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AN EFFECTIVE METHOD OF PERSONAL ORAL HYGIENE*

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NEW ORLEANS

INTRODUCTION

The purpose of personal oral hygiene (usually called home care of the teeth) is (1) maintenance of oral cleanliness, (2) prevention of caries, (3) prevention of periodontoclasia. It is desirable not only to prevent initiation of the lesions of these diseases, but also to prevent, to the maximum extent possible, further advancement of lesions that already exist. An effective method must fulfill all three of these important purposes.

A clean tooth does not decay. Periodontoclasia does not occur about a clean tooth. Therefore, a method which satisfies well the first purpose named, at the same time accomplishes the other two.

No amount or quality of treatment and restorations by dentists can maintain dental health unless they are followed by effective personal oral hygiene. The success and durability of the dentist's work depend largely upon the subsequent personal oral hygiene of each patient.

Of the many methods of personal oral hygiene now generally followed, none is entirely effective. This statement is confirmed by the fact that practically all adults, and many younger persons, have already sustained more or less damage from caries, and that periodontoclasia lesions are present around many or all of the teeth of practically all adults. This means that the methods of personal oral hygiene generally followed, whatever the source of instruction, are inadequate to prevent these diseases.

Several years ago I designed and described [1] the method of personal oral hygiene which is necessary for prevention of caries and periodontoclasia. It was based upon accurate information, secured mostly through laboratory research, upon the etiological conditions at the locations where the lesions of these diseases originate and advance. Measures for successful prevention must provide for effective prevention or minimizing of these local etiological conditions. Correct information (not merely opinions) as to what these conditions are is necessary. Measures based upon incorrect information, erroneous beliefs,

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or misinterpretation of scientific observations, however authoritative they may seem to be, are unlikely to be effective.

The causative organisms in these diseases are microscopic, the pathological processes originate and advance microscopically, the tissues involved are composed of microscopic elements and the lesions themselves, at first, are only microscopic in extent. Therefore, correct information about these conditions must be based upon microscopic research. Opinions and ideas not conforming to the basic facts, which can be confirmed only by microscopic work, are confusing, often entirely erroneous.

During the past several years there has been opportunity to demonstrate, to their satisfaction, and to teach to a considerable number of dentists, the fundamental facts as to the local etiological conditions in these two diseases. With these facts known and understood, and any previously existing confusion or conflicting ideas cleared up, it is evident to anyone that effective personal oral hygiene is necessary for prevention of these diseases; and to significantly retard or prevent further advancement of existing lesions. It is also evident that methods generally taught and practiced are inadequate and could not be entirely effective.

The necessary method, which I have described, [1] is quite different from, and in some particulars quite the opposite of, ineffective methods generally advocated and followed. Dentists who have the necessary information have their expectations confirmed and are profoundly impressed by the effect they themselves experience personally, and the beneficial effect upon their patients whom they successfully teach. Hundreds of people (including a good many dentists) can be named who now, by this method, are maintaining a degree of oral cleanliness and dental health far greater than is possible in any other way.

The purpose of this paper is again to direct attention to this method, I now having had considerable experience in presenting the fundamental facts upon which it is based, and in teaching the method to a considerable number of people (mostly university personnel). But before describing the method it seems desirable to recall briefly or present important information about the local etiological conditions which must be minimized or prevented and to recall or present some of the fundamental facts regarding the disease processes involved.

Clear understanding of the cause of a disease is necessary for the adaptation of means for its prevention. If the disease be local, such as caries and periodontoclasia, clear understanding of the essential local conditions which cause the lesions and promote their advancement, is necessary for adaptation of means of preventing these local conditions.

BASIS FOR THE METHOD

Locations where enamel caries lesions originate.—Caries lesions of the enamel first appear in the occlusal pits and fis-

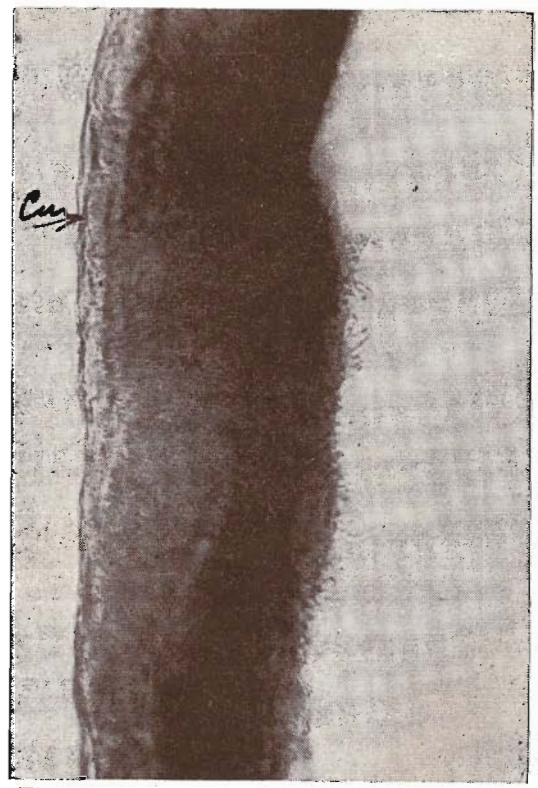

Fig. 1: Section of bacterial film from surface of enamel. Enamel cuticle, cu, at left. Note fruiting heads projecting at surface of pad at right.

sures and on the proximal surfaces between the teeth, at locations where there is more or less protection from natural cleaning. They do not originate on surfaces from which foreign material is frequently removed by functional friction or otherwise.

Nature of the material in protected areas.—In uncleaned areas on the teeth there is continuously present a heavy film (Fig. 1) of bacterial material (usually called plaque) consisting of long rod and filamentous types of microorganisms, characterized by having one end attached to the tooth (Fig. 2) and the other extending outward to the surface of the film where there are the growing ends and

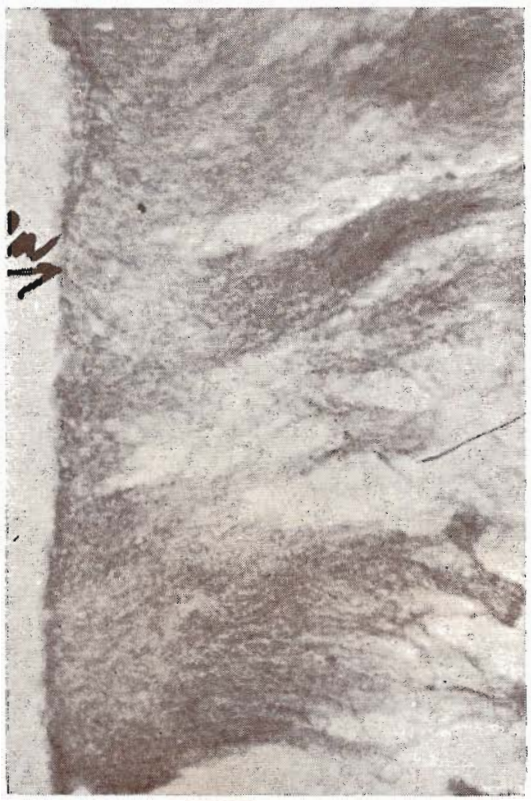

Fig. 2: Section of bacterial film from surface of enamel slightly teased apart to show filamentous type organisms. Enamel cuticle, cu, at left.

usually fruiting heads of the fungi making up the pad. The thickness of this bacterial film varies greatly at different locations, determined largely by the depth of the space and by the exposure to friction by which the ends of the filaments at the surface are frequently rubbed off. At the surface there are not only the growing ends and fruiting heads of the filaments making up the pad but also large

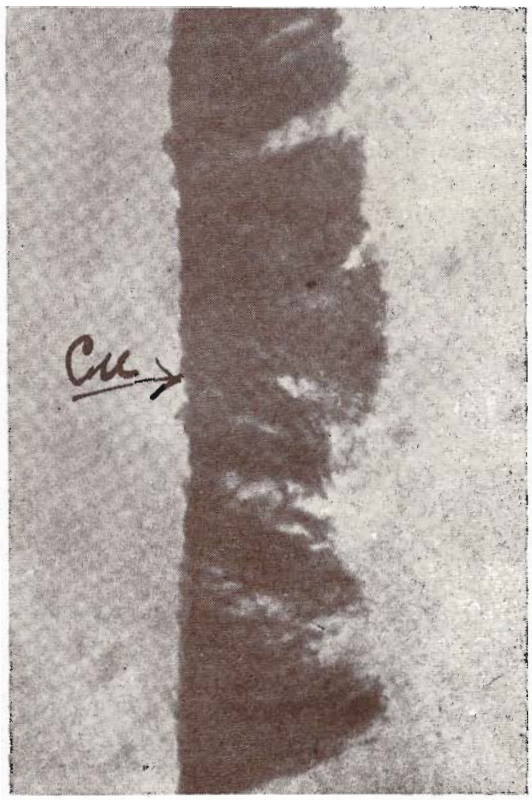

Fig. 3: Bacterial film from over active chalky enamel. Outer part of film brushed off leaving stumps and stems of filamentous type organisms attached to cuticle, cu.

numbers of the very many varieties of other bacteria in the mouth. These are accumulated upon the surface and interspersed, more or less, between the outer ends of the filaments.

Deeper in, however, the pad at any particular place is made up of the filamentous types only, each filament extending outward, at right angles, from the surface of the tooth to which it is attached. Breaking off the ends of the filaments at the surface of the pad still leaves a film consisting of stumps and stems (Fig. 3) attached to the tooth. Most of these are living and capable of growing in length again under favorable circumstances, thus restoring, in time, the former thickness of

the film pack at any given location.

The presence on teeth of filamentous types of microorganisms, usually spoken of as Leptothrix or thread forms, has been long recognized, some authors associating them with caries, others not. More than eighty-five years ago Leber and Rottenstein [2] described filamentous forms of Leptothrix as the "microorganisms of caries." Their Figs. 1, 2, 3 in Plate II, illustrate the organisms and a pad of filamentous forms attached to, and radiating from, a decaying tooth surface.

Wedl [3] in 1870, emphasized the proliferation of Leptothrix in relation to caries and illustrated this in several of the engravings in his *Atlas to the Pathology of the Teeth,* showing uniformly disposed, firmly adherent Leptothrix masses on exposed surfaces of teeth, especialy those at which caries lesions originate.

W. D. Miller [4] in 1883, observed filamentous forms of Leptothrix on the teeth and in caries lesions. He describes, on the border of decaying dentin, indistinguishable masses of fungi from which project numerous threads of *Leptothrix buccalis* (See his Fig. 2). Later, in 1890, in his book [5] on *The Microorganisms of the Human Mouth,* his Figs. 63, 67, 79, indicate filamentous organisms composing the bacterial film on teeth. He was not able to identify these filamentous forms, or any other forms of bacteria found in the mouth, as the specific cause of caries. He evolved his well known chemicoparasitic theory to the effect that acids are produced by the breakdown of carbohydrates by acidogenic bacteria at the vulnerable locations; which acids decalcify tooth material and thereby cause and promote caries. All firm information to this day confirms and supports the correctness of this theory formulated more than sixty years ago.

