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ORIGINAL RESEARCH

Neurology

Point-of-care electroencephalography enables rapid evaluation and management of non-convulsive seizures and status epilepticus in the emergency department

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Abstract

Objectives: To describe our institutional experience with point-of-care electroencephalography (pocEEG) and its impact on the evaluation/management of suspected non-convulsive seizures in the emergency department (ED).

Methods: We retrospectively identified 157 adults who underwent pocEEG monitoring in our community hospital ED in 1 year. We calculated the time to obtain pocEEG in the ED (door-to-EEG time) and examined the impact of pocEEG findings (categorized as seizure, highly epileptiform patterns, slowing, or normal activity) on antiseizure medication treatment.

Results: PocEEG revealed seizures (14%, $n = 22$), highly epileptiform patterns (22%, $n = 34$), slowing (44%, $n = 69$), and normal activity (20%, $n = 32$). The median door-to-EEG time (from initial ED evaluation to pocEEG monitoring) was only 1.2 hours (interquartile range 0.1–2.1) even though 55% of studies were performed after-hours (5 pm–9 am). Most patients were admitted (54% to the intensive care unit, 41% to floor). Antiseizure medication treatment occurred pre-pocEEG in 93 patients (59%) and post-pocEEG in 88 patients (56%). By reviewing the relationship between pocEEG monitoring and antiseizure medication management, we found a significant association between pocEEG findings and changes in management ($P < 0.001$). Treatment escalation occurred more frequently in patients with epileptiform activity (seizures or highly epileptiform patterns, 52%) than patients with non-epileptiform activity (normal or slow, 25%, $P < 0.001$), and avoidance of treatment escalation occurred more frequently in patients with normal or slow activity (27%) than patients with seizures or highly epileptiform patterns (2%, $P < 0.001$).

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Conclusion: Our study, the largest to date describing the real-world use of pocEEG in emergency medicine, found that rapid EEG acquisition in the ED was feasible in a community hospital and significantly affected the management of suspected non-convulsive seizures.

KEYWORDS
emergency medicine, neuro-emergencies, non-convulsive seizure, point-of-care electroencephalography, status epilepticus

1 | INTRODUCTION

1.1 | Background

As a result of expanded access to continuous electroencephalography (EEG) monitoring, non-convulsive seizures and non-convulsive status epilepticus are increasingly recognized as causes of persistent altered mental status, especially after convulsive status epilepticus and acute brain injuries.[1,2] EEG remains the gold standard for diagnosing non-convulsive seizures and guiding treatment with antiseizure medications. However, in the absence of EEG monitoring, patients suspected of having non-convulsive seizures are managed empirically. The resultant diagnostic uncertainty and variability in treatment poses multiple risks, including misdiagnosis (especially of non-epileptic events and toxic-metabolic encephalopathy), undertreatment (which increases the risk of seizure refractoriness), or overtreatment (which can lead to unnecessary intubation and prolonged hospitalization).[3–8]

1.2 | Importance

Many patients with non-convulsive seizures present first in the emergency department (ED), where access to EEG and its interpretation by EEG-trained neurologists are far more limited or entirely unavailable at both academic and community hospitals.[9–11] Point-of-care EEG (pocEEG) could fill this critical gap in the ED, as it has in inpatient and intensive care unit (ICU) settings at both academic and community hospitals.[12,13] Prior studies of pocEEG devices, including Ceribell's Rapid Response EEG system[14–17] (Figure 1), microEEG,[18,19] StatNet EEG,[20] BrainScope device,[21] and other simplified or abbreviated EEG approaches[22–24] have shown that access to EEG in the ED can improve the diagnosis of patients with suspected non-convulsive seizures. However, larger, real-world evaluations of the impact of pocEEG on antiseizure medication treatment are needed.

1.3 | Goals of this investigation

In this study, we aimed to describe our institutional experience with pocEEG and its impact on the evaluation and management of patients with suspected non-convulsive seizures in the ED.

2 | METHODS

This study was reviewed and approved by the Providence Mission Hospital institutional review board (STUDY2021000480) and reported in accordance with Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

2.1 | Study setting

Providence Mission Hospital encompasses 2 medical centers in California's Orange County, 1 in Mission Viejo and 1 in Laguna Beach. Mission Viejo Hospital is a 504-bed hospital designated as a level 2 adult and pediatric trauma center and a comprehensive stroke center with an annual volume of 54,000 ED visits and over 3000 traumas per year. It supports 63 ICU beds, including specialized neurological and cardiac ICUs, an epilepsy monitoring unit that is part of the University of Southern California's Epilepsy Care Consortium (1 bed, around 12–15 admits per year), and the Mission Neurological Institute. Although Mission Hospital does not have an independent emergency medicine training program, it does serve as a rotation site for emergency medicine residents from nearby academic programs. Conventional inpatient EEG infrastructure is supported by 2 EEG technicians (1 full time, 1 part time) who perform adult studies at Mission Hospital during normal business hours (weekdays between 9 am–5 pm) with limited weekend availability except for stat ICU studies. Although the technicians rarely perform pediatric studies at the Mission Hospital campus of Children's Hospital of Orange County (a "hospital within a hospital" located in the Mission Viejo Hospital), they do not have simultaneous outpatient EEG responsibilities.

