LITERATURE REVIEW

MANAGEMENT OF THE PRE-CAVITATION LESION

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أصبحت الخطط الوقائية ضد نخر الأسنان ناجحة الان وموكدة خارج البلاد وداخلها لدرجة أن الاحتياج للتدخل العلاجي قد قل و أصبحت المسئولية الاولى للوقاية من نخر الأسنان والرعاية بالإصابات الأولية ترجع للمريض ، يشارك فقط طبيب الأسنان في العلاج الترميمي عند معايير معينة متضمنة بعض ما جاء في قائمة هذه المستندات والتي أصبحت مقنعة .
ولأهمية المحافظة على الترميمات السنية ، يجب وضع الترميم الراتنجية الواقية (مبدأ المادة السادة اللاصقة) في الاعتبار كبديل لترميم الأملغم التقليدي صنف أول ، قد تستعمل (الترميمات الراتنجية الوقائية) .ب. . عندما يحكم على التسوس بأنه أعمق إلى طبقة العاج ويكون من غير المناسب للعلاج استعمال المادة السادة للشقوق فقط خاصة في عدم وجود ترامين تقليدية على سطح السن المعالج .

Preventive measures against dental caries are so successful today in certain countries and regions within countries, that the need for operative intervention has decreased and the primary responsibility of preventing dental caries and managing early lesions has been assigned to the patient. The dentist should only intervene with restorative treatment when specific criteria, including some of those listed in this paper, have been satisfied.

In the interest of conservation of tooth structures, the preventive dentistry restoration - the ***preventive resin restoration (PRR),*** (composite resin-sealant concept) or the glass ionomer/composite resin laminate (the so called "sandwich" or "double-laminated" technique) should be considered as an alternative to traditional Class I amalgam restorations. The PRR or sandwich technique concept can be used when the carious lesion is judged to be deeper into the dentin than is appropriate for management by fissure sealant alone, especially if no restoration exists in the tooth surface in question.

The use of sealants has spawned an entirely different concept of conservation of occlusal tooth structure in the management of deep pits and fissures early in caries involvement.

The preventive dentistry restoration embodies the concepts of both prophylactic odontotomy (enameloplasty) and extension for prevention, yet requires only a minimum or no cutting of tooth structure at the carious site. Pain and apprehension are slight, and aesthetics and tooth conservation are minimized. Several options are available in selecting preventive dentistry restorations, depending on the professional's judgement.

The first option is to simply place a conventional sealant over the ***incipient*** lesion as well as over the remaining occlusal fissure system.

The second option, advocates the use of the smallest cavity preparation, but to remove the carious material from the bottom of a pit or fissure and then use an appropriate instrument to place either sealant or composite. Sealant is then placed over the polymerized material as well as flowed over the remaining fissures. Aside from protecting the fissures from future caries, it also possibly protects the composite from abrasion.

A third option reported involves the use of a glass ionomer cement as the preventive glass ionomer restoration (PGIR). The glass ionomer cement is used only in the cavity preparation involving dentin. The occlusal surface is then etched with a gel etchant, (avoiding etching the glass ionomer, if possible). The conventional resin sealant is placed over the glass ionomer and the entire occlusal fissure system. In the event that the sealant is lost, the fluoride content of the glass ionomer will help prevent future primary and secondary caries formation.

Each of these options requires a judgement decision by the dentist. That judgement can well be based on the criterion than if an overt lesion cannot be ***visualized,*** it should be sealed, if it can be ***visualized,*** the smallest possible preventive dentistry restoration should be used along with its required sealant "topping." It was pointed out that the first option could provide the preferred model for conservative treatment of ***incipient*** and ***minimal, overt*** pit-and-fissure caries. These options would be especially valuable in areas of the world with insufficient professional dental personnel and where preventive dentistry auxiliaries have been trained to place sealants under supervision. In all cases, the preventive dentistry procedure should be considered as an alternative to the traditional Class I amalgam with its accompanying extension for prevention that often includes the entire fissure system.

Introduction

The pre-cavitation lesion is an area of the tooth where the carious process has commenced, but has not yet resulted in the breakdown of the enamel.[1] The surface will remain smooth and the lesion cannot be detected by a probe,[2] but there is a difference in colour and translucency and the lesion can be seen as a "white spot," i.e., an early carious lesion[3,4] (Fig. 1). Saliva has the potential to aid remineralization particularly if it contains fluoride ions.[5] As we are now moving towards an era of a more preventive approach, the pre-cavitation lesion can be managed so that this early lesion can be arrested or reversed.[6] Treatment requires a knowledge of the caries and remineralization processes. It involves the commitment of the dentist and the patient to surveillance and motivation. It may involve non-invasive techniques or the pre-cavitation lesion may require invasion, i.e., cutting a *"minimal"* cavity,[7] or a conventional preparation. The decision will depend on the extent of this pre-cavitation lesion,[8] and the position of the lesion on the tooth.

