

## ■ CASE REPORTS

Anesthesiology  
78:981-983, 1993  
© 1993 American Society of Anesthesiologists, Inc.  
J. B. Lippincott Company, Philadelphia

### Motor Paralysis of the Lower Extremities Following Lumbar Sympathetic Block

Winston C. V. Parris, M.D.,\* Howard S. Kirshner, M.D.†

PARAVERTEBRAL lumbar sympathetic nerve blocks<sup>1</sup> are performed for a variety of acute and chronic pain syndromes.<sup>2</sup> The complications of correctly performed lumbar sympathetic nerve blocks are rare and relatively minor. We present a case of permanent partial motor paralysis of the lower extremities following lumbar sympathetic nerve block performed for chronic low back pain syndrome.

#### Case Report

The patient was a 39-yr-old man with an 8-yr history of chronic low back pain radiating to his left lower extremity. His past history revealed that he sustained a traumatic injury to his back 8 yr earlier. Because of an exacerbation of pain and suffering, he was referred for evaluation and treatment. Physical examination was unremarkable except for palpable trigger points in the right paraspinous area at the L3 level. There were no other positive neurologic, musculoskeletal, or cardiorespiratory findings. His blood pressure was 134/92 and pulse was 56 beats/min. The patient was classified as an Emory Class II<sup>3</sup> patient with low pain pathology and low pain behavior.

Thermographic evaluation of his lumbosacral area and both lower extremities revealed moderate hypothermia of the left lower extremity consistent with reflex sympathetic dystrophy, and he was scheduled for a series of left lumbar sympathetic blocks as part of his multidisciplinary pain management. He received the first left paravertebral lumbar sympathetic nerve block with 20 ml 0.25% bupivacaine on the day of the evaluation. The technique used to perform the lumbar sympathetic block was a modification of that described by Carron *et al.*<sup>4</sup> using an initial test dose (2 ml) and the loss of resistance technique. The patient experienced a sensation of warmth in both legs, but he was able to walk to the psychologist's office for his psychologic evaluation (using the symptom checklist 90).<sup>5</sup> His psychologic profile was within normal limits. Ibuprofen (800 mg three times/day) was prescribed, and he returned to the Pain Clinic 1 week later indicating that his pain had decreased from a subjective pain intensity rating of 90% to 70%. He received a second and third

lumbar sympathetic nerve block without complications using the identical technique and solutions at weekly intervals.

When the patient returned for a fourth lumbar sympathetic nerve block, he indicated that the pain intensity had plateaued at a subjective pain intensity rating of 50%. Using the same aseptic precautions and technique as described previously, a 22-G 12.7-cm spinal needle was introduced at the L2 level approximately 3 cm from the midline on the left side. The transverse process of L2 was identified and the needle was withdrawn partially and redirected slightly cephalad and medially. Just as the loss of resistance test was about to be carried out, arterial blood began flowing from the needle after removal of the stylet. The needle immediately was withdrawn and reintroduced at the L3 level using the same technique. After eliciting loss of resistance and a negative aspiration for blood or other fluids, 20 ml 0.25% bupivacaine was injected into the prevertebral fascial plane. Immediately following the injection, the patient experienced a burning pain in the lower back area (L2-L5 area), and this pain radiated down both legs. Shortly afterward, he became hypertensive, with a blood pressure of 220/120, but his pulse remained at 55 beats/min. Almost simultaneously with the onset of hypertension, a circumscribed erythematous, papular cutaneous eruption developed between T5 and T9 on the left side, starting from the midline posteriorly and extending laterally and anteriorly to the midline. The rash receded and disappeared within 20 min. Antihistamine medications were considered, were not administered because of the relative brief duration of the cutaneous eruption. The blood pressure decreased gradually and spontaneously over the next 12 min to 143/96. The electrocardiogram remained normal throughout those events. The patient experienced no cardiorespiratory symptoms except for transient occipital headache at the peak of the hypertensive episode.

The burning back and leg pains changed to bilateral lower extremity numbness with progressive motor paresis. His deep tendon reflexes were decreased, but there were no other positive neurologic findings. After 6 h of observation, we made a presumptive diagnosis of profound lumbar somatic motor block. Ten hours later, his motor function had not improved and urinary retention developed. His bladder was catheterized, and physical examination revealed partial motor paralysis of both lower extremities with a sensory level at T8 on the right side and T12 on the left side, while proprioceptive and vibratory senses were preserved. Beevor's sign with positive "clasp knife rigidity" was present on the right side. Bowel function appeared to be grossly intact. Vital signs and laboratory investigations including a coagulogram were normal.

An emergency neurologic evaluation revealed that a motor deficit had developed in the patient's lower extremities secondary to possible mechanical (hematoma) or ischemic etiology. The possibility of temporary prolonged motor blockade secondary to inadvertent intrathecal anesthesia also was considered, but was dismissed because of the prolonged, profound, and nonreceding character of the motor blockade and the absence of signs of sympathetic block. Magnetic

\* Professor of Anesthesiology, Director, Pain Control Center.

