Continuous EEG monitoring in the Intensive Care Unit

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Abstract. Continuous electroencephalography (cEEG) seems a suitable modality to detect both cerebral ischaemia and epileptic seizures. In this review we will provide background information on EEG monitoring, discuss features of cerebral ischaemia and epileptiform activity and describe the pitfalls and caveats in performing and interpreting cEEG in the Intensive Care Unit.

Introduction

Whereas intensivists can closely monitor the function of various organ systems of Intensive Care Unit (ICU) patients, they are less well informed about the function of the brain. Common practice is a repeated neurological examination, but this can be hampered when sedative and/or anaesthetic agents are administered. Patients with an acute brain disorder are prone to various kinds of central nervous system complications, particularly epileptic seizures and cerebral ischaemia [1]. Both insults may further impair outcome, and can be treated [2]. As outlined in this article, continuous electroencephalography (cEEG) may detect both types of secondary injury.

Other frequently-employed techniques are less suitable to detect these types of injury for various reasons. Magnetic resonance imaging (MRI) and computed tomography (CT) provide a detailed ‘snapshot’ of the anatomy of the brain, but cannot record pathologic processes in contrast to cEEG which measures brain function and pathologic processes continuously. Also, transportation to the radiology suite for MRI and CT is an attendant risk for critically ill patients. The use of MRJ is further limited as most patients need metal devices for support. Transcranial Doppler (TCD) measures changes in blood flow velocity in the main intracranial arteries and can be used for continuous monitoring. A low flow state as well as high flow velocities may indicate cerebral ischaemia [3]. However, using this technique, it is not possible to measure blood flow at tissue level or diagnose epileptic seizures. Another frequently-used modality is intracranial pressure measurement (ICP). ICP monitoring however, provides only indirect information on cerebral oedema and cerebral blood flow. Oxyhemoglobin in the jugular bulb (SjvO\textsubscript{2}) reflects the balance between cerebral oxygen delivery and consumption. Although low SjvO\textsubscript{2} may indicate ischaemia, the method is not accurate, as overall cerebral blood flow in the whole hemisphere is estimated, and no information is obtained on possible regional differences [4].

Less frequently used diagnostic tools for brain monitoring are positron emission tomography (PET), single photon emission computer tomography (SPECT), Xenon-enhanced CT (Xe/CT), brain tissue oxygen monitoring (PbO\textsubscript{2}), cerebral microdialysis, Laser Doppler flowmetry (LDF) and near infrared spectroscopy (NIRS) [1,5]. An overview of frequently-used monitoring modalities is presented in Table 1.

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<td>Continuous EEG</td>
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TCD - transcranial doppler, ICP - intracranial pressure, NIRS - near infrared spectroscopy, EEG – electroencephalography, PbrO\textsubscript{2} - brain tissue oxygen monitoring, LDF - laser doppler flowmetry, CT - computed tomography, MRI - magnetic resonance imaging, PET - positron emission tomography, SPECT - single photon emission computer tomography, Xe/CT - Xenon-enhanced CT

Table 1: Summary of frequently-used neuromonitoring techniques

EEG is a cheap and non-invasive technique to assess cerebral activity. EEG has a reasonable spatial and high temporal resolution, is tightly linked to cerebral metabolism, sensitive to ischaemia, and the best available method for the detection of epileptic activity [6,7]. EEG may also be used to assess prognosis and level of sedation. Digital EEG and improvements in storage capability make 24-hour a day registration possible. Despite these strengths, EEG is still not part of standard monitoring in the ICU.

In this review we will provide background information on the EEG, discuss EEG features of cerebral ischaemia and epileptiform activity, and describe the pitfalls and caveats in performing and interpreting continuous EEG in the ICU.

The EEG

The EEG is a registration of cerebral electrical activity of the brain. It detects potentials of 1000 times smaller amplitude than the electrocardiogram (ECG). The potentials recorded with EEG are summed excitatory and inhibitory postsynaptic potentials in neuronal dendrites, particularly in the most superficial regions of the cerebral cortex. However, also deeper located structures such as the thalamus and brain stem generate potentials, albeit of lower amplitude, which reflect the functional state of these brain structures. The EEG can be regarded as a composition of numerous signals, each with its own frequency and amplitude. The frequency
of these waves is expressed in hertz (Hz), and can be divided in five bands: alpha (8-13 Hz), beta (13-35 Hz), gamma (>35 Hz), delta (<4 Hz) and theta (4-8 Hz). Figure 1 shows a normal EEG recording. EEG characteristics are described in Table 2. The amplitude usually varies from 20-200 microvolts. Another important feature is reactivity of the EEG to various intrinsic and extrinsic factors such as behavioural state (awake, drowsy, sleeping), opening of the eyes, painful stimuli and noise.

