Pediatric Endodontics

Current Concepts in Pulp Therapy for Primary and Young Permanent Teeth

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This book is dedicated to
Moises Fuks, my beloved husband and
long-term companion and friend
and to
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and precious family

Anna B. Fuks and Benjamin Peretz

Preface

The initial idea for writing this book came because we felt that there has been an explosion of scientific knowledge on the understanding of the pulp tissue in the last two decades, which, in turn, affect the proper treatment for various pulp pathological conditions. This immense advancement has included the primary pulp also, and pediatric dentistry today, with regard to pulp treatment, can provide a better, more problem-oriented therapy and treatment to the affected primary pulp. Therefore, when we were approached by the Springer representative to write this book, we gladly agreed.

We felt that there was a need for students, undergraduate and postgraduate alike, as well as for the professional community to be familiarized with the current "state of the art" on pediatric endodontics. We made all efforts to cover the various aspects of the dentin-pulp complex in pediatric dentistry: from the understanding of biological concepts of the healthy pulp, through the pulp reactions to the deleterious effects of caries, to the various treatment modalities for each type of pulp injury, to the adverse reactions to various pulp dressing materials, and to the postoperative prognosis.

The better understanding of these topics led us to conclude that a conservative approach in the treatment of reversibly inflamed pulp needs to be emphasized. Thus, considerable attention has been given to the conservative approach to pulp treatment in primary and young permanent teeth. Our message stresses the paradigm shift toward conservative treatment modalities, relying on an accurate diagnosis based on signs and symptoms to assess the appropriateness of the technique for a specific case.

Notwithstanding, the traditional modes of treatment are also covered.

Understanding the new concepts regarding pulp treatment will guide practicing pediatric dentists and general dentists to select the proper mode of treatment.

A special emphasis has also been given to the future of pulp treatment, in light of the innovative knowledge on stem cells. At present, there is a consensus that the future of medicine and dentistry, particularly of pulp treatment, lies in the thorough research on stem cells.

We hope that this text will be useful to all students and dentists who treat children, to provide a better care for their teeth.

Jerusalem, Israel — Anna B. Fuks
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Contents

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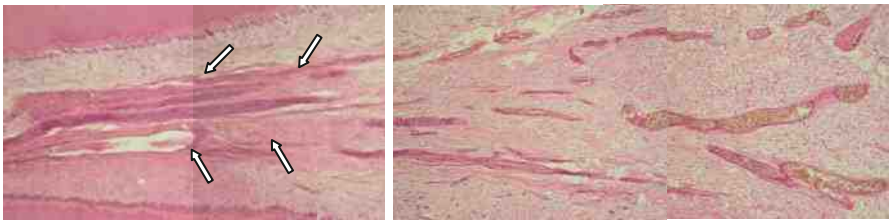


Fig. 2.4 Sections obtained from human sound teeth showing the radicular (*left*) and coronal (*right*) portions of the pulp tissue. Note the fibrous connective pulp tissue with vascular–nervous sheath close to the apical foramen (*arrows*). Conversely, the coronal pulp exhibits loose connective tissue with a number of blood vessels. H/E, 32×

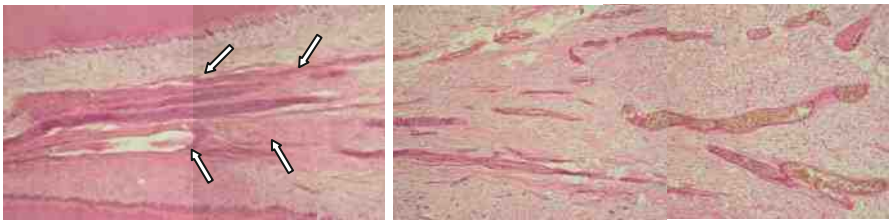
production of dentin, the pulp remains enclosed within the central part of the tooth, having a coronal and a radicular portion. In uni-radicular teeth, the coronal and radicular pulp tissues are contiguous, but in multi-radicular teeth, the floor of the pulp chamber has a clear distinction: the coronal pulp is rich in cells and extracellular matrix, while the radicular pulp has more fibers, and the vascular–nervous sheath is more concentrated, with less anastomosis (Fig. 2.4).

2.3.1 Odontoblasts

The odontoblasts have been traditionally described as cells lining the periphery of the pulpal space and extending their cytoplasmic processes into the dentinal tubules. These cells have several junctions, which allow for intercellular communication and help to maintain the relative position of one cell to another. In young permanent teeth, the pulp tissue exhibits defined zones. The cell-free zone is located just below the odontoblastic layer and contains an extensive plexus of unmyelinated nerves and blood capillaries. The cell-rich zone, which presents a number of undifferentiated mesenchymal cells, is observed adjacent to the cell-free zone. The core of the dental pulp contains larger blood vessels and nerves, which are surrounded by large area of extracellular matrix. This pulp morphology is similar to that observed in primary teeth, but the zones are not so well defined (Fig. 2.5a, b).

Although this description is correct during active dentinogenesis, it is now accepted that the size of the odontoblasts and the content of their cytoplasmic organelles vary throughout their life cycle and are closely related to their functional activity. The relationship between the size of the odontoblasts and their secretory activity can be demonstrated by differences in their size in the crown and in the root, and different dentinogenic rates may be expressed in these two areas of the tooth [7].

The odontoblasts are highly specialized cells and are responsible for the formation of dentin. Due to the extension of their cytoplasmic processes into the dentinal tubules, these cells compose the main part of the dentin–pulp complex. When this complex is damaged by disease or attrition, or is affected by operative procedures, it reacts in an attempt to defend the pulp tissue.



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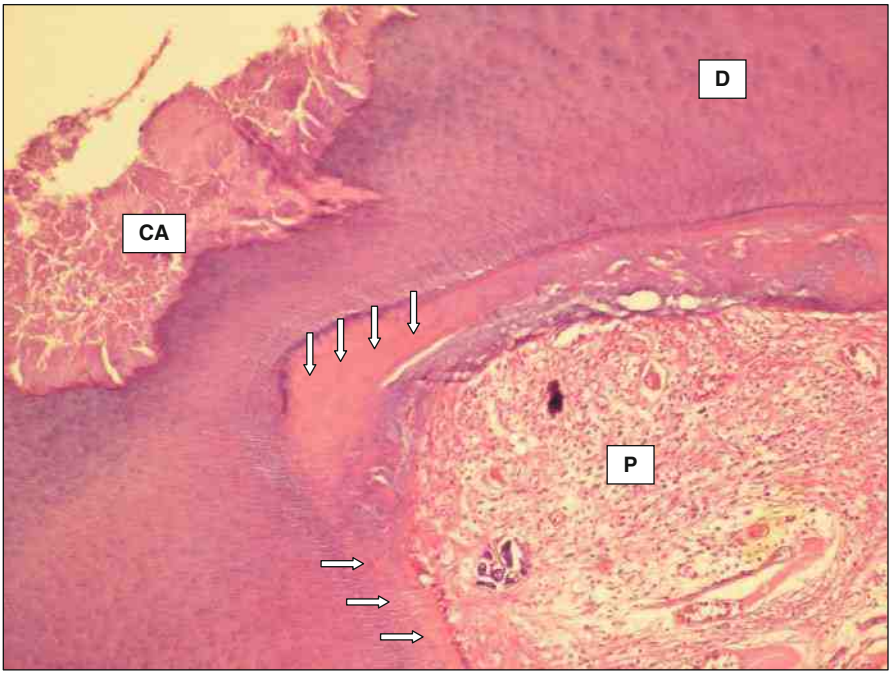

Fig. 2.7 Section obtained from primary teeth with acute occlusal caries (*CA*). Note the layer of tubular reactionary dentin (*horizontal arrows*) deposited in the lower part of the picture where the primary odontoblasts were far from the intense stimulus (pay attention to the dentinal tubule orientation). However, in the upper area of the picture, where the pulp is close to the very deep caries lesion (*CA*), a thick layer of reparative dentin with no tubules, termed as reparative dentin (*vertical arrows*), can be observed. H/E, 32× (*D* Dentin, *P* Pulp)

calcospherites (Fig. 2.8). Failure of the calcospherites to fuse leads to the formation of hypomineralized areas, known as interglobular dentin. These areas are more visible in the radicular dentin, where the dentin is produced simultaneously with the eruptive process, and on the most external portion of the coronal dentin, at the limit between the mantle and the circumpulpal dentin. Predentin consists mainly of types I and III collagen, glycoproteins, and proteoglycans.

Another type of hypocalcification is the Tomes' grainy layer that is formed by the terminal loops and branches of the odontoblastic membrane. This membrane configuration is developed during the formation of the radicular dentin, giving to the peripheral dentin a grainy appearance.

Dentin is composed of tubules. As the odontoblasts secrete the organic matrix, they emit a projection that is surrounded by liquid, providing the tubular aspect. The tubules have a lightly conical shape, due to the mineralization process of the peritubular dentin that occurs throughout the life of the tooth. The tubules extend through the entire thickness of the dentin, following the sinuous track of the odontoblasts. The number of dentinal tubules varies according to the area when different teeth are

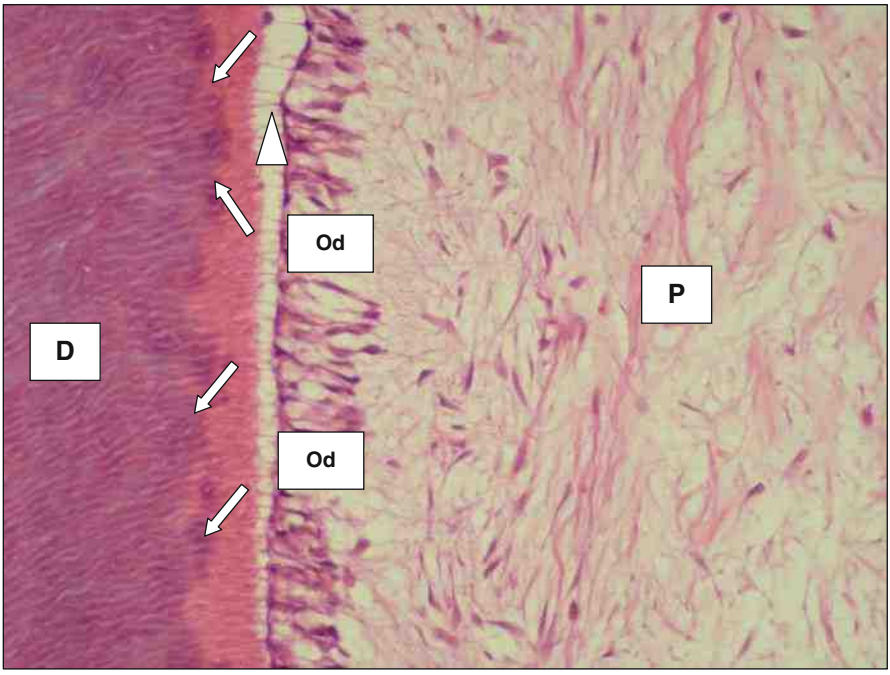

Fig. 2.8 Section obtained from sound primary teeth. Between the tubular dentin and the thin layer of predentin one can see the fronts of mineralization – calcospherites (*oblique arrows*). Note the odontoblasts (*Od*) with their cytoplasmic processes which get inside the dentinal tubules (*head arrow*) *D* dentin. *P* pulp. H/E, 125×

compared. It has been demonstrated that at the enamel–dentin junction (superficial dentin), there are approximately 20,000 tubules/mm^2, while near the predentin (deep dentin), this number increases to approximately 75,000 tubules/mm^2.

The dentin surrounding the periphery of the dentinal tubules is known as peritubular or intertubular dentin. Intertubular dentin is present between dentinal tubules. The odontoblast cytoplasmic processes remain within the dentinal tubules (Fig. 2.8).

Communications among the dentinal tubules, known as dentinal canaliculus, are frequently observed. The peritubular dentin that constitutes the walls of the dentinal tubules is four times harder than intertubular dentin, since it consists of approximately 96 % of hydroxyapatite crystals. Mild stimuli from the external environment, such as attrition and caries, may cause obliteration of the dentinal tubules, resulting in dentin sclerosis. Intertubular dentin is partially composed of collagen fibrils positioned perpendicularly to the long axis of the tubules, surrounding the tubules (Fig. 2.9a, b). The conditioning of the dentin substrate with acidic agents or chelating substances decreases or removes the peritubular dentin on the surface, leaving a mesh of intertubular collagen exposed to the action of bonding agents or to bacteria from decay [9, 10].

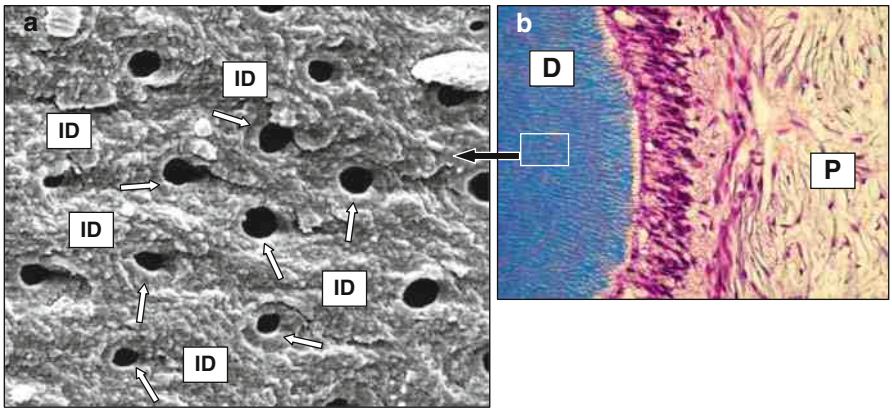

Fig. 2.9 (**a**) Morphology of the dentin structure. Note the dentinal tubules surrounded by peritubular dentin (*arrows*) as well as a large area of intertubular dentin (*ID*). MEV, 3.000×. (**b**) Dentin–pulp complex. *D* dentin. *P* pulp. Cytoplasmic processes from odontoblasts are observed inside the dentinal tubules. Masson's trichrome, 125×

2.5 Factors Affecting the Dentin–Pulp Complex Response to Stimuli in Primary Teeth

Although the life span of primary teeth is shorter and their dentin is thinner when compared to that of permanent teeth, the dentin–pulp complex response to dental caries in human primary teeth is similar to that of permanent teeth, including a reduction in the number of the odontoblasts and an increase in the number of inflammatory cells. These are found under very deep lesions and are less numerous in more distant regions, being almost absent in the radicular apical pulp [11]. The primary dentition is frequently subjected to stimuli such as trauma or caries with associated pulpal inflammation [12]. The same factors affect both the dentin–pulp responses in primary as well as permanent teeth, with respect to external stimuli.

2.6 The Deleterious Effects of Bacterial Infiltration at the Restorative Material Margins

A significant number of studies have implicated the presence of bacteria and their products as responsible for induction of the most severe forms of pulp inflammation. The role of bacteria in the inflammatory reaction was demonstrated by spontaneous healing of pulp exposures in germ-free animals [13] and subsequently by cavities restored with different materials and surface sealed with zinc oxide–eugenol cement to prevent any bacterial contamination originating from microleakage [14].

The presence of bacteria in cavities with a remaining dentin thickness (RDT) of less than 0.25 mm stimulates a more severe pulpal inflammatory reaction than in similar cavity preparations in the absence of bacteria [15]. Thus, the presence of bacteria always increases the mean grade of pulpal inflammation regardless of the RDT [16]. The presence of bacteria in class V cavity was also observed to result in a significant

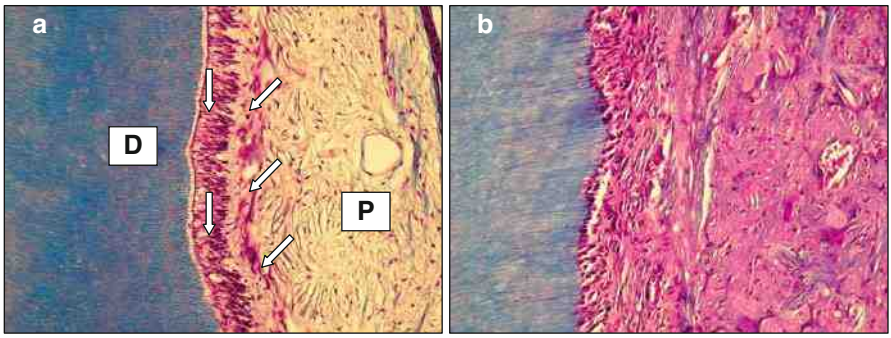

Fig. 2.10 (**a**) Section obtained from a young sound premolar in which a very deep class V cavity was prepared and the cavity floor lined with hard-setting calcium hydroxide cement. Note the pulp tissue with normal histological characteristics. Masson's trichrome, 64× (*D* Dentin, *P* Pulp; *vertical arrows* odontoblast layer; *oblique arrows* cell-rich zone). (**b**) In this human premolar, the cavity floor (dentin) was conditioned and a resin-based material was used as liner. Note the intense inflammatory response associated with complete pulp tissue disorganization. No microleakage at the cavity walls was observed after using a specific staining technique (Brown and Brenn) widely employed for disclosing bacteria. Masson's trichrome, 64×

decrease in the number of odontoblasts per unit area; this effect was more pronounced in deep cavities with RDT less than 0.5 mm than in cavities with RDT greater than 0.5 mm [16]. One can conclude that the ability to maintain an effective seal to protect the pulp from recurrent injury resulting from bacterial microleakage is a decisive factor in the clinical success of restorative products [17]. However, a number of studies performed in human teeth have shown pulpal inflammation in the absence of bacteria [8, 18–22], clearly indicating that other factors, such as the toxicity of dental material components capable of diffusing through dentinal tubules, are also responsible, at least in part, for pulp injury after restorative treatment (Fig. 2.10a, b).

2.7 The Protective Role of the Remaining Dentin Thickness (RDT)

It was found in an in vivo study that the cavity RDT is an important factor mediating pulpal inflammatory activity, particularly when the RDT is reduced to less than 0.3 mm [8, 22]. With an RDT less than 0.25 mm, a significant decrease in the number of odontoblasts was observed together with minimal reactionary dentin repair [23, 24]. It was recently demonstrated that very deep class V cavities prepared in human premolars (RDT thinner than 0.3 mm) which were subjected to adhesive restorations resulted in inflammatory pulp reaction associated with inner dentin resorption [8, 22, 25]. The presence of an RDT of more than 500 mm delays the diffusion of noxious materials into the dental pulp. In this clinical situation, the odontoblasts maintain their metabolism, or, in case of a slight stimulus, they may secrete a reactionary dentin, increasing the total distance between the restorative material and the pulp [8]. Any additional decrease in the dentin thickness to less than 500 mm results in a significant reduction in the number of odontoblasts.

