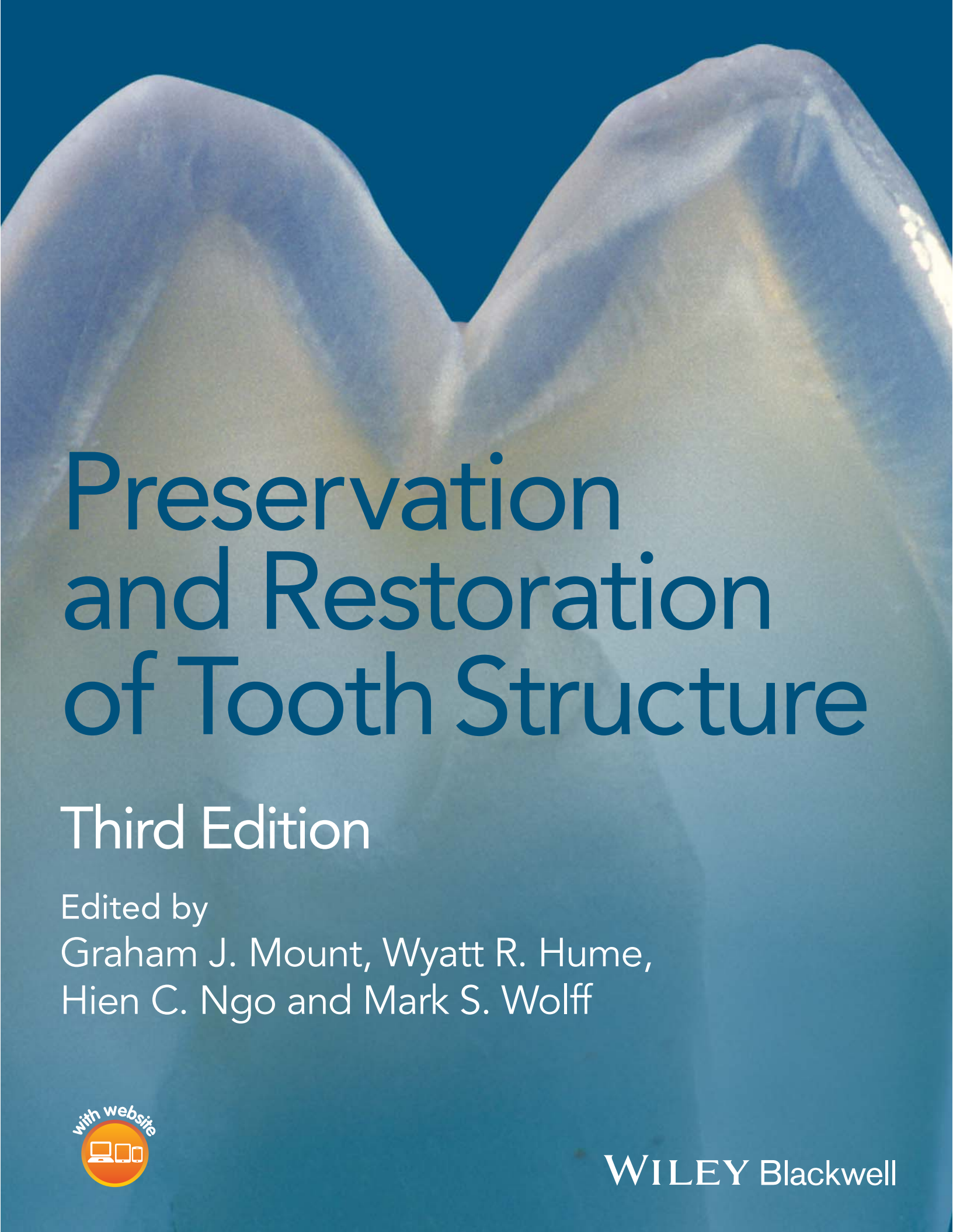

Preservation and Restoration of Tooth Structure

Third Edition

Edited by
Graham J. Mount, Wyatt R. Hume,
Hien C. Ngo and Mark S. Wolff

with website

WILEY Blackwell

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Acknowledgments

We are pleased to have worked with excellent contributors throughout the three editions of this text, and we express our profound gratitude to all who have so willingly participated.

In the first edition we focused on Australia as our primary source of input because the two of us who were editors at that time were based there, and we were pleased to be part of the rapid advances in the concepts and quality of dental care that were occurring in that country. Increasingly, as the first and second editions gained very welcome and gratifying acceptance in other parts of the world, we sought input more broadly, adding two contributors from Singapore in the second edition. In this edition we have a co-editor from the United States, and for the first time contributors from both the USA and the United Kingdom.

This edition builds on the previous two. Many of our earlier contributors have stayed with us as we have revised and updated the text and supporting material. Some have retired, in all cases leaving with us the benefits of their earlier contributions. We express special gratitude to those individuals for their generosity of spirit and hope that they will continue to take pride in the outcome.

As was the case with the previous editions, royalties from sales after expenses will be donated to support dental research. It is dental research that has enabled the improvements in the quality of care that this book celebrates, and with our contributors we are pleased to be able to support the future of our profession and the well-being of the patients that we care for in this way.

We acknowledge in the text those figures and tables that have previously been published elsewhere, and wish to note here our gratitude to the publishers and authors of that material for granting their permission for its continued use.

Some individuals deserve recognition by name. Michael Williams made enormous contributions to the structure and style of the first and second editions, and continued to provide advice for the third. Rob Watts, of Knowledge Books and Software, the publisher of the second edition, gave us new ways of thinking about presentation of our material and also displayed great generosity as we moved on to Wiley Blackwell for this, the third edition. The editorial team at Wiley Blackwell has also been superb.

Our wives and partners have been extremely tolerant of our absences and obsessions, and to them we express special thanks.

About the companion website

This book is accompanied by a companion website:

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The website includes:

- Interactive Multiple-Choice Questions for each chapter
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1 The password for the figures on the book companion website is the first word of the figure legend for Figure 4.8. Please use all lowercase.
2 Go to **www.wiley.com/go/mount/preservation** to enter this password.

Introduction

Since the profound changes in diet that have occurred through the development of agriculture and then urbanized food manufacture and distribution, humans have become subject to dental caries and other diseases that cause damage to tooth structure. Left untreated, caries, in particular, can have devastating consequences for the individual. We are not well adapted to dealing with these problems alone.

It is now 100 years since the work of G.V. Black allowed serious consideration of restorations as an ethical alternative to tooth extraction as a treatment for caries. Despite Black's excellent work and teaching, for the first half of that century, restorations tended to have a short life span. They were large initially, and when they failed and were replaced, they became even larger. The end point was, most often, tooth extraction. Restorations could delay that event, but it was clear that they were not an effective or enduring treatment for the disease.

During the last half-century there has been a profound change. This book focuses on the nature of that change, and its continuing progression. First, the beneficial effects of fluoride in preventing and treating caries became evident through systematic, scientific research. Then, again through scientific research, an understanding developed of the balance between demineralization and remineralization of dental hard tissues. This represented a fundamental change in the understanding of the nature both of caries and the diseases related to corrosion. Together with some advances in dental materials science, in particular, the development, through research, of adhesive restorative materials, these things gave rise to a new concept of both prevention and treatment of defects in tooth structure.

The diseases that cause damage to tooth structure are now sufficiently well understood that, with the knowledge and understanding of the patient, they can be prevented and, when they do occur, can be treated predictably and effectively. Restorations, when they are needed, can be small, strong and enduring. Dental professionals can now primarily be physicians, preventing, diagnosing and treating the diseases non-surgically. When surgical interventions are required to provide the necessary care, they can be minimally invasive, if performed with precision and attention to detail. Extraction need no longer be contemplated as part of the treatment of caries. Neither should repeated cycles of tooth restoration be necessary or expected.

Science continues to advance, and the dental profession's ethical standards and effectiveness continue to evolve and improve with it. We hope that this book will contribute to that process.

Graham J. Mount
Wyatt R. Hume
Hien C. Ngo
Mark S. Wolff

1 The Oral Environment and the Main Causes of Tooth Structure Loss

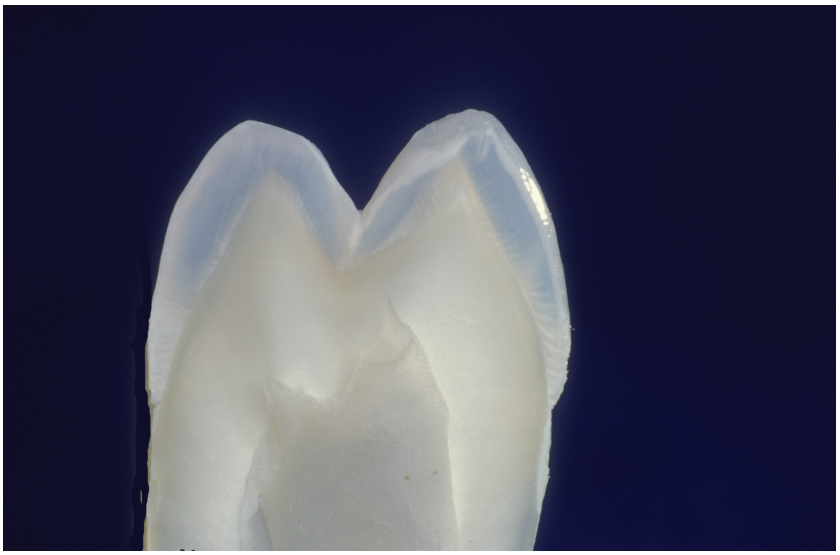
J. Kaidonis, G.C. Townsend, J. McIntyre, L.C. Richards & W.R. Hume

The human oral environment evolved within a Paleolithic (Stone Age) hunter-gatherer setting, reflecting well over 2 million years of evolution from primate origins of even greater antiquity. The interplay between genes and differing environments over many tens of thousands of generations resulted in various oral adaptations that together provided good oral health and function.

