CASE REPORTS

Anesthesiology
79:376–378, 1993

Evaluation of Central Spinal Cord Injury Pain with Diagnostic Spinal Anesthesia

Paul G. Loubser, M.B., Ch.B., Rebecca R. Clearman, M.D.†*

THE prevalence of chronic pain associated with spinal cord injury (SCI) varies considerably (5–94%).[1] Donovan *et al.* describe five categories of chronic SCI pain: musculoskeletal, segmental, central, visceral, and psychogenic.[2] In approximately 30% of SCI patients, pain is disabling, producing additional social handicaps.[3] Recently, attention has turned to identifying mechanisms of nociception and effective treatment modalities for neuropathic SCI pain.[4]

Spinal anesthesia has been used effectively to demonstrate the mechanism and origin of nociception in various pain conditions.[5] Furthermore, in SCI patients, it may elucidate the site of the "neural pain generator" with respect to the actual neurologic level of injury. However, in patients with cervical SCI, the use of diagnostic spinal anesthesia has not been well defined. The following report describes the response to diagnostic spinal anesthesia in a patient with chronic central pain associated with cervical SCI.

Case Report

A 53-yr-old man with a 2-yr history of C6 SCI secondary to a motor vehicle accident presented with chronic dysesthetic pain. In the lower extremities, pain was distributed from the knees to the soles of the feet bilaterally with radiation to the right anterolateral thigh region. Pain occurred mainly with movements (active/passive) or spasticity, and in the right thigh region was described as cramping in nature. In the upper extremities, dysesthesias were distributed from the elbows to the tips of the fingers and were associated with movements. Simple physical modalities such as physical therapy, local heat, and transcutaneous electrical nerve stimulation were ineffective in controlling pain. Similarly, various oral pharmacologic agents such as nonopioid analgesics (75 mg indomethacin twice daily, 50 mg diclofenac three times daily), anticonvulsants, (100 mg phenytoin three times daily, 300 mg carbamazepine three times daily), antidepressants (75 mg amitriptyline twice daily, 150 mg trazodone twice daily) provided only partial relief of pain. Though oral baclofen (20 mg four times daily) was effective in reducing pain by controlling spasticity, it produced functional impairments by decreasing lower extremity strength during attempted ambulation.

The patient's SCI produced an incomplete motor and sensory deficit below the neurologic level of injury. On examination, increased patellar reflexes, hip flexor spasticity, and ankle clonus were present bilaterally. Voluntary movements of the lower extremities such as hip and knee flexion were present bilaterally, though reduced in strength and limited functionally by hypertonicity. Sensory examination revealed the preservation of light touch sensation in the lower extremities, but loss of sensation to pinprick. No allodynia or hyperpathia was present in the lower extremities during cutaneous stimulation.

Clinical neurophysiologic measurements of sensory function were performed. This included a quantitative evaluation of temperature that compared the response to heat pain and cold pain in the lower extremities. In addition, quantitative evaluation of touch, graphesthesia, and joint position were recorded with a quantitative evaluation of vibratory perception thresholds. These evaluations detected absent temperature and thermal pain perception but preservation of some touch and vibratory perception below the C5 dermatome bilaterally. These findings demonstrated a more severe spinothalamic dysfunction in comparison to preservation of dorsal column function, consistent with a presentation of central dysesthesia syndrome.[6] A temporary trial of deep brain stimulation was recommended to the patient before surgical implantation of a thalamic electrode/stimulator ensemble. However, before this trial, confirmation of the central etiology of pain was sought with diagnostic spinal anesthesia.

A 23-G intrathecal catheter (Bizzarri-Guifridda, Becton-Dickinson, Franklin Lakes, NJ) was placed *via* a 20-G Tuohy needle in the L3–L4 lumbar interspace and threaded cephalad for 3 cm. Double-blind pain assessments (patient and nurse evaluating pain were both blinded to the injected agent) included a 10-cm visual analog scale and somatic diagram demonstrating spatial distribution of pain. Following aspiration of clear cerebrospinal fluid from the catheter hub, 2 ml of placebo (normal saline) was injected. The patient was monitored

* Associate Professor, Department of Anesthesiology, The University of Texas Health Science Center Medical School; Director of Anesthesiology Services, The Institute for Rehabilitation and Research, Houston, Texas.

† Assistant Professor, Department of Physical Medicine and Rehabilitation, The University of Texas Health Science Center, Houston, Texas.

Received from The University of Texas Health Science Center Medical School, The Institute for Rehabilitation and Research, and The University of Texas Health Science Center, Houston, Texas. Accepted for publication April 8, 1993. Supported in part by a grant from the National Institute on Disability and Rehabilitation Research, U.S. Department of Education, Grant #H133N00016-91 to Texas Model Spinal Cord Injury Center, The Institute for Rehabilitation and Research, Houston, Texas.

