Association of Time to Continuous EEG Initiation With Outcomes in Critically Ill Patients

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Purpose: Continuous electroencephalography (cEEG) is used in the critical care setting for seizure detection and treatment, sedation management, and ischemia detection. Further evidence is needed to support whether early cEEG use can improve outcomes. We examined whether time from admission to cEEG initiation affects outcomes.

Methods: This is a single-center cohort study of critically ill adults (age $>$ 18 years) who underwent cEEG monitoring within 7 days of admission from January to December 2019. Patients with anoxic brain injury were excluded. Time (hours) from admission to cEEG was recorded. Outcomes were in-hospital mortality and poor discharge modified Rankin Score (4–6). Results are reported as median [quartile range] and odds ratio (OR) [confidence intervals, CI].

Results: In total, 464 patients met eligibility. Median time to cEEG was 23 hours [13, 52]. On multivariable analysis, increasing time to cEEG was associated with discharge mortality (OR, 1.006 [CI, 1.0002–1.013], 0.1%/hour [CI, 0.02–0.2]) and poor outcome (OR, 1.013 [CI, 1.005–1.020], 0.2%/hour [CI, 0.07–0.3]). Median time to cEEG initiation in patients with clinical concern for seizures/status at presentation ($n = 121$) was 12 hours [6, 17] and in patients without clinical concern for seizures at presentation ($n = 343$) was 31 hours [18, 66]. In patients without clinical concern for seizures/status epilepticus at presentation, time to cEEG continued to be associated with mortality (OR, 1.007 [CI, 1.001–1.014)] and poor outcome (OR, 1.012 [CI, 1.003–1.021]).

Conclusions: Increasing time to cEEG initiation was associated with higher mortality and worse outcomes. We hypothesize earlier cEEG results in timely interventions including treatment escalation and de-escalation that may improve outcomes.

Key Words: Continuous EEG, Seizures, Critical care, Health services, Outcomes.

(J Clin Neurophysiol 2026;43: 17–22)

Continuous electroencephalography (cEEG) is used in the critical care setting for diagnosis and treatment of seizures.[1] Higher quantitative burden of seizures and other epileptiform abnormalities including periodic and rhythmic patterns independently increase the probability of mortality and worse functional outcomes in critically ill patients.[2–5] Other indications for cEEG include ischemia detection, management of sedation and prognostication.[1] National studies have shown that cEEG utilization is associated with lower in-hospital mortality in critically ill patients.[6–8] We hypothesize this may potentially be mediated by cEEG-guided treatment decisions (e.g., treatment of seizures, discontinuation of antiseizure medications or anesthetics when not indicated, and early detection of ischemia in at-risk patients).[8,9] However, further evidence is needed to support the utility of cEEG use and cEEG-guided treatment decisions in improving outcomes. This is particularly important because cEEG is resource intensive and access to cEEG is not universal, particularly in low-resource settings.[6–8] Here we sought to determine whether increasing time from admission to initiation of cEEG is associated with in-hospital mortality and discharge functional outcomes as measured by the modified Rankin Scale.

S. F. Zafar is a clinical neurophysiologist for Corticare, received speaking honoraria from Marinus, and received royalties from Springer publishing, unrelated to this work. M. B. Westover is cofounder of Beacon Biosignals unrelated to this work.

The data that support the findings of this study are available from the corresponding author, (S.F.Z.) on reasonable request.

This work was supported by the National Institutes of Health K23NS114201 (PI: S.F.Z.).

This work was previously presented as a poster and platform presentation at the American Epilepsy Society Conference, 2022.

