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Defining, Classifying, and Placing Incipient Caries Lesions in Perspective

Graham J. Mount, AM, BDS(Syd), FRACDS, DDSc(Adel)

The University of Adelaide, Frome Road, Adelaide, 5006 South Australia, Australia

Up to the present time, identification of the disease of caries has relied on recognition of demineralization or cavitation on the tooth surface. Caries is a bacterial disease, and therefore the real beginning occurs with the introduction of a combination of a specific bacterial population, capable of surface demineralization, into an environment that has been modified to the extent that those particular species can flourish. Modifications to the environment include alterations to the texture, to the acid level, and to the flow of the saliva, and the frequent and repeated presence of refined carbohydrate or low pH food or drink. The frequency and efficiency of oral hygiene routines will have a bearing, particularly on the state of the periodontium, but are relatively secondary in the generation of demineralization of tooth structure.

The earliest stages of the disease can remain hidden until there has been some surface damage to a tooth crown, and it becomes visible to the naked eye as a white spot lesion, it can be detected on a radiograph, or, worst of all, located through the use of a sharp probe. Until recently, surgical intervention was considered the essential cure for such a lesion, and the profession has been trained to undertake operative procedures immediately. However, it has now been shown clearly that the disease progresses reasonably slowly in the early stages. In fact, it can be controlled or even eliminated before surface cavitation. A lesion is reversible and can be healed up until there is a cavity deep enough to retain bacterial plaque [1].

Recognition of the initiation and early establishment of the disease should be the primary concern of this profession rather than the search for

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E-mail address: gjmount@ozemail.com.au

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cavities. The combination of factors that can vary from normal and lead to the demineralization of the tooth surface needs to be understood, and methods of identification of these variations must be refined [2]. A dentist should become a physician first to understand the oral environment in detail before becoming a surgeon to repair the ultimate damage that may result from the later stages of the disease. On the other hand, if the disease has already progressed to surface cavitation before identification, recognition of the aberrant factors is imperative before surgical intervention, because it is no good repairing the damage caused by the disease if it is allowed to remain active [3]. Identification of hidden caries should arise from recognition of the presence of the disease before there is evidence of white spot lesions or actual surface cavitation.

A constant cycle of demineralization followed by remineralization takes place on the tooth surface because of continual fluctuations in the intraoral pH levels. However, the tooth surface is protected to a degree by the oral biofilm, the combination of pellicle and plaque, which closely adheres to the tooth surface, because this helps to prevent total loss of calcium and phosphate ions from the immediate environment. Even though ions are released from the enamel in the presence of a lowered pH, many may remain trapped within the biofilm and are available to return into the tooth surface as the pH level rises again [4].

This is a normal healthy ion exchange, which takes place because of the constant fluctuations of pH values within the oral environment. There are frequent episodes of lowered pH through ingestion of various food and drink, and there are also extensive variations in the salivary flow throughout the day and night. Nature can accommodate these changes and, particularly in youth, there may be a net gain in mineralization over time leading to maturation of the tooth surface. It is only when there are vigorous and prolonged periods of lowered pH in this oral balance that there may be a net loss of mineral, leading in the long run to surface cavitation. Once this has occurred, there will be a modification to the bacterial flora in the immediate environment of the damaged enamel, leading eventually to proteolysis of the collagen support for the dentine and extended cavitation.

The following article is a brief review of the main factors involved in maintenance of the oral balance. The profession must become aware of the importance of identifying the early evidence of disease, rather than just seeking cavities [5]. After all, it can take as long as 4 years for a caries lesion to penetrate the enamel and a further 4 years to reach the depths of the dentine [6,7]. It is most important to recognize that it is a complex of factors that will lead to the net loss of mineral from the tooth surface, and this is the precursor to cavitation. No one factor is paramount, and testing methodologies already are available for each of them. The tests should be used routinely for the identification of the susceptible patient long before cavitation becomes apparent. However, if the patient does not present in time, the imbalance of the oral factors that have led to the disease state must

be identified and countermeasures instituted before finite restorations are placed. In the presence of rampant caries, the concept of transitional restorations represent sound treatment planning because many cases of recurrent caries are simply continuing caries arising through failure to control the disease before placing restorations. All of the following factors must be assessed and taken into account.

Bacterial population

Streptococci are the first bacterial species to adhere to teeth and begin plaque formation [8,9]. Other species progressively infiltrate the plaque and after a few days of unimpeded growth, gram-negative bacilli predominate. The most cariogenic organisms are adherent streptococci such as *Streptococcus mutans* [10], *S sobrinus*, and the bacillus *Lactobacillus*. These organisms not only produce organic acids rapidly from refined carbohydrates (ie, they are acidogenic) but also are able to withstand highly acidic environments (ie, they are aciduric). *S sobrinus* is the most rapid acid producer, though it is usually present in much reduced numbers relative to *S mutans*. The lactobacillus is one of the predominant organisms in already carious dentine. The polysaccharides secreted by *S mutans* and other plaque bacteria, provide adherence to the tooth structure via pellicle and will produce further carbohydrate for bacterial metabolism when dietary sources have been exhausted. The oral biofilm covers all teeth and after removal with hygiene procedures will immediately begin to reform.

Several methods have been developed for testing the bacterial population [11]. Those currently available are semiquantitative and involve placing a saliva sample on a culture medium and incubating for 48 hours. The bacterial count is obtained by comparing the culture to a chart supplied by the manufacturer. There are clinical limitations because it takes up to 48 hours to incubate the sample, and correct interpretation of the results requires training. This methodology is tedious and lacks immediate impact on the patient. Further generations of bacterial tests are being developed, and these will be based on the use of monoclonal antibodies or enzymes and are expected to provide rapid chairside results.

