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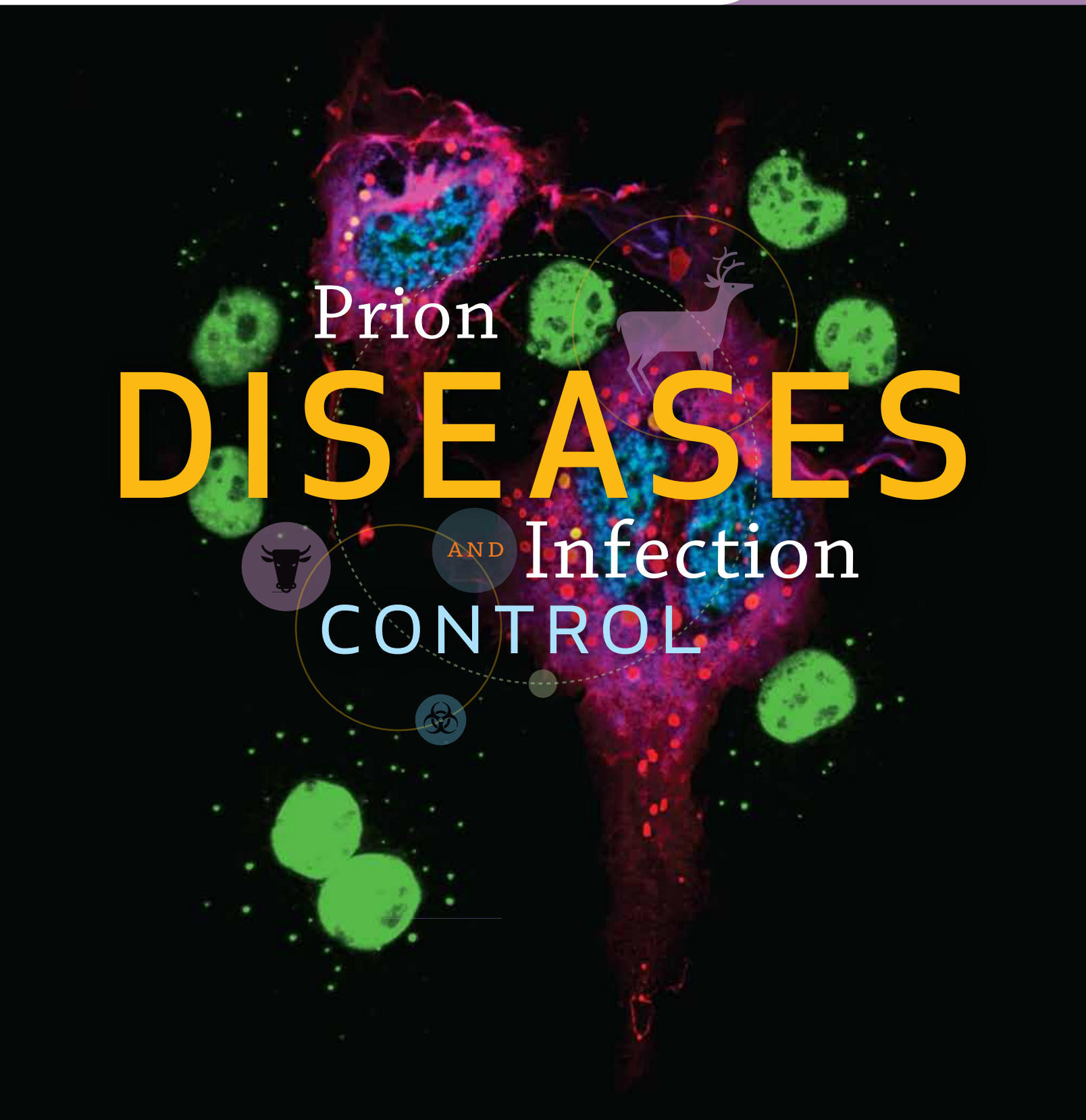

Prion DISEASES AND Infection CONTROL

OF THE CALIFORNIA DENTAL ASSOCIATION
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DEPARTMENTS

FEATURES

CDA JOURNAL, VOL 35, Nº1

The Power of One — Revisited

ALAN L. FELSENFELD, DDS

Last month I discussed the interrelationship of large organizations within dentistry in California and how, with cooperation, we can achieve good things for our profession. Collaborative efforts and common goals have had significant effect on the way we practice today. This is a pragmatic elaboration of the aphorism "In Unity There is Strength."

It is reasonable to believe that this is a reason for organized dentistry to exist. But what of the individual member? One may think that an individual can do little to have impact on our professional or personal lives on a global level. Not so.

It is not uncommon in the arguments over issues that we face as an organization or society that disparate views exist as to what is best for the body. Discussion, debate, and often political maneuvering are the rule for the completion of the work that will provide guidance for our lives. Many of us are privileged, through positions of leadership and representation, to have altered the course of dentistry in California throughout the years. This is not meant to be a tribute to the egos of our leadership, rather a demonstration of how one person with a goal can introduce a resolution, educate others in the preparation of policy statements or effectively debate, and achieve success in effecting change.

Most of us will never be the president or on the boards of the California Dental Association, the American Dental Association, or any other major association of our profession. That does not mean that as a single member we cannot be influential. We should all continue to participate, for it is important for us to give back as individuals.

One may think that an individual can do little to have impact on our professional or personal lives on a global level. Not so.

A good example of an individual's ability to effect change and influence the profession is in the recent enactment of a bill that mandates dental screenings in addition to medical evaluation of children before they enter the first grade. The concept for this bill came from a member pediatric dentist who believed that, given the magnitude of dental disease and the education days lost in the early years, preschool evaluation would benefit a population at risk. While it was not without a significant amount of debate, testimony, discussion and effort, the member was able to garner the support of the California Dental Association, who championed the bill through the Legislature. As the bill was signed, the member could feel proud of his impact on dental health in the state.

Even without that level of effort, a member may have significant impact on where the profession is headed. The election for the office of president-elect of the American Dental Association is by their House of Delegates — a body of more than 400 individual representative members. In 2005, the election was a contest with two individuals in pursuit of the office. Much to the surprise of the house, the first ballot ended in a tie with the second ballot ceding the election to the winner by just a few votes. Consider the power of the few delegates that altered their vote for the second ballot.

There are similar stories on the social political scene. As the municipal and state election season recently passed, and on a historically reasonably consistent basis, many of the contests necessitated recounts or were decided by a handful of voters.

As a member of organized dentistry, you are not required to participate to any extent other than being a dues-paying or "checkbook" member. In reality, most of our members fall into this category for a litany of reasons. Practices are too busy, family requirements prevent their participation, or they are involved in other nondental groups and cannot be involved with depth in dental policy making or political issues. Our organization can accept and encourage membership at this level. Clearly, if everyone participated at a maximum level, there might not be a need for a representative governing body, and such involvement on a grand scale might engender a degree of chaos or confusion in getting anything done.

But for those who do elect to participate, the achievements absolutely outweigh the frustrations. To see your idea develop and be enacted gives one a sense of accomplishment that is difficult to measure. To influence the day-to-day

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activities of your professional life provides a sense of self-actualization that is important for many of us.

That you think it or say it does not make it so. But, if you have the conviction of your beliefs and can argue, prove, substantiate, negotiate, articulate, or convince others then, you can be very successful in being a catalyst for change at the local, state, or national level. An individual who is properly motivated and willing to put in an effort to effect change can make it happen. When it does, there is no greater feeling of success than the legacy you leave or the mark you make on your profession or community. The costs of doing so are great in terms of time, money, and frustration. The rewards are greater. ■■■■

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

Dan Hubig

Toothpaste Tech: What's the Latest News in Toothpaste?

BY DEBRA BELT

With all the advances in oral health care, it's easy for the humble toothpaste to get lost in the barrage of new technology and products. Yet, the item millions of people reach for every day makes strides and keeps pace with an increasing number of offerings and claims.

When it comes to variety, there is no shortage of options in the toothpaste aisle. ConsumerSearch.com reports that Colgate now has approximately 49 varieties of toothpaste, and Crest has 21 varieties in 24 different flavors. In sorting through all the claims made by various manufacturers, cavity prevention, plaque reduction, and fresh breath are long-touted benefits. Whitening, low abrasiveness, and sensitivity reduction are also

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COSMETEMP
by Cosmedent

Cosmedent introduces COSMETEMP, a vastly improved temporary crown and bridge material. COSMETEMP features all the necessary qualities of an ideal temporary material including excellent handling properties, a unique formula that delivers high strength and natural esthetics, and an easy dispensing and retrieving system to eliminate unnecessary hassles and cleanup. Because COSMETEMP marginates beautifully and trims and polishes easily, patients are delighted with the esthetic results that look and feel like natural teeth. When cured, COSMETEMP has virtually no oxygen-inhibited layer. Additional features include a rapid two-minute set time, fluorescence, excellent gingival compatibility and shades that are color-matched to the Vita shade guide and the Renamel Restorative System. COSMETEMP comes in four shades and is dispensed in any 4:1 or 10:1 mixing gun. For more information regarding COSMETEMP, call 800-621-6729 or visit www.cosmedent.com.

A New Look, a New Feeling

This month the *Journal of the California Dental Association* debuts its new look. It is the first redesign of the *Journal* in 10 years, and a visual representation of CDA's desire to stay up-to-date with a changing world. But the changes go beyond the surface.

As you may know, CDA has recently undergone a significant "rebranding" effort to become more relevant to modern dentistry and our members. This effort includes an update of the CDA look as manifested by its logo, Web site and letterhead, and, more importantly, a new approach to the services it provides to members at all stages of practice and the integration of those services with all the CDA companies, including The Dentists Insurance Company and TDIC Insurance Solutions.

Along those lines, the *CDA Journal* has been retooled to reflect these concepts and is making changes to satisfy the interests of its readers. One of those changes can be seen on this page. Our members have expressed high levels of interest in new products and technologies, and they will now be featured prominently in the Impressions section, rather than toward the back of the issue. The *Journal* also is providing a greater variety of scientific content. In addition, this year, TDIC will begin providing its well-regarded Risk Management Case Studies within the *Journal's* pages. This is only the beginning of CDA and the *Journal's* efforts to serve as a significant resource to your practice of dentistry.

We're eager to hear your comments and suggestions. Please e-mail them to me at alan.felsenfeld@cda.org.

ALAN L. FELSENFELD, DDS, EDITOR

"The ADA believes scientific and clinical information should be easier to access and more relevant to dental practitioners."

DAVID C. SARRETT, DMD, MS

New Dental Product Review Publication

Dentists seeking the inside track on products used in patient care literally have a new resource at their fingertips.

The American Dental Association's *Professional Product Review*, a quarterly newsletter, is aimed at assisting dentists with product selection for their practices.

"The ADA believes scientific and clinical information should be easier to access and more relevant to dental practitioners," said David C. Sarrett, DMD, MS, review editor. "We think the ADA *Professional Product Review* does just that, by providing comprehensive dental product information that is unbiased, scientifically sound, clinically relevant, and user-friendly."

The publication, which is free of charge to ADA members and available by subscription to nonmembers, will generally feature three professional dental product categories per issue and include both clinical and laboratory evaluation of numerous products within each category. Some issues will include additional data such as buyer's checklists, expert panel discussions, and technology updates.

"The *Professional Product Review* represents a fundamental change in how the ADA evaluates dental products and communicates those results," said Sarrett. "A unique feature of the PPR is that input comes from the professional opinions and clinical experiences of practicing dentists."

Product selection is based on input from dentists who volunteer as members of the ADA Clinical Evaluator Panel. Panel members respond to product evaluation surveys and participate in panel discussions and interviews. Their feedback appears in the newsletter and online at the review Web site, www.ada.org/goto/ppr.

Posture, Airway and the Tongue in Clinical Dentistry ↓

The new publication Posture, Airway and the Tongue in Clinical Dentistry, authored by Dr. Robert Jankelson, covers the etiology and management of upper airway problems commonly encountered in the dental practice. Impact of body posture and the tongue on occlusion are also covered. Restricted upper airway during facial development is perhaps the single, largest cause of dentofacial abnormalities and malocclusion. No other single factor has such an impact as upper airway obstruction in the young patient. The earlier the condition is treated, the easier the correction is because of the elastic nature of the tissues. Understanding airway restriction can result in more accurate diagnosis and elimination of sometimes unexplained problems in treatment outcome. For more information on this new publication, please go to www.myotronics.com or call 800-426-0316.

Dentaltown and Dentalcompare Partner Up to Offer Product Directory

Dentalcompare, an online buyer's guide for dental professionals, recently announced its partnership with Farran Media, parent company of Dentaltown and Hygienetown.

"Our alliance with Farran Media demonstrates our commitment to furnish the best possible service to dental professionals," said Matthew McLean, chief operating officer of Dentalcompare. "We believe in empowering dental professionals by providing thorough product information and editorial. By expanding our audience to the Dentaltown and Hygienetown visitors, we will be able to help an unprecedented number of dentists make informed purchasing decisions and find the best products for their practice."

Farran Media offers dental professionals an opportunity to share and learn experiences through free online message board communities and forums, as well as press releases, personal referrals, and online continuing education.

The partnership allows Dentaltown and Hygienetown users to access Dentalcompare's unbiased product reviews, directory, and technology directly from their Web sites and magazines. Visitors accessing Dentalcompare product information through Dentaltown or Hygienetown can search and compare products without leaving the respective Web sites. Dentalcompare's editorial content also will be searchable directly from the Dentaltown or Hygienetown Web sites and featured in product spotlights in the corresponding magazines.

UPCOMING MEETINGS

2007	
April 15-21	United States Dental Tennis Association, Sarasota, FL, www.dentaltennis.org.
April 17-21	American Academy of Oral Medicine Annual Meeting, San Diego, www.aaom.com.
May 3-6	CDA Spring Scientific Session, Anaheim, (866) CDA-MEMBER (232-6362).
June 27-July 1	Academy of General Dentistry Annual Session, San Diego Convention Center, (888) 243-3368.
Sept. 27-30	American Dental Association 148th Annual Session, San Francisco, www.ada.org.
Nov. 27-Dec. 1	American Academy of Oral and Maxillofacial Radiology 58th Annual Session, Chicago, www.aaomr.org.
2008	
May 1-4	CDA Spring Scientific Session, Anaheim, (866) CDA-MEMBER (232-6362).
Sept. 12-14	CDA Fall Scientific Session, San Francisco, (866) CDA-MEMBER (232-6362).
Oct. 16-19	American Dental Association 149th Annual Session, San Antonio, Texas, www.ada.org.

To have an event included on this list of nonprofit association 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.

D-Caries Mini →

Neks Technologies introduces the D-Carie Mini, a new pen-sized device designed to make the process of locating and diagnosing even the tiniest caries simple, fast, and accurate. The D-Carie Mini, recently available in the United States is a lightweight, cordless device that uses fiber optic technology and light-emitting diodes to accurately detect both occlusal and interproximal caries—even when lesions are in their earliest stages. When used as a diagnostic aid in conjunction with an X-ray, the Neks D-Caries Mini allows dentists to assess a third dimension, the volume of caries, prior to opening the tooth. The device also provides dentists with an option for examining and diagnosing children, pregnant women, and patients who prefer to forgo X-rays or limit their exposure to them for health or personal reasons. For more information, call 800-873-7683 or visit www.pattersondental.com.

Researchers: Certain Caregivers Are at Potential Risk for Periodontal Disease

According to a new study published in the *Journal of Periodontology*, caregivers of people under physical or psychological stress, including those with the conditions themselves, should not overlook their oral health.

The results suggested that being a caregiver to relatives with hypercortisolemia, dementia, or stress were associated with increased gingival bleeding and higher plaque levels. The study examined adults 50 years and older.