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ISSN: 1537-1603/25/4301-0017
DOI 10.1097/WNP.0000000000001161

METHODS

We conducted an observational cohort study of critically ill patients (age $>$ 18 years) who underwent cEEG monitoring at a single center from January to December 2019. The study was approved by Massachusetts General Brigham Institutional Review Board. Informed consent was waived for this observational study. We included patients who underwent cEEG monitoring within 7 days of admission, because this is a high-risk period for acute symptomatic seizures.[10] We excluded patients with anoxic brain injury given their distinct prognostic profile. Disease severity was measured using the Sequential Organ Failure Assessment (SOFA) Score. Continuous electroencephalography findings were documented according to American Clinical Neurophysiology Society nomenclature.[11] We defined seizures and rhythmic and periodic patterns (RPP) using the American Clinical Neurophysiology Society definition of epileptiform findings: seizures, lateralized periodic discharges, generalized periodic discharges, lateralized rhythmic delta activity, and generalized rhythmic delta activity. Generalized rhythmic delta activity (GRDA) is not significantly associated with seizures, ischemia or unfavorable outcome, as opposed to periodic discharges and lateralized rhythmic delta activity. Therefore, isolated GRDA may help in risk stratification and triaging decisions on duration of monitoring and treatment escalation or de-escalation. We extracted antiseizure medication data, including escalation of treatment (increase in the dose or number of medications) during cEEG monitoring. We recorded time from admission to cEEG initiation in hours. Outcome measures were discharge mortality and modified Rankin Scale.

Median, quartile range, and frequencies were calculated for descriptive statistics. Multivariable analysis was performed to determine the association between time to cEEG initiation and outcomes. We adjusted for variables likely to affect time to cEEG and/or outcomes. Results are presented as odds ratio (OR), 95% confidence intervals (CI).

RESULTS

Of 547 patients admitted to intensive care units that underwent cEEG monitoring, 464 met eligibility. Table 1 summarizes the clinical and demographic variables. The most common diagnosis was acute brain injuries (34%). Clinical seizures at hospital presentation occurred in 25% ($n = 119$) of patients. Electrographic seizures were seen in 10.8% of patients, and other RPPs (lateralized periodic discharges, generalized periodic discharges, lateralized rhythmic delta activity, generalized rhythmic delta activity) were seen in 58.6% of patients. Antiseizure medications (ASMs) were escalated during monitoring in 35% of patients.

The median time to cEEG monitoring was 23 hours [Q1, Q3: 13, 52]. To examine differences in baseline variables with increasing time to cEEG initiation, we divided time to cEEG into four categories roughly corresponding to the first to fourth quartiles on descriptive analysis. Most patients (>60%) presenting with clinical seizures or status epilepticus underwent cEEG monitoring within 12 hours. Patients who underwent cEEG within 12 hours also had higher SOFA scores (median score of 7 [Q1, Q2; 4, 9] vs. 3 [Q1, Q2: 2, 7] among those who underwent cEEG after 48 hours). Patients with longer time to cEEG initiation were monitored for longer (median duration of monitoring was 34 hours when cEEG was started within 12 vs. 49 hours when cEEG was started after 48 hours from admission). The frequency of seizures (average across the cohort 10.8%) and other RPP (average across cohort of 58.6%), and frequency of ASM escalation (35.3%) did not significantly change with increasing time to cEEG (see Table 1 for frequencies, *P*-value). Although the frequency of ASM and anesthetic de-escalation increased modestly with time, this finding was not significant (Table 1).

In patients with clinical suspicion for seizures/status epilepticus at presentation ($n = 121$), the median time to cEEG initiation was shorter at 12 hours [Q1, Q4: 4, 18], compared with 31 hours [Q1, Q4: 18, 66] in patients without clinical suspicion for seizures/status epilepticus at presentation ($n = 343$), $P < 0.0001$.

On multivariable analysis, after adjusting for age, sex, SOFA, etiology (primary neurologic vs. non-neurologic, acute brain injury, brain tumors), history of epilepsy, and seizures/status epilepticus at hospital presentation, increasing time from admission to cEEG was associated with discharge mortality (OR, 1.006 [1.0002–1.013]) and poor outcome (OR, 1.013 [1.005–1.020]). Figure 1 shows the probability of mortality and poor outcomes with increasing time to cEEG. Each increasing hour to cEEG monitoring increased the probability of mortality by 0.1% [CI, 0.02–0.2]. The probability of poor outcome increased by 0.2% [0.07–0.3] with each increasing hour to cEEG initiation.

