

THE INS AND OUTS OF ANAESTHESIA

STANLEY MALAMED

A discussion with Dr Stanley Malamed

In your view, what are the important advances in dental anaesthesia?

Stanley Malamed: In the past several years there have been a number of ‘innovations’ in the area of pain control in dentistry. These include computer-controlled local anaesthetic delivery systems (CCLADS); intraosseous anaesthesia; the introduction, as a result of CCLADS, of several new techniques – the anterior middle superior alveolar (AMSA) nerve block and the palatal approach anterior superior alveolar (P-ASA) nerve block and the introduction of articaine HCl.

CCLADS, such as The Wand and the Comfort Control Syringe System, allow the (almost) pain-free delivery of LA anywhere in the oral cavity, including the palate. Designed to be more psychologically pleasing to the patient, CCLADS deliver the anesthetic solution at a constant rate, regardless of the density of the tissues into which they are deposited. Their use is most notable in any palatal injection and the periodontal ligament injection (PDL).

Intraosseous anaesthesia (IO) was ‘rediscovered’ about 10 years ago when the Stabident was introduced. Subsequently the X-Tip made the technique even more simple to successfully complete. IO anaesthesia has been adopted by most endodontists who have found it to be highly effective when treating the ‘hot’ mandibular molar: the most difficult teeth to anaesthetise. Success rates of over 90% have been achieved in clinical trials, compared to success rates of around 28% for traditional nerve blocks (inferior alveolar nerve block) in this situation.

The AMSA and the P-ASA nerve blocks arose as a result of research with CCLADS. In the AMSA nb deposition of 2/3 of a cartridge of LA into a site midway between the midline of the palate and the papilla between the two maxillary premolars provides pulpal anaesthesia of the five anterior maxillary teeth, their supporting buccal and palatal soft tissues and bone. There is no extraoral anaesthesia of the patient’s face, a finding that is universally lauded by patients. The AMSA has found great popularity among periodontists, dental hygienists and general practitioners. The P-ASA is similar to the AMSA, except that by depositing LA into the incisive foramen (located beneath the incisive papilla just palatal to the maxillary

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central incisors) pulpal anaesthesia is achieved of the incisors and canines bilaterally, using approximately three quarters of a cartridge of LA.

Probably the most important advance in dentistry in the UK and USA has been the clinical introduction of articaine HCl (Septanest (UK), Septocaine (USA)). Introduced in Germany in 1973 and Canada in 1987, articaine has become the more used LA in those countries (in Germany more than 90% of LA use in dentistry is articaine). Categorised as an amide-type LA, like lidocaine, mepivacaine, prilocaine and bupivacaine, articaine also possesses properties of the older ester-type LAs such as procaine. Though no published clinical trial has ever demonstrated any superiority of articaine to other LAs, practising dentists worldwide find the drug has desirable clinical properties that appear to make the drug more efficacious than other commonly used LAs. These anecdotal findings include:

- ‘Works faster’
- ‘Works better’,
- ‘More profound’,
- ‘Don’t miss as often’
- ‘Works when and where other LAs don’t’
- ‘Gets the palate numb when I infiltrate on the buccal’
- ‘Gets mandibular molars numb following buccal infiltration’.

Many, if not most of these statements are unsubstantiated scientifically, however wherever I speak throughout the world I continue to hear these same statements from doctors over and over again. Also heard is the question from these same doctors: ‘Should I use articaine for a mandibular block? I’ve heard that there is an increased risk of paraesthesia with 4% drugs.’ As with the above positive statements there is absolutely no scientific proof that articaine, or prilocaine (the other 4% LA) are associated with increased risks of paraesthesia. All reports have been strictly anecdotal. In the USA, articaine has become the second most popular LA in dentistry in only four years.

Normal and maximum dose of lignocaine: what is the difference?

The ‘normal’ dose of LA for any given injection is the minimum volume which would provide the ‘expected’ duration of pulpal (hard tissue) anaesthesia. For example, using lignocaine with epinephrine one expects to achieve approximately 60 minutes of pulpal anaesthesia following injection of one third of a LA cartridge. Administration of a larger volume does not increase either the depth or the duration of the observed anaesthesia.

The ‘maximum’ recommended dose of a LA is determined

from the patient's body weight. For example in the case of lignocaine the MRD (maximum recommended dose) is based upon 4.4 mg/kg, not to exceed a total dose of 300 mg in a 90-minute period. This number is based upon patient safety. The greater the number of milligrams of LA administered in a brief period of time, the higher the resulting LA blood level will be. Toxic (overdose) reactions develop when this blood level is too high. Exceeding the MRD does not mean that a problem will result. Exceeding this dose simply increases the likelihood of an overdose developing. Though an important factor in all patients, determination of the MRD and not exceeding it becomes more critically important when the patient is a younger, lighter weight child.

