AMERICAN ACADEMY OF DENTAL SCIENCE

HORACE L. HOWE, *Editor*

Meeting held February 9, 1921. Young's Hotel, Boston, Mass.
President L. M. S. Miner in the chair.

Scientific Proceedings. Paper by John Oppie McCall, A.B., D.D.S.,
Buffalo, N. Y., on "the etiology of periodontoclasia" (this page).
Discussion, by Drs. Edward C. Briggs and E. Melville Quinby, of
Boston, Mass., not reported.

THE ETIOLOGY OF PERIODONTOCLASIA

JOHN OPPIE MCCALL

Buffalo, New York

CONTENTS

I. Introduction.....	lix
II. Disturbances of function.....	lxi
III. Traumatic occlusion.....	lxii
IV. Congenital non-occlusion.....	lxiii
V. Etiology is dependent upon more than one factor.....	lxiii
VI. Extrinsic trauma.....	lxv
VII. Bacterial influences.....	lxv
VIII. Nutritional disturbances.....	lxvii
IX. Systemic disorders.....	lxvii
X. Local conditions affecting blood supply.....	lxvii
XI. Anatomical deficiencies.....	lxix

I. INTRODUCTION

To trace the history of the development of periodontal etiology would furnish a very interesting review, dealing as it does with the introduction of many theories that are widely divergent in the views they represent. It is not my intention to discuss the various theories regarding the etiology of dental periclasia that have been advanced. Unfortunately, the paucity of results that followed the application of the earlier ideas on etiology threw students of the disease into a state of mental chaos, in which the only

lix

THE JOURNAL OF DENTAL RESEARCH, VOL. III, NO. 3

stabilizing influence lay in the pronouncements and work of the pioneer Riggs. It has perhaps been the good fortune of students in this field that Nature has been less tolerant than usual in yielding results, in the clinic, when treatment founded on incorrect theories of etiology was instituted. In many branches of medicine, it has been found that treatment, even when its basis was only partially correct, yielded results that seemed to substantiate the verity of the proposed theory. Not so in periodontoclasia. Not only are all periodontal diseases progressive, resisting even the intermittent efforts of the wonder-working "*vis medicatrix naturae*," but they also refuse to yield any but the most meagre results to treatment that is not founded on the correct etiology of the case. Students of periodontal etiology, then, while constantly discouraged in their efforts to solve their problem, have at least been definitely and speedily informed of their lack of success. To obtain uniform and satisfactory results from treatment based on a given theory of etiology has, therefore, seemed a very definite and acceptable proof of the correctness of that theory.

The theory of periodontal etiology that I hold was presented in detail in a previous paper.¹ Since a full presentation of that theory would involve the recapitulation of many items that may be disregarded in a discussion of the underlying hypotheses, I respectfully refer you to that paper for details.

Etiology and pathology are naturally very closely interwoven. In studying the etiology of periodontal disease, we look to pathology for the reason why the various etiological factors produce the results we observe in mouths into which these agencies have found an entrance. The pathology of to-day deals not only with injurious agencies, but it also lays much stress upon interference with functions in the parts attacked. The underlying thought is that only when function is deranged can pathogenic agencies bring about a reaction that results in disease. Disturbance of function is the keynote of modern pathology, and it is upon this condition that the periodontist focuses his attention in studying periodontal disease. This thought, therefore, furnishes the foundation upon which our present concept of the etiology of dental periclasia is erected.

The functions of the periodontium, which include gingivae, alveolar process and pericementum, are complex. These functions center around the one chief function, namely, that of supporting the teeth in the performance of their function of mastication. The functions of providing nourishment for the teeth and for themselves, of aiding in the cleansing of the teeth,

¹ McCall: *Dental Items of Interest*, 1920, xlii, p. 116.

of protecting the cementum from caries-producing agencies, etc., all are subsidiary to the purely mechanical function of furnishing support against the stresses imposed by the muscles of mastication.

