CDA

Acute and Chronic Pain
Local Anesthesia
Nitrous Oxide

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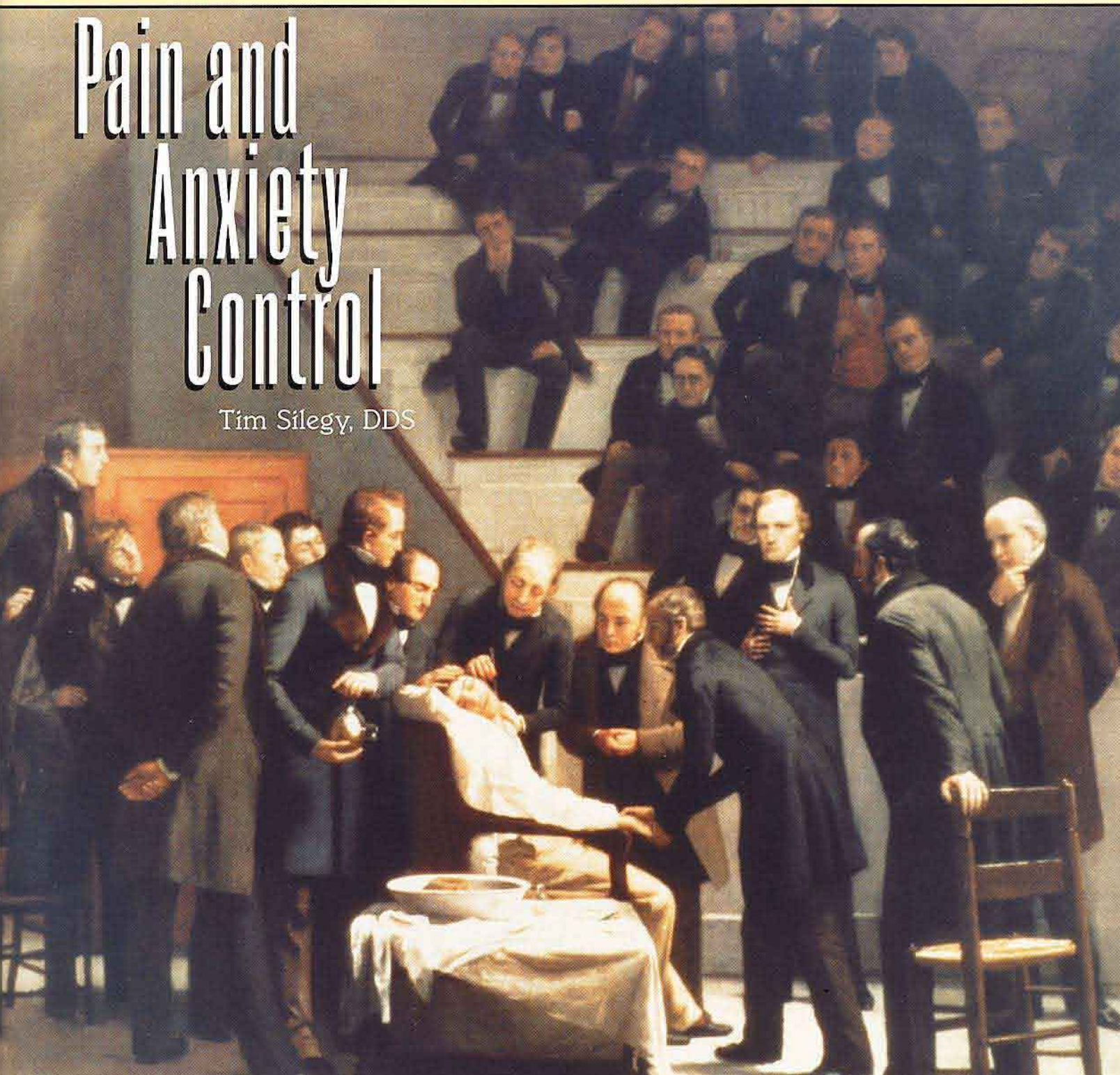
Pain and Anxiety Control

Tim Silegy, DDS



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DEPARTMENTS

FEATURES

CDA JOURNAL, VOL 31, Nº5

Saboteurs Within

Jack F. Conley, DDS

Dentistry can point to many standards and accomplishments, both old and new, in which it can take pride.

Strong codes of ethics and professional conduct are long-time standards that are at the very foundation of the profession. They remain at the center of the profession's efforts to govern itself and develop discipline and guidelines for its members. These standards have contributed significantly to the image of the profession.

Additionally, both individually and collectively, dentists have done much to advance efforts aimed at preventing dental disease.

Many dentists can take pride in their efforts to lift the image of the profession through endeavors to fluoridate community water supplies, contributions to community dental health education, participation in community dental health screenings, treatment of children in underserved communities in California or abroad, or direct charitable giving. A new example of this contribution was the recent "Give Kids a Smile" day, which brought dentists, dental students, dental staff, dental association staff, and many others together in a cooperative effort to provide care for countless numbers of children across the country.

Another effort that has grown mightily in recent years is charitable giving aimed not only at providing care for those unable to afford it, but also at advancing research efforts that will help to further control dental disease. Dentistry can be proud of its increasing contributions to oral public health, whether it has been the California Dental Association Foundation, the Pierre Fauchard Academy, the programs or projects of many other organizations such as Su Salud and AYUDA, or individual contributions that have supported dental care or dental research. These achievements are regularly detailed in dental publications. They are worthy of discussion by the profession when debate on dental health issues is before legislators.

However, beyond these limited audiences, dentistry's standards and accomplishments tend to become invisible to the public. And the public sector is where the saboteurs working within the profession seem to be regularly bashing the image that so many have worked so hard to establish and maintain.

Some of the saboteurs are licensed dentists, some of them are dentists not licensed in California, and others aren't dentists at all but, when located, are found practicing dentistry or managing practices!

The relative obscurity of the activities of these saboteurs makes it unlikely that they become accountable to the peer review and judicial review procedures of organized dentistry. Yet they ultimately become very visible to the public. Why? They make the headlines all too regularly in newsprint or on television. Their negative contributions to the public good seem to get more attention than the positives that the profession contributes. In the process, these saboteurs tear down the good image so many others continually work so hard to build, because the news stories that describe their deeds always include references to such familiar terms as dentistry, dentist, and dental clinics. The public in many instances is unable to separate these individuals from the profession they are demeaning by their villainous acts.

Of what then are these saboteurs guilty? They design and carry out entrepreneurial schemes that have an objective of fraudulently billing the Medi-Cal system (Denti-Cal) for care not delivered, or for care that is not appropriate or not delivered by properly trained individuals. Dentists who engage in these schemes are of course the worst offenders, as they violate the codes of their profession and are guilty of public fraud. It is equally distressing that some nondentists or unlicensed "dentists" plan or participate in these crimes that bring negative public attention to the dental profession.

Within the past few months, we noted two such reports in the public press. One involved a dental clinic owned and operated by a licensed dentist (who recently had became a new member of a component dental society) that employed a nondentist who produced much of the treatment that was billed to Medi-Cal. In eight months during 2002, investigators apparently uncovered $60,000 in fraudulent Denti-Cal billings before closing the office.

Less than 30 days later, another report titled, "Dental Clinics Charged With Bilking Medi-Cal" appeared. Eight individuals, most of whom the reader of this press report would presume were not dentists, were charged with bilking Denti-Cal out of $380,000 during their operation of four clinics.

Too often we have been hearing of fraudulent business practices in dentistry. New or young dentists, often shouldering heavy educational debt, are often targeted

and ultimately become victims of these schemes. They may become victims while engaged in a practice or even after they have left employment in these practices. Fraudulent Denti-Cal billings are often made using the license and Denti-Cal provider numbers of former dentist employees. Young dental graduates must become more vigilant in protecting their ID information, although in many cases it is extremely difficult when they have left an office to protect their personal information from illegal use by people who have previously gained access to it.

Due to the fact that some of the perpetrators of this activity are either unlicensed, or are not even dentists, there is no easy solution to controlling this problem for the dental profession. While dentists are directly responsible for many of these crimes, it is not an activity that the profession has a self-governance mechanism to identify. This fraud is invisible until suspicious practice billing trends, reports from patients or former employees, or suspicions of other dentists in the community lead to audits conducted by state investigators. There are apparently about two dozen active investigations involving suspected dental Medi-Cal fraud cases currently under way in California, according to the press reports we have reviewed.

Fraudulent Denti-Cal billing IS a black eye to the image of the dental profession. The dental profession does not have a good deterrent that will discourage these individuals who work quietly and invisibly until a billing trend is uncovered or a suspicion helps to bring them to justice. Potentially, the best deterrent may be better education of the young professional to identify and avoid this danger.

New members of the profession who are heavily in debt must be counseled or mentored on the importance of protecting their personal identities and resisting the temptation to engage in suspicious or risky business opportunities without carefully evaluating them. Only under those circumstances will we be able to remove the most gullible targets of these saboteurs at work within the dental profession.

Educational efforts will not eliminate the entire problem, but they will at least help to prevent well-meaning members of the profession from being victimized and prosecuted for crimes they did not consciously commit, a scenario that is too often encountered.

CDA JOURNAL, VOL 31, Nº5

Editorial Provides Inspiration

ROSEANN MULLIGAN, DDS, MS

I am hopeful that your editorial "Side by Side" in the February 2003 issue of the Journal of the California Dental Association (Page 97) will provide the springboard for renewed involvement in organized dentistry. It was copied and sent by the National Dental Society of the Bay Area (a component of the National Dental Association) to its members. We had no expectation that the CDA Journal would publish an enlightened editorial that avowed our history as African-American dentists and sincerely deplored the sordid period in our history when African-Americans were denied membership in the American Dental Association. The expressions from my colleagues emanated sensitivity, courage, morality, ethics, and vision.

As a member of the National Dental Association House of Delegates, I have observed the shift in relationship between ADA and NDA, moving from strictly congenial to collaborative. This improvement was initiated with the appointment of Dr. James Bramson as the executive director of ADA. This collaborative relationship motivated me to become a member of organized dentistry -- ADA, CDA, and the Alameda County Dental Society.

In the relatively short time I have been a CDA member, I have noticed that the organization is changing for the better. I joined CDA in time to witness Dr. Jack Broussard's championing of the strategic plan, which incorporated the ad hoc diversity committee's recommendations and adapted the leadership and members to the concept of inclusiveness. I await Dr. Debra Finney's assent to the CDA presidency, which will signal another empowering message that CDA is changing for the better with our first woman president.

Your editorial has challenged me, inspired me, and placed an onus of responsibility on me to assume an active member role. As you wrote in your editorial, there were African-American dentists who risked great humiliation to protest professional racial segregation so that African-Americans could join ADA. I owe a great debt of thanks to them and to the formidable and visionary ADA leaders who voted to ban all racially motivated membership restrictions.

As a member of the National Dental Association and ADA/CDA, I can embrace this juncture where "dentists of every ethnicity and background must stand side by side to vigilantly guard our profession." I must thank you for the eloquent and passionate reminder, which purveys the message of Dr. Edward Scott, former NDA president, "Let us focus on the common ground that unites us and not on the issues that divide us."

PAMELA ARBUCKLE ALSTON, DDS, MPP
OAKLAND, CALIF.

Good for a Horselaugh

Bob Horseman's piece in the February 2003 issue of the Journal ("Don't Make Me Take off My Belt," Page 174) was pure gold!

I am one of the 37 dentists sill alive who clearly recall the Doriot handpiece. And my wife is one of the four living registered dental assistants who remember it. I read the article to her in its entirety. This, by the way, was one of the few times in our marriage that I have been allowed to complete more than three sentences without interruption. My wife loved the article, and we had several good laughs together in the process. Thank you for helping to save our fragile marriage of 51 years.

I wish Bob Horseman many more years of health and happiness. Please keep those great articles coming.

GERALD L. VALE, DDS, MPH, JD
POWAY, CALIF.

CDA JOURNAL, VOL 31, Nº5

Grant Promotes Diversity of State's Dental Work Force

The California Endowment recently approved a $6.3 million grant to fund up to four California dental schools to increase the enrollment of minority and low-income students. The project also aims to improve access to dental care for underserved populations through dental resident and student rotations in community clinics and practices that provide care to disadvantaged patients.

In 2000, less than 5 percent of California dental students were African-American, Hispanic, or Native American, and only 8 percent of California dentists are from these minority communities. Oral Health in America: A Report of the Surgeon General pointed out the need to increase the diversity of the dental workforce and linked this to improving the health of minority populations.

California has 68 dental health professional shortage areas, many in rural areas. Along with financial and administrative limitations in public and private insurance programs, these access problems are the primary reasons for the low percentage of California Medi-Cal eligible children who visit the dentist.

To address these workforce and access issues, California dental schools that receive support from the Endowment will participate in Pipeline, Profession & Practice: Community-Based Dental Education, a nationwide, 11-school initiative started by the Robert Wood Johnson Foundation in September 2002. Dental schools eligible to apply for these $1.3 million grants are Loma Linda University, University of the Pacific, University of California at Los Angeles, and University of Southern California. The University of California at San Francisco School of Dentistry is one of the 11 schools in the Robert Wood Johnson Foundation's $19 million "Dental Pipeline" project.

"It is critical for one of the nation's most diverse states to have an equally diverse health care work force. We are pleased to join forces with the Robert Wood Johnson Foundation in this important endeavor, and look forward to increasing the number of minority and low-income students enrolled in California dental schools," said Jai Lee Wong, senior program officer for the Endowment.

"What a tremendous opportunity for the California Endowment and the Robert Wood Johnson Foundation, to mesh our individual philanthropic efforts into a focused area of concern," added Judith S. Stavisky, MPH, MEd, senior program officer at the Robert Wood Johnson Foundation.

The "Dental Pipeline" program office is based at Columbia University's Center for Community Health Partnerships under the direction of Allan Formicola, DDS, MS, at Columbia and Howard Bailit, DMD, PhD, from the University of Connecticut Health Center and Hartford Hospital. Kim D'Abreu Herbert, MPH, also at Columbia, serves as the program's deputy director.

"Since there is such a limited pool of minority and low-income dental school applicants, it will be critical for all of the Dental Pipeline schools to work collaboratively; not only to increase their minority recruitment and enrollment efforts, but also to review policies affecting dental education and the attractiveness of the dental profession to underrepresented students," Formicola said.

Bailit stated that "outreach by dental schools to underserved communities has a twofold purpose: It provides more care to disadvantaged patients and gives dental students and residents experience in caring for a diverse group of patients in community settings."

The Center for Community Health Partnerships is a resource center than enables physicians, dentists, nurses, and public health professionals at Columbia University to collaborate with community-based organization on projects that reduce health care disparities.

For more information about Pipeline, Profession and Practice: Community-Based Dental Education visit http://dentalpipeline.columbia.edu.

The California Endowment was established in 1996 to expand access to affordable, quality health care for underserved individuals and communities, and to promote fundamental improvements in the health status of all Californians. The Endowment has regional offices in Los Angeles, San Francisco, Sacramento, Fresno, and San Diego with staff working throughout the state. The Endowment makes grants to organizations and institutions that directly benefit the health and well-being of the people of California. For more information, visit their Web site at www.calendow.org

CDA JOURNAL, VOL 31, Nº5

Link Found Between Perio Disease and Oral Lesions

Dental researchers from the University at Buffalo have found a significant association between one measure of periodontal disease and oral precancerous lesions and tumors.

Analyzing data from the Third National Health and Nutrition Examination Survey, researchers found that people with serious periodontal disease were at double the risk of having a precancerous lesion and at four times the risk of having an oral tumor of any kind than people without serious gum disease.

Results of the study were presented at the American Association of Dental Research meeting in San Antonio in March.

"This is the first finding of a potential link between oral cancer and oral infection," said Sara Grossi, DDS, a co-author on the study, "but there is evidence of an infection link in other cancers." She noted research showing an association between Helicobacter pylori and stomach cancer, human papillomavirus and cervical cancer, and cytomegalovirus and Kaposi's sarcoma.

"The potential implications of this association of gum disease and oral cancer is enormous," said Grossi, clinical assistant professor of oral biology at the University of Buffalo School of Dental Medicine.

"Survival from oral cancer, as with most cancers, depends on the stage of the disease at diagnosis. If further studies demonstrate that periodontal disease is a significant risk and a warning sign, screening and examinations for oral cancer can be targeted in order to improve prevention and early detection of oral cancer."

NHANES III was conducted in the general U.S. population from 1988 to '94 by the Centers for Disease Control and Prevention. Physical examinations of participants included an assessment of oral health, including the amount of clinical attachment loss, a measure of gum detachment from the underlying bone, and a standard indicator of periodontal disease.

For this study, people in the NHANES III database who were at least 20 years old and had a minimum of six natural teeth -- a total of 13,798 -- were placed into one of two groups based on clinical attachment loss: less or more than an average of 1.5 mm for all teeth..

Researchers then determined the presence of oral tumors (any unusual growth), precancerous lesions, or any kind of soft tissue lesion (including canker sores, abrasions, redness, irritations, and general sore spots) in the two groups.

Results showed that oral tumors were four times more prevalent and precancerous lesions were twice as prevalent in people with periodontal disease (as assessed by clinical attachment loss) than in those without periodontal disease.

Researchers controlled for various conditions known to be risk factors for oral cancer, such as smoking, alcohol consumption, age, gender, race, education, occupation, diet, and number of dental visits.

"These findings suggest strongly that infection is associated with oral cancer," Grossi said, "but they don't prove that oral infection is causally related to oral cancer. If clinical studies prove that to be true, the implications for public health would be tremendous. Additional research in this area could significantly improve oral-cancer screening and early detection programs, and help reduce mortality from oral cancer."

On the Cover

The First Operation Under Ether (detail) Robert C. Hinckley

The First Operation Under Ether, an oil on canvas by Robert C. Hinckley (1853-1940), depicts the introduction of anesthesia into surgery. It is actually an amalgamation of the three operations that were required to convince the medical community of Boston that painless surgery was possible. The painting, known worldwide as one of the best depictions of a surgical operation, shows actual surgeons who attended at least one of the three surgeries, though not necessarily the first. The painting was completed in 1894 and acquired by the Boston Medical Library in 1903. Used with permission of the Boston Medical Library in the Francis A. Countway Library of MedicineInformation on OSAP conferences, newsletters, training programs, and other C.E. offerings is available at www.osap.org.

CDA JOURNAL, VOL 31, Nº5

Bilingual Helpline Connects Hispanic Families With Health Services

A new bilingual health helpline has been opened to help Hispanic families get basic health information to help them prevent and manage chronic conditions, and to refer them to local health providers and federally supported programs.

The National Hispanic Family Health Helpline Su Familia, (866) 783-2645 or (866) SU-FAMILIA, is open Monday through Friday, 9 a.m. to 6 p.m. Eastern Time. It was developed and is operated by the National Alliance for Hispanic Health and is supported by the U.S. Department of Health and Human Services' Health Resources and Services Administration and Office of Minority Health.

"Hispanics continue to face health disparities. This is unacceptable," Health and Human Services Secretary Tommy G. Thompson said. "That's why we are committed to getting information and resources to those communities where the health gap exists. By establishing the Su Familia helpline, we are helping families get access to the best health information."

Su Familia bilingual information specialists are able to refer callers to one of more than 16,000 local health providers, including community and migrant health centers, by using the callers' zip code. Callers can also request basic health information, referrals to information sources, or receive consumer-friendly, bilingual Su Familia fact sheets. Fact sheets are available for a wide variety of topics including asthma, cancer screening, cardiovascular disease, child and adult immunizations, diabetes, domestic violence, and HIV/AIDS.

Audiologists Say Dental Staff May Need Hearing Protection

Recent clinical experience and related research reveal that dentistry may be an at-risk profession for hearing loss, wrote audiologists at the Medical College of Georgia in an article in the December 2002 GDA Action, journal of the Georgia Dental Association.

They noted that dentists and their staffs have joined the ranks of others who are vulnerable to hearing loss: rock musicians, railroad and construction workers, and military personnel.

According to the National Institute for Occupational Safety and Health, noise-induced hearing loss is the most common occupational injury, the researchers write. The majority of the recent studies suggest that the noise levels experienced by the dental team have the potential to result in hearing loss and/or tinnitus (ringing or buzzing in the ears) for dentists and their staff who work with high-speed air turbine handpieces.

The articles cited one survey of California dentists that showed that every dentist who had purchased a high-speed handpiece also had some measurable degree of hearing loss, and that over half experienced moderate to severe tinnitus.

The researchers noted that loud noise levels in the dental environment can also affect patients. For some patients, the high noise levels can add to overall anxiety. Patients with sensorineural hearing loss often experience an acoustic phenomenon known as "recruitment," an abnormal perceptual increase in loudness due to the hearing loss. These patients, the authors said, may be extremely sensitive to even moderately loud sounds.

The audiologists suggest that hearing protection and conservation needs to become a regular part of dental office protocol, especially for those directly exposed to noise-generating equipment.

Treatment Acceptance Hinges on Relationship With Patient

Dentists need to understand more about the total care of patients, addressing not only their physical needs but also their emotional or psychological needs, wrote Cathy Jameson, PhD, in Dental Practice Management, winter 2002 edition.

The days of telling a person what they need and having them immediately accept have come to an end, Jameson wrote. Informed patients want to participate in treatment planning and decision making. She stressed that each person on the dental team plays a significant role in making effective presentations.

Case presentations provide the challenge of educating and motivating people. Jameson said the purpose of the consultation is to make it possible for patients to go ahead with the dental treatment.

