

depending on many variables, such as anesthesia techniques, baroreflex sensitivity, or underlying cardiovascular pathology. Therefore, as with most findings from laboratory studies, our findings should not be extrapolated simply to the clinical situation.

The statement by Dr. Hoka that "a maximal change in blood pressure in response to acute blood loss is an outcome of reduced blood volume and baroreflex-induced compensation" may not be true unless full baroreflex compensations (increases in peripheral vascular resistance, heart rate, and cardiac contractility) are achieved at the conclusion of acute blood loss. Our data show that the same amount of blood loss (5 ml/kg) over the same period (10 s) produced almost the same reduction of MAP regardless of whether dogs were in a normotensive state or under induced hypotension before acute blood loss, and regardless of which hypotensive agent was being used before acute blood loss. This indicates that maximum reduction of MAP secondary to acute arterial blood loss may not have been affected by the arterial baroreflex in our experimental procedures. This is because acute arterial blood loss was the initial event and presumably because baroreflex compensation had not been operating fully enough to modulate the impact of acute blood loss on the degree of maximum reduction of MAP. As a matter of fact, maximum reflex increases of HR and RSNA occurred after MAP had reached the lowest level following acute blood loss. We found that the maximum increases in HR and RSNA were greater with PGE<sub>1</sub> than with SNP and therefore stated that "baroreflex response to rapid blood loss was significantly greater during PGE<sub>1</sub> than with SNP-induced hypotension . . . despite the same baseline-induced hypotension . . . and the same degree of further hypotension from rapid blood loss." Although we only measured and evaluated maximum gains in HR and RSNA, a restoration of MAP may have been faster, cardiac output values may have been higher, and the renin-angiotensin system may have been activated more after reaching maximum reduction of MAP due to acute blood loss during PGE<sub>1</sub>-induced hypotension, as compared to SNP-induced hypotension.

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## Myofascial Pain Syndrome Can Cause Right Upper Quadrant Pain

*To the Editor:*—Haynsworth and Noe present an interesting case of right upper quadrant pain.<sup>1</sup> In their discussion of the causes of abdominal pain, "trigger points" are mentioned but myofascial pain syndrome (MFPS) is not. Trigger points associated with a MFPS are an important and often overlooked cause of somatic abdominal pain.<sup>2</sup>

Muscles having a referral pattern that includes the abdomen are the rectus abdominus, serratus anterior, external oblique, iliocostalis thoracis, erector spinae, and possibly the intercostal muscles.<sup>2-5</sup> The inter-spinous ligament, costal cartilage and lower intercostal joints may also refer pain to the upper abdomen.<sup>2,3</sup> Most of these structures are not located within the anterior abdominal area and therefore are not usually palpated during examination of the abdomen.

Visceral complaints as seen in their patient also have been noted as part of a MFPS.<sup>3-5</sup> Perhaps the reproduction of this patient's pain at the T11 area represented a trigger point in the serratus anterior or intercostal muscle. This undiagnosed MFPS may also respond to rhizotomy.

We believe that for completeness, the possibility of a MFPS and its appropriate treatment (TENS trial, trigger point injections, spray and stretch, and physical therapy) should be addressed before the diagnosis of intercostal neuralgia and the relatively destructive treatment of rhizotomy is suggested.

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*In Reply:*—Dr. Romanoff and Dr. Ellis are correct in stating that myofascial pain syndrome is a common cause of abdominal wall pain. In fact, it is probably the most common cause of abdominal wall pain seen in our clinic. Conservative treatment with trigger point injections, spray and stretch, and physical therapy is a very reasonable option in the patient in whom muscle tenderness, trigger point areas, or a history suggesting increased pain with muscle movement are identified. However, our patient did not have this type of symptomatology elicited either by history or physical exam. There were indeed no specific tender muscle regions or trigger points identifiable to inject or treat with spray and stretch techniques and physical therapy.

It was not the intent of our case report to discuss etiologies of abdominal wall pain. The etiology of pain in our patient still remains unknown. Because the pain resolved with local anesthetic blockade of a single intercostal nerve, it was believed that this pain was secondary to an area of irritated peritoneum innervated by a single intercostal nerve, or entrapment of the nerve itself. It is unusual for pain secondary to myofascial pain syndrome to resolve with blockade of a single intercostal nerve, because of the overlap in innervation to the abdominal wall musculature.

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This case serves as a good example to remind practitioners that patients with visceral-type symptoms may have etiologies arising from structures outside the abdominal cavity, and this was our primary intent. We also wanted to show that partial rhizotomy of an intercostal nerve is an alternative to phenol or alcohol neurolytic techniques.

Interestingly, recent follow-up shows that this patient remains pain-free at 12 months and has required no further hospitalizations or pain medications.

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## Nerve Stimulation and Residual Neuromuscular Block

*To the Editor:*—The recent paper by Pedersen *et al.*,<sup>1</sup> raised many interesting and troubling questions. The investigators designed a protocol where ten anesthesiologists were not blinded to the purpose of the study. The latter centered on two groups of patients undergoing gastrointestinal surgery, where a peripheral nerve stimulator was used in one group and not in the other.

The anesthesiologists, described as experienced in the use of a peripheral nerve stimulator, were told to maintain relaxation with either pancuronium or vecuronium at a level such that one or two responses to train-of-four (TOF) were felt. The same anesthesiologists were instructed to give the relaxant to the other group only on the basis of detection of spontaneous muscle activity (I suppose: movement, spontaneous breathing, or tightening of abdominal wall). These patients were maintained on 66% nitrous oxide in oxygen and minimal fentanyl (50 µg), given only if the systolic blood pressure and heart rate exceeded 30% of control. These anesthesiologists also were instructed to reverse the block with 2.5 mg neostigmine and had an option to use an additional two doses of 1.25 mg each, only when spontaneous breathing or other muscle activity and/or the presence of one or two responses to TOF could be demonstrated. They even were given the criteria the investigators considered sufficient for recovery following reversal, *i.e.*,

sustained head lift with no manually detectable fade to TOF in the monitored group or sustained head lift in the nonmonitored patients.

As clinicians, we would have predicted that all patients in the four groups would have completely recovered neuromuscular function following the conditional reversal, taking into consideration the small doses of either relaxant administered (table 2 in their article) for procedures lasting over 3 h in the absence of potent inhalation anesthetics. We also suspect that these patients would have met the above-mentioned criteria of neuromuscular recovery before going to the recovery room (RR), especially during the 15–33-min waiting period in the operating room (OR) following the end of surgery.

It is difficult therefore to reconcile the differences between the OR events and the investigators' findings in the RR. Ten patients in the RR were found to have residual blockade (unable to head lift for 5.0 s), and 17 patients required an additional supplemental dose of neostigmine despite all the restrictions on relaxant dosage and full reversal following these lengthy procedures. Could it be that the neuromuscular block was overreversed?<sup>2,3</sup> Electromechanical twitch recordings of TOF ratios in the RR were also of concern. One patient in group 1 was found to have a TOF ratio of 0.06. How can this be missed in the OR by the experienced anesthesiologists who evaluated tactile TOF fade