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, eg, 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 electrode-skin 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
regard for appropriate electrode size and care of placement in order to prevent skin burn due to too high a current density. Unfortunately, such stimulators are usually available for research use only.

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Never Use Neuromuscular Blocking Agents for Sedation

To the Editor:

We read with appreciation and concern the review article by Isenstein et al., which appeared in the October 1992 issue of Chest. The authors have provided clinically relevant information that is familiar to most anesthesiologists, but it may not be the case for other specialists. With more newly available drugs and better monitoring techniques, there is a current trend toward increasing usage of neuromuscular blocking agents (NMBAs) in the ICU. Unless they are applied appropriately for specific indications, however, the resurgence of the use of these agents may not be a welcome sign. As mentioned in the Isenstein article, in addition to failure of coughing during tracheal suctioning and prolonged paralysis, there may also have been direct damage to the neuromuscular junction.1,2 Prolonged use of NMBAs may be associated with prolonged neuromuscular dysfunction with neurogenic or myopathic abnormalities as shown on electrodiagnostic studies and muscle biopsies. Recovery of strength may take months, and weaning from a mechanical ventilator can become difficult. The risks of nosocomial infection and disuse atrophy are increased as well.3-5 Gooch et al6 suggested that the blockade of neuromuscular transmission might sometimes be severe enough to lead to actual denervation of some muscle fibers.

Nevertheless, our major concern is what we believe to be an inadvertent mistake made by the authors. We strongly feel that the subject title Sedation under the section of Indication is inappropriate, misleading, and needs to be corrected. It should be replaced with something like "Paralysis for Diagnostic and Therapeutic Procedures." It is well known to anesthesiologists as well as intensivists, but not to other physicians, that NMBAs not only do not reduce anesthetic requirement but also do not cause sedation or amnesia at all.6,7 It is paralysis not sedation that NMBAs can provide. This point cannot be overemphasized or a disaster may occur, particularly if mechanical ventilation is not instituted. Adequate medications for amnesia and sedation should always be provided concomitantly. Without them, tremendous stress reaction under that situation can be extremely harmful to certain patients. A syndrome of traumatic neurosis can also be induced by awakened paralysis due to NMBAs.8 For those who are not acquainted with NMBAs, nothing is more dangerous than using them for sedation.

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REFERENCES

Use of Heparin in Prevention of Venous Thromboembolism

To the Editor:

In an article that appeared in the supplement to the October 1992 issue of Chest, Clagett et al1 provide recommendations for prophylaxis against venous thromboembolism in various patient populations at risk for this serious complication. I found this extensive article quite informative.

I was concerned to find that the authors stated in their "Summary and Recommendations" section regarding spinal cord injury (SCI) that low-dose heparin (LDH) "appears ineffective and is not recommended." Many prior observations from SCI centers in the United States and throughout the world suggest the efficacy of LDH. Virtually all SCI patients admitted to our SCI rehabilitation unit are given LDH (every 8 h) prophylaxis unless contraindicated. In over 280 patients admitted to the unit in the past 4½ years, fewer than 12 percent have developed clinically evident deep venous thrombosis (DVT), and fewer than 5 percent have developed pulmonary embolism (PE); there have been no deaths in the patients with these complications. Roughly one half of those diagnosed with DVT or PE were diagnosed either prior to admission or at the time of admission, and most of these patients had not received this prophylactic regimen. Although I realize that this is not a controlled study, I am not yet convinced that LDH given every 8 h is ineffective prophylaxis, and am disturbed regarding the summary detailed.

The authors cite in support of their conclusions three studies, which compare LDH to controls,2 to adjusted-dose heparin,3 and to low-molecular-weight heparin (LMWH).4 I assume that the authors include both every-12-h and every-8-h dosing in the definition of LDH used in their recommendations, as both regimens were employed in the studies cited. They cite a small study by Frisbie and Sashara,5 who found no difference in the incidence of DVT in patients given 5,000 U of subcutaneous heparin every 12 h and in those who received no prophylaxis as observed using impedance plethysmography. They did not mention that the authors themselves felt that prophylaxis for DVT in this prospective study could not be assessed due to the low incidence of DVT found in the control subjects;6 they reported about a 60 percent incidence in a prior study.

This variability in incidence of thromboembolic complications observed within institutions is common, as reported in the literature. Watson7 has shown a highly variable incidence seen yearly in his SCI patients (6 to 25 percent for DVT; 0 to 18 percent for PE). This is also evident at my institution. This variability in incidence makes it extremely important to utilize larger populations when comparing prophylactic regimens.

Clagett et al cite in support of the use of adjusted-dose heparin the study by Green et al8 at the Midwest Regional SCI Care System, who compared every-12-h dosing of LDH with adjusted-dose heparin. The incidence rates in the LDH and adjusted-dose heparin groups were 31 percent and 7 percent, respectively. It should be noted that in 24 percent of the patients treated with adjusted-dose heparin, treatment was interrupted due to bleeding. The authors

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