oroscopic control with contrast, and spread appears as in figure 1, intralymphatic injection may be differentiated from intravascular injection by the lack of washout within a period of seconds as one would expect if arterial or venous injection was made.

References


**Paraplegia after Continuous Subdural Meperidine Infusion**

Graf Hilgenhurst, M.D.,* Andrew W. Sukinnik, M.D.,† Mary L. Anderson, M.D.,‡ W. Heinrich Wurm, M.D.§

NEUROLOGIC injury, including paraplegia, continues to be a rare but devastating complication of epidural anesthesia. In most cases, the mechanism of the injury remains unexplained. We report a patient who developed an incomplete mixed sensory and motor neurologic deficit after an epidural catheter was used to provide surgical anesthesia and postoperative pain management.

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**Case Report**

A 63-yr-old woman was scheduled for a hysterecomy and resection of a mature teratoma. Because of severe bronchial asthma, an epidural anesthetic was chosen. A 17-G Weiss needle was placed using a loss-of-resistance technique to introduce an 18-G bullet-tipped side-holed polyamide catheter. Placement of the catheter was technically difficult, and blood was encountered on the first three attempts. The fourth attempt was successful at L1–L2, with no blood or cerebrospinal fluid on aspiration of the catheter. After a test dose of 3 ml 2% lidocaine with epinephrine 1/200,000, anesthesia was achieved at level of T4 bilaterally with 12 ml 2% lidocaine with epinephrine, and surgery was begun. A total of 12 ml 0.5% bupivacaine was given over 3.5 h. Blood pressure was labile and treated with fluids and phenylephrine infusion. Preoperative blood pressure had been 140/80 mmHg. Phenylephrine infusion was begun at 85/40 and titrated to maintain 110–130/70/80. This was tapered and discontinued in the postanesthesia care unit, and an epidural infusion of preservative-free meperidine 0.2% was begun at 7 ml/h.

On the morning of the first postoperative day, the patient was moving uneventfully with assistance from bed to chair. At 12:00 pm she complained of inability to move her legs. This was not accompanied by any pain in her legs or back. She was evaluated and found to have motor weakness from the level of the hip flexors down, with loss of sharp and dull discrimination below the knees and sharply diminished reflexes at the knee and ankles bilaterally. The epidural infusion was discontinued, and 7 ml blood-titled fluid was aspirated from the catheter. Laboratory studies at that time revealed normal prothrombin time, partial thromboplastin time, platelet count, and bleeding time. The catheter was left in place. The Department of Neurosurgery was consulted, and a magnetic resonance imaging scan of the thoracic and lumbar spine was obtained. This revealed multiple compression fractures of thoracic and lumbar vertebral bodies. A fluid collection surrounding the thecal sac was identified from T4 to L5 (fig. 1). Multiple air bubbles were present within the fluid collection. The fluid and air collection was in the subdural space.

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* Assistant Professor of Anesthesia.
† Instructor of Anesthesia.
‡ Assistant Professor of Radiology.
§ Associate Professor and Chairman of Anesthesia.

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Address reprint requests to Dr. Hilgenhurst: Department of Anesthesia, New England Medical Center Hospital Box 298, 750 Washington Street, Boston, Massachusetts 02111.

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external to the cerebrospinal fluid in the subarachnoid space, but did not occupy the epidural space (fig. 2). The fluid was low-signal on T1-weighted images and was high-signal on T2-weighted images. The signal characteristics were not consistent with a hematoma. The air was low-signal on both T1- and T2-weighted images. The catheter was not identified and was removed intact that afternoon. The Department of Neurology recommended immediate decompression. The patient refused further surgery.

The patient's neurologic status did not improve, and a computed tomographic myelogram was scheduled and performed 2 days later. Intrathecal contrast was placed via a C1–C2 puncture because of the abnormal appearance of the lumbar spine. This study demonstrated an unchanged spinal fluid and air collection and confirmed the subdural location of this collection (fig. 3). Multiple compression fractures were noted from T4–T5 to L3, with maximal distortion at T12. The appearance of the fluid showed a derangement of normal structure, suggesting marked cord compression from T4 to L3, particularly in the lower thoracic region. A second magnetic resonance imaging scan obtained 1 week later revealed a resolving fluid collection and a new increased T2 signal in the spinal cord from T7 to L1, consistent with cord edema or infarction.

At the 1-month follow-up examination the patient continued to have mixed sensory and motor deficit characterized by loss of bowel and bladder function (which now is resolving), essentially absent motor function below the hip flexors, and loss of sharp and dull sensations below the knees bilaterally. Reflexes were absent at the knees and ankles. Sensory and motor losses were more pronounced on the right side. Proprioception remained intact bilaterally, and vibration sense was intact on the left but not on the right. At the 6-month follow-up the patient was wheelchair-bound.