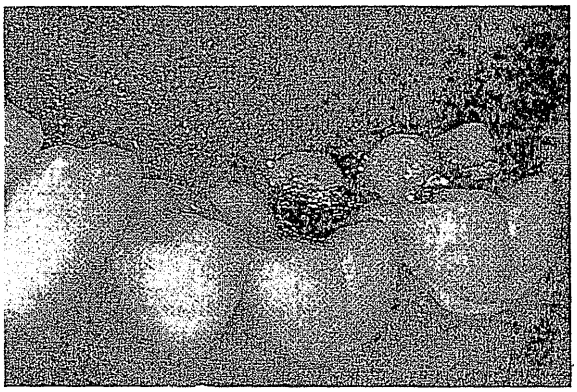

Fig. 1. White spot enamel lesions at the cervical margins of both molar teeth.

From a preventive dentistry standpoint, the early identification of the pre-cavitation (*incipient*) lesion is extremely important because it is during this stage that the carious process can be *arrested or reversed.* Clinically, it is often difficult to recognize and diagnose the early lesion and for this reason, it is important to be familiar with its features from aetiological and histologic standpoints.[9]

New concepts in restorative dentistry concentrate more on preserving the integrity of the tooth rather than filling a cavity. The use of materials possessing both cariostatic properties and long-term adhesion is changing the approach to the treatment of the early pre-cavitation lesion.

A pre-cavitation lesion starts on the enamel surface and is due to loss of minerals from the orderly arrangement of the apatite crystals in the enamel rods. The optical properties are changed, light is scattered, and the increasing porosity makes the enamel less translucent. It is seen clinically as a "white spot." Morphologically, although the lesion has an intact surface, there is subsurface demineralization.[10]

Probably the most important fact is that the surface of the enamel is relatively intact (although microscopically the surface is much more porous than sound enamel). The implication is that the *caries process can be retarded, arrested, or indeed reversed before any physical cavitation requiring clinical intervention has occurred.*

It usually takes a period of months or even years for a carious lesion to develop. Dental caries is not simply a continual, cumulative loss of material, but rather a dynamic process, characterized by alternating periods of *demineralization and remineralization.*[11] Demineralization is the dissolution of the calcium and phosphate ions from the hydroxyapatite crystals, which are lost into the plaque and saliva. In remineralization, calcium, phosphate and other ions in the saliva and plaque are redeposited in previously demineralized areas. It is possible to have demineralization and remineralization occurring without any loss of tooth mass. A *lesion results when the cumulative, negative mineral balance exceeds the rate of remineralization over an extended period.*[3] *The disease can be arrested.*[12]

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The caries process is initiated by micro-organisms which colonize the tooth surface in the form of dental plaque. As soon as the plaque is removed from any tooth, it *immediately* begins to build up again. This should not be unexpected, since by definition, dental plaque is composed of salivary residue, bacteria and their end products, all of which are always present in the mouth. Thus a good plaque control programme must be *continuous.* It must be a daily commitment over a lifetime.

Both demineralization and remineralization occur during caries development. Carious lesions develop when the rate of acid-induced demineralization of teeth exceeds the capacity of the saliva to remineralize the damaged enamel components. Following the intake of sugar, a localized demineralization of the enamel occurs as a result of the acid produced by the plaque bacteria. This negative mineral balance, if continually repeated, eventually results in a carious lesion. It often requires months or even years, for the lesion to develop.[13] During this time, under proper conditions, a compensatory remineralization of the damaged area can occur by mineral components in the saliva. There is a precedent for such a mineralization potential. Immediately after eruption of the teeth, the outer layer of the enamel is not completely mineralized; the maturing (mineralization) of this outer layer occurs within the first year as a result of being bathed in the saliva.[12] The rate at which the disease progresses varies between different individuals, and is a function of factors which include carbohydrate intake, oral hygiene, microbial and salivary conditions, the tooth surface and time (Fig. 2). The tooth surface is in a dynamic equilibrium with the saliva and with any bacterial plaque that may be present. The plaque concentrates mineralizing ions such as calcium, phosphate, magnesium and carbonates from the saliva to provide the chemical environment for the precipitation and formation of *calculus*, a concretion that adheres firmly to the tooth. If the plaque is not removed by flossing and brushing *before* the calculus begins to form, the resultant mineralized mass provides a greater surface area for even more damaging plaque accumulation. Calcium, phosphate and other ions, may pass one way or the other.[14] Progression to caries or balance of ionic exchanges towards remineralization depends on the above factors.[15] Whether to cut and fill the pre-cavitation lesion, or to manage and remineralize it, depends on accurate diagnosis, on the position of the lesion on the tooth surface assessment of the question - *Is there an active caries, i.e., is it progressing or can it be arrested?*[3]