"We found that short-term psychological stress was a risk indicator to elevated plaque levels, and long-term physical stress was a risk indicator to gingivitis," said Fernando N. Hugo, DDS, on the faculty of Dentistry of Piracicaba, Brazil. "These findings support the health impact of psychosocial risk factors from chronic stress, which may lead to malfunction of some biological functions."

Research indicated that the demanding task of care giving, typically associated with increased stress, may also be a risk factor for poor oral hygiene. These findings pointed out that stress may contribute to a disinterest in performing oral hygiene.

"Flossing and brushing the teeth and gums had a protective effect against plaque and gingivitis," said Kenneth A. Krebs, DMD, American Academy of Periodontology president. "That said, future research is needed to explore the relationship between stress and oral hygiene negligence."

Two-hundred thirty people were evaluated in the study, with nearly 52 percent as caregivers. The caregivers of dementia patients were examined because they represent a well-known group suffering from the impacts of chronic stress on human health and immune functions. The results were among the first in literature to suggest that caregivers of relatives with dementia are at risk of having more plaque and gingivitis than noncaregivers.

TOOTHPASTE, CONTINUED FROM 9

Dan Hubig

frequent claims. More recent claims to hit the market include anti-bacterial and anti-inflammatory action, and a patented salivary enzyme system.

Of all the benefits claimed by toothpaste manufacturers, whitening receives a lion's share of the attention. When the editors of *Consumer Reports* tested 41 varieties of toothpaste earlier this year, they identified whitening as the most prominent claim. They also said whitening presents "something of a gray area" with the fine print on products promising to whiten teeth by removing stains, not by lightening the base color of the teeth.

The editors of *Consumer Reports* noted that only products bearing the ADA Seal have had appropriate clinical and/or laboratory studies, and scientific data reviewed by the ADA Council on Scientific Affairs.

Consumer Reports found "no correlation between whitening claims and stain-removing ability" even with the seven toothpastes they tested that contained peroxide. The testers favored Ultrabrite All in One Advanced Whitening, reporting that it "proved excellent at stain removal — and with only average abrasiveness." The two closest competitors in stain removal, according to *Consumer Reports*, were Colgate Max Fresh and Colgate Luminous.

Beyond whitening, there are brands bearing the ADA Seal that offer a combination of benefits.

Crest Pro-Health has a trademark Polyfluorite System containing stannous fluoride and sodium hexametaphosphate. Product research for Pro-Health includes more than 70 publications and research presentations. The ADA Council on Scientific Affairs' acceptance of Crest Pro-Health toothpaste is based on its finding that the product is effective in helping to prevent and reduce tooth decay, gingivitis, and plaque above the gumline, to relieve sensitivity in otherwise normal teeth, and to whiten teeth by removing surface stains, when used as directed.

Colgate Total also carries the ADA Seal and contains triclosan, a patented copolymer that fights bacteria and oral inflammation implicated in periodontal disease. Colgate Total has been studied in more than 10,000 patients. Colgate pioneers the claim that the toothpaste can "break the inflammation cascade" of plaque bacteria and gingival inflammation "associated with disease throughout the body."

Colgate Total Whitening Gel received a mention in the *Consumer Reports* testing where the editors said it was "the only toothpaste we tested that's ADA-accepted for plaque and gingivitis prevention."

Among innovative toothpaste claims is Biotène's patented LP3 salivary enzyme system recommended for dry mouth treatment. Biotène claims that the toothpaste works like the body's natural defenses to fight cavities, periodontal disease, and oral infections due to dry mouth. The toothpaste contains three primary enzymes: glucose oxidase, lactoperoxidase, and lysozyme, which Biotène reports to function in boosting and replenishing saliva's own antibacterial defenses.

Biotène toothpaste is also made with xylitol, a five-carbon sugar alcohol that is used as a sugar substitute and can be extracted from birch, raspberries, plums, and corn. The makers report that xylitol inhibits the growth of pathogenic bacteria because bacteria cannot use it to grow and metabolize.

Biotène also promotes the lack of sodium lauryl sulfate in its products, citing recent reports that some people get fewer canker sores when switching to a toothpaste that does not contain sodium lauryl sulfate. Research has also speculated that sodium lauryl sulfate dries out the protective mucous lining in the mouth, making it vulnerable to irritants.

Somewhat in the shadow of pioneering claims are more routine claims such as desensitizing toothpastes. The ADA recognizes two effective ingredients in treating sensitive teeth and gums: strontium chloride and potassium nitrate. These "block the tube-like channels that pass through teeth and connect to nerves," thereby reducing "the ability of the nerves to transmit pain," according to the ADA. Crest Sensitivity Original Formula Maximum Strength was recommended by *Consumer Reports*, based on the product's ADA Seal.

Earlier this year, Biotène launched Biotène Sensitive toothpaste, which contains potassium nitrate combined with Biotène's LP3 salivary enzyme system. Biotène also promotes the taste of its product based on the ability of xylitol to cover the strong taste of potassium nitrate.

Low abrasiveness is a little heralded claim that considers to what extent a toothpaste scrapes away at the dentin layer under the enamel and gumline. Abrasives are usually in the form of silica and are useful for removing plaque. All ADA-approved toothpastes contain mild abrasives; heavily abrasive toothpastes are not recommended as they can cause gum recession and damage to tooth enamel.

Donated Textbooks Will Help Cambodia's New Dentists

A concerted effort to rebuild Cambodia's oral health infrastructure involves a call for used dental school textbooks.

Robert P. Renner, DDS, who recently volunteered his services in Cambodia to treat the "street children," wrote about his experience in the August 2006 issue of the *Suffolk Dental Bulletin*, the publication of the Suffolk County (New York) Dental Society. The article also emphasized the need for used dental school textbooks for the country's dental students.

In the 1970s, approximately 1 million people were killed in genocidal social experiments. The Khmer Rouge had effectively eliminated the professional classes. At the close of the 1990s, there were only 35 dentists in a country of 10 million people.

Those who wish to donate books (English-language books will be accepted), should send them to Dr. Heng Sopanha, University of Health Sciences, Faculty of Odonto-Stomatology, 73 Monivong Blvd., Sangkat sras Chak Khan Duan Pehn, Phnom-Penh, The Kingdom of Cambodia.

ADA Develops Practice-Related Podcasts

In response to an increasing demand for information through avant-garde tools, the American Dental Association has produced a series of practice-related podcasts, which are available for free download at www.ada.org/goto/podcasts.

The podcasts are each about 20 minutes long and feature one or more subject matter experts and a moderator. While primarily targeting new dentists, other practitioners will find the podcasts pertinent and useful. Podcasts can be found in major directories such as Odeo, iTunes, and Podfeed.net. The ADA's podcast feed is managed by Feedburner. Listeners can subscribe to the podcast, save it to their hard drives to listen later on their digital mp3 player, iPod or PC, or stream it directly from ada.org.

Newcomers to podcasts should view the ADA's Podcast frequently asked questions site at www.ada.org/goto/podcast. Additionally, listeners are encouraged to share their thoughts on the podcasts by going to the same Web site and clicking on "What do you think?" to provide feedback.

Podcast topics include "Internet Marketing"; "Finding and Keeping the Patient"; "Strategic Planning and Systems Development" (part 1); and "Taxes and Business Planning" (part 2).

Listeners can subscribe to the podcast, save it to their hard drives to listen later on their digital mp3 player, iPod or PC, or stream it directly from ada.org.

Honors and Awards

Paul Subar, DDS

Peter K. Moy, DMD

Paul Subar, DDS, has been named the first recipient of the California Dental Association's Arthur A. Dugoni Faculty Award. This award will annually recognize an educator from the state's five dental schools who has made contributions to the scholarly and creative activities of his or her respective school. Subar, of Mill Valley, is an assistant professor at University of the Pacific Arthur A. Dugoni School of Dentistry.

Peter K. Moy, DMD, Los Angeles, has been installed as treasurer of the Academy of Osseointegration. Moy is director of implant dentistry, Department of Oral and Maxillofacial Surgery, and professor at the University of California, Los Angeles, School of Dentistry, a faculty member of the UCLA Hospital and Clinic's Department of Hospital Dentistry. He also maintains a private practice limited to oral and maxillofacial surgery in Brentwood.

The University of California, Los Angeles, School of Dentistry has received a pledge of $2.5 million from Nobel Biocare. The school's largest, single endowment to date, it will be used to establish the Nobel Biocare Endowed Chair in Surgical Implant Dentistry.

"This endowment is a very important and exciting development for the section of oral and maxillofacial surgery, the division of diagnostic and surgical sciences, and the school as a whole," said No-Hee Park, DMD, PhD, dean of the UCLA School of Dentistry. "The Nobel Biocare Endowed Chair in Surgical Implant Dentistry is a wonderful way to advance the teaching and research activities of a leading scholar while helping to strengthen UCLA's standing at the forefront of dental education."

"We are extremely grateful for Nobel Biocare's generous support, which will benefit the UCLA School of Dentistry's students, faculty and patients for many generations to come," said Thomas Mitchell, assistant dean and director of development at the UCLA School of Dentistry.

No-Hee Park, DMD, PhD

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Prions

Transmissible Spongiform Encephalopathies and Dental Transmission Risk Assessment

BY JANYCE HAMILTON

AUTHOR

Janyce Hamilton is a freelance writer working out of Naperville, Ill. Her previous articles for the *Journal of the California Dental Association* are "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 Ties Taking Toll," and the "Dental Implications of the Human Genome Project."

ACKNOWLEDGMENTS

The author gratefully acknowledges the research assistance of Ruth Schultz at the Library of the American Dental Association in Chicago; editorial comments of Heather Larson of Phoenix and Ermias Belay, MD, of the national Centers for Disease Control and Prevention in Atlanta; and reviewer comments by James Mastrianni, MD, PhD, at the Prion Laboratory, University of Chicago.

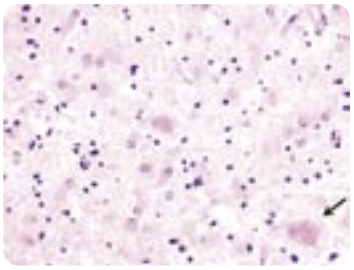
Are instruments used on the dental patient with possible variant Creutzfeldt-Jakob disease a theoretical remote risk of little concern, or is the unknown potential transmissibility one that warrants disposal or a prion-inactivating regimen for contaminated instruments?

No test. No cure. No foolproof disinfection. No wonder surgeons feel uneasy about prion diseases or bristle when asked about preventing "mad cow" risk.

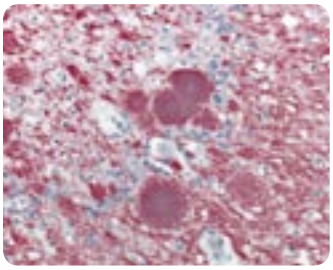
The national Centers for Disease Control and Prevention wants dentists and oral and maxillofacial surgeons to know that when it comes to minimizing risks of a transmissible spongiform encephalopathy (or "prion disease"), which is known in humans as Creutzfeldt-Jakob disease, there is nothing different they need to be doing in 2007 than, say, 2003. That's the year of the landmark study by Kohn and colleagues, "Guidelines for Infection Control in Dental Health-Care Settings — 2003."[1] The guidelines draw upon the World Health Organization's important report, "WHO Infection Control Guidelines for Transmissible Spongiform Encephalopathies: Report of a WHO Consultation, Geneva, Switzerland, 23-26 March 1999" for examples of sterilization.[2] The precautions in these documents would be pertinent for the known Creutzfeldt-Jakob disease (CJD) patient, or during major dental procedures where neurovascular tissues may be exposed, including using single-use items (needles, anesthetic cartridges); disposing of hard-to-clean equipment after one use (files, broaches, burs); avoiding flash sterilization; keeping instruments moist so tissues don't dry before cleaning and sterilization; and using one of WHO's transmissible spongiform encephalopathy sterilization methods.[2]

In 2006, a new set of WHO transmissible spongiform encephalopathy guidelines on tissue infectivity was released, which confirmed the first human-to-human transmission route via blood transfusion.[3] This development points to a previously unmapped way in which prion diseases have been spreading. Reportedly, the British Dental Association is going to release revised guidance based on a 2006 Spongiform Encephalopathy Advisory Committee Position Statement on variant CJD and endodontic dentistry[4] to update the use and decontamination of dental instruments.[5] Concerns over this issue arose well before a study of endodontic files using high magnification, postclinical use, and subsequent decontamination found 75 percent had residual adherent biomass.[6]

This article looks at the complex



Histopathologic changes in frontal cerebral cortex of the patient who died of variant Creutzfeldt-Jakob disease in the United States. Marked astroglial reaction is shown, occasionally with relatively large florid plaques surrounded by vacuoles (arrow in inset) (hematoxylin and eosin stain, original magnification x 40).



Immunohistochemical staining of cerebellar tissue of the patient who died of variant Creutzfeldt-Jakob disease in the United States. Stained amyloid plaques are shown with surrounding deposits of abnormal prion protein (immunoalkaline phosphatase stain, napthol fast red substrate with light hematoxylin counterstain; original magnification x 158).

questions around the implications of emerging data on the abnormal prion protein (PrP^{TSE}) and infection control during hospital-based procedures as well as dental and oral and maxillofacial surgeries. Reviewed is the literature from a PubMed search of dental journals since 1999 using keywords "prion diseases."[1,4,6-24] The aim of the article is to summarize data for weighing potential prion transmission risks, so dentists can decide how to approach infection control.

The problem: a menacing family of diseases. On the English countryside, black cattle dot the hillsides, munching away. The story begins here, where a family of transmissible spongiform encephalopathies first showed up in these cows. Bovine spongiform encephalopathy afflicted the cattle that were fed a supplement of bone and tissue meal rendered from processing plants disposing of cattle, goat, sheep, and other carcasses. This disease, which is known as "scrapie" in sheep and goats and chronic wasting disease in deer, has several human forms, including CJD, fatal insomnia, and Gerstmann-Straussler Scheinker disease. Prion diseases have been described in the medical literature for more than 150 years, but no one understood what they were. Molecularly, neither suspicious viruses nor bacteria were in found ante- or postmortem assays. All people said is that formerly gentle animals and level-headed people stared into space more, bumped into things, walked wobbly, lost their appetite, attacked others unprovoked, and generally acted a tad crazy.

The biological villain. American Carleton Gajdusek studied and reported on kuru, a brain wasting death in tribes who consumed human tissues as part of burial rituals. William Hadlow, a veterinary researcher, noted that the pathology of kuru was the same as scrapie in sheep. He suggested to Gajdusek that he try to transmit kuru, since Hadlow knew scrapie was transmissible. Gajdusek did this in the 1960s — kuru was transmitted to chimpanzees. Thus, Gajdusek is credited with making the connection between sheep scrapie and kuru, and was a recipient of the 1976 Nobel Prize in Medicine for this work.

In 1972, Stanley Prusiner, MD, searched for a biological commonality shared by scrapie in animals and kuru in humans. In 1982, he published a report on an unusual folded protein — termed a "prion" that was thought to be connected. Of the 30,000 or so different proteins in human blood, this one was acting independently. It seemed this type of prion sent a signal to surrounding proteins, which "listened," as they'd soon conform their structures and fold too. Interestingly, the manifestation of the disease didn't seem to occur until much later, sometimes decades.

The growing numbers of PrP^{TSE} converts lurk silently, incubating before starting mass foldings within otherwise healthy-acting animals and people. Scientists are discovering bacteria communicate by releasing signaling molecules — a process known as "quorum sensing." It is unknown if this is similar to the activity of prions.

