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# Characteristics and role in outcome prediction of continuous EEG after status epilepticus: A prospective observational cohort

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## Summary

**Objective—**Continuous electroencephalography (cEEG) is important for treatment guidance in status epilepticus (SE) management, but its role in clinical outcome prediction is unclear. Our aim is to determine which cEEG features give independent outcome information after correction for clinical predictor.

**Methods—**cEEG data of 120 consecutive adult patients with SE were prospectively collected in three academic medical centers using the 2012 American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology. Association between cEEG features and two clinical outcome measures (mortality and complete recovery) was assessed.

**Results—**In the first 24 h of EEG recording, 49 patients (40.8%) showed no periodic or rhythmic pattern, 45 (37.5%) had periodic discharges, 20 (16.7%) had rhythmic delta activity, and 6 (5%) had spike-and-wave discharges. Seizures were recorded in 68.3% of patients. After adjusting for known clinical predictive factors for mortality including the STatus Epilepticus Severity Score (STESS) and the presence of a potentially fatal etiology, the only EEG features (among rhythmic and periodic patterns, seizures, and background activity) that remained significantly associated with outcome were the absence of a posterior dominant rhythm (odds ratio [OR] 9.8; p = 0.033) for mortality and changes in stage II sleep pattern characteristics (OR 2.59 for each step up among

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these categories: absent, present and abnormal, present and normal; p = 0.002) for complete recovery.

**Significance—**After adjustment for relevant clinical findings, including SE severity and etiology, cEEG background information (posterior dominant rhythm and sleep patterns) is more predictive for clinical outcome after SE than are rhythmic and periodic patterns or seizures.

### Keywords

Neurocritical care; Terminology; Status epilepticus; Electroencephalography background activity

Status epilepticus (SE) is a potentially fatal condition requiring comprehensive assessment and rapid treatment.[1] Continuous electroencephalography (cEEG) has an important role in this setting for seizure detection and treatment guidance[2] and is recommended for SE management.[3] The role of continuous or repeated routine electroencephalography (EEG) in outcome prediction is less clearly defined,[4] and with inconsistent findings.[5,6] Moreover, most available data regarding EEG patterns and clinical outcome association were published before the introduction of the 2012 American Clinical Neurophysiology Society's (ACNS) Standardized Critical Care EEG Terminology.[7] This terminology clearly defines rhythmic and periodic patterns (RPPs) and also EEG background features. Recently a high interrater agreement has been reported using this terminology.[8] Although recent studies have examined some EEG pattern and seizures using this terminology,[9] the relationship between EEG patterns categorized by this terminology and SE outcome has not been evaluated. Herein, we describe EEG patterns recorded during the first 24 h of cEEG in a prospectively collected cohort of adult patients with SE and their association with outcome.

**Key Points**

- Rhythmic or periodic patterns (RPPs) on continuous electroencephalography (cEEG) are present in more than half the patients after status epilepticus.
- Sixty-eight percent of cEEG recordings show definite seizures, with half of seizures being purely electrographic.
- EEG background provides independent information regarding outcome after adjustment for relevant clinical findings, as compared to RPPs or seizures.
- The absence of a posterior dominant rhythm is associated with a greater likelihood of mortality
- Normal stage II sleep patterns are associated with the likelihood of complete recovery.

## Methods

### Primary research question

The primary research question was to evaluate cEEG yield in outcome prediction after SE.

### Standard protocol approvals, registrations, and patient consents

The institutional review boards of each center approved this study. Because this observational study involved no risk for patients and focused on the acute phase in critically ill patients, consent was waived.

### Cohort and SE definition

This observational cohort included all consecutive adult patients (>16 years of age) with SE of all etiologies (with the exception of postanoxic SE) admitted to three university tertiary care centers in Boston, Massachusetts, U.S.A., from June 1, 2013 at the Brigham and Women's Hospital and the Massachusetts General Hospital; and from November 1, 2013 at the Beth Israel Deaconess Medical Center, all through March 31, 2014. Subjects were screened through daily review of all EEG studies ordered during that period; all patients with suspected SE at each institution had continuous EEG within 24 h as part of routine clinical care. SE was defined as the occurrence of an ongoing epileptic seizure or repeated epileptic seizures, without full recovery between seizures for >5 min.[1] EEG diagnosis was required for nonconvulsive SE in accordance with recently published criteria.[10]