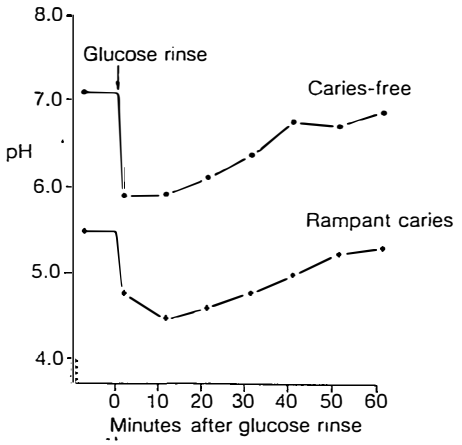

Fig. 2. Changes in the pH in plaque following a glucose rinse (Stephan Curve). The fall in plaque pH is greater in a person with rampant caries than in a caries-free individual because the former has not only a high but frequent consumption of sugar.

The conditions for optimum remineralization are the *same* as for preventing the initiation of a lesion:

(1) plaque control to reduce the negative effects of bacterial acidogenesis
(2) sugar discipline to minimize the number of acidogenic episodes and
(3) the use of fluorides that potentiate the remineralization process.

Thus with the same primary preventive dentistry routines, an individual can simultaneously protect teeth into the *future* and compensate for limited *past* damage.

Diagnosis implies more than just recording the number of cavities, their location and appearance. It is necessary to know whether the patient is likely to develop new cavities and whether existing cavities are likely to progress. Only then can preventive and operative treatment be prescribed suitable to the needs of the individual patient.

Diagnosis requires clean dry teeth, good ligh-

ting, radiographs and sharp eyes. A probe to feel with, not to penetrate with, will be required and perhaps dental floss.[6] Assessment of any lesion will depend on the age of the patient, the number of lesions, diet and to some extent, time[6] (Fig. 3). The clinician must decide at the first visit of a patient whether the disease is *incipient;*[17] this may require constant monitoring.[17,18]

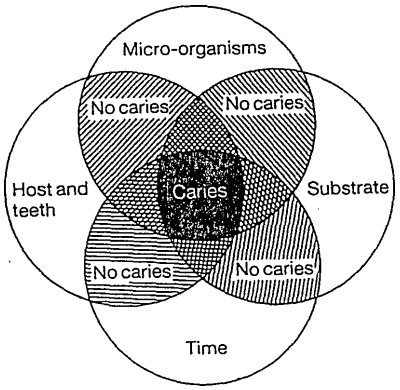

Fig. 3. The four circles represent the interplay of the aetiological factors in dental caries. All four factors must be acting simultaneously for caries to occur.

Smooth surface caries, which arises on intact enamel surfaces other than at the location of the pits and fissures can be divided into free-smooth-surface caries (i.e., caries affecting the buccal and lingual tooth surfaces) and approximal caries, affecting the contact area(s) of adjoining tooth surfaces (i.e., mesial or distal surfaces).

On the buccal and lingual surface of a tooth, the white spot may be localized, or it can extend along the entire gingiva, sometimes involving multiple teeth. Interproximally the *pre-cavitation* lesion usually starts as a small round spot immediately gingival to the contact point and then gradually expands to a small kidney shape, with the indentation of the kidney contour directed coronally.[18] In fissure caries, the initial lesion comparable to the white spot usually occurs *bilaterally* on the two surfaces at the *orifice of the fissure* and eventually coalesces at the base.[19] Occasionally, lesion formation begins along the wall of the fissure or at the base, either *unilaterally or bilaterally.*[20]

The diagnosis of an approximal lesion is best seen with radiographs and it must be borne in mind that carious lesion is larger in the tooth, than it actually appears on a radiograph.[21] An initial lesion is not detectable radiographically, and it is not until further demineralization has occurred that it is visible on an x-ray. *There will still be no cavitation.* Although a bitewing radiograph will not reveal the full extent of a lesion, it gives some indication of zoning. Approximal caries is usually a slow disease process. However, the clinician should bear in mind that there are always exception to the rule and treatment of patients should not be ruled by statistics, which sometimes are skewed. How do we interpret the radiographic appearance of the carious lesion? Clearly, where no lesion in the enamel is apparent, no treatment is required. Where radiolucency is confined to the enamel, preventive measures should be instituted. Lesions that reach the amelodentinal junction or are spreading laterally in dentine need more consideration. It could be argued that a lesion just entering the dentine should be left and remineralizing procedure adopted. However, the radiological appearance will not reflect the true histological state. This lesion is the most difficult to treat and requires close monitoring with bitewing radiographs if it is to be controlled. In these cases, a year can make a difference since the caries has penetrated dentine where it can range far and wide. For this reason, if the patient cannot be examined regularly, it is better to treat the lesion with the minimal cavity approach.