Figure 2 shows differences in time to detection of electrographic seizures across discharge outcomes. The median time from admission to electrographic seizure detection was longer in patients deceased versus those alive at discharge, but not significant (48 [27, 113] vs. 27 [20, 52], $P = 0.180$). The median time to seizure detection was significantly longer in patients with poor outcomes (modified Rankin Scale 0–4) versus those with good outcome (39 hours [23, 84] vs. 20 [7, 46] $P = 0.022$). We also examined time to any seizure/RPP detection (because these patterns exist along a continuum, and many patients had both seizures and RPP (Fig. 3). Time to first detection of any seizure/RPP was significantly longer in patients deceased at discharge (40 [21, 92] vs. 29 [17, 56], $P = 0.027$). Time to first detection of any seizure/RPP was longer in patients with poor versus good outcomes, but did not reach significance (34 [17, 81] vs. 25 [17, 46], $P = 0.050$).

Subgroup Analysis

We performed a subgroup analysis in patients without clinical concern for seizures at presentation ($n = 343$). In the subgroup, the median time to cEEG initiation was 31 hours [Q1, Q4: 18, 66]. In patients without a clinical concern for seizures/status epilepticus, 31 (9%) had electrographic seizures and 207 (60%) had RPPs. In this subgroup, 78 (23%) had antiseizure treatment escalation during cEEG monitoring and 9 (3%) had treatment de-escalation. After adjusting for age, sex, SOFA, and etiology (primary neurologic vs. non-neurologic, acute brain injury, brain tumors), increasing time from admission to cEEG continued to be associated with discharge mortality (OR, 1.007 [CI, 1.001–1.014]) and poor outcome (OR, 1.012 [CI, 1.003–1.021]).

DISCUSSION

Increasing time to cEEG initiation is independently associated with increased risk of mortality and worse discharge functional outcomes. Based on our results, we hypothesize that increasing time to cEEG initiation can delay treatment interventions that, in turn, may worsen outcomes. These interventions include antiseizure treatment escalation, treatment de-escalation when not indicated, detection of new focal findings or ischemia, and associated interventions.

The frequency of detection of seizures and RPPs did not significantly change with increasing time to cEEG initiation. There are several potential hypothesis for these findings: (1) The median time to cEEG initiation was 12 hours in patients with clinical concern for seizures and 23 hours for the entire cohort, and, therefore, most patients received relatively early cEEG; (2)

