Identification of the Epidural Space: Is Loss of Resistance to Air a Safe Technique?
A Review of the Complications Related to the Use of Air

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Background and Objectives. The major determinant of successful epidural anesthesia is the localization of the epidural space. The manual loss of resistance technique is widely used by anesthesiologists in identifying the epidural space. Should air or saline be used in detecting the point of loss of resistance? No consensus exists as to which technique is superior, and individual providers currently use the technique with which they are most comfortable. The incidence of adverse effects associated with the use of epidural air is unknown and may be underreported as the effects may be unrecognized or considered trivial. The authors comprehensively review the complications of epidural air from published reports.

Methods. Using the appropriate key words, the authors searched the Medline (National Library of Congress) scientific data bank from 1966 to 1995, for case reports of epidural complications.

Results. There are few prospective, controlled, double-blinded studies comparing the relative merits of using air versus saline for the loss of resistance technique of epidural placement. There are, however, numerous case reports. Complications associated with the use of air for the loss of resistance technique included pneumocephalus, spinal cord and nerve root compression, retroperitoneal air, subcutaneous emphysema, and venous air embolism. Additionally, inadequate analgesia and paresthesia have been associated with the loss of resistance technique using air. Transient and permanent neurologic sequelae have been attributed to some of the complications. The simultaneous administration of nitrous oxide and positive pressure ventilation has also been reported to expand localized collections of air, resulting in heightened symptoms.

Conclusions. The potential complications associated with the use of air for identifying the epidural space may not outweigh the benefits. The use of saline to identify the epidural space may help to reduce the incidence of these complications. Key words: epidural, anesthesia, air, complications, loss of resistance, saline.

Epidural anesthesia has been a part of anesthetic practice since 1901, when Sicard and Cathelin of France independently popularized the caudal approach (1). This technique has since undergone many modifications as a result of improvements in needle, syringe, and catheter technology, and as a result of advances in the pharmacology of local anesthetics and adjuvant medications. However, correct localization of the epidural space still remains the major determinant of successful epidural anesthesia. Several devices have been manufactured to assist in the identification of the...
epidural space (2), but the manual loss of resistance is still the preferred approach of most anesthestiologists.

The loss of resistance technique is based on the fact that the interspinous ligament and the ligamentum flavum are relatively dense tissues (3). As the tip of the epidural needle pierces the ligamentum flavum, there is an abrupt decrease in resistance and the contents of a well-lubricated syringe may be injected easily and smoothly through the needle and into the epidural space. Recently, a controversy has arisen as to the contents of the syringe. Should it be air or saline? There is currently no consensus as to which is superior. The decision as to which technique to use should not be based on bias and/or training alone but also on the relative possibility of complications that can occur with each technique. There have been no complications directly related to the use of saline, however complications associated with the loss of resistance technique using air have been reported. We review these complications in this paper.

**Pneumocephalus**

Most headaches subsequent to spinal or epidural anesthesia related to dural puncture appear 1–3 days after the procedure have been reported and persist 1–5 days (4,5). In 1979, Abram reported several headaches of a different character observed in 8 of 604 patients undergoing epidural anesthesia (6). These headaches occurred shortly after the procedure, occurred only when the patient sat up, and were partially relieved by reclining. In all cases, the headache followed dural puncture during attempted epidural placement using air for the loss of resistance technique. We have identified 13 similar case reports of pneumocephalus resulting from the use of the loss of resistance to air technique. Table 1 provides a summary of the cases. The amount of air utilized ranged from 2 to 20 mL. In two of the case reports (8,9) the details of the technique were not provided while in another (14) the volume of air was not stated. Six of the cases specifically reported an inability to aspirate during placement of the epidural needle (6,7,11–13,16). While some patients were asymptomatic (9) others suffered from severe headache, nausea and/or vomiting, generalized seizures, and/or hemiparesis (8). In the case described by Katz (10) pneumocephalus was believed to be responsible for the delayed awakening in a patient who had total spinal anesthesia after an attempted epidural anesthesia and received nitrous oxide in oxygen as part of sedation. Pneumocephalus was radiologically confirmed in 12 of the 13 cases. Headache is a symptom common to both pneumocephalus and dural puncture. However, the onset of headache is typically immediate or at least, earlier with pneumocephalus. It is important to differentiate between the two types of headache, because treatment differs. A computed tomography (CT) scan is diagnostic for air within the cranium, and management is usually symptomatic. Administration of 100% oxygen often reduces the severity of the headache (11,13,16). In contrast, administration of nitrous oxide makes the headache worse in a patient with pneumocephalus as it may result in a threefold increase in subarachnoid pressure (17).