Address reprint requests to Dr. Loubser: Department of Anesthesiology, Baylor College of Medicine, Smith Tower, Suite 1003, 6550 Fannin, Houston, Texas, 77030.

Key words: Anesthetic techniques: spinal. Pain: chronic; thalamic. Spinal cord: injury.

for 30 min after injection of placebo, which failed to produce any change in the patient's pain status. Thereafter, 50 mg lidocaine in 7.5% dextrose was injected *via* the catheter. This was followed by the disappearance of spasticity, loss of voluntary movements, and disappearance of dysesthetic pain in the lower extremities. A sensory level (touch) of T8 was detected on the anterior abdomen. Clinical assessments were repeated every 20 min, and approximately 60 min after injection of intrathecal lidocaine, the patient's spasticity and pain status returned. In comparison, no changes in pain status or sensory or motor function were recorded in the upper extremities. The catheter was removed on completion of the study.

The response to diagnostic spinal anesthesia required reassessment of the central etiology of the patient's pain. In turn, the patient declined the temporary trial of deep brain stimulation, and instead, an aggressive course of conservative therapy was reattempted. This included high-dose amitriptyline (150 mg twice daily), group psychotherapy, and biofeedback relaxation. These measures produced some relief of pain, which the patient deemed compatible with normal activities of daily living. Other invasive modalities such as dorsal column stimulation or a temporary trial of intrathecal opioid also were declined by the patient.

Discussion

Several theories based on experimental data have been forwarded to explain the pathophysiology of central pain. The original suggestion by Melzack and Loeser,[7] that phantom pain in paraplegics developed secondary to "pattern generating mechanisms" occurring centrally in response to deafferentation, is widely regarded as the most plausible explanation. The term "central" is used in this context to denote a location within the dorsal horn and somatosensory projection systems, rostral to the actual spinal cord transection. Central dysesthesia syndrome was described by Beric *et al.*[6] in patients with SCI who manifest loss of spinothalamic but preservation of dorsal column function. Misinterpretation of residual dorsal column system input in the absence of suppression *via* integrated spinothalamic system activity within the thalamus or lower brainstem is thought to produce widespread dysesthesias.

Visceral/abdominal pain may occur in high-thoracic and cervical SCI patients regardless of varying degrees of sensory anesthesia and/or paralysis. Despite interruption of spinal sensory pathways, autonomic connections to the viscera and mesentery may remain intact, *e.g.*, *via* the vagus nerve or sympathetic chain. In most cases, a comprehensive diagnostic workup will indicate the source of pain, *e.g.*, gall bladder, bowel, bladder, or kidney, and appropriate treatment will alleviate pain. However, a study by Juler and Eltorai[8] of 36 SCI patients with visceral disease suggest that the diagnosis may be delayed in spinal injuries above the splanchnic outflow. Sometimes, no apparent etiology or source of pain can be identified. This form of chronic visceral pain is idiopathic and poorly understood. Little is known about its exact prevalence or its significance to patients with SCI; however, it may represent a form of central pain.

Local anesthetics injected in the lumbar intrathecal space accumulate along the dorsal and lateral portions of the spinal cord and nerve roots, areas that are heavily myelinated.[9] In 1951, Pollock *et al.*[10] described the use of diagnostic spinal anesthesia to elucidate the source of nociception in patients with SCI. In four patients, spinal anesthesia administered above the level of the lesion was associated with disappearance of pain. More recently, Loubser and Donovan[4] described the use of diagnostic spinal anesthesia *via* an intrathecal catheter in 21 patients with chronic SCI pain. The level of spinal anesthesia was titrated to anesthetize segments of the spinal cord below, at or above the SCI. Spinal anesthesia administered below the level of injury to three patients with cervical SCI having clinical symptomatology suggestive of central dysesthesia syndrome did not abolish pain.[4]

Recently, a positive response to diagnostic spinal anesthesia was demonstrated by Crisologo *et al.* in two patients with central post-stroke pain.[11] The present case report confirms Crisologo *et al.*'s findings and suggests that peripheral nociceptive mechanisms contributed significantly to the patient's symptomatology. According to Devor, neuropathic pain develops secondary to activation of low threshold mechanoreceptors, which then become functionally effective in driving the central pain circuit *via* dorsal horn cells.[12] In turn, spinal anesthesia may interrupt these low threshold afferents, thereby producing reduction of pain.

The response to diagnostic spinal anesthesia in this patient provoked a significant alteration in the planned management. Other possible nondestructive treatment alternatives aimed at modulating peripheral sensory input to the dorsal horn cells, such as epidural dorsal column electrical stimulation and intrathecal pharmacotherapy (opiates, adrenergic agonists/antagonists), may have a place in the treatment of this condition. These modalities are usually not indicated for central pain syndromes; however, a positive response to diagnostic spinal anesthesia might support further evaluation, *e.g.*, temporary percutaneous trials.