### Definition of variables

The STatus Epilepticus Severity Score (STESS) was calculated for each patient using age (<65 years = 0 pt; ≥65 years = 2 pts), seizure type (simple-partial, complex-partial, absence, and myoclonic in the context of idiopathic/ genetic epilepsy = 0 pt; generalized-convulsive = 1 pt; nonconvulsive SE in coma = 2 pts), level of consciousness (alert, somnolent or confused = 0 pt; stuporous or comatose = 1 pt), and history of previous seizures (yes = 0 pt; no = 1 pt), with a total score between 0 and 6 points.[11] This score is a tool designed to predict mortality and has been externally validated on an independent cohort.[12] The onset time of SE was determined as precisely as possible from prehospital chart and emergency department summaries. The time last seen well was considered the beginning of SE for episodes without clear times of onset (e.g., unwitnessed or subtle nonconvulsive SE). Etiology was determined based on clinical chart review and classified as potentially fatal if not specifically treated (or not) as previously described in other studies[13,14] including: acute (<7 days) large vessel ischemic stroke, acute cerebral hemorrhage, acute central nervous system infection, severe systemic infection, malignant brain tumor, acquired immunodeficiency syndrome (AIDS) with central nervous system (CNS) complications, chronic renal insufficiency requiring dialysis, systemic vasculitis, metabolic disturbance or acute intoxication sufficient to cause coma in the absence of SE, eclampsia, and intracranial tumor surgery.

The presence or absence of structural brain lesions was assessed using available imaging information and categorized as: no lesion, or remote or acute structural lesion. Clinical outcome at hospital discharge was categorized as return to premorbid clinical state, new morbidity, or death.

### cEEG recordings and data classification

Recordings were acquired using the international 10–20 system with 21 electrodes (XLTEK; Natus Medical Incorporated, San Carlos, CA, U.S.A.). Filters were set at 0.5 and 70 Hz, and

a notch filter was used as needed. All EEG recordings were reviewed by trained electroencephalographers using the 2012 ACNS Critical Care EEG terminology. One author certified for ACNS Standardized Critical Care EEG Terminology (V.A.) prospectively reviewed all EEG tracings to ensure accurate classification. Missing information was obtained during this secondary review. For the rhythmic and periodic patterns RPPs, “Main term 1” (lateralized, generalized, multifocal, bilateral independent) and “Main term 2” (periodic discharges [PDs], rhythmic delta activity [RDA], spike-and-wave [SW], or no periodic pattern) were recorded. Modifiers regarding their prevalence, duration, frequency, and sharpness were recorded as ordered categorical variables. The “plus modifiers” were also collected when applicable.[7] For background activity, data regarding symmetry, frequency, and continuity were collected as ordered categorical variables. The posterior dominant rhythm and its reactivity were collected as categorical variables: present or absent. The presence of stage II sleep was also recorded as ordered categorical variables (absent, present but abnormal, or present and normal). Stage II sleep patterns were considered as normal if K complexes and spindles were present, symmetric and synchronous; otherwise it was considered as present and abnormal or absent. Presence of definite seizures and their frequency (number of seizures/h) were also assessed. Each patient was categorized as to the presence of electrographic-only seizures or not. For nonconvulsive seizures, the Young criteria were used.[15]

### Statistical analysis

Data were analyzed using Stata 13 (StataCorp, College Station, TX, U.S.A.). Patients with different types of RPPs were compared using chi-square, Fisher’s exact, and Kruskal-Wallis tests, as appropriate. Univariate analyses for clinical measures, etiology, and cEEG features (RPPs, seizures and background) were conducted for the two outcomes of interest (mortality and complete clinical recovery). Variables with a p-value $\leq 0.05$ were entered into a stepwise backward logistic regression model (significance level for removal from the model: $p < 0.05$). Model goodness-of-fit was evaluated with the Hosmer-Lemeshow chi-square. Model performance was assessed through receiver operating characteristic (ROC) curve analysis.

## Results

During the study period, 120 adult patients undergoing cEEG for SE met criteria for inclusion in the study. The mean age was 58 years (standard deviation [SD] 16.65). Fifty-six patients were male (46.7%); 62 (51.6%) had premorbid seizures. The median STESS value was 2 (range 0–6), and 50% of patients had a potentially fatal etiology.

cEEG recordings were obtained at a median time of 14.4 h (range 0–143 h) after commencement of SE. Twenty-two SE episodes (18.3%) started while the cEEG was being acquired. Table 1 summarizes the clinical findings for patients with different RPP types. Although 49 patients (40.8%) did not show any periodic pattern, 45 (37.5%) had PD, 20 (16.7%) RDA, and 6 (5.0%) SW. The type and presence of a periodic pattern were differentially distributed depending on age. Patients without RPPs were younger (mean 51.0 years old) than patients with PD (61.0 years), RDA (66.3 years), or SW (63.5 years) ($p < 0.001$). Patients with SW and without RPPs had a potentially fatal etiology less frequently (p

