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Determination of Intravascular Migration of an Epidural Catheter Using the Air Technique

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EPIDURAL hematoma associated with epidural anesthesia and anticoagulation can cause irreversible neurologic sequelae. This complication may occur in patients in whom anticoagulation with intraoperative heparin after atraumatic epidural catheterization had occurred;[1] intraoperative intraarterial thrombolytic therapy may pose an additional risk.[2]

We report a case in which we injected a small amount of air into the epidural catheter to confirm our suspicion that an epidural catheter had migrated into a blood vessel of a vascular surgery patient who was about to receive intraarterial urokinase. By making an unequivocal diagnosis of catheter migration, we avoided the use of the thrombolytic agent and the potentially disastrous consequences of epidural hematoma.

Case Report

Our patient was a healthy 22-yr-old man with a history of claudication in his right leg after blunt trauma, who presented for operative reconstruction of his right popliteal artery with a saphenous vein graft. He had several uncomplicated general anesthetics in the past, and his only medical problem was an asymptomatic variant of β-thalassemia. Medications before surgery included naproxen 500 mg *bid*, and intravenous heparin, which had been discontinued the day before surgery. Preoperative laboratory results included a hematocrit of 40%, PT (prothrombin time) of 13 s (normal 10–14 s), and PTT (partial thromboplastin time) of 29 s (normal 24–36 s).

We planned to use a combination of epidural and general anesthesia intraoperatively, and to infuse dilute local anesthetic *via* the epidural catheter to provide sympathetic blockade postoperatively. After administering 800 ml of lactated Ringer's solution intravenously, we used a loss-of-resistance technique to insert a 20-G nylon multiorifice catheter (Portex, Keene, NH) at the L3–L4 interspace. Neither blood nor cerebrospinal fluid could be aspirated from the catheter, no paresthesias occurred, and two 3-ml test doses of lidocaine 1.5% with 5 μg/ml epinephrine did not cause tachycardia[4] or symptoms of local anesthetic toxicity. After administration of 6 ml 0.5% bupivacaine with 5 μg/ml epinephrine, the patient developed a sensory level of T-6 on the left and T-10 on the right.

We induced general anesthesia and muscle relaxation with 0.25 mg fentanyl, 350 mg sodium thiopental, and 10 mg vecuronium, and maintained anesthesia with 0.5–1.3% isoflurane in a 50% N_2O/O_2 mixture during the harvesting of the saphenous vein graft. An additional 3-ml dose of 0.5% bupivacaine with 5 μg/ml epinephrine administered during the dissection did not cause an increase in heart rate. We then turned the patient to the prone position for the vascular reconstructive procedure.

Shortly thereafter, we administered an additional 3-ml dose of 0.5% bupivacaine with 5 μg/ml epinephrine. This time, however, the injection was followed, within 20 s, by an increase in heart rate from 71 to 82 beats/min. Although suggestive of intravascular epidural catheter migration, this 11-beats/min increase in heart rate after epinephrine seemed inconclusive compared with the 32-beats/min mean maximum increase in heart rate described by Moore and Batra.[4] A repeat test with epinephrine-containing solution was similarly inconclusive. No change in blood pressure occurred after either injection. We were unable to aspirate blood from the catheter, even after

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flushing it with saline. The patient had just received 5,000 units of heparin intravenously, in preparation for the arterial repair, when the surgeon advised us that he was about to administer intraarterial urokinase to eliminate small, distal mural thrombi that he detected during an earlier popliteal arteriogram.

To definitively establish whether the catheter had migrated into an epidural vein, we injected air into the epidural catheter, as described by Leighton and Gross.[3] After discontinuing the N_2O, we applied a Medasonics D-8 Versatone Doppler probe (Mountain View, CA) to the patient's right precordium, and adjusted its position to maximize the Doppler heart tones. Within 10 s after injection of 1 ml of air *via* the epidural catheter, the Doppler heart tones changed to a roaring sound, indicative of intracardiac air. Having documented the intravenous location of the epidural catheter, we did not give urokinase, and were careful to maintain activated clotting time as close as possible to twice its control value.

Postoperatively, when results of the PTT indicated that the heparin effect had subsided, we removed the epidural catheter. Clotted blood was present in the distal 3 cm of the catheter. We inserted a new catheter at L 2–3 without incident, and established sympathetic blockade with a continuous infusion of dilute bupivacaine. This provided adequate sympathetic blockade, with no evidence of sensory or motor block; the patient never developed signs or symptoms of epidural hematoma. The second catheter was removed without incident 3 days postoperatively, and the patient made an uneventful recovery.