TABLE 1. Clinical and Demographic Variables

		Time to EEG Initiation				
	Full Cohort $N = 464$	**≤12 hours** $N = 108$	**13–24 hours** $N = 131$	**25–48 hours** $N = 100$	**>48 hours** $N = 125$	***P***
Age, median (Q1, Q3)	64 (50, 74)	61 (46, 72)	66 (51, 77)	62 (52, 75)	64 (51, 73)	0.127
Sex, female N (%)	221 (47.6)	46 (42.6)	53 (40.5)	58 (58.0)	64 (51.2)	**0.033**
Primary diagnosis (%)*						
Epilepsy/breakthrough seizures	23 (5.0)	10 (9.2)	7 (5.3)	3 (3.0)	3 (2.4)	
Acute brain injury	161 (34.7)	22 (20.3)	53 (40.5)	45 (45.0)	41 (32.8)	
Ischemic stroke	36 (7.8)	2 (1.2)	13 (9.9)	11 (11)	10 (8)	0.096
Intracerebral hemorrhage	69 (14.9)	7 (6.4)	22 (16.9)	19 (1.09)	21 (16.8)	**0.001**
Subarachnoid hemorrhage	69 (14.9)	12 (11.1)	25 (19.1)	19 (19.0)	13 (10.4)	**0.032**
Traumatic brain injury	51 (11.0)	10 (9.3)	19 (14.5)	12 (12.0)	10 (8.0)	0.196
Brain tumor	52 (11.2)	9 (8.3)	8 (6.1)	16 (16.0)	19 (15.2)	**0.003**
Other neuro diagnosis	84 (17.9)	14 (13.0)	31 (23.4)	18 (18.0)	21 (16.8)	
Non-neuro diagnosis	137 (29.6)	36 (33.3)	27 (20.6)	24 (24.0)	50 (40)	
History of epilepsy (%)	55 (11.9)	24 (22.2)	17 (13.0)	8 (8.0)	6 (4.8)	0.000
History of brain injury (%)	111 (22.8)	29 (26.9)	38 (29.0)	18 (18.0)	26 (20.8)	0.175
SOFA score	6 (3, 8)	7 (4, 9)	7 (4, 9)	5 (2, 8)	3 (2, 7)	**0.000**
Clinical seizures at hospital presentation (%)	119 (25.6)	65 (60.2)	32 (24.4)	14 (14)	8 (6.4)	0.000
Clinical status epilepticus at hospital presentation (%)	50 (10.3)	34 (31.4)	12 (9.1)	4 (4.0)	0 (0.0)	0.000
Median time to EEG, hours (Q1, Q3)	23 (13, 52)					
Duration of monitoring, hours (Q1, Q3)	41 (24, 97)	34 (24, 94)	29 (22, 26)	41 (24, 97)	49 (28, 114)	0.015
EEG findings (%)						
Electrographic seizures	48 (10.8)	12 (11.1)	15 (11.5)	7 (7.0)	14 (11.2)	0.570
Electrographic status epilepticus	18 (3.9)	5 (4.6)	6 (4.6)	2 (2.0)	5 (4.0)	0.744
RPPs	272 (58.6)	57 (52.8)	81 (61.8)	56 (56.0)	78 (62.4)	0.404
Sporadic discharges	219 (47.2)	55 (50.9)	64 (48.1)	47 (47.0)	54 (43.2)	0.694
ASM prescribed during admission (%)	425 (91.6)	105 (97.2)	124 (94.6)	87 (87.0)	109 (87.2)	0.006
ASM escalation during EEG monitoring (%)	164 (35.3)	42 (38.9)	50 (38.2)	31 (31.0)	41 (32.8)	0.530
ASM and anesthetic de-escalation during EEG monitoring (%)	29 (6.3)	6 (5.4)	7 (5.4)	5 (5.1)	11 (8.9)	0.620
Length of stay, days median (Q1, Q2)	12 (7, 21)	9 (6, 19)	10 (7, 18)	12 (7, 22)	15 (9, 23)	0.000
Discharge mRS (%)						
0	4 (0.8)	3 (2.8)	1 (0.7)	0 (0.0)	0 (0.0)	0.002
1	7 (1.5)	4 (3.4)	1 (0.7)	0 (0.0)	2 (1.6)	
2	39 (8.4)	14 (13.0)	9 (6.9)	10 (10.0)	6 (4.8)	
3	64 (13.9)	18 (16.7)	18 (13.7)	17 (17.0)	11 (8.8)	
4	151 (32.5)	31 (28.7)	44 (33.6)	34 (34.0)	42 (33.6)	
5	91 (19.8)	13 (12.0)	30 (22.9)	18 (18.0)	31 (24.8)	
6	138 (23.1)	25 (23.1)	28 (21.4)	21 (21.0)	33 (26.4)	

*Not mutually exclusive.
ASM, antiseizure medication; mRS, modified Rankin Scale; RPP, rhythmic and periodic patterns; SOFA, sequential organ failure assessment.

within the time frame for cEEG initiation captured in our study, with increasing time to cEEG, while the likelihood of detecting seizures/RPP did not significantly change, the increasing time to detection and treatment decisions, particularly in those without a clinical concern for seizure, may have exposed patients to higher burden of seizures; (3) there may be patients who receive cEEG that do not require monitoring; (4) those with later initiation of cEEG and findings of seizures/RPP may have developed the electrographic findings as a secondary complication after admission (e.g., organ dysfunction, metabolic derangements, secondary brain injury, infections) or had higher illness severity that may have contributed to worse outcomes; (5) with increasing time to cEEG, the number of patients who had ASM and anesthetic de-escalation slightly increased, and though not significant, this supports the hypothesis that earlier cEEG initiation can guide early treatment de-escalation when not indicated, potentially minimizing exposure to the adverse effects of ASMs and anesthetics and (6) finally, early initiation of cEEG may also enable earlier recognition of clinical changes in comatose patients (e.g., development of ischemia in patients with subarachnoid hemorrhage).

