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SPECIAL ISSUE INSIGHT

EEG monitoring after cardiac arrest

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Hypoxic–ischaemic brain injury (HIBI) is the main cause of death and disability in patients who are comatose after return of spontaneous circulation (ROSC) from cardiac arrest [1]. The electroencephalogram (EEG) is a useful tool to assess the severity of HIBI and provide prognostic information. In addition, EEG can be used to diagnose epileptiform activity in patients with suspected seizures and monitor the effectiveness of antiepileptic treatment.

EEG for prognostication

The EEG signal reflects the function of cortical and subcortical neural networks. In the comatose patient after cardiac arrest, it provides a non-invasive means to assess the gradual recovery of these networks on time scales of hours to days. EEG is the most used prognostic tool after cardiac arrest [2] and it is widely available. Intermittent 30-min routine EEG is the most common approach, but many centres have adopted continuous EEG (cEEG) monitoring, facilitating the assessment of the evolution of brain activity over time.

The EEG signal is complex and the information from EEG experts may be difficult to interpret for the intensive care unit (ICU) physicians. Wide-consensus classification of EEG patterns in critical care has been included in the standardised terminology of the American Clinical Neurophysiology Society (ACNS). This terminology, initially published in 2013 and updated in 2021 [3], has been increasingly adopted in clinical literature and it contributes to a consistent definition of the main EEG patterns in HIBI.

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EEG background voltage, continuity, and reactivity

The basic EEG components are the background rhythms and the eventual superimposed patterns. The background is described according to its frequency, voltage, continuity, and reactivity to external stimulation. According to ACNS, the EEG background voltage is categorised as normal, low voltage (< 20 μV) or suppressed (< 10 μV). In terms of continuity, it is categorised as continuous, nearly continuous, discontinuous, burst attenuation/burst suppression, or suppressed (see Fig. 1 and definitions in ESM Table E1).

Time course of EEG background after arrest

Most patients have suppressed patterns immediately after cardiac arrest. A normal recovery process is characterised by gradually increasing amplitude and continuity. A transition towards a continuous normal-voltage EEG background (all activity ≥ 20 μV) within the first 12–24 h after cardiac arrest is among the most specific predictors of good neurological outcome [4]. The earlier this normalisation is detected, the better is the prognosis [5]. Presence of reactivity (defined as a change in frequency or voltage, including attenuation, following a predefined stimulus [3]) in a continuous or discontinuous normal-voltage EEG is associated with a higher likelihood of a good outcome [6]. However, interpretation of EEG reactivity is prone to interindividual variability [7]. A further source of variability is the type and intensity of stimulation.

On the other hand, persistent abnormalities of either EEG background voltage (suppression) or continuity (burst suppression) are strong predictors of a poor outcome after cardiac arrest and are often referred to as 'highly malignant' patterns [6]. While in the first 12–24 h after ROSC suppressed patterns have been reported in patients with good recovery, specificity for poor outcome prediction becomes close to 100% afterwards [8, 9]. For that reason, the 2021 ERC-ESICM guidelines for post-resuscitation care [10] recommend using EEG not earlier

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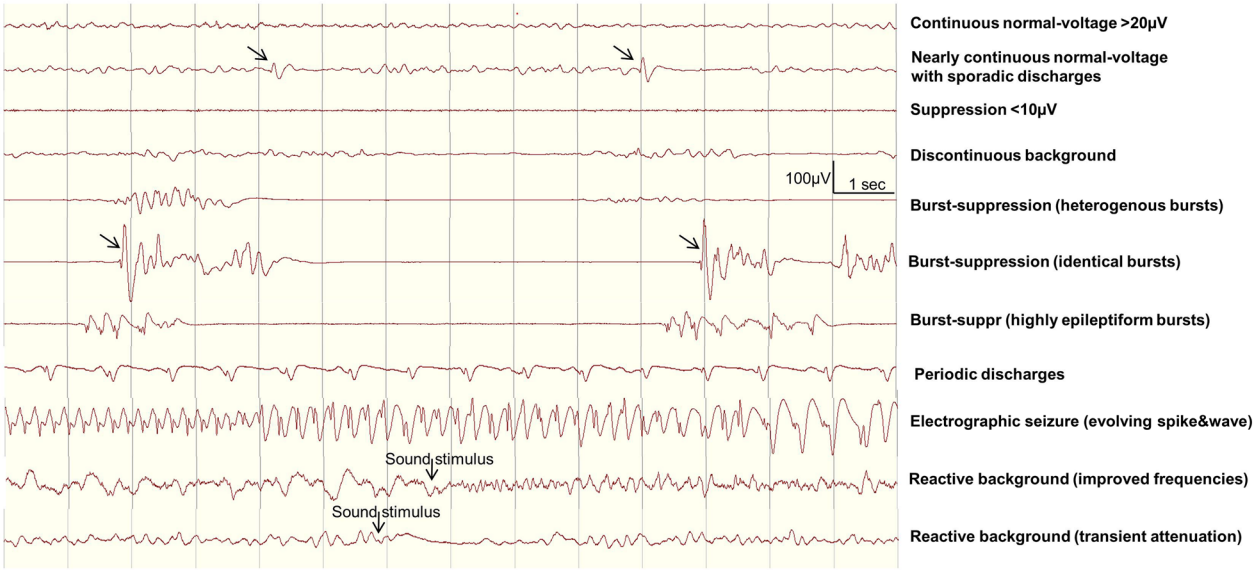

