with the more traditional vacuum suction machine (Fig. 37.15) that basically draws the EVA stock plate over the plaster cast. It is possible to use either the single layer or prefabricated multilayered EVA in these machines; but it is very difficult to create a multi-laminated mouthguard from separate plates using this technique, as adaptation of layers will always be poor. There is a high risk of the mouthguard coming apart during use and its benefit lost.

There are several critical steps that must be followed when this type of mouthguard is made. Each must be carefully executed, otherwise the benefits of the mouthguard could be severely compromised. First, to improve the suction and thereby adaptation, a hole is drilled in the palatal area of the cast (Fig. 37.16) to ensure good suction in the palatal region. Second, the EVA plate is heated to adapt well to the cast and cool in place. If the formed mouthguard is removed while still warm, there is great risk of deformation, which will result in poor adaptation to the teeth and surrounding tissue and thereby poor retention. The third step is the one that is most often overlooked, that is, proper trimming of the mouthguard after it has cooled completely.

As stated before, the mouthguard should extend as far into the vestibule as tolerated by the patient, with appropriate clearance of the buccal and labial frena. It should extend as far back on the palate as reasonable to increase anterior strength and retention (73) (Fig. 37.17). Distally, the mouthguard should cover at least up to the second molar (75) (Fig. 37.18). The mouthguard could be trimmed with a specially heated knife on the cast or removed from the cast and scissors used (Fig. 37.19). It is advisable to replace the mouthguard on the cast after trimming is complete and



A B C

**Fig. 37.15** A. A traditional vacuum suction machine used to make custom-made mouthguards. At top there is a heating element; the stock plate is sandwiched in a jig right underneath the element. The cast is placed on a perforated plate and care must be taken that it is as much in the center as possible. When the stock plate heats up it will start to droop, so care must be taken not to overheat it because that will result in too thin mouthguard. Most commonly used EVA materials should droop about 2–2.5 cm (3/4–1 inch) before being sucked down on the cast. C. The plate needs to completely cool down on the cast before it is removed from the cast.

**Fig. 37.16** To improve the suction and thereby the adaptation drilling a hole in the palatal area of the cast in necessary. Alternatively trim the cast such that some of the palatal structure is removed. This is however only possible for individuals with a high palatal vault, because otherwise there is a danger of removing too much of the alveolar process.

**Fig. 37.17** The mouthguard should extend as far up in the vestibule as tolerated by the patient, with appropriate clearance of the buccal and labial frenum.

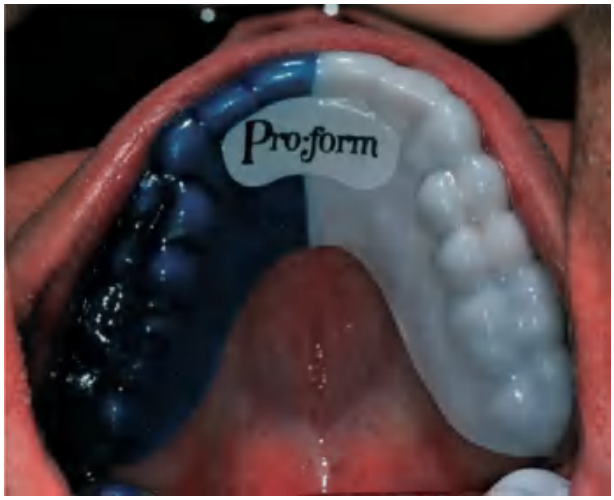



**Fig. 37.18** The mouthguard should extend as far back on the palate as reasonable to increase both anterior strength and retention, and it should distally cover up to the second molar at least if at all possible.

flame the edges with a torch. Smooth the flamed edges with wet fingers or a small spatula. Alternatively, the edge can be smoothed with a small rag-wheel in a hand trimmer (Fig. 37.20). To further improve the athlete's comfort, it is possible to gently heat the occlusal surface and then have the athlete bite together with the guard in place. This is done to even the occlusal contact between sides. Alternatively, this can be done on an articulator, if casts of both arches and bite registration are made.

The second method is with positive pressure machines, such as **Drufomat®**, **Erkopress-2004®** or **Biostar®**. In these machines, the stock plate is pressed, after being heated, onto the cast with pressure from above the plate rather than being drawn down onto the cast with negative pressure from below. This will ensure very close adaptation of the material

**Fig. 37.19** The mouthguard can be trimmed with a specially heated knife on the cast or removed from the cast. Heavy duty as well as fine curved scissors can be used.

**Fig. 37.20** The edges of the mouthguard can be smoothed with a small rag-wheel or sanding wheel in a simple hand trimmer that can even be taken to the field house.

to the cast and it is relatively easy to add multiple layers, as the positive pressure will always ensure that the new layer adapts to the existing one. It is much easier to control the thickness of the mouthguard using this technique and the stiffness of the mouthguard can quickly build up with multiple layers without losing much adaptability to the teeth. However, there must be some resilience in the mouthguard, so care must be taken not to make too stiff an appliance. An interesting possibility with this technique is the possibility of sandwiching names, numbers or logos between layers (Fig. 37.21). This will not only make it easier to know whose mouthguard is whose, but also might encourage the athlete to wear one if the team logo was clearly visible.

Once the mouthguard is made, it should be inspected for quality. The main issue, especially with the vacuum technique is thickness. There has been some effort made to find the most ideal thickness of a mouthguard to ensure comfort as well as yield optimal protection. Consensus seems to be that the thicker the mouthguard is, the more protection it affords. However, it would appear that if thickness exceeds

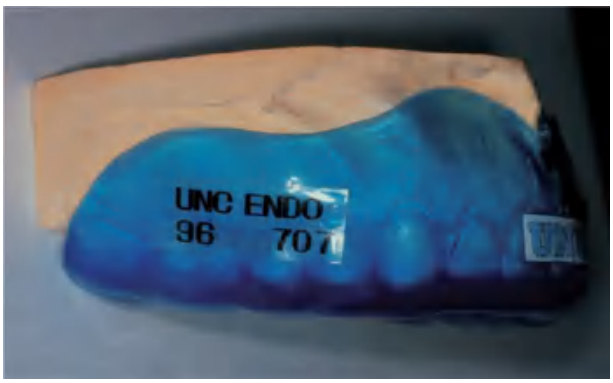



**Fig. 37.21** An interesting possibility using the multilayered technique is the possibility to sandwich in names, numbers or logos.

more than 4 mm, performance is only improved marginally and is much less comfortable for the wearer (63, 76).

### Common pitfalls in fabricating custom-made mouthguards (Fig. 37.22)

- *Extension above the teeth*: The more of the gums that are covered above the teeth, the greater the retention as well as increased strength of the mouthguard. Overextension is uncomfortable and could lead to injury in the vestibule. The mouthguard should be trimmed so that the frenum is free.
- *Extension over the molar area*: The mouthguard should cover at least one molar tooth on each side. Some extension over the gums in the molar region is recommended for retention.
- *Extension in the palatal area*: The mouthguard needs to cover some of the tissue above and behind the anterior teeth. This will both increase retention and strength of the mouthguard.
- *Rough edges*: Rough edges decrease comfort and could cause additional injuries. Smooth them with a flame, sandpaper or rag-wheel.

**Fig. 37.22** An example of a poorly made mouthguard. First, the cast is not properly trimmed so that there is a lip in the vestibule area, preventing a good adaptation in that area. Second, the mouthguard is too thin as is evident from the color of the cast showing well through the blue EVA; the extensions into the vestibule are too short in the anterior area and the edges are not smooth.

- *Too thin or too thick*: Too thin a mouthguard will not be strong enough to provide protection; too thick might be uncomfortable to wear.

### *Special considerations for mouthguards*

For patients undergoing orthodontic treatment involving fixed appliances, the brackets and arch wires may be covered with boxing wax before impressions are taken. The mouthguard may be made of a less elastic material to protect the teeth, but care should be taken not to increase the thickness so that lips or other soft tissues are put under tension. It is not clear whether mouthguards that are specifically sold to athletes who wear orthodontic appliances appear to offer any protection. Adapting a boil-and-bit mouthguard might be more beneficial if cost or convenience is an issue. This also applies for children in mixed dentition. If a custom mouthguard is made for these children, it must be remade with new impressions at least every 6 months, thus making cost a strong factor. A well-made mouth-formed guard might be as effective.

In those sports where the athletes are wearing a helmet and some kind of facemask, the mouthguard is frequently attached to the mask for convenience. The issue has been raised there should be an automatic release mechanism, so that if the helmet and/or the facemask comes off the athlete, there will not be a forceful pull on the mouthguard and thereby the teeth or the dental arch (Figs 37.23 and 37.24).

**Fig. 37.23** The connection between a facemask and the mouthguard should have self-releasing mechanisms so that if the helmet and/or the facemask comes off the athlete, there will not be a forceful pull on the mouthguard that could damage the teeth or the dental arch.

### *Care of mouthguards*

When fitted, instructions should be given for the care of the mouthguard (Figs 37.24 and 37.25). It has been reported in the past that only half of athletes clean their mouthguards after use. Eight percent even lend them to other athletes (77). Bacteriological studies have led to the recommendation that

**Fig. 37.24** The mouthguard should never be tied to the facemask or other appliances that could come loose.

**Fig. 37.25** Everyone who receives a custom-made mouthguard needs to be provided with an appropriate storage box that will allow ventilation and they should be taught how to regularly clean the mouthguard by brushing it with toothbrush and tooth paste.

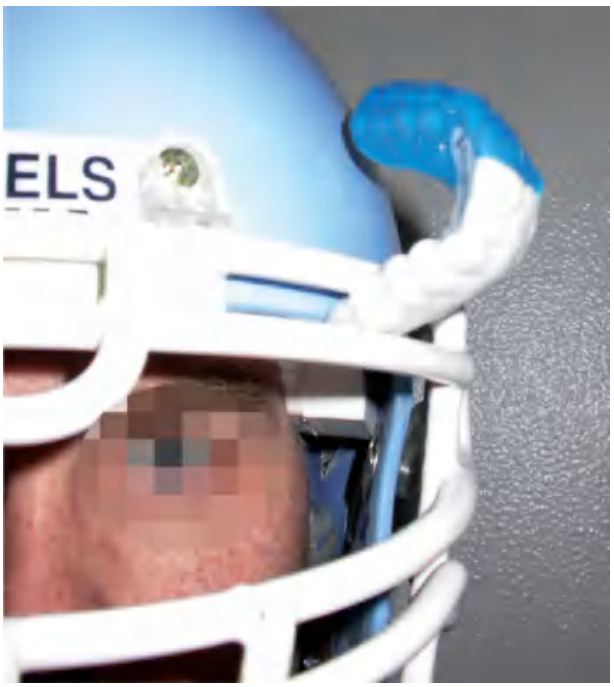



**Fig. 37.26** Athletes should also be taught proper care of the mouthguard during play, such as not chewing on it or placing it somewhere where it can easily be lost.

mouthguards should (a) be washed with soap and water immediately after use; (b) be dried thoroughly and stored in a perforated box; and (c) be rinsed in mouthwash or mild antiseptic (e.g. 0.2% chlorhexidine) immediately before use. Additionally, it should be recommended to the athletes to not consuming a sugar containing sports right before inserting a mouthguard, rather right after. This is because it has been shown that that consuming sports drinks just prior to using a custom-made mouthguard can affect the buffering capacity of the free-flowing saliva and thereby a possible risk factor for dental caries (78).

The mouthguard should be inspected regularly to check its fit, especially in children who are still growing. During use it may become distorted or torn by chewing. Athletes should be warned that chewing the mouthguard will shorten its life (Fig. 37.26).

## *Use of mouthguards and other protective appliances in various sports activities (27)*

Oral and dental injuries have been reported to be a relatively large portion of all injuries reported for certain sports (14, 79). Nysether found for example that approximately 20% of all reported injuries among Norwegian soccer players were dental injuries (79). In every fifth case the expenses for necessary dental treatment exceeded the maximum compensation from the insurance company. The best protection available for the oral and dental area should be a priority for everyone involved in sports activities, not only for the benefit of the participants but also to reduce the potential cost and liability of the organization.

### American football

As stated previously in this chapter the introduction of facemasks and proper helmets virtually eliminated oral and dental injuries (25–27). For dental protection the choice of mouthguard for American football does not appear to matter: boil-and-bite mouthguards appeared to provide as good protection as the custom-made ones (70). However, the comfort of the custom-made is unquestioned especially for those who have to communicate frequently during the game.

### Baseball

The best protection for baseball players is a faceguard or a cage attached to the batting helmet. An intervention study in juvenile baseball found that wearing a faceguard resulted in significantly less oculofacial injuries than not wearing one (30). For other players on the field it is possible that they would benefit from wearing a custom-made mouthguard but as of yet no published study has investigated this.

### Basketball

Mouthguards have been shown to significantly reduce dental injuries and referral to dental offices for male players (40). For this sport there is no question that a custom-made mouthguard is the only choice because both stock and boil-and-bite require the wearer to clench their jaws to keep it in place. The air flow is not affected by a custom-made mouthguard but can be with a stock type (80).

### Boxing

Mouthguards were initially created for boxers to protect their soft tissue (33) but it soon became apparent that they protected the teeth much more than the lips and gingiva (33, 34). There has been mandatory wear of a mouthguard in boxing at almost all levels for a long time and it is of note that despite repeated blows to the chin and face dental injuries appear to be relatively infrequent, most likely due to the mouthguards. It has been reported that none of 250 participants in an amateur boxing championship who were evaluated after the game had any visible dental trauma (81). All kinds of mouthguards have been suggested for boxers, both stock and custom-made maxillary, as well as various forms of 'bimaxillar' that covers both arches. However once again no comprehensive study has been done to show that one type is more beneficial than other.

### Field and ice hockey

Hockey has been traditionally considered to be very dangerous for the oral and dental structures. A report from Helsinki, Finland found that of 106 patients with sports-related dental traumas, 39% of cases happened in ice hockey or skating. Many amateur and college level sports organizations have by

now mandated faceguards as well as mouthguards for their players. Those actions have virtually eliminated lower facial injuries (29), but only in the case of full facial guards because it has been shown that the so-called half mask that only covers the upper half of the face does not protect the jaws or oral cavity (31). In field hockey a systematic review and meta-analysis including 11 articles showed that on average proportion of field hockey players who had sustained at least one dentofacial injury varied from 12.7% among junior and senior players to 45.2% among elite players indicating ongoing need for further protection (82). However, the study indicated that actually fewer players were using a mouthguard now compared to the 2000s (82). The additional benefit of wearing a mouthguard to a full face-mask in hockey is not clear and needs to be further investigated.

#### Horseback riding

Facial injuries have been reported to be common among those who ride horses (83). However, the use of custom-made mouthguards is not promoted by those in the equestrian industry. Use of helmets with face protectors have become more common, especially for the younger generation of riders but data is lacking about the effectiveness of those (84).

#### Rugby

Several papers have reported on the protection, or lack thereof, of mouthguards (38, 41) in rugby. Once again a large intervention study is needed to clearly demonstrate how much a mouthguard does protect rugby players. Custom-made mouthguards are probably the best for these players, primarily for comfort.

#### Soccer

Soccer is without a doubt the most common sports activity in the world. The incidence of oral injuries has been reported surprisingly high, for example 28–32% in high school soccer players in a 2-year observation (85, 86). Yet in many areas there has been a very little discussion about need for dental protection. The risk seems to be especially high for goalkeepers and forwards, as well as for inexperienced teams (79). A custom mouthguard should be considered for those who are at risk.

#### Team handball

Team handball is another aggressive contact sport where oral and dental protection does not seem to have gained much interest although there seems to be a high rate of injuries for participants (87). Effort should be made to make players aware of the risk as well as to investigate whether a custom mouthguard does offer protection.

### Cost–benefit of impact protecting devices

An unanswered question at present is how the cost of protecting individuals from injuries relates to the cost of treating actual injuries in a non-protected situation. This question becomes substantial in many sports activities where traumas are not that frequent like fore example skiing or fencing (88). Before such a question can be answered for individual sports disciplines the following questions should be answered:

- What is the incidence of traumatic dental and maxillofacial injuries in that particular sport activity?
- Is there a typical trauma pattern in the particular sport?
- How effective are different protective devices?
- What are the costs of these devices?
- What is the cost of treating a typical traumatic injury related to that particular sport activity?
- Can the protective device possibly cause a shift in injury pattern, like protecting the crown but causing instead alveolar or root fractures?

As stated previously very few studies have been conducted in a scientific manner and on a large scale on the actual effectiveness of all the above-mentioned protective devices. This was strongly highlighted in 2002 when a Task Force on Community Preventive Services (formed by the USA Government Agency: Centers for Disease Control and Prevention in Atlanta, GA) did systematic reviews of all papers that reported on effectiveness, applicability, other positive and negative effects, economic evaluations and barriers to use of selected population-based interventions intended to prevent or control sports-related craniofacial injuries (89). The report stated that there was not a single study that was sufficient to meet minimum requirements of the Task Force for inclusion in the report on the issue. The Task Force concluded: 'According to *Community Guide* rules of evidence, evidence is insufficient to determine the effectiveness of population-based interventions to encourage use of helmets, facemasks, and mouthguards in contact sports in increasing equipment use or reducing injury-related morbidity or mortality' (89).

There is no question that we need in the very near future multiple large epidemiological studies on sports injuries and their prevention, to confirm the clinical impression of effectiveness of protective devices. Detailed analysis of cost effectiveness should also be carried out for every sporting activity once that data is collected. This would be a good foundation for rational accident prevention in sports.

## Other applications for the use of mouthguards

### Protection of the dental tissues during general anesthesia

Oral endoscopy and orotracheal intubation may result in fracture or displacement of teeth (90–92). Damage may be inflicted by using the incisal edges of the anterior teeth as a fulcrum when inserting a laryngoscope, retractors or endoscopes. The fracture of prosthetic crowns has also been

reported and injuries to the teeth are one of the most frequent complications during the delivery of general anesthesia (93). However, it is not at all clear how frequent this kind of trauma is, for it is likely that in many cases the damage is not discovered until months if not years later because the tooth or teeth only sustained mild luxation during the trauma rather than crown fracture or frank avulsion.

It has been suggested that patients who present with discolored and/or symptomatic anterior teeth for which there are no obvious etiologic factors should be carefully questioned regarding their past surgical history. If the patient denies a history of trauma yet relates a recent (2–5 years) history of general anesthesia, a differential diagnosis including trauma secondary to endotracheal intubation should be considered (94).

There are a number of injures to teeth during general anesthesia. The most frequent of all anesthesia-related medicolegal claims was for dental injuries: up to one-third of all medicolegal claims against anesthetists (95). Concern for dental injuries of this type has increased due to an awareness of the medicolegal responsibility of the surgeon or anesthetist for iatrogenic injury (93). A number of methods have been recommended to prevent injuries in these circumstances; but there is little evidence of their success. Custom-made mouthguards have not been considered cost-effective. This might be in part because anesthesiologists fear that the mouthguard could get in the way during intubation. However, a recent study showed that when comparing 80 patients, where half had a mouthguard and the other not, there was only on average 7 seconds difference between the two groups (96). When one considers the possible cost of legal action due to dental trauma, even as infrequent as they may be, the case for a dental protection of some sort during intubation becomes even stronger. At least in cases where there is a great risk of complications due to restorations, limited mouth opening etc., this approach might be advisable (97, 98).

## Prevention of oral injury from traffic accidents

As it appears from Fig. 37.2, traffic accidents are the cause of a significant amount of oral injuries. In that regard, some preventive measures can be made.

### *Bicycle injuries*

These injuries are very common in some countries, due to the popularity of bicycles (99). In addition, a relatively new extreme sport where a rider does tricks, jumps over obstacles and slides on rails etc. on BMX bikes has raised concerns by many dentists who treat dental injuries about the potential for serious injuries and the apparent lack of protectors for the lower portion of the face. No study has of yet has been completed on this subgroup of bicyclists but there is every indication that they do suffer as frequent and serious oral injuries as extreme skateboarders and rollerblades.

Bicycle riders are extremely exposed to head injury during accidents (28). In most of these injuries the impact direction is either direct and frontal or tangential to the face resulting in a combination of dental and soft tissue wounds when the face hits or slides along the ground (Fig. 37.27). The use of bicycle helmets has been common in professional bicycle sport and is now becoming popular among both children and adults. In some countries laws have been passed to require at least all children and even everyone riding a bicycle to wear an appropriate helmet. The bicycle helmet is primarily designed to prevent brain injuries and has been shown to reduce the risk of these injuries by 48% (28) but it offers no protection against injuries to the lower part to the face, including dental injuries (99–101).

**Fig. 37.27** Head and facial injuries are frequent in bicycle accidents. In most of these injuries the impact direction is either direct and frontal or tangential to the face resulting in a combination of dental and soft tissue wounds when the face hits or slides along the ground.

### *Motorcycle injuries*

These traffic injuries are the most dangerous of all traffic accidents. In most countries the mandatory use of helmets has been legislated for. This preventive measure has been found to reduce the mortality rate significantly and also the frequency of oral and maxillofacial injuries (102, 103). Motorcycle helmets with chin-bars are likely to provide the best protection for the lower half of the face.

### *Motor vehicle injuries*

Head injuries are common in car accidents and vary significantly according to the person's position in the car. The driver is usually affected by direct impact with the steering wheel and/or the windscreen whereas the front seat passenger suffers from injuries related to a collision with the front seat panel and/or the windscreen. The back-seat passenger usually faces collision with the front seat. In all of these instances a frontal impact is the result with a combination of soft tissue and dental and/or maxillofacial injuries.

Since the introduction of safety belts and later airbags a significant reduction in not only the mortality after automobile injuries has been noted but also a reduction in the severity of oral and maxillofacial injuries (Fig. 37.28) (104).

**Fig. 37.28** The introduction of safety belts and later airbags has significantly reduced not only mortality after automobile injuries but also the severity of oral and maxillofacial injuries.

## Essentials

The majority of dental and oral injuries appear unexpectedly and during daily life activities, which make their prevention difficult if not impossible. The best approach is education, both on how to avoid injuries as well as how to manage them when they occur.

At present preventive measures can be indicated in the following situations:

- Contact sports
- Certain traffic activities (bicycle, motorcycle, motor vehicle)
- Oral endoscopy and oro-tracheal intubation in cases with extensive restorations or a weakened periodontium.

Altogether, 7–25% of dental injuries appear to have a nature where preventive measures have a chance of reducing or preventing those from occurring.

### Preventive devices

- Helmets may prevent skull fractures and to a certain degree brain concussion but not dental injury if they are without faceguard or chin bar.
- Faceguards may prevent facial and oral injuries.
- Mouthguards may prevent or reduce the severity of oral traumas.

### Design of mouthguards

An ideal mouth guard should ideally have the following functions:

- Preventing or reducing injuries by absorbing or deflecting frontal and axial impacts
- Shielding the oral soft tissue
- Providing the mandible with resilient support during forceful occlusion that may prevent crown root fractures of premolars and molars, jaw fractures and brain concussion.

### Types of mouthguard

- Stock – prefabricated
- Mouth formed
- Custom-made

Among these the custom-made has the optimum comfort for the wearer and according to some the best trauma preventive capacity.

### Use of mouthguards and other preventive measures in sport

#### *American football*

Helmets and face masks significantly reduce the chance of oral injury.

#### *Basketball*

Mouthguards significantly reduce the frequency of oral injuries for males.

#### *Boxing*

Mouthguards reduce the chance of dental injuries.

#### *Field and ice hockey*

Face masks and/or mouth guards significantly reduce facial and oral injuries.

#### *Horseback riding*

There is a high frequency of facial injuries. There is no report on the effect of preventive measures.

#### *Rugby*

Mixed protection effects of mouthguards have been reported.

#### *Soccer*

Possibly goal keepers and forwards may need mouth guard protection. Unfortunately no studies have verified this.

#### *Team handball*

This sporting activity is attended by a number of injuries. There are no data of oral protective measures.

### Use of preventive measures in traffic

#### *Bicycle injuries*

The use of helmets appears to reduce the frequency and severity of facial injuries.

### *Motorcycle injuries*

Helmets appear to reduce the frequency and severity of head injuries.

### *Car injuries*

Safety belts reduce the frequency, severity and mortality of facial injuries.

## Use of preventive measures in endoscopy and orotracheal intubation

Mouthguards are indicated in cases of maxillary anterior restoration (crowns, bridges and implants) or reduced periodontal support. The effectiveness of this procedure has not yet been documented.

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# 38

# Prognosis of Traumatic Dental Injuries: Statistical Considerations

P. K. Andersen, F. M. Andreasen & J. O. Andreasen

## Identifying prognosis related factors

### Univariate and stratified analysis

In most dental trauma studies, a univariate analysis is performed where healing outcomes are compared against various pre-injury and injury factors (e.g. age, root development, severity of trauma) and treatment factors (e.g. type of splinting, antibiotics, treatment delay). A minimum observation time should be used (usually one year) in order for a given complication to become manifest. A *univariate analysis* is then performed using Chi square or other tests. This will normally reveal a series of significant relations among which a number are due to associations between related parameters (e.g. age and root development). To reveal these associations a *stratified analysis* should be performed where for instance root development (mature and immature) is subdivided for given age groups. In this way a certain number of associations can be revealed, and redundant factors can be excluded from having a significant influence on healing outcome.

## Expressing prognosis quantitatively

In the dental literature, the most frequently used method for expressing prognosis is as proportion survival, where n treatments are followed for time intervals of varying lengths and $x/n \times 100\%$ of these procedures develop a given complication x. Prognosis is then related to an average or median interval (1, 2).

When analyzing the occurrence of events, such as pulp necrosis (PN), pulp canal obliteration (PCO) and root resorption (RR) after dental trauma, *time* becomes very important. First, from a clinical point of view, where the time after injury at which complications are likely to occur, and the order in which different complications can be anticipated, play an important role for clinical decision making.

As healing complications usually appear with some time variation, the length of observation period of patients followed is decisive (Fig. 38.1). If, for example, the observation period for an overwhelming majority of patients in a trauma study is so long that most complications will have appeared (period C), conventional frequency calculations will provide a reliable picture of the likelihood of a given event. However, if prognosis is calculated from a material with a very short observation period (period A), an unrealistically optimistic picture of complication events will be presented. In period B, a varied picture of the appearance of complications will be seen due to variation in length of observation periods and their relation to the chance of detecting a given complication (1).

It can be seen from Figs 38.2–38.4 that pulp necrosis, pulp canal obliteration and root resorption after luxation or avulsion with subsequent replantation of permanent teeth appear with relatively large variations in time (3–8). Thus, expression of prognosis for these complications will show great variation from one investigation to another, making comparison of results difficult, if not impossible. However, using the method of meta-analysis (see later), it is possible to summarize the results from similar investigations in a quantitative manner.

Also, from a statistical point of view, the time factor is important because teeth are observed at varying time intervals due to staggered entry and a fixed closing date, implying different probabilities of experiencing a given complication within the period of observation. This fact must be taken into account in statistical analyzes of clinical data on dental trauma. This problem can be overcome by focusing on events during a time interval of fixed length and expressing prognosis as proportion survival as explained above; but such a procedure may involve a serious loss of information.