= 0.043). Approximately half of patients without RPPs and half of those with SW did not have any structural lesion, whereas the majority of PD and RDA were associated with a remote or acute structural brain lesion (p = 0.009). Of note 116/120 patients (96.7%) had brain imaging; 111 patients had computed tomography (CT), 60 had magnetic resonance imaging (MRI), and 55 patients had two imaging modalities. All four patients without imaging had an SE in the setting of a known epilepsy (three with AED-related events and one without any provocative factor) and were not considered in this analysis.

Seizures were less frequent in patients without RPPs, but the clinical nature of seizure type (clinical or purely electrographic) was the same across the four groups.

RPP characteristics are shown in Table 2. Although 73.4% of PD were abundant or continuous, and SW were mostly continuous, 55% of RDA were frequent or occasional (p = 0.003). The duration of the pattern (when non-continuous) also differed, with PD being more prolonged than RDA of SW (p = 0.006). The RPP frequency was also lower in the PD group than in the RDA or SW groups (p < 0.001, Kruskal-Wallis). The descriptions of the sharpness and the “plus modifiers” are shown, but their comparisons were not performed because these terms do not apply uniformly to all RPP. Of note, only 2 (4.7%) of 45 of the PD had triphasic morphology.

EEG features, clinical information, and etiology of survivors and nonsurvivors are compared in Table 3. RPPs were more frequently absent in the survivors (43.1% vs. 33.0%), and generalized rhythmic delta activity (GRDA) was more frequent in the nonsurvivors (27.8% vs. 2.0%) (p = 0.002). A background with a posterior dominant rhythm (PDR) was present in nearly half of the survivors but in only 5.6% of patients who died (p = 0.001). The background frequency was also slower in the nonsurvivors, with more patients with delta activity and fewer patients with alpha activity than in the survivors (p = 0.01). Clear background reactivity was seen twice as often in the survivors (p = 0.018). All other EEG features regarding RPP modifiers, seizures, and background (including background symmetry, voltage, and continuity; not shown in Table 3) were similar between both groups.

The STESS was significantly higher in nonsurvivors: median of 4 (range 1–6) versus 2 (0–6, p = 0.0018). Non-survivors were also twice as likely to have a potentially fatal etiology (p = 0.004, Fisher). In multivariate analysis, only the absent of a PDR (OR 9.8; 95% confidence interval [CI] 1.2–79.8; p = 0.033) and the STESS (OR 1.48/point; 95% CI 1.06–2.05; p = 0.02) remained associated with mortality (model goodness-of-fit: p = 0.72, $\chi^2$). The area under the ROC curve using the PDR and the STESS to predict mortality was 0.79 (95% CI 0.695–0.894).

Patients with full recovery at hospital discharge and those presenting new morbidity (including nonsurvivors) are compared in Table 4. Patients with good recovery had RPPs less frequently (32.1% vs. 61.5%, p = 0.007) and had less frequent seizures (53.9% vs. 75.3%, p = 0.022). Nevertheless, when present, seizure types and frequency were similar in both groups. A PDR was also seen more frequently in patients with favorable outcomes. Stage II sleep patterns were present and normal in more than half of patients with complete recovery, but in only 13.6% of patients with new morbidity or death (p < 0.001). All other

EEG features regarding RPPs, seizures, or background (including background symmetry, voltage, and continuity; not shown in Table 4) were similar. STESS was lower in patients with good outcome: median of 2 (range 0–6) versus 3 (0–6, $p < 0.001$). Those patients also had a potentially fatal etiology less frequently. In the multivariate logistic regression model, STESS (OR 0.51/point; $p = 0.005$; 95% CI 0.32–0.82) and the presence of a potentially fatal etiology (OR 0.12; $p = 0.001$; 95% CI 0.03–0.4) remained significant. The only EEG feature that remained significantly associated with complete clinical recovery was stage II sleep pattern characteristics (OR 2.59/each step up: from absent to present but abnormal or from present but abnormal to normal; $p = 0.002$; 95% CI 1.4–4.77) (model goodness-of-fit: $p = 0.85$, $\chi^2$). The area under the ROC curve using the STESS, the presence of potentially fatal etiology, and sleep II stage categories is 0.88 (95% CI 0.82–0.94).

