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Total Spinal Anesthesia, A Late Complication of Epidural Anesthesia

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A common complication associated with thoracic or lumbar extradural block is inadvertent dural puncture. In 1969, Dawkins gave the incidence of dural puncture as 0.4–7.5 per cent depending on the type of "extradural indicator" used. In 48,292 cases reviewed by Dawkins, the incidence of unrecognized dural puncture followed by a full extradural injection and subsequent total spinal anesthesia was 0.2 per cent.¹ The onset of total spinal anesthesia, with its concomitant signs and symptoms of hypotension and hypoventilation, is usually very abrupt. The following is a case report of an unusual complication of continuous epidural anesthesia.

REPORT OF A CASE

A 23-year-old black woman, gravida 5, para 4, was admitted to the hospital in active labor. Past medical history was significant only in that she had hypertension and was on a low-salt diet. Prenatal blood pressures ranged from 130/90 to 160/100 torr. Upon admission to the labor and delivery suite, the patient experienced a precipitous delivery of a healthy female infant, without complication. The only anesthesia administered for the delivery was 10 ml of 1.0 per cent lidocaine, infiltrated locally for the episiotomy.

The following day, the patient requested sterilization, and she was subsequently scheduled for bilateral tubal ligation under epidural anesthesia. Postpartum blood pressures ranged from 110/70 to 170/110 torr. Laboratory studies were all within acceptable limits. The patient's height and weight recorded 24 hours postpartum were 159 cm and 71 kg, respectively.

After an iv route was established, blood pressure cuff applied, and appropriate sterile preparation, an epidural catheter was placed without difficulty via an 18-gauge thin-walled Tuohy needle in the L3–4 interspace. The patient was in the sitting position, and loss-of-resistance technique was utilized. Aspiration tests of cerebrospinal fluid were negative in 360 degrees and also after the epidural catheter was placed. A 2-ml test dose of anesthetic solution, which consisted of a mixture of .75 per cent bupivacaine and 3.0 per cent chloroprocaine in a ratio of 2:1, was injected into the epidural space. After monitoring vital signs, waiting 3–5 minutes, and after negative aspiration tests, an additional 12-ml volume of anesthetic solution was injected (total anesthetic dose injected = 70 mg bupivacaine and 140 mg chloroprocaine). The patient was placed in the supine position, level, and after 15 minutes analgesia to T8–9 was obtained without a significant decrease in blood pressure. The operation was carried out uneventfully. Postoperatively, when the

patient and operating room staff were readying to move the patient from the operating room table to the transport stretcher, the patient began complaining of shortness of breath. Ventilation was assisted via mask with 100 per cent oxygen and the anesthetic level, when retested, was at T2–3. Blood pressure and pulse remained stable. Fifteen minutes later, an hour after epidural injection, the patient was completely apneic and unable to move. Anesthetic level was again tested and revealed a sensory level of C2–3. Other vital signs remained stable. Aspiration of cerebrospinal fluid through the epidural catheter was again attempted, but was negative. After the situation had been explained to the patient, the trachea was intubated nasally after 3 mg *d*-tubocurarine 150 mg thiopental, and 100 mg succinylcholine. The patient was then taken to the recovery room, aspiration of cerebrospinal fluid via the epidural catheter was again attempted, and 10–15 ml of clear cerebrospinal fluid were obtained. Over the course of the next two hours the level of motor and sensory blockade rapidly descended, and the trachea was extubated.

Before removal of the epidural catheter a prophylactic blood patch was performed with 10 ml of autologous blood. Upon discharge, the patient's neurologic status was normal, and she had no symptoms of headache.

COMMENT

Although total spinal anesthesia is an infrequent complication of extradural block, its life-threatening potential is well recognized. The onset of total spinal anesthesia is usually very abrupt, and its effects on blood pressure profound. The case reported is unusual in both respects, in that onset occurred almost an hour after epidural injection and blood pressure remained stable.

