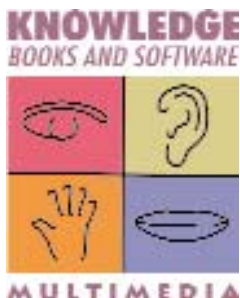


PRESERVATION AND RESTORATION OF TOOTH STRUCTURE

Graham J. Mount and W. R. Hume



Acknowledgements

Graphics imaging Brian Stewart
Publisher Rob Watts
Layout design John Faulds
Graphics Dean Maynard

© 2004 Knowledge Books and Software

All rights reserved. Published 2004.

This book is copyright. Apart from any fair dealing for the purposes of private study, research, criticism and classroom use, as permitted under the Copyright Act, no part may be reproduced by any process without written permission. Inquiries should be addressed to the publisher.

Knowledge Books and Software

ABN 75003053316

PO Box 50, Sandgate, Queensland, 4017, Australia

40 Seaview Street, Brighton, Queensland, 4017

Telephone (07) 3869 0994; 1-800-773353

Facsimile (07) 3269 6444

Email: orders@kbs.com.au

Website: www.kbs.com.au

Printed in Australia.

ISBN ~~1920824 34 0~~

Product code: Dent02.

Foreword

It is a pleasure and a privilege to write a foreword to this new edition of the *Preservation and Restoration of Tooth Structure*. This is a book for students of all ages: undergraduates, postgraduates and experienced practitioners. I will, however, address my remarks to the undergraduates who will need to study this excellent textbook in depth.

At undergraduate level, subjects are often taught in compartments such as anatomy, pathology, dental materials, operative dentistry, periodontology. However, as soon as you meet patients, these packages must merge into an holistic approach to the dental care of the person in your chair. This text takes the holistic approach to the teaching of operative dentistry, showing you the relevance of these individual subjects to the preservation and restoration of tooth structure. Thus you are led from relevant anatomical considerations, to the pathology of dental caries and tooth wear. The role of operative dentistry is set in the context of controlling these pathological processes. When repair is needed, as part of disease management, you are shown the principles of tooth restoration and this inevitably involves a careful consideration of the materials available. Patients have gums as well as teeth that meet and move across each other, and for this reason, chapters covering periodontal and occlusal considerations are an essential part of the text.

We are now in the era of adhesive dentistry. An appropriate amount of diseased tissue is removed and the tooth repaired with a tooth-coloured rest-

orative that bonds to, and supports, the remaining tooth structure. There is no such thing as a 'standard' cavity preparation. To make sense of the subject, your preclinical course should have been taught on real carious and restored teeth. I sincerely hope that you were not taught to cut stereotyped holes in plastic counterfeits because this would be so counterproductive to your understanding, as to be worse than a waste of time!

The authors have placed the chapters in a logical progression envisaging you working systematically through the text; however, there are other ways to use the book. It is beautifully illustrated, so try just looking at the pictures and their figure legends. Alternatively, when exams loom and you are too tired to revise, just concentrate on those 'note' 'be aware' and 'summary' boxes.

Finally, notice the quality of the operative work illustrated here. You can achieve this from your first day in the clinic provided you are critical of your efforts and demand that your teachers are prepared to pick up a handpiece, an instrument, and demonstrate how to perfect what you have done. This is when you will really learn the 'art' of restorative dentistry and the results will give you the buzz of satisfaction that is the key to your continued enjoyment of the technicalities of the subject.

Edwina Kidd
Emeritus Professor of Cariology,
Guy's, King's & St. Thomas's Dental Institute,
University of London

a

Contents

Introduction

Acknowledgements

Contributors

1	Tooth Structure	1
	<i>W. R. Hume, G. C. Townsend</i>	
	Enamel	
	Dentine	
	Dental Pulp	
	Tooth Root and Cementum	
	Periodontal Tissues	
2	Disease Dynamics and the Dental Pulp	11
	<i>W. R. Hume, W. L. K. Massey</i>	
	Insults to the Pulp	
	Defence within Dentine	
	Inflammation in Response to Mechanical,	
	Thermal and Chemical Insults	
3	Dental Caries –	
	The Major Cause of Tooth Damage	21
	<i>J. M. McIntyre</i>	
	The Multifactorial Aetiology of Dental Caries	
	Mechanism for Caries Development	
	The Progressing Caries Lesion	
	Identification of Caries Lesions	
4	Preventive Management of Dental Caries	35
	<i>J. M. McIntyre</i>	
	The Most Effective Approach to Prevention	
	Assessing Dietary Factors in Caries Development	
	Evaluating and Improving Oral Hygiene	
	Evaluating and Enhancing Salivary Protective Factors	
	Function and Prescription of Fluorides	
	Prescription and Application of Chlorhexidine	
5	Non-carious Changes to Tooth Crowns	47
	<i>J. A. Kaidonis, L. C. Richards, G. C. Townsend</i>	
	Terminology	
	Aetiology of Tooth Reduction	
	Diagnosis	
6	Risk Assessment in the Diagnosis	
	and Management of Caries	61
	<i>H. C. Ngo, S Gaffney</i>	
	Introduction	
	Traffic light-Matrix (TL-M) Risk Assessment Model	
	Risk Assessment for the Individual Patient	
	Clinical Application of TL-M	
7	Lifestyle Impacts on Oral Health	83
	<i>L. J. Walsh</i>	
	The Importance of Saliva	
	Lifestyle Factors and Dental Caries	
	Modifications in Treatment	
8	Additional Aids to the Remineralisation	
	of Tooth Structure	111
	<i>E. C. Reynolds, L. J. Walsh</i>	
	Introduction	
	Anticariogenic Casein Phosphopeptides	
9	Instruments Used in Cavity Preparation	119
	<i>G. J. Mount, L. J. Walsh, A. Brostek</i>	
	Rotary Cutting Instruments	
	Speed Groups	
	Air Abrasion Techniques	
	Pulsed Erbium Lasers (Er:YAG and Er,Cr: YSGG)	
	Chemo-mechanical Caries Removal (Carislov™)	
	Conventional Hand Instruments	
10	Basic Principles for Cavity Design	145
	<i>G. J. Mount</i>	
	Introduction	
	Principle Techniques for Placement	
	Protection of Remaining Tooth Structure	
	Other Significant Factors	
	The Final Selection	
	The Use of Rubber Dam	
11	Glass-ionomer Materials	163
	<i>G. J. Mount</i>	
	General Description	
	Properties	
	Clinical Considerations	
	The Lamination or ‘Sandwich’ Technique	

12 Composite Resins	199	16 Vital Pulp Therapy	299
<i>J. C. L. Neo, A. U. J. Yap</i>		<i>G. J. Mount, W. R. Hume</i>	
Introduction		Indirect Pulp Therapy	
Composition, Setting and Classification		The A.R.T. Technique	
Properties			
Clinical Considerations			
13 Dental Amalgams	219	17 Periodontal Considerations in Tooth Restoration	309
<i>R. W. Bryant</i>		<i>G. J. Mount</i>	
Description of Dental Amalgam		Normal Gingival Tissue	
Properties		Problems Which Compromise Periodontal Tissues	
Clinical Manipulation		Effect of Restorative Dentistry on Gingival Tissue	
Clinical Aspects of Amalgam Restorations			
Biocompatibility – Mercury and Dental Amalgam		18 Occlusion as it Relates to Restoration of Individual Teeth	323
14 Classification and Cavity Preparation for Caries Lesions	243	<i>G. J. Mount</i>	
<i>G. J. Mount, W. R. Hume</i>		Basic Principles of Occlusion	
Introduction		19 Choosing Between Restoration Modalities	337
A New Cavity Classification		<i>G. J. Mount</i>	
Site 1 Lesions		Introduction	
Site 2 Lesions		Glass-ionomer	
Site 3 Lesions		Composite Resin	
		Amalgam	
15 Pulp Protection During and After Tooth Restoration	289	Gold	
<i>W. R. Hume</i>		Ceramics	
Avoidance of Pulpal Damage Due to Caries		20 Failures of Individual Restorations and Their Management	347
Avoidance of Pulpal Damage During Cavity Preparation		<i>G. J. Mount</i>	
Protective Measures During Restoration Placement		Failure of Tooth Structure	
Chemical Diffusion and Fluid Flow Through Dentine		Failure of Restorative Material	
Risks to the Pulp from Plastic Restorative Materials		Fracture or Collapse of a Restorative Material	
Materials Used in Pulp Protection		Total Loss of a Restoration	
		Change of a Restorative Material	

Introduction

In the overall scheme of personal health the art and science of operative dentistry has little to do with the patient's life span but a lot to do with their lifestyle. Physical comfort, enjoyment of food and drink, overall bodily health, aesthetics and personal pride are all affected by the state of the oral cavity and the dental profession took sole responsibility for this over a century ago. There has been considerable improvement in the abilities of the profession and the attitude of the public to dental health, particularly over the last fifty years, and this is as it should be.

This book is presented again in modified form to acknowledge further change since it was first written in the early 1990s. It was designed then to identify the changes that were taking place and this second edition is written to expand upon the further changes that have been recognised and accepted in the last ten years. Understanding of the disease process is becoming more sophisticated, techniques for early identification, prevention and healing are improving, terminology is changing and patient expectations are rising.

It would seem that the greatest fundamental change is recognition of the concept of 'minimal intervention dentistry'. The dictionary defines 'minimal' as the 'smallest possible in amount or least possible in extent'. 'Intervention' is defined as 'an action undertaken to prevent something undesirable'. The concept therefore is to carry out operative dentistry in the most conservative manner possible and thus to limit the amount of undesirable consequences and this is now widely recognised.

It is suggested that there is sufficient evidence now available for the profession to modify its approach to the treatment of dental caries which, for a long time, has involved a very heavy handed technique based upon the concept of a surgical cure for a bacterial disease. Probably the greatest problem for both operator and patient has been to connect the two – the introduction of the disease process in to the oral environment and the ulti-

mate visible end result – that is, white spot lesions and frank cavitation. It can take up to four years for demineralisation to penetrate the full depth of the enamel and a further four years to reach the pulp through the dentine so the connection can be difficult to explain. But the level of knowledge is such now that the profession should concentrate on the disease process and overcome that, before considering what is necessary to repair the damage done in the form of surface cavitation. In fact, many early lesions can be healed and remineralised through elimination of the disease with no need to resort to surgery at all.

The average life span of a restoration is 10-15 years. The average life span of our patients is extending and is now in the vicinity of eighty years. The restorative materials currently available continue to improve but they remain a poor substitute for natural tooth structure. With current knowledge it is now possible for the individual patient to minimise the problems that still occur from caries and non-carious tooth loss and help to ensure that their teeth will last well in to the 8th and 9th decade of life.

The first discovery of serious significance to challenge and change the G. V. Black approach was the recognition of the importance of the fluoride ion in the demineralisation/remineralisation cycle which may lead to a caries lesion. This occurred just over 50 years ago and has led to a dramatic reduction in the caries rate in fluoridated communities. The modes of function are becoming well understood but it is important to recognise that fluoride is not the only important ion in the oral environment. Calcium and phosphate ions are essential components of saliva as well as the major components of teeth themselves.

There is, quite deliberately, considerable emphasis on saliva in this volume. The importance of the nature, the components and the health of the saliva are finally being recognised in the maintenance of oral health. Apart from calcium, phosphate and fluoride ions the saliva contains bicarbonate

buffers to assist in breaking down the acids generated from food and drink or from bacterial activity in plaque. The normal flow, texture and buffering capacity can vary considerably in an otherwise healthy patient and is subject to rapid change as a result of variations in good health. As the mouth is a major portal of entry to the body there is always a bacterial flora, some of which are both aciduric and acidogenic. But the flora can be controlled or modified.

There are two distinct formats for loss of tooth structure – carious demineralisation caused by bacteria and non-carious tooth loss resulting from long term low pH in the oral environment. Non-carious tooth loss is an insidious process that is becoming more common because of changes in diet and lifestyle and the early stages are difficult to identify. The damage done can be just as serious as caries and early recognition is imperative.

This book attempts to gather the current knowledge and understanding of the health of the oral environment and the caries process and to offer logical alternative methods of returning the situation to normal. It begins with a brief study of what is regarded as normal and then investigates the disease state, both caries and non-carious tooth loss. Modern methods of diagnosis and treatment planning are detailed as well as innovative methods of remineralisation and healing of the early lesion. There follows a detailed discussion of methods of cavity preparation both old and new with emphasis on minimal intervention.

Three chapters in the book discuss the present understanding of the principle direct restorative materials on the understanding that these are the logical materials to use in minimal intervention dentistry.

One of the most significant discussions covers the introduction of a new method of identification and classification of lesions of the tooth crown so that, in future, the profession will be encouraged to consider preservation of tooth structure as the main aim during restoration of lesions. It is imperative to recognise that the classification is there only to allow identification of lesions and in no way dictates either the cavity design or the restorative material to be used in each case.

It is accepted, of course, that the old style G. V. Black dentistry will be with us for a long time yet in the form of replacement dentistry, that is, replacement of restorations that have failed through the effluxion of time. The only constant in any profession should be change and this profession is no exception. If all dentists, from this time on, were to concentrate on early recognition of the disease, and its elimination, and then adopt minimal intervention principles for the treatment of new lesions, our patients would be grateful and the profession would raise itself to new heights as ‘dental physicians’ rather than ‘dental surgeons’.

Graham Mount and Rory Hume

Acknowledgements

As with the first edition of this text book this is the result of a lot of work from a lot of people and it is hard to know where to begin to express appreciation. The inspiration to publish again came from a number of academics, in particular those who have the responsibility for teaching operative dentistry. The concept of minimal intervention dentistry is evolving so fast that both teachers and students, let alone the practising profession, are finding it difficult to keep pace and a single text containing as much as possible of this philosophy is desirable. We make no claim that this is the complete story but we feel it is a move in the right direction and will hopefully continue the evolution of the greatest change in this discipline in a hundred years.

There have been changes in the list of authors mainly because knowledge is expanding and techniques are evolving. Also it was recognised that it was rational to eliminate all reference to the indirect methods of tooth restoration. We felt the concentration should really be on minimal intervention and conservation of natural tooth structure. By the time indirect techniques become necessary the cavity is quite extensive and remaining tooth structure is in need of support and protection. We remain grateful to David Southan who covered most of the indirect section in the first edition and I know he acknowledges the reasons behind the modification.

I am grateful to all our coauthors for their cooperation and tolerance of my editing techniques. They have worked hard to make sure this edition is available for the next academic year and they have kept to a tight time schedule. The illustrations come from the libraries of the authors and

many of the old ones are still present. However, there are plenty of new ones and hopefully they are all relevant.

There is a CD-ROM available again this time but it comes with a different purpose. There did not appear to be a great demand for the disc in the previous edition and it was locked so the illustrations were not readily available. This time the disc is an optional extra and is aimed at the teaching profession. The illustrations are readily accessible and can be downloaded for teaching purposes and their origin is clearly acknowledged on each slide.

In addition, another version of this material is available on a website. The address is

www.midentistry.org

and readers are encouraged to visit it because it reinforces the contents of the book and provides another view of the subject.

I remain grateful to my good friend Michael Williams whose skill in detecting errors and omissions within the text is unsurpassed. There are not many with the dental knowledge and powers of observation required to carry out such a demanding task. Finally I must acknowledge the skill and dedication of the staff at Knowledge Books and Software, our new publishers, who saw to the production in what to me is record time. It is nice to find that we here, on the far side of the world, are capable of producing our own version of modern knowledge in such excellent form.

There is a lot to be said in favour of a productive retirement. I remember my wife made a promise “for better, for worse – but not for lunch” but in spite of it all she has remained as loyal and tolerant as ever and I am very grateful. Maybe this time we will really go caravanning!

Contributors

A. M. Brostek B.Sc. (Monash), B.D.Sc.(WA)
Visiting Lecturer
OHCWA, The University of Western Australia

R. W. Bryant MDS (Syd), PhD (Syd), FRACDS
Professor of Conservative Dentistry
The University of Sydney

S. Gaffney BDS, MASH
Faculty of Dentistry
The University of Adelaide

W. R. Hume BSc (Dent) BDS PhD DDSc (Adel) FRACDS
Professor Emeritus
University of California

J. A. Kaidonis BDS, BScDent, PhD (Adel)
Senior Lecturer in Clinical Dentistry
The University of Adelaide

J. M. McIntyre AM, BDSc (Qld) PhD (Adel)
Visiting Research Fellow
The University of Adelaide

W. L. K. Massey BDS PhD
Harvard School of Dental Medicine
Harvard University

G. J. Mount AM, BDS (Syd), FRACDS, DDSc (Adel)
Visiting Research Fellow
The University of Adelaide

J. C. L. Neo BDS (S'pore), MS (Oper. Dent.)
Assoc. Professor, and Head
Department of Restorative Dentistry
National University of Singapore

H. C. Ngo BDS, MDS (Adel)
Associate Professor
The University of Adelaide

L. C. Richards BDS BScDent(Hons) PhD (Adel)
Professor
Dental School
The University of Adelaide

E. C. Reynolds BSc (Hon.), PhD
Professor and Dean
Faculty of Dentistry
University of Melbourne

G. Townsend BDS, BScDent, PhD, DDSc (Adel)
Professor of Dental Science
The University of Adelaide

L. J. Walsh BDSc(Qld), PhD, DDSc (Qld), FFOP(RCPA), GCED
Professor of Dental Science, and Dean
The University of Queensland School of Dentistry

A. U. J. Yap BDS(S'pore), MSc(London), PhD(S'pore), FAMS
Associate Professor
Department of Restorative Dentistry
National University of Singapore

1 Tooth Structure

W. R. Hume ♦ G. C. Townsend

It is essential to have a good knowledge of tooth structure in order to understand both the nature of the defects and diseases that can occur and to then make rational decisions on their prevention, treatment and repair.

Teeth are composed of four different tissues: enamel, dentine, dental pulp and cementum. Each of these is made up of structural elements found elsewhere in the body, but arranged in unique ways.

In the brief description that follows a basic knowledge of the embryology and histology of the developing tooth is assumed. Readers interested in further information are referred to the reading list at the end of this chapter.



Enamel

Calcification

Ameloblasts differentiate from the inner layer of endothelial cells of the enamel organ of the tooth bud in response to the laying down of dentine by **odontoblasts** derived from the dental papilla. The ameloblasts secrete a mixture of enamel matrix proteins (amelogenins and enamelines) from their basal border to form an extracellular matrix protein gel. Apatite* begins to precipitate within this gel immediately adjacent to each ameloblast.

It is likely that the amelogenin provides an ideal substrate for the precipitation of carbonated hydroxyapatite from the locally supersaturated environment of calcium and phosphate. As each apatite crystallite grows, the amelogenin immediately adjacent to it and much of the enamelin goes into solution. Crystallite growth continues, leaving long apatite crystallites stacked in arrays (enamel rods) corresponding to the parent ameloblasts, with an enamelin-rich boundary layer between rods (**Figures 1.1-3**).

Modifications to calcification

During enamel formation the rate of dissolution of the matrix protein seems to be temperature dependent, episodes of fever during enamel formation cause defects in enamel structure. The

rate of dissolution may also respond to levels of fluoride in the hydroxyapatite crystals, since very high levels of fluoride also cause defects in enamel mineralisation (mottling), while at optimal levels fluoride induces the formation of enamel of low solubility.

Progress of calcification

The process of matrix protein secretion and its almost immediate replacement by hydroxyapatite, with ameloblast withdrawal, continues for a period of years. The ameloblasts leave behind stacks of crystallites that are aligned to form long rods. There is a change in the crystal orientation at the rod boundaries, with individual rods being separated by varying amounts of inter-rod enamel.

Enamel prisms

Human enamel has a physical structure, or 'grain', because of the enamel rods. When enamel fractures it usually breaks along the 'grain' of the prisms. However, 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 some parts of the outermost, layers of enamel are more homogeneously mineralised and are termed 'prismless'.

* The term 'apatite' is used here to describe the mineral of teeth; apatite and its chemistry is described in more detail in **Chapter 3**.

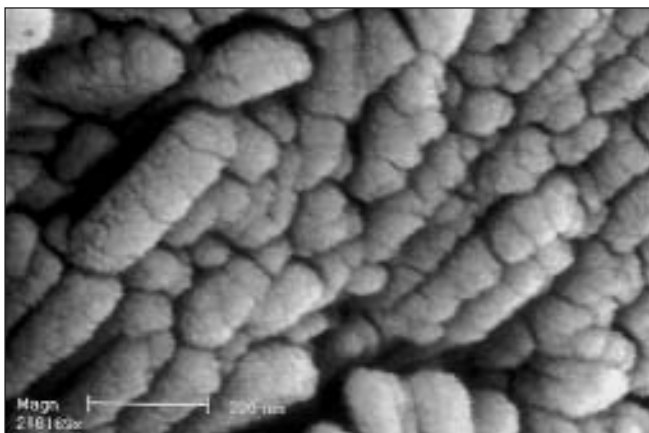


Fig. 1.1. An SEM of the surface of an enamel rod showing the enamel crystals. Note the water filled space around each crystal. Mag. x216,000. Courtesy Dr H. C. Ngo.

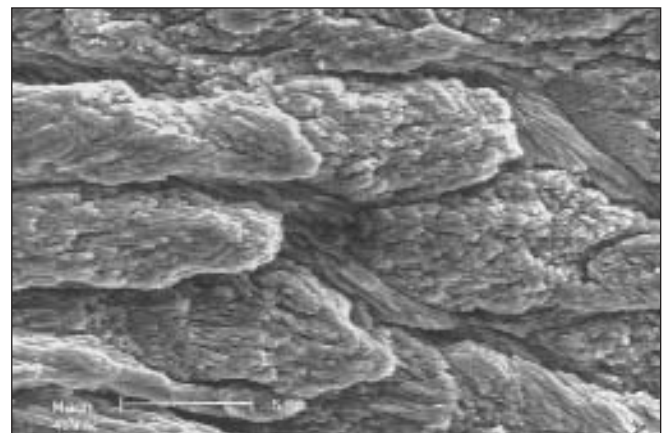


Fig. 1.2. An SEM of fractured enamel showing the rods consisting of bundles of crystals. Note the 'grain' along which fracture may occur. Also note spaces which are water filled. Mag. x5000. Courtesy Dr H. C. Ngo.

Pre-eruption maturation of enamel

Once the ameloblasts have completed secreting matrix they take part in the process of pre-eruption enamel maturation during which the hydroxyapatite crystals continue to grow, with protein and water being lost from the matrix. There is less time for this process in deciduous than in permanent teeth. By the time permanent teeth erupt the enamel is normally 96-98% carbonated hydroxyapatite by weight, and about 85% by volume. The remainder is protein, lipid and water. Pores exist between the enamel crystallites, by volume the water space is about 12%. It is within this aqueous phase of enamel that the dynamics of post-eruption maturation, demineralisation and remineralisation take place, as described below.

Reduced enamel epithelium

Once matrix secretion is completed, the ameloblasts become part of the reduced enamel epithelium covering the tooth crown. When the tooth emerges into the oral cavity most of the reduced enamel epithelium is quickly worn off, although some cellular remnants may remain in occlusal grooves as an amorphous layer (see **Chapter 14, page 248**) Some cells of the reduced enamel epithelium also contribute to the formation of the dento-gingival attachment. On exposure to saliva, the coronal enamel becomes covered by a coating of pellicle that consists of strongly adsorbed salivary proteins and lipids.

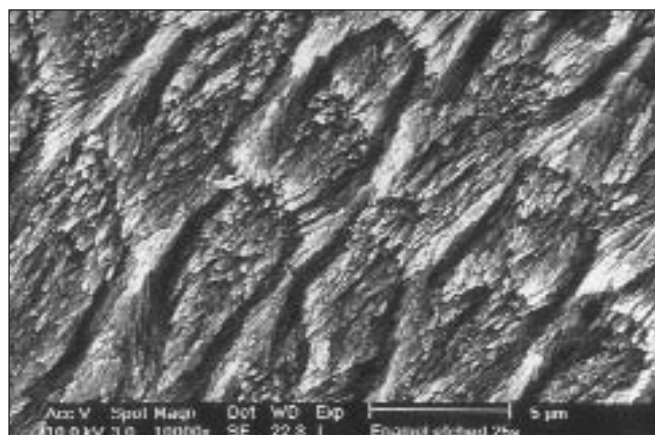


Fig. 1.3. Enamel surface of a tooth following 15 seconds of etching with 37% orthophosphoric acid. Note the ends of the rods with the enamel crystals dissolved from the outer surface. Mag. $\times 10,000$. Courtesy Dr H. C. Ngo.

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 darker. The degree of mineralisation also influences its appearance; hypo-mineralised areas appear more opaque than normally well-mineralised regions, which are relatively translucent.

Enamel striations

Enamel is formed in an incremental manner and fine cross striations may be seen within prisms, representing daily increments of matrix production. Larger striations, the striae of Retzius, probably reflect a 7-10 day rhythm. 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 this in turn will vary the reflection of light rays.

Post-eruption mineralisation

Enamel is quite highly mineralised before the tooth erupts, but further calcium and phosphate deposition in crystal defects continues following eruption because saliva is supersaturated with these ions.

The percentage by volume of mature enamel is approximately 85% inorganic, 12% water and the remaining 3% protein and lipid. Tooth mineral is highly substituted with various ions, including sodium, zinc, strontium and carbonate, which make it more reactive than pure hydroxyapatite.

The apatite crystals of enamel, particularly those at and near the surface, are in dynamic equilibrium with the adjacent aqueous phase of saliva or dental plaque. Over time, carbonate is progressively replaced with phosphate, and fluoride replaces some hydroxyl groups, depending on local fluoride concentration at the tooth surface. In

time, the enamel surface becomes very well mineralised if the pH of its local environment is neutral or alkaline.

Continuing change in enamel

Almost all of the enamel matrix protein disappears as enamel forms. Enamel contains no cells, yet it is far from an inert tissue. Ionic exchange of calcium, phosphate and fluoride both in and out of enamel occurs continually, depending on local concentrations and pH. This is of central importance to many aspects of dental care.

NOTE



There is a continuous exchange of ions between the tooth surface and the oral environment.

Effect of ambient pH

If the enamel in the erupted tooth is high in carbonate and low in fluoride content the critical pH for demineralisation will be pH 5.5. This means that if the oral environment drops below pH 5.5 mineral can be lost from the surface and the central core of enamel crystallites. However, with less carbonate and more fluoride in the enamel the critical pH for mineral loss decreases, and can be as low as 4.5.

BE AWARE



- Low fluoride content enamel – critical pH 5.5
- High fluoride content enamel – critical pH 4.5

When the pH rises above the critical level lost mineral can be regained from salivary calcium, phosphate and fluoride. The dynamics of mineral loss and gain are described in more detail in Chapter 3.

Tissue fluid flow

Filtered tissue fluid moves very slowly outward through enamel in vital, erupted teeth because the pressure inside the tooth is higher than outside. This tissue fluid is called ultrafiltrate and contains no protein, only water and inorganic ions. Ultrafiltrate has the potential to slowly hydrate the inner surface of restorative materials bonded to enamel.

Dentine

Early formation

Concurrently with enamel formation, the ectomesenchymally derived odontoblasts secrete both collagen and relatively complex mucopolysaccharides from their outer end to form the dentinal matrix. The collagen acts as a matrix for mineralisation both during tooth formation and throughout life.

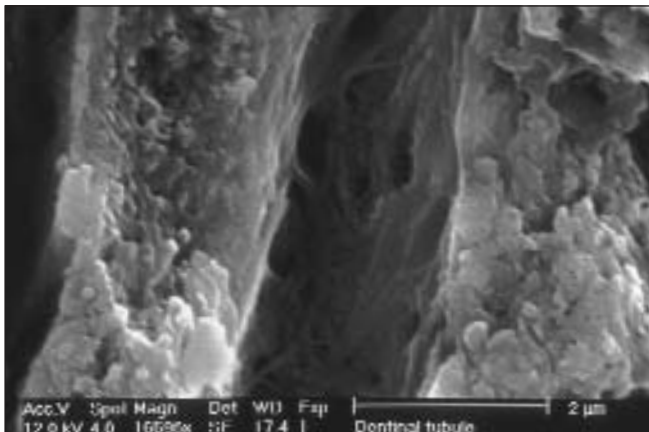


Fig. 1.4. A specimen of dentine split vertically down the length of the dentine tubules. Note the entrances to the lateral canals on the inner walls of the tubule. Mag. x16,600. Courtesy Dr H. C. Ngo.

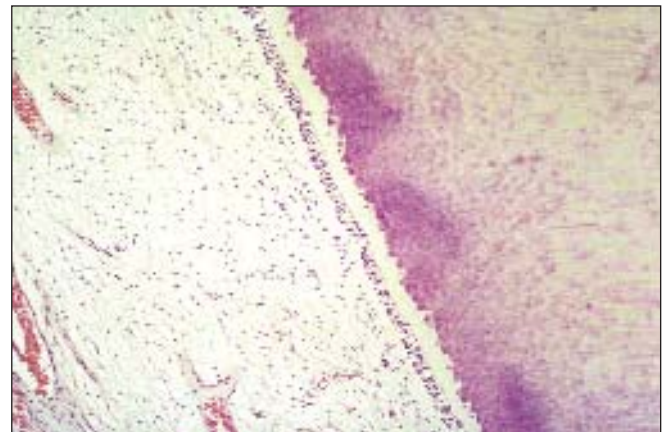


Fig. 1.5. Histology of dentine: Low power view of dentine showing dentine, predentine, odontoblasts and dental pulp. Mag. x100.

Development of dentinal tubules

Most of the odontoblast cell body withdraws towards the pulp as matrix secretion continues, but a thin and continuous tube of protoplasm called the odontoblastic process or 'Tomes' fibre remains. This phenomenon and the unique structure which develops because of it, the dentinal tubule, are central to the form and nature of dentine and determine many of its properties.

The complexity of dentine

The components of dentine are similar to those of bone, but the arrangement of the protoplasmic cell processes and the tubules in which they lie is unique (**Figure 1.4**). Unlike bone, dentine contains no blood vessels, nor does it contain the equivalent of osteoclasts, so it does not undergo cellular remodelling as bone does. The presence of collagen, mucopolysaccharide ground substance and odontoblastic processes lead to the formation of a relatively complex tissue.

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 mineralised and brittle enamel.

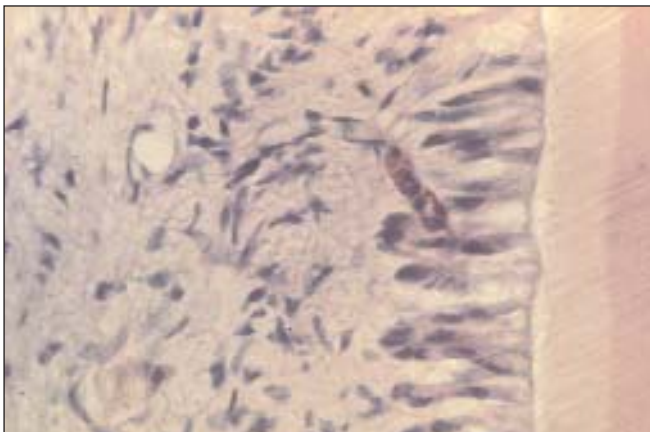


Fig. 1.6. Histology of dentine: A higher power view of the odontoblast region. Mag. x400.

Anatomy of dentine tubules

The non-calcified tubule created by the presence of the odontoblastic process extends from the dentino-enamel junction to the odontoblastic cell body which lies on the outer surface of the pulp chamber. When the dentine is completely formed this can be 5 mm or more in length (**Figures 1.5 and 1.6**). The dentinal tubules have unique characteristics. They are tapered, with the diameter near the pulp 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.

NOTE



Dentinal tubules are pathways for movement of

- fluid
- chemicals
- bacteria

Continuing maturation of dentine

The calcification of the dentinal matrix is most rapid in the months following its secretion, but the process will continue slowly throughout life. In particular, the dentine immediately adjacent to the tubule lumen becomes more heavily calcified and the tubule diameter itself decreases as more hydroxyapatite precipitates from the supersaturated dentinal fluid. The increasing thickness of the peritubular dentine increases the density of the whole tissue as the diameter of individual tubules decreases.

Odontoblasts

Odontoblasts normally remain for the life of the tooth, with their cell bodies on the inner surface of predentine and their processes extending into it (**Figures 1.7 and 1.8**). They retain their capacity to secrete matrix protein and to form additional dentine.

Secondary 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 the odontoblast comes into contact with the products of bacterial metabolism at levels below those which would kill it, i.e. in advancing caries or beneath a leaky restoration. In these circumstances the odontoblasts can lay down additional dentine relatively rapidly. This tissue is termed tertiary reparative dentine (Chapter 14).

NOTE



Dentine is a living organ and constantly changing

- primary dentine
- secondary dentine
- tertiary dentine
- constant outward fluid flow

Irregular reparative dentine

If sufficient damage occurs to kill 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 may lack the usual tubular structure but include cell bodies.

Dentine is wet

The odontoblastic tubules are full of fluid, some intracellular and some extracellular. The extracel-

lular fluid moves outward because of 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 fluid flow is much more rapid.

Factors affecting wetness

Dentinal wetness depends primarily on the size and number of the tubules, so it is wetter closer to the pulp where they are larger in diameter and more closely packed. Dentine becomes less wet with age, because of continuing peritubular dentine deposition throughout life. If the pulp dies the dentine stays wet, but outward flow is likely to be considerably reduced.

Smear layer

If dentine is cut or polished during dental treatment the tubule orifices become, at least partially, occluded with debris called 'smear layer' which consists primarily of tooth debris but also contains other contaminants such as plaque, pellicle, saliva and possibly blood (Figure 1.9). Following fracture, the tubules may become blocked by natural deposition of salivary components. Smear layer can be removed by acids, as will be described in more detail in Chapters 11 and 12 (Figures 1.10 and 1.11).

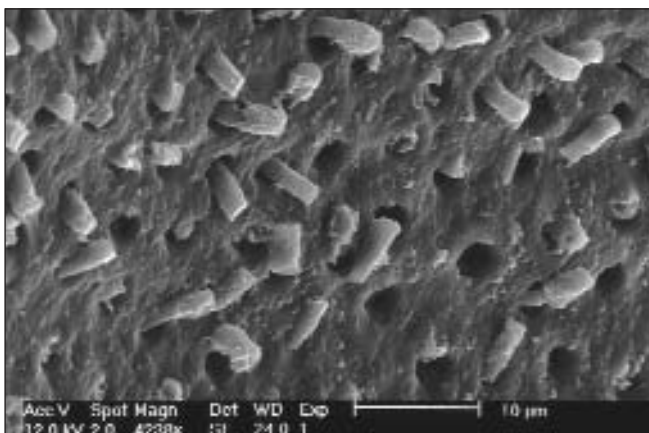


Fig. 1.7. A specimen of dentine split across the dentinal tubules. The tooth was freshly extracted so the odontoblasts have been torn apart and the ends show within each tubule. Mag. x4200. Courtesy Dr H. C. Ngo.

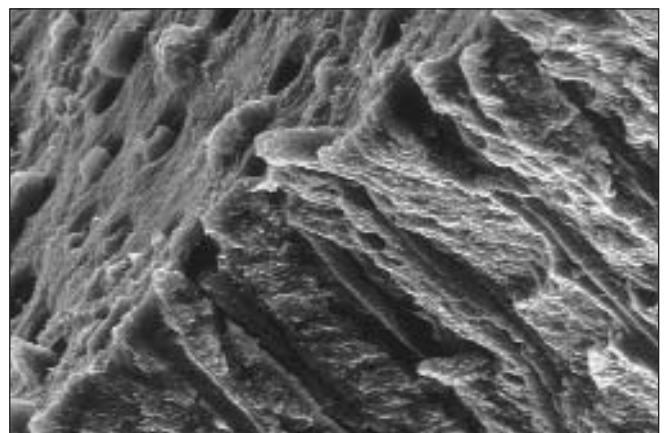


Fig. 1.8. A specimen of dentine from a freshly extracted tooth similar to that shown in Figure 1.7, split vertically along the tubules. Note the presence of the odontoblasts within the tubules. Mag. x4200. Courtesy Dr H. C. Ngo.

Diffusion through dentine

Chemicals can diffuse through the dentine tubules just as they can through any water-based medium. Dentine behaves as if it is an impermeable solid traversed by water-filled tubules. The rate and amount of diffusion is dependent on the concentration gradient, the molecular size of the solute, the temperature, the thickness of dentine, the diameter and number of tubules, and whether or not the tubules are partially blocked with smear layer.

BE AWARE



Dentine is an impermeable solid traversed by water-filled tubules



Fig. 1.9. Dentine with smear layer. Smear layer left on the surface of the floor of a cavity following cavity preparation. Mag. x800.

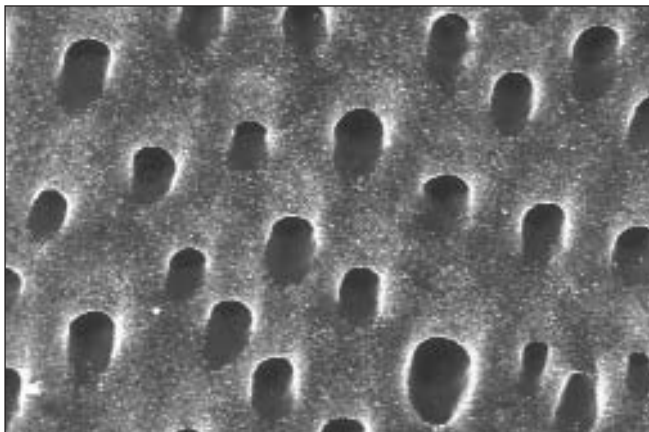


Fig. 1.10. The floor of a cavity in an extracted tooth following etching for 15 seconds with 37% orthophosphoric acid. Note the lack of smear layer and odontoblasts. Mag. x4,000. Courtesy Dr H. C. Ngo.

The natural wetness of dentine, the tubule structure and smear layer are all important factors to be considered when replacing missing tooth tissue.

Dental Pulp

Development

The growth of dentine inward from the epithelial cap slows dramatically as the tooth matures encompassing an area of tissue which is the dental pulp. The rate of dentine formation thereafter is sufficiently slow that the pulp usually remains throughout life although it becomes progressively smaller.

Constituents

The outer layer of the pulp, which is also the inner layer of 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

BE AWARE



Dentine is an extension of the pulp
Odontoblasts can regenerate

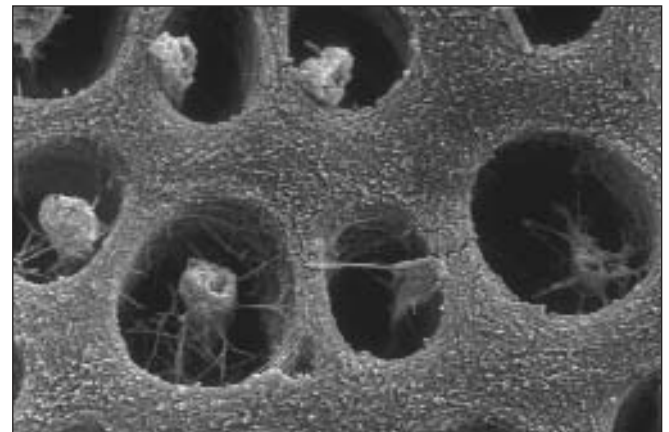


Fig. 1.11. A specimen similar to the one shown in **Figure 1.10** but the tooth has just been extracted. Note the presence of the odontoblasts that appear to be shrivelled by the etchant. Mag. x25,000. Courtesy Dr H. C. Ngo.

elsewhere, being 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 then new odontoblasts can differentiate from the pulpal ecto-mesenchyme to lay down irregular reparative dentine.

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 which causes movement of these cell bodies may trigger action potentials within the sensory nerve network. Fluid movement within the dentinal tubules therefore elicits sensation, which is interpreted as pain. Cutting dentine, drying dentine, osmotically-induced fluid flow in the tubules, heat and cold, can all cause pulpal 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 (Chapter 2). It is reasonable to propose that the rich sensory innervation of the pulp serves a protective function for the mouth. It is also of great diagnostic value in dental practice, since reported pain symptoms can give a strong indication of the presence and nature of pathological processes in dentine and pulp.

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 also helps the tissue to overcome chemical and bacterial insult. Because of the large number of capillaries present in the sub-odontoblastic layer there will be an 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 aging

With advancing age a number of changes occur in the pulp 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.

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 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 which mineralise to form root dentine.

Development of cementum

As the roots continue to form the outer surface becomes covered with cementum which is 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 adjacent ecto-mesenchyme of the dental follicle. Enmeshed in the cementum are the collagen fibres of the periodontal ligament and it is this which connects the tooth root to the adjacent bone.

NOTE



The pulp has very strong powers of recovery particularly in youth

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 organised. 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 keratinised to form 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, which is related to the gingival sulcus or crevice around the neck of the tooth,
- junctional epithelium, which forms an attachment to the enamel via a laminar structure and a system of hemidesmosomes.

As long as it is 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. The significance of the maintenance of gingival health is further described in **Chapter 17**.

Further reading

Avery, JK. *Essentials of Oral Histology and Embryology: A Clinical Approach*. St. Louis: Mosby, 1992.

Mjör, IA and Fejerskov, O. *Human Oral Embryology and Histology*. Copenhagen: Munksgaard, 1986.

Sasaki, T. *Cell Biology of Tooth Enamel Formation*. Basel: Karger, 1990.

Ten-Cate, AR. *Oral Histology: Development, Structure, and Function*. St. Louis: Mosby, 1994.

2 Disease Dynamics of the Dental Pulp

W. R. Hume ♦ W. L. K. Massey

An awareness of the events which occur in the pulp following insults enables the dentist to both protect the tissue and to provide appropriate treatment if it is damaged. Interceptive therapy may make the difference between pulp survival through healing and pulp death. Various therapies may also reduce or eliminate pulpal pain.

In very general terms, the pulp responds to damage in ways similar to other connective tissues, i.e. it can undergo various forms of inflammation, it can heal, or it can die. However the pulp is unique among connective tissues in that it is entirely enclosed in dentine and it has processes which extend throughout the dentine so that the pulp and the dentine should be regarded as a single entity

Any trauma or therapy applied to the dentine should be regarded as trauma or therapy applied to the pulp. Insults, such as the caries process and tooth restoration, are unlike those found elsewhere in the body and will challenge the pulp. It is not surprising, therefore, that some aspects of the pulpal response to insult are unique. Some therapies used to treat the dental pulp are also unique.



Insults to the Pulp

The pulp can be damaged or die in a variety of ways. The cause can be caries, microleakage, mechanical, chemical or thermal trauma, either alone or in combination.

Dental caries

Most commonly, the dental pulp can become inflamed and may die as a consequence of the advance of the caries process through dentine.¹ The process can be halted by effective treatment of the disease or through the application of preventive measures, either alone or in combination with tooth restoration.

Microleakage in restored teeth

Although the restoration of a caries defect in tooth structure is usually in the interests of the patient's health and well-being, the action of restoration may not in itself be sufficient to prevent the ultimate death of the pulp, over time, even when carried out in parallel with effective caries preventive measures. The principal reason for pulpal inflammation following restoration is microleakage, the existence of a gap between the restorative material and the dentine, in which bacteria can propagate. Evidence for this phenomenon, and the measures which can be taken to minimize it, are described in detail in **Chapter 15**.

Mechanical, thermal or chemical trauma

Direct mechanical trauma to a tooth can interrupt the blood supply by tearing the fine blood vessels at the root apex, leading to avascular necrosis of the tissue within the tooth. There is little that can be done, except to encourage those at risk of injury (e.g. contact sports players) to wear protective mouth guards.

Some pulpal cells, in particular the odontoblasts, can be killed by

- direct trauma

- by heat generated during tooth cutting
- by chemicals applied to dentine, particularly when freshly cut
- exposure of pulp tissue during cavity preparation

It is rare for the entire pulp to be killed in this way and, although these events cause pain the pulp will usually heal in the weeks after the damage occurs, unless bacteria or their products can also reach the damaged tissue.

Defence Within Dentine

Dentine has a limited capacity for its own defence. Dentinal tubules are a potential pathway for the diffusion of noxious chemicals from the external environment to the pulp and for the inward movement of micro-organisms. However, they can be reduced in diameter or totally closed by one or more of several processes.

Dentinal sclerosis

Dentinal sclerosis is the narrowing of dentinal tubules by the formation of peritubular dentine, dense calcific material laid down through an active, odontoblastic, metabolic process. Sclerosis may progress relatively rapidly in dentine beneath the advancing carious lesion or in dentine which has been exposed to the oral environment through abrasion, attrition or erosion. Sclerosis also occurs more slowly as a natural part of aging.

SUMMARY ⓘ

Dentine can protect itself through mineral deposition from

- saliva
- pulp

Calcium phosphate deposition

Crystals of calcium phosphate may also be deposited deep within dentinal tubules as a response to slowly advancing caries by a mechanism which is not understood, but which is presumably mediated by odontoblasts (**Chapter 15**).

Salivary precipitation

Precipitation of salivary calcium and phosphate can occlude dentinal tubules exposed to saliva and effectively desensitise hyper-sensitive dentine. Remineralisation will not occur in the presence of actively advancing caries or in acid erosion because of the low pH which is integral to these processes. In the presence of active abrasion or attrition there is unlikely to be precipitation but it will start again if any of these processes are arrested (**Chapter 5**).

Reparative dentine

Dentine is formed by odontoblasts and, as long as they remain alive, these cells will retain the capacity to make additional dentine in response to injury. There are other cells in the pulp which can form new dentinogenic cells if odontoblasts die, providing the rest of the pulp remains vital. The hard tissue which they form has all the constituents of dentine, but may vary in form.

Mild injury

If the injury to dentine is mild, such as attrition or the preparation of a shallow cavity, most odontoblasts beneath the damaged area survive. Regular reparative dentine, which contains relatively normal tubules, will be laid down at the pulp-dentine interface in relation to the area of damage as a continuation of the main body of dentine.

Moderate to severe injury

If more odontoblasts die because the injury is more severe, in either degree or duration, the pulpal cells can produce reparative hard tissue of widely varying types. The form will depend on the nature and the stage of differentiation of the cells which effect the primary calcific repair process. Secondary dentinogenic cells are derived by mitotic activity from the cell-rich sub-odontoblastic layer. This layer contains a high proportion of both fibroblasts and more primitive mesenchymal cells, either of which may be precursors of the replacement cells. The reparative dentine laid down in this way may be totally atubular, or may be poorly mineralised. In some cases cells in the area of calcification may become entrapped and

die, leaving substantial defects in the reparative tissue.

Chronic injury

In the chronically inflamed pulp diffuse calcification may occur, presumably because of activation, differentiation and calcific matrix secretion by mesenchymally-derived cells. Relatively well organised dentine may also be laid down within a chronically inflamed pulp to form 'pulp stones'. On rare occasions pulp stones will form in an otherwise normal pulp for no apparent reason.

Pulpal inflammation in response to bacteria

Inflammation is a series of events in vascular connective tissue which, ideally, neutralise or eliminate damaging factors and initiate tissue repair.² Inflammatory response in the pulp to bacterial insult can be identified through degenerative changes in the tissue which are not very different from changes seen in other tissues. Early changes include fibrosis and thickening of the basement membrane in associated small vessels. Frustrated repair will often induce calcific foci associated with amorphous, partially mineralised connective tissue matrix with degenerate cells and tissue.

Chronic inflammation

Whatever the microbial mix and its pathogenic vectors it is likely that the first response to caries or microleakage within pulpal tissue will be a low grade, chronic inflammation, characterised by the presence of greatly increased numbers of T-lymphocytes in the extra-vascular space. Mild chronic inflammation is usually symptomless, since the lymphocytes do not cause the release of factors which change the sensitivity of the pulp's sensory nerves. An infiltrate will form with varying numbers of lymphocytes, monocytes/macrophages and plasma cells within the pulpal connective tissue. The capillaries may become engorged and increase in number and many small vessels in the area may display features resembling high endothelial venules which are specialised for the exchange of inflammatory and immune cells.

As the inflammation progresses there may be haemorrhagic changes in relation to extensive

leukocyte infiltration. There may be extravascular haemorrhage, complete loss of connective tissue architecture and scattered chronic inflammatory infiltrate as well.

It is likely that the level of response depends to a large degree on the particular bacteria involved. In one individual there may be a small zone of dentinal caries, well removed from the pulp, which evokes a vigorous response because either the lesion contains particularly pathogenic micro-organisms or the pulpal response includes a specific immune component related to prior challenge. On the other hand, the microbial mix may be particularly benign in another patient and there may be little or no response until the lesion is large and close to the odontoblast layer. Similarly, a relatively small number of bacteria beneath a leaking restoration may evoke a chronic inflammatory response in one patient but not in another.

BE AWARE

There can be considerable variation in response between patients.

Acute inflammation

Foci of acute inflammation can develop within the chronically inflamed tissue if microbially-derived toxins damage pulpal cells, bringing about the local synthesis of histamine, bradykinin or prostaglandins. Alternatively, humorally-mediated hypersensitivity reactions to microbial components or products themselves may cause tissue damage. If such acute foci develop, the pulpal nerves may become sensitised to normal stimuli and the patient may report sensitivity of short duration to hot or cold food or drink, to cold air, or to osmotic change while eating. The pulp will not necessarily die under these circumstances and the institution of appropriate therapy may well lead to healing.

Reversible pulpitis

If, at the stage of simple, chronic inflammation or chronic inflammation with small, acute foci, the aetiological factors are removed by debridement, or denied substrate by the creation of an effective seal, the inflammation may resolve. That is to say, the inflammation is reversible. However, it is like-

ly that the pulpal tissue, after such a cycle of inflammation and repair, will be less vascular, less cellular and more fibrous than before, and may therefore be less able to withstand subsequent insults.

BE AWARE

- Bacterial insults are the most damaging
- Each insult will lead to reduced response
- Accumulated insults may lead to pulp death

Irreversible pulpitis

If the injury is more severe or there is a major immune-mediated response to the microbial challenge the tissue changes may become irreversible. The pulp may then die painlessly over time, or alternatively total necrosis may take place quite rapidly and cause considerable discomfort. If the level of stimulus remains relatively constant, such as under a leaking but otherwise stable restoration, the ability of the tissue to resist bacterial toxins will decline over time and a reversible inflammatory process may become irreversible. Some form of surgical ablation of the pulp tissue, such as pulpotomy or pulpectomy, will then be required if the tooth is to be retained.

The boundary between reversible and irreversible pulpitis is impossible to define. Remove the cause and a pulp in a healthy young patient may heal from a state of chronic, suppurative inflammation such as a pulp micro-abscess. On



Fig. 2.1. Following symptoms of pain on temperature change the restoration was removed and the pulp chamber exposed revealing a suppurating pulp. The pulp tissue is beyond recovery and endodontics is indicated.

the other hand, in older individuals or in teeth which have been subjected to previous episodes of inflammation, a pulp micro-abscess is more likely to spread because the tissue is less able to elaborate repair. In such circumstances toxic products of cell lysis may kill adjacent cells.

Prediction of outcome

Clinically it is important to develop the ability to discriminate between a pulp which might heal following conservative therapy and one which will not.³ In an adult, irreversible inflammation is characterised by symptoms of severe pain of long duration in response to hot or cold, or spontaneous unstimulated pain particularly at night. The patient may not be able to accurately identify which tooth is causing the pain, or whether it is in the maxilla or mandible.

Histological examination of the tooth responsible will generally reveal at least one pulpal micro-abscess, often in the area of a pulp horn. A micro-abscess is an accumulation of polymorphonuclear leucocytes and dead and dying pulp cells in the form of pus. The area of pus formation will be surrounded by fibrous connective tissue infiltrated with polymorphonuclear leucocytes and, slightly

NOTE



Total isolation of the lesion with an adhesive provisional restoration is the only treatment for an inflamed pulp.

further away, chronic inflammatory cells.

A diagnosis can be made on whether the inflammation is reversible or not by removing the infected layer of carious dentine or the leaking restoration and totally sealing the lesion from the oral environment with a glass-ionomer or zinc oxide/eugenol temporary restoration. If the inflammation is reversible the pain will cease almost immediately and, after a delay of at least three weeks to allow healing in the pulp, a definitive restoration can be placed.

Open-form chronic pulpitis

In the presence of a more advanced necrosis of the pulp tissue drainage may occur from the pulp chamber through the overlying carious dentine (**Figure 2.1**). The pulpitis is then regarded as ulcerative or open-form, and may not be painful. Drainage allows the development of chronic pulpitis, with the inflammatory response being confined to the superficial area (**Figures 2.2 and 2.3**). This may persist for a considerable period of time, even years, because of the development of a balance between the injurious agents and tissue resistance. At this point the elimination of drainage by the placement of a temporary or permanent restoration by a dentist can lead to severe pain, total pulp necrosis and progression to a peri-apical lesion.

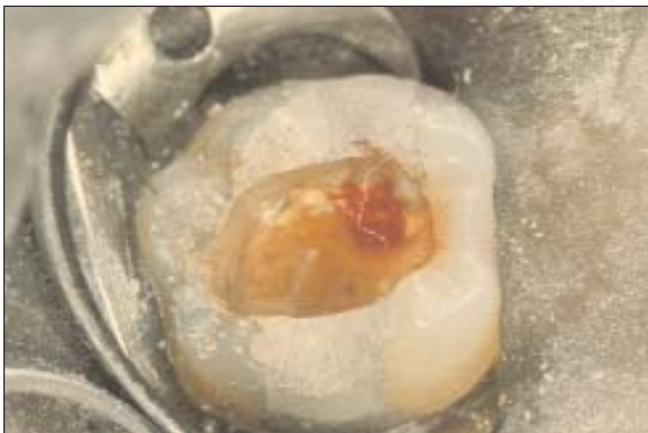


Fig. 2.2. This tooth also showed symptoms following change of temperature so the situation was investigated. The pulp was exposed and immediately showed signs of positive haemorrhage.



Fig. 2.3. The tooth shown in **Figure 2.2**. The cavity was blotted dry but the cavity immediately filled with blood once more showing that the pulp was positively and irreversibly inflamed.

Pulp polyp

In young people with untreated, gross carious lesions exposing the body of the pulp, chronic ulcerative pulpitis may lead to proliferation of hyperplastic granulation tissue into the carious cavity. The hyperplastic tissue, known as a 'pulp polyp', may have a relatively thin pedicle connecting it to the remainder of the pulp, and may be covered with a well developed epithelial layer, presumably seeded from desquamated oral epithelial cells via saliva (**Figure 2.4**).

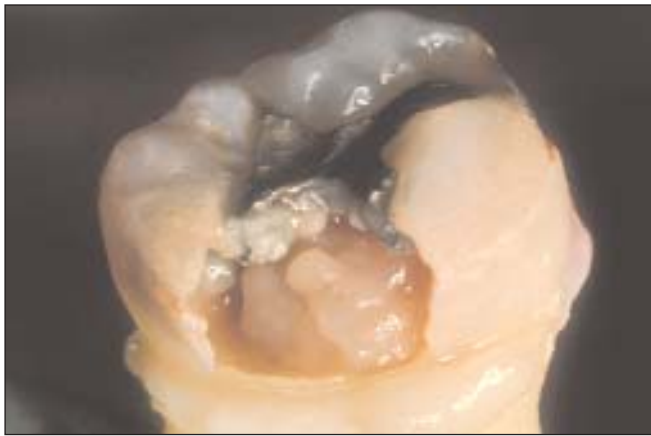


Fig. 2.4. The pulp in this tooth was exposed by rampant caries that had progressed so fast that the pulp was exposed before it could die. The inflammatory response resulted in proliferation of the tissue into a pulp polyp.

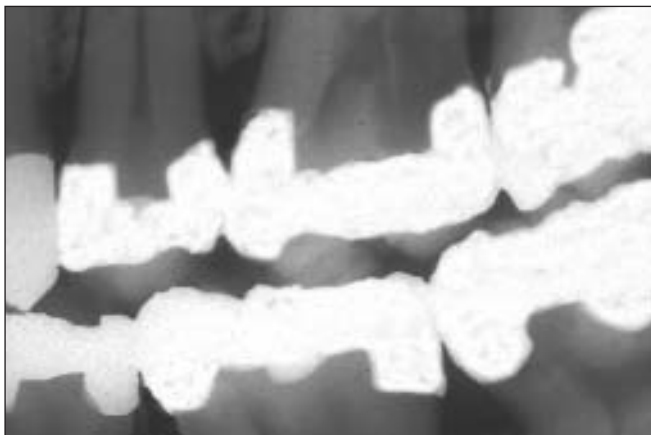


Fig. 2.5. A bitewing radiograph reveals an area of vigorous ideopathic resorption on the distal of the upper first molar beneath the restoration. As it was considered to be beyond treatment the tooth was extracted.

Diffuse calcification

Chronic pulpal inflammation may also induce the secretion of ectopic dentinal matrix by fibroblasts or undifferentiated mesenchymal cells, causing either diffuse or well organised calcification, often leading to narrowing or obstruction of the root canal.

Ideopathic resorption

In a small percentage of cases, for reasons which are not fully understood, osteoclasts may proliferate instead and cause resorption of dentine from the internal surface of the pulp chamber^{4,5,6} (**Figures 2.5 and 2.6**).

Parallel with the resorption there will be a disorganised calcification occurring so that there is a continuing destruction and rebuilding of dentine at the same time and progress will be intermittent and irregular. The resorption can commence internally within the pulp tissue or externally at the cemento-enamel junction. In both cases it is difficult to recognise in the early stages.

Ideopathic internal resorption begins within the pulp tissue, probably at the interface between the

BE AWARE



Ideopathic resorption can be

- internal
- external

identify one lesion and seek others in the same patient.



Fig. 2.6. The tooth similar to the one shown in **Figure 2.9** was sectioned following extraction to reveal the resorbed area. It would appear that the resorption commenced in relation to the cemento-enamel junction on the lingual surface so it was then classified as ideopathic external resorption.



Fig. 2.7. The upper lateral incisor shows the typical 'pink tooth' syndrome. There is an area of ideopathic internal resorption within the crown that shows first as a translucent area close to the gingival margin.

pulp and the dentine and, if it is not diagnosed early, can lead to the loss of a tooth. It is generally associated with trauma, including cavity preparation, or an external blow to a tooth. It will remain asymptomatic and the earliest signs will show radiographically as an ill-defined radiolucency in relation to the pulp chamber. Ultimately it will show as a pink 'blush' through the enamel which, on careful examination will be revealed as the pulp tissue occupying a large area of the crown (**Figures 2.7 and 2.8**). An external lesion may be found at this time at the cemento-enamel junction, often disguised within the gingival crevice. In the early stages, pulpectomy is the only available treatment but if it is allowed to progress until it reaches the external surface the tooth will probably be lost.

The alternative form of resorption commences on the external root surface and is known as ideopathic external resorption. It is sometimes associated with trauma or with orthodontic movement of a tooth and generally commences in the region of the cemento-enamel junction. In the early stages it may be successfully treated by careful debridement of the lesion and cauterising with trichloroacetic acid followed by placement of a glass-ionomer restoration. However, this lesion is very prone to recurrence along the gingival margin in relation to inflammation in the gingival tissue (**Figures 2.9**).



Fig. 2.8. A lesion similar to the one shown in **Figure 2.7** has been opened and curetted in an attempt to arrest the resorption. It will be cauterised with trichloroacetic acid and restored with glass-ionomer.



Fig. 2.9. A lower molar showing a proliferation of soft tissue in relation to an area of ideopathic resorption. It is not clear if the tissue arose from the pulp or the gingival tissue.

Inflammation in Response to Mechanical, Thermal and Chemical Insults

Reversible acute inflammation

Non-bacterial, traumatic stimuli of short duration may kill or damage a few odontoblasts only. The action of cutting dentine or over-heating it during cavity preparation, or the placement directly onto dentine of restorative materials which release toxic chemicals, such as composite

resin, may cause a simple, acute inflammatory response. The effect may be direct sensitisation of sensory nerve endings, short-term vasodilatation, a reversible increase in vessel wall permeability, followed by increased local tissue fluid pressure and, possibly, increased lymphatic flow. These effects will be mediated by the release of lysosomal proteins from damaged cells into the extracellular space. Later there may be release of histamine from mast cells and the synthesis of lysyl-bradykinin from kininogen and of cell membrane fractions following the synthesis of prostaglandins. The sensory nerve effects may include increased responses to otherwise sub-threshold stimuli such as hot or cold foods and represent the body's attempt to remove debris and initiate repair. The pain thus generated may serve no apparent physiological function.

Similarly, a mild, acute inflammatory process may be reversible if the aetiological factors do not persist. The absence of bacteria or their byproducts means the likely course of events may be relatively simple and predictable. The important variables will be the degree of cell damage and the capacity of the host tissues to elaborate inflammation and repair. Histamine and bradykinin both have a short half life, in the order of minutes only, and prostaglandin synthesis ceases when the cell membrane fractions are cleared away by phagocytes, usually within a few days. Repair involves the return to normal tissue fluid dynamics, the redifferentiation of odontoblasts and, subsequently, the deposition of reparative dentine may occur. Although the inflammatory events described above can be regarded as reversible, the pulpal tissue will subsequently be less cellular, less vascular and more fibrous. That is, it will have 'aged', in a manner similar to pulp tissue after chronic inflammation, and will also be less able to withstand subsequent insult in any form.

BE AWARE

'Aging' of pulp tissue without bacterial involvement

- repeated low level chronic insult
- isolated acute episodes

leads ultimately to pulp death

Acute inflammation leading to pulp death

More severe, non-bacterial insults causing a greater degree of cell damage or death may bring about more marked vasodilatation and the movement of substantial amounts of blood fluid and protein into the injured tissues. In the young individual healing may occur through the sequence of events described above despite the severity of the injury and the initial response. However, in an older patient or where a pulp has been compromised by previous episodes of inflammation and repair, such damage may lead to the death of the entire pulp. Cell death and disintegration may release lysosomal enzymes into the extracellular environment causing the death of more cells. The process of cell death almost always ends just short of the root apex. The periapical tissues are more able to resist damage than those of the aged or compromised pulp because they have a rich collateral circulation.

Direct pressure measurements in various areas of normal and inflamed pulps have shown that the tissue behaves as a gel, not a fluid, and that local pressure increases will not spread. The assumption that a pulp is destined to die if it suffers an episode of acute inflammation is therefore not warranted, particularly if bacteria are not involved.

Treatment of the dead pulp

Once blood supply and vitality have been lost the only predictable therapy which will allow tooth retention, in the long term, is the removal of necrotic tissue debris from the pulp space and its replacement with an inert filling material. Unfortunately, if this is not done, the tissue debris is likely to become infected at some later time, resulting in periapical infection, inflammation and pain. The anticipated mode of infection is by anachoresis, the lodgment of bacteria from adjacent tissues, or the blood supply, into the pulp chamber where they can survive and multiply.

Periapical inflammation

There is continuity between the pulp and the periodontal ligament through either the apical foramina or the lateral accessory canals. Therefore,

inflammatory processes within the pulp tend to affect the periapical or periradicular regions and induce what are generally termed periapical lesions. Most commonly, chronic apical periodontitis develops as a consequence of, and concomitantly with, chronic pulpitis of bacterial origin. Depending on the nature and number of the micro-organisms invading the dentine and pulp, this apical response can become well established while all or most of the pulp tissue is still alive.

Pulpal pain and sensation

The pulp is richly supplied with sensory nerves, many of which end close to the odontoblastic cell bodies, and will respond to stimuli such as change of temperature. Application of stimuli may also cause pain through movement of the odontoblast cells through fluid in the dentinal tubules. Finally cell damage and inflammation within the body of the pulp may fire the sensory nerves and also cause pain.

Complete loss of sensibility of the dentine and pulp usually indicates that the entire pulpal tissue is dead. However there is not an absolute link between sensibility and vitality, since patients may report apparently normal sensory responses in teeth which, on histological examination, show

no evidence of vital pulp, while others have no sensibility in teeth which are otherwise normal.

The periodontal ligament at the root apex is also well innervated. Sensory nerves within the ligament normally provide information to the brain-stem nuclei on pressure or mechanical load and tooth displacement. Such sensory information subconsciously contributes to masticatory control and may also be noted consciously as touch, pressure and pain. Inflammation in the periapical tissues decreases the critical firing threshold of the sensory nerves of the region and allows the initiation of pain by relatively minor tooth movement. Palpation through gentle movement of the tooth with finger pressure or alternatively percussion by gently tapping with a solid instrument may well elicit pain under these circumstances.

Tests of pulpal and periapical status

No one test alone can give an accurate picture of the state of the dental pulp or periapex. It is only by the correlation of all available information that the clinician can arrive at a diagnosis. A careful visual examination, using good illumination and magnification, plus a radiographic examination should be used in conjunction with the tests described below.

TABLE 2.1: Methods for testing pulpal and periapical status

Heat	Sensibility to heat may be tested by the selective application of hot water from a syringe, heated gutta percha sticks or thermostatically controlled heat applicators
Cold	Selective application can be carried out using cold water in a syringe, a stick of water-ice, solid carbon dioxide (dry ice), or ethyl chloride on a cotton pellet.
Percussion	Tap the tooth gently, vertically then laterally, with a suitable solid instrument such as the handle of a mouth mirror. Sharp pain may be elicited which may then persist for a brief time.
Palpation	Digital pressure on the tooth itself, then on the soft tissues adjacent to the root apices may elicit pain or may reveal soft or hard tissue swelling.
Electrical	Electric pulp testers may indicate the presence of viable pulp nerves but they do not give a reliable indication of the state of pulpal tissue. They should not be used in individuals with cardiac pacemakers.
Differential anaesthesia	Despite using all of these tests it may necessary to use either sub-periosteal infiltration or intra-ligamentary local anaesthesia for individual teeth to help reach a decision on which tooth is causing pain.

Further Reading

1. Kim S, Trowbridge H. Pulpal reactions to caries and dentine procedures. in: *Pathways of the pulp*. 6th Edition. Cohen S and Burns RC, editors, St Louis: Mosby, 1994.
2. Smulson MH, Hagen JC, Ellen SJ. Histopathology and diseases of the dental pulp. in: *Endodontic therapy*, 5th Edition, Weine FS, editor, St Louis: Mosby, 1996.
3. Smulson MH, Hagen JC, Ellen SJ. Pulpo-periapical pathology and immunological considerations. in: *Endodontic therapy*, 5th Edition, Weine FS, editor, St Louis: Mosby, 1996.
4. Heithersay GS. Treatment of invasive cervical resorption: an analysis of results using topical application of trichloroacetic acid, curettage, and restoration. *Quint Int* 1999 Feb; 30(2):96-110.
5. Heithersay GS. Invasive cervical resorption: an analysis of potential predisposing factors. *Quint Int* 1999 Feb; 30(2):83-95.
6. Heithersay GS. Clinical, radiologic and histopathologic features of invasive cervical resorption. *Quint Int* 1999; 30:27-37.

3 Dental Caries – The Major Cause of Tooth Damage

J. M. McIntyre

Damage to the structure and function of the tooth crown can arise from several causes. Demineralisation of the apatite mineral component leads to caries and erosion and physical factors such as wear and trauma can lead to attrition, abrasion, abfraction and fracture. This chapter will focus on dental caries, with the remaining categories of damage being covered in separate chapters.

The concepts of the cause and the progress of dental caries have changed over the last three decades. It is now realised that it is normal for a significant exchange of ions to take place between the tooth surface and the covering oral biofilm (pellicle/plaque/saliva) following every episode of eating or drinking. Demineralisation of apatite can be rapidly reversed from the reservoir of calcium and phosphate ions stored in the saliva. However, there are circumstances in which the demineralisation will exceed the body's capacity to remineralise and this will lead to an accumulated loss of the mineral content of both enamel and dentine. Surface cavitation will follow.

Dental caries is therefore identified as a continuing chronic loss of mineral ions from either the enamel crown or the root surface stimulated largely by the presence of certain bacterial flora and their byproducts. The loss will initially only be visible microscopically but it will eventually become evident in enamel as a white spot lesion or as softening of root cementum. Failure to intervene and reverse the mineral loss will lead to cavitation, with eventual irreversible bacterial damage to the dental pulp.

The profession has a responsibility to detect, control and manage the initiation and progress of this disease, and this means it is essential to understand the imbalance of intraoral factors which may lead to dental caries.



The Multifactorial Aetiology of Dental Caries

It is now recognised that there are numerous factors contributing to the initiation of a caries lesion on a specific tooth location and the following five factors have been found to exert most influence on both caries and erosion.¹

- Plaque accumulation and retention leads to increased opportunities for carbohydrate fermentation by the acidogenic bacteria contained in the oral biofilm, leading to the production and storage of organic acids at the plaque/tooth interface.
- Frequency of carbohydrate intake continues to be the major contributing factor in cases of high caries risk. Plaque bacteria metabolise carbohydrate and produce concentrations of organic acids capable of dissolving apatite.
- Frequency of exposure to dietary acids is

also considered to be a factor which can enhance the rate of both caries and erosion.

- The natural protective factors of pellicle, saliva, and to a lesser extent good plaque (free of acidogenic bacteria) plays a major role in preventing caries or limiting its progression.
- Fluoride and some other trace elements contribute to controlling the development of caries.

The stability of the oral environment in relation to tooth tissue is dependant on the maintenance of a homeostatic balance between these factors. The introduction of large quantities of fermentable, processed carbohydrates into the diets of the industrialised countries has lead to a significant imbalance of these factors. The result is higher concentrations of organic acids being present, at more frequent intervals, on the tooth surface. This means that the levels of apatite dissolution will overwhelm the protective repair systems so caries and/or erosion will result.

It is necessary to be familiar with the precise nature of each of the factors and the resultant activity which occurs on the tooth surface.



Fig. 3.1. Interaction of aetiological factors in the oral cavity

TABLE 3.1: Main contributing factors to the demineralisation-remineralisation balance

Destabilising factors	Protective factors
Diet + plaque = plaque acids	Saliva Buffering capacity
Reduction in salivary flow	Ca ²⁺ and PO ₄ ³⁻ levels
Low buffering and oral clearance	Buffering and remineralisation
Acidic saliva Erosive acids	Oral clearance proteins/ glycoproteins
	Fluoride exposure (pre- and post-eruptive)

Plaque formation and retention

Plaque is the semitransparent layer of polysaccharides which adheres strongly to the tooth surface and contains pathogenic organisms as well as some which simply thrive in the environment it creates. Plaque forms on all teeth every day, irrespective of food intake.² Many types of bacteria live in the oral cavity and some are able to colonise the tooth surface and form plaque continuously. Many bacteria rely on the pellicle, a glycoprotein film formed from saliva, to gain adherence to the enamel or exposed root surfaces. The combination of plaque, pellicle and bacteria is known as the oral biofilm. Thick plaque is held in the pits and fissures on the otherwise smooth surfaces of the crown of a tooth, between interproximal surfaces where teeth contact and around rough or overcontoured restorations. Mechanical oral hygiene procedures are not very effective in removing plaque totally from these sites, which are therefore the most common areas for caries initiation.

Several chemical solutions with antibacterial properties are able to kill up to 35% of the plaque organisms, thus partially modifying the pathogenicity of the plaque layer. However, unless the concentrations of these antibacterials can be maintained for several hours, the remnant bacteria will quickly use any further carbohydrate intake to regrow thick plaque and produce more acids.

SUMMARY

Plaque and plaque PH

There are a number of factors to take into account

- bacterial flora – *Streptococcus mutans*
- plaque retention contact areas overhangs over-contour pits and fissures sticky foods
- plaque content thickness
- salivary buffering salivary flow
- fluoride time in contact
- carbohydrate intake frequency

The role of bacteria in plaque formation and activity

Streptococci are the first bacterial species to adhere to teeth and begin plaque formation. Other species progressively infiltrate the plaque and after a few days of unimpeded growth, gram negative bacilli predominate. The most cariogenic organisms are adherent streptococci such as *Streptococcus mutans*, *Strep. sobrinus* (formerly known as *Strep. mutans* serotypes 'd' and 'g'), and the bacillus *Lactobacillus*.³ These organisms not only produce organic acids rapidly from refined carbohydrates, that is they are acidogenic, they are also able to withstand highly acidic environments, that is they are aciduric. *Strep. sobrinus* is the most rapid acid producer, though it is usually present in much reduced numbers relative to *Strep. mutans*. The lactobacillus in particular flourishes in acidic environments and is one of the predominant organisms in already carious dentine. The polysaccharides secreted by *Strep. mutans* and other plaque bacteria provide adherence to the tooth structure via pellicle and will produce further carbohydrate for bacterial metabolism when dietary sources have been exhausted.

Bacterial metabolism of high levels of refined carbohydrate in plaque can cause an immediate 2-4 point drop in pH at the tooth surface. The degree of fall depends on plaque thickness, the number and mix of plaque bacteria and the efficiency of salivary

buffering, along with other factors. Recovery to normal resting pH takes from 20 minutes for the average patient to several hours for those with a high susceptibility to caries. A very high salivary flow rate may return the pH towards neutral quite rapidly, but local retention of sticky foods may delay the rise in pH until the food is completely dissolved or removed. Carious demineralisation is proportional to the pH level and the duration of contact of low pH plaque with the tooth surface.⁴

SUMMARY

Cariogenic bacteria include

- *Streptococcus mutans*
- *Streptococcus sobrinus*
- lactobacillus – these are all aciduric/acidogenic

Frequency of fermentable carbohydrate intake

The most significant patient behaviour factor leading to an increase in caries risk is the frequency of consumption of fermentable carbohydrate. There is good evidence that it is the frequency of eating rather than the total quantity of fermentable carbohydrate consumed that causes caries.²

The mono and disaccharides are the most vulnerable to rapid fermentation, though some of the highly processed starches have also been shown to contribute to acid production. The acids resulting from carbohydrate fermentation are weak organic acids and in most cases will only cause chronic low grade demineralisation. However, when a high frequency of sugar consumption is maintained over a prolonged period, or there is a serious deficiency of natural host protective factors, caries will progress more rapidly.

BE AWARE

Sources of acids include

- fermentable carbohydrates
- carbonated soft drinks
- fruit juice
- gastric reflux

Other sources of acids

In some circumstances the addition of strong dietary food acids, or even refluxed gastric acids, will exacerbate the problem. Strong dietary acids are available from a variety of extrinsic sources such as carbonated soft drinks, sports drinks, cordials and fruit juices.⁵ Frequent or prolonged exposure to these can lead to rapid demineralisation and can turn mild caries into a rampant attack. A common example is seen in infants who are allowed to sleep suckling a bottle of fruit juice or syrup. The oral pH will drop rapidly to a very low level and may be sustained for long periods. Gastric reflux is another problem often not recognised by the patient who may think it is normal and not potentially damaging to the teeth.

Protective dietary factors

Some foods provide protective factors that may mitigate against demineralisation of the tooth surface. Plaque is less able to attach to the tooth surface in the presence of fat. Milk products, specially cheese, and some nuts fall in this category. Other foods may themselves act as buffers. Foods which require vigorous chewing can be considered protective, since they will stimulate salivary flow and, therefore, increase buffering capacity. When incorporated into the diet they can assist in returning the pH in plaque to neutrality quite rapidly.²

Salivary protective factors

Saliva plays a major role in protecting the teeth against acid challenge, as well as protecting the soft oral and alimentary tract tissues against dehydration and potential pathological irritants.¹ Around 1 to 1.5 litres of saliva are secreted into the mouth and swallowed every day. The most convincing clinical evidence of protection against dental caries is the serious and rapid damage to tooth structure which follows the sudden loss of saliva. Xerostomia can be caused by prescription and

over the counter drugs, the use of recreational drugs, from excessive exercise leading to physical dehydration, from irradiation of the salivary glands, prolonged stress or from certain medical conditions such as Sjögren's Syndrome which is related to rheumatoid arthritis (**Chapter 7**).

Other protective factors

Saliva itself is the best protection against acid attack on tooth structure and the main protective factors are

- saliva is supersaturated with Ca and HPO_4 ions so they are available to replace ions lost from the tooth surface as a result of acidic demineralisation,
- the HPO_4 ion in particular provides significant buffering capacity at resting pH and in the early stages of an acid challenge,
- pellicle – a layer of glycoproteins from saliva is part of the oral biofilm coating of the tooth surface and provides a high level of protection against acid challenge. It acts as a barrier to diffusion of acid ions into the tooth, as well as the movement of dissolution products from apatite out of the tooth. It may also limit mineralisation of apatite leading to formation of calculus from the release of Ca and HPO_4 ions from saliva once it reaches levels of supersaturation,
- bicarbonate buffering – there is a very effective bicarbonate buffering system in stimulated saliva which contributes a high level of protection against both organic and erosive acids on the tooth surface,
- salivary flow rate – salivary flow and oral clearance rates influence removal of food debris and micro-organisms. However, a high salivary flow may also dilute topically applied therapeutic agents, e.g. fluoride, resulting in the need to increase the concentration required to maintain optimal levels for tooth protection,
- fluoride ions – contribute to the overall protection and repair of the tooth mineral. Normal fluoride ion content in saliva is only 0.03 ppm on average but the level will vary following intake of extra fluoride ions from

SUMMARY



Salivary protective factors

- Ca^{2+} and HPO_4^{2-} ions
- Pellicle
- Buffer with bicarbonates
- Salivary flow
- Oral clearance rate
- Fluoride ion content

dietary sources, topical fluoride, toothpaste etc.

Salivary flow rates

Saliva provides the major source of natural protection and repair to teeth following acid challenge. Both the quality and the quantity of saliva being secreted will vary throughout the day but will be depressed during sleep. Unstimulated saliva contains little bicarbonate buffer, with less Ca ion but more HPO_4 ion than plasma. Reflex stimulation of salivary flow by chewing, or through the presence of acidic foods, e.g. citric acid, can

increase the flow by a factor of more than ten. Following stimulation, bicarbonate buffer concentrations can increase sixty times. Also Ca ion levels will increase slightly, but PO_4 ions will not increase in proportion to the flow rate.

Reduction of maximum salivary flow to less than 0.7 mL/minute may increase caries risk, although this depends on many other interacting factors (Figures 3.2 and 3.3).

Mechanism for Caries Development

Chemistry of the acid ion interaction with apatite

In order to understand the mechanism of the caries process it is necessary to understand the basic nature of the chemical reactions which occur at the tooth surface.

Demineralisation

The mineral component of enamel, dentine and cementum is hydroxyapatite (HA) consisting essentially of $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$. In a neutral environment HA is in equilibrium with the local aqueous environment (saliva) which is saturated with Ca^{2+} and PO_4^{3-} ions.

HA is reactive to hydrogen ions at or below pH 5.5, known as the critical pH for HA. H^+ reacts preferentially with the phosphate groups in the aqueous environment immediately adjacent to the crystal surface. The process can be described as conversion of PO_4^{3-} to HPO_4^{2-} by the addition of H^+ and at the same time the H^+ is buffered. The HPO_4^{2-} is then not able to contribute to the normal HA equilibrium because it contains PO_4 , rather than HPO_4 , and the HA crystal therefore dissolves. This is termed demineralisation.⁴

Remineralisation

The demineralisation process can be reversed if the pH is neutralised and there are sufficient Ca^{2+} and PO_4^{3-} ions in the immediate environment. Either the apatite dissolution products can reach

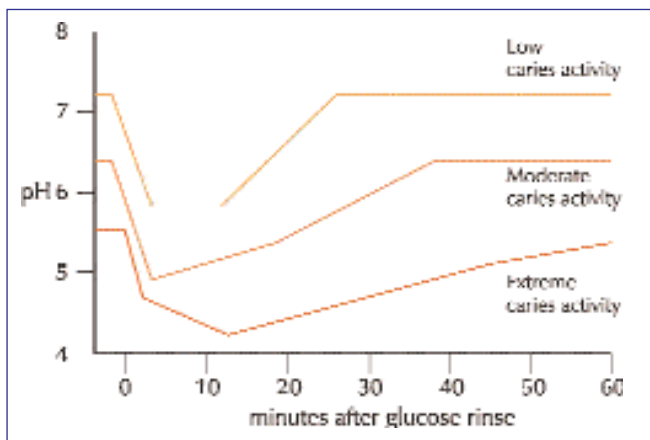


Fig. 3.2. Salivary pH after a glucose challenge: Note that with low caries activity saliva buffering is rapid and adequate. As the activity increases recovery to higher pH is slower.

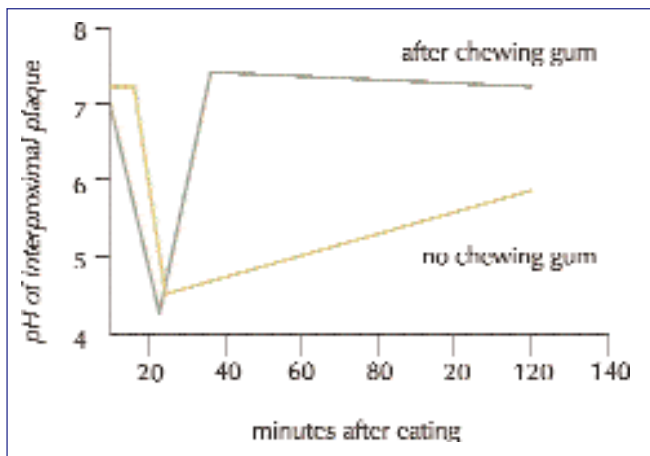


Fig. 3.3. Effects of chewing gum on interproximal plaque: Two hours after eating and not cleaning interproximal plaque still shows low pH. Chewing 2 pellets of non-sugared gum for 20 minutes after eating raises pH to normal.

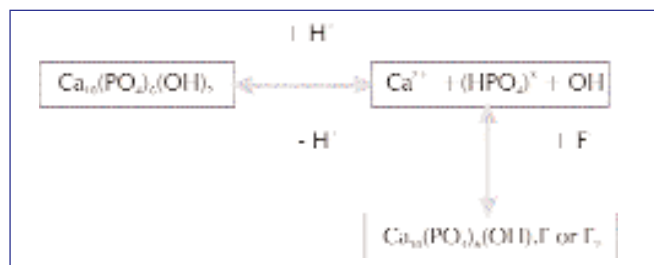


Fig. 3.4. Conversion of hydroxyapatite to fluorapatite (F₂) or fluoride enriched apatite (OH.F): The chemical reaction taking place at the tooth surface is shown.

neutrality by buffering, or Ca^{2+} and PO_4^{3-} ions in saliva can inhibit the process of dissolution through the common ion effect. This enables rebuilding of partly dissolved apatite crystals and is termed remineralisation.

This interaction can be greatly enhanced by the presence of fluoride ions at the reaction site. The overall reaction, which may be characterised as the demin/remin process, can be symbolised in general terms as in **Figure 3.4**.

The chemical basis of the demin/remin process is similar for enamel, dentine and root cementum. However the differing structures and relative quantity of the mineral and organic tissue content of each of these materials results in significant differences in the nature and progress of the carious lesion. These differences will be described later.

The progressive reaction of acid ions with apatites

Following eruption of a tooth there is a process of continuing mineralisation of enamel because of the presence of calcium and phosphate ions in the saliva (**Chapter 1**). Initially enamel apatite contains carbonate and magnesium ions but these are highly soluble in even mild acidic conditions. Thus, there will be a rapid and extensive exchange of hydroxyl and fluoride ions as the magnesium and carbonate are dissolved, leading to a more mature enamel with a greater resistance to acid challenge. The level of maturity, or acid resistance, can be great-

ly enhanced in the presence of fluoride. When a pulse of acid ions is generated at the tooth surface, regardless of the level of maturity, the general reaction may be symbolised as in **Figure 3.4**.

As the pH decreases the acid ions react, principally with the phosphates in saliva and plaque (or calculus), until the critical pH for dissociation of HA is reached at approximately pH 5.5. Any further decrease in pH results in progressive interaction of the acid ions with the phosphate groups of HA resulting in partial or full dissolution of the surface crystallites. Stored fluoride released in this process reacts with the Ca and HPO_4 ion breakdown products, forming FA, or fluoride enriched apatite. If the pH decreases below 4.5, which is the critical pH for FA dissolution, even FA will then dissolve. If acid ions are neutralized, and the Ca and HPO_4 ions are retained in this hypothetical model, the reverse process of remineralisation is able to occur as described in **Figure 3.5**.

In reality, in terms of the cycle described, there will be variation in both the level of acid ion production as well as neutralisation under differing situations in the oral cavity. Furthermore, Ca and HPO_4 ions usually diffuse to the tooth surface and may be lost, particularly in the presence of more severe levels of demineralisation. Partial replacement by salivary ions may result in remineralisation occurring in the surface layers and, over

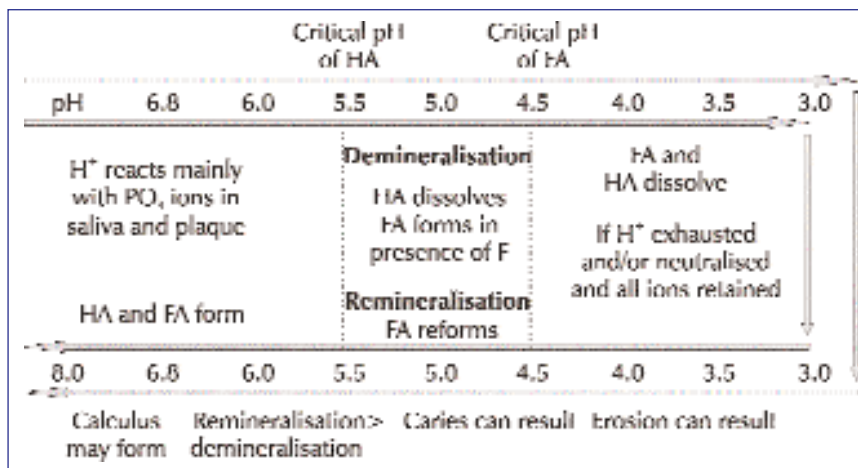


Fig. 3.5. The demineralisation-remineralisation cycle:

A conceptual chart to demonstrate the levels of pH at which the stages of demineralisation/remineralisation cycle occur.

F: Fluoride; FA: Fluorapatite; HA: hydroxyapatite.

time, even in the deeper regions of demineralisation within the lesion.

Possible sequelae

It is apparent from the pH cycle diagram, that depending on the strength of the acid present, the frequency and duration of production and the remineralisation potential in each particular situation, any one of the following sequelae can occur:

- the enamel may continue to mature – becoming more acid resistant
- chronic caries may develop – slow demin with active remin (subsurface lesion)
- rapid (rampant) caries may arise – rapid demin with inadequate remin
- erosion may occur – very rapid demin with no remin at all⁵

It has been shown *in vitro* that the chemical nature of the acid attack on the tooth surface can be rapidly modified from one causing erosion to one causing caries, and back again, by minor changes to the saturation level of acid ions with calcium and phosphate ions, or by other means.⁶

It is important for the clinician to be able to distinguish erosion from caries lesions. Erosion lesions are usually ‘cupped’ in shape with a smooth firm base, in contrast to active caries, which has a soft irregular base of demineralised collagen. Differentiation is important because erosion is far more difficult to control than caries (Chapters 4 and 5).

It is important for the clinician to identify whether the carious process is chronic or rapidly active as this will determine the degree of urgency and intensity of the control phase. Rampant caries may involve pH levels bordering on those causing erosive demineralisation and at those levels remineralisation is difficult to achieve. This means that control is much more urgent, and requires a greater range and intensity of preventive measures.

The Progressing Caries Lesion

Early enamel lesion

The initial enamel lesion results when the pH level at the tooth surface is lower than that which can be counterbalanced by remineralisation in depth, but is not low enough to inhibit surface remineralisation. The acid ions penetrate deeply into the prism sheath porosities leading to subsurface demineralisation. The tooth surface may remain intact because remineralisation occurs preferentially at the surface due to increased levels of Ca and HPO_4 ions, fluoride ions, and buffering by salivary products. This process is demonstrated in Figure 3.6.

The clinical characteristics of these lesions include

- loss of normal translucency of enamel with a chalky white appearance, particularly when dehydrated
- a fragile surface layer susceptible to damage from probing, particularly in pits and fissures
- increased porosity, particularly of the subsurface with increased potential for uptake of stain (Figure 3.7)
- reduced density of the subsurface, which may be detectable radiographically or with transillumination

SUMMARY



Summary of reactions at the tooth surface

Following any food intake there will be an acid-induced demineralisation in areas of any tooth surface that is covered by mature plaque.

Mineral loss will be reversible i.e. remineralisation will occur if

- eating frequency is low
- local fluoride concentration is high
- salivary buffering is good

On the other hand if

- eating frequency is high
- local fluoride concentration is low
- salivary buffering is poor, then demineralisation will outweigh remineralisation

This is dental caries.

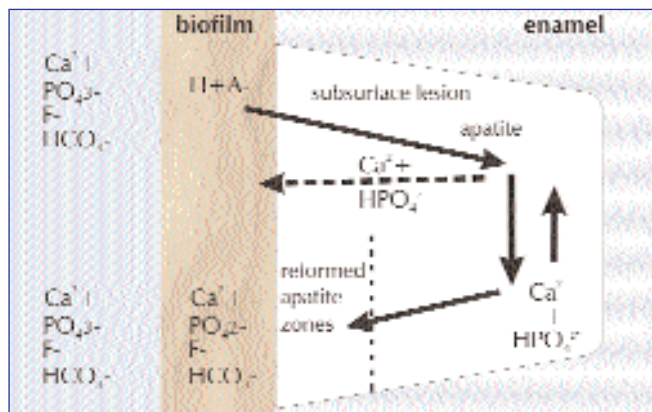


Fig. 3.6. The remineralisation cycle:

Note that the factors which favour this cycle include increased Ca^{2+} , increased PO_4^{3-} , raised pH and the presence of F⁻.

- a potential for remineralisation, with an increased resistance to further acid challenge particularly with the use of enhanced remineralisation treatments (**Chapter 8**)

The size of the sub-surface lesion may progress until the underlying dentine becomes involved and demineralised. Interproximal lesions will then become detectable radiographically. Even so, the surface of the tooth may remain intact, and the lesion may still be reversible.

In reversing incipient enamel lesions, the ideal is to regain the original density of enamel (**Chapter 8**). In reality, there may be only partial replace-

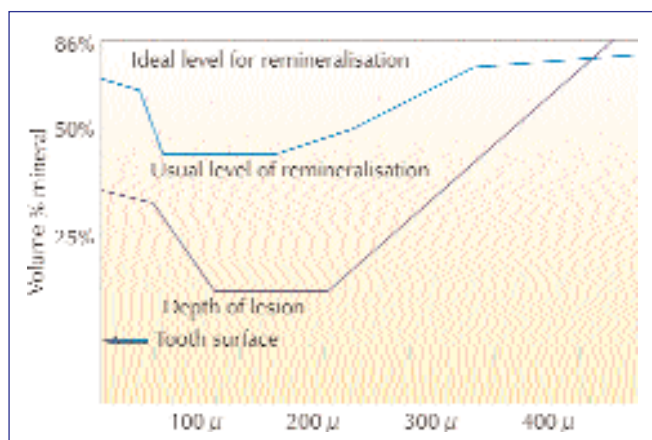


Fig. 3.8. Calcium levels following remineralisation:

A schematic concept of the amount of remineralisation which may take place in enamel following demineralisation. The level will not achieve the theoretical normal, but will be adequate to enhance the physical properties of the enamel.

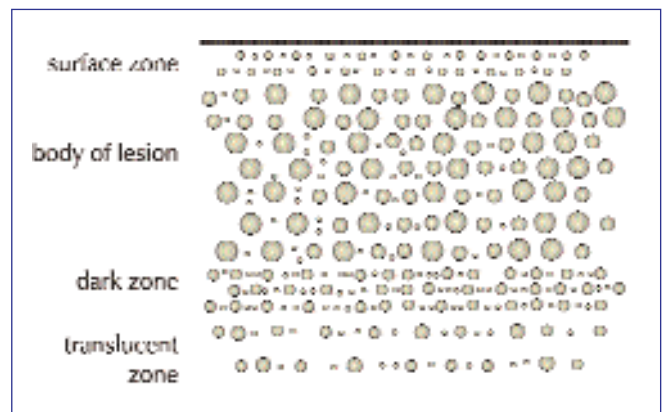


Fig. 3.7. Distribution of pore sizes in early caries lesion in enamel

ment of subsurface density. Even so the partially remineralised incipient lesion in the enamel will be more resistant to further acid demineralisation than normal enamel and physically stronger as well. Hence, it is preferable, where the patient is maintaining good home care, to observe the lesion over time rather than restore immediately and deny possible remineralisation⁷ (**Figure 3.8**).

Problems of diagnosis

It must be emphasised that assessment of the rate of progress of the lesion at both the incipient and more advanced stages is largely subjective. The best method for differentiation is to carry out a Caries Risk Assessment and the details of this will be discussed fully in **Chapter 6**. This will include an analysis of plaque retention, diet, saliva and fluoride exposure, together with a range of pertinent past and present dental and medical historical factors.

For those countries where water and/or tooth paste fluoridation have been available for some time, the overall caries profile of the community is usually significantly modified, leading to the need for a more cautious and detailed approach to the clinical diagnosis of active caries.⁸ In Australia, this has resulted in

- a change in risk profiles, with 15-30 year olds and the elderly now being at greatest risk of active caries

- a great proportion of the population remaining caries free, though a small proportion of all age groups experiences caries rates similar to those that were common prior to the introduction of fluoride supplementation
- caries being more easily hidden within dentine with little visual clinical evidence in enamel
- the need for more thorough clinical examinations of both crowns and exposed roots to identify the caries lesion at its earliest stage.
- erosion caused by frequent use of dietary erosive acids, and endogenous acids becoming a more frequent cause of tooth damage

The advancing coronal lesion

If the demin/min remin imbalance continues, the surface of the incipient lesion will collapse through dissolution of apatite or fracture of the weakened crystallites, resulting in surface cavitation. Bacteria-laden plaque can then be retained within the depths of the cavity and the remineralisation phase will be rendered more difficult and less effective. The dentine/pulp complex will become more actively involved at this point but there can still be fluctuations in the degree of activity. It is interesting to note that the pulp will produce an immediate response to invasion of acid in to the outer dentine tubules. There will be degree of mineralisation of the lateral canals that unite the dentine tubules to the extent that the properties of light transmission through the dentine will be

altered producing the so-called translucent layer. This will not be readily clinically visible but maybe revealed radiographically and can certainly be seen if all demineralised dentine is removed during cavity preparation. This is essentially a defense reaction by the pulp proving that the pulp and the dentine are one and the same organ and is capable of a degree of healing (**Chapter 14**).

Caries into dentine

Once demineralisation has progressed through the enamel into dentine and bacteria become permanent inhabitants of the cavitation they will be able to progress into the dentine itself. Demineralisation will still be driven by dietary substrate but the bacteria will also produce acid to dissolve the hydroxyapatite of the deeper dentine. Thus there will be a region of demineralisation in advance of the bacterial invasion. There may be some pioneer bacteria in, or even beyond, the area of demineralisation, but these are not clinically relevant.

Both the texture and the colour of dentine will change as the lesion advances. The texture (hardness) change is due to demineralisation. The colour will darken due to bacterial byproducts or stain from foods and beverages. In chronic lesions the colour change will be more pronounced and the floor of the cavity will be firmer in texture (**Figures 3.9 and 3.10**).

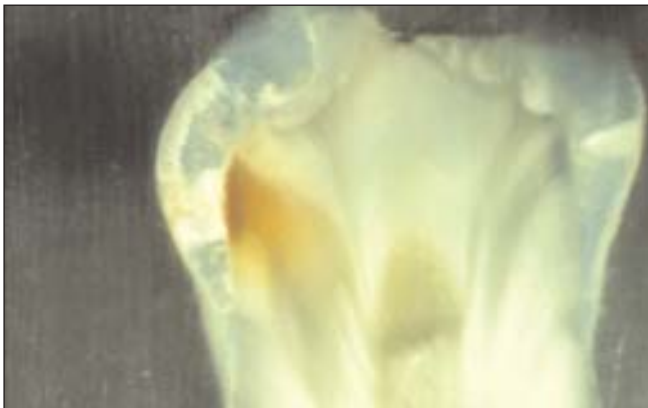


Fig. 3.9. Progress into dentine: The pattern of progress of caries into dentine. The demineralisation follows the dentine tubules downwards and inwards towards the pulp.



Fig. 3.10. Progress into dentine: Note in the proximal lesion on the left the typical penetration towards the pulp. The occlusal lesion on the right shows penetration approximately twice as deep as it is wide.

The slowly progressing lesion

If the lesion is neglected and allowed to extend through the dentine the enamel will become progressively undermined and weakened. Collapse of the unsupported enamel may eventually result in an open cavity that is relatively self cleansing and 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 which is very dark and more or less inactive.

The rampant lesion

In rampant caries the process evolves rapidly. Cavitation in enamel occurs quickly and the dentine floor of the cavity becomes softer to the touch but without significant colour change. The pulp will be at risk of irreversible damage because the remineralising and sclerosing process, which normally reduces the permeability of the tubules, will be unable to keep pace. Rapid protection of the dentine/pulp complex is essential if loss of vitality is to be avoided.

Control of the lesion

It is possible to arrest the progress of dentinal caries at any stage by sealing the cavity and isolating the bacterial flora from its nutrient dietary carbohydrate source. The remaining bacteria will become dormant and progress in the lesion will cease (**Chapter 15**).

Zones in the caries lesion

Two distinct stages of demineralisation can be observed in dentinal caries. These have been identified as the infected zone (outer layers), and the deeper (pulpal) affected zone. The infected zone is characterised by a high level of bacterial contamination, complete demineralisation of the dentine leading to total or partial collapse of the dentine tubular structure, and loss of dentinal sensitivity. The affected zone has sufficient mineral content to retain dentine tubular structure and sensitivity, even though the mineral content is partially lost. Providing there is at least 10% of the original level of mineral remaining, remineralisation is possible. This demarcation can be seen in naturally occurring root caries lesions

(**Figure 3.11**) and reproduced in artificial caries models as shown in **Figure 3.12**. The recognition of this demarcation means that a greater level of pulp protection can be achieved through retention and remineralisation of the partially demineralised dentine.⁹ This can be achieved through use of the so-called atraumatic approach – that is, completely sealing the lesion with an adhesive, bioactive cement that will arrest progress and allow a degree of healing (**Chapter 16**).

Root surface caries

Although the process of demineralisation of the root surface, that is, root surface caries, is essentially identical with the process of enamel caries there are important differences that need to be recognised. In enamel caries the early lesion is identifiable as a white spot lesion. The early root surface lesion may be very difficult to detect because there is likely to be minimal or no colour change but only a modification in surface texture.¹⁰ The mineral content of dentine is much lower than that of enamel so, when demineralised, it will rapidly expose the collagen matrix which may retain its physical structure as long as it remains well hydrated.¹¹ The exposed matrix is susceptible to physical damage but it can be readily remineralised through the repair mechanisms of saliva providing the disease is eliminated, and therefore the demin/remin balance, is stabilised. The surface of advanced root caries lesions may

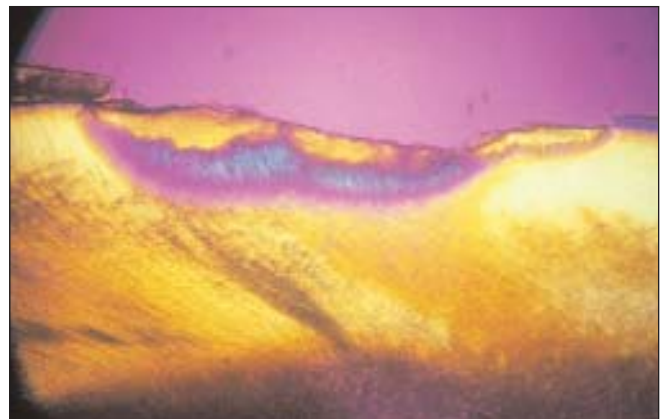


Fig. 3.11. Natural root caries lesion under polarised light: Note surface yellow zone of highly demineralised dentine. Purple zone is partially demineralised. Mag. x35.

therefore be rehardened through the application of topical fluorides or remineralising solutions (**Chapter 8**) and the progression modified or arrested. The enamel is generally not involved in the early stages but the lesion may extend up and under the cervical margin of the enamel crown as the lesion progresses (**Figure 3.13**).

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.¹² As with all dentine caries there will be an affected zone where the demineralisation is in advance of the bacterial infection. This will be a softened, demineralised, colourless zone of dentine on the floor of the cavity which should not be removed during cavity debridement because it can be sealed from the oral flora and subsequently remineralised. Sealing the surface assists the natural repair mechanisms and leads to reduced challenge to the pulp.

Identification of Caries Lesions

When G. V. Black developed a classification for carious cavities, methods for identification of a lesion were restricted to direct visual detection or tactile identification with a sharp

probe. He recognised and described the white spot lesion and suggested that, as surgery was the sole effective method of control, then it should be removed and restored. The advent of radiography in the early years of the last century allowed earlier recognition of demineralisation in the interproximal regions but was unable to define the difference between the early stages and actual surface cavitation. As a result many lesions that could have been healed by remineralisation were surgically treated using the basic principles of extension for prevention, leading to extensive loss of otherwise sound tooth structure.

In recent years methods of early identification have become more sophisticated and the profession now has the ability to be more conservative and to minimise the extent of the damage to remaining tooth structure. The use of a sharp probe was abandoned long ago because, in the presence of demineralised enamel or dentine, probing is the most efficient method of producing a cavity, even if one was not previously present. It is apparent that preparation of even the smallest cavity will structurally weaken a tooth crown so early identification and remineralisation is the preferred technique for the treatment of any lesion.

The following techniques are now available or in the process of refinement and no doubt further techniques will be developed.

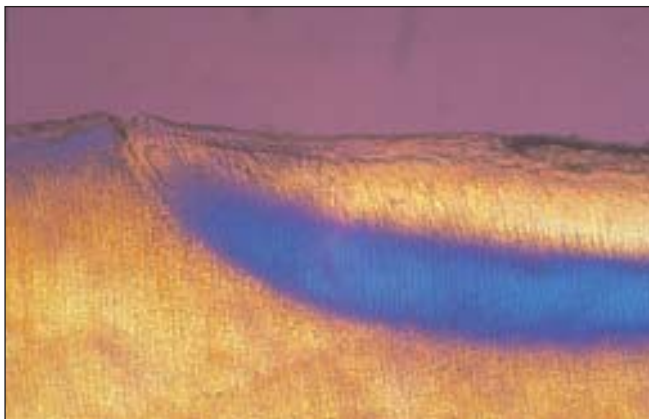


Fig. 3.12. Artificial caries lesion under polarised light: Note demarcation between highly demineralised and part demineralised zones. Mag. x100.



Fig. 3.13. Natural root caries lesion shown under transmitted light: Note it extends laterally under enamel as well as following the tubules toward the pulp canal. Mag. x35.

Conventional radiography

Basic radiographic techniques became available in the early years of the last century and were widely adopted by the profession by the 1940s. In the light of modern knowledge, exposure times for the patient were far too long with the average for a standard film being in the vicinity of four seconds. The patient was at some risk but the operator was at much greater risk of overexposure. Methods for protection of both operator and patient were soon adopted.

Today standard exposure times are greatly reduced to less than one second per film and surrounding protection methods mean that all concerned are relatively safe. Definition with standard techniques, properly applied, is adequate for diagnostic purposes and, when properly developed, the archival value of the film is high.

Digital radiography

The main area of progress in radiography has been the introduction of digital techniques. These systems are not yet in wide use but they hold promise for the future.¹³ It is generally agreed that, at present, the value of the image for diagnostic purposes is not quite as high as the conventional radiograph but results are improving. The machines involved are also improving with a wireless machine, that is, a machine free of all wire connections, that uses radio waves, already available.

A number of variations in the software programmes are being tested for their ability to enhance the image and their use may well become universal.¹⁴ One of the main advantages is that the exposure time is reduced to fractions of a second thus offering a high level of safety for both operator and patient. Also the image appears immediately on a computer screen where it can be manipulated, enhanced, controlled, transferred to another screen and permanently stored.

Laser fluorescence

Laser light can be used in the visible region (blue or red) as a tool for the detection of carious lesions. Techniques developed to date for early detection by laser light rely on natural fluorescence from the tooth material or from bacterial

byproducts and these are proving to be reasonably reliable and compare favourably with radiographs.¹⁵ However, some authors report a wide variation between operators so that a degree of prior training is desirable.¹⁶ An interesting variation on this methodology has been reported which may have value in treatment of the incipient occlusal lesion.¹⁷ Laboratory studies have shown that specific laser irradiation that is absorbed strongly by the carbonated hydroxyapatite mineral of the teeth can briefly heat a thin layer at the surface, altering its composition and making it strongly resistant to subsequent acid attack in the caries process. This resistance leads to major inhibition of subsequent subsurface caries progression and shows promise for the treatment of susceptible sites on the tooth surface such as pits and fissures.

Quantitative Laser-induced Fluorescence (QLF)

A variation on the use of laser energy to detect caries is in the process of development using quantitative laser-induced fluorescence and this also is based upon the natural fluorescence of tooth structure.¹⁸ Currently the method is being tested *in vitro* and *in vivo* and the advantage appears to be an ability to assess both demineralisation as well as remineralisation levels in a white spot lesion.¹⁹ The added advantage of assessing the progress of healing a lesion is obvious but it may be limited to readily accessible lesions only.

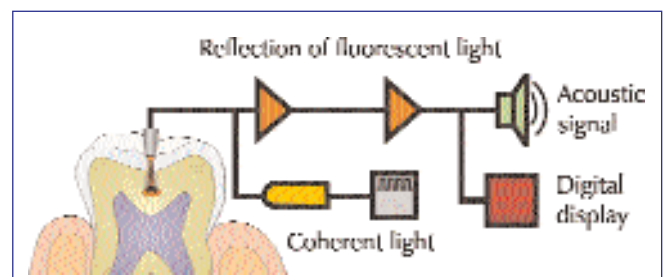


Fig. 3.14. Laser fluorescent machine: A diagrammatic illustration of the function of this machine. Note fluorescence is reflected back to the machine and recorded as a number which is simply indicative and not finite.

Limitations

It is acknowledged that all the above techniques have some essential value but it is important to note that none of them should be relied upon for making a final diagnosis. Early recognition of the interproximal lesion is generally undertaken using radiographs. It is noted elsewhere that, in the average patient, progress of a caries lesion through proximal enamel can take a considerable period of time. The earliest white spot lesion will not be available for direct recognition on the contacting surfaces. The early stages of demineralisation of dentine will show clearly beneath a patch of radiolucent enamel. However, there will not necessarily be surface cavitation at that stage and, with proper use of remineralising techniques (Chapter 8), the lesion may still be healed. Actual surface cavitation should be the determinant, rather than radiolucency, because it is only at that stage that plaque can no longer be reliably removed routinely and progress of the lesion arrested. In other words the operator should not rely solely on radiographs or lasers to make a final clinical decision.

On the other hand it must be recognised that the occlusal fissure lesion presents yet another problem. As shown in Chapter 14, a fissure has a very complex anatomy and it varies in depth and width without any external evidence. In a high percentage of cases it will narrow down to a width of 200 μ or less when close to the occlusal surface and then open out again down near the cemento-enamel junction. Plaque can gain access to the depths and, where there is high caries activity,

demineralisation will occur at the lower levels and early detection is difficult. Direct pressures applied through mastication will tend to compress plaque further into the depths and its removal is impossible.

Identification of demineralisation within the subjacent dentine is difficult with a radiograph but is now becoming possible with a reasonable degree of accuracy using instruments producing laser fluorescence or similar techniques. However, it is emphasised again that the presence of caries activity within dentine does not, of itself, mean that surgical interference is justified. It has been shown that placement of a seal that is proof against microleakage is sufficient to arrest all progress of the lesion for long periods.²⁰ It is suggested therefore that, in the presence of a medium to high level of caries activity, placement of a sealant could be readily justified even if dentine involvement is already suspected. This should then be kept under observation and if, in the future, there appears to be further activity, surgical intervention may be justified.

The above comments are made, not to denigrate rising technology, but to emphasise the advantages that can accrue from a conservative approach to the treatment of caries. All of the above statements are predicated upon the assumption that every effort has been undertaken to eliminate the active disease in the first place and in recognition that surgery is not a cure for caries. Surgery is only required to overcome the damage that the disease has caused.

Further Reading

1. Kidd EAM and O Fejerskov, eds. *Essentials of dental caries; the disease and its clinical management*. Copenhagen: Munksgaard, 2003.
2. Murray, JJ, ed. *The prevention of dental disease*. Oxford: Oxford University Press, 1989.
3. Marsh P, Martin, MV. *Oral Microbiology*. Oxford: Wright Publishers 1996.
4. Thylstrup and Fejerskov, eds. *Textbook of clinical cariology*. Copenhagen: Munksgaard, 1994.
5. Lussi A, Kohler N, ZeroD, Schaffner M, Megert B. A comparison of the erosive potential of different beverages in primary and permanent teeth using an in vitro model. *Euro J Oral Sci* 2000; 108: 110-114.
6. Larsen MJ. Dissolution of enamel. *Scand J Dent Res* 1973; 81:518-522.
7. Ten-Cate JM. *In vitro* studies on the effects of fluoride on de- and remineralisation. *J Dent Res* 1990; 69 (Special Issue): 614-619.

8. Weatherell JA, Deutsch D, Robinson C and Hallsworth AS. Assimilation of fluoride by enamel throughout the life of the tooth. *Caries Res* 11; Suppl. 1; p 85, 1977.
9. Massler M. Changing concepts in the treatment of carious lesions. *Br Dent J* 1967; 123:547-548.
10. Nyvad B, Fejerskov O. Root surface caries; Histology and microbiological features and clinical applications. *Int Dent J* 1982; 32:312-326
11. Symposium – Exposed root interactions. *Am J Dent* 1994; 7:225-296.
12. Featherstone JDB, McIntyre JM, and Fu J. Physio-Chemical aspects of root caries progression, In *Dentine and Dentine Reactions in the Oral Cavity*, eds, Thylstrup A, Leach SA and Qvist V, 1987, Oxford: I R L Press, 127-137.
13. Miles DA. The deal on digital: the status of radiographic imaging. *Compend Contin Educ Dent* 2001; 22: 1057-1062.
14. Gakenheimer DC. The efficiency of a computerised caries detector in intraoral digital radiography. *J Am Dent Assoc* 2002; 133:883-890.
15. Lussi A, Megert B, Longbottom C, Reich E, Francescut P. Clinical performance of a laser fluorescence device for detection of occlusal caries lesions. *Eur J Oral Sci* 2001 Feb; 109(1):14-9.
16. Fung L, Smales R, Ngo H, Mount GJ. Diagnostic comparison of three groups of examiners using visual and laser fluorescence methods to detect occlusal caries in vitro. *Aust Dent J* 2004; 49:67-71
17. Featherstone JD Caries detection and prevention with laser energy. *Dent Clin N Am* 2000; 44:955-69.
18. Heinrich-Weltzien R, Kuhnisch J, van der Veen M, de Josselin de Jong E, Stosser L. Quantitative light-induced fluorescence (QLF) – a potential method for the dental practitioner. *Quint Int* 2003; 34:181-8.
19. al-Khateeb S, Oliveby A, de Josselin de Jong E, Angmar-Mansson B. Laser fluorescence quantification of remineralisation in situ of incipient enamel lesions: influence of fluoride supplements. *Caries Res* 1997; 31:132-40.
20. Mertz-Fairhurst EJ, Smith CD et.al. Cariostatic and ultraconservative sealed restorations: six year results. *Quint Int* 1992; 23:827-838.

4 Preventive Management of Dental Caries

J. M. McIntyre

Every dentist carries the ethical responsibility to not only treat active caries lesions, but also to ensure that each patient learns how to control any further caries activity. This can be achieved most effectively when the dentist is able to determine the nature of the imbalance of factors leading to caries, and can persuade and advise each patient of strategies to prevent or reverse the imbalance. Success rewards both dentist and patient, though failure of the patient to comply and control the problem can result in severe frustration for both.

There are a number of methods available to assist the patient to break the cycle and this chapter will review those methods that will assist the operator to devise a suitable regime for each individual patient.



The Most Effective Approach to Prevention

As caries has a multifactorial aetiology, it is generally necessary to adopt a multifactorial approach to prevention. As discussed in **Chapter 3**, the most common factors causing demineralisation of tooth structure are

- excessive frequency of fermentable carbohydrate in the diet
- excessive frequency of low pH in saliva, extrinsic or intrinsic
- inadequate plaque control
- deficiencies in salivary protection
- inadequate exposure to fluoride ions
- failure to control the microflora

The significance of each of these items will vary in each patient so the advice and treatment regime will need to vary.¹ For example, an excessive intake of fermentable carbohydrates will be the dominant aetiological factor for the majority of young patients. However, inadequate plaque control accompanied by a lack of fluoride may be more significant for one patient while loss of salivary flow in the presence of an acceptable diet could be the key factor in another. This means that, to minimise the level of demineralisation and enhance the level of remineralisation, it is necessary to help the patient by offering advice, relevant to their particular circumstances. In other words the hierarchy of aetiological factors must be understood for each patient.

Assessing Dietary Factors in Caries Development

This is the most common and significant cariogenic factor. If acid ions are persistently produced in plaque from an excess of refined carbohydrate, they will exhaust the buffering capacity of the saliva, and the remineralising process will no longer effectively counteract the demineralis-

ing factors. Assessment of the diet requires a motivated, cooperative patient prepared to record detailed dietary routines on each day of a three day diet chart.² The most fermentable carbohydrates are the mono and disaccharides, though highly processed starches have been shown also to be major contributors. In excess of four exposures of fermentable carbohydrates per day is considered potentially cariogenic. However, this is a relative concept and it must always be considered alongside all other demineralisation promoting factors before its full significance can be determined.

Measures to improve diet

It is necessary to carefully assess the contents of the diet to determine the most cariogenic item/s so that alternatives can be recommended. Sugar substitutes are available³ or it may be sufficient to just reduce the frequency of inclusion of the most fermentable carbohydrates. Long term modification of the diet is difficult to achieve and frequent monitoring of the outcome will be required for success in achieving change in both the short and long term. Assistance of the entire dental team will make success more achievable.

Extrinsic and intrinsic acid

The second aspect of the food intake to be assessed is the extrinsic acid content. This is usually present in beverages such as carbonated drinks, fruit juices. A high intake of these may significantly increase the concentration and strength of acid ions on the tooth surface sufficient to hasten demineralisation. In addition, if vigorous tooth cleaning is undertaken immediately after ingestion of these liquids there is likely to be erosion of already demineralised tooth structure. The inclusion of sugar substitutes in acid beverages will not reduce demineralisation because of the intrinsic low pH.

Intrinsic acid will arise from gastric reflux, regurgitation, frequent vomiting and problems such as bulimia. These are often difficult to diagnose and may require the involvement of other health professionals.

Evaluating and Improving Oral Hygiene

A number of studies have shown that, unless tooth brushing is carried out efficiently, it achieves little reduction in caries development. This means that a thorough assessment of plaque control with tooth brushing is essential before advising each patient on modifications required to achieve success.⁴ The following should be noted:

- suitability of the brush and potential difficulties in its use. Patients with muscular or arthritic difficulties may find electric tooth-brushes more effective
- method of application of the brush
- frequency and time taken for brushing
- the routine use of a fluoridated dentifrice significantly increases the benefits of tooth brushing
- disclosing systems can assist both dentist and patient in assessing the effectiveness of daily plaque control routines

The first oral hygiene routine should be carried out in the morning either before or after breakfast. The object is the removal of plaque rather than the elimination of food debris so cleaning immediately before eating is just as effective as cleaning

after. In fact, if breakfast is to include a low pH drink, such as orange juice, cleaning before will reduce the potential for mechanical erosion of demineralised root surfaces. The second oral hygiene routine should be carried out just before retiring for the night. During sleep the salivary flow virtually ceases and any available buffering capacity is lost. Therefore removal of all plaque should be completed with diligence and any prescribed preventive medicament, such as topical fluoride or chlorhexidine, should be applied at this time. In the absence of plaque the fluoride

will be taken up into the tooth structure more effectively and the subsequent lack of saliva will be of no consequence.

Need for more frequent daily cleaning

In the presence of rampant caries oral hygiene routines should be undertaken either before or after each food intake to encourage the patient to recognise the important part played by fermentable carbohydrates in the caries process. A fluoridated dentifrice **must** be used because maintenance of fluoride on the tooth surface is highly desirable.

Additional cleaning aids

Where a high level of plaque control is essential, patients should be advised in the correct use of dental floss or other interdental cleaning aids, whichever is most acceptable to the patient. There are many therapeutic mouth rinses available designed to reduce oral bacteria and the most effective of these contain chlorhexidine gluconate.⁵ Care should be taken to avoid frequent use of mouthrinses containing high concentrations of alcohol, particularly with patients with reduced salivary protection because alcohol can contribute to further dehydration of the mucosal tissues and exacerbate the problem.

Recent evidence suggests that a mouthrinse of 10% povidone-iodine⁶ can cause a significant reduction in salivary bacterial counts, particularly in children.

Evaluating and Enhancing Salivary Protective Factors

Deficiencies in salivary protection are generally a result of depletion in salivary secretion. Clinical and visual clues to assist in the detection of xerostomia include

- visual evidence of dry oral mucosa
- patient may be seen to lick their lips frequently
- patient reports that they have to sip fluids frequently
- patient with a high caries rate appears to

NOTE



Tooth brushing is to remove plaque – not food debris

- clean before eating or
- clean after eating
- **most important** – clean before retiring

have a normal noncariogenic diet and effective oral hygiene

- patient routinely uses medications that cause hyposalivation
- some medical conditions cause xerostomia, e.g. Sjögren's syndrome, rheumatoid arthritis etc. (Table 4.1)

It is recommended that salivary tests be conducted for any patient where xerostomia is even a remote possibility. The main parameters to assess are briefly described here and will be discussed later in Chapters 6 and 7.

Salivary parameters to be assessed

- Flow rate: assess stimulated saliva where flow rate has been increased by chewing gum or citric acid contact with the tongue. Normal flow rate is 1.5-2.5 mL/min. Less than 0.7 mL/min is considered xerostomic
- Buffering capacity: assess using commercial tests which determine the pH reduction achievable by saliva
- Bacteriological tests: estimate the quantity of *Strep. mutans* or *Lactobacillus* in saliva. Alternate test methods:
 - a) culture bacteria on a selective agar medium for 48 hours
 - b) antibody tests for rapid estimation of bacterial counts
- Acidogenicity tests: an innovative impression material containing pH indicators to demonstrate sites of low pH around the teeth.

Most of these tests will indicate potential caries

risk but must be considered with all the other evidence. Control of a high caries rate is still possible if the patient takes extreme care with diet and oral hygiene and makes maximum use of topical fluoride. However, in the presence of a dry mouth, patients will often seek comfort by eating sweets or drinking sugared drinks more frequently, seriously exceeding the buffering capacity of the saliva. Even a mildly cariogenic diet might result in some caries without other protective action.

Causes of hyposalivation

A number of factors can contribute to salivary protective deficiency including the fact that a few patients have a genetically determined low level of salivary flow.⁷ The basis of this is not understood. The most common factors which are known to cause hyposalivation are:

- Mood altering drugs such as tricyclic antidepressants and anti-Parkinsonian drugs. Nonprescription psychotropic agents such as marijuana can produce a similar effect. Where there is severe salivary reduction resulting from a particular prescribed drug, it may be possible to try an alternative. However, changing drug routines and altering the balance of a prescribed series is often a long term, complex process and should be undertaken only with the cooperation of the other health professionals involved. Modification may be justified if the caries rate is excessive.
- Radiotherapy of the head and neck region. While care is taken to avoid the salivary glands it may be impossible to carry out effective radiotherapy without severe damage. Xerostomia may reach a peak within six weeks of commencement of radiotherapy. A slight increase of flow may then gradually occur but severe xerostomia may persist for many years.
- Rheumatoid conditions such as Sjögren's syndrome leads to severe depletion of excretion from all secretory glands including salivary, lacrimal etc.
- Other medical conditions such as uncontrolled diabetes or extreme stress can lead to salivary depletion.

TABLE 4.1: Reduced Salivary Flow

Drug-induced	antihypertensives anticholinergic anti-Parkinsonian psychotropic sedatives
Anxiety	severe emotional disorders
Medical complications	diabetes, malnutrition glandular infection or obstruction, radiation of head or neck (>70 Grays in six weeks = total xerostomia) Sjögren's syndrome

BE AWARE**Xerostomia**

- Stimulated flow rate below 0.7 ml/min
- Generally drug-related
- Not related to age

Enhancing salivary protection

Enhancement of salivary flow may be difficult, particularly when it is caused by systemic disease. Chewing sugar free gum may have a limited positive effect. Prescription of low levels of pilcarpine, administered intraorally, will increase flow rates but patient reaction varies. Some show a degree of allergy. Prescription sialogogues often contain citric acid and this will lower the intra oral pH thus increasing the risk of caries rather than modifying it.

BE AWARE

Sustained enhancement of salivary flow is difficult to achieve safely – for preference eliminate the cause of dysfunction.

Xerostomia alleviating products

A number of therapeutic products will alleviate the discomfort of xerostomia. There is an artificial saliva available which contains a variety of electrolytes normally present in saliva, has a similar viscosity and can therefore provide short term comfort.

There are a number of gels for application to oral mucosa that assist with moisture preservation for limited periods. These will improve infection control and replace essential electrolytes. The foaming agent, sodium laurel sulphate, normally present in toothpaste, may cause irritation of the dry oral mucosa, in which case, toothpastes without this agent are available.

Compensating for diminished salivary protection

In the presence of xerostomia it is essential to provide compensation for the loss of the protective factors. The following alternates should be considered, alone or in combination:

- topical fluoride: see **page 42** for various prescriptions

- chlorhexidine gel applied once or twice per day for limited periods either as a mouth-wash or a gel (see **page 44** for prescriptions)
- replacement of calcium and phosphate ions with calcium phosphopeptide (CPP) preparations, available in a variety of forms, from lozenges to topical paste (**Chapter 8**).

Function and Prescription of Fluorides

Following the discussion in **Chapter 3** it is necessary to consider the effect of fluoride on the initiation and progress of the caries lesion and then consider levels of prescription. It is a little over 50 years since the role of the fluoride ion began to be appreciated. This led to efforts to supplement its contact with teeth either by increasing the fluoride content in the diet through the artificial fluoridation of drinking water, incorporating it in toothpaste or applying it directly to the tooth surface. Adding it to the water supply leads to the so called pre-eruptive effect through incorporation of higher concentrations into the apatite structure of developing teeth. This means it is immediately available during an acid challenge to inhibit apatite dissolution and enhance any potential remineralisation.

A topical application will provide a post-eruptive effect because it will increase the fluoride concentration on the tooth surface and it can be stored in plaque. Thus it is immediately available to inhibit demineralisation of the surface apatite and, following demineralisation, it will enhance repair and remineralisation in conjunction with the calcium and phosphate ions present in saliva.⁸

NOTE

The most important aspect of fluoride supplementation: it helps to control caries for 50-80% of people exposed to it even though the amount and frequency of refined carbohydrate in the diet remains unchanged.

The presence of fluoride raises the tolerance of the oral balance to increased amounts of refined carbohydrate, making it more difficult for apatite to demineralise, and if it does occur, then enhancing remineralisation.

How does it work?

