CASE REPORTS

Anesthesiology
80:939–941, 1994

J. B. Lippincott Company, Philadelphia

Total Spinal Anesthesia after Interscalene Blockade of the Brachial Plexus

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BLOCKADE of the brachial plexus has been used to provide anesthesia for all types of surgery of the hand, upper extremity and shoulder.[1] Several reports have described spinal or epidural anesthesia resulting from attempted blockade of the brachial plexus using an 8.9-cm needle by the interscalene route.[2–11] We report a recent case where cervical spinal anesthesia, possibly complicated by epidural or prevertebral spread of anesthetic agents, ensued after successful brachial plexus blockade with a 2.5-cm needle.

Case Report

A 30-yr-old man presented for debridement and closure of a traumatic injury of the left forearm. The planned anesthetic technique was a brachial plexus block of the left arm, by the interscalene approach, in combination with a light general anesthetic.

An 18-G intravenous catheter was already in place, so the patient was brought into the operating room and a pulse oximeter, automated blood pressure cuff (Dinamap®, Critikon), and ECG monitor were applied. The patient was supplied with supplemental oxygen by face mask at 5 l/min, and the left neck and shoulder were sterilized with Betadine solution and draped in sterile fashion. After topical anesthesia of the skin with 1% lidocaine, a 22-G, 2.5-cm, short bevel nerve blockade needle (Plexufix®, Burron) was advanced between the bellies of the anterior and middle scalene muscles at the level of C6, as described by Winnie.[7] A left median nerve paresthesia was elicited, and a mixture of 20 ml 0.5% bupivacaine and 20 ml 2% lidocaine was injected slowly, with negative aspiration after each 3–5 ml. Toward the end of the 5-min injection, the patient moved his head and neck slightly. He was cautioned not to move, and after repeated negative aspiration, the remaining 5 ml local anesthetic was injected.

Immediately after the injection, the patient became unresponsive and apneic, with loss of muscle tone in all extremities. The blood pressure decreased from 140/80 to 95/60 mmHg and the pulse from 85 to 70 beats/min. No seizure activity was noted. Intravenous fluid was administered, cricoid pressure was applied, and the trachea was intubated after the administration of thiopental 100 mg and succinylcholine 100 mg (because the level of muscle relaxation and amnesia could not be determined). Mechanical ventilation was instituted, and because the patient's vital signs were stable, surgery was begun. It was assumed that a total spinal anesthetic had occurred.[2,3] Subsequent hemodynamic and neurologic signs are summarized in table 1. The surgical procedure lasted 2 h, during which time general anesthesia was maintained with 50% nitrous oxide in oxygen. The patient's heart rate and blood pressure remained stable during the first h postinjection, and then the heart rate gradually decreased to 60 beats/min, while the blood pressure remained constant. The pupils were initially noted to be widely dilated, but they gradually constricted to pinpoint size over the course of the first h. Peripheral motor tone remained flaccid until near the end of the procedure. There was no response by the patient to surgical manipulation of the left arm.

At 2 h postinjection, the patient was able to breathe spontaneously, with adequate tidal volumes, and could follow commands to move his legs and right arm. The trachea was extubated at this time, but the patient complained of a "choking sensation" and an inability to clear his secretions. Fifty milligrams thiopental was administered intravenously, and the patient's trachea was reintubated easily, at which time the vocal cords were noted to be moving spontaneously. The patient was then transferred to the postanesthesia care unit, spontaneously breathing 100% oxygen. Pinprick testing revealed a dense band of sensory anesthesia extending from C6 to T2 bilaterally. The left (operative) arm had proximal motor strength of 0/5, whereas the right arm had a strength of 2/5. Thirty minutes later, right arm strength and sensation had returned to normal, while the left arm remained flaccid. The trachea was extubated without incident. Left arm anesthesia and motor blockade gradually resolved over the next 4 h. Close questioning on postoperative day 1 revealed no recall of perioperative events.

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Received from the Uniformed Services University of the Health Sciences and National Naval Medical Center, Bethesda, Maryland, and Harvard Medical School and Massachusetts General Hospital, Boston, Massachusetts. Accepted for publication November 23, 1993.

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Key words: Anesthesia, regional: interscalene. Complications: total spinal anesthesia.

Table 1. Neurologic and Hemodynamic Signs

Time after Blockade (min)	Blood Pressure (mmHg)	Heart Rate (beats/min)	Pupil Size (mm)	Sensory/Motor Level
Preinjection	140/80	85	3	Intact
10	95/65	70	10	General anesthesia
60	100/60	60	Pinpoint	General anesthesia
120	130/80	80	3	C-6 to T-2 Bilateral segmental analgesia
180	140/80	80	3	Left arm blockade
360	135/80	80	3	Intact

Discussion

Both spinal and epidural anesthesia have been reported to occur after interscalene blockade of the brachial plexus with a variety of needle lengths from 5.1 to 8.9 cm. A number of mechanisms have been postulated to explain these complications, including direct injection into the epidural or subdural space secondary to incorrect needle placement, perineural or intraneural injection of drug, prevertebral spread of injected agent, or injection through a correctly placed blockade needle into an abnormally long dural root sleeve. Our interpretation of the events in this case is as follows.