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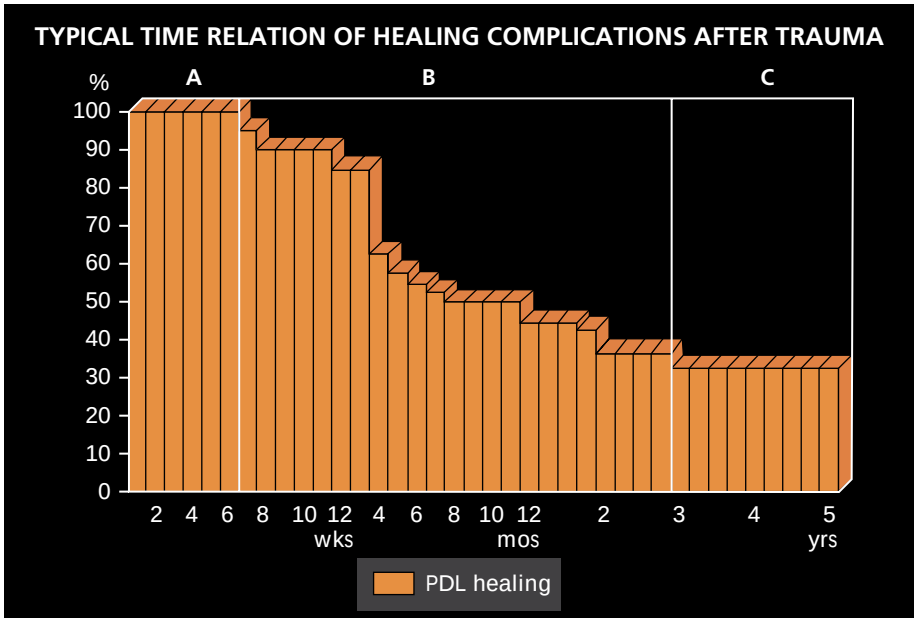


**Fig. 38.1** Typical time relation between occurrence of healing complications after dental trauma. Source: Reproduced from (1).

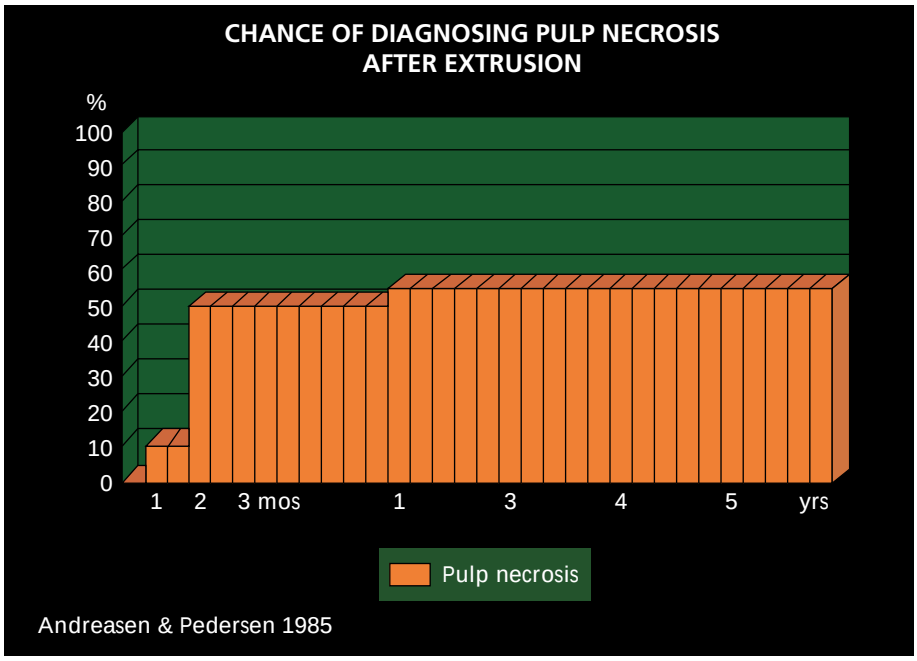


**Fig. 38.2** Observation time and probability of diagnosis of pulp necrosis after extrusion of mature permanent teeth. Source: Reproduced from (3) with permission.

## Life table analysis

An informative expression of prognosis is achieved by means of *life table*, or *actuarial statistics*, by which variation in prognosis with variation in observation period can be described. Such an analysis permits full utilization of all 'survival' information accumulated up to the closing date of the study, including information from patients with partial follow-ups (i.e. patients not followed through to the occurrence of a given complication or terminal event). This form of analysis is well known and widely applied in studies of life expectancy with cancer (9–12). It has also been applied to long-term dental investigations, such as the longevity of dental filling materials (13–16), pulp capping procedures (17), tooth transplantation (18, 19), endodontic procedures (25–27, 29, 38) and long-term prognosis of various dental trauma entities (3–8, 20, 21, 35–37). Non-technical text books on survival analysis are also available (12, 22–24).

A crucial difference in survival data from, e.g., cancer trials and data on occurrence of complications after dental trauma results from the non-fatal nature of the latter complications. Typically, the occurrence of a dental complication must be diagnosed in a clinical investigation, and this means that only at some predetermined observation periods can it be seen whether a given complication has arisen since the previous follow-up examination; but the precise time of occurrence in the time interval cannot be determined. Thus, the relevant statistical analysis of occurrences of complications is an analysis of *grouped survival data* (3).

A calculation illustrating the principles of life table analysis is presented in the following example of patients suffering

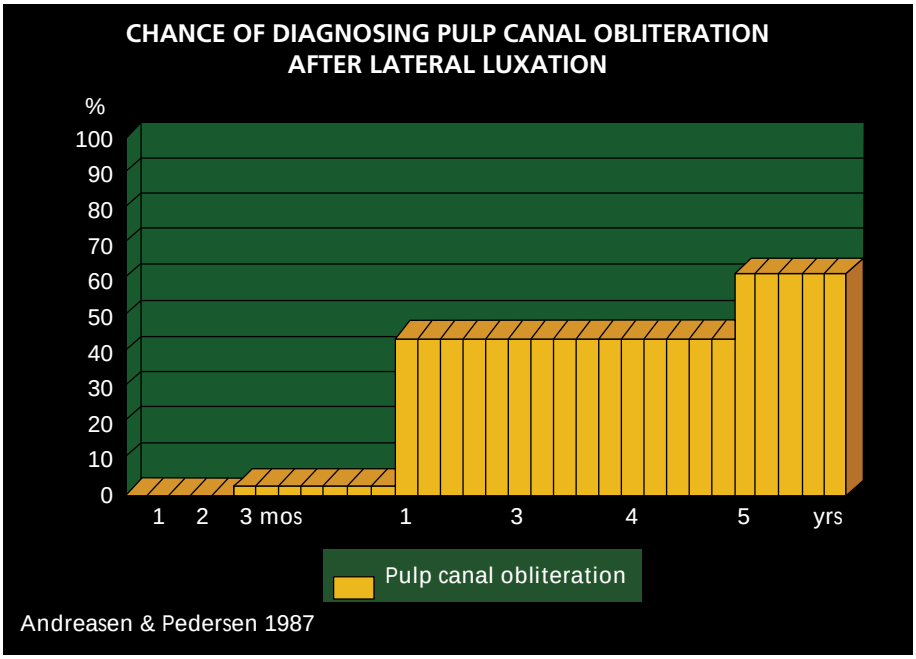


**Fig. 38.3** Observation time and probability of pulp canal obliteration subsequent to lateral or extrusive luxation of mature permanent teeth. Source: Reproduced from (5) with permission.

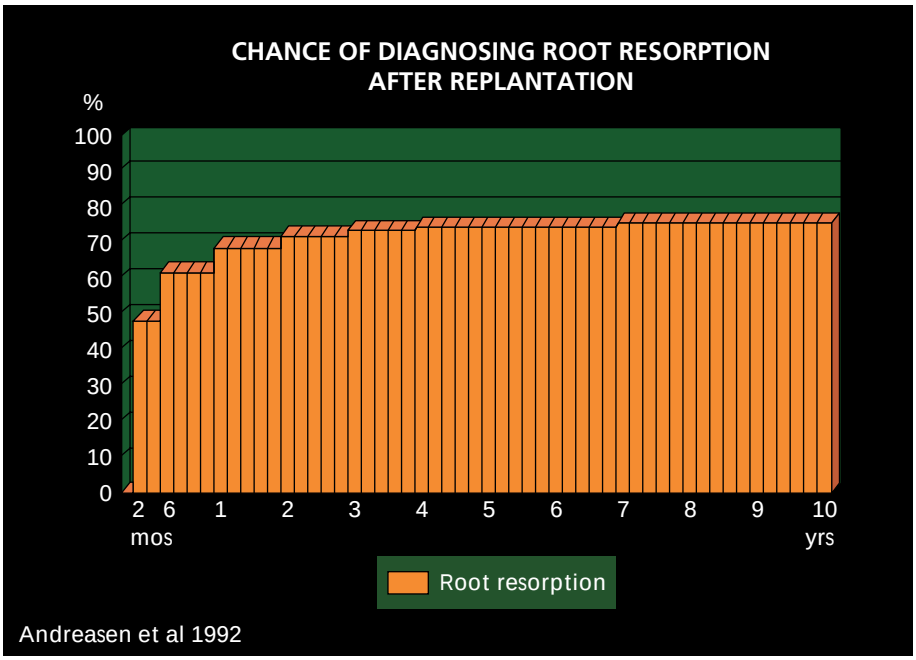


**Fig. 38.4** Observation time and probability of diagnosis of external root resorption after replantation of avulsed permanent teeth. Source: Reproduced from (7) with permission.

dental trauma (e.g. replantation of avulsed teeth) (Fig. 38.5). Each patient will represent one of the following three outcomes:

- *Failed* – if the replanted tooth has developed one of the complications under study.
- *Withdrawn* – if the tooth (patient) is lost from the trial for any reason other than healing complications from the injury. For example, if a patient stopped attending recall examinations and was lost to follow-up, or if a tooth was lost due to causes unrelated to the initial trauma, e.g. a new acute injury.
- *Censored* – if the tooth is still in function at the end of the investigation and does not show the complications being studied.

The following example will show the principles of the calculations behind a tooth survival analysis: Ten patients with a total of 10 replanted incisors participate in a prospective investigation. There are no teeth showing failure at the end of the first observation period (a). Tooth survival at the end of the first interval is therefore 100%. At the end of the next interval (b), failure is seen in two teeth. Tooth survival is now:

$$(10-2)/10\times 100\% = 80\%$$

In the third observation period (c), there are now only eight teeth at risk. Of these eight teeth, four fail; that is, 4/8 of 80% have survived, yielding 40% survival. No complications are registered in the fourth interval (d), but two patients have

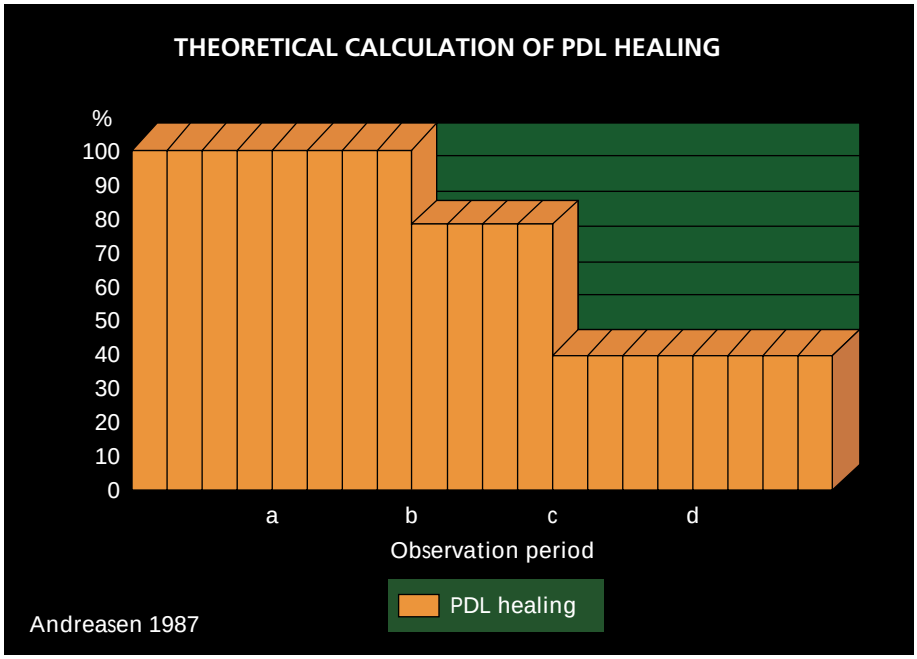


**Fig. 38.5** Calculation of periodontal ligament survival in a theoretical clinical dental trauma study. Source: Reproduced from (1) with permission.

such short observation periods that they are withdrawn from the study. There are now only two teeth left. Tooth survival is therefore 2/2 of 40% = 40%. It can be seen from Fig. 38.5 that the risk for a given complication can now be calculated for a given observation period (3).

Life table statistics are sensitive to a number of factors, such as definitions of failed, withdrawn and censored patients, as well as recall procedures and sample size. The significance of these factors will therefore be discussed.

## Importance of definition of failures

*Failures* can be defined according to the type of dental trauma analyzed, such as loss of tooth, root resorption, development of pulp necrosis, pulp canal obliteration or loss of marginal bone support. Each of these conditions (apart from tooth loss) represents unique diagnostic difficulties. Thus, *pulp necrosis* is dependent upon a number of diagnostic procedures, where none is conclusive (see Chapter 16). *Pulp canal obliteration*, *root resorption* and *loss of marginal bone support* are primarily dependent upon radiographic examination procedures (see Chapters 2 and 16). This implies that a variation in technique could influence the sensibility of the analysis. Concerning *tooth loss*, it should be established whether the tooth in question was otherwise intact and in function, and that the extraction was *not* related to the previous trauma (e.g. a traffic accident, extraction for orthodontic or prosthetic purposes). Otherwise serious bias might arise.

## Importance of censored patients

The definition of a censored event (i.e. tooth survival or tooth presence without pathology), like failures, is entirely dependent upon the established criteria for pathology. Furthermore, it is important that the majority of censored data represent adequate observation periods for the complication to have had a chance to appear. Otherwise problems may arise regarding the reliability of the survival analysis.

## Importance of withdrawn patients

The usual reason for withdrawal of patients from continuing examination appears to be one of the following:

- The patient cannot be traced (moved without a forwarding address, emigration, death).
- The patient has received a recall notice and chosen not to respond (28).
- The tooth in question is lost.
- The tooth in question has survived and the patient does not feel the need for a follow-up examination.
- The patient ignores the recall notice for other reasons (e.g. inconvenience or lack of interest).

If the reasons for no-show at a scheduled follow-up are centered around the first two reasons for withdrawal, the analysis may be seriously jeopardized. The relevance of no-shows has only been studied in a few dental investigations (25–27). Thus, it was shown in a long-term study of healing after surgical endodontics that patients not initially responding to recall notices, when later examined after extensive recall efforts demonstrated the same healing pattern as the rest of the material (27). This illustrates that, at least in the cited study, the no-shows were usually related to inconvenience for the patient of the scheduled examination. Also, the situation where patients feeling well tend to withdraw may cause bias since the patients remaining in the study are no longer representative for the treated patient population (so-called 'dependent censoring').

### Importance of the length of the investigation period

This is a crucial factor and should be related to the mean or median observation period for the occurrence of the event under investigation. Thus, root resorption requires extended observation periods for the material before any meaningful survival statistics can be established (7, 8) (see Chapter 4).

### Importance of sample size

The sample size becomes very important especially when the pathological event under investigation occurs infrequently.

### Effect of recall schedule

As mentioned above, it can usually only be established at some prescheduled follow-up examinations whether a given complication has arisen since the previous examination, whereas the precise time of occurrence within that period cannot be determined. The recall schedule is therefore important. Short time intervals between successive follow-up examinations are recommended when the rate of failure is high, whereas longer time intervals may be chosen when few failures are likely to occur.

## Comparing life tables

### The logrank test for comparison of life tables

Life table estimates for the survival probability in two groups, e.g. two randomized treatment groups, may be compared at a single point in time, t, using the variance estimate based on Greenwood's formula (12). It would be more efficient, however, to compare survival of the two groups on the entire survival functions and not only on their value at a single point in time. This may be done using the *logrank test statistic* for the case of continuously observed i.e. *non-grouped survival data* (9, 10). For *grouped survival data*, a similar test has been developed (12).

### Further treatment comparisons, multiple regression analysis

In almost all clinical investigations, the identification of clinical factors which have an effect on the occurrence of healing complications after trauma has been achieved using univariate statistical analysis, where it is assumed that all teeth in the group for which the estimate is calculated have the same survival probability. In this type of analysis, each potential causative factor is considered individually, usually with the result that numerous clinical factors can be identified that are significantly related to the given healing complication. In many cases, however, the groups are heterogeneous, and the relationships found may have little biological relevance, as they may be due to associations between registered clinical parameters. As an example, assume that the stage of root development is the only clinical parameter related to a given complication, and a given type of treatment (e.g. repositioning and splinting) is chosen according to stage of root development. In such a case, in a univariate analysis, there may be a significant relationship between treatment procedure and complication due to this association.

In order to identify and eliminate such associations, various multiple regression analyzes have been developed whereby the effect of several factors is studied simultaneously.

In the case of continuously observed data, the *Cox regression model* is frequently used (12, 22–24, 30). It is also possible to use such a model for grouped survival data when, e.g. treatment comparisons are to be made in the presence of other prognostic factors (3, 31). An alternative model which is frequently used is the *logistic regression model* for grouped survival data (30). This model leads to effects of prognostic factors being quantified as *odds ratios* (Table 38.1). Using such multiple regression models, associations can be disclosed and/or eliminated, and this has been shown to significantly reduce the number of predictors for healing complications after dental traumas (3, 6–8, 32).

### Odds ratio (OR) and risk ratio (RR)

OR represents the risk for an experimental group compared to that of the control group. RR is defined as the ratio of risk in the intervention group to the risk in the control group. Risk is the ratio of people with an event in a group to the total in the group.

In the following example, the action of OR and RR is shown for a constructed example in a trauma situation (Table 38.1).

$$\mathrm{OR} = \frac{a/b}{c/d} = \frac{10/90}{20/80} = 0.44$$

$$\mathrm{RR} = \frac{a/e}{c/f} = \frac{10/100}{20/100} = 0.50$$

An OR or RR of 1 indicates no difference between comparison groups. For undesirable outcome, an OR that is <1 indicates that the intervention was effective in reducing the risk of outcome. Likewise figures above indicate increased risk in the intervention group.

### The use of predictors and confidence limits

From a regression model it is possible to estimate the probability that a given complication will arise during a given interval for a patient having a tooth with the given characteristics. Confidence limits for such a predictor may also be estimated. It is important to note that, due to the association between prognostic factors, such individual predictions

**Table 38.1** Effect of antibiotics upon fracture healing.

| | Infected cases | Healed cases | *n* |
|---|---|---|---|
| Antibiotics (intervention) | 10 (a) | 90 (b) | 100 (e) |
| Control | 20 (c) | 80 (d) | 100 (f) |

*must* be based on a multiple regression model and cannot be obtained from a series of single-factor analyzes.

### Comparing tooth survival or healing results from various studies

In the evaluation of the effect of various treatment modalities, the usual approach is to report the individual findings from clinical studies, the so-called narrative review (33). Such an analysis usually results in a range of treatment effects from which it is difficult to summarize the effect of a given treatment. To overcome this difficulty, *meta-analysis* can be used. This method uses statistical techniques to summarize estimates from a series of studies with common underlying characteristics (34) and thus allows estimation of the magnitude of a treatment effect. The principle is that when the effect of the same treatment has been estimated in a number of similar studies (together with confidence limits), a quantitative overview can be obtained as a weighted average of the individual estimates using as weights the precision of the individual estimates. It should be mentioned that any conclusion based on meta-analysis is only as valid as the included studies. If there is a high risk of bias of the included studies, there is also a high risk of bias in summation of the studies and the meta-analysis. Because of this, the appraisal of the risk of bias is a key element in performing a systematic review (39).

## Essentials

### Expressing prognosis quantitatively

- Percent calculation (i.e. disregarding relationship to observation period)
- Life table statistics (i.e. incorporating relationship to observation period)

### Factors of importance for life table analysis

- Definition of healing and non-healing
- Censored patients
- Withdrawn patients
- Length of period under consideration
- Sample size
- Recall schedule

### Comparing life tables

- Log rank test for treatment comparisons

### Multiple regression analysis

### Odds and risk ratio

### Predictors and confidence intervals

### Comparing healing results

- Meta-analysis

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# 39
# Splinting of Traumatized Teeth

J. O. Andreasen & K. S. Oikarinen

Until the 1970s, splinting of traumatized teeth was primarily accomplished using methods employed in the treatment of jaw fractures, with cap splints, arch bars and wires (1–3). This was not only because of a lack of knowledge of the healing mechanisms of injured teeth, but also because of lack of appropriate splinting materials. Since the discovery of adhesive techniques in the late 1960s, a wide range of splinting devices has been developed (4, 5). However, the most important discovery in recent decades has been that splinting in general can have an adverse effect on healing processes in the periodontium and pulp after trauma. See Chapter 3 for a detailed analysis of the effect of splinting after dental traumatology. Furthermore, the reader is referred to a detailed review article by Kahler & Heithersay (2008) (49). Thus, non-physiologic fixation of displaced teeth can induce periodontal and pulpal healing problems; and long-term splinting may not only prolong the wound healing process in the periodontal ligament (6) but may also lead to preservation of an otherwise transient ankylosis (7–10). Arrest of revascularization of the pulpal has also been observed in animal experiments (9). In autotransplantation of teeth, both ankylosis and increased risk for pulp necrosis have been observed more frequently in rigidly splinted than in flexibly splinted autotransplanted molars in humans (11). In the following, the effect of various splinting devices on the healing of dental tissues after injury will be discussed.

## Influence of splinting on dental tissues

### Influence upon gingiva

It has been shown that gingival damage caused by arch bars fastened with steel wires is reversible if the periodontium was healthy before splinting (12–19). Wire-loop splints (used in intermaxillary splinting) have been found to lead to gingival changes which are, however, reversible after wire removal (20). The presence of wires placed in contact with the gingiva may lead to invasion of bacteria through rupture or tears in the epithelial attachment.

### Influence upon periodontal healing

An early experimental study in 1974 by Andreasen (7), and supported by later clinical (11, 21) and experimental studies (8–10, 22), demonstrated that optimal periodontal healing (i.e. with minimal ankylosis) after extraction and replantation of teeth in animals was obtained in a non-splinted situation compared to rigid splinting. It is assumed that slight mobility in the initial healing period activates resorption of initially formed ankylosis sites. Only a single experimental study did not support these findings (23).

### Influence upon pulp healing

In a monkey model of extracted and replanted teeth, it was shown that splinting could decrease pulp revascularization and increase the extent of pulp necrosis and inflammatory root resorption compared to non-splinting (9). In humans, splinting of autotransplanted teeth for only one week (with a suture splint, see later) has been found to improve pulpal healing as compared to rigid splinting for four weeks (21).

### Enamel changes after splinting

The staining of labial enamel after acid etching could present a problem, especially if filled composite resin has been used, where there is no clear distinction between enamel and the splinting material. However, experimental studies have shown that treatment of enamel with methods simulating

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various phases of splinting does not cause permanent staining; and discoloration can be removed with careful polishing (24).

## Requirements for an acceptable splint

The splint should preferably have slightly vertical and horizontal flexibility in order to support healing (see above). In the following, the rigidity of splints will be described as: *flexible* i.e. more mobility than a non-injured tooth, *semi-rigid*: equal to normal tooth mobility, and *rigid*: less than normal tooth mobility. An optimal splint should fulfil most or all of the following requirements:

- Direct intraoral application
- Easy to construct with materials available in dental practice
- Does not increase periodontal injuries or promote caries
- Does not irritate oral soft tissues
- Passive; does not exert any orthodontic force on teeth
- Versatile in achieving rigid, semi-rigid or flexible splint
- Easy to remove and causes minimal or no permanent damage to the dentition
- Allows pulp testing and endodontic treatment
- Hygienic and esthetic.

## Splinting methods

Since the discovery of the acid etch technique of enamel by Buonocore in 1955 (4), this procedure has had a major influence on splinting devices (5). Since the 1970s, several splinting methods have been introduced which allow slight horizontal and vertical movement, many of which utilize the acid etch technique, various types of wires or orthodontic appliances (25–29). Thus, composite and similar materials can be attached firmly to the enamel surface. The area needed for acid etching can be limited to a small surface of the crown (3).

## Testing the mechanical properties of various splinting types

An experimental *acrylic model* jaw-tooth was designed to simulate the clinical mobility of teeth and the influence of various splinting devices. The lateral and vertical flexibility of two *arch bar splints* (Schuchardt and Erich arch bars), which are frequently used in the immobilization of jaw fractures, and a *wire-composite splint* were examined (30). The tests showed that a 0.3 mm thick wire-composite splint had a flexibility which closely resembled that of a control situation, while arch bar splints with and without acrylic coverage virtually prevented vertical and horizontal movement (30).

In a second study, using a *sheep jaw model*, a variety of different splinting materials were tested for their flexibility and grouped into rigid, semi-rigid and flexible (31). In this study, light cured composite resin splints, Fermit® (Vivadent, Germany) and Triad® gel composite splint (light cured gel form acrylic, Dentsply, USA) yielded a *rigid* splint. *Semi-rigid* splinting was achieved by Kevlar®, Fiber-splint®, Protemp II® (multiple functional methylacrylic esters, Espe, Germany) and flexible wire-composite splint. The latter splint types provided adequate lateral support for a loose injured tooth and also allowed slight vertical flexibility. Also, a cadaveric model used to test composite splint showed significant less mobility than other flexible solvents (50).

In order to establish optimal splinting stability, the foremost requirement is to define to what extent a loose tooth or tooth-bone segment should be immobilized in order to ensure optimal healing. Unfortunately, no such analysis exists. However, experience from vertical distraction osteogenesis indicates that limited mobility can advance osteogenesis (see Chapter 34).

Properties of various suitable splinting materials are compared in Table 39.1. The most versatile splints are resin splints (Protemp®, Luxatemp®), wire-composite and TTS splints (titanium trauma splint), all of which fulfil the demands of modern dental splinting.

In the following, the various types of splints will be described.

**Table 39.1** Comparison of different types of splints. Various properties are assessed using a three-point system. Plus (+) illustrates that the property is strongly related, plus-minus (±) that the property is slightly related and minus (–) that the property is do not exist for the splint concerned.

| Type of splint | Accuracy of reposition | Easily discolored | Flexibility | Rigidity | Easily fractured | Easy to construct | Suitability after dental trauma |
|---|---|---|---|---|---|---|---|
| Suture splint | + | | + | – | + | + | ± |
| Arch bar splint | – | – | – | + | – | – | – |
| Arch bar splint with acrylic | – | – | – | + | – | – | – |
| Flexible wire-composite | + | ± | + | – | – | + | + |
| Rigid wire-composite | + | ± | – | + | – | + | ± |
| Composite splint | + | ± | – | + | + | + | ± |
| Protemp®, Luxatemp® | + | ± | + | – | ± | + | + |
| TTS splint | ± | ± | + | ± | – | ± | + |
| Orthodontic splint | ± | ± | ± | ± | – | ± | ± |

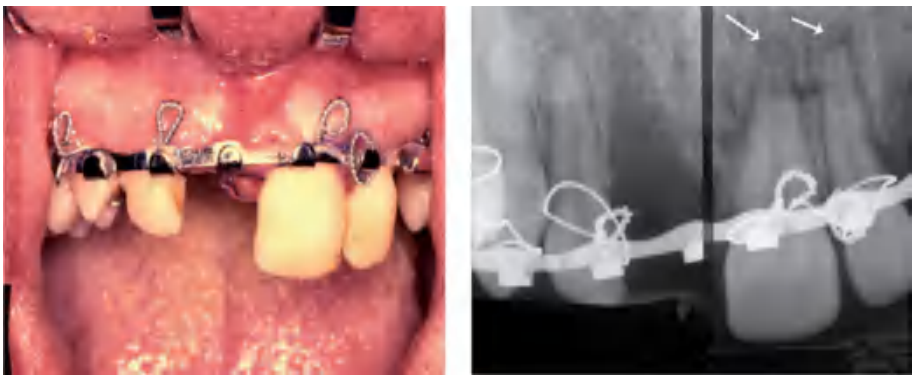


**Fig. 39.1** Displacement of an originally subluxated left central and lateral incisors by an Erich's arch bar (see arrows).

