CASE REPORTS

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Ventricular Tachycardia and Brief Cardiovascular Collapse in Two Infants after Caudal Anesthesia Using a Bupivacaine-Epinephrine Solution

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CAUDAL anesthesia, combined with general anesthesia, has gained wide acceptance in pediatric anesthesia. Dalens et al. reported no major complications or neurologic sequelae following caudal anesthesia in 750 children. However, he reported three cases of accidental intravenous injections of the test dose, resulting in instantaneous cardiac arrhythmias. We report two cases of ventricular tachycardia and brief cardiovascular collapse after caudal anesthesia with bupivacaine-epinephrine in two infants, aged 4 and 10 months.

Case 1
A 4-month-old male infant weighing 4 kg was scheduled for inguinal herniorrhaphy. His medical history was significant for prematurity at 32 weeks, without a history of apnea. On arrival to the operating room, his systolic blood pressure and heart rate were 90 mmHg and 120 beats/min, respectively. Under general anesthesia with halothane (1.5%), nitrous oxide (67%), and oxygen (33%) via a mask, and receiving assisted ventilation, the patient was placed in the right lateral decubitus position. Hemoglobin oxygen saturation measured by finger pulse oximetry (SpO2) was 100%; end-tidal carbon dioxide (PetCO2) was not measured. After skin preparation, a standard 23-G, 50-mm-long intramuscular needle (Becton-Dickinson, Franklin Lakes, NJ) was inserted into the sacral canal. A test dose was not used. Gentle aspiration before and during injection of 4 ml of 0.25% bupivacaine with 1:200,000 epinephrine was negative for blood and cerebrospinal fluid. The solution was injected within 30-40 s. On turning to the supine position, the patient was noted to be pale in color. His heart rate had increased from 130 to 200 beats/min, his systolic blood pressure had decreased from 97 to 46 mmHg, and his SpO2 had decreased from 100 to 76%. The electrocardiogram revealed a pattern consistent with ventricular tachycardia. Halothane and nitrous oxide were discontinued and the trachea was intubated. PetCO2 was 17 mmHg, measured at the proximal end of the endotracheal tube. The patient's lungs were ventilated with 100% oxygen. Approximately 60 s after the onset of tachycardia, the heart rate slowed to 120 and returned to normal sinus rhythm. The patient's color improved as the SpO2 increased to 98% and systolic blood pressure returned to 97 mmHg. The case proceeded uneventfully under isoflurane (1-1.5%), nitrous oxide (60%), and oxygen (40%) anesthesia. There was no evidence of a satisfactory caudal block. The patient was discharged the following day after an uneventful postoperative course.

Case 2
A 10-month-old female infant weighing 8 kg was scheduled for pyloroplasty for uretero-pelvic-junction obstruction. General anesthesia was induced with halothane (1.5%), nitrous oxide (67%), and oxygen (33%). After receiving 10 mg/kg intravenous atropine and 0.4 mg/kg intravenous atracurium, the trachea was intubated. PetCO2 was 38 mmHg, measured at the proximal end of the endotracheal tube. SpO2 was 100%. Caudal anesthesia was performed with the patient in the lateral position, using a standard 23-G, 50-mm-long intramuscular needle (Becton-Dickinson), and after verification of negative aspiration for blood or cerebrospinal fluid before and during the injection, 8 ml of 0.25% bupivacaine with 1:200,000 epinephrine was injected over 30-40 s. A test dose was not used. On turning to the supine position, the patient was noted to be pale in color. The femoral pulse was barely palpable. The heart rate had increased from 120 to 180 beats/min, the systolic blood pressure had decreased from 98 to 40 mmHg, and the finger pulse oximeter failed to detect a pulse signal. The electrocardiogram revealed a pattern consistent with ventricular tachycardia. Halothane and nitrous oxide were discontinued and the patient's lungs were ventilated with 100% oxygen. The patient was given 1 mg/kg lidocaine intravenously. Within 1 min of the onset of symptoms, there was return of the heart beat and blood pressure to preinjection values. A serum bupivacaine level (determined by a Hewlett Packard 5890 gas chromatography model; Hewlett Packard, Wilmington, DE) drawn from a vein 10 min after the injection, was 1.9 µg/ml. The surgical procedure was cancelled, and the patient was transferred to the pediatric intensive care unit for monitoring. There was no evidence of sensory or motor blockade. Postoperative electrocardiogram and echocardiogram were normal.

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Discussion

The most interesting points of the two cases described here are the immediate onset of hemodynamically significant tachyarrhythmias (either ventricular tachycardia or supraventricular tachycardia with aberrant conduction) indicating intravascular or intraosseous injection, and rapid resolution of the symptoms with or without treatment. There was no block, implying intravascular injection in both cases. It has been reported that accidental intravascular injection could occur after a negative aspiration test for blood.§ Dalens² reported a higher incidence of intravascular injections with needles ordinarily used for intramuscular injections than with a short level needle (10.6% vs. 1.6% of times used). In addition, three inadvertent intravenous injections of the test dose resulting in instantaneous cardiac arrhythmias occurred, all in the intramuscular needle group.

