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EEG monitoring in the intensive care unit

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Abstract

Performing EEG in the intensive care unit (ICU) has many technical, environmental and resource-related challenges. Recording audio and video with EEG is always preferable for recognising artifacts and stimulus-evoked changes. Non-epileptic movements are very common is the critically ill, and their recognition prevents inappropriate overuse of AEDs. Nonconvulsive status defined by EEG is commonly detected in the ICU by continuous EEG monitoring. It is unknown whether such EEG findings are a cause or consequence of coma, or whether they worsen or simply reflect outcome. EEG is the first and only real-time monitor of epileptic seizures, and continues to be a powerful measure of cerebral function in the seriously ill.

INTRODUCTION

EEG is the first and only real-time monitor of epileptic seizures, and is a powerful measure of cerebral function in the seriously ill. Performing EEG well in the ICU has many technical, environmental and resource-related challenges. Sources of artifacts include patient movements (active and passive), sweating and extrinsic (electrical and mechanical).

The electrode - skin interface is the weakest link. Meticulous attention to electrode application and maintenance are required. Standard metal disc electrodes with adhesive paste are adequate for a diagnostic recording, but for longer studies application with collodion is more secure. Skin breakdown can be a problem after a prolonged period. Electrode caps are quick, easier and are helpful for very restless patients, but are artifact prone and vulnerable to inaccuracies of placement. MRI compatible silver-silver chloride impregnated plastic disc electrodes are also available.[1]

Placement of a limited number of electrodes is no substitute for the full 10-20 array because of poor sensitivity and specificity for epileptiform abnormalities.[2]

Recording of audio and video with EEG is always preferable for recognising artifacts mimicking seizures and stimulus-evoked changes.

The duration of the EEG in ICU is determined by the clinical context (and resources): whether a diagnostic EEG of 20-30 minutes, sometimes leaving leads on, or continuous EEG (cEEG). Resourcing determines who does the EEG, when and for how long, and who reads it and when. For cEEG, various techniques of digital EEG processing can allow efficient review of data but these techniques easily mistake artifacts for cerebral activity, so it is essential to verify changes with an informed examination of raw EEG.[3]

Questions we are expected to answer in the intensive care unit include:

IS HE SEIZING OR NOT?

Non-epileptic movements with posturing, rigidity, tremors, chewing and autonomic changes are very common is the critically ill, and whilst they could be seizures, they usually are not[4] (no seizures in 98% of 371 consecutive EEGs in the ICU at Royal Perth Hospital). Excluding the diagnosis of seizures prevents inappropriate overuse of AEDs, and this is a core role of EEG in the ICU.

Clinical misdiagnosis of status epilepticus may occur with rapid use of neuromuscular blockade and intubation of patients presenting with convulsive movements. EEG can produce valuable clarification, in association with withdrawal of sedation. On the other hand, up to 14% of generalised convulsive status epilepticus may evolve into nonconvulsive status (NCSE).[5]

What EEG patterns are ictal in the comatose? Recurrent seizures are easiest to recognise, with rhythmic discharges having spatiotemporal evolution in frequency (>4Hz), and morphology. Periodic patterns are the most difficult. Generalised periodic epileptiform discharges (GPEDs) may or may not be ictal, and the clinical context determines approach. PLEDs are usually interictal, but indicate a predisposition to seizures. Triphasic waves are non-ictal, may be sharply contoured

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and are recognised by their other characteristics including morphology, reactivity and phase-lag. Triphasic waves (and almost all periodic patterns) are suppressed by benzodiazepines, along with consciousness and respiration. Such suppression, in the absence of clinical improvement, does not diagnose NCSE. Ambiguous patterns can be seen, with stimulus-induced rhythmic, periodic or ictal discharges (SIRPIDs), commonly seen during cEEG.[6] They usually have no clinical correlate, and their treatment and prognostic implications are unknown.

Even in known status epilepticus, no prospective data compares the EEG endpoints of electrographic seizure termination versus burst suppression. Burst suppression may represent our goal in the treatment of status, but can also been seen with severe myoclonic encephalopathy, sometimes inappropriately described as "status myoclonicus". Burst suppression may have a clinical accompaniment during the burst with rapid eye opening, eyelid/face twitches, limb jerks or body stiffening. Severe myoclonic encephalopathy may follow any severe brain insult, especially after cardiac arrest, and the EEG may show burst suppression, unreactive GPEDs separated by isoelectric intervals, ± electrographic seizures.[7] Whilst outcome is aetiologically dependent, after cardiac arrest this clinical and electrographic picture has a very poor prognosis that is uninfluenced by aggressive treatment.[8-11]

Nonconvulsive status defined by EEG is commonly detected in the ICU by cEEG.[3] It is unknown whether such EEG findings are a cause or consequence of coma, or whether they worsen or simply reflect outcome. Thus far outcome of NCSE in the comatose patient is determined by the underlying aetiology and its complications, with interventions having little or no effect on outcome, with the exception of epilepsy. Unresolved questions include what EEG patterns should prompt aggressive treatment.

WHY ISN'T HE AWAKE (FAILURE TO AWAKEN)?

A normal EEG excludes impairment of consciousness at that time, and points to an altered psychological state. An abnormal EEG confirms abnormal cerebral function at that time, *but* the EEG may have been normal before treatment. Removal of sedating medications is required.

Coma patterns of various frequencies (alpha, theta, delta) may evolve, with prognosis determined by cause and progress with time. Whatever the frequency, invariant and non-reactive EEG patterns are associated with a poor prognosis, especially if ≥12 hours after the insult and if the patient is not sedated. They are mostly transient, and evolve into patterns that are more prognostically definite.[12]

IS HE ASLEEP?

Sedation is one of the commonest intensive care treatments. Excessive sedation is prevalent, and may prolong mechanical ventilation, increases complication rates and lengthens ICU stay. Monitoring techniques include clinical judgement and various sedation scores. EEG can assess consciousness in the paralysed and the level of "induced coma", but otherwise has a limited role, if any. The black box with one or two EEG channels and EEG signal processing is generally unhelpful, and it has no role in epilepsy monitoring.[13] Daily interruption of sedation is far more helpful, allowing neurological assessments and lowering mortality.[14]

IS HE DEAD?

We are frequently asked to give a prognosis in the critically ill. If the aetiology is known, EEG can often be a reliable predictor of poor outcome, for example coma after cardiac arrest with GPEDs, burst suppression or electrocerebral silence. However, in the ICU there are increasing problems recording electrical silence, let alone electrocerebral silence. In addition, 20% of patients with clinical diagnosis of brain death have residual EEG activity lasting for days.[15] Brain death is first and foremost a clinical and not an EEG diagnosis.

CONCLUSION

EEG is an essential diagnostic investigation in the critically ill, and it is likely that ongoing refinements will lead to its more extensive use.

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