

by a reduction in the pH of the solution. The pKa of lidocaine is 7.91 at 25°C and 7.57 at 38°C.⁴ The order of the percentage of the ionized forms of lidocaine existing in the solutions should be LE 4.9 > LE 6.2 > LE 7.5, and this should be the reverse in order of the anesthetic potency and the speed of onset of the anesthetic action of the solution.^{5,6} The mechanism of action of lidocaine in accelerating epinephrine absorption is not known. Possible mechanisms are local tissue irritation⁶⁻⁸ and vasodilating action.^{6,7} Enhancement of the action of lidocaine in tissue irritation is reported to be correlated with a reduction in the pH of the solution, which increases the percentage of the ionized forms of lidocaine.⁸ Vasodilation by lidocaine is expected at the concentration used in this study,⁷ but further studies will be needed to elucidate the effect of pH on vasodilating activity of lidocaine. Provided that the mechanisms are the local tissue irritation and the vasodilating action, which would increase the blood flow and the permeability of the blood vessels, the effect of molarity of the solution on the rate of absorption of an injected substance may be diminished.⁹ The regression equations obtained from the values of groups LE may indicate that the effect of sodium concentration on the rate of absorption of epinephrine is less obvious than that of pH in the presence of lidocaine.

Commercial preparations of lidocaine with epinephrine have a pH lower than 5. Lidocaine and epinephrine in such a low pH solution not only promote the increase in the blood level of epinephrine, but also reduce the

anesthetic action.^{5,10} Epinephrine should be added to plain lidocaine prior to use to avoid increasing the acidity of local anesthetic solutions containing epinephrine. Sodium bicarbonate can also be used to increase the pH of the anesthetic solution.^{5,8}

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Anesthesia-related Complications in Children with Duchenne Muscular Dystrophy

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Duchenne Muscular Dystrophy (DMD) is the most common and severe form of childhood myopathy. Pa-

tients suffering from this disease often require anesthesia to obtain a muscle biopsy or to correct orthopedic deformities. Occasional case reports have described perioperative complications in these patients.¹⁻⁴ This study was designed to determine the incidence and type of anesthesia-related complications occurring in these children and identify risk factors.

METHODS

The medical records were reviewed retrospectively of all patients with DMD who were known to have had surgery with general anesthesia at our institution during a 5-yr period ending January, 1985. The diagnosis of DMD was based upon: 1) a characteristic history and

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physical examination which included a positive family history in male members and progressive proximal muscle weakness; 2) elevation of serum creatine phosphokinase (CPK) levels; and 3) electromyography and muscle biopsy consistent with myopathic disease. One child did not have a muscle biopsy, but had all the other criteria (see case 1).

RESULTS

There were 25 patients, all males, with a mean age of 7.3 yr (range 1–18 yr). Each patient underwent one operation, except one boy who had three procedures. Preoperative serum CPK levels ranged from 158–14,600 iu with a mean of 6303 iu (normal is less than 50 iu). Electrocardiograms (ECG) were performed in 19 patients and were abnormal in 13. The abnormalities were typical of DMD and included tall R-waves in the right precordium, deep Q-waves in limb and left precordial leads, biventricular hypertrophy, and sinus tachycardia.⁵ Echocardiograms were performed at rest in three patients and were normal.

The type of operations are listed in table 1. Premedication was administered to 16 patients and included narcotics, tranquilizers, barbiturates, and/or anticholinergic drugs. Nineteen of the 25 patients received either halothane or isoflurane after intravenous (iv), rectal, or inhaled induction of anesthesia, including the one patient who had three separate inhaled anesthetics. The remaining six patients had iv anesthesia with combinations of barbiturates, narcotics, and tranquilizers. Nitrous oxide was administered to all patients. Seventeen patients had their tracheas intubated. Non-depolarizing muscle relaxants were administered to 11 patients, and one patient received succinylcholine uneventfully.

Four patients developed complications which are described below. The one child who underwent multiple procedures had no anesthetic difficulties.

Case 1: A 5-yr-old boy with a clinical diagnosis of DMD was scheduled for a confirmatory muscle biopsy. His past medical history and physical examination were unremarkable except for mild weakness. There was no evidence of cardiac disease and his preoperative ECG was normal. He received no premedication and was anesthetized with halothane, nitrous oxide, and oxygen *via* face mask with assisted ventilation. Maximum inspired halothane concentration attained during induction was 2%. Approximately 10 min after beginning anesthesia, peaked T-waves were noted on ECG, which progressed to widened QRS complexes and asystole. Cardiopulmonary resuscitation was initiated immediately and continued for 90 min until spontaneous cardiovascular function returned. The highest serum potassium level measured was 8.9 meq/l. He made a full recovery without neurological sequelae. This case has recently been reported in detail.⁶

Case 2: A 13-yr-old boy was scheduled for scoliosis repair. He was wheelchair-bound with no apparent cardiac disease. His preoperative ECG revealed deep Q-waves in V6 and increased anterior forces characteristic of DMD. An echocardiogram showed normal left ventricular

