Available online at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Clinical paper

Assessing both early and late EEG patterns improves prediction of outcome after cardiac arrest

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Abstract

Objective: Previously proposed "synchronous EEG patterns" predict poor outcome within 24 h after cardiac arrest (CA). We investigate the prognostic performance of these early EEG predictors in addition to the late EEG predictors (>24 h) recommended in the European post-resuscitation guidelines.

Methods: Observational substudy of the TTM2-trial including consecutive comatose resuscitated patients. Continuous EEG-monitoring (cEEG) was blindly assessed using the American Clinical Neurophysiology Society´s standardised EEG terminology and categorised into early EEG predictors (burst-suppression with identical or highly epileptiform bursts, or suppression with generalised periodic discharges) and late EEG predictors (heterogenous burst-suppression or suppression). Poor outcome was defined as modified Rankin Scale 4–6 at six months.

Results: Of 191 included patients, 53 % had poor outcome. Early EEG predictors had 100 %[CI 96–100] specificity at all time-points and maximal sensitivity 30 %[CI 21–40] *before* 24 h. Late EEG predictors had 100 %[CI 96–100] specificity *beyond* 24 h with maximal sensitivity 32 %[CI 21–43]. Using both early and late EEG predictors, and gradually adding cEEG-information from consecutive time-epochs, sensitivity increased to 49 %[CI 39–59] up to 36 h after CA (p = 0.001). A continuous background within 12 h predicted good outcome (sensitivity 61 %[CI 50–71]; specificity 87 %[CI 79–93]).

Conclusion: Searching for both early EEG predictors (e.g. identical burst-suppression) and late EEG predictors (e.g. heterogenous burst-suppression > 24 h) significantly improved sensitivity of poor outcome prediction without false positive survivors in this cohort. A self-fulfilling prophecy may have affected our results. cEEG during the first two days after CA identified half of the patients with a long-term poor outcome and half of the patients with a good outcome.

Keywords: Cardiac arrest, Prognostication, continuous EEG monitoring

Introduction

Despite advances in prognostication techniques, predicting neurological outcomes in post-anoxic coma remains a clinical challenge in the intensive care unit (ICU). Early and accurate prediction of good and poor outcomes is important for guiding treatment decisions, informing families, and optimising the allocation of healthcare resources.

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https://doi.org/10.1016/j.resuscitation.2025.110762
Received 21 May 2025; Received in Revised form 25 July 2025; Accepted 28 July 2025

Both the European and the American guidelines for post-resuscitation care recommend EEG as part of multimodal prognostication.[1,2] EEG is a valuable tool in the post-cardiac arrest setting for its ability to detect seizure activity and provide prognostic information.[3]

The European guidelines recommend using burst-suppression and suppression with or without periodic discharges as predictors of poor outcome in multimodal prognostication when seen beyond 24 h after cardiac arrest but suggest to delay this to even after the end of targeted temperature management (TTM) and clearance of sedatives.[2,4]

In post-anoxic coma, the EEG background pattern changes over time, both in patients with good and poor outcomes. It is well known that even in patients with eventual long-term good outcome, the EEG can be severely affected very early after CA. Thus, the prognostic value of EEG patterns is time-dependent due to the evolution of post-anoxic brain injury. The EEG patterns currently recommended in the European guidelines predict outcome only beyond 24 h after CA.[5–8]

There is evidence from the large multi-center study by Ruijter et al that so-called "synchronous EEG patterns" predict poor outcome with very high specificity regardless of timing and are reliable already in the first 24 h, during ongoing sedation and temperature management.[5,9–11] These patterns, with the exception of "abrubt-onset generalized burst-suppression", were recently included and strictly defined in the American Clinical Neurophysiology Society (ACNS) critical care EEG terminology, version 2021,[12] and include burst-suppression with identical bursts, burst-suppression with highly epileptiform bursts and suppression with periodic discharges.