2.2 | Study design and cohort selection

We retrospectively identified all adult patients (age ≥18 years) who underwent at least 1 episode of pocEEG monitoring with Ceribell's Rapid Response EEG at Providence Mission Hospital Mission Viejo between January 1, 2020 and December 31, 2020. Indications for EEG monitoring (preceding clinical event concerning for seizures without return to neurological baseline despite a reasonable interval of observation, unexplained encephalopathy, or cardiac arrest) were drawn

from consensus guidelines.[25–27] The flow diagram of the study cohort selection is shown in Figure 2 **(top)**. Of the 319 patients in this cohort, we identified 157 patients who had their first episode of pocEEG monitoring in the ED; the remainder underwent their first episode of pocEEG monitoring either in the ICU ($n = 92$) or the floor unit ($n = 70$). Sixteen patients who had their first episode of pocEEG monitoring in the ED underwent a total of 27 repeat pocEEG studies during their hospital course that were not assessed in this study.

The institutional pocEEG workflow is shown in Figure 2 **(bottom)**. Before the deployment of pocEEG throughout the hospital, all emergency physicians and nurses were trained on the use and setup of pocEEG. Although the order to initiate pocEEG monitoring typically came from the emergency physician, any consulting or admitting hospitalist, intensivist, or neurologist could recommend or order pocEEG monitoring. The emergency nurse would then acknowledge the order and set up the device. The emergency physician would notify the EEG-reading neurologist for a more urgent preliminary read according to an algorithm (developed through the collaboration and consensus of clinical leadership) based on the clinical context; however, the reading neurologist would provide a final read after reviewing the entire study. During business hours, the treating physician could decide whether to obtain conventional EEG or pocEEG monitoring; however, pocEEG was the *de facto* modality for EEG monitoring during after-hours (5 pm–9 am on weekdays and all-day on weekends). The duration of pocEEG monitoring was determined by shared decision-making between the emergency physician and reading neurologist based on the patient's clinical status and preliminary pocEEG findings.

2.3 | Measurements

We reviewed each patient's electronic medical record (EMR) to extract demographic and clinical information to determine the relationship between pocEEG findings and changes in antiseizure medication treatment. We collected data on the timing and duration of pocEEG monitoring only for the initial pocEEG study done in the ED (excluding repeat studies). PocEEG findings from the original clinical EEG report written by an EEG-trained reading neurologist (author P.J.D.) were categorized by an EEG-trained neurologist (author K.G.) as either seizure or status epilepticus, highly epileptiform patterns (HEP, including epileptiform discharges and rhythmic or periodic activity), slow activity (including non-epileptiform burst suppression and generalized rhythmic delta activity), and normal activity based on the most abnormal epileptiform finding present throughout the recording in accordance with prior studies of pocEEG.[14,15] PocEEG studies were not reinterpreted because we were interested in the impact of the findings that actually guided the patient's clinical care. Antiseizure medication treatment timing was tabulated as prehospital, pre-pocEEG, and post-pocEEG (patients may have received treatment multiple times).

We also collected data on the time between patients' initial evaluation in the ED (which was not necessarily the time of pocEEG request) and the start of pocEEG monitoring (door-to-EEG time), subsequent conventional EEG monitoring at any point in the hospital course, ED length of stay (in hours), ED disposition (admit to ICU, admit to floor, discharge to home or hospice care), and hospital length of stay (in days). PocEEG study timing was dichotomized into business hours (weekdays 9 am–5 pm) and after-hours (weekdays 5 pm–9 am and weekends) defined according to EEG technician hours and therefore their availability.

The Bottom Line

The authors conducted a retrospective study on the use of point-of-care electroencephalogram (EEG) in emergency departments. The study provided evidence that obtaining point-of-care EEG in the emergency department is feasible and could help both reduce the unnecessary use of antiseizure medications and improve the efficiency and throughput of emergency care for these patients with suspected non-convulsive seizures.

2.4 | Outcomes

We defined the clinical impact of pocEEG on management in terms of either appropriate antiseizure medication escalation (for pocEEG findings of seizure/HEP) or avoidance of inappropriate antiseizure medication escalation (for pocEEG finding of normal/slow activity). Two emergency physicians (authors R.K. and M.K.) more broadly categorized each patient's antiseizure medication management plan as either: antiseizure medication treatment affected by pocEEG, antiseizure medication treatment preceding pocEEG, or no antiseizure medication treatment. Each patient case was not reviewed by both emergency physicians; rather each reviewer coded a subset of the cohort and discussed their coding to ensure a concordant interpretation of the management plan categories.

We tabulated the number of antiseizure medication treatments given to assess whether normal/slow activity was associated with decreased antiseizure medication treatment. We also calculated the time (in hours) to the first antiseizure medication (either benzodiazepine or non-benzodiazepine anticonvulsant) from the start of pocEEG monitoring to evaluate whether pocEEG findings of seizure/HEP were associated with appropriately hastened antiseizure medication treatment.