In general terms, fluoride works in three ways. It slows down the development of a caries lesion by inhibiting the demineralisation process. It increases the resistance of enamel to acid attack and enhances the normal remineralisation process by preferentially reacting with hydroxyapatite to form fluorapatite or a fluoride enriched apatite. Finally, at high concentrations it can inhibit bacterial metabolism.

However, it is important to note that, in an acid environment, the fluoride ion reacts strongly with free Ca and HPO_4 ions, forming fluorapatite (FA) crystals $[\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})\text{F}_2]$. These crystals are less soluble than pure HA because of better subunit stacking and are therefore more resistant to dissolution by acid ions above pH 4.5. This is the critical pH for FA in contrast to HA where the critical pH is 5.5.

SUMMARY



Fluoride

Reacts directly with enamel and dentine and produces several effects.

- Forms fluorapatite which is less soluble than hydroxyapatite
- Inhibits demineralisation
- Enhances remineralisation
- Inhibits bacterial metabolism
- Reduces 'wettability' of tooth structure
- Inhibits plaque formation

Optimal levels of fluoride

Fluoride ions have to be at the site of an acid attack on the tooth surface to be effective. This is best achieved either through incorporation of fluoride into tooth structure during its development or frequent daily contact with low concentrations of fluoride ion on the tooth surface.⁷ The optimal level to achieve control will vary for each person depending on the level of acid ions present and

the level of protective agents. Resting phase saliva may contain around 0.03 ppm fluoride ion, and even this concentration has been found to result in areas of inhibition of demineralisation in *in vitro* experiments.⁷

In healthy teeth without fluoride supplementation, there may be up to 2,500 ppm fluoride ion present in enamel and dentine.⁸ These ions are obtained from a variety of food and beverage sources including sea foods and beverages like tea and beer that contain high concentrations. However, this is not sufficient to cope with the elevated concentrations of plaque acids resulting from frequent refined carbohydrate consumption in the modern diet.

Supplementation will increase the fluoride ion concentration to around 4,000 ppm or more throughout tooth structure. This helps to increase the resistance to acid challenges sufficiently to reduce caries rates, on average, by 60% in children, with a benefit being experienced across all age groups.

Regular use of a fluoridated toothpaste (1,000 ppm fluoride ion) has been shown to reduce caries prevalence, even without supplementary dietary fluoride. Initial data suggested this is in the order of a 30% reduction over a two-year period, though long term epidemiological data shows an increase with time to a level equivalent to or higher than for water fluoridation. Obviously, used together, both forms of fluoride provide the optimal benefit for the majority of the population.

Increasing fluoride exposure

The greatest benefit of fluoride supplementation has been in caries prevalence in children, with slightly lower reductions across the adult population. However, there remains a small proportion across all age groups who will continue to have a high caries rate despite access to fluoride supplementation. It is not possible to increase concentrations stored in tooth structure by dietary means alone as the maximum intake is 1.0 mg/day. However, the level of fluoride at the tooth surface can be increased by the use of frequent applications of concentrated forms in vehicles such as mouthrinses, gels or varnishes. The level of fluoride in such preparations range from 1,000

ppm in some mouthrinses, gels and varnishes to 12,300 ppm in APF gels, and 26,000 ppm in one varnish. Applications will increase uptake levels into the surface tooth structure, and store excess fluoride ion as CaF_2 around the apatite crystallites. This may lead to heavy remineralisation at the surface of enamel lesions but the fluoride ion may not be able, initially, to penetrate more deeply into the subsurface body of the lesion. Subsequent acid challenges will progressively ionise this layer to permit free fluoride ions to penetrate more deeply. However, even the additional CaF_2 is quickly lost in the acid environment found in the highly caries active patient and needs to be replenished more frequently to be effective.

This suggests that the availability of fluoride supplements alone will not be sufficient for some patients and auxiliary chemical means of overcoming demineralisation would be desirable. Such means are discussed in detail in **Chapter 8**.

Effect on established lesions

The fluoride ion will not only prevent initial lesions developing, but will also stabilise established lesions. Fluoride can

- contribute to remineralisation of incipient enamel caries
- partly remineralise carious dentine and thus slow down or arrest the caries process in the cavitated coronal lesion
- remineralise root surface lesions to the extent that restoration may not be necessary

Topical fluoride is more effective in inhibiting smooth surface caries. It is less effective in fissure or interproximal caries because of the difficulty of removing stubborn or mature plaque. Daily application of topical fluoride to demineralised root surfaces over a period of 2-4 months will lead to significant hardening of the exposed dentine indicating that a remineralising balance has been established. The surfaces of such remineralised lesions can become glass like in texture, as a result of this hypermineralisation.

TABLE 4.2: Vehicles for topical fluorides

Fluoride containing dentifrices

- Usually as NaF (1.0%), Na_2FPO_3 (0.76%) or SnF_2 (0.4%) (concentration of fluoride ion by weight).

In general there is approximately 1 mg/g of available fluoride (1000 ppm).

A toothbrush completely covered in paste holds approximately 1.5 mg of fluoride.

Recent developments include

- a low fluoride toothpaste for young children (400 ppm)
- a 5000 ppm dentifrice for high caries risk adults (not recommended for children)

Concentrated gels

- APF 1.23%: contains approximately 12.3 mg of fluoride ion/gm or ml of gel or 12,300 ppm fluoride ion at pH 3.5.
- NaF 2%: contains approximately 10 mg of fluoride ion/gm or ml of gel or 10,000 ppm fluoride ion at pH 7.0.

Note that APF gel is more effective than NaF in providing prolonged protection against caries and in counteracting the effects of strong acids. However, it is contraindicated where glass based restorative materials are present – such as ceramics, glass-ionomers and some glass filled composite resins.

Concentrated solutions

- SnF_2 20%: dissolved under heat in glycerine for stabilisation, diluted for local topical application as required.

Mouth rinses

- Ranging from 0.02-0.2% NaF (0.1-1.0 mg of fluoride per ml (100-1,000 ppm.) of mouth rinse. Some mouth rinses may be acidulated.

Varnishes

- 0.2% NaF in viscous resins/varnishes, contains 1 mg of fluoride per ml of varnish (1,000ppm).
- 5% NaF in viscous varnishes contain 25 mg of fluoride per ml (approx 26,000ppm).

NOTE: For patients with a high caries rate, supplemental topical fluoride use should be considered.

Prescription of fluorides

Table 4.2 describes the various methods of fluoride application.⁹

The most common fluoride compounds available for topical application are

- NaF (sodium fluoride)
- SnF₂ (stannous fluoride)
- APF (acidulated phosphate fluoride)
- Na₂FPO₃ (sodium monofluorophosphate)

The vehicle most commonly used is a dentifrice, although there is a variety of other methods of routine application, involving solutions, gels and varnishes. Care must be taken in prescribing because some of them may do superficial harm. For example, the acidulated gels provide the highest fluoride uptake but at the same time they are likely to remove the glaze from ceramics or roughen the surface of a glass-ionomer or glass filled composite resin if applied too often. Also, the safety factor must be considered when prescribing highly concentrated fluoride products in some age groups.

Fluoride containing dentifrices

A dentifrice may contain NaF (1.0%), Na₂FPO₃ (0.76%), or SnF₂ (0.4%) = (% F ion). In general there is approximately 1 mg/gm of available fluoride (1000 ppm). A tooth brush completely covered in paste holds approximately 1.5 mg of fluoride.

Recent developments include

- lower concentration fluoride dentifrice for young children (400 ppm F ion) which will reduce unintended fluoride ingestion in children up to six years who are unable to control their swallowing reflex,
- higher concentration dentifrices, with 5,000 ppm fluoride ion which should be restricted to adults with a high caries risk.

Concentrated gels

- APF 1.23% gel: contains approximately 12.3mg F ion/gm of gel or 12,300 ppm fluoride ion, at pH 3.5.
- NaF 2%: contains approximately 10mg F ion /gm of gel or 10,000 ppm F ion at pH 7.0.

APF gel is more effective than NaF in providing prolonged protection against caries and in counteracting the effects of strong acids. However, it is

contraindicated in the presence of glass based restorative materials such as ceramics, glass-ionomers and some glass filled composite resins.

Concentrated solutions

- SnF₂ 20%: dissolved under heat in glycerine for stabilisation, diluted for topical application as required.
- mouth rinses range from 0.2-0.02% NaF (1,000-100 ppm – 1mg F/ml to 1mg F/10ml.) and may be acidulated.

Varnishes

- 1.7% NaF in a viscous resin varnish contains around 1,000ppm fluoride ion.
- 5% NaF in a viscous shellac type of varnish contains around 26,000 ppm fluoride ion.

Varnishes have the advantage of prolonged retention, and through dissolution, allows slow release of the fluoride ion. Gels prolong contact with the enamel for up to a few hours, but if they are swallowed, the fluoride ion is quickly released.

Schedules of application

- The minimum use of topical fluoride for all patients, irrespective of the apparent caries risk, should be a morning and evening application of fluoride dentifrice as part of the basic daily oral hygiene routine.
- Retention rate depends on initial concentration applied. Normally retention rate from a low concentration mouthrinse is relatively high.
- Use concentrated gels only in the most caries active cases.
- Time of day is important. Application immediately prior to retiring offers prolonged retention because of decrease in resting saliva flow rate during sleep.
- Duration of application should be at least three minutes.
- Neutral gels work well on porous enamel or exposed dentine and an acid environment will aid in fluoride transport into the tooth structure.
- APF gel provides higher uptake as stored

fluoride, therefore a more prolonged period of protection.

- With low caries risk, use an acidulated gel, professionally applied, at 6-12 month intervals.
- With high caries rate use the acidulated gel at six week intervals. This can be applied at home using a custom made stent or tray. However, acidulated gels are not recommended for this application because of the potential for etching ceramic or glass containing restorations.

SUMMARY



Factors affecting efficiency of fluoride application

- Oral clearance rate
- Concentration applied
- Time: overnight best
- Duration: three minutes minimum
- Form: acidulated phosphate fluoride gel offers best uptake

Fluoride safety factors for adults

The probable toxic dosage of fluoride ion is 5 mg/kg body weight/per day¹⁰. For the frail, chronically ill adult, this dosage should be considered high, and prescribed doses kept well below this level.

Steps should be taken to minimise ingestion during application. In the office, use adequate suction and, during home application, advise patients to allow drooling of excess over a sink. Spit out excess for one minute after each application. The amount swallowed will then be well below those levels considered necessary to raise total blood levels to the 1 ppm considered likely to cause chronic toxicity.

It is essential to

- prescribe the minimal dose necessary to gain the required result
- instruct patients very clearly in the correct means of self application

TABLE 4.3: Guidelines for additional fluoride therapy

Clinical situation	Therapy guideline
To maintain a low rate of caries.	Morning and night fluoride toothpaste plus 12-monthly topical fluoride gel/varnish.
Extra protection – orthodontic treatment (> 8 yrs), partial dentures, pregnancy.	Morning and night fluoride toothpaste plus 0.2% NaF mouthrinse 2-3 times per week.
One to two cavities per year, over 8 years old.	Morning and night fluoride toothpaste plus 0.2% NaF mouthrinse twice per week or 2% NaF gel every week.
Three or more new cavities per year, over 8 years old.	Morning and night fluoride toothpaste plus 0.2% NaF mouthrinse daily before bed plus 2.0% NaF gel weekly.
Children under 6 years of age with high caries rate.	Supervised brush twice per day with low fluoride paste. 1.23% APF gel: very small quantity painted on teeth by parent weekly, F Varnish six-monthly.
Very dry mouth, or a patient scheduled for radiation, surgery or drugs affecting salivary glands.	Morning and night fluoride toothpaste. 0.2% NaF rinse after lunch, and before bed, or 1.23% APF gel or 2% NaF gel nightly. May use artificial saliva.
Severe erosion – acid reflux, frequent vomiting, excess citrus, wine taster.	Morning and night fluoride toothpaste. 1.23% APF or 2% NaF gel self-application AM and PM during active erosion phase.
Hypersensitive teeth.	Use desensitizing dentifrice twice daily. Caution with brushing technique. Paint area with 2% NaF gel twice per day until sensitivity controlled after initial application of F Varnish.
Noncompliant home users, e.g. adolescent, severely disabled, who need to control caries.	Six-weekly visits to the clinic for supervised self-application of 1.23% APF gel.

- monitor outcomes and reduce the dose as increasing control of the caries is evident

BE AWARE



Fluoride safety for adults

Maximum dose

- 5 mg of fluoride per kilogram of body weight per day

Fluoride safety factors for children

The probable toxic dose of fluoride for children is 5 mg/kg of body weight. Containers of fluoride tablets or drops used to supplement systemic fluoride intake should not contain more than 100 mg fluoride ion in total. Any fluoride should be kept well out of the reach of young children to avoid accidental overdose. Careful supervision of the amounts of fluoride toothpaste used daily is important because ingestion of more than 1 mg fluoride ion daily may lead to fluorosis. Regular fluoride containing dentifrice holds up to 1 mg. fluoride ion per gram of paste so a full brush head of paste contains approximately 1.5 mg of fluoride ion.

BE AWARE



Fluoride safety for children

Probable toxic dose

- 5 mg of fluoride per kilogram of body weight per day

NOTE: A daily dose greater than 0.07 mg of fluoride per kilogram of body weight per day for children with developing teeth may result in fluorosis. Use topical fluorides with caution.

Particularly for small children, use a junior paste which contains only 0.4 mg/gm of fluoride ion. Children under three years are likely to swallow any unused paste unless carefully watched, and up to six years of age may regularly ingest approximately 30% of paste used.

Concentrated gels, and mouthrinses containing 0.2% NaF, should not be prescribed for routine use in young children even when infant caries has occurred. It is better to use a junior paste containing 0.4 mg F/gm fluoride ion three times a day,

with only partial rinsing of excess from the mouth. Localised application of varnish, or a more concentrated solution, may be undertaken by a parent or dental professional.

Prescription and Application of Chlorhexidine

There is a large number of therapeutic mouth rinses designed to reduce the oral bacterial population⁵ and the most effective of these contain chlorhexidine gluconate. Several contain high concentrations of alcohol and this may contribute to further dehydration of the mucosal tissues, particularly with patients with reduced salivary protection. The ones with a water base or contained in a gel are recommended.

Chlorhexidine has proven to be the most effective of the therapeutic plaque control agents because it is able to ionically adhere to the teeth and oral mucosal surface in high concentrations for many hours. This ability to be retained results in prolonged high levels of antibacterial action. Most of the other agents, such as sodium benzoate or cetyl pyridinium chloride, will produce an immediate 30% reduction in susceptible oral bacterial numbers but they regain normal concentrations within a few hours of application. This means that mouthrinses containing chlorhexidine need to be used less frequently than those containing most other antibacterial agents for effective plaque control. Note however, that they should not be used until half an hour after cleaning the teeth with traditional dentifrices as the sodium laurel sulphate saponification component in dentifrice can reduce chlorhexidine activity.

Chlorhexidine is available as a gluconate at 0.2% concentration in mouthrinses and 2.0% concentration in a gel form. The mouthrinses are also available with and without 10% ethyl alcohol as a preservative and taste enhancer, and as indicated above, only those without alcohol should be prescribed for patients experiencing hyposalivation. Twice a day rinsing at this concentration will significantly reduce plaque bacterial counts and

maintain them at a low level. The more concentrated gel form requires only once a day application for the same antibacterial effect. It will also assist in maintaining moisture at the mucosal surface for patients with mild hyposalivation. Alternate application of topical fluoride gels and chlorhexidine gels, one in the morning and one at night, is recommended for patients with high caries risk.

The only negative aspect of routine use of chlorhexidine is the tendency to stain any remaining plaque. This is not a permanent stain, except perhaps around leaking or defective margins, and is readily removed with professional tooth cleaning.

NOTE



Chlorhexidine is the most effective antibacterial

- prescribe water based for preference
- apply 2.0% twice a day, short term for maximum effect
- apply 0.2% daily for long term control
- do not apply within one hour of using toothpaste
- maybe alternate fluoride in morning, chlorhexidine on retiring
- optimum time for application on retiring because of longterm effect

Protective sealants

Fissure sealants are a well proven protective measure against the development of carious lesions in pits and fissures in children. However, sealants are now recommended for all age groups where the caries risk is high, and particularly where the individual's ability to control the causes is diminished, e.g., severe physical or psychological disability. The materials used are unfilled resins or glass-ionomer cements. Even if the seal is applied subsequent to some degree of demineralisation the process will be arrested and bacterial activity is likely to become dormant (**Chapter 14**).

A new glass-ionomer with a high fluoride release is now available to provide surface protection for early smooth surface lesions and newly erupting teeth (**Chapter 11**). A light application will prevent demineralisation of the underlying tooth structure, while allowing some degree of maturation or remineralisation of the underlying enamel to take place. Resin sealants will also prevent further demineralisation but will not allow normal maturation and remineralisation to take place.

Further Reading

1. Kidd EAM and Joyston-Bechal S., eds. *Essentials of dental caries; the disease and its management*; Oxford: Wright, 1987.
2. Nikiforuk G. ed. *Understanding Dental Caries. Prevention: Basic and Clinical Aspects*. Basel: Karger, 1985. Ch 8.
3. Lussi A, Kohler N, Zero D, Schaffner M, Megert B. A comparison of the erosive potential of different beverages in primary and permanent teeth using an *in vitro* model. *Euro J Oral Sci* 2000; 108:110-114.
4. Murray, JJ. ed. *The prevention of dental disease*. Oxford: Oxford University Press, 1989.
5. Nikiforuk G. ed. *Understanding Dental Caries. Prevention: Basic and Clinical Aspects*. Basel: Karger, 1985. Ch 11.
6. Amin SM, Harrison RL, Benton TS, Roberts M and Weinstein P. Effect of Povidone-iodine on *Streptococcus Mutans* in Children With Extensive Dental Caries. *Ped Dent* 2004; 26(1):5-10.
7. Edgar WM and O'Mullane DM. Factors affecting salivary flow rate and composition; in *Saliva and Dental Health*. *Br Dent J*, London, 1990; 11,12.
8. Fejerskov O, Ekstrand J and Burt B. eds. *Fluoride in Dentistry*. Copenhagen: Munksgaard, 1996.
9. Proceedings from a Workshop; Dentine and dentine reactions in the oral cavity. Silverstone LM, Hicks MJ and Featherstone MJ. Dynamic factors affecting lesion initiation and progression in human dental enamel II. Surface morphology of sound enamel and caries like lesions of enamel. *Quint Int* 1988; 19(11):773-785.
10. Whitford GM. The physiological and toxicological characteristics of fluoride. *J Dent Res* 1990; 69 (spec issue): 539-549.
11. Manton DJ and Messer LB. Pit and fissure sealants: A major cornerstone in preventive dentistry. *Aust Dent J* 1995; 40: 22-29.

5 Non-carious Changes to Tooth Crowns

J. A. Kaidonis ♦ L. C. Richards ♦ G. C. Townsend

Apart from dental caries and iatrogenic damage (eg. the dental handpiece), the main processes that can change the morphology of a tooth during its lifetime are abrasion, attrition, erosion and fracture. Fossil records, anthropological research and studies in comparative anatomy, show that the processes responsible for tooth reduction have acted on teeth since prehistoric times. Selective forces such as environmental stress have produced evolutionary changes, over generations, to the morphology and physiological function of the stomatognathic system.

Physiological adaptation of the body, in response to environmental stress, includes production of secondary dentine, continual eruption, changes to masticatory patterns, remodelling of bone (e.g. temporomandibular joint) and especially the ability to remineralise both enamel and dentine. There appears to be a perpetual balance between environmental stress and physiological adaptation. It is only when the body is too slow to adapt, or is unable to adapt, that pathology will become evident.

This biological approach to the oral structures recognises that they will change throughout life, a view that varies from past concepts. The focus on modern dentistry has for many years



been on caries and periodontal disease, and has evolved into an art and science aimed at restoring the broken down dentition to its original newly erupted morphology on the assumption that the unworn tooth has the ideal functional form. A variety of geometric concepts of occlusion have evolved over the years and occlusal reconstruction has tended to follow formal guidelines regardless of the great variability that exists in the architecture of the stomatognathic system within and between populations, as well as in the same individual over time.

By recognising progressive change in tooth form as a physiologically dynamic process, premature and unnecessary dental intervention may be avoided.

Terminology

There is a lack of consistency in the dental literature in the terminology used to distinguish and describe the different types of noncarious tooth reduction. The accepted terms **abrasion** and **attrition** are often used interchangeably. The term **erosion** is sometimes considered as tooth wear when in reality it is the result of chemical dissolution of tooth structure, not the rubbing together of surfaces. The confusion has probably arisen because all three forms of tooth loss often occur simultaneously and because of the lack of understanding of how these mechanisms present clinically. Although fracture is a separate process leading to loss of tooth structure and should be considered as pathological damage, it should be remembered that microfracture is what defines the wear process of abrasion and attrition. In addition, the dentist must be aware that the definitions are purely dental descriptions. From a tribological perspective (Tribology: a subdiscipline of engineering associated with the study of wear and lubrication), attrition and abrasion are essentially two and three bodied abrasion respectively while, what dentists call erosion, is in fact corrosion. The universal acceptance of erosion is when particles (solid or liquid) moving at high velocity cause wear upon a surface.¹



Fig. 5.1. Abrasion on anterior teeth. The notching on the incisors was caused by crushing dried water melon seeds held 'vertically' between the teeth.

The term **tooth reduction** is therefore a useful generic description because it covers all processes that lead to the loss of tooth substance. In this Chapter the terms abrasion, attrition, erosion and fracture will each be defined and described as currently observed in the dental literature and do not follow tribological definitions.

NOTE

Tooth reduction =	
abrasion –	3 body wear
attrition –	tooth to tooth wear
erosion –	chemical reduction

Aetiology of Tooth Reduction

Abrasion

Every² described abrasion as: “the wearing of tooth substance that results from friction of exogenous material forced over the surface by incisive, masticatory, and grasping functions.” To this must be added the wear caused by tooth cleaning. Within this definition exogenous material is anything foreign to tooth substance. The most common material forced over tooth surfaces is the food itself. Included are sand, grit and foreign material found in the food bolus, the natural abrasivity of some foods, and any solid material



Fig. 5.2. Note the excessive wear on the incisal edges of the upper left central and lateral incisor. The patient has consistently held a pipe between the teeth while working over many years.

held by or forced against the teeth. Abrasion may therefore occur during mastication, when the teeth are being used as tools, or during tooth cleaning where the foreign body is the toothbrush and the dentrifice. The ability to use the teeth as tools is an important evolutionary advantage, a purposeful function and not a parafunctional activity.³

In general, the action of abrasion from food is not anatomically selective on the tooth surface. In other words, the abrasive influence of a bolus of food occurs on the whole occlusal surface affecting the cusp tips, cusp inclines and fissures plus and to a lesser degree, the occlusal aspects of the buccal and lingual surfaces.

An exception to this lack of specificity may occur when the same two or three teeth are used repeatedly as tools for grasping an object. This may lead to more severe abrasion on these teeth and examples of this type of abrasion may be related to a broad range of occupations and pursuits, from hunter-gathering to pipe smoking (**Figures 5.1 and 5.2**).

An abrasion area produced by food, as distinct from an attrition facet, is generally not well defined as abrasion tends to round off or blunt tooth cusps or cutting edges. In addition, the tooth surface will have a pitted appearance (**Figure 5.3**). Where dentine is exposed it may be scooped out since it is softer than enamel.

Interestingly, dentine exposed by abrasion is

not sensitive because it will be covered by a smear layer typical of that seen with other mechanical interference such as a dental bur. The dentine tubules can be 'burnished' by mechanical action so closing them over. This suggests that dentinal sensitivity on an abraded area may indicate the presence of erosion (corrosion) as well.

Abrasive dentinal wear is relatively shallow in nature when compared to that of erosion. The ratio of depth to bucco-lingual width is relatively constant for any particular diet. As the enamel rim wears the dentine will be proportionally scooped out as well.⁴ In addition, the maximum depth of dentine loss shifts towards the buccal of the scooped area for the lower posteriors and towards the palatal of the upper posteriors as the cusps are worn flat and the masticatory stroke becomes broader. There are situations where abrasion will not lead to scooped dentine, such as wear caused by a pipestem, because of its solid structure.

Microscopically, an abraded surface shows haphazardly oriented scratch marks, numerous pits, and various gouge marks (**Figure 5.4**). However, abrasive scratches will be almost parallel when the abrasive material is forced in one direction only across the tooth surface. This occurs during the last phase of the masticatory stroke when opposing teeth come close to each other between 1 and 3 mm out of centric occlusion and then slide into intercuspal position with food between the



Fig. 5.3. Abrasion on the occlusal surface. Australian Aboriginal teeth exposed to excessive abrasion. Note gouge marks and pitting on the enamel and the dentine is scooped.



Fig. 5.4. Scanning electron micrograph of an abraded occlusal surface: Note the random pattern of scratch marks. Mag. x100.

surfaces. The length, depth and width of this microdetail varies depending on the abrasiveness of the food, and the pressures applied during mastication.

The distribution and extent of abrasive wear over the dentition is influenced by many variables including type of occlusion, diet, lifestyle and age.

Influence of occlusion

The type of occlusion is a prime factor in the distribution and pattern of abrasion. As the variability of upper and lower tooth positions is almost limitless, the distribution and pattern of abrasion can also be extremely variable. As a general rule, in an Angle Class 1 molar relationship, with normal anterior overjet and overbite, abrasive wear will occur on the occluso-buccal aspect of the lower teeth and the occluso-palatal aspect of the upper teeth producing an ad-palatum occlusal slope. This will normally hold true for the premolars and first permanent molars, but the occlusal slope may be reduced to neutral around the second molars, and finally may be negative or ad-linguum on the third molars. The occlusal twist that develops on the occlusal surface of posterior teeth with advanced abrasion is called the helicoidal plane⁵ (Figure 5.5).

Diet and lifestyle

Molnar⁶ described how abrasion is intricately related to diet and culture: “The varieties of foods consumed by primitive man and the specialised tool function of the teeth have left significant marks in the form of worn occlusal surfaces over the dental arches”. For example, nonindustrial populations living in a harsh environment, masticating hard, fibrous foods show more extensive abrasion than those in industrial urban societies consuming soft processed foods.

Age

There is a high correlation between age and tooth wear within all populations. Obviously, newly erupted teeth have less wear than those that have been in function for a longer period. In general, the older the individual the more extensive the abrasion, although there will be individuals in modern cultures who show very little wear indeed.

Oral hygiene techniques

Although routine tooth cleaning is desirable to reduce the risk of periodontal disease and caries, the cleaning process itself may result in the loss of tooth structure through abrasion. The use of an abrasive dentifrice, combined with vigorous brushing with a hard toothbrush, can result in abrasive defects particularly near the gingival margin on the facial surfaces. Such loss of tooth structure can pose a significant problem. When dentin is exposed by abrasion alone the tubules may remain closed by the so called smear layer. In the presence of acid, the dentinal tubules may be opened through loss of this layer causing the pulp to become inflamed and respond to changes in temperature, osmolality and tooth drying. This painful condition is called cervical hypersensitivity. Loss of tooth structure from abrasion may become so severe that the strength of the tooth is threatened.

While closure of dentinal tubules can overcome cervical hypersensitivity on a temporary basis, for long-term resolution it is essential to determine the cause of the problem. As will be described below, exposure of the tooth surface to low pH food or drink prior to brushing may lead to rapid demineralisation leaving the collagen matrix exposed to damage from a tooth brush. This may exacerbate loss of structure and prevent the natural closure of dentinal tubules by salivary precipi-



Fig. 5.5. Abrasion pattern on an ancient skull specimen: Note the helicoidal wear pattern on the occlusal of the posterior teeth emphasising the slope to the lingual in the third molars.

tate. Cervical hypersensitivity is discussed in more detail in **Chapter 7**.

Attrition

The term attrition is used to describe tooth wear caused by tooth-to-tooth contact without the presence of food. It was defined by Every² as “wear caused by endogenous material such as microfine particles of enamel prisms caught between two opposing tooth surfaces”. The enamel prisms break off and become caught as the tooth surfaces are forced over one another, producing characteristic parallel striations when viewed microscopically.

The characteristic feature is the development of a facet which is a flat surface with a circumscribed and well defined border. There will be fine parallel striations in one direction only and within the border of the facet. One facet will match perfectly with another facet on a tooth in the opposite arch and the parallel striations will be lying in the same direction.

In general, incisors and canines show facets with striations that are orientated in an anterior-lateral direction (**Figure 5.6**), while facets on posterior teeth show striations that are either transverse (i.e. a bucco-lingual orientation) when on the working side, or oblique (i.e. running in the direction of the opposite canine) on the nonworking side. This general pattern is common among

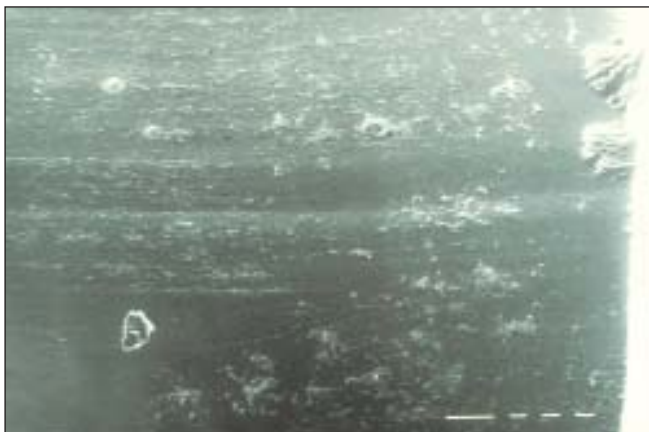


Fig. 5.6. Scanning electron micrograph of the surface of a facet: Note the parallel striations. Mag. x100.

human populations and occurs from a lateral mandibular movement, where the mandible may go past the canine edge-to-edge position.

The distribution of attrition is influenced by the type of occlusion, the geometry of the stomatognathic system and the characteristic grinding pattern of the individual.

Bruxism and parafunction

In the past, such terms as bruxism and parafunction have been used synonymously to describe persistent tooth grinding and clenching. It has been described as a pathologic habit leading to various craniomandibular disorders and it has been suggested that occlusal interferences, deflective inclines and stress have all acted, alone or in combination, as trigger mechanisms.

Parafunction implies ‘outside that of normal function’ and includes habits such as pencil chewing, nail biting etc. which are considered as pathological. It must be noted that parafunctional activities, taken on their own, are in fact within the range of normal function, that is, using the teeth as tools. In a pre-industrialised population, a person would ‘strip’ a piece of wood to make it sharp, or bite to remove a piece of damaged finger nail and this is, in fact, normal functional activity. However, when pencil chewing and nail biting are performed on an habitual basis, uneven wear and possible related pathology may become apparent. Other more obvious pathological parafunctional habits such as cheek and lip biting should be included within this category.

NOTE

Parafunction is outside of normal.

Bruxism can be considered as a physiological behaviour. When tooth grinding is quantified from the frequency of faceting observed on teeth within the general population, and especially faceting observed on the tips of canines, then tooth grinding can be considered as universal.⁷ In fact, over 90% of people in both preindustrialised and industrialised populations show evidence of tooth grinding. Children frequently grind their teeth, and even infants grind their gums prior to tooth eruption. This suggests that tooth grinding is a universal behaviour rather than a habit, because habits are learned behaviour patterns. So tooth grinding

should be regarded as a common physiological behaviour of central origin. It is only when stress levels become too high that grinding intensity increases to the point where there is likely to be adaptive changes to the craniofacial structures, including the muscles and joints. When these structures are too slow to adapt, or fail to adapt, then pathology may become evident in a variety of forms such as craniomandibular disorders.

It is logical, therefore, to accept that it is a behaviour of central origin, and that only acquired habits such as persistent nail biting and pencil chewing should be regarded as parafunction. Occlusal interferences cannot be entirely discounted and should be observed and understood in the context of treatment planning. Not that they initiate bruxism, but they are likely to provide an environment where the direction and intensity of grinding forces may affect teeth, muscles or joints. Current opinion within the literature suggests no association between malocclusions and craniomandibular disorders.

The development of malocclusions during growth will be slow enough so the stomatognathic system will have time to proprioceptively learn and develop a functional pattern which may include avoidance mechanisms for interferences which become part of the functional envelope. However, there is a potential for problems to develop if the functional pattern undergoes an acute change without the body having time to adapt. Such changes may be rare, for example facial trauma, but acute changes to the occlusion caused by general operative dentistry must be considered responsible for some craniomandibular problems.

The physiological approach to tooth grinding has been suggested by many researchers, in particular by Every², who proposed the theory of thegoxis. This theory suggests that tooth grinding is a phylogenetic behaviour pattern designed to enhance specific facet edges and hence the efficiency of the masticatory system. In other words, while function causes abrasive wear on enamel, tooth grinding will reinstate the sharp edges and enhance the efficiency of enamel blades.

Interproximal attrition

Interproximal attrition occurs on the contacting proximal surfaces of adjacent teeth when they move against one another during occlusal loading, such as mastication or tooth grinding. Examination of interproximal wear facets in teeth does not show the microwear described above. Instead, the interproximal surfaces show grooves which are orientated vertically, that is occluso-gingivally, and match well with grooves on the adjacent tooth surface. Interproximal wear on the mesial of a tooth is often greater than wear on the distal interproximal contact. In vitro research has accurately modelled this pattern⁸, indicating that, provided alveolar bone support remains intact the predominant movement is either vertical or near vertical with a minor mesial tilt, rather than bucco-lingual as has been suggested in the past. This leads to a gradual shortening of the dental arch length over time. This movement is distinctly different from periodontally affected teeth with bone loss because these can be displaced buccally or lingually as well under load depending on where the bone loss is situated.

Erosion

Erosion of tooth structure is defined as the superficial loss of dental hard tissue due to a chemical demineralisation not involving bacteria. The clinical appearance will vary (**Figures 5.7 and 5.8**). In



Fig. 5.7. Active erosion on a premolar tooth. Note the glazed surface, the loss of microanatomical detail and the scooping of the dentine.

generalised erosion the whole tooth crown may be affected with loss of surface definition leading to a glazed, lifeless appearance with no sharp enamel ridges as they become rounded off. The enamel surface may become relatively concave until the dentine is exposed, whereupon the erosion accelerates due to the relative lack of mineralisation of the dentine. This leads to a scooped out appearance. Dentine exposed by active erosion is temperature sensitive because the repeated acid attack keeps the dentinal tubules open to the oral environment. In fact, sensitivity to cold is a good diagnostic feature for active erosion. Dentinal scooping from erosion can become very deep when compared to that from abrasion.

The extent and pattern of erosion that occurs in a particular patient may help to identify the source of the acid causing the problem but great variation is possible. The direction of acid movement within the mouth, the variations in saliva flow and even the pattern of swallowing may have an effect.

Significance of saliva

One of the main predisposing factors for erosion can be a lack of either quality or quantity of saliva (Chapter 7). The biofilm on the tooth surface is an important natural barrier to acid. However, in the presence of a low pH, the biofilm is readily removed, leaving the tooth surface looking very clean but exposed to acid attack. One of the diag-

nostic features of active erosion is a pristine mouth with no evidence of staining or plaque.

It must be remembered that the biofilm found on teeth is also the result of many years of evolution. The salivary pellicle is responsible for the closed system at the pellicle/tooth interface. Following normal conditions of demineralisation the basic ingredients, Ca^{2+} , PO_4^{3-} and OH^- ions of hydroxyapatite will be released and entrapped below the biofilm. They will then be available for remineralisation following modification of the pH. Therefore, when there is no biofilm present, an acid attack on the tooth surface will cause instant demineralisation without the potential for subsequent remineralisation. In other words there is no closed system and the essential ions will be lost permanently. The surface dissolution will be rapid, resulting in dished out lesions previously described. Surface loss from erosion is different from the relatively slower subsurface demineralisation associated with the pathogenesis of caries and the white spot lesion.

Abrasion, especially from tooth brushing, and/or attrition may be superimposed over eroded surfaces leading to excessive tooth reduction and further difficulties in diagnosis. For example, erosion will be greatly exacerbated if the teeth are brushed while the acid level in the mouth is high. Brushing at this point will remove the organic framework so that remineralisation cannot then take place even if supersaturated conditions were possible. After the acid intake it will be sufficient to wash the mouth vigorously with water to remove the acid residue and delay brushing for up to three hours. This allows sufficient pellicle formation and therefore the natural environment for remineralisation will be re-established.

In a chronic situation such as the professional wine taster, application of a fluoride mouth wash prior to a tasting session, while the biofilm is still present, will allow for remineralisation with fluoroapatite and therefore minimise the problem.

The acids which cause erosion of the tooth surface may originate from either extrinsic or intrinsic factors.



Fig. 5.8. Active erosion lesions on the occlusal and buccal of the lower bicuspid. The erosion is active because there is sensitivity to cold.

SUMMARY

Chemical erosion can be the result of

extrinsic factors

- acid food
- acid drinks – cola drinks, wine, sports drinks
- medications – asthma puffers

intrinsic factors

- regurgitation of gastric acid
- gaseous reflux (burping)
- chronic vomiting

Extrinsic factors

Acids of extrinsic origin arise from outside the body. Industrial acids can be carried in gaseous form in the air in heavily polluted areas and may cause demineralisation of the labial surfaces of anterior teeth, particularly in a mouth breather. Progress of the erosion may be relatively slow and, therefore, diagnosis is often difficult.

A variety of foods and drinks have a low pH and frequent ingestion may cause problems. For example, low pH cola drinks (including so called diet colas), cordials and fruit juices may cause erosion. However, individual variations in the method of consumption of these liquids before swallowing may lead to differing patterns.

Certain medications are also acid in nature and the potential for demineralisation must be recognised and the patient counselled. For example, a lack of gastric acid may be compensated by the oral administration of concentrated hydrochloric acid with advice that it should be taken through a straw or glass tube. However, there is still a tendency to force some of the acid into the oral cavity by the act of swallowing. Other examples include asthma medications, especially puffers. These have a very low pH and the mouth should be rinsed with plain water immediately after use to neutralise the acid. These medications also tend to relax the smooth muscle of the gastrointestinal tract leading to the possibility of acid reflux which can affect the teeth.

Intrinsic factors

Generally, intrinsic factors can be subdivided into recurrent vomiting and gastro-oesophageal reflux disease (GORD). GORD is subdivided into three categories, regurgitation, that is when stomach contents reach the mouth but are immediately swallowed again, rumination, when stomach contents are chewed then reswallowed and gaseous reflux, that is burping. Regurgitation and gaseous reflux are common while rumination occurs only among infants and some bulimics. Intrinsic factors may be differentiated from extrinsic acids by observing the distribution of the affected areas.

Chronic vomiting will affect the palatal surface of the upper teeth because they are in the path of the gastric contents when emitted, while the lower teeth will be protected to a degree by the tongue. However, chronic gastric reflux may erode both upper and lower teeth because the constituents of the reflux are in a gaseous form and may be more widely distributed around the oral cavity.

In the presence of modified salivary flow and reduced buffering capacity, the effect of both extrinsic and intrinsic factors will be exacerbated. The buffering capacity of the saliva against acid attack is the best defence against both caries and erosion, but routine use of fluoride and casein products, either professionally or home applied, will assist in reducing the damage (**Chapter 8**).



Fig. 5.9. A lesion on an upper premolar has developed over the last 5-6 years, yet the lowers have been missing for 50 years. The cause may be tooth brushing rather than abfraction.

Abfraction

Although toothbrush abrasion has for many years been considered responsible for the typical wedge-shaped lesion observed on labial and buccal surfaces of teeth, there is growing evidence that excessive buccal and lingual forces on teeth may be responsible for some of these lesions. This concept, termed abfraction, proposes that flexure of the tooth at the cervical margin while under load is responsible for the progressive breakdown of the brittle dental tissues (**Figure 5.9**). If a cusp remains under load at the beginning or end of a masticatory cycle, there is a possibility of flexure or compression in the crown, either of which may lead to dislocation of enamel and/or dentine at the point of rotation. However, it is suggested that there is no unanimity on the theory although it remains a possibility (**Figure 5.10**).

Tooth fracture

Tooth fracture is a relatively common occurrence, particularly on teeth which have been restored. It may be the result of direct trauma but there are other reasons as well and a careful diagnosis is required rather than just smoothing over the roughened area. The following forms of tooth loss from fracture should be noted:

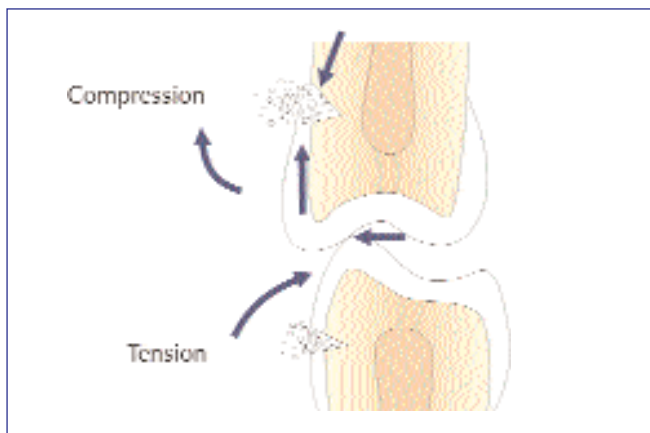


Fig. 5.10. Abfraction is thought to result from undue load on relatively flexible teeth. It can result from either compression or tension as shown.

Enamel flaking

Slivers of enamel of various sizes may fracture from the incisal edges of anterior teeth or from the buccal or lingual edges of posterior teeth, particularly if the occlusal table is flat. Occasionally large areas of buccal or lingual enamel plate may split off leaving dentine exposed. It is important to distinguish between chipping from direct trauma and that arising from pernicious habits such as biting cotton, biting fingernails or opening hair clips with the teeth. However, enamel flaking may be the result of tooth grinding and the pattern that results reflects the direction of the mandible during the forceful phase of the grinding stroke (**Figure 5.11**). As described above, the microwear detail over the dental arches is the blueprint produced by a lateral mandibular movement where the mandible starts from centric occlusion and moves outwards past the canine edge-to-edge. This produces a pattern of enamel flaking affecting the labial incisal edges of the upper incisors and the lingual incisal edges of the lower incisors. During this grinding action, it is the lateral pterygoid on the contralateral side that is active and responsible for the movement.

Occasionally the direction of a forceful grinding stroke is affected by a deflective incline on a posterior tooth, which has become a guiding factor, as



Fig. 5.11. Extreme lateral grinding movement past the canine edge-to-edge leading to enamel chipping. The facets are distinct and the wide bucco-lingual groove corresponds with the upper canine. The patient is a stressed 16-year-old.

a result of a change in the distribution of the posterior teeth following extractions or from restorative procedures. Such guidance may produce a subtle change in the wear pattern specific for that individual or may lead to fracture (**Chapter 18**).

Extreme wear patterns

Extreme lateral grinding patterns extending past the canine edge-to-edge position are common. These extreme positions cannot be achieved voluntarily by patients without discomfort or strain, but it can be shown that the wear facets match entirely. This suggests that these extreme positions may be attained during sleep where the body's protective reflexes are turned off. The forces applied during such movements are relatively high and explain not only enamel chipping, but failure of labial veneers, cracks in porcelain crowns and fracture of cusps. These extreme lateral mandibular movements may cause the temporomandibular joint on the contra-lateral side to move past the eminence – a position where the condyle is physiologically disarticulated – and this position may be sustained during sleep. This may lead to some of the temporomandibular joint problems observed clinically, where the affected joint is opposite to the side of the heaviest wear.

Although it is possible for an unrestored tooth to fracture during tooth grinding, it is far more common in teeth weakened iatrogenically by the placement of restorations. Cavities designated #2.2 in the new classification (**Chapter 14**) will double or even triple the length of cusps, substantially increasing the torque at the cusp base and leaving the tooth more prone to fracture. Endodontically treated teeth are also at increased risk due to loss of tooth structure related to access for root canal therapy.

As the patient ages, teeth develop minor cracks in the enamel which are usually repaired by precipitation of salivary pellicle followed by mineral deposition. However, if the tooth is subject to heavy occlusal load the crack can propagate through to the dentine. Movement of the cusp under function may then be extremely painful due to hydraulic stimulation of odontoblast sensory nerve receptors. Treatment involves identifying, protecting and strengthening the cusp

(**Chapters 10 and 14**). The cusps most prone to split and fail are the lingual cusps of lower molars and the buccal or lingual cusps of upper first and second premolars.

Crown fracture

The crowns of anterior teeth are most at risk from extrinsic forces such as direct trauma. The main predisposing factors are the age of the patient and tooth position. From the time of emergence of the permanent anterior teeth to the late teen years there is a combination of immature physical activities with immature facial structures. Teeth that tend to protrude are therefore at a higher risk. In the older patient the presence of caries, restorations, erosion, abrasion or attrition may have already weakened the crown structure and, even a minor blow, may lead to loss of part or all of a crown. Both crowns and roots are at increased risk of fracture in endodontically treated teeth.

Adaptation and pathology

The human dentition should remain functional throughout life. Dental caries and periodontal disease leading to premature tooth loss are modern day diseases, since the incidence of these diseases in prehistoric populations was relatively very low. It is not uncommon to find ancient skeletal material with completely intact dentitions and no evidence of caries, only tooth reduction.

The craniofacial structures are made up of individual units including the teeth, temporomandibular joints, musculature and the supporting craniofacial skeleton. Any change to one component of the craniofacial anatomy may lead to alterations in associated structures. Because of their physiologic plasticity, the craniofacial structures are in a state of continuous change throughout life, the extent and rate of change being related to a combination of the genetic makeup of the body and the influence of environmental forces (i.e. environmental stress). Functional demands imposed upon the system are one of the factors responsible for change and, only when the body cannot adapt or is too slow to adapt to these demands, will tissues break down pathologically.

Stability of occlusal vertical dimension

Advanced tooth reduction may lead to a quantitative change in the craniofacial complex. In the absence of any compensatory or adaptive response from associated structures, a reduction of the occlusal vertical dimension, or face height, would be expected. However, research suggests that the occlusal vertical dimension is generally maintained through compensatory mechanisms of continual eruption of teeth. Further evidence suggests that, if the amount of tooth reduction is small, there may even be an increase in occlusal vertical dimension over time. Face height seems to be dependent on the balance between the rate of occlusal tooth reduction and the adaptive bodily responses of tooth eruption and alveolar bone growth.

NOTE



The vertical dimension is expected to remain essentially unchanged throughout life in spite of wear, abrasion and attrition.

Adaptation within the tooth

Progressive tooth reduction also leads to an adaptive change within the tooth with the production of secondary dentine within the pulp chamber. When the rate of loss of tooth substance is slow secondary dentine will form without damage to the vital pulp, although at times it may become completely calcified.

However, if the response is not adequate there may be loss of vitality with associated periapical pathology. Furthermore, the gradual loss of cusps generally leads to a wider masticatory stroke resulting in adaptive anatomical changes to the temporomandibular joint including modification or flattening of the articular eminence.

Diagnosis

The causes of tooth reduction have been outlined and it must be noted that more than one process may be acting on teeth simultaneously with varying intensity and duration. A diagnosis

cannot therefore be made on the basis of surface appearance alone. Facet borders may not be distinctively sharp, or may not even exist at all, due to the extent and duration of action of other mechanisms such as abrasion and erosion. Similarly, erosion may remove all fine detail and overwhelm evidence of abrasion. Tooth grinding combined with abrasion upon an eroded surface, may remove more tooth substance than normal because of the weakened enamel surface.

This confusion and interplay of forces may complicate clinical diagnosis. However, with a clear understanding of the ways tooth reduction may take place, and a thorough medical and dental history, the causes will often become self evident. Questioning patients in relation to tooth reduction should form a normal part of the history taking process. The following factors should be taken into account in diagnosis and treatment planning.

Age of the patient

The degree of tooth wear will generally be related to the age of the patient. An elderly patient with a fully functional dentition may show loss of more than half of the clinical crowns, but in the absence of pain and assuming that aesthetics is of no concern, the situation can be considered to be physiologic. However, the same degree of wear in a 20-year-old patient could be interpreted as being pathologic and the chance of retaining a complete dentition into old age may be remote.

Random loss of teeth

Random loss of posterior teeth will lead to additional load being borne by the remaining teeth and they are then more prone to attrition and abrasion, particularly if the posterior support has fallen below the theoretical minimum (**Chapter 18**) (**Figures 5.12 and 5.13**). The presence of deflexive inclines may promote the development of unusual wear patterns. Restoration of posterior support to within the minimum, along with restoration of freedom of movement through the absence of deflexive inclines, may well prevent further loss of tooth structure and stabilise the situation in a relatively simple fashion.

Evidence of active tooth grinding

A diagnosis of attrition may be difficult to make because of the other mechanisms that may be present concurrently. Observation of the following signs and symptoms may lead to a diagnosis of attrition:

- Shiny facets – well defined and polished facets indicate active tooth grinding. The facets should normally be capable of being matched between opposing arches but, occasionally, a patient is capable of adopting a bizarre inter-occlusal position during intense concentration or during sleep and will develop a facet in an apparently impossible position such as the labial incisal edge of an upper canine. However, in the presence of erosion, the facet may not appear shiny even if the bruxism is active.
- Enamel flaking – active grinding can cause enamel flaking on the incisal edges of teeth. Any staining associated with these fractures can imply past activity.
- Myofascial pain dysfunction – the presence of MPD syndrome may indicate active tooth grinding with associated pain and tenderness in the temporomandibular joints. Attrition facets may be detectable.
- Stiff jaw – an acute episode of stiffness in the muscles of mastication may result from traumatic injury or infection, but chronic stiffness may indicate active tooth grinding, par-

ticularly if it is apparent upon waking after a nights sleep.

A simple method of detecting the presence of active tooth grinding is to construct a night guard. Polish the occlusal surface to a matte finish only and subsequent tooth grinding activity will show as highly polished wear facets on the acrylic surface.

Evidence of erosion

Early signs of erosion may be difficult to detect and demonstrate but the following are often indicative of erosion:

- If, in the presence of active tooth grinding, there are no well defined facets there is probably active erosion.
- Sensitivity – active erosion will demineralise the dentine surfaces and lead to exquisite sensitivity through the open dentine tubules. This sensitivity can be on both cervical areas or scooped occlusal surfaces.
- Staining – eroded surfaces that show evidence of staining can be considered inactive.

Careful history taking is required to confirm the diagnosis because patients are often reluctant to disclose unusual dietary habits. Patient education and counselling is important if the process is to be arrested.



Figs. 5.12 & 5.13. Models of the occlusal relationship of a patient with serious loss of posterior support. In conjunction with occlusal guidance there is severe wear on the incisal edges of the anterior teeth.

Evidence of abrasion

As defined above most patients will undergo some degree of abrasion simply through mastication (**Figures 5.14 and 5.15**). However, the degree will vary depending on the enthusiasm for chewing and the type and consistency of the food being consumed. The decision as to whether the situation is pathological, and in need of treatment, will depend upon many factors. Generally erosion and attrition are the primary aetiological factors and abrasion may be a complicating factor.

Diagnosis of active tooth reduction

The best method of making a diagnosis is to study accurate impressions or replicas of teeth under a

low power microscope to reveal the microdetail of attrition, abrasion and erosion. Make a defined scratch with a sharp explorer or a No. 12 scalpel on a facet or an eroded area. Make a rubber based impression immediately and compare with further consecutive impressions obtained one to four weeks later. The disappearance or reduction of definition of the scratch over a period of 2-4 weeks would suggest that tooth reduction is active. The scratch should be viewed in the impression itself under magnification. The diagnosis of cause will be difficult and may require careful history taking and continuing observation.



Fig. 5.14. Extreme erosion and abrasion in a stressed and nervous patient. Note the effect of different restorative materials. The porcelain crowns have not worn but the surrounding and opposing teeth show considerable wear.



Fig. 5.15. This patient lacks posterior support but the outstanding feature is the extreme wear on the lower anterior teeth caused by the single porcelain crown.

Further Reading

1. Mair LH. Wear in Dentistry – Current terminology. *J Dent*, 1992; 20:140-144.
2. Every RG. *A new terminology for mammalian teeth : Founded on the phenomenon of thegnosis*. Christchurch: Pegasus Press, 1972:1-64.
3. Richards LC. Form and function of the masticatory system. In: Ward GK. ed. *Archaeology at ANZAAS*, Canberra: Australian Institute for Aboriginal Studies. 1984.
4. Bell EJ, Kaidonis JA, Townsend GC and Richards LC. Comparison of exposed dentinal surfaces resulting from abrasion and erosion. *Aust Dent J* 1998; 43:362-366.
5. Richards LC and Brown T. Development of the helicoidal plane. *Hum Evol* 1986; 1(5):385-398.
6. Molnar S. Tooth wear and culture: A survey of tooth functions among some pre-historic populations. *Curr Anthropol*, 1972; 13:511-526.
7. Kaidonis JA, Richards LC, Townsend GC. Nature and frequency of dental wear facets in an Australian Aboriginal population. *J Oral Rehabil* 1993; 20:333-340.
8. Kaidonis JA, Richards LC, Townsend GC. Abrasion; an evolutionary and clinical view. *Aust Prosthodont J* 1992; 6:9-16.

6 Risk Assessment in the Diagnosis and Management of Caries

H. Ngo ♦ S. Gaffney

As caries is now recognised as a complex disease it is necessary to redefine the methods of diagnosis and treatment planning if the profession is to treat patients in the most efficient and effective manner. Over recent years there have been a number of formal methods of treatment planning offered by different authors and the authors of this chapter acknowledge their priority. No doubt each method has its advantages and disadvantages and most of them are quite broadly based. The following chapter offers a plan for diagnosing the disease status of each individual patient so a customised treatment schedule can be prescribed and is therefore more likely to be successful. There is no doubt that the disease belongs to each patient and until they accept responsibility for their status then success will be limited. The advantage of the system offered here is that the plan is strictly personalised and therefore will have a higher potential for success.

The concept of the use of a traffic light colour system of recognition for a number of health concepts is not new and the authors offer no apology for its further use. It is an effective method which is widely understood and readily recognised by all age groups and there is no reason why the dental profession should not make use of a 'good thing'.



Introduction

This chapter has been written by clinicians for clinicians. It describes a method of integrating the current knowledge of the caries process into everyday clinical practice with the focus on the treatment of individuals with caries rather than general caries prevention at a population level. It supports the need to diagnose the disease early, well before the presence of cavities indicates that it has been present for some time.

In many developed countries, caries is no longer a pandemic disease so clinicians see fewer patients with active caries than twenty five years ago. A Commission of the FDI reported a decline in caries rates in nine countries and suggested that there is a polarisation of the population in these countries into a low caries majority and a caries active minority. It is recommended therefore that treatment of caries should be patient centred, that is, treatment should be designed to meet the specific needs of the individual. For this reason, in the guidelines for the treatment of individuals aged between 6 and 16 years, the Scottish Intercollegiate Guidelines Network recommended “an explicit caries risk assessment should be made for each child presenting for dental care”.¹

The aetiology of caries and the influence of various risk factors in the caries process have been well understood for some time. What has been missing is a way to use this knowledge to develop personalised treatment recommendations for individual patients. A dentist in general practice is now faced with two challenges. The first is to diagnose the level of caries activity. Caries is a slowly progressing disease controlled by numerous interacting factors and the ultimate consequence is the cavities which are found on tooth surfaces. The cause of the disease cannot be determined by a physical examination of the hard tissues – a cavity is the ultimate outcome of the complex of factors.

The second challenge is to identify caries prone individuals and to convert them to caries resistant ones. It is essential that all contributing factors be considered so that an appropriate management

program can be designed to meet the needs of each individual.²

A ‘Traffic Light–Matrix’ model has been developed to address that problem. It offers a systematic approach to the assessment of all the risk factors which contribute to caries activity. Information is gathered and used to produce a risk profile from which a patient centred treatment regime can be developed. The model is based on the fact that the caries process is multifactorial and it will be driven by changes to one or more aspects of the overall oral environment. This means that sustainable treatment outcomes can only be achieved by long term modification to the factor, or factors, that have changed in that environment.

The concept of using the colours red, yellow and green in a Traffic Light colour system to convey the different levels of risk has been used previously in both dentistry and health education. Some school canteens label sugar containing food and drinks red or yellow and those free of refined sugar are labelled green to raise awareness of the cariogenicity of these snacks. A dental manufacturer³ recently introduced the concept with a range of saliva diagnostic tests and preventive products to facilitate communication with patients.

Some myths and facts about caries

The current understanding of the development of caries has reached a stage where it is apparent that clinicians need to accept a new understanding of its diagnosis and treatment. At the same time some widely held myths about the nature and treatment of caries must be challenged.

Myth – The diagnosis of caries should be based upon the detection of cavities.

Fact – Caries is a transmissible bacterial infection and a multifactorial disease that reflects change in one or more significant factors in the total oral environment. Diagnosis involves recognition of those changes rather than simply noting cavities.

Myth – Placement of a restoration in a cavity is the primary effective method of treatment of caries.

Fact – The treatment of caries requires behavioural modification and/or chemical agents to rectify changes in the oral environment. Surgical intervention is required only when there is a physical defect or cavitation on the tooth surface. It may well be part of a comprehensive treatment plan but surgical intervention only removes the demineralised tissue and repairs defects. It is not a cure for caries.

Myth – There is one treatment regime that can be applied to all individuals.

Fact – Every individual has a unique oral environment, therefore treatment must be specific for the conditions that exist in that individual's mouth.

In order to manage caries effectively, there must be a clear understanding of

- all factors contributing to the development of caries
- accurate diagnosis of aberrant factors
- detection and assessment of active lesions

NOTE



Caries is the reflection of adverse changes occurring in the oral environment over time. Effective treatment will only be achieved by making long term, sustainable changes to the oral environment.

For each patient, the disease can only be managed by addressing all of the contributing factors.² This chapter reviews current understanding and management of the disease at the individual level and introduces a Traffic Light-Matrix system to personalise the relationship between the patient and the disease. This varies from the community based approach but it is recognised that there is a need to combine both to ensure the overall health of the population.⁴

Understanding the caries problem

There have been essentially three hypotheses advanced to explain the caries process:

• Non specific plaque hypothesis

This theory suggests the disease is the outcome of the activity of the micro-organisms found in plaque. The total acid production is important suggesting that diagnosis and

treatment are not aimed at any specific part of the microflora. Treatment is based on the principle that a clean tooth cannot decay because caries activity is the result of failure to remove plaque. This theory has been disproven.

• Specific plaque hypothesis

This theory suggests that only a few species of the organisms found in plaque contribute to the disease process so therefore plaque, per se, is not pathogenic. Only those individuals with high levels of specific bacteria such as *S. mutans* and *Lactobacilli*, can develop caries. Treatment is based on the reduction or elimination of the disease causing bacteria. This theory is faulty.

• Ecological plaque hypothesis

The current theory, widely accepted, suggests that caries results from a shift in the balance of the resident microflora driven by modifications in local environmental conditions. For example, repeated frequent sugar intake leads to recurring episodes of low pH in the biofilm and this will favour the growth of acidogenic and aciduric species, thus predisposing the individual to caries.⁵ That is to say, the composition and behaviour of the biofilm is a reflection of a modified environmental condition. Other modifications in the environment, such as changes to salivary flow may lead to the same result. Treatment begins by identifying and modifying any factors that are tilting the balance of the biofilm in favour of a cariogenic activity.⁶ To achieve long term success, treatment should be aimed at modifying all aspects of the oral environment over the long term.

The term oral biofilm

Oral biofilm is the term used to describe the total complex formed when pellicle adheres to the tooth surface and becomes populated with bacteria and their extracellular products. Demineralisation can only occur at the interface between the biofilm and the tooth surface. It grows continuously and, following removal by hygiene procedures, it will reform within minutes.

There will be natural metabolic fluctuations

within and beneath it leading to regular cycles of demineralisation and remineralisation of the tooth surface. These cycles can not be prevented because they are absolutely normal.⁷ Note that most of the ions released during a normal episode of demineralisation will be held within or under the biofilm and remain available for remineralisation. The metabolic fluctuations can be modified and therefore the demineralisation/remineralisation cycle can be modified. As long as the cycle is not in favour of demineralisation over too long a period of time, cavitation will not evolve. That is, both sides of the cycle must be in balance.

NOTE



The behaviour and composition of the biofilm are a reflection of the oral environment and caries is a reflection of adverse changes occurring in that environment.

Site specificity

A further element to be considered in the caries process is the tooth surface itself. Caries activity will not be evenly distributed throughout the mouth because some sites are more susceptible than others. This means treatment should include consideration of where the caries activity is occurring. To accommodate this, the Traffic Light-Matrix model differentiates between the three

types of tooth surface that vary in susceptibility to caries:

- **Exposed smooth surfaces**

All those surfaces that are exposed to good salivary flow and the mechanical cleansing effect of the tongue, lips and cheeks. They are readily accessible to tooth brushing and show the lowest caries incidence.

- **Hidden smooth surfaces**

Smooth surfaces that are less accessible to mechanical cleansing such as interproximal surfaces between all teeth. Other examples include lingual surfaces of lower molars, distal surfaces of the last teeth in the arch, occlusal surfaces of slowly erupting teeth (**Figures 6.1a and 6.1b**).

- **Pits and fissures**

Pits and fissures that appear on any surface, particularly occlusal surfaces. The enamel is structurally different from and matures more slowly than enamel on smooth surfaces (**Chapter 1**) and fissures cannot be completely cleaned with normal oral hygiene measures.

This suggests that if the exposed smooth surfaces are extensively carious the disease is rampant. On the other hand caries in the depths of fissures is so difficult to prevent that the occasional lesion should not cause undue alarm. Note also that a smooth surface may be hidden at a particular stage and exposed at another. For example the



Fig. 6.1a and 6.1b. Arrested root caries lesion on the lingual of a lone bicuspid in an area that is difficult to clean for an aging patient. A thin film of a low viscosity, high fluoride glass-ionomer has been flowed over the area to protect it and increase the fluoride content.

distal surface of the first molar will be exposed until the eruption of the second molar when it becomes hidden. A slowly erupting permanent molar may pose a problem but once eruption is completed the occlusal surface becomes exposed and relatively self cleansing (**Figure 6.2**).

Identifying the risk factors

The factors listed in the summary can all be significant in modification of the metabolic cycle in the biofilm and it is important to identify which of these are operating in concert in a particular case so that a management program can be prepared in a logical and effective manner.^{8,2} Medical and social history should be taken into account as well as the patient's dental history including exposure to fluoride. Also the intraoral factors such as salivary flow and plaque control will all play a part.

These are relevant to patients in all age groups, but the significance of the different factors will vary over time. For example, frequency of refined carbohydrate intake is more significant in youth but salivary factors become more dominant as the

NOTE

The dentist has two roles to play: first, as a physician to diagnose and manage the disease process, second, as a surgeon to manage and repair caries defects.



SUMMARY



The risk factors can be divided into two broad categories:

Primary factors

- the biological factors that act directly on the biofilm:
 - saliva
 - diet
 - fluoride

Modifying factors

- indirect influence on the biofilm:
 - socio economic status
 - lifestyle
 - past dental history
 - compliance history

The success of a treatment plan relies upon patient cooperation and that depends on the patient developing a good understanding of the factors that cause the disease. The dentist, therefore, has two distinct roles to play. First, as a physician to diagnose and assist the patient in understanding and managing the disease process. Second, as a surgeon to manage and repair defects on the tooth surfaces.

Assessment of risk factors and physical examination of tooth surfaces and the soft tissues are two essential elements of a complete diagnosis of the oral condition. At the start of a course of treatment both patient and dentist should agree on a set of well defined objectives. At the completion a further evaluation should be carried out, and the interval for recall determined according to the patient's risk profile.

It is not easy to affect changes to the modifying factors so the three primary factors are the most significant from a clinical point of view. The socio-economic status is a modifying factor that exerts

NOTE



Dental care is ongoing, so the identification and assessment of risk factors will need to be repeated periodically as part of a life time monitoring programme.



Fig. 6.2. Excessive plaque accumulates on the occlusal surface of an erupting molar because this surface is hidden from the patient's oral hygiene efforts. Once fully erupted it will become exposed and relatively self cleansing.

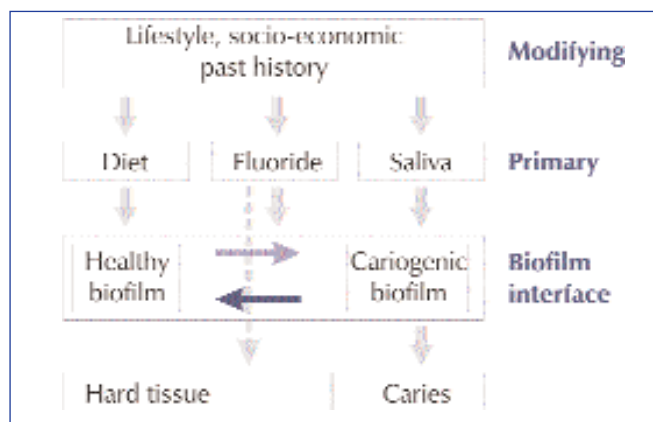


Fig. 6.3. The relationships between the primary factors, modifying factors and the behaviour of the biofilm in the caries process*.

an influence on the primary factors and can lead to change in the biofilm, but this is largely beyond the operators control.⁹ On the other hand the dentist may be able to influence lifestyle as long as the patient can be convinced of the value. The current understanding of the caries process is illustrated in **Figure 6.3**.

Traffic Light-Matrix (TL-M) Risk Assessment Model

Desirable features of a risk assessment model

The concept of risk assessment is fundamental to the effective treatment of caries. A treatment philosophy that is based on managing the oral environment must have the means of identifying and recording the factors that influence that environment. To be clinically useful a risk assessment model should have the following features:

- the risk indicators used must have a good correlation with caries incidence
- the tests should be accurate, produce rapid results and be technically simple
- the model must be capable of being used by hygienists and other auxiliaries
- an assessment of patient motivation and potential for compliance is essential

- the data collected should lead to a well defined clinical pathway

The Traffic Light system

The traffic light is the first element of this system and it builds on the existing risk assessment models as well as including an assessment of patient motivation and lifestyle activities. It does not attempt to predict caries incidence but rather it acts as an early warning system that alerts the clinician to the presence of risk factors that are capable of changing the oral environment. Few people can be considered to be caries free for their entire life as the status can change with the different challenges and circumstances over a life time. It is designed to help clinicians to select appropriate clinical pathways for individual patients based on their current personal risk profiles. It must be understood that the caries process needs to be continuously managed over the years at an individual level. There is very little research that describes how the interplay between the different risk factors influences the overall risk of developing caries at the individual level.

The TL-M model allocates a threshold value for each risk category. If the information elicited from questioning or by clinical testing yields results which exceed the predetermined threshold values the model alerts the clinician to a possible problem. The threshold values used have validity for individual risk factors but the model does not attempt to make any assumptions about either the relative importance of individual risk factors or their relationship to each other.

The model investigates sixteen risk factors and scores a red light, a yellow light or a green light for each risk factor depending upon predetermined criteria. For example, if the resting pH of unstimulated saliva is below 5.8, that particular risk factor scores a red light. This means that, even if all other salivary parameters test within acceptable limits, the red light for resting pH is enough to alert the clinician to a possible problem with either the quantity or quality of the saliva. Any restorative treatment carried out should be regarded as transitional until that problem is identified and, if

* Personal communication, Dr. Chris H. Sissons, Wellington, New Zealand

possible, corrected or compensated for. The system uses a specially designed form to record risk factors and the tests can be carried out either by a dentist or an auxiliary who has been trained to collect the data. The involvement of auxiliary personnel makes the model more economically attractive for everyday clinical practice.

The 16 risk factors used in the TL-M model

Each of the components involved in the risk assessment process is further divided into subcategories (Figure 6.4):

Saliva

1. ability of minor salivary glands to produce saliva
2. consistency of unstimulated (resting) saliva
3. pH of unstimulated saliva
4. stimulated salivary flow rate
5. buffering capacity of stimulated saliva

Diet

6. number of sugar exposures per day
7. number of acid exposures per day

Fluoride

8. past and current exposure

Oral biofilm

9. differential staining
10. composition

11. activity

Modifying factors

12. past and current dental status
13. past and current medical status
14. compliance
15. lifestyle
16. socio economic status

The Matrix

The second element of the TL-M model is the matrix. This is designed as a means of assessing the patient's present disease status and attitude to maintaining their own dental health. It is not intended to be anything more than a subjective assessment by the clinician on a particular day based on the clinician's instinctive understanding of that patient. However, collecting and re-collecting this information over a period of time gives the clinician a very useful measure of the patient's ability, or willingness, to comply with treatment directives. It is also a simple way to provide information about the potential for patient compliance between different operators in the same practice or upon referral to another practitioner.

Attitude towards dental health is scored as A, B or C on the vertical axis of the grid shown below. Current disease status is scored as 1, 2 or 3 and is recorded on the horizontal axis. The two scores position the patient in the grid (Figure 6.5).

The scoring criteria are listed on the next page.

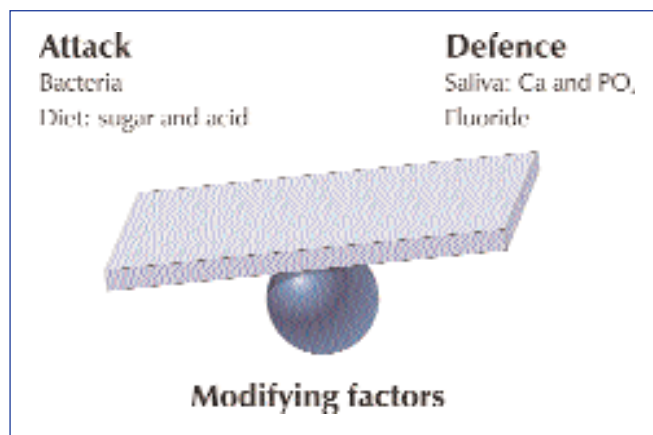


Fig. 6.4. There is a need to find a balance between the aetiological factors to obtain and retain stable oral health.

		DISEASE STATUS		
		1	2	3
ATTITUDE	a			
	b			
	c			

Fig. 6.5. Example of a Matrix, the ultimate goal of a clinician is to bring every patient into the A1 category.

Attitude

- a) **Self-motivated** – dentally aware and maintaining dental health is a high priority.
- b) **Dentally aware** – but still dependent on the dental team for motivation and help in staying healthy.
- c) **Unmotivated** – a low level of dental awareness and dental health is a low priority.

Disease status

1. **No apparent disease** – no treatment required at this time but there may or may not be restorations or evidence of past disease.
2. **Controlled disease** – there may be treatment required for functional reasons, such as defective restorations, but there is no sign of active disease.
3. **Active disease** – active disease is apparent either as new lesions or disease activity around existing restorations.

Risk Assessment for the Individual Patient

The Traffic Light-Matrix model (TL-M) is designed to assess the current level of disease and allow the development of a treatment regime tailored to specific needs. There have been other risk assessment and computerised caries management models developed in the past and the best known is the Cariogram.^{10,11} All models and all systems collect similar information but the TL-M model offers a more practical interpretation and application of that information. The previous models attempt to rank individual risk factors according to the relative importance by assigning a weighting to each risk factor. The information is then used to predict whether the individual is at low, moderate or high risk of new caries activity.

The TL-M model does not attempt to weight individual risk factors according to importance nor does it attempt to predict or quantify future risk. Its primary function is to alert the clinician to the immediate presence of a caries conducive environment so that the presence of one or more

risk factors can be taken into account in the diagnosis of disease and the formulation of a treatment plan for each patient. Being a manual system, the TL-M can be a very useful teaching and training system, the only requirement being a sound knowledge of the problems as well as appropriate solutions.

Factors to be assessed

As noted above, the following five components of the oral environment need to be assessed and analysed:

- saliva
- diet
- fluoride
- biofilm (plaque)
- modifying factors

Assessment of saliva

Saliva plays a major role in oral health and modification in salivary function may have detrimental effects on both hard and soft tissues as well as a negative impact on the quality of life of the patient (Chapter 7). Sreebny published an excellent review of the literature on this topic and the design of the saliva assessment is based on this paper.¹² Manufacturers of risk assessment kits have also published good communication tools on saliva³ and bacteria testing.¹³

SUMMARY



Saliva has three principal functions:

- to clear dietary acid and sugars from the mouth
- to buffer acid produced at the biofilm to tooth interface
- to provide a reservoir of ions for remineralisation

Whole saliva is a mixture of unstimulated and stimulated saliva. The production, composition and function of the two types are different so they need to be evaluated separately. The measurement of saliva should be included in a regular examination so that changes can be detected and the patient advised.

Unstimulated (resting) salivary flow rates will

fluctuate throughout the day and will be affected by a variety of causes. This means that, while a single observation may be indicative, it will not necessarily be conclusive and repeated observation at a similar time of the day and in similar circumstances may be required before making a finite assessment. The time of day is important because of diurnal variation and proximity to meals may have an effect. The pharmacokinetics of therapeutic medications may also be significant. This means xerostomia may be most marked in the period following absorption and distribution of a drug and then return to normal well before the next dose.

Regular investigation of the following properties are recommended:

unstimulated saliva (to be undertaken before the stimulated saliva test)

- functional efficiency of the minor salivary glands
- consistency of unstimulated saliva
- pH of unstimulated saliva

Salivary glands	Contribution
Submandibular	60%
Parotid	20%
Sublingual	5%
Minor	15%

Table 6.1. Contribution from different salivary glands to the total daily production of unstimulated saliva.¹⁷



Fig. 6.6. Droplets of saliva forming at the orifices of the minor salivary glands.

stimulated saliva

- flow rate
- buffering capacity

Function of the minor salivary glands

Unstimulated saliva is important for oral comfort as stimulated saliva is only produced during the short periods of mastication. The minor salivary glands account for 15 percent of the daily saliva production¹⁴ and the submandibular glands are the major contributor (**Table 6.1**). There is wide variation in the flow rate from the minor salivary glands located in different areas of the mouth. There may be a reduction in unstimulated flow rate from glands located in the palate as the patient ages but there are no age related changes from the glands located in the buccal and labial areas.¹⁵ This is why minor salivary glands located on the inside of the lower lip are generally selected for examination. Walsh described a simple way to assess the functionality of these minor salivary glands¹⁶ (**Chapter 7**).

Clinical steps

- Sit the patient upright.
- Roll the lower lip outwards and dry with a gauze square.
- Measure the time taken for droplets of saliva to appear at the orifices of the ducts of the minor glands (**Figure 6.6**).
- Apply a single ply of tissue paper to make it easier to see the droplets of saliva (**Figure 6.7**).

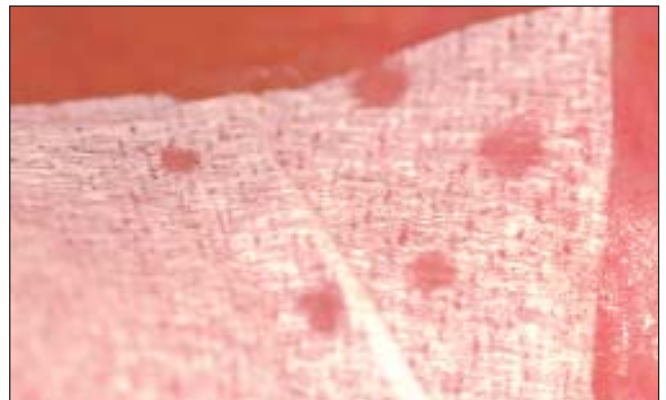


Fig. 6.7. A ply of tissue can be used to visualise the droplets.

Results and interpretations

A red light means apparently no minor salivary gland function which can be due to

- severe dehydration
- damage to the salivary glands by radiotherapy or pathology
- hormonal imbalance
- side effects of medication

A yellow light means a delay in saliva production. May be due to a mild level of

- dehydration
- side effects of medication

A green light means normal function.

In severe cases of salivary dysfunction, such as Sjögren's syndrome, the mucosa on the inside of the lip looks dry but can also show signs of trauma (Figure 6.8).

The time taken for the droplets to form will indicate the level of performance of the minor salivary glands.

Droplets of saliva need more than 60 seconds	Red	
Droplets of saliva appear between 30-60 seconds	Yellow	
Droplets of saliva appear in less than 30 seconds	Green	

Consistency of unstimulated saliva

Having noted the function of the minor salivary glands then check the unstimulated saliva itself. This is composed of 99% water and 1% proteins



Fig. 6.8. A case of Sjögren's syndrome showing extremely dry mucosa with small areas of trauma showing at the arrows.

and electrolytes¹⁸ so it should look clear, watery and contain a small amount of bubbles and have the ability to form a very thin coat on all hard and soft tissues. The thickness of the film will vary between 10-100 μm , depending on the different locations in the mouth.¹⁹ Viscosity should be graded using a web test. Use a tongue blade or dental mirror to lift pooled saliva from the floor of the mouth. As the instrument is withdrawn a web will form which, when stretched, will eventually break. Normal saliva can maintain a salivary web for 2-5 cm. but the web distance for the viscous saliva of the xerostomic patient can be as much as 15 cm.

Clinical steps

- Sit the patient upright.
- Ask the patient to stop swallowing for 30 seconds.
- Tilt the head slightly forward.
- Open the mouth and take note of appearance of the saliva (Figure 6.9).
- Asked the patient to touch the palate with the tip of the tongue.
- Check the appearance of the mucosa on the floor of the mouth and the formation of a shiny film of saliva (Figure 6.10).
- Perform a web test and note the result.

Results and interpretations

One of the important functions of saliva is to provide salivary clearance, that is, diluting and



Fig. 6.9. Healthy unstimulated saliva pooling in the anterior of the mouth some bubbles can be observed.

removing debris from the oral cavity. Ropey or frothy saliva has a lower water content and is therefore less protective to the hard and soft tissue for two reasons:

- it offers a lower salivary clearance rate
- it does not form an effective coating on the tooth surfaces.

Thick, ropey, frothy, extended web test	Red	
No visible pooling of saliva, a little sticky	Yellow	
Watery with pooling of saliva, shiny thin film on the floor of the mouth	Green	



Fig. 6.10. Healthy unstimulated saliva forms a thin and shiny film, coating the mucosa on the floor of the mouth. Some bubbles can be observed.



Fig. 6.11. Ropey, stringy unstimulated saliva.

pH of unstimulated saliva

The pH of unstimulated and stimulated saliva can differ by up to two pH units¹² and can range between pH5.3 to pH7.8.¹¹ As the tooth surface should be coated with a thin film of unstimulated saliva, it follows that the pH of this saliva will have an influence on the biofilm on the tooth surface.

BE AWARE

The hard and soft surfaces of the oral cavity are coated, not bathed, by saliva.

Clinical steps

- Collect a small sample of unstimulated saliva by asking the patient to dribble into a plastic container.
- Place a strip of pH paper into the saliva.
- After 10 seconds, check for pH level according to manufacturer's instructions³ (**Figure 6.13**).

Results and interpretations

The pH level of unstimulated saliva is a general indicator of the acid level of the oral environment. Normally the critical pH of hydroxyl apatite is 5.5 so the closer the pH of unstimulated saliva gets to this level the more chance there is for demineralisation.²⁰

A red light means that the patient's oral environment is highly acidic and urgent action should be undertaken to implement countermeasures.



Fig. 6.12. Frothy or bubbly unstimulated saliva.

Unstimulated saliva pH <5.8*	Red	
Unstimulated saliva pH = 5.8 to 6.8	Yellow	
Unstimulated saliva pH >6.8	Green	

Stimulated saliva flow rate

The composition of stimulated saliva is dependent on the flow rate and it represents the combined production from both major and minor glands. The average flow rate in a group of healthy adults shows a wide variation across the group but consistency within the individual. The mean flow rate is 1.6ml/min.²¹ Ask the patient to chew on a piece of paraffin wax for five minutes



Fig. 6.13. pH paper should be dipped into a small sample of unstimulated saliva for 10 seconds then compared with the colour chart supplied.

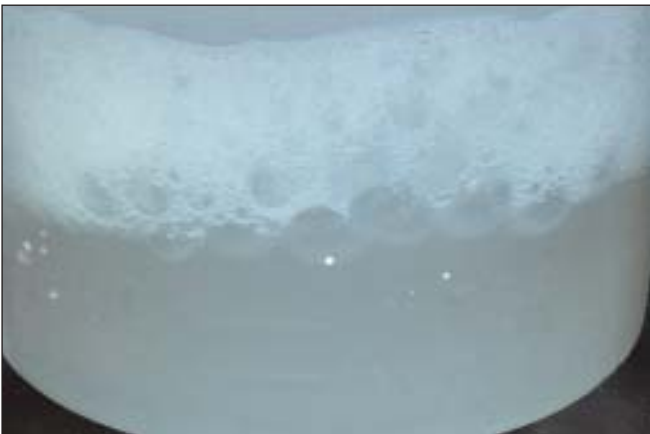


Fig. 6.14. Only measure the liquid portion of the stimulated saliva.

without swallowing and harvest the saliva in a plastic measuring cup. A stimulated salivary flow of 0.7ml/min. is accepted as the threshold below which there is an increased risk of caries.²²

Clinical steps

- Sit the patient upright.
- Ask them to chew on piece of non flavoured wax.
- Discard the first quantity of saliva after 30 seconds.
- Set the timer for five minutes then leave the patient alone chewing the wax.
- The patient must keep chewing for five minutes, spitting into a plastic measuring cup at regular intervals.
- At the end of the five minutes collect the measuring cup and note the volume.
- Prepare for buffering capacity test and bacteria count (**Figure 6.14**).

Results and interpretations

Ericsson et al categorised stimulated saliva flow into three groups: very low, low and normal.²³ This has been adapted into the traffic light system for ease of communication.

* GC Corp recommendations

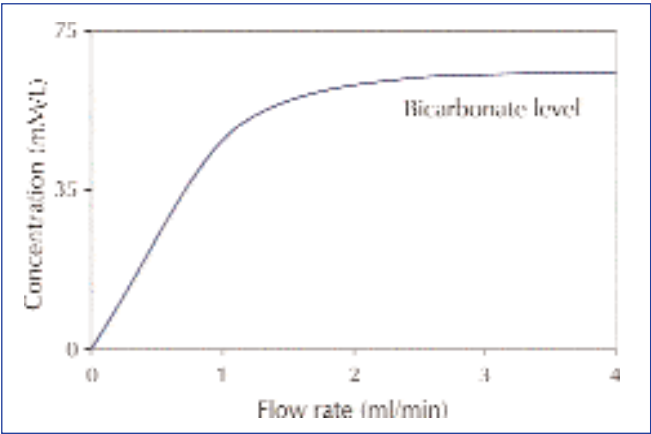


Fig. 6.15. The concentration of bicarbonate increases with high flow rate of saliva.

Stimulated saliva after 5 min <3.5ml	Red	
Stimulated saliva after 5 min. = 3.5 to 5 ml	Yellow	
Stimulated saliva after 5 min > 5ml	Green	

Buffering capacity of stimulated saliva

Buffering capacity is a measure of the ability of saliva to neutralise acid and this depends upon the concentration of bicarbonate. At very high flow rates, the level of bicarbonate concentration in some patients can exceed the plasma level. As illustrated in **Figure 6.15**, the concentration of bicarbonate in stimulated saliva is greatest at the start of a masticatory cycle.

Clinical steps

- Take a sample of the saliva collected for the stimulated flow rate test.
- Thoroughly wet the pads on the test strip with saliva.
- Remove excess saliva on the pads by placing the strip at 90° to a tissue to ensure a constant volume (**Figure 6.16**).
- Allow the strip to stand for five minutes. As the buffering effect is time dependant, it is important to read the test at the five minutes mark.
- Compare the colour with the standard and assign a score based on the manufacturer's instruction.

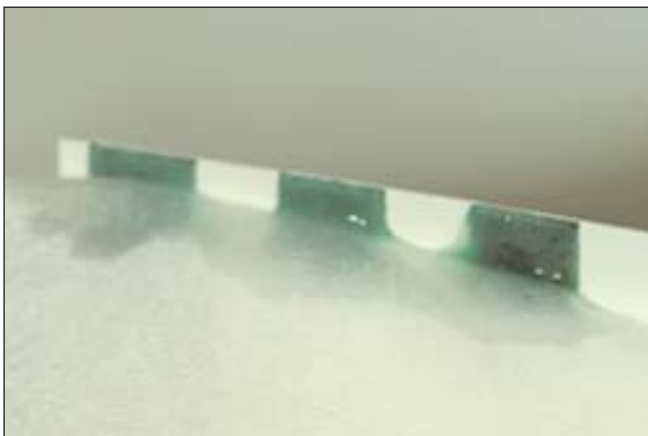


Fig. 6.16. Saturate the pre-acidified pads with stimulated saliva then use a tissue to remove the excess saliva.

Results and interpretations

There are two systems readily available at this time for measuring buffering capacity of stimulated saliva: CRT Buffer (Vivadent) and Saliva Check Buffer (GC Corp)

- Vivadent: the buffering capacity can be scored as high, moderate or low with only one test pad.
- GC Corp: there are three separate pads with different levels of acid built in with a numerical scoring system and a high level of sensitivity.

The results from both of these tests can be translated into the TL-M scale.

GC Corp	Vivadent		
Final score 0-5	Low	Red	
Final score 6-9	Moderate	Yellow	
Final score 10-12	High	Green	
The buffering capacity as scored using the GC and Vivadent system.			

Assessment of the biofilm

The bacteria most commonly implicated in the caries process, *S. mutans*, *S. sobrinis* and *Lactobacilli*, belong to a group of approximately 40 species in this population, all of which have a cariogenic potential and they need to be identified and controlled. About 50% of the organisms in plaque cannot be cultured with current laboratory technology.⁴ They interact with neighbouring cells and they function as a coordinated and metabolically integrated microbial community.²⁴

The matrix of polysaccharide varies in density so there are concentration gradients that will influence the diffusion properties of the mature biofilm.²⁵ This means the thicker the biofilm the more difficult it is for saliva to buffer the acids within. It is possible to roughly gauge the diffusion gradient with a disclosing gel which stains thin plaque red and thick plaque blue (**Figures 6.17 and 6.18**).

Streptococcus mutans

Streptococcus mutans is a general term describing several closely related species of streptococcus. The specific name *S. mutans* is now given to

human isolates and it is the species most commonly found in human dental plaque. There is an association²⁶ between the salivary level of *S. mutans* and the number of carious lesions and it is a particularly accurate predictor of caries activity in small children.²⁷

SUMMARY



Relationship between salivary *S. mutans* count and caries:

- concentration of *S. mutans* in stimulated saliva reflects concentration in biofilm
- low *S. mutans* count in saliva suggests a low level of caries activity
- high *S. mutans* count in saliva is possible with **high or low** caries activity

Lactobacilli

Lactobacilli require retentive sites and are therefore found in deeper lesions. The population level is strongly influenced by dietary sugar intake and, in the absence of open lesions, a high level suggests a diet high in fermentable carbohydrate.

Test methods

The test currently available for both of these species is semiquantitative and involves placing a saliva sample on a culture medium and incubating for forty eight hours (**Figure 6.19**). The bacterial count is obtained by comparing the culture to a chart supplied by the manufacturer. There are

clinical limitations because of the time needed to incubate the sample and correct interpretation of the results requires training.

Clinical steps

- Take a sample of the saliva collected for the stimulated flow rate test.
- Thoroughly wet both sides of the test strip.
- Place the NaHCO₃ tablet into the container.
- Close the container tightly and place into incubator for 48 hours.
- Read the results and discard the strip safely.

The next generation of tests will be based on the use of monoclonal antibodies or enzymes and is expected to provide rapid chairside results.

Results and interpretations

The results must be recognised as being semi-quantitative only and the counting of the culture strip can be subjective. A red light in both *S. mutans* and *Lactobacilli* suggests a high caries risk.^{28,29}

<i>S. mutans</i>	<i>Lactobacilli</i>		
>10 ⁶ CFU	>10 ⁵ CFU	Red	
Not applicable	Not applicable	Yellow	
<10 ⁵ CFU	<10 ⁴ CFU	Green	
Caries risk levels according to the CRT system from Vivadent (Vivadent 2003).			



Fig. 6.17. Stagnant plaque suggests an increased level of localised risk and the rich colours may encourage the patient to engage in better self care routines.



Fig. 6.18. Two colour disclosing gel stains thick plaque (stagnant) blue and thin plaque red.

Dietary analysis and caries risk

The positive correlation between caries and the frequency of refined carbohydrate intake versus the total amount of fermentable sugar is well documented.^{30,31,32} In this case, the term 'frequency' refers to the number of times per day these types of food are eaten. However, it is also important to look at the way the food is consumed. For example, if a soft drink is consumed over a long period of time by taking numerous small sips there will be a higher risk when compared with drinking quickly within a short period of time. The risk increases dramatically when combined with poor plaque control and a lack of fluoride intake.

The term diet analysis can be misleading so it is important that patients understand the true purpose of this exercise. Some 'over-the-counter' (OTC) medications contain a high level of sugar but are not considered as food or drink. Some of the cough lozenges or breath freshener products are high in sugar and are designed to remain in the oral environment for extended periods.

Sugar

The change in plaque pH is time dependent as illustrated by the series of Stephan curves in **Figure 6.20**. This means that, following intake of sugar, the pH of the biofilm drops sharply and may take over 60 minutes to regain its original value.³³



Fig. 6.19. A culture test for *S. mutans* and *Lactobacilli* by Vivadent requires 48 hours of incubation.

There is overwhelming evidence that frequent intake of fermentable carbohydrate increases the risk of caries and individuals with high sugar diets consistently show elevated levels of *S. mutans* and *Lactobacilli*. The Vipeholm study provided evidence that the frequency of consumption is directly related to caries activity.³⁰ It has also been shown that chewing sugared gum twice a day leads to a significantly higher caries increment after 24 months.³⁴ Based on this the TL-M system assigns a red light to a patient with more than two exposures to sugar in between the main meals.

Acid

Dietary sources of acid, such as carbonated soft drinks, fruit juices and sports drinks, have pH lower than the critical pH of apatite (pH=5.5) and they may lead to increased caries and erosion (**Chapter 5**). Many of these sources also contain significant concentrations of sugar resulting in a synergistic effect thus markedly increasing caries risk.

Clinical steps

- Patients keep a record of what goes into their mouth for five days i.e. three weekdays and over a weekend.
- Avoid using term 'diet analysis' as it tends to exclude non-food sources of sugar and acid such as medication, sport drinks etc.

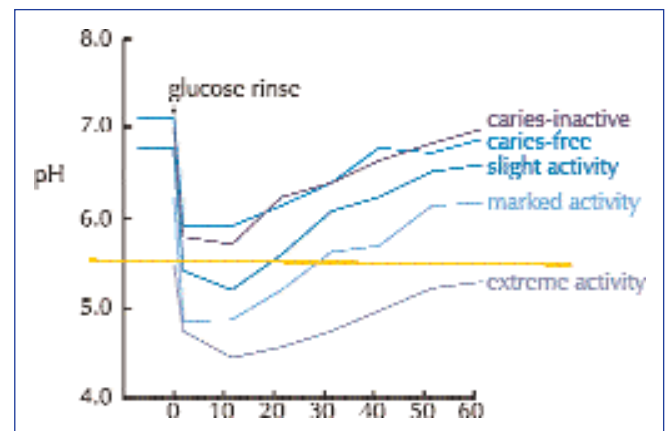


Fig. 6.20. A drop in pH in plaque after a 10% glucose rinse (adapted from Nikiforuk, 1985).

- Highlight the sugar and acid exposure on the record sheet with
 - red for every sugar exposure in between meals,
 - yellow for every acid exposure in between meals.
- Use a unit of 30 minutes per tick. For example,
 - a cola drink score one tick for sugar and one tick for acid as these drinks have a low pH,
 - consuming a cola drink and sucking on a sweet at the same time score one tick for sugar and one tick for acid,
 - drinking tea/coffee with sugar over a period of one hour score two ticks.
- The number of between meal exposures to sugar and acid over the five days will be used to classify the patient's risk profile.

Results and interpretations

Food consumption and a caries lesion are events that occur in a different time frame. Therefore changing a dietary routine can not produce an immediate response because the cavities found today are the outcome of a dietary pattern of the past. This is very important if the patient is to accept the correlation. As caries is a multifactorial and complex disease, the classification below should be interpreted with due consideration for the other factors.

Sugar: exposure in between meals	Acid: exposure in between meals	
>2	>3	Red
>1	>2	Yellow
Nil	1	Green

Fluoride

There is a discussion about fluoride in **Chapter 4**. This segment will offer a brief summary on fluoride use related to the TL-M system.

Fluoride provides protection against caries at three levels:

- it increases the resistance of enamel to demineralisation
- it enhances the reservoir of ions for remineralisation

- it interferes with bacterial cell metabolism in the plaque

It is now accepted that constant exposure to low levels of fluoride in drinking water and toothpaste will have an effect on enamel throughout the life of a tooth. Note that for smooth surface lesions, fluoride in toothpaste may be more beneficial than drinking fluoridated water. However, the effectiveness of fluoride is reduced as the pH of the saliva falls and this has important implications when interpreting the interrelationship of the risk factors.

Exposure to fluoride		
No fluoridated water or toothpaste	Red	
Either fluoridated water or toothpaste	Yellow	
Both fluoridated water and toothpaste	Green	

Modifying factors

Dental history

The presence of restorations and/or active caries lesions is the strongest evidence of high caries activity even if they are identified as white spot lesions only. Useful information can often be derived from the location and pattern of these lesions. For example, active lesions in regions where there is normally saliva accumulation, such as lower anteriors or buccal surfaces of upper molars, tends to indicate high caries activity.

Lifestyle

Lifestyle can be defined as a set of consciously chosen personal behaviour patterns (**Chapter 7**). While it does not actually cause caries, it may predispose the patient through behaviour and environment to an increased risk of developing caries or non-caries tooth loss, often in a most unexpected manner. The patient will not necessarily relate the behaviour to problems in the oral cavity and a high level of education may be necessary to develop recognition.

The following list covers the obvious lines of enquiry the dentist should follow to try to elicit the required information to lead to a correct diagnosis. This is covered in detail in **Chapter 7** and this list is offered only to draw attention to the detail:

- **Medical history**

There are numerous medical conditions and medications which can result in altered salivary function. Common ones include diabetes, depression and rheumatoid conditions such as Sjögren's syndrome, rheumatoid arthritis and irradiation of salivary glands for head and neck cancer.

- **Prescription medications**

Hundreds of prescription medications lead to reduced salivary flow as a side effect. The most common of these are anti-depressants, anti-hypertensives, anti-cholinergics, anti-psychotics, diuretics and anti-Parkinsonian drugs.

- **Self-administered medications – over the counter (OTC)**

The active ingredients of many of these reduce salivary flow and may be coated with sucrose. Some are meant to be held in the mouth over periods of time so the ambient pH can be lowered for extended periods. Hay fever and asthma medications will affect saliva flow and lower the pH.

- **Recreational drugs**

Caffeine, tobacco, alcohol, marijuana and amphetamines all reduce saliva flow and are addictive. Cola style soft drinks and high-energy athletic drinks are particularly destructive as they contain significant amounts of caffeine and create an addiction to a product which has high sugar levels and low pH. Even the so called diet or sugar free varieties have a significantly low pH and are therefore dangerous.

Patients have a very restricted view of what they consider to be medications and they have no idea of the potentially harmful effects of many of the substances they ingest. It is therefore, extremely important that a careful history be taken if the risk factors are to be fully and accurately determined.

Socio economic status

Social factors can have an overriding influence on general health status and this is especially true for multifactorial and complex problems such as diabetes and cardiovascular disease. Children

from lower socio economic strata whose parents have low educational levels are at a much greater risk of developing caries and the potential for changing their lifestyle is influenced by the parents' level of understanding and motivation. However this is difficult to assess and it is most unwise to jump to conclusions.

The effect of financial stringency can affect dental treatment at two levels:

- compliance: is there spare money in the family budget for the patient to comply with the clinician's instructions? This factor cannot be readily changed so it is important to devise acceptable home and professional care regimes,
- indirect costs: for some patients, even a free visit to the surgery can be expensive in terms of loss of earnings, child care and travel expenses.

Compliance

While it is important to discover as much as possible about the various risk factors it is more important that the patient is willing to make the necessary changes to eliminate or at least modify these factors. Compliance is dependent on two factors:

- patient attitude to improving their own oral health, that is, the patient has to take ownership of the problems,
- ability of the dental team to devise an acceptable treatment regime.

Effective management is ongoing and requires continuous monitoring. A course of treatment will have an element of both non operative and operative treatment. There should be clear objectives which are discussed and accepted by the patient and at the conclusion of a course of treatment, both the dental team and the patient must evaluate the outcomes and decide on the next course of action. The patient must be made aware of the dynamic and changing nature of their relative risk and disease activity.

Clinical Application of TL-M

Advantages of TL-M model

The TL-M model offers a significant number of advantages to the clinician who wants to apply modern concepts of caries treatment. The TL-M model provides a systematic approach to the collection of risk assessment data using a standardised checklist for each patient.

- There is a good correlation between the identified risk factors and the potential to develop caries.
- The tests used are straightforward and the collection of data can be carried out by auxiliaries.
- Because the system does not involve a lot of clinical time it is cost effective.
- The model is an excellent communication tool between dentists, auxiliaries and their patients.
- The involvement of auxiliary staff in the collection of data improves team morale which in turn creates an effective dental team. All staff members feel that they are helping to produce good treatment outcomes for patients rather than simply observing the process.
- The TL-M model collects data from individual patients and allows the development of personalised treatment solutions.
- The model provides a simple way to monitor patient progress towards the ultimate goal of managing their own oral health.

The intelligent use of the TL-M model allows the clinician to design a suitable treatment plan for problems ranging from a full mouth rehabilitation to a specific lesion on a particular tooth. Because the treatment plan is based on the systematic collection of clinically relevant information a successful treatment outcome is more likely to be achieved.

SAFE against BAD

It is important to engage patients in the treatment of their own disease of caries because a high level of compliance is required for success. Because of the multifactorial nature, effective control may

require major lifestyle changes which can only be achieved through education and effective communication. A useful tool, in the form of two sets of acronym has been designed to assist in explaining the major aetiological factors and their interactions so that a mutually acceptable treatment plan can be designed and monitored (**Figure 6.21**).

To manage the disease successfully, both dentist and patient must continuously juggle these two sets of factors, maximising the SAFE and minimising the BAD sides of the equation.

There is no simple and universal solution because each patient displays a unique combination so the treatment plan must be customised to their particular situation. However, the following guidelines can assist in leading to a consensus:

- the patient needs to identify the areas that they can readily change
- treatment recommendations must have a definitive end point with clear objectives which are acceptable to both the patient and clinician
- the effectiveness of the recommendations must be closely monitored
- progress over time must be noted and approved to maintain enthusiasm

Managing caries is a lifetime process in which a healthy oral environment can be achieved by tipping the balance in favour of remineralisation by ensuring that the SAFE factors can overcome the effects of the BAD.

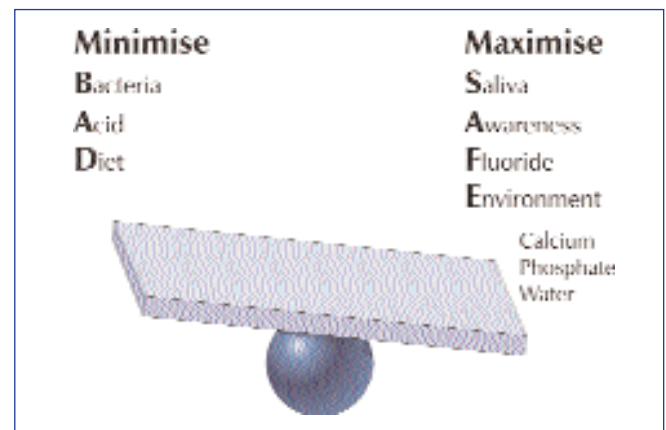


Figure 6.21. It is essential to achieve a balance between the BAD factors and the SAFE factors to obtain and retain stable oral health.

Charts

The charts on the following two pages are samples of the type of records that can be used for the assessment of a patient. They are set up in this manner so that the reader can photocopy them and modify them to suit their purpose. There are many variations possible and the operator is encouraged to incorporate some related form of record in to their practice routine and to repeat the record periodically depending upon the response from the patient.

Summary

The current understanding of the caries process and the factors which influence that process have led to a radical reappraisal of how the caries susceptible patient should be treated. It is essential to understand that the presence of caries should not be recognised simply through the presence of cavities. The disease has been present for some time before surface cavitation becomes apparent. Caries is a reflection of change in the oral environment and the risk factors that are capable of inducing that change, either directly or indirectly,

and are clearly identifiable shortly after they first occur. This knowledge is not new but until recently it has not been widely applied in the practice of everyday clinical dentistry.

One of the factors preventing the routine application of this knowledge in the past was the lack of a systematic risk assessment model that provided relevant information about caries risk factors and was both simple to use and cost effective. The TL-M model meets all of the desirable criteria. It is clinically relevant because the information collected can be used for early identification of changes occurring in the oral environment. The identification of these changes can then be adapted to the production of a specific treatment regime designed to meet the needs of each individual patient.

Acknowledgement

The authors would like to express their appreciation to Dr. Adrian Gaffney and Dr. Tony McLaughlan for their assistance in formulating the Traffic Light-Matrix system, as described in this chapter.

Risk Assessment for Caries

Patient: _____	Date: _____
DOB: _____	Clinician: _____

MATRIX: Attitude and Disease Status

		CARIES RATE		
		1	2	3
ATTITUDE	a			
	b			
	c			

NOTE: Patient's Matrix score should be reassessed regularly

PATIENT's self-assessment of attitude

Q: Are you willing to change the way you care for your oral health?

A: If answer YES assign **A**
 NOT SURE assign **B**
 NO assign **C**

CLINICIAN's assessment of disease

- No evidence of current caries activity **1**
- No current caries activity but need for restorative care due to structural breakdown **2**
- Active disease present **3**

RISK FACTORS

Saliva

Unstimulated

Minor salivary gland function

Droplet of saliva requires > 60s	R
Droplet of saliva requires < 60s	Y
Droplet of saliva requires < 30s	G

Stimulated

Flow rate

Stimulated saliva after 5 min <3.5mL	R
Stimulated saliva after 5 min >3.5 <5.0	Y
Stimulated saliva after 5 min > 5mL	G

Consistency

Thick, ropey, frothy	R
Sticky, no visible pooling	Y
Watery, shiny film on floor of mouth	G

Flow rate

GC	Vivadent	
Score: 0-5	Low	R
Score: 6-9	Moderate	Y
Score: 10-12	High	G

Resting pH

Unstimulated saliva pH < 5.8	R
Unstimulated saliva pH > 5.8 & <6.8	Y
Unstimulated saliva pH > 6.8	G

Plaque (biofilm)

Activity (preliminary check)

Two-tone disclosing gel

Blue	R
Not applicable	Y
Red	G

Composition

Composition (based on CRT from Vivadent)

<i>S. mutans</i>	<i>Lactobacilli</i>	
> 10 ⁶ CFU	> 10 ⁵ CFU	R
Not applicable	Not applicable	Y
< 10 ⁵ CFU	< 10 ⁴ CFU	G

Diet (four-day history)

Number of exposure in between meals

Sugar	Acid	
> 2	> 3	R
> 1	> 2	Y
Nil	< 2	G

Fluoride

NO fluoridated water or tooth paste	R
EITHER fluoridated water or tooth paste	Y
BOTH fluoridated water and tooth paste	G

Modifying factors

Q1: Any drugs which can decrease salivary flow (prescribed/OTC/recreational)?

Q2: Any diseases which can cause dry mouth?

Q3: Any fixed or removable prostheses (including orthodontic appliance)?

Q4: Is compliance likely to be poor?

Q5: Does patient have a recent episode of active caries?

YES to ANY ONE of the above questions	R
Not applicable	Y
NO to ALL the above questions	G

OVERALL TRAFFIC LIGHT ASSESSMENT

	G	Y	R
Saliva			
Plaque			
Diet			
Fluoride			
Modifying factors			

MANAGEMENT STRATEGIES

Minimise

Bacteria

Acid

Diet

Maximise

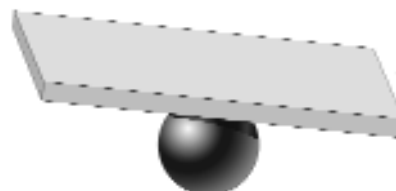
Saliva

Awareness

Fluoride

Environment

Calcium
Phosphate
Water



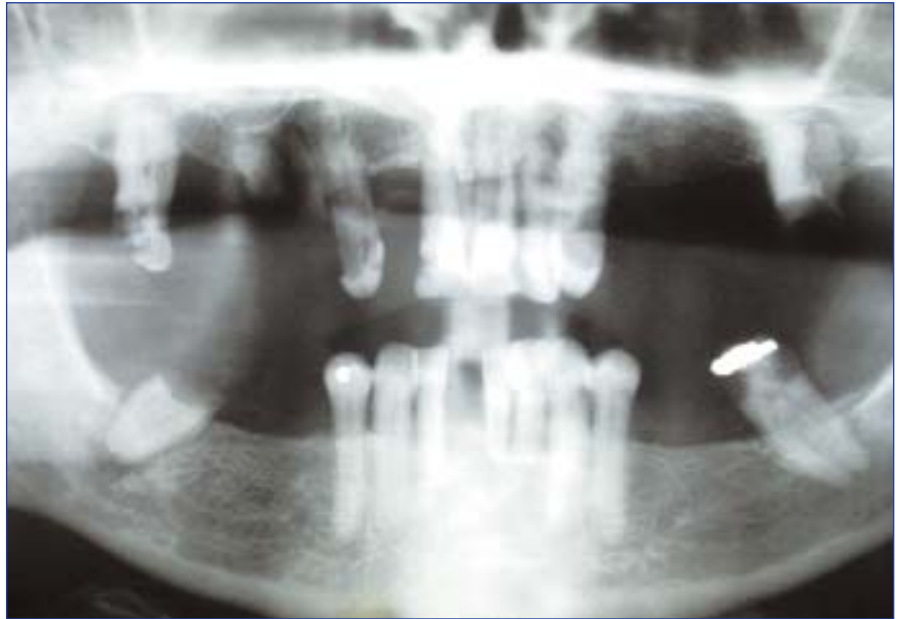
Further Reading

1. Network, S. I. G. *Targeted Caries Prevention in 6 to 16-year-olds Attending for Dental Care*. Edinburgh: Scottish Intercollegiate Guidelines Network; 2000.
2. Kidd, E. and B. Nyvad. Caries control for the individual patient in O. Fejerskov and E. Kidd, *Dental Caries: The Disease and its Clinical Management*. Oxford: Blackwell & Munksgaard; 2003; 303-312.
3. GC Corporation. *Saliva Resting: Good Practice, Good Sense*. Tokyo: GC Corporation; 2002.
4. Pitts, N. Are we ready to move from operative to non-operative/preventive treatment of dental caries in clinical practice? *Caries Res* 2004; 38:294-304.
5. Marsh, P. D. The oral microflora and biofilm on teeth in O. Fejerskov and E. Kidd, *Dental Caries: The Disease and its Clinical Management*. Oxford: Blackwell & Munksgaard; 2003; 29-47.
6. Marsh, P. D. Microbial ecology of dental plaque and its significance in health and disease. *Adv Dent Res* 1994; 8:263-271.
7. Fejerskov, O. Concepts of dental caries and their consequences for understanding the disease. *Comm Dent Oral Epidemiol* 1997; 25:5-12.
8. Bratthall, D. and G. Tynelius-Bratthall. Diagnostics as basis of causal treatment: tools and tests for evaluation of caries and periodontal diseases in A. E. Wolff, *Professional Prevention in Dentistry*. Baltimore: Williams & Wilkins; 1994; 31-68.
9. Hunt, R. J. Behavioral and sociodemographic risk factors for caries in J. D. Bader, *Risk Assessment in Dentistry*. Chapel Hill: University of North Carolina Dental Ecology; 1990; 29-34.
10. Bratthall, D. Dental caries: intervened-interrupted-interpreted. Concluding remarks and cariography. *Eur J Oral Sci* 1996; 104:486-491.
11. Suddick, R. P. and M. W. Dodds. Caries activity estimates and implications: insights into risk versus activity. *J Dent Educ* 1997; 61:876-884.
12. Sreebny, L. M. Saliva in health and disease: an appraisal and update. *Inter Dent J* 2000; 42:287-304.
13. Vivadent, I. *CRT Bacteria Caries Risk Test*. Schaan: Ivoclar Vivadent AG; 2003.
14. Dawes C. Factors influencing salivary flow rate and composition. Saliva and Oral Health. W. M. Edgar and D. M. O'Mullane. London, *Br Dent J* 1996; 27-41.
15. Shern, R. J., Fox PC, et al. Influence of age on the secretory rates of the human minor salivary glands and whole saliva. *Arch Oral Biol* 1993; 38: 755-761.
16. Walsh, L. Preventive dentistry for the general dental practitioner. *Aust Dent J* 2000; 45: 76-82.
17. Dawes, C. Factors influencing salivary flow rate and composition in W. M. Edgar and D. M. O'Mullane, *Saliva and Oral Health*. London: British Dental Association; 1996; 27-41.
18. Nauntofte, B, Tenovuo J. O, et al. Secretion and composition of saliva in O. Fejerskov and E. Kidd, *Dental Caries: the Disease and its Clinical Management*. Oxford: Blackwell & Munksgaard; 2003.
19. Tenovuo, J. Saliva in A. Thylstrup and O. Fejerskov, *Textbook of Clinical Cariology*. Copenhagen: Munksgaard, 1996; 17-43.
20. Dawes, C. What is the critical pH and why does a tooth dissolve in acid. *J Can Dent Assoc* 2003; 69:722-724
21. Heintze, U, Birkhed D, et al. Secretion rate and buffer effect of resting and stimulated whole saliva as a function of age and sex. *Swed Dent J* 1983; 7:227-238.
22. Ericsson, V, Hardwick I. Individual diagnosis, prognosis and counseling for caries prevention. *Caries Res* 1978; 12 (Supplement): 94.
23. Marsh, P. D, Bradshaw D. J. Microbial community aspects of dental plaque in H. N. Newman and M. Wilson, *Dental Plaque Revisited: Oral Biofilm in Health and Disease*. Cardiff: Bioline; 1999; 237-253
24. Wood, S. R, Kirkham J, et al. Architecture of intact natural human plaque biofilms studied by confocal laser scanning microscopy. *J Dent Res* 2000; 70:21-27.
25. Beighton D. The value of salivary bacterial counts in the prediction of caries activity in N. W. Johnson, *Risk Markers for Oral Diseases*. Cambridge: Cambridge University Press; 1991; 1:313-326.
26. Hausen, H. Caries prediction – state of the art. *Community Dent Oral Epidemiol* 1997; 25:87-96.
27. Anderson M. H, Bales D. J., et al. Modern management of dental caries: the cutting edge is not the dental bur [see comments]. *J Am Dent Assoc* 1993; 124:36-44.
28. Krasse, B. Caries risk: a practical guide for assessment and control. Chicago: Quintessence Publishing; 1985.
29. Gustafsson, B. E, Quensel C. E., et al. The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. *Acta Odontol Scand* 1954; 11:232-264.
30. Sreebny, L. M. Sugar and human dental caries. *World Rev Nutr Diet* 1982; 40: 19-65.
31. Karlsbeek, H, Verrips G. H. Consumption of sweet snacks and caries experience of primary school children. *Caries Res* 1994; 28:477-483.
32. Nikiforuk, G. *Understanding Dental Caries/Etiology and Mechanisms, Basic and Clinical Aspects*. Basel: Karger. 1985
33. Glass, R. L. Effects on dental caries incidence of frequent ingestion of small amount of sugars and stannous EDTA in chewing gum. *Caries Res* 1981; 15:368-372.

7 Lifestyle Impacts on Oral Health

L. J. Walsh

The dental clinician needs a keen insight into the various ways in which a patient's lifestyle may interact with and influence their oral health. A proper history and evaluation of a dental patient should address not only their medical history but should also routinely examine lifestyle issues such as smoking and the intake of water, alcohol, and caffeine. Similarly, additional questions for the dental patient with an obvious caries problem should include use of non-prescription medicines, and dietary intakes of acidic foodstuffs as well as fermentable carbohydrates.



The Importance of Saliva

Saliva performs a multiplicity of roles within the oral cavity. Regrettably, its importance is usually not appreciated until it is absent. Salivary dysfunction is a common problem and is frequently undiagnosed, at least in part, because the patient's symptoms are not a reliable indicator of salivary gland function. Patients will rarely be aware of diminished output until the resting flow rate is less than half the normal rate¹ (**Figure 7.1**).

The functions of saliva include

- lubricating the oral tissues for swallowing and speech,
- assisting the sense of taste by acting as a solvent for ions and through proteins such as gustin,
- maintaining health of the oral mucosa through growth factors to promote wound healing, and cystatins to inhibit destructive enzymes e.g. cysteine proteases,
- assisting in digestion through amylase and lipase,
- dilution and clearing of material from the oral cavity,
- buffering acids from dental plaque,
- buffering weak acids from food and drink e.g. wine or black cola soft drinks,
- buffering short term exposure to strong acids e.g. reflux and vomiting,
- serving as a reservoir for the ions calcium, phosphorus, and fluoride used in remineralisation,
- controlling oral microflora through immunological (IgA), enzymatic, peptide and chemical mediators.

Salivary flow is stimulated by the taste and mastication of food. The increased flow will lead to a raised pH with an increase in bicarbonates and, therefore, an increase in buffering capacity. Calcium and phosphate levels will also rise and this will influence the balance between demineralisation and remineralisation of tooth structure. There will also be an improved clearance of food debris due to more rapid movement of the salivary film as well as greater activity of the antimi-

crobial mechanisms available from saliva.

Reductions in the quantity of salivary secretions or changes in the properties of saliva are responsible for a host of related oral and dental problems which impact directly upon quality of life. These include

- difficulties in chewing and swallowing food
- difficulties in speech (dysphonia)
- alterations in taste (dysgeusia)
- increased plaque formation
- increased risk of caries
- increased risk of erosion and tooth wear
- cervical dentinal hypersensitivity
- mucosal abrasions and mucosal irritation
- halitosis
- oral fungal infections
- impaired retention of complete maxillary dentures
- lack of lubrication beneath complete mandibular dentures

These oral problems may in turn influence medical status, in that patients may lose interest in eating and suffer from poor nutrition. Because salivary dysfunction can have numerous implications

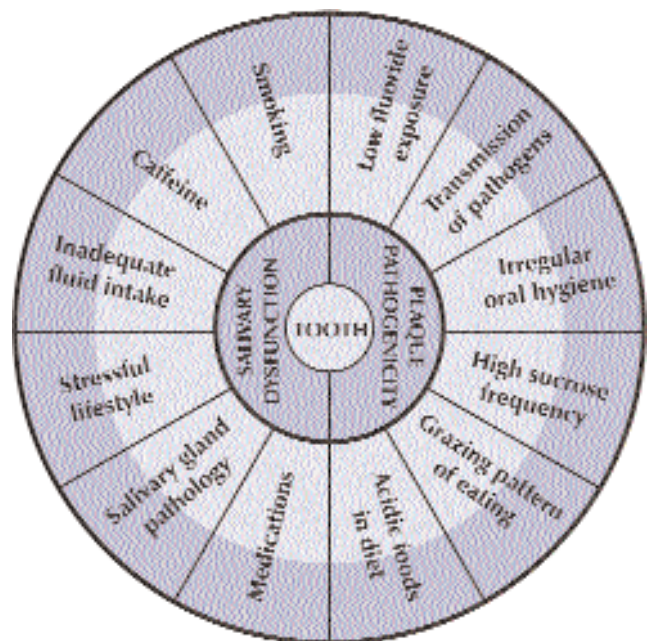


Fig. 7.1. Caries Causation: Schematic representation of lifestyle, behavioural and medical factors which reduce protective properties of saliva or increase pathogenicity of plaque. Increase the factors and increase the caries rate.

for general as well as oral health, screening for salivary parameters should be an essential component of patient assessment.

Components of saliva

Saliva is a composite of the secretions of major and minor salivary glands as well as material derived from the gingival sulcus, a point which has diagnostic relevance in terms of salivary markers of periodontal destruction. The composition varies from site to site within the mouth, and is also related to the time of day and proximity to meals. Its properties are affected by both the overall level of hydration and the general health of the individual.

Saliva is derived from the blood and is therefore a filtrate of serum. This means that production is linked to the fluid balance of the body and that blood flow through the salivary glands from branches of the maxillary and other arteries has a major effect. Approximately 99% of the volume is water, and this serves as the solvent for the other components of the saliva.

NOTE



Total flow rate, stimulated + unstimulated = 500 to 1500 mL per day
Average volume of resting saliva present in the oral cavity = 1 mL.

The resting saliva arises from

- submandibular glands (60%)
- sublingual glands (5%)
- parotid glands (20%)
- minor glands (15%)

Parotid saliva (serous saliva) is high in bicarbonate ions and amylase, while submandibular gland secretions (mucinous saliva) is high in mucins and calcium. The concentration of calcium in submandibular saliva (3.7 mmol/L) is considerably higher than that found in plasma (2.5 mmol/L) or in pooled whole saliva (1.35 mmol/L).

An increased salivary flow causes the oral environment to become alkaline, and this will lead to mineralisation of foci within supragingival plaque and the formation of dental calculus. The higher

pH allows formation of highly ionised phosphate ions (PO_4^{3-}) in the saliva and plaque because of breakdown of organic phosphates by salivary phosphatase enzymes. Patients with cystic fibrosis have modified exocrine gland function leading to increased levels of Ca^{2+} and PO_4^{3-} in their submandibular saliva and may therefore show a marked tendency to form supragingival calculus.

Salivary enzymes and mucins

Saliva is such a complex biological fluid that it is practically impossible to replicate it from individual components. It has a limited role in assisting digestion, beginning with a pre-digestive function mediated by a number of enzymes, including amylase, lipase, and a range of proteases and nucleases. Amylase can break down starch and glycogen into smaller components such as dextrins and maltose. By breaking down complex carbohydrates it may also serve a minor protective role by limiting their adhesion to the teeth.

The majority of the components are hydrophilic but a few are hydrophobic, such as the enzyme lipase. This is secreted by the von Ebner's glands on the posterior aspect of the tongue and can enter globules of fat to assist in breaking down fatty acids.

The mucins of saliva are glycoproteins with many short oligosaccharide residues on each molecule. They exist in both high and low molecular weight forms. Low molecular weight sulpho-mucins assist in clearing bacteria from the oral cavity by binding to and aggregating micro-organisms but the level, in resting saliva, of these low molecular weight mucins tends to decrease with age.

The interplay between water and mucins has a dramatic effect on the viscosity of saliva, particularly for the secretions from the submandibular salivary glands. The mucins bind water by hydrophilic interactions, and this bound water is essential for maintaining hydration of the oral mucosa. Reduced water results in a relative increase in the concentration of mucins, making the saliva more viscous in consistency and sticky in nature.²

Mucins are essential for the lubricating functions of saliva. When flow rates are low, patients wearing mandibular dentures suffer trauma to

the underlying mucosa and, for those with full upper dentures, there will be a lack of retention due to loss of cohesion and possibly chronic fungal infections.

Salivary mucins also protect the mucosal tissue and limit the extent of damage to surface cells caused by normal masticatory function. An even coating of mucins also gives a smoother surface for the flow of air during speech.

Salivary antibacterial systems

Saliva contains a broad range of antibacterial agents. Immunoglobulin A (IgA) is a major component of saliva proteins, and is able to aggregate bacteria and prevent adhesion. IgG and other immunoglobulins derived from the gingival crevice are also present in saliva, however little complement fixation is possible in saliva as levels of key complement components are too low. The contribution of gingival crevicular fluid to resting salivary flow is very small, in the order of 10-100 μL /hour. Antibacterial systems other than IgA are as follows:

- amylase can inhibit the growth of some species of bacteria
- lysozyme breaks down the peptidoglycan in the cell wall of some Gram positive bacteria, including *Streptococcus mutans*
- lactoperoxidase catalyses the oxidation of salivary thiocyanate by hydrogen peroxide to the toxic molecule hypothiocyanite which inactivates bacterial enzymes
- histatins (histidine-rich proteins) inhibit growth of *Candida albicans* and *S. mutans*
- lactoferrin binds ferric ions and inhibits bacteria from accessing iron, but it can be degraded by some bacterial proteases
- apolactoferrin has antimicrobial effects on a range of micro-organisms, including *S. mutans*

Salivary buffer systems

Healthy resting saliva maintains a pH in a narrow range between 6.7 and 7.4 and contains a very effective bicarbonate (HCO_3^-) buffer system. In the peripheral blood stream the combination of sodium bicarbonate, carbonic acid, and gaseous carbon dioxide removes protons (hydrogen ions)

from the system. Saliva works in a similar way. It is 5% dissolved carbon dioxide, compared with 1% in normal room air, and it is present both as bicarbonate ($\text{H}_2\text{O} + \text{CO}_2 \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{HCO}_3^- + \text{H}^+$) as well as dissolved CO_2 gas.

The concentration of bicarbonate ion in resting saliva is approximately 1 mmol/L, and this increases to over 50 mmol/L on stimulation. As the concentration of bicarbonate ion increases, so does the pH and the buffering capacity³ and this is a key point in interpreting diagnostic tests. Because of diurnal variations in resting flow rate, there are corresponding variations in levels of bicarbonate and thus in the pH and buffering capacity. The resting pH will be lowest during sleep and immediately upon waking, and then varies throughout the waking hours.

Any increase in salivary pH and buffering capacity will facilitate remineralisation and will also have some effect on the oral flora. Specifically, it will suppress propagation of aciduric micro-organisms, particularly cariogenic *S. mutans* and *Candida albicans*.

There are a number of ways of stimulating salivary flow such as mastication of foods and chewing gum. If they gain contact with the tongue, strong acids like phosphoric acid or acidulated fluoride products are powerful gustatory stimuli. Salt, sweet and bitter are less effective. On the one hand, salivary stimulation has an obvious protective function in a normal patient, but it has little if any therapeutic value in the problem patient. In fact, in patients with salivary gland dysfunction, frequent use of citric acid drops to enhance the flow is contraindicated because of the limited buffer capacity of their saliva.

The presence of phosphate, particularly in the resting saliva, also contributes to the buffering capacity. A range of proteins, as well as peptides such as sialin, play a minor role. They assist by promoting the production of amines, which exert an alkalinising effect from enzymatic breakdown of salivary proteins and oral bacteria. Similarly, urea in the saliva can be broken down to ammonia.

Salivary proteins involved in remineralisation

Saliva contains a range of inorganic ions including calcium, phosphates, fluoride, magnesium, sodium, potassium, and chloride. There are other components that help to maintain supersaturation of saliva with calcium and phosphate ions including -

Statherin: This inhibits precipitation and crystal growth of calcium phosphate. It is a phosphoprotein with a strong affinity for calcium and for enamel and other apatite surfaces. Many of the key biochemical properties of human salivary statherin are possessed by phosphopeptides derived from casein (CPP) (**Chapter 8**). Statherin and CPP share partial sequence homology with phosphoproteins from mineralised tissues such as dentine and bone.