When masses of prions result, clumped together by their sticky surfaces, they travel throughout the body. How they are transported in the body is uncertain, but the lymporeticular system has been suggested. The favorite stop for the traveling prions is the brain. Evidence of the occupation of the brain by prions is not stains but sponge holes, as if "eaten." The cascading neurological impact of rapidly increasing numbers and sizes of holes makes the brain look like coral, which alters mental and physical function rapidly,

Wasting disease in deer is known as 'scrapie' in sheep and goats. The human forms of this disease include CJD and fatal insomnia.

driving the afflicted "mad" until death. Sometimes a patient lives a few years, but it's not unusual for death to occur in as little as a few months or even weeks.

PrP^{TSE} diehard superpowers. The prion that causes CJD is unlike what most infection control manuals have addressed; you can't seem to kill a prion with standard disinfecting techniques used on more fragile viruses such as HIV, hepatitis, or TB bacterium.

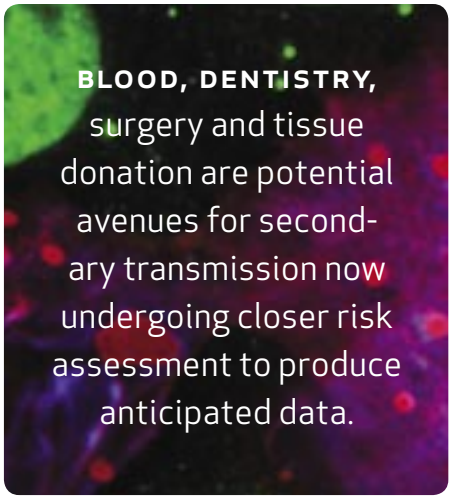
In late 2006, researchers meekly reported that they boiled and baked prions with exceedingly long and high temperatures, yet tests still detected the faint "signaling" of prions still active on the surface of stainless steel instruments.

PrP^{TSE} is a cyborg — as no biological proteinase can touch it. Proteinases, which usually eat abnormal proteins, can't seem to digest the prion, which goes about its business mechanically destroying its human host impervious to the usual macrophages and other biosoldiers of defense.

Types of human prion disease. The data for solving the mysteries of prion diseases is scanty, and most are in animal models. There are incidence statistics medical epidemiologists have collected about the three known types of transmissible spongiform encephalopathies in humans:

- Sporadic CJD (80-85 percent) (average age at death 67 worldwide[3] or 68 in the United States[25]) — spontaneous cases of unknown cause;
- Familial CJD (10-15 percent) — genetic origin;
- Variant CJD (4 percent) — least in reported numbers but not least feared is this newest form. It afflicts younger populations (average age 28[26]). vCJD traumatized the British population, mainly transmitted from an innocuous activity: eating beef unknowingly infected with bovine spongiform encephalopathy. There were more than 184,453 U.K. cases of bovine spongiform encephalopathy in cows as of Sept. 30, 2006.[27]

While vCJD — the mad cow type — is the disease that has highest infectivity risk, all types could theoretically be transmitted through direct brain, blood, or tissue transplant/transfusion, which is why no close family relative with prion disease should be a tissue, blood, or organ donor. The thousands of sporadic cases in the past few decades are theorized, but unproven, as a cell's random error — a genetic triggering that mistranslates normal to warped protein. Less popular with public health scientists are the unproven theories published that a portion of sporadic cases should be investigated more carefully to look at iatrogenic or person-to-person etiology.[28] The United States is not the United Kingdom, but American epidemiologists do look there for clues to worst-case scenarios that could come to pass on this side of the pond. The peak number of U.K. vCJD deaths in one year was 28; in 2006, five vCJD cases were counted (as of Nov. 22).[26] The total number of children diagnosed with vCJD in the United Kingdom is four definite and two probable, the youngest age 12.[29]



vCJD British Cases Raise Questions of 'Subclinical Carriers' and 'Self-Sustaining Epidemic'

Blood, dentistry, surgery, and tissue donation are potential avenues for secondary transmission now undergoing closer risk assessment to produce anticipated data.[26,30] For example, a study of 100,000 tonsils from the National Anonymous Tonsil Archive begins in 2007 in the United Kingdom to test for abnormal prions.[31] The U.K.'s Spongiform Encephalopathy Advisory Committee strongly recommends this testing progress with "all possible urgency."[26] Imagine the epidemiological quagmire that would ensue should any of the potential 5,000 subclinical vCJD carriers donate blood or share their prion disease by infecting instruments even after a current universal sterilization regimen. These newly exposed patients themselves become vectors, and on it goes.

It's the "what-ifs" that drive the Spongiform Encephalopathy Advisory Committee epidemiology subgroup to wonder whether instituting attributable rather than anonymous tissue testing would be a valuable public health tool so that routes of transmission could be understood, and further spread stemmed.[26] The proposed pragmatics are likely to be contentious in discussions, however. Studies have shown people with familial cases, such as those with a family predilection for Huntington's disease, prefer not to know their own carrier status.

The knowledge of an increased risk of a lurking biological killer infecting the brain stem for a neurological stranglehold doesn't sit well. Today, learning you have HIV or many cancers — which have treatments that can

prolong and even save your life — isn't good news, but at least you can try to do *something*. For CJD exposures, should they turn into clinical manifesting disease, there is no treatment.

For oral health clinicians, two principles should be kept in mind when reading the animal studies: 1) not every PrP^{TSE} exposure will infect you with CJD and 2) it is sensible to minimize all potential opportunities for human exposure. In animal studies, during a single time point in the incubation or clinical illness, bovine spongiform encephalopathy infectivity has been found in sternal bone marrow and palatine tonsil.[3] Meanwhile, sheep blood has infectivity during the "silent portion" of the prion affliction, as does cow brain.

While a cosmetic case has not been reported, tell the Botox crowd that full, pouty lips are courtesy of injectable bovine-sourced poison and they'd frown if they could.[32] The 2006 WHO report addressed such varied concerns as minimizing transmissible spongiform encephalopathy risk in vaccines, many produced with delicate animal-sourced organisms that would be destroyed by harsh purification/extraction methods; and gelatin used in food and vitamins should pose little risk after processing as long as cow skulls and vertebrae are excluded; milk is considered noninfectious if from healthy cows "fit for human consumption."[3] Keep in mind that the oral route of administering PrP^{TSE} is inefficient — no matter the level of the food infectivity consumed; while blood transfusion, especially for those with hemophilia, is efficient even in low titer infected blood or clotting factor.[33]

For tissue infectivity concerns during oral surgical procedures in the United States, William G. Kohn, DDS, from the CDC, said there has been "nothing related to dental tissues, not root canals, tooth extractions, or periodontal

TABLE 1

High, Low, or Nondetectable TSE Infectivity By Tissue*†

Tissue	One or more Human TSEs Infectivity Shown	
	Yes	No
HIGH TITER INFECTIVITY TISSUES		
Brain	Yes	
Spinal cord	Yes	
Retina	Yes	
Optic nerve	Unknown‡	
Spinal ganglia	Yes	
Tigeminal ganglia	Yes	
Pituitary gland	Yes	
Dura mater	Yes	
LOW TITER INFECTIVITY TISSUES		
Peripheral nerves	Yes	
Enteric plexuses	Unknown	
Spleen	Yes	
Lymph nodes	Yes	
Tonsil	Yes	
Thymus	Unknown	
Esophagus	Unknown	
Stomach/abomasum	Unknown	
Duodenum	Unknown	
Jejunum	Unknown	
Ileum	Unknown	
Appendix	Unknown	
Large intestine	Yes	
Placenta	Unlikely (report unconfirmed)	
Lung	Yes	
Liver	Yes	
Kidney	Yes	
Adrenal		None yet, more testing needed
Pancreas	Unknown	
Bone marrow		Preliminary data, more testing needed
Skeletal muscle		Preliminary data, more testing needed
Tongue	Unknown	
Blood vessels	Unknown	
Nasal mucosa	Unknown	
Salivary gland	Unknown	
Cornea	Yes	
CSF	Yes	
Blood	Yes	

TABLE 1 CONTINUED

High, Low, or Nondetectable TSE Infectivity By Tissue*† *(continued)*

Tissue	One or more Human TSEs Infectivity Shown	
	Yes	No
NO DETECTABLE INFECTIVITY TISSUES		
Testis		Preliminary data, more testing needed
Prostate/epididymis/ seminal vesicle		Preliminary data, more testing needed
Semen		Preliminary data, more testing needed
Ovary	Unknown	
Uterus (non-gravid)	Unknown	
Placenta fluids		Preliminary data, more testing needed
Fetus	Unknown	
Embryos	Unknown	
Bone	Unknown	
Heart/pericardium		Some data, more desired
Tendon	Unknown	
Gingivae		Some data, more desired
Dental pulp	Unknown	
Trachea	Unknown	
Skin	Unknown	
Adipose tissue		Preliminary data, more testing needed
Thyroid gland		Preliminary data, more testing needed
Mammary gland/udder	Untested	
Milk		Preliminary data, more testing needed
Colostrum		Preliminary data, more testing needed
Cord blood		Preliminary data, more testing needed
Saliva		Some data, more desired
Sweat		Some data, more desired
Tears		Some data, more desired
Nasal mucus		Some data, more desired
Bile	Unknown	
Urine		Some data, more desired
Feces		Some data, more desired

* Table condensed and adapted from Tables 1A-C, Annex 1, 2006 WHO Guidelines[3] and 2003 WHO Guidelines.[34]

† Noninfectivity does not mean PrP^{TSE} were not present.

‡ Unknown because not yet tested for infectivity.

bone grafting procedures." (See **TABLE 1**.) Not in human studies, but there is hamster scrapie transmission dentally.[9]

Iatrogenic CJD (1 percent) has occurred when the disease was transmitted by transfusion with infected blood (three vCJD cases), and transplanted with infected tissues (human dura mater allografts, corneas) or organs or on surgical tools (neurosurgical) and equipment (EEG electrode). In a study by Gibbs and colleagues, electrode probes used in a middle-aged woman who had CJD that were cleaned and sterilized accidentally transmitted CJD in two younger patients.[35] More than two years passed, and the electrode probes were implanted in the cortex of a chimpanzee. While they had been cleaned thrice, and sterilized in ethanol and formaldehyde vapor repeatedly, the chimp got sick with CJD 18 months later.

More than 362 cases of iatrogenic CJD transmission have been counted worldwide.[12] No bone-derived graft tissues for periodontal repair or jaw buildup have transmitted CJD. Chewing (surgical bone augmentation), walking (hip replacement), and seeing (cornea implants), and extending life (bone marrow transplant) are all case-by-case risk-benefit proposition that involve demographic risk and manufacturers' safeguards.

Controversy Doesn't Die With Its Victims

Those left behind may never be satisfied with the official explanations when a family member dies of CJD. Heather Larson of Phoenix has first-hand knowledge of the familial genetic form of CJD. Her mom lived in Phoenix and worked at home. Her symptoms were classic for CJD but add nausea, hallucinations, and onset of incontinence. Heather's mom died at age 56 of familial CJD in a matter of weeks — not years — after

being diagnosed. Afterward, Heather said, the problems were far from over.

"I worked with the National Prion Disease Pathology Surveillance Center to arrange the autopsy as my mother died. It took several days of phone calls, but the [center] called me and said they had finally found someone who would do an autopsy on my mother — in New Mexico. I wanted to identify her body once it was back in Phoenix and before it was cremated. I stood alone in that room and looked at her body in a white bag marked 'biohazard.'"

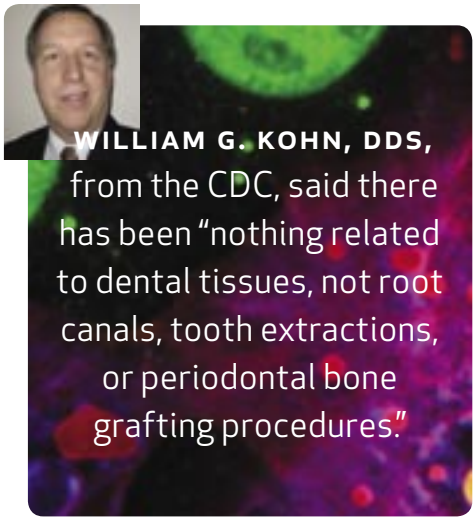
Months later, her mom's dentist called to remind her it was time for a cleaning. "I had to tell them that she was deceased. They asked how she died, and I said 'CJD — the human form of mad cow disease' and they quickly got off the phone."

"As for myself, I don't know if it would even be something to bring up for fear of being discriminated against, however, I wouldn't want to infect anyone either. There is a 50/50 chance that I carry the gene too. I haven't gotten tested. I don't see any benefit."

The British Dental Association has urged dentists not to discriminate against suspected CJD patients. England's chief dental officer of the Department of Health mailed his dental colleagues a letter in 2005 saying nothing special need be done beyond the satisfactory standards of decontamination for all patients for the CJD symptomatic or "at-risk for CJD for public health purposes" dental patient, as long as treatment doesn't include surgery.[36]

Blood Supply Concerns

No patient with a clotting disorder has ever been reported as contracting vCJD — including those in the United Kingdom where the risk has been highest. That is perhaps the most encouraging news of all, which should allay some the fears over bloodborne vCJD risks. Efforts in medical product manufacturing are believed to reduce or eliminate most risk should a vCJD-infected donor unknowingly give plasma. While it's easy to let a phrase like "it is still hypothetically possible that a person getting a blood transfusion or clotting agent can be exposed to the agent that causes vCJD if the donor(s) were incubating the disease," the lack of a single case says something. This is precisely what U.S. government public health officials want us to keep in mind.



The Department of Health and Human Services Administration, including the Food and Drug Administration, CDC, the National Institutes of Health, and the Office of Public Health and Science, released a joint statement Nov. 27, 2006, applicable to those with blood clotting disorders visiting dentists:

"At this time the U.S. Public Health Service does not believe there is a need for pdFVIII recipients to inform their surgeons or dentists about the recipient's potential exposure to vCJD. Also, there is no recommendation for surgeons and dentists to take any special precautions based on such potential exposures.

"In the U.K., public health authorities notified recipients of plasma-derived products such as pdFVIII that they may have an increased risk of vCJD in addition to the risk from eating potentially contaminated beef products. The U.K. health authorities notified patients to inform their surgeons and dentists about their potential exposure as a public health precaution intended to prevent possible secondary spread of the disease from dental and surgical instruments. The PHS, including the FDA, CDC, and NIH, does not believe that such notifications are necessary in the U.S. This is based on the extremely small risk in the transmission from plasma-derived clotting factor products in the U.K. or anywhere else in the world. Given this information, the PHS believes that the potential risks of altering the standard current precautions with respect to reusable surgical and dental instruments, and instruments used for invasive procedures outweigh any potential benefits."

In the United Kingdom, from 2004 on, people with hemophilia are to tell their dentists of their possible risk of CJD from receiving pool blood clotting agent so disposable instruments can be readied as needed. The dentists are instructed not to turn them away.

U.S. Public Health Service: 'Little Cause for Worry' Here

In at least five countries as of January 2007, blood donors have gone on to develop vCJD, so unrealized spread of the disease is not out of the realm of possibility.[3] The United States is not among them. Those unable to give blood in the United States due to their travels outside the country are not required to tell their dentist or oral surgeon.[37] In one person's circulating

blood outside the central nervous system, PrP^{TSE} titer is likely lower but it cannot be sterilized.[3] It's hard enough to get the news that you are at "higher risk" for the agent causing an incurable fatal disease. That could cause significant attacks in someone with anxiety problems. People would rather not know they are at risk of something. Now the FDA would like a test to identify subclinical incubators of vCJD. This would be handy information, because then doctors would know whether you are at risk and if you would subsequently require an extraordinary approach to minimize infection risks via surgical and dental tools.