In a recent survey of the literature relative to the cause of caries Boedecker [6] came to the general conclusion that "intact enamel surfaces in vitro exhibit widely variable resistance to attack by acids produced by acidogenic bacteria. There is no evidence that acidogenic bacteria and carbohydrates can initiate dental caries". (I do not accept this conclusion. Acidogenic bacteria and carbohydrates are essential for the initiation of caries lesions). "The progress of dental caries, once initiated, is effected by bacteria acting during the acidogenic phase on a carbohydrate substrate." "There is probably no specific microorganism associated with dental caries . . ."

The conclusions reached by Bartels [7] from a survey of the recent literature on the subject do not conflict with or contradict Miller's [5] original theory, but rather support it.

The fact that bacterial action on cariogenic food material within the mouth is necessary for production of caries is further substantiated by the work of Kite, Shaw, and Sognnaes [8] who showed that susceptible rats fed a cariogenic diet by stomach tube do not develop caries; and by Orland [9] to the effect that rats maintained entirely free from bacteria, reared upon a cariogenic diet, do not develop caries, whereas controls on the same diet have a high percentage of caries.

The work of J. Leon Williams, toward the close of the nineteenth century, contributed more than all others had previously, to the cause of caries and the microscopic conditions at the locations where the lesions originate. His paper [10] in 1897, not only shed much light upon the microscopic caries process, but it showed clearly the nature of the bacterial film upon the surface of teeth beneath which the caries lesion originates and advances. In numerous photomicrographs (See his Figs. 61, 62, 63, 64, 66, 67, 69, 71, 75) he showed beyond question that the film pack is composed of long filaments radiating outward from the surface of the tooth to which they are attached. His work substantiated the claims of Miller [5] and of G. V. Black [11], that caries is caused by acids produced by conditions at the location of the lesions, and showed the morphologic characteristics of the principal microorganisms composing the film pack.

Later, Williams [12] directed attention to the numerous microorganisms found in material scraped from the surface of the teeth, some of them long rods and filaments, together with many other forms—large and small rods, and coccal forms. He directed attention especially to an organism which had been described previously by Vicentini [13] and named by him *Leptothrix racemosa.* This organism consists of a long relatively coarse filament or stem, attached to the tooth, on the outer portion of which numerous spores are produced. This spore-bearing conidiophore, which they called a fruiting head, is found projecting at, and just beyond, the surface of the film pack.

The striking, characteristic fruiting head serves as a convenient means of identifying the organism. Although it contributes largely to the bacterial film on many teeth, there is no evidence that it bears any specific relationship to caries. However, it is usually one of the several thread-form and filamentous type organisms making up the film on teeth, beneath which caries lesions originate and advance. In many specimens the deeper part of the film is made up largely of the stems of this fungus.

Vicentini [13] claimed that it is present in all mouths. Williams, in his first paper describing it [12], states that it "is common to all mouths which I have examined." I have found it present in material from most of the hundreds of extracted human tooth specimens, from many different sources, which I have examined.

The morphologic characteristics of this organism and the way in which it often makes up a large part of the film pack are shown by Williams [12] by numerous photomicrographs. (See his Figs. 14, 17, 18, 20, 31, 32, 33, 34, 37). I have also shown [1] (See my Figs. 6, 7, 8, 9) how the fruiting heads of this organism tend to project above the surface of the film pack and to accumulate in masses at the surface.

Throughout the literature since the historic paper of Williams [10] in 1897, there may be found numerous pictures of preparations which confirm the fact that the bacterial film on teeth is composed largely of long filamentous forms, each one extending outward from the surface to which they are attached. Since an effective method of personal oral hygiene must be based upon correct information as to the characteristics of this bacterial film, it may be helpful to refer to a few of such illustrations: [10] (Figs. 6, 8, 9, 11, 24), [11] (Figs. 2, 3), [12] (Figs. 82, 84, 109), [13] (Figs. 3, 4), [14] (Figs. 86, 87), [1] (Figs. 4, 5, 6, 7, 8,), [15] (Fig. 3), [16] (Fig. 3), [17] (Fig. 3, 4), [18] (Figs. 86, 87), [19] (Fig. 2), [20] (Fig. 3).

Conditions within cavities.—If the conditions on the tooth which initiate and promote the first stage, chalky enamel, caries lesions continue long enough, sooner or later a cavity develops and advances, usually at an accelerated rate. Active cavities in enamel finally reach, and advance into the dentin; thus progressively increasing in depth and in width.

Cavities, and to a less extent normal pits and fissures, present favorable receptacles for lodgment and retention of food material. The larger and deeper the cavity, the more food material it will hold. Many cavities are never free of remnants of food, and this material is continuously in various stages of decomposition. Before lodgment in the cavity food is heavily inoculated (or contaminated) with the many different kinds of bacteria [5, 12, 20-23] of the oral flora. In the cavity it is exposed to the abundant transient bacterial flora there, arising from decomposition that is going on of previously introduced food material, and also to the more or less permanent or continuous bacterial film lining the base and walls, (Figs. 4, 5) of the cavity.

Such a conglomerate bacterial environment always contains many species which produce acids when growing in the presence of fermentable carbohydrates, and others which do not. Food material (particles) retained within a cavity may, and usually does, contain variable amounts of

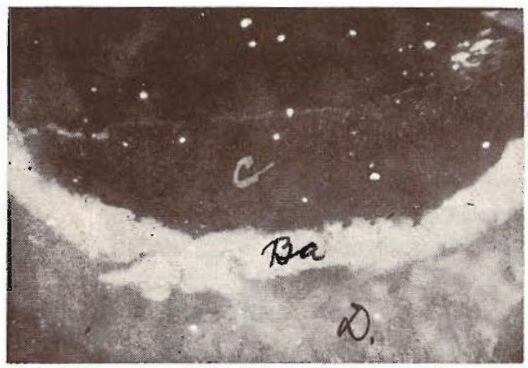

Fig. 4: Section including bottom wall of cavity in dentin, photographed with incident light. Ba-bacterial film, D-dentin, C-remnant of celloidin in which specimen was embedded.

Fig. 5: Section of sidewall of cavity in dentin showing filamentous bacterial pad at extreme right, disintegrating dentin to which film is attached, and spindle shaped expansion of tubules packed with bacteria.

such carbohydrates. In addition to that present in the particular material when it was introduced into the cavity, the carbohydrates may be replenished, more or less, from carbohydrate containing fluids (drinks) taken into the mouth subsequently. The production of acids, upon which caries activity and enlargement of the cavity depend, is determined largely by the retention and break-down of fermentable carbohydrates within the cavity.

The nature of the more or less continuous bacterial film lining the walls of the cavity is of great importance, from the standpoint of oral hygiene for prevention of further advancement of the lesion. This film is made up of countless millions of filamentous type organisms (Fig. 6) similar to those making up the film on the surface of teeth beneath which caries lesions originate. One end of the filament (Fig. 7) is attached to the decaying walls of the cavity, from which it extends outward to the surface of the film, where there are the growing ends and fruiting heads.

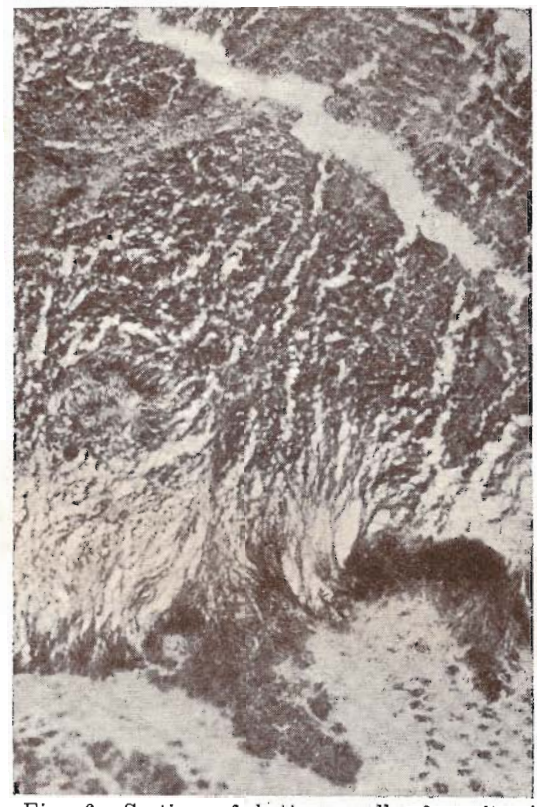

Fig. 6: Section of bottom wall of cavity in dentin. Deeper part of bacterial film consists of filamentous type organisms attached to decaying dentin.

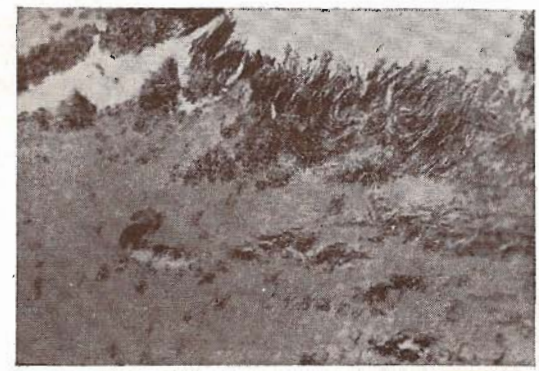

Fig. 7: Section of bottom wall of cavity in dentin. The outer part of the bacterial film has been torn away leaving stumps and stems of the filamentous type organisms attached to the disintegrating dentin.

The thickness of this bacterial film within the cavity varies greatly, depending largely upon the dimensions of the cavity and how frequently and completely its contents are disturbed or removed. Many of the films within cavities in extracted tooth specimens which I have measured range from about 100 to 400 microns (Fig. 8); some considerably thick-

Fig. 8: Heavy bacterial film, Ba, lining bottom wall of cavity in dentin, D. Thickest part of film pad measures 430 microns.

er. Most of the filaments of the different kinds of organisms composing such films are less than 0.5 micron in thickness. Therefore, the attached film within the cavity is made up of millions of separate elements, each of which is many times—usualy several hundred times—as long as it is thick.

Every specimen of decaying teeth shows precisely the same thing. Covering the surface or lining the cavity where decay had commenced there is always to be found a thick, felt-like mass of filamentous types of organisms, each radiating outward from the surface to which it is attached.

Pertinent facts relative to the caries process in enamel.—If a tooth specimen is suspended in a strong solution (5 to 10 per cent) of HCl, the enamel is rapidly dissolved. The enamel cuticle is set free within a minute or two, by the dissolving of the enamel immediately beneath it, and this loosened keratin-like membrane now may be removed or brushed off. If the acid is greatly weakened by sufficient dilution, or by less dilution plus the addition of tribasic calcium phosphate, then decalcification proceeds very slowly; and in the latter case only the carbonates are removed at first[24, 25]. Only partial decalcification occurs. The enamel becomes porous and softened like chalk and it may be dug into easily with suitable sharp instruments. If the quantity of diluted, weakened or buffered acid is frequently renewed, this partial decalcification may extend deep into, or in time all the way through, the enamel. In this way I have partially decalcified the entire enamel cap, which could then be shelled off in large pieces from the underlying dentin. If, on the other hand, all the enamel is covered with wax, except for a limited area, then only the uncovered area which is exposed to the acid is partially decalcified. This corresponds to the natural early stage caries process.

If we use organic acids such as lactic (which is supposed to be the principal one involved in the natural caries process) the same phenomena occur except that, in general, the action is much slower. When such weak acid solutions are used the enamel cuticle remains intact and in place. Thus weak acids pass through the cuticle and the products of their action on enamel pass back out through this very delicate osmotic membrane without destroying or impairing it.