In this substudy of the international multicenter Targeted Hypothermia vs Targeted Normothermia After Out-of-Hospital Cardiac Arrest (TTM2) trial,[13] we evaluate the prognostic performance of guideline recommended "late EEG predictors" (burst-suppression with heterogenous bursts or suppression) and previously proposed "early EEG predictors" (burst-suppression with identical bursts, burst-suppression with highly epileptiform bursts and suppression with periodic discharges), in isolation and in combination. The two strategies has been extensively investigated previously but to the best of our knowledge the added value of the combined approach, e.g. evaluating both early identical burst-suppression and late non-identical/heterogenous burst-suppression has not been explored previously. The combined approach may improve sensitivity for predicting poor outcome with EEG. This is important since prognosis remains uncertain in a large proportion of patients.

We hypothesise that burst-suppression with identical or highly epileptiform bursts or suppression with periodic discharges (early predictors), and suppression or heterogenous burst-suppression beyond 24 h (late predictors), each have high specificity for poor outcome, and that combining these improves sensitivity.

When performing continuous EEG-monitoring (cEEG) after CA the recordings are commonly reviewed multiple times during the first days. In the present study we also investigate the added value of assessing consecutive time-epochs of the cEEG-recording, thus gradually collecting prognostic cEEG information during the first 72 h for prediction of poor outcome.

There is extensive evidence that restoration of a continuous normal-voltage background at an early time-point predicts good outcome, but the proposed time-points vary in the literature. Therefore, we additionally aimed to further explore the temporal characteristics of cEEG for prediction of good outcome.

Methods

This is a retrospective observational substudy of the TTM2-trial (NCT02908308, 2017-2020), in which 1900 adult comatose patients were randomised to either temperature control with hypothermia at 33 °C or normothermia with early control of fever (≥37.8 °C), following out-out-hospital cardiac arrest.[13,14] Ethical approval was obtained in all participating countries.

cEEG-monitoring was performed at seven European sites in Sweden (Malmö, Lund, Helsingborg, Halmstad, Linköping), France (Versailles) and Switzerland (Lausanne). Patients underwent cEEG as soon as practically possible after ICU admission and were otherwise treated in accordance with the TTM2-trial protocol.[14] All patients were sedated to Richmond Agitation and Sedation Scale level -4/-5 for the full intervention period (40 h after randomisation). Sedation was allowed according to standard care also after the intervention period. After 96 h multimodal neurological prognostication was performed and if a poor prognosis was presumed according to trial criteria, withdrawal of life sustaining treatment (WLST) was allowed. Other non-neurological reasons for WLST included irreversible organ failure, a serious medical comorbidity, or other reasons that deemed further life-sustaining therapies unethical. Presence of two of the following signs predicted a poor outcome according to the trial protocol: Bilateral absence of pupillary and corneal reflexes, status myoclonus, unreactive burst-suppression or suppression beyond 24 h, neuroimaging with signs of global ischaemic injury, bilaterally absent somatosensory evoked cortical potentials (SSEP) and high serum levels of neuron specific enolase (NSE). At prognostication and WLST decisions during the trial "high NSE levels" were defined according to local routine, but in this retrospective analysis the definition was fulfilled if the highest NSE value between 48–72 h was above 60 ng/ml. If the prognosis was uncertain, active intensive care should be continued, and patients reevaluated daily. The treating team was not blinded to the results of prognostication, including the local EEG report. Thus the guideline recommended late EEG patterns (burst-suppression or suppression) were available during prognostication, but should be used in combination with other prognostic tools. The early EEG predictors were not part of the prognostication protocol, which stated that EEG patterns during ongoing targeted temperature management or sedation should not be used in decisions regarding WLST.

Functional neurological outcome was assessed using the modified Rankin Scale (mRS) at six months after CA. Follow-up was performed either face-to-face or by telephone. Good outcome was defined as an mRS score of 0–3 (no symptoms, no clinically significant disability, slight disability, or moderate disability) and poor outcome was defined as mRS 4–6 (moderately severe disability, severe disability, or death).[15]

cEEG was recorded with at least 8 electrodes, placed according to the international 10–20 system, including a reference and a ground electrode. The cEEG results, interpreted by local EEG-reviewers, were available for the treating critical care teams. After the trial ended inclusion, all cEEGs were retrospectively reviewed in consecutive 6-h epochs (from 6 ± 3 h to 72 ± 3 h post arrest) by one EEG specialist (SB or EW) blinded to all clinical data. Each EEG epoch was assessed for dominant background pattern, including presence of identical and highly epileptiform bursts, and presence of rhythmic and periodic patterns. EEG scoring was performed according to the ACNS standardised EEG terminology,

version 2021.[12] For each patient the reviewer also noted the time of first appearance of a nearly continuous normal-voltage (≥20 μV) background. See Fig. 1 and **supplemental Table S1** for definitions of early and late EEG predictors.

