The manuscript for this paper was submitted to, and declined for publication by, several of the foremost national dental journals in America. I believe that the information will be interesting to dentists who have been interested in some of my other papers and teachings in this field. Therefore I have had it printed to send to dentists on my list, and to others upon request.

THE AUTHOR

PREVALENCE OF PERIODONTOCLASIA*

by

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PREVALENCE OF PERIODONTOCLASIA

by C. C. Bass, M.D.

At the present time there is much confusion and lack of sound information[1] as to the prevalence, etiology, diagnosis and prevention of periodontoclasia. Testifying before a U. S. senate committee in 1956, Dr. Harry Lyons,[2] then president-elect of the American Dental Association, dean of the School of Dentistry of the Medical College of Virginia and member of the National Advisory Dental Research Council of the National Institute of Dental Research, stated that "so-called gum diseases are essentially complete mysteries as far as their causes and prevention are concerned." This probably correctly indicates the general state of knowledge about this disease.

In the November, 1957, number of the J.A.D.A.[3] there are a total of 17 articles relating to periodontal disease, including an editorial discussion. The authors include specialists in the field, one dean and several professors, associate and assistant professor in dental schools. These articles, as a whole, indicate continuing widespread confusion and mystery as to the prevalence, cause, and prevention of one of the most prevalent and most important diseases of man.

The purpose of this paper is to point to the fact that periodontoclasia is a universal disease of man, always present in some stage and advancing from early childhood until the teeth are finally lost (extracted or exfoliated) mostly during the second half of the normal life expectancy period. Simple procedures will be given whereby the interested worker, with suitable facilities, can see for himself and know his own knowledge the facts as to the prevalence of this disease, and will not have to depend upon the ideas and opinions of others, which too often may be misleading.

Pus in Gingival Crevices Indicating Inflammation.

For illustration start with any average 7 or 8-year-old child and make microscopic examination of material from the gingival crevices, especially the interproximal crevices. Very simple technic is sufficient for this purpose. Material is taken from the crevice with a suitable instrument,* spread upon a slide, fixed with heat, stained with a drop of 0.5 per cent solution of crystal violet, washed, dried, and examined with the microscope for pus. More or less pus cells are always found present in properly collected and prepared material from any such child. This proves the presence of more or less inflammation and suppuration at the location from which the material was taken.

For illustration take next any average 10 or 11-year-old child. Properly collected material from the gingival crevices always contains pus cells, usually in noticeably greater amounts than in the younger child. This shows that there was continued inflammation and suppuration of the crevicular epithelial tissue at the location from which the specimen was taken.

* The best instrument for this purpose I have ever seen is similar to one which I designed for my own use and briefly described several years ago.[4] It has since been made in quantity by O. Suter, Dental Mfg. Co. of Chico, Calif., and is designated the "Bass 55" explorer and scraper. The 0.55 mm wide blade is properly shaped for best application into the gingival crevices and for removing material therefrom for microscopic examination. I would be badly handicapped without this instrument for exploring and demonstrating periodontoclasia lesions.

The next illustration may be made by taking any average 13 or 14-year-old child. Purulent material is more abundant from the crevices and, upon microscopic examination, contains still greater amounts of pus cells. This shows that there is continued inflammation and suppuration at the location.

Next consider any average 16 or 17-year-old boy or girl. Deeper lesions can be found around some of the teeth, often the inflamed gums bleed easily when disturbed and properly collected material from the crevices (periodontoclasia lesions) contains a great abundance of pus cells. The inflammation and suppuration has continued. Often some receeding of the epithelial attachment at particular locations can be recognized.

The personal oral hygiene habits of the individual, at any age, profoundly affect the progress of the disease process. There is a great variation in the rate of progress, from those with total neglect, to those who do try to follow the best method they know. By this age, and sometimes before, increased interest in personal appearance and oral cleanliness, plus usually more or less trouble and expense on account of caries damage, lead to increased attention to brushing the teeth. Although the methods of brushing generally known and followed[5] are inadequate to entirely prevent periodontoclasia they do tend to retard the advancement of the disease.

The age range of 20 to 25 years may be taken next for illustration. "Pyorrhoea pockets" are found around some of the teeth, especially around the molars and biscuspids, usually the deepest between the first and second molars. Pus is always obtainable and matetrial from the lesions (crevices) always contains an abundance of pus cells.

