HIGHLIGHTS

Intrathecal Magnesium Sulfate Protects the Spinal Cord from Ischemic Injury during Thoracic Aortic Cross-clamping

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VASCULAR anesthesiologists and surgeons have long sought for their Holy Grail: a means to decrease the incidence of paraplegia in patients undergoing thoracoabdominal aortic aneurysm repair. Depending on the location of the aneurysm, its extent, and whether it has dissected or is repaired emergently, the incidence of paraplegia can range from 2% to 40% with a mean incidence of 16%.[1]

In this issue of ANESTHESIOLOGY, Simpson *et al.* (page 1493) report remarkable results. In their canine model of ischemic spinal cord injury, the intrathecal administration of 3 mg/kg magnesium sulfate before thoracic aortic cross-clamping provided complete protection against ischemic injury; none of the eight animals receiving intrathecal magnesium had any measurable neurologic injury. Conversely, their control group of animals had an incidence of spinal cord injury of 87.5%

Simpson *et al.* give no explanation for why magnesium given in a very small volume (0.1–0.15 ml) into the cisterna magna should provide spinal cord protection from an ischemic insult. They speculate that magnesium decreases spinal cord metabolic rate or that it might inhibit N-methyl-D-aspartate (NMDA) activation, thereby preventing initiation of the excessive release of excitotoxic amino acids. Macintosh *et al.* reported that the administration of MK-801, an NMDA receptor blocker, promoted significant recovery of intracellular-free magnesium concentrations that decreased precipitously after brain injury.[2] However, Simpson *et al.* provide no evidence that this occurred in their animal model. They also hypothesize that perhaps the exogenous magnesium vasodilated cerebral vessels or prevented vasospasm of vessels supplying the spinal cord. Because nitroprusside, a known cerebral vasodilator, has been associated with an increase in intracranial pressure with a decrease in spinal cord perfusion pressure,[3] it is difficult to see how vasodilation could be responsible for these results. Whether vasospasm plays a role in spinal cord ischemia associated with thoracic aortic repair has not been reported previously. We are left then with an effect of magnesium on the NMDA receptor, though we can only speculate how the magnesium might have reached the distal spinal cord.

We can object to the small sample size in their study, the type of ischemic insult studied, *i.e.*, hemorrhage followed by 45 min of thoracic aortic cross-clamping, the unblinded nature of the study, and so on. In the final analysis, however, because both groups were treated in an identical fashion, except for the second group receiving intrathecal magnesium, we doubters must explain why the group given the intrathecal bolus of magnesium had such notable outcomes compared to the control group.

Ten years ago, Vacanti and Ames reported that mild hypothymia and intravenous magnesium protected against irreversible damage during central nervous system ischemia.[4] Because of hypothermia's well known protective effect in the central nervous system ischemia, many of us assumed that it was the hypothermia and not the magnesium that provided protection in the Vacanti and Ames' animal model. In the Simpson *et al.* study, the temperature was carefully controlled between both groups at 36°C. Robertson *et al.* reported results similar to Vacanti and Ames by administering magnesium intravenously in the absence of hypothermia.[5] Of nine rabbits given 100 mg/kg magnesium sulfate intravenously and subjected to 40–50 min of spinal cord ischemia, only 44% (four of nine) were normal. Of course, there may be species differences, and the magnesium was given intravenously and not intrathecally, but their results are not nearly as dramatic as those of Simpson *et al.*

The discrepancy between the Simpson *et al.* and the Robertson *et al.* studies underscores the importance of duplicating these results in a larger group of animals and perhaps with another animal model. If the results are duplicated, then while we attempt to identify magnesium's mechanism of action, clinical studies may be warranted given the relatively low incidence of adverse effects associated with parenteral magnesium administration, the ease of this particular approach (compared to regional spinal cord cooling, for example), and the potential risk-benefit ratio.

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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