Statistical analyses were performed in R studio (2023.12.1 + 402), running on R (4.3.1).[16] Baseline characteristics, clinical variables and follow-up details are presented using descriptive statistics. Prognostic performance of EEG predictors is presented using sensitivity and specificity. Confidence intervals (CI) were calculated as 95 % Clopper Pearson exact intervals.

To assess the relationship between timing of recovery of a (nearly) continuous normal-voltage EEG background pattern (amplitude ≥ 20 μV and < 10 % suppression) and prediction of good outcome, sensitivity, specificity, and 95 % CI were calculated for each hour after CA.

The additional value of repeated cEEG evaluations over time was assessed in patients with cEEG recording started within 12 h after CA, having at least one epoch of cEEG between 12 and 24 h, and one epoch between 24 and 36 h after CA. Sensitivity, specificity, and 95 % CI were calculated for each additional cEEG epoch and statistical added value was assessed using McNemar test.

Results

In total, 191 patients were included in the cEEG substudy (**supplemental figure S1**). Demographic data is presented in Table 1. Median age was 68 years [IQR 61–75], and 144 patients (75 %) were male. At six months follow-up, 101 patients (53 %) had poor outcome. cEEG monitoring was started at a median of 7.4 h [IQR 5.8–9.2] after CA. The majority of patients received propofol sedation (96 % of poor outcome patients and 94 % of good outcome patients) and/or midazolam (24 % of poor outcome patients and 23 % of good outcome patients). Dosage is presented in Table 1. Multimodal prognostication was performed in 87 patients (46 %). WLST was performed in 71 (70 %) of the patients with poor outcome due to multi-organ failure, cardiac reasons or neurological reasons. In 42/71 (58 %) patients a presumed poor neurological prognosis was the only reason for WLST. In 30/42 patients an unreactive burst-suppression or suppression pattern beyond 24 h was documented in the local EEG-report which was available at the time-point of the decision to withdraw care.

Prediction of poor outcome

Early EEG predictors

Early EEG predictors of poor outcome, including suppression with superimposed periodic discharges and burst-suppression with identical or highly epileptiform bursts were exclusively observed in patients with poor outcome. Thus, specificity for prediction of poor outcome was 100 % [CI 96–100] in all time-epochs, see Table 2 and Fig. 2. Sensitivity for prediction of poor outcome was highest in individual cEEG epochs ≤ 24 h after CA with 30 % [CI 21–40], and decreased gradually after that to only 8 % [CI 3–16] after 36 h, see Fig. 3A and Table 2.

