(Shanghai Journal of Stomatology, China 2006)

Minimal Invasive Dentistry: A Review and Update

Brostek AM, Bochenek AJ & Walsh LJ.

Dr. Andrew M. Brostek. BSc, BDSc
Dr. Andrew J. Bochenek. BDSc, LDS, FRACDS, FPFA
Professor Laurence J. Walsh. BDSc, PhD, DDSc, GCEd, FFOP (RCPA), FICD, FADI, FPFA

Biosketch:

Drs. Andrew Brostek and **Andrew Bochenek** are in general dental practice in Perth, Western Australia. Both have been using a minimal invasive dentistry approach in their clinical practices for many years and have been early adopters of technologies which support this approach to patient care, including air abrasion, lasers and ozone. They have taken a holistic and comprehensive approach to patient care which exemplifies the approach described in this article.

Dr. Laurence Walsh is Professor of Dental Science at the University of Queensland in Brisbane, Australia. He has worked clinically in the field of special needs dentistry for 20 years and published extensively on preventive and minimal intervention dentistry. Through his research work, he has contributed to the development and clinical assessment of a number of the technologies which underpin minimal intervention dentistry which are now in clinical use in Australia and elsewhere.

Affiliations:
Dr. A.M. Brostek and Dr. A.J. Bochenek, part-time lecturers, School of Dentistry, Oral Health Centre, University of Western Australia, 17 Monash Av. Nedlands, WA 6009.

Professor L. J. Walsh, Professor of Dental Science, School of Dentistry, Head of School, University of Queensland, 200 Turbot Street, Brisbane, QLD 4000.

Address for Correspondence: Noranda Dental Surgery
5 / 36 Benara rd Noranda, Perth, Western Australia, 6009.

Phone: +61 892752186
Fax: +61 892753292
Email: andrew@mylaserdental.com

Abstract:

The term Minimal Invasive Dentistry can best be defined as the management of caries with a biological approach, rather than with a traditional (surgical) operative dentistry approach. Where operative dentistry is required, this is now carried out in the most conservative manner with minimal destruction of tooth structure. This new approach to caries management changes the emphasis from diagnosing carious lesions as cavities (and a repeating cycle of restorations), to one of diagnosing the oral ecological imbalance and effecting biological changes in the biofilm. The goal of Minimal Invasive Dentistry (MI) is to stop the disease process and then to restore lost tooth structure and function, maximizing the healing potential of the tooth. The thought process which underpins this new minimal invasive approach can be organized into three main categories:

- **Recognize** = Identify patient caries risk
- **Remineralize** = Prevent caries and reverse non-cavitated caries
- **Repair** = Control caries activity, maximize healing and repair damage

The disease of dental caries is not just demineralization, but a process of repeated demineralization cycles caused by an imbalance in the ecological and chemical equilibrium of the biofilm /tooth interface (the Ecological Plaque Hypothesis). Dietary and lifestyle patterns, especially carbohydrate frequency, water intake and smoking, play an important role in changing the biofilm ecology and pathogenicity. Tools for chairside assessment of saliva and plaque, allow risk to be assessed and patient compliance to be monitored. The remineralizing properties of saliva can be enhanced using materials which release biologically available calcium, phosphate and fluoride ions (CPP-ACP and CPP-ACFP). Use of biocides can also alter the pathogenic properties of plaque. Use of these MI treatment protocols can repair early lesions and improve patient understanding and compliance. This review article introduces some of the key concepts and practical aspects of Minimal Invasive Dentistry.

Keywords: Minimal Invasive Dentistry, CPP-ACP Recaldent®, biomimetic

Introduction

The term “Minimal Invasive” (MI) Dentistry can be defined as the management of dental caries with a biological/medical approach, as compared to traditional operative dentistry which is based on surgical and mechanical principles. It focuses on the end results of the disease (cavitations), but does not address the preceding disease process.

The MI approach has been developed because of the many failures associated with traditional operative dentistry. Traditional diagnosis involves detecting carious lesions at a late stage (frank cavitation) and then restoring these cavities, usually with a G.V. Black cavity preparation and the use of amalgam or composite. Unfortunately, removing the evidence of the disease does not stop the disease process from continuing at other sites. In other words, placing dental restorations does not ‘cure’ caries. No restoration can be considered permanent. In fact, unless the ongoing causes of the disease are addressed, restoration failure from recurrent caries is highly likely, and this premature failure results in on-going restoration replacement, with resulting larger and larger cavities, and weakening of tooth structure. To overcome this, the caries disease process needs to be managed in partnership with the patient and over the lifetime of the patient.

The Dental Caries Process

Dental caries is a complex diet and saliva- modified infectious multi-factorial bacterial disease **(Walsh, 2000 A),** which results from alterations in the plaque biofilm and the ionic interactions between saliva, plaque fluid, and dental enamel **(Fejerskov & Kidd, 2003)**. Recognizing that the formation of cavities in the teeth is an end result of the dental caries disease process, the goal of Minimal Invasive Dentistry is to arrest (or reverse) this disease process, to maximize the healing potential of the dental hard tissues and then to restore lost structure and function with biomimetic materials.
The dental plaque biofilm which forms continuously on tooth surfaces is a critical factor in the disease process, because it can mature to become a complex three dimensional structure which allows the persistence of the acidogenic and aciduric bacterial species (mutans streptococci) which initiate the caries process.

This microbial biofilm is a normal structure, which forms on all surfaces exposed to the oral fluids. The first stage of its formation is the deposition of a layer of salivary glycoproteins (pellicle), onto which bacteria adhere in a well defined sequence. As the film matures, the blend of bacteria and inter-bacterial polysaccharide gel matrix provides a protective environment for the bacteria within the plaque. Plaque is a complex mosaic of interrelating bacterial micro-environments, and can be described as a sophisticated community *(within an established infrastructure).*

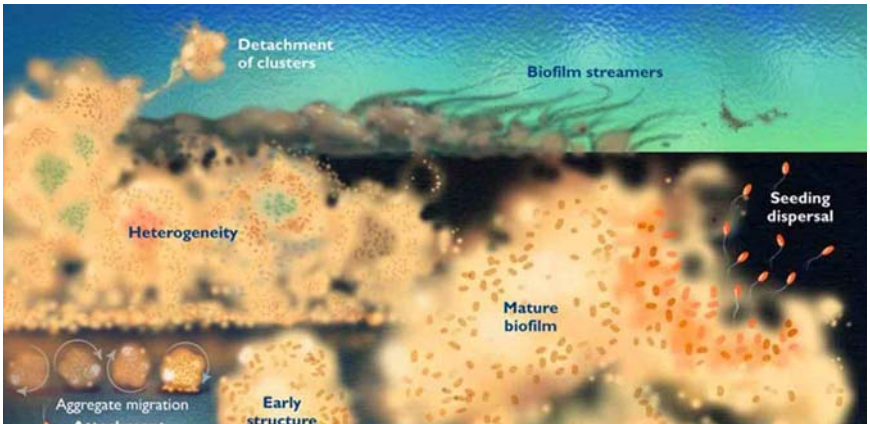


Figure 1. A schematic illustration of biofilm development – Courtesy of Dr. Peg Dirckx, Montana State University Centre for Biofilm Engineering, USA.

The age and thickness of the plaque biofilm influences the cariogenicity of the plaque. Mature plaque has a lower oxygen potential in its deepest aspects, which facilitates the growth of facultative anaerobes such as mutans streptococci. These microorganisms can produce both intra- and extra-cellular polymers which are a storage vehicle for carbohydrates, allowing extended periods of fermentation between meals. By suppressing the growth of non-pathogenic bacteria and enhancing the growth of aciduric bacteria, frequent intake of dietary fermentable carbohydrates and acidic drinks place ecological pressure on the plaque biofilm. Naturally, peroxide rinses (which raise oxygen levels) and sodium bicarbonate rinses (which elevate pH) will have a positive and beneficial effect on the supragingival plaque ecology.

A key fact is that repeated conditions of low pH (rather than sugar availability per se), select for mutans streptococci, as well as lactobacilli. The dominance of aciduric (acid-tolerant) and acidogenic (acid-producing) bacteria results in shifts in the demineralization-remineralization balance. This well recognized event has been termed the 'Ecological Plaque Hypothesis' **(Marsh, 2003)**.
Implicit in this hypothesis is the concept that dental caries can be prevented, not only by directly inhibiting the growth of these pathogens, but also by interfering with the environmental factors driving the selection and enrichment of these bacteria. Thus, a more holistic approach can be taken in disease control and management strategies. Examples of applying this "ecological manipulation" approach in clinical practice include:

- Regularly disrupting plaque (through mechanical oral hygiene) to reduce its thickness
- Avoiding dietary patterns such as frequent snacking or drinking fizzy drinks which place ecological pressure on the "healthy" (non-pathogenic) plaque bacteria
- Discouraging the growth of aciduric bacteria (and encouraging remineralization) by increasing the pH of the oral fluids using sodium bicarbonate
- Avoiding habits which lower oxygen tension (such as smoking)
- Elevating oxygen levels by using hydrogen peroxide mouthrinses.

It follows from the Ecological Plaque hypothesis, that such manipulation of the oral environment will favour the development and persistence of "good" (non-cariogenic) dental plaque. This plaque will have limited fermentation capabilities, and any glycolysis will be incomplete, resulting in the production of weak organic acids (such as acetic, propionic, and butyric acids). These weak organic acids play a vital role in buffering pH changes in the plaque biofilm and in so doing they help protect the tooth from demineralization by strong organic acids.

In contrast, "bad" plaque which is formed in a highly cariogenic environment will ferment carbohydrates readily, with glycolysis producing strong organic acids, (such as lactic, formic, and pyruvic acids), which will readily demineralize enamel (**Reynolds & Walsh, 2005**).

Streptococcus mutans is a normal plaque inhabitant; however it is the principal bacterial species responsible for initiation and progression of dental caries in infants, young children and adults. It is both aciduric and acidogenic and can produce the extra-cellular glucans involved in enhancing plaque mass, changing the diffusion properties of the plaque matrix and further promoting colonization. *Streptococcus sobrinus* is another important bacterial initiator of dental caries, whereas Lactobacilli are recognized as secondary colonizers of cavitated lesions and are mainly responsible for caries progression within dentine. The preferential sites for colonization of *Streptococcus mutans* are retentive areas of teeth (such as fissures and proximal surfaces) and high levels at these sites are an indicator of high caries risk (**Loesche, 1979; Krasse, 1984**).

Transmission of *S. mutans* from mother to child by salivary transfer has been demonstrated in various studies, through behaviours such as kissing, sampling of food, and sharing spoons and other utensils. This process has recently been shown to occur in pre-dentate infants, in whom by 6 months of age up to 60% of infants have been infected with *S. mutans*. By the age of 24 months, 84% of the infants have become infected **(Wan et al, 2001)**.

Detailed studies of these transmission events in the early years of life have shown that early colonization and high levels of *S. mutans* in infants and young children is strongly associated with enhanced risk for early childhood caries. Behavioural and lifestyle factors which also contribute to this heightened caries risk include**:**

- 'Grazing' eating patterns, with a high frequency of intake of sucrose and other fermentable carbohydrates
- Use of sweetened agents (such as syrups) on pacifiers
- Intake of acidic fruit juices, cordials and soft drinks **(Wan et al, 2003)**.

Since *S. mutans* transmission and infection of children is inevitable, improving parental dental health with a community based education and maintenance approach is important to help reduce the dental caries rate in children. Regular (daily) use of chlorhexidine gel has been shown to interfere with acquisition of mutans streptococci in infants and young children **(Wan et al, 2003, Twetman & Grindefjord, 1999)**.
Chlorhexidine has been used for the past 35 years in the treatment of caries with varying degrees of success. While the literature is ambivalent on the success of chlorhexidine when used in isolation to prevent dental caries, its performance as an antimicrobial against *Streptococcus mutans* is very consistent and powerful. For adult patients, current recommendations for chlorhexidine usage in high risk patients are for weekly use of a 0.2% gel (0.5 mL) or mouthrinse (20 mL) once per week, to suppress levels of mutans streptococci within the dental plaque biofilm **(Walsh, 2000; Anderson, 2003)**.

A number of organic acids produced by mutans streptococci and other acidogenic bacterial species initiate the process of demineralization of enamel and root surfaces. The fermentation process (glycolysis) which produces these organic acids is a direct result of the ecological conditions in the mature plaque biofilm (low pH and low oxygen tension). The type and frequency of fermentable carbohydrate in the diet are critical factors, since these drive the processes of bacterial fermentation **(Reynolds & Walsh, 2005)**. The

organic acids produced from this normal metabolic activity of bacteria deep within the plaque biofilm (and thus close to the enamel surface), can cause the plaque pH to drop below 5.0 within 1-3 minutes following a sucrose exposure **(Kidd, 2005)**. A newly developed test (the Plaque-Check test from GC Corporation) allows this process to be evaluated chairside in real time using the patient's plaque and also demonstrated to patients as a motivational strategy. This test is an important development, because previously this normal metabolic activity of the biofilm could not be seen or assessed by the clinician. In fact, it was easy for the dentist to forget that the biological activity of fermentation occurs within the plaque biofilm (**Kidd, 2005**).

Repeated falls in the plaque pH below the critical pH of enamel result in demineralization of the tooth surface (Figure 2). In contrast, saliva can buffer this acid attack and can provide replacement minerals as part of the natural repair process. This replacement of minerals through the biofilm into the tooth is called remineralization. The process of remineralization can be enhanced dramatically through the use of bio-mineralizing materials such as casein phosphopeptides-amorphous calcium phosphate (CPP-ACP) (Figure 3). The development of these materials has heralded a new era in prevention, since their use clinically can result in predictable reversal of enamel white spot lesions (**Shen et al, 2001**). This has moved the "goalposts" for the treatment of white spot lesions from caries arrest to caries reversal.

