Combining the theory and practice of endodontology, this book uses a problem-based approach to allow readers to apply their existing knowledge to a range of clinical scenarios, as well as to gain new knowledge and confidence in the field. Aimed at dental students, those working towards specialisation in dentistry and clinicians, the book encourages readers to transfer knowledge and diagnostic skills from one problem to another and improve their self-directed learning and reflective skills.

Pitt Ford’s Problem-Based Learning in Endodontology includes a wealth of cases that span topics such as the maintenance of a vital pulp, root canal treatment, surgical endodontics and trauma. Each case is accompanied by full colour photographs and radiographs that illustrate the key stages in diagnosis, treatment planning, treatment and prognosis. In addition, the detailed commentary provides information on viable alternative treatment strategies, rationale for the treatment described, evaluation of the current evidence for and against the course of treatment and finally the prognosis.

Key features
- Clinically relevant to every day practice and thoroughly grounded in scientific evidence
- Uses a problem-based approach to help readers solve problems for themselves
- Contains clinical cases spanning a wide range of topics
- Richly illustrated in full colour throughout with high quality photos and radiographs
- Written by an international list of contributors

The Editors
Shanon Patel is a specialist in endodontics; his time is divided between working in specialist referral practice in London, and teaching in the Endodontic Postgraduate Unit at King’s College London Dental Institute where he is involved in the training of future specialists. He has published over 30 papers in peer reviewed scientific journals, written two book chapters, co-authored the textbook Principles of Endodontics and is completing a PhD assessing the applications of Cone Beam CT in endodontics.

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The discipline of Endodontics encompasses the diagnosis and treatment of human dental pulp and periapical tissues in both health and disease. The principle aim of Endodontics is to preserve the natural dentition and provide function, comfort and esthetics. Many advances have been made in the field of Endodontics within the last few years. Successful root canal treatment relies upon elimination of microorganisms present in the root canal systems, prevention of recontamination after treatment and restoration of these teeth to function. The subject of Endodontontology is often viewed as both a science and an art. The science of Endodontics deals with biological and pathological conditions of pulp and periapical tissues, while the art of Endodontics covers technical procedures performed during root canal treatment. However, perhaps the real art is establishing a connection between the information gathered at examination and integrating this with the dentist’s knowledge, experience and technical skill. The problem-based learning approach employed in this book is an innovative way in which to connect the science and art of Endodontics using the best clinical evidence.

This book contains information regarding diagnosis and treatment planning for a range of pulp and periapical conditions, as well as technical advice regarding access preparations, cleaning and shaping, obturation and temporization and restoration. The distinctive features of this book are: each chapter lists specific objectives, provides the patient’s chief complaint, medical and dental histories, clinical examination details, diagnosis and treatment planning. It then gives a scientific description of the condition, its treatment, relevant references and asks pertinent questions relating to the condition.

Professor Tom Pitt Ford was a master of simplifying complex problems. What he had in mind when he discussed the format of this book to its current editors was to be interactive with the reader and keep his or her focus on a specific problem and issue. I really think they have delivered what Tom envisaged. In other words, this book teaches pre-doctoral students and new practitioners why, when and how to perform root canal treatment. This unique approach provides the reader with the necessary information to perform root canal treatment and gives them an opportunity to understand the technical aspects of Endodontics and the science underpinning them.

Mahmoud Torabinejad
Professor of Endodontics
Loma Linda University
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Preface

The aim of this textbook is to challenge the reader to assume the role of problem solver. The process of problem-based learning is an interactive process, in which the participants develop their own learning goals through careful consideration of clinical scenarios before sourcing the answers. It is our aim for the reader to be stimulated by the clinical scenarios described, and to delve further into the literature should they desire; in this sense the text can be seen as an adjunct to existing conventional textbooks. We accept that this book does much of the ‘brainstorming’ aspect of problem-based learning, supplying the reader with a basic but not exhaustive answer. It is hoped that adopting a problem-based approach and providing a skeleton answer will lead to a deeper knowledge of clinically relevant problems.

When planning the book content, there were numerous discussions between the co-editors on what should, and should not be included. From the outset we unanimously agreed that we wanted to emphasise the biological rationale of Endodontics, rather than produce a ‘recipe’ book on ‘how to do it’. It was not our intention to encompass every aspect of Endodontology, instead, our aim was to describe the key areas of clinical endodontics by way of vignettes with pertinent questions contained within the text. Broader consideration was also given to areas not covered in standard endodontic texts such as pain of non-odontogenic origin, and technological advances including the use of cone beam computed tomography in Endodontics.