After the near completion of injection of a successful interscalene block (as argued by the 5–6 h of anesthesia that resulted), the last few milliliters of local anesthetic was injected into either the epidural or the subarachnoid space, or both, at C6, probably as a result of advancement of the block needle into the intervertebral foramen. The rapidity with which symptoms developed (ranging from inability to phonate to unconsciousness, apnea, hypotension, and bradycardia) argues for some degree of subarachnoid injection, although the short needle used was intended to minimize the risk of this complication. It is possible that the trachea could have been easily intubated without further intervention, but succinylcholine and a small dose of thiopental were administered to insure good conditions. Clearly, some amount of local anesthetic entered the cranium: the observation of initial dilated and nonreactive pupils is consistent with a loss of parasympathetic efferent activity from the Edinger-Westphal nucleus, and the observed bradycardia can be most easily explained by cervicothoracic spinal anesthesia with blockade of the cardiac accelerator fibers (T1–T4).[7] These early signs persisted for 45 min, at which time the pupils constricted to pinpoint size and the heart rate decreased further, to 60 beats/min. This evidence of a high, but not intracranial, blockade persisted for approximately 2 h. At the conclusion of the case, the patient seemed to have recovered completely; vital signs had returned to baseline, he was breathing spontaneously, and he was able to move the three nonoperative extremities in response to command. It was only after a failed attempt at extubation that his high band of segmental analgesia and right upper extremity motor weakness was appreciated. This too resolved in due course, and the patient was left with the intended brachial plexus block.

This unexpected blockade of the right arm could have been the result of segmental spinal blockade, occurring as the cerebrospinal fluid concentration of anesthetic receded, or of epidural or prevertebral spread of anesthetic. Although several authors have observed this phenomenon and theorized as to its cause,[8–10] there seems to be no easy way to determine the actual route by which local anesthetic agents reached the contralateral nerve roots.

The sudden total flaccidity and subsequent lack of recall experienced by this patient are consistent with previously published reports by Ross and Scarborough[2] and McGlade.[11] Although it was of some concern to the anesthesiologists in attendance, the patient displayed no evidence of the seizure activity or myocardial depression that might have resulted from an intravascular injection of lidocaine or bupivacaine, respectively.[12,13] Durrani and Winnie have recently described a "locked-in syndrome" resulting from probable intraarterial injection accompanying a successful brachial plexus block.[14] In our case, unlike theirs, the patient had no recall of the event, and there was no seizure activity. These differences may represent the distinction between applying local anesthetic agents to the brain *via* the bloodstream or *via* the cerebrospinal fluid.[14]

However, the effect of local anesthetic administered *via* the cerebrospinal fluid on brain stem and cortical function is poorly understood at best. Haranath demonstrated that unlike intravenous or intraarterial injection, cerebrospinal fluid administration of local anesthetics such as procaine caused nystagmus, defecation, vomiting, respiratory depression and loss of consciousness after 15–30 min, but was not associated with seizure activity in a dog.[15] Various pupillary and autonomic findings have been reported in animal studies,[16] and it may be that subclinical electrical seizure activity occurs that can not be appreciated without the use of electroencephalography.

Also of interest to us was the cardiovascular response that our patient displayed, which was similar to that of the patient described by McGlade.[11] Despite high sympathectomy and some degree of parasympathetic blockade at the brain stem level, the patient's heart rate and blood pressure remained at acceptable levels throughout and the patient did not require vasopressors or chronotropic drugs. Direct application of local anesthetics into the medullary region of the central nervous system can result in hypotension, bradycardia, and ventricular arrhythmias.[16] Even if one postulated unopposed parasympathetic stimulation as the intracranial local anesthetic level receded, the patient remained quite stable, as suggested by the decrease in heart rate and pupillary constriction. However, it is important to note that our patient was young, healthy and euvolemic; the same complication in an elderly patient or one with serious cardiovascular disease may have produced profound hemodynamic change.

In summary, we report an extremely unusual combination of complications occurring after a brachial plexus block by the interscalene approach. Our report should once again emphasize the importance of careful technique, but more importantly, the need for appropriate patient monitoring and immediate access to resuscitation equipment when performing regional nerve blocks. The question of what effect local anesthetics have on the brain stem and cerebral cortex when applied *via* the cerebrospinal fluid remains unclear.

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