REVIEW

ELECTROENCEPHALOGRAPHIC EVALUATION IN PATIENTS WITH IMPAIRED CONSCIOUSNESS

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ABSTRACT

The electroencephalographic examination (EEG) records spontaneous electrical activity generated in the cerebral cortex. The EEG is abnormal in almost all conditions associated with impairment of the level of consciousness. When consciousness is impaired the EEG becomes slowed (episodic or continuous), unresponsive to afferent stimuli, amplitude diminishes until eventually becomes flat.
Coma may have various causes. EEG is a diagnostic and prognostic tool in acute anoxic coma. EEG is the only diagnostic tool for detecting a non-convulsive epileptic status.

Key words: electroencephalography (EEG), coma, impaired consciousness

INTRODUCTION

The electroencephalographic examination records spontaneous electrical activity generated in the cerebral cortex.

Even more than 80 years after its introduction by Hans Berger, the electroencephalogram (EEG) remains as an important supplementary examination in the investigation of neurological disorders, because gives valuable and accurate information about cerebral function.

The value of EEG is limited by its high sensitivity to the electrical environmental noise, its dependence on sedative drugs, and its inability to test the brainstem.

The EEG is abnormal in almost all conditions associated with impairment of the level of consciousness.

Coma may have various causes. EEG is the only diagnostic tool for detecting a non-convulsive epileptic status. Locked-in- syndrome has usually normal EEG findings.

EEG FINDINGS IN COMATOUSE PATIENS

Altered state of consciousness may result from many cause, therefore EEG findings are variable, non-specific.

When consciousness is impaired the EEG becomes slowed (episodic or continuous) (figure 1), unresponsive to afferent stimuli, amplitude diminishes until eventually becomes flat.

Some special EEG patterns could be disclosed on EEG in comatose patients (1, 2):

- In **alpha-pattern coma** EEG discloses activity in alpha-frequency range, but distributed widely over cerebral regions (not only in posterior regions as alpha rhythm) and unresponsive to sensory stimuli**.** It was reported in comatose patients with brainstem stroke and hypoxia caused by cardio-pulmonary arrest and is usually associated with a poor prognostic.
- In **theta-pattern comas** EEG disclose widespread, persistent, unreactive activity from theta-frequency.

Sometimes the two patterns coexists (alpha-theta coma).

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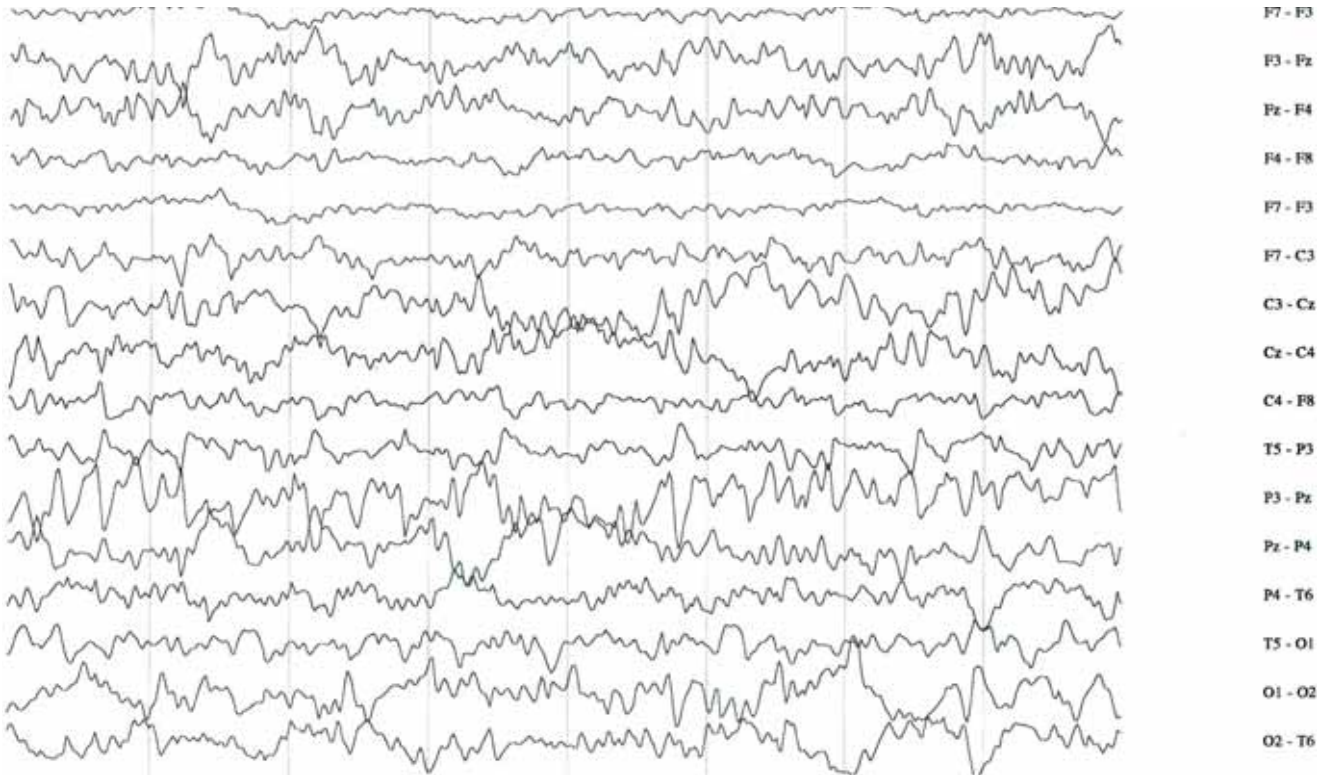