## Suture splint

The simplest type of splint is a suture placed over the incisal edge from the palatal/lingual gingiva to the buccal gingiva. This fixation can be used, for example, in preventing repositioned incisors from extruding, but will only be effective for a short period of time (32–34). After autotransplantation of premolars, the suture is placed over the occlusal surface of the transplant (see Chapter 33). Suture splints have been found to improve the prognosis of autotransplanted teeth compared to rigid splints (11).

## Arch bar splint

Several decades ago, rigid splinting of luxated teeth was considered necessary, and the types of splints used were either arch bars or cap splints. These splints caused considerable damage to the injured teeth, due to inaccurate repositioning, which could press the loose tooth against the socket wall or extrude subluxated teeth (Fig. 39.1). Furthermore, there was risk of bacterial invasion into the periodontal wound due to the close proximity of the splints and wires to the gingival margin (35).

## Orthodontic appliances

Orthodontic ligature wire bonded with composite or attached to brackets has been advocated (36). However, orthodontic bracket wires and composite may cause irritation of oral mucosa, impairment of oral hygiene and discomfort, especially at the start of the splinting period (37). Furthermore, demands for a passive splinting (i.e. with the tooth in a neutral position) are endangered if brackets are united by rectangular orthodontic wires. It is therefore recommended that malleable steel wire is used (38).

## Composite resin

A splint composed entirely of composite resin is esthetic and easy to construct but has been found to fracture in the interdental area, as the material is fragile. The splint is rigid and thereby violates the demands for splinting in most cases. Moreover, due to color match and bonding strength to etched enamel, it is difficult to remove without damaging underlying tooth structure. If a splint of this material must be used, it is advisable to splint the luxated tooth to only one adjacent tooth (39).

## Wire-composite splint

Wire-composite splinting was introduced in 1987 (40) and has since been reviewed (3) and tested both *in vitro* (12, 24, 30, 52) and *in vivo* (39, 51). One of the major benefits is that the splint is constructed of materials that are routinely available in dental offices. Detailed illustration of construction of a flexible wire-composite splint is illustrated in Fig. 39.2. Wire-composite splinting is easily modified into a rigid splint by changing the dimension of the wire or by adding composite along the labial wire up to the interdental space (Fig. 39.2). Examples of the use of wire-composite are shown in Figs 39.3 and 39.4. However, there is the same problem concerning risk of potential damage to underlying enamel as with a composite splint.

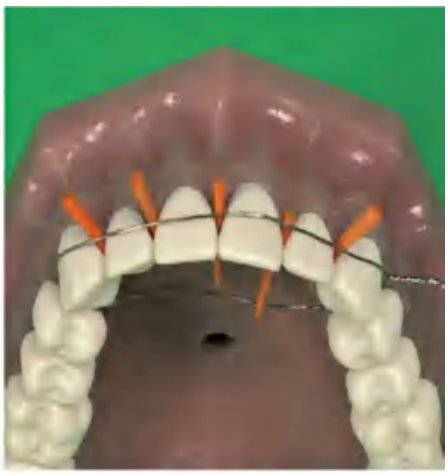
In a recent comparative study of various types of splints in volunteers, a wire-composite splint proved to be well accepted, did not cause major damage to the oral mucosa and allowed the volunteer to maintain good oral hygiene (37).

In several studies the use of fiber glass or fishing line instead of wires has been described and is frequently in use (27, 37, 42–44, 51, 52). Fiber glass ribbon is soaked in composite resin and no filler material is used. Flexibility can be varied with the numbers of layers and extension of the splint (27). Recently the use of power chains has been tested (52).

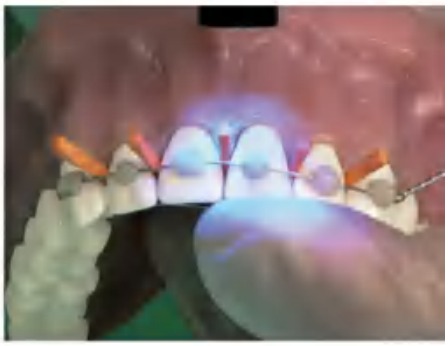
## Resin splint (e.g. Protemp® and Luxatemp®)

Protemp® and Luxatemp® are multi-phase resin materials used in temporary prosthetic restorations and for lining prefabricated crowns. Protemp® is chemically cured; whereas Luxatemp® is dual cured (i.e. chemical and light cured). It is possible to apply the material in stages, an advantage with multiple displaced and repositioned teeth (Fig. 39.5). These materials do not exert forces on teeth during application and are esthetically and hygienically

**Fig. 39.2 Application of a wire composite splint**
(Left) Instruments needed. (Right) Loop of a 0.4mm diameter soft round steel wire is twisted around the injured and neighboring teeth. Dental tooth picks both keep the labial part of the wire to the mid-portion of the crown and at the same time decrease papillary bleeding. It is important to ensure that the dental tooth picks keep the injured tooth in a neutral position and do not displace the loosened teeth. They should not be used to stabilize the luxated tooth.



**Etching enamel and application of composite**
Only a small area on the mid-portion of the crown is etched. A small amount of resin is applied on the surface. Flowing light-cured composite is applied with a syringe. Composite is applied first on non-injured teeth. The luxated tooth is finally included in the splint. The position of the tooth is ensured with finger pressure.



**Composite application and polymerization**
Flexibility of the splint is achieved by leaving some free wire interdentally. Wire is cut with a diamond bur at both distal ends.

**Finished splint**
(Left) The splint is complete after the palatal part is removed and sharp edges of composite polished. (Right) Final splint from frontal view (upper view). The splint can be altered from a flexible into a rigid one by adding composite on the wire and also in the interdental areas as is done here in the right-hand-sided teeth (lower view).

**Fig. 39.3** An extruded tooth splinted with a wire-composite splint.

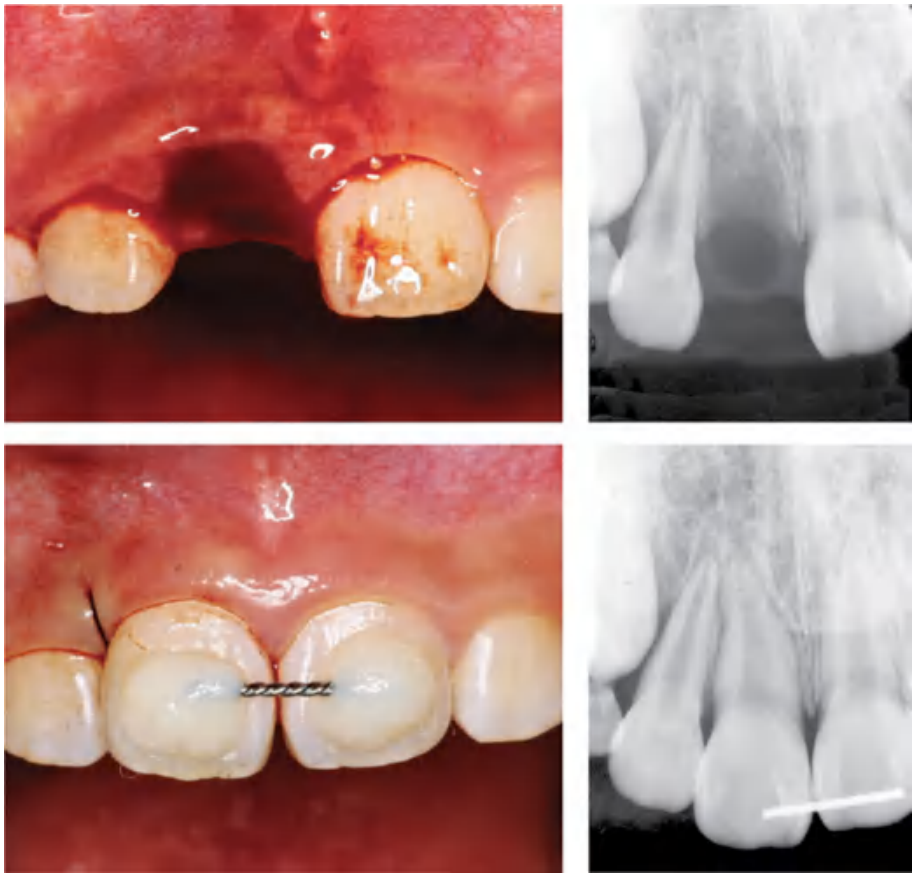


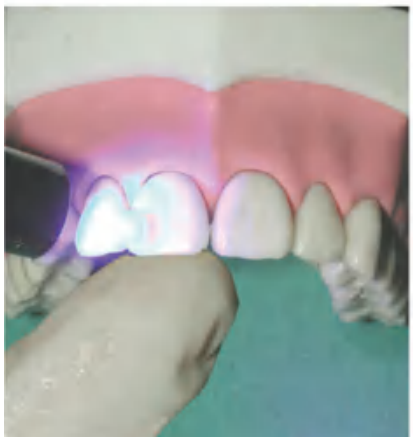
**Fig. 39.4** An avulsed tooth splinted with a wire-composite splint.

**Fig. 39.5 Application of a Luxatemp® splint**
(Left) Instruments needed. (Right) Extruded central incisors repositioned with finger pressure.

**Etching of enamel**
The now repositioned incisors are etched incisally.

**Rinsing and application of Luxatemp®**
The acid etch gel is rinsed away with saline or water and air dried, then Luxatemp® is applied to the laterals and the centrals.

**Curing the splint**
The resin is immediately cured with UV light. During this procedure the tooth is kept in situ with finger pressure. This process is continued until the four incisors are united with the resin splint.



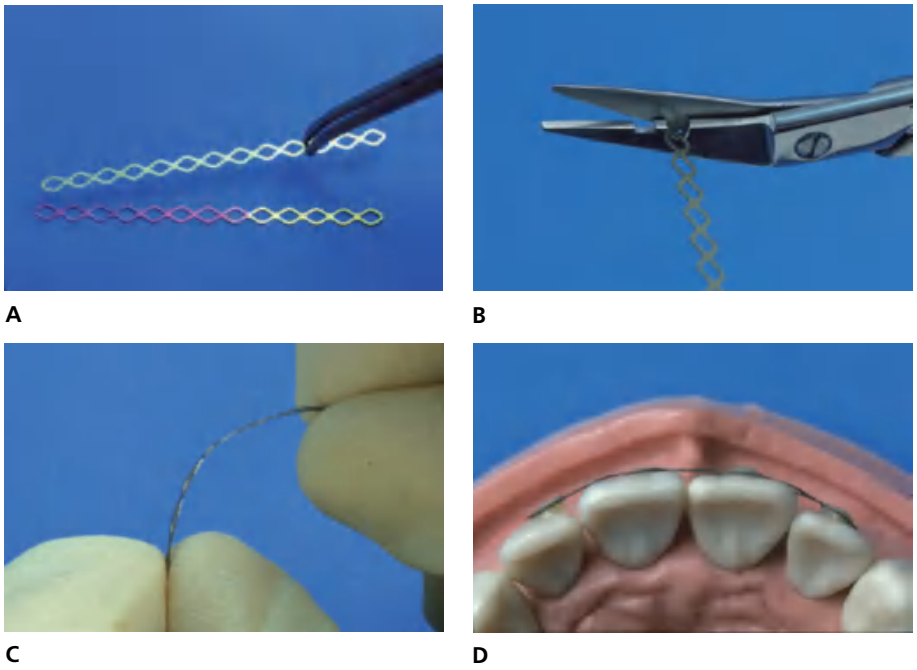



**Fig. 39.6** Construction of a Titanium Trauma Splint (TTS) (Mediartis AG, Switzerland). This is available in two lengths of 51 and 100mm (A). The TTS is cut to the desired length with a pair of scissors (B). Since the TTS is only 0.2 mm thick, it can be easily bent with the fingers (C). A well-adapted TTS is characterized by only small amounts of composite placed into the rhomboid openings (D). Source: Courtesy of Dr. von Arx, Berne, Switzerland.

acceptable. Furthermore, they have been shown to allow semi-rigid splinting (31).

In a case of missing teeth or in a mixed dentition, where neighboring teeth are not fully erupted, it is necessary to span the edentulous area. In these cases, reinforcement is necessary. This can be accomplished with metal bars, orthodontic wires, nylon lines, glass fibers, or synthetic fibers or tapes which are presently on the market (e.g. Kevlar®, Dupont Corp., Fiber-splint®, Polydent Corp., Mezzovico, Switzerland) and which can fuse with resin. If these are not available, even paperclips can be straightened out for the purpose. The material allows some flexibility and the splint is applied directly to etched crown surfaces.

### Prefabricated metal splinting materials

Commercially available dental splints have recently been introduced. Prefabricated splints made of titanium have been reported by von Arx and co-authors (37, 44, 45). The prefabricated Titanium Trauma Splint (TTS) is only 0.2 mm thick and can easily be bent with fingers and adapted to the dental arch. Because of the rhomboid design of the splint, it can also be adapted in length. TTS is bonded to enamel with a light cured composite resin and removed by 'peeling' it off the tooth surface. The splints have been found to be well tolerated and cause only slight patient discomfort (45) (Fig. 39.6).

### Removable splint

Recently a new type of splint has been described where a removable splint made of polycarboxylate and polyacrylic was made after impression taking.

These splints were used in cases where initial reposition and splinting was not considered optimal, resulting in occlusal trauma (45). The indications for this splint appear questionable.

## Recommendations for splinting type and duration

Length and rigidity of splinting is primarily determined by the trauma scenario. In the following, various types of injuries will be described as well as their need for fixation and fixation period (see Table 39.2).

### Extrusion

Extrusive luxation implies rupture of the periodontal ligament fibers. It has been shown that the periodontal ligament achieves approximately 70% of its original strength 2–3 weeks after injury (6). Thus, extrusion represents a simple healing scenario and only 2 weeks of splinting is needed to allow healing of the PDL as well as to keep Hertwig's epithelial root sheath in immature teeth in position adjacent to vital apical tissues to ensure continued root development (see Chapters 2 and 18). A semi-rigid splint is recommended.

### Lateral luxation

Traumatic dislocation in a lateral direction often causes damage to the PDL and bone. Hence the splinting time must be longer than for extrusively-luxated teeth in which the alveolar socket is intact. In these cases, a semi-rigid splint should be used for 4 weeks. Before removal of a splint a radiograph

**Table 39.2** Fixation periods after various dental injuries. In multiple injuries, the length and rigidity is chosen according to the injury which requires the longest or the most rigid fixation.

| Type of injury | Extrusive luxation | Lateral luxation | Intrusive luxation | Avulsion | Root fracture in the cervical third | Root fracture in the middle or apical third | Alveolar fracture |
|---|---|---|---|---|---|---|---|
| Fixation period | 2 weeks* | 4 weeks* | 6–8 weeks | 1–2 weeks | 4 months | 4 weeks | 4 weeks |
| Type of fixation | Flexible | Flexible | Flexible | Flexible | Rigid | Flexible | Flexible |

*Fixation period following lateral luxation starts at 4 weeks, but might be increased, depending upon healing of marginal bone (see Chapter 18).

should be taken. If there are radiographic signs of periodontal breakdown, additional splinting time is necessary (usually 3–4 weeks) (see also Chapter 18).

## Intrusion

Intrusive luxation causes serious damage to the alveolar socket. If the tooth is surgically repositioned, the splinting time must be long enough to support the tooth during remodeling of the bony socket, a process which normally takes 6–8 weeks (see also Chapter 19). To prevent permanent ankylosis a semi-rigid splint should be used.

## Avulsion

The duration of splinting is dependent upon the extent and nature of additional damage to the socket (fracture). Replantation into an intact alveolar socket requires a splinting period of no longer than 7–10 days to prevent ankylosis (see Chapter 20). In cases with incomplete eruption a suture placed over the incisal edge can provide adequate support for an avulsed replanted tooth (34).

## Autotransplantation

Autotransplantation can be considered a well-controlled avulsion but differs in the initial instability due to the expanded socket. In autotransplanted teeth, healing usually takes place without ankylosis and with complete pulp revascularization. A suture placed across the occlusal surface is usually sufficient and has been found to lead to significantly better pulp and periodontal healing than a rigid splint (11, 21). (See also Chapter 3.)

## Root fracture

Root fractures can be located in the apical, middle or cervical third of the root. Displacement of the coronal fragment can be classified as concussion, subluxation, extrusion and lateral luxation (see Chapter 12). Root fractures in the apical third of the root and without coronal fragment displacement do not normally require any splint.

In the past, root fractured teeth have been stabilized by a variety of splints, such as cap splints, orthodontic appliances, bonded metal wires, composites and fiber-glass splints.

Previous recommendations of rigid, long-term splinting for all root fractures have been questioned in recent studies (41, 46, 50). It has been shown that splinted and non-splinted teeth did not differ in outcome of treatment, i.e. whether the healing takes place by hard tissue union, inter-position of connective tissue and/or bone or no healing (see Chapter 15). The type of splinting – cap splint or wire-composite splint – does not influence outcome either in transverse or in oblique cervical root fractures (41). Based on controversial results in recent studies, it has been recommended to splint root fractures located at mid-root for 4 weeks (47). In cervical root fractures, a longer splinting time might be indicated. However, a recent study could not demonstrate an effect in healing pattern of longer splinting time (41).

## Alveolar bone fractures

The principles of treatment of alveolar fractures are related to the treatment of both jaw fractures and tooth luxations. After proper repositioning, splinting is assumed to support both PDL and bone healing. Fixation should be semi-rigid and the splinting period 4 weeks.

## Splinting in primary dentition

In most cases, splinting of luxated primary teeth is not possible due to lack of patient cooperation. In the case of alveolar or mandibular fractures, a resin or a cap splint is indicated. The cap splint should be cemented only on non-traumatized teeth, leaving the injured teeth free within the splint in order not to damage them when the splint is removed (48, 49).

## General comments

Gingival fibers heal within one week, which is enough to provide some stability. Where ankylosis may be a problem (avulsions), a short fixation period may in some cases prevent permanent ankylosis (see Chapters 2 and 20).

In cases where ankylosis is not a significant risk, a fixation period of 2–3 weeks is indicated. During the splinting period, it is essential that good oral hygiene be maintained. Careful toothbrushing and rinsing with chlorhexidine are recommended. When there is an associated injury to the bone, an additional 1–2 weeks is indicated. If remodeling of the socket takes place (e.g. following crushing injuries, as after intrusions and lateral luxation), 6–8 weeks of splinting may be required. Concerning length of the splint (number of adjacent teeth), mobility tests have shown that there is no need to extend the splint to more than one non-injured adjacent tooth to the injured tooth (39).

## Essentials

Splinting may negatively influence:

- Gingival healing
- Periodontal healing
- Pulp healing

### Splinting properties

- *Flexible* and *semi-rigid*: optimal for pulp and periodontal healing.
- *Rigid*: to be used in cervical root fractures and replantation of teeth after PDL removal and fluoride treatment.

### Splinting types

a. Suture splint
b. Arch bar
c. Orthodontic appliances
d. Composite
e. Wire-composite acid etch bonded
f. Resin
g. Metal(TTS)splint

Splint types e, f and g appear to be the most versatile in dental traumatology.

### Splinting periods

- Extrusive luxation: 2 weeks
- Lateral luxation: 4 weeks
- Intrusive luxation: 6–8 weeks
- Avulsion: 1–2 weeks
- Root fracture; middle or apical third: 4 weeks
- Root fracture; cervical third: 3 months
- Alveolar fracture: 4 weeks

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# 40

# Bleaching of the Discolored Traumatized Tooth

J. E. Dahl, S. E. Kopperud & U. Pallesen

This chapter focuses on the treatment of discolored traumatized teeth, most of them being non-vital and subsequently, endodontically treated. Tooth bleaching based upon hydrogen peroxide as the active agent, applied directly or produced in a chemical reaction from sodium perborate or carbamide peroxide, must be regarded as the treatment of choice, alone or preceding a prosthetic restoration. More than 90% immediate success has been reported for intracoronal bleaching of non-vital teeth, but in a period of 1–8 years after treatment, 10–40% of the initially successfully treated teeth needed retreatment.

## Etiology of discoloration

The discoloration of the traumatized tooth is most often a consequence of pulpal response to the trauma (1). Trauma to a newly erupted tooth may cause pulpal bleeding and lead to pink discoloration due to penetration of blood into the dentinal tubules. The dark staining of non-vital teeth is caused by deposition of blood degradation products or necrotic pulp tissue in the dentinal tubules (2, 3). Medicaments used in endodontic procedures like endodontic sealers, mineral trioxide aggregates (MTA) or special antibiotics (eg. minocycline in triple antibiotic paste (TPA)) may also lead to discoloration (4–6). Formation of tertiary dentin can be an effect of pulp inflammation, and the dentin sclerosis and obliteration of the pulp chamber affects the light-transmitting properties of the tooth resulting in a gradual darkening of the tooth. Trauma to the primary dentition may also damage the developing tooth that erupts discolored due to disturbances of matrix deposition and mineralization (7). These types of discoloration are termed intrinsic (1) and may be exaggerated by extrinsic discoloration from aging, coffee, tea, red wine, carrots, oranges, and tobacco (1, 8).

## Medicaments

### Historical overview

The first publication on bleaching of discolored, pulpless teeth appeared in 1864 (9), and since then, medicaments like chloride, sodium hypochlorite, sodium perborate, and hydrogen peroxide, have been used; alone, in combination and with and without heat activation (10). In the walking bleach technique that was introduced in 1961, the bleaching was performed between the visits (11). A mixture of sodium perborate and water was placed in the pulp chamber that was sealed off between the visits. The method was later modified replacing the water with 30 – 35% hydrogen peroxide to improve the whitening effect (12). The use of carbamide peroxide for bleaching teeth was first published in 1989 (13) and was based on an observation made in the late 1960s by an orthodontist who had prescribed an antiseptic containing 10% carbamide peroxide to be used in a tray for the treatment of gingivitis and found tooth lightening as an additional effect of the treatment (14).

### Today's medicaments and mode of action

Tooth bleaching today is based upon hydrogen peroxide (CAS No 7722-84-1) as the active agent. Hydrogen peroxide may be applied directly or produced in a chemical reaction from sodium perborate (CAS No 7632-04-4) (15) or carbamide peroxide (CAS No 124-43-6) (16). Hydrogen peroxide is a strong oxidizing agent through the formation of free radicals (17), reactive oxygen molecules, and hydrogen peroxide anions (18). These reactive molecules attack the long chained, dark colored chromophore molecules and split them into smaller, less colored and more diffusible molecules. Carbamide peroxide also yields urea (16) that

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theoretically can be further decomposed to carbon dioxide and ammonia. It is, however, unclear how much ammonia that will be formed during tooth bleaching using carbamide peroxide. The high pH of ammonia facilitates the bleaching procedure (19). This can be explained by the fact that in a basic solution, lower activation energy is required for the formation of free radicals from hydrogen peroxide, and the reaction rate is higher resulting in an improved yield compared to an acidic environment (18). The outcome of the bleaching procedure depends mainly on the concentration of the bleaching agent, the ability of the agent to reach the chromophore molecules, and the duration and number of times the agent is in contact with chromophore molecules. In Europe, bleaching products are today classified as cosmetic products, not as medical devices. According to EU Council Directive 2011/84/E, products intended for tooth bleaching shall not contain or release more than 6% of hydrogen peroxide and not be used on patients younger than 18 years of age (20). It is the dentist's responsibility that a clinical examination is carried out preoperatively and that the bleaching products are used as intended. Using products with higher concentration of hydrogen peroxide than 6% and treatment on patients younger than 18 years of age may be necessary in some cases. In such cases the treatment must be specially justified even though the safety and efficacy of the treatment are always the responsibility of the dental surgeon.

### Toxicity of bleaching agents

In animal experiments, exposure of the gingiva to 1% hydrogen peroxide for 6 - 48 h resulted in epithelial damage and acute inflammation in the sub-epithelial connective tissue (21). Long-term application of 3% or 30% hydrogen peroxide in the hamster cheek pouch twice weekly resulted in inflammatory changes (22). In clinical trials using 10% carbamide peroxide in custom made trays, 25–50% of the patients reported gingival irritation during the treatment (23, 24).

A single dose of carbamide peroxide in rats induced a concentration dependent toxic reaction, including respiratory depression, reduced weight gain and water consumption, changes in estrous cycle, and, at necropsy, histological abnormalities of the stomach as ulceration, necrotic mucosa and disrupted gastric glands (25–27). Long term administration of hydrogen peroxide has resulted in deleterious localized effects on the gastric mucosa, decreased food consumption, reduced weight-gain, and blood chemistry changes in rats (28), and the formation of small duodenal mucosal hyperplasia in catalase deficient mice that seemed to be reversible during a 6-week recovery period (29). Catalase is a scavenger of reactive oxygen species, and long-term exposure to hydrogen peroxide reduced the plasma catalase levels significantly in rats (30). These results (29, 30) indicate that the toxicity of hydrogen peroxide is mediated through the formation of oxygen radicals.

Several studies of DMBA carcinogenesis in mice skin and hamster cheek pouch indicate that hydrogen peroxide may act as a tumor-promoter (22, 31). In an evaluation of the genotoxic potential of hydrogen peroxide in oral health products, it was concluded that hydrogen peroxide seems to have a weak potential to induce local carcinogenic effects (32). Due to degradation of hydrogen peroxide in the oral cavity, it is unlikely that the use of tooth whitener will represent a cancer risk in persons that do not have an increased risk of oral cancer due to tobacco use, alcohol abuse or genetic predisposition. The risk of oral cancer may increase with repeated treatments with tooth whiteners (32). This is in line with statements from the International Agency for Research on Cancer that found limited evidence in experimental animals and inadequate evidence in humans for the carcinogenicity of hydrogen peroxide (33). The chemicals used for tooth bleaching have a toxic potential, and accidental ingestion may occur (34). However, based upon the amount of the chemicals used for the treatment of single tooth or few teeth, that is the most likely situation in a trauma case, this treatment seems to represent a small or no risk to the patient.

### Effects of bleaching agents on restorations

Bleaching agents can reduce the micro hardness and increase the surface roughness of all restorative materials (35, 36). Glass ionomer cements seem to be more affected than composite resins, and alterations such as cracks and pits in the surface has been shown to occur. Amalgam restorations are the least affected, but corrosion and release of metal ions, including mercury, has been detected. However, the release of mercury has not been shown to exceed the limits defined by the World Health Organization (37). In general, it is questionable whether the changes in surface texture have any clinical relevance (35, 36).

In vivo studies have shown a considerable reduction in enamel and dentin bond strength of resin composite restorations if performed immediately after bleaching (38, 39). This has been explained by the presence of hydrogen peroxide residuals in the enamel that inhibit the polymerization of resin-based materials and thus reduce the bond strength (40). In a previous review, however, (36) it was unclear if the reported changes in surface structure caused by bleaching agents were of any clinical significance. The length of time in which the radicals inhibit the polymerization is a matter of controversy. Lag time from 24 hours (40–42) up to 1–3 weeks (43–45) has been suggested to obtain the best bond strength.