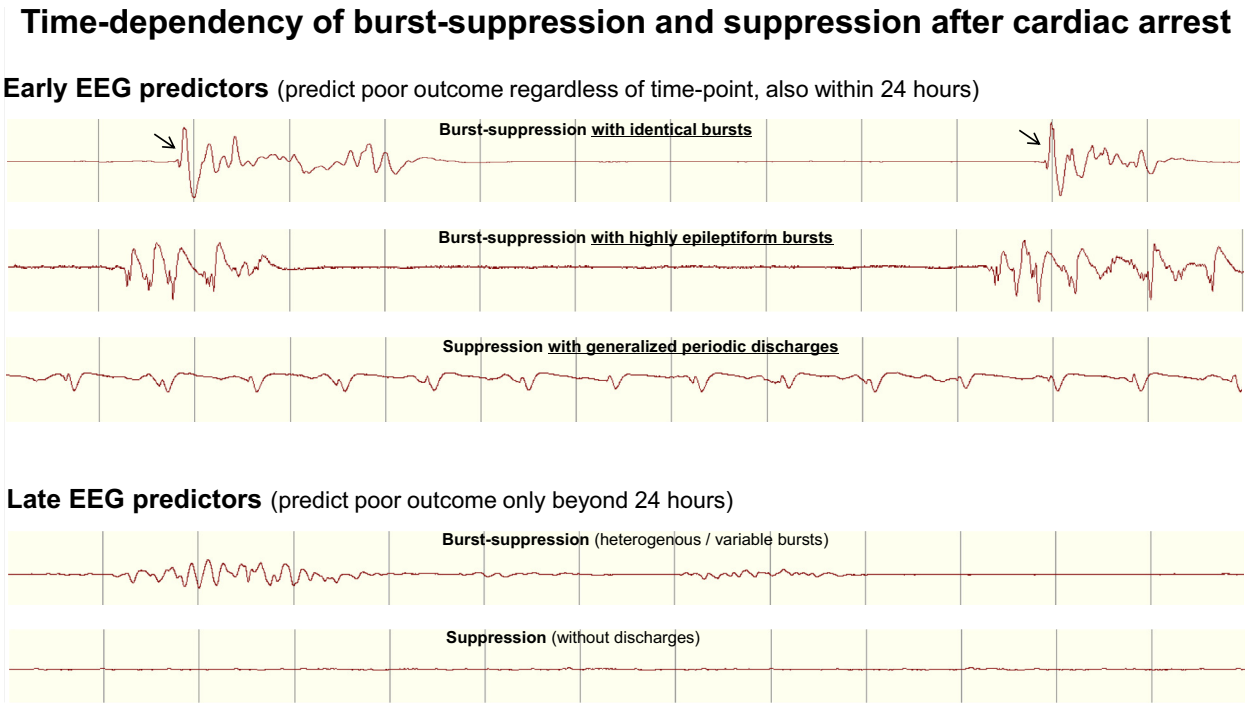

Fig. 1 – Subtypes of burst-suppression and suppression after cardiac arrest defined using the American Clinical Neurophysiology Society´s 2021 critical care EEG terminology. Early EEG-predictors also known as synchronous patterns (Ruijter et al, Annals of Neurology 2019) are predictive of poor outcome regardless of time-point after cardiac arrest also within 24 h (time-independent). Late EEG-predictors are predictive of poor outcome only beyond 24 h (time-dependent).

Table 1 – Prediction of poor outcome based on early EEG predictors (i.e. synchronous patterns) and late EEG predictors (heterogenous burst-suppression or suppression), and a combined approach. Prediction based on assessment of individual cEEG windows, as well as the additional value of evaluation of consecutive cEEG windows.

	Sensitivity	Specificity	TP	TN	FP	FN	N
A. Early EEG predictors[a]							
Start to 12 h	28 [19–39]	100 [96–100]	25	81	0	64	170
>12 h to 24 h	30 [21–40]	100 [96–100]	28	86	0	66	180
>24 h to 36 h	20 [12–29]	100 [96–100]	18	85	0	74	177
>36 h to 48 h	8 [3–16]	100 [96–100]	7	84	0	82	173
>48 h to 72 h	8 [3–16]	100 [91–100]	6	40	0	70	116
B. Late EEG predictors[b] (only presented > 24 h)							
>24 h to 36 h	30 [21–41]	100 [96–100]	28	85	0	64	177
>36 h to 48 h	24 [15–34]	100 [96–100]	21	84	0	68	173
>48 h to 72 h	32 [21–43]	100 [91–100]	24	40	0	52	116
C. Combined early[a] and late[b] EEG predictors (only presented > 24 h)							
>24 h to 36 h	42 [32–53]	100 [96–100]	39	85	0	53	177
>36 h to 48 h	29 [20–40]	100 [96–100]	26	84	0	63	173
>48 h to 72 h	34 [24–46]	100 [91–100]	26	40	0	50	116
D. Additional value of consecutive cEEG evaluations using combined early [a] (all time-epochs) and late[b] (>24 h to ≤ 72 h) EEG predictors. Prognostic ability from start of cEEG (median 7 h) up to a time-point is presented							
Start to 12 h[(a)]	28 [19–39]	100 [96–100]	25	81	0	64	170
Start to 24 h[(a)]	35 [25–45]	100 [96–100]	34	88	0	64	186
Start to 36 h[(a+b)]	49 [39–59]	100 [96–100]	49	89	0	51	189
Start to 48 h[(a+b)]	50 [40–60]	100 [96–100]	50	90	0	50	190
Start to 72 h[(a+b)]	51 [41–62]	100 [96–100]	52	90	0	49	191

[a] Includes suppression with generalised periodic discharges, burst-suppression with identical or highly epileptiform bursts.

