The Root Canal Anatomy in Permanent Dentition

Marco A. Versiani
Bettina Basrani
Manoel D. Sousa-Neto
Editors

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ISBN 978-3-319-73443-9 ISBN 978-3-319-73444-6 (eBook)
https://doi.org/10.1007/978-3-319-73444-6

Library of Congress Control Number: 2018942639

Printed on acid-free paper

This Springer imprint is published by the registered company Springer International Publishing AG part of Springer Nature
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Born in 1452 in the Tuscan village of Vinci, Leonardo was not only a gifted artist but also one of the greatest geniuses who has ever lived. According to him, "practice must always be founded on sound theory, and to this perspective is the guide and the gateway; and without this nothing can be done well." Obviously, this concept can be extended to innumerous human activities, including dental practice. In other words, essential practical and clinical experiences are very important for managing complex cases, but they cannot be a substitute for knowledge and theory. Dentistry was founded in empirical-based research and clinical experience, which means that clinicians often use empirical reasoning to make diagnoses and treatment plans, based on thoughts and follow-up of cases over the years. For several decades, the understanding of the influence of canal anatomy on endodontic procedures was based on empiric observation rather than on rigorous experimentation. Consequently, several authors from the past have stated the impossibility of succeeding in treating infected teeth because of the complexity of the root canal system, which was revealed at the end of the nineteenth century. With the improvement and application of the scientific method in health sciences, however, empirical approach started to be followed by systematic observation, measurement, and experiment. At this point, knowledge of the root and root canal anatomical complexities started to be applied into clinical practice, root canal therapy became more predictable, and endodontics, as a respectful specialty, has born. As the Greek physician Hippocrates postulated that the anatomy is the foundation of medicine, root canal anatomy is undoubtedly the foundation of endodontics!

This book is intended for students undergoing specialist training, general practitioner with special interest in endodontics, and specialists alike, being particularly dedicated to the memory of the pioneers in the endodontic field who overcame enormous obstacles to pave the way not only for their own careers or personal wills, but also for giving us inspiration to keep going with their outstanding work and write this book.

Foreword I

Science is the father of knowledge, but opinion breeds ignorance.

—Hippocrates

Root canal anatomy is the foundation of the art and science of endodontic therapy and succeeding post-treatment healing. Human dentition presents a wide range of anatomical variations in each tooth type. The root and canal morphology is learned to vary greatly between populations, within populations, and even within the same individual. The studies on root canal anatomy from the first half of the nineteenth century highlighted the number of root canals, their configurations, and complexities in teeth, while studies from the second half of the nineteenth century and early twentieth century gained insight on the apical terminus of the root canal anatomy and the periapical tissues that surround it. The knowledge obtained from this cluster of studies formed the bottom line for the biological basis in endodontic treatment. The conception of pulp and periodontal tissue as a continuum, association between endodontic disease and periapical host immune response, as well as the therapeutic significance of apical termination were all emphasized by these studies. Besides, leveraging on the current knowledge, it is recognized that effective nonsurgical root canal treatment and endodontic surgery requires a thorough knowledge of tooth morphology and root canal anatomy. Unfortunately, some of the therapeutic issues associated with complex root morphology and root canal anatomy still remain as challenges.

At the moment it is quite evident that the dental profession needs a broad review of this complex topic—anatomy of the root canal systems and their implications in root canal treatment. Drs. Versiani, Basrani, and Sousa-Neto recognized this requirement and have put together a comprehensive body of knowledge for endodontology. They have compiled some of the finest authorities from around the world to contribute knowledge and insights to this book. The broad list of chapters covered in this book has left no stone unturned. Drs. Versiani and Sousa-Neto as a team have studied root canal anatomy with micro-CT for the last couple of decades. Dr. Sousa-Neto has also used the method of diaphanization since the early 1980s to study root canal anatomy. This team has studied more than 15,000 teeth through these years and published many impactful articles. The experience of this group in this field is obvious from the chapters covered in this book. This book is the most comprehensive overview of root canal anatomy and their clinical implications the dental profession has had the opportunity to review.

There are several reasons for me to be delighted to write the foreword for this book. First, this book represents the maiden attempt to review the root canal anatomy and their related topics using modern high resolution imaging techniques. The dental profession must be updated about this important topic in a structured manner. I am also very pleased because I have known Dr. Versiani for almost 5 years and I am aware of his dedication to endodontology. Dr. Basrani, my colleague at the University of Toronto, was one of the very first internationally trained endodontists to move from Argentina to Canada with great passion for endodontics. This book is a product of their sacrifices, passion, and commitment. I am confident that this book will serve our profession well.

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Foreword II

The ability to understand and to anticipate root canal anatomy prior to rendering endodontic therapy has remained a challenging issue. Indeed, this issue confronts clinicians every day as they peer into access openings and search for canal orifices. When we read the endodontic literature, we read from time-to-time that a case failed because a second distal canal was overlooked, a third mesial canal was not noticed, a second canal in a lateral incisor was missed, etc. So, what can be done to significantly reduce these oversights?

Dr. Marco Versiani, Dr. Bettina Basrani, and Dr. Manoel Sousa-Neto took up this challenge and engaged tooth anatomy experts from around the world and combined their collective knowledge to prepare a textbook on internal tooth anatomy that should be required reading for every dentist who aims to provide the very finest endodontic therapy. As a reader would expect from a textbook of this caliber, each chapter is enriched with high-quality images and exhaustive citations from peer-reviewed literature.

This textbook is enhanced by also providing online videos and countless photographs derived from some of the most current technologies enabling researchers to even view the complexity of the root canal system in three dimensions. Based on new discoveries, this textbook by Drs. Versiani, Basrani, and Sousa-Neto provides a new nomenclature founded on expanded knowledge that sophisticated technologies have revealed.

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…superstition and pseudoscience keep getting in the way, […] providing easy answers, dodging sceptical scrutiny, casually pressing our awe buttons and cheapening the experience, making us routine and comfortable practitioners as well as victims of credulity. […] Pseudoscience is easier to contrive than science, because distracting confrontations with reality - where we cannot control the outcome of the comparison - are more readily avoided. […] Pseudoscience speaks to powerful emotional needs that science often leaves unfulfilled. […] Science thrives on errors, cutting them away one by one. False conclusions are drawn all the time, but they are drawn tentatively. Hypotheses are framed so they are capable of being disproved. […] Proprietary feelings are of course offended when a scientific hypothesis is disproved, but such disproofs are recognized as central to the scientific enterprise. […] Science is far from a perfect instrument of knowledge. It's just the best we have.

Carl Sagan
The Demon-Haunted World: Science as a Candle in the Dark

Acknowledgments

We would like to acknowledge Springer International Publishing for giving us the opportunity to edit this textbook on root canal anatomy. A special thanks goes to Andrei Berdichewsky, who was responsible for offering us this project.