Prior work in pediatric critically ill patients also showed that time from ICU admission to cEEG initiation was associated with increased mortality (OR, 1.001 [CI, 1.0002–1.001]).[12] Like our findings, they found that the probability of mortality increased by 0.1%/hour [0.02%−0.1%].[12] In patients at high risk for seizures, delays in cEEG initiation can result in delayed detection and potential exposure to a higher burden of subclinical seizures. As shown in a different cohort of pediatric critically ill patients, each 1% increase in maximum hourly seizure burden increased the

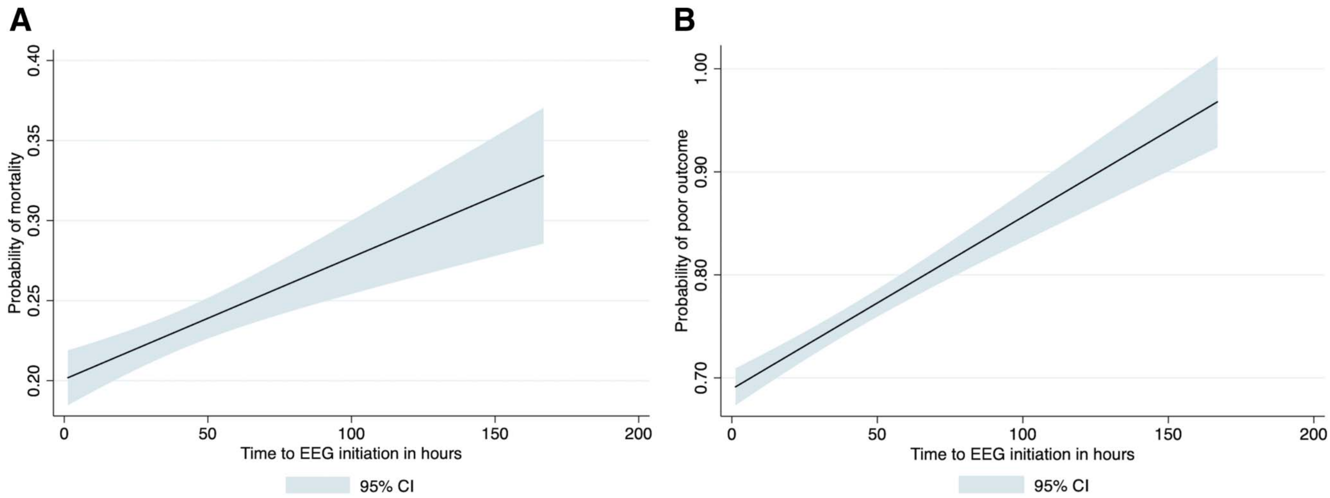

FIG. 1. Time to cEEG initiation and outcomes. **A**, Probability of mortality. As the time to cEEG initiation increases, the probability of discharge mortality increases, adjusted for age, sex, SOFA, etiology (primary neurologic vs. non-neurologic, acute brain injury, brain tumors), history of epilepsy, and seizures/status epilepticus at hospital presentation. **B**, Probability of poor outcome. As the time to cEEG initiation increases, the probability of poor outcome increases, adjusted for age, sex, SOFA, etiology (primary neurologic vs. non-neurologic, acute brain injury, brain tumors), history of epilepsy, and seizures/status epilepticus at hospital presentation. cEEG, continuous electroencephalography; SOFA, sequential organ failure assessment.

odds for neurologic decline (OR, 1.13 [CI, 1.05–1.21]).[3] Similarly, in a cohort of adult patients with subarachnoid hemorrhage, every hour of electrographic seizures was associated with increasing probability of 3-month disability (OR, 1.10 [CI, 1.01–1.21], $P = 0.04$).[2] Finally, among hospitalized adult patients, increasing burden of seizures and other RPPs was associated with increased liklihood of poor neurologic outcomes.[4,5]. Lateralized periodic discharges, generalized periodic discharge, and lateralized rhythmic delta activity measured in 6-[5] and 12-hour[4] sliding windows independently increased the probability of worse discharge functional outcomes. Our findings support the hypothesis that earlier cEEG initiation and earlier detection of seizures or RPP may improve outcomes.