Fig. 1 Relevant EEG patterns after cardiac arrest. See ESM Table 1 for definitions

than 24 h to predict poor outcome (ESM Fig. 1). As for other predictors, the guidelines also recommend that malignant EEG patterns should be used in combination with other unfavourable clinical signs, to limit the risks of falsely pessimistic predictions.

Malignant EEG patterns are mostly transient, and their sensitivity for detecting patients with poor outcome may decrease to 25% or less at 48–72 h [11]. When bursts are highly epileptiform or appear stereotyped and repetitive ('identical' bursts, see definitions in ESM Table E1) [12] the prognosis is particularly poor, even if observed in the early hours after ROSC [9].

Sedation alters the EEG signal in a dose-dependent manner. With increasing dosing of sedation, the EEG background may decrease in amplitude, frequency, and continuity, but the typical 'highly malignant' patterns are not induced by usual sedative regimens [13]. Therefore, while ongoing sedation always needs to be considered when interpreting the EEG, it does not preclude its use for prognostication. Temperature control targeted at hypothermia may also potentially affect EEG. However, although ion channel kinetics and neurotransmitter release are temperature dependent, EEG effects of a body temperature of 32–34 °C are small. Moreover, the routine use of controlled hypothermia in HIBI is no longer recommended [14].

EEG to detect and treat seizures

Superimposed rhythmic and periodic EEG patterns (RPPs) that may reflect electrographic seizures have been reported in 10–35% of comatose cardiac arrest survivors [6]. Although isolated discharges on EEG hold no predictive value, generalised periodic discharges or electrographic seizures are associated with a poor neurological outcome [6]. An earlier occurrence of epileptiform activity, evolution from a suppressed background pattern, and lower background continuity are associated with a higher likelihood of unfavourable outcome [5, 6].

There is currently no consensus on what the optimal treatment strategy of seizures after cardiac arrest is [10]. Prolonged seizures may cause further brain damage through excitotoxicity, in which case aggressive treatment could be beneficial. In the recently published multicentre TELSTAR trial [15], a stepwise administration of antiepileptic agents and protocolised sedation (intravenous phenytoin plus benzodiazepines, followed by levetiracetam or valproic acid plus propofol in case of failure, and thiopental if the second step was unsuccessful) achieved complete suppression of all RPPs during 48 consecutive hours in 49/88 (56%) patients vs. 2/83 (2%) with standard care. However, at 3 months, neurological outcome did not differ between the two groups. The overall rate of poor neurological outcome was very high (92%). While the TELSTAR trial suggests that aggressive anti-seizure therapy may be futile in the most severe patients with post-anoxic status epilepticus, the benefit of seizure suppression in patients with seizures or status epilepticus lacking other conclusive unfavourable signs remains to be determined.

Continuous EEG recording

Full-montage, 21-electrode, cEEG recording is often perceived as labour intensive and mainly used in large university hospitals. However, cEEG eliminates the need for repeated measurements, facilitates appreciation of the evolution of EEG rhythms over time, and allows instantaneous detection of electrographic seizures. Since HIBI is diffuse, reduced montages hold promise to provide equally reliable results as full montages [16]. Simplified six-channel cEEG allowed ICU physicians to interpret the most clinically relevant EEG features after brief training and make decisions on patient management [17]. Remote expert interpretation of the EEG (tele-EEG) is an attractive way to make this technology available for hospitals lacking experienced personnel. Computer-assisted quantitative analyses, such as amplitude-integrated EEG [18] (ESM Fig. 2), can facilitate EEG interpretation at the bedside and help identify the most relevant features of HIBI. Finally, deep learning of artificial neural networks has recently been tested in its ability to predict neurological outcome from the EEG [19]. Results showed that the accuracy of this technique was comparable to standard visual EEG assessment by trained experts. These innovative approaches may facilitate bedside EEG monitoring in the future.

Supplementary Information
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Declarations

Conflicts of interest
CS is Associate Editor of Intensive Care Medicine. The other authors declare they have no conflict of interest.

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