Jameson says there are six steps in a successful case presentation:

* Build the relationship. Confidence and trust must first be established before people will accept a service

* Establish the need. To establish the need, careful and caring listening is required to understand how patients feel about their dental needs.

* Educate and motivate. These are ongoing but are most critical at the time of the consultation.

* Ask for a commitment. Jameson said it is necessary to ask for a commitment or people can walk out without deciding. Asking for a commitment also determines whether patients have any objections or barriers.

* Make a financial arrangement. This should be done before scheduling appointments, Jameson said.

* Schedule appointments.

Jameson said that dentists who follow this six-step process of case presentation will find acceptance rates will increase.

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CDA JOURNAL, VOL 31, Nº5

Tooth Loss Can Be Emotional Experience for Some Patients

A study by Canadian researchers found that 53 percent of partially dentate people found it difficult to come to terms with their tooth loss, according to an article in the November 2002 Oral Health.

The study investigated the reactions to tooth loss in a partially dentate group of 100 people using a questionnaire-based study.

The researchers found that partially dentate people who experienced difficulties in accepting their tooth loss were more likely than those who had no difficulties to feel less confident, restrict food choice, enjoy food less, avoid laughing in public, and avoid forming close relationships.

Of the 100 people in the study, 91 completed the questionnaire, 38 men and 53 women. In 78 percent of the cases, people had missing anterior teeth and 22 percent had only posterior teeth missing. Of the participants, 81 were wearing partial dentures and 10 were in the process of having their first partial dentures constructed.

When asked if they found it difficult to accept losing teeth, 53 percent reported difficulty accepting tooth loss and 41 percent said they had no difficulty accepting it. The overriding emotion felt by those who had no difficulty in accepting the loss of their teeth was one of relief. People who experienced difficulties expressed a wider and more complex range of emotions. The most common were sadness, depression, and feeling old.

The researchers also found that 51 percent of those who experienced difficulties with tooth loss felt that they had been unprepared for the effects that tooth loss had on them. Those people said that an explanation from the dentist would have helped.

Hygienists Speak up on What Makes Them Stay at Their Jobs

Dental staff members who think their employers are fair and generous are happier than those who don't, wrote Janyce Hamilton in the January/February 2003 CDS Review, journal of the Chicago Dental Society. And, Hamilton added, when staff is happy, retention and recruitment is not a problem.

Hamilton's article detailed the responses from hygienists across the country who were asked: What do dentists have to do to make their staff happy? The responses came in the form of first-hand stories of what the hygienists love about their employers.

In their responses, the hygienists noted many reasons for liking their employers, including:

* Excellent pay;
* Better-than-average benefits;
* Easy to talk to;
* Pride in their work and compassion for patients; and
* Day-to-day kindness and mutual respect.

Hamilton wrote that many of those who responded to the survey noted that small things matter. Little kindnesses do not go unnoticed, according to many of the respondents.

CDA JOURNAL, VOL 31, Nº5

Pain and Anxiety Control in Dentistry — This Is No Humbug

Tim Silegy, DDS

Contributing Editor

Tim Silegy, DDS, is a diplomate of the American Board of Oral and Maxillofacial Surgery. He maintains a private practice in Long Beach, Calif

Pain and suffering are an unfortunate part of human experience. It is also perhaps dentistry's greatest paradox.

Patients will refrain from seeing the dentist for years fearing the relatively minor discomfort of the dental injection. They will reject even the simplest of treatment plans stating, "If my tooth doesn't hurt, Doc, why bother?"

Paradoxically, the pain of a toothache (avoidable with regular preventive care) is frequently what drives our patients to see us.

For many, "pain" and "dentistry" are synonymous, so much so that this association has been incorporated into our popular vernacular.

A daunting task is "like pulling teeth," a difficult decision, akin to a root canal.

John Patrick's play The Teahouse of the August Moon chronicles the struggles of Okinawan villagers during the Second World War. The play ends with the protagonist making this statement. "Pain makes man think. Thought makes man wise. Wisdom makes life endurable."

I saw the play almost 20 years ago, and this closing statement has stayed with me ever since.

I am reminded of it almost daily, as I induce general anesthesia in an apprehensive patient for third-molar removal, see the look of relief in the eyes of a toothache patient after having received a mandibular block, witness the blank stare of a pediatric patient breathing nitrous oxide who was crying only moments before, or explain to a patient with facial pain that in spite of what others may think her pain is real and she is not crazy.

As dentists, we are judged not by our ability to carve tertiary anatomy into a shiny new amalgam, but rather by our ability to make the procedure painless.

It is appropriate, then, that we dedicate this issue of the Journal of the California Dental Association to pain and anxiety control in dentistry.

* Dentists utilize local anesthesia more than any other health professionals do. Dr. Alan Budenz reviews basic concepts of local anesthesia, introduces new agents and armamentaria, and gives protocols for managing the hard-to-numb patient.

* Drs. Stanley Malamed and Morris Clark review nitrous oxide and address myths that may be preventing its more frequent use. They make the case that with proper administration and well-maintained equipment, the nitrous oxide-oxygen technique has an extremely high success rate and a very low rate of adverse effects.

* Dr. Roger Kingston and I discuss the principal pharmacologic modalities that appropriately trained dentists can use to manage pain and anxiety in apprehensive dental patients.

* Dr. Scott Jacks and I review oral conscious sedation and demonstrate that it can be a safe and cost-effective alternative to intravenous sedation and general anesthesia for children who could otherwise not be treated.

* Dr. John Yagiela challenges traditional assumptions about the appropriate management of acute and postoperative dental pain by reviewing the literature on currently available oral analgesics used in dentistry.

* Patients suffering from chronic pain and headache can be found in all dental practices. Dr. Steven Graf-Radford reviews current methods of diagnosing chronic pain and discusses the physical, pharmacologic, and behavioral interventions currently used to manage it.

The work of these individuals reflects dentistry's ongoing commitment to reducing and, it is hoped, one day eliminating pain and anxiety in our patients.

It is a noble cause now, as it was 150 years ago when, in amazement, Dr. John Warren uttered, "Gentlemen, this is no humbug!" after having painlessly removed a tumor from a patient under the effects of ether anesthesia administered by, none other than a dentist, William T.G. Morton, DDS.

CDA JOURNAL, VOL 31, Nº5

Local Anesthetics in Dentistry: Then and Now

Alan W. Budenz, MS, DDS, MBA

ABSTRACT Local anesthetics have been in use in dental practice for more than 100 years. The advent of local anesthetics with the development of nerve blockade injection techniques heralded a new era of patient comfort while permitting more extensive and invasive dental procedures. A brief history and summary of the current local anesthetics available in the United States is provided, and some of the newest techniques for delivering local anesthetics are reviewed. General guidelines for addressing difficulties encountered in anesthetizing patients are also discussed.

AUTHORS

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The first local anesthetic agent to be widely used in dentistry was cocaine. Centuries before European exploration of the New World, Peruvian Indians had found that chewing leaves of the coca plant produced exhilaration and relief from fatigue and hunger. Following the import of coca leaves to Europe, much research was conducted to elucidate the properties of the coca leaf extract. In 1859, Albert Niemann refined the coca extract to the pure alkaloid form and named this new drug "cocaine." Niemann recognized the anesthetic effect of cocaine when he noted that "it benumbs the nerves of the tongue, depriving it of feeling and taste."1 In the summer of 1884, Carl Koller, a junior resident in the University of Vienna Ophthalmological Clinic, conducted experiments to test the topical anesthetic properties of cocaine on the corneas of various lab animals and on himself (self-administration being common in medical research at that time). He found that the drug rendered the corneas insensitive to pain. In September of that year, Koller performed the world's first operation using local anesthesia induced by topical cocaine on a patient undergoing glaucoma correction.2 The noted American surgeon William Halsted was the first person to inject cocaine for nerve conduction blockade, performing infraorbital and inferior alveolar nerve blocks for dental procedures in November 1884.3 Halsted subsequently developed numerous other regional nerve block injection techniques, many of which are still fundamental to dental practice.

Despite its promise for pain management during surgery, cocaine had major drawbacks, such as a high propensity for addiction and a short duration of action. The latter factor necessitated injection of large doses of the drug, increasing the potential for severe systemic toxicity. One technique developed to counteract this short duration/high dose problem was to apply a tourniquet near the operative site. In addition to the risk of local tissue damage, this approach had limited success in many regions of the body and was impractical for anesthesia of the oral cavity. In 1903, Heinrich Braun reported that epinephrine could be used as a "chemical tourniquet" when added to a

Table 1

Characteristics of Local Anesthetic Agents

Local anesthetic	Onset*	Protein binding (%)	Duration of pulpal anesthesia**	Maximum adult dosage***
2% lidocaine plain	Fast: 1 to 4 minutes	65	Short: 5 to 10 minutes	2.0 mg/lb
2% lidocaine with epinephrine	Fast: 1 to 4 minutes	65	Moderate: 60 to 90 minutes	3.2 mg/lb
3% mepivacaine plain	Fast: 1 to 5 minutes	75	Short: 20 to 40 minutes	3.0 mg/lb up to 400 mg total
2% mepivacaine with levonordefrin	Fast: 1 to 5 minutes	75	Moderate: 60 to 90 minutes	3.0 mg/lb up to 400 mg total
4% prilocaine plain	Fast: 2 to 5 minutes	55	Moderate: 10 to 20 minutes	4.0 mg/lb up to 600 mg total
4% prilocaine with epinephrine	Fast: 2 to 5 minutes	55	Moderate: 60 to 90 minutes	4.0 mg/lb up to 600 mg total
4% articaine with epinephrine	Fast: 1 to 6 minutes	95	Moderate: 60 to 90 minutes	3.2 mg/lb up to 500 mg total
0.5 bupivacaine with epinephrine	Moderate: 2 to 10 minutes	95	Long: 1.5 to 7 hours	0.6 mg/lb up to 90 mg total

*Time of onset: Individual variances are common. Lower number provided is average for infiltration injections; higher number is average for nerve block injections.

**Duration of pulpal anesthesia: Individual variances are common. Lower number provided is average for infiltration injections; higher number is average for nerve block injections.

***Maximum adult dosage: Maximum dosage should always be adjusted for the injection site characteristics (vascularity, tissue density), the injection technique used, and the physical status of the patient. Total maximum dosage numbers are for all patients regardless of other factors. The lowest possible dose required is always the safest.

solution of cocaine by producing localized vasoconstriction to slow the rate of vascular uptake, and thus reducing the required dose of cocaine.4 However, the drawbacks of cocaine were still significant, and research to find a synthetic substitute was widely undertaken. In 1905, Alfred Einhorn and his associates in Munich reported their discovery of procaine, an ester-based synthetic local anesthetic.5 Procaine was immediately accepted as a safe substitute for cocaine. Some historians consider the discovery of procaine to mark the beginning of the modern era of regional anesthesia. Several other ester-type local anesthetics were subsequently developed and remained in wide use in the United States throughout most of the 20th century.

In 1943, Nils Löfgren, a Swedish chemist, synthesized a new amide-based local anesthetic agent, derived from xylidine, and named it "lidocaine."6 Lidocaine was more potent and less allergenic than procaine and the other ester-based anesthetics. Since Löfgren's discovery of lidocaine, several other amide anesthetics have been developed for use in dental procedures: mepivacaine, prilocaine, bupivacaine, etidocaine, and articaine. The advantages of the amide-based anesthetic agents, particularly their very low rate of allergenicity as compared to the ester-type anesthetics, led to their gradual and complete replacement of the ester-based anesthetics in dental use. The last ester anesthetics packaged in a dental syringe cartridge were discontinued in the mid-1990s.

Current Dental Anesthetic Agents

Today's availability of a variety of local anesthetic agents enables dentists to select an anesthetic that possesses specific properties such as time of onset and duration, hemostatic control, and degree of cardiac side effects that are appropriate for each individual patient and for each specific dental procedure. Table 1 lists the anesthetic agents available for dental use in the United States and briefly summarizes their properties. It should be noted that these properties, particularly duration and depth of anesthesia, are only approximations and are variable due to a number of factors:7

* Individual variation in response to the drug administered;
* Accuracy in administration of the drug;
* Status of the tissues at the site of drug deposition (vascularity, pH);
* Anatomical variation; and
* Type of injection administered (supraperiosteal ["infiltration"] or nerve block).

Lidocaine

Lidocaine is considered the prototypical amide anesthetic agent. At its introduction in 1948, it was roughly twice as potent and twice as toxic as procaine, producing a greater depth of anesthesia with a longer duration over a larger area than a comparable volume of procaine. Consequently, lidocaine quickly became the most popular local anesthetic in dentistry. It is available in the United States in three formulations: 2 percent without vasoconstrictor (plain), 2 percent with 1:100,000 epinephrine vasoconstrictor, and 2 percent with 1:50,000 epinephrine.

Lidocaine without vasoconstrictor has a soft-tissue anesthetic duration of one to two hours, but a pulpal duration of only five to 10 minutes and is therefore of limited use for most dental procedures. Both formulations with the epinephrine vasoconstrictor have a pulpal duration of one to 1.5 hours and a soft-tissue range of three to five hours. The 1:50,000 epinephrine concentration may be advantageous for hemostasis in surgical sites but has no significant advantage for duration of pulpal anesthesia.

Mepivacaine

Introduced in 1960, a 2 percent solution of mepivacaine has potency and toxicity ratings roughly equivalent to a 2 percent solution of lidocaine. The greatest advantage of mepivacaine is that it has less vasodilating activity than lidocaine (all anesthetic agents without an added vasoconstrictor are vasodilators to some degree) and can therefore be used reliably as a nonvasoconstrictor-containing solution for procedures of short duration.7 Mepivacaine is available on the U.S. market as either a 3 percent plain solution or a 2 percent solution with 1:20,000 levonordefrin. The plain solution has a pulpal anesthetic duration of 20 to 40 minutes with a soft-tissue duration of two to three hours. The vasoconstrictor-containing solution has a pulpal duration equivalent to that of lidocaine with vasoconstrictor, that is, pulpal anesthesia for one to 1.5 hours and soft-tissue duration of three to five hours. It should be noted that although the levonordefrin vasoconstrictor in mepivacaine is less likely to produce cardiac side effects, such as palpitations, than is epinephrine, it is more likely to increase blood pressure and does have a higher potential for interaction with tricyclic antidepressants such as amitriptyline hydrochloride.8-10 At the time of this writing, levonordefrin production has been discontinued in the United States and existing supplies of mepivacaine with levonordefrin are expected to be exhausted by early to mid-2003. However, a potential new producer of levonordefrin is currently running production tests and may have mepivacaine with levonordefrin back on the U.S. market by mid to late 2003.11

Prilocaine

Prilocaine, also introduced in 1960, is slightly less potent and considerably less toxic than lidocaine as a local anesthetic agent. Like mepivacaine, prilocaine produces less tissue vasodilation than lidocaine and can be used reliably in plain solution form for short-duration procedures. Prilocaine is available as a 4 percent plain solution or as a 4 percent solution with 1:200,000 epinephrine. The plain solution has a pulpal duration of 40 to 60 minutes with soft-tissue anesthesia for two to three hours. It is worth noting that the duration of anesthesia with plain prilocaine is more dependent upon the type of injection given than are other anesthetics. Infiltration injections of prilocaine plain may only provide five to 10 minutes of pulpal anesthesia while regional block injections typically show the commonly described 40- to 60-minute durations. The vasoconstrictor-containing solution provides pulpal anesthesia for one to 1.5 hours like lidocaine and mepivacaine with a potentially longer soft-tissue duration of three to eight hours.7 Anecdotally, prilocaine has been said to have greater efficacy in patients who are difficult to anesthetize, for example, patients with a past or present history of substance abuse. An additional advantage is the decrease in cardiac side effects due to the lower vasoconstrictor concentration. Relative contraindications for the use of prilocaine include a patient history of methemoglobinemia, anemia, or cardiac or respiratory failure due to hypoxia.7

An additional precaution is raised by reports of a significantly increased risk of nerve paresthesia with the use of prilocaine and articaine, particularly for inferior alveolar and lingual nerve block injections.12,13 Haas, the lead author of a number of these studies, has speculated that chemical toxicity may be the cause of these increased paresthesias since the only common feature of prilocaine and articaine is that they are both 4 percent concentration anesthetic agents.14 His hypothesis is supported by reports of neurologic deficits with 4 percent lidocaine in animal studies15 and in human studies using 5 percent lidocaine for spinal anesthesia.16-18 This suggests that reduction of dosage to the absolute minimum amount required for effective anesthesia and the use of a slow, atraumatic injection technique with repeated aspirations are wise precautions if either of these anesthetic agents is selected for use with inferior alveolar and lingual nerve block injection techniques at all.

Bupivacaine

Bupivacaine is an analogue of mepivacaine that exhibits a fourfold increase in potency and toxicity and a remarkable increase in the duration of anesthesia. Released in the United States in 1983 and available only as a 0.5 percent solution with 1:200,000 epinephrine, bupivacaine may exhibit a slightly slower time of onset in some patients, approximately six to 10 minutes compared with two to seven minutes for lidocaine and mepivacaine.4 The longer duration of anesthesia for which bupivacaine is known is achieved primarily via regional nerve block injection techniques with mandibular blocks frequently having greater duration than maxillary blocks. As a block, pulpal durations of 1.5 to seven hours are common with soft-tissue anesthesia of five to 12 hours. When administered via infiltration technique, bupivacaine provides anesthetic depth and duration comparable to other local anesthetic agents.

Etidocaine

This long-acting amide anesthetic has been discontinued in the North American market.

Articaine

Articaine is an analogue of prilocaine in which the benzene ring moiety found in all other amide local anesthetics has been replaced with a thiophene ring. Al-

though not released in the United States until April 2000, articaine has been available in Germany since 1976 and in Canada since 1983 in a number of formulations. To date, only one formulation has been approved in the United States, a 4 percent solution with 1:100,000 epinephrine. With a higher per-cartridge unit cost and a pulpal anesthesia duration of approximately one hour with soft-tissue anesthesia for two to four hours, it would initially appear that articaine is a less attractive agent for dental applications. However, with a slightly faster onset of action (1.4 to 3.6 minutes19), reports of a longer and perhaps more profound level of anesthesia,20,21 and most notably frequent practitioner anecdotes of a greater ability to diffuse through tissues, articaine has become a very widely used anesthetic in the European and Canadian markets. The tissue diffusion characteristics of articaine are not well-understood; however, in a variable percentage of patients, a maxillary infiltration injection in the buccal vestibule will result in adequate palatal anesthesia for tooth extraction. Similar results have been claimed for the mandibular anterior and premolar teeth with buccal infiltrations.19 As discussed with prilocaine, reports of a significantly increased risk of nerve paresthesia with the use of articaine and prilocaine, particularly for inferior alveolar and lingual nerve block injections,12,13 warrants practitioner caution in the use of these anesthetic agents.

The Difficult-to-Anesthetize Patient

Many factors may affect the success of local anesthesia, some within the practitioner's control and some clearly not. While no single technique will be successful for every patient, guidelines exist that can help reduce the incidence of failure. For this discussion, a failure will be defined as inadequate depth and/or duration of anesthesia to begin or to continue a dental procedure. Due to a number of factors, such as thicker cortical plates; a denser trabecular pattern; larger, more myelin(lipid)-rich nerve bundles; and more variable innervation pathways,22-29 more problems of inadequate anesthesia occur in the mandibular arch than in the maxillary. Although failures are more common in the mandibular arch, maxillary failures do occur and can be equally frustrating.

Table 2

Common Accessory Innervations[20-30,34,35]

Tooth	Primary innervation	Possible accessory innervation
Maxillary molars and premolars	Molars: posterior superior alveolar nerve Premolars: middle superior alveolar nerve	Greater palatine nerve
Maxillary centrals, laterals, and cuspids	Anterior superior alveolar nerve	Nasopalatine (long sphenopalatine) nerve
Mandibular molars and premolars	Inferior alveolar nerve	1. Long buccal nerve 2. Mylohyoid nerve
Mandibular centrals, laterals, and cuspids	Incisive branch of inferior alveolar nerve	Mylohyoid nerve

The Maxilla

Most problems with maxillary anesthesia can be attributed to individual variances of normal anatomical nerve pathways through the maxillary bone (Table 2).30 While the pulpal sensory fibers of the maxillary teeth are primarily carried in the anterior, middle, and posterior superior alveolar nerves, which also supply the buccal soft tissues, accessory pulpal innervation fibers may be found in the palatal innervation supplied by the nasopalatine and greater palatine nerves.30 By careful application of topical anesthetics, distraction techniques (application of pressure and/or vibration), and slow delivery of the anesthetic agent, palatal injections can be given with very little to no patient discomfort. With the availability of articaine hydrochloride 4 percent with epinephrine in the United States, many practitioners are finding that palatal injections may not be necessary when it is injected into the maxillary buccal vestibule.20 Additionally, new computer-controlled anesthetic delivery systems are particularly adept at eliminating, or at least minimizing, the discomfort of palatal injections.31-33 Such systems are discussed in greater detail under New Delivery Systems and Techniques.

