Brain death and bioelectrical brain activity

G. Pfurtscheller¹, G. Schwarz² and W. List²

¹Department of Computing, Institute of Biomedical Engineering, Technical University of Graz, and
²Institute of Anaesthesiology, University of Graz, Austria

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Abstract. The effect of mechanical vibration and light stimulation on the ongoing and evoked bioelectrical activity was studied in two cases with clinically defined brain death and two other patients with severe head injury, one of them with an isoelectric EEG. The importance of such stimulation sequences for the definition of brain death is discussed, with particular emphasis on mechanical vibration.

Key words: Quantitative EEG – Mechanical vibration – Brain death – Long-latency VEP – Long-latency SEP

Material and methods

Patients

Two ICU patients, both with clinically defined brain death after severe head injury, were subjected to combined quantitative EEG and evoked potential measurements. Further data from two other patients with severe head injury are also reported.

Somatosensory evoked potential (SEP)

For the study of the SEP we used a mechanical vibration stimulus lasting 1 s, connected via a plastic ring of 30 mm diameter to the distal digit of the index finger [13]. This type of stimulus is suitable for the ICU, because it can be applied quickly and easily, is much more natural for the sensory system than electrical stimulation of mixed peripheral nerves [11] and can be applied with the same stimulus strength in coma and after clinical recovery (the strength of electrical nerve stimulation has to be increased in unconscious patients [12]), does not show electrical interference, elicits a large amplitude SEP [7], and can also be used to study alpha or beta spindles in the comatose state [14]. A disadvantage of the vibration is the minimal mechanical noise; we did not make any effort to protect the patients from this noise, for instance by masking both ears. The vibration stimuli were generated 60 times at intervals of 10 s.

Visual evoked potential (VEP)

Light stimuli were applied by an array of red LEDs inserted in a pair of goggles, which allowed complete protection from outside light. The light stimuli lasted 1 s and were presented 60 times at intervals of 10 s.
EEG recording

Beside the normal clinical EEG (16 channels, time constant 0.3 s, upper cut-off frequency 70 Hz), four bipolar recordings were made from Cz-C3, Cz-C4 and approximately from O1-O2 and F3-F4 for computerized EEG and EP studies. An additional recording was made between the vertex and the left mastoid to obtain the vertex EP. A time constant of 0.1 s and an upper cut-off frequency of 35 Hz were used in the EEG machine. For the definition of “brain death”, clinical criteria and the clinical EEG were used.

Data processing

A minicomputer (PDP 11/23) was used for data processing. After anti-aliasing filtering (30 Hz, 120 dB/octave), each channel was sampled with 64/s, starting 3 s before stimulation onset and lasting 6 s, and checked automatically for artefacts.

The maximal energy of the late EP components is close to 10 Hz [9]; the sampling rate of 64/s is therefore sufficient to study late EP components, especially when only the shape and amplitude are of interest. For EP plotting, a reconstruction algorithm between sample points was used, resulting in a final resolution of 1.6 ms.

Results

Case 1

A 20-year-old man had a traffic accident with severe head injury on 11 April 1984, 10.00 a.m. The patient had a cardiac arrest during helicopter transport; cardiac resuscitation was successful. Upon arrival in the ICU clinical examination revealed deep coma, apnoea, wide pupils without reaction to light and no brain nerve reflexes; cerebral hemorrhage and massive brain edema were found in the CT. Other symptoms were polyuria and hypothermia. Catecholamines were administered. The first clinical EEG made at 14.30 showed some small-amplitude residual activity. Two hours later the EEG was isoelectric (Fig. 1). After starting periodical vibration stimulation, EEG activity returned (Fig. 2), first in the Cz-C4 recording and later in the Cz-C3 and Cz-A2 recordings, and persisted for about 10 min after the end of mechanical stimulation. Half an hour later the same procedure was repeated and EEG activity appeared again over the central region. Visual stimulation gave no response. The clinical status of brain death was unchanged during recurrence of bioelectrical activity.

Case 2

This was a 6-year-old girl with severe head injury (18.3.1982) and a subdural hematoma, massive brain edema and a small cerebral hemorrhage in the basal ganglia. The hematoma was removed surgically. Initially the clinical picture was dominated by signs of a midbrain syndrome; the brain edema increased and a mass delay occurred (CT control). The EEG was isoelectric; furthermore, hypothermia, hypotonia, bradycardia and apnoea were observed. Besides the isoelectric EEG, monophasic VEPs were preserved over the occipital pole (19.3.1982, 11:30; Fig. 3). Repetition of the measurement 5 h later again revealed an isoelectric EEG and a clearly preserved VEP. On both occasions the VEP had a wave at 150 ms and an amplitude of about 1 μV. During both VEP measurements, the spinal and brain nerve reflexes were negative, the pupils were wide and without light reactions; clinical signs of brain death already existed. No VEP was found in the other derivations from frontal or central regions.