be selected based on sex,[2] this formula may also be applicable to these adult sizes. This formula is easy to remember and, we hope, helpful in clinical situations.

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Paraplegia after Cystectomy and Epidural Anesthesia

To the Editor:—Paraplegia after epidural anesthesia performed for urologic surgery in a hyperlordotic position has been previously described.[1,2] We report two additional cases of postoperative paraplegia after cystectomy and ileal bladder construction in which the nature of the neurologic deficits suggests that the injury was not related to the epidural anesthetic.

A 50-yr-old man (172 cm, 90 kg) was scheduled to undergo radical cystectomy. Epidural puncture was performed at the L3-L4 interspace, using a 17-gauge Tuohy needle. A catheter was then inserted 4 or 5 cm cephalad into the epidural space. No paresthesia was elicited at any time, and there was no return of blood or fluid through the catheter. After a test dose of 3 ml lidocaine, 1%, with epinephrine 1:200,000, 14 ml of a 1:1 mixture of lidocaine, 2%, and ropivacaine, 1%, was injected. After achieving a sensory anesthesia level at T8, general anesthesia was induced with etomidate and sufentanil. Paralysis was achieved with cisatracurium.

After induction, the patient was placed in a hyperlordotic position on the operating table, with a folded sheet used as a wedge under the sacrum. The surgical procedure was uneventful. Preoperative blood pressure was 100/70 mmHg. During the procedure, pressure ranged from 90/60 to 120/90 mmHg and was less than 100/70 mmHg for a total of only 10 min. Central venous pressure was maintained between 7 and 10 mmHg. Estimated blood loss was 1,000 ml. At admission to the recovery room, the patient was extubated and an extradural infusion of ropivacaine, 0.2%, at a rate of 4 ml/h was administered. Seven hours later he first complained of a loss of sensation in the left and right lower limbs. This was thought to be caused by the action of ropivacaine and hence the rate was reduced to 2 ml/h.

In the early morning on the day after surgery, the patient continued to complain of a loss of sensation in the left and right lower limbs. Epidural analgesia was discontinued. Neurologic evaluation, performed 5 h after discontinuation of the epidural analgesia, revealed flaccid paralysis for hip flexion, and 3/5 weakness for hip, knee, and ankle extension and for ankle and knee flexion bilaterally. Babinski sign was absent bilaterally. Perception of pinprick and cold on the medial and posterior aspect of his legs was also decreased. There was no impairment of perianal sensation and no incontinence. A lumbar plexus lesion was suspected, and subsequent electromyograph studies showed small compound muscle action potentials and slow motor nerve conduction velocities in the ankle to knee and sciatic to knee. Magnetic resonance imaging of the spine on the third day failed to reveal any spinal cord abnormalities. After 3 months, the patient had an increase in strength in most of the muscles around his hips, knees, and ankles. He was able to walk with a walker.

A 72-yr-old woman (160 cm, 67 kg) underwent radical resection of the bladder. Epidural and general anesthesia were performed as the first case. Twelve milliliters of a 1:1 mixture of lidocaine, 2%, and ropivacaine, 1%, was injected through the epidural catheter. The patient was placed in a hyperlordotic position. The surgical procedure was uneventful and blood pressure, heart rate, central venous pressure, and oxygen saturation were maintained within normal limits. Twenty minutes after the end of surgery the patient was conscious and extubated without difficulty, and postoperative analgesia was maintained with 5 ml/h ropivacaine, 0.2%. Eight hours after extubation she was still unable to move her legs. Ropivacaine infusion was reduced to 3 ml/h.

On the first postoperative day the patient continued to complain of inability to move her legs. Epidural analgesia was discontinued. Neurologic evaluation performed 5 h after discontinuation of the epidural analgesia showed significant motor dysfunction in the hip flexors and quadriceps bilaterally. There was no hypalgesia or hypesthesia, and reflexes were normal. The Babinski sign was absent. There were no impairment of perianal sensation and no incontinence. A femoral nerve lesion was suspected and electromyograph studies showed small compound muscle action potentials and slow motor nerve conduction velocities in femoral nerve bilaterally. Magnetic resonance imaging of the lumbar and thoracic spine on the third day was normal. After 5 months of rehabilitation, some motor improvement was noted.

Discussion

Neurologic deficits after epidural anesthesia are usually attributed to needle-catheter-induced trauma, spinal stenosis (perhaps combined with hypotension) leading to cord ischemia, or direct spinal cord drug toxicity. These factors seem unlikely to be relevant in these two cases. No paresthesia was elicited during needle or catheter placement, and there was no evidence that either the needle or the catheter entered the subarachnoid

space. Moreover, lesions of the spinal cord or caudal equina were unlikely in these patients. There was no magnetic resonance imaging evidence of cord disease and the presence of paraplegia without either a sensory deficit or perianal anesthesia–incontinence suggests that the lesion must lie outside the spinal cord.[3] In our opinion, the most likely explanation may be patient position. Paralysis after surgery in the hyperlordotic position has been reported.[1,2] This could result from increased tension on or stretch of nerves in the lumbar plexus. The potential for damage would also be increased by the long duration of surgery. Finally, direct nerve injury during the surgical dissection may have contributed. As a result, we would urge caution when long-duration procedures in the hyperlordotic position are planned.

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