

## Who's in Charge Here?

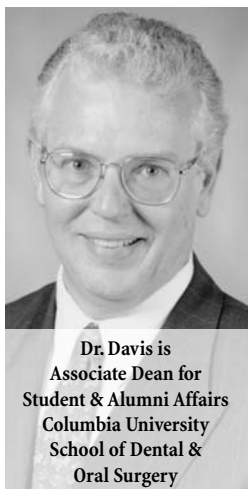
In order for dentists to regain control over the practice of their profession, they must be willing to embrace change.

CHANGE IS DIFFICULT...in any situation. We have heard that said in so many ways at so many times. In a private dental practice, especially a solo practice, where the dentist is driven by careful attention to the best for his or her patients but still must keep an eye on the economic exigencies of the practice, change can be particularly fraught with difficulty. We often find ourselves considering patient care issues and thinking, "But that is what I learned in dental school." Or, "Our patients expect it." Or, "Third parties pay for it." Or, "They don't cover that." These are classic barriers that we confront frequently when adopting new technologies, novel methods and recently published guidelines.

Life should be so simple that we can readily, given the economic climate of health care in the United States, adapt to new findings to benefit our patients.

Dr. Bruce Baum of the Gene Therapy and Therapeutics Branch of the NIDCR/NIH wrote in the summer 2004 Journal of the American College of Dentists (p. 28), "Current dental practice, though biologically based, has not yet begun to adapt to the realities of modern biology for most diagnosis and treatment."

Examples of this restricted adaptation or movement to new technologies are easily identified. Since the mid 1980's, pit and fissure caries have constituted approximately 80% of new carious lesions in the United States. In 2000, Surgeon General David Satcher, with the support of a huge project team and multiple contributors, produced "Oral Health in America: A Report of the Surgeon General." Among a wide variety of findings and recommendations, that report noted that "...as much as 90% of all dental caries in school children occur in pits and fissures," and "...the teeth at highest risk by far are permanent first and second molars."



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The report references the NIH Consensus Development Conference of 20 years ago (1984), which noted, "...an extensive body of knowledge has firmly established the scientific basis for the use of sealants." The Surgeon General also cites the federal government's "Healthy People 2010" objectives, which state that at least 50% of first permanent molars in children under age 8 years should have received sealants by 2010.

With approximately five years until 2010, recent data indicate that only 25% or fewer children have such sealants. Why? It may be because many of us simply do not yet "trust" sealant technology, even though it has been evaluated in practice since the early 1960's.

Perhaps some of us are intimidated by reports, although anecdotal in nature and with no evidence base, that "new" caries have been seen under a sealant, thus providing a rationale for never placing sealants. This contradicts meta-analyses conducted since the mid-1980's that clearly demonstrate the success and extraordinary cost-effectiveness of well placed, carefully monitored sealants.

### **No More Six-Month Recall?**

Similarly, because patients have been taught by us that "you need a cleaning" at least annually or better, biannually, and because "it is easier" to carefully examine teeth with plaque and debris removed, we convinced patients and third parties that they must pay twice a year for a thorough rubber cup/pumice prophylaxis.

Evidence, which has accumulated over the last decade, makes it quite clear that we are in fact removing significant amounts of fluoride-rich surface enamel with every prophylaxis. The reality is, if there is no visible stain or remaining trace of calculus, soft dental

plaque is readily removed with a toothbrush prior to our thorough examination. Such an approach, the toothbrush prophylaxis, is somehow viewed as not a “real” service, and that is compounded by patient/parent expectations and our economic “exigencies.”

How many of us have truly moved forward to employ individual patient “risk assessment” for caries, periodontal disease or oral cancer, and, as a result, modified how we educate and manage such patients with an anticipatory guidance protocol? It is clear that not every patient is best suited by our classic six-month recall. Those with higher incipient disease rates would better be seen at three months; for those 50% of children under age 12 who have never had a carious lesion and have sealants in place, based on the evidence, an annual recall makes more sense. After all, the “science” of the six-month recall derives from the label on a proprietary tooth powder produced back in the 1930’s, which included the statement, “...see your dentist every six months.”

Our diagnostic techniques and our risk assessment technology now enable us to make much more informed decisions and to be more accessible to patients who genuinely require more intensive and more frequent care.

### **Out With the Old**

In another venue, our technology is growing rapidly for diagnosis. New methods for discovering new carious lesions are jumping from the drawing board in rapid succession. We all learned in dental school that the gold standard diagnostic method for pit and fissure caries, 80% of new lesions in the United States, is the use of an ultra-sharpened explorer with some serious pressure. And yet, it is clear that an explorer with serious force behind it can actually chip, crack and damage healthy enamel adjacent to pits and fissures. We may even be transporting and inoculating cariogenic bacteria to uninfected fissures!

With the advent of a variety of new technologies, beginning with simple magnification for visual evaluation of pits and fissures and now adding enamel fluorescence, computer analyzed transillumination and other new technologies, it is time to relegate the explorer to more appropriate applications, such as checking weakened tooth structure or restoration margins.

We all accept that oral hygiene clearly plays a major role in the prevention of gingivitis and periodontal disease through the removal of plaque, bacteria and toxins, thereby preventing the inflammation that is the actual disease process. But one of the most shocking concepts for all of us to accept is that a direct relationship between oral hygiene and caries activity is more tenuous. There simply is not significant evidence-based literature that poor oral hygiene is the main etiology of caries! We all have seen the brother with no caries and terrible hygiene whose sister has excellent hygiene and four new proximal lesions. The secret herein is that she snacks all day; the incipient lesions never have a chance to remineralize at an almost continuous oral pH of 5.5.

As noted again in the Surgeon General’s report, in the section on “Daily Hygiene and Dental Caries Prevention,” there is clear value in tooth brushing and flossing for periodontal disease prevention and to rid the mouth of food debris, eliminate odor and improve appearance. We do and should depend on parents to maintain reasonable levels of hygiene for their children to give them an understanding of the importance of such efforts.

More important for caries prevention, however, is low concentration fluoride exposure on a daily basis. This is best provided topically by toothpaste and by fluoridated water through its systemic reflection in constant low salivary fluoride levels. Even in the mouth of the “frequent snacker,” where continual pH drops initiate demineralization, low levels of fluoride can shift the balance to remineralization and save the day. We now know this is the critical fluoride mechanism, not the original pre-eruption fluoride incorporation of developing teeth. In even the cleanest mouth, the ongoing battle of demineralization is real; it is the continued availability of fluoride ions in the mouth at low levels that shifts the balance to success in remineralizing incipient lesions.

A superb example of not quite understanding these new biological considerations is found in a recent “rule change” by the Northeast Regional Board. Examinees are now allowed to use live patients with proximal carious lesions radiographically only “halfway through the enamel.” This replaces the former requirement that the lesion had to have reached the DEJ. These earlier incipient lesions are exactly the lesions, which with careful monitoring of patient compliance, the new remineralization technologies are designed to address.

### **Seize Control**

The question, “Who is in charge here?” is focused on what we the practitioners, the educators, and our associations, academies and societies must do to maintain control of contemporary oral health care practice. It is we who are trained to not only understand, but to document through carefully controlled, evidence-based research, the data on which we make every decision. Our patients’ expectations have been developed through education by our profession. If we need patients to understand that perhaps, “this time, at this visit, you may not need a rigorous pumice prophylaxis,” or that, “your teeth are, in fact, as white as they should be,” it is for us to provide that “new” education.

It is understood by all of us that third party payment for services is critical to the survival of many independent private practices and to the institutional “safety nets” for our less economically viable patients. Should not the third parties and the specific services they cover reflect our most current understanding of the underlying biology, technology and state-of-the-art research that our profession is producing? It is for our organizations and each of us as individuals to educate third party payers and patients and to push emphatically for change. The new world in which we are part-

ners in treatment decisions with our patients is appropriate, but our patients need as much relevant information as we can provide on which to base their decisions.

Our many years of study and practice and the resultant knowledge and its application are the ultimate value to our patients. Yet our economic survival still hinges on “procedures” produced. It is time for action by each of us working together through our organizations to begin to again place the education of the public and third parties in the fore. Only then can we offer what is truly proven best for our patients.

As a final example, there is clear evidence that the implementation of preventive protocols, individualized risk-based timing for recalls, and patient education about sealants, frequency of eating and personal oral hygiene are the critical factors in oral wellness. A large, third party-based, multiple-provider pediatric practice in Minnesota recently reported\* the results of several years of individual risk assessment and anticipatory guidance efforts in a pediatric setting. Using technologies we now have, such as fluorides, sealants and varied recall lengths, the outcomes were clear that more care was provided to the children who most needed it, and that total disease levels throughout the entire population of patients decreased. A clear result was that the per capita average cost of providing the care decreased, and the practice’s net income increased. This model was of a large patient population over a sufficient time to allow these conclusions.

Change will forever be difficult, especially for each of us in our individual settings. As it has been said about the challenge of change, “What doesn’t kill us, makes us stronger.” Let us again be in charge as the educators and clinicians.

Finally, again quoting Dr. Bruce Baum of the NIDCR/NIH in that same article, when discussing our need to adapt new technologies, Dr. Baum said, “...I believe that a considerable and multifaceted effort, by the organized profession and the dental education communities, will be required to prevent dentistry from being left behind altogether.” ■

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\* Dr. Paul Walker, professor of pediatric dentistry and past president, American Board of Pediatric Dentistry, presentation at 2001 ADEA Annual Session.

# Dental Caries

## An Infectious and Transmissible Disease Where Have We Been and Where Are We Going?

*Page W. Caufield, D.D.S., Ph.D.*

### **Abstract**

Dental caries is often referred to as an infectious disease, which means different things to different people. Pediatric dentists may be tempted to liken caries to other childhood diseases they encounter yet treat caries from mostly a mechanical or surgical approach. On the other end of the spectrum are clinicians who view the oral cavity as a site teaming with bacteria and think that reducing the overall microbial mass

using antimicrobial agents will somehow affect this infectious disease. Here, we argue for another view, that is, to combine the most effective portion of each therapy to yield an effective outcome: the treatment and prevention of dental caries.

Because primary teeth are eventually shed, pediatric dentists may be in an advantageous position to implement this approach since restorations to primary teeth need not be as durable as those made to permanent teeth.



Moreover, pediatric dentists use sealants extensively and are used to working, for example, with rubber dams, especially in young patients. It may turn out that after more research is completed on the ontogeny of the oral microbial flora, changes or ecological shifts made in childhood become lifelong. More specifically, drastic reductions or eliminations of cariogenic bacteria, along with their sites for coloniza-

tion in the primary dentition, may prevent or delay their transfer to the permanent dentition. Similarly, reductions in a mother's levels of cariogenic bacteria may delay or interrupt the transfer of cariogenic bacteria to her infant.

Here, the goal is to discuss several concepts before their final inception into practices and to stimulate discussion of ideas, reframing the design to meet the demands of everyday practice. Ultimately, the proof will come from a large clinical trial conducted in many practices and patient populations.