Also, systemic effect of lidocaine could not have accounted for the observed response,[13] because pain in the upper extremities was unchanged. The presence

of other peripheral nociceptive mechanisms such as sympathetically maintained pain seem unlikely in this patient, in view of the extensive clinical neurophysiologic testing, which clearly supported a central pain etiology.

In conclusion, this report provides further evidence that diagnostic spinal anesthesia may be associated with a positive response in central pain syndromes. This suggests that neural blockade with local anesthetics be incorporated into the diagnostic workup of patients with possible central pain syndromes. The response to neural blockade may either confirm a predominant central nociceptive mechanism or identify patients in whom peripheral sensory input contributes to the pain symptomatology, which may influence selection of long-term therapeutic options. Further clinical research is needed to characterize central pain syndromes in SCI patients and objectively evaluate long-term treatment modalities using neuraxial electrical stimulation or pharmacotherapy.

The authors gratefully acknowledge the secretarial assistance of Ms. Beverly Streety.

References

1. Burke DC, Woodward JM: Pain and phantom sensation in spinal paralysis, Handbook of Clinical Neurology: Injuries of the Spine and Spinal Cord. Part II. Edited by Vinken PJ, Bruyn GW, Braakman R. New York, Elsevier, 1976, pp 489–499

2. Donovan WH, Dimitrijevic MR, Dahm L, Dimitrijevic M: Neurophysiological approaches to chronic pain following spinal cord injury. Paraplegia 20:135–146, 1982

3. Botterell EH, Callaghan JC, Jousse AT: Pain in paraplegia: Clinical management and surgical treatment. Proc R Soc Lond [Biol] 47: 281–288, 1953

4. Loubser PG, Donovan WH: Diagnostic spinal anesthesia in chronic spinal cord injury pain. Paraplegia 29:25–36, 1991

5. Winnie AP, Collins VJ: Differential neural blockade in pain syndromes of questionable etiology. Med Clin North Am 52:123–128, 1968

6. Beric A, Dimitrijevic MR, Lindblom U: Central dysesthesia syndrome in spinal cord patients. Pain 34:109–116, 1988

7. Melzack R, Loeser JD: Phantom body pain in paraplegics: Evidence for a central "pattern generating mechanism" for pain. Pain 4:195–210, 1978

8. Juler GL, Eltorai IM: The acute abdomen in spinal cord injury patients. Paraplegia 23:118–123, 1985

9. Greene NM: Physiology of Spinal Anesthesia. Baltimore, Williams & Wilkins, 1981, pp 12–15

10. Pollock LJ, Brown M, Boshes B, Finkleman I, Chor H, Arieff AJ, Finkle JR: Pain below the level of injury of the spinal cord. Arch Neurol 65:319–322, 1951

11. Crisologo PA, Neal B, Brown R, McDanal J, Kissin I: Lidocaine-induced spinal block can relieve central poststroke pain: Role of the block in chronic pain diagnosis. ANESTHESIOLOGY 74:184–185, 1991

12. Devor M: Central changes mediating neuropathic pain, Proceedings of the Fifth World Congress of Pain. Edited by Dubner R, Gebhart GF, Bond MR. New York, Elsevier, 1988, pp 114–128

13. Day FJ: Value of spinal block in central pain (letter). ANESTHESIOLOGY 74:1167–1168, 1991

Anesthesiology
79:378–381, 1993

Indigo Carmine-induced Severe Hypotension in Patients Undergoing Radical Prostatectomy

Yoram Shir, M.D., Srinivasa N. Raja, M.D.†*

INDIGO carmine (indigotindisulfonate sodium, American Regent, Shirely, NY; IC) is a blue dye routinely used during urologic procedures to localize the ureteral orifices and to identify severed ureters and fistulous communications.[1,2] Sporadic cases of untoward reactions to IC following its intravenous administration—mainly hypertension and bradycardia[3–6,‡,§] but also

* Assistant Professor of Anesthesia.

† Associate Professor of Anesthesia.

Received from the Department of Anesthesiology and Critical Care Medicine, The Johns Hopkins University School of Medicine, Baltimore, Maryland. Accepted for publication April 8, 1993.

Address reprint requests to Dr. Shir: Department of Anesthesiology and Critical Care Medicine, The Johns Hopkins Hospital, 600 North Wolfe Street, Osler 304, Baltimore, Maryland 21205.

Key words: Hypotension. Indigo carmine. Radical prostatectomy.

‡ Schwerin GS: Severe reaction to indigo carmine. Illinois Medical Journal 101:48–49, 1952.

§ Wu CC, Johnson AL: The vasopressor effect of indigo carmine. Henry Ford Hospital Medical Journal 17:131–134, 1969.