Letters to the Editor

Dehydration: Cause of Acute Renal Failure

To the Editor:

I read with interest, the case report describing acute renal failure during spinal cord stimulation trial (1). The authors attributed the episode of acute renal failure to spinal cord stimulation. They speculated that decreased renal blood flow and peripheral vasodilation from sympathetic blockade by spinal cord stimulation contributed to acute renal failure.

The effects of spinal cord stimulation on renal blood flow have not been studied. Epidural anesthesia can produce sympathetic nerve system blockade. However, high epidural anesthesia (T6) does not result in a significant change in renal blood flow (2). The degree of sympathetic blockade by epidural anesthesia is higher than by spinal cord stimulation. Sympathetic blockade can cause peripheral vasodilation, which may contribute to hypotension. However, one study showed that spinal cord stimulation did not result in significant changes in mean arterial pressure (3). These studies did not support the theory advanced by Larkin, Dragovich, and Cohen.

I believe that the cause of acute renal failure in the Larkin et al case report was hypovolemia. This patient in their study demonstrated signs and symptoms of hypovolemia. He had postural dizziness, hypotension, increased heart beat, and tachypnea. The patient did not urinate for 2 days. After 3 liters of ringer's lactate, he had normal blood pressure and heart rate. He began to produce urine. His lab result returned to normal.

The most common cause of hypovolemia is dehydration. Preoperative dehydration is common in surgical patients due to preoperative fasting. The symptoms of dehydration may be most evident in minor surgical procedures, where intraoperative fluid requirements are low (4). Poor fluid intake postoperatively further intensified dehydration, and resulted in acute renal failure. Therefore, it is important to emphasize how to prevent perioperative dehydration after minor surgery.

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In Response:

About 50 years ago, reports began to trickle into the literature about depressed patients treated with tricyclic antidepressants and monoamine oxidase inhibitors whose chronic pain symptoms were also alleviated (1,2). Although most physicians were skeptical about a possible link between antidepressants and analgesia, anecdotal reports of inadvertent pain relief during the pharmacological treatment of depression continued to emerge, until in 1964 Lance and Curran published the first randomized controlled study demonstrating that TCA can effectively reduce pain in non-depressed patients (3). Today, TCA are considered a first line treatment for a wide array of chronic pain conditions (4,5).

This narrative is not intended to demean Dr. Huang or disparage his supposition, but rather to highlight two salient points. First, almost all serious complications from medical interventions are initially described via anecdotal reports similar to ours.6 Second, it would be imprudent to summarily dismiss any possibility of a link between spinal cord stimulation and renal failure just because it has never been reported. This would be akin to the position Merck initially took with rofecoxib and the possible association with an increased risk of cardiovascular events, and we all know how that turned out (7,8).

In conclusion, our manuscript was never intended to establish a definitive connection between spinal cord stimulation and renal failure. Instead, it was merely meant to raise the possibility of such an association. We concede the possibility that dehydration in and of itself could have caused our patient's renal failure, but at the same time we also believe it is in the best interest of our specialty not to discount the possibility that spinal cord stimulation did contribute to this complication. After all, time will be the final arbiter of the truth.

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