Proline-rich proteins: These work in a similar fashion to statherin and bind to the surface of calcium phosphate crystals to prevent their growth. Together with citrate, they bind a considerable portion of the total calcium in the saliva, and help to maintain the correct calcium-phosphate ionic ratio. They are also a key component of pellicle and bind strongly through their amino terminus to enamel. The trailing carboxyl terminus is the site of adhesion for some bacteria in the early stages of plaque formation, and is also the binding site for tannins from the diet.

Saliva and remineralisation

Saliva controls the equilibrium of mineral gain and loss in an erosive or cariogenic oral environment. Its importance is demonstrated graphically in patients with salivary dysfunction and in desalivated animals.

Protective properties of saliva that increase on stimulation of salivary flow include oral clearance, buffering ability and degree of saturation of tooth mineral. This suggests that stimulation of flow after the consumption of fermentable carbohydrates may reduce the fall in plaque pH and thereby increase the potential for remineralisation. Studies by Edgar *et al.*⁴ showed that chewing gum immediately after a carbohydrate intake will neutralise plaque acid production and remineralise incipient lesions.

Saliva affords both static and dynamic protective effects such as buffering and sugar clearance. The former is probably the more important because of its direct link with enhanced remineralisation.

Fluoride ions accumulate in dental plaque and the levels in resting saliva correlate well with healing and remineralisation of early caries lesions.^{4,5} Low to moderate levels of fluoride, up to 40 ppm, inhibit glycolytic fermentation of sugars by plaque bacteria.

Remineralisation also depends upon the presence of fluoride which should be delivered to the early lesion in moderate concentrations to achieve the greatest effect. Topical applications of very high concentrations encourage the formation of an extremely dense surface layer, effectively reducing the permeability and hindering further repair. On the other hand, topical application of fluoride-containing phosphopeptides will bolster the capacity of the intra-oral fluoride reservoir to supply ions for a prolonged period (**Chapter 8**). This is crucial to the success of topical treatments in preventing and arresting both caries and erosion⁵ (**Figure 7.2**).

Clearance of substrates and acids

The term 'oral clearance' refers to the elapsed time between the introduction of a substance to the oral cavity and the moment when its presence there can no longer be detected. It can be expressed as either

- half life = 50% of the original concentration,
- detection threshold = time when a substance can no longer be detected.

The oral clearance rate can be tested as follows:

- apply glucose solution or glucose sweet e.g. a jelly bean
- place a test tape containing glucose oxidase, peroxidase and a chromogenic substance, selectively into the mouth
- leave in place for some time, observing for colour change
- check the time taken for glucose to no longer be detected

Oral clearance is affected by the stimulated flow rate and is site-dependent to the extent that the

most rapid clearance occurs adjacent to the ducts from the major salivary glands. This means that the test should be conducted at several sites such as the anterior floor of the mouth and the maxillary vestibule labial of tooth #11. A range of measurement times will reveal that clearance lingual to tooth #31 occurs within 30 seconds in most individuals. In contrast, the labial maxillary site near the midline typically requires 20 minutes to clear glucose completely.

All of this suggests that strategies for salivary stimulation should be considered as part of an overall preventive regimen for a patient with active caries. These may include foods and eating patterns which lead to saliva stimulation, as well

as the use of sugar-free chewing gum. Geddes⁶ has shown that if a meal includes a carbohydrate, such as sucrose, glucose, or fructose that is fermented rapidly in plaque, there will be a rapid fall in plaque pH. Moreover, frequent repetition of carbohydrate intake will enhance the demineralising potential.

Other food items can have a positive influence on the plaque pH. For example, when milk or cheese products are consumed at the end of a meal, the proteins they contain can buffer the pH and can also exert a topical effect through phosphopeptides. Ending an evening meal with a low fat cheese platter, or using low fat cheese sticks as a between meal snack, are examples of how a



Fig. 7.2a. Common patterns in saliva testing results: This 35-year-old female patient shows caries on buccal surfaces of canines and premolars resulting from salivary dysfunction.

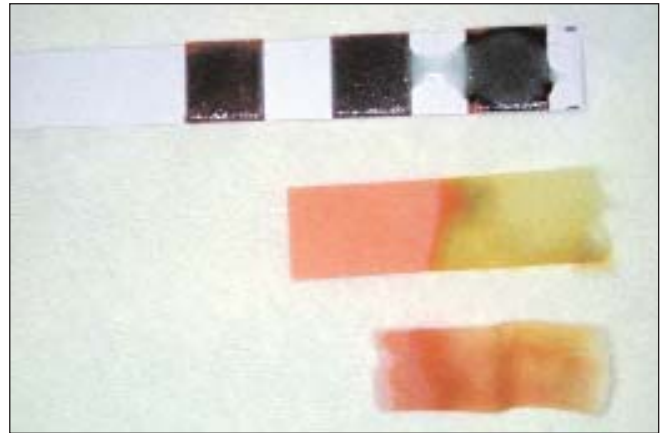


Fig. 7.2b. Test shows depressed pH with resting saliva (bottom strip), but normal pH with stimulated saliva.



Fig. 7.2c. Female patient (28 years) has cervical hypersensitivity on buccal of maxillary teeth following long periods in a swimming pool leading to subclinical dehydration.

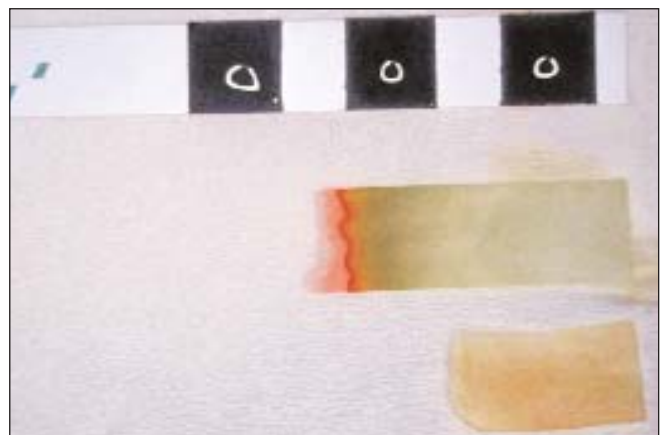


Fig. 7.2d. After modifications to lifestyle, tests show resting saliva at pH6.8 (bottom strip) which is above the critical pH value for enamel.

dietary pattern can be altered to increase these natural caries preventive actions.

Control of salivary secretion

The time of day has a substantial influence on the resting flow rate of saliva. It decreases during sleep and increases during the waking hours reaching the maximum flow during midafternoon. This pattern has relevance when assessing the resting flow rate in the clinical setting.

With a typical resting flow rate of 0.03 mL/minute, the total amount of saliva secreted during eight hours of sleep will be only 15 mL. There will be about two hours of stimulated flow during eating and then an additional 14 waking hours of resting flow leading to a total daily production of 700-1000 mL.

A complex of mechanisms work in partnership to control salivary flow. Autonomic parasympathetic and sympathetic nerves are the primary influence while taste and tactile stimuli from the tongue and oral mucosa also play a part. Stimulation of parasympathetic nerves causes the release of water and ions, but not proteins, whereas sympathetic stimulation causes the release of proteins packaged within acinar cells. Proprioceptive stimuli from masticatory muscles and the periodontal ligament will excite the inferior and superior salivary nuclei within the brain and these are also influenced by the cerebral cortex (**Figure 7.3**).

These neurological influences underpin the significance of the psychological status of the patient on resting salivary flow rates.

Clinical assessment of salivary parameters

Resting saliva flow can be examined by visual means, including

- visual assessment of lower lip labial gland secretion
- visual assessment of resting salivary volume in the oral cavity (pooling)
- visual scoring of salivary viscosity (watery, bubbly, or frothy)
- measurement of resting salivary pH using pH paper or a pH meter

Visual assessment of resting saliva is a simple and rapid method for screening patients for sali-

vary dysfunction (**Chapter 6**). In the presence of xerostomia the saliva will pool and froth in the sublingual regions and white mucinous strands of saliva residues will appear on the oral mucosa.¹

Assessment of stimulated saliva flow involves

- measurement of the volume collected over a defined period
- measurement of pH using pH paper or a pH meter
- estimation of buffer capacity using challenge strips with weak acids

NOTE



Saliva flow rates in healthy patients

- Resting flow rate for pooled saliva = 0.3-0.4 mL/minute
- Less than 0.1 mL/minute = xerostomia
- Stimulated flow rates = 1-2 mL/minute
- Less than 0.7 mL/minute = pathology

Systematic assessment of salivary parameters

1. Listen to symptoms

- Oral dryness during the waking hours
- Oral dryness on waking
- Lack of lubrication during eating, talking or swallowing
- Salivary web formation during swallowing
- Altered taste perception
- Impaired retention of full upper dentures
- Impaired lubrication of lower dentures
- Mucosal irritation from foods and dental home care products
- Other potentially related complaints such as halitosis

2. Listen to the history

- Duration and severity of symptoms
- Known exacerbating and relieving factors
- Medical conditions associated with salivary dysfunction
- Other medical conditions
- Prescribed medications
- 'Over-the-counter' medications
- Past medical treatments
- Past dental treatment
- Use of home care products

3. Listen to the patient's lifestyle

- Patterns of fluid intake
- Dietary patterns for fermentable carbohydrates
- Preferred snacking patterns
- Intake of caffeine
- Intake of alcohol
- Intake of acidic foods and drinks
- Intake of nicotine
- Intake of illicit substances
- Patient's occupation

- Patient's recreational habits
- Major stressful events in the patient's life

4. Look for signs

Soft tissue changes

- Dryness of the vermillion border of the lip
- Dryness of the oral mucosa
- Loss of filiform papillae of the tongue
- Cratering and fissuring of the tongue
- Increased plaque formation on the tongue
- Related mucosal pathology such as oral candidal infections



Fig. 7.3a. Subclinical dehydration: Male (25 years) shows occlusal tooth wear and cervical hypersensitivity on buccal of premolars in both arches.



Fig. 7.3b. Loss of occlusal tooth structure in the left mandibular quadrant.



Fig. 7.3c. Loss of occlusal tooth structure in the right mandibular quadrant.



Fig. 7.3d. Resting salivary pH of 5.6 (upper strip), but pH 7.4 for stimulated saliva (middle strip). Buffer capacity for stimulated saliva (bottom strip) satisfactory suggesting subclinical dehydration. Added problem was high intake of black cola drink.

- Absence of saliva in response to gland palpation
- Enlargement of major salivary glands

Hard tissue changes

- Increased caries rate (particularly cervical caries)
- Increased rate of noncarious loss of tooth structure by dental erosion
- Multiple teeth with cervical dentinal hypersensitivity from dental erosion
- Failure to form supragingival calculus from plaque in the lower incisor region
- Increased plaque accumulation on the teeth and appliances

Aetiological factors in depressed salivary parameters

1. Dehydration

- Inadequate fluid intake
- Strenuous physical activity
- Swimming
- Outdoor occupations
- Dehydrating work environment
- Driving/travelling long distances
- Caffeine including black cola softdrinks, energy drinks, coffee, tea, etc.
- Alcohol
- Polyuria in uncontrolled diabetes mellitus

2. Salivary gland pathology

- Head and neck or total body irradiation
- Lymphocytic sialadenitis in HIV, hepatitis C, and diabetes mellitus
- Primary Sjögren's syndrome
- Secondary Sjögren's syndrome associated with connective tissue diseases including rheumatoid arthritis, sarcoidosis, systemic lupus erythematosus, scleroderma, dermatomyositis, and polymyositis
- Graft v host disease in bone marrow transplant recipients

3. Medical conditions

- Psychological stress
- Depressive illnesses
- Chronic renal failure
- Menopausal hormone imbalance
- Thalassaemia major
- Chronic protein energy malnutrition

4. Side effect of recreational drugs

- Nicotine

- Alcohol (dehydration, liver cirrhosis)
- Cannabis
- Opiates (heroin, methadone, narcotics, etc.)
- Amphetamines

5. Medications

- Anticonvulsants
- Antiemetics and antinauseants
- Anti-Parkinsonian agents
- Antipsychotics
- Antidepressants (TCA, SSRI, Monoamine oxidase inhibitors)
- Antipruritics
- Antihistamines
- Antihypertensives
- Antispasmodics
- Antineoplastic agents
- Anxiolytics, tranquillisers, and sedatives
- Cardiac antiarrhythmics
- Expectorants and decongestants
- Diuretics
- Narcotic analgesics
- Systemic bronchodilators
- Skeletal muscle relaxants

Impact of medications

Many major medical disorders are associated with xerostomia, and these must be recognised by health professionals. However, other common factors in the aetiology are often overlooked, particularly the effect of medications.

Several hundred medications will induce salivary dysfunction, and this important side effect is not always listed as an adverse effect in prescribing guides. Over-the-counter medications such as expectorants and decongestants are particularly important. Polypharmacy is common amongst elderly and medically compromised patients so all in this category should be tested for salivary dysfunction, even in the absence of overt caries or other pathology. Salivary dysfunction can also be a side effect because of delayed metabolism and clearance of drugs by the liver and/or kidneys.

Because of the circadian rhythm, which allows the flow to be highest in midafternoon, it is wise to take repeated measurements of resting saliva before making a diagnosis of xerostomia. A false negative diagnosis is most likely to occur when

patients are seen only in mid afternoon. It should be remembered that salivary dysfunction is not the only oral side-effect of medications – others include taste dysfunction, gingival overgrowth, mucosal ulcerations or lichenoid lesions, mucosal pigmentation, tooth discolouration, involuntary facial movements, facial pain and dysaesthesia.

Psychological stress

A patient's psychological state can influence their resting salivary output considerably.⁷ Individuals with a high level of stress in their workplace or home environment can show prolonged reductions in resting salivary flow rate because of the alterations in sympathetic and parasympathetic output that occur with emotional and psychological stress.

Hormonal variations

Hormones can stimulate water saving mechanisms thus influencing salivary flow and composition through direct action on acinar or ductal elements within the salivary glands. Anti-diuretic hormone, which increases water reabsorption in the distal tubule in the kidney, has the same action on salivary gland ducts. As a consequence, a reduced volume and increased viscosity of saliva in the anterior floor of the mouth contribute to a sensation of thirst. However, thirst itself is an imperfect indicator of body fluid balance.

There are several known effects of hormonal changes on salivary parameters in female patients. Oral contraceptive use causes a higher buffer capacity. Menstruation, ovulation, and premenstrual syndrome do not directly affect salivary flow, pH or buffer capacity, however, the overriding effect of psychological stress should be borne in mind. In pregnancy, there is reduced buffer capacity. This effect declines, and then reverses in very late pregnancy, just before delivery, when salivary buffer capacity rises. Finally, menopause can cause lowered resting flow rates, but this effect is not seen in all women.⁸⁻¹⁰ Similarly, the male sex hormone testosterone is also known to increase resting salivary flow.

Gum chewing and salivary function

Because regular gum chewing leads to a pro-

longed increase in both the stimulated and unstimulated salivary flow rate, chewing sugar-free gum is an important preventive oral health behaviour. It augments the protective properties of saliva such as oral clearance, buffering, pH, and supersaturation with minerals. Chewing sugar-free gum elevates plaque pH and this can be enhanced by including phosphopeptides in the gum (**Chapter 8**). This has been demonstrated by Reynolds et. al., who assessed demineralised slices of enamel mounted in intra-oral appliances.¹¹⁻¹⁴ A similar elevation in pH can be achieved by using alkalinising agents such as urea within the gum formulation.¹⁴ Long-term chewing of xylitol or xylitol-sorbitol gums has been shown to lead to a reduction in caries risk over five years.^{15,16}

During gum chewing, the flow rate peaks during the first minute and can be maintained by continual chewing. It will not increase dramatically by chewing faster, with flow rate being maintained over a range of chewing frequencies between 35 and 130 chewing actions per minute. It is important to stress that gums containing sucrose must be avoided. They are less stimulatory to salivary flow than sugar-free gums, do not promote mineralisation, but are directly cariogenic through sustained release of sucrose.³

Lifestyle Factors and Dental Caries

Dental caries is an infectious bacterial disease which can be modified by dietary carbohydrate and saliva. The caries process represents the interaction of many factors over time:

- Salivary factors – flow, pH, F and bicarbonate (HCO_3) levels. Reduced salivary flow rates result in reduced salivary pH and buffer capacity.
- Tooth factors – location, fluoride levels, hypoplasia, etc.
- Plaque factors – amount, composition of plaque, the levels of *S. mutans* and other aciduric microorganisms.

- Dietary factors – form, frequency, exogenous acid content particularly a high intake of acidic foods and drinks which shifts the flora to aciduric organisms.
- Oral hygiene – frequency and efficacy, use of fluoride-containing dentifrice which affects the loading of the intraoral fluoride reservoir.

This section examines strategies for caries prevention, with an emphasis on behavioural and lifestyle issues. The co-existence of several factors such as bacterial plaque, fermentable carbohydrates, a susceptible tooth surface and time, is necessary for caries to occur. It is sensible, therefore, to base caries preventive strategies on several independent elements, rather than one element alone. For example, lifestyle factors which may suppress salivary flow include smoking and caffeine and a low water intake. On the other hand, actions that elevate salivary pH and buffer capacity include regular use of sugar-free chewing gums, and sodium bicarbonate mouthrinses.

Bacterial species that are both acidogenic and aciduric flourish in low pH environments. Refined carbohydrates and acidic foodstuffs therefore encourage growth of bacteria such as *Streptococcus sobrinus* and *Streptococcus mutans*. As saliva is the milieu in which the disease progresses it is not surprising that salivary parameters such as flow rate, ionic composition, buffer capacity, and fluoride concentration exert a major influence on the equilibrium between de- and re-mineralisation.

Salivary assessment can provide valuable clues to assist the clinician in determining causal factors in the patient who presents with dental caries, dental erosion, or cervical hypersensitivity. Repeated saliva testing is a useful part of monitoring patient progress, and the level of compliance regarding changes to lifestyle (Chapter 6).

Reduced salivary protection

Because low resting saliva flow rates encourage growth of aciduric organisms such as *S. mutans*

and *lactobacilli*, it is essential that salivary dysfunction be recognised. As outlined in **Aetiological factors in depressed salivary parameters**, it can be induced by medications, irradiation of salivary glands, and salivary gland diseases such as Sjögren's syndrome¹⁷ (Figure 7.4).

Exposure to fluoride

Frequent exposure to fluoride achieves optimal low level loading of the salivary fluoride reservoir sufficient to prevent the initiation and progression of caries. At low concentrations, fluoride inhibits demineralisation and enhances remineralisation through precipitation of calcium phosphates, and the formation of fluorapatite (Chapter 4).

When present in high concentrations, fluoride can inhibit bacterial enzymes and reduce the utilization of fermentable substrates by *S. mutans*. At levels of 0.5 mmol fluoride/L within plaque, the cell cytoplasm of *S. mutans* is acidified, and glycolysis is inhibited once the intracellular pH reaches pH 5.2. This accounts for 75% of the caries inhibition effect of high concentrations of fluoride, with the remaining 25% due to the combined effect of fluoride on enamel solubility. However, because this does not reduce the number of viable bacteria, some form of antibacterial agent is essential for caries control in the high risk patient.

There is evidence that glass-ionomer can suppress cariogenic micro-organisms through fluoride release (Chapter 11). Clinical studies have examined bacterial counts in carious dentine under resin modified glass-ionomer and amalgam restorations over a six month or two year period. Patients with occlusal dentine caries in molar teeth underwent a minimal procedure in which the overlying enamel was removed to allow the underlying carious dentine to be sampled in the region just beneath the dentinoenamel junction. The teeth were then restored without further removal of carious dentine, and re-evaluated after six months or two years. At that time all caries was removed and a permanent restoration placed. While both materials showed a substantial decrease in numbers of *S. mutans* and *lactobacilli* over the course of the study, the greatest suppressive effect occurred with the glass-ionomer.

BE AWARE 

Salivary factors that influence de- and remineralisation

- flow rate
- ion composition
- buffer capacity
- fluoride content

The fluoride reservoir can be supported up to twice daily through the use of fluoridated toothpaste whereas fluoridated water will achieve a sustained loading. That is, fluoridation of water supplies exerts additional anticaries properties and explains why caries increases in a population which is using a fluoride dentifrice after community water fluoridation is removed.

Optimal loading can be achieved with a twice daily dentifrice combined with professional products, such as neutral sodium fluoride gels, applied on retiring when the saliva flow is lowest. Bioadhesive fluoride releasing tablets are also available.

Fermentable substrates in the diet

Lifestyles with a high frequency of intake of fermentable substrates will increase caries rates so assessment must include questioning regarding patterns of snacking. Snacks include carbonated drinks or energy drinks, particularly those containing caffeine (**Figure 7.5**), as well as tea and coffee.

Sucrose, glucose and other fermentable substrates in the diet can be replaced with non-fermentable sweeteners such as xylitol, sorbitol or isomalt (a chemically modified carbohydrate), or an artificial sweetener with no chemical similarity to carbohydrates such as aspartame or saccharin.

Fig. 7.4. Sjögren's syndrome: Female (50yrs) with primary Sjögren's syndrome. Positive serological testing for anti-SS-A and anti-SS-B antibodies, and for antibodies to nuclear antigens including double stranded DNA.



Fig. 7.4a. Teeth show arrested root surface caries, abrasion of labial enamel and gingivitis despite low plaque levels.

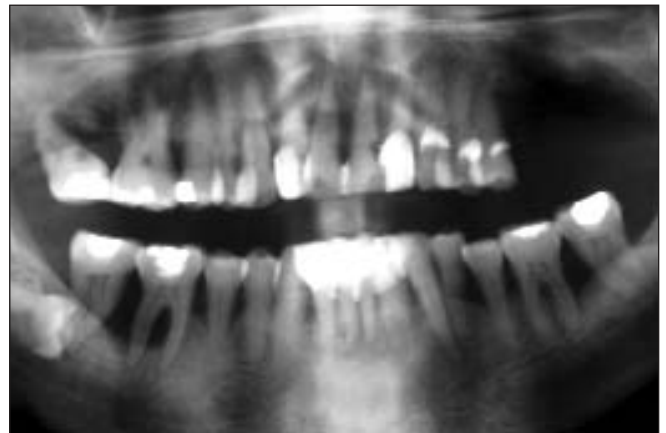


Fig. 7.4b. Initial OPG radiograph shows extensive restorations, posterior periodontal bone loss. Home maintenance included chlorhexidine gel, high fluoride (5000 ppm) dentifrice.

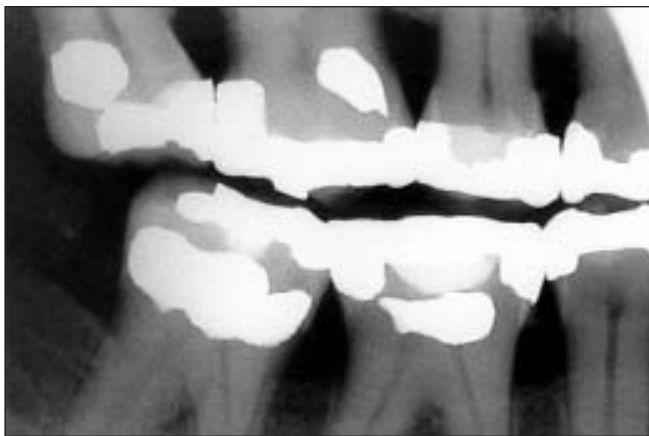
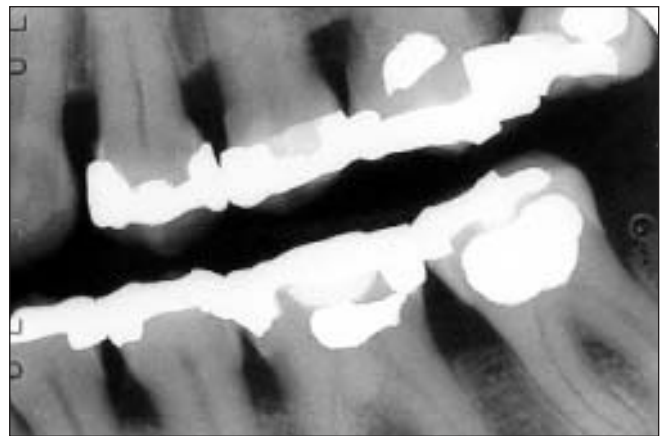


Fig. 7.4c & d. Radiographic situation 15 years later. Restorations remain intact, no further periodontal bone loss.

Xylitol is taken up by *S. mutans*, requiring the organisms to expend energy, but is not fermentable, so no acid fermentation byproducts will be released. This means it is not cariogenic, however, recently attention has been directed to the effects of prolonged consumption of xylitol. Chewing a xylitol gum regularly for extended periods can suppress salivary levels of *S. mutans*, and appears to select for *S. mutans* with impaired adhesion properties, that is, these bacteria bind poorly to teeth and shed easily from plaque to saliva. This means that frequent chewing of xylitol gum by pregnant women may be associated with reduced mother to child transmission of these bacteria. This conclusion is supported by clinical trials.

Soft drinks are often a major contributor to total sucrose intake because they contain typically 12–13% sucrose by weight. This means a 375 mL can of soft drink contains 50 grams of sucrose, and a 600 mL bottle about 80 grams. A high daily total intake of sucrose can lead to insulin resistance and Type 2 diabetes and this is a leading cause of kidney failure and blindness, and a major risk factor for death from cardiovascular disease and stroke. It can also lead to obesity and it must be noted that there is a recorded epidemic of this in Western countries.

Items to include in a diet history

- Plain milk
- Flavoured milk



Fig. 7.5a & b. Caries from black cola soft drink: Male (25 years) shows caries and erosion. Drinks 1.5 litre/day as well as high caffeine energy drinks.



Fig. 7.5c & d. Male (50 years), extensive caries, Type 2 diabetes mellitus and obese. Drinks 4 litres/day of black cola drinks. Lower incisors partially protected by resting saliva from the floor of the mouth.

- Cheese
- Icecream
- Yoghurt
- Chocolate
- High energy foods such as Sustagen
- Bakery items such as cakes, muffins, sweet biscuits, pastry
- Confectionery such as lollies, mints, jellies
- Cough lozenges
- Cordial
- Vitamin C tablets
- Fruit bars
- Dried fruits
- Bread
- Custard

- Black cola soft drink
- Energy drinks
- Other soft drinks

Transmission of *S. mutans*

Dental caries is a true infectious disease which fulfils Koch's postulates. The transmission of *S. mutans* from mother to child via salivary transfer has been demonstrated in numerous studies. Behaviour patterns include kissing, sampling of food, and sharing spoons or feeding utensils. The child's own saliva provides a level of protection from early childhood caries but this is limited to the mandibular incisor teeth (**Figure 7.6**).

The higher the salivary levels of *S. mutans* in



Fig. 7.5e & f. Female (18 years) lost three maxillary teeth from caries. Drinks 3 litres/day of black cola drinks. Home prevention included chlorhexidine before restorative treatment was undertaken



Fig. 7.5g. Male (14 years) swished mouthfuls of black cola drink for 20 minutes before swallowing. Drinks 1 litre/day but extended contact time dissolves labial enamel.



Fig. 7.5h. Opiate addict (32 years) high frequency black cola soft drink because of craving for sweetness. Note extensive enamel caries.

the mother, the greater the number of organisms transmitted. Analysis of the strains present in families has shown that strains are highly conserved within mother to child pairs and also between siblings. The presence of either or both *S. mutans* and *S. sobrinus* in approximal plaque is strongly correlated with early carious lesions in children. *S. mutans* is normally acquired much earlier in life than *S. sobrinus*, however some adults and elderly patients harbour *S. mutans* alone and not *S. sobrinus*. It is rare to find a patient with coronal or root surface caries who does not harbour *S. mutans* in high numbers.

For many years, *S. mutans* were thought to lack a suitable ecological niche in the predate oral

cavity so that predentate inoculation was unlikely to lead to colonization. However, inoculation with *S. mutans* can occur early in life through repeated exposure from the mother and siblings.¹⁸⁻²¹ In 2001, the work of Wan and others demonstrated conclusively that predentate acquisition could occur. Approximately one-third of 3-month-old infants examined in their studies showed positive cultures of *S. mutans*.²⁰ This raises the possibility of interrupting transmission by

BE AWARE



Streptococcus mutans

- can be transmitted from mother to child
- can colonise the predentate mouth
- will stimulate early childhood caries



Fig. 7.6a. Caries in the deciduous dentition: Caries at cervical of the maxillary incisors and proximals on lower first molar. Early lesions at cervical of posteriors, mandibular incisors not affected.



Fig. 7.6b. In another patient all the maxillary teeth are completely destroyed by caries and will require removal.



Fig. 7.6c & d. Compare caries in maxillary teeth to the state of mandibular teeth in the same patient.

selective decontamination of both mother and infant using intermittent home application of chlorhexidine. Delayed acquisition of *S. mutans* is likely to reduce the caries experience in both the primary and permanent dentitions at a later age.

The preferred site for colonisation of *S. mutans* are non-shedding, hard surfaces,^{22,23} so the early introduction of a hard surface into the mouth will provide an alternative site to the teeth for colonization. This may occur with the cleft palate patient who is fitted, shortly after birth, with a maxillary acrylic appliance to obturate the cleft. These are typically worn until 18 months of age when the cleft is surgically repaired.

Early colonisation and high levels of *S. mutans* in infants and young children is promoted by a range of factors and is associated with high caries experience.

- A high carbohydrate intake
- Sweetened pacifiers
- Fruit juices, cordials, soft drinks are a source of carbohydrate, are aciduric and have erosive effects through low pH.
- A grazing pattern of eating
- Poor pre-natal nutrition and peri-natal illnesses may cause enamel hypoplasia and predispose to early childhood caries.

DNA fingerprinting has shown that genotypes of *S. mutans* are relatively stable within the one individual, and persist for several years. There is a high degree of consistency in children between three and eight years of age as well as in adults. However, some individuals can also gain and/or lose genotypes. There is a particularly high degree of consistency in mother-child pairs, which reflects the usual vertical mode of transmission of *S. mutans*.

Mother-child behaviours

S. mutans colonization occurs more frequently in pre-term children due to their relative immaturity. In a recent study of 172 preterm, six-month-old infants²¹, 50% of preterm and 60% of full term children harboured *S. mutans*. In both groups,

increased frequency of sucrose was the most important factor, followed by breast feeding and saliva transfer from mother to infant. Mothers with infected infants who had *S. mutans* levels greater than 5×10^5 CFU/mL in saliva, came from a lower socio-economic status, and snacked more frequently.

Greater percentages of infected infants were given demand breastfeeding, and slept next to their mothers. In full term infants a higher percentage of infected children received more than one feeding during the night. In both groups infected infants were reported to have sucked adults' fingers, to have shared foods with other individuals, and to have drunk from a training cup and had their food pre-tasted.

Frequent acid intake

A high frequency of exposure to dietary acids in the form of soft drinks, sports drinks, and fruit juice can shift the supragingival oral flora toward aciduric microorganisms. As the intra-oral pH falls, the numbers and proportions of *S. mutans* and Lactobacilli increase, and the proportions of acid-sensitive species such as *Fusobacterium nucleatum*, *Streptococcus gordonii* and *Streptococcus oralis* will fall. Thus, the fall in pH not only enhances the competitiveness of cariogenic organisms, but inhibits the growth and metabolism of species not associated with caries. Chemo-stat studies of plaque bacteria have shown that certain species other than *S. mutans* or *Lactobacilli* are competitive at pH values low enough to demineralise enamel, e.g. *Veillonella dispar*. They may be found within the plaque involved in the initiation of dental caries, but are likely to be secondary colonisers rather than primary pathogens.

The challenge with this strategy, if used in isolation, is that mature plaque is a highly ordered structure with numerous metabolic interactions between bacteria. The microbial community has already achieved a high level of homeostasis which will make it somewhat resistant to deliberate external manipulation.

NOTE

The frequency of low pH in the saliva can be reduced by

- restricting the intake of fermentable substrates or acidic foods
- inhibiting acid production, e.g. using high concentrations of fluoride
- using nonfermentable sugar substitutes, e.g. xylitol
- using alkalinising mouthrinses and reducing acidic drinks in the diet
- promoting alkali generation, e.g. from arginine or urea supplements

Endogenous acids

Acidic conditions can be created by eating disorders or gastrointestinal conditions, such as chronic gastric reflux, in which the source of the acid will be hydrochloric acid from the stomach. Stronger acids can degrade pellicle by proteolysis, and thus predispose the tooth surface to mineral loss from erosion (**Figure 7.7**). However, these conditions predispose to caries as well because of the likelihood of greatly increased counts of *S. mutans* through their preference for highly acid conditions. A preventive program for these patients must address the microbial risk factors for caries as well as the erosion potential.



Fig. 7.7a. Bulimia: Female (19 years) with bulimia shows typical loss on palatal of upper incisors. The inset shows saliva at rest pH 6.2, and stimulated pH 7.4.



Fig. 7.7b. Female (55 years) with chronic gastric reflux shows erosive loss of palatal tooth structure.

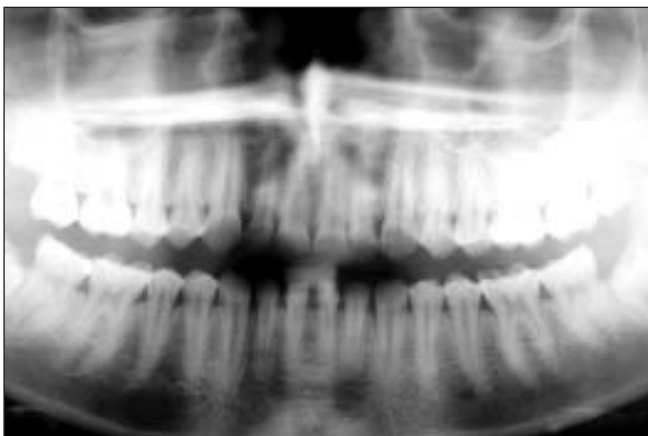


Fig. 7.7c. OPG radiograph of bulimic patient in **a**) shows erosion of anteriors, and flat occlusal on posteriors.



Fig. 7.7d. Female (six months pregnant), nausea and vomiting since week 12 shows erosion on lingual of upper anteriors.

Eating disorders

Eating disorders such as anorexia nervosa, bulimia, and binge eating are most prevalent in adolescent girls and young adult women. They can have dramatic effects on oral health, particularly non-carious loss of tooth structure through erosion. Typically there will be adverse physiological changes beyond the oral impact.

They are psychiatric disorders characterised by abnormal eating patterns which result in both dramatic weight loss and serious general health consequences. Bulimia is more common in late teenage and early adult years. There will be refusal to maintain body weight over a minimal normal weight for age and height, an intense fear of gaining weight, and a distorted body image. There are two subtypes –

- those who drastically limit their food intake and avoid eating
- those who purge after eating

Gastric purging can cause dramatic erosion from the corrosive effects of gastric hydrochloric acid. Proteolysis of pellicle removes this important protective layer of salivary glycoproteins, exposing the tooth surface directly to the gastric contents. Patients typically will clean their teeth after purging, resulting in abrasion of the softened enamel. Purging can also induce oesophageal inflammation, protein malnutrition, dehydration, and electrolyte imbalances. Bruises, caluses, scarring, fungal infections of the nail beds and abrasions of the fingers may be present from repeated episodes of self-induced vomiting.

Patients presenting with sensitive exposed dentine and palatal erosion of the maxillary anterior teeth are likely candidates for a disorder involving regurgitation of gastric contents. However, consideration should be given first to the more common condition of gastric reflux, which can be attributed to the side effects of several medications, as well as to pregnancy and hiatus hernia. Caffeine will increase gastric acid production and may increase the severity of erosive and abrasive tooth wear on palatal surfaces of maxillary incisor teeth.

Hydration and caffeine

Negative fluid balance and systemic dehydration decrease resting saliva flow and pH and can lead

to erosion, with both accelerated tooth wear²⁵ and increased caries risk. As the body moves into negative fluid balance the resting saliva volume will decrease and viscosity will increase in the anterior floor of mouth contributing to a sensation of thirst. However, thirst itself is an imperfect indicator of body fluid balance. Severe dehydration affects both resting and stimulated flow rates, whereas in mild dehydration, only the resting flow is abnormally low.²⁶

Water constitutes approximately 70% of the mass of body cells and approximately 60% of the total mass of the human body. Regulation of thirst through the hypothalamus is a relatively poorly developed reflex and lags behind the body's need for water. There is a level of obligatory daily water excretion related to the disposal of waste through the kidneys, faeces, lungs and skin. Typical daily water loss from the kidneys is in the order of 500 mL to 1.5 litres; from insensible perspiration through the skin 450-900 mL; from the lungs 350 mL; and from faeces 150 mL. To compensate for this normal total water intake should be 2.3 litres. This is obtained generally as follows:

- from the metabolic production of water, approximately 200-300 mL
- from foods 700-1000 mL
- from liquids 550-1550 mL

These figures explain the rationale behind the advice to drink five to six glasses of water per day. During hot weather water loss increases in two ways: sensible perspiration, where the skin and clothing is visibly wet (1400 mL) and insensible perspiration, together with loss through the lungs (700 mL). Thus, total fluid intake in hot weather, assuming no significant physical activity should be 3.3 litres – an increase of one litre. During prolonged exercise large amounts of fluid can be lost through sensible perspiration. Amounts up to 5 litres are not uncommon, thereby increasing the daily water requirements to well over 6.5 litres per day.

A reduction of 2% in body weight will generally trigger the thirst reflex. Once body weight loss from water loss is in the order of 3-4%, complete cessation of resting salivary output will occur because of the release of antidiuretic hormone. At the same time there will be a dramatic reduction

in urinary output as the kidneys attempt to save water.

Patients with a high intake of alcohol and caffeine should be aware of the need to take in additional water because of their diuretic effects. When assessing a patient's intake of caffeine, it is recommended that coffee, tea, cola type soft drinks and energy drinks be carefully noted as well, since these are the major sources. The net effect of a high intake will be loss of fluid and this can precipitate adverse changes in the salivary environment, particularly the resting saliva.

A typical mean daily caffeine intake is 4 mg/kg, which equates to approximately 280-300 mg/day in an adult with normal body weight of 70-75 kg. Demonstrable physiological and psychological effects from caffeine may occur at doses of 60-70 mg in most individuals. Because it is addictive, acute cessation of caffeine intake is associated with a characteristic withdrawal syndrome²⁷ so reductions should be achieved gradually.

BE AWARE



Caffeine

- is highly addictive
- found in many beverages
- mean daily intake = 4mg/kg
normal body weight = 70-75 kg;
dose = 300 mg/day
- effects felt at 70mg in most people
- acute withdrawal may be a problem

Caffeine is the most popular, widely consumed, legal, and socially acceptable psychoactive drug in the world. Hollingworth's 1911 investigation was amongst the first to systematically study behavioural effects of caffeine. His research was necessitated by a United States federal government suit against the Coca-Cola Company for marketing a beverage with an addictive ingredient, namely, caffeine.²⁸

Caffeine is a psychostimulant and at dose levels of 50-200 mg will raise mental alertness, induce a faster and clearer flow of thought, wakefulness, and restlessness. Higher doses will stimulate the vasomotor and respiratory centres. It also has a slight stimulant action on the heart, increasing

cardiac contractility, and output and dilating the coronary arteries. It constricts cerebral blood vessels but causes bronchial relaxation, increases urine output and increases secretion of gastric acid. This latter effect explains why a high caffeine intake exacerbates gastroesophageal reflux. Excessive intake of caffeine causes insomnia, anxiety, tachycardia, hypertension, cardiac arrhythmias and gastrointestinal disturbances. Chronic use is associated with habituation and tolerance, and, as noted above, discontinuation may produce a withdrawal syndrome.²⁹ There are well known psychological and physiological effects from modification to caffeine intake, and this needs to be borne in mind when advising patients.^{27,30}

Very high levels of caffeine have deleterious effects on the performance of the heart and the cardiovascular system. Intakes above 680 mg per day, equivalent to five cups of drip filter coffee, can be associated with an elevated risk of cardiac arrest in patients who are non-smokers.³¹

The medical literature has drawn attention to linkages between high caffeine beverages and osteoporosis. As well, consumption of soft drinks containing phosphoric acid has been shown to be an independent risk factor for hypocalcaemia in post-menopausal women. Consumption of one bottle per day of cola soft drinks has been associated with depressed blood calcium levels³² in elderly women, while black cola beverages have been

TABLE 7.1: Caffeine content of some common foodstuffs

4 mg	cup of decaffeinated tea or coffee
4 mg	hot chocolate drink
10 mg	small bar of chocolate
35 mg	375 mL can of black cola soft drink
35 mg	cup of regular tea
66 mg	200 mL can of energy drink
70 mg	cup of instant coffee
125 mg	cup of percolated coffee

Based on product labels, data from Barone and Roberts 1996 (31), and information from web sites from manufacturers of black cola soft drinks.

associated with an elevated risk for fractures in teenage children, particularly in females.^{33,34}

Use of chlorhexidine

Chlorhexidine (CHX) is regarded as the 'gold standard' as a mouth-rinse or topical treatment for suppressing *S. mutans* (Chapter 3). Unlike fluoride, chlorhexidine does not directly affect the ionic reactions of remineralisation, but is effective solely on the cariogenic microflora. This means that, because CHX is highly effective against *S. mutans*, it is particularly useful as part of caries prevention programme, as distinct from a caries control programme. It can cause a 1000-fold reduction in salivary levels with a single treatment. However, the levels will re-establish over time so intermittent use is required.^{24,1}

CHX can be delivered in a variety of ways including varnishes, mouthrinses, gels, dentifrices, and chewing gums. The gel form can be applied using a toothbrush or in a tray. Delivery using a chewing gum vehicle has the advantage of simplicity and the possibility of combining chlorhexidine with xylitol to reduce salivary levels of *S. mutans*, *lactobacilli* and yeasts.

Problems with **CHX mouthrinse** include

- taste alterations
- staining of the teeth, tongue, and restoration margins
- increased calculus formation
- mucosal irritation from alcohol based mouthrinses

Advantages of **CHX gel** products over rinses are

- no mucosal irritation
- reduced influence on taste perception
- reduced staining if used intermittently
- lower cost
- excellent patient compliance.

NOTE



CHX is particularly useful in caries prevention because

- it is highly effective
- it has an acceptable toxicological profile
- it is very poorly absorbed if ingested accidentally
- no significant resistance develops in the oral microflora
- allergy is uncommon

Synthetic or natural phenolic agents in oral health products

Triclosan is a synthetic phenolic agent which suppresses Gram-negative anaerobes, particularly periodontopathic organisms, to a greater extent than Gram-positive cariogenic bacteria. Both triclosan and essential oils are natural phenolic compounds and have a greater antimicrobial effect on *S. mutans* than on other streptococci. However, their effect on *S. mutans* is modest compared with chlorhexidine. Rinsing twice daily for 30 seconds with an essential oil mouthrinse (Listerine®) or using a Triclosan-containing co-polymer dentifrice (Colgate Total®) will give modest suppressive effects on *S. mutans* in supragingival plaque and saliva.²⁴

Cigarette smoking

There is an extensive literature addressing the impacts of smoking on oral health. The vasoconstrictive effects of nicotine can result in suppressed resting saliva flow, with consequential depressed pH and buffer capacity. In addition, smoking alters the ecology of the oral flora through enhanced growth of facultative anaerobes in supragingival and subgingival plaque. Smokers have higher levels of dental plaque and higher caries rates than non-smokers.

The saliva film in smokers is thinner and slower moving. Its poorer defensive capabilities explain why smokers are more likely to develop carious lesions on the proximal surfaces of posterior teeth and the cervical aspects of maxillary anterior teeth and mandibular premolars (Figure 7.8).

Smoking also suppresses the healing of oral wounds, and this contributes to an increased severity of destructive periodontitis. There is an increased risk of malignancies in the oral cavity and in other sites such as the larynx, bladder, and pancreas. Acute necrotising ulcerative gingivitis, mucosal pigmentation, and halitosis are also strongly linked to smoking.

Alcohol intake

Regular intake of large quantities of alcohol can exert a range of effects on oral health, including salivary dysfunction, as a consequence of the diuretic effects of ethanol and the resulting nega-

tive fluid balance. This can contribute to both caries and erosion and the latter can be exacerbated by the reflux of gastric acid contents following binge drinking. Mixing alcohol with a range of carbonated drinks containing high levels of sucrose, combined with depressed saliva flow rate from dehydration, can result in increased caries activity.

Chronic alcoholism will lead to cirrhosis of the liver and the resultant scarring will influence the production of a range of water binding proteins such as albumin, leading to disturbances in fluid balance. These changes, in turn, reflect back into the production rate of saliva. Reductions in resting saliva flow and pH have been seen in a range of liver conditions including both chronic Hepatitis B and chronic Hepatitis C infections, as well as in alcoholic cirrhosis.

Other oral health impacts of alcohol abuse

include neglect of home oral health care, interactions with numerous drugs including the antibiotic metronidazole, erratic attendance at dental appointments and a tendency to bleeding. Impaired wound healing will be a secondary effect from liver dysfunction if alcoholic liver cirrhosis has developed. Trauma to the teeth and face from falls and other accidents will heal slowly. High levels of alcohol ingestion have been linked with an increased risk of oral malignancy in smokers via a co-factor effect, however this association does not occur with mouthrinses containing alcohol.

Illicit drug use

Numerous illicit substances cause salivary dysfunction, either through direct pharmacological effects on the central nervous system, or through secondary pathways such as release of antidiuretic hormone (ADH). Examples of illicit substances



Fig. 7.8a & b. Cigarette smokers: Pattern of cervical demineralisation from reduced saliva protection.



Fig. 7.8c & d. Severe cervical caries, ranging from small lesions to frank cavitations with a predominantly cervical pattern.

linked to dry mouth and extensive dental caries include cannabis (marijuana), LSD, cocaine, amphetamines, and the opiates, heroin, morphine, endone, methadone.

Depressed resting saliva flow rate will contribute to an increased risk of caries through impaired clearance of cariogenic fermentable substrates, reduced repair of dental hard tissues through impaired remineralisation, and reduced salivary antimicrobial mechanisms. Addicts may neglect regular oral hygiene practices such as toothbrushing and generally have a high intake of sweets, soft drinks, and foods with a high sucrose level. The salivary dysfunction combined with this cariogenic diet explains why there is a higher caries rate and greater tooth loss than others in the same age group. There are behavioural issues too in as much as opiate-injecting drug users attend for dental care on an irregular basis, and usually only when there is a need for relief of acute dental pain.³⁵

Methadone may be used as treatment for drug users as part of their management, essentially the replacement of one opiate with another. It is delivered orally in a syrup of sorbitol and glycerol and has been identified as being cariogenic because of the pharmacological effects rather than personal neglect.³⁶⁻⁴⁰ However, there is a misconception that the increased caries risk is due to the syrup, in the mistaken belief that this is formulated with sucrose. In fact the base is sorbitol and this is a naturally occurring polyol which yields little in the way of acidic fermentation products from the pathway of glycolysis. It has a cariogenicity

approximately 10% of that of sucrose on a per-weight basis, and cannot be fermented effectively by *S. mutans*, even though other oral organisms can ferment it (Table 7.2).

In reality, dietary alterations are of greater significance due to pharmacological effects of methadone on those regions of the brain that influence food preference. Methadone causes a desire and craving for sweetness^{38,39}, which in turn leads to a high sugar intake in the form of refined sugars with high cariogenicity.³⁸

Diabetes mellitus

In poorly controlled diabetes mellitus there will be a reduction in both resting and stimulated salivary flow rates and a reduced pH. Poor blood glucose control can increase the polyuria associated with diabetes leading to negative fluid balance and dehydration. With long-standing disease lymphocytic infiltration of the major salivary glands may lead to reductions in both resting and stimulated salivary flow. In some patients, bilateral enlargements of the parotid salivary glands may occur.

Diabetics, like patients with bulimia, may collapse in the dental surgery from hypoglycaemia. Loss of consciousness may be preceded by a range of symptoms, including heart palpitations, sweating, confusion, irritability, headache, and seizures. To assist in the rapid and effective management of hypoglycaemia, glucose syrups or other readily available sources of glucose should be available in all dental practices. Susceptible patients should be advised to eat a light meal or snack prior to dental appointments, to reduce the risk.

Strong linkages have been reported between diabetes mellitus and a range of oral health conditions particularly periodontitis, oral fungal infections and dental caries (Figure 7.9). An underlying dehydration in the poorly controlled patient can induce salivary dysfunction with depressed resting flow and pH resulting in an aciduric oral flora. This may lead to the development of caries and oral fungal infections with *Candida albicans*. In addition, lymphocytes may infiltrate the salivary glands resulting in a reduced stimulated salivary flow as glandular capacity reduces over time.

Patients with Type I diabetes and severe

TABLE 7.2: Ability to ferment selected substrates

Micro-organism	Sucrose	Glucose	Sorbitol
<i>Streptococcus mutans</i>	+	+	-
<i>Lactobacillus casei</i>	+		+
<i>Enterococcus faecalis</i>	+	+	-
<i>Streptococcus salivarius</i>	-	-	+
<i>Moraxella catarrhalis</i>	+	-	+
<i>Corynebacterium spp.</i>	+	-	+

periodontal disease show a higher prevalence of a range of diabetic complications compared with diabetics with only minor periodontal disease because of the release of potent pro-inflammatory mediators into the circulation. Side effects can include renal disease, stroke, transient ischaemic attacks, angina, myocardial infarcts and heart failure.⁴³

Noninsulin-dependent Type II diabetes mellitus has been linked with a high carbohydrate lifestyle, and this, of course, has implications for coronal and root surface caries. Poorly controlled diabetes results in polyuria which will in turn impact on resting salivary parameters. Periodontitis in these patients may exacerbate the severity of the diabetic state by decreasing glycaemic control.⁴⁴



Fig. 7.9a. Diabetic patients: This patient has attachment loss, xerostomia and root surface caries.



Fig. 7.9c. This patient has attachment loss, xerostomia, root surface caries, coronal caries and a periodontal abscess on the buccal of tooth 43.

Modifications in Treatment

As discussed above, the general health status and lifestyle of a patient can have a considerable influence on oral health. At the same time there are many factors to be taken in to account before deciding on a final treatment plan. The following items may need consideration:

- the physical environment of the practice including the positioning of the dental chair
- choice of local anaesthetic agent or vasoconstrictor
- timing and duration of an appointment
- pre-operative medications such as antibiotics, anxiolytic agents or steroids



Fig. 7.9b. An OPG radiograph of another diabetic patient shows extensive bone loss and coronal and root surface caries.



Fig. 7.9d. Bone loss in the furcation of tooth 14 has been followed by root surface caries.

- techniques for haemostasis
- need for relative analgesia
- pre-treatment medical consultation
- pre- or post-operative analgesia
- changes in prescription medications or homecare products
- strategies used to preserve oral health, e.g. retention versus removal of teeth for patients undergoing radiotherapy for head and neck malignancies.

Dental caries and general health

Severe dental caries can impact on general health through a range of mechanisms. Loss of teeth can affect masticatory function leading to changes in food selection and nutrition. Dental pain can cause significant morbidity and alterations in lifestyle as particular types of foods or mandibular movements are avoided. It will also disrupt sleep and affect work performance.

When endodontic lesions develop as a consequence of untreated caries, a range of acute phase proteins and cytokines are released into the circulation with possible systemic effects including fever and malaise. Bacterial infection may arise from an endodontic focus in patients with severely compromised immune systems.⁴⁶

Masticatory function may decline with tooth loss leading to modification to the nutritional status. Food may be overcooked to soften it leading to reduced levels of vitamins. Masticatory efficiency will reduce as opposing tooth units are lost or become painful and unstable dentures can lead to a reduction in resting salivary flow. Several studies have shown an important link between masticatory function and the loss of teeth. One study⁴⁷ showed that 38% of 1,160 elderly patients reported difficulties in chewing at least one of six common foods, despite the fact that 57% of the dentate patients and virtually all of the edentulous patients wore dentures. There was strong linkage between difficulty in chewing and the loss of teeth. Nearly 60% of edentulous patients reported difficulty in chewing, whereas only 6% of patients with fewer than nine missing teeth reported problems chewing common foods.

It seems that edentulous patients consume fewer vegetables, less fibre, less carotene (a pre-

cursor of vitamin A), more cholesterol, more saturated fat, and more calories than those who have twenty five or more natural teeth. These changes may enhance the risks of cardiovascular disease and various forms of cancer and this in turn suggests that preservation of a healthy functioning dentition is an important goal in general health care.⁴⁷

Osteoporosis in the elderly, both females and males is attracting increasing interest. It has been linked to a range of lifestyle factors including high alcohol intake, lack of exercise, smoking, caffeine and the intake of black cola soft drinks. A number of these same factors exert an influence on resting saliva flow and the oral flora. This means that patients being counselled on these measures for their oral health, may also be receiving advice that is useful for their general health.

Dietary advice for all patients should stress balance across the diet and also address the issue of so called empty calories. The term empty calories describes foodstuffs which do not contain fibre protein, vitamins or minerals, for example black cola soft drinks. A glass of black cola soft drink (250 mL) contains approximately 150 kilocalories of energy which is equivalent to two large slices of whole wheat bread or a handful of grapes. However, only the grapes and the bread contain dietary fibre, protein, vitamins and minerals.

Home care programs for patients with salivary dysfunction

Frequent use of bland rinses containing saline or sodium bicarbonate and the application of rehydrating or mucosal protective products, such as Laclede Oral Balance gel, can provide relief from symptoms of oral dryness. Patients with profound xerostomia suffer from mucosal irritation and many are unable to tolerate the use of some commercial rinses which contain alcohol or flavouring agents. Salivary stimulants, such as pilocarpine may be useful but may lead to side effects. It is essential that sugar and acid-containing sweets be discouraged as salivary stimulants because they accelerate caries in the presence of low saliva flow. Patients should be encouraged to apply a lip lubricant at regular intervals.

Use of sugarless chewing gums such as

Recaldent™ (Chapter 8) is of major importance for promoting salivary function. The antibacterial, buffering, and lubricant qualities of saliva improve with increasing flow rates, so that stimulating the flow with gum chewing will increase the protection from the salivary secretions. Other ingredients in chewing gums, such as xylitol and phosphopeptides, provide additional preventive benefits and assist in maintaining oral health.

Home use of a neutral sodium fluoride gel or high fluoride dentifrice on a daily basis will maintain salivary fluoride concentrations and inhibit mineral loss. Neutral fluoride gel may be applied on a toothbrush but acidulated fluoride products are contraindicated for patients with severe salivary dysfunction because they may cause erosion, dentine hypersensitivity, or mucosal irritation.

Xylitol and phosphopeptides exert modest suppressive effects on *S. mutans*, however additional antimicrobial targeting is worthwhile in patients with very high caries activity. A water based 0.2% chlorhexidine gluconate gel, applied with a toothbrush, is the agent of choice for chemical plaque control in patients with salivary dysfunction, since it possesses proven antiplaque, antigingivitis, and anticaries activity. The alcohol content of some of the other chlorhexidine rinses is not recommended because it may cause mucosal burning and may reduce saliva flow.

Topical CPP-ACP preparations such as GC Tooth Mousse™ are available as topical thick pastes for clinical use in tray carriers or brushed directly onto the teeth. In addition to their value in preventing caries or erosion, they control cervical hypersensitivity, a condition often associated with reduced salivary flow or low pH conditions. The thick creamy consistency of CPP-ACP products is well suited for self-application by patients and is completely safe if ingested and this is an important consideration for products which will be used by patients in their home.

Antifungal therapy may be required in xerostomic patients with a history of recurring oral candidosis. Chlorhexidine gel exerts mild antifungal effects and can be used together with alkalinising mouthrinses, such as sodium bicarbonate, to suppress levels of *Candida* species. If dedicated topical antifungal agents are required, care must be

taken that the preparation is free of sucrose or other fermentable substrates. Suspensions or lozenges of nystatin or amphotericin are used commonly for treatment of candidoses. Tissue surfaces beneath dentures and the denture surface itself are typically heavily contaminated with fungal organisms. The denture surfaces can be cleaned and then the denture immersed for a short period in dilute sodium hypochlorite to reduce the fungal load. In addition, antifungal creams or suspensions such as miconazole can be directly applied to the fitting surface of a denture, and the denture returned to the mouth to give a sustained effect. Severe denture-related fungal infections require expert attention.

Regular dental review (at least three monthly recall) is important for ensuring that oral hygiene is maintained at an adequate standard, and caries and other conditions are controlled. Maintenance dental sessions should include oral hygiene reinforcement, removal of plaque, and surface treatments with fluoride varnish or phosphopeptides. Adequate monitoring will ensure that the oral health status is maintained, and the need for extensive restorative work is minimised.

Home care for patients with salivary dysfunction

1. Ensure adequate hydration

- Limit intake of caffeine, alcohol, and other diuretics
- Ensure adequate water intake
- Use an oral hydrating gel
- Apply lip balm regularly
- Sleep on the side to avoid mouth breathing at night
- Construct a denture with an internal reservoir

2. Increase the pH and buffer capacity of saliva

- Gain extrinsic bicarbonate via mouthrinse or toothpaste
- Increase extrinsic bicarbonate via chewing a sugar-free gum
- Rinse with water after ingesting acidic foods and drinks

3. Promote remineralisation

- Use a phosphopeptide chewing gum or topical gel at home
- Use a fluoride dentifrice (1000 or 5000 ppm)

- for tooth brushing
- Tooth surface protection with high fluoride glass-ionomer
- 4. **Use dentifrice as a self-applied topical agent for early cervical and proximal lesions**
 - Use home fluoride gel (1.23% neutral NaF) in high risk patients
 - Apply fluoride varnish at recall appointments
 - Use minimal intervention approach for cavity design
 - Use glass-ionomers to restore lesions as much as possible
- 5. **Suppress cariogenic micro-organisms**
 - Dietary modification
 - Reduce the frequency of cariogenic snacks between meals
 - Reduce levels of acidic foods and drinks between meals
- Use a xylitol or CPP-ACP chewing gum
- Apply 0.2% chlorhexidine gel intermittently once a week
- 6. **Improve oral hygiene**
 - Regular toothbrushing twice daily
 - Regular interdental cleaning once daily
 - Use a detergent free dentifrice if mucosal burning occurs i.e. free of sodium lauryl sulphate
- 7. **Assess outcomes**
 - Review salivary parameters at recall appointments
 - Monitor salivary levels of *S. mutans*
 - Monitor caries increment
 - Look for caries arrest and reversal
 - Look for changes in the mineralising potential of saliva

Further Reading

1. Walsh LJ. Preventive dentistry for the general dental practitioner. *Aust Dent J* 2000; 45:76-82.
2. Edgar WM, Higham SM. Role of saliva in caries models. *Adv Dent Res* 1995; 9:235-238.
3. Imfeld, T. Chewing gum- facts and fiction: a review of gum-chewing and oral health. *Crit Rev Oral Biol Med* 1999; 10:405-419.
4. Edgar WM, Higham SM, Manning RH. Saliva stimulation and caries prevention. *Adv Dent Res* 1994; 8:239-245.
5. Pearce EI, Coote GE, Larsen MJ. The distribution of fluoride in carious human enamel. *J Dent Res* 1995; 74:1775-1782.
6. Geddes DA. Diet patterns and caries. *Adv Dent Res* 1994; 8:221-224.
7. Queiroz CS, Hayacibara MF, Tabchoury CP, Marcondes FK, Cury JA. Relationship between stressful situations, salivary flow rate and oral volatile sulfur-containing compounds. *Eur J Oral Sci* 2002; 110:337-340.
8. Frutos R, Rodriguez S, Miralles-Jorda L, Machuca G. Oral manifestations and dental treatment in menopause. *Med Oral* 2002; 7:26-35.
9. Laine M, Pienihakkinen K, Ojanotko-Harri A, Tenovuo J. Effects of low-dose oral contraceptives on female whole saliva. *Arch Oral Biol* 1991; 36:549-552.
10. Kivela J, Laine M, Parkkila S, Rajaniemi H. Salivary carbonic anhydrase VI and its relation to salivary flow rate and buffer capacity in pregnant and non-pregnant women. *Arch Oral Biol* 2003; 48:547-551.
11. Reynolds EC. The prevention of sub-surface demineralisation of bovine enamel and change in plaque composition by casein in an intra-oral model. *J Dent Res* 1987; 66:1120-1127.
12. Reynolds EC. Anti-cariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. *Spec Care Dentist* 1998; 18:8-16.
13. Reynolds EC. Remineralization of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions. *J Dent Res* 1997; 76:1587-1596.
14. Fure S, Lingstrom P, Birkhed D. Effect of three months frequent use of sugar-free chewing gum with and without urea on calculus formation. *J Dent Res* 1998; 77:1630-1637.
15. Hujoel PP, Makinen KK, Bennett CA, Isotupa KP, Isokangas PJ, Allen P, Makinen PL. The optimum time to initiate habitual xylitol gum-chewing for obtaining long-term caries prevention. *J Dent Res* 1999; 78:797-803.
16. Makinen KK, Bennett CA, Hujoel PP, Isokangas PJ, Isotupa KP, Pape HR. Xylitol chewing gums and caries rates: a 40 month cohort study. *J Dent Res* 1995; 74:1904-1913.
17. Macpherson LMD, Chen WY, Dawes C. Effects of salivary bicarbonate content and film velocity on pH changes in artificial plaque containing *Streptococcus oralis* after exposure to sucrose. *J Dent Res* 1991; 70:1235-1238.
18. Edwardsson D, Mejare B. *Streptococcus milleri* and *Streptococcus mutans* in the mouths of infants before and after tooth eruption. *Arch Oral Biol* 1978; 23:811-814.

19. Milgrom P, Riedy CA, Weinstein P, Tanner ACR, Manibusan L, Bruss J. Dental caries and its relationship to bacterial infection, hypoplasia, diet and oral hygiene in 6- to 36-month-old children. *Comm Dent Oral Epidemiol* 2000; 28:295-306.
20. Wan AKL, Seow WK, Walsh LJ, Purdie DM, Bird PS, Tudehope DI. Association of *Streptococcus mutans* colonization and oral developmental nodules in predentate infants. *J Dent Res* 2001; 80:1945-1948.
21. A K L Wan, W K Seow, D M Purdie, P S Bird, Tudehope DI, Walsh LJ. Oral colonization of *Streptococcus mutans* in six-month-old predentate infants. *J Dent Res* 2001; 80:2060-2066.
22. Berkowitz RJ, Jordan HV, White G. The early establishment of *Streptococcus mutans* in the mouths of infants. *Arch Oral Biol* 20:171-174.
23. Berkowitz RJ, Turner J, Green P (1981). Maternal salivary levels of *Streptococcus mutans* and primary oral infection in infants. *Arch Oral Biol* 1975; 26:147-149.
24. Walsh LJ. *Microbiology*. 4th Ed. Sydney: Royal Australasian College of Dental Surgeons Incorporated. 2003. pp. 21-40.
25. Khan F, Daley T, Young WG. Dental erosion and bruxism. A tooth wear analysis from south east Queensland. *Aust Dent J* 1998; 43:117-27.
26. Walsh NP. Saliva flow rate, total protein concentration and osmolality as potential markers of whole body hydration status during progressive acute dehydration in humans. *Arch Oral Biol* 2004; 49:149-154.
27. Watson JM, Lunt MJ, Morris S, Weiss MJ, Hussey D, Kerr D. Reversal of caffeine withdrawal by ingestion of a soft beverage. *Pharmacol Biochem Behav* 2000; 66:15-18.
28. Benjamin LT, Rogers AM, Rosenbaum A. Coca-Cola, caffeine, and mental deficiency: Harry Hollingworth and the Chattanooga trial of 1911. *J History Behav Sci* 1991; 27:42-55.
29. Zorc B, Grga D. Caffeine. *Farmaceut Glasn* 1998; 54:437-441.
30. Barone JJ, Roberts HR. Caffeine consumption. *Food Chem Toxicol* 1996; 34:119-129.
31. Weinmann S et al. Caffeine intake in relation to the risk of primary cardiac arrest. *Epidemiology* 1997; 8:505-508.
32. Fernando GR. Consumption of softdrinks with phosphoric acid as a risk factor for the development of hypocalcaemia in post-menopausal women. *J Clin Epidemiol* 1999; 52:1007-1010.
33. Petridou E. The role of daily products and non-alcoholic beverages in bone fractures amongst school-aged children. *Scand J Soc Med* 1997; 25:119-125.
34. Wyshak G, Frisch RE. Carbonated beverages, dietary calcium, the dietary calcium/phosphorus ratio, and bone fractures in girls and boys. *J Adolesc Hlth* 1994; 15:210-215.
35. Carter EF. Dental implications of narcotic addiction. *Aust Dent J* 1978; 23:308-310.
36. Sheedy JJ. Methadone and caries. Case reports. *Aust Dent J* 1996; 41:367-369.
37. Titsas A, Ferguson MM. Impact of opioid use on dentistry. *Aust Dent J* 2002; 47:94-98.
38. Zador D, Lyons Wall PM, Webster I. High sugar intake in a group of women on methadone maintenance in south western Sydney, Australia. *Addiction* 1996; 91:1053-1061.
39. Asghar K. Role of dietary and environmental factors in drug abuse. *Alc Drug Res* 1986; 7:61-83.
40. Colon PG. Heroin abuse and its effects on teeth. *Quint Int* 1975; 6:103-108.
41. Coates EA, Brennan D, Logan RM, Goss AN, Scopacasa B, Spencer AJ, Gorkic E. Hepatitis C infection and associated oral health problems. *Aust Dent J* 2000; 45:108-114.
42. Coates EA, Walsh LJ, Logan R. The increasing problem of hepatitis C virus infection. *Aust Dent J* 2001; 46:13-17.
43. Thorstensson H. Periodontal disease in adult insulin dependent diabetics. *Swed Dent J (Suppl)* 1995; 65:1068-1071.
44. Kinane DF, Marshall GJ. Periodontal manifestations of systemic disease. *Aust Dent J* 2001; 46:2-12.
45. Walsh LJ. Serious complications of endodontic infections. Some cautionary tales. *Aust Dent J* 1997; 42:302-306.
46. Slade GD, Spencer AJ, Roberts-Thomson K. Tooth loss and chewing capacity among older adults in Adelaide. *Aust NZ J Publ Hlth* 1996; 20:76-82.
47. Joshipura KJ. The impact of edentulousness on food and nutrient intake. *JADA* 1996; 127:459-467.

8 Additional Aids to the Remineralisation of Tooth Structure

E. C. Reynolds ♦ L. J. Walsh

Progress of the caries lesion from the very earliest signs of demineralisation is now well understood. The challenge in recent years has been to find reliable techniques to reverse this action. Much work has been carried out using the fluoride ion and it has now been shown that this has multiple actions in relation to caries. It certainly discourages demineralisation because, if it is incorporated with the enamel, it forms fluorapatite and this has a lower critical pH than hydroxyapatite. It also discourages the formation of bacterial plaque on the tooth surface and, if present at low levels in the saliva, it is always available during the process of remineralisation to again form fluorapatite.

However, the remineralisation developed by these means is superficial only leaving lower levels of demineralised enamel and dentine with a degree of porosity and incomplete mineralisation. A technique has now been developed to overcome these limitations wherein calcium, phosphate and fluoride ions can be persuaded to remineralise in depth, virtually eliminating white spot lesions and restoring full aesthetics to enamel and resistance to further acid attack.



Introduction

This chapter describes the early stages of caries development by examining the chemical processes of enamel demineralisation.¹ It then focuses on reversal of that process leading to enamel subsurface lesion remineralisation. Even though remineralisation of early enamel caries is now well established through the use of fluorides, it seems the full potential has not previously been realised. Tooth mineral is predominantly composed of calcium phosphate in the form of apatite and even though fluoride can drive the formation of fluorapatite ($\text{Ca}_{10}(\text{PO}_4)_6\text{F}_2$), it is clear that for every two fluoride ions, ten calcium ions and six phosphate ions are required to form one unit cell of fluorapatite. This, together with the intrinsic insolubility of calcium phosphates and in particular calcium fluoride phosphates, is the reason why fully effective products have not been developed for professional use to reverse early enamel caries.

A new remineralisation technology has been developed based upon phosphopeptides from milk casein. These casein phosphopeptides (CPP) contain multiphosphoserine sequences that have the ability to stabilise calcium phosphate in nanocomplexes in solution as amorphous calcium phosphate (ACP). Through their multiple phosphoserine sequences, the CPP bind to ACP in metastable solution thus preventing its growth to the critical size required for nucleation and phase transformation to an insoluble crystalline calcium phosphate. The casein phosphopeptide-amorphous calcium phosphate nanocomplexes (CPP-ACP) have been shown to localise at the tooth surface and prevent enamel demineralisation in laboratory, animal and human *in situ* trials. The CPP-ACP have also been shown to remineralise enamel subsurface lesions *in situ* and *in vivo* when delivered in consumer oral care and professional products. The CPP-ACP interact with fluoride ions to produce an amorphous calcium fluoride phosphate stabilised by the CPP at the tooth surface. This provides soluble calcium, fluoride and phosphate ions to promote remineralisation with

fluorapatite that is more resistant to acid challenge. Oral care products containing CPP-ACP (Recaldent™) are now commercially available.

Dental caries

Dental caries is a pathological process of localised destruction of tooth tissue by micro-organisms. The disease is initiated via the demineralisation of tooth hard tissue by organic acids, produced from fermentable carbohydrate, by the cariogenic bacteria resident in dental plaque. Even though, in most developed countries, the prevalence of dental caries has decreased through the use of fluorides, the disease remains a major public health problem. The estimated economic burden of treating dental caries in most developed communities is higher than that for other dietary related diseases including coronary heart disease, hypertension or stroke.

The processes involved in the development of an incipient enamel carious lesion are now well understood. Frequent exposure to dietary fermentable carbohydrate, in particular dietary sugars, leads to an increase in the population of cariogenic bacteria² (e.g. *Streptococcus mutans*, *Lactobacillus* spp. and other species) in the biofilm. These bacteria are both acidogenic and aciduric, that is they are prolific producers of organic acids, in particular lactic acid and actively produce that acid at a pH value around 5, which is below the critical pH range for enamel apatite, resulting in its dissolution. Commensal supragingival plaque bacteria can subsist on sugars obtained from salivary glycoproteins at low concentrations, and metabolism in this restricted environment produces metabolic end products like acetic acid, formic acid and ethanol. Although this energy-efficient pathway will lead to the excretion of organic acids, the low rate of production and the intrinsic buffering capacity of plaque and saliva generally results in a plaque pH which is not below the critical pH range for enamel. This means that, under most conditions, there may be growth of a complex bacterial community on the surface of the tooth without net enamel loss.

However, frequent exposure to high concentrations of dietary sugars will produce a transition in

the composition of the bacterial community and dominance of cariogenic bacteria. These organisms have evolved to cope with high concentrations of sugar by utilising lactate dehydrogenase, as well as other glycolytic enzymes, to efficiently convert the excess sugar to lactic acid, and then excrete it. This leads to an acidic extracellular environment which is hostile to the majority of commensal bacteria. However, the cariogenic bacteria survive well through efficient intracellular pH control mechanisms involving transmembrane H^+ translocating ATPase enzymes. This means that cariogenic bacteria can proliferate and produce lactic acid in an acidic environment below the critical pH range for enamel.

The critical pH is defined as the highest pH value where tooth enamel undergoes net mineral loss. The actual value depends on the activities of Ca^{2+} and PO_4^{3-} ions in the hydration layer around the enamel crystals, and these are generally in equilibrium with the concentrations of the same ions in saliva and plaque fluid. Based on the known average Ca^{2+} and PO_4^{3-} concentrations in saliva and plaque fluid, supported by laboratory studies, the critical pH for tooth enamel is reported to be between 5.2 and 5.5. However, natural tooth enamel is a calcium-deficient, carbonated enamel and the higher the carbonate content the greater the solubility and, therefore, the higher the critical pH. It is also known that the presence

of fluorapatite in enamel lowers solubility and therefore lowers the critical pH.

During the caries process the organic acids produced by the plaque bacteria, predominantly in their undissociated form (e.g. lactic acid shown as HL in **Figure 8.1**), diffuse into the tooth enamel via the water-filled interprismatic spaces and lower the pH of the hydration layer around the enamel crystals. Once the pH falls below the critical value the enamel crystals will undergo net mineral loss in a process referred to as demineralisation. Lactic acid is effective at dissolving apatite crystals as the acid (H^+) reduces PO_4^{3-} and OH^- activities and, through chelation, the lactate anion (L^-) reduces Ca^{2+} activity.

The 'white spot' lesion

The loss of calcium phosphate from the enamel structure results in the development of an incipient subsurface enamel lesion which can be recognized as a 'white spot'. The white appearance is due to optical phenomena associated with increased enamel porosity. At this stage, the process is reversible and it is possible for calcium and phosphate ions, particularly the neutral ion pair $CaHPO_4^0$, to diffuse into the subsurface lesion to restore the lost apatite in a process referred to as 'remineralisation'. However, the clinical application of remineralisation solutions containing calcium and phosphate ions has not been successful

up to date due to the low solubility of calcium phosphates, particularly in the presence of fluoride ions. Insoluble calcium phosphates are not easily applied, do not localize effectively at the tooth surface and require acid, for solubility, to produce ions capable of diffusing into the subsurface lesion. Soluble calcium phosphate ions will be at low concentrations, will not substantially incorporate into the biofilm or localise at the tooth surface, and will wash away before

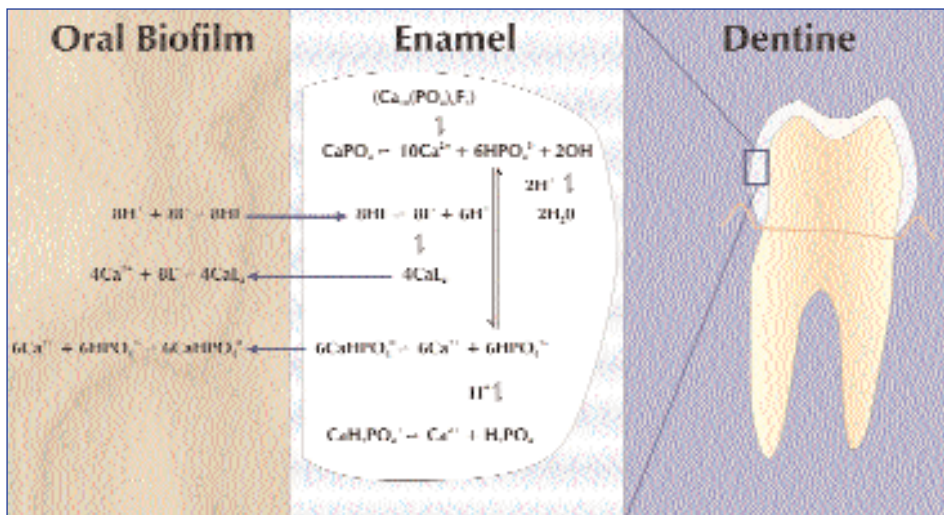


Fig. 8.1. Enamel demineralisation: The chemical speciation of the demineralisation process at the plaque-enamel interface resulting in an incipient carious lesion (white spot).

effective diffusion into the subsurface lesion can occur. A new technology has now emerged which will generate an amorphous form of calcium phosphate, stabilised by phosphopeptides from the milk protein casein, which will make calcium phosphate available for effective remineralisation.

Anti-cariogenic casein phosphopeptides

The food group most recognised as exhibiting anti-caries activity is dairy products (milk, milk concentrates, powders and cheeses). Using *in vitro*, animal and *in situ* caries models, the components largely responsible for this anti-cariogenic activity have been identified as casein, calcium and phosphate.⁴ The bovine milk phosphoprotein, casein, which is known to interact with calcium and phosphate and is a natural food component is an obvious candidate for an anti-cariogenic food and toothpaste additive. However, this was originally precluded by organoleptic properties (such as taste, smell, texture), from the use of the very high levels required for activity, and its relative insolubility in the presence of calcium ions. Using an intraoral appliance in a human model, it was shown that digestion of caseinate with trypsin enhances the protein's ability to prevent enamel subsurface demineralisation. In fact, it substantially increases solubility as well as calcium phosphate stabilisation. Tryptic peptides of casein were found incorporated into the plaque associated with a substantial increase of calcium and phosphate. It was concluded that the tryptic peptides that were responsible for the anti-cariogenic activity were the calcium phosphate sequestering casein phosphopeptides (CPP) containing the active multiphosphoseryl sequence -Ser(P)-Ser(P)-Ser(P)-Glu-Glu-⁴

These peptides are 10% w/w of caseinate and through their multiple phosphoseryl residues are able to sequester their own weight in calcium phosphate to form colloidal nanocomplexes. The CPP are not associated with the unpalatability or allergenicity of the caseins and they are soluble at acid pH values. They also have the potential for a specific anti-cariogenicity at least ten times greater on a weight basis, so their potential as

additives to oral care products is considerably better than that of the intact proteins.

Interaction of casein-phosphopeptides with calcium phosphate

The CPP have a substantial ability to stabilise calcium phosphate in solution. The calcium phosphate phase, stabilised by the CPP, is a basic amorphous calcium phosphate phase ($\text{Ca}_{3.0877}(\text{PO}_4)_2(\text{OH})_{0.1754x}\cdot\text{H}_2\text{O}$). In neutral and alkaline supersaturated calcium phosphate solutions basic ACP nuclei spontaneously form. The CPP bind to forming ACP nanoclusters, thus preventing their growth to the critical size required for nucleation and phase transformation, thereby producing a metastable solution. A 1.0% w/v CPP solution can stabilise 60 mM CaCl_2 and 36 mM sodium phosphate at pH 7.0 to form colloidal amorphous calcium phosphate-CPP nanocomplexes (CPP-ACP). This solution has been studied using a variety of *in vitro*, human *in situ* and animal caries models.

Anti-cariogenicity of CPP-ACP in the rat

The ability of casein-phosphopeptide amorphous calciumphosphate nanocomplexes (CPP-ACP) to reduce caries activity has been investigated using specific-pathogen-free rats orally infected with *Streptococcus sobrinus* 6715WT-13. CPP-ACP solutions, applied to the animals teeth twice daily, significantly reduced caries activity, with 0.1% w/v

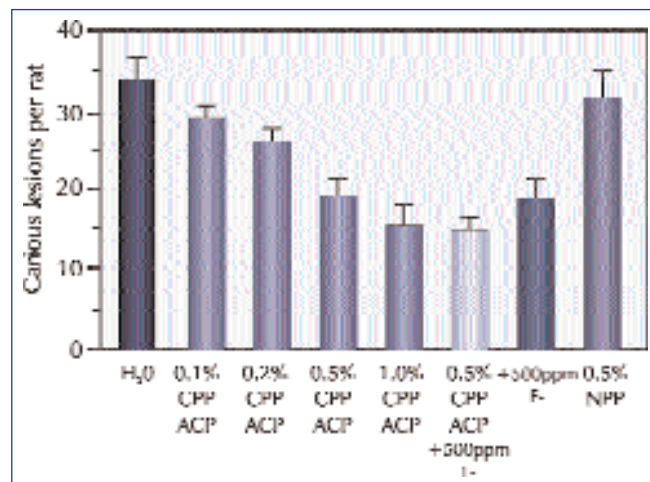


Fig. 8.2. The effect of CPP-ACP solutions on caries activity of rats.

CPP-ACP producing a 14% reduction and 1.0% w/v CPP-ACP a 55% reduction relative to the distilled water control (Figure 8.2).

CPP-ACP at 0.5-1.0% w/v produced a reduction in caries activity similar to that of the 500 ppm F⁻ solution. It was also shown that the anti-cariogenicity of CPP-ACP in combination with fluoride was additive so that animals receiving 0.5% CPP-ACP plus 500 ppm F⁻ had significantly lower caries activity than those animals receiving either CPP-ACP or fluoride alone. The non-phosphorylated casein peptides did not reduce caries activity of the animals.

Anti-cariogenicity of CPP-ACP in human *in situ* studies

Localisation of CPP-ACP at the tooth surface has been demonstrated in an *in situ* model. Enamel specimens were subjected to twice daily exposures of a 1% CPP-ACP solution. This led to a substantial reduction in enamel mineral loss relative to the control enamel. In this study and other mouthrinse clinical trials, supragingival plaque exposed to CPP-ACP solutions contained substantially increased amounts of calcium and phosphate ions when compared with the control plaque. Electron micrographs of immunocytochemically stained sections of supragingival plaque, exposed to the CPP-ACP, revealed localisation of the peptide predominantly on the surface of micro-organisms⁶ but also in the extracellular matrix (Figure 8.3).

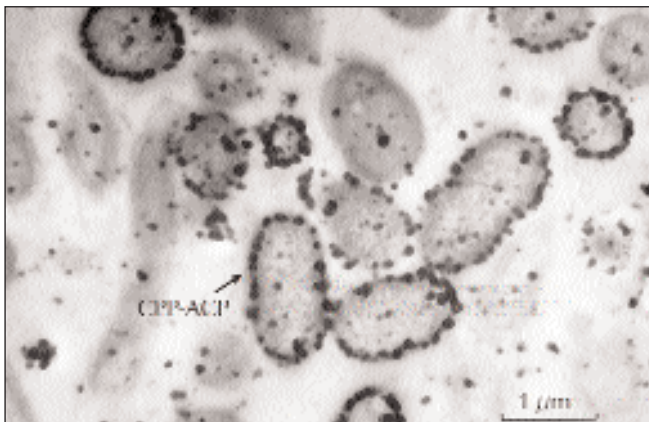


Fig. 8.3. The incorporation of CPP-ACP into supragingival plaque from a mouthrinse.

The CPP-ACP has also been shown to bind to exposed dentine and to occlude tubules (Figure 8.4), possibly explaining the reduction in tooth sensitivity that has been observed in a number of clinical studies.

Enamel remineralisation *in situ*

The ability of CPP-ACP added to sugar-free chewing gum (Recaldent™) to remineralise enamel subsurface lesions has been demonstrated in randomised, controlled, double-blind *in situ* clinical studies.⁵ The sugar-free gums (control and CPP-ACP containing gums) were tested over a period of 14 days. Specimens were chewed for either 20 minutes four times a day or for five minutes, seven times a day. Microradiography and computer-assisted densitometric image analysis demonstrated that gum containing 18.8 mg and 56.4 mg of CPP-ACP, chewed for 20 minutes four times per day, increased enamel subsurface remineralisation by 101% and 151% respectively, relative to the control sugar-free gum (Figure 8.5). Microradiographs of the enamel lesions before and after remineralisation showed that the CPP-ACP promoted remineralisation throughout the body of the lesion. SEM-EDAX (elemental) analyses of sections of the remineralised enamel revealed that the mineral deposit was hydroxyapatite with a higher Ca:P ratio than normal apatite. Acid challenge of the enamel remineralised by CPP-ACP *in*

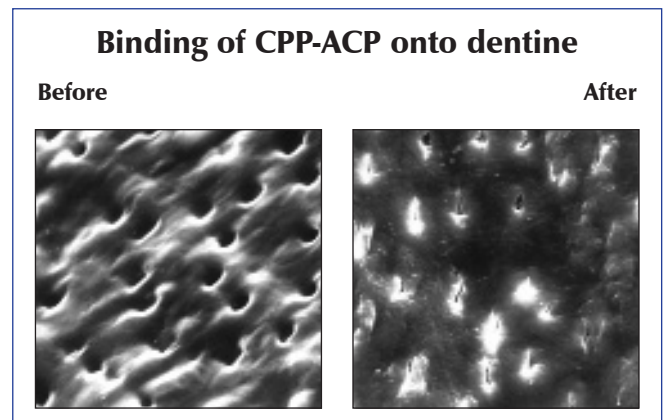


Fig. 8.4. Electronmicrograph demonstrating occlusion of exposed dentinal tubules by CPP-ACP.

situ showed that the remineralised apatite was more resistant to acid challenge than the normal calcium deficient carbonated tooth enamel.

CPP-ACP remineralisation mechanism

From a series of *in vitro* experiments it has been established that the activity of the neutral ion pair CaHPO_4^0 is correlated highly with the rate of rem-

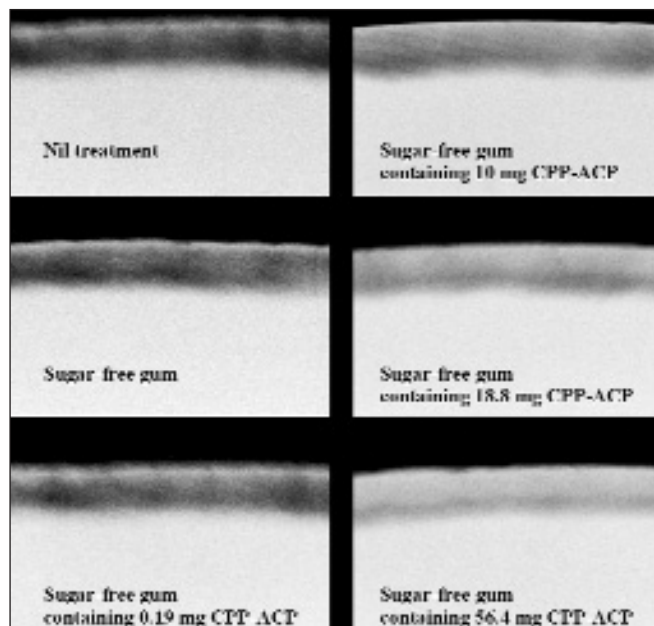


Fig. 8.5. Microradiographic images of enamel subsurface lesions remineralised *in situ* by sugar-free gum containing CPP-ACP.

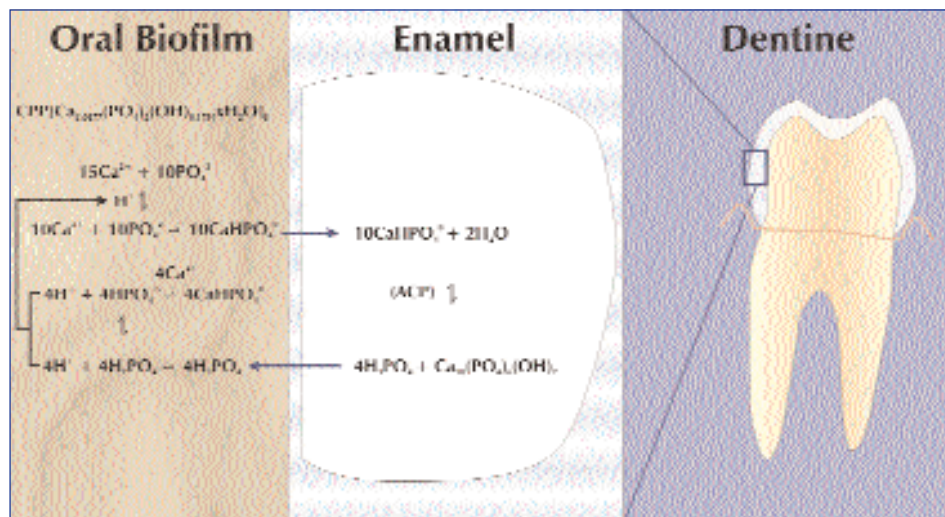


Fig. 8.6. Enamel remineralisation: Proposed mechanism of enamel subsurface lesion remineralisation by CPP-ACP.

ineralisation of enamel subsurface lesions.³ The diffusion coefficient for the remineralisation process was estimated at $3 \times 10^{-10} \text{ m}^2 \text{ s}^{-1}$ which is consistent with the coefficients of diffusion for neutral molecules through a charged matrix. CaHPO_4^0 and associated species diffuse into the enamel lesion and, by the formation of Ca^{2+} and PO_4^{3-} ions, increase the degree of saturation with respect to hydroxyapatite (HA). The formation of HA in the lesion would lead to the generation of acid and phosphate including H_3PO_4 , which would diffuse out of the lesion down a concentration gradient. The results indicate that the CPP-bound ACP, acts as a reservoir of the neutral ion species CaHPO_4^0 that is formed in the presence of acid. (Figure 8.6) The acid could be generated by dental plaque bacteria and under these conditions, the CPP-bound ACP would buffer plaque pH and produce calcium and phosphate ions, in particular CaHPO_4^0 . The increase in plaque CaHPO_4^0 would offset any fall in pH thereby preventing enamel demineralisation. Acid is also generated in plaque as H_3PO_4 by the formation of HA in the enamel lesion during remineralisation. This, therefore, explains why the CPP-ACP are such efficient remineralising agents as they would consume the H_3PO_4 produced during enamel lesion remineralisation, generating more CaHPO_4^0 , thus maintaining its concentration gradient into the lesion. These results are therefore consistent with the proposed anti-cariogenic

mechanism of CPP-ACP being the inhibition of enamel demineralisation and enhancement of remineralisation through the localisation of ACP at the tooth surface (Figure 8.6).

Interaction of CPP-ACP with fluoride

The CPP-ACP interacts with the fluoride ion to produce a novel amorphous calcium fluoride phosphate (ACFP) phase of apparent composition $\text{Ca}_8(\text{PO}_4)_5\text{F} \cdot x\text{H}_2\text{O}$. The identification of this novel

amorphous calcium fluoride phosphate (ACFP) phase led to speculation that it is probably responsible for the additive anti-cariogenic effect of the combination of CPP-ACP with F. It is suggested that the anti-cariogenic mechanism of fluoride arises from the localisation of the fluoride ion at the tooth surface, particularly in plaque, in the presence of Ca and phosphate ions. Localisation increases the degree of saturation with respect to fluorapatite (FA), thus promoting remineralisation of enamel with FA during an acid challenge. It is clear that for the formation of FA ($\text{Ca}_{10}(\text{PO}_4)_6\text{F}_2$), calcium and phosphate ions must also be present with the fluoride ions. The additive anti-cariogenic effect of CPP-ACP and F is therefore attributable to the localisation of ACFP at the tooth surface by the CPP, which, in effect, co-localises Ca, Pi and F.

In randomised, controlled, mouthrinse trials, a rinse containing 4.0% CPP-ACFP significantly increased calcium, inorganic phosphate and fluoride ion content of supragingival plaque when compared with a rinse containing the equivalent concentration of fluoride ions as sodium fluoride. In spite of these marked increases in calcium, phosphate and fluoride ions, calculus was not observed in any of the subjects, suggesting that the calcium fluoride phosphate in the plaque remained stabilised at the tooth surface by the

CPP as the bioavailable amorphous phase, and did not transform into a crystalline phase. The increases in the supragingival plaque levels of bioavailable Ca, phosphate and fluoride ions are markedly greater than those obtained in similar studies with toothpastes containing 1000ppm F (MFP or NaF). These results suggest that the CPP are an excellent delivery vehicle to co-localise bioavailable Ca, fluoride and phosphate ions at the tooth surface in a slow release amorphous form with superior clinical efficacy. Recently, a dentifrice containing 2% CPP-ACP plus 1100 ppm F was shown to promote remineralisation of enamel subsurface lesions with mineral that was resistant to acid challenge. A dentifrice containing 1100 ppm F only with no CPP-ACP was far less efficient.

Commercial development of CPP-ACP

The CPP-ACP technology has been commercially developed and is now available in sugar-free chewing gum (Recaldent™) and in a range of dental professional products such as Tooth Mousse™ manufactured by GC Corporation. The Tooth Mousse™ product contains 10% w/w CPP-ACP and has been used clinically for the treatment of mild to moderate fluorotic lesions (**Figures 8.7a and 8.7b**) and for the reversal of white spots after orthodontic debanding (**Figures 8.8a and 8.8b** following page).



Fig. 8.7. Treatment of mild to moderate fluorosis by microabrasion followed by GC Tooth Mousse™. **a)** Before treatment and **b)** after treatment.

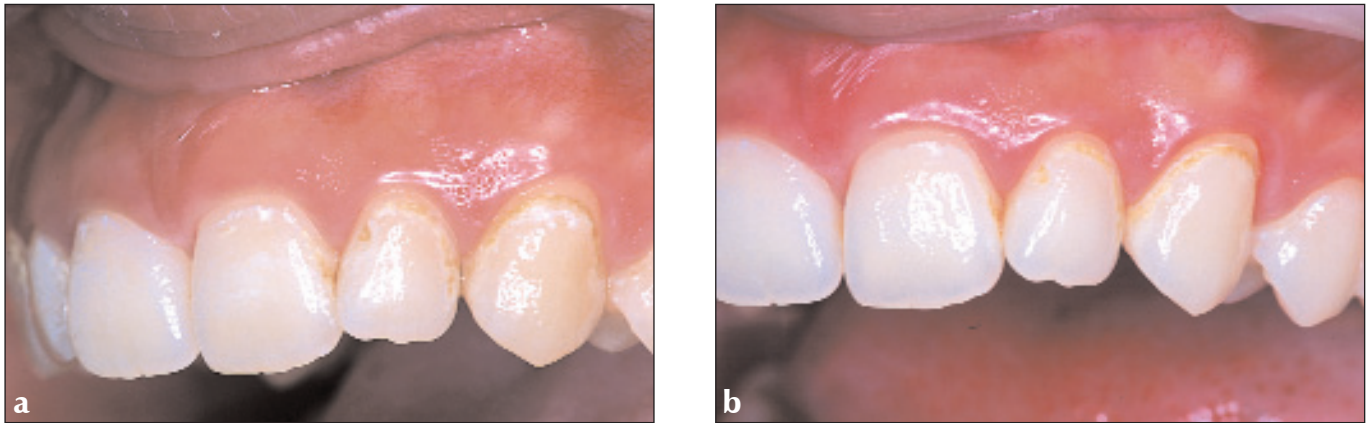


Fig. 8.8. Tooth Mousse™ treatment of white spots after orthodontic debanding. **a)** Before treatment and **b)** after treatment. Images courtesy of Dr Hayashi, Yokohama, Japan.

SUMMARY



The recommended clinical application protocols are

- white spot reversal (post-orthodontic bracket removal, fluorosis, incipient carious lesions)
- post-bleaching
- post-scaling and root planing
- dentinal hypersensitivity
- erosion lesions
- caries prevention
- post-fluoride treatment

Further Reading

1. Featherstone JDB, Duncan JF, Cutress TW. A mechanism for dental caries based on chemical processes and diffusion phenomena during in vitro caries simulation on human tooth enamel. *Arch Oral Biol* 1979; 24:101-112.
2. Loesche WJ. Role of *Streptococcus mutans* in human dental decay. *Micro Rev* 1986; 50:353-380.
3. Reynolds EC. Remineralisation of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions. *J Dent Res* 1977; 6:1587-1595.
4. Reynolds EC. Anti-cariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides. A review. *Spec Care Dent* 1998; 18:1-9.
5. Reynolds EC, Black CL, Cai F, Cross KJ, Eakins D, Huq NL, Morgan MV, Nowicki A, Perich JW, Riley PF, Shen P, Talbo G and Weber FW. Advances in enamel remineralisation: anti-cariogenic casein phosphopeptide-amorphous calcium phosphate. *J Clin Dent* 1999; 10:86-88.
6. Reynolds EC, Cai F, Shen P, Walker GD. Retention in plaque and remineralisation of enamel lesions by various forms of calcium in a mouthrinse or sugarfree chewing gum. *J Dent Res* 2003; 82:206-211.

9 Instruments Used in Cavity Preparation

G. J. Mount ♦ L. J. Walsh ♦ A. Brostek

The stage may be reached in the progression of dental caries where preventive therapy and remineralisation techniques will no longer be successful. In the presence of frank cavitation in both enamel and dentine it will no longer be possible to prevent plaque accumulation. It then becomes essential to surgically debride the lesion and restore the tooth to original anatomy to allow proper cleaning and prevent further plaque accumulation. It is also sometimes necessary to shape and restore teeth damaged by trauma and to remove defective restorations and replace them. Each of these actions requires cutting either the tooth tissues or hard restorative materials.

Until the end of the 1800s hand instruments were the preferred method of cavity preparation and even after the introduction of rotary instruments they were essential to the final refinement of a cavity designed in the G. V. Black tradition. Rotating cutting instruments travelling at increasing speeds then evolved and still remain the most effective means of reducing both tooth tissue and old restorative materials. However, recent advances with both air abrasion and lasers techniques have provided additional instruments to the armamentarium. Furthermore chemo-mechanical techniques for the removal of carious dentine are becoming more sophisticated and are contributing to a reduction in patient discomfort during the surgical phase of operative dentistry. All of these techniques deserve further research and investigation, particularly in the light of the trend to minimal cavity designs.



Rotary Cutting Instruments

Classification of burs

There is a vast array of sizes and shapes of rotary cutting instruments available and, in an attempt to rationalise the selection, the International Standards Organisation has developed a classification (ISO 6360). The essential dimensions of a bur, including the surface material on the bur head, shank type, overall length, shape and type of finish of the working head and the size of the head, are all numerically ordered (Figure 9.1).

‘Annoyance Factor’

The term ‘annoyance factor’ is used rather loosely to describe the patient’s subjective reaction to cavity preparation and is a combination of the pressure applied to the tooth, the vibrations and noise recorded through the bones of the skull, the heat and smell generated at the interface between the tooth and the bur and the time taken to perform a given task (Figure 9.2).

Vibration and noise generation are related closely to the coarseness of the cutting instrument and the speed of rotation. Within limits, the higher the speed and the smaller the instrument the lower

the vibration but concentricity is also a factor and requires maintenance of the handpiece, the bur and the equipment in general. The generation of heat and smell are both closely related to the adequacy of lubrication and the efficiency of the instrument in the removal of the shavings being taken from the tooth surface. All of these factors can be combined to give a rough idea of the problems faced by the patient when a tooth needs to be surgically prepared.

Selection of burs

In general, rotary cutting instruments will remove tooth structure either by chipping it away or else by grinding.^{1,2} There are essentially three types of bur designed for cavity preparation although there are many variations for polishing, contouring etc. This discussion will be confined to those related to preparation of caries lesions only.

Steel burs

These were originally used when rotary cutting instruments were developed well over one hundred years ago.³ Initially only foot drills were available with speeds of about 50-500 rpm. Electric motors followed, allowing speeds up to 5000 rpm, and they remain valuable for removal of caries and development of retentive elements in dentine. Each bur generally has eight blades and

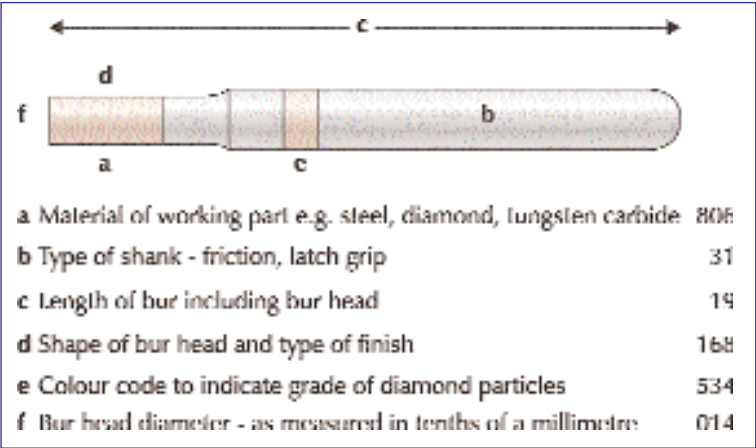


Fig. 9.1. International Standards Organisation (ISO) numbering system for rotary cutting instruments. This is the basic code recommended by ISO to enable a universal numbering system to be developed for recognition of all rotary cutting instruments.

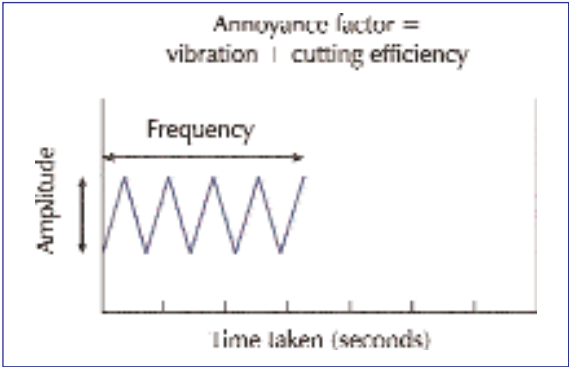


Fig. 9.2. ‘Annoyance factor’: A diagrammatic representation of the relative discomfort suffered by the patient depending on the selection of bur size, shape and surface as well as the speed of rotation and pressure applied during cutting. These factors are then related to the time taken to complete the task and a factor number can be allocated from 1-10.

some of them have a positive rake angle to facilitate the cutting of the dentine or removal of caries. This, however, makes them relatively fragile and subject to chipping along the leading edge and they should not be expected to have a long life in normal practice. In fact single use is recommended.

The use of air/water spray during cutting with steel burs will increase efficiency but is not essential. Generally the shanks are designed for latch type handpieces, but some manufacturers now make friction grip handpiece heads to hold these burs.

The friction grip helps to maintain concentricity and thereby reduce vibration. Any bur rotating at low speeds will deliver a rather high annoyance factor to the patient and a good tactile sense is essential for the operator to avoid over cutting. Burs must always be in good condition and visibility must be excellent at all times.

Tungsten carbide burs

Following the development of higher speed motors and handpieces there was a need to provide stronger burs to withstand the heavier stresses involved and to lengthen their useful life.

SUMMARY ⓘ

- High annoyance factor
- Lubrication **not** essential
- Relatively fragile
- Easily eccentric
- Low speed only

Tungsten carbide burs were designed almost exclusively for friction grip handpieces because concentricity is essential and they only cut efficiently at greatly increased speeds. In fact, they do not begin to reach effective cutting capacity until 100,000 rpm and are best used at speeds beyond 300,000 rpm. The efficiency of these instruments for removing particles of enamel or dentine depends on the number of blades, the rake angle of the blades and other variations in the design. Blades which twist around the shank of the bur will remove debris more readily and blades which are cross cut are more efficient still (**Figure 9.3**). Burs with eight blades or fewer are designed for rough or gross cutting and the greater the number of blades the finer the surface and the smoother the cut. Tungsten carbide burs with thirty blades or more are used for polishing (**Figure 9.4**).

The usual bur has six blades and a negative rake angle to provide better support for the cutting edge. For the same reason, many have a radial clearance too. They cut metal and dentine well but are prone to produce microcracks in enamel thus weakening the cavosurface margin.

It is essential that tungsten carbide burs be used

SUMMARY ⓘ

- Low annoyance factor
- Lubrication essential
- Relatively fragile
- Easily eccentric
- Ultra-high speed only

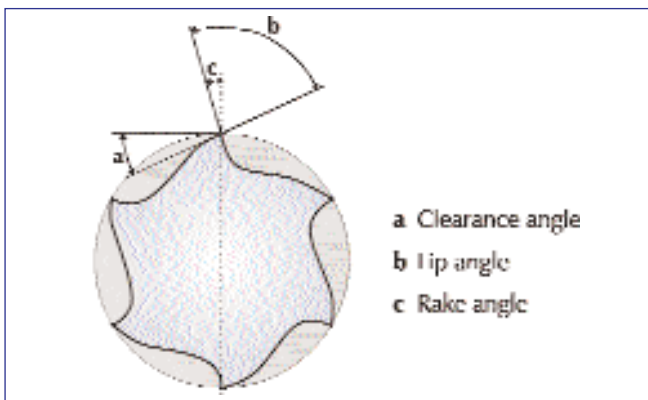


Fig. 9.3. Design of burs: The design of the blades is predicated on the need to eliminate debris rapidly, at the same time preserving the strength in the blades to avoid chipping. Tungsten carbide burs generally have a negative rake angle for additional strength. Many steel burs have a positive rake angle, but are prone to chipping.



Fig. 9.4. Two tungsten carbide burs compared to a fine diamond bur. The one on the left is a crosscut but with six blades and horizontal cuts through the blades to allow better removal of debris. The second tungsten carbide bur is a six bladed bur for fine finishing. The diamond is a fine tapered cylinder bur for opening fissures.

at speeds above 100,000 rpm. Air/water spray is mandatory for the removal of debris and temperature control and they must be mounted in a friction grip cartridge for concentricity. Probably only a new bur will be truly concentric because any loss of a blade, or even a section of a blade, will alter the balance so that only every third or fourth blade will actually contact the tooth. This means that the clinical life is generally quite short. The annoyance factor with a new bur, at high linear surface speed, is very low but a lack of concentricity will be immediately discernible to the patient.

Diamond burs or stones

Diamond burs will grind away tooth structure and they are available in a range of particle size from about 150 micron (μ) down to 5 μ . Diamonds with a particle size of 150 μ are extremely coarse, with a high annoyance factor, so the regular 80 μ particles are the usual selection for basic cavity preparation. Finer diamonds with particle sizes in the 25 μ range are recommended for finishing all margins where adhesive restorative materials are to be placed, while polishing procedures can be carried out with particles down to 5 μ . In recent times there have been considerable improvements in the methods of embedding the diamond particles in the metal of the bur head so they last longer and there is a far better distribution of particle

size. The particles are attached to the shank of the bur through either a galvanic metal bond or a sintering process and the quality and efficiency of the bur is dependent upon the efficiency of attachment of the particles to the bur head, as well as the clearance of the shavings.

Diamonds abrade tooth structure rather than cut or chip it and are therefore more efficient over a greater range of speeds and are less likely to chip or break either themselves or the tooth. They are most efficient when used against hard materials, such as enamel and porcelain, although very fine diamonds are excellent for reducing dentine to a fine finish (**Figures 9.5 and 9.6**).

Air/water spray is mandatory to enhance clearance and to control heat development, which will be greater as the particle size reduces and clearance is slowed down. Diamond burs will cut efficiently over a wide range of speeds although, logically, the annoyance factor will be least with the finer grain size if lower speeds are to be utilised.

SUMMARY



- Low annoyance factor
- Lubrication essential
- Not so fragile
- Maintains concentricity
- Various speeds

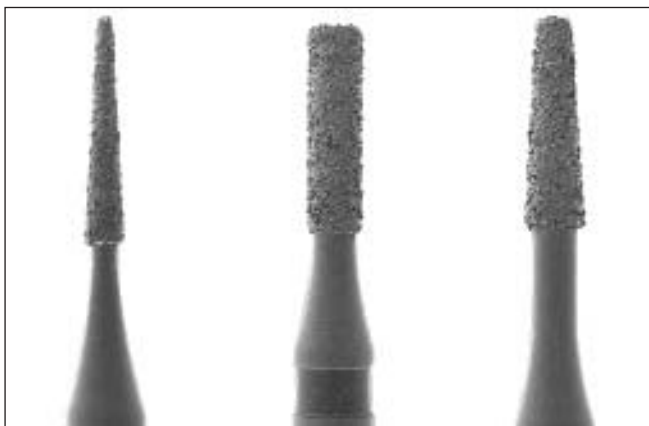


Fig. 9.5. A set of three diamond burs with the same grit size showing the different shapes available. The first is a fine tapered useful for opening fissures. The second is a cylinder for extending a cavity and the third is a tapered fissure useful for entering a proximal lesion or a tunnel.



Fig. 9.6. Two diamond burs showing the difference in grit size. On the left the grit size is 80 μ while the one on the right is 25 μ .

Size and shape of rotary cutting instruments

The original steel burs designed for slow speed handpieces were divided into shapes broadly described as follows (**Figure 9.7**):

- Flat fissure – a parallel sided cylindrical bur of varying length designed to extend a cavity along a fissure or widen a proximal box.
- Tapered fissure – similar to a flat fissure but with the sides tapered towards the tip, designed to develop retentive grooves and pin holes.
- Round – spherical burs designed to remove caries and to develop retentive pinholes.
- Inverted cone – a bur with a flat base and sides tapered towards the shank, designed to develop a flat floor in a cavity as well as undercuts adjacent to the floor for retention of plastic restorative materials.

With the advent of increased speeds these shapes have been retained but modified to a considerable extent. The original subdivision is still valid and burs and their application can still be discussed under those headings, though slightly modified. One complicating factor has been the plethora of rather bizarre bur shapes produced by manufacturers at the whim of individual operators who develop an unusual shape to fulfil one task only in tooth reduction. The result can be a vast array of burs with limited functions which

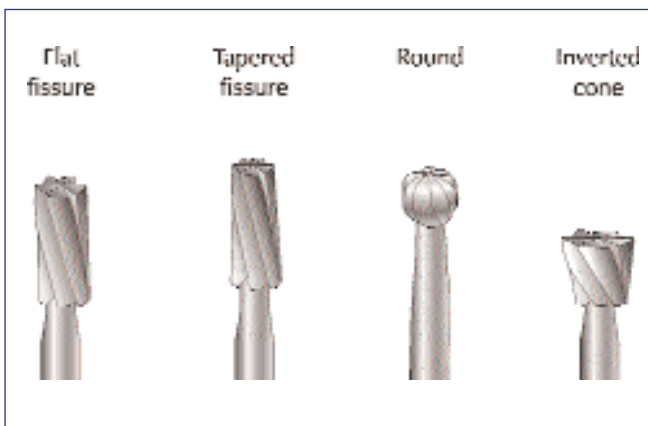


Fig. 9.7. Basic bur shapes: Shown are the four basic shapes in which burs are manufactured. However, the inverted cone is not in common use any longer because the production of undercuts for retention is not required in the presence of adhesion.

tend to clutter up bur storage and serve a very limited purpose. It is recommended that the bur shapes and sizes be strictly limited and the various shapes be used to their full extent.

Elimination of inverted cone

Simplification of bur selection is achieved by eliminating the inverted cone. The original concept behind this shape was that it should undercut sound enamel and then remove it by being withdrawn vertically through the enamel. This is no longer necessary for cavity preparation. The sharp acute angle which will be produced between the floor and the walls is difficult to restore properly with plastic restorative materials because they do not flow readily into such acute angles. Retentive features can be better developed with the other shapes.

BE AWARE



- Inverted cone burs are not useful
- Develop sharp angles that complicate placement of restorative materials

Cutting efficiency

Tooth structure can be removed at different rates depending upon the intended outcome. If it is necessary to remove a large quantity in a short time then, within the constraints of the annoyance factor, a large diameter coarse cutting diamond or tungsten carbide bur with good clearance, rotating at ultra-high speed, with copious water spray, will accomplish this. On the other hand, precise cavity outline form requires greater tactile sense leading to conservation of remaining tooth structure and better adaptation of adhesive restorative materials. This can be achieved with slower speeds and smaller burs, preferably finer diamonds.

End cutting/side cutting

It is also important to consider whether a bur will cut efficiently along the side or the end or both. Entry through intact enamel requires an end cutting bur, development of cavity outline requires side cutters. It is possible to combine the two functions in one bur. A diamond cylinder with a

flat end and a slightly rounded corner will achieve both aims rather efficiently. It will penetrate sound enamel by engaging the surface of the tooth with the bur at a slight angle and then it will extend a trench while held at right angles to the surface. A round bur will also penetrate the surface by using the side only rather than the base. However, the bur head then disappears into the cavity and is lost to sight. A logical choice of bur should be made according to the task in hand and the number of burs in a kit can then be kept to a minimum.

Visual contact with the bur head

It is important to maintain visual contact with the working head of the bur during cavity preparation, particularly if the cavity is small and in a confined position (**Figure 9.8**). The enamel on the occlusal surface of a molar will be approximately 2-3 mm thick so, if a bur with a working head less than 4 mm long is used to open a fissure, the cutting blades will disappear from view on reaching the dentine. The operator can then be disoriented resulting in deep overcutting and loss of direction. Penetration into the dentine may be over extended and the tooth may be unnecessarily weakened.

If the bur head is about 5 mm long it will be possible to maintain visual contact at all times and cavity preparation will be more conservative. A diamond cylinder of this dimension is both end cutting and side cutting and the fissure can be opened both quickly and efficiently even though

access and visibility is limited. Round or pear shaped burs are contraindicated for these tasks.

Linear surface speed

Within limits, the faster the surface of one material passes over the surface of another material the faster will be the abrasive effect and the more material will be removed. Therefore the linear surface speed

(LSS) of the rotating bur is significant. The speed can be calculated using the formula shown.

NOTE

Linear Surface Speed

$$V = \pi d n$$

d is the diameter of the bur

n is the revolutions per minute

It must be noted that the LSS of a bur will vary according to the geometry of the bur so that the tip of a tapered bur will not be travelling as fast as the butt. This means that cutting efficiency will be different at different levels of the same bur.

In general, the higher the speed the smoother the operation, in as much as, providing the bur is running concentrically, vibration will be reduced as the speed increases. However, there is a limit, because centrifugal forces take precedence at ultra-high speeds. For example, a bur with a diameter of 2 mm should not exceed 300,000 rpm or it may bend and break. Also, it is difficult to maintain concentricity in a rotating bur but, at higher speeds, this problem is somewhat overcome. The fact that only every third or fourth blade is actually working is less significant at ultra-high speeds and vibration is reduced.

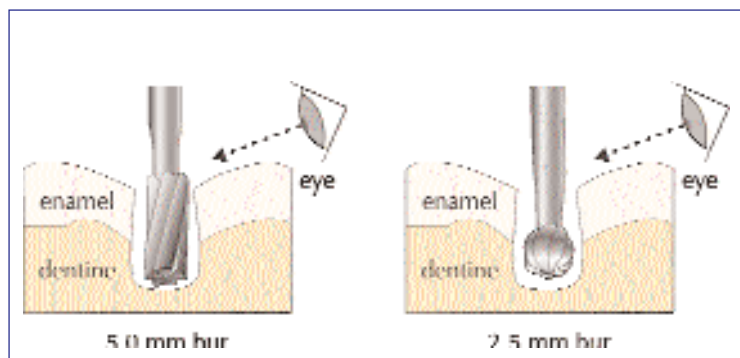


Fig. 9.8. Visual contact: It is important, wherever possible, to maintain visual contact with the working area of the bur head to minimise depth of penetration and thereby limit the amount of sound tooth fabric removed.

Application of load

Because of torque limitations with air turbines, as the speed increases the load that can be applied without the bur stalling decreases. This means that the tactile sense of the operator also decreases. It is therefore possible to remove too much tooth structure with ultra-high speed burs if visibility is less than excellent. With electric micromotors torque can remain reasonably high through an electrical feedback circuit and tactile sense remains high.

Load before stalling will also be dependent upon the linear surface speed of the bur, the

presence or absence of an adequate lubricant and the sharpness of the bur. There is a temptation to continue the life of a bur beyond its useful time, simply by applying greater load, but in so doing the temperature at the work face will be increased, there will be greater wear and tear on the handpiece bearings and the concentricity of the handpiece will degenerate more rapidly.

The exact amount of force is variable, but generally, in between 60 gm and 120 gm must be regarded as a maximum. With a given load the rate of cutting increases in relation to the speed of rotation but this is not in direct proportion. At speeds above 100,000 rpm the rate of cutting does not increase greatly and beyond 400,000 rpm the risks of losing concentricity outweigh the cutting advantages and torque will be substantially reduced.

Lubrication

A coolant applied to the tooth surface and the bur head during cutting reduces the temperature rise and increases the cutting rate by acting as a lubricant.⁴ The coolant can be air, water or a combination of the two in the form of a spray. A copious stream of water is the most effective, providing it can directly reach the operative area, but an air/water spray is adequate. It has been shown that cutting dentine with no lubricant may result in a temperature rise, at the surface of the tooth in

contact with the bur, of up to 136°C in only two seconds.⁵ Air alone will maintain a low temperature but, if used for other than short periods, may dehydrate the tooth and damage the pulp. Using an air/water spray with a water flow of 35-50 mL per minute the temperature rise can be limited to 20-30°C. Water alone is even more effective in as much as the temperature rise can be limited to 10°C with a flow of only 10 mL per minute.⁶ However, ensuring that the bur head is adequately bathed with water is not always easy in the oral cavity. For example, the tooth itself may 'shadow' a water jet from the gingival margin of a proximal box in an upper molar. The combined air/water spray is generally regarded as, clinically, the most effective.

SUMMARY



Stalling load =

- LSS of bur
- lubrication
- sharpness of bur

Increased load =

- greater wear
- increased temperature
- early eccentricity

NOTE



Optimum lubricant –
air/water spray @ 35-50mL/minute

Speed Groups

With modern handpieces, and the motive power to drive them, rotational speeds ranging from under 1,000rpm up to approximately 400,000rpm are readily available and, taking into account the above discussions, it is logical to make use of the entire range. However, considering the essential requirements of operative dentistry, it is equally logical to subdivide the stages of cavity preparation and assess the appropriate

TABLE 9.1: Colour coding for handpieces

Speed grouping	RPM	Handpiece colour	Lubricant
Low speed	500-25,000		Optional
Intermediate high speed	20,000-120,000		Mandatory
Ultra-high speed	250,000-400,000		Mandatory

Table 9.1. Colour codes used by most manufacturers to indicate the speed at which a handpiece will work best. The variations are achieved by modifications in gearing ratios.

speed for each activity. Most manufacturers colour code handpieces according to the operating speed (**Table 9.1**).

It is generally necessary to enter the lesion through the enamel. Any demineralised infected dentine can then be removed and the final cavity outline refined to receive the ultimate restorative material. Each stage of cavity preparation has its own special requirements in regard to

- bur selection – diamond, tungsten carbide, steel
- speed of cutting – ultra-high speed, intermediate high speed, low speed
- lubrication – water jet, air alone, combination air/water spray
- visibility – direct, indirect
- reduction of the annoyance factor

Lubrication with air/water spray is desirable at all speeds for temperature control and removal of cutting debris but short periods using air alone are acceptable for enhanced visibility.⁷

The speed and efficiency of a rotary cutting instrument will depend to a degree on the engi-

neering and quality of manufacture. It is also necessary to define the optimum size of a bur relative to the linear surface speed and then be clear on the load to which that bur can be subjected without producing a greater annoyance factor. The following diagram is designed to define these parameters (**Figure 9.9**).

Low speed

500-10,000 rpm – green band handpiece
1,000-25,000 rpm – blue band handpiece

Steel burs are indicated in this speed range and the use of a lubricant is optional. Visibility is better without a lubricant, but cutting is faster and cleaner using air/water spray.

Because of the negative rake angle on modern tungsten carbide burs they are relatively ‘blunt’ at this speed. Diamond burs are designed to cut hard tooth structure and are not effective in removing soft caries.

Diameter of steel burs can range from 3.0 mm down to 0.5 mm and the smaller the bur the less the annoyance factor. The size should be selected to fit the task in hand. Tasks include removal of caries and development of retentive designs, placement of pins, grooves and ditches, as well as all stages of polishing to a final finish.

Intermediate high speed

20,000-80,000 rpm – red band handpiece
20,000-120,000 rpm – orange band handpiece

Diamond burs with a medium to fine grit are the most efficient in this range and use of a lubricant is mandatory. Air alone for very short periods is acceptable

because it will enhance visibility but cutting will be faster under air/water spray. Minimal cavities can be opened rapidly and effectively in this speed range using an 85 μ diamond. Final cavity outline, with walls suitable for adhesive restorative materials, can be achieved using a 25 μ diamond without

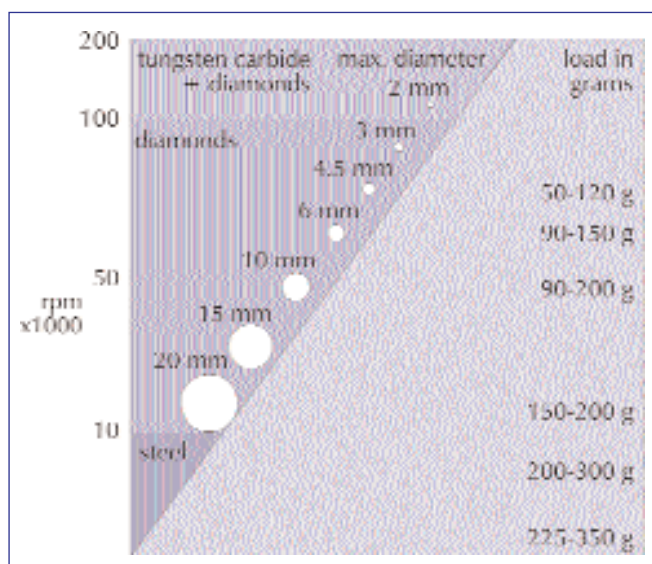


Fig. 9.9. Bur selection chart: The chart shows the maximum diameter of a bur relative to the speed available as well as the maximum load which can be applied for optimum efficiency. Note that diamonds have a wide range of speeds available. Tungsten carbides are efficient only above 100,000 rpm. On the other hand, steel burs should only be used below 10,000 rpm. Water spray must be used as a lubricant at speeds above 10,000 rpm.

Annoyance Factor = 8



High amplitude, low frequency, long time

Annoyance Factor = 3



Low amplitude, low frequency, shorter time

overextension and undue loss of tooth structure.

Tungsten carbide burs tend to ‘chatter’ at this speed and may cause microcracks in enamel and an increased annoyance factor. Steel burs will not cut at these speeds and the annoyance factor would be far too high. Diameter of the burs to be used in this group cover a wide range but, logically, the smaller the bur the lower the annoyance factor and the more conservative the cavity design.

There is a very fine tactile sense available within this speed range and the risk of overcutting is minimal. Therefore it should be used in the development of small cavities as well as to refine final

cavity outline for all restorations. It is also useful for initial contouring of most restorations leading to a final polish. It is the correct speed group for refining the occlusion (**Figures 9.10-9.12**).

Ultra-high speed

250,000-400,000 rpm – air turbine only

Tungsten carbide burs are at their most efficient in this range but diamond burs are also very useful.⁸ Lubrication is mandatory with a copious water jet being the most efficient for temperature control.

Tungsten carbide burs cut dentine very smoothly providing they are not chipped or eccentric. They can also develop a fine margin in enamel although it must be noted that they cut more smoothly along the margin where the rotating bur enters the cavity.

The opposite, exit margin, is likely to chip more readily. As tungsten carbide burs are essentially side cutting they should not be used to enter through healthy enamel into a new lesion. They cut old metal restorations well.

Diamonds are more versatile and a coarse end cutting diamond is the preferred bur to enter a new lesion or remove bulk enamel, even though both entry and exit margins will be relatively rough, depending upon the grit size being used.

Annoyance Factor = 2



Low amplitude, low frequency, shortest time

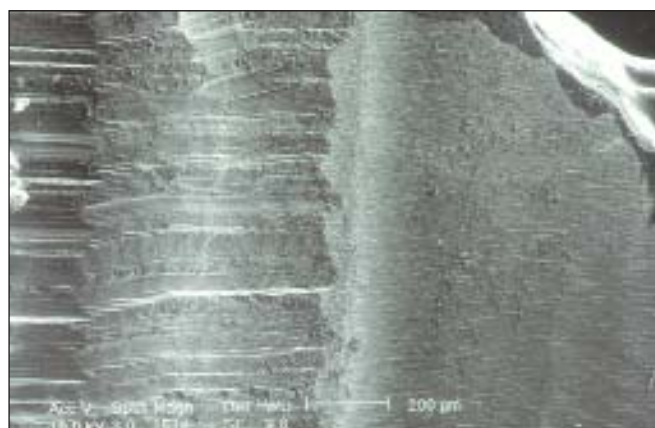


Fig. 9.10. An SEM illustration to show the difference in the finish of the enamel margin of a cavity following the use of a diamond with 80 μ grit (on the left) and a 25 μ grit on the right.



Fig. 9.11. The 80 μ diamond is being used to open a cavity and to remove an old amalgam restoration. The resultant margin will be rough (see **Figure 9.10**) and not suitable for placement of an adhesive restorative material.



Fig. 9.12. The 25 μ diamond is being used rapidly and lightly to polish the margins to an acceptable finish for use of an adhesive restorative material (see **Figure 9.10**).

The diameter of burs for this speed group range from 2 mm down.