So the 'normal' dose is that volume of LA administered to achieve the desired clinical action of the drug, while the maximum dose is based more on safety and the avoidance of overdose.

When calculating the correct dose of local anaesthetic, should practitioners take into account the amount of topical anaesthetic injected, and if so, why?

Topical anaesthetic is an essential component in the delivery of painless injections. When properly administered, only a small quantity of topical anaesthetic should be applied to the soft tissues at the site of needle penetration. If applied in this manner then the dosage of topical anaesthetic will not be a factor in determining MRD (see above).

In those rare situations in which a doctor liberally applies large quantities of topical anaesthetic over a wide area of the mouth, the blood level of topical anaesthetic may become significantly elevated and then, if injectable LA is administered, an overly high blood level may ensue, producing an overdose reaction.

As mentioned previously, this is an exceedingly rare event in dentistry.

Do you recommend dentists use lignocaine with adrenaline solution, or is an adrenaline-free formulation your preference?

As a basic rule, I strongly recommend the inclusion of epinephrine in the LA solution as a matter of routine. That is, unless there is a compelling reason against its use. These reasons include:

- A brief appointment combined with minimal cutting on the tooth
- A patient whose cardiovascular system is so severely damaged that even sitting in the dental chair might prove to be overly dangerous.

The addition of epinephrine to the LA does several things of importance. It provides a longer duration, greater depth of anaesthesia and it retards absorption of the LA into the cardiovascular system, thereby helping to minimise the risk of an overdose.

Epinephrine should always be included in the LA solution unless there is a compelling reason for using

a plain drug.

How would you suggest dentists control the important area of post-operative pain control?

Post-surgical pain control has been managed effectively by USA dentists through the combined use of two classes of drug: NSAIDs (non-steroidal anti-inflammatory drugs) and LA.

One hour prior to the planned surgical procedure, the patient takes an oral dosage of a NSAID (e.g. ibuprofen). The 'usual' (LA preferred by doctor) local anaesthetic is administered for pain control during the surgery. Depending upon the duration of the surgical procedure, a long-acting LA such as bupivacaine is injected into the same site, either immediately following the initial LA (when the surgical procedure is brief) or at the very conclusion of the surgery (in a longer procedure). Bupivacaine will provide the patient with up to 12 hours of anaesthesia following the surgery, thus 'covering' the period in the first three to five hours when post-surgical pain is usually most intense. However, since it is impossible to guarantee that the patient will remain 'numb' for 12 hours (people vary in their response to drugs, sometimes quite significantly), the doctor will prescribe oral NSAIDs on a 'timed' basis, for example every six hours or every 12 hours, depending on the drug chosen for use. In this manner, when the feeling of numbness dissipates and 'feeling' returns, the patient will have a therapeutic blood level of the NSAID, thus precluding pain from developing.

This regimen has been in use in the USA for more than 10 years and has proven to be highly effective and gives great patient satisfaction.

Can you suggest a simple guideline that will help practitioners find the right local anaesthetic for his or her patients and what factors are most important when considering the right product for each individual?

The dental practitioner should have available in their surgery a variety of LAs. Selection of an appropriate LA for use in a given patient at a particular appointment will be based primarily upon the patient's need for pain control at that visit. LAs may be categorised by their expected duration of pulpal anaesthesia. As such, there are three categories: short-duration, intermediate-duration and long-duration. Mepivacaine (plain) is an example of a short-duration LA, providing pulpal anaesthesia ranging from 20-40 minutes and soft tissue anaesthesia from two to three hours. Intermediate-duration LAs include lignocaine + epinephrine, prilocaine + epinephrine or felypressin; mepivacaine + epinephrine or levonordefrin and articaine with epinephrine. All of these products will provide the typical patient with approximately one hour of pulpal and three to five hours of soft tissue anaesthesia. Bupivacaine + epinephrine is a long-duration LA, providing pulpal anaesthesia from 90 to 180 minutes and soft tissue anaesthesia from five to 12 hours. The dental practitioner should have available in the surgery one drug from each of those categories that are appropriate for the practice.

Once the LA has been selected for use in the patient, the doctor must determine if there are any contraindications to that drug's administration. Fortunately for the drugs available in dentistry, few such contraindications exist.

Please discuss prolonged anaesthesia following local anaesthesia administration

Paraesthesia, defined as a prolonged anaesthesia, is a potential complication whenever a needle is inserted into tissue and a drug deposited near a nerve. All LAs can, and have, produced paraesthesia. Case reports of lidocaine, mepivacaine, prilocaine and articaine have appeared in the dental literature. Of late, however, it appears that an increasing number of case reports implicate articaine as a cause of paraesthesia.