[b] Includes suppression without discharges and burst-suppression with heterogenous bursts.

Late EEG predictors

Beyond 24 h after CA, late EEG predictors of poor outcome, including heterogenous burst-suppression (without identical or highly epileptiform bursts) and suppression (without periodic discharges), were observed exclusively in patients with poor outcome, therefore predicting poor outcome with 100 % specificity [CI 96–100], see Fig. 3A. Sensitivity for prediction of poor outcome was similar for all evaluated individual time-windows beyond 24 h (24–32 %). There were patients with good outcome and presence of these EEG patterns within the first 24 h after CA, see Fig. 2.

Combined approach and additional value of cEEG monitoring

When either an early EEG predictor (burst-suppression with identical or highly epileptiform bursts and suppression with periodic discharges) or a late EEG predictor (heterogenous burst-suppression and suppression) were present in individual time-epochs after 24 h, specificity to predict poor outcome was 100 % [CI 96–100] and sensitivity varied between 28 and 42 % with a maximum between 24–36 h after CA, see Fig. 3A.

When gradually adding prognostic information from early and late EEG predictors in consecutively evaluated cEEG windows, sensitiv-

Table 2 – Patient characteristics. Data presented as number (percentage) of patients unless otherwise indicated.

Characteristic	Poor outcome *N* = 101	Good outcome *N* = 90
Baseline characteristics		
Age, median [IQR], years	71 [63–77]	65 [59–72]
Male	70 (69)	74 (82)
Witnessed arrest	96 (95)	83 (92)
Shockable rhythm	55 (54)	75 (83)
Minutes to ROSC, median [IQR]	34 [22–47]	20 [13–30]
Hospital stay		
TTM 33 °C	51 (50)	43 (48)
Length of ICU stay, median [IQR], days	5 [4–7]	3 [3–5]
Highest NSE value up to 72 h, median [IQR] (N tested), μg/L	69 [35–171] (*N* = 88)	24 [20–30] (*N* = 80)
Propofol, cumulative dose first 72 h, median [IQR] (N patients), mg/kg	99 [46–175] (*N* = 97)	120 [74–155] (*N* = 85)
Midazolam, cumulative dose first 72 h, median [IQR] (N patients), mg/kg	0.66 [0.09–2.70] (*N* = 24)	0.70 [0.06–1.80] (*N* = 21)
Mortality	94 (93)	0 (NA)
WLST for any reason, No. (%)	71 (70)	0 (NA)
WLST for neurological reason only, No. (%)	42 (42)	0 (NA)
Time to death, median [IQR], days	5 [4–10]	NA

