[Review]

Implications of Remineralization in the Treatment of Dental Caries

Theodore Koulourides, D.M.D., M.S.

Professor of Dentistry
Senior Scientist, Institute of Dental Research
University of Alabama School of Dentistry
University of Alabama at Birmingham

1.0 INTRODUCTION

1.1 THREE STAGES IN THE EVOLUTION OF DENTISTRY

Historically, dentistry evolved in three stages (Figure 1). The first stage was tooth pulling. Usually barbers or physicians treated the toothache by extracting decayed teeth. This required considerable skill to operate in the least painful way. The usual training of the tooth pullers was through apprenticeship.

Around the beginning of the 19th century restorative dentistry emerged as the second stage. This new period in dentistry involved the development of instruments for drilling the tooth and the formulation of appropriate filling materials. In addition to skill, the restorative period required scientific knowledge; part of this effort was directed towards a better understanding of the etiology and treatment of oral disease and the development of new dental materials. Dental schools offered the training needed for the profession.

Currently, we live in the transition period toward a third stage, that of disease prevention. In addition to science and skill, this stage of dentistry requires the capacity to teach the patient the etiology of disease and the rationale for the preventive treatment. With a better understanding of the disease, people are more likely to comply with the instructions for prevention. Thus, communication between dentist and patient becomes an important factor in preventive dentistry. The dentist must motivate the patient to improve his diet and oral hygiene, and the patient must understand the etiology of dental disease. Dental caries can be controlled quite effectively when the patient cooperates with the dentist.

1.2 CONTENTS OF THIS ARTICLE

This article is mainly a review of our studies of the tooth factor in caries evaluated with both *in vitro* and intraoral experiments. It will include:

1. Background information on the interaction of the tooth with its fluid environment (saliva, diet, and plaque fluid).

THE EVOLUTION OF DENTISTRY

Stages of :	Needed Attributes :
Tooth Pulling	Skill
Restorative Dentistry	Skill, Science
Preventive Dentistry	Skill, Science, Communicaton

Fig. 1. The Evolution of Dentistry

FACTORS IN DENTAL CARIES

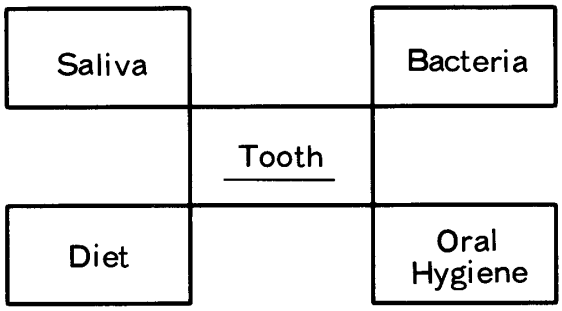

Fig. 2. Factors in Dental Caries

2. Experiments with acids *in vitro* on the development of posteruptive tooth resistance.

3. The intraoral cariogenicity test (ICT) that we designed in our Dental Research Institute for studies of caries in the human mouth.

4. The therapeutic management of dental caries.

5. Recommendations for the practice of clinical preventive dentistry.

1.3 FACTORS IN CARIES (Figure 2)

Caries is a multifactorial disease that includes the interaction between 5 cardinal factors, the TOOTH, SALIVA, BACTERIA, DIET, and ORAL HYGIENE. The presence of fluoride in the diet and in agents of oral hygiene is of particular importance to caries. Dramatic changes in any of the above factors, while relatively rare, are very influential in caries causation. Certainly, no caries is possible with out teeth. Decrease of salivary secretion, as in xerostomia, is associated with rampant caries (Brown et al. 1975). The amount and frequency of sugar consumed is another established causative factor(Newbrun, 1967 ; Gustafsson, et al, 1954). Caries is considered by some investigators as a specific infection by *Strep. mutans*, but the ubiquitous presence of these bacteria in the mouth of most people weakens the theory of classical infection. There is no single microbial species associated with caries. Bacteria constitute the physiologic lining of our gastrointestinal track, and in most cases, serve a useful function. Undoubtedly, *Strep. mutans* is one of the prime candidates responsible for the production of acid that dissolves the tooth mineral, but it is not the only possible acidogen.

The saliva／bacteria／tooth interaction is usually in a state of dynamic equilibrium that can be deflected into a pathogenic relationship by predominance of the attacking factors, mainly the acid produced at the plaque／tooth interface. This fluid at the tooth surface is either saliva or the fluid within the plaque which is modified by the diet and microbial fermentations. For example, with sucrose entering the plaque the bacteria will produce acids and lower the pH near the tooth surface 6.6—7.4 (pH of saliva) to a level below 5.0, indicating 100 times the acid (H^+) activity of saliva. With

the slow diffusion of salivary buffers through the gelatinous plaque, such acidities may cause enamel demineralizaton, the first step to caries. It is obvious that frequent removal of the plaque will bring a healthier fluid (i. e., saliva) to the tooth surface interface and reverse the process of demineralization by remineralization. One very pertinent point on the pathogenicity of caries is whether the tooth surface can acquire sufficient resistance to ward off the damaging effect of the produced acid ; in such a case the acidogenic bacteria on the tooth surface become innoccuous parasites, part of the physiologic oral flora. A regular and thorough practice of plaque removal contributes to caries prevention by adjusting the dynamic equilibrium with a lower cariogenic attack (Axelsson et al. 1976).

Normally, caries is not the result of drastic changes of one of the above five factors, but the result of slight or pronounced changes of a combination of factors. In a hypothetical case, a patient may consume a high sugar diet and neglect plaque removal. The combination of these two factors can be devastating for the teeth unless measures are taken to counteract the cariogenic attack with intensive fluoride therapy which may increase the tooth resistance in areas of challenge.

1. 4 THE HUMORAL CAUSATION OF DISEASE

Philosophically, I like the very old Humoral theory on the pathogenicity of disease. Expressed by a Greek philosopher, Embedocles, of the 5 th century B. C., the theory suggests that, before organic disease occurs, there is an imbalance of the fluids circulating in the body. At that time, they recognized four body humors : blood, phlegm, yellow bile, and black bile. The balanced proportion of these humors maintained health, while local or general disturbance of the proportions of the four humors caused disease. For example, a person with a high proportion of black bile was melancholic.

