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Pay inequality is more than trending. Famous actors like Jennifer Lawrence, tennis stars like Serena Williams and executives like Sheryl Sandberg raise our awareness every day. It’s an issue in this year’s presidential election. We cheered when Patricia Arquette challenged “… every woman who gave birth …” to fight for wage equality. Newsweek reports that a female professional soccer player is paid 38 percent of her male counterpart. And, although Serena Williams is acknowledged as the highest paid female athlete in the world, she still earns 15 percent less, per victory, than her male counterpart, Novak Djokovic.

The Institute for Women’s Policy Research states that women earn 79 percent of the income of men. In dentistry, a CBS MoneyWatch article states that female dentists earn only 72 percent of what male dentists earn — compare that to female physicians who earn 82 percent of their male counterparts.

This is all just so hard to take. Women are almost half of the new dental workforce, and 40 percent of all women are the breadwinners in their families. A study out of the National Bureau of Economic Research tried to make sense of this disparity in wages, reviewing data over the last 30 years. It found that the time women take off, whether it is a long leave or a shortened daily schedule, in order to care for their families, is a large contributing factor. Other studies have suggested that pay may be affected because females may not be the best negotiators, or in health care professions, they may choose lower paying specialties.

It’s true. At first, my beautiful baby resulted in decreased office hours. However, it also brought a new appreciation that I had chosen a great profession. We can set our own hours and still ensure that we see patients and maintain production. My mother, who is also a dentist, set her hours around our schedules. She drove us to school every morning. On the way, we ran through our multiplication tables, recited our memorized prayers and practiced our vocabulary words. She came to our soccer games, tennis tournaments and piano recitals. She also worked hard, spending many hours in the office. But as the boss, she could also bring her kids into the office after school. She was a rock star at both roles — dentist and mother. She was able to provide a beautiful life for us, even as a single parent. She has always credited her choice of profession as the reason for being able to balance it all.

Like my mother, most of my female colleagues have a strong desire to succeed, are financially astute and do well. So where does this pay gap come from? Are female dentists less aggressive at presenting treatment plans? Do we make less per hour because we spend more time with more patients? Do fewer female dentists own their practices or are they employee dentists who possibly earn less? Are male dentists better at negotiating contracts with dental plans? We must find the answers to these questions.

The U.S. Equal Employment Opportunity Commission has a proposal to collect data about employees’ pay beginning in 2017 for employers with more than 100 employees. Adding pay data to existing employer reports that report race, gender and ethnicity, they say, will provide a tool to identify discriminatory pay practices.

Last year, Fortune reported, in an interview with Salesforce CEO Marc Benioff, that he addressed this issue by spending about $3 million to bring the salaries of female employees up to the level of their male counterparts.

If the economists are right that in the future, most dentists will transition out of private practice and into being employees, such a proposal may benefit us as well.

Venus Williams shed an introspective light on gender wage disparity, “Imagine you’re a little girl. You’re growing up. You practice as hard as you can, with girls, with boys. You have a dream. You fight, you work, you sacrifice to get to this stage. You work as hard as anyone you know. And then you get to this stage, and you’re told you’re not the same as a boy. Almost as good, but not quite the same. Think how devastating and demoralizing that could be.”

Our dental school classes are comprised of more and more women.

Dentistry’s Pay Gap
Ruchi K. Sahota, DDS, CDE

In dentistry, a CBS MoneyWatch article states that female dentists earn only 72 percent of what male dentists earn.
Eventually, the gender gap within our profession will close. We will rise to more leadership positions. We will lean in. We will band together. And, we will soon arrive at a day where the pay gap narrows enough to just disappear. Sure, we cannot have it all, but the flexibility of our profession allows us to have a lot. And for that, I cannot be more grateful.

Ruchi K. Sahota, DDS, CDE, practices family dentistry in Fremont, Calif., and serves as faculty at the University of the Pacific, Arthur A. Dugoni School of Dentistry. She is also a certified dental editor, a consumer advisor for the American Dental Association, past president of the Southern Alameda County Dental Society and a fellow of the American College of Dentists, International College of Dentists and the Pierre Fauchard Academy.

The Journal welcomes letters

We reserve the right to edit all communications. Letters should discuss an item published in the Journal within the last two months or matters of general interest to our readership. Letters must be no more than 500 words and cite no more than five references. No illustrations will be accepted. Letters should be submitted at editorialmanager.com/jcaldentassoc. By sending the letter, the author certifies that neither the letter nor one with substantially similar content under the writer’s authorship has been published or is being considered for publication elsewhere, and the author acknowledges and agrees that the letter and all rights with regard to the letter become the property of CDA.
Impressions

The nub:
1. The small stuff matters the most in dental ethics.
2. It is easier to bend a green twig than move a mighty oak.
3. There is something everyone can do this afternoon to improve the ethical tone of dentistry.

Small Stuff

David W. Chambers, EdM, MBA, PhD

Before there was Watson that blitzed the experts on “Jeopardy,” and even before computers regularly humbled chess masters, there was Arthur Samuel and his checker-playing computer. That was 1949, and Samuel probably had to hand-crank his machine.

Samuel started by giving his gadget some great tactics, such as jumping an opponent or getting to the king row. He had the computer assign high point values to the big payoff moves. At each move, the computer searched a modest range of available options and picked the one with the most points. The machine was allowed to adjust the point distribution. Following each move and the opponent’s response, the computer would re-evaluate the approach and adjust point values. That was the learning part.

At first, Samuel pretty regularly beat the computer. But it got better and eventually Samuel had to give up the contest as being hopeless.

What did the computer learn that made it so effective? Mostly it was the small stuff. Open a space toward the middle rather than the edge of the board, reduce the opponent’s possible moves, that sort of thing. Most checkers games are won or lost in the scrum at the beginning, based on slight adjustment in balance. After establishing control of the territory with multiple very small wins, even a 6-year-old child can run the board with kings and triple jumps.

And so it is with dental ethics. Disciplined licenses, lawsuits against unfair practices, Medicaid fraud and the like may get the headlines. But it is the daily, nuanced decisions — winking at a colleague’s questionable dealings or causal depreciation of the aspirations of auxiliaries — that establish the tone of the profession.

A few years ago, I tested this idea, and the results have been published. I looked to see what effect we could get by having enforcers, such as state boards, clamp down on devious practitioners. I also looked at the relationship between dentists who are ethically upright and unafraid to point out the behavior of the few bad apples and the dentists who are ethically upright but decline to get involved.

Like Samuel’s checker-playing computer, the real action depended on the small stuff. The relationships among good practitioners and how willing they are to put the details of good practice on the table were more important than what was done to the devious few. The effect is many orders of magnitude in favor of daily details.

The next time someone tells you not to sweat the small stuff in dental ethics and wait until something really big and important comes along, you can be pretty sure the person you are talking with has been waiting a lifetime for the right opportunity to do something ethical.

David W. Chambers, EdM, MBA, PhD, is professor of dental education at the University of the Pacific, Arthur A. Dugoni School of Dentistry, San Francisco, and editor of the Journal of the American College of Dentists.
Tobacco Smoke Houses
Pathogens in the Mouth

Tobacco smoke is a proven environmental stressor by allowing bacteria colonization and “immune invasion” in the mouth, according to a new study from the University of Louisville School of Dentistry.

Specifically, smoke, which is comprised of thousands of chemical components, acts as an environmental stressor to which oral bacteria respond by altering the expression of multiple genes and proteins, including virulence factors that promote colonization and immune evasion. Recent evidence has demonstrated that tobacco smoke and components alter the bacterial surface and promote biofilm formation in several important human pathogens, including Staphylococcus aureus, Streptococcus mutans, Klebsiella pneumonia, Porphyromonas gingivalis and Pseudomonas aeruginosa.

University of Louisville School of Dentistry researcher David A. Scott, Ph.D, explained his findings in a press release.

“One pathogen establishes itself within a biofilm, it can be difficult to eradicate as biofilms provide a physical barrier against the host immune response, can be impermeable to antibiotics and act as a reservoir for persistent infection,” Scott said.

“Furthermore, biofilms allow for the transfer of genetic material among the bacterial community and this can lead to antibiotic resistance and the propagation of other virulence factors that promote infection.”

Of course, dental plaque is a biofilm and it can lead to gingivitis and chronic periodontitis.

Kentucky ranks No. 2 for cigarette use among adults, according to the Centers for Disease Control and Prevention. Studies from the National Survey on Drug Use and Health show that 90 percent of smokers start as teens. In California, 21,000 kids get hooked on smoking every year, and half of them will die from tobacco-related illnesses. California has approximately 3.4 million adult smokers and 200,000 youth smokers.

Dental patients can be reminded that smokeless tobacco use puts them at risk not only for oral cancer, but also cancer of the pharynx, larynx and esophagus, as well as may lead to tooth abrasion, increased tooth decay, gum recession, nicotine dependence, tooth discoloration and bad breath. Danger signs of oral cancer include a sore that does not heal; a lump or white patch; a prolonged sore throat; difficulty in chewing; restricted movement of the tongue or jaws; and a feeling of something in the throat. According to the National Institute of Dental and Craniofacial Research (NIH), the five-year survival rate for those with localized disease at diagnosis is 83 percent compared with only 36 percent for those whose cancer has metastasized.

Photos on Cigarette Packages May Be Strong Smoking Deterrent

Photos related to the dangers of smoking on cigarette packages are a strong deterrent and provoke more users to quit, according to researchers at the University of North Carolina at Chapel Hill. A clinical trial, which was recently published by JAMA Internal Medicine, was conducted over four weeks and included 2,149 smokers (1,901 completed the study). Those enrolled received warnings either through text or photos. According to the results, 40 percent of the participants who were exposed to pictures attempted to quit. Among the smokers who received only a text warning, 34 percent attempted to quit. Further, 5.7 percent of the photo group quit for at least a week.

“Implementation of pictorial cigarette pack warnings in the United States is on hiatus. Our trial findings provide timely and important information as the United States and other countries consider requiring pictorial cigarette pack warnings. The World Health Organization Framework Convention on Tobacco Control now recommends pictorial warnings but stops short of requiring them. Our trial findings support strengthening the treaty to require pictorial warnings on cigarette packs,” the study concludes.

The limitations of the study include not having an understanding of what long-term exposure to photos may have and whether the participants coming into the study may have been more inclined to quitting than the general population.

Stock image of a cigarette pack from Brazil, where images on cigarette packs are required.
Study Describes Damage Caused to Mouth From Exploding E-Cigarette

A report has been published in scientific literature that describes the extent of damage an exploding e-cigarette can cause to the mouth. The University of Cincinnati studied the mouth of an 18-year-old who had an e-cigarette explode while using it. The report, published in the *Journal of Oral and Maxillofacial Surgery*, describes the burns, lacerations and lost and fractured teeth that resulted from the explosion.

E-cigarettes work by heating up liquid nicotine, the neurotoxin derived from tobacco that is as addictive as cocaine, producing a vapor that users inhale. The U.S. Fire Administration has been evaluating the safety of e-cigarettes after reports of explosions and fires have increased.

The authors of the study state that multiple procedures will need to take place to “reconstruct lost tissue, and to re-establish functional and cosmetically acceptable results.”

In addition to fire risk, e-cigarettes carry a variety of health risks for users and those around them. E-cigarette aerosol contains formaldehyde and lead, as well as at least 10 toxic chemicals on California’s Proposition 65 list of chemicals known to cause cancer and birth defects. E-cigarette use among middle and high school students tripled from 2013 to 2014, according to the Centers for Disease Control and Prevention and more than a quarter of a million youth who had never smoked a cigarette used e-cigarettes in 2013. Ninth graders who use e-cigarettes are eight times more likely to later smoke traditional cigarettes than their peers who have never tried e-cigarettes. E-cigarettes are widespread with 450 brands currently available and some 7,600 flavors. E-cigarettes are sometimes touted as the healthy alternative to traditional cigarettes, but some reports have cited negative effects of the vapors.

The National Institute of Dental and Craniofacial Research has awarded more than $2 million in first-year funding to seven research grants centered on studying the effects of e-cigarettes on oral and craniofacial tissues. The timing of the awards is critical as research shows an increasing number of high school students, approximately 13.4 percent, are now using e-cigarettes. Additionally, evaluating the risks of e-cigarettes has been challenging due to the lack of research regarding their harmful effects.

Facial Blueprint Studied Through Stem Cells

A new study has identified the role of molecular signals in the development of facial structures. Specifically, USC stem cell researchers looked at two types of molecular signals – Jagged-Notch and Endothelin1 (Edn1). These two signals are critical when the face is shaped. The researchers discovered that the Jagged-Notch and Edn1 “work in tandem to control where and when stem cells turn into facial cartilage.”

Edn1 signals speed up the formation of cartilage in the early stages of development in the lower face and Jagged-Notch signals stop the development of cartilage until later in the upper face. This timing is critical for the development of the regions of the face, according to the authors.