Initial entry to most lesions, and the removal of old restorations, is achieved best in this speed range. The tactile sense is minimal and over cutting is possible if visibility is limited. Use these speeds for gross reduction of tooth structure only and then step back to intermediate high speed to refine the cavity (**Figure 9.13**).

Air Abrasion Techniques

Description

While rotary instruments are still used for the majority of restorative procedures, in the past decade there has been a dramatic expansion in the utilization of methods which can augment, or indeed replace, rotary instruments for caries removal and cavity preparation. Alternative methods of air abrasion, pulsed erbium lasers and chemo-mechanical caries removal, provide the clinician with the ability to work more conservatively and in different quadrants at the same appointment, with a greatly reduced need for local anaesthesia. Overall, this facilitates a minimalist philosophy and reduces stress for both patient and operator.

Air abrasion, also called kinetic cavity prepara-

tion, particle beam or kinetic abrasion technology, uses the kinetic energy of microfine (20-50 μ diameter) particles of alumina (aluminium oxide – Al_2O_3) in a high pressure air stream to remove tooth structure by brittle micro-fracture.^{9,10} The failure of brittle materials during air abrasion occurs by a process of crack creation, extension, and erosion. When the abrasive particles impact such a surface, the depression grows, and radial and lateral cracks are generated in the area. The cracks ultimately join together to isolate and remove a piece of the material. The alumina particles are delivered intraorally using a handpiece with contra-angle or sickle configuration, fitted with a nozzle through which the particle beam is directed on to the tooth.¹¹ Alternative powders that can be used for air abrasion include sodium bicarbonate, urea, and dolomite.

History

The technique of air abrasion was invented in 1943 by Robert Black (a cousin of G. V. Black), but was not pursued with vigour as a technology for cavity preparation because of the advent of the air turbine. It re-emerged in the 1990s, largely due to the work of J. Tim Rainey, a dentist in Texas who was a protégé of Robert Black. The technique attracted interest because it simplifies the preparation of very conservative cavities and reduces the need for local anaesthesia in many patients.

It should be regarded as an adjunct to, but not a



Fig. 9.13. Standard bur kit: The burs in sequence from the left are as follows – #200 diamond 80 μ , #200 diamond 25 μ , #168 diamond 80 μ , #168 diamond 25 μ , #156 diamond 80 μ , #156 diamond 25 μ , #008 mild steel round, #012 mild steel round, #016 mild steel round, #168 mild steel tapered fissure, #140 tungsten carbide, #168 tungsten carbide.

universal replacement for rotary instruments. The main indications include the preparation of minimal access through enamel for occlusal lesions on posterior teeth, as well as limited debridement of dentine lesions without local anaesthesia. However, it must be noted that large areas of carious infected dentine are not hard and brittle so it has limited ability to undergo brittle fracture. Therefore, these areas cannot be cut efficiently with air abrasion and alternate techniques are recommended. The traditional hand excavators are still of value but are not particularly accurate. Slowly rotating round burs are the usual method but now there is a chemo-mechanical technique which has a number of advantages for removal of grossly softened tooth structure.

Air abrasion units are also useful for microetching of metallic and ceramic crowns prior to cementation or repair, and for removal of extrinsic stains from teeth using low particle velocity and softer abrasives to avoid enamel or root surface damage.

Available equipment

There are now several suppliers of air abrasion technology and a range of equipment is available. Quite complex units with advanced features such as supersonic handpieces, which increase particle velocity and thus cutting speed, are available. Pulsing of the particle stream through the cyclical operation of valves is possible and air desiccation

and particle agitation can be incorporated to prevent clumping and blockages of the powder. Microprocessor control of air pressure is available and the particle parameters can be varied. With more complex equipment, a remote control unit, small enough to fit into the palm of a hand, provides control of the basic parameters of the instrument, allowing fingertip control without having to move away from the patient. It is also possible to integrate the foot control with that of the dental unit so one foot control can be universal.

Clinical application

There is a distinct learning curve required for the use of air abrasion, since the cutting action created by the particle beam occurs ahead of the handpiece and there will be no tactile feedback at all. Typically, a rotary cutting instrument is side cutting and there is tactile sense at all times. In the absence of this 'feel' there is a tendency for over-cutting of sound tooth structure which will only be reduced with experience and attention to detail.

NOTE

Advantages include

- very low annoyance factor
- reduced need for anaesthesia
- reasonably wide application

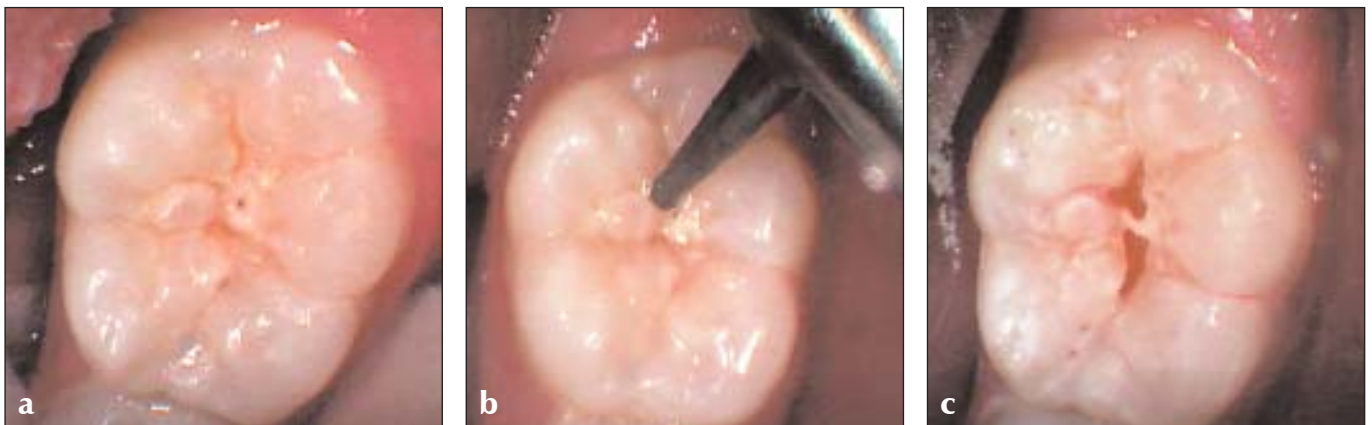


Fig. 9.14. Air abrasion used for a minimally invasive approach to a small fissure caries lesion. **a)** The initial situation. **b)** Air abrasion nozzle in place. **c)** Final preparation before restoration.

The energy delivered by the abrasive particles is directly related to the airflow and the particle size.¹² Doubling the particle size from 27-50 μ multiplies the mass, and thus the kinetic energy, by a factor of eight. While this will accelerate tooth removal it will lead to more discomfort for the patient, particularly during the cutting of dentine, although the relationship between energy and discomfort is not linear. In addition, there may be an increase in dehydration which is often interpreted as a cold sensation. There is, therefore, an inherent trade-off between increasing cutting power (energy) and the level of patient comfort i.e. the annoyance factor (**Figures 9.14 and 9.15**).

In practice, this means that a range of particle sizes need to be employed such as

- 27 μ for removing caries
- 34 μ for cutting enamel
- 50 μ for extraoral/laboratory microblasting

Note also that air abrasion will not effectively remove the smear layer from dentine, but rather creates one. Acid etching should be used to remove the smear layer prior to bonding of resins to dentine or enamel.

BE AWARE



Air abrasion is contraindicated for

- removal of grossly carious dentine
- removal of smear layer

Clinical efficiency

Factors affecting the clinical efficiency of air abrasion, that is, removal of tooth structure, include

- nature of the tooth surface – carious enamel cuts preferentially to sound enamel but sound dentine is cut more readily than carious dentine
- moisture level – dry dentine cuts more readily than wet dentine
- nozzle size (fine, ultrafine)

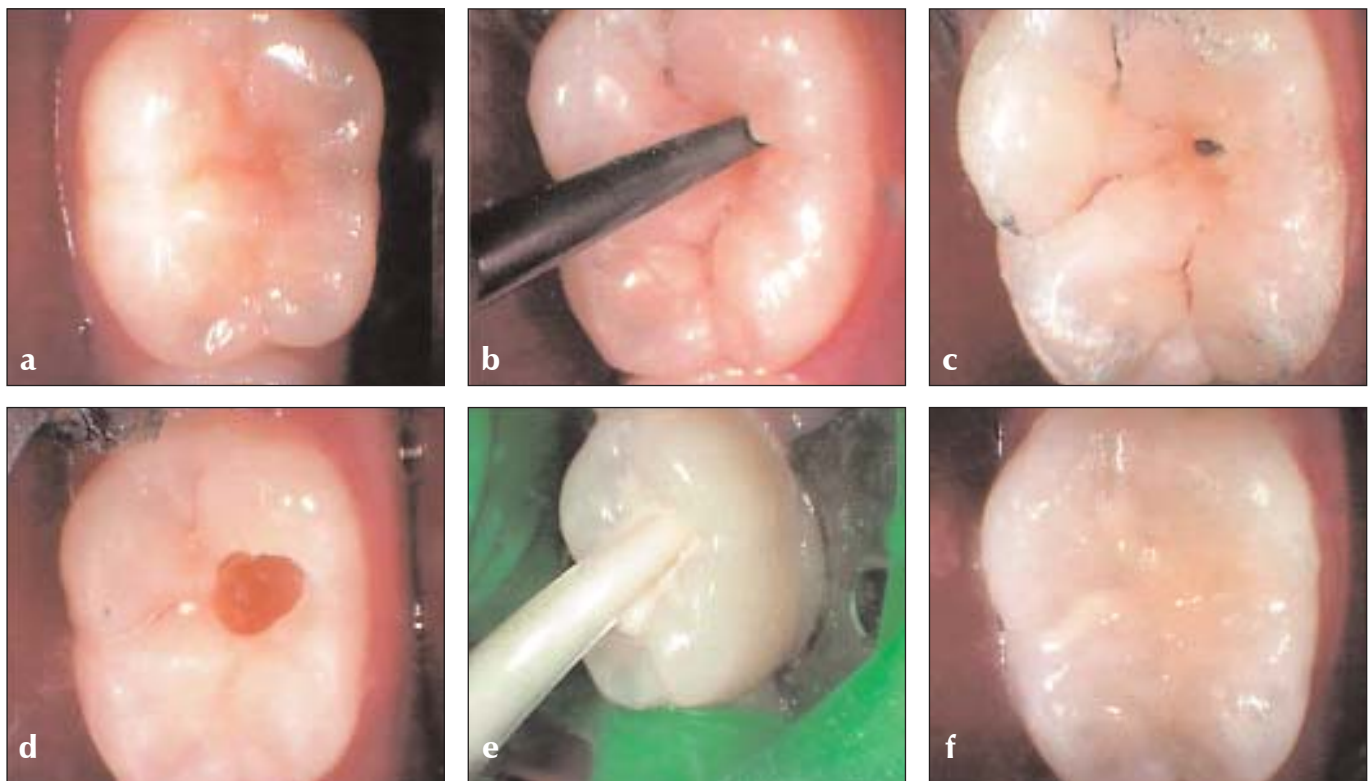


Fig. 9.15. Air abrasion used for a minimally invasive approach to an occult ('hidden') lesion detected with laser fluorescence using the DiagnoDENT™. **a)** The initial situation showing an apparently intact fissure system. **b)** Air abrasion nozzle in place. **c)** Access gained through the occlusal enamel into the carious lesion. **d)** Preparation after caries removal using rotary instruments. **e)** Placement of glass ionomer. **f)** Final restoration.

- air pressure (typically 45-80 psi, but can be as high as 120 psi)
- particle flow rate (usually 2 g/minute but may be up to 8 g/minute)
- particle size and velocity (e.g. 27, 34, 50 or 100 μ particle sizes)
- nozzle-target distance (typically 0.8-1.0 mm minimum)
- nozzle-target angle (45, 66, 90 degrees)
- speed of movement of the handpiece over the tooth surface
- pulse frequency (mark-space interval or dwell) – only for pulsed instruments.

Air abrasion has a variety of applications including entry to a caries lesion, removal of carious enamel and dentine, stains, calculus, organic plugs, composite resin restorations, and fissure sealants.¹³ The cutting action comes from firing alumina particles against the target surface. This means it delivers an end cutting action. When the nozzle is close to the teeth, the particle stream is narrow and a small diameter straight cut is achieved. As the nozzle to target distance increases, the beam becomes wider and more conical and thus a shallower and wider pattern of removal occurs.

BE AWARE



- Nozzle close to tooth surface = narrow cut
- Nozzle away from tooth = wider cut

Using very high air pressures, ranging from 7–12 bar, increases the cutting speed of the particle stream, but increases dramatically the level of discomfort for the patient as well as the theoretical risk of injury from air emphysema. A further disadvantage of increased air pressure is that, as the abrasive air fluid exhausts from the instrument, there is an immediate drop in pressure. This will cause a substantial decrease in air temperature that can lead to extreme discomfort for the patient.

Scanning electron microscope investigations reveal rounded cavosurface margins and internal line angles.¹⁴ The surfaces are microscopically rough and the dental tubules are occluded but surface microcracking is minimal or absent. In contrast, cavities prepared using conventional high speed tungsten carbide or diamond burs show sharp line angles, chipping of the cavosurface margins, and striated internal surfaces, with surface microcracking to a depth greater than 50 μ (see **Figure 9.10**).

The effect on the smear layer is more complex. Short exposures to air abrasion of less than five seconds may remove some of the smear layer, whereas longer exposures lead to obstruction of

BE AWARE



- Increased air pressure –
- increased patient discomfort
 - increased risk of emphysema
- Lower temperature –
- greater discomfort

TABLE 9.2: Typical operating features of air abrasion equipment

Application	Removal of organic plug	Removal of composite resin	Roughen porcelain before repair	Removal of surface stains from enamel
Air pressure	2.8-4.3 bar	4.3 bar	4.3 bar	1.4-2.8 bar
Powder size	27.5 μ alumina	27.5 μ alumina	27.5 μ alumina	27.5 μ sodium bicarbonate
Powder flow rate	3-5 g/min	3-5 g/min	5 g/min	2-3 g/min
Nozzle diameter	280 μ	356 μ	356 μ	457 μ
Mode	Micro-pulse	Micro-pulse	Continuous	Continuous
Time per tooth	5-10 sec	As required	5-20 sec	As required

(Based on Elrod et al. 1999, using a Kreativ™ Mach 5.0 air abrasion unit)

dentinal tubule openings, probably from abrasive powder residues.

Recommendations for use

The primary indications for the intraoral application of air abrasion are as follows:

- conventional and ultra conservative cavity preparations in enamel and dentine such as occlusal, cervical and proximal cavities as well as tunnel preparations – that is Site 1, Size 1 & 2 and Site 2, Size 1 & 2 minimal lesions
- preparation and cleaning of cavity surfaces prior to placement of adhesive restorations
- removal of old composite resin restorations and sealants
- removal of the surface of anterior composite resin restorations for refacing
- removal of superficial enamel discolourations

There are also a number of extraoral indications, particularly in the laboratory, where it can be very useful:

- extraoral microetching of restorations
- divesting casting investments and enhancing the mechanical retention of porcelain or cast metal crowns.
- microblasting of cast metal crowns with 50 μ alumina as an alternative to acid pickling
- removing residues of cement from crowns and bridges prior to recementation
- roughening the tissue surface of dentures to increase the adhesion of liners and relines

Air abrasion is not suitable for

- removal of grossly carious dentine
- preparation of large cavities
- removal of caries in subgingival sites
- cutting preparations for full crowns, inlays or onlays
- preparing precise retention features in cavity preparations such as slots, grooves or ditches
- removal of crowns, bridges, or porcelain restorations

Occupational hazards

Alumina, the most common powder material used in air abrasion, is an insoluble white odourless nonflammable inert powder. Grey alumina is composed of flakes of alumina, while white alumina contains globules of alumina mixed with titanium dioxide.

The use of alumina raises three occupational health and safety issues:

- it must not be used in a dental practice where operatory staff suffer from asthma, reactive airways disease, or other respiratory diseases, because of the risk of exacerbation from fine alumina dust, classified as a 'nuisance dust',
- in the absence of rubber dam, the patient must tolerate alumina powder buildup in the mouth. Wet gauze squares placed on the buccal mucosa and tongue can help contain these residues. However, alumina is not a chemical irritant and is not absorbed through mucous membranes or the skin,
- dust problems in the treatment area tend to



Fig. 9.16. Prototype of a modified air abrasion hand-piece showing the powder/water slurry being emitted from the tip. The width of the abrasive stream is slightly wider than with powder only.



Fig. 9.17. The operative opening of the modified air abrasive hand-piece showing the powder aperture in the centre and the water aperture surrounding that opening.

increase as the air abrasion dust adds to other environmental sources of dust.

Both conventional intraoral high velocity evacuation and additional extraoral high performance vacuum systems are required. The extra-oral system removes the small amount of dust, approximately 10%, which escapes the intraoral evacuation system, and also provides filtration of the entire operatory air. In terms of personal protective equipment, a mask is considered adequate protection but both staff and patient must wear eye protection because alumina particles may cause mechanical injury to eyes.

Forthcoming developments

Research is continuing at present into the advantages of providing a parallel water stream concurrently with the abrasive powder stream. Apparently the water absorbs some of the energy of the exiting air stream and this can reduce the lateral damage that can be done by the abrasive powder particles. It seems that, due to slurry formation, the abrasive effect is greatly enhanced and there will be a reduction in the dehydration effect. There would appear to be some advantages including

- improved efficiency of removal of soft demineralised dentine
- reduced patient sensitivity from air pressure drop effect

- incorporation of warm water reduces sensitivity still further
- reduction of nuisance dust problems

However, it must be noted that the cutting effect appears to be widened in the presence of the parallel water spray thus negating, to a degree, the efficiency of air abrasion for minimal preparation techniques. Further developments envisage the production of a handpiece capable of both methods of delivery thus enhancing the versatility of this technique (Figures 9.16 and 9.17).

Pulsed Erbium Lasers (Er:YAG and Er,Cr:YSGG)

History

Laser technology for caries removal and cavity preparation is currently at a high state of refinement, having had several decades of development up to the present time. The process of effective cutting of tooth structure using pulsed Erbium:YAG laser energy in combination with a water mist spray was first characterized in detail by Raimund Hibst at the University of Ulm in Germany in the mid 1980s.^{15,16} Following this, a range of clinical trials was undertaken in which

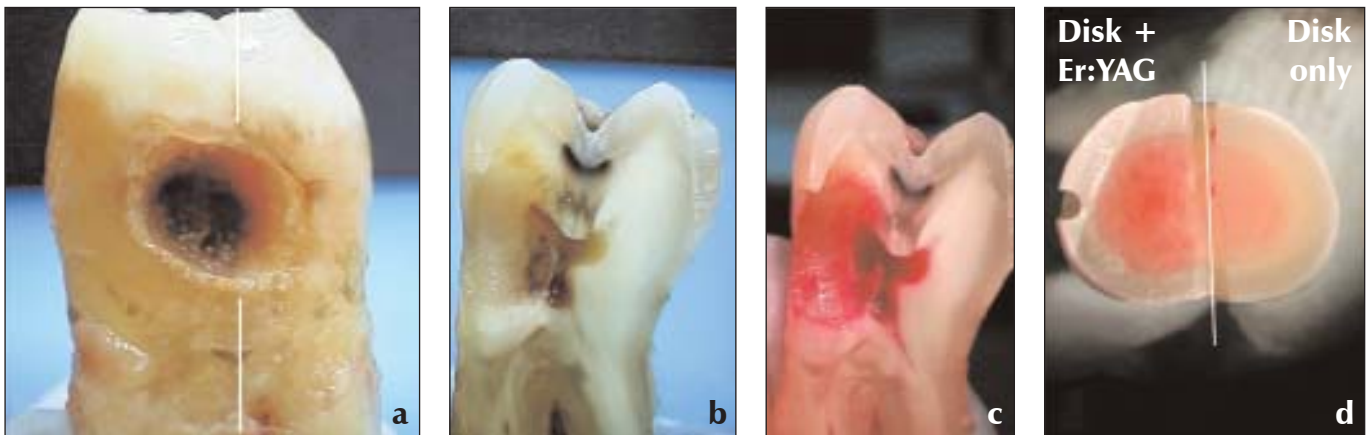


Fig. 9.18. False positive staining with acid fast red caries detector dye. **a)** Extracted tooth prior to caries removal using an Er:YAG laser. After caries removal, the tooth was sectioned along the plane shown. **b)** After caries removal. **c)** Dye applied to the cavity gives a consistently strong false positive stain because of the lack of smear layer. **d)** Demonstration of the same effect on an extracted sound premolar tooth sectioned horizontally with a disk. The left side has then been laser-treated and the right side untreated, before dye was applied. False positive staining is present on the laser-treated dentine surface.

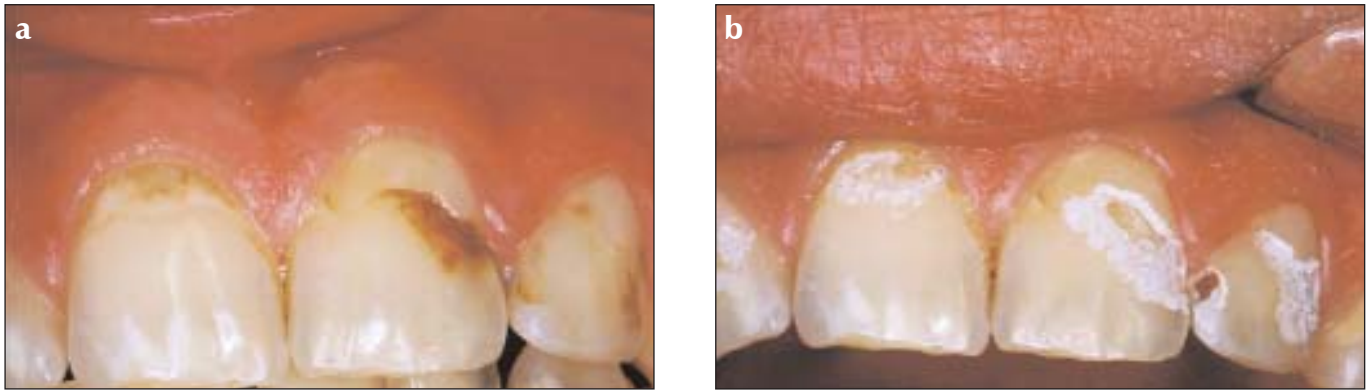


Fig. 9.19. Restorative procedures using the Er:YAG laser, in anxious dental patients, without local anesthesia. The Er:YAG laser was used with a non-contact hand-piece.

a) Pre-operative appearance of a 22-year-old male with salivary dysfunction, and associated cervical and approximal caries.

b) Areas of caries and defective resin composite have been removed. The intense white appearance of the margins is typical of laser etching.

c) The restored teeth immediately post-operatively. The etched appearance of the margins disappears once bonding resin has been placed.

d) 30-year-old female patient with areas of hypoplastic enamel.

e) The enamel surface has been 'peeled' using a series of pulses from the laser.

f) The two areas have been restored with resin composite.

g) 65-year-old female patient undergoing anti-cancer chemotherapy, with recurrent caries at the margins of several restorations.

h) Areas of caries and undermined resin composite have been removed.

i) The cavity preparations have been restored.

From the *Australian Dental Journal* 2003; 48:151. Used with permission.



teeth, destined for extraction for orthodontic purposes, were treated with the Erbium:YAG laser and conventional restorations placed. The teeth were then removed and the histology of the dental pulp examined. The studies showed no histological or biochemical injury to the dental pulp arising from the laser cutting procedure. Moreover, the patients were routinely treated without local anaesthesia, and experienced little or no discomfort during cavity preparation (**Figures 9.18 and 9.19**).

At this time a range of lasers have been tested for cavity preparation purposes and it seems the solid state laser systems based on erbium (Er:YAG and Er,Cr:YSGG) are the most effective. Detailed studies of these two systems by Hibst and others suggest that the mechanism for enamel removal is basically an explosive subsurface expansion of interstitially trapped water, with rapid ejection of apatite crystals in the opposite direction from the incoming laser beam. As carious tooth structure contains even more water, the use of lasers which absorb into water is even more effective in the removal of demineralised dentine.¹⁷

Alternative systems have been explored but not all have proven effective. Excited dimer lasers, operating within the ultraviolet portion of the spectrum, can ablate enamel by photo-ablation, a non-thermal (cold) process. This involves breaking chemical bonds within apatite mineral and enamel proteins utilising the high energy of the photons. However, this process is rather slow in as much as it takes 3-5 minutes to ablate 1 mm³ and is therefore unsuitable for clinical use. Intensive research over the past three decades on other non-erbium laser-based cavity preparation systems, including super-pulsed CO₂, Ho:YAG, Ho:YSGG, Nd:YAG, Nd:NLF, Nd:YAP, and GaAs lasers, has not led to clinical application due to concerns about effectiveness, safety, or cost.

Modus operandi of lasers

At this point the most common laser systems used for cavity preparation rely on the intense absorption bands within tooth structure in the middle and far infrared regions. Tooth enamel is a mixture of apatites, including hydroxyapatite (HA;

Ca₁₀(PO₄)₆(OH)₂), fluorapatite (Ca₁₀(PO₄)₆F₂) and carbonated apatite (Ca₁₀(PO₄)₆CO₃). The major absorbing components in enamel are hydroxyl (2800 nm), water (2940 nm), carbonate (7000 nm), and phosphate (9000-11000 nm). Regardless of the balance of these components, water is the major absorbing agent from the standpoint of ablation of both sound and carious tooth structure.¹⁸

The major absorption areas for water are within the middle part of the infrared spectrum, specifically in the region near 3000 nm wavelength. A wavelength of 2940 nm will match exactly the maximal absorption of water, and thus provide effective ablation of tooth structure and this corresponds with that of the Erbium:YAG laser. Lasers which operate at more or less than 2940 nm absorb less well in water and thus tend to show less rapid ablation of tooth structure when measured on a weight or volumetric basis. The same is true for ablation of bone.

Tooth structure is naturally 12% by weight of water, and one third of this is bound into the chemical structure of the tooth as a monolayer of molecules surrounding each crystal of apatite mineral within both enamel and dentine (**Chapter 1**). The remaining two thirds is found in pores in the enamel and dentine and this can be removed by dehydration. A laser which can transfer the water into steam by rapid heating can therefore explode tooth structure and eject the crystallites. The evaporation of the water creates a cooling effect and this mitigates against the inherently thermal nature of the process. In fact, Erbium-based laser systems can achieve effective ablation at temperatures well below the melting and vapourisation temperature of enamel.^{19,20}

Since carious enamel and dentine has an increased water content it will be ablated selectively at lower pulse energies with middle infrared lasers. The absorption rates of various lasers are given in **Table 9.3**. With the middle and far infrared lasers, there are high rates of absorption (>100 cm⁻¹). This means the energy is absorbed within the outer 100 μ of enamel and is converted efficiently into heating the water. The Er:YAG, Er:YSGG and Er,Cr:YSGG lasers operate at wavelengths of 2940, 2790 and 2780 nm, respectively. Absorption in water of the Er:YAG laser is

13,000 cm^{-1} and this is much higher than that of the Er:YSGG at 7000 cm^{-1} as well as Er,Cr:YSGG at 4000 cm^{-1} . Since all three lasers rely on water-based absorption for cutting enamel and dentine, the efficiency of ablation in terms of volume and mass removal of tooth structure is greatest for the Er:YAG laser. These lasers are used with a water mist spray to ensure both effective energy coupling of the applied laser energy as well as cooling between pulses.

NOTE



Water and lasers

- Water is essential
- Converted to steam and ‘explodes’ the enamel
- Maintains lower temperature
- More water in carious dentine, therefore more effective

Clinical application

To deliver pulsed Erbium laser energy to the tooth, a variety of handpieces have been designed including straight, contra angle and curved configurations to suit a variety of clinical situations. Most are noncontact handpieces and use a sapphire window to deliver the energy onto the tooth surface. The window also transmits a visible red light beam, to allow the clinician to target the area of interest. An alternative is to use a short curved fiber or a straight sapphire tip which can improve access to deep proximal preparations and tunnel preparations. However, the use of longer fibers and tips of this type requires modification in the

settings, since they show greater energy loss than the relatively thin sapphire window on a noncontact handpiece.

Sound effects

A variety of photo-acoustic effects occur during cavity preparation, caused by the volume expansion of rapidly heated water, and these have clinical relevance. The pressure increase is influenced by several factors including the speed of sound in the tissue, tissue density, and pulse duration. Because of ejection of material from the tooth and ablative recoil, there is little amplitude (loudness) until the ablation threshold is reached. Above the ablation threshold, the loudness increases as active ejection occurs. For this reason, erbium-based laser systems produce a characteristic popping sound when the laser is operating on dental hard tissues above the ablation threshold. Both the pitch and resonance of this sound relates to the propagation of an acoustic shock wave within the tooth, and varies according to the presence or absence of caries. These sounds assist the user in determining that caries removal is complete.

NOTE



Healthy tooth structure produces ‘click’ sound

- high pitch
- rapid attack/decay

Carious tooth structure transmits a loud ‘thud’ sound

- low pitch
- slow attack/decay

TABLE 9.3: Absorption coefficients of enamel

Spectral region	Wavelength	Laser types	Absorption coefficient (cm^{-1})	Relative performance for enamel ablation
Ultraviolet	240-300 nm	ArF, KrF, XeCl	10	+
Visible	400-700 nm	Ar, KTP, He-Ne	<1	-
Near infrared	830 nm	GaAs	<1	-
Near infrared	1,064 nm	Nd:YAG	<1	+
Middle infrared	2,780 nm	Er,Cr:YSGG	450	++
Middle infrared	2,940 nm	Er:YAG	770	+++
Far infrared	9,300-10,600 nm	CO ₂	825	+++

Analgesic effect

Generally, patients undergoing cavity preparation with erbium lasers do not need local anaesthesia. Large scale clinical trials report that only 2-5% of patients will request local anaesthesia although many patients experience slight, intermittent sensations of cold in their teeth, probably caused by the cooling effects of water evaporation during laser pulses. Some report subtle 'earthquake' sensations within the pulp which, while unusual, are not perceived as unpleasant. Many patients have no sensation whatsoever which suggests that the annoyance factor is very low. It also raises the intriguing possibility that there is an analgesic effect created by the laser.

There have been a number of animal and human studies conducted that tend to confirm analgesic effects created by erbium lasers. These appear to operate at the level of dental pulp, with an increase in pain threshold of incisor teeth after irradiation. The animal studies are particularly informative, since they allow direct measurement of intradental nerve electrical activity after standardised mechanical stimuli have been applied, and they remove the possibility of placebo effect. Suppressive effects on nerve firing have been shown, leading to an analgesic effect with a duration of 10-15 minutes. The effect can be induced again by further lasing. The suggested mechanisms include altered haemo-dynamics and a depressed nerve excitability by blockade of the Na/K pump on intra-dental nerves. Erbium laser energy appears to selectively block depolarisation of A delta fibers leading to gate control. It may also exert an effect on C fibers within the dental pulp.

Deliberate 'pre-emptive' analgesia can be created by applying subablative pulses to the teeth or their root apices. At this level the energy penetrates well through both soft tissues and teeth, through to the pulp, without causing thermal injury. Typical parameters are 60-120 mJ/pulse, at a frequency between 10-30 Hz, applied for 30 seconds, with the optimal effect at about 20 Hz. The use of the appropriate frequency may relate to membrane bioresonance, a process through which oscillations from the laser pulses, created in the nerve cell membrane, temporarily disrupt

the action of the Na/K pump. The development of this analgesic effect can be significant for patient comfort allowing minimal intervention treatment in several teeth, in one appointment, without the need for local anaesthesia.

SUMMARY



Advantages

- Reduced need for anesthesia
- Low annoyance factor
- Cavity surface suitable for adhesive restorations

Disadvantages

- Lack of tactile sense
- Slower removal of tooth structure

Effect on bonding

Preparation of teeth using erbium lasers creates a macroroughening effect, and this can be used to increase the retention of adhesive resin materials, such as bonding agents. A range of studies have examined the efficiency of laser etching versus conventional acid etching, and most of these show little difference in terms of bond strengths, despite the fact that the morphologies created on the tooth structure are not identical. Furthermore, clinical studies have shown that the integrity of a margin created with the laser is identical to that of a high speed turbine. There has been no difference reported in microleakage, marginal discolouration, or other restorative parameters over time.²¹

Further research

In addition to the above mentioned 'auditory guide' to caries removal, selective ablation can also be employed. Other means of control include real time analysis of fluorescent light emitted by bacterial products (quantitative light fluorescence), or by light emitted by plasma formation (sparking) if no water spray is used (laser-induced breakdown spectroscopy). Both methods are currently being explored, with the intention of providing a reliable guide to the clinician of the presence of residual caries in a cavity preparation. This would be of great importance with tunnel preparations, where visibility is constrained.

Caries detecting dyes are unsuitable for use with lasers since these methods do not create a smear layer, but rather leave a highly energized (or charged) dentine surface (Figure 9.18).

Chemo-mechanical Caries Removal (Carisolv™)

Introduction

Chemo-mechanical caries removal is a minimally invasive method for the removal of advanced dentine caries during cavity preparation. Specifically, the method targets the outer layer, generally referred to as the infected or necrotic zone of the caries lesion. This most superficial part of the lesion contains collagen with broken crosslinks, which is susceptible to complete proteolysis by the chemical action of sodium hypochlorite and chloramines. Because of breakdown of the collagen and loss of apatite minerals, infected carious dentine is soft and insensitive. It contains high levels of bacteria, such as *Lactobacillus* species and *Actinomyces odontolyticus*, which are susceptible to inactivation or destruction through the combined action of a high pH and the release of chlorine. Both sodium hypochlorite and chloramines exert a rapid antibacterial effect on caries pathogens.

History

The current embodiment of the chemo-mechanical caries removal method was developed by Dan Ericsson in Sweden in the early 1990s,²² and carries the trade name Carisolv™. It is a water-based gel containing

- 0.5 % sodium hypochlorite
- amino acids leucine, lysine, and glutamic acid
- sodium chloride to make the solution isotonic
- sodium hydroxide to make the solution pH 11
- carboxymethylcellulose

The inclusion of carboxymethylcellulose as a thickening agent makes the gel easy to place in the cavity, and allows surface tension effects to retain the gel in a cavity against the forces of grav-

ity. The clinical technique requires access to the lesion followed by application of the gel onto the floor and walls of the cavity. The dentine will then soften and can be removed using noncutting custom designed hand instruments or ultralow speed rotary instruments (Figure 9.20). Five different instruments, with ten different tips, are available for caries removal (Figure 9.21). These instruments have a sharp edge but a blunt cutting angle, to provide depth control and give a whisking rather than penetrating action.

In some variations, a coloured dye is included in the gel to highlight the changes in opacity resulting from the breakdown of dentine collagen proteins. Active breakdown of the proteins releases peptides into the gel, leading to the development of opacity in the solution. At the point where fresh liquid no longer becomes opaque, breakdown of the carious dentine collagens has been fully completed.

Modus operandi

The principle of operation of the gel involves proteolytic breakdown of dentine collagen proteins in carious dentine by the chloramines, without accompanying demineralisation.²³ Despite its effects on carious dentine and the bacteria involved, the gel does not damage the oral mucosa. The inclusion of the three amino acids with different

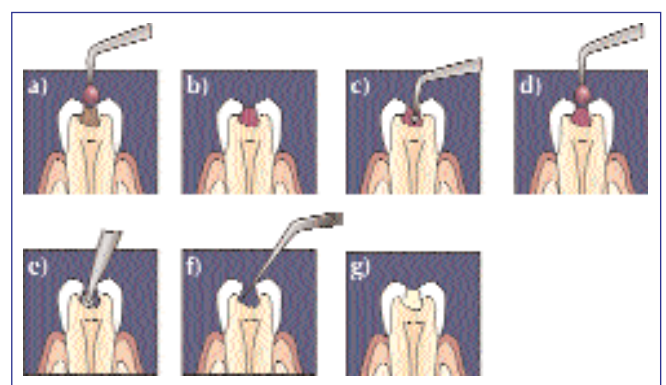


Fig. 9.20. Technique for chemo-mechanical caries removal. **a)** Gel is transferred to the lesion. **b)** The gel is allowed to penetrate the infected carious dentine. **c)** Hand whisk instruments are used to remove increments of the softened carious dentine. **d)** Further amounts of gel are replaced and the process repeated as necessary. **e)** The cavity is blotted with a moist cotton pellet. **f)** The preparation is inspected. **g)** An adhesive restoration is placed. Illustration courtesy of Mediteam, Sweden.

charges at pH 11 makes the action of the sodium hypochlorite and chloramines specific for denatured collagen. Normal collagens in sound dentine and oral soft tissues are not affected.

The clinical technique involves gaining access by a conventional technique followed by applying the gel onto the carious infected dentine.²⁴ The denatured dentine can then be removed in a step-wise incremental fashion. The gel soaks into the tooth structure and fresh gel needs to be placed at intervals to maintain the proteolytic reaction. After 'whisking away' the softened carious dentine, additional gel is applied, and the procedure repeated until no more dentine can be removed and the surface is sound as judged by normal clinical criteria. Removal of softened dentine is generally accomplished using specially designed star shaped hand instruments with a whisking action. There are also specially designed burs for use in an ultralow speed (450 rpm) handpiece, powered by a portable control unit with a microprocessor that controls torque to 12 Nm (Powerdrive). The bur rotates in both directions, with different cutting edges coming into play, giving slow or rapid removal as desired. Alternatively, a pulsed Er:YAG laser may be effective.

Treatment of the infected layer with the gel leaves a surface that is free of bacteria but may be still mildly demineralised. However, in the presence of a bioactive restorative material it is capable of remineralisation. Both the chemical and the mechanical part of the treatment play an impor-

tant part in achieving this. Caries detecting dyes will be of no value since there is no smear layer, and nonspecific staining of dentine will occur. The surface topography of the remaining healthy dentine is macroscopically rougher than that seen after conventional caries removal with rotary instruments, but this is not clinically relevant.

SUMMARY



Advantages

- Reduced need for anesthesia
- Low annoyance factor
- Removes totally denatured dentine only
- Preserves healthy tooth structure

Disadvantages

- Takes more time
- Limited to use in large cavities
- Removes infected dentine only
- Will not complete the entire cavity design

Benefits of this method include no damage to sound tooth structure, an absence of a smear layer, and reduced pulpal irritation and pain compared with conventional mechanical methods. Long term clinical studies have not found significant differences in the treatment outcomes between teeth treated with the gel and those treated conventionally. Preparation with rotary instruments or by regular hand excavation is reported to be significantly more painful. It must be noted that the time involved using the gel technique is usually greater than when using rotary instruments although root surface caries can be dealt with quite rapidly. However, if the time taken to administer and achieve local anaesthesia is included in the procedural time, the total time taken for the different techniques is more or less the same.

Clinical indications

The technique may be a useful treatment adjunct in adult patients with dental fear and anxiety, phobia of needles or an allergy to local anaesthetics. It is also well suited to paediatric patients where there is a risk of soft tissue damage when using rotary instruments in combination with



Fig. 9.21. A selection of hand excavating instruments used with the Carisolv technique.

local anaesthesia. A number of studies have documented patient responses to this treatment in the general practice setting. It has been shown to provide substantial benefits to both clinicians and patients, because of a reduced need for local anaesthesia and an ability to undertake treatment on a number of teeth in a time effective manner (Figures 9.22 and 9.23).

Because the gel does not affect sound tooth structure or restorations, conventional rotary cutting instrumentation or other methods are required to remove existing restorations to gain

access to recurrent caries. As with all methods of caries removal, it is essential to have good access to the inner aspects of the lesion, and to check all surfaces of the cavity for complete caries removal, specially the dentino-enamel junction.

When first using the chemo-mechanical method it may appear to be time consuming. However, once the clinician is familiar with the technique, the gel can be applied simultaneously onto several open cavitations in the same time period, to maximize efficiency of proteolysis, rather than treating one tooth at a time.

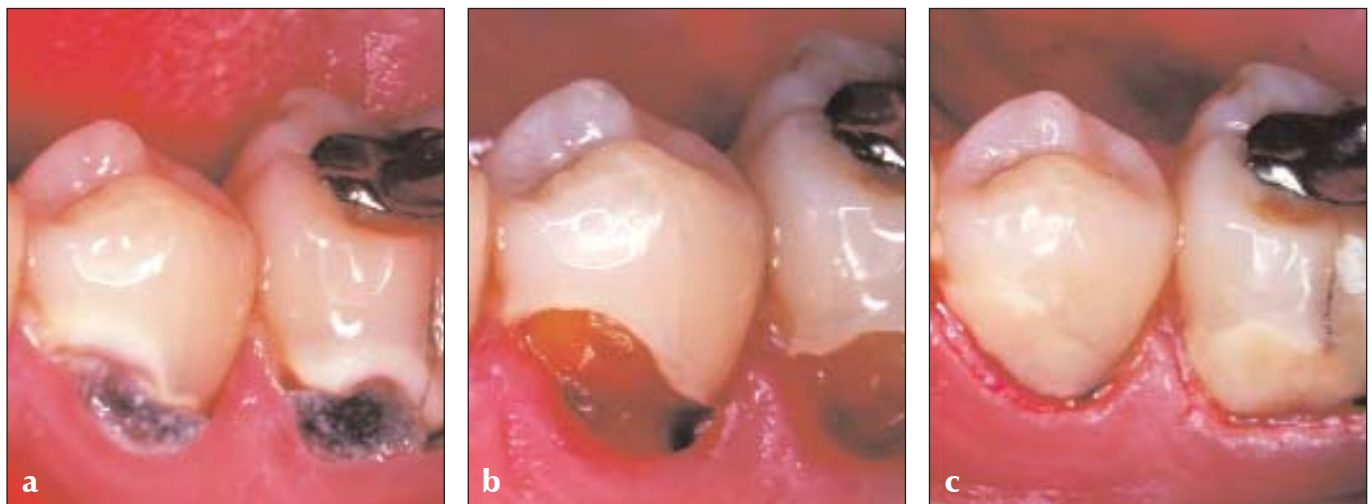


Fig. 9.22. Treatment of rampant caries with Carisolv. **a)** The initial situation. **b)** After caries removal using gel followed by the specially designed hand excavators. **c)** The lesions restored with glass ionomer.



Fig. 9.23. Caries removal on anterior teeth using Carisolv. **a)** A star-shaped hand instrument is used to whisk away softened carious dentine. The gel is opaque because of proteolysis. This dye has a red colouring agent present. **b)** After caries removal has been completed. The outline of the preparation is the shape of the caries lesions and not a pre-conceived outline form.

It has been reported that some patients may experience slight discomfort from the mechanical scraping action and undue drying of the completed cavity preparation can contribute to discomfort. Multicentre clinical trials suggest that patients generally perceive less discomfort than with conventional rotary instrumentation, may be in part, because of the lack of evocative stimuli such as the sound of an air turbine or the sight of local anaesthetic syringes and needles. In fact, many patients report no sensation whatsoever which means the annoyance factor is very low.

Conventional Hand Instruments

Hand instruments preceded rotary cutting instruments by many years and, until the advent of modern high speed handpieces and adequate lubrication of the bur, they were still used for refining the cavity outline. In fact, in the beginning they were essentially the sole means of entering a cavity and G. V. Black and others developed a wide range of chisels, hatchets and hoes for the efficient removal of enamel and dentine and spoon excavators for cleaning out remaining caries. Now that rotary cutting instruments are sophisticated, fast and efficient, the need for hand

instruments is greatly reduced. Two instrument types still have a place (**Figure 9.24**):

- gingival margin trimmers for planing the enamel margins in small spaces where access for rotary cutting instruments is limited

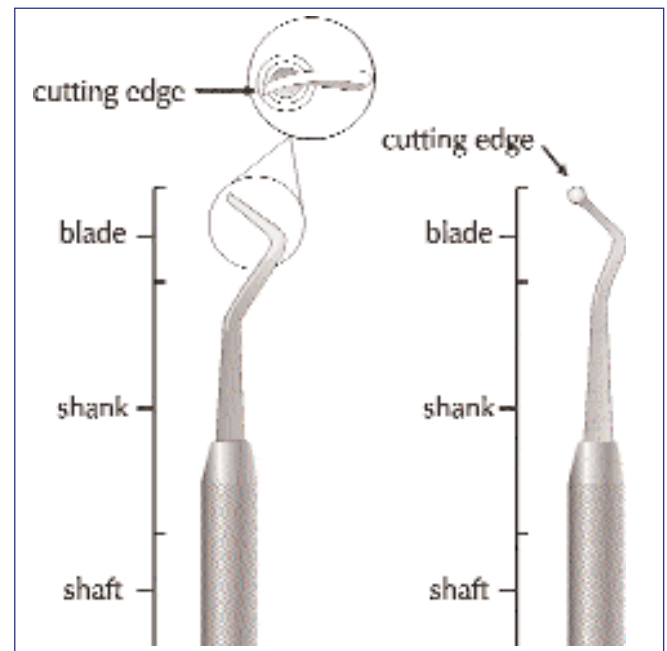


Fig. 9.24. Design of hand instruments: These are typical hand instruments showing the elements in the design. There are infinite variations available in both size and angulation of the blade to the shank as well as the shank to the shaft. Most are double-ended with a left-handed and right-handed functioning blade on each instrument.

TABLE 9.4: Relative effectiveness of cavity preparation technologies

Technique	Sound enamel	Sound dentine	Carious dentine	Metallic restorations	Non-metallic adhesive restorations
Hand excavators	-	-	++	-	-
Rotary burs	++	++	+++	++	++
Ultrasonic instruments	+	+	+++	+	+
Air abrasion	++	+	+	+	++
Carisolv	-	-	+++	-	-
Er:YSGG laser	++	++	+	-	++
Er:YAG laser	++	++	+++	-	++

- no effect; + indicates slow cutting; ++ indicates moderate cutting, +++ indicates rapid cutting or removal

- spoon excavators for the final removal of softened demineralised infected dentine. Take care not to remove all softened dentine because some of it will be affected only and subject to remineralisation (**Chapter 16**)

Gingival margin trimmers

According to the classification of instruments defined by G. V. Black, gingival margin trimmers are modified hatchets. The two modifications incorporated in these instruments are, firstly, that the cutting edge is at an angle to the shaft rather than a right angle and the second is that the blade is curved i.e. it is a double-plane instrument. It is the curve in the blade that makes it a lateral scraping instrument. They are paired instruments where the blade angle is set for either a left or right function and, in the partner instrument, the blade angle is set for planing the enamel rods in either a mesial or distal proximal box.

The width of the blade varies between 1.0 mm and 1.5 mm and it is the finer of the two which is recommended for continuing use. Typical numbers in the Black classification are 10.80.7.14 and 10.95.7.14 and these are fine enough to trim the cavosurface margins of the average minimal cavity without extending it unduly or damaging the adjacent tooth. It is essential that these instruments be kept very sharp and are used with a light touch or there is a risk of introducing micro-cracks in the enamel walls or over extending the cavity outline.

Gingival margin trimmers may be necessary in small cavities where close proximity of the adjacent tooth makes it impossible to use burs without causing damage to adjacent tooth structure. They should be used gently to remove isolated and weakened enamel rods around the cavosurface margin and care must be taken to remove no more than is essential of the remaining enamel. There is a temptation to remove unsupported enamel

from the gingival margin simply because it is weak and unsupported by sound dentine. However, when it is not subject to occlusal load, there is no danger of fracture and its retention may keep the margin of the restoration out of the gingival crevice. In many situations it can be supported and retained by the use of an adhesive glass-ionomer.

Spoon excavators

The spoon excavator is also a double plane instrument that can be regarded as a modified hatchet. However, the cutting blade is curved or rounded. It is also designed for lateral scraping and is generally provided with left and right blades at opposite ends of the same shaft. The typical spoon excavator number is 12.90.8.12 for the right side and 12.10.8.12 for the left and the blades vary in diameter.

It is essential that the blades are maintained in a sharp condition and that they are used with a very light touch so that the demineralised layer on the floor of the average cavity known as the affected dentine (**Chapters 3 and 16**) is maintained rather than removed.

Spoon excavators are designed for the removal of softened dentine at the base of a cavity. However, care must be exercised to avoid producing an unnecessary pulp exposure. The progress of the carious lesion through dentine must be well understood because the deepest layers of softened dentine are merely demineralised and not infected and therefore need not be removed (**Chapters 3 and 16**) Certainly there is no need to remove discoloured dentine because this generally represents the translucent zone (**Chapters 1 and 14**). It may be difficult on occasions to define the parameters of affected dentine and a sharp excavator may penetrate deeper than is required for control of the lesion.

Further Reading

- Hartley JL. Comparative evaluation of newer devices and techniques for the removal of tooth structure. *J Prosthet Dent* 1958; 8:170-182.
- Hudson DC. Cutting properties of dental burs. National Bureau of Dental Standards, Technical News Bulletin. *J Am Dent Assoc* 1955; 39:154-56.
- Schuchard A, Watkins CE. Comparative efficiency of rotary cutting instruments. *J Prosthet Dent* 1965; 15:908-23.
- Thompson EO. Clinical application of the washed field technique in dentistry. *J Am Dent Assoc* 1955; 51:703-13.
- Schuchard A, Watkins CE. Thermal and histologic response to high speed and ultra-high speed cutting in tooth structure. *J Am Dent Assoc* 1965; 71:1451-8.
- Woods RM, Dills WE. Temperature changes associated with various dental cutting procedures. *J Can Dent Assoc* 1969; 35:311.
- Schuchard A, Watkins CE. Thermal and histologic response to high speed and ultra-high speed cutting in tooth structure. *J Am Dent Assoc* 1965; 71:1451-8.
- Watkins CE. Cutting effectiveness of rotary instruments in a turbine handpiece. *J Prosthet Dent* 1970; 24:181-5.
- Rainey JT. Air abrasion: An emerging standard of care in conservative operative dentistry. *Dent Clin of NA* 2002; 46:185-209.
- Christensen GJ. Cavity preparation: cutting or abrasion? *J Am Dent Assoc* 1996; 127:1651-3.
- Elrod DR, Forehand JM, Kutsch VK, Moore BG. *Dental instrument and process*. US Patent 5,934,904. 1999.
- Goldstein RE, Parkins FM. Air-abrasive technology: its new role in restorative dentistry. *J Am Dent Assoc* 1994; 125: 551-7.
- Myers TD. Advances in air abrasive technology. *J Calif Dent Assoc* 1994; 22:41-4.
- Laurell KA, Hess JA. Scanning electron micrographic effects of air-abrasion cavity preparation on human enamel and dentin. *Quint Int* 1995; 26:139-44.
- Hibst R, Keller U. Experimental studies of the application of the Er:YAG laser on dental hard substances: I. Measurement of the ablation rate. *Lasers Surg Med* 1989; 9:338-344.
- Hibst R, Keller U. Experimental studies of the application of the Er:YAG laser on dental hard substances: II, Light microscopic and SEM investigations. *Lasers Surg Med* 1989; 9: 345-351.
- Belikov AV, Erofeev AV, Shumilin VV, Tkachuk AM. Comparative study of the 3 μ laser action on different hard tissue samples using free running pulsed Er-doped YAG, YSGG, YAP and YLF lasers. *SPIE* 1993; 2080:60-67.
- Altschuler GB, Belikov AV, Sinelnik YA. A laser-abrasive method for the cutting of enamel and dentine. *Lasers Surg Med* 2001; 28:435-444.
- Walsh JT, Flotte TJ, Deutsch TF. Er:YAG laser ablation of tissue: effect of pulse duration and tissue type on thermal damage. *Lasers Surg Med* 1989; 9:314-326.
- Walsh JT, Deutsch TF. Er: YAG laser ablation of tissue: measurement of ablation rates. *Lasers Surg Med* 1989; 9:327-337.
- Walsh LJ. The current status of laser applications in dentistry. *Aust Dent J* 2003; 48:146-155.
- Ericson D, Zimmerman M, Raber H, Gotrick B, Bornstein R, Thorell J. Clinical evaluation of efficacy and safety of a new method for chemo-mechanical removal of caries. A multi-centre study. *Caries Res* 1999; 33:171-177.
- Maragakis GM, Hahn P, Hellwig E. Chemomechanical caries removal: a comprehensive review of the literature. *Int Dent J* 2001; 51:291-299.
- Morrow LA, Hassall DC, Watts DC, Wilson NH. Chemo-mechanical method for caries removal. *Dent Update* 2000; 27:398-401.

10 Basic Principles for Cavity Design

G. J. Mount

When a caries lesion has progressed to the point where it is beyond remineralisation and healing it is imperative to remove that part which is broken down and place a restorative material. If the restoration is to be retained for the long term and successfully restore the tooth to its original form and function there are a number of factors to be taken into account. This chapter discusses those factors that, in particular, relate to the effective retention of the material within the tooth as well as the problems of protection of remaining tooth structure that may be weakened by the ravages of caries.

No material is universal and correct selection is important to ensure longevity. In the following chapters the three principle plastic restorative materials will be discussed in sufficient detail to enable the clinician to make a logical choice as to which material to select for each restorative problem.



Introduction

When a caries lesion has progressed to a point where it is beyond stabilising through remineralisation, as discussed in **Chapter 3**, it is necessary to remove that part of the tooth which is irretrievably broken down and replace it with a restorative material. Selection of that material is significant in relation to the longevity of the restoration, because that will help to dictate the longevity of the tooth itself.

Natural tooth structure is a finite commodity and should be preserved and protected as far as possible at all times. There must be good reason to remove healthy enamel and dentine because it can never be regained. The best restorative material is a poor substitute and rebuilding a tooth to full contour, anatomy and aesthetics is both difficult and expensive. Preparation of a cavity will inevitably weaken remaining tooth structure and each time a restoration is replaced the tooth will become weaker still. Replacement dentistry exacerbates tooth loss on every occasion so that longevity of each restoration is of paramount importance.

This means that several aspects of each material should be considered prior to selecting the most appropriate for a particular task

- method of retention of the material within the tooth structure
- prevention of recurrent caries – elimination of microleakage
- ability to assist remineralisation of surrounding tooth structure
- longevity under occlusal load – fracture and wear resistance
- ability to protect remaining tooth from further mechanical failure
- aesthetics

Principle Techniques for Placement

The materials currently in use can be roughly divided into two groups dictated by the method of clinical placement.

Direct restorative materials

These are classified thus because they are plastic, i.e. readily deformable when first mixed, and are placed into a prepared cavity in a tooth whilst still in this condition. During placement they can be modelled or moulded into the appropriate form before they become set and rigid. They vary in their physical properties but are expected to achieve a level sufficient to effectively replace missing tooth structure. They can be used to restore a cavity of considerable complexity because of their plasticity but their physical properties are not always adequate for the tasks imposed. This means that the larger the cavity within the tooth crown the greater the risk of early failure.

Indirect restorative materials

These materials are usually formed on the laboratory bench and the finished restoration is luted into the tooth. This means that the first requirement is the preparation of a cavity in remaining tooth structure, with near parallel walls, that is completely free of undercuts. An accurate model is made using an impression of the prepared tooth so that the restoration can be fabricated on the model and placed within the cavity without distortion or stress and retained with a luting cement.

Whilst many of the basic principles and requirements apply equally with these two groups of materials further discussion on indirect materials would open up entirely new fields which are beyond the bounds of this treatise. Therefore there will only be passing reference to them and the student will need to look further afield for discussions in depth.

Principles of retention

A restorative material can be retained on or within a cavity in a tooth either by adhesion or mechanical interlocking. Adhesion can be further subdivided into a micromechanical interlocking system or a chemical union through an ion exchange.¹ Either way, successful retention is required to prevent the restoration from falling out under occlusal or masticatory load as well as to prevent microleakage of bacteria into the interface between the restoration and the tooth.

Prevention of microleakage depends largely upon careful manipulation and placement of each of the restorative materials but retention can be divided into three categories according to the material used. Composite resins will rely entirely upon micromechanical interlocking between the tooth structure, both enamel and dentine, and glass-ionomer unites with both of these through an ion exchange mechanism. Both are capable of providing a good adhesion free of microleakage and, under most circumstances, will require no mechanical interlocks. However, amalgam and the rigid restorative materials which are manufactured by indirect techniques rely primarily upon mechanical interlocks. A degree of adhesion can be developed through the use of luting or bonding agents but the strength of this form of adhesion is tenuous at best and should not be relied upon.

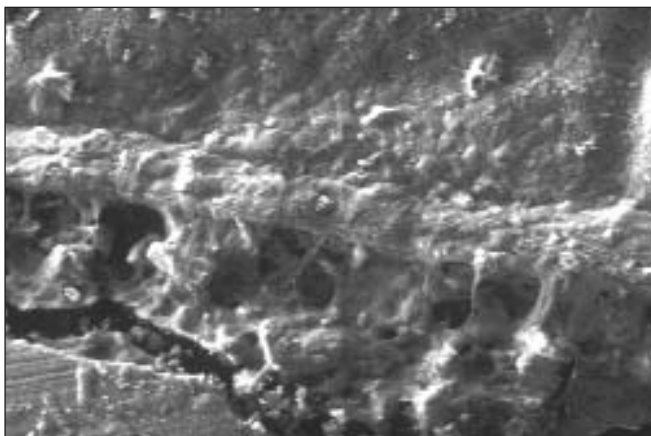


Fig. 10.1. The ion exchange union between glass-ionomer (below) and enamel (above). The specimen was sectioned through the union then lightly etched before viewing under a SEM with a cryo-vac stage. With the water balance stabilised there are no cracks and a true view is obtained of the ion exchange region between the two materials. Courtesy Dr HC Ngo.

SUMMARY



Three types of retention

- Chemical = glass-ionomer
- Micromechanical = composite resin
- Macromechanical = amalgam

Each of the materials will be discussed in relation to their method of retention.

Adhesion with glass-ionomer

Adhesion with glass-ionomer arises entirely through an ion exchange between tooth structure and the cement.² As the glass powder is mixed with a poly-alkenoic acid, calcium/strontium and aluminium ions will be released and will form a matrix which will set and hold the particles together. There will ultimately be formation of orthosilicic acid which will convert to a silica gel as the pH rises and the cement matures, and this will assist further in binding the glass particles together. As the freshly mixed cement is applied to the tooth surface, free polyalkenoic acid will penetrate into both enamel and dentine displacing calcium and phosphate ions and these will combine with the cement matrix forming a new ion enriched material between the tooth and the restoration which is firmly bound to both parent materials. The result is a diffusion based

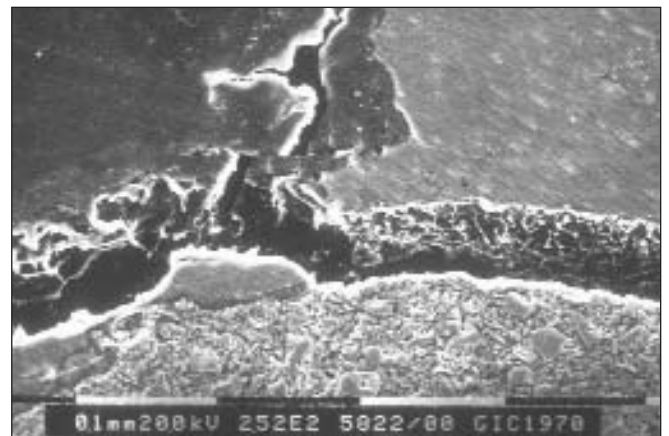


Fig. 10.2. A similar specimen viewed without the cryo-vac stage so severe artifacts are visible with cracks in several areas. The glass-ionomer has split from the dentine but the ion exchange layer is still visible. On the left side pieces of enamel have split away but remain attached to the cement. The dentine and the enamel have separated.

adhesion between the matrix and the glass particles on the one side and the matrix and tooth structure on the other. It would appear that the matrix holding the glass particles together is the weakest material in this mix so failure, if it occurs, will be cohesive within the cement rather than adhesive at the interface (**Figures 10.1 and 10.2**).

Whilst the initial reaction is between the inorganic components of both materials, there is a slow ongoing chemistry which will eventually lead to a degree of union with the collagen component of dentine as well. The cement contains water and water is a byproduct of the chemical reaction, so the presence of further water within the enamel and dentine, is of little consequence and, within limits, will not effect the efficiency of the adhesion.

In clinical practice the rather prolonged setting reaction, as described, must be taken into account. The cement contains a certain amount of water which is 'bound-in' and this is essential for the reaction to occur. There is also some 'unbound' water which, desirably, should be retained to allow the development of full physical potential and acceptable translucency in the finally set cement.

Hence, it is necessary to maintain the water balance until the set material has reached a degree of maturity. This can be achieved by sealing the restoration with a resin enamel bond for up to 24 hours. However, this problem is being overcome and some of the more recent materials do not require this level of protection.³

Physical properties are dictated to a considerable degree by the powder content so care must be exercised in both dispensing and mixing. The higher the powder content the stronger the set restoration up to the point where there is insufficient liquid to adequately wet the powder. Failure will invariably occur as cohesive failure in the cement leaving behind the ion enriched layer firmly attached to the enamel and dentine. This layer will maintain a seal over the dentine tubules thus preventing bacterial microleakage, sensitivity and recurrent caries.

SUMMARY



Adhesion is dictated by

- use of a high powder:liquid ratio
- conditioning to remove smear layer
- maintenance of the water balance during setting

The glass-ionomers adhere in the same manner to both enamel and dentine. The technique of placement is relatively simple and straightforward and longevity is assured following careful application of clinical procedures. This subject is discussed in further detail in **Chapter 11**.

Adhesion with composite resin

It is relatively straightforward to develop a mechanical interlock type of adhesion between enamel and composite resin and, in the presence of sound well supported enamel, this is probably the strongest adhesion available in dentistry.^{4,5} However, the basic resin that is the foundation of composite resin is anhydrous and therefore incompatible with water. This means that adhesion to dentine poses problems because dentine is such a complex material and it is always wet because of the dentine fluid flow from the pulp.⁶

Enamel to composite resin

Etching with 37% orthophosphoric acid for 15 seconds will lead to the development of micropores in the surface of the enamel allowing ingress of an unfilled low viscosity resin to the depth of approximately 50-100 μm . When the resin sets the tensile strength of the union can be as high as 30 MPa. However, it must be noted that, in setting, the resin contracts dimensionally 1-5% and can exert considerable stress upon the union. When bonded to a single surface there may be some flow relaxation during setting but, if placed in a three dimensional cavity, shear stresses of up to 17-20 MPa are possible.⁷ This means that only sound enamel, well supported by healthy dentine, should be bonded and, even so, it is possible to over stress weakened cusps in heavily prepared teeth (**Figures 10.3 and 10.4**).

NOTE

Micromechanical adhesion between composite resin and enamel is

- the strongest adhesion available
- dependant upon the strength of surrounding enamel

Dentine to composite resin

Much of the difficulty of bonding composite resin to dentine lies in the complexity of the histologic structure and the variable composition of the dentine itself. Dentine is only 45% inorganic and the mineral component is randomly arranged within an organic matrix which is essentially collagen. It is intimately related to the pulp through the dentine tubules, which are always fluid filled, and each tubule contains an odontoblastic process. There is a constant intrapulpal pressure of approximately 25-30 mm Hg. resulting in an outward flow of dentinal fluid. Each tubule is surrounded by a hypermineralised collar, peritubular dentine, with the remaining, intertubular dentine, being notably less mineralised. The relative area of dentine occupied by tubules decreases as they diverge from the pulp with about 45,000 per mm². at the pulp surface but only 20,000 at the dentino-enamel junction. In addition the tubules become narrower as they reach the surface. At the pulp they occupy 22% of the cross sectional area, while

near the enamel they occupy only 1%. The challenge then is whether to attempt to adhere the composite resin chemically to the dentine or to develop a micromechanical attachment within the tubules, similar to that achieved so readily with enamel. The former requires a chemical union between a resin, which is essentially hydrophobic, and the dentine which is always wet and contains a complex mix of inorganic and organic material, with greatly varying levels of mineralisation. The latter requires removal of sufficient water for long enough to allow penetration of the resin far enough down into the tubules to provide an effective mechanical interlock (**Figures 10.5-10.8**).

A further method of adhesion involves a micromechanical attachment through development of a 'hybrid layer'. The surface of the intertubular dentine can be demineralised and denatured to a depth of approximately 5 µm. through the application of an acid such as 37% orthophosphoric acid. Unfilled resin can then be lead down into the exposed collagen fibres with a hydrophilic leader, such as acetone, so that, following activation of the resin, there may be a mechanical interlock with the dentine. The composite resin can then be placed over and united to the adhesive resin layer. Recent *in vitro* results, using extracted teeth with no positive dentine fluid flow, suggest high retention strength but there remain unanswered questions concerning longevity of retention in vital

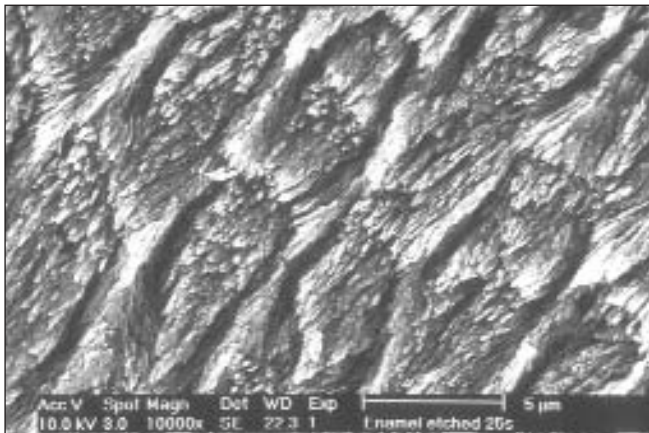


Fig. 10.3. The traditional view of etched enamel showing some enamel crystals have been lost from the outside of the enamel rods leaving the surface quite porous to a depth of about 100 µ. The resin bond will penetrate well into this porous surface. Courtesy Dr H. C. Ngo.

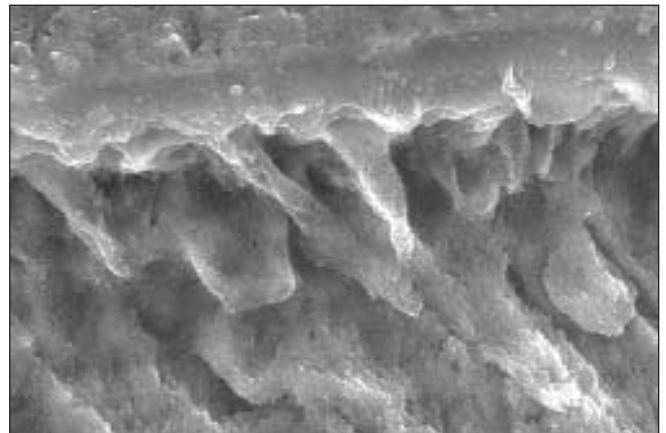


Fig. 10.4. A composite resin restoration was placed on enamel surface then sectioned. The surface was subjected to etching to remove a layer of enamel exposing the resin that has penetrated down into the porosity shown in **Figure 10.3**. Courtesy Dr H. C. Ngo.

teeth with a positive dentine fluid flow. The long term effect on collagen is not yet clear and it is possible that demineralising and denaturing it may lead to it being discarded and separating out from healthy dentine over time. The placement technique is operator sensitive and overall longevity has yet to be confirmed.

To date the results of all techniques, up to generation 7, are equivocal. Relatively effective adhesion figures can be obtained *in vitro* but maintaining these figures under the stresses of the oral environment has proven difficult. The main hazards to date are the relative dimensional instability during setting as well as the hydrophobicity of

the resins which bind the filler particles together and are the principal constituent of the composite resins. With improvements in these two properties long term micromechanical adhesion to dentine may be developed.

BE AWARE



Major hazards for adhesion of composite resin to dentine

- Hydrophobicity of the resin
- Difficulty of eliminating water from the union
- Setting shrinkage of the resin

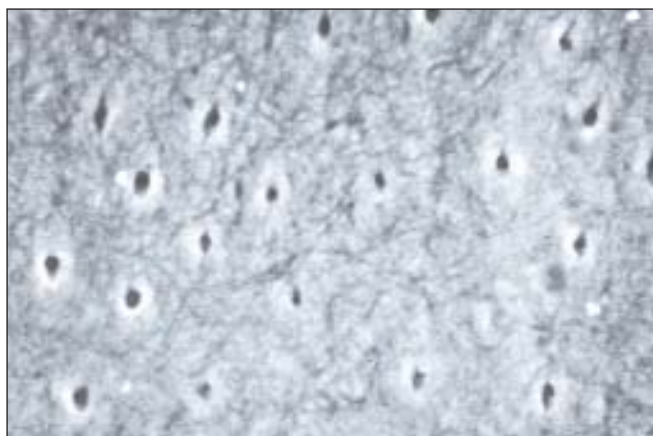


Fig. 10.5. A section through dentine at a level close to the dentino-enamel junction showing the frequency and diameter of the dentine tubules. Only about 10% of the surface area represents tubules. Courtesy Dr H. C. Ngo.

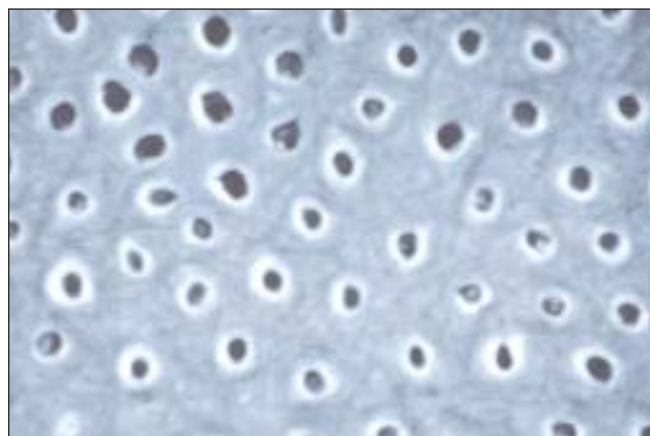


Fig. 10.6. A section through dentine just above the pulp chamber showing the frequency and diameter of the tubules in this area. Note that about 40% of the area represents tubules. Courtesy Dr H. C. Ngo.

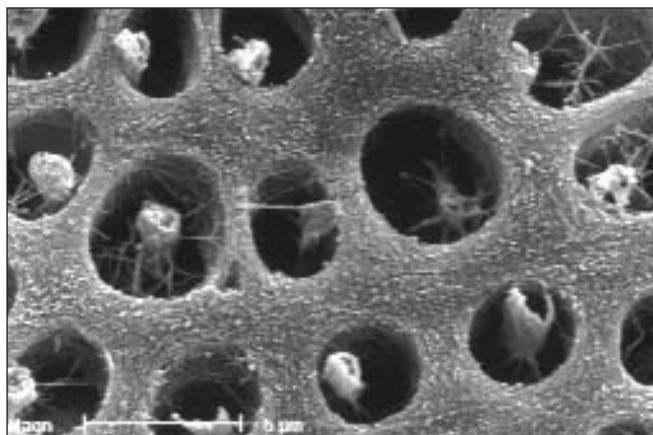


Fig. 10.7. The etched surface of dentine in a vital tooth shown at a magnification of 20,000x. Note the diameter of each tubule as well as the presence of the odontoblast process in each tubule. This emphasises the difficulty of keeping the surface dry during placement of a restoration after etching.

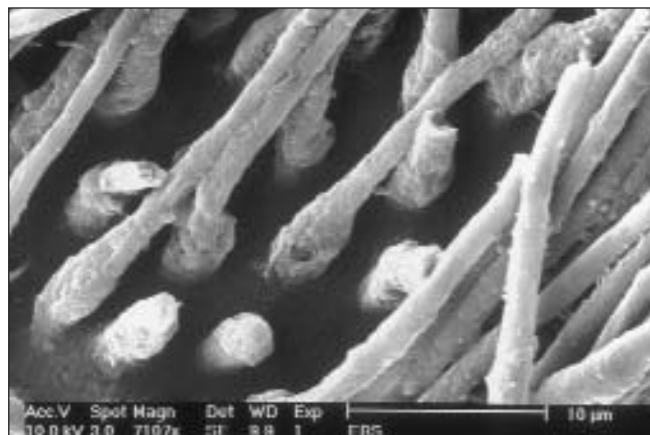


Fig. 10.8. A composite resin was placed in an extracted tooth using normal clinical standards. The tooth was sectioned across the floor and dentine etched away. Note resin tags in the tubules, but there was no dentine fluid or odontoblast in the tubule. Courtesy Dr H. C. Ngo.

The filler particles play no part in adhesion to tooth structure. They are held within the resin matrix either mechanically or through a silane coating which offers some degree of retention to the filler but not to the tooth.

The preferred method of attaching composite resin to dentine is through the use of glass-ionomer as the bonding agent using the lamination technique as discussed in **Chapter 11**.

Retention of amalgam

Neither mechanical nor chemical adhesion is available with amalgam. For a small conventional G. V. Black type cavity it is sufficient to provide parallel or slightly convergent walls to retain a section of amalgam but as the cavity gets larger the problem of retention increases. The major area of strength in remaining tooth structure will lie in the central core of dentine in the gingival one third of the crown of the tooth which suggests that development of mechanical retentive elements surrounding that core will offer the best results for extensive restorations. A further consideration is that, often, a large amalgam restoration will subsequently become the foundation for an extracoronary restoration, so maintenance of the central core of dentine, with mechanical interlocking of the amalgam around it, is very desirable.

Bonding amalgam

There has been a recent trend to use either an unfilled resin or a low powder/liquid ratio glass-ionomer to bond amalgam in to a cavity. These techniques will have value in sealing the interface between the amalgam and the tooth surface to prevent microleakage but will add little or nothing to the physical retention of the restoration. Neither material is strong in tension so the potential for actually reinforcing the tooth over time is very limited. An unfilled resin will shrink on setting and will subsequently take up water and hydrolyse. As discussed above, retention to dentine will not be long term. Glass-ionomer is also relatively weak in tension although there may be some value in its potential for remineralisation. The *in vitro* results to date appear moderately satisfactory but longevity *in vivo* is unproven.

BE AWARE



Bonding amalgam with resin or glass-ionomer

- will seal against microleakage
- may assist in remineralisation
- **will not** enhance retention

Mechanical interlock

The best method of developing retention for amalgam within tooth structure is to place ditches and grooves in remaining dentine in such a manner as to engage the central core of dentine.^{8,9} Use a small tapered fissure bur at low speed and cut the retentive elements at angles to each other so as to reciprocate from side to side - buccal to lingual or mesial to distal. Individual cusps, which are at risk of splitting, can be 'tied on' and a tooth showing a mesiodistal split can be 'tied together'. Each groove can be made self retentive by turning the tip of the bur outwards at the end of the groove thus, in effect, converting each into a dovetail.

A tapered fissure bur is recommended because the resultant ditch will be wider at the entry than at the base, and this will facilitate the condensation of amalgam into the groove.¹⁰ Spherical particle amalgam will flow readily so that it can be fully condensed into a groove up to 3 mm deep (**Figures 10.9-16**).

Each section of an amalgam restoration should be individually retentive so, as the cavity is extended, ditches and grooves should be incorporated in the design. Any extensive restoration should normally be regarded as a foundation and core for a subsequent extracoronary restoration, which will almost certainly be required as the patient ages. This means it is essential that each section is firmly locked into place so that the amalgam will then become a base, or a dentine replacement, to support the final restoration.

Supplementary pins

The use of cemented, friction grip or selfthreading pins has been recommended in the past to enhance retention.^{11,12,13} However, both cemented and friction grip pins offer very limited mechanical advantage and selfthreading pins place sufficient stress on the dentine to lead to subsequent development of microcracks.¹⁴



Fig. 10.9. A traditional G. V. Black- Class II cavity in the distal of an upper molar. Note that it has been conservatively prepared but it has still removed the entire proximal wall of the tooth thus weakening the surrounding cusps.



Fig. 10.10. A large cavity taking in the distal and occlusal of an upper molar. This represents replacement dentistry so the cavity is extensive and retention of the restoration requires modification of the design. Note the grooves and ditches in the proximal box as well as the ditch between the two buccal cusps.



Fig. 10.11. The amalgam restoration in this tooth was placed clinically about ten years before the tooth was lost. It has been sectioned to show the depth and extent of the groove in the gingival floor of the proximal box.



Fig. 10.12. Grooves were cut in the floor of an occlusal cavity in an extracted tooth to demonstrate the ability to pack amalgam effectively into the grooves regardless of the depth. They were cut using a mild steel tapered fissure #700 to a depth of 1.0, 2.0 and 3.0 mm. and then the cavity was restored with a high copper spherical alloy. Note the condensation is adequate in all three grooves.

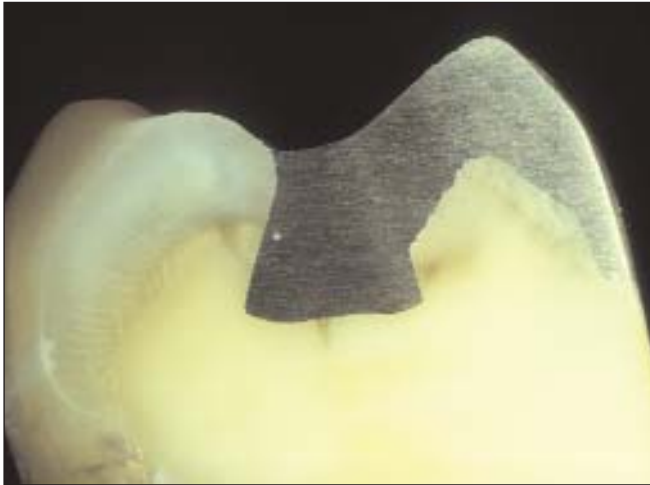


Fig. 10.13. An extensive cavity was cut in an extracted tooth to show the effect of retentive grooves in locking an amalgam restoration to place. The grooves were cut using a mild steel tapered fissure #700. Having reached the required depth the tip of the bur was turned out at each end of the groove leading to a dovetail effect to lock the amalgam in to place. The tooth was then cut away to demonstrate the effect.



Fig. 10.14. The upper first molar is being prepared for a full crown. An extensive amalgam had been placed previously as a core and reduction of the buccal surface reveals the effect of the dovetail lock placed in the extension of the buccal fissure.



Fig. 10.15. A very extensive amalgam core is being placed in to this extracted molar tooth in preparation for a crown. Careful examination will reveal the grooves and ditches prepared to provide retention.



Fig. 10.16. The tooth shown in **Figure 10.15** has been restored with amalgam and then sectioned mesio-distally to demonstrate the efficiency of the grooves in locking the amalgam to place around the central core of dentine. Both the mesial and the distal groove are turned in slightly toward the centre of the tooth so the amalgam cannot be lost.

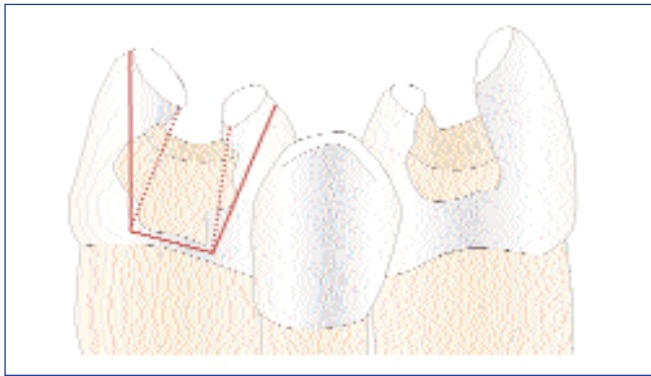


Fig. 10.17. A diagrammatic representation of a protective design for supporting a weakened cusp and preventing it from splitting. Note that working cusps require more protection than nonworking cusps but at the same time this design will maintain cusp height more effectively than the traditional cusp cap design.

Their use is not recommended in general, but there may be unusual circumstances where placement is warranted.¹⁵

A supplementary pin must be well surrounded by sound tooth structure and will need to lie parallel to both the external tooth surface and the wall of the pulp chamber. It is worth noting that, at any given point on the root surface, the surface of the root is parallel with the inner surface of the pulp chamber. This means that a periodontal probe placed against the root surface, will give a guide to the direction in which the pulpal wall will be lying. The pin hole should be 2-3 mm deep into dentine and no more than 1 mm of the pin should be left standing into the cavity. It is unwise to bend pins after placement because of the risk of propagating cracks in the dentine. If they are properly placed bending is unnecessary. The pin can be shortened if essential using a small diamond bur at ultra-high speed under air/water spray.

BE AWARE

Pins to enhance retention

- cemented
- friction lock
- self-threaded

are **NOT** recommended

Protection of Remaining Tooth Structure

Preparation of a cavity will compromise the strength of the remaining crown. Preparation of a trench into dentine along a fissure system on the occlusal surface will effectively double the length of a cusp and leave it more susceptible to flexure and fracture. Further preparation of a proximal box in the same tooth will double the length of the cusp again and development of a split at the base becomes more predictable.

Split cusps

Split cusps are frequently encountered in heavily restored teeth and can occur even in unrestored teeth. They are generally the result of frequent loading on medially facing, sharply angled cusp inclines, often through working side contacts in lateral excursions, and may be difficult to identify. The patient will report pain on pressure, or possibly, following release of pressure, which will become progressively more consistent over time. The tooth will eventually become temperature sensitive as well. Identification of the failing cusp is difficult in the beginning and testing by the application of pressure to each individual cusp is necessary but not always reliable.

The split generally begins at the level of the pulpal floor of the cavity, at either the buccal or lingual line angle, and progresses on a gentle curve outwards to the cemento-enamel junction. When the cusp is lost the margin will often be just supragingival. However, on occasions, the split will commence more medial to the line angle and may progress through the pulp chamber and out on to the root surface below the epithelial attachment or even the alveolar bone crest. The tooth will then probably lose vitality and restoration may be rather complex or even impossible.

BE AWARE

A split cusp poses a serious diagnostic quandary.

Prevention of splits

Prevention of splits at the base of a cusp or their propagation to full fracture is highly desirable. If a cavity is so extensive that it undermines one or more cusps the cavity should be designed in such a way that the restorative material accepts the occlusal load over the cusp/s.¹⁶ Both tooth structure and plastic restorative materials are weak and brittle in thin section but are strong in bulk. If the remaining tooth is weak it is necessary to provide bulk in the restoration to protect the tooth from occlusal load. In the #2.2 cavity there is sufficient strength in both the buccal and lingual walls to support the restoration. The #2.3 category deals with the reverse situation where it is necessary to rely on the restoration to protect the remaining tooth structure (Chapter 14).

Restoration of a posterior tooth requires reconstitution of the occlusal anatomy at the correct height with a proper relationship to the opposing tooth. Maintenance of the original cusp height on the tooth to be restored makes it easier to restore the occlusal level so it is worthwhile trying to retain full cusp length when designing the cavity (Figure 10.18).

As long as the cavity is simple and the marginal ridge remains intact, the risk is not great and the tooth will be strong enough to protect the restora-

tion. Placement of an adhesive restorative material will, in fact, return the tooth close to its original strength. However, following removal of the proximal surface as well as trenching the occlusal surface (as in the conventional Class II cavity), it is necessary to consider the need to protect the remaining cusps from undue occlusal load. Further involvement of the central core of dentine will exacerbate the problem and modification to cavity design becomes essential. The stage will be reached where it is necessary to rely upon the restoration itself to accept the occlusal load and this involves both cavity design as well as selection of a restorative material capable of providing strength and protection.

Cavity design for cusp protection

Tooth structure and most restorative materials, apart from gold, must be regarded as relatively brittle. They are strong enough in bulk to withstand masticatory stress but in thin section will fail rather easily. Therefore modification to cavity design should aim at provision of the restorative material in bulk to provide protection for the tooth structure which is now regarded as weak and brittle. At the same time it is desirable to retain as much of the gingival one third of the crown as possible because therein lies strength



Fig. 10.18. A lower molar that was responding with pain on pressure. Removal of the restoration reveals a split at the base of the mesio-lingual cusp that will require a cavity design as suggested in Figure 10.17.



Fig. 10.19. Completing a cavity design in an extracted lower molar in which both buccal and lingual cusps are being protected as suggested in Figure 10.17. Note the use of a mild steel tapered fissure #700 bur to develop the retentive ditch and groove under the buccal cusp.



Fig. 10.20. The completed restoration in the tooth shown in **Figure 10.19** showing the level of protection provided for the buccal and lingual cusps. Maintaining height makes it easier to maintain proper occlusion with the opposing tooth.

and retention for the extracoronal restoration which will, so often, be the final restoration.

It is essential first to remove the weakened tooth structure from undue occlusal load. This can be achieved by eliminating the medially facing inclines from the occlusal end of the cusp and at the same time retaining as much as possible of the original cusp height.^{17,18}

With all plastic restorative materials it is necessary to provide bulk in the restoration. The combined effect can be developed by leaning the facial and lingual walls out from the gingival floor, in a straight line to, or just beyond, the tip of the cusp. A non-working cusp does not require a great deal of support so it is sufficient to provide approximately 0.5 mm of coverage. However, a working cusp will still be subject to a heavy load and therefore requires up to 2.0 mm of coverage depending on the type of occlusion. By leaning the walls outwards in this fashion the restorative material can be built over the cusp with a cavosurface margin of close to 90° without compromising the strength at the gingival base of the cusp (**Figure 10.17**).

NOTE



Cusp protection

- Amalgam has the strength
- Glass-ionomer is too brittle
- Composite resin is too flexible, and the enamel margin will be too fragile

Care must be taken not to reduce the cusp height any further than necessary because one of the more difficult problems in building a restoration in a plastic material is to restore the full occlusal height to the tooth. Failure to do so may well compromise the relationship with the opposing tooth and thereby alter the occlusion (**Chapter 18**).

Other Significant Factors

Prevention of recurrent caries

Elimination of the active disease is the recurring theme of this text. Prevention of microleakage depends to a high degree on the quality of handling and placement techniques. Development of the correct contour is entirely in the hands of the operator. However, there are other aspects which should be considered.

BE AWARE



Recurrent caries will arise from

- failure to overcome the active disease
- microleakage – generally poor placement
- over contour allowing plaque accumulation

Glass-ionomer

Because of the ion exchange adhesion available there is essentially no microleakage possible in the presence of a properly placed glass-ionomer. Any failure of adhesion will be cohesive within the cement leaving a thin layer of ion enriched cement behind which will maintain a seal over dentine.

The continuing fluoride release will also discourage the formation of bacterial plaque as well as encouraging a degree of remineralisation of

surrounding tooth structure, particularly areas already demineralised.

Composite resin

The micromechanical adhesion available between enamel and composite resin will prevent ingress of bacterial contamination providing the enamel is soundly based and does not fracture as a result of the setting shrinkage. The union with dentine is regarded as less durable because of the hydrophobicity of the resin. The present recommendation is that a glass-ionomer base be placed first to provide adhesion with dentine, and then a composite resin can be laminated over this, thus providing adhesion to both enamel and dentine.

Attempts have been made to develop a composite resin which will release a reliable flow of fluoride at a level similar to that attained from glass-ionomer. However, ion migration is only possible in the presence of water so, as resin is anhydrous, a reliable long term ion release is not possible. So far there are a few materials in this class that claim fluoride release but at a level and a consistency substantially below the amount required to have any bearing on a caries rate.

Amalgam

Amalgam shows no adhesion to tooth structure but it does corrode reasonably rapidly in the oral environment. A fortunate side effect from this is that it has a tendency to seal the interface between the restoration and the tooth with corrosion by-products, which in turn will prevent microleakage. The development of the sealing effect can be controlled to a degree by painting the cavity surface with one or two layers of copal varnish prior to placing the restoration. The varnish is soluble in the oral environment so it will slowly wash out of the interface. As it dissolves the corrosion will occur and the byproducts – largely tin ions – will seal the margin.

An alternative, currently being explored, is to apply a resin or glass-ionomer bond to the tooth surface and then pack the amalgam directly into the cavity while the bond is curing. *In vitro* testing suggests acceptable levels of retention but the strength will only be as strong as the weakest link, and the bonding material is likely to be the first to

give. There may be an advantage to be gained from the fluoride release of glass-ionomer but such bonding agents should not be relied upon to retain an amalgam restoration in place or to reinforce the tooth structure in the presence of poor cavity design.

Amalgam has no in-built resistance to plaque accumulation and attempts to incorporate fluoride have not been successful.

Longevity under occlusal load

All the restorative materials are capable of withstanding normal occlusal loads although it must be accepted that there is a wide variation within the bounds of normal.¹⁹ The actual physical properties of each of the plastic restorative materials will be discussed in the following chapters and development of these properties to their full extent, when placing them in the oral cavity, is entirely in the hands of the operator.

Aesthetics

Aesthetics must be regarded as a matter of opinion and warrants full discussion with the patient. Nothing can be regarded as permanent and stable in as much as natural tooth structure is constantly maturing and changing colour. This means that a good colour match initially may not remain so. It is unwise to select a material entirely on the grounds of aesthetics. Longevity is by far the more important property because every time a restoration is replaced there will be further loss of tooth structure resulting in progressive weakening of that which remains. Selection of the appropriate material in each case warrants a full discussion with the patient and properties such as aesthetics should be kept firmly in perspective.

BE AWARE



Selection of a restorative material is critical

- longevity is the primary motive
- ability to accept the occlusal load
- ability to protect remaining tooth structure
- aesthetics should be the final parameter

In the light of the current tendency for an extended life span for our patients it is desirable to extend the life span of each restoration, keeping in mind the fact that no restoration can be regarded as permanent.

The Final Selection

The following factors should be taken into account when deciding the final treatment plan for the selection of materials to be used as well as the other factors discussed above.

Direct plastic restorative materials

- Possibility to minimise cavity design thus conserving remaining tooth structure.
- Relatively simple to place, to repair and to replace.
- Relatively economical in time, material and therefore cost.
- Some materials offer acceptable aesthetics.
- Some materials allow mechanical adhesion to enamel and some allow chemical adhesion to both enamel and dentine.
- Some materials allow a level of ion exchange and therefore encourage healing of tooth structure and discourage further caries.

Factors in favour

Keeping these factors in mind, a direct plastic restorative material is the obvious choice for the small initial lesion where removal of natural tooth structure can be kept to a minimum. Removing enamel and dentine to make room for the restorative material is highly undesirable and the use of modern adhesive materials obviates this to some degree.

The fact that these restorations can be readily, and relatively inexpensively, added to, modified, repaired or replaced makes them the logical choice in the young patient where caries activity is difficult to monitor or is unpredictable. Also for the older patient, who presents with an apparent acute attack of active caries, it is probably wise, in the first instance, to restore with these plastic

materials until such time as the caries rate is again stabilised.

Factors against

Because the operator is working in a very confined space, with limited access and only one direction of approach, there are limitations to the ability to rebuild a tooth crown to its original anatomy. This means there is a constant risk to both the occlusal relationship with opposing teeth, as well as proximal relationship with adjacent teeth, with the ever present risk of leading to a continuance of dental disease rather than its elimination. These problems will be addressed further in **Chapters 16, 17 and 18**.

Indirect rigid restorative materials

- Restorations are usually constructed indirectly on the laboratory bench.
- Accurate reproduction is possible for occlusal anatomy in relation to opposing teeth and proximal anatomy in relation to adjacent teeth and gingival tissues.
- Some materials can be used in thin section to protect remaining tooth structure from occlusal load.
- Some materials are ideal for the longer term reproduction of aesthetics.

Factors in favour

The ability to reproduce the original anatomy of the crown of a tooth is of great value to ensure the longevity of a single restoration, or a series of restorations. Failure to maintain the correct envelope of movement of the mandible against the maxilla can lead to bruxing, clenching and grinding of teeth and may result in further splitting of remaining tooth crowns (**Chapter 18**). Maintenance of correct emergence profile and contact areas is essential for the health of periodontal tissues and the avoidance of recurrent caries (**Chapter 17**).

Ceramic restorations provide the best aesthetic results available and can only be constructed indirectly.

Factors against

It is necessary to develop a cavity with divergent walls to make it possible to obtain an accurate model of the tooth and to provide a path of insertion for the restoration without stress. This will invariably require further destruction of sound tooth structure. It is possible, when using gold, to protect a remaining cusp with the metal in very thin section but there is still undue tooth reduction necessary to develop a suitable line of withdrawal and occlusal protection.

There are many stages required for the completion of the restoration and each stage is open to error. Excellence in the end result demands excellence at each stage and this inevitably leads to additional cost factors. Therefore, indirect restorations are relatively costly and time consuming. However, these factors must be balanced against the potential longevity and aesthetics before a final decision is made.

Further discussion

The next four chapters will deal with the direct plastic materials ending with cavity preparation procedures and placement techniques. A modified classification of caries lesions will also be presented in **Chapter 14** which will take into account the present ability to develop reliable adhesion in the oral environment as discussed above.

Discussion will begin with glass-ionomers, composite resin and amalgam, because these are the materials used most commonly in general dental practice. The nature and use of the rigid restorative materials will not be pursued in this treatise because it brings into focus a number of complexities which are not appropriate for discussion at this time.

The purpose of these chapters is to provide information that will enable the dental student and practising dentist to achieve optimum clinical results by understanding better the reasons for material selection, and the manipulative requirements for success. To achieve this, three closely linked aspects of each material are discussed:

- composition, setting mechanisms and structure relevant to material selection and clinical performance,

- properties and clinical characteristics likely to influence success and failure,
- clinical considerations which are relevant to the development of an optimum end result, under the limitations imposed by placing the materials directly into the oral cavity.

The Use of Rubber Dam

The emphasis in the preceding pages has been on quality of handling methods and procedures to ensure longevity for each restoration placed. This has to be one of the greatest challenges to the operative dentist because the work has to be carried out in a confined area with limited access and the constant threat of saliva contamination. None of the restorative materials – direct or indirect – can withstand the challenge of contamination so any technique that will assist in avoiding this should be utilised in routine restorative dentistry. It is accepted that the most effective method is the use of rubber dam (**Figures 10.21-24**).

There have been arguments offered both for and against the use of rubber dam as a routine during cavity preparation and restoration and there are many substitutes available designed to maintain access and saliva control.²⁰ None are as effective nor as simple to place and maintain as dam and its use is strongly encouraged. In the presence of really tight contact areas it is possible to open one cavity first before trying to apply the dam. Freeing one contact will allow some degree of movement of remaining teeth and ease placement interproximally, if the team is inexperienced. The following points should be considered in selection and placement:

- Always use light or medium weight dam – it is easier to place but still effective.
- Placement is a four handed operation with a well trained chairside assistant. As a team effort the dam should always be placed with in three minutes.
- The assistant holds the dam out in front of the operator, retained by the four corners only, to allow the operator to visualise the entire square of dam.



Fig. 10.21. A standard set of wingless clamps suitable for general application in operative dentistry. Note the limited number because too many will only create confusion.



Fig. 10.22. A standard set up ready for quick application of dam under most circumstances. Note the bi-angle forceps for placement of the clamp and the scissors with the curved tip which facilitates cutting the interproximal dam when removing it.



Fig. 10.23. The dam properly placed ready for action. Note the use of the napkin which, while not essential, is a comfort for most patients. Low volume suction is not essential for salivary control and an additional hole can be cut high in the dam to ease breathing if required.



Fig. 10.24. The obvious main reason for the routine use of dam. Entrapment and retention of scrap such as this can be a great comfort to the patient and represents good mercury hygiene.

- The operator punches the required holes making sure, if possible, to begin one tooth distal to the tooth to be restored and include the entire quadrant.
- Identify the top left hand corner of the square of dam by punching a single hole so the orientation does not become confused during placement.
- Use castor oil as lubricant because this will not affect the rubber and will provide excellent lubrication.
- Select the appropriate clamp for the anchor tooth. Use wingless clamps.
- Modify the shape of a clamp by grinding the inner margin to ensure a firm fit.
- Tie a length of floss to the clamp in case it flips off the tooth during dam placement.
- Guide the last hole in the dam over the anchor clamp.
- Now, using the four hands available, position the frame, anchoring the four corners positively on to the frame.
- Next engage the teeth at the midline first so the remaining holes are now sited over their appropriate teeth.
- Both the operator and the assistant now ease

the dam over the crowns of each tooth in turn using one finger on the buccal and one on the lingual. This will stretch the dam over the contact area of each pair of teeth.

- Use the blade end of a small #1 flat plastic instrument to separate the teeth gently to allow the dam to slip through the contact points. It may be necessary to use floss to persuade the dam right through.
- Invert the margin of the dam using a small binangle probe while blowing compressed air onto the tooth to dry it and hold the dam in place.
- If there is any likelihood of damaging the dam while preparing a proximal cavity place a wooden wedge to force the dam and the soft tissue gingivally.

As a general routine it is best to expose a full quadrant on every occasion even when restoring a single tooth. This means going at least to the mid-line and, for preference, extend to the next tooth beyond. Where possible place the clamp at least one tooth distal to the one to be restored although a matrix can be positioned to replace the clamp when the restorative material is to be inserted.

SUMMARY



The major advantages can be listed as follows

- enhanced visibility for both operator and chair-side assistant
- high degree of cleanliness available for operator and patient
- complete sterility can be achieved
- operator and assistant have both hands free to concentrate on the task in hand
- patient can relax the tongue and cheeks knowing access is constantly available to the operator and there is no need to swallow constantly

With practised team work it should be possible to place dam in even the most difficult situation within three minutes. Some degree of training and discussion may be required for a patient with a high gag reflex but there is no doubt they will be more comfortable and secure once it is placed. If necessary cut a small hole high up in the dam to make it easier for the patient to breath through although mostly they will breath around it. The placement of oral evacuation behind the dam is optional and not always required.

Further Reading

1. Phillips RW. *Skinner's Science of Dental Materials*, 9th Edition. Philadelphia: WB Saunders; 1991, Chapt. 2.
2. Mount GJ. Adhesion of glass-ionomer cement in the clinical environment. *Oper Dent* 1991; 16:141-148.
3. Leirskar J, Nordbo H, Mount GJ, Ngo H. The influence of resin coating on the shear punch strength of a high strength auto-cure glass ionomer. *Dent Mater* 2003; 19:87-91.
4. Glantz PO. Adhesion to teeth. *Inter Dent J* 1977; 27:324-31.
5. Proceedings of the International Symposium on Adhesives in Dentistry. *Oper Dent*, Supplement 5, July 1991.
6. Oilo G. Bond strength testing - what does it mean? *Inter Dent J* 1993; 43:492-498.
7. Ausiello P, Apicella A, Davidson CL. Effect of adhesive layer properties on stress distribution in composite restorations-a 3D finite element analysis. *Dent Mater* 2002; 18:295-303.
8. McMaster DR, House RC, Anderson MH, Pelleu GB. The effect of slot preparation length on the transverse strength of slot retained restorations. *J Prosthet Dent* 1992; 67:472-7.
9. Garman TA, Outhwaite WC, Hawkins IK, Smith CD. A clinical comparison of dentinal slot retention with metallic pin retention. *J Am Dent Assoc* 1983; 107:762-3.
10. Outhwaite WC, Garman TA, Pashley DH. Pin vs slot retention in extensive amalgam restorations. *J Prosthet Dent* 1979; 41:396.
11. Davis SP, Summitt JB, Mayhew RB, Hawley RJ. Self threading pins and amalgapins compared in resistance form for complex amalgam restorations. *Oper Dent* 1983; 8:88-93.
12. Durkowski JS, Pelleu JB, Harris RK, Harper RH. Effect of diameter of self-threading pins and channel location on enamel crazing. *Oper Dent* 1982; 7:86-91.
13. Evans JR, Wetz JH. The pinned amalgam restoration. Part 1: A review. *J Prosthet Dent* 1977; 37:37-41.
14. Khera SC, Rittman BRJ. Dentinal crazing and inter-pin distance. *J Prosthet Dent* 1978; 40:538-43.
15. Papa J, Wilson PR, Tyas MJ. Pins for direct restorations. *J Dent* 1995; 21:259-264.
16. Salis SG, Hood JA, Kirk EE, Stokes AN. Impact-fracture energy of human premolar teeth. *J Prosthet Dent* 1987; 58:43-8.
17. Mount GJ. The use of amalgam to protect remaining tooth structure. *NZ Dent J* 1977; 73:15-20.
18. Mount GJ. The three stages of the amalgam restoration. *Aust Dent J* 1978; 23:75-80.
19. Plasman PJJM, Kusters ST, de Jonge BA, van't Hof MA, Vrihoef MMA. In vitro resistance of extensive amalgam restorations using various retention methods. *J Prosthet Dent* 1987; 57:16-20.
20. Benavides R, Herrera V. Rubber dam with washed field evacuation: a new approach. *Oper Dent* 1992; 17:26-28.

11 Glass-ionomer Materials

G. J. Mount

Glass-ionomer was developed in England and first reported by Wilson and Kent in 1972. At that time it was recommended for the restoration of cervical abrasion lesions but the early versions lacked aesthetics and translucency. Subsequent development and research by various manufacturers has evolved a number of very useful materials which can be used for a variety of functions in restorative dentistry including luting rigid restorative materials, lining and basing cavities prior to placement of other restorative materials as well as an aesthetic translucent restoration in its own right. Since their development glass-ionomers have increasingly become an essential part of the dentist's armamentarium for providing treatment that conserves tooth structure and assists in its remineralisation while, at the same time, maintaining aesthetics.

Of the numerous attractive characteristics of the glass-ionomers, the most significant are the ability to bond chemically to dentine and enamel through an ion exchange mechanism, as well as an ability to exchange ions with remaining tooth structure and the oral environment. This leads to a long-term release of fluoride and other ions without increasing solubility and the ability to re-absorb these ions and therefore acting as an ion reservoir. As a result it will assist in the remineralisation of surrounding enamel and dentine and therefore lead to a degree of healing of a caries lesion. The principal negative feature, at this point, is susceptibility to brittle fracture – the material is unable to withstand undue occlusal/incisal loading.



General Description

Glass-ionomer is a water based cement formed from the acid/base reaction between a polyalkenoic acid as the liquid and an ion-leachable, fluoride containing, calcium or strontium aluminosilicate glass as the base.¹ The official ISO terminology for glass-ionomer cements, 'glass polyalkenoate cements', indicates the principal components. The terminology used is consistent with current understanding of the chemistry and the properties of the materials and has received general endorsement (Proceedings of the 2nd International Symposium on glass-ionomers, Philadelphia, 1994).

Developments in the glass-ionomer class of materials in recent years have resulted in the availability of a range of materials extending from the original restorative glass-ionomer to a luting material, a lining material and a base or dentine substitute. Some of them have been modified by the inclusion of various resins, such as HEMA, in an attempt to improve physical properties and increase resistance to early water contamination. However, there will come a point where the ion exchange properties will be overwhelmed in the presence of a resin thus negating one of their principle advantages. In general the autocure materials, free of resin, are once again the preferred versions under most circumstances.²

The basic glass powder

The approximate composition of the calcium or strontium fluoroaluminosilicate glass that forms the basis of the powder component in glass-ionomers, is shown in **Table 11.1**. The mixture is fused, quenched, ground and sieved to obtain a particle size ranging between 4-50 μm depending upon the proposed clinical application for each material.

Generally, finer particles are used for luting cements and lining cements and coarser particles are used for restorative materials because these are likely to provide better translucency. The range and particle size distribution will have a bearing on the working properties, the setting

speed and the final physical properties of each material.³

The glass powder may be modified in several ways. The reactivity of the glass is controlled by the fusion temperature and the heat history during manufacture. The glass mix will be taken to a given temperature, allowed to cool for a specified period and then quenched in water. Materials formed with higher temperature glasses set more quickly and reactivity can be varied by modifying the annealing process. These modifications will be utilised to vary setting time as well as physical properties.

Various ions can be added to enhance radiopacity such as strontium and lanthanum. It is interesting to note that strontium is close to calcium on the periodic table and can therefore substitute entirely for calcium. This has been incorporated into some of the materials and, as a side issue, has made it easier to study remineralisation of demineralised tooth structure under a glass-ionomer restoration. This arises because strontium ions will be taken up into the demineralised dentine instead of calcium ions and tracing the strontium is relatively easy and accurate.

Additions to the glass

Cermet (ceramic-metal) powder particles are formed by sintering a metal such as silver or gold to the surface of the glass particles (**Figure 11.1**). The powdered glass is mixed with finely powdered metal and then heated again to a given temperature. Some of the metal becomes sintered to

TABLE 11.1: Approximate composition of a calcium fluoroaluminosilicate glass

Component	Weight %
SiO_2 (quartz)	29.0
Al_2O_3 (alumina)	16.6
CaF_2 (fluorite)	34.2
Na_3AlF_6 (cryolite)	5.0
AlF_3	5.3
AlPO_4	9.9

Adapted from Prosser *et.al.*

the surface of the particles and the excess metal has then to be washed out. Up to 5% (weight) of titanium dioxide may then be added to the powder to restore the material to a clinically acceptable colour.⁴

The alternative of adding metal flakes or spheres, such as amalgam alloy (**Figure 11.2**) to the powder, may improve the abrasion resistance and radiopacity but does not appear to improve other physical properties to any extent.⁵ In fact, there appears to be a reduction in the integrity of the ion exchange adhesion. The physical properties of the basic material is of greater importance than the inclusion of metals so these technologies are no longer as popular as they were.

Fluoride content of the glass

Fluoride is an essential component of a glass-ionomer because of its effect on the temperature of fusion of the glass and its action as an oxide scavenger during the melt. It can affect the working characteristics and the ultimate physical properties of the finished restoration, although it is not a physical part of the matrix. The fluoride ion is very small, almost as small as the hydroxyl ion, so it can move reasonably freely both out of and back into the set glass-ionomer, without bringing about any dissolution or breakdown. Its presence is appreciated for its involvement in

remineralisation in surrounding tooth structure.

The fluoride content of the glass can be varied and a moderate reduction will enhance translucency without unduly reducing the remineralisation potential. It may also have some inhibitory effect on plaque formation in the vicinity of a restoration although it is becoming apparent that other metal ions, such as zinc, may be as effective, or more so, in this action.

The liquid

The scientific terminology of glass-polyalkenoate arises from the acid which is one of the polyalkenoic acid family, only a few of which have so far been tested in this context. A typical liquid contains a 40-55% solution of 2:1 acrylic acid-itaconic acid copolymer in water or, alternatively, a copolymer of maleic acid/acrylic acid. The use of copolymers improves storage compared with the aqueous solution of polyacrylic acid used in the original glass-ionomers because they tend to become quite viscous relatively quickly. It is also possible to incorporate variations in the liquid. There has been limited experimentation on variations from the original polyalkenoic acid with trials being conducted with polyphosphonic acids but only the polyalkenoic acid has so far been clinically successful.

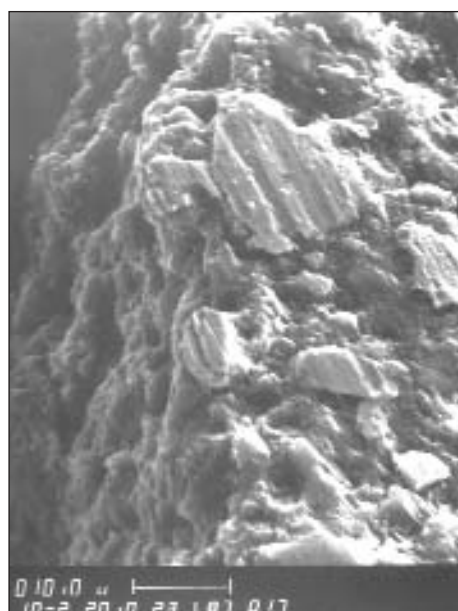


Fig. 11.1. A SE micrograph showing a section through a cermet material. Note the relatively rough glass particles that are covered with very fine silver particles on the surface.

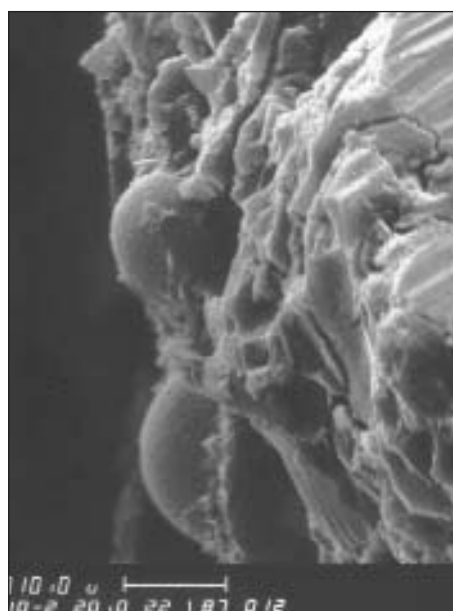


Fig. 11.2. A SE micrograph showing a section through a glass-ionomer that has been mixed with a spherical amalgam alloy. Note the rounded alloy particles that are surrounded by the glass-ionomer.

Tartaric acid

To control the setting reaction, small quantities (5-15%) of the optically active isomers of tartaric acid are added to the liquid. This assists the extraction of ions from the glass powder, retains the working time and sharpens the setting time. It also allows the use of lower fluoride-containing glasses which are more translucent, thereby improving the aesthetic characteristics of the set cement.

Anhydrous forms

The term 'anhydrous' has been suggested for some varieties inferring that water is not part of the liquid. However, it is a misnomer in as much as the glass-ionomers are a water based cement and water is an essential component of all types. In this particular group the polyacrylic acid, normally used as the liquid, has been vacuum-dried and premixed with the glass powder so the liquid, which is then incorporated during mixing, is either water or a dilute aqueous solution of tartaric acid. On mixing, these cements have a relatively low viscosity and are particularly suitable for luting or lining purposes. Another advantage is that a polyacrylic acid of higher molecular weight can be used in a dehydrated form and this can lead to moderately improved physical properties.

Modifications to the basic formula

Since the introduction of the original autocure glass-ionomers in 1976 there have been a number of variations developed with the original intention of strengthening the ultimate restoration, increasing its resistance to early water uptake, modifying its chemical properties and enhancing translucency.⁶ Some of these have been successful and remain in common usage but others interfere with the ion exchange mechanism to such an extent that they can no longer be included within this class of materials. The following are some of the materials currently on the market.

Resin-modified glass-ionomer

These materials consist of the components of a glass-ionomer, as described above, modified by the inclusion of a small quantity of additional resin – mostly HEMA – as well as photo initiators

and traces of other resins and reactive chemicals.⁷ They set partly by an acid/base reaction and partly by a photochemical polymerisation. In addition, in some materials, the polymerisation of the resin component also contains a chemical initiator mechanism similar to that used in the old autocure composite resins. These are known as 'dual' or 'tri' cure materials.

The liquid contains 15-25% resin component in the form of HEMA as well as less than 1% polymerisable groups and a photoinitiator. Depending upon the powder/liquid ratio used in the mix there will be a residual HEMA content in the set cement ranging from 4.5%, where there is a high powder content, to possibly 15% in a thinly mixed lining cement. As HEMA is hydrophilic there is a potential for water uptake and subsequent degradation with release of HEMA into the surrounding dentine.

In the first stages of the setting chemistry, the acid/base reaction between the glass and the polyalkenoic acid, will still take place as described below but the moment light activation is undertaken the resin component will polymerise and the risk of water contamination will be eliminated. The polyacrylate chain formation will continue in the same fashion as the regular autocure glass-ionomer but at a slower rate under a protective 'umbrella' of set resins. In the long run, any resin not set by light activation will set by autocure, because of the presence of an incorporated reduction/oxidation (redox) reaction, and achieve the same physical properties. This means that, even in the absence of light activation, these materials will set completely over time. It has been shown that their physical properties are not downgraded if allowed to autocure only.⁸

In some of the resin-modified materials a small quantity of the light activator catalyst is incorporated in the powder, thus making the powder susceptible to ambient light. Always follow the manufacturer's instructions and do not dispense either the powder or liquid on to the mixing slab until immediately prior to mixing. This is not a problem if the materials are encapsulated.

Resin-modified autocure material

Variations on the resin-modified materials include ones which are either fully autocure or else subject to a combination of light/heat plus autocure.⁹ For these materials the powder component will generally be very finely ground although the particle size will vary according to the proposed function. There will also be a small quantity of a catalyst incorporated that is designed to stimulate the autocure reaction of the resin component. The liquid will contain the usual polyalkenoic acid, tartaric acid, water and HEMA as well as a small amount of a cross-linkable monomer. Some products also claim to contain a polycarboxylic acid modified with pendant methacrylate groups.

Following mixing of the powder and the liquid the usual acid/base setting reaction will be initiated. At the same time the catalyst in the powder will initiate polymerisation of the HEMA and the cross-linkable monomers. Ultimately there will be cross-linking between the two systems and the entire mass will set hard, with uniform physical properties, in spite of the fact that there has been no light activation at all.

Most of this group are luting or lining materials and are of value where it is not possible to reliably light initiate the setting reaction. They have a higher flexural and bonding strength and may be particularly useful for orthodontic bracket and band cementation. At present, the materials already on the market have relatively low physical properties commensurate with the designated tasks to be performed, but there is a probability of applying the same principle with stronger materials, such as restorative materials, in the future.

Light-initiated autocure material

Another variation incorporates a method for speeding up the setting reaction of the autocure materials. This involves enhancing the speed of the acid/base setting reaction by utilising a simple physical principle. If a dye is incorporated in a glass-ionomer and the colour is specified such that the L^* value, when expressed by an $L^*a^*b^*$ colorimetric system in a standard light D_{65} is 60 or less, the curing reaction will be further promoted and the setting reaction will be accelerated. Thus if a glass-ionomer is coloured red, then mixed,

placed and irradiated by the standard blue dental halogen activator light, the acid/base setting reaction, which is already under way, will take place more rapidly. The material can now be regarded as light initiated but there is a complete absence of any added resin. Setting time will be dramatically reduced but there will be no significant generation of heat and the physical properties of the set material will not be downgraded in any way.

This factor has recently been incorporated into a low powder/liquid ratio glass-ionomer to which has been added a small quantity of a red pigment. As an additional benefit it appears to be highly bactericidal due to a high fluoride release. Due to its rapid command set, it can be recommended for a number of applications such as fissure protection, root surface protection or as a lining material.

Polyacid-modified composite resin – ‘Compomers’

There is a further group of materials that contain either or both of the essential components of a glass-ionomer but at levels insufficient to promote the acid/base setting reaction in the dark, i.e. the immediate acid/base reaction is not available because it is overwhelmed by the amount of resin present.¹⁰ These materials will not set without light activation. They do not belong in the glass-ionomer category and have been classified as polyacid-modified composite resins. Commonly called ‘compomers’ they can be quite useful materials as long as their limitations are recognised.¹¹ It is not possible for a compomer to bond to tooth structure through an ion exchange mechanism because the acid/base reaction does not occur for some time after placement. This means that placement of a restoration has to be preceded by an application of a resin bonding agent. For the same reason the ion exchange mechanism of the glass-ionomers, which is so useful for remineralisation, is not available to tooth structure, although there is a small fluoride release that can be detected some time after placement.

The critical difference which distinguishes glass-ionomers from polyacid-modified composite resins (compomers), is the ability of the glass-ionomers to set in the dark in a clinically acceptable time through the normal acid/base reaction.

Clinical test to distinguish 'resin-modified glass-ionomer' from 'poly-acid modified composite resin'

For the clinician to differentiate between the resin-modified glass-ionomers ('dual' or 'tri' cured) and a polyacid-modified composite resin there is a simple test that can be carried out in the clinic. Mix a sample of the appropriate material and place a small quantity on to a glass slab for testing. Immediately after mixing place it under a light proof cover and test it at brief inter-

vals to check the degree of set. The true glass-ionomer material will be reasonably hard to touch within seven minutes. The other materials will not set at all although a few may achieve a rubbery consistency within 15 minutes. But they will not set beyond that without being subject to light activation.



This is a property that applies equally with both glass-ionomers and resin-modified glass-ionomers but not with compomers.

Giomers

A recent addition to the range of materials has been classified as a 'giomer'. This contains pre-polymerised glass-ionomer particles, in a range of sizes, supported by a light activated resin vehicle. As the vehicle is resin the ion exchange potential will not be available to surrounding tooth struc-

ture and the material clearly falls in to the same group as the compomers.

Potential modifications

Research continues and the potential for further modifications is broad.^{12,13} It must be noted that, to date, there has been little variation in the polyalkenoic acids used in these materials and also no great variation in the formula for the glasses. Both sides of the basic formula are subject to considerable variation in as much as there

are some hundreds of different kinds of the polyalkenoic acid available, and in the formula for the glass melt there are a number of metals that could be tried in different combinations and concentrations.

Considerable effort has been expended in the search for inclusions that will enhance fracture resistance but so far without success. Glass or resin fibres have been tried but, as with silicate cements, collateral problems appeared such as an inability to develop and retain a smooth surface on the restoration. Recently experiments have been carried out to enhance the therapeutic value by the inclusion of antibacterial agents,¹⁴ or remineralising agents such as CPP-ACP¹⁵ (**Chapter 8**). Results are so far equivocal and the real value of such inclusions is questionable in the light of the already valuable ion exchange properties that should not be inhibited or affected.

The primary object in further research should be to increase fracture resistance because, even in the best materials currently available, this property is less than ideal. However, it is essential that the original ion exchange potential be not interfered with or reduced. The ion exchange with tooth structure is of immense value because it is the only material that will ensure a union which is proof against microleakage between restoration and tooth structure. It is therefore possible to seal a lesion and isolate it from further ingress of bacteria and/or their nutrients. In addition, the ion release has now been shown to enhance the remineralisation of tooth structure that has been ravaged by caries thus allowing the use of minimal intervention cavity preparations and preservation of natural tooth structure, even though demineralised (**Chapter 16**).

Methods of dispensing

The first materials marketed were provided as a powder and a liquid and required mixing on a glass slab or paper pad. A small spoon was used for measuring the powder and the dropper bottle was intended to dispense a standard sized drop of liquid. However, it was apparent from the beginning that dispensing with the high degree of accuracy required was not possible, particularly when

the person responsible was under the normal pressure of clinical practice. It was not long before the manufacturers developed methods of capsulation for accurate standardised dispensing and machine mixing.

Powder/liquid

Physical properties are heavily dependent upon the powder content and for optimum results full and correct measure is imperative. A small spoon is provided by the manufacturer to dispense the powder. Instructions specify that the spoon be filled to capacity and the excess be wiped off the top using a lip at the top of the bottle. Laboratory testing of this method shows that, no matter how carefully the operator tries, there will be a variation as much as 10% either way in the weight dispensed. This is clinically unacceptable. If hand mixing is to be reliable it is necessary to weigh the powder on a set of scales accurate to two decimal points.

The bottle of liquid is provided with a dropper mechanism designed to accurately dispense one drop at a time. It must be noted that the first drop out of a bottle is always deficient and should be discarded. Subsequently, if the liquid to be dispensed is a rather viscous polyalkenoic acid, it is essential to turn the bottle on its side first while the liquid flows into the dispensing tip. Hold briefly, turn vertical and only then dispense the drop. Any drop that contains an air bubble should



Fig. 11.3. The three commonly used capsule systems for glass-ionomers. All show a high degree of accuracy in dispensing both powder and liquid.

be discarded. Again, the best way is to weigh the drop on the scales and then dispense the amount of powder that will give the right P/L ratio with that drop.

With some materials the liquid is water or a dilute tartaric acid. This is easier to dispense with a dropper bottle but must still be done with care. Simply because it has a low viscosity it is easy to dispense more drops than required.

Capsules

There are now several designs of capsule available and all offer accurate proportioning of the powder and the liquid (**Figure 11.3**). Laboratory experiments show a high level of reliability in both the powder and liquid proportions. Most of the capsules are designed to be both a mixing capsule as well as a dispensing syringe and placement with these allows for considerable accuracy. Each manufacturer will issue their own instructions for storage, mixing and dispensing. However, in general, it is desirable to gently disturb the powder in the capsule by tapping on the bench top before placing the capsule in the mixing machine. When activating the capsule to admit the liquid into the powder, take great care to follow the manufacturer's instructions and to activate the capsule completely.

Paste/paste

There is a further variation in dispensing now available where the material is presented as a two



Fig. 11.4. One of the double syringe systems used to dispense the paste/paste systems for glass-ionomers. Equal lengths can be dispensed with accuracy while the quantity can be varied.

paste system packaged in a twin syringe apparatus (**Figure 11.4**). The main advantage is that there will always be the correct proportions of both components dispensed on to the slab where it will then be handmixed. It is now possible for the operator to prescribe variations in quantity to be mixed without any problems in maintaining the quality of the final mix. At this point these systems are used for luting cements, lining cements, and for endodontic and orthodontic applications only.

They generally have a finer particle size and therefore an ultimate film thickness in the range of $5\ \mu$ and a setting time within three minutes. There is a small quantity of resin included as well as of traces of a catalyst that is required to bring the setting reaction of both the resin and the glass-ionomer to completion but the ion exchange mechanism with tooth structure remains unaffected.

Mixing

Handmixing

Handmixing is possible for all types of cements, although great care is required when measuring out to avoid under or over dispensing. It is not possible to reliably vary the size of a drop of liquid and it is difficult to accurately hand dispense a spoon of powder, so it is not possible to dispense a partial mix in an attempt to conserve material.

The principal object during handmixing is to wet the surface of each glass particle without completely breaking down and dissolving the powder in the liquid. The strength of the set cement lies in the remaining glass particles rather than in the matrix. Therefore the mix should be undertaken quickly on a cool dry glass slab, or a paper pad, without spreading the mix around or spatulating heavily. Dispense the liquid immediately prior to mixing in the centre of the slab and spread it out over a small area. Do not allow it to stand exposed to air for longer than necessary because it will either lose or take up water depending upon ambient conditions. Measure the powder carefully on to the slab then divided the powder in half so it can be mixed in two parts. The first part should be incorporated by gently but rapidly rolling the powder into the liquid within ten seconds. Then

Handmixing



Fig. 11.5 (left). For handmixing take care to be accurate when dispensing. The bottle of liquid should be turned on its side and held for a moment to allow the viscous liquid to enter the dispensing tip. Then turn vertical and dispense a drop. If the drop contains an air bubble then discard and try again.

Fig. 11.6 (below left). Divide the powder into equal parts with the spatula. Spread the liquid drop across a small area of the pad and draw in one half of the powder for mixing.

Fig. 11.7 (below right). By 10 seconds the first part of the powder should be fully mixed. Draw in the second half and complete the mixing.

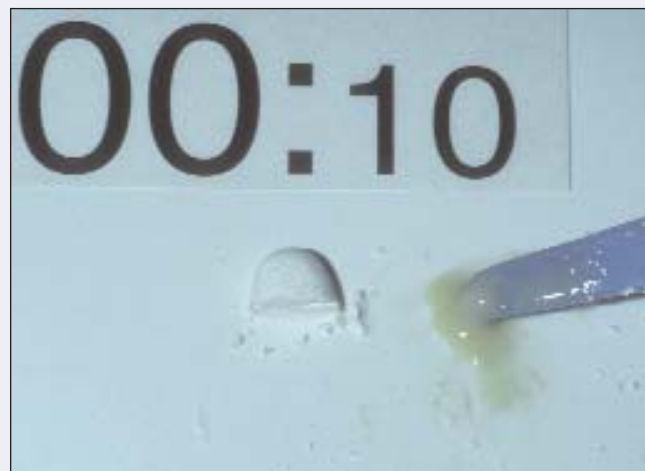
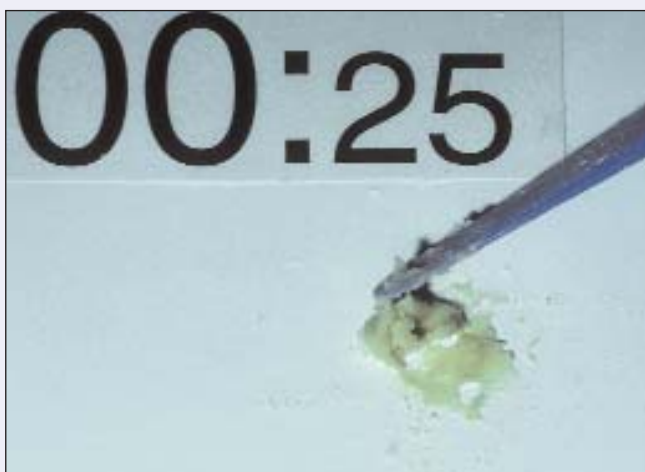


Fig. 11.8. By 25 seconds the mix should be complete. Do not spend more time testing it because it is already starting to set.