Discussion

Although the placement of epidural catheters is generally contraindicated after anticoagulation,[5] the preoperative placement of epidural catheters in patients who will receive heparin intraoperatively can be safely performed. Rao and El-Etr documented no major neurologic complications in 3,164 patients who received continuous epidural anesthesia for lower extremity vascular procedures with intraoperative anticoagulation with heparin. The investigators carefully monitored and controlled intraoperative anticoagulation, and excluded patients with a history of bleeding disorders, as well as those in whom epidural puncture was traumatic.[6] However, epidural catheterization and subsequent anticoagulation can result in epidural hematoma formation with the risk of serious neurologic complications, as described in several case reports.[1,2,7,8]

Despite proper technique and atraumatic placement, epidural catheters can migrate into epidural blood vessels.[9] Therefore, during continuous epidural anesthesia or analgesia, catheters should be tested for intravenous location, not only on placement, but also before each supplemental dose of local anesthetic.[4,10] Our inability to aspirate blood from the catheter could reflect partial or unidirectional obstruction of the catheter orifice(s) by the clot that we observed when the catheter was removed.

Although the results of our epinephrine-containing test dose indicated the possibility of intravascular catheter migration, the observed change in heart rate was equivocal: Moore *et al.* demonstrated, in volunteers and patients, that the administration of intravenous 15 μg epinephrine, with and without local anesthetic, resulted in a mean maximum increase in heart rate of 32 beats/min.[4] We injected air into the epidural catheter to support a definitive diagnosis of intravascular epidural catheter. From the surgeon's standpoint, the use of a thrombolytic agent was desirable to attempt to dissolve any residual thrombus present in the more distal arteries of the leg; from the anesthesiologists' standpoint, the concern was that systemic thrombolysis may cause epidural bleeding and hematoma.

At least two possibilities can explain the equivocal heart rate response to epinephrine in our patient. First, it is possible that the distal orifice of our multiorifice catheter migrated into an epidural vein, while the proximal orifices remained in the epidural space. Thus, only a fraction of the administered test dose entered the epidural vein. In a study of multiorifice epidural catheters, Ward *et al.* demonstrated this effect in an experimental model of the epidural space.[11] In fact, lack of response to epinephrine injected *via* epidural catheter has been demonstrated in nonanesthetized patients with multiorifice catheters radiologically proven to have been in epidural veins.[12] Second, patients may have a decreased response to catecholamines during general anesthesia.[13]

The intravenous injection of air poses the theoretical concern of paradoxical air embolism and subsequent neurologic or cardiac morbidity in patients with probe-patent foramina ovale. However, in adults, small air emboli often occur during routine intravenous therapy and drug administration, with no case reports or series describing any complications. In fact, Naulty *et al.* detected venous air emboli in 8 of 17 healthy laboring parturients during lumbar epidural catheter placement with an air "loss of resistance" technique; none developed clinically significant complications.[14]

Recent reports described epidural hematomas and irreversible neurologic sequelae after epidural anesthesia in patients receiving heparin anticoagulation and thrombolytic therapy.[2,15] The recently described air test of epidural placement enabled us to definitively establish the intravascular migration of an epidural catheter after standard tests proved inconclusive. As a result, we

did not administer thrombolytic agent intraoperatively; this may have prevented the devastating consequences of an epidural hematoma.

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Cardiopulmonary Bypass in Hereditary Angioedema

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AN absence of functional inhibitor of the activated form of the first complement component (C1INH) causes uninhibited activation of the complement cascade and the clinical picture of angioedema. Complement activation is seen in cardiac surgery, and is caused by a number of factors, including exposure of blood to the bypass circuit and administration of protamine.[1–3]

Twenty-five patients with hereditary angioedema undergoing 41 noncardiac operations were recently reported by Wall *et al.*[4] This constitutes the largest series of such patients to date. To our knowledge, only two previous case reports of patients with C1INH deficiency undergoing cardiac surgery with cardiopulmonary bypass exist in the literature. One patient died with evidence of unregulated complement activation after separation from bypass.[5] The other, a child with biochemical, but no clinical, symptoms of hereditary angioedema, underwent ASD closure uneventfully.† We report the case of a patient with hereditary angioedema

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