Transferring the idea of humors to the present day knowledge about dental caries, we can express it in terms of saturation and undersaturation of the plaque fluid with respect to tooth mineral. The bacterial plaques shield the tooth surfaces from the protective effect of saliva. Furthermore, the acid production by the bacteria, when supplied with fermentable sugars, lowers the pH of the plaque fluid and causes tooth demineralization. Our effort, therefore, should be directed toward the development and maintainance of a healthy equilibrium between the tooth and its fluid environment. As long as the mineral loss and mineral gain are equal there is no decay, even though various ions pass across the interface in both directions. An important aspect of this dynamic equilibrium is that, by changing the tooth composition, the ionic exchange can improve the properties of the tooth surface. If, with every cycle of demineralization and remineralization, the redeposited mineral is less soluble than the mineral lost, the challenged tooth surfaces become increasingly more stable to an acid environment ; the problem of caries will be diminished to the extent that these areas become resistant to

the challenge.

It is also pertinent to remember that normally caries develops locally only on areas of the tooth surface that shelter the bacterial plaques from the natural oral defense, such as the salivary flow and the cleansing effect of mastication. In these areas the plaque fluid becomes pathogenic and causes tooth demineralization. Our task in caries research is to demonstrate the optimal conditions related to the development of maximal tooth resistance.

2.0 THE SCIENTIFIC EVIDENCE

2.1 ENAMEL REMINERALIZATION

The possibility of repair of early carious lesions through remineralization was suggested by Joseph Head in 1910. Other dental investigators suggested remineralization for early caries (Andressen, 1919 ; Ruthrauff, 1923), but the lack of scientific information on the tooth／oral fluid equilibrium did not favor acceptance of remineralization in the practice of clinical dentistry. Our knowledge of the equilibrium between calcified tissues with their fluid environment has improved remarkably with extensive research in the last 50 years. The following are some of the cardinal demonstrations that clarified our knowledge regarding the dynamic mineral／tissue fluid equilibrium and the stability of tooth mineral related to caries resistance.

In experiments using powdered enamel and dentin exposed to organic acids, Volker (1939) showed that fluoride treatments reduce the acid solubility of tooth mineral. By grinding successive layers of enamel from the surface inwards, Brudevold and coworkers (1960) found that fluoride concentrations are higher at the surface than in inner enamel, and that the surface layers are more insoluble than those of inner enamel. Studies of the composition of arrested caries lesions indicated an increased fluoride incorporation in relation to normal enamel, again indicating the connection between fluoride and some kind of defense reaction based on mineral exchange that favors retenton of in soluble mineral (Little et al. 1962). Actually, acid resistant zones within lesions were demonstrated repeatedly with studies of sections with micro radiography, microhardness, and polarized light (Gustafsson, 1957 ; Kostlan, 1962 ; Crabb, 1972).

Another typical characteristic of arrested caries lesions was their high content of organic material considered to be part of the tooth defense against caries (Bibby, 1932). There are reasons to believe that this organic material within the lesion, or an enamel pellicle on the tooth surface, may contribute to the increase of mineral stability by blocking the active sites of crystal dissolution. The outer enamel layer of carious lesions was studied extensively by Von der Fehr (1967). By abrading the outer surfaces of teeth, he demonstrated the influence of the oral environment, and of fluoride treatments, on the formation of this surface layer of enamel.

A new approach to studying the enamel/fluid equilibrium was introduced by Pigman and coworkers (Caldwell et al. 1957) who used the study of enamel hardness change as influenced by demineralizing and remineralizing fluids. The advantage of this approach was that it enabled the investigator to study the mineral flux in and out of the lesion with a very sensitive technique applied directly on enamel surfaces. The direct measurements make it possible to make repeated assessments of the demineralizing/remineralizing influences of biological and synthetic fluids in contact with the test surface. It would have been extremely difficult to obtain this information from chemical analyses of these fluids due to difficulties in sampling and to their composition, which includes the same elements as those of the dissolving/reforming enamel mineral. Furthermore, most other techniques for studying changes of enamel density are destructive, and can be used only once on the same specimen. For these reasons, we adopted the microhardness technique for our *in vitro* and *in vivo* studies of the tooth/fluid mineral equilibrium (Koulourides, 1980).

This paper presents the highlights of investigations of enamel remineralization conducted at the University of Alabama School of Dentistry over the past 25 years.

2.2 THE *IN VITRO* AND *IN VIVO* EXPERIMENTAL MODELS

2.21 THE TEST OF ENAMEL MICROHARDNESS: The typical subsurface lesion of caries indicates that the chemical reactions in the mouth tend to preserve the outer layer of enamel through remineralization and the formation of acid resistant minerals at the surface, even though the acid penetrates through the resistant layers and dissolves the underlying susceptible minerals. For the measurements of surface microhardness, a diamond indenter of precise geometry is pressed onto the test surface with a preselected load (in most of our experiments we used 500g). Because of the lack of elastic recovery, the test leaves a permanent deformation on the surface which is measured with a microscope. The length of indentations, or the calculated depth (Length : 30.5), reflects the degree of microporosity of the superficial layers of enamel as affected by the various test fluids. The test is non-destructive, therefore the microhardness of the specimens can be measured repeatedly on enamel surfaces that are homogeneous enough to respond similarly to demineralizing and remineralizing activities of their environment. This is an important requirement of the experimental design because two indentations cannot be placed on the same spot of the test surface.

Figure 3 shows a picture of an enamel surface tested for microhardness at various stages of the experiment. The small indentation at the extreme left represents the condition of enamel initially, while the next is large, indicative of extensive softening after 2 days immersion in an acid buffer. The other 3 indentations represent readings at 2, 4, and 6, of mineralizing treatments. The decreasing size of these indentations indicates progressive remineralizaton of the subsurface lesion. The mineralizing environ-

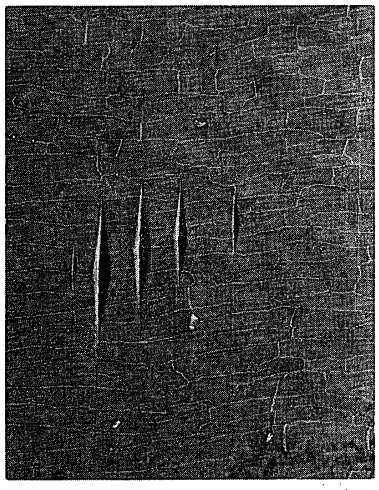

Fig.3. SEM picture of enamel surface microhardness demonstrating softening and rehardening.