In this way the early stage* lesion in natural enamel caries is produced. Acids produced at the surface of the bacterial film, and perhaps also deeper within it, pass along through this sponge or wick-like material, which we have seen is composed of countless millions of long filamentous type organisms, down to the cuticle and thence through it into the enamel.

This is a long slow microscopic and microchemical process resulting from the often repeated slow action of delicate amounts of weak acids produced on or

* (For convenience the term early stage may be applied to caries lesions before there is any break of the surface or cavity, and advanced stage where a cavity, of whatever size, has developed.)

within the bacterial film at the particular location. Time is required for the passage of such weak acid through the bacterial film, (Fig. 9) at the same time interchanging with, and being diluted by fluids of different reaction and composition before it reaches the surface of the tooth which is covered by enamel cuticle. Time is also required for osmotic passage of acid through the cuticle against substances of different reaction and composition resulting from the action of previously introduced acids upon the enamel.

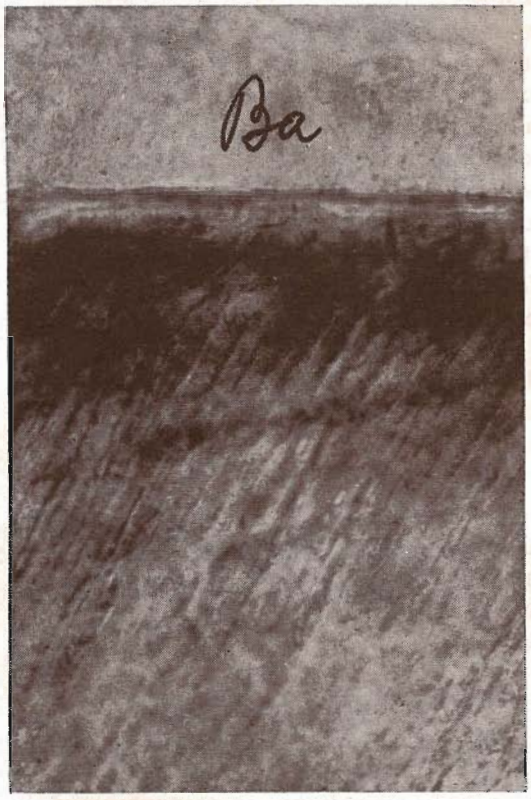

Fig. 9. Ground section (unstained), very early stage caries. Note variation in depth to which process has advanced in different enamel rods. Also note pad of bacterial film, Ba, at surface.

Still more time must be required for passage of the acid into the enamel, especially as the partial decalcification extends deeper and deeper. Not only must the acid pass through the already partially decalcified enamel to the depth of the process but there is a great tendency for this process to follow the course of individual enamel prisms. In suitable preparations under high enough magnification, we usually find the acid action extending along one prism farther than another (Fig. 10) in the immediate vicinity. This means that acid arriving at already partially decalcified enamel passes along the course of the individual prisms until it reaches the farthest point to which previous acid action has extended. Along this course of the prism the minute acid must interchange with products of previous acid action and finally reach the point of activity in effective quantity and strength. Again this is a very slow process. Thus the chalky enamel caries lesion represents the cumulative effect, over considerable periods of time, of minute amounts of weak acids produced, at various times, by bacterial action upon carbohydrate food material at the particular location.

Fig. 10: Ground section through farther advanced chalky enamel. Note variation in depth to which process has advanced in different rods and in intensity at different locations.

Pertinent facts relative to the caries process in dentin.—If the partial decalci-

fication of enamel (early stage caries lesion) continues to progress, it finally reaches the dentin. Minute quantities of acids produced at the surface now slowly pass through the chalky enamel, especially along the cracks (lamella) and channels that may exist there, thus reaching the dentino-enamel junction and on into the dentin. The caries process in dentin differs substantially from the process in enamel. This results largely from the different composition and structure of dentin.

Dentin consists of about 30 per cent organic matter and water, and 70 per cent inorganic material.[26] Acids remove the inorganic material leaving the cartilage-like dentinal matrix material which is now softer and can easily be cut or torn apart with suitable instruments.

During the dentinal caries activity decalcification advances deeper and deeper into the dentin, and the decalcified dentin at the surface (bottom and walls) of the cavity slowly disintegrates, thus increasing the depth and size of the cavity. Some of the bacteria of different kinds within the cavity grow and advance into the dentin by way of the open-ended dentinal tubules, towards the pulp chamber.

Near the surface of the cavity wall all the tubules are packed with bacteria. (Fig. 11). The pressure of the growing and enlarging mass of multiplying bacteria within the tubules in the decalcified dentin expands the tubules, compresses the decalcified softened matrix material and tends to split and break it up. Dislodged particles of the decalcified dentin are continuously being shed off from the disintegrating surface, thus increasing the depth and size of the cavity. (Fig. 5). Much deeper in only part of the tubules contain bacteria, many others at the same level do not. (Fig. 12). This means that the forward advance of bacteria into the dentin proceeds down separate tubules in-

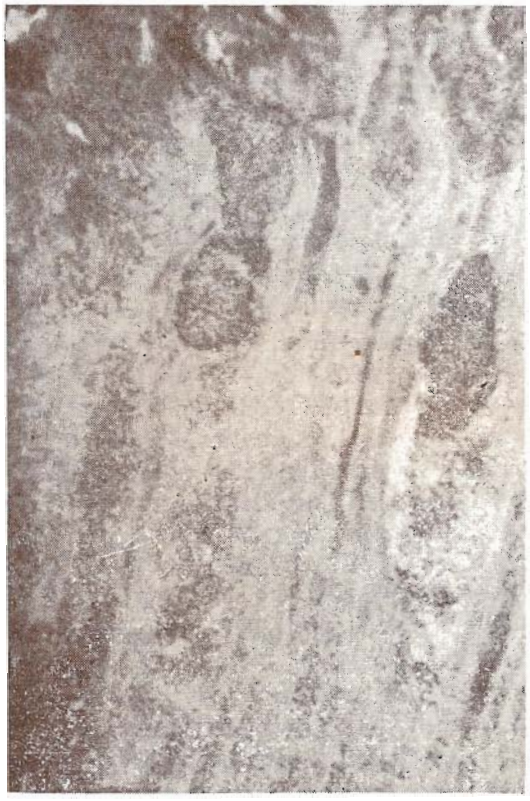

Fig. 11: Section through bottom wall (at top) of cavity in dentin. Note bacterial masses in softened dentin and some extending into dentinal tubules from surface.

Fig. 12: Bacteria advancing pulpward down separate tubules. Others at this same level not yet invaded.

dependently of other nearby tubules.

The dentinal tubules extend through the dentin from the pulp chamber to the dentino-enamel junction. Their size near the pulp is several times that near the outer surface.[26] They contain long protoplasmic projections (Tomes fibers)[27] from the odontoblasts. In normal vital dentin a nutritional fluid, dental lymph,[28-30] circulates in the dentin, principally through the dentinal tubules and their many anastomosing branches.

Irritation from severe wear, erosion and caries stimulates the formation of secondary dentin (irregular dentin, Orban[26]) at corresponding areas on the pulpal wall. This new-formed secondary dentin effectively closes off the tubules of the involved regular dentin. This is now spoken of as a dead tract. Dental lymph cannot pass through the now closed tubules of the area and bacteria cannot advance toward the pulp, as they can in open tubules.

No doubt laying down of this wall of secondary dentin which closes the tubules and shuts off the passage of dental lymph into them, is nature's most important protection against advancement of bacteria into the tubules, and ultimately infection of the pulp. Bodecker[29] has called these changes "protective metamorphosis."

Cervical Caries.—The cervical caries lesion begins at the cemento-enamel junction, and only after the gum has receded enough to expose the area. For unknown reason some people seem to be especially susceptible to this type of lesion. This is probably related to varying relationships in the area where enamel and cementum join.[26-31] In some instances their edges do not quite meet, thus leaving an area of dentin covered only by soft tissue. After this is removed there is left an area of exposed dentin with open-ended tubules, which does not have the usual protective covering of either cementum or enamel. They are now open to invasion by bacteria.

In other instances the cemento-enamel junction presents a V-shaped groove of considerable size which, after the soft tissue is removed, offers a favorable situation for accumulation of filamentous type bacteria, as occurs elsewhere in grooves and depressions.

Whatever the special conditions may be that initiate the cervical caries lesion, it advances into the dentin in much the same way as do lesions originating in the enamel after they reach dentin.

Inactive Caries (Arrested Caries).—Caries lesions usually begin at the vulnerable locations during the first two or three years following eruption of the particular tooth. If the local etiological conditions which initiated the lesion continue, the disease process continues to advance. In time a cavity (advanced stage lesion) develops large enough to be recognized by the patient or by the dentist. The preceding early stage chalky enamel lesion has existed and progressed unrecognized for months previously.

If at any time, the local conditions which cause the lesion and promote its advance are prevented or altered sufficiently, the progress of the disease process slows down or stops entirely. This may occur at any stage of the lesion from the earliest (microscopic) chalky enamel stage to the advanced cavity stage. Obviously the earlier this occurs the less damage will have been done.

A large part of all proximal caries lesions stop advancing—become inactive—before cavities are formed. Often small cavities in this region also cease to advance. This slowing down or cessation of proximal caries activity coincides with the incidence and increasing activity of periodontoclasia in the same region. Bibby[32] has recently reviewed the literature relative to the apparent antagonism between caries and periodontol disease. The available information cited tends to support some such antagonism.

Gingivitis is characterized by a continuous flow of minute quantities of inflammatory exudate which consists of serum containing pus cells and some blood cells. Constant presence of such exudate in the

environment of the caries lesion between the teeth tends to neutralize and counteract the effect of minute quantities of weak acids in the area. It is possible also that it may influence the local bacterial flora and promote the growth of proteolytic bacteria in the environment, thereby lessening acid production by acidogenic bacteria. On the other hand, periodontoclasia has little or no influence upon occlusal caries. Inflammatory exudate does not reach the area in effective amounts and concentration.

Early stage caries—chalky enamel—always appears pure white throughout the life of the tooth, as long as the cuticle remains intact. Usual stains of the mouth do not pass through normal cuticle. On the other hand, chalky enamel from which the surface has been destroyed becomes stained—usually varying shades of brown. Stains are also carried into chalky enamel through cracks (lamella) and other channels that may exist there. The staining of such lesions tends to increase, although they may remain inactive, throughout the life of the tooth. Usually chalky enamel lesions which had advanced sufficiently to be stained because of loss of cuticle or penetration of lamella or small cavities remain white around the borders in areas in which the cuticle is still intact.

Occlusal caries lesions start at the entrance and sidewalls of occlusal pits and fissures of molars and bicuspids where the depressions favor accumulation of bacterial film, retention of food material, and production of acids. The partial decalcification—chalky enamel stage—progresses and finally cavities develop. There is a tendency for the decay process to extend down any channels and after it reaches the dentin, to spread and undermine the enamel along the dentino-enamel junction.