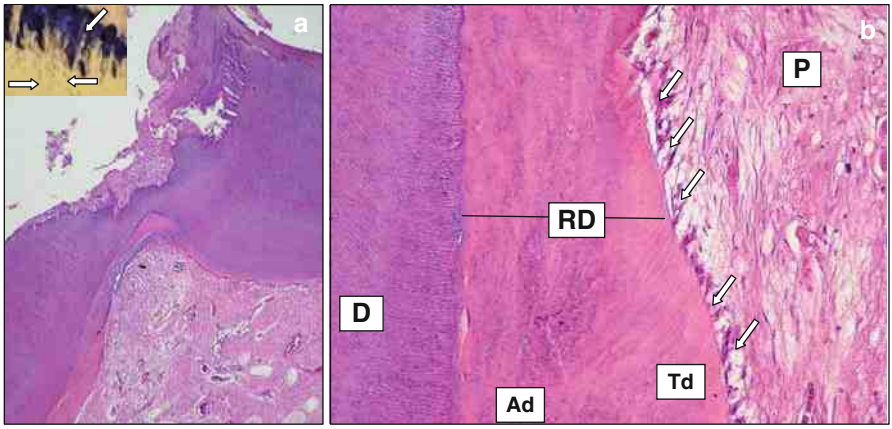

Fig. 2.11 (**a**) Deep carious lesion in primary first molar. H/E. 32×. The small image characterizes the necrotic dentin (*oblique arrow*) and the presence of microorganisms inside the dentinal tubules (*horizontal arrows*). Brown and Brenn technique, 125×. Note the intense inflammatory pulp reaction associated with complete tissue disorganization. (**b**) Detail of (**a**). Note the intense deposition of reparative dentin (*RD*) adjacent to the primary dentin (*D*). A heterogeneous and atubular dentin matrix (*Ad*) containing parts of dead odontoblasts as well as a tubular dentin (*Td*) deposited by the new odontoblast-like cells (*arrows*) can be observed. H/E, 125× (*P* Pulp)

This reduction may be compensated for by the differentiation of odontoblast-like cells from progenitor pulp cells, which migrate to the injury site and secrete reparative dentin. The reparative dentin decreases the dentin permeability and increases the distance between the restorative material and the pulp, protecting it from noxious products. However, in this specific condition, the number of mesenchymal stem cells decreases, interfering with the potential of pulpal healing in case of further damage to the dentin–pulp complex. Thus, the RDT appears to provide an important protective barrier against toxins, bacterial infiltration, or any noxious material applied to dentin. In this way, it seems adequate to protect the pulp tissue against irritant stimuli by using biocompatible materials as liners in very deep cavities [8].

Based on the remaining dentin thickness, three situations can be taken into consideration:

1. Initial carious lesion or shallow cavity preparations (RDT > 500 mm): a localized reactionary dentin may be secreted facing the restoration site, and intratubular mineralization (dentin sclerosis) occurs, resulting in a significant decrease in the dentin permeability and pulp protection. It has been suggested that this stimulation may be due to signaling molecules (i.e., TGF-b1, BMP-2 liberated from the dentin during demineralization) [26].
2. Carious lesion progression implying a deep cavity preparation (RDT < 500 mm): these lesions may lead to partial death of odontoblast. Depending on the pulp inflammatory intensity, progenitor/stem cells can migrate to the injury site and differentiate to give rise to a new generation of odontoblast-like cells. These cells are responsible for the deposition of a specific type of tertiary dentin termed as reparative dentin, as described above [27, 28] (Fig. 2.11a, b).

3. During a subsequent restorative process, deep cavity preparations with RDTs between 250 and 40 mm lead to poor tertiary dentin repair activity [15]. These result from impaired odontoblast dentin secretory activity due to cellular injury [29]. The study demonstrated that the mean number of intact odontoblasts found beneath this kind of cavity preparation was 36% lower than the number found beneath similar preparations with an RDT between 500 and 250 mm. This lack in the ability of the odontoblasts to provide adequate pulp repair and pulp protection after deep cavity preparation has been supported by observations of a persistent inflammatory pulpal response and odontoblast displacement following such deep cavity preparations [29].

2.8 Clinical Recommendations

In clinical situations, conservative careful cavity preparations should be carried out: intermittent cutting movement, air/spray cooling, and use of new burs. In addition, biocompatible, antibacterial, and bioactive dental products must be used as liners to protect the pulp tissue against toxic components released from restorative materials capable of diffusing across the dentin [20, 30].

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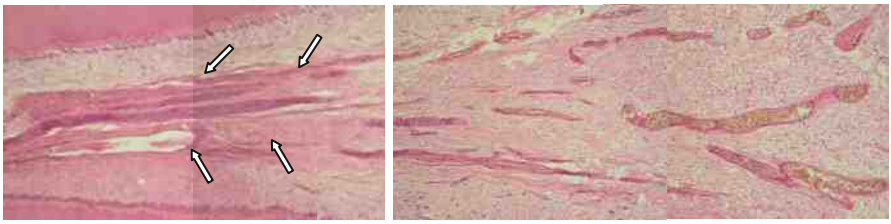

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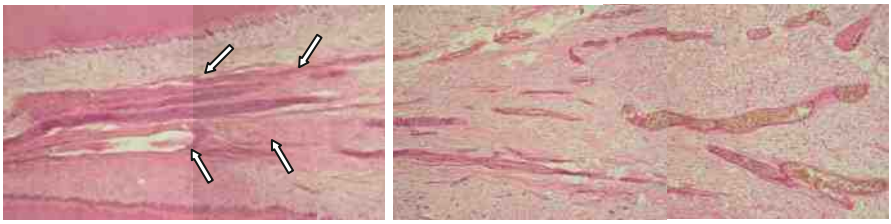

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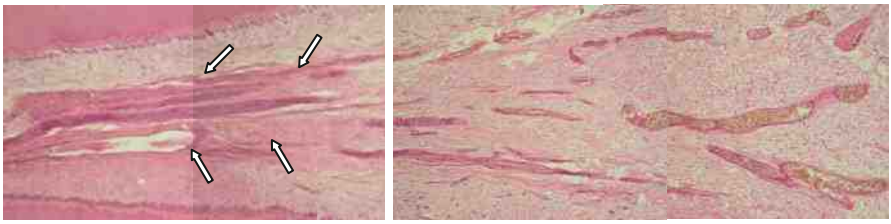

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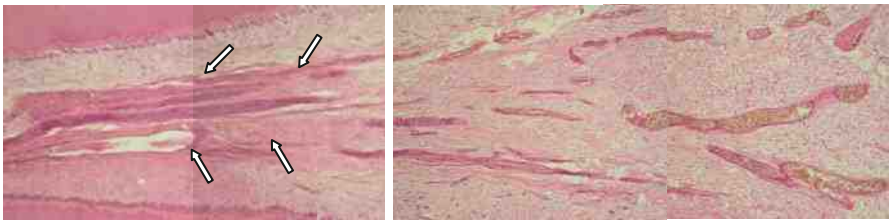

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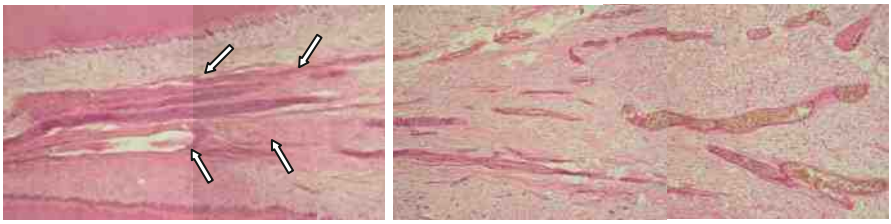

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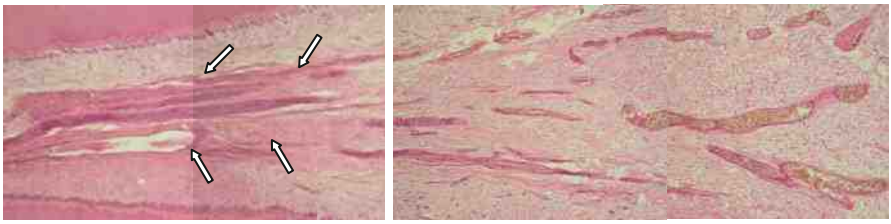

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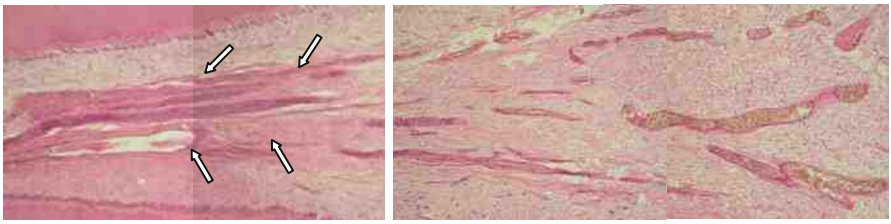

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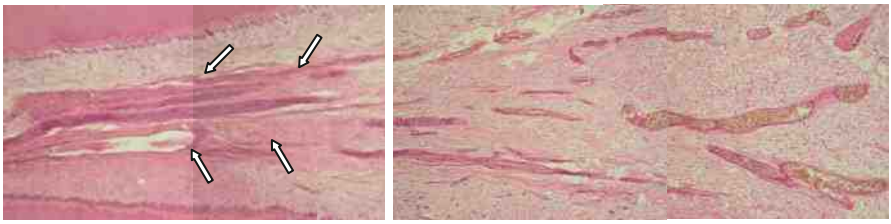

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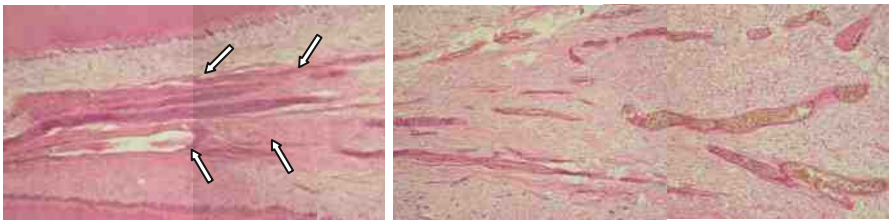

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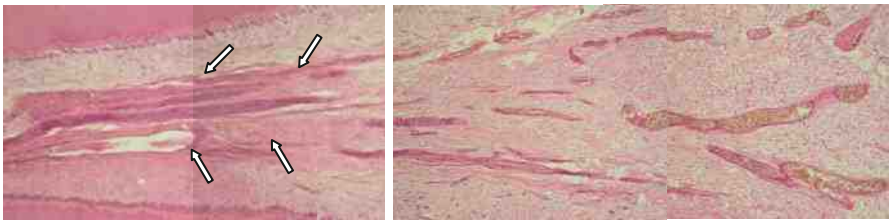

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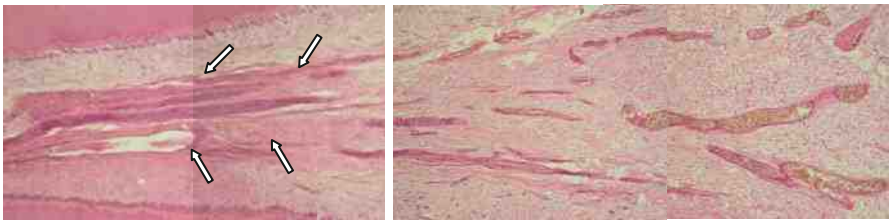

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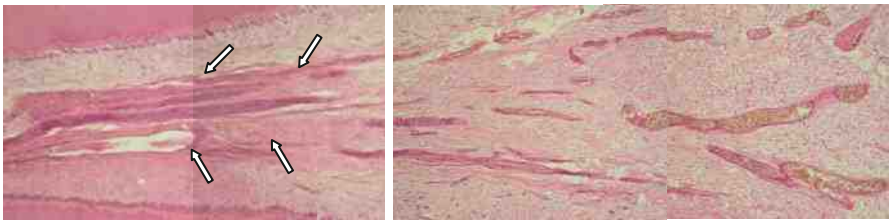

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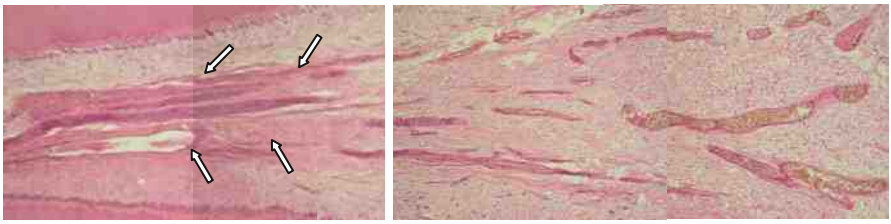

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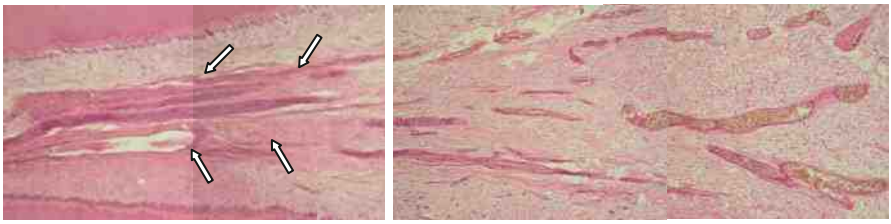

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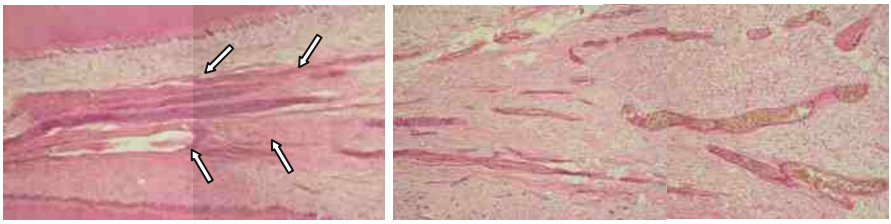

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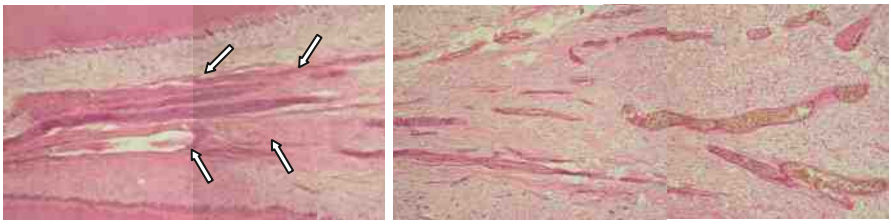

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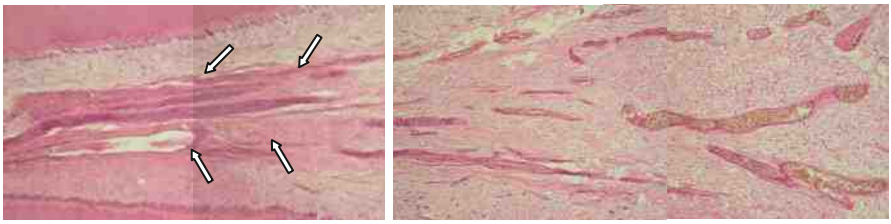

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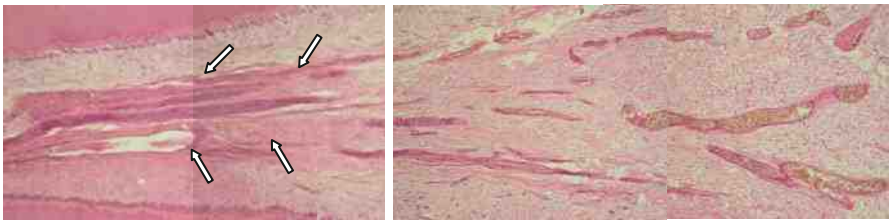

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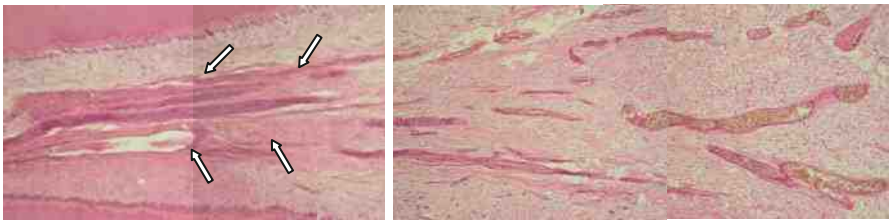

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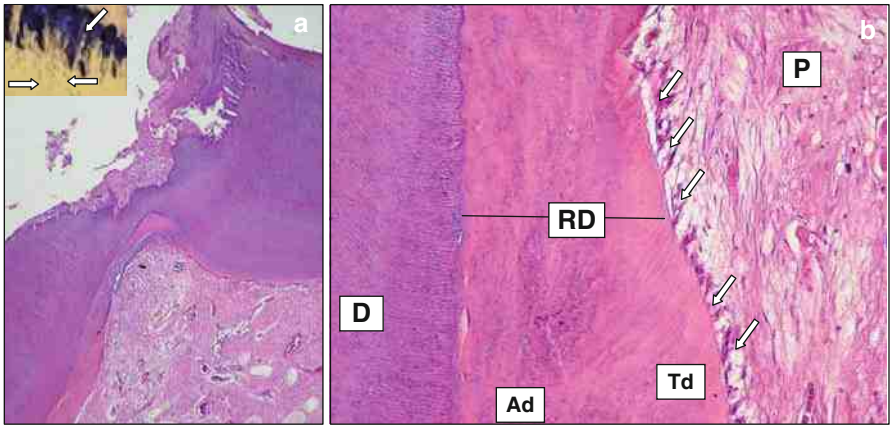
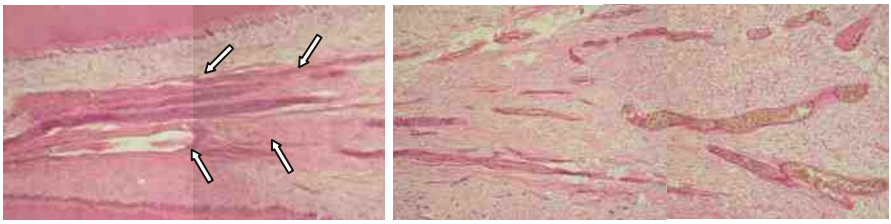


Fig. 2.11 (**a**) Deep carious lesion in primary first molar. H/E. 32×. The small image characterizes the necrotic dentin (*oblique arrow*) and the presence of microorganisms inside the dentinal tubules (*horizontal arrows*). Brown and Brenn technique, 125×. Note the intense inflammatory pulp reaction associated with complete tissue disorganization. (**b**) Detail of (**a**). Note the intense deposition of reparative dentin (*RD*) adjacent to the primary dentin (*D*). A heterogeneous and atubular dentin matrix (*Ad*) containing parts of dead odontoblasts as well as a tubular dentin (*Td*) deposited by the new odontoblast-like cells (*arrows*) can be observed. H/E, 125× (*P* Pulp)

This reduction may be compensated for by the differentiation of odontoblast-like cells from progenitor pulp cells, which migrate to the injury site and secrete reparative dentin. The reparative dentin decreases the dentin permeability and increases the distance between the restorative material and the pulp, protecting it from noxious products. However, in this specific condition, the number of mesenchymal stem cells decreases, interfering with the potential of pulpal healing in case of further damage to the dentin–pulp complex. Thus, the RDT appears to provide an important protective barrier against toxins, bacterial infiltration, or any noxious material applied to dentin. In this way, it seems adequate to protect the pulp tissue against irritant stimuli by using biocompatible materials as liners in very deep cavities [8].