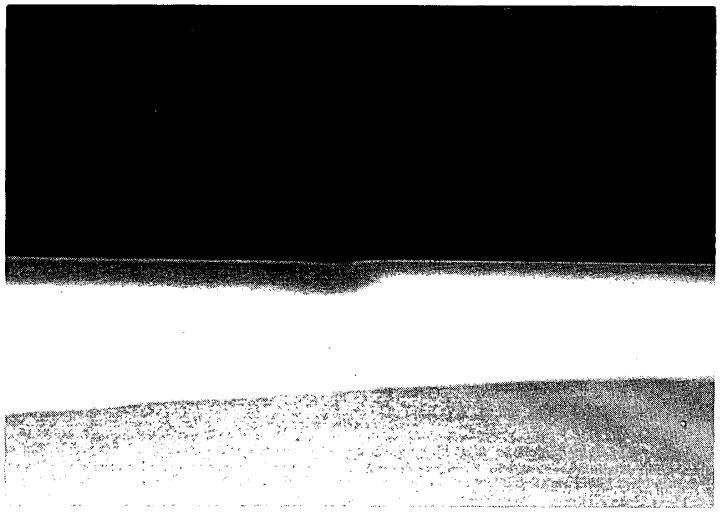

Fig.4. Microradiograph of abraded bovine enamel exposed to the acid buffer for 4 days (right) and 8 days (left).

ment forms new crystals, or assists the growth of old ones, thus reducing the microspaces in the subsurface of enamel. The increased density presents higher resistance to indenter penetration reflected in smaller indentations.

2.22 MICROADIOGRAPHY : Figure 4 is a microradiograph of a lesion on bovine enamel. The right side was exposed to the acid buffer pH 4.0 for 4 days and the left side for 8 days, with buffer changes every 2 days. Areas of high density appear white in contrast to those of low density which appear black. Notice that the lesion has subsurface characteristics, and that it is parallel to the test surface, indicating that the demineralizing activity had a similar effect all across the test surface. This pattern is typical with bovine enamel when the outer surface (approximately 100−200 um) has been removed. With human enamel, the variability from one site of the test surface to the other is greater. For this reason, it is better to use bovine enamel in experiments assessing the demineralizing and remineralizing acivities of synthetic and natural fluids.

2.23 EFFECT OF FLUORIDE ON ENAMEL REMINERALIZATION : One of the most important findings of the remineralization studies using the microhardness test was the effect of trace amounts of fluoride. This is shown in Figure 5. In the acid environment, enamel softens ; in a mineralizing environment, it rehardens. Fluoride added to mineralizing solutions accelerated the remineralizing process by 3 to 4 times, even at the low concentration of 1 ppm (Koulourides et al, 1961). Furthermore, fluoride incorporation increaes the enamel resistance to a subsequent scid attack.

2.3 *IN VITRO* ACID RESISTANCE OF ARRESTED CARIES

Our *in vitro* experiments addressed two aspects of the mineral equilibrium between the tooth surface and its environment. The first aspect was the natural tooth resistance of early caries that had been arrested and formed the yellow or brown spots on

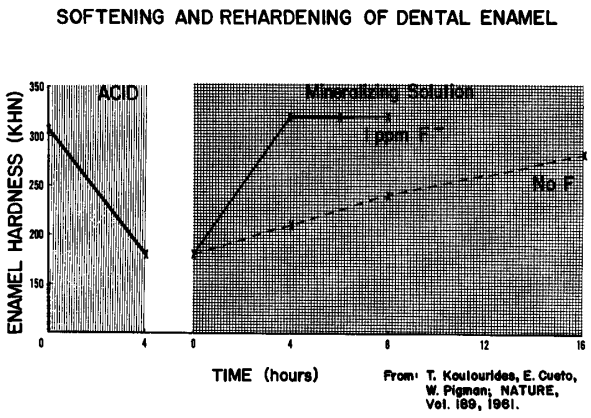

Fig. 5. NATURE

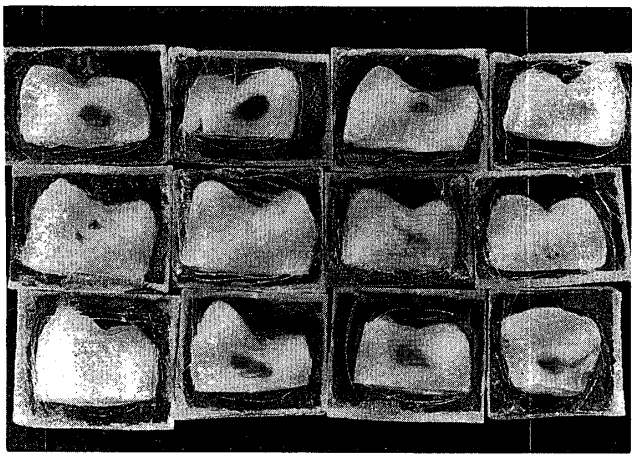

Fig. 6. A group of approximal surfaces cut from extracted teeth with consolidated lesions.

the attacked surfaces. The second was the experimental replication of tooth resistance with repeated fluoride treatments and acid exposures. For the first project, surfaces from extracted teeth with naturally consolidated lesions and adjacent sound enamel (Fig. 6) were exposed to acid buffers (0.01 M lactic acid containing 3.0 mM Ca and 1.8 mM P at pH 4.0) for one week. Then the teeth were sectioned and microradiographed (Koulourides and Cameron, 1980). Bibby (1932) called these areas consolidated lesions. Consolidated lesions contain higher organic material and higher fluoride, and are more resistant to acids than normal enamel (Little et al. 1962 ; Stack, 1964).

Figure 7 is a microradiograph of a section cut through a consolidated lesion that had been subjected to acid demineralization for one week. Clearly, the acid produced more severe demineralization on the sound enamel above and below the consolidated lesion. The shape of the consolidated lesion approaches a triangle similar to the shape of carious invasion in approximal areas of the teeth. Apparently, the reactions of lesion consolidation create families of mineral of different resistance to acid. The presence of resistant mineral within the lesion is an important reaction, because it indicates a natural way of caries arrestment. In a way, the reaction can be considered as local immunization ; the tissue resistance increases to the point that the acid produced by the bacterial plaques will not be sufficient to advance the lesion on the consolidated areas. The process of lesion consolidation is the equivalent of "wound healing", although the reactions are mainly chemical, whereas in wound healing the connective tissue cells play a major role in the formation of the new matrix.