FIGURE 1. *EEG in HIV encephalitis – diffuse irregular high-voltage, arrhythmic theta-delta slowing centro-temporal bilaterally*

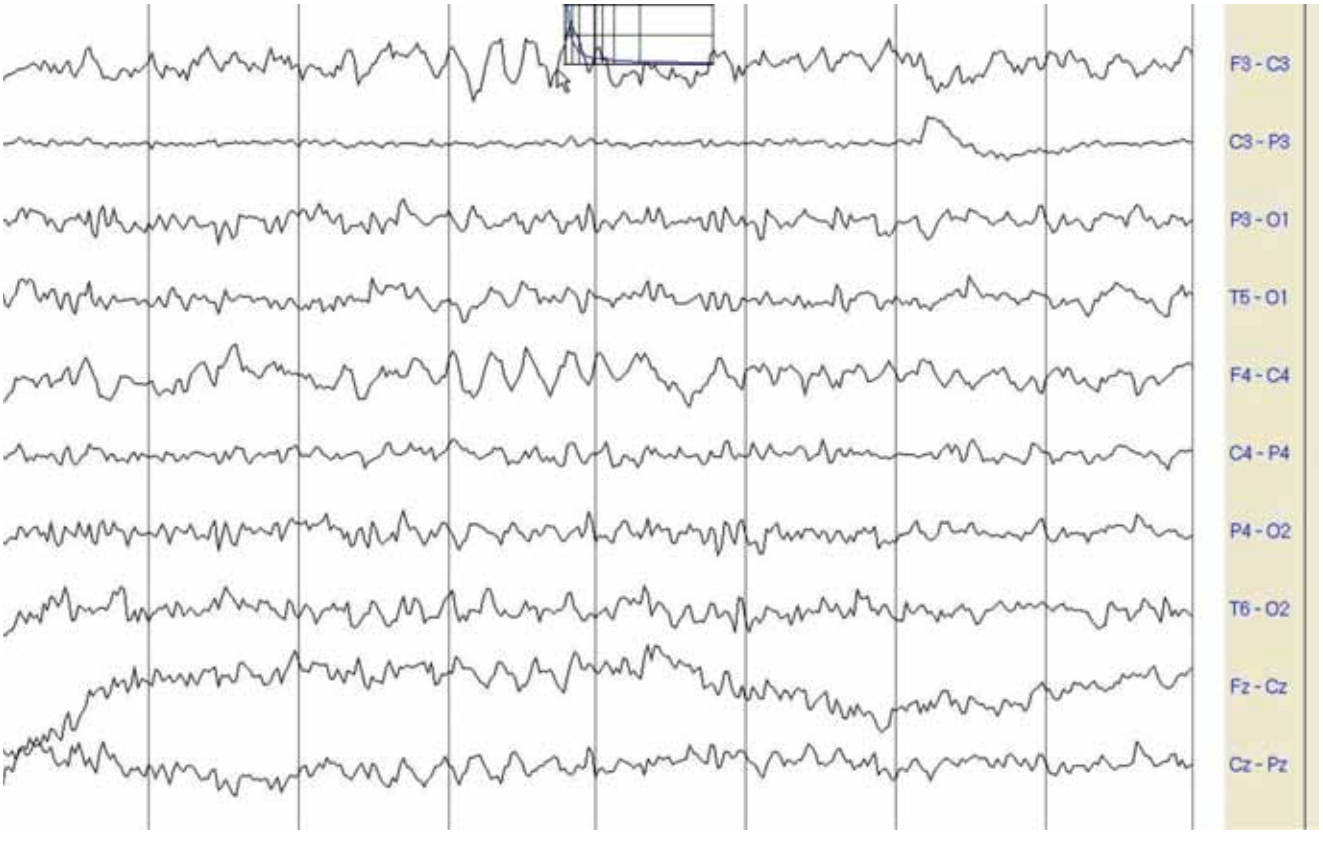

FIGURE 2. *EEG in paraneoplastic encephalitis – 47 years old women with lung tumor, FIRDA („frontal intermittent rhythmic delta activity")*

- In **spindle-coma** EEG shows activity like "sleep spindles", but with more diffuse distribution then normal sleep spindles, that could be related to dysfunction of midbrain reticular formation.
- **Intermittent rhythmic delta activity** is paroxysmal, with constant frequency, synchronous over two hemispheres, mainly frontally in adults (FIRDA – „frontal intermittent rhythmic delta activity") and occipitally in children (OIRDA – „occipital intermittent rhythmic delta activity") and reflects dysfunction of subcortical centers that influence cortical activity. (Figure 2)
- **Repetitive complexes** are disclosed on EEG in comatose patients.

PLEDs ("periodic lateralized epileptiform discharges") with 50-300μV and periodicity between 0.3-4 seconds occurs commonly in acute hemispheric lesions (infarcts, tumors).

When PLEDs occur independely over both hemispheres are named BIPLEDs ("bilaterally periodic lateralized epileptiform discharges"). The most common cause of BIPLEDs is diffuse pathologic involvement of the brain (anoxic encephalopathy, CNS infection).

- **Burst-suppression pattern** is characterized by bursts of high-voltage, mixed-frequency activity, separated by intervals of apparent inactivity that last few seconds-minutes. The bursts may be asymmetric or bisynchronous.

This burst-suppression pattern occurs during deeper stages of anesthesia, in comatose patients due to overdose of CNS depressant drugs. Also it can be found in diffuse encephalopathy following cardiac arrest, when is associated with a poor outcome.