† Professor and Vice Chairman, Department of Neurology.

Received from Vanderbilt University Medical Center, Nashville, Tennessee. Accepted for publication January 8, 1993.

Address reprint requests to Dr. Parris: Department of Anesthesiology, Vanderbilt University, Nashville, Tennessee 37232.

Key words: Lumbar sympathetic block complications. Motor paralysis.

## CASE REPORTS

resonance imaging (MRI) of the spine was normal, and there was no evidence of epidural hematoma or other spinal cord compression syndrome. The patient was assessed as having a T10 myelopathy, and his neurologic status remained stable. Over the next 24 h, there was improvement in motor function, but the bladder remained dysfunctional. Intermittent bladder catheterization was carried out every 6 h, and intensive physical therapy was commenced. Occupational therapy was also planned for a later date.

Seven days after the onset of his neurologic problems, the patient's general physical and medical condition improved and his motor paralysis receded. Hip flexion, knee extension, and ankle dorsiflexion were 80% of normal, with greater weakness on the right side. Sensory evaluation revealed that there was numbness and paresthesia inferior to the groin on the right side and from the mid-thigh distally on the left side. His bladder function continued to improve gradually. At this point in the course of his management, a tentative diagnosis of anterior spinal artery syndrome was made, since epidural or subdural hematoma was ruled out by MRI. Angiographic studies were considered but dismissed because of the possibility that the contrast media used for angiography may reactivate vasospasm of the "damaged" vasculature, resulting in exacerbation of the original neurologic lesion and possible reversal of his improving clinical condition. Instead, ultrasonographic studies of the abdomen were done, and the results were within normal limits. Computed tomographic scan of the abdomen was normal excluding abdominal aortic aneurysmal dissection or related pathology. Review of the MRI of the cervical, thoracic, and lumbar spinal cord suggested that a possible linear "cystic" area was present at the T10 level of the spinal cord and that this cystic lesion was possibly consistent with syringomyelia or related spinal cord cystic disease. No clear diagnostic consensus emerged among the radiology consultants who reviewed the MRI films.

The patient continued intensive physical therapy and occupational therapy, and his motor function continued to improve steadily. He was able to stand and ambulate haltingly with the assistance of a walker. Twenty days after admission to the hospital, the patient was discharged home with a significant, but resolving motor deficit, and he continued to receive aggressive rehabilitation therapy as an outpatient. Twenty-seven months after his neurologic problems, he continues to make satisfactory progress and is able to ambulate fairly well with the aid of a walker. His bowel function is normal except for persistent constipation, which he manages with stool softeners. He has occasional urinary retention with incontinence and catheterizes himself intermittently.

## Discussion

In the case presented, an immediate burning pain developed in the low back area and radiated down both legs. This was followed by a papular erythematous eruption confined to the region between T5 and T10. Immediately afterward, headache and hypertension developed. Cumulatively, these symptoms were unusual but initially were suggestive of an acute allergic response, although the patient never became hypotensive. Further, the motor paralysis, though much improved, persists 24 months later. The presumptive diagnosis is the development of the anterior spinal artery

syndrome<sup>6</sup> or some variation of that syndrome. It was noted that, during initial injection, blood flowed freely from the needle prior to injection of local anesthetic. It is possible that this blood flow resulted from inadvertent puncture of the anterior spinal artery or the artery of Adamkiewicz,<sup>7</sup> and the resulting arteriospasm (following puncture) may have produced progressive ischemia to the anterior (motor) segment of the spinal cord. The fact that the posterior cord was not involved (vibration and proprioception senses were intact) makes the possibility of ischemia of the anterior segment of the spinal cord plausible.<sup>8</sup> It is likely that the ischemia may have been transient, but that the damage to the cord was sufficient enough to produce some residual motor impairment. This theory is partially confirmed by progressive improvement in the patient's neurologic condition. Further, ultrasonographic studies were done and the results were normal. The hypothesis that this patient's problems were due to an ischemic event affecting the vascular integrity of the anterior segment of the spinal cord has not been proved.

Spinal cord ischemia with resultant motor paralysis (including paraplegia) has been reported as a complication of celiac plexus block.<sup>9,10</sup> The same mechanism could be wholly or partially responsible for motor deficits experienced by our patient following lumbar sympathetic block. The possibility of preexisting intraspinal pathology has been raised by the presence of linear cystic intraspinal lesions that were alluded to by some of the radiology consultants after review of MRI films.

In summary, it is possible that the artery of Adamkiewicz or an associated branch may have been punctured during the performance of the lumbar sympathetic block, resulting in spinal cord ischemia. Whereas the precise mechanism of this ischemia is unknown, the complication of spinal cord ischemia with subsequent motor deficit should be considered one of the possible though uncommon complications of lumbar sympathetic blocks.

