appropriate use of body ventilators,\textsuperscript{4,5} and mechanical insufflation-
exsufflation for eliminating airway secretions\textsuperscript{6,7} are all necessary.

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To the Editor:

We were and are very much aware of Dr. Bach's contribution to the literature on noninvasive ventilation in patients with neuromuscular respiratory failure. We had no intention to obscure that; in fact, our original submission was approximately twice as long, with a proportionally larger number of references, including several of those he cites. Despite this, his work is cited five times in our list of references. However, although Dr. Bach's work is well known to us and to the relatively small group of physicians interested in long-term ventilation in general, and in neuromuscular patients in particular, it is our perception that many general pulmonologists are less familiar with the options available.

The point of our report was the presentation of the first well-documented case of conversion of tracheostomy to nasal positive pressure ventilation (NPPV). We were aware of and cited Bach's reference 10; we found it difficult to sort out the patients receiving NPPV, and did not regard their course as "well documented." The other reports address other forms of noninvasive ventilation, with which our report was not concerned. One portion of our article that he quoted should have read " . . . with substitution of this invasive method . . . " which represents a copy editing error on our part.

As regards the remainder of the letter, we are aware of and applaud Dr. Bach's remarkable success with mouth IPPV and intermittent abdominal pressure ventilation. Interestingly, our experience with tracheostomy positive pressure ventilation does not seem to be as unfavorable as his, and, assuming good quality speech via cuff-down ventilation with or without one-way valve, our muscular dystrophy patients have found tracheostomy positive pressure ventilation quite satisfactory. Obviously, we cannot rival Dr. Bach's sheer volume of experience in this regard (can anybody)!

Finally, while daytime P\textsubscript{CO\textsubscript{2}} elevation implies the need for partial ventilatory support in neuromuscular disease patients, we have not found that mild to moderate elevations in P\textsubscript{CO\textsubscript{2}} predict need for increasing ventilatory support of those receiving nocturnal ventilation in the short to intermediate term. Indeed, we have several such patients with vital capacities of less than 15 percent predicted who have been comfortably maintained on nocturnal support only for several years.

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Neuromuscular Blockade in the ICU

To the Editor:

Although neuromuscular blocking agents (NMBAs) are commonly used in ICUs, the use of peripheral nerve stimulators to monitor the depth of neuromuscular blockade is not routine.\textsuperscript{1} Little information is available regarding the proper use of the peripheral nerve stimulator, the standard of care for monitoring paralysis, despite the fact that incorrect use can lead to inaccurate dosing of NMBAs as Isonstein et al\textsuperscript{2} noted in the October 1992 issue of Chest. We began a brief study after noting difficulty in obtaining a response to peripheral nerve stimulation in a patient with massive arm edema.

The aim of the study was to determine the effect of peripheral edema on the train-of-four stimulation.

We monitored neuromuscular function in two ICU patients with anasarca: a 50-year-old Hispanic male AIDS patient with respiratory failure and a 78-year-old white man with COPD. Neither patient was receiving an NMB or had a peripheral neuropathy. Each patient had two standard 5×6.5-cm ECG electrodes placed over the left ulnar nerve distribution. The electrodes were connected to a peripheral nerve stimulator (MiniStim, Life-Tech Inc, Houston) via wires fitted with alligator clamps. Thumb adduction was monitored after train-of-four stimulation. In both patients, a train-of-four stimulus at maximal stimulus amplitude (50 mA into a 2,000-ohm load) did not elicit a response. With the electrodes remaining in place, the patient's forearm was wrapped in Ace bandages and elevated to decrease edema. After about 2 h, the patients had a decrease in arm edema from 2+ to trace. The patients' arms were returned to the initial position, and train-of-four stimulus was repeated. Both patients regained normal response to submaximal electrical stimulation. The same protocol was repeated on the right arm of each patient, with reappearance of a twitch response after resolution of edema.
We postulate that as peripheral edema increases the distance between the nerve and the electrodes, the resistance increases and the amount of voltage required for nerve depolarization will also increase according to Ohm's law. This relationship may be similar to that of transthoracic resistance and defibrillation current. Human studies have shown that transthoracic resistance increases with chest size and decreases with increased contact pressure between paddles and chest wall. In the patient with COPD, when contact pressure between electrodes and skin was increased, a train-of-four stimulation elicited a twitch response despite massive edema.

