Fatal air embolism

To the Editor:

I commend Davies and Campbell for their work in reporting the three deaths and two cases of morbidity relating to dental implant surgery[1] and Dr. R.L. Matthews for the excellent editorial in the same issue.

I would like to point out two inaccuracies in Davies' and Campbell's report. Citanest Forte is the proprietary name for prilocaine HCl four per cent with epinephrine 1/200,000, not mepivicaine two per cent with levonordefrin 1/20,000 as reported.[2]

The implication that "injection of catecholamines into the peridontal (sic) ligament is virtually the same as direct intravenous injection" cannot be found in the referenced paper.[3] The paper by Lilienthal and Reynolds deals with intraosseous anaesthesia, and while injection into the bone may result in blood levels of catecholamines and anaesthetic agent comparable to that of an intravascular injection,[4] this is not true of the periodontal ligament injection commonly employed for dental anaesthesia.

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3 *Lilienthal B, Reynolds AK.* Cardiovascular responses to intraosseous injections containing catecholamines. Oral Surg Oral Med Oral Pathol 1975; 40: 574–83.
4 *Allen GD.* Dental Anesthesia and Analgesia. 3rd ed. Baltimore: Williams and Wilkins, 1984.

REPLY

We thank Dr. Wright for his critical appraisal of our paper. He is correct concerning the proprietary name of one of the local anaesthetics used in the reported cases. Patient 2 received mepivacaine, the proprietary name of which is Carbocaine.

The paper by Lilienthal and Reynolds[1] describes the cardiovascular response to the intraosseous (IO) injection of catecholamines and clearly demonstrates the "rapidity with which the catecholamines are absorbed into the general circulation." Dr. Wright is correct in stating that this paper does not refer directly to periodontal ligament (PDL) injection, a method of administering local anaesthetic "directly adjacent to a tooth to be anesthetized."[2] However, the results are the same with either the IO or PDL technique. Indeed, two papers on the subject of periodontal ligament injection equate this technique to that of intraosseous injection, on the basis of the spread of the injected solution and the systemic effects of the injectate.[2,3] Periodontal ligament injection results in local anaesthetic being "distributed widely by passing through the cribiform plate and the medullary bone spaces and into the vasculature in and around the tooth and adjacent teeth."[2] Solutions injected by the PDL technique are rapidly absorbed into the systemic circulation, and the results are similar, "whether the injections were done intravenously, intraosseously or periodontally."[3] Smith and Walton also state that the "name of the technique refers to the intended site of needle insertion and not to the path or spread of the injected solution... In reality, the periodontal ligament injection is an intraosseous injection."[2]

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Massive tongue swelling after uncomplicated general anaesthesia

To the Editor:

We present a case of an uncomplicated general anaesthetic requiring postoperative tracheal intubation due to massive tongue swelling possibly due to the glutaraldehyde solution used for sterilization.

A 67-yr-old man, following removal and debridement of infected components of a left total knee arthroplasty under uncomplicated general anaesthesia, presented for a second debridement. The patient weighed 104 kg and had a 25 pack-year history of cigarette smoking. His medical history included hypertension, exertional angina relieved by sublingual nitroglycerin, and well-controlled psoriasis. He had had an anaphylactic reaction to penicillin ten years ago which required tracheal intubation. His medication included metoprolol, furosemide, captopril, and cefotaxime.

After premedication with midazolam and fentanyl, thiopentone and succinylcholine were administered. The trachea was intubated atraumatically with a size 7.5 mm cuffed tracheal tube (Sheridan Catheter Corporation). A No. 2 Miller blade previously cleaned and sterilized in two per cent glutaraldehyde solution[1,2] was used for intubation. Anaesthetic maintenance was with isoflurane in nitrous oxide/oxygen (50/50).

After several hours of uneventful surgery and anaesthesia the trachea was extubated and the patient was taken to the recovery room. He was given oxymorphone for pain relief. Fifteen minutes later he complained of swelling of his tongue. On examination, the tongue was twice its normal thickness; however, his airway and ability to swallow were intact. Diphenhydramine 50 mg IV and methylprednisolone 300 mg IV were administered. Over the next ten minutes the swelling of his tongue rapidly progressed to fill the entire oral cavity and force his mouth wide open. The patient complained of inability to clear secretions and his speech was obviously impaired. With no topical or regional anaesthesia, a fibreoptic nasal intubation was performed with a 7 mm tracheal tube. The epiglottis and vocal cords appeared normal. The patient was taken to the intensive care unit, breathing spontaneously without difficulty. Four hours after his arrival in the intensive care unit, the patient's tongue had returned to normal size. The tracheal tube was removed and the subsequent postoperative course was uneventful.

Discussion

The cause of this life-threatening complication is unclear. Three mechanisms for swelling of the tongue and oropharynx can be postulated: (1) mechanical trauma or obstruction of venous or lymphatic flow, (2) hypersensitivity response to intravenous medication, or (3) tissue reaction to a substance applied locally.

It seems unlikely that mechanical trauma would cause this symmetrical, profound, isolated swelling of the tongue – the intubation was atraumatic. Also, it is unlikely that the reaction was due to a systemic hypersensitivity response because it was confined to the tongue. The remainder of the mucous membranes in the oropharynx were normal, as were the epiglottis, vocal cords, and tracheal lining. A skin test for previous sensitization to thiopentone was negative. The most likely explanation for this patient's problem is a local reaction to an applied substance. The lidocaine jelly had been used with each previous occasion at this institution without complication. The tracheal tubes used are implantation-tested and carry the Z-79 designation. The most likely explanation for this problem may be the glutaraldehyde solution.

A skin test was performed with 0.02 per cent glutaraldehyde solution. The patient did not complain of burning or irritation in response to the intradermal injection.[3] However, there was a wheal-and-flare response that was interpreted by the allergist as indicating sensitization. While we cannot be certain that the result of this skin test was not due to local irritation by the glutaraldehyde, the response is strongly suggestive of prior sensitization.

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2 Leads from the NMWR (Morbidity and Mortality Weekly Report) (Vol. 34/Nos. 43, 45, 1985), Centers for Disease Control, Atlanta: Summary: Recommendations for preventing transmission of infection with HTLV-III/LAV in the Workplace. JAMA 1985; 254: 3162–7.

3 Material Safety Data Sheet, 2% Glutarladehyde. Arlington, Texas: Surgikos, 1988: 1–4.

Acute pulmonary oedema after tourniquet release

To the Editor:

We would like to describe the unusual case of a fit adult man undergoing arthroscopy for septic arthritis who developed acute pulmonary oedema shortly after deflation of a limb tourniquet.

Case report

A fit 20-yr-old man, diagnosed as having septic arthritis of the knee, was scheduled for emergency arthroscopy and irrigation. Preanaesthetic assessment revealed: temperature 39° C; pulse rate 120 per minute; cardiorespiratory assessment was otherwise normal. All investigations were normal except for a leucocytosis of $19.5 \times 10^9 \cdot L^{-1}$. The patient was adequately fasted. Anaesthesia was induced with thiopentone 5 $mg \cdot kg^{-1}$ and maintained with