Case Report

Recovery of N100 component of auditory event-related potentials and EEG after cardiac arrest during propofol sedation

S. M. Westerén-Punnonen1*, H. Yppäriä1, T. Musialowicz2, I. Korhonen3, M. Hynynen4 and J. Partanen1

1Department of Clinical Neurophysiology and 2Department of Anaesthesiology and Intensive Care, Kuopio University Hospital, Finland. 3VTT Information Technology, Tampere, Finland. 4Departments of Anaesthesiology and Intensive Care Medicine, Helsinki University Hospital, Jorvi Hospital, Espoo, Finland

*Corresponding author. E-mail: susanna.westeren-punnonen@kuh.fi

We report on the EEG monitoring of a patient who suffered an episode of postoperative ventricular fibrillation (VF) following coronary artery bypass grafting (CABG). VF initially caused a considerable suppression and slowing of the EEG. The recovery of cerebral function was evaluated by recording both EEG and auditory event related potentials (ERPs). Six hours after the episode of VF, when the patient was asleep but arousable to voice command, the N100 component of the auditory ERPs had recovered to the level measured before the operation, whereas the EEG was still very slow for that level of sedation. This may have been due to VF having less effect on the N100 component than on the background EEG. Our findings suggest that measuring evoked potentials may improve the evaluation of brain function after cardiac arrest.

Br J Anaesth 2005; 94: 626–9

Keywords: heart, ventricular fibrillation; monitoring, electroencephalography; monitoring, event related potentials; monitoring, intensive care

Accepted for publication: December 15, 2004

Cardiac arrest rapidly leads to acute cerebral anoxia and ischaemic neuronal damage in the brain. This is reflected in the EEG by an overall decrease in amplitude, an increase in slow activity and the disappearance of the fast activity. Both the reduction in amplitude and slowing of the EEG occur ~3–21 s after circulatory arrest.1 With continued severe ischaemia, all electrical activity will finally disappear (isoelectric EEG). Evoked potentials contain complementary information on brain function by reflecting the conduction and processing of a sensory stimulus in the brain. The activation of different parts of the sensory pathway results in variations in electrical activity, which can be recorded as evoked potentials. After a successful resuscitation, the recovery of brain function can be evaluated by monitoring both the spontaneous (EEG) and evoked (e.g. event related potentials [ERPs]) electrical activity from the scalp. We report this case to highlight the different behaviour of EEG and evoked potentials during recovery after a cardiac arrest.

Case report

A 68-yr-old female with a history of angina pectoris (NYHA III–IV) and dyslipidemia was scheduled for an elective CABG operation with cardiopulmonary bypass. The patient was initially included in a neuromonitoring study,2,3 for which informed consent had been obtained. The patient received propofol, alfentanil, isoflurane and pancuronium, and the operation was completed without complications. In the postoperative unit, sedation was started and maintained with propofol to keep the sedation level between Ramsay scores of 4 (asleep, a brisk response to a glabellar tap) and 6 (not responding to any commands) until she was haemodynamically stable. One hour after the operation, while EEG was being continuously monitored, the patient had an episode of ventricular fibrillation (VF). Successful defibrillation was completed within 3 min and sinus rhythm was restored. After resuscitation, the patient was hypertensive and ST-segment changes were detected on the ECG. Propofol infusion was continued, and nitroprusside and nitroglycerine infusions were started. The amplitude of the background EEG reduced remarkably 24 s after the onset of VF. Total spectral power dropped by 72% from the value measured before the VF. The EEG signals recorded at 2.5 h and 6 h after the VF reflected mainly slow-wave activity (Fig. 1) and the EEG and auditory ERPs were recorded as follows: 1 day...
before the operation (baseline), during deep sedation (2.5 h after VF; corresponding to a Ramsay score of 6) and 6 h after VF, when the infusion of propofol had been discontinued and patient was asleep but arousable by a loud auditory stimulus (corresponding to a Ramsay score of 4). Delta power was increased and SEF95 was reduced when compared with the values during the pre-arrest period.

At 4 h the patient was haemodynamically stable and propofol infusion was discontinued. She was extubated 8 h after the VF episode. Dobutamine infusion was started 13 h after the VF because of metabolic acidosis indicating inadequate organ perfusion. One week after VF, the EEG signal and computed spectral parameters were similar to the baseline values (Fig. 1 and Table 1). The N100 component of the auditory ERPs, which measures detection of the auditory stimulus, was not identifiable 2.5 h after the VF (Fig. 2). However, 6 h and 1 week after the VF it had returned to the baseline value. The patient recovered completely within 1 week, without any neurological deficits, and was discharged from hospital.

The EEG and ERPs were recorded with a four-channel EEG and ERP measuring machine (EMMA, Department of Clinical Neurophysiology, Kuopio University Hospital, Finland). During the recordings auditory stimulation at 60 dB above the absolute hearing level was applied to the right ear through headphones. The total number of stimuli

---

**Table 1** Computed EEG parameters at baseline and at three time points after VF. The data are presented as Fz (Cz). RS, Ramsay score