Fig. 11.9. Draw the mix up in to a disposable syringe tip and dispense it in to the cavity as soon as possible.



Fig. 11.10. A quantity of the glass-ionomer powder with enhanced wettability has been dispensed on to the pad and a drop of liquid placed in the centre. Courtesy 3M/ESPE.

include the second part entirely leaving no residue and finish the mixing within a further 15 seconds. The finished mixed material should be 'glossy wet' on the surface and the working time should now be between 60-90 seconds. For convenience it should be transferred to a disposable syringe for accurate and positive placement into the cavity.

Improved wettability

One of the original problems with hand mixing was the relative reluctance of the liquid to actually wet the powder particles. For example, if a drop of liquid is placed on the top of a pile of powder (**Figure 11.10**) it will show no sign of absorption, so some degree of persuasion is required and mixing by hand becomes tedious. It has now been shown by one manufacturer that agglomeration of the particles and elimination of the dust from the powder will improve the wettability of the powder and reduce this problem considerably (**Figure 11.11**). As a result of the internal porosity of the granulated particles the liquid is absorbed immediately and, while the granulate is strong enough for transport, it will break down rapidly with mixing. There appears to be some minor improvements in physical properties and it is possible to achieve a suitable mix in less than half the normal time. It is suggested that there would be some advantages to be gained from the use of a granulated powder in areas where machine mixing is not available.



Fig. 11.11. Almost immediately the liquid disappears into the powder thus making it easier to mix by hand. Courtesy 3M/ESPE.

Machine mixing

Quality control in the manufacture of the capsules is generally very high but care must be taken in activating a capsule to ensure the full release of the liquid into the chamber containing the powder. Pay careful attention to the manufacturer's directions for machine mixing. The time of mixing will vary from one manufacturer to another and there may well be variations in the consistency of the energy provided by different mixing machines. The energy of a mixing machine is likely to decline with age and once in a while the efficiency should be tested as discussed below. Mixing time can be varied to a small degree, either up or down, but it is unwise to rely upon changing the time of mixing. Rather, it is far better to maintain the efficiency of the mixer.

Paste/paste

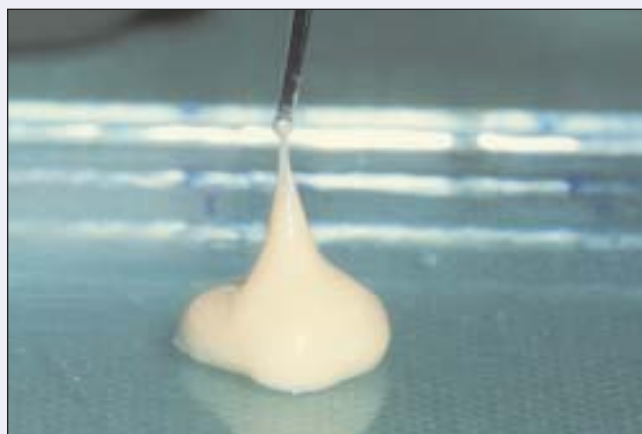
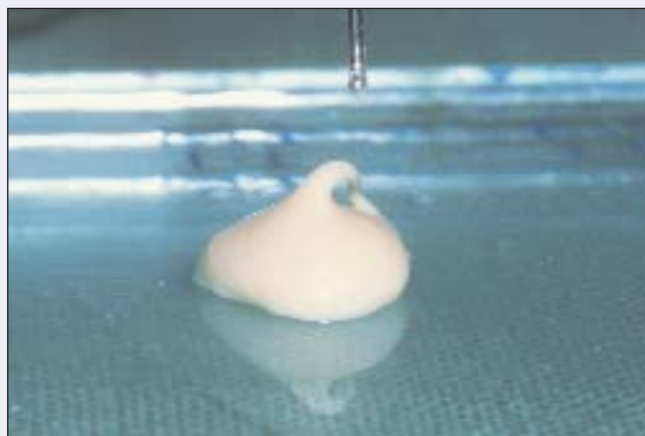
The paste/paste materials will logically be hand-mixed on either a glass slab or a paper pad. As with all handmixed materials reduction of the temperature of the glass slab to just above the dew point will allow for an extended working time but care must be exercised to avoid incorporating additional water into the mix. If the slab is stored in the refrigerator then it should be wiped dry before placing on to the bench and then checked again for further deposition of dew before dispensing the cement. Mix the paste/paste materials quickly and thoroughly with a flexible spatula and complete the mix within 10-15 seconds. Apply

Loss of gloss/slump test¹⁶

Learn to recognise a properly mixed capsulated specimen by carrying out a 'loss of gloss/slump test'. Make a trial mix and express the cement on to a glass slab. Lift the top of the pile of cement with a small instrument until the cement breaks and slumps back on to the slab. There will come a point where the

cement breaks but does not slump. Measure that time and subtract 30 seconds to determine the working time.

Normally this should be between 60 and 90 seconds but for the resin-modified glass-ionomers it will be nearer to three or three and a half minutes in the absence of light activation.



to the cavity using a disposable syringe for preference.

Setting reaction of autocure materials

The chemistry of the setting reaction of a glass-ionomer is very important if full advantage is to be gained from the bioactivity which is inherent with this group of materials.^{17,18} The true autocure glass-ionomer will undergo a simple acid/base reaction which leads to a diffusion based adhesion between the glass particles and the matrix. This

process must be sufficiently complete within a clinically acceptable time period.

Calcium and aluminium ions are released by proton attack on the surface of the glass particles and these ultimately cross-link the polyacid chains into a network. This is a relatively open network that will remain porous allowing free passage of hydroxyl, fluoride and other ions out of and back into the cement matrix. Three stages have been described in this setting reaction which is regarded as a long-term chemical exchange continuing for at least one month after placement

and probably much longer. The calcium chains form first, producing a clinically acceptable early set, but they are relatively fragile and highly soluble in water. Their presence is the key to translucency. The aluminium chains form thereafter and are strong and insoluble and provide the major physical properties of the set restoration. With the early versions of the glass-ionomers, the matrix formed in the initial stages of the setting reaction was predominantly based upon calcium and was therefore highly susceptible to both water loss or water uptake. The restoration was regarded as unstable and had to be immediately sealed from water uptake. However, the speed of setting has been modified by removal of excess calcium ions from the surface of the glass particles and modern autocure glass-ionomers are based mainly upon aluminium and are far more tolerant of early exposure to water. However, it is still wise to protect most of them with a waterproof sealant immediately after placement.

The setting mechanism involves the dissolution of the surface of the glass particles with the release of ions and can be divided into three clearly defined stages (**Figures 11.12 and 11.13**).

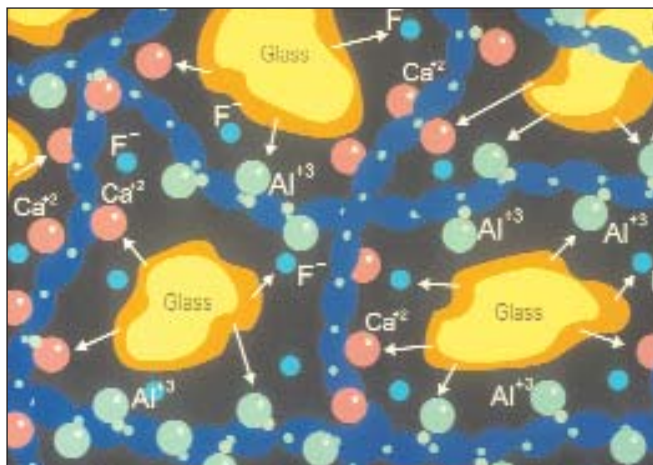


Fig. 11.12. A theoretical diagram of the initial interaction between the powder and the liquid of any of the glass-ionomers. The polyalkenoic acid immediately attacks the surface of each of the powder particles releasing both calcium and aluminium ions, as well as some phosphate ions.

Three stages of the setting reaction

Stage 1: Dissolution

The surface layer of the glass particles is attacked by the polyacid to produce a diffusion-based union between the two. Approximately 20-30% of the glass is decomposed and ions, including calcium/strontium, aluminium and fluoride, are released leading to formation of a cement sol.

Stage 2: Precipitation of salts – gelation/hardening

During this stage, calcium/strontium and aluminium ions bind to polyanions at the carboxylate groups. The initial clinical set is achieved by cross-linking of the more readily available calcium ions. This reaction is relatively rapid, usually forming a clinically 'hard' surface within 4-10 minutes from the start of mixing. Maturation occurs over the next 24 hours as the less mobile aluminium ions become bound within the cement matrix leading to more rigid cross-linking between the poly(alkenoic acid) chains. Fluoride and phosphate ions form insoluble salts and complexes. Sodium ions contribute to the formation of an orthosilicic acid on the surface of the particles and, as the pH rises, this converts to a silica gel which assists in binding the powder to the matrix.

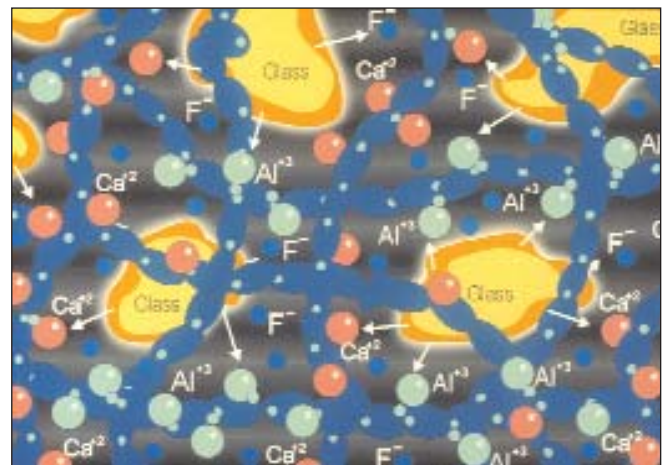


Fig. 11.13. A later stage showing the increasing complexity of the calcium and aluminium polyacrylate chain formation as well as the early stages of the generation of the silica gel on the surface of all particles which helps to bind the particles together.

Stage 3: Hydration of salts

Associated with the maturation phase is a progressive hydration of the matrix salts, leading to a sharp improvement in the physical properties.

Rate of setting

The rate of setting depends on a number of manufacturer controlled variables, such as glass composition, temperature at which glass fusion occurs, temperature at annealing stage, powder particle size and size distribution, tartaric acid concentration, the composition of the liquid, and the possible inclusion of a resin component and photoinitiators. The rate is also dependent on several operator controlled factors, such as

- **mixing temperature**

Storage of the mixing slab and the bottle of powder in a refrigerator increases the working time by up to 25%. The liquid must not ever be refrigerated because it is likely to gel.

- **powder: liquid ratio**

Although a higher powder content will yield improved physical properties, sufficient liquid must be present to wet the surface of all the powder particles. Inadequate liquid

results in a decline in translucency as well as a decline in physical properties. Variations in the ratio of powder to liquid will alter clinical handling characteristics. A luting cement, for example, will be mixed with a lower powder content because it needs to be able to flow sufficiently to allow a fine film thickness between the restoration and the tooth. However, physical properties will be lower.

BE AWARE



Refrigerate powder and mixing slab to extend working time.

NEVER refrigerate the liquid – it will gel.

Setting reaction of resin-modified glass-ionomer

In theory, two distinct types of curing/setting reactions take place in the resin-modified glass-ionomers (**Figures 11.14 and 11.15**):

- the acid/base neutralisation reaction identical with the autocure
- free radical methacrylate cure

The relationship between these two reactions may take one of two forms:

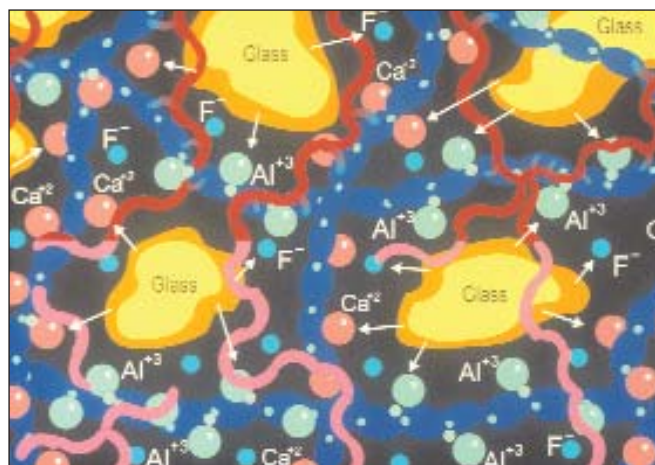


Fig. 11.14. The setting reaction of the resin-modified glass-ionomers. The chemistry is essentially the same as the standard glass-ionomer but there is a resin framework being generated over and around the glass-ionomer matrix. This will seal the process against short-term water uptake or water loss. Note that resin polymerisation will occur to the depth of light penetration only.

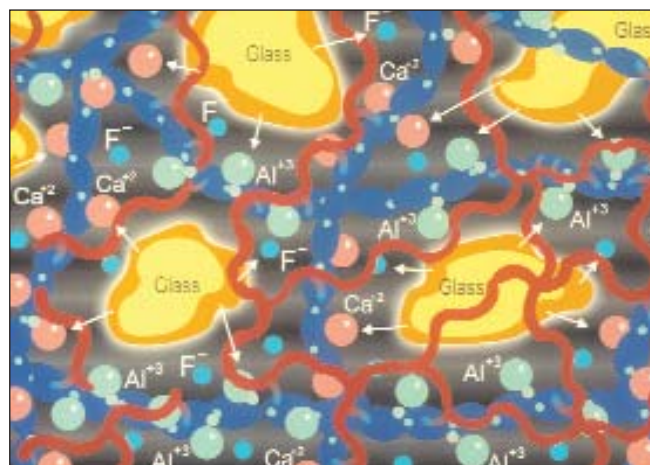


Fig. 11.15. The later stages of the setting reaction showing completion of the resin polymerisation as a result of the autocure reaction.

- formation of two separate matrices – an ionomer salt hydrogel and a polyHEMA matrix. This system may cause complete inhibition of the acid/base reaction,
- multiple cross-linking – pendant methacrylate groups may replace a small fraction only of the carboxylate groups of the polyalkenoic acid thus preventing the separation of the two potential matrices.

Cross-linking of the polymer chains may then take place through one or more of the following reactions:

- acid/base reaction
- presence of a photoinitiator (camphorquinone) i.e. light cure mechanism
- reduction/oxidation reaction (redox) i.e. resin autocure mechanism

Following mixing and placement of the material, application of the activator light will result in rapid hardening to the depth of penetration of the light. There will be photo cross-linking of both the methacrylate groups of the polymer as well as the HEMA and the restoration can be regarded as clinically set. However, full physical properties will not be achieved for some days while the acid/base reaction continues in the same way as for the autocure materials, although probably at a reduced rate.

BE AWARE



The setting chemistry is slow with all types

- autocure will snap set in three minutes then continue to full set over next one month
- resin-modified will snap set with the activator light then continue to full set over next one month

It must be noted that initial light activation will lead to hardening only to the depth of light penetration. This suggests that it is desirable to build a restoration incrementally to ensure complete polymerisation. On the other hand the autocure component will ensure that the restoration will be fully cured in the long run so this aspect is not as important as it is when placing composite resin restorations.

Structure of the set glass-ionomer

The set material contains particles of unreacted glass, surrounded and supported by a siliceous hydrogel, embedded in a polysalt matrix of cross-linked polyalkenoic acid molecules rich in calcium and aluminium ions.¹⁹ The set cement contains water, both bound in and free, so it is regarded as a water based cement. Ion migration can only occur in the presence of water so this material is ideal to allow for the transit of ions both inwards and outwards within the oral environment. In particular, ions with small dimensions such as hydroxyl and fluoride ions will move both ways through the material and will be available for remineralisation of damaged tooth structure.

NOTE



- ion migration can only occur in the presence of water
- glass-ionomer is the only water based restorative material

In the early stages, immediately following mixing, the calcium polyacrylate chains can take up further water and be washed out and lost. On the other hand if the cement is exposed to air it may lose some of the unbound water and dehydrate, losing both physical integrity and strength. These problems can be modified at the time of manufacture by washing off some of the excess calcium ions from the surface of the glass powder thus producing a relatively fast setting cement. However, as the calcium polyacrylate chains are essential for translucency in an aesthetic restoration, the fast set materials generally are not as aesthetic as the slower setting autocure materials. As discussed below the alternative is to apply a water proof seal over the restoration immediately after removal of the matrix to maintain the water balance.

Water balance

Once the material is set the water content is arbitrarily classified as either 'loosely bound water' or 'tightly bound water'. The loosely bound water can be lost quite readily in the early stages if the

cement is left exposed to air but the tightly bound water cannot be removed at all. The latter is associated with the hydration shell of the cation-polyacrylate bond, particularly that of aluminium and, to a degree, some of the silica gel water as well.

Successful management of the water balance of the glass-ionomer material is probably the most significant factor in ensuring long term success with the autocure cements.²⁰ Approximately 24% of the set glass-ionomer is water and susceptibility to water uptake and loss is summarised in **Figures 11.16** and **11.17**. The cement is most susceptible to moisture contamination during Stage 2 of the setting process before the calcium/strontium ions have been locked in to the resistant gel matrix. It also remains susceptible to water loss for some time after placement. The period of vulnerability to water loss and water uptake will vary with the material and considerable research has been undertaken to minimise the problem. Most of the present materials and particularly the resin-modified glass-ionomers will be resistant to water uptake within minutes of placement but they all remain susceptible to water loss for anything up to two weeks.²¹

BE AWARE



The water balance is critical for all glass-ionomers

- water in is immediately critical for autocure aesthetic cements
- water out is critical for six months for autocure aesthetic cements
- water in is less critical for resin-modified and all others
- water out is critical for two weeks for all cements

The best method for protecting the cement from either water loss or water uptake is to paint a liberal coating of a single component, light activated, resin enamel bond over the restoration immediately it is set.^{22,23} Most manufacturers supply the appropriate material. Trim any excess cement through the seal and apply more resin bond if required and then light activate it to create the seal. This will maintain isolation for about 24 hours and thereafter the water balance will be stable. Note that not all the glass-ionomers are equally unstable (**Figure 11.18**).

Except for the slow set aesthetic materials, all types of autocure cements have been modified during manufacture by removing some of the excess calcium/strontium ions thereby limiting



Fig. 11.16. Two early ASPA restorations on the labial of the upper central incisors. It is apparent that both are faulty as a result of failure to seal them properly leading to water uptake with loss of translucency. In addition the restoration in the right central incisor was placed first and allowed to dehydrate while the other restoration was placed. Hence cracks from water loss.



Fig. 11.17. The restorations shown in **Figure 11.16** have been replaced with new restorations of another glass-ionomer. They were properly sealed immediately the matrix was removed to maintain the water balance. They had been in place for eight years at the time they were photographed.

the formation of calcium polyacrylate chains. This reduces their susceptibility to water so that they are fast setting, and therefore, resistant to water uptake at about four minutes after mixing. There is at least one material that is so fast setting that it is best exposed to water immediately after it has achieved its preliminary set because it appears to require additional water to achieve full strength.²⁴

The main limitation is that these fast setting materials lack translucency although they do have superior physical properties. It must be noted that they remain susceptible to water loss, and therefore cracking, for about two weeks after

placement. If, in that period, the cement is likely to be exposed to air for more than five minutes it should be sealed with a low viscosity resin sealant as discussed above.

Modifications to allow for light initiation of the glass-ionomers mean that they can then be regarded as a fast set cement with immediate resistance to water uptake and some protection against water loss.²⁵ The addition of 5-15% HEMA, as well as light activation catalysts, offers protection to the calcium/strontium polyacrylate chains against dissolution in water. Meanwhile the usual acid/base reaction continues. However, because of the HEMA, the resin-modified materials seem to perform like mild hydrogels with rapid water uptake over the next 5-7 days which can lead to swelling and some possible stain uptake. Following adequate light activation the material can immediately be contoured under air/water spray at intermediate high speed without interfering with the setting reaction. But it should be noted that it is still possible to dehydrate the restoration any time over the next two weeks. Also it is logical to seal the surface of the newly placed and contoured restoration with a low viscosity resin sealant because it will fill any exposed porosities and scratches and leave a smoother surface for patient comfort and aesthetics.

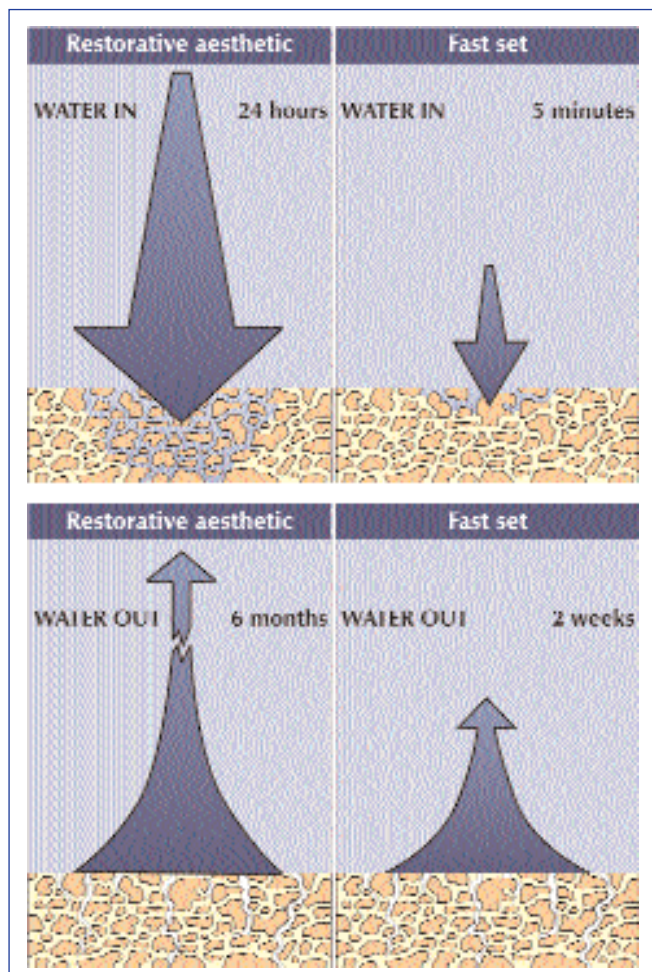


Fig. 11.18. Water balance: The autocure glass-ionomer cements are susceptible to water uptake and water loss during the early setting stages. The restorative aesthetic cements, in particular, need to be protected because water uptake will severely reduce the degree of translucency of the set cement as well as reduce physical properties.

Adhesion to tooth structure

Bond to mineralised tissue

One of the most important characteristics of glass-ionomer materials is their ability to adhere chemically to mineralised tissues (**Figures 11.19-22**). The probable mechanism is based upon both diffusion and adsorption phenomena²⁶ and is initiated by the polyalkenoic acid when freshly mixed material, that still contains available unreacted acid, contacts tooth surface. Phosphate ions will be displaced from the apatite by carboxyl groups, each phosphate ion taking a calcium ion with it to retain electrical neutrality. The setting of the glass-ionomer, combined with the release of mineral ions from the surface of the enamel or dentine, leads to buffering of the polyacid, a rise in the pH and reprecipitation of minerals at the cement/tooth interface. It appears that chemical

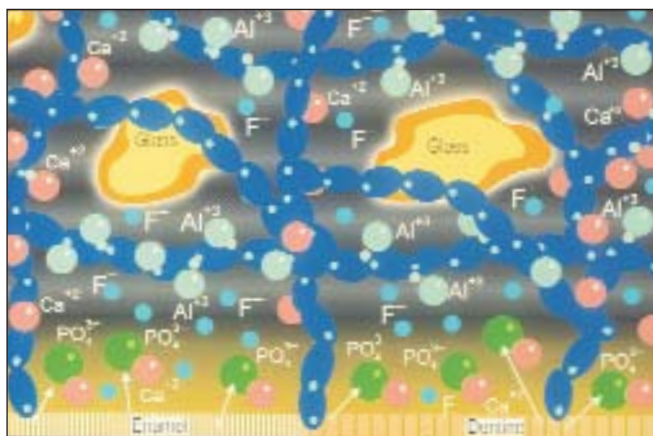


Fig. 11.19. A theoretical diagram showing the ion exchange system of adhesion of glass-ionomer to tooth structure, both enamel and dentine. The free polyacrylic acid that is still available from the newly mixed glass-ionomer displaces calcium and aluminium ions from the cement and calcium and phosphate ions from tooth structure. These form a new material which, as it sets, binds the two together.

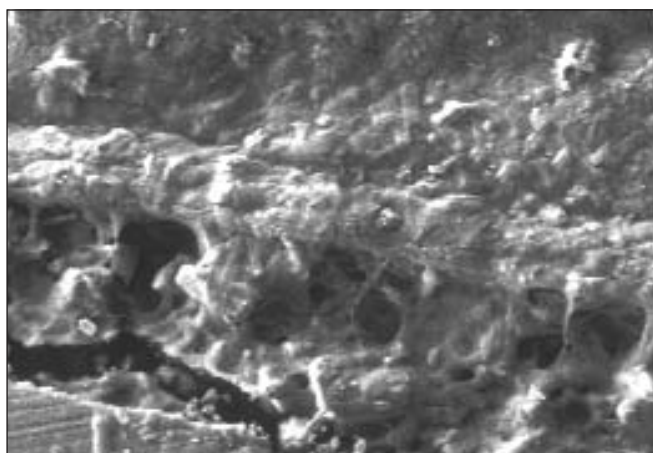


Fig. 11.21. The same specimen at a higher magnification shows the ion exchange layer more clearly. The glass-ionomer is below and tooth structure above.
Courtesy Dr H. C. Ngo.

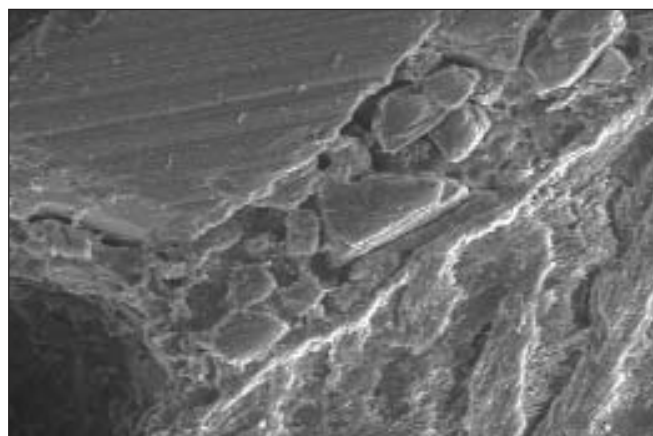


Fig. 11.20. A scanning electron micrograph showing a specimen of a restoration adhering to a tooth on a cryo-vac stage. Because it is deep frozen the water balance is maintained and the specimen does not crack. The surface has been lightly etched so the ion exchange layer remains slightly dominant and clearly visible. Glass-ionomer above, enamel below.

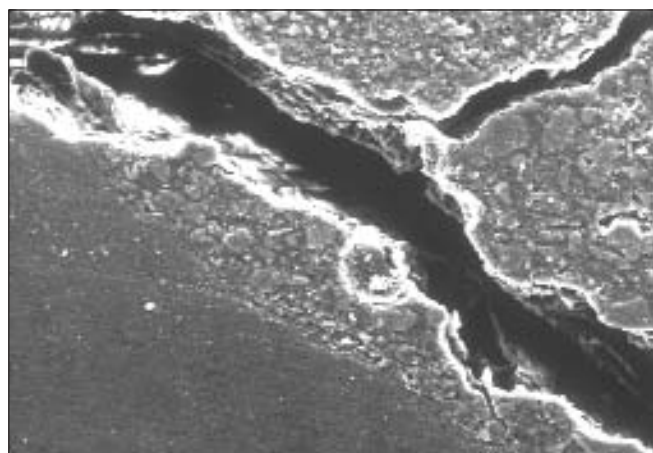


Fig. 11.22. A specimen viewed under the SEM without the cryo-vac stage. There has been dehydration leading to cracking with part of the restoration remaining attached to the tooth. This is the normal mode of fracture leading to loss of a glass-ionomer with the ion exchange layer staying behind on the tooth surface.

bonding is therefore achieved through the combined calcium/strontium phosphate/polyalkenoate crystalline structure acting as an interface between both enamel or dentine and the set material. This can be described as a 'diffusion-based adhesion'.²⁷⁻²⁹

Bond to collagen

Adhesion to the organic component of the dentine may also occur through either hydrogen bonding

or metallic ion bridging between the carboxyl groups on the polyacid and the collagen molecules of the dentine.³⁰

It has been suggested that there will be a level of micromechanical attachment between the tooth structure and the resin-modified glass-ionomers. However, as the total content of resin in the mixed material is less than 5% this is unlikely to make a significant contribution to the overall strength of adhesion. Some researchers have

demonstrated penetration into dentine tubules in a manner similar to composite resin. However, the tensile strength of any material in the tubules will be quite low so this is unlikely to play any part in the retention of the restoration.

The strength of the union has not yet been measured because failure under normal circumstances is cohesive in the cement leaving the ion exchange layer firmly adherent to the underlying tooth structure. As the ion exchange layer is always present, tests purporting to reveal the strength of adhesion will reveal the tensile strength of the relevant cement rather than the adhesion.

Conditioning the tooth surface

Logically, adhesion will take place best in a clean environment. Various agents have been proposed to remove some or all of the smear layer and possibly pre-activate the enamel and dentine in preparation for adhesion. Low molecular weight acids such as citric acid or hydrogen peroxide were recommended initially but the most desirable material has proven to be a low concentration polyacrylic acid, applied for a brief period and then washed thoroughly from the tooth surface.³¹ Polyacrylic acid is a part of the glass-ionomer system and therefore any remaining residue will not interfere with the setting reaction. Both glass-

ionomer and the tooth structure have a high surface energy and an application of 10% polyacrylic acid will lower the surface energy of the tooth and thus increase the wettability of the surface and encourage the adaptation of the cement to the tooth. It is a mild acid with a large molecular weight and will not demineralise the tooth surface unduly nor penetrate the dentine tubules. Following an application time of ten seconds there will still be plugs in the dentine tubules that will discourage dentine fluid flow and help to keep the cavity relatively dry until the restoration is in place (**Figures 11.23 and 11.24**).

NOTE

- Conditioning is essential to prepare the cavity surface
- **NEVER** etch the tooth because it will reduce the potential for ion exchange

An alternative to removal of the smear layer is to apply a mineralising solution to the tooth surface such as ITS solution³² or 25% tannic acid and leave it in place for 1-2 minutes before washing thoroughly. The smear layer will now be included in the ion exchange layer and will not interfere with the adhesion.

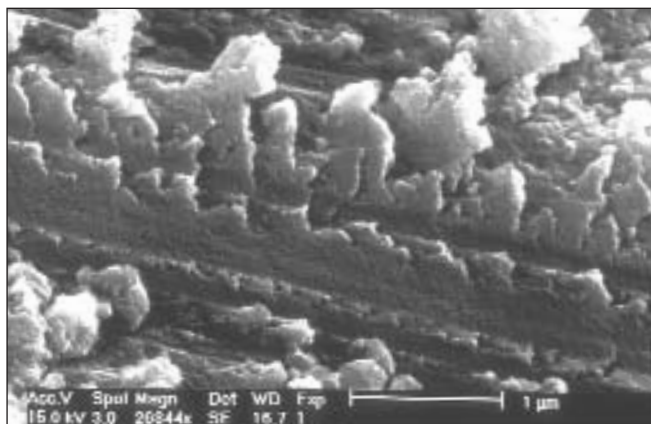


Fig. 11.23. A scanning electron micrograph showing the smear layer remaining on the floor of a cavity following cavity preparation.

Courtesy Dr H. C. Ngo.

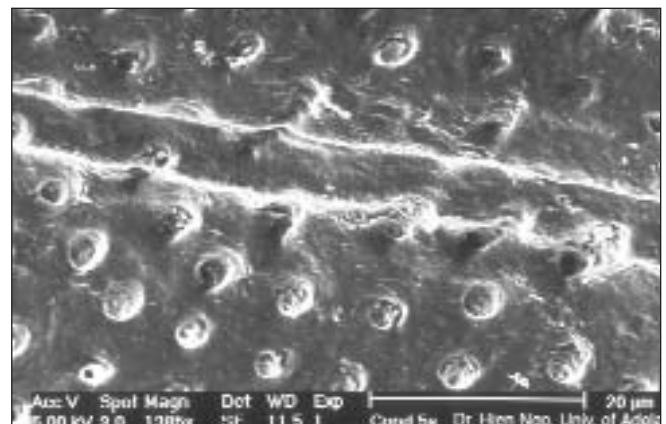


Fig. 11.24. A scanning electron micrograph showing the dentine surface following conditioning with 10% polyacrylic acid for 10 seconds. The smear layer is removed but the dentine tubules remain relatively closed over thus limiting the dentine fluid flow. Courtesy Dr H. C. Ngo.



Fig. 11.25. There are sensitive erosion lesions at the gingival margins of both the upper right lateral and canine. It is proposed to restore these using a Type II.1 restorative aesthetic glass-ionomer. A soft tin matrix is bent to place first and set to one side ready for use.



Fig. 11.26. The area for adhesion is conditioned with 10% polyacrylic acid for 10 seconds then washed and lightly dried. It is permissible to scrub lightly with pumice and water prior to this but it is not necessary and the risk is that the gingival margin will be abraded and will then bleed.



Fig. 11.27. The glass-ionomer is machine mixed, syringed to place and the soft tin matrix is used to gently push the cement on to the tooth and provide a suitable contour. The matrix is left in place until the excess cement has been shown to be set hard.



Fig. 11.28. As soon as the cement is set, the excess is broken away, the matrix is removed and the restoration is immediately coated with a resin enamel bond. Any further contouring can now be undertaken as required and the resin seal can be light activated to provide a completely water tight seal for at least 24 hours.



Fig. 11.29. The two restorations photographed one week later. Note the level of translucency and the successful aesthetic result.



Fig. 11.30. The restorations shown in the previous series photographed eight years after placement. Note the continuing aesthetic success.

Placement routine

Restoration or base

The following routine is recommended for the placement of any glass-ionomer material^{33,34} (Figures 11.25-30):

- prepare the cavity surface as smooth as possible (cement will adapt better to a smooth surface)
- apply a liberal coat of 10% polyacrylic acid for 10 seconds
- wash vigorously with air/water spray for 10 seconds
- dry lightly but do not dehydrate the surface

The glass-ionomer can now be syringed into place immediately and supported positively with a matrix to assist adaptation between the glass-ionomer and the dentine and enamel. Observe the stage of set of the excess cement around the matrix and, as soon as it is brittle hard, remove it to release the matrix. Now flick off the matrix and immediately cover the cement with the resin sealant to maintain water balance. Trim further, only if essential, through the sealant using a sharp blade or a slowly rotating steel bur with no air/water spray. Add further sealant if required and light activate. Check for possible excess sealant, particularly at the gingival margin, because it may retain plaque and irritate the gingival tissue. Review in about one week to assess the aesthetic result and trim or polish further if essential.

Luting

When using these materials for luting crowns or inlays, it is more logical to apply a mineralising solution, as suggested above, or a dentine bonding agent containing a poly(alkenoic acid) before crown cementation. Either of these will seal the dentine tubules thus avoiding the risk of them opening and allowing the hydraulic pressure of cementation to permit permeation of polyacrylic acid towards the pulp. There have been reports of post-insertion sensitivity in the past but it has been shown to be no more prevalent than it is when using zinc phosphate or resin cement and is almost invariably associated with removal of the smear layer (Figures 11.31-36).

Classification

The following is the accepted classification for glass-ionomers:

Type I – Luting

Use: cementation of crowns, bridges, inlays, orthodontic appliances

Setting rate: fast set

Powder: liquid ratio – 1.5: 1

Radiopaque: generally

Film thickness: 20 μ or less

Type II – Restorative

• Type II.1 Restorative Aesthetic

Use: aesthetic restorations

Setting rate: autocure – slow resistance to water uptake and loss

resin-modified – fast set, immediate resistance to water uptake

Powder: liquid ratio – 3: 1 or greater

Radiopaque: most materials

• Type II.2 Restorative Reinforced

Use: where increased physical properties are required but aesthetics not important

Setting rate: fast set

Powder: liquid ratio – 3: 1 or greater

Radiopaque: always

Type III – Lining or Base

• Lining

Use: in thin section as thermal barrier under metal restorations

Setting rate: autocure – rapid resistance to water uptake

dualcure – autocure plus light assisted cure

Powder: liquid ratio – 1.5 :1

Radiopaque: always

• Base – dentine substitute

Use: in combination with composite resin in lamination technique

Setting rate: fast set

Powder: liquid ratio – 3: 1 or greater

Radiopaque: always



Fig. 11.31. Cementation of crowns. Remove any residue from the temporary crowns and flush well with water. Dry lightly and paint a little cement over the crown preparation.



Fig. 11.32. Load the crown generously with cement and place into position. Apply positive pressure to ensure full placement. There is no need to maintain pressure for more than a few moments because of the thixotropic flow properties of the cement.



Fig. 11.33. As soon as the cement has set hard flick off the excess. There is no need to seal the cement because it is a fast setting material with rapid resistance to water uptake.



Fig. 11.34. Explore thoroughly for retained scraps of cement in the gingival crevice because they can gather plaque.



Fig. 11.35. A VMK crown has been sectioned and removed because of loss of porcelain. Note the even film thickness of the cement. Most of it has remained attached to the gold rather than the tooth but retention is reliant upon the crown design rather than the cement.



Fig. 11.36. Another VMK crown has been removed because of loss of vitality. Note the fine even film thickness of the cement.

It is accepted that the tensile strength of these cements is no greater than zinc phosphate cements but it must also be understood that no cementation medium is proof against poor design so the luting cement does not need to be very strong. Each restoration must be individually self retentive and the cement is there only to seal the interface between the restoration and the tooth.

SUMMARY



Advantages of GIC as a luting cement

- Fine film thickness and thixotropic flow
- Biocompatibility
- Fluoride release
- Ease of handling

Properties

Biocompatibility

Biological potential of glass-ionomers

Up to this time all the restorative materials used in dentistry have been biologically inert and have shown no activity in relation to the tooth structure they replace. As long as the profession concentrated on trying to eliminate caries through a surgical approach there was not a great deal of interest in the potential for bioactivity, apart possibly, from the release of fluoride ions. It was recognised in the 1950s that silicate cement released fluoride and this was regarded as a novelty. When the glass-ionomers were released in 1976 it was noted that they also released fluoride ions in the same manner and, for that and other reasons, they rapidly replaced the silicates.

Recent investigations have looked more deeply into the exchange of ions both out of the tooth structure and back into it from a variety of sources. The saliva is, of course, the most important element from this point of view because, in a healthy patient, it is a super saturated solution in relation to both calcium and phosphate ions (Chapter 7). However, it is now known that glass-ionomer is also a rich source of ions and it is

possible to trace the exchange from the cement both into and out of tooth structure as well as other skeletal structures.³⁵ There is already considerable clinical evidence of the value of these techniques and clearly it opens the way to far more conservative methods of dealing with the ravages of caries.

Probably the one most important factor arising from these materials being water based lies in the basic chemical principle that it is only possible to have ion mobility in the presence of water. Obviously ion mobility is essential for both demineralisation and remineralisation of tooth structure so totally anhydrous materials can play no part in these activities. However, glass-ionomer contains fluoride, calcium, strontium and aluminium ions and all of these are capable of migration. Therefore they can be harnessed, to some degree, to assist in the healing process.

SUMMARY



Ion exchange can only occur in the presence of water. Glass-ionomer is a water based material, therefore, glass-ionomer is the ideal material for remineralisation.

When contemplating the problems of de- and remineralisation of tooth structure there are two surfaces of the restoration to be considered. The external surface of the restoration is in direct contact with the saliva and the ever changing environment of the oral cavity. Ion exchange is a continuum on those surfaces. However, the inner surface of a restoration is completely isolated and sealed from the oral environment and there is a potential for an ion exchange in that region as well. The interface between the cavity floor and the dentine will be wet from dentine fluid flow and both the pulp and the glass-ionomer can make a contribution towards remineralisation.

NOTE



There are two surfaces to every restoration:

- outer surface in contact with the oral environment
- inner surface in contact with the floor of the cavity

Both surfaces can be involved with remineralisation.

In the presence of water, it is possible for fluoride ions to migrate out of the cement into the surrounding saliva from where they may be taken up into adjacent tooth structure or accumulated in the oral biofilm complex. On the other hand, if the ambient concentration of fluoride in the saliva, and therefore the pellicle-restoration interface, is high enough, it is possible for excess fluoride to return into the cement. This suggests therefore that glass-ionomer will act as a fluoride reservoir. In addition glass-ionomer, through its ion exchange adhesion with tooth structure, will be an ideal sealant for a cavity, preventing the ingress of bacterial nutrients and reducing any that may be present on the floor of the cavity to a dormant form. This means that, because glass-ionomer inactivates the bacteria, it can be placed in very close proximity to the pulp without the risk of developing an irreversible pulpal inflammation. The placement of a further sub-lining, such as calcium hydroxide, under a glass-ionomer is therefore generally not justified.

The actual chemical reaction which takes place under a freshly placed glass-ionomer requires further investigation but it is logical to assume that, immediately following placement, there is likely to be quite a ferment of chemical activity. The pH of the polyalkenoic acid itself is less than pH2.0. On the assumption that the cement is placed on a

newly conditioned dentine that is free of debris, the remaining mineral ions in subjacent dentine will be released by the polyalkenoic acid and will be free to mix with the ions released from the glass-ionomer. It can be assumed that there will then be a free exchange of ions in this isolated area until such time as the acid level is buffered by the newly released calcium and phosphate ions from the tooth and the calcium/strontium and phosphate ions from the cement. As the pH rises the cement will set and the ion exchange will slow down. It has been shown that strontium ions can penetrate a considerable distance into dentine that has been partly demineralised by caries leading to the development of what could be regarded as a type of dentine scar tissue.²¹

Resistance to plaque

It has been shown that bacterial colonies fail to thrive on the surface of glass-ionomer and this in turn means that there is a high level of tolerance to this material in surrounding soft tissue.^{36,37} The *mutans streptococcus* is the major pathogen found in dental plaque and it has been shown that it is unable to thrive in the presence of fluoride. Thus the response of all soft tissues to glass-ionomer restorations is favourable (**Figures 11.37 and 11.38**).



Fig. 11.37. This restoration lies approximately 2.0 mm subgingivally and has been in place for six years. Note the healthy appearance of the tissue at the gingival margin, probably because of the continuing low level fluoride release.



Fig. 11.38. There are two glass-ionomer restorations at the lingual gingival margin of the lower right canine and lateral incisor. Note the level of plaque formation on the adjacent teeth and the relative lack of plaque on the restorations.

Pulp response to glass-ionomer

The pulpal response to glass-ionomer materials is favourable.³⁸ The freshly mixed material is very acid with the pH ranging from 0.9-1.6. However, dentine is an excellent buffer and even thin layers of dentine remaining between the restoration and the pulp are sufficient to prevent a reduction of pH within the pulp tissue. A mild inflammatory response has been noted by several authors but, as the pH rises again within the first hour, the inflammation will resolve within 10-20 days.

Recent work suggests that glass-ionomer can be used to cover and protect a mechanical or traumatic exposure of an otherwise healthy pulp and there can be formation of a dentine bridge in spite of the lowered pH. This supports the previously held concept that it is only bacteria or their toxins that will cause a continuing pulpal inflammation and not a restorative material per se. However, if there is any doubt, particularly concerning the possibility of bacterial infection, a small quantity of calcium hydroxide over the actual exposure will have a predictable result.

Sensitivity to luting materials

It has been suggested that glass-ionomer can be the cause of post-insertion sensitivity when used as a luting agent under full crowns.³⁹ However, it has been shown that the results are the same when using zinc phosphate cement so there are likely to be other causes. In a tooth, which requires a crown, it is likely that the pulp is already inflamed because of existing damage and this may be exacerbated further by crown preparation procedures. Further problems can arise from an ill-fitting temporary restoration. It is therefore desirable to treat the tooth with considerable care at the time of cementation. Do not remove the smear layer by conditioning or scrubbing the dentine in an attempt to develop or enhance adhesion. For preference, seal the dentine tubules by applying a mineralising solution, or a resin dentine bond that contains maleic acid, at the time of cavity preparation and before recording the impression. Then, at the time of cementation, mix the cement at the correct powder/liquid ratio and take care not to dehydrate the dentine prior to placement (**Figure 11.39**).

BE AWARE



When luting with glass-ionomer, do not

- scrub
- condition
- etch

Seal the dentine tubules with

- original smear layer
- remineralising solution
- resin bond containing maleic acid

Note that, in view of the low powder content of a luting cement, the tensile strength is not high. It is therefore unwise to expect it to add significantly to retention. No cementing medium should be used to compensate for a poor cavity design.

Fluoride release

The prolonged and substantial release of fluoride ions from all glass-ionomer materials is of major clinical significance.^{40,41} It is likely that a further release is available from the glass particles themselves because the glass can be regarded as being porous to ions such as these. The large release of fluoride ions during the first few days after placement, declines rapidly during the first week and stabilises by the end of two to three months. There is evidence to indicate a continuing release for at least seven years after placement of a restoration and, almost certainly, longer.

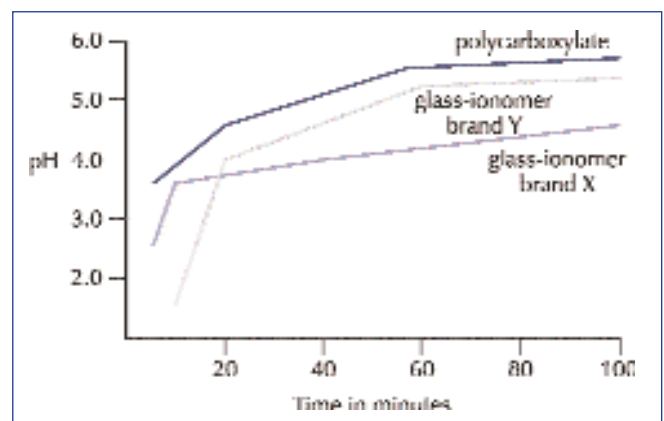


Fig. 11.39. Variation in pH for luting cements: Glass-ionomers show a low pH when first mixed but it rises rapidly in the first 20 minutes so the early acid level is probably not the cause of postinsertion sensitivity.

The fluoride ions come initially from the surface of the glass powder and are held in the siliceous hydrogel matrix. The fluoride ion is not a structural part of the matrix and is approximately the same size and mobility as the hydroxyl ion. This means that a continuing exchange of fluoride ions can occur, depending on the gradient of fluoride available in the oral environment generally, at any given time. Fluoride release normally occurs from the matrix into the adjacent environment but, in the presence of a high fluoride concentration in the mouth, such as during a professional application of fluoride as a preventive measure, fluoride ions can be taken up into the cement again. Glass-ionomer materials can, therefore, be regarded as a fluoride reservoir (**Figures 11.40 and 11.41**).

It is suggested that, for the fluoride concentration to be effective in initiating remineralisation, the concentration in saliva should be at least 10 parts/million. It is this level which is available from glass-ionomer materials and any concentration below this level will be of little or no clinical significance.

Solubility and disintegration

In a clinically relevant organic acid solution, such as lactic acid, solubility is low compared with that of zinc phosphate and zinc polycarboxylate cements.⁴² In water, solubility is less than that of silicate cements but slightly greater than that of

several other cements including resin materials.

However, the surface of glass-ionomer can be damaged in the presence of a low pH such as during the application of some topical fluoride solutions. Acid phosphate fluoride solution has a pH 3.0 while neutral sodium fluoride is about pH 6.5. Therefore, it is possible for roughening of the cement surface to occur if a phosphate fluoride solution is applied regularly (**Chapter 4**). Caution should be exercised when using these solutions near glass-ionomers, particularly in relatively hostile environments such as those found in patients with xerostomia. In conditions of severe xerostomia, such as found in Sjögren's Syndrome, autocure glass-ionomers will often only last for a year or so before disintegrating unless they are laminated with composite resin.

resin-modified glass-ionomers appear to be more resistant to dissolution and disintegration than the autocure glass-ionomers but it is still wise to use topical fluorides with a higher pH.

BE AWARE

Acidulated fluoride phosphate applications can damage glass-ionomer.

Dimensional change

A free-standing specimen of a glass-ionomer material, correctly manipulated and protected from early exposure to hydration or dehydration, will show a volumetric setting contraction of

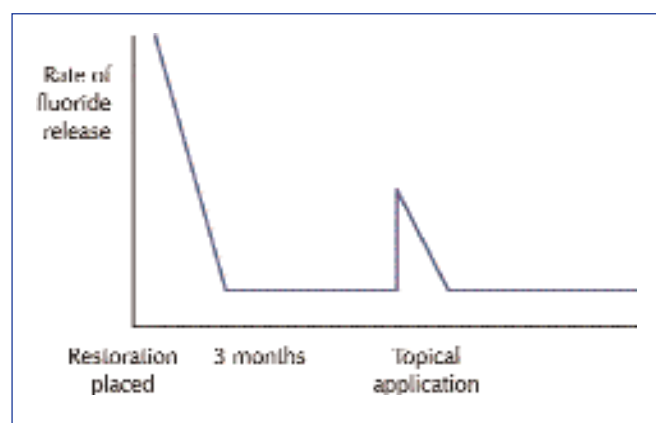


Fig. 11.40. Fluoride release from glass-ionomer: Initial fluoride release is high but declines rapidly over the first 3 months. It will continue probably for the life of the restoration but can be 'recharged' by a topical application for another short period.

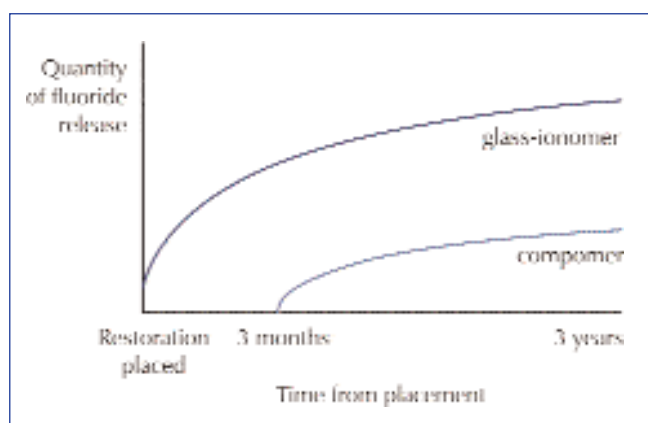


Fig. 11.41. Variation in pH for luting cements: This graph shows the total fluoride release over time for the average glass-ionomer compared with a compomer which will not show a fluoride release for some time after placement.

approximately 3% which develops slowly through the prolonged setting process. In the presence of adhesion, through the ion exchange with tooth structure, the shrinkage will be controlled and, in view of the time taken for the setting reaction, there will be a degree of stress relaxation leading to a reduced marginal discrepancy.

The resin-modified glass-ionomer restorative materials contain less than 5% additional resin and show a very small initial shrinkage of the resin component at the time of light activation. Subsequent shrinkage from the continuing acid/base reaction will develop rather slowly and will be controlled to some extent by the adhesion. Over time, the set material will take up water and expand to some degree and this will more than negate any setting shrinkage. In contrast, light activated composite resins show immediate shrinkage upon light activation with development of considerable stress at the tooth to resin interface.

Resistance to fracture

One of the few limitations of glass-ionomers is their susceptibility to brittle fracture. Compared with hybrid composite resins and dental amalgams, glass-ionomer materials are weak and lack rigidity.⁴³ Clinical use should avoid situations that subject the restoration to heavy occlusal load or bending. There is a difference in strength among various glass-ionomers with a substantial difference between the original autocure glass-ionomers and composite resins or amalgams. The resin-modified glass-ionomers are stronger, with

the best of them showing more than double the fracture resistance so that they are nearly comparable with the microfil composite resins. Recent research has led to improvement in the physical properties of the autocure materials and there are a number now on the market with fracture resistance superior to the resin-modified materials and very close to microfilled composite resins. Further improvements are expected.

Typical 24-hour strengths are shown in **Table 11.2**.

It should be noted that the presence of silver does not impart additional fracture toughness to silver cermets although it does improve abrasion resistance. Cermets are not indicated where occlusal loading precludes the use of other glass-ionomer materials.

The modulus of elasticity, which measures rigidity, ranges from 7 GPa to 13 GPa and the silver cermets tend to have a relatively low elastic modulus.

Abrasion resistance

Immediately after placement glass-ionomers are less resistant to abrasion than composite resins but their resistance improves considerably as they mature. Providing the material is well supported and protected by remaining tooth structure, abrasion resistance is satisfactory.⁴³ Because abrasion results in loss of matrix, there will be an increase in surface roughness over time with exposure of the glass particles and internal porosities (**Figures 11.42 and 11.43**).

The silver cermet materials, where the silver particles are incorporated on to the glass particles, have more resistance to abrasion because, under

TABLE 11.2: Strengths (after seven days) of glass-ionomer cements

Type of glass-ionomer	Compressive strength (MPa)	Diametral tensile strength (MPa)	Flexural strength (MPa)	Shear punch (MPa)
Type II.1 aesthetic	70-220	12-20	8-40	30-40
Type II.2 reinforced	140-220	13-16	22-30	35-45
Type I luting	70-150	6-15	4-18	20-25
resin-modified	110-220	15-16		60-70
Type II.2 new version	161-274	19-26		70-80

Differences among commercial products and in experimental conditions account for the large variations.



Fig. 11.42. These two lower molars have had the fissures sealed with a high strength Type II.2 glass-ionomer. This photograph was taken three years after placement showing that the resistance to wear is high.



Fig. 11.43. The cermet restoration in the lower molar has been in place for 14 years and shows no sign of wear. This is not now a recommended treatment because there are more aesthetic restorations. However, it suggests the potential for longevity for glass-ionomers.

loading, the silver is smeared across the surface of the set cement during finishing and function. The result is a so-called Beilby layer of burnished metal such as is found following polishing of most soft metals.

Thermal diffusivity

This property governs the insulating efficiency of a material under transient conditions. Represent-

ative values for several materials are listed in **Table 11.3**. Thermal diffusivity increases with increased powder/liquid ratio. Therefore the Type III lining cements show a higher figure than the Type II restorative cements. Despite its silver content, the silver cermet behaves as a cement rather than as a metallic material and has a diffusivity lower than polycarboxylate cements.

TABLE 11.3: Thermal expansion and diffusivity		
Type of material	Linear coefficient of thermal expansion ($K \times 10^{-6}$)	Thermal diffusivity $\times 10^{-6}/^{\circ}C$ (mm^2/sec)
Casting gold	15.0	90.00
Dental amalgam	22-28	9.40
Composite resin	20-60	0.25
Glass-ionomer materials	10-11	
Type I. luting		0.15
Type II.1 aesthetic restorative		0.19
Type II.2 silver – cermet		0.19
Type III base/lining – autocure		0.35
Type III lining dual-cure		0.19
Tooth	11.4	
Dentine		0.18
Enamel		0.47
Water		0.14

Table 11.3. Note that the glass-ionomers show figures that are very close to those of tooth structure.

Colour and translucency

The Type II.1 restorative aesthetic materials, both autocure and resin-modified, provide adequate colour matching and translucency, although translucency will take several days to develop for the autocure cements.⁴⁴ The greatest problem with the autocure type is that it will be seriously affected by early exposure to water; therefore, sealing immediately after placement is essential. Careful clinical placement with final contouring being undertaken at least 24 hours later can lead to entirely satisfactory results providing colour selection is correct. If the colour match or translucency is still not satisfactory after maturation over one week the restoration can be laminated with composite resin to improve aesthetics (**Figures 11.44 and 11.45**).

The resin-modified glass-ionomers, on the other hand, show excellent translucency immediately after light activation. Over the following day or two, the actual measure of translucency will decline although this is not clinically detectable. Within the first week the restoration will have recovered completely and may in fact have surpassed the original degree of translucency. The ultimate restoration compares favourably with composite resin and lamination is normally not justified.



Fig. 11.44. There are three Type II.1 restorative aesthetic glass-ionomer restorations in the upper canine and two bicuspids that have been in place for 12 years. They were properly sealed at the time of placement.

Radiopacity

Radiopacity can be achieved by the incorporation of barium (Ba), strontium (Sr) or lanthanum (La), by fusing metal to the glass particles, as in cermet powders, or by mixing in dental amalgam alloy or zinc oxide. Radiopacity can be important in many applications of minimal intervention dentistry but is unnecessary and undesirable for materials used where aesthetics is of prime importance.

Most of the glass-ionomers are more radiopaque than dentine and several exceed the radiopacity of enamel. Some of the autocure Type II.1 restorative aesthetic cements are entirely radiolucent because the incorporation of a radiopacifier may alter their translucency. The silver cermet has a radiopacity approaching that of dental amalgam.

Clinical Considerations

Taking into account the foregoing factors it is suggested that, during clinical placement of each of the types, consideration should be given to the following factors.



Fig. 11.45. Three glass-ionomer restorations. The canine was restored five years ago with a resin-modified glass-ionomer. The two bicuspids were restored with Type II.1 restorative glass-ionomers 15 years ago and were not properly sealed at the time of placement. The aesthetic result is unsatisfactory.

Type I – Luting

Reasons for use

The following are the main reasons for regarding glass-ionomer as useful for luting indirect restorations:^{45,46}

- fine ultimate film thickness, easily obtained because of good flow properties
- low solubility in the oral environment
- tensile strength and abrasion resistance equivalent to zinc phosphate
- presence of continuing fluoride release
- high tissue tolerance of both pulp and gingival tissue

Strength of retention

Strength of retention with an indirectly fabricated restoration is directly proportional to the area of the vertical walls of the preparation and the accuracy of fit. It is unwise to expect to enhance the retention with a luting agent because the tensile strength of the cement is in the order of only 2-4 MPa. Therefore glass-ionomer should not be used for its retention properties and conditioning of the tooth structure, prior to cementation, to enhance adhesion is undesirable.

Ultimate film thickness

The powder in the luting cement should contain particle sizes of 10 μm or less to allow for optimum film thickness.^{47,48} The freshly mixed cement shows properties of thixotropic flow so it is unnecessary to maintain pressure on the newly placed restoration once it is fully seated. For preparations with long parallel sides it may still be wise to vent the crown to encourage full seating.

Correct powder/liquid ratio

The prescribed powder/liquid ratio is normally 1.5:1 and it is important to achieve this during mixing.⁴⁹ Capsulation is the preferred method of dispensing and mixing. Too little powder will leave an excess of liquid with the risk of post-insertion sensitivity and increased solubility. An excess of powder will reduce the ability to achieve complete seating of the restoration thus altering the fit and the occlusion.

Type II.1 – Restorative aesthetic

Reasons for use

The following are the main reasons for using these materials as restoratives in their own right.^{50,51}

- adequate aesthetics and translucency are available in both the autocure and the resin-modified materials
- physical properties are sufficient as long as the restoration is fully supported by surrounding tooth structure and it is not subject to undue occlusal load
- adhesion can be achieved with underlying tooth structure through the ion exchange mechanism, thus completely eliminating microleakage.
- the material acts as a fluoride reservoir. The continuing release inhibits plaque formation on the restoration, thereby enhancing tissue tolerance, as well as providing a source of fluoride for remineralisation of any adjacent demineralised tooth structure.⁴⁸
- it is a bioactive material so there is a continuing exchange of fluoride and other ions available from the restoration to the tooth structure as well as the surrounding oral environment.

Dispensing and placement

The prescribed powder/liquid ratio is 3.5:1 or greater and, for optimum physical properties, the powder content should not be reduced. Capsulation is the preferred technique for dispensing and mixing because it is always reliable.

The use of a matrix is desirable to improve adaptation of the cement to the cavity floor and to help restore the original crown form. It will also minimise the subsequent need to contour and polish the restoration.

Water balance

Maintenance of the water balance of the autocure cements in this category is essential. Some manufacturers claim to provide a cement which is resistant to water uptake within 15 minutes of mixing but all these materials will develop superior translucency if they are protected with a water-proof sealant for at least the first hour.

Light activation

Manufacturers recommend 20-40 seconds of light activation for the resin-modified materials. However, as it is not possible to light activate excessively, it is recommended that the light be applied for at least 20 seconds in each of several directions to ensure the material is fully cured.

Contour and polish

Initial contouring is best carried out using a matrix. Any excess can then be modified using fine diamonds at intermediate high speed under air/water spray and progressing finally to graded aluminium oxide discs, also under air/water spray. Take care not to dehydrate the cement in the first six months after placement.

Type II.2 – Restorative reinforced**Reasons for use**

In situations where a fast setting material is desirable with increased physical properties, but the colour match of the restoration with the tooth, and the translucency, are not important.

Physical properties

The physical properties of this group, particularly the tensile and ultimate compressive strengths

and the fracture resistance are not yet comparable with amalgam and will never be as good as gold. However, considerable progress has been made in improving these properties so their uses will expand. There are now materials with strength values, assessed by a shear punch test, close to microfilled composite resins.

Core build-up

The term 'core build-up', although widely used in relation to these materials, is really a misnomer. Whilst it can be used to a limited degree for rebuilding a badly broken down tooth in preparation for the placement of a full crown, its physical properties are such that it cannot be relied upon to accept any undue occlusal or lateral load. It is essential that there is still adequate remaining natural tooth structure strong enough to withstand the stresses to which the crown is likely to be subjected. There should be at least one half of the original crown bulk present with a collar of at least 3 mm of natural dentine around the gingival margin involved in the preparation. Reinforcement of glass-ionomer with pins and posts will not be sufficient, on their own, in the absence of sound dentine.



Fig. 11.46. A low powder/liquid ratio glass-ionomer has been placed on the floor of this cavity as a lining only. It will be covered with amalgam because it is not strong enough to withstand the setting shrinkage of a composite resin.



Fig. 11.47. A moderate sized cavity in an upper molar has been based with a high strength, fast set glass-ionomer prior to placing a composite resin. Note that the gingival margin is still in enamel so this will be lightly bevelled and the composite resin will adhere to it.

Type III – Lining and base cements

Reasons for use

There are two different applications for this group based on the powder/liquid ratio employed

- low powder content (ratio 1.5:1) should be used primarily as a lining under another restoration to prevent thermal change irritating the pulp
- high powder content (ratio 3.5:1 or greater) should be used as a base or dentine substitute

Lining cements

Lining cements have relatively low physical properties and are designed to be entirely covered by another restorative material. They should be used in thin section to fill voids in cavity design and act as a thermal insulator to prevent pulpal insult. Because of their physical properties they should not be expected to act as a bonding agent for the overlying restorative material. Under no circumstances should they be left exposed to the oral environment (**Figure 11.46**).

Dentine substitute

When the cement is mixed with a high powder content, it can be regarded as a dentine substitute and can act as a significant part of a total restora-

tion. Always use the strongest material available with the highest possible powder content and completely restore the cavity with glass-ionomer. It can then be cut back to make room for a stronger material, such as amalgam or composite resin, to be laminated over it (**Figure 11.47**).

The Lamination or 'Sandwich' Technique

The concept of using two different materials to form one final restoration is new to dentistry and leads to some confusion.^{53,54} The rationale behind the technique is to make the most of the biological, physical and/or aesthetic properties of each material and, in the presence of adhesion, to achieve as close as possible to a single monolithic reconstruction of a tooth. It has been shown that a tooth can be restored to its original physical strength by this method with no compromise in aesthetics (**Figures 11.48-54**).

As long as there remains doubt about the efficiency and longevity of techniques for bonding composite resin to dentine the lamination technique must be the preferred system for the



Fig. 11.48. The following is a laboratory series on the placement of a lamination restoration using composite resin over glass-ionomer. Note that there is still some affected dentine on the floor of the cavity but as it will be completely sealed there is no need to remove it.



Fig. 11.49. A short length of mylar strip was used as a matrix and the entire cavity restored with a high strength, fast set glass-ionomer.



Fig. 11.50. A cavity suitable for composite resin has now been prepared in the glass-ionomer regarding the cement as a dentine substitute. The enamel walls are exposed to make use of the available micromechanical adhesion but the gingival margin remains covered with glass-ionomer.

placement of these restorations in any cavity where at least one margin is located on dentine.⁵⁵ Also, where a cavity extends deeply interproximally beyond the cemento-enamel junction, and the ultimate restoration is to be amalgam, then it is relatively simple to place a glass-ionomer base in the gingival half of the proximal box and laminate over that.

The result will be that, with either composite resin or amalgam, the section of the restoration which is likely to be subgingival will have an ion exchange adhesion to the dentine, will release fluoride and will show a resistance to plaque formation. In both situations the physical limitations of the glass-ionomer will be compensated by having the stronger material laminated over it. There are further practical advantages to be gained from placing the glass-ionomer as a dentine substitute:

- there will be less composite resin to be placed, thus minimising the ultimate sum total shrinkage of the composite resin, that occurs during light activation
- it will minimise the number of increments of composite resin to be placed and light activated, thus saving time as well

Principles of lamination

To obtain the maximum benefits from the technique the following principles must be observed:



Fig. 11.51. The base is viewed from another angle to show that there is sufficient depth in the cavity to allow for the relative flexibility of the composite resin.

- use the strongest glass-ionomer available, place it in quantity and regard it as a dentine substitute – note that resin-modified is not necessarily the strongest.
- avoid the placement of a sub-base, such as calcium hydroxide, because it will reduce the area for adhesion of the glass-ionomer
- condition the cavity as usual to develop full ion exchange adhesion with the underlying tooth structure
- place the glass-ionomer and allow it to set fully, either chemically or by light initiation, before proceeding
- once set, cut the glass-ionomer back to expose all the enamel margins because the composite resin union to sound enamel is the strongest available
- allow sufficient space for a reasonable thickness of composite resin, taking into account its relative flexibility under load
- build the contact area in composite resin rather than glass-ionomer
- develop a union between the glass-ionomer and the composite resin by lightly etching the glass-ionomer, while etching the enamel, so as to obtain optimum advantage from the 'monolithic reconstruction' concept
- always use a radiopaque glass-ionomer



Fig. 11.52. Both glass-ionomer and enamel are etched for 15 seconds, washed thoroughly and lightly dried. A resin enamel bond is painted over the surface and light activated. A complex dentine bonding agent is not needed because there is no dentine involved.

Lamination of autocure glass-ionomer

It is possible to develop a mechanical interlock between an autocure glass-ionomer and composite resin similar to the union between composite resin and enamel. As soon as the glass-ionomer is set, apply a standard acid etchant to both the glass-ionomer and enamel and leave for 15 seconds only. Wash thoroughly and dry lightly but do not dehydrate the glass-ionomer. Apply a thin layer of a low viscosity enamel bonding resin and light cure. There is no need to use a complex dentine bonding routine because there is no dentine still exposed to which to bond. Finally place the composite resin using a standard incremental build-up technique.

Lamination of resin-modified glass-ionomer

It is unnecessary to etch a resin-modified glass-ionomer when laminating it with composite resin, because there appears to be sufficient bonding capacity in the HEMA, incorporated in the cement, to ensure an adequate chemical bond between the two materials. If acid etchant does flow over the cement, however, it will do no harm. Follow the same routine as described above but etch the enamel only.

Lamination of amalgam

This combination is likely to be used in restoration of a molar tooth where the gingival margin of



Fig. 11.53. The composite resin can now be built incrementally with care to ensure maximum conversion of the resin bonds and minimum ultimate shrinkage.

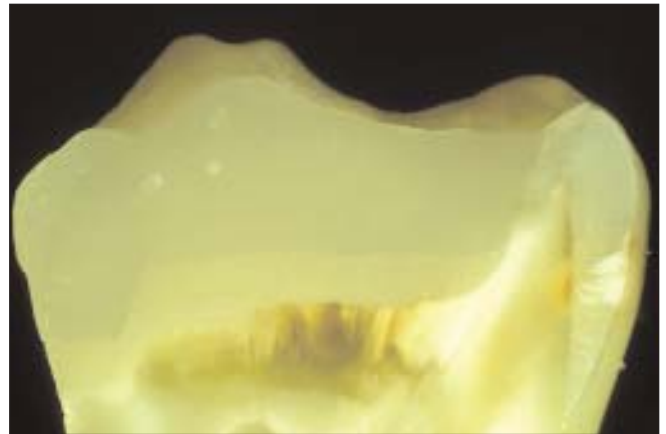


Fig. 11.54. The tooth has been sectioned mesio-distally to show the relationship between the glass-ionomer and the composite resin.

the proximal box is well subgingival. Use the strongest glass-ionomer available as the base and develop the full ion exchange adhesion to tooth structure by conditioning the cavity. Ensure the material is fully set and then cut it back reasonably generously to allow sufficient space to compensate for the relatively brittle nature of the amalgam. Expose all the enamel margins and develop a mechanical interlock design in both the glass-ionomer and the remaining tooth structure so that the amalgam is firmly locked into position in the usual style.

It is possible to develop a degree of chemical union between the glass-ionomer and the amalgam by wiping a small quantity of 45% polyacrylic acid (standard glass-ionomer liquid) over the set cement just prior to packing the amalgam. Wipe

off any excess but leave a fine layer of acid on the cement only. The freshly packed amalgam will now unite with the glass-ionomer to some degree, although the value of such union is open to question.

Further Reading

1. Wilson AD, McLean JW. *Glass-Ionomer Cement*. London: Quintessence; 1989.
2. Tyas MJ, Burrow MF. Adhesive restorative materials: A review. *Aust Dent J* 2004; 49:112-121.
3. Wilson AD, Nicholson JW. *Acid-base Cements: Their Bio-medical and Industrial Applications*. Cambridge: Cambridge University Press; 1993.
4. McLean JW, Gasser O. Glass cermet cements. *Quint Int* 1985; 5:333-43.
5. Simmons JJ. The miracle mixture. Glass-ionomer and alloy powder. *Texas Dent J* 1983; October:6-12.
6. Arita K, Lucas ME, Nishino M. The effect of adding hydrox-yapatite on the flexural strength of glass-ionomer cement. *Dent Mater J* 2003; 22:126-136.
7. Nicholson JW, Anstice M. The development of modified glass-ionomer cements for dentistry. *Trends in Polymer Science* 1994; 2:272-6.
8. Mount GJ, Patel C, Makinson OF. Resin-modified glass-ionomers; Strength, cure depth and translucency. *Aust Dent J* 2002; 47:339-343.
9. Ellakuria J, Triana R, Minguez N, Sloer I, Ibaseta G, Maza J, Garcia-Godoy F. Effect of one-year water storage on the surface microhardness of a resin-modified versus conventional glass-ionomer cement. *Dent Mater* 2003; 19:286-290.
10. Ruse ND. What is a 'compomer'? *J Can Dent Assoc* 1999; 65:500-4.
11. McLean JW, Nicholson JW, Wilson AD. Proposed nomenclature for glass-ionomer dental cements and related materials. Guest editorial, *Quint Int* 1994; 25:587-589.
12. Brook IM, Hatton PV. Glass-ionomers: bioactive implant materials. *Biomaterials* 1998; 19:565-71.
13. Osinaga PW, Grande RH, Ballester RY, Simionato MR, Delgado Rodrigues CR, Muench A. Zinc sulfate addition to glass-ionomer-based cements: influence on physical and antibacterial properties, zinc and fluoride release. *Dent Mater* 2003; 19:212-7.
14. Botelho MG. Inhibitory effect on selected oral bacteria of antibacterial agents incorporated in a glass-ionomer cement. *Caries Res* 2003; 37:108-114.
15. Mazzaoui SA, Burrow MF, Tyas MJ, Dashper SG, Eakins D, Reynolds EC. Incorporation of casein phosphopeptide-amorphous calcium phosphate into a glass-ionomer cement. *J Dent Res* 2003; 82:914-918.
16. Bass EV, Wing G. The mixing of encapsulated glass-ionomer cement restorative materials *Aust Dent J* 1988; 33:243.
17. Nicholson JW. Chemistry of glass-ionomer cements: a review. *Biomaterials* 1998; 19:485-94.
18. Nicholson JW, Wasson EA. The setting of polyalkenoate (glass-ionomer) cement: *Transactions of the Academy of Dental Materials: Proceedings of a Conference on Setting Mechanisms*. June, 1992.
19. Nicholson JW. Glass-ionomers in medicine and dentistry. *Proc Inst Mech Eng (H)* 1998; 212:121-6.
20. Mount GJ. Clinical placement of modern glass ionomer cements. *Quint Int* 1993; 24:107.
21. Mount GJ. An Atlas of glass-ionomer cement : *A Clinician's Guide* 2nd ed. Martin Dunitz: London; 1994.
22. Watson TW, Banerjee A. Effectiveness of glass-ionomer surface protection treatments : a scanning optical microscope study. *J Prosthodont Rest Dent* 1993; 2:85-90.
23. Earl MSA, Mount GJ, Hume WR. The effect of varnishes and other surface treatments on water movement across the surface of a glass-ionomer cement : II. *Aust Dent J* 1989; 34: 326-329.
24. Leirskar J, Nordbo H, Mount GJ, Ngo H. The influence of resin coating on the shear punch strength of a high strength auto-cure glass ionomer. *Dent Mater* 2003; 19:87-91.
25. Mount GJ, Papageorgiou E. Some curing properties of light activated glass ionomer cement. *Aust Dent J* 1992; 37:309.
26. Akinmade AO, Nicholson JW. Review- glass-ionomer cements as adhesives. Part 1. Fundamental aspects and their clinical relevance. *J Material Sci Med* 1993; 4.
27. Mount GJ. Adhesion of glass-ionomer cement in the clinical environment. *Oper Dent* 1991; 16:141-148.
28. Tyas MJ, Burrow MF. Clinical evaluation of a resin-modified glass ionomer adhesive system: results at five years. *Oper Dent* 2002; 27:438-41.
29. Watson TF, Billington RW, Williams JA. The interfacial region of the tooth/glass-ionomer restoration: a confocal microscope study. *Am J Dent* 1991; 3:303-310.
30. Akinmade A. Adhesion of glass-polyalkenoate cement to collagen. *J Dent Res Special Issue* 1994, Abstr. 633, p. 181.
31. Glasspoole EA, Erickson RL, Davidson CL. Effect of surface treatments on the bond strength of glass ionomers to enamel. *Dent Mater* 2002; 18:454-462.
32. Causton BE, Johnson NW. Improvement of polycarboxylate adhesion to dentine by the use of a new calcifying solution. *Br Dent J* 1982; 152:9-11.
33. Matis BA, Cochran MA. Technique on restoring cervical lesions. *Oper Dent* 2002; 27:525-7.
34. Hewlett ER, Mount GJ. Glass-ionomers in contemporary restorative dentistry – a clinical update. *J Calif Dent Assoc* 2003; 31:483-492.

35. Ngo HC. Biological potential of glass-ionomers. In *An Atlas of Glass-ionomers* 3rd edition, Mount GJ. Martin Dunitz: London; 2002, Chapt 2.
36. Sidhu SK, Schmalz G. The biocompatibility of glass-ionomer cement materials: A status report for the American Journal of Dentistry. *Am J Dent* 2001; 14:387-396.
37. Sanders BJ, Gregory RL, Moore K, Avery DR. Antibacterial and physical properties of resin-modified glass-ionomers combined with chlorhexidine. *J Oral Rehab* 2002; 29:553-558.
38. De Souza Costa CA, Hebling J, Garcia-Godoy F, Hanks CT. *In vitro* cytotoxicity of five glass-ionomer cements. *Biomaterials* 2003; 24:3853-3858.
39. Rosenstiel SF, Land MF, Crispin BJ. Dental luting agents: A review of the current literature. *J Prosthet Dent* 1998; 47: 280-301.
40. Forsten L. Fluoride release of glass-ionomers. in: *Glass-ionomers; the next generation*. Proceedings of the 2nd Symposium on Glass-ionomers. Ed. Hunt P, Philadelphia, 1994; 241-249.
41. Forsten L. Fluoride release and uptake by glass-ionomers and related materials and its clinical effect. *Biomaterials* 1998; 19:503-508.
42. Cranfield M, Kuhn AT, Winter G, Factors relating to the rates of fluoride release from a glass-ionomer cement, *J Dent* 1982; 10:333-4.
43. Mitsuhashi A, Hanaoka K, Teranaka T. Fracture toughness of resin-modified glass ionomer materials: effect of powder/liquid ratio and powder particle size reduction on fracture toughness. *Dent Mater.* 2003; 19:747-757.
44. Mount GJ, Papageorgiou E. Some curing properties of light-activated glass ionomer cements. *Aust Dent J* 1993; 37:309.
45. White SN, Zhaokun Yu, Film thickness of new adhesive luting agents, *J Prosthet Dent* 1992; 67:782-5.
46. White SN, Zhaokun Yu, Kipnis V, Effect of seating force on film thickness of new adhesive luting agents, *J Prosthet Dent* 1992; 68:476-81.
47. McComb D. Retention of castings with a glass-ionomer cement. *J Prosthet Dent* 1982; 48:285-288.
48. Dahl BL, Oilo G. Retentive properties of luting cements: an *in vitro* investigation. *Dent Mater* 1986; 2:17-21.
49. Metz JE, Brackett WW. Performance of a glass-ionomer luting cement over 8 years in a general practice. *J Prosthet Dent* 1994; 71:13-15.
50. Mount GJ. Longevity in glass-ionomer restorations: review of a successful technique. *Quint Int* 1997; 28:643-650.
51. Davidson CL. Glass ionomer cement, an intelligent material. *Bull Group Int Rech Sci Stomatol Odontol* 1998; 40:38-42.
52. Nicholson JW. Chemistry of glass-ionomer cements: a review. *Biomaterials* 1998; 19:485-494.
53. Mount GJ. Clinical requirements for a successful 'sandwich' – dentine to glass-ionomer cement to composite resin. *Aust Dent J* 1989; 34:259-65.
54. McLean JW, Glass ionomer cements. *Br Dent J* 1988; 164: 293-299.
55. Sidhu SK, Pilecki P, Cheng P-C, Watson TF. The morphology and stability of resin-modified glass-ionomer adhesive at the dentine/resin – based composite interface. *Am J Dent* 2002; 15:129-136.

12 Composite Resins

J. C. L. Neo ♦ A. U. J. Yap

The first of the resin restorative materials was launched in the early 1950s but, as it was unfilled it was not successful and was rapidly abandoned. The concept of adhesion between resin and enamel was first investigated by Buonocore about the same time but it was not until the concept of adding fillers to the resin matrix was evolved in the 1960s that there was any sign of success. From that time on there has been considerable research undertaken until at this time the so-called composite resins are the material of choice in many circumstances. It is possible to achieve a very high level of aesthetic replacement of tooth structure and, in the presence of an enamel margin around the full circumference of the cavity, a restoration will show minimal signs of microleakage. The longevity of the individual restoration is highly dependent on the care and skill of the operator who must work within the limitations of the material.



Introduction

The continuum of direct aesthetic restorative materials

The clinical use of direct aesthetic restorative materials has increased substantially in recent years fuelled by increased aesthetic demands by patients and the decline in the popularity of amalgam as a restorative material. The latter may be attributed to the fear of the potential toxicity and the possible environmental effects of mercury.

Glass-ionomers and composite resins form the two extreme ends of the continuum of direct restorative materials (**Figure 12.1**). Glass-ionomers consist of basic glasses and acidic co-polymers and set by an acid/base reaction. Composite resins are basically ceramic filled polymers which set by resin polymerisation. The advantages of glass-ionomers include chemical bonding to tooth structure through an ion exchange mechanism, fluoride release and a high level of bioactivity. The qualities of composite resin include excellent aesthetics and acceptable physical properties but placement techniques are quite demanding. Attempts to combine the properties of the two materials have led to the development of a number of hybrid materials. These include resin-modified glass-ionomers, compomers and, more recently, giomers.

Resin-modified glass-ionomers are hybrid materials that retain an acid/base setting reaction as a significant part of their overall curing process, as well as the bioactivity and ion exchange adhesion of the glass-ionomers.¹ In contrast, compomers are materials that contain either or both of the essential components of glass-ionomers but at levels insufficient to promote the acid/base setting reaction or the ion exchange adhesion.² The filler is an ionomer glass and a variable amount of dehydrated polyacrylic acid is incorporated into a resin matrix. The acid is activated when water is absorbed into the restoration and subsequently reacts with the glass. However, the delayed reaction does not allow for development of an ion exchange adhesion nor a prolonged fluoride release.

Giomers, the latest category of hybrid materials,

are based on the incorporation of prereacted glass-ionomer technology. The basic glass filler is reacted with acidic polymers in water prior to inclusion into a silica filled resin. Manufacturer's claims include fluoride release and recharge, biocompatibility, clinical stability, excellent aesthetics and a smooth surface finish. Independent studies on giomers are currently limited and do not fully substantiate these claims. The few that have been published suggest that they have a surface finish that compares well with compomers and composite resins.³ However, the long term fluoride release is questionable⁴ and the ion exchange adhesion is not available so they belong with composite resins and not with glass-ionomers.

Composites, compomers and giomers

Composites resins may be defined as three-dimensional combinations of at least two chemically different materials with a distinct interface.⁵ They were introduced to the dental profession in the early 1960s. Micromechanical bonding of unfilled resins to enamel via acid etching was already established in the 1950s but the unfilled resin materials then in use suffered a high polymerisation shrinkage, a high coefficient of thermal expansion and poor physicomachanical characteristics so that they were not satisfactory as a restorative material. They were originally chemically activated and light activated versions were subsequently developed in the 1970s. Combined materials using both chemical and light activation

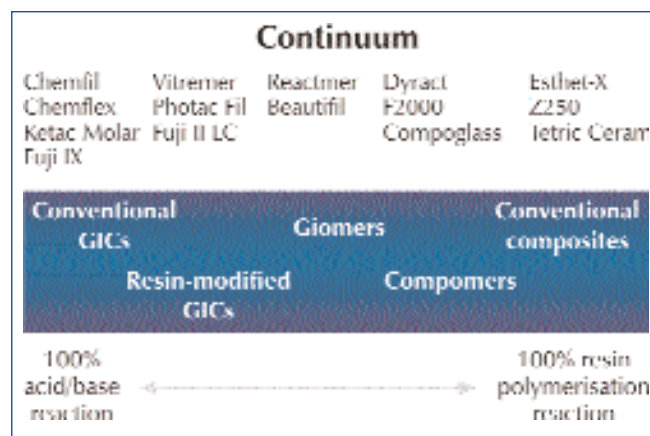


Fig. 12.1. Continuum of direct tooth coloured restorative materials.

systems are now available for core build-ups and luting purposes.

The current versions of composite resins, compomers and giomers are all packaged as a single paste system, are light polymerised and require bonding systems to promote adhesion to tooth structure. They are made up of an organic phase in the form of a resin matrix, and this contains a dispersed phase in the form of inorganic filler particles. At the interface between the two there is a coupling agent. There are also minor additives such as polymerisation initiators, activators, coloring pigments and stabilisers.

The base monomers include Bis-GMA (Bisphenol A glycidyl methacrylate) or UDMA (urethane dimethacrylate) as well as diluents like TEGDMA (Triethyleneglycol dimethacrylate). The organic phase of a compomer also contains resins with functional acid groups, such as TCB, which is a reaction product between butanetetracarboxylic acid and hydroxyethyl methacrylate (HEMA) and/or CDMA which is an oligomer derived from citric acid. At present the commercially available giomers are UDMA-based.

Many of these materials contain amorphous silica with an average particle size of $0.04\ \mu\text{m}$ as the filler and these may be included as individual particles or as pre-polymerised fillers. To achieve radiopacity, silicate particles based on aluminium, strontium or barium oxides are also incorporated. Compomers and giomers also contain fluorosilicate glass or ytterbium trifluoride fillers to provide some level of fluoride release.

Silane coupling agents are commonly employed to ensure bonding between filler particles and the resin matrix. Silane molecules have silanol and methacrylate groups at their extreme ends and these attach to the silica based fillers and the methacrylate groups in the resin matrix respectively.⁶

Composition, Setting and Classification

Composition

The major components of a composite resin are as follows:

- **Organic phase**
 - most composite resins contain the aromatic oligomer Bis-GMA dimethacrylate
 - some contain urethane dimethacrylate.
 - Bis-GMA is highly viscous and shows polymerisation shrinkage and water sorption. A diluent like TEGDMA is generally added to reduce the viscosity.
- **Inorganic phase**
 - filler particles are usually glasses containing aluminium, barium, strontium, zinc, zirconium or quartz with size ranging from $0.1\text{--}10\ \mu\text{m}$
 - alternate fillers can be silica with particle sizes from $0.04\text{--}0.2\ \mu\text{m}$
- **Interfacial phase – coupling agent**
 - to bind the filler to the matrix
 - to act as stress absorber allowing stresses in the resin to be transferred between filler particles via the weaker matrix
- **Miscellaneous phases**
 - accelerators and initiators

Filler loading is expressed as volume % or weight % and contributes to the physical and mechanical properties of strength, stiffness, dimensional change, setting contraction, radiopacity and improved handling. Both the size and the size distribution of the filler particles play a part in the characteristics of each material. The smaller the filler particle size, the better the polishability. The ideal combination of filler size involves a combination of two or more sizes of filler particles to allow for more efficient packing.

Chemically cured composite resins contain benzoyl peroxide as the initiator combined with a tertiary aromatic amine accelerator to produce free radicals for polymerisation.

Light activated composite resins contain camphorquinone and a tertiary amine as the photoinitiators, pigments and UV absorbers. Pigments in

the form of oxides are added to provide a variety of shades. Ultraviolet absorbers stabilise potential colour changes.

Setting reactions

The setting reaction is a free radical addition polymerisation. The production of free radicals propagates the reaction leading to a highly cross-linked polymer. For the chemically cured resins, the initiator, benzoyl peroxide, is activated by the tertiary aromatic amine. For photocured resins, visible light with an approximate wavelength of 470 nm activates the photoinitiator. The co-initiator interacts with the activated photoinitiator producing free radicals for the start of the polymerisation process. For dual curing resins, the process of chemical and light curing are both incorporated in the system.

Classification systems

The system of classification in recent years has been based on the particle size and size distribution and the percentage filler loading of the resin. The classification introduced by Lutz and Phillips 1983⁷ describes the following groups:

TABLE 12.1			
Category	Particle size (μ)	Filler loading (by weight)	Particles
Traditional/Macrofillers	1-15	70-80%	quartz
Microfine/microfillers	0.04-0.2	50-60%	amorphous silica
Fine particles	0.4-3	70-90%	ground glass/quartz
Hybrids/blends		77-84%	macrofillers plus microfillers

After Lutz and Phillips

Subsequently Bayne⁸ offered a further classification based also upon particle size and divided into six categories.

TABLE 12.2

Category	Particle size (μ)	Category	Particle size (μ)
Megafill	0.5	Minifill	0.1-1
Macrofill	10-100	Microfill	0.01-0.1
Midfill	1-10	Nanofill	0.005-0.01

After Bayne *et.al.*

In recent years, two further categories of materials were introduced, this time related to variations in their filler loading.

Flowables

Launched in late 1996, in response to dentists' requests, the flowables were developed to improve handling characteristics. The filler particle sizes range from 0.04-1 μ m and there is a reduction in filler content down to 44-54%, thus reducing the viscosity of the material allowing for better flow to poorly accessible areas.

They have a higher resin content so it is understandable that mechanical properties are less than the traditional composites. With a lower modulus of elasticity and better wettability, they are expected to serve as an intermediate layer between the adhesive layer and the overlying resin to reduce contraction stress and consequently improve the seal of the restoration.⁹ Initial indications included a long list but increasing evidence has limited their applications. There may be some benefit from improved flow as long as they are not associated with high stress.¹⁰ Examples include cervical restorations, or as a liner at the gingival margin in the proximal box in a posterior restoration. However, the data to support its use to decrease polymerisation stress or microleakage is not conclusive.^{11,12}

Packables

Shortly after the introduction of the flowables, the packable or condensable composites were marketed as a posterior restorative material with the handling properties of dental amalgam. The filler loading is within the range of 48-65 % by volume, and the filler particle sizes vary from 0.7-20 μ m. The fillers include fused particle agglomerate,

fibrous filler or improved filler particle packing.¹³ These are claimed to impart better physical and mechanical properties so they can be used in load bearing situations. Clinically, the material is less sticky and more viscous making it easier to establish proximal contact. Other reported advantages include placement in bulk and reduced polymerisation shrinkage. However, the data to support claims that they are better than the traditional hybrid materials have not been substantiated.

Properties

Setting time

The setting time of a composite resin depends on the method of activation. Two-paste chemically activated systems have a setting time ranging from 3-6 minutes from the start of mixing. The setting time of a one-paste light activated system is highly dependent on the light source and the exposure time. Light curing time ranges from 3-40 seconds depending on a variety of factors including type and intensity of the light source, the shade and the thickness of the material. A minimum light intensity of 400 mW/cm² has been recommended for routine curing of all dental composites.¹⁴ The use of a higher intensity light will reduce the curing time and increase polymerisation but it may also increase shrinkage and therefore microleakage.¹⁵

Light activated materials continue to polymerise and shrink after removal of the curing light¹⁶ because for both chemical and light activated materials, conversion of monomer to polymer is never quite complete. Approximately 25-40% of double bonds remain unreacted¹⁷ and there is a possibility that these unbonded monomers may be cytotoxic to the pulp.¹⁸

NOTE



- Composite resins can never be over cured
- Curing lights must be checked regularly to ensure the intensity is beyond 400mW/cm²

Depth of cure

Although depth of cure is not a problem with chemically activated materials, it presents a problem for those that are light activated. The presence of a hard surface on a newly placed restoration does not necessarily mean that there has been adequate polymerisation throughout the entire restoration.¹⁹ Inadequate curing of the lower layers of a restoration may result in gap formation, marginal leakage, recurrent caries, pulpal sensitivity and ultimate restoration failure.²⁰

The depth of cure is affected by many factors including

- filler type and composition
- resin chemistry
- shade and translucency
- activator-initiator concentration
- intensity, spectral distribution and duration of light curing²¹

Composite resin restorations should be built in increments no greater than 2 mm thick in order to obtain maximum, uniform polymerisation despite some manufacturers' claims.²² The exit window of the light curing tip must be constantly held within 3-4 mm of the surface of the restoration to obtain optimum effect.

NOTE



- Composites must be cured in increments no greater than 2 mm
- Light curing tips must be held within 4 mm of the surface of the restoration at all times

Thermal properties

Ideally the thermal expansion of a restorative material should be similar to tooth structure in order to maintain the integrity of the interface between the restoration and the cavity wall. The coefficient of thermal expansion of a composite resin is approximately three times greater than tooth structure and this varies with the percentage filler content. Composite resins with lower filler volume, such as a microfil, will therefore have a higher thermal expansion compared to a more highly filled material. Despite this, it seems that few clinical problems arise from this property. It is possible that this may be attributed to the

transient nature of thermal stressors as well as the low thermal conductivity of composite resins. Glass-ionomer bases are often incorporated under composite restorations to reduce the bulk of composite required and therefore, indirectly, the polymerisation shrinkage, rather than for thermal insulation.

Water sorption and solubility

Composite resins have been shown to absorb water and expand but the sorption process takes time and may not be sufficient to compensate for polymerisation shrinkage.

BE AWARE



- Composite resins take up water
- Compomers take up more water than composites
- Giomers take up the most water, leading to greater marginal gap reduction²³

Water sorption is largely dependent on the resin matrix²³ and has been shown to reduce physico-mechanical properties of composite resins including hardness and wear resistance.²⁴ Curing a restoration with an activator light that does not have the full intensity required for proper polymerisation will compromise water sorption and solubility.²⁵ The solubility is also dependent on the resin composition.²⁶

BE AWARE



Composite resins absorb water over time causing marginal staining.

Color stability

The color changes associated with composite resins can be either extrinsic or intrinsic in origin. Extrinsic color changes result from the incorporation of staining agents such as coffee, tea or black cola drinks into the surface of a restoration. This may be related, at least in part, to the surface finish and porosity of the materials. The use of certain mouthrinses and bleaching agents may also cause discoloration of some composites and compomers.^{27,28} The intrinsic color stability of light activated composites is superior to chemically activated ones²⁹ and that of composites is superior to compomers which are more hydrophilic.^{30,31}

Color stability may, to a degree, be shade dependent with lighter shades undergoing a greater colour shift.³² It is also important to note that both composite resins and compomers undergo some degree of color change during light polymerisation³³ so shade selection can be compromised. It is suggested that, at the time of shade selection, accuracy of choice can be enhanced by polymerisation of a small piece of the relevant material on to wet tooth structure adjacent to the cavity.

NOTE



Where aesthetic demands are high, confirm shade selection by polymerising some of the selected resin on to the tooth involved before it has become dehydrated.

Radiopacity

Radiopacity is an important property as it enables the clinician to detect recurrent caries and marginal defects. It is particularly important when the cervical margin of a composite resin restoration is in dentin. Radiopacity is achieved by the incorporation of silicate particles based on the oxides of heavy metals such as barium (Ba), zirconium (Zr) and strontium (Sr). Ytterbium trifluoride is also used in some materials.

Mechanical properties

The mechanical properties of composite resins vary with filler volume percent. This means that increased filler loading will increase hardness, stiffness, strength and fracture toughness. As microfill and flowable composites have lower filler loading than minifill and midifill composites, their mechanical properties are expected to be lower. Therefore the first two should not be used in stress-bearing areas such as the occlusal surfaces of posterior teeth.

However, the lower stiffness or modulus of elasticity is an advantage in the restoration of non-carious cervical lesions where it is desirable that the restoration is able to flex in response to cervical deformation during function or parafunction.³⁴ The mechanical properties of composite resins are generally superior to those of compomers and

giomers especially when exposed to an acid environment.

NOTE

- Composite resins with higher filler volumes generally have better mechanical properties
- In carious or non-carious cervical lesions, composite resins with lower filler volumes are preferred

Wear

Wear occurs whenever two surfaces move in contact with each other. Clinical wear mechanisms can be divided into occlusal contact area (OCA) and contact free area (CFA) wear.³⁵ OCA wear results from the combined effect of direct tooth contact during function and indirect tooth contact via trapped particles during the closed phase of mastication. CFA wear or slurry wear occurs during the open phase of mastication and toothbrushing. In restorations of conservative size, the wear of many current composite resins is similar to that of amalgam being in the range of 10-20 μm a year.³⁶ However, it has been shown that the OCA wear of composites can exceed that of CFA wear by three to five times. Hence, composite resin wear is still a problem for large restorations as substantial wear may lead to changes in functional occlusion and deeper intercuspation (**Chapter 18**).

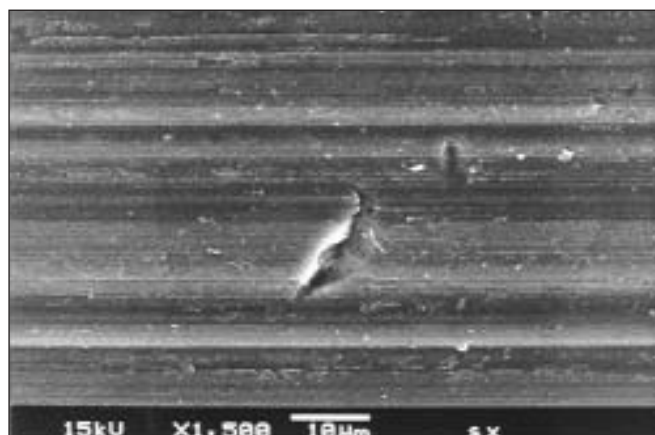


Fig. 12.2. Presence of microscopic cracks that may contribute to catastrophic failure of microfill composites after extensive OCA wear.

The wear resistance of restorative materials is best assessed clinically as it is difficult to simulate the complex loadings, movement and chemical environment of the mouth. *In vitro* wear testing remains popular due to the cost and time involved in running clinical trials, problems associated with patient recruitment/fallout and large variance in clinical data arising from widely differing cavity shapes, locations and stresses. Despite the large number of complex wear devices/tests available, the correlation between *in vitro* and *in vivo* findings is still poor.³⁵ The wear resistance of particular composite resins may also vary between OCA and CFA wear patterns. Due to differences in experimental designs and methods of measurement, the best way to draw conclusions from various studies is to consider the ranking of the evaluated materials within each study. Although the wear resistance of microfill composites is generally better than minifill and midifill composites,³⁷ they appear to fail catastrophically after prolonged fatigue³⁸ (**Figure 12.2**). The wear mechanism of different composites is dependent on material microstructure and a wide variety of conditions including contact stress, duration and chemical environment. In general, the wear resistance of composite resins is superior to compomers and giomers.³⁹

BE AWARE



Composite resin wear is a problem in large stress-bearing restorations.

Polymerisation shrinkage

Despite advances made in resin formulation, polymerisation shrinkage continues to be a problem with all resin-based restorative materials. Shrinkage will vary between 1-5% volume⁴⁰ and can be divided into pre-gel and post-gel phases.⁴¹ During the pre-gel phase, the composite resin is able to flow and stress within the structure will be relieved. After gelation, that is, in the post-gel phase, flow ceases and cannot compensate for shrinkage stresses. This will result in significant stress in the surrounding tooth structure as well as the composite resin to tooth bond.⁴² This can lead to postoperative sensitivity, tooth fracture, microleakage (**Figure 12.3**) and secondary caries.

There are a number of methods available to

reduce the effects of polymerisation shrinkage including

- incremental placement of composite resin in to the cavity
- placement of a glass-ionomer base to reduce the volume of composite required
- glass-ionomer liner to act as a shock absorber
- the development of stronger bonding agents
- the use of 'soft-start' 'pulse cure' or other modified methods of light curing

There are a number of light curing regimens available designed to allow for the composite resin to flow during setting by means of controlled polymerisation. It has been shown that shrinkage stresses can be reduced through some of these techniques but the problem remains. In general, the smaller the filler content, the greater the polymerisation shrinkage. Hence, microfill and flowable composites have greater shrinkage than their more highly filled counterparts. Compomers, by nature of their setting chemistry, absorb water and expand. Although hygroscopic expansion may compensate for polymerisation shrinkage, the process takes time to occur and will allow other problems to develop such as marginal staining of restorations. The ultimate solution to the problem lies in the development of nonshrinking polymers.

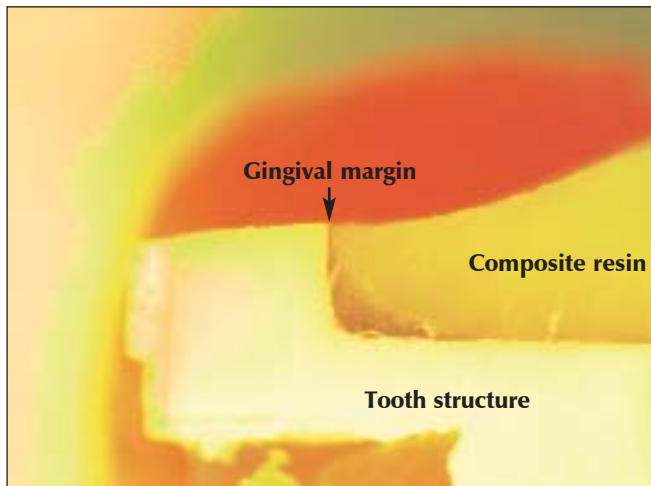


Fig. 12.3. Microleakage at the dentine margins of composite restorations arising from polymerisation shrinkage.

BE AWARE



- Polymerisation shrinkage continues to be a problem with composite resins
- Take care to reduce this clinically during placement
- Shrinkage is generally towards the light source

Bonding to enamel

Bonding of composite resins, compomers and giomers to both enamel and dentine is primarily micromechanical in nature.⁴³ The bond to enamel is entirely micromechanical. The enamel margin should be etched with 37% phosphoric acid for 15 seconds then washed thoroughly to remove all etchant and debris. This results in selective dissolution of the outer surface of the enamel rods leading to the development of microporosities of up to 30 μm in depth. A low viscosity resin is then applied with the expectation that it will penetrate into the microporosities leading to the development of resin-tags. Although resin bonding to enamel is reliable and strong, there are a number of factors of importance that can reduce its efficiency –

- poor clinical techniques
- contamination of the enamel surface after etching
- development of microcracks in the enamel during cavity preparation
- presence of unsupported or fractured enamel margins

Bonding to dentine

Bonding composite resin to dentine is less reliable than bonding to enamel primarily due to differences in composition and structure. The composition of enamel and dentin⁴⁴ is shown in **Table 12.3**. Dentine contains approximately twelve times more water and twice the amount of organic material than enamel. Water competes with bonding agents for substrate surface and can also hydrolyse resin bonds.⁴⁵ While enamel is relatively homogeneous in structure, dentine is heterogeneous with the various constituents unevenly distributed in peritubular and intertubular dentine.

Dentine bonding systems can be divided into three groups based on the mechanism of adhesion.³⁷ The first group aims to modify the smear layer and incorporate it into the bonding process. The smear layer has been defined as ‘any debris, calcific in nature, produced by reduction or instrumentation of dentine, enamel or cementum’.⁴⁶ A second group dissolves the smear layer while the last group completely removes it.

The third group is the one that is commonly in use now and the technique involves the use of three components – acid etchant, primer and adhesive. In the first generation of these systems the components were applied in three consecutive steps. The technique is known as the ‘total etch technique’ and involves etching both enamel and dentine at the same time. The etchant removes the smear layer and also demineralises the superficial layer of dentin, leading to exposure of a microporous scaffold of collagen fibrils. The second step involves the application of a primer that contains hydrophilic monomers with an affinity for the exposed collagen fibrils. The primer also contains hydrophobic monomers to copolymerise with the adhesive which is an unfilled or lightly filled resin. The monomers are usually dissolved in organic solvents, such as ethanol or acetone, which, due to their volatility, displace water from the moist collagen network.⁴⁷ The final step is to apply the adhesive and this engages the exposed collagen and forms resin tags deep in the dentinal tubules and thereby generates a hybrid layer.

To simplify this time consuming three-step procedure, recent innovations have been directed towards combining the various components into two-step or one-step systems (**Figure 12.4**). Primers have been combined with adhesives to form ‘one-bottle’ systems. Etchants have been combined with primers to form ‘self-etching primers’ and all

three components can be mixed together to form ‘all-in-one self-conditioning adhesives’. The latter systems usually employ weaker acids such as maleic, citric, oxalic or nitric acids as well as phosphoric acid in lower concentrations.

An alternate method of effectively bonding composite resin to tooth structure is to place a glass-ionomer as a base, a lining or a bonding agent. There is a variety of materials on the market and the main advantage lies in the ion exchange adhesion of glass-ionomer to tooth structure that will ensure the absence of microleakage. When used as a base a high strength material is placed, trimmed to fit the cavity design, then etched to allow a micromechanical union between the composite resin and the glass-ionomer. On the other hand there is a low powder, low viscosity, light activated material available that can be placed as a bonding agent on the floor and walls of the cavity immediately prior to placement of the composite resin.⁴⁸ Both materials can then be light activated at the same time. These techniques are discussed in detail in **Chapter 11**.

NOTE

- Composite resin bonding to dentine, though significantly improved, still remains a problem
- Three-step smear removing (total-etch) systems are generally more effective than two and one-step systems

TABLE 12.3		
Content	Enamel	Dentine
Inorganic	86% (hydroxyapatite)	50%
Organic	12%	25% (mainly type I collagen)
Water	2%	25%

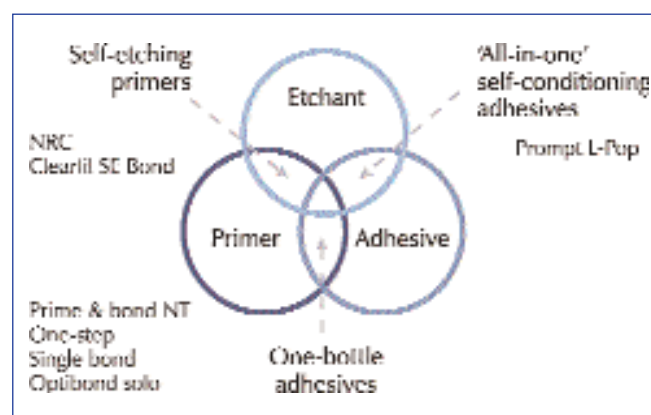


Fig. 12.4. Smear removing bonding systems.

Clinical Considerations

Indications and contraindications

Composite resins were first developed over 40 years ago and have shown considerable progress since. The following are the essential requirements for successful placement:

- the best bond is obtained with enamel around all the margins
- the cavity must be isolated from contamination so preferably use rubber dam
- cavity must be accessible to the activator light for adequate polymerisation



Fig. 12.5a. Indication for the use of composite resin to restore a proximal lesion in an anterior tooth: Old composite resin restorations require replacement.



Fig. 12.5b. The old restorations have been removed with minimal extension of the original cavity.

- contraindications include patients with excessive wear and bruxism

Successful restorations in anterior teeth are shown in **Figures 12.5-11**. Approved applications for restoration of posterior teeth have been set out by the American Dental Association Council on Scientific Affairs⁴⁹ and include its use in small to moderate sized occlusal proximal and cervical lesions.

Selection criteria

Placement of a composite resin restoration is a highly technique sensitive procedure and is therefore heavily dependent on the knowledge and skill of the operator. The whole process from selection of the case, to the cavity preparation to the final finishing demands an understanding of the material and the rationale behind each step of the placement technique.

Preoperative procedures

The tooth to be restored must be cleaned with a paste of pumice and water only to remove plaque and debris to ensure the correct shade is recorded. Proprietary prophylaxis pastes often contain fluoride and are best avoided as they may interfere with bonding to enamel.

Selection of shade

The shade of a tooth is a highly complex issue of the interplay of hue, chroma and value, including metamerism, and the source of the light involved.



Fig. 12.5c. The completed restorations shortly after insertion.



Fig. 12.6a. Indication for the use of composite resin to restore a proximal lesion in an anterior tooth involving the incisal angle: A prior restoration has fractured and requires replacement.



Fig. 12.6b. The completed restoration shortly after replacement.
Case by courtesy Dr. Betty Muk.



Fig. 12.7a. Indications for the placement of composite resins to restore labial cervical lesions on anterior teeth: There are multiple shallow cervical noncarious lesions on these anterior teeth requiring restoration with an aesthetic material.



Fig. 12.7b. The lesions have been restored using composite resin with a dentine bonding agent.



Fig. 12.8a. Reshaping anterior teeth and closing diastema with composite resin: There is spacing between all anterior teeth for this patient who requests closure of the spaces.



Fig. 12.8b. Careful addition of resin on the proximal surfaces resulted in good size, proportion, physiological contours and pleasing aesthetics.



Fig. 12.9a. Modification of anterior teeth using composite resin: The maxillary right central incisor shows a size discrepancy in comparison with its matching neighbour. The lateral incisor is missing and the first premolars are discoloured.



Fig. 12.9b. The size of the central incisor has been corrected, the canine has been reshaped to simulate a lateral incisor and the first bicuspid were veneered to mask discolouration.



Fig. 12.10a. The use of composite resin to disguise discoloured teeth: The patient was unhappy with the colour of the upper and lower anterior teeth. Bleaching was not available at that point in time.



Fig. 12.10b. This subsequent photograph shows 20 direct resin veneers, placed to modify the shade, 14 years after placement. There is only a small amount of labial abrasion.



Fig. 12.11a. The use of composite resin as an immediate repair following trauma: The patient was involved in an accident resulting in fracture of the maxillary central incisors.



Fig. 12.11b. The teeth have been restored with composite resin as an emergency procedure. The restorations will probably last for some time.

Shade guides have serious limitations.⁵⁰ The most reliable technique is to cure a small amount of composite resin on to the actual tooth involved before it has been allowed to dry out (**Figure 12.12**). Many of the materials on the market encourage the use of a layering technique to mimic the combination of enamel and dentine and/or body and incisal shades.

Cavity preparation – guidelines

On the assumption that the disease of caries has been controlled first it is only necessary to remove sufficient tooth structure to allow access to the area that is beyond remineralisation and healing and therefore has to be replaced. There is no prescribed cavity form required. However, as the best and strongest bond is that obtained between composite resin and enamel, it is desirable to maintain an enamel margin around the full circumference of the lesion. The following basic requirements can be prescribed:

- internal cavity form should be rounded to avoid incorporation of stress points
- bevel enamel margins to enhance the seal between composite resin and enamel
- where aesthetics is important enlarge the bevel to provide a smooth transition of composite resin to tooth structure
- do not place a bevel on occlusal margins to avoid allowing thin sections of the restoration to come under occlusal load
- do not place a bevel on the gingival margin of a proximal box if it is in dentine
- access to a proximal lesion on an anterior tooth should be from the lingual to preserve the facial tooth structure and maintain aesthetics

Selection of matrix

The purpose of the matrix is to enhance adaptation of the restoration to the gingival margin and to provide some degree of contour to the proximal surface.

- For an anterior tooth use a mylar or plastic strip wedged firmly to place.
- For a posterior tooth, use a precontoured transparent matrix for preference.
- A sectional matrix with a 'bitine' or 'G-ring'

will generally improve the proximal contour and contact.

Wedging

Wedging a matrix firmly in to place will achieve the following:

- it will assist in maintaining a good contact, particularly in posterior restorations
- it will protect the interproximal gingival tissues
- it will improve the shape, the proximal contour and embrasure space

The value of plastic transparent wedges to assist in directing the activator light into interproximal regions has not been substantiated and clinically it is much easier to work with a regular wooden wedge.

Bonding procedures

As discussed above the development of a micro-mechanical adhesive union between enamel and composite resin is possible providing the procedures are carried out with due care.⁵¹ Bonding to dentine is quite different and, in spite of much research, remains not entirely reliable. Alternatives have been suggested including the placement of a base of glass-ionomer to act as a dentine substitute with composite resin laminated over it to provide strength and aesthetics (**Chapter 11**).



Fig. 12.12. This is the recommended procedure for verification of shade selection prior to commencement of restorative procedures. Place the trial piece of composite resin on the labial of the tooth to be restored before it has become dehydrated by further treatment.

Another alternative has been suggested with the placement of a base of a flowable composite resin followed by a hybrid material over this to provide the required strength and wear resistance. It is suggested that the flowable material will allow for improved adaptation to the cavity floor as well as provide a degree of flexibility and a stress breaker effect.⁵² However, it will not bond with the dentine any better than a hybrid composite resin and the glass-ionomer base remains the preferred option.

The total etch concept discussed above is probably the best resin to dentine bonding system currently available,⁵³ particularly those systems that maintain the separate three-step procedure.

Bonding to enamel

The preferred acid is 37% orthophosphoric acid in a gel form and the application time is 15 seconds. The gel form is preferred because it allows for better control of placement, is easily seen and complete removal can be easily monitored. Clinically, a well etched surface should exhibit a white, frosted appearance indicating porosities to a depth of about 30 μm . This will now be a high energy surface with a greatly increased surface area that is highly receptive to the uptake of the dentine adhesive.

Etching is highly technique sensitive and success depends upon the following:

- type and concentration of acid
- duration of etch
- complete rinsing away of the acid and etching debris
- surface must now remain free of contamination – preferably under rubber dam
- application of low viscosity adhesive will allow optimum penetration
- adhesive must be as thin as possible with no pooling at the margins

Note that the type of enamel can vary so the duration of etching will vary. The enamel of the primary dentition is relatively prismless and enamel that has been subject to heavy applications of fluoride may require longer etch times to achieve the desired white frosted appearance.

The placement of a primer is not required if all margins are in enamel. When priming dentine, it

may inadvertently be applied to the enamel, but this will not affect the bond strength. The preferred adhesive is an unfilled or lightly filled resin of low viscosity that will penetrate deeply into the microporosities in the enamel resulting from the etching process. This is a highly effective micro-mechanical union and, on the assumption the enamel margin is strong, will provide excellent adhesion between composite resin and tooth structure.

Bonding to dentine

As discussed earlier, bonding to dentine is not as straightforward because the composition and structure of dentine is not homogeneous and it is subject to change over time. Secondary, tertiary and sclerotic dentine can be formed under various circumstances and each will respond differently to etching. Relative to enamel it is highly organic and it is subject to constant hydrostatic pressure because of the positive dentine fluid flow. Over recent years several generations of bonding systems have been developed in an attempt to cope with the demanding requirements of a good dentine bond. The current systems are still dependent on a three-step approach although attempts have been made to modify this by combining steps together.

The steps required for effective etching, priming and bonding dentine are discussed above and the results are the same whether the steps are undertaken separately or in combination. The primary aim of demineralisation of the dentine is readily achieved but keeping the collagen fibres standing discretely and ready to be invested with the bond is not easy. It is necessary to maintain some degree of hydration – but not too much – or the fibres will just lie down and resist intake of bond. Having succeeded in developing a hybrid layer,⁵⁴ it has to be recognised that the collagen fibres have been devitalised through etching and, in time, may separate out and release the bond. There is still the possibility of resin tags in the dentine tubules although this assumes that the tubules have been sufficiently dehydrated to allow penetration of the resin at the time of bonding (**Figure 10.8**).

NOTE

The bonding of

- enamel is micromechanical through acid etching and resin bonding
- dentine is through hybridisation through acid etching, priming and adhesive

Placement of composite resin

The effective placement of composite resin depends on two factors:

- control of polymerisation shrinkage
- full cure of the resin in depth

It is essential, therefore, that composite resins are placed incrementally with no greater than 2 mm thickness of resin in any one increment. Each layer must be built one upon the other ensuring that no voids are incorporated in between.

Curing regimens

As mentioned earlier in this chapter, one of the more recent approaches to reducing polymerisation shrinkage stresses is the use of controlled light polymerisation. This can be achieved by

- soft start polymerisation
- pulse delay activation
- pulse cure
- a combination of techniques

Soft start or exponential/step polymerisation involves prepolymerisation at low light intensities followed by final cure at high intensity. This technique has equivocal results with some studies finding no reduction in polymerisation shrinkage,^{55,56} while others have reported a significant reduction.^{57,58} The variations may be attributed to differences in testing methodology, light energy density and/or materials. Hybrid composites appear to benefit most due to their high modulus of elasticity⁵⁹ but the technique does not appear to affect the mechanical properties or degree of conversion of composites.⁶⁰

Pulse delay activation involves an initial pre-polymerisation at a low light intensity followed by a delay period during which the composite restoration is finished and polished. The restoration is then finally cured at high intensity. This technique has been shown to reduce polymerisa-

tion shrinkage and contraction stresses but studies suggest that the degree of conversion may be reduced or, at best, remains unaffected.^{64,65} However, it has also been suggested that polymers cured this way demonstrate increased susceptibility to ethanol softening, reflecting a less cross-linked structure in the final restoration.⁶⁶

The pulse cure technique employs the use of short pulses of light energy throughout placement of the restoration but this technique has not been widely investigated, compared to the pulse delay technique.

NOTE

- Stiffer composite materials may benefit more from the use of controlled light polymerisation
- The clinical durability of composites may be compromised with the use of some controlled light polymerisation techniques

Depth of cure

There are a variety of factors that will affect the depth of cure of an increment of composite resin. It must be noted that, even when great care has been taken to ensure a full cure, no more than 70% of the resin bonds will be fully polymerised. The percentage may increase a little over time but the important period to ensure an acceptable result is the time of placement. So far no curing light will penetrate further than 2 mm into a composite resin so this automatically becomes the maximum thickness of an increment. The factors affecting the depth of cure are as follows:

- thickness of the resin increment
- duration of exposure to the light
- the particle size of the fillers
- percentage of filler by volume
- shade of the composite resin
- distance from light tip to resin
- curing through tooth structure
- diameter of light tip
- efficiency of the curing light

Finishing the restoration

The proper selection and construction of the matrix followed by careful placement of the composite resin increments will limit the amount of

finishing procedures required to complete a restoration. Jefferies⁶⁷ classified the instrumentation required into

- cutting instruments such as tungsten carbide burs
- abrasive instruments – including bonded, coated and loose abrasives
- finishing and polishing devices

In addition, any small proximal or cervical excess can be effectively removed with a #12 scalpel blade. (Figure 12.13) The occlusal contacts are also checked at this final stage.

Longevity of posterior composite resin restorations

There are many published reports on the clinical performance of composite resin restorations and recent reports suggest that the modern varieties last well (Figures 12.14 and 12.15). However, it must be noted that this is highly dependent on a number of factors including the size of the cavity, the position of the tooth in the arch and the occlusal load. The documented failure rate of 0-45 % for a series of studies from 1996-2003⁶⁸ demonstrates clearly that the material needs to be placed selectively and fastidiously (Figure 12.16). It must be noted that a large proportion of these studies are



Fig. 12.13. This demonstrates the use of a #12 scalpel blade to remove excess composite resin at the gingival margin immediately upon completion of a restoration.

short term and it remains for the profession to continuously track their individual results and assess the long term survival.

BE AWARE



The clinical success of composite resin restorations is highly dependent on the operator who must be meticulous right from diagnosis to selection of the material, placement technique and completion of the final restoration.



Fig. 12.14a. Restoration of a small posterior lesion using composite resin: Caries lesions have been identified interproximally at the mesial of the first molar and the distal of the second bicuspid.



Fig. 12.14b. The lesions have been located and limited extension cavities prepared ready for restoration.



Fig. 12.14c. The completed restorations. Note the physiological contours and the aesthetic end result. Courtesy of Dr Pranee Wattanapayungkul.



Fig. 12.15a. Restoration of more extensive lesions with composite resin: There are defective restorations in both the upper bicuspid that need to be replaced.



Fig. 12.15b. The old restorations have been removed revealing further caries.



Fig. 12.15c. The second restoration has been removed showing that there is further caries in the first bicuspid.



Fig. 12.15d. The completed restorations in both teeth. Note the physiological contours. It could be suggested that the translucency of the restorations could be improved. Courtesy of Dr. Pranee Wattanapayungkul.



Fig. 12.16a. A series of composite restorations showing some common modes of failure (the reason for failure is not always obvious but poor clinical handling has to be a primary reason): Worn and discoloured direct composite resin veneers.



Fig. 12.16b. Defective, leaking, discoloured, worn and partially dislodged composite resin restorations showing defective margins with recurrent caries.



Fig. 12.16c. There has been failure of the bond with this restoration leading to dislodgement of the restoration.



Fig. 12.16d. A combination of wear, marginal leakage and a poor colour match indicates the need for replacement of the restoration.



Fig. 12.16e. There is recurrent caries with staining and a marginal fracture leading to a need for replacement of this restoration.

Further Reading

1. Tyas MJ & Burrow MF. Clinical evaluation of a resin-modified glass-ionomer adhesive system: results at five years. *Oper Dent* 2002; 27:438-441.
2. McLean JW, Nicholson JW, Wilson AD. Proposed nomenclature for glass-ionomer dental cements and related materials. *Quint Int* 1994; 25:587-9.
3. Yap AUJ, Mok YY. Surface finish of a new hybrid aesthetic restorative material. *Oper Dent* 2002; 27:161-6.
4. Yap AUJ, Tham SY, Zhu LY, Lee HK. Short-term fluoride release from various aesthetic restorative materials. *Oper Dent* 2002; 27:259-65.
5. Phillips RW. Past, present and future composite systems. *Dent Clinics NA* 1981; 25:209-218.
6. Ferracane JL. Current trends in dental composites. *Crit Rev Oral Biol Med* 1995; 6:302-318.
7. Lutz F, Phillips RW, Roulet JF, Setcos JC. *In vivo* and *in vitro* wear of potential posterior composites. *J Dent Res* 1984; 63: 914-920.
8. Bayne SC, Heyman HO, Swift EJ. Update on dental composite restorations. *JADA* 1994; 125:687-701.
9. Labella R, Lambrechts P, Van Meerbeek B, Vanherle G. Polymerisation shrinkage and elasticity of flowable composites and filled adhesives. *Dent Mater* 1999; 15:128-37.
10. Bayne SC, Thompson J, Swift E, Stamatiades P, Wilkerson M. A characterization of first-generation flowable composites. *JADA* 1998; 129:567-577.
11. Jain P & Belcher M. Microleakage of Class II resin-based composite restorations with flowable composite in the proximal box. *Am J Dent* 2000; 13:235-8.
12. Braga R, Hilton T, Ferracane J. Contraction stress of flowable composite materials and their efficacy as stress-relieving layers. *JADA* 2003; 134 :721-28.
13. Leinfelder K, Bayne S and Swift E. Packable Composites: overview and technical considerations. *J Esthet Dent* 1999; 11:234-249.
14. Rueggeberg FA, Caughman WF & Curtis JW. Effect of light intensity and exposure duration on cure of resin composite. *Oper Dent* 1994;19:26-32.
15. Jain P & Pershing A. Depth of cure and microleakage with high-intensity and ramped resin-based composite curing lights. *J Am Dent Assoc* 2003; 134:1215-23.
16. Yap AU, Wang HB, Siow KS, Gan LM. Polymerisation shrinkage of visible light cured composites. *Oper Dent* 2000; 23: 98-103.
17. Ølio G. Biodegradation of dental composites/glass ionomer cements. *Adv Dent Res* 1992; 6:50-54.
18. Yap AUJ, Saw TY, Cao T, Ng MM. Composite cure and pulp-cell cytotoxicity associated with LED curing lights. *Oper Dent* 2004; 29: 92-99.
19. Pilo R & Cardash HS. Post-irradiation polymerisation of different anterior and posterior visible light-activated resin composites. *Dent Mater* 1992; 8:299-304.
20. Ferracane JL. Dental composites: Present status and research directions. *Second International Congress on Dental Materials* 1993; 45-53.
21. Shortall AC, Wilson HJ & Harrington E. Depth of cure of radiation-activated composite restoratives – influence of shade and opacity. *J Oral Rehabil* 1995; 22:337-342.
22. Yap AUJ. Effectiveness of polymerisation in composite restoratives claiming bulk placement: Impact of cavity depth and exposure time. *Oper Dent* 2000; 25:113-120.
23. Toledano M, Osorio R, Osorio E, Fuentes V, Prati C & Garcia-Godoy F. Sorption and solubility of resin-based restorative dental materials. *J Dent* 2003; 31:43-50.
24. Yap AUJ, Chew CL, Ong KL, Teoh SH. Environmental damage and occlusal contact area wear of composite restorative. *J Oral Rehabil* 2002; 29:87-97.
25. Hofmann N, Renner J, Hugo B & Klaiber B. Elution of leachable components from resin composites after plasma arc versus standard of soft-start halogen light irradiation. *J Dent* 2003; 20:223-32.
26. Ortengren U, Wellendorf H, Karlsson S & Ruyter IE. Water sorption and solubility of dental composites and identification of monomers released in an aqueous environment. *J Oral Rehabil* 2001; 28:1106-15.
27. Lim BS, Moon HJ, Baek KW, Hahn SH, Kim CW. Color stability of glass-ionomer cements and polyacid-modified resin-based composites in various environmental solutions. *Am J Dent* 2001; 14:241-6.
28. Lee YK, Zawahry M, Noaman KM & Powers JM. Effect of mouthwash and accelerated aging on the color stability of esthetic restorative materials. *Am J Dent* 2000; 13:159-61.
29. Schulze KA, Marshall SJ, Gansky SA & Marshall GW. Color stability and hardness in dental composites after accelerated aging. *Dent Mater* 2003; 19:612-9.
30. Folwaczny M, Loher C, Mehl A, Kunzelmann KH, Hickel R (2000) Tooth-colored filling materials for the restoration of cervical lesions: a 24-month follow-up. *Oper Dent* 2000; 25:251-8.
31. Iazzetti G, Burgess JO, Gardiner D, Ripps A. Color stability of fluoride containing restorative materials. *Oper Dent* 2000; 26: 520-5.
32. Uchida H, Vaidyanathan J, Viswanadhan T, Vaidyanathan TK. Colour stability of dental composites as a function of shade. *J Prosthet Dent* 1998; 79:372-377.
33. Yap AUJ, Sim CPC, Loganathan V. Polymerisation color changes of esthetic restoratives. *Oper Dent* 1999; 24:306-311.
34. McGuckin RS, Tao L, Thompson WO, Pashley DH. Shear bond strength of Scotchbond 2 *in vivo*. *Dent Mater* 1991; 7: 50-53.
35. Mair LH, Stolarski TA, Volwles RW, Lloyds CH. Wear: mechanisms, manifestations and measurement. *J Dent* 1996; 24: 141-148.
36. Leinfelder KF, Taylor DF, Barkmeier WW, Goldberg AJ. Quantitative wear measurement of posterior resins. *Dent Mater* 1986; 2:198-201.
37. Lundin SA, Andersson B, Koch G & Rasmusson G. Class II composite resin restorations: A three-year clinical study of six different posterior composites. *Swed Dent J* 1990; 14: 105-114.

