INTRODUCTION

I would like to thank the contributors to this project for their efforts on behalf of the Texas Society of Periodontists. We strived to put together information that is current, topical and applicable to clinical practice. We commend the Texas Academy of General Dentistry for cooperating in our effort to get this information to our colleagues and for their interest in disseminating information on the subject. Special thanks also go to Dr. Douglas B. Willingham and the staff of the Texas Dental Journal for their guidance and help.

Our series, entitled: “Periodontics: On the Verge of a New Era,” covers a wide range of topics in periodontics, and every attempt has been made to provide consistent integrated information. It is our feeling that one would gain the most by reading the articles in order, because each builds on its predecessors.

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PERIODONTICS —
on the verge
of a new era

McGuire

Treatment decisions in modern periodontics can often be confusing. This is not surprising considering the fact that each day we are bombarded by conflicting claims carried in many popular non-refereed journals and even in some refereed journals and continuing education courses. Headings such as “Gum Disease Alleviated by Pellets,” and “String Heals Periodontitis,” catch our attention and tend to confuse rather than clarify the problems of treating periodontal diseases. This media blitz is just beginning, fueled by big business entering the field of periodontics. Advertisements now focus on periodontal disease instead of dental caries. Unfortunately, their message might be designed to improve their earnings rather than your patients’ problems with periodontal disease.

Rather than a time of great confusion, I would like you to view today as a time of great opportunity. Dr. Kenneth Kalkwarf, dean of the San Antonio dental school, suggested an excellent analogy to explain much of the confusion in periodontics. It is taken from the book MegaTrends, by John Naisbitt. In that best seller, Naisbitt suggests that we are moving from the industrial era to a new era based on informational services. He states that we are living in the “time of the parenthesis” . . . a time between eras where there is great uncertainty, but great opportunity. If we can learn to make uncertainty our friend, we can achieve much more than in stable eras. In stable eras, everything has a name and everything knows its place, and we can leverage very little. But in the time of the parenthesis, we have extraordinary leverage and influence — individually, professionally, and institutionally — if only we can get a clear sense, a clear conception, a clear vision of the road ahead. It is this vision of the road ahead in periodontics that I will attempt to present. But before you can look ahead, you have to understand the past.

. . . modern periodontics has been through at least two separate and stable eras and is entering a third.

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that modern periodontics has been through at least two separate and stable eras and is entering a third. The first era of modern periodontics began with Loe's discovery in 1965 that plaque causes gingivitis. For the next ten years following this discovery, we were in an era dominated by the non-specific plaque theory. That theory stated that all plaque was essentially the same and too much of it caused disease. It was thought that there was no difference between plaque from individuals with gingival health and from those with periodontitis. It was the amount of plaque present that caused disease. This theory came from studies that, at the time, were primarily epidemiological and looked at supragingival plaque and its relationship to the severity of disease.

In retrospect, it is not surprising that these types of studies yielded conclusions that supported the non-specific plaque theory. We used several basic techniques to treat all periodontal disease. If the patient did not respond to the technique, then there was something wrong with the patient. It was a simple time to practice, and we slept well at night confident of our therapy.

As our research techniques grew more sophisticated, we began to see that supra gingival plaque was different from subgingival plaque. We also began to identify many of the individual microorganisms, and found that some of them seemed to be related to different periodontal diseases. This line of thought represented the bacterial specificity theory. This theory dominated a new era that spanned the decade from 1975 to 1985. During this period, we were confident in our thinking that it was the quality and not the quantity of plaque that was important. The role of the host was largely ignored.

It now seems that we are moving into a new era, and it is because of this transition that we are faced with so many conflicting theories and therapies. A dogma has not yet been established, and this does make it confusing when we are trying to treat our patients. On the other hand, the potential is very exciting because our treatment is not limited by strict guidelines. We might call this new era the host-bacteria interrelationship era.

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We are beginning to understand that while individual microorganisms are important, they are no more important than the way the host interacts with them. It is this dynamic interrelationship that controls whether or not disease is present. This host-bacteria relationship permeates the important changes that have occurred in periodontics in the last few years.

In the non-specific plaque era, we recognized four periodontal diseases: gingivitis, periodontitis, necrotizing ulcerative gingivitis (NUG), and periodontosis (which we now call juvenile periodontitis). At that time, we assumed that untreated gingivitis would always progress into periodontitis, and we were not certain that periodontosis should even be classified as a periodontal disease because it was not plaque-related. Many of these concepts were questioned and changed during the bacterial specificity era. During that era, we found that there were more than four different periodontal diseases (see Wilson in the following article). We not only discovered that juvenile periodontitis was plaque-related, but we also found that many of the other periodontal diseases seemed to be associated with particular microorganisms. It also became evident that not all gingivitis progressed into periodontitis.

We have come a long way from our non-specific plaque theory. We talk with confidence about bacterial specificity, but the truth of the matter is that while we have learned much, there are many bacteria in the periodontal pocket that we have yet to identify. The periodontal pocket is the most complex ecosystem in the body, having more anaerobic bacteria than the intestine. Our knowledge is further limited by the fact that most research done on plaque in the past has been concerned with plaque maturation, not disease etiology. We therefore know much about how plaque is formed, but until recently we have known little regarding plaque's relationship to attachment loss. It is also important to realize that we have found certain very destructive bacteria that do not produce plaque. It is, therefore, dangerous to inform our patients that they do not need to be seen for routine recalls because they do not have any visible plaque. Along with our visual inspection, we need to rely on other clinical parameters such as an increase in probing depth or bleeding upon probing.

