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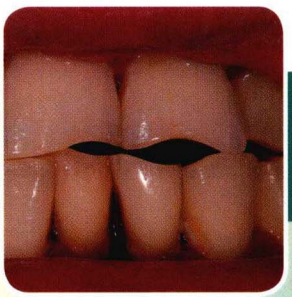
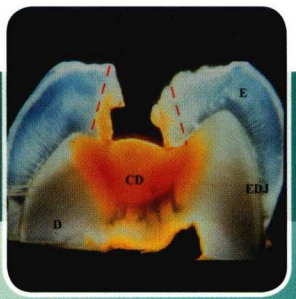
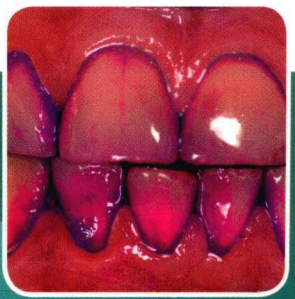
Pickard's Guide to Minimally Invasive Operative Dentistry

Tenth edition







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TENTH EDITION

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Foreword

It was a 'great pleasure and honour' to have been asked to prepare the foreword for the previous edition of Pickard. To have been asked to prepare the foreword for the new-look, new-style tenth edition is a huge honour and pleasure, given that I have been selected to introduce this ground-breaking book in the rapidly developing field of patient-centred and oral healthcare team-delivered, biologically based, minimally invasive, minimum intervention (MI) care dentistry. To practise operative dentistry in any other way in the twenty-first century is not only old-fashioned, but contrary to the best interests of patients.

If Professor Pickard were alive today, I believe he would be delighted to be associated with the ways in which operative dentistry and relevant dental biomaterials science have evolved and developed since his pioneering days as a leader in the field, and to see the tenth edition of his book promote and encourage modern MI approaches to the clinical practice of operative dentistry. I also anticipate that Professors Bernard Smith and Edwina Kidd, who were entrusted with the 'Manual' by Pickard, will be most pleased to see this new edition pushing back the frontiers of the clinical practice of operative dentistry. As described in the accompanying preface by the present custodians of the Pickard legacy—Professors Avijit Banerjee and Timothy Watson—the 'Manual' has undergone major change during the preparation of the new, more user-friendly tenth edition, including a change in title to reflect all that is new in the state-of-the-art management of diseased and damaged teeth and tooth tissues. Professors Banerjee and Watson are to be congratulated not just on the excellence and attractiveness of the tenth edition of Pickard, but on their forward-looking approach and leadership in the promotion of patient-centred, minimally invasive, minimum intervention care dentistry.

In my foreword to the ninth edition of Pickard, I referred to a 'watershed between the traditional and modern art and science of operative dentistry.' With the publication of this tenth edition of Pickard, the transition from traditional, mechanistic, and traumatic to modern, truly tooth-preserving, conservative operative dentistry may be considered to have been largely completed, one of the major outstanding challenges being the translation of all that is described and beautifully illustrated in this book into the everyday clinical practice of dentistry. For established practitioners and teachers this may necessitate a fundamental change in thinking and approach. For students (present and future) a door has been opened to an innovative, much more challenging approach to patient-centred care. For future researchers, this book highlights the many different ways in which operative dentistry and relevant dental biomaterials science must continue to evolve.

From the style developed by Professors Banerjee and Watson, it is apparent that they fully appreciate the challenge that this tenth edition of Pickard poses in terms of putting aside traditional thinking and long-established procedures and techniques in order to embrace new, preventatively orientated concepts and principles. Of particular note is the introduction in this new edition of the '5Rs' concept to enhance and extend the life expectancy of restorations, and in turn teeth, through a slowing down of the so-called 'restorative death spiral.' If you are unfamiliar with the '5Rs' concept, that alone is sufficient justification for you to acquire and study this book.

I applaud the work of Banerjee and Watson and unreservedly recommend this new edition of Pickard to all members of the oral healthcare team. I very much hope that the new knowledge and understanding that it imparts will be widely and effectively translated by all team members into clinical practice in the best interests of existing and future generations of patients. For this to happen, it is hoped that teachers will revise their curricula accordingly, and funders of oral healthcare provision will critically review the extent to which their systems allow and encourage the practice of modern, evidence-based, minimally invasive operative dentistry as described in this book.

Professors Banerjee and Watson have produced a state-of-the-art text on operative dentistry. We must now rise to the challenge to practise twenty-first rather than late twentieth-century operative dentistry. Anyone who reads this book will be hard pressed to find good reason for continuing to practise 'old-style' traditional operative dentistry. The move to 'new-style' MI operative dentistry may be challenging, but it will bring countless benefits both to patients and to the profession.

Nairn Wilson CBE DSc (*h.c.*) FDS FFD FFGDP FCDSHK FACD FADM FHEA FKC

Preface to the tenth edition

The first edition of this textbook was published in 1962. From its origins, initially with Professor Pickard at the helm, the subsequent editions have always promoted operative dentistry principles that placed tooth preservation first and foremost. Since this time there have been major advances in the science underpinning our subject. These include a better understanding of the complex pathological processes that cause hard tissue disease, along with its detection, diagnosis, and the operative technologies and adhesive/sealing dental materials used to manage the damaged tissues. This *minimally invasive* biologically based approach is now recognized as the gold standard, and we have embraced this with the change of book title of this current edition, to *Pickard's Guide to Minimally Invasive Operative Dentistry*.

