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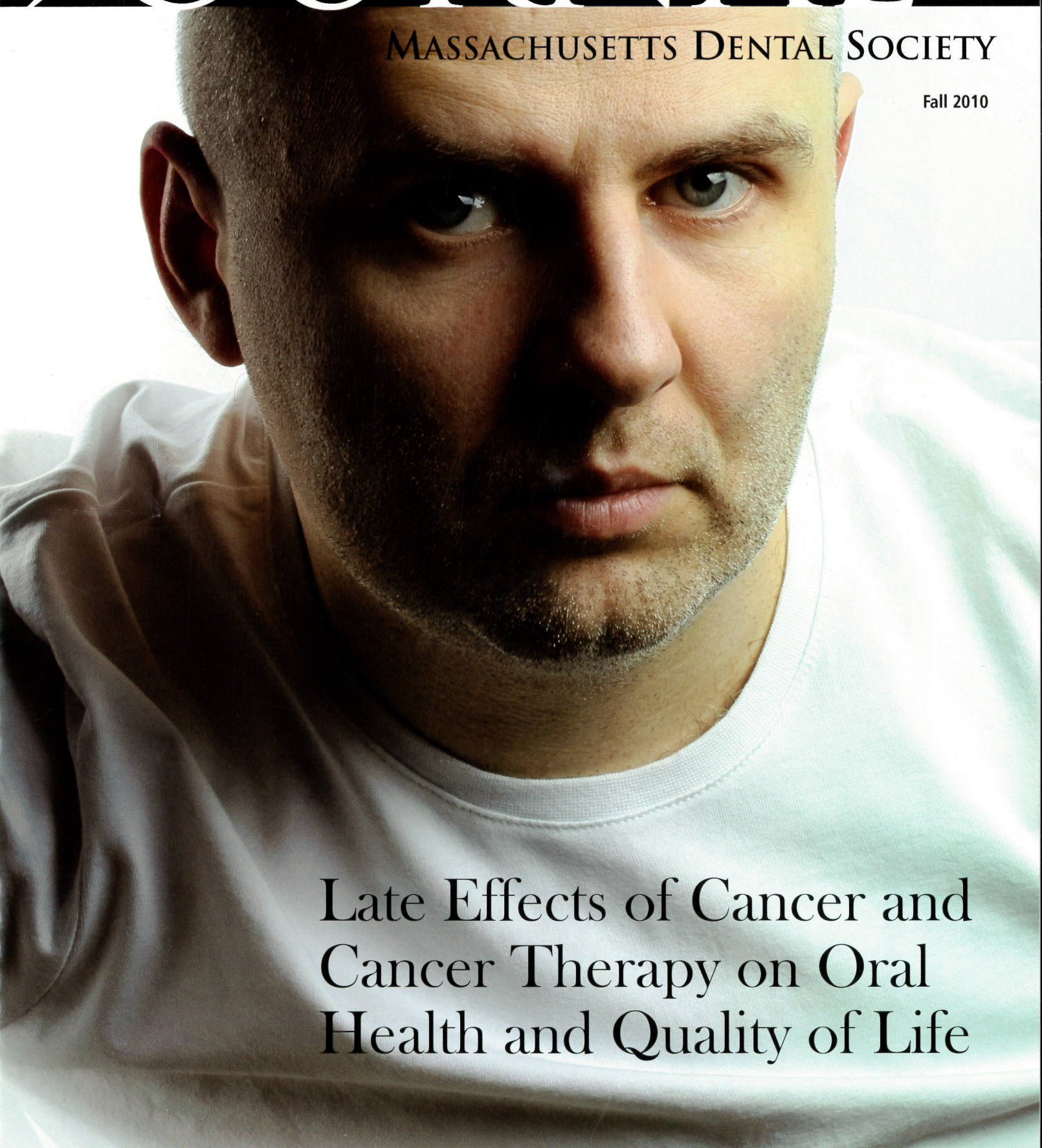

Late Effects of Cancer and Cancer Therapy on Oral Health and Quality of Life

ARE YOU DOING YOUR PART?

The MDS leadership has a responsibility to communicate important information to our members, who, in turn, have a responsibility to open their emails and read them. It is a two-way street. You pay your dues, but are you paying due diligence attention?

Every year, there are issues before our state legislature that directly impact the way we practice. Recent legislative sessions have seen bills dealing with such pivotal issues as allowing unsupervised, independent practice by dental hygienists; allowing retired dentists to maintain licensure for the purpose of volunteering their services; expanding the training, certification, and role of dental auxiliaries; and looking at dentistry's relationship with insurance companies.

You must not think that someone else will take care of things and "watch your back." Grassroots participation is essential—the more voices that speak, the more our elected officials pay attention. Any message we want communicated is so much more effective when there is a large number of constituents behind it. Our paid legislative agents plant the seeds, and our collective voice cultivates them. Elected officials pay attention to phone calls and emails. They care if they receive opinions from you, their constituents. When the issue of allowing the independent practice of dental hygiene came before the legislature, hygienists inundated their elected officials with phone calls and correspondence. At that time, dentists were much less involved and almost lost the issue because of lack of input. The message that legislators heard was that hygienists cared more than dentists did about improving access to dental services. We cannot allow such misconceptions regarding the issues that directly affect the practice of dentistry and quality of care we offer our patients.

Here is an example of why participation by all is needed. In this past legislative session, the MDS filed an amendment to stop insurance companies from capping our fees on noncovered services. These caps would directly affect our uninsured patients, who would, in effect, be challenged with higher fees to make up for lost remuneration due to the capped fees on insured patients.

Statistically, the largest uninsured population is people 65 and over. If insurance companies dictate what we can collect for services they don't even cover, then the uninsured will have higher fees because a dental practice has to meet its overhead costs.

The MDS sent out an Action Alert email on this issue to 3,137 members, requesting that a simple, prewritten email letter be sent to our state senators. All that each member had to do was forward the prewritten response—at most, two minutes of effort was required. This is how our membership responded:

- 861 members opened the email
- 160 members accessed Capwiz (the automated email response)
- 55 members contacted their state senators

Sadly, only 1.75 percent of those 3,137 members responded. Your involvement counts. We need our elected officials to pay attention to our legislative causes. We owe it to our patients and our profession to take, at least, a very small amount of our time to be proactive and effective. Our futures, collectively and individually, continue to be in our own hands. ■

David B. Becker

Arthur I. Schwartz

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I strongly disagree with Dr. Vincent DeAngelis's article exploring whether orthodontics is heading in the right direction, "A 50-Year Journey from Begg to Straight Wire and Beyond: Is Orthodontics on the Right Course Today?" (Vol.59/No. 2, Summer 2010, pages 38–42). When I first encountered this profession 40 years ago, orthodontics was an arcane specialty. Dental educators were advising their undergraduate students to avoid practicing orthodontics if they felt unprepared to deliver "ideal" orthodontics. But the objectivity of this advice could not be defined, and orthodontic education at the undergraduate level stagnated. Today, unfortunately, not much has improved, and this demagogy still largely influences generalists.

After serving in the U.S. Naval Dental Corps, attaining pediatric specialty certification, and then launching a private pediatric practice, the early orthodontic needs of my patients became increasingly clear—and glaringly obvious.

In 1972, the article "Six Keys to Normal Occlusion" by Dr. Lawrence F. Andrews[1] came across my desk. I have to say, without reservation, that this reading was an epiphany in my dental education. For the first time, a text gave objectivity to orthodontic diagnosis and treatment. I immediately became a continuing student of Dr. Andrews's Straight Wire Appliance and treatment techniques.

The specific point I want to make is the implication made by Dr. DeAngelis in his article that the Straight Wire Appliance directly causes gross root resorption is false. The Straight Wire is an appliance and not a treatment philosophy. It does not preclude the use of other personalized techniques. The Straight Wire Appliance may appear to resemble an Edgewise Appliance, but that is where the similarity ends. The Straight Wire Appliance is a fully programmed appliance when correctly sited with the referents obtained from Dr. Andrews's research and applied in the Andrews treatment mechanics. The Andrews System will allow the operator, if desired, to achieve the Six Keys, plus a mutually protected functional occlusion scheme, efficiently and effectively.[2-4]

The Straight Wire concept and appliance are being taught in most, if not all, North American orthodontic departments and selected pediatric programs. I have actively taught and shared my experience, knowledge, and expertise in the Straight Wire technique with numerous dentists throughout the U.S. and abroad. The Straight Wire technique has allowed the general and pediatric dentist in more than 90 percent of the so-called "normal" malocclusions to deliver an objective quality of orthodontic care within the standard of care of the specialty. Since 1975, I have offered my fully documented Straight Wire cases for review as evidence to support that statement.[5,6] Root resorption has never been an issue or major concern of the Straight Wire Technique. Also, Dr. Andrews has stated in print that it is rare for his patients to have root resorption.[7] This is logical, because the Straight Wire Technique is a direct-vector movement with a minimum of "round tripping" or "jiggling"—both common occurrences in the techniques that Dr. DeAngelis describes.

Root resorption is of multifactorial etiology, and all evidence indicates that 1 to 2 mm of apical root loss, if it does occur, seems inconsequential, particularly in light of the functional and esthetic benefits of orthodontic treatment—the scars of the operation, so to speak.[8,9]

I believe that we are likely to remain in the Straight Wire era for some time, because most of its advantages have yet to be discovered. Most orthodontists don't yet understand the huge differences between a nonprogrammed, partly programmed, and fully programmed appliance, and few are employing Dr. Andrews's major contributions to this area of dentistry.

In trying to chart the course of orthodontics in the 21st century, Dr. DeAngelis promotes his Amalgamated Technique based on a falsehood that is not evidenced by those of us using second-generation Andrews's Techniques into the 21st century.

Leonard J. Carapezza, DMD
Wayland

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Author's Response:

Dr. Carapezza's response to the "50-Year Journey" article is not surprising coming from an advocate of the Straight Wire Appliance. I am quite certain that proponents of the other appliances (Speed, Damon, Tip Edge, and Begg) referenced in the article would be equally vehement in their defenses.

The comments in the article concerning the shortcomings of the Straight Wire Appliance are clear and irrefutable, and need not be repeated in this concise rejoinder. They are also supported by others, such as Dr. James Kaley, adjunct professor of orthodontics at the University of North Carolina School of Dentistry and a Diplomate of the American Board of Orthodontics, et al., in an *Angle Orthodontist* article in which they reported that of their 200 consecutively treated Straight Wire cases, more than 90 percent had root resorption.[1] The authors observed that, statistically, the most severely resorbed apices—greater than one quarter of the maxillary central and lateral incisor roots—were subjected to lengthy rectangular archwire intraslot torque.

Additionally, Wehrbein et al. had the sad but rare opportunity to examine the maxilla and mandible of a deceased teenager who had been in treatment with the Straight Wire Appliance for only 19 months (Kaley's average treatment time was 34 months).[2] Their examination revealed severe root resorption of incisors and molars, fenestrations of the maxillary buccal and mandibular lingual alveolar plates, and perforation of the maxillary sinus by the molar palatal roots. These findings were not discernible radiographically. The authors opined that the action of intraslot torque by the rectangular archwire was directly responsible for this irreversible damage to the roots and paradental tissues. These are objective reports from advocates of the Straight Wire Appliance. It should be noted that my interest in the Amalgamated Technique is strictly educational. Entrepreneurs continue to perpetuate the myth of one-size-fits-all malocclusions by proselytizing the "fully programmed" brackets of Andrews and Roth.

The Andrews's Straight Wire Appliance, as modified by Roth,[3,4] is programmed to deliver intraslot torque, a procedure that Thurow, an expert in biomechanics and engineering in dentofacial orthopedics, warned should be avoided due to its inadvertent, superfluous roundtripping of root apices.[5] Dr. Andrews, unfortunately, ignored that admonition as he developed his appliance. Newton's third law of physics is incontrovertible, even in orthodontics.

Root resorption should not be considered a *sine qua non* for orthodontic treatment. The mentality of "scarring of the operation, so to speak" is no more than a poor excuse for faulty biomechanics. Sadly, the biology in biomechanics is ignored by the clinician who favors perfect dental alignment within the Six Keys to normal occlusion at the expense of damaged root apices and paradental structures over ideal alignment with biologically sound physiologic, nonpathologic results. "Do you want root apices and intact paradental tissues at the end of treatment or ideal occlusion?" The discerning clinician should demand both.

And finally, orthodontic academicians who admonish the undergraduate dental student against treating complex malocclusions without sound, extensive postgraduate education in growth and development and orthodontics provide sage advice for the aspiring orthodontic practitioner who, without formalized training by competent orthodontic instructors in university-based programs, does not comprehend the nuances of orthodontic diagnosis, treatment planning, biomechanics, and growth and development. This is not demagogy; this is prudent advice. Likewise, the orthodontist who is not a surgeon must resist a hankering for performing orthognathic surgery for the patient with a skeletal dysplasia; thus, the need for a specialist in maxillofacial surgery. ■

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Woburn

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UNDERSTANDING MUTUAL FUND EXPENSE RATIOS

Every mutual fund must disclose certain costs associated with running the fund. Those costs represent a fund's expense ratio, which is expressed as a percentage of a fund's assets. For example, a fund that has $100 million in assets and annual expenses of $1 million would report a 1 percent expense ratio (1 percent of $100 million = $1 million).

Why is a fund's expense ratio important? First, it can help you gauge how efficiently the fund operates. A high expense ratio reduces the amount that is paid to you as a shareholder. Second, a fund's expenses affect your net returns, particularly over the long term. For example, let's look at a hypothetical illustration (which doesn't reflect the performance of any actual security). Assume you have $10,000 in one stock fund that earns a 5.5 percent return and $10,000 in another stock fund that earns exactly the same return but that costs you an extra half-percent in expenses. The difference between 5.5 percent and 5 percent over 20 years means a $2,645 reduction in your bottom line.

That's not to say that you should automatically reject a fund just because it has a high expense ratio; the fund's performance may be worth the higher cost. However, you do need to take expenses into account, especially if you're investing for the long term.

Some general categories of funds tend to have higher expense ratios than others. For example, a stock fund that specializes in emerging markets may have to spend more on research than a fund that invests only in large-cap U.S. stocks for which a great deal of information is readily available. A fund that is actively managed may have higher expenses than a fund that mirrors an index.

Each mutual fund's prospectus must include a table in the front that you can use to compare the expenses of various funds. The table lists the fund's expense ratio, as well as a breakdown of the costs included in it, which fall into three general areas: management fees, marketing costs, and administrative fees.

Management Fees

Every fund has an investment management or advisor firm that manages the fund and makes investment decisions. Even an index fund, which does relatively little trading and whose investments basically duplicate those of an index, will have a firm or an individual who handles any transactions. Management fees often represent the single largest portion of a typical fund's expense ratio.

Marketing Costs

These costs also are known as 12b-1 fees, after the legal provision that permits them. They were originally designed to let funds recoup costs associated with distribution and advertising, on the theory that attracting new investors and additional assets would help make a fund more cost-effective for each investor. In recent years, there has been discussion regarding whether 12b-1 fees should be eliminated—especially for funds that are closed to new investors and therefore should have little need to market themselves—but they are still very common.

Administrative Fees

This category of fees includes the cost of recordkeeping, custodianship, taxes, and legal, accounting, and auditing services.

What's Not Included in an Expense Ratio

Trading expenses represent the cost of buying or selling securities, and also can have a substantial impact on your net return over time. Trading costs, which include commissions paid by the fund when it buys or sells a security, aren't included in a fund's expense ratio. However, funds are required to report the per-share cost of their annual commissions; this can be found in a fund's annual report or statement of additional information.

Also, not included in the expense ratio is any redemption fee a fund might charge if you sell your shares before a specified time, or any sales charge the fund might impose at the time of purchase or sale.

Before investing in a mutual fund, carefully consider its investment objectives and risks, as well as its charges and expenses. This information is available in the prospectus, which can be obtained from the fund. Read it carefully before investing.

Comparison Shopping

The "Tools and Calculators" section of the Financial Industry Regulatory Authority (FINRA) Web site includes an online Fund Analyzer that lets you compare the impact over time of the fees and expenses of as many as three funds. ■

GEORGE GONSER

Mr. Gonser is CEO of MDSIS–Spring Insurance Group.