Testing the bacterial population in isolation will be of limited value because of the continuing variation in concentration in the average patient. It is likely that *S mutans* for example will always be present but the relative population level will vary. However, when assessed in relation to other factors it may take on additional significance. Control of bacterial flora is relatively simple through the use of mouthwashes such as chlorhexidine. A 2% water-based gel applied in a tray or used as a mouthwash over 2 weeks will reduce the bacterial population dramatically. Alternative mouthwashes containing either triclosan or essential oils, which are both natural phenolic compounds, are also available and reasonably effective.

Saliva

Saliva performs a multiplicity of roles within the oral cavity. Regrettably, its importance is usually not appreciated until it is absent, and the profession has tended to ignore it. Salivary dysfunction is a common problem and is frequently undiagnosed, at least in part because the patient's symptoms are not a reliable indicator of salivary gland function [12]. Patients are rarely aware of diminished output until the resting flow rate is less than half the normal rate.

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

Healthy resting saliva maintains a pH in a narrow range between 6.7 and 7.4 and contains a very effective bicarbonate (HCO_3-) buffer system [13]. In the peripheral bloodstream, the combination of sodium bicarbonate, carbonic acid, and gaseous carbon dioxide removes protons (hydrogen ions) from the system. Saliva works in a similar way. The concentration of bicarbonate ion in resting saliva is approximately 1 mmol/L, and this increases to over 50 mmol/L on stimulation. As the concentration of bicarbonate ion increases, the pH rises, and the buffering capacity increases [3], and this is a key point in interpreting diagnostic tests. Because of diurnal variations in resting flow rate, there are corresponding variations in levels of bicarbonate and thus in the pH and buffering capacity. The resting flow rate will be lowest during sleep and immediately after waking, and then varies throughout the waking hours.

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

A serious modern-day problem is that several hundred pharmaceutical products will induce or encourage salivary dysfunction, and this important side effect is not always listed as an adverse effect in prescribing drug guides. It can also be a side effect resulting from delayed metabolism and clearance of drugs by the liver or kidneys. Over-the-counter (OTC) medications such as expectorants and decongestants are particularly important. Polypharmacy is common among elderly and medically compromised patients, so all in this category should be tested for salivary dysfunction, even in the absence of overt caries or other pathology.

A patient's psychologic state can also influence their resting salivary output considerably. A high level of stress in the workplace or home

environment can lead to prolonged reductions in resting salivary flow rate because of the alterations in sympathetic and parasympathetic output that occur with emotional and psychologic stress. Age, per se, will have no direct effect [14], but the elderly patient whose epithelial attachment has migrated apically will be particularly susceptible to root surface caries, and this is difficult to detect in the early stages.

Salivary flow rates can be tested in the office, but there has to be a complete understanding of the measurements being recorded [15]. The unstimulated flow rate, otherwise known as the resting flow rate, is probably the most significant because this is the saliva that is constantly available on the tooth surface. Stimulated saliva has different properties, is only available in relation to eating or drinking, and can provide high-volume and high-quality buffering. This suggests that both resting and stimulated flow rates, as well as quality and buffering capacity, should be measured under similar circumstances on a number of occasions before a decision is reached for an individual patient. Restitution of flow rate can be complicated and will often involve monitoring of OTC medications as well as consultation with other health professionals. Careful supervision may be required, particularly for patients who are obliged to maintain drug therapies over long periods of time.

Frequency of carbohydrate intake

The most significant patient behavior factor leading to an increase in caries risk is the frequency of consumption of fermentable carbohydrate [16]. There is good evidence that the frequency of eating rather than the total quantity of fermentable carbohydrate consumed causes caries. The mono- and disaccharides are the most vulnerable to rapid fermentation, though some of the highly processed starches have also been shown to contribute to acid production. The acids resulting from carbohydrate fermentation are weak organic acids and in most cases will only cause chronic low-grade demineralization. However, when a high frequency of sugar consumption is maintained over a prolonged period in the presence of a cariogenic bacterial flora, or there is a serious deficiency of natural host protective factors, caries will progress more rapidly.

In some circumstances, the addition of strong dietary food acids, or even refluxed gastric acids, will exacerbate the problem. Strong dietary acids are available from a variety of extrinsic sources such as carbonated soft drinks, sports drinks, cordials, and fruit juice [17]. Frequent or prolonged exposure to these can lead to rapid demineralization and can turn mild caries into a rampant attack. Gastric reflux is often not recognized by the patient who may think it is normal and not potentially damaging to the teeth.

Bacterial metabolism of high levels of refined carbohydrate in plaque can cause an immediate 2 to 4 point drop in pH at the tooth surface [18]. The

degree of fall depends on plaque thickness, the number and mix of plaque bacteria, and the efficiency of salivary buffering, along with other factors. Recovery to normal resting pH takes from 30 minutes for the average patient to several hours for those with a high susceptibility to caries. A very high salivary flow rate may return the pH toward neutral quite rapidly, but local retention of sticky foods may delay the rise until the food is completely dissolved or removed [19]. Demineralization is proportional to the combination of the pH level and the duration of contact of low pH plaque with the tooth surface (Fig. 1) [20].

The only available method of altering or modifying carbohydrate intake is through patient counseling, which is difficult and time consuming [21]. The most important factor is the frequency of intake rather than the quantity, and this relates to the period of time over which the sugar is actually in the mouth. A bottle of soft drink imbibed in one gulp is relatively harmless. Sipped slowly over 30 minutes it is much more dangerous. Breath-freshening lozenges sucked for prolonged periods are equally dangerous and so on.