THE CONCEPT OF DENTAL CARIES being an infectious and transmissible disease was convincingly demonstrated by Paul Keyes,<sup>1</sup> then at the National Institute of Dental Research. Almost half a century later, clinicians and researchers alike have only begun to gain insight into the role and relative contribution bacteria make to the caries process. I will review some of the major trends, both practical and philosophical, proposed and practiced in this last half century, and then describe where I think dentistry is going next. It is not the intention of this thesis to give an exhaustive review of the literature. To do so would only highlight the contradictory nature of the present knowledge base. Many detailed and focused reviews of the literature are available. Some are more and some are less biased than what appears below.

After realization in the 1960s that specific microbes might be responsible for dental caries, focus was directed to controlling the microbial mass colonizing the teeth, dental plaque, euphemistically referred to as plaque control. Prior to that time, the *Lactobacillus* group was a focus of attention, later to be replaced in prominence by the mutans streptococci (MS). Plaque control became part of every dental practice, and brushing and flossing soon became household routines. Coupled with topical and systemic fluoride's reported anticaries effect, many people felt that the reign of caries was over, at least in the U.S. While we teach with almost religious furor and unquestioning rigor the holy triad of oral hygiene, regular<sup>2</sup> visits to the dentist and fluoride, many people can now make the argument that the actual effectiveness of these pillars of prevention are overstated and overly optimistic.<sup>1</sup>

One measure of the misconceptions and discordance in dental education and research circles can be found in the summary of polling of 55 experts as to why dental caries was declining.<sup>2</sup> No unifying consensus was found, but there were many different theories. What seemed evident was that the decline could not be attributed to any specific treatment or preventive measure; systemic fluoride, the obvious choice among most dentists, fared poorly when considering caries was declining in many countries without fluoride, Japan, for example.

### **Mechanical Approaches**

The first of the approaches to caries prevention I term mechanical. It makes sense that dentists would think mechanical; we are surgeons and we cut, debride and repair. Plaque control via brushing and flossing is an example of a mechanical approach. Fluoride's anticaries effect is thought by many to be mechanical in the sense that fluoride is alleged to make the enamel surface more resistant to acid demineralization. Other mechanical approaches include the handpiece and bur debridement of lesions. Like toothbrushing and flossing, it's the surgical way—scrubbing and debriding lesions. Ambroise Paré, the father of modern surgery, taught us that.

How effective are mechanical approaches for preventing caries? Flossing and brushing came up short, in study after study in terms of caries prevention. The World Health Organization report<sup>3</sup> gave disappointing results despite reports to the contrary by Axelsson and coworkers in Sweden.<sup>4</sup> Remember, most caries are

found on the occlusal surfaces, marginalizing both brushing and fluorides in terms of prevention because these two means are mostly ineffective on these surfaces.

Also arriving in the late '60s were the first sealants, which I will discuss in more detail later in this article. As a mechanical barrier, sealants are surprisingly underutilized and still remain controversial as to their overall effectiveness. A major part of the problem is they are not properly placed, that is, good etch, a moisture-free surface and adequate polymerization.

Overall, I think it's safe to say that a strictly mechanical approach to caries treatment and prevention falls short of addressing the bacterial etiology of the disease. It's somewhat puzzling why clinicians treating caries lesions do not disinfect the preparation before placing a restoration. Conventional restorative materials like amalgam are notoriously bad at leakage. Both bacterial culturing and scanning electronic microscopy show clearly the removal of decayed dentin falls short of removing bacteria within the dentinal tubules and at the caries front. Historically, even G.V. Black mentioned bacteria in dentinal tubules, recommending "toileting" the preparation prior to placing a filling; but this meant cotton and air spray to remove particles and moisture. I will revisit this concept later.

### **Antimicrobial Approaches**

The next phase of treatment regimens directed against the infectious disease aspects of caries was launched with great zeal in the 1970s. First introduced as a plaque-control agent by Loe, chlorhexidine, along with a long assortment of mouthrinses, grew in popularity. Agents used included antibiotics, chlorhexidine, iodine, zinc chloride, high concentrations of fluoride—including stannous fluoride and combinations of the above—among many others. (For reviews, see<sup>5,6</sup>) The idea was that both caries and periodontal diseases were bacterial infections. Accordingly, antimicrobials were applied that could reduce the numbers of bacteria, regardless of whether those bacteria were beneficial or benign to the host.

This "shotgun" approach to caries and plaque control continued for more than a decade. Intensive therapies were applied to children. These included SnF<sub>2</sub>, chlorhexidine gels and high concentrations of acidulated fluorides (high in HF and bactericidal to many bacteria). In perhaps the most intensive application of a chemotherapeutic agent to the dentition, accompanied by the most extensive microbiological survey of sites of re-colonization, Emilson and coworkers<sup>7</sup> applied a 1% chlorhexidine gel for 24 exposures to the dentition of young adults with extensive caries experience (DMFS = 34). Suppression of MS lasted for two to six months. The reappearance of MS was noted on the posterior teeth, especially the molars. Third molars were particularly difficult sites to disinfect, leading the authors to suggest that young adults without third molars would be more amenable to disinfection. These investigators also noted that the high number of restorations, which provided retentive sites, were a significant factor leading to the recolonization of these dentitions.

Another study reported the complete elimination of mutans streptococci, with the effect in some adults lasting two years.<sup>8</sup>

Others did not respond as well. The reason for this disparity will be discussed in the next section. Our own experience with iodine and chlorhexidine varnishes was that they failed to exert lasting effects on cariogenic bacteria when applied to prevent transmission of mutans streptococci from mothers to their infants.<sup>9-11</sup>

An antimicrobial approach to caries prevention and treatment has fallen short of its original expectations, probably for three reasons:

1. Antimicrobials are incapable of penetrating existing lesions, fissures and pits, or precarious or white spot lesions.
2. The shotgun approach toward indiscriminately eliminating bacterial populations from the oral cavity or from the dentition is not only difficult to sustain, but may not be in the best interest of the host. One of the key roles of the indigenous biota of the oral cavity is to exclude exogenous pathogens from colonizing. This is accomplished by physically occupying space or receptor to which pathogens can bind. In addition, many of the oral bacteria produce antimicrobial substances (e.g., bacteriocins) that are lethal to many types of bacteria, including pathogens.
3. Topically administered antimicrobials may be toxic not only to bacteria, but to humans. High doses of fluoride, for example, have led to illness and, in a few cases, to death. To avoid possible toxicity and for safety reasons, many antimicrobials used orally require dilutions that in many applications, such as mouthrinsing, result in very low antibacterial potency.

This is not to say that antimicrobials have no future. An overall evaluation of the use of antimicrobials in the literature suggested that this approach might have promise if used in a focused or targeted way, with a specific and measurable bacterial outcome or endpoint.<sup>12</sup>

Before discussing how the two methods of caries prevention and treatment can be combined in a more effective manner, it is worth mentioning the current thinking on designating one or a few members of the dental plaque as “odontogenic pathogens.” The leading candidate and most widely researched and characterized is the group of bacteria called the *mutans streptococci* (MS).

*Streptococcus mutans* is the member of MS most common in humans and most associated with disease. Decades of work have contributed to our collective knowledge of *S. mutans*, including its carriage rate, transmission and acquisition, physiological and genetic composition. Its importance to the research community was underscored when it was selected as a candidate for complete sequencing of its genome. In the insightful words of Coykendall, “this organism appears to have been intentionally designed from the ground up to be a cariogenic bacterium.”<sup>13</sup> Without a lengthy review of a massive amount of accumulated literature on the properties of *S. mutans*, it’s becoming clear that while it is an important contributor to dental caries, it is by no means the only cariogen. Plaque bacteria, forming what is called a biofilm, represent a

## *Is it possible to take the best attributes of the mechanical and antimicrobial approaches and merge them into a more effective way to treat and prevent caries?*

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diverse and compact community in which *S. mutans* is less than 1% of the total bacterial counts. We now think of dental plaque as a polymicrobial community of indigenous bacteria, just as the human body is made up of diverse cell types, each type with a specialized function or task.

In a classic study in schoolchildren, Burt and Loesche concluded that in the most common form of caries, fissure caries, “*S. mutans* was necessary but not sufficient.”<sup>14</sup> A recent survey of the composition of cariogenic plaque bacteria using genetic rather than culture medium argues against the notion that a single group of organisms is associated with caries.<sup>15</sup> New methods that use DNA markers are showing the presence of bacteria never cultured before—in fact, at least one-third of the bacteria in plaque have yet to be identified. So we need to conceptualize that caries results from a constellation of bacteria in the biofilm, not just as a mono-infection by a single or a few bacterial groups. This area of research is being hotly investigated and promises better delineation of the bacterial etiology of caries.

Let’s assume that the mutans streptococci and *Lactobacilli* are important contributors to the caries process. Certainly, these two groups are almost universally found in lesions. As caries progresses, *Lactobacilli* become more predominant, as they prefer a stagnant, retentive site to colonize and an existing lesion provides that site. The mutans streptococci require teeth to colonize, preferring retentive sites to smooth surfaces by an order of magnitude. Elimination of all retentive sites in the dentition would preclude colonization of these two cariogens—retentive sites include pits, fissures, white spot lesions, existing lesions, and margins around existing restorations.

Can this be achieved? Assume that dentists could somehow render the dentition “non-retentive” for these two cariogens. Could colonization of these two groups be inhibited or denied? Could modern materials using bonded composite resins and sealants render the dentition non-retentive? The answer may be “yes.”

### **Combined Mechanical and Antimicrobial Approaches to Caries Management/Prevention**

Is it possible to take the best attributes of the mechanical and antimicrobial approaches and merge them into a more effective way to treat and prevent caries? I think it can be done, but with some degree of caution because the proposal has not undergone the full test of efficacy, the Phase III clinical trial. Nonetheless, many of these procedures have been advocated and used successfully, especially in pediatric dental practices. Combining them could yield complementary and synergistic results.

For clarity, I will divide the treatment strategy into segments of common elements and approximate sequence in terms of patient care. I will not describe routine aspects of standard of care in terms of diagnostic and operative procedures.

### **Debridement**

Ideally, in a single visit, debridement of all open lesions would be achieved with rotary or hand instruments. The idea is to remove most of the carious dentin and make a retentive area for placing a bonded glass ionomer cement in the defects. Care must be taken to form a good seal across the pulpal floor. Contrary to what most of us were taught, removal of all carious dentin may not be necessary, and, in some cases, may compromise the vitality of the pulp.

Debridement and placing GIC as a provisional restoration allows for at least two effects: 1. lesions do not progress and the pulp is protected; 2. retentive niches harboring cariogenic bacteria have been removed and remaining bacteria sealed off from the oral cavity. Assuming carious dentin is the site of cariogenic bacteria, disinfect the preparation before placing GIC. Many oral antimicrobials are available. Except for the antimicrobial “toilet,” the above is a key principle of the ART approach.<sup>16</sup>

### **Disinfecting Procedures**

We don’t know if disinfecting or “toileting” the preparation will kill whatever microbes are left in the remaining dentin, but the idea is consistent with treating caries as an infection. I prefer either sodium hypochlorite (0.5%) or 2% iodine disclosing solution as oral antimicrobial agents, but others might use a chlorhexidine or Betadine solution. Placing a cotton pellet saturated with antimicrobial into the prepared lesion for a predetermined time, maybe 30 seconds, might reduce the microbial counts. Additional applications of antimicrobial solutions to the entire dentition before and after restoration/sealant placement could eliminate additional microbes on the tooth surfaces.