**Spinal Cord and Nerve-Root Compression**

Neurologic defects following epidural anesthesia are fortunately rare, but they can occur due to spinal cord compression from hematoma (18), trauma to the spinal cord or roots, vascular compromise, and coexisting transverse myelitis (19). Table 2 summarizes four cases, one case related to us by K. Rothenberg, S. Lipman, A. Shander, and E. Fredman (case presentation interview, 1993), of spinal cord compression as a result of the use of air in identifying the epidural space (20–22). Symptoms ranged from sharp excruciating pain to motor weakness, loss of sensation, and paraplegia. In these cases, CT scan showed air impinging on the spinal cord at a level corresponding to the pattern of neurological deficit. Kennedy et al. described nerve root compression from accumulation of air in the epidural space in a patient receiving a continuous infusion of opioid and local anesthetic for the treatment of cancer pain (23). The patient had repeatedly silenced the alarm that signaled the presence of air in the pump system and purged the air into her epidural catheter. Cuerden et al. described delayed recovery after epidural analgesia in four obstetric patients in whom loss of resistance to air was used to identify the epidural space (24). The neurologic defects described included sensory loss, paresthesia, motor weakness, decrease in muscle tone, and diminished deep tendon reflexes of the lower limbs. One of the patients had an increase in muscle tone and exaggerated knee and ankle reflexes, suggesting an upper motor neuron lesion. Fortunately, all patients made a full recovery within 48 hours. The authors felt that the neurologic deficits in these patients were compatible with spinal cord compression from air which was
then reabsorbed over the next 24–48 hours. No imaging studies were performed to confirm the diagnosis in these cases.

The use of air in identifying the epidural space may have contributed to the development of a cauda equina syndrome in a patient who subsequently received nitrous oxide as part of general anesthesia for a radical retropubic prostatectomy (25). Although the postoperative magnetic resonance imaging was noncontributory, it is possible that the air which caused the cauda equina lesion had been absorbed. Neurological deficits were still present 12 months postoperatively.

**Subcutaneous Emphysema**

There have been nine published case reports of subcutaneous emphysema as a result of loss of resistance to air in identifying the epidural space (26–31), with subcutaneous emphysema observed in the supraclavicular (29), cervical (27,30,31), thoraco-lumbar (28), and abdominal (20) regions. A common factor in these cases was multiple attempts, using upwards of 20 mL of air, before the epidural space was identified. The main clinical feature was crepitus, and in all cases the subcutaneous air was reabsorbed over several days. In the case described by Viel et al. the presence of subcutaneous emphysema in a patient with a diagnosis of severe acute pancreatitis led to misdiagnosis as an anaerobic soft tissue infection (28). The epidural catheter was removed prematurely in spite of effective analgesia. In one of the cases described by Carter the author felt that presence of air in the extradural space probably contributed to a less effective extradural anesthetic block (27). Potential clinical problems that may arise as a result of subcutaneous emphysema include extrinsic airway compression and air embolism (via the valveless vertebral venous plexus of Baston), both of which could become more serious with the use of nitrous oxide.