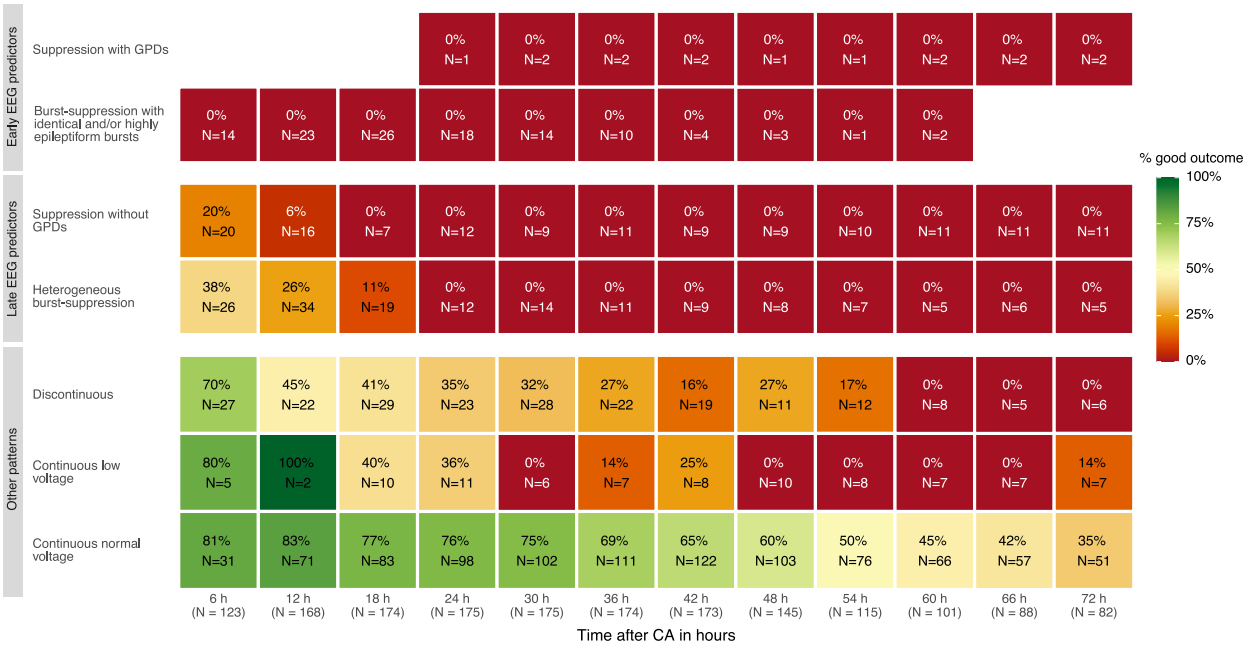

Fig. 2 – Each tile shows the percentage of patients with good outcome (or in other words, the false positive rate for prediction of poor outcome) and the total number (N) of patients with that EEG background pattern as the dominant pattern in each 6-h time-window, centralised around the given time. The x-axis also shows the total number of patients with cEEG evaluated in each time-window.

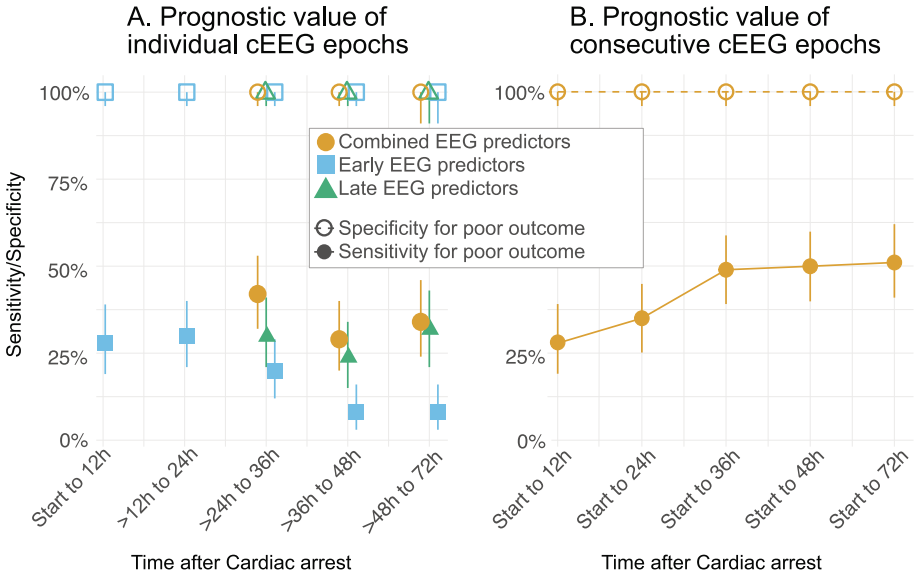

Fig. 3 – A. Prognostic performance of early EEG predictors, i.e. synchronous patterns (periodic discharges on suppressed background, burst-suppression with identical or highly epileptiform bursts), late EEG predictors (suppression, heterogenous burst-suppression). Performance of the early EEG predictors (blue) are presented for all individual time-windows and the late EEG predictors (green) are presented for time-windows beyond 24 h. The combined performance of early and late EEG predictors (yellow) in individual time-windows are presented beyond 24 h. B. Prognostic performance of combined early and late EEG predictors (yellow) and gradually adding prognostic information from consecutive windows, evaluated from start of cEEG (median 7 h) up to a time-point. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

ity to predict poor outcome significantly increased (p = 0.001) with monitoring up to 36 h, reaching 49 % [CI 39–59], see Fig. 3B. Sensitivity did not improve significantly if monitoring was further continued up to 72 h (sensitivity 51 %), see Table 2.

