Correspondence

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Hypotension from a Nodal Rhythm

To the Editor:—I enjoyed Dr. Lappas’ article but found myself confused by figures 7 and 8 and the corresponding text. In figure 7, the PAP shows an increase similar to the increase in RAP during the nodal rhythm. But the text describes it differently. Heart rate did not increase appreciably with conversion from nodal to sinus rhythm (fig. 7) or from nodal rhythm to atrial pacing (fig. 8). However, the low arterial pressure during nodal rhythm in both fig. 7 and fig. 8 is impressive. Obviously, heart rate alone is not the factor. A discoordination of atrial and ventricular contraction could explain it. We often observe nodal rhythm following a second dose of succinylcholine or when un-premedicated patients are anesthetized with halothane. This arrhythmia is resolved by the administration of atropine or by letting the patient breathe spontaneously. We have not observed such marked hemodynamic changes as noted by Dr. Lappas. Could this be because the patients studied by Dr. Lappas had poor intrinsic myocardial function?

Anyway, I wouldn’t have thought of countershocking someone with nodal rhythm before I read this article.

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Reference

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EEG Changes during Anesthesia

To the Editor:—I was most interested to read Dr. Tinker’s article in which he observed an abrupt appearance of anterior amplitude dominance during induction of anesthesia and that this EEG change is coincident with loss of eyelid reflex and loss of ability to respond to command. Of particular interest is the difference in EEG activity between anterior and posterior areas. I have monitored the EEG continuously during anesthesia in many patients and have repeatedly observed that amplitude-dominant rhythmic activity originates from the frontal area. Dr. Tinker surmises that amplitude-dominant rhythms in awake man originate from posterior areas. It is known that normal EEG rhythms in awake man are divided into four types: alpha, beta, theta, and irregular. Dr. Tinker’s hypothesis may hold true for patients with alpha rhythms. However, it may not hold true for patients with beta, theta, or irregular rhythms. Furthermore, in about 20 per cent of adult patients, alpha rhythms are not observed in the posterior area (rare alpha type). Instead, low-voltage, fast activity is dominant in the posterior area of these patients. Also, I question the importance of the difference of amplitude between 20 μV obtained anteriorly and 18.5 μV obtained posteriorly. During continuous EEG recording of EEG activity in more than 500 patients, who were given various preanesthetic medications, I found low-voltage, fast EEG activity in all leads. Posterior amplitude dominance obtained from the patient with an alpha rhythm when recorded in the EEG laboratory (silent, dark, no premedication) was not always observed when the same EEG studies were made in the operating room. Dr. Tinker concludes correctly that the clinical usefulness of the EEG has been limited by unwarranted emphasis on differences among anesthetics, enthusiasm for complex frequency analyses, and lack of recognition of the importance of level of stimulus at clinically useful concentrations. However, it has been confirmed that as anesthesia deepens, EEG amplitude increases and frequency decreases compared with the awake state. This change does not diminish the value of the EEG as a monitor of cerebral function during clinical anesthesia and operation.
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References

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In reply: — Dr. Yanagida correctly states that amplitude-dominant EEG rhythms are not always seen posteriorly in awake man. We agree that posterior alpha amplitude dominance occurs in awake patients about 80 per cent of the time. Whether or not there is any awake posterior amplitude dominance in a given patient seems irrelevant to our observation that frontal amplitude dominance (never seen in awake normal man) develops during induction of anesthesia, and our use of this rather abrupt development as an endpoint for comparison with MAC. Dr. Yanagida has not always observed posterior amplitude dominance after premedication, but before “anesthesia.” We wonder just how heavily his patients were premedicated, and whether loss of awareness of surroundings had already occurred. Dr. Yanagida questions the closeness of the amplitude measurements shown in our figure 4. We agreed, and concluded in our paper that the shift had not yet occurred in figure 4, but was present by figure 6, where anterior dominance is obvious. We therefore considered the EEG change to have occurred midway between the two concentrations, at which time the anesthetic concentration was still well below MAC.

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