## Discussion

The main finding of this study is that among the cEEG characteristics considered, background activity and sleep features appear to be better clinical outcome prediction factors than RPPs and seizure features, which lost their predictive value after adjustment for other known predictors. Regression analysis, including cEEG, clinical information, and etiology found that the absence of a PDR was the only EEG feature associated with mortality. When absent, it increased markedly the likelihood of mortality, with an odds ratio of 9.8. Similar findings were shown for good outcome prediction. Stage II sleep characteristics were the only significant predictor of an increased likelihood of complete recovery, with an OR of 2.59 for each step up in the following categories: absent, present and abnormal, or present and normal. Both mortality and complete recovery models resulted in very good goodness-of-fit and prediction performance, supporting these findings.

EEG oscillations, including the posterior dominant rhythm[16] and the sleep spindles[17] are caused by intact connections between the neocortex and the thalamus, also known as the thalamocortical system. Moreover a recently published study[18] suggests that EEG background organization and sleep architecture correlate with preserved cognition assessed with functional MRI and brain metabolism assessed with positron emission tomography in patients with disorders of consciousness. Those findings associated with the present study suggest that the presence of a posterior dominant rhythm and sleep spindles may be clinical surrogates of the thalamocortical system integrity and confirm EEG as a valid biomarker in the epilepsy field.[19]

Reports assessing the role of EEG in outcome prediction in SE are few, but some have reported similar findings. A study evaluating periodic lateralized epileptiform discharges (PLEDs) (now labeled as LPD according to the 2012 ACNS terminology) in 62 prospectively collected SE episodes also found that outcome was more closely related to age and etiology than to the ictal EEG pattern.[6] Although abnormalities on EEG 1 h after treatment predicted seizure recurrence in another prospective study of 70 cases of SE,[20] they were not associated with mortality. In a retrospective study of the prognostic implication of different types of periodic epileptiform discharges (PEDs),[21] within the same PED pattern, the likelihood of death was decreased in patients with chronic etiologies (as opposed to acute etiologies) after regression analysis. Moreover, no statistical association was found between

outcomes and several EEG features including RPP amplitude, inter-PD interval, and duration of epileptiform complexes.

Others have found that RPPs or seizures may be useful in outcome prediction. Jaitly et al. found that “after SE ictal discharges” the “presence of a burst-suppression pattern” and, to a lesser extent, LPDs (formerly called PLEDs) were associated with increased morbidity and mortality after adjustment for etiology in a prospectively collected cohort of 180 patients with SE. In that study,[22] however, brain anoxia represented 20% of the cohort; anoxic patients were excluded from our study. Because postanoxic SE after cardiac arrest usually carries a dismal prognosis and is frequently associated with RPPs,[23] this might explain the difference in findings. In addition, in that study, EEG was categorized into seven different patterns and not assessed using the detailed classification used in this study[7] that allowed evaluation of each different feature of the EEG. PDs were the only EEG feature associated with outcome in a retrospective evaluation of 50 SE cases,[5] with 44% of patients with PDs having a poor outcome versus 19% in the group without PDs. Still, these results were based on a retrospective univariate analysis without adjustment for etiology or clinical findings. In our analysis, RPPs were also significant predictors for both good outcome and mortality in univariate analysis, but this association was lost after adjustment for etiology and STESS. Moreover, the study by Nei et al.[5] focused on ictal or PDs without assessment of the EEG background. There are also divergent results regarding the value of RPPs in outcome prediction in different patient populations. No association was found between PD and mortality in comatose patients,[24] even when PDs were prolonged,[25] but the presence of PDs was a strong outcome predictor in the setting of CNS infection[26] or intracerebral hemorrhage.[27] The fact that PDs are associated with outcome in cohorts with specific brain pathology, such as CNS infection or intracerebral hemorrhage, and not when assessing comatose patients in general or in patients with SE from all etiologies, reinforces the importance of the underlying etiology in this setting.

Among patients with RPPs, PDs were very frequent (two thirds of patients), followed by RDA and SW. Of interest, the same rate of seizure occurrence (~80%) was found in the PD, RDA, and SW groups, but in only 51% when RPPs were absent. The similarity of seizure occurrence in the LPD and lateralized rhythmic delta activity (LRDA) groups has also been described in critically ill patients.[9] Indeed, both EEG patterns were associated with a rate of seizure occurrence of 60% in that cohort. That we included patients in SE only, while and Gaspard et al. included unselected patients undergoing cEEG, may explain the higher rate of seizure occurrence in our cohort. Half of patients with SW and those without any RPP did not have structural brain lesions, as opposed to patients with PD or RDA, among whom 86% and 85%, respectively, had structural lesions. This reinforces the association of PDs with structural lesions[28] and suggests a more nonstructural etiology to the spike-and-wave pattern, as seen in the genetic generalized epilepsies.[29]