II. DISTURBANCES OF FUNCTION

With this thought of disturbances of function as our principal theme, we divide the disturbance of the mechanical function of the periodontium into *hyper-function or function plus*, and *hypo-function or function minus*. In a mouth having the normal number of teeth, arranged in normal occlusion, hyper-function is never encountered, since Nature applies no more pressure to the entire periodontium than it can successfully withstand. Tendencies toward hyper-function, in such a mouth, stimulate the bone cells of the parts to lay down larger and denser bones, by which the increased pressure is accommodated. A striking example of this is furnished by the Esquimaux, whose heavy maxillary structures have long been known to anthropologists. When one or more teeth are missing, or when one or more teeth are out of their normal positions in the arch and in such relations as to increase the stress to which they are subjected, disturbance of function, in this case a plus or hyper-occlusion, is instituted. In such a case, a potential disease-producing agency is at work, sometimes compensated by osteoblastic activity, more often constituting the entering wedge for a destructive complex that is destined to undermine these wonderful structures. On the other hand, when the opposite state of affairs is encountered, that is, a mouth in which a tooth has erupted in such a position that it has never come into occlusal contact with its natural antagonist, there is a condition of hypo-function—the occlusal relation known as congenital non-occlusion. Even when such teeth are sufficiently near their antagonists to permit of some degree of use in the incision or mastication of food, the absence of actual contact of tooth against tooth determines the condition as one of hypo-function. Pathological sequelae not only may, but usually do, actually ensue as a result of this disturbance of function.

Other disturbances of function that may enter into the causation of periodontal disease are mechanical interference with circulation, or with secretion and excretion in the marginal gingiva. The pressure or even the presence of bands, or the presence of deposits of salivary calculus, may impede circulation in a mechanical way, and may interfere with the free escape of fluid from the gingival crevice, whether this fluid be considered as a secretion or an excretion. In either event there is a disturbance of the protective and cleansing function of the marginal gingiva. Disturbances

of nutrition, whether due to dietary deficiencies or to neurotic influences, may be considered as agencies that undermine the function of these tissues, through the derangement of cell constitution. The presence of such agencies, and also their relative importance in the etiologic complex, is often difficult to determine but they are unquestionably factors of importance. When we include, with these, extrinsic mechanical injury, such as improper brushing and use of clamps, we have stated all the primary or inaugurating causes of periodontoclasia.

III. TRAUMATIC OCCLUSION

Inasmuch as the chief function of the periodontal tissues is support of the teeth in the process of mastication, it is but natural that interference with this function should furnish the dominating feature in the etiologic complex of many cases. When we realize also that the interlocking of cusp and opposing sulcus is usually extremely close, we find that only slight malposition of tooth or malformation of cusp is required to bring about the condition now known as traumatic occlusion. It is to be expected that traumatic occlusion will occur in mouths in which the teeth are obviously malposed, or in mouths in which drifting of the teeth has followed extraction; but it has been an astonishing thing to find traumatic occlusion in mouths that present the usual complement of teeth and when the teeth have an alignment that, even under careful scrutiny, appears to be normal. This fact can be readily demonstrated by the experienced periodontist.

Traumatic occlusion has been noted by several observers as a causative agency in periodontoclasia. They have noticed it chiefly in cases presenting marked loosening of the teeth, and have attached varying shades of importance to it as an etiological factor in such cases. It has remained, however, for Stillman to point out its full significance in such cases, and also to trace its influence back to the point where incipient symptoms alone could be detected, such as hyperemia of the pulp, gingival recession, marginal gingivitis, obscure alveolar pain, early pocket formation, etc.

A paper that is intended to deal with the entire field of periodontal etiology should not emphasize one part of that subject to such an extent that it would unduly minimize other important phases. Nevertheless, traumatic occlusion so frequently occurs in the etiologic picture, and its importance relatively to other factors is so great, that it seems almost impossible to overemphasize it. Traumatic occlusion, when present, overshadows the other etiological factors in the case to such an extent that no completely satisfactory curative result can be obtained except through its elimination.

In cases from which occlusal trauma cannot completely be eliminated, it remains a stumbling block in treatment and produces a constant retrogressive tendency. Since it produces an excessive occlusal function, it will be understood that the consequent injury is felt throughout the entire pericementum and in the entire alveolar structure. Keeping this thought in mind, the attendant alveolar absorption, deep invasion of bacteria with pocket formation, and pronounced circulatory disturbance, are readily explained.

