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Bupivacaine Cardiotoxicity in a Pregnant Patient with Mitral Valve Prolapse

To the Editor:—A 22-year-old woman, 38.5 weeks gestation, was admitted for management of labor and delivery. Although obstetric history was negative, medical history was significant for occasional dizziness, dyspnea and palpitations, and several episodes of syncope during the prior 2 years. Neurologic evaluation revealed no etiology; however, cardiac consultation was not obtained prior to admission (postoperative echocardiogram showed mitral valve prolapse). The patient's weight was 65 kg, height was 160 cm, and physical examination was unremarkable.

Following 9 hours of labor, the decision was made to perform a cesarean section. An epidural catheter placed at L3–4 revealed blood on aspiration. Therefore, the catheter was removed and another inserted at L4–5. Aspiration was negative for blood or cerebral spinal fluid, and test doses of 3% chloroprocaine (2 and 5 ml) produced no sign of subarachnoid or systemic injection. However, shortly after injection of the therapeutic dose (20 ml, 0.75% bupivacaine) a generalized seizure occurred. Endotracheal intubation, utilizing cricoid pressure and facilitated by succinylcholine administration, was carried out within 1 min. Immediately after intubation, the cardiac rhythm was ventricular tachycardia (200 bpm), which remained the predominant rhythm for the next 26 min. Resuscitation, with a right hip wedge in place, included ventilation with oxygen, intravenous boluses of lidocaine (100 mg, 100 mg, and 50 mg) and sodium bicarbonate (44 mEq twice), and cardioversion (50, 50, and 100 watt-s, the last of which resulted in normal sinus rhythm). Subsequent cesarean section was performed with good neonatal outcome, and the postoperative course was unremarkable for mother and infant.

We feel this case illustrates an example of bupivacaine cardiotoxicity. Although all local anesthetics may exhibit cardiovascular toxicity, several studies suggest that the relative cardiotoxicity (compared with central nervous system toxicity) is greater for bupivacaine than for lidocaine.[1–5] Additionally, the cardiotoxicity of bupivacaine (but not of lidocaine) is enhanced by hyperkalemia,[6] hypercarbia,[7] hypoxia,[7] and acidosis,[7] all of which occur with generalized seizures. In our patient, however, the presence of mitral valve prolapse (which predisposes to arrhythmias) may have contributed to the ventricular tachycardia.

This case also illustrates several points that are pertinent to prevention of local anesthetic toxicity when epidural anesthesia is used following puncture of an epidural vessel. First, aspiration and the test dose did not reveal intravascular placement of the catheter (and we have found that in parturients, 5 ml of 3% chloroprocaine uniformly produces signs of CNS toxicity without producing seizures). However, toxicity may have resulted from the rapid passage of bupivacaine into the systemic circulation via the open epidural vessel, aided by an elevated epidural pressure resulting from injection of the large dose (20 ml). Obviously, fractionating the dose (*e.g.*, 5 ml per min) would reduce or eliminate this risk, or possibly one should consider an alternative anesthetic technique if puncture of an epidural vessel occurs. Finally, although we feel that endotracheal intubation was the appropriate initial management (to prevent pulmonary aspiration in a parturient with a full stomach), the use of succinylcholine may have increased the cardiac effects of bupivacaine by increasing serum potassium.[6]

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