Based on the remaining dentin thickness, three situations can be taken into consideration:

1. Initial carious lesion or shallow cavity preparations (RDT > 500 mm): a localized reactionary dentin may be secreted facing the restoration site, and intratubular mineralization (dentin sclerosis) occurs, resulting in a significant decrease in the dentin permeability and pulp protection. It has been suggested that this stimulation may be due to signaling molecules (i.e., TGF-b1, BMP-2 liberated from the dentin during demineralization) [26].
2. Carious lesion progression implying a deep cavity preparation (RDT < 500 mm): these lesions may lead to partial death of odontoblast. Depending on the pulp inflammatory intensity, progenitor/stem cells can migrate to the injury site and differentiate to give rise to a new generation of odontoblast-like cells. These cells are responsible for the deposition of a specific type of tertiary dentin termed as reparative dentin, as described above [27, 28] (Fig. 2.11a, b).



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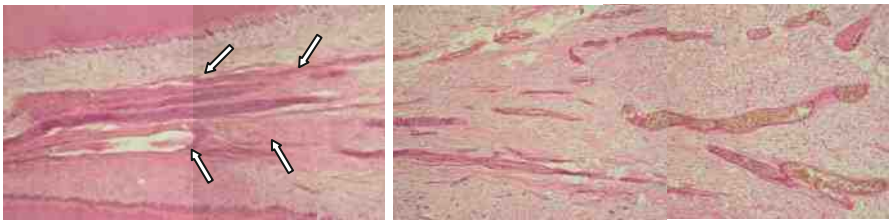

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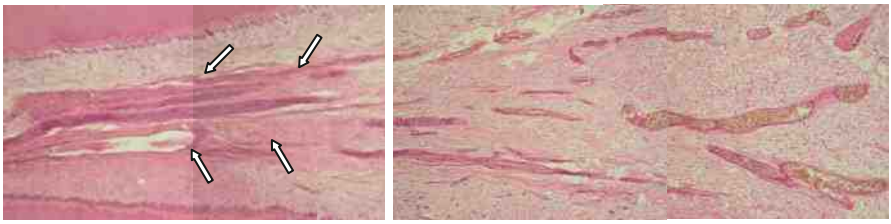

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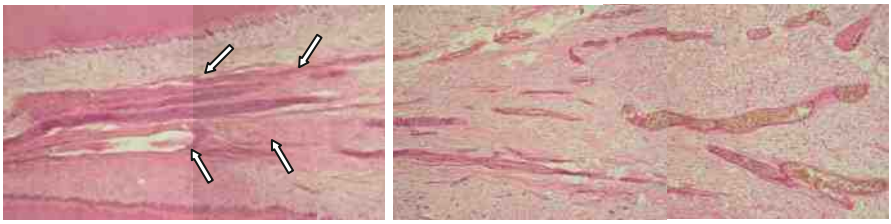

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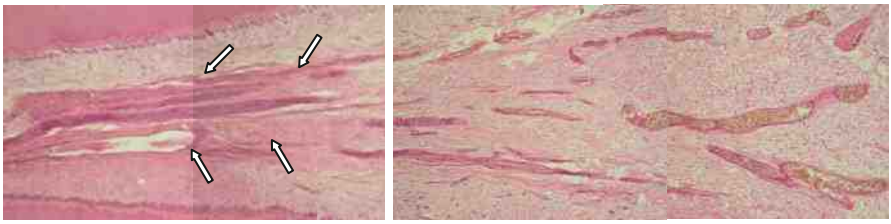

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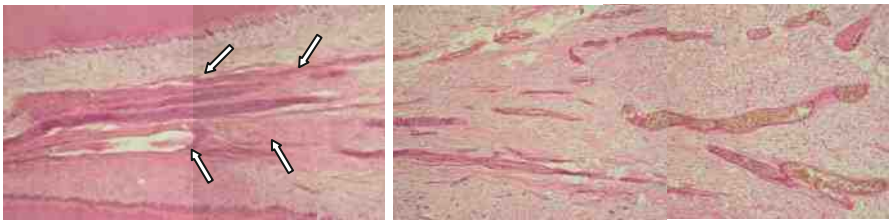

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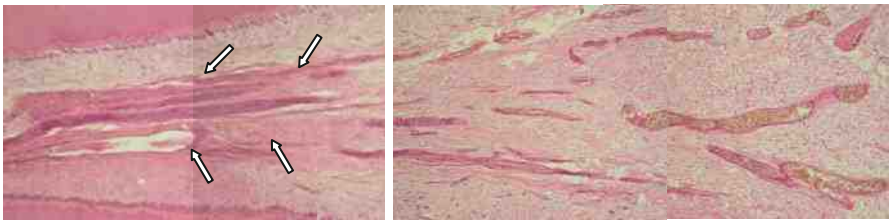

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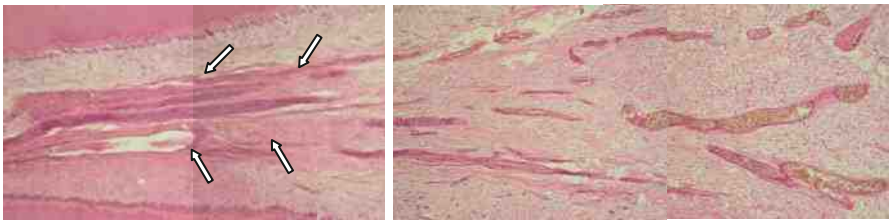

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Pediatric Endodontics

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This book is dedicated to
Moises Fuks, my beloved husband and
long-term companion and friend
and to
Tamar, Neta and Alona Peretz, my beloved
and precious family

Anna B. Fuks and Benjamin Peretz

Preface

The initial idea for writing this book came because we felt that there has been an explosion of scientific knowledge on the understanding of the pulp tissue in the last two decades, which, in turn, affect the proper treatment for various pulp pathological conditions. This immense advancement has included the primary pulp also, and pediatric dentistry today, with regard to pulp treatment, can provide a better, more problem-oriented therapy and treatment to the affected primary pulp. Therefore, when we were approached by the Springer representative to write this book, we gladly agreed.

We felt that there was a need for students, undergraduate and postgraduate alike, as well as for the professional community to be familiarized with the current "state of the art" on pediatric endodontics. We made all efforts to cover the various aspects of the dentin-pulp complex in pediatric dentistry: from the understanding of biological concepts of the healthy pulp, through the pulp reactions to the deleterious effects of caries, to the various treatment modalities for each type of pulp injury, to the adverse reactions to various pulp dressing materials, and to the postoperative prognosis.

The better understanding of these topics led us to conclude that a conservative approach in the treatment of reversibly inflamed pulp needs to be emphasized. Thus, considerable attention has been given to the conservative approach to pulp treatment in primary and young permanent teeth. Our message stresses the paradigm shift toward conservative treatment modalities, relying on an accurate diagnosis based on signs and symptoms to assess the appropriateness of the technique for a specific case.

Notwithstanding, the traditional modes of treatment are also covered.

Understanding the new concepts regarding pulp treatment will guide practicing pediatric dentists and general dentists to select the proper mode of treatment.

A special emphasis has also been given to the future of pulp treatment, in light of the innovative knowledge on stem cells. At present, there is a consensus that the future of medicine and dentistry, particularly of pulp treatment, lies in the thorough research on stem cells.

We hope that this text will be useful to all students and dentists who treat children, to provide a better care for their teeth.

Jerusalem, Israel — Anna B. Fuks
Tel Aviv, Israel — Benjamin Peretz

Contents

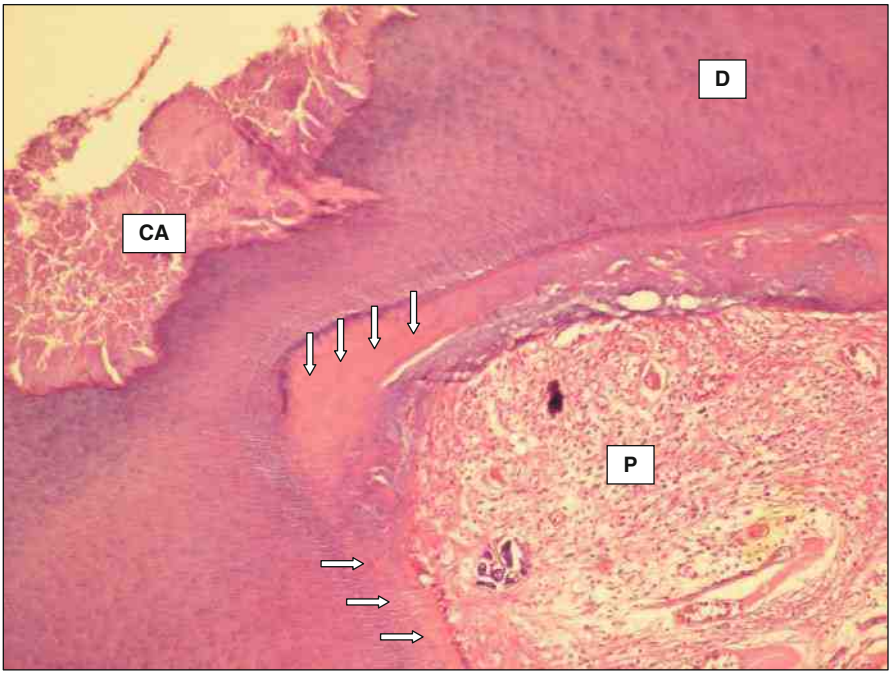

Fig. 2.7 Section obtained from primary teeth with acute occlusal caries (*CA*). Note the layer of tubular reactionary dentin (*horizontal arrows*) deposited in the lower part of the picture where the primary odontoblasts were far from the intense stimulus (pay attention to the dentinal tubule orientation). However, in the upper area of the picture, where the pulp is close to the very deep caries lesion (*CA*), a thick layer of reparative dentin with no tubules, termed as reparative dentin (*vertical arrows*), can be observed. H/E, 32× (*D* Dentin, *P* Pulp)

calcospherites (Fig. 2.8). Failure of the calcospherites to fuse leads to the formation of hypomineralized areas, known as interglobular dentin. These areas are more visible in the radicular dentin, where the dentin is produced simultaneously with the eruptive process, and on the most external portion of the coronal dentin, at the limit between the mantle and the circumpulpal dentin. Predentin consists mainly of types I and III collagen, glycoproteins, and proteoglycans.

Another type of hypocalcification is the Tomes' grainy layer that is formed by the terminal loops and branches of the odontoblastic membrane. This membrane configuration is developed during the formation of the radicular dentin, giving to the peripheral dentin a grainy appearance.

Dentin is composed of tubules. As the odontoblasts secrete the organic matrix, they emit a projection that is surrounded by liquid, providing the tubular aspect. The tubules have a lightly conical shape, due to the mineralization process of the peritubular dentin that occurs throughout the life of the tooth. The tubules extend through the entire thickness of the dentin, following the sinuous track of the odontoblasts. The number of dentinal tubules varies according to the area when different teeth are

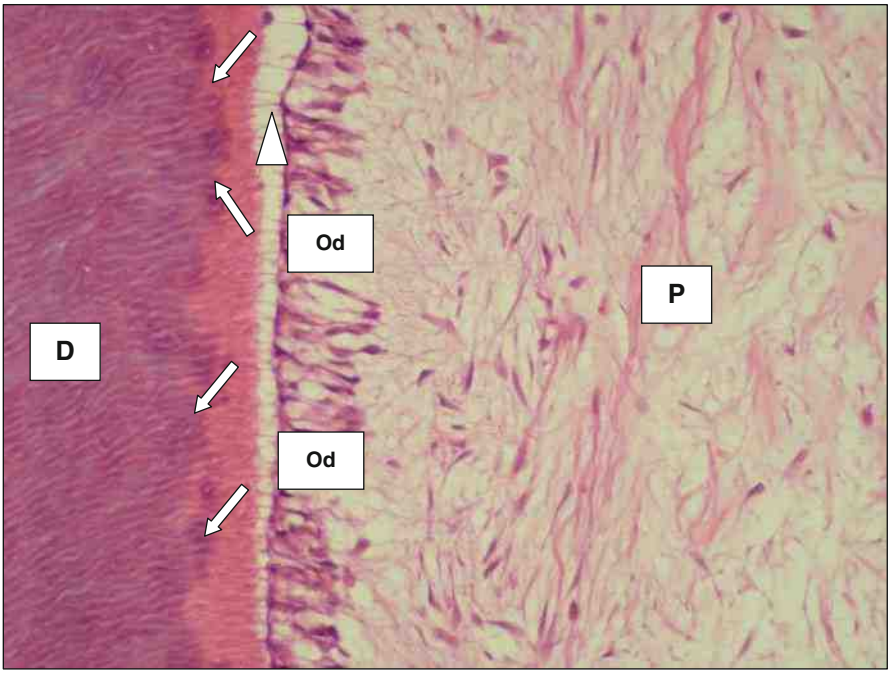

Fig. 2.8 Section obtained from sound primary teeth. Between the tubular dentin and the thin layer of predentin one can see the fronts of mineralization – calcospherites (*oblique arrows*). Note the odontoblasts (*Od*) with their cytoplasmic processes which get inside the dentinal tubules (*head arrow*) *D* dentin. *P* pulp. H/E, 125×

compared. It has been demonstrated that at the enamel–dentin junction (superficial dentin), there are approximately 20,000 tubules/mm^2, while near the predentin (deep dentin), this number increases to approximately 75,000 tubules/mm^2.

The dentin surrounding the periphery of the dentinal tubules is known as peritubular or intertubular dentin. Intertubular dentin is present between dentinal tubules. The odontoblast cytoplasmic processes remain within the dentinal tubules (Fig. 2.8).

Communications among the dentinal tubules, known as dentinal canaliculus, are frequently observed. The peritubular dentin that constitutes the walls of the dentinal tubules is four times harder than intertubular dentin, since it consists of approximately 96 % of hydroxyapatite crystals. Mild stimuli from the external environment, such as attrition and caries, may cause obliteration of the dentinal tubules, resulting in dentin sclerosis. Intertubular dentin is partially composed of collagen fibrils positioned perpendicularly to the long axis of the tubules, surrounding the tubules (Fig. 2.9a, b). The conditioning of the dentin substrate with acidic agents or chelating substances decreases or removes the peritubular dentin on the surface, leaving a mesh of intertubular collagen exposed to the action of bonding agents or to bacteria from decay [9, 10].

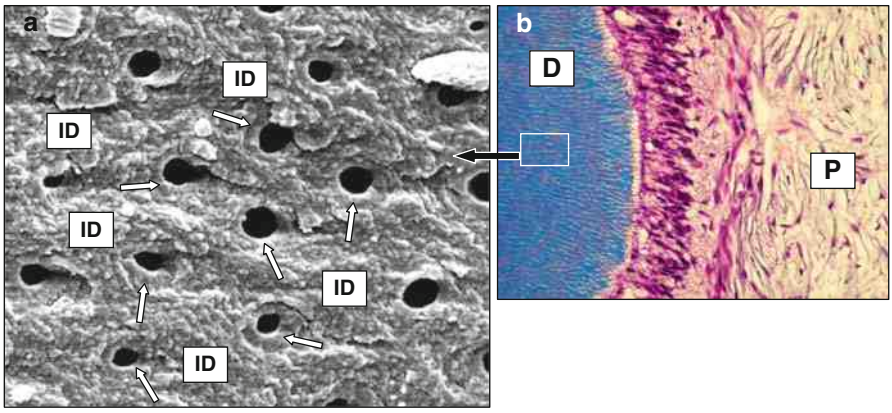

Fig. 2.9 (**a**) Morphology of the dentin structure. Note the dentinal tubules surrounded by peritubular dentin (*arrows*) as well as a large area of intertubular dentin (*ID*). MEV, 3.000×. (**b**) Dentin–pulp complex. *D* dentin. *P* pulp. Cytoplasmic processes from odontoblasts are observed inside the dentinal tubules. Masson's trichrome, 125×

2.5 Factors Affecting the Dentin–Pulp Complex Response to Stimuli in Primary Teeth

Although the life span of primary teeth is shorter and their dentin is thinner when compared to that of permanent teeth, the dentin–pulp complex response to dental caries in human primary teeth is similar to that of permanent teeth, including a reduction in the number of the odontoblasts and an increase in the number of inflammatory cells. These are found under very deep lesions and are less numerous in more distant regions, being almost absent in the radicular apical pulp [11]. The primary dentition is frequently subjected to stimuli such as trauma or caries with associated pulpal inflammation [12]. The same factors affect both the dentin–pulp responses in primary as well as permanent teeth, with respect to external stimuli.

2.6 The Deleterious Effects of Bacterial Infiltration at the Restorative Material Margins

A significant number of studies have implicated the presence of bacteria and their products as responsible for induction of the most severe forms of pulp inflammation. The role of bacteria in the inflammatory reaction was demonstrated by spontaneous healing of pulp exposures in germ-free animals [13] and subsequently by cavities restored with different materials and surface sealed with zinc oxide–eugenol cement to prevent any bacterial contamination originating from microleakage [14].

The presence of bacteria in cavities with a remaining dentin thickness (RDT) of less than 0.25 mm stimulates a more severe pulpal inflammatory reaction than in similar cavity preparations in the absence of bacteria [15]. Thus, the presence of bacteria always increases the mean grade of pulpal inflammation regardless of the RDT [16]. The presence of bacteria in class V cavity was also observed to result in a significant

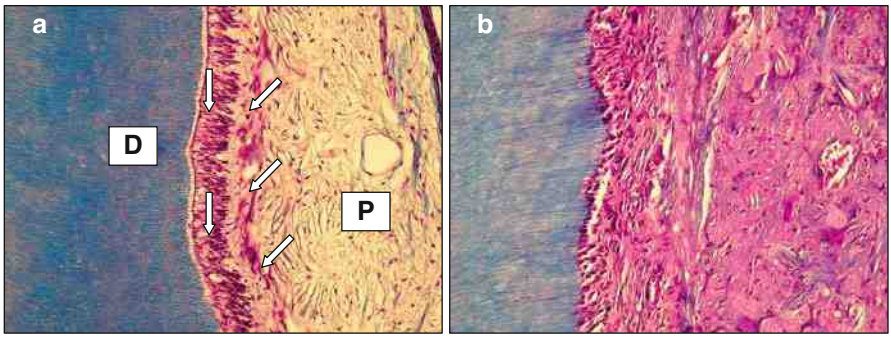

Fig. 2.10 (**a**) Section obtained from a young sound premolar in which a very deep class V cavity was prepared and the cavity floor lined with hard-setting calcium hydroxide cement. Note the pulp tissue with normal histological characteristics. Masson's trichrome, 64× (*D* Dentin, *P* Pulp; *vertical arrows* odontoblast layer; *oblique arrows* cell-rich zone). (**b**) In this human premolar, the cavity floor (dentin) was conditioned and a resin-based material was used as liner. Note the intense inflammatory response associated with complete pulp tissue disorganization. No microleakage at the cavity walls was observed after using a specific staining technique (Brown and Brenn) widely employed for disclosing bacteria. Masson's trichrome, 64×

decrease in the number of odontoblasts per unit area; this effect was more pronounced in deep cavities with RDT less than 0.5 mm than in cavities with RDT greater than 0.5 mm [16]. One can conclude that the ability to maintain an effective seal to protect the pulp from recurrent injury resulting from bacterial microleakage is a decisive factor in the clinical success of restorative products [17]. However, a number of studies performed in human teeth have shown pulpal inflammation in the absence of bacteria [8, 18–22], clearly indicating that other factors, such as the toxicity of dental material components capable of diffusing through dentinal tubules, are also responsible, at least in part, for pulp injury after restorative treatment (Fig. 2.10a, b).