The ninth edition received a major update in style and content based upon the sound foundations from its previous editions authored by Professors Smith and Kidd. In this new edition we have responded to reader feedback and have made the following changes:

- We have included a chapter on the principles of the operative management of the badly broken down tooth, leading the reader towards an appreciation of the intimate link between direct and indirect restorations.
- We have expanded the final chapter covering the long-term clinical management/maintenance of direct restorations, using the minimally invasive '5Rs' concept—*review, refurbish, reseal, repair*, and *replace*.
- With respect to referencing the information in the book, we felt that traditional reference lists at the end of each chapter become rapidly outdated in areas that are undergoing continuous evolution. Therefore in this edition we have introduced QR code images to allow the reader to access with their mobile devices original and supporting material resources through digital media. The use of keywords for searching online databases will allow the reader to review references that will evolve dynamically.

This edition has been significantly enhanced by the inclusion of more high-quality images to help illustrate scientific concepts and clinical scenarios. We must thank Dr Louis Mackenzie, who has kindly provided many of these additions. In addition, we wish to thank our many colleagues who have allowed us to use their illustrations. They are acknowledged in the captions to the relevant figures, together with the source of the original publication where applicable.

In the previous edition we reinforced the link between prevention, operative dentistry, and overall patient care. This *minimum intervention* care philosophy continues to underpin the current edition, with increasing emphasis placed upon the differing important roles of the oral healthcare team. The operative skill set of a new dental graduate has evolved to encompass not only the techniques, materials, and science of minimally invasive dentistry, but also, increasingly, the behaviour management of their patients. Without patients taking responsibility for their oral health, even the best operative dentistry will fail, regardless of the materials used. In this regard, the amalgam debate has not gone away. Indeed, as a result of the United Nations Minamata Treaty in 2013, the global environmental impact of dental amalgam has led to recommendations for a phase down in its clinical use. The treaty has highlighted the need for increased dental research into caries prevention and alternative restorative materials in conjunction with better professional education in their use. We sincerely hope that this book goes some way towards achieving the latter.

A.B.
T.F.W.
October 2014

Contents

1

Dental hard tissue pathologies, aetiology, and their clinical manifestations

Chapter contents

1.1 Introduction: why practise minimally invasive (tooth-preserving) operative dentistry?

Minimally invasive operative dentistry is that aspect of restorative dentistry which repairs and/or restores damaged and defective tooth structure directly in order to maintain pulp vitality, function, and aesthetics (see Figure 1.1). The primary goal is to respect tooth structure during this process, retaining viable and biologically repairable tissues to maintain tooth vitality for as long as possible. The hard tissue damage or defects can be caused by one or more of the following:

- caries
- tooth wear
- trauma
- developmental conditions.

Minimum intervention oral healthcare is that approach to patient management where the oral healthcare team (comprised of the dentist, nurses, oral health educators, hygienists, therapists, technicians, reception staff, and practice managers), led by the dentist, act as one to provide individualized patient-centred care and advice to encourage the patient to take responsibility for and maintain their own oral health. Minimum intervention care revolves around methods of detection/diagnosis/risk assessment of oral disease, non-operative control/prevention of these conditions, minimally invasive operative repair of tissue damage, and review/maintenance/recall of the patient and the advice/care offered by the dentist/team (see Figure 1.1). The process of care planning involves the patient, including disease prevention by behaviour change and adherence, not just listing those operative procedures offered to restore damaged or defective teeth in isolation. It must be understood from the outset that even though minimally invasive operative dentistry has a pivotal role in the 'surgical' repair of damaged teeth, it alone does not provide the actual cure for dental disease—please understand that '*drilling and filling teeth does not cure caries!*' The following sections will provide an overview of the four conditions mentioned previously with respect to their aetiology, histopathology, and microbiology where relevant. An attempt will be made to relate these features to the clinical manifestations of each condition, namely carious lesions and tooth-wear lesions.

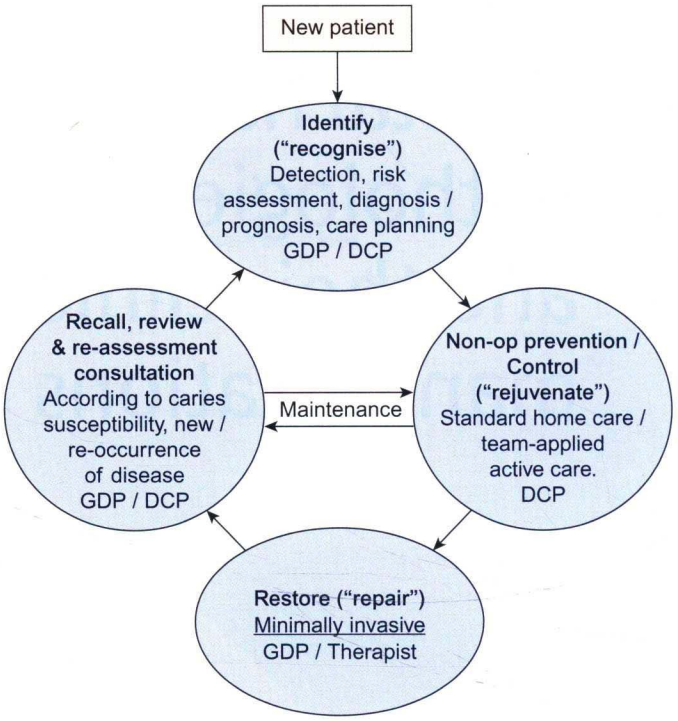

Figure 1.1 The patient-centred minimum intervention care cycle showing the four interlinking phases of patient assessment/diagnosis, non-operative prevention of lesions/control of disease, *minimally invasive* operative intervention, and review (recall). The arrows indicate the direction of patient flow through this cycle, and within each bubble an indication is given of the members of the dental team who might be included (GDP, general dental practitioner; DCP, dental care professional, including oral health educator-trained nurses, hygienists, therapists, practice managers, and reception staff).