2.4 LESION LAMINATIONS

The formation of zones of high acid resistance within lesions has been demonstrated experimentally by periodic treatments of teeth with fluoride during periods of exposure to acid buffers (Koulourides, 1977 ; Koulourides, 1983). Figure 8 illustrates the reac-

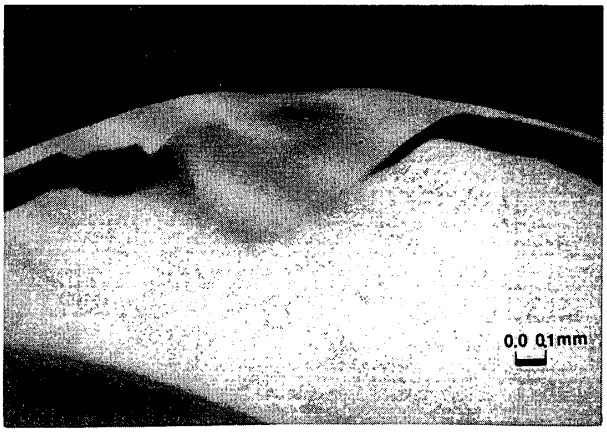

Fig. 7. A typical microradiographic picture of a consolidated lesion and sound enamel above and below the area of consolidation.

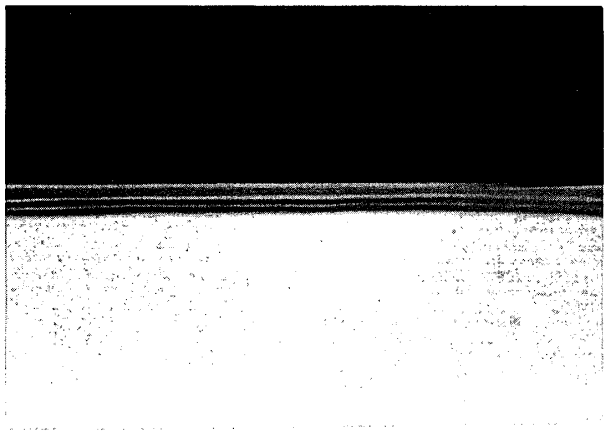

Fig. 8. Lesion laminations produced "*in vitro*" by 4-minute intermittent treatments with 2% NaF and 2-day exposures to the lactic acid buffer.

tion leading to the formation of resistant mineral within lesions. Each of the three laminations in the lesion was produced by a 4-minute NaF treatment followed by a 2-day immersion to acid buffer. Apparently, the fluoride treatments converted a layer of enamel mineral at the tooth surface to the resistant family minerals, while the subsequent 2 day in acid caused the first body of the lesion by dissolving deeper layers of mineral unaffected by the fluoride treatment. A similar reaction was repeated with the second and third fluoride treatment, with the end result of three laminations of resistant mineral formed at the demineralizing front.

The lamination phenomenon points out another very important aspect of tooth resistance to decay. The enamel minerals affected by the fluoride treatment are so resistant that even three continuous 2-day exposures to strong acid (pH 4.0) did not cause dissolution of the acid resistant zones. This is apparent from the fact that the first and third laminations do not differ appreciably in mineral density, although the first lamination received the acid treatment three more times than the third. Conceivably, with frequent fluoride treatments, the resistant laminations would form a continuous thick layer of resistant mineral, a barrier to the acid penetration toward the susceptible minerals of the inner enamel. If the tooth mineral could become resistant to such a strong acid (pH 4.0 acting for 2 days), most likely it would also be immune to the transient attack of natural caries. Actually, in natural caries the formation of a salivary pellicle on the tooth surface and incorporation of organic material within the lesion may be additional factors contributing to higher resistance of consolidated lesions. High organic material within the lesion may buffer the acidogenic activities and also provide pathways of penetration of fluoride to crystals located in deeper layers of enamel.

3.0 THE INTRAORAL CARIOGENICITY TEST (ICT)

The ICT was developed in an effort to study the interactions between the tooth

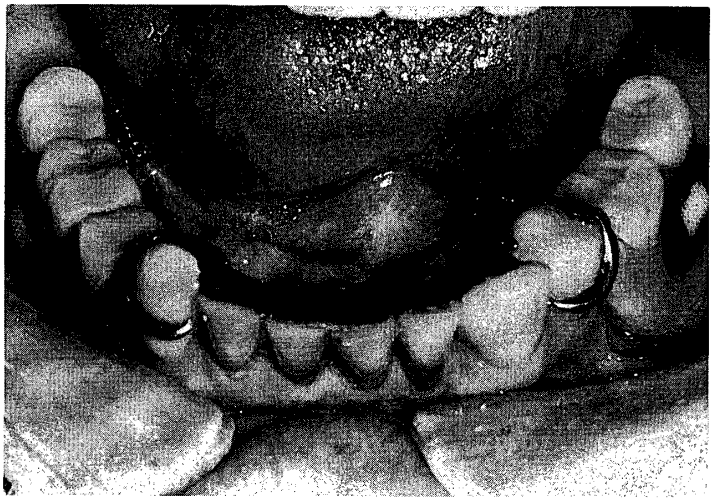

Fig. 9. A picture of a partial denture used for the Intraoral Cariogenicity Test (ICT).

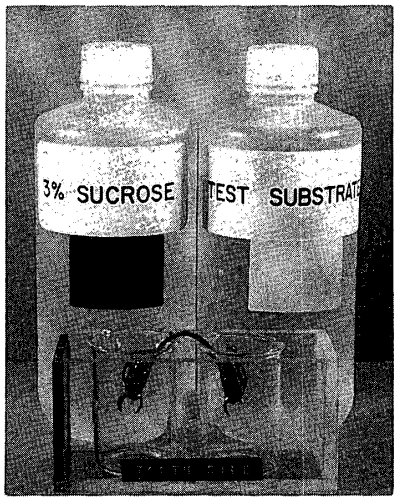

Fig. 10. The immersion box designed for the evaluation of two regimens, on the left and right experimental sites, in the same experiment. The two regimens can be two types of sugar, or two different fluoride treatments.

and its oral environment in the human mouth (Koulourides and Volker, 1964). Enamel slabs excised from human or bovine teeth are mounted with a Dacron mesh cover in partial dentures (Figure 9). The bacteria retained on the mesh and supplied with suger cause exerimental caries on the test surfaces within one week of intraoral exposure. The extent of caries is measured with the test of enamel surface hardness before and after the ICT. The cariogenic activity can be enhanced without damaging the subject's teeth by asking the subject to immerse his denture into sugar slutions for specified times and frequencies. Actually, two sugars have been compared simultaneously by immersing one side of the denture to the test sugar and the other side to the reference solution (usually sucrose) for 10 minutes 4 times daily (Figure 10) (Koulourides et al, 1976). In this manner the softening of the enamel in the two experimental sites will reflect the cariogenicity of the two sugars superimposed on that of the diet. Similar experimental designs can be applied for the comparison of two topical fluoride treatments, each applied to one experimental site. Therefore, intraoral experiments can be designed for testing various parameters, such as the cariogenic potential of subjects, the cariogenicity of sugars, the cariostatic effectiveness of fluoride, the effect of frequent plaque removal, etc.