The serum bupivacaine level obtained in the second case was 1.9 μg/ml at 10 min. This is consistent with an intravascular injection, as reported by Sharpe et al.,† during which one patient, administered 0.375% bupivacaine with 0.5 ml/kg 1:200,000 epinephrine, had plasma levels of 3.72, 2.82, 1.79, and 1.21 μg/ml at 2, 5, 10, and 15 min, respectively.

Do the clinical signs in these two cases represent epinephrine toxicity or bupivacaine toxicity? Arrhythmias are known to occur after administration of epinephrine in the presence of halothane. § Arrhythmias were seen with test doses of epinephrine in halothane-anesthetized lambs. In pigs, premature ventricular contractions or ventricular tachycardia resulted when epinephrine was infused intravenously during halothane anesthesia. ** The arrhythmogenic dose of epinephrine in the older pigs (aged 50–60 days) was 10.5 μg/kg.

The accidental intravenous injection of 5 μg/kg epinephrine in both of our cases is ten times the dose used as a test dose (0.5 μg/kg),³ and approximately 50 times what one would use clinically for its inotropic effect (0.1 μg·kg⁻¹·min⁻¹).

Arrhythmias, often refractory to resuscitation, can also occur with bupivacaine.⁴ Because SPO₂ was well maintained in both patients before and during the block, and the second patient was receiving controlled ventilation with a PetCO₂ of less than 40 mmHg, hypoxia in both cases, and hypercarbia in the second case, are unlikely causes for the arrhythmia.†† The toxic plasma levels of bupivacaine in adults range from 3–5 μg/ml.⁵ In children, toxic plasma levels of bupivacaine associated with convulsions appear to range from 2–10 μg/ml.⁶–⁸ In young pigs, toxic plasma concentrations of bupivacaine were found to be age dependent.⁹ The younger pigs were found to be more resistant to central nervous system or cardiac toxicity after rapid bupivacaine infusion. Human infants, however, are at a greater risk of bupivacaine toxicity because α₁-acid glycoprotein, which binds bupivacaine, is lower in infants compared with older age groups.¹⁰ Scott¹¹ suggests that the absolute toxic plasma concentration may be more dependent on the rate of increase than on any exact concentration, with the higher absolute levels tolerated when the rate of increase is slow.

The possible advantages of adding epinephrine to a bupivacaine solution could be epinephrine’s effect of slowing vascular absorption, and its beneficial positive inotropic cardiac effects in case of accidental intravascular injection.¹² In rats, it has been shown that epinephrine actually increases cardiorespiratory toxicity when administered with bupivacaine.¹³ Finally, epinephrine may also serve as a useful marker for intravascular injection, although, under halothane anesthesia, it is more reliable when treatment with intravenous atropine has been given previously.³

Lidocaine, although useful in treating ventricular arrhythmias, may be additive with bupivacaine in lowering the seizure threshold,¹⁴ and may lower the ventricular tachycardia threshold in some animals.¹⁵ Animal data indicate that ventricular arrhythmias from bupivacaine are probably better treated with bretylium than with lidocaine.¹⁵

In conclusion, we report two accidental intravascular injections of bupivacaine and epinephrine during the performance of caudal anesthesia in infants. These were associated with wide complex tachycardias and hy-

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† Sharpe TDE, Goresky GV, Sabourin MA: Comparison of plasma blood levels of bupivacaine after caudal injection of 0.25% and 0.375% solution with 1:200,000 epinephrine (abstract). Anesthesiology 69:A764, 1988.


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potension that were short lived, and probably represented epinephrine toxicity. Although the accuracy of test dosing before administering full doses of caudal local anesthetics remains uncertain, we recommend routine test dosing and slow fractional administration of the entire local anesthetic dose when performing caudal epidural blocks in children. Furthermore, with the recent commercial availability of equipment specifically designed for providing pediatric regional anesthesia, we also recommend that, in the future, needles specially designed for regional anesthesia in children be employed.

References

Operating Room Fires Initiated by Hot Wire Cautery

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OPERATING room fires continue to occur,1–3 because of the simultaneous presence of a source of ignition, a flammable material, and an oxidizing atmosphere.2,3 Electrocautery and lasers are common sources of ignition. Flammable materials found in the operating room include a variety of objects, such as cotton towels,4 plastic tapes, endotracheal tubes,4,5 surgical drapes,6 and cotton gauze sponges.4 The oxidizing nature of the atmosphere usually is caused by supplemental oxygen or nitrous oxide.2,3

We report four cases of operating room fires caused by contact of disposable hot-wire cauteries to flammable materials (cotton gauze or eyelashes). An experimental study was performed to investigate whether these cauteries can ignite cotton gauze and other ma-

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