The bloodborne risk of prions is unknown but small. But until researchers produce data that shows the prion load needed to conduct infection and the corresponding risk level, the "logic" is theoretical too. Should even a single prion be left on a dental drill that is reused, we will be in the dark until there are investigations showing that one prion does or does not confer infection and the role of a host's response. In 2007, to needlessly alarm the U.S. dentist that special precautions or disposable instruments are needed is not warranted yet, and may never be.

Authorities Dispute Claim By Some That CJD Is Misdiagnosed as Alzheimer's

Because it is so rare, physicians reportedly have misdiagnosed CJD as Pick's disease, vascular degeneration, paraneoplastic syndromes, viral encephalitis, and meningitis. U.S. research teams have looked for prion gene ties with Alzheimer's and other dementias.[38] In England, however, there is a greater interest shown by public health officials in this area. Among the 2006 Spongiform Encephalopathy Advisory Committee recommendations is enhanced clinical surveillance in the aged to learn if there may be any "under-ascertainment of cases in the elderly due to misdiagnosis."[26]

Cases of secondary transmission of vCJD, if any, may be detected in the elderly as they are more likely to have had surgeries and received blood transfusion.[26]

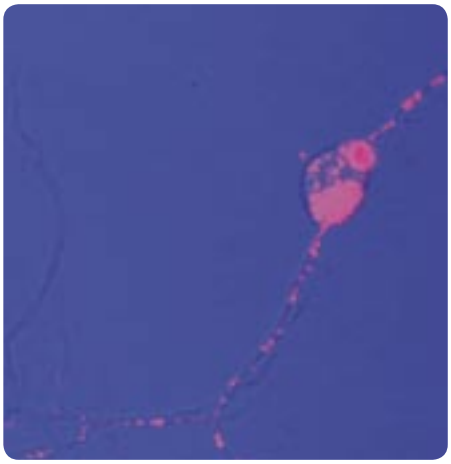
Beef and Bovine Spongiform Encephalopathy in America

Is mad cow just more Y2K and the sky is falling type nonsense? To be sure, there have only been three vCJD cases in the United States, and assurances come from some public health leaders are that there's probably "few if any others in the population." Knowing this, even if current standards in dental offices do not inactivate prions on at-risk patients, will it make a difference? Why throw considerable sums of money at protections if there is no data projecting that there will be substantial new cases of other vCJD carriers in America? Also, it is unknown if some who are infected will never have the disease. So, dentists and surgeons have mostly been shrugging off theoretical risks.

Beef products may be safer in the United States today than a decade ago. Processed meats like beef hot dogs and bologna used to have the paste- and batter-like meat product produced by forcing bones, with attached meat, under high pressure through a sieve to separate the bone from the edible meat. This mechanically separated meat was banned to protect consumers against bovine spongiform encephalopathy for human food in 2004 standard changes by U.S. Department of Agriculture's Food Safety and Infection Service.

Background on animal transmissible spongiform encephalopathies. The small numbers of North American bovine spongiform encephalopathy cattle have been minor compared with the outbreaks in the United Kingdom and Europe. Scientists are not sure how bovine spongiform encephalopathy jumped species to humans. Luckily, the numbers of infected cattle peaked in the United Kingdom and Europe in the mid-1990s, and continues to fall.[39] Unfortunately, the number of countries with "mad cows" increases each year. As of fall 2006, three cows had tested positive for bovine spongiform encephalopathy in the United States; five in Canada; 83 in the United Kingdom; 189,000 worldwide. At this writing, Japan will trade U.S. cattle if 20 months or younger, and they test each of their own cows. The United States' previous wider net of testing found few in the past several years, so the decision in 2006 was to limit testing to "downers" (falling down animals) and other targeted abnormal cows because to do otherwise seemed cost-excessive. The 90 percent cutback on animals tested saves about $35 million a year and still amounts to 40,000 animals tested per year.[40] Before the first bovine spongiform encephalopathy-positive cow was identified in America, the U.S. exports of beef product were $3.8 billion (2003); afterward they had fallen to $1.4 billion (2005) because of fears that the U.S. testing isn't rigorous enough.[41]

W. Ron DeHaven, administrator of



Prion trafficking in nerve cells. Prions branded with a fluorescent dye (pink) were added to nerve cells taken from a hamster brain. The prions initially were in the form of large clumps on the cell, but over time the clumps were broken into smaller units and transported along wire-like nerve cell projections.

the Animal and Plant Health Inspection Service for the USDA, said that he eats U.S. beef and knows there isn't a significant problem and never has been. In a 2006 editorial, DeHaven wrote that USDA's recently released surveillance data over seven years and 764,000 samples finds "the prevalence of bovine spongiform encephalopathy in the USA is less than one case per 1 million adult cattle, with the most likely range of infected animals being four to seven."[42]

In Wisconsin, the Department of Natural Resources has spent $27 million battling the disease since it surfaced in 2002 in that state, one of at least 14. Bovine spongiform encephalopathy jumped the species barrier to humans from cow, so who is to say chronic wasting disease won't go from deer to human?[43]

Dental Instruments

The most recent frontier is surgical instruments, and the data are accumulating but have not yet been pooled to give any index of risk by type of tool or equipment, type of surgery, or exact questions to identify vCJD vs. probable vs. at-risk/possible vs. no/low-risk patient. Some organizations are releasing their assessment of the estimated infectivity in each human bodily tissue and fluid.

In California, one dental school admitted to tackling the potentially infected CJD patient a little differently. The University of California at San Francisco is sending patients with the potential risk of a prion disease to its medical center hospital, where gloved safety specialists pick up the contaminated waste and arrange for disposal/incineration all within one hour of its exposure. In clinics, like UCSF's Memory and Aging Center, patients are screened for prion disease risk, and if it cannot be ruled out as a possibility, should dental care be necessary, dental instruments used are either quarantined pending diagnosis, incinerated, or cycled 10-20 times at the direction of health authorities who intend the precautions to prevent possible secondary iatrogenic spread of the disease via contaminated dental and surgical instruments.

There have been three people in the United States with vCJD, 197 elsewhere in the world. While human "mad cow" might

one year spike into an epidemic in the United Kingdom, the CDC isn't predicting an epidemic here.[44] Even if that number rose by 100 percent, that would be six people. What's the chance that one of those highly infectious "mad cow" people was sitting in your dental chair today?

Exposure Scare Over CJD-Tainted Surgical Tools Shutters Hospital, Causing Cancellation of Surgeries, and Resulting in Patient Lawsuit

One of the first hardships of operating in a prion-potential world is how upsetting it is to patients. At Emory University Hospital in Atlanta during 2004, some 500 patients had to be telephoned with a particularly unpleasant incident report — their own.[45] The surgical instruments used on their brain/spine surgeries were previously used on a patient who tested positive for sporadic CJD.

Last year, a teacher who had been among those patients informed of the potentially contaminated tools used on her surgery filed a lawsuit for monetary damages and a fund for her medical costs, should she develop the disease. Among other errors, the complainant lists not having quarantined surgical tools or using a Joint Commission on Healthcare Accreditation sterilant that "dissolves brain tissue."[46]

Trying to avoid this kind of legal entanglement, hospitals are starting to gear up for these prion-risk cases. Last month, surgeries were canceled for a few days at South Ontario Hospital in Canada, as the Public Health Agency of Canada's Creutzfeldt-Jakob Disease Surveillance System was busy testing surgical instruments used on a brain surgery in a suspected CJD patient.[47] One can imagine a bank of telephones, scripts ready, hospital representatives dialing the residences of patients who may have come into contact with the instruments used in the operation. Fortunately, the more comprehensive tests of the instruments were negative as of this writing.

These hospital-based neurosurgical risk-disclosures occurred in the United States, and are educational for oral and maxillofacial surgeons and dentists performing complex protocols. All over the United Kingdom and Europe, dentistry is now a highly monitored profession because of the mad cow cases there. Patients are to inform their dentists if they have a family member with the genetic form of CJD, if they received any tissues or fluid donation, or received oral or other surgery with instruments used on someone who later was diagnosed with CJD.

If you were a dentist in Scotland

and you suspected your dental instrument accidentally abraded the tonsil ("medium" infectivity) of a patient at risk of CJD, that instrument is to be sterilized at least 10 times to minimize prions remaining on it.[48] While routine dentistry is considered low-risk, if you are an oral and maxillofacial surgeon doing procedures on the head, neck, face, or orbital regions, additional precautionary measures may be needed to reduce possible CJD transmission.[36]

No evidence-based data and projection models have been generated to indicate whether dental transmission of vCJD has, is, or likely will occur. Indeed, the threat of transmission remains a possibility.[49]

UCSF Medical and Dental Centers Adopt Highest 'As If' Precaution Level Infection Control Theory That Most Human Tissues and Fluids Are Infective

Few would disagree that UCSF, through the Memory and Aging Center, is on the leading edge of research into understanding prion diseases worldwide. Ermias Belay, MD, an epidemiologist with the CDC, is not alone when he points out that there are other theorems worth pursuit, "Still, there is controversy on whether prions are the only agents that cause CJD."[50]

In reading the UCSF Medical Center Infection Control Manual Guidelines on treating CJD patients, they seem to come from a different direction that overlays its own policies and procedures above the existing national and international guidelines for minimizing prion transmission.[51] Because UCSF is the premier location of prion research in the United States, it is very aware of the implications of its data and protocol for infection control.

"I have read many articles from the U.K., and there are no uniform recommendations and lots of 'refer to your infection control coordinator for protocol,'" said Molly Newlon, DDS, MA, director of Health and Safety, UCSF School of Dentistry.

"I assume that all used items, including the metal instruments are incinerated. I do know that deactivation of prions on stainless steel is very difficult, and the instruments must be in an autoclave for so long that it destroys the instrument. The research that is currently being done in the Prusiner lab involves testing a soaking solution that may be used prior to autoclaving that will deactivate the prion. As I understand it, this product is still being tested and is not yet available for use in the marketplace. Also, it is my understanding that conventional sterilization procedures cannot guarantee prion deactivation, which is why any instrument used on a CJD patient in dentistry is single-use only."

Brown and colleagues conducted a 2005 instrument study that is cited in medical literature to show how corrosively damaging the WHO protocol for prion deactivation can be.[52]

Can't Touch That

Prions on instruments are difficult to inactivate. Instead of providing a list of all the decontamination protocols that are ineffective against prions (16 minimum processes), dentists should know that when it comes to highly infectious tissues and contaminated instruments, everything you were taught in dental school can't touch it.

"Our studies show that the standard sterilization techniques in use are insufficient to inactivate human prions.[53] However, acidic sodium dodecyl sulfate combined with autoclaving can inactivate prions beyond the level of even the most sensitive detection methods," said Kurt Giles, DPhil, assistant adjunct professor, Institute for Neurodegenerative Diseases, UCSF.

Regarding 2006 *Journal of Virology* findings of Prusiner and coauthors, Giles said, "The main findings of our paper were that human prions are 100,000 times more difficult to inactivate than hamster prions.[53] This is of great significance since hamster prions have historically been used as the standard for prion inactivation protocols. We also found, as have others, that prions bound to stainless steel are even more difficult to inactivate than prions in suspension (again, historically prions in tissue suspension have been used as the standard)."

U.S. Dental and Oral Craniofacial Leaders Confident While U.K. Equivalent 'On Watch'

While American physicians and dental surgeons read relatively little about prion diseases in their journals, in England, a flurry of updated guidelines at the end of 2006 has dental practitioners sitting up. They know there will likely be word from the Department of Health that summarizes updates since the 2005 let-

ter from the chief dental officer advising that routine dentistry requires no special adaptations for infection control even on patients who may have CJD.

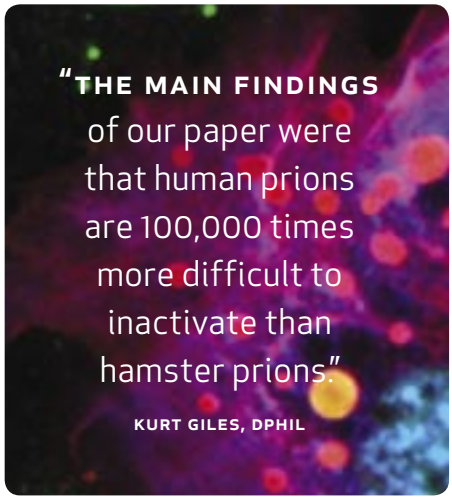
From the two Spongiform Encephalopathy Advisory Committee reports last year, the latest issued Nov. 30, 2006, it appears a Department of Health preliminary risk assessment recently was completed of vCJD on difficult-to-clean endodontic instruments, infectivity of dental pulp, and subclinical vCJD carrier state.[4,26] To date, no data show that dental pulps are infectious, yet peripheral nerves and blood are both in close proximity. Said the report, "Although data are limited and indirect, it is reasonable to assume that the dental pulp of individuals subclinically infected with vCJD may be infectious although the level of infectivity in unknown."[4]

Although refinements in risk assessment and direct data from vCJD human pulps will improve predictions, the Department of Health preliminary assessment seems more concerned about endodontic equipment, which had been suggested but not required as one-use anyway: "Dental pulp is as infective as peripheral nerve tissue and if a subclinical carrier population for vCJD exists, a self-sustaining vCJD epidemic arising from endodontic surgery is plausible."[4]

William G. Kohn, DDS, associate director for Science, Division of Oral Health, CDC, said he feels at ease about the status of the information put forth for dentists. Kohn said "there is no more risks than we determined there'd be, so no updates are needed. vCJD transmission is of no greater concern for dentistry in the U.S. now than what it was in 2003."

At the time the 2003 guidelines from the CDC were printed, vJCD wasn't associated with convincing evidence of prion detection in human blood. The 2006 WHO guidelines acknowledged that blood transfusions infected recipients with "mad cow."

"But you can't jump from three blood transfusions in U.K. transmitting vCJD means all blood for any dental procedure in the U.S. is now infectious with prions potentially," Kohn said. "Saying 'all blood is potentially infectious' is panic-laden statement that is invalid."



Updating Medical History Forms

In the United Kingdom, to identify patients who are about to have surgery who may be at-risk of developing CJD, individuals are questioned on family dementia before age 65; dura mater grafts; corneal transplant; pituitary hormone injection pre-1986; insulin injection pre-1989; problems concentrating, reasoning, remembering; and unsteadiness walking, jerky movements, or lacking previous coordination. If any answer "yes" to the aforementioned, they are referred to a neurologist for more careful screening of risk category.

A 2006 article written about the U.K. situation suggests that screening all patients to learn who relies on blood transfusions and clotting agents is necessary to discern patient risks.[33]

It is believed that there are lower titers of vCJD prions in lymphoid tissues of non-U.K. individuals so chances of transmission from "any" surgical procedure would be somewhat less of an issue.[53]

Whether dentists should have any screening questions to identify patients who may be at risk of developing CJD in medical history, as done in the United Kingdom and suggested elsewhere prior to any type of surgery (patient CJD risk categories are definite, probable, possible [includes diagnosis unclear], unlikely, definitely not) remains something the CDC doesn't think is even worth going into.[21,54]

"It's so rare in the U.S. that a dentist would likely never hear the answer 'Yes, CJD,'" Kohn said. Given the current prevalence of this disease, he added, "It doesn't make any sense to ask more specific questions to screen for it and all the other rare diseases, or you'd go on forever and screen for all kinds of conditions and it would take too long."

T. Forcht Dagi, MD, MPH, of Harvard-MIT Division of Health Sciences and Technology, agreed with the CDC's Belay on this point. Dagi explained that irrespective of diagnosis status or organism, "from a policy perspective, the American College of Surgeons has taken the route of recommending and endorsing the highest level of universal precautions for all patients."

