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]

HASSAN H. ALI, M.D.
Associate Professor of Anesthesia
Harvard Medical School

GEORGE SHORTEN, F.F.A.R.C.S.(I.), FCANAES.
Clinical Research Fellow

Department of Anesthesia
Massachusetts General Hospital
Boston, Massachusetts 02114

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

(Accepted for publication January 25, 1991.)

Anesthesiology
74:957–958, 1991

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-

sen."[4] Our study on residual paralysis in the recovery room from 1979 did not include any patients monitored with a nerve stimulator.

It is very difficult to document an effect of any monitoring device, and our study design may not have been ideal. However, as advocates for the use of monitoring devices, it is not enough that we *claim* benefit for the patients or ourselves from the use of these devices. We do have an obligation to try to prove or disprove that we are correct in our assumptions. Our study was an attempt to evaluate the effectiveness—as opposed to the efficacy—of tactile evaluation of the TOF response in daily clinical work. Other studies in this direction are in progress.

TOM PEDERSEN, M.D.
ULLA BANG, M.D.
NIELS V. OLSEN, M.D.
ERIK JENSEN, M.D.
JENS ENGBÆK, M.D.
JORGEN VIBY-MOGENSEN, M.D.*
Departments of Anaesthesia
*Herlev Hospital and *Rigshospitalet*
University of Copenhagen
DK-2100 Copenhagen, Denmark

REFERENCES

1. Eriksson LI, Jensen E, Viby-Mogensen J, Lennmarken C: Train-of-four (TOF) response following prolonged neuromuscular monitoring: Influence of peripheral temperature (abstract). ANESTHESIOLOGY 71:A827, 1989
2. Mitterschiffthaler G, Khunl-Brady KS, Koller J, Mair P, Wieser Ch, Wierda M: Residual neuromuscular blockade: A comparison of vecuronium (Vc), atracurium (Ac), alcuronium (Alc), pancuronium (Pc), and pipecuronium (Pip). Acta Anaesthesiol Scand 33(suppl 91):140, 1989
3. Bevan DR, Smith CE, Donati F: Postoperative neuromuscular blockade: A comparison between atracurium, vecuronium, and pancuronium. ANESTHESIOLOGY 69:272–276, 1988
4. Viby-Mogensen J, Jorgensen, BC, Ording H: Residual curarization in the recovery room. ANESTHESIOLOGY 50:539–541, 1979

(Accepted for publication January 25, 1991.)

Anesthesiology
74:958–959, 1991

Transesophageal Echocardiography in Dogs

To the Editor:—We read with interest the recent editorial by Vandenberg and Kerber[1] concerning the use of transesophageal echocardiography (TEE) for intraoperative monitoring of left ventricular function. The authors state that "experimental animal studies of ischemia and infarction have not been performed with TEE" because "the canine left ventricle does not image well by TEE." We wish to call attention to the fact that high-quality TEE images of the left ventricle *can* be obtained in the dog. The use of a canine model offers an excellent opportunity to study TEE under conditions of altered hemodynamics or graded myocardial ischemia and thereby address some of the unresolved questions discussed by Dr. Vandenberg and Dr. Kerber.

Long-axis views of the left ventricle can easily be obtained in the dog in the same manner as they are in humans, *i.e.*, by positioning the transducer behind the left atrium and angling it in a caudad direction (toward the cardiac apex). The difficulty comes when attempting to obtain short-axis, midpapillary views analogous to those reported in the human investigations of ischemia. This is because the dog has an accessory lobe of the right lung interposed between the esophagus and the inferior wall of the left ventricle,[2] blocking acoustic transmission from the esophagus. We have reported a method to obtain short-axis views in the dog—by performing a right thoracotomy or median sternotomy, retracting the accessory lobe with an umbilical tape, excising the pleural attachments to the posterior pericardium, and filling the resulting space with an acoustical transmission medium (*e.g.*, saline).[3] We have obtained excellent images[3] and other derived data[4,*] from dogs in this manner. From a review of the literature, we also found that other quadrupeds, many sea mammals, and primates (other than the great apes) have similar anatomy to the dog in this regard.[5]

Short-axis imaging in dogs may actually offer advantages over human imaging. First, the wedge shape of the accessory lobe causes the apex of the dog's heart to angle away from the esophagus. This tends to make the dog's epicardial outline appear entirely within the sector of the ultrasonic scan, unlike in humans, in whom part of the epicardium often lies outside the visible sector.[5] Having the full epicardial outline available would make it easier to assess wall thickening in conjunction with wall motion analysis. In addition, it has been our impression that the cardiac apex is more readily visualized in dogs than it is in humans.

The belief that short-axis TEE imaging of the dog's left ventricle is difficult appears to be widely held, inasmuch as we have been unable to find any publications (other than our own) in a Medline search on the subject. We hope that these observations will encourage other investigators to consider using TEE in the animal laboratory.

G. BASHEIN, M.D., PH.D.
Associate Professor of Anesthesiology and Bioengineering (Adjunct)
ROY W. MARTIN, PH.D.
Research Professor of Anesthesiology and Bioengineering
University of Washington
Seattle, Washington 98195

REFERENCES

1. Vandenberg BF, Kerber RE: Transesophageal echocardiography and intraoperative monitoring of left ventricular function (editorial). ANESTHESIOLOGY 73:799–801, 1990
2. Evans HE, Cristensen GC: Miller's Anatomy of the Dog. Second edition. Philadelphia, WB Saunders, 1979, p 540

* Martin RW, Graham MM, Kao R, Bashein G: Measurement of left ventricular ejection fraction and volumes with three-dimensional reconstructed transesophageal ultrasound scans: comparison to radionuclide and thermal dilution measurements. J Cardiothorac Anesth 3: 260–268, 1989.