### **Placement of Sealants**

Occlusal fissures, pits and margins around existing restorations constitute the major ecological sites for colonization of two major cariogenic bacteria, the MS and the *Lactobacilli*. Sealing these sites would essentially change a retentive site into a smooth surface, hence, eliminating a retentive site for new colonizers and at the same time covering existing bacteria, cutting them off from the oral cavity. Sealing over existing amalgam and resin restorations is a proven way to eliminate retentive sites. Sealing proximal surfaces is possible with some attention to detail and use of orthodontic separators.

### **Final Restorations**

Bonded composite resins seal off the dentin from the oral cavity. Even amalgams can be bonded and sealed. Before final restorations are placed, however, the entire dentition should be disinfected and this process repeated with the idea that cariogenic bacteria will

either be killed or denied a retentive site to recolonize. Clinical studies will be needed to tell us how many times or how often disinfecting is necessary. In studies we conducted in the past, a 1% or 2% iodine disclosing solution was applied every other day for a week, then monthly, but the study design and patient population were unique for that study. If all the lesions, fissures, pits and margins are well sealed, one or two antimicrobial applications will probably suffice.

### **Expected Outcomes**

The main outcome from the proposed approach (or its final reduction to practice from clinical research) would be a shift in the oral bacterial ecology. A cariogenic flora would be replaced by a non-cariogenic flora. The reason an ecological shift would occur is because the dentition was altered to preclude the colonization of those cariogens that require the retentive sites to colonize. Further research will monitor the extent of this shift as well as its duration. Of course, the final proof of concept will be a decrease of dental caries; this proof will require at least another decade.

I encourage clinicians in practice and in academia to formulate their approach to caries control based not on the limitations of dentistry from the past, but on first asking what objectives they want to accomplish for their patients. If the answer is a healthy dentition with no or minimal restorative needs, then this approach may have some merit.

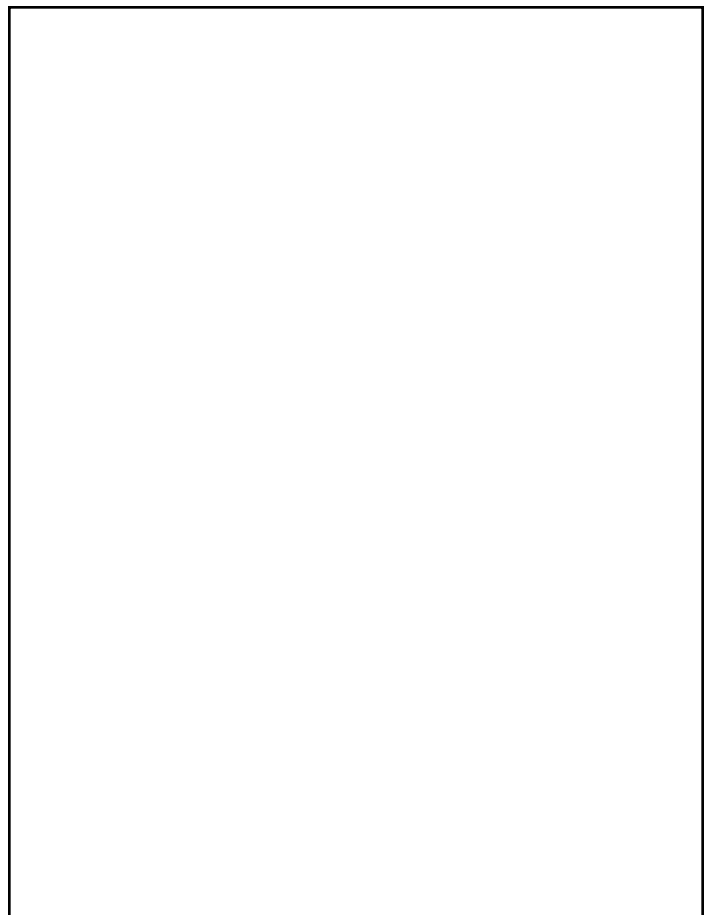
Although it is difficult to abandon some of the skills we learned early on in our dental schools, we now know many of those procedures were the best available based on the dental materials and equipment of the day. With modern composite resins, sealants, combined with our knowledge of the infectious components of the oral microbial flora, it seems prudent and reasonable to adopt our approach to caries control to the newer concepts. It's hard to do, I know, because of the difficulties we encounter everyday in changing our faculty's concepts of caries control and which are taught to our students.

Nothing I have written is really that novel or new. Indeed, I know pediatric dentists in this country and in other countries, such as Germany and Brazil, who practice along the lines outlined above. I think common sense would go a long way, even if to only question some of our existing beliefs and practices. The full-blown proof of a definitive approach is yet to be offered, but very few of the concepts of how we practice dentistry today have a scientifically proven basis or are evidence-based. This is changing, and we must change as well. ■

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# Oral Candidiasis in Pediatric HIV Patients

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## Abstract

Oral candidiasis can be an early sign of illness or disease progression in HIV/AIDS and other immuno-compromised states. Oral lesions associated with fungal infections present in a variety of forms, including a few of previously unknown etiology. Diagnosing these variants of disease can be challenging because of their atypical clinical presentation. Moreover, the emergence of new candida species, drug resistance and immature immune systems add to the complexity of this condition, especially in children.

Candida species reside as part of the normal flora of the oral cavity in about 40% of the general population known as carriers.<sup>1</sup> In the event of immune suppression, there is a shift from commensalisms to an exponential increase in colonization, which eventually leads to clinical signs and symptoms of oral candidiasis (OC).

OC in children has been less well studied, with only limited numbers of individuals examined. However, between 20% and 70% of children with HIV infection or AIDS have been reported to show clinical signs of oral candidiasis.<sup>1,2</sup> It is also the first infection to appear in approximately half of all HIV-infected children. Esophageal candidiasis, an AIDS-defining illness, according to the CDC, is reported to develop in approximately 20% of children. The combination of an immature immune system and suppressed cellular immunity provides optimal conditions for rapid disease progression. As a result, pediatric AIDS and associated infections have become a major challenge.

## Oral Manifestations

Oral candidiasis may be present as clinically distinct forms, including pseudomembranous, erythematous and hyperplastic variants. Other lesions, such as linear gingival erythema, median rhomboid glossitis and angular cheilitis, are now also believed to be associated with candidiasis.<sup>3,4</sup>

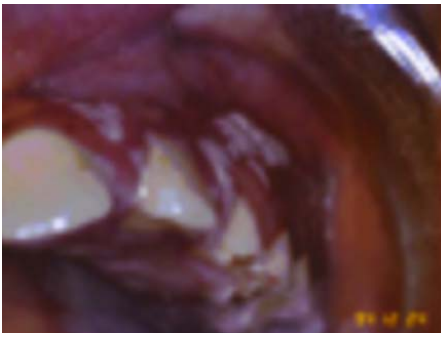
The pseudomembranous type (thrush) is characterized by the presence of creamy plaques on any part of the oral mucosa (Figure 1). The white plaques can be removed easily, and may reveal a superficially hemorrhaging surface at times. Common locations for infection include the palate, gingiva, buccal mucosa and dorsum of the tongue.

Chronic hyperplastic candidiasis, or candidal leukoplakia, presents as white patches on the buccal mucosa that do not rub off. This condition is least common and somewhat controversial, as it is believed to be a combination of candidiasis superimposed on a leukoplakic lesion. The diagnosis is confirmed by the presence of pseudo-hyphae upon microscopic smear examination and resolution of the lesion after anti-fungal therapy (Figure 2).

When the tongue is affected, patchy depapillated areas appear on the dorsal surface (Figure 3). The palate can also be affected secondarily because of its close proximity to the tongue lesion.

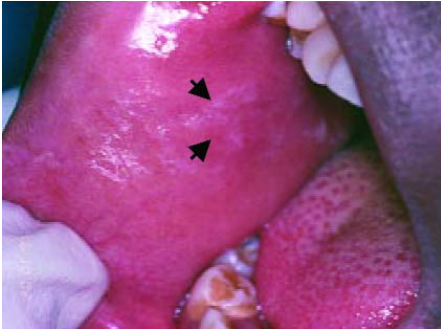
Median rhomboid glossitis, or central papillary atrophy, a condition with a previously uncertain etiology, has now been associated with candidiasis.<sup>4</sup> It is seen exclusively on the midline of the posterior dorsal aspect of the tongue and is asymptomatic. The erythematous mucosal changes are due to the loss of the filiform papillae, and the condition usually resolves following topical anti-fungal therapy.

Angular or labial cheilitis, characterized by fissuring at the corners of the mouth or lips, has also been implicated with a fungal origin and may appear either alone or in conjunction with any of



**Figure 1. Pseudomembranous candidiasis. Characterized by presence of white, creamy patches of plaque on oral mucosal surfaces.**

*(From New York Department of Health AIDS Institute's Clinical Guidelines Development Program Web site, [www.hivguidelines.org](http://www.hivguidelines.org).)*



**Figure 2. Hyperplastic candidiasis. Seen here as speckled white and erythematous areas that do not rub off.**



**Figure 3. Candidal glossitis. Characterized by central papillary atrophy of tongue.**

*Printed with permission: Neville BW, Damm DD, Allen CM, Bouquot JE. Oral & Maxillofacial Pathology, 2nd Edition. Philadelphia:W.B. Saunders, 2002.*

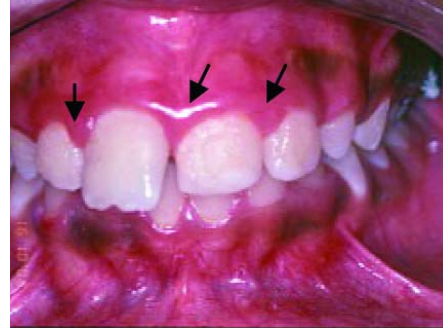
the other forms<sup>4</sup> (Figure 4). Chronic oral habits in children may spread this infection to adjacent sites, resulting in exfoliative lesions of the vermilion zone and perioral skin, creating a clinical pattern known as cheilocandidiasis.

Atrophic or erythematous candidiasis (EC) appears clinically as a red lesion. The color intensity may vary from fiery red to a hardly discernable pink spot and is usually without clinical symptoms. It often presents as mild, localized inflammation of the gums and can be easily mistaken for gingivitis (Figure 5). An accurate diagnosis in such cases is difficult and can be further complicated in the presence of plaque and poor oral hygiene. Recent studies utilizing microscopic examination of mucosal swabs of such lesions report an overall increase in the prevalence of erythematous candidiasis.<sup>5</sup> This is a shift from previous literature that suggests that pseudomembranous candidiasis is the most commonly occurring variant of oral fungal infections.<sup>6</sup> It is unclear whether EC has been misdiagnosed in the past or simply underreported in children.<sup>3</sup> There is also the possibility that perhaps EC lesions have a different clinical presentation in children than in adults.