1.2 Dental caries

1.2.1 What is it?

'A reversible (in its earliest stages) but progressive disease of the dental hard tissues, instigated by the action of bacteria upon fermentable carbohydrates in the plaque biofilm on tooth surfaces, leading to bacterially generated acid demineralization and ultimately proteolytic destruction of the organic component of the dental tissues.'

1.2.2 Terminology

Primary caries is the developing pathological biochemical process and physical lesion occurring on a previously sound tooth surface.

Root caries is primary caries on an exposed root surface (often after gingival recession has occurred), penetrating more easily into the exposed dentine. The pathological biochemical process for both primary and root caries is the same (see Figures 1.2 and 1.3).

Recurrent (secondary) caries is primary caries occurring at the margin of a failing restoration. An alternative definition of this is 'caries associated with restorations/sealants (CARS).' The aetiology is the same—metabolic activity in the stagnant plaque biofilm.

Residual caries is a term describing that portion of caries-affected, demineralized tissue retained purposely after minimally invasive cavity preparation, which is then sealed over and restored (see later).

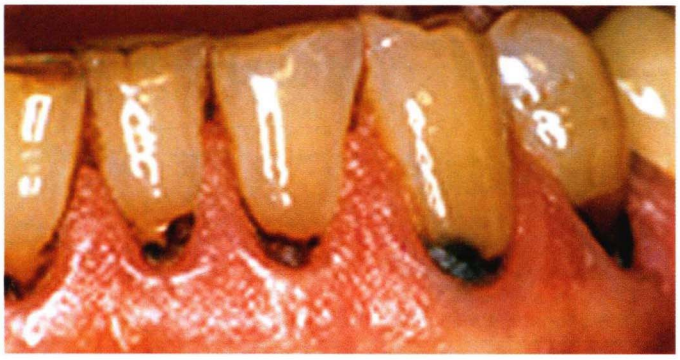

Figure 1.2 Slowly progressing root surface lesions with dark, leathery dentine surfaces and some plaque deposits. This is the mouth of a 70-year-old patient with a dry mouth (xerostomia, secondary Sjögren's syndrome) and rheumatoid arthritis, making oral hygiene difficult due to impaired toothbrush manipulation and painful mucosae.

1.2.3 Caries: the process and the lesion

The caries process

The caries process originates as metabolic activity in the plaque biofilm resident on the tooth surface. This biofilm begins to form just a few minutes after the tooth surface has been brushed, and is adsorbed initially as the *acquired pellicle* containing an admix of salivary proteins and glycoproteins. Within a short time, oral bacteria colonize the pellicle, thus forming the dental plaque biofilm, associated closely with bacterial extracellular polysaccharides and salivary proteins. The increased density of this developing biofilm, changing bacterial population, pH, and oxygen tension all combine to create a cariogenic environment on the tooth surface. This ubiquitous natural metabolic process cannot be prevented. However, disease progression can be *controlled* by the patient, with the help of the dentist and their oral healthcare team, so that a clinically visible enamel lesion never forms. The de- and remineralization metabolic processes can be modified, particularly by regular disturbance of the biofilm with a toothbrush and fluoride toothpaste. If the biofilm is partially or totally removed at regular close intervals, mineral loss may be stopped or even reversed towards mineral gain (especially in early or *incipient* lesions). The fluoride in toothpaste delays lesion progression primarily by inhibiting demineralization and encouraging remineralization processes.

The carious lesion

The carious lesion forms as a direct consequence of the metabolic activity in the biofilm on the tooth surface (i.e. the caries process). If factors tip the demineralization/remineralization balance towards demineralization (plaque, diet, salivary factors, mineral ion concentrations, and time), the histological stages of progressive lesion formation leading to cavitation can eventually be detected clinically and dealt with accordingly.

1.2.4 Aetiology of the caries process

Occurring in the plaque biofilm, the main factors that interact in the aetiology of the carious process are as follows:

- *Bacteria*: with colonization within the plaque biofilm, several hundred different species exist within a complex ecology, dependent on the age and relative stagnancy of the plaque on the tooth surface. *Streptococcus mutans*, classically thought to be the primary causative bacterial species, is now considered to have an associative role in the