IV. CONGENITAL NON-OCCLUSION

Hypo-function due to congenital non-occlusion, far from being a cause of injury as is traumatic occlusion, produces its harmful effects by inducing subnormal tone. The periodontal tissues in such cases have little resistance to infection or to mechanical injury, and thereby fall a prey to minor irritations and low grade infections. Not only are such tissues easily and quite deeply invaded by bacteria, but their reaction to injury is low grade, and they respond poorly to the stimulating agencies which may be employed during treatment. Quite the reverse is true of the hyper-function cases.

V. ETIOLOGY IS DEPENDENT UPON MORE THAN ONE FACTOR

We are prone to judge by the obvious, and when a case of periodontoclasia presents, if there is palpable calculus on the tooth surface or if a crown band extends beneath the marginal gingiva, it is usually concluded that this irritant is the chief cause of the trouble. A perfect scaling or trimming in such cases may give a very agreeable result, but how long does the case remain well? Does the tooth, if loose, become firm? For the correct adjudication of the etiology we must not only consider the obvious, but also search for every possible factor that may be implicated. We may frequently judge the relative importance of the various causative factors from the character of the lesion.

Reference has already been made to the "etiologic complex." It is a term particularly suited to indicate one of the peculiarities of periodontal etiology. Past writers have suggested almost every conceivable disease-producing agency as a "cause of pyorrhea." Anxious dentists, and patients too, ask the specialist whether anyone has found the "cause of pyorrhea." There is no *one* "cause," any more than there is one disease of the investing structures of the teeth. Not only are the causes numerous, including many of those suggested in the past but there must always be a combination of forces to inaugurate the disease. No *one* of them acting alone can do this.

The periodontist, then, has a wide range of factors for which he must search. And while each group of factors tends to produce certain types of lesion, the effects produced are subject to considerable modification by the interactions of the various possible combinations. For instance, traumatic occlusion tends to produce absorption of alveolar bone. If local and general resistances to infection are high, such absorption may be the only direct effect, the tooth becoming somewhat loose in its socket without visible inflammation, recession or pocket formation. If, however, resistance is low, the factor of bacterial invasion enters the picture, and the tendency is toward gingival inflammation and pocket formation, in addition to mobility of the tooth. On the other hand, if the strength of the alveolar structure is more than adequate to meet the mechanical demand, and resistance is high, a traumatic occlusion is apt to concentrate its irritation at the tooth apex, producing thereby a pulp irritation, which may express itself as an obscure pain in the affected or nearby region, as an active hyperemia in the pulp of the tooth itself, or as a hypersensitiveness of the tooth surface. When traumatic occlusion is complicated by bacterial activity, the tendency is toward a deep invasion, producing not only deep pockets but even infection of the pulp before the pocket has involved the apical region.

When calculus or other irritants of the marginal and cemental gingiva are present, and are not complicated by traumatic occlusion, there is little effect on these structures unless there is a recognizable susceptibility to infection. Such infection does not penetrate deeply into the pericementum but its effects are confined almost wholly to the marginal and cemental gingiva. The continuous accumulation of calculus, together with the uninterrupted activity of the bacteria, may produce a gradual wasting away of these tissues. In such cases, however, the pericementum is destroyed at about the same rate as the marginal and cemental gingiva, and there is consequently no marked pocket formation. A slight change in the balance of the influences of calculus, bacteria, and local resistance, may produce a hypertrophic gingivitis, instead of a pure destructive process.

When we realize that one of the functions of the marginal gingiva is to secrete a fluid in the gingival crevice, which assists in the cleansing of the enamel surface that is overlapped by this tissue, we find it only reasonable to assume that the presence of calculus or a crown band will cause, in addition to irritation, a partial damming up of this crevicular secretion, and a consequent slight engorgement of the gingival tissue with the serous fluid. What effect this has on the composition of the secretion will be difficult to determine, but clinical evidence points to a change in its composi-

tion. Cases involving traumatic occlusion also give evidence that is strongly suggestive of a marked change in the composition of this crevicular fluid. We are here concerned, however, with the effect that its partial retention may have on the gingival tissue itself. Like all retained secretions or excretions it is undoubtedly somewhat toxic, and must at least tend to interfere with the normal circulation in the part.