2.7 The Protective Role of the Remaining Dentin Thickness (RDT)

It was found in an in vivo study that the cavity RDT is an important factor mediating pulpal inflammatory activity, particularly when the RDT is reduced to less than 0.3 mm [8, 22]. With an RDT less than 0.25 mm, a significant decrease in the number of odontoblasts was observed together with minimal reactionary dentin repair [23, 24]. It was recently demonstrated that very deep class V cavities prepared in human premolars (RDT thinner than 0.3 mm) which were subjected to adhesive restorations resulted in inflammatory pulp reaction associated with inner dentin resorption [8, 22, 25]. The presence of an RDT of more than 500 mm delays the diffusion of noxious materials into the dental pulp. In this clinical situation, the odontoblasts maintain their metabolism, or, in case of a slight stimulus, they may secrete a reactionary dentin, increasing the total distance between the restorative material and the pulp [8]. Any additional decrease in the dentin thickness to less than 500 mm results in a significant reduction in the number of odontoblasts.

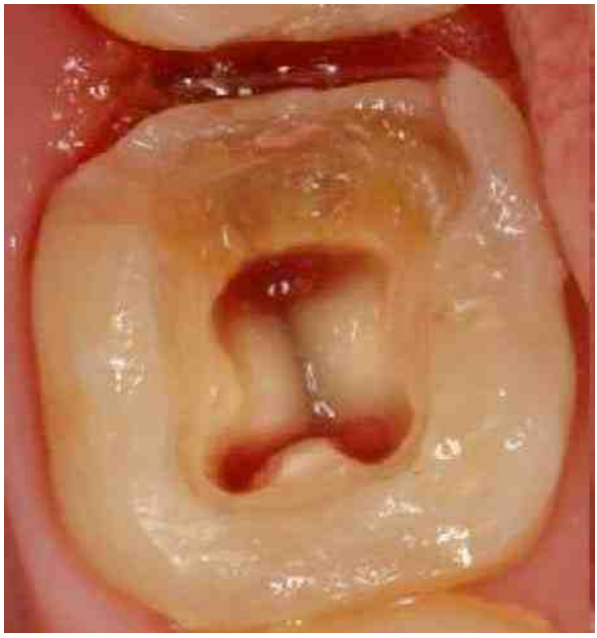

Fig. 5.10 Hemostasis obtained after placement of a dry cotton pellet

adverse events rendering MTA as, perhaps, the most exciting new pulp therapy material in decades.

One of the downsides of MTA is that the presence of iron renders the tooth a dark gray color. Recently white MTA that was supposed to reduce or eliminate the discoloration became available on the market. The results have been slightly inferior to those with gray MTA, and discoloration was still present. Another issue that has to be dealt with especially in the realm of pediatric dentistry is the cost of MTA.

In primary teeth, MTA is predominantly used for direct pulp capping and pulpotomy procedures. The major benefits of MTA are that it is biocompatible, it is bactericidal (high pH, 12.5), and it is able to stimulate cementum-like formation, osteoblastic adherence, and bone regeneration (see Chap. 2). Moreover, its sealing, mineralizing, dentinogenic, and osteogenic potentials make it the preferred choice for numerous clinical applications [48]. Success rates for MTA have ranged from 66 to 100 %, and results have not been too far different from those obtained with formocresol, ferric sulfate, or $NAOCl_2$ [18].

5.8.2.1 MTA Pulpotomy Technique

1. Once the pulp chamber is accessed, the coronal pulp is removed and hemostasis is achieved with a cotton pellet.
2. A 3:1 MTA to sterile saline is mixed into a paste and applied to the pulpal floor (Figs. 5.11 and 5.12).
3. ZOE or IRM is placed over the MTA and the tooth is restored.

3. During a subsequent restorative process, deep cavity preparations with RDTs between 250 and 40 mm lead to poor tertiary dentin repair activity [15]. These result from impaired odontoblast dentin secretory activity due to cellular injury [29]. The study demonstrated that the mean number of intact odontoblasts found beneath this kind of cavity preparation was 36% lower than the number found beneath similar preparations with an RDT between 500 and 250 mm. This lack in the ability of the odontoblasts to provide adequate pulp repair and pulp protection after deep cavity preparation has been supported by observations of a persistent inflammatory pulpal response and odontoblast displacement following such deep cavity preparations [29].

2.8 Clinical Recommendations

In clinical situations, conservative careful cavity preparations should be carried out: intermittent cutting movement, air/spray cooling, and use of new burs. In addition, biocompatible, antibacterial, and bioactive dental products must be used as liners to protect the pulp tissue against toxic components released from restorative materials capable of diffusing across the dentin [20, 30].

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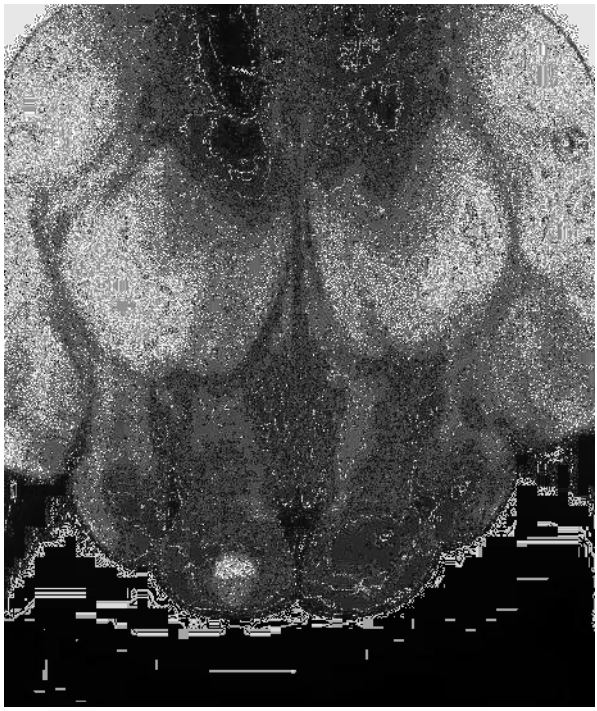

Fig. 5.15 A 2-year result showing a dentin bridge under the pulpotomy area and a vital pulp

partial pulpotomies has had very little success and as a technique is not advocated. It has been shown that $CaOH_2$ has no beneficial effect on inflamed pulps [54]. However, under "sterile" conditions, the use of partial pulpotomy is something that may need to be reconsidered as a possible treatment alternative [55]. The authors suggested performing partial pulpotomy to preserve pulp vitality in a young primary tooth with a wide open apex and thin root dentin walls (Fig. 5.15).

Petel and Fuks (personal communication) have reported the use of this technique for the resolution of idiopathic internal resorption [56].

5.10 Summary

This chapter has presented a number of materials that have been used as pulpotomy medicaments since the early 1900s, as well as alternative thinking and revisiting of old techniques in a new context.

A recent Cochrane Review of pulpotomy medicaments showed that none of the materials was significantly superior, although the trend was for better results with MTA and ferric sulfate [49].

It is possible that the reason why pulpotomies do not present 100 % success may be due to inadequate case selection. If the pulp tissue is truly unaffected or minimally affected, then there should be no reason why pulp treatment should fail. One

should always keep in mind that bleeding status may or may not be a true indicator of pulp status. In case of a carious exposure, one can assume that the radicular pulp may have also been adversely affected. The decision may have to be to perform a pulpectomy instead of a pulpotomy, since the radicular pulp may have already begun a process of degeneration, or to perform a pulpotomy with an expected less favorable prognosis.

Therefore, clinically, one should take into consideration numerous factors before deciding what treatment to perform. These factors include:

1. Pulp status
2. Extent of the carious lesion
3. Age of the patient at the time of treatment
4. Goals of the treatment
5. Cost of the treatment

If the child is very young and there is a carious exposure, a pulpectomy may be the treatment of choice. Conversely in an older child with a similar carious exposure, where no long-term maintenance is needed, a pulpotomy may be adequate with any of the materials discussed.

In asymptomatic carious teeth where the lesion radiographically appears close but not overlapping with the pulp space, or in cases when a clear barrier between the caries and the pulp is evident, an indirect pulp treatment (IPT) may be the recommended technique regardless of the age of the child.

Diagnosis is the key to the success of any of these materials and techniques and should always be at the forefront of our thought process when making any decision on pulp therapy.

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Pediatric Endodontics

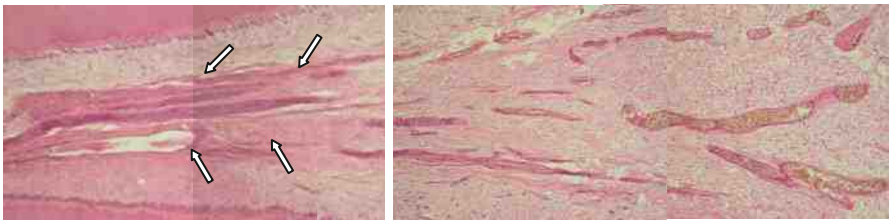

Fig. 2.4 Sections obtained from human sound teeth showing the radicular (*left*) and coronal (*right*) portions of the pulp tissue. Note the fibrous connective pulp tissue with vascular–nervous sheath close to the apical foramen (*arrows*). Conversely, the coronal pulp exhibits loose connective tissue with a number of blood vessels. H/E, 32×

production of dentin, the pulp remains enclosed within the central part of the tooth, having a coronal and a radicular portion. In uni-radicular teeth, the coronal and radicular pulp tissues are contiguous, but in multi-radicular teeth, the floor of the pulp chamber has a clear distinction: the coronal pulp is rich in cells and extracellular matrix, while the radicular pulp has more fibers, and the vascular–nervous sheath is more concentrated, with less anastomosis (Fig. 2.4).

2.3.1 Odontoblasts

The odontoblasts have been traditionally described as cells lining the periphery of the pulpal space and extending their cytoplasmic processes into the dentinal tubules. These cells have several junctions, which allow for intercellular communication and help to maintain the relative position of one cell to another. In young permanent teeth, the pulp tissue exhibits defined zones. The cell-free zone is located just below the odontoblastic layer and contains an extensive plexus of unmyelinated nerves and blood capillaries. The cell-rich zone, which presents a number of undifferentiated mesenchymal cells, is observed adjacent to the cell-free zone. The core of the dental pulp contains larger blood vessels and nerves, which are surrounded by large area of extracellular matrix. This pulp morphology is similar to that observed in primary teeth, but the zones are not so well defined (Fig. 2.5a, b).

Although this description is correct during active dentinogenesis, it is now accepted that the size of the odontoblasts and the content of their cytoplasmic organelles vary throughout their life cycle and are closely related to their functional activity. The relationship between the size of the odontoblasts and their secretory activity can be demonstrated by differences in their size in the crown and in the root, and different dentinogenic rates may be expressed in these two areas of the tooth [7].

The odontoblasts are highly specialized cells and are responsible for the formation of dentin. Due to the extension of their cytoplasmic processes into the dentinal tubules, these cells compose the main part of the dentin–pulp complex. When this complex is damaged by disease or attrition, or is affected by operative procedures, it reacts in an attempt to defend the pulp tissue.

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This book is dedicated to
Moises Fuks, my beloved husband and
long-term companion and friend
and to
Tamar, Neta and Alona Peretz, my beloved
and precious family

Anna B. Fuks and Benjamin Peretz

Preface

The initial idea for writing this book came because we felt that there has been an explosion of scientific knowledge on the understanding of the pulp tissue in the last two decades, which, in turn, affect the proper treatment for various pulp pathological conditions. This immense advancement has included the primary pulp also, and pediatric dentistry today, with regard to pulp treatment, can provide a better, more problem-oriented therapy and treatment to the affected primary pulp. Therefore, when we were approached by the Springer representative to write this book, we gladly agreed.

We felt that there was a need for students, undergraduate and postgraduate alike, as well as for the professional community to be familiarized with the current "state of the art" on pediatric endodontics. We made all efforts to cover the various aspects of the dentin-pulp complex in pediatric dentistry: from the understanding of biological concepts of the healthy pulp, through the pulp reactions to the deleterious effects of caries, to the various treatment modalities for each type of pulp injury, to the adverse reactions to various pulp dressing materials, and to the postoperative prognosis.

The better understanding of these topics led us to conclude that a conservative approach in the treatment of reversibly inflamed pulp needs to be emphasized. Thus, considerable attention has been given to the conservative approach to pulp treatment in primary and young permanent teeth. Our message stresses the paradigm shift toward conservative treatment modalities, relying on an accurate diagnosis based on signs and symptoms to assess the appropriateness of the technique for a specific case.

Notwithstanding, the traditional modes of treatment are also covered.

Understanding the new concepts regarding pulp treatment will guide practicing pediatric dentists and general dentists to select the proper mode of treatment.

A special emphasis has also been given to the future of pulp treatment, in light of the innovative knowledge on stem cells. At present, there is a consensus that the future of medicine and dentistry, particularly of pulp treatment, lies in the thorough research on stem cells.

We hope that this text will be useful to all students and dentists who treat children, to provide a better care for their teeth.

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Tel Aviv, Israel — Benjamin Peretz

Contents

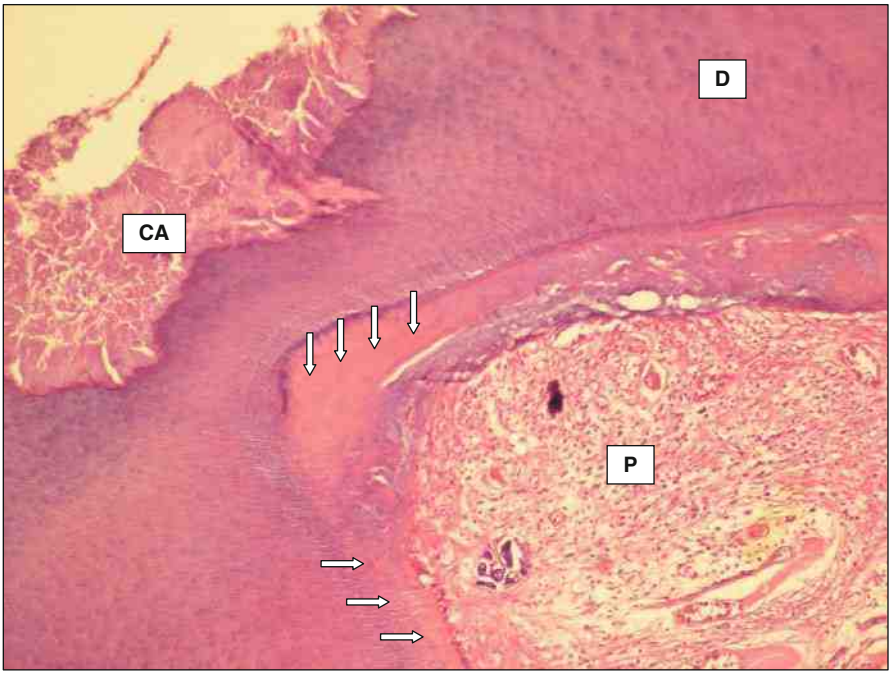

Fig. 2.7 Section obtained from primary teeth with acute occlusal caries (*CA*). Note the layer of tubular reactionary dentin (*horizontal arrows*) deposited in the lower part of the picture where the primary odontoblasts were far from the intense stimulus (pay attention to the dentinal tubule orientation). However, in the upper area of the picture, where the pulp is close to the very deep caries lesion (*CA*), a thick layer of reparative dentin with no tubules, termed as reparative dentin (*vertical arrows*), can be observed. H/E, 32× (*D* Dentin, *P* Pulp)

calcospherites (Fig. 2.8). Failure of the calcospherites to fuse leads to the formation of hypomineralized areas, known as interglobular dentin. These areas are more visible in the radicular dentin, where the dentin is produced simultaneously with the eruptive process, and on the most external portion of the coronal dentin, at the limit between the mantle and the circumpulpal dentin. Predentin consists mainly of types I and III collagen, glycoproteins, and proteoglycans.

Another type of hypocalcification is the Tomes' grainy layer that is formed by the terminal loops and branches of the odontoblastic membrane. This membrane configuration is developed during the formation of the radicular dentin, giving to the peripheral dentin a grainy appearance.

Dentin is composed of tubules. As the odontoblasts secrete the organic matrix, they emit a projection that is surrounded by liquid, providing the tubular aspect. The tubules have a lightly conical shape, due to the mineralization process of the peritubular dentin that occurs throughout the life of the tooth. The tubules extend through the entire thickness of the dentin, following the sinuous track of the odontoblasts. The number of dentinal tubules varies according to the area when different teeth are

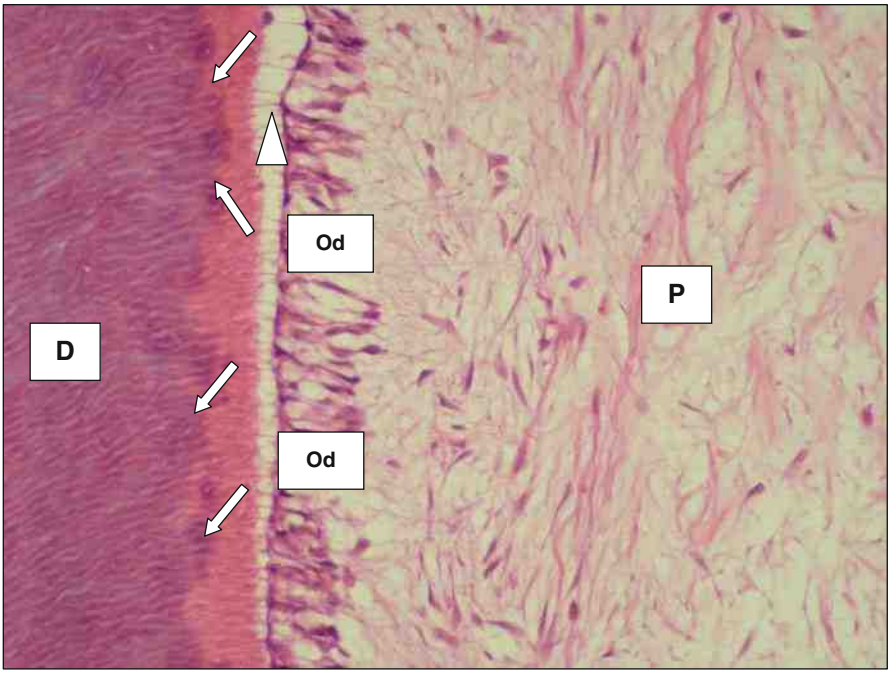

Fig. 2.8 Section obtained from sound primary teeth. Between the tubular dentin and the thin layer of predentin one can see the fronts of mineralization – calcospherites (*oblique arrows*). Note the odontoblasts (*Od*) with their cytoplasmic processes which get inside the dentinal tubules (*head arrow*) *D* dentin. *P* pulp. H/E, 125×

compared. It has been demonstrated that at the enamel–dentin junction (superficial dentin), there are approximately 20,000 tubules/mm^2, while near the predentin (deep dentin), this number increases to approximately 75,000 tubules/mm^2.

The dentin surrounding the periphery of the dentinal tubules is known as peritubular or intertubular dentin. Intertubular dentin is present between dentinal tubules. The odontoblast cytoplasmic processes remain within the dentinal tubules (Fig. 2.8).