We are indebted to the following collaborators for their hard work and invaluable contributions to make this book possible: Stephen Cohen, Anil Kishen, Antonis Chaniotis, Arnaldo Castellucci, Carlos Murgel, Clóvis Monteiro Bramante, Craig Barrington, Christos Boutsioukis, Diogo Guerreiro, Domenico Ricucci, Elizeu A. Pascon, Francisco Balandrano, Gustavo De-Deus, Hany M. A. Ahmed, Holm Reuver, Hugo Sousa, Isabela N. Rôças, James L. Gutmann, Jesus D. Pécora, Jojo Kottoor, Jorge N. R. Martins, José F. Siqueira Jr., Leo Tjäderhane, Mário R. Pereira, Nicola Perrini, Nuno Pinto, Oscar von Stetten, Paul M. H. Dummer, Ronald Ordinola-Zapata, Sergiu Nicola, and other colleagues who allow us the privilege of sharing their images in courtesy. Thank you very much!

To our families, thanks for your encouragement, assistance, and support that allow us to progress in our professional lives.

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1 Historical Overview of the Studies on Root Canal Anatomy

Nicola Perrini and Marco A. Versiani

Abstract

The fundamental basis of the endodontic specialty is the knowledge of root canal anatomy. Thus, a thorough understanding of the canal morphology and its variations in all groups of teeth is a basic requirement to improve the outcome of the endodontic therapy. In the past, a lot of research work was done on this subject, and the findings have had a noteworthy influence on clinical practice as well as on dental education. Therefore, it would be appropriate to take a brief look to the past to understand contemporary research approaches on the study of root canal anatomy. Authors that preceded this new image-processing technological era, to whom endodontics are greatly indebted, must be revisited.

1.1 Introduction

From the Latin *anatomia*, dissection, and from the Greek *anatome*, where *ana* means "up" and *temnein* means "to cut," this word represents the study of the body, the determination of the regions in an organism that are to be considered its parts. Writing about the historical aspects of the studies on root canal anatomy does not simply mean to place the stages that lead to the current knowledge chronologically or compiling the biography of the authors. In fact, the history of endodontic as a dental specialty is relatively recent and was born with the better understanding of the morphology and biology of teeth, as well as the development of endodontic techniques in the brief period of 30 years, from 1900 to 1930, after centuries of immobility [1]. Consequently, this historical overview was carried out considering the result of a long cultural and scientific evolution that simultaneously influenced medicine and its other biological branches, including dentistry.

1.2 From Classical Antiquity to Thirteenth Century

The ancient Greek anatomist *Herophilus* (c.335–c.280 B.C.), founder of the school of anatomy of Alexandria, and his disciple *Erasistratus* (c.310–c.250 B.C.) were the first physicians to perform systematic dissection of human bodies. *Herophilus* has been widely acknowledged as the "father of anatomy" and hailed as one of the greatest anatomists that ever lived. His revolutionary discoveries represented an important step in the ancient understanding of the human anatomy [2]. Some

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M. A. Versiani et al. (eds.), *The Root Canal Anatomy in Permanent Dentition*,
https://doi.org/10.1007/978-3-319-73444-6_1

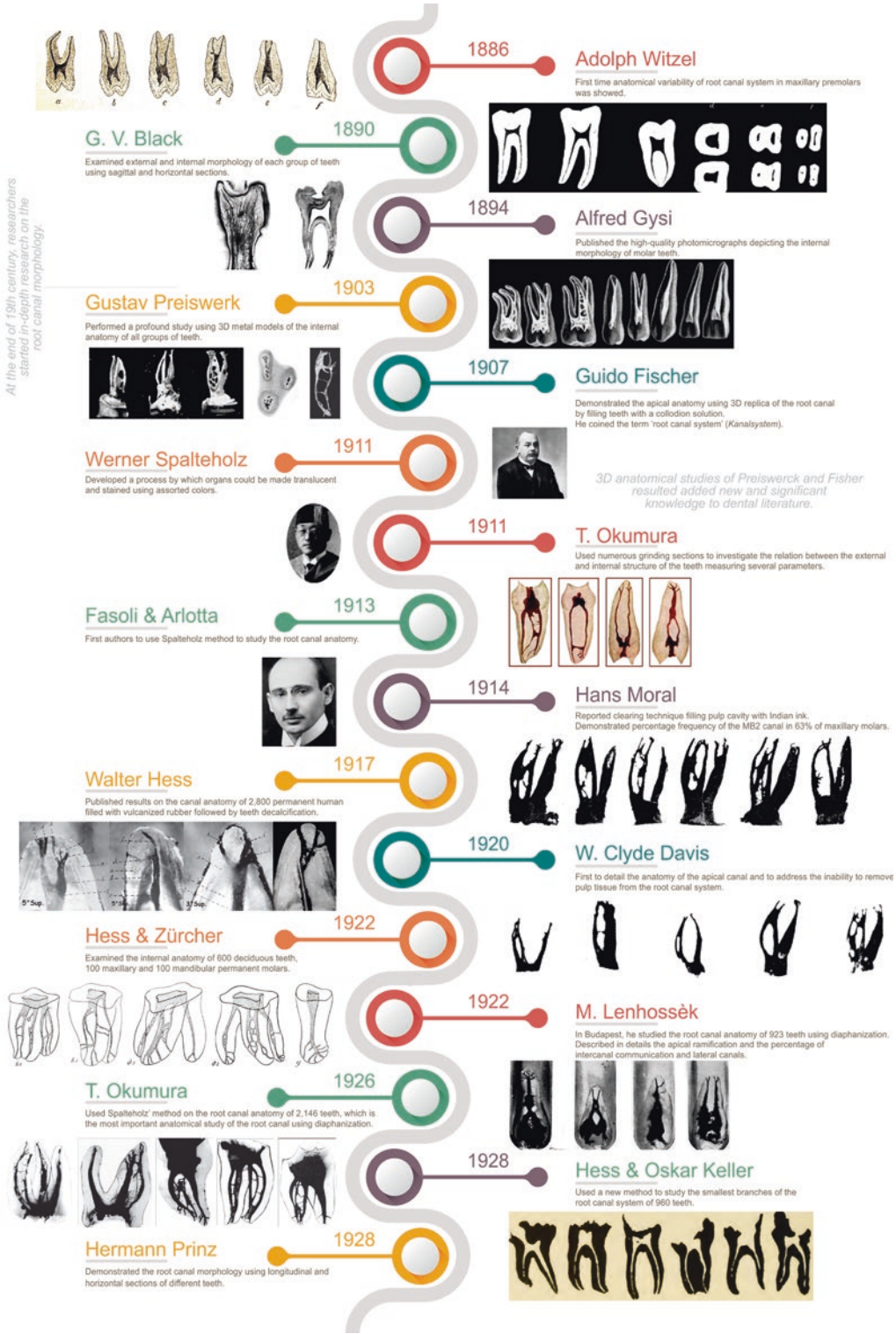

Fig. 1.2 Timeline infographic of the studies on root canal anatomy (from 1886 to 1928) (images reproduced from Perrini [1] with permission)

