III. THE MECHANISM OF PERIODONTAL BREAKDOWN

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Relations between Periodontal Disease and Systemic Disease

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The effect of systemic factors has been implicated primarily in the form of periodontal disease designated as "periodontosis." Periodontosis presents somewhat of an enigma because it is often defined as a form of periodontal disease due to systemic factors, but the systemic factors are often not recognizable, and there is no consistent relationship between a specific disease and the development of periodontosis.

What constitutes periodontosis is not clearly defined, and there is much disagreement between investigators and clinicians[1, 2] as to what constitutes the disease. This makes it difficult to know how to attack the problem of etiology.

A multitude of systemic factors and some specific diseases have been suggested in the etiology of both periodontitis and periodontosis. These factors have been indicted because of clinical observation in human subjects but especially as the result of studies on laboratory animals.

Some of the more frequently suggested systemic factors are nutritional[3] ones. Both specific nutritional factors and general nutrition have been suggested as significant in the etiology of periodontal disease. Protein deficiency in laboratory animals has been shown by Goldman[4] to produce periodontal changes, and others have suggested from clinical observation that protein deprivation is significant in the progress of periodontal disease. However, no one has demonstrated that periodontal disease has greater prevalence among individuals whose diets are low in protein.

If we consider the nature of periodontal disease—as a chronic destructive process of the periodontium, which progresses at various rates, depending on the individual's ability to repair the damaged tissue—one would expect the progress to be more rapid in any condition which influences repair. Coller[5] and others have shown that protein deficiency delays repair; therefore, in the individual unable to carry out normal reparative procedures because of protein deficiency, periodontal disease will progress more rapidly than in the individual who can repair tissue normally. However, it has never

been demonstrated that periodontal disease can be initiated by means of dietary deficiency.

When specific nutritional factors are mentioned in relation to periodontal disease, vitamin C is the one that is usually given first consideration. The reason for this is primarily historical, but also because vitamin C deficiency is a purpuric disease and gingival disease is also characterized by bleeding. Vitamin C deficiency is not an initiating factor in periodontal disease, but, because vitamin C is necessary for the normal metabolism of endothelium and connective tissue, it may influence the course of periodontal disease. It will enhance gingival bleeding due to local factors because of altered capillary permeability. It will also prevent repair of the periodontium in the face of a chronic destructive periodontal disease, so that the disease may progress much more rapidly in the individual with vitamin C deficiency. But there is no evidence to show that gingival tissues are particularly susceptible to vitamin C deficiency or that vitamin C deficiency will initiate periodontal disease.

Glickman[6, 7] studied the effect of vitamin C deficiency in periodontal disease. He states that it is not an initiating factor but that if some local factor is superimposed on deficiency of vitamin C, then it is of some importance in the progress of destruction.

It has also been shown that gingival tissues are not particularly susceptible to vitamin C deficiency and that periodontal disease is not a characteristic feature of scurvy. This was well demonstrated by Crandon and his group[8] in subjects who were placed on a vitamin C–deficient diet. He first determined that they were in a good state of general and periodontal health, and put their mouths in a state of complete dental health. He then proceeded to produce scurvy in these individuals and found that, even after they had shown frank manifestations of scurvy for a period of 30 days or more, none of them demonstrated any gingival bleeding. If gingival disease or periodontal disease is a manifestation of scurvy, it must be a late manifestation.

Endocrine factors have been given considerable attention in relation to periodontal disease, particularly hypoinsulinism or diabetes mellitus. Much has been written about the effect of diabetes on periodontal disease, but most of the work has been based on clinical observation. Glickman[9] has demonstrated that diabetes per se is not responsible for periodontal disease but that, if local factors are present, periodontal disease will develop.

Here I would digress for a moment from the effect of systemic disease on periodontal disease and mention the effect of periodontal disease on systemic disease. Because of periodontal disease, it may be difficult, if not impossible, to control the level of blood sugar in the diabetic. The individual may go out of control because of exacerbations of his periodontal disease, and it may be almost impossible to maintain diabetic control in the face of the chronic destructive process in the periodontium, with the associated infection.

Another relation between endocrine activity and periodontal disease is demonstrated in the effect of the gonadal hormones upon the development of pregnancy gingivitis. Increased hormonal stimulation during pregnancy will increase vascular proliferation and enhance gingival bleeding. However, pregnancy gingivitis occurs only in the presence of pre-existing gingival disease and is eliminated with the removal of the local factors, even though the hormonal factors are still active. The hormonal alterations thus are not initiating factors but contributory ones.

Leukemia involves the gingival tissue, with the production of gingival enlargement and bleeding. This is usually secondary to pre-existing gingival inflammation. Invasion of the neoplastic cells into the tissues lowers their vitality, and superimposition of fusospirochetal organisms produces ulceromembranous gingivitis and a rapidly progressing periodontitis. The gingival changes in leukemia are chiefly secondary to the pre-existing gingival disease.

Another group of systemic diseases that should be discussed are the collagen diseases. Any disease characterized by collagen degeneration could initiate or influence the progress of periodontal disease. This is most evident in scleroderma, in which there is radiographic evidence of widening of the periodontal space and histologic evidence of degeneration of the periodontal tissues. However, the effects of these changes in the production of progressive destructive periodontal lesions are minimal in the absence of local destructive factors.

Evidence of the effects of other collagen diseases, such as rheumatic fever, periarteritis nodosa, and dermatomyositis, upon the periodontal tissues has not been demonstrated. Some of the rarer syndromes with alterations in ectodermal or mesodermal elements may predispose the individual to periodontal disease, or they may markedly accelerate the progress of the disease.

Gingival disease may occur as a manifestation of a dermatologic disease such as lichen planus. In gingival lichen planus the disease is usually erosive and provides a suitable environment for an exaggerated effect by local factors.

Other forms of systemic disease could be discussed, but these should suffice to point out that in most instances systemic disease is a contributory factor rather than an etiologic factor in periodontal disease. In those instances in which it initiates periodontal disease the course of the periodontal disease is determined by the local factors superimposed.

These observations would indicate that our research in periodontal disease should be directed toward the reaction of the periodontal tissues to injury, whether due to local or systemic insult. It seems that the answers will best be found in the study of the histochemical, biologic, and morphologic characteristics of the periodontal tissues.

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