In a press release, USC Stem Cell researcher and lead author Lindsey Barske said, “We’ve shown that the earliest blueprint of the facial skeleton is set up by spatially intersecting signals that control when stem cells turn into cartilage or bone. Logically, therefore, small shifts in the levels of these signals throughout evolution could account for much of the diversity of shapes we see within the skulls of different animals, as well as the wonderful array of facial shapes seen in humans.”

Interestingly, many of the genes required to shape the facial skeleton in fish and man are the same. The researchers used the zebrafish system to understand the skeletal components of the face.

A three-day-old zebrafish head skeleton with newly differentiated cartilage cells (magenta) emerging from a pool of skeletal progenitor cells (green). (Image by Lindsey Barske)
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Strength of Dentin Tested

Dentin is a strong material. The reason as to why teeth in humans can come into contact so often (some 5,000 per times day) and rarely break is largely considered to be because of dentin. Researchers at Charité — Universitätsmedizin Berlin have found that “it is the mechanical coupling between the collagen protein fibers and mineral nanoparticles that render dentin capable of withstanding extreme forces.” To test this, the researchers enhanced compressive stress on the inside of dentin samples and put it under heat as high as 125 degrees Celsius. The result was the discovery that dentin has the same yield strength of construction-grade steel. A gradual reduction in the size of the CHAP crystal lattices toward the inner part of the tooth was present.

Paul Zaslansky is from the Charité’s Julius Wolff Institute.

“Tissue found near the dental pulp, which is formed during the later stages of tooth development, contains mineral particles that are made up of smaller cell units,” Zaslansky said in a press release.

Bacteria related to dental decay can soften the mineral, destroy collagen fibers and cause breakdowns in teeth. The researchers said that dentists should make sure to keep teeth moist when performing dental procedures.

“Avoiding dehydration may very well prevent build-up of internal stresses, the long-term effects of which remain to be studied,” Zaslansky said.

Chemicals in Plastics and Fungicides Could Harm Children’s Teeth

Chemicals commonly found in plastics and fungicides could be harming the teeth of children. This happens when the chemicals disrupt hormones that promote growth of dental enamel, according to a new study by the French National Institute of Health and Medical Research. According to a press release, “Endocrine disruptors are chemicals that interfere with mammalian hormones. Bisphenol A (BPA) is one of the most prevalent, found in everyday items, including refillable drink bottles and food storage containers. Vinclozolin is another endocrine disruptor that was commonly used as a fungicide in vineyards, golf courses and orchards.” The study analyzed how permanent first molars and incisors in children between the ages of 6 and 9 have sensitive areas that can be prone to cavities. This is referred to as molar incisor hypermineralization (MIH).

Researchers “cultured and studied rat ameloblast cells, which deposit enamel during the development of teeth. They found that the presence of sex hormones like estrogen and testosterone boosted the expression of genes making tooth enamel, especially male sex hormones. As BPA and vinclozolin are known to block the effect of male sex hormones, the findings reveal a potential mechanism by which endocrine disruptors are weakening teeth.”

Rats were used in the study and received a daily dose of BPA either by itself or with vinclozolin. The dosage was the same as an average dose a human would have every day from the time they are born to when they are 30 days old. Cells from the teeth of the rats were collected. The doses had changed the “expression of two genes controlling the mineralization of tooth enamel.” Another part of the experiment led to the discovery that a mechanism by which endocrine disruptors are weakening teeth.

Katia Jedeon, lead author of the study warned pregnant women to avoid endocrine disruptors.

“ Tooth enamel starts at the third trimester of pregnancy and ends at the age of 5, so minimizing exposure to endocrine disruptors at this stage in life as a precautionary measure would be one way of reducing the risk of enamel weakening.”
Marijuana use can have a long-term affect on oral health, according to a new study by Arizona State University. The study, which collected data from 1,037 individuals who were born in New Zealand in 1972 and 1973 and followed until they were 38 years old, found that “Cannabis was associated with poorer periodontal health at age 38 but was not associated with the other physical health problems.” Among the participants, 484 had ever used tobacco daily and 675 had ever used cannabis.

The researchers labeled the topic important because after major policy changes in the U.S., policymakers, health care professionals and the general public are looking for information on if cannabis use is related to physical health problems later in life. The specific goal of the study, which was discussed in an article published by JAMA Psychiatry, was to determine if marijuana use from the ages of 18 to 38 had a correlation with physical health problems at 38 years old. The researchers obtained laboratory measures of physical health (periodontal health, lung function, systemic inflammation and metabolic health), and self-reported physical health.

A press release on the topic stated, “This study has a number of implications. First, cannabis use for up to 20 years is not associated with a specific set of physical health problems in early midlife. The sole exception is that cannabis use is associated with periodontal disease. Second, cannabis use for up to 20 years is not associated with net metabolic benefits (i.e., lower rates of metabolic syndrome). Third, our results should be interpreted in the context of prior research showing that cannabis use is associated with accidents and injuries, bronchitis, acute cardiovascular events, and, possibly, infectious diseases and cancer, as well as poor psychosocial and mental health outcomes.”

According to the study, “Unlike cannabis use, tobacco use was associated with worse lung function, systemic inflammation and metabolic health at age 38 years, as well as within individual decline in health from ages 26 to 38 years.”

A little more than half of the participants were male. Some limitations of the study were self-reported marijuana use and the fact that physical health in all of the candidates was assessed in early midlife.

Cells Around Vital Organs Speed Tissue Repair

Cells that live in the cavities surrounding various organs (such as the heart, lung and liver) play a significant role in rapid tissue repair, according to a new study from the Cumming School of Medicine. The study, published in the journal Cell, specifically looked at macrophages. Macrophages “patrol within the cavity, and upon organ damage, adhere themselves to the damaged area for quick repair,” according to a press release.

Jing Wang, PhD, is the lead author of the study and member of the Snyder Institute for Chronic Diseases.

“The traditional thought of how organs are repaired after injury is that monocytes (a type of immune cell found in the blood) are recruited to the site of injury, move out of blood vessels and mature into macrophages in two to three days,” Wang said. “In our study, the mature macrophages are already in the cavity and can infiltrate the injury site in visceral organs directly, thereby initiating immediate and rapid repair.”

The cells were viewed in real time after experiencing thermal and toxin-induced injury. “The cells behaved in the same way for both types of injury. Further to the observation, when the macrophage supply was depleted in the abdominal cavity, tissue repair did not take place as quickly. When the cells were reinfused back into the animal models, they resumed their role,” the press release stated.

Wang believes the findings may change how clinical procedures and surgeries are performed. “Washing out” a cavity to clear out foreign pathogens could be “hindering the healing process.”

Electron microscope image of a macrophage in the alveolus, showing the nucleus and cytoplasmic organelles, such as golgi and mitochondria.
Dental Student Research

Parish P. Sedghizadeh, DDS, MS

Dental research not only advances the field of dentistry and oral health care, it directly impacts patient management and informs medicolegal standards of care. Our understanding of the pathogenesis of oral diseases, the risk factors for disease in our patients and therapeutic guidelines all come from peer-reviewed research findings. Whether basic science or clinical research, both are often required for ultimate translation of research findings to patient care. Therefore, dental research in its entirety is necessary to safeguard the well-being of our patients and to ensure the growth of our profession in a rapidly evolving health care field.

Naturally, the future of dental research rests in the hands and minds of young and new investigators. They must carry the torch to assure a bright future for dentistry and dental research. Therefore, in this issue of the Journal we celebrate and recognize the work of young dental researchers and their mentors. We pay homage to the fact that student investigators often go above and beyond what is required of them in their training or curriculum to make dedicated time for conducting research. We also recognize their mentors who foster student inquiry and help maintain a pipeline of dental investigators. As an educator and mentor, I know that student research is challenging and involves higher-order learning in the cognitive domain for these students; they are challenged to generate data, synthesize information or apply analytical and problem-solving skills. What these student researchers and their mentors do is by no means easy and involves commitment and sacrifice. Despite the challenges, the research efforts of our young investigators can also be rewarding as evidenced by the peer-reviewed publication of their work, which is highlighted in this issue.

I would like to thank our student researchers and their mentors for their hard work and diligence in writing these articles. I am thankful for the opportunity to be the guest editor for this issue, and I hope you will enjoy (as I did) seeing the scholarship and research efforts of our young dental investigators.
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Antifungal Activity of Alkaloids Against Candida albicans

Juliana Noguti, DDS, MS, PhD; Mersedeh Rajinia; Bruna Raquel Zancope, DDS, MS; Maria Carolina Salome Marquezin, DDS, MS; Dalia Seleem, DDS; Vanessa Pardi, DDS, MS, PhD; and Ramiro M. Murata, DDS, MS, PhD

ABSTRACT Candida albicans is a pathogen in the mouth responsible for opportunistic infections that are usually harmless. Natural products have been used to develop several drugs, mostly anticancer and anti-infective agents. Among these, alkaloids have been studied for their medicinal properties. In this study, we examined their antifungal activity against C. albicans in vitro. Among the alkaloids studied in this work, berberine hydrochloride showed the best activity against C. albicans.

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Among infections observed in patients, especially when there is an imbalance in the normal flora,1,2 Candida albicans is the most prevalent human fungal pathogen.3 Its ability to form single biofilms increases exponentially the virulence4 as well as the resistance to antimicrobial drugs.5 Oral candidiasis vary from the large white plaques of pseudomembranous layers on the tongue and buccal mucosa to the palatal erythematous lesions of chronic atrophic candidiasis and moreover, to angular cheilitis on the labial commissures.6 Generally, C. albicans biofilm can be present in those who have dentures and implants.7 When systemic diseases or immune deficiencies affect the individual, this opportunistic fungus promotes very uncomfortable infections,8 thereby decreasing the patient’s quality of life.9 In light of these pathologies from C. albicans,
there is a growing need to research and develop new approaches that might be used against this pathogen. In addition, limitations of the currently available antifungal agents present a challenge in the treatment of oral candidiasis.

Natural products are still major sources of innovative therapeutic agents for infectious diseases. Alkaloids are secondary metabolites found in many plants and microorganisms that have shown several pharmacological activities, including antimicrobial activity.

The aim of this study was to evaluate the antifungal activity of several alkaloids against Candida albicans in vitro and to determine the cytotoxicity on oral fibroblast cells.

Material and Methods

Antimicrobial Compounds

In this in vitro study, we performed a screening using several natural alkaloids. A library of 131 FDA-approved drugs was purchased (Selleck Chemicals, Houston). Compounds were stored as 10 μM stock solutions in dimethyl sulfoxide (DMSO) at 4 degrees Celsius until use. Berberine hydrochloride, cinchonidine, cytisine, gramine, lappaconite hydrobromide, matrine, oxymatrine, piperine, pinosylvin, sophocarpine, and synephrine were selected for further analysis based on their antimicrobial activity as determined by minimum inhibitory concentrations (MIC) using the microdilution broth method (Clinical and Laboratories Standards Institute (CLSI), 2002). The concentrations tested for all compounds used were in the range 0.01-100 μM in 1% DMSO. All in vitro tests included the following tested groups: 1-negative control, 2-vehicle (1% DMSO v/v) and 3-positive control (fluconazole 32.65 μM).

Microbial Growth Conditions

C. albicans MYA-2876 (ATCC-American Type Culture Collection, Manassas, Va.) was used in this study. It was the selected strain based on its proven virulence and genomic sequencing. C. albicans strains were subcultured on BBL Sabouraud’s dextrose agar (BD, Sparks, Md.) and incubated at 37 degrees Celsius with 5% CO₂ for 36 hours. The culture medium of C. albicans was replaced once daily during the 36-hour incubation period. The inoculum strain concentration was standardized according to the microdilution broth method.

The initial stock suspension was obtained by suspending five colonies of C. albicans from 24-hours-old cultures in saline. The cell density of the initial stock suspension was in the range of 1 x 10⁶ - 5 x 10⁶ cells per ml, which was determined using a spectrophotometer (DU-650, Beckman Coulter, Fullerton, Calif.), by measuring the absorbance of 0.08-0.1 at 625 nm wavelength. The working suspension of the inoculum used in this experiment was standardized to 5.0 x 10⁷ - 2.5 x 10⁸ colony forming units (CFU)/ml by diluting the initial stock solution in medium 1:2,000. The 36-hour biofilm was established based on our preliminary data (not published), illustrating the peak mature phase of C. albicans biofilms. Our preliminary results on such findings were obtained by comparing biofilm density over a five-day period. Analyses were both qualitative, done by examining biofilm morphology microscopically, as well as quantitative, performed by comparing the biomass or total dry weight of the biofilms collected over the five-day period.

Minimum Inhibitory Concentration and Minimum Fungicidal Concentration

The minimum inhibitory concentration (MIC) and minimal fungicidal concentration (MFC) were performed according to the CLSI guidelines.

