A NEUROPHYSIOLOGICAL BEDSIDE TECHNIQUE FOR MONITORING URAEMIC BRAIN DYSFUNCTION

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Introduction

Encephalopathy is a typical sign of uraemia and is regarded as its most serious complication. Haemodialysis treatment is able to reverse cerebral symptoms, but can induce brain dysfunction itself, too — e.g. the disequilibrium syndrome. Early detection and sensitive monitoring of encephalopathy could give valuable information about threatening cerebral complications and about adequacy of regular dialysis treatment (RDT).

Patients' complaints and the neurological status are not sensitive enough for detection of slight brain dysfunction. Reproducibility of findings is insufficient for follow-up-studies. The electroencephalogram (EEG), used in many clinical investigations, is influenced to a considerable extent by drugs and changes of vigilance, too, so that the validity of the EEG for the follow-up studies is reduced. For such purposes quantitative neurophysiological data are preferred. The suitability of evoked EEG potentials for detection of uraemic encephalopathy was tested in several studies [1,2,3,4]. Evoked potentials are EEG waves occurring with constant latency after auditory, visual or somatosensory stimuli. Evoked potentials are usually superimposed and masked by irregular, i.e. not stimulus-related EEG activity. Averaging of many EEG responses increases amplitude of evoked, i.e. stimulus-related potentials, but decreases amplitude of irregular EEG activity. Thus isolation of evoked potentials from background is possible [5].

Latencies of visual and somatosensory evoked potentials were found to be prolonged in renal patients [1,2,3,4]. Latencies of late components analysed in these studies are influenced by changes of vigilance, drugs and habituation phenomena, however, so that reproducibility is not yet sufficient [6]. Early components of auditory evoked potentials (AEP) were found to be relatively constant and are not influenced by changes of vigilance or habituation [5,6,7,8]. Therefore the suitability of early AEP components for detection and follow-up control of uraemic brain dysfunction was investigated in patients on RDT.
Methods

The normal range of AEP latencies was examined in 32 healthy subjects. Investigations were done in 43 patients (19 males, 24 females) on RDT. Mean age of patients and of controls did not differ significantly.

A diagram of signal processing is given in Figure 1. Amplifiers, averager, scopes, plotter and stimulator were mounted in a rack and could be moved to the bedside in the renal unit. EEG was recorded by Ag-AgCl surface electrodes from vertex and mastoid. Artefacts were avoided by using isolation pre-amplifiers placed near the head of the patient. The system frequency bandpass ranged from 2 to 5000Hz. 1024 responses were averaged by a 20 bit-minicomputer (2048 addresses, bin-width: 40μs). Auditory click stimuli of 0.1ms length and 10s⁻¹ frequency were applied by earphones at 60 dB above hearing threshold. Averaging of EEG segments was triggered by the stimulator.

![Diagram of signal processing](image)

Figure 1. Diagram of signal processing in analysis of auditory evoked EEG potentials (AEP)

Results

Early AEP components regularly observed are shown in Figure 2. Positive components were called P₁, P₂, P₃, P₄, negative components N₁ and N₂. Latencies were measured from stimulus to peak. Latencies of these early AEP components were found to be exactly reproducible at different days and were not influenced by drugs, changes of vigilance or habituation phenomena (Figure 3).

AEP latencies were found prolonged in most patients on RDT, but were found prolonged in any patient with uraemic symptoms (nausea, asterixis, drowsiness) (Figure 4). During initiation of RDT the first haemodialysis in some
patients caused an actual increase of AEP latencies, possibly due to disequilibrium, but then AEP latencies improved within a few weeks (Figure 5).

Figure 2. Early components of AEP. Two thousand and forty eight responses averaged. 
t: time

Figure 3. Reproducibility of AEP-latencies: AEP (each representing average of 1024 responses) of the same subject at 10 different days
Figure 4. Latencies of AEP-component N2 in healthy subjects and in patients on regular dialysis treatment. Hatched zone: normal range

Figure 5. Latencies of AEP-component N2 during initiation of regular dialysis treatment

Conclusions

1. Measurement of AEP latencies using a minicomputer is a practicable bedside technique well suited for routine purposes.
2. AEP latencies are sensitive indicators of even slight brain dysfunction.
Reproducibility of AEP latencies is high, since early AEP components are not influenced by drugs, changes of vigilance or habituation phenomena.

3. AEP latencies are valid parameters of uremic encephalopathy. The test makes a follow-up control of brain dysfunction in RDT patients possible. In several studies AEP latencies were well correlated with clinical conditions.

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