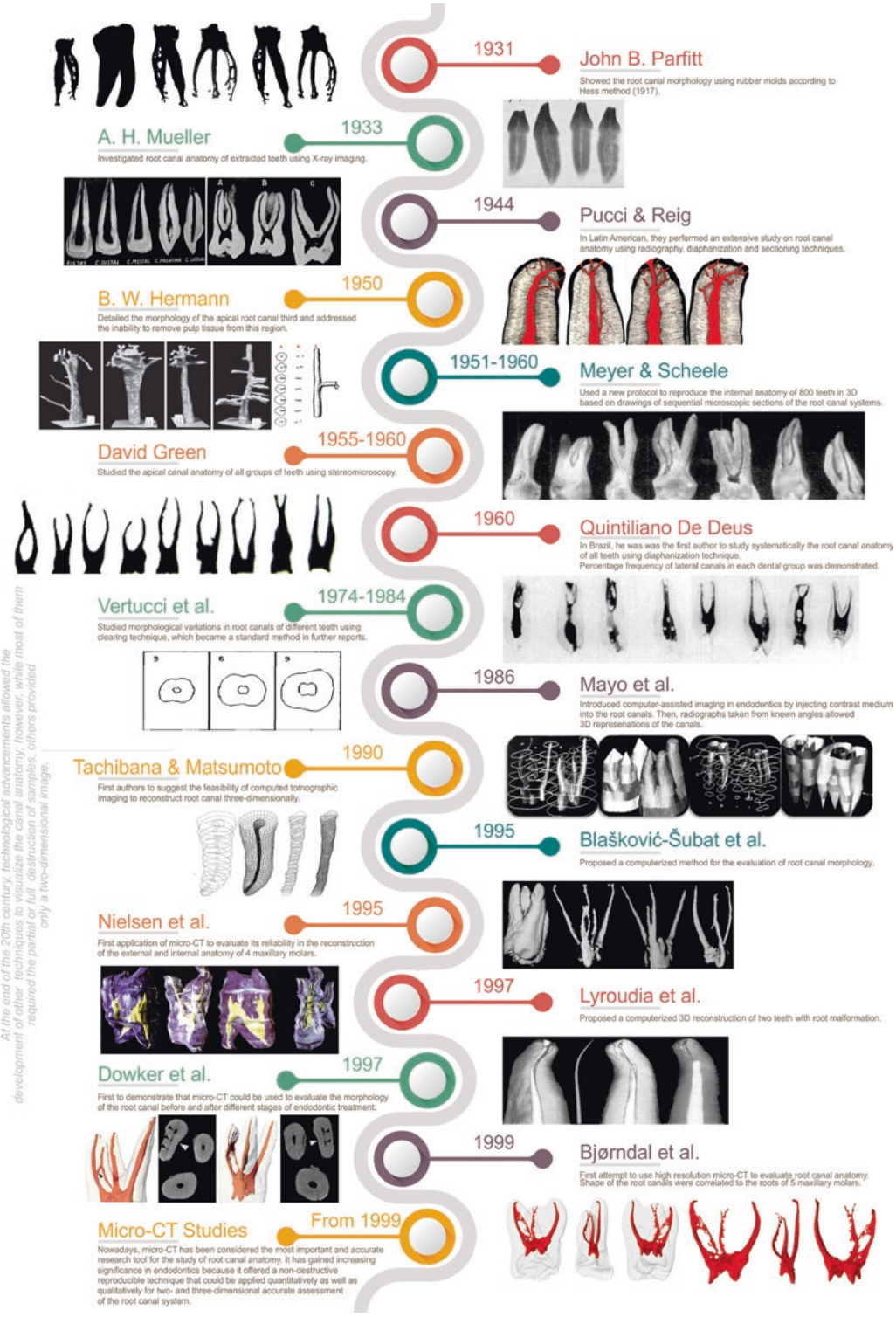

Fig. 1.3 Timeline infographic of the studies on root canal anatomy (from 1931 to nowadays) (images reproduced from Mueller [57], Pucci and Reig [58], Green [60], De Deus [68], Vertucci [70], Mayo et al. [89], Tachibana and Matsumoto [100], Blašković-Šubat et al. [93], Nielsen et al. [107], Lyroudia et al. [94], Dowker et al. [106], Bjørndal et al. [111] and Perrini [1], with permission)

2 Dentin Basic Structure, Composition, and Function

Leo Tjäderhane

Abstract

Dentin is the largest structural component of the human tooth. Dentin provides support to enamel, preventing enamel fractures during occlusal loading. It also protects the pulp from microbial and other potentially harmful stimuli. As vital tissue, dentin is not only a passive mechanical barrier between the oral environment and the pulp tissue but, in many ways, participates in the overall protection of the continuum of the hard and soft tissue often referred as the dentin-pulp complex. For example, dentin contains several growth factors that may be liberated during wear or caries and participate in the regulation of the defensive reaction at the dentin-pulp border or the pulp proper. Odontoblasts project their cell processes into dentinal tubules, and also therefore the division of the "vital" pulp and "dead" mineralized dentin is artificial. Different parts of the dentin in a particular tooth may also qualitatively differ from each other, which enables it to meet the requirements of that specific location.

2.1 Introduction

Dentin can be described in various ways, based on the composition, structure, or type of dentin. Usually, dentin is described as extracellular organic matrix that has been mineralized, much like the bone. As a matter of fact, dentin is nanocrystalline-reinforced biocomposite, which gives it its unique properties. About 70% (55% in volume) and 20% (30% in volume) in weight are minerals and organic components, respectively, the rest being water. However, due to the tubular nature of dentin and the occlusion of tubules by peritubular (intratubular) dentin with age and as defensive reaction, these values are only average.

The structure of dentin can also be divided into intertubular and peritubular dentin. Vast majority of the organic component is located in the intertubular dentin formed by the odontoblasts at the dentin-pulp border. Due to the tubular occlusion, the amount of minerals and division between inter- and peritubular dentin can vary significantly between the different parts of the tooth, as the peritubular dentin slowly occupies the tubular lumen. At the same time, water content in these areas is, respectively, reduced. And finally, dentin

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M. A. Versiani et al. (eds.), *The Root Canal Anatomy in Permanent Dentition*,
https://doi.org/10.1007/978-3-319-73444-6_2

is frequently classified by its phase of formation: dentin-enamel junction, mantle dentin, primary dentin, secondary dentin, and tertiary dentin. Tertiary dentin formation is part of the dentin-pulp complex defensive reaction aiming to protect the pulp and can further be divided into reactionary and reparative dentin, depending on the structure and cells forming the dentin (primary or replacement odontoblasts, respectively) [1].

2.2 Dentin Formation

2.2.1 Odontoblasts

Dentin is almost exclusively formed by the odontoblasts that are derived from embryonic connective ectomesenchymal cells from the cranial neural crest [2]. The differentiating odontoblasts start the secretion of the predentinal proteins, followed by the initiation of enamel matrix secretion by the differentiating ameloblasts, at the site where the dentin-enamel junction (DEJ) is formed. During and right after the differentiation, the odontoblasts organize into a distinguished odontoblast cell layer, and the mineralization of organic matrix completes the formation of the first layer of dentin, mantle dentin [2].

In the coronal part of the tooth, odontoblasts are tall cells, and their morphological and cell membrane polarization [3] is unique in the group of collagen-synthesizing cells. Odontoblasts form a single layer of cells between dentin and pulp, with the cell body located on a pulpal wall of dentin and odontoblast processes inserted into dentinal tubules (Figs. 2.1 and 2.2a). The cell bodies are 20–40 μm tall, depending on dentinogenic activity. The odontoblast process is a cytoplasmic process which penetrates into mineralized dentin tubules. The process has the 0.5–1 μm main trunk and thinner lateral branches [4].

