CDA

Toxicity & Allergies
Vasoconstrictors
Complications

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LOCAL ANESTHESIA

Stanley F. Malamed, DDS

OF THE CALIFORNIA DENTAL ASSOCIATION
Journal
CDA Journal
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DEPARTMENTS

644 The Editor/*A Lesson in Diversity*
645 The President/*The Historical Perspective*
647 Commentary/*Reaching for Higher Standards*
650 Impressions/*Odontology Often Is Final Piece to Grim Puzzle*
708 Dr. Bob/*Geezerhood*

FEATURES

658 MAXILLARY NERVE BLOCK: THE PTERYGOPALITINE CANAL APPROACH

Providing a profound block of the nerve V2, the maxillary nerve block via the pterygopalitine canal allows the dentist the ability to perform procedures anywhere in the maxillary quadrant that has been anesthetized.

by J. Mel Hawkins, DDS and David Isen, DDS

665 THE DIFFICULT TO ANESTHETIZE TOOTH

A discussion of anesthesia strategies available for the "difficult to anesthetize cases".

by Christine L. Quinn, DDS

668 VASOCONSTRICTORS AND THE HEART

The use of vasoconstrictors in local anesthetics as topical hemostatic agents and in gingival retraction cord remains controversial although data exists from which to formulate reasonable guidelines.

by Thomas J. Pallasch, DDS, MS

677 LOCALIZED COMPLICATIONS FROM LOCAL ANESTHESIA

A review of possible adverse events that may result from local anesthetic administration.

by Daniel A. Haas, DDS, PhD

683 TOXICITY AND ALLERGY TO LOCAL ANESTHETICS

Inspired by addictive and often toxic cocaine, local anesthetics used today are safe and effective, although some rare toxicity and allergic reactions do exist.

by Andrew Chen, DDS

CDA JOURNAL, VOL 26, Nº9

A Lesson in Diversity

Jack F. Conley, DDS

Last month, the Journal published an opinion by CDA member Jay M. Hislop, DDS, entitled, "Losing control -- The Deprofessionalization of Dentistry." A portion of that commentary linked AB 1116 legislation to the theme of the commentary and generated significant negative reaction from many members of the association. It also elicited concern within leadership ranks of the association because the discussion was contrary to CDA policy which was instrumental in the support and passage of AB 1116. I will return to these issues shortly.

Two points must be made initially. First, every member of the Association has the freedom or right to express his/her point of view in the CDA Journal or Update publications. Second, this does not mean that publication of a member opinion, feedback letter, or commentary represents the position of the association, the editor, or editorial staff. Publication does not imply that any of these entities is sympathetic to the opinion or to any side of a controversy that results from its dissemination. To be succinct, I quote an excerpt from the disclaimer which is published under the title "Contributions" on the masthead page of every issue of the CDA Journal:

"Neither the editorial staff, the editor, nor the association are responsible for any expression of opinion or statement of fact, all of which are published solely on the authority of the author whose name or initials are indicated."

Returning to the issues at hand, feedback received has demonstrated that there really are two issues in this controversy rather than one, and that is why we have a controversy with some angry members. To be specific, the usual discussions in this publication involve philosophical debates on dental science and technique issues such as TMD or cosmetic dentistry, or matters of dental policy or politics of the association. In this instance, the matter of conflict with association policy on AB 1116 was recognized immediately. The review process resulted in an Editorial Note that was appended to the article in an effort to clarify the legislation and CDA's stance. In retrospect, even that effort, while well intended, was probably not adequate to manage that issue.

The other issue that emerged from this controversy is not normally part of the discussions that appear in the Journal and failed to attract our attention when the material was reviewed. It is really at the heart of the controversy that has angered many loyal CDA members who have foreign training. I have learned from this experience that degrees earned and the skills and knowledge they represent are very "personal," and any opinion that deals with personal matters should be handled with great sensitivity. To the extent that we fell short of that standard in this case, we certainly apologize to any and all who were offended. No insult was intended.

Nonetheless, we must protect the right of members to be heard, and, at the same time do our best to provide a balanced perspective on controversial matters. While the process is intended to encourage member dialogue, it will always place contributing members at some risk in that others may oppose their view. It is not the position of the editor or other leadership to side for or against the position of an individual member, except to very clearly educate the membership to the adopted association positions and/or legislative initiatives.

In the space that follows, we offer some presentations that will hopefully place the controversy into careful focus and address the other side of the issue raised by the commentary. Central to our purpose is the objective of achieving a complete and accurate understanding of the AB 1116 legislation by every member of California Dental Association. CDA President Kenneth E. Lange will address the historical perspective and association position in detail, followed by member commentary and feedback.

It is my hope that this presentation will fully carry out our responsibilities to resolve the controversy that has arisen this month and will help to restore the understanding that is so important to our profession in this era of diversity.

CDA JOURNAL, VOL 26, Nº9

The Historical Perspective

Kenneth E. Lange, DDS

A recent opinion piece by member Dr. Jay Hislop generated significant controversy and misunderstanding. The section of the article dealing specifically with AB 1116 (Keeley) is central to the concerns that have been expressed. Outlined below are the factors involved which culminated in the passage of AB 1116. Following that, CDA was an active proponent of the legislation. I remain convinced it is in the best interest of California's dentists and consumers.

The California State Legislature first addressed this subject almost thirty years ago because of a perceived shortage of U.S. trained dentists to serve the state's growing population. The Legislature directed that State Board of Dental Examiners to establish a procedure to license dentists who were not educated in U.S. accredited dental schools. In response, the State Board adopted regulations in providing for a restorative technique examination, or bench test to evaluate the competency of foreign-trained dentists seeking a California license.

Over subsequent years, the California Dental Association and a coalition of foreign-trained dentists became increasingly concerned about the efficacy of the restorative technique examination. The object of AB 1116, in part, is to raise the licensing standards for foreign-trained applicants.

During consideration of the provisions of AB 1116, the CDA leadership and Council on Legislation solicited input from membership through numerous articles in the UPDATE and JOURNAL regarding provisions of the proposed legislation and the importance of establishing uniform educational and clinical standards to obtain a California license.

The statement in the commentary that the Board of Dental Examiners "floods" the professional market with practitioners of limited skills and experience" is incorrect. Many of the foreign-trained dentists applying for license in California have exceptional training and considerable professional experience. This statement has been construed as an insult to foreign-trained members of the coalition, CDA members themselves, who conscientiously sought to correct the inequity in the state licensing procedures, as well a all foreign-trained dentists licensed currently, and applicants who qualify in the future.

The provision of AB 1116 which seems to have created the most controversy permits foreign-trained dentists to append the DDS designation to their names. After considerable deliberation, the Council on Legislation and Executive Committee concluded that its focus must be on assuring comprehensive and uniform educational and clinical training rather than debate over title. Moreover, concern about the inference of a dentist's skill and education by the designation is eliminated by the elevated standards established by AB 1116.

I am in complete agreement with CDA's position to support AB 1116 establishing uniform educational and clinical standards for all California dental license applicants. If anyone has a question about the CDA position on this issue, I suggest they review these comments again in Brian Blomster's in-depth article in the July 1997 CDA Journal.

CDA JOURNAL, VOL 26, Nº9

Reaching for higher Standards

Ed de la Vega, DDS

I read with very keen interest, the commentary of Jay M. Hislop, DDS, the President of the San Joaquin Valley Dental Society in the August 1998 CDA Journal. Some of his comments made sense, but a whole lot of what he said about AB 1116, and foreign-trained dentists is absolute nonsense! Following is my response to Dr. Hislop's commentary.

Where was Dr. Hislop when the CDA House of Delegates was discussing this issue? Change has been upon us. And further change is inevitable. Yes, there is a lot of value in this thing called "diversity." CDA and ADA are overly-focused on diversity because of its impact on membership in big urban cities like Los Angeles and San Francisco. Give it time. Soon it will be in every city throughout California. No matter what is said, and for that matter, whatever barriers are placed, graduates of foreign schools will continue to come to California. These dentists, because of cultural and family values that emphasize hard work and good clean lifestyle will continue to prosper much like the others who blazed the path for them. A lot of them will eventually join CDA and some may even become the leaders who in the next millennium will lead CDA and the profession to a higher level than what it is today.

I advise everyone to become familiar with the AB 1116 legislation. Nowhere does it say that the California Board of Dental Examiners "confers" the title DDS to anyone as was stated by Dr. Hislop. It merely states that the bill will "authorize any person who holds a valid license, unrevoked and unsuspected certificate as a dentist in California to append the letters DDS to his or her name regardless of the degree conferred upon him or her by the dental college from which the licensee graduated." This is not the first time this has been done in California. People with the Doctor of Osteopathy (OD) degree can append the letters "MD" after their names if they pass the State Medical Board. What is in the degree or name anyway? What counts most is not the title or degree, but who you are as a person and a professional, and, what kind of dentistry you deliver!

The CDA and the coalition of ethnic minority dental societies recommended the changes and the State legislature agreed, because some dentists, U.S. graduates included, who practice in California have degrees that directly translate to DDS, although they are not written in English. (Even the old Northwestern dental school diploma is in Latin!) The general idea was to have one common designation so that the public will not be confused, and, the "playing field will be level."

I must take issue with any claim that the dental training of foreign-trained graduates is "nothing more than a technician course in a foreign country." I served the California State Board of Dental Examiners for four years as a member of the Examining Committee and for four years as a Member of the Board. I know what I saw in those eight years. While I respect the values of "accreditation," I do not agree that all graduates of accredited schools are necessarily better than those from foreign schools. Dentists should not be stereotyped simply because of the origin of their dental degrees!

If foreign-trained graduates are inferior, why is it that California schools have secured "special permits" for some of us to teach in their schools? Just browse through the faculty rosters of the dental schools in the state and you will find foreign graduates as department chairpersons. The former Dean of UCLA School of Dentistry, who recently was appointed as a UC Chancellor, is foreign-trained. Furthermore, foreign graduates are authors of a good number of books found in our schools.

I believe that the school deans agreed with the provisions of AB 1116 because it was the best way to clarify the licensing standards. I sensed this when I testified in Sacramento with University of the Pacific Dean Arthur Dugoni in support of AB 1116. As you know, by the year 2003, all applicants for the board exams must have a degree from an accredited school. Schools may be located in Canada, the USA, or in any foreign country as long as California accredits the school. AB 1116 makes the Board of Examiners responsible for approving foreign dental schools, and thus, in the near future, as CDA Past-President Eugene Sekiguchi stated in the CDA Update in February 1997, "We need not worry about this state collecting all the unaccredited dentists in the world." In the year 2003, foreign graduates who have not graduated from an accredited school must complete an approved two year program at a California school of dentistry.

We, the foreign-trained dentists, suspect that the equivalency in training "issue" raised by Dr. Hislop is instead a smokescreen based upon fears of economic impact. We believe that California is a large enough state to sustain all dentists who choose to practice here and meet the state's licensing requirements.

I believe that allowing a few CDA members who are licensed dentists with unblemished records (whose only fault is that they are foreign-trained) to append the letters DDS after their names is a fair exchange for establishing the new standard in the year 2003. Rather than deprofessionalize dentistry, the new requirements should reach for the higher standard that Dr. Hislop seeks.

CDA JOURNAL, VOL 26, Nº9

Odontology Often is Final Piece to Grim Puzzle

A man fails to return home after a weekend fishing trip in the mountains. His wife reports him missing, and a weeklong search ensues. Months go by before a hiker stumbles upon some sun-bleached human bones near an alpine stream.

The discovery is reported to authorities, and the county coroner calls in an expert in identification -- a forensic dentist, one of a small cadre of specialized volunteers assisting California's Department of Justice in identifying missing individuals.

Forensic dentists, also known as forensic odontologists, help match nameless human remains with some of the Golden State's approximately 2,000 unidentified victims from as far back as 1959.

Identification is accomplished through studies of dental remains, which include postmortem X-rays, photography, and creation of dental records to catalog unique restorations or other identifying qualities. The postmortem information is sent to Sacramento, Calif., where it is compared with dental records of missing people on file at the Department of Justice.

The identification process is important in forensic odontology and can present substantial challenges depending on circumstances surrounding the case.

"One remarkable case was when a single tooth was found in addition to some pelvic bones that enabled us to determine it was a female," says Norman D. "Skip" Sperber, DDS, a senior forensic odontology volunteer with more than 30 years' experience. "It was a central upper incisor containing a particular restoration. We searched the Department of Justice records of missing women, made a positive identification, and her killer was later identified and apprehended."

Before the late 1970s, solving missing-persons cases was haphazard at best. No coordinated statewide procedure existed, and Sperber thought there had to be a better way. In the first effort by a state to organize dental records to make matching them with unidentified deceased people easier, he and two other forensic dentists convinced a member of the state Legislature that a law was needed. Their work resulted in a statute enacted in 1979 requiring dental records of all missing persons to be sent to the Department of Justice.

The system has worked. Duane E. Spencer, DDS, a pediatric dentist practicing in the East Bay who since 1974 has participated in identifying more than 250 missing people, recalls one case in particular.

"A number of years ago, a case made television's America's Most Wanted show after a man was murdered in Berkeley," Spencer says. "His dismembered body was scattered. Months later the skull finally turned up. The skull and teeth helped us identify the victim, eventually leading authorities to the murderer. He was found guilty and received the death sentence."

Spencer said the identification was relatively straightforward because the victim's dentist had kept good records of his care.

Another volunteer forensic odontologist, James D. Wood, DDS, in Cloverdale, north of San Francisco, also emphasizes the importance of good recordkeeping.

"Without good records, especially good radiographs, there is no way to compare a possible match to a set of postmortem remains. That's the critical feature," Wood says. "The written record is also incredibly important because it documents what was done on a patient. I can't stress enough the importance of not destroying records, and that means X-rays as well. Everyone deserves the dignity of their identity."

California's handling of missing-persons cases has had a nationwide effect, leading to a federal law that mirrors what the state has done, according to Gerald L. Vale, DDS, an associate dean of the USC School of Dentistry.

"Many remains found here came from other states, so working with our own data we won't be very successful," says Vale, a veteran of more than 30 years of forensic dentistry. "We need a nationwide database, and we're moving in that direction with proposed new federal legislation called Jennifer's Law."

"The benefit would be more people identified, and this will benefit families because of the obvious emotional and financial concerns. From the law enforcement point of view, it's tremendously important, because the solution of homicides results from knowing who the victim is."

White Smile Equated to Healthfulness

Being perceived as healthier and more attractive may be as simple as flashing a healthy white smile, according to the Crest Extra Whitening Smile study. Conducted in conjunction with Custom Research Inc., and research and psychology experts, the study surveyed 200 male and female respondents of various ages and ethnic backgrounds in eight cities in the United States.

Study participants were asked to rate two photos of the same people before and after their teeth had been digitally manipulated to be whiter. Each participant looked at 20 pairs of photos. Respondents were not informed of the manipulation process, and the tooth color changes were slight.

The results show that a 90 percent of study respondents viewed the people with whiter teeth as healthier and more attractive, regardless of age, sex, or ethnic background.

CDA JOURNAL, VOL 26, Nº9

Find the Target, Hit the Mark

The Internet has greatly increased people's ability to access research material and other information. Search engines are invaluable when it comes to sifting through mounds of information, but sometimes the results can be overwhelming. How can you narrow your search? Following are some suggestions:

- Quotation Marks. Placing words within quotation marks creates a phrase. A match is returned only when the exact word sequence is found. Example: "dentistry in the 18th century."
- A plus sign. Adding a plus sign (+) directly in front of a word requires that the word be included in all search results. Example: tooth+decay.
- A minus sign. Adding a minus sign (-) directly in front of a word indicates that the word should not be found in search results. Example: porcelain-teacup.
- An Asterisk. An asterisk (*) is a wild card. It must be placed on the right-hand side of a word or embedded within a word with at least three characters to the left. Use an asterisk to find various spellings or related words. Example: dent* will return matches of dent, dents, dental, dentist, dentistry, among others.
- And: Search results must contain all words joined by the "and" statement. Example: restorations and ceramics.
- Or: Search results must contain at least one of the words joined by the "or" statement. Example: needles or syringes.
- And Not: Search results cannot contain the word that follows the "and not" statement. Example: crowns and not royalty.
- Parentheses: Use parentheses to build complex search queries that incorporate other special words and characters. Example: San and (Francisco or Diego) results in lists of sites about either city.
- Capitalization: Searches typed in all lowercase letters will match for either uppercase or lowercase letters. Uppercase letters in a search word will match only to uppercase letters. Generally, it is better to use lowercase letters in your search phrases.