Communications among the dentinal tubules, known as dentinal canaliculus, are frequently observed. The peritubular dentin that constitutes the walls of the dentinal tubules is four times harder than intertubular dentin, since it consists of approximately 96 % of hydroxyapatite crystals. Mild stimuli from the external environment, such as attrition and caries, may cause obliteration of the dentinal tubules, resulting in dentin sclerosis. Intertubular dentin is partially composed of collagen fibrils positioned perpendicularly to the long axis of the tubules, surrounding the tubules (Fig. 2.9a, b). The conditioning of the dentin substrate with acidic agents or chelating substances decreases or removes the peritubular dentin on the surface, leaving a mesh of intertubular collagen exposed to the action of bonding agents or to bacteria from decay [9, 10].

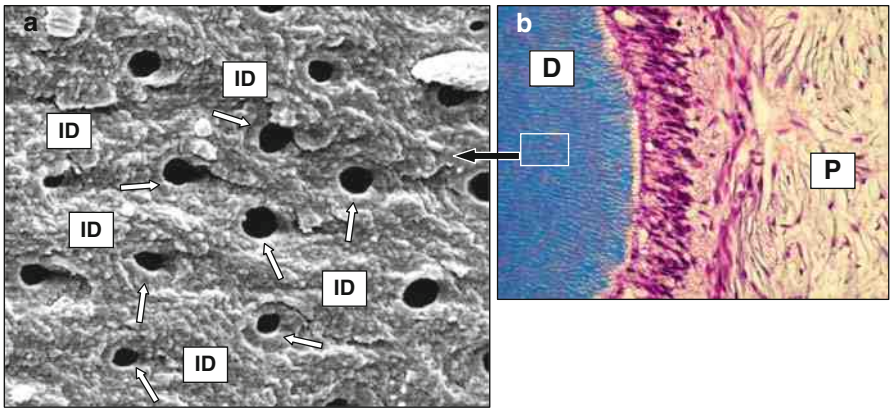

Fig. 2.9 (**a**) Morphology of the dentin structure. Note the dentinal tubules surrounded by peritubular dentin (*arrows*) as well as a large area of intertubular dentin (*ID*). MEV, 3.000×. (**b**) Dentin–pulp complex. *D* dentin. *P* pulp. Cytoplasmic processes from odontoblasts are observed inside the dentinal tubules. Masson's trichrome, 125×

2.5 Factors Affecting the Dentin–Pulp Complex Response to Stimuli in Primary Teeth

Although the life span of primary teeth is shorter and their dentin is thinner when compared to that of permanent teeth, the dentin–pulp complex response to dental caries in human primary teeth is similar to that of permanent teeth, including a reduction in the number of the odontoblasts and an increase in the number of inflammatory cells. These are found under very deep lesions and are less numerous in more distant regions, being almost absent in the radicular apical pulp [11]. The primary dentition is frequently subjected to stimuli such as trauma or caries with associated pulpal inflammation [12]. The same factors affect both the dentin–pulp responses in primary as well as permanent teeth, with respect to external stimuli.

2.6 The Deleterious Effects of Bacterial Infiltration at the Restorative Material Margins

A significant number of studies have implicated the presence of bacteria and their products as responsible for induction of the most severe forms of pulp inflammation. The role of bacteria in the inflammatory reaction was demonstrated by spontaneous healing of pulp exposures in germ-free animals [13] and subsequently by cavities restored with different materials and surface sealed with zinc oxide–eugenol cement to prevent any bacterial contamination originating from microleakage [14].

The presence of bacteria in cavities with a remaining dentin thickness (RDT) of less than 0.25 mm stimulates a more severe pulpal inflammatory reaction than in similar cavity preparations in the absence of bacteria [15]. Thus, the presence of bacteria always increases the mean grade of pulpal inflammation regardless of the RDT [16]. The presence of bacteria in class V cavity was also observed to result in a significant

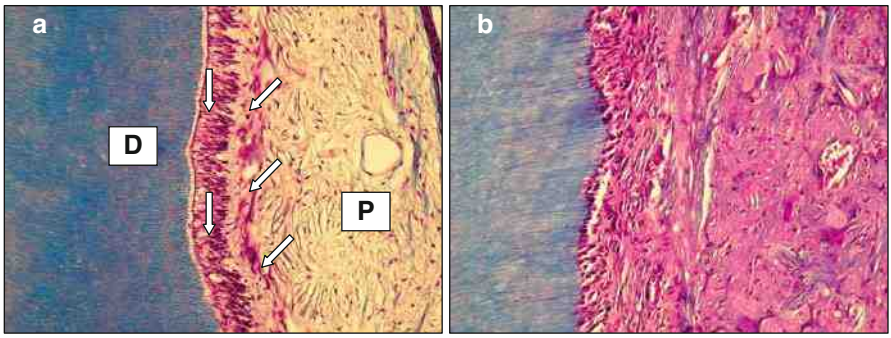

Fig. 2.10 (**a**) Section obtained from a young sound premolar in which a very deep class V cavity was prepared and the cavity floor lined with hard-setting calcium hydroxide cement. Note the pulp tissue with normal histological characteristics. Masson's trichrome, 64× (*D* Dentin, *P* Pulp; *vertical arrows* odontoblast layer; *oblique arrows* cell-rich zone). (**b**) In this human premolar, the cavity floor (dentin) was conditioned and a resin-based material was used as liner. Note the intense inflammatory response associated with complete pulp tissue disorganization. No microleakage at the cavity walls was observed after using a specific staining technique (Brown and Brenn) widely employed for disclosing bacteria. Masson's trichrome, 64×

decrease in the number of odontoblasts per unit area; this effect was more pronounced in deep cavities with RDT less than 0.5 mm than in cavities with RDT greater than 0.5 mm [16]. One can conclude that the ability to maintain an effective seal to protect the pulp from recurrent injury resulting from bacterial microleakage is a decisive factor in the clinical success of restorative products [17]. However, a number of studies performed in human teeth have shown pulpal inflammation in the absence of bacteria [8, 18–22], clearly indicating that other factors, such as the toxicity of dental material components capable of diffusing through dentinal tubules, are also responsible, at least in part, for pulp injury after restorative treatment (Fig. 2.10a, b).

2.7 The Protective Role of the Remaining Dentin Thickness (RDT)

It was found in an in vivo study that the cavity RDT is an important factor mediating pulpal inflammatory activity, particularly when the RDT is reduced to less than 0.3 mm [8, 22]. With an RDT less than 0.25 mm, a significant decrease in the number of odontoblasts was observed together with minimal reactionary dentin repair [23, 24]. It was recently demonstrated that very deep class V cavities prepared in human premolars (RDT thinner than 0.3 mm) which were subjected to adhesive restorations resulted in inflammatory pulp reaction associated with inner dentin resorption [8, 22, 25]. The presence of an RDT of more than 500 mm delays the diffusion of noxious materials into the dental pulp. In this clinical situation, the odontoblasts maintain their metabolism, or, in case of a slight stimulus, they may secrete a reactionary dentin, increasing the total distance between the restorative material and the pulp [8]. Any additional decrease in the dentin thickness to less than 500 mm results in a significant reduction in the number of odontoblasts.

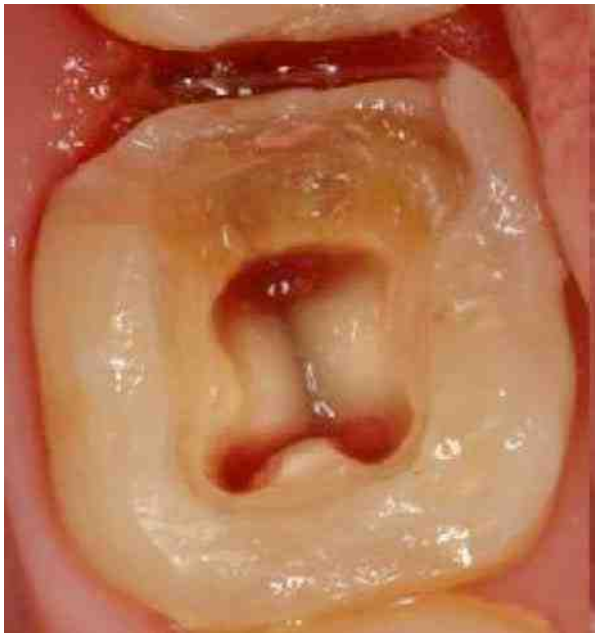

Fig. 5.10 Hemostasis obtained after placement of a dry cotton pellet

adverse events rendering MTA as, perhaps, the most exciting new pulp therapy material in decades.

One of the downsides of MTA is that the presence of iron renders the tooth a dark gray color. Recently white MTA that was supposed to reduce or eliminate the discoloration became available on the market. The results have been slightly inferior to those with gray MTA, and discoloration was still present. Another issue that has to be dealt with especially in the realm of pediatric dentistry is the cost of MTA.

In primary teeth, MTA is predominantly used for direct pulp capping and pulpotomy procedures. The major benefits of MTA are that it is biocompatible, it is bactericidal (high pH, 12.5), and it is able to stimulate cementum-like formation, osteoblastic adherence, and bone regeneration (see Chap. 2). Moreover, its sealing, mineralizing, dentinogenic, and osteogenic potentials make it the preferred choice for numerous clinical applications [48]. Success rates for MTA have ranged from 66 to 100 %, and results have not been too far different from those obtained with formocresol, ferric sulfate, or $NAOCl_2$ [18].

5.8.2.1 MTA Pulpotomy Technique

1. Once the pulp chamber is accessed, the coronal pulp is removed and hemostasis is achieved with a cotton pellet.
2. A 3:1 MTA to sterile saline is mixed into a paste and applied to the pulpal floor (Figs. 5.11 and 5.12).
3. ZOE or IRM is placed over the MTA and the tooth is restored.

3. During a subsequent restorative process, deep cavity preparations with RDTs between 250 and 40 mm lead to poor tertiary dentin repair activity [15]. These result from impaired odontoblast dentin secretory activity due to cellular injury [29]. The study demonstrated that the mean number of intact odontoblasts found beneath this kind of cavity preparation was 36% lower than the number found beneath similar preparations with an RDT between 500 and 250 mm. This lack in the ability of the odontoblasts to provide adequate pulp repair and pulp protection after deep cavity preparation has been supported by observations of a persistent inflammatory pulpal response and odontoblast displacement following such deep cavity preparations [29].

2.8 Clinical Recommendations

In clinical situations, conservative careful cavity preparations should be carried out: intermittent cutting movement, air/spray cooling, and use of new burs. In addition, biocompatible, antibacterial, and bioactive dental products must be used as liners to protect the pulp tissue against toxic components released from restorative materials capable of diffusing across the dentin [20, 30].

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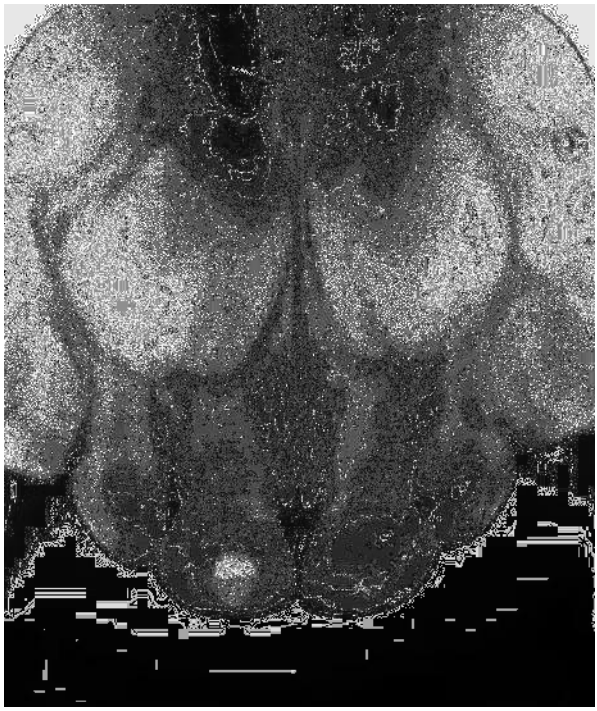

Fig. 5.15 A 2-year result showing a dentin bridge under the pulpotomy area and a vital pulp

partial pulpotomies has had very little success and as a technique is not advocated. It has been shown that $CaOH_2$ has no beneficial effect on inflamed pulps [54]. However, under "sterile" conditions, the use of partial pulpotomy is something that may need to be reconsidered as a possible treatment alternative [55]. The authors suggested performing partial pulpotomy to preserve pulp vitality in a young primary tooth with a wide open apex and thin root dentin walls (Fig. 5.15).

Petel and Fuks (personal communication) have reported the use of this technique for the resolution of idiopathic internal resorption [56].

5.10 Summary

This chapter has presented a number of materials that have been used as pulpotomy medicaments since the early 1900s, as well as alternative thinking and revisiting of old techniques in a new context.

A recent Cochrane Review of pulpotomy medicaments showed that none of the materials was significantly superior, although the trend was for better results with MTA and ferric sulfate [49].

It is possible that the reason why pulpotomies do not present 100 % success may be due to inadequate case selection. If the pulp tissue is truly unaffected or minimally affected, then there should be no reason why pulp treatment should fail. One

should always keep in mind that bleeding status may or may not be a true indicator of pulp status. In case of a carious exposure, one can assume that the radicular pulp may have also been adversely affected. The decision may have to be to perform a pulpectomy instead of a pulpotomy, since the radicular pulp may have already begun a process of degeneration, or to perform a pulpotomy with an expected less favorable prognosis.

Therefore, clinically, one should take into consideration numerous factors before deciding what treatment to perform. These factors include:

1. Pulp status
2. Extent of the carious lesion
3. Age of the patient at the time of treatment
4. Goals of the treatment
5. Cost of the treatment

If the child is very young and there is a carious exposure, a pulpectomy may be the treatment of choice. Conversely in an older child with a similar carious exposure, where no long-term maintenance is needed, a pulpotomy may be adequate with any of the materials discussed.

In asymptomatic carious teeth where the lesion radiographically appears close but not overlapping with the pulp space, or in cases when a clear barrier between the caries and the pulp is evident, an indirect pulp treatment (IPT) may be the recommended technique regardless of the age of the child.

Diagnosis is the key to the success of any of these materials and techniques and should always be at the forefront of our thought process when making any decision on pulp therapy.

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associated with an increased failure rate when compared to underfilling or flush finishing [19, 51].

Studies have shown that remnants of ZOE may be found in the alveolar bone in 50 % and up to 70 % of the exfoliated primary teeth, and this material was still retained in more than a quarter of the patients after three years [16, 42, 79]. Figure 6.13 demonstrates a case of a ZOE-overfilled primary central incisor whose remnants remained in the tissue even after the eruption of the succedaneous permanent incisor.

According to Coll and Sadrian, RCT correctly done do not cause adverse effects on the succedaneous teeth, but have a 20 % chance of altering the path of permanent tooth eruption when using ZOE filling material [1]. Incorrectly performed root canal treatment may cause arrest of eruption of the succedaneous teeth [90]. Ectopic eruption of permanent incisors following ZOE pulpectomy of their primary predecessors was also described by Tannure et al. [91].

It could be hypothesized that ectopic eruption of a permanent incisor might not be caused by a RCT on the primary incisor but due to severe trauma to the primary incisor affecting and/or dislodging the developing permanent bud.

Studies have shown either no or low incidence of enamel hypoplasia in succedaneous teeth when the primary teeth have been treated with pulpectomy [1, 16].

Failure of root canal treatment occurred in 3.3 % of the primary molars that presented with a new radiolucent lesion or enlargement of existing periapical radiolucency after being treated with zinc oxide and iodoform paste. When a periradicular radiolucent lesion is present before the RCT, the likelihood of failure (e.g., increase in size of the lesion) is higher than in the absence of such a defect before the treatment. No association was found between RCT in primary molars and the appearance of enamel defects or ectopic eruption of the following premolars [92].

Previous studies raised the possibility of radicular cysts developing in concurrence with low-grade irritation to the dental sac of a permanent successor by root canal filling material leaking from resorbed primary apices [92, 93]. In those studies, enlargement of the dental sac in association with a root-treated primary tooth occurred in 3.3 % of the followed cases, but the development of a true radicular or dentigerous cysts was a rare occasion. Despite the low occurrence, dentists should be aware of this phenomenon and radiographically monitor root canal treated teeth until shedding (Fig. 6.16).

Most radicular cysts in the primary dentition do not demonstrate clinical signs, but if the cyst attains a certain size, displacement of the developing tooth bud might occur [1, 14, 94, 95], accompanied by expansion of the buccal cortical plate (Fig. 6.17) [94, 95].

6.10.1 RCT in Canines and Incisors

Iodoform pastes have better resorbability and disinfectant properties than ZOE, but commonly produce a dark-brown discoloration of the tooth crowns compromising the esthetics [30, 33, 96].

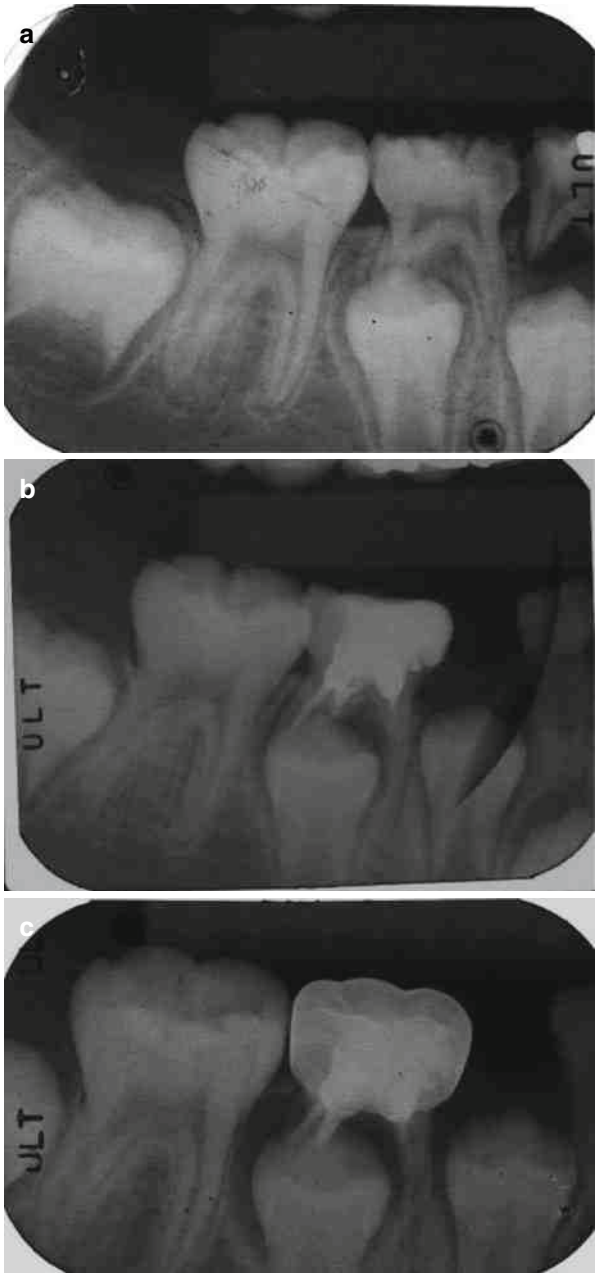

Fig. 6.16 Root canal treatment using Endoflas, performed in a primary right second mandibular molar. Figures (**a**, **b**) are pre- and postoperative radiographs. Radiograph (**c**) demonstrates enlargement of the dental sac of the mandibular right second premolar 3 months later

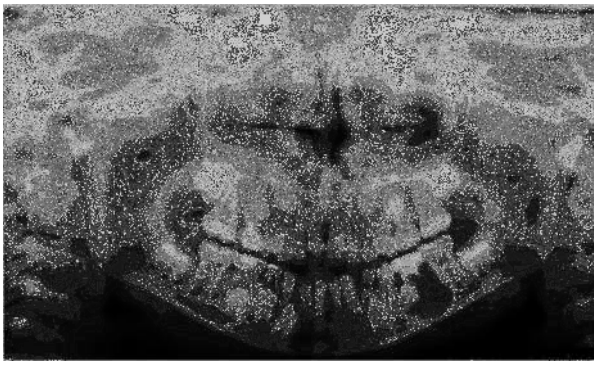

Fig. 6.17 A radicular cyst associated with a lower second primary molar with a root canal treatment. The tooth germ is deflected mesially. The large cystic lesion may endanger the lower border of the mandible (Courtesy of Emanuel Twito, DMD)

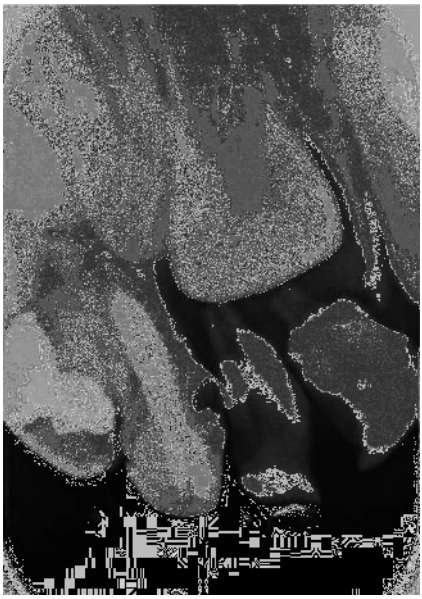

Fig. 6.18 Primary right canine with a classic SURTEX® Dentatus prefabricated metal post (Courtesy of Maya Dotan, DMD)

6.10.2 Metal Post

Figure 6.18 illustrates a primary upper right canine with a classic SURTEX® Dentatus prefabricated metal post. This procedure is not acceptable in primary teeth as the roots are predestined to resorb and the metal post may injure the permanent tooth bud. Furthermore the post and core are not leakage-free and as demonstrated in this case. The root canal filling material is washed away from the canal exposing it to reinfection. In case of unrestorable teeth, the preferred option is extraction.