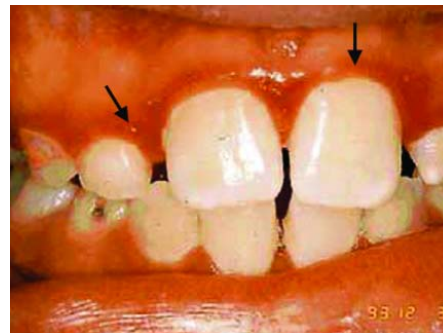


**Figure 4. Angular cheilitis. Seen as fissuring or hypo-pigmentation at angles of mouth. Fissuring can occur on lip as well (labial cheilitis) and resembles "chapped lips."**

*(From New York Department of Health AIDS Institute's Clinical Guidelines Development Program Web site, [www.hivguidelines.org](http://www.hivguidelines.org).)*



**Figure 5. Atypical erythematous candidiasis. Seen here as mild inflammation of maxillary anterior gingiva. Characterized by loss of stippling, mildly edematous and closely mimicking gingivitis.**



**Figure 6. Linear gingival erythema (LGE). Seen as erythematous band 2-3mm in width along free gingival margin.**

*(From New York Department of Health AIDS Institute's Clinical Guidelines Development Program Web site, [www.hivguidelines.org](http://www.hivguidelines.org).)*

HIV-associated gingivitis (HIV-G), a condition thought to be exclusive to HIV, has also been reported in other immunocompromised patients. It has since been renamed linear gingival erythema (LGE). As the name suggests, LGE is characterized by a linear erythematous band 2-3mm in width along the free gingival margin (Figure 6). Data from recent studies seem to suggest a fungal involvement that responds positively to anti-fungal therapy.<sup>3</sup> Thus, the term "HIV-associated gingivitis" may yet be renamed "HIV-associated candidiasis." Regardless, the result is there may be a significant number of children with an undiagnosed variant of oral candidiasis.

The big question is, what are the long-term effects of undiagnosed (therefore untreated) oral fungal infections on the course of HIV/AIDS itself? Would treating the oral infection offer the body a better chance to deal with the HIV? The answers to these questions are largely unknown, although logic dictates that treating these infections may provide the body with a better chance of coping with the HIV. After all, morbidity and mortality from HIV infection are not because of the virus per se but result from the ensuing opportunistic and secondary infections.

## Speciation

The majority of candidal infections in children and adults are caused by *C. albicans*; however, reports of the emergence of other novel species of candida have begun to appear.<sup>7,8</sup>

Masia Canuto, et al. recently evaluated 153 HIV-positive patients and found that 21% of these patients had non-*C. albicans* species. Similarly, Morace found that 25% of yeast species isolated from individuals with AIDS were non-*C. albicans* species, the most common being *C. dubliensis*, *C. glabrata* and *C. krusei*. Recent studies in children show that some species, exclusive to HIV/AIDS, have the ability to develop stable resistance to fluconazole in vitro and have significantly higher levels proteinase activity, a property considered a virulence factor in fungi.<sup>8</sup>

## Management

When signs and symptoms of candidiasis are atypical, diagnosis is confirmed by exfoliative cytology, culture of the specimen obtained from the lesion and by a positive response to anti-fungal therapy.

Treatment of oral candidiasis in children involves the use of antifungal drugs, such as 1:500,000 nystatin suspension rinse. Clotrimazole oral troches may be prescribed for older children; the tablets are sucked five times a day. Candida can be resistant to all

these forms of therapy, and even when responsive, may reappear soon after therapy is completed.<sup>7,9</sup>

Candidal esophagitis usually requires hospitalization and intravenous therapy with amphotericin B. Drug resistance is often an undesirable outcome of therapy, and the goal of the dentist should be to develop preventive strategies, such as periodic regimens of chlorhexidine and fluoride rinses. Chlorhexidine has been shown to diminish the mucosal adherence of fungi, thereby eliminating the onset of infection.<sup>10</sup> In addition, an aggressive recall/monitoring system should be developed for individual patients based on oral findings and the use of immunological markers to identify patients at risk.

The need for anticipatory guidance and communicating closely with the child's guardian and physician is paramount. Based on the immune status, pediatric HIV/AIDS can be classified into three categories. An immunological profile for each category has been adapted from the CDC and is presented in Table 1.

## Discussion

Increased oral carriage of *C. albicans* is a common finding in HIV-infected patients. In a study done by Korting, "the microbiological recoveries of oral *C. albicans* from 62 HIV-infected adults were 57.5% for CDC stage I patients, 76.5% for stage II patients and

**TABLE 1**

HIV MARKERS	VIRAL LOAD (HIV RNA) ¶ - According to the CDC		CD4+ COUNT AND PERCENTAGE * - According to the CDC					
	Viral Load (< 30 months of age)	Viral Load (> 30 months of age)	CD4 + Absolute Count (< 12months)	CD4 + % (< 12 months)	CD4 + Absolute Count (1-5 years old)	CD4 + % (1-5 years old)	CD4 + Absolute Count (6-12 years old)	CD4 + % (6-12 years old)
<b>Category 1 No suppression</b>	< 1000 – 150,000 copies/mL	< 1000 – 15,000 copies/mL	≥ 1,500 cells/ µL	≥ 25%	≥ 1,000 cells/ µL	≥ 25%	≥ 500 cells/ µL	≥ 25%
<b>Category 2 Moderate suppression</b>	150,001 – 1,700,000 copies/mL	15,001 – 150,000 copies/mL	750 – 1499 cells/ µL	15%-24%	500 – 999 cells/ µL	15%-24%	200-499 cells/ µL	15%-24%
<b>Category 3 Severe suppression</b>	> 1,700,000 copies/mL	> 150,000 copies/mL	< 750 cells/ µL	< 15%	< 500 cells/ µL	< 15%	< 200 cells/ µL	< 15%
	CD8+ COUNT §		NEUTROPHIL COUNT § ANC COUNT		¶CDC: Guidelines for the Use of Antiretroviral Agents in Pediatric HIV Infection. MMWR 1998; 47 (no. RR-4): 1-31. Palumbo PE, Raskino C, Fiscus S, et al. Disease progression in HIV-infected infants and children: predictive value of quantitative plasma HIV RNA and CD4 lymphocyte count. JAMA 1998;279:756-61. *CDC: 1994 Revised classification system for HIV infection in children less than 13 years of age. MMWR 1994; 43(no. RR-12): 1-10. §Sandberg JK, Fast NM, Jordan KA, Furlan SN, Barbour JD, Fennelly G, Dobroszycki J, Spiegel HM, Wiznia A, Rosenberg MG, Nixon DF. HIV-specific CD8+ T cell function in children with vertically acquired HIV-1 infection is critically influenced by age and the state of the CD4+ T cell compartment. J Immunol 2003 Apr 15;170(8):4403-10. §A Guide for Patients by Buchanan, 1994, Children's Medical Center of Dallas. <a href="http://public2.bcm.tmc.edu/pediatrics/documents/4120.pdf">http://public2.bcm.tmc.edu/pediatrics/documents/4120.pdf</a> David A J Moore, Ann Sullivan, Paul Hilstead, Brian G Gazzard. A retrospective study of neutropenia in HIV disease. Int J of STD & AIDS, Volume 11, Number 1 (January 1, 2000), pp. 8-14.			
HIV MARKERS	Strength of CD8+ T cell response (SFC/ million CD8+ T cells)		(Absolute Neutrophil Count)					
<b>Category 1 No suppression</b>	400 SFC/million CD8+ T cells (correlates to <400 CD4 count)		> 1500 cells/ mm <sup>3</sup>					
<b>Category 2 Moderate suppression</b>	1000 SFC/million CD8+ T cells (correlates to 400-1500 CD4 count)		500-1500 cells/ mm <sup>3</sup> (mild neutropenia)					
<b>Category 3 Severe suppression</b>	300 SFC/million CD8+ T cells (correlates to >1500 CD4 count)		< 500 cells/ mm <sup>3</sup> (infection can occur)					

87.5% for stage III patients.<sup>11,12</sup> Similarly, Fong, et al. found yeast carriage rates in HIV-positive individuals to be 67% for those with CD4-cell counts above 500 cells/ $\mu$ l, 86% for patients with counts of 200 to 500 cells/ $\mu$ l and 82% for patients with CD4-cell counts below 200 cells/ $\mu$ l.<sup>11,13</sup>

Investigators have also demonstrated an increase in the intensity of candidal carriage in immunocompromised patients. The intensity of carriage is a quantitative measure of candidal species in the oral cavity. Its sensitivity can allow for early detection of infection even prior to the onset of mucosal changes. Normal values range from 350 to 400 colony forming units (CFU) per ml of saliva and show an increase with disease progression.<sup>11</sup> Therefore, there has been considerable interest in recent years in using candidal carriage and intensity of carriage as early markers for disease progression in immunocompromised patients.

Although the basic pathogenesis of HIV infection is the same for adults and children, some laboratory findings are unique to children. For example, a CD4 count alone is not as reliable a marker of disease status because children tend to have higher and less consistent CD4 levels compared to adults, and a low CD4 count is often a late finding.<sup>14,15</sup> There also are profound alterations in T-cell assays, hence, susceptibility to infections cannot always be correlated with immunological markers.

Because oral fungal colonization remains one of the most common opportunistic infections observed in both adult and pediatric HIV patients and is usually the first to appear, it can have a prognostic value<sup>16</sup> independent of CD4 status or other more commonly used immunological markers. Interestingly, two AIDS-defining conditions of pneumocystis carinii pneumonia and intestinal lymphoma were associated with pseudomembranous oral candidiasis in two case reports.<sup>16</sup>

## Significance

Standard antibody testing is now available to determine an individual's HIV status at an early age. However, because of the expense of complex technology, health workers in developing countries, "where 95% of the world's pediatric AIDS cases are found," must rely on simple, inexpensive and early clinical tests for HIV infection.<sup>3</sup> The monitoring of candidal carriage and colonization by dentists and other trained clinicians can lead to early diagnosis of HIV infection in both child and mother, or serve as a marker for disease progression.