The MIC was determined by the microdilution broth method using 96-well plates. Initial C. albicans inoculum was prepared and adjusted to 5.0 x 10⁷ - 2.5 x 10⁸ colony forming units (CFU)/ml. Stock solutions of the compounds were prepared in 100% DMSO followed by a serial two-fold dilutions in Roswell Park Memorial Institute (RPMI) 1640 medium. The tested compounds’ drug concentrations were in the range 0.01-100 to μM in 1% DMSO. The plates were incubated at 37 degrees Celsius for 24 hours. The MIC was determined as the lowest concentration of the compound that inhibited microorganism growth. The MIC results were compared to the vehicle control (1% DMSO), positive control (fluconazole; fluconazole concentration of 32.65 μM was used in this experiment based on its MIC against C. albicans established from our previous experiments) and negative control (RPMI 1640). The MFC was performed by subculturing 20 μl of the samples from each of the microplate wells on agar Sabouraud plates, which were incubated for 48 hours. MFC was determined as the lowest concentration of the compound to show no fungal growth. Results were compared to the vehicle control (1% DMSO), the positive control (fluconazole) and the negative control (RPMI 1640).
Co-Culture Model

Cytotoxicity Test

Cytotoxicity assays were performed on fibroblast cells (ATCC:CRL2014), grown in Dulbecco’s Modified Eagle’s Medium (DMEM)/10% FBS (Lonza, Walkersville, Md.) at 37 degrees Celsius in 5% CO2. Based on the initial antifungal screening, the alkaloids were added simultaneously with the C. albicans and the fibroblasts at day two of the experiment, after the initial 24-hour period of seeding the fibroblasts on the wells. Prior to adding the yeast, the morphology of the fibroblasts was examined in an optical microscopy to ensure cell viability. The alkaloids remained incubated in the well plates of C. albicans and fibroblasts with medium in a volume ratio of 1:10 for 72 hours in order to allow for adequate exposure to the treatment as well as maturation of biofilm. Tested compounds at concentrations in the range of 0.01-100 μM were dissolved in 1% DMSO and added to the plates. After the screening of the alkaloids selected for this study, berberine hydrochloride (BER–HCl) was selected for further in vitro tests using a co-culture model and colony forming unit (CFU). In addition, a toxicity test was performed with fibroblast cells. The fibroblast suspension was prepared and adjusted to $1.0 \times 10^5$ cells/ml and plated in 96-well plates. The plates were incubated at 37 degrees Celsius in 5% CO2 for 24 hours, which were then replenished with fresh medium. Toxicity tests performed with BER HCl were repeated with higher concentrations up to 500 μM. The plates were then incubated at 37 degrees Celsius for 24 hours. Cell viability analysis was performed by adding CellTiter-Blue (CellTiter-Blue Viability Assay, Promega Corp., Madison, Wis.) to the cells and incubating the plates for three-and-a-half hours. Fluorescence was

<table>
<thead>
<tr>
<th>Substance</th>
<th>Molecular formula/ weight (g/mol)</th>
<th>Molecular structure</th>
<th>Antimicrobial activity</th>
</tr>
</thead>
</table>
| Berberine hydrochloride    | $C_{20}H_{18}ClNO_4/371.81$       | ![Berberine structure] | C. albicans SC5314  
MIC: 20 μM  
MFC: 90 μM |
| Cinchonidine               | $C_{19}H_{22}N_2O/294.39$         | ![Cinchonidine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Cytisine                   | $C_{11}H_{14}N_2O/190.24$         | ![Cytisine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Gramine                    | $C_{11}H_{14}N_2/174.24$          | ![Gramine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Lappaconite hydrobromide   | $C_{25}H_{45}BrN_2O_4/665.61$     | ![Lappaconite structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Matrine                    | $C_{15}H_{24}N_2O/248.36$         | ![Matrine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Oxymatrine                 | $C_{15}H_{24}N_2O_2/264.36$       | ![Oxymatrine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Palmatine chloride         | $C_{21}H_{22}ClNO_4/387.85$       | ![Palmatine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Piperine                   | $C_{17}H_{19}NO_3/285.33$         | ![Piperine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Rotundine                  | $C_{21}H_{25}NO_4/355.43$         | ![Rotundine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Sinomenine                 | $C_{19}H_{23}NO_4/329.39$         | ![Sinomenine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Sophocarpine               | $C_{15}H_{22}N_2O/246.35$         | ![Sophocarpine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
| Synephrine                 | $C_{8}H_{13}NO_2/167.20$          | ![Synephrine structure] | C. albicans SC5314  
MIC: >100 μM  
MFC: >100 μM |
read using the DU-650 spectrophotometer plate reader with excitation at 550nm and emission at 585nm. Cytotoxicity of the tested groups was compared to those of the vehicle control (1% DMSO), positive control (fluconazole) and negative control (RPMI 1640).

**Fluorescence Microscopy and Colony Formation Unit**

A co-culture model was conducted by culturing fibroblast cells and *C. albicans* together in a sterile 48-well plate. First, oral fibroblast cells (ATCC: CRL2014) were seeded in DMEM with 10% fetal bovine serum (FBS) at 37 degrees Celsius in 5% CO2 for 24 hours. The medium was then replaced with an inoculum of 5 × 10^2 to 2.5 × 10^3 CFU/ml *C. albicans* (ATCC: SC5314) grown in DMEM without FBS. Fibroblast cells and *C. albicans* were treated daily with 20μM, 50μM, 100μM and 200μM of BER HCl. The plate was then incubated at 37 degrees Celsius in 5% CO2 for 24 hours. The vehicle control tested was 1% DMSO, positive control was fluconazole and negative control was pure DMEM. The distribution of dead and live fibroblast cells was examined using the Viability/Cytotoxicity Assay Kit for Animal Live and Dead Cells (green: live fibroblast cells; red: dead fibroblast cells). (Sigma-Aldrich, St. Louis) was used to stain *C. albicans*. The fluorescence images only provided qualitative examination of the fibroblasts, both live and dead, as well as *C. albicans* distribution. However, the cytotoxicity test was used to measure the percentage of cell viability by incubating the plates with CellTiter-Blue added to the cells and then measuring the fluorescence using a spectrophotometer. The percentage of cell viability for each sample tested was normalized to the vehicle control group, which was set to have 100 percent viability. Fluorescent images of the double staining were captured using fluorescence microscopy (EVOS microscope, Life Technologies, Carlsbad, Calif.). After 72 hours of treatments, colony formation unit (CFU) was determined by suspending each sample of biofilm in 1 ml of PBS and plating 20 μl of the suspension on Sabouraud dextrose agar plates, which were incubated at 37 degrees Celsius in 5% CO2. After 24 hours of incubation, the number of *C. albicans* colonies was counted and the data was transformed in Log 10. All procedures were repeated at least twice for reproducibility.

**Statistical Analysis**

All results were expressed as the mean ± SEM, using one-way analysis of variance (ANOVA) and Tukey test. The level of statistical significance was set at 0.05.

**Results**

**Susceptibility Test**

The **TABLE** shows the MIC and MFC values of 13 alkaloid compounds against *C. albicans*. Based on the initial MIC and MFC screenings, BER HCl had a strong antifungal potential with the lowest MIC in the range of 20-50 μm and was, therefore, selected for further in vitro analysis using a co-culture model and colony-forming unit (CFU) assessment of biofilms.

**Co-Culture Model Fluorescence Microscopy**

After treatments of berberine hydrochloride (20-200 μM), *C. albicans* growth was inhibited (**FIGURE 1**). To rule out the possibility of cytotoxicity in the bioassay, gingival fibroblasts were exposed to various concentrations of BER HCl at varying concentrations, as indicated in **FIGURE 1**. At a fourfold dilution, all dilutions of the compound inhibited Candida formation. However, when compared to the negative control group, higher concentration of BER HCl of 200μM had a visible adverse effect on fibroblast cells. In regard to *C. albicans* formation, the positive control group and all concentrations of BER HCl were able to reduce fungus growth.
Colonies Forming Unit

The table shows that the number of colony forming unit (Log 10) of the tested groups was significantly reduced in comparison to that of the negative control group (p < 0.001). Moreover, the inhibition of Candida formation was significantly higher in groups treated with fluconazole or in those treated with BER HCl at high concentrations of 100 and 200 μM (p < 0.001).

Toxicity Test

The cytotoxicity result of BER HCl showed nontoxic effects on fibroblast cells, up to 90μm with 100 percent cell viability (Figure 3). At higher concentrations up to 500μm, cell viability was slightly greater than 50 percent.

Discussion

A new spectrum of human fungal infections is increasing because of the systemic diseases responsible for the immunocompromised system failure. Historically, natural products have been a rich source of antifungal drugs, and among them, alkaloids from some plants have already been reported to affect fungus' biological functions at very low concentrations. In this study, we reported an antifungal activity provided from a screening of alkaloids in order to evaluate their potential antifungal activity against C. albicans.

Numerous studies with alkaloids support the benefits of this group of compounds. Vindoline I-IV were isolated from the Catharanthus roseus, a common plant used by folkloric medicine in countries such as India, South Africa, China and Malaysia to treat diabetes. A study performed with breast cancer stem cells showed antiproliferative activity from two alkaloids: noscapine and papaverine. Moreover, antibacterial activity was found in vasicine, an alkaloid extracted from Adhatoda zisata Nees, a plant in Ayurveda and Unanni medicine, against E. coli. Antifungal properties against C. albicans were also demonstrated.

Some studies showed that alkaloids extracted from plants exhibited potential antifungal activities, which was in agreement with our results. However, from the natural compounds library used in this study, only BER HCl demonstrated promising antifungal activity against C. albicans. Among the natural products described so far in the literature that possess antifungal activity, another example is matrine, which at 1 mg/ml was shown to inhibit nearly 80 percent of planktonic growth. However, our results did not show the same potential when compared to the negative control, as both MIC and MFC were determined to be at concentrations greater than 100 μM. Future research is needed to confirm matrine's potential and its possible mechanism of action against Candida albicans.

Palmitine, an alkaloid found in a plant called Phellodendron amurense (commonly known as the Amur cork tree), has shown antifungal activity against Microsporum canis, a fungus that causes dermatitis in humans and other animals. However, it did not show strong antifungal potential on C. albicans, as our results illustrate a high MIC/MFC greater than 100 μM. However, further studies are necessary to confirm its antifungal activity against C. albicans.

BER HCl is an isoquinoline alkaloid commonly used in Chinese medicine with broad bioactive properties against a large number of diseases, including fungal and inflammatory diseases. Our study provided new information on the antifungal potential of BER HCl against C. albicans. Nakamoto et al. (1990) investigated the antifungal effects of BER HCl in comparison to amphotericin B and determined that MIC values of BER HCl were higher when compared to amphotericin B. Other studies have illustrated a strong synergistic effect of berberine and fluconazole, as the combinatorial effect resulted in the inhibition of C. albicans.
growth. Our results showed that BER HCl (100 and 200 μM concentrations) decreased CFU/ml to similar values as those of the positive control treated with fluconazole 32.65 μM (Figure 2).

Gu et al. suggested that berberine at high dosage (> 0.05 mg/ml) exhibited cytotoxicity on fibroblasts. Our study also demonstrated that the cytotoxicity result of BER HCl showed nontoxic effects on fibroblast cells, up to 90 μM with 100 percent cell viability. Berberine is usually well-tolerated by humans, however, higher doses might cause nausea, diarrhea and allergies. Nevertheless, in this study, at higher concentrations up to 500 μM, cell viability was slightly greater than 50 percent. Morphological changes were observed in human promonocytic U937 cells with 75 mcg/ml of berberine for 24 hours leading to apoptosis. It is necessary to test and regulate berberine dosage according to safe levels and further to ensure its use for pharmaceutical purposes.

Recently, an in vitro study reported that berberine treatment had detrimental effects on the cell wall integrity of C. albicans. In addition, berberine was able to interfere with the calcineurin pathway, leading to dysfunctional mitochondria followed by apoptosis due to the reactive oxygen species by the action of berberine. C. albicans can survive and recover from stressful conditions because it has the ability to tolerate stress by adopting different regulatory routes and thus, influence the susceptibility of Candida cells to react and block other drugs’ antifungal activity. BER has a potential to overcome Candida protective mechanisms for survival and growth. Future research may investigate the multitarget effects of berberine as well as other alkaloids against C. albicans. In addition, future in vivo studies are necessary to validate the efficacy of this natural compound for preclinical use in the prevention and/or treatment of oral candidiasis.

In conclusion, a screening of alkaloids against C. albicans was performed to identify natural products with antifungal activity. Berberine hydrochloride (BER HCl) has shown activity against C. albicans at low concentration. Furthermore, studies in order to evaluate its cytotoxicity and antimicrobial activity must be explored, thus, guiding the clinical and pharmaceutical intentions in a secure and safe manner.

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Prevalence of Enamel Markings on Third Molars

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ABSTRACT The purpose of this study was to measure the prevalence of enamel markings in routinely extracted third molars. One hundred donated third molars were examined. All had some marking(s). Caries was almost universal; white snowcapping of cusps and ridges was extremely common; pit and valley defects were very common; spots and bands were very common, most were white; horizontal grooves were common; linear enamel hypoplasia, considered to be a true developmental defect, was rare.