The American Association of Oral and Maxillofacial Surgeons refers media questions to one of its members especially interested in prion diseases: oral and maxillofacial surgeon Eric R. Carlson, DMD, MD, FACS, chairman of the Department of Oral and Maxillofacial Surgery, University of Tennessee Medical Center, Knoxville. Carlson said obtaining a thorough history, particularly

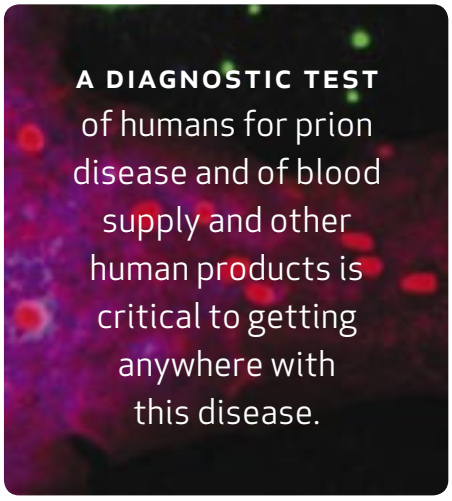
related to any observations of dementia, is the starting point. Because eye tissue is contacted during facial surgeries, "I recommend that surgeons performing orbital and facial surgery follow the guidelines established by the National Institute for Health and Clinical Excellence so as to reduce the risk of transmission of Creutzfeldt-Jakob disease. This involves the adherence to steps taken to ensure that surgical instruments can be tracked," he said. Moreover, surgeons electing to implant freeze-dried allografts, particularly dura, should obtain these from an American Association of Tissue Banks-accredited tissue bank.

Research Frontiers

The National Institute of Dental and Craniofacial Research hasn't conducted or funded any CJD studies, likely because there hasn't been a single report of prion disease from dentistry. If data emerges showing surgical instrument transmission during dentistry, with solid evidence of oral tissue infectivity, it may reprioritize research funds for the good of public health. For now, the U.S. Army Medical Research and Materiel Command of the Department of Defense awarded $42.5 million for prion disease research in fiscal year 2002. Some of those recipients began in 2003, but all will conclude their projects in 2008. We are just beginning to understand that there are prion-based diseases, so there very well may be more prion-based diseases than CJD.

A CJD test. Available tests for prion diseases are mostly postmortem, invasive, nonsensitive, and nonquantitative. A diagnostic test of humans for prion disease and of blood supply and other human products is critical to getting anywhere with this disease. In animal models, an in vitro "asymptomatic prionemia" blood test in development is encouraging, with at least one team publishing promising results in 2006.[55] Human vCJD prion-laden blood has yet to be rigorously investigated. A PrP^{TSE} test for the sporadic form of CJD in the nasal olfactory mucosae is also beings studied.[3]

Respected University of Chicago neurologist James A. Mastrianni, MD, PhD, at the Prion Laboratory, is trying to make infectious prions. If he and his team can learn the mechanism behind the folding machine, identifying novel therapies for treating prion diseases would be a next step. He said he'd like to define sites on the PrP^{TSE} to target with a "designer peptide" to bind and block interaction that causes nearby normal prions to go abnormal.



Vaccine. More cases of vCJD in the United States would have to occur before enough human agent is supplied to work on developing a vaccine. The few, precious vCJD blood samples for researchers aren't enough to go around for the world community of scientists, and this is a major hurdle for any potential developer.

Therapeutic directions. With prion discovery relatively new — compared to fungi, parasites, bacteria, and viruses — drugs or gene therapy could involve inactivating prion formation and eradicating existing clumping prions. Again, there is the idea to "unfold" them to make them vulnerable to destruction.[56]

Effective infection control method for prions. Paul Brown, MD, leading epidemiology bovine spongiform encephalopathy expert, has researched and published on various decontamination regimens. He suggested to dentists that a regimen of immersion in NaOH/bleach for at least an hour, washed, then autoclaved at 132 C for at least 20 minutes isn't perfect but better than what many are now doing.

FDA and CDC Monitoring Disease Etiology and Incidence Patterns for Clusters Around Healthcare Workers and Pathologists

The CDC's Belay is not alone in the reassurance that he hasn't seen any cluster of CJD among dentists or any other profession.[6] "The group who we would think to be maximally at risk of CJD infection because of professional exposure would be neuropathologists, and no cluster of cases among neuropathologists has been reported. We don't expect to see it in dentists either."

The CDC's Kohn assures that if the rate of incidence changes and more vCJD patients are sitting in dental chairs, or any new data emerges of dental mechanisms that spread PrP^{TSE} that put patients or providers at risk, they will act. "But three cases of vCJD transfusion in the U.K. doesn't warrant any changes at this time in the U.S."

In 10 to 20 years, we'll know "the last chapter."

Until then, any measures introduced for infection control in American dentistry in response to the three "mad cow" cases will need to be evidence-based and proportionate to the risks. ■■■■

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Putting the Practice into Evidence-based Dentistry

IAN D. COULTER, PHD

ABSTRACT Whenever a new field emerges in health care, a period is experienced in which the field tries to define itself. This is the position evidence-based dental practice finds itself in at the moment. In this paper, it is argued that, for dentistry to enter into the brave new world of evidence-based practice, it will require some rethinking of the research enterprise in the profession.

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The standard definition is given by Sackett.[1] Evidence-based practice is "the conscientious, explicit and judicious use of the current best evidence in making decisions about the care of individual patients ... means integrating clinical expertise with the best available external clinical evidence from systematic research."

This is usually contrasted to traditional dental practice where "Emphasis is placed on accumulated knowledge and experience, adherence to accepted standards and the opinion of experts and peers. It is practical, prudent, personal."[2]

The definition for evidence-based practice, therefore, begs one or two questions. Words like "conscientious, explicit and judicious use" are not only a bit subjective but raise questions about who is to be the judge. Then, the added problem, what is the "best available external clinical evidence from systematic research"? To answer the second question, one needs to look at evidence-based research.

Evidence-based Research

It is necessary to distinguish two different but intimately linked movements: evidence-based practice and evidence-based research. The latter refers to the process, by which evidence is generated, the methodologies such as systematic reviews, meta-analyses. But the former refers to the application of evidence to actual practice.

Though one might assume these two would be highly related in that the second should lead logically to the first, it is not necessarily the case. This occurs for a couple of reasons.

The first is that evidence-based research puts a premium on the quality of the research. It must be rigorous and be able to be replicated. While a wide range of evidence may be considered, evidence-based research has established a hierarchy in terms of the quality of research. The principle underlying this hierarchy is which methods give the most definitive answer in determining that the therapy used was responsible for the health outcome measured.

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The clear answer is that studies that can answer questions about efficacy are the most preferred, and the method that does that most clearly is the random controlled trial. The dominant focus of evidence-based research practice, therefore, has been the random controlled trial. However, it is not the single, random-based trial that is the gold standard. Rather, it is the systematic review of numerous random controlled trials that is the most significant, particularly those that result in a meta-analysis. In fact, the results of a single, even double-blinded, trial can be misleading, particularly if the number of subjects is insufficient to power the study (in effect, give it statistical legitimacy). Meta-analysis overcomes that problem by combining studies that are homogeneous so that the subject pool is larger.

Other forms of study design may be included in an evidence-based research systematic literature review (nonrandom trials, cohort studies, and simple pre-post and post-case series) but the random controlled trial is given most weight since it is the design that most clearly establishes efficacy.

Unfortunately, such studies generally test a therapy under ideal conditions and often with homogeneous populations to ensure comparability of the groups when comparing outcomes. But evidence-based practice ultimately requires therapies that can be applied in normal practice, that is, effectiveness studies.[3] While on logical grounds, a therapy without any efficacy will not be effective; a therapy that has efficacy may not have effectiveness when applied to heterogeneous populations and normal practice conditions. Furthermore, therapies with equal or comparable efficacy may differ considerably in terms in effectiveness.

In contrast, however, random controlled trials test therapies under ideal conditions and therefore, do not often help with determining effectiveness in everyday practice as opposed to efficacy in a controlled, and usually perfect, setting.[4] There are some very strict ethical limitations to conducting clinical trials that prevent certain populations from participating. If there is a very high risk but low benefit for a subgroup of patients, this might mitigate against them being included, such as patients with high co-morbidities. Conversely, some low-risk patients may not be included because too large a number would be needed to be enrolled to make the study feasible.[5] The end result, therefore, is that clinically, it is not possible to know if the therapy can be applied to groups that were not included in the trial.

THE TYPE OF CLINICAL detail essential for a provider to decide if a given patient is a candidate for a drug, procedure, therapy, is seldom provided in a random controlled trial.

Although providers do treat populations, they treat them one at a time. Random control trials seldom contain the "soft data" about individual variations, particularly in response to therapy. The type of clinical detail essential for a provider to decide if a given patient is a candidate for a drug, procedure, therapy, is seldom provided in a random controlled trial.[6] They provide the results of average patients, and, even then, it is an average of those who meet the inclusion criteria.

This problem can be solved through observation studies, but there is a dilemma about the role of observational studies. On one hand, they may seem more clinically relevant and include the populations and subpopulations of interest to the health provider, but, on the other hand, they do not provide the type of definitive evidence that might persuade the provider to recommend the procedure to the patient. Despite this ambivalence, observation studies continue to be widely published. Ray, in a survey for two months in 1998 of three leading medical journals, found that observational studies comprised 68 percent to 87 percent of their featured articles and communications and only 32 percent, 13 percent and 26 percent of their publications were random controlled trials.[7] He noted while it is now known how observational studies impact practice or policy, given the propensity to publish them, the journals must feel they are important to their readers.

One solution to the dilemma in evidence-based research has been to create a hierarchy of evidence. A standard hierarchy is the following, from the highest to the lowest: evidence provided by at least one appropriately designed random controlled trial; evidence provided by a controlled trial that is not randomized; evidence provided by a well-designed cohort or case-control study; evidence provided by a multiple time series; descriptive studies, case reports, and opinions of experts or respected authorities.[8]

Evidence-based Dental Practice vs. Evidence-based Research

The problem is not so much that the practitioner and researcher disagree that practice should have some evidence to support. It is more to do with how

TABLE 1

The Problem

The dentist	The researcher
Evidence means what works well for me in my practice and clinical experience is the basis for deciding this.	Evidence means what has efficacy and why, and clinical experience is a very problematic source for this.

TABLE 2

The Difference

The dentist wants truth on his/her side.
The researcher wants to be on the side of truth.

that evidenced is defined. This is shown in TABLE 1. This can also be represented in another way as shown in TABLE 2.

There is, therefore, a disconnect between research and practice. Research can be both rigorous and clinically useful, unfortunately, a case therefore that "never the twain shall meet."

Is Dentistry Capable at the Moment of Doing Evidence-based Practice?

Clearly, dentistry does a lot of scientific research, but much of this is in the laboratory and focuses on what might be termed as biomedical research. This involves the use of the basic and biological sciences for the investigation of disease, its biological mechanisms, and reparative processes. One can also point to the large field of materials research, work in implants, periodontal disease, maxillofacial surgery, genetics, wound healing, and so on. But if one takes the most common of all oral diseases and evaluates the "evidence" for its diagnosis and treatment, the results are a bit sobering.

In 2001, the National Institutes of Health convened a consensus conference, bringing together a panel of experts, reviewing the research evidence, and hearing submission and testimonies from experts on this topic.[9] The overall conclusion of the panel was that the evidence base for most practices in the diagnosis and management of caries is weak. Across the whole spectrum of research from epidemiology, to diagnosis, to treatment, to outcomes, they found a lack of studies, in particular, those that could establish efficacy. There were not only a very low number of trials (only seven where there was definitive evidence the patient had caries), the quality of the trials was also problematic.

The question is not so much "is dental research collecting evidence?" but more a case "is it relevant to practice, can it be translated into practice, and is it in fact being used to determine practice even within our teaching institutions?" Is sufficient work being done on trials to establish efficacy? Even where that is occurring, are further studies being done on effectiveness, which would determine real outcomes in real practices with real patients?

The Problem

Until very recently, it was not at all clear the research was driven by the needs of practice. One of the benefits of evidence-based practice as a movement might be to help refocus the effort. But even there, it tends to be a one-way movement. The research mostly occurs in large institutions (i.e., National Institutes of Health, universities); much of it is in the laboratories. The results are published, disseminated, taught in dental schools, and in continuing education programs. One might term this the trickle-down theory of research. As in economics, the expectation is that all the boats will benefit and float a little higher. There is very scant evidence that the theory actually works.

In many ways, what happens in research is not dissimilar to the person who searches under the streetlamp for their keys. Upon being asked if they were sure they lost the keys there, the person answers "No," but is looking there because that is where the light is. Very little research is done in dentistry in practice settings. Most research follows the medical model and is university- and hospital-based. But there is a huge difference between dentistry and medicine in this regard. Most dental patients are not in hospitals, and only a very small number of them are in the dental teaching clinics. Those who are tend to be atypical patients.

In the case of medicine, the universities are attached to huge teaching hospitals with access to very large populations of patients. So one can study cancer through the National Cancer Institute Centers with some assurance that trials can be conducted on the major cancers, and that these will not be significantly different from the cancers encountered in general practice. But this kind of infrastructure does not exist for dentistry, which may help explain the low number of trials conducted.

In the Caries Consensus Conference it became clear that two fundamental pieces of evidence were missing, which impact drastically on trying to conduct trials. The first is that the epidemiology of caries is insufficiently studied. The committee was unable to establish the natural history of caries. Without this it would be impossible to know whether any given treatment is actually performing better than leaving the disease untreated.

The second great gap in the evidence is in knowing what dentists are actually doing in their practice. There is almost a complete lack of descriptive studies, using random samples that would allow one to generalize about the practice of dentistry. The real answer to the question "Is dentistry evidence-based?" cannot be found in the evidence of evidence-based research, but can only be answered by knowing what is happening in the dental practices. The correct answer

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to the question "Is dentistry evidence-based?" is we do not know. Despite all the scientific research being done within dentistry, the truth is we do not know how much of dentistry is evidence-based.

If one looks to medicine, the data would suggest the figure is not likely to be very high. There is considerable debate about how much of medical clinical practice is evidence-based. The initial estimates by the Office of Technology Assessment in 1979 and 1983 were that only about 10 percent to 20 percent of medicine could claim to be evidence-based.[10,11] As noted by Imrie and Ramey, this figure was simply an estimate.[12] These authors further note that other commentators have given figures as low as 15 percent for practices based on any evidence.[13]

The problem of establishing any figure is one first needs to define what will constitute the evidence. How one does that has a great impact on the result. If, for example, one demanded only one good single, random controlled trial, the figure will be much higher than if one required repeated random controlled trials. The use of a single, random control trial, no matter how good the study, poses methodological problems. Single studies can be contradicted by later studies. To overcome the problem of a single study, studies are pooled if they are homogeneous enough to permit a meta-analysis. This also greatly increases the sample sizes on which analyses can be done.[14,15] Examples of misleading meta-analysis have already been documented in the literature.[16] Furthermore, studies with negative results are less likely to be published.[17] This itself has a tremendous impact on the "evidence."

If the figure for medicine for evidence-based practice is as low as has been estimated, it is very unlikely that the amount of evidence-based practice in dentistry is any higher.

The Solution: Putting the 'P' Back Into Evidence-based Practice

One solution is to begin focusing on a different kind of research, what one might term practice-centric research. In an earlier article, the author suggested that one solution is a move toward health services research.[18] Briefly, such research with its focus on patients, access, utilization, services, costs, quality of care, appropriateness of care, the health encounter and outcomes would place the focus squarely on the practice of dentistry. Health services research is defined as the investigation of the relationship among social structure, process, and outcomes for personal health services.[19] In fact, it is difficult to see how evidence-based dental practice is going to be possible without this type of evidence.

