OF THE CALIFORNIA DENTAL ASSOCIATION

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DEPARTMENTS

FEATURES

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Thank You to the *Journal* Reviewers

Authors have their names on their articles. Contributing editors, staff members, and outside vendors have their names in the masthead. But there are more people involved in putting out the *Journal* than those whose names are printed in each issue. There are also the professionals who formally review manuscripts and offer their recommendations. Below is a list of the people whose reward comes in the form of a thank you letter and a listing here. In addition, there are many others who have provided information counsel to the *Journal*. It is impossible to list them all. The *Journal* extends its thanks to the following people and everyone else who assists us in our endeavor.

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Happy Holidays

ALAN L. FELSENFELD, DDS

What happens during the rest of the year to the needy people who receive the benefits of our munificence?

Happy Thanksgiving, Christmas, Hanukkah, Kwanzaa, and whatever else you may celebrate. Now that the holidays are over, one can pause to think about the season and our feelings about others and ourselves. That time of year generally brings out the selflessness in us. There were presents for our families and loved ones, cards for friends and acquaintances, and merriment in our business and personal lives. We tend to be more charitable toward the needy. A few dollars at Salvation Army collection posts, a contribution to our place of worship, feeding the needy at a nearby shelter, or food donations to charitable organizations are all part of our spirit for the season. We feel worthy for what we do and what we give. This is a good thing.

The holiday season and these good feelings last for six to eight weeks each year, not counting the merchandising campaigns that start earlier and earlier. But what happens during the rest of the year to the needy people who receive the benefits of our munificence?

A similar feeling of self-actualization occurs within many of us as we provide dental services to the needy in foreign countries on missions with organizations who are committed to these laudable tasks. Many of our students spend their school vacations away from the United States providing care to developing nations' underserved populations under the supervision of our colleagues. It is a wonderful experience traveling to another culture and helping those who do not have the wherewithal to receive medical or dental care. Thousands of indigent children and adults benefit every year from these programs. Costs of travel and lodging generally are borne by the participants who not only pay their own way but also take time out of their practices to participate.

But what of people in our own communities who live in a state of health care not unlike that of our neighboring countries? What programs allow them to have the benefit of our ability to provide superb health care? Certainly many of these individuals are qualified for Denti-Cal or Healthy Families as their safety net providers. This population may elect to participate or not, or they may or may not have access to care in their neighborhoods. With those programs, at least there is some possibility for health care.

Unfortunately, there is a large group of people who are the working poor, or those who do not have sufficient resources to pursue dental care; not for lack of access or lack of desire but for lack of sufficient resources to budget this care.

California has several programs that could benefit this group of patients as well as allow our members to give back to their own communities. These programs do not require you to spend significant sums of money to travel to distant regions nor spend time away from your practice.

One of the easiest means to provide for those that need and cannot otherwise receive dental treatment is through a CDA-sponsored program called Donated Dental Services. This national program serves elderly, disabled, and medically compromised individuals who are unable to afford their own dental treatment. It is simple and rewarding to participate. Make one telephone call to CDA and you can sign up to participate. The only requirement is to provide dental care to one or two patients a year in your office and with your treatment plan at no cost to the patient.

You control the patient flow and the treatment. It takes minimal effort; however, the returns are maximum to you (and the patients) in helping those less fortunate who have needs but a limited ability to receive care. Since the Donated Dental Services program began in California in 1995, 612 volunteer dentists have generously provided what is estimated to be $2,660,724 worth of services for 1,204 disabled or elderly individuals. In addition, at the end of the 2006-2007 fiscal year, there was an increase in services rendered with 156 individuals receiving $416,635 worth of donated dental treatment, which indicates the generosity of the volunteers and the often involved nature of the benefit provided.

Having participated in this program, it is easy to understand the pride one feels in doing something good for someone who has unmet needs. It has minimal cost and does not interrupt our daily routines. The individuals who receive the benefit of our expertise have a genuine need and are appreciative of what we do for them. According to the Academy of General Dentistry, American dentists have provided more than

$116 million worth of free dental care to more than 77,000 individuals through this program.[1] If each of us in California annually treated two patients within this or similar programs, we could help more than 60,000 people in one year. Think about that, and then think about how good we can feel about ourselves this year when the holiday season is again upon us. ■■■■

REFERENCE

1. Letter to the Editor, *New York Times*, Academy of General Dentistry, Oct. 15, 2007.

Address comments, letters, and questions to the editor at alan.felsenfeld@cda.org.

Journal's Impartiality Questioned

We are concerned about the reputation, integrity, and impartiality of the *Journal of the California Dental Association* due to the content and advertising in the October 2007 issue. First, dedicating all the scientific articles to the Caries Management By Risk Assessment, CAMBRA, topic at the same time as the CDA House of Delegates debates adoption of the main principles of CAMBRA places the *Journal* in the potential position of CAMBRA advocate. This dilemma was realized when Mr. Jon Roth, executive director of the CDA Foundation, argued that the House of Delegates should adopt the CAMBRA principles since the *Journal* had already agreed to publish the principles.

Of greater concern was the prominent placement of the two-page centerfold CariFree ad promoting "The Complete CAMBRA Solution" in the middle of a CDA article on CAMBRA. This ad, for a product never before marketed in the *Journal*, easily leaves the impression that the selection of CAMBRA as a topic was financially motivated.

To maintain the reputation, integrity, and impartiality of the official publication of the California Dental Association, the *Journal* must more carefully monitor the choice and timing of topics and articles as well as the placement and content of ads within the *Journal*. Avoiding even the appearance of a conflict of interest needs to be the highest priority of the *Journal of the California Dental Association*.

JASON W. PAIR, DDS
GARY HERMAN, DDS
GEORGE MARANON, DDS
MARTIN C. COURTNEY, DDS

This ad, for a product never before marketed in the *Journal*, easily leaves the impression that the selection of CAMBRA as a topic was financially motivated.

The Author Responds: Electropolishing Needs Further Study

I am writing this rebuttal to address the Letter to the Editor titled "The *Journal* Can Do Better" (December 2007 issue) with regard to my article your *Journal* published in its September issue titled, "Simplifying Endodontics With EndoSequence Rotary Instrumentation."

I would direct readers to the October issue of the *Journal of Endodontics* and the Anderson et al. article titled "Fracture Resistance of Electropolished Rotary Nickel-Titanium Endodontic Instruments."

This paper studied cyclic flexural fatigue and torsional strength comparing EndoWave, ProFile and RaCe rotary NiTi files. The authors stated in the article, "Overall, electropolished instruments performed significantly better than nonelectropolished instruments in cyclic fatigue testing and, to a lesser extent, in static torsional loading." Concluding, "Electropolishing may have beneficial effects in prolonging the fatigue life of rotary NiTi endodontic instruments. The benefits of electropolishing are likely to be caused by a reduction in surface irregularities that serve as points for stress concentration and crack initiation."[1]

This appears to be supported by a 2006 article by Tripi et al., which concluded in a study comparing fatigue resistance of rotary nickel titanium endodontic instruments when looking at ProFile, RaCe, Hero and Mtwo that, "instrument design often proves to be an important factor in the fatigue resistance of NiTi rotary instruments" and "In RaCe instruments the electropolishing surface treatment increases the fracture fatigue resistance."[2]

I think we can all agree further study needs to be made in electropolishing before we conclude if this offers positive or no clinical benefits to endodontic rotary NiTi files.

GREGORI M. KURTZMAN, DDS

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1. Anderson ME, Price JW, Parashos P, Fracture resistance of electropolished rotary nickel-titanium endodontic instruments. *J Endod* 33(10):1212-6, October 2007.
2. Tripi TR, Bonaccorso A, Condorelli GG, Cyclic fatigue of different nickel-titanium endodontic rotary instruments. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102(4):e106-14, October 2006; E-pub July 14, 2006.

Matt Mullin

Resistant Bacterium Cases on the Rise

BY PATTY REYES, CDE

What once was common in health care surroundings and confined settings, such as prisons, methicillin-resistant *staphylococcus aureus* is now in the community. So, something as innocuous as a handshake, a brief high-five, or other skin-on-skin contact could be life-threatening, or at the very least, result in chronic compromised health.

The recent outbreaks in various parts of the country, including those that struck two children in California and caused the deaths of two teenagers on the East Coast late last year, prompted some dentists to contact the American Dental Association, who in turn encouraged them to contact the Centers for Disease Control and Prevention.

"This is a significant public health problem," said Scott K. Fridkin, a medical epidemiologist at the CDC, in a newspaper interview. "We should be very worried." And rightfully so. MRSA claimed

CONTINUES ON 13

Straumann Launches New Generation Bone Level Implant

Straumann recently announced the launch of its new generation Bone Level Implant. It comes in three diameters and four lengths and is suitable for all dental implant indications. There is a full matching prosthetic portfolio comprising 125 components, and a CAD/CAM custom abutment service in titanium and ceramic. The new implant line extension will be available initially in most parts of Europe, North America, Australia, and New Zealand, and the rest of the world in later this year. For additional information, go to www.straumann.com.

Venom Tech: New Adhesive Derived From Snakes Useful in Oral Surgery

A study in the October issue of the *Journal of Periodontology* found that an adhesive made from an enzyme found in snake venom was a more effective and beneficial adhesive when used to close surgical incisions than traditional sutures.

The study, "Fibrin Adhesive Derived From Snake Venom in Periodontal Surgery," by Mônica D.S. Barbosa, PhD; Sebastião Luis A. Gregh; and Euloir Passanezi explores a new fibrin adhesive made of buffalo plasma-derived fibrinogen and a thrombin-like enzyme obtained from snake venom and evaluates its applicability in periodontal surgery. Free gingival grafts that were sutured (control group) were compared to others immobilized through the use of the adhesive (experimental group), according to the abstract.

The study followed 15 patients during the healing process after a gingival graft. When the adhesive derived from snake venom was used, those patients had faster recovery and better results than those treated with traditional sutures.

"This unique type of adhesive may stimulate faster tissue repair. It is a more natural form of adhesive in comparison to traditional sutures used after surgery," explained study author Barbosa of the Bauru Dental School at the University of Sao Paulo. "More studies are needed to fully evaluate the effectiveness of this alternative."

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Caries in China: Fluoride Toothpaste to the Rescue

According to the latest figures from the Chinese Stomalogical Association, dental caries in China remains a pressing issue with the caries rate being 77 percent for deciduous teeth and up to 65 percent for permanent teeth.

While there has been significant improvement over the past decade, dental caries is still one of the most prevalent oral diseases in China, said Luan Wenmin, a professor, and vice president of CSA.

Working to address this issue, more than 70 experts in stomatology gathered last fall for a conference on "Oral Health through Fluoride for China and Southeast Asia" in Beijing, China. The conference was jointly organized by the World Health Organization, the FDI World Dental Federation, the International Association for Dental Research, and CSA.

The stomatological experts confirmed in a final conference statement that fluoride toothpaste remains the most widespread and significant form of prevention of and protection against tooth decay used worldwide. It is also the most rigorously evaluated vehicle for fluoride use.

Prior to the Beijing oral health conference, the World Health Organization adopted a resolution on oral health, which urged the establishment of national plans for the use of fluoride based on appropriate programs for automatic administration through drinking water, salt or milk, or topical use, such as affordable toothpaste, said Ramon J. Baez, DDS, and a WHO representative. "We hope that this conference will invigorate those in China and Southeast Asia to move the agenda item forward."

FDI World Dental Federation President Michèle Aerden, DDS, said the implementation of affordable access and appropriate exposure to fluoride has been successful in many parts of the world.

"It's important for dental team members to adapt their workplace and tasks in order to work as safely and as comfortably as is possible."

MARK S. RITZ, DDS, MAGD

ADA-OSHA Alliance Posts New Ergonomics Resources

New resources about avoiding injuries now are available to dentists and dental office staff, thanks to the ADA's collaboration with OSHA to help dentistry voluntarily addresses ergonomic issues.

"It's important for dental team members to adapt their workplace and tasks in order to work as safely and as comfortably as is possible," said Mark S. Ritz, DDS, MAGD, a member of the Council on Dental Practice and chair of its ergonomic subcommittee. "This means they should have access to knowledge about ergonomics and how it can be applied to the dental office."

A tip sheet about hand pain and how dentists can adapt their offices and habits to lessen stress on their hands, as well as an article about musculoskeletal disorders in dental hygiene, now are posted on ADA.org.

The ADA-OSHA Alliance, originally signed in April 2004, established a means of collaborating on ergonomics matters that both the American Dental Association and the U.S. Occupational Safety and Health Administration view as important to the health and well-being of dentists and the dental team.

A team representing both OSHA and the ADA meets quarterly to learn the best ways to reach joint goals in communicating information about workplace safety and health.

Information about ergonomics in dentistry, as well as the hand pain tip sheet and accompanying article on musculoskeletal disorders, is available online at www.ada.org/prof/prac/wellness/ergonomics.asp#alliance. Additional information is available on OSHA's Web site, www.osha.gov.

UPCOMING MEETINGS

2008	
May 1-4	CDA Spring Scientific Session, Anaheim, 800-CDA-SMILE (232-7645), cda.org.
Sept. 12-14	CDA Fall Scientific Session, San Francisco, 800-CDA-SMILE (232-7645), cda.org.
Oct. 16-19	American Dental Association 149th Annual Session, San Antonio, Texas, ada.org.
2009	
May 14-17	CDA Spring Scientific Session, Anaheim, 800-CDA-SMILE (232-7645), cda.org.
Sept. 11-13	CDA Fall Scientific Session, San Francisco, 800-CDA-SMILE (232-7645), cda.org.
Oct. 1-4	American Dental Association 150th Annual Session, Honolulu, Hawaii, ada.org.

To have an event included on this list of nonprofit association continuing education meetings, please send the information to Upcoming Meetings, CDA Journal, 1201 K St., 16th Floor, Sacramento, CA 95814 or fax the information to 916-554-5962.

BACTERIUM, CONTINUED FROM 11

more deaths than AIDS in 2005, according to CDC stats. In that same year in the United States, there were 94,000 infections and 19,000 people died from MRSA.

While staph infections are common in the population, according to the National Institute for Occupational Safety and Health, 1 percent carry the MRSA bacteria. Severe complications can develop when colonization leads to infection. When the bacteria is present and spread by casual contact, minor skin infections, like an abscess, can turn into major health complications including necrotizing abscesses that consume tissue.

It is thought this strain of bacteria emerged due to the overuse of antibiotics and the introduction of a vaccine that was developed to protect against the infection.

Draining and lancing sores, and the application of other antibiotics are ways to treat the infection. However, the microbe can develop in the lungs leading to pneumonia, for example, or affect vital organs, bone, and bloodstream leading to other serious and potentially fatal complications. There is also the threat of getting reinfected. Those with weakened immune systems, including the elderly and very young children, are most susceptible.

According to the Nov. 2, 2007, issue of *Cal-OSHA Reporter*, several preventive measures can be taken easily in the workplace, as well as personally, to guard against MRSA infection. Among them:

- Wash hands frequently with warm water and soap,
- Keep wounds covered and clean with dry bandages,
- Emphasize worker health and safety on the job,
- Do not use others' personal effects such as uniforms, protective equipment, towels, washcloths, razors or clothing,
- Keep an ample supply of hygiene products, and
- Be diligent with cleanliness in the workplace; ensure contaminated surfaces and equipment are adequately sanitized.

Massachusetts-based Institute for Health Care Improvement encourages doctors to limit prophylactic use of antibiotics but it can be a hard sell, said Debby Rogers, vice president for the California Hospital Association, in the article in the *Cal-OSHA Reporter*. While many physicians are receptive to the message, some

Matt Mullin

of their counterparts prefer to adhere to their practice and training, she added.

"This really is a societal change, said Rogers in the interview. "There's a lot of education that needs to be done."