Common themes were evident among Paleolithic groups. First, dentitions showed extensive wear when compared to many modern societies, yet the teeth remained functional. Second, although the diet was generally acidic, erosion and the non-carious cervical lesions that are endemic in current populations were not apparent. Third, oral bacterial biofilms were present, yet the prevalence of dental caries and periodontal disease was so low that it could be considered insignificant.

With the advent of farming in human societies about 10,000 years ago and increasingly over the last 400 years, as food manufacture and distribution became more common, changes in our diet have led to profound changes in the oral ecosystem. These changes were sudden from an evolutionary perspective and they have been primarily responsible for the modern oral and many of the systemic diseases now afflicting human populations.

Among those diseases are several that lead to the loss of tooth structure.



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The Human Oral Environment in Health

The sialo-microbial-dental complex in a state of balance

The human mouth is centrally involved in the first stages of digestion, as is the case in all species with alimentary canals. The need to chew and swallow a variety of foods in order to survive in different environments caused the evolution of oral and dental tissues, salivary secretions and a unique oral microbial ecology. These elements together can be referred to as the sialo-microbial-dental complex. 'Sialo' means to do with saliva. All elements were, until recent centuries, in relative harmony or balance in the great majority of individuals.

Tooth structure, diet, the oral microbial mix, saliva and biofilms[1] (dental plaque) are closely associated and interrelated physically, functionally and chemically. Although each is described separately in the sections below, the components have evolved together and function as an integrated system. All contribute to health, and under some circumstances all contribute to disease.

Tooth Structure

Enamel

Dental enamel, the strongest substance in the human body, is a highly mineralized tissue with a well-defined structure. It is formed by the precipitation of crystals of apatite, a compound comprised of calcium, phosphate and other elements, into an extracellular protein matrix secreted by specialized cells called ameloblasts.

The precipitation of apatite is called 'calcification' or 'mineralization'. It commences before the emergence of the tooth in the mouth. The protein matrix dissolves as the crystals grow, leaving a tissue comprised almost entirely of apatite crystals in a unique physical arrangement.

Of central importance to the understanding of the chemical dynamics relating to the inorganic components of tooth structure is the fact that ions can move in and out of apatite crystal surfaces, that is, the crystals can shrink, grow or change in chemical composition, depending on local ionic conditions.

Tooth shape is determined by ameloblast proliferation

Ameloblasts are derived from epithelial cells of the embryonic mouth. In a process that is closely controlled genetically, they proliferate into underlying tissue to create the enamel organ, which has a shape or form unique to each tooth crown, defining the shape of the future dentino-enamel junction.

The underlying tissue, which becomes the dental papilla, is derived from ecto-mesenchymal neural crest cells that have previously migrated into the region of the developing oral cavity. These mesenchymal tissues give rise to all of the other dental tissues: dentine, pulp, cementum and to the adjacent periodontium.

Differentiation of ameloblasts and odontoblasts, which are mesenchymally derived, results from a series of reciprocal interactions between the adjacent cells of the enamel organ and those of the dental papilla, mediated by various signalling molecules and growth factors among and between the two groups of cells.

Calcification and the formation of enamel prisms

Stimulated by the deposition of pre-dentine by adjacent odontoblasts, ameloblasts secrete an extracellular matrix of protein gel made up of amelogenins and enamelins. The local ionic environment of the extracellular fluid is supersaturated with calcium and phosphate, and the matrix proteins form an appropriate lattice for the precipitation and growth of apatite crystals.

Also sometimes called hydroxyapatite (HA), apatite is a molecule with the basic structure $Ca_{10}(PO_4)_6(OH)_2$ highly substituted with other ions, most notably carbonate in place of some phosphate ions, and fluoride in place of some hydroxyl ions. Strontium, sodium, zinc and magnesium may also be present in place of some calcium ions, usually in trace amounts.

Highly carbonated apatite is much more soluble than apatite with low carbonate, a significant factor in post-eruption enamel maturation as carbonate is replaced with phosphate from saliva by ion exchange. The degree of solubility of apatite also varies with the level of substitution between hydroxyl and fluoride ions. Extensive research has demonstrated that optimal levels of fluoride within apatite, most probably achieved also through ion exchange once in the oral environment rather than incorporation during initial formation, lead to the creation of enamel surfaces of very low solubility.

The apatite crystals coalesce within the gel matrix, their orientation being aligned to a strong degree, very probably because of orientation of the matrix protein molecules. The proteins dissolve as the crystals grow, creating a tissue comprised largely of apatite crystals.

As crystal growth continues, the ameloblasts continue to secrete matrix proteins and move away from the developing hard tissue, leaving long, generally parallel apatite crystals in arrays that form enamel rods, or prisms, with a relatively enamelin-rich boundary layer between rods corresponding to the interface between individual ameloblasts in the secreting layer. There is a change in the crystal orientation near the rod boundaries, with individual rods being connected by varying amounts of inter-rod crystallites (Figures 1.1 and 1.2).

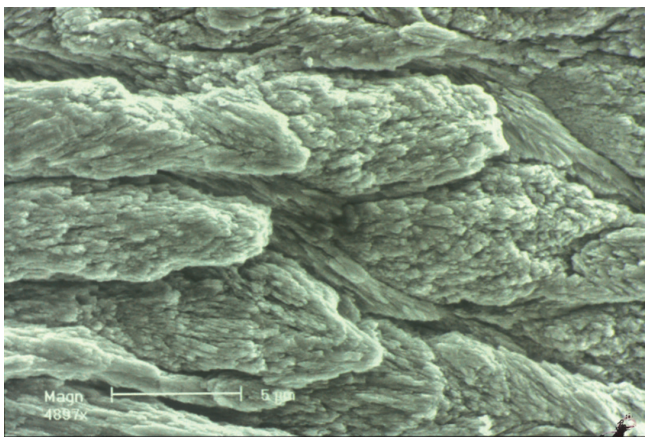

Figure 1.1 The surface of a specimen of fractured enamel showing the enamel rods, which consist of bundles of enamel crystals. The rods lie relatively parallel with each other so there is a distinct 'grain' along which fracture of the enamel is likely to occur. Note also the spaces between the rods that will be filled with ultrafiltrate in life. Magnification x 4,800. Courtesy of Professor Hien Ngo.

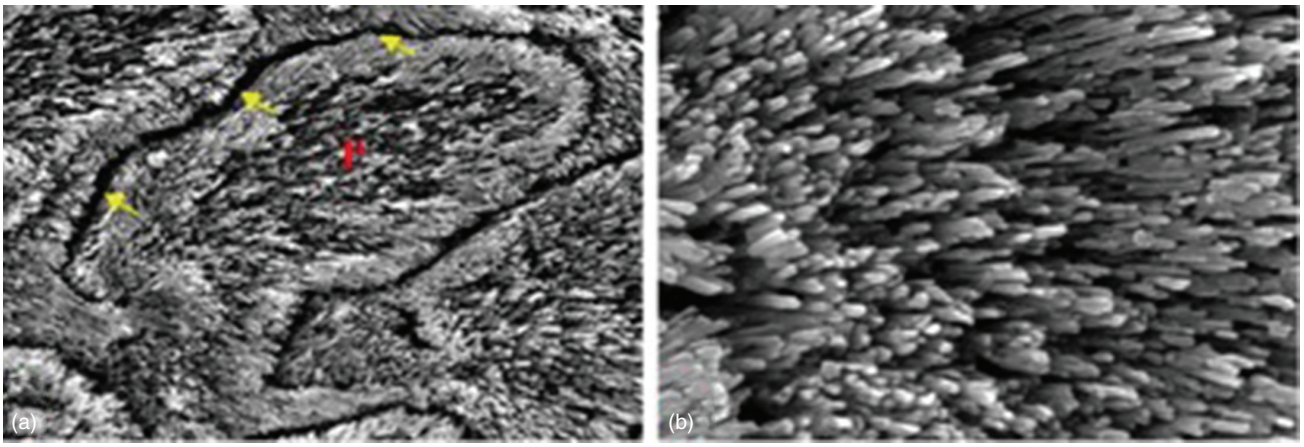

Figure 1.2 (a) Scanning electron micrograph of enamel prisms showing the interprismatic space (yellow arrows) with a periphery of densely packed crystals in parallel arrays. Courtesy of Professor Hien Ngo. (b) Higher magnification scanning electron micrograph showing the space between the apatite crystals, which in life is occupied by an aqueous organic gel matrix, and their orientation. Courtesy of Professor Hien Ngo.

By the time permanent teeth erupt, the enamel is normally 96–98% carbonated HA by weight, and about 85% by volume. The remainder of the enamel, consisting of about 3% protein and lipid and about 12% water by volume, exists in laminar pores between the enamel crystals (see Figures 1.2 (a) and 1.2 (b)).