The Mandible

Problems with mandibular anesthesia are most common in the molar region but are by no means limited to these teeth.23-29,34 As in the maxilla, most anesthesia problems encountered in the mandible are due to individual variations in the nerve pathways, in other words, accessory innervation (Table 2).34,35

The first, and simplest, guideline relates to the extent of anesthesia achieved. If, for example, a patient reports profound anesthesia of his or her lower lip and tongue after receiving an inferior alveolar and lingual nerve block injection, but the tooth in question is still sensitive, it is probable that those two nerves have been successfully anesthetized and that the tooth sensitivity is very likely due to accessory innervation. This conclusion is based upon nerve morphology. Fibers near the periphery of a nerve bundle tend to innervate the most proximal structures, i.e., molars in the case of the inferior alveolar nerve; while fibers in the center of the nerve bundle tend to innervate the most distal structures, i.e., the incisors in this example.7 If a patient

reports that his or her lower lip and the tip of his or her tongue are anesthetized, structures that are innervated by the most central fibers of the inferior alveolar and lingual nerve bundles respectively, than it seems reasonable to conclude that these two nerves are indeed anesthetized and that accessory innervation to the sensitive tooth likely exists in this patient.

For mandibular molars, a common, and therefore important, accessory pathway to be considered is the long buccal nerve.27,36-38 This nerve branches from the anterior division of the mandibular portion of the trigeminal nerve high within the infratemporal fossa and crosses the anterior border of the mandibular ramus above the retromolar pad to enter and innervate the mucosa and overlying skin of the cheek, including the mandibular buccal attached gingiva. Due to the possible branching of this nerve as it descends along the medial surface of the mandibular ramus, a high injection site along the long buccal nerve pathway may offer a greater likelihood of successfully anesthetizing more of these accessory branches.26 Such a site for blocking the long buccal nerve is to inject into the soft tissue just medial to the anterior border of the ramus at or above the same level above the mandibular occlusal plane as the inferior alveolar block injection is given, i.e., using the depth of the coronoid notch anteriorly as the landmark for the horizontal level of the injection.4 An added benefit of this site is improved patient comfort by injecting medial to the anterior border of the mandible rather than into the lateral tissue.

An additional source of accessory innervation to any mandibular tooth is the mylohyoid nerve.23-25,28,39 This nerve arises from the inferior alveolar nerve at a variable level above the mandibular foramen and may not be consistently anesthetized with a conventional inferior alveolar block injection.40 Although it is anatomically described as a motor nerve innervating both the mylohyoid and the anterior belly of the digastric muscles, the mylohyoid nerve has been clearly shown to carry sensory fibers to mandibular teeth.28,39 A mylohyoid nerve block may be delivered by injecting into the floor of the mouth between the medial surface of the mandible and the sublingual fold formed by the sublingual salivary gland. The injection should be given just distal to the sensitive tooth, and the depth of the injection should approximate the root apices.41 An alternative technique to anesthetize the mylohyoid nerve is to administer a second inferior alveolar nerve block at a higher and/or deeper site.34 This may better approximate the origin of the mylohyoid nerve as it branches from the inferior alveolar nerve, but this technique does carry an increased risk of intravascular injection and possible hematoma.35,42

A potentially more efficient method for dealing with accessory innervations in the mandible is to use a more complete mandibular block technique such as the Gow-Gates43 or Vazirani-Akinosi44 techniques (Table 3). These injections, first described in the early 1970s, are given at higher sites on the mandibular ramus (Figure 1) and are aligned relative to the maxillary occlusal plane rather than the mandibular. Properly performed, these techniques have a very high success rate coupled with a very low risk of positive vascular aspiration.45 It should be noted, however, that even a high mandibular division nerve block technique, such as the Gow-Gates, may not have a 100 percent success rate in anesthetizing all possible nerve branches to mandibular tooth pulpal tissues.46,47 For this reason, the best advice is to be proficient with a variety of mandibular injection techniques as described in detail in the dental literature.

Another concern is the situation where anesthesia of all apparent nerve pathways is achieved, but the duration is short and/or the depth of anesthesia is poor. Giving a second injection into the same site as the first injection may prove adequate simply due to the increased volume of anesthetic solution. However,

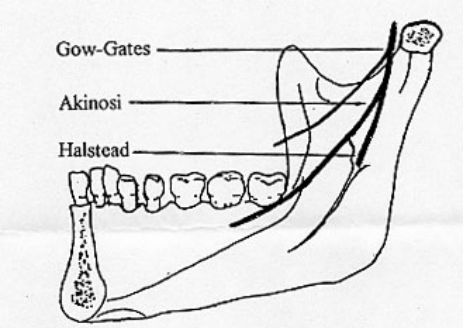

Figure 1 Sites for anesthetic solution deposition

using a different anesthetic agent for the second injection may increase the likelihood of successful duration. This difference may be explained by individual variances in tissue pH conditions and differing characteristics of each anesthetic agent, such as dissociation characteristics, lipid solubilities, and receptor site protein-binding affinities. No contraindication exists for using any of the amide anesthetic agents in combination with one another; however, care must be taken to limit the total dosage of anesthetic given to the maximum amount allowable for the agent with the lowest permissible dosage. For all injections given, the precise amount of each agent injected and the specific site of each injection should be recorded in the patient's treatment record. It is particularly helpful to note if one agent appears to have worked better than another. In these cases, this "better" agent should be used for the first injection at the next appointment.

The "Hot" Tooth

Anesthetizing the "hot" tooth, a condition generally indicating an irreversible pulpitis, can be one of the most frustrating problems for any dental practitioner. Whenever possible, prescribing antibiotic therapy to reduce inflammation and allowing the site to settle down may constitute the best course of action. When such a course is not an option, the first step in working through this situation is to de-

liver an appropriate nerve block injection as far back as possible along the innervation pathway of the hypersensitive tooth. If all of the surrounding soft tissues are numb, but the tooth itself is still sensitive, use of an intraosseous technique, which has a highly predictable success rate, is recommended.48-50 Less predictable, but also potentially effective, is a periodontal ligament injection technique.51-53 A last resort is to quickly access a pulp horn, creating a hole just large enough to insert a needle, and injecting anesthetic directly into the pulp chamber of the tooth. The major limitation of all three of these injection techniques is the inability to anesthetize multiple teeth with a single needle penetration and the relatively short duration of anesthesia achieved.53,54

New Delivery Systems and Techniques

In the past decade, two delivery systems have been developed that utilize computer technology in the administration of local anesthetics to patients. The Wand (Milestone Scientific) and the Comfort Control Syringe (Dentsply) both recognize that the more slowly an injection is given, the less traumatic it is to the tissues of the injection site and therefore the more comfortable the injection is to the patient. The Wand precisely controls the flow rate and modulates fluid pressure by use of a computer microprocessor and an electronically controlled motor to deliver the anesthetic solution at a slow rate regardless of tissue resistance.55 This allows the operator to deliver the anesthetic solution into any injection site, including the palate, at a rate that is potentially below the threshold of pain. An additional advantage is the smaller diameter of the syringe/handpiece itself, which permits the operator to use a more comfortable and stable pen grip on the syringe, allowing for more natural use of finger rests while injecting. The smaller size of the syringe may also be less intimidating to patients, a significant consideration when working with a dental-phobic patient.33,56-58 Disadvantages of the Wand system include the initial cost of the unit, approximately $1,400; the cost of the disposable syringe/handpiece assembly per patient, approximately $1; the longer/slower injection time; and, due to the volume of the tubing connecting the motor unit to the handpiece, only 1.4 ml of anesthetic solution can actually be delivered from each anesthetic cartridge.57 Additionally, the system does require some time to get accustomed to: The system is operated by a foot-pedal control, and the anesthetic cartridge is not directly visible in the operator's hand. This latter factor is addressed by a series of audible sounds that inform the operator of how much anesthetic solution has been delivered.

Introduction of the Wand delivery system has renewed interest in the palatal approach to anesthesia of the anterior and middle superior alveolar nerves.58,59 Using the palatal approach, anesthesia of the pulpal tissues of the maxillary incisor and premolar teeth, as well as anesthesia of the buccal and palatal gingival tissues, may be accomplished without the side effect of facial anesthesia found with the infraorbital nerve approach. Preservation of normal facial sensation and movement is an advantage for mid-procedure smile line assessment of maxillary anterior cosmetic procedures, and patient acceptance is an additional advantage. On a precautionary note, it is imperative that this injection be administered very slowly with constant visual monitoring by the operator to avoid excessive tissue blanching. The recommended injected volume is 0.6 to 0.9 ml administered over a 60- to 90-second, or longer, interval. If excessive tissue blanching is observed during the injection, a momentary pause to allow return of normal blood supply, indicated by return of pink coloration to the tissue, is recommended. A risk of palatal tissue ulceration must be recognized if marked ischemia occurs.58,59

The Comfort Control Syringe is a newer entry in the electronic, computer-controlled anesthetic delivery system market. This preprogrammed unit controls the delivery rate of anesthetic solution for a selection of injection techniques (block, infiltration, palate, PDL, intraosseous) preselected by the operator. Although bulkier than the Wand syringe/handpiece, the Comfort Control Syringe also enables the operator to use a pen grip while injecting. The Comfort Control Syringe houses the anesthetic cartridge directly behind the needle, just as in a traditional syringe; and the injection controls are fingertip accessible on the syringe rather than via foot pedal. The initial unit cost is approximately $900 with disposable supplies costing approximately 55 cents per patient.58-61

Although the technique of delivering local anesthetics directly into alveolar bone in close proximity to root apices is not new, recent technology has greatly improved the convenience of intraosseous injections. Systems marketed by Stabident, X-tip, and Intraflow have been incorporated into many dental practices. The intraosseous technique is quite reliable for pulpal anesthesia for one or two teeth and is particularly useful for anesthetizing the "hot tooth." Primary pulpal anesthesia using an intraosseous technique is effective in 45 percent to 93 percent of cases with short duration of approximately 30 minutes.54 When used as a supplement to an inadequate conventional infiltration or nerve block injection, the intraosseous technique is effective in 80 percent to 90 percent of cases with profound anesthesia of moderate duration (60 to 90 minutes).54

Intraosseous injections require a system for penetrating the cortical plate of bone so that the anesthetic agent may be injected into the cancellous tissue space from where it then diffuses to the desired root apices. The Stabident System (Fairfax Dental) is a two-part system with a separate perforator needle that mounts to a low-speed handpiece. The anesthetic injection needle is then passed through the perforation into the cancellous bone. One cause of difficulty with this system is the necessity of aligning the injection

needle precisely with the perforation channel to gain access to the cancellous space. This problem has been addressed in the Stabident System by adding a funnel-shaped needle guide that is inserted into the perforation channel. The X-Tip System (X-Tip Technologies) has also addressed this problem in its system design. The X-Tip is also a two-part system, similar to the Stabident, with the exception that removal of the perforator needle leaves a cannular guide for insertion of the anesthetic injection needle into the cancellous bone. The Intraflow System (IntraVantage) is based upon a special low-speed handpiece with a clutch and foot-pedal control system that permits perforation and injection with the handpiece in place, thus removing the need to switch from handpiece to syringe. The Intraflow handpiece system is about $900; the cost of disposable supplies is similar for all three systems, ranging from $1.50 to $2.

Because intraosseous injections are into the highly vascular cancellous bone tissue space, use of vasoconstrictor-containing anesthetic agents is generally not advised due to the rapid uptake of the agent into the circulatory system with a subsequent increase in patient heart rate.49,50, 62-64 In a number of studies, from 2 percent to 15 percent of patients reported moderate to severe pain during perforation, needle insertion, or injection of the anesthetic solution; and equal numbers of patients reported postoperative pain, swelling, or bruising at the injection site.54

A variety of electronic anesthesia systems have come and gone from the dental marketplace. Although these systems had their clinical successes, most practitioners found them frustrating to use in routine practice. The increased time for patient education about use of the system and the large variance in predictable anesthesia from one patient to the next, and even between different sites on the same patient, have ultimately led to their discontinued use. In general, the systems were only useful for relatively non-invasive procedures on a small percentage of patients.

Summary

What might be next on the front for dental anesthesia? As dental lasers continue to evolve and become increasingly refined, they may yet reach their early promise of providing "painless dentistry without the needle or the drill." Such an event will surely usher in a new era of patient comfort, potentially decreasing the number of dental-phobic patients. The prospect is truly exciting.

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Nitrous Oxide-Oxygen: A New Look at a Very Old Technique

Stanley F. Malamed, DDS, and and Morris S. Clark, DDS

ABSTRACT Inhalation sedation utilizing nitrous oxide-oxygen has been a primary technique in the management of dental fears and anxieties for more than 150 years and remains so today. Though other, more potent, anesthetics have been introduced, nitrous oxide is still the most used gaseous anesthetic in the world. Administered properly with well-maintained equipment, the nitrous oxide-oxygen technique has an extremely high success rate coupled with a very low rate of adverse effects and complications.

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Discovered in 1772, nitrous oxide was, for almost 70 years, a recreational drug. It was not until Dec. 10, 1844, in Hartford, Conn., at a traveling "popular science" exhibition that the potential of nitrous oxide to relieve pain was appreciated. On that night, "Professor" Colton demonstrated nitrous oxide. Attending the performance was a local dentist, Horace Wells. Wells noted that one of the men who had volunteered to inhale nitrous oxide had seriously injured his leg but was apparently unaware of any pain. The next day, a reluctant Colton served as the anesthesiologist as another dentist, Dr. John Riggs, extracted a wisdom tooth from Dr. Wells' mouth. After recovering from the effects of the nitrous oxide, Wells stated that he had been totally unaware of the procedure and that there had been no pain associated with it.1

Wells -- recognized by both the American Dental Association in 18642 and the American Medical Association in 18703 as the discoverer of anesthesia -- committed suicide while in jail on May 30, 1848, while under the influence of the anesthetic gas chloroform, to which he had become addicted.

Though other, more potent, anesthetics have been introduced, nitrous oxide remains the most used gaseous anesthetic in the world. It is commonly administered as a part of every general anesthetic technique for the purpose of enabling a lesser amount of a more potent (and usually more toxic) general anesthetic agent to be employed.

Surveys by the American Dental Association demonstrate that the percentage of American dentists employing nitrous oxide-oxygen (the procedure changed to include oxygen in the 1860s) is about 35 percent.4

Interestingly, nitrous oxide-oxygen has found an important niche in the area of emergency medicine.5 Under the proprietary names Entonox and Dolonox, nitrous oxide-oxygen (in a 40 percent to 60 percent ratio), is employed by paramedical

CDA JOURNAL, VOL 31, Nº5

personnel in the prehospital management of pain associated with acute myocardial infarction.6 In some areas of the world, it is used in emergency medicine in lieu of opioid analgesics for the management of painful injuries.

Technique

The technique for administration of nitrous oxide-oxygen inhalation sedation has changed little during the past several decades. One of the most important safety features associated with the technique is the ability of the dentist or dental hygienista to administer to a patient the precise amount of nitrous oxide required to provide the desired level of central nervous system depression (sedation). This ability is termed "titration," and it represents the most important safety feature of this technique. When titrated properly (Table 1) the success rate of nitrous oxide-oxygen inhalation sedation is extremely high. Unpleasant side effects -- such as nausea, vomiting, and bizarre behavioral responses -- do not occur when titration is performed. The administration of a fixed concentration of nitrous oxide-oxygen (for example 50 percent to 50 percent) routinely to all patients at each visit simply makes no sense given the ease with which titration may be carried out. The above-mentioned side effects are much more likely to be observed when titration is not employed.

The recommended technique of administration of nitrous oxide-oxygen inhalation sedation for the cooperative adult or child patient (a patient who willingly accepts the nasal hood) is presented in Table 1.7

Properly employing the technique described described above will allow the overwhelming majority of apprehensive dental patients to be successfully sedated, receiving their dental treatment in a much more comfortable and stress-free environment. Review of patient records from more than 29 years of administration of nitrous oxide-oxygen inhalation sedation at the University of Southern California

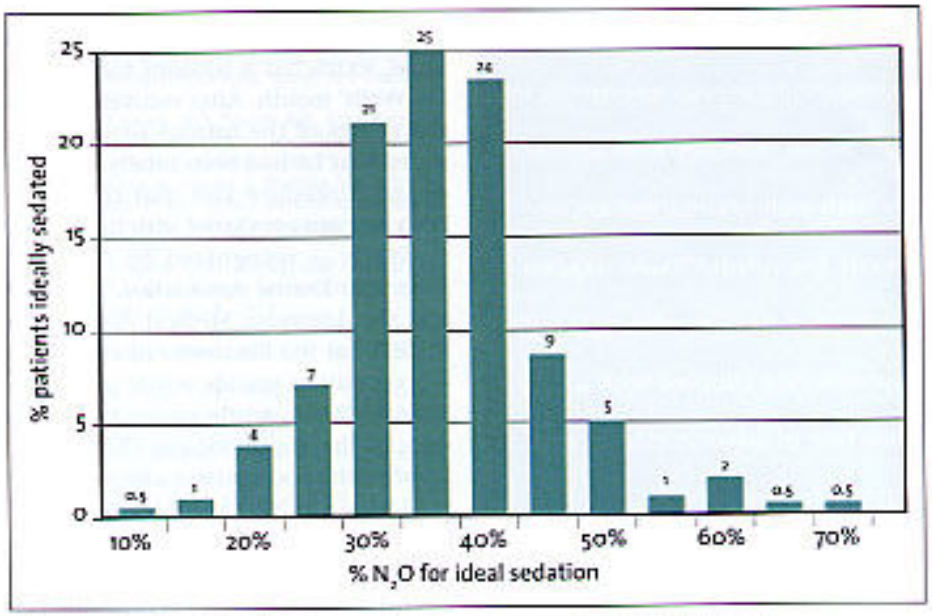

Figure 1. Normal distribution curve for nitrous oxide-oxygen inhalation sedation.

School of Dentistry demonstrates that the typical inhalation sedation patient (middle of the bell-shaped curve) requires from 30 percent to 40 percent nitrous oxide to achieve ideal sedation (Figure 1).7

It is important to note that there are patients at either end of this normal distribution curve who respond well to significantly lower concentrations of nitrous oxide (hyperresponders -- left side of the curve), while others require significantly higher levels of nitrous oxide to achieve the same clinical effect (hyporesponders). Titration enables the dentist to adequately sedate all of these patients.

In the precooperative pediatric patient (younger than 5) acceptance of the nasal hood is not likely. Indeed, with a screaming or crying combative young patient, it is difficult to employ inhalation sedation successfully. In addition to the difficulty in placing the nasal hood in this situation, the screaming/crying patient is primarily breathing through his or her mouth. In the hands of a trained pediatric dentist, an acceptable technique is placement of the nasal hood over the mouth of the screaming/crying patient. In this manner, the patient receives large amounts of nitrous oxide-oxygen, which eventually leads to his or her calming down, at which time the nasal hood is repositioned on the nose and treatment commenced. This procedure is repeated as often as needed.

The Equipment

Although the technique of nitrous oxide-oxygen delivery has changed little, during the past decades the inhalation sedation machine has undergone considerable revision. Despite the introduction of the ester-type local anesthetics (e.g., procaine hydrochloride) into dental practice in 1904, nitrous oxide continued to be administered as the sole agent in general anesthesia. The administration of 100 percent nitrous oxide produced what is called "anoxic anesthesia." It wasn't until the introduction in the late 1940s of the first amide local anesthetic -- lidocaine hydrochloride -- that the need for nitrous oxide to provide pain control disappeared. Langa demonstrated that nitrous oxide in combination with oxygen would provide excellent "relative analgesia," which in combination with local anesthesia provided the dental patient with pain-free treatment 8 The ability of

the inhalation sedation unit to deliver 100 percent nitrous oxide became a potential liability. The American Dental Association's Council on Dental Materials, Instruments, and Equipment adopted an Acceptance Program for inhalation sedation units that permitted the doctor to better evaluate those units being considered for purchase.9 The primary emphasis of this program has been the addition of safety features to these units aimed at making it difficult, if not impossible, to administer less than 20 percent oxygen to the patient. To receive a satisfactory classification, the manufacturer had to submit its devices to the Council on Dental Materials, Instruments, and Equipment for evaluation. The council publishes a listing of acceptable devices in the Journal of the American Dental Association, in the ADA Guide to Dental Therapeutics,10 and on the ADA Web site, www.ada.org.

The many safety features incorporated into the modern inhalation sedation unit are listed in Table 2.

Another significant, and more recent, change relates to the technology used to control the precise flow of gasses delivered through the inhalation sedation unit. Although the old flow tube flowmeter technology is still available, it is being replaced by the state-of-the-art digital electronic flow control devices, such as the Centurion Mixer and Digital MDM (Figure 2). Both of these devices are percentage devices and overcome the limitations of the older flow tube technology. The devices have resolution of the gas flow in increments of 0.1 liter per minute, and the total flow and percent of oxygen are displayed digitally, eliminating the guesswork or calculations required with simple flow tube devices. The ability to clean the front panel with just a wipe reduces the potential of cross patient infection, an issue associated with the crevices created by knobs and levers. Patient safety is ensured with built-in alarms for all gas depletion conditions along with servo control of the gas delivery (what you see is what you get). Continuous internal self-monitoring of all operational parameters by the device frees the practitioner to concentrate on the patient's needs. The device alerts the practitioner or staff to unusual parameters requiring attention, similar to those seen in larger hospital-based systems.

The digital units deliver pure oxygen during the "flush" function by electronically shutting off the nitrous oxide flow, as opposed to the flow tube units, which only dilute the nitrous oxide delivered. Again, the removal of extra steps in shutting down the nitrous oxide supply before pressing the "flush" button is removed and greatly simplifies the practitioner's tasks.

The units contain flashing LEDs to afford the practitioner a simple method of ensuring that the individual component gas is flowing and that the relative ratio and amount of flow is correct. Additionally, the digital unit provides the capability of displaying the flow rate of either of the constituent gasses. The nonsilenceable alarm function for oxygen depletion ensures patient safety. The air intake valve located on the bag tee provides room air to the patient whenever the patient's breathing demand is greater than the combined output of the mixer head's settings and reservoir bag volume.

