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The Pathogenesis of Periodontal Disease and Treatment Implications

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Graduate School

THE PATHOGENESIS OF PERIODONTAL DISEASE AND TREATMENT IMPLICATIONS

by

Richard C. Oliver

A Thesis in Partial Fulfillment

of the Requirements for the Degree

Master of Science in the Field of Periodontology

August 1962

61786

I certify that I have read this thesis and that in my opinion it is fully adequate, in scope and quality, as a thesis for the degree Master of Science.

Charles T. Smith, Dean, School of Dentistry

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I. INTRODUCTION

One way in which we learn is by experience and reflection thereon. The perceptive clinician dealing with periodontal disease must be disturbed by the fact that the same local etiologic factors are associated with varying degrees of periodontal destruction in different individuals. Similarly, the same therapy and control of the local etiologic factors produces varying degrees of success in curing periodontal disease for these patients. The observation of these patients when they are on a maintenance basis can improve our understanding of the basic nature of periodontal disease.

There is a wide variation in the therapy and home care procedures required to maintain the periodontal health of these patients. Many need extensive oral physiotherapy in the form of interdental tip massage, the use of a wide variety of auxiliary cleansing aids, vigorous brushing, and prophylaxis on a two or three-month basis. Despite these efforts, some patients develop new pockets and continue to lose alveolar bone. Their tissues vary from one recall visit to the next, and are frequently referred to as "weak," or "of poor tone and quality," or perhaps "improved." They seldom seem to achieve the firm, pink, well-stippled soft tissue that characterizes the normal attached gingivae. Yet, the gingival tissues of other recall patients rapidly return to this tissue condition following therapy, in spite of less than ideal oral hygiene and very little oral physiotherapy. These patients do remarkably well on six-month (or longer) recalls, and cause us to question whether the extent of their original therapy (if it included extensive surgery) was really necessary.

Scaling, curettage, and minimal toothbrush instructions might have achieved the same result. In fact, most of us have patients who for one reason or another did not have the benefits of periodontal surgery (although we believed it was indicated) and have kept their teeth with no apparent further periodontal destruction. These experiences and the reflections thereon pose the problem: what is the difference in individuals that accounts for the varying responses to local etiologic factors as well as to therapy?

II. REVIEW OF THE LITERATURE

CURRENT CONCEPTS OF THE ETIOLOGY OF PERIODONTAL DISEASE

Discussions of the etiology of periodontal disease in most current textbooks differentiate between "systemic" and "local" factors. Local factors include calculus and other deposits on teeth, food impaction, other irritants, occlusal trauma, architectural deformities of the soft tissues, etc. Systemic factors generally recognized are faulty nutrition, debilitating diseases, blood dyscrasias, endocrine dysfunctions, psychogenic factors, etc.

There are varying concepts of the importance of the systemic factor in the etiology of periodontal disease. Some seem to discount it completely. Others, such as Fish, describe the etiology on a morphological basis. He has stated that "once the col is detached from the teeth, it becomes acutely inflamed and rapidly breaks down, establishing a deep interdental ulcer that has little chance of healing without surgery."[1] Miller refers to the "Etiologic Pile" which describes various systemic and local factors piled one on top of the other. These lower the tissue resistance and result in the breakdown of the periodontium.[2] Glickman describes the basic interrelation of local and systemic factors and represents the causation of periodontal disease as: (1) pathological local factors in the presence of physiologic systemic influences; (2) pathological systemic factors in the presence of physiologic local influences; or (3) pathological systemic and local factors.[3] Goldman has formulated the relationship as follows: local environmental factors x

frequency of injury x duration of disease process modified and governed by the resistance and reparative factors of the individual = periodontal manifestation.[4]

Current concepts point to bacteria as the most important etiologic factor.[5,6] Some clinicians believe that occlusal trauma plays the major role in periodontal destruction. There is little unanimity, but where systemic factors are acknowledged, one must be able to diagnose specific diseases. When this cannot be done, the systemic factors are grouped together and described by terms such as "resistance," "repair capacity," and "systemic quotient," whose only virtue is in their vagueness. And being vague, they sufficiently frustrate the clinician that he readily dismisses the systemic factor as intangible and immeasurable, and concentrates his attention on looking for and treating the local factors. If he is sufficiently disturbed by the appearance of the patient or his oral tissues, or his medical history, he may request a medical examination and perhaps even certain laboratory tests. Almost invariably the results are "non-contributory."

There is one other consistent concept in discussions of the etiology and pathogenesis of periodontal disease. It is the belief that as long as the local etiologic factors persist, the disease will continue to progress. The origin of this concept may have been clinical observation, or it may have resulted from attempts at describing etiology and prognosis to students or patients. Treatment seems imperative if periodontal disease is pictured as one in which the infection spreads and destruction progresses. Kramer, in his brochure for periodontal patients, states: "Without treatment there is a continuation of the destruction of the supporting tissues to a point where the teeth loosen and fall out."[7] Other authors give similar reasons for periodontal therapy.[8]

PROBLEMS IN APPLYING THESE CONCEPTS

Obviously, the classical discussions of etiology and pathogenesis leave unanswered the problem of the varying responses of different patients to the same etiologic factors and the same periodontal therapy. What can be learned from examining the periodontal tissues of the patient? We are able to assess his past periodontal disease by observation of the tissue destruction that has occurred. These observations result from the correlation of tissue architecture, pocket measurements, and radiographic interpretation of alveolar bone loss. We are also able to assess the present intensity of the periodontal disease by noting the tooth mobility, the degree and nature of the inflammatory process, the presence of purulent exudate, or hemorrhage, and by correlating these signs with the patient's symptoms. We may attempt to evaluate the future potential for periodontal disease by projecting our ability to eliminate the etiologic factors that we recognize, correct the architectural deformities and pockets, and teach and motivate the patient to practice adequate maintenance procedures.

But the question remains: Are these observations, etiologic concepts, and therapeutic procedures sufficient to justify our prognosis? If so, how do we account for the variability in our results? Cohen recently stated that "it has not yet been proven that periodontal therapy even alters the outcome of periodontal disease."[9] Glickman writes that "at the present state of our knowledge, however, it should be recognized that comparable local measures do not attain the same result in all individuals, and that one of the reasons for the variability in the outcome of treatment is the difference in the systemic influences upon the cellular changes associated with healing."[10] Perhaps the present concepts of periodontal disease are too limited.