Vale reminds all dentists of other ways they can help.

"By law, California dentists must put a patient's name or social security number in all full or complete dentures," he says. "We've had cases of bodies identified because of this."

Department of Justice officials welcome the efforts of California's volunteer forensic dentists.

"As a result of dentists who volunteer for us, we've been able to identify five additional unidentified deceased persons in the last year-and-a-half," says Jeannine Willie, supervisor of the Missing/Unidentified Persons Unit, Office of the Attorney General, California Department of Justice. "In each of these cases, if it weren't for the dentists an ID could not have been made. All leads were exhausted. The dentists who volunteer for us are the greatest."

Dentists who want to know more about forensic dentistry can attend a lecture being presented by the California Society of Forensic Dentistry. The next lecture will be Sept. 25, 1998, in San Diego. Vale, Sperber, Spencer, Wood, and six others will make presentations. Contact Rick Cardoza, DDS, at (619) 444-6195 for more information.

Soul-Searching Authors Sought

Chicken Soup for the Dental Soul is looking for authors. On the heels of several "Chicken Soup" bestsellers, including Chicken Soup for the Teenage Soul and Chicken Soup for the Pet Lover's Soul, a new book is in the works about the "soul" of dentistry. Dental professionals are invited to submit one or more true, dentistry-related short stories for the new Chicken Soup for the Dental Soul story contest.

Suitable story subjects might deal with parenting, the Tooth Fairy, kindness, kids at the dentist's office, self-esteem, attitude, overcoming obstacles, perspective, teaching and learning, special moments, and living one's dream.

Contest-winning stories will be selected by the Chicken Soup organization. Author of the first-place story will receive $2,000; the second-place winner will receive $1,000; and the third-place winner will receive $500. Authors of all other stories published in Chicken Soup for the Dental Soul will receive $300. All awards will be paid within two months of publication of the book. Receipt of all stories submitted will be acknowledged by postcard. Entries are due Dec. 1, 1998.

For more information, contact Don Dible at (408) 739-4020 or E-mail at dentalsoul@aol.com.

CDA JOURNAL, VOL 26, Nº9

Communication Can Lead to Complaint Resolution

Many peer review complaints involve communication problems between the patient and the dentist. With direct and specific communication with patients, many misunderstandings can be avoided. Following is a list of suggestions by the Oregon Dental Association to help dentists better communicate with patients:

1. Discuss fees for services to be performed before commencing treatment.

2. Discuss all treatment options and risks with the patient before commencing treatment. Don't simply make a treatment decision for the patient. Document discussions with patients and agreements made in each patient's chart.

3. Pre-authorize treatment to be done with the patient's insurance company before performing the procedure, and share the outcome of prior authorization with the patient before beginning treatment.

4. If a patient is dissatisfied with treatment received, the dentist should communicate with the patient. Staff should not be assigned to attempt to resolve the issue. The patient wants to talk to the dentist. Staff communication with patients in that circumstance often shifts the focus to a billing dispute instead of addressing the patient's discomfort or dissatisfaction with treatment.

5. Suggest peer review if dentist and patient disagree on the quality or appropriateness of treatment you have provided.

6. Communicate verbally with the patient before his or her verbal complaint turns into a written complaint or demand.

7. Discuss office policies with patients before commencing treatment. Document office policies and have the patient sign or initial that they understand these policies.

8. Handle patient problems or concerns immediately. Don't let issues fester. Handle them at the onset.

9. Forward patient records within 14 days when requested either by the patient or another practitioner.

10. Seek help from a colleague or local dental association when dealing with a difficult patient or if treatment is not working out as planned.

HIV Spread Drops in Some, But Increases in Others

National Cancer Institute researchers report a "striking drop" in the spread of HIV infection among young men through homosexual contact and drug use during the early 1990s.

Published in the June 17 Journal of the American Medical Association, the NCI study found "the best available data indicate an increase in heterosexual transmission to young women, particularly young black women."

The study, conducted by Philip S. Rosenberg, PhD, and Robert J. Biggar, MD, of the National Cancer Institute, is the most detailed yet of HIV infection trends among young adults, Rosenberg said in an NCI news release. He added that it provides some insight into the effectiveness of public health efforts to battle HIV.

Biggar and Rosenberg estimated that 50 percent fewer white men aged 18 to 27 were living with HIV infection in 1993 than in 1988, NCI reported. The number of young minority men living with HIV, though, was about the same in 1988 and 1993. But the number of black women aged 18 to 27 living with HIV infection in 1993 rose more than 60 percent from 1988.

As for the years since 1993, Rosenberg is optimistic that the trends showing declines in new cases of HIV attributable to homosexual contact and drug use have continued.

"If fewer people have the disease, there is less chance of encountering an infected person, which tends to slow the spread of the disease," he says.

He added that it is possible new therapies reducing the viral load may also mean people carrying HIV are not as infectious.

The researchers are particularly concerned by the high rate of heterosexual transmission among young minority individuals.

"Most HIV-infected young people, about two-thirds, are black or Hispanic," Rosenberg says, but those minority groups comprise just 27 percent of the population of the age groups studied.

CDA JOURNAL, VOL 26, Nº9

Caries Protection Doesn't Come in a Bottle

Some public health officials are concerned about the increased reliance on bottled water and the potential impact it might have on dental caries, especially in young children, says Warren LeMay, DDS, in the April 1998 WDA Journal.

Parents, dentists and other health care providers should consider the consequences of children drinking water with inadequate fluoride levels, LeMay says.

According to LeMay, unlike many community water systems, most types of bottled water lack sufficient fluoride. Says LeMay, "It seems clear that unless an individual uses enough of other sources of fluoride, there is an increased risk for dental caries. Appropriate levels of fluoride in the drinking water can reduce dental caries by up to 30 percent."

Americans drink nearly 3 billion gallons of bottled water a year, reports LeMay. Increased consumption may be particularly significant among consumers with health concerns or with compromised immune systems.

LeMay suggests that dentists and physicians evaluate the fluoride intake of children and discuss with parents the possible need for dietary fluoride supplements, fluoride mouthrinses or more intense professional supervision.

Dentists should also inquire whether a home is equipped with a water filtration system. Two of the most common water filtration systems are carbon filtration and reverse osmosis. Carbon filtration systems do not remove fluoride, but reverse osmosis systems can remove about 95 percent of fluoride from the water. Parents with a reverse osmosis system may be unaware that their system is removing most of the fluoride from their drinking water, LeMay says.

CDA JOURNAL, VOL 26, Nº9

Maxillary Nerve Block: The Pterygopalitine Canal Approach

J. Mel Hawkins, DDS and David Isen, DDS

ABSTRACT Over the past few years, the dental industry has introduced a number of new local anesthetic delivery systems. Despite the advantages of many of these newer techniques, conventional needle and syringe procedures remain the cornerstone of local anesthesia for most dental practitioners. Most of these "tried and true" approaches are both popular and comfortable for the dentist since they are widely taught in undergraduate dental curriculum.

There is one technique, however, that although relatively safe, effective and reliable, is not usually included in undergraduate dental programs. This technique, the maxillary nerve block via the pterygopalitine canal, provides the patient with a profound block of nerve V2 and allows the dentist the ability to perform procedures anywhere in the maxillary quadrant that has been anesthetized.

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Regional block anesthesia is very common in dentistry -- primarily in the mandible. The mandibular block is an essential procedure to anesthetize the lower dentition due to the thick cortical plate of bone. It is this bony plate that, in most cases, inhibits the ability of infiltrated local anesthetics to diffuse to the root apicies. Although true for mandibular molars, the anatomy of the maxilla is different in that a thinner cortical plate will allow infiltration anesthesia in most situations. Despite this fact, the ability to block the maxillary nerve has many advantages.

The literature describes three techniques to block the maxillary nerve; one extraoral and two intraoral approaches. The extraoral approach, which is described in detail by Nish et al, will not be discussed here because it is not a common or practical procedure in most dental offices. 1,2,3

Intraorally, the two techniques to block the maxillary nerve are the high tuberosity approach (similar to the posterior superior alveolar nerve block) and the greater palatine canal approach. Although less predictable and prone to more complications, the high tuberosity approach may be the more comfortable procedure for the dentist because it is essentially a high buccal infiltration. The goal of this technique is to direct the needle superiorly, medially and posteriorly along the zygomatic and infratemporal surfaces of the maxilla to enter the pterygopalatine fossa. The depth of needle insertion is judged by

measuring the distance from the gingival crest of the maxillary premolar region to the infraorbital ridge approximating the foramen on the face. This depth can be anywhere from 24 to 44 mm.4

Complications associated with this technique include the lack of profound anesthesia either due to inadequate volume of local anesthetic (it may take more than one cartridge of solution to obtain complete anesthesia) or improper positioning and inadequate depth of penetration. The pterygoid plexus of veins is in close proximity to the area approached with this technique and there is a relatively high risk of hematoma.

In view of the disadvantages associated with the extraoral and high tuberosity maxillary nerve blocks, this paper will focus on the greater palatine canal approach as the most predictable, reliable and safest means of obtaining profound maxillary anesthesia.

The maxillary nerve block via the greater palatine canal was first described in 1917 by Mendel.5 Despite this technique's longevity however, it is not used frequently. This is perhaps due to the fact that it is not taught as a routine approach in a significant number of dental facilities and because the need to block the hemimaxilla does not arise as frequently as the need to block the mandible.

Advantages

As with any local anesthetic technique, there are advantages and disadvantages. The advantages of the maxillary nerve block include the ability to do quadrant dentistry without the need for multiple injections. The presence of oral infections may not allow a practitioner to infiltrate either because the infection is in the area to be injected or because of the difficulty anesthetizing a tooth with an apical abscess.6 In these cases, a maxillary block via the palatine canal may bypass the area of infection and thus not disseminate an infection by injecting in or around the involved areas.6 This approach is also useful for sinus procedures, and for the diagnosis of chronic maxillofacial pain syndromes. As a final advantage, one must consider that this technique has a greater than 95 percent success rate, with minimal risk.7

Disadvantages

The disadvantages and contraindications with this technique are few, but important. As with any block, hemostasis will not occur in the proximal areas of operation. One must infiltrate in order to obtain hemostasis. If there is infection in the area of the palatine canal, this technique cannot be used. There are also some anatomical limitations. Difficulty negotiating the canal with the needle tip may occur due to bony exostosis that can preside on the anterior pterygoid plate which compromises the posterior border of the pterygopalatine canal. Approximately 5 percent of the population have been shown to have tortuous canals that impede the needle tip.8 Also, in a study done by Westmoreland and Blanton, 300 skulls were examined and of those, 53 (18 percent) were found to have a relatively horizontal course with respect to the palatal anatomy.9 This horizontal opening would disallow the passage of the needle in a relatively superior orientation.

Anatomical Considerations

Any technique of local anesthesia must be based on a thorough and sound knowledge of the anatomy of the area to be injected and anesthetized (Figure 1).10

The second division of the trigeminal nerve arises from the gasserian ganglion in the medial cranial fossa and exits the skull via the foramen rotundum. The

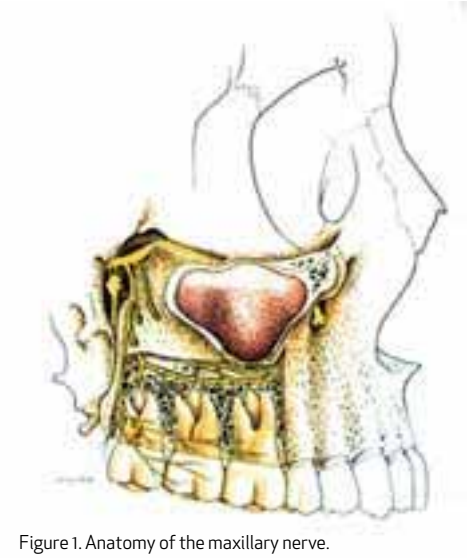

Figure 1. Anatomy of the maxillary nerve.

nerve then traverses the superior aspect of the pterygopalatine fossa, where it divides into three major branches: the pterygopalatine nerve, the infraorbital nerve, and the zygomatic nerve.

The Pterygopalatine Nerve

The main portion of the pterygopalatine nerve passes down the ptergyopalatine canal and exits toward the oral cavity at the greater palatine foramen as the anterior palatine nerve. It is the greater palatine foramen and the pterygopalatine canal that are traversed to achieve the second division nerve block.

The pterygopalatine or sphenopalatine ganglion is attached to the medial side of this nerve. However, none of the fibers of the pterygopalatine nerve synapse there. The nasal branches are emitted and the medial branches traverse the septum, ending as the nasopalatine nerve which exits at the incisive foramen.

The Infraorbital Nerve

The main trunk of the maxillary nerve continues through the pterygopalatine fossa and gives rise to the posterior supe-

rior alveolar nerve, which divides into an external gingival branch and the internal dental branch. The main nerve trunk then enters the inferior orbital fissure and travels through the floor of the orbit in the infraorbital canal. While in this canal, it emits the middle superior and anterior superior alveolar nerves, which sometimes exist as a nerve plexus. These nerves travel along the lateral sinus wall to the premolar and anterior teeth, respectively, and send sensory branches to the maxillary sinus mucosa. In approximately 30 percent of the population, the middle superior alveolar nerve does not occur and, if this is the case, its areas of innervation are assumed by the posterior and anterior alveolar nerves.11 The infraorbital nerve exits the maxilla at the infraorbital foramen and divides into three terminal sensory branches -- the superior labial nerve to the upper lip, the lateral nasal nerve to the side of the nose, and the inferior palpebral nerve to the lower eyelid.

The Zygomatic Nerve

This branch emerges from the infraorbital nerve and has two divisions of its own. One communicates with the postganglionic parasympathetic fibers from the pterygopalatine ganglion to the lacrimal gland. The other exits the body of the zygoma and the zygomatic orbital foramen and supplies sensation to the malar eminence.

Anatomically, once the greater palatine foramen is located, a number of structures will be approximated by the needle tip. As the needle advances, it will transcend the greater palatine fossa and the associated neurovascular bundles. Although damage to these structures can occur theoretically, the incidence of such damage appears to be low. The incidence of positive aspiration has been reported to be only 8 percent and traumatic injury to the nerve less than 1 percent.12 The reasons for these low percentages are theorized to be due to two factors. One, the practitioner is advancing the needle virtually parallel to these neurovascular structures and therefore puncturing them is unlikely. Two, the tough perineurial fibrous sheath may offer some protection from the needle tip.1 If a dentist does enter a blood vessel, it is unlikely that it will be an artery. If the needle tip does in fact approximate an artery, the patient feels pain and moves their head away as a protective response, the artery goes into spasm, and the smooth muscle wall of the vessel constricts. This is evidenced by blanching on the ipsilateral side of the patient's face. Therefore, in most cases, a practitioner must be rather aggressive to actually enter an artery. If, however, one punctures a vein, it is possible for a hematoma to occur. This extravasation will be limited to the pterygopalatine fossa and not be clinically evident.

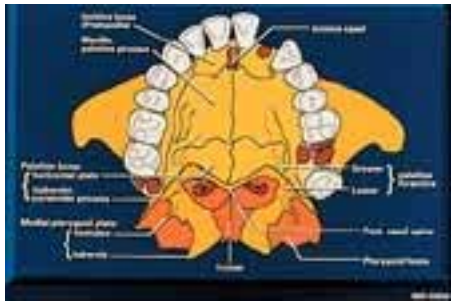

Figure 2. Anatomy of the hard palate.

Hard and Soft Tissue Landmarks

In most cases, the challenge of this technique centers around locating the greater palatine canal and being familiar with the anatomy of the palate (Figure 2). With practice, finding the canal entrance and orienting the needle correctly with respect to the midsagittal plane becomes easier. To maximize one's ability to locate the greater palatine canal, there are five landmarking steps. These are as follows:

1. Using the dentition, providing the patient has a full complement of teeth in that arch, the greater palatine canal is located between the middle portion of the third molar and the middle portion of the second molar 84 percent of the time. It is located mesial to the mid portion of the second molar 10 percent of the time and distal to the mid portion of the third molar in 6 percent of the population.9 If there is not a complete dentition in the arch on the ipsilateral side of the injection, these landmarks should be adjusted.

