Recording Cortical EEG Subcortically — Improved EEG Monitoring from Depth-Stimulation Electrodes

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***Abstract*—The electroencephalogram (EEG) generated by cerebral cortex can be recorded far away from the cortex, analogous to the electrocardiogram (ECG) that can be recorded far from the heart. ECG is often seen as an artifact in EEG recordings. In this paper we demonstrate that the burst suppression pattern of EEG, which is generated by the cerebral cortex, can be recorded at a distance from the cortex with a pair of electrodes in the subthalamic nucleus and also with an electrode pair on the masseter muscle below the zygomatic arch. We then present a fundamental theoretical model which explains the currents inside and outside the cranium, which produce the EEG at these locations.**

I. Introduction

Extensive literature exists regarding the recording locations of the elecrical activity of the brain and its evoked elecrical activity, i.e. evoked potential (EP). These electrical sources are generated in cortical or subcortical structures using the electroencephalogram (EEG) or the magnetoencephalogram recordings. It is obvious that all EEG currents generated by the cortex flow both above and below the cortex to form current loops [1]. These return currents are usually omitted in the calculations. Even when they are included, the assumption is that the subcortical structures are a homogeneous mass, omitting the fact that they in fact are inhomogeneous and anisotropic.

The generators of EEG and EPs are often assumed to be dipole surfaces consisting of gray matter, which can be modeled with a dipole. This dipole may change in shape and orientation continuously or be stationary. However, much of the cortical EEG activity is synchronous in the whole cortex which covers the hemispheres with the exception of the part where the brainstem originates. An example of such activity is the cortical slow oscillation of sleep, as well as much of the slow oscillations during anesthesia. It follows that this kind of generator cannot be modeled with a single dipole. Furthermore, surface recordings measure the amplitude difference at two scalp surface locations corresponding to underlying cortical locations according to the lead field of the measurement [2]. Obviously, also the large dipole surfaces induce currents in the subcortical brain, but models of these currents are few and relatively rough [3].

In the diagnosis of epilepsies, particularly in the search for structures for epilepsy surgery, subdural and intracortical EEG has been recorded extensively during the last 50 years. Recording with multi-contact intracerebral electrodes enables localizing the onset zone of epileptic seizures, but usually only roughly, for instance either the temporal lobes or the frontal lobes. The exact location of seizure activity in three dimensional space remains uncertain and depends on the knowledge of structures and localization of the electrodes given by imaging studies. The localization is reasonable, when the electrode is in the gray matter, which either contributes to or is involved in the propagation of the focal seizure; however, when the recording electrode is far from the focus, its value in localization is limited.

In the same way, event-related potential recordings from intracerebral structures have been used to localize function such as the neural network of pain perception and the role of insula in pain. Attempts to localize early components of somatosensory evoked potential with subthalamic electrodes have been less successful due to the lack of appropriate models.

During the past few decades, stimulation of deep midline structures has been increasingly used for treatment of disorders like Parkinson's disease and seizure disorders. These implanted electrodes have created a possibility also to record event related potentials and spontaneous activity, such as sleep and epileptic spikes [4] or anesthesia [5], [6]. This has also drawn attention to the fact that not only subcortical but also cortical epileptic activity is recorded by deep midline electrodes [7].

Although advanced models exist for the effects of stimulation currents in the adjacent structures, these models do not explain to what extent these currents spread to the cortex and scalp. On the other hand the spread of thalamic currents to the scalp has been modeled by [8], which also takes into account anisotropy of the structures. Also, evoked potentials

TABLE I
TISSUES AND CONDUCTIVITY (RESISTIVITY) VALUES INCLUDED IN OUR SPHERICAL HEAD MODEL [10].

Tissue	Conductivity [S/m]	Resistivity [Ω cm]
Skull/Bones	0.029	3450
White matter	0.14	700
Scalp	0.43	230
CSF	1.82	55

have been recorded from depth electrodes, but the results have been explained with simple models traditionally used in scalp recordings, such as phase reversal [7].

