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.
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 in competent orthodontic instructors in university-based programs, do not comprehend the nuances of orthodontic diagnosis, treatment planning, biomechanics, and growth and development. This is not demagoguery; 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.

Vincent DeAngelis, DMD

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 invalid and irrelevant, 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 consistently treated Straight Wire cases, more than 90 percent had root resorption. They 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 intracoronal 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). 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 sinuses by the molar palatal roots. These findings were not discernible radiographically. The authors opined that the action of intrarotational force by the rectangular archwire was directly responsible for this irreversible damage to the roots and associated 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 Straight Wire Appliance, as modified by Roth,1,2 is programmed to deliver intrarotational torque, a procedure that Thowur and biomechanics in engineering dentofacial orthopedics, warned should be avoided due to its inadvertent, superimposed root apex erosion. 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 parodontal structures over ideal alignment with biologically sound physiologic, nonpathologic results. “Do you want root apices and intact parodontal tissues at the end of treatment or ideal occlusion?” The discerning clinician should demand both.

References
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 EARNED A 5.5 PERCENT RETURN AND $10,000 IN ANOTHER STOCK FUND THAT EARNED 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.
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 fuming. 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 co-pays. 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.
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 VARIOUS 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 BIO, 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, ENCOURAGING 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 FOUNDATIONAL 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, SUCH AS 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’RE NOT RANKING HIGH ENOUGH 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 TO 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 ORMESSAGES 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 EARN 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 STRATEGIES AND SOLUTIONS 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 patients requires inter disciplinary care. The purpose of this article is to review the common oral complications in oral 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 symptoms, 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, xerostomia, nausea, vomiting, and fatigue. Thrombocytopenia, anemia, and neutropenia may develop, along with other side effects associated with chemotherapy, for example, peripheral neuropathy, nausea, and vomiting. These side effects can result in chronic neurosensor y symptoms; salivary gland dysfunction may also be chronic. 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 with orofacial pain 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 post radiation therapy has been identified as a common persistent symptom, including dry mouth (92 percent), change in taste (75 percent), and xerostomia (40 percent). The majority of patients experienced pain (58 percent), and 17 percent rated pain as moderate or severe within the past month. Measurements of salivary flow after IMRT, where the major glands are spared high-dose exposure confirm less severe hyposalivation and improved quality of life. Oral complications during and following cancer therapy depend upon the disease under treatment, the stage and location of disease, the medications and dosage, the schedule of therapy, and any patient comorbidities, including individual susceptibility. Radiation and chemotherapy may affect oral tissues, mucosa, salivary glands, neurosensory function, dentition, periodontium, and musculature 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 and may lead to more severe and prolonged effects on oral tissues. Posttreatment chemoprevention and management of radiation-induced complications may be necessary, and the benefit of maintenance chemotherapy and to develop a bolus for depletion. Dietary shifts are seen in HNC patients, affecting taste changes, and increasing consumption of high-carbohydrate foods of mood or pureed consistency. Saliva also possesses antimicrobial and remineralizing properties 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. Salivary flow may be decreased during and following radiation therapy. The impact of advanced radiation technology, such as intensity-modulated radiation therapy (IMRT) to spare salivary tissue, has been demonstrated in HNC patients. Quality of life has improved in patients undergoing IMRT compared to conformal radiation therapy. Xerostomia and other symptoms associated with dry mouth may be important in protecting against dental damage. Prevention requires excellent oral hygiene and a noncariogenic diet. The bacterial component can be managed with chlorhexidine rinse. Remineralization of tooth surfaces can be achieved with 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 Chemotherapy in breast cancer patients has been shown to cause mucosal lesions, affect salivary function leading to a macropolyp to carious and fungal flora, and cause taste change that may persist for more than six months. Decreased phosphate and secondary IGA food molecules to taste receptors and to develop a bolus for degradation. Dietary shifts are seen in HNC patients, affecting taste changes, and increasing consumption of high-carbohydrate foods of mood or pureed consistency. Saliva also possesses antimicrobial and remineralizing properties 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. Salivary flow may be decreased during and following radiation therapy. The impact of advanced radiation technology, such as intensity-modulated radiation therapy (IMRT) to spare salivary tissue, has been demonstrated in HNC patients. Quality of life has improved in patients undergoing IMRT compared to conformal radiation therapy. Xerostomia and other symptoms associated with dry mouth may be important in protecting against dental damage. Prevention requires excellent oral hygiene and a noncariogenic diet. The bacterial component can be managed with chlorhexidine rinse. Remineralization of tooth surfaces can be achieved with fluoride and by providing calcium and phosphate in the oral environment (remineralizing products).