Each enamel crystal exists within a local hydration shell, and is in ionic equilibrium with it. It is within this local aqueous phase, which itself is in ionic balance with its adjacent environment, that the dynamics of post-eruption maturation will take place, and it is within this phase of enamel, influenced by the adjacent biofilm, that post-eruption demineralization and remineralization may occur under appropriate conditions of local pH.

Filtered tissue fluid moves very slowly outward through enamel in vital, erupted teeth because the pressure inside the tooth is higher than that outside. This tissue fluid is called ultrafiltrate and it contains no protein, only water and inorganic ions. Ultrafiltrate does not appear to be a significant factor in local ion dynamics near the enamel surface, very probably because of its very slow rate of outward flow.

Macroscopic structural elements affecting appearance and strength

Enamel is formed in an incremental manner and fine cross-striations may be seen within the prisms, representing daily increments of matrix production. Larger striations, the striae of Retzius, probably reflect a 7–10-day rhythm in the rate of matrix production and mineralization. Where the striae of Retzius reach the surface, mainly in the cervical region, they can produce distinct grooves or depressions referred to as enamel perikymata. These run circumferentially around the crown giving it a slightly rough surface texture and disperse reflected light in a way that gives the tooth its characteristic appearance.

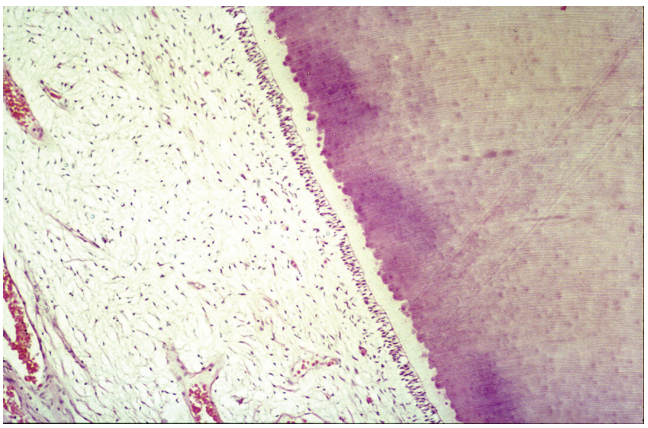

Figure 1.3 Histology of dentine. Low power view of dentine showing, from right to left, dentine, pre-dentine, odontoblasts and dental pulp. Magnification x 100.

The orientation of the enamel rods give the human enamel a physical structure, or 'grain' (Figure 1.3). This makes the enamel 'anisotropic', meaning that its physical properties differ depending on orientation. The enamel rods in the regions of cusp tips and incisal edges are often arranged more irregularly. They are referred to as gnarled enamel and it is believed that this twisting increases strength. The innermost and the outermost layers of enamel are more homogeneous and are the most highly mineralized regions of the enamel cap. The enamel in these regions is referred to as 'prismless' or 'amorphous'.

Modifications to calcification

During enamel formation, the rate of dissolution of the matrix protein seems to be temperature-dependent, because episodes of high body temperature, or fever, during enamel formation can lead to defects in enamel structure. The rate

of dissolution may also respond to the levels of fluoride in the hydroxyapatite crystals, with very high levels of fluoride causing defects in enamel mineralization, or mottling. As noted above, at optimal levels, fluoride is associated with low enamel solubility.

Thickness of enamel and the effect on colour

The thickness of enamel varies in different parts of the crown, being thickest at the cusps and incisal edges and thinnest in the cervical region. The natural colour of the enamel is moderately translucent white or whitish-blue. This colour shows in the incisal region of teeth and the cusp tips where there is no underlying dentine. As the enamel becomes thinner, the colour of the dentine shows through and the enamel appears to be more yellow. The degree of mineralization of the enamel also influences its appearance; hypo-mineralized areas appear more opaque than normally well-mineralized regions, which are relatively translucent.

Dentine

Dentine forms concurrently with enamel through the mineralization of a quite different matrix protein, collagen, which is secreted by mesenchymally-derived odontoblasts. Although similar to bone in terms of its main constituent elements, dentine has a unique structure consisting of dentinal tubules that radiate out from the pulp and which are surrounded by mineralized collagen. The tubules are formed because the odontoblasts, in addition to secreting matrix, leave a thin, cellular process, the odontoblastic process, behind as they withdraw from the advancing front of secreted matrix that then mineralizes. This structure, and in particular the dentinal tubules which contain tissue fluid and for some of their length cell processes, give dentine many properties that are very relevant to dental health and disease and to the restoration of defects.

The dentino-enamel junction

The junction between dentine and enamel, the dentino-enamel junction, is not a flat plane but is 'scalloped', especially in those areas subject to high occlusal stress. Dentine physically supports the overlying enamel and shows some degree of flexibility, which may help to prevent fracture of the highly mineralized and relatively brittle enamel.

Dentinal structure

Collectively, dentine is about 70% by weight inorganic material and about 20% organic. The inorganic material is composed of crystals of apatite, in a similar association with collagen as occurs in bone, but with a different physical arrangement that is determined by the presence of dentinal tubules. The perimeter of each tubule, the peri-tubular dentine, is more mineralized than the inter-tubular dentine, and neither is as highly mineralized as enamel. Unlike bone, dentine contains no blood vessels, nor does it normally contain the equivalent of osteoclasts. It is in all senses a vital and biologically reactive tissue because of its intimate association with odontoblasts and their cellular processes within tubules, and therefore with the adjacent dental pulp, but it does not undergo cellular remodelling as bone does.

Anatomy of dentine tubules and their continuing maturation

The non-calcified tubule, created by the presence of the odontoblastic process during and after matrix deposition and subsequent calcification, extends from the odontoblastic cell body on the outer surface of the pulp chamber to the dentino-enamel junction. When the dentine is completely formed, this can be 5 mm or more in length (Figures 1.3 and 1.4). Dentinal tubules have unique characteristics. They are tapered, with the diameter reducing by about half as it approaches the enamel. In adult dentine, the odontoblastic cell process may only occupy the inner one-third to one-half of the tubule but the entire tubule can remain patent. The non-protoplasmic portion of the tubule is filled with tissue fluid.

With advancing age, peritibular dentine continues slowly to calcify, and the internal diameter of each tubule progressively decreases. The ions that precipitate to form this additional mineral come from the pulpal extracellular fluid, which is supersaturated relative to apatite. This progressive mineralization throughout life leads to a tissue that is denser, less permeable and less flexible. Each of these changes is relevant to decisions in the restoration of structural defects, as will be described in Chapter 15.

There are also protoplasmic connections between odontoblastic processes, and therefore connections between adjacent tubules by what are termed lateral tubules in a quite complex arrangement (Figure 1.5).

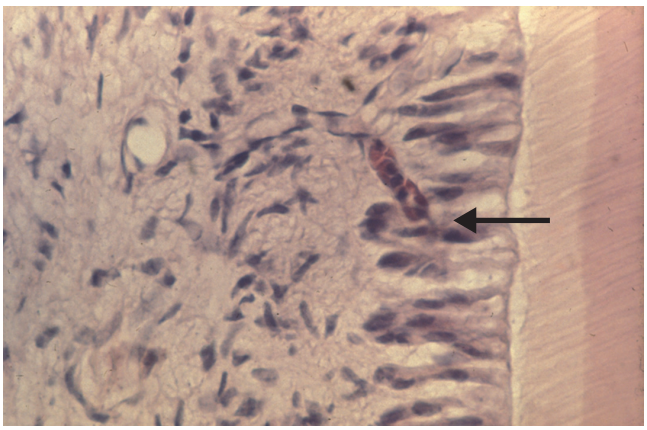

Figure 1.4 Histology of dentine. A higher power view of the odontoblast region. Magnification x 400.

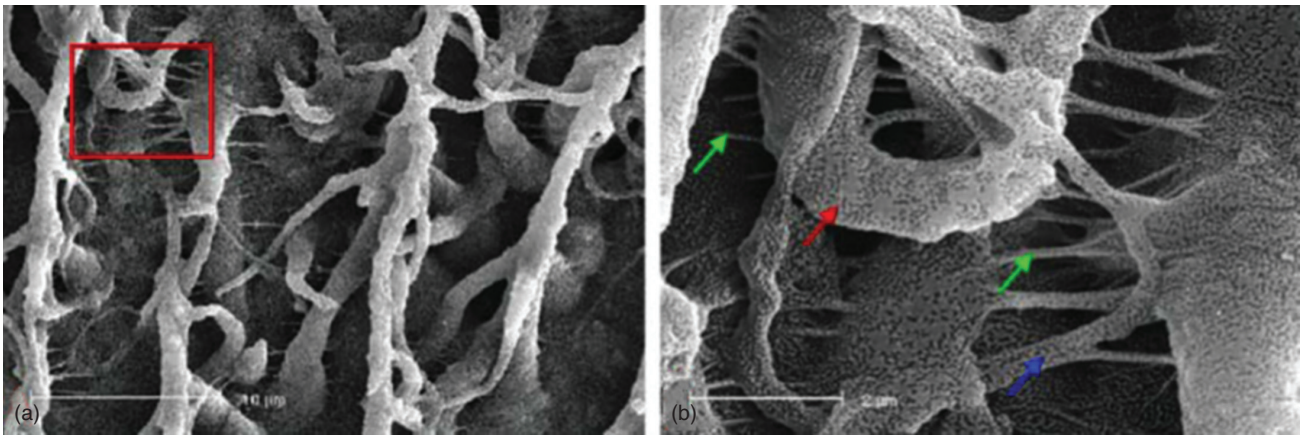

Figure 1.5 (a) Scanning electron micrograph showing the primary dentine tubules as well as the lateral tubule network. Courtesy of Professor Hien Ngo. (b) Higher magnification of the selected area in Figure 1.5 (a), showing the lateral branches (red arrow) extending from the major tubules. This network was further divided into sub-micron (blue arrow) and even finer divisions at the nanometer level (green arrow). Courtesy of Professor Hien Ngo.