Our findings contrast with a recent post hoc analysis of the continuous versus routine EEG in critically ill adults (CERTA) trial.[13,14] The investigators examined the correlation between time from admission to EEG and 6-month outcomes (mortality, modified Rankin Scale, Cerebral Performance Categories). They

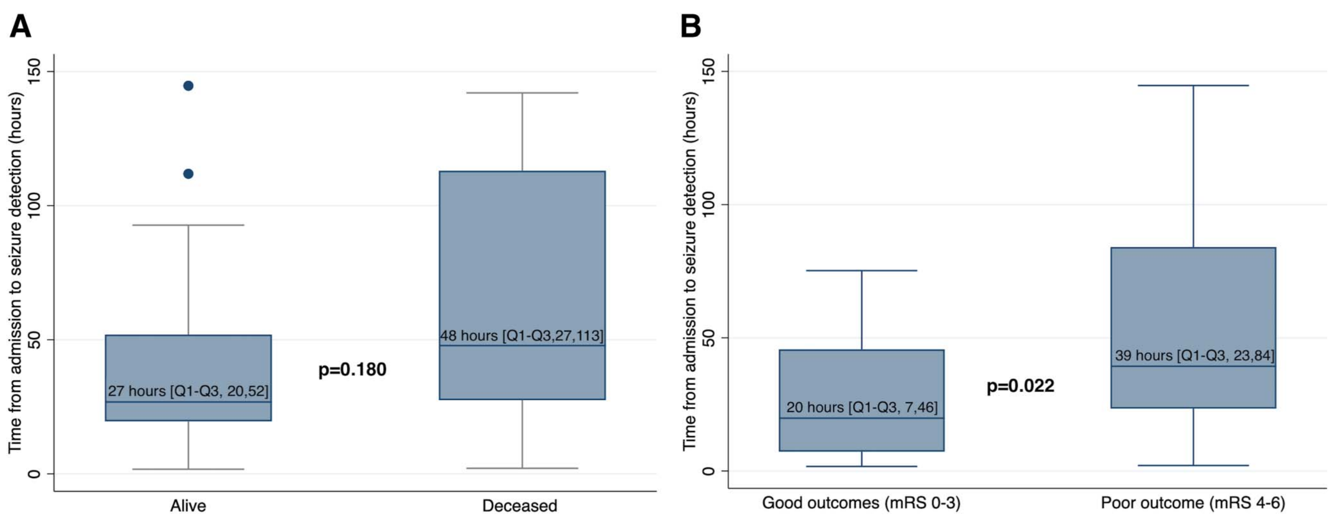

FIG. 2. Time to cEEG initiation and detection of electrographic seizures. **A**, Time from admission to detection of electrographic seizures between patients alive versus those deceased at discharge. The median time from admission to seizure detection was 27 hours [20, 52] in patients alive versus 48 hours [27, 113] in patients deceased at discharge, $P = 0.180$. **B**, Time from admission to detection of electrographic seizures between patients with good versus poor outcome at discharge. The median time to seizure detection was 20 hours [7, 46] in patients with good outcome versus 39 hours [23, 84] in patients with poor outcome, $P = 0.022$. cEEG, continuous electroencephalography.