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pain following treatment is reported in the majority of patients at follow-up be-
tween six and 12 months and likely con-
tinues indefinitely. 34 The persistence of mucosal sensitivity may be due to atro-
phy of the mucosa, mucosal neuropathy, and hyposalivation. Chemotherapeutic agents may result in peripheral neuro-
opathy, including orofacial neuropathy. Post-
radiation 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 compli-
cations 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 pleas-
sure or desirable flavor, and loss of umami has been suggested to have the strongest correlation with impact on quality of life. 35 Taste is mediated by epithelial re-
ceptors, is impacted by hyposalivation, and may be affected by microbial shifts and retention of food in the mouth. Ad-
ditionally, 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 or with and without chemotherapy. 36 Recov-
ery of taste is variable, in some studies improving in two to six months follow-
ing cancer therapy, although taste change may continue indefinitely. The impact of taste change includes reduced inter-
est 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 chemo-
therapy, such as that received by breast cancer patients. Chemotherapy may be secreted in saliva, resulting in taste change until the drug is cleared; how-
ever, taste change may continue due to direct damage to taste receptors. Tissue necrosis, oral bleeding, and postsurgical wounds may contribute to taste change, halsaltosis, and altered smell. Taste dis-
orders may also follow oncologic surgery, which may damage the lingual branch of the glossopharyngeal nerve or the chorda tympani, resulting in hyposalivation. 37

IMRT may spare salivary glands and thus reduce the impact of radiation therapy on taste. However, low-dose ir-
radiation to areas of the oral cavity may impact taste. Radioprotectors, such as amifostine, may have utility in protecting taste by protection of tissue or indirectly by maintenance of saliva. 38 Dietary counseling/modification, addi-
tion of seasoning to food, avoidance of unpleasant foods, and food rotation are recommended. Local infection and hypo-
salivation should be managed if possible. Zinc supplementation may affect taste dysfunction. 39

### Postradiation Fibrosis

Radiotherapy and surgery may lead to limited oral opening, limited mobility of the tongue, and trismus that may af-
fect 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. Radia-
tion-induced fibrosis has been reported in patients with squamous cell carcinoma of the head and neck. 40 Trismus may be exacerbated by modifying radiation therapy fields and by introduc-
ing active jaw range-of-motion exercises during radiation therapy. Pentoxifylline, which affects fibrogenic cytokine produc-
tion, has been shown to improve established trismus 41 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. Reactiva-
tion of latent organisms and exacerbation of chronic oral infections, including dental and periodontal infection, may occur. Cancer therapy may lead to changes in oral mi-
crobiota that can lead to infection. Chemotherapy can compromise oral mu-
osal immune defense mechanisms and reduce antinociceptive functions of saliva, myelosuppression and immunosuppres-
sion may lead to exacerbation of pre-
existing sites of chronic infection or pre-
dispose the patient to new infection and increase the risk of systemic infection. Latent herpes simplex virus infections ex-
acerbate when host immune defenses are compromised due to malignant disease or the chemotherapeutic regimens. Manage-
ment may include prophylaxis for sero-
positive patients who will become myelo-
suppressed, 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 mar-
row. Oral hemorrhage can occur when platelet counts are below 25,000/mm³, is more likely in patients with gingivitis or periodontal disease, and may occur in ulcerative oral mucositis.