Exposure to fluoride

Frequent exposure to fluoride achieves optimal low-level loading of the salivary fluoride reservoir sufficient, at least to a degree, to prevent the initiation and progression of caries. At low concentrations, fluoride inhibits demineralization and enhances remineralization through precipitation of calcium phosphates, and the formation of fluorapatite [22,23].

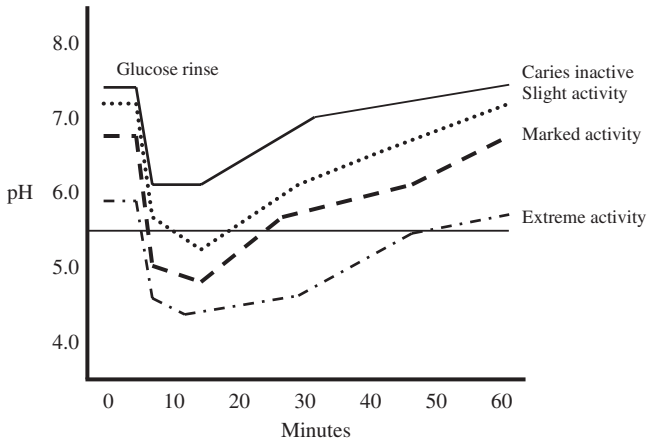

Fig. 1. The approximate time taken for the pH of the oral environment to rise again after a glucose challenge. (*Adapted from* Nikiforuk G. Understanding dental caries, volume 1. Prevention, basic and clinical aspects. New York: Karger; 1985. p. 152; with permission.)

When present in high concentrations, fluoride can inhibit bacterial enzymes and reduce the use of fermentable substrates by *mutans streptococci*. At levels of 0.5 mmol fluoride/L within plaque, the cell cytoplasm of *mutans streptococci* is acidified, and glycolysis is inhibited once the intracellular pH reaches pH 5.2. This accounts for 75% of the caries inhibition effect of high concentrations of fluoride, whereas the remaining 25% is due to the combined effect of fluoride on enamel solubility. However, because this does not reduce the number of viable bacteria, some form of antibacterial agent, such as chlorhexidine [24,25], is essential for caries control in the high-risk patient.

The correct exposure to fluoride will also result from patient counseling, and it is important that it is well understood. Not all communities have fluoridated water, but alternatives are available and must be appreciated and used correctly [26]. A constant low level in the saliva is notably more efficient in controlling demineralization than an occasional professional topical application.

Demineralization

The mineral component of enamel, dentine, and cementum is hydroxyapatite (HA) consisting essentially of $Ca_{10}(PO_4)_6(OH)_2$. In a neutral environment, HA is in equilibrium with the local aqueous environment, that is the saliva, which is saturated with Ca_{2+} and $(PO_4)^{3-}$ ions [27].

HA is reactive to hydrogen ions at or below pH 5.5, because this is the critical pH for HA. H^+ reacts preferentially with the phosphate groups in the aqueous environment immediately adjacent to the enamel crystal surface. The process can be described as conversion of $(PO_4)^{3-}$ to $(HPO_4)^{2-}$ by the addition of H^+ and at the same time the H^+ is buffered. The $(HPO_4)^{2-}$ is then not able to contribute to the normal HA equilibrium because it contains PO_4, rather than HPO_4, and the HA crystal therefore dissolves. This is termed demineralization [4]. Specifically, it results in dissolution of the surface crystals on the enamel rods and follows down the prism sheaths, making the enamel more porous. The central core of the rods may remain, but the light transmission properties will be altered and the white spot lesion will become apparent (Figs. 2–4) [28].

Ultimately the porosity will penetrate as far as the dentine, whereas the central core of the rods may remain substantially intact and this may allow demineralization of the dentine itself with the ions moving back out along the same pathways. There will be an immediate response from the pulp, which will stimulate a degree of mineralization in lateral tubules in the dentine subjacent to the lesion thus forming the translucent zone [29,30]. This represents a pulpal defense mechanism, and this zone will precede the lesion as it penetrates the dentine all the way to the pulp chamber (Figs. 5 and 6).

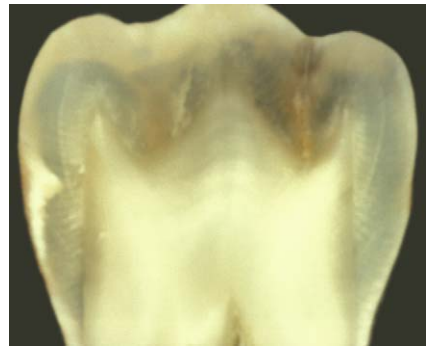

Fig. 2. A cross-section through a molar tooth shows the early white spot lesion on one proximal surface. Note the roughly triangular outline with the deepest penetration of demineralization in the center and the fine spread occlusally and gingivally.

Initially the physical properties of the enamel will not be significantly reduced, but over time the surface will become fragile and easily damaged by instruments such as a dental probe. Eventually the external surface will collapse, plaque bacteria will begin to permanently colonize the cavitation, and the process will become irreversible. The speed and depth of penetration will depend on the presence or absence of fluorapatite in the enamel as well as the concentration and frequency of acid attack.