Various models of the electronic gas mixing head allow mounting as a wall unit, portable unit, countertop unit, or as a flush-mount unit in modern cabinetry. Digital heads have the most flexibility, especially when combined with various remote bag tee options provided by the manufacturer. The units are fully compatible with central gas supply systems such as the popular Flo-Safe Manifold, Centurion Gas Manifold, and all existing scavenging systems. It is available with the American Dental Association recommended 45 liter per minute scavenging control valve in various mounting configurations. 11

Electronic digital administration heads for delivery of conscious sedation advance the art of dentistry. The digital heads once considered the wave of the future are the standard today. The digital accuracy and exacting control is highly recommended for patient comfort and safety.

Current Concerns About Nitrous Oxide

Several concerns have been addressed regarding the safety of inhalation sedation with nitrous oxide-oxygen inhalation sedation. These include the problem of abuse of nitrous oxide by health care professionals, sexual awareness related to nitrous oxide, and potential biohazards of chronic exposure to trace anesthetic gas.

Abuse of nitrous oxide by health care professionals: Nitrous oxide causes euphoria and, therefore, as Sir Humphrey Davy discovered in 1798, has a potential for abuse.12,13 This abuse is usually not as addictive as some drugs, but nonetheless can be a steppingstone to other drugs and can cause incapacitation of the affected person. Nitrous oxide should be given the same respect given to all drugs.14,15 When chronically abused, nitrous oxide can have serious health consequences.16

The typical abuser of nitrous oxide is usually older and probably from the middle to upper class. If he or she has an inhalation sedation unit available, it has probably been altered in an attempt to deliver a higher concentration of gas. A dentist living in Colorado placed a blanket over his head to increase the concentration even more. He became asphyxiated and could not be revived. Chronic inhalation (abuse) of nitrous oxide may lead to various neuropathies. This is particularly concerning if the loss of tactile sensation is associated with interference with their occupation, i.e., dentist. The neuropathy is generally reversible but can be permanent.

Nitrous oxide is used for mood alteration, sedation, and analgesia. It is the weakest of all general anesthetic agents. In the right circumstances, it has the potential to cause unconsciousness.

Sexual awareness related to nitrous oxide: There have been reports of sexual abuse of patients while under the influence of a variety of anesthetics.17-19 As expected, nitrous oxide has also been

associated with scattered reports of impropriety between male practitioners and female patients. Nitrous oxide does cause euphoria and, in high concentrations, dreaming hallucinations and, as described by Sir Humphrey Davy in 1798, "voluptuous sensations." The cases of record always involve three elements that place the practitioner at risk: treating a patient without an assistant in the operatory, high concentrations of nitrous oxide, and failure to titrate the patient to avoid the extension beyond their range of therapeutic sedation.

Nitrous oxide requires hosing that can drape around the shoulders for retention of the mask. It is important to allow the patient to adjust the mask on his or her face and to help the patient understand that it is connected to the hosing. The hosing on a euphoric patient can be misconstrued to be an inappropriate contact. Also, the patient should be allowed to fully recover. It may take longer than three to five minutes. Jastak and Malamed have reported a series of cases involving nitrous oxide and sexual phenomena.17 Malamed reports in an unpublished survey that a percentage of dental hygiene students reported increased feelings of sexuality and/or arousal while under the effects of nitrous oxide.20 They also reported some instances of orgasm. Nitrous oxide should be employed with confidence. Employing simple guidelines will ensure there are no difficulties with any sexual issues and the administrator of nitrous oxide.

Potential biohazards of chronic exposure to trace anesthetic gas: Nitrous oxide is found naturally in the atmosphere in minute quantities. It is quickly reversible in action, but is it totally harmless?

Little was known about the possible effects of inhalation of minute amounts of anesthetic vapors until the late 1960s. Until this time, little was done to eliminate anesthetic vapors being delivered into the ambient air from the anesthesia machines. In 1967, Vaisman21 published the results of a survey of Russian anesthesiologists in which it was demonstrated that they suffered a higher incidence of irritability, headache, fatigue, nausea, pruritus, spontaneous abortion, and fetal malformation than non-operating room personnel. It must be emphasized that in these studies nitrous oxide was but one of many gases under investigation. Because it is the most commonly used inhalation anesthetic, nitrous oxide will be found in all samples of air taken from operating rooms. It is used in conjunction with oxygen and other more potent inhalation anesthetics such as isoflurane, desflurane, and sevoflurane. Therefore, it has been impossible to separate the effects of any one of these gases from the others. Because of the special nature of dental practice, in which virtually the only inhalation anesthetic employed is nitrous oxide, the findings of these operating room studies were not applicable to the dental profession.

In the United States, Cohen and colleagues22,23 published articles dealing with anesthetic health hazards in the dental setting. One article contained a study that surveyed more than 50,000 dentists and dental assistants who were exposed to trace anesthetics. The results suggested that long-time exposure to anesthetic gases could be associated with an increase in general health problems and problems with the reproductive system in particular. While this study was retrospective in nature, it only fueled the concern regarding the safety of nitrous oxide in the dental office. Unfortunately, this "study" did not contain any measured data of these trace gases that were involved in any of the environs reported.

In 1974, Bruce, Bach, and Arbit24 investigated the possibility of nitrous oxide affecting perceptual cognition and psychomotor skills of personnel exposed to varying concentrations of the gas. They reported that just hours of exposure to as little as 50 ppm could result in audiovisual impairment. Despite multiple attempts to duplicate their results, all efforts failed. The National Institute of Occupational Safety and Health became interested in these studies and established 50 ppm as the maximum exposure limit for personnel in the dental setting. It was determined that 25 ppm was achievable in the operating room, and therefore this became the standard for that setting. Multiple attempts to reproduce the research results of Bruce, Bach, and Arbit have failed;25 interestingly, these researchers have retracted their conclusions, indicating the results were not based on biologic factors.26

The results of this "research," as one would expect, caused a concern and subsequent decline in the use of nitrous oxide. Indeed, there was alarm in the manufacturing and equipment industry for nitrous oxide that bordered on a crisis. In 1995, a worldwide literature search on the topic of biohazards associated with nitrous oxide use was conducted by Clark.27 Eight hundred and fifty citations were retrieved, of which 23 met the predetermined criteria for scientific merit. The conclusion drawn from this literature review indicated that there was no scientific basis for the previously established threshold levels for the hospital operating room or the dental setting. This research became the impetus for a meeting of interested parties representing dentistry, government, and manufacturing. A result of the September 1995 meeting, sponsored by the American Dental Association's Council of Scientific Affairs and Council of Dental Practice, was the formal position statement that a maximum nitrous oxide exposure limit in parts per million has not been determined.28

The specific biologic issue is the inactivation of methionine synthetase. This enzyme is linked to vitamin B-12 metabolism. Vitamin B-12 is necessary for DNA production and subsequent cellular reproduction. Nitrous oxide does affect methionine synthetase and does, in high concentration and with long exposure (longer than 24 hours), have an effect on reproduction.29 However, to date there is no evidence that a direct causal relation-

Figure 2. Digital electronic flow control device, Digital MDM. Photograph courtesy of Matrx (Orchard Park, N.Y.).

ship exists between reproductive health and scavenged low levels of nitrous oxide.30,31 Sweeney and colleagues32 were the first to link reproductive problems in humans with chronic nitrous oxide exposure. He used a sensitive test -- the deoxyuridine suppression test -- to accurately determine the first signs of this biologic effect in humans. Sweeney found that chronic exposure levels of 1,800 ppm of nitrous oxide did not exert any detectable biologic effect in humans. Sweeney suggests that 400 ppm is a reasonable exposure level that is both attainable and significantly below the biologic threshold.

Today, it is below the standard of care not to have a scavenging nasal hood.27 The scavenging nasal hood is a double mask: an inner mask contained within a slightly larger outer mask. The inner mask receives a fresh supply of nitrous oxide-oxygen from the inhalation sedation unit and delivers gas to the nose of the patient through tubes that are slightly larger in diameter than the other tubes. The outer, slightly larger, mask connects to slightly smaller tubes that connect with the vacuum system. On exhalation through the nose all exhaled gases are vented into the outer nasal hood and then, via the vacuum, are carried away from the patient and the treatment area.

Two of the most common causes of nitrous contamination in the office are from patients' talking and mouth breathing. The clinician should recognize this problem and attempt to modify the situation to make the interaction with the patient as brief and concise as possible. The use of a rubber dam is highly effective at decreasing trace nitrous oxide exposure.

Monitoring of trace nitrous oxide: The most accurate and effective method of determining nitrous oxide levels in ambient air is through an infrared nitrous oxide analyzer.33 The infrared analyzer can detect gases from the previously mentioned 1 ppm to an upper limit of 2,000 ppm. These devices are very expensive but can be rented from a gas service company. A gas supplier will have the resources available for dentists to rent an infrared spectrophotometer for nitrous oxide analysis.

In 1997, the American Dental Association published recommendations for responsible maintenance and monitoring of nitrous oxide and its equipment. They are listed in Table 3.28

Summary

Inhalation sedation utilizing nitrous oxide-oxygen has been a primary technique in the management of dental fears and anxieties for more than 150 years and remains so today.

Administered properly with well-maintained equipment, the technique has an extremely high success rate coupled with a very low rate of adverse effects and complications.

Notes

a. The administration of nitrous oxide-oxygen inhalation sedation by trained dental hygienists is permitted by some state dental boards

Table 1. Inhalation Sedation Technique[7]

1. A 5 or 6 Lpm (liter per flow minute) of 100 percent oxygen is established, and the nasal hood is placed on the patient's nose. The patient is instructed to adjust the mask as needed for comfort.

2. If necessary, the flow rate is adjusted (more, less, the same) while the patient is breathing 100 percent oxygen. The patient must be able to breathe comfortably, in and out, through his or her nose with the nosepiece in position.

3. A flow of nitrous oxide is started, at approximately 20 percent initially. Nitrous oxide is then added in ~10 percent increments every 60 seconds until an ideal sedation level* is reached.

4. When the patient states that he or she feels pleasant and more relaxed, the ideal level of clinical sedation* has been achieved.

5. Once the ideal level of sedation* is achieved, local anesthetic is administered and the planned dental/surgical procedure completed.

6. Nitrous oxide flow is terminated, and the patient is permitted to breathe 100 percent oxygen at a flow rate equivalent to the established Lpm for the patient. This may be started earlier than at the absolute completion of the procedure to ensure a more expedient recovery. Oxygen is given for minimally three to five minutes, longer if clinical signs of sedation persist.

7. The patient may be dismissed from the dental office unescorted if it is the doctor's belief that he or she is completely recovered from sedation.

* Ideal sedation has been achieved when the patient states that he or she is experiencing some or all of the following: feeling of warmth throughout his or her body, numbness of the hands and feet, numbness of the soft tissues of the oral cavity, a feeling of euphoria, and a feeling of lightness or of heaviness of the extremities. Note that not all patients will experience the same symptoms.

Table 2. Safety Features Incorporated Into Modern Inhalation Sedation Units

Alarm
Color coding
Diameter index safety system
Emergency air inlet
Lock
Minimum oxygen liter flow
Minimum oxygen percentage
Oxygen fail-safe
Oxygen flush button
Pin index safety system
Quick-connect for positive-pressure oxygen
Reservoir bag

Table 3. ADA Recommendations for Maintenance and Monitoring of Nitrous Oxide-Oxygen and Equipment[28]

1. The dental office should have a properly installed nitrous oxide delivery system. This includes appropriate scavenging equipment with a readily visible and accurate flow meter (or equivalent measuring device), a vacuum pump with the capacity for up to 45 liters of air per minute per workstation, and a variety of sizes of masks to ensure proper fit for individual patients.

2. The vacuum exhaust and ventilation exhaust should be vented to the outside (for example, through the vacuum system) and not in close proximity to fresh-air intake vents.

3. The general ventilation should provide good room air mixing.

4. Each time the nitrous oxide machine is first turned on and every time a gas cylinder is changed, the pressure connections should be tested for leaks. High-pressure line connections should be tested for leaks quarterly. A soap solution may be used to test for leaks. Alternatively, a portable infrared spectrophotometer can be use to diagnose an insidious leak.

5. Prior to first daily use, all nitrous oxide equipment (reservoir bag, tubings, mask, connectors) should be inspected for worn parts, cracks, holes, or tears. They should be replaced as necessary.

6. The mask may then be connected to the tubing and the vacuum pump turned on. All appropriate flow rates (that is, up to 45 L/min. or per manufacturer's recommendations) should be verified.

7. A properly sized mask should be selected and placed on the patient. A good, comfortable fit should be ensured. The reservoir (breathing) bag should not be over- or underinflated while the patient is breathing oxygen (before the administration of nitrous oxide).

8. The patient should be encouraged to minimize talking and mouth breathing while the mask is in place.

9. During administration, the reservoir bag should be periodically inspected for changes in tidal volume; and the vacuum flow rate should be verified.

10. On completing administration, 100 percent oxygen should be delivered to the patient for five minutes before the mask is removed. In this way, both the patient and the system will be purged of residual nitrous oxide. An oxygen flush should not be used.

11. Periodic (semiannual interval is suggested) personal sampling of dental personnel, with emphasis on chairside personnel exposed to nitrous oxide, should be conducted (for example, through use of diffusive sampler [dosimeter] or infrared spectrophotometer).

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An Overview of Outpatient Sedation and General Anesthesia for Dental Care in California

Tim Silegy, DDS, and Roger S. Kingston, DDS

ABSTRACT This paper reviews the principal pharmacologic interventions for the management of pain and anxiety in the apprehensive dental patient, including oral sedation, intravenous sedation, and general anesthesia.

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Disclosure

Dr. Silegy gives basic, advanced, and pediatric life support lectures for CME Associates in Orange, Calif., and is the director of the oral and maxillofacial surgery assistant's course sponsored by the California Association of Oral and Maxillofacial Surgeons in Roseville, Calif.

Access to dental care is a highly debated issue at present, as dentists, public health officials, politicians, and patient advocates examine the demographics and distribution of dental care in California.

One of the barriers to dental care for a significant population subgroup, possibly as large as 30 percent, is fear and anxiety.1 At best, dental patients who experience fear, anxiety, and hypersensitivity to pain will move in the dental chair and recall dental visits negatively. Most significantly, many patients will avoid needed dental care, consequently suffering unnecessary morbidity.

Perhaps dentistry's greatest contribution to humankind has been the introduction of anesthesia and anesthetic agents to dull or eliminate the physical and psychological effects of pain. Beginning in the 1840s with the popularization of ether by Dr. William T.G. Morton and nitrous oxide by Dr. Horace Wells, the interest of dentists in improving the dental experience is well-documented and voluminous.

In the 1960s, Southern California pioneers such as Drs. Nils Jorgensen, Oran K. Bullard, Adrian Hubbell, Howard Davis, Ed Boller, Lee Reeve, John Lytle, and Frank McCarthy introduced and refined intravenous sedation and general anesthetic techniques for in-office dentistry. These pioneers had concern for patient safety and advocated for safety standards and governing legislation.

Currently, the dental anesthesia faculties of the California dental schools teach undergraduate dental students, graduate students, and postgraduate practicing dentists to provide dental care with sedation and general anesthesia. The University of California at Los Angeles and Loma Linda University offer two-year residency training programs in dental anesthesiology.

Dental anesthesiology, while not recognized as a specialty by the American Dental Association, is a field wherein trained and qualified dentists limit their practice to teaching and providing anesthesia services for dentistry. Professional organizations such as the American Soci-

CDA JOURNAL, VOL 31, Nº5

Table 1

Training Requirements for Anesthesia Permits in California

Permit	Education	Other Cert.	Related C.E.
General anesthesia	4-year OMFS residency or 1-year anesthesia fellowship	BLS ACLS	15 hours biennially
Conscious sedation	1-year general practice residency or 60 hour course and 20 cases	BLS	15 hours biennially
Pediatric oral conscious sedation	1-year general practice residency or pediatric dentistry residency or 25-hour course with 1-hour patient contact	BLS	7 hours biennially

ety of Dentist Anesthesiologists and the American Dental Society of Anesthesia exist to promote excellence in dental anesthesia through education and research.2

Equipped with portable monitors, many dentist anesthesiologists provide a full range of anesthesia services, ranging from light sedation to general anesthesia, for children, adults, and individuals with special needs, in the offices of primary care dentists. This represents a considerable cost savings compared to treating these same patients in outpatient surgical centers and hospitals.

California Law

The Dental Board of California has implemented legislative regulations to establish minimum standards for training, facilities, and monitoring for most anesthetic techniques. Guidelines developed by the Southern California Society of Oral and Maxillofacial Surgeons served as a framework for this legislation.

Guedel was the first to describe anesthetic levels.3 His observation of patients undergoing ether anesthesia provides a basis for our understanding today. The American Society of Anesthesiologists, the American Dental Association, the American Academy of Pediatric Dentists, and others have defined anesthetic depth.4-6 Because airway protective reflexes are progressively depressed with increasing depth of anesthesia, more training and experience is needed to manage the potential complications associated with deeper levels of anesthesia. Therefore, the California Dental Practice Act defines three anesthetic levels and regulates their administration .7

Pediatric Oral Conscious Sedation

Pediatric oral conscious sedation is a minimally depressed level of consciousness in a minor patient, produced by oral medication, that retains the patient's ability to maintain independently and continuously an airway, and respond appropriately to physical stimulation and verbal command. "Minor patient" means a dental patient younger than 13.

Conscious Sedation

Conscious sedation is a minimally depressed level of consciousness produced by a pharmacologic or nonpharmacologic method, or a combination thereof, that retains the patient's ability to maintain independently and continuously an airway and respond appropriately to physical stimulation and verbal commands. Conscious sedation does not include conditions resulting from the administration of oral medications or the administration of a mixture of nitrous oxide and oxygen, whether administered alone or in combination with each other. The drugs and techniques used in conscious sedation shall have a margin of safety wide enough to render unintended loss of consciousness unlikely.

General Anesthesia

General anesthesia is a controlled state of depressed consciousness or unconsciousness, accompanied by partial or complete loss of protective reflexes, produced by a pharmacologic or nonpharmacologic method, or a combination thereof.

In California, all licensed dentists may administer local anesthesia and nitrous oxide-oxygen to adults and children. The same dental license also allows oral sedatives to be administered to patients 13 years of age and older. Dentists wishing to administer oral sedatives to children, intravenous sedation, or general anesthesia, require a special permit to do so. The requirements for these permits are listed in Table 1.

Indications

Conscious sedation and general anesthesia in the dental office are generally limited to healthy (ASA Class I and Class II) patients. When interviewing the patient at the time of consultation, in addition to reviewing a patient's medical history and performing a dental exam, it is important for the dentist to evaluate the patient's level of anxiety. Patients with a history of "bad" dental experiences in childhood, claustrophobia, low pain tolerance, or substance abuse are likely to benefit from some type of pharmacosedation.

Sedation is defined as a state of drowsiness or mental clouding. Frequently used

synonyms include anxiolysis or relaxation. The limbic system is responsible for awareness, and inhibition of this area of the brain results in a sense of tranquility and well-being. Hypnosis refers to central nervous system depression, principally of the reticular activating system, resembling normal sleep. Agents that produce these effects are collectively referred to as sedative-hypnotics. As in normal sleep, appropriately sedated patients may close their eyes and have dreams but can independently maintain their airway and be easily awakened.

Commonly Used Agents

The principal inhalational anesthetic agent in use today for sedation is nitrous oxide.8 The use of nitrous oxide is extensively reviewed elsewhere in this issue by Dr. Stanley Malamed. While some have advocated the use of halogenated hydrocarbons (desflurane and sevoflurane) to provide sedation, the need for anesthesia machines and vaporizers limits their use principally to outpatient surgical facilities and hospital operating rooms.9-10

A number of non-inhalational drugs have sedative and, in higher doses, general anesthetic properties. Benzodiazepines, barbiturates, opioids, dissociatives, and others are commonly used alone or in combination. These agents are summarized in Table 2.

Benzodiazepines

Benzodiazepines act on gamma-amino-butyric-acid receptor complexes in the central nervous system. When activated, these receptors increase the passage of chloride ion through discrete channels, hyperpolarizing nerves in the limbic system, which results in a sense of relaxation. Benzodiazepines have little analgesic effect and are frequently used in conjunction with opioids when used for intravenous sedation.

Diazepam was first synthesized in the early 1960s and commonly referred to as "mother's little helper." At one time, it was the most frequently prescribed medication in the United States.11 As is the case for all benzodiazepines, diazepam effectively reduces anxiety and is a profound amnestic. Diazepam is available as a pill or dissolved in propylene glycol for injection. Propylene glycol can irritate smaller veins and has been associated with phlebitis. Diazepam has a wide margin of safety and is effective both orally and intravenously. In the case of intravenous sedation, its long duration of action makes it a good choice for longer procedures. Diazepam is metabolized in the liver. An active by-product of this metabolism is oxazepam, which contributes to a reduced yet prolonged sedative effect. Care must be taken when using the drug on elderly patients, as they are typically much more sensitive to the sedative effect and tend to need a longer period of recovery.

Midazolam is about 2.5 times as potent as diazepam and has greater amnestic properties. It is water-soluble and is distributed as an aqueous solution for injection. It is also available premixed with cherry syrup for oral administration. Midazolam undergoes rapid redistribution and has a significantly shorter half-life than diazepam, making it an ideal agent for short procedures. Additionally, because it has no active metabolites, there is little "hangover" effect as is the case with diazepam.

