

Cortical Electroencephalogram from Subcortical Electrodes rather than Electrosubcorticogram

To the Editor:— In the August 2007 issue of ANESTHESIOLOGY, Velly *et al.*¹ reported the difference in electroencephalograms from cortical and subcortical electrodes during anesthesia and concluded that they reflect different actions of anesthetics on cortical and subcortical structures. However, the authors failed to fully explore what they really were recording from scalp and depth electrodes. Our group has done several similar recordings,² and based on these, we want to make some corrections in the interpretation of Velly *et al.*

The electroencephalogram consists of several patterns and components that may occur simultaneously and with varying topology. During anesthesia, we can record slow wave oscillations, which resemble the slow waves of sleep and are widespread. Some other patterns are seen in limited areas, such as the spindles.^{2,3} In figure 1, we show examples of both during burst suppression in propofol anesthesia. The patient data are from our published material.² The uppermost trace is bipolar scalp derivation P4-F4. The next traces show depth 4–depth 1, where depth 1 is the contact at the tip of the electrode and depth 4 is the one closest to the vertex, in a four-contact electrode in the subthalamic nucleus, similar to the depth electrode of Velly *et al.*¹ The next two traces are the right frontal electrode F4 and the right parietal electrode P4 referred to depth 1. The lowest trace shows that the spindle is recorded between two surface electrodes. P4 and A1, although simultaneously it is not visible in P4-F4. In all traces positive at the first, active electrode is down.

Notice that the suppression, slow wave burst with approximately 10 Hz activity on it, and the spindle occur synchronously in derivations between scalp electrodes and between two contacts in the depth electrode. Their relative amplitude, however, changes depending on the relative amplitude of these waves at electrodes F4 and P4. The uppermost trace is the difference of the third and fourth traces. The slow wave and spindle are on the average of the same amplitude in scalp–scalp derivation, uppermost trace, and between the two contacts of the depth electrode. Note that from a recording between two electrodes in a volume conductor we cannot conclude where is the source of the electrical activity. Conclusions about the generator must be based on multielectrode recordings, as in the case of location of epileptic foci, and similarity with patterns with known generator such as the cortical slow waves.

The voltage fluctuation generated by the cortex spreads by volume conduction through cerebrospinal fluid, bone, and skin-to-scalp electrodes. It also spreads through brain tissue, which is a volume conductor. When the current passes the contacts of the depth electrode, a voltage is recorded that equals the product of the current and the impedance of the brain tissue between the two contacts of the electrode. This is also how voltage, scalp electroencephalogram, is recorded between two scalp electrodes. The signal recorded between the two contacts of the depth electrode therefore is the electroencephalogram recorded with a depth electrode in the subthalamic nucleus. It probably has little contribution from the nearby structures, because the differences between transcortical and scalp traces and the depth electrode trace can be explained by the sensitivity distribution of the electrodes.

In conclusion, the signals recorded by Velly *et al.*,¹ both the signal from scalp electrodes and that from depth electrodes, are probably mainly electroencephalogram generated by the cerebral cortex, which is also evident from the illustrations of their article. The reason why they get different spectra and different correlation dimensions from the two signals is the different topography of the different cortical electroencephalographic patterns and therefore different contributions to the depth electrode–recorded electroencephalogram and scalp electrode–recorded electroencephalogram. The derivation they use for scalp electroencephalogram minimizes the slow waves, which are the