One of the longest controversies in dentin-pulp complex research has been the extent of odontoblast processes into dentinal tubules. This is caused by the conflicting results obtained with different research methods and by the possible differences between the species [4]. In human teeth, most studies indicate that the odontoblast cell processes would not extend far from the dentin-pulp border (200–700 μm) (Fig. 2.2b).

2.2.2 Predentin and Mineralization Front

The 10–30 μm layer of unmineralized predentin is located between odontoblasts and mineralized dentin (Figs. 2.1 and 2.2a). This is where the dentin organic matrix is organized before the controlled mineralization at the mineralization front

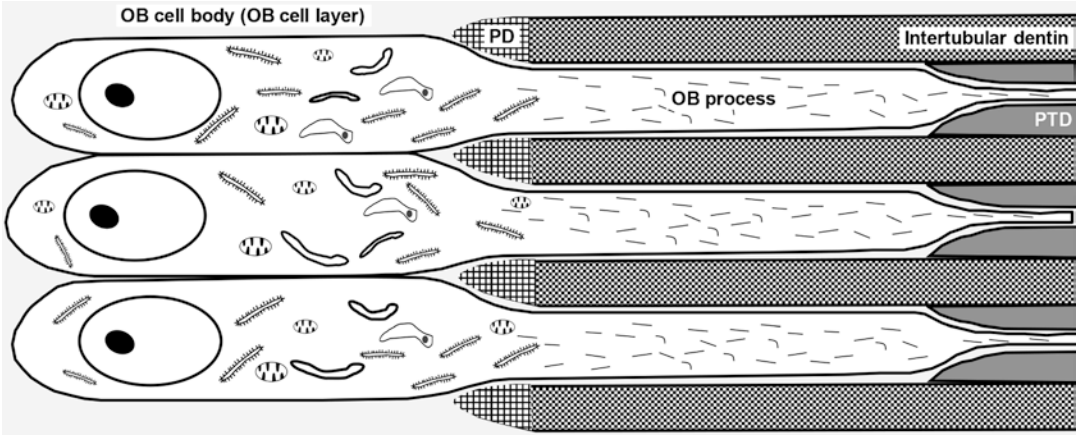

Fig. 2.1 Drawing of the dentin-pulp border. Odontoblast (OB) cell bodies with large nucleus at the pulpal terminus of the cell and cytoplasmic organelles form a tight cell layer separated from the mineralized dentin by predentin (PD) where the intertubular dentin organic matrix is organized before mineralization. Odontoblast processes penetrate into mineralized intertubular dentin in dentinal tubules. The cell processes are devoid of cytoplasmic organelles. Peritubular dentin (PTD) formation starts some distance from the mineralization front (modified from Tjäderhane and Haapasalo [4], with permission)

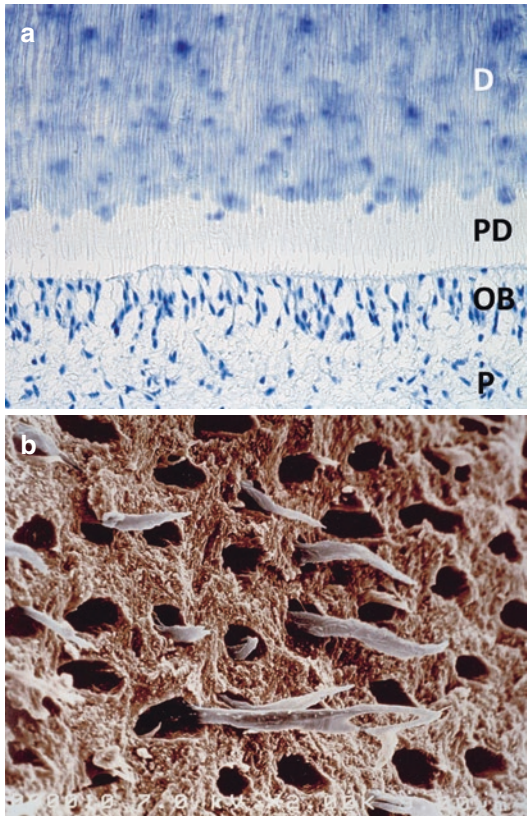

Fig. 2.2 (**a**) Dentin-pulp complex interface, displaying odontoblasts (OB), predentin (PD), tubular dentin (D), and pulp proper (P) beneath the odontoblasts. Note that instead of being straight, the mineralization front is irregular, presenting the mineralization in the form of calcospherites. Demineralized section, toluidine blue. Magnification 20×. (**b**) Odontoblast processes protruding from the dentin fracture site exposed at 0.4 mm from the pulp. Magnification 2000× (from Goracci et al. [47], with permission)

to form intertubular dentin. The backbone of the organic matrix is type I collagen, whereas non-collagenous proteins—glycoproteins, proteoglycans, and enzymes—control the matrix maturation and mineralization. The mineralization front is often considered to be linear, but actually mineralized globular protrusions called calcospherites are common (Fig. 2.2a) [4].

2.3 Dentin-Enamel Junction

The dentin-enamel junction (DEJ) is not just an inactive interface between enamel and dentin but seems to be much more complex and interactive structure than previously believed [1]. Phylogenetic, developmental, structural, and biological characteristics have led to the suggestions that instead of the DEJ, this structure should be termed the dentin-enamel junctional complex [5].

The DEJ of human tooth is wavy, scalloped line between two mineralized structures. Laser-induced autofluorescence and emission spectroscopy demonstrate the DEJ as 7–15-μm-wide structure, which is distinct from both enamel and dentin [6]. The "primary" scallop size varies from 25 to 50 μm. They contain smaller (0.25–2 μm) "secondary scallops" and intermingling sub-micrometer-sized ridges of dentin and enamel [7, 8]. The scalloped form of the interface is thought to improve the mechanical attachment of enamel to dentin [1]. However, human is among the very few species in which the scalloping DEJ has been demonstrated, questioning the role of this wavy structure to the enamel-dentin attachment. Instead of the scalloped form of the DEJ—or in addition to it—hydroxyapatite crystals extending through the DEJ into both structure [9–11] and dentinal collagen fibrils reaching into enamel [12] may contribute to the durability and toughness of the DEJ under occlusal forces [1].

2.4 Mantle Dentin

The mantle dentin is 5–30-μm-thick layer of the outermost dentin that in many aspects is different from the rest of the dentin. This is due to the different process of formation. The mantle dentin organic matrix is laid down during and immediately after the terminal differentiation of the odontoblasts and before their spatial organization into distinct cell layer. It also contains the remnant components of dental papilla, and the mechanisms of mineralization are different from what occurs at the mineralization front [4]. It is devoid of large tubules; instead, multiple small ramifications of each tubule are present in the mantle dentin. The organic matrix in mantle dentin is less regular and contains so-called von Korff fibers consisting mainly of type III collagen [13]. The mineral content of mantle dentin has also been thought to be lower than in circumpulpal dentin,