According to experimental studies, the burst-suppression pattern could be due to hyperpolarisation of cortical neurons that determine EEG silence. This could be the consequence of increased inhibition of cortical synapses that also leads to the disconnection of the cortex from thalamic input.

Scollo-Lavizzari and Bassetti proposed classifications of EEG in comatose patients as follows (3):

– Grade I EEG- is dominant, normal alpha activity with theta-delta activities;
– Grade II EEG- is dominant theta-delta activity with still detectable normal alpha activities;
– Grade III EEG- is theta-delta activity without alpha activities;
– Grade IV EEG – is delta activity, low voltage, possibly with short isoelectric intervals or dominant, monomorphic, nonreactive alpha activity (alpha coma), or periodic generalized phenomena (spikes, sharp waves, slow waves) with very low-voltage background activity;
– Grade V EEG – is very flat to isoelectric EEG (less than 10-20 mV).

EEG Grades I to III are classified according to the definition of dominant finding. There can be multiple different waves in a single EEG recording. When a similar rhythm patterns comprise more than 50% of the result is defined as a dominant finding.

A typical wave pattern observed on EEG result is classified as Grade IV.

Grade V EEG results showed only isoelectic activity.

In *locked-in syndrome (de-efferented state)* the EEG is usually normal, even the patient is mute and tetraplegic because the lesion is on brainstem (basis of pons, bilaterally midbrain). Also, EEG is normal in hysteria mimiking coma.

In *akinetic mutism* a non-paralyzed, alert patient, capable of movement and speech lies motionless and silent for days-weeks. Usually there are bifrontal lesions.

EEG AND CAUSES OF COMA

Coma is a dynamic condition that may have various causes. Important changes may take place rapidly, often with consequences for treatment.