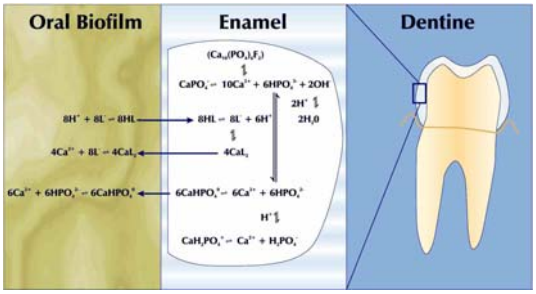


Figure 2. The process of enamel demineralization. From Reynolds & Walsh, 2005. Courtesy of Dr. G. Mount.

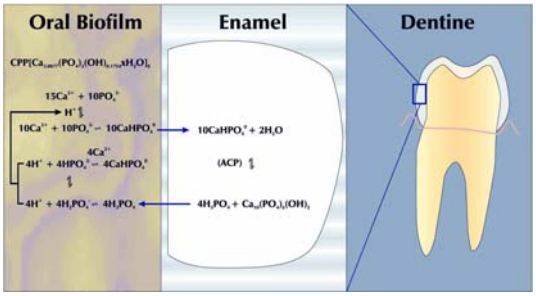


Figure 3. The process of enamel remineralization using CPP-ACP. From Reynolds & Walsh, 2005. Courtesy of Dr. G. Mount.

The cumulative results of the demineralization – remineralization cycle dictate whether or not carious lesions occur. Cavitations develop in enamel once the net loss of mineral has exceeded the point where the process of re-precipitation of ions from the subsurface reaches the point of exhaustion, and catastrophic collapse of the surface occurs. At this point, the white spot becomes a cavity. This represents a fundamental turning point in the disease process, since bacteria are now able to gain access to the internal aspects of the

tooth structure and are protected from mechanical oral hygiene by the walls of the newly formed cavity.
Having now addressed the bacterial nature of the caries process, the foundations of a minimal invasive approach to dental caries can now be presented as three steps:

- **RECOGNIZE** = Identify patient caries risk
- **REMINERALIZE** = Prevent caries and reverse non-cavitated early lesions
- **REPAIR** = Control caries activity and repair damage

To further detail this approach, these can be expanded into:

1. RECOGNIZE

- **Caries history and early lesions**
- **Biofilm assessment**
  Plaque Checking and pH analysis
  Bacterial testing
- **Primary Caries Risk Factors**
  1) **Diet** – Dietary assessment
  2) **Fluoride**
  3) **Saliva** – Saliva testing
- **Modifying Risk Factors - Lifestyle analysis – Medications and Disease**

2. REMINERALIZE

- **Fluid balance**
- **Dietary cariogens**
- **Habits –**Smoking, Caffeine and alcohol
- **The role of Biocides –** Flouride, Chlorhexidine and Xylitol
- **Biomineralization – the use of chemicals: CPP-ACP (Recaldent®)**
- **Mechanical disruption of plaque** - Tooth brushing / Flossing / Tongue scraping
- **Increase in intra-oral pH**

3. REPAIR

- **Fissure sealing and tooth surface protection**
- **Biomimetic restorative materials - Internal healing of dentine**
- **Minimal operative techniques**
  Micro-preparation techniques:
  Micro-preparation and Fissurotomy burs, Chemomechanical methods, Sonic tooth preparation, Air abrasion and Lasers
- **The role of disinfection in arresting dental caries**
  Ozone – *Chemistry, Indications*
  Photo-activated disinfection (PAD) – *Mechanism of action*

1. RECOGNIZE

- **Caries history and early lesions**

Past caries experience can be regarded as one useful predictor of tooth caries risk, particularly amongst patients under the age 16 years of age. "The clinical implication is that past caries experience can be used as one factor to predict future caries risk" **(Mickenautsch et al, 2006).** This relationship exists because of the persistence of bacterial, dietary, salivary and lifestyle factors over time.
An important part of the clinical visual examination is a determination of the type and number of caries lesions. Early 'white spot' active lesions which are located beneath thick plaque deposits are indicative of the level of current caries activity. White spot lesions which are supra-gingival and not covered by plaque (associated with healthy gingival tissues or gingival recession) are likely to be arrested and thus "historical" in nature. As these lesions trap stains and chromogens from the diet over time, they may become dark as well as hard.

As already mentioned active white spot lesions are non-cavitated and are reversible with conservative remineralization treatments using CPP-ACP.

Clinical examination for dental caries should also include local predisposing factors for plaque retention such as:

- Newly erupted teeth (partial coverage by soft tissues, and immature enamel)
- Exposed root surfaces (recession)
- Crowded teeth
- Deep occlusal fissures or other "natural" retentive sites
- Retentive sites caused by dental treatment (such as rest seats for chrome cobalt partial dentures).

The complex relationship between the biofilm and the major caries risk factors is shown in Figure 4. These factors relate directly or indirectly to the continuous changes occurring in the ecological equilibrium of the dental biofilm.

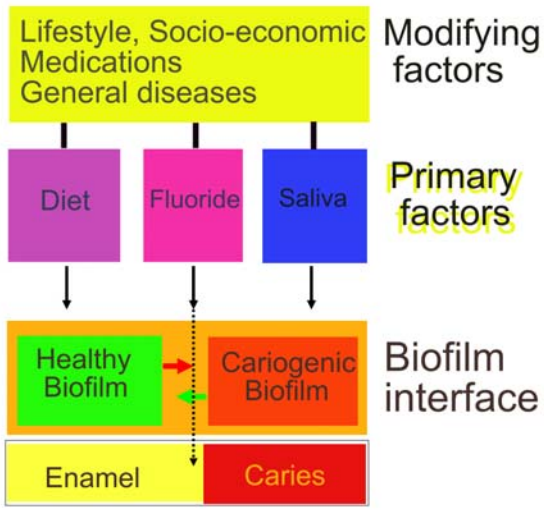


Figure 4. Caries risk factor diagram - Source: Dr. Hien Ngo, Australia.

The PRIMARY risk factors are Diet, Fluoride and Saliva and whereas the MODIFYING factors are patient lifestyle choices, socioeconomic factors, patient medications and the effect of general diseases.

- **Biofilm assessment :**

Plaque Checking and PH analysis

The development of effective screening methods for plaque cariogenicity now allows improved assessment of individual caries risk and for monitoring of the effect of preventive measures. Left undisturbed, the dental plaque biofilm thickens with time and undergoes maturation with the development of complex features such as stacks, water channels and waste channels **(Marsh, 2003)**. As facultative organisms such as mutans streptococci increase numerically, they create a more complex biofilm physical structure by synthesizing extra-cellular polymers which give the plaque a sticky, gel-like nature. The thicker the biofilm, the more difficult it is for the saliva to buffer the acids produced deep within the biofilm. As already discussed, this mature cariogenic biofilm encourages growth of aciduric and acidogenic bacteria. These bacteria ferment carbohydrate substrates, resulting in the production of a large number of organic acids. For this reason, clinical assessment should not focus on detecting any one particular species of bacteria or a specific acid, but rather the entire plaque biomass and the net result of fermentation after sucrose challenge.

Rapid assessment of the results of plaque fermentation can now be carried out easily at the chairside with the **Plaque Check + pH Test kit (GC Corporation, Japan)**, see Figure 5.

Figure 5. Plaque Check + pH test kit (GC Corporation, Japan)

The test kit allows two types of assessment to be carried out. The first is a 2-tone disclosing reagent that is used to stain plaque in the mouth. Different retention patterns for the two dyes allow the user to determine whether the plaque is “mature” (and thus capable of fermentation) or newly formed (thin) as in Figure 6. The second test in this kit measures the pH of the total biomass of plaque both before and after challenge with a sucrose solution. A low pH result from this fermentation test gives an indication of a high level of acidogenic bacteria in the plaque. In this, it is important to collect the sample on the disposable collection instruments provided, which allow the clinician to ‘scoop’ up

the plaque from the tooth surface with the applicator tip. This preserves the integrity of the plaque biomass.

Both tests give visible results which are used to educate the patient about the importance of daily effective tooth brushing and the dietary patterns which influence the cariogenicity of their plaque. Studies of plaque fermentation after sucrose challenge have been undertaken over 40 years, however the technology used (intra-oral micro-electrodes) is not suitable for everyday clinical use. The Plaque-Check test makes the pH results easily interpreted using a traffic-light series of colour changes in a dye. There is a strong correlation between the levels of cariogenic bacteria in dental plaque and its acid production as assessed using this test **(Matsumoto & Yoshii, 2005)**.

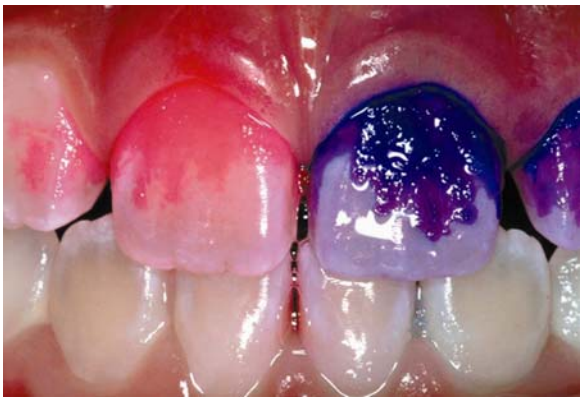


Figure 6. Two tone staining of mature plaque (purple) versus newly formed plaque (pink) - Dr. Hien Ngo, Australia

This plaque test kit is an important development, because it allows chairside clinical testing by the general dentist to give an instant biofilm analysis (evidence of "bad" cariogenic plaque) and at the same time can be used to educate the patient about their individual plaque risk. Patient hygiene compliance may improve if the patient is provided with an inexpensive plaque disclosing product, to allow the patient to repeat the assessment of plaque levels as part of their home-care oral hygiene activities.

Another related approach is to examine the levels of lactic acid produced by the aciduric bacteria (sampled from the tongue) using an enzymatic test impregnated onto strips **(ClinPro™ Cario L-Pop™ from 3M-Espe Seefeld, Germany)**.

Bacterial testing

Salivary samples contain bacteria from plaque and thus samples of stimulated saliva can be used as a surrogate for assessing plaque samples directly. Bacterial testing can be undertaken using microbiological culture-based tests (such as the CRT from Vivadent), which use semi-specific growth media and require a 48 hour incubation period. An alternative approach which is specific to an individual species of bacteria is the use of monoclonal antibodies for a chair-side rapid solid-phase immunoassay (such as the **Saliva Check SM from GC Corporation, Japan**). High *S. mutans* levels in the culture-based or immunoassay tests correlate with high *S. mutans* levels in the mouth and an associated high cariogenicity of the plaque. A high lactobacillus count reflects a high frequency of dietary carbohydrate intake and the presence of cavitated lesions. The latter

explains why high plaque lactobacillus levels have been associated statistically with caries progression. Thus, while levels of mutans streptococci are informative regarding the likelihood of caries initiation, high salivary levels of lactobacilli cannot in themselves be regarded as a predictor of caries risk, except when there are also high levels of *S. mutans* in the oral cavity. Chairside lactobacillus bacterial tests cannot therefore be used on their own to assess caries risk.

- **Primary Caries Risk Factors**

To enable a comprehensive assessment of oral health, primary factors such as dietary intake of acidic foodstuffs and fermentable carbohydrates, even including non-prescription (traditional) medicines, must be evaluated.

1) Diet

There is a positive correlation between the frequency of refined carbohydrate intake and dental caries as confirmed by the Vipeholm, Hopewood House and other classic studies in cariology in the 1940's and 1950's **(Sreebny et al, 1982)**. **Stephan in 1944** first showed that dental plaque exposed to sucrose would rapidly produce acids, with a subsequent rapid drop in pH, followed by a gradual recovery toward the baseline plaque pH. It is well accepted that there is a direct causal association between the pattern of strong acids produced in response to sucrose and caries activity.
With a number of different acidogenic bacteria involved, the final shape of the Stephan curve represents the balance between the different metabolic outcomes of this bacterial activity. Figure 7 presents Stephan curves which show the variation in pH in subjects with different caries activity. The reason for these inter-subject differences is the varying level of acidogenic organisms ("bad" plaque) between these individuals.

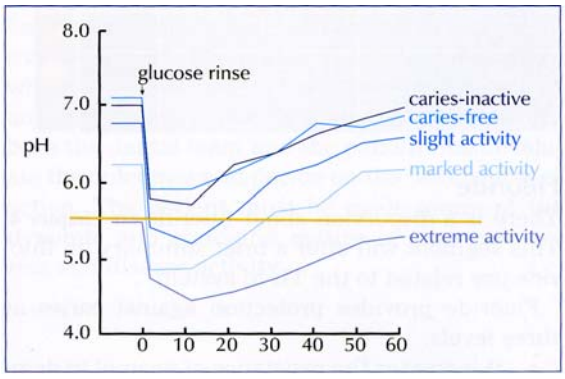


Figure 7. Changes in plaque pH after a 10% glucose rinse (adapted from **Nikiforuk, 1985**) Courtesy of Dr. G. Mount.

A high frequency of intake of fermentable carbohydrates (especially sucrose, which is much more cariogenic than glucose and other sugars) will increase the caries rate. Carbohydrate products that are sticky, retained for long periods in the mouth, or consumed with high frequency have a higher cariogenicity than foods that are eliminated quickly from the oral cavity. Human pH telemetry studies have consistently shown that

subjects who consume only three meals a day have short periods of demineralization which are counteracted by long periods of remineralization.
In contrast, with frequent meal or snack periods (grazing behaviour), demineralization cycles will predominate, leading to tooth mineral loss (Figure 8).