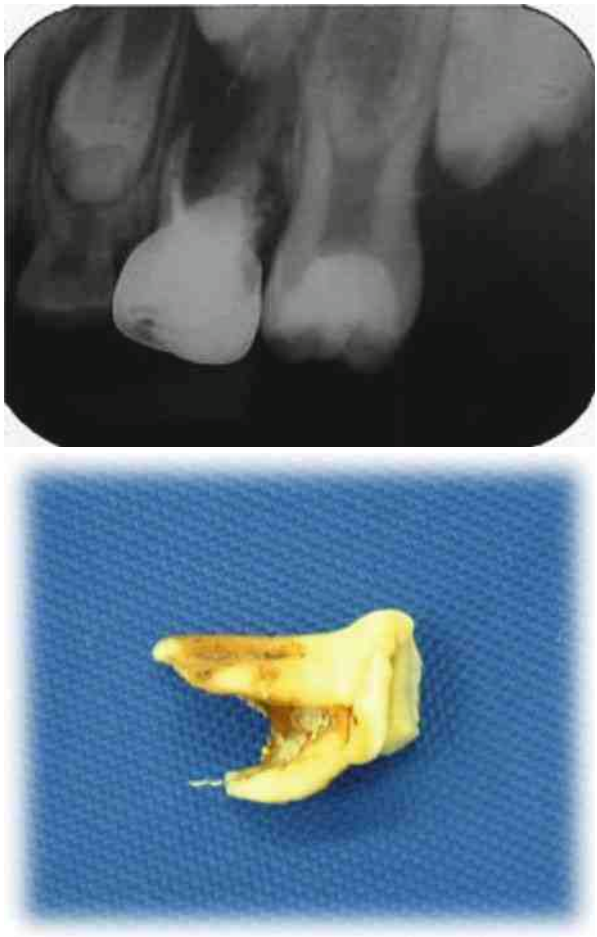

Fig. 6.19 (**a**) Radiographic view of a broken lentulo spiral filler in a canal post RCT. (**b**) The tooth was extracted. The lentulo spiral filler is bulging from the apex

6.10.3 Lentulo Spiral Fillers

Lentulo spiral fillers are dental instruments with constantly spaced spirals used to distribute sealer or cement in root canals. Care must be taken to avoid the possibility of the spiral breaking inside the root canal. Clinicians should always perform correct use of the instrument especially in primary teeth with tortuous root canals and dispose of old instruments. In case of a broken instrument in the canal, one should consider extracting the tooth or performing a close follow-up and extracting the tooth as soon as the tooth bud is approaching the edge of the lentulo in the resorbing root (Fig. 6.19).

6.10.4 Turner Tooth

An enamel defect in the permanent teeth caused by periapical inflammatory infection in the overlying primary tooth is referred to as Turner's tooth. Turner's hypoplasia is the result of disruption in the process of enamel matrix formation, which in

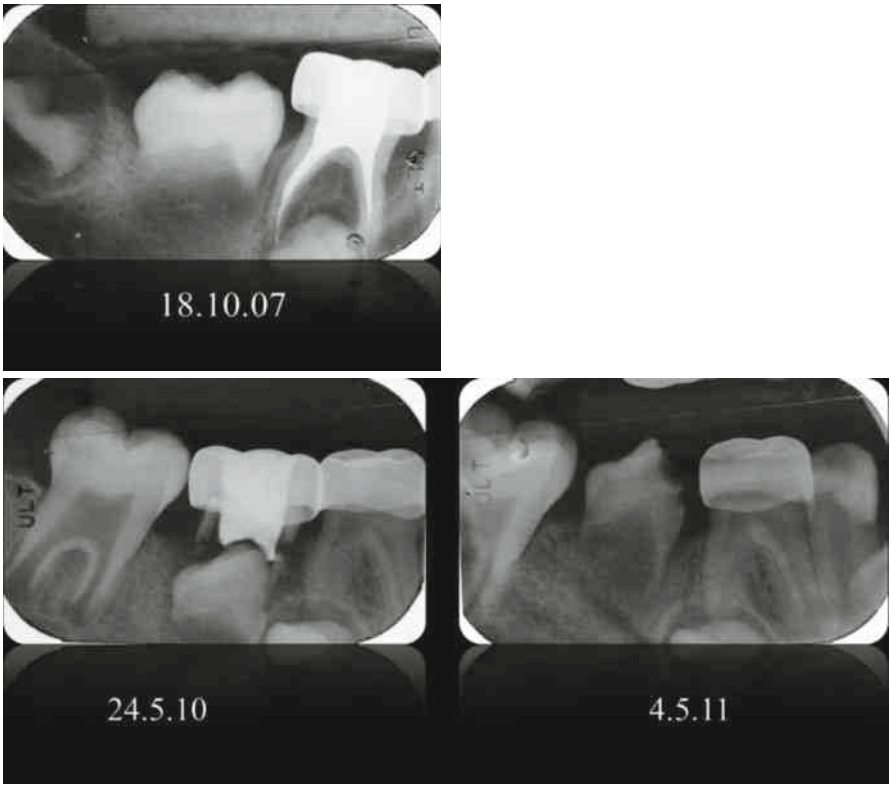

Fig. 6.20 Although rare, hypoplasia in the permanent successor as a sequela to a RCT in a primary tooth may occur even after a successful RCT (Courtesy of Gal Wallenstein, DMD)

turn causes both quality and thickness of enamel to be defective (See Chap. 2). Turner's hypoplasias in permanent teeth that resulted from periapical lesions in non-vital primary teeth have been reported [97].

Although rare, hypoplasia in the permanent successor as a sequela to a RCT in a primary tooth may occur even after a successful RCT. Those incidences emphasize the crucial need for periodical follow-up visits to evaluate the treated primary tooth (Fig. 6.20).

6.11 Case: Root Canal Treatment in a Primary Molar

A healthy 5-year-old Caucasian boy presented at the clinic. Patient appeared apprehensive and uncooperative, objecting dental examination. Father stated that the child "had severe pain awaking him from sleep a few weeks ago." Intraoral examination revealed soft tissue swelling and redness around the lower right primary first molar.

Two bitewing x-rays were performed (periapical x-rays of the lower right second primary molar was not taken) (Fig. 6.21). Based on the history of pain, the clinical examination, and radiographic findings, the most probable diagnosis was exacerbation of chronic dentoalveolar abscess in the lower right first primary molar.

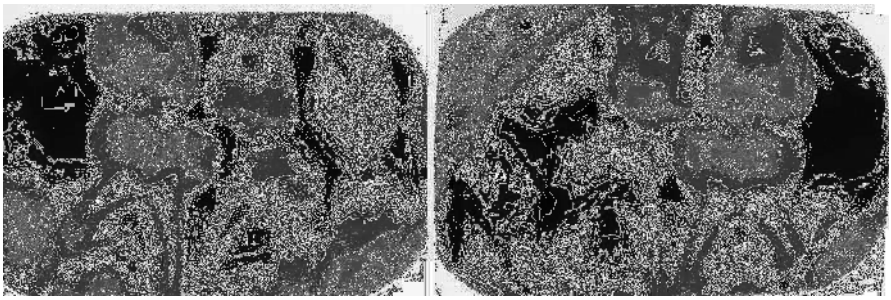

Fig. 6.21 Two bitewing x-rays were performed the first time the child presented, 30.8.12

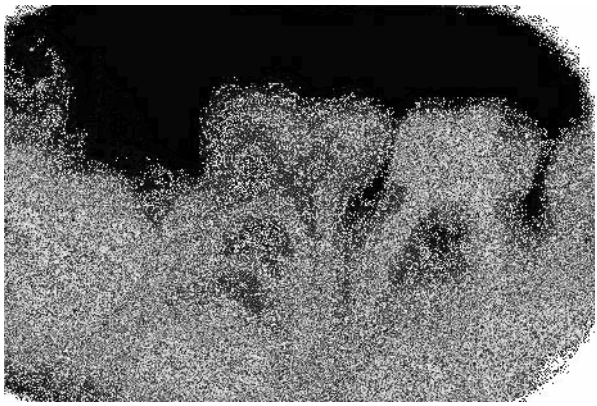

Fig. 6.22 RCT was performed as an emergency treatment in the lower right mandibular first primary molar, 30.8.12

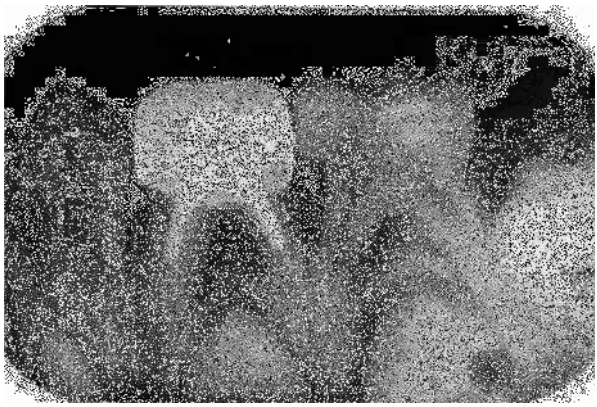

Fig. 6.23 Post-op x-ray of RCT in the lower left mandibular first primary molar, 23.5.13

On the same appointment, using conscious sedation, RCT was performed as an emergency treatment in the lower right mandibular first primary molar (Fig. 6.22). Due to parents delaying the next appointment, another emergency treatment using conscious sedation was performed, this time a RCT in the lower left mandibular first primary molar (Fig. 6.23). Comprehensive treatment of other carious lesions was accomplished using inhaled sedation.

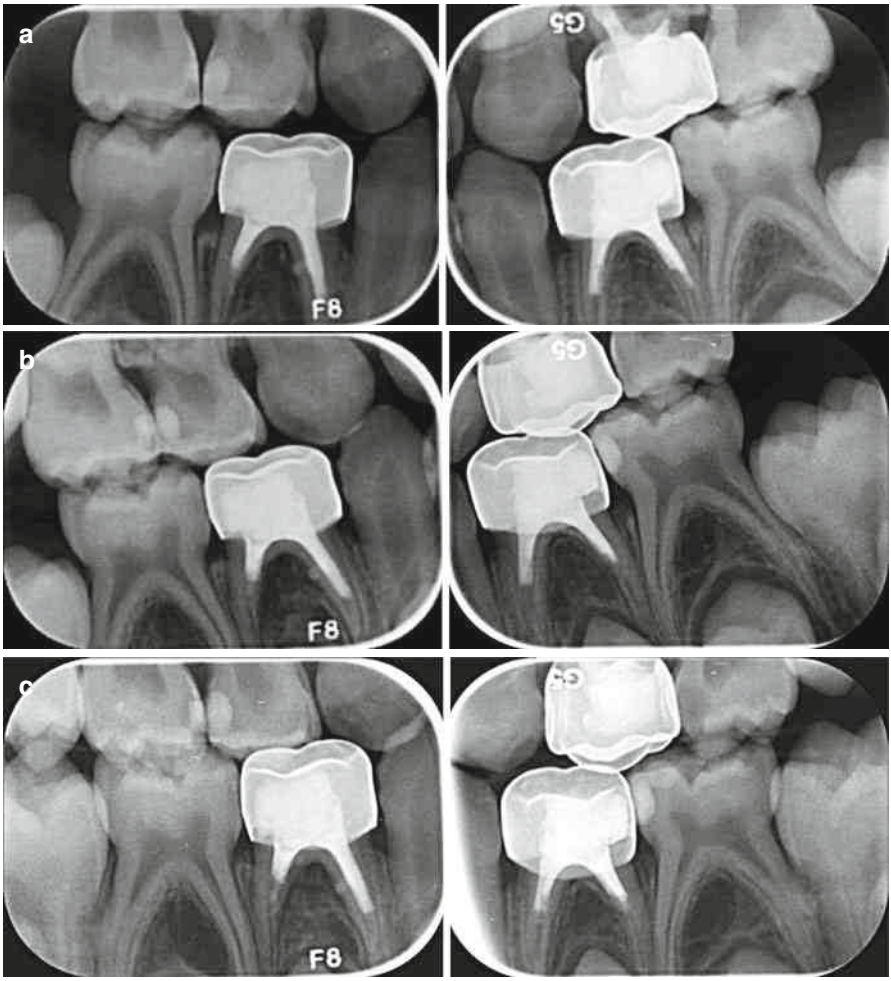

Fig. 6.24 Recall appointments demonstrated new carious lesions that needed treatment, but both (lower first mandibular teeth) RCT teeth appeared to have no clinical or radiographic pathologies. (**a**) First recall appointment after 15 month, (**b**) Second recall appointment 23 months after treatment and (**c**) Third recall appointment 2 years and 9 months after the RCT treatment.

During the next 2 years and 9 month, recall appointments demonstrated new carious lesions that needed treatment, but both (lower first mandibular teeth) RCT teeth appeared to have no clinical or radiographic pathologies (Fig. 6.24).

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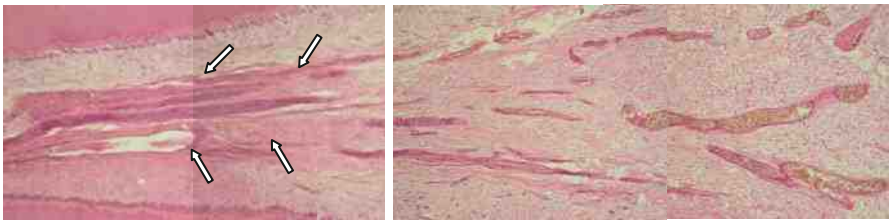


Fig. 2.4 Sections obtained from human sound teeth showing the radicular (*left*) and coronal (*right*) portions of the pulp tissue. Note the fibrous connective pulp tissue with vascular–nervous sheath close to the apical foramen (*arrows*). Conversely, the coronal pulp exhibits loose connective tissue with a number of blood vessels. H/E, 32×

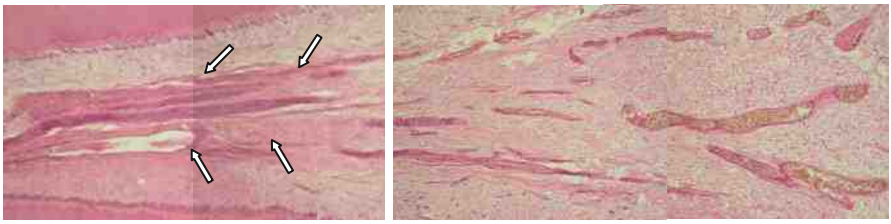
production of dentin, the pulp remains enclosed within the central part of the tooth, having a coronal and a radicular portion. In uni-radicular teeth, the coronal and radicular pulp tissues are contiguous, but in multi-radicular teeth, the floor of the pulp chamber has a clear distinction: the coronal pulp is rich in cells and extracellular matrix, while the radicular pulp has more fibers, and the vascular–nervous sheath is more concentrated, with less anastomosis (Fig. 2.4).

2.3.1 Odontoblasts

The odontoblasts have been traditionally described as cells lining the periphery of the pulpal space and extending their cytoplasmic processes into the dentinal tubules. These cells have several junctions, which allow for intercellular communication and help to maintain the relative position of one cell to another. In young permanent teeth, the pulp tissue exhibits defined zones. The cell-free zone is located just below the odontoblastic layer and contains an extensive plexus of unmyelinated nerves and blood capillaries. The cell-rich zone, which presents a number of undifferentiated mesenchymal cells, is observed adjacent to the cell-free zone. The core of the dental pulp contains larger blood vessels and nerves, which are surrounded by large area of extracellular matrix. This pulp morphology is similar to that observed in primary teeth, but the zones are not so well defined (Fig. 2.5a, b).

Although this description is correct during active dentinogenesis, it is now accepted that the size of the odontoblasts and the content of their cytoplasmic organelles vary throughout their life cycle and are closely related to their functional activity. The relationship between the size of the odontoblasts and their secretory activity can be demonstrated by differences in their size in the crown and in the root, and different dentinogenic rates may be expressed in these two areas of the tooth [7].

The odontoblasts are highly specialized cells and are responsible for the formation of dentin. Due to the extension of their cytoplasmic processes into the dentinal tubules, these cells compose the main part of the dentin–pulp complex. When this complex is damaged by disease or attrition, or is affected by operative procedures, it reacts in an attempt to defend the pulp tissue.



