# Treatment of Endodontic Infections

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# Preface

Endodontics is the dental discipline that plays an essential role in promoting oral health and may also contribute to systemic health by helping reduce the total oral infectious bioburden. As a healthcare discipline, endodontics is primarily concerned with prevention and treatment of apical periodontitis, an inflammatory disease of infectious etiology that may present with different clinical manifestations and potential complications. Along with caries and marginal periodontitis, apical periodontitis is one of the most frequent biofilm-induced oral diseases that affect humans. Given its infectious origin, it is widely recognized that understanding the microbiology of endodontic infections and the host response to them is the foundation for establishment of evidence-based treatment that offers a predictable outcome.

This book was conceived and built upon the perception that basic science knowledge needs to be integrated with clinical endodontic practice, especially when it comes to host-pathogen interaction issues. To accomplish this purpose, it is divided into two sections: the first one deals with diverse aspects of microbiologic and immunologic issues related to apical periodontitis. This part intends to provide the reader with a strong basis and solid background for a thorough understanding of the treatment strategies that are discussed in section 2. The latter, in turn, focuses on the principles and practice of treatment of the different types of endodontic infections based on current concepts of etiology and pathophysiology of the disease, which were provided in the first section. Ultimately, this is a book about the core of endodontics that documents the latest research on the subject and attempts to narrow the gap between research and clinical practice. It is expected that students, clinicians, professors and researchers will all benefit from the information presented in this book.

Most chapters were written by myself. By that, I intended to provide a uniform view of diverse aspects of the basics and treatment of endodontic infections. I have been working intensely on these specific aspects of endodontics over the last 15 years, so it was time to put all this information together in a single work. In six chapters, I had the invaluable help of my associate in the lab, wife and soul mate Isabela Rôças as a co-author. Actually, none of this would have been possible without all of her love, passion and support. In another chapter, I was honoured to share its authorship with Hélio Pereira Lopes, with whom I have already written many papers and three editions of a textbook about endodontics in Portuguese. To me, he is certainly one of the greatest authorities in the field of endodontics in the world.

I am also indebted to so many friends that in different ways lent a hand for me to finish this book. They include Michael Hülsmann, Domenico Ricucci, Gilberto Debelian, Craig Baumgartner, and my team in Estácio de Sá University, especially Julio Machado de Oliveira, Flávio Ferreira Alves and José Cláudio Provenzano. I would also like to express my gratitude to the Quintessence team, especially Mr. Johannes Wolters, who believed in this project and made it possible, and to Daniel Jenk and Suzyon O'Neal Wandrey, for the patient, competent and brilliant work of editing this material.

José F. Siqueira, Jr. DDS, MSc, PhD

## Dedication

TO MY WIFE, ISABELA; MY CHILDREN, ESTHER, MARCUS VINÍCIUS AND THAÍS; AND MY PARENTS JOSÉ AND LÉA, FOR ALL THEIR LOVE, PATIENCE AND SUPPORT.

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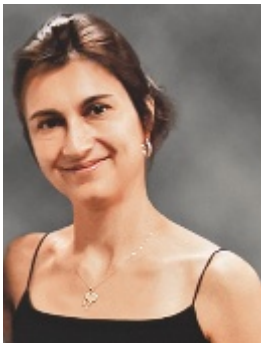


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# Introduction to Section 1

Apical periodontitis is essentially an inflammatory disease of microbial origin caused primarily by infection of the root canal system (Figure S1-1).[8] Although chemical and physical factors can induce periradicular inflammation, a large body of scientific evidence indicates that endodontic infection is essential to the progression and perpetuation of the different forms of apical periodontitis.[1-3,13] Endodontic infection only develops in root canals devoid of host defenses, either as a consequence of pulp necrosis (as a sequel to caries, trauma, periodontal disease, or iatrogenic operative procedures) or pulp removal (treatment).

Although fungi and, more recently, archaea and viruses have been found in association with endodontic infections,[7,10,12,14] bacteria are the primary microorganisms implicated in the pathogenesis of apical periodontitis. More than 460 bacterial species and phylotypes belonging to 100 genera and 9 phyla have been detected in the different types of endodontic infections.[11] In the advanced stages of the endodontic infectious process, bacterial organizations resembling biofilms can be observed adhered to the canal walls.[4-6,9] Therefore, apical periodontitis has been included in the group of biofilm-induced oral diseases.[6]

Bacteria colonizing the root canal system gain access to the periradicular tissues via apical and lateral foramina and root perforations. As a consequence of the clash between bacteria and host defenses, inflammatory and immunologic reactions take place in the periradicular tissues (Figure S1-2). Although protective, these defense mechanisms can also be destructive and induce the development of apical periodontitis. Depending on several bacterial and host-related factors, endodontic infections can lead to acute or chronic forms of apical periodontitis. Because histopathological conditions do not always correlate with clinical symptoms, apical periodontitis may be clinically symptomatic or asymptomatic.

