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Quadraplegia in a Patient with Cervical Spondylosis after Thoracolumbar Surgery in the Prone Position

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Patients with cervical spondylosis may be at increased risk for postoperative neurologic dysfunction after neurosurgical procedures. Previous reports have focused on the central cord syndrome after decompressive cervical laminectomy and midcervical quadriplegia after craniotomy in the sitting position.[1,2] We are unaware of either of these conditions as complications of procedures performed in the prone position. We report the development of postoperative quadraparesis, despite seemingly optimal anesthetic management, in a patient with severe cervical spondylosis who underwent thoracolumbar spine surgery in the prone position.

CASE REPORT

A 60-yr-old man with severe degenerative joint disease and spinal stenosis required a decompressive laminectomy from T11 through L5. The patient's symptoms consisted of mild but progressive lower-extremity weakness and spasticity. Radiologic evaluation, including a myelogram and magnetic resonance imaging scan, revealed severe cervical, thoracic, and lumbar spinal stenosis with evidence of spinal cord compression in all three areas, although the stenosis was more profound in the thoracic and lumbar segments (fig. 1). The remainder of the past medical history was unremarkable. The physical examination was notable for a blood pressure of 110/70 mmHg and heart rate of 78 beats per min. The patient was mildly obese and had severe limitation of cervical range of motion in all directions. Upper extremity strength was 4+ to 5/5, and lower extremity strength was 3 to 4/5 (where 5 = full strength and 0 = complete paralysis). The upper-extremity reflexes were normal, and spasticity was present in the lower extremities. Bilateral shoulder joint movement was severely limited. The hematocrit was 32%. The remainder of the examination and laboratory evaluation was unremarkable.

Because of the severity of the patient's cervical spinal stenosis, awake tracheal intubation and positioning were planned. Premedication consisted of droperidol and fentanyl, and a state of mild sedation was achieved. A radial arterial catheter was inserted. Topical lidocaine was applied to the oropharynx; superior laryngeal nerve blocks were performed; and transtracheal lidocaine was administered. Awake oral tracheal intubation under direct visual laryngoscopy was performed without difficulty and with minimal cervical extension. The patient was turned prone and positioned on a Wilson frame and "horseshoe" head rest; the patient remained cooperative and was encouraged to assist in positioning so that his cervical spine would be aligned in a position comfortable to him (approximately neutral). Careful examination of upper- and lower-extremity strength revealed no change, and anesthesia was induced with sodium thiamylal and fentanyl. Pancuronium was administered to facilitate muscle relaxation, and anesthesia was maintained with approximately 1% isoflurane and 60% nitrous oxide in oxygen and with fentanyl 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$. Monitors consisted of electrocardiogram, pulse oximeter, intraarterial pressure, blood pressure cuff, temperature probe, esophageal stethoscope, capnograph, Foley catheter, and nerve stimulator.

Decompressive laminectomies were performed from T10 through L5; in addition, a large, bony mass that extended through the dura was resected at the T11–T12 level. Except for an unexpectedly long duration of surgery (approximately 6 h), the operation proceeded uneventfully. Arterial oxygen saturation remained at 100% throughout the procedure, and systolic blood pressure usually was maintained at between 100 and 120 mmHg. (Systolic pressure did briefly decrease to between 90–100 mmHg near the beginning of surgery; however, mean arterial pressure remained greater than 65 mmHg throughout this period.) Blood loss was estimated to be 1,000 ml, and two units of packed red blood cells were administered to maintain a hematocrit of 30%. At the conclusion of surgery, the inhalation anesthetics were discontinued; neuromuscular blockade was reversed with neostigmine and glycopyrrolate; and the lungs were ventilated with 100% oxygen. Since the patient remained asleep, a soft cervical collar was applied to aid in stabilizing the neck, and the patient was turned to the supine position with great care. He was then transported to the recovery room, breathing spontaneously *via* the endotracheal tube and a Mapleson C system.