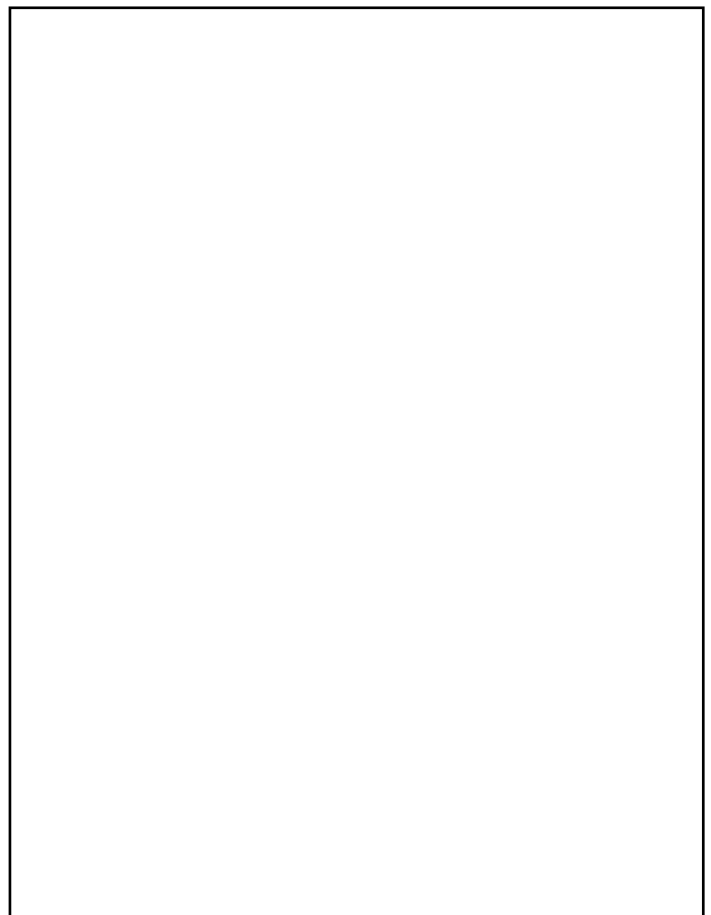
Furthermore, prevention or early treatment of opportunistic oral infections may provide a better prognosis for the HIV infection, leading to an overall sense of well-being for these children with very special needs. ■

*The authors thank Derek Park for his assistance with this article.*

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# Contemporary Space Maintenance for the Pediatric Patient

*Renuka R. Bijoor, D.D.S., M.D.S.; Kavita Kohli, D.D.S.*

## Abstract

Primary teeth play a critical role in the growth and development of a child. In addition to their role in esthetics, eating, speech, and to encourage normal function and resultant expected growth, the other main function of a primary tooth is to hold space for the permanent successor until it is ready to erupt. Space maintenance forms an integral part of preventive and interceptive orthodontics. After the premature loss of a tooth, not only do space maintainers maintain function and preserve arch length,<sup>1</sup> they also maintain esthetics and eliminate any potential psychological damage a child could face as a result of the premature loss of teeth.<sup>2</sup> The space maintainer also allows the permanent tooth to erupt unhindered into proper alignment and occlusion.

LOSS OF SPACE in the primary and mixed dentition is a fairly common presentation. Early interception would provide a great service to the patient by avoiding or, at the least, limiting the length and extent of comprehensive orthodontic treatment needed later,<sup>3</sup> and sometimes even surgical intervention. Causes of loss of space are multifold. They include: premature loss of teeth (traumatic, idio-

pathic or as a result of extractions); nonrestored proximal carious lesions; congenitally missing teeth; ankylosis; infraclusion; and ectopic eruption. The key points are diagnosis of a potential space loss and identification of a need for prevention or intervention.

Diagnosing space problems and treating cases of mild, moderate and severe crowding require an understanding of the etiology of crowding and the development of the occlusion stage, as presented by Ngan, et. al.<sup>4</sup>

In addition to severe crowding and a high caries index, another obvious contraindication for use of a space maintainer would be when the permanent tooth is very close to eruption and appears to be very close to the crest of the alveolar ridge. Dean, et al.<sup>3</sup> advise using the amount of bone over the tooth as a guideline; erupting premolars need four to five months for every mm of bone as measured on a bite-wing radiograph. The loss of primary anterior teeth does not indicate the need for a space maintainer unless the patient or parent requests one for esthetic purposes.<sup>5</sup>

Space is maintained in the maxillary arch by the interlocking of the opposing arch canines.

Devising a treatment plan should first and foremost address elimination of the etiologic factor. Some authors advise that it is important that diagnostic records are taken for the study of the case using an arch length analysis (eg., the Nance analysis, the Tanaka-Johnston analysis or the Moyer's mixed-dentition analysis) for defining the problem.<sup>3</sup> If a significant discrepancy is noted, the clinician must then assess where the tooth mass problem is and decide the best method to resolve it. Space maintainers can be clas-



Figure 1. Lower pedo partial.



Figure 2. Upper pedo partial.



Figure 3. Removable lower lingual arch.



Figure 4. Band and loop.

sified broadly by their various applications,<sup>7</sup> but mainly they are removable or fixed.

### Removable Space Maintainers

Removable space maintainers are relatively easy to fabricate, require less chairside time, and are relatively inexpensive, compared to fixed space maintainers. However, they have limited use in pediatric dental practice because of inadequate compliance by the patient—and sometimes even the parent—with regard to their use and care. Indeed, they have the advantage that they can be removed and cleaned easily, potentially eliminating the possibility of caries or soft-tissue inflammation.

Removable space maintainers are always bilateral, and in both the upper and lower arch would mainly constitute the “pedo” partial denture<sup>6</sup> (Figures 1 and 2). Components of the “pedo” partial denture are an acrylic major connector, with retentive features consisting of clasps (“C”/ Adam’s/ ball clasps or variations) and an arch wire, and for further retention, other features such as occlusal rests or direct bonded buttons. As the permanent teeth erupt, the acrylic pontics must be removed with an acrylic bur.

Certain cases warrant the use of a removable lower lingual arch (Figure 3), as, for example, in the caries-prone patient. This appliance requires special brackets to allow easy removal for adjustments or for adding attachments.

### Fixed Space Maintainers

Although relatively more expensive, fixed appliances decrease the need for patient and parent compliance. They also are less bulky, less cumbersome for the patient to accept and manage, and require less regular care. With the fixed appliance, vigilance is critical through regular follow-up, to ensure the removal of the appliance in coordination with eruption of the permanent successor.

The cements that can be used for luting these appliances are zinc phosphate, zinc polycarboxylate, ZOE, glass ionomer, resin-modified glass ionomer, compomer and resin cements.<sup>6</sup> Glass ionomer cements, in particular, have useful fluoride-releasing properties.

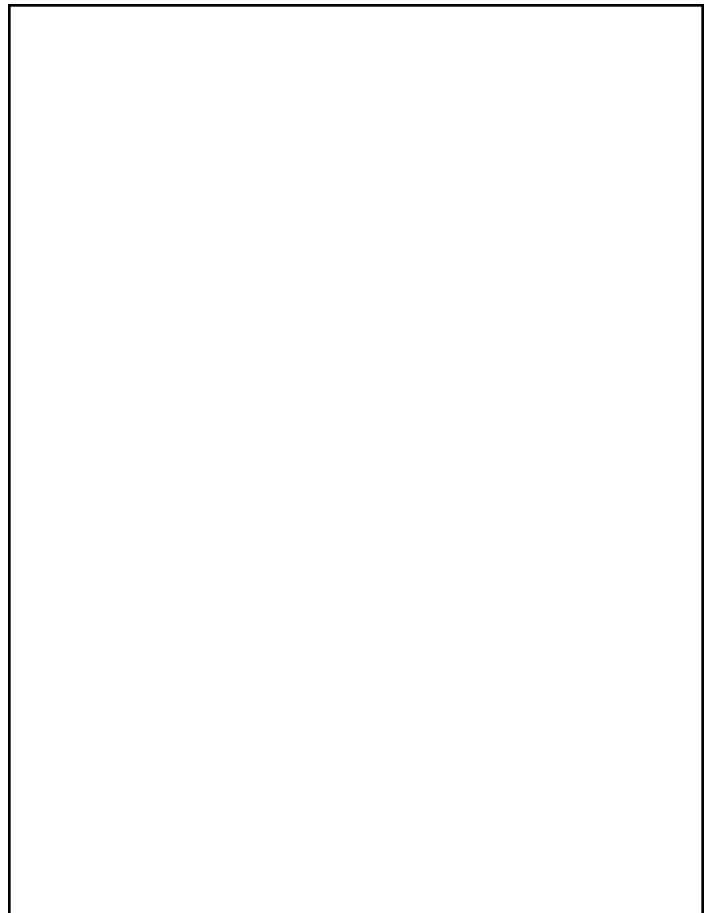
Fixed appliances can be classified as unilateral or bilateral, maxillary or mandibular.



Figure 5. Crown and loop.



Figure 6. Distal shoe.



*Given the issue of compliance and the dynamics of the mixed dentition, the longevity of space maintainers is not ideal.*

### **Unilateral Fixed**

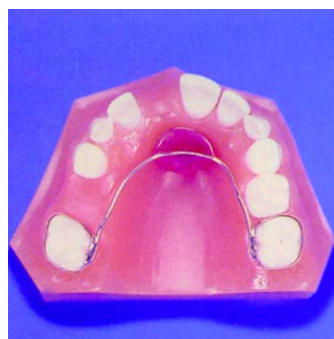
These space maintainers are in either arch and are mainly:

- **Band and Loop Appliance (Figure 4)**—The band and loop can be used after the loss of the primary first or second molar. The major components of this appliance, as the name signifies, are a band on the abutment tooth and a loop that traverses the edentulous space and is wide enough to allow eruption of the succedaneous tooth. This appliance can have various modifications, such as the use of an occlusal rest to prevent mesial tipping of the adjacent tooth. This rest could also be bonded into position for added retention. It may be modified for a long span by utilizing two bands and a buccal bar with a loop, especially when the paths of insertion of the abutment teeth are not parallel. Adjustment of the loop also helps to regain a small amount of space. There could be further modifications, such as using an occlusal bar to prevent supra-eruption of opposing teeth or an occlusal pad to provide a functional surface for the opposing teeth.
- **Crown and Loop Appliance (Figure 5)**—The crown and loop can be used when the abutment tooth has inadequate tooth structure, or has been restored after a pulpotomy, the crown being either fitted or provided separately with the impression by the dentist.
- **Distal Shoe Appliance (Figure 6)**—The distal shoe appliance is used after the loss of the primary second molar to prevent the mesial migration of the erupting permanent first molar. It uses a single band with a distal extension beneath the tissue to contact and guide the erupting tooth.

### **Bilateral Fixed**

These space maintainers can be classified as:

- **Maxillary**—There are mainly two appliances that help to maintain the position of the maxillary molars with retention via bands on the maxillary first permanent molars, and one appliance to replace anterior teeth. These are:
  1. The Nance Holding Arch (Figure 7): This appliance maintains maxillary arch length when one or more of the primary molars are lost. The acrylic button on the palate is to add stability; however, there is skepticism about the use of this button as it may cause some degree of palatal mucosal irritation, especially when oral hygiene is poor.
  2. The Transpalatal Bar (Figure 8): The transpalatal bar consists of a wire attached to bands on the permanent first molars. This wire is adapted to the contour of the palatal vault, is comfortable and does not interfere with speech or oral hygiene.



**Figure 7. Nance holding arch.**



**Figure 8. Transpalatal bar.**



**Figure 9. Groper partial.**



**Figure 10. Lower lingual holding arch.**

3. Groper Fixed Anterior Bridge (Figure 9): When esthetics are a prime concern, an anterior fixed bridge is provided to replace the missing incisors. The appliance consists of a lingual arch wire, which is attached to bands on the primary second molars. In the anterior region, the arch wire is a broader band, to which individual teeth are soldered. Indeed, the esthetics and the absence of an inconvenient removable appliance are great advantages of these appliances.

- **Mandibular**—The lower lingual holding arch (LLHA) (Figure 10) prevents mesial migration of the permanent molars and lingual tipping of the anterior teeth, which would cause the arch to collapse. The appliance is simple and consists of a wire soldered with bands fitted on the permanent first molars. The appliance could have distal stops, to prevent distal migration of anterior teeth, placed anywhere along the arch wire. This appliance cannot be fabricated for a patient with the four lower permanent incisors unerupted. If placed prior to eruption of the permanent incisors, the lingual arch will block the eruption and the labial migration of those teeth into the arch.