It is hoped that this book will be read not only by senior undergraduates who wish to consolidate their knowledge, but also by postgraduates preparing for the Royal College diplomas in the United Kingdom and Ireland, and Advanced Education in General Dentistry Programs in North America. In addition, US Board candidates, United Kingdom and Australian specialists in training candidates taking their final ‘exit’ diploma in non-Endodontic disciplines such as Prosthodontics and Periodontics should find this book invaluable as they also require a sound understanding of Endodontology and its relevance to their disciplines.

This book was the brainchild of the late (and very great) Professor Tom Pitt Ford, he initially led this project. It was his desire to deliver a problem-based endodontic textbook authored by a truly international group of academics and clinicians. It is particularly sad for us that he is not present to witness the publication of this book. Many of the contributors to this book were lucky enough to have been taught by him and/or worked alongside him, and will never
Preface

forget his encyclopaedic knowledge of Endodontology, patience and meticulous attention to detail. He enlisted a group of editors and contributors, and indeed was the lead co-editor himself prior to his untimely death in 2008. The title of the book bears his name as recognition not only of Tom’s contribution to this book, but more importantly to his contribution to Endodontology.

Shanon Patel, London, UK
Henry F. Duncan, Dublin, Ireland
Section 1
Aetiology and Diagnosis

Case 1.1 Microbiology of primary periapical periodontitis
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Case 1.2 Chronic periapical periodontitis
Domenico Ricucci

Case 1.3 Chronic periapical periodontitis with suppuration
Domenico Ricucci

Case 1.4 Chronic periapical periodontitis with an extraoral sinus
Philip Mitchell

Case 1.5 Periodontal-endodontic lesions
Markus Haapasalo and Hanna Haapasalo
Case 1.1
Microbiology of primary periapical periodontitis
José F. Siqueira Jr and Isabela N. Rôcas

Objectives

Periapical periodontitis is an inflammatory disease that affects the tissues surrounding the apical portion of the root, and is primarily caused by microorganisms infecting the root canal system (Figure 1.1.1). At the end of this case, the reader should be able to recognize the infectious origin of periapical periodontitis as well as understand the basic aspects of the microbiology of endodontic infections.

Introduction

A 29-year-old female was seeking replacement of defective and discoloured composite restorations in her upper incisor teeth (12,11 and 21). These teeth had been restored approximately 5 years ago.

Chief complaint

The patient complained of the poor appearance of the existing restorations in her maxillary incisor teeth. She recalled that these teeth were sensitive to cold and/or sweet foods or drinks. She saw her dentist who advised her that she had several carious lesions in these teeth, with her consent these teeth were then restored with direct composite restorations. The teeth had been asymptomatic since then.

Medical history

Unremarkable.
Dental history

The patient’s last dental check-up was 3 years previously. At that time, no periapical lesions were detected radiographically. The patient had also addressed her diet, including reducing the quantity and frequency of carbonated drink consumption.

Clinical examination

The extraoral examination was unremarkable. The patient had a moderately restored dentition, and her oral hygiene status was good. Composite restorations in all upper incisors were defective and discoloured.

All the maxillary anterior teeth responded normally to vitality test except for the 12. A periapical radiograph revealed a periapical radiolucency associated with the 12 (Figure 1.1.1). No swelling or sinus tracts were detected. Removal of the defective coronal restorations from the 12 resulted in exposure of the pulp, however, the root canal was necrotic. Caries remnants on the cavity walls were observed underneath the mesial composite restoration.

Diagnosis and treatment planning

What was the diagnosis?

The diagnosis was chronic periapical periodontitis associated with an infected necrotic root canal. The cause of pulp necrosis was very likely coronal leakage and caries exposure, although the possibility that the pulp may have been iatrogenically exposed during caries excavation.

Figure 1.1.1

(a) Periapical radiograph of the 12, demonstrating an apical radiolucency, (b) schematic drawing covering the same radiograph to illustrate the major biologic events involved. The root canal is necrotic and infected and an inflammatory response associated with bone resorption developed at the periradicular tissues (periapical periodontitis). This is an attempt to prevent spread of the infection to the bone and other body sites.
Microbiology of primary periapical periodontitis

should be considered. The exposed pulp may have been vital (although irreversibly inflamed) or already necrotic at the time of the previous course of restorative treatment. If the pulp was still vital at that time, caries associated with a leaking restoration may have maintained the insult to the pulp tissue, resulting in pulp necrosis.

**What treatment should be carried out in this case?**

Endodontic treatment should be performed, followed by placement of a well-adapted plastic composite restoration. The defective and discoloured restorations in the other maxillary incisors should also be replaced.