Where caries in the dentin is visible on a radiograph, often the enamel is cavitated and has reached a point of no return. Monitoring of these lesions with bitewing radiographs teeters on the brink of disaster because failure to diagnose correctly can result in either pulp exposure or fracture of marginal ridges - with all its attendant problems in the future.

Interproximal caries can sometimes be diagnosed by very careful looking it may be seen as a shadow between the teeth which appears as grey or pinkish discoloration (Fig. 4) It can sometimes be detected by transillumination or with floss. (If the floss shreds and there are no interproximal fillings there, then there is probably cavitation). Progression of a smooth surface lesion may regress if it is on a buccal or lingual surface and it may disappear. If it is a smooth surface

interproximal lesion, the progression may take three to four years to proceed through enamel.[6] Progress through dentine is much more rapid[4] and once the lesion has reached or passed the dentino-enamel junction, diagnosis is important, whether an invasive approach is used or not.

Fig. 4. A carious lesion is present on the distal aspect of the first premolar tooth. The lesion is shining through the marginal ridge which shows a pinkish gray discoloration.

It is recommended that the following guidelines be used for treating the pre-cavitation lesion:

1. When enamel is sound on bitewing radiograph, treatment should not be done.
2. When radiolucency is confined to the enamel, preventive measures and attempt to remineralize should be instituted.
3. When radiolucency is confined to the enamel but has reached the amelo-dentinal junction, it should be monitored closely with bitewing radiographs and, if lesions are progressing, restoration via minimal cavity preparation should be done.
4. When radiolucency has entered the dentine, and the patient has a high caries rate, treatment should be instituted immediately via minimal cavity preparation. When caries incidence is low, monitoring through bitewing radiographs should be carried out. If the patient is not available for regular inspection, restoration should be performed immediately.
5. When radiolucency in dentine is close to the pulp, restoration should be done immediately; where possible, use a minimal occlusal cavity approach and always line with calcium hydroxide.

In all cases, preventive measures should be introduced. An improvement in oral hygiene can often make a dramatic change and tilt the balance toward remineralization.

Pit and fissure lesions can be diagnosed by sight also. A pre-cavitation lesion may show up as a white spot lesion, or a brown discoloration in the sides of the fissure, where there may be opacity. A cavity may not be visible, even though caries is present. However, lesions will show up with carefully taken and standardized radiographs[22] (Fig. 5). The progression of pit and fissure lesions depends on the initial diagnosis (it may have been the structure of the fissure and not a cavity) but once caries has started in a fissure or pit, it can progress rapidly.[23]

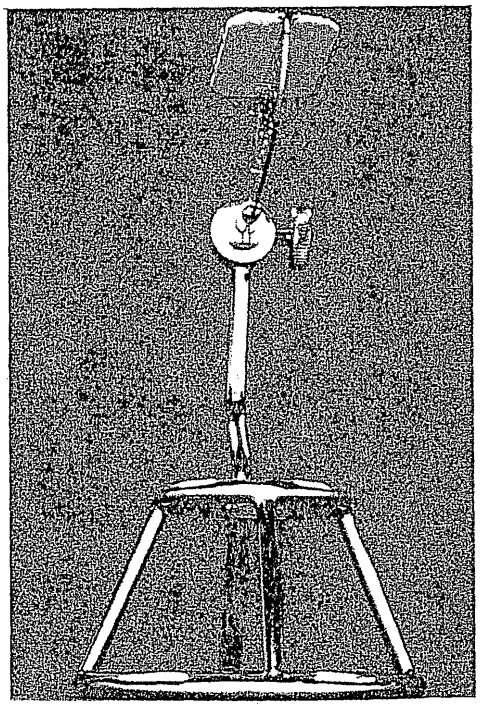

Fig. 5. The Backer-Dirks holder to aid in the production of reproducible bitewing radiographs.

Fluorides are highly effective in reducing the number of carious lesions occurring on the smooth surfaces of enamel and cementum. Unfortunately, fluorides are not equally effective in protecting the occlusal pits and fissures where 95% of all carious lesions occur.[24] Considering the fact that the occlusal surfaces constitute only 12% of the total number of tooth surfaces, it means that the pits and fissures are approximately

eight times as vulnerable as the smooth surfaces.[25] Historically, several solutions have been tried to deal with the deep pits and fissures on occlusal surfaces.