### **Longevity of Space Maintainers**

Given the issue of compliance and the dynamics of the mixed dentition, the longevity of space maintainers is not ideal. In a recent study by Rajab,<sup>8</sup> the overall performance was 30.7% failed, and

19.9 % lost. The mean survival time was 18 months, with the lingual arch showing the lowest median survival time and the Nance the highest. Solder breakage was the most common cause of failure, followed by cement loss.

In a similar study of 190 space maintainers by Qudeimat and Fayle,<sup>9</sup> 9.36% of failures were due to cement loss, 24% to breakage, 10% to design problems, and 9% were lost. The researchers discovered that band and loop space maintainers had the longest mean survival time and that unilateral space maintainers survived longer than bilateral ones.

## Modifications

There have been numerous articles in recent literature with ingenious designs of fixed space maintainers, such as using a modified bonded bridge space maintainer,<sup>10</sup> a nickel titanium space regainer-retainer<sup>11</sup> and, more recently, a glass-fiber reinforced composite resin space maintainer.<sup>12</sup>

As an interim restoration after loss of the permanent first molar in the mixed dentition, the construction of a functional and cost-effective stainless steel crown bridge using a rectangular mesh made of orthodontic wire soldered to stainless steel crowns on abutment teeth and a resin tooth as a pontic has been described.<sup>13</sup> In order to maintain occlusion in the mixed dentition, it has been suggested that the preformed stainless steel crown could be an interim restoration for a permanent tooth until a more desirable full cast crown is possible.<sup>14</sup>

Certain specific situations warrant other methods of treatment. Congenitally missing lateral incisors have always posed a challenge to the general dentist, pediatric dentist and orthodontist. Whether to close, open or maintain the space is an orthodontic decision based on various factors, says Wexler.<sup>15</sup> He lists these factors as: skeletal base relationship, severity of crowding, overjet, overbite, position unto which the canine erupts, crown sizes, crown shapes, gingival contours, gingival display on smiling, patient and parent attitude towards options, availability of appropriate prosthetic services, time delay before “final” prosthetic treatment, space and bone required for implant fixtures, and financial and biological costs.

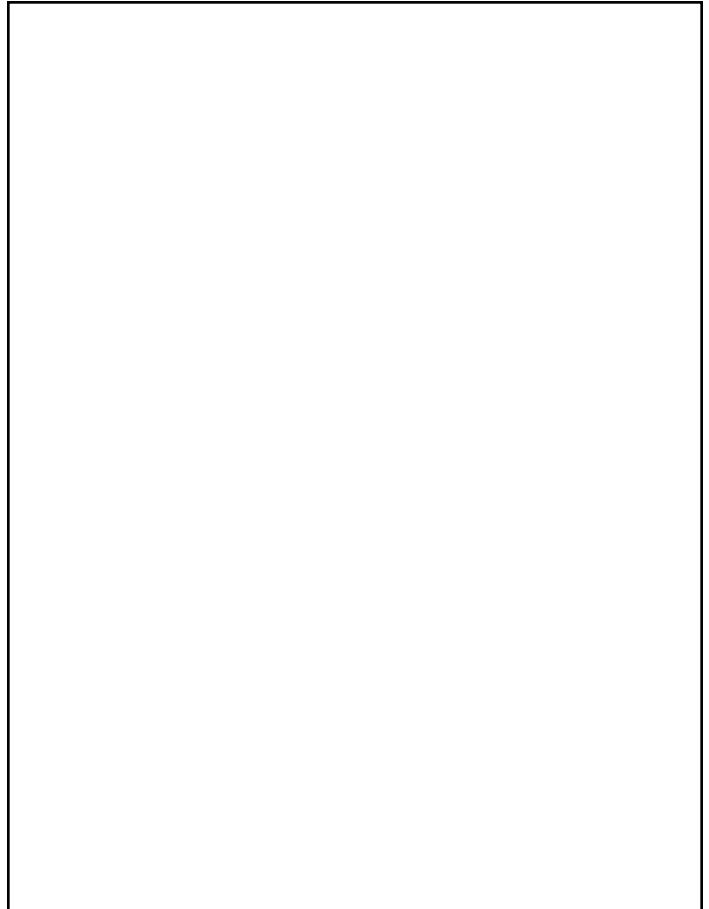
Gerald R. Dickinson<sup>16</sup> has discussed the challenge posed by young patients who are congenitally missing lateral incisors and have received orthodontic treatment to maintain this space until placement of implants when skeletal growth has ceased and soft tissue has matured.

The treatment of the potential or actual loss of space in the mixed dentition is essential to maintaining the health and balance of the developing dentition and facial structure. Timely intervention can, thus, be a true service. Any of these forms of treatment can be applied as suited when a case that needs space management presents. ■

*The authors thank Space Maintainer Laboratory for allowing them to use the illustrations that appear with this article.*

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# Root Fracture

## Report of Case Supporting Decreased Splint Times

*Joseph McManus, D.M.D., M.S., M.H.A., M.B.A.; Martin J. Davis, D.D.S.*

### Abstract

This is a case presentation of a root fracture that was treated and followed intermittently for 23 years. The treatment outcome supports the newly evolving protocol that root fractures are most successfully treated using a flexible splint over a relatively short duration.

TRAUMATIC INJURY to the oral facial region of children is a common occurrence. The peak incidence of injury is between the ages of 2 and 4, and 8 and 10. Statistics reveal that 30% of children suffer a traumatic injury to the primary dentition and 22% suffer trauma to the permanent dentition by age 14.<sup>1</sup> Continuous attention in the dental literature and research centers on best practices for the care of traumatic injuries. However, one type of oral injury that is relatively uncommon and is a source of varying recommendations is root fracture.

The prevalence of root fractures varies between the primary and the permanent dentition. Fractures of permanent tooth roots are far more frequent given the much higher bone density and resistance to whole tooth displacement. Root fracture rates range between 2% for the primary dentition and 7% for the permanent dentition.<sup>2</sup> The most affected age range is between 3 and 4 years for deciduous teeth and 11 and 20 years for permanent teeth.<sup>3</sup>

The recommended management of root fractures has evolved over the last two to three decades. In 1974, McDonald

recommended that the fractured root be stabilized minimally for three to four weeks.<sup>4</sup> In 1981, Andreasen recommended absolute immobilization, with rigid fixation for two to three months.<sup>5</sup> This longer stabilization is then echoed in McDonald's more recent editions.<sup>6-9</sup>

Both Andreasen and Camp recommended a relatively long stabilization period for teeth with fractured roots. In particular, Camp, a pediatric dentist and endodontist, historically recommended 10 to 12 months.<sup>7</sup> Mc Donald believed that the application of a rigid splint was conducive to calcified tissue repair. He recommended the use of a heavy wire (.032-.036) when stabilizing a tooth with a fractured root.<sup>8</sup> However, recent studies by Cvek and Andreasen cast doubts on the necessity, efficacy and advisability of long-term, rigid splinting.<sup>9</sup> Untoward outcomes of splinting, which clearly increase with rigidity and with time of fixation, include both inflammatory and replacement resorption. In a functional anatomical sense, normal callous formation, mineralization of that callous, and concomitant periodontal repair with normal filler group orientations at different locations all occur best with slight function occurring.

### Case Presentation

A 10-year-old healthy female presented on an emergency basis with trauma to the permanent maxillary right central incisor (#8). The patient had suffered a face forward fall in her backyard two days previously, striking a hard object and causing the dental injury. The patient had no untoward medical or dental history.



**Figure 1. Baseline and date of first appearance radiograph.**



**Figure 2. Radiograph at 10 days.**



**Figure 3. Radiograph at 6 months.**



**Figure 4. Radiograph at 14 months.**



**Figure 5. Radiograph at 17 years.**

Oral examination of #8 revealed tooth mobility of 1.5 out of 3.0. There was neither soft tissue laceration nor evidence of other dental trauma. A baseline periapical radiograph (Figure 1) revealed a mid-root, horizontal fracture of the root with the coronal and apical segments in acceptable apposition. Baseline electric pulp testing revealed a vital, non-hyperemic pulp, using #10, a clearly non-traumatized, contra lateral tooth as a reference. There was no evidence of any occlusal interference on this incisor.

The tooth was stabilized with an etch-retained labial wire using a light .016 wire. The splint time was limited to four weeks. At the time the wire was removed, the tooth had a physiologic plus- $\frac{1}{2}$  mobility. In addition, an electric pulp test registered within normal limits, again using the maxillary left lateral incisor as a reference. Radiographs of the tooth were taken at date of first appearance (Figure 1), at 10 days (Figure 2), at 6 months (Figure 3) and at 14 months (Figure 4); these revealed no periapical pathology, a periodontal ligament of normal width and a visible mineralizing repair of the fracture. Clinical and electric pulp tests remained negative during this interval.

Subsequent to treatment, the family relocated and the patient left the practice. All moved back to the area 17 years after the injury. Examination at that 17-year interval revealed no pathology in the maxillary right central incisor or supporting tissues. The tooth was of identical color and normal sensitivity upon percussion, and electric pulp testing compared to the reference teeth. The radiograph (Figure 5) showed excellent healing of the root fracture, which was now difficult to visualize.

The patient left the practice again, only to return to the area again at 23 years after the fracture. Oral examination was once again within normal limits. The patient refused any radiographs because she was pregnant. Intraoral photographs (Figures 6 and 7) were taken and showed no evidence of pathology.

## Discussion

In the case presented here, the root fracture was diagnosed by appropriate radiographs and mobility evaluation. In addressing the imperative to visualize the apex, it is not uncommon in patients



**Figure 6. Photograph at 23 years (mirror view).**



**Figure 7. Photograph at 23 years (anterior view).**

such as this one, with narrow, high vaulted palates, to obtain radiographs that are slightly foreshortened. Most important is consistency in film angulation for follow-up visits, to enable comparison.

Root fracture prognosis depends upon a number of variables, such as extent and length of the fracture line, fragmentation, apposition of segments and the overall health of the patient. Significant dislocation of the fragments, detectable pulpal hyperemia, high mobility of coronal segment, and traumatic occlusion are also very important factors.<sup>10</sup>

The type of splint used and the length of splinting time are additional considerations for healthy outcomes.

The fracture in the reported case had a number of favorable variables. The fracture line was simple and limited in length; the pulp was vital; the occlusion was atraumatic; and the patient was young and healthy. These conditions lead to a very favorable prognosis.

The management of root fractures with minimal complications and a strong prognosis should not dictate rigid fixation for a lengthy period of time.<sup>11</sup> Such aggressive treatment modalities are not indicated unless a compromised outcome is probable. The case that was presented lends support to a shortened fixation period with a physiologically flexible splint.

## Conclusion

Recent studies regarding the rigidity type and length of fixation of root fractures are helping us reassess and improve our treatment

protocols. This case supports newer, less-lengthy protocols for simple root fracture management. It is hoped that future studies and case reports will concur. ■

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## Fourth District Offers Ninth Dental Congress

THE NINTH ANNUAL SARATOGA DENTAL CONGRESS, "The Meeting at the Springs," will take place May 26-27 at the Saratoga City Center in Saratoga Springs. Again this year, the meeting will be held over two full days. The meeting is sponsored by the Fourth District Dental Society.