### Neurotoxicity

Some chemotherapeutic agents are neu-
rotoxic (e.g., vincristine, platinum agents, and taxanes) and may lead to orofacial dysaesthesia and pain that can be compounded by dental 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. The symp-
Infection, nutrient/caloric demand, GI dysfunction

### Compromised Nutrition

Compromised nutrition may occur due to nausea, emesis, and altered oral func-
tion. Oral function may be affected by hyposalivation, taste change, xerosthyn-
Chorda tympani

### Table 1. Chronic Oral Complications of Cancer Therapy

<table>
<thead>
<tr>
<th>Oral Complication</th>
<th>Potential Direct Risk Factors</th>
<th>Potential Indirect Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyposalivation</td>
<td>Radiation, chemotherapy</td>
<td>Dehydration, medications, anticholinergic, antihistamine, antidepressants, antihypertensive, and analgesic drugs</td>
</tr>
<tr>
<td>Dental demineralization/caries</td>
<td>Hyposalivation, compromised oral hygiene, microbial shifts, diet change</td>
<td>Antibiotics causing microbial shifts; emesis, reflux</td>
</tr>
<tr>
<td>Dental sensitivity</td>
<td>Dental hypersensitivity, gingival recession, dental demineralization</td>
<td>Neuropharynx</td>
</tr>
<tr>
<td>Periodontal attachment loss</td>
<td>Dental hypersalivation, oral hygiene, microbial shifts</td>
<td>Individual susceptibility</td>
</tr>
<tr>
<td>Mucosal sensitivity</td>
<td>Mucosal atrophy, neuropathy, mucositis, hyposalivation, physical/spinal/chemical trauma</td>
<td>Mucosal infections, reactivation of herpes viruses</td>
</tr>
<tr>
<td>Taste reduction/taste change/halitosis</td>
<td>Radiation, chemotherapy receptor toxicity; neuropathy; tumor necrosis; oral hygiene; diet, emesis, reflux</td>
<td>Secondary infection (candida, periodontal disease, hyposalivation)</td>
</tr>
<tr>
<td>Fungal infection</td>
<td>Hyposalivation, tobacco use, prothrombin, antibiotics, steroids</td>
<td>Altered local and systemic immunity, myelosuppression, immunosuppression</td>
</tr>
<tr>
<td>Bacterial infection</td>
<td>Oral mucositis, ulceration, inflammation, infections, poor oral hygiene</td>
<td>Altered local and systemic immunity, myelosuppression, immunosuppression</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Oral mucositis, ulceration, inflammation</td>
<td>Altered nutrition, immunosuppression</td>
</tr>
<tr>
<td>Oral function, dysphagia, hyposalivation, dental status, necrosis</td>
<td>Oral function, dysphagia, hyposalivation, taste change, orofacial and mucosal pain, dental status, necrosis</td>
<td>Altered nutrition, immunosuppression, mucosal and salivary gland pathosis</td>
</tr>
<tr>
<td>Temporomandibular disorders</td>
<td>Mandibular discontinuity, tissue fibrosis, graft-versus-host disease</td>
<td>Myelosuppression, anemia, nutritional status, diabetes mellitus, tobacco use, immunosuppression</td>
</tr>
<tr>
<td>Compromised wound healing</td>
<td>Vascular supply, tissue cellularity, radiation therapy, chemotherapy</td>
<td>Salivary hypofunction, secondary infection</td>
</tr>
<tr>
<td>Soft-tissue necrosis, osteonecrosis</td>
<td>Radiation therapy, trauma, bisphosphonate drugs, possible antiangiogenic drugs, tobacco use, trauma</td>
<td>Diabetes, tobacco use, nutritional compromise; immunosuppression, mucosal and salivary gland pathosis</td>
</tr>
<tr>
<td>Soft-tissue necrosis, osteonecrosis</td>
<td>Radiation therapy, chemotherapy, regional cancereization, tobacco use, alcohol, viral agents (e.g., HPV, EBV)</td>
<td>Immunosuppression</td>
</tr>
<tr>
<td>Recurrent, secondary, or other cancers</td>
<td>Radiation therapy, chemotherapy, regional cancereization, tobacco use, alcohol, viral agents (e.g., HPV, EBV)</td>
<td>Immunosuppression</td>
</tr>
<tr>
<td>Compromised systemic health and nutritional compromise</td>
<td>Oral function, dysphagia, hyposalivation, taste change, orofacial and mucosal pain, dental status, necrosis</td>
<td>HIV, diabetes mellitus, tobacco use, immunosuppression, mucosal and salivary gland pathosis</td>
</tr>
<tr>
<td>Dental and skeletal growth and development (pediatric patients)</td>
<td>Radiation therapy, chemotherapy, direct tissue toxicity</td>
<td>Hormonal effects on growth and development, stage of dental and skeletal maturation at time of therapy</td>
</tr>
</tbody>
</table>