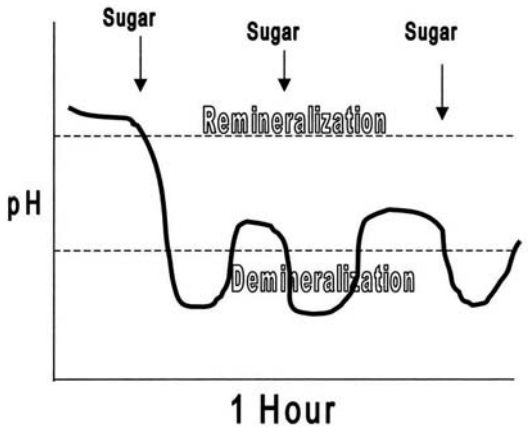


Figure 8. Example of fluctuations in plaque pH with a high frequency of carbohydrate intake, for example from sipping a sugar-containing soft drink over a one hour period. Source: Tinanoff N, 2005.

To improve the usefulness of dietary assessment, patient questioning should address patterns of snacking or 'grazing' behaviour. Regrettably, lifestyle changes in many countries have resulted in dramatic increases in the consumption of sugar-containing carbonated ("fizzy") drinks (such as cola drinks and energy drinks), which have a very low pH (2.0-3.5) and a high sucrose content (11-14% by weight). These and other dietary sources of acid such as fruit juices have a pH lower than the critical pH of apatite (pH 5.5), which may contribute not only to an increased caries rate but also to dental erosion (Figure 9).

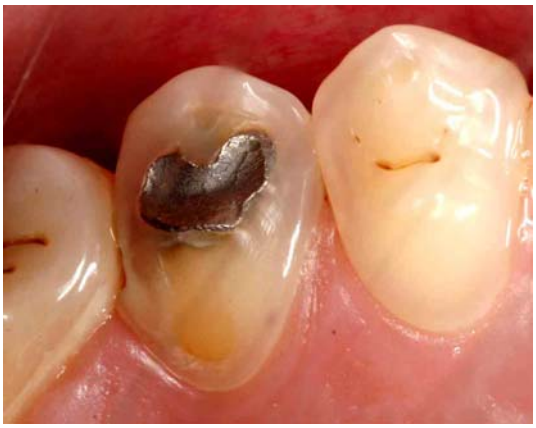


Figure 9. Patient with high acid drink intake with resultant dental erosion and accelerated tooth wear.

The importance of the repeated dietary intake of these low pH (acidic) foods is that the plaque ecology will change over time, with the ecological pressure favouring aciduric (acid-tolerant) bacteria. This raises the cariogenicity of the plaque as the number and proportion of S. mutans increases, with corresponding drops in the growth and metabolism of bacterial species not associated with caries **(Walsh, 2005)**.

For similar reasons, endogenous acid exposure due to eating disorders, chronic gastric reflux diseases, or reflux caused by pregnancy or medications, will lower the intra-oral pH, and promote higher levels of *S. mutans* in the plaque biofilm, thus increasing caries risk (as well as causing dental erosion).

The addition of caffeine to softdrinks and energy drinks, as well as high tea and coffee consumption, will also further increase caries risk by causing subclinical dehydration which reduces saliva production and pH, particularly at rest. Further risks associated with high sucrose consumption include the development of Type 2 diabetes mellitus and resistance to insulin. Diabetes mellitus is a leading cause of kidney failure and blindness, and a major risk factor for death from cardiovascular disease or stroke **(Walsh, 2005)**.

The concept of promoting general health by controlling a small number of risk factors can have a major impact on a large number of diseases in a population at a lower cost **(Fejerskov, 2003)**.
The literature also supports the view that patients' sugar intake can be useful for caries risk prediction (especially in conditions of low fluoride exposure). The clinical implication is that "Dietary history with a focus on sugar intake remains a useful tool for assessing caries risk" **(Mickenautsch et al, 2006)**.

Dietary Assessment:
Some of the key factors are the frequency of sugar exposures and acid exposures between meals, where more than >4 daily episodes in either category is very favorable for the initiation and progression of dental caries. Patients should keep a 5 day record of everything they put into their mouth, so that a risk profile can established. An excellent risk charting system, using a 'traffic-light' analogy to allow easy patient education and recording has been established **(Ngo & Gaffney, 2005)**. Patient diet history questions should also include: flavoured milk, icecream, yoghurt, chocolate, bakery items (cakes, biscuits), sweet confectionary (candy, jellies), traditional medicines (e.g. Royal Jelly), cough lozenges, fruit bars, dried fruits, black cola and other soft drinks, energy drinks, and fruit juices.

2) Fluoride
Low concentrations of fluoride ions are present in saliva and accumulate in dental plaque when it is thin. These low levels (0.1 ppm and less) are consistent with healing and remineralization of early caries lesions (**Edgar et al, 1994; Pearce et al, 1995**). The reason for this is that the natural repair process of remineralization occurs when calcium and phosphate from saliva together with fluoride enter the subsurface region of a white

spot lesion and form a new veneer on the existing crystal remnants within the lesion (**Featherstone, 2004)**. Because the dynamic balance between demineralization and remineralization determines the end result, dental caries is reversible if detected early enough.
As well as enhancing remineralization, higher levels of fluoride can influence bacterial metabolism. Fluoride levels of up to 40 ppm can inhibit glycolytic fermentation of sugars by plaque bacteria, while high fluoride products (such as high fluoride toothpastes and gels) can impair energy utilization and thus acid production via the fermentation process, with its effects on the two enzymes enolase and H+/ATPase **(Bradshaw et al. 1990; Bowden, 1990; Wahab et al, 1993; van Loveren et al, 1995)**. High levels of fluoride (0.16-0.3 mol/L) will kill bacteria **(Bowden, 1990)** and this can be achieved through topically applied professional products such as gels and varnishes.

3) Saliva
Saliva is a complex biological fluid made up of a mixture of the secretions of the major and minor salivary glands as well as some material from the gingival sulcus. Approximately 99% of the volume is water, with the addition of bicarbonate ions, enzymes including amylase, immunologic factors (IgA), enzymic peptide and chemical mediators, plus mucins and calcium.

Its functions include:

- Lubrication and oral clearance
- Limited pre-digestive functions (Amylase, lipase, proteases, nucleases)
- Assisting taste sensation (as an ion solvent and gustin)
- Maintaining oral health via growth factors to promote healing
- Buffering of plaque acids and buffering weak acids from food and drink
- Buffering brief strong acids, from reflux and vomiting
- Acting as a reservoir for calcium, phosphorus and fluoride ions used in remineralization
- Control of the oral microflora (immunological factors) **(Walsh, 2005)**.

A reduction in salivary flow rates (xerostomia) or changes in the quality or ionic properties of saliva, are responsible for many other oral and dental conditions, as well as dental caries. For example, with regard to lubrication, the ratio of water to mucins in the saliva has a dramatic effect on viscosity, particularly for the mucin-rich secretions from the submandibular salivary glands. These mucins are essential for lubrication and protection of oral mucosal tissues during normal function. Furthermore because saliva coats and protects the oesophageal mucosa, a lack of saliva can increase the likelihood of erosive oesophageal inflammation.

Increased salivary flow and pH results in an alkaline oral environment, leading to mineralization of plaque foci and the formation of dental calculus. In a high pH environment, the action of salivary phosphatase enzymes breaking down organic phosphates, allows the formation of highly ionized phosphate ions (PO_4^{3-}). Salivary flow is stimulated by taste and mastication, and as a consequence of enrichment with

bicarbonate, stimulated saliva has a higher pH and buffering capacity. With a parallel increase in calcium and phosphate levels, the diffusion gradients for these ions are altered in the plaque fluid, with an increase in remineralization of tooth structure.
The normal total flow rate (stimulated and unstimulated) is between 500 and 1,500 mL per day, while the average volume of resting saliva present in the oral cavity at any one time is only 1.0 mL (**Walsh, 2000**). There is extensive evidence that impaired salivary flow rates are related to an increase in caries risk.

From the point of view of preventing dental caries, the essential role of saliva can be considered to be -

- Clearance of cariogenic substrates, erosive and cariogenic acids
- A reservoir of calcium, phosphate and fluoride ions
- Saturation of the plaque/tooth interface changing the critical pH level
- A growth environment for microbiota.

Signs of xerostomia include –

- Increased plaque deposits
- Reduced antimicrobial action of saliva
- Recurring Candida albicans infections
- An increased caries rate
- Increased plaque pathogenicity (increased aciduric microorganisms)
- Increased tooth wear from softening of tooth structure in the acidic environment
- Cervical dentinal sensitivity and dental erosion **(Walsh, 2000; Walsh, 2002)**.

Xerostomia symptoms include taste dysfunction, halitosis, mucosal irritation (from foods and toothpastes), a lack of cohesion or lubrication for denture wearers and problems with chewing food and swallowing. The importance of these problems cannot be understated with regard to the impact on the quality of life of dental patients.

Saliva controls the tooth mineral equilibrium gain or loss in an erosive or cariogenic environment. Stimulating the salivary flow will increase the protective properties of saliva, including oral clearance, buffering ability and remineralization potential. Stimulation of saliva flow immediately after fermentable carbohydrate consumption (such as by sugar-free chewing gum), has been shown to neutralize plaque acids and to promote remineralization of incipient carious lesions **(Edgar et al, 1994)**.

Salivary flow is controlled by a complex of neurological mechanisms of mainly autonomic parasympathetic and sympathetic nerves, plus taste and tactile stimuli (Figure 10). As a result, the anxiety level of the patient will affect resting salivary flow rates, as will the time of day. Salivary flow decreases during sleep and increases during waking hours, reaching a maximum rate in the mid-afternoon. These patterns are relevant to obtaining accurate clinical testing of saliva flow rates.

Common causes of salivary dysfunction are:

- Negative fluid balance – mild subclinical dehydration decreases resting saliva flow rate and pH, while severe dehydration affects both resting and stimulated

flow rates (**Walsh et al, 2004)**. Diuretic agents such as alcohol and caffeine will influence fluid balance and can impact dramatically on resting salivary flow rate and pH. Sources of caffeine include coffee, tea, black cola soft drinks, and energy drinks. Caffeine addiction is a major problem in many societies.

- Emotional and psychological stress
- Smoking and tobacco chewing
- Drugs of abuse – such as heroin, opiates, cocaine, cannabis, amphetamines.
- Medications – a wide variety of drugs, including anti-depressants (TCA, SSRI, MAOI), anti-psychotics, anti-emetics, anti-Parkinsonian agents, decongestants and expectorants, anxiolytics and sedatives, diuretics, systemic bronchodilators **(Walsh, 2002; Newbrun, 1989)**.
- Salivary gland pathology – infection, inflammation, neoplasia, sialoliths, radiation of the head and neck
- Medical conditions such as Sjögren's syndrome and connective tissue auto-immune disorders, diabetes mellitus (I and II), HIV infection, chronic hepatitis C, cystic fibrosis, chronic renal failure, liver dysfunction, menopausal hormone imbalances, and chronic protein-energy malnutrition.

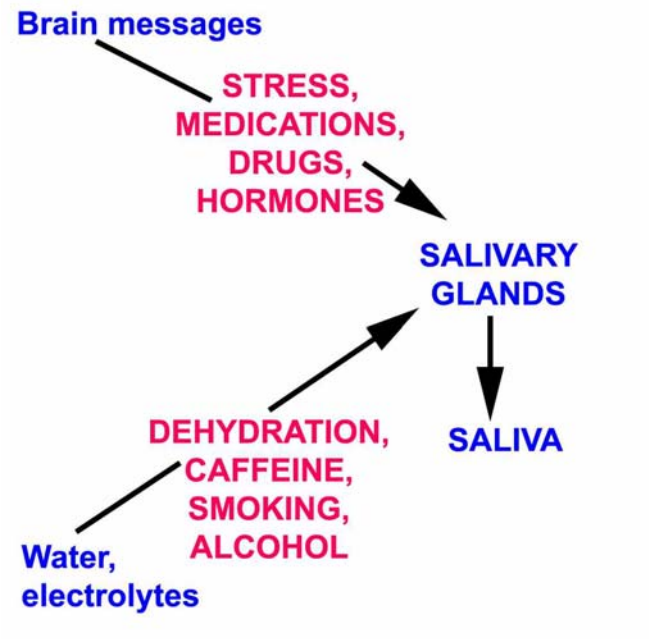


Figure 10. Schematic of saliva production. Modified from **Walsh, 2002**.

Saliva testing

The purpose of saliva testing is to identify whether there are salivary deficiencies in the patients presenting with dental diseases, discomfort or sensitivity.

The typical dental problems driven by an underlying salivary dysfunction include –

- Dental erosion
- Tooth wear – especially scalloping of occlusal surfaces
- Cervical dental hypersensitivity

- High rates of coronal or root surface caries

This testing is valuable for adults and teenagers in analyzing these typical problems and should be part of comprehensive assessment of new patients.

Which salivary parameters should be measured?
In healthy patients, measured saliva flow rates should be as follows:

Normal resting flow rate for pooled saliva = 0.3-0.4 mL/ minute Resting flow rate less than 0.1 mL/minute = Xerostomia
Normal stimulated flow rates = 1-2 mL/minute If less than 0.7mL/minute, salivary gland pathology is present.

Chairside saliva testing can be used to measure –
- pH – both at rest and when stimulated
- Flow rate – both at rest and when stimulated
- Viscosity at rest (either frothy, sticky or bubbly)
- Buffer capacity when stimulated

The **Saliva Check Buffer test kit (GC Corp. Japan)** (Figure 11), allows these six tests to be performed in a reproducible fashion (Figures 12, 13, 14 & 15). The results are scored on a summary sheet that is included in the patient's chart for future reference (Figure 16).

Figure 11. GC Saliva Check Buffer testing kit (GC Corp. Japan).

The following shows the six step saliva testing process.