Taking all this into account, we have concluded that it is impossible to say what part of the signal recorded by depth electrodes in subthalamic nuclei comes from nearby structures and what comes from the cerebral cortex. The same is true about sleep activity and epileptic spikes [4].

As stimulation electrodes are increasingly applied, the possibilities for continuous long term recordings become more widely available. In general, the stimulation current is applied in the form of relatively short pulses. If stimulation current is non-continuous, the time intervals between stimulation should allow for EEG recordings either from the (same) stimulation contacts or additional electrodes on the shaft of the stimulation electrode. During the off-phase, i.e. the non-stimulating portion of the cycle, the dual usage of the stimulating electrodes would depend on the switching capability of the amplifiers.

Recording from such electrodes has many benefits compared with scalp or subdermal recordings, particularly less electrode movement artifacts and electromyogram (EMG), sometimes also electroculogram (EOG), which often render scalp or subdermal EEG recordings impossible to interpret. However, accurate studies on the spread of EMG, and EOG to subcortical electrodes, are also missing.

In this paper we show a simple model, which explains the spread of cortical currents to deep thalamic electrodes and show how these electrodes pick up cortical EEG during sevoflurane and desflurane anesthesia. We then discuss the possibilities of long term recordings from these electrodes and the possible relevance of these recordings in the treatment of the patients with electrodes implanted for treatment of seizures or Parkinson's disease.

This paper demonstrates that EEG can be recorded during deep anesthesia, burst suppression, between two electrode contacts of a stimulation electrode [6], [9]. We then show that burst suppression EEG can also be recorded with two electrodes on the skin of face, on the masseter muscle below the zygomatic arch, which are also far from the generator, the cerebral cortex. Finally, we illustrate with a spherical model how the electric field of the cerebral cortex flows inside the brain past the depth electrodes and under the skin of the face past the electrode on the masseter, so that electrode pairs at these sites record cortical activity.

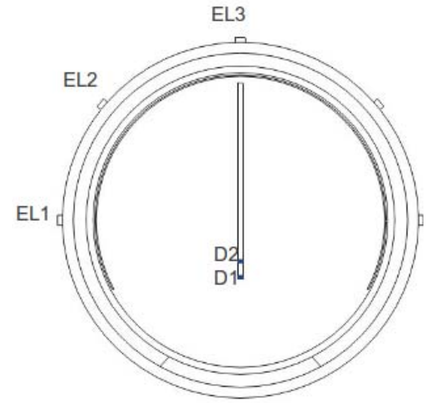


Fig. 1. The geometry of the spherical head model illustrates the subthalamic electrodes D_1 and D_2. Surface electrodes E_1, E_2, and E_3 reflect standard electrode locations for the comparison of lead placement.

II. METHODS

A. Physiological Measurements

We recorded the EEG of patients undergoing anesthesia for surgery for implanting stimulation electrodes in the subthalamic nucleus. The indication for the stimulator implantation was Parkinson's disease. In the first set of recordings, the patients were anesthetized with the volatile anesthetic desflurane. We recorded electrical activity from the scalp (i.e. EEG) and subthalamic electrodes simultaneously and plotted scalp–depth, scalp–scalp EEG as well as EEG recorded between two contacts in the stimulation electrode i.e. depth–depth. The digital EEG recording was done with a sampling freuency of 512 Hz, and a bandpass of 0.016 Hz – 200 Hz. The study was approved by the ethics committee of the Oulu University Hospital.

The second set of recordings are from two electrodes attached to the masseter muscle, vertically 3 cm apart. The patient was anesthetized to EEG burst suppression level with propofol. We compared the masseter measurements to scalp recordings, a submental electrode pair, and the patient's electrocardiogram (ECG). The study was approved by the ethics committee of the Tampere University Hospital.