## Intracoronal bleaching

### Methods

Intracoronal bleaching is a conservative alternative to more invasive esthetic treatment of non-vital discolored teeth (Fig. 40.1). Several aspects of the methods have been reviewed recently (3, 34, 46). Careful examination of the teeth is necessary, as the method requires properly obturated

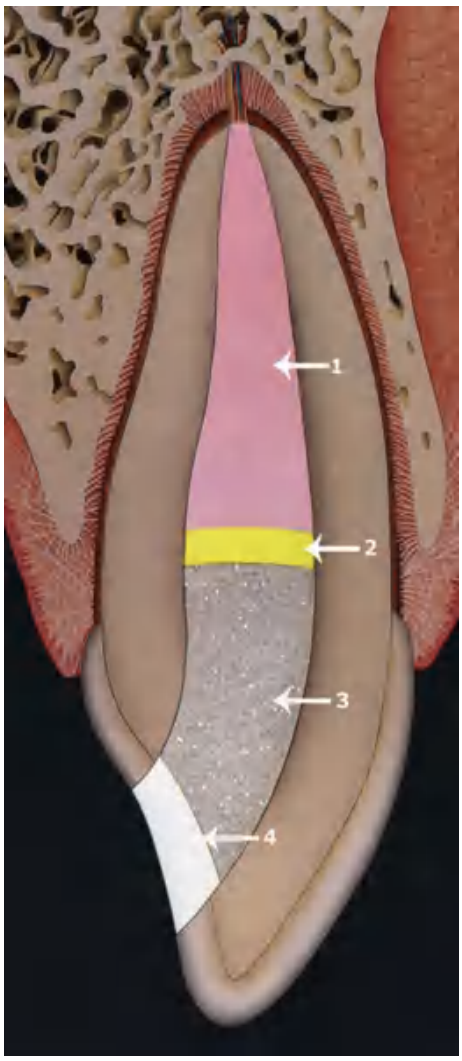


**Fig. 40.1 Internal bleaching – the walking bleach method**
Sketch of a tooth where internal bleaching is being carried out. Arrows at root-filling material (gutta-percha) (1), coronal seal of the root-channel (eg. IRM® or Cavit®) (2), bleaching agent (sodium perborate suspended in water) (3), and temporary seal (eg. IRM® or Cavit®) (4). If the root is discolored it is necessary to remove 2–3 mm of the root filling material from the cervical part of the root canal. The depth can be estimated by measuring the distance between the incisal edge of the tooth to a level about 2–3 mm below the buccal gingival margin. The method is further described in Table 40.1.

root canal to prevent the bleaching agent from reaching the periapical tissues (47). In addition, it is suggested to remove any residual caries and restorations from the pulp chamber and to have a healthy periodontal tissue (47). Discolored dentin is left *in situ*. If the root is discolored, the root canal filling is removed 2–3 mm past the cement-enamel junction; if the filling is not removed to this level, the cervical part of the crown will not be bleached due to the orientation of the S-shaper dentinal tubules. To prevent leakage in apical direction, exposed root filling material is sealed with cement (e.g., IRM®, Cavit® or glass ionomer cement (GC)). Both hydrogen peroxide and sodium perborate have been used, and various heat sources applied to speed up the reaction and improve the bleaching effect (10). A combination of sodium perborate and water (11, 48, 49) or hydrogen peroxide (12) has been employed in the "walking bleach" technique. Carbamide peroxide has been suggested as an alternative (50, 51). The medicament is placed into the pulp chamber and the access cavity is sealed with a temporary filling (e.g., IRM® or GC), that provides an adequate seal without risk of leakage. The bleaching agent is left for 3–7 days. Thereafter the bleaching agent is replaced regularly until acceptable lightening is achieved (Fig. 40.2). For the light yellow or grey discolorations 1–2 treatments may be sufficient, whereas the dark yellow and black discoloration may need 2–4 times or more (49). If the tooth has not responded satisfactorily after 2–3 treatments, the "walking bleach" technique can be supplemented with an in-office bleaching procedure (47). If a slightly brighter color than that of the neighboring teeth can be obtained, account is taken for a possible color relapse over time. Then the pulp chamber and the access cavity is obturated with a tight seal of resin composite or glass ionomer, as further discoloration is likely to be a result of restoration breakdown and uptake of food stains into the tooth rather than being due to chemical reduction of the oxidation products produced by the bleaching itself (49).

A modification of the method that reduces the number of in-office appointments has been suggested as the bleaching treatment is performed at home by the patient (52, 53). Access to the pulp cavity is gained, and the root filling is sealed off with a glass ionomer filling. The patient places the bleaching agent, usually 10% carbamide peroxide, intracoronally with regular intervals and covers the lingual aspect of the tooth with a plastic splint. By this method the pulp chamber is left unsealed during the weeks of treatment. If the seal of the root-filling is insufficient the periapical tissue may be contaminated leading to endodontic treatment failure. Bleaching agent left due to insufficient rinse of the sticky gel could be ingested, and intra coronal dentin is subjected to discoloration from pigments in food or beverage. The saved chair-side time using this bleaching method does not compensate for the risk of adverse biological consequences.

## Efficacy

The efficacy of the different medicaments used for internal tooth bleaching has been evaluated *in vitro* on artificially stained teeth. The outcome of the "walking bleach" method, the thermo-catalytic method, and a combination of the two methods using sodium perborate mixed with 35% hydrogen peroxide, and 35% hydrogen peroxide was compared. No difference was observed between the methods and medicaments (54). The results for sodium perborate mixed with 30% hydrogen peroxide were superior to those of sodium perborate in water (93% vs 53% of the artificially stained and bleached teeth recovered their initial shade) (55, 56). Other studies found no difference in the bleaching efficacy using sodium perborate mixed with 30% hydrogen peroxide, sodium perborate mixed with 3% hydrogen peroxide, or sodium perborate mixed with water (57–59). Increased lightening of the teeth was observed with longer bleaching time. The immediate results after intracoronal bleaching with 10% carbamide peroxide was better than that of sodium

A B

**Fig. 40.2 Internal bleaching**
Result of bleaching with sodium perborate and water. A. Conditions before bleaching. B. A successful aesthetic result after 3 weeks of bleaching.

perborate in 30% hydrogen peroxide, but the final outcome after 3 treatments over 14 days was in favor of sodium perborate mixed with 30% hydrogen peroxide (50). Both 35% carbamide peroxide gel and 35% hydrogen peroxide gel lightened artificially stained teeth to the same extent after 7 days with a somewhat better result for 35% carbamide peroxide gel after 14 days (51). In the same study, sodium perborate-treated teeth lightened significantly less. Another study demonstrated a greater diffusion of bleaching agent through the wider dentinal tubules in young dentin, suggesting a more efficient and effective bleaching of teeth in young patients (60).

The cited *in vitro* studies showed that sodium perborate in water, sodium perborate mixed with 30% hydrogen peroxide, and 10%–35% carbamide peroxide all were efficient in the bleaching of non-vital teeth. This conclusion was based on results from bleaching artificially stained teeth and the efficacy might be different in a clinical situation.

## Esthetic results

The esthetic outcome of a bleaching treatment is a subjective evaluation, and the patient may have a different opinion than the dental surgeon (61). In addition, different terms and definitions of the outcome have been applied in studies making comparison difficult (48, 61, 62). Immediate treatment success has usually been defined as no or slight deviation in color between the treated teeth and the neighboring teeth (Fig. 40.2). More than 90% immediate success rates have been reported using the thermo-catalytic method (10) or the conventional "walking bleach" procedure (48) (Figs 40.3 and 40.4). A recent publication studied 255 internal bleached teeth in 203 patients and found the color modification to be "good" in 87.1% and "acceptable" in 12.9% (49). This study also showed that patient age and tooth type did not affect the outcome, but light yellow and grey teeth responded better than dark yellow and black teeth.

To evaluate the long-term esthetic results of internal bleaching, a determination of treatment failure rate may be useful. Failure was, however, not defined in the cited long-term studies (48, 61–63), but the intuitive definition is teeth that need to be retreated. The need for retreatment increased with the observation time, 10% after 1–2 years (62), 20–25% after 3–5 years (48, 63) and 40% in teeth observed up to 8 years (60). In a more recent study the failure rate was reported to be 7% after 5 years, but the majority of cases in this study were defined as ideal for bleaching (no other filling than the palatinal endodontic opening) (61). In a study of tetracycline-stained teeth that were endodontically treated, internally bleached and followed for 3–15 years, 4 out of 20 patients needed re-treatment (64). At present, there is no good predictor for the long-term outcome of internal bleaching, but for the recommendation that teeth with many fillings are not suitable for the procedure (10, 61).

## Adverse effects

Cervical root resorption (Fig. 40.5) is an inflammatory mediated external resorption of the root, which has be seen after trauma and internal bleaching (62) and sometime followed by ankylosis (Fig. 40.6). Reviews of published case reports on cervical root resorption revealed 28 such cases following internal bleaching (3, 34). A majority of these cases involved teeth with a history of trauma, and trauma has been regarded as a risk factor for development of cervical root resorption after internal bleaching (65). High concentration of hydrogen peroxide in combination with heating seemed to promote cervical root resorption (48, 62), in line with observations made in animal experiments (66–68).

The occurrence of cervical root resorption has also been evaluated in clinical studies. Fifty-eight bleached (30% $H_2O_2$ and heating) pulp-less teeth were followed for 1–8 years, and 4 cases (7%) of external root resorption were observed (62). A study of 204 teeth treated with a combination of thermo-catalytic and "walking bleach" techniques with an observation time of 1–19 years revealed 4 teeth with cervical resorption and all these had a history of trauma (69). Another 95 teeth examined three years after treatment, using the walking bleach technique (sodium perborate in water), revealed no cervical resorption (48). In a four-year follow up of 250 teeth with severe tetracycline discoloration using sodium perborate in oxygen-water as the bleaching agent, no evidence of external resorption was found (70). An analogous study comprising 112 teeth bleached with a paste of sodium perborate in 30% hydrogen peroxide and observed for 3–15 years, reported no external root resorption (64).

Darcey and Qualtrough explored the pathological process involved in dental resorption and suggested that internal bleaching agents could be a predisposing factor (71).

**Fig. 40.3 Long term results of internal bleaching**
The tooth of a 21-year-old woman had been endodontically treated 6 years earlier due to trauma. Discoloration was visible immediately after the endodontic treatment which subsequently became more intense. A. Right central incisor with dark blue discoloration. B. The result after 3 weeks of internal bleaching with sodium perborate suspended in water and a weekly change of bleaching agent. C. Five years after internal bleaching. Only slight discoloration is visible, and no re-treatment was necessary. D. After 8 years the patient still did not ask for re-bleaching. E. Ten years after internal bleaching. Recurrence of the discoloration is visible to the patient who needed re-treatment. The relapse after 10 years was not as severe as the discoloration before the initial bleaching. F. Ten years after initial bleaching the tooth was re-bleached for 2 weeks to a satisfying result. G. Eleven years after the second bleaching (21 years after the first bleaching) the patient needed retreatment. H. Again, the tooth responded positive to a new 2 weeks internal bleaching. Although internal bleaching does not have – as do most other treatments – indefinite durability, the long-term aesthetic results of this non-invasive treatment are considered to be of high quality.

Two mechanisms for the bleaching induced resorption have been suggested; direct initiated inflammation and autoimmune reaction caused by changes in the dentin surface subsequent to diffusion of hydrogen peroxide through the dentin (72, 73). In vitro studies using extracted teeth showed that hydrogen peroxide placed in the pulp chamber penetrated the dentin (74). Heat and cervical defects of the cementum increased the penetration (75–77). Hydrogen peroxide also enlarged the permeability of dentin (78) that may enhance the effects of hydrogen peroxide by repeated exposures.

Tooth crown fracture has also been observed after internal bleaching (79) most probably due to extensive removal of the intra-coronal dentin. In addition, internal bleaching with 30% hydrogen peroxide has been found to reduce the micro-hardness of dentin and enamel (80), especially when the thermos-catalytic methods was applied (81), and to weaken the mechanical properties of the dentin (82).

The general recommendation today is not to heat the bleaching agent in the access cavity (83). In addition, extensive removal of discolored dentin must be avoided to prevent weakening of the tooth. Taking these recommendations into consideration, internal bleaching of the traumatized tooth represents low risk treatment.

## External tooth bleaching

External tooth bleaching may be performed as the only treatment of the discolored traumatized tooth or complementary to internal tooth bleaching. The efficacy for

**Fig. 40.4 Examples of different aesthetic results after internal bleaching with sodium perborate**. Conditions before and 3 years after bleaching. A. Good aesthetic result. B. Acceptable aesthetic result. C. Unacceptable aesthetic result.

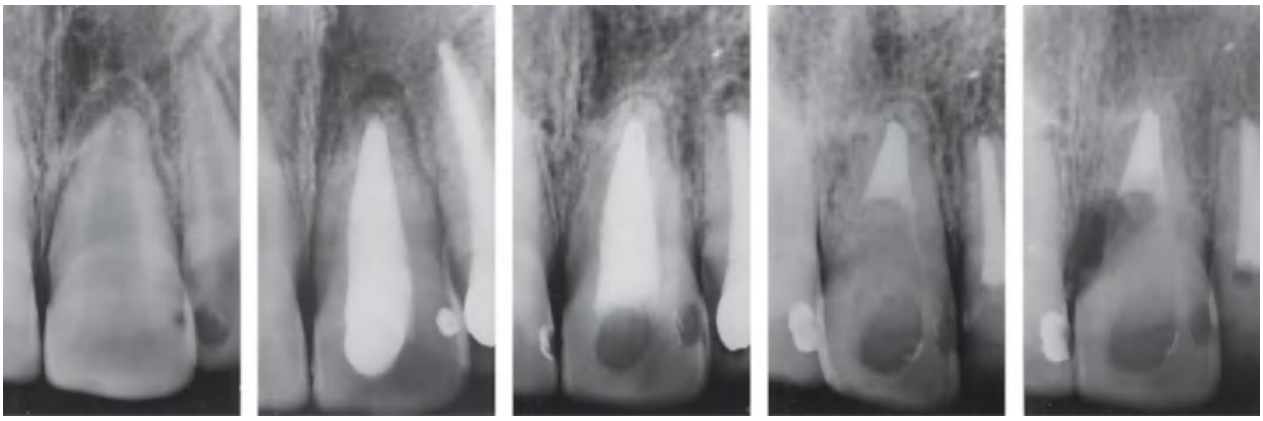


**Fig. 40.5 Inflammatory root resorption after internal bleaching**
Central incisor was bleached with 30% hydrogen peroxide and a light beam from a heat producing lamp. The tooth had previously suffered a luxation injury with subsequent pulp necrosis and root filling. Extensive cervical inflammatory root resorption is seen 1 year after bleaching. Source: Reproduced with permission from (72).

treating discolored traumatized teeth by external compared with internal bleaching has, however, not been evaluated. The most relevant approach for external bleaching of non-vital teeth is in-office bleaching, using high concentration of hydrogen peroxide (30–35%) (83). Care must be taken to secure bleaching only of the traumatized (tooth) teeth to prevent persisting color mismatch to the other teeth. To enhance or accelerate the whitening process, heat-activation of the bleaching agent by light, heat or laser has been promoted. It is, however, still debatable, whether the activation results in superior tooth brightening compared with non-activated bleaching therapies (84). In a previous study

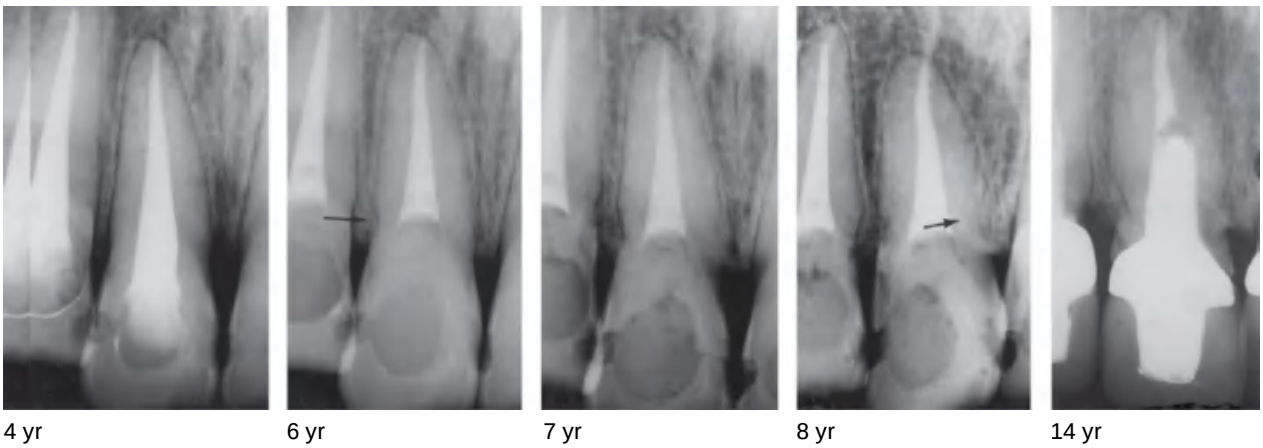


**Fig. 40.6 Ankylosis after bleaching**
A discolored central incisor crown was bleached with 30% hydrogen peroxide. After the first bleaching a cavity with arrested resorption was seen cervically (arrow). After the second series of bleaching 2 years after, a slowly progressing root resorption associated with ankylosis developed in the cervical part of root. Source: Reproduced with permission from (72).

evaluating a combination of irradiation and tooth bleaching agent, the immediate results favored the additional use of irradiation; however, the difference to teeth treated with bleaching agents without irradiation was insignificant after 6 months (85).

## Clinical recommendations

Based on the literature, bleaching of discolored traumatized teeth must be regarded as safe and predictable biological treatment. Bleaching should be the initial treatment in both young and old patients, when a discolored, endodontically treated tooth needs aesthetic improvement. Bleaching may be performed alone or as a supplement to composite or ceramic restoration of the injured tooth. Although the bleaching procedure may need to be repeated over the years, bleaching will also be necessary prior to more invasive and expensive treatments such as laminates or full-crowns if required later in life to obtain the best esthetics. Contraindications for bleaching the endodontically treated tooth are unhealthy periodontal tissue and insufficient root filling.

When discoloration is caused by degradation of necrotic material in the pulp chamber, internal bleaching should always be the treatment of choice. External bleaching only cannot be recommended, as this method gives the bleaching agent less favorable access to the discolored dentin. An example of a recommended protocol for internal bleaching – the walking bleach method – where focus is put on proven efficacy and minimizing side effects, is shown in Table 40.1 and Fig. 40.1. If this method does not lead to a satisfactory result for the patient, it may be supplemented with in-office bleaching, both internally and externally. A recommended protocol for in-office bleaching as a supplement to walking bleach method is shown in Table 40.2.

In teeth with obliterated pulp chambers, the discoloration is not caused by pigments from blood degradation products, but as result of the light transmitting properties of thick dentin, known from yellow canines, and a successful and long-lasting effect of bleaching cannot be expected. Chromophore molecules may, however, be present in the superficial layers of the enamel. External in-office bleaching may thus give a minor effect provided the neighboring teeth are not bleached. To obtain this, the adjacent teeth have to be protected by rubber dam during the bleaching. The procedure in Table 40.2 may be used, omitting the internal placement of the bleaching agent. Long lasting aesthetic treatment of obliterated teeth is seldom possible to obtain without invasive treatment like thick porcelain veneer or full crown.

## Essentials

### Indications

Bleaching of discolored endodontically treated teeth can be regarded as a safe and predictable biological treatment in most cases.

### Efficacy

Internal bleaching has up to 90% immediate success.

### Precautions

Thermo-catalytic bleaching may constitute a risk factor for the development of cervical root resorption.

The use of high percentages of hydrogen peroxide in bleaching procedures may impair the mechanical properties of dentin.

The dental surgeon is solely responsible for safety when using hydrogen peroxide above 6% concentration and when bleaching on patients younger than 18 years of age (according to EU regulations).

**Table 40.1** Recommended protocol for internal bleaching – the walking bleach method – with focus on proven efficacy and minimizing the side effects.

| Steps | Comments |
|---|---|
| (1) Radiographic control | To secure healthy periodontium and complete root filling. |
| (2) Opening | To all areas of the pulp chamber and pulp horns. Discolored dentin shall not be removed. The superficial layers of old approximal restorations can be left in place, but must be non-leaking, and replacement due to lack of color match may be necessary after bleach. |
| (3) Coronal seal of root filling | The entrance to the root channel is normally sealed off with a thin and tight layer of temporary cement (IRM®, Cavit®) or glass ionomer cement. If the root is discolored and needs bleaching, it is necessary to remove 2–3 mm of the coronal gutta-percha in the channel. |
| (4) Preparation of the pulp chamber | The smear layer is removed by phosphoric acid-etch for 10 sec., followed by water spray and dehydration with air and ethanol. |
| (5) Bleaching agent | The pulp chamber is filled with a thick mix of sodium perborate and water, excess water is soaked by cotton pellets, and the mix is condensed by e.g. an amalgam condenser. |
| (6) Temporary seal | A 2 mm layer of Cavit® or IRM® cement (thick consistence) is pressed into undercuts and towards the clean cavo-surface margin. |
| (7) Bleaching time | The bleaching agent is changed weekly 2–4 times, until the tooth is slightly lighter than the adjacent teeth. |
| (8) Restoration of the cavity | The cavity is filled with a dual cured white-shaded resin material bonded to the tooth structure. The white color of the restorative material may lighten the tooth further and makes it possible to distinguish between dentin and resin material if re-treatment becomes necessary. The enamel part of the cavity is filled with a tooth colored light-cured resin material. It is recommended to have the final restoration placed 1–3 weeks after completion of the bleaching. |

**Table 40.2** A short protocol for in-office bleaching as a supplement to walking bleach of the discolored endodontically treated tooth, with focus on minimizing side effects and proven efficacy. Indication for this treatment is teeth where discoloration remains after 3–4 weeks of walking bleach procedure according to Table 40.1.

| Steps | Comments |
|---|---|
| (1) Protection of the patient | Gingival tissue and adjacent teeth are covered of a well-fitting rubber dam, and the patient is told to give information about any pain from the gums during bleaching. The patient's eyes are shielded with safety glasses. |
| (2) Pre-treatment | The pulp chamber and tooth surface is cleaned and dried with air and ethanol. No acid etch is used. |
| (3) Bleaching agent | 25–40% hydrogen peroxide or 35–40% carbamide peroxide gel is applied to the tooth internally and externally. Heating or light devise is not recommended. |
| (4) Bleaching time | Bleaching agent is changed every 10–15 min. The procedure is continued 1–1½ hour and may be repeated after 1–2 weeks. |
| (5) Restoration of the cavity | See Table 40.1. |

## Prognosis

Internal bleaching is normally long lasting in that a minority (10–40%) of the initially successfully treated teeth is in need of retreatment after 1–8 years.

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# Economic Aspects of Traumatic Dental Injuries

U. Glendor, L. Andersson & J. O. Andreasen

## Introduction

Traumatic dental injuries (TDI) frequently occur, especially in childhood and adolescence, with consequences for time and cost (1–6). Though increased research during recent decades has enhanced our knowledge of clinical and biological aspects of tooth and bone healing, the knowledge of spent resources in time and costs on dental trauma has been given little attention. Until now only a few population based studies from Scandinavia, including urban and rural populations treated by hospital and public dental health clinics, have addressed this matter (1–4, 7–10). TDI often affects different tissues in a young and growing individual. Thus, long, time-consuming and costly treatments in childhood will often continue into adulthood.

## Costs of traumatic dental injuries

In a prospective and longitudinal study carried out in Sweden, a total cost (including direct and indirect costs) of US$ 3.3–4.4 million was estimated per million individuals per year in the age interval 0–19 years (4). In Denmark, the annual cost of treatment of TDI ranges from US$ 2–5 million per million inhabitants per year irrespective of age (11). Health care service costs and total costs of the emergency visit and for each subsequent visit in Sweden is presented in Table 41.1. The results indicate that the emergency visit is the most expensive visit concerning both health care service cost and total costs. This probably depends on the character of the emergency visit (unplanned) and also the involvement of parents or other companions, compared with subsequent visits.

### Degree of severity

To calculate resources spent on TDI on a population basis, relevant information must be presented in a simple and representative manner. One of the most important pieces of information is the severity of the injury, e.g. whether the injury is *uncomplicated* or *complicated*. An injury should be regarded as complicated when there is an *increased risk of complications* (e.g. pulp necrosis or root resorption) such as when the pulp has been exposed by fracture or the periodontal membrane has been injured by dislocation of the tooth (12–15) (Fig. 41.1). Thus, each traumatic episode should be grouped according to the most severe diagnosis (9, 16, 17), for example a tooth with intrusion and crown fracture without pulpal exposure should be grouped as a tooth with intrusion alone (10, 11, 18). If there has been more than one traumatic episode, all episodes should be grouped according to the most severe episode (1). The classification into degree of severity (16, 19), and into uncomplicated and complicated traumas (1–4, 6, 10, 11, 18, 20) has been used earlier. Studies have shown that a complicated trauma, especially to permanent teeth, is of major significance with respect to time and costs (1–3, 6, 10, 11).

Estimated average health care service costs for uncomplicated and complicated traumatized primary and permanent teeth are shown in Table 41.2. The results from Glendor et al. (3) are presented per injured patient during a period of two years in the age interval 0–19 years, while Borum and Andreasen (11) presented their results per injured tooth during a period of 11 years irrespective of age, and Wong and Kolokotsa (6) during a median number of eight visits in the age interval 7–18 years. Despite differences in material and methodology between these three studies, it is clear that complicated traumas to permanent teeth are the most expensive. The average treatment costs in Denmark and Sweden correspond well, taking into account that in the

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Swedish study more than one tooth could have been injured per patient during a shorter period with an increase of costs, and that in the Danish study there were probably several re-treatments per injured permanent tooth throughout childhood, adolescence and adulthood. In the Danish study this was a 'pessimistic' estimate. Treatment costs also differ according to treatment regimens thus most treatments in Denmark were performed at the hospital trauma center (University Hospital, Copenhagen), in the United Kingdom at a dental hospital, and in Sweden both public dental health clinics and specialist clinics in hospitals were involved.

**Table 41.1** Health care service cost and total costs in US$ in Sweden of the emergency visit and mean cost of subsequent visits of TDI to primary and permanent teeth in the age interval 0–19 years.

| Dentition | Type of costs | Emergency visit ($) | Per subsequent visit ($) |
|---|---|---|---|
| Primary | Health care service cost | 42 | 35 |
| | Total costs[1] | 85 | 75 |
| Permanent | Health care service cost | 81 | 54 |
| | Total costs[1] | 133 | 81 |

[1] Total costs include direct and indirect costs (for definition see How to present costs, p. 986).
Source: Reproduced from (2, 3) with permission.

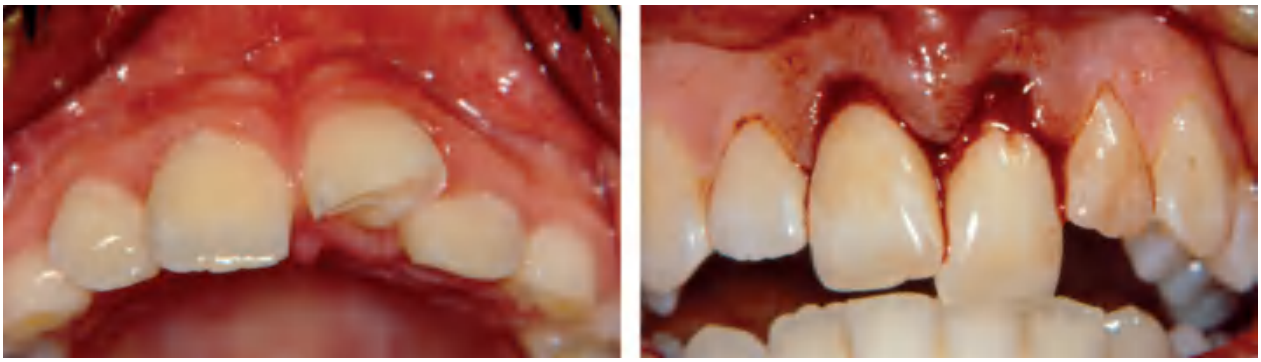
Borum and Andreasen (11) calculated the final treatment (health care service) costs for traumatized primary and permanent teeth in Denmark (Table 41.3). Treatment costs for TDI to primary and permanent teeth vary between dentitions and type of injuries. The acute trauma treatment (health care service cost) of both primary and permanent teeth correspond well with the results from Sweden (see Table 41.1).