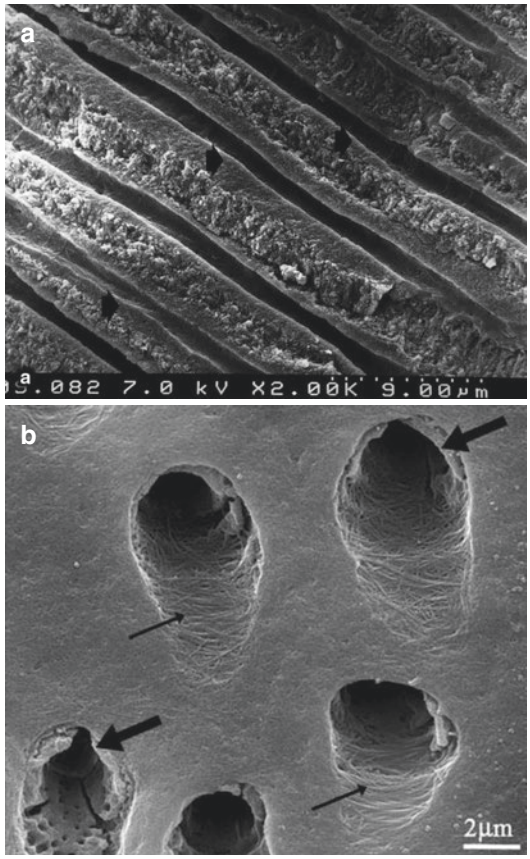

Fig. 2.3 (**a**) Scanning electron micrograph of fractured dentin about 1 mm from the dentin-pulp border. Peritubular dentin (arrows) is present already this close to the pulp. Magnification 2000× (from Goracci et al. [47], with permission). (**b**) SEM image of dentin surface. In the majority of tubules, peritubular dentin has fractured and was lost during the polishing. Two tubules retain the peritubular dentin (thick arrows) which appears non-fibrillar and porous/perforated. The internal surfaces of tubule walls exposed by the fragmentation of the peritubular dentin contain intertubular collagen fibrils (thin arrows) (from Gotliv et al. [26], with permission)

wear or caries, the tubules may also be occluded by mineral crystals formed due to reprecipitation of minerals or from the mineral ions delivered from the pulpal side via dentinal fluid. This phenomenon is—confusingly—also called dentin sclerosis, although "reactive (dentin) sclerosis" might be the more appropriate term [1].

Peritubular dentin is a separate phase from intertubular dentin, forming a distinct annulus within each tubule instead of intertubular dentin matrix-mediated crystallization (Fig. 2.3b). However, peritubular dentin is often heterogeneous, and several separate or connected mechanisms may occur at the same time [1, 4]. Peritubular dentin is perforated by tubular branches but also by several small fenestrations [26] (Fig. 2.3b), which allow tubular fluid and its components pass back and forth across the peritubular dentin. Peritubular dentin may thus not act only as a passive blockage of dentinal tubules but also contribute to the vitality and possibly even remodeling of mineralized dentin as a whole.

2.6 Tertiary Dentin

Tertiary dentin formation is a response to external irritation, such as wear, erosion, trauma, caries, or cavity preparation. The growth factors present in mineralized dentin and liberated during caries or wear are believed to initiate and control the tertiary dentin formation and structure [27]. Tertiary dentin increases the mineralized barrier thickness between oral microbes and other irritants and pulp tissue, aiming to retain the pulp tissue vital and noninfected. The form and regularity of tertiary dentin depend on the intensity and duration of the stimulus. There are two kinds of tertiary dentin, namely, reactionary dentin, formed by original odontoblasts, and reparative dentin, formed by newly differentiated replacement odontoblasts (Fig. 2.4) [2, 4]. Reactionary dentin is tubular and relatively similar to secondary dentin in structure, while reparative dentin is usually atubular (or poorly tubularized) and may present variable forms (Fig. 2.5). Reparative dentin is believed to be relatively impermeable, forming a barrier between tubular dentin and pulp tissue.

2.7 Root Dentin

Root dentin bears strong resemblance but also certain distinct differences to coronal dentin. The outermost layer of root dentin, the granular layer of Tomes, is located right beneath the root cemen-

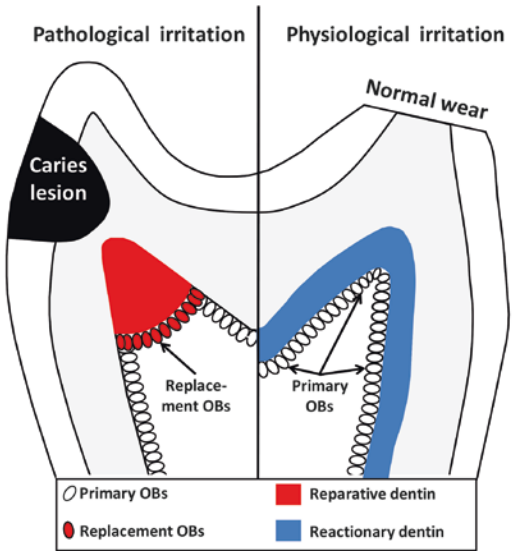

Fig. 2.4 A model of the fate of odontoblasts under pathological and physiological irritation. Intensive pathological irritation (deep and/or active caries, extensive wear, trauma), bacteria, and/or dentinal growth factors induce local odontoblast death. At the same time, the pulp stem cells migrate and differentiate into replacement odontoblasts and start the local synthesis of reparative dentin, usually distinctly different from primary dentin (lack of dentinal tubules, lamellar osteodentin-type calcification, etc.). In teeth with physiological wear or other mild irritation, slow but continuous dentin formation (either physiological or reactionary) by primary odontoblasts leads to decrease in pulpal space (modified from Mitsiadis et al. [2], and Tjäderhane and Haapasalo [4], with permissions)

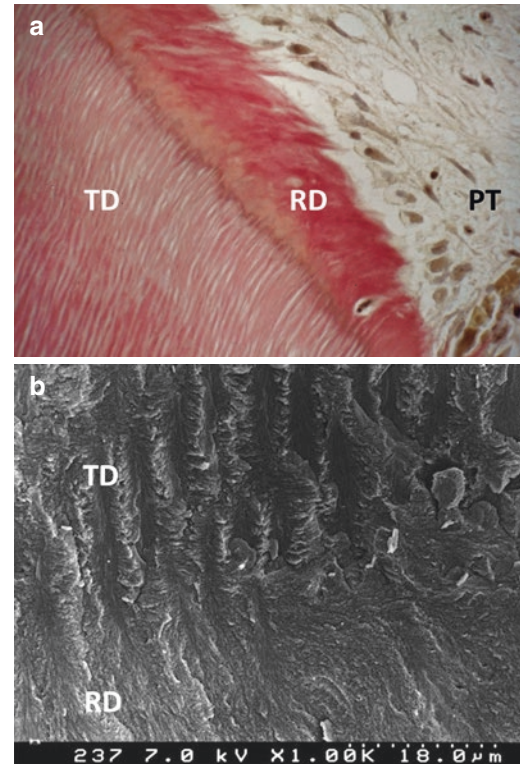

Fig. 2.5 (**a**) Histological view at the dentin-pulp border with physiological tubular dentin (TD) and reparative dentin (RD) lacking tubular continuity. Also note the poorly organized odontoblast-like cell layer. PT: pulp tissue. Light microscopy, magnification 250×. (**b**) SEM image of the borderline between normal tubular dentin (TD) and atubular reparative dentin (RD). Magnification 1000× (both images from Goracci et al. [47] with permission)

tum. It is thought to represent the mantle dentin with thin canaliculi and poorly fused globules which perhaps represent the mineralization pattern in the early stages of root dentin/cementum formation [1]. The tubular density in root dentin is at least moderately [24, 28] or even drastically [29] lower than in coronal dentin, especially in the most apical part [24, 28, 30, 31]. The apical portion of human dentin has also other structural variations, such as relatively large number of accessory root canals, transient and repaired surface resorption, and cementum-like lining the apical root canal wall [1]. Interestingly, age-related root tubular sclerosis starts from the apical region and advances coronally [25, 32], and it may be the main factor influencing permeability of root dentin [33, 34] (Fig. 2.6a). Root dentin has also other regional differences in permeability, as buccal/lingual root canal dentin has patent tubules, while the mesial/distal dentin-pulp borders may be completely occluded with minerals [33, 34] (Fig. 2.6b). This kind of patterns of tubule patency may correspond to local stress distributions of the roots under occlusal loading [1].