Thursday's program will feature Dr. Paul Belvedere, speaking on "Direct Composite Techniques Made Easy." Programs for dental hygienists, assistants and staff will include Mary Govoni, C.D.A., R.D.H., M.B.A., whose topic is, "How Ergonomics and Efficiency Impact Your Practice." For staff, there is, "Stop Telling Patients About Their Dental Insurance," presented by Tom M. Limoli Jr.; and "CPR Skills, First Aid and Automated External Defibrillators," an initial certification course, given by ERT Safety Training and Consulting.

The Congress will resume on Friday with more continuing education for the entire dental staff. On the program are: "Infection Control—Let's Get Real," a lecture by Derek K. Heing; "Recognize Childhood Development Delays and Habits of Effective Dental

Offices," by Ann-Marie C. DePalma; "Fabulous Office Systems," by Linda W. Drevenstedt; "Risk Management of the Dental Staff," by Frederick Wetzel; and "CPR Skills, First Aid and Automated External Defibrillators," a recertification course, by ERT Safety Training and Consulting.

Attendance at previous meetings has exceeded 1,000 persons. Registration will open at 8 a.m., with access to the Exhibit Hall. Courses both days will begin at 9 a.m. Fees for dentists are \$175 for one day, or \$275 for two days. For staff, fees are \$50 each day. Students and residents will be admitted free of charge. Coffee and pastries will be served in the morning, and lunch will be available both days. The chairman of the meeting is Fourth District President Thomas J. Milko.

For information on dentist and auxiliary registration, call Fourth District Dental Society Executive Director Robert C. Westcott at (518) 793-3160. For exhibitor registration, call Robert Sharp at (518) 793-5903. ■

## "The Meeting at the Springs"

City Center, Saratoga Springs, NY



Sponsored by Fourth District Dental Society  
Registration 8:00 am  
Exhibits open at 8:00 am

For more information, contact  
Fourth District Dental Society  
84 Glenwood Avenue  
Queensbury, NY 12804  
518-793-3160

### Plus

- ◆ Table Clinics
- ◆ Exhibitions
- ◆ Luncheons
- ◆ Raffles

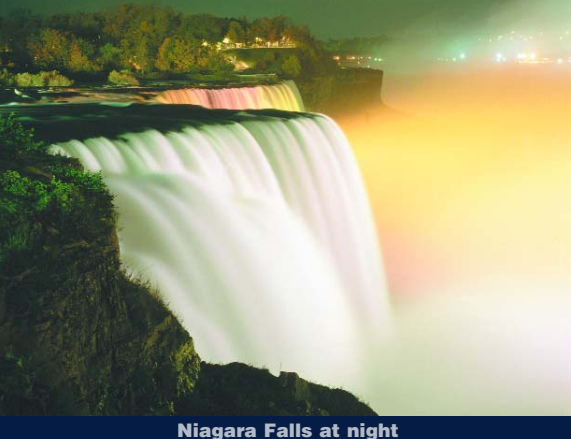
### Thursday, May 26, 2005

- ◆ Lecture – *Direct Composite Techniques Made Easy*  
*Dr. Belvedere*
- ◆ Lecture – *How Ergonomics and Efficiency Impact Your Practice*  
*Mary M. Govoni*
- ◆ Lecture – *Stop Telling Patients About Their Dental Insurance!*  
*Tom M. Limoli*
- ◆ "Hands-On Course" – ERT Safety Training & Consulting  
*"Initial Certification"*

### Friday, May 27, 2005

- ◆ Lecture – *Infection Control – Let's Get Real*  
*Derek K. Hein*
- ◆ Lecture – *Recognize Childhood Development Delays and Habits of Effective Dental Offices* – *Ann-Marie C. DePalma*
- ◆ Lecture – *Fabulous Front Office Systems*  
*Linda W. Drevenstedt*
- ◆ Lecture – *Risk Management of the Dental Staff*  
*Dr. Frederick Wetzel*
- ◆ "Hands-On Course" – ERT Safety Training & Consulting  
*"Recertification"*

*Continuing Education for the Entire Staff*



Niagara Falls at night



Bass Fishing



Shea's Marquee



## NYSIDA Semi-Annual Meeting Moves West

THE EIGHTH DISTRICT DENTAL SOCIETY will host the 2005 NYSDA Semi-Annual Meeting June 9-12 in Buffalo. The meeting will celebrate the presidency of Lawrence E. Volland, D.D.S., a Lockport general dentist, who has been serving as NYSDA President since January. Dr. Volland and his fellow officers will be installed at a formal Dinner Dance on the evening of Saturday, June 11. Standing up with Dr. Volland will be President Elect Alfonso J. Perna, Vice President Steven Gounardes, Secretary-Treasurer John P. Asaro and Past President Brian T. Kennedy.

The base of operations for the meeting is the Buffalo/Niagara Marriott in suburban Amherst. But a variety of social and recreational events have been planned to take advantage of and show off some of Western New York's many natural and manmade attractions. Descriptions of these events and locales are presented here.

The Board of Governors will hold its opening session from 2:30-4:30 p.m., Thursday, June 9. It will continue deliberations on Saturday during an all-day session that will begin at 9 a.m. Throughout the meeting, the New York State Dental Foundation will offer continuing education for both dentists and their staffs. Complete descriptions of courses and presenters appear in the February NYSDA News, as well as on the NYSDA Web site, [www.nysdental.org](http://www.nysdental.org).

Among other events planned for NYSDA members during their stay in Buffalo is the annual luncheon of the New York State Chapter of the Pierre Fauchard Academy, which will take place on Saturday.

Arrangements for the Semi-Annual Meeting are being coordinated by this year's Annual Meeting Chair Joseph Accardo. Requests

for information should be made to NYSDA Assistant Executive Director Beth Wanek at (800) 255-2100.

### Out and About in Buffalo

Semi-Annual Meeting events are casual, formal and purely relaxing.

**COCKTAILS AT THE BRINK** (*Thursday, June 9*): The first social event of the meeting starts with a trip to Goat Island, located between the American and Canadian Falls, where a cocktail reception will be held from 7 – 10 p.m. at the Top of the Falls Restaurant, noted for its spectacular view of the Horseshoe Falls. A short ride away, ADA dignitaries will officiate at the dedication of a plaque commemorating the founding of the American Dental Association in Niagara Falls in 1859. All are invited to attend. Attendees will also be free to roam and see the sites of Niagara Falls, or try their luck at the Seneca-Niagara Casino. Continuous shuttle service between venues will be provided. Buses will depart Niagara Falls at 10 p.m. and return to the Marriott by 10:30 p.m. Our thanks to the Niagara County Dental Society, Ivoclar Vivadent, Inc., and Benco Dental Co. for underwriting the cost of this event. *Fee: \$35 per person.*

**GOLF TOURNAMENT** (*Friday, June 10*): NYSDA's annual Golf Tournament will be held at Lockport Town & Country Club, just a short ride from the Marriott. One of the most scenic and challenging golf courses in the area, this 6,600-yard course has been host to the USGA qualifiers, Western New York PGA tournaments, New York State championships, Buffalo district titles and more. This is a scramble



Waterfront



Fuzak Garden

tournament. Shotgun start is at 8:30 a.m. Soft-spiked golf shoes are required. Entry fee includes greens fees, cart, Continental breakfast at 7:30 a.m., lunch and prizes. Transportation will leave the Marriott at 7 a.m. Please make sure you list your handicap or your 18-hole average score on the event registration form. Our thanks to NYSDA Support Services, Inc., Sullivan-Schein, Dental Dynamics, Graser's Dental Ceramics and Patterson Dental for underwriting the cost of this event. *Fee: \$100 per person.*

**TROPHY BASS FISHING** (*Friday, June 10*): Join us for a day of small mouth bass fishing on Lake Erie during "trophy bass season," which is held prior to the opening of regular bass season. Typically, a good number of bass, whose size and fight will impress even the most avid fisherperson, are caught. And it is not uncommon to catch multiple small mouth bass in the 4-6 pound range! Each person is allowed to keep one fish for a "trophy mount." Rod, bait and tackle will be provided. Transportation departs the Marriott at 6:15 a.m. and returns at 2 p.m. Box lunches provided. Our thanks to Liberty Mutual Insurance Co. for underwriting the cost of this event. We need to reserve fishing boats as early as possible, so, if you plan to sign up for this event, call Beth at NYSDA Headquarters (1-800-255-2100) ASAP!

*Fee: \$125 per person.*

**LOCKPORT LOCKS & ERIE CANAL CRUISE** (*Friday, June 10*): This unique two-hour cruise includes "locking through" and being raised the 49-foot elevation of the Niagara escarpment in the only double set of locks on the Erie Canal. You will pass under bridges that rise straight up, see water cascade over Lockport's famous "Flight of Five" 1840's locks, and travel through the solid walls of the "rock cut," as your captain describes the architecture and sights along the way. He'll also throw in some folklore for your entertainment. Be sure to bring your camera. Rain or shine, you'll enjoy this cruise. Lunch follows at the Canalside restaurant. Your tour will conclude with a short visit to the nearby Market Street Art Center, a complex where you'll find original artwork, prints, note cards, pottery and jewelry, all produced by local artisans. Bus departs the Marriott at 9:30 a.m. and returns at approximately 3 p.m. Thanks to the Tri-County Dental Society for underwriting the cost of this event.

*Fee: \$35 per person.*

**JAMAICAN BEACH PARTY** (*Friday, June 10*): That's right, a little bit of Caribbean in Western New York! Friday night's gala is a

Jamaican Beach Party/Bar-B-Que at Beaver Island State Park. Move to the rhythm of a cool West Indies island band while sipping Red Stripe beer or rum punch at the tropical bar. You will dine from an authentic Jamaican buffet, grilled on the beach as the sun sets over the Niagara River. Transportation will be provided. Buses depart the Marriott at 6:30 p.m. and will return at approximately 10:30 p.m. Our thanks to the Eighth District Dental Society, Erie County Dental Society, the Lockport Dental Society, and Ivoclar Vivadent, Inc., for generously defraying the cost of this event.

*Fee: \$35 per person.*

**YOGA WAKEUP** (*Saturday, June 11*): Wake up your mind, body and spirit with a gentle yoga class. Whether or not you have practiced yoga, this one-hour session that begins at 7:30 a.m. will leave you energized and refreshed for the day. Certified Himalayan Institute Instructor Karen Muscarella will guide you through a series of gentle postures and relaxation techniques to rejuvenate your body, calm your mind and lift your spirit. Wear comfortable clothing. Mats are provided. A light, healthy breakfast will follow.

*Fee: \$20 per person.*

**ALLENTOWN ARTS FESTIVAL SHUTTLE** (*Saturday, June 11*): One of the largest art festivals in the nation, the Allentown Festival takes place each year in the enchanting Allentown historic preservation district of Buffalo. Tens of thousands of art patrons visit the festival. They come to enjoy the warmth of a late spring day in Buffalo, the charm of the Allentown area, and the quality of the arts and crafts presented by the more than 400 juried exhibitors. Continuous shuttle service, compliments of the Erie County Dental Society, will be available from the Marriott from 9 a.m. to 4 p.m.