VI. EXTRINSIC TRAUMA

Extrinsic trauma, such as a bruise, operative injury, improper brushing, etc., does not directly disturb function, but is more nearly a pure irritant. It naturally follows that the injury produced is neither far-reaching nor serious, but is usually confined to the vicinity of the marginal gingiva and alveolar crest. Here again, various combinations of causative factors produce widely varying clinical pictures. Food impaction against the gingival tissue is properly considered an extrinsic injurious agent, since it is introduced from without. A distinction is thus made between such injuries and the injuries caused by traumatic occlusion, calculus, etc., which are always in the mouth, and are thus acting continuously. Food impaction itself causes pain and a circulatory disturbance in the affected gingival tissue, but pocket formation is absent or is shallow. Food impaction combined with traumatic occlusion and bacterial activity, however, is apt to cause pocket formation.

VII. BACTERIAL INFLUENCES

The secondary etiological factors in periodontoclasia include all agencies that tend to cause a breaking down of periodontal tissue but which are without ability to inaugurate this destructive process. By far the most conspicuous member of this group is bacterial injury. It is difficult for many writers to concede the secondary influence of bacteria in diseases of the periodontium. It is true, also, that certain characteristic features of these diseases do not occur except when the tissues become infected. The destruction of pericemental fibres is accomplished only by forcible mechanical pressure, as by the driving of a crown band, or through bacterial activity. Thus, it is possible for a typical pus pocket to be formed only when bacteria penetrate and proliferate in the pericementum. Looking at the question in this light, it might be said that the bacteria were the prime cause of the pocket formation. It is also true that, if these bacteria could be entirely kept away from the gingival crevice, pocket formation could never occur. This again might seem to suggest that bacteria are primary factors.

As for the last point, it is possible both theoretically and practically for pathological conditions to develop in the periodontium without the tissues becoming infected and without the pericementum losing its continuity. The chief reason why bacteria should be considered secondary invaders, however, is the fact that their injection into previously healthy gingivae fails to produce periodontoclasia. Moreover, the typical suppurative types of disease invariably exhibit evidence of a previous preparation of the diseased tissue by some agency that lowers its resistance. This preparatory agency may be a disturbance of function, an undermining of the resistance by nutritional disturbance, or an extrinsic mechanical injury, in short, any of the agencies which we have already denoted as primary etiological factors.

It cannot be too strongly impressed upon all students of periodontal disease that the bacteria found in these cases are incapable of proliferating within the gingival tissue except as its resistance has been lowered by some other agency. This other agency may be so slight in the beginning as to escape observation, but it is unwise to assume, because of this fact, that it is lacking. Even in the virulent form of gingival infection known as phagedenic gingivitis or Vincent's gingivitis, there is so universally a palpable preparatory irritation or tone depression, that the secondary character of this infection is maintained. It has been held that, when this infection reaches epidemic proportions, as it did during the late war, the Vincent bacterial group is capable of infecting previously normal tissue. Even in the camps, however, an appreciable percentage of men failed to acquire this disease, a fact explainable only through the possession on their part of an adequate general and local resistance.

The significance of the secondary nature of infection in periodontoclasia will surely not be overlooked when the subject of treatment is to the fore. Antiseptic treatment has a value proportioned largely on the virulence of the bacteria, and the importance of their position in the etiologic picture. In the chronic types, the infection is distinctly low grade and the important etiological element is that of disturbance of function. Treatment by antiseptics, vaccines or stimulants, gives comparatively little benefit. The benefits of such treatment are short-lived, also, unless the primary factors have received adequate attention. In the acute types, of which phagedenic gingivitis is the most severe, antiseptics play a more important rôle. But even in this type of disease, antiseptic treatment has very definite limitations when used alone, and the final clearing up of the infection depends on the mechanical and surgical treatment, rather than on the antiseptic.

VIII. NUTRITIONAL DISTURBANCES

The usual position of nutritional disturbances, including dietary deficiencies, is in the group of secondary factors, although we have already mentioned them as occurring in the group of primary factors. It is true that dietary deficiencies and scorbutic influences may be primary undermining factors in periodontal disease. It is seldom possible, however, to demonstrate this through the medium of dietary treatment except in the young. Given a patient who has reached maturity with a fairly normal periodontium, dietary shortcomings seem to exert but little influence on these tissues, except as a secondary agency. Scurvy in a definite form is so rare, in the present-day American adult, that no authoritative statement can be made as to whether it is primary or secondary in etiology. While diet is very important during the developmental period of the periodontal tissues, it is safe to say that after maturity, it becomes distinctly a secondary factor in periodontoclasia.