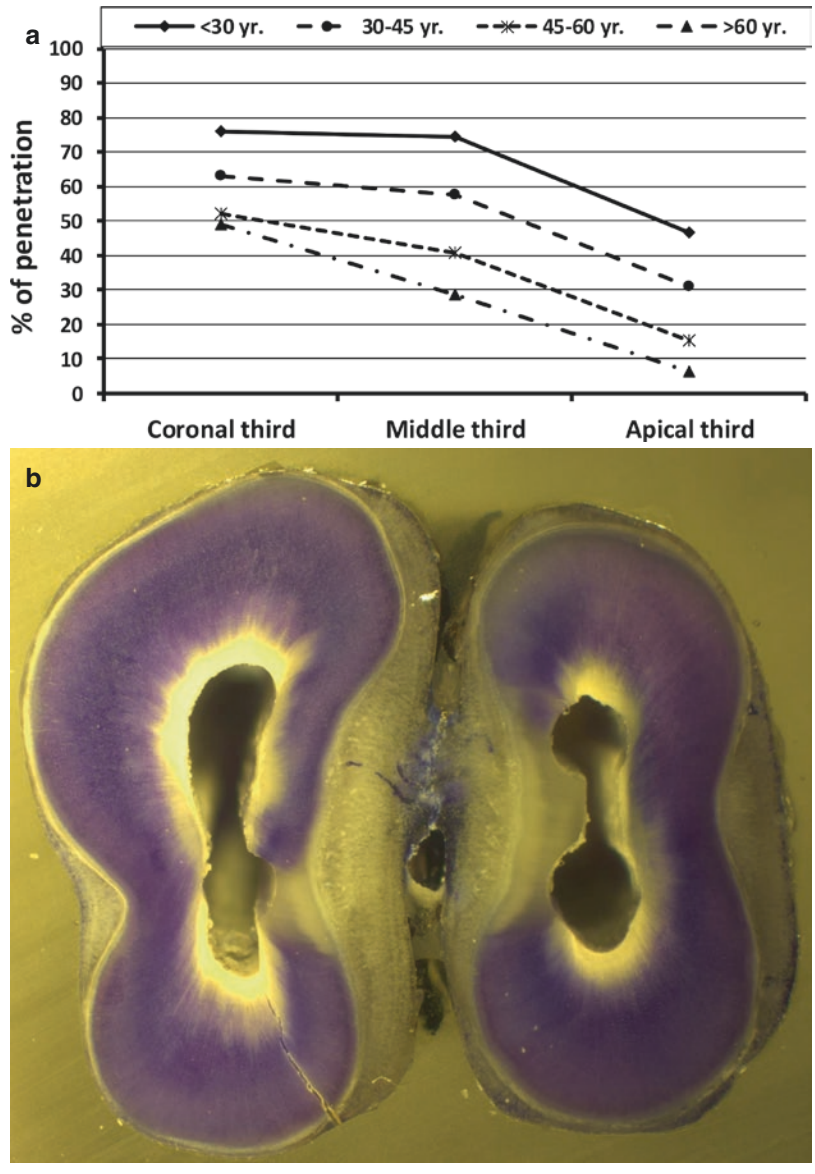

Fig. 2.6 (**a**) Relative mean dye penetration (in percentage of complete dentin area) after 2-month methylene blue incubation in instrumented root canals (data from Thaler et al. [34]). (**b**) The patency of tubules demonstrated in lower molar roots with the removal of methylene blue stain with 5.25% NaOCl irrigation. Buccal and lingual curvatures of canals demonstrate clear penetration, while in approximal and especially furcal sides, the effect is less pronounced. At furcal sites, the lack of tubular patency is also seen with the lack of methylene blue staining. Reflective light microscope, 10× magnification

2.8 Physiological and Pathological Changes in Dentin

2.8.1 Age-Related Changes

The best known—and the most important in terms of clinical endodontology—age-induced changes in human dentin-pulp complex are the obliteration of the pulp chamber and root canals even in intact teeth, due to physiological slow-rate secondary dentin formation. In incisors, canines, and premolars, the physiological age-related obliteration usually advances from the coronal direction, while in molars the dentin in the pulp chamber floor may also grow toward the roof, contributing to the pulp chamber occlusion. The clinical relevance of these phenomena is

discussed in more details in other chapters of this book.

The effects of aging on dentin mechanical properties are less known and have been a subject of debate for decades. However, the more recent studies strongly indicate that mineralized dentin may not be as stable as previously indicated, and the aging induces changes that should be taken into consideration in clinical work. Perhaps the most important aspect is the increased mineralization—or more precisely, increased mineral-to-collagen ratio—in aged dentin that increases the hardness especially in outer dentin [35]. This is mostly due to the peritubular dentin occlusion of dentinal tubules [35, 36]. At the same time, the mechanical properties of dentin change: the fatigue crack growth exponent is about 40% lower [37], the endurance strength about 48% lower [38], and the fatigue crack propagation over 100 times faster [37] in old than in young dentin. As a result, dentin flexure strength has been calculated to reduce approximately 20 MPa/decade [36, 38], and this reduction correlates well with the occlusion of tubules with age (Fig. 2.7). Reduction of the lumen diameter and increase in mineral content may not be the only factors contributing to the changes in mechanical behavior of human dentin with age, as changes in the organic components have also been speculated to contribute to the structural response [38]. While the potential age-related changes in, e.g., dentin collagen cross-link remain to be shown, loss of matrix-degrading enzymes has already been demonstrated [39–41] and may implicate also changes in their substrates, including collagen and non-collagenous proteins.