In order to evaluate tooth resistance developed by the conditions of the ICT experiment, the slabs recovered from the 1-week ICT exposure and the various fluoride treatments are exposed to either a second, prolonged ICT period, or to acid buffers *in vitro*. The demineralization damage to the enamel is assessed with measurements of microhardness, and finally with microradiography of thin sections of the slabs.

The ICT provides very important information on the intraoral demineralizing or remineralizing activity.

3.1 THE EFFECT OF A NaF MOUTHRINSE ON NORMAL AND PRESOFTENED ENAMEL

In this study we evaluated a 0.02 % NaF mouthrinse applied as an extraoral immersion for 1 minute twice daily for its effect on normal and presoftened enamel (Koulourides and Housch, 1981). The enamel slabs, presoftened on one half of the test surface for 16 hours (in 0.01 M lactic acid/sodium hydroxide buffer in 1 % CMC containing 3.0 mM Ca and 1.0 mM P, and adjusted to pH 4.0 and 37 degrees C., 20 ml/3×5 mm slab), were mounted in recesses in partial dentures, as shown in Figure 9. A Dacron gauze cover was used to enhance plaque-like microbial retention on the test surface. The subjects were the appliance at all times, except for the immersions in the assigned solutions and the daily cleaning (taking care not to brush the test site). In order to enhance the subjects' cariogenic activity, two ten-minute immersions into 3 % sucrose were assigned, one at mid-morning and another at mid-afternoon. The intraoral test lasted one week. Then the slabs were recovered and exposed to *in vitro* acid for an additional week in order to assess the enamel resistance acquired during the intraoral exposure. The demineralizing/remineralizing reactions were followed with microhardness tests after the ICT and after days 1, 3, and 7 of the acid resistance test (ART). Separate slabs were used to assess the incorporation of fluoride on fully presoftened slabs.

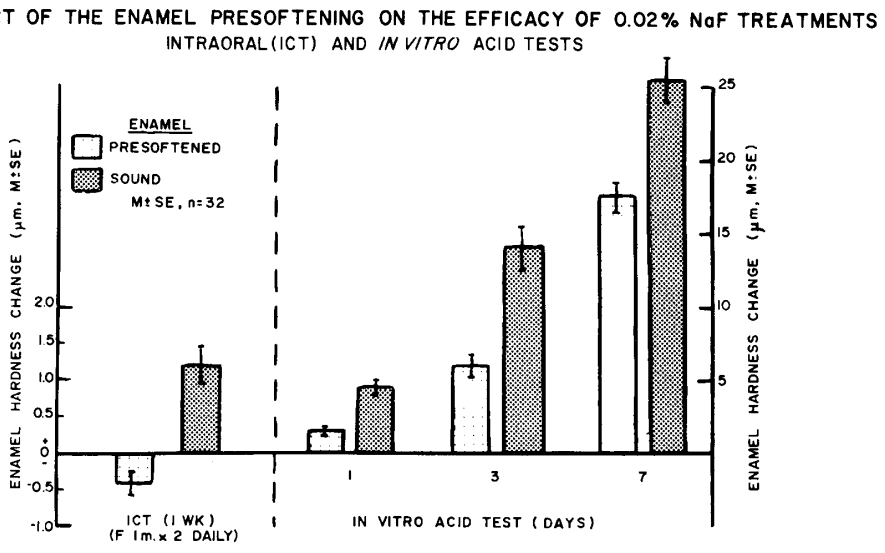

Fig. 11. Microhardness data demonstrating a higher benefit for presoftened than the normal enamel from the two daily 1-minute immersions to 0.02% NaF solutions.

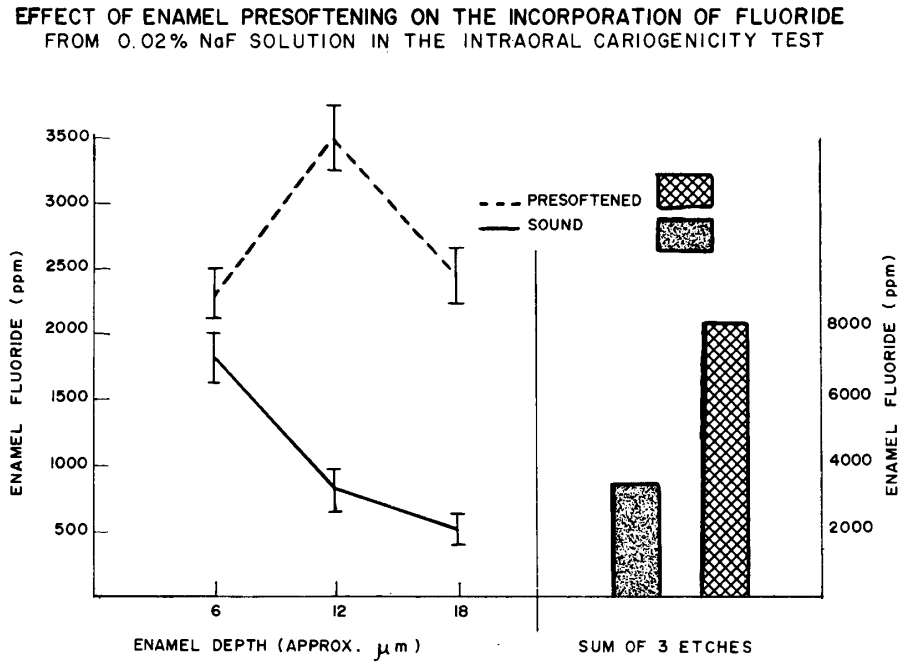

Fig. 12. Fluoride biopsies of presoftened and sound enamel exposed to the ICT and the 0.02% NaF immersions.

Figure 11 presents the change of enamel microhardness of the normal and presoftened sides of the slab. During the ICT, there was some softening on the normal side and a minor remineralization on the presoftened side. During the test of acid resistance, the presoftened side was consistently more resistant than the normal enamel. Since both normal and presoftened enamel were exposed to the same solutions, these results indicate that the remineralization, which was more intense on the presoftened enamel, contributed to the formation of a resistant mineral in the microspaces created by the presoftening.

Figure 12 shows the pattern of fluoride incorporation. Presoftened enamel incorporated more fluoride than normal enamel during the remineralization reaction ; particularly in the subsurface (about 20 μm from the surface) where there were more microspaces available for the formation of new crystals. Figures 11 and 12 present a good association between the remineralization, fluoride incorporation, and the resultant higher resistance of the remineralized enamel to the *in vitro* acid.