LOOKING BEYOND THE MASS. RATE CAP ISSUE

If you had told me a year ago that the health insurance market would be turned on its head and a national Health Care Reform Law would be steaming forward toward implementation, I would have thought you were crazy. Scott Brown's election to the U.S. Senate last January seemingly put a nail in the proverbial coffin of the national health care reform discussions. Or so it was thought.

While the national health care effort seemed to be tucked in the "nice try, but no go" drawer, a stimulant was festering. In California, the rate increases handed out to small businesses by California's largest insurer were, on average, in excess of 30 percent. Companies protested, employees fumed, and the media reported on the situation, thereby turning it into a national issue. The California Division of Insurance rejected the rates and demanded that the carrier go back and recalculate them. As a result, President Barack Obama leveraged this uprising and the anger of the U.S. public to reinvigorate the seemingly dead national health care reform issue. A few weeks later, the national Health Care Reform Law was finalized.

Meanwhile, back in Massachusetts—the incubator of the national Health Care Reform Law—small businesses were delivered an average increase of 25 percent after nearly 10 years of annual double-digit increases. Spurred on by California and Rhode Island rate cap efforts, Massachusetts Governor Deval Patrick and the Division of Insurance (DOI) rejected the filed April 2010 renewal and new business rates, and thereby entered into a battle with the state's carriers over "proper" rating. As a result, the renewal and new business rates were held to 2009 rates, with minor adjustments for census and address changes. Small businesses and employees rejoiced. Undeniably, insurance rate relief was welcomed, but is it sustainable?

Let's look at some of the facts of this contentious situation.

Background—Health insurance rates going up at a double-digit clip are clearly unsustainable. Businesses can't afford the increases, especially with the economic climate we are currently experiencing. They are forced to increase the cost sharing with the employees, which makes or has made it too pricey for them as well. While something had to be done, is the rate cap the long-term answer?

Insurance Carriers—Insurance carriers in Massachusetts use approximately 90 percent of collected premiums to pay for claims, and that represents one of the highest percentages in the country. Suffice it to say that the carriers in Massachusetts are administratively lean. So, if carriers are keeping costs down and paying a high percentage of premium dollars on claims, what is driving costs? According to the carriers, the contracts with providers are the root of the problem. For 2010, the carriers are estimating that 75 percent of premium increases are tied to the provider contracts. If that is truly the case, then the DOI's capping of increases for 2010 at 2009 rates (artificial rates) will create a situation where carriers simply won't be able to cover costs. Short-term reserves will cover the immediate shortfall, but what about long-term?

Providers—We, as consumers, still want the best, most innovative care; however, there is a price for innovation. The reputation of Massachusetts providers is among the best in the world; however, there is also a price for excellence. Combine these two factors and you have an expensive model of providing care. The provider community must balance government payment deficiencies and increased payment delinquency issues, while providing excellent care and cutting-edge procedures and technology. Shouldn't they get paid for the services they provide? Another issue is the disparity of payments between community hospitals and teaching hospitals. In some cases, there is upwards of a 300 percent disparity in cost for the same procedures. Maybe the bundled/capitated arrangement to eliminate cost differentials and spur competition is the answer?

General Population—We have all had to deal with skyrocketing health insurance costs, reduced benefits, and higher copays. A common sentiment is that we really don't care what it costs after paying so much, but in actuality, we need to utilize the system more efficiently and carefully. With plans being devised regarding cost and quality factors, there will more incentivized decision making going forward.

An argument can be made for all parties involved—government, carriers, providers, consumers—that they are justified in their individual actions. However, it will take a combined effort by all these groups to correct this situation. From a more educated and involved consumer, to the restructuring of provider contracts, to the continued vigilance on the carriers' behalf, tough decisions and sacrifices must be made. It is not one entity's fault, but a collective need for change and improvement.

For now, the rate cap issue rages on. And if we are concerned about costs and challenges, we haven't even really touched on the myriad issues involving the national Health Care Reform Law and its potentially far-reaching effects. We will save that for the next issue—and maybe beyond. ■

TECHNOLOGY TODAY

GLENN LOMBARDI

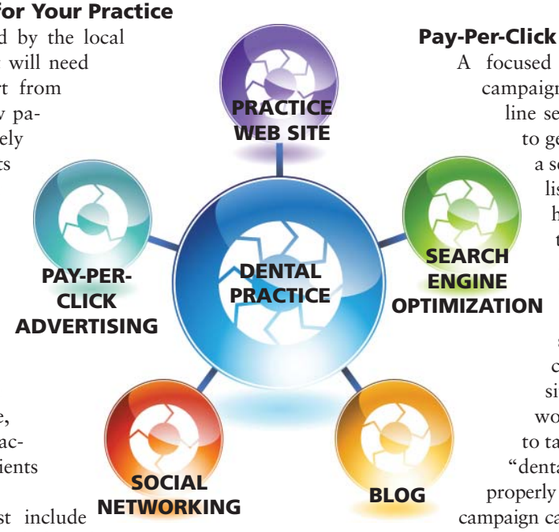
Mr. Lombardi is president of Officite, a provider of customized dental and medical Web sites based in Downer's Grove, IL.

FIVE WAYS TO INCREASE PATIENT FLOW TO YOUR PRACTICE

The growth of your dental practice relies heavily on the effectiveness and depth of your comprehensive marketing plan. To compete with other dentists and attract new patients to your practice, you must start with a professionally designed Web site—keeping it at the core of all of your varied marketing strategies. Getting your practice front and center when patients are looking for dental services is both necessary and possible with the right online marketing solutions.

Build an Online Presence for Your Practice

In a profession that is saturated by the local competition, a successful dentist will need to set his or her practice apart from the rest in order to generate new patients. How? Begin with a creatively designed Web site that reflects the values and quality of your practice. Patients are online searching for dental care in your neighborhood, but if your practice hasn't entered the vast online world, you're missing out on valuable patient leads. A Web site will enable you to compete with dentists who are already gaining visibility online, plus you'll be promoting your practice 24/7, reaching out to patients even when your office is closed.



An effective Web site must include the essential tools that patients look for when searching for a dentist online, so make sure your Web site is a patient-friendly resource for easily accessed information. Post your practice details, including clinician bios, hours of operation, maps, phone numbers, and services. Enhance the performance of your site by providing patients with an educational library of oral health information, and consider posting videos, photos, and patient testimonials that highlight your work. But most important is to present patients with a strong call-to-action, enticing your online visitors to contact your practice for further information.

Search Engine Optimization

Your Web site is only as valuable as a patient's ability to find it in the search engine's results. And while there are several strategies for driving targeted traffic to your site, search engine optimization (SEO) is one of the most fundamental and effective tactics for doing so. SEO involves fine-tuning the internal components of your Web site to improve its ranking in search engines, including content optimization, strong keywords, and link-building. When patients visit major search engines, like Google and Yahoo!, to search for dentists in their town, they visit the dental Web sites that appear in the top positions. The higher your site appears for your targeted keywords, such as "your town" and "dentist," the more visibility your practice will earn. And that visibility leads to higher patient volume.

Pay-Per-Click Advertising

A focused pay-per-click (PPC) advertising campaign is another key component of online search marketing. PPC enables you to get your practice listed at the top of a search engine page in the sponsored listings, even if you aren't earning high rankings naturally. You can then modify your campaign to create ads that are brief and enticing, encouraging patients to click on them, directing them to your Web site. You only pay when someone clicks-through the ad to your Web site, and you choose the specific keywords and service electives you want to target, such as "tooth whitening" or "dental implants." When implemented properly and combined with SEO, a PPC campaign can reach patients beyond your local town to surrounding communities, maximizing the performance of your site and online marketing strategies.

Social Networking

Social networking is fast becoming one of the most effective means of online communication and information sharing available today. Facebook, Twitter, MySpace, and other social media sites have fueled the growth of word-of-mouth referrals, one of the most efficient ways to build awareness for your practice and produce qualified leads. Establish a space on the major social media sites, and you'll gain additional exposure for your practice, connect with current and potential patients, and have the ability to broadcast your news or messages to your entire online network. With that being said, you'll want to seamlessly integrate your Web site into the relatively new and rapidly growing world of social networks in order to enhance the visibility and reputation for your practice online.

Expand Your Reach with a Blog

Finally, consider creating a blog for your practice's Web site. When managed properly, a blog can be an extremely effective avenue for highlighting your expertise in the dental profession, as well as a valuable means for developing a rapport with your potential and existing patients. But the greatest benefit of establishing a blog is the higher ranking it earns your Web site in the search results. Because each blog page is a separate Web page for your site, frequent posts provide numerous pages for search engines to index. As long as you keep your blog consistently up-to-date with fresh and valuable content, you'll notice an increase in your page ranking, and this will dramatically increase your online presence and attract new patients to your site.

The benefits of a professional Web site combined with results-oriented online marketing solutions and strategies can bring you a higher return on your investment than any other form of marketing available today. If you want to thrive in today's dental profession, you need to position your practice where your patients are—the Internet. Not to mention that when your Web site acts as the focal point for all of your marketing efforts, you'll maximize the performance of your site, which translates to more patients for your practice. ■

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Late Effects of Cancer and Cancer Therapy on Oral Health and Quality of Life

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Abstract

Persisting and chronic oral complications of cancer therapy are common. Oral complications in cancer survivors are underreported but impact oral function and quality of life. Prevention and management of oral complications in cancer survivors requires interdisciplinary care. The purpose of this article is to review the common oral complications in cancer survivors.

Introduction

Oral complications of cancer and cancer therapy, which arise during and continue following therapy, affect oral function and impact general health and, subsequently, survivors' quality of life. Prevention and management of oral complications are required throughout the course of the disease, from diagnosis, through treatment, and following cancer therapy. The impact of acute oral complications in cancer patients is generally recognized; however, the potential negative impact of late oral health problems on symptom burden, oral function, and overall health are underappreciated. As survivorship continues to rise, there is an increased need to determine the impact of late treatment effects and the most effective means of prevention and treatment.

Head-and-neck cancer (HNC) and therapy for the disease cause acute oral complications that impact quality of life. These complications include mucositis and associated pain, hyposalivation, viscous oral secretions, taste change or taste loss, difficulty with mastication and dysphagia, function of oral prostheses, and affected speech. While patients recover from some of the acute effects of therapy, many experience persisting oral complications that impact oral health, general health, and quality of life.

Prior to cancer therapy, the oral and dental status of the patient must be carefully examined, and any medically necessary dental care must be given to prevent or minimize oral and systemic complications during therapy and survivorship. Following cancer therapy, it is critical for the dental team to understand the prior cancer therapy that may limit dental treatment, ongoing medical management, any comorbidities, and prognosis. This is best accomplished with close communication between oncologists and dentists.

Oral Quality of Life and Symptom Burden

Oral complications during and following cancer therapy depend upon the disease under treatment, the stage and location of disease, the medication(s) and dosage, the schedule of therapy, and any patient comorbidities, including individual susceptibility. Radiation and chemotherapy may affect oral tissues, oral mucosa, salivary glands, neurosensory function, dentition, periodontium, and muscular and joint function.

Advances in the chemotherapy management of malignant disease over the past decade include therapy directed at molecular targets expressed by tumor cells and improvements in surgery, radiation, combined therapies, and supportive care. Induction and concurrent chemotherapy is increasingly incorporated in the management of HNC[1] and may lead to more severe and prolonged effects on oral tissues. Posttreatment chemoprevention and maintenance therapy is under investigation for a number of cancers and may become common in cancer control.

Acute complications may lead to persisting mucosal symptoms that result in chronic neurosensory symptoms; salivary gland dysfunction may also become chronic, thereby increasing the risk of late oral and dental complications. Effective prevention and management of oral mucositis during therapy may reduce the severity of chronic symptoms. Chronic sequelae of radiation include mucosal pain, atrophy, infection, fibrosis, salivary gland dysfunction, possible change in taste, and an increased risk of dental and periodontal disease, with risk of mucosal and bone necrosis. The sequelae of chemotherapy include mucosal atrophy/inflammation, neurosensory change (taste and/or pain), salivary gland dysfunction, and impairment of craniofacial and dental growth and development in children.

Quality of life is affected in patients with late effects of cancer therapy. Quality of life in HNC patients more than six months postradiation therapy identified common persisting symptoms, including dry mouth (92 percent), change in taste (75 percent), and difficulty eating (40 percent).[2] The majority of patients experienced pain (58 percent), and 17 percent rated pain as moderate or severe with one-third reporting that the pain interfered with daily activities. Oral health outcomes were reported in 357 HNC patients who were followed for up to five years after cancer therapy and who had reported that dental problems, such as trismus, xerostomia, and thick saliva, increased after one year and continued at their last follow-up.[3] Another study assessed patients up to five years posttreatment, identifying dry mouth, thick saliva, speech changes, dental problems, and sleep disturbance that affected quality of life (all $p<.01$).[4] Similar findings in another study showed a gradual improvement in depression and global quality of life over five years.[5] A prospective study of nasopharyngeal cancer (NPC) patients treated with radiation therapy up to 24 months found poorer global health, fatigue, loss of appetite, and dysphagia (all $p<.01$); xerostomia and thick saliva ($p<.001$); taste change and dental problems ($p<.05$); and pain and emotional function ($p<.005$).[6] These findings were confirmed in another study.[7]

Hyposalivation

Saliva is a complex secretion that provides oral lubrication and wetting, and allows food molecules to reach taste receptors and to develop a bolus for deglutition. Dietary shifts are seen in HNC patients following treatment, with increased consumption of high-carbohydrate foods of moist or pureed consistency. Saliva also possesses antimicrobial and remineralizing effects, as well as growth factors that may be important in tissue repair. Saliva is necessary to maintain dental integrity by providing calcium and phosphate, maintaining pH, and effecting oral flora.

Several approaches have been examined to reduce hyposalivation in cancer patients. Amifostine (WR-2721) is a free radical scavenger approved to prevent hyposalivation in patients undergoing radiation therapy for HNC. A recent meta-analysis demonstrated that amifostine resulted in a decrease in acute and late hyposalivation.[8] Salivary gland transfer out of the radiation field has been discussed; however, the use of advanced radiation technology, such as intensity-modulated radiation therapy (IMRT) to spare salivary tissue, has become standard in HNC radiation therapy, limiting the consideration for this surgical approach. Measurements of salivary flow after IMRT, where the major glands are spared high-dose exposure confirm less severe hyposalivation and improved quality of life.

Sialagogues, such as pilocarpine, cevimeline, and bethanechol, may improve hyposalivation in patients with residual salivary gland function.[9] IMRT with salivary gland sparing may allow stimulation of residual gland function with sialagogues. Products for mouth wetting (salivary substitutes) should be considered for palliation when saliva production cannot be stimulated. Despite these products, patients often rely on carrying water for frequent mouth wetting. There has been no assessment of saliva viscosity and related function, and while mucolytics such as guaifenesin and acetylcysteine can be considered for patients with thickened secretions, their effectiveness is not well documented.

Chemotherapy in breast cancer patients has been shown to cause mucosal lesions, affect salivary function leading to a microbial shift to cariogenic and fungal flora, and cause taste change that may persist for more than six months.[10] Decreased phosphate and secretory IgA also is reported. In stem cell transplantation, hyposalivation persists after six months and at three years.[11] In addition, medications commonly used in supportive care of chemotherapy patients (e.g., antiemetics, analgesics, antianxiety/antidepressants) may cause hyposalivation. Xerostomia and sore mouth are seen in patients who have undergone stem cell transplantation, with more frequent and severe symptoms in myeloablative transplant compared to reduced-intensity conditioning.[12]

Dental Health

Oral hygiene may be compromised following cancer therapy, due to limited intraoral access, increased plaque accumulation, and microbial shifts associated with hyposalivation. Periodontal bone loss is increased in people with hyposalivation and in fields of high-dose radiation therapy. Progressive periodontal disease and periodontal management within the high-dose radiation field represent a risk factor for osteonecrosis.[13] In addition, hyposalivation limiting remineralization and diet change lead to risk of dental demineralization that may progress rapidly, causing rampant tooth destruction. Buffering capacity, mineral exposure, and antimicrobial factors are affected. Fluoride shifts the equilibrium toward deposition of calcium in enamel, and it has antibacterial effects that may be important in protecting against dental damage.