**Epidural Air and Extracorporeal Shock Wave Lithotripsy**

Palmer and Norman reviewed 1,344 patients that underwent extracorporeal shock wave lithotripsy (ECSWL) (32) and noted 8 patients who had retroperitoneal air on plain radiographs performed after the procedure. Use of air in locating the epidural space was common to all 8 patients. In 2 patients, radiographs taken after the epidural but before ECSWL also showed retroperitoneal air, indicating that epidural placement was the most probable source of the retroperitoneal air. Air appeared to have migrated along spinal nerves in radiographs of 5 patients while in 3 it appeared localized within tissue planes.

Deam and Scott reported a neurological deficit following ECSWL in a patient who had an epidural placed using 2 mL of air for a loss of resistance technique (33), and again CT scan revealed an air loculus in the right lateral recesses of S1 and T12. The neurological deficits gradually improved over the next 18 days, although she still had residual backache and some limitation of straight-leg-raising at 6 weeks. Roberts et al. noted interstitial emphysema on abdominal radiographs in 38 (15%) of 150 patients treated with ECSWL (34), and all 38 patients had undergone epidural anesthesia with the loss of resistance to air technique. The interstitial emphysema resolved over several days. Abbott et al. in their paper on anesthesia for ECSWL stated the following: “Anesthetists should pay particular attention to the problems associated with epidural anesthesia. If the loss of resistance to air technique is employed to locate the epidural space, air may spread into the paravertebral spaces and along the paths of adjacent nerves. The presence of air-water interfaces, especially in the epidural space, may result in trauma to the spinal cord and nerves. We therefore believe that the loss of resistance to saline method is mandatory (35).” They also stated that epidural air might have been responsible for transient paresthesia in one of their patients. Furthermore, concomitant administration of nitrous oxide and intermittent positive-pressure ventilation may result in expansion of air within tissue planes, resulting in additional tissue trauma.