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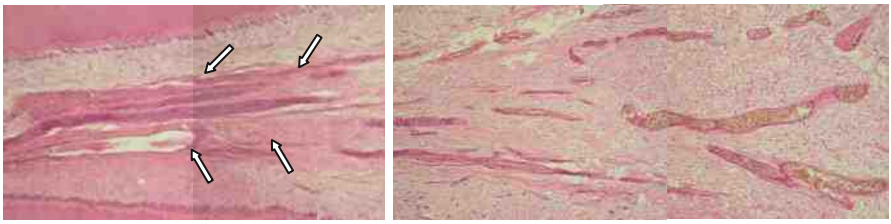

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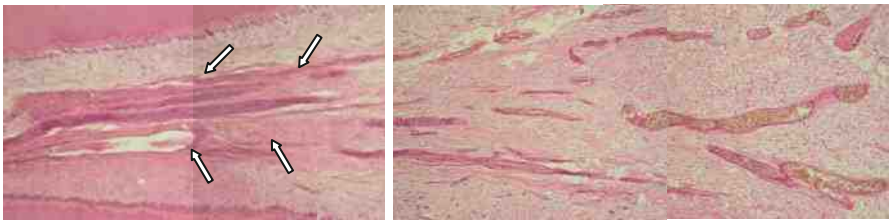

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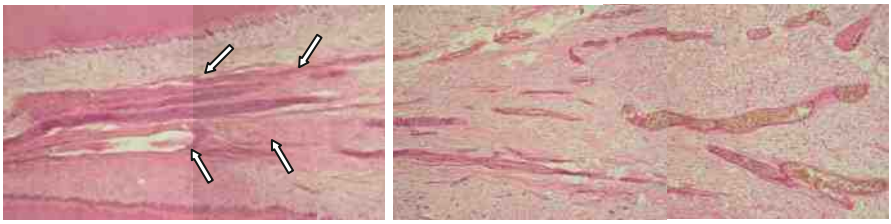

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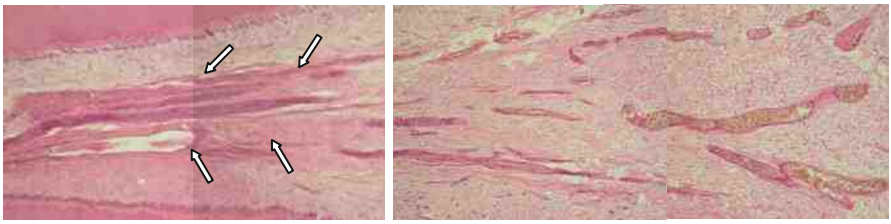

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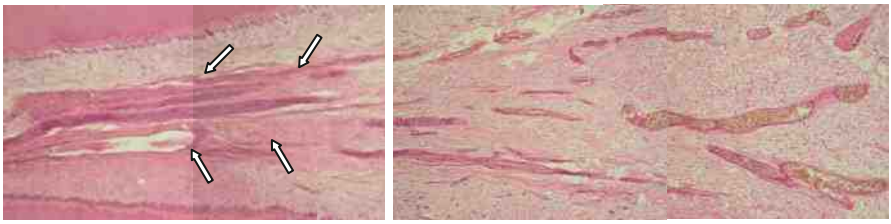

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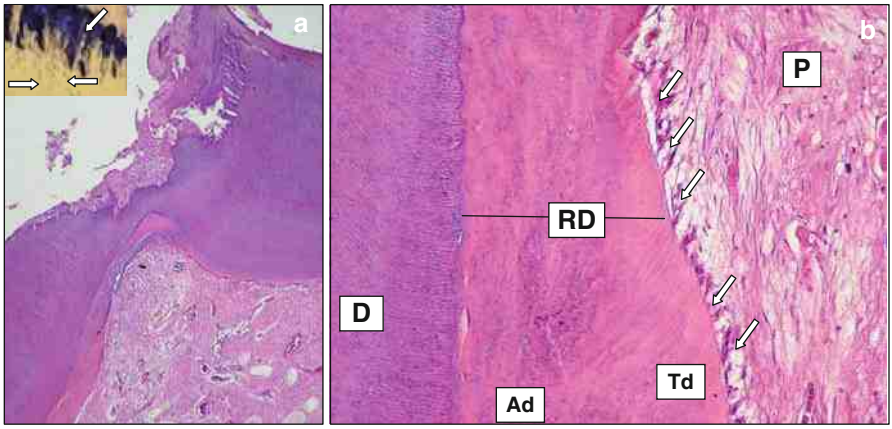

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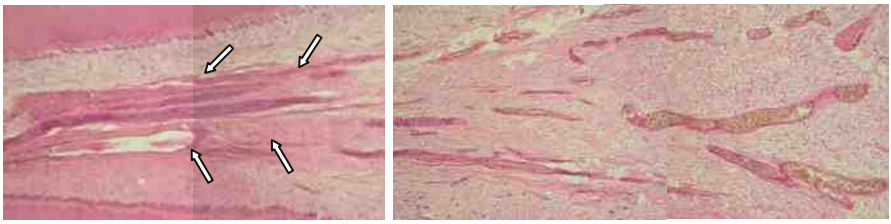

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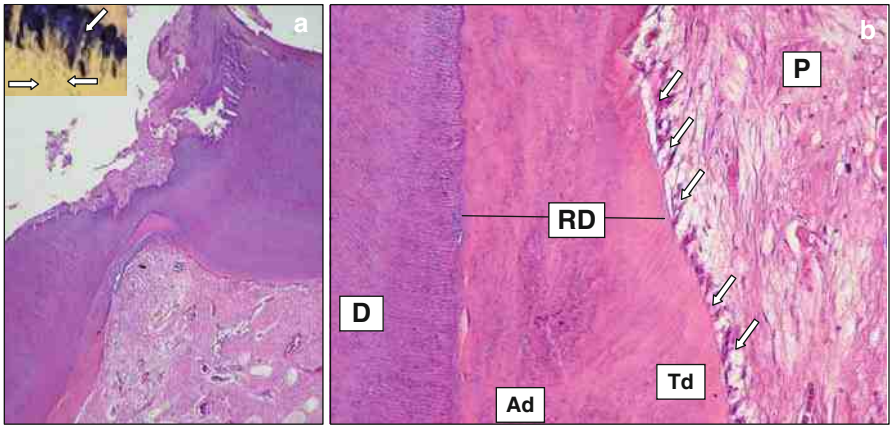

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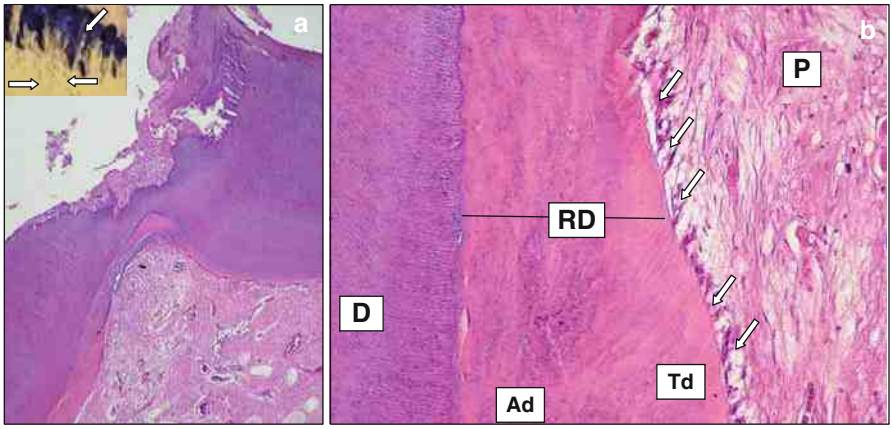

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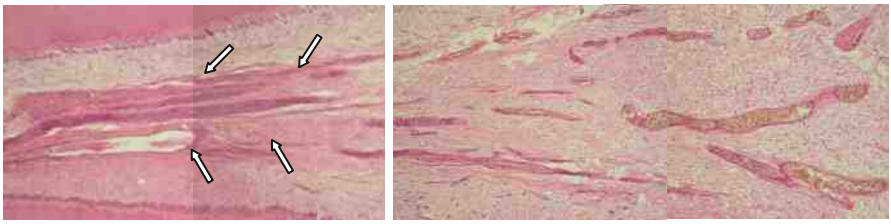

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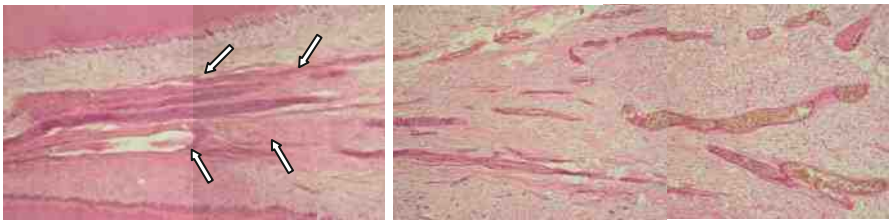

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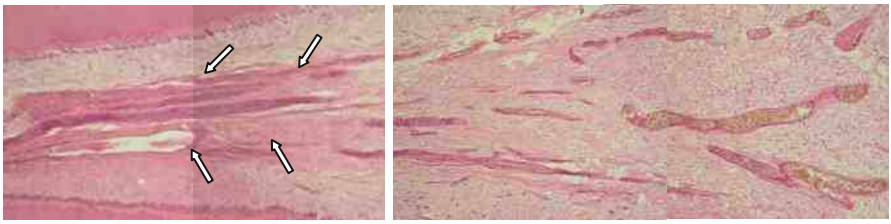

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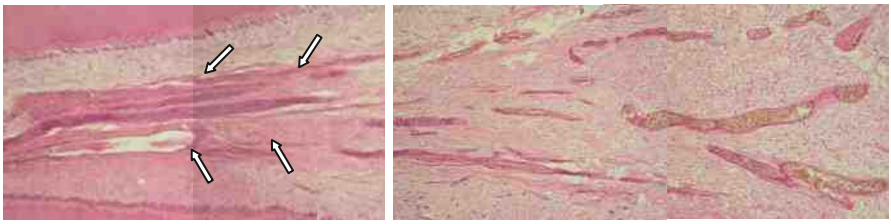

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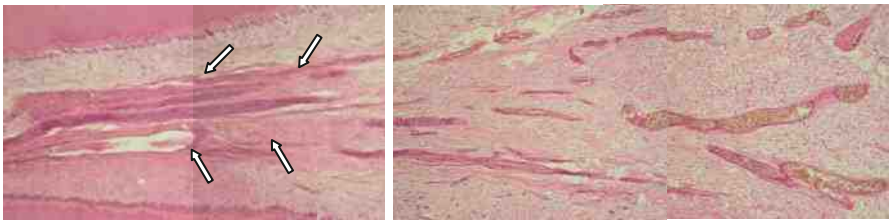

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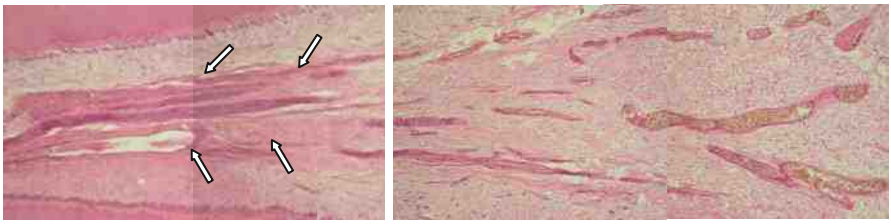

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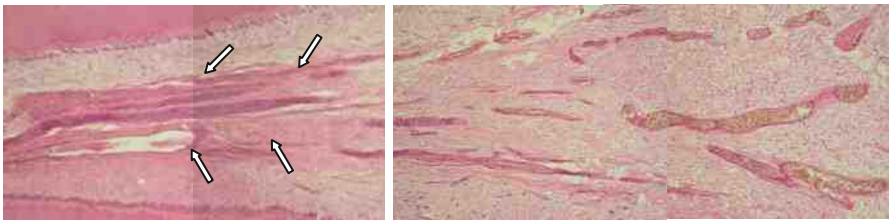

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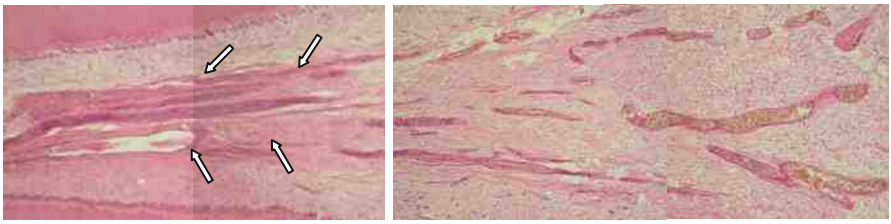

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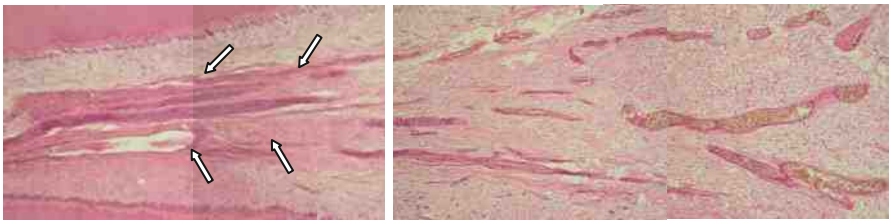

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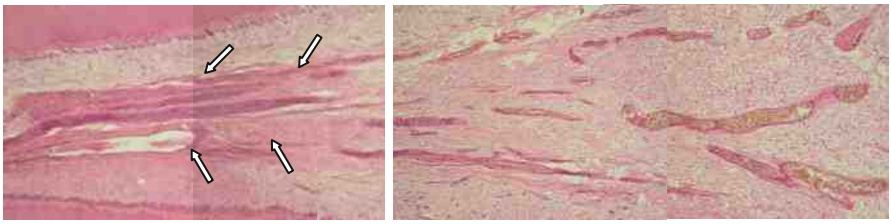

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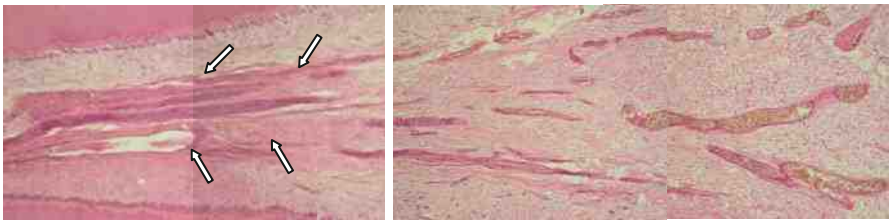

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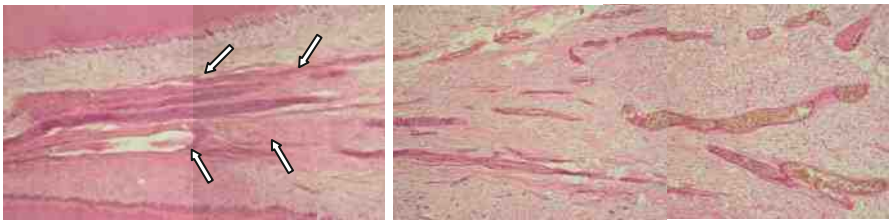

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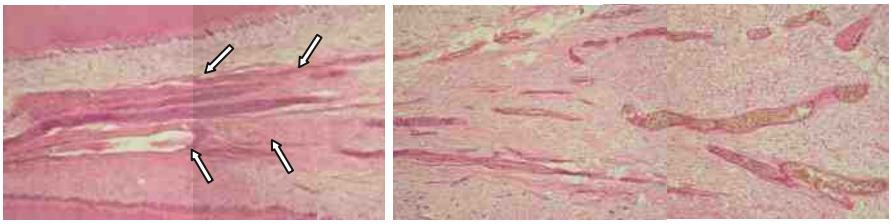

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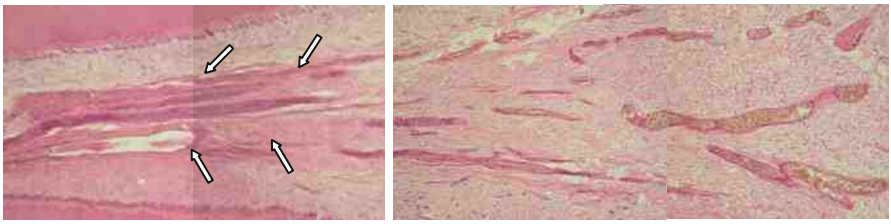

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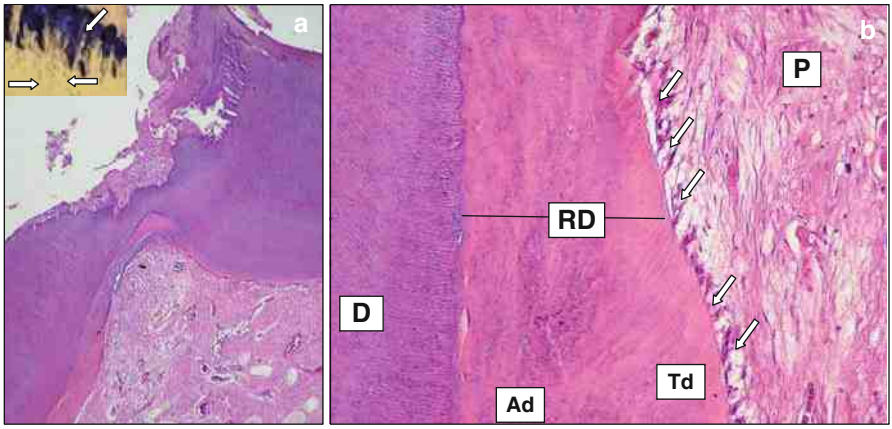

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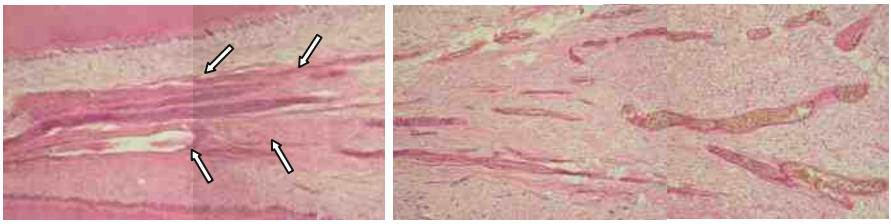

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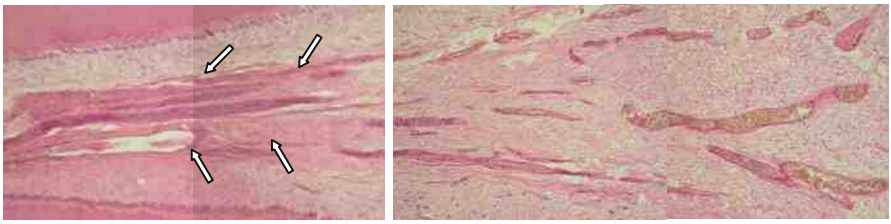

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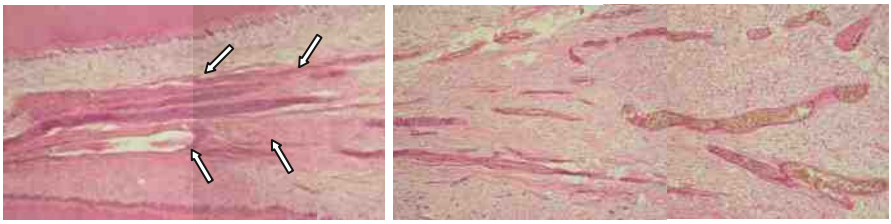