This study has several strengths. First, it includes data from 120 patients with SE studied prospectively using the new American Clinical Neurophysiology Society’s (ACNS) Standardized Critical Care EEG Terminology,[7] which has been demonstrated to be suitable for research on the clinical significance of critical care EEG features,[8] as opposed to previous studies which used less-uniform EEG terminology.[5,6,20–22] Moreover, the yield of

cEEG in outcome prediction was adjusted for known important clinical factors, further confirming the robustness of the STESS for outcome prediction.[11,12]

This study also has some limitations. First, we limited our analysis to the first 24 h of cEEG and we could not exclude the possibility that later assessment or pattern evolution might have led to different findings and increased the yield of some other EEG features in outcome prediction. Nevertheless, the prognostic value of SE duration is largely lost after 10 h,[30] and early EEG evaluation has been shown to be as reliable as later assessment for outcome prediction in a different clinical setting.[31] In addition, the outcome prediction value of some RPP modifiers (sharpness and "plus modifiers") was not evaluated. Still, these particular terms cannot be applied to all RPPs, and the relatively poor interrater agreement reported[8] may lessen the predictive value of the "plus modifier." Furthermore, stepwise backward logistic regression model was employed in the present study to identify variables with independent prediction value, a technique that some consider suboptimal.[32,33] However, a stepwise regression model in logistic regression is an efficient and widely used way to analyze the effect of a group of independent variables on a binary outcome.[34,35] Careful variable selection was made (know predictors, statistically significant variables in the univariate analysis) and necessary assumptions for regression[34] were checked (absence of colinearity, numbers of variable) to minimize the potential limitations of this procedure. Finally, this study is based on cEEG data, which is not routinely available in every center that manages SE.

## Conclusion

This study is the first report of the predictive value of EEG features in cEEG monitoring for outcome prediction in SE since the publication of the 2012 ACNS report on Standardized Critical Care EEG Terminology. It provides class III evidence that when adjusted for clinical predictors, EEG background information gives independent information on outcome prediction as opposed to rhythmic or periodic patterns or seizure characteristics. Indeed, the absence of a posterior dominant rhythm is associated with a greater likelihood of mortality. Stage II sleep patterns are associated with the likelihood of complete recovery. Although STESS and etiology are confirmed as robust predictors, RPPs and seizures are not predictive of SE outcome after statistical adjustment. These findings need to be confirmed but may be important for EEG interpretation and daily clinical practice for critically ill patients in SE.

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## Biography

**Dr. Vincent Alvarez** is a neurologist/epileptologist at the Valais Hospital and University Hospital in Lausanne and a visiting scientist at Brigham and Women's Hospital.

**Table 1**

Clinical and seizures characteristics based on rhythmic and periodic patterns

| | Total | No RPP | PD | RDA | SW | p-Value (test) |
|---|---|---|---|---|---|---|
| n | 120 | 49 (40.8%)[a] | 45 (37.5%)[a] | 20 (16.67%)[a] | 6 (5%)[a] | |
| Age (mean, SD) | 58.18 (±16.65) | 51 (±14.5)[b] | 61 (±16.43)[b] | 66.25 (±15.33)[b] | 63.5 (±20.8) | **<0.001** (ANOVA) |
| Male gender | 56 (46.7%) | 22 (44.9%) | 19 (42.2%) | 12 (60%) | 3 (50%) | 0.62 (Fisher) |
| Premorbid seizures | 62 (51.6%) | 27 (57.1%) | 23 (51.11%) | 7 (35%) | 5 (83.3%) | 0.19 (Fisher) |
| STESS (median, range) | 2 (0–6) | 2 (0–5) | 2 (0–6) | 3 (0–6) | 2.5 (0–6) | 0.45 (Kruskal-Wallis) |
| Potentially fatal etiology | 62 (52.1%) | 21 (42.86%) | 26 (59.09%) | 14 (70%) | 1 (16.67%) | **0.043** ($\chi^2$) |
| Structural lesion (116 with brain imaging) | | | | | | |
| No lesion | 33 (28.45%) | 22 (47.83%) | 6 (13.33%) | 3 (15%) | 2 (40%) | |
| Acute | 40 (34.48%) | 12 (26.09%) | 17 (37.78%) | 10 (50%) | 1 (20%) | |
| Remote | 43 (37.07%) | 12 (26.09%) | 22 (48.89%) | 7 (35%) | 2 (40%) | **0.009** ($\chi^2$) |
| Definite seizure | | | | | | |
| Presence of definite seizure | 82 (68.33%) | 25 (51.02%) | 36 (80%) | 16 (80%) | 5 (83.33%) | **0.01** (Fisher) |
| With clinical manifestations | 42 (51.22%) | 15 (60%) | 14 (38.89%) | 10 (62.5%) | 3 (60%) | |
| Electrographic seizure only | 40 (48.78%) | 10 (40%) | 22 (61.11%) | 6 (37.5%) | 2 (40%) | 0.27 ($\chi^2$) |

In bold: statistically significant.