TABLE 1. Operations

Type	No. of Procedures
Muscle biopsy	11
Posterior spinal fusion	5
Heelcord lengthening	4
Tendon transfer	4
Abscess drainage	3

size and a shortening fraction of 27–30%. He did not receive premedication. Anesthesia was induced with thiopental iv and maintained by inhalation of isoflurane (0.4–1.5%), nitrous oxide, and oxygen and the iv administration of fentanyl and pancuronium. The anesthetic course was uneventful until 7 h into the procedure, when he suddenly developed bradycardia and hypotension unresponsive to the iv administration of fluids and sympathomimetic drugs and closed-chest cardiac massage. At autopsy, the heart was enlarged with biventricular dilatation and hypertrophy. The myocardium contained numerous scattered broad bands of fibrosis most pronounced posteriorly and laterally in the subepicardial areas (fig. 1). Microscopic examination of the lungs revealed small areas of fat embolization and platelet aggregation in the pulmonary vessels.

Case 3: A 6-yr-old boy underwent a heelcord lengthening procedure. Past medical history and preoperative ECG were unremarkable. He was premedicated with pentobarbital rectally and morphine and atropine im. Anesthesia was induced with thiopental iv and maintained with halothane, nitrous oxide and oxygen. Maximum inspired halothane concentration was 2%. The trachea was intubated without muscle relaxants, and ventilation was controlled. Body temperature and heart rate increased progressively during the first 2 h of surgery, despite undraping the patient in a cool operating room and the use of an unheated, nonbreathing anesthesia system. At the time, esophageal temperature had risen from 35.5 to 38.6° C, arterial blood gases revealed pH_a 7.52, PaO₂ 205 mmHg, and PaCO₂ 25 mmHg with an FIO₂ of 0.5. Body temperature decreased spontaneously after halothane was discontinued and fentanyl was substituted (fig. 2). The remaining intraoperative and postoperative course was uneventful.

Case 4: An 8-yr-old boy underwent tendon transfers in the lower extremities. Preoperative ECG revealed left ventricular hypertrophy,

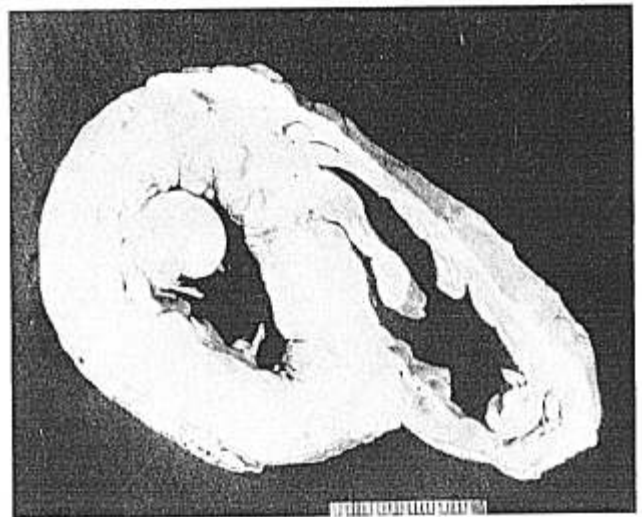


FIG. 1. Sectioned heart (case 2). Enlarged dilated hypertrophic ventricles with scattered fibrous tissue replacing myocardium.

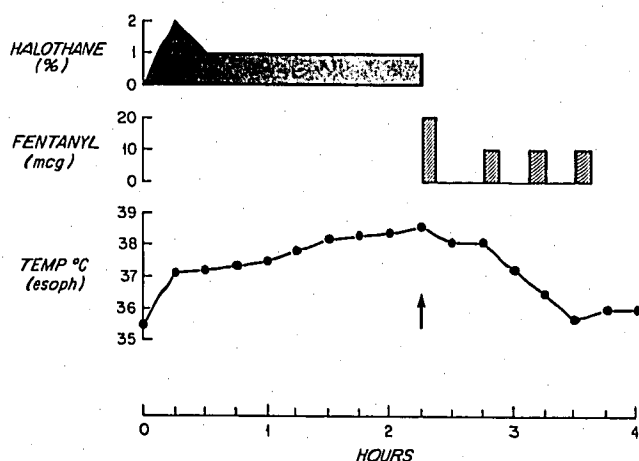


FIG. 2. An example of unexplained temperature elevation during heeler cord lengthening in a 6-yr-old boy responding to discontinuation of halothane. The arrow indicates the time when an arterial blood gas analysis was performed; $pH_7.50$, PaO_2 205 mmHg, $PaCO_2$ 25 mmHg with an FiO_2 of 0.5.