Edema is a major problem in the ICU and accumulates quickly in critically ill patients secondary to a number of factors, e.g., low albumin, increased venous pressure secondary to mechanical ventilation, and massive volume resuscitation. Therefore, the absence of a twitch response in patients with new or persistent edema may erroneously suggest overparalysis. Consider the effect of peripheral edema on resistance. Perhaps in patients with peripheral edema the extremity should be elevated to minimize the edema, or alternate monitoring sites, such as the facial nerve, should be considered.

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To the Editor:

We think that the observations of Drs. Casale and Siegel are very important. There are many patients in whom it may be very difficult to obtain an accurate response to wrist-nerve stimulation, especially those with massive arm edema (and those with cold extremities). Furthermore, it is a common finding to detect a change in response when pressure is applied to one or both of the electrodes applied to the wrist for nerve stimulation. However, we have some differences in opinion as to the underlying biophysical principles and explanations for their findings regarding edema and electrode pressure.

The main resistance between the two stimulating electrodes is determined by the electrode-skin interface (ie, metal anode/cathode, gel, and skin) and the volume of underlying subcutaneous tissue (Fig 1 and 2). These two elements are in series. The volume of underlying subcutaneous tissue in turn is made up of extracellular fluid, fat, muscle, and nerve—all in parallel. The current that is supplied by the stimulator (of either constant-voltage or constant-current type) is thus divided between the two parallel elements—the nerve and nonnerve subcutaneous compartments (which are in parallel). Of the total stimulator current, it is likely that only a small part actually stimulates the nerve by activating one or more areas of electrically excitable membrane. Most of the current that passes in or out of the skin is shunted around the nerve by the nonnerve subcutaneous tissue.

As edema forms in the subcutaneous wrist tissue, the shunt resistance (R) decreases and thus shunts current away from the nerve (Fig 2). The nerve is farther from the skin (as suggested by Casale and Siegel), thus decreasing its "share" of the total current in the subcutaneous compartment. Both of these factors (ie, increased shunting and increased distance) cause less stimulation of the nerve.

Without very careful measurements, it may be difficult to know whether the total current from the stimulator goes up or down with edematous conditions (this, in turn, also depends in part on the type of stimulator—constant current or constant voltage).

As an explanation of increased twitch with pressure applied to the electrodes: (1) This pressure probably decreases the electrodeskin resistance (eg, by thinning the gel or squeezing out air, thus decreasing the R-skin/electrode in Figure 2); and this pressure decreases the distance from the skin to the nerve (eg, squeezes out subcutaneous fat, thus decreasing the R-series in Figure 2). Both of these factors tend to increase the amount of current reaching down to and stimulating the nerve in the subcutaneous compartment.

The variability of the response of the nerve secondary to such factors as edema and cold and NMBA administration is the main rationale for developing strategies such as "train-of-four" and "tetanic-fade" as attempts to use the pattern of the response (vs the amplitude) in order to titrate the level of NMBA dosage. Therefore, it is very important for those in an ICU setting to become familiar with such techniques before attempting to use electrical stimulation to titrate the level of NMBA (eg, from amplitude alone).

As an addendum, patients with very large wrists for any reason (eg, edema or large size) almost certainly need more current for adequate stimulation than is available from the usual clinical stimulators. That is, the effects of the increased size (mainly the increased shunting around the nerve) should theoretically be overcome by increasing the strength of the stimulus, with due caution.

FIGURE 1. Geometry of the relationships between the stimulator, skin, and wrist nerve. Note that part of the current passing from the stimulator and through the skin never passes through the nerve and that stimulation of the nerve occurs near the cathodal electrode.

FIGURE 2. Electrical equivalent of Figure 1. Each of the circuit components (resistances) can vary somewhat independently with events such as edema and pressure applied to the electrode. Sub-Q and S.Q. = subcutaneous.

1640

Communications to the Editor