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline (RS 2)</th>
<th>2.5 h after VF (RS 6)</th>
<th>6 h after VF (RS 4)</th>
<th>1 week after VF (RS 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS power (µV^2)</td>
<td>10.4 (12.3)</td>
<td>10.4 (9.1)</td>
<td>19.1 (16.6)</td>
<td>13.5 (16.1)</td>
</tr>
<tr>
<td>MF (Hz)</td>
<td>8.9 (10.3)</td>
<td>1.1 (1.4)</td>
<td>2.0 (3.0)</td>
<td>8.4 (9.2)</td>
</tr>
<tr>
<td>SEF95 (Hz)</td>
<td>23.4 (24.5)</td>
<td>8.4 (11.2)</td>
<td>8.4 (10.0)</td>
<td>23.4 (23.7)</td>
</tr>
<tr>
<td>Delta (%)</td>
<td>24.0 (11.6)</td>
<td>69.3 (64.0)</td>
<td>73.8 (54.7)</td>
<td>24.5 (17.3)</td>
</tr>
</tbody>
</table>
in each recording session was 700 (595 standard stimuli and 105 deviant stimuli). The EEG signal was recorded from the midline (Fz and Cz) and the left and right central locations (C3 and C4). All electrodes were referenced to the right mastoid. Electrode–skin impedances were <5 kΩ. The raw signal was amplified and digitized at a rate of 279 Hz and stored on a PC for offline analysis.

The background EEG, recorded during the auditory stimulation, was analysed in 5-s epochs with a 50% overlap (300 s from the beginning of the recording). Serious artifacts were excluded by checking the maximum amplitude for each epoch; if the amplitude was >75 µV, the epoch was excluded. First, the RMS power was calculated from the EEG epochs. Then, the power spectral density (PSD) for each epoch was estimated using Welsh’s averaged periodogram method. Spectral entropy, spectral edge frequency (SEF95) and median power frequency (MPF) were computed from the PSD using a frequency range of 0.5–32 Hz. Relative powers for total (0.5–32 Hz), delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta-1 (13–20 Hz) and beta-2 (20–32 Hz) bands were also computed. Finally, the median of the accepted epochs was computed for all the EEG parameters.

For ERP analysis the data were transformed to epochs of −100 to 900 ms relative to the onset of each stimulus. After artifact rejection (rejection levels +75 µV and −75 µV), the responses to standard and deviant tones were averaged separately. The averaged data were filtered digitally with low-pass cut-off frequency at 15 Hz. The N100 component was defined as a maximum negative deflection between 80 and 150 ms after onset of the stimulus.

A CS3 monitor with an EEG module (Datex-Ohmeda, Helsinki, Finland) was used for additional EEG monitoring during postoperative recovery. The EEG signal was recorded from the mastoid–midline (A1–Fz and A2–Cz) and central–parietal (C3–P3 and C4–P4) derivations. A Datex-Ohmeda recording system digitized the EEG signal at 100 Hz, which was then stored and analysed offline. The spectral power was calculated for 5-s epochs with a 50% overlap in the frequency band 0.5–32 Hz. The median values were computed before the beginning of the VF (440 s determined from the ECG signal) and after the EEG pattern had changed as a result of the VF (290 s).

**Discussion**

The auditory ERPs recovered faster than the EEG after cardiac arrest. Six hours after VF, when the patient was asleep but arousable to a voice command, the difference between the EEG and the ERPs was marked. At that time, the N100 component had recovered to baseline level, whereas the background EEG still reflected mainly slow activity. The evolution of the EEG activity within the first hours after resuscitation from cardiac arrest provides an early prognosis of the recovery of brain function. Adding recordings of somatosensory or visual evoked potentials further improves the predictive performance.
Auditory evoked potentials reflect the conduction of an auditory stimulus in the cochlear nerve and brainstem (brainstem auditory evoked potentials [BAEP]), and further to the auditory cortex (middle-latency auditory evoked potentials [MLAEP]). Thus auditory stimulus processing in the brain is best reflected in the ERPs. The ERPs are affected by both the physical properties of the auditory stimulus and the psychological state of the subject. Therefore sleep, sedation and coma modify the amplitude, latency and shape of the ERP peaks. The most prominent ERP peak is the N100 component, which appears \( \sim 100 \) ms after the onset of the stimulus and has been associated with early discrimination of incoming stimuli. The loss of the N100 component during propofol sedation has been proposed as a marker of the transition from consciousness to unconsciousness, but other reports suggest that this component remains visible during general anaesthesia and deep sedation postoperatively. In addition, the visible ERP components have been shown to correlate with regaining of consciousness and a generally good prognosis in comatose patients.

Cardiopulmonary bypass may cause cerebral hypoperfusion and EEG changes. Furthermore, anaesthetic drugs and sedation level alter the EEG waveform. Vasoactive drugs given after the resuscitation may also contribute to the EEG waveform. Therefore the effect of these factors on various parameters of the EEG cannot be completely excluded in this case. During sedation, the patient had considerably reduced values in some EEG parameters (MPF and SEF95) after VF compared with patients who recovered from CABG without complication. In our case the delta power was increased when compared with that of patients recovering normally. This may imply that the slowing of the EEG was mostly caused by the cardiac arrest.

The N100 component was not identifiable when the patient was deeply sedated. This might have been due to the pharmacological effect of sedation. We have reported recently that six of 26 patients recovering normally from CABG did not have a detectable N100 component during deep sedation. In the present case, the N100 component had recovered to baseline level during moderate sedation, even though the background EEG was still very slow. This may suggest that the N100 component recovers from propofol sedation more quickly than the EEG. However, VF initially caused a considerable suppression and slowing of the EEG, and during moderate sedation the background EEG was still very slow for that sedation level. Therefore it can also be speculated that VF had a lesser effect on the N100 component than on the background EEG. Thus our findings might also suggest that the recording of evoked potentials may improve the evaluation of the brain function after cardiac arrest.

**Acknowledgement**

Funding for the work reported here was provided by the IBIS Project (EU–BIOMED2, BMH4–97–2570).

**References**