In the meantime, researchers are working to develop antibiotics to treat MRSA. One company has created a test that can provide results in one hour. Typically, it takes 24 hours to get the results. According to the article in *Cal-OSHA Reporter*, with the new test, patients can start their treatment sooner "with medications that are still effective against the virulent staph."

The ADA and the CDC partnered to develop the CDC's infection control recommendations for dentistry. These recommendations were updated five years ago. The guidelines are available online at www.ada.org/prof/resources/topics/cdc/index.asp#guidelines.

Those with questions about MRSA are encouraged to consult the CDC's Web site, www.cdc.gov/ncidod/dhqp/ar_mrsa.html.

Capsaicin is capable of opening pores found only on the cell membrane of pain-sensing nerve cells.

A Hot Approach to Anesthesia

Capsaicin, the chemical that gives chili peppers their kick, chased with a local anesthetic, could be an improved way to treat pain in surgery, dentistry, and childbirth, according to a recent Harvard Medical School study.

Researchers reported in the journal *Nature* that a combination of capsaicin and QX-314, a derivative of the local anesthetic lidocaine, effectively silences pain-sensing nerve cells without disturbing other neurons that control motor function and other sensations. The innovative combination holds the potential to end pain in the dentist's chair without the temporary paralysis and numbness of current local anesthetics.

The two chemicals take advantage of a unique characteristic of pain-sensing neurons to block their activity without blocking signals from other nerve cells. Lidocaine interferes with electric currents in all nerve cells. But QX-314, by itself is unable to enter cell membranes to block their electrical activity.

That's where the hot chili chemical came into play.

Capsaicin is capable of opening pores found only on the cell membrane of pain-sensing nerve cells. With these pores opened by capsaicin, QX-314 can then enter the cell membrane and selectively block the activity of the pain-sensing neurons while leaving alone other nerve cells.

Researchers demonstrated the approach in rats and feel confident it will also work in people.

Updated Anesthesia Guidelines Ready for Use

After two years of work to update anesthesia guidelines, the American Dental Association has posted the new guidelines online at ada.org. The revised documents include "Guidelines for the Use of Sedation and General Anesthesia by Dentists"; "Guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students"; and the policy statement: "The Use of Sedation and General Anesthesia by Dentists."

According to the ADA, the changes bring the materials in line with other dental and medical organizations that had recently made significant changes to their documents.

"The revised anesthesia guidelines will provide practicing dentists with safe parameters for patient care at different levels of sedation," according to Frank Maggio, DDS, chair of ADA Council on Dental Education and Licensure. The council, along with its Committee on Anesthesiology, proposed the guideline changes, which were approved by the ADA House of Delegates last October.

The committee also held a "Proposed Sedation and Anesthesia Guidelines: Q&A" at the ADA annual session in San Francisco, and similar Q&A seminars at the annual meetings of the Academy of General Dentistry and American Association of Dental Examiners to explain the changes and address concerns.

The ADA is in the process of transmitting the guidelines to state boards of dentistry and other communities of interest. The guidelines are available immediately for use by the profession. The link to the guidelines is http://www.ada.org/prof/resources/positions/statements/index.asp#pain.

Honors

Patrick J. Ferrillo, Jr., DDS, San Francisco, dean of the Uiversity of the Pacific, Arthur A. Dugoni School of Dentistry, has been named president of the International Federation of Dental Educators and Associations and an honorary fellow of the Academy of Dentistry International.

Arthur A. Dugoni, DDS, MSD, Palo Alto, Calif., dean emeritus at University of the Pacific, Arthur A. Dugoni School of Dentistry, has received the Lifetime Achievement Award from the Pacific Coast Society of Orthodontists.

Patrick J. Ferrillo, Jr., DDS

Arthur A. Dugoni, DDS, MSD

CORRECTION

C.E. LISTING WEB SITE: The Web site address for online C.E. courses from the California Society of Pediatric Dentistry was incorrect in the December *Journal*. The correct address is http://cspd.org/oce.

Magnolia Bark Means No Bite From Bad Breath

Magnolia bark extract proved to be effective against bacteria responsible for bad breath, according to a recent report in the *Journal of Agricultural and Food Chemistry*. Researchers Minmin Tan and colleagues tested magnolia bark's germ-killing ability in a Wm. Wrigley Jr. Co. lab and concluded that it can be added to mints or gum for improved breath-freshening benefits.

Magnolia bark extract, a traditional Chinese medicine used to treat fever, headache and stress, has proven effective against germs that cause ulcers, and recent studies have shown it has low toxicity and few side effects.

In the study, magnolia bark extract and its two main components, magnolol and honokiol, were evaluated. In the lab, researchers found it highly effective against three types of oral microorganisms, killing 99.9 percent of bad breath bacteria within five minutes.

"Magnolia bark extract can inhibit bacteria responsible for producing hydrogen sulfide and methyl mercaptan and even a gram positive bacteria, *S. mutans* responsible for dental cavities," Michael Greenberg, Wrigley director, summarized on the American Chemical Society Web site.

In vivo tests on nine healthy Wrigley employees who chewed mints and gum containing the bark after lunch produced less dramatic but still potent effects.

The mints killed off more than 61 percent of the germs that cause bad breath within 30 minutes — comparable to some commercial mouthwashes. Mints without the extract were only 3.6 percent effective.

The gum didn't work as well, reducing oral bacteria by 43 percent within 40 minutes, compared with an 18 percent reduction in gum with no extract.

"Because bacteria is the major cause of breath odor, products containing effective germ-kill compounds will provide a long-lasting reduction of oral malodor," the researchers wrote.

However, the product is not expected in stores any time soon.

"It's a long way from scientific research to a commercializable product, and there are a lot of perils and pitfalls along the way," said Chris Perille, Wrigley spokesman.

"Magnolia bark extract can inhibit bacteria responsible for producing hydrogen sulfide and methyl mercaptan and even a gram positive bacteria, S. mutans responsible for dental cavities."

MICHAEL GREENBERG

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CELL TO CELL COMMUNICATION:

Implications for Oral Health, Pathogenesis, and Drug Therapies

BY JANYCE HAMILTON

Humans talk so much that strategic interruption is a required part of conversation. Animals vocalize or thump out their own language. Now it turns out that molecularly, communication between cells is not so different. As Alice in Wonderland said on her strangely plausible trip through a rabbit hole, it just gets "curiouser and curiouser."

AUTHOR

Janyce Hamilton is a freelance writer working out of Naperville, Ill. Her previous articles for the *Journal of the California Dental Association* include "Transmissible Spongiform Encephalopathies and Dental Transmission Risk Assessment," "Robots, Bionics, and Bioengineered Replacement Parts in Dentistry," "The Link Between Periodontal Disease and Systemic Diseases: State of the Evidence 2005," "Assessing 'Real Science': Poor Studies, Industry Taking Toll," and "Dental Implications of the Human Genome Project."

Sure cells respire, have a nucleus, and make waste as if little critters. But in recent years, a surprisingly personified trait has been cocking the heads of research scientists — it has been shown that a cell can *talk* to another cell. Better yet, the other cell hears and responds. Among the known cell types that communicate throughout their communities are bacterial, animal, and human.

This report talks about how scientists continue to understand what was once so mysterious: cell-cell communication. Also explained is exactly how cells communicate and how questions are raised about whether or not cells have a sentient or "knowing" quality. Examples of how these molecular communication processes are being studied by university and government oral craniofacial researchers are provided. Novel pharmacotherapeutic approaches to one day molecularly dissuade colonization of biofilms and periodontal pathogens are not far-fetched. Strange stuff is going on, too, including fooling cells into thinking nothing is wrong, while the clinically dead are revived, and trying to prevent "back talk" so communities of cells accept a newly implanted cell that will contain the first artificially created chromosome.

How it Was Discovered That Cells Talk

Bonnie Bassler, PhD, is an investigator with the Howard Hughes Medical Institute, Chevy Chase, Md., and professor of molecular biology, Princeton University, Princeton, N.J. She has been dubbed "The Bacteria Whisperer" for her entertaining and affectionate lectures about how bacterial cells talk.[1] She credits the Hawaiian bobtail squid as "ground zero" in which molecular detail of cell-cell communication was first comprehended.[2]

Explained Bassler, the 2-inch long squid lives buried in the sandy floor of the coastal Pacific shallow salt water by day and emerges at night. It's different than most sea creatures in that it has a glowing light organ filled with luminescent bacteria known as *Vibrio fischeri*. In the 1960s and '70s microbiologists K. Nealson and J.W. Hastings of Harvard University studied the properties of *V. fischeri* bacte-

ria. That team's first clue to bacterial cells communicating among each other was that if the bacteria were outside of the organ, they "knew it" and didn't glow; there had to be a dense number of them together first. The team theorized that a chemical molecule, which they dubbed an "autoinducer" (AI), was released so the cells got the message that they had amassed in sufficient numbers and so it was time to flip the gene switch on to light up.

Nealson and Hastings published a paper in 1973 that they had found the autoinduction phenomenon and, furthermore, that they isolated, characterized, and identified the signal molecule (autoinducer).

Another piece of the mystery came in during the 1980s when Dr. Mike Silverman and grad student Joanne Engebrecht, from the Agouron Institute, La Jolla, Calif., chopped up the glowing chromosome — the structural genetic enzymes for luminescence and the density sensing mechanism — from the *V. fischeri* bacterial cell, injected it into *Escherichia coli* to see if it also glowed on a piece of DNA. It did and that part was the AI, the density-sensing mechanism. Yet, more proof there was communicating going on was that when the squid was propelling itself around at night to hunt for food, it used biochemical sensors on its back to detect how much moon and starlight were hitting it. To counter-illuminate the light shining upon it through the water's surface, it would open its skin flap covering the light organ, and the *V. fischeri* cells needed to project the precise same amount of light from its bottom side, would communicate "the light has changed," and begin to amass. Now the amount of glow emitted from the bottom side matched that hitting the topside so that the squid didn't cast a shadow on the sandy seafloor. The Hawaiian bobtail squid propelled about, covertly evading predator detection while sneaking up on shrimp.

Knowing all of this, Bassler commenced upon new research and nailed things down. In another glowing sea creature, *Vibrio harveyi*, she identified not one but two signal circuitry systems and published the definitive proof of bacterial cell-cell talk and quorum sensing.

Quorum sensing. The "butterfly effect" goes like this: Will the flap of a butterfly's wings affect a tornado in Texas? Likewise, is there such an effect with cells? If one cell's receptors receive a signal at the cell surface, they dive through the cytoplasm and to the brain and genes, which translate and activate a response (even if it's a nonresponse). One little cell kicking out an inflammatory agent may not be perceived as arthritic knee pain, but cells make sure they "call all the butterflies in Texas" for the biggest effect on the tornado. Turns out that a bacterial cell knows, said Bassler, that one cell dribbling out toxins isn't going to amount to much kick, so the bacteria "wait" and count heads. They sense when they are alone versus together via "quorum sensing" AI molecules, and when they know there are enough of them together, then they all secrete their toxins at the same time (**FIGURE 1**).

Said Bassler, "Bacteria are using a rich chemical lexicon to coordinate population-wide behaviors and carry out tasks in groups that they could never manage if they simply acted as individuals."

BASSLER SAID CELLS
are not socially reclusive loners, but have a language for their own species, and are in effect multilingual for communicating between species.

How One Cell Talks to Another Cell

Bassler said cells are not socially reclusive loners, but have a language for their own species, and are in effect multilingual for communicating between species. They aren't psychic and they don't have "eyes," so within the dark confines of intercellular broth, they talk to tell each other when they are there and who is there. "They coordinate gene expression, which ultimately coordinates behavior so they can all act as one, as if multicellular organisms," she said.

Many questions remain that science still needs to work out. For example, how is information preserved, where is it stored in the cell circuitry? Physicists work on this too. Also, how do bacterial cells decode mixes of signals and know to tweak the functions of multiple genes given various messages simultaneously? What can impede, stop, or introduce noise-to-signal flow out of and in between cells — can the signals be eaten, tricked, eavesdropped, and tattle-taled upon?

Chemical signaling used to be thought a trait of only higher organisms, but it's not. With the talking talents of multicellular organisms, signaling makes cells function much like the functions of a human body, where our brains signal and chemically coordinate the whole population so things get done that couldn't get done alone, such as metabolism.

Therapy to treat oral pathogens one day might involve taking bacterial mutants that are of the same species found in a biofilm but have enhanced virulence and that can't "talk" or "hear." If they are injected into the oral cavity (or through a dentifrice or rinse) and dominate the nor-

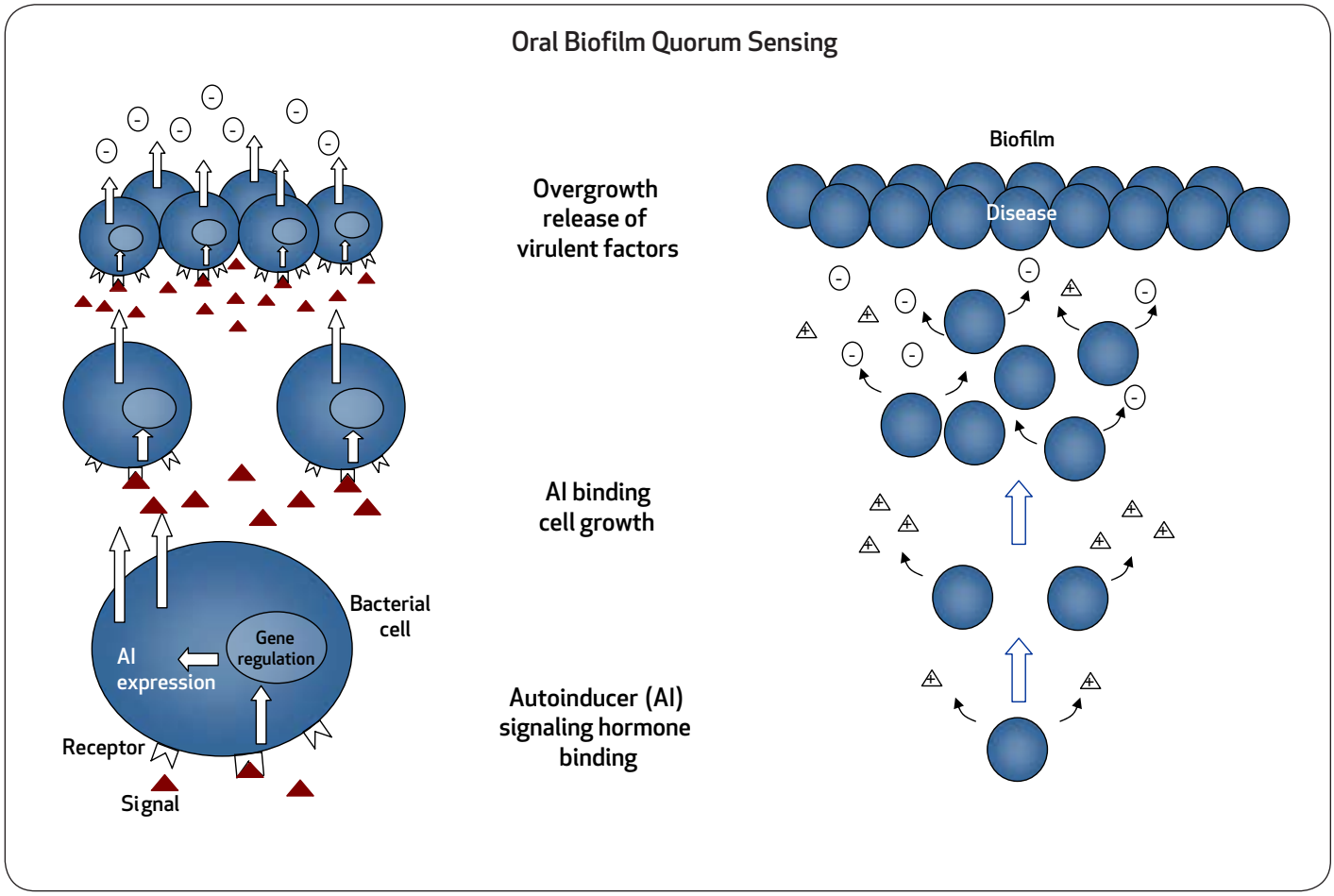


FIGURE 1. Oral biofilm quorum sensing. Quorum sensing — releasing and receiving chemical signaling molecules called autoinducers between bacterial cells — allows the cells to coordinate information to control gene expression as population density of cells increases or decreases. While no one has found or demonstrated the bacterial cell quorum-sensing system in oral biofilms yet; it is, however, suspected partly because monospecies, which do not alone form biofilm (in vitro) do so in vivo where there are multiple species. Since the precise signaling molecule has not been identified in oral biofilm production, it has been theorized that they are likely types of autoinducers "talking" and "hearing" each others' messages to coordinate population-wide gene expression. At low concentrations, bacterial cell growth remains low, as are the numbers of autoinducer molecules released. Growth rates of all climb steadily. When the cell density results in a threshold of sufficient autoinducer numbers ("+") released, the cells sense this and the behavior/functions of clusters of cells begin acting in sync, indicating gene expression is being coordinated. This may alter the release of autoinducers and begin the release of other factors (host-damaging virulence factors) ("-"). As the cell population reaches a critical threshold, a biofilm forms which can result in disease. *(Illustration courtesy of Robert Dorsam and Janyce Hamilton.)*

mal bacterial population — voila! Nothing gets done as planned as mutants rule — the good kind, the therapeutic "Franken cells" our molecular researchers created.