III. PATHOGENESIS OF PERIODONTAL DISEASE

UNDERSTANDING THE NATURE OF DISEASE

A discussion of the pathogenesis of periodontal disease must properly begin with a discussion of disease in general. Disease has many definitions. One simple definition analyzes the word and its parts, i.e., "dis - ease," meaning separation from ease. Engel has defined disease as "failures or disturbances in the growth, development, functions, and adjustments of the organism as a whole or any of its systems."[11] This definition does not limit us to any one parameter of disease, such as searching out the biochemical defect, or the causative organism, or the "stressor," which the cellular concept of disease and other narrower concepts have done. This definition tries to get away from the implicit assumption that disease is a thing in itself, unrelated to the patient, the patient's personality, bodily constitution, and mode of life. We as physicians and dentists have preferred to think of disease as something external, something that afflicts the patient. If we can see the cause as something we can attack and destroy, we can treat the disease. Our language has furthered this concept. We talk about "catching cold," "treating disease," "infection," etc. We have attempted to draw direct cause-effect relationships between specific etiologic factors and specific diseases. We have failed to think of disturbances at all levels: biochemical, cellular, organic, psychological, interpersonal, and social.

Many of the limitations of our present concept of disease can be traced back to the dominance of the germ theory of disease in medical

thinking. Koch and Pasteur were the founders of this theory that equated the microbe with the causation of disease. History offers many examples substantiating the theory. Epidemics and plagues are evidence that a microbial agent may strike down the weak and the healthy alike when newly introduced into a susceptible population. But the theory does not account for the fact that large percentages of healthy and normal individuals harbor potentially pathogenic microbes without suffering any symptoms or lesions. Examples include the tubercle bacillus, streptococci, and many viruses. These microbes are only associated with disease where there is a loss of "resistance" such as occurs in uncontrolled diabetes, overwork, improper nutrition, overindulgence, or even in an unhappy love affair. Neither does the germ theory explain the failure of our modern chemotherapeutic agents to reduce the amount of infectious disease on an epidemiologic basis.

What other evidence is there for assuming that a disturbance in the host is often necessary for the disease response? The most common evidence is that healthy cells tend to ignore the presence of virulent bacteria and their toxins. In addition, many microbial enzymes are inducible, e.g., altered host cell substrate releases hyaluronic acid, which in turn induces the production of hyaluronidase by the microbe. Further evidence is the association of disturbances of the host with the onset of specific diseases. Necrotizing ulcerative gingivitis and herpes are two examples. The proper role of the microbe, then, is as the agent of infection. The disease response is instigated by the unrelated disturbance of the host.

What are the agencies responsible for natural resistance to infection, and what factors interfere with the operation of these agencies? There are those which restrict the infective agents, such as the blanket

of fluids covering all body surfaces and the barriers formed by the epithelium and connective tissue. Stress, which produces a generalized inflammation, can depolymerize the ground substance and result in the destruction of the collagen fibers. There are also the agencies that clear infective agents. These include the cells that are responsible for phagocytosis, as well as specific antibody reactions and tissue substances.

It has been shown that animal tissues normally contain substances that possess anti-microbial activity. Properdin and lysozyme are two such substances. For example, in animals exposed to irradiation, properdin disappears, the bacteria normally present in the G.I. tract multiply, and the animal becomes susceptible. Lysozyme is able to partially hydrolyze the cell wall of many bacteria such as staphlococcus, streptococcus, and pneumococcus. It may be that these are considered non-pathogens because of lysozyme.[12] It is also obvious that the nutritional state of the individual determines susceptibility and resistance. As previously mentioned, staphlococcus and streptococcus infections are frequent in the uncontrolled diabetic, but resistance returns to normal when insulin is given. Therefore, susceptibility can be the temporary expression of a physiologic disturbance.

Dubos has made a number of important observations on natural resistance.[13] He points out that the tissues of man contain everything required for the life of most microbes. It has also been demonstrated in germ-free animal experiments that simple, normal, non-pathogens cause disease. Therefore, the fact that a population survives and flourishes in a given environment implies that its members have a high degree of natural resistance to the microbes normally in that environment. This

natural resistance stems in part from evolutionary selection of the strains best endowed with mechanisms for withstanding infection, and also from the development of adaptive reactions in response to early exposure to the microbes. There exists a biologic equilibrium between man and the microbes in his environment. This equilibrium is only stable, and the mechanisms of resistance are only effective, within the narrow range of conditions constituting the normal environment. Common microbes, ordinarily harmless, produce disease when physiologic circumstances are sufficiently disturbed. They then cause many ill-defined ailments, major and minor, that constitute a large part of the diseases of everyday life. Therefore, the microbe is the prerequisite but not the determinant of these diseases.

Disease is thus considered a natural response of the patient, and the problem is to identify the state of disease regardless of its severity, etiology, or the particular system involved. We must be able to identify the potential for breakdown in the apparently healthy individual. Engel states that, "The presence of a complaint must be regarded as presumptive evidence of disease."[14] It indicates a disturbance in the physiological equilibrium. Conventionally, we as physicians and dentists have tended to dismiss such complaints when we were unable to find something wrong objectively, i.e., pathosis. We have also equated the correction of a physiologic or anatomic abnormality with cure regardless of the patient's subjective experience. Both responses indicate a limited concept of the etiology of disease.

ETIOLOGIC FACTORS OF DISEASE

What are the etiologic factors in the genesis of disease? Engel has divided them into: (1) those which determine the capacity of the

organism to grow, survive, and adapt; and (2) those which strain the current capacities of the organism.[15] The former include genic factors and developmental factors. At any point in time, what constitutes stress to an individual will be determined to a large degree by the nature of his past experience plus his genic endowment. The second category includes: (1) factors which injure by virtue of physical or chemical properties (both external, e.g., poisons, heat, etc.; and internal, e.g., excess insulin and gastric juices); (2) factors which lead to injury when insufficient or unavailable, such as oxygen, vitamins, or hormones; (3) microorganisms and parasites; and (4) psychologic stress. Psychologic stress has been defined as:

> All processes whether originating in the external environment or within the person, which impose a demand upon the organism, the resolution or handling of which necessitates activity of the mental apparatus before any other system is involved or activated.[16]

The action of a microorganism does not satisfy these criteria, but the idea of an infection does, just as the idea of losing one's teeth constitutes a psychologic stress.

Some generalizations may be made about these etiologic factors. They may operate through the impact of something added to the system as well as through a deficiency. They may originate within the organism as well as in the environment. Factors which have etiologic significance with respect to a disease process at one time may not at another time. We never deal with a single factor in disease, although one factor may be more important than others.