Part of this shift in emphasis would also mean a shift from a focus on efficacy (trials) to a focus on effectiveness (what works in practice under normal conditions, with normal patients with normal dentists). When something has been shown to be effective in practice, then one should move to trials to determine efficacy. When something is known to be effective and efficacious, then one should move to understanding the biological mechanisms involved. This would be an almost complete reversal from the way research proceeds at the moment, and the way the National Institutes of Health funds research.

But in addition to a change in the type of research methods, there must also be a change in the sites of research. To advance evidence-based practice, practice-based research needs to be advanced. The first need here is simply to determine what dentists are doing in their practice (what is being practiced). This is known as descriptive studies and is virtually unfunded by the National Institutes of Health, which prefers hypothesis-driven research. It cannot be obtained simply by looking at patient files or by interviewing patients and dentists. All of these are valuable, necessary, but inadequate. It requires observation of practices using methods such as a rapid ethnographic assessment to compile a comprehensive account of what dentists are actually doing in practice. The view of practice obtained by such areas as epidemiology/health services research, and that obtained by sociological and anthropological observation, are so different as to lead one to conclude the two groups are viewing totally different animals.[20]

A second requirement is that practices have to become the sites for collecting data. As noted before, hospitals, universities, and laboratories cannot provide the kind of data needed to determine what kind of dentistry is being practiced, or to study the outcome of particular therapies as used in practices. In many ways, dentistry is often seduced by its close relationship to medicine. Its research thrust for the most part attempts to replicate an approach that has been highly successful in medicine.

Unfortunately, the practice of dentistry is in fact quite unlike medicine. Dentistry much more resembles the practice of solo practitioners, such as optometrist and chiropractors. What the latter has recognized, and, in fact, what the entire Complementary and Alternative Medicine group has recognized, is that they must form practice networks for the assembling of data. Since they are not part of hospitals, and for the most part, not part of universities, they have recognized there is no alternative as they also face the challenge of substantiating that their practices are evidence-based. Hawk, Long and Boulanger in 1998 established such a network of practices for the chiropractic profession.[21]

Conclusion

For dentistry to enter into the brave new world of evidence-based practice, it will require some rethinking of the research enterprise in the profession. Not only must the focus of the research change, but we must also see changes in the methodologies used, and the sites in which the research is conducted. If it is intended to label "evidenced practice" to mean what it actually says, putting the practice back into the equation is needed. That involves recruiting the dental profession, those in practice, to "come on over" to the world of research. While most dentists are used to their alma mater asking them to open up their wallets, in the future they may be asking them to open up practices. Open them up to the inconvenience of research. It is difficult to see how, without doing this, we can determine how much dentistry is evidence-based, and secondly, how we might transform the practice so that it is. ■■■■

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Management of the Hypertensive Dental Patient

BY JOHN A. YAGIELA, DDS, PHD, AND T. LANT HAYMORE, DDS

ABSTRACT Hypertension is a common malady and a harbinger of such diseases as heart attack and stroke. Because millions of Americans are not aware they are hypertensive or it is not adequately controlled, dentists can contribute significantly to national health by screening their patients. Dentists must also be cognizant of the implications high blood pressure has for dental practice. Specific treatment recommendations include limiting dental care in patients with severe hypertension, reducing stress, and periodically monitoring blood pressure.

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Hypertension is the most prevalent systemic disorder in the United States. One-third of American adults, or about 70 million individuals, have high blood pressure.[1] A similar number have prehypertension, which predisposes them to developing clinical hypertension in the future, and still others have one or more risk factors for it. Worldwide, hypertension may afflict as many as 1 billion individuals and be responsible for 7.1 million deaths per year.[2] It is inevitable that most dentists will be called upon to treat hypertensive patients on a weekly basis. The purpose of this review is to provide the practitioner with an understanding of (1) the basic pathophysiology of hypertension, (2) how hypertension and its management can affect dental treatment, and (3) steps the dentist can take to provide optimal care for the hypertensive dental patient.

Hypertension

Hypertension is defined in adults by a mean systolic blood pressure of 140 mm Hg or greater, a mean diastolic blood pressure of 90 mm Hg or greater, or when the individual is taking antihypertensive medication for blood pressure control.[3] Hypertension can be characterized as either primary or secondary. Primary hypertension is the term used for high blood pressure where no specific etiology can be found. When a specific cause is identified, the increase in blood pressure is called secondary hypertension.

The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure now classifies blood pressure values into four categories (**TABLE 1**).[4] The designation of normal blood pressure as less than 120/80 mm Hg and prehypertension as 120-139/80-89 mm Hg reflects recent data on the progressive development of high blood pressure with age, its profound negative influence on cardiovas-

TABLE 1

Classification of Blood Pressure for Adults

BP classification	SBP (mm Hg)	DBP (mm Hg)
Normal	<120	And <80
Prehypertension	120-139	Or 80-89
Stage 1 hypertension	140-159	Or 90-99
Stage 2 hypertension	≥160	≥100

SBP: systolic blood pressure; DBP: diastolic blood pressure
From Chobanian, et al.[4]

cular health, and the resultant increased emphasis on preventing its development.

PATHOPHYSIOLOGY

Primary hypertension, also known as essential or idiopathic hypertension, comprises 95 percent of all cases of high blood pressure. It is the default diagnosis when all other causes for the disorder have been excluded. As is the case with obesity, that other scourge of modern life, primary hypertension may not be a disease in the classic sense. It is instead most likely a consequence of homeostatic mechanisms that were appropriate in prehistorical times but are now problematic given the sedentary lifestyle and salt- and calorie-rich diet common to Western nations.[5] Indeed, the term essential hypertension, reflecting the once widespread belief that the condition was necessary to support normal tissue perfusion, may have held some truth in previous epochs.

An increase in arteriolar constriction and total peripheral vascular resistance is frequently observed in primary hypertension.[6] Despite the resultant heightened myocardial afterload, cardiac output remains in the normal range. Increased sympathetic nervous system activity helps explain the arterial vasoconstriction, as well as the augmented myocardial function and activated renin-angiotensin axis that collectively maintain the cardiac output. Over time, the increased hemodynamic load, and some of the mediators supporting it, cause the heart and blood vessels to hypertrophy and become less compliant.

Although the exact cause of primary hypertension remains elusive, a unifying theme has been proposed whereby any one of several inciters — sympathetic nervous system activity, renin-angiotensin axis stimulation, or hyperuricemia — causes renal vasoconstriction.[7] Decreased renal blood flow then activates the body's mechanisms to increase blood pressure and volume. Variables that influence these inciters include genetics, physical and emotional make-up, diet, physical activity, and environmental conditions.

Secondary hypertension accounts for only 5 percent of all cases of high blood pressure. This diagnosis is important, however, because it suggests that effective treatment of the underlying disease process will lead to its cure and because it notifies the clinician of an associated health problem with medical significance.[6] **TABLE 2** lists the leading causes of secondary hypertension along with some of their identifying characteristics. Other instigators of secondary hypertension include pregnancy (preeclampsia), various neurologic disorders (brain tumors, dysautonomias, etc.), and vascular abnormalities (e.g., arteriovenous shunts).

Many prescription and nonprescription drugs and several herbal products can cause secondary hypertension or exacerbate primary hypertension.[4] For example, immunosuppressive agents such as cyclosporine, tacrolimus, and methylprednisolone increase blood pressure in up to 90 percent of solid-organ transplant recipients.[8] Cigarettes and most nicotine-replacement products (but not the transdermal patches), decongestants, amphetamine-like drugs, and other sympathomimetic agents likewise promote hypertension. The hypertensive effects of ephedra (ma huang) caused the Food and Drug Administration in 2004 to place limits on its sale. Nonsteroidal antiinflammatory drugs and cyclooxygenase 2 inhibitors do not cause hypertension by themselves but are capable of destabilizing blood pressure control in patients with hypertension.[9]

CONSEQUENCES OF HYPERTENSION

End-organ damage is a natural consequence of chronic, uncontrolled hypertension. The heart, brain, and kidneys are most at risk. Hypertrophy of arterial muscle and other arteriosclerotic changes in small arteries that occur in response to high blood pressure and related mediators such as angiotensin II begin to impair perfusion of affected tissues. The development of ventricular hypertrophy and atherosclerosis of large arteries further affects blood flow to end-organs by reducing cardiac output and arterial elasticity. In the kidneys, the reduced renal tissue perfusion promotes local ischemia, tubular injury, and interstitial inflammation. Eventually, loss of adequate renal blood flow and associated ischemic changes can reduce glomerular filtration to the point of clinically evident renal insufficiency and end-stage renal failure. As renal failure progresses, there is increased fluid retention and accelerated release of vascular mediators.

TABLE 2

Primary Disorders Associated With Secondary Hypertension

Disorder	Signs and Symptoms
Acromegaly	Enlargement of hands, feet, tongue, jaw; headaches; fatigue; visual problems
Alcoholism	Alcohol-seeking behavior; intoxication; confusion; unsteadiness; skin capillary enlargement
Aldosteronism	Hypernatremia; hypokalemia; fatigue; thirst
Coarctation of the aorta	Decreased or delayed femoral pulses; cold; legs; heart murmur; abnormal chest radiograph
Cushing's syndrome	Weight gain; moon face; dorsal hump; truncal obesity; fatigue; weakness; hirsutism; amenorrhea; purple striae; hypokalemia
Drug and herbal therapy*	Medication history
Hyperparathyroidism	Kidney stones; osteoporosis; depression; lethargy; muscle weakness
Hyperthyroidism	Heat intolerance; weight loss; palpitation; tachycardia; exophthalmos; tremor
Hypothyroidism	Fatigue; muscle weakness; weight gain; alopecia
Obstructive uropathy	Pain; reduced urine output; hyperkalemia
Pheochromocytoma	Headaches; diaphoresis; palpitation; tachycardia
Renal parenchymal disease	Renal insufficiency; edema; elevated BUN, creatinine; proteinuria
Renovascular disease	Systolic/diastolic abdominal bruits; elevated plasma renin
Sleep apnea	Snoring; daytime somnolence; obesity

BUN: blood urea nitrogen

* Drugs and herbs that may increase blood pressure include acetaminophen (long-term use); antidepressants (e.g., bupropion, desipramine, venlafaxine); appetite suppressants (e.g., phentermine, sibutramine); bromocriptine; corticosteroids (e.g., dexamethasone, prednisone); epoetin alpha; ergotamine; immunosuppressants (e.g., cyclosporine, tacrolimus); liquorice; mineralocorticoids (e.g., fludrocortisone); monoamine oxidase inhibitors (with tyramine-containing foods); nicotine; nonsteroidal antiinflamatory drugs and cyclooxygenase (COX)-2 inhibitors (e.g., celecoxib); sex steroids (e.g., estrogen, testosterone); St. John's wort; sympathomimetic amines and related stimulants (e.g., amphetamine, methylphenidate, pseudoephedrine); and yohimbine.

Cardiac problems develop when the heart begins to decompensate from the increased workload and decreased perfusion of the myocardium. The heart enlarges as it increasingly depends on Starling's principle (in which stretching of cardiac muscle up to certain limit increases contraction force). Myocardial perfusion is further impaired by the increased intramural tension and by the accompanying tachycardia, and angina pectoris may occur when the oxygenation of tissues cannot keep up with the demand. Signs and symptoms of left and right congestive heart failure — including dyspnea and ascites — develop as blood backs up, respectively, into the pulmonary and systemic venous vasculatures. Arrhythmias may develop in response to the myocardial enlargement and ischemia. Sudden death from coronary thrombosis is a common terminal outcome.

Vascular pathology affecting the central nervous system includes arteriosclerotic changes and the development of microaneurysms. Occipital headaches are common early manifestations of hypertension. Episodes of dizziness or syncope, representing transient ischemic attacks, may herald the danger of stroke. Hemorrhagic strokes follow the breakage of weakened arteries or microaneurysms; ischemic strokes are the result of atherosclerosis and thrombus formation in, or embolization of, cerebral arteries.

HYPERTENSIVE CRISIS

Hypertensive crisis is a term used to indicate an acute, severe increase in blood pressure and is often defined by a systolic blood pressure of 180 mm Hg or more, and/or a diastolic pressure of 120 mm Hg or more.[3] Because individuals with chronic hypertension can tolerate higher blood pressures than their normotensive counterparts, emergency treatment of a hypertensive crisis is determined more by the rate of increase in blood pressure and its associated signs and symptoms rather than by absolute pressure values.[10] When the acute hypertension is accompanied by ongoing or impending target organ damage, the crisis becomes a true

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hypertensive emergency. In this situation, the acutely elevated blood pressures progressively damage blood vessels, which may then precipitate cerebrovascular accidents (hemorrhagic or ischemic) or myocardial infarction, or lead to acute forms of encephalopathy, congestive heart failure, renal failure, or ocular damage. Signs may include pulmonary edema, acute angina, vision loss, and seizures. Hypertensive emergencies require immediate hospitalization and treatment.

A hypertensive urgency occurs when there is no associated target organ dysfunction. Although the patient may experience severe headache and/or anxiety, shortness of breath, or epistaxis, the terms crisis and urgency are misnomers in the sense that hospitalization is usually not necessary. Instead, early outpatient medical treatment with oral medications and close follow-up by a physician is suitable. In the dental office, activation of emergency medical services is the preferable choice to ensure proper treatment when there is any question about the patient's condition.

Implications for the Dentist

Because only one-third of individuals with hypertension have their blood pressure under control to the degree recommended by the JNC, the average dentist will, perforce, regularly encounter dental patients with uncontrolled hypertension.[4] Almost half of these patients will not even know they have the disorder. Thus, the dentist should take the blood pressure of all new patients regardless of their medical history.[11] The dentist can expect to uncover untreated or inadequately treated hypertension in about one of every five adult patients tested. Because of the almost linear increase in the prevalence of hypertension with age, and the possibility that secondary hypertension may develop at any time, recall patients should also have their blood pressure checked.

Significant benefits accrue from the participation of dentists in screening their patients for hypertension. Current statistics indicate that one death will be prevented for every 11 patients the dentist identifies as hypertensive who then receive treatment sufficient to reduce their mean systolic blood pressure by 12 mm Hg for 10 years.[12] Acutely, identification of the dental patient in hypertensive crisis may be life saving for the patient and protect the dentist against malpractice litigation.

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ORTHOSTATIC HYPOTENSION

Some patients with high blood pressure, especially the elderly, and those with diabetes mellitus or autonomic dysfunction, have an increased risk of developing acute hypotension when they stand after having lain in the dental chair for a length of time.[7,13] In addition, orthostatic hypotension is a known side effect of certain drugs used to treat high blood pressure, including the α-adrenergic blocking drugs, β-adrenergic blocking drugs, and other sympatholytic agents. The use of conscious sedation to reduce operative stress can exaggerate postural changes in blood pressure. Physical injuries in the form of broken bones and facial lacerations are common sequelae of the impaired consciousness and balance caused by the lack of effective cerebral circulation. In most cases, having the patient gradually assume more vertical postures after dental treatment prevents orthostatic hypotension.

XEROSTOMIA

Dry mouth is a common side effect of many drugs, including agents used in the management of high blood pressure. Centrally acting antihypertensives, diuretics, sympatholytics, angiotensin-converting enzyme inhibitors, and calcium-channel blockers have all been identified as causing xerostomia.[14,15] These effects are generally additive, so most patients with clinically significant xerostomia are usually on multiple medications. Oral complications associated with xerostomia include angular cheilosis, altered taste (dysgeusia), candidiasis, difficulty swallowing (dysphagia), painful, burning tongue (glossodynia and glossopyrosis, respectively), thirst, and increased caries. Because there is considerable variation in the degree to which different drugs cause dry mouth in specific patients, a useful strategy for alleviating persistent xerostomia is for the physician to seek an alternative antihypertensive regimen with less inhibition of salivation. Salivary stimulants in the form of gums or tablets containing nonsucrose sweeteners and/or citric acid may be helpful, along with oral moisturizers (plain water, salivary substitutes).[14,16] Sialogogues (pilocarpine, cevimeline) taken before meals can help provide the necessary saliva for mastication; topical fluorides are beneficial in reducing caries.