2. The greater palatine foramen is located anterior to the junction of the hard and soft palate. This junction is seen as a color change such that the tissue overlying the soft palate is darker pink than the tissue overlying the hard palate. This border can also be palpated using the dental mirror. The foramen can occur anywhere from 1.8 to 12mm anterior to this border with an average distance of 7mm.9, 13

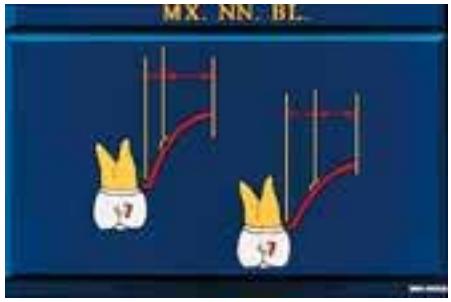

Figure 3. Depth of the palatal vault.

3. The greater palatine canal is usually located at the junction of the horizontal and vertical bony plates of the hard palate. In patients who have a very deep palatal vault, the canal opening will appear closer to the dentition. Conversely, in patients with a more shallow palatal vault, the foramen will appear closer to

Figure 4. Location of greater palatine canal.

the sagittal midline (Figure 3).

4. The greater palatine canal is located along an imaginary line from the ipsilateral hamular process to the ipsilateral cingulum of the lateral incisor (unpublished data) (Figure 4).

5. The soft tissue depression which covers the greater palatine canal is a blanched or whitish area overlying serous and mucus glands. This lighter colored tissue can also be used to locate the canal.

The lessor palatine canal is located posterior to the greater palatine canal on the posterior aspect of the hard palate. This canal is of no clinical significance to this technique since it is not possible to advance the needle into this foramen due to its extremely small size and tortuous path (see Figure 2).

Armamentarium and Technique Modifications

There are modifications which the authors believe will assist in the success of this block. For patient comfort, it is absolutely essential to do a preliminary infiltration in the region of the greater palatine foramen. This, in essence, now becomes a two-injection technique.

The first syringe is used for the preliminary injection and can be done with a 30 gauge short needle with a solution containing 1:100,000 epinephrine. The rationale for this is twofold. First, the 30 gauge needle tip will, in fact, having lined up all the landmarks, be very close to the greater palatine foramen, and, with its small needle gauge, disallow significant backflow from the small puncture. Second, as vascularity is encountered during the preparatory injection, the 1:100,000 epinephrine solution may minimize extravasation submucosally or onto the surface of the hard palate. The suggested volume is 0.5 cc.

The orientation of the pterygopalatine canal is superior and posterior. The angle that the canal creates with the horizontal hard palate can range from 20 to 70 degrees. However, 75 percent of the population have been found to have this angle

Figure 5. Bent needle.

occurring between 37 to 57 degrees.13

Therefore, the second syringe can be prepared with a bend of 45 degree at the hub, utilizing a 25 gauge or 27 gauge needle with the bevel facing posteriorly, towards the anterior border of the pterygoid plate (Figure 5). In this way, the needle bevel can 'ski' off the above-mentioned surface and advance upwards through the pterygo-maxillary fissure.

Another technique modification which the operator may wish to attempt is the "dart or pen grip". This involves holding the syringe as a pen (Figure 6). The advantage of this positioning is that it allows the dentist an extra degree of motion such that one is able to roll the syringe

Figure 6. "Pen" or "Dart" grip.

barrel between the thumb and forefinger. This may help to maneuver the needle tip inside the palatine canal, especially if it touches bone before the final depth of insertion. The dentist must never push in order to advance the needle if this occurs. If one encounters bone before the final depth of insertion, one should pull back 1 mm, re-angulate and try to gently redirect the needle tip. No force should be required for the advancement. With too much force, it is possible to penetrate bone and deposit local anesthetic into the nasopharynx. If this occurs, the patient will complain of a bitter taste in their throat. As well, the practitioner will be aware that they have entered the nasopharynx by aspirating and observing only air being withdrawn into the cartridge.

Clinical Procedure

The patient must be asked to lift their chin off their chest and to open wide. If this does not occur, it will be extremely difficult to visualize the injection site and the mandibular dentition will impede the barrel of the syringe.

The practitioner will use the five landmarks previously described in order to pinpoint an area of injection. Assuming there is no gag reflex, place the mouth mirror at the junction of the hard and soft palate and observe that the greater palatine canal will occur an average of 7 mm anterior to this (Figure 7). If there is a

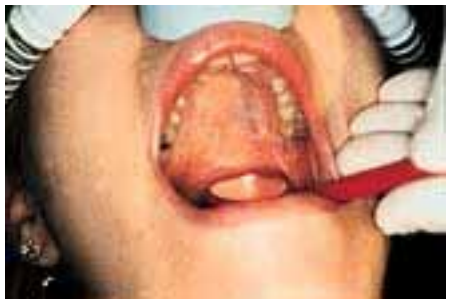

Figure 7. Hard and soft palate junction.

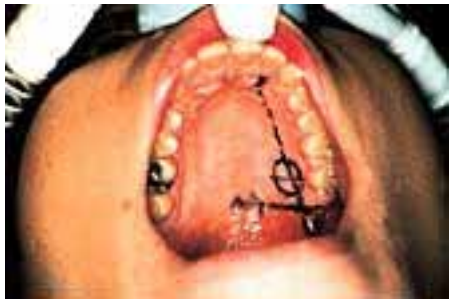

Figure 8. Location of canal.

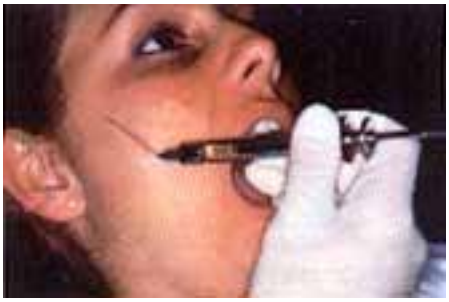

Figure 9. Angular direction of canal.

Figure 10. Depth of injection.

Figure 11. Depth of injection.

full dental arch present, the foramen will be located

between the distal marginal ridge of the second molar and the middle aspect of the third molar approximately 85 percent of the time. Next, line up the ipsilateral hamular process and lateral incisor and observe the junction of the vertical and horizontal bony plates of the palate. Finally, look for a whiter depression of soft tissue beneath which lies the opening of the greater palatine canal (Figure 8).

Now that a target zone has been established, the practitioner can administer the preparatory injection directly into this area. A local anesthetic with a vasoconstrictor concentration of 1:100,000 should be used for this injection. This is absolutely essential in order to administer a painless maxillary nerve block. To help the patient experience less discomfort from the preparatory injection, a topical anesthetic, which must be isolated from the tongue, should be used. Counterpressure from the mirror would be beneficial as well. One must ensure that this injection is given 3-5 mm submucosally so that the local anesthetic solution, in an approximate dose of 0.5 cc, does not leak back into the oral cavity. Allow two to three minutes before proceeding with the next injection during which time the second needle and syringe can be prepared.

With a 25 or 27 gauge needle bent at 45 degrees and the bevel facing posteriorly, the procedure can begin. The length of the needle should be parallel to the midsagittal plane and using the needle tip as a probe, the foramen is located (Figure 9). Gently rotate the needle until it falls up into the canal without any resistance. If bony resistance is encountered, pull back 1 mm and gently try to tease the needle tip around this. Advance in a superior and posterior direction to the final depth as measured from the gingival crest in the maxillary bicuspid region to the infraorbital foramen. This is almost the same as the depth measurement for the high tuberosity approach previously mentioned. It can be anywhere from 24-44 mm with most adults falling into the 25-30 mm range (Figure 10 & 11.) If a patient is found to have a distance of 30 mm from the gingival crest in the bicuspid region to the infraorbital foramen we must visualize 5 mm of needle remaining outside of tissue once we have reached our final depth.

Long needles are generally 35 mm in length. If the dentist encounters an obstruction that is not negotiable or if the patient has a very long face (a depth of 44 mm in the extreme), then the final depth will not be obtainable. This may not

matter since it has been suggested that if the practitioner is within 15 mm of the pterygopalatine fossa and a full cartridge of solution is injected, success should occur.12

When the final depth has been achieved, aspirate and slowly inject a full cartridge of either a plain local anesthetic solution or one with a vasoconstrictor concentration of 1:200,000 or 1:100,000 epinephrine. It should take 30-45 seconds to inject the full cartridge and the patient should be instructed that they will feel slight pressure in the middle of their face.

The time of onset for the block to occur is 3-10 minutes and, with a 1:200,000 solution, the duration of anesthesia is 1.5 - 2 hours for hard tissue and 2.5 hours for soft tissue.

Complications

The complications of positive aspiration, hematoma and neural damage have been discussed, as has the inability to introduce the needle the entire depth into the canal.

Diplopia of the ipsilateral eye may occur 35.6% of the time.12 This results from the local anesthetic diffusing superiorly and medially to anesthetize the orbital nerves. The patient must be assured that this phenomenon is transient. There are no known reports in the literature of permanent diplopia.14

Depositing anesthetic solution into the nasopharynx can occur if too much pressure is exerted on advancement of the needle. In this case, the posterior wall of the canal can be perforated. If a dentist enters the tissue of the palate too far distally with the initial needle puncture it is possible to miss the hard palate completely and advance up into the soft palate. The needle advances very easily in this area, and one may have the false sense that they are in the pterygopalatine canal. When solution is deposited in this case, it again is deposited into the nasopharynx and the patient will complain of a bitter taste and may cough. No block will occur in this situation.

Conclusion

The maxillary nerve block in the greater palatine canal is a safe and effective method for achieving anesthesia of the hemimaxilla providing that there is strict adherence to the landmarks and techniques described. This technique carries with it a high success rate and with specific applications can allow a practitioner to provide painless dental treatment.

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Injection Techniques to Anesthetize the Difficult Tooth

Christine L. Quinn, DDS

ABSTRACT Failure to achieve anesthesia can be a significant problem in the day-to-day practice of dentistry. The usual strategy following an anesthetic failure is to reinject. Therefore, a good understanding of conventional anesthetic techniques is important. But the practitioner also needs to have a broad armamentarium of injection strategies available for the "difficult-to-anesthetize cases". These strategies include the use of 5 percent lidocaine, intrapulpal injection, periodontal ligament injection and intraosseous injection. This paper will be a brief discussion of those techniques with an emphasis on the intraosseous injection.

AUTHOR

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Failure to achieve anesthesia can be a significant problem in the day to day practice of dentistry. In a survey conducted by Kaufman1 it was concluded that about five anesthetic failures occur each week in a general practice, with the inferior alveolar nerve block being identified as the injection with the greatest number of failures. Eleven percent of the dentists responding to the survey reported that they had an anesthetic failure (within the survey week) that resulted in uncompleted treatment. These patients may eventually lose confidence in the dentist, questioning if they will ever be able to provide them with adequate pain control.

The usual strategy following an anesthetic failure is to reinject. Therefore, a good understanding of conventional anesthetic techniques is important. But the practitioner also needs to have a broad armamentarium of injection strategies available for the "difficult to anesthetize cases". There are several causes for failure of anesthesia: incorrect needle placement, anatomic variation, presence of inflammation, and patient anxiety. This discussion will focus on the difficult-to-anesthetize tooth and some ways in which to improve anesthetic success.

It is difficult to achieve anesthesia in the presence of acute inflammation. It was thought that because the inflamed tissue has a low pH (5 to 6), relative to nonimflamed tissue (pH 7.3), the local anesthetic was acidified. This would decrease the amount of nonionized anesthetic that would be available to cross into the nerve. Inflammation also leads to increased blood flow in the tissue. This would speed the removal of the anesthetic from the injection site. Conventional wisdom has traditionally suggested to provide nerve block anesthesia at a site distant to the inflammation to avoid the decrease in pH. This, in fact, does not always guarantee anesthesia. One of the most difficult

situations to achieve profound anesthesia is in the mandibular molar with acute pulpitis in which the local anesthetic is delivered some distance away from the inflammation. Rood2 conducted clinical trials comparing buffered 2 percent lidocaine with epinephrine with commercially available lidocaine with epinephrine. He demonstrated that there was no difference in effectiveness between the two solutions when used for maxillary infiltration on inflamed teeth. Rood concluded that pH is not the major factor causing anesthesia failure in the presence of inflamed tissue.

It has been shown that morphological changes occur along the nerve some distance away from the inflammation..3 There is also interest in the mediators of inflammation in terms of how they may affect nerve transmission and local anesthetics.4,5

Concentrated local anesthetic solutions may be one way to anesthetize the acutely inflamed tooth. Eldridge6 demonstrated, in a double blind study, that 5 percent lidocaine with epinephrine was able to successfully anesthetize teeth with acute pulpits. There was a 93 percent success rate with 5 percent lidocaine when used for inferior alveolar block anesthesia versus a 22 percent success rate with 2 percent lidocaine. It should be noted, however, that a concentrated anesthetic solution is more toxic than standard formulations.

Another approach to the difficult-to-anesthetize tooth is the incorporation of supplemental injection techniques into the anesthesia armamentarium. These techniques include the intrapupal injection, periodontal ligament injection and the intraosseous injection.

Intrapulpal Injection

This technique is a supplemental injection technique that may be used when trying to achieve pulpal anesthesia for root canal treatment. The technique is as follows: A small opening is made in the pulp chamber. The needle is wedged against the pulp canal walls and a small amount of local anesthetic is injected with considerable back-pressure. Anesthesia will occur immediately allowing for instrumentation of the canal. The pressure effect has been shown to produce the anesthesia rather than the local anesthetic itself. VanGheluwe7 found that saline was as effective in providing anesthesia, when given intrapulpally, as 2 percent lidocaine (1:100,00 epinephrine). A disadvantage to this technique is that the patient will initially experience some pain during the injection. It may be possible to decrease this pain by placing a cotton pledget soaked in the local anesthetic solution on the exposure site prior to the intrapulpal injection.

Periodontal Ligament Injection

The periodontal ligament injection is useful in situations of incomplete anesthesia. This technique relies on intraosseous spread of the local anesthetic.8 A 27-gauge (or 30-gauge) short needle is placed between the periodontal ligament and the tooth to be anesthetized on either the mesial or distal aspect of the tooth. A small volume of anesthetic, 0.2 ml, is deposited under pressure. A separate injection is required for each root of a multirooted tooth. The onset of anesthesia is immediate and the duration is unpredictable. Patients may experience some sensitivity postoperatively in the area of the injection. Periodontal ligament damage and bone resorption does occur at the site of needle insertion but these changes are transient. Care should be taken when using a local anesthetic with epinephrine in patients who may be sensitive to the vasoconstrictor effects. Anesthetic solutions given intraosseously will enter the blood system rapidly.9

Success of the periodontal ligament injection is dependent on the anesthetic being delivered under pressure into the periodontal ligament space. There are specialized syringes available to give the dentist a mechanical advantage and a metered dose when injecting, but most dentists are capable of administering a periodontal ligament injection with the traditional syringe.

Intraosseous Injection

The intraosseous injection is not a new technique. The original technique required surgical exposure of the cortical bone and drilling a hole with a small round bur. A new technique has been developed in which an introducer device prepares a small hole in the cortical bone. A 27-gauge ultrashort needle is then placed in the perforator hole and local anesthetic is injected into the cancellous bone. There are two systems currently on the market: Stabident and Cyberdent. The Stabident system uses a perforator that fits on a contra-angle slow-speed handpiece. The injection needle is the same length and diameter as the perforator. Cyberdent is a unit in which the perforator and needle are mounted on a specially designed slow-speed handpiece.