As a professor of anaesthesia at a US university, I have been intimately involved with the review of cases involving LA-associated paraesthesia. My feelings about this subject are, hopefully, unbiased. I mention this because I try very hard to evaluate each and every case that is presented to me to seek to determine which one or combination of the following might be responsible: (1) the needle, (2) haemorrhage, (3) edema, (4) surgical trauma, or (5) the LA drug.

Most reports of paraesthesia involve the mandible, specifically the lingual nerve, either as a loss of sensation or on occasion the loss of taste (the chorda tympani nerve). Frequently the paraesthesia also involves the inferior alveolar (IA) nerve, with associated paraesthesia of the chin, lip and buccal soft tissues in the anterior aspect of the mandible.

Where an IA nerve block was administered and the paraesthesia involves only the lingual nerve, it is difficult to fault the LA drug (why wouldn't the IA nerve also be involved?). Many of these cases also involve reports of the patient experiencing a 'lightning bolt-like' feeling as the needle was advanced through their tissues.

When LA is deposited against the lingual aspect of the mandibular, ramus fibres of the IA nerve are blocked. When a paraesthesia develops in this situation and involves branches of the IA nerve, there is a greater likelihood that the LA drug may be responsible, although the needle, haemorrhage and edema may still be possibilities.

When a paraesthesia resolves over days to about two weeks, the likelihood is that it is a result of either edema or haemorrhage. More prolonged paraesthesia might be secondary to either needle trauma or neurotoxicity of the LA drug.

The drug: anecdotal reports seem to indicate that there is a greater incidence in the chance of a paraesthesia with more highly concentrated LAs (4% ' 3% ' 2% ' 0.5%). However, and it is important to realise, that there is absolutely no scientific evidence that this is the case. Haas reported in 1993 that the risk of a permanent paraesthesia following LA administration for all drugs was 1:786,000 injections. With 0.5%, 2% and 3% LAs the risk was 1:1.2 million. With 4% prilocaine the risk was 1:550,000 and articaine 4% was

1:440,000.

However, his statistics were derived from voluntary reports of paraesthesia from Canadian dentists to their insurance carriers and estimates of the number of LA cartridges of each drug sold in Canada during a 21-year period. Additionally the nature of the dental procedure (surgery, conservative) was not known, nor was the length and gauge of the needle. Dr Anthony Pogrel, an oral and maxillofacial surgeon in the San Francisco Bay area has evaluated many patients with post-dentistry paraesthesia and states that the risk of a permanent inferior alveolar nerve paraesthesia is in the range of about 1:26,000. He further states that it is likely that during the practice career of every dentist, one patient will suffer a permanent paraesthesia following IA nerve block (independent of the drug administered). Pogrel states, and I concur, that the cause of the paraesthesia is usually unknown, that there is no way to prevent it (short of never administering a LA again) and there is no known treatment.

How does a practitioner go about successfully anaesthetising the difficult-to-reach and treat tooth?

In one of my rare short-and-concise answers the ability to anaesthetise the 'difficult' tooth has become markedly easier because of two 'innovations'. I speak of (1) the intraosseous injection and (2) articaine, both of which were described earlier. Either used alone has led to greatly increased rates of successful anaesthesia in the 'hot' mandibular molar, traditionally the most difficult teeth in the mouth to anaesthetise. The use of articaine for the intraosseous injection should lead to clinically successful anaesthesia in almost all situations.

As an aside, anaesthesia of maxillary teeth should not pose a problem since infiltration anaesthesia, if it does not work, can be supplemented by the PSA (posterior superior alveolar nerve block), the ASA (anterior superior alveolar nerve block), or the AMSA nerve block. Anaesthesia of mandibular incisors, canine and premolars may easily be obtained through the incisive (mental) nerve block that has a success rate of almost 100%, when administered properly.

Can you explain new LA techniques including the computer controlled LA delivery systems (CCLADS)?

CCLADSs employ a computer to assess the amount of pressure required to administer the LA into the tissues. They are designed to administer the LA at a steady pressure regardless of the dentistry of the tissues, thereby making injections such as the PSA, infiltration and palatal equally comfortable. Additionally these devices are designed to be more aesthetically pleasing to the patient, unlike the more threatening look of the classical metal dental local anaesthetic syringe. Indeed, almost universally patients 'like' the look of these CCLADS devices. CCLADSs, in my opinion, enable the doctor to administer LA more comfortably anywhere in the oral cavity than the traditional hand-held syringe.