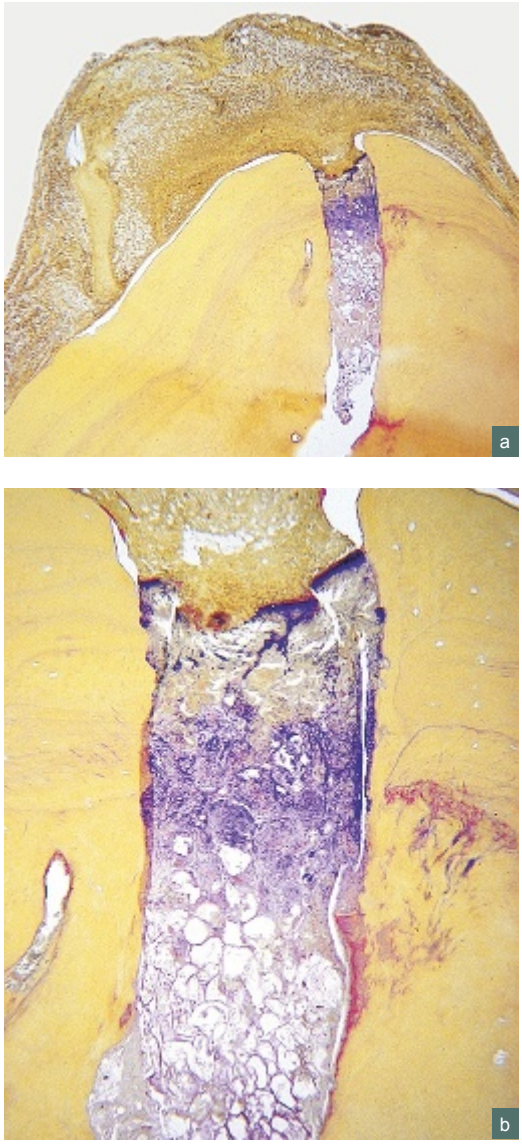


**Fig S1-1 a)** Apical periodontitis is an inflammatory disease primarily caused by bacteria infecting the root canal system. **b)** Note the border between infection and defense near the apical foramen (courtesy of Dr. Domenico Ricucci).

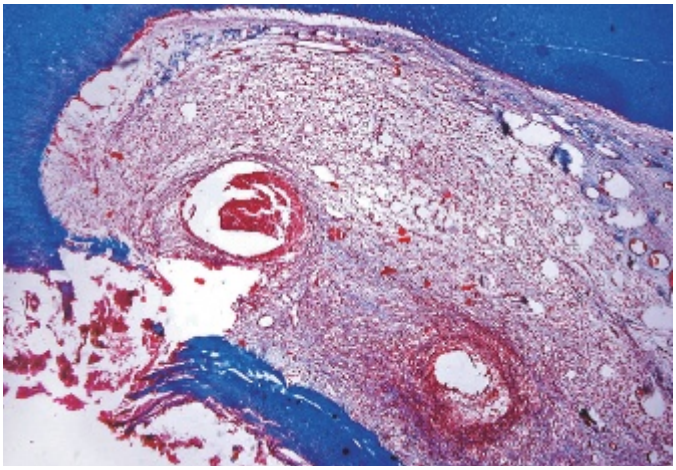

**Fig 4-3** Pulp exposure due to caries. Note the presence of microabscesses in the tissue area adjacent to exposure.

encased in a low-compliance environment. If the aggression is severe, tissue pressure may exceed that of thin-walled venules, which are compressed and collapse. Consequently, drainage is impeded and stagnation of blood flow not only promotes increased blood viscosity, but also impairs removal of waste products. This may lead to cell death and tissue necrosis. In addition, several bacterial products are toxic to host cells and may contribute to necrosis in the area. Neutrophils attracted to the area may also contribute to tissue damage by releasing enzymes and oxygen-derived products that degrade tissue components. The intensity and duration of the aggression will influence the severity of the tissue response.