Remineralization

The demineralization process can be reversed if the pH is neutralized and there are sufficient Ca^{2+} and PO_4 ions available in the immediate environment. Either the apatite dissolution products can reach neutrality by buffering, or Ca^{2+} and $(PO_4)^{3-}$ ions in saliva can inhibit the process of

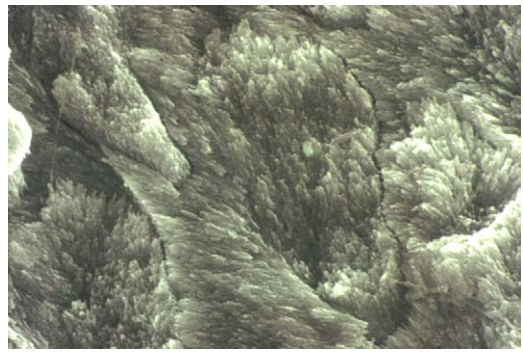

Fig. 3. A freeze-fractured specimen of enamel showing the enamel rods intertwined together with the enamel crystals on the surface. Original magnification $\times 4000$. (Courtesy of H.C. Ngo, BDS, MDS, PhD, Adelaide, South Australia.)

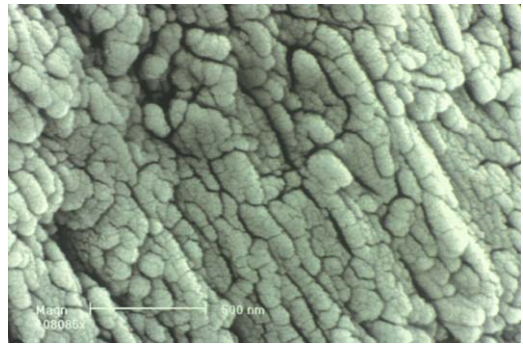

Fig. 4. A specimen similar to the one shown in Fig. 3 where the fracture passes through a white spot lesion. Note the loss of surface crystals from the enamel rods leading to a degree of porosity. Original magnification ×8000. (Courtesy of H.C. Ngo, BDS, MDS, PhD, Adelaide, South Australia.)

dissolution through the common ion effect. This enables rebuilding of partly dissolved apatite crystals and is termed remineralization.

Normally remineralization is a surface phenomenon that is self-limiting and prevents penetration of ions into the depth of the lesion, especially in the presence of fluoride [22]. Rapid deposition of fluorapatite will form a firm surface layer, which is notably more resistant to further demineralization but at the same time is resistant to penetration of the calcium and phosphate ions required to rebuild the lesion in depth. The result is often the visible persistence of the white spot lesion, including some level of stain uptake that emphasizes its presence.

A new remineralization technology has been developed based on phosphopeptides from milk casein [31,32]. The casein phosphopeptides (CPP) contain multiphosphoseryl sequences that have the ability to stabilize

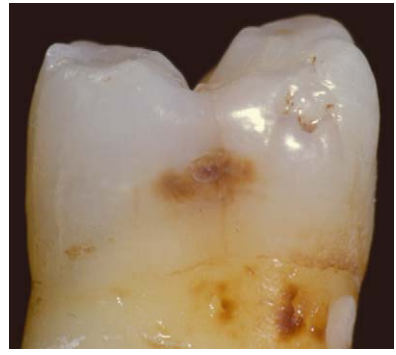

Fig. 5. The proximal surface of a bicuspid tooth showing the typical white spot lesion in relation to the contact area. Note the surface has taken up stain but is still smooth and not cavitated.

Fig. 6. The tooth shown in Fig. 5 has been sectioned through the white spot lesion. Note the demineralization has already penetrated through to the dentine and the pulp has generated its own response with the development of the translucent zone. This lesion can still be remineralized and healed because the surface is not cavitated.

calcium phosphate in nanocomplexes in solution as amorphous calcium phosphate (ACP). Through their multiple phosphoseryl sequences, the CPP bind to ACP in metastable solution, thus preventing its growth to the critical size required for nucleation and phase transformation to an insoluble crystalline calcium phosphate. The CPP-ACP nanocomplexes have been shown to localize at the tooth surface and prevent enamel demineralization. They have also been shown to remineralize enamel subsurface lesions in depth in situ and in vivo by producing an amorphous calcium fluoride phosphate, stabilized by the CPP at the tooth surface, and capable of penetrating to the base of the lesion. The soluble calcium, phosphate, and fluoride ions will promote remineralization with fluorapatite that is more resistant to acid challenge and capable of full penetration into the depths of the enamel lesion. One of the advantages of full penetration can be the restoration of normal translucency in the enamel and elimination of the white spot lesion (Figs. 7–9).

The chemical basis of the demineralization/remineralization process is similar for enamel, dentine, and root cementum [33]. However, the differing structures and relative proportion of the mineral and organic tissue content of each of these components of tooth structure results in significant differences in the nature and progress of the carious lesion.

Early enamel lesion

The initial enamel lesion results when the pH level at the tooth surface is lower than that which can be counterbalanced by remineralization in depth but is not low enough to inhibit surface remineralization. The acid ions penetrate deeply into the prism sheath porosities leading to subsurface