Triazolam is a benzodiazepine designed principally to treat insomnia. It is distributed in pill form and has been found to be very effective when used for sedating anxious dental patients. Following oral administration, it has a peak

Table 2

Commonly Used Anesthetic Agents and Dosing Ranges

Generic name	Trade name	Oral dose	IM dose	IV dose (titrated to effect)	Wide margin of safety
Diazepam	Valium	Adult 2.5-10 mg Child 0.2-0.5 mg/kg	n/a	Adult 2.5-10 mg Child 0.04-0.2 mg/kg	Y
Midazolam	Versed	Child 0.5-1 mg/kg	0.07-0.15 mg/kg	Adult 1-5 mg Child 0.01-0.1 mg/kg	Y
Triazolam	Halcion	Adult 0.125-0.5 mg Child 5-20 mcg/kg	n/a	n/a	Y
Meperidine	Demerol	n/a	1 mg/kg	0.5-1 mg/kg	Y
Fentanyl	Sublimaze	n/a	n/a	0.5-1 mcg/kg	Y
Methohexital	Brevital	n/a	n/a	0.5-1 mg/kg GA	N
Propofol	Diprivan	n/a	n/a	1-2.5 mg/kg GA	N
Ketamine	Ketolar	n/a	3-5 mg/kg GA	1-2 mg/kg GA	N

*GA indicates dose indicated for general anesthesia only

effect at one hour and a half-life of two to three hours. Kaufman and colleagues found a 0.25 to 0.50 mg oral dose of triazolam taken one hour prior to treatment to be as efficacious as a mean dose of 19.3 mg of diazepam titrated intravenously.12 Baughman and colleagues found 0.5 mg of oral triazolam to produce significant amnesia.13

The administration of multiple doses of triazolam with early discharge after reversal with sublingual flumazenil has been advocated by several continuing education seminars.14 To date, no controlled clinical studies have demonstrated this to be a safe technique.

Opioids

Opioids exert their effect on the central nervous system by interacting with specific receptors in the brain and spinal cord blunting the response to excitatory neurotransmitters, namely acetylcholine and substance P (Table 3). Because the opioid agonists are not specific for any one receptor, the desirable effects of euphoria, sedation, and analgesia are frequently accompanied by nausea, dysphoria, and respiratory depression, especially in higher doses. Consequently, they are seldom used as a lone agent for sedation.

Opioid agonist-antagonists like pentazocine, nalbuphine, and butorphanol agonize some receptors but antagonize others. They were once popular because of self-limiting respiratory-depressant effects but are seldom employed by anesthesia providers today.

Meperidine hydrochloride and fentanyl citrate are the most commonly used opioids for intravenous sedation and general anesthesia. When used in conjunction with benzodiazepines, they produce a synergistic sedative result, effectively decreasing the overall dosage of drugs needed to elicit the desired effect.

About one-tenth as potent as morphine sulfate, meperidine is a relatively weak analgesic. When used intravenously it has a duration of 30 to 60 minutes. Unwanted side effects include histamine release, nausea, vomiting, and postural hypotension. As is also the case with fentanyl, meperidine should not be used in patients taking monamine oxidase inhibitors, as delirium and cardiovascular collapse have been reported.15

Table 3

Opiate receptors

Mu	Kappa
Euphoria	Sedation
Indifference	Meiosis
Respiratory depression	
Sigma	**Delta**
Dysphoria	Sedation
Mydriasis	Euphoria

Fentanyl citrate is perhaps the most commonly used opioid in anesthesia. It is 100 times more potent then morphine and has a duration of action much shorter than that of meperidine. Corresponding with increased potency is an increased incidence of respiratory depression. Too rapid an administration of the drug can also result in a spasm of the diaphragm and skeletal muscles commonly referred to as "stiff-chest syndrome." 16 This occurs rarely in sedative doses and can be easily managed by administering a muscle relaxant.

Barbiturates

Barbiturates, like benzodiazepines, act on the gamma-amino-butyric-acid receptor complex. They are available in pill form or as a powder reconstituted for injection. Prior to the synthesis of diazepam, they were used frequently as minor tranquilizers. Today, barbiturates are used mainly to induce general anesthesia. Sodium pentothal is commonly used in the hospital environment and was the first barbiturate to be used by oral and maxillofacial surgeons for third-molar removal.17 Its tendency to cause respiratory depression and long duration of action have limited its use in the dental office, and it was superseded by methohexital in the early 1960s.

Until last year, sodium methohexital was the most commonly used general anesthetic agent in the oral and maxillofacial surgery office. Its rapid onset and relatively short duration of action make it ideal for inducing general anesthesia for short procedures like third-molar removal. Respiratory depression, laryngospasm, hiccups, and resistance are common side effects that are easily managed by experienced practitioners. Due to a manufacturing shortage in 2002, many surgeons have switched to alternative agents such as ketamine and propofol.

Ketamine

Ketamine is a phencyclidine derivative that produces profound analgesia and amnesia. Unlike other anesthetic agents, it does not depress the reticular activating system; rather, it blocks transmission of sensory impulses from the reticular activating system to the cerebral cortex at the thalamus. Consequently, there is little respiratory depression; and protective airway reflexes remain intact. It is available in liquid form for intravenous and intramuscular injection and has been administered orally. Many practitioners are concerned with reports of hallucinations while emerging from the effects of the drug. This can be prevented with the simultaneous administration of a benzodiazepine.18 Ketamine has both sympathomimetic (increases heart rate and causes bronchodilation) and cholinergic (causes hypersalivation and bronchial secretion) effects. Consequently, an anticholinergic such as atropine or glycopyrolate is frequently administered concomitantly.

Propofol

Propofol is a di-isopropyl-phenol unlike any other anesthetic agent. It is highly lipid-soluble, which accounts for its rapid uptake and short duration of action. It is a potent amnestic and has been found to be a powerful antiemetic.19 In low doses, it is an effective sedative. Due to its rapid

redistribution, it is frequently administered as a continuous infusion using an infusion pump.

Anesthetic Technique

Oral Sedation

Oral (enteral) sedation is ideal for slightly anxious patients requiring traditionally nonpainful dental procedures such as cosmetic and restorative dentistry. The principal advantages of oral sedation include ease of administration, as there is no need for intravenous access, and favorable patient acceptance. Oral benzodiazepines such as diazepam and triazolam are particularly effective. When used in recommended dosages, they are very safe and carry with them very few direct contraindications. Midazolam is also an effective oral sedative; however, its use is chiefly reserved for premedication in children.

A major disadvantage of oral sedation is the inability to tailor dosages for individual patients. When taken orally, anesthetic agents are absorbed by the portal circulation and pass through the liver, where they are metabolized prior to entering the central circulation. This first pass effect makes it difficult to predict the bioavailability of the agent and, consequently, just the right dose. Drugs that are unstable in an acidic environment make be deactivated by gastric acid in the stomach.

As is the case for patients who receive parenterally administered anesthetic medications, dental patients who have taken oral sedatives should be driven to and from their dental appointments by a responsible adult and should not drive or operate dangerous machinery until fully recovered from the effects of the medication.

Intramuscular Injection

The intramuscular route of drug administration parallels oral administration. Agents undergo first-pass metabolism in the liver, making the end effect difficult to predict. The major advantage of intramuscular injection is that it requires little cooperation from the patient. Ketamine is an ideal intramuscular drug. With an onset of 10 minutes and minimal associated respiratory depression, it renders patients cataleptic, facilitating treatment or the establishment of intravenous access.

Intravenous Sedation

Contrary to popular opinion, the utilization of intravenous sedation does not directly provide for a deeper level of sedation than oral administration. The major advantage of IV sedation is that it allows the clinician to maintain precise control over the patient's level of sedation through the administration of small incremental dosages of medication, a technique known as titration. By titrating medications, a patient's level of consciousness can be carefully adjusted and the level of sedation controlled.

The procedure involves gaining intravenous access in the hand or forearm and maintaining it throughout the course of the procedure. Those individuals with "needle phobia" benefit from the use of topical anesthetics such as lidocaine prilocaine cream or spray refrigerants such as ethyl chloride. Patients are typically given supplemental oxygen via a nasal canula or nasal hood in conjunction with nitrous oxide. Their blood pressure and heart rate are monitored periodically and oxygen saturation is continuously monitored with pulse oximetry. A precordial stethoscope is commonly used to evaluate heart rate and breath sounds during the procedure.

Benzodiazepines are frequently used as initial agents. Small doses are administered until the patient's eyelids droop (Verril's sign) and his or her speech is slurred. An opioid may be added next to deepen the level of sedation and provide analgesia. While effective in reducing anxiety and blunting the pain response, IV sedation alone does not eliminate the need for local anesthesia. Consequently, care must be taken to ensure that the patient is numb prior to starting and throughout the course of treatment.

An added benefit of using a benzodiazepine-opioid combination for IV sedation is that in case of a medical emergency, both benzodiazepines and opioids are readily reversible. Flumazanil competitively displaces benzodiazepines from the gamma-amino-butyric-acid receptor complex. Naloxone is the reversal agent for opioid analgesics. Care must be taken to observe the patient after the administration of a reversal agent, as patients may become re-sedated if the half-life of the anesthetic agent exceeds that of the reversal agent. While propofol and ketamine have been used in low doses to provide sedation, it is the opinion of the authors that they do not have a margin of safety wide enough to render unintended loss of consciousness unlikely, and hence do not fall within the legislative guidelines of "sedative" agents.

General Anesthesia

General anesthesia is indicated for management of highly anxious and fearful (phobic) patients requiring restorative dentistry and for oral surgery patients undergoing procedures where local anesthesia alone would not provide sufficient comfort. This form of anesthesia is the most uniformly dependable anesthesia modality.

In the outpatient environment, a general anesthetic begins in much the same way as IV sedation. Because a deeper plane of anesthesia is expected, sedative medications may be administered more rapidly; and commonly a third agent is added to induce unconsciousness. This "balanced" anesthesia technique, provides anxiolysis, analgesia, and amnesia in a predictable and safe manner.

Dr. Adrian Hubble popularized the use of sodium methohexital for induction of general anesthesia in patients undergoing third-molar removal.20 The surge technique involves injecting an initial large bolus of the agent followed by frequent smaller boluses or "bumps."21 This technique is still practiced by many oral surgeons today when using sodium methohexital or propofol for third-molar anesthesia.

The use of a continuous small infusion of agent is another frequently used technique, being particularly useful for longer surgical and restorative cases. Following an initial large bolus, a slow intravenous drip, or an infusion pump, administers a continuous predetermined amount of an agent or agents, allowing the practitioner to maintain tight control on the level of anesthesia. Propofol and propofol/opioid and propofol/ketamine combinations have been effectively used to provide deep sedation and general anesthesia in the ambulatory setting.22-24

For short cases such as third-molar removal, endotracheal intubation is rarely necessary when office-based general anesthesia is employed. However, because the patient's protective airway reflex is obtunded, care must be taken to protect the airway. Placement of a 4-x-4-inch gauze as a pharyngeal shield (throat pack) is useful.

In cases where the dental procedure is expected to last longer than an hour, or when considerable water spray is going to be used, the airway can be protected with a laryngeal mask or by endotracheal intubation.

Regardless of the airway management technique employed, a dedicated person (other than the operator) must be present during a general anesthetic to manage the airway and monitor the patient.

Safety Concerns

Ambulatory outpatient sedation and general anesthesia in dentistry has an enviable safety record. Lytle and Stamper reported a mortality rate of seven deaths in 4.7 million office anesthetics administered from 1968 to 1987 by Southern California oral and maxillofacial surgeons.25 A similar survey of Massachusetts oral and maxillofacial surgeons found no deaths in 1.5 million office anesthetics during a five-year period.26

This exemplary safety record can be attributed to several factors.27 First, as previously mentioned, the sedation and general anesthesia modalities used in the dental community are generally reserved for healthy patients.

Second, most practitioners surround themselves with a highly trained team, thoroughly prepared and equipped to manage anesthetic emergencies. All personnel involved in patient care are required by law to have current training in basic life support. Practitioners holding general anesthesia permits are required to be current in advanced cardiac life support. Advanced training programs in anesthesia assisting for dental assistants are available from the American Association of Oral and Maxillofacial Surgeons and the California Association of Oral and Maxillofacial Surgeons.

Third, state law mandates that all conscious sedation and general anesthesia permit holders undergo a rigorous office anesthesia evaluation every six years. The office anesthesia evaluation was developed by the Southern California Society of Oral and Maxillofacial Surgeons in the late 1960s and was subsequently incorporated into the state Dental Practice Act. There are six components of the evaluation. The first four involve the inspection of the office physical plant, emergency equipment and monitors, emergency drugs, and records. The anesthetic team is then evaluated as common anesthetic emergencies are simulated. Finally, an actual sedation or general anesthetic is observed.

Other Options

Not all patients are good candidates for in-office sedation and general anesthesia. Patients with serious health conditions, young children with significant restorative needs, and individuals who are physically challenged, may be poor candidates for office-based treatment. Some hospitals have dental equipment that can be moved into an operating room and encourage dentists to join their medical staffs. Patients who could not otherwise have dental care -- such as those with severe physical, mental, or psychological disabilities -- can receive comprehensive dental care while anesthetized by the hospital's anesthesia service.

Conclusion

Although access to dental care is still an issue in California, no patient should find it impossible to receive needed dental care because of fear and anxiety. Many levels of anesthetic intervention can be provided by appropriately trained dentists. Available techniques include local anesthesia, nitrous oxide/oxygen analgesia, oral sedation, intravenous conscious sedation, and general anesthesia administered in the office, a surgical center, or a hospital. The challenge for every dentist is to formulate an anesthetic treatment plan to suit each patient's need. When dentists are consistently able to do this, patients will no longer have to fear dental treatment and our success as doctors and humanitarians will be elevated to a new, higher level.

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Pediatric Oral Conscious Sedation

Tim Silegy, DDS, and Scott T. Jacks, DDS

ABSTRACT As the young indigent population of this state grows, access to dental care continues to be a problem. Studies show that children from poor families suffer from a higher caries rate than those from a higher socioeconomic class. The management of pain and anxiety with intravenous sedation or general anesthesia in the young, precooperative patient, can be a significant adjunct to the delivery of dentistry. However, because children in this demographic group frequently lack the financial resources necessary for these treatment modalities, they will either not receive the necessary care because they are deemed unmanageable or will have a traumatic experience causing them to become even more resistant to future dental care. This article demonstrates how oral conscious sedation can be a safe and cost-effective alternative to intravenous sedation and general anesthesia in facilitating dental care for children who could otherwise not be treated.

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Disclosure

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Access to dental care for the children of California has become an important issue for both the California Dental Association and the California Society of Pediatric Dentists. The 2000 surgeon general's report on Oral Health in America confirms the need for action in this area. It states that "dental caries is the single most common chronic childhood disease -- five times more common than asthma and seven times more common than hay fever."1

The executive summary of the report also states that "the social impact of oral disease in children is substantial." Nationally, "more than 51 million school hours are lost each year to dental-related illness. Poor children suffer 12 times more restricted-activity days than do children from higher-income families. Pain and suffering due to untreated diseases can lead to problems in eating and speaking and attending to learning."1 Another significant finding of the report is that "25 percent of poor children have not seen a dentist before entering kindergarten."1 Hence, the child's first dental experience is frequently unpleasant because it involves major restorative dentistry.

Managing the dental needs of the pediatric population imparts a unique challenge to the dental practitioner. He or she must not only be skilled at diagnosing and treating the deciduous and succedaneous dentition, but also be able to assess and address the emotional and behavioral status of children. Many prekindergarten children are not capable of sitting for protracted periods. This fact makes them poor candidates for restorative dental procedures and represents a troubling issue. These children, ranging in age from 2 to 4 years, are developmentally at a precooperative age.2 The typical attention span of such a young child is four to eight minutes.3 In addition, many find restorative dentistry emotionally stressful; and research has shown that children of this age, when placed in emotionally stressful situations, tend to regress, further impeding the ability of the dentist to perform dentistry effectively.

CDA JOURNAL, VOL 31, Nº5

Some of these young patients with extensive dental caries require general anesthesia to facilitate treatment. Others, with no complicating medical conditions, can be ideal candidates for intravenous sedation. IV sedation and general anesthesia are expensive, ranging in cost from hundreds to thousands of dollars, before a penny is spent restoring the mouth. In many instances, these financial issues further block access to care.

Oral conscious sedation is another, significantly less expensive, option. An appropriately trained and permitted dentist (see Silegy and Kingston in this issue) can safely perform this procedure in the office. Children, who may not be able to tolerate restorative procedures with nitrous oxide-oxygen and local anesthesia alone, are frequently ideal candidates. Oral sedation enables the dentist to perform the necessary dental treatment with minimal stress to the patient and dental team. In some instances, it may not be the treatment of choice; but when appropriate, oral sedation may make the difference between treatment and no treatment at all.

Preoperative Evaluation

Not all children are good candidates for oral sedation. Successful management of pediatric dental patients requires the dentist to have an understanding of age-dependent behavior, medical conditions that could complicate sedation, and the complexity of the anticipated dental treatment.

Behavioral Evaluation

During the initial exam, it is important for the dentist to evaluate and classify behavior so that an estimate of the child's cooperative ability can be determined. This determination will assist the treating dentist in deciding whether the patient is a candidate for nonpharmacologic intervention, oral conscious sedation, IV sedation, or general anesthesia. Numerous systems have been developed to accomplish this task. Two prominent classification systems have been developed by Wright and Frankl.4

Wright's system places children into one of three categories based upon their behavior:

* Cooperative;
* Lacking in cooperative ability; or
* Potentially cooperative.

Frankl's behavior rating scale divides behavior into four categories:

* Rating 1 -- definitely negative;
* Rating 2 -- negative;
* Rating 3 -- positive; and
* Rating 4 -- definitely positive.

Some dentists develop their own scale and use it to evaluate the behavior of young children in the dental setting. No matter what system a practitioner uses, the essential issue is that he or she documents preoperative behavior and considers it when formulating the treatment plan.

Medical Evaluation

The medical condition of the pediatric patient can have a profound effect on the dental treatment plan. To be considered for oral sedation in the dental office setting, children should be free of systemic disease (ASA Class I) or have a well-controlled medical condition such as mild asthma or diabetes (ASA Class II).

Because most cases of morbidity and mortality associated with pediatric oral conscious sedation involve airway and/or respiratory complications, it is imperative that special attention be paid to these areas.5 The most common acute medical condition affecting young children is the upper-respiratory tract infection or common cold. Preschool-aged children suffering from an upper-respiratory tract infection are more prone to complications because they frequently are obligate nose breathers.8 The hypersecretion and edema associated with an upper-respiratory tract infection can dramatically diminish their ability to keep their airway clear, especially after having received a sedative and local anesthetic. Additionally, nitrous oxide-oxygen administered via a nasal hood, will have little effect on the child with nasal congestion.7 In this instance, treatment should be deferred for two weeks from the cessation of symptoms.8

Dental Evaluation

Necessary dental procedures should be categorized based upon the anticipated time needed for their completion. The authors believe that the child whose dental work can be completed in an hour or less makes the best candidate for oral sedation. Children requiring significantly more chairtime might be better served by an IV sedation, general anesthetic, or additional appointments using oral sedation.

Standardized treatment protocols that consider the child's behavioral, medical, and dental evaluation can aid the practitioner in determining the best course of treatment.

Agents and Techniques

Many agents and techniques have been used to sedate the pediatric patient.9,10 The administration of oral sedative medications is generally well-tolerated by children. While most agents are unpleasant to taste, when mixed with sweetened drink powders or juice, they are generally palatable to patients, particularly when thirsty from preoperative fasts.

While effective, oral sedation is much less predictable than intravenous sedation. When a sedative agent is administered intravenously, the plasma concentration rises quickly to elicit an immediate dose-dependent response. The same agent, when administered orally, may be subject to deactivation in the highly acidic environment of the stomach. Upon passing into the small intestine, there is a generally rapid uptake of the agent into the portal circulation. In the liver, a significant portion is metabolized by the cytochrome p-450 complex (phase I metabolism), conjugated with glucuronic acid (phase II metabolism), and transported to the kidneys, where it is excreted in the urine.11 Consequently, after a considerable delay, only a fraction of the administered agent enters the plasma.

A list of agents commonly used for pediatric oral conscious sedation in dentistry

Table 1

Commonly Used Oral Sedatives

	Chloral hydrate	Diazepam	Midazolam	Meperidine	Hydroxyzine
Dispensed	500 mg capsule 250 mg or 500 mg/5 ml syrup 324 mg, 500 mg or 648 mg suppository	2, 5, 10 mg tabs 5 mg/ml	5 mg/ml 1 mg/ml 2 mg/ml syrup	50 mg tab 50 mg/5ml	10, 25, 50 mg tabs
Dose	25-75 mg/kg	0.25-0.5 mg/kg	0.25-1 mg/kg	1-2 mg/kg	1-2 mg/kg
Onset	30-60 min	30-60 min	15-30 min	15-20 min	30 min
Duration	60-90 min	60-180 min	30-60 min	60-100 min	60 min
Half-life	8 hours	25 hours	1-2 hours	5-6 hours	4 hours
Nausea	+++	+	-	+++	-
Reversible	no	yes	yes	yes	no
Side effects	resp. dep nausea	resp. dep.	resp. dep	resp. dep., nausea	dry mouth
Contraindications	Coumadin/lasix	glaucoma	glaucoma	MAO inhibitors	none

*Doses must be adjusted when used in combination with other drugs

is given in Table 1. Of the agents listed, chloral hydrate has been and continues to be a popular sedative.12-14 Developed in 1832 by Leibig, it is currently available in capsule, syrup, and suppository form. The sedative properties of chloral hydrate are attributed to the active metabolite trichloroethanol.15 An alcohol, it follows zero-order kinetics and as such, has no definitive half-life. Consequently, the duration of the sedative effect can be highly variable and unpredictable when compared to agents that follow first-order kinetics.