ST and T-waves changes, and deep Q-waves in leads I, III, AVF, V4-V6. He received no premedication and was anesthetized by inhalation of halothane, nitrous oxide, and oxygen and iv morphine. His trachea was intubated without muscle relaxants, and ventilation was controlled. The maximum inspired concentration of halothane was 4% during induction, but rapidly reduced to about 1.5% throughout most of the case. Esophageal temperature gradually increased from 36.0° C to 38.2° C over 2 h, accompanied by a rise in heart rate from 100 to 140 bpm. There was no change in arterial blood pressure. The elevation in body temperature did not respond to undraping the child and administration of unheated inspired gases. Temperature and heart rate gradually decreased after halothane was discontinued and returned to pre-induction values prior to termination of the operation. Recovery was uneventful.

DISCUSSION

Four of the 25 patients had complications during anesthesia. Although all complications occurred in patients who received volatile inhaled anesthesia, many of the patients who did not develop complications received similar anesthetic management. There did not appear to be any factors which preoperatively identified patients who developed problems.

Intraoperative cardiac arrest developed in two children (cases 1 and 2). In the first child, asystole resulted from acute hyperkalemia. Although acute hyperkalemia has been reported in DMD patients following administration of succinylcholine and halothane,^{2,7} this case is unusual in that cardiac arrest developed abruptly following induction with halothane, nitrous oxide, and oxygen alone. Although the etiology of this reaction is not known, there is good reason to suspect it was secondary to the acute onset of malignant hyperthermia (MH). Hypoxia despite supplemental oxygen, hypercarbia despite hyperventilation, metabolic acidosis,

myoglobinuria, and massive elevation of CPK are typical findings in this condition, and all occurred with this patient. Hyperthermia frequently develops late and may not occur if circulatory arrest is sudden.⁸ While there are reports which describe uneventful administration of general anesthesia to many patients with muscular dystrophy using a variety of techniques,^{9,10} there are numerous case reports of anesthesia-related perioperative complications in children with DMD. Some of these appear to be typical MH reactions.^{1,3,4} Others manifest only some of the features of MH (such as rhabdomyolysis), and perhaps they represent an abnormal response distinct from MH in these patients.¹¹ The relationship of such abnormal responses to typical MH remains undefined.

The cause of cardiac arrest and death in the second patient was likely related to heart failure secondary to chronic cardiomyopathy. The severely weakened heart may have been unable to compensate for massive blood loss, large amounts of transfused blood and crystalloid solutions, and variations in systemic arterial blood pressure and vascular tone which occurred during the operation. Dystrophic involvement of the myocardium does occur in teenagers with DMD,¹² but may be difficult to appreciate preoperatively, since exercise tolerance is limited. Echocardiography in the resting state may not reflect the ability of the myocardium to respond to stress. All volatile anesthetics cause a dose-related depression of myocardial contractility, although isoflurane appears to have less effect than halothane.¹³ Nevertheless, even isoflurane may produce a significant negative inotropic effect in patients with a diseased heart. Microscopic pulmonary embolization may have contributed to the failure of cardiopulmonary resuscitative efforts in our patient, but was not thought to be extensive enough to cause cardiac arrest.

Complications were less severe in the other two patients. However, they both exhibited unusual responses to halothane anesthesia. Body temperature does not commonly rise during anesthesia, and mild unintentional hypothermia is a well-recognized phenomenon in children undergoing halothane anesthesia.¹⁴ Temperature elevation in these two patients did not respond to routine measures of enhancing body heat loss in the operating room, but did abate promptly with discontinuation of halothane. Unexpected hyperthermia has occurred in children with DMD anesthetized with enflurane.¹⁵ This is of concern because hyperthermia may be one sign of an MH episode.

In summary, serious complications can occur in children with DMD during anesthesia. These problems may be minimized by using narcotics instead of potent volatile anesthetics, and nondepolarizing muscle relaxants instead of succinylcholine. Temperature should be

monitored and dantrolene should always be available for immediate use in case an MH episode occurs. Local and regional anesthesia may be adequate for many surgical procedures these patients require.

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Atracurium and Vecuronium Do Not Affect Extraocular Muscle Function After Outpatient Surgery

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Postoperative visual disturbances, such as double vision, may affect up to 71% of patients who have received long-acting nondepolarizing muscle relaxants at the time of discharge from the hospital.¹ Such problems

may continue for a day or more after surgery.² These disturbances may be especially serious for outpatients. Extraocular muscle tone (EMT) of the medial recti may be measured in prism diopters using horizontal scale of the Maddox wing test,³ a simple hand-held device (fig. 1). This study was designed to compare EMT after the new intermediate-acting nondepolarizers atracurium (ATR) and vecuronium (VEC) with succinylcholine (SDC) in outpatients after a standardized anesthetic technique.

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MATERIALS AND METHODS

After approval by the Human Subjects Research Committee, 35 ASA physical status I or II females undergoing outpatient laparoscopy were selected. Written informed consent was obtained from each patient. All patients received fentanyl 50-150 µg iv and glycopyrrolate 0.2 mg iv when the intravenous catheter was inserted. After baseline Maddox readings, measured be-