**Venous Air Embolism**

Venous air embolism (VAE) is a well-recognized complication of many surgical and diagnostic procedures (36–38). A rich plexus of veins, largely located lateral and anterior to the spinal cord, occupy the epidural space. These veins are valveless, so when a patient is in the lateral position, epidural pressure closely approximates central-venous pressure (40). Factors predisposing to development of VAE include a tear in a vein and perivascular pressure that is higher than venous pressure. Thus, unrecognized epidural-venous puncture with rapid injection of air used for loss of resistance may result in VAE. There have been two pediatric case reports that draw attention to the hazard of VAE during initiation of epidural anes-
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<td>6</td>
<td>56-year-old woman, epidural steroid for radiculopathy</td>
<td>L4-5 interspace, LOR to 3 mL of air. Negative cerebrospinal fluid aspiration. 5 mL 1% lidocaine + 25 mg triamcinolone.</td>
<td>Mild frontal headache. Progressed to severe generalized headache on sitting up. Partially relieved by supineposition. Duration: 1 h.</td>
<td>Upright lateral skull x-rays revealed very small amounts of intracranial air probably in the basal cisterns.</td>
<td>Nil.</td>
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<td>6</td>
<td>50-year-old woman, epidural steroid for lumbar radiculopathy</td>
<td>L2-3 interspace, LOR to 3 mL of air. Reconfirmed by injection of a few additional mL of air. Negative cerebrospinal fluid aspiration. 8 mL 0.5% Lidocaine + 50 mg triamcinolone.</td>
<td>Sensory anesthesia below T10, partial motor block lower extremities. Sitting up 1 h later resulted in immediate, severe, generalized headache, nausea and vomiting. Duration: 3 h.</td>
<td>Upright lateral skull x-ray. Small quantity of air in basal cisterns and peripheral subarachnoid space.</td>
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<td>7</td>
<td>33-year-old woman, epidural analgesia for labor and delivery.</td>
<td>L3-4 interspace, LOR to saline and 2 mL of air. Inadvertent dural puncture recognized. Rectified at L2-3 space with LOR to saline.</td>
<td>Immediate mild frontal cephalgia with the first needle placement. Headache exacerbated by elevation of the head.</td>
<td>No imaging studies.</td>
<td>Pretreated with 40 mL of NaCl via epidural catheter with immediate relief.</td>
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<td>7</td>
<td>31-year-old woman, epidural analgesia for labor and delivery.</td>
<td>L3-4 interspace. Epiduralspace localized after two attempts and total of 9 mL of air injected. Negative cerebrospinal fluid aspiration. Epidural needle removed because of symptoms. Analgesia provided using N₂O in oxygen via mask.</td>
<td>Severe fronto-occipital headache upon epidural entrance associated with paresthesia in the right leg.</td>
<td>No imaging studies.</td>
<td>Headache persistent and on the third epidural analgesia for postpartum day an epidural blood patch was performed with resolution of symptoms.</td>
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<td>8</td>
<td>64-year-old woman, epidural anesthesia for amputation of toes without apparent complications intraoperatively.</td>
<td>Details not provided.</td>
<td>Postoperative severe headache, nausea, and vomiting. Discharged on day 4 while symptomatic. Readmitted 48 h later via emergency room with headache, vomiting, confusion, and dehydration. She was obtunded, dysarthric, and had four generalized seizures with a right focal onset. There was flaccid right hemiparesis, right central facial weakness, absent gag reflex, decreased d-p tendon reflex on the right and positive Babinski sign on the right foot. Electroencephalogram—Diffuse slowing, more marked over the left hemisphere.</td>
<td>Two areas of low density in the posterior fossa which appeared to be air in the subarachnoid space. This was confirmed on skull films. No other abnormalities were found.</td>
<td>Conservative medical management with rehydration and anticonvulsant therapy. Headache, nausea, and vomiting resolved but required vascular surgical service for continued pain and ischemic changes in the lower extremities.</td>
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<td>9</td>
<td>58-year-old woman. Extracorporeal shock wave lithotripsy with aid of epidural anesthesia.</td>
<td>Details not available. One day prior to discharge, she fell out of bed, striking her left temporal area. Neurological examination was normal. A skull film was obtained to rule out a skull fracture. Skull film revealed a partial air ventriculogram with no evidence of orbital, basal skull fracture or sinus air fluid level. This was confirmed on computed tomography scan. Pseudocerephalus was attributed to the epidural.</td>
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<td>10</td>
<td>25-year-old woman. Epidural anesthesia for cesarean section.</td>
<td>Multiple attempts with the LOR test with repeated injections of air (estimated total of 20 mL). 16 mL bupivacaine hydrochloride and 1:200,000 epinephrine injected into the epidural space. Immediate cessation of respiration and hypotension. She was treated with tracheal intubation, IPPV, crystalloids, and vaso-pressors. Following delivery of the baby, anesthesia was maintained with 66% N2O in oxygen, 10 mg diazepam, and 0.2 mg fentanyl. 4 h later, patient remained drowsy, stuporous, and had involuntary movements even though spontaneous respiration had returned. Computed tomography scan showed a large subarachnoid air-filled cavity in the parieto-frontal cerebral cortex with an estimated volume of 25 mL. Neurological status improved by the next day. She was discharged on third day postpartum.</td>
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<td>11</td>
<td>29-year-old woman. Epidural analgesia for labor and delivery.</td>
<td>There was difficulty localizing the epidural space due to repeated contact with the bone. On the 4th attempt, 5 mL of air was injected at L2-3 space at loss of resistance. No cerebrospinal fluid was observed. Epidural needle was removed because of symptoms. A general anesthetic without N2O was subsequently administered because of persistent occipito posterior position. Immediate complaint of severe bifrontal headache with occipital radiation. Several episodes of violent emesis followed. There was no neurological deficit alteration in her level of consciousness. Computed tomography scan revealed air in the ventricles. Subarachnoid space and basal cisterns. Headache resolved by the first postoperative day and she was discharged on day five.</td>
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<td>12</td>
<td>77-year-old woman with lumbar stenosis. Epidural for low back and bilateral leg pain.</td>
