Incidence of Venous Air Embolism during Epidural Catheter Insertion

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Air embolization has been reported during many surgical and diagnostic procedures and as a consequence of intravenous therapy.¹⁻³ It can occur whenever the local venous pressure is less than the pressure in the air source.⁴ Bromage has suggested that these conditions may exist in patients undergoing the insertion of an epidural catheter.⁵

Therefore, we have attempted to systematically assess the incidence of detectable air embolus during epidural catheter insertion. This was accomplished using a widely employed, safe, non-invasive system of Doppler detection of minute quantities of intracardiac air.

MATERIALS AND METHODS

The patients admitted to the study were 17 healthy primiparous women (ages, 18–32 years), classified as ASA class 1 who received epidural anesthesia for labor and delivery. We excluded patients with: signs of toxemia of pregnancy, obstetric complications, congenital or acquired heart disease, or psychiatric problems. An intravenous infusion of lactated Ringer’s solution was started prior to the epidural puncture, and the amount of fluid infused before the procedure was recorded, as was the interval between the last oral intake of food and the epidural puncture.

The protocol was approved by The Human Research Committee of the Brigham and Women’s Hospital. Informed consent was obtained from the patients prior to the procedure, and the patients were placed in the left lateral decubitus position. A modified Brattle Electronics Model 101 ultrasonic Doppler transducer was placed over the fourth intercostal space along the right sternal border as described by Michenfelder et al.⁶ This position is usually optimal for detection of intravenous air embolism.⁷ In order to test the position of the Doppler, 5 ml of normal saline were rapidly injected intravenously. The Doppler positioning was deemed correct if a characteristic change in the Doppler heart sounds was noticed by one of the investigators (JSN). If such a sign was not noticed, the position of the Doppler was changed until the characteristic sounds were obtained on three consecutive injections.

With the patient still in the left lateral decubitus position, the placement of a catheter in the lumbar epidural space was accomplished by our standard epidural technique, utilizing the midline approach with a 17-gauge Weiss (winged) epidural needle through an intradermal skin wheal of local anesthetic. Entry into the epidural space was determined using the “hanging-drop” sign. When the epidural space was identified, or if the hanging drop sign was equivocal, the position of the needle was checked using a “loss of resistance” test with 5 ml of air. Once entry into the epidural space had been confirmed, the needle was turned so that the Huber tip was facing caudal and the loss of resistance sign repeated with 5 ml of air, rapidly injected (2 s). The epidural catheter was then passed 2 cm caudal beyond the tip of the needle, and the needle withdrawn over the catheter. The Doppler sounds were monitored during the entire epidural catheterization.

Two minutes following threading of the catheter into the epidural space, the position of the Doppler was again confirmed with a 5-ml rapid intravenous saline injection. If this confirmatory test was positive, the presence or absence of Doppler-detected embolization was recorded for the patient. If the characteristic change in the Doppler heart sounds was not observed in the second test, the patient was not admitted to the study. A continuous record of the patient’s electrocardiogram during the procedure was made, and the blood pressure was checked using the Riva-Rocci method with a mercury sphygmomanometer at one-minute intervals during the procedure. In addition, the occurrence of such signs as a “gasp” or hyperpnea were noted.¹⁴ If the presence of an embolus was noted on the Doppler, the heart was auscultated for a murmur. The patients were
divided into two groups as determined by the presence or absence of change in Doppler heart sounds, and the data were analyzed for statistical significance using Student's t test for parametric data testing and chi-square for non-parametric data.

RESULTS

The characteristic Doppler sounds of venous embolism were heard in eight of the 17 patients. In one patient, the Doppler detected an embolus during the "hanging drop" test. In this patient and six others, emboli were detected at least once during the "loss of resistance" test. In the one remaining patient, embolization was detected shortly after insertion of the epidural catheter. In this patient, symptoms of an intravascular injection (tinnitus and tremulousness) followed a 3-ml injection of 0.5% bupivacaine through the catheter, and blood was then aspirated from the catheter, which was subsequently removed. There were few symptoms noted in the patients in whom an air embolus was detected. One patient "gasped" at the time of embolization, and two patients had a short (less than one minute) period of mild hypotension (decline in MAP of 15 mmHg). In one of these hypotensive patients, the electrocardiogram revealed a 20-s period of frequent premature ventricular contractions. No murmurs were heard in any patient.

There were no significant differences between the two groups of patients in the mean values for height, weight, age, parity, and pre-epidural mean arterial pressure. There were, however, significant differences noticed in the mean values of the time since the patient's last meal (NPO status) and the amount of intravenous Ringer's lactate solution administered prior to the epidural anesthetic (table 1). The patients in whom embolism was detected had been NPO approximately twice as long as those in whom embolism was not detected, and had received significantly less intravenous fluid.