38. Lambrechts P, Braem M & Vanherle G. Evaluation of clinical performance of posterior composite resin and dentin adhesives. *Oper Dent* 1987; 12:53-78.
39. Yap AUJ, Tan CH, Chung SM. Wear behaviour of new composite restoratives. *Oper Dent* 2004; 29:277-282.
40. Davidson CL & Feilzer AJ. Polymerisation shrinkage and polymerisation shrinkage stress in polymer-based restorations. *J Dent* 1997; 25:435-440.
41. Davidson CL & De Gee AJ. Relaxation of polymerisation contraction stresses by flow in dental composites. *J Dent Res* 1984; 63:146-148.
42. Feilzer AJ, de Gee AJ & Davidson CL. Setting stress in composite resin in relation to configuration of the restoration. *J Dent Res* 1987; 68:1636-1639.
43. Van Meerbeek B, Inoue S, Perdiago J, Lambrechts P & Vanherle G. Enamel and dentin adhesion. In *Fundamentals of Operative Dentistry*, 2nd ed. Quintessence Publishing; 2001; 236-259.
44. Van Meerbeek B, Perdigao J, Lambrechts P & Vanherle G. The clinical performance of adhesives. *J Dent* 1998; 26:1-20.
45. McLean JW. Dentinal bonding agents versus glass ionomer cements. *Quint Int* 1996; 27:659-667.
46. Ishioka S & Caputo AA. Interaction between the dentinal smear layer and composite bond strengths. *J Prosthet Dent*. 1989; 61:180-185.
47. Jacobsen T, Ma R, Soderholm KJ. Dentin bonding through interpenetrating network formation. *Transactions of the Academy of Dental Materials* 1994; 7:45-52.
48. Peumans M, Van Meerbeek B, Lambrechts P & Vanherle G. Two-year clinical effectiveness of a resin-modified glass ionomer adhesive. *Am J Dent* 2003; 16:363-368.
49. ADA Council on Scientific Affairs: ADA Council on Dental Benefit programs, Statement on posterior resin-based composites. *JADA* 1998; 129:1627-8.
50. Yap AUJ, S Bhole and KBC Tan. Shade match of tooth-coloured restorative material based on a commercial shade guide. *Quint Int* 1995; 26:697-702.
51. Yap AUJ, Ho KS & Wong KM. Comparison of marginal sealing ability of new generation bonding systems. *J Oral Rehabil*. 1998; 25:666-671.
52. Bayne SC, Thompson JY, Swift EJ et.al. A characterization of first-generation flowable composites. *JADA* 1998; 129:567-577.
53. Kanca J. Bonding to tooth structure: A rational rationale for a clinical protocol. *J Esthet Dent* 1989; 1:135-138.
54. Nakabayashi N, Kojima K, Masuhara E. The promotion of adhesion by the infiltration of monomers into tooth substrates. *J Biomed Mater Res* 1982; 16:26-273.
55. Rahiotis C, Kakaboura A, Loukidis M, Vougiouklakis G. Curing efficiency of various types of light-curing units. *Eur J Oral Sci* 2004; 112:89-94.
56. Yap AU, Soh MS, Siow KS. Post-gel shrinkage with pulse activation and soft-start polymerisation. *Oper Dent* 2002; 27: 81-87.
57. Hofmann N, Markert T, Hugo B, Klaiber B. Effect of high intensity vs. soft-start halogen irradiation on light-cured resin-based composites. Part 1. Temperature rise and polymerisation shrinkage. *Am J Dent* 2003; 16:421-430.
58. Lim BS, Ferracane JL, Sakaguchi RL, Condon RL. Reduction of polymerisation contraction stress for dental composites by two-step light-activation. *Dent Mater* 2002; 18:436-444.
59. Rueggeberg FA, Caughman WF, Curtis JW Jr. Effect of light intensity and exposure duration on cure of resin composite. *Oper Dent* 1994; 19:26-32.
60. Bouschlicher MR, Rueggeberg FA. Effect of ramped light intensity on polymerisation force and conversion in a photoactivated composite. *J Esthet Dent* 2000; 12:328-339.
61. Chye CH, Yap AU, Lai YC, Soh MS. Post-gel shrinkage associated with different light curing regimens. *Oper Dent* (accepted for publication).
62. Sahafi A, Peutzfeldt A, Asmussen E. Effect of pulse-delay curing on in vitro wall-to-wall contraction of composite in dentin cavity preparations. *Am J Dent* 2001; 14:295-296.
63. Suh BI, Feng L, Wang Y, Cripe C, Cincione F, de Rijik W. The effect of the pulse-delay technique on residual strain in composites. *Compend Contin Educ Dent* 1999; 20(S):4-12.
64. Hackman ST, Pohjola RM, Rueggeberg FA. Depths of cure and effect of shade using pulse-delay and continuous exposure photo-curing technique. *Oper Dent* 2002; 27:593-599.
65. Asmussen E, Peutzfeldt A. Influence of pulse-delay curing on softening of polymer structures. *J Dent Res* 2001; 80:1570-1573.
66. Soh MS, Yap AU. Influence of curing modes on crosslink density in polymer structures. *J Dent* 2004; 32:321-326.
67. Jefferies SR. The art and science of abrasive finishing and polishing in restorative dentistry. *DCNA* 1998; 42:613-627.
68. Brunthaler A, Konig F, Lucas T et al. Longevity of direct resin composite restorations in posterior teeth. *Clin Oral Investig* 2003; 7:63-70.

13 Dental Amalgams

R. W. Bryant

An amalgam is an alloy of one or more metals with mercury. Dental amalgam refers to a particular type of amalgam and, until recently, it was the most commonly used and one of the oldest dental restorative materials. It is the product of an amalgamation reaction between particles of an alloy, containing essentially varying amounts of silver, copper and tin, and possibly traces of other metals, with mercury.

There is still no adequate, economic alternative for dental amalgam as a restorative material for a moderate sized caries lesion in a high load bearing area.

The combination of reliable long-term performance in load bearing situations and cost per unit restoration is unmatched by any other dental restorative material.



Description of Dental Amalgam

Although, for much of the 20th century, dental amalgam was overwhelmingly the principal restorative material used in dentistry, its use has declined since the 1980s. This reflects

- improvements in methods of prevention and control of caries,
- an emphasis on conservation of tooth structure and the development of new restorative materials and techniques,
- altered perceptions by patients seeking toothcoloured restorations and their acceptance of a shorter life span for a restoration,
- negative emphasis on the mercury content of dental amalgam. The potential for allergy to or toxicity from the mercury content has been recognised for many years and intense investigations and research have been undertaken. Currently, it appears likely that any restriction in the use of amalgam will be related to environmental concerns rather than to the health of individual patients and there appears to be no justification either to cease using it or to replace it entirely in the hope of altering a patient's state of health.

Currently, amalgam is being used less frequently overall, down from 67% of all direct restorations placed in Australia in 1983 to 34% in 1998. It is now being more selectively used for larger, multiple surface restorations and cuspal coverage.

Extensive clinical studies have provided information on performance, longevity and reasons for failure, and laboratory tests have lead to a greater understanding of the structure and performance. Dental amalgam has always been regarded as a very tolerant material that will perform well in spite of great variations in placement techniques.

Terminology and types of dental amalgams

Dental amalgam alloy refers to the combination of metals, such as silver, tin, copper and sometimes zinc, indium, palladium or platinum. Since the 1960s the so-called high copper amalgams have

largely replaced high silver containing alloys that had changed little in composition since the 1890s.

A range of amalgam alloys is available commercially and these may be distinguished from each other by the following descriptors:

Copper content

Low copper amalgam alloys have a total copper content less than 6%. The formula is approximately 70% Ag, 26% Sn, 3-4% Cu with some minor elements. Until the mid 1960s, almost all dental amalgam alloys were of this type.

High copper amalgam alloys have a total copper content greater than approximately 12%. Most modern alloys are in this range, containing approximately 41-61% Ag, 28-31% Sn, 12-27% Cu and some minor elements. They have superior physical properties and clinical performance relative to the low copper amalgams mainly because of the absence of a tin/mercury (γ -2, γ_2) reaction product and therefore reduced corrosion and creep.

Zinc content

Alloys containing more than 0.01% zinc are described as zinc containing and those with less than 0.01% zinc are referred to as zinc free. Zinc was originally included in the initial melt for an alloy because it acted as an oxide scavenger to produce clean castings of the ingot. Modern techniques of casting into an inert atmosphere have

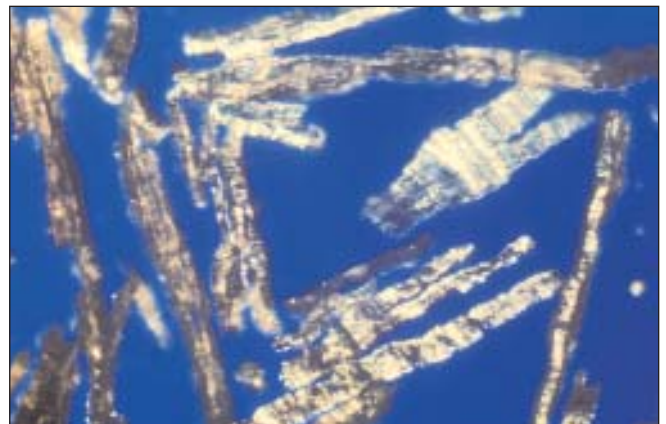


Fig. 13.1. An old style lathe cut amalgam alloy. Note long fragile 'sticks' of alloy to be broken into shorter lengths during trituration. Note also finer particles of dust.

simplified the production of zinc free alloys. Amalgams that contain zinc appear to exhibit a lower rate of margin fracture under clinical loading but they tend to exhibit an excessive delayed expansion if contaminated with moisture during placement.

Minor elements

A number of elements, such as indium, palladium and platinum, may be included in the alloy in minor quantities. Although the proportion of these is usually less than 1%, reference is frequently made to the presence of one of these elements in advertising the alloys.

Gamma 2-phase content (γ_2)

Amalgams may be described as γ_2 -containing or γ_2 -free. Low copper amalgams contain the Sn-Hg reaction product which is called the γ_2 phase to distinguish it from the γ phases of the Ag-Sn and Ag-Hg alloy systems. This reaction product is slow-setting, weak and corrodes easily. Within several hours after amalgamation, all correctly manipulated high copper amalgams are γ_2 free.

Particle shape and type

There are two particle types depending on method of manufacture (**Figures 13.1-13.3**).

- lathe cut (or chip) refers to the irregularly shaped filings produced by cutting an ingot of alloy on a lathe. Prior to cutting, the ingot

is traditionally homogenised to produce an alloy with a Ag-Sn phase (γ) as well as some regions of Cu_3Sn (ϵ)

- spherical particles are produced by atomising the alloy, whilst still liquid, into a stream of inert gas. They are usually not subjected to an homogenising heat treatment and therefore contain Cu_3Sn (ϵ) finely dispersed in a Ag-Sn (γ) matrix

Some alloys contain a blend of lathe-cut particles and spherical particles and, depending on the proportion of each in the mix, the handling properties, particularly the packability will be modified. Packability is a clinical characteristic referring to the resistance offered by the amalgam to packing loads during amalgam placement.

Classification of alloys and amalgams

Based on the copper content, the shape of the particles and the major elements present in the spherical particles of a blended particle alloy, dental amalgam alloys and their corresponding amalgams can be simply classified into six types, namely

- low copper, lathe cut
- low copper, spherical
- high copper, lathe cut
- high copper, spherical
- high copper blend, Ag-Sn-Cu
- high copper blend, Ag-Cu

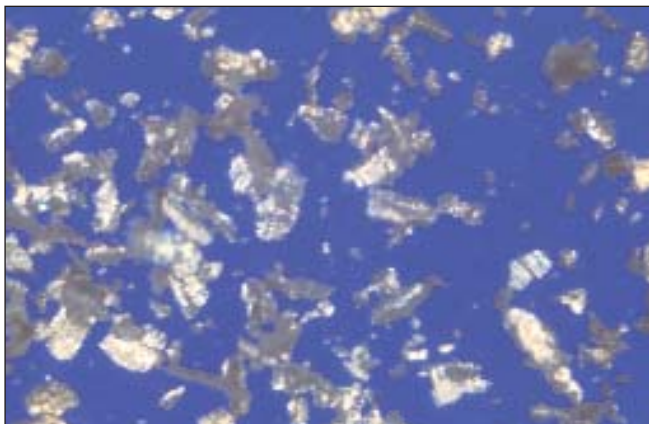


Fig. 13.2. Regular sized particles of a fine grain alloy. Long filings have been reduced for easier machine trituration. There are still fine dust particles.

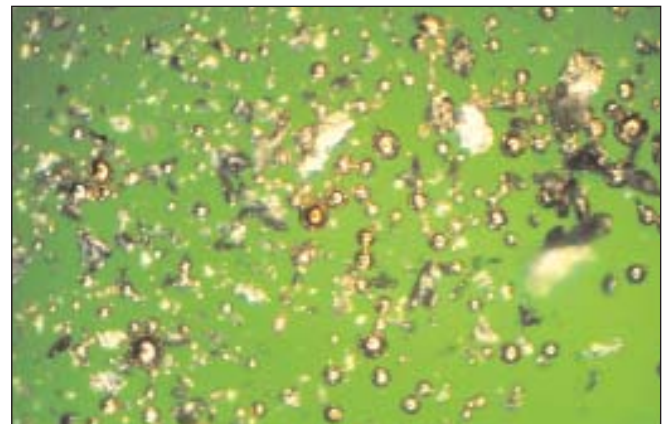


Fig. 13.3. An alloy with a mixture of regular and spherical particles. A spherical alloy is easy to pack but some rough cut alloy is included to add resistance to the plugger.

The two types of blended particle alloys contain both lathe cut and spherical particles and the spherical particles, which have a high copper content, mainly contain either Ag, Sn and Cu or Ag and Cu. Examples of four types are illustrated in **Figure 13.4**.

There has been confusion over the terminology

used with these different types but the above classification is preferred. The first two have been called 'conventional' or 'silver tin' and the 'high copper spherical' has also been known as 'single melt high copper' or 'ternary'. Terms such as 'dispersion modified' and 'Ag-Cu dispersed' have been applied to the last one and a general label of

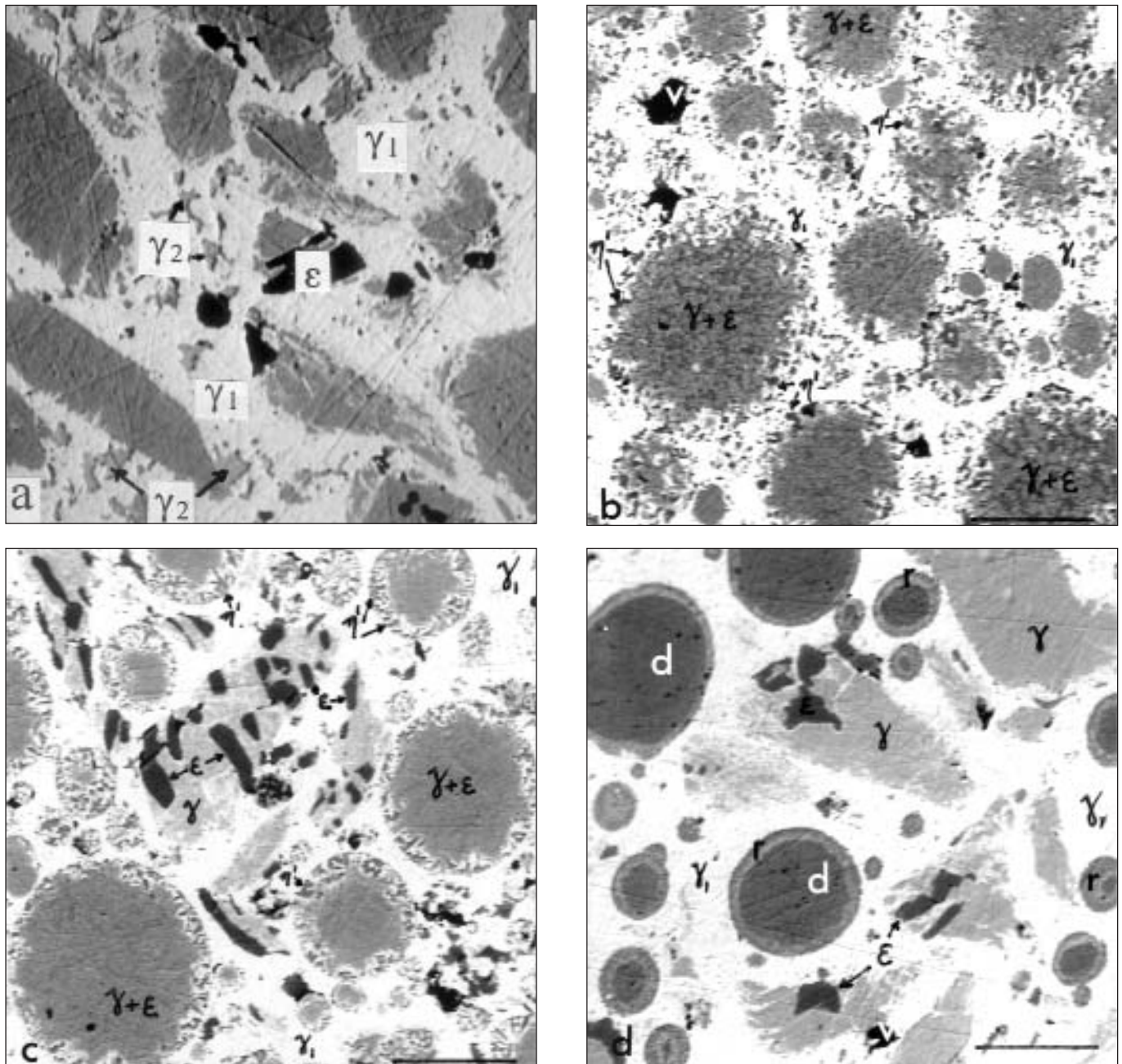


Fig. 13.4. Back scatter electron photomicrographs of a group of amalgams. **a** Shows a low copper lathe cut alloy, **b** is high copper spherical alloy, **c** is a high copper blend, Ag-Sn-Cu alloy (Permite C-SDI) and **d** is a high copper blend Ag-Cu alloy. The phases shown include γ , ϵ , d , η' , γ_1 , γ_2 , v (void), r (reaction zone, alloy containing γ_1 and η' phases). Bar lines indicate 20 μm .

‘admixed’ has been applied to all alloys containing both lathe cut and spherical particles.

The range of high copper amalgam alloys available, with different handling characteristics, ensures that there is no longer any justification for the clinical use of a low copper amalgam.

Amalgamation and structure

Phases present in unreacted alloy

When mercury reacts with the particles, the metals in the alloy are usually present as phases which approximate stoichiometric composition and are shown in **Table 13.1**. Two phases, γ and ϵ are present in all unreacted alloys and a third, the dispersant component (d), is only present in the ‘high copper blend, Ag-Cu’ type of alloy.

Components of the amalgamation reaction

In amalgamation, mercury comes into contact with the surface of the alloy particles and approximately 3-5 μm of the surface of each particle undergoes a reaction. The metals in the particles are combined together in phases and these go into solution in the mercury and then precipitate out in the form of new reaction products. Two of these (γ_1 and η^1) are present in all amalgams and the third product, the γ_2 phase, is only present in low copper amalgams.

The product of this reaction becomes the matrix which, once it is set, holds the remaining particles together as a coherent mass.

Initial mercury content

Depending on the shape, size and composition of the alloy particles, the amount of mercury required to provide good amalgamation can vary from 40-53% by weight. Low copper microfine lathe cut particle alloys require a relatively large amount of mercury initially and high copper spherical alloys require the least. The final mercury content is influenced by the initial content and the clinical technique used when placing the amalgam and

is approximately 37-48%. Retention of excess mercury in the matrix will lead to loss of physical properties. A lack of dispensed mercury, or faulty trituration, will lead to failure to wet the surface of all the particles, a weak particle/matrix interface and a reduction in physical properties

Structure of the set amalgam

Providing the minimal amount of mercury, commensurate with complete amalgamation, is incorporated initially, and then proper condensation techniques are carried out, approximately 35-50% of the final volume of the set amalgam will consist of unreacted portions of alloy particles, held together by the γ_1 (or Ag-Hg) phase matrix.

Clinically, amalgams deteriorate with time as a result of corrosion. Corrosion products will be formed within the amalgam as well as on the surface and Sn-Hg phase may be lost from low copper amalgams. A clinically significant result of corrosion is an increase in the number of voids in the amalgam.

Properties

Corrosion

Corrosion is defined as the electrochemical destruction of a metal by reaction with its environment. Corrosion is the single most important property of amalgam influencing the clinical

TABLE 13.1: Composition of the major phases in dental amalgam

Code	Phase or component	Weight%			
		Ag	Sn	Cu	Hg
γ gamma	Ag ₂ Sn	73.2	26.8		
ϵ epsilon	Cu ₃ Sn		38.4	61.6	
d dispersant	Ag-Cu eutectic	71.9		28.1	
γ_1 gamma 1	Ag ₂ Hg ₃ Ag ₂₂ Sn Hg ₂₇	30.7	1.8		67.5
γ_2 gamma 2	Sn _x Hg _y		82.5		17.5
η^1 eta prime	Cu ₆ Sn ₅		60.9	39.1	

longevity of the restoration. Amalgam restorations may undergo one or more types of corrosion.

Tarnishing

Tarnishing arises from surface corrosion caused by oxidation of the Sn-Hg phase in low copper amalgams or the Cu-containing phases in high copper amalgams with the formation of a film of oxides, sulphides and hydroxides. Polarisation may also take place with breakdown of the film and formation of a corrosion product leading to pitting and roughening of the surface.

Crevice corrosion

A differential oxygen concentration cell may develop at the tooth/restoration interface resulting in the surface facing the cavity walls becoming anodic in relation to the outer surface of the restoration. Selective attack on phases in the amalgam, as well as the release of tin and copper, result in the formation of corrosion products containing tin and copper as well as Ca, Cl, Fe, S and Zn. These become lodged in the crevice and can seal the interface producing the so-called selfsealing of the margin between the amalgam and the cavity wall.

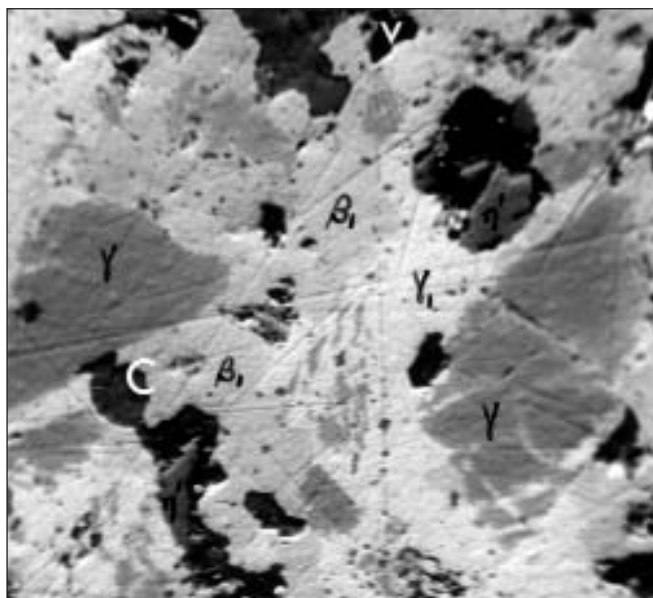


Fig. 13.5. Corroded amalgam: A clinically-aged amalgam showing corrosion. **c** is chloride containing corrosion product, β_1 = Sn rich AgHg matrix phase. Note the absence of γ_2 phase in this low copper amalgam.

Corrosion fatigue

This occurs particularly around the margins of restorations. Fine, branchlike penetrations, having the appearance of fatigue cracks, extend along the grain boundaries of the γ_1 matrix phase in regions subjected to occlusal load.

The γ_2 phase is most susceptible to corrosion in low copper amalgams and the process extends through the restoration via the γ_2 phase, between grains of γ_1 phase, and through voids. In high copper amalgams the η^1 , d, e (and possibly the γ_1) phases appear to corrode concurrently. Corrosion is a slower process in high copper amalgams and extends along the grain boundaries of the γ_1 phase (**Figure 13.5**).

Galvanic corrosion

Contact between dissimilar metals within an electrolyte may lead to galvanic corrosion. Saliva will act as an electrolyte between amalgam and cast gold, between amalgam and prosthodontic alloys, and between fresh amalgam and old amalgam. Occasionally, this can cause sharp pain shortly after placement of a new amalgam restoration sufficient to require its urgent removal. There will also be the potential for longterm corrosion with pitting and roughening of adjacent metal restorations. This means that the margin of a crown prepared over an amalgam core should be placed on tooth structure beyond the gingival extent of the amalgam.

SUMMARY



Corrosion

All amalgams always corrode

- tarnish
- crevice corrosion
- crevice fatigue
- galvanic corrosion

High copper amalgam shows controlled corrosion
Conventional amalgam corrodes more rapidly

Creep

Creep is progressive permanent deformation of a set amalgam under load. Low copper amalgams show high creep values of greater than 2.5% which

can be associated clinically with greater margin fracture.

The presence or absence of the γ_2 phase is the principal factor influencing creep. The presence of the γ_2 phase enhances the ability of the γ_1 phase grains to slide under load, particularly when the γ_1 grains are small. High copper amalgams, with no γ_2 phase and with creep resisting η^1 crystals at γ_1 grain boundaries, show less than 0.2% creep at seven days.

Strength

The importance of laboratory testing of the strength of an amalgam in determining clinical success or failure is uncertain, although some tests may be representative of the clinical situation. Compressive strength and transverse strength values are shown in **Table 13.2** for products that are representative of four amalgam types. Operator variables may significantly influence the achievement of these values.

High copper spherical amalgams have the highest physical properties of all amalgams at one hour after placement and this may be of clinical importance if the restoration is to be subjected to early load. However, they are not fully set at this time and it is unwise to subject a new restoration to immediate stress. The tensile strength is only about 20-38 MPa at 1 hour so there is a need for care when checking the occlusion. At seven days the properties of high copper amalgams are substantially greater than for low copper amalgams. The transverse strength relates directly to the final mercury content and this may be of clinical importance in large restorations which are to be subjected to heavy occlusal loads.

BE AWARE



Operator variables will be the major influence on all physical properties.

Rigidity

The rigidity of restorative materials is an important property if they are to be used in load bearing sites. The modulus of elasticity of high copper amalgams (approximately 55 GPa) far exceeds that of glass-ionomers and composite resins (3-5 GPa and 4-16 GPa, respectively) and may be contrasted with dentine (18 GPa) and enamel (82.5 GPa).

Fatigue strength

This is the response to repeated loading at relatively low subfracture loads for extended periods of time. High copper amalgams show low creep and take extended periods of time before fracturing. If fracture does occur it is preceded by very little bending.

Thermal expansion

Amalgam has a linear coefficient of thermal expansion of $25 \text{ mm/mm}^\circ\text{C} \times 10^{-6}$, compared with glass-ionomers (10-11), composite resins (30-60) and tooth structure (11.4). The larger the coefficient, the greater the dimensional change of the material following change of temperature.

Dimensional change

Most amalgams exhibit a slight contraction on setting and this is of relatively minor clinical significance. Zinc containing amalgams, particularly low copper amalgams, may exhibit delayed

TABLE 13.2: Significant physical properties of amalgams in common use

Amalgam type	Comprehensive strength in MPa		Transverse strength in MPa 1 week	Modulus of elasticity (GPa)	Static creep (%)
	1 hour	1 week			
Low Cu – lathe cut	155	390	139	41	2.5
High Cu – spherical	325	590	148	53	0.1
High Cu – blend, Ag-Sn-Cu	190	570	142	56	0.1
High Cu –blend, Ag-Cu	190	500	122	52	0.2

Table 13.2.

The clinical significance of differences between high copper amalgams is uncertain.

expansion about 3-5 days after placement reaching in excess of 400 $\mu\text{m}/\text{cm}$. It appears to be the result of incorporation of water during clinical handling leading to an electrolytic reaction between the water and zinc. Hydrogen gas will be generated within the amalgam leading to expansion out of the cavity (**Figure 13.9**), increased marginal leakage and pain.

BE AWARE



All amalgams contract minimally on setting. Expansion is generally caused by moisture contamination.

Table 13.3 shows a comparison of the dimensional change associated with several different amalgams following condensation with or without water contamination. All contaminated amalgams exhibit increased tarnish and zinc-containing amalgams will undergo excessive delayed expansion.

Clinical Manipulation

Cavity design

There is not a great deal of latitude available in the design of a cavity for restoration with amalgam. G. V. Black laid down a set of principles and many of those still apply although not so rigidly. Extension for prevention is out of date but elimination of the disease is essential because

amalgam has limited resistance to margin penetration of bacteria and has no bioactivity. Inclusion of fluoride is of no benefit because there is no water available to allow ion migration. In the absence of adhesion it is generally necessary to remove unsupported enamel around the margins and it is important to try to develop margins where the angle of the amalgam margin will be at least 70° to limit the potential for fracture (**Figures 13.6-13.8**).

Trituration

Trituration (or mixing) of the alloy with the mercury is carried out in a mechanical mixer (amalgamator) with the alloy and mercury in single use (ideally) or reusable capsules. The object is to wet the entire surface of the alloy particles with the mercury, to bring about the process known as amalgamation. The effectiveness of the mixing process will be influenced by the speed of the particular unit, the length and the direction of movement of the capsule in the machine, the presence of a pestle in the capsule and the length of time of mixing.

The clinician can determine the maximum and minimum mixing times for a new amalgam alloy in a particular machine by carrying out several trial mixes. If, after mixing, the amalgam feels hot to the hand or is difficult to remove from the capsule, the mixing time should be reduced. To set a

Table 13.3. There are serious side effects from water contamination during condensation, particularly if the alloy contains zinc. (Adapted from Nelson and Mahler 1990.)

TABLE 13.3: Effect of water contamination					
Amalgam	Type	Zinc (%)	Dimensional change ($\mu\text{m}/\text{cm}$) with time		
			Dry 3 month	Wet 1 week	Wet 3 month
Low Cu – lathe cut	N	0.93	-13.3	+39.1	+286
High Cu – spherical	T	0.03	-15.2	-9.2	-11.7
High Cu – blend, Ag-Sn-Cu	V	0.04	-9.2	-8.4	-13.3
High Cu – blend, Ag-Sn-Cu	P	0.18	-0.4	+3.0	+5.5
High Cu –blend, Ag-Cu	D	0.97	-12.5	+2.1	+26.6
N= New True Dentalloy (SS White); T=Tytin (Kerr); V=Valiant PhD (Caulk/Dentsply); P= Permite C (SDI); D= Dispersalloy (J&J)					

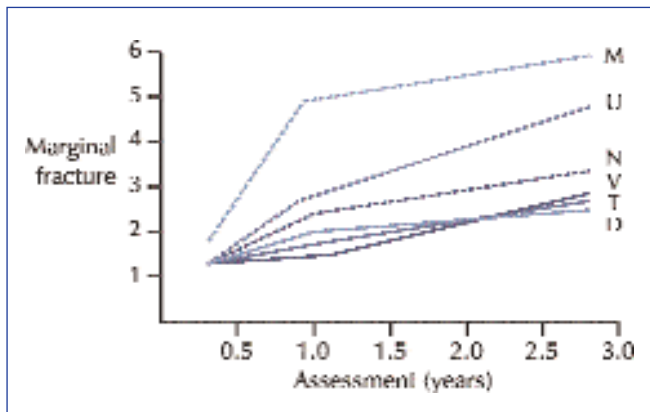


Fig. 13.6. Increase in marginal fracture over time:

M=microfine low copper lathe cut; U= Low copper spherical; N=fine particle low copper lathe cut; V=high copper blend Ag-Cu-Sn; T= high copper spherical; D= high copper blend Ag-Cu.

minimum time limit, drop the freshly mixed mass on to the bench from a height of approximately 30 cm. If the mix is dry and crumbles on hitting the bench, the trituration time should be increased. A well-mixed amalgam should stay together when dropped but should be a little flattened and retain a wet gloss on the surface. It is unwise to substantially change the trituration time in an attempt to modify working time. Manufacturers control the working time of each alloy and it is generally preferable to use their recommendation.

It is also better to slightly over triturate rather than under triturate an amalgam. The following should be kept in mind:

- prolonged trituration may reduce plasticity, shorten working time and increase final contraction
- inadequate trituration may result in incomplete wetting of the surfaces of the alloy particles by the mercury, a weak interface between the matrix (γ_i) and the particles, lower strength, increased porosity, a rougher surface and increased corrosion

Although it is preferable, from the perspective of mercury hygiene, to use single use, predis-

SUMMARY

Test for correct mix

Over trituration

- alloy will be hot
- hard to remove from capsule
- shiny wet and soft

Under trituration

- alloy will be dry
- will crumble if dropped from 30cm

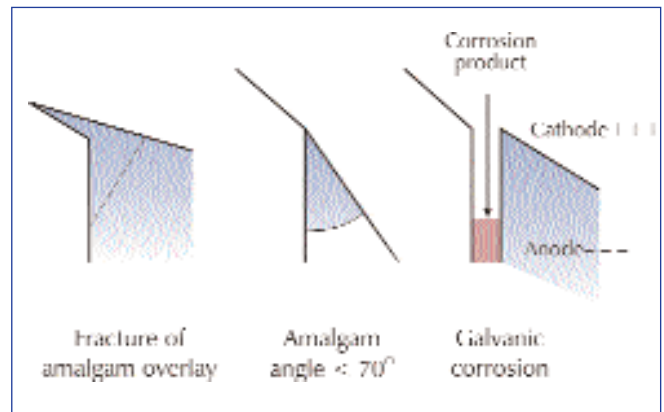


Fig. 13.7. Some reasons for failure of amalgam margins:

The main reasons for failure of amalgam/tooth interface are shown but corrosion products will seal the interface and recurrent caries is unlikely to arise.



Fig. 13.8. Three amalgams showing different degrees of marginal failure, none of which is necessarily clinically significant.

pensed capsules, some clinicians prefer alloy pellets/powder and mercury dispensed in reusable capsules. In this case, the lowest possible mercury proportion, in accordance with the manufacturer's instructions should be used, so that excess mercury does not have to be removed after trituration and before placement into the cavity. Dispensing and trituration of the mass must be aimed at ensuring optimum plasticity. Probably no other characteristic has as much influence on the adaptation of the amalgam to the cavity floor and walls thus optimising the seal and minimising postinsertion sensitivity.

BE AWARE

Essential requirements for correct placement

- proper plasticity
- minimal mercury content

Packability

Packability refers to the resistance offered by the amalgam to the forces of condensation used in placement. Amalgams can be described as offering a high or low degree of packability and this will vary according to the size and proportional distribution of lathe cut or spherical particles. Amalgams containing only spherical particles are relatively easy to pack because they move readily from under the condenser. Use a larger packing instrument with these amalgams. A substantial load will still be required to achieve proper adaptation to the cavity and to eliminate as much mercury-rich material as possible during condensation.

Purpose of condensation

Condensation refers to the incremental placement of the amalgam into the prepared cavity and compression of each increment into the others to form a continuous homogeneous mass.

The aims of condensation are to

- adapt amalgam to the margins, walls and line angles of the cavity
- minimise voids and layering between increments within the amalgam
- develop maximum physical properties
- remove excess mercury to leave an optimum alloy/mercury ratio

It is best carried out using hand instruments with a smooth flat face which can deliver reasonable force, per unit area, and compress the layers together. This means that the smaller the diameter of the face of the plugger the greater the pressure that can be applied.

Mechanical condensers are available and reduce the need for application of load. However, they tend to lead to unreliable condensation, as well as the generation of heat and mercury vapour, both of which are undesirable. Ultrasonic condensation is unacceptable because of the release of mercury vapour.

Speed of placement

Immediately amalgam is triturated, phase formation commences and the setting reaction is under way. It must be used in a plastic state so there should be no delay between trituration and condensation. No amalgam should be placed more

than three minutes from the start of mixing. Attempting to condense a partly set amalgam into a cavity will result in poor adaptation, increased post insertion sensitivity, reduced marginal seal, layering between increments, a weak restoration, and increased potential for corrosion.

Moisture contamination

Keep the amalgam completely free from moisture contamination during the entire condensation procedure. The newly mixed amalgam must not be touched by bare hands and the cavity must be completely dry and free of gingival seepage or haemorrhage. For preference, work under rubber dam. Inclusion of water will lead to increase in corrosion and tarnish with a reduction of physical properties. A zinc-containing amalgam will, in addition, develop a delayed expansion and rise out of the cavity or show localised 'bubbles' on the surface (**Figure 13.9**).

BE AWARE



Avoid contamination of amalgam during placement into the cavity

- use rubber dam

Contamination with moisture at this stage results in

- increased tarnishing of all amalgam types
- increased corrosion of all amalgam types
- excessive delayed expansion of zinc-containing amalgams



Fig. 13.9. Result of moisture contamination during placement of the amalgam into the cavity. There is evidence of 'bubbles' on the occlusal of the restoration resulting from gas formation.

Need for a matrix

Because of the need for appropriate condensation pressures, it is essential to use an effective matrix for any cavity that is not entirely enclosed within four walls. A variety of metal matrices is available with different kinds of retainers and no particular one is superior to the others. The important point is that the matrix strip be metal and of sufficient size for the particular task. It must be adapted tightly to the gingival margin, using a wooden wedge, to minimise the risk of developing an overhanging excess of amalgam beyond the confines of the cavity. Firm wedging will also slightly separate the teeth and this will compensate for the thickness of the matrix band and ensure firm proximal contact between the restoration and the adjacent tooth. Redevelopment of the proximal contour of a posterior tooth is difficult at the best of times and calls for considerable clinical skill.

If an excessive amount of tooth structure is missing it is possible to reinforce the matrix strip with greenstick compound. First place and contour the matrix as best possible and support the gingival margin with a wedge. Then take a short length of greenstick compound and soften one end in a bunsen burner. Condition the hot end in hot water momentarily then push the softened material in to the proximal area of the matrix and mold it to shape to offer maximum support as it sets.

Following successful restoration of the cavity with amalgam, heat the tip of a No. 6 flat plastic instrument and push it into the compound to soften it before gently levering it away from the matrix, taking care not to overload the newly setting amalgam.

Placement

Place the amalgam into the cavity in relatively small increments and condense rapidly on to the walls and in to the line angles using appropriate load of short duration. Pack laterally as well as vertically to ensure complete adaptation, particularly into the angles of the cavity. Use a smooth faced condenser of appropriate size.

SUMMARY



Placement of amalgam

- complete within 3 minutes of mix

Condense with

- smooth flat pluggers
- lathe cut – use small pluggers
- blended – use small flat pluggers
- spherical – use largest plugger to fit cavity

Use optimal load at all times.

The appropriate load to apply for a particular amalgam is related to the type of amalgam and the size of the condenser. Always remove the mercury-rich surface of the last increment before placing a subsequent load and increase the size of the condenser when packing excess beyond the confines of the cavity.

When placing very large restorations (e.g. cuspal coverage), large increments of amalgam may be placed providing each increment is spread widely so that it is thin and can be thoroughly condensed.

First burnish (precarve burnish)

On completion of condensation carry out a pre-carve burnish using a large burnisher for 15 seconds. Use light force and move from the centre of the restoration outwards across the margins. This will bring further mercury to the surface, which must be removed, as well as improving adaptation to the cavity margins.

Carving

Carving instruments must be of appropriate shape and very sharp so as to create minimum disruption to the amalgam at the margins. Do not carve a deep or sharp occlusal anatomy but use the remaining enamel as a guide and take care to maintain marginal ridges and occlusal spillways. Deep, sharply carved patterns on the occlusal surface may look nice but they act as crack initiators and are undesirable. Inadequate bulk of amalgam is the principal cause of amalgam fracture. It is also very easy to over carve and take a restoration out of contact with the opposing tooth thus altering occlusal relationships leading to over-eruption

and lateral interferences. However, well defined marginal ridges and lateral spillways will dissipate load from the occlusal table and lead the food bolus away from contact areas.

- Remember that excess flash left over the surface of the cusp inclines will be fragile and will break away easily, usually leaving a crevice or ditch at the margin that is liable to gather plaque and therefore make the enamel below susceptible to further demineralisation.
- Always be prepared to adjust the occlusal anatomy of the opposing tooth to avoid developing deep intercuspation between the arches because this may lead to interferences during lateral and protrusive excursions and prejudice the longevity of the restoration or the tooth. It is wise to anticipate this before cavity preparation and advise the patient accordingly at that time.

SUMMARY



Completion of placement

- finish packing and burnish
- carve to final contour
- final burnish to close margins
- polish to matte finish only

Final burnish (postcarve burnish)

Following carving, check the occlusion, particularly in relation to lateral excursions and then carry out a brief final burnish. Use a large burnisher at a low load and burnish outwards across the margins to finally adapt the amalgam at the margin on both the occlusal and the proximal surfaces where access permits.

Finishing

The amalgam restoration should be finished at a later appointment to

- refine the amalgam/tooth margin,
- adjust occlusal contacts,
- refine (but not deepen) occlusal anatomy,
- smooth the surface of the restoration to minimise plaque retention. Note that a high gloss is not required and over polishing represents poor mercury hygiene.

Do not increase the depth of fissures or increase the angle of the cuspal inclines because this may increase margin failure over time. Carry out all finishing procedures under an air/water spray to minimise the generation of heat because that may lead to the liberation of mercury vapour (**Figures 13.10-12**).

Clinical Aspects of Amalgam Restorations

Adaptation and seal at the margins

The clinical experience of sharp sensitivity to cold stimuli, particularly during the first 3-5 weeks after placement, is usually associated with incomplete adaptation to the cavity walls and a lack of seal at the restoration/tooth interface. Occasionally, postinsertion sensitivity can also be associated with an occlusal interference, so post-insertion review of the occlusion is essential. In the long term, a lack of adaptation may be associated with marginal deterioration, accumulation of debris, recurrent caries and pulpal reactions.

Manipulative techniques influencing adaptation

Generally speaking, blended particle and lathe cut amalgams have better adaptation and seal than spherical particle amalgams. The following manipulative factors will all have a bearing on the efficiency of the initial marginal seal:

- Over-trituration may result in reduced plasticity as the amalgam may set too quickly leading to poor adaptation and excessive contraction on setting.
- Good condensation is essential to adapt the amalgam to the cavity walls and margins.
- Spherical amalgams can be condensed using larger condensers and do not require the same load to achieve optimum physical properties.
- Lathe cut and blended particle amalgams require smaller diameter condensers, more care and a heavier load to achieve the optimum degree of adaptation.

- Both precarve and postcarve burnishing of the margins may improve adaptation.

Self-sealing

Over a period of time, a well placed amalgam restoration will begin to exhibit a reduction in leakage which can be attributed to the formation of corrosion products at the amalgam/tooth interface, a process which is termed 'self-sealing'.

Low copper amalgams seal within 2-3 months but high copper amalgams corrode less and therefore take 10-12 months to provide a comparable seal. This is the only clinical disadvantage of high copper amalgams compared with low copper amalgams.



Fig. 13.10. A quadrant of amalgams showing cuspal protection designs that will be followed for more than 15 years. Note the satisfactory conservative occlusal anatomy.



Fig. 13.11. The same quadrant as shown in **Figure 13.10** eight years after placement. Note wear facet on the buccal cusp of the second bicuspid. Possibly the occlusal was carved too deeply allowing the opposing bicuspid to overerupt.

Clinical techniques to seal the dentine tubules

A number of techniques have been used over the years to minimise or prevent the problems associated with lack of seal at the restoration margin and of the dentine tubules. Current techniques in common use are

- a thin glass-ionomer lining over all the dentine to seal the tubules. Condition the dentine as usual before placement,
- a low viscosity resin/dentine adhesive to seal the dentine tubules. Etch the dentine as usual before placement,
- a higher viscosity chemically cured or dual cured luting resin, in a bonded amalgam restoration.

Note: Techniques using a copal resin varnish or application of an oxalate solution have been largely superseded. These techniques failed to provide an adequate long-term seal of the dentine tubules.

SUMMARY



Seal at the margin

- crevice corrosion provides 'self-sealing' after 3-12 months
- glass-ionomer lining seals dentine and releases fluoride
- resin bond seals the dentine
- copal resin varnish and oxalate solutions are now largely superseded



Fig. 13.12. The same quadrant of amalgams shown 15 years after placement. Note the corrosion and marginal chipping. These do not affect longevity but aesthetics is poor.

Bonded amalgam restorations

In recent years there has been a trend to the placement of bonded amalgam restorations. In this procedure the walls of the cavity are prepared as for resin-to-dentine bonding then coated with a thin layer of a relatively high viscosity chemically cured or dual cured luting resin. Amalgam is mixed and packed into the cavity so that the amalgam and viscous resin are setting at the same time and therefore develop a mechanical interlock.

Advantages suggested for this procedure include:

- Conservation of tooth structure because a retentive cavity form is not required. However, research suggests that regular retentive form should still be provided so that the bonding will simply optimise the longevity of the seal.
- Fracture resistance of the tooth is enhanced. Research suggests that this is a relatively short term benefit lasting perhaps one year only due to hydrolytic degradation of the bond.
- Seal of the dentine tubules will eliminate postrestoration sensitivity. This benefit has been difficult to confirm in controlled clinical studies carried out by clinicians who are competent in optimising amalgam adaptation by traditional means.

The technique has the following difficulties:

- uniform placement of the viscous resin onto cavity walls is difficult
- in a large restoration, setting of the resin may prevent optimal carving of the amalgam
- the viscous resin may pool at the gingival margin of the cavity
- the bond of the resin to tooth is likely to degrade over time

At present, it is difficult to justify this technique because simpler, more reliable methods are available for sealing dentine tubules and minimising the effects of leakage at the margins.

Margin fracture of amalgam

Margin fracture has also been referred to as margin breakdown, ditching and crevice formation. The problem has been widely researched and discussed and several restorations exhibiting different amounts of margin fracture are shown in **Figures 13.13** and **13.14**. Regardless of the type of amalgam, margin fracture increases with time. The rate of increase is greater for low copper amalgams than for high copper amalgams.

Margin fracture is the result of an ongoing process of occlusal loading, deformation (creep), corrosion fatigue, fracture, formation of a crevice corrosion as well as further occlusal loading. Margin fracture correlates poorly with further caries, unless the crevice exceeds 300 μm in width, so that care must be exercised in making the deci-



Fig. 13.13. A quadrant of old amalgam restorations showing chipped and ditched margins. The cause is unknown but cavity design is partly to blame.



Fig. 13.14. Bulk fracture of an amalgam restoration. The main cause is the poor cavity design leaving a broad wedge of amalgam with no support.

sion to replace a restoration if the margin simply looks less than ideal. Few amalgams appear in an as new condition after three to five years and control of the disease of caries is the proper method of extending restoration longevity.

Clinical techniques to prevent margin fracture

The single most effective and proven method of reducing the rate of margin fracture is the optimal use of a high-copper amalgam although there are other factors that can influence the development of margin fracture.

- Excess amalgam, left lying over the occlusal or proximal surface through failure to carve correctly, is always subject to fracture leading to

margin ditching or crevice formation.

- The margin angle of the restoration should, where possible, be close to 70° and the cavity should be designed to allow for this. This may be difficult to achieve on occlusal margins.
- Where possible, design the cavity to avoid undue occlusal stress on the margin because of the potential for plastic deformation (creep) and corrosion fatigue under clinical loading.

Bulk fracture of amalgam

Bulk fracture of amalgam restorations is a common cause of failure during the first five years after restoration placement. Although physical strength as well as resistance to corrosion and plastic deformation are of importance, the operator related factors of cavity preparation and manipulation of the amalgam are the major causes leading to early failure (**Chapter 20**). Research has found that fractures at the isthmus of Site 2, Size 2, 3 and 4 restorations can generally be attributed to inadequate depth in the prepared cavity at this site. Poor manipulation during placement is another cause. High copper amalgams exhibit less evidence of early bulk fractures than low copper amalgams (**Figures 13.15-17**).



Fig. 13.15. The proximal box has fractured from this amalgam due likely to inadequate depth in the isthmus region.



Fig. 13.16. The same restoration as shown in **Figure 13.15** with cause of the failure now clear. The amalgam lacked bulk in the isthmus because of inadequate cavity depth.

Restoration of deep cavities with amalgam

A difficult clinical challenge is to restore a proximal cavity in a posterior tooth which extends



Fig. 13.17. Bulk failure of an extensive amalgam restoration which has come about because of the non-retentive cavity design.

beyond the cemento-enamel junction without incorporating an overhanging excess of amalgam somewhere in relation to the gingival margin. Placement of an adequate matrix is difficult and supporting that matrix sufficiently to withstand proper condensation loads is a further challenge. Attempts to remove the overhang subsequent to matrix removal are rarely completely successful.

A two-stage amalgam placement is one option. An amalgam restoration is initially placed, concentrating on optimal gingival contour. The restoration is then refurbished at the next appointment by the preparation of a cavity within the amalgam. This next restoration is designed to optimise proximal contact and contour. A lamination technique similar to the one proposed for composite resin is another option and is discussed in **Chapter 11**.

Repair of amalgam restorations

Occasionally there is a need to repair an existing restoration by adding fresh amalgam to existing amalgam that has been retained and now forms a portion of the walls of the newly prepared cavity. Alternatively, addition or repair with more amalgam may be required at the initial appointment.

A bond of up to 50% of the unrepaired strength can be obtained when a repair is carried out subsequent to the first appointment. However, additions carried out within the initial appointment can achieve a bond strength of up to 75% of the unrepaired strength. The preparation for the new amalgam should meet all the usual requirements for depth and mechanical retention. In all repairs, a retentive cavity form should be provided.

Sometimes it may be adequate to simply repair a broken margin with amalgam or glass-ionomer rather than replace the entire restoration, keeping in mind that replacement will almost invariably involve further loss of tooth structure and pulpal irritation.

If repairing the defective margin with amalgam, it is a wise precaution to place a glass-ionomer lining on the dentine at that site. Particularly if the margin is not under direct occlusal loading it may be sufficient to open conservatively, make sure there is no hidden caries, and then seal the margin with a glass-ionomer. Choose the strongest

available cement and condition the surface with 10% polyacrylic acid for 10 seconds, prior to placement.

Integrity of the repair

The surface to be repaired should be freshly cut and free of saliva and debris in order to optimise the chemical bond that may be achieved between available mercury from the new mix and the sectioned unreacted alloy particles of the previous amalgam. Use of a mercury rich mix of the new amalgam does not appreciably improve the strength of the bond and it represents unacceptable mercury hygiene. The use of a resin bonding layer has been shown to interfere with the repair process and prevents formation of a chemical bond of the new amalgam to the old.

Replacement of amalgam restorations

Regardless of the material, when a restoration is replaced some sound dentine and enamel are likely to be removed. Studies have demonstrated that amalgam and glass-ionomer may be removed from a cavity with substantially less loss of sound tooth structure compared with the removal of compomer and composite resin.

Galvanic effect

Patients may complain of a metallic taste following placement of an amalgam restoration. This is caused by a rapid, short lived corrosion that takes place between the new restoration and other older restorations, of either amalgam or gold, in the vicinity. The galvanic effect is mediated by the saliva and produces an electric current of sufficient intensity to elicit a metallic taste or even a mild pain. On rare occasions the pain may be acute and require immediate removal of the new amalgam. Following the formation of a passivating layer of corrosion products, the current is much reduced and the taste is usually no longer of concern. Sealing the new restoration with either a varnish or resin sealant may restore patient comfort and confidence. Amalgams of the high copper blend, Ag-Cu type may exhibit greater initial corrosion than other high copper amalgams because of the presence of γ_2 phase for from 1-3 hours.

Clinical performance of amalgam restorations

A large number of longitudinal clinical studies and cross-sectional surveys have been conducted to examine all aspects of the clinical performance of high copper and low copper amalgams.

Surveys have reported 50% survival times for Site 1 restorations of more than 19 years, for Site 2 restorations of 24 years, and for complex Site 2, Size 3 and 4 restorations, replacing at least one cusp, of 11.5 years. Recent studies of high copper amalgams report a failure rate at five years of only 2-3% and ten years survival rates of 75-91%. These results must be compared with survival rates of low copper amalgams of 39-80%.

Early failures of amalgam restorations are largely iatrogenic and can be reduced by more careful attention to cavity preparation and proper placement and carving of the amalgam.

The replacement of amalgam restorations can also be delayed by

- elimination of the disease of caries by control of sugar intake, by other preventive measures and routine fluoride applications,
- using a high copper amalgam. Use of a blended particle alloy will improve the adaptation/seal and reduce the leakage at margins,
- use of a glass-ionomer lining, or alternative technique, to seal dentine tubules beneath the restoration,
- careful diagnosis to minimise unnecessary intervention. Particular care should be taken in diagnosing margin fracture because this only correlates with the presence of caries when the defect is relatively large. Replacement of the entire restoration will invariably lead to further loss of tooth structure, weakening of cusps and additional pulpal irritation,
- careful cavity design to avoid placing occlusal load directly on to restoration margins.

Biocompatibility – Mercury and Dental Amalgam

The biocompatibility of amalgam has been the subject of extensive investigation, particularly in relation to the presence of mercury.

Unfortunately many patients have been advised, or have requested, to have existing amalgam restorations replaced in the mistaken belief that this will improve their general health, or result in the cure of chronic diseases and afflictions. Health authorities have consistently rejected these reasons for the removal, or nonplacement, of amalgam restorations.

The US National Institutes of Health in 1991, the Swedish Medical Research Council in 1992, the US Public Health Service in 1993, and the Swedish Board of Health and Welfare in 1994 all reported that there was no evidence of ill health resulting from the use of dental amalgam, with the exception of relatively rare and well localised allergic type reactions on adjacent mucosa.

Potentially available forms of mercury

From the toxicological viewpoint, there are potentially four available forms of mercury: liquid, vapour, inorganic compound, and organic compound.

Liquid mercury (elemental)

Elemental mercury has little or no toxic effect when swallowed. Droplets of mercury which may be swallowed are poorly absorbed and most liquid mercury passes harmlessly through the gastrointestinal tract.

Mercury vapour (elemental)

Significant toxicity can occur when mercury vapour is inhaled and this accounts for most of the occupational and accidental exposures resulting in mercury intoxication. Mercury vapour is rapidly absorbed into the blood via the lungs; it can cross the blood/brain barrier where it can accumulate over long periods of time and from which it exits only slowly.

Mercury is a nonspecific enzyme inhibitor and interferes with cellular function.

Chronic exposure to mercury vapour can result in mild to moderate central nervous system (CNS) effects that include, with increasing concentrations, insomnia, irritability, memory loss, headaches, depression and muscle tremors. Conditions that can mimic this mercury toxicity include alcoholism, lead poisoning, Parkinson's and senile dementia. Diagnosis of mercury intoxication requires testing the mercury level of urine (μg mercury/litre of urine)

- 10 μg is normal
- 100 μg is a significant exposure
- 300 μg is the level at which symptoms/signs appear

Although dental staff may be at risk if very poor mercury hygiene is practiced, the levels of mercury vapour to which patients are exposed is well below the level known to pose risks to health (Refer to **Concerns for those at special risk**).

Inorganic mercury compounds

Although dental amalgam contains several inorganic mercury compounds (e.g. Ag-Hg), of low or very low toxicity, they are apparently harmless when swallowed and are effectively excreted. In contrast, the lower molecular weight, mercuric chloride is caustic to the lining of the gastrointestinal tract and is listed as a violent poison. It is also irritating to the skin but is sometimes used in topical antibacterial agents.

Organic mercury compounds

Some organic compounds of mercury, especially methyl and ethyl compounds, are highly toxic at low concentrations but none is known to form in the oral environment through the use of dental amalgam. (Refer to **Environmental concerns**).

Concerns for the patients

The major pathway for the global transportation of mercury is via the atmosphere. For those people with no occupational exposure to mercury, with no amalgam restorations and with a low fish diet, the daily intake of mercury from air, food and water is 10-20 μg . The average daily dose of mercury from

dental amalgam, for patients with more than 12 restored occlusal surfaces, has been estimated at up to 3 μg . This represents approximately 15-20% of the normal daily intake of up to 20 μg of mercury from all sources for the average person.

The intake can be increased by actions that remove the passivating surface oxide film although this reforms rapidly. Such actions include habitual grinding of teeth (bruxing), chewing gum, eating coarse foods or toothbrushing.

Mercury release during dental procedures

Mercury release (μg) has been quantified for a number of procedures:

1-2 μg	Trituration of amalgam
6-8 μg	Placement of an amalgam restoration
44 μg	Dry polishing an amalgam to a high gloss
2-4 μg	Wet polishing to an acceptable finish
15-20 μg	Removal of an amalgam under water spray and high velocity suction
2-4 μg^*	Removal of an amalgam under water spray with high velocity suction extended for 30 seconds

* The extended use of the suction **after** removal of all the amalgam from a cavity is an important component of routine clinical protocol.

Some of the released mercury enters the saliva and is either evaporated from the saliva and respired into the lungs or released into the environment. Alternatively, it may be ionised and bound in complexes and carried to the gastrointestinal tract.

Significant clinical studies

A study of over 1,000 Swedish women, aged 38-60, carried out over a 20-year period failed to show any adverse effects on general health in those with amalgam restorations compared with those with no amalgam restorations. Removal of all amalgam restorations did not improve general health (Ahlquist *et al*, 1988 and 1995).

A study, on more than 100 Catholic nuns, aged 75-102, compared those with and without amalgam restorations and failed to identify any difference in mental function (Saxe *et al*, 1995).

SUMMARY

Minimising mercury release during dental procedures

- use high velocity suction for 30 seconds after all amalgam has been removed from previous restoration
- carry out **ALL** finishing procedures with a water spray
- do **NOT** attempt to achieve a 'high gloss'

Statement

Because the release of very small quantities of mercury from amalgam has been demonstrated, there have been anecdotal reports of cases of mercury toxicity resulting from dental amalgams. The evidence has been subjected to scrutiny from multidisciplinary panels of experts, who report 'although it is not possible to completely rule out adverse effects in a minority of susceptible patients, it is concluded that there is insufficient evidence to justify the claims that mercury from dental amalgam restorations has an adverse effect on the health of the vast majority of patients' (Eley and Cox, 1988).

SUMMARY

Significant clinical studies on effects of mercury

- no adverse effects of amalgam on general health (Ahlquist *et al*)
- no difference in mental function (Saxe *et al*)
- removal of all amalgams did not improve general health (Ahlquist *et al*)

Concerns for those at special risk

Based on potential toxicity hazard threshold levels, it has become customary in recent times to restrict the use of amalgam in those groups in society perceived to be most at risk. These groups are children, the pregnant and those with compromised immune systems.

The rationale for these restrictions is questionable as it appears to be based **not** on proven risks but, rather, on the threat of possible legal action at some time in the future.

Two types of threshold levels are described in industry – the **lowest** observed adverse effect level (LOAEL) and the **no** observed adverse effect level

(NOAEL). Threshold levels for the general public are based on industry levels and a generous allowance for safety and continuous exposure. The levels refer to equivalents to air mercury exposure ($\mu\text{g}/\text{m}^3$) where 100 equals clinical mercurism (LOAEL), 50 equals nephrotoxicity (LOAEL), 25 is the W.H.O. industrial level (NOAEL) and 5 is the general public (NOAEL) while 1 equates to those potentially at risk (NOAEL). To reach a level of 1 $\mu\text{g}/\text{m}^3$ from dental amalgam would require

- absorption of 20 $\mu\text{g}/\text{day}$ of mercury vapour via lungs
- absorption of 400 $\mu\text{g}/\text{day}$ of mercury in saliva via gastro-intestinal tract

To understand this scale, recognise that patients with, on average, 27 amalgam restorations would normally have a daily mercury dose from those restorations of 1.7 μg by inhalation and 1.3 μg by gastrointestinal absorption.

Numerous large studies have sought for a possible effect of mercury associated with pregnancy. Most of these studies have included females occupationally associated with dentistry and the use of dental amalgam (dentists, chairside assistants, laboratory technicians). Although a higher concentration of mercury was identified in the placenta of some dental personnel, no study has found a difference between dental personnel and females who are not occupationally exposed in regard to spontaneous abortions, birth weight, infant survival, congenital abnormalities.

Amalgam illness/sickness

Over recent years, dentists have become aware of patients claiming to have toxic signs and symptoms which they attribute to mercury from their dental amalgam restorations.

Typically, these patients report a wide range and diverse group of symptoms and signs. Many of these are typical of chronic fatigue syndrome. Others range from miscarriage to gastritis to aggressiveness to sensitivity to infection. Many patients report a range of signs and symptoms considered by the 'Association of patients damaged by dental treatment' to be typical of amalgam illness.

These people have often been to many health care practitioners before they attend a dentist

because it has been suggested to them that removal of all amalgam restorations would lead to a substantial improvement in their health status. Many of these patients have been described as being manipulative, and limited scientific studies have failed to identify a resting urine mercury level greater than the general population. A number of psychologists and dentists have been involved in attempting to provide answers to assist both patients and dentists to overcome amalgam illness. Many of them have a history of significant psychic trauma, often associated with triggering the symptoms. The history may include death of a parent, spouse or loved-one, miscarriage, divorce, marriage, childbirth, adoption. For the patient and the dentist, the most important aspect is the need to consider that these reported signs and symptoms may, in fact, be an indication of a serious condition, which is being overlooked, while the patient focuses on a series of relatively easy-fix causes, such as the mercury release from amalgam restorations.

SUMMARY



Patients complaining of 'amalgam illness'

- often present with many and diverse symptoms
- frequently exhibit symptoms of chronic fatigue
- have previously visited many health care practitioners
- may have undiagnosed serious health problems
- patient is 'focused' on 'easy-fix' causes e.g. presence of amalgams
- frequently have history of significant psychic trauma e.g. divorce, death of loved one etc.

Allergy to amalgam restorations

A direct allergic response to the mercury associated with dental amalgam is rare but may be manifested in one of two ways. An immediate sensitivity may follow placement of an amalgam restoration with external skin lesions being more common than intraoral lesions. An urticarial rash may appear on the face and limbs and this may be followed by dermatitis. Once the amalgam has set and the level of free mercury is greatly reduced, the skin lesions resolve.

Alternatively, there may be a long term response

in the form of oral lichen planus or lichenoid reactions with erosive areas on the tongue or buccal mucosa adjacent to an amalgam restoration. Frequently these restorations are poorly contoured and corroded (**Figures 13.18 and 13.19**).

BE AWARE



Any one of three factors may cause mucosal lesions

- allergy to mercury
- cytotoxicity related to the corrosion products
- trauma from rough restoration

Patients with lesions related spatially to amalgam restorations should be patch tested for sensitivity to the constituents of dental amalgam by a dermatologist experienced in this field. If allergy is proved, the restorations should be replaced using another material, although, on occasions, the patient may demonstrate an allergy to an alternative material such as composite resin. If there is no apparent allergy, new amalgams can be placed to eliminate corrosion products and to improve the contour.

Amalgam tattoo

The so called amalgam tattoo, in which components of amalgam are trapped in the oral tissues, appears clinically as a macular or slightly elevated blue, black or grey pigmentation, generally in close proximity to an existing amalgam. Possible causes are as follows:

- scraps of amalgam may fall into an open surgical or extraction wound
- excess amalgam may be left in the tissues while sealing the apex of a root canal with a retrograde amalgam
- pieces of amalgam may be forced into the mucosa

Usually there is no local tissue response, although chronic inflammation and dystrophic calcification at the site have been reported.

Concerns for dental staff

In the 1970s and 1980s the findings of several large studies on urine mercury levels in dentists resulted in the establishment of firm Mercury Hygiene protocols – refer to **Recommendations of**

the World Dental Federation. As a result, in less than 10 years, the average urine mercury level in dentists in the USA declined from 14.2 $\mu\text{g/l}$ to less than 5 $\mu\text{g/l}$ compared with the average for the general population of 1-3 $\mu\text{g/l}$. The first neurobehavioural effects of a raised mercury level are said to appear at a urine level of approximately 100 $\mu\text{g/l}$.

Environmental concerns

"If environmental contamination by mercury containing waste from dental practices is not cut down to very low levels, then it is likely to be the main reason for government action against the use of amalgam in the future." (Eley, 1997)

Particles of waste amalgam are produced when restorations are removed. Waste that is not removed by the primary and secondary waste traps within the dental surgery enter the waste water and sewer system. It has been estimated that, each year, dental facilities in the USA release 40 tons of mercury in this way.

The concerns

Mercury containing particles enter the food chain from the country's waterways. Micro-organisms methylate the mercury and these forms of organic mercury are concentrated as they move up the

food chain, particularly affecting people for whom fish and/or shellfish is a major part of the diet. There have been several serious environmental disasters caused by prolonged industrial discharge of mercury.

Prevention

It has been calculated that, in the dental environment, approximately 40% of the amalgam waste that is removed from teeth when replacing restorations is collected in the regular chairside and pump traps. That is, approximately 60% of the waste escapes the primary and secondary solid separators and is released into waste water. Use of efficient centrifugal separators or sedimentation separating techniques can achieve a 99% reduction in mercury concentration in waste water and their use should be mandatory.

The challenge

In future years, the challenge is not only to ensure the presence of effective amalgam particle traps and the installation of efficient centrifugal separators in dental practices but also to develop commercially viable techniques for collecting the constituents of the amalgam restorations that are less than 10 μm in size.



Figs. 13.18 & 19. Examples of long standing erosive lesions on the buccal mucosa and the side of the tongue that lie adjacent to old amalgam restorations. Removal of the restorations resulted in rapid improvement.

RECOMMENDATIONS FROM THE WORLD DENTAL FEDERATION



Dental mercury hygiene

1. All personnel involved in the handling of mercury should be alerted, especially during training, to the potential hazard of mercury vapour and the need to observe good mercury hygiene practices.
2. The workplace should be well ventilated with fresh air exchange and outside exhaust. Air filters such as those in air-conditioning systems may act as mercury reservoirs and should be replaced periodically.
3. The surgery atmosphere should be checked periodically for mercury vapour.
4. Do not lay carpet in dental surgeries. Continuous seamless sheet flooring carried up the walls for at least 10 cm is recommended.
5. Mercury should be stored in unbreakable, tightly-sealed containers away from any source of heat.
6. Mercury and amalgamation equipment should be used only in areas that have an impervious surface with a lip along the leading edge so that spilled mercury or excess amalgam is confined and recovery facilitated.
7. Use single-use capsules rather than reusable ones or any other method of dispensing the alloy and mercury.
8. Reusable capsules should be kept closed between uses. Single-use capsules should be reassembled after use and immersed in used radiographic fixer solution. Alternatively, store them in a screw-top container pending proper daily disposal.
9. Avoid the need to remove excess mercury before or during packing by selecting an appropriate alloy:mercury ratio.
10. Use only capsules that remain sealed during amalgamation.
11. Use an amalgamator with a completely enclosed activator arm.
12. Mercury dispensers should be handled with care and checked periodically for leakage.
13. The orifice of the mercury dispenser should be examined after use for residual mercury.
14. Mercury and unset amalgam should not be touched with the bare hands.
15. All amalgam scrap and free mercury should be salvaged and stored in a tightly-closed container under used radiographic fixer solution.
16. Spilled mercury should be cleaned up immediately and placed in the scrap jar.
17. Do not heat mercury or amalgam or any equipment used with amalgam. Instruments contaminated with amalgam should be cleaned before heat sterilisation.
18. Do not use ultrasonic amalgam condensers.
19. Remove old amalgams and polish new ones under copious air/water spray with high-volume evacuation. The exhaust for the system should be outside the surgery.
20. Wear a mask which is fine enough to prevent the inhalation of amalgam dust.
21. Disposable materials contaminated with mercury or amalgam should be placed in a polyethylene bag, sealed and disposed of every day.
22. Waste systems that amalgam scrap may enter (for example, cuspidors, sinks and suction systems) should have plastic traps from which the scrap can be recovered and stored as described in recommendation No. 15.
23. Skin accidentally contaminated by mercury should be washed thoroughly with soap and water.
24. Do not eat, drink or smoke in the surgery.
25. If a mercury hygiene problem is suspected, personnel should undergo urinalysis to detect mercury levels.

Further Reading

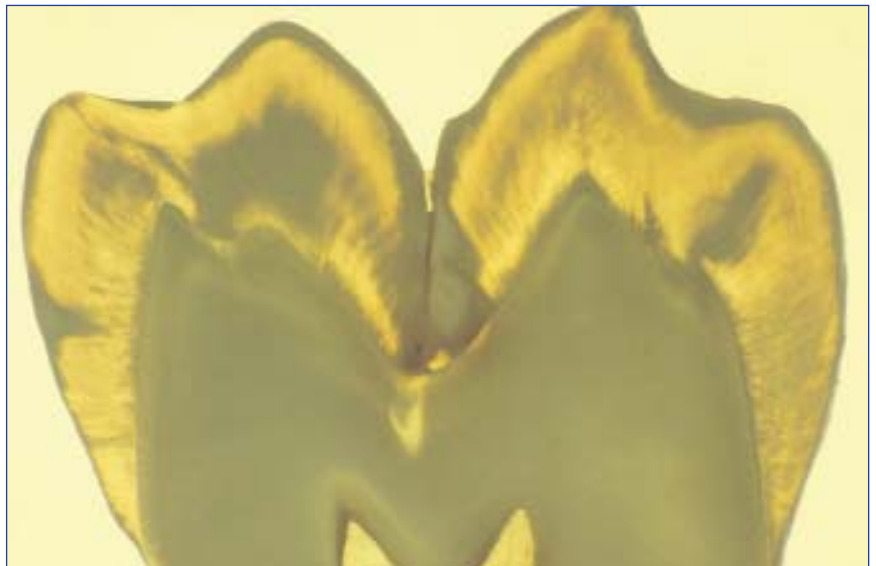
- Adegbembo AO, Watson PA, Lugowski SJ. The weight of wastes generated by removal of dental amalgam restorations and the concentration of mercury in dental wastewater. *J Can Dent Assoc* 2002; 68:553-558.
- Alhquist M, Bengtsson C, Furunes B, Hollender L, Lapidus L. Number of tooth fillings in relation to subjectively experienced symptoms in a study of Swedish women. *Comm Dent Oral Epidem* 1988; 16:227-231.
- Alhquist M, Bengtsson C, Furunes B, Hollender L, Lapidus L. Number of amalgam fillings in relation to cardiovascular disease, diabetes, cancer and early death in Swedish women. *Comm Dent Oral Epidem* 1993; 21:40-44.
- Bergman M. Side effects of amalgam and its alternatives: local, systemic and environmental. *Int Dent J* 1990; 40:4-10.
- Bonella E, White SM. Fatigue of resin bonded amalgam restorations. *Oper Dent* 1996; 21:122-126.
- Bryant RW. Marginal fracture of amalgam restorations. A review. *Aust Dent J* 1981; 26:162-166 and 222-224.
- Council on Dental Materials, Instruments and Equipment. Council on Dental Therapeutics. Safety of dental amalgam - an update. *J Am Dent Assoc* 1989; 119:204-205.
- Department of Health and Human Services. *Dental amalgam: a scientific review and recommended public health service strategy for research, education and regulation*. Washington, DC: Public Health Service, 1993.
- Eley BM. Dental amalgam: a review of safety (occasional paper, issue 3). London: British Dental Association, 1993.
- Eley BM. The future of dental amalgam: a review of the literature. Part 2: Mercury exposure in dental practice. *Br Dent J* 1997; 182:293-297.
- Hamilton JC, Moffa JP, Ellison JA, Jenkins WA. Marginal fracture not a predictor of longevity for two dental amalgam alloys: a 10 year study. *J Prosthet Dent* 1983; 50:200.
- Hawthorne W, Smales R, Webster D. Longterm survival of restorative materials in private practice. *J Dent Res* 1994; 73:747, Abst. 85.
- Lindberg NE, Lindberg E, Larsson G. Psychologic factors in the etiology of amalgam illness. *Acta Odontol Scand* 1994; 52: 219-228.
- Mahler DB, Marantz R. The effect of the operator on the clinical performance of amalgam. *J Am Dent Assoc* 1979; 99:38.
- Mahler DB, Nelson LW. Factors affecting the marginal leakage of amalgam. *J Am Dent Assoc* 1984; 108:51-54.
- Markley MR. Silver amalgam. *Oper Dent* 1984; 9:10-25.
- Mjor IA, Smith DC. Detailed evaluation of 6 Class II amalgam restorations. *Oper Dent* 1985; 10:17-21.
- Mount GJ. The condensation of amalgam by a group of general practitioners. *Aust Dent J* 1972; 3:222-227.
- Osborne JW. Mercury, its impact on the environment and its biocompatibility. *Oper Dent Supplement* 2001; 26:87-103.
- Osborne JW, Albino JE. Psychological and medical effects of mercury intake from dental amalgam. *Am J Dent* 1999; 12:151-156.
- Phillips RW. *Skinner's Science of Dental Materials*, 9th edition. Philadelphia: WB Saunders, 1991, Chapt. 17-18.
- Recommendations on dental mercury hygiene. Revision of FDI technical report number 7. *Int. Dent. J* 1988; 38:191-192.
- Saxe SR, et.al. Alzheimer's disease, dental amalgam and mercury. *J Am Dent Assoc* 1999; 130:191-199.
- Szep S, Baum C, Alamouti C and others. Removal of amalgam, glass-ionomer cement, and compomer restorations: changes in cavity dimensions and duration of the procedure. *Oper Dent* 2002; 27:613-620.
- Wing G. The condensation of dental amalgam. *Dent Pract*, 1965; 16:52-59.

14 Classification and Cavity Preparation for Caries Lesions

G. J. Mount ♦ W. R. Hume

As discussed in other chapters of this book, when demineralisation becomes dominant and remineralisation fails, a carious lesion will develop on the enamel or the root surface of a tooth. Once the lesion has progressed into the dentine there is a need for some level of surgical intervention to remove the infected dentine, to eliminate surface cavitation and avoid further accumulation of plaque. In most situations this will involve removal of a certain amount of enamel to achieve access but it must be noted that both enamel and dentine are capable of being remineralised and therefore conserved. The principle of minimal intervention operative dentistry is based upon maximum preservation of natural tooth structure to maintain the strength and integrity of the tooth crown. This Chapter offers a new look at the identification, classification and treatment of lesions from initial demineralisation to the treatment of extensive coronal breakdown.

Up to the present time the profession has used a classification of cavities proposed by G. V. Black over one hundred years ago. The classification was designed before the widespread use of radiographs so lesions were not diagnosed until they were visible to the naked eye and were therefore, by modern standards, relatively large.



A further problem was that it was a classification of cavity designs for amalgam as this was the principal restorative material available. The result was that, regardless of the size of the lesion, a specific cavity design was required to deal with it. Today current knowledge offers many alternatives ranging from earlier diagnosis of caries activity, along with effective methods of control, to the application of adhesive and bioactive restorative materials. If our patients are to reap the full benefit of these advances it is necessary to review both the classification and the approach to the surgical treatment of lesions when they progress beyond remineralisation alone.

Introduction

Defects on the crown or root surface of a tooth can arise from one or more of the following four causes:

- developmental defects in the enamel surface
- bacterial caries
- chemically stimulated dissolution or erosion
- physical abrasion

Probably the most common problem arises from a combination of bacterial caries beginning in relation to a developmental defect. This is confirmed by repeated surveys showing that the most frequent lesion requiring treatment is occlusal caries, primarily in molars but also in bicusps. The next lesion in terms of frequency is bacterial caries developing in relation to the contact point between pairs of teeth - both posterior and anterior.

In recent years there has been an increasing problem in relation to chemical erosion of both enamel and dentine and this can generally be traced to increased intake of acid food and drink allied to vigorous tooth brushing shortly after intake. Physical abrasion is generally related to occlusal irregularities but can often also be related to chemical dissolution at the same time.

All of these problems can lead to sufficient loss of tooth structure to require repair or replacement but, at the same time, all can be prevented, stabilised or healed to some degree. It is important that there is a means of properly classifying and identifying all these lesions at the time of initial examination so that a proper logical treatment plan can be formulated to not only repair the damage but, more importantly, eliminate the cause.

With this approach in mind this chapter outlines a proposal to introduce a new classification for lesions of the crown of the tooth and then goes on to offer some suggestions for repairing the lesions. It is important to note that the classification does not specify a cavity design. These essential details must be left to the informed and sound clinical judgement of the operator whose main aim at all times should be preservation of as much natural tooth structure as possible.

A New Cavity Classification

The reasons for a new classification

The classification used at present by the profession goes under the name of its author, Dr G. V. Black.¹ The centenary for the introduction of this classification is well past and there have been many changes and much progress in the understanding of caries, as well as other forms of progressive loss of tooth structure. The inherent limitations of the present classification are far too rigid for simple modification and it is suggested that it is time to get serious about reviewing the concept.

Probably the most significant discovery that has had a major impact on the practise of operative dentistry is the understanding of the ion migration that occurs, both out of and back into tooth structure, as a result of the caries process. It is now recognised that this is reversible, so the early lesion can be healed and recognition of the initiation of the disease process is imperative. After all, a cavity (loss of tooth substance) is an advanced symptom of a bacterial disease (or chemical dissolution) that has been in progress for some time. It is also apparent that there is a gradation of mineral loss from the heart of the lesion outwards to the periphery of the lesion. This implies that, simply because some section of the tooth is partly demineralised, it does not necessarily have to be removed because remineralisation may still be possible.

The second significant discovery is the development of sound long term adhesion between restorative materials and tooth structure. This not only reduces the potential for microleakage between restoration and tooth but also offers the possibility of reinforcing the tooth crown, at least to the limit of the tensile strength of the material.

A third innovation is the development of a restorative material that is capable of supporting an ion exchange within the tooth crown. This not only leads to an ion exchange mechanism for adhesion but also assists the remineralisation of demineralised enamel and dentine. These three discoveries alone significantly undermine the original precepts behind the G. V. Black classifica-

tion and suggest that there should be change. One of the greatest advantages of introducing a new classification is the possibility of recognising all new lesions from the very earliest stage and treating them in the most conservative minimally invasive manner possible.

At the same time it is necessary to accept that all restorative dentistry up to the time of the introduction of change will have been carried out using Black's principles. In other words, it is essential to take both concepts into account at the same time because it is not possible to carry out a simple substitution of one for the other. Breakdown of old restorations needs to be recognised separately as 'replacement dentistry' and there is little or nothing that can be done for these apart from minimising the loss of further tooth structure.

The following apologia to G. V. Black is offered to assure the reader that the authors understand the historical significance of a great man and a leader of the profession.

The G. V. Black Concept

When Black defined the parameters for his classification, the cavity designs were controlled by a number of factors many of which no longer apply. Caries was rampant and the role of bacterial flora and the significance of fluoride were not understood. Radiographs were not in general use so, on average, a cavity was not diagnosed until it was large enough to be identified with a sharp probe or seen by the naked eye. By modern standards that meant it was well advanced. There were limitations in the available instruments for cavity preparation as well as the selection of restorative materials. The classification offered a series of cavity designs related to the site of the lesion but the list was then modified to suit the intended restorative material. Because all cavities, by today's standards, were large he did not take into account the increasing dimensions of a cavity nor the varying complexity of the method of restoration. Black suggested that it was necessary to

- remove additional tooth structure to gain access and visibility
- remove all trace of demineralised enamel

and dentine from the floor, walls and margins of the cavity

- make room for the insertion of the restorative material in sufficient bulk to provide strength
- provide mechanical interlocking retentive designs
- extend the cavity to self cleansing areas to avoid recurrent caries

In his designs Black showed commendable respect for remaining tooth structure as well as occlusal and proximal anatomy but it was necessary to sacrifice relatively extensive areas of enamel and dentine to achieve his goals. Other far more effective methods of dealing with a carious lesion are now available. With modern understanding of adhesion and remineralisation it is no longer necessary to remove all unsupported demineralised enamel around the cavity margin, the concept of self-cleansing areas has been discarded and removal of all affected dentine from the axial wall of the cavity is strictly contraindicated because of the potential for remineralisation and healing.

Many of the old limitations no longer apply and it is now appropriate to think again about the problems presented by a carious lesion. Without in any way denigrating the achievements due to Black's concepts and work, the following thoughts are offered and a new approach to the definition of cavity design is outlined. The proposed classification is designed for the identification of lesions from the very earliest stage of demineralisation and to define their increasing complexity as the lesion extends. It is expected to provide benefits for both the profession and their patients.^{2,5}

Classification of lesions of the exposed tooth surface

It is suggested that caries lesions occur in only three sites on the crown or root of a tooth, that is, in those areas subject to the accumulation of plaque. Therefore, the first parameters for the classification are these sites:

- **Site 1** – pits, fissures and enamel defects on occlusal surfaces of posterior teeth or other smooth surfaces

- **Site 2** – approximal enamel in relation to areas in contact with adjacent teeth
- **Site 3** – the cervical one third of the crown or, following gingival recession, the exposed root

However, as caries can be a progressive disease, it is desirable to be able to define the size and extent of the lesion at the time of identification and, therefore, the potential complexity of the restorative procedures required for treatment. It is possible then to define five separate sizes as the lesion progresses:

- **Size 0** – the earliest lesion that can be identified as the initial stages of demineralisation. This needs to be recorded but will be treated by eliminating the cause and should therefore not require further treatment,
- **Size 1** – minimal surface cavitation with involvement of dentine just beyond treatment by remineralisation alone. Some form of restoration is required to restore the smooth surface and prevent further plaque accumulation,
- **Size 2** – moderate involvement of dentine. Following cavity preparation remaining enamel is sound, well supported by dentine and not likely to fail under normal occlusal load. The remaining tooth is sufficiently strong to support the restoration,
- **Size 3** – the lesion is enlarged beyond moderate. Remaining tooth structure is weakened to the extent that cusps or incisal edges are split, or are likely to fail if left exposed to occlusal load. The cavity needs to be further enlarged so that the restoration can be designed to provide support to the remaining tooth structure,

- **Size 4** – extensive caries or bulk loss of tooth structure e.g. loss a complete cusp or incisal edge, has already occurred.

The Size 0 lesion will be new and may be difficult to identify. The immediate treatment will be to eliminate the disease and thereby bring about remineralisation. Size 1 lesions will necessarily also be a new lesions and minimal cavity designs, followed by restoration with adhesive materials, will be indicated. Sizes 2, 3 and 4 may mean a new lesion that has progressed to a considerable extent without the patient presenting for treatment or it may be replacement dentistry following breakdown of an old restoration. The same basic principles for developing a cavity design will apply in both cases and, for obvious reasons, the larger the cavity the greater the problems in restoration and the shorter the probable longevity of the plastic restorative materials. The selection of the most suitable material for the larger restorations will be dictated by such properties as resistance to fracture and flexure as well as abrasion resistance.

To assist in communication the relationship between Black's classification and the modern site and size concept is shown below.

Site 1: Size 0, 1, 2, 3 and 4 - Pit and fissure caries

- Cavity located on the occlusal surface of a posterior tooth or any simple enamel defect on an otherwise smooth surface of any tooth.
- **Black Class I** – the smaller Sizes 0 and 1 could not be carried out previously because suitable restorative materials were not available so the Black classification begins with Site 1, Size 2 (1.2).

TABLE 14.1: Classification of caries lesions

SIZE SITE	No cavity 0	Minimal 1	Moderate 2	Enlarged 3	Extensive 4
Pit/fissure 1	1.0	1.1	1.2	1.3	1.4
Contact area 2	2.0	2.1	2.2	2.3	2.4
Cervical 3	3.0	3.1	3.2	3.3	3.4

Table 14.1. A diagrammatic representation of the proposed classification so that the user can visualise the relationship of the Site and Size concept for the description of lesions of the crown of a tooth.

Site 2: Size 0, 1, 2, 3 and 4 – Approximal lesion commencing in relation to contact areas

- Cavity located on the approximal surface of any tooth (anterior or posterior) initiated in relation to the contact area between two teeth.
- **Black Class II** – lesions occurring between posterior teeth only. Because of difficulties of identification and materials limitations there was no equivalent of Size 0 or 1 so the Black classification begins with Site 2, Size 2 (2.2).
- **Black Class III** – lesions occurring between anterior teeth only.
- **Black Class IV** – an extension of a Class III lesion involving the incisal corner or incisal edge of an anterior tooth. An alternative cause would be traumatic fracture of the incisal corner – now classified Site 2, Size 4 (2.4).

Site 3: Size 0,1,2,3 and 4 – Cervical lesions

- Lesion located in the cervical region anywhere around the full circumference of a tooth including exposed root surface following recession.
- **Black Class V** – this classification does not recognise lesions on the gingival third of the approximal surface, particularly root surface



Fig. 14.1. The crown of a bicuspid tooth showing the three Sites where caries is normally initiated: 1. occlusal fissures, 2. proximal contact areas, 3. cervical regions around the full circumference of the tooth.

Table 14.2. Demonstrating the difference between the original G. V. Black classification and the new proposal. The main difference is that the earliest signs of demineralisation can be recorded with this system and the numerical identification fits well with computerisation of records.

TABLE 14.2: Comparison

Proposed classification	Equivalent Black classification
Site 1 – Pits and fissures and smooth surfaces	Class I – Pits & fissures
Size 0 – fissure seal	Not classified
Size 1 – minimal surgery	Not classified
Size 2 – equivalent to Black Class 1	Class I
Size 3 – requires protection of remaining tooth structure	Class I
Size 3 – lost cusp or similar	Class I
Site 2 – Contact area, all teeth	Class II – contact area, posterior teeth
Size 0 – surface demineralisation	Not classified
Size 1 – beyond remineralisation	Not classified
Size 2 – moderate involvement	Class II
Size 3 – requires protection of remaining tooth structure	Class II
Size 4 – bulk loss of tooth structure	Class II
	Class III – contact area, anterior teeth
	Not classified
	Not classified
	Class III
	Class III
	Class III
	Class IV – incisal edge lost, anterior tooth
	Not classified
	Not classified
	Class IV
	Class IV
	Class IV
Site 3 – cervical third	Class V – cervical third
Size 0 – surface demineralisation	Not classified
Size 1 – minimal intervention	Not classified
Size 2 – more extensive	Class V
Size 3 – approximal root surface	Class II
Size 4 – two or more surfaces	Class V

caries, as being different from Class II lesions. An erosion/abrasion lesion or a small carious cavity on the buccal or lingual surface would be a Site 3, Size 0 (3.0) if it was expected to be arrested. If restoration was required it would be Site 3, Size 1 (3.1). A larger carious lesion would be classified as Site 3, Size 2 (3.2). An interproximal lesion would generally be Site 3, Size 3 (3.3) because of difficulty of access. The Site 3, Size 4 (3.4) classification is reserved for a complex lesion involving more than one tooth surface.

Cavity design and preparation

It will be noted from the above that the Black's classification did not allow for the Size 0 or Size 1 lesion in either Site 1 or 2 because, in the absence of radiographs, they could not be identified. Also, in the absence of adhesive restorative materials the Size 1 could not be repaired in the proposed minimal manner.

It must be recognised that there is a clear division between restoring a new lesion and replacing a failed restoration. When dealing with new active caries the cavity design should be very conservative because it is possible to remineralise both enamel and dentine which is only partly demineralised and not denatured and cavitated. Margins need be extended only to smooth surfaces which are capable of remineralisation and the concept of removal of all demineralised tooth structure on the theory of extension for prevention no longer applies. Cavity outline form should be dictated only by actual cavitation of the surface so this means it is often possible to maintain tooth to tooth contact interproximally. In fact, with the Size 1 and 2 lesion the prime object of the restoration is simply to restore the smooth surface of the crown to prevent further plaque accumulation.

When dealing with an erosion/abrasion lesion it is essential to diagnose and eliminate the cause to ensure longevity for any restorative material chosen for repair.

On the other hand, in replacement dentistry, the cavity outline is already defined and will often be more extensive than ideal. For these restorations most of the principles laid down by Black will still

apply, if for no other reason than tooth structure cannot be replaced. In fact, for both Size 3 and Size 4 lesions very little has changed.

Whether the problem presenting is a new lesion or replacement of a failed restoration, the limitations of the physical properties of both the remaining tooth structure and the restorative material must be taken into consideration. A small restoration can be reliably supported by remaining tooth structure, particularly in the presence of adhesive restorative materials. In fact, it is claimed that a tooth crown can be restored to full physical strength by placing these materials. However, as the cavity enlarges the tooth becomes weaker until it reaches a point where the restoration must be designed in such a way that the restorative material itself will support remaining tooth structure and protect it from occlusal load. This requires modification to cavity designs and some consideration as to which material to utilise. These factors are taken into account within the classification.

With the foregoing in mind, treatment of each of the lesions mentioned in the classification will be discussed. However, it must be noted that there is no intention of specifying the actual cavity design or the method of restoration for any lesion. These decisions are left to the operator to decide according to prevailing conditions for each patient.

SITE 1 LESIONS

Lesions identified under this classification will generally commence in fissures on the occlusal surface of a posterior tooth. Pits on the lingual of upper anterior teeth are not uncommon and may also occur on the buccal surface of lower molars and the lingual extension of the distal occlusal groove of upper molars. Erosion and attrition lesions on the occlusal surfaces of posteriors and the incisal edges of anteriors should also be included.

- **Site 1 – Size 0 (1.0)**

No equivalent in the G. V. Black classification

A pit or fissure on any tooth or an erosion lesion on an incisal edge that is regarded as

suspicious and in need of observation and preventive measures.

- **Site 1 – Size 1 (1.1)**

No equivalent in the G. V. Black classification

Small defect in one section of a pit or fissure and will often be restored in combination with placement of a fissure seal on the remainder of the fissure system.

- **Site 1 – Size 2 (1.2)**

Moderate size lesion with all fissures involved or replacement of an existing Black Class I restoration.

- **Site 1 – Size 3 (1.3)**

A larger lesion requiring incorporation of protection of one or more cusps within the design.

- **Site 1 – Size 4 (1.4)**

Extensive lesion with one or more cusps already missing

Site 1 – Size 0, designated 1.0

No equivalent in the G. V. Black classification

The typical lesion is generally represented by an occlusal fissure on a posterior tooth. However, there are similar defects in the enamel that can be noted in areas like the cingulum pits at the lingual of upper laterals or the buccal pits on lower molars. The eroded tips of the cusps of posteriors or incisal edges of anteriors can also be recorded

in this category. Specifically these lesions do not need to be restored, simply identified. It is important to diagnose the cause of the lesion and the caries risk situation and prevent progress (**Chapter 6**). This is not always simple but will necessarily require investigations in to the caries status of the patient.

Anatomy of the fissure system

Before considering the prevention or restoration of a fissure lesion it is necessary to understand the development and anatomy of fissures.^{6,7} Diagnosis and treatment of the fissure systems of the posterior teeth has always been difficult and controversial. It seems that nearly all posteriors have complex fissures but for a variety of reasons only a percentage of them become carious (**Figures 14.2 and 14.3**). Fissures form during calcification of the crown of the tooth. Calcification commences at the tip of the cusps and, as the cusps grow, they will fuse to some degree at the completion of the occlusal surface of the crown. Fusion will not always be complete and in a high percentage of cases there will be defects within the area of fusion ranging from single deep pits to extended grooves with limited opening to the outer surface but relatively large defects in the depths of the fissure. It is virtually impossible to determine the

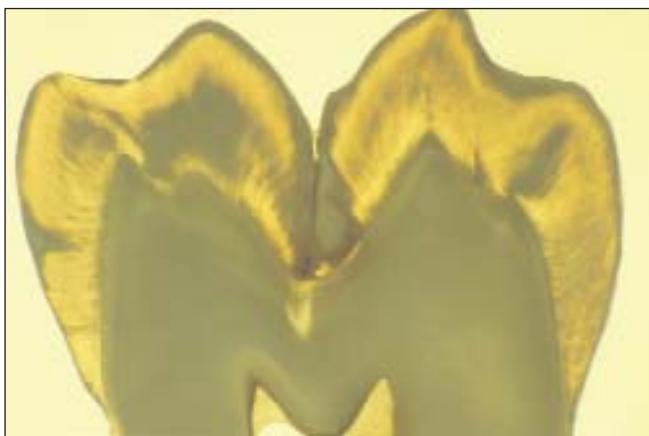


Fig. 14.2. A photomicrograph using transmitted light showing the earliest signs of a caries lesion at the base of an occlusal fissure. Note the signs of the development of the translucent dentine below the fissure resulting from the deposition of additional mineral in the lateral tubules as a result of stimulation of the pulp arising from the presence of the caries.

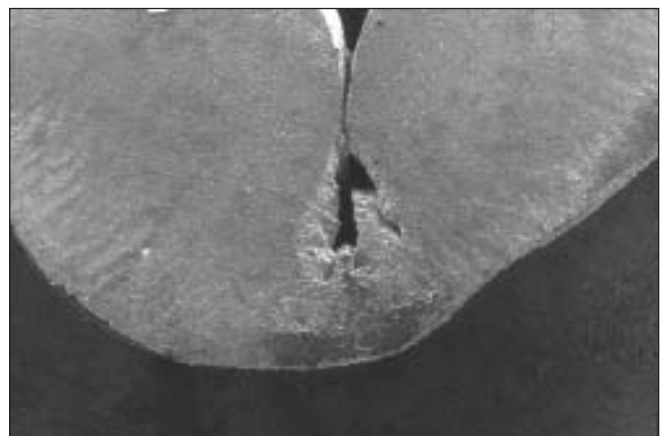


Fig. 14.3. A scanning electron micrograph of the same lesion shown in **Figure 14.2**. Note the level of development of the lesion in the enamel without overt signs in the dentine. However, the dentine is already involved as demonstrated in the previous figure.

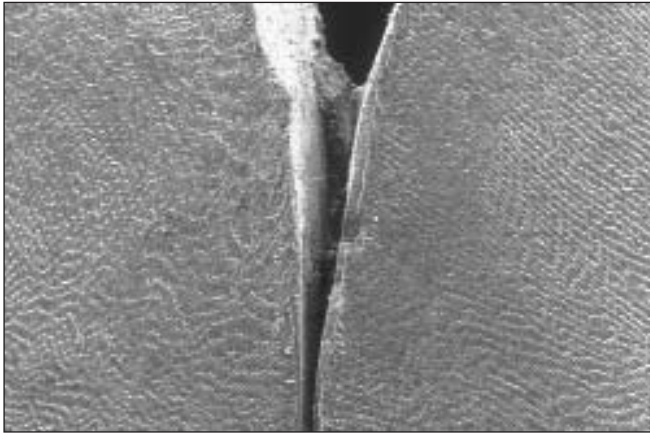


Fig. 14.4. A scanning electron micrograph showing the detail of an occlusal fissure. Note the debris at the base and the layer of amorphous enamel on the surface of the enamel.

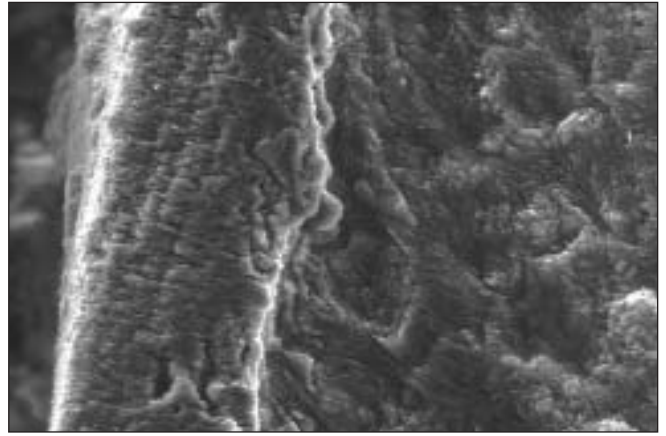


Fig. 14.5. A scanning electron micrograph at a higher magnification showing more clearly the layer of amorphous enamel on the surface of the fissure. This will complicate the development of the micro-mechanical adhesion with composite resin. Courtesy Dr H. C. Ngo.

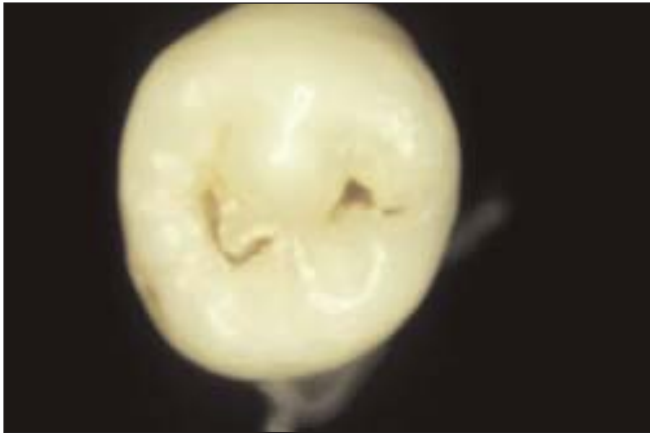


Fig. 14.6. An occlusal view of a bicuspid showing the occlusal fissures. Careful examination suggests the possibility of some carious involvement in the right fissure.

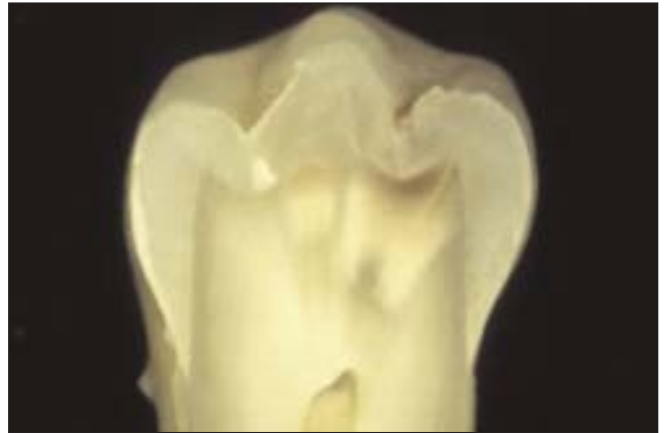


Fig. 14.7. The tooth shown in **Figure 14.6** has been sectioned to show the level of demineralisation beneath the right hand fissure. Note the translucent layer below the caries which represents the level of involvement of the pulp at this early stage.

complexity by visual examination or by radiographs so many situations go undetected. Modern laser fluorescence equipment has been developed to assist in diagnosis but should not be entirely relied upon for accuracy in detection of the presence of caries (**Chapter 9**).

The surface of the enamel within a fissure is covered with a layer of so-called amorphous enamel.⁸ This consists of a layer of reduced enamel epithelium along with rather jumbled enamel rods lying more or less parallel to the surface rather than at right angles (**Chapter 1**). It is likely that all enamel is formed with this covering layer but on the outer exposed surface the layer will

wear away shortly after eruption leaving the traditional enamel formation where only the ends of the rods are visible or available for adhesion through etching (**Figures 14.4 and 14.5**).

Within a relatively short period of years the complexities of the fissure will generally be filled with plaque and pellicle and because of the narrowness of the fissure it is impossible to remove it all in preparation for sealing.⁹ However, these defects provide an ideal milieu for the generation of active caries and the demineralisation can progress to a considerable extent before detection (**Figures 14.6 and 14.7**).

Sealing the fissures of the newly erupted tooth



Fig. 14.8. Careful examination of the occlusal fissure system on this lower molar suggests that there may be some caries involvement in the lingual fissure. A fissure seal is recommended.



Fig. 14.9. The tooth shown in **Figure 14.8** has been fissure sealed with a Type II.1 restorative aesthetic glass-ionomer.



Fig. 14.10. The same tooth as shown in **Figure 14.9** photographed approximately 12 years after placement of the fissure seal suggesting the longevity of such a seal is satisfactory.



Fig. 14.11. A similar fissure seal in an upper molar photographed approximately 14 years after placement.

is widely regarded as a logical preventive treatment and has been utilised for many years.^{10,11} There is a long history of success but it remains far from universal in its application.¹² It is suggested that, in the application of this classification, the Size 0 should be recorded for a situation where there is a complex fissure but none of the tests applied reveal decalcification. This is recorded as a warning that the fissure should remain under close observation and it should be sealed in the near future if there is any sign of change or the caries activity level for that patient is in the danger zone. Fissure sealing is regarded properly as a preventive treatment rather than a restora-

tion but is included for discussion here because it generally utilises normal restorative materials - composite resin or glass-ionomer¹³ (**Figures 14.8-11**).

Site 1 – Size 1, designated 1.1

No equivalent in the G. V. Black classification

Lesions identified under this classification will generally commence in fissures on the occlusal surface of a posterior tooth. Pits on the lingual of upper anterior teeth are not uncommon and may also occur on the buccal surface of lower molars and the lingual extension of the distal occlusal groove of upper molars. Erosion and attrition

lesions on the occlusal surfaces of posteriors and the incisal edges of anteriors should also be included once they are extensive enough to require restoration.

The complex anatomy of a fissure lends itself readily to the admission of bacterial plaque into the restricted depths along with periodic infusions of refined carbohydrate for nutrition. Demineralisation can then progress intermittently without being readily observed from the outside. The speed of penetration will be determined by the caries activity level in each particular patient and progress can be very fast with devastating results. This is particularly so in the presence of well fluoridated enamel which is likely to be very strong and resistant to fracture under occlusal load. In these circumstances it is possible for the dentine to become involved to the extent that the pulp is also involved before the tooth exhibits any form of symptoms.

Generally, progress will be slower and possibly intermittent. In many cases only one section of a fissure system will become involved at a time. Previously, when amalgam was the material of choice, it was necessary to eliminate all sign of the fissure system by trenching the occlusal surface because it is not possible to effectively finish an amalgam partially along a fissure. There will always be a defect at the interface between amalgam and the fissure in the enamel. In the presence of the adhesive materials it is possible to be highly selective and to instrument only that section of the fissure that is obviously cavitated and demineralised and then simply seal the remainder at the time of placement of the restoration (**Figures 14.12-17**).

This technique can be used under many circumstances with safety and will result in saving considerable areas of natural tooth structure. For example, when restoring a proximal Site 2 lesion the occlusal fissure will sometimes become involved in the cavity design. Previously removal of the entire fissure was mandatory, partly to ensure that caries would not develop later in the fissure and partly as one element in the retention of a non-adhesive restoration. At this time the fissure can be sealed at the time of placement of the proximal restoration using an adhesive material.

The materials used for sealing fissures are discussed separately in their own chapters but it is logical to briefly review them here particularly in relation to the anatomy of fissures.

Resin sealants

There have been many materials used to seal fissures and prevent the development of active caries ranging from silver nitrate to lasers. However, the most popular sealant over many years has been resin in various forms¹⁴ and now glass-ionomer is also proving to be of value.

Resin, either unfilled or lightly filled, was originally used over thirty years ago and has a long history of success. It is now available in both a light cured or an autocured form and many are tinted for easier identification. The intention is to flow the resin into the depths of the fissure to obtund it completely so preventing the future admission of more plaque and bacteria. Prior to placement the enamel should be etched to both reduce the surface energy of the enamel and to allow development of the usual micromechanical attachment of the resin. Unfortunately, the enamel on the walls of a fissure is covered with an amorphous layer, as described above, and may not accept a good etch pattern so attachment of the resin may be tenuous (**Figure 14.5**).

The presence of plaque and pellicle can be a serious interference to the admission of resin into the depths of the fissure. It has often been suggested that the occlusal surface should be well cleaned prior to placement of the sealant but it is apparent that most methods of cleaning will only add to the debris trapped in the opening to the fissure. Also it is apparently not possible to flow the resin into the fissure beyond the point where the fissure is less than 200 μm wide so complete success is unlikely.

Failure may occur through partial lifting of the sealant with exposure of one segment or another of the fissure. As resin has no antibacterial properties there is the possibility of caries initiation, particularly if the segment of resin is retained but not sealed. In spite of these possible limitations the effectiveness of such a preventive measure has been reported to be acceptably high.



Fig. 14.12. A laboratory technique exercise undertaken on the fissures of a lower molar to test the efficiency of the placement of a glass-ionomer as a sealant for a Site 1, Size 1 lesion.



Fig. 14.13. The tooth shown in **Figure 14.12** has been sectioned bucco-lingually to demonstrate the penetration of the glass-ionomer into the depths of the fissure. It is apparent that the restoration is very efficient.



Fig. 14.14. A Site 1, Size 1 lesion. This patient had been caries free up to date but at the age of 18 years there is evidence of caries in the distal fissure of the lower molar. Caries activity was monitored and eliminated and limited intervention was planned.



Fig. 14.15. The lesion shown in **Figure 14.14** has been investigated in a very conservative manner and the full extent of the lesion determined. It is now being conditioned ready for placement of a glass-ionomer sealant.



Fig. 14.16. The lesion shown in **Figure 14.14** has been restored with a fast setting autocure Type II.2 glass-ionomer using a gloved finger as the matrix to force the glass-ionomer in to place. The photograph was taken as the finger was removed leaving a slight excess of the cement.



Fig. 14.17. The tooth shown in **Figure 14.14** following removal of the excess cement. Note the remainder of the fissure system is now sealed as well as the lesion site.

Glass-ionomer sealants

Glass-ionomer is also a very effective protection material for open fissures although there has not been a great deal of long term research published to date.^{15,16} Its value lies in the fact that it is possible to develop adhesion to the enamel, both normal and amorphous, through the ion exchange mechanism and, in addition, it has a continuing ion exchange with the environment.¹⁷ The same limitations apply as described above for a resin seal in as much as it will not flow into a fissure beyond the point where the fissure is 200 μm wide and neither is it possible to completely clean it.

However, it is sufficient to condition the tooth surface with 10% polyacrylic acid for 10 seconds without scrubbing or cleaning. Wash thoroughly with water only and dry lightly. This will remove plaque and pellicle and reduce the surface energy of the enamel to allow the cement to adapt readily and develop a good adhesion. A fast setting, high strength, Type II.2 autocure restorative cement should be mixed with a high powder/liquid ratio, and flowed into the fissure. The tip of a gloved finger, very lightly lubricated, makes an excellent matrix to apply pressure to the cement to ensure maximum penetration into the fissure.

The use of a low powder content weak glass-ionomer material is contraindicated because these cements do not last for long. They wash out or wear out. However, it has been shown that, even though the cement is lost, the caries activity is reduced because of the fluoride uptake into the enamel or, possibly, there is a glass-ionomer residue left in the entrance to the fissure.

Mechanical preparation of fissures

As discussed above, it is virtually impossible to clean out a fissure once a tooth has been in the oral environment for more than a year or two. In many cases, as the patient ages, it will become desirable to mechanically clean the fissure even though this involves a limited sacrifice of some enamel. There is a variety of methods of achieving this with minimal reduction of tooth. These variations are discussed in **Chapter 9** and include a very fine tapered diamond stone, a small tungsten carbide bur, a laser or air abrasion. The diamond

stone offers the simplest and most effective method mainly because the operator retains some degree of both visual and tactile sense and is therefore less likely to over extend the preparation. As the preparation is primarily in enamel, local anaesthesia is not generally required. The tungsten carbide bur is less desirable because it is easy to lose sight of the head of the bur and therefore lose control of depth of penetration. However, the other methods are claimed to be painless and do not require local anaesthesia either and therefore have some patient appeal. Neither is there any tactile sense so over extension is possible. Whichever preparation method is applied preservation of natural tooth structure is of paramount importance along with long term adhesion of the restorative material.

Having gained access to a carious area it is only necessary to develop clean walls to the cavity to ensure proper adhesion. Leave the floor untouched even though it may be demineralised. Once it is sealed and isolated it will remineralise and heal.¹⁸

Site 1 – Size 2, designated 1.2

G. V. Black Classification – Class 1

Preparation

This may be a new cavity in which caries has progressed some distance before the patient presented for treatment. Generally only one part of the fissure system will be involved and remaining fissures can be sealed without further exploration¹⁹ (**Figures 14.18-21**).

However, it will often mean replacing an existing restoration, such as an old amalgam ‘prophylactic odontotomy’, where all of the fissure system has already been involved. A tungsten carbide bur should be used at ultra high speed to remove old restorations taking care not to extend the cavity any further than necessary. A tapered or parallel sided diamond cylinder at intermediate high speed under air/water spray is then preferred to explore the extent of the problem. Small round burs can be used to remove remaining caries from the walls but removal of all affected dentine from the floor is contraindicated. Occlusal enamel



Fig. 14.18. A Site 1, Size 2 caries lesion. This was a lesion similar to the one shown in **Figure 14.14** but on opening the lesion it was apparent that it had progressed further.



Fig. 14.19. The tooth shown in **Figure 14.18** following complete restoration with a Type II.1 glass-ionomer.



Fig. 14.20. The tooth shown in **Figure 14.18**. Following restoration with the glass-ionomer it was decided that the occlusal surface required greater strength than could be offered by the cement so it was cut back to allow lamination with composite resin.



Fig. 14.21. The tooth shown in **Figure 14.18** following lamination with composite resin in the main load bearing area. Note that the rest of the fissure system remains restored in glass-ionomer.

should be retained, even though it is unsupported, providing the margins are sound and there are no microcracks.²⁰ A base of glass-ionomer will provide considerable support and reinforcement to undermined enamel.

Restoration

Glass-ionomer is the best material for the initial restoration and it can be laminated as required with composite resin if the occlusal load is excessive. Use the strongest cement available, for preference an autocure cement, providing it is radiopaque and mixed at a high powder/liquid ratio to allow for the development of optimum

physical properties. A sublining is not required because of the high level of biocompatibility of the glass-ionomer. Condition the cavity and place the cement with a syringe starting at the floor of the cavity.

Composite resin should not be used alone because of its shrinkage on curing with the consequent risk of microleakage. If it is to be used it should be laminated over glass-ionomer. The combination of the two materials is sufficient to restore the physical properties of the tooth very close to the original. The modern high strength autocure glass-ionomer will set rapidly and within three minutes it can be cut back approximately

2-3 mm to allow room for the composite resin. Clean the enamel margins free of cement and polish and bevel as necessary. Etch both the enamel and the cement for 15 seconds. Wash thoroughly and dry but do not dehydrate. Apply an appropriate unfilled resin enamel bonding agent followed by incremental build up of the composite resin. After completely light curing, adjust the occlusion and polish as required.

Site 1– Size 3, designated 1.3

G. V. Black Classification – Class 1

When the cavity reaches this size there will be extensive undermining or breakdown of at least one cusp with the possibility of a split developing at the base.²¹ It may be a new lesion involving almost the entire dentine of the crown or it may be an old restoration which has become recurrent (Figures 14.22-25).

Preparation

Tungsten carbide burs should be used at ultra-high speed to remove any old remaining restorative material and a small diamond cylinder is best to open the enamel to determine the extent of the problem. Round burs can then be used to remove infected dentine from the walls around the entire periphery to ensure a sound ion exchange adhesion. Be careful not to remove all affected dentine from the floor of the cavity to avoid the problems arising from pulp exposure.

If it is a new cavity resulting from active caries it may be desirable to carry out an indirect pulp capping as described in **Chapter 16**. Open the cavity with a small diamond cylinder, only as far as required, to gain access to the infected dentine. Clean the walls around the entire periphery using a round bur of appropriate size to ensure a complete seal. Leave affected dentine on the floor because it may remineralise and it is better at this time not to stimulate the pulp any more than essential. Seal the cavity with glass-ionomer for a minimum of three weeks and then reassess the cavity design when preparing the final restoration. At that stage carefully check all remaining cusps to determine the need to protect them from occlusal load. If a cusp has a column of sound den-

tine providing adequate support for the enamel and there is more than one half of the medial facing cuspal incline still present it can remain standing without protection.

If a cusp is undermined and the medial cuspal incline is subject to occlusal load it requires protection or it will develop a split at the base. These aspects of cavity design are discussed in detail in **Chapter 10**. The cusp will need to be beveled outwards to reduce lateral load regardless of the restorative material. If amalgam is the material of choice it may be necessary to include retentive elements in the cavity design such as grooves and ditches placed in remaining sound dentine to ensure that the restoration is soundly locked in. If the adhesive materials are to be used the cusp still requires to be bevelled and the restorative material will need to be well constructed to take the load and relieve the cusp.

Restoration

Of the plastic materials available amalgam is the material of choice for such an extensive restoration. It is easier to build and to carve the occlusal anatomy to the extent required and the wear factor is similar to natural tooth structure. Also amalgam has a superior resistance to flexure and therefore is better able to provide positive protection to the weakened tooth structure. Eventually it will make a more satisfactory base for the crown which, under these circumstances, will probably be required at a later stage.

When using amalgam as the restorative material a lining of glass-ionomer should be placed over the floor of the cavity at least 0.5 mm thick to optimise the bioactivity available from the cement. However, the lining should not significantly reduce the bulk of the restorative material because it may reduce its physical properties. It may also be desirable to place a resin or glass-ionomer/amalgam bond to provide a degree of adhesion between the amalgam and remaining tooth structure. The remaining enamel is generally too weak to withstand the stress of the setting contraction of composite resin even when the restoration is built incrementally. It may be possible to use a lamination technique over glass-ionomer, as described in **Chapter 11**, providing the



Fig. 14.22 A Site 1, Size 3 lesion. Initially this was classed as a Size 2 lesion but following full exploration of the lesion it was classified as a Size 3 because there is a need to protect the disto-buccal cusp.



Fig. 14.23. The same lesion shown in **Figure 14.22**. There was some degree of affected dentine remaining on the floor and this required remineralisation so a glass-ionomer base has been placed.



Fig. 14.24. As this was a load bearing area on an upper molar, and there were no aesthetic considerations of concern, amalgam was the material of choice for the final restoration.

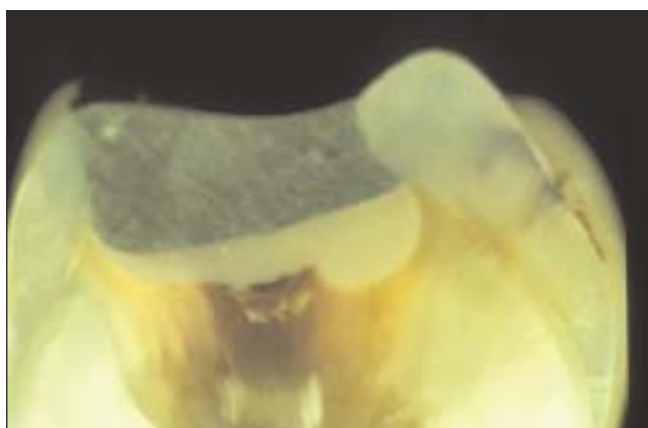


Fig. 14.25. As this was a laboratory exercise the tooth has now been sectioned to show the relationship between the amalgam, the level of protection provided to the buccal cusps and the glass-ionomer base.

occlusal load is not excessive. Observe carefully the anatomy of the opposing tooth and be prepared to modify the length of an opposing working cusp to minimise the depth of the intercuspation between the two teeth. This will reduce the splitting stress on the heavily restored tooth and help to eliminate undesirable contacts during lateral excursions (**Chapter 18**).

Site 1 – Size 4, designated 1.4

G. V. Black classification – Class 1

Preparation

This will be an extensive cavity most likely in a molar tooth. At this size there will have been a fur-

ther breakdown with complete loss of one or more cusps and full restoration with a plastic restorative material will be complex. Amalgam could be utilised for a reasonably satisfactory restoration but generally an indirect extracoronary restoration such as a full or three quarter crown will be required subsequently to completely restore coronal anatomy and occlusion (**Figures 14.26 and 14.27**).

Cavity preparation should be carried out as described above for a cavity classified as Site 1, Size 3. Old restorative material is best removed using tungsten carbide burs at ultra high speed. Use a diamond cylinder at intermediate high speed to enter enamel and round burs to remove the infected dentine. Make sure the walls are clean enough to accept the ion exchange adhesion



Fig. 14.26. A Site 1, Size 4 lesion. A laboratory exercise to demonstrate the extent of a larger lesion where most of the distal cusps appear to be seriously weakened.

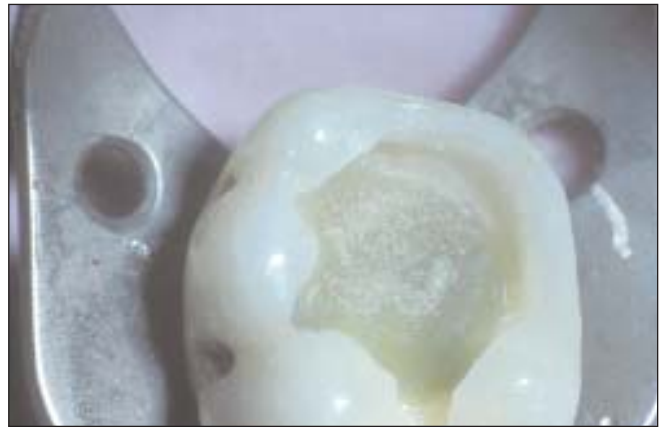


Fig. 14.27. The same tooth as shown in **Figure 14.26** following completion of the cavity design. Note that more than one half of the distal cusps have been reduced so this becomes a Size 4 lesion.

but leave affected dentine on the floor to avoid undue stimulation to the pulp.

In those cases where the caries rate is highly active it may be desirable to carry out the indirect pulp cap technique as described in **Chapter 16**.

Restoration

If amalgam is to be used as the restorative material, mechanical interlocks with the remaining tooth structure are essential using ditches and grooves strategically placed in the gingival area. A resin or glass-ionomer bond may enhance retention still further. Generally, one or more of the remaining cusps will need protection as described before.

As there will have been considerable loss of dentine it will be necessary to lay down a base of glass-ionomer at least 0.5 mm thick to take advantage of its bioactivity and help to remineralise the dentine on the floor of the cavity. It will also be necessary to place a matrix to compensate for the missing enamel wall. Build the amalgam incrementally and condense well. Overbuild the occlusal contour and be prepared to adjust an opposing working cusp to minimise the degree of intercuspation and free the occlusion in lateral excursions (**Chapter 18**).

SITE 2 LESIONS

These are lesions which arise on the proximal surface of either an anterior or a posterior tooth beginning in the region of the proximal contact area.

- **Site 2 – Size 0 (2.0)**

No equivalent in the G. V. Black classification

This lesion represents early demineralisation of the enamel without surface cavitation. It is expected that it can be healed through elimination of the disease and remineralisation.

- **Site 2 – Size 1 (2.1)**

No equivalent in the G. V. Black classification

Surface cavitation of the enamel with minimal dentine involvement which has reached a point beyond stabilising or repair through remineralisation only. It will be identified by radiographs or transillumination.

- **Site 2 – Size 2 (2.2)**

More extensive involvement of the dentine with the marginal ridge weakened or broken down but sufficient tooth structure remaining, following cavity preparation, to support the restoration. It may be a replacement for a small Black Class II or Class III restoration.

- **Site 2 – Size 3 (2.3)**

On a posterior tooth there will be considerable involvement of the dentine with a split at the base of a cusp – or at least the potential for a

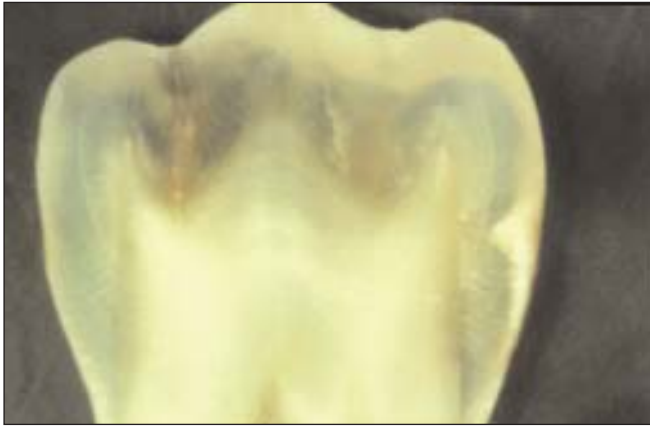


Fig. 14.28. A Site 2, Size 0 lesion. A section through a molar tooth showing the very earliest stage of demineralisation of the proximal surface just below the contact point. The lesion has not yet penetrated the full depth of the enamel and can almost certainly be remineralised and healed. This is essentially a 'white spot lesion'.



Fig. 14.29. There is a 'white spot lesion' on the proximal surface of this bicuspid representing demineralisation but the surface remains smooth and cannot retain plaque.



Fig. 14.30. The tooth shown in **Figure 14.29** has been sectioned showing the lesion has in fact reached dentine. There is the usual translucent zone below the lesion representing the pulpal reaction to the advancing caries. However, the enamel surface is still smooth so it will not retain plaque and it can probably be healed. Surgical intervention should at least be delayed while remineralisation is attempted.



Fig. 14.31. A tooth similar to the one shown in **Figure 14.30** has been sectioned. However, there was surface cavitation already and surgical intervention is essential to overcome plaque retention.

split – with the need to protect one or more cuspal inclines from occlusal load. On an anterior tooth there will be extensive proximal caries with loss of support for the incisal corner which will be deeply undermined.

- **Site 2 – Size 4 (2.4)**

There will be complete loss of at least one cusp from a posterior tooth or extensive loss of the incisal edge of an anterior tooth as a result of either caries or trauma.

The Size 0 or 1 lesion in a posterior tooth will

have to be identified by radiograph or by transillumination. However, neither method is completely accurate and there may remain doubt as to the presence or absence of surface cavitation.