B. Sensitivity distributions

This study simulated the sensitivity distribution of the synchronously active cortex. The lead field maps the direction and sensitivity of each measurement lead [2], [11]. It is created by feeding a reciprocal current I_R to the measurement lead. There are two ways to explain and depict the lead field – either as a current field or a potential field. The lead voltage relates the measured signal to the current sources in the volume conductor such that

$$V_{LE} = \int_v \frac{1}{\sigma} \mathbf{J}_{LE} \cdot \mathbf{J}^{\,i} dv, \tag{1}$$

where V_{LE} is the measured EEG voltage in the volume conductor v. The reciprocal current field $\mathbf{J}_{LE}$ is the lead field,

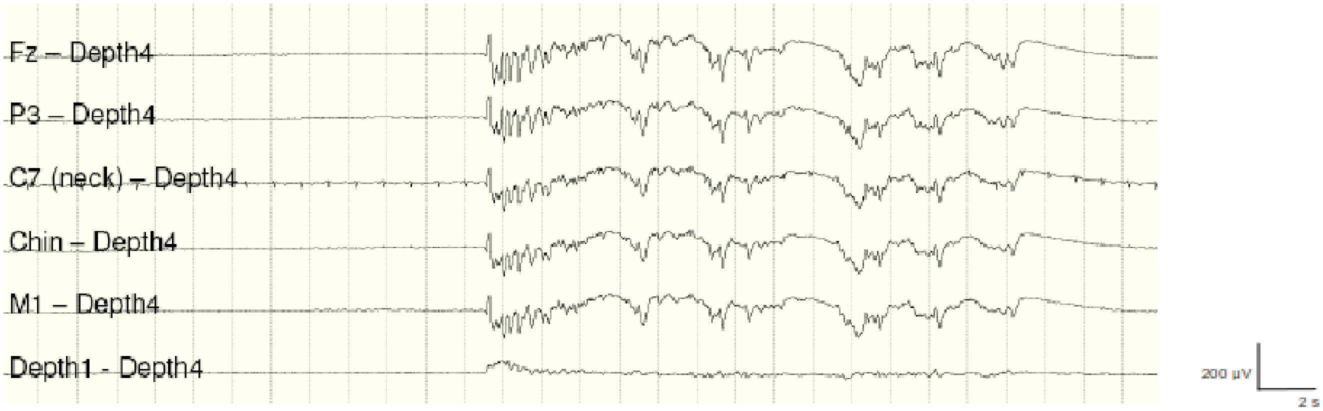


Fig. 2. Burst suppression consists essentially of cortical slow oscillations of sleep, surface positive (down!) slow waves, which appear synchronously on the whole cortex, and phase-locked faster activity. These can also be recorded with two electrode contacts in the depth electrode as a local field potential. $Depth_1$ is the contact at the tip of he electrode; $Depth_4$ is approximately 1.5 cm above it. M_1 is an electrode on the mastoid process.