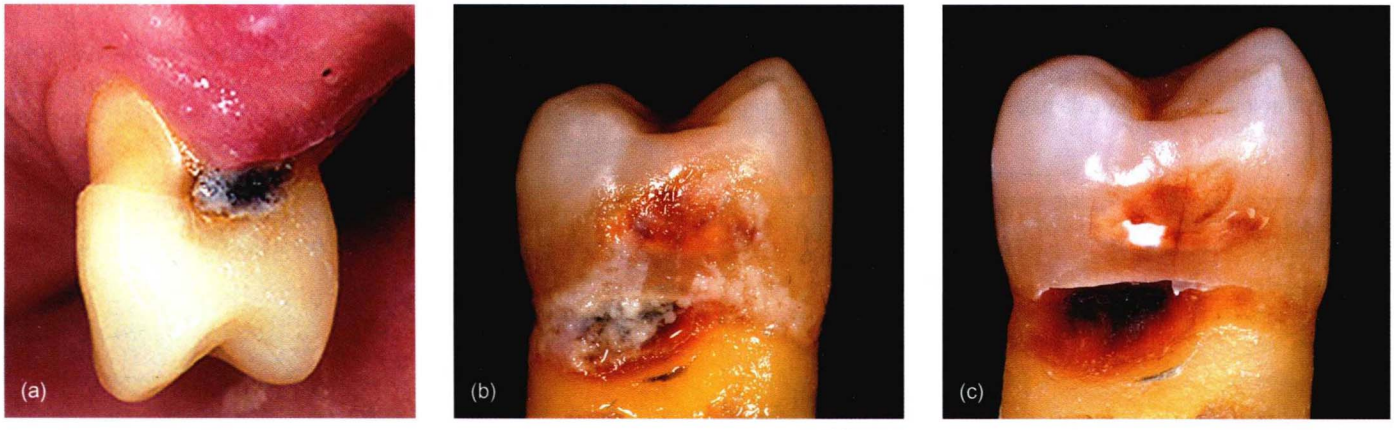

Figure 1.3 **(a)** An active root caries lesion with overlying plaque deposit in an area of stagnation alongside the margins of a partial denture. The buccal cervical abrasion cavity has been caused by excessive toothbrushing. **(b)** A stagnant plaque biofilm present on the proximal root surface, which when removed **(c)** reveals an active root caries lesion. (Images (b) and (c) courtesy of L. Mackenzie.)

Q1.1: What differences in clinical appearance are there between the coronal and root surface caries lesion in Figure 1.3 (c), and how may they relate to lesion activity?

caries process, and may act as a potential microbiological marker for caries. *Lactobacillus* and *Bifidobacterium* species have been shown to be significant in the caries process, and it is likely that species interaction within the biofilm will instigate and allow the carious lesion to progress.

- *Susceptible tooth surfaces* (see Chapter 2): carious lesions occur on tooth surfaces that have accumulated plaque, stagnating for a prolonged period of time, which may include the following:
  - The depths of pits and fissures on posterior occlusal/buccal surfaces of those teeth that the patient cannot clean effectively with a toothbrush. These areas on newly erupting molars are particularly susceptible to carious attack.
  - Proximal surfaces (mesial and distal) *cervical* to the contact points of adjacent teeth (where the patient may not floss regularly, or at all). These surfaces of particularly imbricated (crowded) teeth can be more susceptible due to the lack of access for oral hygiene aids.
  - Smooth surfaces adjacent to the gingival margin (again an area that the patient may often miss with their toothbrush), especially of those teeth that are imbricated, rotated or in-standing.
  - The ledged/overhanging/defective margins of restorations (a plaque trap created, often not evident to the patient, and inaccessible to a toothbrush or floss) (see Figure 1.4).
- *Fermentable carbohydrates*: plaque bacteria are capable of metabolizing certain dietary carbohydrates (including sucrose and glucose), producing various organic acids (lactic, acetic, and propionic acids) at the tooth surface, causing plaque pH to fall within 1–3 minutes, and initiating demineralization if the pH drops to below 5.5 (critical pH of enamel). The pH can take up to 60 minutes to climb back to normal levels, this normalization being aided by the protective buffering capacity of saliva (pH 7.0; see Figure 1.5). This demineralization/remineralization cycle occurs continuously at any tooth surface, all the time.
- *Time*: even though the drop in pH commences rapidly, sufficient time is required for the plaque biofilm to produce a *net* mineral loss equating to histological hard tissue damage at the tooth surface.

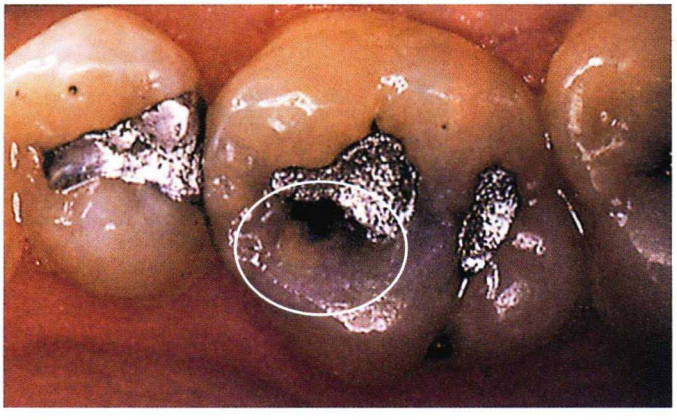

Figure 1.4 Caries at the margin of the failed dental amalgam restoration (white circle) on the occlusal surface of UL6.

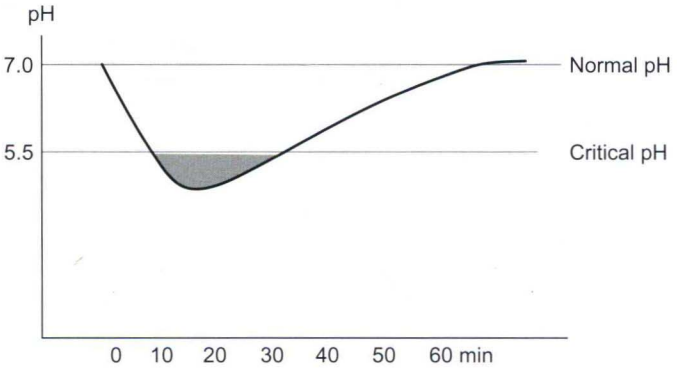

Figure 1.5 The Stephan curve, showing the changes in plaque pH over time after an oral glucose rinse at time 0 minutes. The critical pH of enamel (5.5) is that below which the hydroxyapatite crystals begin to dissociate into their constituent ions. Note that the critical pH varies, depending on an inversely proportional correlation with the concentration of available calcium and phosphate ions in the plaque biofilm fluid at the tooth surface. The grey-shaded portion of the graph indicates the 20-minute period in which the tooth surface is under threat of mineral loss. The critical pH of dentine is 6.2, which again is not fixed.