Extensively undermined enamel may crack off leaving the decaying dentin widely exposed. Bacterial film and food material in such wide open occlusal cavities may be disturbed or rubbed off several times every day. Occasionally dentinal decay in wide open cavities ceases to advance and the dentin becomes very hard, similarly to the way in which dentin exposed by severe wear becomes almost as hard as enamel. This is an example of what often happens when such a wide open cavity is frequently cleaned out by whatever means.

Sometimes radical changes in diet habits or in oral hygiene habits and methods at any time, may slow down or stop the activity of early stage occlusal caries lesions. However, in most instances, no such circumstance arises, especially during the age period (up to 12 or 15 years of age) of greatest activity of such lesions.

Periodontoclasia-Definition. — Various terms have been used to indicate the disease process encompassed by periodontoclasia. Such terms as periodontal disease, periodontosis, periodontitis, pyorrhea, pyorrhea alveolaris, alveolodental pyorrhoea, Riggs disease, gingivitis have been applied to the disease in general or to some particular phase or prominent clinical condition. The disease is characterized by inflammation and suppuration of the epithelial tissue within the gingival crevice, and by long continued progressive chronic inflammation in the parodontal tissues resulting in destruction and resorption of periodontal fibers and alveolar bone.

Periodontoclasia is caused by foreign material, consisting of bacterial film and/or concretions (mostly calculus) upon the surface of the tooth at the entrance to, and within, the gingival crevice. Prevention of the disease and prevention of further progress of existing lesions can be accomplished only by preventing or minimizing these local etiological conditions—conditions without which lesions do not originate or advance.

Early stage of the disease.—All periodontoclasia lesions begin at the gingival margin and are, at first, only microscopic in extent, therefore not recognized.

It has been pointed out [1, 10, 12, 14] and emphasized above, that bacterial film (plaque) on protected areas on the surface of the tooth consists of a pad of filamentous types of micro-organism, one end attached

to the tooth and the other extending outward towards the surface of the pad. At the surface of the pad there may be found more or less of any and all of the many other kinds of bacteria in the mouth.

At the entrance to the gingival crevice this bacterial material tends to grow or advance into the crevice and to impinge upon the epithelial surface there. The deeper part of the bacterial material continuously present upon the tooth tends to harden, calcify, and form rough concretions there. (Fig. 13). Normally, the marginal gingival tissue rests upon the soft smooth non-irritating enamel cuticle. Hard, rough material superimposed upon the cuticle acts as a mechanical irritant like a foreign body.

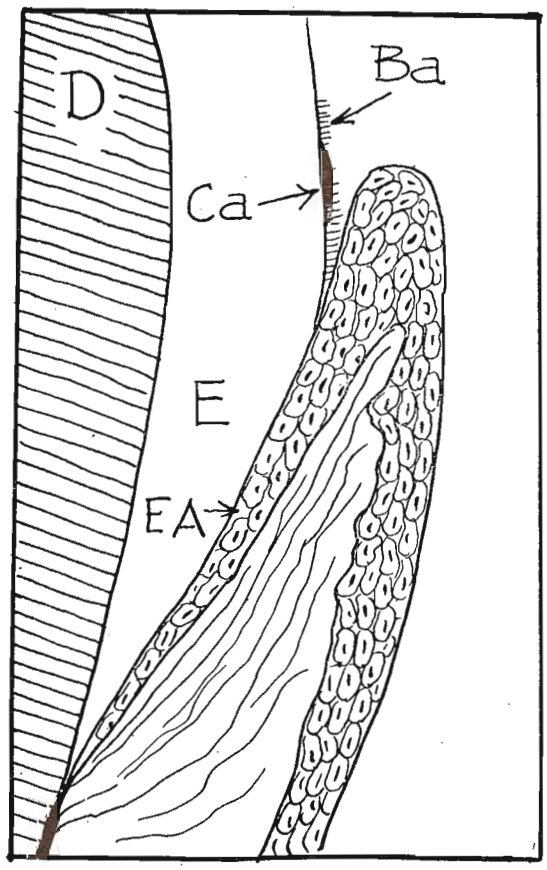

Fig. 13: Drawing to indicate foreign material at entrance to gingival crevice in early stage periodontoclasia. Ca-calculus, Ba-bacterial film, E-enamel, D-dentin, EA-epithelial attachment.

Soon the gingival margin is irritated by this foreign (and perhaps toxic) material against which it rests. Inflammation of the epithelial tissue ensues, characterized by migration of polymorphonuclear cells through the affected tissue (Fig. 14) to the point of irritation, where they tend to accumulate as pus cells.

Fig. 14: High magnification of section of marginal gingiva showing polymorphonuclears passing through the epithelial tissue at right which was in contact with irritating material on tooth. Good oral epithelium at left.

At first the amount of inflammatory exudate, which consists of pus cells and serous fluid, is not sufficient to be recognized by the unaided eye. However, material properly collected from the location always contains pus cells, upon microscopic examination; and, sections always show polymorphonuclear cells passing through the epithelial tissue opposite the irritating foreign material on the tooth against which it rested.

As the inflammation continues the bacterial film on the tooth tends to extend deeper and deeper into the gingival crevice, (Figs. 15, 16) the amount of the associated calculus on the tooth within the crevice increases, and there is increase in the amount of pus produced. The inflamed slightly congested and edematous condition of the marginal gingiva may now be recognized upon careful examination. It bleeds easily upon slight pressure or manipulation. The condition may now be noticed by the individual and by the dentist. Usually the importance and significance of gingivitis, as the early stage of the periodontoclasia lesion is not recognized. However it is, in fact, the begin-

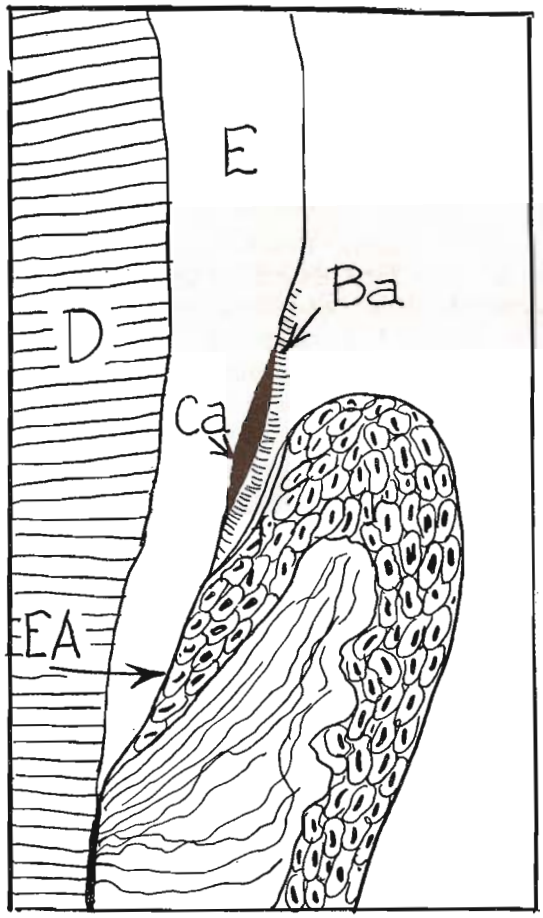

Fig. 15: Drawing to indicate foreign material within the gingival crevice in an advancing periodontoclasia lesion. Ca-calculus, Ba-bacterial film, E-enamel, D-dentin, EA-epithelial attachment.

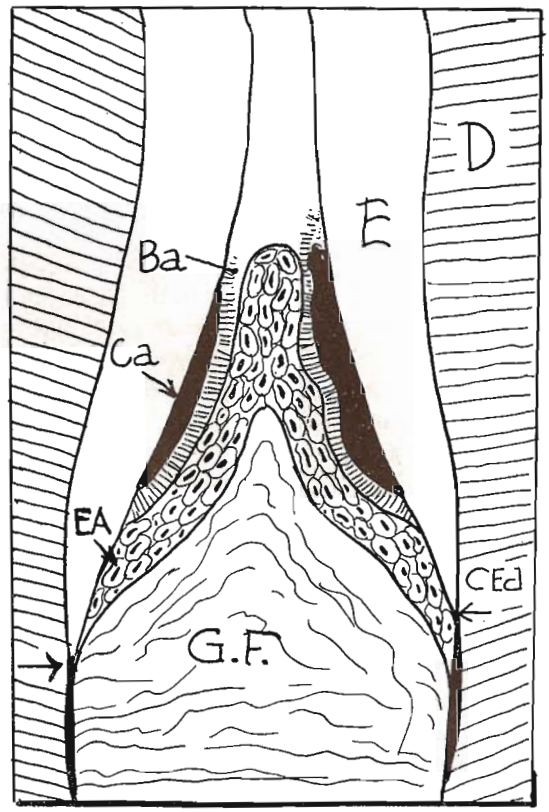

Fig. 16: Drawing to indicate foreign material within the gingival crevices in advancing interdental periodontoclasia lesions. Ca-calculus, Ba-bacterial film, E-enamel, D-dentin, CEJ-cemento-enamel junction, GF-gingival fibres.

ning of a progressive pathological process which never ends spontaneously, until the tooth is finally lost. The origination, activity, and rate of advancement of early stage lesions, and also, of the more advanced stage lesions, are determined largely by the effectivenes of the personal oral hygiene habits of the individual. These vary greatly with different individuals, at different times, and as applied to different tooth areas.

Nature of the subgingival bacterial film and calculus.—Once the foreign material (bacterial film and calculus) has advanced into the crevice and inflammation is set up there, more or less inflammatory exudate is continuously present in the crevice thereafter. This exudate consists of diluted serum (lymph) containing pus and blood cells. It provides a favorable environment for the establishment and growth of types of micro-organisms which are favored by partial anaerobiosis and by the serum-rich substrate. Such filamentous type organisms as actinomycetes and certain leptotrichia are encouraged.

Subgingival calculus consists of calcified bacterial material.[33, 34]. The inorganic portion is derived from the inflammatory exudate (serum). The bacterial portion consists largely of filamentous types of microorganisms, especially actinomyces.[35]

A pad or film of filamentous type organisms is always present on the surface of the tooth, and on the surface of any calculus, within the periodontoclasia pocket. (Fig. 17). This constantly present

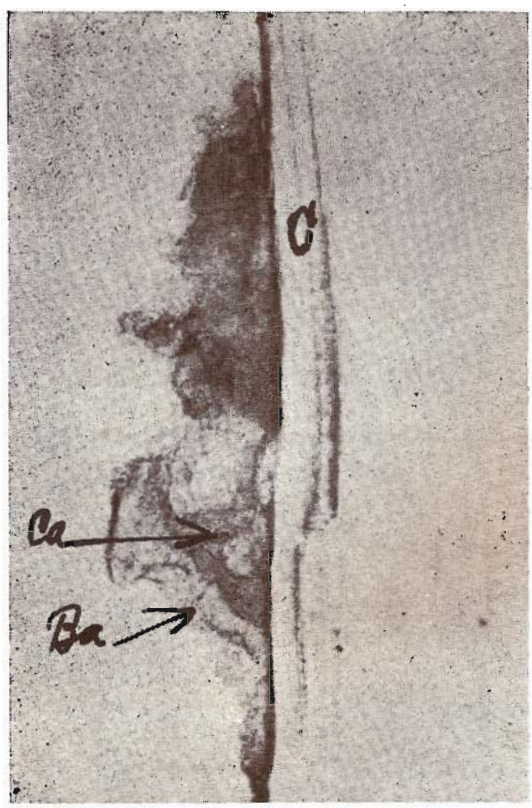

Fig. 17: Section through foreign material, which was located on the tooth within the periodontoclasia pocket. C-cementum, Ca-calculus covered by heavy lighter-colored bacterial film Ba.

bacterial film is composed of long filaments with one end attached to the tooth or to the calculus, (Fig. 18) and extending outward in palisade form to the surface of the pad which is in contact with the inflamed crevicular epithelial surface. At the surface of the pad there are the growing ends and fruiting heads of the particular organisms composing the film pack.