IX. SYSTEMIC DISORDERS

Much has been written about the vicious effects produced in the mouth by gastro-intestinal disorders, diabetes, syphilis, and kindred diseases. These diseases do have a depressant effect on the oral tissues, frequently leading to the formation of more or less characteristic lesions. Were they primary in their significance, treatment of cases complicated by these factors could not give satisfactory results except after the abnormal systemic condition had been eliminated. This has not been the experience of competent periodontists. Cases having diabetes and syphilis have yielded quite as well to proper local treatment as have those without systemic disease, although sometimes more slowly. We accordingly rate systemic conditions as secondary among etiological factors.

X. LOCAL CONDITIONS AFFECTING BLOOD SUPPLY

Of the local conditions affecting blood-supply, much has been written regarding the so-called end-organ produced through the arrangement of the arteriole-venule-capillary mechanism by which the epithelium of the marginal and cemental gingivae are nourished. It has been assumed that the return of venous blood, from the finger-like processes that carry these blood vessels into the epithelium of the external gingival surface, was obstructed or that the current was too sluggish to bring about prompt return of this blood to the larger veins. Abundant anastomosis of vessels in the body of the gingiva protects against complete obstruction. Moreover,

the first evidence of pathology in the marginal and cemental gingiva is to be found at or near the crevicular surface of the marginal gingiva, or at the base of the gingival crevice. In these locations, there are no deep finger-like processes carrying blood-vessels, hence nothing approximating end-organ conditions.

A sluggishness of circulation may be brought about in many ways and by a variety of agencies. It seems obvious, however, that of the two factors, the one producing the slowing-up of the circulation is primary, while the circulatory disturbance itself is secondary. Having become established, it, like the bacterial invasion, is important in its influence on the lesion, and it is frequently difficult to restore the circulation to its original freedom. In such cases treatment is disappointing, and must be repeated at frequent intervals.

Among the most prominent causes of circulatory insufficiency are hypo-function and insufficient tooth-brush massage. Of these the hypo-function has been mentioned.

Tooth-brush massage is essentially an artificial measure. It is a means of accomplishing certain ends which should be attained through the proper exercise of occlusal function. Inasmuch as very few individuals in our so-called civilization use their teeth vigorously enough, or partake of sufficiently fibrous foods, the use of the tooth brush is a universal necessity. Properly used it stimulates the circulation in the gingiva to a remarkable extent. Unfortunately, this salutary effect does not carry very far into the pericementum and attains to an indeterminate depth into the bone. Whatever its deep effect, however, failure to use the brush properly lowers perceptibly the barriers against bacteria.

Decomposition products from adherent food particles, and other poisons including products absorbed into the blood from the intestines, have an undoubted effect in lowering the local defenses against bacteria. Their direct effect on the cells of the part, even in cases of circulatory slowing-up, are often vague. Mercury seems to have the most definite effect on the pericementum of any of the poisons. Even this agent seems rather to aggravate previously existing lesions, and does not apparently inaugurate disease in normal periodontal tissue. Tobacco smoke is irritating, but seems to produce no disease in the previously clean healthy mouth. In the neglected mouth it aggravates a slight chronic gingivitis into a semi-acute type; and in such a mouth it is particularly apt to pave the way for attack by the fuso-spirillary group of organisms.

XI. ANATOMICAL DEFICIENCIES

Anatomical deficiencies, especially frail alveolar structures, may play a prominent part in the etiologic complex, but always as secondary factors. An example often encountered is the type of case having slender teeth with long sharp cusps and correspondingly deep sulci, the alveolar structure being thin and delicate. Unless the points of the cusps are considerably and evenly abraded during the period of youth, when the pericementa are fairly thick and capable of withstanding occlusal shocks, the frail buccal and lingual plates of the alveolar process become more or less absorbed under the continued strong bucco-lingual pressure. This condition then becomes the entering wedge for infection and tissue solution.