

Guest Editorial I Am Worried

I have been thinking about our specialty a great deal lately. And quite frankly, I am worried. Don't misunderstand me. I am not worried about our specialty; I am worried about myself. I am having a difficult time keeping up with all of the advances in periodontology. And to make matters worse, there seems to be no end in sight.

It all seemed so straightforward in the beginning. I arrived at Harvard in the summer of 1970, eager to become a periodontist. And over the next 3 years, with the help of dedicated teachers, I finished my training program armed with all I would need to know for a career in periodontology. I knew that periodontal disease was worldwide and affected most people, if not all, beginning at age 35. I knew that all people were at equal risk for developing the disease. I knew that when untreated, the disease progressed slowly and steadily until teeth were lost. I knew that bacterial plaque caused periodontal disease and that the main pathogen was *Bacteroides melaninogenicus*. I knew that the host was involved somehow in the disease process, but likely minimally. I knew the importance of root planing and split-thickness surgical flaps. I knew that loose teeth should be splinted with orthodontic wire prior to surgery to foster good wound healing. I knew that Darvon Compound 65 was best for postsurgical pain management. And last but not least, I knew that my patients needed to perform meticulous home care. To that end, I had a phase contrast microscope and could show my patients all of the strange and wiggly things in the small plaque scrapings from their teeth.

And so, it was an exciting day in June when I embarked on my career as a periodontist. I was eager to use all of my knowledge and skills to treat patients presenting with periodontal disease. I couldn't imagine much of the information I had learned in my program changing in the coming decades.

Alas, the next month, July, I encountered a glitch in my understanding of periodontal disease causation. Sig Socransky's group at Forsyth Dental Center reported that when using anaerobic techniques to sample periodontal pockets in children with periodontosis, they found bacteria never before been seen in the human mouth. The bacteria at severely diseased sites were primarily, of all things, Gram-negative anaerobic rods.¹ How could I know at the time that periodontology was entering the "golden age of oral microbiology"? Soon, a completely new understanding of the bacterial etiology of periodontal disease would emerge, and periodontal pathogens such as *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans* would be found. Still, I decided to not let this small breakthrough in the understanding of periodontal disease etiology rattle my confidence. I pressed forward.

Alas, in August, Paul Goldhaber and his colleagues at Harvard reported that bone resorption in tissue culture could be blocked by inhibiting specific inflammatory mediators such as prostaglandins.² How could I know that the work of Goldhaber, as well as the research of investigators such as Max Goodson, Roy Page, and Robert Genco, would begin to explain the role of the susceptible host in periodontal disease initiation and progression? Within a few years, understanding the role of the host would become a key element in explaining the pathogenesis of periodontal disease. And yet—full of confidence—I pressed on.

Alas, in September, Sig Ramfjord and his colleagues at Michigan reported the initial findings from their longitudinal study of the efficacy of periodontal treatment in human subjects.³ How could I know that this research would open the door to the future of clinical trials research in periodontal disease management and that randomized controlled clinical trials and evidence-based periodontics would steadily emerge in my specialty? Who knew that Jan Lindhe and his group would further define clinical trials research in periodontics and that workshops in the US and Europe would be convened to further understand the design and conduct of clinical trials research? But unfazed, I continued onward as an exceedingly well-informed young periodontist.

Alas, October brought news that a new drug for pain management called Motrin would be available the first of the year. Apparently, it was called an NSAID and would be preferred to Darvon Compound 65 or Empirin Compound #3. I confess that I had never heard of Motrin or an NSAID, so this was a new concept for me in pain management.⁴ Nonetheless, I moved forward as an in-the-know periodontist.

Alas, in November word arrived from Denmark that Harald Löe and his colleagues were attempting to regenerate periodontal attachment structures with the concept of epithelial exclusion.⁵ How could I know at the time that the concept of guided tissue regeneration and guided bone regeneration would emerge in our specialty and forever change the way we treat patients?



Finally, December arrived, and to my recollection, there were no breakthroughs in periodontology that month. Thank goodness. I needed a break. I paused and reflected on my first 6 months as a periodontist. I wondered just how much our specialty might change. I was already scrambling to keep up. But surely this period was a fluke. Surely periodontology wouldn't continue to change this much.

But now, after all these years, I think I know the answer. I realize I chose a specialty that attracts phenomenal clinicians and scientists to its ranks, and that through their genius and hard work, our specialty will always be evolving. For me, it has been quite the ride so far.

Our specialty can now regenerate lost periodontal attachment structures using new surgical techniques, devices, and biologics. Tissue engineering is no longer a concept but a reality. Signaling molecules such as growth and differentiation factors are being used daily to foster wound healing. New therapies to prevent or slow periodontal disease are being validated through clinical trials. Both novel anti-infective and host modulatory therapies are actively studied. We now know that tissue-destructive components of inflammation can be modulated or blocked. Teeth can be replaced with root-form dental implants. Implant surfaces continue to be improved, providing more predictable and faster wound healing. And if bone is needed for implant therapy, it can be regenerated. Plastic surgical techniques allow us to treat a variety of soft tissue defects. Recent research suggests that soon we may be using live connective tissue cells to rebuild lost papillae. And certainly, evidence continues to emerge that periodontal disease is a risk for several systemic conditions. The area of our specialty that we now call "periodontal medicine" is intensely focused on advancing this issue. New responsibilities are emerging in our specialty, demanding that we maintain not only our patients' oral health, but also their health overall. Further, we will need to work much more closely with medical colleagues to ensure that periodontal disease does not contribute to systemic disease. Recent symposia or workshops in collaboration with cardiology or endocrinology have started us on this pathway.

Quite frankly, there seems to be no end in sight for the continued expansion and change of periodontology. I think I have every right to be worried that I cannot keep up with our specialty. And yet, I also cannot hide my sense of excitement.

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