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.[4] 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.[5] 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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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 Jäntti 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.[1,2] However, I must add a few comments.

When I received Jäntti'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.* Michenfelder also makes similar use of the term in his monograph "Anesthesia and the Brain."[3] 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 Jäntti'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 Jäntti indicates that those terms also imply nonresponsiveness to somatosensory, visual, or auditory stimuli, which, as he pointed out, is *not* a concommitant 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 Jäntti'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 μV 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

residual activity that is frequently in excess of 2 μV, *i.e.*, it really is not "isoelectric" by anybody's definition. Nonetheless, we all have a common understanding of what we are describing when we use the term.

Finally, Jäntti describes circumstances in which activity can be elicited during anesthetic-induced maximal suppression (?) of the EEG. He provides references to support several of his suggestions though not for his assertion that somatosensory evoked responses to median nerve stimulation can be elicited during EEG suppression. I am pleased to provide references to support that assertion as well, in part to assure Jäntti that this phenomenon was well known to me.[4,5]

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A Novel Use for the Pulmonary Artery Catheter

To the Editor:—I would like to report a novel use for the pulmonary artery catheter.

The patient was a 58-yr-old man involved in a high-speed motor vehicle accident. After initial evaluation and stabilization, including tracheal intubation, he presented to the operating room for open reduction and internal fixation of bilateral femur fractures.

During attempts at inserting a right internal jugular venous catheter, I found that the vein could be cannulated readily with a 20-G catheter, but the "J" tip guidewire could not be advanced without meeting moderate resistance. The left internal jugular vein then was cannulated with a 20-G catheter, and the "J" tip guide wire was advanced without resistance. An 8.5-French catheter (Arrow-Flex Sheath, Arrow International) subsequently was inserted over the guidewire. After full insertion, however, air was aspirated from the three-way stopcock. The catheter then was slowly withdrawn while negative pressure aspiration was maintained. When the catheter was approximately 50% withdrawn, blood was aspirated easily from the three-way stopcock.

At this point, a Swan-Ganz Thermodilution Paceport catheter (Baxter Healthcare) was inserted through the 8.5-French catheter. With the balloon inflated, the pulmonary artery catheter was advanced sequentially into the right atrium and through the right ventricle into the pulmonary artery. The 8.5-French catheter then was inserted "over" the pulmonary artery catheter. After full insertion, blood was aspirated easily from the three-way stopcock.

It is not clear why air was aspirated initially from the three-way stopcock after full insertion of the central venous catheter. I was concerned that the guidewire may have exited the venous system through a small disruption at the junction of the left internal jugular vein and left subclavian vein and that the central venous catheter tip was located within the mediastinum. This hypothesis is not unreasonable considering that the patient had been involved in a high-speed motor vehicle accident and the difficulty encountered in attempting to advance the guidewire through the right internal jugular vein. This may have been the case even without a traumatic disruption, because inadvertent placement of a central venous catheter into the mediastinum *via* the left internal jugular vein approach has been reported.[1] In general, venous perforation caused by central venous catheter placement is more likely when the left internal jugular vein approach is used rather than the right internal jugular vein approach.[1] I thought that using the pulmonary artery catheter with the balloon inflated, which is blood-flow directed, instead of using the guidewire, which is not blood-flow directed, would initially allow passage of the pulmonary artery catheter into the right side of the heart and then serve as a "guidewire" for passage of the central venous catheter past the junction of the left internal jugular vein and left subclavian vein.