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Wide varieties of markings appear on human tooth enamel. Some are of shape, others are of color or texture. Pit and valley defects may be produced by perturbation to ameloblastic function or they may simply be a variation of anatomic form. Linear enamel hypoplasias (LEHs) are generally thought to represent severe systemic stress events, affecting an entire cohort of ameloblasts contemporaneously during mineralization. LEHs are generally reported to be narrow horizontal grooves consistent with a group of contemporaneous ameloblasts being affected by a stressor, but vertical LEHs have also been reported. Wide rounded grooves may reflect a period of ameloblastic hypofunction, but they may simply be a variation of normal anatomy. White spots may reflect hypomineralization during tooth formation, acquired carious demineralization, fluorosis or rare genetic defects. Brown spots are considered to be less common and can be associated with fluorosis or caries. Likewise, white or brown bands may reflect a period of ameloblastic hypofunction or acquired carious demineralization. Cusps and cuspal slopes of newly erupted molars are often marked by white markings or “snowcapping.” It is unclear as to whether these markings are of developmental or acquired origin and what is normal or abnormal.

Caries is one of the most prevalent acquired diseases worldwide, with enormous impact, suffering and cost. Caries begins as reversible white spot demineralization lesions in enamel. These may remain static, progress slowly or progress rapidly. The term molar-incisor hypomineralization (MIH), a clinical syndrome, has been used to describe the presence of acquired, demarcated opacities and posteruptive enamel breakdown in the permanent incisors and first molars. However, MIH has also been described as being a developmental defect of enamel (DDE). Reported prevalence rates of MIH have varied widely, from 2 percent to 75 percent. The etiology of MIH, developmental or acquired, remains a contentious issue. One recent systematic review found that DDE was associated with caries. However, it is not clear that the enamel markings ascribed as being DDE were actually of developmental origin. It is possible that white markings ascribed to DDE were in fact acquired through demineralization after eruption. Without either longitudinal evaluation of affected surfaces from the time of eruption or of destructive subsurface histological evaluation, definitive knowledge of the etiology of enamel markings cannot be understood. 

Standard dental anatomy texts describe the most common anatomical variants rather than attempting to capture the wide range of human tooth enamel form and color. Furthermore, markings that appear to be similar clinically may have very different origins. Knowledge of the types and prevalence of enamel markings would assist clinicians in distinguishing the normal from the abnormal and in identifying those patients affected by or at risk for developmental or acquired defects, as well as in advancing understanding of the etiologies of enamel markings.

The purpose of this study was to create a third molar model for evaluating enamel markings and to measure the prevalence of enamel markings in routinely extracted third molars.

**Materials and Methods**

**Patient Population**

Patients presenting for extraction of lower wisdom teeth were recruited at the University of California, Los Angeles, School of Dentistry, Oral and Maxillofacial Surgery Clinic. All consenting patients requiring third molar extractions between June 2011 and September 2013 were included. This clinic has a socioeconomically diverse patient population because it provides specialty services to patients covered by Medi-Cal and is located in an extremely affluent part of Los Angeles. Census data has indicated that the proportion of Los Angeles city and county residents from disadvantaged backgrounds and racial and ethnic diversity is higher than California or national averages. Some selection biases may have occurred. A sample size of 100 was attained. Given prior prevalence data, this was sufficient to measure the major types of enamel markings. Most wisdom teeth are extracted from adults 18 years or older. Our subjects in this age group were 18 years old or older and able to provide informed consent. In order to ensure patient confidentiality, no personal identifiers were collected. Although the city of Los Angeles has long provided fluoridated water, many smaller communities in Southern California still receive mixtures of fluoridated and nonfluoridated water. Hence, the fluoridation status, even for subjects who grew up in Southern California, cannot be certain. Essentially, this investigation was an observational study of a convenience sample.
**Subject Recruitment**

Institutional Review Board approval was obtained (UCLA IRB #10-001874). Subjects were recruited using a posted flyer asking interested patients to contact the clinic receptionist. Next, recruiters used a short eligibility screening according to a standardized script to determine eligibility. Finally, completion of informed consent occurred in a private room. Study personnel provided participants with instructions to apply an anonymous coded sticker to a plastic bottle containing thymol solution for their extracted teeth on the day of their extractions. Subjects were given a $10 gift card upon consent, whether or not they later donated their extracted teeth. Plastic bottles with extracted teeth were collected at the end of each day.

**Third Molars**

Wisdom teeth were used because their enamel forms during childhood and they are available. They are commonly extracted prophylactically or because of painful pericoronitis in young adults. Thus, sufficient quantities of third molars, which would otherwise be discarded, could be obtained for examination. This study was limited to third molars, whereas most prior studies have focused upon the entire dentition or on selected groups of teeth. Third molar enamel development starts at ~8 years of age and is typically completed three years later, representing key prepubertal developmental periods. In this study, all consenting patients had already been treatment planned for third molar extraction and were willing to donate their extracted teeth. The sample potentially included a range of teeth from the unerupted to the long exposed. However, subjects overwhelmingly appeared to be young adults rather than middle aged or older individuals. Children younger than 18 were specifically excluded from eligibility. Additionally, subjects generally appeared to be attending for extraction of partly erupted third molars following episodes of pericoronitis. No personal identifiers, including date of birth, were collected. The tooth specimens were solely identified by bar codes. Some of the included teeth may have been misidentified as being third molars by the surgeons performing the extractions, however, all had anatomy consistent with third molars upon extraoral examination.

Diagnostic criteria were based on the Winter and Brook and Witkop and Sauk classifications for use in general purpose epidemiological studies. Those classification systems were modified in consideration of the defects actually seen in our tooth sample. Six main types of enamel defects, namely pit and valley defects, LEH, grooves, spots, banding and snowcapping were seen (Figure). Pits included single, multiple and valley defect variants (Figure). LEH included horizontal and vertical subtypes. Grooves included horizontal shallow rounded and vertical types. Spots contained the above subtypes, as well as single, multiple and translucent variants (Figure). Banding included diffuse, well-defined white and brown subtypes. In addition, the prevalence of prominent perikymata was recorded. The International Caries Detection and Assessment System (ICDAS) was used to measure caries. The ICDAS scale is reproducible, valid and correlates with histology. It is possible that acquired demineralization prevented the identification of some developmental defects. Although wear could also potentially prevent the identification of some defects, none of the teeth exhibited any facetting upon observation or histology, again suggesting that these teeth had been extracted shortly after eruption.

The prevalence of markings and their subtypes were calculated as percentages.

**Results**

Unwanted extracted third molars were readily available for our study. Participating subjects donated 100 unwanted, freshly extracted third molars. Many purported enamel defects were very common in this sample (Table). All teeth had at least one marking; many teeth had multiple markings. Pit or valley defects, snowcapping, spots

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**Tooth Evaluation**

After extraction, each tooth was carefully cleaned to remove any gross plaque or debris and stored in a 0.1% thymol solution. Enamel defects were analyzed using plain eyesight, 2.5x magnification loupes and projected images made using macrophotography with five views per tooth (buccal, distal, lingual, mesial and occlusal). A consensus decision of two trained and calibrated examiners described each tooth. After calibration, levels of interexaminer agreement for the assessment of each third molar were substantial to almost perfect (kappa value = 0.81 − 0.95). If a patient donated more than one wisdom tooth, the tooth with the largest number of visible defects was included.

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**The fluoridation status, even for subjects who grew up in Southern California, cannot be certain.**
and banding were found in a majority of the teeth. Shallow rounded grooves were also quite common, but LEHs were rare. Acquired caries was common, being found in 95 percent of the sample. Prominent perikymata, a normal feature, were no more common than some of the purported anomalies or defects.

Pits and valley defects were extremely common with almost two-thirds of the sample exhibiting pitting (Table). Teeth with multiple pits were more common than teeth with single pits. The size of the pits was evenly split between those bigger or smaller than 1 mm. Linear valley defects were considerably less common than pits.

LEH was rare, found in only 8 percent of the sample (Table). All LEHs were horizontal; no vertical LEHs were identified.

Shallow rounded grooves were quite common, exhibited by 40 percent of the sample (Table). Most of the shallow rounded grooves were located in the cervical quarter of the crowns. However, vertical grooves in positions unrelated to cuspal form were rarer, being exhibited in just 10 percent of the sample.

Spots were extremely common and were exhibited on almost two-thirds of the sample (Table). Teeth with multiple spots were more common than teeth with single spots. White spots were approximately an order of magnitude more common than brown or translucent spots.

Banding was extremely common and was exhibited by almost two-thirds of the sample, as was spotting (Table). Diffuse banding was approximately an order of magnitude more common than well-defined banding. White banding was approximately an order of magnitude more common than brown banding. Banding was generally located in the cervical third of the crown, often coincident with shallow, rounded grooves.

Snowcapping was also extremely common at 83 percent (Table). All but one of the teeth exhibiting snowcapping also exhibited decay, having an ICDAS score of 1 or more.

Caries was extremely common, affecting 95 percent of the sample; 61 percent of the sample had established decay according to the ICDAS criteria (Table).
Prominent perikymata were discerned on approximately two-thirds of the sampled teeth (TABLE). Perikymata are a normal surface feature, the manifestation of striae of Retzius as cohorts of ameloblasts reach the tooth surface and senesce.

Few subjects donated contralateral or opposing third molar teeth, but in those cases, like findings were discerned.

**Discussion**

Enamel markings were extremely common. Pit and valley defects, grooves, spots, bands and snowcapping were found on a majority of teeth, whereas LEHs were much rarer.

LEHs are considered to be a useful biomarker of severe stress in anthropologic study. The high prevalence of enamel markings in this population could be attributed to several factors. Unusual, these teeth were examined with great care using photography with indirect lighting and high magnification. Small defects that usually go uncounted or unseen may have raised the found prevalence.

Snowcapping was an extremely common enamel marking. The vast majority of the teeth in this sample exhibited snowcapping of cusp tips and slopes (TABLE). Snowcapping almost universally presented as multiple bright white areas on cusps and slopes (FIGURE). This study could not determine the origin of this snowcapping whether developmental, acquired or normal. It is possible that this white cuspal enamel becomes worn away in the years after eruption and that snowcapping might become less evident over time.

The ameloblasts forming the outer snowcapped enamel cusp tips and ridges were at the end of their lives, whereas other ameloblasts were contemporaneously forming normal unaffected enamel. Therefore, a change in this outermost enamel cannot be ascribed to developmental stresses because other contemporaneously formed enamel appeared normal.

Snowcapping may be related to cellular senescence and death, but it was generally localized to the cusp tips and slopes and not to the other tooth surfaces. Amelotin, a recently discovered protein, is involved in the formation of the last-formed final enamel and might be related to snowcapping. However, a genetic etiology appears unlikely because snowcapping was only found on the cusps and their slopes, not over the entire tooth surface, which is all eventually covered by last-formed enamel.

The patterning of cuspal snowcapping could be explained by the pattern of eruption of these molars; the cusps erupt first and have the longest exposure to the oral environment. If the oral environment had favored demineralization or caries, these areas would have the longest exposure and could be more likely to show whiteness caused by demineralization. Little is yet known about snowcapping. It is possible that young adults included in this study had moved from a more nutritive home environment to independent living with behavioral and dietary habits that put them at high risk to carious demineralization.

Shallow rounded grooves were common, mostly being found in the cervical areas; all were horizontal. Although shallow rounded grooves could be indicative of true enamel hypoplasia, they were so common as to be considered normal in this sample of third molars.

Spots and bands were common. Banding of developmental origin in the horizontal plane can be indicative of stress affecting multiple ameloblasts simultaneously. The vast majority of spots were white and a few were brown. Translucent spots were rare. No translucent bands were identified, but in the cases with multiple translucent spotting, the spots were generally formed contemporaneously. It is unknown whether translucent enamel is inferior, equal or superior to normal enamel, whereas it is widely believed that white or brown enamel is less dense than normal enamel. Well-defined banding was considerably rarer than diffuse banding.

Caries was extremely common in this cohort, with 95 percent of teeth being affected. This estimate may appear high.
However, there is no doubt that almost three-quarters of the teeth that had established or severe decay. The probability of carious attack is the highest in the couple of years following eruption of first and second molars and the same is likely true for third molars. Many teeth exhibited a variety of defects, most often including decay and snowcapping. Decay could have prevented identification of other defects.

This study was limited to third molars. Most prior studies have not focused on third molars, but on the entire dentition or on representative groups of teeth. Most prior studies examined teeth in vivo, whereas this study examined teeth ex vivo using magnification and optimal lighting conditions. Data on third molar markings is rare, but these teeth are available for donation and their enamel is formed during childhood. It is possible that a similar study could be performed using unwanted premolars extracted from children for orthodontic purposes.

Now that the high prevalence of a wide variety of enamel markings has been established, further study is needed to differentiate the causative etiologies of markings that may appear to be similar, but are of completely different origin. Surface viewing at a single moment in time cannot determine etiology. Unneeded, donated third molars provided an ideal basis for study because they can be obtained in sufficient numbers and can be sectioned for hard tissue histology to examine subsurface features microscopically for evidence of etiology without any harm to their donors.