The four direct causes previously discussed can be affected or modified by several other indirect patient factors to affect ultimately the disease pattern experienced by each individual patient. These determinants include the patient's:

- income (the cost of dental care)
- knowledge about their own oral health
- attitudes to healthcare (general and oral)
- social class
- behaviour
- education.

The relative importance of these factors can be determined during verbal history taking (anamnesis) and oral examination (see Chapter 2), and helps to form the basis for determining the individual's risk and likelihood of developing caries in the future—the caries risk assessment/likelihood analysis (see Chapter 3). It is these factors in combination with the collective skills of the oral healthcare team that will help the patient to overcome or 'cure' their condition of dental caries.

1.2.5 Speed and severity of the caries process

The caries process in the normal oral environment, whose metabolic activity is tipped in favour of demineralization, will take several weeks to become detectable clinically as a lesion with signs and symptoms. This is because the overall process, with its continuously fluctuating metabolic balance at the ionic level, is relatively slow and can be moderated by oral hygiene techniques, dietary modification, and the use of fluoride or other agents. The presence of saliva, with its capacity to buffer plaque acids, provide a source of remineralizing calcium and phosphate ions to the tooth/lesion surface, remove food debris, and lubricate/protect tooth surfaces, also helps.

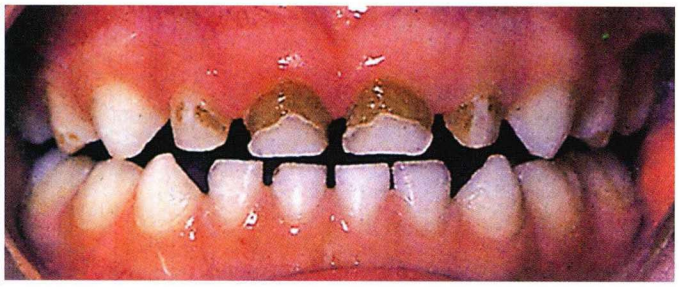

Figure 1.6 Early childhood caries affecting deciduous anterior teeth.

Q1.2: What habit(s) may have contributed to this pattern of disease?

'Rampant' caries

However, clinical scenarios exist where the process is accelerated and many biochemically active lesions form rapidly, often involving surfaces of teeth ordinarily expected to be caries-free—described classically as 'rampant' caries. This condition affects the primary dentition, where it is now more appropriately termed *early childhood caries* (see Figure 1.5), teenagers, or young adults with a highly cariogenic diet (frequent sugar episodes; see Figure 1.6) and/or addicted to recreational drugs, or adult patients with a dry mouth (xerostomia) (see Figure 1.7). Radiation to the region of the salivary glands, used in the treatment of an orofacial malignant growth, and Sjögren's syndrome, an autoimmune condition that may involve the salivary glands, are the most common causes of severe xerostomia. In addition, a large number of therapeutic drugs, such as antidepressants, tranquillizers, antihypertensive drugs, and diuretics can retard salivary flow and affect its quality, especially when taken together (polypharmacy).

Arrested caries

In distinct contrast to rampant caries, the term *arrested caries* describes those lesions that have stopped progressing and are inactive metabolically. It is observed when factors in the oral environment have changed from conditions predisposing to caries to conditions that tend to slow, or even reverse, lesion progression. These 'arrested' lesions often have a dark, hard, shiny exposed dentine surface (see Figure 1.8 and Table 1.1).

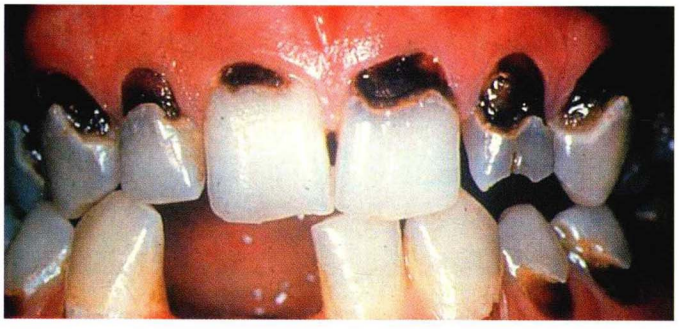

Figure 1.7 Rampant caries in an adult patient with cavities affecting sites not normally associated with caries due to their accessibility for adequate oral hygiene.

Q1.3: What aetiological factors may have contributed to this pattern of disease?

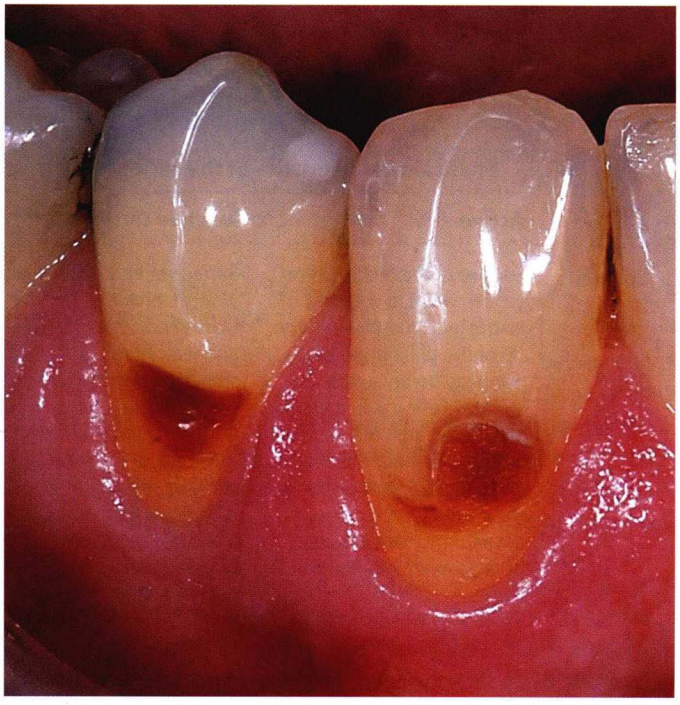

Figure 1.8 A hard, shiny, and stained arrested root surface lesion is present on the buccal cervical aspect of the LR4. However, the lesion on the LR3 has a matte, stippled surface appearance indicative of lesion activity. (Courtesy of L. Mackenzie.)