Management of the pre-cavitation lesion is either invasive or non invasive and the decision depends on the individual clinician and the position of the lesion and is also based on whether the tooth is sensitive to hot or cold or sweet foods and whether the pulp is in jeopardy. Smooth surface lesions on accessible surfaces can be treated preventively. Smooth surface lesions interproximally must be treated according to the depth of the lesion on the radiograph. There is a difference of opinion between certain experts. Elderton 1985[3] suggests that once a lesion has progressed to half way through the dentine, the lesion should be treated by invasive techniques. Kidd 1984[22] and many other clinicians would treat a pre-cavitation lesion that has penetrated to, or just beyond the dentino-enamel junction, by cutting a cavity, perhaps by using a *minimal restoration* (Fig. 6) or a *tunnel* preparation[26,27] (Fig. 7). The reason for this is that the bacteria and their products can diffuse down the dentinal tubules and damage the pulp. Also, the rate of progress through dentine is much less predictable, but at a much rapid pace as mentioned previously, that it is through enamel. More research is needed and certainly great care when a decision is made at this stage of the progress of dental caries. If the lesion is in enamel and it is decided that the process will be arrested, then a programme for careful monitoring and motivation should be implemented.

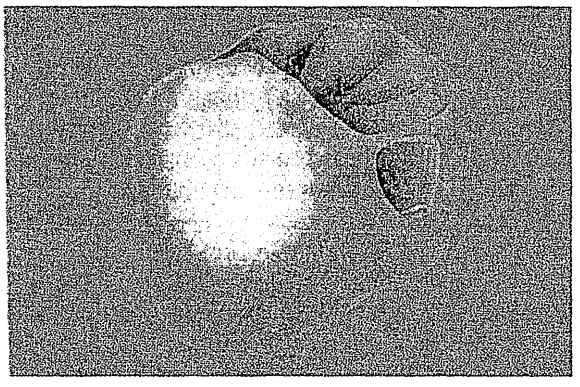

Fig. 6. Approximal Class II microcavity using lateral marginal ridge approach.

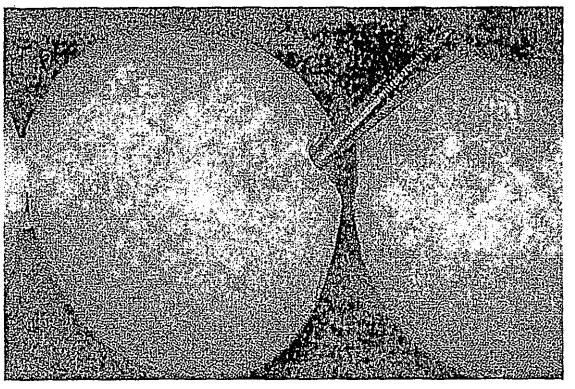

Fig. 7A. Access gained to approximal lesion using a small round diamond bur.

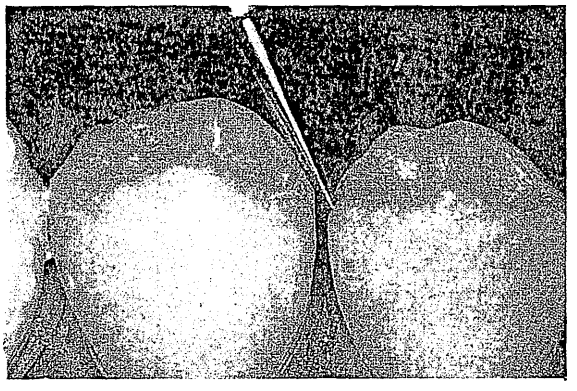

Fig. 7B. Where caries is present in adjoining tooth, access is easily gained via cavity opening in the Class II micro-activity.

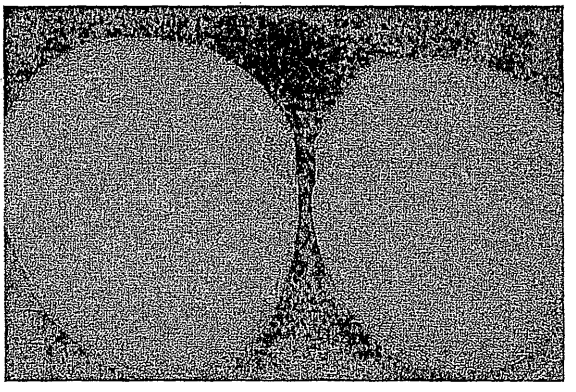

Fig. 7C. Completed Class II microcavities after surface conditioning with 15% polyacrylic acid.