Conclusions
We drew the following conclusions from our research:

- Freshly extracted third molars were readily available for study.
- Enamel markings were universally common in the studied third molar cohort.
- Caries was extremely common, almost universal.
- Snowcapping was extremely common.
- Pit and valley defects were very common.
- Spots and bands were very common; most were white.
- Horizontal grooves were common.
- Many marking types were so prevalent that they may be considered to be normal.
- Linear enamel hypoplasia, considered to be a true developmental defect, was rare.

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Free eDelivery.

Available for iPhone, Android or Kindle Fire. Check it out at cda.org/apps.
Nonodontogenic Sources of Dental Pain

Scott E. Schames, BS; Michael Jordan, RN, MSN, MBA; Hila Robbins, DMD; Lenard Katz, BA; and Kaitlyn Tarbert, RDH

ABSTRACT Nonodontogenic sources of dental pain can be extremely challenging to diagnose. It is critical to establish a proper diagnosis to ensure that treatment is directed toward the source of the pain rather than the site of the pain.

Nonodontogenic pain can be a major source of frustration for patients and dentists. Dentists are trained to detect the source of a patient’s dental and orofacial pain based on the description and clues provided by patients and results of diagnostic tests. Odontogenic causes of dental pain — decay, abscess, cracked tooth, periodontal disease, sinus infection, etc. — can be challenging, but are part of the routine examination process in determining a proper diagnosis to ensure a successful treatment plan.

However, when the dental pain is of a nonodontogenic origin, the route to discovery may be less straightforward. Not only can nonodontogenic dental pain be extremely painful, but such pain can also be indicative of other, sometimes serious health problems. Therefore, it is important to identify the cause of pain as quickly as possible, even when the source of the pain is located in another part of the body.

The key to proper diagnosis of nonodontogenic dental pain is to try to recreate and mimic the chief complaint of pain during the examination; otherwise, the diagnosis may be incorrect and suspect. If the source of the dental pain is not identified during the examination, the dentist must search for other causative factors. It is critical to establish a proper diagnosis to ensure that treatment is directed toward the source of the pain rather than the site of the pain. Until the source of the pain is found, the diagnosis is actually a differential diagnosis, which can only be verified with further testing, consultation with other medical specialties and ultimately confirmed by successful treatment.

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Myofascial Odontalgia From Facial Muscles

Myofascial causes of pain should be investigated during an examination of dental pain. Myofascial pain from a muscle trigger point (TrP) usually causes patients to feel a regional, dull ache at the muscle site, but it can also refer pain to distant ipsilateral sites, including teeth.1,2 According to Travell and Simons, pain

referral patterns vary based on the muscle. For example, TrPs in the masseter muscle can refer pain ipsilaterally to maxillary and/or mandibular posterior molar teeth, the temporalis muscle can refer pain ipsilaterally to each of the maxillary teeth and the anterior digastric muscle can refer pain to mandibular anterior incisors.

In a case reported by Mascia et al., a patient complained of pain in the upper and lower left quadrants of her teeth, radiating to the ear and temporal region. A thorough examination did not return an odontogenic cause; therefore, a myofascial examination was performed. Palpation of the left masseter at the angle of the mandible duplicated the patient’s pain. By isolating the TrP in the muscle between the middle and index finger, a diagnosis of myofascial pain was established. The patient was treated with a non-epinephrine-containing injection near the trigger point, which was successful in eliminating the pain. Mascia et al. note that epinephrine is not used because of its toxic effect to muscle and fat tissue.

The tongue is another organ/muscle that can be the source of nonodontogenic tooth pain. Ankyloglossia or tongue-tie is commonly characterized as a short lingual frenum that limits tongue mobility. The tongue, which has origin and insertion points ranging from the internal portion of the mandible down to the hyoid bone, has many myofascial and muscular attachments to the cervical muscles.  

Olivi et al. report that ankyloglossia is associated with hyperactivity of the suprahyoids and forward displacement of the head. The restricted tongue requires the use of accessory muscles to perform functions such as breathing, breastfeeding, chewing and swallowing. This hyperactivity of the cervical muscles causes shortening of the muscles, induces forward head posture and may ultimately lead to the formation of TrPs that directly refer pain to the teeth. Therefore, restrictions of the tongue should be examined when searching for nonodontogenic sources of tooth pain.

Additionally, ear infections may refer and mimic dental pain. In some cases, orofacial pain that is associated with the eruption of molar (primary and/or permanent) may actually be caused by an ear infection that has referred pain to that region. The area around the ear has multiple nerve innervations including the trigeminal, facial, glossopharyngeal and vagus nerves, as well as superficial sensory branches from the cervical plexus. The referral pattern of pain is due to the overlapping sensory innervations of the trigeminal nerve around the mouth and ear during development.

Myofascial Odontalgia From Muscles Beyond the Head

Muscles in the neck and torso can also cause orofacial pain. Travell and Simons described that masseter muscle TrPs may originate as satellites from TrPs in the trapezius muscle as well as the sternocleidomastoid muscle, both of which can refer pain to the facial area and restrict the opening of the mouth. As counterintuitive as it may seem, muscles distant from the head and neck area can also produce dental pain. As Travell noted, the soleus muscle in the leg near the calf refers pain into the orofacial area. Schames et al. also showed that not only is there a pain referral pattern to the orofacial region from a TrP in the pectoralis muscle located in the armpit, but also there appears to be an association between the soleus and the pectoralis referral patterns.

Dental pain can also be referred from the diaphragm, the thin, dome-shaped sheet of muscle and tendon that separates the chest from the abdomen. The phrenic nerve innervates the diaphragm to control the movements that produce breathing. Bordoni and Zanier discuss that the phrenic nerve can affect the spinal trigeminal ganglia, stimulating “the last two branches of the trigeminal nerve, reaching the teeth and periodontal ligaments through the alveolar nerves,” resulting in dental pain.

Body posture, specifically of the head and cervical area, should be evaluated during patient examination for orofacial pain. Examples of poor posture include, but are not limited to, kyphosis of the thoracic spine, anterior rotation of the shoulders and forward head position. Alterations of craniofacial posture may develop from trauma (including birth trauma), thoracic breathing patterns, mouth breathing, habitual poor posture and epigenetic factors such as the Western diet and modern technology. For example, Lee et al. reported that subjects maintained a head flexion of 33-45 degrees from vertical while texting on their smartphones. Such a forward disposition produces a posterior rotation of the cranium, straightens the lordotic curve of the neck and overworks cervical muscles such as the sternocleidomastoid (SCM). Overactivation of the SCM can lead to malocclusions, compression of the temporomandibular joints, orofacial pain and as documented, the creation of TrPs that directly refer pain to the mandibular teeth.
Cardiac Odontalgia

As many case studies have shown, orofacial pain can be of cardiac origin. Heart problems such as angina pectoris or acute myocardial infarction can refer pain to teeth and facial areas, with or without chest or arm pain.18

One of the first signs of cardiac distress can be pain on either side of the face due to stimulation of the trigeminal nerve. Rothwell explains that referral of ischemic pain to the face and mandible may stem from connections between the vagus and the trigeminal nerve nuclei.19

If a patient presents with dental or orofacial pain, and odontogenic, myofascial and nasal causes have been ruled out, it is prudent to consider that the pain could be of cardiac origin, particularly if the patient has one or more cardiac risk factors, such as advanced age, high blood pressure, obesity, etc. Anecdotally, there have even been cases in which young people have had dental and orofacial pain that was cardiac in origin.

According to Kreiner and Okeson, improper diagnosis of cardiopathic dental pain frequently leads to unnecessary dental treatment or, more significantly, delayed treatment of underlying cardiac disease, such as myocardial infarction.20

Diabetic Odontalgia

While periodontal infections, ulcers and inadequate wound healing are known issues associated with diabetes mellitus (DM),21 DM can also increase the sensation of dental pain. Because it affects the vascular system, DM causes reduced circulation in the tiny blood vessels entering the apices of the teeth, which in turn affects the exchange of nutrients, oxygen and waste, impairing healing.22

In a study examining the influence of adult-onset diabetes on orofacial pain, Rahim-Williams et al.22 reported that diabetic patients were more likely than nondiabetic orofacial pain sufferers to:

- Have daily pain.
- Experience disruption of daily activities and sleep.
- Make an emergency room visit for orofacial pain.

The study concluded that nociceptive pain is exacerbated by diabetes, particularly where diabetic patients can have a subjective experience of increased orofacial pain. According to the study results, the frequency of orofacial pain is more prevalent and more frequent among adults with diabetes than among those without diabetes.

One of the first signs of cardiac distress can be pain on either side of the face due to stimulation of the trigeminal nerve.

Nasal Sinus Lining Odontalgia

Harold Wolff, MD, a pioneer in the field of neurology, performed experiments uncovering referral patterns of pain between other parts of the cranium and the teeth. Dr. Wolff demonstrated that dental pain could originate from the sinuses. In his experiments, Dr. Wolff pressed a probe or faradic electrode against the wall of the nasal cavity at various trigger points.23 Dr. Wolff demonstrated that the nasal sinus lining, which is innervated by trigeminal afferents, has trigger points that refer pain to specific teeth. Dr. Wolff documented that stimulation in the superior nasal cavity produced pain ipsilaterally in the upper teeth, including the canine, the premolars and the first molar, with pain also felt in the maxilla above those teeth. Stimulation of the sphenoid sinus produced pain in the maxillary teeth. Stimulation of the lower lateral wall of the maxillary sinus resulted in pain in the maxilla and posterior maxillary teeth.

Dr. Wolff also applied electrical stimulation to patients’ teeth to determine if pain was referred to other areas of the upper body. He documented that stimulation of a maxillary premolar referred pain in the temporal region, forehead and scalp on the same side of the head in which the tooth was stimulated. Stimulation of a premolar in the mandible caused pain throughout the maxilla and mandible as well as the areas over the zygoma, temple and top of the ear. When a maxillary molar was stimulated, headache was induced in the temporal region, forehead and up to the vertex, with erythema (redness) occurring on the side of the face, mandible and neck.

Bruxism

Bruxism is a common cause of dental and facial pain. The excess pressures can cause stress, not only in the teeth, but also in the surrounding periodontal ligaments and facial musculature.24 To diagnose bruxism, dentists generally examine for dental wear facets from grinding, but it is also important to check for evidence of clenching by looking for scalloping (indentations on the sides of tongue) or linea alba (bite mark lines on the inside of the cheeks). Stress or pain can contribute to bruxism, and some antidepressant medications can also have the side effect of contributing to bruxism.25 Schames et al.26 discussed that nocturnal bruxism is a result of airway obstructions during sleep, so due diligence must be exercised when patients have evidence of dental or facial pain originating from bruxism.

Most recently, in January 2015, Kloeffler wrote, “The tightened muscles of the jaw narrow the airway during nocturnal bruxism, creating
intermittent hypoxia to the brain.” Therefore, we can infer that bruxism causes obstructions of the airway and must be treated appropriately.

Traditionally, dentists treat bruxism with an oral appliance, generically called a night guard appliance. However, it has been documented that when a regular bruxism night guard appliance is given to a patient to wear during sleep, this appliance can exacerbate nighttime obstructions of the airway. Therefore, the standard of care is that a patient cannot use a daytime bruxism oral appliance while sleeping, day or night. Rather, patients must use an oral sleep appliance, such as a mandibular advancement or tongue-retaining appliance during sleep. The oral sleep appliance serves not only to protect the patient from the ill effects of bruxism, but it also serves to bring the patient’s mandible and tongue forward, thereby promoting the opening of the airway.

Signs and symptoms of bruxism are also present in children with obstructive sleep apnea, with a variable prevalence of 3.5 to 40.6 percent. Tonsiloadenoidectomy is often rendered as the first line of treatment, but recent research has documented that signs and symptoms of obstructive sleep apnea were retained in a large percentage of pediatric patients following the removal of tonsils and adenoids. Furthermore, recent studies describe the importance of restoring continuous nasal breathing through the use of myofunctional therapy, as a solution to managing pediatric obstructions of the airway long term. Nasal breathing is associated with a reduction in the inflammation of oral tissues, a more favorable growth pattern of the face and a reduction in the apnea hypopnea index. Myofunctional therapy aims to promote neuromuscular repatterning of the oral and facial muscles, and to promote proper physiology of the orofacial complex during functions such as breathing, eating, speaking, sleeping and swallowing. Any patient who has bruxism should be referred to a sleep physician for more detailed polysomnographic sleep studies to determine the exact type of additional definitive care that the patient may require for nocturnal airway obstructions, such as the use of continuous positive airway pressure (CPAP), myofunctional therapy and/or surgery.

When a regular bruxism night guard appliance is given to a patient to wear during sleep, this appliance can exacerbate nighttime obstructions of the airway.

Barodontalgia
Frequent air travelers are aware of the phenomenon of a potato chip bag expanding during flight then contracting upon landing. During flight, a similar change in pressure can occur within the dental pulp chamber as well as within the bony trabeculae within the mandible. Barodontalgia is the occurrence of a toothache, maxillary sinus ache and/or mandibular bone ache due to the sudden increase or decrease of atmospheric pressure. Barodontalgia can be brought on by changes in cabin pressure during airline flights, climbing to higher altitudes and/or by the changes in water pressure while diving in water. Kollman concluded that the majority of the perceived pain was due to an inflamed pulp affected by altitude pressures. The nonodontogenic phenomenon of barodontalgia should be considered when seeking to identify the source and cause of the patient’s pain complaint.