CELLULAR CHANGES IN DISEASE

Ultimately, the health of any tissue or organ, or of the body, depends upon the normal function of cells. The normal function of body

cells depends upon the alternate rhythmic activity and rest of the cell membrane through which all nourishment enters, and from which all cell products and waste matter emerge. Both excessive prolongation and restriction of the active state of the cell membrane affect the integrity of the cell. The inflammatory response to an irritant prolongs the active state of the cell membrane. Stress also prolongs the active state of the cell membrane, and results in cellular alterations identical to the inflammatory response.[17] Then the cell contents "leak" through the hyperactive and excessively permeable membrane. This results in a loss of cell content, a diminished cell function, and cell destruction. The cell material increases the osmotic pressure of the substrate resulting in prolongation of the active permeable state in neighboring cells. The cell injury is also associated with depolymerization of the ground substance, diminished tonus, increased permeability of the connective tissue, and enhanced diffusion of tissue metabolites. The metabolites in turn provide increased nourishment for resident microbial flora, and in some instances trigger the elaboration of adaptive microbial enzymes, e.g., hyaluronidase. Thus the ultimate effect of either an irritant or stress is to increase the concentration and activity of the microbial flora.

THE NATURE OF THE ADAPTIVE REACTIONS

The response of the body cells and fluids to the influence of corticoids, histamine, and active microbial products, particularly endotoxins, involves a number of adaptive reactions. One is localization of tissue breakdown by the inflammatory response. A second is the modulation

of connective tissue cells, such as fibroblasts, to defense cells, such as macrophages. Proliferation of the epithelium is the adaptive response of the lining tissues. Another adaptive reaction is the release of specific antibody and discharge of the available phagocytes into the area. The exact nature of the adaptive reactions depends upon the ability of the local tissues to limit the effects of the stressor. There is an interreaction between this local adaptation syndrome and the general adaptation syndrome. The hormones produced as part of the general response to stress determine the tissue or cellular response at the site of injury.[18] If anti-inflammatory corticoids (glucocorticoids) predominate, the tissue reaction is one of spreading inflammation, degeneration, and cell death. If pro-inflammatory corticoids (mineralocorticoids) predominate, stimulation, enlargement, and multiplication of cells occur at the local level. Anti-inflammatory hormones predominate in a patient under stress. Adaptation which depends on serologic immunity and/or inflammatory barricades is broken down under stress. The result is cellular fatigue and spreading inflammation. Therefore, a generalized, diffuse, inflammatory response with the loss of fibrous tissue integrity would characterize the tissue response of a patient under stress. Conversely, localized inflammation and hyperplasia would characterize the tissue response of the patient in whom stress has not been a predominant factor.

In summary, disease must be considered as part of the patient rather than something apart or external that afflicts the patient. Disease is the response of the patient to a physiologically unacceptable condition. Frequently the condition is physiologically unacceptable because of disturbances within the patient due to hereditary or developmental impairments, or his way of life. Cellular changes take place which lower the

natural resistance of the patient. As a result, bacteria and other normal conditions become pathogenic. The disease response is the effort of the body to eliminate or restrict these aggravating conditions. Awareness (due to pain or malaise which tends to restrict activity), expulsive forces (such as vomiting, diarrhea, and coughing), elevation of body temperature (which increases metabolic activity and often in itself reduces the activity of the pathogens), the release of the available white blood cells into the blood stream, and inflammation on the local level at the site of the aggravating condition--all are elements of the disease response. Diagnosis must recognize that the observable changes may represent an inadequate response and a progressive process, or a response which arrests the process, or a response which eliminates the aggravating condition. The treatment of disease must be directed at the total patient, and must have as its objectives the recognition and correction of the disturbances within the patient as well as the assistance of his response by control of the aggravating conditions.

CORRELATION OF PERIODONTAL DISEASE AND DISEASE IN GENERAL

What correlations can be made between disease in general and periodontal disease? The work of Moulton and others has shown that emotional factors (psychologic stress) are important etiologic factors in both acute periodontal involvements, such as necrotizing ulcerative gingivitis, and in chronic periodontitis. They report that "the gums simply did not respond to local treatment, and recurrence of the disease was rapid."[19] Schwartz has concluded from his studies of temporomandibular joint problems that the majority of these result from nocturnal bruxism, which is usually due to emotional tension.[20] Thaller, using the Cornell

Medical Index, has also shown a correlation between bruxism and anxiety.[21] Bruxism and the resultant occlusal traumatism are etiologic factors in many patients with periodontal disease. Alexander has emphasized the importance of emotional factors in chronic disease.[22] He has carefully pointed out that emotional factors alone do not account for the complete etiology of chronic disease, but must be considered with specific organ vulnerability (analagous to Engel's genic factors and developmental factors). Hileman has discussed specific periodontal patients in which emotional factors were important in the etiology and treatment.[23] Schluger, in his study of necrotizing ulcerative gingivitis, concluded that the microorganisms were not the cause of the disease, but rather the opportunistic parasite when the resistance was lowered due to other factors such as inadequate diet, insufficient rest, and psychological stress.[24] There have been repeated correlations of stress with periodontal disturbances in experimental animals by Ratcliff,[25] Stahl,[26] and others.

Manhold in his text on psychosomatic dentistry has stated that in dentistry, as in medicine, one-third of all dental disease is due to psychological causes, and still another third of all patients have emotional factors that complicate their dental disease.[27] In his studies, he has been able to relate periodontal disease to personality type. Periodontal pathology is greater in patients with neurotic tendencies, as well as in patients with tendencies toward introversion. He also found that in patients with a non-authoritarian personality, periodontal disease could be mediated by neglect. Miller has listed a number of mechanisms by which psychosomatic factors can initiate periodontal disease. These include a reduction of local nutrition through vascular changes, neglect, excessive clenching or grinding, and taste perversions and alterations of diet due to limitation of gastro-intestinal function.28

The dental literature is voluminous with the correlations between nutritional deficiencies and periodontal disturbances. A new textbook devotes an entire chapter to nutrition and periodontal disease.[29] Since the earliest classical signs of scurvy, such as "bleeding gums," "loose teeth," and "foul odor," were recognized, there has been an unquestioned relationship between Vitamin C deficiency and periodontal disease. Cheraskin has compared Vitamin C tissue levels with tooth mobility, and the relationship of blood glucose to gingival findings and other oral symptoms.[30-33] Vitamin A and estrogen levels both influence the keratinization of the gingival epithelium, and in turn the integrity of the protective barrier. Vitamin D, protein deficiency, and lack of estrogen may all contribute to osteoporosis.[34] It is common knowledge among periodontists that the alcoholic patient presents a difficult, if not an impossible, treatment problem. This may be due to nutritional deficiencies usually associated with alcoholism, the actual effect of the alcohol on the cells, or psychological stress.