Dentinal fluid

The odontoblastic tubules are full of fluid, some intracellular (i.e. within the odontoblastic processes) and some extracellular. The extracellular fluid moves outward due to the pressure gradient between the extracellular fluid of the pulp and the inside of the mouth. In the normal erupted tooth, the movement is slow because of the very limited permeability of enamel, but if the enamel is missing, as occurs in some advanced defects and often during restoration of defects, fluid flow is much more rapid. Dentinal wetness depends primarily on the size and number of the tubules, so dentine is wetter closer to the pulp where the tubules are larger in diameter and more closely packed. Dentinal wetness may be a relevant factor in many tooth restorative procedures.

Chemicals can diffuse through the dentine tubules just as they can through any water-based medium. The rate and amount of diffusion are dependent on the concentration gradient, the molecular size of the solute, the temperature, the thickness of the dentine, the diameter and number of tubules, and whether or not the tubules are partially or completely blocked. The natural wetness of dentine, the tubule structure and factors that occlude tubules are important factors to be considered when replacing missing tooth tissue. As noted above, dentine becomes less wet with age, because tubule diameter tends to decrease. If the pulp dies, the dentine remains wet but outward flow is likely to be considerably reduced.

Dentinal sensibility

The movement of fluid within dentinal tubules causes pain. Such movement can occur through osmotic pressure differential when dentine is pathologically or surgically exposed, through large changes in temperature, or during and after tooth cutting and drying in restorative procedures. The sensibility is very probably mediated by nerve endings in the odontoblastic and sub-odontoblastic layer of the pulp. When the pulp is acutely inflamed, or when there are areas of acute inflammation in the chronically inflamed pulp, sensibility is increased. These phenomena are described in more detail below and in Chapter 12.

Secondary dentine

Odontoblasts normally remain for the life of the tooth, with their cell bodies on the inner surface of pre-dentine and their processes extending into it. They retain their capacity to secrete matrix protein and to form additional dentine. Dentine is slowly laid down throughout the life of the tooth, leading to a gradual reduction in the size and shape of the pulp cavity. This so-called secondary dentine is laid down particularly on the roof and floor of the pulp chamber.

Tertiary (reparative) dentine

Thickening of the dentine occurs more rapidly when the dentinal surface is exposed to the oral environment by accident or wear, or when odontoblasts come into contact with the products of bacterial metabolism at levels below those that might cause death, for example, in advancing caries or beneath a leaking restoration. In these circumstances, the odontoblasts can lay down additional dentine relatively rapidly. This tissue is termed tertiary reparative dentine.

Irregular reparative dentine

If sufficient damage occurs to kill some odontoblasts but the adjacent pulpal tissue survives, new dentine-forming cells can differentiate from the pulpal ecto-mesenchyme. The resultant tissue is called irregular reparative dentine and it may lack the usual tubular structure and may include cell bodies.

Smear layer

Mechanical action on exposed dentine such as the mastication of hard fibrous foods or cutting teeth with metal instruments will produce a smear layer (Figure 1.6) that seems to be a

Figure 1.6 Dentine with smear layer. Smear layer left on the surface of the floor of a cavity following cavity preparation. Courtesy of Professor Hien Ngo.

combination of 'burnished' dentine and, in the case of chewing-induced wear, proteins derived from saliva, or pellicle (see saliva and pellicle, below). In such cases, the dentinal tubules may be effectively occluded, which reduces or eliminates associated sensibility. Following cusp fracture, where no mechanical 'rubbing' of the dentine occurs, the tubules may become blocked by natural deposition of salivary components and, if the salivary circumstances allow, subsequent calcification of those components is likely to occur.

Carbonate, fluoride and dentinal apatite solubility

As described above, surface enamel can become less soluble than the newly formed tissue by ion exchange with saliva. This very probably occurs only to a limited degree within dentine, unless or until it is exposed to saliva. In that circumstance the dentine is initially at relative high risk of loss of mineral, because the apatite is of relatively high solubility. However, as with enamel, if the tissue remains in contact with biofilm, and through that with saliva of neutral or slightly alkaline pH, and if high concentrations of fluoride are available, even episodically, over days and weeks such dentine will become progressively more highly mineralized and less at risk of decalcification.

Dental pulp

Development and constituents

During tooth formation, the growth of dentine inward from the epithelial cap slows dramatically as the tooth matures, encompassing a small body of tissue that is the dental pulp. The rate of dentine formation thereafter is sufficiently slow that a pulp cavity usually remains throughout life although it becomes progressively smaller.

The outer layer of the pulp, which is also the inner layer of the dentine, is comprised of the odontoblastic cell bodies. Immediately beneath this layer is a relatively cell-free zone, rich in sensory nerve endings and blood capillaries. The great bulk of the remaining central pulp tissue is similar to connective tissue elsewhere in the body. It is made up of mesenchymal cells, defence cells and fibroblasts, collagen fibres, ground substance, blood vessel networks (from arterioles to capillaries to venules with accompanying sympathetic nerves), lymphatics, sensory nerve trunks and free sensory endings. This tissue provides metabolic support for the odontoblasts during rapid dentinal deposition, both in initial growth and during repair. If odontoblasts die, but the remainder of the pulpal tissues survives, new odontoblasts can differentiate from the pulpal ecto-mesenchyme to lay down irregular reparative dentine.

As is clear from the dental adaptations that occur in hunter-gatherers and which are described in more detail later in this chapter, teeth have a vigorous capacity to deposit new dentine in response to loss of structure caused by wear. In fact, the entire crown may be lost over only a few decades through wear and the remaining root structures remain vital, functional and healthy.

Sensory innervation of the pulp

Bare sensory nerve endings are in intimate association with the odontoblastic cell bodies, and some extend a short distance into dentinal tubules. Any stimulus that causes movement of these cell bodies may trigger action potentials within the sensory nerve network. As noted above (see section on dentinal sensibility, p. 5), fluid movement within the dentinal tubules therefore elicits sensation, which is interpreted as pain. Cell damage, inflammation or touch within the main body of the pulp also cause pain. The degree of stimulus necessary to bring about a pain response depends upon the sensitivity of the receptors and this will be substantially increased by inflammation within the tissue. It is reasonable to propose that the rich sensory innervation of the pulp serves a protective function for the oral cavity.

The blood supply to the pulp

The blood supply of the pulp is particularly rich, with the rate of blood flow per gram of tissue being similar to that found in the brain. This probably reflects the high metabolic activity levels of the odontoblasts during dentine formation and repair. It may also help the tissue to overcome chemical and bacterial insult. Because of the large number of capillaries present in the sub-odontoblastic layer, there will be a strongly hyperaemic response to local trauma. It is the blood supply of the pulp that determines the vitality of a tooth, not its innervation.

Effect of ageing

With advancing age a number of changes occur in the pulp even in the absence of significant wear, including a decrease in cellularity and an increase in the incidence of pulp stones and diffuse calcification. As the size of the pulp chamber decreases with continued deposition of dentine, the degree of vascularity decreases and so does the capacity of the pulp to withstand various insults.

The pulp best able to respond to rapid wear appears to be that of a relatively young individual. This is a significant factor to be considered in the management of acid-augmented wear, or the wear that can occur following loss of a significant number of teeth, and therefore occlusal support, when it occurs in an elderly individual.

Tooth root and cementum

Root formation

After the crown has formed, the cellular events at the proliferating cervical loop of the enamel organ change and the cemento-enamel junction begins to form. The cells no longer differentiate into functional ameloblasts but continue to induce the formation of odontoblasts, and therefore dentine. The odontoblasts grow inwards, each leaving behind a cell process and matrix proteins that mineralize to form root dentine.

Development of cementum

As the roots continue to form, the outer surface becomes covered with cementum, the fourth tissue unique to teeth. This bone-like tissue is formed by the calcification of matrix protein secreted by cementoblasts, which are cells derived from the adjacent ecto-mesenchyme of the dental follicle. Enmeshed in the cementum are the collagen fibres of the periodontal ligament and it is this system of fibres that connects the tooth root to the adjacent alveolar bone.

Periodontal tissues

Formation of the periodontal ligament

By the time crown formation is complete, ossification of the maxilla and mandible is well advanced. As new bone is formed around the erupting teeth, collagen fibres link alveolar bone to the cementum of the tooth root and the periodontal ligament becomes organized. While a detailed description of the development of periodontal tissues and the process of tooth eruption is beyond the scope of this book, it is relevant to note that by the time the tooth erupts, the oral mucosa overlying the dental arches has become keratinized to form the gingivae, which then adapt closely to the enamel of the tooth crown.