Is Cell-Cell Communication Chemical Mimicry of Talking and Listening, or in Fact Proof of 'Consciousness'?

In Dr. Seuss' book, *Horton Hears a Who!*, an elephant hears a speck of dust trying to get his attention. The tiny speck of dust is a miniature planet, upon which is a town called "Whoville" with microscopic residents called "Whos." Horton the elephant befriends them, and repeats "a person's a person, no matter how small" throughout the book. Well, all this "talking" going on between cells makes them sound personified as if sentient, "knowing" little Whos. Certainly, these terms explain a theory of "behavior" or action of a cell so that we can understand processes more easily. But is this just analogy in using these descriptors, or is there evidence that cells have "thoughts," or an intelligence that drives them to fulfill their functions and change course when conditions warrant?

Neurologists have long explained the coordinated interactions among nerve cells in directing muscle cell contraction (and other activities) as akin to little electrochemical "children" switching games on the playground. This theory is that a group of cells — operating as if pixels, switches, and or transistors — together make up the whole of conscious thought. But just because schools of fish zigzag in unison isn't to say each minnow doesn't have its own thought on the matter. Likewise, molecularly, some cognitive neurophysicist researchers are

questioning if each cell has "thought" too — that is, could each cell be recognizing and thereby decoding visuals?

Neural cell-cell cognition example. Neurons are the "building blocks of cognition" according to Itzhak Fried, a physicist and professor of neurosurgery and psychiatry and biobehavioral sciences at the University of California, Los Angeles, Medical Center. He also is director of the epilepsy surgery program and co-directs the seizure disorder center there as well as heads the cognitive neurophysiology laboratory and does studies through the Brain Research Institute. For a 2007 study published in prestigious *Journal of Cognitive Neuroscience*, Fried was part of a team of researchers reporting on their nine patients having brain depth electrodes inserted in order to evaluate their seizures for subsequent surgery.[3] While subjected to electrodes in their brains anyway, the team took advantage of the rare invasive opportunity to gauge single neural cell response (measured using local field potentials [LFP] while being exposed to images of objects, landmarks, animals or individuals — some chosen per patient preference). The reaction of each nerve cell monitored was recorded. For this study, as well as one a few years earlier by Fried and colleagues, it was found that a lone neural cell in effect "lit up" both when the subject looked at an image he or she recognized, and when he or she closed their eyes and recalled the image from memory. A single neuron's activity spikes selective to each category of visual stimuli and it is invariant to "different views of the same person."[4] The findings of these studies are among the small body of evidence that, at minimum, visually perceived image and imagining an image have the same shared neural circuits.

If preliminary investigations that have quantified amplitude and spiking measured in LFP continue to replicate findings and increase in number, it will be reckoning day. The neuroscientists pooh-poohing the concept of a single neural cell recognizing, say, the dog of its human host? Well, let's just say that those neuroscientists should keep working out their alternative explanations.

"Like a moving front of migrating geese, the cancerous cells descend into the connective tissue."

BRIAN L. SCHMIDT, DDS, MD, PHD

Pain and malignancy cell-cell talk example. How signals are used by a cell to respond to real pain versus phantom pain stimuli is interesting. Also, how is cell-cell communication relevant to cancer cells, tumor formations, and decisions to metastasize? How do cells know where to form secondary site tumors? Why are there clinical patterns that are reproducible — for example, certain cancers of the breast always spread to specific bones?

All good questions that researchers pick away at continuously.

Brian L. Schmidt, DDS, MD, PhD, oncology fellowship director, Department of Oral and Maxillofacial Surgery, University of California, San Francisco, and a federally funded oral cancer researcher, said cell communication and signaling is not an area in *oral cancer* research that has been studied as extensively as it has been in other more common cancers such as breast, prostate, and lung cancer. "Gene swapping, molecular vaccines, and cell communications are not something reported on widely in the dental journals because oral researchers have only begun looking at these questions during the last few years," he said.

Oral mucosal cells use an intricate form of communication to produce an intact epithelial barrier in the mouth. As the carcinogenesis process is initiated, cells change their cell-to-cell signaling and receptor communication. Once this change occurs, he said, the cancerous mucosal cells no longer cooperate to maintain a mucosal barrier.

"Soon an ulcer appears and the patient is in the dental office complaining of pain. The cancerous mucosal cells also change their communication with the deeper connective tissue cells. Like a moving front of migrating geese, the cancerous cells descend into the connective tissue," Schmidt explained, adding that once the epithelial mucosal cells invade beyond the basement membrane into connective tissue, the diagnosis of squamous cell carcinoma is made. Next, the carcinoma cells "talk" about how they can survive free of the main tumor. The cancerous cells identify the lymphatic system and metastasize to the neck.

Schmidt said not all oral cancers metastasize but there is currently no way of determining which ones will. His NCI-supported study is focused on identifying molecular markers in oral SCC that can be used to predict metastasis.

"Looking at quality of life studies, the head and neck cancer patient's pain is the highest across all cancers," Schmidt explained, because oral cancers produce

proteins that sensitize nerve fibers, lowering the pain threshold and leading to debilitating pain just to talk, eat, or drink. Schmidt's creation of the UCSF Oral Cancer Pain Questionnaire may be used in his NIDCR-supported study looking for ways to test experimental analgesics to directly target oral cancer pain.[5,6] Little doubt that he'll be reading the literature on studies of head, neck, and oral cancer pain signaling with interest as this is an emerging frontier of experimental analgesics.

Cell-Cell Communication in Healthy Versus Diseased States

The existing molecular study model in science had been "reductionism" — looking at one cell, and isolating and identifying smaller and smaller aspects of its independent traits. In recent years, however, that model has shifted to "holism" — seeing how one cell intermingles with others in health and pathological processes. These ideas are new to biology and medicine, so for dentistry it is not surprising there are few dental journal articles and books yet on cell-cell communication (confirmed by library staff at the American Dental Association in Chicago for this report). Instead, academic and government medicodental researchers, often with PhD and MD degrees, are thus far publishing their work first in medical and science journals. The pioneer author of overall cell behavior in the dental discipline is the former NIDCR director, and current University of Southern California School of Dentistry Dean Harold C. Slavkin, DDS (see sidebar).

"Cell talk (with scientists and clinicians listening) is a very intriguing topic in molecular biology as well as many human diseases and disorders. It is the cornerstone of modern dentistry and medicine and the cutting edge for pharmaceutical advances," he said.

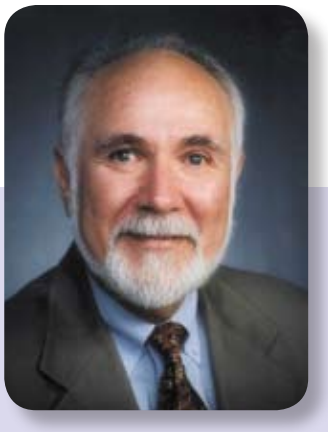
Even Slavkin is excited to know what he's suspected of several oral bacteria all along is now being proven — they communicate. Examples of bacteria using the universal lexicon of AI signaling are *Helicobacter pylori, Porphyromonas gingivalis*, and *Streptococcus mutans*.

(On this note, Bassler commented that AI "communication helps oral bacteria adhere to surfaces and take up iron … [and] is involved in the formation of mixed species biofilms. Bacteria without the ability to 'talk' with the (universal language) can form mono-species just fine, but they cannot form the mixed species biofilms that are present on teeth").

But Slavkin says cell-cell communication is not just about the formation of biofilm, this signaling directs the virulence levels of oral pathogens, the oral mucosal immune response, the inflammatory response of the periodontal tissues, and subsequent tissue destruction.

Interspecies bacterial cell talk and interaction is just now coming into its own with relatively new publications and databases, such as the *Journal of Molecular Signaling, Signal Transduction Knowledge Environment Database of Cell Signaling*, and the *Journal of Cell to Cell Communication and Signaling*, struggling less and less for manuscripts, contributors, and reviewers. In fact, an entire Fall 2007 issue of the mainstream magazine, *Science*, was devoted to cell signaling.[7]

"This is a new field, and not many people are working on it," admitted Wenyuan Shi, PhD, from his desk at the Section of Oral Biology, UCLA School of Dentistry, who is working on it. Shi is a coauthor with K.H. Kuramitsu; X. He; R. Lux; and M.H. Anderson from UCLA; State University of New York, Buffalo; and a biotech company called C3 Jian, of the article "Interspecies Inter-



Dental Pioneer's History of Cell-Cell Signal Research

"In the late 1960s, I was captivated by the problems related to cell-cell communication — How do cells recognize self and nonself, how do they 'choose' to assemble or not assemble into tissues/organs, and how do they sustain specific phenotypes? I chose the developing mammalian tooth organ as my model. I cultured rat teeth in artificial environments, such as the chick chorioallantoic membrane. I dissected these teeth, dissociated into cell isolates, and performed studies to define what, when, and how these cells reorganize to form teeth. Some 30 scientific papers and perhaps 15 years later, we learned that the signals for communication were often ions or small polypeptides termed 'growth factors' and the reception or receptors were specific to bind the growth factor ligand. We later learned that once the signal was bound to a specific receptor the process invoked an intracellular mechanism termed 'signal transduction' by which signals were propagated to specific regions of the genome and acted to regulate gene expressions including positive and negative controls. For me it began with my paper in *Nature* in 1967."

— HAROLD C. SLAVKIN, DDS

actions within the Oral Community" in the December 2007 issue of *Microbiology and Molecular Biology Reviews*. The article is an important publication by privately and NIH grant-funded researchers from dental schools collaborating with those from multidisciplinary departments of microbiology, molecular genetics, and immunology. Such "multidoctoral" collaboration is breaking down barriers between professions while unearthing fresh ground to understand how communities of bacterial cells talk and otherwise interact.

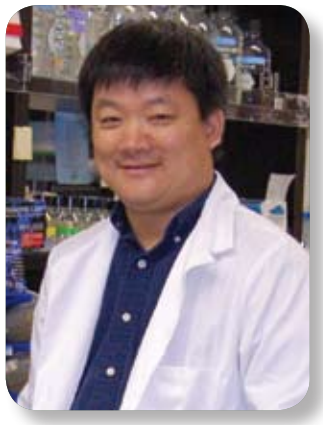
"Bacterial cells are not a bunch of things just sticking together. If you read the literature in detail, their 'brains' are truly functional. They actually are talking with extensive signal transduction," Shi said, adding "I think of them as a different species of animals but instead of on earth, in their own ecological micro-community."

The basis for ecology on earth involves extensive interactions between different organisms. The similar rules apply to different microorganisms in the oral cavity, Shi explained.

Same species cells speak one language, while other species of cell types speak another, he said. "So some cells might speak English, and some French, but there is also a universal language so different species can cross talk — something like hand signals," Shi explained of autoinducers. Interspecies conversing would be necessary for a complex oral biofilm of some 700 species to form. There's no denying that gunky plaque contains a high degree of organization. Emitted are very specific directions regarding who goes where so each cell "sits" akin to one another in a mosaic pattern, building into a grand scheme that either magically or with maestro-like intention confers adherence or growth advantage — you be the judge.

Whatever it is termed and whomever/whatever is at the helm, the behavior of the same cell differs in liquid suspension in vitro versus on a solid surface in vivo because, conceivably, they "sense" and communicate amongst themselves these distinctions regarding their surroundings. The positioning as biofilms aggregate is ordered not randomly distributed. Cells in effect know their group's size, they know who — cell-wise — is there around them and what they are all doing, and that is the only way it makes any sense that their behavior is purposeful. To create oral biofilm, they "select their neighbors" as if school students lining up by height in rows for a class photo. This shuffling and choosing of location can and does respond to change if, say, a mouthrinse disrupts the colony and repairing/rebuilding processes must commence all over again, or the football player doesn't brush his teeth all weekend so nutrients and metabolism are on overdrive. The community adapts and balances in the oral ecosystem by cell-cell communication and then action.

Extraordinary.



"I think of them as a different species of animals but instead of on earth, in their own ecological micro-community."

WENYUAN SHI, PHD

Proteomic and Other Pharmacotherapies Targeting Oral Pathogens That Involve Cell-Cell Communication

It is no surprise that Shi and his colleagues are viewing cell community-wide communication and behavioral interaction as the next frontier for pharmaceutical advances.

The old model is to use antibiotics to kill off all bacteria, which often creates conditions for the return of pathogenic bacteria with virulence. Meanwhile, scientists are beginning to wonder if ecology-based therapy could be applied to control oral microbial infections. A quorum sensing regulatory molecule known as autoinducer-2 (A-2) is needed for some interspecies bacterial cells to be able to form oral biofilms. A cell type that can inactivate A-2 in oral biofilm hasn't been identified. A gene known as luxS, if taken away in *S. mutans,* abolished its ability to produce a mutacin (a virulence factor), even at high cell density. There has been signaling observed between two dental plaque species in vitro, but the identity of the precise signaling molecule is not determined.

Work at the Los Alamos National Laboratory with its oral pathogens sequence database is interjecting that an "inducible repressor may be used as a suppressor" of a mutacin gene. Could *S. mutans* tinkering impact the quorum sensing system and bacteriocin production? Maybe Shi and colleagues will find out. Their published review looks at several areas that could have potential or are in development to be products, like mouthwash with gene modified bacteria, for dentistry:

- ***Selectively inhibiting adherence of pathogenic bacterial cells in oral colonies by producing antagonist bacteria that generate "bacteriocins" also called "mutacins," to inhibit the growth of certain other bacteria***. Examples are *S. mutans* to inhibit *S. sanguis*, and *S. salivarius* to

inhibit biofilm formation by *S. pyogenes;*

- ***Passive or active immunization with antibodies in a vaccine that may penetrate the matrix of oral biofilm to interfere with the normal properties of a functional pathogenic bacterial cell;***
- ***Bacterial cell replacement therapy — rinsing with mouthwash containing bacteria to overtake or knock down virulence capabilities of existing bacterial populations.*** It is now likely that genes are being naturally horizontally transferred between bacterial cells in dental plaque. Although not proven yet, this is highly plausible because *S. mutans* strains have diversely different genes, thus have likely swapped DNA from other *S. mutans* strains or maybe even other species to get what they need. Researchers can either inject more of a bacteria that has genes either naturally or therapeutically altered, which, as a source, will dilute the population from fully functioning pathologically. For example, a noncariogenic *S. mutans* strain (with bacteriocin against other more harmful *S. mutans* strains) could be introduced to grow abundantly and largely render the latter pathologically insignificant in number or function. In theory, this just might reduce colonization of the harmful *S. mutans* to protect enamel. This showed promise in animal studies and awaits approval for human studies.
- ***Probiotic approaches.*** Instead of killing problem bacteria, try aiding the helpful bacteria in the microbial balance. Bacterial supplementation is an example; in human diet probiotics, this includes eating yogurt to improve bacterial flora population for improved gut function.
- ***Interference (for example, messing with metabolization or transporting) with signaling mechanisms of one cell that would normally be received by another to tell it to grow or to stay sensitive to a harmful agents.*** For example, while it is

not yet proven in vivo for oral biofilm cells, in vitro it seems that *S. gordonii* can inactivate the competence sensing peptide of *S. mutans* to antagonize its quorum-sensing-dependent abilities, leaving them more vulnerable and sensitive to antimicrobials agents like histatins.