<td>Epidural space identified with loss of resistance to 3 mL of air at the first attempt. A further 6 mL of air was injected in 3 mL increments to confirm needle placement due to symptoms. Following negative aspiration and a test dose, 8 mL 0.125% bupivacaine and 3 mg betamethasone was injected. Immediate onset of bifrontal and bitemporal headache. Nausea, dyspnea, shallow respiration, hypotension, bilateral upper extremity weakness with a sensory level of C6. Discharge after 2 h with residual temporal headache. 12 h later, the patient presented in emergency room with persistent headache. Plain skull x-rays revealed subdural air at the apex of the tentorium. Conservative management with analgesic medications. Headache resolved after 5 d.</td>
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<td>13</td>
<td>37-year-old woman with right lower extremity radicular pain due to herniated L4-5 disc.</td>
<td>Epidural space found at L4-5 level after two attempts. Total of 4 cc of air was used for loss of resistance. Cerebrospinal fluid could not be aspirated. 5 mL 1% lidocaine + 50 mg triamcinolone was injected into the epidural space.</td>
<td>Near the end of injection of lidocaine, a peculiar sensation like the passage of a bubble between the scapula up to the base of the skull, staying to the left of the midline was described. It was immediately followed by a severe hatcher like pain in the occiput.</td>
<td>Skull x-rays 30 min after the procedure revealed air over the clivus.</td>
<td>Oxygen was given by face mask, pain abated over 45 min. There was no long-term sequelae.</td>
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<td>14</td>
<td>45-year-old woman. Epidural injection for chronic low-back pain.</td>
<td>Unintentional dural puncture took place at the first attempt with LOR to air. Repeat attempt at same space was successful. Air was repeatedly injected to check accurate placement of the needle. 120 mg of triamcinolone acetate in 10 mL of saline was injected.</td>
<td>She developed severe frontal temporal headache without radiation and not dependent on position. It was associated with dizziness, pallor, bradycardia, and hypotension.</td>
<td>Cranial x-rays obtained 4 h later revealed pneumocephalus.</td>
<td>Intravenous crystalloid, vasopressors, and caffeine. Headaches improved, and she was discharged the next day. There was no neurologic deficit follow-up 6 months later.</td>
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<td>15</td>
<td>29-year-old pregnant woman in preterm labor had diagnostic lumbar puncture for sudden onset of severe headache. Computed tomography scan and cerebral angiogram were negative even though cerebrospinal fluid was blood stained. She developed symptoms of PDPH which did not respond to conservative management. An epidural catheter was placed for continuous saline infusion to treat PDPH.</td>
<td>Sedation was provided with midazolam 2 mg and fentanyl 100 mg. Epidural space was identified at the first attempt at L3-4 space using loss of resistance to 3 mL of air. A test dose of 3 mL 1.5% lidocaine + 1:200,000 epinephrine and a 30 mL bolus of saline was injected, followed by a 30 mL/h infusion of saline. The infusion of saline was discontinued.</td>
<td>50 min after the procedure, she complained of a severe frontal headache which was different from her presenting PDPH.</td>
<td>Computer tomography scan revealed an estimated volume of 12-15 mL of air in the cranial cavity.</td>
<td>Headache resolved spontaneously over 2 h.</td>
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<td>16</td>
<td>30-year-old woman. Epidural analgesia for labor and delivery.</td>
<td>L3-4 interspace, LOR to 5 mL of air. Space identified easily. Negative cerebrospinal fluid or blood aspirate. The needle was removed from her back due to the severity of the symptoms. She required cesarean section 8 h later, and a general anesthetic without N₂O was administered.</td>
<td>Immediate onset of severe backache, posterior neckache, approximately and bifrontal-temporal headache. She became uncooperative, vociferously complaining that she was experiencing the worst headache of her life. Neurologic examination was within normal limits.</td>
<td>Computed tomography scan revealed 5 mL of air in the intracranial subarachnoid space and the basal cisterns.</td>
<td>Following emergence from general anesthesia, she noted a decrease in the intensity of the headache which resolved completely the following morning.</td>
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LOR, loss of resistance; IPPV, intermittent positive pressure ventilation; PDPH, postdural puncture headache.
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<td>20</td>
<td>40-year-old woman with degenerative L5-S1 disc disease presented with disabling radiating pains associated with marked motor weakness and sensory loss in the right leg.</td>
<td>Epidural catheter placed at L3-4 and 40 mg methylprednisolone acetate and 4 mL 0.5% bupivacaine was injected with relief of right leg pain. Bupivacaine was injected every 4 h. The catheter was occluded twice within 3 d and was replaced.</td>
<td>The patient complained of increasing motor weakness and numbness in the left leg despite pain relief in the right leg. The patient also experienced headaches and hypersensitivity in the upper extremities.</td>
<td>Computed tomography scan of the spine showed a fairly large amount of posteriorly located epidural air displacing the thecal sac anteriorly in the cervical and thoracic spine and to a lesser amount in the lumbar area.</td>
<td>The catheter was removed and there was a gradual disappearance of neurologic symptoms and signs. A right discectomy at L5-S1 was performed 1 week later.</td>
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<td>21</td>
<td>64-year-old man with adenocarcinoma of the prostate with bony metastasis required epidural analgesia.</td>
<td>Temporal epidural catheter was sited at T4 using LOR to 5 mL of air. A permanent catheter was placed at the same level 3 d later following two attempts using LOR to 10 mL of air. Correct placement was confirmed intraoperatively fluoroscopy and nonionic contrast material.</td>
<td>Attempt to inject 30 mL of morphine solution (1 mg/mL) met with sharp excruciating pain across the left shoulder, chest, and arm.</td>
<td>Spot radiograph using contrast revealed serpiginous and nodular filling defects at T1-2. Computed tomography scan showed air in the epidural space dorsal to the dural sac. The catheter was noted to be in the dorsal space.</td>
<td>The morphine solution was concentrated to 10 mg/mL and the patient received this without sequelae. Review at 8 months showed that the catheter was still in place and he was comfortable on the same dosage.</td>
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52-year-old woman with long-standing severe back pain requested general anesthesia for placement of epidural steroids and local anesthetic. Anesthesia was given using propofol and 70% nitrous oxide in oxygen. Using a 16-gauge Tuohy needle, four separate attempts were made using LOR to air before satisfactory placement. Approximately 40 mL of air was employed during the procedure. 20 mL of 0.25% bupivacaine and 80 mg methylprednisolone in 20 mL normal saline was injected through the epidural catheter which was then removed.