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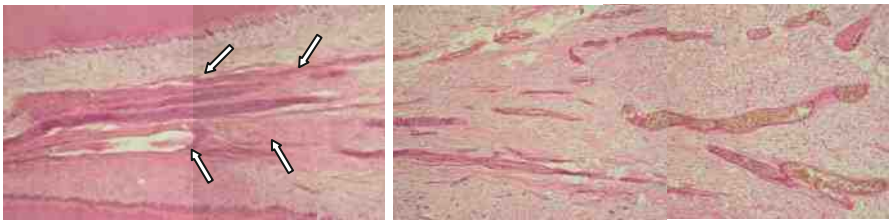

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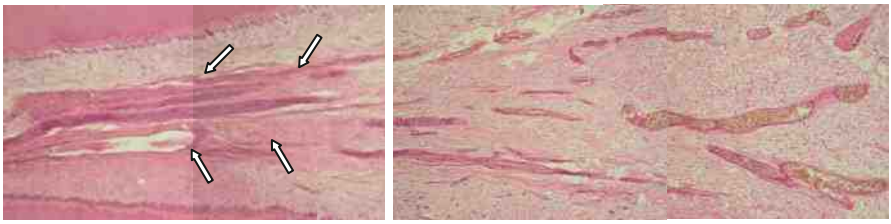

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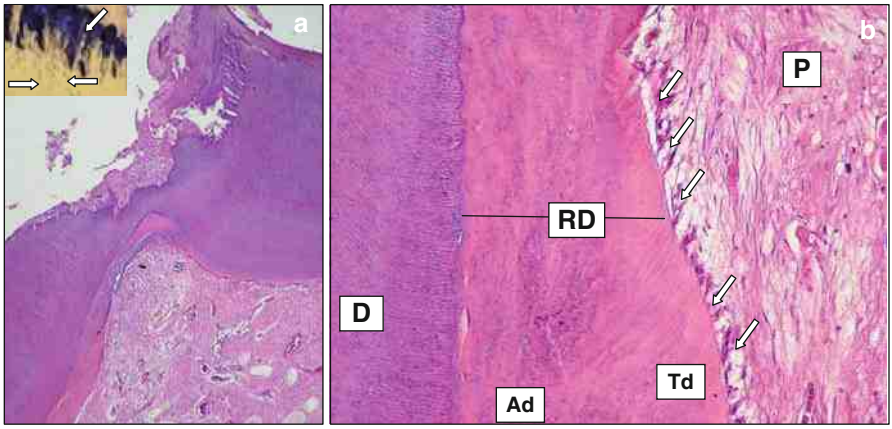

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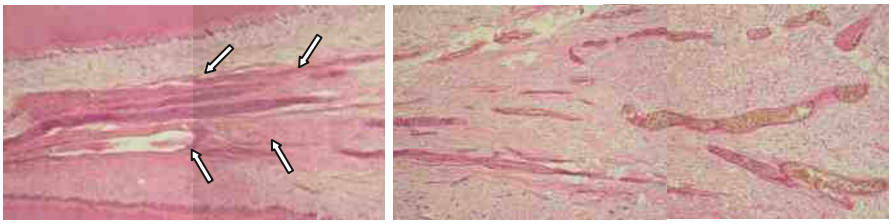

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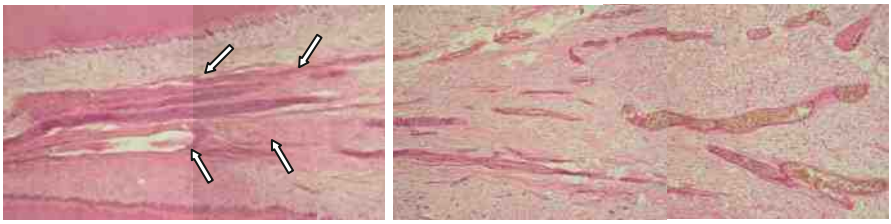

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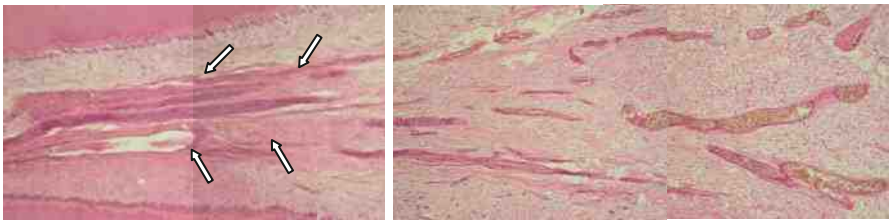

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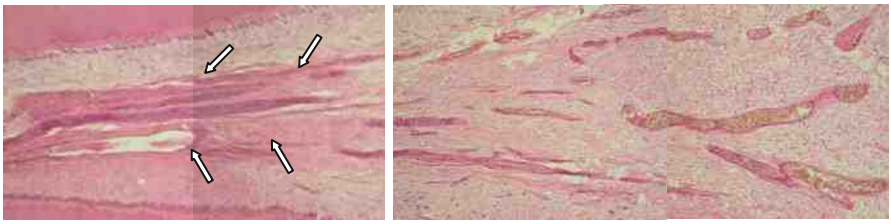

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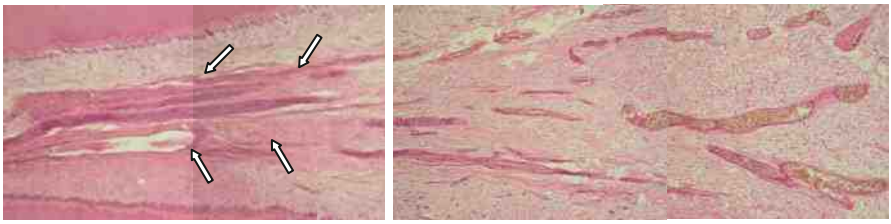

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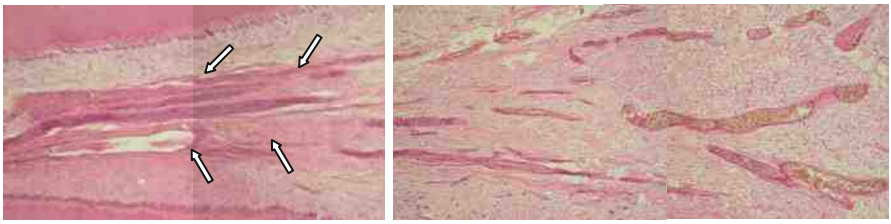

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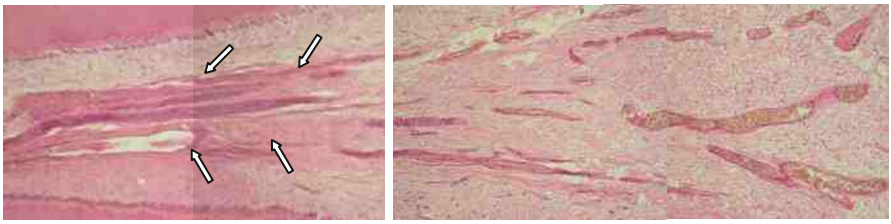

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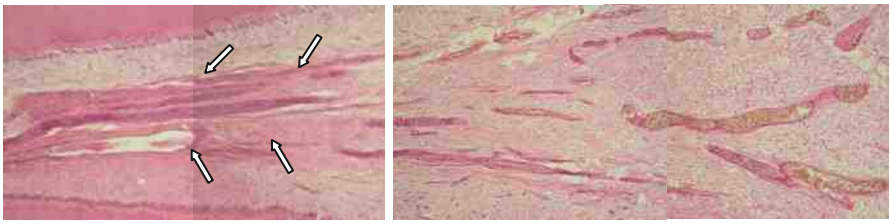

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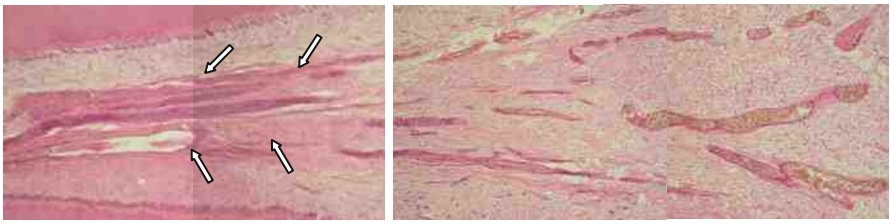

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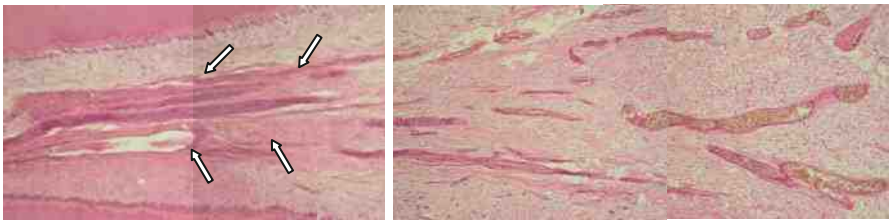

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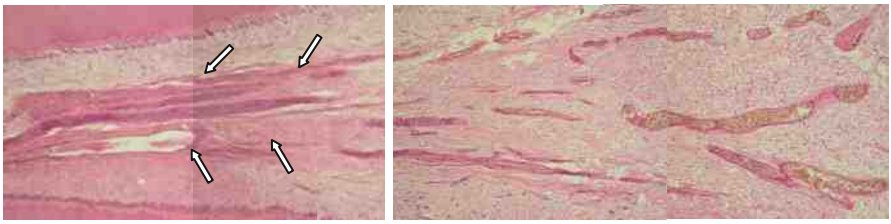

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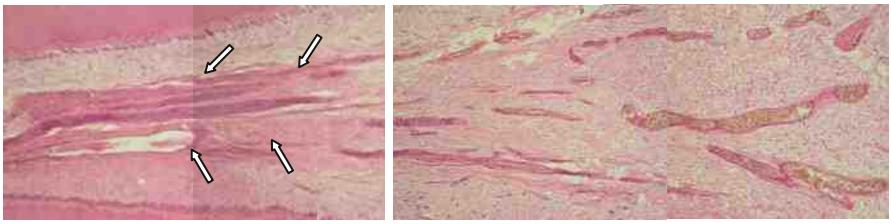

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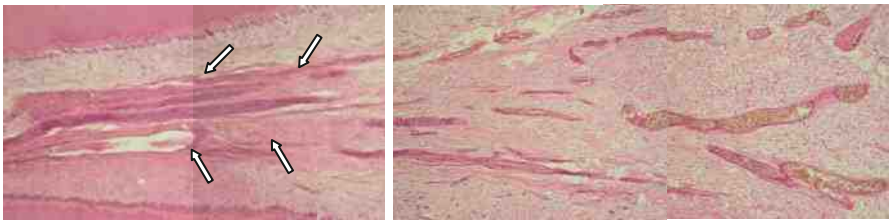

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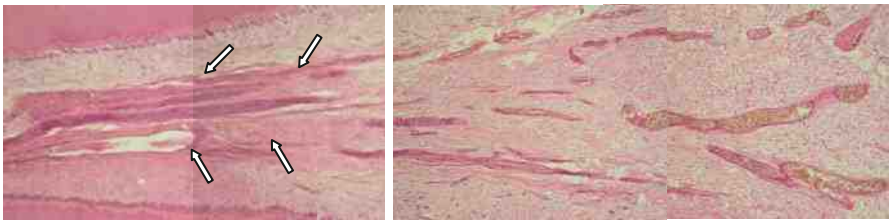

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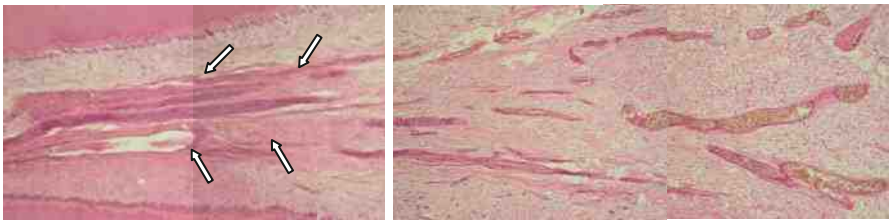

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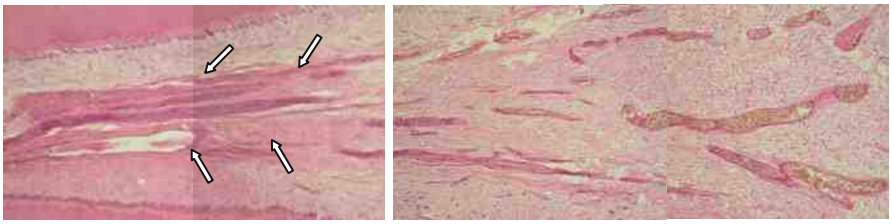

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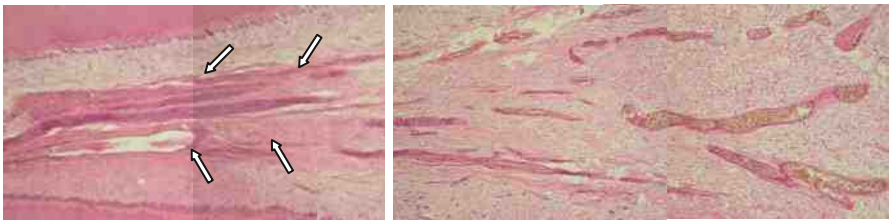

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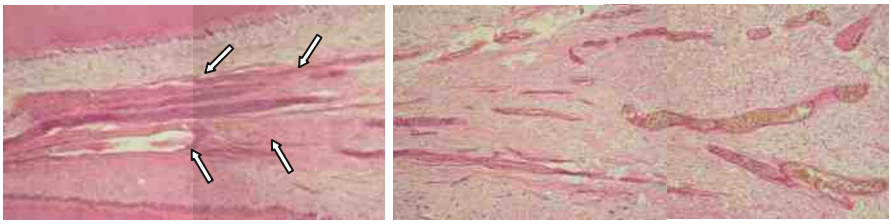

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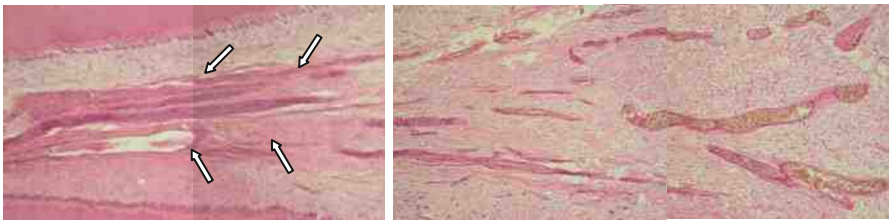

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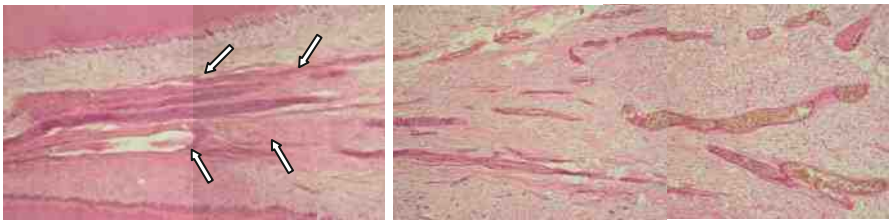

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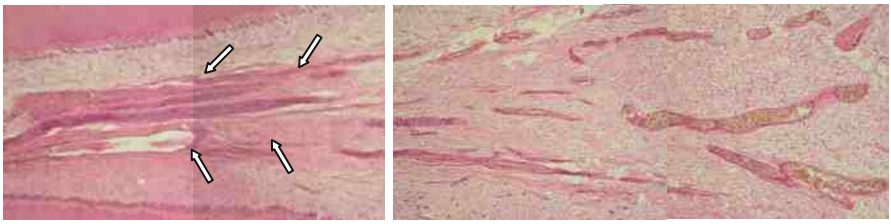

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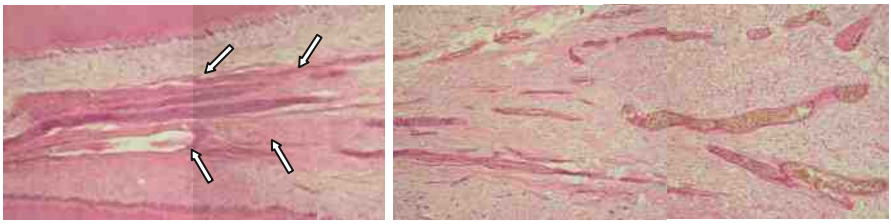

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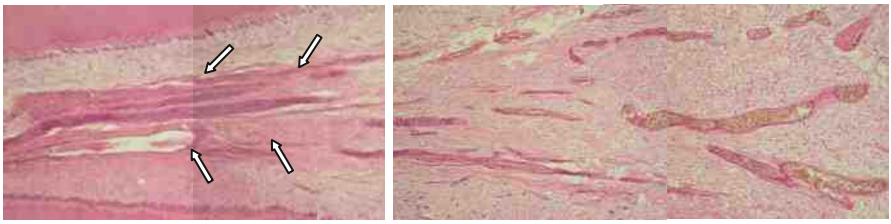

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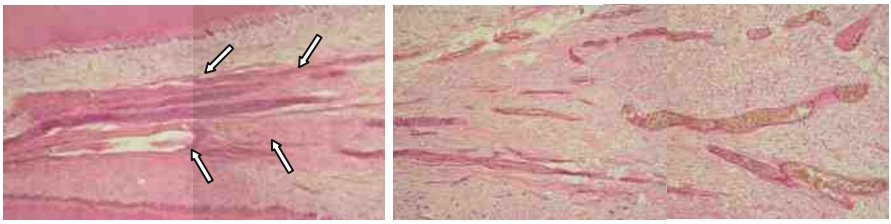

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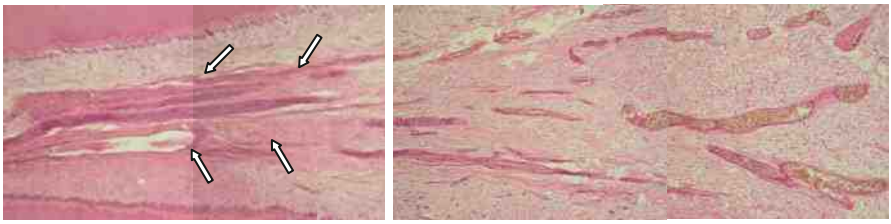

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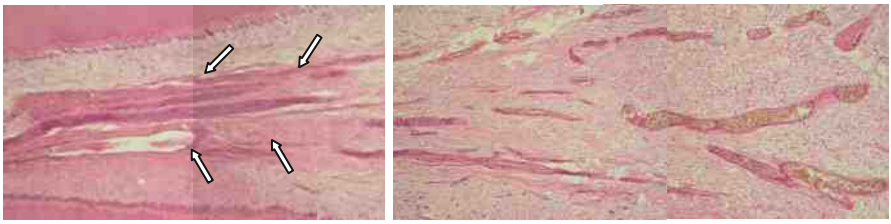

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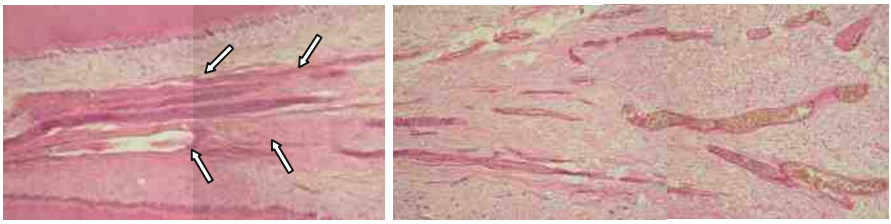

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