■ ***Targeted antimicrobial therapy via a novel specific targeted antimicrobial peptide (stamp) technology.*** This is something Shi's lab team is working on. This is to be the "smart bomb" of oral bacteriology therapy. He described it as instead of applying a broad-spectrum herbicide to the lawn that kills the dandelion and grass, applying a STAMP-type agent would kill the dandelion, spare the grass, and also encourage the grass to flourish and fill in the empty space. Said Shi, "We are working with companies to put our product on the market in a few years."

DNA separation techniques using PCR can look at a bacterial cell population profile in dental plaque. Genomic pharmacotherapeutic researchers can excise a band of DNA, clone it, and sequence it to determine which bacterial species it is. T-RFLP is helpful too, and goes well beyond the existing culturing and 16S reran analyses methods, for assessing the cell identities in communities of cells too, as dentistry remains at the biofilm composition discovery phase. This is why looking at cell-cell communication is a lofty high specificity pursuit and not yet priority — we don't even know all the cell types that are present in healthy versus disease oral aggregated bacterial communities and tissues yet.

But we're getting there.

First New Artificial Life Form on Earth Nearly Complete

A synthetic chromosome based on a DNA bacterium *Mycoplasma genitalium*, which reportedly has one-fifth of its genetic makeup removed and synthetically replaced — dubbed *Mycoplasma laboratorium* by the Nobel Laureate Hamilton Smith's team at the J. Craig Venter Institute, Rockville, Md., is slated to be finished during 2008, according to an institute representative contacted in late 2007. How will its signal be sensed? Will its receptors hear normally and respond appropriately? Will it replicate its synthesized DNA into new cells?

"We are now learning why cancer cells grow unrestrained and subvert surrounding healthy cells to satisfy their increasing need for nutrients and oxygen."

J. SILVIO GUTKIND, PHD

The fake chromosome is said to be "watermarked with inks" for easy microscopic identification. Once it is implanted into a live bacterial cell, things get interesting. It may or may not take control of the cell and in effect become a new life form. It may or may not normally metabolize and replicate itself, but the team hopes it will. These scientists already completed a genome transplant of one type of bacterium into the cell of another, an engineered change over from one to another cell species.

The new life form will depend for its ability to replicate itself and metabolize on the molecular machinery of the cell into which it has been injected, and in that sense it will not be a wholly synthetic life form. However, its DNA will be artificial, and it is said that the DNA that controls the cell is credited with being the building block of life.

The spontaneous issues that could erupt during molecular cell-cell communication and signaling to be encountered when it is inserted into a bacterial cell (knowing there will be community-wide sensing of the newly engineered *M. laboratorium*, whether or not it will be distinctively differentiated by the nonsynthetic cells in the Petri dish) will be better than watching the Superbowl. Like the old days of testing a farm animal organ transplant, gene geeks are gambling on whether the au natural bacteria will be fooled or not into being controlled by artificial DNA bacteria. If they are, the team has plans to engineer, over time, simple artificial-cellular hybrid life and organisms.

And so it goes.

Oral cancer cell-cell communication example. While Schmidt of UCSF toils away at finding molecular markers for cancer metastasis and studies cancer pain, the cell-cell communications between a healthy and a cancer cell are being scrutinized by others. In few other diseases are there so many action verbs that connotate a brain at work, a malignant cell *manipulates* the signaling network, *attracts* chemicals, *instructs* cells, and *alerts* immune response to drop their weapons. Even the phrase "crosstalk" is used in highly technical papers when it comes to cell-cell signaling in cancer.

Scientists at the Oral and Pharyngeal Cancer Branch of the National Institute of Dental and Craniofacial Research, National Institutes of Health, Bethesda, Md., are no exception. A 2007 review, "G-Protein-

Coupled Receptors and Cancer," by Robert Dorsam, PhD, and J. Silvio Gutkind, PhD, of the NIDCR, is being widely disseminated as part of the body of knowledge that is used to piece together the mysteries of cell signaling and circuitry, including whether interfering with these signals may help prevent and treat cancer.[8]

What the coauthors of the paper learned is that G-protein-coupled receptors, GPCRs, make up the bulk of cell surface molecules whose role is signal transmission (send and receive messages). Recently, Dorsam and Gutkind highlighted that malignant cells, which take command of their environment to promote cancer cell proliferation and migration, can "hijack" the functions of GPCRs, thereby disrupting normal inter- and intracellular communications. The havoc wreaked by the malignant hijacker starts with its chemical invisibility, it "flies under the radar," remaining undetected by the immune system. The malignant invader simulates GPCR function to increase the supply of blood, oxygen, and nutrients, too. It then sends trick signals to naïve surrounding tissue telling it to loosen up their tight structure and make the blood vessels permeable so the malignant cells can stream inside and proliferate. Since the malignant cell has now taken over the virtual "air traffic control system" of multiple cells' GPCRs, it sends signals and even a second round of messengers acting on GPCRs in distant organs telling them to make a safe haven for migrating invading cancer cells that would be arriving soon on a lymphatic current.

Like the wartime strategy of jamming the signal of the enemy's radio-broadcasted propaganda, Gutkind and Dorsam are part of a task force exploring the effects of running interference on GPCRs and their downstream targets. Changes in the interactions and cancer progression would be informative.

"Because of increasing knowledge about communication within and among cells, we'll be able to treat malignant processes in a new way."

ROBERT DORSAM, PHD

The team also reported on the findings of researchers detailing how chronic inflammation of tissues can increase metastasis likelihood by releasing chemicals to stimulate the conditioning of "pre-metastatic niches" in lymph nodes and secondary organs favorable for tumor colonization.[9,10] While those studies reported on colon and lung inflammation-cancer research, no one knows yet whether or not chronically inflamed sites of gingival, periodontal and mucosal epithelial tissues are rife with similar interactions.

Commented Gutkind, "We are now learning why cancer cells grow unrestrained and subvert surrounding healthy cells to satisfy their increasing need for nutrients and oxygen. These processes involve a constant communication among cells, and deciphering the molecular complexity of the underlying mechanisms provides golden opportunities for the early diagnosis and new preventative strategies and treatment options for human malignancies." Dorsam discussed that, "Oral health care clinicians should be aware of the very complex network of processes underneath a seemingly innocuous oral lesion, that there is something beneath the surface. Now because of increasing knowledge about communication within and among cells, we'll be able to treat malignant processes in a new way."

Both molecular biologists agreed that dental school pharmacology classes could benefit from more fully addressing cell communication. Dental students are not taught what happens and what is involved in cell-cell signaling, just why disease happens and what medicine is needed. But dentists need new drug therapies, and now those drugs are in the alpha phase of development. Thus, in the next five to 10 years, it is going to become important for the dental students to be taught the basic molecular principles behind oral health and disease so they know how the new drugs coming their way work.

For now, among the emerging uses of GPCR-targeting agents, drug delivery using radio-labeled and binding peptides are candidates for targeting malignant cells that overexpress GPCRs. The result could be that cancer treatments would have fewer side effects. With GPCR communicating such a core part of aberrant signaling and metastatic commanding, drug developers will be eyeing these cell-surface molecules as lead candidates for getting the job done.

Temporarily Tricking Cells, Systems and Organs in the Dead to Stall for Emergency Resuscitation

Since it has been shown that a cell has receptors to "hear" and signals to "send out" messages, then another question becomes in what ways can researchers try to fool cellular surveillance mechanisms to not switch on a gene function. For example, investigators are pursuing repressing the cell

apoptosis process — programmed initiation of cell death — for "an hour or more" so surgeons can repair heart and lungs of the newly dead. This stunningly strange medical research (near) breakthrough is in the works and involves distracting normal cell reactions to delay or circumvent functions like apoptosis. In the United States, one such effort comes from the director of the recently established Center for Resuscitation Science at the University of Pennsylvania in Philadelphia.

In a 2007 report, Lance Becker, MD, an emergency medicine physician researcher at U Penn, explained that contrary to the belief that four to five minutes after a "fatal" heart attack where the failed pump means noncirculating blood flow causes the brain to "begin dying," turns out it's just in shut-down mode to conserve oxygen that cells are also still alive.[11] They looked at heart cells under a microscope in their "very fresh cadaver" and found, although starving for oxygen since they were cut off from a circulating blood supply, they were alive. Only hours later did they die. But you cannot resuscitate a person who is clinically dead for an hour from a heart attack, Becker's team learned, because when the blood supply resumes, the cells die from the suddenly resumed oxygen supply. It's too much of a shock.

In fact, emergency room protocol of administering an oxygen mask to heart attack sufferers, administering epinephrine and shocking the heart muscle to rush in the flow of blood to restore oxygen supply may be a backward approach, in a sobering theorem put forth from the Center. The oxygen flush may be misinterpreted as a tsunami of a terrible problem such as a cancer attack, and a cell is programmed to kill itself rather than compromise to this sudden change of status.

A four-site study published in 2006 showed using a heart-lung bypass machine (to keep the brain fed blood mechanically circulated and the heart in suspended animation until its restarting was worked out) had a 70 percent higher survival rate than traditional methods of resuscitation.[11]

Becker's center will be supporting the investigation and information sharing of a host of novel principles for newly "dead":

WHAT IF THE
chemical alphabet of signals is in effect, decoded? Will we one day have transcripts of dialogues between cells?

- slowing down metabolism through methods such as cooling blood with ice and salt or other hypothermia-induction strategies;
- continuing trials of circulation with heart-lung bypass to keep brain oxygen levels going;
- molecular approaches to blocking chemical communications of genetically programmed switch-on of mitochondria that initiate apoptosis;
- acting as an information hub in publishing and soliciting/reviewing papers and study proposals for additional approaches in alternative resuscitation ideas; and
- training investigators who are willing to take risks, and accepting such patients willing to submit to such unorthodox end-of-life treatments to help advance science, and maybe even increase their odds of not making it over to the other side.

Knowing that all types of cells communicate with each other, and that investigators are intent on not only understanding how they do this, but knowing that they can control it regarding apoptosis, will no doubt bring therapeutics to a level of biomolecular tricksterism and passive means to faux-signal the body's trillions of cells into a nitrous-oxide like stupor: "You are getting sleepy, all is well, relaaaxxxxx …" as your body sits dead on a gurney, blood corpuscles kept viable by flowing via a machine-pumped circuit like some lazy river, while perhaps a cloned human heart muscle is retrieved, jump-started, and surgically sewn into your arteries by the fastest resuscitation cardio ER team in the history of medicine.

Science moves fast, and there are no flies on Dr. Lance Becker.

Conclusion

The general concepts of cell signaling and receptoring — cell to cell communicating — are the same in all cell types. But, signaling pathways between different cell species, whether inside the common housefly or the stench of "morning breath" tongue, are distinctively different. Unlocking one door, leads to 10 more. There's no stopping now, so while interference with cell signaling will be the first round of therapeutics, what if the chemical alphabet of signals is in effect, decoded? Will we one day have transcripts of dialogues between cells? Then can we script artificially encoded signals that we can broadcast to "turn on" and to command gene functions? Imagine if, tailored to the patient's disease progression, molecular surgeons will one day use a wireless mouse chairside to click away at instructing the DNA function to switch on/off and to horizontally share chromosomes for customized therapy?

No one thought cloning would happen in our lifetime either. ■■■■

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Comparison of Patient and Surgeon Assessments of Pain in Oral and Maxillofacial Surgery

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ABSTRACT Pain perception is a physical sensation interpreted in the light of experience and is influenced by a great number of interacting factors. Clinicians are constantly required to combine subjective and objective information to determine optimal treatment of pain. In this study, the authors prospectively compare patients' subjective complaints of pain using the visual analog scale to the surgeons' assessment of pain using standard history and physical examination findings.

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Pain is one of the most common reasons why many individuals seek medical care. It is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[1] Pain perception is a physical sensation interpreted in the light of experience and is influenced by a great number of interacting physical, mental, biological, physiological, psychological, social, cultural, and emotional factors. Each individual learns the application of the word through experiences related to injury early in life.[1] The response to pain is very variable both subjectively, behaviorally (crying, yelling, teeth clenching, wincing), and physiologically through various individual ranges of sympathetic nervous system manifestations (hypertension, tachycardia, nausea, pupillary dilation, pallor, perspiration).

A remarkable aspect of pain perception is the extreme variability of reactions that it evokes. Many factors affect the perception of pain. Comparison of pain perception in civilians and soldiers showed that 83 percent of civilians about to undergo major surgery asked for pain killers, but only 32 percent of wounded soldiers requested pain medications.[2] Stress plays a key role in pain perception. Stress-induced analgesia is the reduction of pain that results when people are under stress.[3] This is partially explained by the effect of the sympathetic nervous system on pain. Anxiety can also have a profound effect. The greater the anxiety of the

individual, the more likely that the response to a stimulus is interpreted as painful.[4] Additionally, the pain threshold is lowered with increasing anxiety.[4]

The perception of pain can also be affected by individual cognitive and psychological factors. In one study, volunteer subjects received a series on increasingly intense electric shocks while in the presence of experimental models who appeared to be getting similar intensity electric shocks. Some experimental models reacted tolerantly to the shocks while others reacted intolerantly. They found that subjects reported less pain and accepted more intense shocks when in the presence of a tolerant model.[5]

The influence of gender on pain assessment and management was reviewed by Nevin.[6] Her findings suggest a trend toward women reporting a lower pain threshold and tolerance and higher pain intensity than men. A more recent study by Cepeda in 2003 looked at gender differences in pain perception in a prospective cohort including 423 women and 277 males emerging from general anesthesia after surgical procedures. The level of pain was reported on a Visual Analog Scale, VAS. Equal doses of morphine were administered until the VAS rating was ≤4. They concluded that women experience more pain and require more morphine than men to achieve a similar degree of analgesia.[7]

Todd reviewed pain assessment and ethnicity and found very inconsistent results in the literature.[8] The need for accurate and specific categorization of ethnicity and measurement of variables such as socioeconomic status and acculturation, in addition to ethnicity was highlighted. In a previous study, Todd looked at the effects of ethnicity on physician estimates of pain severity in patients with isolated extremity trauma.[9] It was concluded that the physicians' ability to assess pain severity does not differ for Hispanic and non-Hispanic white patients.

Previous emergency room studies have suggested that physicians need to be more attentive to their patients' analgesic needs.[10-12] In a study comparing patient and practitioner assessments of pain from commonly performed emergency department procedures, it was found that practitioners and patients easily identified painful procedures. However the correlation between patient and practitioner pain assessments was highly variable.[12] They suggested that practitioners need to be more attentive to anesthetic needs of patients before performing painful procedures. Patient and physician evaluations of outcome after total hip arthroplasty was studied by Lieberman.[13] Their study highlighted a discrepancy between patient and physician evaluations that was greater when the patient was not satisfied with the outcome.