2.8.2 Caries-Affected Dentin

The concept of *minimally invasive dentistry* limits the cavity preparation to the removal of caries-infected dentin, leaving the restoration to be adhesive-bonded to caries-affected dentin. The immediate bond strengths to caries-affected dentin are commonly 20–50% lower than to sound dentin and even lower with caries-infected dentin [42]. Caries-affected dentin has lower mineral content, increased porosity, and altered structure and distribution of dentin collagen and non-collagenous proteins [43]. These changes increase dentin wetness and significantly reduce dentin mechanical properties, such as hardness, stiffness, tensile strength, modulus of elasticity, and shrinkage during drying [42] (Fig. 2.8), which make the dentin in and under the hybrid layer more prone to cohesive failures due to the polymerization shrinkage (Fig. 2.9) and under occlusal forces. *In vitro* experiments have shown

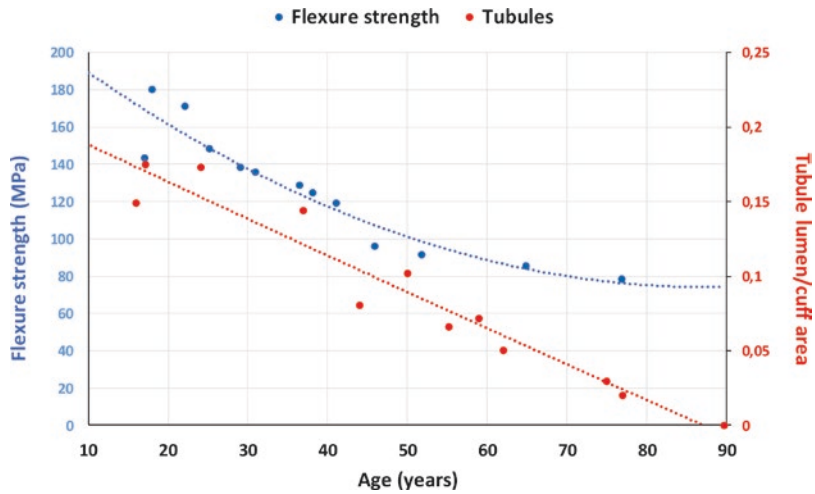

Fig. 2.7 The change in average dentin tubular lumen dimensions (red) and the influence of age on the strength of coronal dentin (blue) in adult human third molars. "Cuff" indicates peritubular dentin. The average reduction in strength over the adult age span is 20 MPa per decade at least until approximately 50 years of age (data adapted from Arola et al. [36])

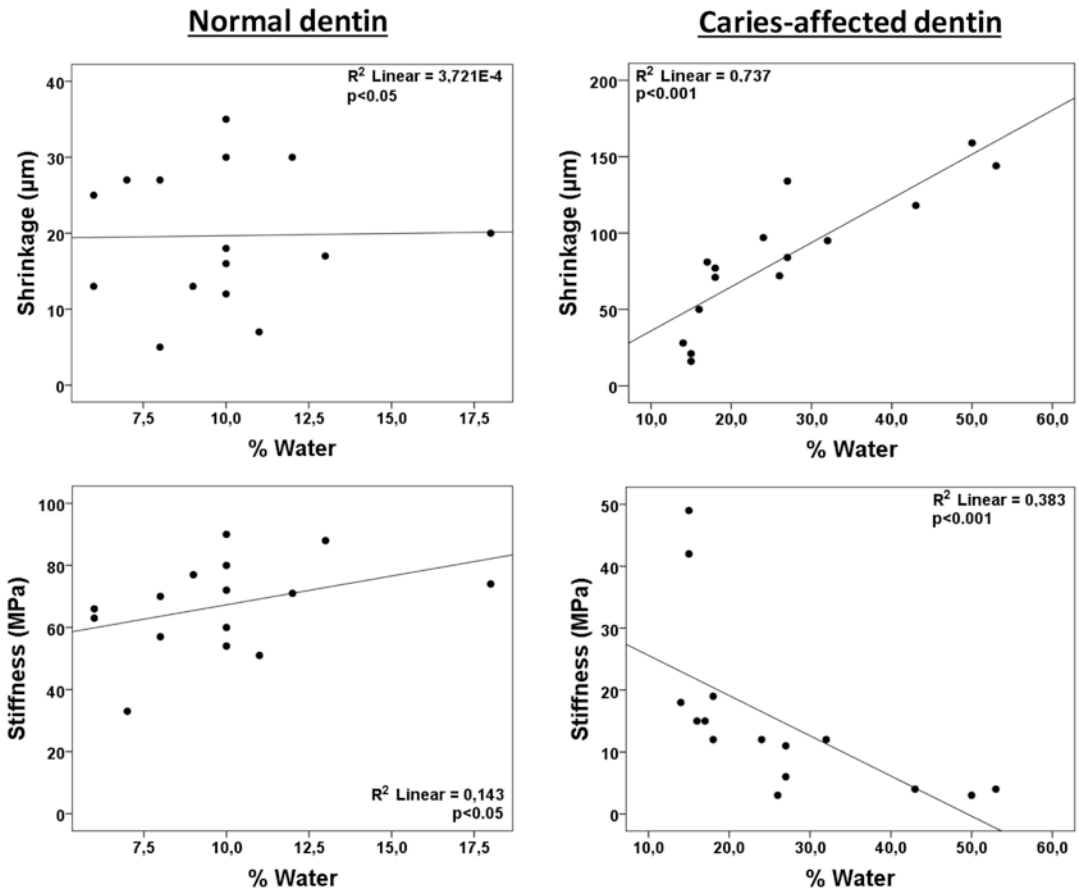

Fig. 2.8 Shrinkage and stiffness of normal (intact) and caries-affected dentin. Note the differences in both shrinkage and stiffness scales (*Y*-axis) and water content (*X*-axis) between the normal and caries-affected dentin (data adapted from Ito et al. [48])

that even short exposure of dentin to lactic acid (the acid produced by *S. mutans* and mainly responsible for caries demineralization) at pH 5 significantly reduces dentin fatigue strength, increases the rate of crack extension, and reduces the fatigue crack growth resistance [44, 45] in a way that is not prevented by sealing the tubular lumens with adhesive resin [45]. Since fatigue crack and its growth are precursors to unstable fracture, lactic acid exposure, which has occurred in caries-affected dentin and may again occur, e.g., in secondary caries, substantially increases the likelihood of restored tooth failure by fracture at lower mastication forces [45]. And finally, deep restorations (typically present in endodontically treated teeth) are more prone to cracks and fractures, not only because of the weaker structure due to loss of tooth tissue but also because of the incremental crack extension with significantly lower cyclic stresses in deep vs. superficial dentin [46].

Taken together, the age- and caries-related changes in dentin composition and structure that may have deleterious effects on dentin mechanical cannot be avoided. However, the dramatic consequences, such as catastrophic tooth fractures, can be avoided if the restorative procedures are performed not only to repair and limit the damage from caries but also to protect and preserve the tooth structure.

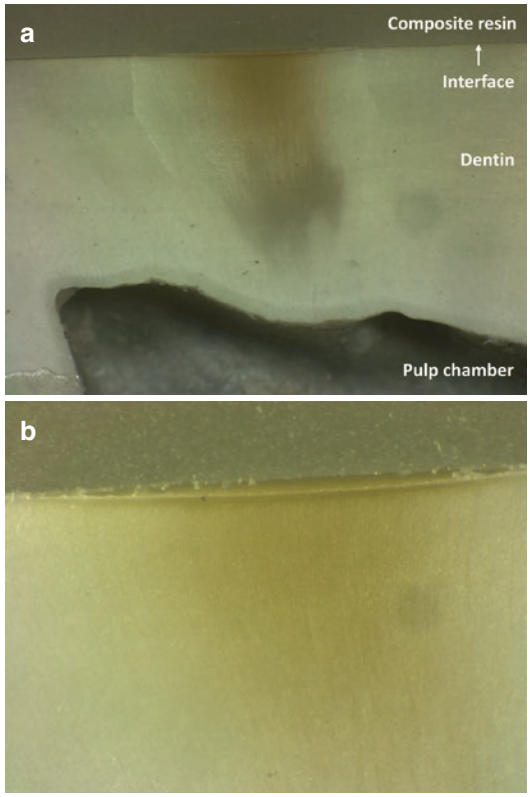

Fig. 2.9 (**a**) Dentin bonding over caries-affected dentin that clinically appeared sound without apparent discoloration. The interface between intact dentin and composite resin is tight and intact, while over caries-affected dentin the interface is clearly defective. Due to polymerization shrinkage stress and shrinkage of the less mineralized caries-affected dentin, cohesive fracture lines can be seen on both sides of the caries-affected dentin. Reflected light microscope, 20× magnification. (**b**) Higher magnification of the interface at the caries-affected dentin site, clearly demonstrating gap formation at the resin-dentin interface. Reflected light microscope, 64× magnification