After reviewing this information, the inescapable conclusion must be that periodontal disease has the same characteristics and complex etiology as disease in general. Systemic variables seem to be not only important contributing factors, but indeed the necessary condition for periodontal breakdown.

BROADENED CONCEPT OF THE PATHOGENESIS OF PERIODONTAL DISEASE

Most current concepts of the pathogenesis of periodontal disease assign bacteria the primary role.[6,35] The bacteria are either affixed to the tooth or calculus as plaque, or incorporated within the structure

of the calculus. The problem with this concept arises with the realization that all people with plaque and calculus do not have periodontal disease. Every clinician has seen many patients with excessive calculus and plaque with no evidence of destruction of the supporting tissues. Conversely, they have also seen patients with little, if any, calculus or plaque, but with marked alveolar bone loss. The same observations can be made about occlusal discrepancies. If one considers dental care statistics and the entire population, the remarkable fact is that so many people retain as many teeth as they do despite the tremendous accumulation of calculus and bacteria in their mouths and the lack of adequate dental care.

The proper role of bacteria, calculus, and other local factors is that of aggravating conditions. A disturbance within the host must precede periodontal destruction. The following observations seem to fit this concept. The bacteria present in periodontal disease are endemic to the oral cavity, but do not always result in disease. Bacterial enzymes, such as hyaluronidase, associated with the breakdown of the periodontal tissues, are inducible by altered tissue substrate. Mandel has stated that salts resulting from inflammation, or altered tissue substrate, combine with the bacterial mucoprotein matrix to produce calculus, and that it seems highly possible that the mineralized subgingival deposit is the result, not the cause, of periodontal disease.[36] It seems probable that with a disturbance of the host, the magnitude of the aggravating conditions dictates the degree of periodontal destruction that occurs. This is consistent with clinical observation.

The pathogenesis of periodontal disease may now be described as follows. Each individual patient has physiologic boundaries which are

the result of his genic and developmental background as well as of his current way of life. Within these boundaries the organism is able to adapt to the multiplicity of local factors without the loss of supporting tissue. When these boundaries are exceeded, cellular alterations take place in the periodontal tissues which increase their susceptibility to the local factors, such as calculus, occlusal trauma, microbial flora, etc. This results in destruction of the periodontal tissues. The character and degree of the destruction will depend on the response of the patient, as well as on the character and magnitude of the local factors. There is one other important dimension in this concept of the pathogenesis of periodontal disease. That is time. The physiologic boundaries vary at different times. In other words, the same local factors may result in breakdown of the periodontal tissues at one time but not at another time. The calculus in a patient's mouth may have contributed to periodontal destruction several years ago, but may not be destroying tissue at present. The reverse is equally true.

Such a concept neither minimizes the importance of the local factors nor accepts the inability to diagnose a specific systemic problem as an indication that the "patient" is unimportant in the pathogenesis of periodontal disease. The concept recognizes that successful therapy will not always result from the control of the local etiologic factors alone.

But the problem remains. How can we evaluate whether a patient is within his own physiologic boundaries? Are there laboratory tests available that will give us the answer? What is the best way of determining the disease state of the patient at present and thereby understanding his periodontal problem?

III. CLINICAL PARAMETERS OF DISEASE

THE VALUE OF LABORATORY TESTS

This has been called the machine age of medicine. For as disease came to be considered only as a disorder of organs and cells, medicine more and more defined specific ailments and contented itself with detailed laboratory investigations. To what degree are we able to depend on the laboratory results?

In the area of nutrition, the only deficiency levels that are known are those at which frank disease occurs. For example, the Vitamin C level is known at which scurvy will develop. But the optimal level, the subclinical deficiency level, and the variation in individual requirement remain uncertain. This is equally true of other vitamins. Cheraskin has attempted to compare plasma ascorbic acid levels with intradermal ascorbic acid test times and with lingual ascorbic acid test times. The correlations are all under 50 per cent.[37] Which one can we use or depend upon? One seems to measure dietary intake while the others measure tissue levels. Tests are also available to determine protein utilization by the body. These, too, are valuable only where gross deviations exist.

In the area of metabolic disturbances, glucose may be used as an example. The standards for the most sophisticated laboratory study were set by Mosenthal and Barry after they had studied the glucose tolerance curve of 50 ambulatory hospital workers.[38] Cheraskin has found varying results, and if glucose tolerance testing, fasting blood sugar, and urinary sugar levels are compared, it becomes evident that only frank diabetes can be detected by these tests.

The present laboratory emphasis is on the attempts to measure stress. Two laboratory determinations being utilized are blood cholesterol levels and 17-hydroxycorticosteroid levels in both saliva and blood. While they may be indicative of stress, the values vary markedly (as stress does) with time, and no physiologic norm exists. Again, they are useful in determining extreme variations at a particular time.

Systemic studies have been done attempting to establish a correlation between periodontal disease and certain laboratory analyses. O'Leary, Shannon, and Prigmore have recently published a paper on a study of sodium, potassium, phosphate, chloride, calcium, urea nitrogen, glucose, glucose tolerance tests, and 17-hydroxycorticosteroid levels compared with periodontal involvement.[39] They were able to find a statistically significant difference between the experimental and control subjects in only the 17-hydroxycorticosteroid levels, and this was slight. However, if periodontal destruction occurs only when the patient has exceeded his physiologic adaptability, then periodontal involvement may represent destruction that occurred weeks, months, or years before, or at various times but not necessarily at the time the tests were made. Little difference should have been expected between the experimental and control groups.

There are numerous reports in the literature correlating various salivary enzymes with periodontal disease.[40-44] Cholinesterase and lysozyme are two of these enzymes. Lysozyme is a bacteriostatic enzyme found in the saliva, as well as in the tissues, that has been correlated with both periodontal disease and stress. We recently attempted to repeat these correlations. We used the periodontal index described by Sandler and Stahl.[45] Two hundred patients collected saliva upon arising

in the morning. This was analyzed for lysozyme, and correlated with the patient's periodontal disease rate (PDR). The criteria used in this index for periodontal disease on an individual tooth basis were: (1) gingival necrosis, or hypertrophy, or inflammation encircling the tooth, or a purulent exudate from the gingival crevice; (2) a gingival crevice depth of 3 mm. or more; (3) tooth mobility greater than 1 mm. in any direction; (4) roentgenographic evidence of alveolar bone extending more than 3 mm. apically from the cemento-enamel junction.