Relation between WLST and EEG prediction of poor outcome

During the whole monitoring period (until 72 h) in total 52 patients had an early or late EEG predictor of poor outcome. All of these patients had a poor outcome, WLST was performed in 47 (90 %), whereof 27 for neurological reasons only, and the median duration of intensive care was 4 days [IQR 3–5].

Five (10 %) of the 52 patients had one other pathological prognostic sign, apart from EEG. 36 (69 %) patients had two or more other pathological signs and fulfilled criteria for a poor prognosis according to both the TTM2-trial protocol and the European post resuscitation guidelines even without considering EEG.

Prediction of good outcome

The proportion of patients with a restored nearly continuous or continuous normal-voltage background gradually increased with time in both good and poor outcome patients, see Fig. 2. The restoration of the background occurred both earlier and in a larger proportion of good outcome patients compared to the poor outcome patients (median 10 h [IQR 8–17] vs 25 h [IQR 16–40] and continuity restoration in 98 % vs 60 % of patients within 72 h). The positive predictive value (PPV) for good outcome was highest, 81 % [CI 70–89], when return of a nearly continuous normal-voltage background pattern occurred $\leq$ 12 h. Thereafter, PPV to predict good outcome decreased with time and the majority of patients who had late restoration of background activity had a poor outcome, see supplementary table S3.

Discussion

In this prospectively collected cohort of a multicenter trial, we found that EEG patterns categorised as early and late EEG predictors reliably predicted poor outcome, with no false positives in this cohort. Early EEG predictors, including synchronuous patterns such as identical burst-suppression, had the highest sensitivity at time-points before 24 h after CA and thereafter sensitivity declined confirming previous studies.[5,9] Guideline recommended late EEG predictors, including non-identical/heterogenous burst-suppression and suppression, showed a relatively stable sensitivity between 24 and 72 h after CA, in line with previous studys.[4,21] Searching for both early and late EEG predictors is a novel strategy which identified even more patients with poor outcome and performed best between 24 and 36 h after CA. Since EEG patterns are often transient, assessment of an individual time-window was less informative compared to continuously evaluating the EEG. When using this combined strategy and gradually adding EEG information from before 12 h up to 36 h after CA (requiring cEEG) sensitivity significantly increased and half of all patients with long-term poor outcome could be identified.

A large previous study investigated the additional value of longitudinal EEG evaluations and found additional value up to 24 h only.[5] In that study so-called “synchronous EEG pattern” and suppression were included in the prediction of poor outcome. Our results also show that cEEG starting at an early time-point (within 12 h) and repeated evaluations of the cEEG are of additional prognostic value. In our study we included additional late EEG predictors such as heterogenous burst-suppression beyond 24 h after CA, which likely explains the added value up to 36 h.

Although the absolute number of patients per EEG pattern is limited, we found no patients with good outcome presenting with identical or highly epileptiform bursts or periodic discharges on a suppressed background (early EEG predictors) at any time-point, consistent with previous studies.[5,7,9,17] Late EEG predictors (heterogenous burst-suppression and suppression), on the other hand, were seen in patients with good outcome, but only within 24 h after CA, stressing the importance of the timing when these EEG patterns are used for outcome prediction. Given the high specificity for prediction of poor outcome, our results suggest that the separation into early and late EEG predictors of poor outcome is adequate, as illustrated in Fig. 2.

In this work, we made a clear separation of the burst-suppression patterns into heterogenous bursts in distinction to identical or highly epileptiform bursts, both defined in the latest ACNS terminology for critical care EEG.[12] We found different prognostic abilities of these burst-suppression subtypes in line with previous studies.[5,9] Whereas heterogenous burst-suppression might be seen in patients with mainly cortical damage, burst-suppression with identical bursts was described to be seen in patients with also damage to the deeper structures such as the thalamus and hippocampus.[18] The specificity of burst-suppression with identical or highly epileptiform bursts to predict poor outcome was 100 % in our cohort at all time-points, with sensitivity peaking before 24 h after CA and gradually decreasing afterwards. This in contrast to burst-suppression with non-identical/heterogenous bursts that reached 100 % specificity only beyond 24 h after CA and its sensitivity was similar at later time-points.