Figure 13 shows the typical picture of increased tooth resistance on bovine slabs exposed to the ICT for one week with fluoride and then to *in vitro* acid for another week. One half of the surface was presoftened in acid prior to the exposure to the ICT. The surface preservation on the normal enamel is barely perceptible, while the presoftened enamel (on the left) shows a wide mineral dense zone of resistant new minerals over the main lesion. Also, the severity of the lesion is greater on the normal side than it is on the presoftened enamel. The different distribution of mineral density within the lesion is indicative of the resistance that was acquired from the previous

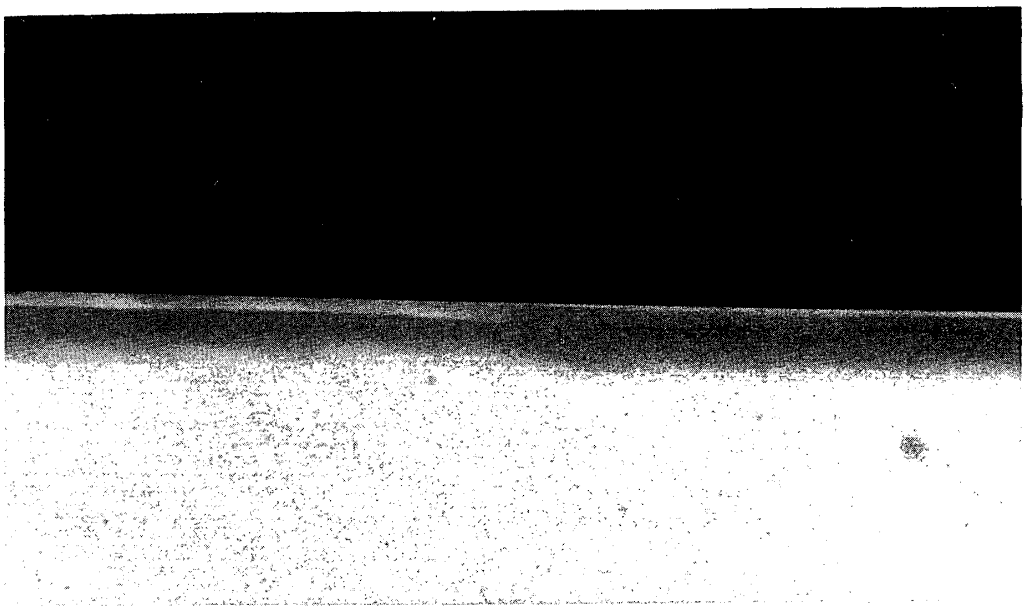

Fig. 13. Microradiograph showing subsurface lesions on one half presoftened bovine enamel exposed to the ICT and fluoride.

intraoral exposure and the influence of fluoride immersions. The dark zone of the microradiograph indicates acid dissolution of the susceptible original enamel mineral that was unaffected by the fluoride treatment.

This experiment demonstrates that the presence of 0.02% NaF (about 90 ppm F) at the experimental site for 1 minute twice a day had a very pronounced effect on the demineralizing-remineralizing equilibrium and on the composition of enamel, particularly the part softened in acid prior to the ICT exposure. Apparently, the highly dense normal enamel does not benefit from the fluoride treatment to the same extent as the porous presoftened enamel. The difference was also obvious in the microradiographic study.

3.2 EXPERIMENTAL LESION CONSOLIDATION

For the *in vivo* experimental demonstration of the fluoride effect in lesion consolidation, ICT experimental caries were developed in one week on one half of the surface of human enamel slabs by covering the other half with nail varnish in order to protect the surface from cariogenic activity. After the one-week ICT, the nail varnish was removed and fluoride was applied to both normal and decayed (primed) enamel for 4 minutes in a way similar to clinical applications of fluoride. Then the slabs were returned to the mouth for an additional 4 weeks of ICT caries, after which they were recovered, sectioned, and microradiographed.

Figure 14 shows the enamel surface with the varnish removed after one week of cariogenic priming ; the "whitish" area on the left is indicative of an early lesion, while the translucent area on the right is normal enamel. Figure 15 is a microradiograph of lesions at the end of this 5-week experiment. It shows that the primed (decayed) side of the enamel surfaces receive much more benefit from the fluoride treatments than the

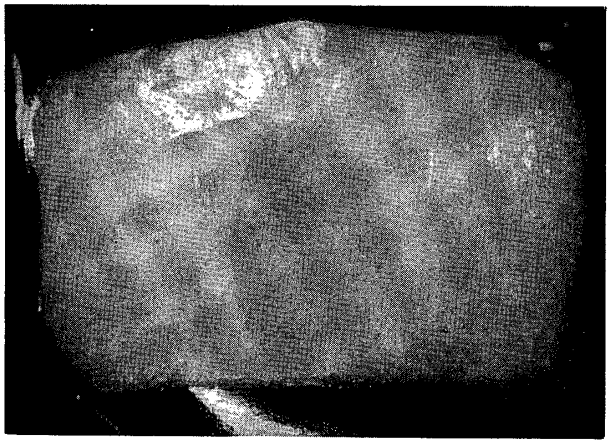

Fig. 14. Picture of human enamel specimen cariogenically primed on one side with 1 week of ICT exposure.

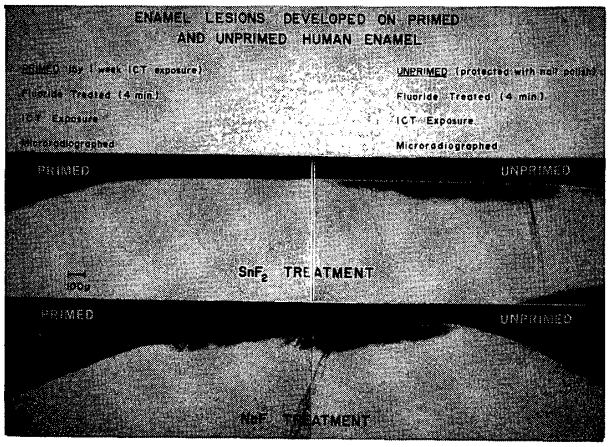

Fig. 15. Microradiograph of human enamel specimen cariogenically primed (1 week ICT) vs unprimed enamel treated with fluoride and then re-exposed to the ICT for 4 weeks.

normal enamel (Koulourides et al, 1980).

The aforementioned two experiments demonstrated that both the acid and the ICT cariogenic priming of enamel enhance the protective effect of fluoride treatments, because they provide the microspaces for the formation of the resistant fluoride—rich mineral. In subsequent experiments, we observed that the increased tooth resistance is associated with a thicker outer layer of the subsurface lesion (Koulourides and Housch, 1983). Apparently, this outer layer is a measure of the extent of formation of resistant mineral as a result of the fluoride treatment and intraoral remineralization.