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Root Canal Components

3

Ronald Ordinola-Zapata, Marco A. Versiani, and Clóvis Monteiro Bramante

Abstract

Essentially, the root canal system can be divided into pulp chamber, located within the anatomic dental crown, and the root canal space, found inside the radicular portion of the tooth. Other important components of the internal anatomy of teeth include canal orifices, apical foramina, apical ramifications, and accessory, lateral, and furcation canals. The purpose of this chapter is to describe common features of the internal anatomy in all groups of teeth, and, in addition, to provide brief comments on its impact on diagnosis and clinical procedures.

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3.1 Introduction

In 1919, Henry Morgan made an interesting analysis about what would be the object of dental education [1]. From a formal point of view, the answer was to make a dentist, a professional that practices a healing art that aims to prevent or relief human suffering. But, what would be the foundation of every healing art? The answer is the anatomy, one of the oldest branches of medicine. Knowledge of root canal anatomy is essential in order to explain the treatment plan to a patient, to properly examine radiological imaging of teeth and surrounding structures, and, most important, to perform invasive procedures. It is also required to avoid iatrogenic injuries during pulp chamber access, canal instrumentation, or post-space preparation procedures. In addition, it allows the detection of additional canals commonly correlated with persistent periapical disease. In summary, a deep understanding of the canal morphology is an imperative requirement for the success of the endodontic therapy.

Root canal treatment is indicated when the pulp tissue of a tooth is damaged or infected because of decay, trauma, iatrogenic operative procedures, or deep fillings. Different bacteria from the oral cavity can adhere to dentinal surfaces, invade the dentinal tubules and produce pulpal inflammation that, lately, may spread throughout the complexities of the root canal system, compromising the periradicular tissues. This

M. A. Versiani et al. (eds.), *The Root Canal Anatomy in Permanent Dentition*,
https://doi.org/10.1007/978-3-319-73444-6_3

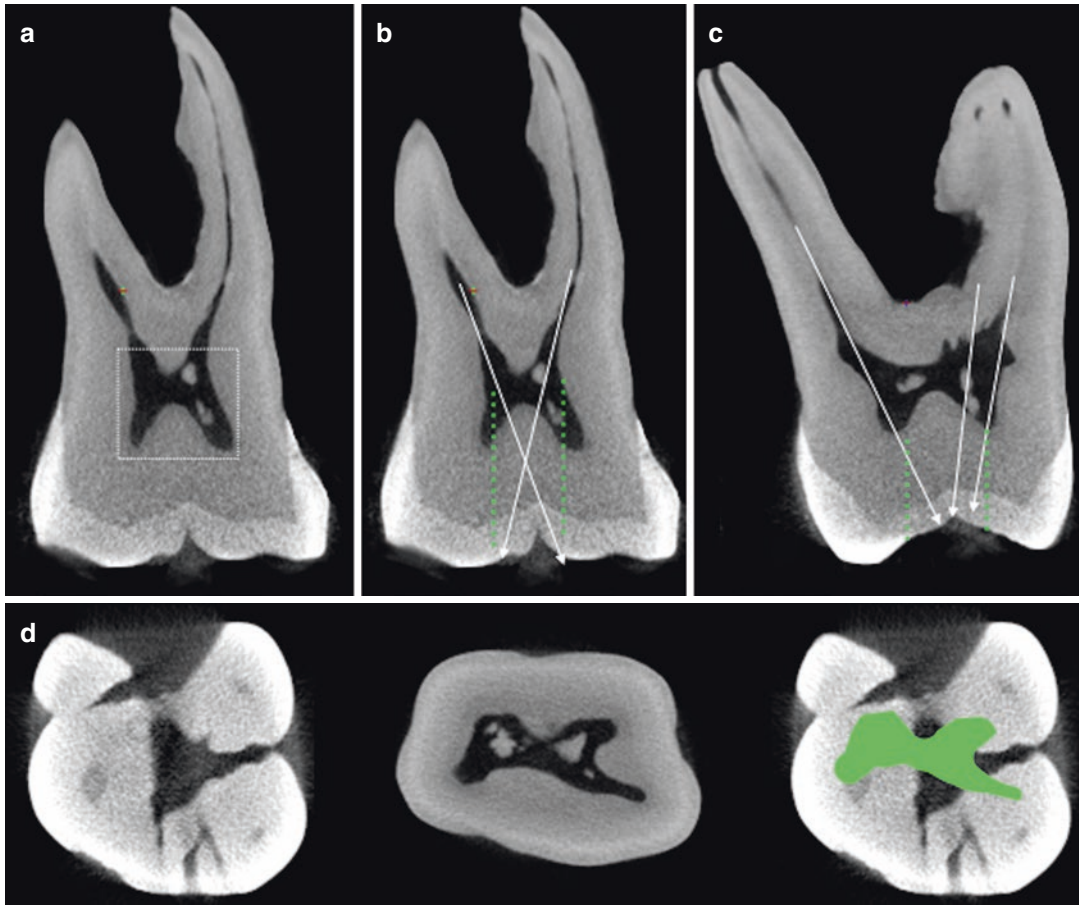

Fig. 3.1 Law of centrality, concentricity, and orifice locations observed through micro-CT sagittal and transverse cross-sectional planes from a maxillary first molar. (**a**) Detail of the pulp chamber; (**b**, **c**) projection of the root canal axis (white lines) converges to the center of the anatomical crown; (**d**) the projection of the pulp chamber floor into the coronal enamel dictates the ideal form of the access (green area)

In addition to knowing these laws, the use of illumination and magnification associated with special instruments, such as ultrasound tips, would provide the best approach to explore all anatomic variations of the pulp chamber in order to locate all canal orifices and avoid missed canals which has been considered one of the main causes of endodontic failure (Fig. 3.3). Amongst the most common missed canals associated to cases with persistent periapical disease are the second mesiobuccal canal of maxillary molars (MB2 canal), lingual canals of mandibular incisors and premolars, and the middle mesial and distolingual canals of mandibular molars.

3.2.1.1 Minimally Invasive Endodontics: Clinical Considerations

In recent years, considerable attention has been given to the anatomy of the pulp chamber. The use of the operative microscope, three-dimensional imaging technology, ultra-flexible files, and superior illumination has opened new possibilities, allowing minimally invasive procedures during endodontic treatment, mostly related to changes in access preparation (Fig. 3.4). This approach aimed to preserve tissue from healthy tooth structure in order to avoid structural failure in the future. This concept overlooks the traditional requirements of straight-line access