We found no statistically significant correlation. We then thought that, since these included both recall and new periodontal patients, and since destruction may not take place except when the patient is physiologically out-of-bounds, we should not expect a correlation. Indeed, the fact that the recall cases were being maintained indicated that periodontal breakdown was not taking place. We then looked for patients with "acute" necrotizing ulcerative gingivitis in which we felt reasonably sure that tissue destruction was occurring at that time. We collected several samples from each of four patients and found no consistency in lysozyme levels. We checked this by taking a series of saliva samples at different intervals and different times of the day from twenty routine patients. We found no consistency whatsoever in salivary lysozyme levels from the same patient taken at various times, and were unable to relate them to periodontal involvement. The levels varied from hour to hour, as well as from day to day.

Glucose levels (both fasting blood glucose and glucose tolerance tests) have been studied, and a relationship has been shown with oral symptoms.[31,33] This might be expected. The relationship was more clearly defined than with gingival findings. In fact, oral symptoms (gingival

tenderness, xerostomia, stomatopyrosis) may be valuable in determining altered glucose metabolism. However, a recent study emphasizes the complexity of diagnosis from symptoms alone.[46] Ascorbic acid levels, regardless of measurement methods, fluctuate depending on bodily needs, psychological stress, intake, etc. Multiple determinations are necessary to be of any value whatsoever.

In his paper, "The Arithmetic of Disease," Cheraskin emphasized the necessity of having both an altered systemic substrate and exciting oral factors to have manifestations of oral disease.[47] All systemic conditions which might alter the systemic substrate are listed and given a weighted value. All local factors are similarly listed. These weighted values are added in both categories, and the product of the systemic substrate value times the exciting oral factors value is equal to the observable evidence of disease. This concept and the equation underscore the purpose of therapy--namely, to reduce all of these values to as close to zero as possible. But several problems exist with the concept. One is that it assumes that we are able to diagnose and define even marginal systemic imbalances that exist at the moment with presently available procedures. Another, which the author points out, is that information is still lacking about the interaction of various factors, i.e., is the cause of a subclinical ascorbic acid deficiency inadequate intake, stress, or smoking? Also, the time dimension is not emphasized, i.e., are all of the observable evidences of disease which he uses, such as alveolar bone loss, related to systemic and local variations that exist at present, or are they related to those that were present at some previous time?

What conclusions can be drawn about the value of present laboratory tests in establishing the clinical parameters of disease for an individual? They all have one or more of four serious shortcomings. First, they may only define frank disease on the basis of presently accepted normal ranges, and do not indicate incipient or subclinical disease. Second, the values are only meaningful at the time the test is done and may differ markedly from the values obtained a day before, a week later, at a different hour, or for an extended period of time. This is understandable when one considers the many etiologic factors that may upset the dynamic physiologic equilibrium. Therefore, multiple determinations must be done to be of value. Third, there is in most of the present tests a question of reliability of the test as to what it actually measures. For example, does an altered glucose tolerance pattern result from metabolic imbalances or psychological stress? The same might be asked of ascorbic acid values. Frequently, the test techniques lend themselves to inaccurate and inconsistent results. Very few laboratories are able to do an accurate 17-hydroxycorticosteroid determination. Finally, there is the matter of individual difference when subjected to a group norm and the applicability of the test. A normal distribution puts a significant per cent of the population at either end of the curve. Therefore, normal for one patient may be a disease response for another.

These comments on laboratory tests are not meant to depreciate their value, but rather to define their usefulness. The tests help to diagnose frank disease, but do not delineate an individual patient's physiologic boundaries. The perceptive clinician must rely on additional information and observations to establish the parameters of disease for his patient.

THE DIAGNOSIS OF DISEASE

How may we evaluate disease in our patient? The most accurate way is by correlating facts gathered by listening to and observing the patient at the time of examination and at each subsequent visit to the office. The perceptive clinician consciously or unconsciously draws a mental picture of the patient that is perhaps the most valuable indication of the physiologic boundaries and disease parameters for that particular patient. Some of the information and observations which are helpful follow.

By Selye's definition, we are appraising whether our patient is under stress at present, and what the various stressors are.[18] Psychological stress is an important factor. Gross difficulties bothering a patient are usually discernible. But we should also attempt to determine his personality type. Manhold has pointed out that periodontal problems may arise due to either neurosis or neglect.[27] The neurotic is characterized as a person who is insecure, unhappy, ill at ease, and riddled with compulsions or feelings of persecution or guilt. He is never able to understand the world or the people in it, yet tries to be one of the group. He finds no peace, and feels as if tied in knots internally. He often complains about his previous dentist. He carefully weighs his words and acts and adjusts in any way possible. Therefore, a neurotic's tension must exhibit itself somatically. Often these patients enjoy being "sick," for it brings them attention.

There are two other personality types of interest: the authoritarian and the non-authoritarian. The authoritarian is one who submits his individuality to a greater power. In our society, the true authoritarian is the superconformist. He does not adjust to

changing situations and needs to be instructed. He is most secure when acting in a particular pattern which does not call for self-reliance but rather demands obedience to set instructions or conventions. He is characteristically more anxious, more rigid, and more dependent than the non-authoritarian. He tends to find his niche in society and is compulsive about maintaining his personal health. Authoritarian personality individuals are often good periodontal patients. However, the authoritarian may become neurotic if his way of life is markedly changed.

The non-authoritarian exhibits little rigidity of thought or behavior. He has no dependency and little hostility, and meets new situations without hesitation. He revels in responsibility, and his interests and aptitudes are of a professional or scholastic nature. His habits are erratic, and he forgets to eat, to brush his teeth, and to keep appointments. He takes little care of his mouth, and gives no one else the opportunity to do so. We have to appeal to his intellect to get any degree of cooperation in periodontal home care. His periodontal problems result from neglect. He is under very little psychological stress, and generally is in excellent health, although he pays little attention to his physical condition.

These are just a few of the personality classifications that are useful. To help define the personality type and assess the psychological stress of a patient without offending or arousing suspicion, a clinician may make observations, take a medical and dental history, and ask a few questions, such as the following: What things bother you the most? Do you consider yourself a sensitive person? How would you describe your personality? What are some of your likes and dislikes? What type of people do you like or dislike? Do you think of yourself as

a perfectionist? How would you feel about losing your teeth? While basic personality types seldom change, the ability to adjust (or adapt), and the modes of adjustment, do vary at different times. Frequently, the dentist's accurate appraisal of the periodontal condition and prognosis will elicit a response from the patient which is an important clue to his personality.