The reactions in enamel remineralization have been the subject of extensive investigations in numerous research centers. It has been demonstrated with both microradiography and polarized light microscopy that the remineralizing reactions fill microporosities and form crystals within the lesion that are different from those of the original enamel. Presumably, these crystals contain more fluoride and present higher resistance to acid dissolution (Johansen and Olsen, 1979).

3.3 THEORY OF ENAMEL ADAPTATION TO THE CARIOGENIC CHALLENGE

The above described observations led us to the theory of enamel adaptation to cariogenic challenge, illustrated in Figure 16 (Koulourides, 1977). According to this theory, the intraoral interactions between the tooth, the cariogenic challenge, and the remineralizing activities will selectively remove soluble minerals from the tooth and incorporate insoluble ones, as long as the appropriate ionic components (such as fluoride) are present in the fluid environment during the remineralization phase of the reaction. The mineral exchange may increase the resistance of the tooth surface to an extent sufficient to ward off the subsequent cariogenic challenges. Essentially, the adaptation

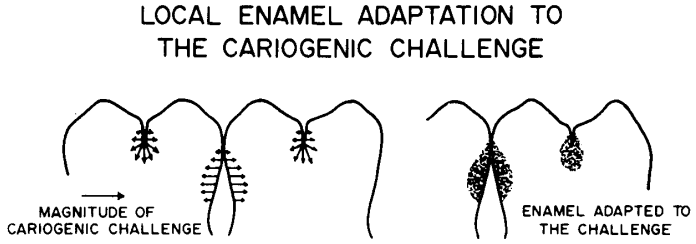

Fig. 16. Diagramatic representation of enamel adaptation to the cariogenic challenge.

reaction is like local immunization of the tooth surface in the areas of cariogenic challenge.

4.0 THE THERAPEUTIC MANAGEMENT OF CARIES

This experimental evidence indicates that there is a stage in the development of caries that can be treated therapeutically. The evolution of carious lesions from the initial enamel demineralization to the subsurface lesion and the open cavity varies widely in different individuals, and even from tooth to tooth in the same mouth. An open cavity can occur within one or two months after tooth eruption (as observed soon after eruption of third molars in cases of poor oral hygiene), or it may develop over a period of several years and possibly be arrested through the process of lesion consolidation.

4.1 CLINICAL DIAGNOSIS OF LESION CONSOLIDATION

The clinical methods of examination for caries are based on visual, tactile (explorer), or radiographic criteria. Early enamel lesions appear as white spots on smooth surfaces and give the impression of "stickiness" when the explorer is used in pits and fissures. Approximal lesions are usually diagnosed in bitewing radiographs showing the typical conic invasion towards the dentin. Lesion consolidation does not completely eliminate the above signs. Apparently, the mineral deposited during remineralization does not reconstitute the original density of enamel. Areas with consolidated lesions are softer, but, as mentioned earlier, they are usually more resistant to acid than adjacent enamel unaffected by caries. Also, in the radiographic examination, the difference in mineral density between active and consolidated lesions is not sufficient to overcome variables in angulation and the procedures of photographic developent. With time, the smooth surface lesions regain some translucency, but they seldom disappear ; actually in areas of food/plaque stagnation, they become yellow or brown by the incorporation of stains from the diet or bacterial products. For these reasons, the best criterion of lesion consolidation is the lack of progress in longitudinal studies. Since the public is exposed to numerous sources

of fluoride, and fluoride is preferentially deposited in lesions, time alone is a factor in favor of lesion consolidation.

In view of the possibility of lesion consolidation, the use of the explorer for the "stickiness" test needs to be re-evaluated. A sharp explorer pressed in pits and fissures will crush the fluoride-rich, outer layer and create grooves that may favor both the retention of bacteria and the acid dissolution of the newly exposed, susceptible enamel. For these reasons, it seems preferable to use only the non-invasive, visual and radiographic examination and try to judge the lesion severity from the discoloration associated with the progress of demineralization to the DEJ. The recently introduced fiber optic illumination may improve our non-invasive diagnosis of early caries and enable us to define the point of irreversibility. Perhaps an appropriate dye that will stain early dentinal caries will further facilitate the diagnostic procedure.

4.2 THE TREATMENT OF EARLY CARIES

Our recommendations for the management of early caries, based on the theory of enamel adaptation to the cariogenic challenge, are :

1. Patient education on the causation of dental caries and the means available for prevention and treatment of early lesions.

2. Reduction of cariogenic challenge by optimizing plaque removal and proper dietary habits. These aspects need to be individualized according to the level of patient education and his willingness to consistently follow required recommendations. The instructions for young children should include the parents since their interest in the oral health of their children and their persistent supervision are necessary components for success of a preventive program. The improvement of oral hygiene can be monitored on each patient by recording the plaque and gingival indices, as well as using the Snyder test which seems to be responsive to the amount and frequency of sugar consumption. In addition to their objective value, the above tests are good motivational tools serving as mental rewards for a worthwhile effort.

3. Increase of tooth resistance by frequently providing fluoride in the fluid environment. The fluoride can be provided by (a) fluoridation of water supplies, (b) professional applications of concentrated fluoride solutions, and/or the use of agents of oral hygiene that contain fluoride. Systemic administration of fluoride will contribute to increased tooth resistance by incorporating fluoride in enamel during the stages of mineralization. Although this is not within the context of our main topic, i. e. caries arrestment, it is an important aspect of a preventive program. In areas where the drinking water is not fluoridated, fluoride tablets should be prescribed, at the doses recommended by the American Dental Association, during the periods of tooth development and prior to eruption. After tooth eruption, the protective effect of systemic fluoride seems to be minor compared to the local effect provided by topical applications, toothpastes, mouthrinses,

or chewable tablets.

4. Intensive topical fluoride therapy should be used in conditions of high cariogenic challenge. Such conditions may involve excessive sugar ingestion, poor plaque removal, or deficiency of salivary secretion. Patients with dry mouth (xerostomia) may require concentrated fluoride gels (1% NaF) applied daily with custom made trays (Brown et al, 1976). The amount of fluoride swallowed from such applications is considerable, but since most of these patients belong to the elderly group, there seems to be no adverse systemic effect. Undoubtedly for younger patients the possibility of fluoride toxicity should be considered when fluoride is taken from many sources. The maximal safe amount of fluoride for each age has not been defined as yet, but, in my opinion, doses of less than 5 mg/day from all sources of ingested fluoride are not likely to have toxic effects for a person over 10 years of age.