Q1.4: Why might the lesion on the LR3 appear active whereas the adjacent lesion on the LR4 in the same patient appears arrested?

1.2.6 The carious lesion

Having summarized the caries process as an ongoing metabolic demineralization/remineralization balance occurring at the interface between the plaque biofilm and the tooth surface, it is important to understand that the resulting carious lesion is a progressive alteration and destruction of the hard tissues (mineral and organic matrix) from the enamel surface through to the pulp. While the lesion is still within enamel, it can be arrested and possibly reversed with net mineral gain in its earliest stages. Once it is into dentine, the process can be made inactive (arrested), but if proteolytic destruction of the organic collagen matrix has occurred extensively, this cannot be reversed as observed histologically. This section will take the reader through key features of the histological and clinical development of a lesion from its earliest enamel stages through to cavitation into the pulp.

An understanding of the basic histological features of healthy enamel and dentine is an essential prerequisite for appreciating the changes

Table 1.1 Summary of differences in physical characteristics between active and inactive (arrested) carious lesions in enamel or dentine

	Physical characteristics of the carious lesion	
	Active	**Inactive (arrested)**
Enamel	Surface of enamel is white/yellow; opaque with loss of lustre; feels rough when the tip of the ball-ended probe is moved gently across the surface. Lesion is in a plaque stagnation area (i.e. pits and fissures), or near the gingival and proximal surface below the contact point. Lesion covered by plaque biofilm prior to examination (WSL)	Surface of enamel is whitish, brownish, or black. Enamel is shiny and feels hard and smooth when the tip of the ball-ended probe is moved gently across the surface. For smooth surfaces, the lesion is typically located at some distance from the gingival margin. Lesion not covered by plaque prior to examination (BSL)
Dentine	Dentine appears moist and matte; feels rough, soft, and wet or leathery on gentle probing	Dentine appears shiny and hard, and is scratchy on gentle probing

WSL, white spot lesion; BSL, brown spot lesion.

that occur within the lesion, and an outline of these is presented in Table 7.1 in Chapter 7. Further information can be obtained from the suggested further reading at the end of this chapter. The relationship between lesion histology and clinical appearance has been used in a caries detection and assessment system which is outlined and discussed in Chapter 2 (see Table 2.3).

Within enamel

Plaque-acid demineralization causes porosities to form within the prism structure, initially beneath the outer surface of enamel: this is termed *subsurface demineralization*. The developing pore volumes through the depth of the enamel lesion, caused by a longer exposure to reduced pH, have been measured using polarized light microscopy (outermost surface zone (< 1% pore volume), body (5–25%), dark (2–4%), and innermost translucent zone (1%); see Figure 1.9).

The existence of the enamel lesion surface zone may be due to increased extrinsic fluoride ion deposition in this area, or as a consequence of remineralization metabolism of the biofilm on the tooth surface. It is essential that this intact surface is not cavitated iatrogenically (i.e. a hole created by a dentist/therapist sticking a sharp dental probe/explorer into the lesion surface; see Chapter 2, Figure 2.7), as it still has the potential to heal if the biofilm can be regularly and effectively removed by the patient, and remineralizing solutions and/or toothpastes containing higher concentrations of calcium and phosphate ions used periodically (see Chapter 4, Section 4.2.3).

Histologically, smooth surface lesions have a cross-sectional shape of an inverted cone (widest superficially, with the apex towards the enamel–dentine junction (EDJ); see Figure 1.10). Fissure lesions can be considered to take the form of two adjacent smooth surface lesions (see Figure 1.11).

Figure 1.9 Longitudinal ground section through a carious lesion on a smooth surface (polarized light and water; E, enamel; D, dentine). The enamel lesion is shaped as an inverted cone, widest at the tooth surface, narrowing towards the enamel–dentine junction, with a relatively intact surface zone (SZ).

Q1.5: What ions have contributed to the creation of the intact surface zone and where have they come from?

- *Clinical manifestations* (see Table 1.1): the active white spot lesion (WSL) is initially smooth, frosty white/opaque, and non-cavitated

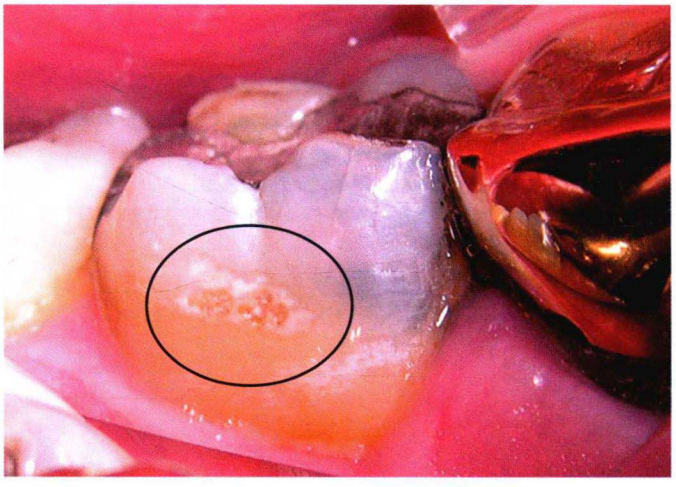

Figure 1.10 Active white spot enamel lesion on the mid-buccal of LL7 (circled). This more developed lesion has a rough surface, acting as a plaque trap.