2.5 | Analysis

Descriptive statistics were calculated for continuous (mean and SD or median and interquartile range [IQR]) and categorical (frequency and proportions) variables. Comparisons of categorical and continuous data, especially differences according to pocEEG findings (dichotomized according to the presence [seizure/HEP] or absence

FIGURE 1 Ceribell point-of-care EEG. Rapid Response EEG is a point-of-care EEG system composed of a headband with 10 electrodes (corresponding to the temporal chains of the conventional 10–20 system) connected to a recorder that can be set up to record EEG data within minutes. EEG data can be reviewed at the bedside on the portable recorder using either the visual display of EEG waveforms or the audio output of the Brain Stethoscope function, which converts the waveforms from each hemisphere into an audible tone conveying rhythmicity and periodicity of brain activity in real-time. EEG data can also be reviewed remotely using an online portal. Rapid Response EEG is also equipped with Clarity, an artificial intelligence algorithm that continuously monitors the burden of seizure and seizure-like activity and displays a trend of seizure burden that is visible on both the bedside recorder and the online portal as well as an alert for impending status epilepticus when seizure burden reaches 4.5 minutes in any 5-minute period. Figure reproduced with permission from Ceribell Inc. (www.ceribell.com). Abbreviation: EEG, electroencephalography.

[normal/slow] of epileptiform activity for certain analyses), were performed using χ^2 tests and one-way analysis of variance (for normally distributed data) or Kruskal-Wallis tests (for non-normally distributed data), respectively. As an exploratory analysis, we assessed the associations between door-to-EEG time (from ED arrival) and time to antiseizure medication treatment (from pocEEG monitoring initiation) with ED and hospital length of stay using Pearson's *r*. Missing data were excluded from analysis rather than imputed. A significance level of $\alpha = 0.05$ was used with Bonferroni correction for multiple comparisons.

3 | RESULTS

3.1 | Characteristics of study cohort

In the cohort of 157 patients who underwent initial pocEEG monitoring in the ED (Table 1), the average age of patients was 57.7 years (SD 22.4), 49% of patients were female, and a history of seizures or antiseizure medication use was present in 38.2% of patients. The indication for pocEEG monitoring was either a preceding clinical event concerning for seizures (45.2%), unexplained encephalopathy (51.0%), or cardiac arrest (3.8%). Median pocEEG monitoring duration was 2.1 hours (IQR 1.5–2.8), with 87 (55.4%) lasting at least 2 hours and 10 (6.4%) lasting 12–24 hours, and 54.8% of studies were performed after-hours (weekdays 5 pm–9 am or weekends). Conventional EEG monitoring was performed in 81 patients (51.6%) at some point in the hospital course after pocEEG monitoring. Patients spent a median of 3.0 hours (IQR 2.0–4.8) in the ED and 3.0 days (IQR 1.0–7.0) in the hospital before disposition/discharge, and most patients were admitted from the ED (53.5% to ICU, 41.4% to floor) with only 5.1% being discharged home.

3.2 | Main results

PocEEG revealed seizures in 22 patients (14.0%), HEP in 34 (21.7%), slowing in 69 (43.9%), and normal activity in 32 (20.4%). Ten of the 22 patients (45.4%) with seizures were confirmed to have status epilepticus on pocEEG, and either brief seizure activity or status epilepticus was noted at the start of pocEEG monitoring in 2 and 8 patients, respectively. Patients with seizures detected on pocEEG were more likely to have a preceding history of a seizure-like clinical event (77.3%, $P < 0.001$) compared to patients with pocEEG findings of HEP (41.2%)