The first step in the intraosseous injection is to locate the perforation site. The perforation site is typically distal to the tooth of interest at an equal distance from the adjacent tooth; 2 mm below a line connecting the gingival margins of the teeth. Once the perforation site is located it is necessary to deposit a small amount of local anesthetic for soft tissue anesthesia, if the patient is not already anesthetized. Perforation may be approached in one of two ways. One approach is to hold the introducer tip perpendicular to the injection

site and penetrate the soft tissues and buccal plate in a series of short bursts. Cancellous bone is reached when there is a sudden loss of resistance. The other approach is to angle the perforator so that the needle is directed apically. A 35-degree angle is used for all maxillary injections. In an anterior mandible a very acute angle is used (10 degrees). This angle will gradually increase for the posterior mandible (60 degrees for the molars). The soft tissues and buccal plate are penetrated until the collar of the perforator touches the gingiva. Once perforation is complete the perforator is removed and the needle is introduced into the hole, following the same path of entry. Single tooth anesthesia may be achieved through the slow deposition of a third of a cartridge of local anesthetic. A full cartridge of local anesthetic will provide anesthesia for multiple teeth. It is not recommended to use this injection near the mental foramen, near the midline, nor close to the maxillary sinus if sinus penetration is likely. It is also not recommended for use when the adjacent teeth are very close together and introduction of the perforator may penetrate the periodontal ligament.

Many patients will experience an increase in heart rate with the intraosseous injection of epinephrine-containing local anesthetics. This effect lasts about 2-3 minutes and is seen in approximately 60 percent-80 percent of individuals receiving the injection. 10-13 Patients should be informed of this effect to decrease their anxiety, should it happen. If the patient is sensitive to epinephrine or cardiovascularly compromised it may be a better choice to use 3 percent mepivacaine plain for the intraosseous injection. 3 percent mepivacaine plain has been shown to be effective when used as a supplemental intraosseous injection in teeth with irreversible pulpitis.14

The intraosseous injection may be used as a primary anesthetic technique.11,15 Replogle11 evaluated, in a clinical study, the efficacy of an intraosseous injection of either 2 percent lidocaine (1:1000,000 epinephrine) or 3 percent mepivacaine plain in mandibular first molars. Anesthetic success was achieved in 74 percent of the first molars injected with the lidocaine solution and only 45 percent of the first molars injected with the mepivacaine solution. The duration of anesthesia decreased over 60 minutes with the lidocaine solution having a longer duration of action.

The intraosseous injection has been very successful as a supplemental technique. Reisman14, in a clinical study, gave patients with a symptomatic mandibular molars an inferior alveolar nerve block with 2 percent lidocaine (1:100,000 epinephrine). Anesthesia was successfully achieved 25 percent of the time. By adding a supplemental intraosseous injection of 3 percent mepivacaine plain, there was an overall success rate of 80 percent. If an additional intraosseous injection was given (with 3 percent mepivacaine plain) the overall success rate was boosted to 98 percent.

Finally, no matter how successful different injection techniques are reported to be, if the patient is anxious they will be very difficult to anesthetize. Anxiety lowers the pain threshold. When this occurs the patient will experience non-painful stimuli as being painful. Decreasing patient anxiety is as important as the local anesthesia. Ways to manage patient anxiety may be through the use of nitrous oxide and oxygen inhalation sedation, oral sedation, or intravenous anesthesia.

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Vasoconstrictors and the Heart

Thomas J. Pallasch, DDS, MS

ABSTRACT The use of vasoconstrictors in local anesthetics, as topical hemostatic agents, and in gingival retraction cord, remains controversial although data exists from which to formulate reasonable guidelines. The value of such vasoconstrictors to increase local anesthetic efficacy and reduce systemic uptake is unquestioned. Elevated blood levels of epinephrine can occur with their use but do not generally appear to be associated with any significant cardiovascular effects in healthy patients or those with mild to moderate heart disease. Reduced dosages or local anesthetics without vasoconstrictors are indicated for patients with more significant disease and epinephrine-impregnated retraction cord should be used cautiously or avoided in certain situations. Endogenous epinephrine released in dental treatment-associated stress may also reach significant blood levels and make it difficult to determine causation of cardiovascular adverse events. The safely record of dental local anesthetics and their vasoconstrictors has been impressive and will remain so with continued judicious use of these agents.

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Anyone in dental practice long enough has probably heard the admonition from a physician: "Don't use adrenalin in this patient." Such advice is based upon certain misconceptions that:

- The vasoconstrictor content (dosage) of dental local anesthetics is comparable to those used for anaphylactic, asthmatic, or cardiac emergencies;
- The resulting loss of anesthetic efficacy is without clinical consequences (the adrenal release of catecholamines seen with inadequate anesthesia is without harm); and
- No guidelines have been established for the safe use of vasoconstrictors in dental local anesthetic solutions in medically compromised patients.

Consequently, the dental health professional is placed in the difficult position of either following inappropriate advice or disregarding it and using the his or her best clinical judgment.

Vasoconstrictors in Local Anesthetics

It is generally accepted that the good achieved by the inclusion of vasoconstrictors in dental local anesthetics greatly outweighs any potential deleterious effects of these agents. Their ability to retard anesthetic absorption, thereby both decreasing local anesthetic systemic toxicity and prolonging and increasing its activity at the site of deposition, is the rationale for their use. Additionally, local control

of bleeding can be very advantageous. That the vasoconstrictor can be absorbed into the systemic blood circulation and at times produce significant plasma levels is well-established, but there are conflicting opinions on the potential consequences. The use of racemic epinephrine gingival retraction cord is of concern and contention. The adrenal release of epinephrine and norepinephrine can also be a significant and sometimes overlooked factor in any adverse cardiovascular effects.

Local anesthetics and their vasoconstrictors are remarkably safe as employed in dentistry. From 1943 to 1952, local anesthetics were responsible for 0.43 percent of all anesthetic deaths in New York City,1 possibly three deaths in 700 million dental injections in Britain from 1970 to 1979 (70 million cartridges/year),2 and 1 death in 490,000 to 1.85 million injections in oral surgery and 1 in 36 million general practitioner-administered local anesthetics.3

Many efforts have been made to determine the potential or actual adverse effects associated with vasoconstrictors in such local anesthetics. Several studies have indicated that under certain conditions the blood levels of epinephrine may attain concentrations of four to 27 times their baseline (pre-injection) level,4-7 but the majority of studies indicate that these elevated levels of epinephrine associated with dental treatment usually result in only minor to moderate changes in cardiovascular parameters (heart rate, blood pressure, stroke volume).4-11

Electrocardiographic changes may also occur in both healthy and cardiovascularly impaired patients both during local anesthetic administration and the dental procedure itself.8,12,13 These may entail slight ST segment (myocardial ischemic) depression usually not considered clinically significant.8,12,13,14 Such electrocardiogram changes may occur with local anesthetics administered without vasoconstrictors15 and can be present more often before than after vasoconstrictor use.14 Forty patients having had a recent myocardial infarction (average 12 days previously) underwent dental anesthesia (one cartridge of 1:100,000 solution) with no significant changes in blood pressure, heart rate or the electrocardiogram.16 Levonordefrin appears to produce norepinephrine-like effects of increased peripheral resistance and mean pressure and reduced heart rate and cardiac output.3

Epinephrine -- through its combined alpha- and beta-adrenergic agonist effects -- usually increases heart rate, stroke volume, systolic blood pressure, myocardial oxygen consumption, and cardiac automaticity, but reduces diastolic blood pressure. There is a certain threshold of increased blood epinephrine necessary to induce these changes: heart rate (50-100 pg/ml), systolic blood pressure (75-150 pg/ml) and diastolic pressure (150-200 pg/ml)16 which translates to 1, 2, and 4 cartridges of lidocaine with 1:100,000 epinephrine respectively.3 This data has been derived from a study of only six healthy patients,17 a number that may be too small to account for the significant variation in response to epinephrine seen clinically particularly with higher doses.4-11 It is hypothesized that some patients may be "hypersensitive" to epinephrine, particularly those already highly anxious;3,18 but there is no data to support this contention. Normal dose-response variations should be expected as with any other drug.

The hemodynamic alterations seen with elevated plasma epinephrine are usually quite short in duration,3,6,19 probably because of the very short plasma half-life of epinephrine (usually less that one minute).20,21 Epinephrine is largely eliminated from the blood in 10 minutes20,21 or less2 due to its metabolism by catechol-O-methyl transferase in the blood, liver, lungs, and other tissues.22

Epinephrine in local anesthetics is often required to control local bleeding. Twice the blood loss in periodontal surgery occurs when concentrations of 1:100,000 are used as opposed to 1:50,000.23 Concentrations of 1:100,000 may usually prove to be adequate.24 Epinephrine-induced surgical vasoconstriction may lead to delayed wound healing and significant "rebound" postoperative bleeding.25,26

Three official statements have been formulated by various bodies regarding the use of vasoconstrictors in dental local anesthetics. In 1986 and again in 1991, the American Heart Association27 stated that "if vasoconstrictors are necessary, care should be taken to use the smallest effective dose. Vasoconstrictor agents should be used in local anesthetic solutions during dental practice only when it is clear that the procedure will be shortened or the analgesia rendered more profound. When a vasoconstrictor is indicated, extreme care should be taken to avoid intravascular injection." In 1964, the American Heart Association and the American Dental Association28 made this joint statement: "The typical concentration of vasoconstrictors contained in local anesthetics are not contraindicated with cardiovascular disease so long as preliminary aspiration is practiced, the agent is injected slowly, and the smallest effective dose is administered." In 1955, the New York Heart Association29 provided the following: "Under these conditions and with these precautions, the use of epinephrine with procaine for dental surgery presents no special hazards in persons with heart disease. We would recommend for any one session that there be used no more than 10.0 cc of 1:50,000 epinephrine -- no more than 0.2 mgm of epinephrine

in any form." The conditions of concern to the New York Heart Association were that the dentist should have information from the physician about the nature and severity of the heart disease in the patient and knowledge of medications the patient is receiving, particularly such medication as might increase the activity of epinephrine. These recommendations remain generally reasonable; and many dentists today are sophisticated enough to assess mild to moderate cardiovascular disease without physician consultation, although such may be indicated in more advanced forms of the disease.

TABLE 1.

American Society of Anesthesiologists Physical Status Classifications (as adapted from Malamed 24).

I: Normal healthy individual
II: Patient with mild to moderate systemic disease
III: Patient with severe systemic disease that limits activity but is not incapacitating
IV: Patient with severe systemic disease that limits activity and is a constant threat to life
V: Moribund patient not expected to survive 24 hours or without an operation

There are no absolute contraindications (never used under any circumstances) to the use of vasoconstrictors in dental local anesthetics as epinephrine is an endogenous neurotransmitter. However, severely ill patients (ASA Class IV or V) may be at too great a risk for elective dental care or require hospitalization for treatment (Table 1).24 It appears reasonable if possible to restrict the amount of epinephrine to 0.04 mg/appointment (Table 2) in patients with unstable (preinfarction, Crescendo) angina, daily anginal episodes, recent (in the past three to six months) myocardial infarction or coronary bypass, hyperthyroidism (thyrotoxicosis), uncontrolled cardiac arrhythmias and/or severe essential hypertension and congestive heart failure.3,18,24 Also, the total dose should be restricted, if possible, to 0.04 to 0.05 mg in patients taking tricyclic antidepressants or nonselective beta-adrenergic blocking drugs.3,24 It has been suggested30,31 that the above conditions are absolute contraindications to the use of vasoconstrictors without any advice as to how then to manage such patients. Local anesthetics without vasoconstrictors may not provide adequate anesthesia resulting in significant adrenal release of endogenous epinephrine, are without hemostatic efficacy, and could necessitate hospitalization for dental treatment with all the attendant risks. Local anesthetics with vasoconstrictors are generally safe in the presence of cardiovascular disease24,32 and the optimal concentration of the vasoconstrictor will depend on the type of anesthetic agent, the duration required, the site of injection and vascularity, and the requirements for local hemostasis.33 Local anesthetics without epinephrine (3 percent mepivacaine and 4 percent prilocaine) can prove advantageous in patients with relative vasoconstrictor contraindications.32

Three groups of drugs have been potentially implicated in drug interactions with local anesthetic vasoconstrictors: phenothiazine and other neuroleptic antipsychotics, antidepressants (tricyclics, monoamine oxidase inhibitors), and beta-adrenergic blocking drugs (Table 3). Two others (selective serotonin reuptake

TABLE 2.

Vasoconstrictor Concentrations and Maximum Dosages (as adapted from Malamed(24) and Felpel (31).

Epinephrine

Normal Healthy Adult			
Concentration	**Micrograms/Cartridge(2)**	**Maximum Dose**	**Number of Cartridges**
1:200,000	9	0.2 mg	22 (40 ml)3
1:100,000	18	0.2 mg	11 (20 ml)3
1:50,000	36	0.2 mg	5 (10 ml)
Significant Cardiovascular Disease (ASA III or IV)			
1:200,000	9	0.04 mg	4 (8 ml)
1:100,000	1	8	0.04 mg 2 (4 ml)
1:50,000	36	0.04 mg	1 (2 ml)

1. The dosage for levonordefrin is 90 micrograms/cartridge, 1.0 mg maximum dose or 11 cartridges maximum in the healthy patient, which exceeds the maximum allowable local anesthetic dose.

2. Each cartridge contains 1.8 ml.
3. Exceeds the maximum allowable local anesthetic dose.
4. Measurement Conversions:
1 milligram (mg) =0.001 gram = 10(-3) gram
1 microgram (ug) = 0.000001 gram (0.001 milligram) = 10(-6)
1 nanogram (ng) = 0.000000001 gram (0.001 microgram) = 10(-9) gram
1 picogram (pg) = 0.000000000001 gram (0.001 nanogram) = 10(-12) gram

inhibitors, cocaine) also merit discussion.

Since the phenothiazines and related antipsychotics possess alpha-adrenergic blocking activity, the potential exists for a combined alpha and beta agonist like epinephrine to interact with the unopposed beta receptor resulting in hypotension and reflex tachycardia. It does not appear that such an interaction has ever occurred clinically in dentistry.34,35

The antidepressant monoamine oxidase inhibitors primarily affect monoamine oxidase A that regulates the norepinephrine available for neuronal release in the sympathetic nervous system. Epinephrine is only a substrate for monoamine oxidase A after it is metabolized by catechol-O-methyl transferase. It does not release norepinephrine (as do indirect and mixed-acting adrenergics) and monoamine oxidase inhibitor-induced adrenergic activity may result in down regulation of the postjunctional adrenergic receptor. For these reasons, epinephrine may be safely employed in patients taking the monoamine oxidase inhibitors.34,36

It may be prudent in patients taking the tricyclic antidepressants to restrict the epinephrine dose to 0.05 mg or 5.4 ml of a 1:100,000 solution,34 although such an interaction may only occur at large doses if at all.37 There may not be a recorded clinical case of such an interaction.38 Currently, there are no drug interactions between epinephrine and the selective serotonin reuptake inhibitors;39,40 and there is no data regarding the miscellaneous antidepressants in Table 3.

Nonselective beta-adrenergic blocking drugs inhibit both the beta1 (cardiac) and beta2 (peripheral) adrenergic receptors, while the cardioselective beta-blockers inhibit only the beta1 receptors. Epinephrine administered to a patient taking a nonselective beta-blocker may result in hypertension and reflex bradycardia due to epinephrine interaction with the unopposed alpha-adrenergic receptor.41-43 In such patients, epinephrine is relatively contraindicated (reduced dosage); no reports have appeared of any such drug interaction in patients taking the cardioselective beta-blockers.41 Hypertension has been seen with the combination of propranolol and levonordefrin.44 There is probably no significant epinephrine drug interaction with the alpha/beta adrenergic blockers.

Sudden cardiac death associated with cocaine is well-documented.45,46 Some of these deaths occurred six hours or longer after acute cocaine ingestion, and it is possible that cocaine metabolites persisting for 48 hours after ingestion may have been responsible.47 Cocaine affects the cardiovascular system primarily by blocking the neuronal reuptake of norepinephrine and dopamine, by neuronal release of catecholamines, direct vasoconstriction, and local anesthetic effects.47 The cardiovascular effects of cocaine are complex and conflicting: adrenergic stimulation, increased heart rate, increased or decreased cardiac contractility, and slowed cardiac impulse conduction.47 Other effects include arrhythmias, myocarditis,

TABLE 3.