As long as the surface of the enamel is smooth there is the possibility of remineralisation in conjunction with elimination of the disease so a finite decision is required (**Figures 14.28-31**).

Exploration with a sharp probe is strictly contraindicated because, if there is demineralisation, it is certain that this will cause cavitation in the

fragile enamel and the lesion will immediately become at least Size 1. It is often possible to develop some degree of spacing between two teeth using an orthodontic rubber band for 48 hours following which a limited impression can be obtained with a silicone impression material. Surface cavitation will generally be revealed with a reasonable degree of accuracy (**Figure 14.36**).

Whatever the result it is recommended that, in the absence of positive surface cavitation, restraint be exercised and surgical procedures be delayed while the disease is treated first and the lesion kept under observation for some months and, hopefully, healed.

Site 2 – Size 0, designated 2.0

No equivalent in the G. V. Black classification

Early demineralisation of the enamel without surface cavitation. It is expected that the lesion can be healed through elimination of the disease and remineralisation. Application of a resin sealant may be indicated.

Site 2 – Size 1, designated 2.1

No equivalent in the G. V. Black classification

The initial lesion generally commences in the enamel immediately below the contact area and extends facially and lingually in an elliptical shape controlled by the extent of the contact area.

It will generally not involve the contact area nor undermine the marginal ridge or incisal corner to any extent until it has progressed to Size 2. It is often possible, therefore, to maintain the entire proximal surface in enamel. Early demineralisation can penetrate the full depth of the enamel but, providing the prism structure does not collapse, it can be remineralised sufficiently to regain its original physical strength. It may be stained and disfigured but, following removal of the infected dentine within and sealing with an ion releasing glass-ionomer, the proximal wall may remineralise and remain intact. If enamel caries has progressed to the extent that there is actual cavitation of the surface there may be a need to tunnel through to the exterior, to a limited extent, via the dentine cavity, so that the cavitation can be obturated to eliminate further plaque retention but the remainder of the proximal wall can remain intact. In the presence of ions released from a glass-ionomer, as well as external topical applications of fluoride and CPP-ACP, remineralisation will support and reinforce the enamel and prevent further breakdown.

All of these considerations apply to both anterior and posterior teeth and there can be three different approaches considered for restoration of these lesions depending upon its position in relation to the marginal ridge or the presence of a larger lesion in the adjacent tooth.²²⁻²⁴



Fig. 14.32. The distal surface of a molar tooth showing a lesion more than 2 mm from the crest of the marginal ridge. A 'tunnel cavity' is likely to be successful and the marginal ridge can be retained.



Fig. 14.33. The distal surface of a molar tooth showing that there is a demineralised lesion within 2 mm of the crest of the marginal ridge. Cavity design for restoration should be approached with a 'slot cavity' in mind.

Internal occlusal fossa approach (tunnel)

When the enamel lesion is at least 2 mm apical to the crest of the marginal ridge or the corner of the incisal edge the simplest and most conservative approach is through the occlusal fossa just medial to the marginal ridge using the internal occlusal fossa or tunnel approach²⁵⁻²⁸ (**Figure 14.32**). The initial access for this lesion should be as small as possible to preserve natural tooth structure. Maintenance of the original proximal contour with a normal contact area is desirable so removal of enamel should be minimal. The entry should begin just medial to the marginal ridge with careful preservation of remaining enamel.

Slot cavity

If the lesion is closer than 2 mm to the crest of the marginal ridge, and a tunnel approach is likely to leave the ridge too weak to be maintained, access can be gained through the marginal ridge itself (**Figure 14.33**). This design has been called a slot cavity and is probably more commonly used in anterior teeth.

Proximal approach

Finally, if the adjacent tooth already has a size 3 or 4 lesion prepared in it with the entire proximal surface missing there may be direct access to the Size 1 lesion via a proximal approach²⁹ (**Figure 14.54**). The occlusal surface and marginal ridge can remain intact and a very conservative cavity can be designed to remove the caries only. In view of the normal direction of progress of the dentinal caries the marginal ridge will generally not be involved or weakened at all.

Site 2 – Size 1 Internal occlusal fossa approach (tunnel) – 2.1

Preparation

In posterior teeth use a small tapered cylinder diamond bur under air/water spray at intermediate high speed³⁰ (**Figures 14.34-39**). Begin in the occlusal fossa just medial to the marginal ridge. Enter the enamel aiming towards the expected carious lesion. Generally the tactile sense available at this speed will allow the operator to feel when the lesion is entered. Approach carefully, observing progress under magnification and good

illumination until the defect is identified. Now turn the same bur into a more upright position, encroaching into the marginal ridge area to a minimum extent, to enlarge the cavity and improve visibility. Lean the bur towards the facial and the lingual to create a funnel shaped access cavity to the lesion. The entry will now be approximately triangular in outline with the apex towards the central occlusal fossa and the base along the medial aspects of the marginal ridge. The carious dentine will now be directly visible and can be removed with small round burs. It may be necessary to use a bur with a long shank to ensure that the walls and the gingival floor are on sound dentine so that a good seal can be developed around the entire circumference. Note that the removal of all affected dentine on the axial wall is unnecessary, particularly if there is a risk of exposing the pulp. Glass-ionomer will be the material of choice for restoration because it will seal and isolate the area, following which the dentine will remineralise.³¹

The extent of the proximal enamel defect will become clear at this point and a decision can be made on the presence or absence of enamel cavitation. If the enamel is only demineralised and not cavitated it should be left alone to be supported and remineralised through the cement along with elimination of the disease. If the enamel is already cavitated, or needs to be broken through, a short length of standard metal matrix should be placed interproximally and wedged into place to protect the adjacent tooth. Small round burs and small fine hand instruments can be used to complete the cavity design.

It is generally possible, with magnification, to see the gingival enamel margin but access to the occlusal margin is more difficult. However, it is only necessary to remove the seriously broken down enamel because remaining demineralised enamel around the cavity margin will remineralise.

No specific retention design is required because a glass-ionomer will be used for restoration. It can be laminated over with composite resin if it seems necessary. If, at this point, it appears the marginal ridge is cracked or severely compromised a decision may have to be made to remove the marginal ridge, i.e. the cavity may now have to be



Fig. 14.34. This series shows, as a laboratory exercise, the development of a 'tunnel cavity' in the distal of the first molar. The teeth have been mounted in a model and will be treated in proper clinical style.

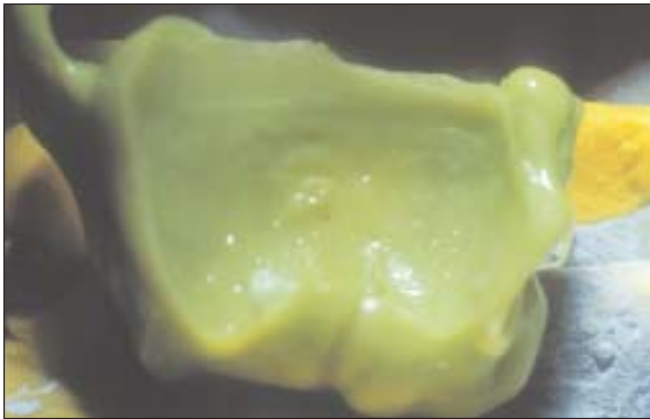


Fig. 14.36. To determine the presence or absence of surface cavitation, and therefore whether or not remineralisation will be sufficient with out surgical intervention, a small silicone-based impression has been taken of the interproximal surfaces. It is apparent that there is cavitation so surgery is indicated.



Fig. 14.38. The distal surface of the restored tooth is exposed to show the limited extent of the proximal exposure. Note the demineralised enamel around the circumference of the lesion. While this enamel is not fully mineralised it is smooth and can remineralise in the absence of disease.



Fig. 14.35. The distal surface of the molar tooth showing the size and extent of the lesion. It is more than 2 mm below the crest of the marginal ridge but is reasonably extensive.



Fig. 14.37. The cavity has been prepared and restored with a fast set, high strength Type II.2 glass-ionomer. Note the limited extent of the cavity entrance.

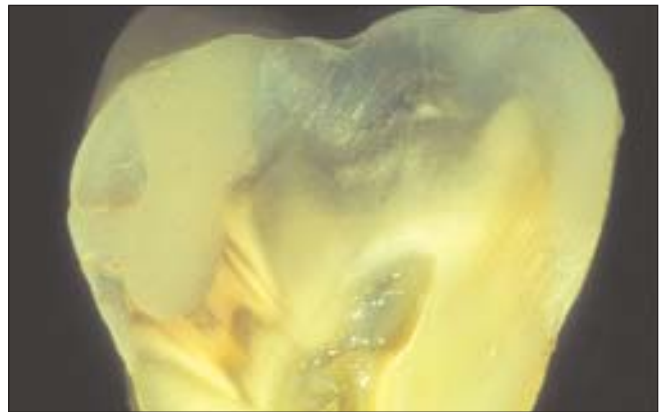


Fig. 14.39. The tooth involved has been sectioned mesio-distally to demonstrate the extent of the cavity and the strength of the remaining marginal ridge. Note the extent of the translucent zone below the restoration representing the defense mechanism of the pulp.

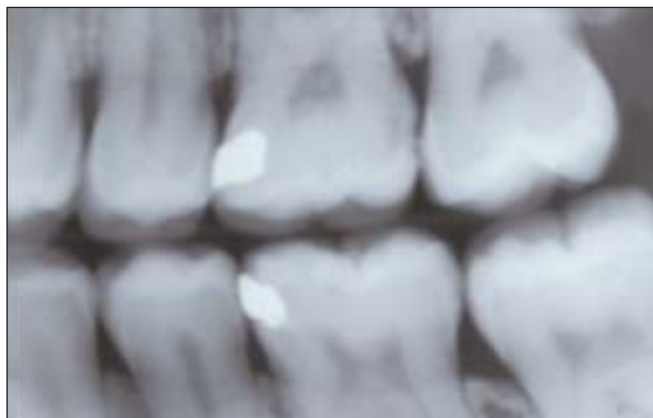


Fig. 14.40. This series demonstrates the advantages of minimal intervention in restorative dentistry. This image shows a radiograph revealing a lesion at the distal of the upper molar. As there was then no alternative it was treated in a conventional manner.



Fig. 14.41. The standard CV Black Class II cavity prepared to restore the lesion. Note that the extension is limited because the patient was expected to control the disease.



Fig. 14.42. The lesion was restored with amalgam and it was photographed 16 years later. Note there is a limited degree of marginal ditching but the restoration is satisfactory.



Fig. 14.43. In the same patient, the contra-lateral upper first molar developed a similar lesion approximately four years later and it was decided to use a minimal intervention approach to restoration.



Fig. 14.44. The lesion has just been restored using a tunnel cavity design and a Type II.1 restorative aesthetic glass-ionomer. The rubber dam is about to be removed.



Fig. 14.45. The same restoration photographed 12 years after placement. It is apparent that the restoration is satisfactory and that the patient is currently free of caries.



Fig. 14.46. A Site 2, Size 1 lesion at the distal of the upper central incisor.



Fig. 14.47. A lingual view of the same lesion. Note the strong marginal ridge on the distal of the crown. It was decided to retain the marginal ridge if possible.



Fig. 14.48. A tunnel design cavity has been prepared and the marginal ridge has been retained thus maintaining the strength in the crown.



Fig. 14.49. Aesthetics was not a problem so the lesion has been restored with a high strength, fast set autocure glass-ionomer.

classified as a Site 2 – Size 2 (2.2) and modified accordingly.

The contrast between the standard G. V. Black Class II cavity design and a tunnel is shown in the series of illustrations (Figures 14.40-45).

In anterior teeth access to the lesion is similar to that described for a posterior tooth and can be gained through either the labial or lingual enamel (Figures 14.46-49). However, in view of the fact that no restorative material can be regarded as a perfect replacement for tooth structure or completely permanent, it is much better to approach from the lingual thus preserving the labial enamel and minimizing aesthetic problems in the future.

An approach from the labial will occasionally be

necessary because of crowding of the teeth with consequent overlapping and difficulty of gaining both access and visibility, particularly when the lesion is in a lower anterior tooth.

Enter the lesion with a very small diamond cylinder at intermediate high speed under air/water spray just medial to the marginal ridge. Extend very conservatively to the incisal and gingival to disclose the extent of the problem whilst maintaining the proximal enamel. Remove caries with small round burs only. There is no need to develop specific retentive elements because normally, simply cleaning the cavity will produce some degree of undercutting. If the proximal enamel is not cavitated in the area of the initial lesion leave it intact so that it can remineralise. In

the presence of cavitation, clean the defect with care whilst protecting the adjacent tooth with a short length of metal matrix strip as described above. Do not extend any further than is essential to eliminate plaque retention because the remaining enamel will remineralise. The presence of undermined enamel is of no consequence because it will be supported by adhesion with the restorative cement. Where possible polish the enamel margins with a 25 μ diamond bur to enhance adhesion.

Restoration

Glass-ionomer is the material of choice for the restoration of both anterior and posterior teeth, with the option of lamination with composite resin if the load bearing area of the restoration involves the entire occlusal support against the opposing tooth.³²⁻³⁴ Condition the cavity as usual. Wash and dry it but do not dehydrate the tooth.

Use a Type II.2 high strength, autocure glass-ionomer that is radiopaque and mixed at a high powder/liquid ratio, preferably capsulated for automated mixing. Use a short length of mylar strip as a matrix and wedge it lightly into place to ensure good proximal contour. Place the cement in two increments using a syringe. Tamp the first increment into the depths of the cavity using a small dry plastic sponge.

If the enamel is cavitated watch to ensure that some excess cement is extruded through between the matrix and the tooth. Add the second increment and tamp again to ensure firm adaptation to the entire cavity wall. As soon as the cement is set, remove the matrix and trim the restoration to a satisfactory occlusion. Apply a resin glaze to seal the cement.

If the occlusal involvement of the restoration is thought to be too great or there is labial exposure of the glass-ionomer and the aesthetic results are less than ideal, it can now be cut back, using a small diamond cylinder under air/water spray, to allow room for lamination with composite resin. Regard the cement as a dentine substitute and remove it to a depth of approximately 2 mm only, sufficient to expose the entire enamel wall. Bevel the enamel as required and acid etch it for 15 sec. only. The glass-ionomer should also be etched for

the same time span. Wash well and apply an enamel bonding resin. Build the composite resin incrementally to full contour and, after polymerising fully, trim and polish as required.

Because it will take up to a week for a glass-ionomer to mature and achieve its final colour and translucency it is desirable to delay a decision to use lamination to improve aesthetics for that length of time.

Site 2 – Size 1 – slot cavity 2.1

There will be occasions when the carious lesion commences high on the proximal surface of a posterior tooth leaving less than 2 mm of the marginal ridge occluso-lingivally or it may already be cracked or otherwise very weak. Under these circumstances it may be wise to approach the lesion through the marginal ridge and produce a small box form cavity sufficient to eliminate the lesion but not extended beyond the demineralised enamel (**Figures 14.50-53**). Under these circumstances it is often possible to maintain a tooth to tooth contact with the adjacent tooth on either the facial or lingual margin, or both, thus facilitating the maintenance of a relatively normal contact area between the two teeth.

Preparation

Open into the lesion using a fine tapered diamond bur at intermediate high speed to maintain a good tactile sense. Extend carefully until the carious lesion is clearly visible and all cavitated enamel is removed. Gentle use of a gingival margin trimmer will allow careful extension without damage to the adjacent tooth. Remove caries with small round burs and ensure clean margins around the entire circumference. If possible maintain a contact with the adjacent tooth. Do not extend medially more than half way through the marginal ridge or the cavity design may have to be modified further (see **Site 2 – Size 2**). Where possible polish the enamel margins with a 25 μ diamond bur to enhance adhesion.

Restoration

In the absence of heavy occlusal load a high strength Type II.2 autocure glass-ionomer alone is



Fig. 14.50. A laboratory exercise to demonstrate a Site 2, Size 1 lesion to be restored with a slot design cavity.



Fig. 14.51. The tooth before mounting in the model showing that the lesion is high on the proximal surface and therefore a slot design is the preferred approach.



Fig. 14.52. The completed cavity showing the limited extension which includes the cavitated enamel only. Note that there remains some areas of demineralisation around the margins which will be expected to remineralise in the presence of the glass-ionomer.

generally sufficient. Use a short length of mylar strip as a matrix wedged firmly into place between the teeth. Condition the cavity as usual and syringe the cement in to place using a fine tip on the syringe or capsule. Allow about three minutes to set adequately, contour if necessary and seal as required with an unfilled resin. Do not allow the cement to dehydrate but it will withstand water contamination the moment it is fully set. If the occlusal load is heavy it may be wise to cut the cement back about 2 mm and laminate it with composite resin.



Fig. 14.53. The model has been reassembled to show the cavity in relation to the adjacent tooth.

Site 2 – Size 1 – proximal approach 2.1

On occasions the preparation of a larger Site 2, Size 3 or 4 cavity will allow good access and visibility to the proximal surface of an adjacent tooth with a Site 2 – Size 1 lesion present on it. Restoration of this cavity is relatively straightforward without the need to involve the marginal ridge or approach through the occlusal fossa (**Figures 14.54–57**).

Preparation

Use a small tapered diamond cylinder bur at intermediate high speed under air/water spray. Access to the lesion and the entry angle will be



Fig. 14.54. A Site 2, Size 1 lesion at the distal of a bicuspid that has been revealed during the preparation of the Size 3 lesion at the mesial of the adjacent molar. It was decided to use the 'proximal approach' to retain strength in the marginal ridge.



Fig. 14.55. The approach to such cavity designs will often be limited by the size and position of the adjacent lesion and long shank endodontic burs may be required.



Fig. 14.56. The final cavity design. Note the presence of demineralised enamel around the circumference of the cavity. However, as this is smooth and will not retain plaque it is expected to remineralise in the presence of the glass-ionomer.



Fig. 14.57. The cavity has been restored with a high strength, fast set, autocure glass-ionomer that is radiopaque.

dictated and controlled to some degree by the cavity in the adjacent tooth but, as the caries is progressing into the dentine in an apical direction and normally does not undermine the marginal ridge at this size, there is no problem removing all the infected layer without unduly weakening the marginal ridge. Remove the enamel as far as is necessary to achieve access to the caries. Use a small round bur with a long shank to clean the cavity, particularly around the full circumference of the walls. A retentive design is not required because an adhesive glass-ionomer will be used for restoration and the walls will generally be slightly undercut.

Restoration

It is essential that the restorative material be radiopaque to avoid subsequent confusion as to whether a lesion is restored or not. As the restoration is not under load, and aesthetics is not generally a problem, a high strength Type II.2 autocure cement is generally the material of choice. Condition the cavity as usual and place a small piece of matrix strip interproximally, lightly supported with a wedge. Syringe the cement into place and apply the matrix firmly over the cement to ensure positive adaptation.

As soon as the cement is set the matrix can be removed and the cement contoured and polished

before proceeding with the adjacent restoration.

Site 2 – Size 2, designated 2.2

G. V. Black Classification – Class II – posterior
– Class III – anterior

A new proximal lesion will be more extensive than a minimal lesion before it is identified with the marginal ridge and proximal surface broken down or so weakened that the previous cavity designs are no longer valid.^{35,36} The extent of the cavitation of the proximal enamel will dictate the classification and, ultimately, the outline form of the cavity. There is no need to remove sound enamel, particularly from the gingival floor, just because it is undermined following removal of caries. The enamel at the gingival is not under occlusal load and can be retained, thus keeping the restoration margin out of the gingival crevice. Neither is there a need to develop dovetail retentive elements on the occlusal of a posterior or the lingual of an anterior tooth because an adhesive restorative material is to be placed which does not require mechanical interlocks. The final proximal outline form will often be curved rather than dovetailed and the generation of sharp point and line angles is strictly contra-indicated because the angles complicate the placement of the restorative material and may lead to stress concentration, resulting in further failure of tooth or restoration.

For a replacement restoration following breakdown of a previous restoration, or the elimination of amalgam³⁷ in favour of a more aesthetic material, the same basic rules will apply, but the cavity will generally be somewhat larger and already extended further than necessary for a modern Site 2 – Size 2 cavity (2.2). It may be verging on a Site 2 – Size 3 (2.3) cavity and a decision will be required on the final design and classification once the cavity is nearing completion (**Figures 14.58-61**).

The decision as to which plastic restorative material to use in a given situation is discussed in **Chapter 19** and the final cavity design will depend on the selection. If amalgam is the material of choice for restoration of a posterior tooth, inclusion of the occlusal fissures is required because it

is not possible to complete an amalgam restoration part way along a fissure. The preparation of an occlusal extension is not relevant to retention of the restoration as a whole in as much as each section of an amalgam restoration must be individually self retentive rather than rely on any one section to retain another. In fact, if a proximal lesion can be restored without involving the occlusal fissures at all then a small proximal box alone may be adequate.

If restoring with amalgam the main retentive form in the proximal box should be placed within the dentine at the gingival as well as in the facial and lingual walls as discussed in **Chapter 10**. If the separate sections of the restoration are individually self retentive, there will not be failure at the narrow isthmus which joins the occlusal extension to the proximal box.

On the other hand, if composite resin and/or glass-ionomer is to be used for the restoration there will generally be no need to open the occlusal fissures any further than recommended for a fissure seal or a Site 1 - Size 1 restoration and there will be no need for a retentive design to be included in the proximal box. Removal of the caries and extension of the cavity outline to include all the cavitated enamel will be sufficient. Weakened or demineralised enamel around the proximal box, particularly along the gingival floor, can be supported and reinforced with glass-ionomer, but facial and lingual enamel must be soundly based on dentine if it is to be a significant factor in retention of a composite resin restoration.

In anterior teeth there are no fissures involved, so a slot preparation is all that is required. Remove the demineralised enamel and infected dentine only and, if possible, avoid including the contact area. Unsupported enamel will be maintained through adhesion with the restoration (**Figures 14.62-65**).

Preparation

For the new lesion, access should be gained with a small cylindrical diamond bur under air/water spray at ultra-high speed beginning just medial to the marginal ridge and aiming towards the carious lesion. Extend facially, lingually and medially with the same bur only as far as is necessary to



Fig. 14.58. A Site 2, Size 2 lesion is to be restored with a composite resin/glass-ionomer laminate. This is replacement dentistry because an old amalgam restoration is to be replaced. This means there is little modification of the cavity design possible.



Fig. 14.59. The amalgam has been removed, a small amount of caries has been removed along the gingival margin and a high strength, fast set, autocure glass-ionomer has been placed as a base.



Fig. 14.60. The glass-ionomer and the surrounding enamel margin is being etched for 10 seconds with a 37% ortho-phosphoric acid. It will then be thoroughly washed and lightly dried. A simple enamel resin bond will then be applied and light activated. There is no need for a complex dentine bonding agent because there is no dentine exposed.



Fig. 14.61. The restoration has been completed with composite resin built incrementally.

expose the extent of the caries. If restoring with amalgam, extend the preparation along the full extent of the occlusal fissure system with the same diamond cylinder. This will produce an extension approximately 1 mm wide, to the full depth of the enamel and just into the dentine, with parallel walls and that will allow adequate condensation of the amalgam. When restoring with composite resin the fissure system need not be involved. However, if fissure caries is suspected the fissure may be opened with a very fine tapered diamond point. There is no need to

remove enamel just to make room for more composite resin (**Figures 14.66-68**).

Remove the caries with small round burs at slow speed. Clean the facial and lingual walls and the gingival floor but leave affected dentine on the axial wall to be remineralised. Cavity outline can now be completed, often with hand instruments such as gingival margin trimmers, removing enamel only if it has been completely denatured. Further extension to the facial or lingual is unnecessary and the walls need not be free from contact with the adjacent tooth. Retain as much gingival



Fig. 14.62. A Site 2, Size 2 anterior restoration is to be replaced because the existing composite resin shows signs of marginal leakage and deterioration in colour match.



Fig. 14.63. The same lesion as shown in **Figure 14.62** seen from the lingual. Note the colour change and the signs of marginal leakage.

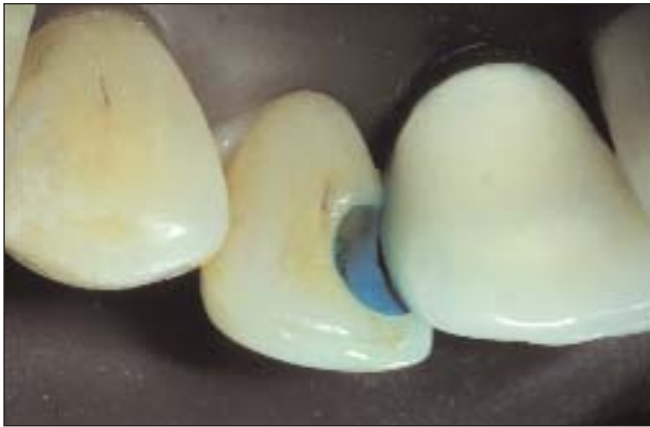


Fig. 14.64. The old restoration has been removed and the cavity is being conditioned with 10% polyacrylic acid to enhance the ion exchange adhesion that will develop between the tooth structure and the glass-ionomer.



Fig. 14.65. The ultimate restoration was a Type II.1 restorative aesthetic glass-ionomer and here it is viewed from the labial. Note the satisfactory aesthetics.

enamel as possible, even if it is undermined and weakened following removal of the caries, because this will keep the gingival margin of the restoration out of the gingival crevice. As this enamel is not subject to occlusal load it can be supported and reinforced through adhesion with glass-ionomer. Weakened and unsupported enamel should not be involved in adhesion using composite resin with the etching technique because it is likely to fail as a result of the setting contraction of the resin.

If the cavity is being modified for replacement of a failed restoration the outline form will already be dictated by the previous cavity design. Remove

old metal restorations at ultra high speed with a tungsten carbide bur taking care not to enlarge the cavity. Cut the metal through and break it out in pieces. Refine the cavity using diamond cylinders at intermediate high speed for improved tactile sense.

Remove caries and old lining material at low speed. If amalgam is to be placed again mechanical retention must be provided in the gingival one third of the crown.

If composite resin is to be placed, mechanical retention is not required because adhesion will be obtained with the enamel through acid etching. If



Fig. 14.66. This is a laboratory exercise showing a new Site 2, Size 2 lesion in the mesial of the upper second molar and a similar replacement lesion in the distal of the first molar.

there are minor undercuts already present following removal of caries these should be restored with glass-ionomer rather than by removal of further tooth structure. Where possible polish the enamel margins with a $25\ \mu$ diamond bur to enhance adhesion.

Restoration

If amalgam is the material of choice for restoration and the caries has progressed to a point further than half way to the pulp chamber, a lining or a base should be placed on the axial wall. A high strength glass-ionomer is the ideal choice because this will act as a thermal barrier as well as provide an ion exchange to stimulate remineralisation on the axial wall.

The cavity and the lining should be covered by a single application of copal varnish or a resin or glass-ionomer amalgam bonding agent. Copal varnish is expected to wash out over a short period of time and this will allow the deposition of amalgam corrosion products to seal the interface. The amalgam bonding agents are expected to provide some degree of adhesion between the amalgam and the tooth structure but the strength will be limited to the tensile strength of the resin or cement. A matrix can now be placed, wedged sufficiently firmly to develop a degree of separation between adjacent teeth, and the amalgam condensed to place. An alloy containing a high percentage of copper with spherical particles is easier to con-



Fig. 14.67. The cavity in the second molar is now completed and shows the limited cavity extension on the occlusal surfaces. The proximal box has been extended to include all the caries but on the occlusal the fissures have been explored in a very limited manner because they are not part of the retention required for adhesive restorative materials.



Fig. 14.68. The replacement dentistry cavity has now been completed in the distal of the first molar. Note the need for full extension in the occlusal sections of the cavity because they were already partly prepared for the original restoration. This is designed as an amalgam cavity and therefore the retentive ditches at the ends of the occlusal grooves.

dense and shows moderately superior physical properties (**Chapter 13**).

If the final restoration is to be composite resin it should always be placed using a lamination technique over a glass-ionomer base (see discussion on lamination in **Chapter 11**). Note that a sublining of calcium hydroxide is unnecessary because the glass-ionomer will provide a seal which is proof against microleakage and it is sufficiently bioactive to assist remineralisation and to cause no lasting pulpal response.

For those same reasons the choice for final

restoration can often be glass-ionomer alone without lamination. In fact the only reason for covering with composite resin would be in situations where the occlusal load is expected to be too great for the glass-ionomer to stand without support. This may well be the case when restoring a posterior tooth but will be rare in an anterior tooth and many deciduous teeth can be safely restored with glass-ionomer alone.

NOTE



Site 2 – Sizes 1 & 2 (2.1 & 2.2) lesions in both anterior and posterior teeth have been described together, because all principles and procedures are the same for both situations. However, when discussing larger Site 2 lesions (2.3 and 2.4) in posterior teeth, which have a potential for split cusps or total loss of cusps, the preparation and restoration phases will be described separately, principally because anterior teeth do not have cusps with their associated potential for fracture.

Site 2 – Size 3, designated 2.3

G. V. Black classification, Class IV – anteriors
Class II – posteriors

2.3. Anterior teeth

This lesion represents an extension of the Site 2, Size 2 (2.2) lesion or, alternatively, a minor traumatic fracture causing damage to the incisal corner of an anterior tooth.

Following trauma the lesion may be relatively minor with adequate healthy enamel around all the margins to develop mechanical adhesion with composite resin through acid etching of the enamel. However, with extensive caries, or replacement of old approximal restorations, the enamel at the gingival margin may be weak and friable and inadequate for retention of the restoration. Under these circumstances it will be desirable to use a glass-ionomer as the dentine substitute and laminate with composite resin over that because the cement does not have sufficient fracture toughness to support the incisal corner on its own.

Depending on the degree of tooth loss this restoration may be followed at a later date by full

crown preparation and under those circumstances it will be desirable to have sound adhesion to remaining tooth structure for the original restoration so that it will remain in place during further preparation (**Figures 14.69-72**).

Preparation

Gain access very conservatively retaining all possible enamel even though unsupported by dentine. Remove all remaining unsatisfactory old restorative material and remove caries from the walls with small round burs. There is no need to remove all affected dentine from the axial wall. Retention through mechanical interlocks, such as dovetail extensions in the lingual enamel, is both undesirable and unnecessary because adhesive restorative materials will be placed and all possible natural tooth structure should be retained. Smooth all enamel margins and remove friable enamel rods. Bevel as required to enhance retention with the composite resin. Where possible polish the enamel margins with a 25 μ diamond bur to enhance adhesion.

Pins are contraindicated because they are very difficult to completely disguise within the restorative material and may result in an unsightly shadow being cast through the restoration. They are also likely to lead to microleakage in the future. There will be occasional exceptions to this where there is extensive tooth loss but under those circumstances an extracoronary restoration is probably indicated.

Restoration

If composite resin alone is to be used the enamel must be well supported with sound dentine around the full circumference because unsupported enamel is likely to fracture as a result of setting shrinkage of the composite. Cover and protect the exposed dentine with a resin modified or high strength autocure glass-ionomer as a dentine substitute.

The micromechanical attachment of the composite resin to enamel through acid etching will then retain the restoration. Etch the enamel and the cement, apply a thin layer of a low viscosity enamel resin bond, blow off the excess with dry air and light activate.



Fig. 14.69. There was recurrent caries beneath a small amalgam restoration in the distal of the canine and the incisal corner was weakened. It was decided to rebuild with a composite resin/glass-ionomer laminate.



Fig. 14.71. The glass-ionomer is now being etched to enhance the union with the composite resin.

If the cavity is extensive and the gingival enamel is either insufficient or too weak to sustain adequate adhesion the restoration should begin with a glass-ionomer as a dentine substitute. The cement should be the strongest possible with a high powder/liquid ratio, preferably capsulated, to ensure optimum physical properties. As soon as the cement is set it can be cut back to expose the enamel margins and make room for the composite resin. Etch the enamel and apply a thin film of a low viscosity resin enamel bond. It is desirable, in extensive cavities, to develop enamel bonding on both the labial and the lingual to enhance the overall strength of retention of the restoration.

Build the composite resin incrementally, begin-



Fig. 14.70. The glass-ionomer has been laid as a base, allowed to set, and cut back to develop a composite resin cavity.



Fig. 14.72. The completed restoration photographed from the labial showing that the composite resin offers some support to the incisal corner but does not fully replace it.

ning with the lingual surface, and use a hybrid resin for strength. Laminate over this on the labial with a microfil composite to enhance aesthetics.

2.3 Posterior teeth

In a posterior tooth, as the carious lesion becomes more extensive, the remaining tooth structure becomes progressively weaker until the point is reached where the restoration must be relied upon to provide some support to the tooth rather than the reverse.^{38,39} Initial opening of an occlusal fissure into dentine will double the length of the cusps and development of a proximal box will tend to double the length again. Occlusal load on



Fig. 14.73. A laboratory exercise where this cavity is expected to become a Site 2, Size 3 lesion because the cusps are likely to require some level of protection against occlusal load.



Fig. 14.74. The cavity is nearing completion and the lingual cusp is being bevelled to allow a buildup of amalgam over it so the amalgam can take the occlusal load.



Fig. 14.75. A tapered fissure bur is now being used to extend a retentive ditch under the base of the lingual cusp as discussed in Chapter 10.



Fig. 14.76. The completed amalgam restoration showing that both the buccal and the lingual cusps have been protected and the amalgam carved to enhance protection.

the cuspal inclines will then produce flexure in the cusps which, on many occasions, will later lead to symptoms of pain on pressure because of the development of a split at the base of the cusp.⁴⁰⁻⁴² Ultimately there is likely to be total failure with loss of tooth structure in bulk (note discussion on protective cavity designs in Chapter 10) (Figures 14.73-76).

Preparation

The steps for preparation of a protective restoration are as follows. Open the cavity conservatively using a small diamond cylinder bur at ultra high speed under air/water spray. To remove an

old restoration use a tungsten carbide bur also at ultra high speed. Remove all remaining caries around the walls but be conservative with affected dentine on the axial wall. At this point determine the extent of the problem and decide if remaining tooth structure requires protection. Identify a split at the base of a cusp if it is present.

For a cusp which is split or at risk modify the cavity outline by leaning the facial or lingual wall outwards in a straight line from the gingival floor to just beyond the cusp tip as described in detail in Chapter 10. Note that this technique can be used to provide protection for any weakened or undermined tooth structure. Support for one half of a cusp or a single cusp is relatively straightforward

but all four cusps on a molar can be protected at once if necessary. However maintenance of full height on at least one cusp will give an indication of the original occlusal height and simplify replacement of the remainder to normal occlusal anatomy. Auxiliary retention is always required⁴³ but pins are contraindicated in most cases.

Retention can now be achieved using the principles laid down in **Chapter 10**. Where possible polish the enamel margins with a 25 μ diamond bur to enhance adhesion.

Restoration

Cavities in this category are generally large and placement of a base of a high strength glass-ionomer is desirable because of its bioactivity. If the floor of the cavity is expected to be very close to the pulp chamber a sublining of a high fluoride, light enhanced, autocure glass-ionomer may be an advantage in stimulating remineralisation.

Because of the size of the cavity, amalgam is generally the material of choice. After establishing the base, condense the amalgam carefully and overfill the cavity by 1-2 mm above each cusp that is being protected. Release the amalgam from the matrix band first with a fine probe until the enamel can be seen below the amalgam overbuild. Shape the buccal and lingual contour before attempting to carve the occlusal surface. The original cusp height can be used as a guide for non-working cusps but care must be taken to maintain the height of the working cusps whilst developing the occlusal anatomy. When adjusting the occlusion with the opposing tooth, be prepared to adjust the opposing tooth as well as the new restoration to avoid deep intercuspation. This is the correct time to relieve lateral stresses on the cusps as well as eliminate interferences in lateral excursions but, at the same time, maintaining a correct centric occlusal relationship between the arches (**Chapter 18**).

If composite resin is preferred for the restoration, glass-ionomer should be placed as a base or dentine substitute with the resin laminated over to replace the enamel. The details of the lamination technique are discussed in full in **Chapter 11** and success will depend on careful application. Of course, by the time a tooth has reached this condi-

tion an extracoronary restoration is advisable but there may be many reasons for avoiding or delaying this move.

Site 2 – Size 4, designated 2.4

G.V. Black Classification, Class IV – anterior
Class II – posterior

2.4 Anterior teeth

This lesion is generally the result of trauma with loss of a major section of the incisal half of the crown. It can also follow extensive caries or breakdown and extension of a Site 2 - Size 2 or 3 restoration. In most cases the occlusal load will not be heavy and the problems of restoration mainly revolve around restoring aesthetics (**Figures 14.77-80**).

Preparation

Following traumatic fracture there will be very little preparation required. Exposed dentine should be protected with a strong glass-ionomer as a dentine substitute or base at the time of restoration. The enamel margins should be bevelled to ensure optimum adhesion with composite resin as well as to blend the union to tooth structure to ensure acceptable aesthetics.

For an extensive carious lesion or replacement of an old restoration care must be taken to preserve as much of the original enamel as possible. Unsupported enamel can be supported to some degree with glass-ionomer so trim the margins to a smooth finish. Remove caries from around the walls only and leave affected dentine on the pulpal wall for remineralisation. Where possible polish the enamel margins with a 25 μ diamond bur to enhance adhesion. Avoid the use of pins for retention because of likely future problems such as microcracks in dentine and a shadow cast by the pin.⁴⁴

Restoration

Composite resin is the only material which can be used successfully under these circumstances but a base of glass-ionomer is essential to act as a dentine substitute and provide reliable adhesion.



Fig. 14.77. A Site 2, Size 4 lesion in an anterior tooth. There was an existing Class III composite resin restoration in the mesial of the upper central incisor. The incisal corner was weakened by the presence of this restoration and eventually failed.



Fig. 14.78. The old restoration has been removed and a resin modified glass-ionomer has been placed to provide adhesion to the dentine. The cavity has been redesigned for the composite resin and the enamel margin only is being etched in preparation for lamination.



Fig. 14.79. The restoration has been completed by incremental buildup of the composite resin.



Fig. 14.80. The completed restoration shown from the labial.

Restore the entire cavity with a high strength autocure or a resin modified glass-ionomer and fully light activate, as required, from all directions. Cut the cement back to expose all the enamel margins, except possibly, the gingival margin if there is no enamel left or it is too weak to allow retention with the resin. Bevel the enamel to develop optimum adhesion with the resin and to smooth over the aesthetic transition of enamel to composite resin. Build the composite resin incrementally, following the lamination technique described in **Chapter 10**. Begin with a hybrid resin on the lingual for optimum strength and laminate with a microfil resin on the labial to enhance the aesthetics. Colour matching with these restora-

tions is a challenge but modern packaging of the materials allows for artistic mixing and blending of various shades and types of composite resin.

2.4 Posterior teeth

Restoration of a Site 2 – Size 4 cavity in a posterior tooth poses further problems because an entire cusp has failed, either from extensive carious attack or as the result of a split. It generally leaves at least one margin beyond the enamel crown margin and on to the root face. Cavity design is then very similar to that described above for the Site 2 – Size 3 cavity except that rebuilding to the correct occlusal height is complicated by the lack



Fig. 14.81. A Site 2, Size 4 lesion in an upper bicuspid that has lost the buccal cusp under occlusal load.



Fig. 14.82. The old amalgam restoration has been removed and the cavity modified to accept a composite resin/glass-ionomer laminate restoration. The entire cavity is fully restored with a high strength, fast set, autocure glass-ionomer which is allowed to set.



Fig. 14.83. The glass-ionomer, once set, is cut back to a composite resin cavity design and the tip of the remaining cusp is lightly bevelled. The entire restoration and the enamel is then etched for 10 seconds, and an enamel bond is applied and light activated.



Fig. 14.84. The restoration is then built incrementally to full contour in composite resin.

of guidance from the missing cusp (**Figures 14.81-84**).

Preparation

Open the cavity and remove all trace of the old restoration using a small diamond cylinder, or tungsten carbide bur as indicated, at ultra-high speed under air/water spray. Remove all caries around the walls to determine the extent of the problem. Remain conservative and retain affected dentine on the axial wall and pulpal floor. Retain any cusp which is based on sound dentine and treat as described for a Site 2, Size 2 cavity design.

Cusps which are undermined or split should be protected as suggested in the design for a Site 2, Size 3 cavity. Retention must be developed in the gingival floor, where ever possible, using ditches and grooves cut with a small tapered fissure bur (**Chapter 10**).⁴⁴⁵⁻⁴⁷ Where possible polish the enamel margins with a 25 μ diamond bur to enhance adhesion.

Restoration

In these extensive cavities amalgam will be the material of choice, where aesthetics is not a problem, because of its superior strength and lack of

flexibility. Placement of a matrix may be complex and adequate condensation will be time consuming. Overbuild and carve back to the correct anatomy. Carve the facial and lingual in relation to protected or restored cusps first. Pay particular attention to the interproximal contour because it is very easy to develop overcontours in such extensive restorations and, at the same time, difficult to develop the correct anatomy in contact areas.

There are limitations on the use of composite resin, particularly for restoration of molars, for a number of reasons. There will be minimal sound enamel available for the development of acid etch adhesion, particularly at the gingival margin, so problems of microleakage may be difficult to overcome. Glass-ionomer can be utilised as a dentine substitute as described in the lamination technique in **Chapter 11** but it, also, requires support from remaining tooth structure. The setting shrinkage of a light activated composite resin can be compensated to some degree by incremental build up but, in an extensive restoration, this becomes more and more difficult and time consuming and the total shrinkage may be too great. Also fatigue under cyclic loading, as well as the long-term water uptake of the present generations of resin-based restoratives, is such that the breakdown under occlusal load may be greater than can be tolerated and may limit longevity of the extensive restoration.

SITE 3 LESIONS

The Site 3 lesion occurs in the gingival one third of the crown or on the exposed root surface of any tooth. Lesions can occur on the open surfaces (facial or lingual) in relation to the contours of the gingival tissue or interproximally, well below, and not related to, the contact area following gingival recession.

- **Site 3 – Size 0 (3.0)**

No equivalent in the G. V. Black classification. Early signs of either active caries or noncaries tooth loss at the gingival margin of any tooth around the full circumference.

- **Site 3 – Size 1 (3.1)**

Early active caries or noncaries tooth loss in the cervical region of any tooth around the full circumference.

- **Site 3 – Size 2 (3.2)**

Advancing caries, replacement dentistry or extensive noncaries tooth loss in the cervical region of any tooth around the full circumference.

- **Site 3 – Size 3 (3.3)**

Advancing caries or extensive noncaries tooth loss at the cervical margin interproximally between any pair of teeth (root surface caries)

- **Site 3 – Size 4 (3.4)**

Advanced caries or non-caries tooth loss at the cervical margin of any tooth where more than one surface is involved and a complex matrix system is required during restoration.

Caries can occur anywhere around the full circumference of a tooth as a result of reduced salivary flow or lack of hygiene leading to plaque accumulation. The carious lesion may have an enamel margin around the full circumference but the usual cavity in this area has an occlusal (incisal) margin in enamel and a gingival margin in dentine.

Root surface caries will also occur anywhere on the root surface following gingival recession. If it occurs interproximally the lesion will be well below, and unrelated to, the contact area. It will be classified as a Site 3 lesion and restoration will generally be undertaken, depending on access and convenience, from either the facial or lingual rather than from the occlusal. If the lesion is approached from the occlusal then it will be classified as a Site 2 lesion.

Abrasion/erosion noncaries lesions are also included in this category. There are three possible causes (**Chapter 5**) and it is desirable to determine the cause and eliminate it at the time of treatment otherwise the tooth loss may continue around the restoration.

Site 3 – Size 0, designated 3.0

No equivalent in the G. V. Black classification

The zero classification suggests that this is an incipient lesion that can be treated simply by elim-



Fig. 14.85. A series of three Site 3 lesions and a decision is required as to whether these are Size 0 or Size 1. The decision will depend upon the presence of active caries, the presence of hypersensitivity or is the lesion lacking in aesthetics. Whatever the cause it must be identified and removed first and then a decision made as to the need for restoration and the material to be used.



Fig. 14.86. The lesion on the upper canine is not hypersensitive but the aesthetics is unsatisfactory so it will be restored with an aesthetic Type II.1 glass-ionomer.



Fig. 14.87. Early signs of active root surface caries suggests that this lesion will need careful monitoring while the disease is treated and arrested. If not successful then a glass-ionomer restoration will be required.



Fig. 14.88. Although this lesion is not active it needs to be restored to improve the aesthetic situation. It is therefore a Size 1 lesion.

ination of the cause. If the lesion shows actual demineralisation it is often possible to simply polish the lesion and lightly recontour the root face. It is essential to draw the attention of the patient to both the lesion and its aetiology and instruct them to modify both plaque removal as well as investigate all other possible causes for the initiation of active caries (**Chapter 3**) (**Figures 14.85-88**).

On the other hand this may represent noncarious tooth loss through erosion (**Chapter 5**). Under

these circumstances elimination of the cause is again the preferred treatment. In the presence of sensitivity there are a number of pharmaceuticals that can be applied for short term relief but it is essential that the patient be discouraged from relying upon these materials. If the cause is not eliminated the lesion will progress to the stage where it becomes a Size 3.1 and requires restoration.



Fig. 14.89. A Site 3, Size 1 lesion is to be restored with an aesthetic glass-ionomer. It was originally recommended that the lesion should be lightly scrubbed with pumice and water but this is optional. There is a risk of damaging the soft tissue so it is not essential.



Fig. 14.90. The soft tissue was slightly damaged so haemorrhage was controlled with a light application of trichloroacetic acid. The restoration is now complete and has been covered with a resin enamel bond to maintain water balance for the next 24 hours.



Fig. 14.91. The same restoration one week after placement. Note the satisfactory aesthetic result.



Fig. 14.92. The same restoration as shown in **Figure 14.91** photographed approximately 12 years after placement. Note the continuing successful aesthetic result.

Site 3 – Size 1, designated 3.1

G. V. Black classification – Class V

Preparation

A carious lesion is generally found at the gingival margin in the presence of a high caries rate and poor oral hygiene routines. A decision will need to be made as to whether remineralisation and improved hygiene will be sufficient to stabilise the situation or whether a restoration should be placed. If a restoration is required it is sufficient to remove the carious dentine only using small round burs. Occasionally, if the enamel is very friable, it may be necessary to extend the cavity out

into the enamel but there will be no need to remove all the demineralised enamel – just that which is beyond remineralisation. Use a small diamond cylinder at intermediate high speed under air/water spray for limited extension. If glass-ionomer is to be placed, the state of the enamel is not important because the continuing fluoride release will encourage remineralisation. If the cavity is to be restored with composite resin the outline will need to be extended to reach sound, fully mineralised enamel which can be safely etched to provide micro-mechanical attachment. As an adhesive material will be used for restoration it is unnecessary to develop a retentive design.

No instrumentation is required for the restoration of an erosion lesion. Take care to avoid damage to the gingival tissue because haemorrhage will interfere with adhesion. Control of gingival seepage and haemorrhage can be achieved with an application of trichloroacetic acid (**Chapter 11**). Alternatively, placement of a short length of gingival retraction cord into the gingival crevice may assist by slightly displacing the soft tissue away from the cavity and to eliminate seepage of gingival fluid (**Figures 14.89-92**).

Restoration

The material of choice is a Type II.1 restorative aesthetic glass-ionomer, either autocure or resin modified, because the aesthetic result is very similar to that which can be achieved with composite resin and the adhesion and fluoride release are superior. An autocure cement can be used and in many cases the aesthetic result will be adequate. Alternatively, if the colour and translucency is not entirely satisfactory, the cement can be laminated subsequently with composite resin.

An erosion/abrasion lesion may be lightly cleaned with a brief scrub with pumice and water on a small rubber cup to remove the biofilm. Do not use proprietary prophylaxis pastes, whether they contain fluoride or not, because they tend to leave a smear layer which will interfere with the adhesion and the ion exchange. Both an erosion lesion or a carious cavity should then be conditioned with 10% polyacrylic acid for 10 seconds, washed thoroughly and dried lightly.

When a resin modified glass-ionomer is to be placed, contour and adapt the cement with a translucent matrix, light activate for 20 seconds, and remove the matrix. Light activate for a further 20 seconds at least to ensure complete activation. Contour and polish immediately with very fine diamonds under air/water spray. Finally, apply a thin coat of the appropriate glaze to seal any remaining surface porosities and scratches.

When using an autocure glass-ionomer prepare the cavity as above (**Chapter 11**). Use a cement with a high powder/liquid ratio, preferably capsulated, and place it with a syringe. Apply a soft tin matrix to adapt the cement well to the surface of the tooth and leave it to set for approximately four

minutes. As soon as it is set remove any excess cement from around the matrix, lift off the matrix and immediately cover the cement with a generous layer of a single component, very low viscosity resin enamel bond to stabilise the cement and avoid water uptake or water loss. Trim the cement further, if required, using a sharp blade only but do not contaminate with water. Develop a reasonable contour and cover with additional resin bond to complete the seal. Light activate the bond and trim the gingival margin if there is an excess of sealant remaining. Complete the contour and polish a minimum of one week later. Seal again with a low viscosity resin to cover porosities and scratches.

If, after a few days, the aesthetics of the glass-ionomer is unsatisfactory, it can be trimmed back with a fine diamond bur under air/water spray to allow for lamination with composite resin. Clean all the enamel at the occlusal margin and bevel it lightly so as to develop optimum micromechanical union. Etch the enamel and the cement for 15 seconds, wash thoroughly and dry lightly. Apply a thin layer of an enamel resin bonding agent, blow off the excess and light activate it. Build the composite resin incrementally beginning at the gingival margin.

Site 3 – Size 2, designated 3.2

G. V. Black classification – Class V

The 3.2 lesion is similar to the 3.1 except that it is somewhat more extensive and possibly more complex to restore. It will normally be a large caries lesion on the facial or lingual surface of a tooth arising from poor oral hygiene technique in the presence of rampant caries. It will sometimes be replacement dentistry (**Figures 14.93-96**).

Preparation

Follow the routine as described above taking care to retain as much natural tooth structure as possible. Note the potential for remineralisation of both enamel and dentine. Use round burs at slow speed to remove infected dentine from the walls and leave affected dentine on the pulpal floor. Use fine diamonds at intermediate high speed to polish the enamel outline and enhance adhesion.



Fig. 14.93. Two Site 3, Size 2 lesions on upper incisors. These restorations have been in place for some years and are now regarded as unsatisfactory. They will be replaced with glass-ionomer.



Fig. 14.94. Both restorations have been removed and a Type II.1 restorative aesthetic glass-ionomer has been placed using soft tin matrices to ensure proper contour.



Fig. 14.95. It was subsequently decided that the aesthetic result with the lateral incisor was not satisfactory so it is to be laminated with composite resin. It has been lightly cut back to make room for the composite and is now being etched for 10 seconds.



Fig. 14.96. The completed restorations photographed three years after placement. Note that it is difficult to determine which is which restorative material. Both are satisfactory.

Restoration

Glass-ionomer is the material of choice and it can be either resin modified or autocure depending on the ability to gain full access for the activation light. Always condition the cavity with 10% polyacrylic acid for 10 seconds before placement. Use the cement at a high powder/liquid ratio for optimum physical properties, place carefully and always use a matrix to apply pressure for complete adaptation.

Site 3 – Size 3, designated 3.3

G. V. Black classification – Class II or Class V

This category represents an approximal lesion which has developed either as primary root surface caries following gingival recession or recurrent caries at the gingival margin of an existing restoration, which is satisfactory in all other respects. Replacement of the entire original restoration may not be necessary. The conservative approach for the maintenance of the maximum amount of remaining tooth structure may be from the facial or lingual, depending on the position of the lesion. Aesthetics will often not be a problem



Fig. 14.97. A Site 3, Size 3 lesion appears as root surface caries at the distal gingival of the lower first bicuspid.



Fig. 14.98. A slot type cavity has been prepared to remove the surface infected zone of caries leaving a soft demineralised affected zone of dentine on the axial floor. Determination of the proper outline of the lesion is often difficult and care should be taken not to overextend.



Fig. 14.99. A short length of metal matrix strip has been placed and wedged between the two teeth to protect the one not involved and the cavity is being conditioned.



Fig. 14.100. The completed restoration at the distal of the first bicuspid showing a satisfactory aesthetic result using a high strength, autocure glass-ionomer.

but access may be limited. Placement of rubber dam will be of assistance and a minor gingivectomy with electrosurgery or laser may be justified (Figures 14.97-100).

Preparation

If there is a risk to the root surface of the adjacent tooth place a short length of metal matrix band and wedge it lightly into place before beginning cavity preparation.

Use a small tapered diamond cylinder at intermediate high speed under air/water spray and approach the lesion from the most occlusal portion

of the caries aiming gingivally towards the most gingival extent of the cavity. Open conservatively sufficient to achieve reasonable visibility. Carry out a gingivectomy if required and control haemorrhage with trichloroacetic acid. Remove caries around the walls and gingival floor with small round burs, with long shanks if required, and define the cavity outline. Take care on the axial wall because demineralised affected dentine can be remineralised^{48,49} and can therefore remain covering the pulp providing the margins are sealed. It is often difficult to identify a pulp exposure in such a cavity and care should be taken to avoid



Fig. 14.101. A series showing restoration of a Site 3, Size 4 lesion. There are three unsatisfactory restorations in place at the gingival margin of the lower right canine. The active disease was treated first before restoration was undertaken.



Fig. 14.102. The lesion viewed from a distal/lingual perspective. Note the defective cermet and the temporary restoration as well.



Fig. 14.103. The completed cavity viewed from the labial. Note that there is a clean wall around the full circumference to allow for the development of the ion exchange adhesion.



Fig. 14.104. The completed cavity viewed from the lingual. Some affected dentine will be left on the axial wall and no calcium hydroxide lining will be placed.



Fig. 14.105. The completed restoration viewed from the labial photographed three years after placement.



Fig. 14.106. The completed restoration viewed from the lingual at the same time interval.

one. If possible retain a wall of tooth structure on the opposite side to the access cavity because this will facilitate construction of a matrix and the ultimate placement of the restoration.

Restoration

A radiopaque, high strength autocure glass-ionomer is the material of choice because of its adhesion and fluoride release. The fast setting Type II.2 cements are useful under these circumstances but, as they tend to lack translucency, aesthetics may be a problem. A resin modified cement can be placed where access for the activator light is not a problem. Condition the cavity with 10% polyacrylic acid and place a matrix and wedge it. If using a metal matrix apply a light coat of a separator such as an unfilled resin. Syringe the cement into place in two increments. Tamp each into place with a small plastic sponge. Wrap the matrix around the tooth and allow the cement to set. Seal with a low viscosity resin sealant to protect the water balance as required. Trim carefully to ensure there is no overhang or overcontour and seal again.

Site 3 – Size 4, designated 3.4

G. V. Black classification – Class V

This category represents a combination of two or more cavities around the cervical margin of any tooth. The typical situation is likely to appear around a lone standing lower canine where a labial 3.2 lesion is joined by another 3.3 lesion on the distal and possibly even another 3.2 on the lingual. Treatment remains similar to the individual lesion but is more complex to carry out (**Figures 14.101-106**).

Preparation

Retain as much natural tooth structure as possible paying particular attention to the pulpal floor of the cavity. The demineralised affected dentine can often be remineralised and, if possible, should not be removed. Follow the recommendations

under the A.R.T. technique (**Chapter 16**) where indicated. Clean the walls only and maintain as much enamel as possible. Condition the cavity as usual prior to placement of the restoration.

Restoration

Glass-ionomer is the material of choice because of the adhesion and fluoride release. The greatest problem will be to construct a suitable matrix to facilitate placement of the cement. One technique is to cut a soft tin matrix to shape and then cut a small hole in an appropriate position through which to syringe the cement. The matrix can then be placed and held with green stick compound or similar and left in place until the cement is set.

An alternative technique is to use a resin modified glass-ionomer and build the restoration incrementally with careful light curing at each stage.

Summary

The foregoing represents a very detailed discussion on the application of a proposed new classification for lesions of the crown of a tooth, both caries and non-caries. It is suggested that the profession should discuss this proposal with some care because it has become quite clear that caries is a disease arising from bacterial activity and this is not subject to elimination or cure through surgery. Simply removing affected tooth structure will not answer the problem and tooth structure is a finite commodity for all our patients. Certainly the profession has become very adept at repairing and restoring tooth structure to a high level of aesthetic success. But this does not overcome the problems of longevity of the restoration and patients are now living far longer than in the past and are becoming even more resistant to being rendered edentulous.

The G. V. Black classification is clearly outdated and does not take in to account the advent of adhesive restorative materials and remineralisation techniques. The profession has a responsibility to review its approach to its basic discipline.

Further Reading

- Black GV. *A Work on Operative Dentistry: The Technical Procedures in Filling Teeth*. Chicago: Medico-Dental Publishing Company; 1917.
- Mount GJ, Hume RW. A new classification for dentistry. *Quint Int* 1997; 28:301-303.
- Mount GJ. Classification for minimal intervention, Letter to the Editor. *Quint. Int* 2000; 31:227.
- Roulet, Desgrange. *Adhesion – The Silent Revolution in Dentistry*. Paris: Quintessence Publishing Co.; 2000.
- Tyas M, Anusavice KJ, Frenken J, Mount GJ. Minimal intervention dentistry – a review. FDI Commission Project 1-97. *Inter Dent J* 2000; 50:1-12.
- Rohr M, Makinson OF, Burrow MF. Pit and fissure morphology. *J Dent Child* 1991; March-April: 97-103.
- Galil KA, Gwinnett AJ. Three dimensional replicas of pits and fissures in human teeth: scanning electron microscope study. *Archs Oral Biol* 1988; 20:493-499.
- Burrow MF, Burrow JF, Makinson OF. Pits and fissures: etch resistance in prismless enamel walls. *Aust Dent J* 2001; 46: 258-262.
- Burrow MF, Burrow JF, Makinson OF. Relative space contribution in fissures from sealants, prophylaxis pastes and organic remnants. *Aust Dent J* 2003; 48:175-179.
- Simonsen RJ. Pit and fissure sealant: a review of the literature. *Pediatr Dent* 2002; 24:393-414.
- Simonsen RJ. Cost effectiveness of pit and fissure sealant at 10 years. *Quint Int* 1989; 20:75-84.
- Roydhouse RH, Richardson AS. The current clinical status of fissure sealants. *J Can Dent Assoc* 1972; 38:219-20.
- Feigal RJ. The use of pit and fissure sealants. *Pediatr Dent* 2002; 24:415-422.
- Roydhouse RH, Richardson AS. The current clinical status of fissure sealants. *J Can Dent Assoc* 1972; 38:219-20.
- Arrow P, Riordan PJ. Retention and caries preventive effects of a GIC and a resin based fissure sealant. *Commun Dent Oral Epidemiol* 1995; 23:282-285.
- McKenna EF, Grundy GE. Glass-ionomer fissure sealants applied by operative dental auxiliaries – retention rates after one year. *Aust Dent J* 1987; 32:200-203.
- Sidhu SK, Schmalz G. The biocompatibility of glass-ionomer cement materials. A status report for the American Journal of Dentistry. *Am J Dent* 2001; 14:387-396.
- Mertz-Fairhurst EJ, Smith CD et.al. Cariostatic and ultraconservative sealed restorations: six year results. *Quint Int* 1992; 23:827-838.
- Elderton RJ. Restorations without conventional cavity preparations. *Inter Dent J* 1988; 38:112-118.
- Mount GJ. Minimal treatment of the carious lesion. *Inter Dent J*. 1991; 41:55-59.
- Mount GJ. The use of amalgam to protect remaining tooth structure. *NZ Dent J* 1977; 73:15-20.
- Hunt PR. Micro conservative restorations for approximal carious lesions. *J Am Dent Assoc* 1990; 120:37-40.
- Hunt PR. A modified Class II cavity preparation for glass-ionomer restorative materials. *Quint Int* 1984; 15:1011-1018.
- Mount GJ, Ngo H. Minimal intervention dentistry – the early lesion. *Quint Int* 2000; 31:535-546.
- Wilson AD, McLean JW. *Glass-ionomer Cements*. London: Quintessence Publishing Co.; 1989.
- Jones SEB. The theory and practice of internal 'tunnel' restorations: a review of the literature and observations on clinical performance over eight years in practice. *Primary Dent Care* 1999; 6:93-100.
- Hunt PR. Micro conservative restorations for approximal carious lesions. *J Am Dent Assoc* 1990; 120:37-40.
- Zenker JEA, Baratieri I, Monteiro S, Andrada MAC, Vieira LCC. Clinical and radiographic evaluation of cermet tunnel restorations on primary molars. *Quint Int* 1993; 24:30.
- Mount GJ. *An Atlas of Glass-ionomer Cements*, 3rd edition. London: Martin Dunitz; 2002.
- Papa J, Wilson PR, Tyas MJ. Tunnel restorations: a review. *J Esthet Dent* 1992; 4:4-9.
- Ngo. HC, Frazer, Mount GJ, Tuisuva J, von Doussa R.J. Remineralisation of dentine by glass-ionomer, an *in-vivo* study. *J Dent Res* 2001; 80: Special Issue (IADR Abstracts) #0919, page 641.
- Hasselrot L. Tunnel restorations: a three and a half year follow up study of Class I and II tunnel restorations in permanent and primary teeth. *Swed Dent J* 1993; 17:173-82.
- Knight GM. The use of adhesive materials in the conservative restoration of selected posterior teeth. *Aust Dent J* 1984; 29:324-331.
- Knight G M. The tunnel restoration – nine years of clinical experience using capsulated glass-ionomer cements. *Aust Dent J* 1992; 37:245-251.
- Mount GJ. The three stages of the amalgam cavity. *Aust Dent J* 1978; 23:75-80.
- Shillingburg HT, Jacobi R, Brackett SE. Preparation modifications for damaged vital posterior teeth. *Dent Clin N Am* 1985; 29:305-26.
- Santos AC, Meiers JC. Fracture resistance of premolars with MOD amalgam restorations lined with amalgambond. *Oper Dent* 1994; 19:2-6.
- Mount GJ. The use of amalgam to protect remaining tooth structure. *NZ Dent J* 1977; 73:15-20.
- Plasmans PJJM, Kusters ST, De Jonge BA et.al. In vitro resistance of extensive amalgam restorations using various retention methods. *J Prosthet Dent* 1987; 57:16-20.
- Doukoudakis S, Doukoudakis A. Amalgam onlay restoration. *J Prosthet Dent* 1991; 66:493-7.
- Cavel WT, Kelsey WP, Blankenau RJ. An *in vivo* study of cuspal fracture. *J Prosthet Dent* 1985; 53:38-42.
- McCulloch AJ, Smith BGN. In vitro studies of cuspal movement produced by adhesive restorative materials. *Br Dent J* 1986; 161:405-409.
- Plasmans PJ, Kusters ST, de Jonge BA, van 't Hof MA, Vrijhoef MM. In vitro resistance of extensive amalgam restorations using various retention methods. *J Prosthet Dent* 1987; 57: 16-20.
- Khera SC, Chan KC, Rittman BRJ. Dentinal crazing and interpin distance. *J Prosthet Dent* 1978; 40:538-543
- Outhaite WC, Garman TA, Pashley DH. Pin vs. slot retention in extensive amalgam restorations. *J Prosthet Dent* 1979; 41: 369-400.

46. Evans JR, Wetz JH. The pinned amalgam restoration. Part 1. A review. *J Prosthet Dent* 1977; 37:37-41
47. Plasmans PJJM, Kustors ST, De Jonge BA et.al. In vitro resistance of extensive amalgam restorations using various retention methods. *J Prosthet Dent* 1987; 57:16-20.
48. Baker C, Noble H, Ngo H, Morris M, Mount GJ. Electron Probe Micro Analysis of in vitro dentine remineralisation. Abstract 1 IADR (ANZ Div.) Perth 2000 75, *IADR* 1998.

15 Pulp Protection During and After Tooth Restoration

W. R. Hume

In **Chapter 2** the dynamics of disease within the pulp were outlined. This chapter describes the measures which may be taken when restoring teeth to prevent pulpal damage, which may cause pain for the patient or lead to the death of the pulp.

The pulp may be damaged and it may die as caries advances through dentine, probably because of the effects of bacteria or their metabolic by-products. Immediately after tooth restoration the pulp can show signs and symptoms of damage, probably because of trauma directly related to the preparation and placement of a restoration. Also, subsequent to restoration, months or years later, the pulp may die, very probably because of the effects of bacterial microleakage.



Avoidance Of Pulpal Damage Due To Caries

Most pulp death is due either directly or indirectly to dental caries.^{1,2} Therefore those strategies which prevent the initiation of caries lesions are likely to preserve pulp vitality at the same time as they conserve tooth tissue. As the pulp will be involved almost immediately caries penetrates through the enamel and reaches the dentine, early interceptive therapy to arrest and cure lesions will protect the pulp from damage (Chapter 3). Dietary control, appropriate administration of fluoride, both systemic and topical, modification of the bacterial flora, fissure sealing, maintenance of salivary flow and good oral hygiene are, therefore, all pulp protective measures.

In the presence of active caries, placement of a restoration is designed to arrest, or at least slow down, the progress of the disease and will therefore contribute to the protection of the pulp. As a general principle, a small restoration is less likely to damage the pulp than a large one. The early diagnosis and accurate prognosis of active caries followed by conservative restoration placement, when indicated, are therefore in the interest of the survival of the pulp, even when total control of the disease is not possible.

Avoidance of Pulp Damage During Cavity Preparation

There is good evidence that cutting dentine damages or kills odontoblasts. The odontoblastic processes may be cut, or sudden movement of fluid, even in the cell-free areas of the tubule, may damage the contents of the odontoblast or disrupt the cell membrane. Deep or extensive cutting of dentine is therefore more likely to cause pulpal damage and should be avoided where possible.

Generation of heat

The generation of heat during tooth cutting has considerable potential for damaging pulpal cells. The use of air/water spray coolant (Chapter 9) is essential when using high speed rotary cutting instruments to clean, lubricate and cool both the tooth and the cutting instrument. The handpiece and the bur must also be in good condition and running concentrically because vibration can also cause damage to the pulp.

Drying the dentine

Dentine is traversed by tubules which normally contain both plasma ultrafiltrate (dental fluid) and odontoblastic processes. Cut dentine is therefore naturally wet. However, it is necessary for the surface of dentine to be relatively dry for the successful placement of some restorative materials. It can be dried using either a jet of high pressure air at ambient temperature or warm air at a lesser pressure from a triple syringe. Organic liquids such as acetone and ether can also be used to take surface water into solution as they wash the dentine, then evaporate. Each method of drying brings with it the possibility of aspiration of odontoblastic cell bodies into dentinal tubules followed by cell rupture. Prolonged drying is more likely to cause damage than that which is relatively brief and gentle, and a risk/benefit assessment may need to be made. The long term benefit may be a more effective bonding of a resin restorative material to the dry dentine surface, while the short term risk is pulpal damage through excessive dehydration.

BE AWARE



Drying and dehydration

There is a difference between drying and dehydration

- drying lightly is acceptable
- dehydration may damage the pulp

Recovery

Odontoblast damage may therefore be inevitable during the preparation of a tooth prior to placement of a restoration and it may be an acceptable

risk, relative to the overall benefit to the patient. If the pulp is free of bacteria at the time of injury there is a strong possibility that the inflammation, which will certainly follow cell damage, can fulfil its physiological function and the tissue will heal. However, the risk is that the pulp may suffer some degree of ‘premature aging’, that is, become more fibrous and less cellular than it was prior to the injury.

Protective Measures During Restoration Placement

Bacterial microleakage

There is strong evidence that bacteria can grow beneath restorative materials and that either bacterial by-products or bacteria themselves can move through dentine to induce pulpal damage, inflammation and death. This concept is generally described as bacterial microleakage. Positive correlations have been demonstrated between the presence of bacteria immediately beneath restorations and pulpal inflammation.³ For example, the species *Prevotella intermedia* and *Prevotella melaninogenica* are known to be able to produce lipopoly-saccharide which may then diffuse through dentine causing a chronic inflammatory response in the pulp, possibly leading on to cell death.

BE AWARE



Bacterial microleakage

- This is the greatest risk to the pulp
- All restorations must be sealed against microleakage

Those restorative materials which are most often associated with pulpal problems following clinical placement are known to permit bacterial microleakage.⁴ Therefore, those materials which are most effective in protecting the pulp probably do so because they provide a seal which prevents such bacterial invasion.

Placement of a lining

If it is necessary to place a restorative material which does not develop a union with dentine a primary barrier should be laid down first using a varnish, a lining or a base, such as glass-ionomer, which adheres fully to the tooth structure. It may also be beneficial to first treat the cavity surface with an antibacterial agent to kill residual bacteria before commencing restoration, providing the agent itself does no harm to the pulp and does not interfere with the adhesion of the subsequent lining or base.^{5,6}

Chemical toxicity

It must be understood that all chemical substances have the potential to be toxic – whether or not they kill cells depends upon the concentration of a substance in relation to a particular tissue. It must also be noted that dentine is an excellent buffer and will limit the diffusion of even the strongest acid to the extent that the pulp will suffer no ill effects, providing there is a reasonably substantial barrier of sound dentine between the two.⁷

However, dentine is not a universal barrier and some restorative materials release chemicals which can diffuse through and damage the pulp. For example, moderate to severe pulpal inflammation may occur within days following placement of an unfilled bonding resin against deeply etched dentine because of the release and diffusion through dentine of toxic chemicals from the resin. Low viscosity resin luting cements, which are placed under considerable pressure during crown seating, can also bypass the protective barrier effect, particularly if the tubules have been opened by acid etching. There are a number of restorative materials which can be placed with relative safety on intact dentine, in spite of the fact that they are sufficiently toxic to cause moderate but reversible pulpal damage if placed directly on exposed pulp tissue. Direct contact

NOTE



Toxicity

All chemicals are toxic depending upon

- concentration in relation to the relevant material
- dentine is an excellent buffer

with pulp means the protective effect of dentine has been lost, and the concentration will then be too high in the pulp.⁸

Recovery

Healthy pulp tissue is able to tolerate mild or moderate physical or chemical damage to the odontoblast layer as long as bacteria and their toxins are not present over a long period. In general, in the absence of bacteria, the death of a limited number of pulp cells will induce a transient, acute inflammation. New cells differentiate from the adjacent pulp to replace those which have died and the patient may only be aware of mild symptoms of pulpitis for a few days⁹ (**Chapter 2**).

Chemical Diffusion and Fluid Flow Through Dentine

The movement through dentine of bacterial toxins, bacteria themselves, or low viscosity water soluble chemicals under pressure, are all possible contributors to adverse pulpal responses. It is therefore worthwhile reviewing briefly what is known about the potential pathways of movement through dentine.¹⁰

Permeability of dentine

Dentine behaves as an impermeable solid traversed by water filled tubules. Fluids can diffuse through dentine only via the tubules and motile bacteria can grow and move within the tubule fluid. The term permeability is used to include all three types of potential movement, i.e. diffusion, fluid flow and bacterial passage through dentine.

The degree of dentine permeability is determined by

- tubule diameter - the diameter decreases with age
- density - there are less tubules at the dentino-enamel junction
- length - proximity of cavity floor to the pulp
- presence or absence of a smear layer

- coagulated protein or calcific deposits e.g. secondary dentine
- restorative materials e.g. cements, resins or varnishes may modify permeability

Modifying factors

Irrespective of other factors, the permeability of cut dentine is substantially increased, by a factor of 4-5 times, by removal of the smear layer which is generated by cavity preparation. Smear layer can be removed rapidly with strong acid etchants, such as phosphoric acid, and a little more slowly with weaker acids, such as ethyldiamine tetracetic acid (EDTA) or polyalkenoic acids. While complete smear layer removal may increase the potential bond strength of some adhesive restorative materials to dentine, it also increases the potential for chemical diffusion from such materials through dentine to the pulp. It will also encourage an outwards fluid flow through dentine and this may prevent the development of an effective seal for the restorative material, thus leading to later ingress of bacteria or their by-products. This means that the decision whether or not to remove smear layer involves an assessment of the risk/benefit ratio.

BE AWARE

Removal of smear layer allows

- increased fluid flow
- increased chemical diffusion
- possible micro-leakage

The relative area of dentine occupied by tubules increases as the cavity floor approaches the pulp. The number of tubules in coronal dentine is approximately

- 20,000 per mm² at the dentino-enamel junction i.e. 1% of dentine area
- 45,000 per mm² close to the roof of the pulp i.e. 22% of dentine area

Dentine permeability increases as remaining dentine thickness decreases towards the pulp because

- tubules are tapered and become larger in diameter
- they converge and become more densely packed
- they become shorter

Dentine is quite dense at the enamel/dentine junction but becomes markedly more permeable over the pulp horns, where tubule density is highest and dentine is thinnest. It is also more permeable through the axial walls of prepared cavities than beneath the occlusal floor. The depth of the cavity, the location of the walls and floor relative to the pulp horns and the surface condition of dentine therefore control the potential for pulpal damage during preparation and restoration placement and must influence decisions concerning the need for pulp protection.

Risks to the Pulp from Plastic Restorative Materials

Glass-ionomer

Although glass-ionomer cement is inherently chemically toxic, the major toxin, unreacted acid, is well buffered by hydroxyapatite following placement into a cavity.^{11,12} Also the ion exchange union between the cement and the underlying tooth structure leads to the development of an ion enriched layer at the interface which effectively seals the tubules against bacterial penetration. Any subsequent failure of the union will be cohesive in the cement rather than adhesive at the interface leaving a fine layer of the ion exchange material still attached to the tooth, sealing the tubules and preventing microleakage.¹³

SUMMARY

Glass-ionomer

- Initial low pH
- Well buffered by dentine
- Seals dentine tubules
- Prevents microleakage

Composite resin

Composite resins present two major problems for the pulp.¹⁴ First they may release chemical toxins, particularly in the first few days after placement, and second, they do not reliably adhere to dentine. Therefore they may be subject to continuing

microleakage. There is some variation between composites in toxin release pattern, and the degree of damage caused by these toxins will depend on the variables, discussed above, which influence dentine permeability.

There can also be some variation according to the cavity geometry. In an intra-coronal box-form cavity which is directly filled with a single increment of composite resin there is a strong likelihood that a gap will form at one or more cavity walls due to the setting contraction of the resin. This is particularly true with the light activated composite resins and the problem needs to be controlled by careful incremental packing and curing. Also, the larger the cavity the greater the problem.

It has been shown that triethylene glycol dimethacrylate (TEGDMA), a small molecular weight diluent used in most composite resins can traverse through dentine, in biologically significant quantities, in the first days after placement. Also most resin restorative materials, particularly those which are light activated, contain varying quantities of hydroxy ethyl methacrylate (HEMA) and this is toxic in tissue culture. The dentine will modify the diffusion to the extent that the pulp response will be mitigated but there should be a degree of caution exercised. The following points should be noted:

- dentine which has just been debrided of active caries is more permeable
- thickness of remaining dentine is very significant
- acid etching of dentine will reduce its buffering effect

It is therefore desirable to first place a base capable of sealing the dentine tubules.¹⁵ Glass-ionomer is the most effective material because it develops an ion exchange adhesion which is proof against microleakage. Use the strongest material available to ensure optimum physical properties because it will need to be strong to counter the setting shrinkage of a light activated composite

SUMMARY

Composite resin

- Releases chemical toxins
- Adheres well to enamel
- Short term adhesion to dentine

resin. The whole of the dentine should be covered so that the cement acts as a dentine substitute and thus provides protection against both chemical toxicity and microleakage. If the lesion is very deep and close to the pulp, carry out an indirect pulp cap routine (**Chapter 16**) first. This will lead to remineralisation of affected dentine with subsequent retention of the maximum thickness between the pulp and the restoration. Removal of all affected dentine from the floor of an active caries lesion is strongly discouraged.

Dental amalgam

Amalgam has a low potential for chemical toxicity, and seals the interface between restoration and tooth structure through corrosion, which commences shortly after placement (**Chapter 13**). There is a moderate marginal gap in the first weeks after placement, but this decreases due to the formation of corrosion products and to the calcification of surface pellicle and other organic materials. If amalgam is used alone as a restorative material without a primary layer of resin, varnish, liner or base there may be pulpal inflammation in the days following placement due to early microleakage. Thereafter, because an effective seal will be developed through corrosion, the pulpitis is likely to subside.

Copal varnish has been recognised for many years as providing an effective seal from microleakage in the short term and, as it washes out, the amalgam corrodes and develops its own organic seal. A low viscosity resin or a thinly mixed glass-ionomer is much more likely to be effective in maintaining an antibacterial seal over a longer term.

SUMMARY ⓘ

Amalgam

- Very low toxicity to pulp
- Marginal seal through copal varnish
remineralising solution
glass-ionomer resin bond

toxicity and will generally be the factor which determines whether or not bacterial microleakage will occur. Zinc phosphate cement, polycarboxylate cement and glass-ionomer cement are the commonly used luting agents and none of these will release chemicals in sufficient concentrations to harm pulpal cells, providing the dentine is intact and the smear layer has not been removed. The liquid for each cement is a low pH acid, but the hydroxyapatite of intact dentine is capable of acting as an effective buffering agent. Undue cleaning or etching of the dentine immediately prior to cementation, however, will open the tubules and, under the high hydraulic pressure which may arise during cementation, may allow some penetration of the liquid through the tubules leading to pulpal irritation. Some of the resin cements, as well as some of the resin modified glass-ionomer luting cements, contain materials with a potential for toxicity or allergy, such as HEMA, and these may find their way into the pulp if subjected to such pressure.¹⁶

Of course, pulpal problems in teeth which require restorations of this magnitude may well be due to pre-operative conditions such as large caries lesions, failing and leaking existing restorations or mechanical trauma during preparation procedures. If there is a pre-existing chronic inflammation, the superimposition of further acute inflammation may well precipitate ultimate pulp death. However, sometimes pulps which appeared to be healthy immediately prior to preparation phase become painful or die shortly thereafter. The cause is not always easy to determine.

Retention of the smear layer

Retention of the smear layer over the surface of a crown preparation is desirable because it occludes the tubules and prevents penetration of the cement liquid, in spite of the relatively high hydraulic pressures which can be developed during cementation.¹⁷ If the smear layer is removed the tubules will be opened, and a low viscosity mix of cement may be forced through the tubules into the pulp space, thus by-passing the buffering effect. The result may then be postinsertion sensitivity. Rather than remove the smear layer it

Risks to the pulp with luted restorations

When indirectly fabricated rigid restorations, such as crowns or inlays, are cemented to place the luting agent may be a potential source of chemical

is better to seal the tubules with a dentine bonding agent, particularly one containing a polyalkenoic acid, or a mineralising fluid such as ITS solution.¹⁸ This is best carried out immediately after preparation of the tooth and prior to taking the impression and making a temporary crown. The preparation will then be completely sealed from microleakage and will remain more comfortable and secure through the further stages to the cementation appointment.

BE AWARE

Luting crowns and bridges

Leave smear layer intact, or seal surface with

- mineralising solution
- two layers of varnish
- resin dentine bond

Resin cements

Lightly filled resin cements can be used for luting crowns and inlays and they have a very low solubility and can be regarded as durable at the margin. However, they do not flow well so the ultimate film thickness is generally greater than desirable. If the dentine is etched there will be a micro-mechanical adhesion but they do not unite chemically with dentine so they are not proof against future microleakage. Also, cleaning and acid treatment of dentine before crown cementation will open the tubules and increase the chance of resin components being forced down tubules under the hydraulic pressures involved with cementation, thus damaging the pulp.

Dissolution of luting cement

Each of the conventional luting cements appears to provide an adequate seal against bacterial microleakage for several years after placement, but the possibility of eventual dissolution of the cements poses a long-term risk to the pulp. Zinc oxide and eugenol cements tend to hydrolyse and convert to a zinc eugenolate and should not be regarded as long term cements. Glass-ionomer cement will last much better and has one additional advantage in as much as the ion exchange layer will remain and will continue to seal the tubules, even after the loss of the cement.

The rate of dissolution may be related to plaque and dietary factors, since cement dissolution tends to be higher in an acidic environment. As long as the crown margins are well adapted and accurate the dissolution will be slower, and it is possible that in some mouths the developing gap may be occluded by calcified debris, or colonised by relatively benign bacteria. However it is not unreasonable to expect pulp death to occur, in some cases, many years after the placement of indirectly fabricated restorations on vital teeth. The best defence is to protect the dentine with an insoluble and impermeable intermediate layer as described above.

Materials Used in Pulp Protection

Varnishes and other surface treatments

Varnishes

A varnish is a material of very low viscosity containing a relatively large proportion by volume of a volatile solvent, and its prime use is for application to dentine in order to decrease permeability. However, because of the high proportion of solvent, and subsequently the large volume reduction on drying, the ultimate film is relatively porous and therefore not very effective. It relies, to a degree, on the presence of an intact smear layer, with which it may combine, to reduce permeability. There are many versions on the market and they were originally used to decrease the early microleakage around amalgam restorations and then wash out to be replaced by corrosion products, and this remains their best use.

Resin sealants

The light activated, unfilled resins may be used as cavity primers or bonding agents with composite resins and they seal dentine more effectively after the smear layer has been removed. Most are relatively viscous and do not set through loss of solvent but by either chemical action or light activation. Some systems are relatively complex and

require several sequential applications to achieve the desired bond. Properly applied they reduce the potential for ingress of bacteria or their by-products. Whether or not the brief, chemical risk they pose to odontoblasts is outweighed by their possible longer term benefits, by enhancing the seal against microleakage, has yet to be determined.

Remineralising solutions

Several chemical treatments using topical fluoride or oxalate salts are designed to reduce dentinal permeability and therefore the risk of ingress of bacteria and their products. Most of these will successfully reduce dentinal sensibility but the long-term benefits of such treatments for pulp health have not yet been established.

Liners and bases

The main differences between a liner and a base is the thickness and strength. The term liner is used for a thin wash. A base is a relatively thick material strong enough to provide resistance form and to become an intrinsic part of the ultimate restoration. A base can be regarded as a dentine substitute.

Calcium hydroxide

Calcium hydroxide was introduced for direct pulp capping on the assumption that it would promote calcification in the wounded tissue against which it was placed, for example, an exposed pulp. It was expected to provide calcium ions thus aiding remineralisation but this has since been discounted. In fact, because it is strongly alkaline at pH 12-13, there will be a degree of necrosis in adjacent soft tissue but, at the same time, bacteria fail to thrive in its presence. Therefore, it will counter bacterial microleakage and, as it is not unduly toxic, exposed and rela-

NOTE

Calcium hydroxide

- Very high pH
- Highly toxic to bacteria
- Causes necrosis to living tissue
- Use in very small quantities only
- Seal with glass-ionomer

tively healthy pulp tissue in the vicinity usually survives. Calcific scar tissue may be laid down beyond any area of necrosis and may successfully bridge the lesion if no bacteria remain.

However, over time, calcium hydroxide is likely to be washed out from under any restoration which does not have a complete marginal seal, so its effect may be transitory. Because of this it is not recommended as a liner or base. Its use should be limited to protection of an actual pulp exposure only. A very small quantity of an auto-cure cement should be placed over the area of soft tissue exposure, then a seal created over it with a glass-ionomer.

Zinc phosphate cement

This cement has been used for many decades as an 'insulating' base material beneath metallic restorations, and also as a luting agent. Despite its acid nature, it is well tolerated by the pulp if placed on intact dentine, presumably because of buffering of the unreacted acid by hydroxyapatite. There may be immediate and short-term pain if placed on the dentine of an un-anaesthetised tooth, probably due to osmotic effects on dentine tubule fluid. As it has no therapeutic effect upon the pulp there seem to be few indications for its use as a lining or base material.

NOTE

Zinc phosphate cement

- Very limited use as a lining only

Zinc oxide-eugenol (ZOE)

ZOE is a useful part of pulp therapy in the management of deep, active carious lesions and has also been used as a lining and base material. Like glass-ionomer, it provides an effective antibacterial seal, probably because any gap between the cement and dentine will contain a high concentration of eugenol, which is strongly bactericidal. Any available eugenol may also inhibit bacterial metabolism within dentine, and if the material is placed on intact dentine it is unlikely to harm pulp cells. It is also possible for it to develop local anaesthetic and anti-inflammatory reactions in adjacent pulp tissue. Despite these therapeutic

benefits, the cement slowly hydrolyses with time, leaving a residue of soft zinc hydroxide. Also, eugenol will inhibit the polymerisation of composite resin, so it must not be used anywhere prior to, or in relation to, resin restorations or resin luting cements.

Neither should it be used in direct contact with exposed pulp tissue, since the release of eugenol by hydrolysis is markedly greater due to the wetness of the tissue. A concentration of eugenol sufficient to kill pulpal cells may develop rapidly in adjacent vital tissue, and the level may be sustained for several days.

Indications for the use of zinc oxide-eugenol are limited to those situations where the dentine is intact and some form of indirect pulp therapy or caries therapy is required. For example, it is an effective temporary restoration in those situations where it is intended to remove infected surface caries and leave demineralised affected caries behind. Generally, within three weeks it will be safe to proceed with a permanent restoration. Because of the problem of long term hydrolysis, its use should be limited to less than three months as a temporary restoration.

NOTE



Zinc oxide and eugenol

- Anti-inflammatory
- Antibacterial
- Mildly anaesthetic
- Good seal against microleakage

Glass-ionomer

As noted above glass-ionomer, when placed without pressure on intact dentine, poses no chemical

risk to the pulp and, with the development of the ion exchange layer, it creates an effective antibacterial seal. It shows a very low solubility, and therefore appears to be the material of choice for use as a base, or dentine substitute, beneath all plastic restorations.

Glass-ionomer also has considerable potential as a long term temporary, or provisional, restoration in the treatment of active caries. Following removal of infected dentine a strong mix of glass-ionomer is placed

in the cavity over the remaining affected dentine and allowed to set. In the presence of dentinal fluid from the affected dentine there is likely to be a reasonable release of fluoride as well as calcium and phosphate ions from the cement and these will be useful in the remineralising process. The cement will adhere to remaining tooth structure through the ion exchange mechanism and, in addition, is apparently mildly antibacterial because of fluoride release.

Glass-ionomer is the material which has been recommended for the A.R.T. technique (**Chapter 16**) and it has been shown to be very effective in stimulating remineralisation of affected dentine over a short period of time, providing it is used with a high powder content.

NOTE



Glass-ionomer

- Ion exchange adhesion
- No microleakage
- Antibacterial
- Stimulates remineralisation

Further Reading

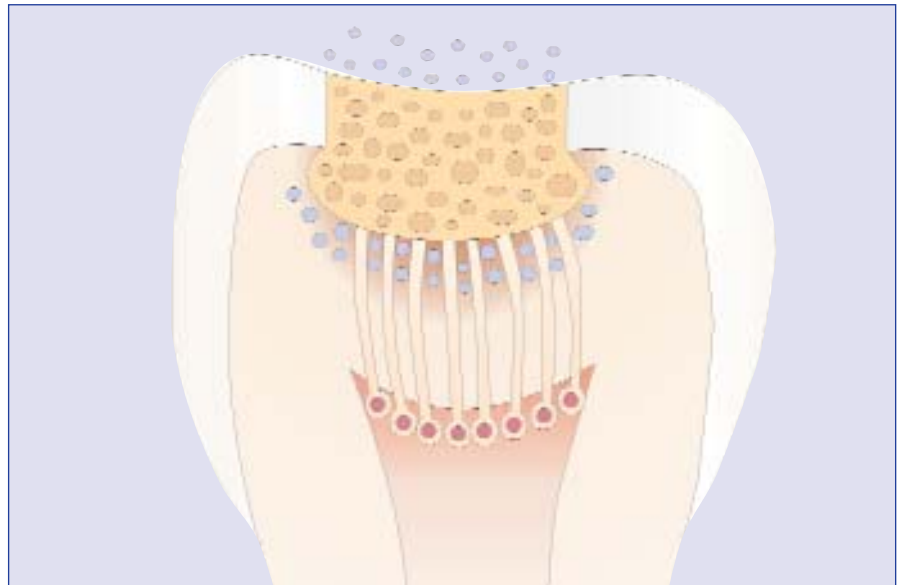
1. Brannstrom M. *Dentine and Pulp in Restorative Dentistry*. London: Wolfe Medical Publications Ltd.; 1987.
2. Brannstrom M, Vojinovic O. Response of the dental pulp to invasion of bacteria around three filling materials. *J Dent Child* 1976; 43:15-21.
3. Cox CF, Keall CL, Keall HJ, Ostro E, Bergenholtz G. Bio-compatibility of surface sealed dental materials against exposed pulps. *J Prosthet Dent* 1987; 57:1-8.
4. Trope M, Orstavik D. Biologic responses of the pulp to restorations. In: *Glass-ionomers: The Next Generation*, ed. Hunt PR. Philadelphia; 1994.
5. Murray PE, Hafez AA, Smith AJ, Cox CF. Identification of heirachial factors to guide clinical decisions for long-term pulp capping. *Quint Int* 2003; 34:61-70.
6. Pashley DH, Depew DD. Effects of the smear layer, copalite and oxybate on microleakage. *Oper Dent* 1986; 11:95-102.
7. Fusayama T. A simple pain-free adhesive system by minimal reduction and total etching. *Ishiyaka Euro America* 1993 pp. 6 & 74.
8. Hamid A, Hume WR. Diffusion of monomer through carious dentin *in vitro*. *J Dent Res* 1995; 74:430.
9. Hume WR. Pulpal responses to restorative materials. *Dentistry Today*, Spring 1989.
10. Hume WR, Gerzina TM. Release of monomers from bonding resin – composite resin combinations through dentin *in vitro*. *J Dent Res* 1994; 73:224.
11. Gerzina T, Hume WR. Movement of glass-ionomer cement through human dentine during crown cementation *in vitro*. *J Dent Res* 1990; 69:934.
12. Hume WR, Mount GJ. *In vitro* studies on the potential for pulpal cytotoxicity of glass-ionomer cements. *J Dent Res* 1988; 67:915-918.
13. Mount GJ. Lessons from the early days. In: *Glass Ionomers: The Next Generation*, ed PR Hunt. Philadelphia; 1994; p92.
14. Gerzina T, Hume WR. TEGDMA elution from resin composite through dentin *in vitro*. *J Dent Res* 1993; 71:162.
15. Snuggs HM, Cox CF, Powell CS, White KC. Pulpal healing and dentinal bridge formation in an acid environment. *Quint Int* 1993; 24:24.
16. Johnson GH, Powell LV, Derouen TA. Evaluation and control of post-cementation pulpal sensitivity. *J Am Dent Assoc* 1993; 124:39-46.
17. Pashley DH, Michelich V, Kehl T. Dentine permeability; effects of smear layer removal. *J Prosthet Dent* 1981; 46: 531.
18. Causten BE, Johnson NVV. Improvement of polycarboxylate adhesion to dentine by the use of a new calcifying solution. *Br Dent J* 1982; 152:9-11.

16 Vital Pulp Therapy

G. J. Mount ♦ W. R. Hume

In addition to procedures designed to keep the pulp alive by avoiding pulpal damage there are several routine measures available to help heal a damaged pulp. Whether the pulp will heal with the aid of a particular therapy or not will depend to a large extent upon the balance between the damaging factors and the host response. The richness of the blood supply could well be a determining factor. Therefore, the chances of success are probably better in youth than in the aging patient because continuing mineralisation within the pulp chamber and root canals will reduce the circulation and therefore the ability to respond to stimulus.

It is impossible to accurately predict the outcome of a particular therapy when the principal pulpal irritant is microbial because both the microbial mix and the host response vary greatly. These two factors can vary between individual patients, between teeth and, over time, in the same patient. Despite this, several therapies have been applied with sufficient likelihood of success to have made them accepted and reasonable parts of the practice of dentistry.



Indirect Pulp Therapy

The ability of the pulp to respond satisfactorily to closed trauma, such as a brief period of dehydration or heat during cavity preparation, is very good. However, the introduction of bacterial infection to an exposed pulp can bring about rapid pulp death. This means that exposure of the pulp to the oral environment carries major risks to the survival of the tissue. So it is desirable to keep the pulp covered at all times, providing this can be

done without compromising other treatment goals. The traditional concept of removal of all softened demineralised dentine from the floor of a cavity is therefore fraught with danger. It must be recognised that the lower layers of dentine, even though softened, are not necessarily infected and can often be retained.¹

BE AWARE



- Bacterial infection is the only irreversible insult to the pulp
- Pulp has very high powers of recovery from other insults

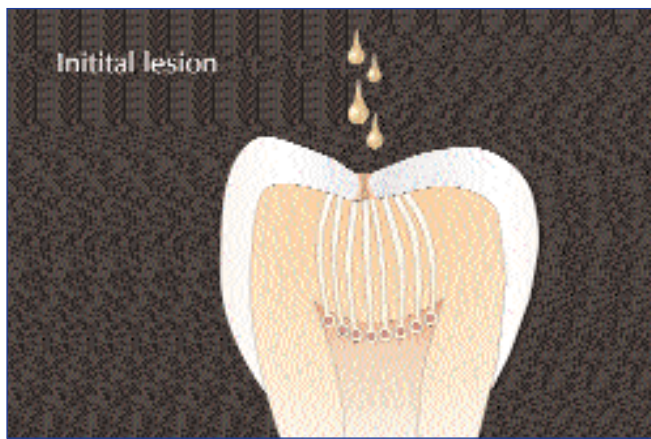


Fig. 16.1. The first of a series of diagrams to show progress of the carious lesion and how to deal with it in the most conservative manner. This represents the earliest penetration of caries down through a fissure with demineralisation down the walls but no involvement of the dentine at this point.

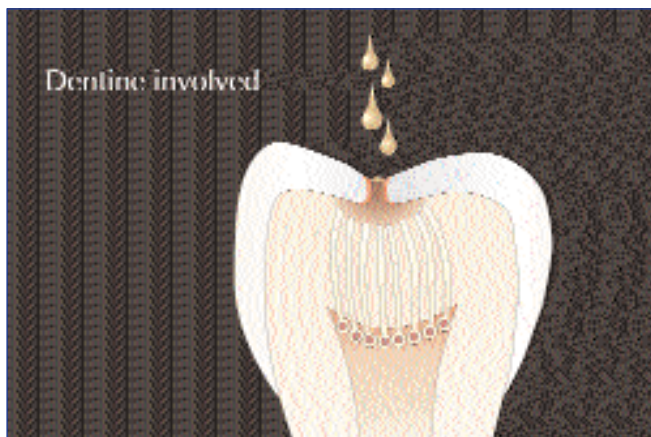


Fig. 16.2. Once the caries reaches the dentine there will be a lateral spread through the lateral canals within the dentine along with a degree of penetration into the bulk of the dentine.

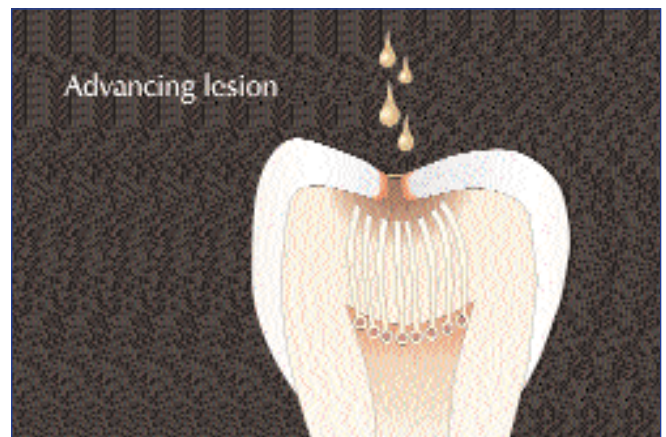


Fig. 16.3. There will be a very early response from the pulp with the generation of a degree of inflammation as well as deposition of mineral ions into the lateral canals of the dentine immediately subjacent to the lesion.

Infected layer

A carious lesion will commence within the enamel as simple demineralisation. Initially there will be no surface cavitation and therefore no retained bacteria except those within the plaque accumulated on the surface. At some point the enamel will become sufficiently porous that there will be a level of response to the demineralisation within the dentine in the form of hypermineralisation of the dentine tubules immediately subjacent to the early lesion. The lateral canals joining the tubules together will become blocked up to some extent through mineralisation suggesting that the pulp

is capable of a very early response to carious attack. This can be seen under the microscope as a translucent layer because the transmission of light through the dentine will be altered. As the lesion extends and the enamel surface breaks down and becomes cavitated, it is possible for bacteria to take up residence. The bacterial population will be modified and the level of activity will expand. The defences of the pulp will be overcome and the dentine, in spite of the hypermineralisation, will become demineralised. Thereafter the outer surface of the lesion, adjacent to the oral cavity, will be heavily infected and the dentine

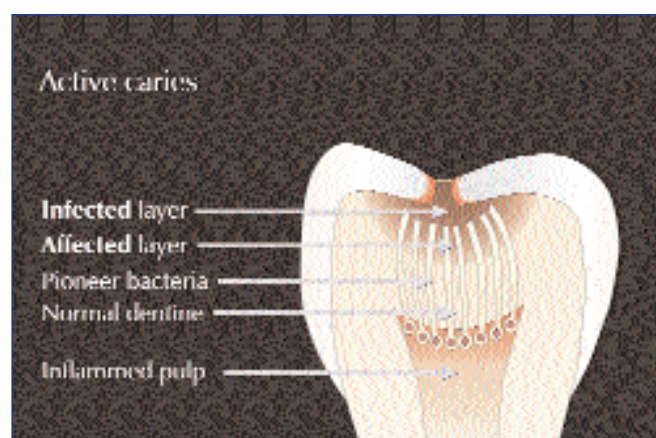


Fig. 16.4. The layers of the caries lesion will now be clearly defined histologically but hard to define clinically.

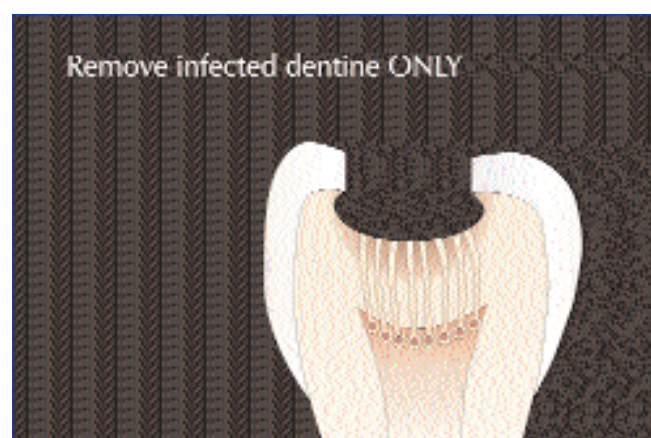


Fig. 16.5. Treatment should aim at removal of the infected layer only with definition of a clean margin around the circumference of the lesion to allow for the development of an ion exchange adhesion with the glass-ionomer transitional restoration.

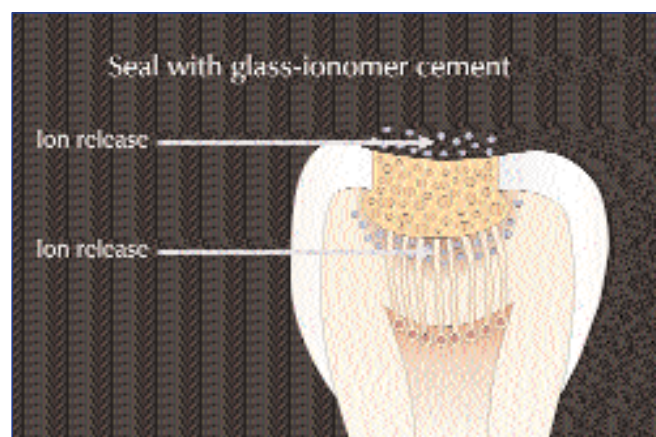


Fig. 16.6. Placement of a transitional restoration that completely seals the remaining lesion will allow for remineralisation and healing of the dentine through the ion release.

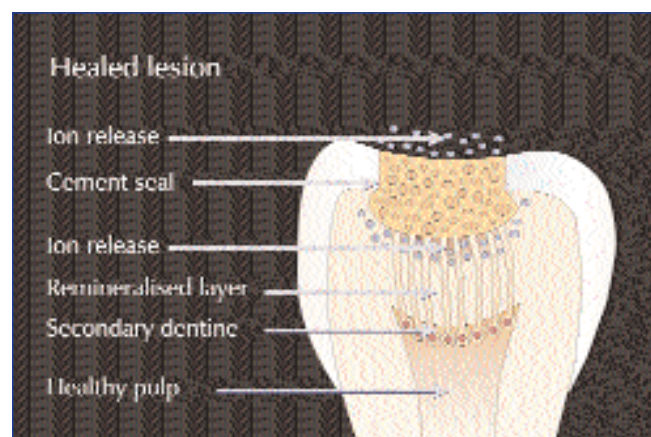


Fig. 16.7. Subsequently the pulpal inflammation will subside quite rapidly and it has been shown that new ions – either calcium or strontium – will be deposited to remineralise the dentine and heal the lesion.

will become seriously involved. The collagen framework of the tubules, now totally demineralised, will be subject to disintegration by proteolytic bacteria and will become denatured and structureless. It will no longer be possible to remineralise this part of the tooth structure so it will need to be removed as part of the reconstruction and repair. This layer is known as the infected layer.

NOTE



Progress of caries

- Demineralisation of enamel
- Demineralisation of dentine
- Pulp initial response
- Hypermineralisation of dentine – translucent layer
- Cavitation of enamel
- Bacterial colonisation
- Breakdown of collagen in dentine
- Further pulp response

Affected layer

However, the demineralisation of the dentine will always progress in stages well in advance of the proteolysis and the complete breakdown. The lower layer of the lesion will be softened but not infected. This zone of demineralised dentine will retain the basic dentine structure with the collagen framework of the tubules still extant, but demineralised to some degree. It would appear that, even where there is only limited mineral remaining attached to the collagen, it is still possible to remineralise the tubules and heal the lesion. This layer will be relatively free of bacteria except for, possibly, a few pioneer bacteria and therefore can be retained and probably healed. It will be preceded always by the translucent layer representing the hypermineralisation stimulated by the pulp. The lateral canals will be sealed and this in turn will limit the speed of penetration of the acid and toxins being produced by the bacteria. It is desirable, then, to retain the affected dentine and offer it some stimulus to completely remineralise itself. Removal is both unnecessary and undesirable because of the potential for further irritating the pulp or, worse still, developing a

mechanical exposure because that will leave the pulp open to the introduction of bacteria. In other words, every effort should be expended to bring about healing of the affected layer rather than its removal. This should be regarded as one of the corner stones of minimal intervention dentistry.

BE AWARE



Arrest caries progress

- Gain access to lesion
- Remove superficial infected dentine
- Seal lesion with adhesive restorative
- Remaining bacteria will be **isolated** and **inactivated**

Clinical routine

The removal of the superficial layer of infected dentine is a major component of the surgical treatment of an advancing caries lesion and this is relatively simple to accomplish. However, the infected zone is not always easy to define because it is quite soft and about the same colour as the surrounding dentine including the softened affected layer beneath it. The caries disclosing solutions that are presently available are not particularly reliable in differentiating between the two layers in as much as they disclose bacteria rather than demineralised dentine. There are certain commercial products that claim to reliably lead to removal of infected dentine only but these also appear to allow excessive removal of dentine that can otherwise be remineralised. It is suggested, therefore, that caries control in an extensive lesion should be carried out in two stages. The first stage is removal of the infected layer to the extent that there is now room for a transitional restoration. The second stage requires sealing the entire lesion to isolate any remaining bacteria from further nutrition and thus inactivate them.

- Stage 1 – Gain limited access to the lesion through removal of enamel around the margins. Carry out limited debridement of the surface of the lesion to remove only the infected dentine and clean the walls around the complete circumference of the lesion sufficient to expose sound healthy dentine. This will allow

for the development of an ion exchange adhesion and a complete seal between the restoration and the tooth thus eliminating micro-leakage. Remove sufficient tooth structure so that the temporary restoration will have the strength to withstand the occlusal load.

- Stage 2 – Place a temporary restoration, using a material which has a level of bioactivity capable of providing a complete marginal seal. Leave in place for six weeks at least.

This routine will allow time for any pulpal inflammation to subside and for the pulp to lay down an initial increment of reparative dentine in those areas closest to the lesion. Over time, secondary dentine formation will follow within the pulp chamber because the tissue fluid in the pulp is naturally supersaturated with calcium and phosphate ions. In the absence of bacteria, the pulp has a high potential for repair. If the pulp survives the inflammation and retains vitality there will also be a degree of remineralisation in the affected demineralised zone. In the presence of a bioactive restorative material this activity is likely to continue for some time although, at this time, it is not possible to guarantee full remineralisation.

Temporary (transitional) restoration

The main function of the temporary restoration is to provide a complete seal so that any remaining bacteria will be deprived of nutrition and will not be able to produce sufficient acid for demineralisation to continue. Further advantages will accrue if the temporary cement has an antiseptic or antibacterial effect on the remaining micro-organisms. In addition, if it is bioactive it will help stimulate remineralisation of the collagen framework of the dentine tubules and the affected layer may heal entirely.

Longevity of the temporary

The temporary restoration should remain for a minimum of three weeks but no longer than a maximum of six months following which the situation should be reassessed. By this time pulpal inflammation will have subsided and there will have been some degree of secondary dentine formed within the pulp chamber. There will now

be two clear alternatives available and these require a level of experience and clinical judgement.

Alternative #1

There may be a sense of insecurity in leaving a relatively substantial layer of affected dentine behind so the operator may choose to remove the entire temporary material for reassurance of success. If glass-ionomer was placed as the transitional restoration there will have been a degree of remineralisation in the pulp as well as the affected layer. There may be some discoloured, demineralised dentine remaining but, in view of the continuing potential for remineralisation, some of this can often be quite safely left on the floor of the cavity. In fact, cleaning the lesion down to a hard shiny dentine surface is unnecessary and undesirable because of the loss of dentine that may still be able to be remineralised. Worse still, it may result in a pulp exposure.

Having determined that remaining demineralised dentine is now firmer and leathery in texture a permanent restoration can be placed using, for preference, a glass-ionomer lining or base. A similar situation may well arise following placement of a zinc oxide/eugenol temporary restoration but, because this material hydrolyses relatively rapidly and does not stimulate remineralisation, it should not remain beyond six weeks.

Alternative #2

If the operator feels confident of success there may be no need to remove all of the transitional restoration. The bulk of the material can be left behind as a base for the final restoration. Cut it back far enough to make room for another material and laminate over it to restore full anatomy and physical properties and create a permanent restoration. Always keep in mind the physical properties of the laminate and make sufficient room to ensure longevity. Some materials can withstand occlusal load better than others.

The decision as to which alternative to undertake can only be decided by the clinician and will depend upon the initial assessment of the amount of affected layer remaining beneath the restoration. If there is doubt and any insecurity then it

may be wise to remove the entire temporary and reassess knowing that the risk of an accidental exposure is always there. On the other hand, assuming the tooth is free of symptoms, the alternative technique is very conservative of tooth structure and therefore the method of choice.

This two stage sequence of debridement covering a period of time is known as indirect pulp therapy because it is designed to avoid a pulp exposure while allowing the tissue to heal. It has also been called indirect pulp capping because it is intended that the potential pulp exposure will be capped by secondary dentine before the area of exposure is reached during the second stage of debridement. The term transitional restoration is coming into common usage particularly if the second alternative treatment is the one of choice. The life span of a glass-ionomer temporary restoration can be quite lengthy, limited mainly by the difficulty of restoring full coronal and occlusal anatomy with proper proximal contour and a well formed marginal ridge.

NOTE



Indirect pulp therapy

- Gain access to the lesion
- Remove **infected** layer of dentine
- Retain **affected** layer of dentine
- Seal lesion with bioactive restorative material
- Leave in place minimum of six weeks
- Make a clinical decision to retain some or remove entirely

Choice of temporary restoration material

For many years the preferred temporary restoration material was a zinc oxide/eugenol paste.

- It provides an adequate seal, excluding most if not all dietary substrate from the remaining micro-organisms in dentine.
- It releases eugenol into adjacent dentine, including demineralised dentine, the rate of release being determined by the wetness of the tissue.
- Eugenol is probably effective in killing residual bacteria.
- Sufficient eugenol may diffuse through the

dentine to the pulp space to inhibit inflammation and pain (**Chapter 2**).

The principal shortcomings of zinc oxide-eugenol in this role are its limited mechanical strength and its limited durability for the longer term because it degrades through hydrolysis. Various resins have been included to modify its formula in order to increase both strength and durability, but fracture strength and wear resistance are not sufficient and the expected life span is therefore limited.

Glass-ionomer is now the material of choice because it is more durable and has the added advantage through its bioactivity of releasing calcium, strontium and phosphate ions as well as fluoride.

- Glass-ionomer is relatively insoluble.
- It is sufficiently strong to withstand reasonable occlusal load.
- It adheres well to clean dentine.
- It is easily placed and relatively easy to remove.
- It releases fluoride which has the potential to kill bacteria in dentine.
- It releases calcium, strontium and phosphate ions, all of which are available for remineralisation of adjacent hard tissues.

The A.R.T. Technique

The term A.R.T. stands for Atraumatic Restorative Treatment and this treatment method was originally developed for use in underdeveloped countries where full dental care is not always available.⁴ In some of these communities Western style dietary routines have been adopted prior to the development of preventive dental measures with a resultant high caries rate. The only alternative dental treatment to date has been extraction of affected teeth leading, usually, to further dental problems.

The A.R.T. technique takes into account the use of the indirect pulp therapy, as described above, through placement of a fast setting glass-ionomer for the stabilisation of rampant caries. It is assumed that there is no electricity to power a



Fig. 16.8. This is the first of a laboratory series showing the purpose of the A.R.T. technique. There is no variation in principle from the first series shown above.

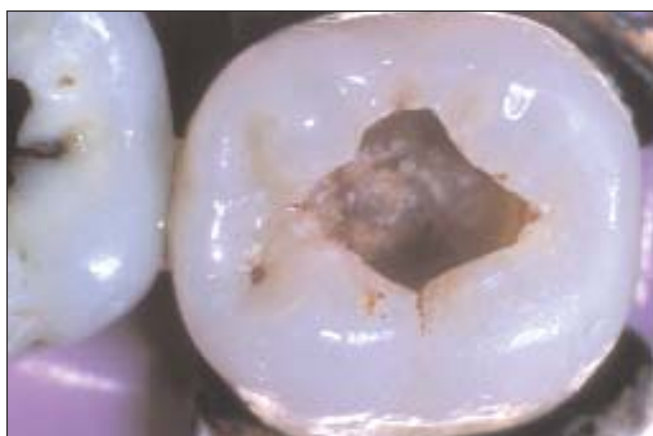


Fig. 16.10. The completed outline of the cavity. There is no need to extend through to the end of the fissures because those can be sealed at the time of placing the transitional restoration. At this point the infected dentine can be removed from the walls around the full circumference using spoon excavators. Enamel may well be undermined but this is of no account.

rotary cutting instrument or proper suction for isolation of the cavity. Auxilliary lighting is limited and the patient is lying recumbent. The routine for restoration placement can be carried out quite simply, using hand instruments to gain access through the enamel and spoon excavators to remove the superficial infected dentine (**Chapter 9**). Care must be exercised to make sure the walls of the cavity are completely clean but leaving a layer of affected dentine over the pulp chamber. Local anaesthesia is not required because the demineralised dentine will not be sensitive. However, some discomfort may be experienced



Fig. 16.9. Access through the undermined enamel has been gained using sharp hand instruments such as chisels or hoes.



Fig. 16.11. The cavity is now conditioned with polyacrylic acid on a cotton sponge for about 10 seconds only. Further sponges can be used with water only to wash the excess conditioner away and leave a clean cavity.

once sound dentine is reached and this can be used as a guide in determining the extent of the cavity (**Figures 16.8-14**).

The completed cavity is then conditioned with polyacrylic acid, applied for 10 seconds on a cotton pellet, then washed with wet cotton pellets and dried again. A high physical property, fast setting, autocure, glass-ionomer is placed in the cavity and allowed to set. A gloved finger can be used as a matrix to apply pressure for positive placement, as well as to keep the cement isolated during the initial setting phase. The restoration should be kept isolated for approximately two minutes at which



Fig. 16.12. The strongest available glass-ionomer is then mixed as directed by the manufacturer and placed into the depths of the cavity. A slight excess is applied on the top and a gloved finger is used to apply pressure to the cement to ensure full adaptation to the cavity walls and floor. Keep the finger in place for 2 minutes to allow full setting of the cement. Roll the finger off the restoration once it has set and finally adjust the occlusion.

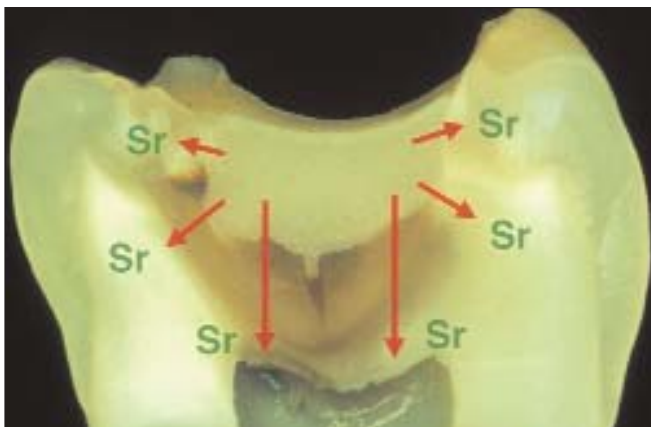


Fig. 16.14. A cross section through a molar tooth in which an A.R.T. restoration had been placed three months prior to extraction. The section was subjected to EPMA analysis and it was shown that there were strontium ions that could be identified all the way to the roof of the pulp.

time the finger can be removed and the restoration exposed to the oral environment.

This routine has been used in several countries now to stabilise cavities regardless of the position on the tooth crown. Site 1 lesions are the easiest to treat but it is reasonably successful for Site 2 and Site 3 lesions. Because glass-ionomer is relatively brittle, restoration of a Site 2 lesion with a heavy occlusal load does not last so well. Restoration of a normal contact area or building up a marginal ridge is generally not reliable so these restora-



Fig. 16.13. The completed restoration immediately following removal of the finger 'matrix'. With some of the slower setting glass-ionomers it may now be necessary to seal the restoration with a varnish or resin seal to maintain a stable water balance.

tions must be regarded as having a limited life potential.

However, Site 1 and Site 3 restorations can be expected to show acceptable longevity. Logically, if normal dental services become available, such restorations should be carefully reassessed and replaced or laminated as required. Results over recent years suggest acceptably high success rates over periods of three to five years with the potential for saving many teeth.

It is important not to confuse the situation by considering the A.R.T. technique to be available only in disadvantaged countries. The major difference between this technique and that recommended for use in general practice (see above) is that, in a normal practice, a rotary cutting instrument can be used to gain access and to develop a clean periphery to the entire cavity prior to placement of the glass-ionomer. In disadvantaged areas where there is no electricity this same stage can only be achieved by hand instruments. Also, of course, the temporary restoration can be replaced or laminated with a longer term restoration at a predictable time in general practice. This may not always be possible in situations where the A.R.T. technique is used. However, the principles are identical in both practices.

Treatment of pulpitis under intact dentine

A transient acute pulpitis may occur due to a long term insult, such as

- an advancing carious lesion
- microleakage under a restoration
- acute traumatic injury
- exposure of extensive areas of healthy dentine during crown preparation

Some form of interceptive therapy may sometimes be appropriate. The most effective therapy in these cases is to remove the insult, mainly by placement of a sound, well sealed temporary restoration. If the pulp is still alive and there is no bacterial infection the potential for resolution of the inflammation and its symptoms are excellent. Two types of material can be applied to dentine to help overcome or suppress pulpal inflammation which has been caused this way.

- Eugenol is antibacterial and has a direct effect upon pulpal inflammation and pain. It inhibits the synthesis of prostaglandins as well as reducing nerve action potentials. A brief application of eugenol to dentine, or the sustained release of eugenol into dentine from a zinc oxide/eugenol base or temporary filling, or luting material results in concentrations of eugenol in the outer layers of the pulp tissue sufficient for these effects to develop. The effect is likely to be prolonged.
- Corticosteroids are widely used in medicine for the temporary suppression of inflammation and the same effect can occur in inflamed pulp tissue. However, a lining or base containing a corticosteroid will not have any long term effect because all the steroid will be released within the first three days.

NOTE



Short term relief of pulp pain

- Eugenol in combination with zinc oxide – long-term effect
- Corticosteroids in topical application – short-term effect

Treatment of the Exposed Pulp

Pulp tissue may be exposed to the oral environment as a result of direct mechanical trauma frac-

turing both enamel and dentine or it may occur, inadvertently, during cavity preparation. Various materials may be placed directly on the exposed pulp, but whether or not the pulp will then survive depends upon

- the age of the patient – because of a relatively high vascularity the younger the patient the more likely the tooth will survive,
- the state of the pulp, including its cellularity and vascularity – previous injury or insult may have lead to the development of fibrous repair tissue thereby reducing vascularity and the ability of the tissue to recover,
- the nature and number of contaminating micro-organisms – the presence of any micro-organism is undesirable and attempts at sterilisation with antibacterial agents may cause further tissue damage. However, in the presence of high vascularity, the pulp tissue may survive minor infection with relatively benign bacteria,
- the material applied – if the pulp tissue is healthy and the area of exposed tissue is relatively small, and if contaminating bacteria are either absent or benign, calcific healing will occur adjacent to many materials. Effectiveness of the seal over the long term, with prevention of further ingress of bacteria should therefore be the principal consideration. Glass-ionomer will create such a seal under most circumstances and the pulp may heal in direct contact with it. However, the response may be better if the pulpal soft tissue only is covered with an autocure calcium hydroxide cement first and then sealed with glass-ionomer.

Pulpotomy

When a small area of uninfected pulp, such as a pulp horn, is exposed by tooth fracture in a young individual the preferred treatment is a Cvek pulpotomy, named after the dentist who first described the technique. The problem is most likely to occur in permanent upper anterior teeth of teenagers and young adults during sport, rough play or fighting and it is likely that, at this age, the pulp tissue will be free of fibrotic degenerative tissue and the blood supply will be rich.

Cvek pulpotomy

A Cvek pulpotomy should be carried out as soon as possible after the injury is sustained to minimise the penetration of bacterial contamination.⁵ Isolate the tooth with rubber dam and clean gently with air/water spray. Under local anaesthesia, a small amount of pulpal tissue and adjacent dentine is then surgically removed with a sterile high speed bur to a depth of approximately 1 mm. This will remove the surface layer of pulp which is the area most likely to be contaminated with bacteria. The wound is washed, bleeding controlled and an autocure calcium hydroxide applied directly to the pulp tissue. The remainder of the tooth defect is then restored, using conventional techniques. Use a material, or combination of materials, which will develop an effective antibacterial seal while restoring function and aesthetics. The high suc-

cess rate in young patients is probably due to a combination of factors, including the low level of bacterial contamination and good response to trauma in that age group.

Deciduous pulpotomy

If larger areas of pulp tissue are removed the response is generally not so effective. A total pulpotomy, in which the entire coronal pulp is removed, is used as an alternative to full root canal therapy in deciduous teeth, and is acceptable since failure is often slow enough to allow natural exfoliation after space retention for several years. In permanent teeth, however, the predictability of a full coronal pulpotomy and its success rate are so poor as to contraindicate the procedure, except as a temporary or economic expedient. Root canal therapy is therefore indicated.

Further Reading

1. Mertz-Fairhurst EJ. Cariostatic and ultra-conservative sealed restorations: six year results. *Quint Int* 1992; 23:827-38.
2. Massler M. Preventive endodontics: Vital pulp therapy. *Dent Clin Nth Am* 1967; 663-73.
3. Brannstrom M. *Dentine and Pulp in Restorative Dentistry*. Italy: Wolfe Medical Publications; 1981.
4. Frencken JE, Holmgren CJ. *Atraumatic Restorative Treatment (A.R.T.) for Dental Caries*. Nijmegen, The Netherlands: STI Books b.v.; 1999.
5. Cvek M. A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture. *J Endodontol* 1978; 4:232-7.

17 Periodontal Considerations in Tooth Restoration

G. J. Mount

The significant factors which require consideration are those situations which can compromise the health of the gingival tissues as the result of operative procedures undertaken for the restoration of a diseased or broken tooth. It is necessary to be able to recognize variations from normal healthy tissue prior to commencement of treatment as well as changes which may occur following restorative procedures. Consideration must also be given to actions which may cause irreversible damage during treatment.



Normal Gingival Tissue

Normal healthy gingival tissue is generally described as firm, coral pink with a stippled surface and a smooth knife edged margin around the circumference of each tooth.¹ There is expected to be a zone of gingival tissue attached firmly to the underlying alveolar bone on the buccal and lingual of each tooth varying in width from 1-5 mm and a crevice about 2 mm deep around each tooth which can be traced with a fine probe. At the base of the crevice lies the epithelial attachment representing a firm biological attachment of the gingival tissue to the root surface. The epithelial attachment normally begins immediately at the cemento-enamel junction of each tooth and is, itself, about 2 mm wide towards the apex (**Figures 17.1 and 17.2**).

Any divergence from the above description is regarded as a disease state which requires diagnosis and treatment. However, as with all pathology, there is a wide variation of acceptable levels as well as a wide variation in treatment method.

Epithelial attachment

At the time of eruption of a tooth, the epithelial attachment is located at the cemento-enamel junction but initially the crevice around the tooth above the attachment will be very deep. By the

time the tooth is fully erupted, the crevice should be only about 2 mm deep. It can remain at that depth for life, but the position of the epithelial attachment may migrate down the length of the root of the tooth for a variety of reasons, not all of which are properly understood. It may not move and patients of advanced age may show no change at all. Also, the zone of attached gingival tissue at the buccal and lingual of each tooth can be reduced to 1 mm or less without any indication of pathology and, in a few patients, can be absent entirely without any untoward result.

Abnormal situations

Reasons for apparent aberrations are not clearly understood and it is suggested that the only indication of pathology or alteration from normal, which is of significance and requiring treatment, is the presence of inflammation. In the presence of normal healthy tissue its position relative to the cemento-enamel junction is of no concern even in the presence of apparently bizarre situations (**Figures 17.3 and 17.4**). There will occasionally be situations where migration of the epithelial attachment or loss of attached gingiva is accompanied by severe loss of supporting bone to the extent that the life and stability of a tooth is compromised and surgical intervention is warranted. However, this will be in the presence of a generalised periodontal condition which is beyond the scope of this treatise.



Fig. 17.1. The gingival tissues of a young man are in near perfect condition. Note the gentle pink colour and light stippling effect with a knife edge gingival margin.



Fig. 17.2. The gingival tissues of a female at the age of 80 years. These are the sole remaining lower teeth and the patient wears a lower partial denture. In spite of this the gingival margin remains at the cemento-enamel junction in perfect health.

Problems which Compromise Periodontal Tissue

Gingivitis

Gingivitis can be identified as a change in the colour, texture and contour of the gingival tissue around part or all of the circumference of a tooth.² The earliest signs will be a subtle change in colour and a tendency to bleed from within the gingival crevice following gentle probing. These changes can progress to hypertrophy of the tissue

with a major change in colour to a dull red and a tendency to be easily damaged by tooth brushing or even eating. The depth of the gingival crevice may increase through hypertrophy of the gingival tissue in an occlusal direction to a depth of 3-4 mm but the position of the epithelial attachment may remain at the cemento-enamel junction, particularly in the early stages of gingivitis (**Figures 17.5 and 17.6**).