RPP, rhythmic and periodic patterns; PD, periodic discharges; RDA, rhythmic delta activity; SD, standard deviation; STESS, STatus Epilepticus Severity Score; SW, spike-and-wave.

[a]Row percentage.

[b]Column percentage.

**Table 2**

Rhythmic and periodic pattern characteristics according to their modifiers

| | Total | PD | RDA | SW | p-Value (test) |
|---|---|---|---|---|---|
| Periodic and rhythmic pattern details | 71 | 45 (63.38%)[a] | 20 (28.16%)[a] | 6 (8.45%)[a] | |
| Main term | | | | | |
| Lateralized | 51 (71.83%)[b] | 36 (80%)[b] | 13 (65%)[b] | 2 (33.3%)[b] | |
| Generalized | 18 (25.35%) | 7 (15.6%) | 7 (35%) | 4 (66.7%) | |
| Multifocal | 0 | 0 | 0 | 0 | |
| Bilateral independent | 2 (2.82%) | 2 (4.4%) | 0 | 0 | 0.051 ($\chi^2$) |
| Prevalence | | | | | |
| Continuous (>90%) | 29 (40.85%) | 21 (46.67%) | 4 (20%) | 4 (66.7%) | |
| Abundant (50–89%) | 17 (23.94%) | 12 (26.67%) | 5 (25%) | 0 | |
| Frequent (10–49%) | 17 (23.93%) | 10 (22.2%) | 6 (30%) | 1 (16.67%) | |
| Occasional (1–9%) | 7 (9.86%) | 2 (4.44%) | 5 (25%) | 0 | |
| Rare | 1 (1.41%) | 0 | 0 | 1 (16.67%) | **0.003** ($\chi^2$) |
| Duration (for noncontinuous RPPs) | 42 | 24 | 16 | 2 | |
| Very long (>1 h) | 4 (9.09%) | 3 (12.5%) | 0 | 0 | |
| Long (5–59 min) | 13 (29.55%) | 10 (41.67%) | 3 (18.75%) | 0 | |
| Intermediate (1–4.9 min) | 11 (25%) | 7 (29.17%) | 4 (25%) | 0 | |
| Brief (10–59 s) | 9 (20.45%) | 4 (16.67%) | 5 (31.25%) | 0 | |
| Very brief (<10 s) | 7 (15.91%) | 0 | 4 (25%) | 2 (100%) | **0.006** ($\chi^2$) |
| Frequency (median, range) | 1 (<0.5–3.5) | 1 (<0.5–3) | 2 (0.5–3.5) | 2.75 (1.5–3.5) | **<0.001** (Kruskal-Wallis) |
| Sharpness (for PD and SW only) | 51 | | | | |
| Spiky | 17 (33.3%) | 11 (24.4%) | – | 6 (100%) | |
| Sharp | 33 (64.71%) | 33 (73.3%) | – | 0 | |
| Sharply contoured | 0 | 0 | – | 0 | |
| Blunt | 1 (1.96%) | 1 (2.2%) | – | 0 | Not assessed |
| “Plus modifiers” (for PD and RDA only) | 65 | | | | |
| None | 39 (60%) | 28 (62.22%) | 11 (55%) | – | |
| Fast activity | 12 (18.46%) | 12 (26.67%) | 0 | – | |
| Rhythmic activity (for PD only) | 4 (6.15%) | 4 (8.89%) | – | – | |

| | Total | PD | RDA | SW | p-Value (test) |
|---|---|---|---|---|---|
| Sharp component (for RDA only) | 9 (13.85%) | – | 9 (45%) | – | |
| Fast and rhythmic activity (for PD) | 1 (1.54%) | 1 (2.22%) | – | – | |
| Fast activity and sharp (for RDA) | 0 | – | 0 | – | Not assessed |
| Triph. Morph. (for PD only) | | 2 (4.65%) | – | – | |

In bold: statistically significant.