Step 1: Test the degree of hydration

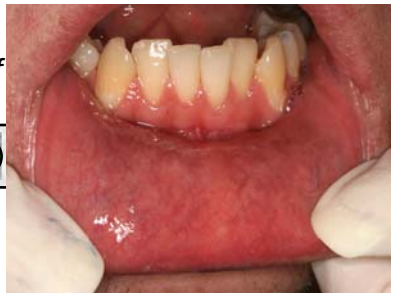
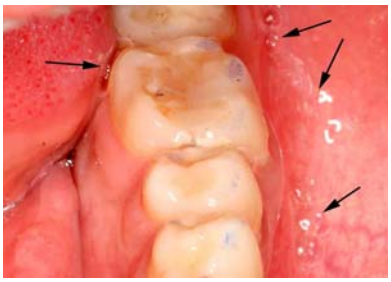
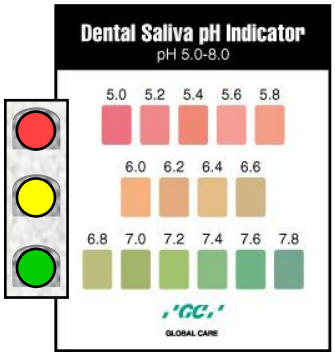


Figure 12. Lip hydration test

Step 2: Assess the resting viscosity. Figure 13. Viscous bubbles (arrows)

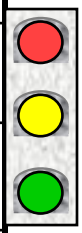


Step 3: Test the resting pH of resting saliva



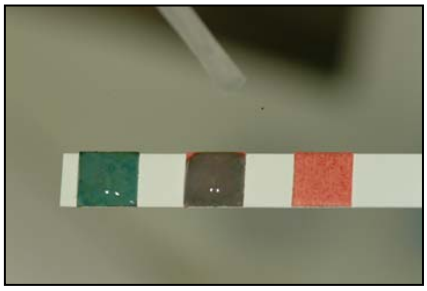
Step 4: Test quantity of stimulated saliva (5 mins)

Volume of Saliva	*Value*
<3.5 mL	very low
3.5-5.0 mL	low
>5.0 mL	normal



Step 5: Test the pH of the stimulated saliva

Step 6: Test the buffering capacity of stimulated saliva (quality) Figures 14 & 15.



Green = 4 points
Blue = 2 points
Red = 0 points

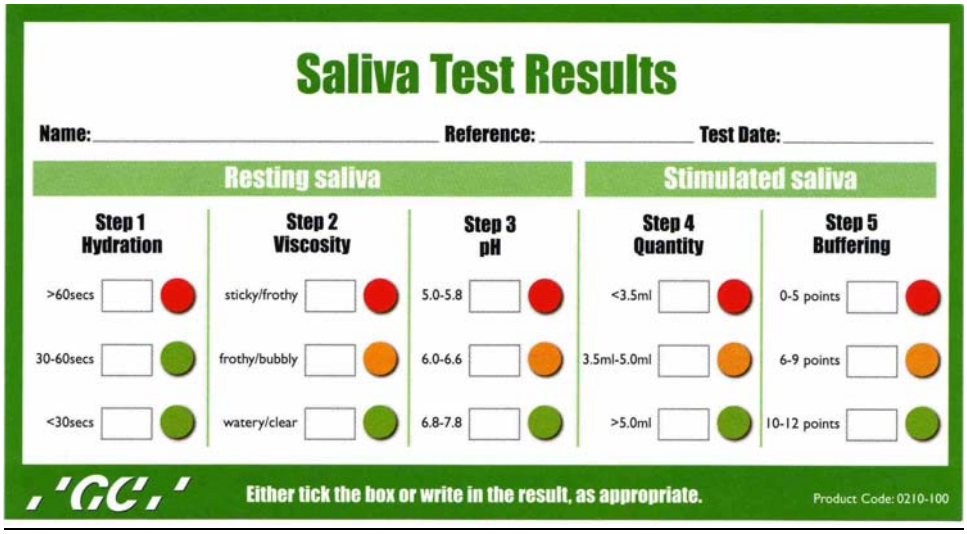


Figure 16. Saliva test result sheet. From GC Corp. Japan.

A repeat Saliva Check Buffer test can be undertaken at 2-3 weeks after the initial test, to reinforce the lifestyle advice and to encourage compliance. The test can be conveniently carried out at the six or twelve month recall examinations. Results can be interpreted in terms of analysis of lifestyle issues and medical /medication-related causes of salivary dysfunction.

It should be noted that pregnant women have reduced buffer capacity in early pregnancy, which reverses in late pregnancy. Menopause can also reduce buffering capacity in some females. Menstruation does not directly affect salivary flow nor pH and buffering, but psychological stress can impair the resting flow rate.

In terms of remineralization of teeth, the importance of the role of saliva cannot be overemphasized. By acting as the major reservoir of and delivery system for calcium, phosphate and fluoride ions, saliva undertakes a key role at the interface with the dental plaque biofilm. Its ionic properties influence the critical pH of the teeth.

- **Modifying Risk Factors - Lifestyle analysis – Medications and Disease**

A medical history including a comprehensive medication history should be a routine part of every dental examination (See common causes of salivary dysfunction). This should include saliva-modifying factors related to habits and lifestyle issues such as smoking, caffeine and water intake, as well as alcohol consumption.

Lifestyle analysis should include a thorough analysis of exercise and work habits. Many athletes including weekend cyclists, triathletes and swimmers, suffer frequent episodes of dehydration which impairs resting salivary flow and can dramatically increase caries risk. Furthermore many athletes quench their thirst with commercial energy drinks which contain caffeine and sugars and also have a low pH. Other weekend or recreational

pastimes can include the exertion of manual work (such as farm-work or gardening), with the attendant risks of decreases in fluid volume due to inadequate water consumption. Difficult social-economic conditions can increase the caries risk considerably, due to a lack of interest or education in oral hygiene, a more cariogenic diet and increases in psychological stress **(Brathall D, 2006)**.

2. REMINERALIZE

- **Fluid balance**

An adequate daily intake of water is critical, because even mild dehydration will reduce saliva flow. Healthy resting saliva contains an effective bicarbonate (HC03) buffer system, plus phosphates, which maintain oral pH in a narrow band of between pH 6.7-7.4.

Saliva also contains inorganic ions of calcium, phosphates and fluoride, plus salivary phospho-proteins (statherin) and proline- rich proteins to maintain a supersaturated calcium and phosphate solution. These proteins have a strong affinity to calcium and apatite surfaces and maintain the correct calcium- phosphate ionic ratio. Through these pathways, saliva controls the mineral balance in an erosive or cariogenic environment. In individuals with salivary dysfunction, the destructive consequences of this to the teeth are graphic.

Water is the major component of saliva by volume, and thus adequate water intake is essential for ensuring sufficient flow of saliva, both at rest and when stimulated. Stimulation of the saliva flow increases oral clearance of acids and can also exert buffering actions. Stimulated saliva will reduce the magnitude of the fall in plaque pH which will occur after carbohydrate intake, and at the same time will increase the possibility that remineralization will occur. Chewing sugar-free gum after carbohydrate intake both neutralizes plaque acid and remineralizes incipient carious lesions **(Edgar et al, 1994)**.

In addition, increases in salivary pH and buffering capacity with flow rate will facilitate remineralization and also will suppress the growth of aciduric microorganisms (particularly *S. mutans* and *S. sobrinus*) in the plaque biofilm. This is another example of changing the ecological balance of the flora by manipulating the oral environment **(Walsh, 2005)**.

It is recommended that patients maintain an adequate daily fluid intake, especially when involved in strenuous activity. For patients with a high caries risk, it would be useful to directly address the low resting pH in the saliva by using alkalinizing mouthrinses (such as sodium bicarbonate) after meals, and reducing the frequency of acidic drinks in the diet, particularly between meals.

- **Dietary Cariogens**

Frequent eating of milk-based foods, such as cheese (as snacks), can prevent or reduce plaque acid production. Dairy products including cheese and milk, can reduce the cariogenicity of fermentable substrates, and this has been demonstrated in a variety of animal and in vitro systems. Clinical studies have shown that processed cheese is hypo-acidogenic, anti-acidogenic, and prevents demineralization as well as enhancing remineralization **(Rosen et al. 1984; Jensen & Wefel 1990; Walsh 2000 B)**.

Dietary advice to patients should include the following (**Walsh, 2000 A; Walsh 2000 B):**

- Dietary restriction of sucrose and other fermentable sugars between meals
- Dietary replacement of sucrose with poorly fermented / non-fermentable substitutes such as maltose, xylitol, sorbitol, sucralose, trehalose and isomalt. Extrinsic sugars derived from fruit are just as cariogenic as sucrose **(Pollard, 1995; Hussein et al, 1996)**
- Dietary restriction of high starch foods between meals. High starch snack foods such as potato chips, donuts, salted crackers have been shown to remain longer on teeth due to slower clearance (compared to low starch, high sucrose foods). Direct breakdown of starches by salivary amylase gives total levels of fermentable sugars similar to high sucrose confectionary foods.
- Dietary restriction of highly acidic foods and drinks, especially soft drinks energy drinks and fruit juices.
- Use of milk based foods such as low-fat cheese or cheese snacks, which can buffer acids and elevate calcium levels. Milk and cheese have been shown to reduce the cariogenicity of fermentable substrates through the action of casein phosphopeptides (CPP).

- **Habits** – Smoking, Caffeine, Alcohol

Smoking results in reduction in saliva flow via a restriction in blood flow to the salivary glands. There is also a local effect of decreasing the oxygen tension in the dental plaque, so encouraging the growth of anaerobic pathogenic organisms.
Other tobacco habits such as tobacco chewing and the use of nicotine patches or gums will have the same dose-dependent vasoconstrictive effects.
Caffeine intake through various dietary sources (coffee, tea, black cola drinks, energy drinks and herbal medicines) has a well described diuretic effect, leading to negative fluid balance with significant reductions in resting saliva flow rate and pH. Alcohol has similar diuretic and fluid balance effects.

- **The role of Biocides**

The goal in using such agents is not to eliminate the plaque flora, since this flora is beneficial to the host, but to suppress the development of a pathogenic plaque. The bacterial nature of the dental caries process means preventive strategies based on the modification, suppression and elimination of *Streptococcus mutans* in the dental plaque are highly effective.

There is no evidence that common usage of chemical agents against dental plaque results in demonstrable adverse effects such as the emergence of resistant strains **(Fejerskov & Kidd, 2003)**.
Biocide delivery systems can include mouthrinses, gels, sprays, chewing gum and varnishes for sustained release.

Fluoride
The mechanisms of action of fluoride have already been discussed in the Primary risk factor section, however at this juncture it is important to recall that the use of high fluoride toothpastes, gels and varnishes can impair bacterial energy utilization, reduce acid production, and impair bacterial viability. In adult patients, optimal fluoride loading is achieved by the intake of fluoridated water combined with twice daily brushing with a fluoride toothpaste (1000 ppm). In high caries risk adult patients, toothbrushing twice daily with a high dose (e.g. 5000 ppm) fluoride toothpaste is indicated.

Chlorhexidine
Chlorhexidine is an antimicrobial agent, which when used intermittently is highly effective for suppressing *S. mutans* with selective bacterial reduction. By interacting with the tooth surface, pellicle, and pioneer colonizing bacteria, it impedes bacterial adhesion to tooth surfaces. Single applications of gels or mouthrinses have an immediate antimicrobial effect with a reduction of 80-95% with a single mouthrinse (**Schiott, 1973**). Some studies show a limited cariostatic effect used without other measures, but reliable results when used in conjunction with oral hygiene, dietary advice and appropriate fluoride products strains **(Fejerskov & Kidd, 2003)**.
Problems which occur with routine daily use, such as extrinsic dental staining, can be obviated using products with anti-discolouration systems which oxidize and de-colour stained compounds. Intensive chlorhexidine use is useful in patients with high levels of *S. mutans*, but must be accompanied by changes in hygiene and diet to sustain the improvements gained. For high caries risk patients, a recommended protocol is toothbrushing once per week (with a separate toothbrush) with a 'pea' sized sample of chlorhexidine gel immediately after breakfast or at least 20 minutes after using fluoride toothpaste.

Xylitol
Use of sorbitol or xylitol containing chewing gums decrease plaque acidogenic potential and increase the protective flow of resting saliva which can neutralize lactate and other strong organics acids from plaque fermentation. Regular use of these agents in sugar-free chewing gums can also promote enamel remineralization (although the results are not as dramatic as those obtained with CPP-ACP).

- **Biomineralization – the use of chemicals: CPP-ACP (Recaldent®)**

The application of products containing Recaldent ® (CPP-ACP), such as GC Tooth Mousse is a major recent development in preventive dentistry. This material works by several mechanisms. First, CPP-ACP binds well to plaque, providing a large calcium reservoir within the plaque, which slows diffusion of free calcium and reduces the ability

of plaque fluid to dissolve the underlying enamel when the pH falls during sugar exposure (**Reynolds et al, 2003)**. The therapeutic importance of elevating dental plaque calcium concentrations has been demonstrated by several research studies, including **Tanaka et al. (1999**) and **Pearce et al (2002)**. Second, CPP-ACP provides a source of calcium for remineralization **(Rose, 2000)**. Third, CPP-ACP also maintains a state of super saturation of phosphate ions with respect to tooth enamel. These phosphate ions can help buffer plaque pH. Finally, CPP-ACP inhibits the growth of *Streptococcus mutans* and other odonto-pathogens. This may be due in part to the increased pool of calcium ions in dental plaque **(Aimutis 2004; Walsh, 2000 B)**. When present in chewing gum, CPP-ACP at a level of 18.8 mg/gum piece has been shown to remineralize enamel subsurface lesions in vivo, and to give a greater effect than gum with xylitol **(Shen et al, 2001)**.
Research to be presented at the IADR General Meeting in Brisbane, Australia in 2006 **(Iijima et al, 2006)**, indicates CPP-ACP gum remineralizes subsurface enamel with mineral of higher crystallinity, making the remineralized enamel less soluble in acid (more acid resistant).
Furthermore a chewing gum containing 54.4mg CPP-ACP significantly slowed progression and enhanced regression of dental caries in a two year clinical trial, relative to a normal sugar-free gum **(Morgan et al, 2006)**.
Significantly, combination of the CPP-ACP with fluoride (CPP-ACFP) demonstrates a significant synergistic subsurface remineralization effect **(Sakaguchi et al, 2006)**.