*No fee.*

**NYSDA OFFICERS INSTALLATION DINNER DANCE** (*Saturday, June 11*): Join your colleagues at NYSDA's annual Dinner Dance as they honor the Association's 2005 President Lawrence E. Volland, his wife, Judith, and the officers of NYSDA. This gala event will be held at the Brookfield Country Club. The evening begins with cocktails and hors d'oeuvres. Dinner and dancing will follow. Attire is formal. Black tie is suggested. Transportation will be provided. Our thanks to MBNA America for underwriting the cost of this event.

*Fee: \$95 per person.*



# Semi-Annual Meeting Registration

June 9 – 12, 2005

Buffalo/Niagra Marriott  
1340 Millersport Highway  
Amherst, New York  
(716) 689-6900

Please complete and return to: Beth M. Wanek, NYSDA, 121 State St., 4th Floor, Albany, NY 12207 (800) 255-2100 FAX: (518) 465-3219

### REGISTRATION

Name \_\_\_\_\_  
Address \_\_\_\_\_  
\_\_\_\_\_  
City \_\_\_\_\_ State \_\_\_\_\_ Zip \_\_\_\_\_  
Telephone (\_\_\_\_) \_\_\_\_\_ Fax (\_\_\_\_) \_\_\_\_\_  
E-Mail \_\_\_\_\_  
Date of Arrival \_\_\_\_\_ Date of Departure \_\_\_\_\_

Please register the following individual(s) for the meeting. Please print names as they are to appear on badges. Badges may be picked up at the NYSDA Registration Desk.

### ACCOMMODATIONS PREFERRED

Rates are subject to state taxes, currently at 8.25%, and local taxes, currently at 5%. (Specific accommodations and room assignments will be determined upon check-in according to room availability at that time.)

Single Occupancy (\$109)     Double Occupancy (\$109)

- I would prefer a king bed, if available.
- I would prefer two double beds, if available.
- I would prefer a smoking room.
- Please check if you have a special need as the result of a disability as defined in the Americans With Disabilities Act. Please explain:  
\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

### RESERVATION GUARANTEE

Your reservation must be guaranteed by the following:

Credit Card \_\_\_\_\_ # \_\_\_\_\_ Exp. \_\_\_\_\_

Signature \_\_\_\_\_

In order to assure room reservations at the Buffalo/Niagara Marriott, all requests must be received in this office on or before May 9, 2005. Reservations not made by the group cutoff date will be accepted based on availability at the current rates. Check-in time is 3 p.m. Checkout time is noon.

For guaranteed reservations, I understand that I am liable for one night's room and tax, which will be billed through my credit card in the event I do not cancel 24 hours prior to the date of arrival.

### AIRPORT SHUTTLE SERVICE

To arrange for complimentary transportation to and from the airport, please complete this section:

Number of Passenger (s): \_\_\_\_\_

INBOUND FLIGHT: \_\_\_\_\_

Date of Travel: \_\_\_\_\_ Airline/Flight # \_\_\_\_\_ Arrival Time: \_\_\_\_\_

RETURN FLIGHT: \_\_\_\_\_

Date of Travel: \_\_\_\_\_ Airline/Flight # \_\_\_\_\_ Departure Time: \_\_\_\_\_

# Event Registration

Name of Guest(s) \_\_\_\_\_  
 \_\_\_\_\_

I/We would like to register for the following events (please make selections below):

\_\_\_ I've enclosed a check payable to NYSDA in the amount of \$ \_\_\_\_\_ .

\_\_\_ Please charge my credit card (NYSDA accepts Visa, MasterCard and AMEX):

\_\_\_ Visa \_\_\_ MasterCard \_\_\_ AMEX Exp. \_\_\_\_\_ .

	# Of Tickets	Total Cost
<b>THURSDAY, JUNE 9</b>		
<b>Cocktails at the Brink</b> \$35 per person		
<b>FRIDAY, JUNE 10</b>		
<b>Golf Tournament</b> \$100 per person My golf handicap or 18-hole average score is: _____ .		
<b>Trophy Bass Fishing</b> \$125 per person		
<b>Erie Canal Cruise</b> \$35 per person		
<b>Jamaican Beach Party</b> \$35 per person		
<b>SATURDAY, JUNE 11</b>		
<b>Yoga Wakeup</b> \$20 per person		
<b>Dinner Dance</b> \$95 per person		
<b>TOTAL</b>		\$

# Council Selects Steven Kerpen to Receive Tillis Award

THOMAS BONOMO, C.J. GARY RECEIVE HONORABLE MENTION

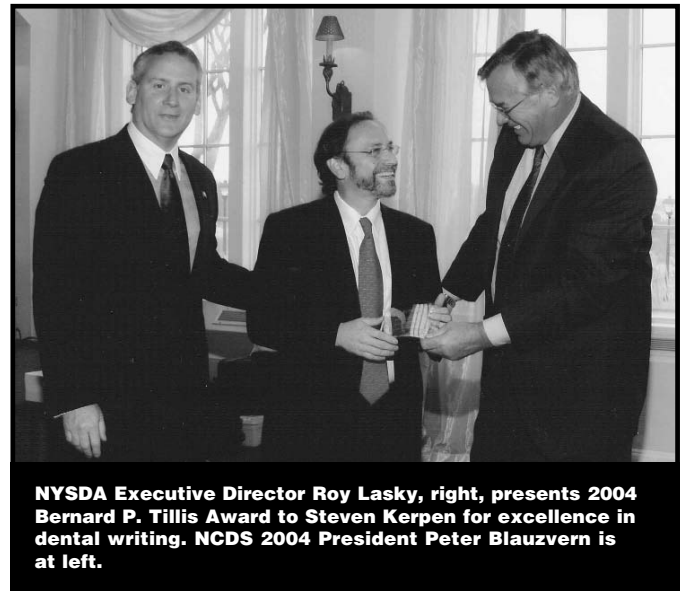
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**STEVEN J. KERPEN, D.M.D., M.P.H.**, of Nassau County is winner of the 2004 Bernard P. Tillis Award for excellence in dental writing.

Dr. Kerpen was selected to receive the award, presented by the NYSDA Council on Membership and Communications, for his editorial, "The Damn Millimeter," in the July/August 2004 Bulletin of the Nassau County Dental Society. He received an inscribed plaque. This is the second time Dr. Kerpen has received the award. He was previously honored in 2002.

The council also cited Thomas J. Bonomo, D.D.S., editor of the Suffolk County Dental Society Bulletin, and Chester J. Gary, D.D.S., J.D., editor of the Eighth District Dental Bulletin, and presented each with an Honorable Mention award. Dr. Bonomo's winning entry was his editorial "Younger Than Springtime Am I," which appeared in February 2004. Dr. Gary was recognized for his editorial "Fight Licensure Abuse," which appeared in spring 2004 and was subsequently republished as a "Viewpoint" in the June/July *NYSDJ*.

The Tillis Award was established in 1996 to honor the memory of the longtime *Journal* editor. It recognizes members of the Dental Association who through their writing in *The NYSDJ* or in any component publication promotes a positive image of organized dentistry.



Dr. Kerpen resigned at the end of last year as editor of the NCDS Bulletin. He had held the post for nine years, during which time the publication went from a two-page newsletter to an award-winning journal, recognized by the International College of Dentists. He received a masters in public health degree from Columbia University and his D.M.D. from the University of Pennsylvania School of Dental Medicine. He completed his general practice residency at Long Island Jewish, Hillside Medical Center, and his postgraduate work in periodontics at Columbia University School of Dental and Oral Surgery.

Dr. Kerpen is in private practice in Great Neck on Long Island. His winning entry is reproduced on the following page.

# From the Editor's Desk

Steven J. Kerpen, D.M.D., M.P.H.

## The Damn Millimeter

"I dreamed things that never were and say, why not?" – *George Bernard Shaw*

AS I SAT ANXIOUSLY in a comfortable dental chair, a reamer was expertly manipulated in the distal canal of my mandibular right first molar. The venerable, yet occasionally salty endodontist to whom I entrusted the removal of my remaining pulp tissue proclaimed, "Only one more millimeter to go." After a brief moment of quiet perspective he turned back to me and said, "With all the s—that's going on in the world today, here we are worrying about a damn millimeter."

While most of us may not have the same flare for language, I think we all at some time or another have experienced the feeling that the universe we work in is small or confining. The intricacies of our profession can seem mundane or over-exacting. It was with these thoughts in mind that I was struck by some fascinating data as I prepared a literature review for the Institutional Review Board of the North Shore/LIJ Health System. I proposed a project to study the impact of periodontal therapy on pre-term delivery of babies.

By this time, most dentists and many patients have become vaguely aware of the significant research concerning periodontal disease and many systemic conditions, including heart disease and diabetes. What goes unappreciated is the minute changes in gingival tissues, the "damn millimeters" that could conceivably have the most profound impacts on people's lives.

Every year in the United States, more than 440,000 babies are born too soon, i.e., pre-term. Of those infants who are born before 37 weeks, many are dangerously small. They are likely to spend days or even weeks in the neonatal intensive care unit. Compared to full-term babies, pre-term infants are at greater risk for several complications, including breathing problems, cerebral palsy and mental retardation, even death. The untoward events have a devastating and lasting impact on those affected and on society as a whole. The annual cost of care for these babies in the intensive care units alone is staggering, and in the billions of dollars. Pre-term delivery is, indeed, a significant social, economic and public health problem, even in the U.S., where approximately 1 in 10 deliveries is pre-term.

The researcher who has had the most profound influence in the field is Dr. Steven Offenbacher of the University of North Carolina at Chapel Hill. Indeed, he has been (no pun intended) the midwife of the concept that periodontal infections can lead to serious neonatal consequences.

Here is a scientist/dentist who dreams things that never were. A review of his publications reveals a profound intellect doggedly pursuing his goal. His studies in this field begin with a hypothesis and go through animal studies, retrospective longitudinal studies, large-scale prospective studies. He is finally now involved in a significant NIH multi-site intervention study that should soon answer the question as to whether periodontal care indeed will be an effective aid in helping to prevent pre-term labor and delivery. A detailed look at this data is illuminating.

Offenbacher, as others, defines periodontal disease in a way that may surprise many of us. Moderate-to-severe periodontitis, which seems to be associated with much neonatal morbidity, is considered to be present when only four or more sites probe at least 5 mm. 5.5% of his patients meet these minimal criteria. The vast majority of patients who show a positive correlation to pre-term delivery have even less periodontal disease. He also finds that women who have periodontal disease progression (even when they start their pregnancy in a healthy periodontal condition) have statistically smaller babies.

Periodontal progression is defined as only four sites with an increase of 2 mm! These are not unusual clinical presentations. In fact, many of us would not even treat these sorts of findings. Any dentist with even the most passing interest in periodontal tissues can appreciate how common, minute and seemingly mundane these changes are, particularly in the pregnant woman prone toward pregnancy gingivitis (50%).

Think for a moment about the potential impact of, say, an over-hanging restoration or of never being taught proper interproximal cleaning techniques. These changes are so subtle that we are the only ones trained and capable of recognizing, treating and preventing them. Dream, if you will, how it seems very possible that your ability to properly handle those mundane, miniscule and seemingly trivial millimeters could have an impact of a most significant and profound nature.

The men and women studying in this field are seeking to add even more meaning to our already rich professional lives. They are dreaming things that never were. It is our domain. We should, like George Bernard Shaw, be asking, "Why not?"

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