### Sports injuries are expensive

TDI in sports can be very expensive. A conservative estimate of the minimum initial cost per dentist's referral for treatment of a serious dental injury in sport is US$ 1,000 per injury (21). Lifetime dental costs of total tooth avulsions have been estimated at US$ 10,000–15,000 per tooth (22). Lang et al. (23) interviewed members of amateur and semi-professional handball leagues regarding their opinion of lifelong subsequent costs for a lost front tooth. The figures varied substantially. On the average, estimates amounted to US$ 10,689 in Germany and US$ 5,373 in Switzerland.

## Time and costs

Resources spent on TDI can be presented in time and costs. The difference between time and costs is that time could be regarded as 'universal' for most countries, given the same



A B

**Fig. 41.1** Uncomplicated (A) and complicated (B) trauma injuries.

**Table 41.2** Estimated average treatment costs in US$ to uncomplicated and complicated traumatized primary and permanent teeth.

| Authors | Year | Country | | Uncomplicated | | Complicated | |
|---|---|---|---|---|---|---|---|
| | | | | Primary dentition ($) | Permanent dentition ($) | Primary dentition ($) | Permanent dentition ($) |
| Glendor et al. (3) | 2001 | Sweden | | 68 | 200 | 114 | 606 |
| Borum & Andreasen (11) | 2001 | Denmark | 'Standard' estimate[1] | 60 | 110 | 200 | 926 |
| | | | Pessimistic estimate[2] | | 420 | | 1490 |
| Wong & Kolokotsa (6) | 2004 | UK | | Permanent incisor: 858[3] | | | |

[1] Survival of the tooth and pulp, and restoration with composite, [2] Pulp extirpation or endodontic treatment, including fixed restoration or extractions,
[3] £1 = US$ 1.65 (in year 2003).

**Table 41.3** Estimated final treatment costs in US$ for traumatized primary and permanent teeth in Denmark treated at a major trauma center.

| | Estimated cost per tooth ($) |
|---|---|
| *Primary dentition* | |
| Acute trauma treatment | 60* |
| Extraction of primary tooth | 35 |
| *Permanent successor* | |
| Composite resin | 50 |
| Crown treatment | 700** |
| Tooth replacement | 2200*** |
| *Permanent dentition* | |
| Acute trauma treatment | 60 |
| Composite resin | 50 |
| Crown or veneer | 700** |
| Endodontics + crown restoration | 1000** |
| Tooth replacement | 2100*** |

* The estimated yearly cost of running the trauma center divided by the average no. of injured teeth treated per year. ** In this amount one composite resin restoration prior to final treatment is included. *** Average for an implant ($2,900), conventional bridge ($2,400) and resin-bridge ($1,000) (62).
Source: Reproduced from (11) with permission.

type of treatment, while costs depend on each country's own market. In some countries treatment costs can be regarded as high, while in others they appear to be moderate. The difference can be due to different welfare systems. Another problem is that it is difficult to present relevant information regarding costs for both patient and society with respect to time. To overcome this, treatment time is more suitable for comparing resources spent both within and between countries. After comparison between countries, time can again be translated to costs for comparison within a country. In Sweden, for example, health care service costs of TDI were found to represent 65% of total costs for the treatment of permanent teeth and 48% for primary teeth respectively, compared to 16% and 11% respectively for treatment time (2, 3). The difference is due to the fact that health care service costs, despite shorter time, are more expensive than companions' costs from loss of production or leisure.

A Canadian study has presented treatment time and cost of permanent incisor replantation during one year (5). Mean treatment time per individual was estimated to be 7.2 hours in the first year and the approximate cost was CAD$ 1465 (US$1 = CAD$11.5 in year 2000). The mean first year cost of a replantation/extraction case including interim prosthesis was CAD$ 1780. Ninety per cent of patients and 86% of parents reported loss of school and work time. Wong and Kolokotsa (6) have estimated the average cost for treating a patient with a traumatized incisor to be US$ 858, taking into account an average of eight visits (Table 41.2). When the cost of the parents accompanying the child to receive treatment was considered, this cost increased to US$ 1,412.

The differences between costs presented in many studies are due to the difference in local costs and also in population material. Some studies presenting costs for TDI are population based (3, 7–10), while others are hospital based (5, 6, 11). The main reason for the high costs in hospital clinics is probably related to the fact that those clinics often receive complicated dental trauma cases on a 24-hour basis and are also often teaching clinics.

## Resources presented in time

Patients with complicated injuries to the permanent dentition have been shown to stay in treatment over a long time, whereas patients with uncomplicated injuries finish treatment more quickly (2). In Fig. 41.2, total time is stratified according to severity of trauma for patients with TDI to permanent teeth. The proportion of patients remaining in treatment for a two-year period following initial trauma is shown for uncomplicated and complicated trauma.

In Table 41.4 treatment time and number of visits per individual of complicated traumatic injuries to the *primary dentition* is much higher than for uncomplicated trauma. This is because treatment of uncomplicated injuries to primary teeth consists primarily of information and follow-ups, while, beyond information, surgery (extraction), sometimes including sedation and follow-ups, is often the result in complicated injuries. In the *permanent dentition*, treatment time and number of visits increase significantly when the injury is complicated, with a spectrum of various therapies and follow-ups, compared to uncomplicated injuries, where restorations and follow-ups dominate (1). The number of follow-ups in uncomplicated injuries to permanent teeth is probably higher than needed with respect to the low risk of complications. A lower number of follow-ups could be considered when planning treatment (1, 10). Almost 50% of all different treatments for TDI to primary teeth are performed in connection with non-trauma-related dental treatment, but only less than 30% for permanent teeth (1). This reflects the unique character of treatment of traumatic injuries to permanent teeth.

Treatment time in permanent dentition has also been found to increase if the first injury occurred before the age of 11 (10). This conclusion, coupled with the fact that there is an increased risk of sustaining multiple dental injuries to permanent teeth if the first trauma episode occurred before the age of 11 (20), highlights the 'pre-teenage child' at special risk of high costs in the event of a TDI.

## Traumatic dental injuries involve several people

Traumatic dental injuries to children and adolescents generally involve one or more professionals and one or more companions, mostly family members, but also neighbors, teachers or sports leaders (2, 3, 5, 24). Only 4 studies to date have analyzed the costs associated with the

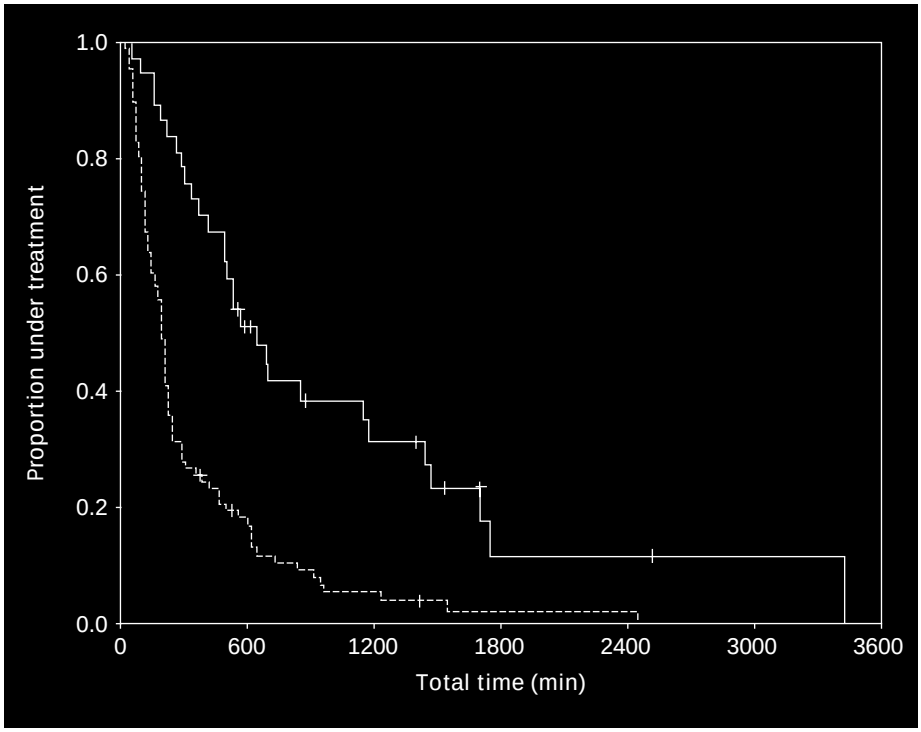



**Fig. 41.2** Total time for permanent teeth, stratified according to severity of trauma. Proportion of patients remaining in treatment for the two-year period following initial trauma is shown for uncomplicated trauma ($n = 86$) ---, and for complicated trauma ($n = 37$) —. Censored observations = +. Source: Reproduced from (2) with permission.

**Table 41.4** The average treatment time in hours and number of visits per individual with uncomplicated (U) and complicated (C) traumatic injuries to primary and permanent teeth in urban (UR) or rural (RU) territories. Treatment is performed by either hospital (HO) and/or public dental health service (PU). Range within parentheses.

| Author | Territory/ caregiver | Dentition | Age years | Type of injury | Period years | Total treatment time | No. of visits |
|---|---|---|---|---|---|---|---|
| Josefsson & Lilja Karlander (63) 1994 | RU, PU | Permanent | 7–17 | – | 10 | 1.2 (0.1–11.2) | 2.9 (1–9) |
| Solli et al. (9) 1996 | UR, RU, HO, PU | Permanent | 6–18 | – | 1 | 0.9 | 1.9 |
| Glendor et al. (1) 1998 | UR, RU, HO, PU | Primary | 2–6 | U | 4 | 0.8 (0.3–3.3) | 2.5 (1–9) |
| | | | | C | | 1.6 (0.4–4.4) | 4.3 (1–12) |
| | | Permanent | 7–18 | U | 12 | 3.2 (0.3–11.0) | 9.2 (1–27) |
| | | | | C | | 8.5 (1.7–20.5) | 16.4 (1–24) |
| Borssén et al. (10) 2002 | UR, RU, HO, PU | Primary | 1–6 | – | 15 | 0.6 (0.1–4) | 2.2 (0–12) |
| | | Permanent | 7–16 | – | 15 | 1.6 (0.3–27.5) | 4.1 (1–41) |
| | | Permanent | 7–16 | C | 15 | 8.0* | |
| Nguyen et al. (5) 2004 | UR, HO | Permanent | 6–18 | C | 1 | – | 9.1 (4–15) |
| Al–Jundi (64) 2004 | UR, HO | Prim & Perm | ? | – | 3 | – | 3–17.2 |
| Wong & Kolokotsa (6) 2004 | UR, HO | Permanent | 7–18 | – | 7 | – | 10.4 (3–27) |

* Children treated by their regular dentist as well as by a specialist had a mean treatment time of 8 h.

involvement of people accompanying patients to dental visits, as well as resources spent, e.g. in transportation, lost working or leisure time and actual costs for damaged personal equipment incurred by traumatic dental injuries to primary and permanent teeth (2, 3, 5, 6). On average, 1.4 (range 0–3) people except health care professionals were involved with each trauma episode, and in 93% of the cases they were parents (2).

## Dental injuries more expensive than outpatient non-oral injuries

Compared to many other outpatient injuries, TDI seems to be more time consuming and costly. The average number of visits treated on an outpatient basis due to TDI to permanent teeth during one year range from 1.9 to 9.1 visits (1, 5, 9), and exceeds the average number of 1.5 visits due to other

accidental bodily injuries also treated on an outpatient basis during one year (25). A comparison of bicycle injuries with oral and non-oral injuries showed that of those with oral injuries, 61% had more than one medical visit, compared to 27% with non-oral injuries (26).

The average cost of treatment of accidental non-oral injuries has been shown to vary between US$ 506 to US$ 1000 in Sweden, the Netherlands and the USA (25, 27, 28). Medical payments per child treated for accidental body injuries in the USA have been estimated to be US$ 800, compared to US$ 506 in a Swedish investigation (25, 27). A study in the Netherlands estimated the total direct medical costs of injury for all ages to be an average of €1000 (1€ ≈ 1US $ in 1997) (28). These 3 studies included inpatient and outpatient care from other non-oral accidental injuries. A comparable figure for outpatient care of accidental injuries for all ages in the Swedish study was US$ 88, which is low compared with the costs of TDI in Tables 34.2 and 34.3. This makes costs of TDI high compared to other bodily injuries, especially for complicated TDI to permanent teeth.

Considering that most studies of costs of TDI have used a relatively short post-trauma period in their reporting methods, and that very few treatments of dental trauma can be regarded as definitive, the costs presented must be regarded as minimum costs. Seen over a lifetime, costs of a dental trauma may rise far over those presented during the last 5–10 years, and in many cases to high levels when including the time spent by the patient and companions in travel and treatment. A six-unit fixed bridge in the front, probably replaced several times during a lifetime, may become an extremely costly treatment (29).

## Other factors contributing to increase in cost

Multiple dental trauma episodes (MDTE) (20, 30–38), and repeated TDI to the same teeth (20, 39, 40) have been shown to jeopardize the ongoing healing of injuries to pulpal and periodontal tissues (41). Factors such as patient information, filling therapy and follow-ups, especially when the same teeth are traumatized again, contribute to increased cost (20) (Table 41.5).

**Table 41.5** Mean values of number of types of treatment per patient in 6–17-year old patients with repeated traumatized permanent teeth related to 2 or 4–7 episodes per patient. Standard deviations in parentheses. Source: Reproduced from (20) with permission.

| Pattern of treatment | Mean values of number of types of treatment | |
|---|---|---|
| | 2 episodes | 4–7 episodes |
| Information | 1.1 (0.3) | 2.9 (1.3) |
| Filling therapy | 0.8 (0.6) | 4.0 (5.4) |
| Endodontics | 1.3 (4.1) | – |
| Surgery | 0.3 (1.0) | – |
| Prosthetics | – | – |
| Consultations, etc | 0.4 (0.7) | 1.1 (1.6) |
| Follow-ups | 2.8 (2.1) | 7.9 (5.7) |
| Total | 6.7 (6.1) | 15.9 (10.1) |

Poor knowledge of how to manage dental trauma among lay people (32, 42–45), physicians, medical students or physical education teachers (46, 47) may also contribute to the high cost of TDI. Other contributing factors may include inadequate emergency care (48–53) and lack of patient satisfaction with the care provided (48, 54), which can result in frequent reconstructions, with extended costs, e.g. of composite fillings (54).

An increase in costs could also be caused by delay in patients seeking treatment (55, 56), and for some dental injuries, e.g. avulsion injuries, this could be an economic disaster, as a good prognosis is highly dependent upon prompt emergency treatment (57).

## Economic evaluations

During the past 10 years, no study addressing costs of TDI on a population level could be regarded as a true economic evaluation. The reason for this is that these studies have not considered clinical outcomes. A true economic evaluation can be defined as 'the comparative analysis of alternative courses of action in terms of both their costs and consequences' (58). The studies presented in this chapter could instead be defined as cost-of-illness studies presenting the economic burden of TDI to a defined population. However, the important benefits of these cost-of-illness studies are that they act as points of reference for economic analyses and provide information for health care policy-makers (59). Cost-of-illness studies should not be confused with other economics studies, such as comparison of different treatments, factors influencing the survival of e.g. resin-retained bridges, cost-effectiveness of dental restorations, cost-benefit of mouthguard protection, and comparison between cost effectiveness of different treatment solutions for anterior tooth loss.

## How to present costs

Assessing costs involves four steps, which are identical in all forms of economic assessment (59). These are:

- Identify the relevant resources used, regardless of whether they can be measured or not.
- Quantify these resources in physical units, such as hospital days, surgical procedures, physician visits, tests.
- Put a value on the different resources used.
- Deal with uncertainty and time (discounting).

Costs can be presented as *direct*, *indirect* and *intangible* costs:

- Direct medical costs (e.g. costs of drugs, analytical procedures required, hospitalization costs, staff time, equipment)
- Direct non-medical costs (e.g. patients' out-of-pocket expenses, transportation costs, community support services)
- Indirect costs (e.g. production losses) due to patients or others being off work
- Intangible costs (e.g. pain, suffering) associated with therapy.

Intangible costs are often omitted from many analyses as they are difficult to measure and value. To measure intangible costs, one must assess the use of quality of life instruments, or direct measurements within the framework of willingness to pay assessments.

To be a true economic evaluation, the outcomes should be measured as health improvements, expressed in one or more natural units (health effects), utilities (preference weights), or associated economic benefits (gains or savings).

## Future research

In times of economic constraints, the need for knowledge of health economics is even more important as a basis for judging health gain in curative and preventive care within dental traumatology as well as assessing epidemiological tools for evaluating the consequences of TDI. Among care providers and insurance companies interest has grown in the total costs of traumatic dental injuries.

A wish for the future would be that economic research in dental traumatology could also include prospective economic evaluations concerning both inputs and outcomes of a dental health care program (58, 60, 61, 64). During recent years, research in dental traumatology has presented evidence-based guidelines on how to manage traumatic dental injuries to both the primary and permanent dentitions. An interesting strategy would be to study those guidelines with respect to how they change time and costs of TDI. Cost-benefit studies or even better, economic evaluations of emergency services, mouthguards and the like should relate the service to cost to the society.

## Cost and longevity of restorations

These two factors are intimately connected, and usually there is an inverse relation. Thus, restorations with a short long-term prognosis are less costly than more expensive restorations which last longer (65–69).

In two studies cost effectiveness of *fixed bridges* and *implants* have been compared, and in both studies the implants came out as the best long-term solution because of a lower failure rate (69). However, one comparative study showed equal cost efficiency of fixed bridges and implants (67).

In a very detailed study the long-term cost effectiveness was compared for tooth replacement of a missing permanent lateral incisor. The following treatment modalities were considered and compared single tooth implant, resin bonded, fixed partial denture, cantilever (FPD), full coverage FPD and autotransplantation of a premolar. An analysis showed the following ranking from the most cost effective to the least: autotransplantation, cantilever FDP, resin bonded FDP, single tooth implants and implant supported crowns and full-coverage FDP (Fig. 41.3) (66).

In another study the cost of treating an avulsed and implanted maxillary central incisor was compared to a situation where the avulsed tooth was not implanted. In this analysis the following treatment solutions were compared: orthodontic closure, resin bonded bridge, fixed partial denture, and single tooth implant and no replantation plus premolar transplantation and restoration. It turned out that replantation of an avulsed tooth was cost effective in relation to a non-replantation situation (Figs 41.4–41.8) (65).

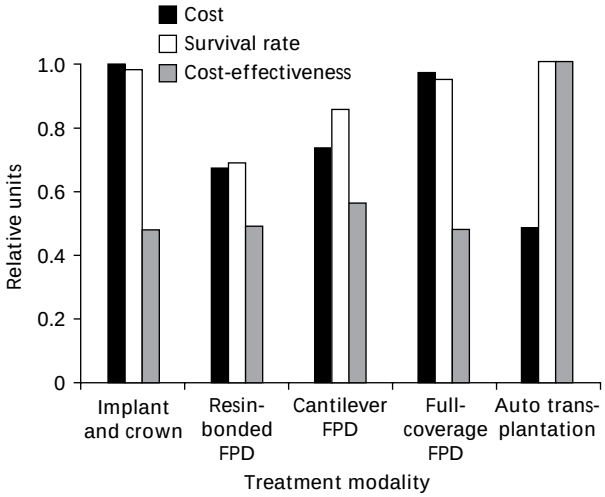



**Fig. 41.3** Relative costs, survival rates and cost-effectiveness. Values are shown in relative units with the most-costly treatment modality represented by a value of 1, the treatment modality with the highest surveil modality represented by a value of 1 (66).

## Essentials

### Degree of severity

- Uncomplicated
- Complicated

### Sports injuries

### Time and costs

- Time is universal
- Cost is unique

### People involved

- Patient
- Companions

### Dental and non-oral injuries

- Outpatient oral injuries

### Other factors increasing cost

- Multiple dental trauma episodes
- Low level of lay knowledge of TDI
- Inadequate emergency care
- Lack of patient satisfaction

### Economic evaluations

- Cost of illness
- Clinical outcome
- True economic evaluation

### How to present costs

- Direct medical costs
- Direct non-medical costs
- Direct costs
- Indirect costs
- Intangible costs

**Fig. 41.4** No replantation, combined cost of orthodontic closure. Cost US$ 10500 (65).

**Fig. 41.5** No replantation, combined cost of temporary treatments followed by an implant. Cost US$ 9000 (65).

**Fig. 41.6** Replantation, decoronation and resin retained bridge. Cost US$ 4700 (65).

**Fig. 41.7** Replantation, decoronation followed by an implant. Cost US$ 8700 (65).

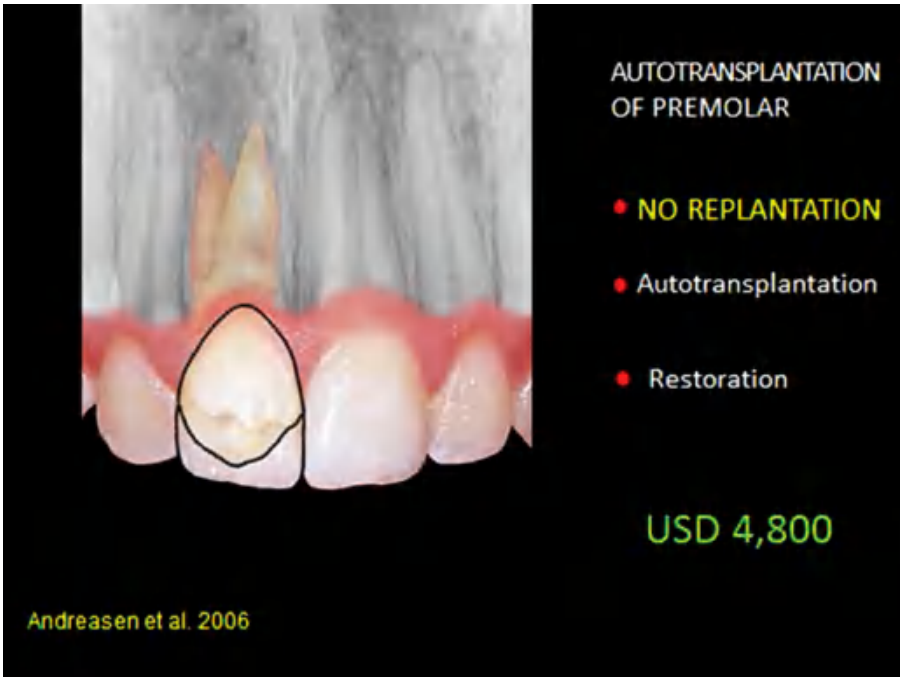



**Fig. 41.8** No replantation, premolar autotransplantation plus restoration. Cost US$ 4800 (65).

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# 42

# Information to the Public, Patients and Emergency Services on Traumatic Dental Injuries

M. T. Flores, M. Al-Sane & L. Andersson

## Introduction

Traumatic dental injuries (TDI) have not yet been widely recognized as severe lesions which carry with them esthetic problems, economic burden, prolonged periods of treatment and sometimes needing psychological support. Self-image may be affected, resulting in low self-esteem and other adverse emotional consequences that go beyond the structural damage to the dentition (see Chapter 9). Moreover, most people fail to grasp the economic impact of some of these injuries because of their high cost of treatment and rehabilitation over a long time (1) (see Chapter 41). Furthermore, those who have suffered the unfortunate experience of a broken or displaced tooth are not usually well informed how this could have been anticipated or prevented.

For many of the injuries the outcome is decided by the measures already taken at the place of accident and the time immediately after the accident. For this reason, it is important that there is knowledge in society on how to manage a TDI at the place of accident. It is also of importance that there are emergency services available and information in the society on how to access such services. In this chapter, we will focus on information to specific groups in the community to which it is important to reach out and to give an overview of methods to inform them.

## Prevention

The urgent need to publicize and promote preventive measures and first aid practices – with the joint objectives of reducing emotional impact, preserving dentition, and reducing short- and long-term costs – cannot be overstated. The rapid advance in knowledge gained through dental research has redirected the professional role toward more preventive and conservative tasks. Hence, the dentist has an ethical duty to provide the community with the necessary information (2), also with regard to TDI to allow each individual to take responsibility for his or her own protection by preventing and limiting damage and improving outcome through appropriate first aid. An example of this is that many TDI could have been avoided if the public (lay people) were aware of activities associated with increased risks, available preventive measures, like the use of a mouth guard in contact sports, and first aid management at the accident site.

Because so little public health education has addressed issues related to dental trauma, educational campaigns focused on the prevention of bodily injuries through accidents (2–6), should also include comprehensive awareness of dental injuries. Campaigns must present their message clearly, making the population aware of its role in saving teeth in case of injury (7). Target groups in this effort would be those exposed to a high risk of accident. Children, adolescents and the adult population should be included in this task. This prevention has been carried out in different ways e.g. picture storybooks (8, 9) (Fig. 42.1), manuals, information included in natural science books, posters, brochures, TV programs, TV and radio talk shows, the local press and multimedia. Because dental injuries may result from so many varied causes, educational campaigns must encourage appropriate first aid and promotion of mouth guards in contact sports and cycling. Even though the use of mouth guards, helmets and facemasks has been widely recommended, there is still insufficient evidence of their effectiveness in reducing the frequency of sports-related injuries to the head, face and mouth (6, 10) (see Chapter 37). Injury prevention campaigns addressing the need for protective devices in sports and bicycling can increase awareness and use. This can rapidly be achieved through legislation or regulation (11).

Several organizations provide information related to dental trauma but there is a high variability in the quality of their contents (12–19). Many web addresses have a commercial interest and information is provided in a direct and

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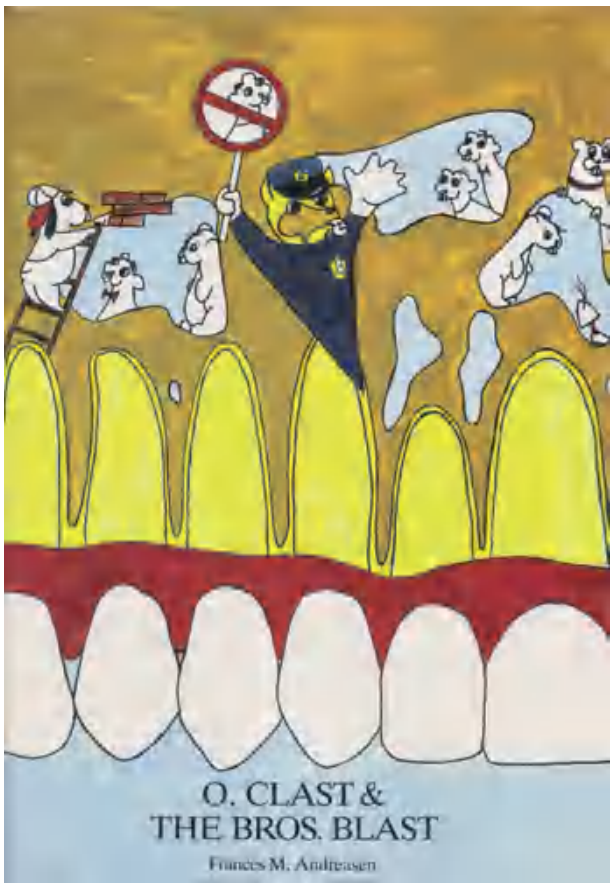



**Fig. 42.1** Children's story books used to inform children what happens after dental trauma.

entertaining manner to the general public. It is necessary to identify instruments to be used to rate the quality and credibility of such information. The American Association of Endodontists (20) and the American Academy of Pediatric Dentistry (21) nowadays endorse and follow the treatment guidelines recommended by the International Association of Dental Traumatology (IADT), which today is considered the gold standard for dental traumatology (22–24).

The prognosis of some TDI are decided by the measures taken already at the place of accident. For this reason, it is important that the knowledge in the society on how to act when TDI is happening.

## Information aiming at increasing the knowledge on how to act at the place of accident

### Who are the target groups?

To be able to increase the knowledge level in society we must first identify the target groups which could be the victims and people close to the victims:

- Lay people in general
- Groups at risk (children, sport players and athletes)
- Parents
- School teachers and sport coaches
- Medical professionals
- Others

#### *Lay people in general*

In recent decades, the level of TDI knowledge among lay people has been the focus of many studies. Hamilton et al. (25), in their study on lay people from North East England, showed that over 80% of their study participants didn't feel confident replanting avulsed teeth due to inadequate knowledge and training.