Abnormal changes in the EEG can be divided in three categories; 1) deterioration of normal background patterns, 2) appearance of abnormal patterns, and 3) disappearance of all activity. Abnormalities recorded by a specific electrode usually have their origin in the underlying part of the cortex. More generalized changes can also arise from deep structures such as the diencephalon, or can reflect a global pathologic process such as metabolic encephalopathy.

Analysis of EEG

Analysis of continuous and emergent EEG data in ICU patients is not straightforward, as intensivists and other ICU staff are not trained in reviewing the EEG, and clinical neurophysiologists are not usually stationed in the ICU. The ability of intensivists and other ICU staff to identify pathologic processes using EEG data is at a level low enough to cause concern; an overall mean correct response rate of 61% for recognition of epileptiform discharges was observed [8]. A simple educational intervention led only to a modest improvement, with an overall mean correct response rate of 67% [8]. Also, there is considerable interobserver variability in EEG interpretation among experts [9]. Furthermore, evaluation of long-term EEG registrations is very labour-intensive.

Recently, various tools for quantitative EEG analysis have been developed to overcome the above drawbacks. These can be fitted with a bedside alarm system to warn the ICU staff whenever significant changes in the EEG are detected by the software. An example is an analysis of frequencies in defined EEG episodes using power spectra, called compressed spectral arrays (CSA). In a power spectrum the square amplitudes in a frequency band of an EEG episode are charted (for an example see Figure 2). In a CSA, time changes in these power spectra are plotted (Figure 3). Several hours of recorded EEG data can thus be compressed into one image which may reveal patterns such as periodically recurring seizures. However, there are several limitations regarding CSA. First, CSA is influenced by factors other than ischaemia and seizures alone and is not a very sensitive or specific method for seizure detection. Second, due to data compression subtle EEG changes are overlooked and therefore CSA can not completely replace the overall visual EEG interpretation [10].

Technical considerations

cEEG monitoring in the ICU has some technical difficulties, which are not present in the EEG laboratory or in the operating theatre. Electrode maintenance is one of the major problems to be faced [10,11]. Electrodes are frequently dislocated during patient transport, nursing care, clinical examination or by the patient if in a confused or agitated state. Other problems around electrode placing are skull oedema or defects, and the presence of drains.

The occurrence of artifacts may be another problem of cEEG monitoring in the ICU. Artifacts may mimic pathologic processes resulting in unnecessary imaging or even treatment. Artifacts may result from environmental noise (surrounding devices such as a haemofiltration apparatus, intravenous fluid pumps, monitors, ventilators), skin conductivity changes (sweating), and mechanically from patting the patient during physiotherapy or from forced-air warming systems. In clinical practice it will not be possible to eliminate all these factors. To minimize unnecessary diagnostic procedures and treatment, it is important to document the origin of artifacts. One approach may be simultaneous video recording [10]. Reducing the number of channels used, may also decrease the odds of artefacts [12].

Cerebral ischaemia

Cerebral ischaemia is a frequent complication in critically ill patients [13,14]. An estimated 30% of patients with aneurysmal subarachnoid
haemorrhage suffer from secondary ischaemia [14]. Ischaemia also often complicates ICU stay in patients after severe traumatic brain injury [15]. Early detection of cerebral ischaemia at a potentially reversible stage, could improve outcome in these patients.

Before the introduction of the CT scan, much experience was obtained in using EEG to diagnose stroke. From these older studies it has become clear that there is a close relationship between cerebral blood flow (CBF) and ischaemic changes in EEG activity. When CBF drops below 25-35 ml/100g/min, reversible neuronal injury occurs, which can be detected with EEG [16,17]. When CBF progressively declines, more severe alterations can be observed (Table 3 and Figure 4) [16-19]. In addition, clinical or angiographic evidence of vasospasm has been associated with ischaemic EEG patterns in 97% of cases [18]. Interestingly, ischaemic alterations can be seen within minutes, allowing prompt intervention. By contrast, ischaemia can only be detected using CT or MRI when it has been present for hours and infarction has occurred [20,21]. It should however be noted that the above changes on the EEG are mainly observed when cortical ischaemia is present. In subcortical ischaemia, the EEG is less sensitive, as no changes or only subtle focal theta activity may be seen [22]. Ischaemic changes of the EEG in patients with acute ischaemic stroke may normalize when haemodynamic augmentation is applied, and this may precede clinical improvement [23]. An improvement of the EEG abnormalities with a decrease in the percentage of slow wave activity has been observed after isovolaemic haemodilution. In addition, older studies reported that on increasing regional CBF after induced hypervolaemia and hypertension, EEG focal slow activity decreased [17].