Discussion

Fortunately, lasting neurologic sequelae after epidural anesthesia are rare. The incidence is variously given as 1 in 10,000,5 1 in 11,000,6 or 1 in 20,000.7 Any of numerous mechanisms can and do lead to injury. In patients with preexisting blood dyscrasias, cord compression caused by hematoma appears to be the most common cause.1 An underlying abnormality of the spine seems to be associated with many cases not involving hematoma.5–8,16 Perhaps these patients are more susceptible to a compromise in spinal cord blood flow caused by increased pressure from the injection of epidural fluid. Other possible causes include arachnoiditis, direct trauma to the spinal cord, prolonged hypotension, patient positioning, and surgical trauma.1–4 Injection of neurotoxic or contaminated substances has rarely occurred.9,10

The current case is further complicated by the subdural (but not subarachnoid) appearance of the fluid. It is well known that catheters may be placed or migrate subdurally, resulting in widespread sensory or sympathetic block after a small injected dose of local anesthetic.11 This was not seen in our case. Although magnetic resonance imaging did not support the di-
agnosis of an epidural hematoma, the catheter was aspirated before removal, and the fluid was blood-tinged. This raises the possibility of a spinal subdural hematoma. A spinal subdural hematoma is clinically indistinguishable from an epidural hematoma, although it is extremely rare: according to Reynolds et al., only 30 reports exist in the world literature. Unlike epidural bleeding, the exact source of bleeding is unclear.

We believe that problems in this case started when the tip of the epidural catheter migrated into the subdural space on the day after surgery. This is corroborated by the uneventful recovery from surgical anesthesia. Meperidine was infused into the subdural space at a rate of 7 ml/h. The capability of the subdural space to absorb and redistribute fluid is unknown. This patient suffered from osteoporosis with compression fractures of several thoracic vertebrae. Narrowing of the spinal canal may have limited the spread of the fluid column in the subdural space. Although resistance to infusion was not measured, it is reasonable to surmise that there was a gradual increase in pressure exerted by the subdural fluid collection, resulting in direct compression of the thoracic spinal cord, increased resistance to venous drainage from the cord, or arteriolar compromise. One or all of these factors may have contributed to the painless paraplegia resembling anterior spinal artery syndrome both clinically and as seen by magnetic resonance imaging. It is unclear whether the contents or volume of the continuous postoperative infusion were a factor. To date, no studies have addressed the possible neurotoxicity of epidural meperidine.

Anterior spinal artery syndrome is characterized by burning dysesthetic back pain from the feet to the level of the lesion; sudden onset of flaccid paraplegia with loss of sphincter control; and loss of pain and temper-
nature with preservation of light touch and proprioception below the lesion. The anterior spinal artery may be at particular risk for occlusion during hypotension because it is unpaired and supplied by anatomically inconstant radicular branches. Lazorthes et al. demonstrated that in 15% of the population the artery of Adamkiewicz has a high (thoracic) origin, further jeopardizing the arterial supply to the lumbar cord. Alternatively, ischemia may result from reduced venous drainage caused by compression of the venous drainage system. Spinal cord ischemia as a mechanism of paraplegia has been suggested after both spinal and epidural anesthesia. A transient case of anterior spinal artery syndrome caused by a spinal cord arteriovenous malformation has been reported by Warner et al. Davies et al. have described a patient who developed anterior spinal artery syndrome 1 day after surgery. All other cases were of immediate onset and were permanent.

In summary, we describe a patient who developed a long-lasting, incomplete sensory and motor deficit after subdural anesthesia. We conclude that permanent neurologic dysfunction continues to be a rare and puzzling phenomenon after regional anesthesia. Bleeding disorders or underlying anatomic anomalies pose increased risks. Rapid neurologic evaluation and diagnostic evaluation are required to ensure timely intervention.
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Anesthesia for a Patient with Gorham's Syndrome:
"Disappearing Bone Disease"

Devanand Mangar, M.D., M.A.,* Patrick A. Murtha, M.D.,† Thomas C. Aquilina, M.D.,† G. Raymond Connell, M.D.‡

GORHAM'S monostotic massive osteolysis is a rare chronic disease associated with vascular abnormality, angiomatosis, or hemangiomatosis.1-3 The syndrome is characterized by progressive regional loss of bone with resultant deformity.4 We describe the anesthetic management of a patient with Gorham's disease for revision of left pleuromedistalbey and placement of right pleurosubclavian shunts to drain chylos pleural effusion during general anesthesia.

Case Report

The patient was a 15-year-old, 35-kg, 166-cm boy with a 2-yr history of Gorham's disease diagnosed by bone biopsy and a history of multiple surgeries including placement of Harrington rods for deformed thoracolumbar spine, complicated by T10 paraplegia. The patient had left a left pleurocophcal vein shunt placed 1 yr earlier for left chylous pleural effusion, and the patient manually pumps the shunt (50 times per day) when he becomes dyspneic, with good relief of symptoms. The patient presented with increasing bilateral pleural effusion and respiratory distress due to a nonfunctional shunt. On the morning of surgery, the patient had a respiratory rate of 40 breaths/min and diffuse rhonchi in both lung fields. Arterial blood

* Assistant Professor, Department of Anesthesiology, University of South Florida College of Medicine.
† Resident, Department of Anesthesiology, University of South Florida College of Medicine.
‡ Staff Anesthesiologist, Grants Medical Center.

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Address reprint requests to Dr. Mangar: Department of Anesthesiology, University of South Florida College of Medicine, 12901 Bruce B. Downs Boulevard, MDC Box 59, Tampa, Florida 33612.

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