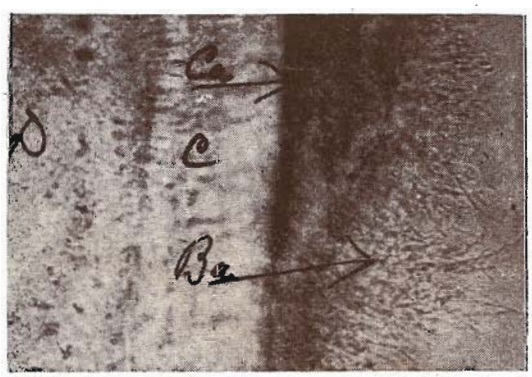

Fig. 18: Section through side of tooth which was located within the periodontoclasia lesion. C-cementum, D-dentin, Ca-calculus (darker) covered by filamentous type bacteria Ba, right. Note dark previously undescribed pathological [89] granules in dentin.

One of the important thread form organisms usually found in the pyorrhea pocket was first described by Beust [36] and named by him *Leptothrix falciformis*. He later [37] called attention to this organism in material from about the teeth, and pointed to the association of it with spirochetes.

I [38] have observed that the habitat of *Endameba buccalis* is the outer surface of the filamentous bacterial film on the tooth within the periodontoclasia lesion. "There they are protected and live, grow and multiply among the strands and fruiting heads of leptotrichia, principally *L. falciformis*." This observation conforms with the observations of others [39-42] as to the distribution of this parasite in the periodontoclasia lesion. Kofoid [39] confirmed especially the observation [43] that ameba are most numerous at the very bottom of the pyorrhea pocket. This ameba cannot be the specific cause of periodontoclasia, as sometimes has been erroneously supposed, for the reason that it is found in the lesions of not more than about 50 per cent of adults under 30 years of age. This does not necessarily mean that the parasite may not be harmful whenever it is present.

The fact has been shown [44] that the film pack is composed of filamentous types of micro-organisms, largely leptotrichia, attached to the surface of the tooth or to the calculus within the periodontoclasia lesion and that there is a tendency of the fruiting heads of *L. falciformis* to extend into the deepest part of the lesion. This conforms with observations of Box [45] in this regard.

It has been observed [44] also that the film pad of *L. falciformis* in the deeper part of the periodontoclasia lesion, and the conditions there, are especially favorable for the growth of spirochetes. These

are found in greatest abundance upon and among the fruiting heads of the leptotrichia at the very bottom of the pocket, and therefore overlying the zone of disintegrating epithelial attachment cuticle (zdeac).[46] This zdeac serves as a useful landmark which accurately indicates, on extracted tooth specimens, the location of the outer border of the epithelial attachment and the bottom of the periodontoclasia lesion[47] at any place around the tooth when it was in situ. The great abundance and constant presence of spirochetes at this location, which is where the lesion is advancing on the surface of the tooth—the location where the outer border of the epithelial atachment is receding apexward—suggests the possibility that spirochetes have some etiological significance in the advancement of the lesion.

While the flora and fauna tend to be limited to fewer varieties in the deeper part of the pocket, the entrance to the crevice and the part just within, are constantly exposed to infection and reinfection by any and all of the many species in the mouth. Material collected from this location always contains more or less of such mouth organisms. It is interesting to mention also that this location is where fusiform organisms are most abundant in mouths in which they and associated spirochetal organisms predominate.

Apexward advancement of the subgingival bacterial film and calculus.—The subgingival bacterial film and/or calculus tend to continuously advance apexward at various rates at different locations on a given tooth and on different teeth in the same mouth. As a result of the inflammation, suppuration, and destruction of the parodontal tissues which this foreign material on the tooth within the crevice causes, the location of the epithelial attachment moves apexward,[47, 61, 66] thus deepening the pocket and leaving more and more of the tooth without its normal soft tissue covering. Such bared tooth surface is always entirely covered with bacterial film right down to the outer border of the epithelial attachment.[44] Calculus formation follows along not far behind.[48] However, there is always a narrow band or space, of variable width, between the inner (or deeper) border of the calculus and the outer border of the epithelial attachment.

The epithelial atachment consists of a thin band or cuff of epithelial cells surrounding the tooth. The width of this cuff varies at different locations. It is widest when it is located on the enamel and becomes narrower as it moves apexward[46, 47]. Waerhaug[49] has presented strong evidence that what has been called the epithelial attachment is, in fact, not organically attached to the surface of the tooth and that a delicate instrument can be passed, with little resistance, into the gingival crevice all the way to the cementoenamel junction. Baume,[50] through phase contrast microscopy, finds evidence that enamel epithelial cells are very delicately

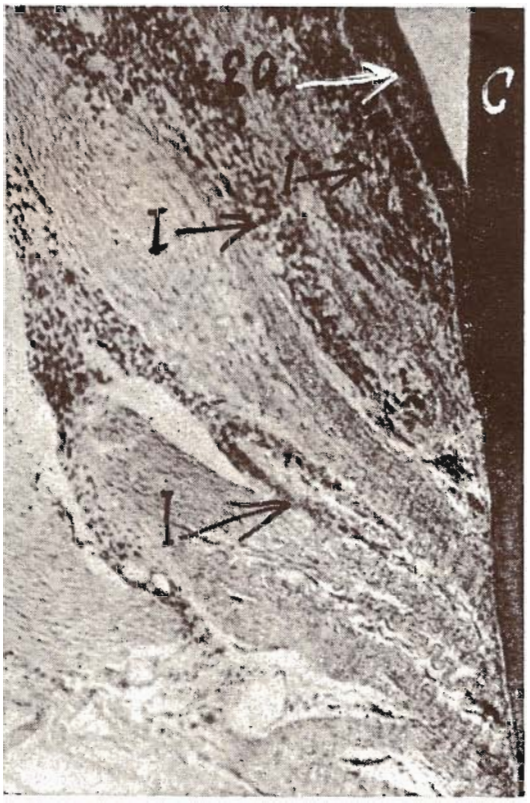

Fig. 19: Epithelial attachment EA, advances apexward over cementum, C, from which periodontal fibers have been destroyed. Note inflammatory cell infiltration I among fibers. (Attachment pulled away from cementum at top.)

attached to the tooth surface by tonofibrils, and that similar fibrils hold together the cells of enamel epithelium, and also those of the adjacent oral epithelium.

The inner border of the epithelial attachment grows apexward to cover over cementum from which periodontal fibers have been destroyed and removed (Fig. 19). As more fibers are destroyed, the epithelial attachment advances farther. Destruction and resorption of the periodontal fibers which make possible the advancement apexward of the epithelial attachment, and which gives to the disease its name—periodontoclasia—results from chronic inflammation extending into the periodontal tissue among and between the periodontal fibers for considerable distances from the bottom of the pocket. (Fig. 20). This inflammatory process is characterized by round cell and plasma cell infiltration, and by accumulation of these inflammatory cells between the bundles of periodontal fibers. (Figs. 21, 22, 23, 24). As the process continues, the involved fibers are broken down and finally resorbed.

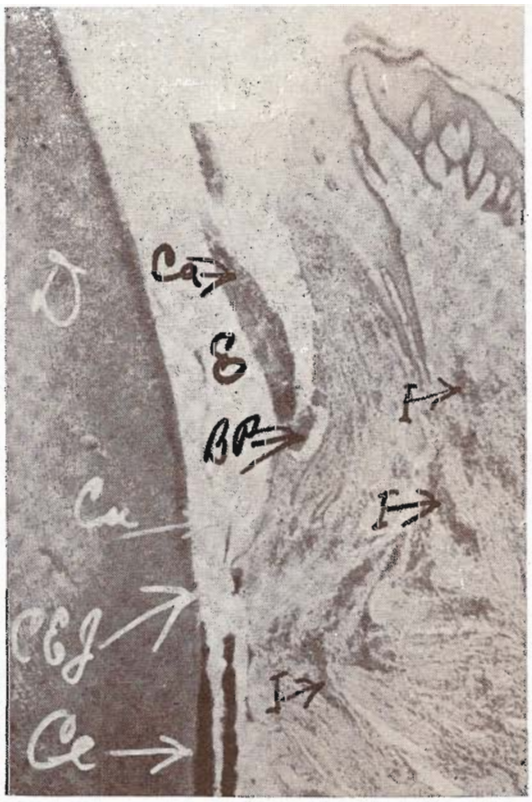

Fig. 20: Section of typical active periodontoclasia lesion (slightly pulled apart). Note especially, location of calculus, Ca, in relation to inflamed gingival tissue. Note also how far round cell infiltration, I, where periodontal fibers are being destroyed, extends from bottom of pocket, BP. E-enamel space, Cu-ribbon of enamel cuticle, D-dentin, Ce-cementum, CEJ-cemento-enamel junction.

The important fact that foreign material (calculus and bacterial film) is always present on the tooth within the inflamed gingival crevice (the periodontoclasia lesion) is well shown in numerous illustrations to be found throughout the literature relative to this disease, or in some instances presented for other purposes. Most pictures show what is usually labelled calculus on the tooth within the pocket. The fact that the calculus and the

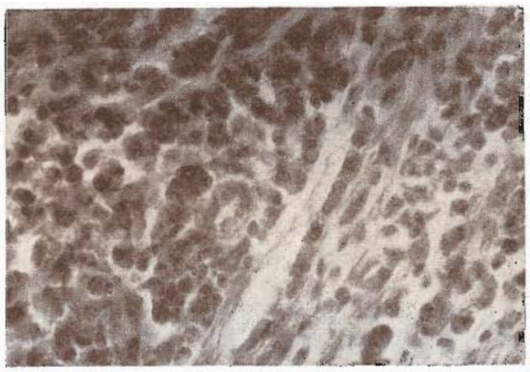

Fig. 21: Heavy round cell and plasma cell infiltration in periodontal tissue resulting in destruction and removal of periodontal fibers. Only a few fibers remain.

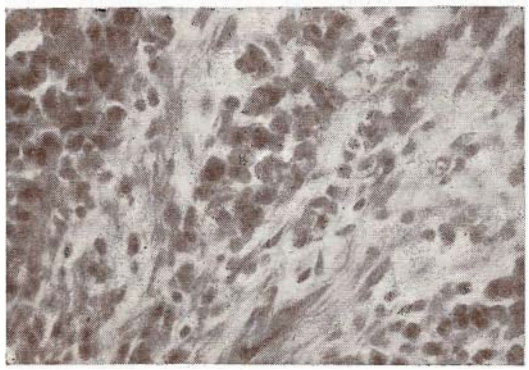

Fig. 22: Round cell and plasma cell infiltration among and between periodontal fibers. Destruction of fibers not quite as advanced as in Fig. 21. More fibers intact.