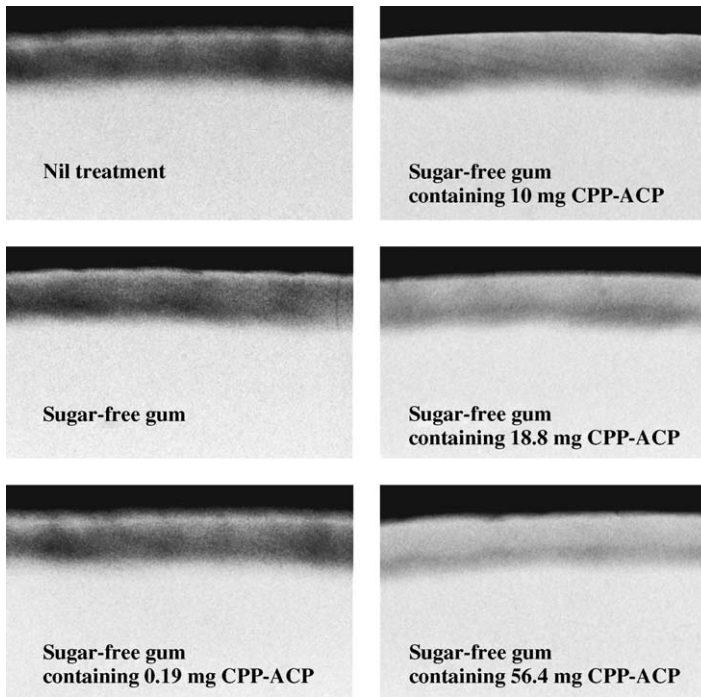

Fig. 7. Microradiographic images of enamel subsurface lesions remineralized in situ by sugar-free chewing gum containing CPP-ACP. (Courtesy of Professor E.C. Reynolds, BSc [Hons], PhD, Melbourne, Australia.)

demineralization. The tooth surface may remain intact because remineralization occurs preferentially at the surface due to increased levels of Ca and HPO_4 ions, fluoride ions, and buffering by salivary products [24].

The clinical characteristics of these lesions include:

- loss of normal translucency of enamel because of altered light properties with a chalky white appearance, particularly when dehydrated;
- a fragile surface layer susceptible to damage from probing, particularly in pits and fissures;
- increased porosity, particularly of the subsurface, with increased potential for uptake of stain;
- reduced density of the subsurface, which may be detectable radiographically, with transillumination or with modern laser detecting devices;
- a potential for remineralization, with an increased resistance to further acid challenge particularly with the use of enhanced remineralization treatments.

The depth and dimension of the lesion will vary from the center to the periphery depending on the thickness of the biofilm and the acid concentration. As shown in Fig. 2, the lesion will be triangular or saucer-shaped,

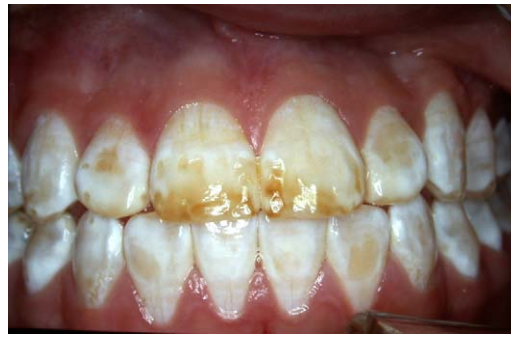

Fig. 8. The patient had a moderate level of fluorosis in all teeth, leading to poor aesthetics. (Courtesy of Professor E.C. Reynolds, BSc [Hons], PhD, Melbourne, Australia.)

varying from very shallow at the periphery to full depth at the center. The center of the lesion may progress until the underlying dentine becomes involved and demineralized. Interproximal lesions will then become detectable radiographically. Even so, the surface of the tooth may remain intact, and the lesion may still be reversible in depth.

However, as the central area of enamel collapses and becomes cavitated, it will be necessary to undertake surgical debridement and repair. But this does not mean that all demineralized enamel around the periphery should be removed. It will remain subject to remineralization as long as it has a smooth surface and can be properly cleaned so operative procedures can remain conservative (Figs. 10–12).

In reversing an early enamel lesion, the ideal would be to regain the original density of enamel, which is possible with the use of CPP/ACP [31]. Even if this is not available, the partially remineralized incipient lesion will be more resistant to further acid attack than normal enamel and physically stronger as well. Hence, it is preferable, where the patient is maintaining

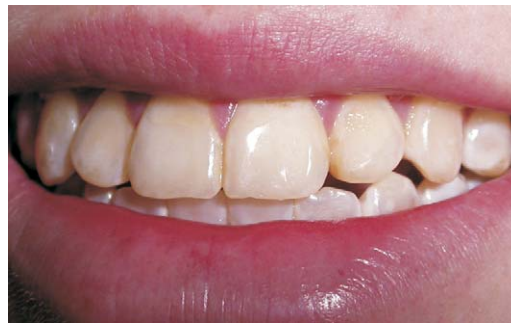

Fig. 9. The same patient shown in Fig. 8 after treatment with microabrasion and the routine application of Tooth Mousse (GC Corporation, Tokyo, Japan) for 2 weeks. (Courtesy of Professor E.C. Reynolds, BSc [Hons], PhD, Melbourne, Australia.)

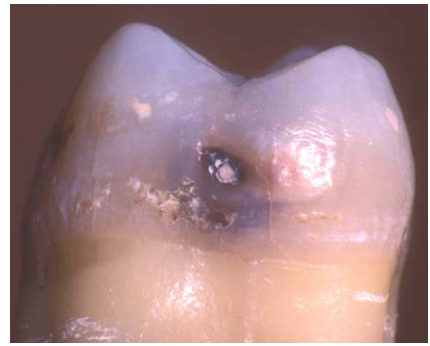

Fig. 10. In a laboratory exercise, this lesion is to be treated using normal clinical protocols and restored using a tunnel approach cavity design. Note the area that is actually cavitated and is surrounded by further demineralization.

good home care and the disease is under control, to observe the lesion over time rather than adopt a surgical approach immediately and deny possible remineralization.

However, although this is the preferred approach to the smooth surface interproximal or cervical lesion, it may be unacceptable treatment for an occlusal fissure. The main problem is that the incipient lesion in a fissure is subject to considerable occlusal load during mastication and the fragile demineralized enamel is readily damaged. Protection with resin or glass-ionomer sealant is recommended at the earliest sign of caries, particularly in the patient who is regarded as a high caries risk (Figs. 13 and 14) [34].