#### *Groups at risk (children, sports players and athletes)*

School aged children are amongst the high-risk groups for experiencing traumatic dental injuries. This is especially true for 9–10 year old boys, who tend to be frequently engaged in vigorous playing and sports activities (26). In a study on a sample of school children from Kuwait, the authors concluded that the knowledge of first-aid measures of avulsion and replantation of teeth among the study participants was low, despite having a good knowledge on managing other type of body injuries (27).

In addition to school children, contact sports players are often prone to orofacial injuries (28, 29). The literature shows that the knowledge level of this group of first-aid measures and prevention practices is less than optimal. Hersberger et al. (30) in their study on water polo players in Switzerland showed that their study participants had inadequate knowledge of the first aid management of TDI. Approximately 45% of players were not aware of the possibility of replanting avulsed teeth. Other studies conducted on participants of different types of contact sports from Switzerland (31–34); Brazil (35,36); Italy (37); Turkey (38, 39); Malaysia (40) and other countries (41, 42) confirmed such findings.

A survey conducted in Columbus, OH, USA on a cohort of high school athletes, reported that only 12.3% used mouthguards (43). O'Malley et al. (44) assessed the frequency of mouth guard use during sport among national school children in the West of Ireland. Only 22% of their studied sample did actually use mouthguards during sports. Similar findings were reported on sport players from Brazil (35, 36), Turkey (38, 39), Switzerland (30–34), and Italy (37, 45), where mouthguard use was minimal among sport players.

#### *Parents*

A good proportion of TDIs take place at home (26) so this means that parents are likely to be the first respondents to such accidents (46). A number of studies assessed the knowledge of parents regarding the first-aid management of traumatic dental injuries and concluded that the knowledge level was inadequate. For example, Hashim et al. (47) among mothers in United Arab Emirates. Additionally, studies conducted on parents from other parts of the world like Brazil (48, 49), Kuwait (50), India (51–53) and Turkey (54) showed similar findings.

### *Teachers and sport coaches*

School teachers play a key role in the immediate management of TDI because they are often present at the accident site. A number of studies assessed teacher's knowledge in that area. For example, McIntyre et al, (55) in their 2008 study on elementary school staff from North Carolina, USA, found that teachers and teaching assistants had little knowledge about the proper management of TDI, their associated costs and the use of proper storage media. Vergotine at al. (56) in their study on academic and physical education teachers from Milwakee public school system, USA, confirmed this finding and concluded that the knowledge level regarding the immediate management of traumatic dental injuries was low, with only 12% of academic teachers would replant an avulsed tooth versus 7% of the physical education teachers. Available literature on school teachers from Kuwait (57), Brazil (58–64), Norway (65), Iran (66, 67), India (68, 69), Turkey (70, 71) and many other parts of the world (72–74) reported similar findings. Overall the outcome of studies conducted on teachers can be summarized in the following points:

- Teachers tend to have poor knowledge of TDI and their management.
- Many do not feel capable replanting avulsed teeth.
- The inadequate knowledge was prevalent among teachers regardless of whether they work in public or private schools.

A limited number of studies reported on the knowledge of sports coaches with regards to the emergency management of TDI and its prevention. Perunski et al (31) in their study on basketball players and coaches from Switzerland reported that the studied group had a low awareness of the immediate management of traumatic dental injuries. Additionally, less than 1% wore mouthguards while playing. Similar findings have been shown with regards to knowledge of mouthguard use among Nigerian sports coaches (75). The majority of their studied sample had inadequate information about mouthguards. Therefore, they were unable to correctly advice and influence the athletes they are training in that regard.

### *Medical professionals*

Dental emergencies are frequently encountered in emergency departments (ED). Several studies reported on the knowledge of ED physicians of TDI management. They all concluded that ED physicians have insufficient knowledge in that area. Trivedy et al. (76) in their study on emergency department physicians from London and other cities in the UK, reported that over 80% of those physicians were not confident in managing TDI.

Ulusoy et al. (77) reported on the results of a questionnaire administered to ED physicians from 11 different hospitals in Turkey. Close to 40% had insufficient knowledge, and close to 80% expressed the need for more education in that area.

Needleman et al. (78) in their 2013 study, reported on the results of a survey mailed out to ED physicians from Massachusetts Emergency Departments, USA. The study reported that the studied physicians showed variable knowledge levels with regard to the first-aid management of traumatic dental injuries, with their knowledge being heightened for avulsion dental injuries. The study recommended that educational campaigns must be undertaken to improve on the ED physician's knowledge of the first-aid management of TDIs.

Similar findings were reported from Israel (79, 80), Kuwait (81), India (82), Pakistan (83) and Chile (84).

In addition to emergency physicians, school nurses who are often involved in the immediate management of traumatic dental injuries taking place at school. Unlike other groups, school nurses demonstrated a good knowledge of managing TDI. Baginska et al. (85) in their 2012 study concluded that the knowledge of the immediate management of avulsion injuries was sufficient among the study participants. Over 90% were aware that time is critical in the management of these injuries, were aware that avulsed teeth should be held from the crown and had a good knowledge of the proper transport medium. Yet, only half of the surveyed nurses would replant the tooth themselves. The study found strong correlations between knowledge level and participation in a lecture on TDI management conducted 2 years prior to the survey (85).

TDI comprise 2–8% of all military dental emergencies (86–88). Lin et al. (80) in their study on the knowledge of military physicians and emergency medical technicians concluded that this group of medical professionals has inadequate knowledge of managing TDIs.

### *Others*

In some parts of the world firefighters receive special paramedical training. They get to provide first-aid management to victims at the accidents site. They might, therefore, have to manage TDIs. Cardoso et al. (89) assessed the knowledge of the emergency management of avulsion injuries among firefighters with special paramedic training. The study reported that their knowledge was insufficient and recommended educating firefighters about TDIs and their first-aid management.

In summary, it has been clearly documented in the literature that the knowledge of TDI risks, their prevention and immediate management is certainly insufficient among various groups in the society likely to encounter TDI, either by experiencing such accidents themselves or being in a position of having to provide first-aid treatment to TDIs victims. So how can we inform the public to increase the knowledge level?

## Traditional ways to inform the target groups

Several attempts were made to educate the public about TDI risks, prevention and its emergency management at the accident site.

### Lectures

Levin et al. (90) conducted a study on 336 Israeli army recruits, half of which received a short lecture on the management of tooth avulsion. The lecture had a statistically significant positive effect on the participant's knowledge

**Fig. 42.2** 'Save your Tooth' poster available in a number of languages.

of the management of avulsion dental injuries. Al-Asfour et al. (57) on their study on intermediate schoolteachers from Kuwait, also reported on the positive effect of lectures on improving the knowledge of TDI management. Similar findings were also reported by Frujeri et al. (91) in their study on different groups of professionals from Brazil.

## Posters

Lieger et al. (92) looked at the effectiveness of posters on raising the knowledge of TDI and their management among schoolteachers from Switzerland and concluded that posters were effective in raising TDI awareness among the studied population. Young at al. reported similar findings on the effectiveness of posters in improving TDI knowledge among schoolteachers (93) and secondary school children (94). In a study from Iran, using an educational intervention by a poster presentation, Ghadim et al (95) found that the knowledge level of TDI increased among school health teachers when they were asked to respond to a questionnaire one month later. Many countries have made special efforts to reach the public with educational material in the form of brochures, posters and informative campaigns (Figs 42.2 and 42.3). Furthermore, the IADT has developed and collected brochures and posters which can be obtained at the website http://www.iadt-dentaltrauma.org (Figs 42.2 and 42.3).

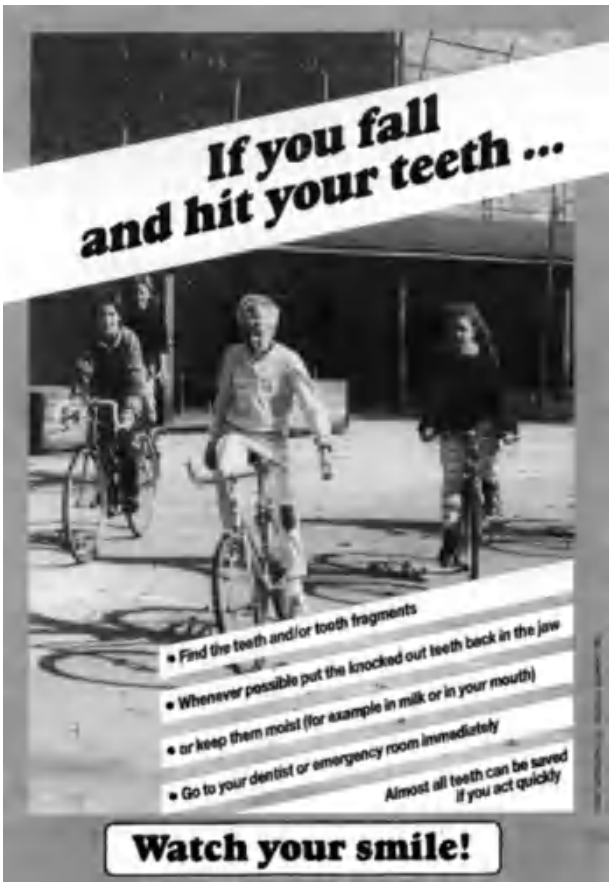



**Fig. 42.3** Poster giving treatment advice after trauma.

## Brochures

Brochures have been used as tools to educate lay persons of TDI and its management. Al-Asfour et al. (96) in their 2008 study evaluated the effect of a leaflet as an information tool to enhance knowledge related to dental avulsions and replantation. A group of 150 parents of primary school children in Kuwait City participated in this study. Half of the parents, served as an intervention group, and received a leaflet with basic first-aid management instructions regarding what to do in cases of tooth avulsions. The other half did not receive any intervention and acted as the control group. One week later the knowledge of the intervention group was assessed using a questionnaire. Results showed that the leaflet had a positive impact on improving the participant's knowledge with regards to avulsion dental injuries and their management. Similar findings were reported from studies conducted on primary school teachers from USA (97), Turkey (70), and Iran (98).

## Other methods

Audiovisual interventions have been successfully employed in educating children about TDI management. Soubra et al. (99) reported on the successful use of movies and storytelling alone or in combination in educating 8–11 year old children on avulsion injuries and their management.

A few studies have reported on using a combination of different educational interventions successfully (97, 100, 101). McIntyre et al. (97), in their 2008 study, reported that reading material alone (pamphlet) is effective in raising awareness of the first-aid management of TDIs among their studied population, However, combining a lecture with reading material gives an improved longer term outcome in terms of knowledge retention. Similar findings were reported by Raoof et al. (101), in their 2014 study, which concluded that a combined educational program based on lectures, discussion session and posters resulted in significant improvement in short term (immediately after the intervention) and long term knowledge retention (36 months later).

These findings indicate that the use of multiple educational interventions can have a synergistic effect on enhancing the outcome. Additionally, the incorporation of lectures and/or discussions increases the chance of interaction during these educational sessions, which may have a positive impact on the efficacy of information transmission and its long-term retention (101, 102).

We conclude that the literature has shown that traditional educational interventions like lectures, brochures and posters are all effective in raising TDI awareness among the groups they were tested on. Lectures and discussions, either alone or combined with other reading materials, however, have shown to be more effective than traditional reading material alone (97–101). This has been attributed to the direct interaction between trauma experts and information recipients, which enhances the effectiveness of the educational experience. Delivering lectures, however, is not cost effective, and requires significant resource investment. Additionally, information delivered through lectures can be forgotten over time. Therefore, lectures and discussion sessions cannot be relied upon when planning nationwide campaigns addressing lay people in the society as a whole. Written information delivered through posters and brochures, although effective, has its own limitations. They may not be thoroughly read, and may carry the risk of being misplaced or even lost altogether. Additionally, they may not be always available at the accident site. Ideally, if knowledge were to be delivered effectively to lay people in the community at large, more practical, cost effective, readily available, easily accessible tools should be considered. This will be discussed in detail in the following sections.

## Role of e-health (the Internet, social media and mobile technology)

Societies are changing rapidly, the Internet, social media and mobile technologies are becoming powerful communication tools. The rapid growth in wireless networks and the marked global increase in the number of smartphone users, have contributed to a worldwide exponential increase in Internet use over the past decade. According to the International Telecommunication Union (ITC), between 2000 and 2015, global Internet penetration grew from 6.5% to 43%. Additionally, in 2011, 3G mobile-broadband signal covered almost 45% of the world's population. By 2015, the coverage has reached almost 70% of the world's population (103). As a result, the Internet has become accessible at low cost to the majority, making it a promising tool for rapid information dissemination.

The World Health Organization (WHO) defines e-Health (electronic health) as the use of information and communication technologies, for health (104). In its broader sense, e-Health is about improving the flow of information, through electronic means to support the delivery of health services (105). The term e-Heath includes the use of the Internet, social media and mobile technology (m-Health) for health purposes.

How effective are these modern communication platforms in information dissemination in public health campaigns? These platforms are interconnect and cannot be separated by clear boundaries. For the sake of discussion, each of these platforms will be addressed individually in chronological order (oldest to newest) assessing their strengths and limitations, as well as the current level of evidence on their effectiveness in information dissemination.

## The Internet

In the last decade, and with the increased popularity of the Internet, access to health information has reached new heights (106). Recently, the Internet has been identified as a preferred source of health information among younger, more educated sectors of the society (106, 107). Hence, the Internet is being increasingly recognized as a medium for delivery of health information, to promote health behavior

change (108). Online based interventions have been successfully utilized in many behavioral health interventions, such as in smoking cessation and in weight loss programs. Chen et al. (109), conducted a meta-analysis on the effectiveness and the cost effectiveness of e-Health based smoking cessation programs. They concluded that compared to conventional interventions, e-Health interventions were slightly more effective, and are likely to be more cost effective.

Although this increase in the dissemination of health-related information holds the potential to empower the public with health-related knowledge, several studies have reported on a number of barriers preventing internet users from making effective use of such information (110). These barriers included inadequate skills to search, evaluate and assess the credibility of online information (110, 111).

The ability for almost anyone to post almost anything on the Internet remains a concern. There is, at the present, no universally adopted means to regulate the content and validity of health related information posted on the Internet (106). For example, Magunacelaya et al. (112) in their study on assessing the quality of online information on mouthguards, reported that the available online information on mouthguards is of variable quality. The majority of the websites included in the study had either wrong or insufficient information on mouthguards.

The effectiveness of the Internet as a tool for information delivery to the public is directly linked to the e-Health literacy level of the public (consumers), which is defined as the ability to conduct the right search and appraise the credibility of the online health information encountered. Peterson et al. (113) assessed the way consumers search for and appraise information on medications on the Internet. Findings of their study indicated that there was a range of search and appraisal skills among their study participants, with many reporting limited awareness of how they found and evaluated Internet- based information on medications. They concluded that educating consumers on how to find and interpret internet-based information on medicines is likely to help with safer and more effective ways of using the health information. Similar findings were reported by Eysenbach et al. (114) in their study on finding and appraising online health information.

But, will health literacy interventions, i.e. teaching people how to search for and appraise health information, lead to improved e-Health literacy levels? And will improved e-Health literacy have a positive effect of health behaviors and health outcome? Car et al. (111) in their 2011 Cochrane review attempted answering these questions. The review concluded that there is currently limited evidence supporting the effectiveness of health literacy interventions and indicated that more high quality studies in this topic are needed to build sufficient evidence. To address the current e-Health literacy gap, health care professional should play a more active role in guiding patients and the public to credible online sources of information. Additionally, they can also suggest appropriate search strategies for individuals interested in broadening their knowledge in specific health related areas (112). The patient information on the International Association of Dental Traumatology (IADT) website, www.iadt-dentaltrauma.org is a good example for a professional initiative to deliver credible health information to the public.

The evidence on effectiveness of the Internet as a health information delivery mode is still insufficient. More research is needed in this area to help further understand the strengths and limitation of the Internet as a source of health information.

## Social media (social network sites)

Social media, also known as online social network sites (SNS) are defined as forms of electronic communication (such as Web sites for social networking and microblogging) through which users create online communities to share information, ideas, personal messages, and other content (such as videos) (115).

In recent years, social media have experienced significant rise in their popularity, particularly with the enormous growth in worldwide use of smartphones. Currently, time spent on social media accounts for almost 25% of all time spent online (116). Additionally, as of September 2013, 73% of online adults were using some kind of SNS, and 42% were using more than one (116). Because of their widespread popularity, and their ability to engage and reach large audience, social media hold tremendous potential as an information delivery tool that can be utilized in nationwide public health campaigns.

Today, among the many other reasons, patients appear to be using the social media to connect with others who have similar medical conditions, and share their illnesses and their management experience (117). Several disease-specific information exchanges exist on Facebook. These modern sources of knowledge, support and engagement have now become important for patients living with chronic disease. The credibility and authenticity of information provided in these platforms, however, appear questionable (118). This remains to be major concern with social media use. Health care authorities and professionals must play an active role in guiding the public to credible sources on social media sites.

But even when health information on social media are scrutinized by health professionals, how effective are these tools in delivering health related messages and promoting health behavioral change? Research in this field is still at its infancy, nevertheless, a number of scholars attempted answering this question. For example, Laranjo et al. (116), in their systematic review and meta-analysis looked at the influence of social networking sites on health behavior change and concluded that social media appear to be effective in delivering information and promoting a change in health related behavior. Yet the evidence is currently insufficient to recommend social media as a trusted mode for health information dissemination. Rice et al. (119), in their systematic review of social networking interventions for the treatment of depression among young adults, concluded that due to the lack of well-designed studies, the evidence on the effectiveness of social media in their tested area is limited.

Maher et al. (120) and Balatsoukas et al. (121), in their review of the literature confirmed that although social media hold great potentials, yet the evidence on their effectiveness is currently very modest.

Social media undoubtedly holds great potentials as a tool in information dissemination in nationwide public health campaigns. To date, our understanding of this tool remains to be limited. Further research is needed to determine how to optimize information delivery, retention, and how to best overcome the information credibility concern that comes with the use of all Internet based information delivery tools.

## m-Health

Mobile health (m-Health) is an emerging term in the literature, describing the use of portable electronic devices, like smartphones, tablet computers and other mobile devices, with software applications (App) for health related purposes (122). WHO defines m-Health is a component of eHealth, that involves the provision of health services and information via mobile technologies such as mobile phones, tablet computers and Personal Digital Assistants (PDAs) (104). Such devices have powerful computing abilities with Internet accessibility, enhanced memories and a diverse range of Apps to download from App stores. Features as such have expanded the range of possibilities and functions of portable electronic devices (106). This is especially true for smartphones, which are currently experiencing worldwide exponential increase in their use. In 2011 up to 10% of the world's population were smartphone users; this is projected to reach almost 36% by the year 2018 (123). Additionally, as of June 2015, more than 100 billion mobile apps had been downloaded from the Apple App Store alone (124).

This expansion in mobile technology presence, use and functions holds promising potentials that can be utilized in health information dissemination.

The above discussion on the role of Internet and social media is directly applicable to smartphones and mobile technology potential role in health information dissemination. In addition to that we will discuss, below, the role of software applications (Apps) in the dissemination of health information.

Currently, countless health Apps are available for smartphone users. Like health information posted on the Internet and social media, many health Apps have no or limited control over the credibility of their content. This poses a serious concern over the kind of health information being communicated to the public. In an effort to address this concern, the National Health Service (NHS) commission board in the UK, piloted an initiative in 2013, the NHS app library. The library employed a peer review process for health related apps, and only those that pass the process are recommended for patient's use http://www.nhs.uk/pages/healthappslibrary.aspx (125). Similar efforts by health care professionals are needed on a much bigger scale, to protect the public and guide them to credible health Apps.

A growing body of literature is highlighting the effectiveness of the app technology in delivering health information and inducing health behavioral change. This is particularly true for self-monitoring apps used in weight loss programs (126) and chronic disease management (122, 127).

Recently, a smart phone App, *Dental Trauma*, has been introduced as a means of disseminating information on management of traumatic dental injuries (Fig. 42.4). This App has been translated into 18 different languages and presents the information in a simple, easy to understand terms for users with no or limited dental background. It is available

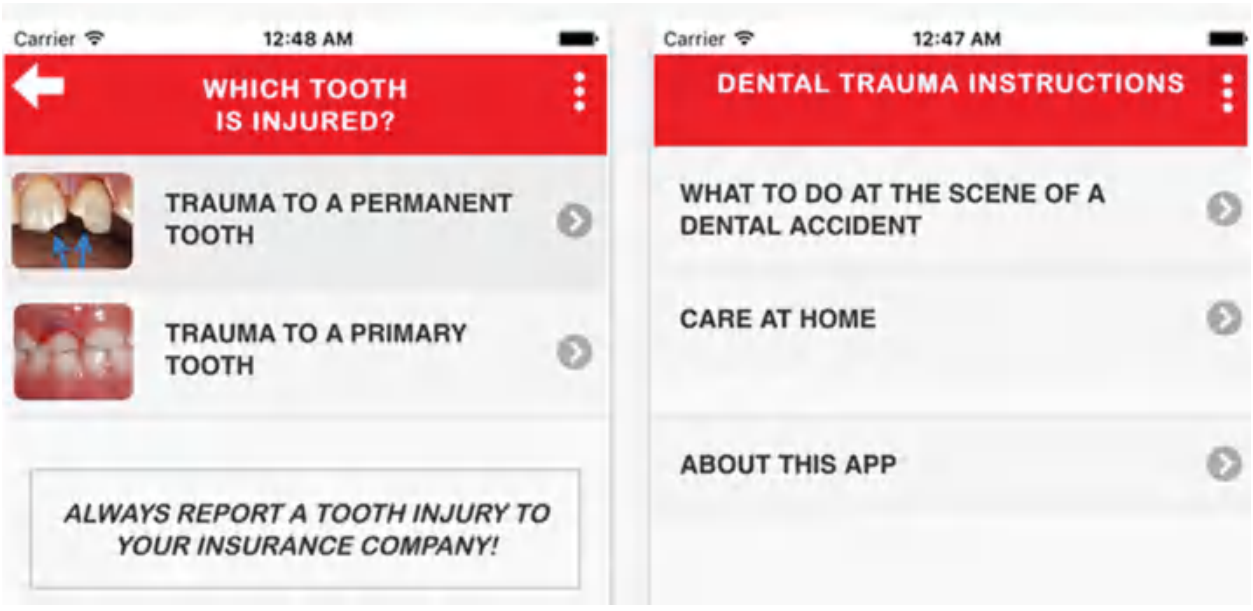



**Fig. 42.4** Dental Trauma is a smartphone app which gives the public instructions how to act at the place of accident.

for Apple and Android phones and can be downloaded from the internet from iTunes Apple (128) and for Android from Google App store (129).

Almusawi et al. (130) attempted assessing the effectiveness of this App in delivering information on the optimal first-aid management of tooth avulsion to a group of school teachers and compared its effectiveness to that of a lecture-based mode of information delivery. The study concluded that the App is an effective information delivery tool. Additionally, its effectiveness is superior to that of lecture-based information delivery (130).

Despite these promising findings, the evidence is still insufficient on the effectiveness of Apps in health information dissemination. More research is needed in this area before Apps can be recommended on a wider scale as a tool for information dissemination in public health information campaigns.

The Internet and mobile technology are widely accessible worldwide. In theory, this provides excellent platforms for health information dissemination in public health campaigns with the potential for improved health outcomes. Currently, however, these platforms are not being widely endorsed. They are being rather perceived as tools with a promising future potential. This is due to two main concerns, the first; is the current inability to ensure the credibility of all health information posted in cyber space and on mobile Apps. The second; is the current insufficient evidence on the effectiveness of these technologies as information dissemination tools in public health campaigns.

Health care professionals have therefore the responsibility of guiding the public to credible sources of information. The introduction on the NHS peer-reviewed library is a good example for protecting the patients and the public (125), http://www.nhs.uk/pages/healthappslibrary.aspx. Additionally, more systematic research on the effectiveness of these technologies in educating the public, and their impact on health care outcomes is warranted.

### How people in the society seek information, and the effect of health information campaigns

In planning health information campaigns, the preference of target population to a particular information delivery mode needs to be taken into consideration. This is likely to enhance their receptivity to the delivered messages. Al-Sane et al. (102) (Fig. 42.5) in their study on lay people's preferred sources of health information on the emergency management of tooth avulsion, found that age and gender as well as other sociodemographic characteristics affected the individual's choice of a preferred source of health information. Additionally, the Internet, health care professionals, and TV were the three most preferred sources of health information across all groups regardless of the sociodemographic characteristics. Hence, health information delivered by healthcare professionals through the Internet, or TV is likely to be effective for the population participated in this study. Several other studies demonstrated a preference of the younger and more educated groups of the society to the Internet as a source of health information (107) (Fig. 42.4). Understanding target populations' preferred sources of health information, their unique sociodemographic characteristics and the influence of that on their choices, is an essential planning step for a successful health information campaign.

## Development of a public awareness campaign

### What message shall be communicated?

The objectives of a dental trauma campaign could be:

- To inform the community about the importance of preventing dental trauma and treating it promptly
- To identify specific methods, appropriate for different age groups, by which dental trauma can be prevented
- To recommend appropriate first aid to be given immediately following dental trauma.

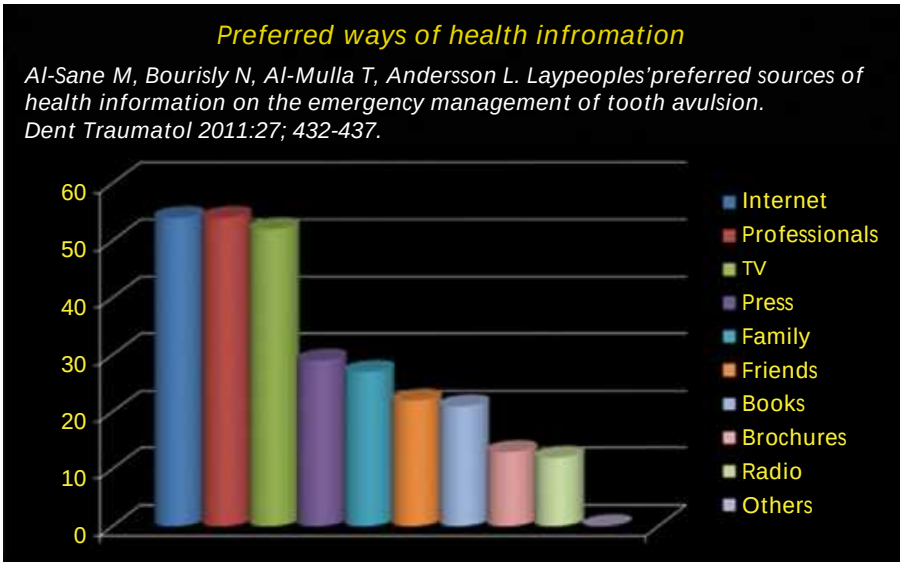



**Fig. 42.5** Preferred ways of health information among people in the society Source: Reproduced from (102) with permission.

## Recommendations for specific groups

### *Dental trauma first aid education in dental schools*

It is important that dental school students receive dental trauma first aid education at the undergraduate level. Dental trauma should be included in dental school curriculum.

### *For government agencies and universities*

Keep records of traumatic dental injuries of patients who attended emergency rooms. A complete registration will give important information for promoting preventive measures. Age, cause of injury, place of injury, diagnosis and emergency treatment offered should be registered.

### *For insurance companies*

Evaluate medical and dental injuries separately and determine treatment costs based on realistic approaches (i.e. worst-case scenario in cases with doubtful prognosis). If various treatment approaches are possible, choose the biologically best procedure with minimal intervention (22–24) (see Chapter 41).