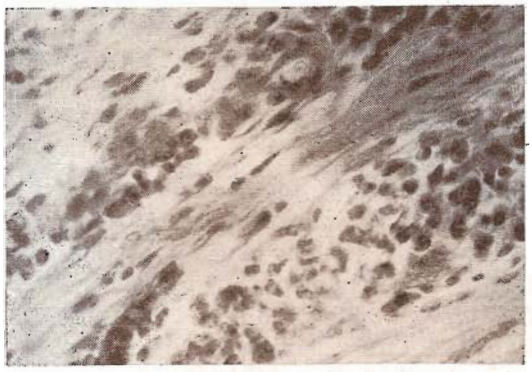

Fig. 23: Inflammatory cell infiltration less than in Fig. 22. Good periodontal fibers intact.

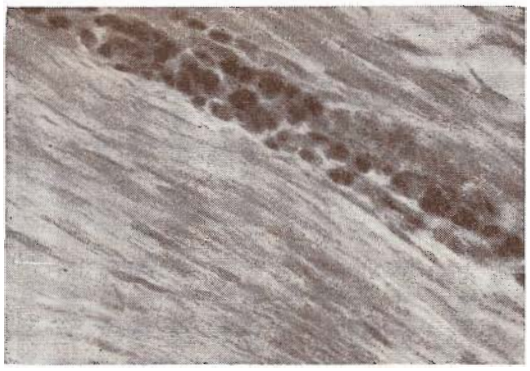

Fig. 24: Inflammatory cells accumulated in a streak between good intact fibers. Condition often found farthest from the bottom of pocket.

part of the tooth between the inner border of the calculus and the outer border of the epithelial atachment are covered by heavy bacterial film,[44,48] is usually overlooked. Following are listed a few of such publications and the particular illustrations which show this important feature of the disease, upon which effective personal oral hygiene must be based: [51](Figs. 1 2, 3, 4), [52] (Fig. 6), [53] (Fig. 8) [54] (Figs. 20, 23, 29), [55](Figs. 2, 7, 8), [56](Figs. 1, 5, 6), [57](Figs. 3, 12, 14, 20, 25, 26, 27, 29, 30), [49](Figs. 17, 18, 20, 39), [58](Fig. 10), [59] (Figs. 24, 26, 31), [60] (Figs. 16 (2), 13 (3), 24 (7)), [16](Figs. 278, 287, 288, 289, 293, 305), [62](Figs. 3, 4), [63](Figs. 3, 5), [64](Fig. 190), [26](Figs. 138, 218, 222), [65](Figs. 59, 64), [18](Fig. 104), [67](Figs. 491, 713, 733, 734, 735, 738, 767B), [68](Figs. 261, 264, 268), [69](Fig. 15), [70](Figs. 6, 8).

The lesion, at first microscopic and later macroscopic, extends around the tooth, but is usually deeper and more active in the interproximal crevices which are deeper and where accumulations of foreign material are less subject to removal by functional friction or customary methods of oral hygiene. The tooth surrounded by concretion and bacterial material adhering to it within the gingival crevice (lesion) is, in effect, a foreign body, constantly subject to infection and reinfection with the many different kinds of bacteria in the mouth.

This foreign body effect was recognized and its importance emphasized more than seventy-five years ago by John W. Riggs.[71] More than sixty-five years ago J. N. Farrar [52] urged the necessity of removing this subgingival foreign material and laid special emphasis upon keeping the area clean. He said "the disease vanishes when the pockets are carefully and thoroughly cleaned, and *kept clean.*" To this day, removal of this material from the tooth, which these pioneers advocated at that time, is still essential for successful treatment of the disease. Not only is it essential, but it is perfectly logical, that the local cause of the disease should be removed and its reformation prevented. Prevention of its reformation is accomplished by the method of personal oral hygiene herein presented.

DESCRIPTION OF THE METHOD

Above, attention has been directed to the essential local etiological conditions in caries and in periodontoclasia—conditions without which lesions of these diseases do not originate, and existing lesions, with rare exceptions, do not advance further. With this information known and clearly understood it is evident that these conditions must be prevented or minimized to prevent the occurrence of the lesions and the advancement of existing lesions.

Treatment of existing lesions is a matter for the judgment and experience of

the dentist to decide. Some caries lesions require fillings; others may require trimming and smoothing of overhanging edges, perhaps enlarging the cavity, so it can be cleaned out well by the patient; still many others require nothing but watching.

It is necessary that all concretions (calculus) on the tooth within the periodontoclasia lesion be scaled off and the surface of the tooth be made as smooth as is practical. Masses and rough surfaces of calculus are very much in the way of the patient's cleaning the tooth, especially within the crevice. It is very important that removal of the calculus include that in the deepest part of the pocket where the lesion is advancing. This calls for skill, patience and conscientious work.

There is no need or justification for the extensive mutilating gingivectomies sometimes done in this disease. Except in the case of some far advanced stage lesions and under special circumstances, thorough cleaning of the teeth by the dentist followed by the method of personal oral hygiene described below, results in rapid subsidence of inflammation and of suppuration of the tissues involved. No further advancement of the lesion occurs.

Locations on the teeth which must be cleaned.—The important locations on the teeth which the patient must clean are occlusal depressions (pits and fissures); areas which are more or less protected from functional friction, such as the embrasures and interproximal surfaces; and the surfaces at the entrance to, and especially those within, the gingival crevices.

The material to be removed (cleaned off or out) consists of more or less food material, in various stages of decomposition, which has been retained since the previous cleaning, and of bacterial film material which has grown there since the previous cleaning. All such material is soft and is easily dislodged by the right (proper) application of toothbrush bristles and of the filaments of dental floss. There is no other way by which they can be physically and effectively dislodged by the patient—no other way by which he can clean his teeth at these important locations.

Right kind of toothbrush and dental floss.—Effective cleaning of the teeth can be done only with a brush and floss, the bristles and filaments of which can be effectively applied to the areas to be cleaned. After having full information as to the functions to be performed and measurements of the spaces and places to be cleaned, and after much experimentation, I have ascertained what I believe are the optimum characteristics of brushes and of floss for the purpose. The exact details have been published,[19,-72] and the reasons for these specifications were given. In my opinion, based upon my own knowledge and experience, brushes or floss that deviate to any considerable extent from these exact specifications are inappropriate and less effective for the purpose, to whatever extent they so deviate.

Briefly, the specifications of the brush are:

1. Plain straight-handle design; over-all length about 6 inches; width about 7/16 inch; 3 rows of bristles, 6 tufts to the row, evenly spaced.

2. High quality nylon bristles, about 80 per tuft, .007 inch diameter, straight trim, finished to 13/32 inch length.

3. Ends of bristles ground and finished to hemispherical shape or at least enough to eliminate all sharp points and rough edges.

4. A similar brush of reduced size for the use of young children should have an over-all length of about 5 inch, .005 inch bristles, finished in the same way to 11/32 inch length.

Briefly, the specifications of the floss are:

1. Material—high tenacity bright nylon yarn (Type 300), 2 denier per filament.

2. Construction—made by twisting to-

gether 5 threads of 70 denier, 34 filament yarn.

3. Twist—3S twist, steamset.

4. Size—350 total denier.

5. Manufacturer's technical designation for this floss is: 70-34/5 S 3, Nylon 300, steamset.

When teeth must be cleaned.—Practically all caries activity occurs at night during rest and sleep. During the daytime movements and activity of the mouth, tongue, cheeks and lips result in frequent washing of the teeth and more or less changing of the saliva around the teeth including that at locations at which food material may be retained and decompose. Acids that may be produced are diluted and/or carried away, more or less, by such frequent changing of the surrounding saliva. Food material is also diluted or washed away, especially the easily dissolved parts of it.

Sugars and fermentable carbohydrates contained in particles of food material which is subject to frequent change of the surrounding fluid tend to leach out. Even food material retained within cavities gives up much of the sugar it may contain, from the frequent changing of the fluid with which it is constantly bathed.

All this activity and functions of the mouth by which fermentable carbohydrates and acid that may be produced are diluted or partially removed necessarily tends to reduce caries activity during the daytime.

On the other hand, during sleep the flow of saliva is greatly reduced. The movements of the mouth by which the saliva surrounding decomposing food material at the caries locations is changed and the products of fermentation are washed away or diluted, are also greatly reduced. The most favorable conditions exist at this time for caries activity.

It has been found by several workers [73-77] that caries incidence and activity are enormously increased in desalivated animals fed a cariogenic diet. Klapper and Volker [78] found that hamsters with only partial salivary gland impairment, when fed a suitable cariogenic diet, develop much more caries than controls. Apparently, the quantity of saliva is an important factor.

In xerostomia (dry mouth), in humans without exception,[79] there is rapid decay of the teeth. This is believed to result from the lessened washing and bathing of the teeth by saliva. In cases in which caries is much in evidence on one side of the mouth only, the patients invariably [80] have a preference for sleeping on the opposite side. This habit tends to dryness on the up-side which is where the greatest caries activity occurs.

It has been pointed out that the changing of fluid and washing of potential caries areas on the teeth which goes on, consciously or unconsciously, during the daytime is enormously decreased during rest and sleep. In addition, and what is even more important, flow of saliva is almost nil. Therefore, during sleep, the conditions are quite similar to those of desalivated animals or of humans with xerostomia. Since conditions which permit and promote caries activity exist mainly at night during sleep, it is evident that to prevent such action at that time, the teeth must be cleaned effectively before retiring.

So far as caries activity is concerned, if the teeth are thoroughly and properly cleaned at night before retiring, there follows a long period during which there is no acid production, because there is no fermentable carbohydrate food material present to produce it. This period coincides with the period during which caries activity otherwise occurs. The cleaning also dislodges or removes much of the bacterial film material at the vulnerable areas. Not much growth of the remnants —stumps and stems—of such microorganisms occurs during the night, for lack of suitable substrate for their growth which food material, if present, would provide.

The time the teeth must be cleaned for prevention of periodontoclasia activity is indicated by somewhat similar consider-

ations to those relative to caries activity. Usually food is taken into the mouth at several different times during the day. Remnants of food—solutions, microscopic particles, some larger—tend to lodge and be retained in protected areas at the entrance to the gingival crevices. Some may be forced into the crevice for short distances. In addition, larger tough particles may be retained between the teeth and press upon the interproximal gingiva, sometimes extending into the crevice. During the daytime the activities and functions of the mouth, and the abundant flow of saliva, result in washing away irritating material. If, however, it is retained over long periods of time during quiet and sleep, and minimum flow of saliva, it causes more or less continuous irritation of the tissues. Most favorable conditions exist for maximum growth of bacteria. If on the other hand the teeth are thoroughly cleaned at these locations, at night before retiring, there follows a long period during which there is no such irritation from decomposing food material, and there is little growth of bacterial film to advance into the crevice.

Thorough proper cleaning of the teeth by the necessary method described herein, not only removes retained food material and dislodges and removes bacterial film at and near the entrance to the gingival crevice, but the bacterial film on the tooth within the crevice all the way down to the very bottom of the lesion is mostly dislodged and removed. Little growth takes place during the night.

Removal of irritating material at the entrance to, and within, the gingival crevice permits rapid subsidence of inflammation and healing of the ulcerated crevicular gingival tissue of existing lesions. It is perfectly evident that for prevention of periodontoclasia and prevention of further advancement of existing lesions, the teeth must be cleaned at night before retiring.