Caries into dentine

Once demineralization has progressed through the enamel into dentine, bacterial plaque will become a permanent inhabitant and it will no longer be

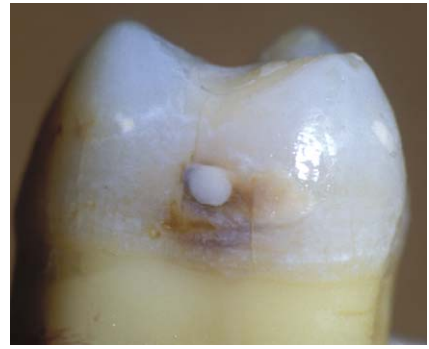

Fig. 11. The cavity has been designed and the restoration placed. Note the area of demineralized enamel surrounding the restoration. Because of the adhesion and ion exchange available with glass-ionomer, that area of enamel can be retained and remineralized.

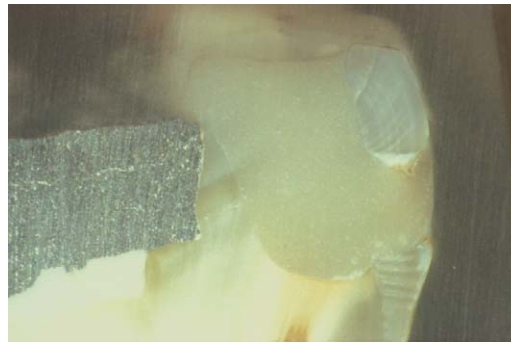

Fig. 12. The tooth shown in Fig. 11 has been sectioned to show the extent of the restoration and the remaining demineralized enamel surrounding the glass-ionomer. Because it is smooth, it will not retain plaque and is likely to heal, providing the disease has been eliminated.

possible to heal the lesion. Demineralization will still be driven by dietary substrate, but there will now also be proteolysis of the collagen framework of the dentine. At this point, some level of surgical intervention is necessary to restore the smooth surface of the tooth crown and eliminate further accumulation of plaque bacteria and their nutrients [1].

If allowed to proceed, there will be continuing production of acid, which will progressively demineralize the HA of the deeper dentine in advance of the collagen breakdown. There will be a gradation of mineral loss as the lesion penetrates, leading to a loss of texture and the uptake of stain. Standard treatment has required the complete removal of all dentine that has been affected down to a level where the dentine is regarded as normal and fully mineralized. However, providing the lesion can be isolated and there is a reasonable level of mineral present on the collagen framework, it is possible to remineralize the dentine [35,36]. This means that the lower layers

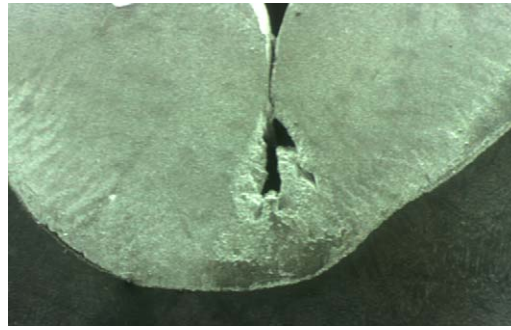

Fig. 13. A scanning electron micrograph of a molar tooth showing the complexity of a fissure. Note the narrow entry and the caries lesion in the depths of the fissure that will not be clinically visible at this point.

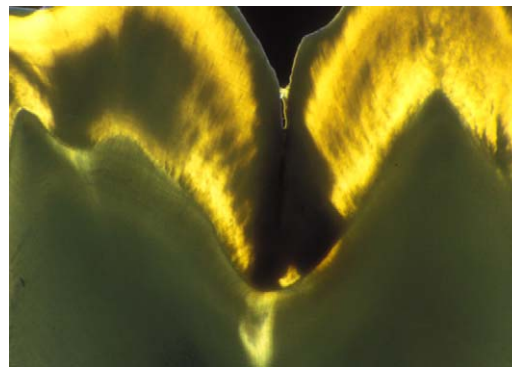

Fig. 14. The same specimen as shown in Fig. 13 illuminated by transverse light. Note the caries lesion and the translucent zone in the dentine beneath representing the pulpal response to caries.

of demineralized dentine can be retained providing the lesion can be completely sealed from any source of bacterial nutrition. Progress will be arrested, and both dentine and pulp will have the opportunity to heal [37]. The ions required for the remineralization under these circumstances can be sourced from either the pulp or a glass-ionomer restorative material that is known to release calcium, strontium, phosphate, and fluoride ions under these conditions (Fig. 15) [38,39].

The challenge

With a clearer understanding of all the factors mentioned previously that are involved in the initiation and progress of a caries lesion, it is time to think again about methods of control and treatment. It is clear that the

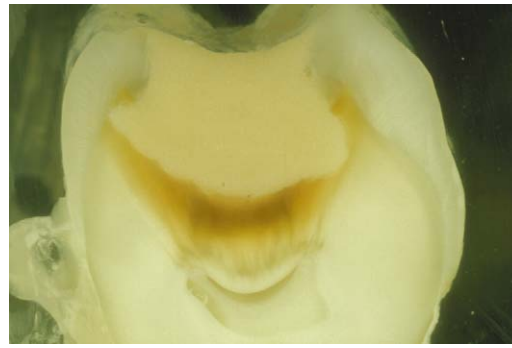

Fig. 15. This tooth was restored clinically using the Atraumatic Restorative Treatment technique and restored using a glass-ionomer as a transitional restoration. The tooth was subsequently lost so it was sectioned to reveal the presence of the affected layer at the base of the restoration. This is expected to remineralize over time.

disease commences well before the development of surface cavitation, and the profession has a responsibility to make an early diagnosis of the hidden disease. It is equally apparent that surgical treatment of a cavity will have little or no bearing on the elimination of the disease. Certainly, restoration of the smooth surface of the tooth crown will remove defects that allowed accumulation of bacterial plaque, but this will still make no difference to the combination of factors that allowed development of the disease in the first place.