Haltis

Haltis in cancer patients can be caused by tissue necrosis, hyperalgesia, mouth breathing, poor oral hygiene, and infections, and can lead to dryness and loss of saliva. Oral conditions contribute to the development of oral infections, which can lead to oral and eye irritation, dry mouth, and 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 treatment, and in patients who received bisphosphonates for oncologic purposes and possibly antineoplastic medications. Bisphosphonates lead to bone resorption and bone exposure can be asymptomatic or may be symptomatic and, therefore, not recognized until progressive and symptomat-

Risks for osteonecrosis of the jaw include previous history of bisphosphonate therapy, radiation, and chemotherapy, and comorbid conditions such as diabetes, immunosuppressive therapy, and smoking. The risk of osteonecrosis is related to the amount of bone exposed during the radiation treatment and follow-up.

Survivors of transplant may be at risk for osteonecrosis and to posttransplant lympho-proliferative disorders, which present in the head and neck and in the oral cavity. This can be caused by congenital or acquired shifts in diet quality that may result in macro- and micro-nutrient deficiencies. All factors associated with oral function and all factors should be addressed in management.

Growth and Development in Children

Radiation therapy and high-dose chemo-
terapy can impact orofacial and dental development in children. Bone growth may be affected by high-dose irradiated tissues. Individuals in whom the hypo-
thalamus is affected may have delayed or altered sexual development. The possible effects on the denti-

Further research on the denti-

eral development of teeth and the occurrence of secondary malignancies.

complications affecting the oral cavity, and the impact on QoL and nutrition are important in evaluating the outcomes of patients following cancer treatment.

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References


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 1970 (Comprehensive Health Manpower Training Act) also helped to create a current 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

Dr. William John 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 1923.²

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 clinicians.³

Shortly after A Survey of Dental Education in the United States and Canada 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 biochemistry and nutrition of dental caries. Throughout his career, he looked at nutrition as the key element in survival, although his center of attention shifted in 1945 to dental nutrition. The relationship of nutrition, immunology, and stress on our indigenous microflora and 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.³

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 Detrick 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 Detrich, 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.⁴

The Birth of Modern-Day Microbiology and Immunology

Two of Dr. Rosebury’s students, Drs. Solomon 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 University in 1945 and later joined the faculty of the State University of New York–Buffalo, where he started and headed up the Department of Oral Biology and, soon after, became the first PhD program in that subject. Robert Genco was a member of his staff; Michael Levine, Frank Scan-napieco, Lawrence Tabak, Martin Taubman, and Thomas Van Dyke were among the future dental scientists trained in that department.⁵ Dr. Levine would inherit Dr. Ellison’s mantle and bring the laboratory of biochemical salivaary analysis into the post-genomic era. The possibility of success ful artificial saliva for xerostomia remained one of his driving forces.⁶

Dr. Macdonald returned to the University of Toronto to establish its first government-funded research lab, and shortly thereafter in 1956, he left Macdonald Building, which houses the Faculty of Dentistry. His article “Science Education: Backdrop for Discovery” was one of the underpinning ideas for his farreaching educational programs in higher education. He gave this as his presiden tial address at the International Association for Dental Research in 1968.⁷

He chose his staff wisely and selected a young graduate, Dr. Ronald Gibbs, from the University of Maryland, who held a PhD in anaerobic microbiology. He was also joined by a former student, Dr. Sigmund Socorsky, 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 Losche, 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.²

¹ 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.² 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.
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 and functional aspects of saliva. Dr. Leon Johnson drew on his knowledge of clinical dentistry to demonstrate the potential of saliva as a mirror of the body. He was one of the first researchers to recognize the significance of saliva in the context of systemic health and disease.

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 then could enter the university milieu and contribute to the growth of health sciences.

As a graduate of Tufts College Dental School, Dr. A. LeRoy Johnson learned early that a dental degree with 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 had recently been 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 at both the University of Michigan and the University of Pennsylvania, Dr. Johnson settled into private orthodontic practice in New York City.

Dr. Pearson suggested that Dr. Johnson try his ideas on the deans of a few medical schools to see if there were no dental schools. Dr. A. LeRoy Johnson

In a more recent study, "Salivary Diagnostics," Dr. Wong elaborates on the potential of saliva as a mirror of the body. 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. 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 then could enter the university milieu and contribute to the growth of health sciences.