Oral and maxillofacial surgeons commonly diagnose and treat patients with head and neck pain. The decision to treat pain is based on subjective and objective findings and can present as a great challenge. Despite the availability of many different pharmacological agents, postoperative pain remains a negative and feared consequence of surgery. This may serve as a deterrent for many patients to seek a necessary surgical procedure. Until better methods are available to quantify pain, clinicians have to rely on subjective parameters and their perception of the patient's response to their physical examination techniques. A patient's vital signs are monitored routinely in hospital wards and clinics. Any deviation of the vital signs from the expected parameters triggers an investigation or intervention by the treating healthcare personnel. A subjective change in the patient's report of pain may also trigger an intervention. The assessment of pain has been introduced as the fifth "vital" sign in addition to the traditional four (temperature, pulse, blood pressure, and respiratory rate).[14] The VAS is a frequently used method to quantify pain severity in pain research.[15] This is a simple tool that can be easily applied by physicians and research assistants, providing reproducible results.[15] It has been validated for use in adults and children as young as age 5.[16,17]

There are several possible explanations for the undertreatment of pain: (1) pain is subjective not objective; (2) the causal basis of pain is often poorly understood; (3) pain is regarded as a symptom, not a disease; (4) there is frequently no definitive treatment for pain; (5) pain does not always fit a scientific model[18]; (6) clinicians face legal and regulatory pressures to restrict the use of narcotics; (7) concerns of side effects and development of tolerance and abuse (narcotics); (8) doctors may assume that patients are over emphasizing their pain level; and (9) adequate funding for pain control is not available

via third-party payers.[18] Undertreatment of pain can have serious consequences to the patients, their families, and society. A decrease in the quality of life and productivity, deterioration of social interactions, increased cost of treatment, prolonged recovery time and the potential for developing chronic pain syndromes. A first step for better treatment of patients' pain is an improved perception of their level of pain.

The objective of this study was to compare the patients' perception of their pain to the surgeons' impression of the patient's pain based on standard history and physical examination techniques during routine consultations, preoperative and postoperative visits at the Emory Clinic, Division of Oral and Maxillofacial Surgery.

Materials and Method

All patients over the age 18 presenting to the oral and maxillofacial surgery clinic at the Emory Clinic in Atlanta for consultation, preoperative or postoperative assessment, were enrolled prospectively for comparison of patient and clinician assessment of pain. Data was collected on 12 clinic days, only when the first author (Bagheri) was available to consistently document the VAS scores. Upon arrival to the clinic, patients were asked by the same investigator to rate their level of pain on a 10 cm VAS. Subsequently, standard history and physical examination techniques were used by faculty or resident surgeons to conduct routine patient evaluations. The evaluating surgeons where asked by the same author to rate his/her perception of the patient's pain on a separate 10 cm VAS. All evaluating surgeons where blinded to the initial pre-evaluation VAS pain rating. The following parameters where also collected: age, gender, race, and the presence of a patient companion in the examination room.

Patients presented to the clinic for a wide range of clinical and surgical conditions at various stages of treatment or consultation. The patients mostly presented for dentoalveolar surgery, disorders of the temporomandibular joint, dentofacial deformities, head and neck infections, obstructive sleep apnea, and other pathologic conditions of the oral and maxillofacial region.

Results

One-hundred twenty-seven patients (N=127) (average age 37.1, range 18-65) presenting to the Emory Clinic OMFS unit were enrolled in the study. **TABLE 1** outlines the demographics of the study group. Patients were enrolled consecutively when the first author was available to conduct the preoperative VAS assessment and document the surgeon's postexamination VAS score. Patient and surgeon VAS scores were compared using the Wilcoxon-signed rank test. A value of $p < 0.05$ was considered statistically significant. **TABLE 2** provides the raw data for the patient and surgeon assessments of pain using the VAS.

The mean pre-examination patient VAS score for the study (N=127) group was 2.4 ± 3.1 (range 0-10) compared to 1.5± 1.8 (range 0-6) for the surgeon. The difference was statistically significantly ($p<0.001$, Wilcoxon signed-rank test). Sixty-five patients reported a pain level of zero on the VAS. Among the 62 patients with some initial pain (VAS ≥1), 43 (69.3 percent) perceived their pain one or more VAS units higher compared to the surgeon. The median difference in pain perception was 2 VAS units higher for the patient compared to the surgeon ($p<0.001$).

The pain assessments of the patient and surgeon were not different between males and females ($p=0.40$, Wilcoxon rank-sum test) or whites and blacks ($p=0.16$). The discrepancies between patient and surgeon scores were not significantly different whether the assessments were performed by residents or attendings ($p=0.63$). The patient's age was not associated with pain assessment differences ($p=0.28$, Spearman rank correlation coefficient). No differences in pain were found based on the presence or absence of a patient companion in the examination room ($p=0.44$ Spearman rank correlation coefficient).

TABLE 1

Patient Demographics

N = 127	
Males	42
Females	85
Average age	37.1
Age distribution	18-65
Race	
White	78
Black	27
Other	22
Patient accompanied	44
Patient alone	83

Discussion

Previous studies in the literature report that physicians frequently undertreat pain.[10-13,18] The results of this study suggest that surgeons underestimate the patients' level of pain and may therefore contribute to inadequate treatment. It is important to notice that the patients evaluated in this study represent a wide range of conditions in oral and maxillofacial surgery. It can be argued that the profile of patients presenting with different categories of problems such as those with temporomandibular joint disorders are significantly different to patients presenting for dentoalveolar surgery or obstructive sleep apnea. Future studies could compare the differences in patients' and surgeons' assessments of pain in different categories of patients. The authors hypothesize that clinicians underestimate the patients' perception

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TABLE 2

Patient and Surgeon Assessments of Pain in Oral and Maxillofacial Surgery

Patient No.	Age	Sex	Patient type	Escort	Patient VAS	Doctor VAS
1	27	F	Orthognathic	No	0	0
2	64	F	Pathology	Yes	4	1
3	25	M	Other	No	0	1
4	22	M	Orthognathic	Yes	2	0
5	36	F	Dentoalveolar	No	0	0
6	29	F	Dentoalveolar	No	5	2
7	56	F	Orthognathic	Yes	3	2
8	18	F	Dentoalveolar	Yes	3	4
9	18	F	Orthognathic	Yes	0	0
10	47	F	TMJ	No	10	6
11	51	F	Dentoalveolar	Yes	7	2
12	64	M	Dentoalveolar	No	0	0
13	24	M	TMJ	Yes	2	2
14	46	F	Orthognathic	No	8	4
15	39	M	Orthognathic	Yes	3	3
16	40	F	TMJ	Yes	8	0
17	45	F	TMJ	No	6	5
18	34	M	Trauma	No	5	2
19	45	F	Orthognathic	No	2	2
20	18	F	Orthognathic	No	5	0
21	20	F	Other	No	8	3
22	20	M	Trauma	Yes	5	3
23	22	F	Other	No	0	0
24	55	F	Dentoalveolar	No	0	0
25	21	M	Trauma	No	0	1
26	77	F	Other	Yes	3	2
27	37	M	Orthognathic	No	0	0
28	65	F	Other	No	0	0
29	24	F	Dentoalveolar	No	1	0
30	18	M	Other	Yes	4	4
31	65	F	Dentoalveolar	No	10	3
32	48	F	Pathology	No	0	0
33	18	M	Other	Yes	0	0
34	25	F	Dentoalveolar	No	3	1
35	32	F	Dentoalveolar	No	0	0
36	43	M	Orthognathic	No	0	0
37	28	F	Pathology	Yes	2	1
38	22	M	Dentoalveolar	No	0	0
39	43	F	TMJ	No	5	6
40	32	M	Other	Yes	0	0
41	54	F	Orthognathic	No	2	0
42	30	M	Pathology	No	0	0

CONTINUES

TABLE 2 CONTINUED

Patient and Surgeon Assessments of Pain in Oral and Maxillofacial Surgery

Patient No.	Age	Sex	Patient type	Escort	Patient VAS	Doctor VAS
43	59	F	Dentoalveolar	No	10	1
44	43	F	Pathology	No	1	1
45	29	M	Dentoalveolar	No	4	1
46	57	M	TMJ	Yes	5	2
47	22	M	Implant	Yes	0	3
48	42	M	Trauma	No	2	3
49	48	F	TMJ	No	6	3
50	29	F	Orthognathic	No	0	1
51	26	F	Implant	No	0	0
52	56	F	Other	No	2	2
53	64	M	Implant	No	0	0
54	56	F	Dentoalveolar	No	0	0
55	52	M	Other	No	0	0
56	38	F	Dentoalveolar	No	0	0
57	19	F	Orthognathic	No	0	0
58	18	F	Orthognathic	Yes	0	0
59	59	F	Other	Yes	10	5
60	45	F	Other	No	0	0
61	52	F	Pathology	No	4	6
62	40	F	TMJ	Yes	10	5
63	25	F	TMJ	Yes	5	2
64	25	M	Dentoalveolar	No	10	5
65	18	F	Dentoalveolar	No	4	3
66	43	M	Pathology	No	0	0
67	31	M	Trauma	No	5	5
68	28	F	Orthognathic	Yes	0	0
69	38	M	Dentoalveolar	No	4	2
70	18	M	Pathology	Yes	0	0
71	19	F	Orthognathic	Yes	0	0
72	65	F	Dentoalveolar	No	2	1
73	31	M	Orthognathic	No	0	0
74	54	F	Orthognathic	No	4	4
75	18	F	Dentoalveolar	Yes	0	0
76	58	F	Other	No	0	1
77	54	F	Orthognathic	No	5	2
78	23	M	Dentoalveolar	No	0	0
79	18	M	Implant	No	0	0
80	29	F	Dentoalveolar	No	0	0
81	38	F	Dentoalveolar	No	0	2
82	18	F	Orthognathic	Yes	0	0
83	82	F	Pathology	Yes	4	6
84	24	F	Dentoalveolar	No	0	0

CONTINUES

TABLE 2 CONTINUED

Patient and Surgeon Assessments of Pain in Oral and Maxillofacial Surgery

Patient No.	Age	Sex	Patient type	Escort	Patient VAS	Doctor VAS
85	60	M	Pathology	No	0	0
86	22	F	Other	No	7	4
87	25	F	Dentoalveolar	No	6	6
88	28	M	Dentoalveolar	No	0	0
89	18	F	Orthognathic	Yes	0	1
90	52	F	Dentoalveolar	No	0	0
91	38	M	Dentoalveolar	No	0	1
92	32	M	TMJ	Yes	6	3
93	28	F	Dentoalveolar	Yes	4	1
94	38	F	TMJ	No	0	1
95	30	M	Other	No	8	4
96	28	M	Dentoalveolar	No	0	0
97	31	F	Dentoalveolar	No	0	0
98	53	F	Pathology	No	8	6
99	39	M	Orthognathic	Yes	3	2
100	43	F	Implant	No	0	0
101	64	M	Implant	Yes	0	0
102	26	F	Orthognathic	No	2	0
103	63	F	Dentoalveolar	No	0	0
104	33	F	TMJ	No	6	6
105	22	F	Dentoalveolar	Yes	0	0
106	19	M	Orthognathic	Yes	0	0
107	28	M	Dentoalveolar	No	0	0
108	25	F	TMJ	No	0	0
109	47	F	TMJ	No	5	3
110	65	F	Dentoalveolar	Yes	0	0
111	29	F	TMJ	No	3	3
112	49	F	Pathology	Yes	0	0
113	52	F	Orthognathic	No	7	3
114	38	F	Orthognathic	No	1	2
115	27	F	Orthognathic	Yes	0	0
116	48	M	Orthognathic	No	0	0
117	49	F	Other	Yes	0	0
118	25	F	Dentoalveolar	No	10	3
119	22	F	TMJ	Yes	5	2
120	27	F	Pathology	Yes	4	4
121	29	F	Dentoalveolar	Yes	0	0
122	52	F	Pathology	No	1	2
123	25	F	Dentoalveolar	No	0	0
124	38	F	Pathology	No	0	0
125	42	M	Pathology	No	0	0
126	30	M	Pathology	Yes	10	3
127	18	F	Pathology	Yes	0	2

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of pain irrespective of the presenting condition. However, a larger sample size is required to confirm this hypothesis.

The results of this study need to be interpreted with caution. The treatment of pain is complex and has puzzled physicians through history. It is not the intention of this study to suggest more aggressive pharmacological treatment of pain. Each case needs to be evaluated independently. However, recognition of the patient's pain level is a critical step in adequate treatment. Psychodynamic psychotherapy is one of the prevailing theories in psychiatry popularized by Sigmund Freud in the early 1900s.[19] It proposed that patients frequently experience improvement in their mental condition through recognition and discussion (free association) of their problems without any active intervention (psychiatric or medical). One can hypothesize a similar relationship with pain. Perception and adequate acknowledgment of pain by clinicians can potentially be therapeutic for the patient. Additionally, the clinician would make treatment recommendations that are more adequate given the patient's condition.

The reported range of pain assessment by the patients on the VAS was from 0 (no pain) to 10 (worst possible pain). However, the highest VAS score by and evaluating surgeon was only 6. The visual analog scale clearly describes a level of pain designated at 10 as the "worst possible pain." One would assume that a patient experiencing a pain of this intensity would show behavioral cues (crying, wincing, moaning, doubling over, clenching the teeth, or covering up the area of pain) that conform to this description of pain. Eight patients (6.3 percent) reported a VAS score of 10. The average VAS score for the same group of patients by the surgeons was 3.9 (range 1-6). It is clear that the assessment differences are

increased on the higher end of the VAS.

Munchausen syndrome (factitious disorder) is well described in the literature.[20] In this disorder, patients overstate their pain for a secondary gain (nonfinancial). The incidence of this disorder is 0.5-1 percent.[21] Therefore, its prevalence does not explain the authors' observation. Malingering is a different condition where patients fake or exaggerate their pain for a primary gain (such as financial gain, work excuse, workers' compensation, insurance, litigation). In a study by Mittenberg using a survey of 33,531 cases of personal injury, 29 percent involved probable malingering and symptom exaggeration.[22] Only a small percentage (3.9 percent) of the patients presented secondary to personal injury. This also would not account for the discrepancy in the authors' observation. However, a more elaborate study on the etiology of injury and the ability of clinicians to perceive the patient's' level of pain would be necessary to shed answers to this question.

Enormous health care costs can arise from both of these conditions or the undertreatment of pain regardless of etiology. It is important to conduct careful history and physical examinations to identify patients that may exhibit either of these conditions and prevent unfavorable treatments and outcomes.

No difference in the assessment of pain between whites and blacks was identified in this study.

The review of the literature by Todd for pain assessment and ethnicity found very inconsistent results in the literature.[8] It would appear a very large sample size would be required to identify any possible ethnic differences in the assessment of pain between clinicians and patients. Sixty-seven percent (85) of the patients in this group were females. That is consistent with previous reports in the literature documenting that females are commonly present to their doctors for the evaluation of pain.[23-25] In the authors' study, surgeons underestimated the patients' perception of pain irrespective of their gender.

The complex anatomy of the head and neck region and the close proximity of multiple specialized tissues and neurovascular structures can make the diagnosis and treatment of pain challenging. Oral and maxillofacial surgeons need to have a low threshold for the referral of patients with chronic pain for evaluation by pain specialists, especially if no surgical etiology is identifiable.

Conclusions

Clinicians using standard history and physical examination techniques in patients reporting a pain intensity ≥1 on the visual analog scale in oral and maxillofacial surgery underestimated a patient's subjective report of pain in more than two-thirds of patients. We also need to be cognizant of other disorders or possible motivations for patients to overstate their pain. ■■■■

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Histopathology Study on Pulp Response to Glass Ionomers in Human Teeth

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ABSTRACT OBJECTIVE: Evaluation of the pulpal response to a resin-modified glass ionomer, a conventional glass ionomer and calcium hydroxide. METHODS AND MATERIALS: Fifty-five deep Class V cavities were lined with Vivaglass Liner, Chembond Superior and Dycal. After seven, 30, and 60 days the teeth were extracted and a histological assessment was performed. RESULTS: There was no statistically significant difference in pulpal response among the three groups for the same time interval (P>0.05). CONCLUSION: Light-cured glass ionomers have similar advantages to conventional glass ionomers.