### *For the media, government departments of health and education, insurance companies and accident prevention organizations*

Educational campaigns must be implemented to increase public awareness of dental trauma. Many serious complications can be avoided if people understand that some simple steps may save teeth at the time of an accident.

### *For government departments of health and sports organizations*

Promote the compulsory use of mouthguards in high-risk contact sports (e.g. ice/field hockey, rugby and football) and recommend its use during the practice of skating and cycling. Teach first-aid practices at schools, gymnasiums, sport centers, and other organized athletic groups.

### *Prevention of sports-related dental trauma*

The American Dental Association and the Academy for Sports Dentistry (17, 18) recommend wearing a properly fitted mouthguard while participating in the following activities: acrobatics, baseball, basketball, bicycling, boxing, equestrian events, extreme sports, field hockey, football, gymnastics, handball, ice hockey, inline skating, lacrosse, martial arts, racquetball, rugby, shot-putting, skateboarding, skiing, skydiving, soccer, softball, squash, surfing, volleyball, water polo, weightlifting, wrestling.

Several types of mouthguards are available, but the most comfortable and the most protective devices are custom made for each individual.

Dentists must be aware of which mouthguards are available on the market and their manufacturing process. Likewise, he or she must educate the community as to the importance of their use and promote them actively among sport participants of all ages (see Chapter 30).

### *Guidelines to the public: first aid and treatment of trauma to primary teeth*

Recommendations to the public when responding to dental trauma in young children include the following measures:

- Dental trauma often includes injuries to the adjacent soft tissue. Therefore, wash the wound with plenty of running water.
- Stop bleeding by compressing the injured area with gauze or cotton for five minutes.
- Seek emergency treatment from a pediatric dentist.

### *Guidelines for dentists responding to emergency injury-related visits include the following regarding primary teeth*

- Intrusion, avulsion and severe trauma to the oral region pose the greatest danger to the development of permanent teeth, especially in children younger than 3 years of age.
- In cases of intrusion, the direction in which the primary root is displaced must be determined. In most cases, the root is displaced toward the lip, away from the developing permanent incisor. In this situation, the tooth generally re-erupts spontaneously within 2–4 months.
- In cases of avulsion, primary teeth should not be replanted to protect the child from unnecessary or inappropriate therapeutic procedure (132) and also to avoid damage to the developing permanent dentition. It should be verified that the missing tooth has not been swallowed, inhaled or completely intruded.
- Good tooth hygienic control must be maintained during the healing phase. Parents should be instructed in careful brushing and the topical use of chlorhexidine in the affected area.

### *Guidelines to the public: first aid and treatment of trauma to permanent teeth*

Tooth fractures (broken tooth), luxation (loosened or displaced teeth) and avulsion (complete loss of the tooth) are the most frequent injuries to the permanent teeth. These injuries could have an improved outcome if the public were well informed about appropriate first-aid measures.

### *Guidelines to the public: first aid following crown fracture*

In a crown fracture, the broken piece of tooth may be repositioned using dental adhesives and composite resins. This is a conservative, recommended treatment, especially in adolescents.

- Find the tooth fragment and keep it wet. (Note to the dentist: we know that a tooth fragment is different from an

avulsed tooth. But in the heat of the situation, the lay public doesn't. Therefore, as general information, avulsed teeth and tooth fragments should be stored in a physiological medium (e.g. physiological saline, saliva, milk). However, an enamel-dentin tooth fragment need only be kept wet – and for this purpose, tap water will do.)
- Seek dental treatment immediately.

### *Guidelines to the public: first aid following tooth avulsion*

Often a permanent tooth can be saved through appropriate first-aid response and immediate treatment.

- Keep the patient calm.
- Find the tooth and pick it up by the crown (the white part). Avoid touching the root.
- If the tooth is dirty, place it briefly (max 10 seconds) under cold running water and reposition it. Try to encourage the patient/guardian to replant the tooth. Once the tooth is back in place, bite on a handkerchief to hold it in position.
- If this is not possible, or for other reasons when replantation of the avulsed tooth is not possible (e.g., an unconscious patient), place the tooth in a glass of milk or another suitable storage medium and bring with the patient to the emergency clinic. The tooth can also be transported in the mouth, keeping it inside the lip or cheek if the patient is conscious. If the patient is very young, he/she could swallow the tooth – therefore it is advisable to get the patient to spit in a container – and place the tooth inside. **Avoid storage in water!**
- If there is access at the place of accident to special storage or transport media (e.g., tissue culture/transport medium, Hanks balanced storage medium (HBBS or saline) such media can preferably be used.
- Seek emergency dental treatment immediately.

The poster "Save a Tooth" is written for the public and is now available in 27 languages and is available at the IADT website: http://www.iadt-dentaltrauma.org

## Information to emergency services

TDI occurs at all hours of the day or night. When it happens – at night or week-ends – and the emergency service staff is contacted, precise questions to the patient or parent can pinpoint the problem and help the clinician decide when a trauma must be treated on an acute basis or when it can be delayed. The best way to manage traumatic dental injuries outside dental office hours is to have an organized emergency service with professionals competent in emergency management of TDI (132, 133). A list with telephone number to the dentist on call should be provided at medical emergency services and to telephone operators of emergency telephone services. A poster in the emergency room can serve as a guideline for these queries (see Fig. 42.6). Injuries requiring immediate (*acute*) treatment include: avulsions of permanent teeth, extrusive luxation, lateral luxation and root fractures, fractures of the alveolar process, jaw fractures, gingival lesions and penetrating lip lesions.

- Injuries which do not require acute treatment (i.e. can wait until the next day, or *non-acute*) include: crown fractures, concussions, subluxations, intrusion of permanent teeth. Also, lateral luxation and intrusion of primary teeth (Fig. 42.6).

## Information to patients

When the dentist is faced with an emergency, it is of the utmost importance to give emotional support to the patient as well as to the stressed and anxious parents. Simple explanations of diagnosis, purpose of the emergency treatment and prognosis will be reassuring.

- Inform the patient and/or parents about the consequences of the trauma.
- Instruct the patient and/or parents on the importance of good oral hygiene techniques.
- Instruct the patient and/or parents about the importance of follow-up examinations.
- Advise the patient and/or parents about dental insurance coverage and the need to file a claim immediately after injury. Particularly with very young patients (i.e. primary tooth injuries affecting the developing permanent dentition) and adults (i.e. simple luxation injuries), post-traumatic complications may show up years later. Most companies will not cover an injury if the injury is reported when complications later appear.
- Information concerning specific types of injuries can be provided on a pre-printed form.

### Primary tooth injuries

Inform about possible complications so that treatment can be sought:

- Appearance of swelling, fever or an abscess
- Changes of crown color associated with a fistula.

In case of intrusion, limit the use of pacifiers and feeding bottles in order to allow spontaneous re-eruption of the intruded tooth.

Inform the patient if severe primary tooth injuries might damage the developing permanent successors.

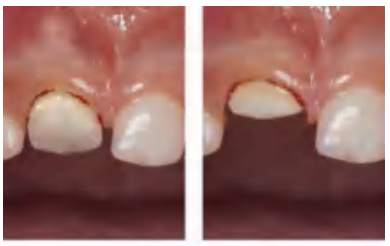
### Tooth fractures

Immediate treatment consisting of reattachment of the tooth fragment will usually be successful; alternatively, a bandage of a white tooth filling material will protect the nerve in the tooth. Later, the tooth can be restored with composite resin

# DUTY INSTRUCTIONS FOR EMERGENCY SERVICES

## CONDITIONS NOT DEMANDING IMMEDIATE TREATMENT

**Crown fracture with exposed dentin** S 02.5*
Exposed yellow dentin is seen. Missing crown fragment should be sought and placed in saline or water until rebonding.



**Lateral luxation and intrusion of primary teeth** S 02.5
The laterally luxated milk tooth is displaced orally (photo to left) The intruded milk tooth is forced up into the jaw (photo to right).

**Crown fracture with exposed pulp** S 02.5
A red and bleeding pulp is seen in the fracture surface.

**Concussion** S 03.2
The tooth is tender to percussion, touch and chewing. The tooth is not loose. There is not change in position and the bite is normal.

**Crown-root fracture** S 02.5
The fracture extends under the edge of the gingiva. There is usually pain from biting.

**Subluxation** S 03.2
The tooth is loose. There is sometimes bleeding from the edge of the gingiva. The tooth hasn't been displaced from normal position. The patient can bite normally.

## CONDITIONS DEMANDING IMMEDIATE TREATMENT

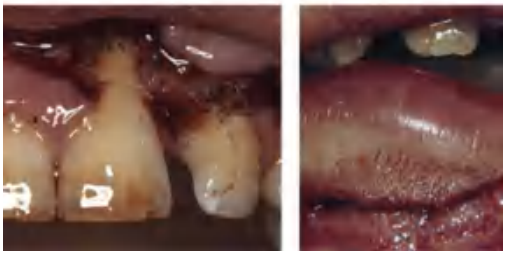
**Extrusion** S 03.2
Note that the tooth is loose and appears longer than adjacent teeth. The patient can't bite together normally.

**Avulsion (permanent tooth)** S 03.2
The tooth is replanted after saline rinse, so that the root surface is clean. This should be done immediately and therefore performed by the physician on duty.

**Lateral luxation** S 03.2
The tooth is angled in relation to adjacent teeth. The patient can't bite together normally.

**Fracture of the alveolar process** S 02.4 (left)
Several teeth are displaced and the entire fragment can be moved by palpation.
**Jaw fracture** S 02.4 (right)
The fracture is exposed to the oral cavity and the patient can't bite together normally.

**Intrusion** S 03.2
The tooth appears too short compared to adjacent teeth. The tooth is firm in its new position.



**Gingival lesion** S 01.5 (left)
The gingiva is displaced and root surface and bone are exposed.
**Penetrating lip lesion** S 01.5 (right)
The teeth have been forced through the lip. Corresponding cutaneous and mucosal wounds are seen. The wound often contains tooth fragments.

**Fig. 42.6** Instructions for emergency room personnel used at the Trauma Center, University Hospital Copenhagen, Denmark. * WHO registration code (see Chapter 11).

or porcelain. In cases where the nerve is exposed, coverage of the nerve will help the nerve heal and allow later restoration of the tooth crown.

### *Duty instructions for emergency services*

#### Conditions not demanding immediate treatment

- Crown fracture with exposed dentin S 02.5* Exposed yellow dentin is seen. Missing crown fragment should be sought and placed in saline or water until rebonding.
- Lateral luxation and intrusion of primary teeth S 02.5 The laterally luxated milk tooth is displaced orally (photo to left). The intruded milk tooth is forced up into the jaw (photo to right).
- Concussion S 03.2 The tooth is tender to percussion, touch and chewing. The tooth is not loose. There is not change in position and the bite is normal.
- Subluxation S 03.2 The tooth is loose. There is sometimes bleeding from the edge of the gingiva. The tooth hasn't been displaced from normal position. The patient can bite normally.

#### Conditions demanding immediate treatment

- Avulsion (permanent tooth) S 03.2 The tooth is replanted with or without local anesthesia, saline rinse, so that the root surface is clean. This should be done immediately and therefore performed by the physician on duty.
- Extrusion S 03.2 Note that the tooth is loose and appears longer than adjacent teeth. The patient can't bite together normally.
- Lateral luxation S 03.2 The tooth is angled in relation to adjacent teeth. The patient can't bite together normally.
- Fracture of the alveolar process S 02.4 (left) Several teeth are displaced, and the entire fragment can be moved by palpation.
- Jaw fracture S 02.4 (right) The fracture is exposed to the oral cavity and the patient can't bite together normally.
- Gingival lesion S 01.5 (left) The gingiva is displaced and root surface and bone are exposed.
- Penetrating lip lesion S 01.5 (right) The teeth have been forced through the lip. Corresponding cutaneous and mucosal wounds are seen. The wound often contains tooth fragments.

## Loosened or displaced permanent teeth

The tooth should be put back in place in the jaw and a splint applied to protect the loosened tooth in the healing period. This splint will be removed after 2–16 weeks, depending on the type of injury (22–24). Chewing is permitted, but hard foods should be avoided. If the splint becomes loose, it should be re-cemented. During the healing period, when traumatized teeth are splinted, the patient should avoid playing contact sports.

In some cases, loosening of the tooth means that the nerve dies. The dead nerve must be removed and replaced by a root filling. The patient should be reassured that this treatment does not hurt.

### *Diet*

A soft diet will be prescribed for the first 14 days, to protect the injured teeth. A soft diet means no hard foods, but not necessarily a liquid diet.

### *Oral hygiene*

Meticulous cleansing of the teeth and gums is necessary to promote healing. It is necessary to show the patients how to perform a gentle and effective tooth brushing, mainly to reduce the anxiety that this procedure may hurt or endanger healing.

Daily oral hygiene includes the following:

(1) Rinse mouth thoroughly with one tablespoon of chlorhexidine gluconate 0,1% alcohol-free twice a day for one-two weeks. For children under 7 years, topical use of chlorhexidine is indicated.
(2) Careful tooth brushing after each meal with a soft toothbrush, working from the gums to the teeth.
(3) After tooth brushing, make sure that splints and teeth are completely clean.
(4) If there are associated lip injuries, recommend lip balm during the healing period to avoid dryness.

### *Follow-up examination*

Some patients will require the first, the day after injury, depending on the severity of injuries. A good recommendation is to see the patient in a week's time, in order to evaluate healing, oral hygiene and control of infection.

Radiographs will be taken 3 or 6 weeks and one year after injury to control healing. Radiographs will allow diagnosis of possible late healing complications, such as root canal infection.

### *Prognosis of treatment*

When treatment has been carried out, the patient and/or parents must receive information about prognosis for the injured teeth.

### *Injury report*

A written report of the accident, including cause, type of injury, treatment and prognosis must be completed for school-related accidents and occupational injuries.

Also, this report will be useful for a court trial when dental injuries are result of violence, assaults or traffic accidents.

## Insurance

Dental needs and dental casualty insurance differ from one country to another. An underlying issue affecting access to care and delivery of services is the cost of care. The dentist should have a general knowledge about dental insurance

coverage following accidents. In many countries there are children without effective dental coverage. Thus, they are at greater risk for experiencing complications of untreated traumatic injuries.

## Essentials

The prognosis of traumatic dental injuries is directly related to the immediate first-aid measures taken at the accident sites. Key individuals likely to encounter such accidents (lay people and medical professionals), have a limited knowledge on the optimal management of TDI.

A number of studies looked at the effectiveness of traditional educational interventions-such as lectures, poster and brochures-in educating those key individuals on the optimal first-aid management. Lectures either alone or in combination with other reading material proved to be more effective than reading material alone in delivering effective messages. The enhanced effect of lectures has been attributed to the increased chance for direct interactions and the possibilities of engaging in a question and answer type of discussion with the health professionals delivering such lectures.

Lectures and discussions are effective in small group settings. When planning nationwide health information campaigns on the optimal management of TDI, however, lectures and discussions may not be the best information delivery mode to use. They require significant resource investment, which raises cost effectiveness concerns on their use in such broad settings.

e-Health whether it is in the form of internet use, or more recently in the form of utilizing mobile communication technologies, offer a promising potential for information dissemination at the broad nationwide scale. The level of evidence supporting e-Health effectiveness is currently very limited. More studies are needed to develop an evidence-based approach to the effective utilization of such platforms for information dissemination in nationwide campaigns.

### Developing public dental trauma awareness

- Dental injuries demand long time treatment, high costs and emotional support.
- Dentists should agree on a standard first-aid message to be delivered to the public.
- According to high risks groups, first aid information should be continuously promoted.

#### *High risks groups and etiology*

- Children 18–30 months
- Adolescents
- Young adults

As long as the public is well informed about dental trauma, the prognosis of injuries may be improved, as correct first aid of avulsed teeth and retrieval of tooth fragments may be performed prior to the patient reaching the dental clinic.

### First aid and treatment of trauma to primary teeth

- Wash the wound with plenty of running water. Often, dental traumas include injuries to the adjacent soft tissue.
- Stop bleeding by compressing the injured area with gauze or cotton for five minutes.
- Do not replace a primary tooth that has been knocked out.
- Seek emergency treatment from a pediatric dentist.

### First aid for a crown fracture

- Find the tooth fragment and keep it wet.
- The broken piece of tooth may be repositioned using dental adhesives and composite resins.
- Seek dental treatment immediately.

### First aid for an avulsed tooth

- Find the tooth and pick it up by the crown.
- If the tooth is dirty, place it briefly (max 10 seconds) under cold running water and reposition immediately.
- If this is not possible, place the tooth in a glass of cold milk. The tooth can also be transported in the mouth, keeping it inside the cheek or lip if the patient is conscious. **Do NOT store the tooth in water.** This will damage vital cells on the root surface.
- If available, place the tooth in special storage media.
- Seek emergency dental treatment immediately.

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# 43

# Dental Trauma Guide

J. O. Andreasen, E. Lauridsen & T. A. Gerds

Evidence-based treatment is the goal in all dental and medical disciplines. Several surveys have documented that significant progress has been made in a number of pedodontics, periodontics and orthodontic disciplines (1). In dental traumatology on the contrary, very few treatment procedures are based on classical randomized clinical studies (2–15). This unfortunate situation is caused by the very sensitive nature of asking children and/or parents about an experimental procedure in an acute treatment situation.

Presently almost all of our knowledge about evidence-based treatment procedures relate to animal experimentation, especially using dogs and monkeys. A series of articles on this topic has shown that dogs in some experiments may demonstrate a more favorable healing, whereas monkeys seem to come closer to healing events in humans (16).

These unfortunate circumstances led to the situation where we have to adopt *best clinical practice*. This concept is presented currently by a group of dentists with a long experience in traumatic dental injuries. Since 2001 a group of dental traumatologists under the auspices of the International Association of Dental Traumatology (IADT) has developed a set of treatment guidelines which are updated every 4–5 years (Chapter 44).

The multiplicity of trauma types makes treatment selection difficult. When it is considered that we have 9 different fracture types and 6 luxation types – and these types can be combined i.e. $6 \times 9 = 54$ treatment scenarios and each of these scenarios has its own treatment need and prognosis – the problem of finding a treatment which will optimize healing becomes tremendous. The final fact to bear in mind is that treatment needs are significantly different in the primary and the permanent dentition, the result being that we have to care for more than 100 different treatment scenarios which makes dental traumatology the most difficult discipline in dentistry (17).

How is the quality of previous dental trauma treatment offered? In 2006 we examined the literature worldwide concerning the proportion of dental trauma patients who received treatment assuring or assisting pulp and periodontal healing. To our big surprise 17 studies showed that only half of the patients received proper treatment, whereas the other half received treatment with either no or even damaging effect (17).

A conclusion of this analysis was that the dentist in charge did not have access to proper treatment guidelines when an actual treatment was instituted.

A possible solution could be to place information of proper treatment for the multitude of different traumas on the internet so that a dentist within few minutes could be familiar with the present IADT treatment guidelines.

## www.dentaltraumaguide.org (Fig. 43.1)

This internet project was started in 2006 based on the large database of trauma treatments collected over 50 years at the Department of Oral and Maxillofacial Surgery at the University Hospital (Rigshospitalet) in Copenhagen, Denmark (17–19). Close to 4,000 patients have been in long-term follow-up in order to register and treat healing complications.

All patients have been treated according to IADT treatment guidelines and all patients had documentation of their injury including 3 photos of the injury and 4 radiographs of the injured teeth plus a standardized patient record (18). Furthermore, a clinical and radiographic follow-up of 1 year or more (17).

On the internet the newcomer can be helped to get to a correct diagnosis by using the trauma type pathfinder (Fig. 43.2).

*Textbook and Color Atlas of Traumatic Injuries to the Teeth*, Fifth Edition. Edited by Jens O. Andreasen, Frances M. Andreasen and Lars Andersson.
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**Fig. 43.1** Front page of dentaltraumaguide.org.

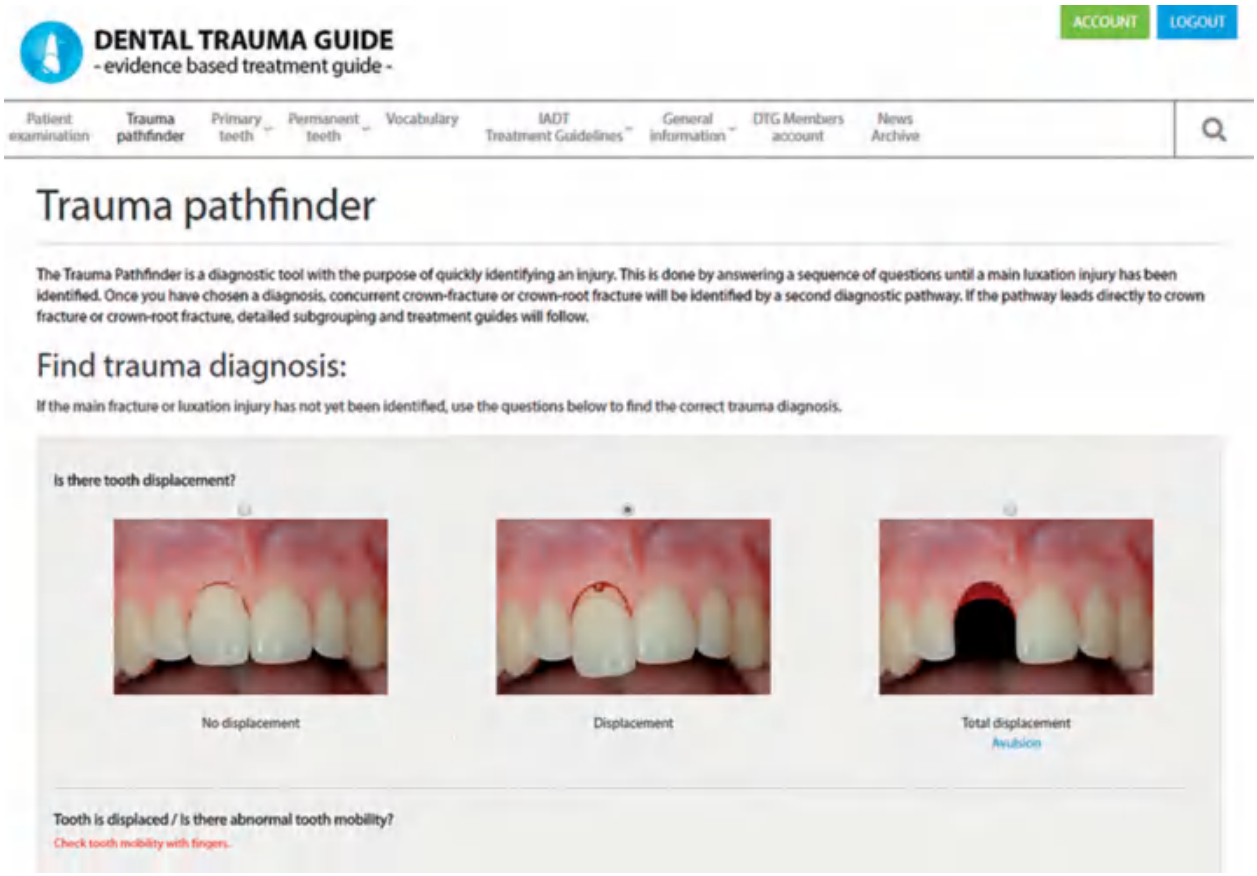


**Fig. 43.2** Trauma pathfinder.

When the diagnosis is established the user will select the primary or permanent dentition and the correct type of injury. When the injury type is selected there is a choice between *etiology, examination, treatment and prognosis*.

The section etiology presented in two planes illustrates the potential damaged tissues (Fig. 43.3). The treatment section illustrated in animated sequences in details how to perform the acknowledged IADT treatment procedures plus suggested follow-up (Fig. 43.4).

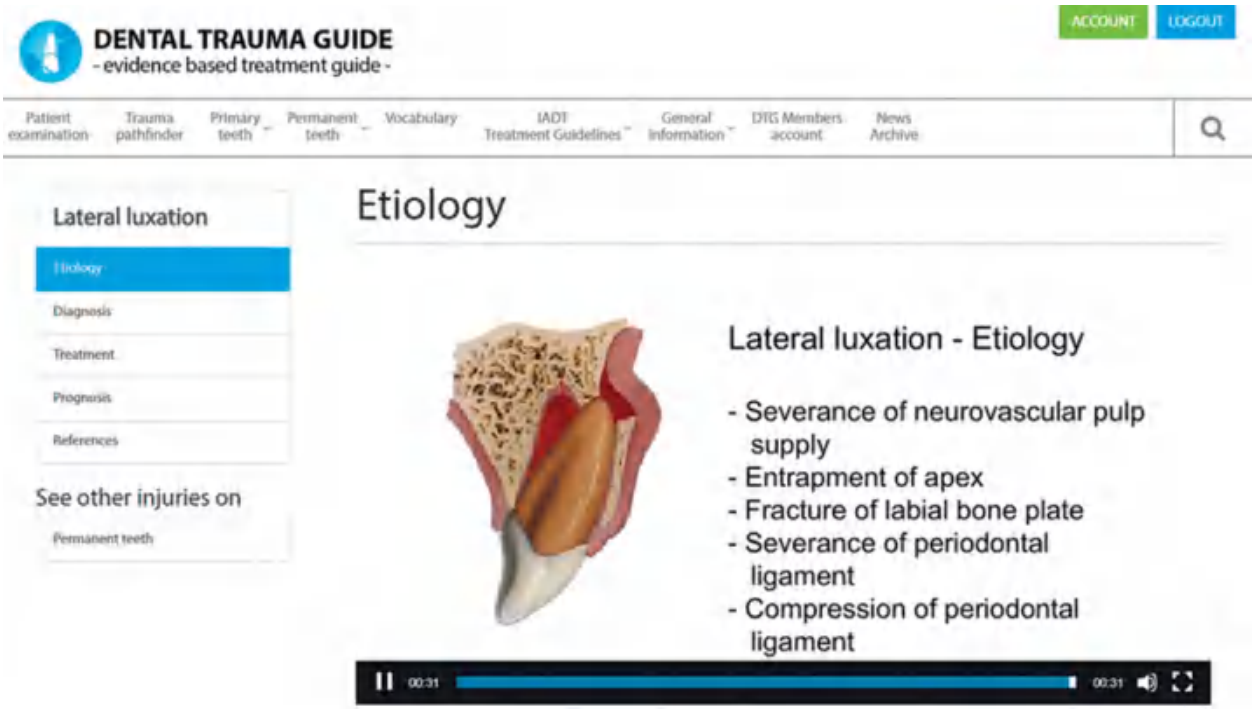


**Fig. 43.3** Etiology of lateral luxation of a permanent tooth with involved tissues.

**Fig. 43.4** Treatment of lateral luxation.

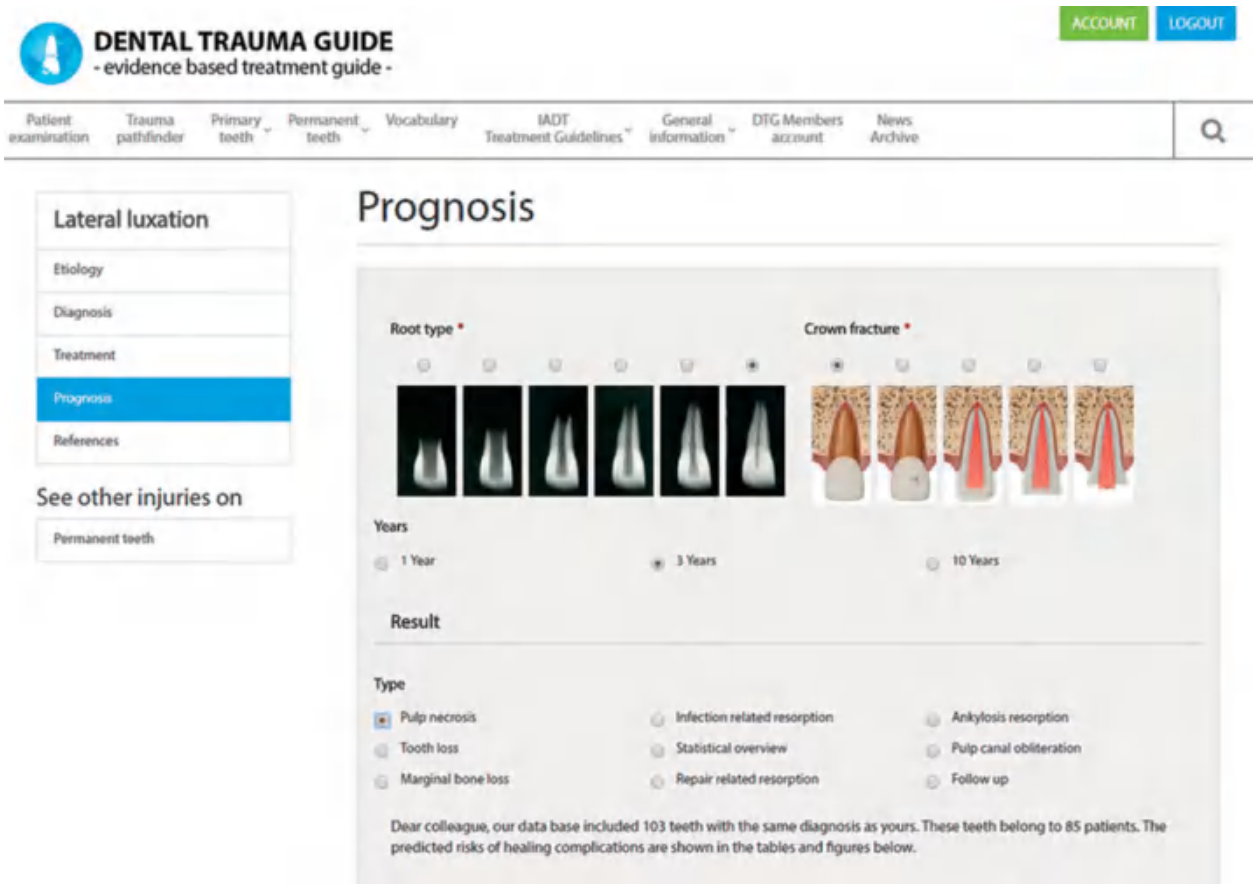


**Fig. 43.5** Prognosis for lateral luxation of a permanent tooth.

In the prognosis section certain predictors for complications are included (Fig. 43.5) (see also Chapter 4). These predictors have been found to have a very strong influence upon the risk of various complications. By using these predictors, the DTG becomes an interactive tool which can predict the risk of *pulp necrosis, repair related resorption, infection and ankylosis related resorption* as well as *marginal bone* and *tooth loss* in an actual case. On the timeline it can also be estimated when these complications will most often become diagnosable (Fig. 43.5).