In a study of 138 patients with sixteen illnesses, including rheumatoid arthritis, peptic ulcer, diabetes, hypertension, hyperthyroidism, coronary occlusion, ulcerative colitis, migraine, asthma, neurodermatitis, dysmenorrhea, degenerative arthritis, glaucoma, and backache, Ring was able to match the disease with the patient on the first choice 66 per cent of the time, and almost 90 per cent of the time with two choices. This was done on the basis of personality alone. He was allowed to ascertain the vital statistics and ask five general personality questions. He was not allowed to observe the patient nor to have any knowledge of the patient's medical history. He classified the patients in three categories according to their reactions, and found certain diseases corresponded with certain categories.[48]

Habits are frequently important clues. Bruxism is a common habit, and usually represents a tension outlet during periods of stress. This can be suspected by noting mobility patterns, wear facets, and the location of periodontal pockets. Frequently, questioning the patient about grinding or clenching his teeth, or whether he wakes up with his teeth sore to biting pressure and with facial muscles that are tense and tender, will verify the suspicion. Patients with occlusal neuroses are well-known dental problems. Smoking is another habit which can reflect stress, and which can alter the character of the oral tissues. Cheraskin

has correlated reduced tissue Vitamin C levels with smoking.[49] In some instances, eating habits will indicate not only inadequate nutritional intake, but also patterns associated with personality types. Overeating can be a symptom of a problem, as can "having a cigarette and a cup of coffee" for breakfast. There are distinct correlations between food preferences and dependency or personality characteristics. As previously discussed, alcoholism is a major stress habit, as well as being a nutritional problem. Alcoholics can seldom be treated successfully for periodontal disease.

The medical history is indispensable in evaluating the parameters of disease. The expression, "when it rains it pours," applies to patients' medical problems and with good reason if non-specific stress and the general adaptation syndrome reduce the physiological boundaries of these individuals. It is not uncommon to find that the patient has more than one of the diseases of adaptation, such as ulcers, asthma, hay fever, other allergies, dermatitis, arthritis, colitis, hypertension, and perhaps diabetes, thyrotoxicosis, and other metabolic diseases. The menopause is accompanied by hormonal imbalance, and is often the source of severe psychologic stress. The most important emotional impact of the menopause for the patient stems not from the obvious, such as fear of pain and fear of change, but from the long-associated connotation of growing old. The fear of losing their teeth has tremendous impact on these patients. Some questions which will bring forth information of value in characterizing the type of patient and his present disease response are: How is your general health? Do you take any medicines? Do you consider yourself nervous?

The dental history will add to the picture of the patient in several ways. His dental habits, such as frequency of past visits to the dentist, the type and quality of his dental work, his oral hygiene habits,

his present concern about his teeth, his chief complaint and why he came to the office, are all important. Oral symptoms may help to define the present state of disease. A few potentially helpful questions are: Do your gums bleed on brushing? How frequently? Are your teeth loose? Are they sore? Are your gums tender? Have you noticed pus around your teeth? Often the onset of symptoms can be correlated with past periods of stress, and comments such as, "they used to bleed but haven't for a long time," attest to the time factor in periodontal disease.

Finally, the present physiologic condition of the patient can frequently be determined by correlating all of the aforementioned information with the clinical observation of the periodontal tissues. The color (such as a diffuse fiery red to magenta, as opposed to pale pink), the tone (glossy and sometimes edematous, as opposed to stippled and firm), hemorrhage to the slightest touch, the appearance of pus at the marginal gingiva with pressure on the periodontal soft tissues, the absence of sufficient local irritation to relate to the tissue changes--all are important clinical signs related to the patient's parameters of disease. The destruction of tissue as opposed to the proliferation of tissue would indicate a less favorable patient response. Most of these tissue signs are similar to those observed with the systemic administration of cortisone.[50]

In summary, the present response of disease or health by a patient can be assessed most accurately by correlation of the observable clinical signs with the present symptoms and the patient's way of life. If disease is due to the inadequacy of his reactions against the microbes, then the physiologic boundaries that determine the healthy response or the disease response can best be determined by observing the nature of these reactions, and understanding man's reactions to his way of life.

The correlation with observable clinical signs and symptoms is necessary because a way of life that is stressing to one patient can be stimulating to another. Although the individual pieces that make the picture are difficult to relate by themselves, the entire picture bears a common-sense relationship to disease that is obvious to the clinician who will look for it.

Several examples may illustrate the concept. A 34-year-old white female presented herself for periodontal examination and treatment. Vital statistics included: divorced, one teen-age daughter, works as a cocktail waitress in a Las Vegas hotel. Her chief complaint was "bleeding gums." Her dental history included infrequent dental care. Her medical history included a hysterectomy two years ago, the present use of tranquilizers, and asthma. Her way of life and habits included arising at noon, having a cup of coffee, and doing odd chores until going to work at 5:00 p.m. She ate the employee's meal about 8:00 p.m. and finished work at 1:00 a.m. She usually had several drinks at that time, and often went out before going home. She smoked at least two packs of cigarettes daily. Clinical observation revealed generalized diffuse gingival inflammation, moderate subgingival calculus, purulent exudate, gingival hemorrhage to touch, periodontal pockets of 4-6 mm. around all teeth, moderate alveolar bone loss and generalized mobility. The patient was aware of nocturnal bruxism. Can the patient be treated successfully for her periodontal disease? The answer is that it is doubtful that treating local factors alone, with no change in her way of life, could promise periodontal cure. And while the actual example is extreme, many of our patients present only minor variations.