Prevention requires excellent oral hygiene and a noncariogenic diet.[13] The bacterial component can be managed with chlorhexidine rinse. Remineralization of teeth can be favored with the use of fluoride and by providing calcium and phosphate in the oral environment (remineralizing products).

Oral Pain

Oral pain may be due to tumor effects and associated with cancer treatment. Recurrence of pain following treatment can be associated with cancer recurrence. While oral pain severity is expected to decrease following cancer therapy, low-intensity

pain following treatment is reported in the majority of patients at follow-up between six and 12 months and likely continues indefinitely.[14] The persistence of mucosal sensitivity may be due to atrophy of the mucosa, mucosal neuropathy, and hyposalivation. Chemotherapeutic agents may result in peripheral neuropathy, including orofacial neuropathy. Postradiation and postsurgical fibrosis and postsurgical defects in the jaw may lead to change in function and promote temporomandibular disorders (TMDs) that may be compounded by surgical complications and anxiety or depression.

Taste Alterations

Taste is related to sensory mechanisms, including taste, texture, temperature, and smell, that are perceived when placing food or other agents in the mouth. Taste is composed of five basic qualities: sweet, bitter, salty, sour, and umami. Umami is the taste sensation associated with pleasure or desirable flavor, and loss of umami has been suggested to have the strongest correlation with impact on quality of life.[15] Taste is mediated by epithelial receptors, is impacted by hyposalivation, and may be affected by microbial shifts and retention of food in the mouth. Additionally, it is affected by oral hygiene, dental and periodontal disease, mucosal infection, and diet.

Reduced or abnormal taste occurs in up to 100 percent of HNC patients during and following radiation therapy with or without chemotherapy.[13] Recovery of taste is variable, in some studies improving in two to six months following cancer therapy, although taste change may continue indefinitely. The impact of taste change includes reduced interest in food, leading to reduced caloric and nutrient intake. Similar findings are noted in stem cell transplantation, with more severe symptoms in myeloablative transplantation as compared to reduced-intensity conditioning. Temporary change in taste occurs due to solid-tumor chemotherapy, such as that received by breast cancer patients. Chemotherapy may be secreted in saliva, resulting in taste change until the drug is cleared; however, taste change may continue due to direct damage to taste receptors. Tissue necrosis, oral bleeding, and postsurgical wounds may contribute to taste change, halitosis, and altered smell. Taste disorders may also follow oncologic surgery, which may damage the lingual branch of the glossopharyngeal nerve or the chorda tympani.

IMRT may spare salivary glands and thus reduce the impact of radiation therapy on taste. However, low-dose irradiation of wider areas of the oral cavity may impact taste. Radioprotectors, such as amifostine, may have utility in affecting taste by protection of tissue or indirectly by maintenance of saliva.[16] Dietary counseling/modification, addition of seasoning to food, avoidance of unpleasant foods, and food rotation are recommended. Local infection and hyposalivation should be managed if possible. Zinc supplementation may affect taste dysfunction.[17,18]

Postradiation Fibrosis

Radiation therapy and surgery may lead to limited oral opening, limited mobility of the tongue, and trismus that may affect oral function. Trismus may be defined as a maximum jaw opening of <35 mm and severe trismus as a maximum jaw opening of <25 mm; it is reported in up to 45 percent of HNC patients. Radiation fields that include the masseter and pterygoid muscles are associated with trismus.[19] While IMRT has been expected to be associated with reduced trismus, this is not seen in recent studies. Prevention of trismus may be achieved by modifying radiation therapy fields and by introducing active jaw range-of-motion exercises during radiation therapy. Pentoxifylline, which affects fibrogenic cytokine production, has been shown to improve established trismus[19] but has not been studied for prevention. Established trismus may show limited response to jaw exercising. Botulinum toxin has also been assessed for the management of trismus, although its benefits are not clearly documented.

Infection

Local oral infections and increased risk of systemic infection from an oral source may occur in cancer patients. Reactivation of latent organisms and exacerbation of chronic foci of infection, including dental and periodontal infection, may occur. Cancer therapy may lead to shifts in microbial flora that can lead to infection. Chemotherapy can compromise oral mucosal immune defense mechanisms and reduce antimicrobial functions of saliva; myelosuppression and immunosuppression may lead to exacerbation of preexisting sites of chronic infection or predispose the patient to new infection and increase the risk of systemic infection. Latent herpes simplex virus infections exacerbate when host immune defenses are compromised due to malignant disease or the chemotherapeutic regimens. Management may include prophylaxis for seropositive patients who will become myelosuppressed, or early recognition and use of antivirals.

Hemorrhage

Thrombocytopenia may occur in patients on high-dose chemotherapeutic regimens or due to disease involving the bone marrow. Oral hemorrhage can occur when platelet counts are below 25,000/mm,[3] is more likely in patients with gingivitis or periodontal disease, and may occur in ulcerative oral mucositis.

Neurotoxicity

Some chemotherapeutic agents are neurotoxic (e.g., vinca alkaloids, platinum agents, and taxanes) and may lead to orofacial dysesthesia and pain that can be confused with pulpal disease, causing pain. Some patients may develop dental hypersensitivity following cancer therapy that may be due to dental demineralization and possibly neuropathy. Patients may experience symptomatic relief with topical fluorides and/or desensitizing agents, including toothpaste. Pain experience may be impacted by anxiety, depression, and sleep disturbances associated with cancer or cancer therapy.

Temporomandibular Disorders

Orofacial pain in cancer patients may include TMDs. Postsurgical complications, including mandibular discontinuity defects, posttreatment fibrosis, and clenching and bruxism, may be increased, resulting in orofacial pain. These patients may benefit from oral habit appliances, physical therapy—such as massage, physiotherapy, and/or muscle relaxants—and management of mood change and sleep dysfunction.

Compromised Nutrition

Compromised nutrition may occur due to nausea, emesis, and altered oral function. Oral function may be affected by hyposalivation, taste change, oropharyn-

Table 1. Chronic Oral Complications of Cancer Therapy

Oral Complication	Potential Direct Risk Factors	Potential Indirect Risk Factors
Hyposalivation	Radiation, chemotherapy	Dehydration; medications: anticholinergic, antinausea, antidepressant, antianxiety, antihypertensive, and analgesic drugs
Dental demineralization/caries	Hyposalivation, compromised oral hygiene, microbial shifts, diet change	Antibacterials causing microbial shifts; emesis, reflux
Dental sensitivity	Dentinal hypersensitivity, gingival recession, dental demineralization	Neuropathy
Periodontal attachment loss	Radiation, hyposalivation, oral hygiene, microbial shifts	Individual susceptibility
Mucosal sensitivity	Mucosal atrophy, neuropathy, mucositis, hyposalivation, physical/thermal/chemical trauma	Mucosal infections, reactivation of herpes viruses
Taste reduction/taste change/halitosis	Radiation, chemotherapy receptor toxicity, neuropathy, tumor necrosis; oral hygiene, diet, emesis, reflux	Secondary infection (candida, periodontal disease, hyposalivation)
Viral infection	Herpes virus infection (HSV, CMV, VZV, EBV)	Myelosuppression, immunosuppression
Fungal infection	Hyposalivation, tobacco use, prostheses, antibiotics, steroids	Altered local and systemic immunity, myelosuppression, immunosuppression
Bacterial infection	Poor oral hygiene, antimicrobials, hyposalivation Mucosal atrophy Acquired pathogens	Altered local and systemic immunity, myelosuppression, immunosuppression
Hemorrhage	Oral mucositis, ulceration, inflammation, tumor necrosis; gingivitis/periodontitis Physical trauma, infections (e.g., HSV)	Thrombocytopenia, acquired coagulopathy; decreased clotting factors (e.g., DIC, liver pathosis) Genetic susceptibility
Neuropathies	Surgery, radiotherapy, cancer chemotherapy (e.g., vinca alkaloids, platinum agents, taxanes, other specific drug toxicity)	Anxiety, depression, sleep disorder
Trismus, limited movement of oral tissues	Postsurgical/postradiation fibrosis; sclerosis with graft-versus-host disease	Myelosuppression, anemia, nutritional status, diabetes mellitus, tobacco use; immunosuppression
Temporomandibular disorders	Mandibular discontinuity, tissue fibrosis	Anxiety, depression, sleep disorder
Compromised wound healing	Vascular supply, tissue cellularity; radiation therapy, chemotherapy	Salivary hypofunction, secondary infection
Soft-tissue necrosis, osteonecrosis	Radiation therapy, trauma, bisphosphonate drugs, possible antiangiogenic drugs, tobacco use, trauma	Diabetes, tobacco use, nutritional compromise; immunosuppression, mucosal and salivary gland pathosis
Graft-versus-host disease (post-stem cell transplant)	Unrelated donor, mismatch transplant	Prior mucosal conditions
Recurrent, secondary, or other cancers	Radiation therapy, chemotherapy, regional cancerization, tobacco use, alcohol, viral agents (e.g., HPV, EBV)	Immunosuppression
Compromised systemic health and nutritional compromise	Oral function, dysphagia, hyposalivation, taste change, orofacial and mucosal pain, dental status, necrosis	Infection, nutrient/caloric demand, GI dysfunction
Dental and skeletal growth and development (pediatric patients)	Radiation therapy, chemotherapy, direct tissue toxicity	Hormonal effects on growth and development, stage of dental and skeletal maturation at time of therapy

geal mucositis, orofacial movement and pain, and altered or limited mastication and deglutition due to posttreatment fibrosis. The long-term impact of diet shifts on diet quality may result in macro- and micro-nutrient deficiencies. All factors associated with oral function and oral intake should be addressed in management.

Growth and Development in Children

Radiation therapy and high-dose chemotherapy can impact orofacial and dental development in children. Bone growth may be affected in high-dose radiated tissues. Individuals in whom the hypothalamus is affected may have delayed or altered maturation and sexual development. The possible effects on the dentition of cancer therapy include agenesis and alterations in tooth formation and tooth eruption, morphologic changes in enamel, altered crowns of teeth, and shortened and/or conical-shaped roots. Dental malformations may result in reduced occlusal vertical dimension and mobility of teeth with agenesis of roots. These changes may not be clinically apparent, but may be identified on imaging.

Compromised Wound Healing

High-dose chemotherapy, radiation therapy, myelosuppression, and nutritional status may compromise tissue healing due to local and systemic effects that can affect patients who have undergone dental procedures. In addition to cancer therapy, comorbid conditions (e.g., diabetes mellitus, myelosuppression, anemia, tobacco use, and nutritional compromise) may affect wound healing. These factors influence the treatment chosen following cancer therapy.

Guidelines for dental extractions in oncology are primarily based on expert opinion. General recommendations are:

- Expert and minimally traumatic extractions >10 days prior to radiation therapy or anticipated absolute neutrophil count <500/mm^3; antibiotic prophylaxis may be recommended if neutrophil count is <1,000/mm^3
- Minimal tissue trauma and primary closure of surgical site, if possible
- Platelet support if platelet count is <40,000/mm^3

Halitosis

Halitosis in cancer patients can be caused by tissue necrosis, hyposalivation, mouth breathing, poor oral hygiene, altered diet, infection, and oral bleeding. Treatment is directed at diagnosis and treatment of the cause(s) when possible.

Soft Tissue and Osteonecrosis

Risk for osteonecrosis of the jaws is seen in patients following head-and-neck radiation therapy, and in patients provided bisphosphonates for oncologic purposes and possibly antiangiogenic medications. Mucosal necrosis and bone exposure can be asymptomatic or minimally symptomatic and, therefore, not recognized until progressive and symptomatic, resulting in limited recognition and underdiagnosis. Comorbid risk factors include diabetes, immunosuppressive therapy and immunosuppression, local trauma, and tobacco use. Prevention is the primary goal, and pretreatment dental management and preventive dental care to reduce local tissue irritation and dental disease following treatment are critical.

In radiation-associated osteonecrosis, management may include antimicrobials, hyperbaric oxygen, sequestrectomy, and surgery with vascularized free flaps in advanced cases.[20,21] Other adjunctive approaches, including the use of pentoxifylline and vitamin E, are in study. In bisphosphonate-associated osteonecrosis, management includes topical antiseptic rinses, antimicrobials, gentle sequestrectomy, and avoidance of surgery, if possible, with a number of approaches under investigation.

Graft-Versus-Host Disease

Graft-versus-host disease (GVHD) in stem cell transplantation occurs when antigen-mismatched transplants are required. This affects 40 to 70 percent of allogeneic stem cell transplantation patients who may develop oral GVHD, which involves oral mucosa, salivary glands, and taste. Oral manifestations may occur as the primary manifestations or as part of systemic findings. In the oral cavity, this may present as mucosal "autoimmune" disease (lichenoid, lupus-like, or systemic sclerosis, Sjögrens-like), which may be symptomatic. When symptomatic, topical approaches for mucosal changes employing immunosuppressive agents may provide benefit.[22] Due to chronic immunosuppression, viral reactivation may occur early in transplant, most often due to herpes viruses (HSV, CMV, VZV).

Second Cancers

Patients with prior cancers are at increased risk for cancer recurrence and new secondary malignancies. In patients following stem cell transplantation, increased risk of oral cancers is seen five to nine years after treatment; three-quarters of these patients have GVHD before oral malignancy.[23] The majority of oral cancers following stem cell transplantation are squamous cell carcinoma of the tongue, followed by salivary gland malignancies. The increased risk is related to prior exposure to carcinogens (e.g., tobacco and alcohol), viral cofactors, and immunosuppression, and possibly related to prior cancer therapy.

Survivors of transplant may be at risk for recurrence of the primary cancer and to posttransplant lymphoproliferative disorders, which present in the head and neck and in the oral cavity commonly as gingival masses. Increased vigilance during patient evaluation and thorough examination is critical for early detection.

Conclusion

Acute complications are universal in HNC patients and stem cell transplant patients, and more common than in cycled chemotherapy. Chronic complications of reduced saliva volume and increased saliva viscosity impact quality of life and have been shown to be the primary persisting complications of HNC therapy. Increased risk of dental breakdown and periodontal disease may lead to increased risk of osteonecrosis. Neurosensory changes, including taste and mucosal sensitivity, may persist following cancer therapy.

By understanding the acute and late effects of therapy, we may be able to identify interventions that reduce symptom burden and improve functional outcomes and symptom clusters that may be impacted by oral disease. Prevention and management of these complications is best achieved by integrated oral and medical care of survivors of head-and-neck cancer. ■

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A Century of Scientific Progress

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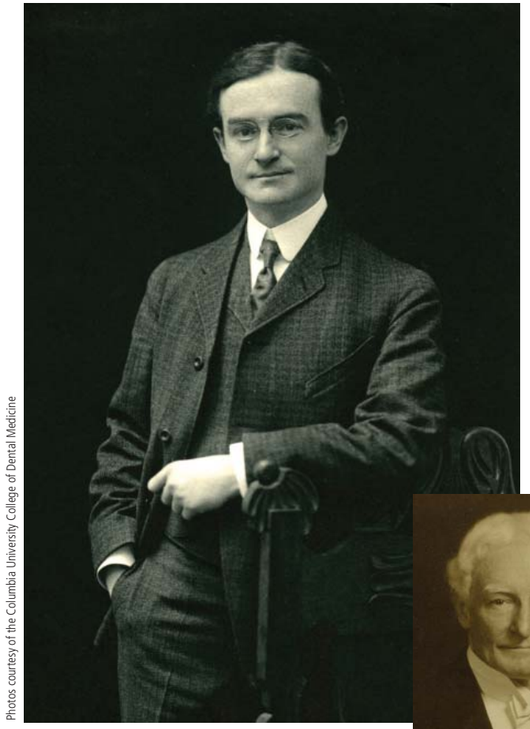
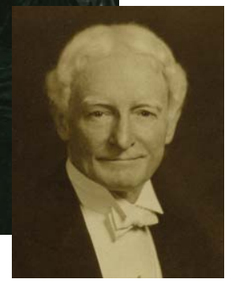
"The highest attainable level of health is a fundamental human right."