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The institute he founded was able to explore the biological and clinical aspects of oral health. Dr. Joseph Lowy, a graduate of Simon Fraser University and UBC Dental School, has become a leader in this subfield. Much of the progress is due to funding from the NIH under the directorship of Dr. Lawrence Tabak. 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 then could enter the university milieu and contribute to the growth of health sciences.

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Dr. Volker's work was completed under the guidance of 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 waive budgetary problems 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. Both he and Dr. Volker became distinguished professors with endowed chairs in dental medicine and had buildings named in their honor at UAB.

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The future will demand scientific innovation and the technologies that it spawns. Dentistry will need a well-trained undergraduate student body, as well as inter-disciplinary scientists, including qualified clinicians. Expanded-duty personnel and automation of processes will continue to bring preventive therapies and curative procedures to our nation's population. Dental education will once again become the preserve of all things related to the second decade of this new century.

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2. Millstein CB. Dr. William J. Gier's contribution to dental research, education, and journalism. JADA. 2002;133(3):144.
19. 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.
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24. Developing Future Dental School Leaders
26. She held an MD degree from Yale, and the second, Dr. Reider Sognnaes, had earned his PhD at Rochester.
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Mapping Dental Establishments in Massachusetts Just Before the Recession

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STEVEN P. PERLMAN, DDS, MS, D, DHL (Hon)
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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 in the population and limited proportional increase in the numbers of dentists and dental establishment ratios. This resulted in small changes in population-to-establishment 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). Between 2000 and 2006—the last year for which this datum is available—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 In 2000, the dentist-to-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.3,4 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 (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.5

Changing Number of Establishments

Between 2000 and 2007, there was an increase of 70 dental establishments (from 2,188 to 2,358). 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.)

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 employees.6,7 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).

In Massachusetts counties, the proportion of dental facilities with fewer than five employees ranged from 26 percent in Hampden 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 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). 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). 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.

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

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.)

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 1. Massachusetts Dental Establishments by County: 2000, 2007

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<tbody>
<tr>
<td>Total State</td>
<td>2,946</td>
<td>3,118</td>
<td>172</td>
</tr>
<tr>
<td>Barnstable</td>
<td>124</td>
<td>127</td>
<td>3</td>
</tr>
<tr>
<td>Berkshire</td>
<td>71</td>
<td>66</td>
<td>-5</td>
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<tr>
<td>Bristol</td>
<td>184</td>
<td>200</td>
<td>16</td>
</tr>
<tr>
<td>Dukes</td>
<td>8</td>
<td>8</td>
<td>-</td>
</tr>
<tr>
<td>Essex</td>
<td>353</td>
<td>394</td>
<td>31</td>
</tr>
<tr>
<td>Franklin</td>
<td>23</td>
<td>24</td>
<td>1</td>
</tr>
<tr>
<td>Hampden</td>
<td>181</td>
<td>179</td>
<td>-2</td>
</tr>
<tr>
<td>Hampshire</td>
<td>49</td>
<td>54</td>
<td>5</td>
</tr>
<tr>
<td>Middlesex</td>
<td>79</td>
<td>83</td>
<td>14</td>
</tr>
<tr>
<td>Nantucket</td>
<td>6</td>
<td>5</td>
<td>-1</td>
</tr>
<tr>
<td>Norfolk</td>
<td>417</td>
<td>432</td>
<td>15</td>
</tr>
<tr>
<td>Plymouth</td>
<td>208</td>
<td>208</td>
<td>0</td>
</tr>
<tr>
<td>Suffolk</td>
<td>242</td>
<td>261</td>
<td>19</td>
</tr>
<tr>
<td>Worcester</td>
<td>301</td>
<td>311</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 2. Massachusetts Population per Dental Establishment by County: 2000, 2007