For smooth surface approximal lesions in low stress bearing area, glass ionomer cement was first suggested in 1980. This required a micro-preparation in which the proximal caries was removed either via a buccal or lingual opportunity approach or, when accessible, below the marginal

Fig. 7D. Matrix band burnished over cement to develop contact area.

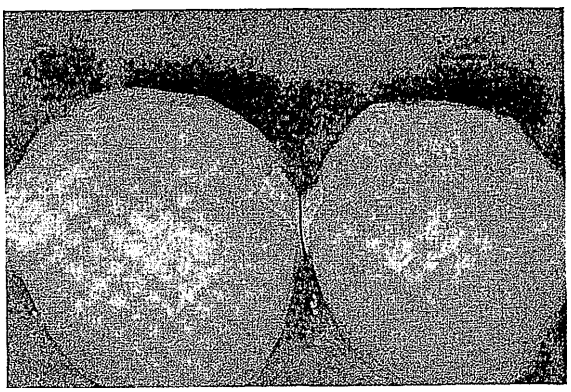

Fig. 7E. Completed restorations after finishing. Apply a coat of light cured bonding agent as an added protection against moisture contamination.

ridge. These approaches preserved and supported the still intact marginal ridge.[7] In 1984, an occlusal approach in which entry was made internally through the fossa, preserving the marginal ridge and removing caries through a tunnel preparation was described.[25] Many clinicians are now adopting these principles of non-destruction of marginal ridges in their treatment of early approximal lesions.

The assessment of caries risk is made from a detailed history and clinical and radiographic examinations. If there is a history of repeated restorations and there are numerous carious lesions, the patient is likely to be at risk. Even more information is gained if the dentist has the opportunity of examining the patient regularly, perhaps every six months, over a number of years. If no new lesions develop and existing "early" lesions remain static and/or darken in colour, it can be concluded that caries risk is currently low and the intervals between examinations may be lengthened. However, many other factors such as the periodontal condition, salivary flow and buffering capacity (Bartlett & Wilson)[28] should be taken into account in arriving at this decision.

The patient's age is also relevant to caries risk. Enamel caries occurs most frequently in young people while later in life, caries risk is often reduced. Reasons for this reduction may include changes in diet and oral hygiene as the patient becomes more mature. However, older people can develop new carious lesions if such factors as diet or salivary flow are altered.

Since diet is one of the main factors in the development of dental caries, a dietary history is an important part of the assessment in patients with a high caries activity. A diet sheet on which the patient is asked to record everything taken by mouth for a seven day period can be a useful record. Such a diet sheet may show the frequency of sweet drinks, sweets and other pre-bed sugar containing snacks which are a potential cause of caries in the particular mouth (Fig. 8).

The maintenance of good oral health requires a partnership between the health professional and the patient. No preventive programme can be a success unless the patient participates in a home self-care regimen to supplement office care programme, with the level of success being proportionate to the amount of participation. Maximum participation can be expected when the patient knows *what* to do, *how* to do it and above all has the motivation. To adhere to recommended procedures, educational strategies can be used to teach facts and skills, but these are useless without motivation. Motivation can be initiated by an individual based on some need or desire, or it can stimulated by persuasion from external sources. With or without motivation, learning is best achieved in sequential steps. As an individual accumulates facts, the facts merge into concepts and ultimately into values, which in turn engender motivation. Motivation will aim towards:

- Elimination of the carbohydrate substrate. Fortunately, complete elimination of sugar from the diet is not necessary to prevent caries. Relatively simple measures, such as reducing the frequency of consumption by confining sugar to meal times and using a sugar substitute in drinks are usually sufficient.

Diet Analysis

	Thursday		Friday		Saturday		Sunday	
	Time	Item	Time	Item	Time	Item	Time	Item
Before Breakfast	7.30	Tea*	7.00	Tea	7.30	Tea*	7.05	Tea*
Breakfast	8.00	2 Wheat slices 2 Crisp break 1 Apple Coffee*	8.00	2 Wheat slices 2 Crisp bread 1 Apple Coffee*	8.30	2 Wheat Slices 2 Crisp bread 1 Apple Coffee*	8.05	2 Wheat slices 2 Crisp bread 1 Apple Coffee*
Morning	9.00	Polo	10.00 11.30	Murray mint Tea* Biscuit	11.15	Tea*	10.00 12.30	Lemon Barley Tea*
Mid-day Meal	12.30	Meat roll Tea*	2.00	Steamed fish Parsley sauce Boiled Potatoes	1.45	Sausage, Boiled Potatoes Ice Cream, Tinned fruit	1.40	Roast lamb, Potatoes, Cabbage, Carrots
Afternoon	2.00 5.30	2 Cream Crackers 1 Dairy Tea Tea* 2 Shortbread biscuits Tea*	2.45 6.00	Tea* Tea*	2.30 5.45	Tea* Tea*	2.00 4.00	Tea* Tea*
Evening Meal	8.00	Chop, leeks, boiled potatoes Choco-ice Tea*	8.30	Bacon Sandwich Tea*	7.30	Fried kipper bread and butter	8.15	Ham salad Bread and butter Tea*
Evening and night	1.00	Horlicks* Biscuits	10.00 1.30	Peanuts Horlicks* Biscuits	9.15 1.45	Chocolate Horlicks* Biscuits	1.15	Horlicks* Biscuits