The second example is a 40-year-old white male referred for periodontal examination and treatment. He worked as a Social Security Administrator. He had no complaint, but had recently moved to San Bernardino and had gone to a local dentist to have his teeth cleaned. His medical history included the loss of his right leg in a hunting accident eight years ago, and a diagnosis of Buerger's disease in the left leg. Otherwise, he was in good health, had good dietary habits, and was not considered nervous. His dental history included periodontal treatment with surgery in 1958. He had moved to another community just after treatment, and had had a "five-minute polish" cleaning annually since 1958. Clinical examination revealed pink, firm, well-stippled gingiva, with little evidence of inflammation despite excessive subgingival calculus and bacterial plaque. The architecture of the soft tissues was deflecting and adequate. Pocket measurements of approximately 5 mm. were made around all posterior teeth. There was no evidence of hemorrhage or purulent exudate. He had no symptoms, and his mouth felt about the same as it had for the last few years. There were no missing teeth or caries. The 1958 radiographs were obtained from the periodontist who had originally treated him. No changes could be seen in the alveolar bone level, despite gross neglect and extensive local irritation. One other significant clue was that, despite having lost one leg and having Buerger's disease in the other, he continued to smoke up to a package of cigarettes a day. Since he was aware that smoking is considered a specific etiologic factor in this disease, we can only conclude that he is not overly concerned about his physical condition, and has adapted well to the problem. His way of life and the character of his tissues indicate that his periodontal condition does not reflect disease

at present. Careful scaling, root planing, and curettage, together with an attempt to re-emphasize the importance of adequate oral hygiene, should be the only therapeutic procedures indicated in this case.

A third example helps to further illustrate the concept. A 31-year-old white female presented for consultation regarding bilateral pain in the temporo-mandibular joints and masseter muscle areas together with gingival bleeding and soreness of three months duration. She had been advised by another dentist to have a full-mouth reconstruction ($5,000). This, he promised, would open her bite and correct the cause of the pain. She had three children, and her husband was a bread salesman. Her medical history included asthma, psychiatric care after her first child was born, a recurrent rash on her neck that she attributed to nerves, and admitted severe emotional tension for the past year. Oral examination revealed diffuse acute inflammation of the gingival tissues, subgingival calculus, and gingival hemorrhage to touch. She had a freeway space of approximately four millimeters. Her teeth were slightly mobile, but this was inconclusive since she had worn a bite plate for one month which opened the bite and relieved the joint pain. When she took the bite plane out, she said she felt severely depressed, and the pain returned. She also reported that tranquilizers given her by her physician had the same helpful effect as the bite plate in relieving both pain and depression. An examination by an ear specialist had been negative. Can either periodontal therapy or a full-mouth reconstruction solve this patient's problem? The cause of her problem is neither calculus nor bite relationship. The answer is obvious that she needs help in solving her way-of-life problems before extensive dental treatment is planned. These three

actual examples have been used to illustrate the importance of the patient in periodontal problems. A direct cause-effect relationship between local etiologic factors and periodontal pathosis cannot be demonstrated in these examples, nor in any other patients, and treatment cannot be based on an assumption of such a direct relationship.

V. TREATMENT IMPLICATIONS

DETERMINATION OF THE PRESENT PERIODONTAL CONDITION

The concept of periodontal disease that has been presented has a number of treatment implications. If the patient must exceed his physiologic adaptability before actual periodontal destruction takes place, the determination of his present response must be made. It is important to decide whether changes in crevicular depth and alveolar bone loss are occurring at present, or whether they are the result of disease that took place at an earlier time and are not changing at present. The presence of deepened crevices and alveolar bone loss indicates periodontal destruction, but does not justify a diagnosis of periodontal disease. If there are neither clinical signs nor other evidences that the patient is physiologically out-of-bounds, the periodontal condition may be assumed to be in a state of equilibrium in which the patient's resistance and reactions are adequate against any local irritants present.

PREVIOUS PERIODONTAL DISEASE

If a patient appears to be well within his own physiologic boundaries, as judged by clinical observations and his way of life, what modifications can be made in treatment? This raises the question of whether present criteria for treatment, which insist that pocket elimination is necessary for a periodontal cure, are applicable to all patients. If pocket elimination means only the surgical removal of the tissue above the base of the pocket, the criteria cannot be applied to

all patients. If pocket elimination also means the change from a periodontal pocket to a deepened crevice with restoration of the tissues to health, the criteria are applicable. If destruction of the periodontal structures is not occurring at present, and if the architecture of the gingivae is adequate, then elimination of the local irritants by careful scaling, root planing, and curettage, the correction of obvious sources of occlusal trauma, and instruction in proper maintenance should be sufficient treatment. Elimination or control of the aggravating conditions provides a margin for error in the event of future disease, and minimizes the potential destruction. The tissues may be expected to readapt around the teeth, and the color, texture, and tone will return to normal. The periodontal pockets become deepened crevices which can be maintained at their present depth. Most clinicians have seen many instances where this has occurred in inoperable areas, reattachment failures, and cases treated with scaling and curettage only.

Brill and Bjorn[51] and Harvey[52] have shown that there is a physiologic movement of tissue fluids from the crevice into the oral cavity. In these cases, the tissues could be expected to resist and resolve the effects of extraneous matter which might be forced into the crevice. However, if architectural deformities of the soft tissues exist, such as craters interproximally, excessive fibrous hyperplasia, and other conditions which make maintenance extremely difficult, if not impossible, surgical procedures are indicated. But surgical pocket elimination results in a number of disadvantages, such as increased susceptibility to root caries, tooth sensitivity, increased complexity of the oral hygiene procedures, frequent esthetic problems, and even the question presently being debated--whether the reconstituted gingiva, which at first

is essentially scar tissue, is as resistant as the normal gingiva. So, in the absence of disease, and with adequate soft tissue architecture (regardless of the underlying configuration of the alveolar bone), surgical pocket elimination can be classified as a prophylactic procedure for these patients. And just as the value and necessity for prophylactic occlusal equilibration has been questioned, one questions whether surgery is essential or desirable for these patients with no evidence of present disease.

PRESENT PERIODONTAL DISEASE

If the patient's way of life and clinical observations indicate disease at present, the clinician has a further obligation. It is doubtful whether minimizing the local etiologic factors alone will arrest the progress of the periodontal disease. He must also point out the inner aspect of disease to the patient. This frequently removes the patient's preoccupation with his teeth and mouth, and involves his intellect. The patient begins looking for the causes of stress in his life, and whether it be psychological, dietary, medical, or something else, this is the first step toward improvement. This knowledge on the patient's part also makes him aware of the dentist's limitations in treating his problem, and helps ease the extreme dependence that the patient is anxious to transfer to the dentist. It outlines the responsibilities of both dentist and patient if periodontal therapy is to be successful.