Few studies have been performed on computerized analysis techniques to detect ischaemia using continuous EEG. In a study in which several EEG parameters were evaluated for the detection of delayed cerebral ischaemia in poor-grade subarachnoid haemorrhage patients, the alpha/delta ratio was pointed out as most sensitive and specific for detecting ischaemia. The alpha/delta ratio is computed by dividing the amplitude power in the alpha range by the amplitude power in the delta range. The described sensitivity was 100% with
76% specificity using a cut-off point of 10% alpha/delta ratio decrease from baseline [24].

EEG monitoring for cerebral ischaemia is a frequently-used tool during carotid surgery to allow timely introduction of a perioperative shunt. EEG recording during carotid surgery has been used as a model to study ischaemia detection using various computerized analysis methods. One of these methods is the Brain Symmetry Index which quantifies the asymmetry of the power spectrum of the EEG in both hemispheres. The Brain Symmetry Index appeared to correlate well with the severity of stroke as estimated with the National Institute of Health Stroke Scale (NIHSS) (correlation coefficient 0.86, p<0.01) [25]. In another study, various computerized analysis methods; spectral edge frequency variables, relative and absolute power from four spectral bands, were investigated for the diagnosis of ischaemia during carotid surgery [26]. In this study, the impact of anaesthetics on EEG parameters was also evaluated. The diagnostic characteristics of the various ischaemia detection programmes differed depending on the type of anaesthesia protocol with which they were used. Some worked better with certain protocols than others.

In summary, cortical ischaemia is closely reflected in EEG alterations, making cEEG monitoring a potential tool for early diagnosis. Several computerized analysis techniques have been developed to make cEEG monitoring feasible. These are however hampered by various interfering parameters such as the use of anaesthetics. Furthermore, it is not completely clear which algorithm has the best diagnostic performance for the detection of cerebral ischaemia in ICU patients.

**Epileptiform activity**

Epileptiform EEG activity can be divided into interictal and ictal activity. Interictal epileptiform EEG activity consists of isolated spikes or spike-and-slow-wave complexes. A spike is a triphasic wave with a duration up to 80 ms with at least twice the amplitude of the background activity. The combination of a spike with a slow wave is called ‘spike-and-wave’ complex; together they usually last around 0.3 s. Ictal EEG alterations include rhythmical runs of spike-and-slow-wave complexes of 2-4 Hz (Figure 5) as well as runs of rhythmical beta, alpha or theta waves with increasing amplitude and decreasing frequency during seizure evolution. These EEG alterations lead to the diagnosis of ‘electrographic seizure’. Thus, not every spike is a seizure, and also not all electrographic seizures consist of spikes. Electrographic seizures have a relatively abrupt onset and last from 5 seconds to 2 minutes. If seizures last longer, status epilepticus must be considered. If electrographic seizures are accompanied by clinical manifestations, they can be called clinical epileptic seizures. If electrographic seizures lack clinical manifestations, especially motor manifestations, they can be called ‘non-convulsive seizures’ or, if prolonged, ‘non-convulsive status epilepticus’. However, the exact definition of non-convulsive seizures (NCSs) remains under discussion, since clinical manifestations may be subtle and easily overlooked. Also, EEG patterns exist that are periodic rather than continuously rhythmical, without clear aetiology. There is no general consensus whether these patterns should also be considered to be ictal epileptiform activity. Only one set of EEG criteria for NCSs has been published so far [27]. There is no scientific information on the interobserver reliability of the diagnosis.

From EEG studies in the ICU, we know that electrographic seizures frequently complicate the course of disease in neurocritical care patients. However, clinical recognition of electrographic seizures in ICU patients is hampered, since a large proportion of electrographic seizures are non-convulsive [1]. Conventional, short-term EEG registration may not detect these events, as seizure activity may be present for relatively short periods only [1], and the optimal moment for registration is unclear as it is not completely known which clinical features correspond with non-convulsive seizure activity. Using long-term EEG monitoring, seizures have been described in 19-34% of neurological ICU patients [6, 27-29]. Of 198 patients with altered mental status undergoing urgent EEG, non-convulsive seizures were

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**Figure 4**: Ischaemic EEG alterations in the left hemisphere
In recordings over the left hemisphere (L), there is an increase in slow activity and loss of beta frequencies. Over the right hemisphere (R), beta activity can be registered.