Q1.6: What features of the lesion in Figure 1.10 will help the dentist to conclude that it is active, how might these be detected, and how might the patient be managed?

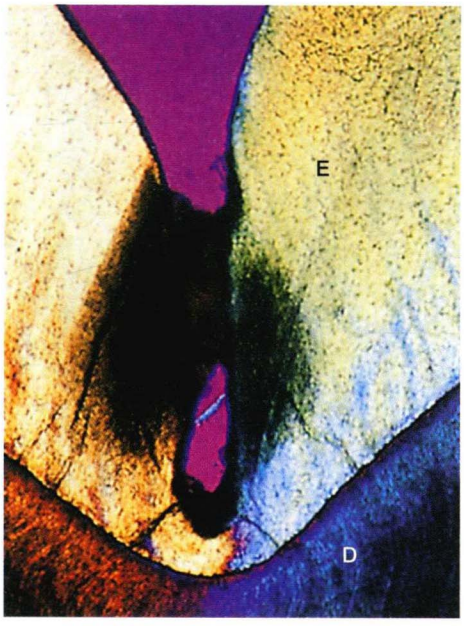

Figure 1.11 A longitudinal ground section (polarized light with water) through an occlusal fissure showing an enamel lesion forming on the two adjacent walls of the fissure (dark regions; E, enamel; D, dentine).

Q1.7: How might this scenario be managed in a high caries risk patient?

clinically (see Figure 1.12). This can be detected more easily if the tooth surface is air-dried for a few seconds using a 3-1 air/water syringe. As the lesion develops over time, it becomes somewhat chalky, eventually becoming roughened or micro-cavitated (roughness can be detected by gently running a rounded/ball-ended probe *across* the lesion surface). This surface irregularity can encourage further plaque deposition (see Figure 1.10). There are no symptoms at this stage, but reactions in the dentine–pulp complex may be mediated by cytokines and bacterial breakdown products within the dentine matrix and tubules (see later).

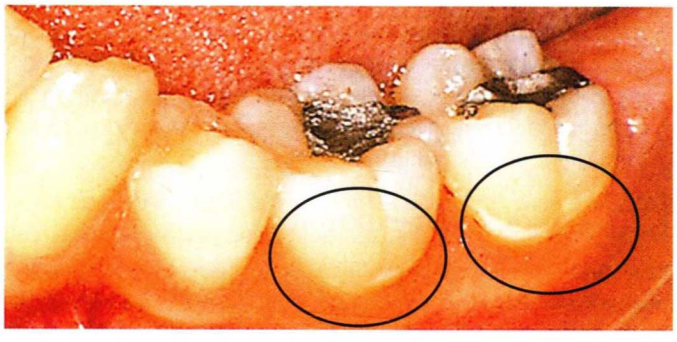

Figure 1.12 Early white spot enamel lesions on the cervical-gingival margins of both mandibular left molars (circled).

- If plaque is removed, these lesions can arrest and porosities can be eliminated by abrasive toothbrushing/tooth wear, resulting in the hard smooth shiny surfaces of arrested lesions. Porosities may also be filled with deposited mineral and dietary molecules causing staining (e.g. tannins), which may be trapped within the mineral lattice. This process creates an arrested brown spot lesion (BSL) with a hard shiny overlying smooth surface (see Figure 1.13b).

The lesion at the EDJ or the amelo–dentinal junction (ADJ)

Histologically, the caries process may reach dentine before clinical cavitation is detectable (a closed lesion: mICDAS score 2; see Chapter 2). Defence reactions in the dentine–pulp complex are stimulated at this stage, with evidence of translucent dentine at the advancing lesion boundary and tertiary dentine deposition at the dentine–pulp interface beneath the advancing lesion (see later). Again, symptoms are unlikely at this stage of lesion development.

The lesion extends in dentine, immediately subjacent to the EDJ (see Figure 1.13a), its lateral extension coinciding with the spread of the overlying enamel lesion at the surface of the tooth, which in turn is dependent on the extent of the resident plaque biofilm at the tooth surface. Relative hypomineralization of this histological mantle dentine zone, greater side-branching of dentine tubules, or defects within the enamel/dentine interface may also contribute to this spread laterally.

The lesion may also penetrate along the dentine tubules towards the pulp.

Within dentine

Once the lesion has spread histologically (and radiographically) further towards the middle third of dentine, it is often cavitated (open) clinically on both occlusal and smooth surfaces, with plaque now able to accumulate on the exposed dentine surface. The further spread of the lesion will undermine the overlying enamel, creating an associated visible grey shadow/opacity, which becomes brittle and prone to fracture under occlusal loading. This undermined and unsupported enamel may need to be removed during cavity preparation if the damage requires operative repair (see Chapter 5, Section 5.9.3).