Fig. 8. A diet sheet completed by a middle-aged patient with a high incidence of caries.
The frequent sweet drinks, sweets and the pre-bed sweet drink and snack are potential cause of caries in this mouth.
Note the frequency of sugar intake eight times per day.
+ 2 spoons of sugar

- Increasing the host resistance by use of fluoride. Deep pits and fissures can be made more resistant by obliterating or "sealing" them with fissure sealants. Fortunately, the fate of potentially pathogenic bacteria inadvertently sealed in dental fissures has been shown to be in the favour of the host. They turn to spore form, and in time decrease in number. A limited number of bacteria may persist in some lesions but do not appear capable of destroying tooth structure under those circumstances. Indeed, the carious lesion may ultimately become sterile. There is therefore convincing evidence that fissure sealants are capable of arresting the caries process.[29]
- Elimination of bacterial plaque. Theoretically, a plaque-free tooth surface will not decay, but complete elimination of plaque from some areas (e.g. fissures) is not possible, and from other areas not always practical (e.g. approximally, where plaque elimination requires the skillful use of floss). However, in other areas (e.g. cervically) effective plaque control by proper tooth brushing will prevent caries.[4]

The site and the size of the lesion must be recorded. The lesion must be demonstrated to the patient. Fluoride should be applied to the lesion and a preventive programme must be personalized for the patient concerned. The patient must be made aware that it is his lesion and his responsibility to help in arresting it. Dietary advice should be given with emphasis on eliminating sugar intake between meals as far as possible. In addition to brushing twice daily with a fluoride toothpaste, it has been suggested by Elderton 1985[23] to encourage the patient to apply fluoride toothpaste directly "to the lesion twice daily for say a month." The patient should also be encouraged to remove all plaque build-up, particularly at that site. It should be arranged to

reassess the patient at regular recall intervals, for example monthly, or bi-monthly, and act as necessary.

The management of pit and fissure lesions by watching and waiting is fraught with danger. It is, therefore, thought inappropriate to do nothing. If caries is visible on the radiograph occlusally, it must be filled, even if there is no physically visible cavity. The choice between an amalgam, composite or glass ionomer filling will depend on the dentist and the conditions for the individual patient. If caries cannot be detected on the x-ray, or clinically, but it is thought that the tooth might be carious, then a fissure sealant should be applied (Figs. 9 & 10). As long as the sealants are retained, no bacteria or bacterial acids can affect the sealed areas. If they are not retained, no damage to the teeth will result from the treatment. The lost sealant can be easily replaced. Sealants should be checked regularly; visually, and radiographically to see that it stays and that it does not leak and decay. There will always be doubt about the presence of bacteria in a sealed restoration, but studies have shown that in small lesions these do not progress.[28,29] In cases where there is no cavitation, but an investigation with a small round bur reveals a very small amount of decay (the size say, of a number one or two round bur), it is suggested by some[30] that a preventive restoration be placed. This is a glass ionomer filling which has been sealed with an unfilled resin. The resin protects the surface of the glass ionomer, until it sets and seals the fissure, and the glass ionomer material leaches fluoride ions into the tooth surface and offers protection from further acid attack. This type of filling is well tolerated by children, and has proven very successful with certain operators, but not with others (so far as its retention, not its clinical properties).

Conclusion

Diagnosis and assessment of pre-cavitation lesions requires great care. Further, the monitoring of the treatment requires flexibility from both dentist and patient. Regular recalls will be necessary to review the situation. Factors taken into account at each visit should include oral hygiene, diet, past and present caries experience, perhaps further caries susceptibility tests, and standardized radiographs taken at intervals of once a year initially, and later every two years or longer. This is a decision that will vary according to individual needs without overexposing the patient.