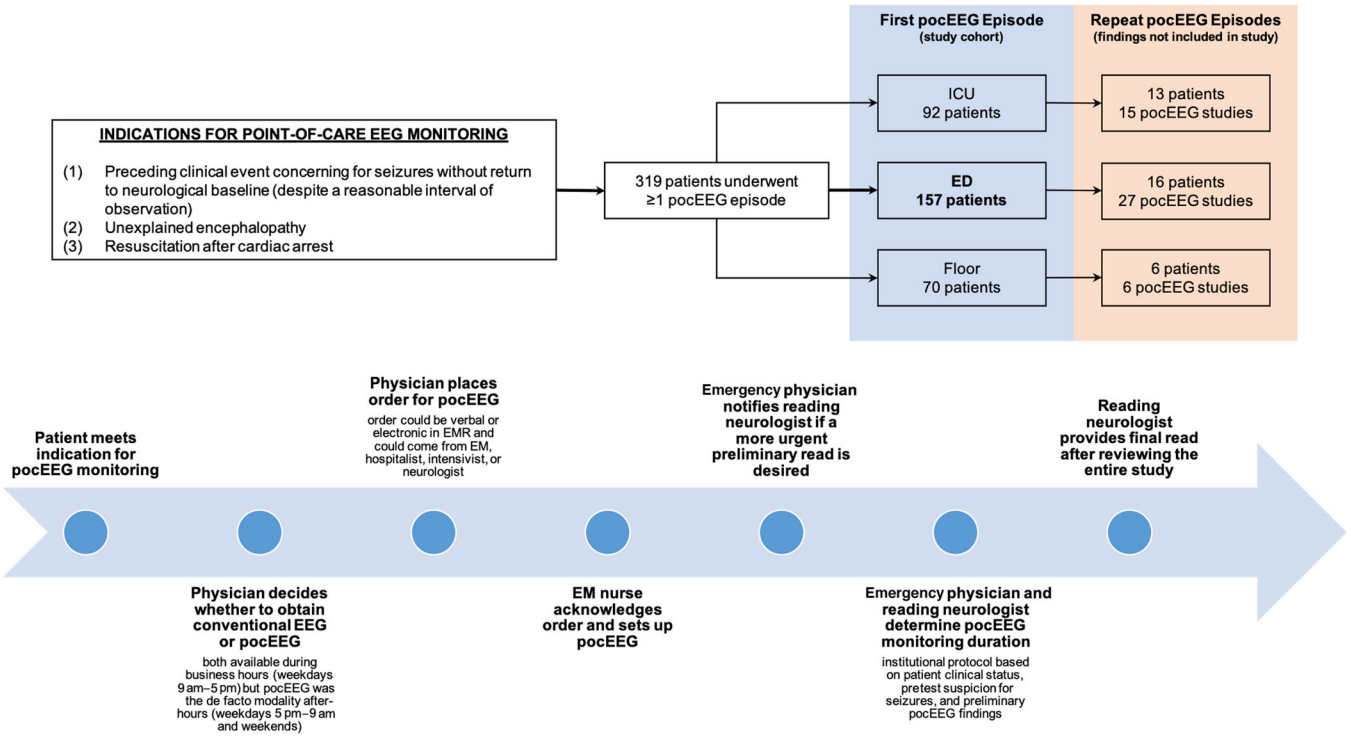

FIGURE 2 Institutional study cohort selection and pocEEG workflow. Flow diagram of study cohort selection (top): 319 patients met at least 1 indication for point-of-care electroencephalography (pocEEG) monitoring and underwent at least 1 pocEEG monitoring episode; the first episode (blue box) occurred in the emergency department (ED, $n = 157$), intensive care unit (ICU, $n = 92$), or floor ($n = 70$), and 35 patients underwent a total of 48 repeat pocEEG studies (orange box). Patients who underwent repeat pocEEG studies were not excluded, but only the first pocEEG episode that occurred in the ED was evaluated in this study. Institutional workflow for initiating pocEEG monitoring in the ED (bottom) was led by emergency medicine (EM) physicians and nurses in collaboration with consulting or admitting hospitalists, intensivists, and neurologists, especially the EEG-reading neurologist. Abbreviation: EEG, electroencephalography; EMR, electronic medical record.

or normal/slow activity (40.6%). Nearly two-thirds of the pocEEG studies that detected seizures and half of the studies that detected HEP were performed after-hours. Conventional EEG monitoring was more likely to occur at some point in the hospital course after pocEEG monitoring that revealed epileptiform activity (seizure/HEP, 69.6%) compared to non-epileptiform activity (normal/slow, 41.6%). Although most patients were admitted from the ED, patients with a normal pocEEG were more frequently discharged home (15.6%) than those with an abnormal pocEEG (2.4%).

PocEEG monitoring occurred a median of 1.2 hours (IQR 0.9–2.1) after initial ED evaluation, with more rapid door-to-EEG times observed in patients with seizures/HEP detected on pocEEG (1.1 hours) compared to patients with normal/slow activity (1.5 hours, $P = 0.02$). Rapid initiation of pocEEG within 60 minutes of initial evaluation occurred in 46 cases (29.3%) and within 90 minutes in 92 cases (58.6%). Delayed door-to-EEG times greater than 180 minutes occurred in only 21 cases (13.4%), which were largely attributable to intervening imaging studies (either as part of a stroke code or otherwise), development of suspicion for non-convulsive seizures after initial evaluation, or primary management of other medical or behavioral problems (Figure 3).

Treatment with antiseizure medications in the ED occurred pre-pocEEG for 93 patients (59.2%, 45 of whom had a history of seizures or antiseizure medication use) and post-pocEEG for 88 patients (56.1%, 38 of whom had a history of seizures or anti-seizure medication use). Divergences between pre-pocEEG and post-pocEEG treatment course occurred in 93 patients (59.2%); 44 patients (50.0%) who were treated post-pocEEG were not treated pre-pocEEG, and 49 patients (52.7%) who were treated pre-pocEEG were not treated post-pocEEG. The proportion of patients who received antiseizure medication treatment pre-pocEEG differed significantly according to specific pocEEG findings ($P = 0.006$) though not according to the presence of epileptiform activity ($P = 0.54$); pairwise comparisons of pre-pocEEG treatment were significant ($\alpha = 0.008$ after Bonferroni correction) between normal activity (34.4%) and both seizures (72.7%, $P = 0.006$) and slowing (68.1%, $P = 0.001$) but not significant between normal activity and HEP (55.9%, $P = 0.08$), seizures and HEP ($P = 0.20$), seizures and slowing ($P = 0.68$), and HEP and slowing ($P = 0.22$). Post-pocEEG antiseizure medication treatment was significantly associated with specific pocEEG findings ($P < 0.001$) and the presence of epileptiform activity ($P < 0.001$); pairwise comparisons of post-pocEEG treatment were significant ($\alpha = 0.008$ after Bonferroni correction) between seizures (81.8%) and both slow (47.8%, $P = 0.005$) and normal activity (34.4%, $P < 0.001$) and between HEP (76.5%) and both slow ($P = 0.005$) and normal activity ($P < 0.001$) but not significant between seizures and HEP ($P = 0.63$) and slow and normal activity ($P = 0.20$).