The duration of the sedative effect can be significantly longer than the working time. Patients who may have moved excessively at the end of a procedure may become quite somnolent when the stimulation of treatment has ceased. This is especially true for patients having received chloral hydrate and is a significant disadvantage in the outpatient setting, where seemingly alert patients are discharged into their parent's care.

Broad dosing regimens for chloral hydrate have been reported. While variability in patient response is possible, a dose of 25 to 50 mg/kg, not to exceed a total dose of 1 g provides adequate sedation. Doses of 75 to 100 mg/kg have also been suggested. These higher doses may render patients incapable of independently maintaining their airways and unable to respond appropriately to verbal command, as required by the California Dental Practice Act.16 An antihistamine may be given in conjunction with chloral hydrate to reduce the incidence of nausea and vomiting.17-19

As a group, benzodiazepines are the safest and most effective sedatives available. Their successful use in the pediatric population is well-documented in the medical and dental literature.20-25 Benzodiazepines act centrally at the gamma-amino-butyric-acid receptor in the limbic system to produce anxiolysis and profound amnesia. The two principal agents presently used to orally sedate children are diazepam and midazolam. Valium is typically crushed and mixed with juice for oral administration. It is administered in a dose of 0.5 mg/kg and has a duration of 30 to 45 minutes. A disadvantage of diazepam is an extended half-life secondary to the active metabolite oxazepam, which can render the patient sluggish for up to 48 hours.

Midazolam is twice as potent as diazepam and water-soluble, making it easy to mix with juice for oral administration. It is also available premixed with cherry syrup. Studies suggest that it is the ideal oral sedative.26-34 Normal dosing is 0.25 to 0.5 mg/kg with a duration of 30 to 45 minutes. Exceptionally anxious children may require dosing up to 1 mg/kg not to exceed a 20 mg dose. Unlike diazepam, midazolam has little if any hangover effect, allowing for a full recovery prior to discharge.

Another benzodiazepine, triazolam as been used to sedate children.23,24 To date, it has not been approved by the Food and Drug Administration for use as a sedative.

In cases of overdosage, benzodiazepines are easily reversed by flumazenil 0.2 mg IV.35,36 Some advocate injecting flumazenil sublingually in cases of emergency. A search of the literature revealed no studies to support this practice.

The sedative and analgesic properties of opioids make them desirable agents for oral sedation. When bound to specific opioid receptors in the spinal cord and central nervous system, opioids attenuate pain and produce sedation and dose-dependent respiratory depression. Meperidine hydrochloride is frequently used as an oral sedative. Administered at

a dose of 1 to 2 mg/kg, it produces analgesia, sedation, and euphoria. Because of the frequently encountered side effects of nausea and vomiting and respiratory depression, meperidine is seldom administered alone. The "DPT cocktail," a mixture of Demerol (meperidine), Phenergan (promethezine) and Thorazine (chlorpromazine), has been used extensively by emergency physicians in hospitals and, to a lesser extent, by pediatric dentists in the dental office.37,38 Chlorpromazine, a neuroleptic, increases the depth of sedation without increasing the incidence of respiratory depression. It is also a potent antiemetic. Promethezine also has sedative properties, decreases the incidence of nausea, and dries the mouth. While generally safe, the DPT cocktail has been associated with seizures and may render a child unresponsive.39

As a group, antihistamines are very safe agents to use on children.40 Blockade of serotonin receptors by antihistamines in the central nervous system produces a sedative-like effect. Although they lack anxiolytic, amnestic, and analgesic properties, antihistamines cause drowsiness and have antiemetic and antisialogogue effects. Commonly used antihistamines include promethezine and hydroxyzine administered in a dose of 0.5 to 1 mg/kg.

The patient evaluation mentioned earlier can aid the dentist in formulating a pharmacologic treatment plan. This plan might make use of one or a combination of drugs. Studies show that the failure rate (being unable to initiate or complete treatment) of oral sedation can be as high as 40 percent and that the regimen used successfully for one appointment may not be effective for the next.41 Because repeat doses of oral sedatives are contraindicated at the same appointment, it is important to have an alternate treatment plan available.

Technique

When feasible, it is advantageous to sedate children in the early morning because a more reliable NPO (nothing by mouth) status can be achieved. Table 2 lists current American Society of Anesthesiologists preoperative fasting guidelines. Pediatric patients should be scheduled to arrive at the office one hour prior to the anticipated treatment time. The treating doctor should quickly reassess the patient by evaluating the airway, listening to the heart and lungs, recording baseline vital signs, and confirming NPO status. A written consent explaining the anticipated dental treatment, the reason for utilizing sedation, the use of restraints, and the risks, complications, treatment alternatives, and expected outcomes should be signed by a parent or legal guardian prior to treatment.

Table 2

American Society of Anesthesiologists Recommendations for Preoperative Fasting

Age	Solids/Nonclear liquids	Clear liquids
>36 months	6-8 hours	2-3 hours
6-36 months	6 hours	2-3 hours
<6 months	4-6 hours	2 hours

*Pain and anxiety can greatly increase gastric emptying time

The oral sedative should then be administered and the patient observed by trained staff. As the patient becomes drowsy, he or she should be casually walked or carried to the treatment area. Oxygen should be administered via nasal mask or nasal canula. If nitrous oxide-oxygen is to be used, it should be titrated to effect via a scavenged nasal hood system (See Malamed in this issue). Adjuncts to pharmacologic anxiolysis such as stuffed animals, ceiling-mounted televisions, and headphones are effective and can help promote cooperative behavior.

As the child settles into his or her environment, a blood pressure cuff, precordial stethoscope, and pulse oximetry probe can be placed and vital signs recorded. Local anesthesia should then be administered slowly with a 30-gauge needle. If 2 percent lidocaine with epinephrine 1:100,000 is used, care should be taken not to exceed a total dose of 4 mg/kg of body weight.42,43 Prior to the dentist's beginning treatment, he or she should protect the airway with a gauze throat pack or rubber dam; and auxiliary staff should be vigilant in suctioning.

At the conclusion of the procedure, the clinician must be sure that the maximum effect of the sedative has passed prior to the cessation of monitoring. If extractions were carried out, gauze hemostasis should be confirmed. Prior to discharge, postoperative vital signs should be close to baseline; and the patient must be able to maintain his or her oxygen saturation on room air. The child should be alert, oriented, and able to ambulate with minimal assistance. Finally, detailed written and verbal postoperative instructions should be given to the parents.

Safety

Anesthetic emergencies occurring in children almost always involve airway and/or respiratory compromise. Because of their disproportionately large tongues, proliferation of lymphoid tissue, and large tonsils, children breathe most readily through their noses. During dental treatment, the most common causes of airway obstruction in a child are occlusion of the posterior oropharynx with the tongue and obstruction of the nares with the nitrous hood.7 It is therefore critical to observe patients for adequate air exchange at all times.

To avoid positional asphyxiation, as much as practical, the child's head position

should be maintained in the sniffing position with the head extended. If obstruction is suspected, the tongue should be pulled forward in the mouth, the posterior oropharynx suctioned, and the nasal hood repositioned.

Apnea in the absence of airway obstruction is a complication of oral sedation rarely seen with normal dosing. When it does occur, it is easily managed with stimulation, positive pressure ventilation, and/or reversal of the sedative agent.

Summary

Thousands of children are sedated safely by dentists daily in the United States. While rare, morbidity and mortality in pediatric patients receiving oral conscious sedation does occur. Retrospective analysis has demonstrated that most mishaps can generally be attributed to four things:44,45

* Inadequate preoperative evaluation;
* Lack of knowledge concerning the pharmacology of drugs employed;
* Inadequate monitoring during the procedure; and
* Lack of training in the management of emergencies.

Competency in the administration of oral sedation is necessary for dentists who choose to treat children on a regular basis. In California, advanced training programs in pediatric dentistry are offered at Loma Linda University, the University of California at Los Angeles, the University of California at San Francisco, and the University of Southern California. Continuing education programs designed to fulfill the educational requirements mandated by the state legislature for an oral conscious sedation permit are taught by the faculties of UCLA, UCSF, Loma Linda, and CME Associates.

In the coming years, California must face and solve the growing issue of access to care for the young children of the state. With dental caries being the single most common childhood disease, the challenge will be daunting. While water fluoridation and other preventive modalities may make significant inroads into solving this problem, for the foreseeable future, the major responsibility for restoring these decayed teeth will fall on the dedicated dentists of California. If the dental needs of the pediatric population are to be addressed safely and effectively, it is vital that dentists who treat children receive appropriate training in oral conscious sedation on a regular basis.

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Current Concepts in Acute Pain Management

Mai-Phuong Huynh, DDS, and John A. Yagiela, DDS, PhD

ABSTRACT Analgesics most commonly prescribed in dentistry for acute pain relief include the nonsteroidal anti-inflammatory drugs, acetaminophen, and various opioid-containing analgesic combinations. The NSAIDs and presumably acetaminophen act by inhibiting cyclooxygenase enzymes responsible for the formation of prostaglandins that promote pain and inflammation. Opioids such as codeine, hydrocodone, and oxycodone stimulate endogenous opioid receptors to bring about analgesic and other effects. Numerous clinical studies have confirmed that moderate to severe pain of dental origin is best managed through the use of ibuprofen or another NSAID whose maximum analgesic effect is at least equal to that of standard doses of acetaminophen-opioid combinations. If an NSAID cannot be prescribed because of patient intolerance, analgesic preparations that combine effective doses of an orally active opioid with 600 to 1,000 mg of acetaminophen are preferred in the healthy adult. On occasion, prescribing both an NSAID and an acetaminophen-opioid combination may be helpful in patients not responding to a single product. In all cases, however, the primary analgesic should be taken on a fixed schedule, not on a "prn" (or as needed) basis, which only guarantees the patient will experience pain.

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The prescription illustrated in Figure 1 has most likely been written more frequently in dentistry for the management of moderate to severe pain (as may accompany the surgical extraction of third molars) than any other during the past three decades and remains highly popular today. Nevertheless, modern guidelines for acute pain management identify three significant problems involving this prescription with respect to the typical dental patient. In addressing these problems, this paper reviews the currently available oral analgesics used in dentistry and summarizes how these drugs may be prescribed to best advantage for acute pain relief.

History

The first "prescription" for pain management can be traced back 2,400 years, when Hippocrates suggested using juices of the poplar and willow bark to alleviate the pain of childbirth. Millennia later, it was found that these juices contained the compound salicin. In 1853, Charles Frederich Gerhardt synthesized aspirin by treating sodium salicylate (a simple derivative of salicin) with acetyl chloride. Aspirin was introduced clinically in 1899 by Heinrich

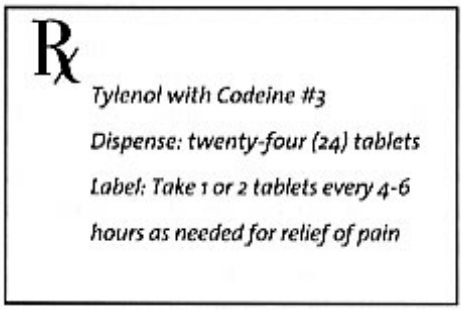

Figure 1. A highly popular prescription for management of moderate to severe dental pain. It has three significant problems.

Dresser of the Bayer Company in Germany and remains one of the most common remedies for acute pain.

Opioid use dates back even further, to the early civilizations of Persia, Egypt, and Mesopotamia. A Sumerian text from 4,000 B.C. was the first to refer to opium derived from the poppy plant. In 1805, Wilhelm Sertürner isolated morphine from opium, giving clinicians for the first time a chemically pure, highly effective analgesic.

Continued advances in the development of aspirin-like and morphine-like drugs have made available a broad spectrum of agents to manage acute pain. In addition, there has been a new understanding of how these analgesics should be used for optimal pain relief in the outpatient setting.

Pain and Analgesia

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience arising from actual or potential tissue damage or described in terms of such damage."1 Pain can be separated into two broad categories: acute and chronic. Acute pain lasts from seconds to days. It generally has a known cause and subsides with removal of the stimulus and healing. Acute pain may be associated with heightened arousal, leading to tachycardia, tachypnea, and anxiety. In contrast, chronic pain typically lasts from months to years. The body has become adapted to this level of pain, and often there is no increased sympathetic response. Chronic pain is associated with depression and decreased function.

Role of Cyclooxygenase and Prostaglandins

Tissue damage stimulates at the site of injury the release of inflammatory mediators such as prostaglandins, kinins, leukotrienes, substance P, and histamine. These mediators help initiate and subsequently magnify nociceptive impulses that are transmitted to the central nervous system for the perception of pain. Of these mediators, prostaglandins are especially important in sensitizing peripheral neurons to the local stimulus. Prostaglandins are also synthesized in the spinal cord and possibly higher brain centers in response to nociceptive impulses and enhance pain sensitivity by recruiting additional secondary neurons that respond to the primary stimulus.

Aspirin and related nonsteroidal anti-inflammatory drugs work at the site of tissue damage, the spinal cord, and/or higher brain centers to prevent prostaglandin formation by inhibiting cyclooxygenase, or COX, activity. With the partial exception of acetaminophen, which has minimal anti-inflammatory effects in most settings, these drugs exert a combination of analgesic, antipyretic, and anti-inflammatory effects.

Tissue COX exists in two well-known subtypes: COX-1 and COX-2. COX-1 is a constitutive form that supports hemostasis (where synthesis of the prostaglandin analogue thromboxane A2 increases platelet degranulation and adhesion), stomach mucosal integrity (where synthesis of prostaglandins protects against acid damage), and kidney function (where prostaglandins help regulate normal renal blood flow). COX-2 is a largely inducible form whose synthesis is activated in damaged or stimulated tissues and leads to the formation of pro-inflammatory prostaglandins. COX-2 plays a major role in inflammation, pain, and fever. It is also constitutively active in regulating renal blood flow.2

There is increasing evidence that one or more additional subtypes of COX may exist. A new COX-3 has been described that is produced by the same gene that encodes COX-1. This COX-3 is found in the brain and is inhibited by clinically achievable concentrations of acetaminophen.3 An additional form of COX that is induced with high concentrations of NSAIDs and is selectively inhibited by acetaminophen may be derived from the COX-2 gene.4

Role of Endogenous Opioids

Nociceptive pathways in the central nervous system are subject to modulation by neurons that release inhibitory transmitters at synaptic sites important for the perception of pain. Brain sites involved in affective responses to pain are also inhibited. Endogenous opioid peptides that stimulate specific opioid receptors play a pivotal role in blunting pain. Evidence has accumulated in recent years that peripheral nerves in inflamed tissues also contain opioid receptors whose activation can produce analgesia.5

Morpine and related opioid analgesics exert most of their pharmacologic effects by stimulating the μ opioid receptor. In addition to relieving pain, μ-receptor stimulation can cause a tranquil euphoria, nausea and vomiting, and constipation. In overdose, respiratory depression is the primary concern; chronic use can lead to physical and psychological dependence.

Pentazocine, nalbuphine, and related opioid agonist-antagonists promote analgesia by stimulating the ? opioid receptor. Analgesic and respiratory depressant effects are similar to those elicited by morphine in normal clinical doses; however, a ceiling effect limits these responses in overdose. Sedation is a common side effect of the agonist-antagonists, but stimulation of ? receptors is more likely to produce dysphoria than euphoria, and psychotomimetic reactions are common with large doses. Physical dependence is possible but less problematic with ? agonists. Precipitation of an acute opioid withdrawal reaction may occur when pentazocine or nalbuphine is administered to an opioid-dependent individual.

Nonsteroidal Anti-Inflammatory Drugs (Including Acetaminophen)

As the prototypical NSAID, aspirin

remains a standard against which other orally active analgesics are compared. It is relatively selective for COX-1 and is therefore prone to causing gastric bleeding and ulceration, especially with high doses and chronic use. Aspirin is unique in that it acetylates the COX enzyme. This property, plus the drug's relative COX-1 selectivity and the inability of platelets to synthesize new COX, provides the basis for the use of low-dose aspirin to prevent thromboembolic heart attack and stroke in susceptible patients. Typical doses of 325 to 650 mg encompass most of aspirin's analgesic dose-response curve in the average adult.

Acetaminophen's analgesic and antipyretic properties are comparable to those of aspirin. However, it does not elicit gastrointestinal irritation or prolong bleeding, which are hallmarks of aspirin use. Acute toxicity is minimal unless an overdose occurs, which may lead to hepatotoxicity. Analgesia by acetaminophen in the average adult becomes readily measurable at a dose of 300 mg and plateaus at 1,000 mg.6

Ibuprofen was the first NSAID to demonstrate analgesic superiority to aspirin. A 400 mg dose of ibuprofen has been shown to have a greater peak analgesic effect and a longer duration than 600 to 1,000 mg of aspirin or acetaminophen, or 60 mg of codeine, and at least comparable efficacy to traditional opioid analgesic combinations (Figures 2a and b).7,8 One meta-analysis has suggested there is a dose-dependent increase in analgesia with ibuprofen up to 800 mg.9 However, the published source from which the statistically analyzed data were obtained for this assertion actually revealed little analgesic improvement with doses beyond 400 mg.10 It is likely that the principal effect of prescribing doses of ibuprofen larger than 400 mg for pain relief is that the duration of maximum analgesia is prolonged.

Naproxen, an NSAID structurally related to ibuprofen, has a half-life of about 13 hours, which allows for less frequent dosing compared to ibuprofen (half-life of two hours).6 A 220 mg dose of naproxen sodium is equivalent to 200 mg of ibuprofen in analgesic onset and peak effect but has a longer duration of action.11 A similar comparison holds for 440 mg of naproxen sodium compared with 400 mg of ibuprofen.12 Diclofenac, ketoprofen, flurbiprofen, meclofenamate, and diflunisal are additional NSAIDs with analgesic activity in the dental setting similar to that of ibuprofen or naproxen. Fenoprofen is also approved for the management of acute pain, but its slow absorption retards the onset of analgesia. Ketorolac, an NSAID commonly used for parenteral administration, is restricted in its oral dosage form to patients who have already received the drug by injection. Lastly, etodolac is a well-tolerated NSAID; however, it has not been proved superior to aspirin for relieving pain of dental origin.

The introduction of selective COX-2 inhibitors has allowed specific targeting of inflammatory prostaglandin production while minimizing adverse side effects such as gastrointestinal ulceration and bleeding problems.13 Clinically available COX-2 inhibitors include rofecoxib, celecoxib, and valdecoxib. As yet, valdecoxib has not been approved for the treatment of acute pain (although recent studies indicate it has strong potential for such use).14 Rofecoxib enjoys two advantages over celecoxib and valdecoxib in that its duration of action is sufficiently long to permit single daily dosing, and it is not contraindicated in patients with a history of sulfonamide allergy. In a dose of 50 mg, rofecoxib is comparable to 400 mg of ibuprofen in onset and peak pain relief.15,16

Opioids and Related Agents

In contrast to NSAIDs, opioids do not have an obvious ceiling effect for analgesia. Thus, increasing the dose increases the pain relief. Unfortunately, opioids cause undesirable side effects that limit their dosing, especially in the outpatient setting. These adverse effects include nausea and vomiting, constipation, sedation, and, in large doses, respiratory depression. Some opioids (e.g., morphine and codeine) also cause anti-inflammatory effects and even immunosuppression; the clinical significance of these findings for dental surgery remains unknown.17,18

Morphine, the prototypical opioid analgesic, is rarely prescribed orally for the management of acute pain because most of the drug is metabolized in the liver before it can reach the systemic circulation. Meperidine and many other opioids have similar problems with the oral route. The three most commonly used opioids for oral administration are codeine, hydrocodone, and oxycodone. These agents have a high oral:parenteral efficacy ratio, in part because a fraction of each drug is converted by the hepatic enzyme cytochrome P450 2D6 (CYP2D6) to a much more active metabolite (codeine?morphine, hydrocodone?hydromorphone, oxycodone?oxymorphone) and released into the bloodstream.19 About 5 percent to 10 percent of Caucasians and 1 percent to 3 percent of African-Americans and Asians have a polymorphic gene for CYP2D6 that cannot support these conversion reactions. In the case of codeine, no analgesia is obtained, nor are such dose-dependent side effects as constipation or respiratory depression. On rare occasions, oral methadone may be prescribed for analgesia. It is chemically unrelated to the aforementioned opioids and requires no metabolic conversion for its opioid action. Propoxyphene is structurally related to methadone but is a much weaker analgesic. At best, 100 mg of propxyphene napsylate is equivalent to 60 mg of codeine.