The healthy periodontium has periodontal ligament fibres connecting cementum to adjacent alveolar bone and, near the cemento-enamel junction, fibres connecting cementum to the gingival tissue. The gingivae are supported by these fibres and by the alveolar bone to form a tight cuff of fibrous, connective tissue covered with epithelium around the enamel of the tooth crowns. The epithelium that becomes closely adapted to the enamel at the dento-gingival junction is comprised of two parts: sulcular epithelium that is related to the gingival sulcus or crevice around the neck of the tooth, and junctional epithelium that forms an attachment to the enamel via a laminar structure and a system of adhesive hemidesmosomes. As long as they are in good health, the closely adapted gingival tissues provide an effective barrier against bacterial movement from the oral cavity into the tissues around the tooth.

Saliva

Saliva is made up of water, organic components, mainly proteins, and electrolytes. Three pairs of glands, the parotid, submandibular and sublingual, produce most of the saliva. Minor glands distributed on the tongue, cheeks, palate and lips produce the remainder.

Salivary secretion is controlled by autonomic innervation. Parasympathetic stimulation will increase salivary secretions. Conversely, sympathetic stimulation, such as occurs during the fight-or-flight response, reduces secretions, as do many drugs that affect mood. In the healthy individual the smell or taste of food, thinking about eating, or most markedly the action of chewing will all increase the salivary flow rate, principally from the parotid, submandibular and sublingual glands.

Salivary flow is significantly reduced by many drugs which affect mood, because those drugs also change autonomic neurotransmission and therefore saliva flow control. For many dental patients this reduction in salivary flow is a major contributor to the development of diseases that result in loss of tooth structure. A more detailed description of saliva in that context is found in Chapter 16.

Composition and function of saliva

Components of saliva contribute to many functions that are essential for good oral health. A brief summary that focuses on those factors related to tooth structure follows (see Table 1.1).

Table 1.1 Functions of saliva

Ion reservoir	Calcium, phosphate and hydroxyl ions for remineralization
Buffer	Buffering both dietary and metabolic acids
Pellicle formation	Promotes biofilm formation for protection against acids
Lubricant	Facilitates swallowing and reduces mechanical wear
Mechanical cleansing	Agglutination of food debris, tissue and bacterial cells for clearance
Antimicrobial action	Oral microbes controlled through immunological mediators
Digestion	Amylase and lipase to break down starches
Taste	Solvent for ions that interact with taste buds
Excretion	Urea
Soft tissue health	Lubrication for comfort and includes growth factors for healing

Water

Water is the solvent for the chemical interactions responsible for taste; for the mechanical process of oral clearance; and with salivary proteins called mucins for the lubrication of all hard and soft tissues in the oral cavity. Water softens the food bolus, enabling chewing and swallowing, and assists in speech.

Electrolytes and buffering

Salivary electrolytes include numerous ions found in other body fluids, Ca^{++}, K^{+}, Na^{+}, Mg^{++}, Cl^{-}, HCO_3^{--}, and PO_4^{---}. Among their functions is the buffering of acids. Some salivary proteins also act as weak buffers.

The principal, initial buffer is the phosphate system associated with resting saliva that is most effective at around pH = 7 (± 0.5) and is associated with the following reaction:

$$H^{+} + HPO_4^{--} \leftrightarrow H_2PO_4^{-}.$$

If there is a drop in pH, the reaction moves to the right, producing more di-hydrogen phosphate ions. As the pH continues to drop, all of the mono-hydrogen phosphate can be used up.

At levels below about pH 6.0, a more powerful buffer, a high concentration of HCO_3^{-} ions, comes into effect from stimulated saliva produced by the parotid glands:

$$H^{+} + HCO_3^{-} \leftrightarrow H_2CO_3 \leftrightarrow CO_2 + H_2O$$

Because CO_2 is continuously lost, the reaction keeps moving to the right, making this an extremely effective buffering system.

Organic components

The organic components of saliva include many enzymes and proteins, some of which have antimicrobial properties. These components not only give protection against external pathogens but also influence microbial growth within the oral biofilm.

Salivary proteins and glycoproteins precipitate onto freshly-cleaned tooth surfaces. This initial layer, also called pellicle, was probably not a significant component of the biofilm in hunter-gatherers, except on surfaces undergoing daily wear. However, it may be significant in present-day dental patients, many of whom clean their teeth on a daily basis or more frequently with mechanical aids including toothpaste that contains abrasive particles. Cleaning with toothpaste is likely to remove the bacterial components of biofilm and some pellicle. Pellicle will rapidly re-form by precipitation from saliva. Newly formed or bacteria-free pellicle will be rapidly colonized by pioneer bacteria, but it may take several days or even weeks for a mature and stable bacterial ecology to develop.

Although it is not yet clear what the precise differences are in local biochemistry between biofilm formed in this way and mature biofilm, it appears likely that some differences are clinically relevant, as will be described in sections below.

Other organic components of saliva are digestive enzymes, such as α-amylase, that help break down starches; proteins such as statharins and proline-rich proteins that bind to Ca^{++} ions to limit precipitation; and mucopolysaccharides and glycoproteins that are important for lubrication.

Saliva flow rate

The rate of saliva production or flow is extremely variable both within and between individuals, ranging from between 0.5–1.5 litres per day. Unstimulated flow rates range between 0.3–0.5 ml/min, while stimulated parotid saliva can range between 1.0–2.0 ml/min.

Multiple factors control flow in a healthy individual, including the level of body hydration and factors that relate to biological rhythms. More saliva is produced when standing, less when sitting and even less when lying down. More saliva is produced in a well-lit room and less in a dark room.

Of particular importance to dental health and disease is the marked increase in salivary flow rate that accompanies chewing. As noted above, stimulated flow can be four or five times that of resting flow. Also notable relative to dental health and disease is the increase in available phosphate, the rise in pH and the increased buffering capacity of high flow-rate saliva. The increase in salivary flow during chewing is not only advantageous to the survival of the individual in terms of swallowing and subsequent digestion of food, but also very beneficial in terms of the maintenance of strong and effective teeth, as will be described further below.

Also important to health and disease are the effects of many prescribed and some recreational drugs in reducing salivary flow. Most drugs that affect mood decrease salivary flow rate, and most individuals receiving three or more prescribed drugs suffer from reduced flow or dry mouth. In addition, there are systemic autoimmune conditions such as Sjögren's Syndrome and other pathologies that also can reduce salivary flow significantly. Some elderly individuals also suffer from reduced salivary flow, and reduction in salivary function and flow can result from radiotherapy and from chemotherapy. Whatever the cause, reduction in salivary flow increases the risk of loss of tooth substance.

Biofilms, Diet and 'Mineral Maintenance'

Located between erupted teeth and saliva is biofilm, composed of salivary proteins and glycoproteins, bacteria and their products, water and ions. Biofilm acts as a semi-permeable membrane between saliva and the tooth surface. It is reasonable to state that the human oral biofilm has evolved in tandem with the diet and oral structures, and that balanced interactions between the saliva, the biofilm and the teeth have evolved to ensure good oral health.

The traditional hunter-gatherer diet varied significantly depending on the environment, and, irrespective of the plant-animal ratio of the diet, the pH of the majority of the food consumed was mildly acidic. It is also relevant that, apart from milk

during the breastfeeding of infants, the only liquid consumed by individuals in hunter-gatherer societies was water. The water, usually ground water from rivers, streams or springs, contained fluoride.

The primary driver in these evolutionary interactions was most probably the advantage to survival by maintaining a strong and functional dentition, despite exposure to mildly acid foods. It is *not* likely that the primary driver of the biofilm was in any way related to dental caries, because until recent centuries that disease was extremely rare.

It appears very likely that in the circumstance into which humans evolved, the hunter-gatherer state, the biofilm was relatively stable in structure, and was a *very effective contributor to the maintenance of oral health*. Current evidence suggests that the changes in biofilm that have resulted from changes in human diet and eating patterns in recent centuries are strongly associated with the oral diseases that now demand the attention of dental professionals and their patients.

A mature oral biofilm in the mouth of an individual with little or no simple carbohydrate in the diet, as occurs in hunter-gatherer societies, contains a large diversity of microorganisms living symbiotically with one another. The main bacterial species in that oral ecosystem and biofilm are the non-mutans Streptococci, including S. mitis, S. oralis and S. sanguinis, and Actinomyces. In such a mouth there will be a very low proportion (~2%) of the mutans Streptococci such as S. mutans, S. sabrinus and other acid-producing and acid-tolerant species.

Non-mutans Streptococci generally live in environments that are close to pH neutral and can survive from salivary glycoproteins alone. That is, they are not dependent on the presence of dietary components for survival. Other species, such as Actinomyces, metabolize urea that is also secreted by the salivary glands. Both of these processes produce ammonia, thereby maintaining a mild alkalinity within the biofilm.

The diet influences the resting pH of such biofilm. In hunter-gatherer communities where the diet is high in meat, the biofilm tends to be slightly alkaline, while a higher plant diet tends to produce a resting pH that is slightly acidic. The resting pH in hunter-gatherer populations therefore varies mildly, between approximately 7.4 and 6.7.