Research in the bacterial specificity era has also demonstrated a number of factors that allowed us to better understand disease progression.

One of these discoveries helps explain why some of our patients with very little plaque seem to have great problems controlling periodontal diseases. It was found that some patients with periodontal disease have a defect in some of their white blood cells. The PMN or polymorphonuclear leukocyte is our bodies' chief cell in...
charge of fighting infection. It makes sense that if there is a defect in this neutrophil that prevents it from reacting to infection, then the disease will progress much further than it would otherwise. This helps explain why some of our patients have a much greater problem controlling their periodontal disease, even though their home care is good. In that patient, even a small amount of bacteria can cause a great deal of attachment loss (increased pocket depth) because the altered neutrophil does not do its job in controlling the infection. PMN defects were first discovered in juvenile periodontitis (periodontosis), and have now been confirmed in many different types of periodontal diseases. Some of these defects in the neutrophil seem to be genetically based, and others seem to be mediated by the bacteria themselves.

Another significant discovery is bacterial invasion. At one time, we thought that the spirochetes found in NUG were the only bacteria that invaded the tissues in any periodontal disease. We now know that bacteria invade, or are at least present, in the tissue in many forms of periodontal disease. This concept has far-reaching consequences. For example, it makes us aware that we not only need to scale the plaque from the root surfaces, but we also need to be concerned with the reservoir of bacteria in the soft tissue walls of the pocket. Much work is now being carried out in bacterial culturing, or identifying bacteria through agents such as DNA probes and monoclonal antibodies. These tests will help identify which antibiotics might be of help in dealing with the bacteria that have invaded into the tissue. Antibiotics may serve as useful adjuncts in the treatment of certain types of periodontal disease such as juvenile periodontitis, rapidly progressive periodontitis, and refractory adult periodontitis. However, it should be pointed out that they seem to have little value in treating gingivitis and chronic adult periodontitis, which are the two most common periodontal diseases. We are very effective in treating these last two diseases through conventional therapies only.

A number of difficulties arise in the treatment of periodontal diseases by antibiotics. One problem is that at the present time, we cannot always determine on a clinical basis whether we have changed the microflora with our antibiotic therapy. Another problem is that periodontal disease as a whole does not act like a classical infection where we have an invading microorganism. It acts more like an opportunistic infection caused by bacteria that are normally present but in an abnormal proportion one to another. A good example of an opportunistic infection is candidiasis produced by antibiotic therapy. The Candida albicans were always present, but the antibiotic changed the environment to one that allowed them to overgrow, creating the disease. This abnormal proportion is contributing to the disease or lack of it helps give us a much better understanding of the progression of periodontal disease. For example, we have always known that pregnant women seem to have more problems with periodontal disease. This was attributed to hormonal changes which made coping with infection more difficult for them. But we now know that in pregnancy, increased amounts of estradiol and progesterone are produced. This changes the environment to one where more virulent microorganisms are favored. So it is not just that pregnant women have a harder time controlling periodontal disease; it is that the environment is changed to one that is more conducive to disease. Similar environmental changes may occur with puberty, smoking, medications, systemic diseases (such as diabetes), or with other stresses. We have even found that improperly contoured subgingival margins on dental restorations are often associated with disease progression, not because of the bulk of plaque that accumulates, but because the environment changes to one that allows more virulent microorganisms to be present.

Another significant change is the way we envision disease progression. It was thought that pocket deepening occurred in a linear fashion in all patients. We felt that it started at a particular time and progressed at a steady rate until the teeth were lost. Evidence now indicates that periodontal disease may be episodic, occurring in bursts of activity that may be site-specific and not generalized throughout the mouth (Figure 1). There appears to be a delicate equilibrium between the host and the microorganisms. Disease occurs only when something happens to upset this balance, such as a defect in the PMN or an amalgam overhang. This again points out the importance of the host, and it helps explain why our patients tend to go through periods of
time where they have more problems controlling their disease. This concept reinforces frequent periodontal maintenance and data collection since we cannot yet predict when one of these destructive episodes will occur.

With all of the changes in periodontics occurring so rapidly, many of us are uncertain about how best to treat our patients with these problems. The January, 1986 issue of AGD Impact stated that “lack of perio knowledge can be hazardous to your career,” citing recent lawsuits against dentists for lack of diagnosis and treatment of periodontal disease.16 While the lack of knowledge is dangerous, misinformation can be just as damaging. It is important to keep informed so that you can differentiate between misinformation and worthwhile changes. Even though our information is changing rapidly, one thing remains constant — our commitment to provide for our patient the very best dental care. It might have been easier for us to provide this care in one of the previous eras when we were comfortable with our knowledge and our therapies. At the present time, many practitioners feel there is no central dogma to rely on. That makes many of us uncomfortable, but it also presents us the opportunity to improve patient care. I hope that through the articles that follow, you will gain “a clear sense, a clear conception, a clear vision of the road ahead,” and you will look at this time of the parenthesis in periodontics not with a feeling of confusion, but with a sense of excitement. ■

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