

## CORRESPONDENCE

**Table 1. Increase in Arm and Leg Heat Content during Prewarming**

(h)	Δ Content (kcal)	Δ Content (kcal/kg)	Fraction of Maximum (%)
0.5	77 ± 18	0.9 ± 0.1	44 ± 6
1.0	133 ± 30	1.6 ± 0.2	77 ± 8
2.0	176 ± 48	2.0 ± 0.3	100

Five volunteers were warmed for 2 h using a Bair Hugger forced-air heater (Augustine Medical, Eden Prairie, MN) set on "high." Total arm and leg (peripheral) tissue heat content increased dramatically with forced-air warming. Most warming occurred within 1 h.

crease in arm and leg heat content explains how prewarming alone can maintain intraoperative normothermia for many hours.<sup>6</sup>

The specific heat of humans is essentially constant.<sup>10,11</sup> A nifedipine-induced increase in peripheral thermal compartment heat content, therefore, must be associated with increased peripheral tissue temperature and a reduced core-to-peripheral tissue temperature gradient. Therefore, we were surprised by the suggestion of Guillaume *et al.* that nifedipine increases peripheral heat storage *instead* of changing the core-to-peripheral tissue temperature gradient. Their error appears to be in assuming that mean skin temperature reliably indicates peripheral tissue temperature. Skin temperature is a complex function of core and ambient temperature, adjacent tissue temperature, and thermoregulatory status. That skin temperatures were similar in untreated patients and those given nifedipine indicates simply that skin temperature generally does not adequately quantify peripheral tissue temperature.

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Anesthesiology

81:526-527, 1994

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(Accepted for publication May 4, 1994.)

**Electroencephalogram Suppression during Anesthesia**

*To the Editor:*—In a recent editorial, Drummond<sup>1</sup> stated that, with a concentration of isoflurane sufficient to render the *electroencephalogram (EEG) isoelectric*, it is reasonable to anticipate that the rate of cerebral oxygen use is strongly reduced. From the following, discussion it is obvious that he means *EEG suppression*, not *isoelectric EEG*.

Isoelectric EEG refers to a situation in which the brain, or at least the cerebral cortex, does not produce electrical activity, *i.e.*, electrocerebral inactivity or electrocerebral silence.<sup>2</sup>

Suppression is not synonymous with isoelectricity. During isoflurane-induced suppression, low-amplitude EEG activity is well described in the literature. Zaret has reported  $\alpha$  coma pattern during suppression.<sup>3</sup> Spindles measuring 13 Hz are seen on the vertex during propofol-induced suppression.<sup>4</sup> Focal or generalized epileptic discharges sometimes are seen during barbiturate anesthesia for the treatment of status epilepticus. In the higher frequencies, the N<sub>20</sub> wave of somatosensory evoked potentials to median nerve stimulation, for instance, can be recorded even during EEG suppression. Hence,

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the cerebral cortex can produce many kinds of electrical activity during suppression.

DC shifts are seen during burst suppression pattern: At suppression onset, the cortical potential changes to a positive level and returns to a negative level at burst onset.<sup>4</sup> Cortical positivity correlates with inhibition, and a negative DC level shift, similar to that seen during bursts, also is seen during generalized epileptic discharges. These DC shifts may give clues to the physiologic role of suppression.

If the EEG is isoelectric, no stimulus produces EEG activity. Conversely, during continuous suppression, bursts can be evoked by minor tactile, sound, or visual stimuli.<sup>5</sup> In certain pathologic conditions, such as the Ohtahara syndrome, a patient can be behaviorally awake when EEG shows this pattern. The pattern then is comparable with interictal epileptic discharges, which may have a minor impact on the patient's behavior.

In summary, burst suppression is readily produced by every healthy brain during general anesthesia with many modern anesthetics. Studying this phenomenon should give us insight in the function of the brain, including the mode of action of anesthetic agents, *i.e.*, what anesthesia is all about. However, indeed, during suppression, EEG is not isoelectric.

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(Accepted for publication May 4, 1994.)

Anesthesiology  
81:527-528, 1994  
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J. B. Lippincott Company, Philadelphia

*In Reply:*—I am one of those people who enjoys cavilling over matters of precise usage of language. So, I suspect that the various coauthors whom I have tormented in past will enjoy seeing me hoisted so publicly on this particular petard.

My response to Jääntti is a qualified "Touché." Although authoritative sources seem more interested in discouraging the use of the term isoelectric than in defining it, it appears that the term should be restricted to states of resting cerebral silence combined with nonresponsiveness to sensory stimuli.<sup>1,2</sup> However, I must add a few comments.

When I received Jääntti's letter, I turned to my bookshelf and quickly found the term, "isoelectric" (used as I used it in my editorial) in *ANESTHESIOLOGY*, *Anesthesia and Analgesia*, *The Journal of Neurosurgery*, *Neurosurgery*, *Stroke*, *The Journal of Neurosurgical Anesthesiology*, and *The Canadian Journal of Anesthesia*. Michelfelder also makes similar use of the term in his monograph "Anesthesia and the Brain."<sup>3</sup> It appears that the term has been widely acceptable to authors and editors alike. That doesn't make it correct, but it certainly means that it is widely understood. To confirm the latter, I consulted neurologists, neurosurgeons, and neuroanesthesiologists in my immediate environment. None of the individuals consulted found the use of the term "isoelectric" to describe a drug-induced state of electroencephalogram (EEG) suppression to be inappropriate or misleading.

What should we do? Well, I have been sensitized by Jääntti's letter (and the need to prepare a response), and I, therefore, probably will reduce my use of the term "isoelectricity." This will be difficult,

because I cannot simply replace the term "isoelectricity" with the term "suppression." Isoelectricity (even if we are somewhat casual with the electrophysiologic definition of isoelectricity) is an absolute term. Suppression is a relative one. If I administer 3-4 mg/kg thiopental to a normal subject, there is no question that it would produce EEG "suppression." However, it would be uncommon for that dose to achieve what I heretofore would have described as "isoelectricity." This means that it will be necessary to use qualifiers to distinguish between relative suppression and maximal suppression. Do the terms "electrocerebral silence" and/or "electrocerebral inactivity" represent alternatives? Not for the purists. The guideline cited by Jääntti indicates that those terms also imply nonresponsiveness to somatosensory, visual, or auditory stimuli, which, as he pointed out, is *not* a concomitant of states of anesthetic-induced maximal suppression (?). Some may choose to use terms such as "maximally suppressed." I suspect, however, that many will still prefer the simplicity and familiarity of "isoelectric."

Next, I acknowledge that, even before Jääntti's letter, I had been uncomfortable with my use of the term "isoelectric." My discomfort stemmed from the knowledge that the term, at least as used in connection with brain death, requires that there be no activity of amplitude greater than 2  $\mu$ V on the surface EEG. It has been my repeated though nonsystematic observation that, in humans, cats, and rats (I have not had the opportunity to make observations in other species), the maximal degree of suppression that can be achieved with anesthetic agents, particularly isoflurane, results in a surface EEG that is frequently referred to as "flat" or "isoelectric" but in reality has