All That Quakes Does Not Necessarily Shiver

To the Editor—Sessler et al.1 utilize surface-recorded electromyography (EMG) in an effort to unravel the etiology of “postanesthetic shivering.” Their article raises interesting questions about shivering; however, the applicability of their electromyographic recording techniques and the resulting data analysis are not convincing. Their hypothesis for the etiology of postanesthetic shivering depends on the analysis of spontaneous EMG activity. The indispensable elements for comprehending this analysis are an adequate description of electrophysiological recording techniques, such as frequency band-width, and the assumptions used in Fourier analysis. This vital information is omitted in their article as published. With any technique that manipulates and reproceses raw data, a clear understanding of a number of implicit (but not often obvious) specifications and limitations of the data-acquisition parameters and associated Fourier analysis must precede the interpretation of the final Fourier transform.

A raw EMG of “postanesthetic tonic EMB activity” is illustrated by the authors in trace 1D of figure 1.2 This recording resembles an EMG “interference pattern”; the typical picture resulting from many motor units firing at rates of 10–50 Hz, all superimposed on one another. In contrast to the raw EMG, the associated power spectrum (depicted in fig. 3) only has a sharp, narrow amplitude peak in the frequency band between 4 and 8 Hz. How can this raw EMG transform into a “tight” power-spectrum? Typically, at the onset of motion, tonically discharging motor units in such postural muscles discharge at frequencies in the range of 5–10 Hz.3 Moreover, the other frequencies evident in the raw EMG are not represented in the power spectrum, thereby emphasizing the significance of the 4–8 Hz spike. Such discrepancies cannot be independently reconciled without knowledge of the recording techniques and Fourier analysis assumptions. Furthermore, the inevitable possibility arises that the authors’ experimental findings result from a methodologic bias in collecting or processing the EMG data.

Another EMG pattern identified by Sessler et al. is a rhythmic bursting discharge at a frequency of 5–7 Hz for which they coin the term “spontaneous EMG clonus.”4 By definition, all clonus is induced by some stimulus; therefore, “spontaneous EMG clonus” is a misnomer. Instead, another descriptor, such as “clonus-like EMG,” would be more appropriate. The authors’ conclusions regarding this “clonus-like EMG” activity also require comment. They observe that the clonus seen in spinal-cord-injured patients and the rhythmic “clonus-like EMG” recorded during emergence from anesthesia each have discharge frequencies in the 5–7 Hz range. Thus, they presume that the two phenomena are identical. This conclusion is then used to postulate that low-dose (0.10–0.19%) isoflurane produces a “functional spinal cord transaction,” the “clonus-like EMG” results from the disinhibition of an “awake” spinal cord from a brain that is “still asleep.”5 In fact, similar spontaneous muscle activity with discharge frequencies in the 5–7 Hz range can occur in several pathological states. For example, the clonic activity observed in major motor seizures, epilepsy partialis continua, various types of tremors, and several focal forms of myoclonus all have rhythmic discharge frequencies in the 5–7 Hz range. In addition, the neuronal generator source for this abnormal activity can manifest at a number of different levels within the neuraxis.6 Therefore, a surface-recorded EMG study of muscle activity cannot be used to positively identify the generator focus for the “clonus-like EMG” as described by these authors.

Finally, Sessler et al. dismiss normal thermogenes as an etiology for postanesthetic shivering. They claim that the EMG patterns collected in individuals subjected to cold-stress do not resemble the EMG patterns recorded during emergence from anesthesia. However, these data are incomplete. In figure 2, the authors contrast multi-channel EMG patterns in a cold-stressed subject and a patient emerging from general anesthesia.1 The data on normal cold-stress shivering is derived from the authors’ own single study and is only published in abstract form; thus, an independent detailed analysis of the recordings is not yet possible. In addition, a clear and exact description of the clinically observed muscle activity occurring at the time of the recording is necessary. Although multiple-channel recordings are a standard method for assessing abnormal spontaneous movements, it is not clearly evident that the EMG pattern in figure 2B comes from a patient who is shivering at the time the recording was obtained. It is as likely that it illustrates the tonic stiffening of muscles as seen during a generalized seizure. This apparent paradox can only be explained by detailed clinical observations made at the moment the recordings were taken. In all, insufficient data exists to categorically conclude that cold-stress shivering fundamentally differs from that observed in postanesthetic shivering.

In summary, the paper by Sessler et al. addresses the important clinical issue of postanesthetic shivering. Flaws in data presentation and in the logical interpretation of results, however, make it difficult to accept the validity of their conclusions. For this study to have any relevance, the authors should clearly indicate how these EMG signals were recorded and analyzed. To adequately buttress their conclusions, additional data on cold-stressed shivering must become available. Finally, evidence needs to be assembled that clearly differentiates generator sources in the neuraxis that can potentially drive the phenomenon of postanesthetic shivering.

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(Accepted for publication November 8, 1988.)