- **Mechanical disruption of plaque -** Tooth brushing / Flossing / Tongue scraping

The aging of dental plaque (particularly if undisturbed for up to 2 days) gives a greater level of acid production than more immature plaque **(Igarashi et al. 1990)**. As dental plaque becomes mature, the non-mutans streptococci with high acidogenicity and acid-tolerance will establish an acidic environment. This environmental shift permits more acidogenic and acid-tolerant bacteria such as mutans streptococci and lactobacilli to enter the dental plaque ecosystem **(Dawes & Dibdin 1986; Takahashi & Yamada 1999)**. Because of the effect of plaque age/maturity on its microbial composition, the general clinical recommendation is that patients should brush twice daily to reduce plaque/ biofilm thickness, and to deliver fluoride, triclosan and other active agents.
Daily tongue scraping has shown to be a most effective method in reducing salivary mutans streptococci levels (**White & Armaleh, 2004)**.

- **Increase in intra-oral pH**

As noted earlier, increases in caries risk can occur when the intra-oral pH is reduced because of salivary dysfunction or through the consumption of acidic drink. Using pH-elevating measures, such as chewing sugar-free gum and rinsing with sodium bicarbonate, can elevate intra-oral pH. This prevents lowering of pH, or reverses a pH drop completely to suppress the growth of acid tolerant bacteria within the plaque biofilm **(Walsh 2000, B)**. Use of fluoride-containing toothpastes with sodium bicarbonate, result in significantly less plaque acid formation after a sucrose challenge than does a conventional (adult strength) fluoride toothpaste **(Blake-Haskins et al, 1997)**. Buffering effects from saliva

or mouthrinses may add to the buffer systems already present in plaque, such as soluble proteins, peptides, organic acids, and phosphate **(Shellis & Dibdin 1988**; **Walsh, 2000; Walsh, 2000 B**). For these reasons, in patients with ongoing salivary dysfunction, routine use of sodium bicarbonate rinses to buffer plaque acids and reduce the aciduric environment is recommended.

3: REPAIR

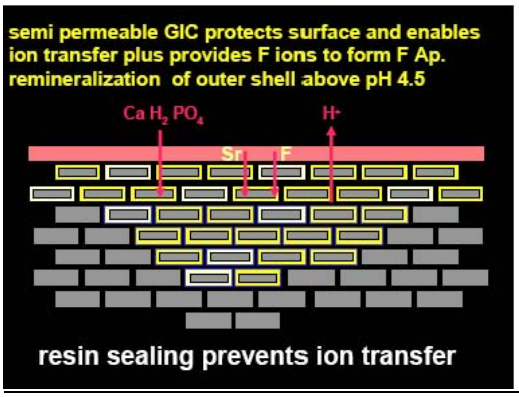
The principle of preservation of tooth structure should dominate decisions about the restoration of new and old carious lesions **(Mount & Ngo, 2000)**. As discussed, once cavitation has occurred, plaque accumulation usually cannot be controlled without surgical (operative) intervention. Deciding how best to intervene and prepare teeth is an everyday problem for the dental practitioner. With traditional operative dentistry, the class I amalgam preparation requires "Extension for Prevention" with the removal of adjacent non-carious pits and fissures, to limit caries development in adjacent areas. Unfortunately, cutting through the enamel into dentine weakens the enamel link between cusps **(Rainey, 2002)**, making them susceptible to movement during mastication. Removal of sound dentine between cusps further weakens teeth, where it is known that increasing the cavity width dramatically reduces tooth strength **(Larson et al, 1981)**. Microscopic tooth movement under function can result in crack propagation over time, leading to cusp fracture and the need for more radical restorative procedures or even actual tooth loss. Use of adhesive restorative materials now allows the minimal removal of tooth structure, without following the traditional "Extension for Prevention" principles, with the restoration of tooth function and plaque control. These minimal techniques are of particular advantage for the restoration of small or incipient pit and fissure carious lesions, whilst maximising tooth strength.

- **Fissure sealing and tooth surface protection**

Sealants are coatings applied by the dentist or hygienist to the grooves of the posterior molar teeth. These coatings are intended to prevent the growth of bacteria that promote decay in the grooves of molar teeth. Based on the work of Simonsen, sealants should be a treatment option for all children immediately after the eruption of the posterior teeth **(Simonsen, 2005)**.
The Cochrane Collaboration has produced a review of pit and fissure sealants **(Ahovuo-Saloranta et al, 2004)**. Its conclusion states that "children who have their molar teeth covered by a resin based sealant are less likely to get dental decay in their molar teeth than children without sealant. The review shows that after 4.5 years, the sealed permanent molar teeth of children aged 5 to 10 had reduction of decay in over 50% of biting surfaces compared to teeth without sealants."
Erupting teeth and newly erupted teeth present a particular problem because the carbonated apatite structure (Ca_{10-x} $(M_x)(PO4)_{6-y}(CO_3)_yOH_2$), is a more soluble form of apatite (which lowers the critical pH).
In studies by Carvalho **(Carvalho et al, 1989, 1992)**, it was found that five times more plaque re-accumulated on the occlusal surfaces of erupting molars compared to fully erupted molars with occlusal function, 48 hours after professional prophylaxis. Most

plaque re-accumulated on the distal and central fossa of the erupting molars. This is due to the difficulty of effective oral hygiene, with the pericoronal tissue sheltering the surface, the adjacent erupted tooth making access difficult, and the low occlusal height of the erupting molar. As a consequence, most carious lesions are initiated in the distal and central fossa during eruption **(Axelsson, 2004)**.
The amount of plaque on occlusal surfaces of erupting molars, compared with erupted molars with full functional wear, explains why almost all occlusal caries in molars is initiated during the extremely long eruption period (12 to 18 months) and why occlusal caries is less common in premolars, which have an eruption period of only 1 to 3 months.

A new concept of **tooth surface protection** for erupting teeth has been developed by Drs. Hein Ngo and Geoff Knight (Australia) in conjunction with GC Corp. Japan, where a high fluoride release glassionomer **(GC FUJI VII ™)** is applied to the complete surface of the erupting tooth. It has been found that in thin layers of half a millimetre or less, this glassionomer cement acts as a semi-permeable membrane, enabling the transfer of calcium, phosphate, fluoride and strontium ions into the apatite crystals in response to acid attack. This protects the underlying tooth and also makes it more resistant to further acid dissolution **(Hicks et al, 2004; Antonson et al, 2006)** (figure17). However, when a newly erupted tooth is sealed with a composite resin, the carbonated enamel is isolated from the oral environment and the process of maturation from a carbonated apatite to a fluoroapatite is prevented **(Knight, 2002)**. These outer layers are then susceptible to acid attack if the fissure seal breaks-down and starts to leak (figure 18).



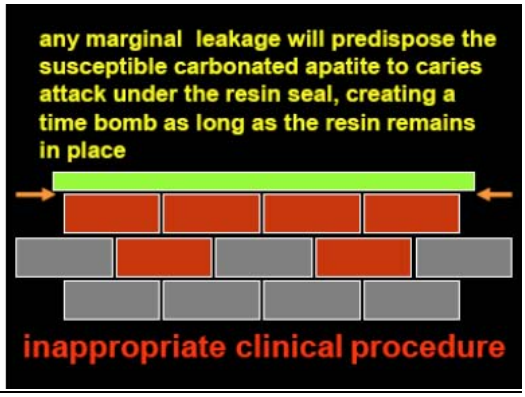


Figure 17. Glassionomer surface protection. Figure 18. Resin surface breakdown
Diagrams courtesy of Dr. GM. Knight: www.Dentalk.com

The effectiveness of fissure sealants has commonly been related to the longevity of sealant coverage i.e., clinical retention in the fissures **(Ripa, 1993)**. Past sealant retention studies show conflicting results between resin sealants and glassionomer sealants **(Arrow & Riordan, 1995; Songpaisan et al, 1995; Poulsen et al, 2001)**. It is relevant that caries prevention and not clinical retention of the sealant should be the main criterion for clinical success. Recent studies show that the use of high viscosity glassionomer materials (e.g.: **GC FUJI IX ™** and **3M-Espe KETAC Molar™**) give a caries-preventive effect of between 3.1 to 4.5 times higher after 3-5 years, compared with resin sealants. Once the fissure sealant is lost, these high strength glassionomer materials have

a 4 times higher chance of preventing caries development in re-exposed pits and fissures of occlusal surfaces in first molars, compared to light cured composite resin fissure sealants over a 1-to -3 year period **(Beirutu et al, 2006)**. This suggests a continuing anticariogenic effect from the glassionomer cement even after apparent macroscopic loss, whereas resin sealants, once lost, do not provide any further preventive effect. This suggests that high viscosity glassionomer sealants should be considered as the material of choice for the fissure sealing of teeth.

For the management of occlusal caries, the flow chart (Figure 19), suggests the following optimal treatment options:

- Monitoring and instituting preventive regimes
- Fissure sealant
- Preventive resin restoration
- Resin composite or Composite resin/GIC laminated restoration or amalgam restoration.

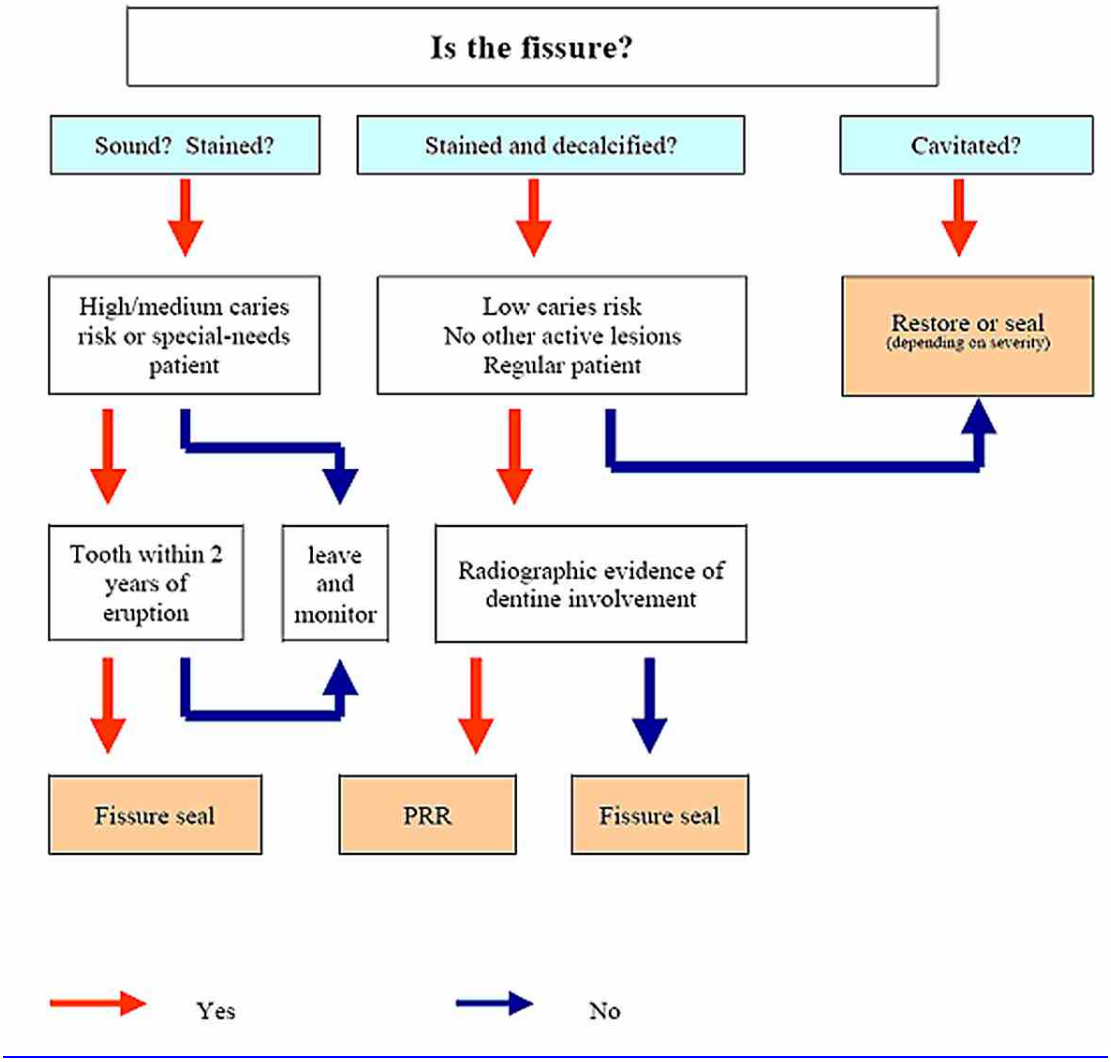


Figure 19. Adapted from Brunton P. *Decision-Making in Operative Dentistry*. Quintessence Publishing Co.

- **Biomimetic restorative materials - Internal healing of dentine**

A new term **"Biomimetic"** or **"interactive**" is being used in regard to restorative materials based on glassionomer chemistry and with particular application in deep carious lesions **(Mount & Ngo, 2000; Wilson, 2001)**.

These *smart* materials have a capacity to self-repair and at least inhibit recurrent and secondary disease. These biomimetic materials are compatible with more inert materials to allow combined applications, such as in the "Sandwich restoration" (composite resin laminated over glassionomer cement).
Glassionomer materials have been recognised to have polyfunctional anticariogenic/ antibacterial action, possibly involving zinc and in certain materials, strontium as well as fluoride **(Wilson, 2001)**.