Influencing the biofilm is saliva, on one hand, and the apatite of the tooth surface, on the other. The electrolytes within saliva diffuse within the biofilm and, if the pH of the biofilm is neutral or close to it, make the immediate environment supersaturated with respect to apatite. The constituent ions of apatite are highly reactive yet they generally do not precipitate out of solution in saliva, or most biofilm, because they are stabilized by salivary statharins and proline-rich proteins. The proteins can be thought of as keeping the ions apart by complexing with Ca^{++} and therefore preventing precipitation of calcium phosphate compounds. This adaptation also tends to prevent mineral precipitation within salivary ducts and the formation of salivary stones.

Biofilm pH relative to acid food

Although many components of the hunter-gatherer diet are mildly acidic, including meat several hours after slaughter and many vegetables, examination of both fossil remains and of the mouths of present-day hunter-gatherers shows little or no evidence of the effects of acid on tooth structure. This reflects the beneficial co-evolution of teeth, the biofilm and saliva in response to the human diet and its variations over many millions of years.

Mature hunter-gatherer oral biofilm and adjacent saliva very probably has the capacity to neutralize or buffer mildly acidic food. Three buffering mechanisms contribute: (i) phosphate buffer in resting saliva and therefore by diffusion in biofilm; (ii) microbial contributors to the pH neutrality of biofilm, as outlined above; and (iii) by increased phosphate concentration and particularly by bicarbonate in high flow rate saliva, stimulated by chewing hard food. Furthermore, it is highly likely that the biofilm in itself is a semi-permeable membrane that acts to a significant degree as an initial physical barrier to extrinsic acid as it enters the mouth.

Biofilm pH relative to bacterial metabolism of food components

Most hunter-gatherer foods contain mainly protein, fats and complex carbohydrates. Of these components, only the complex carbohydrates can be metabolized by biofilm microorganisms, and such metabolism is relatively slow, leading to little or no change in pH.

Although some simple sugars of various types, for example, the monosaccharides, fructose and glucose, and the disaccharides, lactose and sucrose, may be present in the hunter-gatherer diet, they are either at low concentrations or are not eaten frequently. When these sugars are metabolized by the predominantly non-mutans bacteria, acids are produced at a relatively low level, because the microorganisms are neither very acid-tolerant nor highly acidogenic.

Demineralization and remineralization within the local environment

Acidic food and/or metabolic acids produced by bacteria in the biofilm from simple carbohydrates in the diet would cause the pH of the biofilm to fall only if the local buffering capacity was exceeded. If the pH became low enough and the acids were close enough to the tooth surface to influence the hydration cell of enamel crystals at or just beneath the tooth surface, unsaturated conditions would cause the dissolution of the crystal surfaces. This demineralization would reverse (remineralize) once the acids were buffered and the pH rose (Figures 1.7 and 1.8). Simultaneously with these interactions, drops in pH would cause the statharins and proline-rich proteins to release stabilized calcium within the salivary biofilm, also contributing to remineralization.

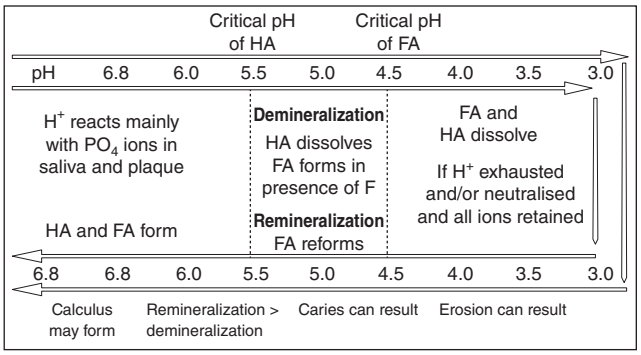

Figure 1.7 The demineralization-remineralization cycle: a conceptual chart to demonstrate the levels of pH at which the stages of the demineralization/remineralization cycle occur. F: Fluoride; FA: Fluorapatite; HA: hydroxyapatite. Source: [6]. Reproduced with permission from Knowledge Books and Software.

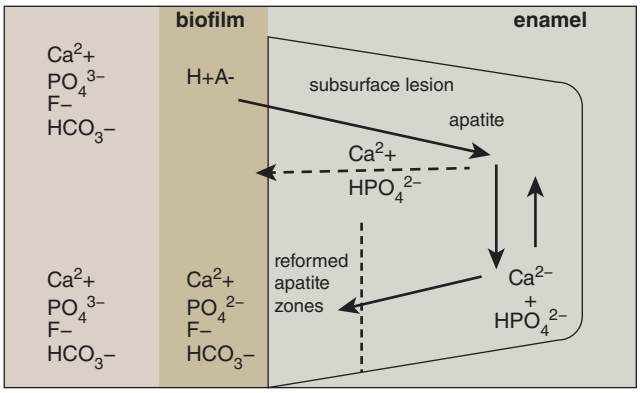

Figure 1.8 The remineralization part of the cycle. Note that the factors that favour remineralization include increased Ca^{++}, increased $PO_4^{—}$, raised pH, and the presence of F^-. Source: [6]. Reproduced with permission from Knowledge Books and Software.

This can be described as a 'closed system' demineralization–remineralization cycle, where the raw products from the dissolution of tooth structure are kept within the local environment and are reused for remineralization.

This local, closed system demineralization–remineralization cycle can only occur in the presence of an appropriately structured oral biofilm. Such a cycle not only protects the tooth from demineralization, but also promotes enamel maturation.

Enamel maturation

As explained above, the apatite of the newly-erupted tooth surface is highly substituted with carbonate, in particular, making it relatively soluble. During closed system demineralization–remineralization cycles as described above, the apatite crystals of enamel at and near the surface are in dynamic equilibrium with the adjacent aqueous phase of the biofilm. Since saliva, and therefore biofilm, have a lower concentration of carbonate, and a higher concentration of phosphate than the adjacent tooth crystal, carbonate is progressively replaced with phosphate over time through the many local closed cycles of demineralization and remineralization.

Similarly, fluoride from natural ground water replaces some hydroxyl groups, depending on local fluoride concentration at the tooth surface. In the modern context, fluoride can also be made available from supplements, topical treatments, water supplies or toothpaste. Whatever the source, fluoride is available for substitution both by simple ion exchange and through the local demineralization–remineralization cycle. The result of both substitutions over time will be a significantly less soluble, or more mature, enamel surface.

The Oral Environment in Disease

Imbalance within the sialo-microbial dental complex

Dental caries, dental corrosion (also called erosion), some forms of abrasion (e.g. the wedge-shaped lesions of toothbrush abrasion, and acid-accelerated wear), and abfraction, are modern-day diseases. They probably result from diet and lifestyle changes that have occurred since the advent of farming some 10,000 years ago, and particularly since the widespread manufacture and distribution of food components, in particular, sucrose, began about 300 years ago.

Each of the conditions listed above is either rare or, in the case of wedge-shaped lesions, has not been found, in hunter-gatherer societies. In contrast, each condition is relatively common in present-day agricultural and industrial societies. In addition, cusp and tooth fracture, commonly found in heavily restored teeth in today's societies, were also probably uncommon in the pre-industrial era, except when caused by external trauma.

The biggest change has been in the human diet for most of the world's population. Foods today are much less abrasive, because they are gathered and prepared in ways that reduce or eliminate sand or similar particles. They are generally easier to chew, so they promote less salivary flow and less wear. They frequently contain sucrose, a simple sugar that, prior to recent agricultural and commercial development, was rare in the human diet. They also contain other simple carbohydrates in much higher concentrations than had been the case prior to developments in agriculture and the commercial manufacture and distribution of food.

Many liquid foods consumed today are extremely acidic, in particular, carbonated beverages, fruit juices, wine and 'sports drinks'. In the hunter-gatherer era, water was only consumed when thirsty, and was pH neutral. Food rarely had a high degree of acidity. When it did, for example, during the opportunistic consumption of unripe fruit, the exposure to acid was seasonal and transient. Many liquid foods now available commercially in the form of carbonated beverages, 'sports drinks' and fruit juices are high in fermentable carbohydrate concentration as well as

being deeply acidic. Many such liquids are consumed for reasons other than hunger or thirst, for example, because of a belief in nutritional benefits in the case of some fruit juices, or because of the addictive effects of caffeine in the case of cola and other commercially distributed drinks.

Each of these changes in diet has strong and adverse effects on the oral ecosystem and on oral health. Factors that reduce salivary flow are probably the next most important contributor, after changes in diet, to the additional increases in incidence of dental diseases in recent decades.

The changes in diet and salivary flow have impacted directly on the nature of the biochemistry internal to oral biofilms, effectively tipping the balance towards demineralization. Although dental caries and the erosion-related group of diseases are separate and distinct from each other, and are described as such below, they have in common the effect of changes in biofilm structure and biochemistry, and therefore on tooth structure loss, principally through demineralization.

Dental Caries

Dental caries is the progressive loss of tooth mineral over time, caused by biochemical circumstances at, and slightly beneath, the tooth surface in which demineralization outweighs remineralization. Caries can begin on enamel surfaces or on exposed dentinal or root cemental surfaces, and becomes a significant problem for the afflicted individual when associated bacteria reach, infect and kill the dental pulp. Bacterial infection can then spread deep into facial bones through the root canal system, leading to localized, then systemic infection, illness and, in the absence of either surgical intervention or natural drainage, which may not readily occur, death.