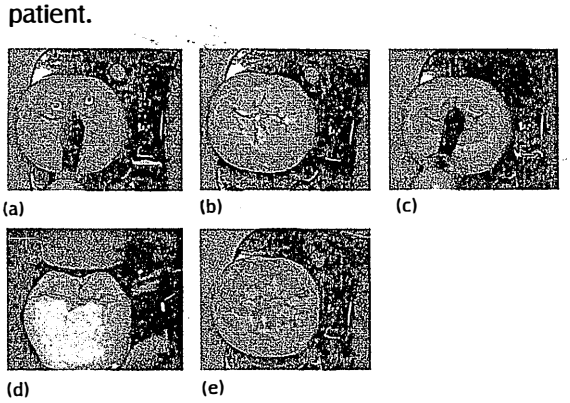

Fig. 9

(a) A brush is used to apply the etchant gel over the occlusal surface of the tooth to be fissure sealed.
(b) Dried etched enamel appears matte, white and frosty.
(c) The sealant is applied to the etched surface using a small disposable brush or a syringe with a disposable tip.
(d) Light curing a fissure sealant.
(e) A fluoride-containing varnish is applied to the etched enamel at the periphery of the restoration where it has not been covered with sealant.

If at the time of the dental examination, emphasis was placed on searching out the incipient lesions for caries (the white spot), preventive strategies could be applied to induce a reversal of the disease process. It is essential that both the profession and the public realize that the biologic `repair' of incipient lesions is a viable alternative to later treatment.

Even when primary preventive dentistry fails, tooth loss can still be avoided. In practice, the early identification and expeditious treatment of caries greatly minimize the loss of teeth. When such routine diagnostic and treatment services are linked with a dynamic preventive dentistry programme, tooth loss can realistically be expected to be reduced significantly.

Should the profession of dentistry be able to control caries effectively through plaque control, systemic (ingested) and topical (local application) use of fluorides, dietary control and use of sealants and preventive dentistry restorations, two important questions need to be asked:

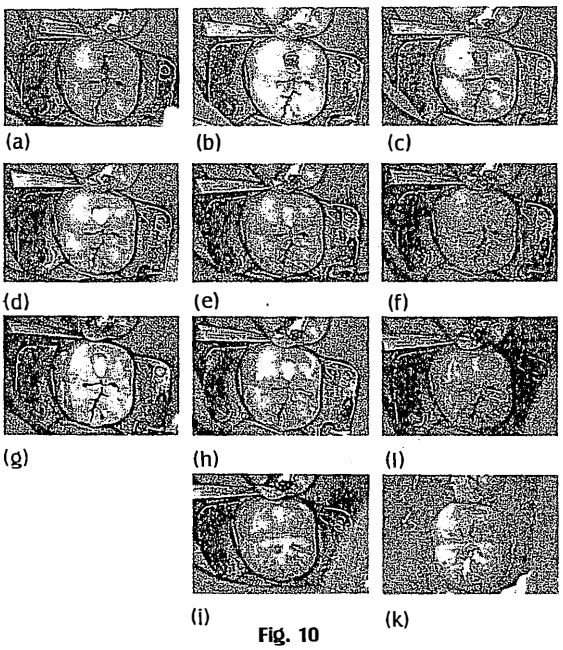

Fig. 10

(a). A lower second molar isolated with rubber dam prior to placing a sealant restoration.
(b). Enamel is removed to gain access to obvious caries.
(c). Soft caries over the pulp is removed.
(d). Exposed dentin is covered with a calcium hydroxide-containing cement.
(e). A second layer of glass ionomer lining may be placed in a deep cavity.
(f). The enamel walls of the cavity and the occlusal surface of the tooth are etched with acid.
(g). Etched enamel after washing and drying.
(h). Bonding resin is applied to the cavity walls and occlusal surface.
(i). The cavity is filled with composite resin. Use of the glass ionomer lining in this tooth allowed the composite to be placed and cured in a single 2 mm increment.
(j). A fissure sealant is applied to the whole occlusal surface.
(k). The occlusion is checked with articulating paper which will locate areas of occlusal contact with a coloured mark.

1) Why do we not have a more effective dental caries control programme?
2) If daily toothbrushing and flossing of teeth remove plaque, why are these simple procedures not used effectively to control caries?

Probably the best answer to these questions is that people must first know that they need to do something as well as how it is to be done. Unfortunately, the public has relatively little information about the tremendous potential of primary preventive dentistry for reducing the ravages of the plaque diseases. Without this information, it is difficult to convince people that they can greatly control their own dental destiny. Instead, people think of dentistry as a treatment-oriented profession that specializes in periodontal treatment, restorations, endodontics, oxodontics and prosthetics. Thus an expanded public education programme is essential to ensure the success of any preventive dentistry programme in which an individual or a community is asked to participate.

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Acknowledgement

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