Tramadol is an analgesic with two complementary mechanisms of action. It is a weak μ-receptor agonist, imbuing the drug with opioid-like activity. In addition, tramadol inhibits the reuptake of norepinephrine and 5-hydroxytryptamine, an antidepressant-like action. Because of its weak opioid activity, tramadol exhibits less respiratory depression, drug dependence, and other side effects commonly associated with opioid use. However, studies have shown that relief of acute oral surgery pain with 50 mg of tramadol is similar to that of 60 mg of codeine but less than that of a full

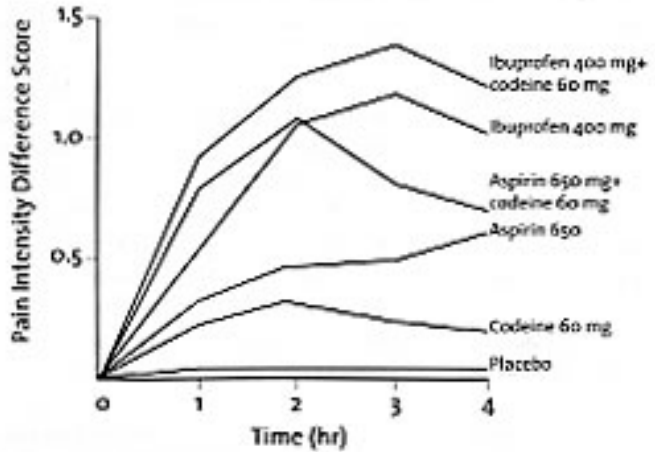

Figure 2A. Pain relief after removal of impacted third molars. Data from Cooper SA, Engel J, et al, Analgesic efficacy of an ibuprofen-codeine combination. Pharmacotherapy 2:162-7, 1982.

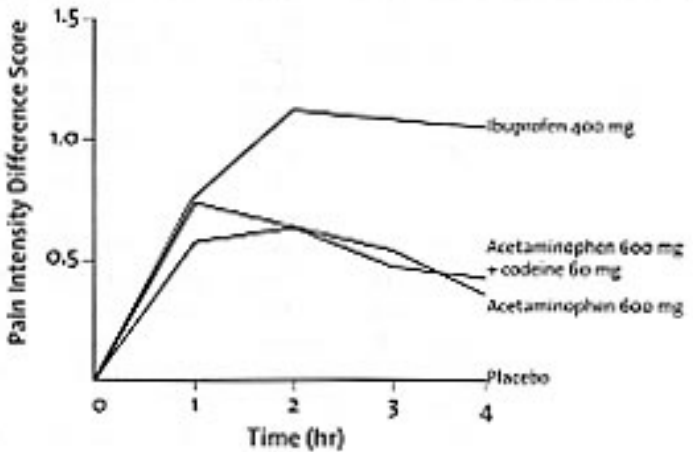

Figure 2B. Pain rPain relief after removal of impacted third molars. Data from Forbes JA, Kehm CJ, et al, Evaluation of ketorolac, ibuprofen, acetaminophen, and an acetaminophen-codeine combination in postoperative oral surgery pain. Pharmacotherapy 10(suppl 6, part2):94S-105S, 1990. (Slightly negative pain intensity difference scores for placebo after two hours are not shown in the graph.)

therapeutic dose of codeine in combination with aspirin or acetaminophen. Tramadol with acetaminophen may be a suitable choice for patients who do not tolerate NSAIDs or opioid analgesics well.20

The agonist-antagonist pentazocine is another alternative to the codeine-like opioids. A dose of 50 mg pentazocine is approximately equianalgesic as 60 mg of codeine. Other agonist-antagonists are not available in oral dosage forms.

Combination Analgesics

No analgesic agent for oral administration is currently available that can relieve all intensities of acute pain. The NSAIDs, acetaminophen, and the opioid agonist-antagonists exhibit a ceiling effect for analgesia in that no greater pain relief can be obtained by increasing the dose beyond a certain limit. In the case of the morphine-like opioids, dose-dependent toxicity restricts the analgesic effect that can usually be obtained. In fact, standard doses of opioids (e.g., codeine 30 to 60 mg, hydrocodone 5 to 10 mg, oxycodone 5 to 10 mg) produce pain relief for dentistry that only approaches the analgesia of two aspirin tablets (650 mg, see Figure 2).

A strategy commonly used to enhance the analgesic benefit of oral medication is to combine two (or more) drugs with different mechanisms of action. The combination of acetaminophen or an NSAID with an opioid allows for increased analgesia because the drugs act through dissimilar mechanisms.21 Because they also have dissimilar side effects, summation of the intensity of these effects does not occur.

Combining an NSAID with another NSAID, or an opioid with another opioid, provides no such benefit. In the case of NSAIDs, the maximum pain relief is already achieved by using a fully effective dose of a single agent. The combination can only produce increased adverse effects. With opioids, the increased analgesia, which could also have been obtained by using a larger dose of a single drug, is accompanied by heightened adverse effects that make such combinations intolerable.

Some practitioners prescribe acetaminophen with an NSAID for postoperative pain control. Clearly, if acetaminophen is insufficient by itself, then an NSAID with a stronger analgesic effect may produce more pain relief than can acetaminophen alone. It is unclear, however, if adding acetaminophen to any NSAID already being taken at a ceiling analgesic dose provides any benefit.22-24 Resolution of this question may ultimately depend on whether acetaminophen and the NSAIDs act on the same or different prostaglandin pathways involved in nociception.

Caffeine is an analgesic adjuvant that exerts no analgesic action by itself in humans but can enhance the potency of such drugs as acetaminophen, aspirin, and ibuprofen. In the case of ibuprofen given after third-molar surgery, the analgesic potency of 100 to 200 mg doses is increased more than twofold by 100 mg of caffeine.25 There are few data, however, to suggest that caffeine can improve the analgesic effect of "ceiling" doses of NSAIDs or acetaminophen.26

Adverse effects of combination analgesics include those for each drug in the combination. In addition, the chronic use of aspirin, phenacetin (an acetaminophen analogue), and caffeine, as once commonly formulated in the "APC" tablet, has long been associated with end-stage kidney disease. The simultaneous use for several days of an acetaminophen-opioid preparation and an NSAID has not been linked to increased renal toxicity.

Precautions and Drug Interactions

Adverse responses to analgesics are more likely to occur in patients who have certain medical conditions or are taking specific drugs. In the case of NSAIDs, all

such drugs (including the COX-2-specific agents) should be avoided in any patient who has exhibited an allergic-like (anaphylactoid) reaction, such as urticaria, angioneurotic edema, bronchial asthma, and acute hypotension, to any NSAID.6 It is believed that inhibition of COX in these patients may result in overproduction of leukotriene mediators of anaphylaxis. NSAID intolerance is also particularly common in patients with rhinitis, nasal polyps, and asthma for which systemic corticosteroids must be used to control bronchospasm. Patients with bleeding disorders or platelet deficiency, or with a history of gastrointestinal inflammatory or ulcerative disease, should not receive NSAIDs with COX-1 activity. Although it is probably safe to prescribe a short course of a COX-2-selective inhibitor in a patient with an ulcer history, such drugs are best avoided in individuals with an existing or recent ulcer because COX-2 is expressed locally during the healing phase. Other conditions in which NSAIDs are not recommended include pregnancy in the second and third trimester (possible premature closure of the ductus arteriosus, excessive bleeding, or depressed uterine contractions during labor and delivery) and congestive heart failure or significant renal impairment (possible fluid retention). The use of aspirin in children may trigger Reye's syndrome in the presence of a viral infection and is best avoided in pediatric and adolescent patients.27

Opioid analgesics are problematic in patients with impaired respiration, as may accompany advanced emphysema or poorly controlled myasthenia gravis. Severe inflammatory bowel disease is also a contraindication to opioid use. Patients with a history of opioid drug abuse present a special set of issues. These patients tend to have a relatively low pain threshold, which may be coupled with a relatively high tolerance to opioids. Consultation with the patient's physician is advised to help balance the need for effective analgesic medication against the concern that such medication may trigger addiction relapse. (It should be noted that there is an extremely low incidence of drug addiction when short courses of opioids are given for analgesia to patients without a history of drug abuse.28)

Geriatric patients often exhibit diminished clearance of analgesic medications, increased plasma concentrations of free drug, and increased pharmacologic effects.

Table 1

Drug Interactions Involving NSAIDs and Acetaminophen

Drug	Effect	Recommendation
Alcohol	Acetaminophen toxicity more likely in chronic alcoholics	Use cautiously. Maintain normal alcohol intake.
Alcohol, corticosteroids, potassium supplements	Increased risk of ulceration with NSAIDs	Avoid combination. Substitute with acetaminophen.
Anticoagulants, thrombolytics, colchicines, broad-spectrum β-lactam antibiotics (e.g., cefoperazone), valproic acid	Increased risk of bleeding with NSAIDs	Avoid combination. Substitute with acetaminophen.
Antihypertensives: ß blockers, ACE inhibitors, diuretics	Decreased antihypertensive effect with NSAIDs	Use NSAIDs cautiously. Monitor blood pressure.
Digoxin	Increased digoxin concentrations	Avoid combination if patient is elderly or has renal disease.
Antidiabetic drugs (insulin or oral hypoglycemics)	Enhanced hypoglycemic effect with NSAIDs	Monitor blood glucose concentrations. Substitute with acetampinophen.
Lithium, methotrexate (high dose)	Increased blood concentrations of these agents when administered with NSAIDs	Avoid combination. Substitute with aceteminophen.
Cyclosporine	Possible nephrotoxicity	Avoid combination if possible.
Probenecid, sulfinpyrazone	Renal and/or biliary excretion of NSAIDs inhibited. Uricosuric effects of probenicid and sulfinpyrazone blocked by aspirin	Avoid combination. Substitute with acetaminophen.
Carbonic anhydrase inhibitors (e.g., acetazolamide)	Increased CNS toxicity with aspirin	Use cautiously.
Antacids, griseofulvin	Reduced aspirin concentrations	Use alternative analgesic.
Hepatic enzyme inducers: barbiturates, isoniazid, phenytoin, rifampin	Possibly altered acetaminophen metabolism and increased hepatotoxicity	Use cautiously.

Table 2

Drug Interactions Involving Opioids

Drug	Effect	Recommendation
CNS depressants: alcohol, antihistamines, sedative-hypnotics, local anesthetics, antidepressants, antipsychotics, central acting antihypertensives	Increased CNS, respiratory depression	Use cautiously.
Anticholinergics, antidiarrheals	Constipation	Use opioids for short periods only.
Agonist-antagonist drugs (nalbuphine, pentazocine)	Antagonism of opioid analgesic effect: loss of analgesia with hypertension and tachycardia, may cause withdrawal syndrome in opioid-dependent patient	Avoid combined use and use of opioid agonist-antagonist in opioid-dependent patients.
MAO inhibitors (phenelzine, selegiline), sibutramine	Hemodynamic instability, convulsions, coma with meperidine	Avoid meperidine. Use other opioids cautiously.
Amphetamines	Hypotension, respiratory collapse with meperidine	Avoid meperidine.
Tobacco	Possible decreased effect of propoxyphene	Avoid propoxyphene. Recommend smoking cessation.
Antihypertensives, diuretics, vasodilators	Potentiation of hypotensive effects	Avoid combination if postural hypotension or dizziness occurs.
CYP2D6 inhibitors: ritonavir, quinidine, propafenone, paroxetine, fluoxetine, sertraline	Decreased effect of codeine, hydrocodone, oxycodone	Monitor analgesic effect. Use alternative drugs if pain relief inadequate.

There is also a heightened risk for drug interactions since many elderly patients are already taking multiple medications, including analgesic/anti-inflammatory drugs.

Drug interactions of concern in dentistry are listed for the NSAIDs and acetaminophen and for the opioid-like drugs in Tables 1 and 2, respectively.

Analgesic Selection

The selection of an analgesic for the management of acute dental pain is ideally based on the pain's actual or expected intensity, the patient's medical history, the drug's pharmacologic profile, and the ease and cost of obtaining the medication. Table 3 lists the authors' recommendations for the typical healthy adult.

Pain of mild to moderate intensity, as may follow extensive restorative dentistry or simple periodontal surgery, is best managed with analgesics listed in Table 3 that are usually found in the home. These drugs, including acetaminophen and ibuprofen, are effective for this level of discomfort; they are also inexpensive and sold over the counter. Patients who have purchased specific analgesics are generally experienced in their use and regard them as effective and well-tolerated.

The NSAIDs listed in Table 3 are the drugs of first choice for controlling moderate to severe pain in dentistry. This degree of pain is characteristically caused by acute pulpitis or the surgical removal of impacted third molars. In doses that produce ceiling analgesia, these NSAIDs are better-tolerated and at least as effective as the more traditional acetaminophen-opioid combinations. Ibuprofen is unusual in that the over-the-counter unit dose of 200 mg can easily be used to duplicate prescription doses (400, 600, 800 mg) of ibuprofen that produce ceiling analgesic effects. While the COX-2 inhibitor rofecoxib exhibits an extended duration of action compared to other NSAIDs, the authors found the cost of a prescription for 50 mg of rofecoxib, every day for three days, to be approximately 15 times that of 800 mg ibuprofen (four 200 mg tablets), three times a day for three days ($14.69 versus $1.09).

Acetaminophen-opioid combinations are the drugs of choice for moderate to severe pain when NSAIDs are contraindicated. The formulations listed in Table 3 ensure that the acetaminophen, which provides most of the pain relief, is taken in a dose of at least 600 mg and that the opioid is used in a dose (codeine 60 mg, hydrocodone 7.5 to 10 mg, oxycodone 7.5 to 10 mg) that significantly and consistently increases pain relief in the oral surgery pain model without usually producing intolerable side effects. Hydrocodone as formulated in Lortab 5/500 or Vicodin (two tablets each, Table 3) is arguably the preferred opioid, since codeine may be less effective in certain patients and oxycodone combined with acetaminophen is a Schedule II drug requiring a triplicate prescription form.

Opioids -- generally combined with ac-

CDA JOURNAL, VOL 31, Nº5

Table 3

Oral Analgesic Recommendations

Drug	Proprietary name	Recommended dose (adult)	Schedule
Mild to moderate pain			
Acetaminophen 325 mg	Tylenol, others	2 tab. q. 4 h.	OTC
Aspirin 325 mg	A.S.A., others	2 tab. q. 4h.	OTC
Ibuprofen 200 mg	Advil, Motrin IB, Rufen	1 tab. q. 4 hr.	OTC
Naproxen sodium 220 mg	Aleve	1 tab. q. 6-8 hr.	OTC
Moderate to severe pain			
Diclofenac potassium 50 mg	Cataflam	1 tab. t.i.d.	Rx
Diflunisal 500 mg	Dolobid	2 tab. to start, then 1 tab. q. 8-12 h.	Rx
Flurbiprofen sodium 50 mg	Ansaid	1 tab. q. 6 hr.	Rx
Ibuprofen 200 mg	Advil, Motrin IB, Nuprin	2 tabs. q. 4 h. or 3 tabs q. 6 h.	OTC
Ibuprofen 400 mg	Motrin, Rufen	1 tab. q. 4 h	Rx
Ibuprofen 600 mg	Motrin, Rufen	1 tab. q. 6 h	Rx
Ketoprofen 50 mg	Orudis	1 cap. q. 6-8 h.	Rx
Meclofenamate sodium 100 mg	Meclomen	1 tab. q. 6 h.	Rx
Naproxen sodium 275 mg	Anaprox	2 tabs. to start, then 1 tab. q. 6-8 h.	Rx
Rofecoxib 50 mg	Vioxx	1 tab. q. d., not to exceed 5 d.	Rx
Moderate to severe pain when opioid desired or NSAID contraindicated*			
Acetaminophen 300 mg and codeine 30 mg	Tylenol with Codeine #3	2 tabs. q. 4 h.	CIII
Acetaminophen 325 mg and codeine 30 mg	Phenaphen with Codeine #3	2 caps. q. 4 h.	CIII
Acetaminophen 500 mg and hydrocodone 5 mg	Hyco-pap, Lortab 5/500, Vicodin	2 tab. q. 4-6 h., max. 8 tabs/24 h.	CIII
Acetaminophen 650 mg and hydrocodone 7.5 mg	Lorcet Plus	1 tab. q. 4-6 h.	CIII
Acetaminophen 650 mg and hydrocodone 10 mg	Lorcet 10/650	1 tab. q. 4-6 h.	CIII
Acetaminophen 660 mg and hydrocondone 10 mg	Vicodin HP	1 tab. q. 4-6 h.	CIII
Acetaminophen 750 mg and hydrocodone 7.5 mg	Vicodin ES	1 tab. q. 4-6 h., max 5 tabs./24 h.	CIII
Acetaminophen 750 mg and hydrocodone 10 mg	Maxidone	1 tab. q. 4-6 h., max 5 tabs./24 h.	CIII
Acetaminophen 325 mg and oxycodone 5 mg	Percocet 5/325	2 tabs. q. 4-6 h.	CII
Acetaminophen 500 mg and oxycodone 7.5 mg	Percocet 7.5/500	1-2 tabs. q. 4-6 h., max 8 tabs/d.	CII
Acetaminophen 650 mg and oxycodone 10 mg	Percocet 10/650	1 tab. q. 4-6 h.	CII
Acetaminophen 650 mg and pentazocine 25 mg	Talacen	1 tab. q. 4 h.	CIV
Acetaminophen 650 mg and propoxyphene 100 mg	Darvocet-N100	1 tab. q. 4 h.	CIV
Acetaminophen 325 mg and tramadol 37.5 mg	Ultracet	2 tabs. q. 4-6 h., max 8 tabs./24 h.	Rx
Severe pain†			
Ibuprofen 200 mg and hydrocodone 7.5 mg; ibuprofen 200 mg	Vicoprofen; Advil, Motrin IB, Nuprin	2 tabs. each q. 6 h.	CII; OTC
Diflunisal 250 mg; methadone 10 mg	Dolobid; Dolophine	1 tab. each q. 6 h. (use 4 tabs diflunisal for 1st dose)	Rx; CII

*May also be prescribed in addition to an NSAID for use if pain relief is inadequate with the NSAID alone.

†Pain unrelieved by combined use of an NSAID and an acetaminophen-opioid analgesic.

etaminophen for prescribing convenience -- may also be used as a supplement to an NSAID for additional pain relief if needed (for example, if the analgesic effectiveness of the NSAID is shorter in duration than the dosing interval). Here, a formulation such as Lortab 10/650 or Vicodin HP reduces the acetaminophen dose somewhat. Ibuprofen-hydrocodone, the only NSAID-opioid combination currently available, is not recommended for routine use because the standard dosing schedule of one tablet, containing 200 mg of ibuprofen and 7.5 mg of hydrocodone, does not provide a ceiling analgesic effect for the ibuprofen.

Several combinations (acetaminophen with either pentazocine, propoxyphene, or tramadol) are listed in Table 3 for patients who are truly allergic to morphine-like opioids. Acetaminophen-tramadol may be the preferred choice based on recent efficacy studies in the oral surgery model.29

On rare occasion, severe dental pain is not satisfactorily relieved with standard analgesic doses. Table 3 lists two regimens in which maximum tolerated doses of an NSAID and an opioid are combined that

may provide some additional analgesia beyond the drugs already mentioned. The first example combines 800 mg of ibuprofen with 15 mg of hydrocodone, a dose greater than generally recommended but nevertheless tolerable in the majority of nonambulatory patients. The second example involves diflunisal and methadone. This approach requires a triplicate prescription for the methadone but may be superior in patients with inherited or acquired CYP2D6 deficiency. Either of these combinations should be prescribed only for a healthy adult patient whose refractory pain is sufficient to require home rest.

Analgesic Use

When prescribing an analgesic for dentistry, the dentist should direct the patient to take the initial dose as soon as feasible and then follow a fixed dosing schedule for at least the expected duration of the most intense pain (i.e., for two days after surgical tooth extractions). This practice ensures the maintenance of effective drug concentrations at the sites of action. Prescribing drugs on a "prn" (pro re nata, Latin for "as needed") basis only helps ensure that pain will be felt. Particularly with NSAIDs, analgesics should be ingested before the pain becomes significant. It takes about two hours after tissue injury for the induction of COX-2 and formation of pro-inflammatory prostaglandins.30,31 Therefore, administration of an NSAID within two hours of tissue injury will be effective in preventing postoperative discomfort.

Because most NSAIDs inhibit both COX-1 and COX-2 nonselectively, their administration before hemostasis is achieved may promote postoperative bleeding. A significant advantage of rofecoxib and other selective COX-2 inhibitors is that they may be given preoperatively for "pre-emptive analgesia" without the worry of decreased platelet function. For surgical cases of extended duration, a single dose of rofecoxib may be beneficial in tiding the patient over until the prescribed analgesics can be taken postoperatively.

Age and body size can significantly influence the use of analgesics in dentistry. Pediatric patients rarely require analgesics beyond those obtainable without a prescription. Ibuprofen oral suspension (100 mg/5 mL) at a dose of 10 mg/kg every four hours (to a maximum of 40 mg/kg/day) probably is the most effective oral analgesic for children; if acetaminophen with codeine oral suspension or solution (125 mg acetaminophen and 12 mg codeine/5 mL) is prescribed, the dose for children 3 to 7 years old is 5 mL (1 teaspoon) every six hours and twice that for children 7 to 12. For elderly patients, regular doses of analgesics listed in Table 3 should be reduced by 50 percent. Dosage reduction is especially important for opioid-containing products because of the marked increase in opioid sensitivity that accompanies advanced age.

Conclusion

Returning to the prescription at the beginning of this paper, three errors that compromise effective pain relief are readily identifiable:

* Tylenol with codeine #3 contains 300 mg acetaminophen and 30 mg of codeine. It is not a preferred drug for a patient who can receive NSAIDs.

* Giving the patient the option of taking one tablet may well result in analgesia inferior to that produced by a standard dose of acetaminophen alone. The clinician should determine the amount of analgesic to be used, not the patient.