It is previously known that if post-anoxic status epilepticus starts from a discontinuous background prognosis is invariably poor.[19–22] We previously described that 25 patients in the present substudy cohort had definite or possible status epilepticus starting within the first day after CA or originating from a discontinuous background and all had a poor outcome. The vast majority (24/25) of these patients with an unfavourable status epilepticus also had an early or late EEG predictor of poor outcome, such as identical burst-suppression before start of status epilepticus or a late heterogenous burst-suppression.[23]

Abortion of cEEG monitoring after detection of an EEG predictor of either poor or good outcome appears likely not to affect the prognostic ability of EEG, but this was not further investigated in the present study. Importantly, early obtained EEG-information cannot be used in isolation for decisions regarding WLST but should rather be used at a later stage when the multimodal prognostication assessment is performed.

A major strength of our study is a well-defined cohort, with a conservative protocol regarding WLST. The TTM2-trial protocol allowed for WLST based on presumed poor neurological outcome after 96 h after CA, well after the period of cEEG monitoring we conducted in this study. However, an unreactive burst-suppression or suppression background pattern after 24 h was part of the multimodal prognostication and therefore, a self-fulfilling prophecy cannot be excluded.

Furthermore, the trial protocol demanded sedation until 40 h after randomisation. Thus, the vast majority of patients were sedated during cEEG monitoring, both in the poor and good outcome groups. Since we had no false positive patients with good outcome when

using EEG to predict poor outcome, we did not investigate the effect of various doses of sedatives on the predictive value of EEG.

Another strength is the early start of cEEG monitoring which allowed us to analyse early time-points, which proved important especially for good outcome prediction. Furthermore, EEG interpretation was centralised and performed by two experts blinded to clinical data, according to the current ACNS critical care EEG terminology, version 2021.

A limitation of our study is that prevalence of some EEG background patterns was low, which is why we remain mainly descriptive. This is also why we have not investigated the relationship between hypothermia or normothermia treatment and the appearance of early and late EEG predictors. However, the allocation to the treatment arms was equally distributed in patients with poor and good outcome. Furthermore, we did not study the additional value of EEG reactivity to external stimuli in this cohort. In this study we had no false positives for prediction of poor outcome with burst-suppression after 24 h, but a previous study indicated that the presence of EEG reactivity might be of value to detect false positives.[24] For good outcome prediction on the other hand, the presence of EEG reactivity has been described to be of additional value.[25,26] Since the treating team was not blinded for clinical EEG reports, we cannot exclude the influence of a self-fulfilling prophecy. However, the trial protocol for assessment of neurological prognosis was conservative and at a delayed time-point (96 h).

Conclusion

cEEG monitoring starting within 12 h and continuing 2 days after CA reliably identifies half of the patients with a poor outcome. Our results confirm that the previously proposed early "synchronous EEG patterns" (e.g. identical burst-suppression) reliably predict poor outcome also within 24 h after CA. A combined strategy searching for both these early EEG predictors and the guideline recommended late EEG predictors (e.g. heterogenous burst-suppression and suppression > 24 h) and assessing consecutive time-epochs significantly improves poor outcome prediction. Furthermore, cEEG identifies more than half of patients with long-term good outcome already within 12 h.

CRediT authorship contribution statement

Marjolein Admiraal: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Sofia Backman:** Writing – review & editing, Supervision, Methodology, Investigation, Data curation, Conceptualization. **Martin Annborn:** Writing – review & editing, Resources, Investigation. **Ola Borgquist:** Writing – review & editing, Resources, Investigation. **Josef Dankiewicz:** Writing – review & editing, Resources, Investigation. **Joachim Düring:** Writing – review & editing, Resources, Investigation. **Marion Moseby-Knappe:** Writing – review & editing, Resources, Investigation. **Stéphane Legriel:** Writing – review & editing, Resources, Investigation. **Hans Lindehammar:** Writing – review & editing, Resources, Investigation. **Anna Lybeck:** Writing – review & editing, Resources, Investigation. **Niklas Nielsen:** Writing – review & editing, Resources, Investigation. **Andrea O. Rossetti:** Writing – review & editing, Resources, Investigation. **Johan Undén:** Writing – review & editing, Resources, Investigation. **Tobias Cronberg:** Writing – review & editing, Resources, Investigation. **Erik Westhall:** Writing – review & editing, Writing – original draft, Supervision, Resources, Methodology, Investigation, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.resuscitation.2025.110762.

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