This sequence of events occurs only in the tissue area adjacent to the bacterial front, not in the entire extent of the pulp. Tissue pressure near the site of inflammation is almost normal and shows no signs of severe inflammation, indicating that tissue pressure changes do not spread rapidly.[33] A pressure difference of 8 to 10 mm Hg has been measured between inflamed and non-inflamed adjacent areas of pulp.[26,77,87] In the area a few millimeters away from the inflamed area, tissue pressure has been shown to be very close to the normal pulp pressure. This difference in pressure can be a result of several edema-preventing mechanisms involved in maintenance of the physiological normality of the tissue not being directly insulted. The increase in tissue pressure may, in turn, force fluid back into lymphatics and capillaries in the nearby non-inflamed tissue, thus lowering the tissue pressure.[27,76,77] Resilience of the ground substance of the pulp tissue may help prevent the spread of pressure throughout the pulp [87].

It is now recognized that total pulp necrosis is a result of the gradual accumulation of local necrosis.[33,84,93] Once a pulp tissue compartment becomes necrotic, the front of infection advances in an apical direction. Consequently, the tissue insult occurs in the area immediately adjacent to the infected region, which responds with the same inflammatory vascular events discussed above. Therefore, after pulp exposure to caries, pulp tissue compartments are subjected to aggression by bacteria and become inflamed, necrotic and eventually infected.