In the recovery room, vital signs remained stable, and cervical neutrality was maintained; there were no episodes of coughing or bucking during this time. The patient's degree of consciousness increased to alertness over 10–15 min; the trachea then was extubated. Neurologic examination revealed a substantial change from the preoperative state; the patient was able to move the lower extremities, but strength of the upper extremities had deteriorated to 3/5 and that of the hands to 2/5. In the upper extremities, reflexes were diminished and in the lower extremities remained hyperactive. The gross sensory examination was normal.

During the next 2 months, the patient's neurologic function gradually returned to near his preoperative baseline, although mild weakness persisted in the upper extremities. Because the patient's function had reached a plateau, an anterior spine decompression was performed. After this procedure, the patient's hand function improved to about 4/5 during the ensuing 90 days.

DISCUSSION

The central cord syndrome and cervical quadriplegia are rare but potentially devastating complications of surgery and anesthesia. These syndromes may result when spinal cord anatomy is disrupted by unusual patient positioning or when spinal cord blood flow is decreased by

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FIG. 1. Magnetic resonance imaging scan showing severe cervical spondylosis.

positioning or hypotension. The mechanisms of injury may differ for the two syndromes.

The central cord syndrome was originally described by Schneider in 1954.[3] It occurs most commonly in patients with severe hyperextension injury of the neck, although the syndrome after much milder hyperextension injury has been reported.[4] Regardless of the injury, the syndrome is more likely to occur if the spinal canal previously has been narrowed by some pathologic process. The mechanism of injury in traumatic central cord syndrome may involve buckling of the ligamentum flavum and direct spinal cord compression, or a "pincher effect" between the posterior elements of the vertebral body below and the lamina above. Levy described the central cord syndrome as a delayed postoperative complication of decompressive cervical laminectomy.[1] Inadequate neck immobilization and orthostatic hypotension postoperatively in the presence of abnormal baseline spinal cord physiology and blood flow were believed to be events provoking the development of the syndrome.

Midcervical quadriplegia has been reported as a rare complication of neurosurgical procedures performed in the sitting position.[2] Antecedent narrowing of the spinal canal by spondylosis or other disease also may increase the risk of spinal cord injury. In contrast to the central cord syndrome, midcervical quadriplegia may be precipitated by extreme flexion of the neck. The syndrome has been described in an infant positioned supine with the neck in extreme hyperflexion intraoperatively[5] and in a man tied up by bandits in a position of extreme cervical flexion for 12 h.[6] The mechanism of injury may involve stretching of the spinal cord and subsequent disruption of blood flow autoregulation.[7] It has also been suggested that anterior displacement of the spinal cord by the taut dura may produce spinal cord compression.[4]

Our patient developed signs and symptoms suggestive of the central cord syndrome after undergoing surgery in the prone position. It is unlikely that direct surgical trauma to the spinal cord produced quadraparesis in our patient, because the operative site was far removed from the area of neurologic injury. In addition, careful attention was directed toward maintenance of cervical neutrality during positioning and surgery. Extreme degrees of hyperextension or flexion were carefully avoided, although subtle changes in position not evident to the eye may well have occurred. Blood pressure was carefully monitored, and significant hypotension did not occur. Although the exact reason for this patient's adverse outcome remains unclear, we assume that subtle alterations in spinal cord blood flow or anatomy or both resulted in central cord ischemia and injury.

When the prone position is used, careful positioning alone does not guarantee the absence of serious neurologic complications in patients with cervical spondylosis. For the intraoperative management of patients with cervical spinal stenosis, we recommend attention to the maintenance of a neutral cervical position and normal blood pressure, despite the lack of efficacy of these maneuvers in our case. Although somatosensory evoked potentials may seem an attractive monitoring adjunct, currently no evidence supports their use in this setting; in fact, evoked potentials may remain normal in patients with isolated motor deficits (*i.e.*, in those with the central cord syndrome).[8] More importantly, in patients with multiple segment spondylosis that includes significant cervical disease, initial cervical decompression should be considered.

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