—From the preamble to the World Health Organization Constitution





Photos courtesy of the Columbia University College of Dental Medicine

Dr. William John Gies

Preface

The exploration of science as it relates to dental health has placed the profession in a unique position as it enters the 21st century. Periodontal disease and dental caries are now well understood, enabling technological advances in prevention and cure. As genomic research unfolds, dental scientists can begin to explore the pathologies of craniofacial diseases and their genetic underpinning. Their goal is to understand the genetic markers and use them in preemptive therapy to eliminate future disease patterns. Looking back at the 20th century, three areas stand out as turning points in our scientific evolution. The first centered on the physiology of saliva, without which existence becomes tenuous. The addition of the fluoride ion to drinking water necessitated the need for research of the saliva and the oral tissues involved in this genuine public health triumph. The second focused on the microbiology and immunology of periodontal disease, which led to better treatment and prevention. Third, dental education served as the foundation upon which the first two areas were built. The solid university training for dental scientists and clinicians became the basis for modern dentistry's success in this century.

In 1926, the Carnegie Foundation published *A Survey of Dental Education in the United States and Canada, Bulletin Number Nineteen,* written by Dr. William John Gies. The new era of dental education that began after this publication slowly evolved over the century into a new paradigm. Several selected examples are presented herein to show some of the major areas in research and education that are keys to understanding how this revolution occurred. Included are exemplars Drs. Gies and Alfred LeRoy Johnson, as well as the university settings that made their successes possible. Government funding played an important role as well, with the birth of the National Institute of Dental Research (NIDR) in 1947, presently known as the National Institute of Dental and Craniofacial Research (NIDCR). Government monies for new school construction in 1963 (Health Professions Educational Assistance Act) and 1971 (Comprehensive Health Manpower Training Act) also helped change the environment and opportunities available to the new cohort of dental scientists, whose education was financed through the Servicemen's Readjustment Act of 1944, also known as the GI Bill. Politicians realized that good dental health was needed for the nation's overall well-being.[1]

Laying the Groundwork

William Gies was an early dental pioneer who received his PhD in physiological chemistry from Yale University in 1897. At Columbia University, he served as professor and chairman of the Biological Chemistry Department. During the years 1910–1918, he applied his skills as a biochemist to dental research, leading him to realize the great need for a basic science approach to dental pathology. Along with several forward-looking dentists, Dr. Gies was instrumental in creating Columbia University's dental school in 1916. As the first university-affiliated dental school in New York, its entrance requirement of two years of college was among the highest in the nation. In 1919, he established the *Journal of Dental Research* and drew up plans for the International Association for Dental Research, whose first official meeting was at the Columbia University Club in December 1920. He subsequently initiated and negotiated the formation of the American Association of Dental Schools in 1923.

Dr. Gies realized the need to restructure dental education by requiring at least two years of specified undergraduate education prior to entry into dental school, as well as a need to upgrade the medical courses given at the dental schools. He felt a pressing need to attract gifted students who would become exemplary teachers of the future, pursuing full-time careers in dental biological research. There was also an obligation to create specialized postgraduate training for clinical dentistry.[2]

Shortly after *A Survey of Dental Education in the United States and Canada, Bulletin Number Nineteen* was published, two young dental students at the University of Pennsylvania studied it carefully. Upon his graduation in 1928, one of them, Dr. Theodor Rosebury, applied to Columbia for a Gies Fellowship in biological chemistry. Dr. Gies inspired Dr. Rosebury to seek out a career in dental research and teaching. After completing the fellowship, he joined the Department of Bacteriology at Columbia Medical School, and with Dr. Gertrude Foley, formulated a guinea pig infection model to demonstrate his hypotheses. During the 1930s, Dr. Rosebury focused on the biochemical and nutritional aspects of dental caries. Throughout his career, he looked at nutrition as the key element in survival, although his center of attention shifted from human to microbial nutrition. The relationship of nutrition, immunology, and stress on our indigenous microflora to health and disease became the main focus of his career. His guinea pig and rabbit models for this work impacted heavily on modern dental microbiology

Dr. Rosebury's professional duties in the 1930s and 1940s expanded to include investigations defining the relationship of fusospirochetal infections to acute necrotizing ulcerative gingivitis (ANUG). His labors confirmed that the oral cavity was an important site for systemic research that had to be conducted in a thorough and comprehensive manner. Its value to science could extend well beyond the mouth. Dr. Rosebury's studies at nearby Fort Dietrich in Maryland for the United States Army increased his awareness of germ warfare. He was lead author of the book *Biological Warfare*, which was published in 1947. While at Fort Dietrich, he developed a lasting appreciation of the importance of interdisciplinary collaboration, for it was there that he witnessed the devotion, cooperation, and success of a group of scientists from diverse backgrounds.

The Birth of Modern-Day Microbiology and Immunology

Two of Dr. Rosebury's students, Drs. Solon A. Ellison and John B. Macdonald, worked on variations of his model for infection. Both went on to train groups of the most prominent dental researchers in modern-day microbiology and immunology. Dr. Ellison earned his PhD at Columbia and soon joined the staff at the State University of New York–Buffalo, where he started and headed up the Department of Oral Biology and, soon after, administered the first PhD program in this subject. Robert Genco was a member of his staff; Michael Levine, Frank Scannapieco, Lawrence Tabak, Martin Taubman, and Thomas Van Dyke were among the future dental scientists trained in that department.[3] Dr. Levine would inherit Dr. Ellison's mantle and bring the laboratory of biochemical salivary analysis into the post-genomic era. The possibility of successful artificial saliva for xerostomia remained one of his driving forces.[4]

Dr. Macdonald returned to the University of Toronto to establish its first government-funded research lab, and shortly thereafter in 1956, he left to become director of the Forsyth Dental Infirmary for Children in Boston. He held a dual appointment with Harvard School of Dental Medicine, which had affiliated with Forsyth in 1954. Dr. Macdonald's task was to convert the infirmary into a research-based facility.

Photo courtesy of the University of British Columbia

Dr. John B. Macdonald

He chose his staff wisely and selected a young graduate, Dr. Ronald Gibbons, from the University of Maryland, who held a PhD in anaerobic microbiology. He was also joined by a former student, Dr. Sigmund Socransky, from Toronto, who had a penchant for microbiology and became the first clinical scholar at the infirmary. The three worked on the mixed anaerobic infection model using the guinea pig. Also included was Dr. Finn Brudevold from the University of Rochester, who would head a hard-tissue laboratory focused on fluoride studies. Many young scientists, including Drs. Max Listgarten, Richard Ellen, Walter Loesche, Ray Williams, Anne Tanner, Donald Hay, Frank Oppenheim, and Philip Stashenko (current president and CEO of the Forsyth Institute in Boston), trained in this interdisciplinary scientific collaboration that had been developed at both Columbia and Rochester.[5]

Dr. Macdonald left Forsyth in 1962 to become president of the University of British Columbia (UBC) and later executive director of the Council of Ontario Universities. At UBC, he established two new competing universities with scientific underpinnings, Victoria and Simon Fraser. Years later, UBC named the John B. Macdonald Building, which houses the Faculty of Dentistry.[6] His article "Science Education: Backdrop for Discovery" was one of the underpinning ideas for his far-reaching educational programs in higher education. He gave this as his presidential address at the International Association for Dental Research in 1968.[7]

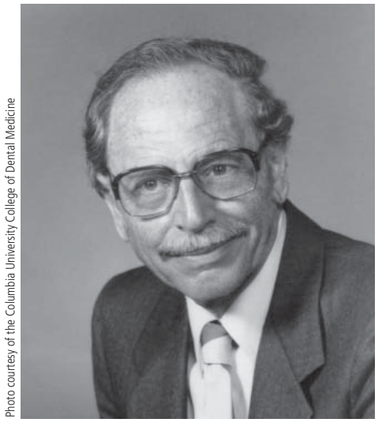


Photo courtesy of the Columbia University College of Dental Medicine

Dr. Irwin Mandel

Dr. John Hein, who held a PhD in chemistry from Rochester, succeeded him at Forsyth and, for the next 29 years, built upon his strong foundation.

During an earlier period, the president of UBC invited Dr. Macdonald to lead a survey on establishing dental education in British Columbia. As part of the lengthy survey, he visited the Harvard School of Dental Medicine, which was one of the factors that led to his being chosen as director of Forsyth. He began his research career at the University of Illinois in Chicago, training with Isaac Schour's faculty in bacteriology and immunology. Dr. Macdonald learned of Dr. Rosebury's work at Columbia and chose to further his studies after completing his master's degree. From the late 1920s through the 1950s, Chicago was one of the best-known areas of dental research due to the leadership of a group of Viennese immigrants.[8]

Salivary Research at Columbia

Columbia Medical School in the early 1940s created an exciting environment in which the young and curious Dr. Irwin Mandel was able to shape his future. His freshman biochemistry teacher, Maxwell Karshan, had demonstrated a special relationship between saliva and dental caries and calculus formation. From 1960 to 1990, Dr. Mandel published more than 100 papers with a major focus on saliva. With his coworkers, he was able to blend the disciplines of basic science and dentistry, creating the field of sialochemistry—the clinical use of salivary analysis for oral disease diagnosis and prognostic purposes. Systemic diseases could now be assessed through the use of saliva. He also played a seminal role in the detailed characterization of the organic components of saliva and their association with oral and gingival health. Saliva became an indispensable tool in oral medicine and preventive dentistry.

The ambience of Dr. Mandel's laboratory set the stage for his large creative output. He worked with highly dedicated technicians, dental students, and foreign dentists trying to establish themselves in the United States. Most of the students were motivated by the desire for an intellectual respite from the rigors of learning clinical dentistry. His excellence as a mentor and teacher motivated future clinical scholars such as Philip Fox, Dana Graves, Roy Stevens, Larry Tabak, and Martin Taubman. Even though the laboratory never had a teaching grant, it had a major effect on many dental faculty members. His modest enterprise provided a vivid example that science was integral to clinical practice and had far-reaching significance for many other researchers.[9]

Bruce Baum, DMD, PhD, lead author of a biographical sketch of Dr. Mandel, has spent much of his career at the National Institutes of Health (NIH), where today he is chief of the Gene Transfer Section, Gene Therapy and Therapeutics Branch at the NIDCR. Dr. Baum's laboratory focuses on gene transfer to salivary glands so that they can produce adequate proteins, facilitating the protective and digestive roles in the mouth and upper GI tract. One of his main targets is to understand the etiology of Sjögren's syndrome, the second most common autoimmune disease in the United States. Presently, with only palliative therapy available, gene transfer is seen as a possible treatment. More research is needed to unravel the biology and chemistry of the genetic system that creates this disorder.[10]

Harvard-trained oral pathologist David Wong, DMD, DMSc, a graduate of Simon Fraser University and UBC Dental School, has been exploring the ability to monitor health status, disease onset, and progression through noninvasive means. Using Dr. Mandel's article on salivary diagnosis as a prelude to his own article, "Salivary Diagnostics Powered by Nanotechnologies, Proteomics, and Genomics," he has become a leader in this subfield. Oral fluid has become a perfect medium

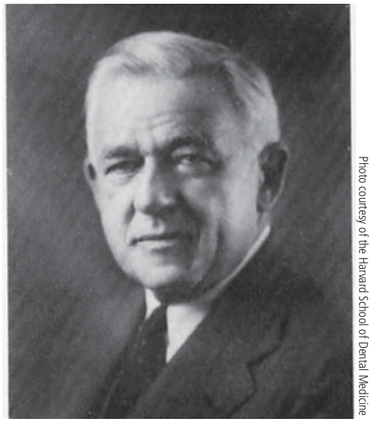


Photo courtesy of the Harvard School of Dental Medicine

Dr. A. LeRoy Johnson

with which to explore health and disease surveillance. Recently, the NIDCR created a road map to identify the specific biomarkers associated with health or disease states and the technologies that can discriminate between the biomarkers. To do this, the NIDCR also funded seven technology groups throughout the United States housed in public and private universities. Dr. Wong believes that the collective efforts and the convergence of salivary diagnostic technologies that include the salivary proteome will present unparalleled opportunities to explore the potentials of saliva in oral and systemic disease.[11] In a more recent paper, "Salivary Diagnostics," Dr. Wong elaborates on the potential of saliva as a mirror of the body.[12] He also recently edited a multiauthored text, *Salivary Diagnostics*. Much of the progress is due to funding from the NIH under the directorship of Dr. Lawrence Tabak.[13] Today, Dr. Wong serves as associate dean of research at the UCLA School of Dentistry.

University Research

During the 20th century, university-trained dental scientists were able to restructure the educational mission of schools of dentistry. Clinical scholarship and new technologies gave dentistry a well-founded scientific background. The profession could then enter the university milieu and contribute to the growth of health sciences.

As a 1904 graduate of Tufts College Dental School, Dr. A. LeRoy Johnson learned early that a dental degree without a college education held little value or respect in the academic world. Trained in the mechanical aspects of orthodontics by Dr. Edward Angle, he realized that dentistry alone could not answer the multitude of biological problems encountered in clinical treatment. His first contact with scientists interested in craniofacial growth and tooth eruption proved that his dental education was lacking in these important areas. After trying unsuccessfully to teach biological orthodontics at both the University of Michigan and the University of Pennsylvania, Dr. Johnson settled into private orthodontic practice in New York City.

The Gies Report had recently been published, and Dr. Johnson called on Dr. Edwin R. Embree of the General Education Board of the Rockefeller Foundation to see if his organization would be interested in funding dental research. Dr. Richard M. Pierce, director of the medical division of the Rockefeller Foundation, had little respect for dental education and suggested funding a medical school program to study oral problems.

Dr. Pierce suggested that Dr. Johnson try his ideas on the deans of a few medical schools at universities where there were no dental schools. Dean Winternitz of Yale was interested and agreed to try the program if the Rockefeller Foundation would finance it. The Foundation granted the money, and two programs were funded. The first, at Yale, admitted a limited number of dental graduates to the first year of medical school, allowing them to progress to an MD degree according to their interest and ability. The second, at the University of Rochester Medical and Dental School, accepted a small number of dental graduates as PhD candidates "to conduct research and to train prospective teachers, investigators, and practitioners in the fundamental biological sciences underlying the problems of dentistry."[14] Earned degrees from respected university programs would allow dental graduates the opportunity to successfully pursue advanced study.

Because the Yale program allowed its graduates to practice medicine, many pursued that profession and disassociated themselves from dentistry, and the dental program eventually closed in 1942. Still, a number of its graduates brought change to academic dentistry. Dr. Lester Burket of the University of Pennsylvania received an MD degree and later went on to become dean of the university's School of Dentistry. His work in oral pathology and oral medicine elevated the status of the disciplines, and the 11th edition of the classic text *Burket's Oral Medicine* was published in 2008. Chief authorship comes from the University of Pennsylvania and includes chapters by 23 specialists on this subject.[15] Dr. Seymour Kreshover, a student of Dr. Burket's, matriculated at Yale and earned a PhD in oral pathology and an MD degree from New York University. He went on to become the third director of the NIDR from 1966 to 1975.[16]

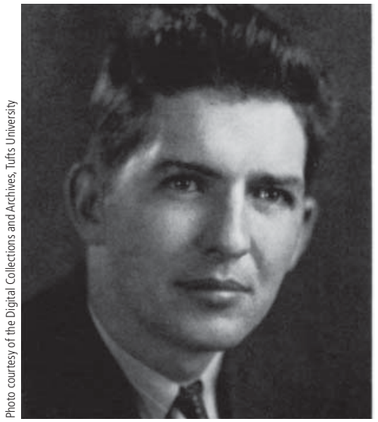


Photo courtesy of the Digital Collections and Archives, Tufts University

Dr. Joseph Volker

The Rochester program educated such dental luminaries as Dr. Basil Bibby and Dr. Harold Hodge, a chemist. As dean, Dr. Bibby brought dental research to Tufts in 1941. In 1947, he returned to Rochester as director of the Eastman Dental Dispensary until his retirement in 1970. At Eastman, he worked with his protégé from Tufts, Dr. Michael Buonocore, who developed the acid etch technique for restorative dentistry and the sealant for preventive dentistry, which changed the dental profession forever. Dr. Hodge, a non-dentist, became chief of the Division of Pathology and Toxicology of the Manhattan Project during World War II and the Atomic Energy Project in 1947. Both would influence such leaders as Dr. John Hein, director of the Forsyth Dental Center, and Dr. Joseph Volker, former dean and chancellor of the University of Alabama system. Both served as deans at Tufts.[17]

In the foreword to a biography of Dr. Volker, Ralph Phillips, DSc, research professor of dental materials at the Indiana University School of Dentistry, noted: "He was a renowned researcher, educator, and administrator, and a pioneer in establishing the mechanism of fluoride in reducing dental caries. He was simultaneously Dean of Tufts College Dental School and the University of Alabama Dental School and eventually a director of a world-famous medical center at the University of Alabama at Birmingham (UAB). He was founding President of a huge university and ultimately the viable Chancellor of one of the nation's leading state university systems."[18] An Indiana native who followed a similar path to Dr. Volker's, including graduate work at Rochester, Dr. Maynard Hine was chancellor of Indiana University–Purdue University at Indianapolis.