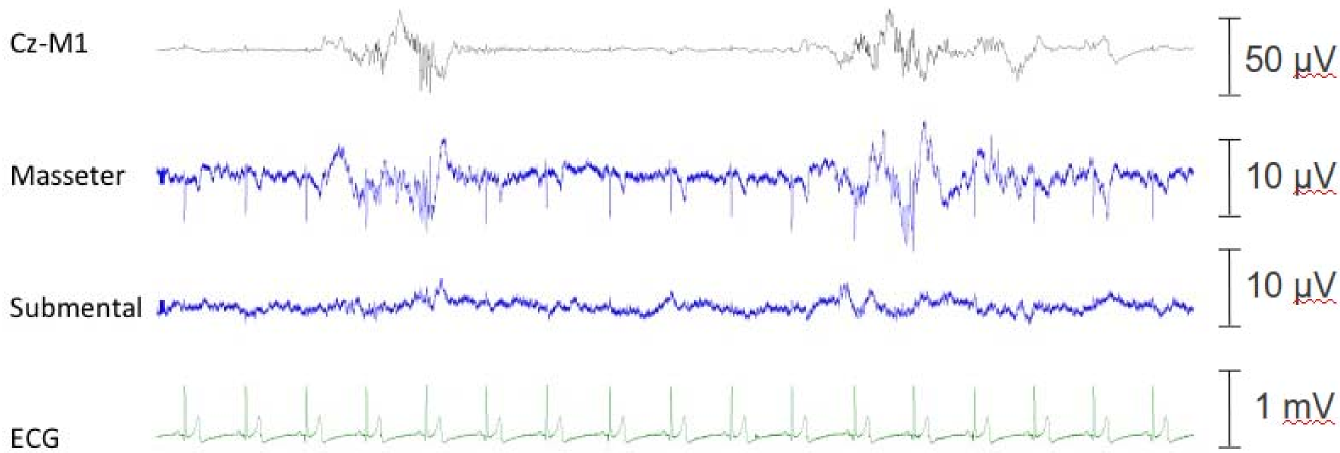


Fig. 3. This recording shows typical EEG burst suppression in propofol anesthesia as recorded from scalp (0.5 - 40 Hz). The second trace is from two electrodes placed on the skin over the masseter M_1 muscle 3 cm apart vertically under zygomatic arch 0.5 - 200 Hz). The patient is relaxed and no EMG is visible, only EEG and ECG. The next trace is from submental muscles, standard sleep recording montage (0.5-200 Hz). No ECG and minimal EEG is picked up by this derivation. Below is the ECG from chest electrodes (0.5- 500 Hz).

$\mathbf{J}^i[A/m^2]$ is the impressed current density field in the volume conductor, and σ is the conductivity tensor [S/m] [2].

The sensitivity distribution in the volume conductor can be established by applying the reciprocity theorem of Helmholtz with the Poisson equation (Eq. 2) applied to describe quasi-static bioelectric source-field problems [12], [13]. A source distribution, $\mathbf{J}^{\,i}$, containing only reciprocal source currents at the measurement electrodes raises a gradient potential distribution, $\nabla\Phi$, i.e. measurement sensitivity, according to the linear Poisson equation

$$\nabla \cdot (\sigma \nabla \Phi) = \nabla \cdot \mathbf{J}^{\,i}\,(\text{in } \Omega), \tag{2}$$

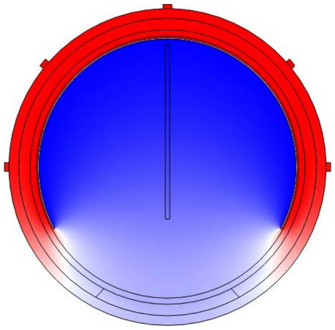
setting the Neumann boundary conditions equal to zero on the scalp

$$\sigma(\nabla \Phi) \cdot \mathbf{n} = 0\,(\text{on } \Gamma_\Omega), \tag{3}$$

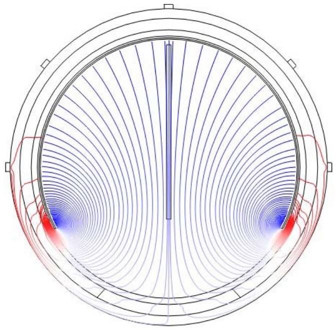
where σ is the electrical conductivity tensor, Φ is the electrical potential, $\mathbf{J}^{\,i}$ is the current source density, $\mathbf{n}$ is a vector normal to the surface, Ω is the volume of the head, and Γ_Ω is the surface of the head [14].

C. *Model and computations*

We calculate the sensitivity distribution in a spherical head model (Fig. 1) based on the original geometry of the Rush and Driscoll head model [15]. The tissues and their corresponding conductivities (resistivities) are listed in Table I [10]. Calculations of the sensitivity distribution are based on the principle of reciprocity and the solution of the numerical finite element method (FEM) of the EEG electrode sensitivity. In the present study we apply the scalp-to-skull conductivity ratio of 15:1 [16]. We calculate the sensitivity distribution of the synchronously active cortex. Electrodes are placeed on the surface E_1, E_2, and E_3 and implanted subthalamically as



(a)



(b)

Fig. 4. These subfigures illustrate the electric potential field generated by the synchronously active cortex. The subfigures depict: a) the potential field and b) the corresponding isopotential lines. The direction of sensitivity at each location is along the electric field, i.e. normal to the isopotential lines. The colors red and blue reflect the positive and negative potentials, respectively, whereas, the intensity of the color reflects the magnitude generated from the synchronously active cortex. The magnitude reflects the sensitivity of the measurements at the surface electrodes E_1, E_2, and E_3 and depth electrodes D_1 and D_2.

a bipolar lead between depth electrode contacts D_1 and D_2 (Fig. 1) to indicate the extent of the cortical generator.