PD, periodic discharge; RDA, rhythmic delta activity; SD, standard deviation; STESS, STatus Epilepticus Severity Score; SW, spike-and-wave; Triph. Morph, tri-phasic morphology.

[a]Row percentage.

[b]Column percentage.

**Table 3**

cEEG features according to the 2012 ACNS Standardized Critical Care EEG Terminology, clinical characteristics, and etiology, and their association with mortality (univariate analysis and after logistic regression)

| | Univariate analysis | | | Multivariate analysis | | |
|---|---|---|---|---|---|---|
| | **Non-survivors** | **Survivors** | **p-value (test)** | **OR** | **95% CI** | **p** |
| EEG | n = 18 | n = 102 | | | | |
| Rhythmic and periodic patterns | | | | | | |
| Main term | | | | | | |
| No rhythmic and periodic pattern | 6 (33.3%)[a] | 44 (43.14%)[a] | | | | |
| LPD (incl. BiPD) | 5 (25.78%) | 33 (32.35%) | | | | |
| GPD | 1 (5.56%) | 5 (4.9%) | | | | |
| LRDA | 0 | 13 (12.75%) | | | | |
| GRDA | 5 (27.78%) | 2 (1.96%) | | | | |
| LSW | 0 | 2 (1.96%) | | | | |
| GSW | 1 (1.56%) | 3 (2.94%) | 0.002[b] ($\chi^2$) | NS | | |
| Prevalence | n = 12 | n = 59 | | | | |
| Continuous (>90%) | 5 (41.67%) | 24 (40.68%) | | | | |
| Abundant (50–89%) | 4 (33.33%) | 13 (22.03%) | | | | |
| Frequent (10–49%) | 2 (16.67%) | 15 (25.42%) | | | | |
| Occasional (1–9%) | 1 (8.33%) | 6 (10.17%) | | | | |
| Rare | 0 | 1 (1.69%) | 0.87 ($\chi^2$) | – | | |
| Frequency (median, range) | 1.25 (0.5–3) | 1 (<0.5–3.5) | 0.33 (Wilcoxon) | | | |
| Seizures | | | | | | |
| Presence of seizure | 13 (72.22%) | 69 (67.65%) | 0.79 (Fisher) | – | | |
| Seizure type | | | | | | |
| With clinical manifestations | 7 (53.85%) | 35 (57.72%) | | | | |
| Electrographic seizures only | 6 (46.15%) | 34 (49.28%) | 1 (Fisher) | – | | |
| Number of seizures/h (median, range) | 2.5 (0.04–30) | 2.43 (0.04–50) | 0.51 (Wilcoxon) | – | | |
| Background | | | | | | |
| PDR | | | | | | |
| Absent | 17 (94.45%) | 55 (53.92%) | 0.001[b] ($\chi^2$) | 9.8 | 1.2–79.8 | 0.033 |

| | Univariate analysis | | | Multivariate analysis | | |
|---|---|---|---|---|---|---|
| | **Non-survivors** | **Survivors** | **p-value (test)** | **OR** | **95% CI** | **p** |
| Frequency | | | | | | |
| Alpha or more | 2 (11.1%) | 41 (40.2%) | | | | |
| Theta | 8 (44.4%) | 44 (43.14%) | | | | |
| Delta | 8 (44.4%) | 17 (16.67%) | 0.01[b] (Fisher) | NS | | |
| Stage II sleep transients | | | | | | |
| Present | 2 (11.11%) | 31 (30.39%) | | | | |
| Present abnormal | 0 | 7 (6.86%) | | | | |
| Absent | 16 (88.89%) | 64 (62.75%) | 0.113 (Fisher) | – | | |
| Reactivity | | | | | | |
| Clear background reactivity | 6 (33.3%) | 66 (64.71%) | 0.018[b] (Fisher) | NS | | |
| STESS (median, range) | 4 (1–6) | 2 (0–6) | 0.0018[b] (Wilcoxon) | 1.48 | 1.06–2.05 | 0.02 |
| Potentially fatal etiology (n,%) | 15 (83.33%) | 47 (46.53%) | 0.004[b] (Fisher) | NS | | |

BiPD, bilateral independent periodic discharge; CI, confidence interval; GPD, generalized periodic discharge; GRDA, generalized rhythmic delta activity; GSW, generalized spike-and-wave; LPD, lateralized periodic discharge; LRDA, lateralized rhythmic delta activity; LSW, lateralized spike-and-wave; NS, nonsignificant; OR, odds ratio; STESS, STatus Epilepticus Severity Score; uV, micro volts.

[a] Column percentage.