The EEG is a diagnostic and prognostic tool in **acute anoxic coma.**

In patients with coma resulting from hypoxic encephalopathy (e.g., after cardiac arrest), the EEG may reflect the severity of brain dysfunction, although the exact relationship among the EEG changes, the extent of neuronal damage, and consequent prognosis is still under study.(3,4,5)

More information might be obtained from EEG pattern changes over time. Particular EEG patterns such as burst suppression or generalized periodic discharges could be disclosed with sequential, repeated, prolonged or continuous EEG monitoring.

Therapeutic hypothermia and aggressive management of post resuscitation disease considerably improved outcome after adult cardiac arrest over the past decade.

Awakening from post anoxic coma is increasingly observed, despite early absence of motor signs and frank elevation of serum markers of brain injury [neuron-specific enolase (NSE), S-100B].

Electroencephalography (EEG) can be performed during therapeutic hypothermia or shortly thereafter. Continuous/reactive EEG background

strongly predicts good recovery from cardiac arrest, on the other hand – unreactive/spontaneous burst-suppression EEG pattern, together with absent N20 on somatosensory evoked potentials (SSEP), is almost 100% predictive of irreversible coma. Diffusion MRI may also help predicting long-term neurological sequelae of hypoxic-ischemic encephalopathy.(6)

EEG remains a unique tool for epilepsy assessment. Some patients with altered mental state have **nonconvulsive seizures or nonconvulsive status epilepticus**. If they lack of an accurate diagnostic and specific treatment, they will have an unfortunate outcome.

Nonconvulsive seizures are electrographic seizures with little or no overt clinical manifestations, EEG are absolutely necessary for the diagnostic.

Patients with clinically fluctuating abnormal mental status, even in the absence of history of epilepsy should perform EEG monitoring in order to disclose nonconvulsive status epilepticus. Also, EEG distinguishes between:

- absence status epilepticus (diffuse, bilaterally synchronous, continuous or discontinuous spike-wave at 2-3 Hz) (figure 3) and
- complex partial status epilepticus (paroxysmal activity unilaterally or bilaterally, alternating sites).

Patients with **nonconvulsive status epilepticus** have a high mortality rate, althought they may have a good outcome when treated with proper anti-epileptic therapy.

Thus, early detection and proper treatment of NCSE are important for good prognosis and to decrease the mortality.

Privitera, et al. reported that 37% of patients with altered levels of consciousness showed EEG and clinical evidence of definite or probable nonconvulsive status epilepticus by 30-45 min of routine EEG recording. (7)

In metabolic comas the slow waves become higher in amplitude as coma deepens, and high-voltage rhythmic delta pattern and a triphasic configuration are disclosed (for example in encephalopathy associated to renal failure). In coma due to barbituric intoxication fast activity initially replaces background rhythms. (8)

Tumors may affect EEG by causing compression, displacement, destruction of the nervous tissue or by leading to obstructive hydrocephalus (figure 4).

During last year we performed EEG in 41 comatose patients (24 females, 17 males; with mean age of 52,16 year old) admitted in University Emergency Hospital of Bucharest.