<table>
<thead>
<tr>
<th>County</th>
<th>2000</th>
<th>2007</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total State</td>
<td>2,159</td>
<td>2,074</td>
</tr>
<tr>
<td>Barnstable</td>
<td>1,800</td>
<td>1,747</td>
</tr>
<tr>
<td>Berkshire</td>
<td>1,888</td>
<td>1,970</td>
</tr>
<tr>
<td>Bristol</td>
<td>2,973</td>
<td>2,726</td>
</tr>
<tr>
<td>Dukes</td>
<td>1,884</td>
<td>1,921</td>
</tr>
<tr>
<td>Essex</td>
<td>2,094</td>
<td>1,907</td>
</tr>
<tr>
<td>Franklin</td>
<td>3,108</td>
<td>2,985</td>
</tr>
<tr>
<td>Hampden</td>
<td>2,552</td>
<td>2,533</td>
</tr>
<tr>
<td>Hampshire</td>
<td>3,109</td>
<td>2,864</td>
</tr>
<tr>
<td>Middlesex</td>
<td>1,885</td>
<td>1,723</td>
</tr>
<tr>
<td>Nantucket</td>
<td>1,595</td>
<td>2,212</td>
</tr>
<tr>
<td>Norfolk</td>
<td>1,561</td>
<td>1,517</td>
</tr>
<tr>
<td>Plymouth</td>
<td>2,280</td>
<td>2,288</td>
</tr>
<tr>
<td>Suffolk</td>
<td>2,854</td>
<td>2,787</td>
</tr>
<tr>
<td>Worcester</td>
<td>2,500</td>
<td>2,514</td>
</tr>
</tbody>
</table>

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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.

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

<table>
<thead>
<tr>
<th></th>
<th>Massachusetts</th>
<th>United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–4</td>
<td>1,508</td>
<td>1,285</td>
</tr>
<tr>
<td>5–9</td>
<td>973</td>
<td>1,130</td>
</tr>
<tr>
<td>10–19</td>
<td>318</td>
<td>404</td>
</tr>
<tr>
<td>20–49</td>
<td>50</td>
<td>73</td>
</tr>
<tr>
<td>50+</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>2,850</td>
<td>2,946</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Number of Establishments</th>
<th>Massachusetts</th>
<th>United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–4</td>
<td>124</td>
<td>127</td>
</tr>
<tr>
<td>5–9</td>
<td>184</td>
<td>200</td>
</tr>
<tr>
<td>10–19</td>
<td>68</td>
<td>8</td>
</tr>
<tr>
<td>20–49</td>
<td>83</td>
<td>144</td>
</tr>
<tr>
<td>50+</td>
<td>23</td>
<td>24</td>
</tr>
<tr>
<td>Total Mass.</td>
<td>1,188</td>
<td>1,285</td>
</tr>
</tbody>
</table>

For the future of the profession. In addition, the American Dental Association commented in past economic reverses that “because patient loads will win the long run, an economic recession should prove to be a minor interruption in improving practice conditions.” 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.

References

Table 5. Dental Establishments by Number of Employees in Massachusetts and the United States: 2000, 2007

<table>
<thead>
<tr>
<th>Number of Establishments</th>
<th>Massachusetts</th>
<th>United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,188</td>
<td>1,285</td>
<td>1,202</td>
</tr>
</tbody>
</table>

Flexibility.

- Temporary and permanent staffing exclusively for the dental industry.
- Dentists, Dental Hygienists, Dental Assistants and Administrative Staff.
- Centrally owned and operated since 1979 with local specialists.
- Flexible temporary rates; proportional rebates should a permanent placement fail to last.
- All dental auxiliary and administrative temps are our employees.
- Personnel are interviewed in person and are pre-screened.
- Extensive live phone coverage by a large team of staffing experts.
- RDH Temps carries professional liability (malpractice) insurance on all Dental Hygienists & Assistants.
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 anything below 7 is acidic. 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.

Conclusion

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

References


Table 1. Acid Levels in Sugarless Candy and Beverages.