GINGIVAL OVERGROWTH

Calcium-channel blocking drugs, especially nifedipine, have been associated with gingival overgrowth.[15,17] The reaction occurs most prominently on the labial surfaces of the incisors. The enlargement is not a true hyperplasia but involves proliferation of noncellular

TABLE 3

Antihypertensive Drug Interactions in Dentistry

Antihypertensive	Dental Drug	Possible Effect	Recommended Action
Diuretics (e.g., furosemide, hydrochlorothiazide	NSAIDs (e.g., ibuprofen)	Decreased renal blood flow, loss of antihypertensive effect	Warn patient about possible interaction; use alternate analgesic if hypertensive response
	Epinephrine, levonordefrin	Transient hypokalemia	Consult physician; avoid use if patient is hypokalemic
β-Adrenergic receptor blockers (e.g., propranolol, metoprolol)	NSAIDs (e.g., ibuprofen)	Decreased renal blood flow, loss of antihypertensive effect	Warn patient about possible interaction; use alternate analgesic if hypertensive response
Nonselective β-blockers (e.g., propranolol)	Epinephrine, levonordefrin	Hypertension and bradycardia	Use cautiously; monitor blood pressure
ACE inhibitors (e.g., captopril)	NSAIDs (e.g., ibuprofen)	Decreased renal blood flow, loss of antihypertensive effect	Warn patient about possible interaction; use alternate analgesic if hypertensive response
Centrally acting α_2-adrenergic receptor agonists (e.g., clonidine)	CNS depressants, opioid analgesics	Increased CNS depression	Use cautiously
Peripheral adrenergic neuron blockers (e.g., guanethidine)	Epinephrine, levonordefrin	Increased cardiovascular responses to vasoconstrictor	Use cautiously; monitor blood pressure

ACE: angiotensin-converting enzyme
Modified from Yagiela JA, Turner RN.[21]

connective tissue elements of the gingiva. Local plaque accumulation and inflammation are involved in the etiology of gingival overgrowth, and meticulous oral hygiene after surgical resection of the enlarged tissues is effective in preventing recurrence. Switching to an alternative antihypertensive drug addresses the problem successfully without relying on the patient's skill with the toothbrush.

OTHER REACTIONS

Diuretics, ACE inhibitors, and several β-adrenergic blocking drugs may cause lichenoid reactions.[15,18] These lichen planus-like lesions are best managed by having the physician change the antihypertensive therapy. If that cannot be done, topical corticosteroids can be used to ameliorate the condition. ACE inhibitors have also been associated with loss of taste and a burning sensation in the mouth. More importantly, both ACE inhibitors and angiotensin II receptor blockers have been implicated in causing angioedema of the oral cavity in up to 1 percent of patients taking the drug.[19] Although edema of the tongue, uvula, and soft palate is most common, laryngeal edema is more serious because of the potential for loss of airway. The angioedema does not appear to be dose related; although most reactions occur early in therapy, angioedema has also developed after months of treatment. Epinephrine, antihistamines, and corticosteroids are used in the emergency management of acute reactions; switching to an alternative class of antihypertensives is required to prevent future attacks.

DENTAL DRUG THERAPY

Perhaps the most controversial issue regarding dental treatment of the hypertensive patient is the use of vasoconstrictors in local anesthetic solutions. (Opinion is fairly unanimous that gingival retraction cord impregnated with epinephrine should not be used in these patients.) The primary concern is the possibility of hypertensive crisis in uncontrolled hypertensive individuals and in hypertensive patients taking drugs that interact with vasoconstrictors. A systematic literature review of the cardiovascular effects of epinephrine during dental treatment found little evidence of risk in uncontrolled hypertensive patients, with mean maximum increases in systolic blood pressure of 15.3 mm Hg when 2 to 4.5 mL of lidocaine with epinephrine was injected, and 11.7 mm Hg when anesthesia without vasoconstrictor was used.[20] Corresponding values for normotensive individuals were 5.0 and 5.0 mm Hg. The diastolic blood pressure was little affected in

any group, nor were there any cases of adverse cardiovascular events.

These results suggest that one to three cartridges of 2 percent lidocaine with 1:100,000 epinephrine or its equivalent can be used safely in patients with uncontrolled hypertension. They are consistent with the fact that low concentrations of epinephrine decrease peripheral vascular resistance by selectively stimulating vasodilatory β_2-adrenergic receptors.[21] Doses beyond three cartridges, which are more likely to activate vasoconstrictive α-adrenergic receptors, have not been well studied. Because epinephrine once absorbed into the circulation is quickly converted to inactive metabolites, injections can be separated over time (e.g., by 30 minutes) to avoid cumulative effects of larger aggregate doses. Little information exists regarding the cardiovascular effects of levonordefrin in uncontrolled hypertensive patients; this fact, plus the greater tendency for levonordefrin to increase peripheral vascular resistance, argues against its use in these patients.

Specific drug interactions involving antihypertensive medications and drugs the dentist may use or prescribe are outlined in **TABLE 3**.[22] Interactions with epinephrine generally occur acutely after administration. In the case of hypertensive reactions (which may result in reflex bradycardia), injecting a small amount of epinephrine (e.g., the equivalent of 1 mL 1:100,000 solution), and monitoring the blood pressure five minutes later will assist the dentist in determining the safety of subsequent doses. Interactions with NSAIDs prescribed by dentists are slower to develop. Here, advising the patient to monitor their blood pressure at home will help uncover the rare case in which substitution with alternative analgesic therapy is warranted.

Management of the Hypertensive Patient

As a matter of course, dentists must provide for the dental needs of their hypertensive patients, including those with uncontrolled high blood pressure, associated diseases (cardiovascular and otherwise), and complex therapeutic regimens. In addition to the specific issues discussed previously regarding these patients, the dentist must be able evaluate these patients, deliver care safely, and respond appropriately to any episode of acute hypertension that might develop.

HYPERTENSIVE CRISIS as a result of invasive dental care gains clinical relevance with Stage 2 hypertension.

PATIENT EVALUATION

Patients with hypertension are often identified by their medical history. Hypertension is obviously an important medical disorder and should be specifically addressed in the medical history. Hypertension may also be identified by the medicines the patient reports taking. (Some patients with controlled hypertension, reasoning they are normotensive with treatment, do not indicate they have high blood pressure.) Medical diseases that are causative for secondary hypertension or increased cardiovascular risk should also be considered in evaluating the patient for hypertension. Consultation with the physician should be sought whenever the dentist needs clarification about the patient's physical status and/or ability to tolerate stress.

Dental patients with hypertension are also often identified by measurement of their blood pressure. Recording of a blood pressure in excess of 139 mm Hg systolic or 89 mm Hg diastolic in the dental office does not automatically mean the patient is hypertensive. The blood pressure recording may be elevated for technical reasons, which is discussed later, or because the patient is fearful or otherwise emotionally stressed in the dental office. Physician referral for all patients with high blood pressure in the dental office is necessary to help ensure they have the opportunity get their blood pressure under control if it is truly high. The actual diagnosis of hypertension will be made by the physician, often after multiple blood pressures have been measured over several weeks to months. Patients who suffer from white coat syndrome, a condition in which their blood pressures are consistently increased in the medical or dental office but not elsewhere, may have to keep a log of blood pressures taken during the course of daily life for an accurate diagnosis to be made.[23]

In general, the dentist should consider the severity of high blood pressure and the existence of other cardiovascular risk factors in making treatment decisions. Although Stage 1 hypertension poses serious cumulative danger to the patient over the course of years to decades, it is usually of little immediate consequence, and regular dental care is appropriate, with the possible addition of steps to reduce operative stress. Hypertensive crisis as a result of invasive dental care gains clinical relevance with Stage 2 hypertension. **TABLE 4** provides reasonable guidelines for treatment decisions based on the patient's blood pressure and the presence of related medical risk factors.[18,24]

TABLE 4

Dental Treatment Recommendations According to Severity of Hypertension

SBP	DBP	MRF	Recommendation
120-139	80-89	Yes/no	Routine dental care OK; discuss BP guidelines
140-159	90-99	Yes/no	Routine dental care OK; consider stress reduction protocol; refer for medical consult
160-179	100-109	No	Routine dental care OK; consider stress reduction protocol; refer for medical consult
160-179	100-109	Yes	Urgent dental care OK; consider stress reduction protocol; refer for medical consult
180-209	110-119	No	No dental treatment without medical consult; refer for prompt medical consult
180-209	110-119	Yes	No dental treatment; refer for emergency medical treatment
≥210	≥120	Yes/no	No dental treatment; refer for emergency medical treatment

MRF: medical risk factor (e.g., history of myocardial infarction, angina pectoris, high coronary disease risk, recurrent stroke prevention, diabetes mellitus, renal disease).
Modified from Merin RL,[23] after Herman WW, et al.[18]

BLOOD PRESSURE RECORDING

Accurate blood pressure recordings are fundamental to management decisions based on the patient's blood pressure. Although they are simple to obtain, attention to certain details is required. The mercury sphygmomanometer is the gold standard for measuring blood pressure, but concerns about mercury contamination have led to a greater use of (1) aneroid sphygmomanometers, which need calibration every six months to ensure accuracy, and (2) electronic devices, some of which are inexpensive but not very accurate, and others that are both expensive and accurate. Each of these devices requires a properly sized and fitted cuff. Size matters, because a cuff that is too small for the extremity will yield falsely high readings and oversized cuffs will give results that are too low. The length of the cuff bladder should equal at least 80 percent of the circumference of the extremity.[4] Although other recommendations have suggested that the cuff width equal 40 percent of the extremity circumference, studies have shown that this criterion does not produce accurate results and that a long cuff length is more important than a correct cuff width in providing accurate results.[25,26] Cuffs that are placed loosely or below the level of the heart will overestimate the blood pressure; the opposite occurs with cuffs that are positioned above the heart.

Falsely high blood pressures may be obtained if the patient has had exercise, nicotine, or caffeine within the past 30 minutes or is not allowed to sit quietly for five minutes before the recording is taken.[4] A quick estimate of systolic blood pressure can be obtained by feeling for the return of an arterial pulse as the cuff is quickly deflated from a high initial pressure. Then, the cuff can be reinflated to a value about 25 mm Hg above the estimated systolic blood pressure and then slowly deflated. Adequate precision is achieved with a bladder deflation rate of 2 to 3 mm Hg/second.

Sphygmomanometry estimates systolic and diastolic blood pressures by listening for the Korotkoff sounds, which result from turbulent blood flow within an artery squeezed by the inflated cuff. Systolic pressure is detected by the initial Korotkoff sound, which appears as blood first squirts past the cuff bladder, and the diastolic pressure is detected by the final Korotkoff sound, which heralds disappearance of discrete auditory pulsations. The disadvantage of this method is its inherent subjectivity. Error can occur because of deficiencies in sound transmission and hearing by the recorder. It is recommended that at least two recordings be taken and then averaged to determine the patient's blood pressure.[4]

STRESS REDUCTION

Hypertensive patients receiving dental care can benefit from treatment modifications designed to reduce physical and psychological stress.[27] Psychological support is especially important for individuals with significant dental fear. Treatment appointments should be scheduled so that the patient will not have to wait in the reception area and the dentist is free to give the patient undivided attention. Pharmacologic anxiety relief in the form of oral (e.g., with diazepam or triazolam) or inhalation sedation (with nitrous oxide and oxygen) should be considered. If fasting guidelines are to be employed, patients should be specifically told to take their antihypertensive drugs as normally scheduled.

Reducing the number of procedures to be performed automatically lowers stress. For example, performing periodontal surgery on two quadrants versus four should, on average, reduce by half the amount of local anesthetic solution that must be administered (and thus the number of injections), the duration of surgery, and the extent of postopera-

tive pain. Controlling pain is an essential treatment goal. Hypertensive patients should be instructed to advise the dentist if they are experiencing discomfort during the procedure, and that they can ask for a "time out" should they so desire. Responding to the patient's needs without delay is vital for the nervous patient to develop a sense of control over the situation.

Blood pressure recordings can provide an independent measure of the stress a patient is feeling and the effectiveness of strategies used to ameliorate it. A baseline blood pressure should be taken each time the hypertensive patient is about to undergo treatment. Subsequent recordings should be taken whenever the dentist suspects the patient's condition may have changed, such as when the patient requests a time out from treatment.

EMERGENCY MANAGEMENT

Acute elevations in blood pressure — for example, to a systolic blood pressure of 180 mm Hg or more or a diastolic blood pressure of 110 mm Hg or more — warn that the patient is experiencing some form of difficulty. Commonly, the underlying problem involves pain, intraoral, or otherwise. The patient may also be anxious, possibly, but not necessarily, as a result of discomfort. Other reversible causes of acute hypertension in the dental office include respiratory depression (in oversedated patients), a full urinary bladder, and vasoconstrictor drug interactions. The dentist should suspend the procedure as soon as the acute hypertension is discovered and seek treatable causes. It may be advisable to terminate the procedure if the blood pressure remains elevated. The patient should not be discharged home, however, until the pressure increase has abated or the patient's physician is consulted.

If a hypertensive crisis, as defined previously, develops, the dentist must stop working and devote full attention to the patient's physical status. Signs and symptoms indicative of a hypertensive emergency should be sought, and the blood pressure should be taken at frequent intervals (e.g., every three to five minutes). Emergency medical services should be requested if the dentist suspects a true emergency may be occurring. Telephonic consultation with the physician can be helpful in making this decision. With no evidence of developing organ damage, the patient may be referred for immediate follow-up care by the patient's physician, if available, or a local emergency medical clinic.

Unless the dentist is formally trained in the pharmacologic management of acute hypertensive reactions, as part of an oral surgery or dental anesthesiology residency, he or she should refrain from administering antihypertensive drugs without consultation from the patient's physician because of the dangers inherent in their use, including tissue ischemia caused by an excessive or too rapid reduction in blood pressure. In one exception to this rule, the dentist should administer sublingual nitroglycerin if the patient's symptoms includes anginal chest pain and there is no specific contraindication to its use.[21,27]

CONCLUSION

Hypertension is a common disorder that must be considered when developing an optimal dental treatment plan. With appropriate treatment modifications — including medical consultation when indicated, effective stress reduction, intraoperative blood pressure monitoring, careful local anesthetic and analgesic use, attention to intraoral manifestations associated with antihypertensive drugs, and avoidance of positional changes that may predispose the hypertensive patient to orthostatic hypotension — all but the most extremely hypertensive patients may be safely treated in the dental office. Detecting hypertension in undiagnosed patients and helping to ensure effective blood pressure control in all patients with hypertension are life-saving contributions that can be made by the treating dentist. ■■■■

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Calculating the Maximum Recommended Dose of Local Anesthetic

JOEL M. WEAVER, DDS, PHD

ABSTRACT Since patients have a wide variance in body size, it is appropriate to base the maximum recommended dose of a local anesthetic on a milligram of drug per kilogram of body weight. Other variables (severe overweight or underweight and cardiovascular compromise) also influence the appropriate maximum recommended dose. By calculating the specific dose limit from the maximum recommended dose for each patient, the chances of a local anesthetic overdose can be significantly reduced.