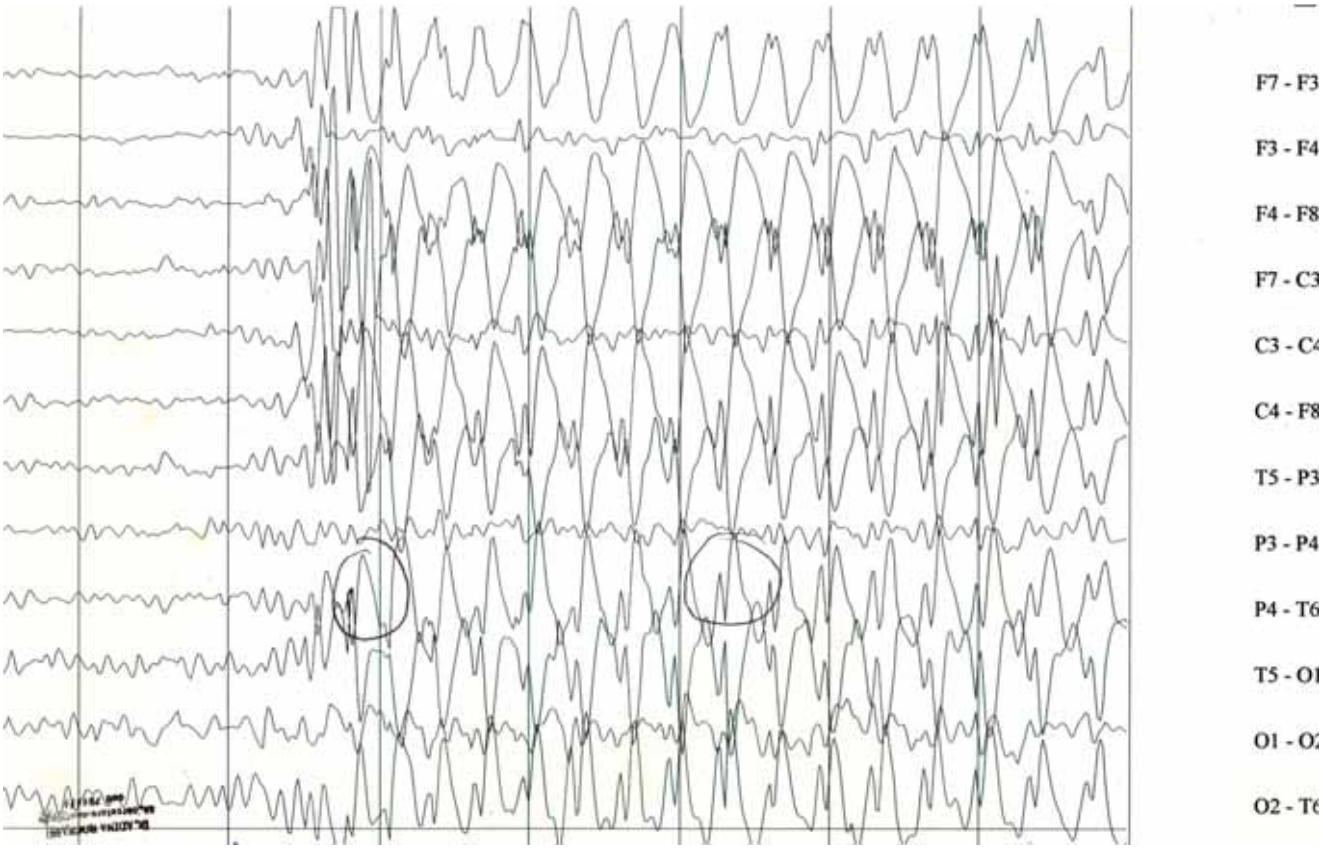

FIGURE 3. *Non-convulsivant seizures (absences) – 15 year old female – spike-wave discharges synchonous, bilaterally at frequency of 3 Hz*