In a guest editorial in the *Journal of Dental Education* in 1976, Dr. Volker reviewed Dr. Gies's contribution to dental education 50 years after its publication. *A Survey of Dental Education in the United States and Canada, Bulletin Number Nineteen* was primarily concerned with undergraduate education, but Dr. Gies also recognized the need for dental schools to involve themselves in graduate education. He realized that advanced training was required in the fields of orthodontics and oral surgery to ensure a cadre of qualified dental specialists. Instruction leading to a master's degree or doctorate in basic science was needed to create a pool of dental investigators. Dr. A. LeRoy Johnson had brought this message to the attention of the General Education Board of the Rockefeller Foundation, which subsequently resulted in the formation of the Yale and Rochester postdoctoral programs.

"In retrospect, the key accomplishment of the report was the close affiliation it fostered between schools of dentistry and universities," wrote Dr. Volker in the editorial. "Once this relationship was achieved, the other recommendations were implemented with relative ease. Very quickly, there was an improved relationship with medicine. The quality of instruction in the basic sciences was enhanced, and the number of full-time faculty in clinical subjects increased greatly."[19]

Photo courtesy of the Forsyth Institute

Dr. Percy Howe

Dr. Volker's work was completed by Dr. Charles McCallum who, upon graduation from Tufts in 1951, followed Dr. Joseph Lazansky, his former professor in oral surgery, to UAB. Dr. McCallum earned his medical degree in 1957 and became a well-known academic oral surgeon. He succeeded Dr. Volker as dean of the UAB dental school from 1962 to 1977 and was followed by another Tufts graduate, Leonard Robinson, DMD, MD, who served until 1986. Dr. McCallum was named vice president of health affairs and eventually became the third president of UAB, a position he held from 1987 to 1993. As president, he encouraged interdisciplinary solutions to society's problems and sought to further harness the strengths of the Medical Center and Academic Affairs (University College) into one university—the Academic Health Center. To preserve this unique historical legacy of a modern university, Dr. McCallum established a university-wide archive.[20] Both he and Dr. Volker became distinguished professors with endowed chairs in dental medicine and had buildings named in their honor at UAB.

Senator J. Lister Hill, who served as U.S. senator for Alabama from 1938 to 1969, was a fundamental asset to the success of UAB. His funding for the Health Center, which included the dental school, added to its strong leadership position.[1] It spawned a series of deans who presided over several new university-affiliated dental schools in the southern United States. These institutions include the University of Texas Health Science Center at San Antonio, the Medical University of South Carolina College of Dental Medicine, the University of Mississippi School of Dentistry, and the University of Louisville School of Dentistry.[21]

A New Undergraduate Paradigm

The inclusion of research at the Forsyth Infirmary was incidental. The infirmary's main function was to train pediatric interns who worked on underprivileged children bused to the facility from Boston's inner city. Over time, its mission changed to investigating the cause and eventual prevention of dental decay and periodontal disease. Dr. Percy Howe began his career as a dentist and self-taught salivary researcher. When the Forsyth Dental Infirmary for Children was built in 1912, it included a small research facility. Dr. Howe joined the faculty a few years later and eventually became director of the clinic, as well as chief of research. His affiliation with Harvard Medical School led to many groundbreaking papers on vitamins and the dentition. With his intellectual curiosity, Dr. Howe inspired Forsyth's international interns, who came for one year, to learn pediatric dentistry, bringing fame to the institute and himself. He understood that the problems of oral disease could only be addressed by a solid basic science education.

Dean Charles Sidney Burwell of Harvard Medical School and President James B. Conant of Harvard University asked Dr. Howe to chair a search committee to find a suitable educator to lead the new Harvard School of Dental Medicine (HSDM). One name came to mind: Dr. A. LeRoy Johnson. On October 2, 1942, Dr. Johnson met with Dean Conant, who named him dean of the dental school. This appointment was instrumental in Dean Conant's overall goal for Harvard University: to change its focus from undergraduate to graduate education.[22] Two of Dr. Johnson's early staff members were clinical scholars who had graduated from the Yale and Rochester programs. The first, Dr. David Weissberger, held an MD degree from Yale, and the second, Dr. Reider Sognnaes, had earned his PhD at Rochester.[23]

In 1942, HSDM began a novel four-year program during which the students spent their first two years in the same classes as the medical students. In 1967, following the resignation of the dean, endocrinologist Dr. Roy Greep, Dr. Paul

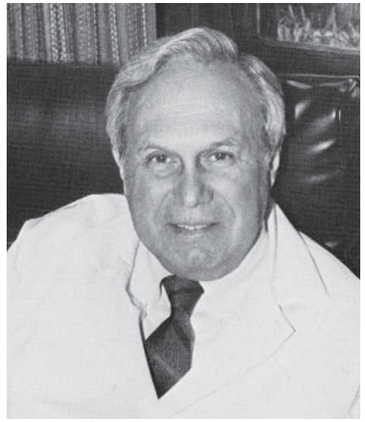


Photo courtesy of the Harvard School of Dental Medicine

Dr. Paul Goldhaber

Goldhaber was chosen as dean with the mandate to better balance the teaching of dental medicine with that of medicine. Dr. Goldhaber had trained in research periodontics at Columbia. For the next 22 years, he oversaw programs that enabled the school to produce a number of skilled researchers who took leadership positions in academic dentistry.

Dr. Goldhaber initiated a doctor of medical science and a master of medical science degree that rewarded scholarship in the dental profession. Areas of expertise were developed in oral medicine, oral pathology, public health, and periodontology. He also worked with Dr. Walter Guralnick in developing one of the earliest dual-degree programs that upgraded oral surgery to a medical specialty.

Developing Future Dental School Leaders

Over the years, other universities, including Alabama, Columbia, and Pennsylvania, adopted this same concept. Oral surgery departments within university-affiliated dental schools have inaugurated research projects in transplantation, bone regeneration, implant surgery, and craniofacial malformations, as well as genomic research. In the early 1970s, Dr. Goldhaber brought in Dr. Chester Douglass to develop the Department of Public Health. With degrees available from the Harvard Schools of Public Health and Public Policy, Dr. Douglass created a major initiative with worldwide recognition.[24]

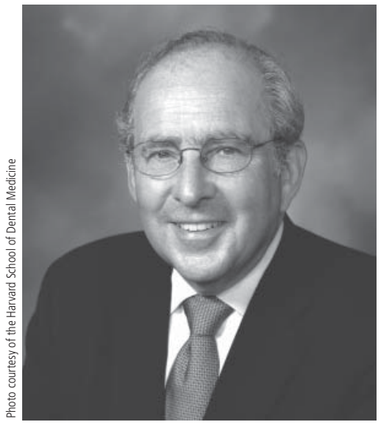


Photo courtesy of the Harvard School of Dental Medicine

Dr. Bruce Donoff

Dr. Bruce Donoff, who holds a DMD/MD degree as an oral surgeon, followed Dr. Goldhaber as dean of HSDM in 1991. He has continued Dr. Conant's mission by growing the programs that have seen its graduates become sitting deans at 10 dental schools nationwide, including Tufts, Columbia, New York University, the University of Michigan, and UCLA. The latter was founded by Rochester graduate Dr. Reidar Sognnaes and today is administered by Harvard alumnus No-Hee Park, PhD, DMD.[25] Dr. Philip Stashenko, the current president and CEO of the Forsyth Institute, is an example of the type of professional that Drs. Percy Howe and A. LeRoy Johnson hoped to create. Dr. Stashenko graduated from HSDM and was awarded his PhD in immunology from Harvard University. He is well respected for his work in cytokine biology.

Conclusion

Recently, Dr. Susan Hockfield, president of the Massachusetts Institute of Technology (MIT), wrote a short essay on the importance of biomedical research to the future of the United States.[26] She noted that innovation-based industries arising from scientific research have been an important factor in our increasing standard of living since World War II. By the early 1950s, the modern research universities, along with the federal funding, were in place. The GI Bill supplied the pool of skilled scientists that were needed. The electronics, nuclear power, communications, and computing industries all grew out of this movement. Today, an accelerating convergence of the life sciences, physical sciences, and engineering has produced a vast array of biomedical innovations. The partnership of government, universities, and industry will be needed to continue the movement forward. The Harvard-MIT Division of Health Sciences and Technology integrates science, engineering, and medicine to solve problems of human needs. The program, now in its fourth decade, is available to medical and dental students.[27]

The future will demand scientific innovation and the technologies that it spawns. Dentistry will need a well-trained undergraduate student body, as well as interdisciplinary scientists, including qualified clinicians. Expanded-duty personnel and auxiliaries will be necessary to bring preventive therapies and curative procedures to our nation's population. Dental education will once again become the primary factor as we enter the second decade of this new century. ■

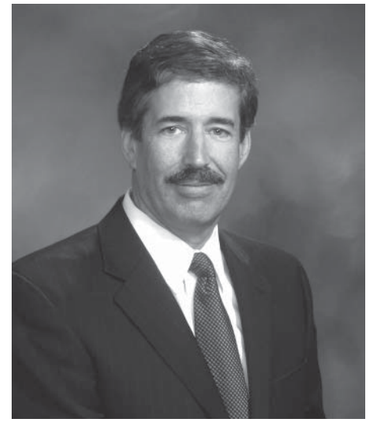


Photo courtesy of the Forsyth Institute

Dr. Philip Stashenko

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Mapping Dental Establishments in Massachusetts Just Before the Recession

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Abstract

Census Bureau data indicate a continuing national increase in the number and size of dental establishments in the time just prior to the recent recession. During this same period in Massachusetts, there were marked increases in the number of individuals employed in dental facilities, and there was a combination of a minor increase of the population and limited proportional increase in the numbers of dentists and dental establishments. This resulted in small changes in population-to-dental facility ratios in most counties in the state. The usual favorable expectations of an economic upswing after a recession for dental establishments may need to be tempered given the increased overhead costs resulting from increased numbers of employees and the fact that the state has the highest dentist-to-population ratio in the country.

Introduction

Between 2000 and 2008, the resident population of Massachusetts increased by 2.1 percent (from more than 6.3 million to almost 6.5 million residents).[1] Between 2000 and 2006—the last year for which this datum is available[3]—the number of professionally active dentists in Massachusetts increased from 5,137 to 5,299 dentists (an increase of 3.1 percent, resulting in a minimal decrease from 1,238 to 1,215 residents per professionally active dentist).[2,3] In 2000, the dentist- and hygienist-to-population ratios were well above the national rates. In 2008, Massachusetts ranked highest in the nation in the number of dentists per 100,000 residents.[4,5]

Mirroring the limited increase in the number of dentists in the state (162 professionally active dentists), there was a comparable increase in the number of dental establishments (172), but an overall increase of almost 3,150 in the number of dental employees in the state (from 18,672 employees in 2000 to 21,818 employees in 2007). (An establishment is defined as a single physical location where services are performed. It is not necessarily identical to a company or enterprise, which may consist of one or more establishments. In addition, one or more practitioners may be present in an establishment. Throughout this presentation, except where specified, the term "dental establishment" refers to those facilities with employees and subject to federal income tax. Government agency programs—hospitals and health department clinics—are not included.[6])

Changing Number of Establishments

Between 2000 and 2007, there was an increase of 70 dental establishments (from 1,288 to 1,358). The major increase in dental establishments (59 facilities) was in Middlesex County. Other counties with notable increases were Bristol, Essex, Norfolk and Suffolk. (See Table 1.)

Statewide, between 2000 and 2007 there was a slight decrease in the number of residents (85 residents) per dental establishment. Among the 14 counties in the state:

- Nine counties had variations of less than 100 residents per establishment;
- Four counties (Bristol, Essex, Franklin, and Middlesex) had decreases that were somewhat greater than 100 residents per facility; and
- Nantucket County had an increase of more than 600 residents per establishment (representing the loss of one of the six establishments during this period). (See Table 2.)

Table 1. Massachusetts Dental Establishments by County: 2000, 2007[6]

	2000	2007	Change 2001–2007
Total State	**2,946**	**3,118**	**172**
Barnstable	124	127	3
Berkshire	71	66	-5
Bristol	184	200	16
Dukes	8	8	—
Essex	353	384	31
Franklin	23	24	1
Hampden	181	179	-2
Hampshire	49	54	5
Middlesex	779	853	74
Nantucket	6	5	-1
Norfolk	417	432	15
Plymouth	208	214	6
Suffolk	242	261	19
Worcester	301	311	10

Variations in the Number of Employees

There has been a progressive increase in the number of employees in dental practices in states throughout the country during the past decades. At the national level (between 1990 and 2007), despite an overall increase of more than 21,000 establishments, there was an actual decrease in the number of "smaller" establishments—those with fewer than five employees. By 2007, 40 percent of U.S. dental establishments had fewer than five employees. Similarly, the number of smaller dental facilities in Massachusetts decreased between 1990 and 2007, with their proportional representation in 2007 below the national level (38.5 percent).

In 2007, the average Massachusetts dental establishment had an average of 7.0 employees (nationally, 6.5 employees) with an average annual salary of $46,800 (nationally, $42,700). Employees may include dentists, dental hygienists, dental assistants, and office staff. (See Table 3.) While there is no such thing as an "average" dental establishment, comparisons between averages (over time and between locales) do provide a picture of the evolving practice of dentistry. The average number of employees was determined by dividing the total number of dental employees in Massachusetts (21,818) by the number of dental establishments (3,118). The average salary was determined by dividing the total annual state payroll figure for dental establishment employees ($1,021,523,000) by the total number of employees (21,818 individuals).

Table 2. Massachusetts Population per Dental Establishment by County: 2000, 2007[1,6]

	2000	2007
Total State	**2,159**	**2,074**
Barnstable	1,800	1,747
Berkshire	1,898	1,970
Bristol	2,913	2,726
Dukes	1,884	1,931
Essex	2,054	1,907
Franklin	3,108	2,985
Hampden	2,522	2,573
Hampshire	3,109	2,864
Middlesex	1,885	1,723
Nantucket	1,595	2,212
Norfolk	1,561	1,517
Plymouth	2,280	2,288
Suffolk	2,854	2,787
Worcester	2,500	2,514

In Massachusetts counties, the proportion of dental facilities with fewer than five employees ranged from 26 percent in Hampshire County to 50 and 51 percent in Dukes and Suffolk Counties and 80 percent in Nantucket County. (See Table 4.)

Dental Establishments with No Employees

In 2007, there were an additional 1,156 Massachusetts dental establishments that were subject to federal income tax, but with no employees. These no-employee dental facilities represented 27 percent of the total number of dental establishments in the state (i.e., 3,118 dental establishments with employees and 1,156 establishments with no employees). The Massachusetts dental facilities with no employees reported a total of $71.7 million in gross receipts (an average of $62,000 in gross receipts per establishment).[8] Nationally, in 2007 there were 39,455 dental establishments with no employees that reported nearly $2.9 billion in gross receipts (an annual average of $73,200 in gross receipts per establishment).[8] Eighty-five percent of the dental establishments in Massachusetts with no employees (987) were located in the Boston-Cambridge-Quincy metropolitan statistical area.