8 h after the procedure, the patient complained of persistent numbness in the left leg. There was a left sided L2 sensory deficit with decreased power of left hip, knee, and ankle flexion with absent left knee jerk. Computed tomography scan revealed a large amount of air in the extradural space which appeared to be displacing the cauda equina.

Intravenous dexamethasone was given and the sensory and motor deficit completely resolved. The tomography scan 3 d postprocedure showed substantial resorption of extradural air. The patient remained pain free with no further neurologic symptoms.

Personal communication with K. Rothenberg, S. Lipman, A. Shander, and G. Fremen.

75-year-old woman with radicular pain from spinal compression fractures had an epidural catheter sited for pain relief. Using fluoroscopy, an initial attempt at T9 level using the hanging drop technique followed by LOR to 2 mL of air was unsuccessful. A repeat attempt at L1 was successful. The catheter was threaded up 5 cm, 5 mL of 0.25% bupivacaine and 50 µg fentanyl followed by depomedrol 80 mg in 5 mL of normal saline was injected in to the catheter.

In the PACU, the patient complained of pain in the thoracic region which was similar to her preprocedure pain. She was given another bolus of 50 µg fentanyl and 5 mL of 0.25% bupivacaine on two occasions resulting in good pain relief. 2 h after the procedure, she developed weakness in her legs. Neurologic evaluation revealed paraplegia with a T6–7 sensory level.