Antidepressants and Beta-Adrenergic Blocking Drugs

Antidepressants	Beta-Adrenergic Blocking Drugs
Tricyclic Antidepressants	fluvoxamine (Luvox)
amitriptyline (Elavil)	paroxetine (Paxil)
amoxapine (Asendin)	sertraline (Zoloft)
clomipramine (Norpramin)	***Cardioselective***
doxepin (Sinequan)	acetutolol (Sectral)
imipramine (Tofranil)	atenolol (Tenormin)
nortriptyline (Aventyl, Pamelor)	betaxolol (Kerlone)
protriptyline (Vivactil)	bisoprolol (Zebeta)
trimipramine (Surmontil)	esmolol (Brevibloc)
Miscellaneous Antidepressants	metoprolol (Lopressor)
buprion (Wellbutrin)	***Noncardioselective***
maprotiline (Ludiomil)	carteolol (Cartol)
mirtazapine (Remeron)	carvedilol (Coreg)
nefazodone (Serzone)	labetolol (Normodyne, Trandate)
trazodone (Desyrel)	nadolol (Corgard)
venlafaxine (Effexor)	penbutolol (Levatol)
Monoamine Oxidase Inhibitors	pindolol (Visken)
phenelzine (Nardil)	propranolol (Inderal)
tranylcypromine (Parnate)	sotalol (Betapace)
Selective Serotonin Reuptake Inhibitors	timolol (Blocarden)
fluoxetine (Prozac)	***Alpha/Beta Adrenergic Blocking***
	carvedilol (Coreg)
	labetolol (Normodyne, Trandate)

cardiomyopathy, cardiac contraction band necrosis, platelet agglutination, accelerated atherosclerosis, and sudden death due to myocardial infarction, arrhythmias or heart block.47 Acute electrocardiogram changes include prolonged PR, QRS and QT intervals along with a prolonged cardiac refractory period. Increased QRS voltage, ST elevation and ST-T changes may occur in up to 39 percent of chronic cocaine abusers.48

It has been suggested24 that any dental patient who has taken or is suspected of taking cocaine recently have dental treatment postponed for 24 hours. This would appear reasonable, and possibly 48 hours would be better considering cocaine metabolites. The detection of dental patients who are substance abusers may be very difficult.46 There are presently no studies or case reports regarding interactions between cocaine or its metabolites and local anesthetics or their vasoconstrictors, but it would appear that the potential exists.

Catecholamine Blood Levels in Stress

Current opinion3,18 appears to minimize the role of endogenous adrenal catecholamine (epinephrine and norepinephrine) secretion in stressful dental situations and maximize the potential for toxic blood levels of epinephrine concentrations in local anesthetics. Previous opinions2,19,49-51 held that conventional doses of epinephrine in local anesthetics were too small to significantly influence the cardiovascular system and that stress/fear-induced adrenal catecholamine secretion was responsible for "adrenaline" adverse reactions. It is probable that both scenarios can operate in any given situation with considerable overlap.

The resting rate of epinephrine secretion for the adrenal medulla is estimated to be 29 to 39 pg/ml for epinephrine,3,4,52 and for norepinephrine is 228 pg/ml.52 This "resting" epinephrine level can be higher (98 pg/ml) prior to dental treatment.10 In chronic stress (severe ICU illness) epinephrine blood levels may rise from 0.034 ng/ml to 0.14 ng/ml (four-fold increase) and in acute maximal stress (cardiac arrest) from 0.034 ng/ml to 0.36 to 35.9 ng/ml (10- to 1000-fold increase) with norepinephrine levels increased by a factor of two and 32 times respectively.52 This is not to equate dental treatment with such intense stress; but norepinephrine and epinephrine blood levels may increase 40 times under stress,19 and certainly dental treatment may be in this category. Merely placing a syringe in a patient's mouth without any tissue penetration may raise blood pressure and heart rate.53 In any given clinical situation, it may be impossible to determine the relative influences of exogenous vs. endogenous vasoconstrictors in the etiology of an adverse event.

Gingival Retraction Cord

The use of gingival retraction cord impregnated with manufacturer-labeled 8 percent racemic (dl) epinephrine is controversial because of conflicting clinical pharmacokinetics and toxicity studies. It has been estimated that each inch of such cord contains between 225.524,54 and 661 micrograms55 of dl-epinephrine (113-330 micrograms of the pharmacologically active l-form) equating with 3.13 to 9.16 cartridges of 1:100,000 anesthetic solution.

One study has demonstrated very significant increases in heart rate (6-120/minute) and blood pressure (0-140 mm/Hg systolic; 0-48 mm/Hg diastolic) in dogs from racemic cord,56 while others see either no or only slightly significant increases/changes in the measured parameters (electrocardiogram, blood pressure, heart rate).57-64 A reported rise in blood epinephrine from 15 pg/ml to 316 pg/ml in a single patient was associated with no hemodynamic changes.61 In another study approximately 64 percent to 94 percent (average 81 percent) of cord epinephrine was "lost" (about 71 micrograms/inch) allegedly due to vascular absorption, however no accounting was made for drug "lost" because of removal by gingival crevicular fluid, saliva or catechol-O-methyl transferase metabolism.54 Some studies61,63 demonstrate little effect of epinephrine placed in intact gingival sulcus (understandable since it is probably not absorbed through intact epithelium), while others indicate that the traumatized gingival sulcus can allow for much greater drug absorption.65 The presence or absence of gingivitis may also play a role, and the tissue manipulation itself may cause hemodynamic changes.64 Interestingly, some studies57,61 leave the cord in place for 30 to 120 minutes, the validity of which is obscure as it relates to clinical practice. Only one study applied 8 percent racemic epinephrine on a cotton pledget (at an unknown volume) in dogs, which resulted in greater elevations in heart rate and blood pressure compared to racemic cord but with some animals exhibiting little or no hemodynamic changes;56 while another employed 8 percent racemic solution to intact gingiva and a gingival laceration with no blood pressure changes, only a slight effect when applied to gingivectomy wounds and very significant blood pressure elevation when applied to an apicoectomy wound.55

It is apparent that the concentration of epinephrine in gingival retraction cord or solution has the potential to induce significant cardiovascular effects, but the consequences in any given patient can be highly variable depending upon the:

- Actual epinephrine concentration in

the cord or solution;
- The length of time the cord (solution) is left in the sulcus;
- The amount of tissue trauma/gingivitis present in the sulcus;
- The number of teeth ligated (area of tissue exposed);
- Dilution/removal by crevicular fluid/saliva;
- Metabolism by catechol-O-methyl transferase;
- Localized vasoconstriction by epinephrine to retard its own absorption;
- Operator trauma to the area in placing the cord; and
- Individual patient threshold to any elevated blood levels of vasoconstrictor.

It is probably best to refrain from epinephrine-impregnated cord in patients known to be at risk from epinephrine as listed above and to use the minimum amount whenever possible.

Conclusions

The record of dentistry in the safe and judicious use of local anesthetics and their associated vasoconstrictors is impeccable. Even though clinical and laboratory studies indicate a potential for significant deleterious effects on the cardiovascular system by exogenous (or endogenous) epinephrine, the lack of clinically significant documentation of these adverse effects in actual patients is noteworthy. Yet complacency could alter these impressive statistics if care is not taken to remember that the proper dose of a drug is "enough"66: the amount that produces a suitable therapeutic benefit with the least attendant toxicity.

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Localized Complications from Local Anesthesia

Daniel A. Haas, DDS, PhD

ABSTRACT The inherent safety of local anesthetics allows practitioners to use them frequently with the confidence that adverse events are rare. Nevertheless, complications can occur. The aim of this article is to briefly review the localized adverse events that may result from local anesthetic administration. Descriptions of each complication will be followed by suggestions for prevention and management. In spite of a dentist's conscientious practice, many of these complications cannot always be prevented.

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The local anesthetics used in dentistry are considered very safe and have a low incidence of adverse reactions associated with their administration. These adverse events may be classified as either systemic or localized. Systemic complications can occur as a result of psychogenic reactions induced by anxiety, toxicity secondary to high levels of the drug in blood, or allergy. Localized complications may manifest in a number of ways, and the focus of this article is to briefly review them. It is important to note that complications are not always preventable, and their occurrence does not necessarily imply poor technique by the dentist. The reality is that with an estimated 300 million injections being administered yearly in the United States alone,1 even those events that may be considered rare will be experienced by many patients. The following is a summary of local complications.

Prolonged Anesthesia or Paresthesia

Complete anesthesia or an altered sensation in the lip or tongue may persist beyond the expected duration of action of a local anesthetic. Commonly referred to as a paresthesia, these neuropathies may manifest as a total loss of sensation (anesthesia), a burning or tingling, pain to non-noxious stimuli (dysesthesia), or increased pain to noxious stimuli (hyperesthesia).2,3 Prolonged anesthesia or paresthesia in the tongue or lip is known to occur following surgical procedures such as extractions,4,5 and it is assumed that the cause is direct trauma to either the lingual or inferior alveolar nerve. However, persistent anesthesia or paresthesia can also occur following nonsurgical dentistry. Most these are transient and resolve within eight weeks, but they may become irreversible. Whereas the former are an annoyance for the patient, the latter are much more distressing.

There are several putative causes of postinjection paresthesia. Hemorrhage into the nerve sheath may lead to an intraneural hematoma, which then causes pressure on nerve fibers, impairing normal conduction. The hematoma and associated edema usually resorb within several weeks, and symptoms subsequently resolve. If scar formation results, there may be permanent loss of sensation. Direct trauma by the needle may also lead to similar damage. In addition, administration of local anesthetic from a cartridge contaminated by alcohol or sterilizing solution may induce paresthesia.6 Finally, neurotoxicity may be a factor, since a review of the literature suggests that local anesthetics have this potential.7-12

How often do paresthesias occur in nonsurgical dentistry? A recent study led to an estimated incidence of 1 irreversible paresthesia out of every 785,000 injections.13 It has been stated in a legal case in Canada that this low frequency would not warrant advising every dental patient of this risk prior to each injection.14 This same study did note that specific drugs were more likely to be associated with paresthesia. Two drugs, articaine (which is available in Canada and parts of Europe under the trade name Ultracaine, among others) and prilocaine (Citanest), were more likely to be associated with paresthesia compared with the other anesthetics, and this was statistically significant when compared to the distribution of use.13 These same two drugs were again found to be significantly more likely to be associated with paresthesia in 1994.15

The reasons for this relationship to the type of anesthetic are speculative only. Differences in metabolism of these drugs would not be relevant since it occurs in organs away from the site of the neuropathy. Their only common feature is that they are the only injectable local anesthetics in dentistry that have a concentration of 4 percent, whereas the others are lower. It may be conjectured that toxicity may manifest simply because of the higher concentration of these drugs, as opposed to any unique characteristic. Needle trauma to the nerve combined with deposition of a large quantity of drug may be more likely to induce residual nerve damage. Supporting a role of drug concentration are reports of neurologic deficits in animal studies using 4 percent lidocaine16 and in human studies of spinal anesthetics with 5 percent lidocaine.10,11,17 This should be contrasted with the rare reports of neuropathy with 2 percent lidocaine (Xylocaine, among others), which is used in dentistry.

Prevention

There is no guaranteed method to prevent paresthesia or prolonged anesthesia. The inferior alveolar nerve block requires the practitioner to advance the needle near the inferior alveolar and lingual nerves. Practitioners attempt to place the needle near these nerves without intentionally striking them, yet this can occur and may be perceived as an "electric shock" sensation by the patient. Interestingly, this sensation does not imply that paresthesia will result.13 Directly contacting these nerves is not an indication of improper technique, it is simply a risk of carrying out intraoral injections.

Prevention of prolonged anesthesia or paresthesia:

- • If the patient feels "electric shock," move needle away from this site prior to injecting.
- Do not store cartridges of local anesthetic in disinfecting solutions.

Most paresthesias are transient and resolve within eight weeks. This is fortunate as there is no definitive means of improving the patient's symptoms. The dentist must show concern and reassure the patient that these events can occur and usually resolve over time. The dentist should note the signs and symptoms and maintain contact with the patient. A change in the character of the symptoms can be an encouraging sign that there may be resolution of the neuropathy. It may indicate that there is healing of the nerve, and with time the patient may regain normal sensation. The patient who has had no change in symptoms over a prolonged period, such as several months, is less likely to have a satisfactory outcome. Restoring sensation by microsurgery may be considered by those with training in this area. It has been stated that microsurgery is most likely to be successful if the patient is evaluated within the first month2 or the first three months.5 There is no guaranteed method of treating prolonged anesthesia or paresthesia.

Management of prolonged anesthesia or paresthesia:

- Reassure the patient that the condition is usually temporary although, rarely, it can remain indefinitely.
- Note signs and symptoms and follow up within one month.
- If symptoms persist for more than two months, refer to an oral and maxillofacial surgeon with experience in this field.

Trismus

Limited jaw opening, or trismus, is a relatively common complication following local anesthetic administration. It can be caused by spasm of the muscles of mastication, which in turn may be a result of needle insertion into or through one of them. The most common muscle to be the source of trismus is the medial pterygoid, which can be penetrated during an inferior alveolar nerve block using any of the three main techniques: the conventional approach, the Vazirani-Akinosi (closed-mouth) technique, or the Gow-Gates. Rarely, the temporalis may be penetrated before it attaches onto the coronoid process if the needle is inserted too far laterally. Even more rarely, the lateral pterygoid muscle may be penetrated if a block is administered too far superiorly. Bleeding into the muscle following injection may also cause muscle spasm and trismus. Furthermore, injection of local anesthetic directly into muscle may cause a mild myotoxic response that can

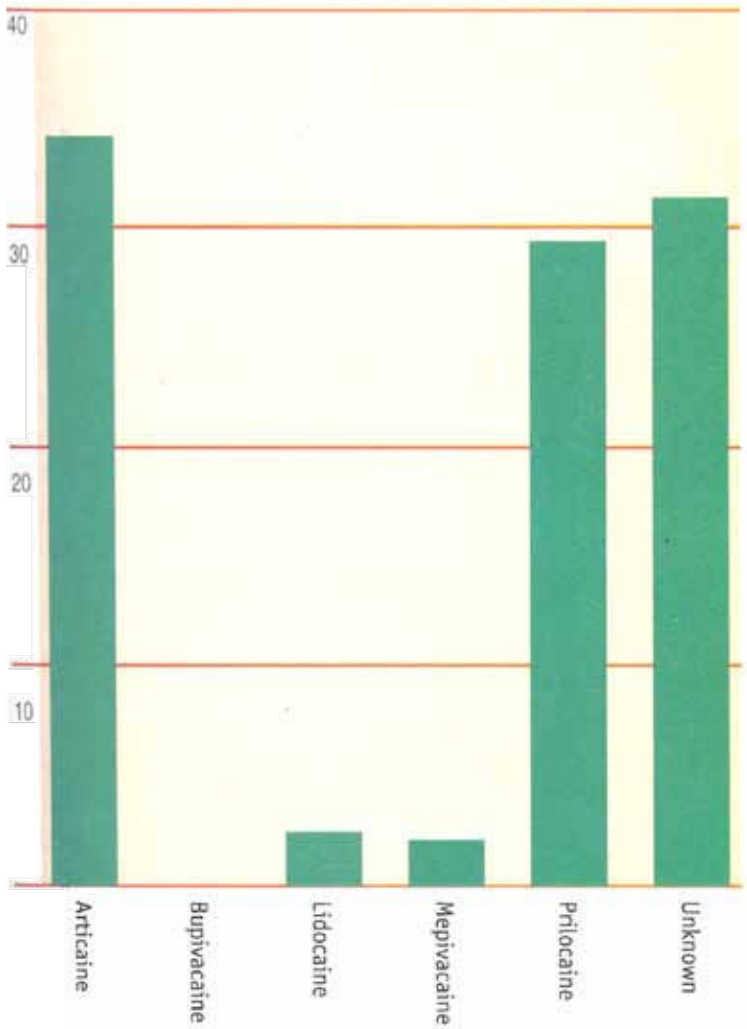

Figure 1. This figure shows the frequency distribution, based on type of local anesthetic, of reported irreversible paresthesias of prolonged anesthesias in Ontario, Canada. The increased frequencies of paresthesia with articaine a prilocaine were statistically significant.[13]

lead to necrosis.18 In the rare situation of an infection from the injection, trismus may also develop.