Neuropathic Pain
Neuropathic pain can be triggered by various factors, but when developed, the pain experienced is generally different from myofascial pain. As indicated above, myofascial pain in usually experienced as a regional, dull, aching pain, while patients with neuropathic pain often report an electrical, burning, shooting or stabbing pain, which can be felt for several seconds when the trigger point (if known) is touched.

Trigeminal Neuralgia
Trigeminal neuralgia (TN) is the most common cause of facial neuralgia. It affects four to five people per 100,000 and affects women older than 40 years of age more often than men. Experiencing severe, shocking pain that can last seconds to minutes, the TN patient will generally be pain-free between occurrences. Although the source of the pain is internal, the patient may report seemingly innocuous sources of pain such as when brushing teeth, shaving or eating. Because patients with TN can suffer from similar symptoms to those caused by pulpitis, dentists should differentiate by administering dental topical surface anesthesia with an 8% xylocaine spray at the trigger zone to observe whether this reduces the pain.
Auriculotemporal Neuralgia

Unlike TN, which generally occurs without an obvious trauma, auriculotemporal neuralgia (AN) is usually caused by a traumatic event, such as a root canal or extraction. The source of pain from AN is believed to be irritation (whether by compression, friction or traction) of the auriculotemporal nerve, particularly where the nerve pathway crosses muscles, the temporal artery or other anatomical structures. Patients with AN may experience moderate to severe pain in the following areas: temporal region, the temporomandibular joint, the parotid and also in the auricular and retro-orbital region. Where pain from TN is experienced as quick flashes of shocking pain, Murayama et al. report that the pain from AN is often:

- Continuous, with exacerbations perceived as stabbing pains.
- Unilateral.

May be triggered by placing pressure on the periauricular region at the level of the tragus. In the case described by Murayama et al., a patient presented with intense pain of short duration near the left external ear and in the ipsilateral maxillary second molar. Pain could be triggered by touching his face and the region of the ear helix. During the examination, a shocking pain was triggered by touching the ear helix region, which referred to the maxillary left second molar and caused a burning pain in the ipsilateral temporal region. Though palpation of the TMJ and masticatory muscles caused no pain, palpation of the region of the auriculotemporal nerve triggered a burning pain radiating toward the temporal region.

Acoustic Neuroma

Acoustic schwannoma, is a benign tumor composed of Schwann cells on the eighth cranial nerve between the brain and the inner ear, which can cause dental pain through compression of the trigeminal nerve. Bisi et al. report that in patients presenting with TN symptoms, approximately 6-16 percent have intracranial tumors (the most common of which is acoustic neuroma). In making a differential diagnosis, early impairment of auditory activity is a common indicator of acoustic neuroma, particularly if the patient experiences hearing loss or tinnitus (ringing in the ears). An MRI should be used to determine if an intracranial tumor is present.

In a case documented by Mehrkhodavandi et al., a patient was initially treated for trigeminal neuralgia after reporting pain radiating from the lower lip to the teeth in the lower left quadrant, without an apparent odontogenic source. The patient also experienced ringing sounds and a congested left ear, as well as numbness, pain and tingling in arms, hands and fingers on both sides. When treatment for TN was unsuccessful, the patient was referred for MRI, which revealed a “moderate-size lobular left cerebellopontine angle mass lesion consistent with acoustic neuroma with compression of the left trigeminal nerve secondary to the lesion.”

Central Sensitization

Dentists should be aware of central sensitization, which is of particular importance in cases of chronic pain. After a traumatic event such as a heavy blow to the masseter muscle, or after repeated heavy bruxism, the perceived pain can become centrally sensitized due to central nervous system hyperexcitability leading to long-term changes in the nervous system. Central sensitization occurs when nociceptive afferent nerve fibers branch extensively to terminate via synapses on many neurons, as opposed to just one synapse. Murray explains that effective synaptic connections result in the activation of neurons in higher centers of the brain for the correct perception of pain. However, there are ineffective synapses in which nerve impulses do not activate the next neuron in the pathway. If there is prolonged or intense pain stimulation, some of the ineffective synapses can become effective connections for other origins by central sensitization and neuroplasticity. In his example,
Murray states that a source of noxious stimulation in the region of the temporalis muscle could activate neurons that typically receive noxious input from the forehead or tooth pulps of the maxillary molars through previously ineffective connections. This means that the brain, and thus the patient, will perceive the pain source as the tooth pulps or forehead, when the pain is actually produced from the temporalis region.

It is important for dentists to realize that muscular and/or dental pain caused by repeated heavy bruxism may no longer respond only to treatment modalities focused toward the effect of bruxism, but may also require neurological treatment for the neuropathic component of the centrally sensitized pathways.

Shingles
Another cause of nonodontogenic pain is shingles (herpes zoster), the syndrome that affects nearly one-third of all Americans at some point in their lives.\(^{39}\)\(^{30}\) Varicella-zoster virus (VZV) initially causes varicella (chickenpox), and then remains dormant in nerves and can reactivate many years later. It is fairly easy to diagnose a patient with orofacial pain stemming from VZV in the trigeminal nerve because it presents with vesicles—ruptures on the skin that follow the neural dermatome. But shingles is much harder to diagnose when a vesicular rash is absent. This condition, zoster sine herpete (ZSH), can be a major source of consternation in patients and their treating dentists. The patient experiences a neurological attack with severe pain, but without the hallmark dermal symptom of vesicles. Many health care professionals may overlook ZSH, shingles without the characteristic appearance of vesicle formations, as a possible cause of dental and facial pain.

According to Kasahara et al.,\(^{30}\) patients describe ZSH pain as “deep, or as a boring, stabbing, aching, burning, prickling, tingling or [an] itching sensation.”

If a patient presents with severe neuropathic-type facial or dental pain, and imaging and other diagnostic tests have ruled out tumors and other neuropathic sources, the dentist should inquire if the patient has had chickenpox in the past to determine if ZSH is a possibility and then proceed with a serological examination. It is important to note that a patient may have unknowingly had the original VZV infection without the main symptom (chickenpox), so if the pain is consistent with shingles, it may make sense to consider it even if the patient states that he or she has not had chickenpox. If the patient is diagnosed with ZSH through a serologic examination, oral antiviral drugs should be prescribed by the appropriate health care professional to treat the disorder.

Neoplasias
Some tumors, aneurisms or cancers can also cause facial pain. While these scenarios are rare, they are extremely important to diagnose, as they may be time-sensitive issues. If neoplasia is being considered, imaging of the face, mandible, temporal, and paraneoplastic syndrome, a symptom that muscular and/or dental pain caused by repeated heavy bruxism may no longer respond only to treatment modalities focused toward the effect of bruxism, but may also require neurological treatment for the neuropathic component of the centrally sensitized pathways.

Sarlani et al.\(^{30}\) reported that facial pain could be the first manifestation of lung cancer, usually presented as unilateral facial pain in the ear, jaws and temporal region. The authors state that the referred pain is caused by two main sources: invasion or compression of the vagus nerve and paraneoplastic syndrome, a symptom of the production of circulating humoral factors by the malignant tumor cells.

Conclusion
Nonodontogenic causes of dental pain can be extremely challenging to diagnose. Because the origin of the dental pain may not be obvious, dentists must consider the details of the pain and patient history and try to recreate the chief complaint of pain. If the source of the dental pain is not recreated during the examination, the dentist must search for other causative factors that may be the source of the perceived pain.


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We’ve all heard the horror stories. The parents who lost their child’s baby pictures. The corporate executive who lost an important sales presentation. The new bride who lost video of her big day. The college student who lost his entire thesis and had to start all over from scratch.

Data loss can happen to anyone, at any time.

Even those on the cutting edge of technology are at risk. The creators of “Toy Story 2” nearly lost the film mid-production when a Pixar employee accidentally deleted data files and the backups failed. Luckily, a technical director had saved portions of the film on her home computer, and Buzz and Woody were brought back to life.

Data loss can even happen to dentists. The Dentists Insurance Company reports one Bay Area dentist lost all of his patient records when his hard drive crashed. When he attempted to restore the data by accessing his backups, he discovered his system hadn’t been backing up for two years.

“Having a backup system for storing information is critical, but it is also critical to check those systems,” said Sheila Davis, assistant vice president, Claims and Risk Management, TDIC. “If you don’t perform regular backups, and you don’t check to make sure those backups are functioning, you run the risk of losing everything.”

In the case above, the dentist did lose everything — and then some. Not only did he have to spend thousands of dollars to rebuild his system, he also had to recreate patient files. Because insurance companies require documentation for claim reimbursement, he had to retake patient radiographs. He also had to cross his fingers and hope that his patients were honest enough to pay what they knew they owed, as he had no billing records.

“There was a chance he would have to write off a significant amount of income, should patients refuse to pay their bills. Most people are understanding when it comes to computer glitches, but it’s still a risk,” Davis said.

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Whether from human error, viruses, technical malfunctions, natural disasters or theft, data loss can have a huge impact on any business. In fact, nearly half of all small businesses have experienced some form of data loss, according to online backup provider Carbonite.

Luckily, there are ways to avoid complete devastation. Topping the list is making sure your backups are run regularly and accurately. “Too often, people forget to check their backups,” Davis said. “Backups are a fail-safe. But even fail-safes should be double-checked.”

Cost is one of the major reasons business owners fail to back up their computers. There are upfront costs, such as hardware, and ongoing costs, such as monthly monitoring fees and storage fees. But the costs associated with a data loss are much greater, so it is a small price to pay.

“Dentists with up-to-date backups can be back to work within a few days,” Davis said. “Those without can spend weeks trying to get up and running again.”

Another reason dentists fail to back up their data simply comes down to time. According to the 2016 Backup Awareness Survey conducted by cloud storage provider Backblaze, 24 percent of computer users never back up their systems, and 42 percent only do it once a year.

“What could be more time consuming than having to rebuild your entire system and reconstruct all of your patient records?” Davis asked.

Most experts recommend real-time backups, also known as continuous backups, in which changes are automatically saved as they are made. That way, should a data loss occur, there will be no gaps in data recovery. Other options include conducting a full back up at a set point in time, such as once a day or once a week.

“The real question is, ‘what’s your risk tolerance?’” Davis asked. “How much data are you willing to lose? A week’s worth? A month? A year?”

Another consideration is where to store your backups. Many practice owners use external hard drives, but these can also fail if connected to a network and a virus strike or a malfunction occurs. Some use thumb drives or portable drives, which can be stored offsite. Many are now opting for cloud-based storage, which allows continuous backup and access.
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“We advise dentists who perform hard backups to disconnect the drive and store it offsite in a secure location. Better yet, we recommend investing in a HIPAA-compliant, cloud-based data backup service,” Davis said.

Christopher said it’s best to have multiple backups, stored in multiple locations.

“Keep one backup offsite in case some type of accident or disaster occurs,” he said. “Automate your backup system so there is less likelihood of human error. Regularly check the data on your backup devices to ensure it is useable and to ensure that backups are performing as expected.”

Whether personal or professional, a data loss can lead to unwanted expense, headache and stress. But by assessing your risk tolerance and taking a few preventative measures, it is possible to avoid catastrophe and get back up and running sooner.

TDIC’s Risk Management Advice Line at 800.733.0634 is staffed with trained analysts who can answer data backup and other questions related to dental practice.
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6106 SACRAMENTO’S EL DORADO HILLS No rush and no chaos here. Staff is Dream Team. Beautiful facility. 6100 SANTA CLARA

6102 SAN RAFAEL

6105 MODESTO Collected $430,000+ on 3-day week. 3-days of Hygiene. 5-ops. Central location. Successor should open 4th day.

6104 SANTA CLARA – CUPERTINO AREA Restorative practice. 2015 collected $1.55 Million with Profits of $694,000. Paperless and digital. Beautiful office. UCR Fees! Extremely attractive selling features available to retain the goodwill.

6103 SAN FRANCISCO’S UNION SQUARE Opportunity to acquire highly regarded practice with condo. Beautiful 5-ops, digital and paperless. 6th op available. 2015 collected $658,000.

6102 MARIN COUNTY’S SAN RAFAEL Great location near shopping mall. Collected $259,000 in 2015 on 3-days. Did $332,000 in 2014. Averages 10 new patients per month. 2-ops in 530 sq.ft. suite. Full price $150,000.

6100 SANTA CLARA Phenomenal launching pad for next Owner. Fantastic location, 5-op facility. Management not taking advantage of what is possible even though 2015 collected $758,000 with Profits of $323,000. Perfectly positioned to be a $1 Million+ year performer immediately! Needs young DDS.