III. Results

We show two examples of burst suppression EEG during anesthesia. Figure 2 shows an example of a burst recorded between a contact on the depth electrode $Depth_4$ and scalp electrodes frontal F_Z, parietal P_3, on the neck C_7 vertebra, chin, and the mastoid process M_1. The burst starts with a surface, i.e. scalp or skin, negative wave, followed by a positive level change.

Fig. 3 shows EEG burst suppression pattern in propofol anaesthesia recored from electrodes on the skin vertically below zygomatic arch. Note that these electrodes pick up both EEG and ECG. The signal below is from submental muscles, similar to the standard recordings in sleep. Note that EEG is at most minimally recordable here.

Fig. 4 depicts a spherical model of the brain, bony cranium, cerebrospinal fluid (CSF), and integrated subdermal and scalp tissue. At the bottom of the model exists a hole corresponding to the brainstem as well as the holes in the bottom of the cranium. The currents created by the cortex loop down and return via the CSF and subdermal tissue. This creates a high current density near the thalamus and therefore even two closely located electrode contacts record the cortically generated current.

IV. Discussion

Our results show that the recordings from depth electrodes are compatible with our model. A scalp positive slow wave of burst, which is synchronous in the whole cortex, creates a current loop which flows downward above the cortex, then turns upward and returns inside the hemispheres to close the loop under the cortex. When these currents pass the electrodes in the subthalamic nucleus, the high current density induces a voltage which reflects the electrical activity generated by the cortex. The exact flow of current is not known because our model geometry is unrealistic. These return currents could possibly be modeled as a double layer. However, we have also recorded burst suppression EEG of propofol anesthesia from electrodes on the masseter (Fig. 3), and this is compatible with the idea that part of the current loop flows under the skin relatively distant from the bony cranium (Fig. 4).

Our results show that cortical EEG can be recorded from electrodes in the subthalamic nucleus. The return currents of EEG, which is generated by the whole cortex synchronously, generates the strongest current in regions at the bottom of the brain, or in other words, near the edges of the cortex. This activity cannot be modeled with a dipole, but a dipole surface. Such dipole surfaces are seen during sleep slow waves, but also during epileptic discharges.

Also activity which involves a part of the cortex, create subcortical current loops and can therefore induce a signal between two contacts of the depth electrodes. These include epileptic spikes [4], spindles in deep propofol anesthsia [5] and even the occipital alpha rhythm. The relatively high voltage at a depth electrode offers the possibility to monitor sleep and seizures.

The size of the electrode is critical. Specifically, the size becomes more critical the closer the electrode is placed to the electrical source, i.e. the comparison of surface versus subdermal, cortical, intracortical and finally depth electrodes [17]–[20]. A sharp needle electrode is routinely used in localizing the nuclei where the stimulation electrode is inserted. These record unit activity, i.e. single cell activity. The stimulation electrodes are larger, and record activity of nearby and distant structures such as the cortex. If the electrode increases in size, for instance, if it is two centimeters long, it will record local activity coupled with distant activity. It is likely to record the large generators such as large areas of cortical activity. At present we do not know, what part of the signal recorded by depth electrodes, comes from subcortical structures and what comes from the cortex [4].

The addition of electrodes along the shaft of the depth electrodes should give an even more accurate measurement and also more local recording of cortical activity at the insertion point of the electrode. As the recording electrodes and electronics can be constructed in the stimulation electrode, depth-recording electrodes are available without extra surgery. The currents needed by amplifiers and transmitting the EEG to receivers, for instance, the skin, have minimal current consumption compared with stimulation. Much of the required electronics is currently available for cochlear implants that could be applied to depth recording technology.

V. Conclusion

Understanding the currents and potential distribution inside the brain is a prerequisite for trying to localize activity there with surface recordings and interpreting the recordings with intracerebral electrodes of different size, shape, and location. This calls for accurate realistic models of subcortical structures. At present the models are crude, but we are developing a theoretical basis for a more realistic model for the electrical fields of the head.

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