Thorough cleaning of the teeth at night before retiring is essential for prevention of both caries and periodontoclasia. Notwithstanding the best job that can be done, there occurs more or less growth and accumulation of bacteria in the mouth at protected locations on the teeth and elsewhere during the night. It makes for somewhat more cleanliness and less decomposition of food in the mouth during the daytime if the teeth are brushed and vigorously rinsed in the morning before breakfast.

How to brush the teeth.—The most important areas to be cleaned with the toothbrush are (a) the occlusal pits and fissures, (b) the proximal surfaces in the sulci between the teeth as far as the bristles may go, (c) the surfaces of the teeth within the gingival crevices to the extent they are accessible to the application of the bristles of the brush. It is done by applying the ends of the bristles to the area with firm pressure and moving the brush back and forth ("vibratory motion") with short strokes, thereby dislodging soft material by the digging action of the ends of the bristles wherever they can be applied.

The bristles of the right kind of brush are smaller, therefore more flexible (easy to bend) than those of other brushes; they are all of the same length; the tufts are properly spaced for most effective use; (microscopically) each bristle is round or smooth on end. This brush was designed and the specifications for it were determined [19] after first having accurate information as to the conditions to be met and the size and shape of the spaces to be cleaned with it.

All the surfaces of all the teeth to which the bristles of the brush can be applied, should be brushed. A good routine is to brush the buccal and labial surfaces of all the teeth first, then the occlusal and lingual surfaces of the grinders in all four quadrants, and finally the lingual surfaces of the anterior teeth.

In brushing the buccal, labial, and lingual surfaces the bristles should be forced directly into the gingival crevices and into the sulci between the teeth, at about a 45 degree angle to the long way of the

teeth. With the bristles forced into the crevices as far as possible, short back and forth movements of the brush dislodge all soft material which they reach on the tooth within the crevice. At the same time the teeth are cleaned above the gum in the sulci and between the teeth, as far as the bristles may go.

At first there is more or less bleeding from the inflamed crevicular gingiva, but this rapidly ceases as the inflammation subsides and the tissues toughen up. Thereafter no reasonable amount of vigorous brushing with this brush causes bleeding, except for such periodontoclasia lesions as may not have healed yet.

A good way to brush the buccal and labial areas is to close the mouth with the teeth nearly together, then put the brush inside of the cheek and brush the lowers and uppers alternately, on one side of the mouth, moving forward and across the anteriors and changing to the other side, brushing the teeth there in the same way.

The occlusal surfaces of the grinders are to be brushed by applying the bristles to the surface, pressing down firmly and moving the brush back and forth with short strokes. This digs out any soft material that may be present in the occlusal pit and fissure depressions.

Usually at this same time the back teeth are brushed on the lingual side in the same way as on the buccal side. The bristles should be directed firmly, at about a 45 degree angle into the sulci between the teeth and especially into the gingival crevices.

This is the time to brush around the back teeth, the most unclean and most neglected place in the mouth. There is a certain relaxation of the jaw (a certain opening of the mouth) which each person must learn for himself, which permits the application of the bristles of the tip (or toe) of this brush to be applied into the space around the back tooth in a way that no other brush could be applied. By the right pressure, application and brush movement one can learn to clean around his back teeth in a surprising and pleasing way. Soon one learns to brush simultaneously the occlusal surfaces, the lingual sides of the grinders, and around the back teeth, in each of the four quadrants.

Finally the anterior teeth are brushed on the lingual side, by directing the bristles of the heel or of the side of the brush into the gingival crevices and the sulci between the teeth at about the same 45 degree angle as at all other places.

Although we have described the brushing in several different locations as if it were done in separate stages actually one proceeds without distinction or separate stages until the brush has been applied, as best it can be, to all areas which can be reached with the ends of the bristles. Anyone who knows how, can brush the teeth as well as is necessary in less than one minute. It is not a matter of how long to brush but to *do it right by this method,* which is quite different—almost the opposite in important particulars—from methods generally advocated or followed. It consists essentially of forcing the ends of the bristles of a brush of appropriate specifications directly into the gingival crevices and into depressions where food material and bacterial film tend to accumulate, and by short stroke movements, dislodging and removing such material.

How to clean the teeth with dental floss.—By the above method of brushing, the bristles of the brush have been stuck in between the teeth and into the gingival crevices as far as they will go, from the buccal or labial side and from the lingual side, but there will be a place along the middle between the teeth where the bristles have not met. The proximal surfaces not reached by the bristles, and those within the gingival crevices, have not been cleaned. The only way by which these important areas can be cleaned is by the proper use of the right kind of dental floss. The optimum characteristics of dental floss for this purpose have been determined,[72] after first having ac-

curate information as to the conditions to be met and the functions to be served by it.

This right kind of dental floss consists of a large number of microscopic nylon filaments, not waxed, and not twisted except just enough to hold it together when in use. It is easy to understand that drawing (scraping) this bundle of microscopic filaments crosswise over a tooth surface would tend to cut off, dislodge and remove microscopic material that may be present there. The bacterial film and other material to be removed consists of microscopic particles.

Each person must be taught how to use dental floss right. Only a few people now know that the use of dental floss is necessary and those who do, in most instances, do not use it effectively for the purpose for which it is required.

For the purpose of cleaning the surface around the contact area, thereby preventing caries activity there, it is only necessary to pass the floss through the contact and back out. This cleans the proximal surfaces above the gum which have not been reached by the bristles of the brush.

For the purpose of preventing periodontoclasia, and preventing advancement of existing lesions, those tooth surfaces within the gingival crevices which cannot be cleaned with the brush, must be cleaned with dental floss. The foreign material on the tooth within the crevice, which causes and promotes the advancement of periodontoclasia, extends to the very bottom of the lesion. Therefore, to remove this material the floss must be carried to the bottom of the lesion.

The floss must be held firmly against the tooth and drawn outward (perhaps sometimes slightly endways also) so as to cut off and dislodge the bacterial and other microscopic material on the tooth within the crevice. The large number of separate nylon filaments of which the floss is composed, when drawn crosswise over the surface of the tooth from the bottom of the lesion outward, not only dislodge microscopic material but much of this is retained between the filaments and is removed with the string.

It does not matter what system is followed in cleaning the teeth with dental floss just so the floss is carried to the bottom of the crevice and then drawn firmly across the surface of the tooth from the bottom of the crevice outward. Different people develop their own technic and manipulations for cleaning their teeth with dental floss. They have to be taught by someone who knows how it should be done and why. The person must be shown that he has diseased gingival crevices, that the teeth are unclean within them, and that the floss must be carried to the bottom of the crevice in order to clean the tooth at this important location.

After considerable experience in presenting this method to a good many dentists and dental students, and in teaching other people how to clean their teeth with dental floss, this author has not found any change needed in the detailed directions he published several years ago,[1] which are quoted verbatim as follows:

1. Cut off a piece of floss about 2 to 3 feet long.

2. Wrap one end with 2 or 3 turns around the first phalanx of the right index finger, for the purpose of anchoring or holding it.

3. Bring the floss over the end of the right thumb which is also held against the finger around which the floss is anchored.

4. Grasp the floss with the left hand and bring it over the end of the first finger of that hand. Thus a length of floss, about 1 inch long, is held between the thumb of the right hand and the first finger of the left hand. (Fig. 25-1.)

5. Now with the thumb inside of the cheek and the finger inside of mouth, the floss is carried to the very bottom of the gingival crevice back of the last right upper tooth, drawn slightly endways through the crevice and crossways outward across the distal surface so as to

scrape off and dislodge the soft bacterial material on the tooth within the crevice and outwards.

6. Holding the floss in the same way, pass it into the next interproximal space. Carry it to the bottom of the posterior

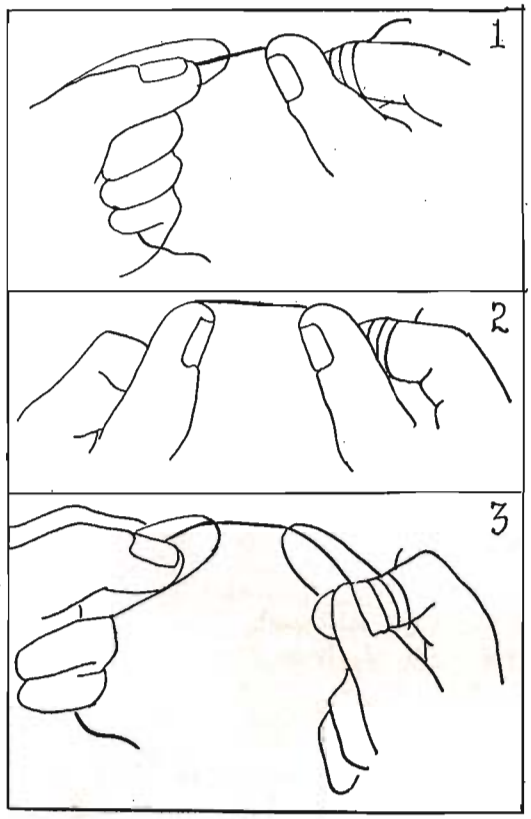

Fig. 25: Good way to hold dental floss in cleaning the teeth. For upper right 1, upper left, 2, lowers 3. See text for details.

gingival crevice and clean the mesial surface of that tooth. Now, before withdrawing the floss from this interproximal space, clean the distal surface of the other tooth in the same way. Then withdraw the floss and move on to the next interproximal space, etc., until the proximal surfaces of all teeth have been cleaned.

7. In passing the floss between contacting teeth it is not forced directly in and out. It should be held over the contact and drawn gently and slightly back and forth endways. This allows the low-twist, unwaxed floss to flatten and pass between the contacting teeth with the greatest ease.

8. After cleaning 2 or 3 teeth the part of the floss used is somewhat soiled and loaded with bacterial material. It is desirable to move along the string to a new place by taking another turn around the anchoring finger. This should be repeated from time to time as needed.

9. The floss is held and manipulated with the same fingers as indicated above until after the surfaces of the teeth in the interproximal space between the left central and lateral have been cleaned.

10. In cleaning the rest of the upper teeth, it will now be found more convenient and practical to hold the floss over the ends of the thumb of the right hand as before and over the thumb (instead of the index finger) of the left hand (Fig. 25—2).

11. All the lower teeth now should be cleaned in the same way. Most people will find that they can carry out the necessary manipulations most successfully with the floss held over the ends of the middle finger of each hand instead of the thumbs or the thumb and first finger as in cleaning the upper teeth. (Fig. 25-3)

12. After cleaning all the teeth with dental floss, the mouth should be thoroughly rinsed by forcing water vigorously back and forth between the teeth in order to remove material that has been loosened or dislodged but not removed by the floss. After a little experience one can clean all his teeth well with dental floss in from two to three minutes.

13. It gives a pleasurable sensation of cleanliness to hastily brush the teeth again after cleaning them with dental floss. But this is not essential.

Since practically all adults and many others have active periodontoclasia lesions of various extent around part or all of their teeth, there will be more or less bleeding of their inflamed gums when they first begin cleaning their teeth with dental floss. This soon ceases and the previously inflamed tissue rapidly heals. No bleeding or discomfort is experienced from any reasonable manipulations of the floss in the future. One soon realizes

that his teeth are cleaner than they have ever been before.