6099 FAIRFIELD Collected $600,000 in 2015. 3-days of Hygiene. 4-ops with digital radiography.

6098 WEST PETALUMA Petaluma has become THE business center of the North Bay! Business parks are growing and young professionals are being drawn to this great family community per the unique amenities of this historic river city. Collected $468,000 with Profits of $199,000. 3-days of Hygiene with 4th day starting September. Full price $300,000.

6089 MOUNT SHASTA Small town living renowned for outdoor lifestyle. Best air and water! Escape Rat Race and corporate intrusion. 3-day week collected $881,000. Available Profits totaled $485,000. Digital radiography including Pano. Full price $350,000.

6070 VISALIA This practice is well positioned for its next caretaker. Strong Hygiene Department, beautiful facility, well equipped. Digital throughout. Collected $727,000 on part-time schedule in 2015. Extend hours and be busier. Best location!

---

**SOUTHERN CALIFORNIA**

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California DRE License 346937

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ANTELOPE VALLEY Has grossed $1.8 Million. Fantastic location. 60,000 autos pass by per day. 8 ops. Partnership for $250,000 or buy all.

ARCADIA Facility only. 3-ops equipped. $65,000 or $95,000 with Ortho.

BAKERSFIELD AREA 5-ops, next to McDonalds. 1,800 sq.ft. includes building. Grosses $40,000/month. Full Price with building $350,000.

BAKERSFIELD Established 55 years. 5-ops in 3,000 sq. ft. Will do $1 Million. Full Price $300,000. Building available for $350,000.

BELLFLOWER Established 60-years. Grossing $350,000. Full Price $240,000.

EAST LOS ANGELES One million Latinos in service area. PPS sold to Seller in 1985. Will do $1 Million in 18 months. Full Price $300,000.

EAST SAN FERNANDO VALLEY Absentee Owner. $8,000 per month Cap Check. 4-ops. Do a Million within a year.

INDIO 4,000 sq.ft. dental building. Full Price $650,000.

LADERA RANCH Grossing $650,000. Shopping center location.

LAGUNA NIGUEL Location, location, location! 4-ops with Panorex. Full Price $185,000.

LA JOLLA Established 20-years. 3-ops. Grossed $150,000. Super opportunity with immediate growth. Full Price $150,000.

LAWNDALE Hi identity. 2-ops. Full price $125,000.

LOS ANGELES HMO Grossing $1.2 Million. 5-ops. Full Price $1.2 Million.

LOS ANGELES HMO Does $4 Million. Full Price $1.2 Million.

NORCO – CORONA Will do $1.5 Million. 8-ops. Exquisite. Full Price $1.2 Million.

NORWALK Fantastic high identity location. 5-ops. Full Price $250,000.

ORAL SURGERY PRACTICE – LOS ANGELES Established 40 years. Beautiful 10 operatory office ready for merger.

ORANGE Established 60 years. 7-ops. Always $1+ Million. Full Price $600,000.

REDLANDS Shopping center. Grosses $350,000. Full Price $250,000.

RIVERSIDE Facility only. 4-ops. Full Price $50,000.

SOUTH ORANGE COUNTY BEACH CITY Grosses $650,000. 4-ops. Beautiful!

PERIO PRACTICE - PRESTIGIOUS BEACH CITY Established 40 years.

TORRANCE Established 12 years. 5 star building. 3-operators. Grossing $250,000. Full Price $195,000.

TUSTIN Dental building. Full Price $1.5 Million.

VENTURA - OXNARD 5-ops. Grossing $850,000. High identity. Full Price $685,000.

YUCCA VALLEY 8/10th of an acre. Great highway visibility. Full Price $250,000.

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4117 SAN JOSE GP  
Incredibly desirable location at the corner of two major intersections in West SJ near the Saratoga border. Offering 40+ yrs of goodwill. 3 ops in 1,200 sq. ft. Practice grossing btw $175K - $215K. Approx 175+ active patients. 2 days of hygiene. Seller will help for a smooth transition. Asking $125K.

4103 SAN FRANCISCO GP  
Vibrant downtown location in historic high-rise bldg. Retiring doctor offering 30+ years of goodwill. 4.5 days of hygiene, 1,500+ active patients, 20-25 new patients/mo. Gorgeous, spacious facility in approx. 2,500 sq. ft. 2015 GR $796K. 2014 GR $768K. Average adjusted net income $274K+ Asking $599K.

4085 SANTA ROSA GP & BUILDING  
Practice and R/E are offered for sale in a well-established medical/dental complex conveniently located near Memorial Hospital. 3 fully equipped ops in 1,200 sq. ft. Approx 750 active patients. Average Gross Receipts of $264K with adj. net of approx. $116K. Seller willing to help for a smooth transition. Price reduced to $125K for the practice and $245K for the real estate.

4108 HUMBOLDT COUNTY GP  
Well-established, high performing general practice boasts 6 fully equipped ops. in 2,900 sq. ft. free standing office w/Digital X-ray, 2 platinum Dexis sensors, & Cerec Omnicam & MCXL units. Loyal & stable pt. base in charming community. 3 fully equipped ops in 1,200 sq. ft. Approx. 500 active patients. Average Gross Receipts of $264K with adj. net of approx. $116K. Seller willing to help for a smooth transition. Asking $1,041K.

4091 HOLLISTER GP & PEDIATRIC  
Country living at it's best – small town feel with affordable housing, in quaint bedroom community to Silicon Valley. Fully equipped 1,600 sq. ft. office with 2 enclosed adult ops and 3 open pedo ops, near Hazel Hawkins Hospital. Turn-key practice, great opportunity for a pediatric dentist. Approx. 565 active patients. 2015 GR $219K. Seller is relocating but will help for a smooth transition. Asking $1,041K.

4114 CONCORD GP  
Well-established practice offering 30+ yrs of goodwill. Concord is on the verge of redevelopment of the old Naval Weapons Base later this year, which will cover 2,300 acres and include 12,000 housing units. The project will include Residential/Commercial/Recreational and Open Space. This practice opportunity is strategically located for growth potential due to the slated re-development. Office has 3 fully equipped ops in 836 sq. Average GR $360K+ on 2 doctor days. Asking $224K.

4121 NAPA GP  
Gorgeous, state-of-the-art office available in beautiful wine country! Incredible location with super high visibility on the corner of two major cross streets near Queen of the Valley Hospital. 7 ops in 3,250 sq. ft. facility. Seller retiring but would like to transition with buyer after the sale. 2,100+ active patients, 9 days of hygiene, 15-20 new patients/mo. 2015 GR $1.56M, 2014 GR $1.62M. Average Adjusted net income of $513K. Asking $1,151K.

4096 MENDOCINO COUNTY GP  
Seller offering well est. 48 year practice. Located in outdoorsman's paradise. Just 2 hours North of SF surrounded by redwood forest, vineyards and mountains. 950 sq. ft. office in single level building w/ 4 fully equipped ops. 2014 GR $565. Asking $300K.

4110 SANTA ROSA GP  
Don't miss this opportunity – absolutely gorgeous, state of the art office located within two major thoroughfares in the heart of Santa Rosa. Practice generating $2.1M+ in GR. Asking $1,436K.

4093 SAN JOAQUIN VALLEY ORTHO  
Established over 35 years with a solid reputation, near several referral sources in seller owned building. 2,500 sq. ft. office with 7 chair open bay in professional center on a well-travelled street with many retailers. Avg. Gross Receipts $763K. Seller retiring and willing to help for smooth transition. Asking $561K. The building is available to purchase as well for $608K.

4085 LAKE COUNTY GP  
Seller retiring from general practice located in a slower paced, relaxed community. Plenty of hunting and fishing and out door activities for the enthusiast. Approx. 1,600 square foot office with 4 fully-equipped operatories. Over 2,000 active patients, average $697K+ in Gross Receipts with an overhead of just 56%, and 4 doctor days per week. Asking $463K.

4105 STANISLAUS COUNTY GP  
Get away to a less demanding commuter friendly town. Seller retiring from practice est. over 30 years ago with loyal patient base in charming community with historic small town feel. 3 fully-equipped ops. in 1,200 sq. ft. office. Approx. 1,400 active pts. w/4 doctor days/week. 5 year avg. GR $647K+ w/approx. 50% overhead. Seller willing to help for smooth transition. Asking $428K.

4120 SF GP  
Well est. downtown family practice grossing over $1M with an avg. overhead of 61%. 5 fully equipped ops., in remodelled office. Retiring seller works 3.5 Dr. days/week. Seasoned, dedicated staff & loyal patient base. Terriff opportunity for experienced & confident dentist. Asking $806K.

4122 SANTA ROSA GP & BUILDING  
Retiring owner/doctor has est. GP in gorgeous 1,500 sq. ft. office w/4 fully equipped ops. & state-of-the-art equipment. Avg. GR $739K+, avg. overhead 60%. Selling building w/practice. Asking $438K building & $544K practice.
Basic Life Support Course, AEDs and Medical Emergency Kits

CDA Practice Support Staff

The Dental Board of California requires that licensees and unlicensed dental assistants complete a basic life support (BLS) course and maintain certification. No more than four units of continuing education credit for license renewal are allowed for a BLS course. The mandatory course requirement must be met by completion of either:

- An American Heart Association (AHA) or American Red Cross (ARC) BLS course.
- A BLS course taught by a provider approved by the AHA, ARC, American Dental Association’s Continuing Education Recognition Program (CERP) or the Academy of General Dentistry’s Program Approval for Continuing Education (PACE).

For the purpose of C.E. credit, a BLS course shall include all of the following:

- Instruction in both adult and pediatric CPR, including two-rescuer scenarios.
- Instruction in foreign-body airway obstruction.
- Instruction in relief of choking for adults, children and infants.
- Instruction in the use of automated external defibrillation with CPR.
- A live, in-person skills practice session, a skills test and a written examination.

Automated External Defibrillators

Dental practices in California are not required to have automated external defibrillators (AED), but if a practice has one then it must comply with state law governing ownership of these devices. Recent changes to the law became effective Jan. 1, 2016. California state law (Civil Code §1714.21) excludes from civil liability the individual or entity that acquires an AED for emergency use if the individual or entity has complied with specific requirements contained in Health & Safety Code §1797.196.

An individual or entity that acquires an AED shall do all of the following:

- Comply with all regulations governing the placement of an AED.
- Notify an agent of the local emergency medical services (EMS) agency of the existence, location and type of AED acquired.
- Ensure that the AED is

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maintained and tested according to the operation and maintenance guidelines set forth by the manufacturer.

- Ensure that the AED is tested at least biannually and after each use.
- Ensure that an inspection is made of all AEDs on the premises at least every 90 days for potential issues related to operability of the device, including a blinking light or other obvious defects that may suggest tampering or that another problem has arisen with the functionality of the AED.
- Maintain records of required maintenance and testing.

Building owners are not required to place AEDs in buildings. However, when an AED is placed in a building, the building owner must do all of the following:

- At least annually notify tenants of the location of the AED units and provide information about who tenants can contact if they want to voluntarily take AED or CPR training.
- At least annually offer a demonstration to at least one person associated with the building so that the person can be walked through how to use an AED properly in an emergency. The building owner may arrange for the demonstration or partner with a nonprofit organization to do so.
- Next to the AED, post instructions, in no less than 14-point type, on how to use the AED.

Any person who, in good faith and not for compensation, renders emergency care or treatment by the use of an AED at the scene of an emergency is not liable for any civil damages resulting from any acts or omissions in rendering the emergency care.

A medical director or other physician and surgeon are not required to be involved in the acquisition or placement of an AED. A manufacturer or retailer supplying an AED must provide all information governing the use, installation, operation, training and maintenance of the AED to the purchaser or owner of the AED.

The protections specified above do not apply in the case of personal injury or wrongful death that result from the gross negligence or willful or wanton misconduct of the person who renders emergency care or treatment by the use of an AED.

### Medical Emergency Kits

Dental practices are required by contract with dental benefits plans to have medical emergency kits. The contents of the kits may be dictated by the plan, although one of the largest plans requires the items recommended by the ADA Council on Scientific Affairs (*J Am Dent Assoc* March 2002). The council recommends a kit contain oxygen, blood pressure monitoring equipment, epinephrine, an antihistamine such as Benadryl, a quick source of glucose, nitroglycerin and a CPR pocket mask. The drugs should be checked periodically to ensure they have not expired. A pharmacy can fill a prescription for an epipen or nitroglycerin if the prescription indicates it is for the office medical emergency kit. Provide the pharmacist with a copy of the ADA article if necessary. For offices where general anesthesia is used, the Dental Board of California requires specific equipment (CCR Title 16 Section 1043.3 of the Dental Practice Act).

Cal/OSHA requires employers to maintain adequate first aid materials, approved by a consulting physician and readily available for employees. Cal/OSHA does not specify what first aid materials are adequate.

One method of obtaining physician approval of a medical emergency kit is to first write a description of the types of injuries that can be anticipated in a dental practice then list the contents of the kit. Provide the list to a physician and request a signature approval on the list.