Computed tomography scan revealed a focal collection of air in the subdural space at T6 with marked compression of the adjacent thoracic cord. Computed tomography scan guided aspiration of subdural air was performed by the radiologist using a 19-gauge needle. Approximately 2 mL of air was aspirated with a decrease in the air volume and a resumption of the spinal cord to a more normal contour. The neurological condition improved initially but deteriorated over the next few hours resulting in significant deficit despite a large steroid dose.

LOR, loss of resistance; PACU, postanesthetic care unit
thesia (40,41). These children were administered approximately 0.4 mL and 0.2 mL air. The authors described a similar sequence of events in both cases: the rapid onset of acute cardiovascular deterioration following forceful injection of air through a misplaced needle and catheter into the lumen of an epidural vein. Fortunately, prompt cardiopulmonary support resulted in the uneventful recovery of both children. A similar case of suspected venous air embolism has been reported in a 40-year-old man undergoing cervical epidural steroid injection using the hanging drop technique (42).

Naulty et al. demonstrated Doppler evidence of intracardiac embolism in 8 out of 17 (46%) patients in whom epidurals were performed using the loss of resistance to air technique (43). In one patient, the Doppler detected an air embolus during the hanging drop test, in six patients the emboli were detected during loss of resistance to air, and in the other patient embolization was detected shortly after insertion of the epidural catheter, which was later found to be intravascular.

The volume or rate of air administration necessary to produce significant physiologic alteration in conscious or anesthetized human patients has not been quantified (44) however, it has been demonstrated that the lowest volume of air required to produce cardiorespiratory instability in dogs is 0.5 mL/kg/min (45). Although a large volume of VAE is necessary to produce right ventricular dysfunction and significant hemodynamic changes, a relatively smaller volume of air reaching the arterial circulation through an intercardiac communication (paradoxical embolus via a patent foramen ovale) can occur. The foramen ovale may open intermittently and allow right to left shunting during crying in 50% of infants during the first week of life. A probe patent foramen ovale exists in 50% of children up to 5 years of age (46) and in 25–30% of adults (47). Gronert et al. described death in one patient and permanent neurological deficit in another during neuromuscular paralysis complicated by paradoxical air embolism via a patent foramen ovale (48). Administration of 50% nitrous oxide in the presence of VAE appeared to increase the size of the air embolus to more than twice its original volume, resulting in significant cardiorespiratory changes (49).

Incomplete Analgesia and Paresthesia

Two cases of incomplete analgesia following identifications of the epidural space by the loss of resistance to air were described by Dalens et al., and in both cases, the location of air bubbles on radiographs of the spinal column corresponded with the dermatomal areas in which there was incomplete analgesia (50). A similar case report of epidural air bubbles causing unblocked segments also was described by Boezart and Levendig (51). Stevens et al., in a carefully controlled radiographic study, were able to demonstrate epidural air bubbles when 3 mL of air was injected into dogs the epidural space (52). The air bubbles were mainly distributed in the region of upper lumbar and lower thoracic vertebrae; but some were seen near the intervertebral foramina, where the nerve roots exited the epidural space. Judging from the small size and scarcity of the epidural bubbles seen on the radiographs, they were not able to condemn the use of a small volume of air in performing the loss of resistance test. However, the authors suggested that the use of more than 3–4 mL of air should be avoided, particularly in patients for whom nitrous oxide is to be used as part of the anesthetic.

A prospective study comparing the incidence of paresthesia in loss of resistance to air versus saline was carried out by Sarma et al. in obstetric patients (53). There were no significant differences in the number of unblocked segments or in the incidence of paresthesia between the two groups, but their sample population of 67 patients was rather small. Tanaka et al. retrospectively analyzed the incidence of complications following epidural anesthesia in 17,439 patients in whom a loss of resistance to saline technique was employed (54) and they found the incidence of paresthesia to be 0.16%. This was a much lower incidence than the 48% reported by Jaucot, who also used saline but incorporated a small air bubble within it (55).