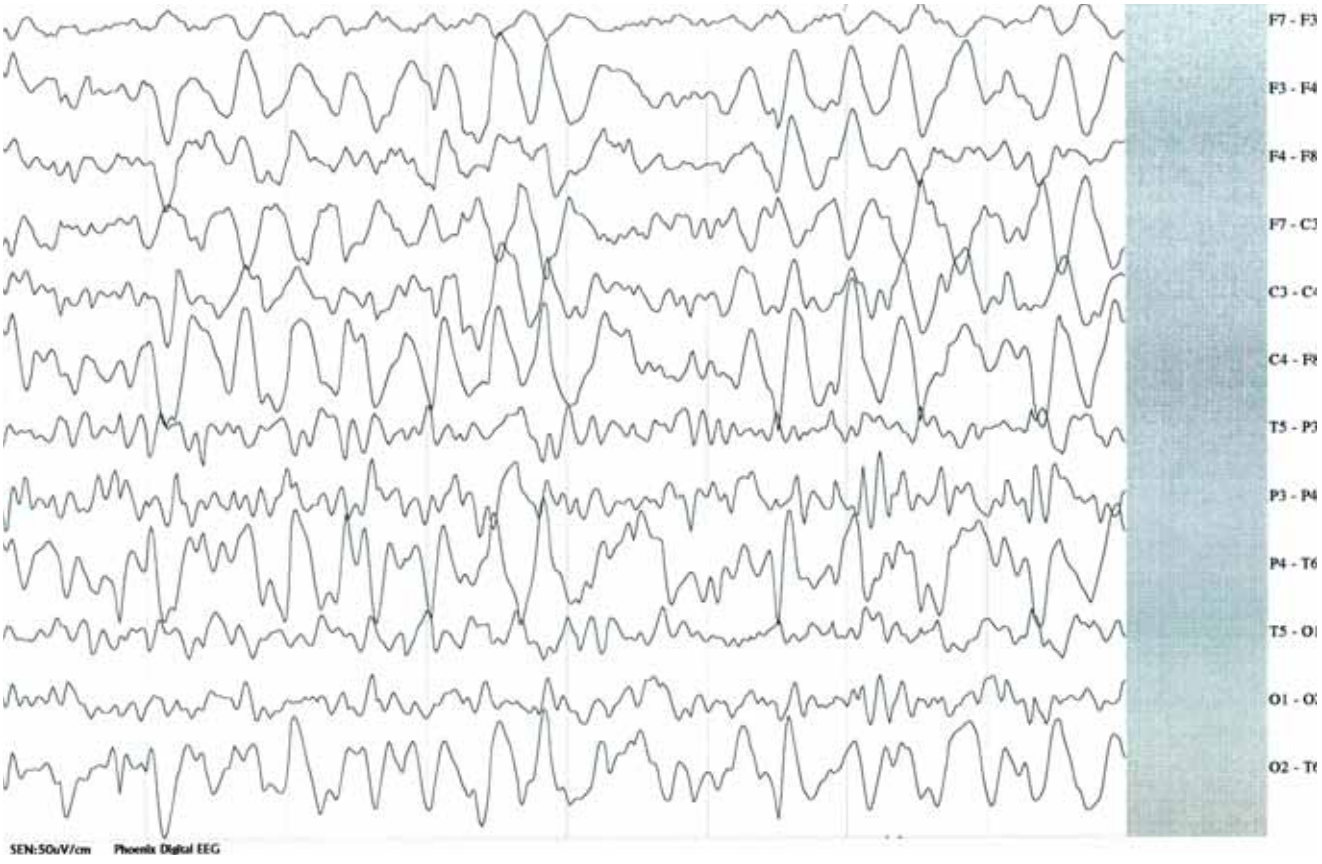

FIGURE 4. *Tumor of third ventricle (colloid chist) with secondary obstructive hydrocephalus – 68 years old male, EEG – massive slowing, high voltage delta activity replacing background activity*

In 10 patients the EEG was recorded repetitively in order to disclose brain death.

The patients were clinically evaluated by neurologists, applying also Glasgow Coma Scale (GCS) as a reliable and objective way of assessing a person's conscious state for initial as well as subsequent assessment.

The EEG recordings were made after the approval of the patient's families and of the local ethic committee.

The causes of coma were (figure 5): in 22% – anoxia; in 22% – epilepsy, in 17 % – cerebral trauma; in 7% – neuroinfections (meningitis, encephalitis); 7% – hemorrhagic stroke and 25% – other causes (cerebral tumor, paraneoplasic encephalitis, metabolic coma drug overdose, cerebral vasculitis).

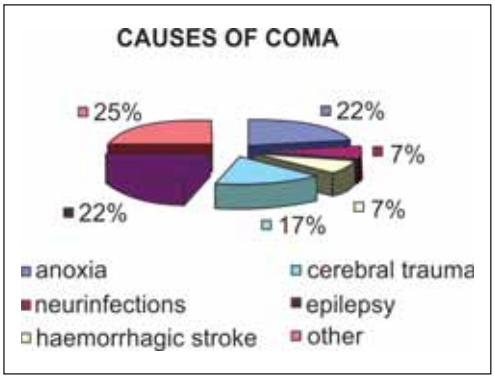

FIGURE 5. *Causes of coma (University Emergency Hospital Bucharest)*

CONCLUSIONS

EEG reflects brain function continuously and therefore holds a key place in the assessment and treatment of coma. EEG reveals immediate changes in coma, and can provide early information on cause and prognosis. EEG is the only diagnostic tool for detecting a non-convulsive status epilepticus.

Therapeutic hypothermia delays the recovery of motor responses and may render clinical evaluation unreliable. Repeated EEG scans increase diagnostic certainty and make it possible to monitor the development of coma. A new multimodal approach – including EEG monitoring – is required in order to improve clinical evolution after cardiac arrest.

The major technical challenge for the future will be the development of reliable tools for continuous EEG monitoring.

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