4.3 CLINICAL STUDIES

Several clinical studies support the conservative, or therapeutic, treatment of carious lesions, even if they are clinically evident (Backer Dirks, 1966; Axelsson et al, 1976; Koulourides and Axelsson, 1976; Kolehmainen and Rytommaa, 1977; Ostrom, 1978).

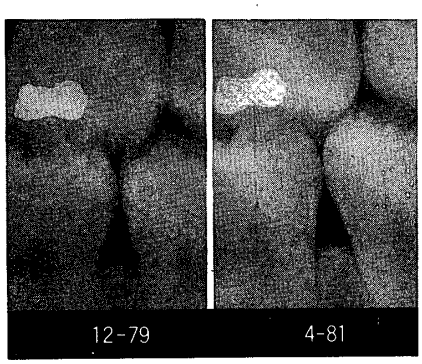

Fig. 17

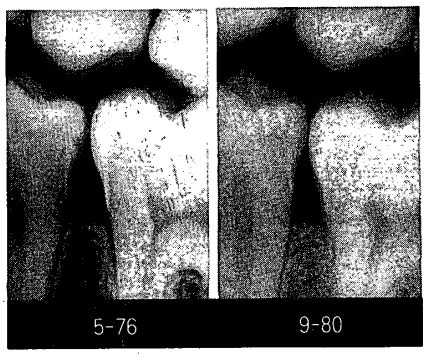

Fig. 18

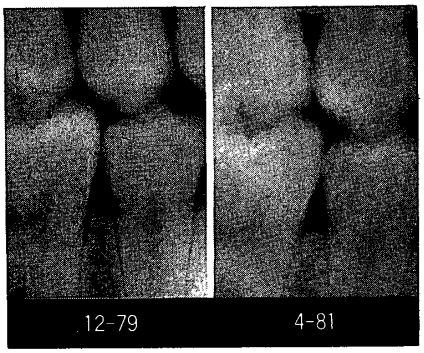

Fig. 19

Fig. 17, 18, and 19. Bitewing radiographs from patients that followed our preventive dentistry program. Most of the incipient lesions showed no progress in our 6-month recall examination.

Figures 17, 18, and 19 show some cases of approximal lesions that we followed for at least four years with biannual examinations and applications of APF gel. There is no appreciable difference in the appearance of consolidated vs early active lesions. As discussed in section 4.1, the main criterion for differentiation is the lack of progress of a lesion in successive radiographs. The majority of these lesions shown in the sequential bitewing radiographs became arrested, or progressed so slowly that the hazard of endangering the pulp within one or two years was minor. However, it should be remembered that the subjects demonstrated compliance with our recommendations for maintaining good oral hygiene. Undoubtedly, the chances of lesion arrestment will be poorer with non-complying patients.

5.0 RECOMMENDATIONS FOR THE PRACTICE OF CLINICAL PREVENTIVE DENTISTRY

In the Preventive Dentistry Clinic of the University of Alabama School of Dentistry in Birmingham, a program was designed by Dr. Carl Ostrom (1978) and associates for patients selected on the basis of their motivation to practice good oral hygiene. Initially, these patients receive five 1-hour instructional visits. These visits are spaced over five consecutive week so that they give the dentist or dental hygienist the opportunity to follow the progress of the patient in improving his oral hygiene habits. Each visit has a specific focus of instruction, (1) proper flossing, (2) proper brushing, (3) dietary habits, (4) possible restorative work, and (5) the final evaluation of the achieved behavioural change. At each visit attempts are made to solve the problems of the individual patient, problems caused either by his lack of understanding of the principles of prevention, or by his lack of skill in carrying out the recommended disciplines. For home care the program recommends toothbrushing with a fluoridated toothpaste after meals, the use of a fluoride mouthrinse once daily (before bed), and flossing once daily. Even with partial compliance with above recommendations, the degree of success is pronounced.

Psychological principles of recognizing and praising the patient for achieving the desired behaviour are extremely effective in improving the compliance of a large number of patients. Like in any other motivational problem, the personality of the motivators (dentists ad hygienists) and their interest in oral health as perceived by the patient are strong factors in the success of the preventive program.

After this initial 5-week program, the patient is assigned to an individualized recall system, which for most patients is every six months. During the recall visits, the patient's compliance is reevaluated on the basis of plaque and gingival index, and the presence of new lesions, or the progression of the lesions marked for observation. Then the patient receives the needed scaling to remove calculus, a prophylaxis, and APF applications. Every two to three years, bitewing radiographs are taken to assess the progression or regression of approximal lesions. Occlusal lesions are assessed visually after blowing the debris with compressed air.

As with any transition, the acceptance of a preventive dentistry philosophy and practice will require changes in the organization of the dental office. The treatment planning should not be based only on the objective criteria of clinical and x-ray examinations, but also on the degree of patient compliance to the prescribed regimen for home care. However, the classification of patients should not be made before an adequate effort to educate, motivate, and train them to adopt better habits of oral hygiene and diet.

Further investigations are needed for a better understanding of lesion consolidation. We will need a better definition of the degree of lesion severity that can be treated therapeutically, and of the optimal fluoride therapy needed for treating the individual patient.

6.0 SUMMARY

Our review included experimental investigations and clinical observations on the importance of local tooth resistance in caries initiation. Together, the combination of research and clinical observations constitute a powerful tool for better understanding of and treatment for disease. All evidence indicates that the tooth resistance may improve in response to cariogenic challenge. If the fluid composition on the tooth areas at risk to decay is conducive to the formation of insoluble mineral, the tooth can become resistant, thereby exhibiting localized immunity against the cariogenic attack ; but the fluid composition in the areas at risk is strongly influenced by both professional and homecare regimens. To mention only the fluoride factor, topical applications in the dental office and the use of fluoride-containing agents for homecare will increase the localized tooth resistance by consolidating incipient lesions. But since the requirements for lesion consolidation include the compliance of the patients, the treatments should be individualized according to the needs of each patient.

Patients that do not comply will need more professional care in contrast to patients willing to adopt homecare favoring oral health. With careful selection and motivation of patients for improved oral hygiene, a large number of early lesions will not need further restorative work. Such lesions should be recorded for observation in the annual or biannual examinations. The clinical signs for recommending restorations of early lesions are : (a) improper compliance of the patient to oral hygiene recommendations, (b) advancement of the lesion from one examination to the next, and (c) pain that may be related to the lesion. The main aspects of the desired patient behaviour are the following : (a) better plaque removal, (b) a healthier diet limiting the amount and frequency of fermentable sugars, (c) the use of fluoride for home care, and (d) regular visits to the dental office for monitoring the oral health status of the patient and topical applications of concentrated fluoride solutions.

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