In the last section of each injury type a bibliography is given on the clinical studies being included in the DTG for prognosis evaluation of the actual trauma type (20) (Fig. 43.6).

Students' use of the Dental Trauma Guide has been tested in a 3D model of various trauma scenarios and it showed that the use of the guide resulted in significantly more correct diagnosis and treatment scenarios compared to students who do not have access to the guide (21).

The DTG is used in 180 countries and it is used more than 2000 times daily (Fig. 43.7).

In January 2017 the DTG was updated to version 2.0.

## Essentials

### Background

- Half of trauma treatment offered today does not assist pulp and periodontal healing.
- International Association of Dental Traumatology (IADT) has published various recommended treatment procedures for all trauma types.
- In 2008 www.dentaltraumaguide.org was launched on the internet to help dentists find the correct diagnosis and the right treatment for the more than 100 different possible trauma scenarios. The basis material for this guide consists of 4,000 dental trauma patients who have been in long-term follow-up to register all types of complications ranging from pulp necrosis, various types of resorption and marginal bone or tooth loss.

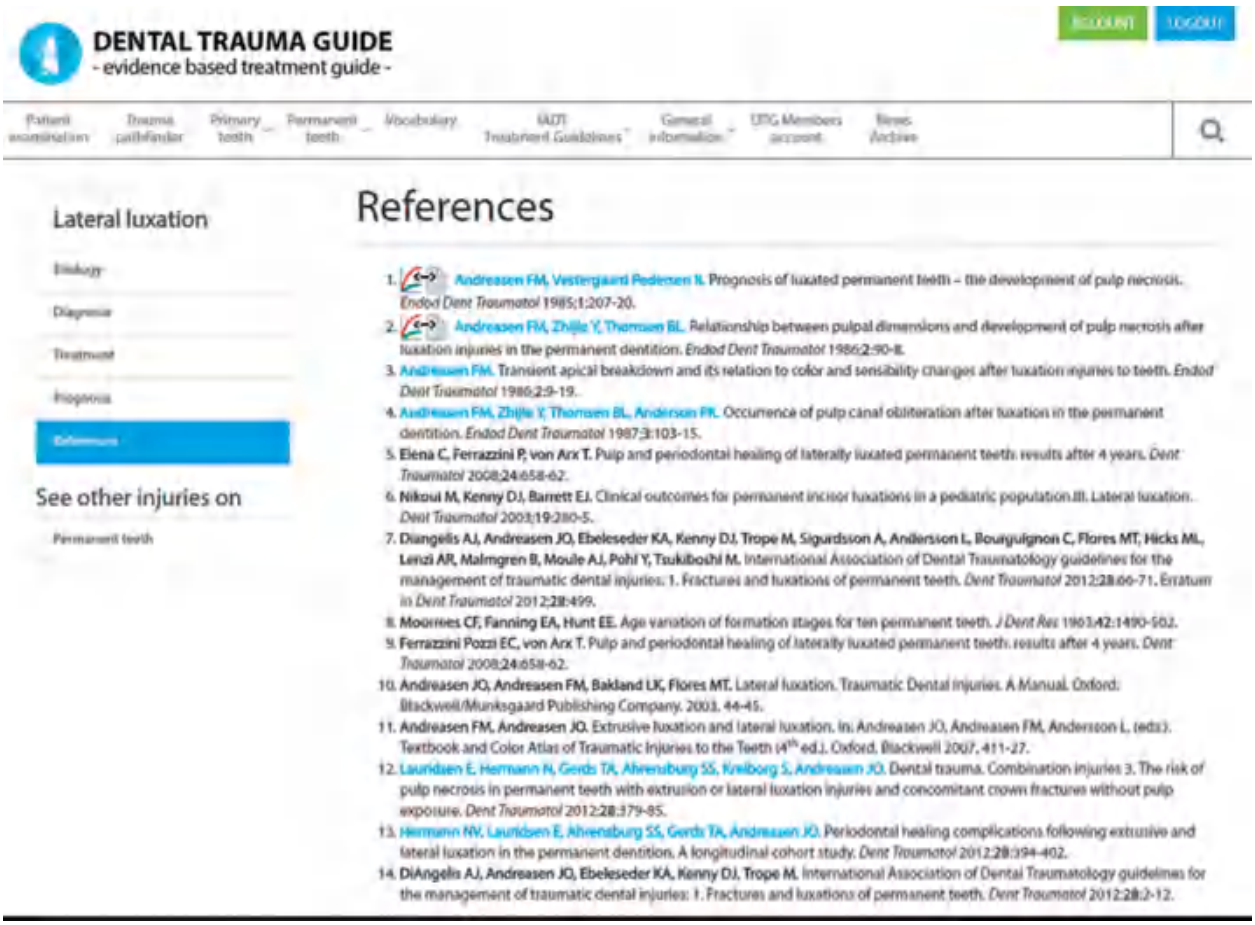


**Fig. 43.6** References related to lateral luxation.

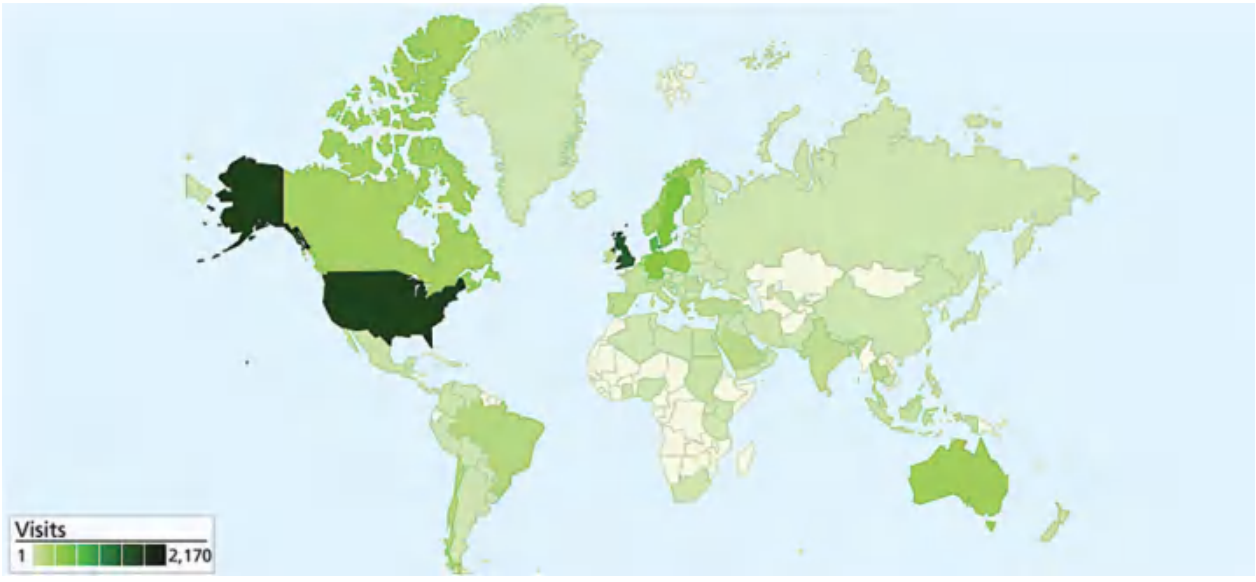


**Fig. 43.7** Use of the Dental Trauma Guide in the world.

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# 44

# International Association of Dental Traumatology (IADT)

L. Andersson, J. O. Andreasen & A. Moule

## The IADT

The International Association of Dental Traumatology (IADT) is a non-profit, public benefit professional organization whose mission is to promote optimal prevention, research, and treatment service in the field of traumatic dental injuries through interaction with dental and medical colleagues, the public, and interested parties in education, sports, industry and appropriate governmental agencies (Fig. 44.1).

## Start of the organization

The idea to form a multidisciplinary international association of dentists interested in dental trauma took shape during a meeting of Scandinavian researchers traveling to a mini-symposium on a cruise ship from Stockholm to Helsinki in February 1989. Dr. Jens Ove Andreasen discussed ideas for promoting research and education in dental trauma with his colleagues. Among those present were Drs. Frances Andreasen, Miomir Cvek, Lars Andersson, Barbro Malmgren, Ingeborg Jacobsen, Annika Skoglund, Lars Kristersson, Bengt Mohlin, Seppo Järvinen and Åke Nygren. During this weekend of brainstorming and discussions they concluded that there was a need to start a world association to promote all aspects of dental traumatology. With the help of Dr. Nygren from the Swedish insurance company Folksam, plans were initiated for an International Conference on Oral Trauma in Stockholm. The IADT was formed during that conference on September 21, 1989 in Stockholm. An important document was adopted at this conference called the Stockholm Manifesto on Accident Prevention, which stated that "all human beings have an equal right to health and safety." That document formed the basis for the IADT's goal of bringing together international healthcare providers with an interest in dental traumatology.

The stated mission of the IADT is to promote optimal prevention and treatment services through education and research. The target groups are dental and medical colleagues, interested parties in education, sports, industry, appropriate governmental agencies and the public. The first officers and directors elected at the Stockholm conference were: Drs. J. O. Andreasen (President), I. Jacobsen (Vice President), O. Sjöström (Secretary-Treasurer) and B. Ericsson, M. Cvek, Å. Nygren, L. Hammarström, K. Oikarinen, L. Tronstad, B. Scheer, A. Kaufmann, M. Matsumoto (Directors). These leaders and many others since then have guided the IADT into becoming a strong multidisciplinary international organization promoting an important component of healthcare worldwide with members in all continents of the world. IADT is governed by an international board of directors. The first president of IADT was Jens Andreasen, followed by Martin Trope, MarieTherese Flores, Asgeir Sigurdssson, Mitsuhiro Tsukiboshi, Lars Andersson, Alex Moule and Nestor Cohenca.

During the early years IADT was run by an enthusiastic core group of members and all the administration was carried out solely by its executive director Leif Bakland successfully leading the organization to a true international association incorporated in the United States. As the IADT grew there was a need for a professional administration to be appointed. Today the administration is run by RES Inc. Administrative and Planning, San Diego, California.

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**Fig. 44.1** Web page of IADT.

### Mission statement

In order to fulfil the mission of the IADT, the following objectives are adopted to guide the Association in its endeavors to promote education in dental traumatology:

- To promote research, both clinical and experimental, in dental traumatology.
- To stimulate development of educational curricula to enhance competency of dental graduates in managing dental injuries.
- To encourage incorporation of educational standards for advanced dental education programs to achieve proficiency in managing dental injuries.
- To promote public awareness of prevention and management of traumatic dental injuries.
- To develop and publish recommended guidelines for treatment of traumatic dental injuries based on current best evidence.
- To assist public health agencies in developing and implementing strategies for prevention and management of dental injuries.
- To represent the interests of dental traumatology in various dental forums.
- To maintain an organizational structure that promotes awareness of dental traumatology.

## IADT World Congresses

The first dental trauma congress was actually arranged in Dallas, USA before IADT was formally founded. Following this the IADT arranged World Congresses in Dental Traumatology in Stockholm, Sweden in 1989, Copenhagen, Denmark in 1991, London, UK 1993, San Diego, USA 1994, Tokyo, Japan 1995, Florence, Italy 1996, Guaruja, Brazil, 1997, Eilat, Israel 1998, Melbourne, Australia 1999, Oslo, Norway 2000, Boston, USA 2001, Santiago, Chile 2003, Reykjavik, Iceland 2005, Nagoya, Japan 2008, Verona, Italy 2010, Rio de Janeiro, Brazil 2012, Istanbul, Turkey 2014 and recently the 19th IADT World Congress in Brisbane, Australia 2016 (Fig. 44.2).

The aim of these congresses has been to spread the knowledge of dental trauma and its prevention to the dentists and health professionals around the world, and to educated practitioners and to stimulate inspiration and collaboration between various dental trauma research centers around the world.

## IADT Treatment Guidelines for Traumatic Dental Injuries

In the late 1990s it was decided that guidelines were needed for treatment of traumatic dental injuries due to the fact that globally very many suggestions have appeared on how to treat the many different types of injuries affecting permanent and primary teeth. For practical reasons the guidelines were divided into primary teeth and permanent teeth, and at a later stage further divided into fractures, luxations and avulsions (1–5).

The first working groups consisted of pedodontists, endodontists and oral surgeons. The first set of treatment guidelines were released in 2001. These were based on a review of current dental literature utilizing EMBAS, MEDLINE and PudMed searches as well as searches of the *Journal of Dental Traumatology*. In cases where data did not appear conclusive, recommendations were based on the consensus opinion of the working groups followed by review by the members of the IADT Board of Directors. The working groups continued to update treatment guidelines and the second version was published in 2007 (6–8). The third and latest version was published in 2012 (9–11). IADT guidelines have been

**Fig. 44.2** IADT Congress in Brisbane, Australia.

translated into 22 languages and are available on the IADT website free of charge. as well as on the website dentaltraumaguide.org (see Chapter 35).

These dental trauma treatment guidelines have been well received and are regarded as the gold standard by many dental societies. They were adopted by the American Association of Endodontists (AAE) in 2013 and the American Academy of Pediatric Dentistry (AAPD) in 2015(12–15).

## Fellowship

In 2015 IADT introduced a successful fellowship program whereby members can gain a Fellowship in Dental Traumatology by fulfilling certain prerequisites and sitting an online examination. Details are available on Fellowship tab on the IADT website www.iadt-dentaltrauma.org The first Fellows to be awarded Fellowship by examination were Els Tijskens from Belgium and Sung Chul Choi from Korea.

## Website

The IADT has a well-designed web page which can be accessed on www.iadt-dentaltrauma.org

Educational material is available for members in the *members only* section on the website. This material includes PowerPoint lectures and many illustrations that are all available free for downloading.

## How to become a member of IADT

Joining IADT is simple. Just log on to the website www.iadt-dentaltrauma.org and follow the prompts.

## *Dental Traumatology*, the scientific journal

As well as publishing the guidelines, IADT publishes the journal *Dental Traumatology*, the only scientific journal in the world solely dedicated to the publication of the science of dental traumatology (Fig. 44.3). The initiative to start a scientific journal was taken by Leif Tronstad in 1985. The name of the journal was *Endodontics and Dental Traumatology* and it is since then Medline indexed. Leif Tronstad was Editor-in-Chief from its start to 2000, when Martin Trope took over as Editor-in-Chief. To meet the increased interest in dental traumatology from other specialists than endodontists, the name was changed to *Dental Traumatology* in 2001. Cooperation with the Academy of Sports Dentistry was started. In 2007 Lars Andersson became the third Editor-in-Chief further developing the structure of the journal by introducing specialized sections with Associate Editors responsible for each section. Priority is given to original articles and review articles which has resulted in an increased impact factor and at the end of 2015 the impact factor reached its highest level ever, and the journal ranked in the second quartile of all Medline indexed scientific journals in dentistry oral surgery and oral medicine. The new Editor-in-Chief from 2016 is Paul Abbott.

## Newsletter

IADT publishes a newsletter on the IADT web page and in the *Dental Traumatology* journal three times a year to try to keep members abreast of happenings in dental traumatology in the wider community as well as reporting on the Associations activities.

**Fig. 44.3** The IADT journal, *Dental Traumatology*.

## Social media

IADT developed a Facebook discussion group in February 2014 https://www.facebook.com/groups/IADT.education/ which now boasts over 15 000 from around the world, confirming the international interest in dental traumatology. Members can comment on, or post cases their own dental traumatology cases for discussion. The discussion page is a good means for IADT to fulfil its mission to promote the prevention and education of dental traumatology in a worldwide forum

## Partnership with Dental Trauma Guide

The IADT works closely with the administrators of the *Dental Trauma Guide*, a non-profit interactive website initiated by Jens Andreasen and his group at the University Hospital in Copenhagen dedicated to optimizing the worldwide treatment of dental trauma. The *Dental Trauma Guide* is an interactive program which can be accessed through the web site www.dentaltrauma-guide.org.

*Dental Trauma Guide* uses the guidelines established by IADT which helped to provide support in the establishment of the guide. www.dentaltraumaguide.org. Interactive step by step instructions and animations on how to treat traumatic dental injuries can be found on this web site (see also Chapter 35).

**Fig. 44.4** Dental Trauma app for public advice about trauma.

## Dental Trauma App

IADT supports and endorses the Dental Trauma App, which is a smartphone app where the public can get First-Aid advice for traumatic dental injuries. This app is available for download through the App Store. The app is designed by Ulf Glendor and translated to more than 20 languages and sold in 61 countries (Fig. 44.4). At the time of writing IADT is working on another free app containing advice for the public, called 'Tooth SOS'. It is due for launch in 2018.

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# Appendix 1

## Emergency record for acute dental trauma

**Page 1**

| | |
|---|---|
| Patient's name<br>Birth date | |

| | |
|---|---|
| Date of examination:<br>Time of examination: | Referred by:<br>Referring diagnosis: |

| | | |
|---|---|---|
| *General medical history*: *Any serious illness?*<br>If *yes*, explain | yes | no |
| *Any allergy?*<br>If *yes*, explain | yes | no |
| *Have you been vaccinated against tetanus?*<br>If *yes*, when? | yes | no |
| *Previous dental injuries?*<br>If *yes*,<br>When?<br>Which teeth were injured?<br>Treatment given and by whom? | yes | no |
| *Present dental injury*:<br>Date: Time:<br>Where?<br>How? | | |
| Have you chipped or fractured any teeth? | yes | no |
| If so, where are these fragments now? | | |
| Have you had or have now *a headache*? | yes | no |
| Have you had or have now *nausea*? | yes | no |
| Have you had or have now *vomiting*? | yes | no |
| Were you *unconscious* at the time of injury? | yes | no |
| If *yes*, for how long (minutes)? | | |
| Can you *remember* what happened before, during or after the accident? | yes | no |

The emergency record is constructed so that wherever a question is answered by *yes*, more details must be provided. Finally, the last question in the record is whether the examiner has re-read the chart. This is a reminder to check that all relevant points have been registered.

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Page 2

Is there pain from *cold air*? yes no
If yes, *which teeth*?

Is there pain or tenderness from *occlusion*? yes no
If yes, *which teeth*?

Is there *constant pain*? yes no
If yes, *which teeth*?

*Treatment elsewhere*? yes no
If yes, *what treatment*?

After *avulsion*, the following information is needed:
*Where* were the teeth found (dirt, asphalt, floor, etc.)?
*When* were the teeth found?
Were the teeth *dirty?*
How were the teeth *stored?*
Were the teeth *rinsed* and *with what* prior to replantation?
*When* were the teeth replanted?
Was *tetanus antitoxoid* given?
Were *antibiotics* given?
*Antibiotic*?
*Dosage*?

Extra-alveolar time:
Dry time:
Extra-alveolar medium: Duration in this medium:
Extra-alveolar medium 2: Duration in medium 2:

**Objective examination** – Extraoral findings

Is the patient's general condition affected? yes no
If yes, *pulse*
*blood pressure*
*pupillary reflex*
*cerebral condition*
Objective findings beyond the head and neck? yes no
If yes, *type* and *location*
Objective findings within the head and neck? yes no
If yes, *type* and *location*

## General injuries

**Soft tissue:**

☐ Skin abrasion
☐ Skin laceration
☐ Skin contusion
☐ Mucosal abrasion
☐ Mucosal laceration
☐ Mucosal contusion
☐ Gingival abrasion
☐ Gingival laceration

**Bony #:**

☐ Alveolar #

☐ Which teeth

Location of # apical to apical foramen ☐

Next to apical foramen ☐

Coronal to apical foramen ☐

☐ Mandibular fracture #

☐ Maxillary fracture #

### Further details:

.....

.....

.....

.....

## Tooth-specific injuries

**(this is to be repeated for each injured tooth)**

**Tooth:**

| PDL | Hard tissue | | |
|---|---|---|---|
| ☐ None | ☐ None | | |
| ☐ Concussion | ☐ Infraction | | |
| ☐ Subluxation | ☐ E# | | |
| ☐ Extrusion | ☐ E/D# | | |
| ☐ Lateral luxation | ☐ E/D/P# | | |
| ☐ Intrusion | ☐ E/D/C # | Supra alveolar ☐ | Sub alveolar ☐ |
| ☐ Avulsion | ☐ E/D/P/C# | Supra alveolar ☐ | Sub alveolar ☐ |
| | ☐ Root # | | |

**Tooth:**

| PDL | Hard tissue | | |
|---|---|---|---|
| ☐ None | ☐ None | | |
| ☐ Concussion | ☐ Infraction | | |
| ☐ Subluxation | ☐ E# | | |
| ☐ Extrusion | ☐ E/D# | | |
| ☐ Lateral luxation | ☐ E/D/P# | | |
| ☐ Intrusion | ☐ E/D/C # | Supra alveolar ☐ | Sub alveolar ☐ |
| ☐ Avulsion | ☐ E/D/P/C# | Supra alveolar ☐ | Sub alveolar ☐ |
| | ☐ Root # | | |

**Tooth:**

| PDL | Hard tissue | | |
|---|---|---|---|
| ☐ None | ☐ None | | |
| ☐ Concussion | ☐ Infraction | | |
| ☐ Subluxation | ☐ E# | | |
| ☐ Extrusion | ☐ E/D# | | |
| ☐ Lateral luxation | ☐ E/D/P# | | |
| ☐ Intrusion | ☐ E/D/C # | Supra alveolar ☐ | Sub alveolar ☐ |
| ☐ Avulsion | ☐ E/D/P/C# | Supra alveolar ☐ | Sub alveolar ☐ |
| | ☐ Root # | | |

# Appendix 2

## Clinical examination form for the time of injury and follow-up examinations

| | | Tooth no. | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| TIME OF INJURY | Date | | | | | | | | |
| | Tooth color:<br>normal<br>yellow<br>red<br>grey<br>crown restoration | | | | | | | | |
| | Displacement (mm):<br>intruded<br>extruded<br>protruded<br>retruded | | | | | | | | |
| | Loosening (0–3) | | | | | | | | |
| | Tenderness to percussion (+/–) | | | | | | | | |
| | Pulp test (value) | | | | | | | | |
| | Ankylosis tone (+/–) | | | | | | | | |
| | Premature occlusal contact (+/–) | | | | | | | | |
| CONTROL | Fistula (+/–) | | | | | | | | |
| | Gingivitis (+/–) | | | | | | | | |
| | Gingival retraction (mm) | | | | | | | | |
| | Abnormal pocketing (+/–) | | | | | | | | |
| | Swelling (+/–) | | | | | | | | |

Each column represents an examination of a given tooth. The first column for each tooth gives the values from the time of injury. *Only* the parameters listed in the top half of the form ('Time of injury') should be recorded at the time of injury. The information from this examination as well as the information collected on the emergency record are used to determine the final diagnoses for the injured teeth. Those parameters *and* four more (fistula, gingivitis, gingival retraction, abnormal pocketing) should be registered at all follow-up controls.

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# Appendix 3

## Summary of treatment and follow-up procedures and recall schedule following the various trauma types

| | Radiographic exposure for various trauma types | | |
|---|---|---|---|
| **Post-traumatic interval*** | **Luxation of 21**** | **Replantation of 21** | **Root fracture of 21** |
| Time of injury | OI 11,21*** | OI 11,21 | OI 11,21 |
| | BI 12,11,21,22 | BI 12,11,21,22 | BI 12,11,21,22 |
| 1 week | | BI 21,22[a,b] | |
| 2–3 weeks | OI 11,21[a,b] | BI 21,22 | OI 11,21 |
| 6–8 weeks | BI 12,11,21,22[a] | BI,12,11,21,22 | BI 12,11,21,22 |
| 3 months | | BI 21,22 | BI 11,21 |
| 6 months | | BI 21,22 | |
| 1 year | BI 12,11,21,22 | BI 12,11,21,22 | BI 12,11,21,22 |
| 2, 3, 4 years | | BI 21,22 | |
| 5, 10, 15 years | BI 12,11,21,22 | BI 12,11,21,22 | BI 12,11,21,22 |

* All examinations include radiographs as well as information from the clinical examination form (see Appendix 2).

** Tooth designation is according to the FDI two-digit system.

*** Regarding radiographic exposure, OI implies 'occlusal identical', or occlusal exposures; BI implies 'bisecting identical'. Both designations imply the use of standardized techniques and filmholder.

[a] Removal of fixation. The following fixation periods are suggested. The reader is referred to the respective chapters for details: Replantation, 1–2 weeks; Extrusion, 2 weeks; Lateral luxation, 4 weeks (depending on radiographic findings); Intrusion, see Chapter 16; Root fracture, 4 weeks.

[b] Begin endodontic therapy: Replantation, after 1 week; Intrusion, after 2–3 weeks.

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# Appendix 4

## Comparison between cost/effectiveness of different treatment solutions for anterior tooth loss

| | Orthodontics | Implants | Resin-retained bridges | Conventional bridges | Autotransplants |
|---|---|---|---|---|---|
| Cost | +++ | +++ | + | +++ | + |
| Long-term service | +++ | +++ | + | ++ | +++ |
| Use in growing individuals | +++ | 0 | +++ | 0 | +++ |
| Bone-inducing potential | + | 0 | 0 | 0 | +++ |

Scale used: 0 = not to be used or non-existent; + = minimum (low); ++ = medium (moderate); +++ = maximum (high).

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