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## References

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# Evaluation of Double Lesion Syndrome with Diagnostic Spinal Anesthesia

*To the Editor:*—Loubser and Clearman[1] described the use of diagnostic spinal anesthesia in evaluating mechanisms of central spinal cord injury (SCI) pain. The following case report highlights another application of diagnostic spinal anesthesia in double lesion syndrome[2] (a subtype of SCI).

A 62-yr-old woman with complete quadriplegia of 2 yr, presented with chronic dysesthetic pain in the buttocks and lower extremities, which commenced approximately 6 months after the SCI. Pain was distributed from the buttocks to the toes and described as continuous and burning or stinging in nature. Dysesthetic pain also occurred in the upper extremities from the shoulders to the fingers in an asymmetric patchy distribution, although not as severe as in the lower extremities. Physical examination did not reveal any obvious source of nociception. The patient was insensate below C7 with allodynia or hyperpathia in the areas of pain. Reflexes, clonus, and spasticity were absent in the lower extremities. All attempts at oral pharmacologic management, including antidepressant (amitriptyline), anticonvulsant (carbamazepine), and opioid and nonopioid analgesics provided limited analgesia. Physical therapeutic modalities, such as range of motion, transcutaneous or neuromuscular electrical stimulation, and local heat, were ineffective.

Radiologic studies of the lumbosacral vertebral column, pelvis, and lower extremities excluded the presence of fractures or other bony abnormality. An extensive urodynamic evaluation of bladder function, including pressure-flow studies with simultaneous videocystourethrography, revealed an areflexic bladder neck. Lumbosacral somatosensory evoked potentials (LSEPs) were recorded from electrodes placed over the lumbosacral vertebrae (S1, L2, T12, reference at T6) after tibial nerve stimulation. An analysis of the R and S components (amplitudes and latencies) compared to control values revealed grossly abnormal LSEPs. These findings suggested the presence of an occult lumbosacral spinal lesion compatible with double lesion syndrome. The patient's pain symptomatology was attributed to the lumbosacral spinal lesion, and diagnostic spinal anesthesia was planned for confirmation before a trial of dorsal column stimulation or intrathecal opioids.

A 23-G intrathecal catheter was placed *via* a 20-G Tuohy needle in the L3–L4 vertebral interspace and threaded cephalad for 3 cm. Double-blind pain assessments included a 10-cm visual analog scale and somatic diagram indicating distribution of pain. After aspiration of clear cerebrospinal fluid (CSF) from the catheter hub, 2 ml of placebo (normal saline) was administered per catheter. However, no change in status was recorded for 60 min, whereupon 50 mg lidocaine in 7.5% dextrose was administered. Pain assessments were continued every 15 min over the next 90 min, without any recorded change in pain intensity or distribution. Similarly, no change in pain symptomatology occurred in the upper extremities. To verify that the catheter had not migrated out of the intrathecal space during spinal anesthesia, aspiration of free-flowing CSF from the catheter hub was demonstrated before catheter removal.

Double lesion syndrome was described by Beric *et al.*[2] as a clinical and a neurophysiologic syndrome characterized by an areflexic bladder, abnormal LSEPs, and cauda equina-like pain in conjunction with a primary cervical or thoracic SCI. Several hypotheses have been advanced to explain the development of the lumbosacral spinal lesion, including nerve hypoxia, arachnoiditis, spinal stenosis, and disc herniation, although at present its pathogenesis is not fully understood.[2] Furthermore, with respect to the pain symptoms, origination of nociception within the lumbosacral region is doubtful in patients with complete SCI (*i.e.,* absence of sensory perception below the level of the lesion). However, Beric *et al.*[2] caution that nonrecognition of lumbosacral dysfunction as the cause of pain could result in pain being interpreted as central in origin, *i.e.,* originating at or above the upper SCI.

The patient response described above suggests that the source of nociception was not within the cauda equina or lumbosacral vertebral column. Spinal anesthesia failed to produce any reduction of pain in the buttocks or lower extremities and response to placebo was negative. In contrast, the clinical presentation of widely distributed dysesthesias in the buttocks and upper and lower extremities correlates more closely with central SCI pain.[3] Subsequently, the patient decided against further intervention, and aggressive medical treatment was pursued.

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# ANNOUNCEMENT

The American Board of Anesthesiology will administer a written examination in CRITICAL CARE MEDICINE on Friday, September 8, 1995. Diplomates of the ABA who apply and are judged to be qualified by virtue of their additional training in the subspecialty will be accepted for examination. An application may be requested by writing to the Secretary, American Board of Anesthesiology, 100 Constitution Plaza, Hartford, Connecticut 06103-1796. The deadline for receipt of completed applications in the Board office is March 1, 1995.