**What are the goals of antimicrobial endodontic treatment?**

The ultimate goal of the endodontic treatment is to maintain or restore health of the periapical tissues. The treatment of teeth with irreversibly inflamed pulps is essentially a prophylactic approach, since the radicular vital pulp is usually free of infection, and so the rationale is to treat the root canal to prevent further pulp necrosis and infection which would eventually result in periapical periodontitis. On the other hand, in cases of infected necrotic pulps like the case described here, an intraradicular infection is already established and, as a consequence, endodontic treatment should focus not only on prevention of introduction of new microorganisms, but also on elimination of those colonizing the root canal.

Entrenched in the protected anatomy of the root canal system, bacteria are beyond the reach of the host defences and systemically administered antibiotics. Therefore, endodontic infections can only be treated by means of endodontic treatment using antibacterial procedures.

Treatment procedures should ideally render the root canal system free of microorganisms. However, given the complex anatomy of the root canal system, it is widely recognized that, with available instruments, irrigants and preparation techniques, fulfilling this goal is virtually impossible for the vast majority of cases. Therefore, the realistic goal is to reduce bacterial populations to a level below that necessary to induce or sustain periapical disease. The clinician should adopt an evidence-based antibacterial protocol that predictably disinfects the root canal and allows this goal to be accomplished.

**Discussion**

**How does caries cause pulp necrosis and subsequent periapical periodontitis?**

Bacteria within carious lesions are organized in authentic biofilms; if left untreated, this carious front advances towards the pulp and simultaneously the tooth structure is destroyed in the process. Diffusion of bacterial products through dentinal tubules induces pulp inflammation long before the pulp is exposed. After exposure, the pulp surface becomes colonized and covered by bacteria from the caries biofilm and becomes severely inflamed (Figure 1.1.2). Some tissue invasion by bacteria may also occur. As a response to the sustained bacterial challenge, the pulp tissue invariably undergoes necrosis and then loses the ability to contain the bacterial
Figure 1.1.2
Histologic section of a tooth with caries exposure. The pulp was vital, but severely inflamed at the area of exposure (Gomori’s trichrome staining).

invasion. Eventually, invading bacteria colonize the necrotic pulp tissue. If left untreated, the events of bacterial aggression, pulp inflammation, necrosis and subsequent infection gradually move towards the apical portion of the root canal until virtually the entire root canal is necrotic and infected.

Bacteria colonizing the necrotic root canal will then induce damage to the periapical tissues and give rise to inflammatory changes. Bacteria exert their pathogenicity by wreaking havoc on the host tissues through direct and/or indirect mechanisms. Bacterial virulence factors that cause direct tissue harm include those that are toxic to host cells and/or disrupt the intercellular matrix of the connective tissue. Furthermore, bacterial structural components stimulate the development of host immune reactions capable not only of defending the host against infection, but also of causing severe tissue destruction. Pus formation in acute apical abscess and bone resorption associated with chronic periapical periodontitis are clear examples of tissue destructive effects indirectly caused by bacteria. They are indirect because of being promoted by the host itself in defence against bacterial infection.

In addition to caries lesions, are there other avenues for endodontic infection?
Under normal conditions, the pulp–dentine complex is isolated and protected from the oral microbiota by the overlying enamel and cementum, the same way the connective tissues elsewhere in the body are segregated from the microbiota residing in body cavities and surfaces by the epithelium of mucosa or skin. Once the integrity of these natural layers is breached (for example; as a result of caries, trauma-induced fractures and cracks, restorative procedures, scaling and root planning, attrition or abrasion) or naturally absent (for example; because of gaps in the cemental coating at the cervical root surface), the pulp–dentine complex will be exposed to the oral environment. This complex is then challenged by microorganisms present in
carious lesions, saliva bathing the exposed dentinal area or in the dental biofilm formed onto the exposed area. The subgingival biofilm associated with periodontal pockets may also represent a source of microorganisms which may access the pulp via dentinal tubes at the cervical region of the tooth or through lateral and apical foramina.

Whatever the route of bacterial access to the root canal, necrosis of pulp tissue is a prerequisite for the establishment of primary endodontic infections. As long as the pulp is vital, it can protect itself against bacterial invasion and colonization. However, if the pulp becomes necrotic as a result of caries, trauma, operative procedures or periodontal disease, the necrotic tissue can be very easily infected. This is because host defences do not function in the necrotic pulp tissue.