The profession must learn to identify problems in the oral environment at a very early stage rather than simply seek cavities. Methods for testing these various factors are becoming more sophisticated and must be incorporated in to normal daily practice so that patients will gain the full benefit of current knowledge. Previous methods of classifying and recording the presence of caries lesions are now out of date, and modifications should be incorporated in to daily practice. It is more than 100 years since the original classification of cavities was developed by G.V. Black [40], and a variation is in the process of being adopted. The following is a brief comparison between the original system and the new classification.

G.V. Black classification of cavities

The origin of this classification goes back to the beginning of last century before the advent of magnification, good illumination, and radiographs, and well before the present sophisticated understanding of the significance of bacterial flora, saliva, fluoride, and the caries process. At that time, there was no alternative method of identifying caries other than by counting visible lesions. Even more significant, the cavity was not readily visible until the enamel surface was already broken down to some extent. In fact, most lesions were quite extensive before recognition.

The Black classification represented a system of cavity designs for restoration rather than caries lesions per se. It was not possible to remineralize the early lesion and, in the absence of an ability to control the disease by any other means, the margins of the cavity were placed out into ''self-cleansing areas'' so patients were theoretically offered the opportunity to control their own disease. This led to the initial cavity being rather larger than necessary. It was not possible to limit the extent of the involvement of an occlusal fissure system because the margin of an amalgam restoration could not cease part way along a fissure. A proximal lesion invariably meant the separation of the cusps and the weakening of the crown to the extent that a split at the base of a cusp became a common problem. The natural aesthetics of a tooth were generally lost because of the extent of the restoration and the occlusion was easily disrupted by extensive involvement of the occlusal surface, leading, undesirably, to deeper intercuspation of opposing teeth [41].

The classification itself was complicated by a need to modify the design to suit the material selected for restoration. For example, the proximal lesion

on a posterior tooth was designed for restoration with amalgam or gold and therefore incorporated retentive designs specific for those materials. On the other hand, a proximal lesion in an anterior tooth anticipated the placement of silicate cement or gold foil and therefore incorporated alternative retention patterns. One of the major deficiencies was that it was not possible to record the difference in size between the initial and the advanced lesion, and this in turn meant that there was no recognition of the increasing complexity of restorative techniques.

New classification of lesions of the exposed tooth surface

It has been suggested that, in the light of current knowledge and understanding of the disease of caries, the G.V. Black classification should now be regarded as out of date. While acknowledging the value of the innovative pioneering efforts of Black, it is time for change with the introduction of a classification that will encourage the profession to adopt minimal intervention concepts in operative dentistry.

An innovative classification, based on the forgoing concept of the progression of the disease of caries, was first proposed in 1997 [42] and modifications have since been incorporated [43,44]. It is already being taught, in parallel with the G.V. Black system, in a number of schools and countries. The following description has already been published in textbooks and scientific articles and is repeated here for further discussion [42–44].

Caries lesions occur in only three sites on the crown or root of a tooth, which are areas subject to the accumulation of plaque. Therefore, the first parameters for the classification are these three sites:

Site 1: pits, fissures, and enamel defects on occlusal surfaces of posterior teeth or other smooth surfaces;
Site 2: approximal enamel in relation to areas in contact with adjacent teeth;
Site 3: the cervical one third of the crown or, following gingival recession, the exposed root.

As caries and noncaries tooth loss represents a progressive disease, it is desirable to be able to define the size and extent of the lesion at the time of identification and therefore the potential complexity of the restorative procedures required for treatment. At the same time, the original classification can remain subject to modification after final identification of the full extent of the lesion. It is possible then to define five separate sizes as the lesion progresses.

Size 0: The earliest lesion that can be identified as the initial stage of demineralization. This needs to be recorded but will be treated by eliminating the disease and should therefore not require further treatment.

Size 1: Minimal surface cavitation with involvement of dentine just beyond treatment by remineralization alone. Some form of restoration is required to restore the smooth surface and prevent further plaque accumulation.

Size 2: Moderate involvement of dentine. After limited cavity preparation, remaining enamel is sound, well supported by dentine, and not likely to fail under normal occlusal load. The remaining tooth is sufficiently strong to support the restoration.

Size 3: The lesion is enlarged beyond moderate. Remaining tooth structure is weakened to the extent that cusps or incisal edges are split or are likely to fail if left exposed to occlusal load [44]. The cavity needs to be further enlarged so that the restoration can be designed to provide support and protection to the remaining tooth structure.

Size 4: Extensive caries or bulk loss of tooth structure (eg, loss of a complete cusp or incisal edge) has already occurred.

The Size 0 lesion is new and may be difficult to identify. The immediate treatment is to eliminate the disease and apply therapies designed to bring about remineralization. Size 1 lesions will also be new, and minimal cavity designs, followed by restoration with adhesive materials, are indicated. Sizes 2, 3, and 4 may mean a new lesion that has progressed to a considerable extent without the patient presenting for treatment or it may be replacement dentistry after breakdown of an old restoration. The same basic principles for developing a cavity design will apply in both cases, and the larger the cavity, the greater the problems in restoration and the shorter the probable longevity of the plastic restorative materials. The selection of the most suitable material for the larger restorations will be dictated by such properties as resistance to fracture and flexure as well as abrasion resistance (Table 1).