## References

1. Bryce-Smith R: Injection of the lumbar sympathetic chain. *Anaesthesia* 6:150-153, 1951
2. Bonica JJ: *Clinical Application of Diagnostic and Therapeutic Nerve Blocks*. Oxford, Blackwell Scientific, 1958
3. Brena SF, Koch DL: The "pain estimate" model for the quantification and classification of chronic pain states. *Anesthesiology Review* 2:8-13, 1975

## CASE REPORTS

4. Carron H, Korbon GA, Rowlingson JC: Techniques and clinical applications, Regional Anesthesia. New York, Grune & Stratton, 1984, pp 132-141
5. Derogatis LR: The SCL-90 Manual. Baltimore, Clinical Psychometric Research, 1975
6. Kozody R, Palahniuk RJ, Wade JG, Cumming MO: The effect of subarachnoid epinephrine and phenylephrine on spinal cord blood flow. *Can J Anaesth* 31:503-508, 1984
7. Crock HV, Yoshizawa H: The blood supply of the vertebral column and spinal cord in man. New York, Springer, 1979
8. Forrester AC: Mishaps in anaesthesia. *Anaesthesia* 14:388-399, 1959
9. Lo JN, Buckley JJ: Spinal cord ischemia: A complication of celiac plexus block. *Reg Anesth* 7:66-68, 1982
10. Woodham MJ, Hanna MH: Paraplegia after coeliac plexus block. *Anaesthesia* 44:487-489, 1989

Anesthesiology

78:983-985, 1993

© 1993 American Society of Anesthesiologists, Inc.  
J. B. Lippincott Company, Philadelphia

## Digitally Assisted Tracheal Intubation in a Neonate with Pierre Robin Syndrome

Paul T. Sutura, M.D.,\* Gregory J. Gordon, M.D.†

IN a recent review of approaches to the difficult adult airway<sup>1</sup> and in a recent report and discussion of tracheal intubation in a patient with Pierre Robin syndrome,<sup>2,3</sup> no mention was made of digitally assisted tracheal intubation. Our experience, reported here, suggests that digitally assisted tracheal intubation may be the technique of choice in this and similar conditions with mandibular hypoplasia.

### Case Report

An 8-day-old, 3.3-kg female was scheduled for glossopexy and tongue-lip adhesion. The diagnosis of Pierre Robin syndrome was made shortly after birth when micrognathia and glossoptosis causing upper airway obstruction were observed.

The infant was brought to the operating room while prone and with an intravenous catheter in place. Precordial stethoscope, percutaneous oxygen saturation, electrocardiogram, blood pressure, and

skin temperature were monitored. When the infant was turned supine, airway patency could be maintained using a nasal airway and gentle jaw-thrust maneuver. Atropine (0.1 mg intravenous) was given and midazolam was titrated in small incremental doses so that a total of 0.3 mg was given intravenously over about 2 h while preparations were made and intubation attempted. Experienced personnel and equipment for performance of an emergency tracheostomy were available immediately. Blind nasotracheal intubation with a 3.0-mm tube was attempted after instillation of phenylephrine-lidocaine nose drops. Despite careful positioning of the head and neck throughout their full ranges of motion plus external manipulation of the larynx, tracheal intubation was not achieved. Direct laryngoscopy by three experienced laryngoscopists, using several different pediatric laryngoscope blades, failed to visualize the glottis. After about 2 h of unsuccessful intubation attempts, minor gingival and palatal bleeding occurred, and it was elected to cancel surgery and reschedule the case for the following week. Airway patency, vital signs, and percutaneous oxygen saturation remained stable throughout.

Eight days later, the infant, with a nasopharyngeal airway in place, was again brought to the operating room. The plan was to first attempt digitally assisted tracheal intubation under local anesthesia and mild sedation. Monitors and emergency preparations were as before. In addition, equipment was available for intubation using a 3.7-mm flexible fiberoptic endoscope (Olympus LF-1, New York, NY) in a modified Seldinger technique and for performance of a retrograde technique. Local anesthesia of the upper airway was achieved by having the infant breathe aerosolized lidocaine *via* a size 0 pediatric face mask for about 10 min. The lidocaine was aerosolized by flowing oxygen through 5 ml 5% lidocaine in a nebulizing chamber linked to several inches cut from a pediatric breathing circuit connected to the mask. Sedation was achieved with continuous intravenous propofol at 20-50  $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ . With this combination, the infant, eyes closed, appeared to suck comfortably on the anesthesiologist's finger throughout the digitally assisted tracheal intubation procedure.

\* Senior Resident in Anesthesiology.

† Assistant Professor of Anesthesiology, Division of Pediatric Anesthesiology.

Received from the MetroHealth Medical Center and Case Western Reserve University, Cleveland, Ohio. Accepted for publication January 12, 1993.

Address reprint requests to Dr. Gordon: Division of Pediatric Anesthesiology, Department of Anesthesiology, MetroHealth Medical Center, 2500 MetroHealth Drive, Cleveland, Ohio 44109.

Key words: Anesthesia: pediatric. Anesthetic techniques: digitally assisted tracheal intubation.