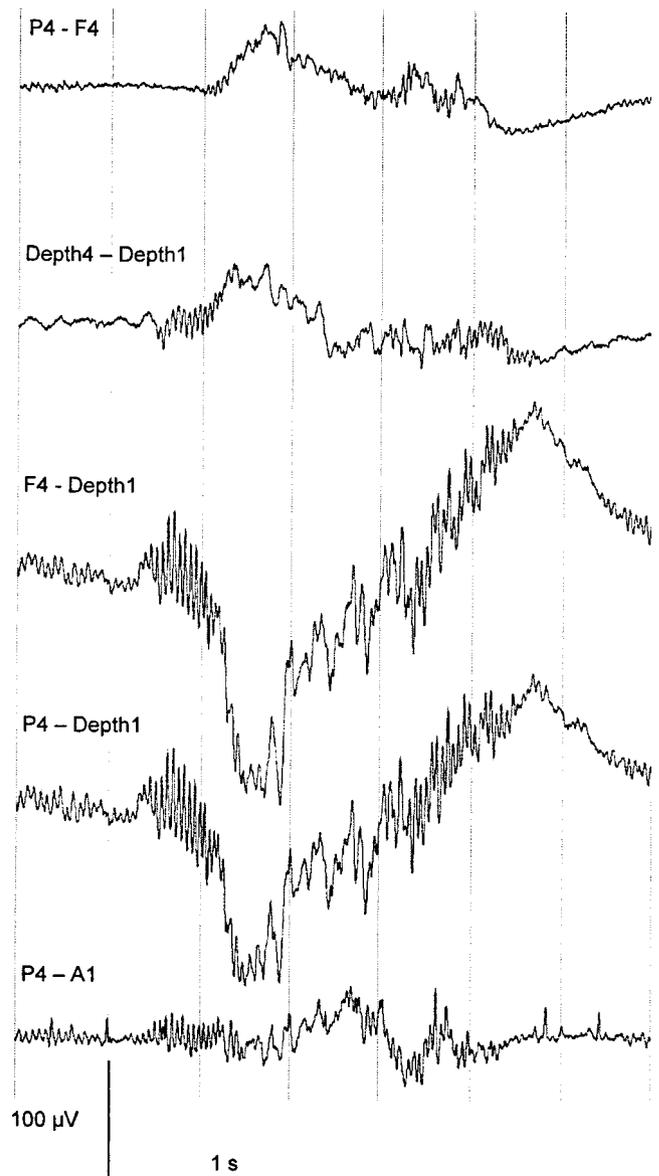


Fig. 1. Electroencephalogram during burst suppression in propofol anesthesia. The *uppermost trace* is the electroencephalogram from two scalp electrodes, the right parietal P4 and the right frontal F4. The *second trace* is the electroencephalogram recorded between two contacts in the depth electrode in the subthalamic nucleus. The *two lower traces* are the electroencephalogram recorded between scalp electrodes P4 and F4, and the electrode contact deepest in the subthalamic nucleus. Notice that practically all waveforms that are recorded between the two contacts of the depth electrode are also recordable from surface electrodes with varying amplitude and polarity. The depth electrode recording and scalp recording therefore are generated by the same structures, most likely the cortex, not two independent generators, one at the thalamus and one at the cortex. The patient data are from Sonkajarvi *et al.*²

most important indicators of the effect of anesthetics.⁴ Rather than the electrosubcorticogram, the signal from the subthalamic electrode is the cortical electroencephalogram, recorded from or near the subthalamic nucleus. It is mainly the same cortically generated signal, the electroencephalogram, but recorded from the other side of the cortex.

The article by Velly *et al.* therefore may not present the differences in electrical activity of deep structures and the cerebral cortex, as the authors claim. Both signals are mainly electrical activity of the cerebral cortex. This shows the importance of understanding the physiology and electrical fields of the electroencephalogram during anesthesia.

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In Reply:—We thank Dr. Jääntti *et al.* for their interest in our study on the differential dynamic of action on cortical and subcortical structures of anesthetic agents during induction of anesthesia.¹ The location of the signal recorded through deep-brain electrodes is clearly a concern for all stereoelectroencephalography measurements. In epilepsy surgery, neurophysiologists and neurosurgeons rely on deep-brain recordings to precisely delimit the brain region to remove surgically. If such a gross contamination due to volume conduction were to occur, the clinical results would certainly be poor. We agree that one problematic issue in combined scalp cortical (electroencephalographic) and subcortical (electrosubcorticographic) electrogenesis recording is that of volume conduction from cortical to subcortical regions or the opposite. But first, we observed different data from each site showing that we observed synaptic activity from different regions of the brain (as shown in fig. 2 in the article). Second, we used a bipolar recording for the scalp and the deep-brain electrode. Wennberg *et al.*² demonstrated that scalp potential recording in the subthalamic nucleus with monopolar montage totally disappears with a bipolar montage.

We do not believe that the illustration provided by Dr. Jääntti *et al.* supports their claim that we recorded only cortical activity both on the scalp electrodes and from the deep-brain electrode. First, the authors suggested that plot 4 of the deep-brain electrode was “close to the vertex,” which is not correct. As shown in figure 1 in the article, this plot is far from the cortex and close to the thalamus. Second, it shows a burst constituted of one slow wave superimposed with rapid activities of various frequencies. These rapid activities are more complex than classic spindles. In addition, the illustration shows, at the beginning of the burst, high-frequency activity in the cortex but not on the depth electrode. The interpretation made to explain this discrepancy is purely speculative. In fact, it demonstrates that cortical and subcortical electrogenesis were different.

We were also surprised to read, in their letter and in the published material from which the patient data came,³ that spindle generation occurred in the cortex, contrary to a large body of data showing the crucial role of the thalamus.⁴ This is not new, because Morrison and Bassett⁵ showed in 1945 that spindles survived in the thalamus after bilateral decortication. We also published in another article that spindles appeared in the thalamus during physiologic sleep before they appeared in the temporal cortex in epileptic patients undergoing stereoelectroencephalography.⁶ In this study, the same surgically implanted electrode was used to record, using bipolar montage, both cortical and thalamic activity. This clearly shows that we recorded deep-brain electrophysiology and not only the electroencephalogram through the implanted electrode.

We found the topographic interpretation of Dr. Jääntti *et al.* confusing. In their article, they state that the phase reversal of the initial component of the burst in figure 2 of this study (Cz-D1/D1-C7, where

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D1 is the depth electrode) is an argument for a thalamic origin. That is correct according to the classic rule of electroencephalographic bipolar interpretation,⁷ but they do not use the same rule for the latter component of the electroencephalographic data (δ activity and “spindle” in fig. 1), and they conclude that there is a cortical origin of this component. We believe this interpretation is incorrect and question their comments regarding topography in our data.

Dr. Jääntti *et al.* also suggest that cortical and subcortical activities are similar at a deep anesthetic state in our study (fig. 2 of the article), demonstrating that we recorded the same signal in both settings. As we write in the article, the activities are similar during deep anesthesia (T4). What were important in our study were the differences in electrophysiology between the electroencephalogram and the electrosubcorticogram we observed during induction of anesthesia, which were obvious from our figures and data, demonstrating that we recorded different activities. We discarded periods with a burst suppression pattern, which in our opinion cannot be interpreted, because electrophysiology tends to be uniform throughout the brain at a very deep anesthetic state (ultimately identical when the electroencephalogram is flat).

Finally, the scalp derivation we used did not minimize slow wave activity. It did not maximize the amplitude of slow waves, but it did not change the dynamics of slow wave appearance. This is clear in our article (figs. 2 and 4) showing the early appearance of δ waves.

We agree with Dr. Jääntti *et al.* that a thorough understanding of basic electrophysiology is mandatory to interpret the electroencephalogram during anesthesia.⁸ We disagree, however, with their interpretation and believe that routine electrophysiologic recordings with depth electrodes, work in our laboratory,⁹ and evidence from the literature support the validity of our data.

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