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The maximum safe local anesthetic dose should be individualized for each patient, based primarily upon their weight (mg drug/Kg body weight) and physical status. Although calculations based on the amount of body surface area may be more accurate, most of the current dosage guidelines are weight-based. For obese patients, the calculation should be based more closely on ideal body weight rather than actual weight. Excessive fat is so poorly perfused with blood that it does not readily participate as a drug reservoir to decrease blood levels of circulating local anesthetic molecules following their systemic absorption from a dental injection site. Severely underweight, starving, bulimic, or debilitated patients typically have low levels of circulating plasma proteins that normally bind a certain percentage of local anesthetic in the blood. These patients may be so cachectic that the fraction of anesthetic bound to blood plasma proteins, and thus held temporarily in an inactive state, may be unusually low. This decreased protein binding may result in unusually high blood levels of free, unbound drug that can cause overdose toxicity when the anesthetic diffuses from the injection site into the blood and rapidly into the brain and heart. Additionally, in these underweight patients, the reduced mass of skeletal muscle tissue that is normally well perfused with blood may markedly decrease the capacity of the muscle tissue to act as a temporary reservoir for the circulating local anesthetic molecules. Thus, without a significant skeletal muscle reservoir to help lower the blood level of the local anesthetic, higher levels of circulating local anesthetic molecules may perfuse vital organs and result in a toxic reaction. The maximum weight-based dose for these patients should

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TABLE 1

Maximum Limit for Lidocaine With Epinephrine for Healthy 3-year-old Child Weighing 33 pounds

- 2% Lidocaine/epi MRD range = 4.4 to 7 mg/Kg
- 33 lbs ÷ 2.2 lbs/Kg = 15 Kg body weight
- 15 Kg x's 7 mg/Kg = 105 mg maximum dose lido/epi (liberal limit)

  or
- 15 Kg x's 4.4 mg/Kg = 66 mg maximum dose lido/epi (conservative limit)
- 2% lido/epi = 36 mg lido/cart x's 3 cartridges = 108 mg (liberal max = Less than 3 cartridges)
- 2% lido/epi = 36 mg lido/cart x's 2 cartridges = 72 mg (conservative max = Less than 2 cartridges)

be lowered accordingly to prevent the hallmarks of toxicity such as loss of consciousness, seizures, respiratory arrest, and even cardiovascular collapse and cardiac arrest. Depending upon the site of the injection, the vascularity of the tissue and the presence or absence of a vasoconstrictor, these signs and symptoms of local anesthetic toxicity typically occur within a few minutes after completion of the dental injections that add up to too many milligrams of drug for the specific weight and health of the patient.

For patients with significant cardiovascular disease or those who take medications like nonspecific beta-adrenergic-blocking drugs (propranolol) that can adversely interact with epinephrine, the maximum safe volume of anesthetic may be limited by the amount of epinephrine rather than the amount of the local anesthetic itself. No more than 40 micrograms (40 mcg) of epinephrine is recommended for these medically compromised patients, which equals 2.2 cartridges of 1:100,000 or 4.4 cartridges of 1:200,000 epinephrine.[1]

Each local anesthetic has its own maximum recommended dose, expressed in mg/Kg. If a combination of two local anesthetics is given, the patient who receives an amount that is calculated to be one-half the maximum recommended dose of one anesthetic should only receive an amount calculated to be one-half the maximum recommended dose of the other anesthetic, since the toxic effects of each drug are believed to be additive.[2] Unfortunately, not everyone uses the same mg/Kg maximum recommended dose for each drug. One package insert recommends 4.5 mg/Kg for lidocaine without epinephrine and 7 mg/Kg for lidocaine with epinephrine.[3] Malamed's text, however, lists another manufacturer's recommendations as 4.4 mg/Kg and 6.6 mg/Kg, respectively.[1] Malamed recommended 4.4 mg/Kg for lidocaine with or without epinephrine, despite the fact that the vasoconstriction provided by the epinephrine reduces the peak blood levels of the anesthetic absorbed from the injection site.[1] None of these recommendations are incorrect, but rather reflect a decision of how conservatively one wishes to calculate the maximum recommended dose of their choice of local anesthetic. This may depend on the dentist's experience, training, and practice venue. A solo office practitioner in a remote area may decide to be more conservative and use a lower maximum recommended dose than the hospital-based dentist in the operating room, or a dentist working with a dentist anesthesiologist where intravenous access is already established and appropriate monitors, equipment, and sophisticated emergency drugs are already in place.

No matter what maximum recommended dose guideline a dentist uses to calculate the dose limit for an individual patient, the process for the calculation of the limit is as follows: Convert the body weight in pounds into kilograms by dividing the number of pounds by 2.2 lb/Kg. Thus, a 33-pound child weighs 15 Kg. Multiplying 15 Kg by the maximum recommended dose, 7 mg/Kg in the first example below, gives the maximum limit of 105 mg of lidocaine with epinephrine. Since each cartridge of 2 percent lidocaine contains 36 mg, three cartridges would equal 108 mg, just slightly over the maximum limit for this 33-pound child. Using Malamed's maximum recommended dose of 4.4 mg/Kg would yield a limit of 66 mg, slightly less than two cartridges (72 mg). A child weighing twice as much (66 pounds) could have twice the number of cartridges (**TABLE 1**).

A 1.8 ml dental cartridge of any 2 percent anesthetic contains 36 mg, while a cartridge of any 3 percent anesthetic contains 54 mg and that of any 4 percent solution contains 72 mg. The maximum recommended dose for 4 percent articaine with epinephrine happens to be the same as for 2 percent lidocaine with epinephrine (7 mg/Kg), so the maximum number of cartridges is reached twice as fast with 4 percent articaine compared to a 2 percent lidocaine. For the 33-pound child, the limit for 4 percent articaine would be approximately 1 to 1.5 cartridges. When calculating the number of milligrams of articaine in a cartridge, the actual volume is much closer to 1.8 ml rather than the 1.7 ml the manufacturer was forced by the Federal Drug Administration to place on the labeling because the volume of

some cartridges weren't always exactly 1.8 ml. Thus, one should calculate that an articaine cartridge has a volume of 1.8 ml.

For 3 percent plain mepivacaine and 2 percent mepivacaine with vasoconstrictor, Malamed's book lists the manufacturer's maximum recommended dose as 6.6 mg/Kg.[1] For the 33-pound child, 99 mg of 3 percent mepivacaine is the calculated limit, and at 54 mg/cartridge, that limit is reached with slightly less than two cartridges (108 mg). For the same 33-pound child, 99 mg of 2 percent mepivacaine with vasoconstrictor is still the calculated limit, and at 36 mg/cartridge, that limit is reached with slightly less than three cartridges (108 mg). Malamed recommended a maximum recommended dose of 4.4 mg/Kg for mepivacaine, so his limit for this child calculates to be 66 mg, which equals a little more than one 54 mg cartridge of 3 percent mepivacaine or slightly less than the 72 mg contained in two cartridges of 2 percent mepivacaine with vasoconstrictor.[1]

Prilocaine's maximum recommended dose, according to Malamed, is 6 mg/Kg (with or without epinephrine), although Yagiela suggested 8 mg/Kg.[1,4] If 7 mg/Kg is a reasonable average maximum recommended dose for prilocaine, the 33-pound child's limit would be about 1.5 cartridges of 4 percent prilocaine (15 Kg x's 7 mg/Kg = 105 mg total dose). At 72 mg/cartridge, 1.5 cartridges of the 4 percent drug equal 108 mg.

Malamed's text recommended a maximum recommended dose for bupivacaine with 1:200,000 epinephrine of 1.3 mg/Kg (but not more than 90 mg total dose) while one manufacturer recommended a limit of 225 mg for bupivacaine with epinephrine for nerve blocks in medical anesthesia, which equals a maximum recommended dose of 3 mg/Kg for a 70 Kg adult.[1,5] Although bupivacaine is not recommended by the FDA for dentistry for children under age 12, a 33-pound child could potentially tolerate a range of 19.5 mg to 45 mg of bupivacaine. Since 0.5 percent bupivacaine contains 9 mg/cartridge, which would translate into a range of two to five cartridges. Because it is a long-lasting anesthetic, toxicity with bupivacaine is also long-lasting, and overdosed patients are resistant to resuscitation efforts. Overdoses of bupivacaine often terminate in lethal central nervous system and cardiac failure. It seems wise for the typical dentist to use the conservative bupivacaine maximum recommended dose of 1.3 mg/Kg. for adults.

CONVERT THE body weight in pounds into kilograms by dividing the number of pounds by 2.2 lb/Kg.

Finally, there is minimal research data to provide an answer to the question of "How long after giving a maximum recommended dose of a local anesthetic can a dentist give more?" Some local anesthetics have active metabolites that contribute to local anesthetic toxicity, in addition to that of the parent compound. Lidocaine, for instance, produces the de-ethylated metabolites glycinexylidide and monoethylglycinexylidide, which are active compounds that can add to the toxicity of additional doses of lidocaine.[6] Because there is insufficient scientific data regarding the "time for safe re-dosing" in dentistry after a maximum dose has already been given, the conservative approach is to not exceed the maximum recommended dose in a single day.

It makes good sense to calculate the maximum dose of anesthetic for every patient, particularly for children, small adults, medically compromised patients, and anyone having extensive procedures, to prevent the tragic consequences of local anesthetic overdose toxicity. If done routinely for every patient, even for one who might need only a half cartridge, we would master the process, and then doing it for the most critical patients would be quick and easy.

Alternatively, a dentist could decide which maximum recommended dose he/she wishes to use for a particular anesthetic, then make a chart of various body weights with a corresponding maximum number of cartridges recommended, and post it in every operatory. That way, there is no excuse for not knowing how much is too much for each individual patient.

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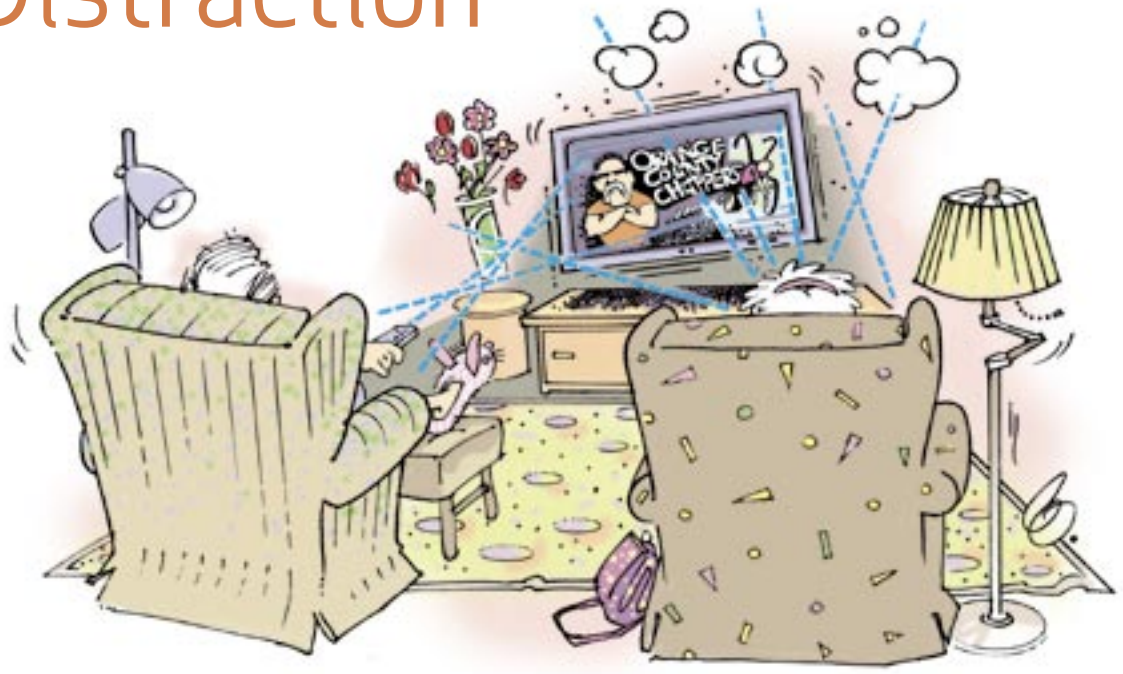
The Laws of Distraction



She has figured out, however, that in speaking softy, she carries a big stick.

Robert D. Horseman, DDS

ILLUSTRATION BY CHARLIE O. HAYWARD.

A body at rest tends to remain at rest.
— SIR ISAAC NEWTON

As a long-time admirer of Sir Isaac Newton (1642-1727), particularly his Laws of Motion, I have always tended to allow my resting body to remain at rest as long as possible. My wife is also a strong advocate of this same law and it is this mutual conformity that has generated a minor hitch in our conjugal bliss.

"Bob," I detected a faint voice emanating from another part of the house. My bride never raises her voice, not even in anger. A summons from six rooms away is delivered at the same volume as a face-to-face encounter. The message could be referring to a mouse seen in the wainscoting or the realization that World War III had just started. No difference.

Without waiting to determine if contact has been established, she then voiced several sentences, the essence of which escaped me entirely. I interrupted with "What?" vigorous enough to be heard next door at the neighbors, trusting this will have encouraged her to speak up. She repeated at the same decibel rating as her previous statements, and with the same predictable results.

I wished desperately to remain comfortably ensconced where I was, obeying Newton's law to the best of my ability. Sometimes I feel obliged to make up some sort of reply. Keeping my voice low, knowing she can no more understand me than I can her, I mouthed a couple of ambiguous remarks, thinking maybe she will materialize at my side to clarify things. This did not happen.

"Bob," she said again, repeating all or portions of what I assumed was the previous message. If I were to announce in no uncertain terms that I can't hear her, the battle is lost, Newton and I are defeated, and I must struggle up to go see what she wants.

This has been going on with minor variations for more than 50 years. You'd think that over that period another law would kick

CONTINUES ON 81

DR. BOB, CONTINUED FROM 80

in — the Law of Averages — and she would get up and come to me. Not once. My wife is not a yeller, and for that I should count my blessings. But it is an attribute that might improve her communication skills. She has figured out, however, that in speaking softy, she carries a big stick, namely to be the winner in the body at rest category.

In another area of marital discord we have reached a compromise. This happened because of a fundamental male/female difference in channel-surfing. When I occasionally have had command of the remote, I move very deliberately from channel to channel. I pause at each one long enough to determine if it contains something I wish to watch, like NASCAR or "National Geographic" specials. This takes me upward of three seconds per channel. My wife, however, has the ability to analyze, in depth, program content, audience appeal, and whether it's a repeat or not in the tiniest fraction of a second. She can go through a 75-channel search in less than a minute. As further proof of her ability to instantly divine the content of a program, she can arrive at a movie a half-hour after it has started and be able to understand everything that comes after.

So we got two remotes. As she races through the whole program spectrum, I wield my own remote to back up or go ahead. This duel of the remotes provides us with many hours of evening merriment without actually coming to blows. The television components that have to do with channel changing are given more work to do in a single hour at our house than would be encountered in a single-remote family in six months. The only time there is a clear-cut victor in these shenanigans happens when battery failure fells one of the contestants. Unless the winner can be conned into forfeiting his or her instrument, the moment is like an unconditional surrender. The loser slinks off to read a book or raid the fridge.

This duel of the remotes provides us with many hours of evening merriment without actually coming to blows.

The ultimate answer is, of course, another TV set. It would have to be identical to the first set. If its screen is even an inch bigger, trouble is inevitable. It should not be sited in the bedroom because the two-hour movie would always be playing there when the non-watcher wants to go to sleep. The bed-watcher can seldom be persuaded to get out of the comfortable bed and retire to the cold living room or den to catch the end of the film that usually finishes at midnight.

A serious student of social mores might conclude that each spouse's willingness to go it alone at the TV controls bodes no good for the future of the marriage. I disagree. With each spouse the master of his or her domain, the likelihood of physical abuse is greatly diminished — unless one of the mates insists on calling the other in a tiny little voice to get up and come look at what is obviously a superior program. Then a body in motion tends to remain in motion, and you know whose body it will be. I'd like to hear how Sir Isaac and Mrs. Newton worked this out. ■■■■