During 2007, most no-employee dental establishments in Massachusetts were individual proprietorships (1,115 facilities) that had average annual gross receipts of $56,700. A smaller number of corporate arrangements (40 facilities) had average annual gross receipts of $205,900.[8]

Given the increasing number of employees per dental establishment with employees, how does one account for the great number of facilities with no employees? Suggested establishment arrangements might include:

- Recent graduates just starting practices
- Older practitioners who are decreasing their time commitment to practice as they prepare for eventual retirement

Table 3. Distribution of Dental Employees by Size of Dental Establishments in Massachusetts and the United States: 1990, 2000, 2007[6,7]

Number of Establishments

	Massachusetts				United States			
				Percent Change				Percent Change
Number of Employees	1990	2000	2007	2000–2007	1990	2000	2007	2000–2007
1–4	1,508	1,285	1,202	-6.4%	57,209	52,036	50,893	-2.2%
5–9	973	1,130	1,198	6.0%	35,750	44,815	50,662	13.0%
10–19	318	454	598	31.7%	9,971	17,007	21,421	25.9%
20–49	50	73	113	54.8%	1,290	2,464	3,204	30.0%
50+	1	3	7	133.3%	111	172	212	23.2%
Total	**2,850**	**2,946**	**3,118**	**5.8%**	**104,654**	**116,494**	**126,392**	**8.5%**
Employees per Establishment								
	5.4	6.3	7.0	NA	5.1	6.1	6.5	NA

Percent Distribution of Establishments

	Massachusetts			United States		
Number of Employees	1990	2000	2007	1990	2000	2007
1–4	52.9%	43.6%	38.5%	54.9%	44.6%	40.3%
5–9	34.2%	38.3%	38.4%	34.2%	38.4%	38.9%
10–19	11.1%	15.4%	19.1%	9.5%	14.5%	15.%
20–49	1.8%	2.5%	3.6%	1.2%	2.1%	2.3%
50+	< 0.1%	0.1%	0.2%	0.1%	0.1%	< 0.1%

Note: Differences in percentage totals are due to rounding.

- An establishment that serves as a secondary activity for an individual who works full-time for someone else
- Contracts with independent outside firms for auxiliary personnel
- Use of family members as auxiliary personnel and where no reports are made for Social Security and income tax purposes
- Establishments of independent corporate arrangements for in-house auxiliaries
- Any number of other alternative practice arrangements (including practice in private homes and other locations) that do not file required quarterly payroll tax reports

Table 4. Dental Establishments by Number of Employees in Massachusetts and the United States: 2000, 2007[6]

	Total Number of Establishments		Fewer Than 5 Employees		Percent with Fewer Than 5 Employees	
	2000	2007	2000	2007	2000	2007
United States	**116,494**	**126,392**	**52,036**	**50,893**	**44.6%**	**40.2%**
Total Mass.	**2,946**	**3,118**	**1,285**	**1,202**	**43.6%**	**38.5%**
Barnstable	124	127	51	45	41.1%	35.4%
Berkshire	71	66	33	20	46.4%	30.3%
Bristol	184	200	54	56	29.3%	28.0%
Dukes	8	8	4	4	50.0%	50.0%
Essex	353	384	144	138	40.7%	35.9%
Franklin	23	24	3	9	13.0%	37.5%
Hampden	181	179	82	61	45.3%	34.0%
Hampshire	49	54	14	14	28.5%	25.9%
Middlesex	779	853	370	358	47.4%	41.9%
Nantucket	6	5	3	4	50.0%	80.0%
Norfolk	417	432	217	193	50.8%	44.6%
Plymouth	208	214	83	74	39.9%	34.5%
Suffolk	242	261	127	133	52.4%	50.9%
Worcester	301	311	100	93	33.2%	29.9%

Just Before the Recession

The dental profession that faced the recent recession is very different from the profession (and population) that confronted the economic reverses in past decades. The combination of a prevention-oriented population, the ability of the profession to provide services unimaginable in the past, and dramatic decreases in the proportion of the population that is edentulous and that has been willing to invest in dental services to retain its remaining dentition bodes well for the future of the profession. In addition, the American Dental Association commented in past economic difficulties that "because patient loads will increase over the long run, an economic recession should prove to be a minor interruption in improving practice conditions."[9] The usual favorable expectations of an economic upswing after a recession for dental establishments, however, may need to be tempered given rapid increases in the number of employees in dental practices and the associated overhead, as well as the fact that Massachusetts has the highest dentist-to-population ratio in the country.

Nevertheless, the increasing size of dental establishments may continue in response to continued third-party inroads into dental practice and the attraction of the next generation of dental students, whose personal experience with dental care would be in a dental establishment with 10, 15, 20, or more employees. ■

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Sugarless Caries

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Figure 1. Sugarless candies to be tested.

In recent years, the author has seen a significant increase in dental decay among his adult patients in his general practice, as have many other dentists. Why have so many dentists experienced the same situation with their patients? Most of these people, having been regular patients for decades, profess their avoidance of sugary drinks, sugary candies, and the many other sources of conventional dietary, fermentable sugars associated with a high caries rate. They claim to rarely sip sodas (regular or diet), a known cariogenic source, and not to consume sugar mints, antacids, candies, chewing gum, or large volumes of citrus (acidic) fruit juices.

In the author's experience, a few of them may just be saying what they think the dentist wants to hear, and a cursory look at their purses and shirt pockets often disclose the tops of candy or mint packages. Most of them are regular patients, sincere, and, over many routine exams/treatments, have not shown any appreciable caries experience for years. Occasionally a restoration will fail or a deep pit will finally etch (decay) into the dentin and require filling-replacement. Yet these are not the patients who have reached old age with the root caries, poorly cleaned teeth, and saliva-drying drug lists so typical of modern-age seniors. These are the 30- to 70-year-olds who brush often, floss religiously, and get regular exams and prophies two to three times a year. They are good patients who give their teeth excellent home care, yet their teeth are decaying like those of plaque-encrusted, Mountain Dew–swilling teenagers. What can be the cause?

Close examination of the patients' diet leads to an interesting phenomenon. Many of these people consume large amounts of "sugarless" candy. They chew sugarless gum, suck sugarless mints, lick sugarless suckers, and consume a whole host of other sugarless confections that are believed to be diet- and dental-safe because they do not contain sugar. The assumption is that they are eating in a healthy manner because the confection has no high-caloric, fermentable carbohydrates—the typical main

Figure 2. Testing equipment: pH paper and digital pH meter.

source of dental decay acids and, thus, tooth decay. This assumption would be wrong.

The question is, however, why do teeth decay if they are bathed in a sugarless product? If there's no sugar, there's no fermentation to lactic (and associated) acid, and thus, no enamel dissolution (decay). This is the way most dentists, sugarless candy makers, and public health people view the process. Teeth will decay if they are exposed to acids with an approximate pH of 5.3 or less.[1–3] With this in mind, one can still have tooth decay without sugar by bypassing the carbohydrate-decay-bacteria-acid-plaque system. How? By just eating acid.

Acid Testing

Since tooth enamel dissolves at a pH of 5.3 or less, anything that brings the oral pH down to such a level (or below) will quickly decay teeth.[1–3] One way of determining whether a particular candy/confection contains acid is to test its pH. A pH level of 7 is considered neutral; anything above 7 is basic and below 7 is acidic. A series of sugarless-labeled candies was purchased at random from a local drug store. Everything labeled "sugarless" was pulled off the shelves and tested using either oral pH test paper (Beutlich, Waukegan, IL) or a Whatman digital pH-microsensor meter (GE Health Care). For the pH paper analysis, several drops of pH-neutral (7.0) water were placed on the candy and measured after a 1-minute delay. The pH meter, when utilized, required several candies to be dissolved in 5–10 cc of pH-neutral water. The meter's sensor bulbs were then immersed in the mixture. Both methods of pH determination used stock laboratory, buffered, pH reference standard (Spectrum Quality Products, Inc., New Brunswick, NJ) to calibrate the meter, pH paper, and pH neutral water. The pH meter measured to the nearest pH 0.1.

Figure 3. Sugarless breath mints being tested using pH paper.

It was more accurate than the pH paper, which measured only to the nearest pH 0.5 with a range of pH 4.5–7.5. The purpose of this study was to roughly measure acidity and identify a previously unrecognized problem, so both systems were applicable to this project.

Results

Most of the candies (and some other sugarless products) tested were citrus-flavored; all were acidic. The diet (sugarless) drinks are listed as a comparison since some patients do not associate coffee, tea, or diet soda with acid and decay. The results are listed in Table 1. All tested products were found to be acidic, although some were more acidic than others. Every 1.0 change of pH represents a 10-fold increase/decrease in acidity: a pH of 4.5 is 10 times as acidic as pH 5.5 and 100 times the acidity of pH 6.5.

Some standard instant coffees and teas, as well as gelatin products, were included because they were advertised as "sugarless" and patients may sip or snack on these products throughout the day. The findings show that a considerable drop in oral pH can occur when consuming sugarless products. It appears that the sugarless candies skip the sugar and deliver acid straight to the teeth. If patients have a caries problem, some sugarless products may not be beneficial and change would be warranted. Patients may be well advised to change their consumption of sugarless candy and beverages based on these findings.

Obviously, the patient's own oral saliva pH, saliva flow rates, buffering capacity (Ca++, HCO3-), and rate of consumption of candies (acid) enter into the formula of whether these products significantly contribute to decay, but individual trends can be easily established. Asking how many candies are consumed during the day, whether they are chewed or sucked, and testing the patients own salivary pH (using pH paper) can assist the dentist or staff members in determining whether the patient's caries problem is sugarless candy–related or not. With the use of pH paper, the candies can be quickly tested in the presence of the patient (place a drop of water on the candy and touch the pH paper to it) for added effect. ■

Table 1. Acid Levels in Sugarless Candy and Beverages.
Tooth enamel dissolves at a pH level of 5.3.

Candy/Beverage	pH	Method (pH paper = p; meter= m)
Generic Walgreens Cough Drop	5.0	p
Sugar Free Popsicles	4.5	p
Breathsavers 3 Hour Mint, sugar free	5.0	p
Pinky Peachment Sugarless Tablets	4.5	p
Sugar Free Altoids, "Simply Mint"	6.5	p
Sugar Free Eclipse Gum, "Winterfrost"	6.0	p
Orbit Sugar Free Gum, "Pina Colada"	6.0	p
Lifesavers Sugar Free:		
Pineapple	5.0	p
Cherry, Watermelon, Raspberry, and Orange	4.5	p
Mentos Gum, "Tropical"	4.5	p
Coastal Bay Sugar Free Fruit Hard Candy	4.5	p
React (5) Sugarless Gum	6.0	p
Jewell Decaf Classic Roast Instant Coffee	5.0	m
Maxwell House Instant Coffee	6.1	m
Chase & Sanborn Special Roast Coffee	6.3	m
Nestea Unsweetened Iced Tea	6.3	m
Sugar Free Jell-O, "Royal Lime"	5.0	p
Luigi's Real Italian Ice—no sugar added	4.1	m
Diet Mountain Dew	3.4	m
Diet Coke	3.3	m
Diet Pepsi	3.0	m
Diet Dr. Pepper	3.2	m
Diet 7Up	3.7	m

Conclusion

You don't need sugar to decay teeth. Many sugarless products contribute significantly to oral acidity and, thus, tooth decay. ■

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Figure 4. Sugarless gum solution being tested with digital pH meter.

Dealing with Difficult Patients

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It has long been known that it's not necessarily what you say, but how you deliver your message that is the most effective tool in dealing with difficult patients. The first step is to identify the people and situations that create the most stress for a dental practice—the "difficult patients." Simply identifying a difficult patient is easy; turning him or her into a great patient and raving fan is quite another story—but it can be done. And sometimes what is discovered is that the difficult patient may not really be difficult after all. Sometimes, the practice's lack of systems and communication can create the difficult patient.

The following protocols identify how to set up the patient visit for success, and how to deal with unfortunate situations and turn that difficult patient into a fan. You'll want to share these with your staff.

The New Patient Experience

The cycle of a new patient begins with that first phone call to the office. The manner in which the phone is answered and how helpful the person answering that phone is will determine a good experience or a bad experience from the patient's point of view. Therefore, it is recommended to have your best communicator answer the phone. Many dental practices have created a special position for just this reason, called the New Patient Coordinator. This person is responsible for the patient's first impression of the practice. Documenting the new patient phone call is equally important. It is crucial to let the patient know what will take place during the first appointment, what to expect, and what he or she needs to provide to help ensure a positive visit.

Financial Systems and Collections

When it comes to the financial aspect of treating a patient, there are two simple rules to follow: Have written protocols for financial options in the practice and inform before you perform. You should discuss the cost with the patient and finalize financial arrangements *before* treatment is rendered. Be positive, confident, and matter-of-fact when discussing the fee and method of payment. If it occurs to you not to ask for payment, it could occur to the patient not to pay, and this is a classic recipe for difficult patients.

Billing Questions

A common scenario for difficult patients is when they receive a statement that they find confusing. The first thing to do is to thank them for calling, because it is difficult for someone to get angrier if you are thanking them. This method can diffuse agitated patients almost immediately. Then, sincerely address their concerns, research the issue, and respond in a timely manner. Verify all the information indicated on the computer and in the chart. Ask leading questions, such as "What questions can I answer about your balance [or statement, or overdue payment, etc.]?" Be concerned and engage your listening skills. When patients reveal that they can't pay the entire balance, ask them, "How much are you short?" The patients may be prepared to pay more than you presume and won't become defensive. Do not presume they can't or won't pay a balance. Ask for their commitment and then commit them to a date when payment will be received in your office.

The Whiner and Complainer

As in all other aspects of life, there are some people who whine and complain, and you will encounter some of these in your practice. Most of the time, they just want to be heard. You should try to ask leading questions to let them know you are listening. "Tell me more" is a magic three-word phrase that can really get patients talking. Is their complaint legitimate? If they are complaining, most likely something caused it. Keep asking them leading questions, and don't let the patients change their story to make you look like the bad guy.

Make sure a front desk staff member follows them into the treatment room and repeats their concern in front of you, the dentist, or another clinical team member. For example, a patient may exclaim at the front desk, "My tooth has hurt terribly the entire time from my last appointment until today!" But back in the operatory, when the dental assistant asks how the tooth has been feeling, the patient says, "Fine." Create more honesty by being present in the situation.

Emergency patients may sometimes fib when trying to get on the schedule. Make sure your staff takes good notes during the phone call and hands those notes off to clinical staff. In this way, you ensure that the same story gets shared with the clinical team as was shared during the phone call.

The Blameful Patient

The same thing works well here. Make sure patients placing blame have an opportunity to relay their concern correctly to all parties. Blame sometimes happens when a patient hasn't finished paying for his or her dentistry, so always make financial arrangements before starting treatment. Some situations require a mediation of sorts. Make sure your patients feel heard.

The Angry Patient

Determine early on what caused the angry situation. Be calm, present, and understanding. Be a good listener and remember not to solve their dilemma or concern too early. Don't patronize them. You should also take steps during any angry situation to continue the conversation when the patient has calmed down.

ReTraining the Difficult Patient

On the Phone

Be an excellent listener. Ask or make leading questions or comments, such as "It sounds like you are frustrated with your billing statement." Write detailed notes. If the complaint is about a team member, be sure to include the team member in the conversation to determine truths or hearsay. Remain calm and concerned. If patients become belligerent, continue the conversation after they have composed themselves and calmed down. Do not raise your voice, as this can just cause more agitation.

When You Receive a Letter

Investigate the truth and respond in a timely manner. Offer to sit down with the patient in person. Remember to document everything and verify all the facts.

Successful Environments

If you're meeting with the patient in person, create a positive environment, and be sure to have good body language and maintain eye contact. Always have a third party present to document the conversation. Do your homework and clarify everything.

Establishing Change

Most people react poorly to change, and changes in the dental office are no different, be it a change to the staff or the fee schedule. When the relationship with a patient's insurance coverage changes, such as your ceasing to be a preferred provider in that particular insurance network, carefully devise what you will say at least six months before you change the relationship with their plan. Clearly state your reasons for the change, and keep in mind that patients will want to know what this means for them. Also, how a patient is introduced to a new team member is crucial. Be sure to have other team members introduce the newest member of your team to the patient so that the patient isn't surprised.

