Fatality Associated with the Combined Use of Halothane and Gingival Retraction Cord


Local anesthetic solutions containing epinephrine frequently are employed for hemostasis and postoperative pain control during oral surgical procedures on patients receiving general anesthesia. Most anesthesiologists are aware of this practice and will avoid the use of halothane or will restrict the total dose of epinephrine to prevent serious cardiac dysrhythmias. However, there is another source of exogenous epinephrine frequently employed during restorative dental procedures that may be unfamiliar to many anesthesiologists.

Gingival retraction cord impregnated with racemic epinephrine often is used by general dentists and prosthodontists to provide gingival retraction and hemostasis during dental reconstructive procedures (i.e., crowns, bridges). Systemic absorption of racemic epinephrine from the gingival sulcus can produce significant dysrhythmias, especially in the presence of halogenated hydrocarbon anesthetics.1-3 We describe a fatality that resulted from the combined use of halothane and gingival retraction cord impregnated with 8% racemic epinephrine.

REPORT OF A CASE

A 36-year-old man weighing 69 kg was admitted to the hospital for extensive dental restorative procedures. The medical history was negative, and preoperative laboratory tests including urinalysis, complete blood count, and chest roentgenogram were within normal limits.

Meperidine 25 mg, promethazine 25 mg, and scopolamine 0.2 mg were given im approximately 1 hour prior to surgery. On admission to the operating suite, the vital signs were BP 138/80 mm Hg, HR 80 bpm, respirations 18 breaths · min−1. General anesthesia was induced with thiopental sodium 200 mg iv and oral endotracheal intubation was facilitated by the administration of succinylcholine 100 mg iv.

General anesthesia was maintained with 1% halothane and 70% nitrous oxide with the patient breathing spontaneously. Blood pressures were not recorded by the anesthesiologist; however, heart rate and respiratory rate remained at 80 bpm and 20 breaths · min−1, respectively, during the first 3 h of the procedure.

Approximately 2 h and 45 min after the induction of anesthesia and within 10 min of the placement of gingival retraction cord impregnated with 8% racemic epinephrine around 21 teeth, the patient became cyanotic with labored breathing. Ventricular fibrillation was noted on ECG lead II.

Halothane was discontinued, and ventilation was controlled with a FiO2 of 1.0. Cardiopulmonary resuscitation was initiated, and electrical countershocks 4 were delivered to the heart with chest electrodes, after which ECG showed ventricular fibrillation. Cardiopulmonary resuscitation was resumed, and percutaneous cannulation of the subclavian vein via the external jugular approach was accomplished. Sodium bicarbonate, 44.6 mEq, was administered iv and a single electrical countershock was repeated. This attempted defibrillation was unsuccessful. Lidocaine 100 mg and NaHCO3 44.6 mEq were administered iv; pH was 7.19, PaCO2 25 mm Hg, PaO2 557 mm Hg, HCO3 10 mEq · l−1, and base deficit −17 mEq · l−1. An additional 44.6 mEq of NaHCO3 was administered iv, as were calcium chloride 1 g, epinephrine 1 mg, and dopamine infused at 550 μg · min−1. Repeated attempts at defibrillation were unsuccessful, and the patient was pronounced dead after 40 min of resuscitative effort.

DISCUSSION

Gingival retraction cord impregnated with 8% racemic epinephrine is used frequently in dentistry to provide hemostasis and allow access and visualization for certain restorative procedures (i.e., crowns and bridges) (figure 1). Some cords may contain as much as 1.0 mg of l-epinephrine per inch.4 The epithelial lining the gingival sulcus is semipermeable, allowing passage of low molecular weight molecules.5 Furthermore, the gingival sulcus is highly vascular. These factors undoubtedly account for the observation that 24-92% of epinephrine applied to the gingival sulcus is absorbed into the systemic circulation.4

In 1956, Raventos1 demonstrated that halothane increased the sensitivity of the myocardium of the dog to epinephrine. Katz et al.2 concluded that the amount of subcutaneous epinephrine administered to adults should not exceed 10 ml of 1:100,000 epinephrine in any 10-min period up to a total of 30 ml per hour.2 More recently, Johnston et al.3 found that the epinephrine dose producing three or more premature ventricular contractions in 50% of their adult patients (ED50) receiving halothane was 2.1 μg · kg−1 · h−1. The arrhythmogenic ED50 for isoflurane and enfurane was 6.7 and 10.9 μg · kg−1, respectively. The
Pacemaker Syndrome during Anesthesia

JOSEPH M. FORAND, M.D.,* AND JOHN F. SCHWEISS, M.D.†

Many patients require anesthesia and surgery previously having had permanent pacemakers inserted for conduction abnormalities, particularly sick sinus syndrome and symptomatic bradycardia. Although many complications of artificial pacing such as power source failure, pulse generator malfunction, electrode fracture, and malpositioning of the electrode are well known, we present an unusual problem in a patient with a permanent ventricular demand pacemaker that has become known as "pacemaker syndrome."

**Report of a Case**

A 77-year-old man had, over several months, noted increasing "dizziness," especially when rising from bed in the morning. On auscultation, bilateral carotid bruits were noted. Carotid arteriograms revealed complete occlusion of the left internal carotid artery and an 80% stenosis of the right proximal internal carotid artery. He also had a history of sick sinus syndrome with profound bradycardia for which a permanent transvenous ventricular demand pacemaker had been inserted 4 years previously. Other significant medical problems were compensated congestive heart failure, chronic obstructive pulmonary disease, and diffuse peripheral vascular disease. His medications included digoxin, procainamide, meclizine, diprydamole, and nitroglycerin. Preoperative evaluation included 12 lead EKGs, with and without a magnet, that revealed a functioning demand pacemaker set at 72 beats/min. The patient was scheduled for a right carotid endarterectomy under general anesthesia.

Premedication consisted of meperidine 25 mg, diphenhydramine 25 mg, and glycopyrrolate 0.2 mg im. A radial artery catheter was inserted percutaneously for continuous arterial pressure monitoring. Following pancuronium (1 mg iv), a rapid sequence induction for endotracheal intubation was accomplished following thiopental 150 mg and succinylcholine 80 mg iv. Anesthesia then was maintained with 60% nitrous oxide, fentanyl 0.35 mg, and pancuronium 5 mg iv.

The course of anesthesia initially was uneventful, with a sinus rhythm at a rate of 75–80 beats/min and a systolic arterial blood pressure varying between 140 and 160 mmHg. Because systolic blood pressure increased to 180 mmHg during initial vessel dissection, a sodium nitroprusside infusion, 35 μg·min⁻¹, was employed for less than 15 min. One per cent lidocaine then was infiltrated into the tissues surrounding

---

Received from the Department of Anesthesiology, St. Louis University School of Medicine, St. Louis, Missouri 63104. Accepted for publication October 24, 1983.  
* Resident in Anesthesiology.  
† Professor and Chairman, Section on Anesthesiology.  
Address reprint requests to Dr. Forand.  
Key words: Complications: hypotension. Heart: pacemakers, artificial.