DISCUSSION AND COMMENT

Although the method of personal oral hygiene herein described has been perfected and some of the information upon which it is based has been established or clarified during the past few years, actually the fundamental ideas as to the cause of these diseases of the teeth and gums, and quite similar effective means of preventing them, were known and taught almost one hundred and thirty-five years ago. If the method then known could have been available and followed generally since that time, countless millions of people would have avoided untold disease, impairment, and final loss of some or all of their teeth; and the resulting disfigurement, discomfort, and other unhappy consequences.

In the Charles Edmond Kells Collection in the Rudolph Matas Medical Library at Tulane University, there is a 200 page booklet [81] entitled "A Practical Guide to the Management of the Teeth, Comprising a Discovery of the Origin of Caries or Decay of the Teeth with its Prevention and Cure," written by Levi Spear Parmly ("Dental Professor") and published in 1819. No doubt copies of this book can be found in other collections and in other libraries. Anyone claiming that our present conception of the cause and prevention of the diseases of the teeth — caries and periodontoclasia — is new, owes it to himself to read this treatise.

Parmly recognized and asserted, without reservation, that these diseases of the teeth and gums are caused entirely by local conditions of uncleanliness and that they can be prevented only by cleaning the teeth effectively. Up to this time most authorities considered that caries originated within the tooth and that gum disease was due to systemic conditions. If not the first, Parmly was certainly one of the first, to claim that local uncleanliness is the essential cause of decay of the teeth and to devise an effective method of cleaning them for prevention of the disease. He was enthusiastic about his "discovery" and asserted that "if the teeth and gums are regularly cleaned with the dentifric apparatus recommended by the author, no caries can possibly take place." "*** the interstices and irregularities of the teeth afford a lodgment for whatever is taken into the mouth, and no contrivance *hitherto discovered* can, from these parts, remove the accumulations."

Parmly came to New Orleans in 1824, taught his methods and gave several lectures on care of the teeth. He was from England where he was celebrated for his knowledge and methods. He also practiced and lectured in this country, in New York, Savannah, and Charleston, in addition to New Orleans. Copies of these lectures in our library present ideas and methods advocated by him at that time with which, with only slight improvement and modernization, our present information and methods entirely coincide. His "dentifric apparatus" consisted of three parts. Although there is no exact description of them, there is general information as to what they were and how they were to be used. "The first part to be used is the brush. It is made hollow in the middle to embrace every part of the teeth except the interstices; and thus, at one operation, the top (a part hitherto entirely neglected), the outer and inner surfaces are completely freed from all extraneous matter. The second part is the polisher for removing roughness, stains, etc., from the enamel and restoring the teeth to their natural smoothness and color." I do not find any description of the polisher.

"The third part is the waxed silken thread which, though simple, is the most important. It is to be passed through the interstices of the teeth, between their necks and the arches of the gums, to dislodge that irritating matter which no brush can remove, and which is the real source of disease. With this apparatus, thus regularly and daily used, the teeth and gums are preserved from disease."

In describing his method of cleaning the teeth with his "untwisted waxed silk" floss, he says "although the gums may first become subject to slight bleeding, yet in a few days, by perserverance in the treatment recommended, this bleeding will soon cease." Persons subject to inflammation of the gums "should clean them often with the waxed silk; when a new and healthy action will be communicated to the gums and they will be restored, in a short time, to their naturally firm and adhesive state."

Some of his other statements are especially interesting. "The first and most important object is cleanliness of the mouth, which is the only preventive of disease." "Where the teeth are kept clean and free from such objectionable matter." (relics of food undergoing putrefaction) "no disease will ever arise. This being the case, the means of prevention are clear and simple; and it is in the mode of cleaning them, that the whole secret of avoiding disease consists." This can be done only "in a proper manner, by using the dentifric apparatus described."

Disease of the teeth is "the effect of carelessness, inattention, or other want of cleanliness. It is in the power, therefore, of every individual to preserve the teeth and gums in perfect health." The advice relative to children's teeth, which he gave then, is quite applicable today. "In childhood the mouth should be cleaned every evening. The relics of food, which have been all the day accumulating, are thus prevented from committing their ravages during the night; and the habit of cleanliness will become fixed, from being so essentially connected with personal comfort." "When the permanent teeth begin to make their appearance, then is the time that the greatest attention to cleanliness is particularly necessary."

In the light of our present knowledge as to the essential etiological conditions in periodontoclasia, it is evident that no method of personal oral hygiene could be effective in preventing it, without the proper use of dental floss. Throughout the literature during the present century, many authors mention the use of floss silk or "flossing" as a part of the home care of the teeth. In most instances methods of using it suggested or implied could not be effective because they do not include cleaning the tooth within the gingival crevice. Joseph Head,[82] in 1917, described his method of "floss-silking" the teeth which evidently did include application of the floss to some extent within the crevice. He says the silk should go "up well under the frenum of the gum ***across the gum and up into the opposite frenum." His statement that "the gums at first naturally bleed" further indicates that the method he advocated included carrying the floss into the crevice.

Proper use of dental floss is necessary for maintenance of both oral cleanliness and dental health. Whatever advocacy and promotion there may have been of this necessary part of personal oral hygiene, it remains a fact that, as yet, very few people use dental floss. Out of more than 900 people—mostly university personnel and dentists and dental students —whom I have examined during the past several years, less than half a dozen were using the available dental floss effectively and thereby maintaining a high degree of oral cleanliness and dental health. Some others used it irregularly and ineffectively.

During recent years there has been a wave of promotion of the idea that the teeth must be brushed soon after each meal. It is based upon the observation by Stephan and others[83-87] that the application of sugar solutions to accumulated dental plaque material ("materia alba") is quickly followed by marked increase in acidity.

Fosdick[88] and a group of cooperating investigators carried out a large clinical experiment to ascertain the effect upon caries incidence, of brushing the teeth within 10 minutes after each ingestion of food or sweets. The brushing was to be followed by thorough rinsing of the

mouth. The controls followed their usual procedure which "generally consisted of brushing their teeth night and morning."

The results over a one or two year period indicate that the incidence of caries is reduced between 50 and 60 per cent by brushing (and/or rinsing) the teeth right after ingesting food or sweets. Both test subjects and controls had a considerable increment of caries lesions during the experimental period. The implication is that these were new lesions which were not present at the beginning. Any method of personal oral hygiene which allows the development of any new caries lesions, is inadequate.

It has long been well known that when fermentable carbohydrates are applied to a mass or accumulation of acidogenic bacteria, acids are produced quickly. If on the other hand, culture media containing the same fermentable carbohydrate is, inoculated with a relatively small number of such acidogenic bacteria, it requires hours of incubation and multiplication before significant amounts of acids are produced. This is common practice in bacteriology in testing acid and gas production of organisms.

In carrying out the experiments to test acid production of plaque material in situ, or to collect a sufficient amount for tests outside of the mouth, it is necessary for the subject to refrain from cleaning the teeth for some time, usually three or four days, to allow time for accumulation of sufficient suitable material for the purpose. How different these conditions are from those that exist when the teeth are properly cleaned of food and bacterial material with both toothbrush and dental floss every night before retiring! When this is done there is not time for harmful amounts to grow and accumulate during the following day before time to clean them the next night; or for sufficient amount for testing purposes either.

Advocating and promoting brushing the teeth after every ingestion of food, for the purpose of lessening caries incidence, encourages the misbelief that caries is the main disease of the teeth (periodontoclasia is the most important for adults), that the teeth can be effectively cleaned with the brush (which they cannot), and it tends to detract attention from the importance and application of the essential information that the teeth must be cleaned with both toothbrush and dental floss every night before retiring.

The oral hygiene (oral cleanliness) habits of different individuals vary greatly, as do the other habits of personal cleanliness. These are acquired in the home. During early childhood the child must learn by experience and the example of others that preparation for retiring at night must include thorough cleaning of the teeth. It should be an established practice in the home that no one may retire with an unclean mouth.

The young child cannot clean his teeth effectively; therefore, it must be done for him by the parent, who must know how to do it. During the first several years, use of the floss is not necessary. The teeth can be cleaned with the brush well enough to prevent caries, if it is done right. The most important part for the child is cleaning the occlusal surfaces of all the chewing teeth by firm application of the bristles to these locations, since almost all cavities in childhood originate on the occlusal surfaces of the grinders.

After a child is several years of age and the permanent teeth are in place, then it is important that the teeth should not only be cleaned with the brush, but that dental floss should be passed (in and out) between contacting permanent teeth, so as to insure against proximal caries activity. At some age, usually between 6 and 10, the child who has been well trained can take over his own oral hygiene procedure, at first under supervision and finally without.

At the present time most children have already sustained caries damage to some or several of their teeth, especially the grinders, by the time they are 12 to 15 years of age. This is entirely preventable and the parents have full responsibility

for it. The responsibility cannot be placed upon the child or others. For a child to develop even one caries lesion is a reflection of neglect and deficient or uninformed guidance in this regard.

In general, as each person advances in age, there is a constantly increasing accumulation of dental disease and injury. Although such damage as has already occurred at any given time, cannot be prevented now, for all practical purposes, and except for some special circumstances, any person can begin at any age and, by the right method of personal oral hygiene herein described, prevent the occurrence of any new caries or periodontoclasia lesion during the remainder of life. The oldest person the author has had under close observation is now 78. For years he has been able, by this method, to maintain the maximum degree of oral cleanliness and dental health.

People depend upon practicing dentists for information and advice relative to dental health. The knowledge, ideas, and methods of dentists are acquired largely through their training in the dental school. Up to the present time, the fundamental facts upon which this method of preventing the diseases of the teeth is based and the method itself, have been taught, and that in a restricted or limited way, in only two of the dental schools of the country, Loyola in New Orleans and University of Texas in Houston. Short course intensive postgraduate instruction has also been given to small numbers of dentists, who then return and enthusiastically teach the method to their patients.

It should be understood that this is not just another method of personal oral hygiene to try on patients. The dentist who understands and knows of his own knowledge (not the opinions of others) the basic facts relative to the local etiological and pathological conditions in these diseases, already knows in advance that this method would be necessary to prevent them. He would be following the method himself. Until he does, he is not prepared to teach it to his patients.

SUMMARY

Attention has been directed to the essential local etiological conditions in caries and in periodontoclasia. The lesions of these diseases are caused, and their advancement is promoted, by uncleanliness at vulnerable locations on the teeth which are not naturally kept clean.

A limited number of illustrations have been included to visualize some of the ideas in the text. Reference is given to a larger number in the literature confirming the etiological conditions at the locations where the lesions of caries and periodontoclasia originate and advance.

Caries activity, and to a large extent, periodontoclasia activity, occur at night during quiet and sleep. To prevent them the teeth must be thoroughly cleaned at night before retiring, thereby securing a long period during which there is no food material about the teeth to ferment or decompose, and relatively little growth of harmful bacteria takes place.

The teeth can be effectively cleaned at the important locations only by the proper application of the bristles of an appropriate toothbrush and the filaments of the right kind of dental floss. Details of the method are given. These differ from, and in some particulars are the opposite of, those usually followed.

To maintain a high degree of oral cleanliness, and to prevent caries and periodontoclasia, *the teeth must be cleaned right, with the right kind of both toothbrush and dental floss, every night before retiring.*

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