**What is the difference between a primary and secondary infection?**

Primary (endodontic) infections occur in untreated teeth. Microorganisms may also be detected in root canals after professional endodontic intervention (secondary infection), either by a breach in the aseptic chain during endodontic treatment, by coronal leakage through temporary/definitive restorations or by tooth/restoration fracture.

**Why do some traumatized teeth develop periapical periodontitis even when the unrestored crown looks intact?**

Bacteria have been isolated from the root canal of traumatized teeth whose pulps became necrotic and periapical periodontitis developed, even in circumstances where the tooth crown was apparently intact. How did those bacteria invade the pulp space? In the past, it was believed that such bacteria originated from the gingival sulcus or periodontal pockets and reached the necrotic canal via severed blood vessels of the periodontium, a phenomenon called anachoresis. This theory has no evidence base. In reality, trauma can induce exposure of dentin by fracturing the crown or inducing the formation of enamel cracks which can be microscopic or macroscopic. A large number of dentinal tubules can be exposed to the oral environment by a single crack. These cracks can become clogged with an oral bacterial biofilm, thus providing portals of entry for bacteria. If the pulp remains vital after trauma, bacterial penetration into tubules is counteracted by the dentinal fluid and/or tubular contents. On the other hand, if the pulp becomes necrotic as a consequence of trauma, it loses the ability to protect itself against bacterial invasion and the dentinal tubules become true avenues through which bacteria can reach the pulp.

**Which microorganisms are commonly found in primary endodontic infections?**

Although fungi and, most recently, archaea and herpes viruses have been found in association with endodontic infections, bacteria are the main microorganisms implicated in the pathogenesis of the different forms of periapical periodontitis.

Primary endodontic infections are dominated by anaerobic bacteria organized in a mixed community (Table 1.1.1). Overall, between 10 and 20 different species can be found per root canal. As for population density, each canal can harbour from $10^3$ to $10^8$ bacterial cells in chronic periapical periodontitis cases and from $10^4$ to $10^9$ in acute forms of the disease. Root canals
associated with large apical lesions harbour a more diverse and populous microbiota. Bacterial
named species, frequently detected in primary infections of teeth with either acute or chronic
periapical periodontitis, are classified in Table 1.1.1.

Is there a difference between the endodontic microbiota in symptomatic (for
example acute periapical abscess) and asymptomatic cases?
The diversity of the bacterial communities is comparatively higher in acute cases than in canals
of teeth with chronic periapical periodontitis. Differences are essentially represented by different
dominant species in the communities and larger number of species in the acute disease. However,
there is no strong evidence supporting the specific involvement of a single species with any
particular sign or symptom of periapical periodontitis. Some gram-negative anaerobic bacteria
have been linked to symptoms; however, the same species also have been encountered in
asymptomatic cases. Other factors, in addition to the presence of a pathogenic species, are
thought to influence the emergence of symptoms (Table 1.1.2).

### Table 1.1.1  Features of the endodontic microbiota in primary periapical periodontitis

<table>
<thead>
<tr>
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<tr>
<td>Community</td>
<td>Mixed</td>
</tr>
<tr>
<td>Mean number of species/case</td>
<td>10–20</td>
</tr>
<tr>
<td>Mean number of cells/case</td>
<td>$10^3–10^5$</td>
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<tr>
<td>Most prevalent groups</td>
<td>Gram-negative/gram-positive anaerobes</td>
</tr>
<tr>
<td>Most frequent taxa</td>
<td>Gram negative</td>
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<tr>
<td></td>
<td><em>Treponema</em> spp.</td>
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<tr>
<td></td>
<td><em>Tannerella</em> forsythia</td>
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<td></td>
<td><em>Porphyromonas</em> spp.</td>
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<tr>
<td></td>
<td><em>Dialister</em> spp.</td>
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<td></td>
<td><em>Fusobacterium</em> nucleatum</td>
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<tr>
<td></td>
<td><em>Synergistes</em> spp.</td>
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<td><em>Eikenella</em> corrodens</td>
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<td></td>
<td><em>Campylobacter</em> spp.</td>
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<td></td>
<td>Gram-positive</td>
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<td><em>Filifactor</em> alocis</td>
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<td><em>Pseudoribamibacter</em> alactolyticus</td>
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<tr>
<td></td>
<td><em>Olsenella</em> spp.</td>
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<tr>
<td></td>
<td><em>Peptostreptococcus</em> spp.</td>
</tr>
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<td></td>
<td><em>Streptococcus</em> spp.</td>
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</tbody>
</table>

8
How do microorganisms flourish in the necrotic root canal? Is there a selective pressure dictating the composition of the infecting microbiota?