The patient may begin to experience initial symptoms of acute pulpitis—a poorly localized sharp pain of a few seconds' duration stimulated by hot, cold, or sweet stimuli (see Chapter 3). The histological components of carious dentine to be considered are the mineral, collagen, bacterial penetration, and tubule structure. Both degenerative and reparative processes act on these simultaneously in different parts of the lesion. The histological changes of the carious dentine biomass through its depth (from EDJ to pulp) are described in the following bullet points, but note that these descriptive zones are not separate biological entities, but blend into one another without clear detectable boundaries (see Figure 1.14).

- *Caries-infected dentine* (zone 1 in Figure 1.14): the outermost, superficial, irreparable, necrotic zone of destruction, often distinguished clinically as a dark brown, soft, wet, 'mushy' layer.
  - Mineral component has dissociated extensively due to bacterial acid attack.

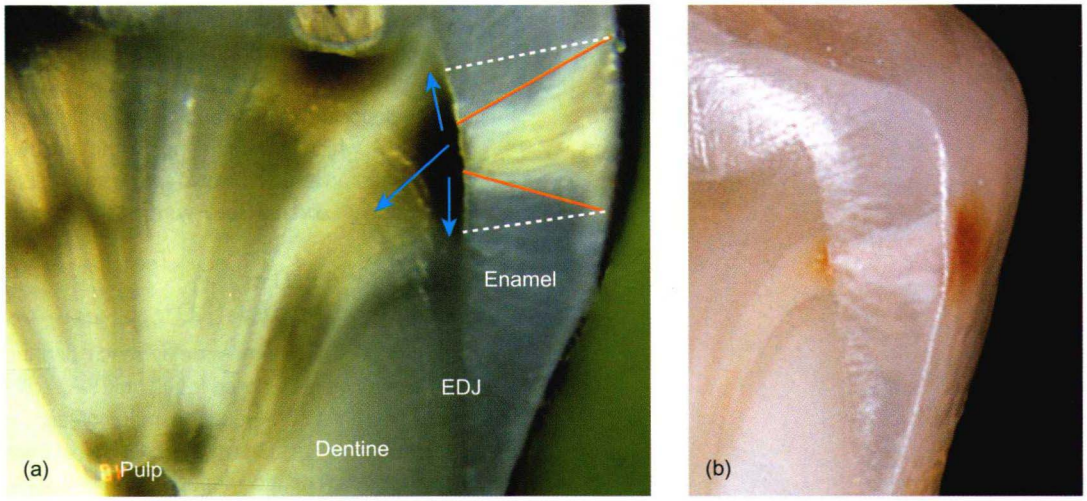

Figure 1.13 **(a)** A mesio-distal section through a carious tooth highlighting a proximal lesion. The red lines outline the 'inverted cone' cross-sectional histological shape of the enamel lesion, and the blue arrows indicate the direction of spread of the lesion having crossed the enamel–dentine junction (EDJ) into dentine. The white dotted lines show how the extent of the spread of the dentine lesion subjacent to the EDJ is associated with the same lateral extent of the enamel lesion on the tooth surface, both governed by the presence of the plaque biofilm at the tooth surface. **(b)** The surface lesion shown is an arrested brown spot lesion, its boundaries clearly aligned with the inverted cone of the enamel lesion beneath. (Figure 1.13 (b) courtesy of L. Mackenzie.)

Q1.8: How will the patient have arrested this once active lesion?

- Collagenous matrix has been denatured (irreparably damaged) by proteolytic enzymes intrinsic to the dentine itself (the zinc-dependent, acid-activated matrix metalloproteinases (MMPs)), as well as from bacteria, activated by those bacterial acids produced during the caries process.
- Bacterial load in this zone is very high.
- Dentine tubule structure is destroyed.

This zone of 'necrotic' dentine should be clinically removed when preparing a cavity, as it cannot be repaired histologically and it provides a poor-quality bonding substrate for adhesive materials to achieve an adequate seal, and inadequate physical restoration support.

- *Caries-affected dentine* (zones 2, 3, and 4 combined together; see Figure 1.14): the inner layer of carious dentine which can be repaired by the dentine–pulp complex, often distinguished as paler brown, harder, 'sticky and scratchy', or slightly leathery dentine (elicited by a sharp dental probe, which should not be used directly over the pulpal floor of a deep cavity).
  - Mineral dissolution still occurs, but to a lesser extent than in infected dentine, as the pH gradually rises towards the advancing front of the lesion and the ionic equilibrium begins to balance.
  - Collagen is still damaged by proteolysis, but to a lesser extent, so permitting dentine repair/mineral gain, as the proteinaceous scaffold for mineral crystal deposition now persists.
  - Bacterial load lessens, but there are still more anaerobic bacteria present.
  - Dentine tubule structure returns gradually within the depths of this zone.

The deepest layer of caries-affected dentine (zone 4 in Figure 1.14) can be described as hypermineralized translucent dentine (due to its glassy appearance in histological cross-section), one of several reparative reactions of the dentine–pulp complex to the caries process (see later). As can be seen in Figures 1.13 and 1.14, the lesion in dentine often has a dark brown discoloration within the caries-infected zone which then pales gradually through the depth of the lesion, towards the pulp. The aetiology of the colour changes is not clear, but a biochemical reaction between proteins and carbohydrates in a moist acidic biological environment, the *Maillard reaction*, may play a part. Not all lesions are uniformly dark brown; some rapidly advancing lesions may have a pale discoloration within the caries-infected zone, and there is no direct link between the colour of dentine and the bacteria present within these zones.

1.2.7 Carious pulp exposure

If the carious process cannot be modified by preventive or controlling measures and the lesion is not treated operatively in time with appropriate minimally invasive excavation techniques and a sealed adhesive restoration within the dentine, then the advancing front of the lesion will approach the dentine–pulp boundary. By this point, bacteria/toxins