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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.

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.

CARL E. NOE, M.D.
Associate Attending
ROBERT F. HAYNSWORTH, JR., M.D.
Associate Attending
Department of Anesthesiology
Baylor University Medical Center
3707 Gaston Avenue, Suite 712
Dallas, Texas 75246

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Nerve Stimulation and Residual Neuromuscular Block

To the Editor:—The recent paper by Pedersen *et al.*,[1] 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?[2,3] 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