With bacterial penetration of the enamel and cavitation, the bacterial ecology within the cavity changes resulting in demineralization of the dentinal tubules (collagen framework) and proteolysis of the collagen (by proteolytic bacteria). The resultant structureless denatured layer is known as the *infected layer* and cannot be remineralized (Figure 20). The softened demineralized *affected layer* lies ahead of this proteolysed *infected layer* and is distinguished by an intact collagen framework which can be remineralized from the pulpal fluids, or from external sources (such as biomimetic materials).

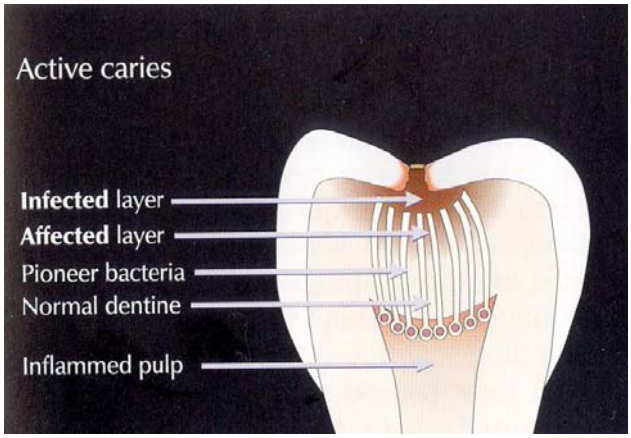


Figure 20. Carious layers within a cavity.

Studies have shown that the *affected dentine* layer can be stabilized if it is isolated from the oral environment **(Massler, 1967; Brannstrom, 1981)**.
Massler and Brannstrom both found that isolation of the caries from the oral environment and elimination of bacteria will bring about stasis in the carious lesion. Mertz-Fairhurst also demonstrated that a lesion can be arrested for many years following isolation with an adhesive material **(Mertz-Fairhurst et al, 1998)**. Sealed lesions which are

radiographically evident have been shown not to progress over a 10 year period **(Handlemann, 1982)**.

The *affected dentine* can be regarded as "pre-carious" rather than actively carious. This layer should be retained and remineralized under most circumstances, reducing unnecessary removal of tooth structure. The optimal clinical approach can then be best summarized, as removal of the infected dentine only, dressing the dentinal wound by sealing the cavity for pulp protection and restoration of the functional anatomy with compatible materials enabling plaque removal.
In their textbook, Mount and Hume state -"*every effort should be expended to bring about healing of the affected layer (of dentine) rather than its removal. This should be regarded as one of the cornerstones of minimal intervention dentistry"* **(Mount & Hume, 2005)**.
The relatively new concept of dentine healing is supported by in-vivo studies showing calcium, phosphate, fluoride and strontium ions from the glassionomer material being incorporated into the remineralized dentine matrix of affected dentine **(Ngo et al, 2001, 2006)**. This demonstrates the interactive biomimetic nature of this material with the *affected layer*.

To achieve internal healing of dentine, a protocol has been suggested by Mount & Hume - A Clinical Protocol for Internal Healing:

Access is gained to the lesion through removal of enamel around the margins of the cavitation. Debridement of the surface of the internal lesion is then carried out to remove the infected dentine and clean the walls around the complete circumference of the lesion sufficient to expose sound healthy dentine (Figure 21).

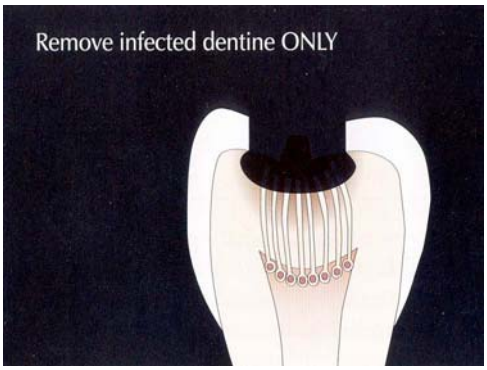
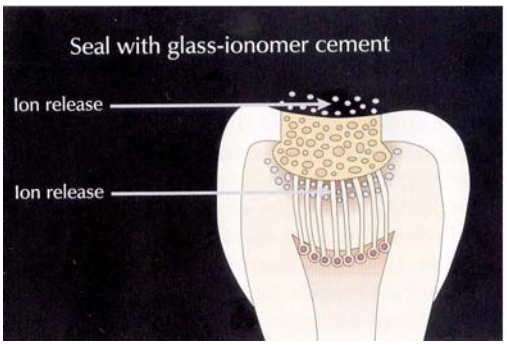


Figure 21. Removal of infected dentine only.

Glassionomer materials such as FUJI VII™ (antibacterial, high fluoride ion levels) can be placed over the affected layer and covered with a thick layer of FUJI IX™. This will allow ion exchange adhesion and the development of a chemical seal between the restoration and the tooth (Figures 22 & 23).



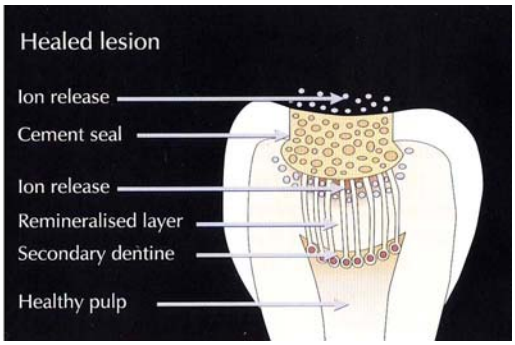


Figure 22. Glassionomer placed over *affected dentine.* Figure 23. The healed lesion
From Mount & Hume. Preservation & Restoration of Tooth Structure 2nd Ed. 2005.

- **Minimal operative techniques**

The advent of the acid-etch technique by Buonocore in the 1950's and 60's brought a huge change to clinical operative dentistry. No longer was it necessary to prepare a tooth to adapt to the limitations of the amalgam restorative material **(Buonocore, 1955)**. Simonsen introduced the "preventive resin restoration" (PRR) in the 70's. The generic term PRR is now accepted as being two separate techniques, one to restore a minimal cavity, and one to prevent future caries (a fissure seal).
As mentioned previously, smooth surfaces of demineralized enamel can be remineralized, as can the *affected dentine* (the softened, non- infected demineralized layer) in the depth of the cavity. Areas of unsupported enamel can be supported with adhesive restorative materials and margins do not have to be extended into theoretically "caries-free" areas. A remineralizing material such as glassionomer cement can be laminated with another stronger material such as composite resin, to better withstand masticatory stresses of the oral environment **(Mount, 1994)**. This has been termed the "Sandwich Restoration" (Figure 24).

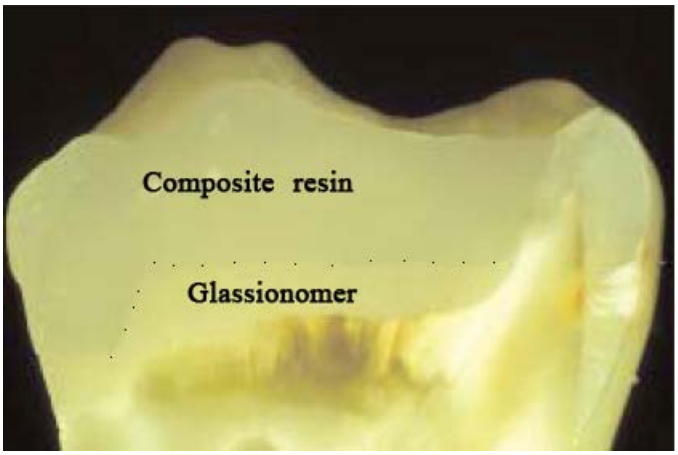


Figure 24. A "Sandwich Restoration" of composite resin and glassionomer (see dotted boundary line).
From Mount & Hume "Preservation & Restoration of Tooth Structure" 2nd Ed.2005.

Minimal clinical cavity access can be defined as the least amount of enamel removal required to enable adequate access for visualisation and removal of the *infected dentine*. This is best accomplished with the use of magnification as this greatly aids the preservation of tooth structure. Use of binocular loupes should almost be considered a pre-requisite for carrying out minimal invasive dentistry.

Micro-preparation techniques:

Micro-preparation techniques can involve a number of cutting modalities:

- Micro-preparation and fissurotomy burs
- Chemomechanical methods
- Sonic tooth preparation
- Air Abrasion
- Lasers

Micro-preparation and fissurotomy burs:

Many manufacturers are now producing smaller burs for cavity preparation using MI techniques. The burs are spherical, tapered or elliptical (Figures 25 & 26) and with the use of magnification, allow very precise preparation of teeth. These allow the dentist to conservatively explore and modify the fissures even when caries has spread laterally along the dentinoenamel junction.

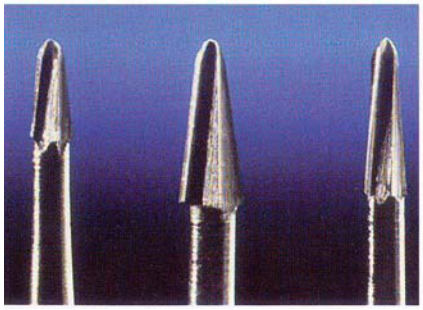


Figure 25. Fissurotomy burs (SS White, USA)

Figure 26. Narrow diamond burs (Brassler, USA)

Chemomechanical methods:

This involves the application of a chemical solution to the caries, selectively softening the carious dentine, facilitating its removal with mechanical hand instruments and without affecting sound non-carious dentine **(Morrow et al, 2000; Ericson et al, 1999)**. The most efficient system available is Carisolv™ (Mediteam Dental – Gothenburg, Sweden), (Figures 27 & 28). Carisolv can be used solely, or in combination with other methods that may be required to gain access to the lesion. This method is particularly suitable for root surface caries and large cavitated coronal cavities. It has the advantage of not usually requiring the use of local anaesthesia.

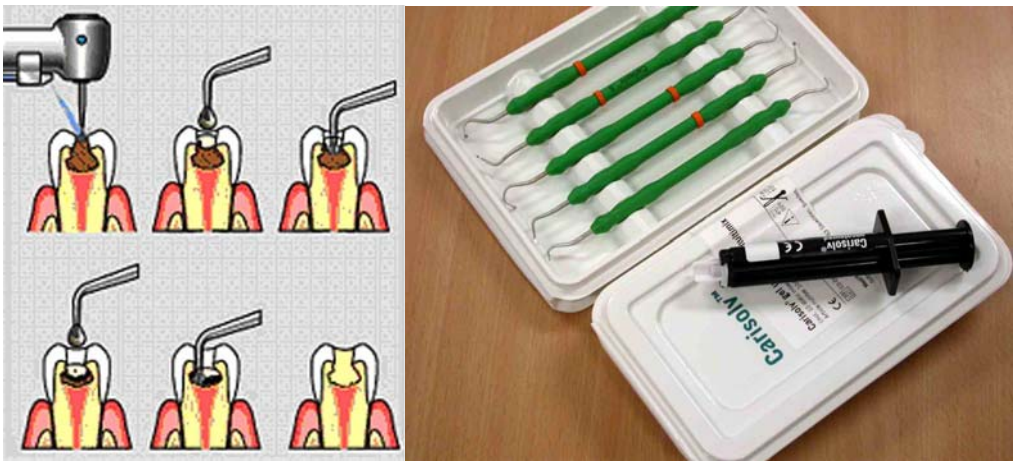


Figure 27. Use of mechanical hand instrumentation with Cariosolv™. Courtesy Mediteam Dental, Gothenburg, Sweden.
Figure 28. Picture of the Cariosolv™ syringe and specifically designed hand instruments.

Sonic tooth preparation:
This utilizes the vibrational energy of ultrasonically vibrated metal tips, rather than rotation. It allows precise minimal cutting preparation using diamond coated tips (Figures 29 & 30).
.

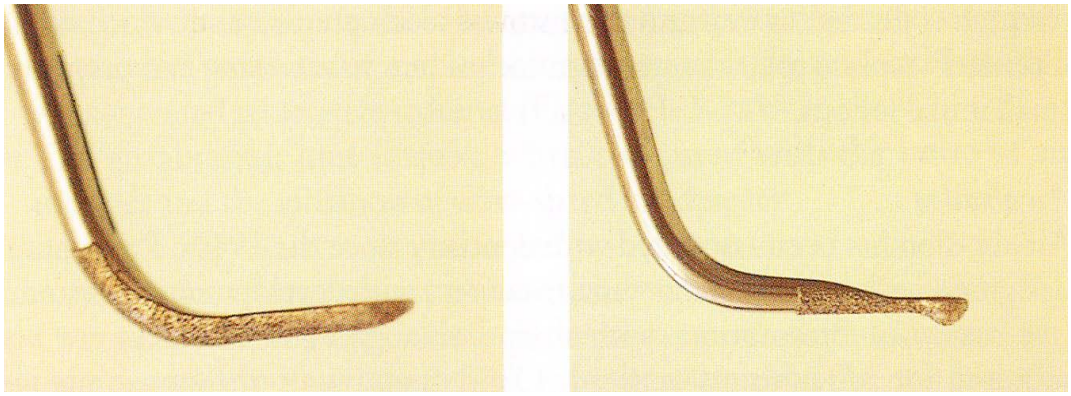


Figure 29 & Figure 30. Diamond coated sonic preparation tips. Courtesy SonicSys system, KaVo Biberach, Germany.

Air Abrasion:
This utilizes a stream of 27.5 micron aluminium oxide particles under air pressure to remove tooth substance by brittle fracture (Figure 13). It produces less heat, sound or vibration compared to high speed instrumentation and does not induce microfractures or microcrazing that follows high speed instrumentation (figure 31)**(Stanley, 1961)**.

