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References

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Paraplegia in a Patient with an Intrathecal Catheter and a Spinal Cord Stimulator

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IMPLANTABLE techniques, such as spinal cord stimulators and intrathecal infusion pumps, have become increasingly popular in the contemporary management of chronic pain states. Potential reasons for the enthusiasm for these techniques are the reversibility of the methods, the relatively low rate of complications, and ongoing refinements in the techniques and devices. In addition, recent clinical reports using disinterested third-party interviews indicate that, when patients are appropriately selected, more than 50% of patients are likely to achieve long-term benefits from these devices.

Neurologic complications are among the most dreaded complications of devices implanted in the epidural or intrathecal space. These complications are particularly alarming when the implantable devices are used for the treatment of chronic nonmalignant pain. Fortunately, they are uncommon.

In this issue of *ANESTHESIOLOGY*, Aldrete *et al.* (page 1542) report a case associated with major morbidity in a patient who had both a spinal cord stimulator and an intrathecal infusion device implanted for the treatment of intractable thoracic pain of unclear etiology. Pharmacologic trials apparently having failed, he underwent a multilevel thoracic posterior rhizotomy. Subsequently, the patient had a spinal cord stimulator implanted above the level of the rhizotomy for persistent pain. The stimulator failed to relieve either the radicular or the midline back pain in this patient. Two aspects regarding the use of the spinal cord stimulator are not clear from this report. First, there is no evidence that a temporary (trial) electrode was placed before the permanent implantation of the spinal cord stimulator. The importance of temporary electrodes as a screening technique has been documented (North *et*

al., *Neurosurgery* 32:384-394, 1993). Second, because the patient did not obtain any significant relief of symptoms, it is unclear not only why the stimulator was implanted permanently but also whether it was still functional and was being used by the patient.

Approximately 3 months after the spinal cord stimulator implantation, a programmable drug pump was implanted and an infusion catheter was introduced into the subarachnoid space. Trial infusion results are not presented; apparently, the patient obtained "partial" relief of his symptoms with the infusion of preservative-free morphine supplemented with oral analgesics. However, about 3 months after the implantation of the infusion system, the patient presented with exacerbations of his thoracic spine pain that required an increase in the dosage of the morphine infusion. Three or four days later, sudden paraplegia developed below D7 level with complete sensory loss below D6. Despite an exploratory laminectomy, the patient had persistent paraplegia. Intraoperative findings were notable for the absence of pathology around the stimulator electrode and the presence of adhesive arachnoiditis, necrosis, and syrinxlike cavity formation of the spinal cord below the level of the tip of the spinal stimulator and in the region of the tip of the intrathecal catheter. The precise anatomic location of the catheter is not specified. Subsequent imaging studies done approximately 3 yr later showed atrophy of the thoracic spinal cord extending over several segments.

There are certain common features between this case and that reported by North *et al.* In both cases, approximately 2-3 months after the pump implantation, sudden paraplegia developed. Both patients had a prior implantation of a spinal cord stimulator that was not

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preceded by a percutaneous trial and was not very effective in controlling the pain. In the case reported by North *et al.*, however, the stimulator had been removed. This case raises a technical question as to the potential interactions between implanted devices used for pain control. There has been no report of interaction between intraspinal opioid infusion and spinal cord stimulation; neither this case nor the earlier case reported by North *et al.* suggest such an interaction. There is, however, potential concern that two programmable electronic devices and their external programming equipment might interact. Nonprogrammable devices can address this; so can parsimonious clinical practice. Both cases appear to be associated with local necrosis of spinal cord tissue following subarachnoid infusion.

The etiology of this serious complication is uncertain in both cases. Aldrete *et al.* suggest that the enthusiasm

for the use of new technical devices should be tempered by the seriousness of the potential risk associated with these methods. It is imperative that screening trials be used to determine the efficacy and possibly predict the subsequent outcome of the implanted devices. This case emphasizes the need for judicious use of the implantable techniques with appropriate patient and drug selection and periodic followup with neurologic evaluations. A cardinal rule is that there be an objective basis for a patient's complaint of pain; the underlying diagnosis in this case is never specified. These potentially morbid procedures should be performed not simply by technicians but by diagnosticians with multidisciplinary support.

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