Assessment of Neuromuscular Block: Aspects of Stimulation

To the Editor.—As a neurologist and electromyographer, I found Brull and Silverman's article to underscore a lack of communication between medical specialties. Several of the findings described in this study are previously reported, accepted tenets of electrophysiological medicine. With regard to the electrode polarity of the stimulator, the authors found that placement of the cathode distal to the anode evokes a greater response. In agreement with this finding and extending it, current textbooks of electromyography state that, in performing motor nerve and repetitive stimulation studies, the cathode must be distal to the anode. This stimulus orientation avoids a failure of impulse propagation termed "anodal block." Anodal block occurs when an impulse that originates beneath the cathode reaches the hyperpolarized segment beneath the anode and is not propagated across this depolarization-resistant segment. Inadvertent placement of the anode between the stimulating and recording electrodes may result in submaximal stimulation and a technically suboptimal study.

With regard to the stimulus intensity required to elicit a supramaximal response, Brull and Silverman found an increasing response amplitude with increasing total stimulus charge, above charge levels previously thought sufficient. In electromyography, the charge required for supramaximal stimulation is recognized to be highly variable. Current practice in electromyography is to individually find the maximal stimulus for each nerve in each patient by progressively increasing the stimulus intensity and duration until the evoked compound muscle action potential (CMAP) no longer increases in amplitude. A supramaximal stimulus is obtained by increasing the maximal stimulus intensity by 20% to ensure recruitment of all the axons in a nerve. This supramaximal stimulus intensity is used for obtaining recordings in which the data are used. With stimulation of the ulnar nerve at the wrist, especially in the presence of neuropathy or local tissue changes, a current intensity of 100 mA with a duration of 0.2 ms or less, frequently 0.5–1.0 ms may be required for supramaximal stimulation. In determination of the maximal stimulus, care must be taken to evaluate the morphology of the evoked waveform to ensure that tissue conductance of the stimulus did not cause excitation of the nearby median nerve. If median innervated thenar muscles are recruited, the evoked CMAP cannot be used because simultaneous supramaximal stimulation of both the median and ulnar nerves cannot be ascertained.

Increased information exchange between electromyographers and anesthesiologists likely would prove beneficial for practitioners of both fields.

Howard W. Sander, M.D.
Assistant Professor of Neurology
New York Medical College
Director of the Electromyography Laboratory
Department of Neurology and Division of Clinical Neurophysiology
Saint Vincents Hospital of New York
New York, New York 10011

References

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In Reply.—We would like to thank Sander for his letter regarding an important (and still not widely understood) topic, the use of electromyography in clinical anesthesia. We wholeheartedly agree with his perception of the "lack of communication between medical specialties."

Although some of the information in our article may have appeared previously in the textbooks of electromyography cited by Sander, the paucity of original investigations published in the anesthesia (or neurology) literature with regard to the effect of electrode polarity on the evoked response was evident.

With regard to the stimulus intensity required to elicit a supramaximal response, the classic anesthesia teaching is to determine the maximal evoked response for each patient by progressively increasing the stimulus intensity and then increasing it further by 20–50% to ensure "supramaximal stimulation." However, contrary to our findings, the understanding had been that, in general, currents
much less than 100 mA were sufficient. This contention was supported by the fact that the vast majority of the clinically available nerve stimulators have maximal current outputs of only 60–70 mA. Furthermore, only a couple of the newer nerve stimulators have the ability to vary the stimulus pulse duration, and only between 0.2 and 0.5 ms.

In conclusion, we are encouraged by the fact that a nonanesthesiologist has taken interest in our literature and that our findings are supported by an expert electromyographer; we are indebted to Sander for his correct call to increase information exchange among medical fields.

Sorin J. Brull, M.D.
David G. Silverman, M.D.
Department of Anesthesiology
Yale University School of Medicine
New Haven, Connecticut 06520-8051

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Cerebral Oxygenation during Hypothermic Cardiopulmonary Bypass: Clinical Findings Support Mathematical Model

To the Editor.—Dexter and Hindman assessed the effect of hypothermia on the relationship between cerebral venous oxygen saturation and cerebral oxygen consumption. Their mathematical model suggests that, during deep hypothermic (17°C) cardiopulmonary bypass (CPB), hemoglobin oxygen affinity might increase (i.e., P50 might decrease) to the extent that oxygen transfer from hemoglobin to brain tissue may be significantly limited. Because of such limitation in tissue oxygen delivery, their model predicts that, at 17°C, brain oxygenation (specifically brain oxygen consumption) will start to decrease when cerebral venous saturation decreases to less than 95, and particularly to less than 90%. In addition, such a model cautions against techniques that use cerebral oxymetoglobin saturation alone as an index of cerebral tissue oxygenation during deep hypothermia. Our findings using near-infrared spectroscopy to measure cerebral intravascular oxygenation (oxyhemoglobin concentration) and mitochondrial oxygenation (oxidized cytochrome aa3 concentration) were consistent with the predictions of the mathematical model of Dexter and Hindman.

We used near-infrared spectroscopy to characterize the relationship between brain intravascular oxygen saturation, which largely reflects cerebral venous hemoglobin oxygen saturation, and cerebral mitochondrial oxygenation, as reflected by the oxidation state of cytochrome aa3. We noted that, at the onset of hypothermic CPB, an increase in cerebral oxyhemoglobin concentration occurred. This increase in intravascular oxygenation, however, was accompanied by a progressive decline in the oxidation state of cytochrome aa3. This suggested a paradoxical dissociation between intravascular and tissue oxygenation occurring during deep hypothermic CPB. Thus, our results agree with the Dexter and Hindman’s predictions that (1) oxygen transfer from hemoglobin to the cerebral parenchyma may become progressively limited during deep hypothermia; and (2) high levels of cerebral venous hemoglobin saturation may be deceptive and may not reflect normal oxygenation at the tissue level.

Adre J. du Plessis, M.D.
Department of Neurology
Jane Newburger, M.D.
Department of Cardiology
Paul Hickey, M.D.
Department of Anesthesia
Richard A. Jonas, M.D.
Department of Cardiac Surgery
Joseph J. Volpe, M.D.
Department of Neurology
Children’s Hospital
300 Longwood Avenue
Boston, Massachusetts 02115

References


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