The main symptom of trismus is the limitation of movement of the mandible, which is often associated with pain. Symptoms will arise from one to six days following an injection. The duration of symptoms and their severity are both variable. Following management, as described below, improvement should be noted within two to three days. If there is no improvement within this time, the dentist should consider other possible causes, such as infection, and treat accordingly.

Prevention of trismus:

- Follow basic principles of atraumatic injection technique.19

Management of trismus:

- Apply hot, moist towels to the site for approximately 20 minutes every hour.
- Use analgesics as required.
- The patient should gradually open and close mouth as a means of physiotherapy.

Hematoma

A hematoma is a localized mass of extravasated blood that may become clinically noticeable following an injection. In this context, it can occur following the inadvertent nicking of a blood vessel during the penetration or withdrawal of the needle. When carrying out intraoral injections, practitioners often pierce blood vessels; but only when there is sufficient blood leaking out can a hematoma be seen. The vessels most commonly associated with hematomas are the pterygoid plexus of veins, the posterior superior alveolar vessels, the inferior alveolar vessels, and the mental vessels. The patient will notice development of swelling and the discoloration of a bruise lasting seven to 14 days. It is important to note that a hematoma will form independently of aspiration results. A negative aspiration does not guarantee an absence of a hematoma, as the needle may nick a blood vessel either on the way in or upon withdrawal. Aspiration results merely report the contents at the needle tip at the time of aspirating. Similarly, a positive aspiration does not imply that a hematoma will result, since the needle may simply have entered a vein at the time of aspiration, and the amount of blood leaking out from this vessel penetration may be clinically unnoticeable.

Prevention of hematoma:

- Follow basic principles of atraumatic injection technique.19
- Minimize the number of needle penetrations into tissue.
- Use a short needle for the posterior superior alveolar nerve block.

Management of hematoma:

- If visible immediately following the injection, apply direct pressure, if possible.
- Once bleeding has stopped, discharge the patient with instructions to:
  - Apply ice intermittently to the site for the first six hours.
  - Do not apply heat for at least six hours.
  - Use analgesics as required.
  - Expect discoloration.
  - If difficulty in opening occurs, treat as with trismus, described above.

CDA JOURNAL, VOL 26, Nº9

Pain on Injection

Occasionally, injection of local anesthetic can be accompanied by pain or a burning sensation. Passing the needle through a sensitive structure such as muscle or tendon may cause pain. It may occur during injection if the solution is administered too quickly and therefore distends the tissue rapidly. Local anesthetic solutions that are too cold or too warm may also cause discomfort. Solutions that are more acidic, namely those with vasoconstrictor, may cause a short-lasting burning sensation. Cartridges stored in a disinfecting solution such as alcohol may have residual amounts of solution on the end of the cartridge that can then be administered inadvertently during injection.

Prevention of pain:

- Inject slowly: Take at least one minute to administer one cartridge.
- Store cartridges at room temperature.
- Do not store cartridges of local anesthetic in disinfecting solutions.

Management of pain:

- Pain or burning on injection is usually self-limiting because it is treated by the onset of anesthesia.

Needle Breakage

This event is very rare. Sudden, unexpected movement of the patient is the primary cause.20,21 It is believed that smaller-diameter needles, i.e., 30 gauge, are more likely to break than larger-diameter, i.e., 25 gauge. Needle breakage usually occurs at the hub, which is the reason for never inserting a needle completely into tissue. Although bending a needle may be considered for injection techniques such as the Vazirani-Akinosi or the maxillary nerve block,22-24 some advise against this practice.25 If it is done, it is important to do so only once because repeated bending will weaken the connection at the hub and predispose the needle to breakage.

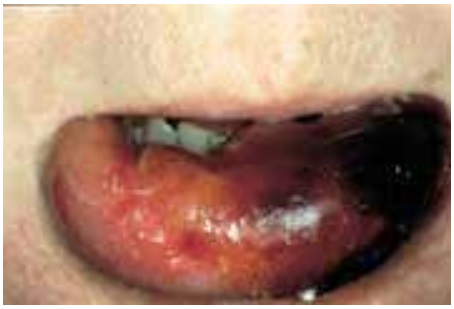

Figure 2. Photograph of a lip bitten by a patient with lymphoma. Picture is courtesy of Professor J.H.P. Main, head of oral pathology, University of Toronto.

Prevention of needle breakage:

- Do not insert a needle into tissues up to its hub; always leave a portion exposed.
- Use long needles if a depth of more than 18 mm is required.
- Use larger-diameter needles (25 gauge is ideal) for the deeper blocks, such as the three mandibular block techniques (conventional, Gow-Gates, and Vazirani-Akinosi) and the maxillary nerve block.
- Do not apply excessive force on the needle once it is inserted in tissue.
- If redirecting a needle is required, withdraw it almost completely before doing so.
- Do not bend a needle more than once.
- Management of needle breakage:
- Remain calm.
- Ask the patient to remain still; keep their mouth open by not removing your hand.
- If a portion of the needle is visible, remove it with a hemostat or similar instrument.
  - If the needle is not visible:
  - Inform the patient.
  - Record the events in the chart.
  - Refer the patient to an oral and maxillofacial surgeon.
  - Surgical removal should only be attempted by someone experienced with surgery of the involved region and after radiographs have been taken to help localize the needle.26

Soft Tissue Injury

Figure 3. An example of the appearance of a patient with a transient facial nerve paralysis. Picture is courtesy of Dr. M. Pavone, Toronto.

With the loss of sensation that accompanies a successful block, a patient can easily bite into his or her lip or tongue. Swelling and pain will result following the offset of anesthesia. This event is most common in children or patients who are mentally challenged or demented, such as those with Alzheimer's disease. The child's parent or guardian, or the caregiver with the mentally challenged patient or those with dementia, should be advised to carefully observe the patient for the expected duration of anesthesia. Nevertheless, soft tissue injury may also be a concern for mentally normal patients who are at risk of an exaggerated response to trauma.

Figure 2. Photograph of a lip bitten by a patient with lymphoma. Picture is courtesy of Professor J.H.P. Main, head of oral pathology, University of Toronto.

Figure 2 shows the consequence of a patient with lymphoma who had bitten his lip. Therefore, patients with bleeding abnormalities should also be warned of the risks of lip and tongue biting.

Prevention of soft tissue injury:

- For pediatric, mentally challenged, or demented patients, use a local anesthetic of appropriate duration.
- Warn the parent, guardian, or caregiver to watch the patient carefully for the duration of soft-tissue anesthesia to prevent biting of tissue.
- In children, consider placing a cotton roll between the mucobuccal fold for the duration of anesthesia.
- Explain the risks of soft tissue injury to

patients with bleeding abnormalities.
- Management of soft tissue injury:
- Use analgesics as required.
- Use rinses or applications with lukewarm dilute solutions of salt or baking soda.
- Consider applying petroleum jelly over lip lesion.

Facial Nerve Paralysis

Anesthesia of the facial nerve may occur if the needle has penetrated the parotid gland capsule and local anesthetic is then administered within. This nerve, the seventh cranial nerve, is contained within the parotid gland and provides motor function through its five branches -- the temporalis, zygomatic, buccal, mandibular and cervical. Needle placement into the parotid may occur if there is overinsertion during an inferior alveolar nerve block or the Vazirani-Akinosi block. The result of anesthesia of these branches of this nerve includes a transient unilateral paralysis of the muscles of the chin, lower lip, upper lip, cheek, and eye. There will be a loss of tone in the muscles of facial expression. In the past, the term Bell's palsy was commonly used to refer to all paralyses of the facial nerve, but it is now restricted to those induced virally.27

Facial nerve paralysis secondary to local anesthetic injection is temporary and will last the expected duration of anesthesia of soft tissue for the particular anesthetic administered. There are risks if there is a loss of the protective reflex to close the eyelid. An example of the appearance of a patient with a transient facial nerve paralysis is shown in Figure 3.

Unwanted anesthesia of other nerves may also occur. Ocular complications following temporary paralysis of cranial nerves III, IV, or VI,28,29 as well as the optic nerve,30 have been described. The proposed mechanism for these events is intravenous transport of local anesthetic to the cavernous sinus.31 Careful aspiration to avoid intravenous injection should prevent this complication.

Prevention of facial nerve paralysis:
- Follow basic principles of atraumatic injection technique.19
- Avoid overinsertion of the needle.
- For the conventional inferior alveolar nerve block, do not inject unless bone has been contacted at the appropriate depth.

Management of facial nerve paralysis:
- Reassure the patient of the transient nature of the event.
- Advise the patient to use an eye patch until motor function returns.
- If contact lenses are worn, they should be removed.
- Record details in the patient's chart.

Infection

With the introduction many years ago of sterile disposable needles, infection is now an extremely rare complication of local anesthetic administration. It may occur if the needle has been contaminated prior to insertion. The normal flora of the oral cavity is not a concern since they do not lead to infection in patients who are not significantly immunocompromised. In fact, bacteria enter the tissues with every needle insertion, yet the body's normal defense prevents a clinical infection. In patients who are severely immunocompromised, a topical antiseptic or an antiseptic rinse such as chlorhexidine could be considered prior to needle insertion.

If an infection does occur, it will likely manifest initially as pain and trismus one day postinjection. If these symptoms persist for three days and continue to worsen, the possibility of infection should be considered. At this stage, this patient should be examined for other signs of infection, such as swelling, lymphadenopathy, and fever.

When there is an active site of infection, such as an abscess, needles should not be inserted. This is not only because the low pH will prevent the onset of local anesthetic action, but also because there is the potential for spreading the infection.

Prevention of infection:
- Use sterile disposable needles.
- Do not contaminate the needle by contacting nonsterile surfaces outside the mouth.
- In severely immunocompromised patients, consider a topical antiseptic prior to injection.

Management of infection:
- Prescribe antibiotics, such as penicillin, in an appropriate dose and duration.
- Record details in the patient's chart and follow up to determine progress.

Mucosal Lesions

Occasionally, the intraoral mucosa may show signs of sloughing or ulceration. The epithelial layer may desquamate from prolonged application of topical anesthetic. It is possible, but not common, that necrosis of tissues may result from high concentrations of vasoconstrictor, such as 1:50,000. Sites of ulceration consistent with a diagnosis of aphthous stomatitis may also result following local anesthetic administration. For each of these, the lesions will be present for one to two weeks and resolve irrespective of treatment. Drug therapy is seldom warranted. Simple measures such as saline or sodium bicarbonate rinses may assist healing by keeping the sites relatively clean.

Prevention of mucosal lesions:
- Do not leave topical anesthetic on mucosa for prolonged periods.
- Management of mucosal lesions:
- Reassure the patient; advise him or her of the expected duration of one to two weeks.
- Use rinses with lukewarm dilute solutions of salt or baking soda, until symptoms resolve.

Summary

The occurrence of localized complications from local anesthesia administration can be minimized by following basic principles of local anesthetic injection

technique and the previously listed suggestions for prevention. Nevertheless, in spite of proper technique, complications may occur. Fortunately, most resolve well without permanent sequelae, and appropriate management will facilitate patient recovery from these events.

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Toxicity and Allergy to Local Anesthesia

Andrew H. Chen, DDS

ABSTRACT Considering the amount of local anesthetic administered on a daily basis, dental professionals must be familiar with the factors that influence the dose and type of local anesthetic that induces a toxic or allergic reaction. In addition to the route and rate of administration, the patient's physical condition and health may also influence the dose of local anesthetic that could be safely administered. This article reviews the different causes of local anesthesia toxicity and allergy. With prevention and early recognition of the warning signs, poor prognosis can be avoided.

AUTHOR

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The first widely used local anesthetic was cocaine. In the mid-1860s, Bennett, a physician, studied the effects and uses of cocaine for surgical procedures. He reported that with increased dosage of cocaine, seizures and respiratory arrest typically followed glossal numbness.1 Despite the reports of toxicity, cocaine provided good anesthesia for a variety of surgical procedures. Hall reported the first use of cocaine by a dental surgeon; Nash operated painlessly on his own upper incisors after an injection of cocaine into the infraorbital nerve. Thus was cocaine introduced as a local anesthetic in dentistry.2,3

Cocaine had certain drawbacks. Its propensity to cause addiction and acute systemic toxicity made it less desirable than was originally thought. Hall himself became addicted to cocaine. Because of this tremendous addiction factor and systemic toxicity, a search for a synthetic substitute was undertaken. In 1905, procaine, an ester local anesthetic, was developed. In subsequent years, other ester-type local anesthetics were used in dentistry and anesthesia. In 1948, Lofgren reported the successful use of lidocaine as a local anesthetic.4 Lidocaine represented a new type of local anesthetic, which differed chemically from the earlier ester-type drugs in several ways. Subsequently introduced anesthetics -- such as mepivacaine, prilocaine, bupivacaine, etidocaine and articaine -- are classified as amide local anesthetics.

As is the case in the development of any new class of medications, the early use of cocaine inspired the development of a safer class of local anesthetics. Though local anesthetics are invariably toxic at higher doses when administered accidentally intravenously, they are devoid of the addiction

problems associated with cocaine.

Typical toxic responses occur in the central nervous and cardiovascular systems, with the most frequent clinical toxic reactions involving the central nervous system. Early toxic effects include alterations in consciousness and seizures, followed by respiratory arrest, myocardial depression and eventually death. In 1970, Lofstrom reported numerous fatal cases involving overdose of local anesthetics. Few cases however, involved small doses administered during dental treatment.5 In 1979, Albright demonstrated a correlation between cardiovascular system toxicity and the longer-acting, highly lipid-soluble local anesthetics (bupivacaine, etidocaine). Of 49 fatal cases, 43 percent involved bupivacaine.6 Subsequently, the Food and Drug Administration recommended against the use of 0.75 percent bupivacaine. The FDA further recommended the slower and more accurate delivery of bupivacaine. Since then, the fatality rate due to cardiovascular collapse secondary to bupivacaine administration has significantly decreased.

Local Anesthesia Toxicity

Local anesthetic toxicity is the result of increased blood levels of a local anesthetic in a very short duration. This can occur with accidental intravenous injection during a regional nerve block, excessive doses, and/or incorrect anatomical locations. The rate of administration, pre-existing medical conditions, acid-base imbalances, age, weight, and other physical and medical factors can also influence toxicity of local anesthetics. Lipid solubility, pKa, rate and route of metabolism change the level of toxicity.7

The high blood concentration of anesthetic necessary to cause toxic overdose comes about in four ways:8

- Too large a dose of local anesthetic drug;
- Unusually rapid absorption of the drug or intravascular injection;
- Unusually slow biotransformation; and
- Slow elimination.

The dose necessary to induce toxicity varies among patients and is influenced by numerous factors:8

- The patient's general health;
- Rapidity of injection;
- Route of administration;
- Amount of local anesthetic administered; and
- Patient's age.

Chemical Structure

The chemical structure of the local anesthetics may also affect the drug toxicity. The right and left stereoisomers of bupivacaine differ in cardiotoxicity, d-bupivacaine being more cardiotoxic than l-bupivacaine.9 The ester local anesthetic is hydrolyzed rapidly by cholinesterases in the blood. Therefore, the toxicity associated with these local anesthetics is typically of shorter duration. The metabolism of the amides in the liver lacks the immediacy of the esters.10 Since local anesthetics do not have active metabolites, there is little risk of complications from hydrolyzed or biotransformed local anesthetics.

Lipid Solubility

The amount of local anesthetic required to produce toxicity tends to parallel the lipid solubility and the potency of the particular drug (Table 1). Because of the lipid bilayer in the cell membranes of nerves and tissues, the more lipid-soluble anesthetics diffuse intracellularly more easily and are more potent. Central nervous and cardiovascular system toxicity are directly correlated to the potency of the local anesthetic -- more potent local anesthetics produce greater toxicity at lower doses than do less potent agents. Furthermore, studies show that the negative inotropic and convulsant effects of procaine are less than those of bupivacaine, procaine being the less potent of the two anesthetics.14,15 Therefore, local anesthetics with low lipid solubility (prilocaine, lidocaine, mepivacaine) are less toxic than anesthetics with high lipid solubility (bupivacaine, etidocaine).13

TABLE 1.
The Amount of Local Anesthetic Required to Produce toxicity as Compared to Lipid Solubility and Potency.