Inadvertent dural puncture during epidural anesthesia can result from dural penetration by the needle or the catheter tip. In the first 1,000 epidural cases reported by Crawford, of 71 dural taps, nine were with the catheter, and in one case dural tap occurred twice.² In his second series of 1,000, Crawford reported 33 dural taps, four of which were caused by the catheter.³ Another factor that may contribute to dural puncture by an epidural catheter is the existence of a thin membranous dura, which can occur either as a congenital abnormality or as the result of an extramedullary tumor that has secondarily thinned out the dura. Last, it can be the result of tethering or adherence of the dura to the internal bony surface of the vertebral bodies, which is common in the lumbar region and not usually associated with any neurologic abnormality.⁴

Another possibility that must also be considered is that of total epidural anesthesia. That the level of

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anesthesia took 15 minutes to go from T₂₋₃ to C₂₋₃ once it started to rise would be consistent with an epidural anesthetic containing a long-acting agent with slow onset, such as bupivacaine. The inability to aspirate cerebrospinal fluid via the epidural catheter at that time would also be consistent with a slow-onset epidural anesthetic.

We feel that the cause of delayed-onset total spinal anesthesia in this patient most likely to have been subarachnoid migration of the catheter tip following epidural injection of the anesthetic solution, thus allowing the anesthetic solution to "leak" slowly from the epidural space to the subarachnoid space. The inability to aspirate cerebrospinal fluid from the

catheter while the patient was in the operating room may have been due to kinking of the catheter while the patient was in the supine position and lying on the catheter.

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Circulatory Changes during Direct Laryngoscopy and Tracheal Intubation:

Influence of Duration of Laryngoscopy with or without Prior Lidocaine

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Circulatory stimulation during tracheal intubation results from both direct laryngoscopy and placement of the tube in the trachea. Pharmacologic attempts to attenuate these blood pressure and heart rate (HR) elevations have included topical anesthesia of the oropharynx,^{1,2} iv lidocaine,[†] adrenergic blocking drugs,^{3,4} vasodilating drugs,⁵ and deep anesthesia.⁶ Although these approaches have been partially successful, we have observed that any protection provided against blood pressure increases may be negated by prolonged laryngoscopy. The influence of the duration of laryngoscopy on circulatory changes during laryngoscopy and tracheal intubation has not been examined. This report describes such changes during direct laryngoscopy lasting 60 seconds followed by tracheal intubation with or without laryngo-tracheal, intravenous or topical oropharyngeal administration of lidocaine.

METHODS

Thirty-six adult patients without known heart disease and scheduled for major noncardiac operations

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† Denlinger JK, Messner JT, D'Orazio DJ, et al: Effect of intravenous lidocaine on the circulatory response to tracheal intubation. Abstracts of Scientific Presentations, Annual Meeting of the American Society of Anesthesiologists, October 1975, pages 43-44.

TABLE 1. Comparative Patient Data from the Study Groups (Mean ± SE)

	Control	Viscous Lidocaine	Intravenous Lidocaine
Age (years)	47 ± 3	51 ± 2	49 ± 2
Weight (kg)	75 ± 3	84 ± 5	76 ± 3
Mean arterial pressure (torr)			
Awake	92 ± 2	92 ± 3	97 ± 3
1 minute after thiamylal-SCh	82 ± 3*	80 ± 4*	84 ± 4*
Heart rate (beats/min)			
Awake	72 ± 4	71 ± 4	75 ± 4
1 minute after thiamylal-SCh	83 ± 5*	85 ± 3*	83 ± 4

* *P* < 0.05 compared with awake value.

were studied. The protocol was approved by the Indiana University School of Medicine Clinical Research Committee, and patient consent was obtained. Preanesthetic medication consisted of im administration of morphine (8-15 mg) and scopolamine (0.4 mg). Upon arrival of the patient in the operating room a radial-artery catheter was inserted to permit continuous recording of mean arterial pressure (MAP) and calculation of HR.

While the patient was breathing oxygen, *d*-tubocurarine (*d*/Tc, 40 µg/kg) was administered iv, followed 3 minutes later by thiamylal (4 mg/kg) and succinylcholine (SCh, 2 mg/kg). Direct laryngoscopy with a straight laryngoscope blade was initiated 1 minute after thiamylal-succinylcholine and maintained for