An off-shoot of CCLADS research was the 'discovery' of the AMSA and P-ASA nerve block injections. Both techniques are

delivered into palatal tissues in specific sites that provide with pulpal anaesthesia of teeth (AMSA = five maxillary anterior teeth on the side of injection; P-ASA = six teeth: the incisors and canines bilaterally), their supporting buccal and palatal soft tissues and bone.

Interestingly, though a good number of dentists love CCLADS, an equally big number find them of no benefit at all. The dentist who is able to administer LA painlessly with a traditional syringe will not feel that the CCLADS is worth the cost. However other dentists, who find that his/her injections are painful to their patients, will find these devices to be of great benefit. I personally find I do not need to use a CCLADS regularly to administer what my patients call a 'painless' injection (I use topical anaesthesia, stretch the tissue before penetrating with the needle, use sharp sterile needles (25 and 27 gauge, never 30 gauge), and probably of greatest importance, I always inject the local anaesthetic slowly). However, I cannot always guarantee that my palatal injections will be painless, so I find myself using a CCLADS more often than not in this area.

My feeling is that these devices work and that each dentist should give consideration to purchasing one (perhaps on a temporary basis), then using it as much as possible to determine if it satisfies the needs of the dentist's patients.

Please talk about sudden cardiac arrest and automated external defibrillators (AED), which I understand, are now available OTC in the USA

In the USA approximately 1,000 persons die from sudden cardiac arrest every day. The survival rate (discharge from the hospital) nation-wide for out-of-hospital sudden cardiac arrest (OOH SCA) is an abysmal 5% for adults and less than 2% for children.

At the moment, a victim that suffers SCA is clinically dead: they are unconscious, not breathing and have no pulse, i.e. they look dead. However, at the precise moment the heart ceases to pump blood, the cells in the victim's body are not yet biologically dead. Cells throughout the body will remain alive until they exhaust all of the remaining oxygen and glucose in the blood that remains around them, then biological (permanent) death occurs.

Basic life support (BLS = CPR), positioning, airway, breathing and circulation, do not change cardiac arrest into a functional heartbeat. Rather BLS keeps the myocardium oxygenated and able to continue functioning (albeit ineffectually) longer, thus extending the period of time over which defibrillation will be effective.

One very elementary fact: the longer the period of time the victim is in cardiac arrest, even with BLS performed, the lower their chance of survival. Statistics show that for every minute the victim is 'down' (in cardiac arrest) without being defibrillated, the chance of their surviving diminishes by about 7% to 10%. In most OOH-SCAs defibrillation is not available

for at least six or more minutes, thereby explaining the abysmal survival rate for OOH-SCA.

Defibrillators are simple computerised devices that are able to detect the present of only two cardiac rhythms, ventricular tachycardia (VT) and ventricular fibrillation (VF). These are the only two rhythms that can be defibrillated. If the AED detects either of these rhythms it states, 'shock indicated'. Absent one of these rhythms the AED says 'no shock indicated', 'check airway, check breathing, check pulse. If no pulse continue CPR'. The AED cannot diagnose which rhythm is present. It simply knows the rhythm is not one of the two that are shockable, and tells the rescuer to reassess the patient for breathing and pulse and administer whichever of these steps are necessary.

Accuracy rates of AEDs are quite high, running above 99.99%. AEDs are currently found in all major USA airports, and on all commercial jet airliners that have more than one flight attendant.

In a three-year clinical trial at Chicago's O'Hare Airport, 18 travellers suffered SCA. In all, 11 survived to be discharged from the hospital. In all cases the AED was used by a non-medically qualified bystander.

In October 2004 the USA Food and Drug Administration approved the over-the-counter sale of AEDs. At present (February 2005) the cost of AEDs in US\$ is between \$1,200 and \$2,500.

The dental board of the state of Florida has legislated that as of 28 February 2008, all dental practices must have an automated electronic defibrillator on the premises. In its absence, the dentist will be considered to be practising below the minimum standard of care.

Stated simply, AEDs save lives by decreasing the time between collapse of the victim in SCA and delivery of a shock to restart the heart.

Would you like to talk about anything in particular in your specialist field, here or in the States?

Anaesthesiology in dentistry has a long and storied history in both the UK and the USA. In the UK men such as Professor Stanley Drummond-Jackson, while in the USA Niels Jorgensen and Leonard Monheim, pioneered the use of techniques of general anaesthesia which led to the later development of today's conscious sedation, be it via intravenous, intramuscular or inhalation.

As local anaesthetics have become ever more effective and safe, the absolute need for general anaesthesia has diminished dramatically. Indeed the combination of local anaesthesia and intravenous benzodiazepine (such as midazolam) can essentially guarantee the patient both a pain-free as well as anxiety-free dental experience.

I am proud to say that our profession has historically been in the forefront of such advances, a role that it continues to maintain today.