More than a third of the people in this age period, even those at the more favorable cultural and educational level, such for instance as college students, already have ameba, *Endameba buccalis (Entameba gingivalis, Gros)*, in their "pyorrhoea pockets."[6] A large proportion of all people of this age or older with negligible or very inferior oral hygiene habits have these ameba. No person, once infected, ever loses the infection until all the teeth are lost. This parasite is easily recognized in preparations made in the simple way described above, after one has become familiar with their characteristic appearance in such preparations. It is not known what influence, if any, this parasite may have upon the progress of the disease process.

A frequent symptom of the disease, bleeding gums, is observed more often perhaps during this period than during any previous or later 5-year period.

Loss of Teeth Mostly After Middle Life.

By the age range of about 35 to 40 the influence upon the progress of the disease from the personal oral hygiene practices of the individual, in addition to individual variation in resistance or susceptibility to the disease, makes for still greater variation in the clinical picture. Those with total neglect not only have demonstrable lesions around all their teeth, but usually by this time some of the teeth have drifted ("traumatic occlusion") more or less and some may have been lost. In addition to the back teeth there are deep lesions about some of the incisors which have heavy subgingival calculus and may be somewhat loose to pressure. Often visible pus can be pressed out from the lesions.

Although some teeth may be lost, rarely all of them, from this disease before the age of 40, the final stage and total loss usually occurs during the age period from 40 to 60, in people who make inadequate, inappropriate or no oral hygiene effort.

Those whose mouth care methods have been good enough often retain several (rarely all) of their teeth beyond this period. The progress of the disease has been retarded but there are always demonstrable lesions and suppuration about every tooth in the mouth.

Occasionally, but rarely, there is some person in the eighties or nineties many of whose teeth are still retained and functioning. In most such cases the history is that since early childhood, certainly before too much damage occurred, a method of personal oral hygiene has been faithfully followed which included vigorous brushing of the teeth and gums at night before retiring.

Receding of the Epithelial Attachments.

Another way of knowing the early beginning and the progressive advancement of periodontoclasia is to examine, by appropriate technic, large numbers of specimens of extracted teeth. Formalin preserved specimens from extraction clinics are suitable. A dissecting microscope with 10x to 15x magnification is necessary. In fact proper use of this instrument for studying extracted tooth specimens is absolutely necessary for anyone to know of his own knowledge, and to fully understand the local etiological and pathological conditions in either caries or periodontoclasia.

As the periodontoclasia lesion advances on any tooth the epithelial attachment recedes apexward. The zone of disintegrating epithelial attachment cuticle, "zdeac,"[7] is a demonstrable line which indicates the exact location of the outer (occlusalward) border of the epithelial attachment when the tooth was *in situ*. Quite simple technic is sufficient for the purpose. Stain the specimen in 0.5 per cent crystal violet for about one minute. Brush off under running water with a suitable soft toothbrush.* Hold the specimen with forceps† and examine, keeping it still wet, under the dissecting microscope. The violet stained line, zdeac, can be found extending part way, sometimes all the way, around the tooth. Wherever it is found it indicates the exact location of the very bottom of the periodontoclasia lesion at any place when the tooth was in the mouth.

On such preparations one can see for himself the close relation of the inner (apexward) border of subgingival calculus (this is not removed by brushing) to the zdeac.[8] On specimens stained but not brushed off one can see how the inner border of the subgingival bacterial film on the tooth and over the calculus extends to the outer border of the zdeac.[9] This is important because the advancing calculus and bacterial film on the tooth within the crevice (pocket) constitute the essential local cause of the disease and of its advancement. Without this foreign material on the tooth within the crevice periodontoclasia does not exist.

