Subdural Injection of Local Anesthetic: A Complication of Epidural Anesthesia

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Several cases of “massive epidural” anesthesia following the presumed injection of small volumes of local anesthetics into the epidural space have been reported.1-3 The authors describing these cases have speculated that such extensive segmental anesthesia may be the result of injection into the subdural space.

A radiographically proven case of subdural catheterization during an attempted epidural anesthesia is reported. Signs and symptoms of subdural anesthesia as well as probable mechanisms for this complication of epidural anesthesia are discussed. Pertinent radiologic and anesthetic literature is reviewed.

REPORT OF A CASE

A 71-year-old woman with diffuse atherosclerotic peripheral vascular disease was scheduled for a left femoral-popliteal bypass and left carotid endarterectomy (CEA) under lumbar epidural anesthesia and cervical plexus block. The patient weighed 70 kg and was 165 cm tall. Her medical history included three left hemispheric cerebral vascular accidents without persistent deficit, adult onset diabetes mellitus controlled by diet only, and hypertension well controlled by propranolol 40 mg po, bid and hydralazine 25 mg po, bid.

The patient was premedicated with propranolol 40 mg and hydralazine 25 mg given orally and morphine sulfate 6 mg im 30 min before induction of anesthesia. The following cannulations had been performed during the night before surgery: a 20-gauge radial arterial catheter, 16-gauge peripheral iv catheter, and a 7 French thermodilution Swan-Ganz catheter in the right subclavian vein.

The patient was transported to the operating room, where a lumbar epidural was attempted in the left lateral decubitus position at the L2–3 interspace with the use of an 18-gauge Touhy needle, by a midline approach with the bevel pointed cephalad. Loss of resistance to air was used to identify the epidural space. Immediately after entering the epidural space, the patient moved unexpectedly and cerebrospinal fluid (CSF) was observed to flow from the needle. The needle was withdrawn approximately 3 mm until no further CSF could be aspirated and a portex catheter was inserted. Two ml CSF was aspirated from the catheter, suggesting the catheter tip was in the subarachnoid space.

We elected to proceed with a continuous spinal anesthetic. Four ml 0.5% isobaric bupivacaine were injected. The catheter was taped into place and the patient was turned supine. After 20 min, no sensory or motor block was evident, and a further 5 cc of 0.5% bupivacaine was injected. Because 15 min later no block was apparent, general anesthesia was induced with 12 mg etomidate and 100 mg succinylcholine iv. The trachea was intubated and anesthesia maintained with N2O/O2 and isoflurane. Shortly after surgery began (about 45 min after the first injection of bupivacaine), the systolic pressure gradually decreased from 155 mmHg to 100 mmHg over a period of 20 min, at which point isoflurane was discontinued and anesthesia was maintained with N2O/O2 and incremental iv dose of fentanyl totaling 0.25 mg.

The surgery, which lasted 3 h, was confined to the femoral-popliteal bypass; the CEA was postponed because the surgeon preferred to do this under regional anesthesia. At the conclusion of surgery the trachea was extubated, and the patient was transported to the recovery room awake and in stable condition. At this point the patient had a bilateral motor and sensory block from T8 to S5. The patient was taken to radiology, where 7 ml of metrizamide (220 mg iodine/ml) was injected into the catheter, and the location of the epidural catheter was demonstrated by fluoroscopy.

Two interesting observations were made. Contrast material was visualized in both the subdural and subarachnoid spaces. The catheter was observed to have taken a caudal position in the subdural space in spite of the cephalad direction of the Touhy needle (figs. 1–3). A bilateral motor and sensory block persisted approximately 8 h after the first injection of bupivacaine. The patient’s right leg and arm remained weak after the sensory block had dissipated, suggesting a perioperative CVA had occurred. This was confirmed by neurologic consultation. The motor weakness resolved over the course of the next 10 days, making it unlikely that this was related to the block.

DISCUSSION

Subdural injection of contrast material is well documented in the radiologic literature. Schultz and Brogdon found a 13% incidence of accidental subdural injection of contrast in 140 myelographies.4 Jones and Newton reviewed 244 myelographies and found a 10% incidence of extrarachnoid injections. They also found that in some cases the contrast material injected into the subdural space may traverse the spinal canal from S2 to intracranial structures.5 During insertion of the spinal needle into the subarachnoid space (usually a long beveled needle), the bevel may come to lie partly within both subdural and subarachnoid spaces. CSF is aspirated, leading to the false impression that the needle point lies entirely within the subarachnoid space. When contrast material is injected, it may pass entirely into the subdural space, which has been distended by leakage of CSF around the needle. Perhaps a one-way valve effect by a flap of pia-arachnoid may prevent contrast material from entering the subarachnoid space as this membrane is pushed away from the needle point by the jet formed during injection. They postulated that a recent diagnostic

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was CSF aspirated, as was seen in our case, and the catheter was inserted into the presumed epidural space after loss of resistance to air or saline. After injection of local anesthetic via the catheter, several common features were noted. Uniformly, there was an extensive, delayed (20–30 min) sensory block after the lumbar injection of a small volume (3.5 to 10 ml) of local anesthetic, often extending to the cervical segments.8–10 A concomitant decrease in arterial blood pressure, easily correctable (in contrast to a total spinal) by the iv

lumbar puncture (LP) might cause CSF to leak into the subdural space and distend it, thus making accidental contrast injections into this space more likely.4 Unrecognized injection of local anesthetic into the subdural space distended by CSF also may lead to failed spinal anesthesia.6

Although there have been several reports of presumed accidental subdural injection of local anesthetic,1–3,7 only three radiographically proven cases of subdural catheterization have been reported during the course of an attempted epidural anesthetic.8–10 In none of these cases

![Image 1](https://example.com/image1)

**Fig. 1. Subdural contrast material overlying L1 and L2 vertebrae. Contrast outline is diffuse; no nerve roots are outlined.**

![Image 2](https://example.com/image2)

**Fig. 2. Subdural contrast seen over L2 vertebral body. Subarachnoid contrast material seen over L4. Subarachnoid contrast outline is more distinct. Catheter ends over L4.**
infusion of crystalloid, was seen. Dyspnea has been reported in several cases, although no cases of apnea were reported as seen in total subarachnoid anesthesia. There was no evidence of respiratory depression in this case during the 35 min before induction of general anesthesia.