To assist in communication the relationship between Black's classification and the Site and Size concept is discussed below.

Site 1: Size 0, 1, 2, 3, and 4—pit and fissure caries

- Cavity located on the occlusal surface of a posterior tooth or any simple enamel defect on an otherwise smooth surface of any tooth.

Table 1
Diagrammatic representation of the relationship of the Site and Size concept for the description of lesions of the crown of a tooth

	Size				
Site	No cavity (0)	Minimum (1)	Moderate (2)	Enlarged (3)	Extensive (4)
Pit/fissure (1)	1.0	1.1	1.2	1.3	1.4
Contact area (2)	2.0	2.1	2.2	2.3	2.4
Cervical (3)	3.0	3.1	3.2	3.3	3.4

- Black Class I: Smaller Sizes 0 and 1 could not be performed originally because suitable restorative materials were not available so the Black classification begins with Site 1, Size 2 (1.2).

Site 2: Size 0, 1, 2, 3, and 4—approximal lesion commencing in relation to contact areas

- Cavity located on the approximal surface of any tooth (anterior or posterior) initiated in relation to the contact area between two teeth.
- Black Class II: Lesions occurring between posterior teeth only. Because of difficulties of identification and materials limitations, there was no equivalent of Size 0 or 1 so the Black classification begins with Site 2, Size 2 (2.2).
- Black Class III: Lesions occurring between anterior teeth only. Now classified as Site 2, Size 2 (2.2).
- Black Class IV: An extension of a Class III lesion involving the incisal corner or incisal edge of an anterior tooth. An alternative cause would be traumatic fracture of the incisal corner. Now classified Site 2, Size 4 (2.4).

Site 3: Size 0, 1, 2, 3, and 4—cervical lesions

- Lesion located in the cervical region anywhere around the full circumference of a tooth including exposed root surface after recession.
- Black Class V: This classification does not recognize lesions on the gingival one third of the approximal surface, particularly root surface caries, as being different from Class II lesions. An erosion/abrasion lesion or a small carious cavity on the buccal or lingual surface would be a Site 3, Size 0 (3.0) if it is expected to be arrested. If restoration is required, it would be Site 3, Size 1 (3.1). A larger caries lesion is classified as Site 3, Size 2 (3.2). An interproximal lesion will generally be Site 3, Size 3 (3.3) because of difficulty of access. The Site 3, Size 4 (3.4) classification is reserved for a complex lesion involving more than one tooth surface.

Cavity design and preparation

Black's classification did not allow for the Size 0 or Size 1 lesion in either Site 1 or 2 because, in the absence of radiographs, they could not be identified. Also, in the absence of adhesive restorative materials, Size 1 could not be repaired using minimal techniques.

There is a clear division between restoring a new lesion and replacing a failed restoration. When dealing with new active caries, the cavity design

should be very conservative because it is possible to remineralize enamel and dentine, which is only partly demineralized and not denatured and cavitated [45]. Margins need be extended only to smooth surfaces capable of remineralization, and the concept of removal of all demineralized tooth structure on the theory of extension for prevention no longer applies. Cavity outline form should be dictated only by actual cavitation of the surface, so this means it is often possible to maintain tooth to tooth contact interproximally. In fact, with the Size 1 and 2 lesion, the prime object of the restoration is simply to restore the smooth surface of the crown to prevent further plaque accumulation.

When dealing with an erosion/abrasion lesion, it is essential to diagnose and eliminate the cause to ensure longevity for any restorative material chosen for repair.

On the other hand, in replacement dentistry, the cavity outline is already defined and will often be more extensive than ideal. For these restorations, most of the principles laid down by Black will still apply, if for no other reason than tooth structure can not be replaced. In fact, for Size 3 and Size 4 lesions, very little has changed in relation to cavity design.

Whether the problem presenting is a new lesion or replacement of a failed restoration, the limitations of the physical properties of both the remaining tooth structure and the restorative material must be taken into consideration. A small restoration can be reliably supported by remaining tooth structure, particularly in the presence of adhesive restorative materials. In fact, it is claimed that a tooth crown can be restored to full physical strength by placing these materials. However, as the cavity enlarges, the tooth becomes weaker until it reaches a point where the restoration must be designed so that the restorative material will support remaining tooth structure and protect it from occlusal load. This requires modification to cavity designs and some consideration as to which material to use [46,47]. These factors are taken into account within the classification.

Summary

Extensive advances have been made in understanding the relationship between the oral environment and the bacterial disease of caries in recent years, so there should be considerable modifications in methods of treatment and control. The initiation of the disease and the progress of the hidden lesion will be surreptitious, relatively slow, and difficult to define until there is surface cavitation. But by then the disease will have been present for some time and in many ways it is too late—there has been permanent damage to tooth structure. The profession has a responsibility to learn to recognize the very beginning of the hidden disease and take steps to cure it before irreversible damage has occurred. The progress of the disease is discussed in some depth and suggestions made to encourage the

profession to recognize the earliest stage of a caries lesion so that cavitation can be prevented, not just restored.

A modified method for identification and recording of lesions is presented because it is designed to encourage the profession to recognize lesions as early as possible, thus leading at least to some extent to preservation of natural tooth structure. It is not possible to abandon the G.V. Black system immediately because so much of modern operative dentistry is performed to repair, restore, and replace old restorations. Once a cavity is prepared to fit in with this system, there is no possibility of improving the cavity design. Therefore, it will be necessary to teach both systems in parallel. However, the profession should be encouraged to recognize the benefits of minimal intervention dentistry. If all new lesions were prepared using minimal cavity designs, future problems with replacement dentistry would be minimized and natural tooth structure preserved to a far greater extent than is possible at present.

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