TABLE 1 Cohort characteristics.

		pocEEG findings			
	Total cohort *N* = 157	**SZ** *n* = 22 (14.0%)	**HEP** *n* = 34 (21.7%)	**SL** *n* = 69 (43.9%)	**NL** *n* = 32 (20.4%)
Age, years, mean (SD)	57.7 (22.4)	52.9 (20.5)	56.4 (22.2)	62.8 (22.7)	51.6 (21.6)
Sex, *n* (%)					
Female	77 (49.0)	13 (59.1)	15 (44.1)	32 (46.4)	17 (53.1)
Male	80 (51.0)	9 (40.9)	19 (55.9)	37 (53.6)	15 (46.9)
Neurological history, *n* (%)					
Seizures or ASM use	60 (38.2)	17 (77.3)	14 (41.2)	18 (26.1)	11 (34.4)
Brain tumor	14 (8.9)	2 (9.1)	6 (17.6)	5 (7.2)	1 (3.1)
CVD/ICH	43 (27.4)	8 (36.4)	5 (14.7)	24 (34.8)	6 (18.8)
Head trauma	20 (12.7)	4 (18.2)	3 (8.8)	6 (8.7)	7 (21.9)
Initial GCS, median (IQR)	11.0 (8.0-14.0)	9.5 (6.5-12.8)	11.0 (5.0-14.0)	10.0 (8.0-14.0)	14.0 (13.8-15.0)
pocEEG indication, *n* (%)					
Clinical event concerning for seizures	71 (45.2)	16 (72.7)	14 (41.2)	27 (39.1)	14 (43.8)
Unexplained encephalopathy	80 (51.0)	6 (27.3)	20 (58.8)	36 (52.2)	18 (56.3)
Cardiac arrest	6 (3.8)	0 (0.0)	0 (0.0)	6 (8.7)	0 (0.0)
Treated with ASMs, *n* (%)					
Pre-hospital	9 (5.7)	4 (18.2)	4 (11.8)	1 (1.4)	0 (0.0)
Pre-pocEEG	93 (59.2)	16 (72.7)	19 (55.9)	47 (68.1)	11 (34.4)
Post-pocEEG	88 (56.1)	18 (81.8)	26 (76.5)	33 (47.8)	11 (34.4)
Intubated during visit, *n* (%)	67 (42.7)	9 (40.9)	19 (55.9)	30 (43.5)	9 (28.1)
pocEEG study timing, *n* (%)					
Business hours	71 (45.2)	14 (63.6)	17 (50.0)	25 (36.2)	15 (46.9)
After hours	86 (54.8)	9 (36.4)	17 (50.0)	44 (63.8)	17 (53.1)
Door-to-EEG time in hours, median (IQR)	1.2 (0.9-2.1)	1.3 (0.7-1.8)	1.0 (0.8-1.9)	1.5 (1.1-2.7)	1.2 (0.9-2.0)
pocEEG study duration in hours, median (IQR)	2.1 (1.5-2.8)	2.1 (1.5-3.6)	2.1 (1.5-2.5)	2.1 (1.6-3.5)	2.0 (1.3-2.5)
Disposition from ED, *n* (%)					
Admit to ICU	84 (53.5)	14 (63.6)	19 (55.9)	39 (56.5)	12 (37.5)
Admit to floor	65 (41.4)	7 (31.8)	15 (44.1)	28 (40.6)	15 (46.9)
Discharge home	8 (5.1)	1 (4.5)	0 (0.0)	2 (2.9)	5 (15.6)
ED length of stay in hours, median (IQR)	3.0 (2.0-4.8)	3.0 (2.0-5.0)	3.3 (2.1-4.1)	3.0 (1.8-4.5)	3.3 (2.1-5.2)
Hospital length of stay in days, median (IQR)	3.0 (1.0-7.0)	4.0 (2.0-6.0)	4.0 (2.0-8.0)	4.0 (2.0-8.0)	1.0 (1.0-3.0)

Note: The number of patients treated with ASMs at different time points (prehospital, pre-pocEEG, post-pocEEG) exceeds the overall sample size because some patients were treated at multiple time points. PocEEG study timing was defined as either business hours (weekdays 9 am-5 pm) or after-hours (weekdays 5 pm-9 am or all-day on weekends) based on EEG technician availability.

Abbreviations: ASM, antiseizure medication; CVD/ICH, cerebrovascular disease or intracranial hemorrhage; ED, emergency department; GCS, Glasgow Coma Scale; HEP, highly epileptiform pattern; IQR, interquartile range; NL, normal activity; pocEEG, point-of-care electroencephalography; SL, slow activity; SZ, seizure or status epilepticus.

By reviewing each patient's ED course and the relationship between pocEEG monitoring and antiseizure medication treatment (Table 2), we found a significant association between pocEEG findings and changes in management as assessed by emergency physicians ($P < 0.001$). Treatment escalation occurred more frequently in patients with epileptiform activity (51.8%) than in normal/slow activity (24.8%, $P < 0.001$), and avoidance of treatment escalation occurred more frequently in patients with normal/slow activity (26.7%) than in patients with seizures/HEP (1.8%, $P < 0.001$). The proportion of patients who were already treated independent of pocEEG findings was comparable between seizures/HEP (46.4%) and normal/slow activity (48.5%, $P = 0.80$). Overall, the number of antiseizure medication treatments given was greater for patients with seizures/HEP (median 3.0 [IQR 2.0–5.0]) than for patients with normal/slow activity (median 2.0 [IQR