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Continuous development of new materials provides a wide range of biomaterials appropriate to various clinical conditions in dentistry. Despite all the improvements, there is still a need for a biomaterial that possesses high biocompatibility, antimicrobial effects, and good mechanical properties. Among the recently developed materials, glass ionomer cements, GIC, have gained popularity since their conception in 1972 by Wilson and Kent.[1] Conventional glass ionomer cements present biocompatibility, nonshrinking setting reaction, chemical adhesion to tooth structure and fluoride release.[2] New formulations have been successively developed to overcome some clinical drawbacks of previous cements, especially aimed at improving physical properties.[3] In many clinical situations, the newer resin-modified glass ionomer cements, RMGICs, are an alternative to conventional glass ionomer cements.

To evaluate the biocompatibility of dental materials, a sequence of tests must be performed including in vitro assay for mutagenesis and cytotoxicity (initial tests), local toxicity reactions by intraosseous or subcutaneous implantation of the material in small laboratory animals (secondary tests) and, finally, the usage tests.[4]

Several studies have shown that the light-cured systems of glass ionomer cements exhibit poor biocompatibility and greater cytotoxicity than conventional cements in cultured cells.[5] In vitro studies of Vitrebond and Vitremer have shown some cytotoxic and mutagenic effects leading the investigators to conclude that they may cause pulp irritation.[5,6] Evaluating indirect pulp capping employing a RMGICs, two recent studies reported acceptable pulpal response, and another reported a less favorable pulpal response.[7-9]

This in vivo study evaluated the histological changes in pulp as a response to light-cured resin-modified glass ionomer and compared it with a conventional

glass ionomer and a calcium hydroxide lining material in deep cavities.

Methods and Materials

The study population consisted of 19 females and 12 males, ranging in age from 13 to 32, with a mean age of 18 years. All of the patients required the extraction of permanent premolars for orthodontic reasons. The participants, and their parents or responsible persons, received an adequate explanation concerning the experimental rationale, clinical procedure, and possible risks. The parents and all the volunteers were asked to read and sign a consent form explaining the research protocol approved by the ethical guidelines.

Patients were required to meet the following criteria.

To be included in the study:

- Permanent first premolars scheduled for orthodontic extraction
- Scores of two or less using the periodontal screening record (evaluation consisted of examining the premolars with a periodontal probe)
- Completed root formation

To be excluded from the study:

- Presence of caries
- Presence of restorations
- Presence of abrasion or erosion
- Presence of pulpal symptoms or radiographic periapical lesions

After local anesthesia the teeth were isolated with a rubber dam. A Class V cavity on the buccal surface of each tooth was prepared with a 440-diamond point (Shofu Inc, Kyoto 605-0983, Japan) in a high-speed handpiece under copious water spray coolant. New diamond points and burs were used after every four teeth. The axial wall was excavated using a carbide round bur at low speed until red feature of the pulp was observed.

The 55 experimental teeth were divided into three groups. In the first group, Vivaglass Liner (Ivoclar Vivadent AG, Schaan, Lichtenstein) was applied to the axial wall of the cavity and then was light-cured for 20 seconds. In the second group, Chembond Superior (Dentsply, Detry, UK) was applied as a liner in the axial wall of the cavity; and in the third (control) group, Dycal (Dentsply, Milford, Del., USA) was applied. All of the materials were used according to manufacturer's directions. After application, two layers of a copal varnish, Copalite (Cooley & Cooley LTD, Houston, Texas) were added. The cavities were restored with a high copper amalgam, Oralloy (Coltene Whaledent, USA). After seven, 30, and 60 days, the teeth were extracted under local anesthesia.

The mesial and distal approximal surfaces of the teeth were reduced with a high-speed diamond bur under spray coolant until the pulp became almost visible through the remaining dentin to facilitate the penetration of the fixative solution. The surfaces were then fixed with a 10 percent neutral buffered formalin solution for one week. The teeth were demineralized with 10 percent ethylenediamine tetracetic acid (ETDA) with PH (7-7.4) as a demineralzing solution at 25 degrees (Celsius) for 60 days, and each tooth was then embedded in paraffin. 5µm-thick serial sections were prepared through the cavities and pulp, obtaining approximately 80 to 100 sections per cavity. They were placed on glass microslides and stained with either hematoxylin eosin for routine histological evaluation or Taylor's modification of Gram's staining technique for detecting microorganisms.[10]

The pulpal responses and the presence of bacteria in their cavities were evaluated using a light microscope (Zeiss, Germany). The RDT, remaining dentin thickness, was ranged as deep (0-0.4 mm), moderate (0.4-0.7 mm), and shallow (more than 0.7 mm). Evaluation criteria for odontoblastic changes, inflammatory cell infiltration and reactionary dentin formation are shown in **TABLE 1**.[11]

The results of odontoblastic changes, inflammatory cell infiltration, and reactionary dentin formation were statistically analyzed using the Kruskal Wallis and Mann-Whitney test at 95 percent level of confidence. Fisher's Exact test ($\alpha = 0.05$) was also used

TABLE 1

Evaluation Criteria[11]

Odontoblastic changes
(Non) Remarkable change was not observed in the pulp
(Slight) Disarrangement of odontoblasts was noted slightly below the cut dentinal tubules
(Moderate) Disarrangement of odontoblasts was seen through most of the cut dentinal tubules
(Severe) Disarrangement of odontoblasts was noted below the remaining dentin.
Inflammatory cell infiltration
(Non) None or a few inflammatory cells were observed through the pulp.
(Slight) A few inflammatory cell infiltrations were noted below the cut dentinal tubules.
(Moderate) Inflammatory cell remarkably observed below the remaining dentin.
(Severe) Severe inflammatory cell infiltration was seen through the pulp.
Reactionary dentin formation
(Non) No abnormal or reparative dentin observed.
(Slight) A small amount of reactionary dentin was noted.
(Moderate) Reactionary dentin was observed below the almost-cut dentin.
(Severe) Complete and large bulk of reactionary dentin was noted.

TABLE 2

Results of Histological Findings

Time Intervals		7 days	30 days	60 days
Experimental groups		VCD	VCD	VCD
Number of specimens		875	566	666
Odontoblastic changes	Non	322	224	221
	Slight	121	121	122
	Moderate	432	221	323
	Severe	000	000	000
Inflammatory cell infiltration	Non	022	325	223
	Slight	212	221	313
	Moderate	640	020	130
	Severe	001	000	000
Reactionary dentin formation	Non	875	253	221
	Slight	000	313	445
	Moderate	000	000	000
	Severe	000	000	000

V: Vivaglass Liner; C: Chembond Superior; D: Dycal

for understanding the correlation between pulpal responses with microorganisms and remaining dentin thickness in each group.

Results

Results of histological findings are shown in TABLE 2.

Bacterial penetration was observed in only six cases (five cases in cavity walls and only one case in pulp). There was no significant correlation between pulpal responses with dentinal thicknesses and microorganisms (P>0.05).

In the Vivaglass Liner, there was a statistically significant difference in inflammatory cell response among three intervals (P<0.05). Inflammatory cell reaction in the seven-day group was significantly more than in the 30- and 60-day groups (FIGURES 1 AND 2). There was no statistically significant difference in odontoblastic changes among three intervals; slight odontoblastic change was seen in each interval (FIGURE 3).

In Chembond Superior there was significant difference only in reactionary dentin formation among three intervals (P<0.05). The mean rank of reactionary dentin formation after seven days was significantly less than in the 60-day group (P<0.05) (FIGURE 4).

The results of Dycal were similar to those of Chembond Superior.

The Kruskal Wallis test showed that there was no statistically significant difference in odontoblastic changes, reactionary dentin, and inflammatory cell response among the three groups for the same time interval (P>0.05).

Discussion

Certain controversy persists regarding the biocompatibility of various RMGIC systems. Some studies have reported an innocuous histologic pulp response to RMGICs in Class V cavities, but in vitro studies often showed some cytotoxicity.[5,6,12,13]

The purpose of this study was to compare the in vivo pulpal response to a resin-modified glass ionomer and a conventional glass ionomer and to evaluate the correlation between the pulpal responses with the presence or absence of bacteria and the remaining dentin thickness. The pulpal responses to these materials were compared with a Ca(OH)2 at three time intervals in accordance with the Craig and Powers protocol.[4]

According to previous studies, each subgroup consisted of five to eight samples and amalgam was used as a filling material.[12,14-16] Although a number of studies claimed that pulp tissue response is caused only by the presence of bacteria, in vitro studies have demonstrated that resin monomers diffuse through the dentin tubules and cause cytotoxicity.[5,17,18] Previous studies have demonstrated that cellular compatibility of RMGICs varies significantly.[19,20] Schmalz and others showed that Vitrebond causes a very strong cytotoxicity effect when evaluated by dentin barrier tests.[21] Nascimento and others applied Vitrebond as a pulp-capping agent in sound human teeth, and no pulp repair or dentin bridge formation was observed even after 300 days.[22] They concluded that Vitrebond is not an appropriate pulp-capping agent to be used in mechanically exposed, sound, human pulps. However, it has been reported that the pupal response to visible light-activated glass ionomer cements may be quite favorable when applied as a cavity liner.[7,23]

The present study showed that al-

FIGURE 1. Cavity preparation, remaining dentin thickness and pulp tissue. The odontoblast layer is disrupted and the cells were displaced into the dentinal tubules. Mild and scattered inflammatory cells are present. (Vivaglass Liner, seven days.) (H & E; 40X.)

FIGURE 2. Moderate to severe aggregation of chronic inflammatory cells under the remaining dentin thickness. (Vivaglass Liner, seven days.) (H & E; 200X.)

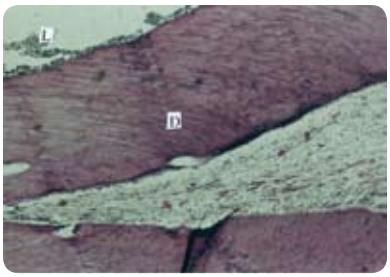

FIGURE 3. A sample of Chembond Superior, seven days. Remnant of liner (L) and remaining dentin thickness (D). Odontoblast layer is disrupted. (H & E; 40X.)

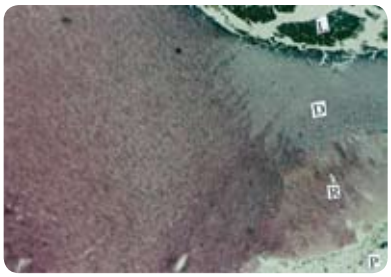

FIGURE 4. Reactionary dentin formation (R) under the remaining dentin thickness (D). Remnant of liner (L) and pulp (P). (Vivaglass Liner, 60 days.) (H & E; 40X.)

though pulpal responses in the same time intervals did not differ significantly among materials, inflammatory cell response in Vivaglass Liner after seven days was significantly more than in the 30- and 60-day groups. According to Geurtsen and others, HEMA and TEGDMA may be released from RMGI in the early 24 hours after polymerization.[5] Buillaguet and others also demonstrated the diffusion of HEMA through dentinal tubules, even against internal pressure.[24] The cytototicity of glass ionomer is reduced with time, as seen in the present study.[6] RMGIC has a burst release of fluoride and may also have a burst release of monomers that will be decreased with time. This finding agrees with the results observed by About and others.[25]

All of the testing materials in this study showed slight-to-moderate inflammatory reactions and no bacterial presence except in six cases. In this study, bacterial-staining data indicated that the lining and filling materials provided an almost complete seal against microleakage through all time intervals. There was only a reversible slight-to-moderate pulpal response, since the testing materials provided an excellent biological seal. This acceptable pulpal response was dependent upon the prevention of bacterial penetration or the lack of toxicity of glass ionomers.

The results of this study showed there was no correlation between the presence or absence of microorganisms and remaining dentin thickness with the pulpal response. The authors' finding corroborates the results of a study done by Sonoda and others.[11] This is probably due to the minimal changes in dentinal thickness prepared in this study and also due to a biologic seal which prevented the bacterial penetration through the pulpal tissue.

If in this study the pulpal response to resin-modified glass ionomer after elimination of carious lesion had also been evaluated, the results of the study could better imitate clinical conditions. It is suggested that a study for evaluation of pulpal response to glass ionomer in deep carious lesions be done in the future.

Conclusions

The glass ionomer systems that were tested provided an almost complete seal against bacterial microleakage through all time intervals. No serious inflammatory reaction of the pulp was observed. The pulpal response to the Vivaglass Liner in seven days was significantly higher than the other intervals.

In all groups, reactionary dentin formation after 60 days was more than other intervals. There was no significant difference in odontoblastic changes, reactionary dentin formation and inflammatory cell response among the groups for the same interval. There was no correlation between pulpal response with dentinal thickness and microorganisms. ■■■■

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Beginning the Discussion of Commercialism in Dentistry

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ABSTRACT There is increasing concern over commercialism in dentistry. Multiple factors contribute to this trend, which has the potential for fragmenting the profession, exacerbating the access issue, and eroding the public's confidence in dentistry. There are both positive and negative aspects of commercialism. Positive approaches for promoting oral health in the face of commercialism hold the greatest promise. The core theme in the recommendations from Ethics Summit on Commercialism is that competent, comprehensive, and continuous oral health care is appropriate and should be promoted to the American public.