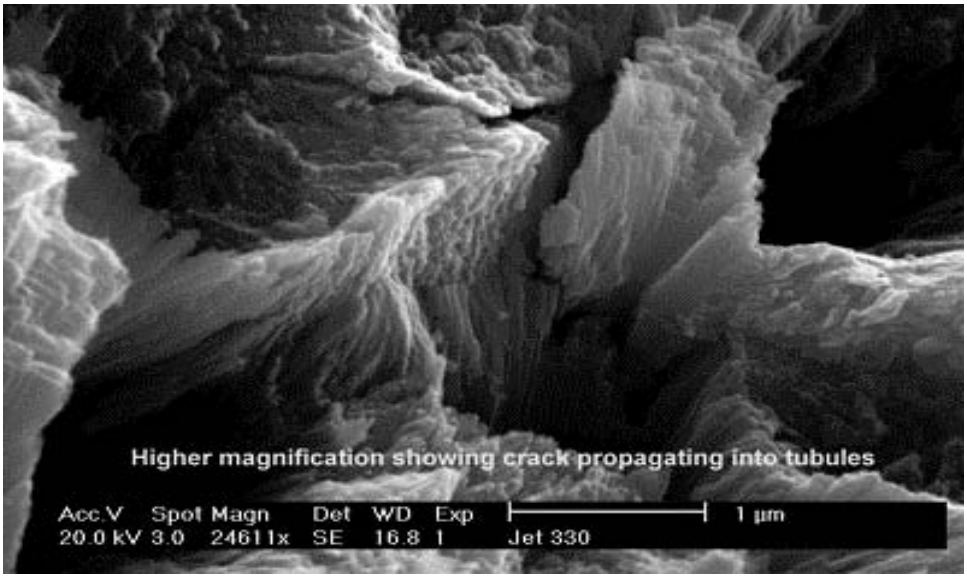


Figure 31. Microcrack propagation into dentine caused by a high speed rotary carbide bur. From Dr. Hien Ngo, Adelaide University, Australia.

Air abrasion can be used for:

- Cutting small pit and fissure cavities
- Removal of composite and porcelain restorations
- Cleaning tooth surfaces of debris and preparation of tooth, metal, porcelain and composite prior to adhesive restoration
- Minor Class II, class III, class IV and class V cavity preparations.

Figure 32. A KaVo air abrasion tip utilizing additional water spray for decreased patient sensitivity.

Air abrasion preparation is conservative and can produce a very fine cut, selectively removing softened carious enamel over dentinal caries to allow access to the infected

dentine **(Rainey, 2002)**. Air abrasion is well tolerated by patients, with the advantage that cavities 2.0 -3.0 mm. deep can be prepared without the use of local anaesthetic.

Lasers:
The process of effective cutting of tooth structure using an Erbium:YAG laser was first reported by Hibst at the University of Ulm in the mid 1980's **(Hibst & Keller, 1989)**. A number of lasers have been investigated for their suitability in cavity preparation and the solid state systems based on erbium (Er:YAG and Er,Cr:YSGG) are the most effective (Figures 33 & 34).
The mechanism of hard tissue removal is basically an explosive subsurface expansion of the interstially trapped water, with the rapid ejection of tooth particles in the opposite direction to the incoming laser beam. As carious tooth structure has a higher water content than sound tooth, it is rapidly and effectively removed by the laser energy **(Belikov et al, 1993)**.
Lasers have the advantages of reduced need for anaesthesia (development of a laser analgesia effect), have a low annoyance factor (less noise and vibration than rotary instrumentation) and leave the cavity surface suitable for adhesive restorations. However the use of laser lacks tactile sense and cutting of tooth structure is slightly slower than with a high speed rotary bur.

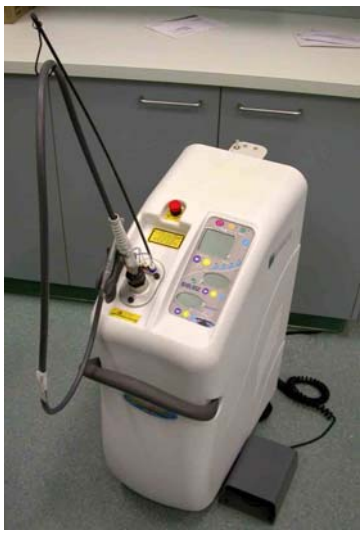


Figure 33. Er,Cr:YSGG dental laser

Figure 34. Er:YAG dental laser.

- **The role of disinfection in arresting dental caries**

Destroying the bacteria present within a carious lesion or changing the ecological niche to encourage remineralization, are current topics of research. These treatment approaches are especially applicable to open lesions such as root caries which cannot be sealed and where the caries process will continue unchecked.

Methodologies that could be considered are:

- Ozone
- Photo-activated disinfection (PAD)

Ozone:

Ozone (O_3) is a powerful oxidizing agent which neutralizes acids and its effects on cell structures, metabolism and microorganisms are well-documented in published papers in both dentistry and medicine **(Bocci, 1993, 1996; Lynch, 2005))**. Research has shown that ozone disrupts the cell walls of microorganisms (bacteria and viruses) within seconds, leading to immediate functional cessation. This rapid effect has clinical significance, as the potential for microbial resistance to this treatment modality is insignificant. In view of its powerful oxidizing properties, O_3 can also attack many biomolecules such as the cysteine, methionine and the histidine residues of proteins and change the surface ecology of the carious lesion. Remineralization from salivary ions occurs readily, due to the surface changes on the exposed dentinal tubules.

Chemistry

Ozone ($O_{3)}$ is naturally produced by the photodissociation of molecular O_2 into activated oxygen atoms, which then react with further oxygen molecules. This transient radical anion rapidly becomes protonated, generating HO_3, which, in turn, decomposes to an even more powerful oxidant, the hydroxyl radical (OH^-).

Indications

Exposed root surfaces in aged individuals with gingival recession are more susceptible to caries **(Hellyer et al, 1990; Galan & Lynch, 1993)**. The micro flora of primary root caries lesions has been shown to contain large numbers of acidogenic and aciduric microorganisms, which correlate with the severity of root caries **(Lynch & Beighton, 1993, 1994; Schuppach et al, 1995; Brailsford et al, 1998; Baysan, 2002)**.

Ozone can now be considered as a clinical alternative management strategy for root caries, and this statement is well supported in the increasing volume of published research. Reversal of primary root caries lesions is associated with remineralization and a corresponding reduction in acidogenic and aciduric micro-organisms **(Beighton et al, 1993; Lynch, 1994)**. This research has shown that ozone also breaks up the acidic products of cariogenic bacteria, which may be important in the aetiology of the developing carious lesion. Research by **Baysan (2002)**, reported that ozone application for either 10 or 20 seconds was effective in achieving a kill of 99% or more (99.9% after 20 seconds) of micro-organisms in primary root carious lesions in vitro and in vivo and an application for a period of 10 seconds was still capable of reducing the numbers of Streptococcus mutans and S. sobrinus in vitro.

Photo-Activated Disinfection:

Photo-activated disinfection (PAD) is a method of disinfecting or sterilizing a site (tissues, wounds and lesions of the oral cavity) by topically applying a photosensitizing agent (a dye) and irradiating the site with laser light at a wavelength absorbed by the photosensitizing agent. Destruction of the microbes occurs without damage to other tissues at the site.

Mechanism of action

The low power laser energy in itself is not particularly lethal to bacteria, but is useful for photochemical activation of the dye.
The photosensitive dyes release reactive oxygen species which cause membrane and DNA damage to the microorganisms. The oxygen free radicals from this process are broken down readily by catalase (present in all tissues and peripheral blood) and lactoperoxidase (in saliva). PAD does not give rise to deleterious thermal effects on teeth or soft tissues. PAD has been shown to be effective for killing bacteria in complex biofilms, which are typically resistant to the action of antimicrobial agents **(Dobson & Wilson, 1992; Sarker & Wilson, 1993; Wilson, 1994)**. It can be used effectively in carious lesions, since visible red light transmits well across dentine **(Burns et al, 1995)**. Major clinical applications of PAD include disinfection of root canals, periodontal pockets, sites of periimplantitis and deep carious lesions **(Walsh, 1997; Dortbudak et al, 2001)**.
An example of a PAD system is the *Dentofex Savedent Red* diode laser **(**Figure 35), with a wavelength of 635 nm that uses tolonium chloride as the photosensitizing dye to sterilize a carious cavity and the infected dentinal tubules. Such systems would have obvious clinical relevance in terms of managing deep dentinal carious lesions. With the ability to rapidly and effectively sterilize the floor of deep carious lesions (to 1.0 mm), more conservative approaches to the removal of demineralized and infected tooth structure could be used. Of note, PAD is still able to exert significant effects even when the cariogenic organisms are protected in a matrix of demineralized dentine **(Burns et al, 1995; Stringer et al, 2000)**.

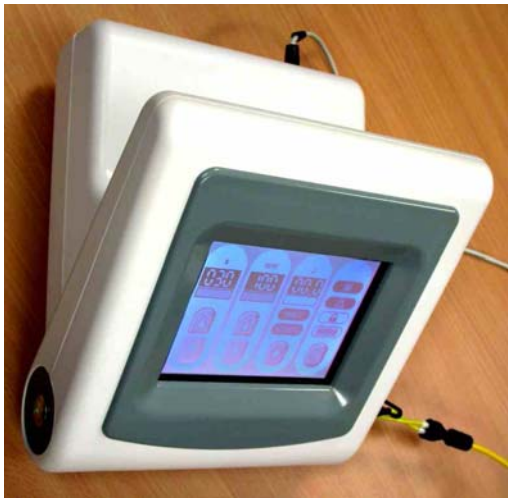


Figure 35. Dentofex PAD Laser. From Dentofex Ltd, Inverkeithing, UK.

Summary

General conservative dentistry is not addressing the causes of the frequent failure of dental restorations. This ‘Repeat –Restoration’ cycle occurs, because general dentists are not trained to diagnose and treat dental caries as a disease, to the detriment of the patients’ oral health.

As described in this article, modern dentistry needs to take a biological (MI) approach to the management of dental caries, where the emphasis must be on diagnosing the oral ecological balance and effecting biological change in the oral biofilm.
By altering dietary and lifestyle patterns and through the use of new diagnostic tests for plaque and saliva, we can monitor patient compliance and modify the disease risk.
New products such as CPP-ACP (Recaldent®) can provide biologically available calcium, phosphate and fluoride ions to enhance tooth remineralization and with the use of sodium bicarbonate rinses (pH elevation) and biocides, we can favorably alter the pathogenicity of the biofilm. New materials and techniques in prevention and repair of tooth damage involve the use of biomimetic restorative materials to heal dentine caries and through the use of new technologies such as ozone or photo-activated disinfection (PAD), we can disinfect carious cavities and encourage remineralization.

References

Aimutis WR. Bioactive properties of milk proteins with particular focus on anticariogenesis. J Nutr. 2004;134:989-995.

Anderson MH. Chlorhexidine and xylitol gum in caries prevention. Spec Care Dentist. 2003;23(5):173-6.

Anderson MH. A review of the efficacy of chlorhexidine on dental caries and the caries infection. J Calif Dent Assoc. 2003;31(3):211-4.

Antonson SA, Wanuck J, Antonson DE. Surface protection for newly erupting molars. Compend Contin Educ Dent. 2006 Jan;27(1):46-52.

Ahovuo-Saloranta A, Hiiri A, Nordblad A, Worthington H, Makela M. Pit and fissure sealants for preventing dental decay in the permanent teeth of children and adolescents. Evid Based Dent. 2004;5(4):93-4.

Arrow P, Riordan PJ. Retention and caries preventive effects of a GIC and a resin-based fissure sealant. Comm Dent and Oral Epidem 1995;23(5):282-5.

Axelsson P. Diagnosis and risk prediction of dental caries, vol.2, Berlin: Quintessence Publishing Co. 2000: 225.

Axelsson P. Preventive materials, methods, and programs. Quintessence Publishing Co. 2004.

Baysan A. Management of primary root caries using ozone therapies. PHD thesis, University of London, 2002.

Belikov AV, Erofeev AV, Shumilin VV, Trachuk AM. Comparative study of 3 micron laser action on different hard tissue samples using free running pulsed Er-doped YAG, YSGG, YAP and YLF lasers. SPIE. 1993;2080:60-67.

Beighton D, Lynch E, Heath MR. A microbiological study of primary root caries lesions with different treatment needs. J Dent Res 1993;73:623-629.

Beirutu N, Frencken JE, van't Hof MA, Taifour D, van Palenstein Helderman WH. Caries preventive effect of a one-time application of composite resin and glassionomer sealants after 5 years. Caries Res 2006;40(1):52-9.

Blake-Haskins JC, Gaffar A, Volpe AR, Banoczy J, Gintner Z, Dombi C. The effect of bicarbonate/fluoride dentifrices on human plaque pH. J Clin Dent. 1997;8:173-7.

Bocci V. Biological and clinical effects of ozone. Has ozone therapy a future in medicine? Br J Biomed Sc.1993;56:270-279.

Bocci V. Ozone as a bioregulator. Pharmacology and toxicology of ozone therapy today. J Biol Regul Homeost agents 1996;10:31-53.

Bowden GH. Effects of fluoride on the microbial ecology of dental plaque. J Dent Res. 1990;69 Spec Iss:653-9.

Bradshaw DJ, McKee AS, Marsh PD. Prevention of population shifts in oral microbial communities in vitro by low fluoride concentrations. J Dent Res. 1990;69:436-41.

Brailsford SR, Lynch E, Beighton D. The isolation of Actinomyces Naeslundii from sound root surfaces and root caries lesions. Caries Res. 1998;32:100-106.

Brannstrom M. Dentine and pulp in Restorative Dentistry. London: Wolfe Medical, 1981.