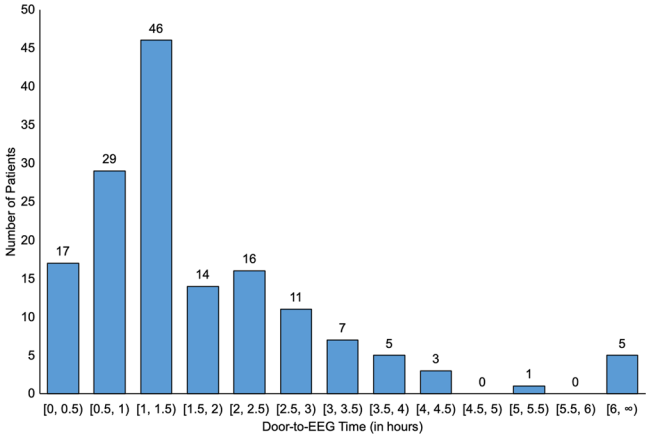

FIGURE 3 Distribution of door-to-EEG times. Door-to-EEG times were calculated as the difference between the time of initial ED evaluation and the time at which pocEEG monitoring was initiated. Each bin was defined as inclusive of the lower bound (indicated by bracket) and exclusive of the upper bound (indicated by parenthesis). Data for door-to-EEG times were missing for 3 patients. Abbreviation: EEG, electroencephalography.

1.0–2.0], $P < 0.001$); all patients with seizures received at least 1 antiseizure medication treatment, and only 1 patient with HEP received no antiseizure medication treatment. Comparing time to first antiseizure medication treatment after pocEEG initiation, patients with seizures/HEP also had a lower median time to first benzodiazepine (0.8 hours [IQR 0.3–2.6]) and non-benzodiazepine anti-convulsant (0.6 hours [IQR 0.2–2.0]) compared to patients with normal/slow activity (benzodiazepine: 3.9 hours [IQR 1.4–11.9], $P = 0.007$; anticonvulsant: 2.4 hours [IQR 0.3-11.8], $P = 0.06$).

In our exploratory analysis, the door-to-EEG time was positively correlated with length of stay in the ED ($r = 0.16$, $P = 0.04$) and the hospital ($r = 0.38$, $P < 0.001$). However, the time to first antiseizure medication treatment post-pocEEG was not significantly correlated with either ED length of stay (benzodiazepine: $r = 0.05$, $P = 0.71$; anticonvulsant: $r = -0.01$, $P = 0.92$) or hospital length of stay (benzodiazepine: $r = -0.14$, $P = 0.30$; anticonvulsant: $r = -0.09$, $P = 0.45$).

4 | LIMITATIONS

Our study was performed using a non-controlled retrospective design using a study period during which pocEEG was already being used as standard-of-care. As such, this study was not designed to prospectively study the real-time impact of pocEEG findings on antiseizure medication treatment, control for variation between physicians in pocEEG-directed treatment patterns, determine the temporal relationship between pocEEG interpretation by the EEG-reading

TABLE 2 Impact of pocEEG findings on antiseizure medication management.

		pocEEG findings			
	Total cohort *N* = 157	**SZ** *n* = 22 (14.0%)	**HEP** *n* = 34 (21.7%)	**SL** *n* = 69 (43.9%)	**NL** *n* = 32 (20.4%)
Impact on management, *n* (%)					
ASM given based on pocEEG	54 (34.4)	13 (59.1)	16 (47.1)	19 (27.5)	6 (18.8)
ASM given before pocEEG	75 (47.8)	9 (40.9)	17 (50.0)	40 (58.0)	9 (28.1)
No ASM given	28 (17.8)	0 (0.0)	1 (2.9)	10 (14.5)	17 (53.1)
Number of ASMs given, median (IQR)	2.0 (1.0-3.0)	3.5 (3.0-5.0)	3.0 (2.0-5.0)	2.0 (1.0-2.0)	1.0 (0.0-2.0)
Time to first ASM after pocEEG in hours, median (IQR)					
BDZ	1.6 (0.4-6.0)	0.7 (0.1-2.5)	1.0 (0.4-2.5)	2.7 (1.6-8.2)	8.5 (1.3-15.9)
AC	0.9 (0.2-4.2)	0.4 (0.2-1.4)	0.7 (0.3-2.4)	1.9 (0.3-13.0)	2.8 (2.5-8.1)

Abbreviations: AC, non-benzodiazepine anti-convulsant; ASM, antiseizure medication; BDZ, benzodiazepine; HEP, highly epileptiform pattern; IQR, interquartile range; NL, normal activity; pocEEG, point-of-care electroencephalography; SL, slow activity; SZ, seizure or status epilepticus.

A significance level of $\alpha = 0.05$ was used for omnibus significance tests and comparisons between pocEEG findings when dichotomized as epileptiform (seizure or HEP) versus non-epileptiform (slow or normal) activity; pairwise comparisons between specific pocEEG findings used a Bonferroni-corrected significance level of $\alpha = 0.008$.