**Figure 5**: Epileptiform activity
Over the right hemisphere (R) spike and wave patterns are registered, indicating focal seizure activity.
observed in 37% [1]. In more specific populations of neurocritical care patients, seizures were detected using EEG in 29% of patients with intracerebral haemorrhage, in 18% with subarachnoid haemorrhage, in 22-33% with traumatic brain injury, in 8% with coma, and in 13-48% after convulsive status epilepticus [1,11,30]. It is difficult, however, to precisely determine the frequency of seizures as in most studies both the study population and the EEG seizure patterns were ill defined. A variety of seizure patterns have been described in the critically ill. However, some EEG patterns including periodic and stimulation-related patterns are not unambiguously ictal. Further research is needed to establish the clinical importance of these patterns, and whether it is necessary to treat them as seizures.

As mentioned above, the vast majority of epileptic seizures in ICU patients are non-convulsive [28,29]. Ongoing seizures can be suspected based on subtle clinical clues [2,6] such as a prolonged “post-ictal state” following generalized convulsive seizures, or acute onset of impaired consciousness or fluctuating consciousness. Also subtle movements including facial twitching, eye deviation and nystagmus may have an epileptic origin. Seizures may further present with episodic blank staring, aphasia, automatisms, elevation of intracranial pressure and acutely altered behaviour without obvious aetiology. Such clinical features may also be seen in ICU patients without seizures. Without EEG it is difficult to judge if these clinical features will correspond with electrographical seizure activity.

Non-convulsive seizures (NCSs) and non-convulsive status epilepticus (NCSE) are associated with a worse outcome especially when they persist for a longer period [2,31,32]. Of patients diagnosed with NCSs, 36% died with NCSs diagnosed within 0.5 hour of onset, 39% died with a diagnostic delay of greater than 1 but less than 24 hours and 75% died with a diagnostic delay of more than 24 hours [2]. The underlying pathophysiological mechanisms remain unclear [31,33]. It could be that NCSE represents an often lethal, intrinsic metabolic cascade caused by epileptic activity, or that NCSs are an epiphenomenon. Experimental research in rats has shown that externally induced status epilepticus (SE) causes increased metabolic demand and blood flow, and elevated intracranial pressure, leading to neuronal necrosis [34]. In humans with underlying brain pathology this increase in CBF, metabolic demand and ICP might explain worse outcome. However, since the underlying brain pathology in patients with NCSs or NCSE is often severe, that by itself could lead to a poor outcome. When adjustments were made for the severity of the underlying pathology, the presence of NCSs was still related to worse outcome [31].

Various tools have been developed for quantitative EEG analysis to detect epileptic activity, including spectral analysis and brain symmetry index, amplitude integration, detection of spikes and rhythmic changes as well as algorithms based on changes in nonlinear dynamics. These tools have been reported to detect 70-93% of seizures, with a false detection rate of between 0.3 to 5/hour [35-41]. Although various strategies have been compared in temporal lobe epilepsy [41], in ICU patients it is currently unclear which seizure detection algorithm has the best diagnostic properties. Also the exact diagnostic value of the various tools remains unclear, since epileptic activity was not studied in a random sample of ICU patients and EEG data was free of artefacts.

**Conclusion**

Advances in computer and networking technology have made long-term EEG monitoring feasible in critically ill patients. In principle by using these techniques, both cerebral ischaemia and epilepsy can be detected early. Further development of computerized analysis will be needed for routine implementation in ICU patients. In addition, various algorithms to detect ischaemia and epilepsy, and to recognize artefacts, should be compared so that the optimal software can be developed.

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**References**


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**Table 3: EEG alterations in relation to variations in cerebral blood flow (CBF)**

<table>
<thead>
<tr>
<th>CBF (ml/100g/min)</th>
<th>EEG alterations</th>
<th>Frequencies (Hz)</th>
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<tr>
<td>25-35</td>
<td>I. Loss of fast beta frequencies</td>
<td>&lt;13</td>
</tr>
<tr>
<td>18-25</td>
<td>II. Slowing of the background</td>
<td>5-7</td>
</tr>
<tr>
<td>12-18</td>
<td>III. Slowing into the delta range</td>
<td>1-4</td>
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<tr>
<td>&lt;8-10</td>
<td>IV. Flatting of the EEG:</td>
<td>- Burst suppression</td>
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<td>- Continuous suppression</td>
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**Abbreviations**

- CBF: Cerebral Blood Flow
- NCSE: Non-convulsive status epilepticus
- NCSs: Non-convulsive seizures
- ICU: Intensive Care Unit
- EEG: Electroencephalogram
- SE: Status Epilepticus
- ICP: Intracranial Pressure
- CBF: Cerebral Blood Flow
- NCSs: Non-convulsive seizures
- NCSE: Non-convulsive status epilepticus
- ICU: Intensive Care Unit
- EEG: Electroencephalogram
- SE: Status Epilepticus
- ICP: Intracranial Pressure
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