[b] Used in regression model because $p < 0.05$ in univariated analysis.

**Table 4**

cEEG features according to the 2012 ACNS Standardized Critical Care EEG Terminology and clinical characteristics and etiology, and their association with a complete clinical recovery at hospital discharges (univariate analysis and after logistic regression)

| | Univariate analysis | | | Multivariate analysis | | |
|---|---|---|---|---|---|---|
| | **Complete recovery** | **New morbidity or death** | **p-value (test)** | **OR** | **95% CI** | **p** |
| EEG | n = 39[a] | n = 81[a] | | | | |
| Rhythmic and periodic patterns | | | | | | |
| Main term | | | | | | |
| No rhythmic or periodic pattern | 24 (61.54%) | 26 (32.1%) | | | | |
| LPD (incl. BiPD) | 7 (17.95%) | 31 (38.27%) | | | | |
| GPD | 2 (5.13%) | 4 (4.94%) | | | | |
| LRDA | 2 (5.13%) | 11 (13.58%) | | | | |
| GRDA | 0 | 7 (8.64%) | | | | |
| LSW | 1 (2.56%) | 1 (1.23%) | | | | |
| GSW | 3 (7.69%) | 1 (1.23%) | 0.007[b] ($\chi^2$) | NS | | |
| Prevalence | n = 15 | n = 56 | | | | |
| Continuous (>90%) | 6 (40%) | 23 (42.07%) | | | | |
| Abundant (50–89%) | 4 (26.67%) | 13 (23.21%) | | | | |
| Frequent (10–49%) | 3 (20%) | 14 (25%) | | | | |
| Occasional (1–9%) | 1 (6.67%) | 6 (10.71%) | | | | |
| Rare | 1 (6.67%) | 0 | 0.39 ($\chi^2$) | – | | |
| Frequency (median, range) | 0.5 (<0.5–3.5) | 1 (<0.5–3.5) | 0.47 (Wilcoxon) | – | | |
| Seizures | | | | | | |
| Presence of seizure | 21 (53.85%) | 61 (75.31%) | 0.022[b] (Fisher) | NS | | |
| Seizure type | | | | | | |
| With clinical manifestations | 13 (61.9%) | 29 (47.54%) | | | | |
| Electrographic szs only | 8 (38.1%) | 32 (53.46%) | 0.31 (Fisher) | – | | |
| Number of seizures/h (median, range) | 1.125 (0.04–20) | 2.75 (0.04–50) | 0.29 (Wilcoxon) | – | | |
| Background | | | | | | |
| PDR | | | | | | |
| Absent | 14 (35.9%) | 58 (71.6%) | <0.001[b] (Fisher) | NS | | |

| | Univariate analysis | | | Multivariate analysis | | |
|---|---|---|---|---|---|---|
| | **Complete recovery** | **New morbidity or death** | **p-value (test)** | **OR** | **95% CI** | **p** |
| Frequency | | | | | | |
| Alpha or more | 19 (48.72%) | 24 (29.63%) | | | | |
| Theta | 15 (38.46%) | 37 (45.68%) | | | | |
| Delta | 5 (12.82%) | 20 (24.69%) | 0.096 (Fisher) | – | | |
| Stage II sleep transients | | | | | | |
| Present | 22 (56.41%) | 11 (13.58%) | | | | |
| Present abnormal | 1 (2.56%) | 6 (7.41%) | | | | |
| Absent | 16 (41.13%) | 64 (79.1%) | <0.001[b] (Fisher) | 2.59 | 1.4–4.77 | 0.002 |
| Reactivity | | | | | | |
| Clear background reactivity | 28 (71.79%) | 44 (54.32%) | 0.076 (Fisher) | – | | |
| STESS (n, %) | 2 (0–3) | 3 (0–6) | <0.001[b] (Wilcoxon) | 0.51 | 0.32–0.82 | 0.005 |
| Potentially fatal etiology | 7 (17.95%) | 55 (68.75%) | <0.001[b] (Fisher) | 0.12 | 0.03–0.4 | 0.001 |

BiPD, bilateral independent periodic discharge; CI, confidence intervals; GPD, generalized periodic discharge; GRDA, generalized rhythmic delta activity; GSW, generalized spike-and-wave; LPD, lateralized periodic discharge; LRDA, lateralized rhythmic delta activity; LSW, lateralized spike-and-wave; NS, non-significant; OR, odds ratio; STESS, STatus Epilepticus Severity Score; uV, micro volts.

[a]Column percentage.

[b]Used in regression model because $p < 0.05$ in univariate analysis.