In addition, when the epidural needle was removed from the patient, blood was seen in the needle lumen in four of the eight patients in the "air detected" group while no blood was noticed in any of the "air not detected" group (table 1).

DISCUSSION

In this study, air emboli were detected during insertion of epidural catheters into the lumbar epidural space in eight (43%) of 17 healthy parturients. The necessary conditions for the occurrence of air embolism are an opening in the vein through which air can enter the vascular system, and a source of air with a pressure greater than that in the vein.

The first condition, an opening in a vein, can be created during epidural puncture. The epidural space is filled with a rich plexus of veins, which during pregnancy, are dilated secondary to chronic vena cava compression by the gravid uterus, with a subsequent increase in epidural venous blood flow. These veins are largely located lateral and anterior to the spinal cord, but are susceptible to trauma from the entering needle, particularly if the puncture is not direct in the midline, or if the needle is rotated while in the epidural space. In four of the eight patients in whom embolus was detected, blood was seen in the needle lumen upon its withdrawal, indicating that at some point during the puncture, a blood vessel was disrupted. In one patient, the catheter obviously was placed intravenously.

The second condition, an air pressure higher than the venous pressure, can also be fulfilled during epidural cannulation. The epidural venous plexus contains no valves, hence, in the lateral position, the epidural venous pressure probably closely follows the central venous pressure. Therefore, if CVP is low, pressure in the epidural plexus may be low as well. Our patients who experienced air embolism had been NPO approximately twice as long as those who did not, and had received approximately half as much intravenous fluid. These characteristics may have predisposed toward a decreased central venous pressure and increased the likelihood of air embolization. In addition, in the left lateral decubitus position, the site of epidural puncture is above the level of the right atrium, and uterine compression of the inferior vena cava is relieved, which would decrease epidural venous pressure and create a situation in which the epidural venous plexus may be subatmospheric. Thus, if the needle enters an epidural vein in this position, air embolism can occur, even during hanging drop testing, as suggested by Bromage. This reduction in the epidural venous plexus pressure may be exacerbated by wide fluctuations in intrathoracic pressure, as may occur during the rapid, gasping respirations occurring during parturition.

In seven of the eight patients in whom venous air was detected, the embolization occurred shortly (15–30 s) after the forcible injection of air during "loss-of-resis-
tance” testing. Usuniha has demonstrated that slow injections of local anesthetic solutions elevate the pressure in the epidural space 10 to 60 cm H₂O. This rise in pressure was more marked in pregnant patients. A rapid injection of air, although more compressible than a liquid, may sharply elevate air pressure in the epidural space to several times the venous pressure. If an opening in the vein has been created during the puncture, a bolus of air may be forced into the vein. It is conceivable that a sharp elevation of pressure may in fact disrupt venules and allow air embolization.

A precordial Doppler device is capable of detecting very small emboli (0.1 ml), but cannot quantify the amount of air which enters the circulation. All the emboli we have noted were of brief duration and were clinically insignificant. Adornato noticed significant symptoms in dogs when air was infused at rates of approximately 0.5 ml·kg⁻¹·min⁻¹. It is unlikely that such volumes of air would be introduced during epidural cannulation. However, paradoxical embolization of small amounts of air into the systemic arterial circulation (e.g., via a patent foramen ovale) may produce organ infarction and serious sequelae. Epidural anesthesia is used in cases of congenital heart disease with right-to-left intracardiac shunts, which could allow ready passage of air from the venous to the systemic arterial circulation, and in these patients, particular care should be taken to avoid the possibility of air embolism. This may be accomplished by careful hydration, performance of the puncture in the right lateral decubitus position, and using a loss of resistance test with saline or local anesthetic rather than air.

Neonatal Sepsis Presenting as Delayed Emergence from General Anesthesia

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Reasons for delayed emergence from general anesthesia in the newborn include hypothermia, metabolic disturbances, and several iatrogenic causes such as an overdose with muscle relaxants or anesthetics. Delayed emergence from general anesthesia is an unique sign of neonatal sepsis which may include variations in temperature, apnea, metabolic acidosis, seizures, respiratory distress, and metabolic derangements. The following case report stresses the importance of considering neonatal sepsis as a factor contributing to delayed emergence in a previously healthy full-term infant.

Report of a Case

This was a 2,750-g 39-week gestation female infant born to a 31-year-old healthy woman after an uneventful pregnancy. Labor was complicated by prolonged rupture of membranes for 19 hours and arrest of labor. The infant was delivered by cesarean section with spinal anesthesia with an one and five minute Apgar score of 2 and 7. With a FiO₂ of 1.0, ventilation as controlled via a mask and bag. Resuscitation was successful, and the infant was referred to our hospital.