Agent	Potency	Lipid Solubility[7]	Absolute Toxicity[16]
Esters			
Procaine	1	1.7	1
Chloroprocaine	1.5	N/A	0.5
Tetracaine	10	76	10
Amides			
Lidocaine 2	43	2	
Prilocaine 2	25	1+	
Mepivacaine	2	21	1.5
Etidocaine	4	800	4
Bupivacaine	4	346	4

The correlation between toxicity and the pKa of a local anesthetic is minimal. However, since the pKa of an anesthetic is directly related to the onset of anesthesia, the onset of toxicity is more rapid in rapid-acting drugs. The closer the pKa of an anesthetic to the pH of the injected tissues, the more rapid the onset of action. Because of the greater number of uncharged anesthetic molecules, more anesthetic is able to diffuse into the nerve sheath and surrounding tissues.

Protein Binding

Protein binding is not a significant a factor in local anesthetic toxicity. Increased protein binding of a local anesthetic allows for tighter and longer binding of anesthetic molecules to drug receptor sites in the nerve, providing for a longer duration of action and potentially increasing the risk of toxicity when additional doses are administered.

Other predisposing factors influencing local anesthetic toxicity are patient-related. They include the patient's age, weight, gender, genetics, and pre-existing disease or medications.

Age

Age is a factor in younger and older patients. The lack of development or dysfunction of the liver and kidneys in these populations increases the level of local anesthetic in the bloodstream.14 However, studies have demonstrated the opposite with immaturity of the other organs. Budgwell and collegaues suggested that immaturity of the central nervous and cardiovascular systems in the younger population might play a role in the resistance to local anesthetic toxicity.15 McIlvaine and colleagues suggested that children have a higher threshold for bupivacaine toxicity than adults.16

Weight

The guideline for maximum dose of a local anesthetic is usually based upon the patient's body weight. Lean body weight is important in determining blood volume of the patient. The greater the body weight, the larger the volume of distribution of the local anesthetic. With a greater volume of distribution, a larger dose of local anesthetic could be administered prior to inducing toxicity. The greater the lean body weight, the more tolerant a person could be to a higher dose of a local anesthetic.14

Pregnancy

Local anesthetic toxicity has been studied extensively in pregnant animals, but not much is known for pregnant women. Any information regarding pregnancy and local anesthetic toxicity is extrapolated from animal studies. From studies with pregnant animals, pregnancy does not seem to alter the level of systemic toxicity with lidocaine and mepivacaine. However, bupivacaine is more toxic in pregnant women than lidocaine, prilocaine, and mepivacaine.17.18

Physical changes occurring in pregnancy may also affect the amount of local anesthetic necessary to cause toxicity. When a woman is pregnant, several changes may occur: a decrease in renal function, increase in volume of blood, and increase in weight. In a person with less than normal renal function, local anesthetic metabolites may accumulate in the bloodstream. However, with the increase in body weight and blood volume, the patient would be able to tolerate a larger volume of local anesthetic.14

Pseudocholinesterase Deficiency

Serum cholinesterase is an enzyme produced by the liver. It hydrolyzes ester anesthetics (procaine, chloroprocaine, tetracaine) and the depolarizing muscle relaxant succinylcholine. A hereditary pseudocholinesterase deficiency retards the hydrolysis of ester local anesthetics. The result is a prolonged half-life of the ester anesthetics. Low serum cholinesterase levels are capable of doubling or tripling the half-life of a local anesthetic. Lack of pseudocholinesterase or atypical pseudocholinesterase could have devastating consequences when large doses of ester local anesthetics are administered. Other causes of low pseudocholinesterase levels include pregnancy, liver disease, and certain drugs (i.e. Echothiophate, Phenelzine, and Trimethaphan).14

Pre-existing Disease

Pre-existing hepatic dysfunction decreases the rate at which amide local anesthetics are metabolized, leading to the elevation of blood levels of local anesthetics circulating to target organs (heart, brain). Renal insufficiency leads to elevated electrolyte levels and an increase in the blood concentration of local anesthetics. This results in hyperkalemia and acidosis, both of which may increase the risk of morbidity and mortality associated with bupivacine administration.19 Cardiac insufficiency reduces blood flow, causing stasis and elevated levels of local anesthetic in end organs, increasing anesthetic risk.

Medications

Besides the drugs that alter pseudocholinesterase levels, drugs that alter functioning of the central nervous and cardiovascular systems may also lower the toxicity threshold of local anesthetics. This is especially so for drugs that decrease liver or cardiac function or stimulate the central nervous system.

Local anesthetics, such as lidocaine and procainamide, are administered in advanced cardiac life support as antiarrhythmics. Patients who are receiving antiarrhythmics and cardiovascular depressants have inhibited myocardial impulse propagation. Therefore, concomitant use of high doses of local anesthetics along with their cardiac medications

may have additive effects on the heart.20 These medications may include digoxin, beta-blockers, and calcium channel blockers. Another concern is patients who are on H1-blockers since these drugs compete for the same enzyme system that metabolizes amide anesthetics. This would result in a delayed metabolism of the local anesthetics in the liver.

Central nervous system depressants may indirectly be detrimental when used to prevent local anesthetic induced seizures. Diazepam and phenobarbital are occasionally used to prevent seizures in the epileptic population. However, clinical practice has abandoned the prophylactic use of these drugs in the prevention of local anesthetic-induced seizures.

As positive as these intentions are, premedication with benzodiazepines may, in fact, lower the incidence of local anesthetic-induced seizures, but it does not alter the threshold for cardiovascular system toxicity.21 In fact, suppression of the initial central nervous system warning signs of overdosage may lead to an increased risk of severe cardiovascular collapse.

The onset of toxicity is the direct result of the rate and site of drug administration. Direct intravenous injection of local anesthetics would induce the most rapid and intense level of central nervous and cardiovascular system toxicity. Highly vascular injection sites, i.e., sublingual regions, have a higher correlation to an increased incidence of local anesthetic toxicity than do less

vascular areas.14 Therefore, multiple injections into highly vascularized areas should proceed with extreme caution.

de Jong and colleagues showed that mixtures of local anesthetics are no more toxic than the parent anesthetic.22 To be safe, alternative injection sites and local anesthetics should be considered. Scott studied the threshold dose for central nervous system toxicity of etidocaine in healthy volunteers. When etidocaine was administered slowly via an intravenous infusion at 10 mg/min and 20 mg/min, the faster rate caused threshold central nervous system symptoms at two-thirds the dose of the slower rate.23 Malamed recommends injection of 36 mg of lidocaine consistently over 60 seconds. Intravenous injections of the same dose in less than 15 seconds significantly increase the risk of overdose reactions.24 It is recommended, therefore, that aspiration and slow injection techniques be utilized for all injections.

The central nervous system is the initial target organ for local anesthesia toxicity. The effects of local anesthetics on the central nervous system are biphasic (stimulation/depression). Although most clinical signs of central nervous system actions related to local anesthetic toxicity are stimulatory, the actual physiologic cause is depressive. The stimulatory effect is the indirect result of a depression of cerebral inhibitory centers. This results in unopposed facilitatory neurons causing random stimulatory firing of the neurons in the brain. This generalized stimulation of the brain causes generalized tonic-clonic seizures. In subtoxic blood levels, 0.05 to 4 mcg/ml of serum blood, lidocaine may produce anticonvulsant and sedative effects.7,8,25,26 At 4 to 7 mcg/ml, mild central nervous system stimulatory signs are seen (Table 2) with lidocaine. The mild signs represent depression of higher cerebral and cortical centers. Cognitive and reasoning centers are depressed. Perioral numbness, which occurs, is not entirely a direct effect of paresthesia; rather this represents the effect of local anesthetic blood levels in the highly vascularized tissue of the oral cavity. As lidocaine blood levels approach 7 mcg/ml, the basal centers and then the medullary centers of the brain are depressed.8 Lidocaine and procaine may produce progressive symptoms (Table 2) without the initial mild signs. If blood levels of lidocaine reach 7.5 to 10 mcg/ml, generalized tonic-clonic seizures occur.19 The seizure is typically of short duration and self-limiting. However, respiratory arrest is common because of the lack of muscle control, which accompanies the seizure. Progression to hypoxia, cyanosis, and cardiac arrest may be rapid because of the detrimental combination of increased oxygen consumption of the tonic muscles and respiratory arrest. Postictal cortical depression may vary from stupor to coma. Respiratory and/or cardiac depression may deteriorate to respiratory and/or cardiac arrest during the postictal medullary depression.

Toxic Effects on the Central Nervous System

TABLE 2.
Signs and Symptoms of Central Nervous System Stimulation

Mild Signs
Talkativeness
Slurred speech
Apprehension
Localized muscle twitching
Lightheadedness and dizziness
Tinnitus
Difficulty focusing the eyes
Disorientation
Circumoral numbness or numbness of the tongue
Progressive Symptoms
Lethargy
Unresponsiveness
Lack of movement of the extremities
Drowsiness and sedated
Lack of muscular tone
Mild drop in blood pressure, heart rate, and respiratory rate

Toxic Effects on the Cardiovascular System

The cardiovascular system is more resistant to elevated local anesthetic blood levels. Similar to the anticonvulsant effects on the central nervous system, subtoxic doses of local anesthetics have demonstrated cardiodepressive actions comparable to quinidine.27 Local anesthetics produce a direct depression of the myocardium, slow conduction of impulses through the AV node, and prolong the refractory period. Lidocaine and procainamide have been used as antiarrhythmics for three decades.28,29 The minimum initial antiarrythmic dose of lidocaine recommended by the American Heart Association is 1 to 1.5 mg/kg, which produces a blood level of 1.5 to 5 mcg/ml in the blood of an average 150 pound adult. At blood levels above 5 mcg/ml, moderate to severe myocardial depression is noted. Bradycardia, negative inotropic actions, and peripheral vasodilation occur progressively until a blood level of 10 mcg/ml. At levels above 10 mcg/ml, severe vasodilatation and bradycardia leads to ventricular fibrillation and asystole.30 Asystole secondary to local anesthetics is virtually irreversible.8 Numerous cases of local anesthetic-induced cardiovascular collapse have been

reported in humans, with a majority of these findings occurring with the potent, highly lipid-soluble, highly protein-bound local anesthetics.31,32,33 Bupivacaine, etidocaine, and tetracaine produced more depressant effects at lower concentrations than did lidocaine, mepivacaine, or prilocaine.27,34,35 Furthermore, bupivacaine is more arrhythmogenic than lidocaine. Bupivacaine blocks the SA node; prolongs the P-R interval and induces more re-entrant type of arrhythmias. All this is due to the fact that bupivacaine is more strongly bound to the receptor site within the sodium channel than is lidocaine, especially in cardiac muscle.36 Liu and colleagues studied the relative cardiovascular toxicity of numerous local anesthetics on dogs and found lethality to be in direct correlation with the relative potencies of the anesthetics. He demonstrated that bupivacaine produced the greatest degree of hypotension and cardiovascular collapse with the lowest lethal dose, followed by tetracaine and etidocaine.13,37 Lidocaine was least likely to induce negative inotropic and chronotropic effects.12,34

Unlike inotropic effects, the exact mechanism of the negative chronotropic action is unknown. It is speculated, however, that the decrease in myocardial contractility may be due to sodium channel blockade or to increased calcium release. De La Coussaye and colleagues reported that the greater negative inotropic effect of bupivacaine is related to the amount of the drug entering into the myocardium.38 The lower lipid solubility of lidocaine prevents a higher intracellular concentration of lidocaine and thereby lessens its negative inotropic potential.

Management of Toxicity

Prevention is the key to local anesthesia toxicity. To prevent intravascular injections, use an aspirating syringe with a slow injection technique. Malamed recommends using needles not smaller than 25 gauge, aspirating in at least two planes before injection, and slowly injecting the anesthetic. Needles smaller than 25 gauge are difficult to aspirate through, especially when located in fibrous tissues. Reorientation of the bevel of the needle ensures that the needle is not resting against the vascular wall, thus providing a better determination of non-intravascular injections. Many reported local anesthetic toxicity cases are associated with a rapid injection of local anesthetics. Injecting 1.8 ml of solution over 60 seconds should prevent a rapid rise intravascularly and minimize toxicity.14

Other techniques to prevent toxicity are paying careful attention to dose and route of administration, using test doses, and using vasoconstrictors. All three techniques should prevent toxicity when injecting into highly vascular areas.7

Vasoconstrictors, such as epinephrine, serve two purposes. As blood vessels are constricted, local anesthetics are absorbed intravascularly more slowly. Second, as a sympathomimetic drug, epinephrine may serve as a marker for intravascular injection. Intravascular epinephrine will typically elicit an acute tachycardic response. Furthermore, during initial cardiovascular system toxicity, the positive chronotropic and inotropic effects of epinephrine will counteract the myocardial depression of the local anesthetic.

Initial signs of toxicity may be difficult to differentiate from anxiety. The patient may appear more verbose and apprehensive. The best treatment is to reassure the patient because, at this stage, the situation is mild and not life-threatening. Planned dental treatment can usually be completed at the same appointment.

If an episode of tonic-clonic seizures arises within two to five minutes after the injection, suspect rapid absorption of the anesthetic from the interstitial tissues. This is more common with injections into the base of the tongue. However, if seizures are seen within 10 to 30 seconds, the local anesthetic was inadvertently administered intravascularly.

Treatment for both instances involves termination of dentistry and immediate airway management. Protect the patient from injuries by moving any loose items away from him or her. Suction should be readily available to remove vomitus, especially during the postictal phase. Patency of the airway should be of primary concern after tonic-clonic seizures. Mechanical ventilation and head tilt/chin lift of the patient may be necessary when seizures stop. Supplemental oxygen and oral airways are also advisable.7

During a severe postictal phase of depression, the patient may be unconscious, unresponsive, and incapable of maintaining a patent airway. The ABCs of resuscitation should be followed. Respiratory arrest is common; therefore, controlled ventilation is required. Cardiac responses may vary from moderate hypotension to asystole. A supine or Trendelenburg position is recommended, producing the least peripheral pooling and increasing vascular circulation. Pulse rate and blood pressure should be closely monitored to prevent complete collapse.

Each patient's response is distinct, thus sensible clinical judgment is recommended to determine the feasibility of continuing with dental treatment. Be attentive to the patient's deteriorating condition. Due to the pre-existing local anesthetic dose present in the cardiovascular system, subsequent local anesthetic injections should proceed with caution.

Central Nervous System Management

Due to the rapid onset and duration of central nervous system toxicity, administration of intravenous anticonvulsant drugs is not expeditious enough to terminate this self-limiting condition, although as a prophylactic measure, barbiturates and benzodiazepines have been administered to prevent seizures.31,37 Typically the administration of 50 to 100 mg of pentobarbital or 5 to 10 mg of diazepam at the earliest sign of toxicity will prevent the development of seizures. These central nervous system depressants decrease the incidence of seizures but do not alter the dose of bupivacaine necessary to cause cardiovascular system collapse.21 In fact, these central nervous system depressants may obscure the primary central nervous system warning signs, which develop prior to cardiovascular system collapse.

Barbiturates and benzodiazepines do not interrupt the neuronal activity during seizures, they merely prevent the tonic-clonic movements exhibited during an episode. However, respiratory function may be obtunded by these drugs; therefore, controlled ventilation may be necessary to prevent the development of hypoxia and hypercarbia.8 This is of extreme importance during the postictal depression, where these drugs could intensify the magnitude of central nervous system and respiratory depression. The primary reason for administration of these central nervous system depressants is to control overt seizure activity and facilitate ventilation if a seizure occurs.7

Cardiovascular System Management

The importance of airway and respiration management supercedes any cardiovascular system support. As in basic life-support, airway is the primary step prior to assessing circulatory function. Thus, before considering the use of any drug therapy for the cardiovascular system, supplemental oxygen and a patent airway must be established. Moore reported successful control of local anesthetic-induced seizures in 84 of 93 patients with oxygen via bag and mask.39

Cardiovascular response to local anesthesia toxicity is extremely complicated. Clinical manifestations vary from simple hypotension to electromechanical dissociation, ventricular fibrillation, or asystole.

