REFERENCES

1. Haynsworth RF, Noe CE: An Unusual Presentation of Intercostal Neuralgia. ANESTHESIOLOGY 73:779–780, 1990
2. Travell JG, Simons DG: Myofascial Pain and Dysfunction: The Trigger Point Manual. Baltimore, Williams and Wilkins, 1983
3. Lewis T, Kellgren JH: Observations Relating to Referred Pain, Viscero-Motor Reflexes and Other Associated Phenomena. Clin Sci 1:47–71, 1939
4. Harman JB, Young RH: Muscle Lesions Simulating Visceral Disease. Lancet 238(1):1111–1113, 1940
5. Young D: The Effects of Novocaine Injections on Simulated Visceral Pain. Ann Intern Med 19:749–756, 1943

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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.

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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

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and head lift? This patient may not even have had a fourth response in the OR. Three patients in group 4 had recorded TOF ratios of 0.1, 0.4, and 0.43, yet none had signs of residual paralysis. We suspect too that these ratios were much lower in the OR or induced by an overzealous reversal. In addition, 35 patients (more than 43%) arrived at the RR with recorded TOF ratios < 0.7, suggesting either inaccurate assessment in the OR, faulty monitoring techniques, additional unwarranted reversal given in the OR or the RR, or the inadvertent peripheral cooling to 23° C.

In summary, we do not find any basis or data to support the misleading conclusion that perioperative manual evaluation of the response to TOF stimulation influenced neither the dose of the relaxant nor the frequency of postoperative residual paralysis, which clearly contradicts an earlier report by the senior author, Dr. Viby-Mogensen.[4]

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REFERENCES

1. Pedersen T, Viby-Mogensen J, Bang U, Olsen NV, Jensen E, Engbæk J: Does perioperative tactile evaluation of train-of-four response influence the frequency of postoperative residual neuromuscular blockade? ANESTHESIOLOGY 73:835–839, 1990
2. Chang CC, Chen SM, Hong SJ: Reversal of the neostigmine induced tetanic fade and endplate potential rundown with respect to autoregulation of transmitter release. Br J Pharmacol 95: 1255–1261, 1988
3. Goldhill DR, Wainwright AP, Stuart CS, Flynn PJ: Neostigmine after spontaneous recovery from neuromuscular blockade. Effect on depth of blockade monitored with train-of-four and tetanic stimuli. Anaesthesia 44:293–299, 1989
4. Viby-Mogensen J, Jorgensen BC, Ording H: Residual curarization in the recovery room. ANESTHESIOLOGY 50:539–541, 1979

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In Reply:—We appreciate the interest of Dr. Ali and Dr. Shorten in our work. Not unexpectedly they have difficulty accepting the results of our study. So had we initially! As would appear from our paper, we were surprised by our findings. After hours of checking all our recordings and data and hours of discussion, we finally had to accept that the data were correct.

Considering our more than 10 yr of experience in monitoring neuromuscular blockade, we do not think that our results are caused by "faulty monitoring techniques." There is no doubt though that the low peripheral temperatures in some patients may have influenced the measured train-of-four (TOF) ratios. We have, over the last few years, many times seen significant fade in a TOF response at low peripheral temperatures in the absence of neuromuscular block. Also, Eriksson *et al.*[1] have recently shown that in the absence of a neuromuscular blocking agent, a linear relationship exists between peripheral skin temperature and the TOF response. For instance, at a peripheral skin temperature of 27° C, *i.e.*, 4° C above the 23° C claimed by Dr. Ali and Dr. Shorten not to influence TOF ratio, a mean (±2 standard deviations) decrease in TOF ratio and twitch height of 0.10 (±0.10) and 20% (±10%), respectively, were found. The great impact of peripheral temperature on the TOF ratio also is illustrated from observations in two patients (included in our study but not described in detail) in which TOF ratios were recorded simultaneously from both arms. Differences in temperature between arms of only 2.8 and 3.3° C resulted in differences in TOF ratios of 0.39 and 0.36, respectively. It is of course unfortunate that the peripheral temperature in some of our patients was so low—a fact that led us to consider not to publish the results. However, the study involved the actual situation seen in everyday work in many operating theaters; the study was performed at a time when we did not know as much as we know today about the influence of peripheral temperature on monitoring (in fact, it was the current study that prompted our studies of this problem); and patients with low peripheral temperatures were evenly distributed among the four groups of patients, and there was no statistically significant differences in peripheral (or central) temperatures among the groups. It is therefore unlikely that peripheral cooling influenced the main conclusion: no clinical effect could be documented of perioperative manual evaluation of the response to TOF nerve stimulation.

Dr. Ali and Dr. Shorten ask whether it could be that some patients' neuromuscular blocks were overreversed at arrival in the recovery room. We do not think so. Only 6 of the 17 patients given supplementary doses of neostigmine (3 monitored and 3 not monitored with a nerve stimulator) showed signs of residual neuromuscular blockade evaluated clinically, and only 4 patients received more than 0.06 $mg \cdot kg^{-1}$. In every case when a supplementary dose of neostigmine was given, the neuromuscular transmission actually improved, indicating insufficient reversal.

Also, Dr. Ali and Dr. Shorten ask, how can a TOF ratio of 0.06 (or for that matter 0.1 or 0.2) be missed in the operating room? We wondered too! However, considering that even at a normal peripheral temperature, it may not be possible to feel fade at a TOF ratio of 0.4, the low peripheral temperature at reversal may at least partly explain why some apparently low TOF ratios were missed by the anesthetist. Further, in the patient with a TOF ratio of 0.06, peripheral skin temperature decreased from 30.5° C at the end of anesthesia to 23.9° C in the recovery room. TOF ratio in this patient may therefore have been higher in the operating room. The decline of TOF ratios during transportation from the operating room to the recovery room has been described by Mitterschiffthaler *et al.*[2]

The finding of more than 40% of the patients arriving in the recovery room with a TOF ratio of less than 0.70 is of no surprise to us. Similar results have been published by Bevan *et al.*[3] and Viby-Mogensen *et al.*[4] In these two studies no notice was taken of the peripheral temperature.

The protocol used was intended to closely simulate every day clinical work, as it is practiced in Scandinavia, when a nerve stimulator is and is not in use (groups 1 and 2 and groups 3 and 4, respectively). The anesthetists were told to "do it as well as possible," whether or not they had access to a nerve stimulator. Under these circumstances, we could not document an effect of perioperative manual evaluation of the response to TOF.

There must be some misunderstanding that our conclusion "clearly contradicts an earlier report by the senior author, Dr. Viby-Mogen-

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