The fight against periodontal disease must be an alliance. The patient cannot say, "Here I am, cure me." He must understand what is causing his disease. Pointing out the inner aspect of disease is only

pointing out what patients instinctively know--that stress affects the body. Periodontal therapy for these patients involves as complete elimination of the local etiologic factors as possible. Scaling, root planing, and curettage are essential, as well as oral hygiene instruction. Maintenance of their periodontal tissues free from local irritation is more important than in healthy patients. Again, if architectural deformities of the soft tissues make it impossible for the patient to remove the bacterial plaque and prevent the formation of calculus with meticulous oral hygiene, periodontal surgery must be done. But surgery should be avoided if possible, at least for awhile. Surgery itself often implies cure to the patient, and puts the burden of responsibility on the dentist. The periodontal prognosis on these patients has to be guarded, and dependent upon their bringing themselves within their own physiologic boundaries, since absolute elimination of local irritants is unattainable. These precautionary comments do not imply that these patients should not be treated periodontally. Judicious local therapy can frequently maintain their tissues with minimal destruction until their way of life is brought within their physiologic boundaries and the prognosis improves. Time is the only judge of the success of these measures. But these patients must understand both aspects of the etiology of their periodontal problem--their own responsibilities and the dentist's limitations. Just as a gastrectomy will not correct the cause of an ulcer, neither will periodontal surgery correct the cause of periodontal disease.

THE AIMS OF PERIODONTAL THERAPY

Periodontal disease has a more complex etiology than is presently implied in most periodontal literature. It is impossible to draw a direct

cause ——→ effect relationship between local factors, such as calculus and alveolar bone loss, without taking into consideration the individual patient. The dentist cannot consider local etiologic factors alone, and arrive at an automatic treatment plan that fits all patients. It is individual patient differences that dictate differences in treatment of their periodontal conditions. While it is not our prerogative to always treat the underlying cause of the periodontal disease, it is our responsibility to recognize it, to help the patient understand and correct it, and to plan our treatment and make our prognosis accordingly.

Our treatment responsibilities for all patients may now be summarized as follows: (1) recognition of the ultimate cause of the patient's periodontal condition as well as the aggravating conditions; (2) education of the patient to help him correct pathogenic factors in his way of life, or to recognize his own physiologic boundaries; and, (3) to assist the response of the patient by removing or controlling the aggravating conditions.

Although the search for the ultimate cause of the periodontal condition does not always lead to a clear and precise answer, the understanding of the patient derived from the search is of immeasurable value. When it is not possible for the patient to correct the pathogenic factors in his way of life, or when the limitations of his genic or developmental background have resulted in a reduced physiologic adaptability, the knowledge of these factors helps the patient and the dentist to determine the treatment methods and treatment potential. Although assisting the response of the patient through control of the aggravating conditions may not in itself cure periodontal disease, it is an extremely important part

of periodontal therapy. Without control of these aggravating conditions, the disease response is magnified, the rate of destruction is increased.

Further correlations should be done on personality profiles and the various manifestations of periodontal disease. Because of its prevalence, and the observable clinical changes, periodontal disease may be a readily available vehicle for the study of disturbances of the patient, including nutritional, emotional, and constitutional disturbances. The further refinement of laboratory tests and values may be accomplished through the study of changes in the periodontal tissues. Also, records must be kept, and statistics gathered on teeth treated with and without surgical pocket elimination. Important information may be derived from correlations on an epidemiologic basis, in which individual variations are minimized.

There is unquestionably much to learn about periodontal disease. The etiology may be as complex as the nature of man. But the concept of the pathogenesis of periodontal disease that has been presented provides an understanding of the varying responses of different individuals to the same local factors and treatment. It also provides a rationale for treatment based upon these individual patient differences.

VI. SUMMARY AND CONCLUSIONS

1. Presently accepted concepts of the etiology and pathogenesis of periodontal disease do not take adequate cognizance of the importance of the individual patient in the disease process.

2. Disease represents a response on the part of the patient to a single or multiple stressor(s).

3. Periodontal disease resulting in pocket formation and alveolar bone loss does not take place unless the patient has exceeded his physiological adaptability. When this occurs, local aggravating conditions increase the intensity of the disease response.

4. Correlation of observable clinical signs and symptoms with the patient's medical and dental history and his way of life gives the most accurate assessment of the patient's disease parameters.

5. Time is an important dimension in evaluating the patient's periodontal condition. The perceptive clinician must differentiate between periodontal disease, in which pocket formation and alveolar bone loss are occurring at present, and periodontal destruction which may have occurred at some previous time.

6. The criteria for treatment, which insist on pocket elimination based upon the concept that pockets will inevitably deepen, are not applicable to all patients. Many patients may be treated successfully by restoring the gingival tissue to health rather than by removing the tissue.

7. Periodontal therapy should be based on the following objectives: (a) recognition of the ultimate cause of the disease response; (b) helping

the patient to correct this cause; (c) assisting the response by control of the aggravating conditions.

8. Successful periodontal therapy results from the mutual efforts of the patient and the dentist to eliminate the etiologic factors. Each must recognize his own responsibilities and limitations.

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LOMA LINDA UNIVERSITY

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THE PATHOGENESIS OF PERIODONTAL DISEASE AND TREATMENT IMPLICATIONS

by

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An Abstract of a Thesis

in Partial Fulfillment of the Requirements

for the Degree Master of Science

in the Field of Periodontology

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Presently accepted concepts of the etiology and pathogenesis of periodontal disease do not adequately explain the following consistent clinical observations: (1) equal local etiologic factors do not result in equal periodontal destruction in all patients; and, (2) equal therapy is not equally successful in all patients. The differences in patient response must be due to differences within the patient. By relating the broadened concept of disease, as described by Engel, to periodontal disease, the reasons for these individual patient differences become apparent. This concept consideres disease in relation to the patient's genic and developmental background, as well as the present environmental factors and the patient's way of life. The concept may be extended to define disease as a response of the patient to physiologically unacceptable conditions. Each patient has varying physiologic boundaries beyond which he is unable to adapt to single or multiple stressors, and the disease response occurs.

These concepts applied to periodontal disease result in the following conclusions:

1. Periodontal disease resulting in pocket formation and alveolar bone loss does not occur unless the patient has exceeded his physiologic adaptability. Local aggravating conditions increase the intensity of the disease response.

2. Correlation of observable clinical signs and symptoms with the patient's medical and dental history and his way of life gives the most accurate assessment of his parameters of disease.

3. Time is an important dimension in evaluating the patient's periodontal condition. The perceptive clinician must differentiate between periodontal disease and periodontal destruction which may have occurred at some previous time.

4. If the patient shows evidence of periodontal destruction but not periodontal disease, surgical elimination of the periodontal pockets may be unnecessary.

5. Periodontal therapy should be based on the following objectives: (a) recognition of the ultimate cause of the disease response; (b) helping the patient to correct this cause; (c) assisting the response by control of the aggravating conditions.