Discussion

Sicard and Forestier introduced the loss of resistance technique using a syringe full of fluid as “un madarin liquide”—that is, a liquid stylet (56). Dogliotti, in the first detailed description of peridural anesthesia in 1933, also used physiologic saline as the locating injectate (57). Over the subsequent 60 years many anesthesiologists substituted a compressible gas (air) for the saline, and this report is presented to indicate that this change carries with it many possible complications, most minor, but some potentially serious.

The volume of air currently used to identify the epidural space with the loss of resistance technique varies widely among anesthesiologists. There is a tendency for a larger volume to be used by less experienced practitioners, particularly when tech-
technical difficulties are encountered in confirming placement of the epidural needle. The volume of air that can be safely injected into the epidural space has not been established and should be determined if air continues to be widely utilized. It is obvious that repeated injections of large volumes of air are more likely to be associated with complications than a single small volume injection, but even when as little as 3 cc has been used, pneumocephalus has been reported to occur (15). Furthermore, clinical features suggestive of VAE have been described during performance of a cervical epidural using the hanging drop technique (43). It was postulated by the authors that prior neck surgery with subsequent surgical scarring could have resulted in a venous malformation with noncollapsible side walls. The presence of a 16-gauge needle in the venous space could have resulted in a positive hanging drop sign with subsequent air entrapment. The potential for air entrainment is further supported by Naulty et al. who demonstrated Doppler evidence of intracardiac air embolism in one patient during the hanging drop technique (43). It is therefore unlikely that a stipulated volume of air can be considered safe to inject into the epidural space, especially in the presence of an unintentional vascular or dural puncture.

Perhaps it is time for us to reconsider our technique of placing epidural needles and catheters. Anesthesiologists are adept at placing central venous catheters with care to avoid VAE. A finger placed over the hub of the epidural needle following disconnection of the syringe and before insertion of the catheter might decrease the possibility of air entrapment into the epidural space, particularly at a cervical level. In discussing the relative merits of a fluid-filled versus an air-filled system, Bromage pointed out that the fluid-filled system is theoretically ideal because the fluid is incompressible: thus, the transition from complete resistance to no resistance is sudden and convincing and provides a crisp and unequivocal end point. The main disadvantage of the loss of resistance to saline technique is that inadvertent dural puncture is less-easily recognized, though unrecognized subarachnoid injection can also occur with the use of air (10). Finally, it has been suggested that the injection of a stream of fluid ahead of the advancing needle pushes the dura away from the needle tip, thus reducing the possibility of dural puncture (58). Anesthesiologists who prefer the loss of resistance to air technique usually do so because they like the sensation of "compressibility" afforded by the column of air within the syringe. Candido and Winnie have described a dual-chambered syringe that allows identification of the epidural space using the loss of resistance to both air and saline (58). The distal chamber contains saline which is injected upon entry into the epidural space, while the proximal chamber contains air which is retained within the syringe. This device may represent the best of both worlds, providing the sensation of "compressibility" without the risks associated with the injection of air into the epidural space.

Conclusion

Complications resulting from the injection of air into the epidural space include pneumocephalus, spinal cord and nerve root compression, retroperitoneal air collection, subcutaneous emphysema, venous air embolism, and, possibly, incomplete analgesia and paresthesia. The cases described indicate that while rare, complications associated with injection of air do occur. It is likely, however, that subclinical presentations occur more regularly and only become clinically significant when there are other associated factors that can produce physiologic compromise, such as a reduction in the compliance of the central nervous system, low-venous pressure and/or the presence of a patent foramen ovale.

Many of these cases also demonstrate that the concomitant administration of nitrous oxide may cause or exacerbate the clinical manifestation of these complications by increasing the size of the entrapped air bubble. It would appear to the authors that since the use of air for the loss of resistance technique offers no advantages over the use of saline, the possibility of the complications described above, no matter how remote, speaks in favor of the use of saline and the abandonment of the use of air for this valuable technique.

References


33. Deam RK, Scott DA. Neurological damage resulting from extracorporeal shock wave lithotripsy when air is used to locate the epidural space. Anesth Intens Care 1993: 21: 455–457.