Dental caries became common in many European societies and then elsewhere in the world following the introduction into the diet of sucrose in large amounts, through its agricultural production, industrial extraction and refining, and widespread commercial distribution at relatively low cost. Until as recently as 100 years ago sucrose was actively promoted as a safe and economical source of calories for human nutrition, with little recognition of its adverse health effects. Despite very clear evidence of its causal link to dental caries and to several systemic metabolic diseases (in particular, obesity, diabetes and cardiovascular disease), sucrose remains an almost ubiquitous component of manufactured food, and is widely used in home and commercial cooking.

The rapid development of what can be a life-threatening disease, caries, in many societies since the commercial development of the sucrose industry led to the development of the dental professions as we now know them.

The pathogenesis of the early carious lesion

It is reasonable first to explain the initiation of carious lesions in terms of the extended ecological plaque hypothesis. The hypothesis proposes that if a previously normal and non-pathogenic biofilm is frequently exposed to fermentable sugars, the acidification of the biofilm becomes more frequent from acid produced by facultative non-mutans bacteria. This initially mild but frequent acidification of the biofilm selects for more aciduric (acid-tolerant) and acidogenic (acid-producing) bacterial strains, causing the biofilm as a whole to gradually become more acidic over time. This change selects for aciduric and acidogenic mutans Streptococci and other acid-tolerant species such as lactobacilli and bifidobacterium, further compounding the effect. At the same time there is selection against those species that produce alkalis and prefer alkaline environments, causing an overall reduction in the biodiversity of the biofilm.

In these circumstances, where the resting pH of the biofilm becomes lower, relatively little simple carbohydrate in the diet is required to produce a further drop in pH to a level where demineralization occurs. This is termed the critical pH. In periods between the ingestion of foods or drinks, pH in biofilm will rise due to salivary buffers and lessened metabolic activity by biofilm bacteria, but more slowly and to a lesser extent than would otherwise have been the case. The balance in what is termed the demineralization-remineralization cycle will therefore be tipped towards demineralization.

In extremely high caries activity individuals, the resting pH of biofilm can be below the critical pH. In such cases the tooth will continually dissolve because the buffering effect of apatite dissolution and the effect of saliva-derived buffers are insufficient to halt the process.

It can also be reasonably proposed that a unique metabolic interaction between sucrose and the mutans Streptococci can add to the acid pathogenicity of such biofilm. Epidemiologically, caries was rare, even in agricultural societies, before the widespread introduction of sucrose into the diet, despite patterns of relatively frequent eating of other simple carbohydrates. In conditions of strict dietary control in human groups where sucrose is effectively eliminated, caries is as well, despite the frequent ingestion of other sugars.

The metabolic and biochemical link between the high prevalence of caries and the presence of sucrose in the diet is probably the production from sucrose, and therefore the addition to the biofilm environment, of substantial amounts of extracellular polyglucan gel by mutans Streptococci and closely related species. Sucrose is the only substrate from which these bacteria can produce a surrounding polyglucan glycocalyx. The glycocalyx confers a definite selective advantage to the producing bacteria in terms of their survival and reproduction. It also probably changes both the thickness and the ion diffusion biochemistry of the biofilm, increasing its caries pathogenicity.

It is clear that the mutans Streptococci are of major significance in the pathogenesis of dental caries. They are highly acidogenic, highly aciduric, and are the sole bacterial species in biofilm capable of synthesizing extracellular polyglucans from sucrose. Although S. mutans is present in most modern human mouths, its proportion can be kept low by restricting both dietary sucrose

and eating frequency. In such mouths, biofilm is very unlikely to contribute to the initiation and progress of caries.

S. mutans infection of the human mouth most commonly occurs during early years by direct or indirect salivary contact with infected parents, siblings or playmates. Parents can be taught how to avoid S. mutans infection in their infant children, which can protect the children from the risk of caries in their early years.

Demineralization outweighs remineralization at the ionic level

The demineralization process during a cariogenic challenge can be summarized as follows. When the acidic conditions are severe within the biofilm, the phosphate and bicarbonate buffer ions, diffusing inwards from saliva, can become overwhelmed, leaving un-buffered H^+ ions that percolate into the enamel laminar pores. These unsaturated conditions within the hydration cell, adjacent to the tooth crystal, will cause the HA to dissolve. In this circumstance there will be net mineral loss from the enamel subsurface, for reasons to be described below.

It should be recognized that there are multiple biofilm ecosystems within one mouth. There are very likely to be significant differences in biofilm ecology between interproximal areas, smooth surfaces, fissures and partially emerged teeth. Together with the differences in salivary oral flow, and therefore buffering capacity in different parts of the mouth, these factors will lead to some areas being more at risk of demineralization than others.

Biofilm is also markedly thicker in some locations than others, namely, within pits and fissures, immediately beneath contact areas between teeth and at the tooth–gingiva junction (see Chapter 2). These locations are the commonest sites for caries initiation in all human populations, probably because of the different demineralization–remineralization biochemistry within thick biofilm.

Subsurface demineralization of enamel

It is a consistent finding that the initial lesion in enamel is beneath the enamel surface. This is probably because of the nature of the diffusion and reactions of different ions during the demineralization–remineralization cycle within the biofilm and the enamel, at the micron scale.

During demineralization, hydrogen ions from biofilm may diffuse into enamel more quickly, and to greater depth, than will saliva-derived buffers and calcium and phosphate ions, also from or via biofilm, during remineralization. The enamel surface may therefore tend to remain more intact than the few microns beneath the surface (Figure 1.9).

Incipient subsurface enamel lesions are more porous and contain more water, changing the refractive index, to the extent that the affected area appears as an opaque white, a 'white spot' area, rather than being translucent (Figure 1.10). The visual detection of 'white spot' areas and steps taken to reverse them by guided remineralization strategies are key stages in the management of early carious lesions, as will be described in Chapter 2.

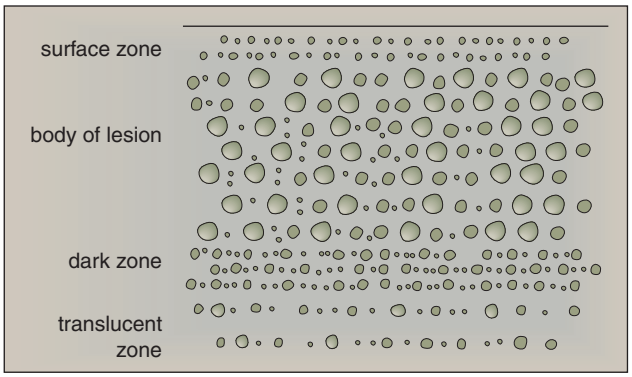

Figure 1.9 Distribution of pore sizes in early caries lesion in enamel. The main initial demineralization is below the enamel surface. Source: [6]. Reproduced with permission from Knowledge Books and Software.

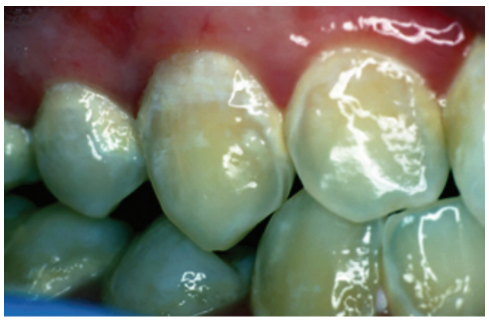

Figure 1.10 Extensive 'white spot' lesions, the early and reversible stage of dental caries in enamel. Most probably due to a combination of poor oral hygiene, frequent contact with simple carbohydrates, and limited salivary flow. Courtesy of M.S. Wolff, New York University College of Dentistry, New York, NY. Reproduced with permission from M.S. Wolff.

Pathogenesis of the advancing coronal lesion

What has been described so far is now widely and appropriately termed the 'reversible carious lesion' of enamel. While the enamel surface remains relatively intact, there is the potential to remineralize the lesion. However, if the demin-remin imbalance continues, the surface of the incipient lesion will become progressively more porous and will finally collapse through dissolution, and therefore weakening, of the mineral connection between crystals. This collapse will result in surface cavitation. The biofilm in the resultant cavity will change in its microbial composition. There is also less access for the salivary buffer. While with profound biochemical change the progress of the lesion can be *halted* at this stage, because of the loss of a remineralizable matrix, the possibility for complete *reversal* of the lesion is lost.

Once demineralization has progressed through enamel into dentine, bacteria become permanent inhabitants of the lesion. Mutans Streptococci constitute most of the flora in biofilm within resultant defects (~30%), while Lactobacilli, Prevotellae and Bifidobacterium are more associated with the advancing

demineralization front within dentine. Demineralization will still be driven by dietary substrate. The bacteria produce acid to dissolve the hydroxyapatite of deeper dentine, diffusing most readily down the dentinal tubules (Figures 1.11 and 1.12). There will be a region of demineralization in advance of the bacterial front.

As demineralization continues into the dentine, the acid front diffusing ahead of pioneer bacteria triggers a dentine/pulpal complex response. Mineralization may occur in the outer dentinal tubules, including the lateral canals that unite the main tubules. This causes the properties of light transmission through the dentine to be altered, producing what is called a translucent layer. At the same time the deposition of reparative dentine can occur within the pulp chamber.

Bacteria in the cavitated lesion will continue the demineralization process and, usually at a later stage, may cause some breakdown of the collagen structure by excreting proteolytic enzymes. Below this softer, fully demineralized carious dentine will be an advancing front of demineralization caused by acid diffusion, in the order of millimeters ahead of the gross bacterial invasion. There may be a few pioneer bacteria in, or even beyond, the area of demineralization within dentinal tubules, but fortunately these are not clinically relevant, as will be described in detail in Chapter 12.