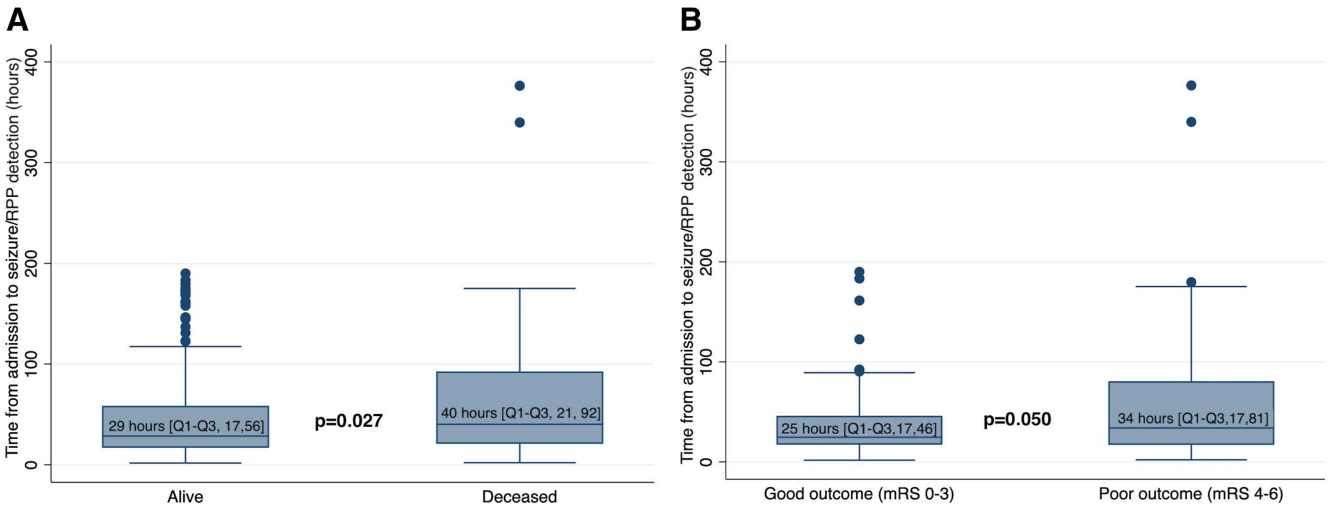

FIG. 3. Time to detection of seizures or RPP. **A**, Time from admission to initial detection of any seizures/RPPs between patients alive versus those deceased at discharge. The median time from admission to seizure detection was 29 [17, 56] in patients alive versus 40 [21, 92] in patients deceased at discharge, $P = 0.027$. **B**, Time from admission to initial detection of any seizures/RPP (LPDs, GPDs, LRDA, GRDA) EA between patients with good versus poor outcome at discharge. The median time to detection of any seizures/RPP was 25 hours [17, 46] in patients with good outcome versus 34 hours [17, 81] in patients with poor outcome, $P = 0.050$. RPP, rhythmic and periodic patterns.

found no significant difference in median time to cEEG between surviving (61.1 hours [24.3–137.7]) and deceased patients (57.5 hours [22.3–141.1]). Similarly, they did not find a correlation between time to EEG and modified Rankin Scale (rho = 0.087, $P = 0.236$), or cerebral performance categories (rho = 0.027, $P = 0.603$). However, one of the major differences between our study and the CERTA trial is that the trial excluded patients who had seizures and status epilepticus within the past 36 and 96 hours of monitoring, respectively. Arguably, these patients are at highest risk for electrographic seizures and RPPs, and in whom early detection and treatment could have the greatest clinical impact. At the same time, the CERTA trial included patients with cardiac arrest/anoxic brain injury, which was also the most common diagnostic category in the trial. This population has a distinct prognostic profile with a baseline high risk of poor outcomes. The median time from admission to cEEG in the CERTA trial was 57 hours among survivors and 61 hours among nonsurvivors, longer than our median time to initiation. The frequency of electrographic seizures/status epilepticus (10%) in CERTA was comparable with our study, while the frequency of RPPs (23% lateralized periodic discharge/generalized periodic discharges/lateralized rhythmic delta activity, and 23% GRDA) was lower than our study. The differences in eligibility and longer time to cEEG initiation in the CERTA trial may explain the differences in our findings. Limitations of our study include that it is a single-center retrospective observational study. We evaluated short-term discharge outcomes, whereas long-term functional and cognitive outcomes, along with medication adverse effects, may provide further insight into the impact of cEEG-guided treatments. Although we adjusted for underlying etiology, and disease severity, and demonstrate that SOFA scores were higher in those receiving early EEG, there may be residual confounding by indication and disease severity. Larger prospective and randomized studies are indicated to validate the findings. Future randomized studies are indicated to determine whether early cEEG-guided treatment decisions, which include both treatment escalation and de-escalation, can improve outcomes.