Regulatory Compliance appears monthly and features resources about laws that impact dental practices. Visit cda.org/practicesupport for more than 600 practice support resources, including practice management, employment practices, dental benefits plans and regulatory compliance.
<table>
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<tr>
<th>BAY AREA</th>
<th>BAY AREA CONTINUED</th>
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<tbody>
<tr>
<td>AC-335 SAN FRANCISCO: Great Practice! 2100sf, 8ops in desirable location of SF. Call for Details: $475k</td>
<td>CC-567 ST. HELENA: Live and Practice in beautiful Wine Country, 5ops in 1842sf, single-story bldg. Price Reduced: $790k</td>
</tr>
<tr>
<td>AC-566 SAN FRANCISCO: Spectacular views of Washington Square. 3ops +2 add’l plumbed in 1400sf office $225k</td>
<td>CG-537 MARIN COUNTY: Rare Opportunity in upscale, highly desirable area. State of the art office. 2400 sf w/ 7 ops $1.1M</td>
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<tr>
<td>AC-585 SAN FRANCISCO: Near Union Sq., 3ops, 566sf. All reasonable offers considered! $270k for the Practice / $160k for the Goodwill</td>
<td>CG-583 SEBASTOPOL: Practice &amp; Real Estate. Seller Willing to consider all reasonable offer. Health Forces Sale PR $125/RE $750k</td>
</tr>
<tr>
<td>AG-564 SAN FRANCISCO: Over 25 yrs goodwill. Large 5,600+ sf w/ 9 ops near Land’s End $2.225M</td>
<td>DC-476 DUBLIN: Shared Facility. Great for Specialist - Endo, Pedo or Ortho. 1100 sf w/ 2 ops+1 add’l $125k</td>
</tr>
<tr>
<td>AG-576 SAN FRANCISCO: Part time practice w/ Amazing Growth Potential. Perfect for 1-3 DDS 4 ops 1,400 sf $550k</td>
<td>DN-497 PLEASANT HILL: Only: $330k</td>
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<tr>
<td>AN-514 SAN FRANCISCO Facility: Located in the bustling financial district! 1,007 sf w/4 ops. Reduced to $125k</td>
<td>DN-504 RICHMOND: Established Practice and Real Estate! 1,450 sf w/ 2 ops + 2 add’l $100k/RE $700k</td>
</tr>
<tr>
<td>AN-565 SAN FRANCISCO: This remarkable opportunity could be your “dream come true”! 2,067 sf w/ 3 ops $2.225M</td>
<td>DN-557 SALINAS: Only: $315k</td>
</tr>
<tr>
<td>BC-361 OAKLAND: Established for over 23+ years! 2,200 sf w/ 7 ops. Seller is retiring. Now Only: $330k</td>
<td>EC-525 SACRAMENTO: Great Location! Excellent Visibility! 1,500 sf w/ 3 ops, 10-15 new pts/mo $220k</td>
</tr>
<tr>
<td>BC-509 SAN LEANDRO: Facility Only, 800 sf, 3ops w/ xray in each op. Call for Details $60k</td>
<td>EC-531 GREATER SACRAMENTO: Practice and Real Estate for Sale! 1,750sf w/ 4ops + 1 add’l, 8npts/mo $800k</td>
</tr>
<tr>
<td>BC-520 HAYWARD Facility: Located in Downtown, 1500 sf, 4 equipped ops, X-Rays in 3 ops. Call for Details $65k</td>
<td>EN-464 ROCKLIN Facility: Don’t miss out on this remarkable opportunity! 2,150 sf w/ 4 ops. Now Only: $100k</td>
</tr>
<tr>
<td>BC-432 PITTSBURG: Own this family-oriented Practice! 1,640 sf w/ 6 ops. Seller is Retiring $350k</td>
<td>EG-479 FOLSOM: History is alive here with tributes to the past! 1,600 sf w/ 3 ops, $225k</td>
</tr>
<tr>
<td>BC-549 LAMORINDA AREA Facility: Excellent Location! Highly Visible, 900sf w/ 3ops +1 plumbed add’l. Reduced $75k</td>
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NORTHERN CALIFORNIA CONTINUED

EN-484 FOLSOM Facility: Fantastic Turn-Key Opportunity! Come live, practice and grow here! 1,934 sf w/ 4 Ops. Now Only $125k
EG-556 SACRAMENTO: Near CSUS Campus. Long-term 2nd generation office. 935 sf w/ 4 ops $389k
EN-534 ROSEVILLE Facility: Location, Location, Location! Turn-key... just needs you! 2,000 sf w/4 ops. $45k
EG-560 CARMICHAEL: Focusing on the philosophy of treating patients as family! 1,200 sf w/ 3 ops + 1 add’l. $130k
EN-558 DAVIS: Designed for maximum office efficiency and patient flow! 1,487 sf w/4 ops + 1 add’l. $650k
EN-573 SACRAMENTO: The goal and focus of this practice is to provide excellent service! 1,075 sf w/ 2 ops. $93.1k
EG-579 ROCKLIN Perio/Gen: Attractive, well-appointed practice in prestigious Whitney Oaks area. 1,600 sf w/3 op + 1 add’l. $325k
FC-415 FT. BRAGG: Excellent Practice! Dr. avgs 18+ pts/day & 20+ npts/mo, 1,800 sf w/ 5 ops + 1 hyg. Op $425k
FC-489 CLEARLAKE: Located on “4-Corners” of Hwy 53, 4ops in shared 3600sf facility. $470k / 50% interest in RE Also Available
FN-527 TRINITY COUNTY: Be the only dentist in town! “Pride Institute” designed! 2350sf w/ 5 ops +1 add’l. $250k
GC-472 ORLAND: Live & Practice in charming small town community. 1,000 sf w/2 ops. Seller Retiring. $160k
GG-386 REDDING: Amazing Practice. Lease or Buy Real Estate! 2,860 sf w/4 ops. Plumbed for 2 add’l! ONLY $260k
GG-453 CHICO: 5,000 sf w/ 7 ops Perfect for 1 or more DDS $325k
GG-454 PARADISE: “2,550 sf w/ 9 ops. 40 yrs goodwill! Amazing Opportunity!” $525k
GN-244 OROVILLE: Must See! Gorgeous, Spacious. 2,500 sf w/5 ops! Collections over $450k in 2013. Only $315k
GN-399 REDDING: Loyal patient base and relaxed workweek schedule. 1,440 sf w/3 ops. $150k
GN-507 CHICO: It just doesn’t get any better than this! 3,000 sf w/7ops. Practice $535k Real Estate $750k
GN-546 CHICO AREA: Office is well-known for offering quality dentistry with sedation. 2,600sf w/4 ops. $350k
HC-461 SONORA: In the beautiful Sierra Foothills, 4ops, 1350sf, free standing bldg. Practice $700k & RE Also Available!
HN-213 ALTURAS: This well managed practice continues to have consistent revenues! 2,200 sf w/ 3 ops + 1 add’l. $115k
HN-280 NO EAST CA: Only Practice in Town 900 sf w/ 2 ops REDUCED! ONLY $60k
HN-539 Central Sierra/Tuolumne Co: The perfect Merger Op in a rural Sierra Community! 2,000 sf w/ 5 ops. $175k
IC-468 SAN JOAQUIN VLY: High-End Restore Practice! 6 ops in 2500+ sf ofce. $425k
IC-572 MODESTO: In desirable Dental/Medical Professional building of town, 3ops in 1300sf ofce. $160k
IN-474 STOCKTON: Too good to be true? Absolutely not! 1,600 sf w/ 3 ops. $95k
IN-506 TURLOCK: Practice in the heart of the Central Valley! 2,000 sf w/5 ops + 1 add’l. $425k
IN-512 MERced: This immaculate practice is an absolute jewel! 1,200 sf w/4ops + 1 add’l. Now Only: $110k
IN-554 TURLOCK: A small town feel but with “big city” amenities! 1,900 sf w/ Sops. $795k
JC-541 FRESNO Facility: 1,210 square feet and consists of 2 fully equipped ops and plumbed for add’l op Call for Details!
JG-491 FRESNO: Well-established. 40-50 new Pt/mo. 1,452 sf w/ 4 fully equipped ops. REDUCED! $395k
IN-551 COALINGA AREA: Serving this community of working families! Paperless Practice. 1,200 sf w/ 3 ops. REDUCED! $395k!

CENTRAL VALLEY

BC-544 ALAMEDA COUNTY Pedo: 1,056sf w/ 4 chairs in growing, revitalized community, Seller Retiring $225k
BG-517 NORTH EAST BAY Endo: 2,750 sf w/ 8 ops! Strong Practice! $500k
CC-346 SO MARIN CO Perio: Beautiful 1,142 sf w/ 3 ops. No reasonable offer will be refused! Reduced $150k
CG-424 NAPA Prosth: Office has Digital X-ray & NEW 3D Imaging Unit! Ready for Experienced, high-end Prosthodontist! On track to collect just under $1m $690k
DC-459 SF PENINSULA Perio: 50% Partnership Buy In! Call for Details! $600k
EG-579 ROCKLIN Perio/Gen: Attractive, well-appointed practice in the prestigious Whitney Oaks area. 1,600 sf w/3 op + 1 add’l. $325k
FN-536 LAKE COUNTY Pedo: Focusing on Prevent dental problems before they begin! 1,750 sf w/ 3ops. Now Only: $225k!
JG-543 CENTRAL VALLEY Ortho: 1,650 sf w/ 5 chair bays & plumbed for 2 add’l, Strong Refs & Satisfied Pts Base $180k
JC-540 FRESNO Sleep Apnea: Motivated Seller retiring! Step right in and make yours! Call for Details!

SPECIALTY PRACTICES

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A look into the latest dental and general technology on the market

Tech Trends

Gboard (Google Inc., Free)

iPhone users know the drill — when texting a friend or colleague about the location of a meeting place, users leave their messaging app for another app to search and copy location information only to return to their messaging app to paste and send. This process and other similar ones are repeated constantly throughout the day. Google Gboard is a third-party, add-on keyboard available for iPhone that brings the powerful Google search engine and commonly used iPhone features together in one simple interface, making repetitive workflows a thing of the past. Gboard is, first and foremost, a swipe gesture keyboard. Users swipe the words they want to spell out by connecting one letter of a word to another without lifting their finger from the screen and Gboard predictively inputs their word with amazing accuracy. Gboard also has emoji and other word suggestions while users are typing in case they were looking for alternatives. The real distinction of this keyboard comes in its integration with search. Users can tap on the Google button directly from the keyboard and a search field appears without ever leaving the app. Search results appear as cards within the keyboard. Users can share the information to their app by simply tapping on the card itself. Gboard knows what kind of information is contained in the cards, such as locations or websites, and shares pertinent information only, which is perfect for texting users. Users can search for images and animated GIFs in the same manner, which further extends the usefulness of this keyboard.

— Hubert Chan, DDS

More Than Half of U.S. Gets News From Social Media

Well above half of U.S. adults get their news on social media, according to a report by the Pew Research Center and John S. and James L. Knight Foundation. According to the study, 62 percent of adults get their news on social media, up from 49 percent in 2012. For the study, researchers completed a survey of 4,654 people. Reddit, not Facebook, is the leading source of news on social media. In fact, 70 percent of Reddit users get news on the platform, compared to 66 percent of Facebook users. In third is Twitter, where 59 percent of its users get their news there. The study also found that 64 percent of the participants get news on just one site, 26 percent get news on two sites and 10 percent get news on three or more sites.

— Blake Ellington, Tech Trends editor

Kids Getting Their First Smart Phone at 10 Years Old

Kids Getting Their First Smart Phone at 10 Years Old

Remember when getting your first bike was the landmark moment of your childhood? Well, that may have been replaced with a smartphone. The average age a child gets his or her first smartphone is 10.3 years old, according to a study conducted by Influence Central. While that may seem shocking to some, this one may get you even more: 31 percent of parents surveyed say their kids have sent them a text while in the same home together. Kids are also using tablets more, as 55 percent said tablets are a kids’ device of choice on car rides.

— Blake Ellington, Tech Trends editor

Longhand Note-Taking Better for Learning Than Laptop Note-Taking

Longhand Note-Taking Better for Learning Than Laptop Note-Taking

The digital landscape has largely replaced many of the paper versions of our world, but a new series of studies has found that there is some benefit to sticking with a pen and paper over a laptop. Researchers at Princeton University and the University of California, Los Angeles, found that students who take notes on laptops performed worse on conceptual questions than those who took longhand notes. Specifically, it was found that those who take notes on a laptop are more geared toward transcribing a lecture rather than processing information and paraphrasing with their own thoughts.

— Blake Ellington, Tech Trends editor

Would you like to write about technology?

Dentists interested in contributing to this section should contact Tech Trends Editor Blake Ellington at blake.ellington@cda.org.
What will you discover in San Francisco?

Innovation. Explore new products and services from hundreds of companies, and try them yourself at CDA Presents. Get a first look at new tech and save big with convention-exclusive discounts.
No one wants to leave the dentist’s office with a gritty film masking their teeth. That’s why we developed Enamelast with an exclusive formula that provides sustained fluoride release and leaves the teeth feeling natural and smooth.

Everything you want in a fluoride varnish. *At last!*