Figure 1.11 Progress of the carious lesion into dentine. The demineralization front and then bacterial invasion follow the dentinal tubules towards the pulp.

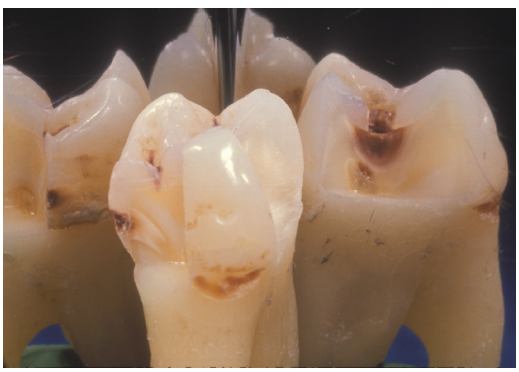

Figure 1.12 Progress into dentine. Note in the proximal lesion of the left the typical penetration towards the pulp. The occlusal lesion seen on the right shows penetration approximately twice as deep as it is wide.

Zones within dentinal caries

Two distinct layers or zones of demineralization can be observed in dentinal caries. These have been identified as the *infected* zone (the outer layers), and the deeper *affected* or partially demineralized zone. The infected zone is characterized by a high level of bacterial contamination, and complete demineralization of the dentine framework leading to total or partial collapse of the dentine tubular structure and intertubular dentine. The affected zone has sufficient mineral content to retain the dentine tubular structure and, importantly, sensitivity, even though the mineral content is partially lost. Providing there is at least 10% of the original level of mineral remaining, remineralization is possible (see also Chapter 12).

The slowly progressing lesion

Under some circumstances, as the lesion advances, the enamel will become progressively undermined and weakened. Collapse of the unsupported enamel may eventually result in a relatively open cavity with good salivary access, even one that becomes relatively self-cleansing, so plaque may not be so readily retained. The caries process may then slow down, leading to the development of a hard leathery floor on the cavity that is very dark and more or less inactive.

At times, slowly advancing lesions allow time for pulpal defences to become clinically significant. These include the deposition of reparative dentine and also the deposition of mineral within the affected dentinal tubules, causing them to become completely obturated. This 'sclerotic' dentine effectively seals the pulp from the advancing lesion. Often such lesions are symptomless.

The rampant lesion

In some biochemical circumstances, caries advances very rapidly. This state is called rampant caries, and is described in detail in Chapter 16. Cavitation in enamel occurs quickly, the dentine floor of the cavity becomes softer, and the pulp will be at risk of irreversible damage because the remineralizing and sclerosing process, which normally reduces the permeability of the tubules, will be unable to keep pace with the advancing lesion. Bacterial by-products can cause the death of the odontoblastic processes and pass through patent tubules, called dead tracts, causing pulpal inflammation and pain.

Root surface caries

Although at the biochemical level the process of demineralization of the root surface is essentially identical to the process of enamel caries, there are important differences that need to be recognized. First among these is that the root surface lesion begins in dentine, rather than within the enamel surface (Figure 1.13).

In enamel caries, the early lesion is identifiable as a 'white spot', whereas the early root surface lesion may be very difficult to detect because there is likely to be minimal or no colour change, but instead only a modification in surface texture or hardness.

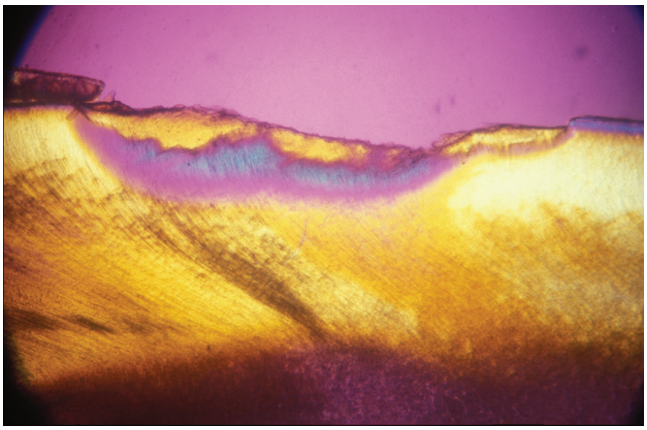

Figure 1.13 Root surface caries in section under polarized light. Note the surface yellow zone of highly demineralized dentine. The purple zone is partly demineralized. Magnification x 35.

The mineral content of root surface dentine is much lower than that of enamel so, when demineralized, it will rapidly expose the collagen matrix, generally retaining much of its protein structure as long as it remains well hydrated. The exposed matrix is susceptible to physical damage but it can readily be remineralized by salivary calcium and phosphate and by fluoride, providing that the causative biochemical circumstances are eliminated and that the demin-remin balance is therefore stabilized. The surface of even quite advanced root caries lesions may therefore be re-hardened through a reduction in eating frequency and the application of topical fluorides or remineralizing solutions.

The enamel is generally not involved in the early stages of root caries but the lesion may extend up and under the cervical margin of the enamel crown as the lesion progresses (Figure 1.14).

The advancing lesion will darken over time through bacterial activity and the uptake of dyes from food. Identification is then easier but it is always difficult to define the full extent of the lesion. As with all dentine caries, there will be an affected zone where the demineralization is in advance of the bacterial infection. This will be a softened, demineralized, colourless zone of dentine on the floor of the cavity that should not be removed during cavity debridement because it can be sealed from the oral flora and subsequently remineralized. Sealing the surface assists the natural repair mechanisms and leads to a reduced challenge to the pulp.

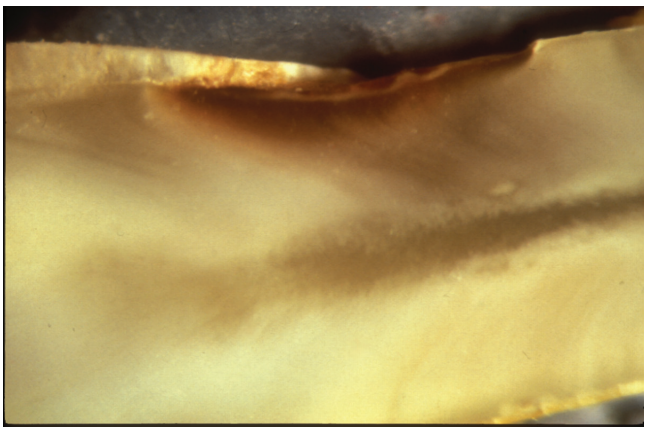

Figure 1.14 Root surface caries in section under transmitted light. Note that the lesion extends laterally under enamel, as well as following the tubules towards the pulp. Magnification x 35.

Dental Corrosion (Erosion)

Dental corrosion can be defined as the superficial loss of dental hard tissue due to chemical demineralization by acids of non-bacterial origin. Although 'erosion' was the word most commonly used in earlier dental texts to describe the condition, 'corrosion' is a more accurate term for what occurs and will be used here. In materials science, the term 'corrosion-erosion' is also sometimes used for acid-induced dissolution of materials. 'Corrosion' means dissolution or damage by acids, whereas 'erosion' means washing away. There is little doubt that what occurs when teeth are exposed to non-bacterial acid is due to the acid.

In Paleolithic populations, the food consumed, irrespective of the environment, was the main source of acid. Clinical evidence of corrosion, as we know it today, was insignificant. With the advent of farming, apart from an increase in carbohydrate consumption, there was also an increase in acid consumption. Communities learned how to ferment food and drinks from plants and milk, culminating in the current 'modern' lifestyle where consumption of acidic drinks has grown greatly, resulting in the high prevalence of corrosion seen in many societies today. Corrosion can also result from frequent exposure to gastric and other non-dietary acids.

The clinical appearance of corrosion varies. In active corrosion, the whole tooth crown may be affected with a loss of surface definition leading to a 'frosted glass' appearance with loss of prominent enamel ridges as they become rounded off (Figures 1.15 and 1.16). The enamel surface may become relatively

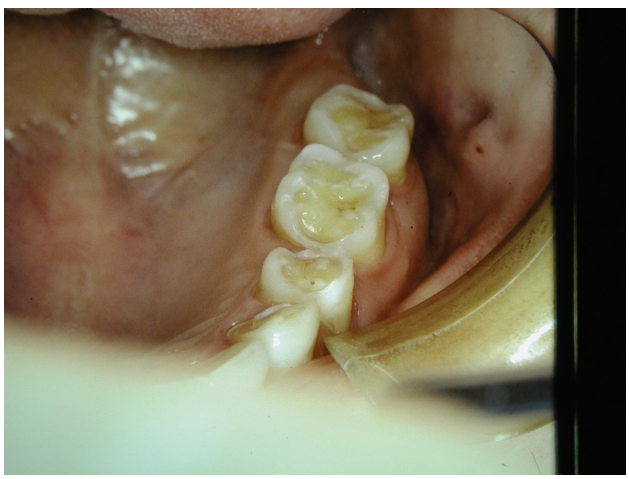

Figure 1.15 Active corrosion-accelerated wear on bicuspid and molar teeth. Note the glazed surface, the loss of micro-anatomical detail and the scooping of the dentine.