Impact on management (coded by 2 emergency physicians) differed significantly between pocEEG findings ($P < 0.001$); patients with epileptiform activity were more likely to be treated than patients with normal/slow activity ($P < 0.001$), and patients with non-epileptiform activity were more likely to not have treatment escalation than patients with seizures or HEP ($P < 0.001$). The proportion of patients who were already treated independent of pocEEG findings was comparable between patients without and without epileptiform activity ($P = 0.80$). The number of ASM treatments also differed significantly between pocEEG findings ($P < 0.001$); this was driven by differences between patients with epileptiform activity and patients with normal/slow activity ($P < 0.001$) rather than between patients with seizures and patients with HEP ($P = 0.26$). Significant differences in the time to first BDZ ($P = 0.04$) after initiation of pocEEG monitoring were also observed, whereas time to first AC treatment did not differ significantly between pocEEG findings ($P = 0.14$); patients with epileptiform activity had lower time to first BDZ (median 0.8 hours [IQR 0.3-2.6], $P = 0.007$) and time to first AC (median 0.6 hours [IQR 0.2-2.0], $P = 0.06$) compared to patients with non-epileptiform activity (BDZ: median 3.9 hours [IQR 1.4-11.8]; AC: median 2.4 hours [IQR 0.3-11.8]).

neurologist and initiation of antiseizure medication treatment, or compare the clinical impact of pocEEG to that of conventional EEG in the ED. Our institutional review board-approved study period did not allow for the collection of historical data to describe pre-pocEEG conventional EEG practice, but we have referred to prior studies that have described aspects of conventional EEG practice (such as delays in EEG acquisition). However, it should be noted that access to conventional EEG in our community hospital ED has anecdotally been very limited, consistent with reports from other community hospitals.[15,16] In addition, the identification of the times of initial ED evaluation and initiation of pocEEG monitoring to calculate door-to-EEG time was based on a retrospective chart review, which may introduce some measurement error in comparison to a prospective study design. It should be noted that this door-to-EEG time is distinct from the time between pocEEG request and setup, which has been reported in prior studies to be roughly 5 minutes for pocEEG.[14,16,28] The typical method of retrospectively ascertaining the time of pocEEG request through the time stamp of the pocEEG order in the EMR can be unreliable, especially in the ED setting, because the EMR order may often have been either preceded by a verbal order or placed retroactively after pocEEG monitoring had already started; as a result, we elected not to quantify or report the time between pocEEG request and setup in our cohort.[29]

Because EEG monitoring (whether with conventional EEG at any hospital that can support it or with pocEEG at hospitals that have adopted it) is standard-of-care for patients meeting the indications established in consensus guidelines[25–27] and applied in our study, it was not feasible to compare characteristics and outcomes of patients who did not undergo pocEEG monitoring to those who did. It should be noted that the majority of patients who underwent pocEEG monitoring were admitted from the ED, perhaps owing to the greater predilection toward more severe illness in this population compared to those who did not warrant pocEEG monitoring. For example, most patients with persistent encephalopathy (even those without seizures) had some underlying medical condition that required further workup and treatment, and all patients with cardiac arrest required intensive monitoring post-resuscitation. Among the population of patients with a preceding clinical event concerning for seizures (including stroke-mimics presenting with aphasia or other focal deficits without radiographic evidence of stroke), pocEEG monitoring was reserved for those who had not returned to their neurological baseline despite a reasonable time interval. Therefore, by and large, these were not patients with single seizures that would otherwise be discharged, and none of the patients in our cohort were monitored for psychogenic events.

The quantification of clinical impact using antiseizure medication treatment data may be affected by nuances of ED seizure management that could not be adequately controlled in a retrospective study. For example, patients who either presented with convulsive status epilepticus or had a history of epilepsy with possible antiseizure medication nonadherence as the cause for breakthrough seizures could reasonably be treated with antiseizure medications before pocEEG and may even have treatment "escalation" after pocEEG ruled out ongoing seizures due to the decision to load the patient's home antiseizure medication regimen to prevent seizure recurrence. Additionally, patients presenting with acute brain injuries or conditions with a high risk of seizures (e.g., intracranial hemorrhage, traumatic brain injury, central nervous system infection, or brain tumors) may have reasonably received antiseizure medications to prevent acute symptomatic seizures, constituting an "escalation" of antiseizure medication treatment despite the absence of clinical or electrographic seizure activity. We are mindful that a non-seizure pocEEG finding after antiseizure medication treatment also does not exclude the presence of preceding seizures; however, patients with a normal pocEEG might be reasonably spared excessive antiseizure medication treatment escalation under the assumption that electrographic seizure activity was ongoing based on clinical suspicion alone. We attempted to address these nuances by having emergency physicians review each patient's medical record and try to assess the overall impact of pocEEG on antiseizure medication treatment. However, we recognize that this is a qualitative assessment, and therefore we also evaluated objective measurements (frequency of treatment pre- and post-pocEEG, number of antiseizure medication treatments, time to first antiseizure medication treatment) that matched outcomes in prior studies and would be reproducible in future studies.[30]

5 | DISCUSSION

Point-of-care diagnostics, from bedside ultrasound to portable serum analyzers, are fundamental to the practice of modern emergency medicine. However, there has long been a dearth of point-of-care tools for empowering emergency physicians in managing neurological emergencies, such as non-convulsive seizures and non-convulsive status epilepticus.[31,32] This is especially important to address given the rising awareness of non-convulsive seizures and status epilepticus among emergency physicians, the considerable diagnostic uncertainty that persists in diagnosing non-convulsive seizures without EEG, and the significant gaps in access to EEG in the ED.[9] A recently developed pocEEG device developed by Ceribell has been shown to be effective in expanding access to rapid EEG monitoring, both in the ED and ICU settings at academic and community hospitals, and facilitating earlier management of non-convulsive seizures.[14,15,17]