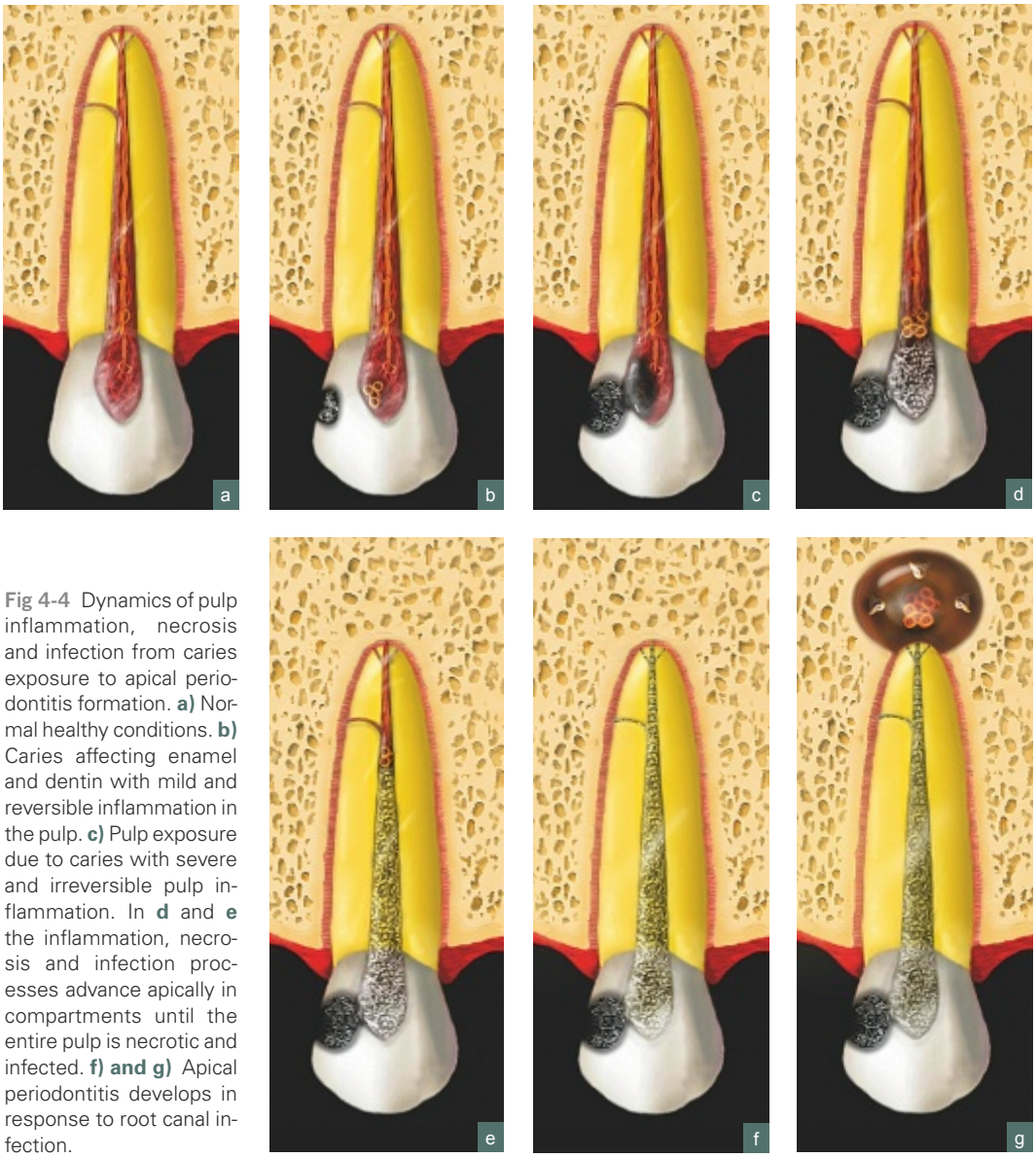


**Fig 4-4** Dynamics of pulp inflammation, necrosis and infection from caries exposure to apical periodontitis formation. **a)** Normal healthy conditions. **b)** Caries affecting enamel and dentin with mild and reversible inflammation in the pulp. **c)** Pulp exposure due to caries with severe and irreversible pulp inflammation. In **d** and **e** the inflammation, necrosis and infection processes advance apically in compartments until the entire pulp is necrotic and infected. **f) and g)** Apical periodontitis develops in response to root canal infection.

In brief, these events of *injury, inflammation, necrosis* and *infection* occur in the pulp in tissue increments that coalesce and migrate apically until the entire pulp is necrotic and infected (Fig 4-4). This explains why different stages of the disease process can be observed throughout the pulp at different times. For instance, the radicular pulp may be non-inflamed even if necrosis is present in the area of pulpal exposure or the coronal pulp is severely inflamed in response to bacterial invasion.[36]