When making changes to financial guidelines, it is important to remain confident. Smile and be matter-of-fact. And try to offer the patient two choices when establishing any changes. This gives the patient a sense of control. Communication really is the key; just keep it positive and simple.

Is It the Patient or the Office?

If you find yourself encountering an inordinate number of difficult patients, you may be asking yourself, "Is it them or is it me?" Recognizing the trends and attitudes in operation at your practice is the key for limiting difficult patients. What is the stress level in the office? If it's high, do you know the cause of the stress? Most dental practices suffer from two things: lack of effective systems and lack of good communication, both of which can lead to stress and poor attitudes.

Releasing a Patient

Like any good business owner, you want to keep your patients; however, in some cases it might be more beneficial to the practice to let the more difficult patient "go." Deciding when that time comes is the hard part. A tolerance level will help you determine if the difficult patient is worth keeping. You should establish rules and guidelines for failed, canceled, and late appointments, and make sure that your entire staff is on board with those rules and enforces them with the patient base.

Also, you should know the state laws regarding what happens if you do not complete dentistry because the patient has not paid his or her bill. In most states, the dentist cannot refuse to complete dentistry when the tooth has been irreversibly altered.

Have a system in place for releasing a patient from the practice. Design a letter to officially release the patient—but be sure to check your state's laws regarding requirements for releasing a patient before you do so. In most states, you must notify the patient in writing. Patients referred for collection of bad debt should be released from the practice immediately upon taking collection action. Two sources for collection guidelines on bad debt and nonsufficient funds check rules and guidelines are the Fair Debt Collection Practices Act (***www.ftc.gov/bcp/edu/pubs/consumer/credit/cre27.pdf***) and Lawdog Publishing (***www.lawdog.com***).

Firing a Patient: What's Legal and What's Not Legal?

You must inform the patient in writing that he or she is being released from the practice. You must give at least 30 days' notice in most states and agree to treat any emergencies during the 30-day time period. Always release patients who permanently refuse their dental care, especially periodontal diagnoses, or patients who have been turned over for collection. Check with the Board of Registration in Dentistry (BORID) for Massachusetts regulations regarding dismissing a patient. ■

A Clinico-Pathologic Correlation

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Figures 1 and 2. Extraoral photograph of the patient shows facial swelling and ecchymosis.

Case Presentation

In January 2009, the oral and maxillofacial surgery service at Tufts Medical Center was consulted to evaluate a 43-year-old female who was transferred to the emergency department from an outside hospital. The patient indicated having a nonsurgical root canal on a maxillary left premolar, #12, performed by an endodontist within the previous 24 hours. The root canal was not completed. The patient noted some discomfort intraoperatively; however, facial swelling started several hours after the first part of the procedure was completed and then progressed, forcing her to seek medical attention.

On initial presentation, the patient was in no acute distress, afebrile with stable vital signs, and initial laboratory data showed a WBC (white blood cell count) of 14.9 (normal is 4.1–10.9x10^3/µL) that quickly dropped to 9.8 after admission. Her medical history was noncontributory; she denied taking any daily medications or supplements and had no history of drug allergy.

Clinically, left periorbital ecchymosis and edema extended from the orbital region to the inferior border of the mandible. She denied any odynophagia, dysphagia, or dyspnea. Lateral pharyngeal and floor-of-the-mouth regions were soft, showing no signs of swelling or ecchymosis. Intraorally, soft fluctuant swelling was present in the maxillary left vestibule, as well as ecchymosis along the left soft and hard palate. Tooth #12 showed signs of recent root canal treatment; however, there were no abrasions, lacerations, or evidence of recent intraoral trauma.

Differential Diagnosis

Hematoma
Subcutaneous emphysema
Cellulitis
Allergic reaction
Angioedema

Radiograph Review

A panoramic X-ray was obtained that showed evidence of gross decay mesial of tooth #4, a three-unit bridge in the upper right quadrant, residual periapical lucency above the apex of the UR molars #2 and #14, and extensive loss of calcified structure on tooth #12.

A face-and-neck computed tomography (CT) scan with IV contrast was ordered to evaluate the progression of the facial swelling. CT findings showed an increased soft-tissue density immediately adjacent to the left maxillary alveolar ridge, measuring approximately 2.5 x 1.6 cm. There was also a mild reticulation of the adjacent fat surrounding the soft-tissue density. There was no significant lymphadenopathy noted, and the salivary and thyroid glands appeared unremarkable. Given the patient's history, the above findings were suggestive for hematoma.

Diagnosis

Hematoma following sodium hypochlorite accident

Figure 3. Panoramic X-ray depicts the offending tooth #12 and the extensive dental history.

Discussion

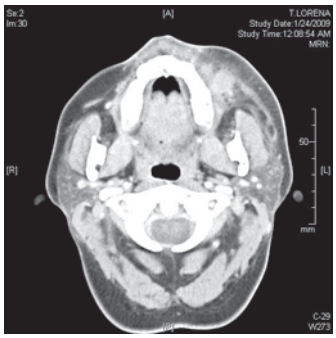
Injection of caustic materials into the tissues, as in the case of sodium hypochlorite (NaOCl), causes necrosis, which leads to separation of the epithelium from the underlying connective tissue, producing a desquamative effect. When NaOCl is inadvertently injected into the bone, the resultant is significant bone necrosis, pain, and perforation to the soft tissues where rupture of the blood vessels occurs, causing the development of hematoma.[1]

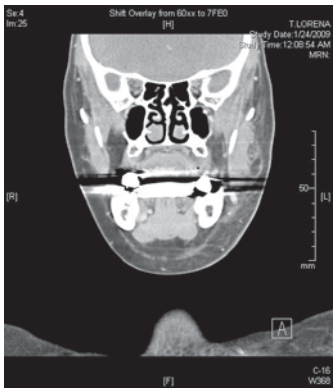
Sodium hypochlorite is routinely used during endodontic therapy as an adjunct to mechanical debridement of the root canal system. It is antimicrobial, dissolves tissue, and provides lubrication; however, it may be very caustic on contact to adjacent soft-tissue beds and vasculature. Upon contact with blood vessels, immediate hemorrhage, ulceration, edema, ecchymosis, necrosis, and stricture are observed. The immediate sequelae have included severe pain, edema, and profuse hemorrhage both interstitially and through the tooth. Reports have described several days of increasing edema and ecchymosis accompanied by tissue necrosis and, at times, paresthesia. The majority of cases resolve within several weeks of the accident.[2] Interestingly, when Kleier et al. surveyed a total of 314 diplomates of the American Board of Endodontics, only 132 reported experiencing an NaOCl accident. The research team found that significantly more women experienced NaOCl accidents compared with men; the condition occurred mostly in maxillary teeth versus mandibular teeth, and more often involved posterior rather than anterior teeth. Patients' signs and symptoms generally resolved within a month. They concluded that NaOCl accidents are relatively rare and that they may be caused by additional factors other than faulty irrigation.[3]

Subcutaneous air emphysema in the head-and-neck region typically develops as a result of trauma, infection, and surgical manipulation. The improper use of air-generating dental instruments during dental extractions and root canals may also result in subcutaneous emphysema without ecchymosis. In some instances, this air may migrate from the head-and-neck region following the path of least resistance through the connective tissue along the fascial planes spreading to distant spaces. The air may enter the retropharyngeal space, which lies between the posterior wall of the pharynx and the vertebral column. It may then penetrate the alar fascia posteriorly entering the Grodinsky and Holyoke's danger space, which communicates with the mediastinum. Once the air collects in this area, it can compress the venous trunks, which may result in cardiac failure, or compress the trachea and thus cause asphyxiation. Some of the severe complications of air emphysema include pneumothorax, pneumopericardium, and mediastinitis. Air emphysema in the dental office almost always develops immediately after exposure to compressed air forced into a wound or the forced entrance of a solution into such a wound.[4]

Excluding our patient's facial swelling, there were no signs or symptoms of acute infection. She continued to deny odynophagia, dysphagia, or dyspnea. She remained afebrile without elevation of her white blood cell count. Her airway remained patent.

Our patient was admitted to Tufts Medical Center for IV antibiotics to prevent infections during the resolution of the hematoma, pain control, and observation. After reviewing the CT images, it was determined that the hematoma was not severe enough to pose a risk for airway embarrassment necessitating surgical evacuation. The patient's two-day hospital course was uneventful, and she was discharged on a two-week course of oral antibiotics. After several weeks of follow-up, the edema and ecchymosis resolved completely. She did not report any paresthesia or sequelae from the condition.





Figures 4 and 5. CT scans show soft-tissue density on the left side with surrounding soft-tissue reticulation.

Conclusion

Sodium hypochlorite accidents are very rare in endodontic practice and although difficult to predict, when they do occur, early intervention is recommended. Supportive treatment is required depending on the severity and should be focused on the patient's symptoms. Prevention is important; therefore, care while using caustic products is highly recommended. ■

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LATERAL PERIODONTAL CYST

The lateral periodontal cyst is a developmental (noninflammatory) cyst that arises in the alveolar bone along the lateral portion of an erupted vital tooth. Such lesions are radiographically indistinguishable from other odontogenic lesions that frequently occur in this location, such as the odontogenic keratocyst, and from lateral radicular cysts that arise secondary to loss of tooth vitality. Typically presenting in adult patients, the lateral periodontal cyst is often asymptomatic and first noted during the course of routine radiographic examination. Although the canine-premolar region of the mandible is the most common location for the lesion,[1] when such lesions arise in the maxilla they typically occur in this same region of the dentition. While most often characterized as a solitary cystic cavity, in some instances the lesion is multicompartmentalized. This multilocular variant is termed the botryoid odontogenic cyst, and a diagnosis of such may portend a higher likelihood of recurrence than its unilocular counterpart.[2]

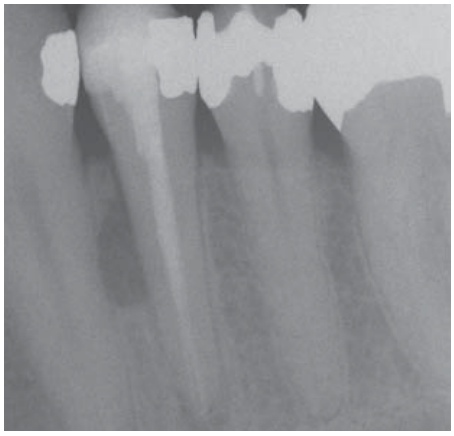

Figure 1. Radiolucent lesion thought to represent a cyst of inflammatory origin. Following enucleation, the lesion was diagnosed as a lateral periodontal cyst.

Though relatively uncommon, a familiarization with this entity is important when forming a differential diagnosis for a radiolucency presenting in a lateral-radicular location. Assessment of tooth vitality is an essential step to avoid unnecessary endodontic therapy and to direct appropriate treatment.

Conservative surgical excision is the standard of care, with submission of lesional tissue for histopathologic evaluation. Given the higher incidence of recurrence in the botryoid variant, patients with this diagnosis may require periodic radiographic follow-up evaluation. ■

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Incidental Dental Radiographic Findings: Dense Bone Islands

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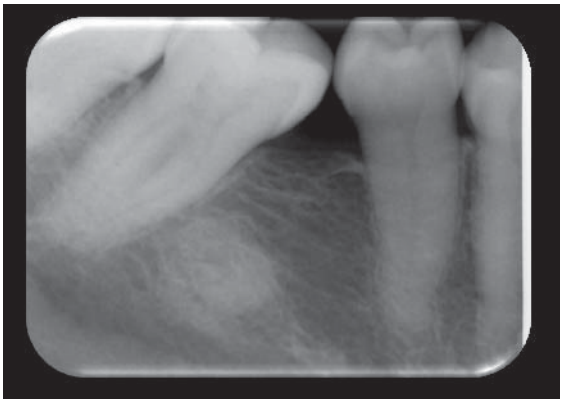

Figure 1b. Periapical radiograph showing the radiopacity in the right posterior mandible.

Figure 1a. Panoramic radiograph showing the radiopacity in the right mandible.

Dense bone islands are synonymous with enostosis or idiopathic osteosclerosis. These present as areas of increased osseous density or radiopacities in the maxilla or mandible with defined borders, located at or around the apical regions of teeth, interradicular area, or with no apparent connection to the teeth. There is a large variation in size ranging from a few millimeters to about 2 centimeters. The effect on adjacent teeth may include indistinct lamina dura and periodontal ligament space and root resorption. The associated teeth are usually asymptomatic. Dense bone islands do not cause osseous expansion; hence, these do not affect the fit of prostheses.

The differential diagnoses for such radiopacities in the jaws could include benign cemento-osseous lesions and inflammatory lesions such as apical sclerosing or condensing osteitis. Dense bone islands may be distinguished from the aforementioned categories by the presence of intact lamina dura and/or periodontal ligament space. However, it may not always be easy to discern the continuity of the lamina dura and periodontal space due to the inherent superimposition of structures in conventional two-dimensional radiography.

Histologically, dense bone islands are characterized by obliteration of marrow spaces by heavy trabeculation or dense cortical bone. The quality or density of bone in the edentulous areas is an important predictor of dental implant success. The available bone can be classified by using the Lekholm and Zarb (1985) classification, in which the quality of bone is divided into four subtypes based on density as follows:

- Type 1: Almost entire jaw is comprised of homogenous compact/cortical bone
- Type 2: A thick layer of cortical bone surrounding a core of dense trabecular bone
- Type 3: A thin layer of cortical bone surrounding a core of dense trabecular bone
- Type 4: A thin layer of cortical bone surrounding a core of low-density trabecular bone

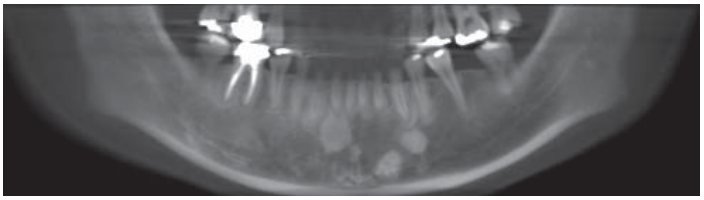

Figure 2a. Panoramic image reconstructed from cone-beam CT data showing multiple radiopacities in the mandible depicting dense bone islands.

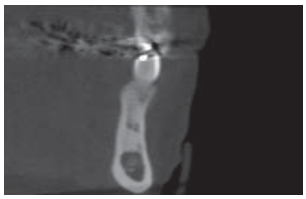

Figure 2b. Cross-sectional image reconstructed from cone-beam CT data showing absence of buccolingual expansion or thinning of the mandibular cortical plates associated with the dense bone islands.

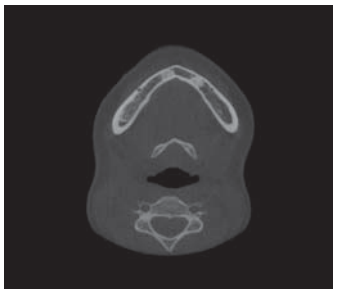

Figure 2c. Axial image reconstructed from cone-beam CT data showing multiple enostosis/ dense bone islands.

A dense bone island is Type 1 bone. Its density provides good cortical anchorage, which is necessary for immediate functional loading of dental implants. However, this type of bone has limited vascularity.

Figures 1a and 1b represent radiographic presentation of enostosis or idiopathic osteosclerosis in the right posterior mandible in panoramic and periapical radiographs, respectively. The dense bone island presents a defined circular corticated radiopacity located mesial to, but not attached to, the mesial root of the second molar in the edentulous region corresponding to the first molar. The periapical radiograph confirms the panoramic presentation. Figures 2a–2c represent reconstructed images from cone-beam computed tomography (CT) data, showing location and characteristics of multiple dense bone islands. ■

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CLINICAL CASE STUDY

PHILIP MILLSTEIN, DMD, MS

Dr. Millstein is a prosthodontist with a practice based in Cambridge. He is editor of the Middlesex District and a former MDS Trustee.