One of the easiest treatments does not involve the use of pharmacology. Repositioning the patient in a supine position with their feet elevated slightly and administering intravenous fluid could overcome the hypotension caused by venous pooling. If further treatment is necessary, vasopressors could be considered. Phenylephrine or ephedrine should be considered prior to epinephrine. Epinephrine has been shown to induce arrhythmias and seizures at lower doses of bupivacaine.40 Because of its immediate and direct cardiac effects, epinephrine sensitizes the heart to arrhythmias, whereas phenylephrine or ephedrine, to a lesser degree, has relatively little or no direct effect on the myocardium or AV node.40

Bradycardia is also commonly seen with local anesthesia toxicity. A heart rate lower than 40 beats per minute in an average patient indicates the need for pharmacologic intervention. Again, rather than use epinephrine, alternatives such as glycopyrrolate or atropine should first be considered. These anticholinergics increase heart rate indirectly via vagal blockade. Both of these medications should be administered intravenously. Atropine 0.5 mg or glycopyrrolate 0.2 mg should prove effective against bradycardia.8

Treatment of arrhythmias induced by local anesthetic overdose is difficult, since local anesthetics are also antiarrhythmics. Bretylium tosylate, calcium chloride, and magnesium sulfate have been effective for resuscitation.41,42 Ultimately, however, cardioversion may be necessary. With the increasing availability of automatic external defibrillators, the mandate may be to have them in all dental offices.

Allergy to Local Anesthesia

True immunoglobin-E mediated allergic reactions to local anesthetics are rare. Many patients and clinicians mistake any idiosyncratic response after local anesthetic injection for an allergic reaction. Giovanitti and Bennett estimated that no more than 1 percent of the adverse reactions to local anesthesia is true allergy.43 However, once reactive to an antigen, the patient is allergic to this drug for the rest of his or her life.

The immunoglobin-E allergic reaction is acquired through exposure to an antigen. With re-exposure, the antigen-antibody response is heightened until a point where mast cells respond with the release of chemical mediators that produce the clinical manifestations of allergy. These mediators include histamines, leukotrienes, chemotactic substances, lysozomal enzymes, prostaglandins, kinins, and platelet-activating factor.44,45 These mediators cause capillaries to leak and permit extravasation of plasma into the surrounding area. True drug allergies manifest as asthma, rhinitis, angioneurotic edema, urticaria, and rash. Urticaria is caused by release of histamine, which induces peripheral capillary leakage along with erythema, pruritis, and edema. Immunoglobin-E anaphylaxis may be severe enough to cause respiratory distress and cardiovascular collapse. Anaphylaxis is the result of a generalized increase in capillary permeability leading to a drop in blood pressure. Furthermore, released leukotrienes cause bronchiolar smooth muscle to spasm,

eliciting an asthmatic-type response.

No matter how doubtful their claim, treat all patients as "allergic to local anesthetics" until the patient is allergy-tested.

Allergy Signs and Symptoms

Mild signs of an allergic reaction include urticaria and rash. Urticaria is associated with pruritis (itching) and wheals (elevated skin patch). These mild dermatological signs are usually visible within six minutes. As the allergic reaction progresses, the cardiovascular, respiratory and gastrointestinal systems become involved. Hypotension is the initial cardiovascular response. Increased histamine release during allergy causes increased plasma extravasation to the interstitial tissues leading to a decrease in blood pressure and to generalized angioneurotic edema. Angioedema typically involves the face, hands, feet, and genitalia. During severe cases, the lips, tongue, larynx, and pharynx are also involved. Angioedema of the upper tracheobronchial tree (laryngeal edema) induces stridor by limiting air exchange to and from the lungs. Spasm of bronchial smooth muscle in the lower tracheobronchial tree causes bronchospasm. Bronchospasm and asthmatic-type reactions are the result of leukotrienes. Leukotrienes, similar to histamines, are chemical mediators of allergy that cause increased swelling and spasm of the tracheobronchial tree. Other symptoms of bronchospasm may include dyspnea, wheezing, flushing, cyanosis, tachycardia, and increased use of accessory muscles of respiration.

Predisposing Factors to Allergy

Many patients claim allergies to Novocain. Ester-type local anesthetics, i.e., procaine, chloroprocaine, and tetracaine, are derived from para-amino benzoic acid, a known allergen. Furthermore, when ester local anesthetics are hydrolyzed by plasma cholinesterase, its metabolites include para-amino benzoic acid. Amide local anesthetics are almost entirely devoid of this problem. After years of countless carpules of amide local anesthetics being administered, only a few cases have been reported with an amide local anesthetic challenge.46

Older commercial preparations of local anesthetics included preservatives, such as methylparaben. Methylparaben is chemically related to para-amino benzoic acid and is also identified as an allergen.47 Methylparaben is a bacteriostatic agent found in many drugs, cosmetics, and foods. Currently, methylparaben has been removed from dental local anesthetic cartridges but is still found in multiple dose vials. Another preservative used is sodium bisulfite or metabisulfite. Bisulfites are antioxidants used to prevent the early breakdown of epinephrine in dental cartridges. No allergic reactions to dental cartridges without epinephrine have been reported. Bisulfites are also found in food, preventing the food from "browning" (oxidizing) when exposed to air. Most patients who are intolerant to bisulfites are also dependent upon inhaled steroids to prevent acute episodes of bronchospasm.

Treatment of Allergic Reaction

Delayed mild cases of an allergic reaction are usually treated by 50 mg of intravenous, intramuscular or oral diphenhydramine (Benadryl). Follow-up doses of 50 mg oral diphenhydramine every four hours is recommended for three days.

If the initial signs of anaphylaxis proceed to conjunctivitis, rhinitis, urticaria, pruritis, and erythema within 60 minutes, 50 mg or IM diphenhydramine (25 mg for a child) and /or 0.3 mg of intramuscular epinephrine (0.15 mg for a child) is recommended.8 Corticosteroids, such as dexamethasone or methylprednisone, are effective in decreasing edema and capillary permeability. Intravenous 8 mg of dexamethasone (4 mg for a child) should be considered if the condition appears to be eminent. When speed is not a factor, Medrol Dosepak is an oral corticosteroid of choice. Subsequently, Benadryl should be prescribed orally for three to four days as precaution.

Bronchospasm or allergic asthma is treatable if diagnosed early. The patient will typically complain of difficulty in breathing and expresses a desire to sit upright. Wheezing may be heard with air exchange. For an asthmatic response, aerosolized albuterol or Medihaler-Epi is considered the first line of treatment.14 Intramuscular antihistimines, like diphenhydramine 50 mg, may also be beneficial. If aerosol or antihistamine treatments are not effective or the patient is unconscious, 0.3 mg intramuscularly of epinephrine will activate the beta-2-agoist receptor sites causing bronchodilation. If these treatments are ineffective or the episode recurs, activate emergency medical services. Medical consultation and follow-up with a physician is recommended.

Although no known cases of anaphylaxis to an amide local anesthetic have occurred within 30 minutes, prevention and immediate intervention is important, especially with laryngeal edema. Laryngeal edema is the one of the most ominous events following an initial allergic reaction. Immediate attention should be paid to evaluation of the patency of the airway. Complete obstruction of the larynx results in no sound and air passage. If a high-pitched crowing sound is heard the airway is partially obstructed. Either obstruction requires the same treatment.8 Emergency medical services should be immediately activated. Administer 0.3mg of intramuscular epinephrine and maintain a patent airway with bag,

mask, and supplemental oxygen. Intravenous steroids and histamine-blockers are also recommended. If the patient does not improve, consider opening an air passage below the obstruction. Typically this involves cricothyrotomy.8,14

Fortunately, anaphylaxis to an amide local anesthetic in a dental office is nearly non-existent. However, generalized anaphylaxis is one of the most urgent emergencies in the dental office. Most of the signs and symptoms have a rapid onset and significant morbidity. In most cases, the patient will become cyanotic and lose consciousness within minutes. All of the above symptoms with laryngeal edema, bronchospasm, and cardiovascular collapse may occur simultaneously. Unless treatment is immediate, the mortality rate is extremely high. Following airway maintenance, intravenous 0.3 mg epinephrine is the first line of treatment; followed by 8 mg dexamethasone. Assisted ventilation with supplemental oxygen is recommended. Management of the cardiovascular component of the reaction requires 15 mg of intravenous ephedrine to combat the severe hypotension, which is often present.8 Chest compressions may be necessary if the patient is pulseless. Activation of emergency medical services is important, however basic life support is the most important step in successful management of anaphylaxis. Prompt and definitive treatment may be the difference of life and death.

Allergy Testing

Due to the rarity of local anesthetic allergy, the simplest in vivo allergy testing technique is recommended. The probability of an anaphylactic response is minimal, especially with the intracutaneous testing technique. However, with any allergy testing, a specialist should complete the testing with an intravenous line started and emergency equipment and drugs at the ready.

Summary

Due to the morbidity and mortality involved with toxicity studies, most clinical studies involve animals. Few case presentations of toxicity and allergy in humans have been reported. The dosages necessary to produce toxicity and allergy vary between species; however, there is a direct correlation between animals and humans with respect to central nervous and cardiovascular system toxicity.

Local anesthetics are central nervous system depressants. At critical low blood levels, their depressant effects can be therapeutic in the prevention of certain types of seizures. At higher blood levels, the suppression of inhibitory pathways results in facilitatory pathways functioning unopposed, resulting in seizures. At very high blood levels, the facilitatory pathways also are blocked, resulting in complete suppression of the central nervous system. This condition is associated with coma and depression of respiratory and circulatory centers ultimately leading to death.

The cardiovascular system tends to be more resistant to the toxic effects. Local anesthetics have a suppressant effect on the heart, reducing myocardial contractile force and prolonging or blocking intracardiac conduction. They are also vasodilators. High doses of local anesthetics cause a reduction in heart rate and blood pressure, cardiac conduction defects, and arrhythmias, including ventricular tachycardia and fibrillation. There is, for the most part, a separation between the dose and blood concentration required to cause central nervous and cardiovascular system toxicity.

In general, the cardiotoxic and neurotoxic effects of local anesthetics do not differ greatly, but their relative potential for toxicity does. At certain dosages, bupivacaine produces effects different from those of other local anesthetics. There is considerable evidence to support the contention that bupivacaine exerts a strong direct effect on the myocardium and the brain.

The treatment of systemic toxicity due to local anesthetics should be instituted rapidly and aggressively. Appropriate equipment and pharmacological agents should be kept close at hand. Maintenance of an open airway and administration of oxygen is important. Support of the circulation and control of arrhythmias are essential for maintaining adequate perfusion of the vital organs as well as assisting in the removal of local anesthetic from the tissue and its detoxification. Persistence in the resuscitation process is essential, as some patients may prove difficult to resuscitate.

Allergic reactions to local anesthetics agents are extremely rare. Ester local anesthetics produce para-amino benzoic acid as a metabolite, and it is a known allergen. Methylparaben is also a known allergen, and it is used occasionally as a preservative in commercial preparations of some amide local anesthetics. Reactions are generally dermatologic when they occur and rarely are systemic or anaphylactoid. Recommendations for screening suspect patients can be found in the literature and generally involve skin tests.47,48

The intent of this article is to review the pharmacology of the local anesthetics and the mechanisms to toxicity and allergy. A good knowledge of the pharmacokinetics and pharmacodynamics of local anesthetics is important; both the patient and the practitioner should have a proper understanding of the consequences of local anesthesia administration. Early intervention can be started when all persons involved know the initial signs and symptoms of toxicity and allergy. Precaution is the best prevention, whether

it is overdose toxicity or allergy; and knowledge is the first key to prevention.

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Geezerhood

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It's good to reach a hale and hearty old age except for seeing your children become depressingly middle-aged. That's where I am now. In a few months, my son will officially enter Geezerhood. There's a pink area appearing on his scalp at the crown as happened to me and my father before me. As a rite of passage, it falls disappointingly short of a first kiss or being granted the keys to the car for the first time.

I asked him, "How does it feel to be almost 50?"

He replied, "How does it feel to have a 50-year-old child?"

We fell silent, each of us thinking regretfully of all the sins we hadn't committed.

I can remember that when I was in my 60s, if anyone called me a sexagenarian, it sounded like flattery. In the catalogue of aging, that period is called Geezer Plus and I reflect on it with mixed emotions. It was when I thought an old person was anybody 10 years older than I was. It was when I realized I was old enough to know my way around but had to concede that I wasn't going anywhere. I gave up going to any movie that didn't have a matinee. Forced to choose between two evils, I always took the one that got me home earlier. On the plus side, I was given a discount at IHOP without having to ask for it. I can go conveniently deaf when I want to, a feat that has stood me in good stead through the last two generations' "music."

Geezerhood is what used to be called the "Golden Years." That term has largely fallen out of favor, particularly with those of us actually enrolled in this period. Ask any Medicare person in a doctor's waiting room how he or she is enjoying the Golden Years if you want to get a cane whacked across your shins.

I am now comfortably in that period that lies beyond Geezer Plus; I am a Super Geezer, formally called an "old-timer." If there is any geriatric nomenclature above that, it falls into the field of paleontology, and I don't want to know about it.

Just over the horizon and coming at me like a tornado bearing down on Kansas is my eighth decade. I'm looking forward to it, surprised and happy to be here. My father used to claim 80 was the best of times, you could do or say anything. If there is an upside to old age, he said, this is it: Eccentricity is not only tolerated, it is expected. And he did his best to uphold the tradition. He claimed in a voice that could be heard clearly throughout the retirement home where he lived in his last years, "Old women are nuttier than old men and there are more of 'em." He often advised me, "What will be, will be, even if it never happens," but I could never work out just what that meant.

So that's definitely my plan. I'm going

for crotchety curmudgeon, maybe throw in a little weird -- I can do that. But first I've got to solve the problem of older men's pants. Something happens to most men sometime between Geezer Plus and Super Geezer. It's a guy thing, and I'm tired of my wife pointing this phenomenon out to me on a daily basis like maybe it's my fault.

What happens is, one night or maybe over a single weekend, a man's belly expands like he was in his ninth month of gestation. At the same time, his rear end diminishes in the same proportion. Cruelly referred to as a "beer belly" and a "cracker bottom," even though the victim may never have consumed either commodity in his lifetime, this anatomical metamorphosis results in a major trouser problem.

He buys a pair of pants that seem to fit reasonably well in the little fitting room with the flimsy curtain that never quite covers the door opening. He adjusts them to what he thinks is his waist, trying to recall from memory just where that is. The definitive landmarks appear to have vanished. The cuffs break nicely over his shoes and he's out of the cubicle before some other guy parts the curtain to reveal him in his underwear.

Like water seeking its highest level, pants on a geezer seek their lowest within 15 minutes of donning them. That is, the belt drops down under the belly. It has no choice. It's a size 36 trying to cope with a size 44 abdomen. Viewed in profile, the belt has assumed a 45 degree angle to the floor, the pant legs are now 4 inches too long, the crotch is just above his knees, and there is enough room in the seat to accommodate a couple of watermelons.

This is the Geezer Look, and pants manufacturers seem at a loss to address the problem. In warmer climes, we geezers have sought to resolve at least part of the error by wearing shorts. This has brought us up against comedic tradition that requires us to wear black socks and dress shoes. And a hat. Geezers are great for hats -- baseball caps, fedoras, Panamas, Greek fisherman caps -- it doesn't matter, as long as it's inappropriate for the occasion.

It is this mean-spirited media portrayal, when coupled with that of the lady-geezer stereotype featuring the all-purpose muu-muu that looks as if came with a center pole and stakes as matching accessories, that tarnishes the luster of the Golden Years.

So what can I tell my son? He doesn't get all misty-eyed when he hears "Sunrise, Sunset" from Fiddler on the Roof. Barbra Streisand doesn't appeal to him with "The Way We Were." Even Doris Day fails to get through with "Sentimental Journey." Perhaps when his descendants begin to outnumber his friends like mine do, he will understand that axiom of Geezerhood: "It's not how old you are, but how you are old." He may even figure out what to do about the belt.

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I am now comfortably in that period that lies beyond Geezer Plus; I am a Super Geezer.