A root canal with a necrotic pulp provides an ideal environment for bacterial colonization as it is moist, warm, nutritious and anaerobic. This environment is by and large protected from the host defences because of lack of an active blood circulation in the necrotic tissue. The main sources of nutrients for bacteria in the necrotic root canal are shown in Table 1.1.3. Although a large number of bacterial species (~700) have been identified in the oral cavity only a limited number of these species (about 10 to 20) can survive within the root canal. The major ecological determinants that influence the composition of the root canal microbiota include oxygen tension, type and amount of available nutrients and bacterial interactions.

The ecology of the endodontic microbiota is influenced by different physico-chemical conditions and type of nutrient availability in the different regions of the root canal. Gradients of oxygen tension and available nutrients are established along the extent of the root canal in such a way that the apical region contains the lowest oxygen tension and the highest concentration of proteins, while in the most coronal region, the oxygen tension and amount of available carbohydrates may be higher. These gradients allow the dominance of certain groups of bacteria.

Table 1.1.2  Factors influencing the development of symptomatic periapical periodontitis

<table>
<thead>
<tr>
<th>Factors influencing the emergence of symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Differences in virulence ability among strains of the same species.</td>
</tr>
<tr>
<td>• Additive or synergistic effects among species in mixed communities.</td>
</tr>
<tr>
<td>• Bacterial populational density (bacterial load).</td>
</tr>
<tr>
<td>• Environment-regulated expression of virulence factors.</td>
</tr>
<tr>
<td>• Host resistance (can be modulated by systemic diseases, concomitant virus infection, environmental factors and genetic patterns).</td>
</tr>
</tbody>
</table>

Table 1.1.3  Main sources of nutrients for bacteria colonizing the root canal system

<table>
<thead>
<tr>
<th>Sources of nutrients for intracanal bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Necrotic pulp tissue.</td>
</tr>
<tr>
<td>• Proteins and glycoproteins from periradicular tissue fluids and exudate seeping into the root canal.</td>
</tr>
<tr>
<td>• Components of saliva that coronally penetrate into the root canal.</td>
</tr>
<tr>
<td>• Products of the metabolism of other bacteria in a mixed infection.</td>
</tr>
</tbody>
</table>
bacteria in different regions of the canal according to their relationship to oxygen and metabolic demands.

**How are microorganisms distributed through the root canal system?**

In advanced stages of the endodontic infectious process, bacterial organizations resembling biofilms can be observed adhered to the dentinal root canal walls (Figure 1.1.3). For this reason, there is a current trend to include periapical periodontitis in the group of biofilm-induced diseases along with caries and marginal periodontitis. In addition to forming biofilms adhered on the canal walls, bacteria can also be observed as planktonic cells suspended in the fluid phase of the main canal or enmeshed in the necrotic tissue (Figure 1.1.4a). Lateral canals and isthmuses connecting main canals can also be clogged with bacteria, primarily organized in biofilms. Bacterial cells originating from biofilms adhered to the root canal walls are often seen penetrating the subjacent dentinal tubules (Figure 1.1.4b). Dentinal tubule infection can occur in around 70–80% of the teeth with periapical periodontitis. A shallow penetration is more common, but bacterial cells can be observed reaching approximately 300 μm in certain teeth.

Bacteria present as planktonic cells in the main root canal may be easily accessed and eliminated by root canal preparation. However, bacteria organized in biofilms attached to the canal walls or located into isthmuses, lateral canals and dentinal tubules are more difficult to reach and may require special therapeutic strategies to be disrupted and eliminated.

**Figure 1.1.3**

(a) Histologic section of the very apical part of the root canal of a tooth with periapical periodontitis. A bacterial biofilm (arrow) is seen adhered to the canal wall, very close to the apical foramen (AF). (b) Higher magnification of the biofilm shown in (a). Planktonic bacterial cells are also seen in the main canal (empty arrow) (Brown and Brenn staining).
Further reading


Case 1.2
Chronic periapical periodontitis
Domenico Ricucci

Objectives
At the end of this case, the reader should appreciate the pathogenesis of chronic periapical periodontitis.

Introduction
A 38-year-old male patient presented for a periodic check-up with no complaints of pain or discomfort.

Chief complaint
The patient was asymptomatic, however, she had received several restorations over the years on his posterior teeth. The lower right first molar (46) was pulp capped 2 years ago as a consequence of pulp exposure that occurred during deep caries excavation. The tooth was restored with amalgam. At a subsequent review visit the patient was asymptomatic.

Medical history
Unremarkable.

Dental history
Regular attender.