The role of plaque

The cause of gingivitis is bacterial activity. Bacteria attach to the surface of a tooth or restoration and



Fig. 17.3. The gingival margin has retreated some distance down the root of the tooth but the patient is nearly 80 years of age and in good health. It is possible that she suffered from some degree of periodontal problems earlier in life but the history is unknown.



Fig. 17.4. A patient in her mid-50s suffered from quite severe periodontal problems successfully treated about 10 years ago. Her periodontium is now in excellent condition despite the disto-buccal root being almost entirely exposed. There is no inflammation so the periodontium is regarded as healthy.



Fig. 17.5. A young patient suffering from a marginal gingivitis. The typical picture shows inflammation along the gingival margin with plaque retained along the full length.



Fig. 17.6. The marginal gingiva looks to be in reasonable condition but gentle probing shows that there is inflammation and ulceration within the gingival crevice. This is often the first sign of gingival problems and requires intensive treatment to arrest migration of the epithelial attachment.

become organised to the extent that they form a soft mass referred to as bacterial plaque. If the plaque is allowed to remain in the vicinity of the gingival tissue it will inevitably result in an inflammatory reaction in response to the presence of toxins from the bacteria. The pathogenic potential of plaque can vary from one individual to another and from one area of the mouth to another. It is accepted that inflammation is an immunological defence mechanism and investigations are still being undertaken to determine if the body responds to a greater degree to one or another bacterial group.

Treatment

Treatment of gingivitis is simply the removal of plaque and/or calculus. Achievement of this aim is not always easy because, over time, the plaque matures and takes up additional calcium and phosphate ions and turns into calculus, a hard accretion on the surface of the tooth which is difficult to remove. However, if plaque is to be removed effectively on a daily basis, the teeth must be maintained in a cleansible condition. Any situation in the mouth which compromises the ability of the patient to remove plaque must be



Fig. 17.7. The typical position for accumulation of plaque is around the lingual of the lower anterior teeth as shown here. This is in part because of the difficulty of cleaning efficiently and in part because the sub-lingual salivary ducts lie just lingual to these teeth.



Fig. 17.8. The same patient as shown in **Figure 17.7** one week after undergoing a complete scale and clean from a hygienist. Note the improved condition of the gingival tissues.



Fig. 17.9. An older patient showing an accumulation of debris over a long period of time. There is considerable marginal gingivitis beneath the plaque which extends well up into the gingival crevice.



Fig. 17.10. The same patient, a month after commencement of treatment, following a series of appointments to undertake deep scaling and cleaning for total removal of plaque and calculus showing considerable improvement. The gingival contour is expected to continue to improve.

avoided or eliminated. This includes maintenance of the correct contour of the crowns and the roots of the teeth, the avoidance of prosthetic replacements which are not cleansible and the elimination of areas of crowding of teeth which compromise cleaning techniques (**Figures 17.7-10**).

Periodontitis

Periodontitis is the usual sequel to untreated gingivitis. It is essentially an extension of the inflammatory process into the connective tissue and the bone surrounding the teeth leading to the progressive destruction of those tissues. It has been suggested that periodontal disease progresses in bursts of activity ranging in length from a few days to a few months.

Migration of epithelial attachment

The first stage involves the loss of the epithelial attachment at the cemento-enamel junction. The crevice surrounding the tooth will become deeper and the patient's ability to remove plaque from the depths of the crevice on a daily basis will be reduced. There is normally only about 2 mm from the base of the epithelial attachment to the crest of the alveolar bone surrounding the root so the bone itself is rapidly involved in the inflammatory process. It will not withstand the presence of bacterial activity and will resorb quite rapidly

developing a pocket around the root within the alveolar bone. By this time the marginal gingival tissue may have resumed a relatively normal appearance with very little obvious inflammation while the destructive process may continue into the deeper structures. Ultimately the loss of bone may progress to the point where the root has lost mechanical support and the tooth becomes mobile and has to be extracted (**Figures 17.11 and 17.12**).

Treatment

Treatment of periodontal disease is essentially the same as treatment of gingivitis in that removal of the cause, the plaque and calculus, is of primary importance. However, resolution of the damage arising from the disease is often very complex. It has proven impossible up to date to redevelop a normal epithelial attachment and very difficult to regain bone height to support the roots of teeth. Therefore it is accepted that periodontal disease can be terminal for remaining teeth unless vigorous treatment procedures are undertaken before bone loss has advanced to the stage that mechanical support for the teeth has been lost.

Removal of the plaque and calculus will allow rapid resolution of the inflammatory response. Once the soft tissue has regained health, at least superficially, steps can be undertaken to ensure that the root surface is entirely clean of calculus



Fig. 17.11. This patient presented following treatment for periodontal disease some years ago. Oral hygiene is now efficient but there remains a problem following migration of the epithelial attachment at the mesial of the upper lateral incisor. Clinically this is not obvious until careful probing of the pockets.



Fig. 17.12. A radiograph of the upper lateral for the same patient as shown in **Figure 17.11**. Note the deep pocket at the mesial of the tooth which is now a major problem with a need for surgical interference if it is to recover.

and that the width of attached gingiva has been reinstated to a reasonable dimension.

Long epithelial attachment

The epithelial attachment will not regenerate in its original form without surgical intervention because the epithelial cells will propagate down the inside of a wound very rapidly and will prevent the development of a normal tissue attachment on the cementum of the root surface. The result, often, is a long epithelial attachment which, whilst not entirely physiological, can remain healthy and free of further inflammation in the presence of careful plaque control routines. Ideally the bone should be regenerated and the epithelial attachment restored to its original dimension but surgical techniques are required to achieve this end.

Effect of Restorative Dentistry on Gingival Tissue

In view of the potential for the ultimate loss of the dentition through periodontal disease it is important to consider all the implications of restorative dentistry on the periodontal tissues.^{3,4} Restorative materials per se will not have a deleterious effect on the soft tissues (**Figure 17.13**). There are three different aspects to this problem

- **Prior assessment and treatment**

It is important firstly, that the condition of the periodontal tissues be assessed prior to restoration of the teeth and that all necessary measures be undertaken to restore normal health.

- **Minimal trauma during treatment**

At the time of carrying out restorative procedures care must be taken to minimise trauma to the gingival tissues.⁵ If the tissue is healthy to begin with it can be expected to heal rapidly and uneventfully. However, it is possible to traumatise the tissues to such a degree during restorative procedures that normal healing cannot take place and some degree of permanent alteration to gingival contour will result.

- **Restore original contour for plaque control**

It is imperative to maintain the teeth in such a condition and contour that plaque control can be achieved readily on a daily basis.⁶ The natural contour of the coronal anatomy of a tooth is generally such that hygiene procedures are relatively effective. However, over contour in any dimension will impose problems in plaque removal. Lack of contour is less of a problem providing it does not lead to food impaction between teeth or under prosthetic appliances. It is generally accepted that it is impossible to achieve a perfectly smooth union between any restorative material and the tooth being restored so, wherever possible, it is desirable to maintain the restoration margin outside the gingival crevice. A further reason for keeping margins supragingival is that some of the common restorative materials, such as composite resin, have a tendency to encourage plaque formation.

Treatment procedures

Improvement of gingival tissue

Restoration of the gingival tissues to a normal healthy state prior to restorative procedures is essential, particularly if the cervical margin of a restoration has to be placed subgingivally. In



Fig. 17.13. It has been argued that some restorative materials are likely to irritate gingival tissues. This is not at all likely providing the margin of the restoration does not accumulate plaque. In this patient there are restorations in amalgam, gold, porcelain and gold foil none of which show any adverse effect around the margin.

many cases it will be adequate to carry out a thorough prophylaxis with removal of plaque and calculus and the topical application of a fluoride gel. The fluoride will remineralise the enamel and dentine and will also decrease the wettability of the surface of the tooth and therefore reduce future plaque formation. There is rarely any justification for the surgical removal of hypertrophied tissue at this point because elimination of the inflammation will lead to resolution of the soft tissue to the extent that surgery may no longer be required. If surgical modification can be justified the tissue will be in good health to begin with and healing will be uneventful.

Improvement of existing plastic restorations

Existing restorations which are over contoured should be recontoured as far as possible at this time even if they are to be removed subsequently.^{7,8} In particular old corroded amalgam restorations and overcontoured composite resins should be contoured and polished so that the patient can use dental floss or interproximal brushes to improve the quality of the gingival tissues.^{9,10} Recontouring can be achieved with very fine tapered diamond stones at intermediate high speed under air/water spray followed by reciprocating diamond blades in a special handpiece to produce a reasonable polish on the surface. Local

anaesthesia is not required even though there will often be a copious blood flow because of the gingival inflammation. Removal of the entire restoration at this point is not recommended because it is very difficult to replace in the presence of gingival inflammation and the final anatomy will not be determined until the inflammation is resolved. Temporary intracoronary restorations are not very successful because of the problems of achieving and maintaining proper interproximal contour and most of the temporary restorative materials are relatively fragile (Figures 17.14-20).

Improvement of existing rigid restorations

Recontouring of ceramic crowns and gold restorations is more difficult but should, nevertheless, be attempted.^{11,12} Diamond burs are indicated on porcelain crowns but multiblade tungsten carbide burs remove gold more efficiently. There is a tendency to gouge into the restoration and it is difficult to achieve a perfect result. Therefore if the original restoration is to be retained it must be done with great care.

If it is intended to eventually remake the crown the construction of a high quality temporary restoration may be justified whilst treatment of the periodontal condition continues. However the gingival tissue will respond rapidly to the initial recontouring and within a week it may be possible



Fig. 17.14. There are acrylic crowns and resin restorations in these three lower anterior teeth, all with overhangs and rough margins. If these are to be crowned again it is important to restore the gingival health prior to commencement of treatment.



Fig. 17.15. The same patient as in Figure 17.14 two weeks after careful recontouring of overhanging margins and deep scaling and cleaning. Note that the gingival contour is greatly improved and it will soon be possible to proceed with reconstruction.



Fig. 17.16. There is an adverse effect on the gingival tissues in the interproximal areas between these bicuspid. Both are restored with amalgam but it is the overhanging margins that retain plaque and cause the problem rather than the amalgam *per se*.

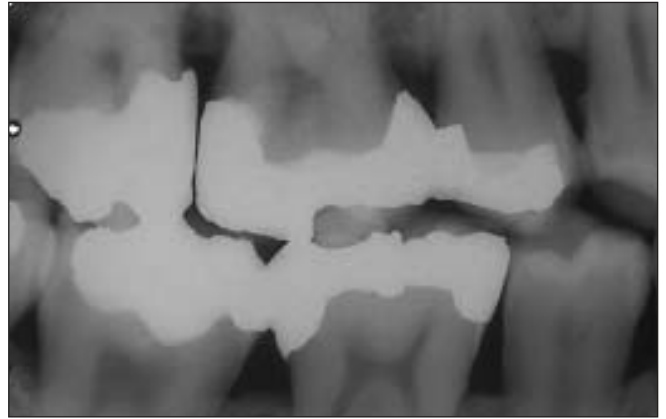


Fig. 17.17. A quadrant of amalgam restorations in the upper left are about to be replaced with gold inlays. Note the overhanging margins particularly at the mesial of the first molar.



Fig. 17.18. The same patient as shown in **Figure 17.17**. The restorations have been removed with some care under rubber dam and the dam has just been removed. Note the level of inflammation in the interproximal tissues.



Fig. 17.19. Temporary restorations were constructed for the same patient and contoured well, particularly interproximally. Two weeks later the temporaries were removed and the gingival tissue appears to have recovered entirely.



Fig. 17.20. A bitewing radiograph taken two years later for the same patient as shown in **Figure 17.17**. Note the gingival contour of the restorations but also note that there has been minor loss at the crest of alveolar bone between the first molar and the second bicuspid.

to determine the final contour of the gingival margin and therefore construct a better temporary crown.

Maintenance of occlusion

There are two factors in relation to the occlusion which are of significance to the periodontal tissues and these must be taken into account when restoring posterior teeth.

Functionally opening contact

Impaction of food debris between two teeth can have a devastating effect on the interproximal periodontal tissues. The cause may, on occasion, be poor contour in relation to the marginal ridge



Fig. 17.21. The patient complains of food impaction between the lower first and second molar teeth. It is apparent that the cause is a functionally opening contact in as much as, during closure, the lingual cusp of the upper molar will engage the forward incline of the occlusal surface of the lower molar and tend to force it distally thus opening the contact area and allowing impaction of food. The presence of the overhanging margins on both amalgam restorations were not the primary cause but did exacerbate the situation.

but more commonly it arises because of deflective inclines on the occlusal surface of adjacent or opposing teeth. For example, when the patient closes towards the centric relation position during mastication of a food bolus, a distal facing incline on a lower molar may engage a mesial facing incline on the opposing molar. The result may be that the upper molar moves distally and opens the contact between it and the adjacent tooth allowing ingress of fibrous food debris. On opening the mouth and relieving the load, the tooth will move mesially again and entrap the food debris. This situation occurs most commonly in relation to the terminal tooth on the arch but it is possible for it to arise between other contacting areas (**Figure 17.21 and 17.22**).

Therefore, in the presence of chronic food impaction, it is necessary to examine the relationship of the opposing teeth to determine the presence or absence of such contacts particularly as the teeth approach each other in the centric relation position. Occasionally there may be a dominant cusp occluding directly into the contact area of two opposing teeth - a situation which has been described in the past as a plunger cusp. However, mostly the problem arises from cuspal inclines not directly related to the contact area at all.

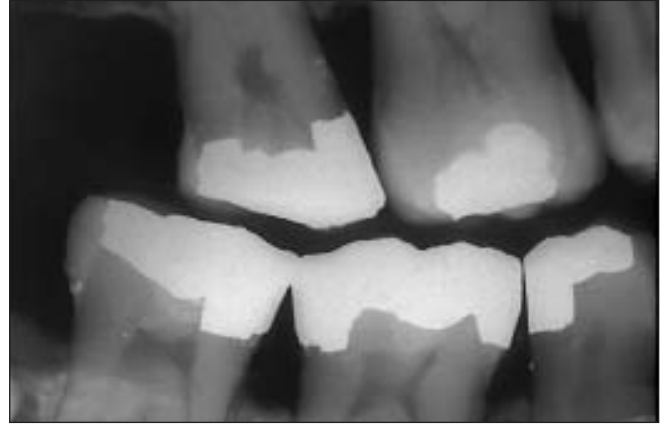


Fig. 17.22. The same patient as shown in **Figure 17.21** immediately after redesign of the two restorations in the upper and lower second molars. Note that the occlusal contour of the lower molar has been levelled off and the prominent lingual cusp of the upper molar has been adjusted to meet evenly with the lower molar. The overhangs are gone.

Modification to the appropriate cusp inclines will overcome the problem.

NOTE



Functionally opening contact

- This term is a more accurate description of a food impaction problem than the previous term, plunger cusp

Maintenance of posterior support

As discussed in **Chapter 18**, a lack of posterior support can lead to undue load on remaining teeth and, if there is already periodontal disease present, it may be severely exacerbated. Maintenance or regeneration of the minimum eight units, in conjunction with treatment of the periodontium, will stabilise the situation.

Placement of gingival margin relative to gingival tissue

There are a number of factors which will need to be taken into account before deciding the final placement of the gingival margin for any restoration. Because of the potential for plaque accumulation along the interface between restoration and



Fig. 17.23. The lower right quadrant is to be reconstructed using gold and porcelain crowns. All existing amalgam restorations have been replaced first to ensure proper contour and a healthy gingival tissue prior to commencement of the rehabilitation.

tooth structure it is desirable to keep the margin out of the gingival crevice. However, the following factors will influence the decision.

Strength of remaining tooth structure

Following caries debridement there may be weakened undermined dentine or enamel along the gingival margin. As this will not be subjected to occlusal load it need not be totally removed but fine acute angles of dentine should be flattened off and rough fragile enamel should be lightly planed with a gingival margin trimmer. It may then be possible to avoid entering the gingival crevice.

Retention of the restoration

The strength of retention of a restoration is dependent upon the length of the vertical walls of the preparation. This is of less significance when placing a plastic restorative material in an intra-coronal cavity because grooves and ditches can supplement retention. But it is very important for both inlays and extracoronal restorations. The situation may often arise where it is necessary to enter the gingival crevice in order to develop sufficient length to ensure retention. Very occasionally gingival surgery with recontouring of the alveolar bone may be indicated but this is undesirable because of the difficulty of redeveloping a normal epithelial attachment.



Fig. 17.24. The quadrant has been prepared for the patient shown in **Figure 17.23** and this shows the hydrocolloid impression at the time it was taken. Note the excellent marginal reproduction as a result of upgrading the gingival tissue first.

SUMMARY



Placement of the gingival margin is controlled by

- strength of remaining tooth structure
- retention for the restoration
- ultimate aesthetic requirements

Aesthetics

Particularly when designing extracoronal restorations on the upper arch it will be necessary to compromise the position of the gingival margin to develop satisfactory aesthetics.^{13,14} Providing the gingival tissue is healthy, the gingival margin of the preparation can be placed up to 0.5 mm into the gingival crevice without provoking an adverse tissue response. Care must be taken, particularly with tissue retraction techniques for impression taking, to minimise tissue damage at the time of operation and skilled technical work is required to develop a smooth union between the restoration and the tooth, with the ideal emergence profile, to minimise plaque retention.

Procedures during restorative work

It is desirable to return the gingival tissue to full health before embarking on restorative procedures so that the position of the gingival contour will be predictable. Following any damage to the gingivae there will be inflammation in the periodontal tissue even if it was perfectly healthy to



Fig. 17.25. The Site 3, Size 1 lesions at the gingival of the two upper bicuspid are to be restored using a resin modified glass-ionomer. To ensure a lack of gingival bleeding during placement the gingival tissue has been cauterised using a brief application of trichloroacetic acid. Note the immediate blanching of the tissue leading to a completely clean field.



Fig. 17.26. The same quadrant as shown in **Figure 17.25** photographed six months after placement. Note that the gingival tissue shows no ill effects at all and has completely healed.

begin with. The heat, redness and swelling which will arise, may alter the gingival contour and subsequent resolution of the inflammation will modify the shape again making it impossible to predict the ultimate contours (**Figures 17.23 and 17.24**).

The following methods can be utilised to maintain gingival health.

Rubber dam and wedges

The most effective method of prevention is proper protection. The routine placement of rubber dam is strongly recommended even though dam, by itself, does not guarantee protection (**Chapter 10**). The dam will displace the soft tissue to some degree but it is still possible to tear the dam or have an instrument pass through it.

When preparing a gingival margin a further precaution is to place a wooden wedge between the teeth thus displacing and protecting the dam as well. A wedge will serve multiple purposes in as much as it will also move teeth mesially and distally sufficiently to enhance access and improve the strength of the contact between the teeth following restoration.

Retraction cord

An alternate method of protecting gingival tissue is to displace the gingival crest laterally by packing a short length of an astringent gingival retraction cord into the gingival crevice. When placed

with care the tissue will be displaced out of the way of any rotary cutting instruments.

Localised removal of excess gingival tissue

Low grade chronic gingival inflammation, sustained over a long period, may resolve into relatively healthy but over contoured fibrous tissue. If it is allowed to remain it may compromise the anatomy of a restoration and also it is easily damaged during operative procedures and will haemorrhage freely. There are several methods of removing such tissue or, at least, controlling the haemorrhage.

A brief application of trichloroacetic acid will arrest the bleeding very effectively and resolve some of the tissue overgrowth. Electrosurgery or laser therapy can be used to remove larger areas of excess tissue (**Figure 17.25 and 17.26**) (**Chapter 9**).

Additional scaling during restorative procedures

Despite following the recommended procedure of thorough scaling and cleaning at the time of recontouring old restorations prior to embarking on final restoration there will often be calculus still present on the interproximal surfaces immediately below the gingival margins of old restorations. A hand instrument or ultrasonic scaler should always be passed over the root face below the gingival margin before placement of the matrix and proceeding with the placement of a



Fig. 17.27. The amalgam in the mesial of this upper molar is overcontoured and subsequently the gingival tissue beneath is chronically inflamed. There is no actual overhang but the mesial surface of the restoration is seriously over contoured.

new restoration. Also the opportunity should always be accepted for polishing the proximal surface of an adjacent restoration which has become accessible during restorative procedures.

SUMMARY



To avoid damage during restorative work

- use rubber dam
- place retraction cord
- use localised cautery or gingival surgery
- use additional scaling
- exercise care in placing matrices

Placement of matrices

Assuming the gingival tissue is in good health, very little damage will be sustained through the placement of a matrix. However, care must be taken to ensure a firm fit with good support for the band using wooden wedges as well as green stick compound as required. Trim the matrix to contour and polish roughened edges. Achieve a degree of separation between the teeth with a wooden wedge so that the ultimate contact with adjacent teeth will be firm and positive.

Correct approximal anatomy

In view of the imperative need to control plaque development and retention in the oral cavity there must be considerable emphasis placed on maintaining the correct anatomy of each tooth. As



Fig. 17.28. The same restoration as in **Figure 17.27** following recontouring using a fine tapered diamond followed by polishing strips to remove the excess amalgam and expose the gingival tissue to normal hygiene. The tissue will recover promptly.

indicated previously over contour of any part of the tooth anatomy will enhance plaque retention particularly in the vicinity of the gingival margin (**Figure 17.27 and 17.28**).

The term emergence profile refers to the contours of the tooth at the gingival margin as it appears out of the gingival crevice. If this profile is overbuilt in any way plaque removal will be more difficult. An overhanging interproximal margin on a restoration of any material is probably the most dangerous alteration to the emergence profile, in as much as it is almost impossible to clean under it. Even a smoothly curved and polished overcontour in a plaque resistant material such as porcelain is undesirable because of the increased problems of plaque removal.

Contact area

The contour of the proximal surface of the crown of a tooth, particularly in the contact area, is also important and is difficult to redevelop when using the direct plastic restorative materials. It is desirable that the surface be smooth and polished but it is also essential that there be a positive contact area with the adjacent tooth. The following factors are important:

- the contact area between posterior teeth should be approximately 1-2 mm below the greatest height of the marginal ridge
- it should be no more than 1-2 mm in height occluso-gingivally and extend bucco-lingual-

ly approximately 50% of the width of the adjacent teeth

- in the upper arch it is generally placed slightly to the buccal of the mesio-distal midline
- in the lower arch it is in the midline
- in the young patient it is narrower bucco-lingually
- as the patient ages it will broaden
- the interproximal space should be as wide open as possible commensurate with the presence of a sound contact area and a smooth vertical emergence profile to allow optimal plaque removal

BE AWARE



Correct anatomy is essential on all surfaces

- emergence profile
- contacting surfaces
- occlusal anatomy

Correct occlusal anatomy

The occlusal anatomy is of equal importance particularly in the area of the marginal ridge (**Figure 17.29**).

- The marginal ridge should rise above the contact area and be smoothly rounded to allow ease of access of dental floss.



Fig. 17.29. A well contoured quadrant of amalgam restorations showing attention to occlusal anatomy with the provision of proper marginal ridges and spillways to relieve the load from the occlusal surface. Note the health of the subjacent soft tissues.

- There are generally shallow grooves running out to the buccal and lingual of the marginal ridge to guide the food bolus away from the contact area and off the height of the cuspal inclines.
- In youth the cuspal inclines tend to be steep but, as the patient ages, the occlusal anatomy assumes a flatter profile.
- Deep intercuspation between opposing arches is undesirable because of the lateral stress which can be imposed on cusp inclines, particularly nonworking cusps.
- It is important to adjust both opposing occlusal surfaces as required during restorative procedures to minimise the intercuspation because deep intercuspation may lead to the development of both balancing and working side interferences during lateral excursions.

Modification to gingival architecture

Surgical modification

The ultimate aesthetic result of restorative dentistry is of importance to both the patient and the operator particularly in relation to the anterior teeth. A lack of balance and symmetry in the gingival margins may be of significance. Surgical techniques have been developed which include recontouring both the bone and the soft tissue. These must be carried out prior to designing the gingival margins of the ultimate restorations and temporary restorations of very high quality should be constructed to ensure maintenance of gingival health during the healing process.

Orthodontic modification

There will be occasions where orthodontic movement of a tooth or teeth is desirable prior to restoration. However, the gingival tissue is likely to remain in the same position, relative to the cervical margin of the crown of the tooth, following movement and surgical intervention may be required to adjust irregularities.

Orthodontic eruption of the remainder of a root following trauma or extensive sub-gingival caries will always be accompanied by migration of the gingival margin and epithelial attachment as well. The gingival tissue will need to be repositioned

and often some degree of alveolar bone recontouring will be involved. The restoration of the crown is further complicated in this technique by the fact that the dimensions of the root face will be notably smaller than the desirable aesthetic shape of the crown and a compromise is generally necessary, particularly in relation to the emergence profile.

In view of the difficulty of re-establishing a normal epithelial attachment, it is suggested that the advantages of such surgery must be weighed carefully against the likely modification to gingival anatomy, and surgery should be undertaken only when essential. The techniques come into the realm of periodontal surgery and will not be discussed further in this treatise.

Further Reading

1. Ramfjord SP. Periodontal aspects of restorative dentistry. *J Oral Rehab* 1974; 1:107-26.
2. Burch JG. Periodontal considerations in operative dentistry. *J Periodontol* 1975; 34:156-63.
3. Block PL. Restorative margins and periodontal health; a new look at an old perspective. *J Prosthet Dent* 1987; 57:683-9.
4. Leon AR. The periodontium and restorative procedures; a critical review. *J Oral Rehab* 1977; 4:105-17.
5. Mount GJ. Crowns and the gingival tissue. *Aust Dent J* 1970; 4:253-258.
6. Grasso JE, Nalbandian J, Sandford C, Balit H. Effect of restoration quality on periodontal health. *J Prosthet Dent* 1985; 53:14-19.
7. Brunsvold MA, Lane JJ. The prevalence of overhanging dental restorations and their relationship to periodontal disease. *J Clin Periodontol* 1990; 17:67-72.
8. Jameson LM, Malone WFP. Crown contours and the gingival response. *J Prosthet Dent* 1982; 47:620.
9. Leon AR. Amalgam restorations and periodontal disease. *Br Dent J* 1976; 140:377-82.
10. Van Dijken JWV, Sjostrom S, Wing K. Development of gingivitis around different types of composite resin. *J Clin Periodontol* 1987; 14:257-60.
11. Youdelis RA, Weaver JD, Sapkos S. Facial and lingual contours of artificial complete crown restorations and their effects on the periodontium. *J Prosthet Dent* 1973; 29:61-66.
12. Behrend DB. Crown margins and gingival health. *Annals of the Royal Australasian College of Dental Surgeons*. 1984; 8: 138-45.
13. Eismann HF, Radke RA, Noble WH. Physiologic design criteria for fixed dental restorations. *Dent Clin N Am* 1971; 15: 543-68.
14. Newcomb GM. The relationship between the location of sub-gingival crown margins and gingival inflammation. *J Periodontol* 1974; 45:151-54.

18 Occlusion as it Relates to Restoration of Individual Teeth

G. J. Mount

When the anatomy of the occlusal surface of a posterior tooth or the incisal or lingual surface of an anterior tooth is to be modified in any way as a result of restoration of that tooth, or an opponent, such modification must be carried out with an understanding of the potential for change in the occlusal relationship between the arches.

Any change must maintain the overall pattern of occlusal harmony.



Basic Principles of Occlusion

A knowledge of occlusion and the articulation of opposing dental arches is of great practical value: hardly a day passes in dental practice without the dentist having to articulate teeth, coincidentally coordinating the jaw muscles and joint components.” (Posselt)¹

Strictly speaking occlusion is a static term describing the relationship of opposing teeth in contact. However it has evolved into common terminology to mean also the movement of opposing teeth during the function of mastication and the empty movements of clenching and grinding.

The basic parameters of articulation have been set out clearly in Hanau’s ‘Quint’² of which the following are the guiding components:

1. angle of the condyle paths
2. angle of incisal guidance
3. occlusal plane
4. occlusal curve
5. angle of the cuspal inclines

There are several factors which can be varied or modified as a result of dental disease or during reconstruction of the dental arches following loss or breakdown of one or more teeth. The angle of the condylar paths will remain constant, although it is possible for them to change slowly over time. The inclination of the incisal path can vary as a result of loss or restoration of posterior teeth, abrasion, attrition or restoration of incisal edges of anterior teeth or a combination of these factors. The curvature of the occlusal plane is difficult to change through the simple restoration of individual teeth but random loss of posterior teeth with subsequent over-eruption of unopposed teeth or depression of overloaded teeth may well lead to change. The inclination of the cuspal inclines and the curvature of the occlusal plane can be changed very readily as a result of repeated alteration to the occlusal surfaces of posterior teeth. This means that the anatomy of every restoration should be considered with great care during reconstruction because of the potential for alteration with untoward results.

The intercuspal relationship of normal teeth in the adult

Whilst there is an infinite variation possible in the actual relationship of opposing teeth it is accepted that certain descriptive positions can be referred to as normal. The terms centric occlusion and intercuspal position are interchangeable and refer to the position at which the teeth are in normal contact with each other with the cusps at maximal intercuspation and, more importantly, the musculature of the temporo-mandibular joints is unstressed and comfortable. Normally all teeth will be in contact although, in some situations, not necessarily regarded as malocclusions, the anterior teeth may be apart, such as in the Angle Class II Division 1 situation. If a tooth becomes unopposed through loss of an opponent it may over erupt or drift, particularly if it is periodontally involved, and thereby modify the centric occlusion position. Also, if the occlusal anatomy of a tooth is altered by deepening the central fossa, or lengthening the cusps, or increasing the angle of the cuspal inclines the tooth may drift or tilt to improve the intercuspal relationship with its opponent, and further modify centric occlusion. Such modifications may lead to an alteration in the tooth to tooth relationship during excursive movements. All mandibular movements are discussed as commencing from the centric occlusion position and will consist of one or a combination of the following.

BE AWARE



Any of the following can lead to alteration to occlusion

- failure to replace a lost tooth
- over eruption of a tooth
- over carving of occlusal surface
- deepened occlusal anatomy
- increase cuspal inclines

The envelope of movement

Centric relation position

The centric relation position or retruded contact position is regarded as the most retruded position that the mandible can achieve with the condyles

in their most upward and forward position within the fossae. In this position the teeth should be in contact amongst the posteriors and this contact should be even on both sides. The mandible should be able to move forwards and upwards to the intercuspal position without deviation to either side. Interferences to this position, or deviation in the movement forward to the intercuspal position, may lead to parafunction because the body may be attempting to eliminate this irregularity by grinding it away. This may lead to muscle stress and excessive attrition on individual teeth or groups of teeth. However, the areas of attrition will not always be clearly related to the actual interference or initial contact and careful evaluation may be necessary.

Lateral excursions

From the centric occlusion position the mandible should be able to make lateral movements for several millimetres to either side without undue stress or interference from the teeth. For the first millimetre or so in an excursive movement, the teeth may remain in contact. Thereafter guidance should be derived from the anterior teeth only, particularly the canines, with no contact between the posteriors at all. The lower canine should move smoothly down the lingual incline of the upper canine and the posterior teeth should

become totally discluded on both sides. This is known as cuspid guidance and under these circumstances very little stress can be exerted on the teeth or the musculature. It is also acceptable to have group function wherein the buccal cusps of one or more posterior teeth remain in contact during the lateral excursion, providing always there is no contact on the opposite side.

If there is a contact maintained during excursion on the opposite nonworking, or balancing side, considerable muscular pressure can be applied because of the relative triangulation between the teeth on both sides and the temporomandibular joint. In other words there is expected to be complete disarticulation from the nonworking side immediately after the initiation of a lateral movement.

Protrusive movement

Similarly during a protrusive movement the posteriors should be discluded, thus minimising the amount of muscular pressure which can be developed. The lower anterior teeth should move smoothly down the lingual surface of the upper anteriors with an even contact on both sides of the midline and the posterior teeth should immediately disarticulate. It really does not matter which of the anterior teeth are in contact providing there is contact on both sides of the midline.

The significance of correct guidance

The factors outlined above in Hanau's Quint will normally be in balance when the occlusion first develops. As tooth wear occurs throughout life this balance should be maintained. However, the introduction of iatrogenic modifications to the relationship will be likely to introduce undesirable stresses and pressures which may well lead to excess wear and muscle stress. In the presence of anterior and cuspid guidance the musculature cannot exert undue pressure to either the teeth or the joint. There can be no fulcrum around which the mandible can rotate distally and the lever arm is so long as to be inefficient in the application of pressure on to the joint.

Proprioception appears to be designed around cuspid guidance of the mandible as it approaches

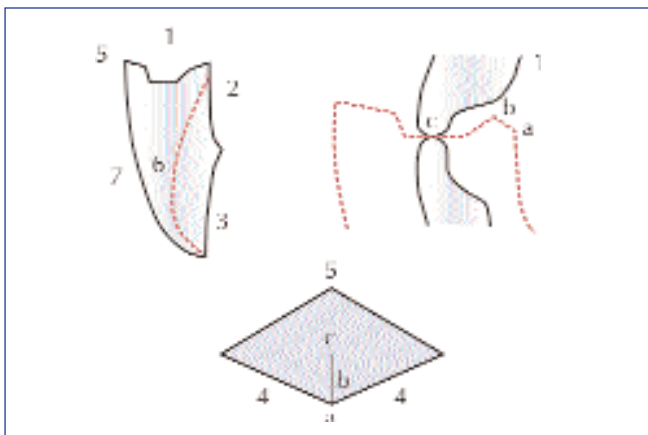


Fig. 18.1. Posselt's diagram of excursive movements: 1. Movement of lower incisors: a) terminal hinge b) intercuspal position c) edge-to-edge position. **2.** Opening from hinge. **3.** Further posterior opening. **4.** Lateral excursions. **5.** Extreme protrusion. **6.** Habitual closing arc. **7.** Extreme opening arc.

the maxilla so cuspid guidance is regarded as normal. However, in the presence of interferences in the envelope of movement, or the development of triangulation between both sides of the mandible and the joint, there is likely to be a subconscious attempt to remove the source of the interference by parafunctional actions, such as clenching and grinding, and this may lead to the development of bizarre wear patterns which are not always immediately obvious as the actual result of the problem.

Factors which may modify anterior or lateral guidance

Changes to cuspal inclines

There are three factors which commonly lead to alteration to the angle of cusp inclines with the potential for complicating the occlusal relationship between the arches. Any change which makes the cuspal inclines steeper will result in deeper intercuspation with opposing teeth sufficient to lead to interferences in lateral excursions, particularly through the development of balancing side contacts.

- When placing a restoration in amalgam or composite resin there is a temptation to deepen cuspal inclines in an attempt to improve the aesthetics of the occlusal surface of a posterior tooth.
- Both of these restorative materials wear away more rapidly than natural tooth structure.

- Routine repolishing of the occlusal surfaces of these restorative materials will bring further change to occlusal anatomy (**Figures 18.2-4**).

Changes to anteriors

Modifications to the incisal edges of anterior teeth can alter anterior guidance and should be undertaken with care:

- Adjustment of the incisal edge of a lower anterior tooth to allow greater thickness in a crown on the lingual of an upper anterior tooth can alter the curvature of the occlusal plane or change the inclination of the incisal path, and may lead to protrusive interferences.
- Aesthetic modifications to the incisal edges of either upper or lower anterior teeth may have a similar effect and should be carried out with care and attention to possible sequelae.

Uncontrolled changes are undesirable because the sequelae of undue change may be complex and can include such things as wear facets in unusual places, split cusps, split roots, lost restorations, broken prostheses, migration of periodontally involved teeth and temporo-mandibular joint pain dysfunction syndrome.

BE AWARE



Minor changes to occlusion can have severe consequences.



Figs. 18.2-4. These cut aways show the relationship of the posterior teeth on both working side and balancing side with contact in both movements because of deep intercuspation developed through over carving amalgam restorations. The result is the potential for clenching and grinding as well as the development of heavy load on remaining cusps.



Fig. 18.5. Overcarving or excessive wear has led to deep intercuspal contact into the occlusal of this lower molar. Removal of the amalgam showed an extensive split at the base of the lingual cusps.

Every restoration should be placed with care to reinstate normal occlusion and avoid undue change. Any sign of unusual wear, or failure of tooth structure or restoration, without obvious cause should be carefully explored to determine if changes to the occlusion could be a contributing factor.

Factors leading to occlusal change

Where there is a relatively intact dentition there are a number of factors which may lead to excessive wear and subsequent changes to occlusion.

Orthodontic movement

Orthodontic movement which does not reinstate a correct cuspid rise and anterior guidance may lead to parafunction and excessive wear. Correction of orthodontic anomalies should be directed, not only to aesthetics, but also to function because, whether the patient is conscious of the problem or not, they may subconsciously attempt to arrange their own correction by parafunction.

Wear on occlusal surfaces and incisal edges

If a restorative material occupies a reasonably large area of the occlusal surface it should have a wear factor which closely resembles that of the opposing and adjacent tooth structure. As a restoration wears away, deeper intercuspal contact



Fig. 18.6. This patient shows a lack of posterior support so that over a period of about 30 years the porcelain crown on the upper central incisor has created considerable wear on the opposing teeth. There is also wear on the remaining natural anteriors and the upper lateral incisor has recently been decoronated.

with the opposing tooth may develop with the potential for a locked occlusion, balancing side contacts in lateral excursions and, possibly, split cusps. Excessive wear cannot be diagnosed simply by examination of the relationship of opposing teeth to see that they are still in contact, because continual eruption will generally maintain a contact. Serial measurement of study models of both arches taken at intervals is essential to determine the speed and extent of wear and to decide whether it is within the restoration or on the opposing tooth or restoration.

Of the current restorative materials gold has a wear factor closest to enamel and some of the ceramics are the most abrasive. Amalgam wears a little faster than enamel and some of the composite resins have posed problems in the past. The modern highly filled hybrid composite resin has an acceptable wear factor which compares favourably with amalgam (**Figures 18.5 and 18.6**).

BE AWARE



Wear on occlusal surfaces is difficult to detect but can have serious consequences.

Failure to replace a posterior tooth

When a posterior tooth is lost and not replaced there is the potential for drifting or migration of opposing or adjacent teeth. Any movement will



Fig. 18.7. These two illustrations demonstrate Thielmanns Diagonal Law. Loss of the lower first molar has lead to over-eruption of the opposing upper molar. This tooth then interferes with excursive movements to the extent that the diametrically opposite upper central incisor has overerupted.



Fig. 18.8. This is not the same patient but it shows the problems that arise from the loss of a lower molar. The second molar has tilted forward and the over-eruption of the upper molar leads to an interference in mandibular movements.



Figs. 18.9 & 18.10. These two illustrations show how to count the units for posterior support. Bicuspid and third molars are allowed one point each and molars score two points. It is essential to have at least eight units with the teeth opposing each other down their long axis.

take place within the first year following loss of normal contact and will occur more readily and to a greater extent in the presence of active periodontal disease.³ Movement will then slow down and, in an otherwise healthy mouth, will cease. However, any alteration to the tooth to tooth relationship between the arches may lead to alterations to contacts during lateral or protrusive excursions or to changes in the direction of the slide between centric relation and centric occlusion.

Loss of a lower first molar, for example, may lead to mesial tilting of the lower second molar followed by over eruption of the opposing upper first molar. The centric occlusion position may

remain apparently comfortable but the initial contact in the centric relation position may then be the distal marginal ridge of the lower second molar against the distal marginal ridge of the upper second molar. The mandible, in fact, may be thrust forward, upward and to one side to achieve a centric occlusion position and, in so doing, load the lingual of the upper anterior teeth, leading to undue wear on the lingual surfaces or migration in the presence of periodontal disease.

Also, if the upper molar erupts far enough, it may act as a guidance in protrusive movements, making the mandible swing laterally as well as foreward, thus producing unusual wear patterns on the incisal edge of the contralateral central

incisor tooth or even encouraging migration, particularly in the presence of periodontal disease. There are many combinations of these modified patterns and it is essential that the occlusion be examined with great care to establish the cause in any situation showing wear patterns outside of a relatively normal range.

BE AWARE



Failure to replace a lost posterior tooth can have serious and complex consequences.

Loss of posterior support

As more teeth are lost the problems become further compounded.^{4,5} It is apparent that it is not necessary to retain thirty two teeth and in fact the average patient has no more than twenty eight teeth in full function. Many get by comfortably and safely with less still. However, below a certain number it may not be possible to maintain a normal vertical dimension and function without remaining teeth migrating or being depressed into the alveolus under excessive load (**Figures 18.9 and 18.10**).

Minimum posterior support

A formula for defining the minimum amount of posterior support required to prevent loss of vertical dimension can be stated in the following terms. If molars are allotted two units and bicuspid and third molars are allotted one unit each, then a total of eight units in function is sufficient to maintain posterior support to the extent that, in a normal healthy mouth, the occlusion will not collapse. This assumes that the teeth oppose each other in a relatively normal relationship and that their periodontal status is acceptable. Whilst it is desirable to be able to record units on both sides of the mouth it is not essential for stability. If there are less than eight units present it is likely that, particularly in the

NOTE



Minimum posterior support

- Molars count as two units
- Bicuspid count as one unit
- Third molars count as one unit
- Minimum units required – total eight units

presence of periodontal disease, the upper anterior teeth will migrate labially, the lower anteriors will tilt to the lingual and remaining posteriors will either drift under load or be depressed into the alveolus leading to loss of vertical dimension.

Distal rotation of the mandible

An additional problem arising from loss of posterior teeth is the potential for distal rotation of the mandible placing extra load on the condyles and the temporo-mandibular joint.⁶ It has been suggested that the fulcrum for this distal rotation lies about the upper and lower second bicuspid in the average occlusion. In other words, as long as the dentition is complete as far as the second bicuspid in both arches, the mandible will not rotate distally. If all eight bicuspid are present and in normal occlusion the parameters of both theories are fulfilled (**Figures 18.11 and 18.12**).

Interpretation of Wear Facets

Careful examination of the occlusal surfaces and incisal edges of remaining teeth will often reveal much useful information and help to lead to a diagnosis and treatment plan. Direct intraoral examination with a good light source is essential and this should be supplemented with mounted study models. There is a further discussion on recognition and diagnosis of wear facets and their causes in **Chapter 5**.

Patient habits

Discussion of the problem with the patient may reveal further information. Both dietary and cleaning habits are significant, particularly in relation to erosion, and it is essential that the patient understands the chemistry of demineralisation. Stress and tension are factors in parafunction and, though there may be little that a dentist can do to relieve this, just discussing the situation may help the patient. In fact, it is important that the patient understands the whole cycle because it will help to rationalise problems ranging from obvious wear patterns to split teeth and loss of cusps.

Occasionally, it will be discovered that the patient can place the mandible in a bizarre position to produce a wear pattern and it seems impossible to reproduce this position on an articulator.



Figs. 18.11 & 18.12. If there are four bicusps in normal apposition on each side then there will be sufficient posterior support and the mandible will be stable against the maxillary teeth. On the other hand, if the lower second bicuspid is missing there will be a point of rotation at the distal of the lower first bicuspid and there is the potential for considerable load on the TMJ.



Figs. 18.13 & 18.14. A typical problem arising from failure to take account of the occlusion. The mesial marginal ridge in the amalgam restoration in the upper bicuspid has been overcarved to allow for the distal corner of the incisal edge of the lower canine. The latter has now over-erupted sufficient to lead to development of a severe incline between centric relation and centric occlusion positions resulting in a heavy load being applied to the upper anteriors.

Deflective inclines

Excessive wear on the lingual surfaces of upper anterior teeth or the incisal edges of lower anteriors may be the result of a deflective incline between the centric relation position and centric occlusion.⁷ This should be readily detectable both in the mouth and on the articulator and the cause defined. The problem frequently arises through under contouring the mesial marginal ridge of an upper first bicuspid allowing the opposing canine to erupt into the void. The situation will be exacerbated if there has been a collapse of the upper anterior segment through loss of teeth or in the presence of a Class II Division 2 malocclusion. The wear pattern on the teeth appears to depend on which teeth lose the enamel first, the uppers or

the lowers, and the wear may be excessive before being detected. In an extreme case the anteriors may fit together so intimately that there appears to be no room in which to place a protective restoration. Elimination of deflective inclines in the posteriors between centric relation and centric occlusion may allow the mandible to move distally sufficient to make room for a restoration and, at the same time, eliminate the cause (**Figures 18.13 and 18.14**).

Balancing side interferences

Excessive wear on canine teeth is generally the result of the development of a balancing side interference. These interferences are mostly on second and third molars and the further distal the



Fig. 18.15. An example of heavy erosion probably arising from heavy toothbrushing after ingestion of low pH food and drink.

contact the greater the load and the leverage that can be applied. Deep intercuspation between upper and lower molars, particularly second or third molars, is generally the result of loss of canine guidance and cuspid rise. Conversely it may arise from deeply overcarving the occlusal anatomy of a molar restoration thus allowing overeruption of the opposing tooth. This in turn may lead to the development of both balancing side, as well as working side, interferences followed by parafunction and excessive wear on the canine.

Chemical erosion

Generalised wear patterns may arise as a result of chemical erosion with abrasion and attrition as a secondary cause. The real cause may be difficult to detect because information from the patient is essential for a correct diagnosis and this is not always forthcoming. However careful observation may lead to a diagnosis. For example loss of enamel on the lingual surfaces of the upper posteriors as well as the upper and lower anteriors is generally caused by highly acidic gastric reflux. It can be the earliest sign of either anorexia or bulimia, both of which may be associated with gastric reflux.

Loss of enamel on the labial surfaces of the upper anterior teeth as well as the lingual of the upper posteriors may arise from frequent ingestion of low pH liquids such as citrus juices, cola drinks, athletic drinks or wine (**Figures 18.15 and 18.16**).



Fig. 18.16. There is probably a chemical origin for this excessive erosion exacerbated by vigorous tooth brushing.

Damage arising from excessive wear

Modification to aesthetics

Probably the most obvious effect of excessive wear is alteration to the aesthetics of the upper or lower anterior teeth. These are the ones the patient sees and the ones to which they are likely to draw attention (**Figures 18.17 and 18.18**) (**Chapter 7**).

Wear on the incisal edges of anteriors will generally arise from parafunction and the cause must be identified before treatment planning for restoration. Elimination of deflective inclines will often allow sufficient distal movement of the mandible to allow restoration where it was apparently not possible before. Restoration of the incisal edge without identifying the cause will inevitably lead to failure.

Split cusps

Split cusps are a common cause of pain and on many occasions alterations to the occlusion will be a factor. Initial symptoms will be intermittent pain on pressure and the patient will have difficulty in determining the actual origin. Pain will become more frequent and eventually the tooth will become sensitive to change of temperature. Finally the entire cusp will fail leaving a surface which is temperature sensitive for a day or two and then is comfortable except that it will probably be sharp and rough to the tongue (**Figures 18.19 and 18.20**).

Occasionally the split will progress through the pulp chamber and this will lead to extreme pain and possibly loss of the tooth. Identification of the



Fig. 18.17. Uneven wear on the incisal edge of the upper central incisors is almost certainly the result of over-eruption of the upper second molar on the opposite side. The result is that the protrusive movement is interfered with and misdirected.

cusps involved is often difficult and may only be confirmed by removal of the old restoration. There are a variety of devices for applying discrete pressure to individual cusps, and one of these may be useful, but it may require lateral stress in a particular direction to reveal the problem.

Functionally opening contact

Functionally opening contacts are a common problem arising from altered occlusal patterns. They generally occur in relation to heavily restored teeth, most often the terminal tooth in the arch, where there has been a large restoration placed with a direct plastic restorative material. Recontouring the occlusal surface is difficult in these circumstances and is largely the result of guesswork. Arising from the atypical occlusal anatomy and the subsequent drifting and tilting of opposing teeth there may be a deflective incline developed between the centric occlusion and centric relation positions. As the patient makes the initial contact in centric relation on a mesial facing incline of, say, an upper molar with a distal facing incline of the opposing lower molar the upper molar will move slightly distally opening the contact sufficiently to engage a piece of fibrous food debris. As the patient opens, the upper molar will move mesially again entrapping the food debris. This situation has, in the past, been called a plunger cusp but this is a misnomer because simply reducing the height of an opposing cusp will not necessarily eliminate the prob-



Fig. 18.18. Generalised wear on all teeth probably arising from a lack of canine guidance as well as lack of posterior support.

lem. It is essential to observe the entire occlusion and identify deflective inclines in relation to the offending area and modify them to be successful.

Restoration of occlusal harmony

Examination and monitoring of the intermaxillary relationship is an essential prerequisite for successful treatment.⁸ For a new patient identify the initial contacts in the centric relation position and then observe the presence or absence of a slide to one side or the other as the patient moves into the centric occlusion position. Determine the cause of the slide. Now observe the lateral excursions and assess the presence or absence of a balancing side contact. This is more difficult to determine and examination of study models will assist. Reassess these movements periodically because the relationship is never static, even in the presence of an apparently stable unrestored dentition.

Working side and balancing side interferences

Look carefully at each tooth, drying them off and varying the angle of the light to seek wear facets on surfaces which should not be worn. Working side interferences can be just as damaging as balancing side contacts and must be identified. They may be reproduced on study models but identification in the mouth will often tell you more than the model.

Careful observation of non-working cusps on molars, in particular, is very important. They are not subject to the heavy load of working cusps



Fig. 18.19. An upper second bicuspid has spontaneously split mesio-distally. This is an unusual failure and is probably the result of clenching and grinding as a result of stress.



Fig. 18.20. The mesio-lingual cusp of the lower molar has failed probably because of over carving or excessive wear on the amalgam restoration.



Figs. 18.21 & 18.22. A large amalgam restoration in the lower molar has been deeply carved to allow retention of the lingual cusp on the upper molar. The resulting deep inter-cuspal contact has led to excessive load on the disto-lingual cusp on the lower molar which is almost certain to split.

and may be left unworn and therefore relatively high. They then become working side interferences with the potential for undue guidance in excursions and are very prone to develop a split at the base. In a situation such as seen in **Figure 18.21** the disto-lingual cusp could be reduced and the cusp incline modified to reduce the stress and eliminate the possibility of interferences in lateral excursions.

Alternatively, if the lower molar requires a restoration the occlusal anatomy can be restored and the opposing cusp reduced in height leading to a similar reduction in intercuspation.

Adjustment directly in the mouth can be carried out before commencing restorative treatment but, on many occasions, the required adjustments can be achieved simply by altering cuspal inclines following placement of a restoration. If there is any

doubt concerning the possible result of adjustment and alteration to cuspal inclines, carry out the modifications on study models first, including removal of inclines or the addition of anatomy to the occlusal surfaces, using a trial wax up technique (**Figures 18.21 and 18.22**).

Orthodontic correction

Orthodontic correction may be an essential prerequisite for successful reinstatement of occlusal harmony. This may become more difficult as the patient ages but, conversely, it may be very important in some situations to include a degree of orthodontic correction. Prevention of relapse is then the main problem and fixed bridgework may be required.

Restore posterior support

Restoration of posterior support is essential for the badly broken down dentition which has included random loss of posterior teeth. However it is only necessary to rebuild the support until there are eight units present and stable as described above. Fixed bridgework or implants are generally necessary because removable partial dentures are not efficient as load bearers and the patient may not wear them conscientiously. A free end saddle on a removable partial denture will not be an effective load bearer at all and a tooth born saddle must be very well seated and supported if it is to make any contribution to posterior support.

Restore vertical dimension

A decision as to whether vertical dimension has been lost may be difficult to resolve and is often controversial. If there are less than eight units of posterior support remaining there is almost certain to be some loss of height but in the presence of an acceptable number of normal posterior units it is almost impossible to lose dimension. Restoration can be carried out quite simply and it is normally only a matter of determining the amount of space required to accommodate the new restorations rather than to go to any length to determine what the patient will tolerate. Providing correct occlusal balance is maintained, with freedom of movement and proper guidance, the patient will accommodate very readily to an increase of up to

one centimetre or more without really being aware of the change (**Figures 18.23 and 18.24**).

It is important to use fixed prostheses rather than removable partial dentures. Any tooth that is left out of occlusion in the process of restoration of the vertical dimension will erupt over a period of time and achieve a new occlusal contact. This may be done deliberately with success. For example, a lone molar tooth which has been under heavy load may be left out of a rehabilitation and within a year it will be in function. It will be necessary under these circumstances to review the occlusion periodically to ensure that the new pattern is stable and acceptable.

BE AWARE



Loss of vertical dimension

- Only if loss of posterior support
- Restore with fixed appliances only
- Tooth taken out of contact will re-erupt

Use of a removable splint

The use of an occlusal splint to test if the patient will tolerate restoration of vertical dimension is not valid and may even deter the patient from further treatment. It may be of value as part of overall treatment of a grossly broken down dentition, particularly if there are signs of a TMJ pain/dysfunction syndrome, but insistence on a trial period prior to rebuilding an occlusion cannot be justified. For a patient already wearing a partial



Figs. 18.23 & 18.24. A set of study models demonstrating the perils of loss of posterior support. It is not so much over eruption of the posterior teeth but the teeth bypass each other and any tooth remaining in contact is likely to be intruded into the alveolus.

denture it may be worthwhile modifying it at the proposed vertical dimension but it must be noted that any tooth not fully engaged within the splint may move to the extent that, with the splint out, the occlusion will be unstable. Essentially, if a removable appliance is to be utilised at any stage, all teeth must be fully engaged when the splint is in the mouth and the occlusion must still be completely stable when the splint is out.

BE AWARE



Restoration of vertical dimension

- Removable splint trial run not essential
- All teeth in contact with splint in and splint out

Restoration over time

Finally, it should be noted that development of a stable harmonious occlusion may well be a long term project. Longevity is the key to success so restorative techniques and materials should be chosen because their future is predictable. Use restorative materials with known wear factors and, where possible, oppose each material with a

similar material. Recognise the long term effect of alterations to the occlusion which may arise from using materials which wear faster than natural tooth structure. Be prepared to modify the occlusal surface of the opposing tooth when an old restoration needs to be replaced. Do not necessarily contour a new restoration to accommodate an existing opponent without contemplating the advantages of recontouring the opponent. Restore vertical dimension with care and ensure that all teeth are stable at all times. Observe patients frequently and reassess the occlusion looking constantly for change. Carefully examine the occlusion after any breakdown to determine if there is an occlusal component in the failure.

BE AWARE



Occlusion will constantly change as a result of

- wear of opposing teeth
- wear of opposing restorative materials
- drifting or tilting of teeth
- parafunctional habits
- occlusal irregularities

Further Reading

1. Posselt U. *Physiology of Occlusion and Rehabilitation*. Oxford: Blackwell Scientific Publications; 1962
2. Hanau RL. Full denture prosthesis. Intraoral technique for Hanau articulator model – H. Buffalo, 1930.
3. Love WO, Adams RL. Tooth movement into edentulous spaces. *J Prosthet Dent* 1971; 25:271-77.
4. Kayser AF. Shortened dental arches and oral function. *J Oral Rehab* 1981; 8:457-62.
5. Zarb GA, McKay HF. The partially edentulous patient: 1. The biological price of prosthodontic intervention. *Aust Dent J* 1980; 25:63-8.
6. Kerveskari P, Alanin P. Association between tooth loss and TMJ dysfunction. *J Oral Rehab* 1985; 2:189-94.
7. Wise MD. *Failure in the Restored Dentition: Management and Treatment*. London: Quintessence Publishing Company Ltd.; 1995.
8. Randow K, Glantz P-O, Zoger B. Technical failures and some related clinical complications in extensive fixed prosthodontics. *Acta Odontol Scand* 1986; 44:241-5.

19

Choosing Between Restoration Modalities

G. J. Mount

It is necessary to take a number of factors into account in selecting the restorative material to be utilised in the restoration of any tooth. There are both advantages and disadvantages with each of the materials currently available and none of them is ideal. Whilst there is considerable pressure at this time to use aesthetic restorative materials where ever possible it must be acknowledged that longevity should be the guiding factor and aesthetics should not be regarded as paramount.

Maintenance of the original tooth structure should be the primary aim in treatment of the caries lesion but as the lesion progresses and the cavity becomes larger there comes a stage where sacrifice of further tooth structure is required to provide protection from occlusal load to ensure maintenance of the remainder. In view of the fact that no restorative material is ideal and that all materials display a potential life span of no more than twenty years a considerable amount of restorative work represents replacement of old restorations.

Generally, replacement is indicated because of recurrent caries, failure of the previous restorative material or fracture of remaining tooth structure. Inevitably, there will be further loss and weakening of the remaining tooth structure. These factors will dictate the choice of the replacement material which must always offer the greatest longevity possible.



Introduction

There is a limited number of restorative materials available within the discipline of operative dentistry and none of them can be regarded as universal. All have their advantages, disadvantages and limitations.¹ There is a degree of pressure at present to place aesthetic restorations at all times and there appears to be a lack of appreciation of longevity as a guiding factor. There is a continuing series of investigations being carried out by both the academic world as well as private practice to test the bounds of longevity and it is suggested that the student of this subject carefully observe the results.² In many cases estimations are based upon restorations placed by undergraduate students but the real test comes from operators working in general practice. The following discussions are based upon the latter.

Glass-ionomer

Advantages

This is the only material currently available that is bioactive and capable of true diffusion based chemical adhesion to both enamel and dentine through an ionic exchange between the restoration and the tooth surface. It is a water based cement and therefore stable in the oral environment. It is bioactive so it is capable of exchanging calcium, strontium and phosphate ions with tooth structure. It is also a dynamic material in as much as the chemistry of the setting reaction will continue for a long period of time after placement. As a result of the method of manufacture of the glass, the cement contains fluoride which is released into the surrounding tooth structure after placement. There is a strong release initially which reduces over the first two months but has been shown to still be present over at least seven years. The fluoride content can be continually recharged from topical applications from many sources, including toothpaste through to professionally applied fluids or gels. As a result, this material

has a strong resistance to the development of recurrent caries at the interface with the tooth structure and there will be little plaque formation on the surface.

Glass-ionomer is available in a number of forms depending on powder/liquid ratio, particle size and variations in the chemistry of the setting reaction. It is used extensively as a luting material for crowns and bridges as well as a lining and a dentine substitute under other restorative materials. Each of these versions is fast setting and develops an early resistance to water contamination. One of the most useful versions is the restorative cement which has an acceptable degree of translucency and colour matching and, because of its adhesive potential, there is no need to modify the cavity design to develop mechanical interlocks for retention.

Both the Type II restorative cement and the Type III lining cement are available in a light activated form (resin modified or dual cure) as well as the original chemically activated (autocure) system. The dual cure mechanism has been developed through the addition of further resins, including HEMA and photoinitiators. The main advantage is resistance to water contamination immediately the cement is set. The original acid/base reaction, which allows the development of adhesion to the tooth, and is therefore the key to the glass-ionomer system, is still present but is protected by the umbrella like presence of the dual cure resins.

SUMMARY



Glass-ionomer

Advantages

- Ion exchange adhesion to tooth structure
- Ion exchange with tooth structure
- Continuing fluoride reservoir
- Acceptable aesthetics
- Good wear factor on maturity
- Low solubility on maturity

Disadvantages

- Low fracture resistance
- Subject to dehydration in absence of saliva

Disadvantages

The main limitation is a relatively low fracture resistance such that it cannot be used alone to withstand undue occlusal load. It is not suitable to rebuild marginal ridges or incisal corners but, providing it is well supported by surrounding tooth structure, the physical properties and abrasion resistance are sufficient to withstand considerable load. Solubility is also low but improves over time due to the long term setting mechanism. On occasions, translucency in the chemically activated (autocure) materials may not be sufficient for colour matching and lamination with composite resin may be required for a satisfactory aesthetic result.

A further important caution is that, as a water based material, it is subject to dehydration, particularly in the early stages after placement. In addition, it will dehydrate and disintegrate in the presence of a low salivary flow. For patients with Sjögrens Syndrome and similar salivary incompetence it is important to confine the use of glass-ionomer as a dentine substitute and to laminate over it with another material to protect it.

Composite Resin

Advantages

Restorations with excellent aesthetics can be built with composite resin particularly with the modern light activated varieties. When the resin is placed incrementally, and properly light activated at each stage, variation in both colour and translucency can be incorporated and anatomical form reproduced reasonably accurately. Much work has been carried out in recent years on the filler particles incorporated within the resin and there is considerable variation between products marketed by different manufacturers. Physical properties and translucency will be affected by the filler content and the ability of a material to take and retain a smooth polished surface will also vary.

Properly placed, its physical properties can be sufficient to withstand moderate occlusal load as

far distally as the bicuspid and the wear factor of the more heavily filled types is sufficient for these restorations. Relative to ceramic restorations it is inexpensive but it has a notably shorter potential lifespan.^{3,4} It is relatively simple to develop a micromechanical union between enamel and composite resins and this is the strongest adhesion available in the oral cavity.

Disadvantages

Placement techniques are extremely complex and require patience and a high degree of clinical skill. One of the main problems at present is the overall shrinkage of the resin mass during the curing phase. The chemically cured composites shrink towards the centre of the restoration thus tending to pull away from the walls of the cavity and towards the floor. When cured by light activation, shrinkage will occur towards the light pulling the resin away from both the floor and the walls. Careful incremental placement will minimise the total shrinkage but it may still be sufficient to place considerable stress on the bond of the restoration to either the tooth or a cement base.⁵ These restorations are therefore subject to microleakage especially in relation to margins placed on dentine. In the presence of microleakage the pulp may become inflamed unless it is adequately sealed and protected with a glass-ionomer base under the composite resin.⁶

Both the Bis-GMA and urethane dimethacrylate resins, which are the basis of most composite resins, are inert materials with no bioactivity at all. As they do not contain water, an ion exchange is not available. However, they are relatively hydrophilic and take up water over time, and this will lead to a degree of breakdown, particularly under occlusal load. This means the wear factor can be significantly high, particularly if the restoration is expected to maintain posterior support. On the other hand some of the composite resin formulae containing large particle sizes may cause wear on the enamel of opposing teeth. Determination of the true extent of the wear factor can be quite complex because, while the teeth may appear to maintain occlusal contact, there may be quite extensive wear taking place. The

only way to confirm this is to take serial impressions and measure the contour of the occlusal surfaces. The risk imposed by continuing wear is development of deeper intercuspation leading to heavy lateral stresses, split cusps and occlusal interferences⁷ (Chapter 18).

Considerable research has been undertaken in an attempt to develop an adhesive union between the composite resins and dentine. *In vitro* testing demonstrates satisfactory results but it has not been possible to repeat these *in vivo* at this point.

Union between the filler particles and the resin matrix is developed through adding a silane coating to the filler particles. This is expected to lead to a chemical union throughout the restoration. However, this bond represents a potential weakness because it may be incomplete or hydrolyse and break down and release the particles thus increasing the wear factor.

Allergies have been reported by both operators and staff, as well as patients, to some of the ingredients. Methylmethacrylate and HEMA have both been identified as allergens since resins were first used many years ago for the construction of dentures. Particles of barium or strontium glass may be released as a result of wear and the long term effects arising from their ingestion is not yet known. Formaldehyde can also be a by-product of the setting reaction.

SUMMARY



Composite resins

Advantages

- Excellent aesthetics
- Excellent adhesion to enamel
- Accepts a quality polish
- Variety available for different tasks
- Wear factor acceptable
- Relatively inexpensive

Disadvantages

- Placement techniques require high level of skill
- Difficult to restore ideal coronal anatomy
- Bond to dentine questionable
- Resin is hydrophilic and takes up water over time
- Longevity questionable

Amalgam

Advantages

Over many years, particularly in small restorations, amalgam has been shown to have a very satisfactory history of longevity with a potential halflife of up to twenty five years. It is relatively easy to handle through standardised methods and placement techniques have become very routine. It seems to be very tolerant of less than ideal placement techniques although over contour, particularly in relation to the gingival tissues can pose problems. Properly placed, it has physical properties which are sufficient to withstand occlusal stresses and it is a very economical material to work with and therefore cost effective. It is not in any way bioactive and it does not have any caries resistant properties in itself. However, it will corrode quite rapidly in the oral environment, and the corrosion products will seal the margins against microleakage within the first three weeks after placement. It is therefore highly resistant to recurrent caries.⁸

Disadvantages

The main disadvantage is that it is aesthetically undesirable. The material itself is a dark grey in colour and as it corrodes it darkens in itself and also releases metallic ions into the surrounding dentine leading to a blue discoloration in the remaining tooth structure. Corrosion is therefore both an advantage and a disadvantage. In modern formulations with a high copper content, the corrosion potential has been reduced sufficiently to minimise the disadvantages although there is still enough corrosion product available to seal the margins against microleakage.

Reproduction of occlusal and proximal anatomy is difficult to achieve in placement of all the direct plastic restorative materials and the problems increase as the cavity gets larger. Relatively sophisticated matrix techniques are available but reproduction of full proximal anatomy, particularly in relation to the marginal ridge and contact area, is difficult.

Occlusal anatomy can be restored for the smaller restoration but becomes more difficult as the cavity extends, particularly if a cusp needs to be protected. It is not possible to determine the success of the reconstruction of the anatomy until the restoration is completed and the patient is able to close the mouth and check the occlusion. The wear factor of amalgam is slightly greater than enamel, therefore, if the restoration is extensive and the occlusal load is heavy the occlusion may not remain stable over long periods and should be continually observed and monitored.

Mercury safety issues are discussed in **Chapter 13**.

SUMMARY



Amalgam

Advantages

- Relatively easy to handle
- Relatively tolerant of poor placement techniques
- Excellent longevity in small to medium sized lesions
- Least expensive of the direct restoratives

Disadvantages

- Very poor aesthetics
- Difficult to restore full anatomical form
- Wear factor too great for extensive restorations

Gold

Advantages

The physical properties of gold alloys are variable with four types of alloys available for the restoration of teeth using either direct or indirect techniques. Direct gold restorations are placed in the form of gold foil which is pure 24 carat gold leaf. This will cold weld to a uniform mass through malleting directly into a cavity. In addition there are three Types of gold for tooth restoration using indirect techniques – Types A, B and C – which range in hardness upwards from Type A which is the softest. With this range available it is possible to select a relatively ideal material for restoration of any situation. It must be noted that

the wear factor with gold is very similar to that of natural tooth structure.

Modern casting techniques allow for very high accuracy in fit and the inherent strength and ductility of gold means that it can be utilised in thin section to protect remaining tooth structure. It is a noble metal and will not corrode and, because of its highly polished surface texture, it is resistant to plaque formation.

One of the major advantages is that, when a restoration is produced by an indirect technique in the laboratory, it is possible to reinstate both the occlusal and proximal surfaces to the full anatomy of the original tooth. This is difficult indeed with any other material, except possibly ceramics.

Disadvantages

Placement of direct malleted gold foil is relatively time consuming and is indicated for small, one surface restorations only. Gold inlays and crowns, constructed by indirect techniques, involve complex laboratory procedures as well as rather lengthy chairside operations. They are, therefore, relatively expensive in the first instance although their longevity will generally justify the initial outlay. In addition the multi-stage production routines allow the introduction of errors at any one of these stages and it is essential that skilled operators are available at all times, both in the clinic and the laboratory. The error, or accumulation of errors, will often only be detected at the final insertion appointment and may require repetition of the entire procedure.

In the current era of aesthetic dentistry the appearance of a gold restoration becomes debatable. Not all patients are opposed to them but many request more aesthetic materials.

SUMMARY



Gold

Advantages

- Wide range of materials available
- Highly accurate fit available
- Reinstate full coronal anatomy
- Wear factor similar to tooth structure
- Longevity justifies the care and cost

SUMMARY**Gold****Disadvantages**

- Multistage production allows for errors
- High skill required at all stages
- Relatively high cost initially
- Aesthetics a matter of choice

Ceramics

Advantages

The art and science of dental ceramics has reached a very high level and it is the material of choice for aesthetic restorations. With careful attention to detail ceramic crowns can defy detection by the naked eye. Because plaque will not readily accumulate on a fully glazed ceramic surface tissue tolerance is very high and a skilled technician can reproduce the anatomy of both the occlusal and the proximal surfaces with great accuracy.

The physical properties of glazed ceramic are of a high order and their abrasion resistance is such that the opposing tooth is more likely to wear than the restoration. The cost of these restorations to the patient is high but the initial outlay can be justified because of the longevity and superior aesthetics available.

Disadvantages

Because of the many stages involved in the production of ceramic restorations there is a high potential for the introduction of errors. All stages of production in both the clinic and the laboratory must be carried out to the highest possible standard if success is to be assured.

The fracture resistance of ceramics is not high and restorations are prone to cracking and chipping, particularly if the occlusion is not properly developed. Repair and replacement is expensive and failure will generally require complete reconstruction. In addition, the wear factor of ceramic

against both natural tooth structure as well as gold and the other materials is rather high, particularly if the porcelain has lost its glaze. If the material is to be used it is better to oppose ceramic to ceramic rather than to oppose it to any other material.

SUMMARY**Porcelain****Advantages**

- Perfect aesthetics available
- Complete reinstatement of anatomy
- Accuracy of fit

Disadvantages

- Multistage production allows for errors
- High skill required at every stage
- Relatively high cost
- Wear factor high on natural tooth structure

Factors governing the selection of a restorative material

Taking into account the advantages and disadvantages of the available restorative materials as listed above the following rationale can be applied in any given situation with the materials discussed in order of preference.

Restoration and maintenance of physical properties of a tooth

Gold is the material of choice in as much as it can be used in thin section to protect and reinforce remaining tooth structure. Also, constructed by indirect techniques, it is possible to reform the ideal contour and anatomy of a tooth and rebuild occlusion with a high degree of accuracy.^{9,10}

Ceramic restorations also are built by indirect techniques and therefore it is possible to rebuild anatomy and occlusion with a high degree of accuracy. However they are too brittle to be designed in thin section and therefore, generally, more of the remaining tooth structure must be removed to allow sufficient room for the restorative material.

Both amalgam and composite resin should be confined to intracoronal restorations only and

therefore it is generally not possible to restore strength to remaining tooth structure. Adhesion with resins is only as strong as the tensile strength of the component parts of the system and neither composite resin, resin bonding agents nor enamel are consistently strong in tension. Cavity modifications and an acid etch union between enamel and resin can provide a degree of protection to weakened cusps, but not substantial reinforcement.

The adhesion available with glass-ionomer cement will restore some of the lost physical properties but the cement itself has a relatively low tensile strength and cannot therefore be relied upon to offer a significant reinforcement to remaining tooth structure.

Restoration and maintenance of occlusion

Gold is the material of choice because, depending on the alloy selected, the wear factor is almost identical with that of natural tooth enamel. Because the restoration is built using an indirect technique, occlusion can be refined to a high degree and will subsequently be maintained over many years, almost without change.

Ceramic restorations are useful for restoring anatomy and occlusion because they must be built indirectly. However the technique is more demanding and the wear factor is much greater than gold. The porcelain surface will not flow and create the Beilby veneer which is seen with metals and therefore they abrade opposing surfaces, particularly as the porcelain loses its glaze. It is desirable to oppose porcelain to porcelain and not porcelain to natural tooth structure or any other restorative material.

Amalgam, composite resin and glass-ionomers are not suitable for the restoration and maintenance of the occlusion because the wear factor is too great with all three. In addition it is almost impossible to reliably recontour the occlusal surface in these materials directly in the mouth with restricted access and vision and, generally, there is no opportunity to add to the restoration if it has been inadvertently carved out of occlusion during placement.

Restoration and maintenance of aesthetics

Ceramic is the material of choice for restoration of the larger lesion because it is possible to simulate the original tooth in colour, translucency and character with a high degree of accuracy. However, in many cases, it is necessary to be relatively destructive of remaining tooth structure to make room for the porcelain. With the advent of reliable adhesion to sound enamel through resin bonding techniques it has become possible to produce ceramic veneers to restore the labial surfaces of anterior teeth with minimal removal of enamel. Care must be taken to avoid over contouring because this may compromise gingival health.

With careful attention to detail it is possible to restore aesthetics using composite resin. More conservative techniques can be utilised but the integrity of the margin depends entirely on the availability of sound, well supported enamel which can be etched so the resin can be bonded to it.¹¹

Glass-ionomer is a useful aesthetic restorative material with the main limitation being an inability to withstand heavy occlusal load. It is the material of choice for an erosion lesion or for any lesion where involvement of the occlusal surface is at a minimum. If the load is expected to be too great it can be laminated with composite resin and this combination has been shown to restore reasonable strength to the remaining tooth structure.

Choice of restorative material according to size of lesion

Natural tooth structure is the best defence against further caries. It can be remineralised and generally the patient can be educated in dietary and hygiene procedures (**Chapters 4 and 7**). Remineralised tooth structure is just as hard as the original tooth and is more resistant to further caries attack. Therefore, even though the surface may be mildly disfigured and stained, the most conservative approach to cavity design should be adopted. The following recommendations are offered as a rational approach to the choice of cavity design and the subsequent selection of the restorative material.¹²

Site 1 lesion – pits and fissures on smooth surface

- Newly erupted immature tooth with deep fissures
Place a protective coating using an unfilled resin or a glass-ionomer. No instrumentation will be required.
- Mature tooth with small carious dentine involvement
Open into the caries very conservatively. Follow out remaining fissures only where there is a possibility of further caries. Restore with glass-ionomer and laminate if essential.
- Moderate size cavity with no undue occlusal load
Open conservatively and restore with glass-ionomer base and a composite resin laminate for areas subject to heavy occlusal load
- Large cavity in a molar with extensive occlusal involvement
Restore with amalgam over a glass-ionomer base using a protective cavity design where required to prevent further breakdown.

Site 2 lesion – contact area, posterior

- New lesion just involving dentine
Restore using glass-ionomer as the principal restorative material. Laminate only if the occlusal load is heavy
- Larger lesion with marginal ridge involved
Use a conservative modified cavity design. If the occlusal fissure is not carious, restore the proximal box with composite resin over a glass-ionomer base and seal the fissure with resin. If the occlusal load is heavy, particularly in molars, restore with amalgam. When replacing a failed restoration, where the occlusal load is acceptable, use glass-ionomer as a dentine substitute and laminate with composite resin.
- Extensive lesion leaving undermined and weakened cusp/s
Use protective cavity design modified for restoration with amalgam or gold inlay so as to provide protection over those cusps requiring support. Gold is the most conservative material when used as an inlay because it can be placed in thin section to protect remaining

cusps. However, the design must be such as to relieve stress on cusps that are already split and retentive elements must be in the gingival one third of the crown.

- One or more cusps already lost
The cavity design is now complex and extensive. Additional retention must be provided using grooves and ditches in the gingival one third of the remaining tooth structure. Restore with amalgam as the primary restoration with a gold or ceramic extracoronary restoration to follow.

Site 2 lesion – contact area, anterior

- Initial lesion
Glass-ionomer is the material of choice for the restoration. Wherever possible the lesion should be entered from the lingual thus minimising problems of aesthetics, for both the present and the future.
- Large lesions or replacement restorations
Glass-ionomer remains the material of choice for the primary lesion. However, once the incisal edge and the labial surface is involved and aesthetics is compromised the cement will need to be placed as a dentine substitute and laminated with composite resin.

Site 2 lesion – involving incisal corner

- Small initial lesion
The small lesion in this classification is likely to occur only as a result of trauma. If the enamel only is involved it may be sufficient to bevel the enamel and restore with composite resin. If there is any dentine involvement it should be protected with a glass-ionomer first before lamination to provide pulp protection and adhesion to the dentine.
- Larger lesions or replacement restorations
Restore the entire lesion with a glass-ionomer. Immediately cut back the cement sufficiently to expose the entire enamel margin on the labial and sufficient of the lingual enamel to ensure a union strong enough to withstand the anticipated incisal load. If the enamel is weak along the gingival margin, leave the cement covering that margin to a

depth of about 2 mm. Bevel the enamel margins, etch and laminate with composite resin.

Site 3 lesion – cervical margin

- Erosion lesion

A Type II.1 restorative aesthetic glass-ionomer cement is the material of choice under most circumstances. No instrumentation is required and the lesion should be cleaned with pumice and water only to remove the pellicle before being conditioned and restored. The aesthetic result should be checked after one week and if it is unsatisfactory the cement can be cut back lightly and laminated with composite resin. The resin-modified glass-ionomers are generally aesthetically satisfactory.

- Carious lesion or replacement restoration

The cavity should be instrumented only as far as is essential to remove active caries. Mechanical retention is unnecessary and demineralised enamel may remain unless badly undermined. Glass-ionomer is the material of choice and the cavity will need to be conditioned before restoration. If the aesthetic result is still unsatisfactory after one week the cement can be cut back and laminated with composite resin.

Extracoronary restorations

- Porcelain or composite resin laminate veneers

A laminate veneer can be regarded as a relatively conservative method of modifying the shape or aesthetics of anterior teeth. As at least half the thickness of the labial enamel must be removed to avoid over contour, this restoration should not be regarded as reversible. Also it must be noted that, as it is almost impossible to know the thickness of remaining enamel, the labial surface should not be cut back to an arbitrary depth for fear of inadvertently removing it all and leaving a bond to dentine only. This applies particularly to the gingival margin where it is most difficult to achieve a good bond to dentine.

Any existing restoration should be fully

upgraded first using glass-ionomer and non-vital teeth should be bleached as close as possible to their correct colour. Composite resin may be the preferred material for minor modifications to one or two teeth only but indirectly built porcelain veneers are recommended for extensive modification of several teeth at a time.

- Three-quarter veneer

This is the most conservative of the extra-coronary restorations and is valuable for reinforcing posterior teeth in particular. Gold is the correct material to use because it can be cast with a high degree of accuracy and used in very thin section. Underlying restorations should be fully upgraded first using amalgam or glass-ionomer. Skilful cavity preparation will minimise the gold display and in many cases the aesthetic result is entirely acceptable.

- Full crown

A full coverage restoration is required where either the remaining tooth structure is so badly broken down that no other method will adequately restore the tooth or it is necessary to upgrade aesthetics or occlusion. If aesthetics is of no concern gold is the material of choice because remaining tooth structure can be conserved to a greater degree and also the occlusal wear factor is the same as tooth structure. Construction of a porcelain crown will require removal of a greater amount of tooth structure to allow bulk in the crown for both fracture resistance and aesthetics.

Existing restorations should be fully upgraded first and, in view of the fact that retention of such a restoration is gained in the gingival one third of the remaining tooth, amalgam is the material of choice for such work. Glass-ionomer may be sufficient to make good small deficiencies, particularly on an anterior tooth but composite resin does not adhere to dentine so it is inadequate for this purpose.

Further Reading

1. Dawson AS, Makinson OF. Dental treatment and dental health: Part I. A review of studies in support of a philosophy of minimum intervention dentistry. *Aust Dent J* 1992; 37: 126-32.
2. Hawthorne W, Smales R, Webster D. Long term survival of restorative materials in private dental practice. *J Dent Res* 1994; 73: Abstr. 85, pp.747.
3. Barnes DM, Holston AM, Strassler HE, Shires PJ. Evaluation of clinical performance of twelve posterior composite resins with a standardised placement technique. *J Esthet Dent* 1990; 2:36-43.
4. Lambrechts P, Williams G, Van Herle G, Braem M. Aesthetic limits of light cured composite resins in anterior teeth. *Inter Dent J* 1990; 40:149-58.
5. Sheth JJ, Fuller JL, Jensen ME. Cuspal deformation and fracture resistance of teeth with dentine adhesives and composites. *J Prosthet Dent* 1988; 60:560-69.
6. Davidson CL, Kemp-Scholte CM. Shortcomings of composite resins in Class V restorations. *J Esthet Dent* 1989; 1:1-4.
7. Bryant RW. Posterior composite resin restorations – a review of clinical problems. *Aust Prosthodont J* 1987; 1:41-50.
8. Pink FE, Minden NJ, Simmonds S. Decisions of practitioners regarding placement of amalgam and composite resin restorations in general practice settings. *Oper Dent* 1994; 19:127-32.
9. Mount GJ. A review of newer restorative materials. Part 1. *Dent Today* 1989; 5:1-6.
10. Mount GJ. A review of newer restorative materials. Part 11. *Dent Today* 1990; 6:1-8.
11. Tyas MJ. Adhesive dental restorative materials and systems. *Annals of the Royal Australasian College of Dental Surgeons* 1989; 10:101-7.
12. Dawson AS, Makinson OF. An alternate philosophy and some new treatment modalities in operative dentistry, Part II. *Aust Dent J* 1992; 37:205-10.

20 Failures of Individual Restorations and Their Management

G. J. Mount

It has been claimed by many authorities that between 70-75% of the clinical time of the average operator is occupied carrying out replacement dentistry to overcome what is loosely called recurrent caries. There are many reasons for failure and there is always a temptation to replace a restoration entirely rather than repair it. However, each time a restoration is replaced there is, inevitably, further loss of tooth structure, and that which remains will be weakened. It is desirable, therefore, that all factors be taken into account before a decision is taken to remove all remaining restorative material on the grounds that, in many cases, repair of the existing restoration may be adequate. Interpretation of failure should never be made on external appearance alone because this may be very deceptive. Firstly, it is essential that the cause of failure be assessed and, if possible, fully determined. One of the most common causes is continuing caries as a result of failure to eliminate the disease and this should be fully investigated in all cases. However, sealing an active lesion with a glass-ionomer is often sufficient to arrest the active phase and allow a lesion to heal. Determination of the physical properties of the remaining restorative material may pose problems because it cannot be assessed without removing it. It is generally not possible to be certain that there is no further active caries under the restoration, or to make a valid assessment of the condition of the pulp. Some restorative materials can be repaired more readily than others but adequate access to the area of breakdown may be difficult.



Failure of Tooth Structure

Continuing caries

There is no doubt one of the most common reasons for the need to replace a restoration is failure to eliminate the disease of caries in the first place. The term recurrent caries is the most usual reason cited in the majority of surveys of replacement dentistry but it should really be used with caution. Is the recorder observing a continuation of the original disease or is this a new lesion resulting from a fresh attack of caries arising from a breakdown of normal oral health? For the sake of the patient it is important to differentiate because general health problems may lie behind it. Caries is clearly a bacterial disease and the cause and control is discussed in earlier chapters. If the original disciplines to control caries are not undertaken then it should not surprise if further lesions develop in relation to the margin between restoration and tooth structure. Obviously the intimacy of the union between the two is a weakness because of the potential for bacterial microleakage into the gap. On the one hand the material needs to be closely adapted to the cavity walls but, on the other hand, over contour or excess material beyond the original contour of the

crown of the tooth may become a prime site for the accumulation of plaque.

Once the bacterial burden has been reduced to an acceptable level, hygiene levels have been established and there is some control of refined carbohydrate intake, minor deficiencies can be tolerated. Elimination of the disease is the primary essential if individual failures are to be controlled. It is not difficult to identify examples of very poor dentistry being tolerated for long periods, with no sign of active caries, in a mouth that is free of disease.¹ On the other hand, the best dentistry may fail if disease is rampant.

However, there are a number of other factors that need to be understood and controlled. Tooth structure can fail at the cavity margin adjacent to a restoration for a variety of reasons, including leaving a margin under direct occlusal load or introducing microcracks in the enamel during cavity preparation. Bulk failure of an entire cusp may follow preparation of a cavity because it is often sufficient to weaken the crown. Alternatively, the restoration itself can fail at the margin or in bulk if it is subjected to excessive load or its full physical properties have not been developed during placement. Either way, failure may lead to the development of further caries in relation to deficiencies or else to loss of aesthetics or function as a result of loss of bulk tooth structure.



Fig. 20.1. The enamel margin along the occlusal edge has failed mainly because the margin of the original cavity was extended too far to the tip of the cusp without taking into account the occlusal load. The margin should have been extended over the cusp tip allowing the amalgam to take the load and protecting the enamel.



Fig. 20.2. The enamel margin has failed around this amalgam in two areas mainly because the areas of contact of the opposing cusps were not taken in to account in the original cavity design. The load at the distal margin in particular is obvious.

Failure of the enamel margin

Enamel is a brittle material with a very specific grain because it consists of serrated rows of enamel rods lying parallel to each other and at right angles to the surface of the crown. Ideally the cavity margin in the enamel should lie at right angles to the surface. This is often difficult to achieve and this means that wedge shaped defects along the margins may arise through failure of the enamel rods which have been foreshortened and left unsupported. Alternatively, the margin may have been placed too far up the medial facing cuspal incline and therefore be subjected to heavy occlusal load (**Figures 20.1 and 20.2**).

Also, the enamel rods can suffer microcracks during cavity preparation following use of an eccentrically rotating bur. If an adhesive material such as composite resin, is then placed, the stresses induced by the setting shrinkage may lead to further development of these cracks.

There will be occasions where the bulk of the restoration is sound and it will be acceptable to rebuild one section only. Conservative treatment of minor ditching at the margin can often be achieved by limited opening, with a very fine tapered diamond bur, and restoration with a glass-ionomer. However, if the defect is of long standing it will be wise to explore as far as the dentine beneath to make sure there is no active caries

within. Because amalgam has the ability to seal its own margins through corrosion, limited repair is often a proposition. However, composite resin has no such safety factor and marginal failure can be dangerous and lead to rapid carious involvement. If the restoration is gold it may be repaired with gold foil although a small defect, which is not under undue occlusal load, can be sealed with glass-ionomer.

If a limited repair is contemplated it is wise to consider the occlusion and the strength of the remaining tooth structure. If the enamel margin has failed because of undue occlusal load then it may be desirable to extend the margin of the restoration further still so that the restoration takes the stress rather than the limited amount of remaining enamel. However, this may involve a complete redesign or selection of an alternate restorative material.

Failure of the gingival enamel margin at the base of a proximal box may arise from poor placement of the original restorative material, but is almost invariably the result of continuing or recurrent caries. Because this is such a caries prone region, elimination of the disease is paramount before repair is contemplated. It is then sometimes possible to prepare a limited tunnel approach to the lesion, generally working from the buccal, to be restored with a glass-ionomer.

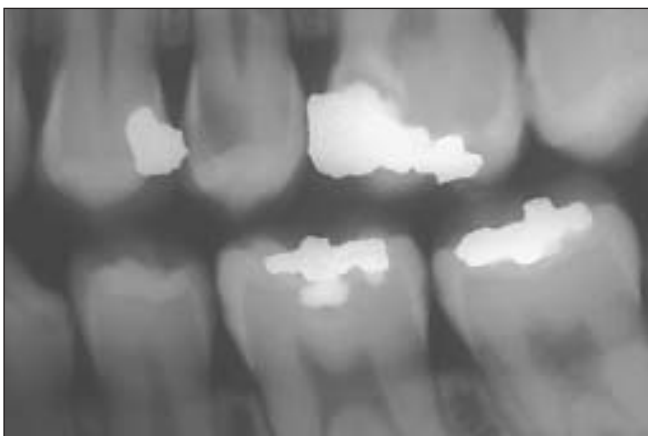


Fig. 20.3. The amalgam in the mesial of the upper molar is faulty, at least in part because of failure to condense the restoration completely. The failure is complicated by the overhang which encouraged further plaque accumulation and therefore caries.

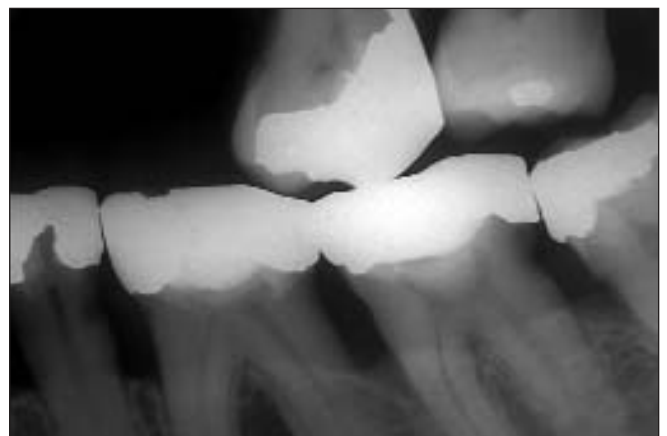


Fig. 20.4. There is further caries below the gingival margin of the restoration in the distal of the second molar, in part through poor placement technique, but also because of the overhang on the restoration in the mesial of the adjacent tooth.

This is the recommended material because of the ion exchange adhesion and bioactivity which will assist in controlling further caries.

Failure of dentine margin

It is generally the gingival margin of the proximal box of a restoration which is in dentine and detection of a fault and subsequent repair may pose problems. Often the cause is an operator error such as failure to adapt or condense the restorative material adequately at the margin. Also failure to develop a good contact with the adjacent tooth may lead to food impaction. Probably the greatest problem arises from an over contour or overhanging margin on a restoration because it will retain plaque (**Figures 20.3 and 20.4**).

Root surface caries is not specifically failure of a dentine margin although it will often be interproximal and easily confused with failure of the adjacent restoration margin. In fact, root surface caries is generally the result of a new attack of caries, mostly in an aging patient following gingival recession. Even the best restoration can fail under these circumstances and successful treatment will rely primarily upon control of the disease in the first place (**Chapter 7**). Remineralisation is often possible, particularly if the lesion is detected early prior to actual cavitation.

The decision on whether to repair the margin or replace the entire restoration will depend on two

factors. Access to the lesion is not always easy without undesirable destruction of remaining tooth although sometimes a tunnel cavity design from the buccal or lingual, with restoration using glass-ionomer, can lead to a satisfactory resolution. Alternatively, the main bulk of the restoration may be of low quality and, under these circumstances, the entire restoration should be redesigned.

Bulk loss of tooth structure

The strength of the crown of a tooth lies in maintenance of the circle of enamel around the full circumference of the crown. Once the circle is broken by the preparation of a cavity on a proximal surface for placement of a restoration the integrity of the cusps is at risk.² This situation is exacerbated by cutting the traditional trench across the occlusal surface to eliminate the occlusal fissure. It is not surprising then that a common failure is the development of a split at the base of a cusp leading ultimately to its loss (**Figures 20.5 and 20.6**).

Preparation of the trench to deal with a fissure, as in the traditional Class 1 cavity, will double the length of a cusp. Preparation of the proximal box, as in the traditional Class II cavity, will double the length again. Occlusal pressure on the remaining medially facing inclines of the cusp will then exert considerable leverage and a split at the base should not be surprising.



Fig. 20.5. This molar responded to occlusal pressure so the extensive amalgam was removed. The crack running mesio-distally shows clearly at the base of the lingual cusps.



Fig. 20.6. A very common failure is the complete loss of the lingual cusps particularly in lower molars following failure to provide sufficient protection from occlusal load.

Prevention of such failures is not easy but begins with the preparation of the initial cavity designed to deal with the earliest lesion. Cavity designs such as the tunnel (**Chapter 14**) are desirable because they minimise the involvement of the proximal enamel. The slot design, also described in **Chapter 14**, is the next choice because it eliminates the occlusal trench. If neither of these modifications can be employed both the width and depth of the occlusal trench should be as limited as possible. Maintenance or restoration of the original, relatively shallow, occlusal anatomy is desirable even to the extent of modifying the height of the opposing cusp to maintain a proper occlusion. Particularly in replacement dentistry it will often be found that the depth of intercuspation is excessive due to previous deep carving of the occlusal anatomy of the restoration being replaced. On many occasions, the problem can be reduced by judicious reduction of the height of the opposing cusp thus eliminating the need for over-carving of the new restoration. This will minimise the intercuspation of the opposing teeth and limit lateral stresses on remaining cusp inclines (**Chapter 18**).

Loss of an entire cusp is distressing for the patient. It often arises through failure to take into account the weakened nature of the remaining tooth structure in an extensively restored tooth and failure to provide some form of protective restoration. It is also necessary to continually monitor changes to the occlusal wear patterns because loss of occlusal anatomy may result in a nonworking cusp eventually standing high and becoming subject to lateral stress. There is good reason to monitor nonworking cusps - such as the lingual cusps of lower molars - because over the years occlusal wear can leave these cusps subject to undue lateral stress. There is no reason why the anatomy cannot be modified by shortening the cusps and altering the cuspal incline to minimise lateral stress and reduce the risk of fracture. This will not alter the vertical dimension but it may, in fact, eliminate balancing side contacts which can, on occasions, be regarded as lateral interferences.

Repair of a lost cusp generally requires replacement and redesign of the entire restoration. Occasionally a protective restoration is already in

place with the occlusion being sustained by the restorative material. Under these circumstances it may be sufficient to simply repair the defect by adding to the existing material or placing a composite resin or glass-ionomer veneer. However there will now be reduced support for the remaining restoration and it will need to be soundly based to accept the extra load. Also, it may be desirable at this point to explore the remaining tooth structure because of the possibility of a split elsewhere requiring further protection. If the restoration is to be converted to an extracoronal design it is essential that the primary restoration be very soundly based and firmly retained by underlying tooth structure with retentive grooves and ditches so that it will not be disturbed or weakened by preparation for the final full crown.

Split root

This occurs generally in the remaining root structure of a nonvital tooth which has been restored with a post crown. The post is essentially an intraradicular restoration which relies on the integrity of the root to sustain it. It will naturally be subjected to considerable lateral stresses, particularly in an anterior tooth, and there is a need to reinforce the root against these forces if at all possible. Minimal enlargement of the root canal during endodontic treatment and subsequent preparation for a post is highly desirable and the best method of prevention. It is sometimes possible to place a cuff around the top of the root as part of the post and core design but the most difficult area in which to prepare for this cuff is around the lingual gingival margin. Considering the direction of the stresses, this is the area which requires the most reinforcement. A split in a root will allow the development of tensile forces on the cement which will eventually destroy the cement and allow the loss of the crown.

Diagnosis of a split root is very difficult and, almost invariably, terminal in the life of the tooth. When a post crown becomes uncemented the remaining root must be carefully explored for signs of a split. The use of magnification and a fiberoptic light to illuminate the tooth from various angles may be sufficient. A caries detecting

dye may help or simply applying leverage may show percolation of gingival fluid on the root face. If the diagnosis is not conclusive recement the crown, adjust the occlusion and advise the patient of a possible further failure at a later date. If the recementation lasts less than 12 months, the cause is almost certainly a split root (**Figures 20.7 and 20.8**).

Once the diagnosis is confirmed it must be acknowledged that repair for the long term is impossible and an alternative restoration should be planned.



Fig. 20.7. The post crown in this upper central incisor became uncemented on two occasions. Careful exploration shows the presence of a split which is now visible at the lingual of the post hole.

Loss of vitality

There will need to be a modification to the treatment plan following loss of vitality whatever the cause. There is likely to be a shift in the translucency or colour of the remaining crown and some further weakening following the enlargement of the root canal during root canal therapy. Any pre-existing restoration will need to be reviewed and possibly redesigned.



Fig. 20.8. A tooth showing a similar failure to the one shown in **Figure 20.7**. The tooth was extracted as it is beyond recovery. This shows the two parts of the root of the tooth demonstrating the typical direction of the split which runs upwards and buccally to a point about two thirds up the length of the post.



Fig. 20.9. These amalgams demonstrate the common ditching along the margins that many amalgams suffer from within a reasonably short period after placement. This is of no concern as long as there is no disease present and it is unwise to polish the amalgam back to eliminate the ditch because this will alter the occlusion.



Fig. 20.10. The ditching along the margins of these amalgams is notably more extensive and is exacerbated probably by poor placement technique and maybe contamination during condensation. There is a greater risk of recurrent caries than with the patient shown in **Figure 20.9**.

Failure of Restorative Material

Failure of the margin of the material

Most of the restorative materials, other than gold, have a poor edge strength and therefore may not withstand undue occlusal load. It is important in designing a cavity to try to place the margin away from an area subject to direct occlusal load. Where the margin must be under load, the edge of the restorative material should have a cavo-surface margin close to 90°. There must be a compromise between strength in the material and strength in the enamel and the other properties of each material will have a bearing on final cavity design and therefore the potential life span of the restoration.

Amalgam

Amalgam has a relatively poor edge strength and ditching along the margins is not uncommon. However, because the interface between the cavity and the restoration will seal itself as a result of corrosion of the amalgam, there will not often be a further caries lesion developing. In spite of the fact that the average amalgam restoration looks less than ideal within a reasonably short period of time after placement, repair of the margins is not normally indicated. Ditching of the margin of a low copper amalgam should be regarded as normal (**Figures 20.9 and 20.10**).

Repolishing the occlusal surface to improve the margins will result in alteration to occlusal anatomy and contact with the opposing tooth and is strictly contraindicated. There are differences between high copper amalgams and other alloys in their resistance to marginal ditching and corrosion and these factors have been discussed in **Chapter 13**.

Composite resin

Composite resin has no resistance at all to a renewed invasion of caries so failure, through loss

of adhesion at the margin, requires immediate attention. If the margin is left open on the occlusal surface, plaque will be forced in to the gap under the high hydraulic pressure generated by mastication and caries will develop rapidly.³ It is essential that the defect be explored in depth with care and, in the majority of cases, extensive replacement of the restoration is necessary. Occasionally, simply resealing the breakdown can be achieved, particularly if the restoration is relatively new, but the repair should be kept under careful observation for some time thereafter.

In view of the fact that it is difficult to obtain long term adhesion between composite resin and dentine, failure at the gingival margin is not uncommon. Repair is not normally appropriate and replacement of the entire restoration is generally indicated. The use of a glass-ionomer base is strongly recommended in order to avoid this type of breakdown in the first place (**Chapter 11**) (**Figures 20.11 and 20.12**).

Glass-ionomer materials

Failure of a glass-ionomer restoration is generally the result of poor handling of the material at the time of placement. Ditching around the margins is generally the result of using a low powder content mix leading to a weak material. Early water contamination before the material is mature could have a similar result. The development of cracks in the bulk of the material is generally the result of failure to protect the newly placed cement against dehydration prior to maturation.

Providing it is well supported by surrounding tooth structure, a glass-ionomer can be used to restore an occlusal lesion and, even under heavy occlusal load, it will not be subject to marginal or bulk failure. However, it does require a certain amount of bulk to resist marginal ditching so it should not be expected to survive as a thin veneer. Also, as the tensile strength is not high it is not generally regarded as being suitable for the restoration of a marginal ridge or incisal corner.

If failure should occur then complete replacement is probably the best solution. As the union between old and new glass-ionomer is not strong, it is generally best to remove all the old material

right down to sound tooth structure so that it will be possible to generate a new ion exchange adhesion layer with enamel or dentine. However, the cause of failure must be determined first and an alternate material placed if the cause is not clear. Another method of repair would be to partially remove the old glass-ionomer and laminate what remains with a composite resin.

Gold

Occasionally, gold will fail along a margin as a result of further wear on the occlusal surface, particularly if opposed by a ceramic restoration with a high wear factor. As gold has no inbuilt resistance to further attack, caries may progress rapidly and the defect can become very extensive in a relatively brief period. This means that any defect should be explored with considerable care. Assuming the original cause can be eliminated, repair of the margin with gold foil may be adequate. If the occlusal load is not great then glass-ionomer can be utilised in a very conservative repair.

Loss of luting cement

All indirectly fabricated restorations carry the risk of dissolution of the luting agent over time. Longevity in the restoration begins with elimina-

tion of the disease. Following this, the use of a low solubility luting cement, combined with high quality laboratory techniques to ensure an accurate fit in the first place, are the best methods of control. Repair is difficult because the margin is often close to, or under, the gingival tissue. If caries is becoming active along the margin, repair can be attempted by opening conservatively and placing glass-ionomer. The alternative is replacement of the restoration.

It is interesting to note that, in a completely healthy mouth, it is possible to have a full crown become uncemented through dissolution of the cement but show no sign of further caries on the tooth surface.

Fracture or Collapse of a Restorative Material

Fracture through the main bulk of a restoration is potentially dangerous, particularly if a segment is retained within the cavity after becoming mobile. Rapid caries will develop because plaque will be admitted under the mobile segment and it will then be forced into the dentine tubules under masticatory pressure. It is preferable that the



Fig. 20.11. The composite resin restoration shows considerable loss of structure over a period of about ten years. Modern composite resins are expected to last longer but this is a typical form of failure with this material.



Fig. 20.12. There are two Site 2, Size 2 composite resin restorations in these upper anteriors both showing marginal leakage and loss of colour after a period of about five years.

entire restoration be lost immediately after failure but, in fact, the directly placed plastic restorative materials are often retained through the retentive design of the original cavity or adhesion to enamel along one margin.

Amalgam

Bulk failure of an amalgam restoration is not uncommon and there are several possible causes. It is essential that each section of a complex amalgam restoration be individually retentive. That is to say both the proximal box and the occlusal extension need to have their own retentive design because neither one can be expected to support the other. Add to that, the material must be properly placed and fully condensed to achieve its proper potential for physical properties. The causes of failure can be

- inadequate retention in a section of the original cavity design,
- failure at the isthmus of a Site 2, Size 2 (2.2) restoration may occur because the proximal box is not locked into the dentine with retentive grooves and ditches. Apparent lack of bulk in the material at the isthmus and the design of the axiopulpal line angle are of little significance,
- placement of an inappropriate lining materi-

al. The use of a lining material which hydrolyses and disintegrates may leave the amalgam without physical support,

- multiple layers of lining materials, or one lining material in excessive bulk, will reduce the volume and therefore the physical properties of the final restoration.

Failure to condense the material adequately during placement or contamination during condensation will also reduce the physical properties although amalgam is a very forgiving material and attainment of full physical potential is rarely achieved. The modern concept of bonding an amalgam into the cavity using a composite resin bond is quite insufficient to retain an amalgam in a cavity. It is essential to incorporate mechanical interlocks as well under all circumstances. The only cure for this type of bulk failure in an amalgam restoration is complete replacement of the entire restoration taking added care with the design of the cavity (**Figures 20.13 and 20.14**).

Composite resin

Composite resin may fail in a similar fashion to amalgam although it is rather flexible and failure will normally occur at the margins rather than in bulk. Reduction in physical properties leading to failure can be attributed to failure to light cure the



Fig. 20.13. Bulk failure of the restorative material itself is not common and only occurs as a result of failure to make allowance for the intrinsic brittleness of amalgam in particular. Note that this failure is not because of weakness in the isthmus but failure to provide proper retention of the amalgam in the proximal box.



Fig. 20.14. The same restoration as shown in **Figure 20.13** following removal of the piece of amalgam. The reason for failure is now apparent. There is no substantial box in the cavity design to support the restorative material and there is too much lining material. The amalgam therefore failed through lack of support.

material for long enough or the inclusion of contaminants between increments. The relatively low depth of cure of the average composite resin is a clinical trap and considerable care needs to be exercised to make sure each increment is fully cured. Both the proximity of the light to the surface of the restoration as well as the length of time of application are significant. If the composite resin has been built over a glass-ionomer base, which has been placed as a dentine substitute, the risk of further caries, immediately following failure, will be reduced over the short term because of the presence of the cement. However, replacement without delay of the entire restoration is generally necessary. The cause of the failure must be determined and a decision made as to the replacement material to be used.

Porcelain

Generally gold does not break but ceramic crowns, inlays and veneers are relatively brittle and therefore subject to bulk failure.⁴ A careful analysis of the reason for failure is essential if the replacement is to succeed. There are several possible causes:

- Occlusion – it is essential to maintain a properly balanced occlusion in the presence of porcelain restorations because irregularities may lead to parafunction on the restoration and bulk failure.
- Design – porcelain requires both adequate bulk and stable support. The marginal ridge of a molar crown made of porcelain bonded to metal should have a metal shoulder below it. The lingual of an anterior crown should have adequate thickness if it is to withstand occlusal load.

Repair of porcelain is difficult and complete replacement is generally required. There are a number of proprietary products offered for the repair of chipped or broken porcelain but it is very difficult to match the color properties of ceramic with any other material and adhesion between the two within the oral environment remains tenuous. Also the wear factor is always greater with composite resin so the life span of repairs with materials other than porcelain remains limited.

Total Loss of a Restoration

Rigid restorations

This is generally the result of loss of cementation of a rigid extracoronal restoration. The fault generally lies in incorrect cavity design although poor handling of materials, failure to study the occlusion or bulk failure of tooth structure will contribute.⁵

Extracoronal restorations should be retained through a fully retentive design, and the luting cement is utilised, essentially, to prevent microleakage between the restoration and the tooth. The physical properties of the cementing medium may be insufficient to withstand undue tensile stresses though compressive properties may well be adequate to accept occlusal load. The main reasons for cementation failure will be improper mixing of the cement or contamination during placement of the restoration. Alternatively, the retentive features of the design may be inadequate. A careful assessment of the cause is required before recementation to avoid repeated failure.

Direct plastic restorations

Amalgam and composite resin will rarely disappear entirely from a conventional cavity but composite resin or glass-ionomer may be lost from erosion lesions without leaving a trace. The cause will generally be failure to develop the full adhesion potential of either material by leaving surface contamination on the cavity at the time of placement. Alternatively abfraction stresses may be involved and the occlusion should be examined to assist in diagnosis (**Chapter 5**). Develop a fresh surface on the dentine before attempting to replace the restoration in case the existing surface is sufficiently demineralised to be unsuitable for chemical adhesion. Similarly, following loss of a composite resin there will be tags of resin remaining in the surface layer of enamel or dentine and it will be necessary to freshen the surface by removing up to 100 μm of tooth structure so that adhesion can be established again.

Change of Restorative Material

When any restoration fails it is desirable to reassess the situation and decide if the existing material is the correct material of choice under the circumstances. Each replacement means that there will be further loss of natural tooth structure and, of course, this is a finite resource. None of the currently available restorative materials can be regarded as totally permanent in the true sense and therefore the longevity of each restoration is important. Selection of the material for restoration of the initial lesion and then for each replacement will need to take into account such factors as

- caries rate
- occlusal load
- ability to protect remaining tooth structure
- aesthetics
- size of the cavity, ie. the amount and strength of remaining tooth structure
- economic considerations

Apart from the essential requirement of controlling the disease of caries no one factor should dominate this decision apart from the patients long term well being and stability. The following factors should be considered for each material.

Glass-ionomer

Indications

- Simple to handle clinically, relatively tolerant of variations in placement technique and inexpensive to use.
- Chemical union with both enamel and dentine with an ion exchange adhesion which is proof against microleakage.
- Continuing ion exchange with tooth structure and the oral environment throughout the life of the restoration leading to some degree of remineralisation and healing of demineralised dentine.
- Adequate for aesthetics and it can be veneered with composite resin if necessary to enhance physical properties and aesthetics.

- Ideal for use in the presence of a high caries rate because of the chemical adhesion and continuing fluoride release.
- The preferred material for long term provisional restorations.

Contraindications

- Unable to withstand heavy occlusal load without adequate support from surrounding sound tooth structure and may require protection through another restorative material laminated over it.
- Water-based and therefore will not survive in the presence of xerostomia.

Composite resin

Indications

- Satisfactory for the restoration of small lesions and areas under moderate occlusal load.
- Has excellent aesthetics, at least in the short term.
- Generally, physical properties are sufficient to accept moderate occlusal load but the wear factor is less than ideal and it should be used on occlusal surfaces of molars with discretion
- Can develop an excellent seal with etched enamel providing the enamel is sound and well supported.
- Long-term union with dentine is doubtful. To develop sound dentine adhesion it should be used in conjunction with a glass-ionomer base.

Contraindications

- It is complex and demanding to place properly in the oral cavity. Therefore it is more expensive to place and has a relatively short clinical life span.
- It has limited ability to restore extensive cavities because of problems associated with achieving both proper interproximal contour and occlusal anatomy.
- It has a relatively large setting shrinkage so the larger the cavity the greater the total shrinkage, thus putting considerable stress on the margins and the union with remaining tooth structure.

- It has no built in resistance to bacterial invasion and should, therefore, be used with caution in the presence of a high caries rate
- It is based on methylmethacrylate which is a known allergen and contains materials such as HEMA which can also cause an allergic reaction. The full degree of toxicity is not yet understood.

Amalgam

Indications

- Relatively simple and inexpensive to use and reasonably tolerant of careless placement technique.
- Physical properties are generally adequate to withstand occlusal load.
- Efficient and cost effective for the restoration of average to medium sized cavities because carving and contouring direct in the oral cavity is straight forward in the presence of guidance from remaining tooth anatomy.
- It can be used to a limited degree to protect remaining tooth structure.
- Excellent in the presence of a high caries rate because it corrodes and seals its own margins and is economical to repair.

Contraindications

- Contains mercury and is a known health hazard to dental staff.
- Has been known to lead to an allergic response in a small number of patients
- Poor aesthetics and tends to produce a blue grey colour change in any tooth.
- It is limited in the restoration of extensive cavities because of the difficulty of restoring correct occlusal anatomy directly in the mouth.

Gold

Indications

- When well constructed gold restorations show the greatest longevity and this will often justify their use in spite of additional cost.
- Physical properties are ideal for the restoration of the occlusion.
- Indirect methods of construction are generally

utilised and this allows for the ideal reconstruction of all aspects of anatomy, both occlusal and proximal.

- It can be used in very thin section for protection of remaining tooth structure

Contraindications

- Gold restorations are complex to construct, with the potential for error at any one of a number of stages, and are therefore relatively expensive.
- It cannot be recommended in the presence of a high caries rate.
- Aesthetics is a matter of opinion and some patients regard it as unsatisfactory.
- Gold itself has no built in resistance to bacterial invasion. However, a glass-ionomer luting cement will allow a continuing ion exchange and may provide some protection.

Porcelain

Indications

- Longevity may well justify its use.
- Excellent aesthetics available, at least over the medium term.
- Physical properties and indirect methods of construction are adequate for reconstruction of the occlusion.

Contraindications

- Ceramic restorations are complex to construct, with the potential for error at any one of a number of stages, and are therefore expensive.
- Porcelain may cause undue wear on natural tooth structure, and other restorative materials as well, so care must be exercised in using it on an occlusal surface.
- Porcelain itself has no built in resistance to bacterial microleakage. However, a glass-ionomer luting cement will allow a continuing ion exchange and may provide a degree of protection.
- It cannot be recommended in the presence of a high caries rate or a heavy occlusion. It is important the occlusal problems be overcome first.

Further Reading

1. Mjör IA. Repair versus replacement of failed restorations. *Inter Dent J* 1993; 43:466-472.
2. Bell GJ, Smith MC, dePont JJ. Cuspal failure of MOD restored teeth. *Aust Dent J* 1982; 27:283-7.
3. Jorgensen KD, Matona R, Shimakobe H. Deformation of cavities and resin restorations in loaded teeth. *Scand J Dent* 1976; 84:46-50.
4. Mount GJ. Repair of porcelain fractures. *Dent Outlook* 1985; 11:84
5. Mount GJ. Failures in crown and bridgework. *Dent Outlook* 1985; 11:53-58.

Index

A

Abfraction	8, 9, 55
Abrasion	48, 59
tooth reduction	48
Acid	36, 98
Dietary	24
Endogenous	54, 99
Exogenous	54, 99
Acidulated fluoride phosphate	41
Activator lights	213
Acute pulpitis	307
Adhesion	
composite resin	206
glass-ionomer	147, 178
Affected layer	302
Air abrasion	128
Alcohol intake, oral effect	102
Amalgam see Dental Amalgam	
Amelogenesis	3
Annoyance factor	120
Apatite	
deposition	2
A.R.T. (atraumatic restorative treatment)	304
Attrition	51
interproximal	52

B

Bacteria	
<i>Lactobacillus</i>	23
<i>S. Mutans</i>	23
<i>S. Sobrinus</i>	23
Biofilm	63, 73
Bisphenol-A diglycidyl dimethacrylate	201
Benzoyl peroxide	201
Bond, composite resin	206
glass-ionomer	147, 178
Bruxism	51
cusp fracture	154
enamel flaking	55
Buffering	
of acid	24

by saliva	73
measuring capacity	73
Bulimia	102,106
Bur selection	120
lubrication	125
speed groups	125
Burnish	
Final	229
Precarve	227

C

Caffeine	100
Calcium fluoaluminosilicate glass	165
Calcium hydroxide	296
Calculus	
origin of	312
removal	312
Casein phosphopeptide-amorphous calcium phosphate	114
plus fluoride	117
Carbohydrate, fermentable	94
frequency	22
Caries lesions, classification of	246
Caries progress	27
advancing	29
lifestyle – effect of	106
myths	62, 112
rampant	30
risk factors	65
Cavity classification	246
by G. V. Black	245
new classification	246
reasons for change	244
Site 1, Sizes 0-4	248
Site 2, Sizes 0-4	258
Site3, Sizes 0-4	278
Sites of lesions	246
Sizes of lesions	247
Cavity design	
general principles	152
G. V. Black's concept	245
reasons for change	244
Cementum	8
Centric occlusion	325

Centric relation position	325
Cermet	165
Chemo-mechanical caries removal	136
Chewing gum	107
Chlorhexidine	44, 102
Chronic pulpitis	303
Cigarette smoking, effect of	102
Cola drinks	101
Compomers	167,200
Composite resin	200
Adhesion	148
Bond – dentine	206, 211
enamel	206, 212
Choice	339
Colour stability	204
Components	201
Curing	213
Depth of cure	203, 213
Effect on pulp	293
Failure	
enamel margin	349
longevity	214
margin of material	355
total loss	356
glass-ionomer base	196
incremental buildup	213
light activation	213
depth of cure	213
Lutz & Phillips classification	202
Mechanical properties	
fracture toughness	205
hardness	205
strength	205
wear	205
packable	202
polyacid modified	200
polymerisation	202, 205
radiopacity	204
setting time	203
shade selection	208
shrinkage	205
thermal diffusivity	203
water sorption	204
wedging	211

Condensation of amalgam	228
Conditioning	180
Copal varnish	231
CPP-ACP	112
chewing gum, in	117
effect of	113
formula	113
gel, mousse	117
Cusp, protection	
failure	351
split	154, 351
Cvek pulpotomy	308

D

Demineralisation	25, 64, 71
Demineralisation/remineralisation	25
cycle see Dental Caries	
Dehydration	100, 102, 106
Dental amalgam	
adaptation	230
biocompatibility	235, 294
bonded	232
bulk fracture	233
burnish	229
choice	340
classification	220, 221
clinical performance	235
condensation	229
contamination	228
copper content	221
corrosion	224
creep	224
cusp protection with	154
dimensional change	225
electron photomicrographs	222
failure	
at margin	232
bulk fracture	233, 355
galvanic effect	224, 234
lamination	236
marginal fracture	232
marginal seal	231
mercury content	223
minor elements	221
particles	
lathe cut	221
spherical	221
placement	228, 229

repair	234, 226
retention	151, 152
self-sealing	231
strength	225
thermal properties	225
trituration	226
water contamination	228
wear factor	235
zinc content	220

Dental caries	
bacterial flora	23
demin./remin. cycle	25
demineralisation	25
fermentable carbohydrate	23
fissure caries	248
fluoride effect	76
indirect pulp capping	290
infected/affected dentine	30
progression	28
rampant	30
recurrent caries	
remineralisation	25
risk assessment	
diagnostic tests	66
patient attitude	76
patient history	77
root surface caries	30
white spot lesions	113

Dental pulp	
ideopathic resorption	16
indirect pulp therapy	300
inflammation	13
necrosis	14
protection	300, 304
pulp response to caries	13
pulp tests	19

Dentifrice, containing fluoride	41
---------------------------------	----

Dentine	
adhesion	149
caries progression	29
conditioning	180
diffusion through	7, 12
ideopathic resorption	16
infected/affected	30
permeability	12,
sclerosis	13
secondary	13
smear layer	7
tubules	5

Dentine bonding agents	206, 211
Diabetes and saliva	104
Diet Analysis	80
Drinks, acidic, erosion	80
frequency of intake	80
Drugs, acidic, erosion	103
illicit	103
prescription	77
over the counter (OTC)	91
recreational	77

E

Eating disorders	100
Electronic fissure testing	32
Emergence profile	314, 320
Enamel	
calcification	2
caries progression	27
crystals	2
failure of margin	349
flaking	55
mineralisation	3
perikymata	3
prisms	3
resin bonding	148
rods	3
Epithelial attachment	310, 313
Erosion	
chemical	52
extrinsic	58
intrinsic	58
Etching	
dentine	148
enamel	148
Eugenol	304

F

Filler loading	201
Fissures	
at risk	249
cavitation	251
Fissure protection	
glass-ionomer	254
Fissure sealants	
composite resin	252
glass-ionomer	254

- Fluorapatite
 critical pH 26
 formation 26
- Fluoride
 application schedules 41
 caries inhibition 24, 36, 40
 compomers, release of 201
 giomers, release of 201
 guidelines for therapy 39
 glass-ionomer, release of 186
 mouth rinses and washes 42
 Safety factors
 adults 43
 children 44
- Functionally opening
 contact 316, 332
- ## G
- Gastric reflux
 chemical erosion 99
 effect on saliva pH 99
- Gingival tissue
 emergence profile 314
 matrix placement 319
 normal, healthy 310
 rubber dam and wedges 319
- Giomers 168, 200
- Glass-ionomer
 abrasion resistance 188
 adhesion 147, 178
 to collagen 179
 aesthetics with 157
 amalgam alloy included 165
 anhydrous 164
 autocure 164
 base, use as a 196
 biocompatibility 184, 297
 capsules 170
 choice of 338
 classification 182
 composition 164
 conditioning of dentine 180
 core build-up 192
 dental pulp 186
 dimensional change 187
 dispensing and mixing 169
 fissure protection 254
 fluoride content 165
 fluoride release 186
 handmixing 172
 indications for 191
 ion exchange mechanism 147, 176
 lamination with 193
 lining, use as 193
 liquid 165
 luting, use as 182, 186
 paste/paste dispensing 172
 placement routine 182
 plaque inhibition 185
 pulp response 293
 radiopacity 190
 resin-modified 166, 175
 restorative, aesthetic 191
 restorative, reinforced 192
 sealing, for water balance 177
 selection of 158
 setting reactions 173, 176
 solubility 187
 temporary restoration 303
 thermal response 189
 translucency 190
 transitional restoration 303
 water balance 176
- Gum, chewing
 sugar free 107
 with CCP-ACP 117
- ## H
- Hand instruments
 gingival margin trimmers 142
 spoon excavators 142
- Handpieces
 noise from 120
- HEMA (hydroxyethyl-methacrylate) 166, 201
- Hydroxyapatite
 acid ion interaction 25
 conversion to fluorapatite 39
 demin./remin. cycle 25
- Hypersensitivity
 cervical 50
- ## I
- Incremental buildup 276
- Indirect pulp therapy 300
 A.R.T. technique 304
 provisional restoration 303
- Indirect restorations 158
- Infected layer 301
- Inorganic fillers 201
 macrofillers 202
 microfillers 202
- Intercuspal relationships 324
- Interproximal attrition 52
- Ion exchange mechanism 147
- ## L
- Lactobacillus* 74
- Lamination technique
 amalgam 236
 composite resin 196
 glass-ionomer 195
 principles 194
- Lasers 133
 diagnosis of caries 32
 safety measures 137
- Lifestyle 84
- Light activation
 composite resin 203
 light source 213
- Linear surface speed 124
- Lining cements 193
- Loss of gloss test 173
- Luting cements 182
 postinsertion sensitivity 186
- Lutz & Phillips classification 202
- ## M
- Macrofillers 202
- Marijuana 103
- Matrix 67
- Mercury
 amalgam allergy 237, 238
 amalgam tattoo 238
 elemental 235
 environmental 239
 hygiene 238, 240
 inorganic 236
 organic 236

vapour	235
Microfillers	202
Microleakage	33, 205
Mouth rinses	
chlorhexidine	44, 102
fluoride	39

N

Nd-YAG laser	133
Nicotine intake	102
Noise – annoyance	120

O

Occlusal harmony	316, 332
anterior guidance	325
balancing side interference	330
vertical dimension	334
working side interference	325
Odontoblast	
cell body	2
dentine formation	4
reparative dentine	6
Oral biofilm	2, 4, 63
Oral clearance	43, 86
Oral hygiene	
abrasion due to	50
first daily clean	37
frequent daily clean	37
second daily clean	37
Orthophosphoric acid	212

P

Parafunction	51
Periodontal disease	314
Periodontal ligament	9
Periodontitis	315
Pins	151-154
Pipe smoking	48
Pits and fissures	249
Plaque (see Biofilm)	63, 73
Polyacid modified composite resin	200
Polyalkenoic acid	165
Polymerisation	
composite resins	203
contraction	205

Posselt's diagram	325
Proximal contour	321
Pulp capping	304
Pulpitis	302
irreversible	291
reversible	17
Pulpotomy	308

Q

Quartz fillers	201
----------------	-----

R

Radiographs	32
Radiopacity	
glass-ionomer	200
Remineralisation	25
Replacement dentistry	245
Resin bonding agents	
amalgam	232
to dentine	207, 212
to enamel	206, 212
Resin fissure seals	252
Resorption, ideopathic	16
Restoration, failure	
amalgam	355
bulk failure	355
composite resin	355
glass-ionomer	353
marginal failure	353
Retention	147
mechanical v chemical	146
with amalgam	151
with composite resin	147
with glass-ionomer	147
Retentive grooves and ditches	151
Rotary cutting instruments	
annoyance factor	120
classification	120
cutting efficiency	121
design principles	120
diamond	121
linear surface speed	124
load application	126
lubrication	125
speed groups	125
standard kit	128
tungsten carbides	121

Rubber dam	
instruments	159
placement	160

S

Saliva	
assessment of	84
bacterial flora	74
bacterial transfer	98
bicarbonate buffering	86
buffering test	73
components of	85
control of flow	89
diurnal variation	69
hormonal variations	92
functions of	82
flow rate	25, 39, 89
medications, effect of	91
oral clearance	87
protective factors	38
reduction in	38, 93
remineralisation	87
resting	70
stimulated	72
unstimulated	70
Salivary glands	
buffer systems	73, 86
dysfunction	89
enzymes	85
minor	69
proteins	87
sublingual	69
submandibular	69
Silane coupling agent	201
Site 1 lesions	249
Site 2 lesions	258
Site 3 lesions	278
Sjögren's Syndrome	93
Smear layer	147, 149, 294, 180
SnF ₂ solution	42
Sodium fluoride (NaF)	42
Sodium monofluorophosphate	42
Soft drinks	
acid level, -pH	98
caffeine in	100
Spoon excavators	141
Split cusp	331

Streptococcus mutans 74, 96
sobrinus 74
 Strontium 164, 185
 Sucrose 92, 95
 Sugar
 intake 104, 107
 substitutes 99, 102

T

Tannic acid 180
 Tartaric acid 168
 Thegosis 52
 Thermal coefficient of expansion
 amalgam 225
 composite resin 200
 glass-ionomer 189
 Thieleman's diagonal law 328
 Tomes fibres 5
 Tooth fracture 350
 cusp 348
 enamel flaking 55
 extreme wear patterns 56, 58
 reduction 55
 Toxicity 291
 Traffic light-matrix system 66, 78
 Transillumination 258
 Transitional restoration 303
 Trichloroacetic acid 280, 281
 Tungsten carbide burs 121

U

Urethane dimethacrylate 201

V

Varnishes 295
 Vertical dimension, stability 57, 334
 Vomiting (chronic) 100

W

Water fluoridation 40
 Wear patterns 56
 White spot lesion 31, 32
 Wine – effect on teeth 84, 53

X

Xerostomia 89, 91
 Xylitol gum 92, 95

Y

Ytterbium 201

Z

Zinc oxide and eugenol 296, 304
 pulp inflammation 297
 pulp protection 307
 temporary restoration 303
 Zinc phosphate 184
 as a luting agent 182