Anatomically, the subdural space is a potential space between the dura mater and the pia-arachnoid throughout the distribution of the meninges covering all neural structures in the central nervous system including the spinal nerve roots. The spinal subdural space has a much smaller volume than the epidural space, so small volumes of local anesthetic travel extensively within this space. Since the pia-arachnoid and the dura mater have separate attachments to the dorsal nerve root but have a common attachment to the ventral nerve root, local anesthetic in the subdural space would be expected to pool dorsally. This anatomic distribution may explain the partial sparing of sympathetic and motor fibers and the relatively moderate fall in BP seen in subdural injection compared with the precipitous fall in BP seen in total spinal anesthesia.

Mehta and Maher have described intentional cervical subdural injection of neurolytic agents with contrast material for treatment of intractable cancer pain. They found that contrast solutions injected into the subdural space ascend slowly against gravity and spread into the cranium and outward onto the cervical nerve roots. In their technique, a short beveled needle is inserted into the midline using loss of resistance to identify the epidural space. The needle then is advanced; the bevel is rotated through 180 degrees until a sudden increase in resistance occurs. The needle now lies in the subdural space. Thus, in performing epidural anesthesia, an increase in resistance after the usual loss of resistance may indicate that the epidural needle lies in the subdural space. Likewise, rotation of the needle after loss of resistance may increase the likelihood of entry into the subdural space.

A small but definite substantiated incidence of subdural injection occurs during the course of epidural anesthesia. This incidence may be increased by a preceding LP, spinal anesthetic, accidental dural puncture, abnormal loss of resistance, or rotation of the needle. With the foregoing in mind, it would seem prudent to always choose another interspace whenever accidental dural puncture has occurred. Also, as this case has demonstrated, the needle probably should not be withdrawn from the subarachnoid space into the epidural space in an attempt to salvage an epidural anesthetic after dural puncture. This phenomenon should be considered when one is attempting to perform a prophylactic “saline patch” after unintentional dural puncture. When this complication is suspected, perhaps a water-soluble contrast medium such as metrizamide should be injected under fluoroscopic control to confirm the aberrant position of the catheter. Nonaqueous contrast media, particularly Pantopaque®, which have been associated with arachnoiditis, should be avoided. A final point demonstrated here is that the Touhy needle does not guarantee the direction that the epidural catheter will take. Perhaps this case will clarify the mechanism of unintentional subdural injection of local anesthetic, enabling the anesthetist to avoid this complication and to recognize it when it occurs.

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Is There a Risk of General Anesthesia Triggering SIDS? Possibly Not!

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Sudden infant death syndrome (SIDS) is the cause of at least 5,000 deaths each year in the United States (1.5–2 deaths per 1,000 live births)1 and is the leading cause of death between the age of one month and one year of life. The accepted definition of SIDS is “The sudden death of any infant or young child which is unexpected by history and in which a thorough post-mortem examination fails to demonstrate an adequate cause for death.”1 It is a condition that haunts parents and physicians alike.2 It also is a condition that alarms the pediatric anesthesiologist, who is concerned that SIDS might be precipitated in the perioperative period. This concern has even prompted some physicians to question whether nonurgent surgery should be deferred in infants who are in the at-risk age range for SIDS. This deferral of nonurgent surgery, however, would not affect most infant patients, as most surgical conditions seen in this age group are considered to be at least semi-urgent (e.g., inguinal hernia).

While some factors have been found to be clearly associated with SIDS, the precise identification of those children who are definitely “at risk” is not yet possible. It is also not known whether general anesthesia and surgery might precipitate the occurrence of SIDS in a child who is at risk, in a manner similar to that which has been shown to predispose the experramenture to perioperative apnea.3 No studies have correlated the association of general anesthesia and SIDS. It is also not established whether infants who have later succumbed to SIDS had previously been exposed to general anesthesia.

STUDY

This study was approved by the Human Ethics Committee. Medical Records of 35 children who died of sudden infant death syndrome were examined to determine whether any of them had a previous history of receiving general anesthesia at any time. Three infants were found to have required a general anesthetic at some time before their final fatal illness.

REPORT OF THREE CASES

Case 1. A full-term female infant (birth weight—3 kg) of a 17-year-old single mother had an uneventful delivery except for a “cord around the neck.” No neonatal resuscitation was required. Mild physiologic jaundice was present until the infant was four days old, when the child was discharged home with the mother. At age 6 weeks the child was admitted to hospital with a history of vomiting after feeding and a diagnosis of pyloric stenosis. Endotracheal anesthesia with halothane in oxygen was uneventful and the patient was discharged home after 1 week.

At age 16 weeks the child was found in bed with cyanosis and no pulse. Cardiopulmonary resuscitation by ambulance personnel was

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