The epithelial tissue attached to and surrounding the tooth—epithelial attachment first so designated by Gottlieb in 1921[10]—normally is located entirely on the enamel and extends from the cemento-enamel junction occlusalward. The outer border is always within the gingival crevice. The epithelial attachment would remain on the enamel throughout life were it not for disease or injury. As the periodontal fibers are destroyed, as a part of the periodontoclasia inflammatory process, the apexward border of the epithelial attachment grows downward over the cementum from which the fibers were removed. Concurrently the outer border of the epithelial attachment recedes leaving more and more of the surface of the tooth with no attached soft tissue. As the disease progresses, over the years, more periodontal fibers are destroyed and the epithelial attachment continues to move apexward. The location of the outer border of the attachment, which was at the very bottom of the (crevice) lesion, is shown by the zdeac on a properly prepared extracted tooth specimen. This landmark is always located on the enamel during the early stages of this disease. In the more

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* Although any soft toothbrush may be used it is better to have one with not larger than .007" diameter nylon bristles, all trimmed to the same length, properly spaced in the head and with the ends ground round or smooth in order to reduce scratching. Such brushes are not available commercially, but I will be glad to send a suitable one complimentary to anyone who wishes to follow up this subject.

† The most suitable forceps I have used for this purpose is Rochester Pean No. 422½ with a longitudinal groove cut in the grasping ends of the blades, as made for me by J. Sklar Mfg. Co., 38-04 Woodside Ave., Long Island City 4, N. Y.

advanced stages it is on the cementum, part way down the length of the root of the tooth. In the still more advanced and final stages it is usually on the apical half of the root.

Typical examples of the advancement of the zdeac on teeth have been illustrated in another publication.[11] However, one can know of his own knowledge and understand what occurs in this regard only by examining large numbers of specimens by the method indicated above.

COMMENT

Employing the method indicated the author has examined material from the gingival crevices of large numbers (more than a thousand) of people within and between the age ranges named above for illustration. Pus, and therefore inflammation and suppuration, was found in all of them, upon thorough examination.

Following the simple procedure indicated above, using the "zdeac" to indicate the location of the outer border of the epithelial attachment, large numbers of (more than two thousand) miscellaneous specimens of extracted teeth have been examined from people of various ages. The location of the epithelial attachment was found to be on the enamel on most of the younger teeth (supposedly specimens extracted within the first 1 to 5 years after eruption). On many of these, especially on the proximal surfaces, it had already receded part way or entirely below the cemento-enamel junction. On practically all older teeth it was found to be located on the cementum at varying distances between the cemento-enamel junction and the apex.

These observations will be confirmed by anyone following the necessary similar methods. The prevalence of periodontoclasia thus indicated by laboratory procedures contrasts sharply with numerous reports of findings in clinical surveys. As an example, in a recent paper[12] Russell, in his Table I, reports 92.1 per cent of Newburgh children, 7 to 14, found "negative for gingival disease." Table II indicates 52.8 per cent of his Colorado Springs group of adults "negative for periodontal disease." In a more recent paper Rovelstead and others[13] report a study of 2,027 random selected naval recruits, average age 18.5 years, entering U. S. Naval Training Center at Bainbridge, Md., from July 16, 1956 to Dec. 10, 1956. They found 20.8 per cent free from gingivitis. My experience is that 100 per cent of people comparable to those in these groups have active demonstrable periodontoclasia lesions in some stage.

These observations support Kronfeld's statement[14] as to the presence of evidence of pathosis in the gums, but not his interpretation of its significance. He states: "The presence of a small number of inflammatory cells in the subepithelial tissues can be considered an indication of gingivitis; but if it is almost every human gingival crevice would have to be considered pathological, which would only cause confusion." On the contrary, I believe refusing to recognize this pathological condition as such and calling it normal, because of great prevalence, is unsound and confusing.

All teeth with caries lesions or fillings have also demonstrable periodontoclasia in some stage. Such teeth, if not lost from caries, are finally lost from periodontoclasia in the same way as sound teeth are lost from this disease.

There are no symptoms by which the individual recognizes the presence of periodontoclasia as such, except perhaps during the last stages. He does not realize the existence or the nature of the pathological process that is going on. He has no way of evaluating its possible systemic effects over the long period of its existence.

SUMMARY

Demonstrable pus indicating inflammation and suppuration was found in material from the gingival crevices of all of a large number of people of different ages from early childhood to advanced age.

Evidence of progressive receding of the epithelial attachment from its normal location on the enamel to different levels along the cementum was found on a large proportion of many miscellaneous extracted tooth specimens.

Simple technical procedures are given for testing and confirming these findings.

These observations indicate that all dentulous people, at any age, have progressive demonstrable periodontoclasia in some stage.

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