TREATMENT FOR A MALPOSITIONED OCCLUSION IN A PATIENT WITH ACROMEGALY

A 62-year-old Caucasian male presented for treatment of a mandibular dysfunction. His chief complaint was that he could "pass a magazine through his teeth," and he had trouble eating. Over the last year and a half, his jaw had been shifting. Ultimately, he was diagnosed with a benign adenoma of the pituitary gland, which resulted in a diagnosis of acromegaly. This disease often affects jaw positioning by causing an uncontrolled growth of the bones in the front of the skull. Treatment consisted of intracranial excision of a part of the pituitary gland. The results appeared to be positive. No medication was required.

The patient was then referred to an oral surgeon, who recommended extensive surgical procedures to help reposition the mandible. The patient refused surgery, and nonsurgical consultation was sought. Conventional dental recommendation included removal of the offending crown (#31) with an attempt, if the jaws came together, to equilibrate and ultimately close the occlusal gap. The results of the nonsurgical procedure are shown in the accompanying figures.

Clinical procedures were followed. Upon removal of the crown, the jaw repositioned itself. Occlusal equilibration was required over several weeks until anterior and posterior occlusal contact was made. Once equilibration was complete, a crown was fabricated for tooth #31. The original occlusion was never regained and never could be—even with surgical procedures. The patient was confident that he could work with such a positive result. ■

Disclaimer: The system used in the treatment of this patient is presently unavailable. It is being developed by the author for eventual commercialization.

Figure 1a–1c. Pretreatment.

Figure 2. After removal of the crown (#31).

Figure 3. After occlusal equilibration.

Figure 4. Final result.

Give Us Your Feedback on This Clinical Case Study

A Clinical Case Study is a written and visual assessment of a clinical case where the author presents before-and-after radiographs and/or photographs as a means to discuss the diagnosis, treatment plan, and actual treatment of a particular situation. The purpose of this study is to encourage Journal readers to contribute a clinical response to the cases presented.

Please address your correspondence to Clinical Case Study, Journal of the Massachusetts Dental Society, Two Willow Street, Suite 200, Southborough, MA 01745, or email ***mcarman@massdental.org***. Include your name, address, and phone number or email address so that we may contact you for follow-up. Responses may be published in a future Journal.

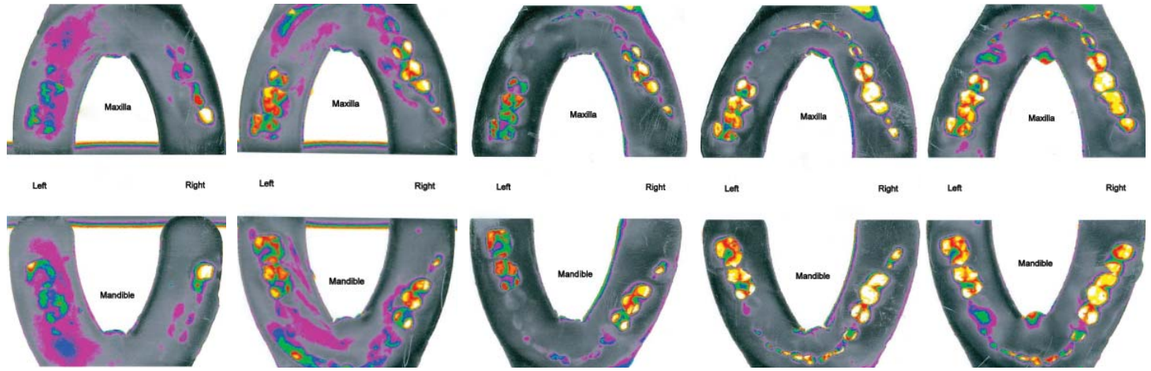
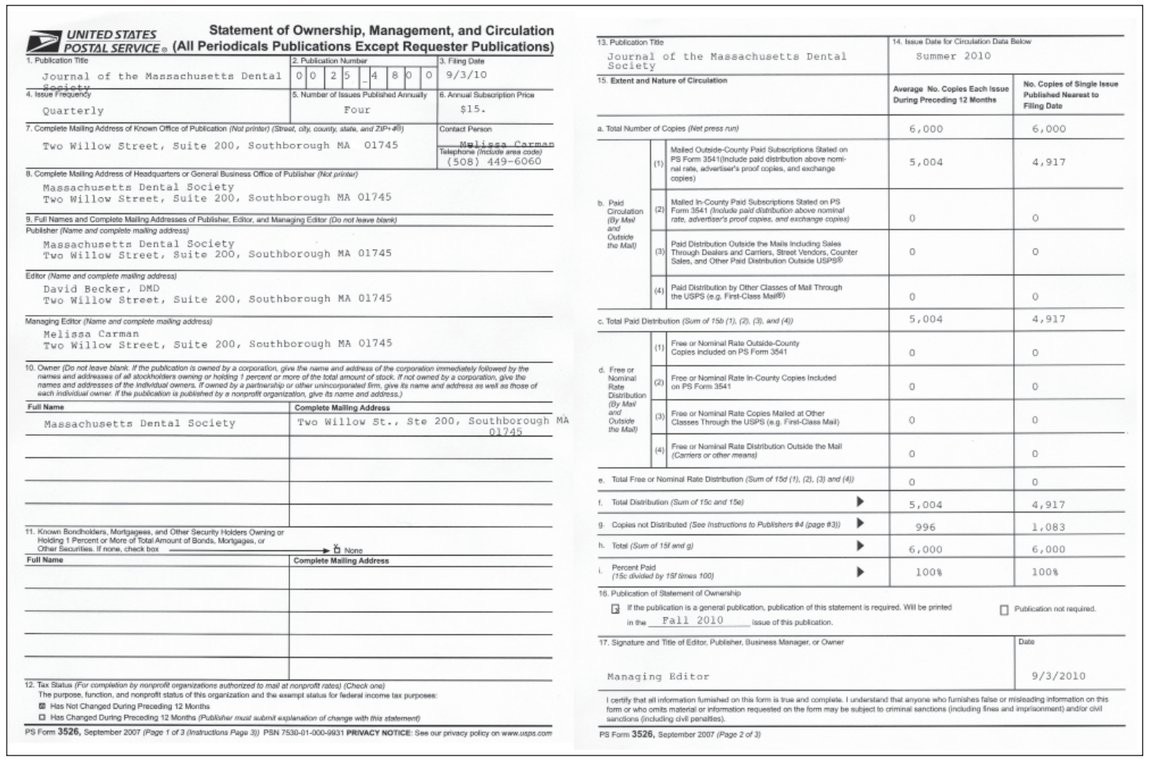


Figure 5a. Pretreatment. **Figure 5b. After removal of crown (#31).** **Figure 5c. One week later.** **Figure 5d. After completion of occusal equilibration.** **Figure 5e. Final result (replacement of crown #31).**



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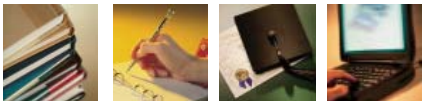


DENTAL EDUCATION

MELISSA CARMAN, MANAGING EDITOR

Highlighting key events taking place in dental education in Massachusetts.

Tufts University

The awards are rolling in for Tufts University School of Dental Medicine's vertical expansion project, including LEED Silver Certification from the U.S. Green Building Council in recognition of its sustainable design.

The Kneeland Street building, which was formally dedicated last November, conforms to LEED (Leadership in Energy and Environmental Design) standards, the nationally recognized green building model. The five-story expansion's 1,700 new windows, designed to bring more light into the building's interior, contribute to its energy-saving profile.

In addition, TUSDM has been acknowleged with a Building of America Award, which recognizes the country's most innovative construction projects; the Boston Society of Architects Honor Award for Healthcare Facilities Design; and the International Facilities Management Association Boston Chapter's Best Practice Award.

Boston University

Dr. Gregory Stoute (right) and Dr. Jean-Marie Betty at one of the clinics.

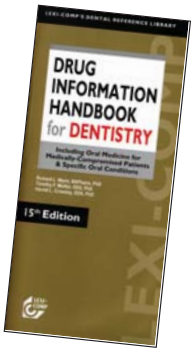
This past June, Gregory Stoute, DMD, traveled to Jamaica as part of an oral health outreach mission organized by the Jamaica Awareness Association of California. Dr. Stoute, an associate professor and director of minority affairs at the dental school, helped provide preventive care, cleanings, and extractions to approximately 130 patients over the course of the five-day mission.

"In Jamaica, even people who have jobs—the working poor—are in many cases unable to afford dental care," said Dr. Stoute. "At one point in the trip, many workers from the hotel we were staying at came in for a dental visit. As in so many nations, including the U.S., and even Massachusetts with the recent MassHealth coverage cuts, working people just do not have access to care."

For the past 30 years, Dr. Stoute has been involved in outreach efforts worldwide, including missions to the Caribbean, South Africa, and South America. ■

NORMAN BECKER, DDS, EDITOR EMERITUS

Drug Information Handbook for Dentistry—15th Edition



RICHARD L. WYNN
TIMOTHY F. MEILLER
HAROLD L. CROSSLEY

Lexi-Comp Reference Library

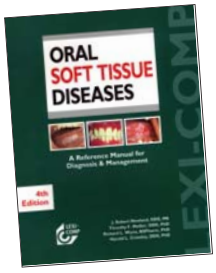
This book should belong in every practitioner's office. Although it includes the caveat that it is "intended to serve the user as a handy reference and not as a complete drug resource," the book contains information on more than 1,600 commonly used drugs.

The cross-references are easy to navigate and explain the specific use of the drugs, including their function, doses, and possible adverse reactions. The book includes drug monographs that list generic names, cross-references, sample prescriptions, brand names, pharmacologic category, and unlabeled/investigational use.

This very useful handbook also details adverse effects, restrictions, dental uses, dosage, mechanism of action, contraindications, warnings/precautions, and drug interactions (e.g., metabolism effect, avoidance of concomitant use, increased toxicity, decreased effect, dietary considerations, duration of action and half-life, and pregnancy and lactation considerations).

There is no doubt in my mind that this handbook has had a positive effect on my prescription-writing habits, and my patients can only benefit from the extra knowledge I have gleaned from this resource.

Oral Soft Tissue Diseases—4th Edition



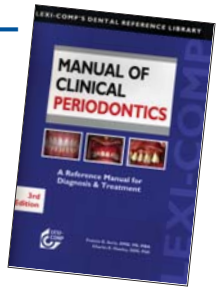
J. ROBERT NEWLAND
TIMOTHY F. MEILLER
RICHARD L. WYNN
HAROLD L. CROSSLEY

Lexi-Comp Reference Library

The fourth edition of this reference manual describes white lesions, red lesions, ulcerated lesions, blistering/sloughing lesions, pigmented lesions, and soft-tissue enlargements in a clear and easily accessible manner. The editors use labeled tabs for clear organization of the topics, making the material easy for the user to navigate. They also utilize clear and precise photographs and text to identify etiology, typical visual clues, useful clinical information, differential diagnosis, diagnostic steps, treatment recommendations, follow-up suggestions, and clinical significance for each of the lesions under study.

A drug section with sample prescriptions, as well as special topics—such as management of the patient undergoing cancer therapy, dry mouth syndrome, fluoride, antibiotic prophylaxis, HIV infection and AIS, and normal blood values—makes this manual a great teaching tool.

Manual of Clinical Periodontics—3rd Edition



FRANCIS G. SERIO
CHARLES E. HAWLEY

Lexi-Comp Reference Library

This book is more than what it claims to be: "A Reference Manual for Diagnosis & Treatment." It is a teaching tool for practitioners and students alike.

Starting with an introduction to health and disease, as well as evidence-based decision making, the authors cover normal anatomy, histology, and physiology of the peridontium, followed by the classification of periodontal diseases, assessment, diagnosis, treatment planning, and therapeutic endpoints.

The tabs allow for an easily referenced manual of all facets of periodontal care, including: prevention and maintenance; nonsurgical therapy; surgical principles; resection and regeneration; periodontal plastic surgery; periodontal emergencies; and implant considerations.

The authors' use of photographs and illustrations, along with a useful bulleted question-and-answer format, makes this manual a valuable addition to any practitioner's library. ■

VIEWPOINT

ROY A. SCHONBRUN, DDS

Dr. Schonbrun practices oral and maxillofacial surgery with Connecticut Valley Oral Surgery Associates in western Massachusetts.

NOT-SO-ACTIVE INGREDIENT?

Remember the old Billy Crystal–Christopher Guest *Saturday Night Live* sketch with two guys making claims of outrageous human feats, such as shoving a meat thermometer in an ear and then banging it in with a ball-peen hammer? Now we are expected to believe the claim of one former Massachusetts state legislator that sorbitol in his toothpaste caused him to fail a home-based breathalyzer test assigned as part of his court-ordered probation, following his arrest leaving the scene of a drunk-driving accident last year. He claimed that sorbitol, a sugar alcohol contained in many toothpastes, has been reported to trigger positive breathalyzer results. This shocking revelation, by a politician no less, could create a whole new category of designated driver—those who don't brush or rinse. Or it could empower the Registry of Motor Vehicles to rescind the licenses of those who do brush their teeth. Sorbitol has been sorely victimized and, since mouthrinses and dentifrices are our profession's *aqua vitae*, it is time to set the record straight.

Sorbitol, aka glucitol ($C_6H_{14}OH_6$), is called a sugar alcohol, but it is actually neither a sugar nor an alcohol. It is a naturally occurring carbohydrate (polyol) found in plants or manufactured from sugars and starches and used as an artificial sweetener. It is found in sugar-free gum, candy, diet sodas, and, yes, toothpastes and mouthrinses, including whiteners. Although the word "alcohol" is used in its name, sorbitol cannot get you drunk. It is not completely absorbed into the bloodstream and ferments in the small bowel, which can cause bloating, gas, and diarrhea—embarrassing, but not inebriating. I looked on the shelves of a local CVS and found dozens of dental-related products—toothpastes, mouthrinses, and whitening agents—and most of them contained sorbitol. No hazardous labels, however.

Now, I suppose if you fed enough sorbitol to a lab rat and put the critter behind the wheel of a car, an accident might ensue. But that is not sufficient proof for this investigator to condemn sorbitol as the causative agent. It is clear, however, that mouthrinse and toothpaste can be used successfully to mask other imbibed substances—such as alcohol. For example, when your kid leaves the house at night smelling minty-fresh and comes home many hours later still smelling minty-fresh, you may find tire tracks across the front lawn the next morning.

I think it is safe to say that we, as dental practitioners, can continue to recommend to or, rather, compel our patients with confidence to use toothpaste and mouthrinses without contributing to their delinquency and without increasing our malpractice premiums.

Which brings up another scathing condemnation of a commonplace dental medicament that has hit the media recently: denture adhesive. An article published in the *Fort Worth Star-Telegram* claimed that denture cream caused severe neurological disorders in a previously healthy 26-year-old woman. Actually, it is the zinc in the adhesive that is the damnable element. According to the article, there is enough zinc in these denture pastes to coat the bottom of a battleship, and it is responsible for severe disruptions of our axons, dendrites, and synapses. Overexposure to zinc can cause numbness and tingling of the extremities, head, and neck. According to the National Academy of Sciences, the largest daily tolerable intake of zinc is 8–11 mg, while researchers at the University of Texas Southwestern reported that denture cream test subjects averaged intakes of 300 mg of zinc daily. It is indeed a most unfortunate, but perhaps avoidable, occurrence. A better-fitting denture, new or re-based, could have helped. Reading the directions regarding proper application (frequency and amount) might have proved precautionary.

But rather than take this at face value, I decided to do my own investigation into the matter of zinc and denture adhesives. The connection of zinc to neurological disorders relates to the balance between zinc and copper. An excess of zinc can cause copper depletion neuropathy. It is still unclear as to the bioavailability of zinc in denture creams, but reports of copper deficiency myeloneuropathy and zinc excess consistently indicated daily amounts of zinc oxide–containing denture adhesive and duration of use far in excess of the levels recommended by the manufacturer. I suspect that the switch now to nonzinc denture creams containing polymethylvinyl ether, when misused to excess, could also lead to interesting pathological findings in the future. Frankly, I think that vinyl should be used on the outside only. Dentists should continue to recommend zinc and nonzinc denture creams to their patients, but perhaps with a more cautionary tone.

What's next? The hazards of choking on dental floss?

Editors' Note: The opinions expressed in this Viewpoint do not reflect the opinions of the Journal, *its editors, or the Massachusetts Dental Society.* ■