* The use of "prn" dosing helps ensure the patient will experience postoperative pain before attempting to treat it. Patient comfort is improved if analgesics are taken on a fixed schedule for the first few days. Thereafter, the patient may reduce intake in response to the waning noxious stimulus. Thus, a prescription for moderate to severe pain in dentistry that meets current knowledge might read as shown in Figure 3.

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Current Concepts in Pain Management

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ABSTRACT The majority of people afflicted with orofacial pain have acute pain that resolves quickly, but some are left with chronic and disabling pain. Therapy must be provided to deal with the nociception, behavior, and suffering. Appropriate behavioral evaluation may be required prior to developing a treatment plan. The treatment should then be carefully outlined and presented in a treatment-planning visit and may include physical, pharmacologic, and behavioral aspects.

AUTHORS

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Orofacial pain is the result of a complex interaction of nociception, pain, suffering, and behavior that afflicts millions. The majority of people afflicted with orofacial pain have acute pain that resolves quickly, but some are left with chronic and disabling pain. Too often, this chronic pain is left undiagnosed; and patients continue to suffer. Because dental schools teach little about chronic pain, a diagnosis is infrequently made; or patients with non-odontogenic pain -- e.g., migraine, cluster headache, trigeminal neuralgia, or myofascial pain -- are misdiagnosed. The term "atypical" is often suggested to categorize the pain and implies a psychiatric or behavioral problem.1 "Idiopathic," when referring to a medical problem, suggests there is something unknown and does not define the problem. The same applies to terms incorporating the word "atypical." It has been reported that patients described as atypical or idiopathic can be diagnosed if evaluated by someone with more experience.2 The International Association for the Study of Pain3 and the International Headache Society4 take the position that there are better terms for facial pain diagnoses than atypical or idiopathic.

The term "idiopathic facial pain" has included diagnoses such as atypical facial pain, atypical odontalgia, masticatory muscle disorders, and traumatic neuralgia. These categories serve to perpetuate the limited knowledge of orofacial pain where the etiology is unclear. The most comprehensive facial pain classification to date incorporates the International Headache Society criteria and expands it to include disorders that were not clearly defined.4 It is suggested that if the clinician does not know what is causing the pain, the term "idiopathic" or "pain of unknown origin" be used. The patient should then be referred for better diagnosis. The major categories of pain are intracranial, extracranial, musculoskeletal, neurovascular, neuropathic, and psychogenic. In many categories, the etiologic mechanism, peripheral and/or central, is

poorly understood. Pain disorders must be classified based on an understanding of the underlying mechanism, etiology, and clinical presentation. It is also necessary to recognize the differences in acute and chronic pain because treatment approaches are different.5

Acute pain may best be described as a useful pain, e.g., acute pulpitis, mucosal irritation secondary to a prosthesis, and active infection. It is the essential component that allows the sufferer and his or her practitioner to know something is wrong. It usually lasts a predictable time. If the duration extends beyond what is expected, further investigation is needed; or a chronic etiology may be expected. There is little difficulty in defining the specific pathology (e.g., denture irritation) of acute pain; and therapy is usually obvious (e.g., removal of irritating source). Chronic pain typically begins with an acute episode and progresses to a chronic condition if it is inappropriately managed, no treatment is sought, or treatment was not completed, e.g., a peripheral neuropathic pain may develop following a herpes infection that causes acute pain. The pain duration is usually defined as being longer than six months. The pain is not necessarily useful and is frequently associated with increased anxiety and depression, possibly because numerous doctors have been seen and numerous procedures tried without mitigation of the pain.

Management Approaches for Chronic Pain

A practitioner must carefully assess and diagnose the pain before developing a therapy. Treatment should be aimed at a specific diagnosis or pain mechanism. Using poorly defined diagnoses such as atypical trigeminal neuralgia or atypical facial pain should be avoided. If a practitioner cannot make an accurate diagnosis, he or she should consider referring the patient. The dentist must try to understand each component of the chronic condition so a comprehensive and structured therapy can be offered. Therapy must be provided to deal with the nociception, behavior, and suffering. Appropriate behavioral evaluation may be required prior to developing a treatment plan. The treatment should then be carefully outlined and presented in a treatment-planning visit. Treatment may include physical, pharmacologic, and behavioral aspects. Using a structured treatment agreement is helpful in explaining therapy to patients.

Treatment Planning

A treatment contract is a written agreement between a patient and health care provider.6 This is imperative in a setting such as a chronic pain center where patients receive simultaneous treatment from multiple specialists. In clinical practice, using this concept helps to prevent patients who are not responding from being overtreated. Patients who suffer from chronic disease often feel a loss of self-control. Too often, they have been given the false promise that the next surgery, medication, or alternative therapy will "cure" the problem, only to be disappointed. It is therefore recommended that a treatment contract be used to provide clear treatment explanations, set treatment goals, set treatment duration, define the patient's and health care providers' responsibilities, and provide specific contingencies to enhance compliance.

When providing a treatment explanation, it is suggested that all treatment possibilities be discussed. Where possible, this should be provided in stages, especially if one component of therapy is contingent upon the success of another. It is recommended that all possible therapies be outlined, allowing flexibility to change to alternative plans if the first choice is not working out. Once a treatment program is established, the treatment contract will help ensure compliance. Additionally, if the patient and clinician sign the treatment contract, the full course of treatment is agreed upon, preventing the patient from dictating changes.

Setting goals is important in a treatment contract. It is recommended that the treatment goals be stated as behaviors (process goals) to be learned, increased, decreased, or eliminated, rather than specific percentage reductions in pain (end goals). The goals need to be realistic and attainable.

Setting treatment duration is useful in allowing specific therapies to be tested prior to re-evaluating and moving forward with alternatives or proceeding with further workup. The treatment contract should specify therapy duration and frequency. In patients for whom noncompliance or tardiness is a possibility, consequences may include stopping therapy or referring the patient for inpatient care. By agreeing to a treatment contract that sets a time limit, the patient is made aware that there is a treatment constraint. Therefore, there is a consequence if he or she misses appointments or does not follow the instructions outlined.

Sharing responsibility for treatment outcome is especially important in chronic pain patients. It is not uncommon for the patients' expectation to be unrealistic. In chronic migraine, it is not possible to "cure" the pain. Migraine is a genetic disorder that at this time can only be managed. Therefore, a treatment contract will define what the patient needs to do to manage the pain as opposed to what the clinician can do for the patient. Pain management usually requires a multidisciplinary approach. Patients who are exposed to more than one clinician

are apt to receive conflicting information or may conveniently interpret information as conflicting to split the team. The treatment contract enables one person, "the pain manager," to coordinate the treatment team. All questions or problems should be discussed with the pain manager. The entire team, including the referring physician and pharmacy, should have a copy of the treatment contract to prevent miscommunication.

The treatment contract can be used to define contingencies. Behaviors that are required and prohibited should be specified. These may include issues related to missed appointments, late arrival, medication usage (decreasing dose schedule, time contingent administration vs. pain contingent administration), noncompliance, and home program. The consequences of undesirable behaviors should be clearly spelled out.

Therapies

Orofacial pain may be addressed with a variety of physical, pharmacologic, and behavioral strategies. Therapy principles and rationales will be discussed, rather than disease-specific therapies.

Physical

Exercise

Posture and body mechanics as they relate to working and relaxation should be considered in musculoskeletal pain and neurovascular disorders.7 Poor posture is thought to affect most people, but when there is nociceptive activity in a muscle, further nociceptive input, caused by poor posture, might trigger a greater pain response. It has been proposed that posture related to sitting, standing, and sleeping be discussed with the chronic head and neck pain sufferer. Patients are trained to sleep on their back or side, emphasizing a neutral spine position. Sitting with appropriate lumbar support and taking regular breaks from sitting hunched over a workstation are recommended. Good head position requires keeping the ear aligned over the shoulder and hip while sitting and standing. Appropriately modifying the workstation and ensuring eyewear is well-adjusted will prevent anterior head position (jutting the head forward) and eyestrain. Jaw position is also significantly affected by anterior head positioning.8 Patients should also be informed to keep the tongue touching the palate with their teeth unclenched. In certain situations, if cervical spine range of motion needs correction, physical therapy involving cervical mobilization techniques may be used. These techniques, whether direct (manual) or indirect (through specific therapeutic exercises), address dysfunctions found in the cervical spine.9 Treatment is aimed at restoring normal joint relationships and range of motion as well as restoring muscles to their original resting length. Traction, moist heat, ultrasound, and massage are used only as needed to facilitate the mobilization.

Trigger-Point Therapy

Trigger-point injections serve as a diagnostic and therapeutic technique. In myofascial pain, injecting local anesthetic (usually 1 percent procaine) into the tender or active trigger point will decrease the pain temporarily.7,8 It is believed this should be done to relieve the pain so that the patient may function normally for some time, thereby allowing central inhibition to activate. Doing the trigger point injections without addressing perpetuating factors and providing an exercise program has limited value.10

There are other means whereby the trigger point may be converted from active to latent, using spray and stretch, massage, moist heat, ultrasound, electrical stimulation, or other distraction techniques.8,10-12 Applying a distraction stimulus (cold in fluoromethane spray and stretch) allows the muscle to be exercised without restriction. This normal function may be the necessary input to trigger central nervous system inhibition. Botulinum toxin has been described for muscle pain and headache.13 Its function is likely independent of the muscle paralysis it creates, instead it may exert its effect by reducing peripheral neural sensitization.14 Acupuncture points often coincide with the trigger points, and needling these points may in part affect pain through peripheral and central processes. Acupuncture may not be more effective than placebo or massage.15,16

Nerve block

Neural blockade is useful in neuropathic pain conditions. Neuropathic pain by definition requires there to be damage to the peripheral or central nervous system to activate the pain mechanisms.17 The injury may be obvious, such as that following nerve severance or stroke, or minor, such as that as following bruising, infection, or compression. Broadly neuropathic pain is defined as sympathetically maintained and sympathetically independent pain.18 Neural blockade is effective in differentiating sympathetically maintained pain (complex regional pain syndrome) from sympathetically independent pain. It may also be effective in controlling sympathetically maintained pain if used repetitively. Stellate ganglion blocks, phentolamine infusion,19 and sphenopalatine blocks have been described as useful in obtaining a chemical sympathetic block. Somatic block may help identify the pain source. Rarely is this effective as an isolated therapy. Steroid combinations may provide pro-

longed relief, but care should be exercised because local submucosal steroid use may result in tissue sloughing or if the steroid is injected into the face, the resultant fat necrosis may produce dimpling. Lidocaine infusion (200 mg over one hour) may be used therapeutically in various forms of neuropathic pain.20 It is suggested that response to intravenous lidocaine may predict who responds to the lidocaine analogue mexiletine.

Spinal blocks, including selective nerve root block and epidural and facet injections are useful if there is a local nociceptive source driving the pain. Often magnetic resonance or other imaging points to the nociceptive source. This may then be addressed using fluoroscopic guided procedures. Placing a steroid at the source may help reduce the inflammatory driver and allow physical therapy and exercises to restore function.

Neural blockade with medications other than local anesthetics and steroids has been described for facial pain. Neural destruction may be intentionally created with alcohol or glycerol. Creating anesthesia dolorosa or deafferentation pain should be considered prior. Studies with streptomycin applied to patients with trigeminal neuralgia have not been effective in placebo-controlled trials.

Topical Therapy

The use of topical therapies has not been well-studied. There is some evidence that capsaicin applied regularly will result in desensitization and relief in neuropathic pain. This may deplete substance P and thereby desensitize the pain site. The recommended dose is five times per day for five days and then three times per day for three weeks. If the patient cannot withstand the burning produced by the application, the addition of topical local anesthetic, such as 4 percent lidocaine gel, may prove useful. Topical anesthetics on their own are effective in acute pain states and have limited use in chronic pain. Lidocaine patches are beneficial if the pain is extraoral, but continuous intraoral delivery is a challenge. Using a neurosensory shield may allow longer applications intraorally.21 The dentist may manufacture a custom acrylic stent to fit over the pain site. This is held in place, and the topical agent is repeatedly applied to the gingival surface. Clonidine can be applied to the hyperalgesic region by placing the proprietary subcutaneous delivery patch where it is most tender. Alternatively, the use of a 4 percent gel can be compounded and delivered over a larger area. Topical clonazepam (0.5 to 1.0 mg three times per day) has been effective at reducing a burning oral pain.22 Patients were instructed to suck a tablet for three minutes (and then spit it out) three times per day for at least 10 days. Serum concentrations were minimal (3.3 ng/ml) one and three hours after application. Woda hypothesized there was a peripheral not central action at disrupting the neuropathologic mechanism. One may consider using other topical agents such as ketamine, carbamazepine, amitriptyline, nonsteroidal anti-inflammatories, and steroids; but their benefits have not been systematically studied.

Splint -- Intraoral Orthotic Device

There are numerous splint designs and as many theories as to how and why they work for pain. The exact mechanism whereby patients are helped by splints is elusive. It is recommended that the stabilization appliance be used because the relative risk is minimal.23 Splint therapy still remains the indicated therapy for temporomandibular disorders and muscle pain. Its use for migraines, where there is no temporomandibular or muscle involvement, is not well-studied.24 It does not seem that occlusion is a major factor in TMD, and significant alterations in occlusion as a first line of therapy should be avoided.

Surgery

Although not suggested as a therapeutic modality for trigeminal neuropathic pain, surgery is an excellent alternative for trigeminal neuralgia. The most effective surgical approach remains microvascular decompression.25 Advances in microvascular decompression include the use of an endoscope. This allows clearer observation and is less traumatic.26 Gamma knife radiosurgery is a recent advance for trigeminal neuralgia.27 This technique offers a relatively non-invasive means for lesioning the trigeminal nerve adjacent to the pons using a 4 mm collimator helmet. Complications are rare, and to date the author has seen one case of trigeminal dysesthesia attributed to the procedure. There are numerous other surgeries that may be useful in orofacial pain. Use of surgery for TMD depends upon the etiology. Arthrotomy and open joint surgery are far less necessary since the improved use of arthrocentesis and arthroscopy.28 Future surgical care may include neural stimulation. Currently this is experimental.

Pharmacologic

Pharmacologic intervention for chronic orofacial pain is sometimes essential to allow central nervous system inhibition and facilitate the peripheral therapies. It is essential that the dentist treating chronic pain understands that the medications used to alter pain in the trigeminal nerve distribution may act centrally or directly on the nerve to reduce the pain and suffering. Often, the medications fall into categories such at antihypertensives

(beta blockers, calcium channel blockers, alpha adrenergic agents), antidepressants (tricyclic antidepressants, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors), antiepileptic drugs (membrane stabilizers, GABAergic drugs), or specific receptor agonists that are not Food and Drug Administration-approved for pain but are commonly used "off label." Certainly, the classes specifically approved for pain conditions such as anti-inflammatories, muscle relaxants, narcotic and non-narcotic analgesics, triptans, and ergots are also commonly used for the chronic orofacial pain patient.

For chronic benign pain, the goal should be to limit narcotic use. In certain circumstances, there are no alternatives; and carefully controlled use is necessary. It is recommended that patients understand the therapeutic goal is to keep them functional. If they maintain the agreed behavioral function, continued narcotic use should be provided. If these behaviors are not met, withdrawal from this treatment may be needed. It may be useful to have this withdrawal performed by a detoxification specialist. If medication is to be withdrawn, a protocol for this must be outlined. At times, a blinded process is used to help reduce anxiety over stopping the narcotic. Consent is obtained when using the blinded pain cocktail. The medication is reduced usually by 20 percent per week. Care should be provided to deal with withdrawal symptoms.

Table 1 summarizes the common medication classes used in orofacial pain. This is not an exhaustive list, and dentists treating orofacial pain may use other groups not listed.

Behavioral

Following a behavioral evaluation, management is directed at the factors that may affect treatment and determining the most appropriate interventions. Consideration should be given to the following factors:

* Behavioral or operant;
* Emotional;
* Characterlogical;
* Cognitive;
* Side effects;
* Medication use; and
* Compliance.

To reduce behavioral stressors, stress management and relaxation skills are used. Cognitive behavioral training provides skills for coping with daily life stresses, depression, and pain.29,30 In addition, patients are presented with information regarding myofascial pain and operant aspects of pain and medication use.

Conclusion

Because pain comprises nociception, behavior, and suffering, careful attention to creating a comprehensive therapy is essential. The clinician should not revert to a psychogenic etiology as a default, rather, understanding that all pain, no matter what the etiology, has a behavioral component will allow the patient the opportunity to receive behavioral therapy alongside antinociceptive modalities. The current understanding that pain may be generated from peripheral and central mechanisms further warrants the therapy to be aimed not only at the peripheral source, but also at the brain pain inhibition systems. A patient who has a toothache after an extraction or root canal therapy may not have a peripheral source for the pain, rather the pain may be generated by an ongoing process in the trigeminal nucleus or elsewhere in the brain. Treatment aimed solely at the tooth site may only worsen the situation, whereas treatment aimed at stabilizing neural excitability or enhancing central inhibition (e.g., medication and/or behavioral intervention) may reduce the pain, behavior changes, and suffering.

Current advances in knowledge regarding pain mechanisms have made therapy for chronic pain patients more successful. Patients should be afforded therapy from as broad and all-encompassing a base as possible.

Table 1. Medications Used in Orofacial Pain

Analgesics
Narcotic
Non-narcotic
Antidepressants
Tricyclic antidepressants
Selective serotonin reuptake inhibitors
Monoamine oxidase inhibitors
Antiepileptic drugs
Membrane stabilizers
GABAergic agents
Antihypertensives
Alpha blockers
Beta blockers
Calcium channel blockers
Muscle relaxants
Serotonin antagonists
Serotonin agonists
Ergots (nonselective)
Triptans

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Pitfalls of Being a Patient Record Pack Rat

Robert E. Horseman, DDS

There are many reasons to look about our office with pride. Our collection of used diamonds, for example, is second to none. The 3M Company has recognized us as being the most innovative users of yellow Post-It notes in the Dental Offices Under 1,000 Square Feet Division. The Southern California Edison Company has often publicly marveled at the number of extension cords we have emanating from a single power outlet.

Yet, there is one area that threatens to erase the smugness of these accomplishments. It is the disposition of patient records. Our custom over the years has been to simply decamp from our venue when patient records reached the point where they occupied 68 percent of the total office space, leaving the next tenant the task of disposal. Nomadic tribes used to do this when their accumulated refuse gave even the most tolerant of them migraines. The heady feeling of the chance to start over with a clean slate is admittedly attractive but can interfere with the continuity of treatment. This does not mean you shouldn't move just beyond the limit that lower denture patients are willing travel to seek you out.

So we came up with Plan B: a simple solution, really, involving the removal from our files of all the patients who had not visited during the past 10 years. Although this has the desirable effect of thinning the herd, so to speak, it has also produced an inactive file approximately 100 times the size of the active file, and that's why we can't get the car in the garage anymore.

Even more depressing is the discovery that we are facing what appears to be thousands of individuals who, because they have not been in for 10 years or more, force us to ask ourselves "Why?"

What immediately comes to mind, of course, is the distinct possibility that the work we did for them was so good they will never require any more dentistry.

We concede that some may have moved out of the area or to that ultimate "beyond," but what about those who didn't return because we hurt them, we didn't live up to their expectations, we were too expensive or, worse yet, too cheap? Were we too old, too hairy, too pushy, too wishy-washy, too fat, too emaciated or so totally lacking in charm and ordinary social graces that wild ponies couldn't drag them back?

Write this legibly on a yellow Post-It and stick it on your forehead: DON'T GO THERE! Analysis of one's shortcomings is an exercise best left to one's spouse. Instead, work on getting your active files, now purged of all these missing hordes, into some sort of recognizable alphabetical order. "Alphabetical" is the keyword here. Sometimes temporary staff has

innovated a filing system involving first instead of last names, or hair color, thus ensuring themselves an indispensable position as the only employee able to find anything.

You realize that all these missing people were the recipients of your recall cards, the ones with the charming little first molar brandishing a toothbrush and asking that they call your office RIGHT NOW for an appointment because it has been six months since their last visit and you are worried sick that their oral health will be endangered if they procrastinate a minute longer. These are the cards that cost 23 cents apiece to mail and carry the same imperative impact that other unsolicited junk mail delivers.

There is a theory that the surest way to see a long-absent patient suddenly reappear is to place his or her records in an inaccessible place, perhaps in an incinerator. This is an unreliable ploy at best, vying with the recall card in results, but cheaper.

The law states that patient records must be maintained for a minimum of seven years. Why seven instead of five or eight, nobody knows. Why are there seven days in a week, or why can a soft drink with a name like 7 UP be bought at a 7-Eleven? It never came up for a vote.

One of the enduring characteristics of dentists is that they never throw anything away. That's why their cupboards are full of stuff for which they have no earthly use. If it weren't for assistants who daringly give the heave-ho to vast quantities of this junk when the doctor is on vacation, the whole profession would grind to a halt for lack of space. We don't need a law to tell us to keep all these records, we would just keep them with all the other useless stuff anyway. We can't help it. But really, who cares what we did on Joe Blow 10 years ago? We can't even read our writing. What we want to know is what are we going to do with Joe right NOW.

Which brings us to Plan C. All the dentists in your town who are speaking to one another gather up all their ancient records and anything else they are willing to relinquish. We stack all this impedimenta in a huge pile after getting the proper permits from the City Council, the Fire Department, the EPA and the ACLU, and torch it.

The act of culling, of purging, of expunging can be a liberating experience. Deny yourselves no longer!