<table>
<thead>
<tr>
<th>Candy/Beverage</th>
<th>pH</th>
<th>Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generic Walgreens Cough Drop</td>
<td>5.0</td>
<td>p</td>
</tr>
<tr>
<td>Sugar Free PopSodas</td>
<td>4.5</td>
<td>p</td>
</tr>
<tr>
<td>Breathavers 3 Hour Mint, sugar free</td>
<td>5.0</td>
<td>p</td>
</tr>
<tr>
<td>Pinky Peachment Sugarless Tablets</td>
<td>4.5</td>
<td>p</td>
</tr>
<tr>
<td>Sugar Free Altoids, &quot;Simply Mint&quot;</td>
<td>6.5</td>
<td>p</td>
</tr>
<tr>
<td>Sugar Free Eclipse Gum, &quot;Winterrind&quot;</td>
<td>6.0</td>
<td>p</td>
</tr>
<tr>
<td>Orbit Sugar Free Gum, &quot;Pine Colada&quot;</td>
<td>6.0</td>
<td>p</td>
</tr>
<tr>
<td>Lifesavers Sugar Free: Pineapple</td>
<td>5.0</td>
<td>p</td>
</tr>
<tr>
<td>Cherry, Watermelon, Raspberry, and Orange</td>
<td>4.5</td>
<td>p</td>
</tr>
<tr>
<td>Mentos Gum, &quot;Tropical&quot;</td>
<td>4.5</td>
<td>p</td>
</tr>
<tr>
<td>Coastal Bay Sugar Free Fruit Hard Candy</td>
<td>4.5</td>
<td>p</td>
</tr>
<tr>
<td>Reast (15 Sugarless Gum)</td>
<td>6.0</td>
<td>p</td>
</tr>
<tr>
<td>Jewel Decaf Classic Roast Instant Coffee</td>
<td>5.0</td>
<td>m</td>
</tr>
<tr>
<td>Marselli House Instant Coffee</td>
<td>6.1</td>
<td>m</td>
</tr>
<tr>
<td>Chute &amp; Samborn Special Roast Coffee</td>
<td>6.2</td>
<td>m</td>
</tr>
<tr>
<td>Nestea Unsweetened Iced Tea</td>
<td>6.3</td>
<td>m</td>
</tr>
<tr>
<td>Sugar Free JeI-O, &quot;Royal Lime&quot;</td>
<td>5.0</td>
<td>p</td>
</tr>
<tr>
<td>Luigis Real Italian Ice—no sugar added</td>
<td>4.1</td>
<td>m</td>
</tr>
<tr>
<td>Diet Mountain Dew</td>
<td>3.4</td>
<td>m</td>
</tr>
<tr>
<td>Diet Coke</td>
<td>3.3</td>
<td>m</td>
</tr>
<tr>
<td>Diet Pepsi</td>
<td>3.0</td>
<td>m</td>
</tr>
<tr>
<td>Diet Dr. Pepper</td>
<td>3.2</td>
<td>m</td>
</tr>
<tr>
<td>Diet 7Up</td>
<td>3.7</td>
<td>m</td>
</tr>
</tbody>
</table>

Table 2. Tooth enamel dissolves at a pH level of 5.3.

It was more accurate than the pH paper, which measured only to the nearest pH 0.1 with a range of pH 4.5–7.3. 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 and 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. The results of this study were used to roughly measure acidity and identify a previously unrecognized problem, so both systems were applicable to this project.

Conclusion

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

References


Figure 1. Sugarless candies to be tested.

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

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

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

Journal of the Massachusetts Dental Society
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.

The Whiner and Complainer

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 lies. Always have a third party present to document the conversation. Do your homework and clarify everything.

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 services and fees. Many dental practices have dictionary-like terms that can be confusing to a patient. 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 angry 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 always find them 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.

A front office 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 complain 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 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 accept responsibility 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 anger—whether the patient is “angry at the system” or “angry at you”—and understand it. 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.

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 system, it is the responsibility of the practice to address 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 to at least six months before you change the relationship with their plan. Clearly state the reasons for the change, and keep in mind that patients will want to know what these 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 options in the practice and inform before you perform. You should discuss the cost with the patient and finalize financial arrangements before starting treatment. Some situations require a mediation of sorts. Make sure your patients feel heard.

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 allow the patient access to their medical records. Check with the Board of Registration in Dentistry (BORD) for Massachusetts regulations regarding dismissing a patient.
A Clinico-Pathologic Correlation

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 4.1–10.9x10³/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
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 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.
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.

References
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

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.

References
Dense bone islands are synonymous with entostosis 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
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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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 uncontrollable 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.

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

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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 acknowledged 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

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.

**RICHARD L. WYNN**
**TIMOTHY F. MEILLER**
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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.


**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 AIDS, 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 periodontium, 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.

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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 (C₆H₁₂O₆), 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. ■