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The things we don't understand are often frightening; and frightening things can be dangerous. So it is with commercialism in dentistry. There is a growing sense that a desire for excessive profit may be leading, in some cases, to unnecessary dental care, to provision of substandard care, or to lack of comprehensive treatment.[1-3] It is possible that commercial orientations could be exacerbating the access problem. There are concerns over inflated claims made to practitioners in advertisements and in the literature, and that some in the profession portray — to their colleagues and the public as well — an image of dentistry that seems to value fragmentation of care and too heavy an emphasis on procedures that have potential for large financial gain rather than oral health.[4-6]

Perhaps at no other time since the turn of the last century when dentistry fought to become a scientifically based profession has there been greater tension between financial and patient motivation. These tensions and the various views regarding how they should be addressed, are causing segmentation within dentistry, both among practitioners who have increasingly divergent values and among Americans who are served, unserved, or misserved by a system that is becoming fragmented.[7]

We can, and we probably should, move the conversation beyond editorials. We are able, and it would likely be beneficial, to distinguish our perceptions of good commercialism from bad commercialism, because there are certainly healthy sides

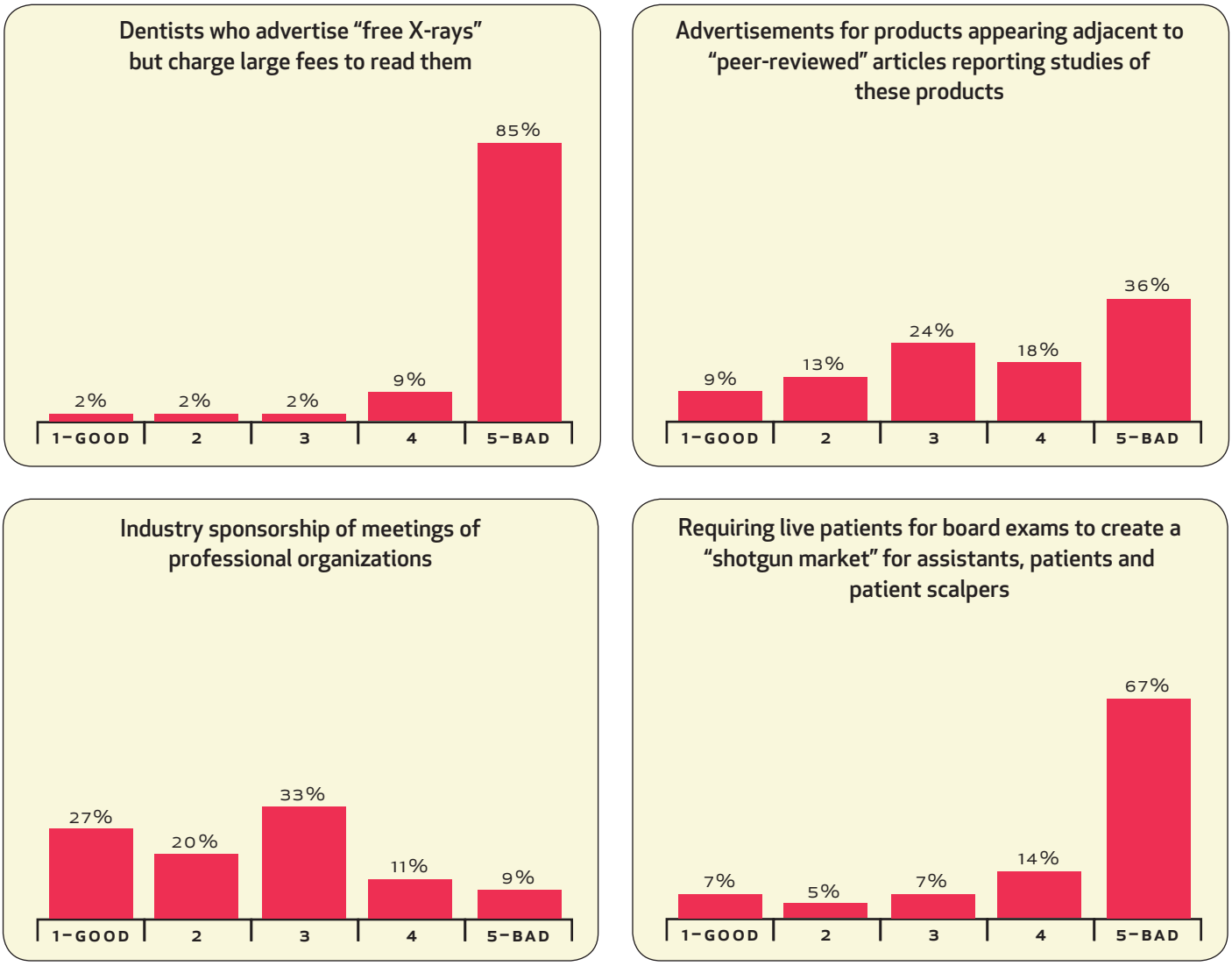


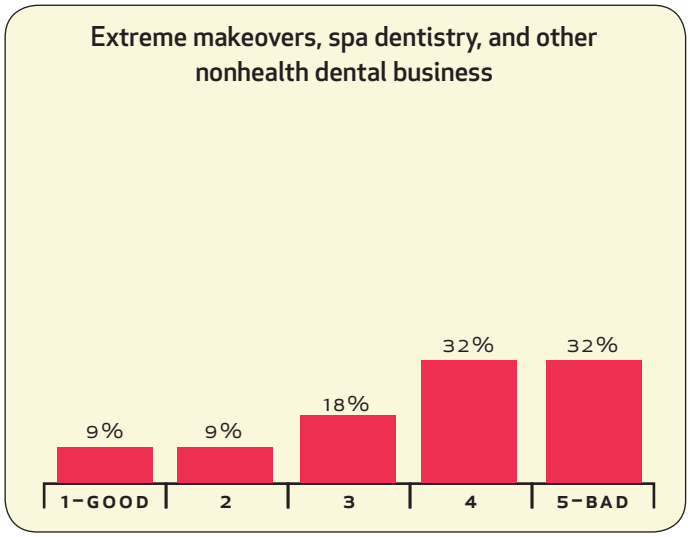
FIGURE 1. Examples of the range of opinion regarding various dental practices, as rated by participants in Ethics Summit on Commercialism.
(Good commercialism is defined as exchanges that benefit both parties because they are based on trust and sharing adequate information; bad commercialism is defined as subordination of trust and information to achieve excessive profits.)

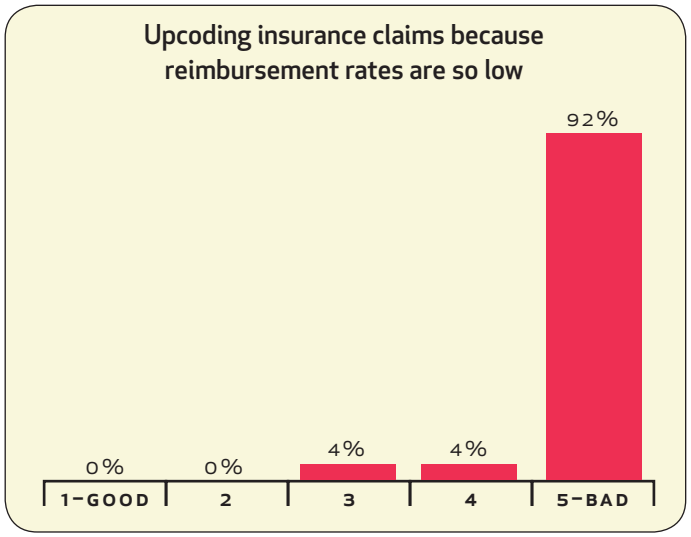
to commerce that need to be separated from the potentially damaging elements. It is time to engage in direct, open, and serious discussion about the effects of rising commercialism on dentistry.

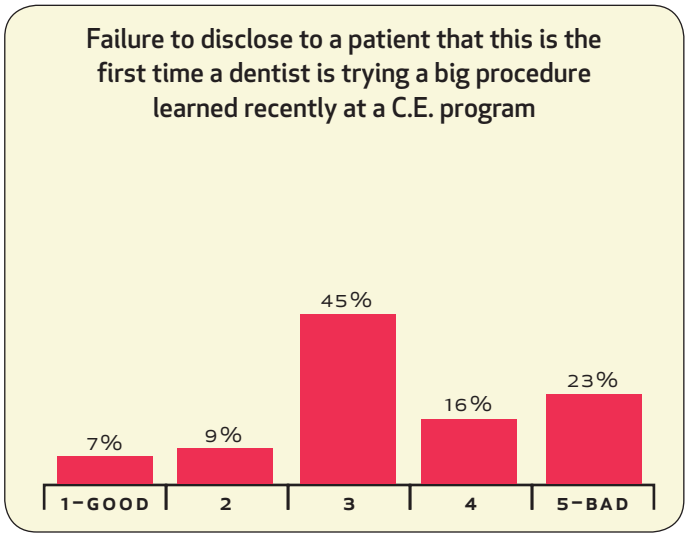
It is in this spirit that the American College of Dentists and the American Dental Association invited approximately 60 representatives from organized dentistry, education, specialties, insurance, research, professional development, and journalism communities to participate in Ethics Summit on Commercialism, a two-day conference at the ADA headquarters building in Chicago on Feb. 28 and March 1, 2006. This is a report of that meeting.

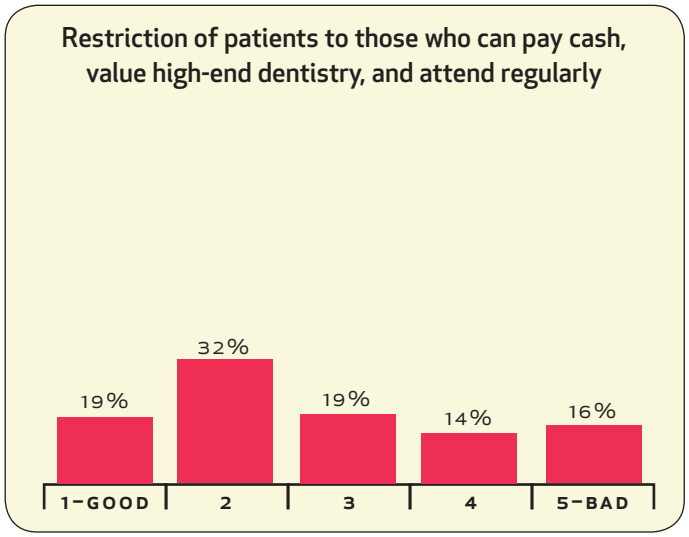
Chief Complaint — Wrestling with the Devil

Dr. Gordon Christensen provided an overview of the extent and nature of the problem. Reflecting on a set of specific examples, it is possible to identify patterns among the concerns over commercial claims made by dentists, advertisers, industry, and those providing continuing education to the profession. These patterns include: (1) claims in which the commercial intent out shouts professional or patient-health concerns; (2) claims that come from many sources; (3) claims that make no pretense at scientific or professional evidence (they play to both patient and dentist short-term needs); and (4) claims that are increasing in both frequency and boldness. Both the public and the profession are being told that, for a price, they can have what makes









money or makes them look good, while the traditional professional values of comprehensive health based on science and realistic expectations are not finding the same level of general expression.[8-10]

But the devil is indeed in the details. What is needed is a definition of commercialism that captures the complex nature of the concept and invites open discussion. There is an inescapable financial element in dentistry. The origins of professions such as medicine can be traced to the 17th and 18th centuries when only gentlemen were welcomed. Each had independent means, making contamination of practice by money interests a rare thing. Those conditions no longer exist, although society continues to expect that professionals will place treatment considerations above financial concern.

Some of the positive aspects of commercialism include exchange of value, building bonds and relationships, and receiving a fair compensation. Commercialism stimulates innovation and support services for the profession. On the other hand, the temptation of money over patient or the public good, working at the level of commodities (lowest common denominator for quality), large-scale operations that fail to respond to individual needs, advertising that is misleading, and making profit a standard for colleagues are negative aspects of commercialism.

Participants in the Summit accepted the definition that commercialism (in the negative sense) consists of "attitudes or methods that excessively emphasize profit or business success."

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TABLE 1

Sources of Commercialism as Rated for Potential Damage by Participants in the Ethics Summit on Commercialism

	8, 9, 10	Avg	St Dev
Practice management courses overemphasize profit and business success	28	7.36	2.76
Society stresses financial success and a "me-first" attitude	27	7.32	2.07
Professionalism is inadequately emphasized in dental schools	24	6.64	3.08
Traditional professional ideals are insufficiently emphasized	23	7.00	2.45
Students model commercial orientation of young faculty and recent grads	23	6.68	2.56
Traditional professional ideas of service are no longer relevant	22	6.07	3.22
States can do little about commercialism because of laws and regulations	22	6.20	3.05
Dental organizations do not provide adequate guidance through codes, etc.	22	6.42	2.84
Dentists ignore organizational guidelines	21	6.47	2.41
Dental school debt adversely affects professionalism of young practitioners	20	6.95	2.39
Dental education is apathetic to commercialism; not considered a problem	20	5.66	3.13
Continuing education courses depict dentistry as a commercial endeavor	19	6.91	2.45
Dental leadership avoids addressing commercialism for legal fears	19	6.34	2.58
Enforcement of organizational guidelines is inadequate	19	6.42	2.42
Dental publications depict dentistry as a commercial endeavor	18	6.32	2.30
Professionalism has been re-defined to encompass commercial qualities	17	6.64	2.33
The "climate" of dentistry conditions dentists to think of it as a business	17	6.36	2.29
Dentists are apathetic to the commercialism issue; not considered a problem	16	6.09	2.41
Dentists believe dentistry is a job and practices are businesses	16	6.30	2.44
Industry emphasizes financial advantage of products over patient benefit	16	6.11	3.07
Dental leadership avoids addressing commercialism for fear of losing members	16	6.12	2.49
Dental meetings depict dentistry as a commercial endeavor	15	6.32	2.35
Dental leadership is apathetic to commercialism; not considered a problem	11	5.32	2.59
Society accepts dentistry as primarily a commercial endeavor	9	4.69	2.77

Commercialism is commonly understood to mean a mutually beneficial method of exchange — essentially a neutral term. It takes on negative overtones, especially in professions such as dentistry, when fiduciary relationship involving trust among patients, peer professionals, related institutions and organizations, and society as a whole are eroded. Because these interactions are sophisticated and claims often are not subject to direct verification, the information contained in informed consent, advertising and research claims, conflict of interest statements, professional contracts, and so forth must be sufficiently complete and accurate that they would not lead to later remorse. Commercialism in a positive sense, or better "good professional business practices," preserves the criteria of fiduciary relationships and valid information.

This bi-directional definition of commercialism can be illustrated by the range of opinions on specific instances in dentistry (FIGURE 1).

History — Who Caused This Mess?

The factors contributing to the status of commercialism in dentistry are complex. One way to explore patterns underlying multifactorial issues is experts' ratings that are then subjected to statistical analysis. This was done by participants in the Ethics Summit. A set of 24 potential factors was considered. These are displayed in TABLE 1, and as originally presented, they were grouped into four sets: (1) general (society and individual practitioners), (2) education, (3) leadership in organized dentistry, and (4) regulatory. Participants expressed their views of the relative power of each of the 24 factors to make commercialism in dentistry a cause for concern.

This exercise revealed the complexity of the issue. Average ratings for the top 22 factors differed by a single point or less on an 11-point scale. This means that general agreement or disagreement masks differences at a deeper level of

TABLE 2

Statistical Analysis for Clusters of Commercial Factors Rated by Participants in the Ethics Summit on Commercialism

	Factor I	Factor II	Factor III	Factor IV	Factor V
	34%	11%	8%	7%	7%
Practice management courses overemphasize profit & business success	0.499	0.479	-0.318		
Society stresses financial success and a "me-first" attitude				0.447	0.602
Professionalism is inadequately emphasized in dental schools	0.575	-0.442			
Traditional professional ideals are insufficiently emphasized	0.626			0.444	
Students model commercial orientation of young faculty & recent grads	0.579	-0.514			
Traditional professional ideas of service are no longer relevant	0.515		0.409		
States can do little about commercialism because of laws & regulations	0.665				
Dental organizations do not provide adequate guidance through codes, etc.	0.504		0.542		
Dentists ignore organizational guidelines	0.526				
Dental school debt adversely affects professionalism of young practitioners	0.595				
Dental education is apathetic to commercialism; not considered a problem	0.696				
Continuing education courses depict dentistry as a commercial endeavor	0.578	0.408			
Dental leadership avoids addressing commercialism for legal fears	0.702				
Enforcement of organizational guidelines is inadequate	0.576	0.417	0.447		
Dental publications depict dentistry as a commercial endeavor	0.669	0.411			
Professionalism has been re-defined to encompass commercial qualities	0.600	-0.592			
The "climate" of dentistry conditions dentists to think of it as a business	0.570	-0.425			0.444
Dentists are apathetic to the commercialism issue; not considered a problem	0.478			-0.554	
Dentists believe dentistry is a job and practices are businesses	0.615				
Industry emphasizes financial advantage of products over patient benefit	0.684		-0.409		
Dental leadership avoids addressing commercialism for fear of losing members	0.730	-0.431		0.549	
Dental meetings depict dentistry as a commercial endeavor	0.577	0.592	-0.431		0.513
Dental leadership is apathetic to commercialism; not considered a problem	0.549				-0.425
Society accepts dentistry as primarily a commercial endeavor	0.513			-0.587	

Factor I	Global factor — commercialism is a large, multifactorial issue
Factor II	Near professional commercial drivers (practice management courses, C.E. courses, publications, meetings, and lack of enforcement; values taught in schools, modeling and mentoring, and professional ideas as antidotes to this driver)
Factor III	Professional driver (service ideal enforced through codes and enforcement; practice management courses, industry, and meetings seen as detracting forces)
Factor IV	Fragmentation of values (traditional professional values need to be stresses in the face of social narcissism and a collaboration of (some) patients with (some) dentists to favor commercial redefinition of practice)
Factor V	Public perception (leadership in organized dentistry fighting general societal trend toward commercialism)

Principal components factor analysis, no rotation. Cronbach's alpha, a measure of consistency among the ratings, is a modest .691.

understanding. Certainly, participants were not indifferent. Sixteen of the 24 factors had both ratings of 0 and 10. The factors are arranged in TABLE 1 in descending order based on the number of 8, 9, and 10 ratings received.