Bratthal D. Malmö University Department of Cariology website: www.db.od.mah.se/car/data/basic.html

Buonocore MG. A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces. J Dent Res, 1955;34:849-53.
Burns T, Wilson M, Pearson GJ. Killing of cariogenic bacteria to killing by light from a GAA diode laser. J Dent. 1994;22:273-8.
Burns T, Wilson M, Pearson GJ. Effect of dentine and collagen on the lethal photosensitization of Streptococcus mutans. *Caries Res.* 1995;29:192-197.
Carvalho JC, Ekstrand KR, Thylstrup A. Dental plaque and caries on occlusal surfaces of first permanent molar in relation to stage of eruption. J Dent Res 1989;68:773-779.
Carvalho JC, Ekstrand KR, Thylstrup A. Results after 3 years of non-operative occlusal caries treatment of erupting first permanent molars. Comm Dent Oral Epidem. 1992;(20):187-192.
Dawes C, Dibdin GH. A theoretical analysis of the effects of plaque thickness and initial salivary sucrose concentration on diffusion of sucrose into dental plaque and its conversion to acid during salivary clearance. J Dent Res. 1986;65:89-94.
Dobson J, Wilson M. Sensitization of oral bacteria in biofilms to killing by light from a low power laser. Arch Oral Biol.1992;37:883-887.
Dortbudak O, Haas R, Bernhart T, Mailath-Pokorny G. Lethal photosensitization for decontamination of implant surfaces in the treatment of peri-implantitis. Clin Oral Impl Res. 2001;12:104-108.
Edgar WM, Higham SM, Manning RH. Saliva stimulation and caries prevention. Adv Dent Res. 1994;8:239-245.
Ericson D, Zimmerman M, Raber H, Gotrick B, Bornstein R, Thorell J. Clinical evaluation of efficacy and safety of a new method for chemomechanical removal of caries; A multi-centre study. Caries Res.1999;33(3):171-177.
Featherstone JD. The caries balance: the basis for caries management by risk assessment. Oral Health Prev Dent. 2004;2 (Suppl 1):259-64.
Fejerskov O, Kidd EAM. Dental caries: The disease and its clinical management. Copenhagen: Blackwell Munksgaard, 2003.
Galan D, Lynch E. Epidemiology of root caries. Gerodontology 1993; 10:59-71.
Handelmann S. Effect of sealant placement on occlusal caries progression. Clin Prev Dent. 1982; 4:11-16.
Hellyer PH, Beighton D, Heath MR . Root caries in older people attending a general dental practice in East Sussex. Br Dent J 1990;169:201-206.
Hibst R, Keller U. Experimental studies of the application of the Er:YAG laser on dental hard substances: 1. 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. Lasers Surg Med.1989;9:345-351.
Hicks J, Garcia-Godoy F, Flaitz C. Biological factors in dental caries enamel structure. J Clin Pediatr Dent. 2004;2:119-24.
Hussein I, Pollard MA, Curzon ME. A comparison of the effects of some extrinsic and intrinsic sugars on dental plaque pH. Int J Paediatr Dent. 1996;6: 81-6.
Igarashi K, Lee IK, Schachtele CF. Effect of dental plaque age and bacterial composition on the pH of artificial fissures in human volunteers. Caries Res. 1990;24: 52-8.
Iijima Y, Nishimura M, Iijima S, IADR General session, 2006 Brisbane, Australia (Abstract 0184)

Jensen ME, Wefel JS. Effects of processed cheese on human plaque pH and demineralization and remineralization. Am J Dent. 1990;3:217-23.
Kidd EAM. Essentials of dental caries: The disease and its management (3rd edition), London: Oxford University Press, 2005.
Knight GM. The fissure seal dilemma *Aust Dental Assoc News Bulletin* 09: 2002
Krasse B. Can microbiological knowledge be applied in dental practice for the treatment and prevention of caries? J Canad Dent Assn 1984;50:221-3.
Larson TD, Douglas WH, Geistfeld RE. Effect of prepared cavities on the strength of teeth. Oper Dent 1981;6(1):2-5.
Loesche WJ. Clinical and Microbiological aspects of chemotherapeutic agents used according to the specific plaque hypothesis. J Dent Res.1979;58(12):2404-12.
Lynch E. The diagnosis and management of primary root caries (thesis). London: University of London, 1994.
Lynch E. Ozone: The Revolution in Dentistry. Quintessence Publishing Co. 2005.
Lynch E, Beighton D. Relationships between mutans streptococci and perceived treatment need of primary root caries lesions. Gerodontology 1993;10:98-104.
Lynch E, Beighton D. A comparison of primary root caries lesions classified according to colour. Caries Res.1994;28:233-239.
Marsh PD. Are dental diseases examples of ecological catastrophes? Microbiology. 2003; 149 (Pt 2):279-94.
Massler M. Changing concepts in the treatment of carious lesions. Br Dent J. 1967; 123:547-548.
Matsumoto Y, Yoshii E. Evaluation of a new plaque acidogenicity test. IADR General Session, 2005, Baltimore (Abstract 0821).
Mertz-Fairhurst EJ, Curtis JW, Ergle JW, Rueggeberg FA, Adair SM. Ultraconservative and cariostatic sealed restorations: Results at year 10. J Amer Dent Assoc. 1998;129:55-66.
Mickenautsch S, Yengopal V, Bönecker M, Leal SC, Bezerra ACB, Oliveira LB. Minimal Dentistry Compendium: A new approach to dentistry (evidence based compendium) 1st edition 2006, Midentistry Corp. www.midentistry.com, P. O. Box 2779, Houghton, 2041, South Africa.
Morgan MV, Adams GG, Bailey Dl, Tsao CE, Reynolds EC, IADR 2006 Brisbane Abstract 2445 ((E.pub ahead of print).
Mount GJ. An atlas of Glass Ionomer Cements : A Clinician's Guide. London, Martin Dunitz, 1994.
Mount GJ, Ngo H. Minimal intervention: A new concept for operative dentistry. Quint Int, 2000;31(8):527-533.
Mount G, Hume WR, Preservation and Restoration of Tooth Structure, (2nd edition) Brisbane, Knowledge Books and Software, 2005.
Morrow LA, Hassall DC, Watts DC, Wilson NHF. A chemomechanical method of caries removal. Dental Update. 2000;27:398-401.
Newbrun E. Cariology. 3rd ed. Chicago: Quintessence Publishing Co, Inc, 1989.
Ngo H.C, Gaffney S. Risk assessment in the diagnosis and management of caries. In: Mount G, Hume WR, Preservation and Restoration of Tooth Structure, (2nd edition) Brisbane, Knowledge Books and Software, 2005 pp.75-76.

Ngo H, Mount GJ, Morris M, McIntyre J, Tuisuva J, Von Doussa R.
J Dent Res 2001; Spec issue 80 (Abst. No. 919).
Ngo HC, Mount G, McIntyre J, Tuisuva J, Von Doussa RJ. Chemical exchange between glassionomer restorations and residual carious dentine in permanent molars: An in vivo study. J Dent. 2006 Mar 13 (E.pub ahead of print).
~~Add Review~~
Nikiforuk G. Understanding Dental Caries: Vol.1, Etiology and Mechanisms, Basic and Clinical Aspects. Vol. 2: Prevention Basic and Clinical Aspects. Karger Pub, 1985.
Pearce EI, Coote GE, Larsen MJ, The distribution of fluoride in carious human enamel J Dent Res. 1995;74:1775-82.
Pearce EI, Sissons CH, Coleman M, Wang X, Anderson SA, Wong L. The effect of sucrose application frequency and basal nutrient conditions on the calcium and phosphate content of experimental dental plaque. Caries Res. 2002;36:87-92.
Pollard MA. Potential cariogenicity of starches and fruits as assessed by the plaque-sampling method and an intraoral cariogenicity test. Caries Res. 1995;29:68-74.
Poulsen S, Beiruti N, Sadat N. A comparison of retention and the effect on caries of fissure sealing with a glassionomer and a resin-based sealant. Comm Dent and Oral Epidem. 2001;29(4):298-301.
Rainey TJ. Air abrasion: An emerging standard of care in conservative operative dentistry. Dent Clin Nth Am (April) 2002; 185-209.
Reynolds EC, Cai F, Shen P, Walker GD. Retention in plaque and remineralization of enamel lesions by various forms of calcium in a mouthrinse or sugar-free chewing gum. J Dent Res. 2003;82:206-11.
Reynolds EC, Walsh LJ. Additional aids to the remineralization of tooth structure. In: Mount GJ and Hume WR. Preservation and restoration of teeth (2nd edition), 2005. pp. 111-8.
Ripa LW. Sealants revisited: an update of the effectiveness of pit and fissure sealants. Caries Research 1993;27 Suppl 1:77-82.
Rose RK. Effects of an anticariogenic casein phosphopeptide on calcium diffusion in streptococcal model dental plaques. Arch Oral Biol. 2000;45:569-75.
Rosen S, Min DB, Harper DS, Harper WJ, Beck EX, Beck FM. Effect of cheese, with and without sucrose, on dental caries and recovery of Streptococcus mutans in rats. J Dent Res. 1984;63:894-96.
Sagakuchi Y, Kato T, Sato S, Kariya S, Nagao S, Chen L, Remineralization potential of CPP-ACP and its synergy with fluoride. IADR General session, 2006 Brisbane, Australia.
Sarker S, Wilson M. Lethal photosensitization of bacteria in subgingival plaque from patients with chronic periodontitis. J Perio Res. 1993;28:204-210.
Schiott CR. Effect of chlorhexidine on the microflora of the oral cavity. J. Periodont Res (Suppl).1973;12:7-10.
Schuppach P, Osterwalder V, Gunnenheim B. Human root caries: Microbiota in plaque covering sound, carious and arrested carious root surfaces. Caries Res 1995;29:382-395.
Shellis RP, Dibdin GH. Analysis of the buffering systems in dental plaque. J Dent Res. 1988;67:438-46.
Shen P, Cai F, Nowicki A, Vincent J, Reynolds EC. Remineralization of enamel subsurface lesions by a sugar-free chewing gum containing casein phosphopeptides-amorphous calcium phosphate. J Dent Res 2001;80:2066- 70.

Simonsen RJ. Preventive Resin Restorations and Sealants in Light of Current Evidence. Dent Clin N Am 49 (2005):815-823.
Songpaisan Y, Bratthall D, Phantumvanit P, Somridhivej Y. Effects of glassionomer cement, resin-based pit and fissure sealant and HF applications on occlusal caries in a developing country field trial. Comm Dent and Oral Epidem 1995;23(1):25-9.
Sreebny LM. Sugar and human dental caries. World Rev Nutrition Diet. 1982;40:19-65.
Stanley HR. Traumatic capacity of high-speed and ultrasonic dental instrumentation. J Am Dent Assoc. 1961 Dec (63);749-66.
Stephan RM. Intra-oral hydrogen ion concentrations associated with dental caries activity. J Dent Res.1944;23:257-66.
Stringer GJ, et al. Lethal laser photosensitisation of Streptococcus mutans with a visible red diode laser. Aust Dent J. 2000;45(suppl):S22.
Takahashi N, Yamada T. Acid-induced acid tolerance and acidogenicity of non-mutans streptococci. Oral Microbiol Immunol. 1999;14:43-8.
Tanaka M, Matsunaga K, Kadoma Y. Effect of rinse with calcium enriched milk on plaque fluid. J Med Dent Sci. 1999;46:123-6.
Tinanoff N, Association of diet with dental caries in preschool children. Dent Clin North Am. 2005;49:725-37.
Twetman S, Grindefjord M. Mutans streptococci suppression by chlorhexidine gel in toddlers. Am J Dent.1999;12(2):89-91.
van Loveren C, Buijs JF, Kippuw N, ten Cate JM. Plaque composition, fluoride tolerance and acid production of mutans streptococci before and after the suspension of the use of fluoride toothpastes. Caries Res.1995;29:442-8.
Wahab FK, Shellis RP, Elderton RJ. Effects of low fluoride concentrations on formation of caries-like lesions in human enamel in a sequential-transfer bacterial system. Arch Oral Biol. 1993;38:985-995.
Wan AK, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. Oral colonization of Streptococcus mutans in six-month-old predentate infants. J Dent Res. 2001;80(12): 2060-5.
Wan AK, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. A longitudinal study of Streptococcus mutans colonization in infants after tooth eruption. J Dent Res. 2003 Jul; 82(7):504-8.
Wan AK, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. The effects of chlorhexidine gel on Streptococcus mutans infection in 10-month-old infants: a longitudinal, placebo-controlled, double-blind trial. Pediatr Dent. 2003;25(3):215-22.
Walsh LJ. The current status of low level laser therapy in dentistry, Part 2. Hard tissue applications. Aust Dent J. 1997;42:302-306.
Walsh LJ. Preventive dentistry for the general dental practitioner. Aust Dent J. 2000 (A); 45(2):76-82.
Walsh LJ. Fifteen strategies for caries prevention: Towards target zero. ADA News Bull. 2000 (B);278:17-20.
Walsh LJ. Lifestyle impacts on oral health. In: Mount GJ and Hume WR. Preservation and restoration of tooth structure (2nd edition). Brisbane, Knowledge Books and Software. 2005. pp.83-109.
Walsh LJ. Saliva testing: good practice, good sense. Tokyo: GC Corporation, 2002.

Walsh NP, Montague JC, Callow N, Rowlands AV. 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;49:149-54.
White GE, Armaleh MT, Tongue scraping as a means of reducing oral mutans streptococci. J Clin Pediatr Dent. 2004 Winter;28(2):163-166.
Wilson M. Bacterial effect of laser light and its potential use in the treatment of plaque-related diseases. Int Dent J. 1994;44:181-189.
Wilson NH. Conference report. Direct adhesive materials: current perceptions and evidence-future solutions. J Dent. 2001 Jul;29(5):307-16.