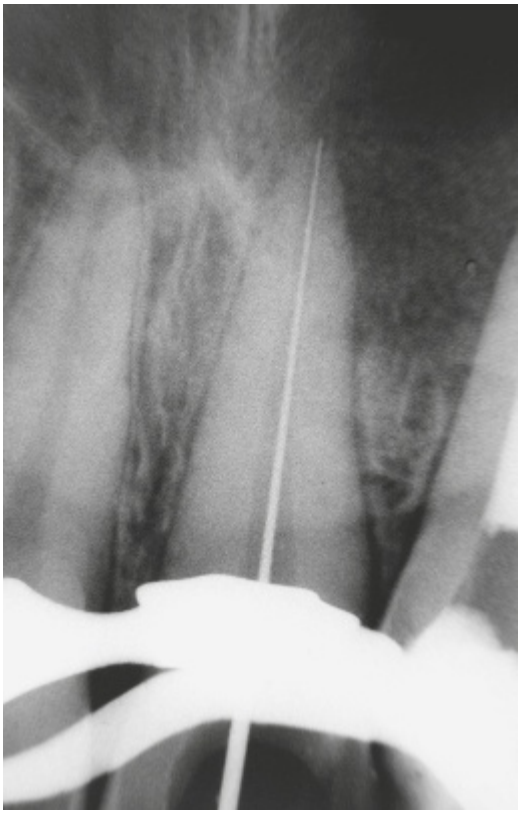

**Fig 12-32** Patency length.

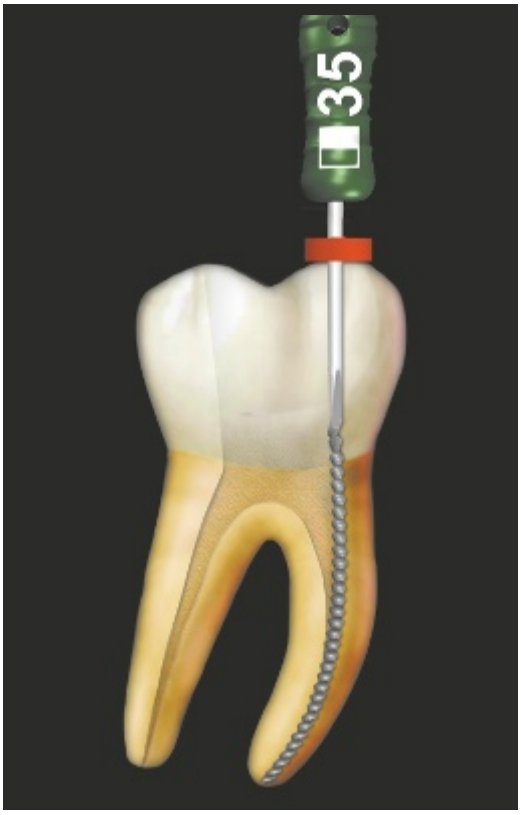


**Fig 12-33** Minimum apical preparation size for proper disinfection.

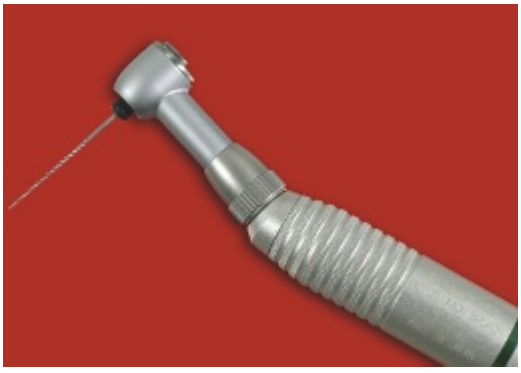

**Fig 12-34** TEP-E10R reciprocating/quarter-turn handpiece (NSK).

An example of the ARM technique for preparation of a curved canal is shown in Table 12-1 and Figure 12-35. This technique has been taught for many years at the authors' dental school and has achieved excellent results as part of the antimicrobial clinical strategy (Fig 12-36).[187,190]

## Engine-driven rotary instrumentation

Following the introduction of NiTi alloy for manufacturing endodontic instruments and their excellent performance in laboratory and clinical studies, a large variety of engine-driven

**Table 12-1** Alternating rotary motion (ARM) sequence for preparation of the mesial canals of mandibular molars

| ARM TECHNIQUE |
|---|
| 1. Coronal access preparation |
| 2. Location of the root canal orifices |
| 3. Negotiation - (estimated radiographic length or apex locator) |
| Stainless steel instruments #08 and #10 |
| Pathfinders (C+ file) |
| 4. Radicular access |
| NiTi instruments #15, 20, 25, 30, 35, 40 (until resistance) |
| GG #2 and #3 or LA Axxess #20 burs (until resistance) |
| 5. Establishment of working length |
| NiTi #15 |
| Patency length (PL) = 22 mm |
| Working length (WL) = 21 mm |
| 6. Establishment of apical patency |
| NiTi #15 (PL = 22 mm) |
| 7. Apical preparation |
| NiTi #25 (WL = 21 mm) |
| NiTi #15 (PL = 22 mm) |
| NiTi #30 (WL = 21 mm) |
| NiTi #15 (PL = 22 mm) |
| NiTi #35 (WL = 21 mm) |
| NiTi #15 (PL = 22 mm) |
| 8. Step-back |
| NiTi #40 (20 mm) |
| NiTi #15 (PL = 22 mm) |
| NiTi #45 (19 mm) |
| NiTi #15 (PL = 22 mm) |
| NiTi #50 (18 mm) |
| NiTi #15 (PL = 22 mm) |
| NiTi #55 (17 mm) |
| 9. Recapitulation and apical clearing |
| NiTi #15 (PL = 22 mm) |
| NiTi #35 (WL = 21 mm) |

NiTi rotary instruments with different designs have become commercially available. NiTi rotary instruments are generally designed to be used in continuous clockwise rotation obtained through air-driven or electric motors usually operated at a low speed (150 to 600 rpm) and low torque (0.1 to 5 Newton centimeter – Ncm). An exception is the LightSpeed instrument, which is recommended for use at 1,500 to 2,000 rpm. Low-speed handpieces on standard dental units have speeds ranging from 5,000 to 20,000 rpm and a working torque of about 2.5 Ncm compared to speeds of 380,000 to 450,000 rpm and torques of 0.1 to 0.5 Ncm in high-speed handpieces. In most electric motors available for use in NiTi rotary instrumentation, speed and torque may be programmed by the clinician and/or preset by the manufacturer.

NiTi alloy has indisputably led to the manufacturing of reliable rotary instruments with improved capabilities for shaping root canals. However, rotary instruments may be prone to a higher risk of fracture during instrumentation.[45,68] Before stainless steel instruments fracture, they usually become deformed, exhibiting severe bending or unwinding of flutes. NiTi instruments, on the other hand, can fracture without any visible signs of deformation. Thus, visual inspection is not a reliable method for evaluating the physical integrity of NiTi rotary instruments.[142]

Fracture of NiTi rotary instruments occurs either by torsional stress or cyclic flexural fatigue.[51] Torsional fracture occurs when the tip of the instrument is locked in the canal while the shaft continues to rotate. Cyclic fatigue occurs when an instrument rotates within its elastic limit in a curved canal. In the latter situation, every bent portion of the instrument is subjected to mechanical loading, represented by alternate compressive and tensile stresses. The continuous repetition of such stresses leads to the low cyclic fatigue of the instrument.

Cyclic fatigue resistance refers to the number of cycles an instrument is able to resist under specific loading conditions. Since the