Both discussion at the workshop and the statistical analysis shown in TABLE 2 tended to point in the same direction.[11] There were no "camps" among the participants. Industry, education, and practitioners did not band together in clumps and point to others as the problem, nor was society at large fingered for blame. This is an optimistic finding since it was felt by participants in the workshop that a solution to the problem could be found that incorporates the interests of

all parties in oral health care. This was confirmed by the very large common factor in the statistical analysis — accounting for one-third of the total variance.

A second pattern to emerge in the statistical analysis, and confirmed in the subsequent work of the group, concerns "near professional." A powerful force, for good or not, includes practice management courses emphasizing profit and success, the continuing education industry when it stresses profit as an outcome, publications with a commercial tone, professional meetings with their heavy emphasis on commercial exhibitors, and lack of enforcement of professional codes. As part of this pattern, the professional values taught in dental schools, modeling and mentoring, and the influence of senior professionals on young colleagues, and articulation of professional ideals generally were recognized as a balancing force to the "near professional" commercial interests.

A third pattern might be labeled "professional participation." It includes positive elements of organized dentistry articulating and enforcing standards of professional conduct in the face of countervailing pressures from industry, continuing education, and meetings of organized dentistry. The final two factors to emerge appear to be very similar. Both include a tension between societal values that appear to be drifting in the direction of short-term personal concerns balanced against clear definitions of comprehensive professional responsibility coming from the profession.

One factor seems to reside at the practice interface between individual dentists and patients; it involves a segmentation of the profession into dentists who are ready to respond to consumerism and those who favor a traditional health-based approach. The final factor raises this tension in values to the level of dentistry as an entire profession and all of society.

It is believed that generational differences play a role in the multiple perspectives on commercialism. Writers on the topic point to the historical pattern of older generations complaining that their juniors are poor at following the rules while the younger generations complain that the rules need to be changed.[12,13] (Currently senior dentists should refer to Douglas More's extensive study of dental students in the 1960s to see what their elders thought of them at the time.[14]) Because this generation of leadership in dentistry must inevitably pass the profession to its juniors, it is imperative to work with them rather than blame them. There is some evidence in both dentistry and other professions that the critical time for the creation of professional values is the first few years of practice — not education.

OLDER GENERATIONS complain that their juniors are poor at following the rules while the younger generations complain that the rules need to be changed.

A multiyear project out of the Harvard School of Education demonstrated a pattern among those in journalism, molecular biological research, and acting, where the successful veterans in each field underestimated the difficulties and pressures on young professionals (after all, they had succeeded).[15] Those in school learned role-played professional values in protected environments. The greatest danger occurred during the first years of independent practice and some number in each group chose a strategy of temporarily suspending the professional values they had learned in school "just until they get established."

There is research showing that educational debt in dentistry has remained roughly constant when compared with dentists' incomes over the past two dozen years. It is also the case that the debt impact of starting a practice is greater than educational borrowing. At one expensive private school, practice debt averages 1.4 times educational debt, with both higher interests rates and shorter pay-back periods. A research study at that school found that there was no correlation between amount of educational debt and reported "unconventional" profiles of procedures performed; there was, however, a slight trend for "unconventional" practice profiles related to debt in establishing a practice.[16]

Diagnosis — What Needs to Be Fixed?

Based on a sense of the challenges of the problem that commercialism poses for the dental profession and an analysis of the multifactorial nature of the situation, it is possible to address the specific paths this trend will take — and ultimately how the perceived bad forms of commercialism can be addressed. There are three interrelated tasks: (1) define the consequences in terms that link specific factors to potential damage for the oral health care of America or to the dental profession; (2) prioritize these consequences in terms of their likely impact, weighing both the damage and the possibility of such damage occurring; and (3) prioritize the potential for mitigating these consequences through appropriate and timely action by the profession. Expert judgment rather than scientific data are

TABLE 3

Concern Over Negative Impact of Commercialism, Priorities by Importance and Potential for Management

	Importance	Management	Concern
Consumerism promoting cosmetic care, decreased health care, altered delivery models	67	64	4247
Segmentation in delivery causing decreased oral health, more regulation	60	63	3774
Erosion of codes opening profession to outside intervention	64	45	2870
Change in public perceptions leading to decreased oral health and loss of status	53	50	2691
Pressures on young practitioners distorting their values	40	62	2500
Questionable C.E., other claims causing decrease in science and care	52	47	2436
Not valuing profession leading to loss of monopoly status	44	55	2432
Lose of participation in organized dentistry diminishing its voice	43	44	1862
Schools not modeling professionalism causing unprofessional young practitioners	37	48	1794
Loss of profession's credibility as oral health experts causing decline in health	45	38	1706
Loss of scientific foundation causing loss of status as professionals	44	35	1532

the appropriate basis for this analysis because the problems are ill-defined and quantitative data are not available.

A first approach to this analysis was made at the workshop. Alternating between small teams and the plenary group, an initial list of 11 factors that could be perceived as threats was generated. These are shown in TABLE 3. Each factor is expressed as a connection between a factor and the damage it would cause if left unchecked. These factors were discussed and then prioritized by the entire group. The top three factors to emerge were (1) a commercial orientation in the public would drive demand for "cosmetic" or other oral services that are unrelated to comprehensive oral health — this could have the effect of decreasing the availability of true oral health care, exacerbating access issues and driving inappropriate changes in delivery models; (2) segmentation in the oral health care delivery system would erode quality of care and increase regulation; and (3) weakened ethics and professionalism — or their incomplete implementation — would open the door for outside intervention that may be less responsive to the true oral health needs of the public. There was also concern over (4) emerging unrealistic public perceptions about what constitutes appropriate oral health care fueled by inaccurate or incomplete public information; (5) questionable continuing education and other claims from within the profession; (6) dentistry's loss of credibility as the authoritative source of oral health care information; (7) maintaining the scientific base of the profession; and (8) not valuing professional behavior or participating in professional activities.

Participants also prioritized these 11 factors based on the ease with which they might be averted or ameliorated. It was felt that efforts to respond to public demand for noncomprehensive care, segmentation of the delivery system, and relieving pressures on young practitioners were especially likely to be fruitful. By contrast, stemming possible losses of the profession's credibility in oral health and preventing the erosion of the scientific base of the profession would present more demanding challenges.

Logic dictates that limited resources should first be allocated to circumstances that are serious and manageable. In order to identify the best candidates, the priorities for significance were multiplied by the priorities for manageability to give an attractiveness score to potential initiatives. The factors identified by attendees as contributing to commercialism in its negative sense are presented in order of their attractiveness for action in TABLE 3. Participants were reluctant to leave any part of the problem unaddressed, so several factors were combined into the "codes" category and several groups spontaneously added mentoring young practitioners to their assignment.

Treatment Plan — What Should Be Done?

Positive steps can be taken to head off and address the consequences posed by the negative aspects of commercialism in dentistry. At the workshop, five teams worked with recombined definitions of the most attractive initiatives that various parts of the profession, and particularly the American Dental Association, can take. They developed the 17 recommendations contained in TABLE 4. Participants prioritized the recommendations within each of the five sets but not across sets.

INAPPROPRIATE CONSUMERISM

In the case of inappropriate consumerism, it was felt that dentistry must take the initiative. Currently, a variety of voices is clamoring about access issues. This is a complex matter, and certainly one that must be framed in terms of overall oral health rather than any of

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TABLE 4

Initiatives to Address the Perceived Negative Effects of Commercialism in Dentistry

A. Inappropriate consumerism	The profession must take the lead in addressing the access issue A significant campaign should be mounted to promote comprehensive oral health Public relations activities should continue to draw attention to dentistry's positive role
B. Segmentation within the delivery system	Increase incentives for practice in underserved communities Increase reimbursement levels for underserved populations Develop guidelines for non-specialty practice areas Increase dental office productivity Bring young practitioners into professional relations early
C. Codes	Create a Patient Bill of Rights and Responsibilities Mentorship and early involvement of young professionals Engage components in education and enforcement
D. Public perceptions	Create realistic expectations for patients — informed consent, comprehensive, continual care, etc. Reinforce message that oral health is part of overall health Create media for patients
E. Information	Educate the public about what comprehensive oral health care means Develop standards for commercialism, e.g., disclosure, and publicize them Increase expectations that dentistry is based scientifically grounded claims

several narrow and competing perspectives. Dentistry is in the best position to lead efforts to address this problem. Public relations are important to maintain positive connections with the diverse constituencies served by dentistry, and patient education is a desirable action that was developed in some detail by three of the five working groups. All of the initiatives proposed by the first team are ones where the American Dental Association should take the lead.

SEGMENTATION OF THE PROFESSION

The team working with the consequences of segmentation to the profession that commercialism is now driving developed five recommendations. These involve incentives for practice in underserved areas, more realistic reimbursement for underserved populations, guidelines for representation of services provided by nonspecialty practitioners who make claims beyond those of general dentistry, increasing office productivity for ethical practice, and bringing young practitioners into dentistry quickly and effectively.

What is noticeable about the recommendations from this team is that they generally involve collaboration between organized dentistry and other partners. Education and the government would be logical partners for reaching underserved communities. Private payers and states are a natural pairing for reimbursement. Nonspecialty groups will need to sit down with the ADA to work through the nature of appropriate claims regarding qualifications. Industry has great experience and interest in dental office productivity. The dental honoraries such as the American College of Dentists would be an obvious partner in mentoring young professionals.

The American Dental Association and its tripartite structure is the appropriate home for education, enforcement of the ADA Principles of Ethics and Code of Professional Conduce, and for a creation of a Patient Bill of Rights and Responsibilities. It is likely that such a bill would include the concerns identified by the team working on inappropriate consumerism that realistic expectations need to be developed and broadly communicated.

REALISTIC PUBLIC PERCEPTIONS OF ORAL HEALTH

The team addressing public perceptions explicitly identified the need to inform patients about the value of sound oral health and to create realistic expectations, including access to competent, comprehensive, and continual oral health care, and to informed consent that discloses and discusses all treatment options.

INFORMING THE PUBLIC

Educating the public about comprehensive oral health care was also the predominant theme in the team working with issues of information. They also reintroduced the notion that "good commercialism" includes information that is complete and accurate. This applies equally to the relationship between dentists and patients, dentists and continuing education providers and industry, and between these groups and the research community. Obviously, this group would expect to see collaborations between organized dentistry and these groups in order to develop appropriate standards for full and meaningful exchange of information.

Conclusion

Although commercialism in dentistry has multiple facets — some healthy and others dangerous — it is possible to identify the basic patterns in the concept. The effect of these on oral health and the den-

tal profession can be understood, at least in broad terms. Based on this understanding, there are concrete steps the American Dental Association and its partners can take to tip the balance strongly in favor of good professional business practices.

The recommendations developed in the Ethics Summit on Commercialism workshop involving leaders from across the dental profession hold realistic promise for addressing the growing concern posed by negative commercialism. Perhaps what is most remarkable about the recommendations, aside from the fact that they are collaborative and doable, is their positive tone. Participants from the Ethics Summit on Commercialism quickly came together around the point that there is no wisdom in complaining and little to be gained in trying to stop others from doing what they think is in their best interests — even if mistaken. The core theme in the recommendations from Ethics Summit on Commercialism is that competent, comprehensive, and continuous oral health care is appropriate and should be promoted to the American public. It is believed that that message is more powerful than the message of commercialism. ■■■■

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High Anxiety

Dentists have not slept well since the last amalgam scare combined with the sterilization of handpieces to make insomniacs of us all.

Robert E. Horseman, DDS

ILLUSTRATION BY CHARLIE O. HAYWARD

Human behaviorists tell us that fear, guilt, greed, and lust are part and parcel of the human condition. It's amazing to me people can actually make a living divulging facts like this to the rest of us, but apparently this knowledge is essential to understand why politicians behave as they do, and to account for the obscene salaries paid to other people whose sole talent is tossing a ball through a hoop or hitting one with a stick.

While greed and lust would seem to be the operative factors here, *envy* might be another if you're on the outside looking in. It is more easily understood how fear and guilt contribute to the smooth running of society. This is what keeps you from testing a hot iron with your tongue or shoplifting a Skilsaw from Sears.

You would think every fear imaginable would have been documented by now and the antidotes disseminated so we could successfully avoid the consequences, but new fears are cropping up every day. Nowhere is this more evident than in our own profession where the media love to be the first to spread the alarm. Dentists have not slept well since the last amalgam scare combined with the sterilization of handpieces to make insomniacs of us all. Here are a few potential fears you may not have thought of:

THE RECEPTION ROOM — Have you read *all* the articles in the magazines in your reception room? What if one of them advocated overthrow of the government? Or recommended some cosmetic procedure that resulted in a less than satisfactory result? Fifty million trial lawyers are ready to hold you personally responsible for providing this material to unsuspecting patients.

"Where did you get the idea for moving your ears forward and your eyebrows up?"

"From my dentist's reception room!"

"And setting fire to the Pentagon?"

CONTINUES ON 81

DR. BOB, CONTINUED FROM 82

"Same place."

"The people rest!"

THE BUSINESS OFFICE — Chances are your business office is an ergonomic nightmare ready to inflict everything from carpal tunnel syndrome to a dowager's hump on your employees. This is a test of employee loyalty you cannot afford to take. It would be a mistake not to allot some of your nocturnal wakefulness to this area.

THE LABORATORY — The potential for fear generated by your lab is so immense, you should definitely dismantle it immediately and move it to some remote place, preferably in the next county. The same reasoning applies to wherever you keep your central vacuum and air compressor. All these things rely on a physical principle called "centrifugal force." Once unleashed, centrifugal force is capable like Hurricane Hugo of decimating everything in a 10-mile radius. You don't want to be there.

YOUR PRIVATE OFFICE — Private? Hah! Grand Central Station is private compared to your sanctum sanctorum. Unless you've installed a door that Chase Manhattan could be proud of, your office is as private as the Million Man March. Most of the sensitive material you harbor in your sanctuary is capable of spontaneous combustion due to laxity in federal regulations involving the corrupt paper industry.

Even though you may have difficulty yourself finding anything on or about your desk, bad people whom you would least suspect will have no problem at all extracting documents that could embarrass you or cause search warrants to be issued by judges antagonistic toward dentists. There is no soporific in the PDR strong enough to counter this.

THE OPERATORY — The operatory, by definition, should be the one place where you are in charge, as much in your element as a goldfish in its bowl. Wrong! Here's where air, water, electricity, vacuum, sharp things, corrosive things, radiation, and infection meet in a vortex of anxiety, apprehension, and resistance. It is true that over the years we've learned to cope with most of these fears to the point where our anxiety level is no higher than you might experience if accidentally buried alive, but the malady lingers on.

THE FUTURE — Managed care. This has been rightly classified as the Fear du Jour. Maybe it will go away. What are the odds? Will mercury fear go away? Will backflow? Will your ulcer? These concerns are expressed in *Horseman's Law of Balanced Inertia* as "For every moment of perceived tranquility, there is an equal and opposite moment of abject fear." It was on this fundamental axiom that dental societies were formed long ago. Ostensibly to further education and promote camaraderie amongst dentists, the real reason that dental societies continue to flourish is that they provide a forum to exchange mutual fears. There is nothing that allays the worries of a fellow practitioner as much as discovering he is not alone. It would be appropriate to stand before each meeting and sing a variation of that old song:

"... For your fears are my fears,
And my fears are your fears,
The more we get together,
The happier we'll be."

To further this concept, we should apply to *Mad Magazine* for permission to use Alfred E. Neuman as our mascot, diastema and all, and dump that pathetic little molar that is featured on too much of our literature. Our new motto would then be, "What, me worry?"

It's worth a shot. ■■■■