Increasing time to cEEG initiation was associated with higher discharge mortality and worse functional outcomes, despite no difference in detection of seizure or RPPs. We hypothesize this may be the result of (1) prolonged exposure to seizures/RPPs, (2) delays in the initiation of antiseizure treatments, resulting in untreated cerebral metabolic stress and secondary brain injury,[15] (3) delays in cEEG-guided de-escalation of ASMs and anesthetics when not indicated, and (4) delays in diagnostic work up (e.g., imaging) of new focal slowing or new focal discharges, particularly in comatose patients or those at risk for ischemia (e.g., patients with aneurysmal subarachnoid hemorrhage). Future pragmatic trials and comparative effectiveness studies are indicated to test our hypothesis.

REFERENCES

1. Herman ST, Abend NS, Bleck TP, et al. Consensus statement on continuous EEG in critically ill adults and children, part I: indications. J Clin Neurophysiol 2015;32:87–95.
2. De Marchis GM, Pugin D, Meyers E, et al. Seizure burden in subarachnoid hemorrhage associated with functional and cognitive outcome. Neurology 2016;86:253–260.
3. Payne ET, Zhao XY, Frndova H, et al. Seizure burden is independently associated with short term outcome in critically ill children. Brain 2014;137:1429–1438.
4. Zafar SF, Rosenthal ES, Jing J, et al. Automated annotation of epileptiform burden and its association with outcomes. Ann Neurol 2021;90:300–311.

5. Parikh H, Hoffman K, Sun H, et al. Effects of epileptiform activity on discharge outcome in critically ill patients in the USA: a retrospective cross-sectional study. Lancet Digital Health 2023;5:e495–e502.
6. Ney JP, van der Goes DN, Nuwer MR, Nelson L, Eccher MA. Continuous and routine EEG in intensive care: utilization and outcomes, United States 2005–2009. Neurology 2013;81:2002–2008.
7. Hill CE, Blank LJ, Thibault D, et al. Continuous EEG is associated with favorable hospitalization outcomes for critically ill patients. Neurology 2019;92:e9–e18.
8. Amerineni R, Sun H, Fernandes M, et al. Real-world continuous EEG utilization and outcomes in hospitalized patients with acute cerebrovascular diseases. J Clin Neurophysiol 2025;42:20–27.
9. Amerineni R, Sun H, Lee H, et al. Using electronic health data to explore effectiveness of ICU EEG and anti-seizure treatment. Ann Clin Transl Neurol 2021;8:2270–2279.
10. Beghi E, Carpio A, Forsgren L, et al. Recommendation for a definition of acute symptomatic seizure. Epilepsia 2010;51:671–675.
11. Hirsch LJ, Fong MW, Leitinger M, et al. American Clinical Neurophysiology Society's standardized critical care EEG terminology: 2021 version. J Clin Neurophysiol 2021;38:1–29.
12. Sánchez Fernández I, Sansevere AJ, Guerriero RM, et al. Time to electroencephalography is independently associated with outcome in critically ill neonates and children. Epilepsia 2017;58:420–428.
13. Rossetti AO, Schindler K, Sutter R, et al. Continuous vs routine electroencephalogram in critically ill adults with altered consciousness and no recent seizure: a multicenter randomized clinical trial. JAMA Neurol 2020;77:1225–1232.
14. Urbano V, Novy J, Alvarez V, Schindler K, Rüegg S, Rossetti AO. EEG recording latency in critically ill patients: impact on outcome. An analysis of a randomized controlled trial (CERTA). Clin Neurophysiol 2022;139:23–27.
15. Struck AF, Westover MB, Hall LT, Deck GM, Cole AJ, Rosenthal ES. Metabolic correlates of the ictal-interictal continuum: FDG-PET during continuous EEG. Neurocrit Care 2016;24:324–331.