This study, the largest to date describing the use of pocEEG in emergency medicine, found that rapid EEG acquisition in the ED significantly affected the detection and treatment of suspected non-convulsive seizures. Epileptiform activity (seizures or highly epileptiform patterns that would warrant either urgent antiseizure medication treatment or close monitoring) was detected in 36% of patients, including non-convulsive status epilepticus in 6.4% of all patients, and 45% of patients with seizures were found to have ongoing seizure activity at the start of pocEEG monitoring. The majority of patients (59%) underwent pocEEG monitoring within 90 minutes of initial ED evaluation with a median door-to-EEG time of 1.2 hours. For comparison, prior reports[14,29] of the delay in obtaining conventional EEG described a median delay of about 4 hours in the ICU and 1.5 hours in the ED at academic hospitals with excellent EEG technologist availability, and it should be noted that this delay was calculated from the time of EEG request,

which could be hours after the time of initial evaluation used to calculate the door-to-EEG time in our cohort. Although over half of patients were treated with antiseizure medications before pocEEG, pocEEG findings were associated with a significant difference in rates of subsequent antiseizure medication treatment escalation, number of antiseizure medication treatments given, and time to antiseizure medication administration that were consistent with expedited and appropriate treatment of patients with non-convulsive seizures and avoidance of overtreatment of patients in whom ongoing seizure activity was ruled out. Furthermore, we found that shorter door-to-EEG time was associated with shorter ED and hospital length of stay.

Our findings regarding EEG-directed antiseizure medication treatment changes are consistent with prior studies of Ceribell's pocEEG, as well as those of other pocEEG devices.[15,18,30] We found relatively high rates of antiseizure medication treatment preceding pocEEG monitoring, however we did not observe any significant differences in pre-pocEEG anti-seizure medication treatment rates associated with subsequent pocEEG detection of epileptiform versus non-epileptiform activity, suggesting that clinical suspicion alone can be unreliable in diagnosing such abnormalities. This same phenomenon was previously demonstrated in the Does Use of Rapid Response EEG Impact Clinical Decision Making (DECIDE) multicenter study of Ceribell pocEEG, and a subsequent economic analysis projected the financial impacts of inappropriate or inaccurate treatment in terms of excess rates of intubation and prolonged ICU and hospital length of stay ranged from $3971 to $17,290 per patient.[4,14]

We also found 55% of pocEEG studies occurred after-hours, during which conventional EEG monitoring was otherwise unavailable. Before the deployment of pocEEG at our hospital, the treating physician would have needed to determine whether EEG monitoring could wait until business hours (which risks missing the diagnosis of non-convulsive seizures and delaying appropriate treatment and triage), an EEG technician would need to be urgently called in (which risks additional call burden on EEG technicians as well as increased expenses due to overtime pay), or the patient would need to be transferred to an EEG-capable hospital (which poses a significant economic penalty to the referring hospital due to transportation costs and lost revenue).[17] Therefore, the opportunity for pocEEG to mitigate these logistical and financial externalities of gaps in conventional EEG infrastructure has the potential to greatly augment the value of pocEEG for patients, physicians, and hospital leadership at many centers.

In addition, the positive correlation between door-to-EEG time and ED length of stay suggests that patients suspected to have non-convulsive seizures may benefit from earlier EEG evaluation to guide appropriate disposition from the ED and improve hospital throughput.[12,33] This has both practical and financial implications for efficiency in patient care and hospital logistics. Furthermore, it should be pointed out that these pocEEG devices, by identifying patients who would most benefit from long-term monitoring, might actually help increase the efficiency of conventional EEG services at hospitals with limited resources. This is especially true at community hospitals, where trained EEG technologists provide both inpatient and outpatient EEG services and may have to delay outpatient EEG procedures to urgently assess a patient in the ED or ICU. The impact of pocEEG on overall health care and hospital efficiency, and the incremental value of automated detection algorithms in directing emergency physicians' treatment at the bedside, may be the subject of future studies.

In summary, point-of-care EEG can empower emergency physicians in their bedside evaluations of patients with suspected non-convulsive seizures, guide appropriate antiseizure treatment, facilitate more efficient patient care, and support novel approaches for the management of neurological emergencies.

AUTHOR CONTRIBUTIONS

Richard Kozak and Matthew Kaplan conceived the study and collected the data. Parshaw J. Dorriz reviewed EEG studies. Kapil Gururangan analyzed the data and drafted the manuscript. All authors contributed to data interpretation and critical revision of the manuscript, had full access to the study data, and approved of the submission of the manuscript. Richard Kozak and Kapil Gururangan take responsibility for the manuscript as a whole.

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CONFLICT OF INTEREST STATEMENT

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PRIOR PRESENTATIONS

Findings from this report have been previously presented at annual meetings of the Society for Academic Emergency Medicine (SAEM; May 10-13, 2022 in New Orleans, LA and May 16-19, 2023 in Austin, TX), American College of Emergency Physicians (ACEP; Oct 1-4, 2022 in San Francisco, CA), American Neurological Association (ANA; Oct 22-25, 2022 in Chicago, IL), and American Epilepsy Society (AES; Dec 2-6, 2022 in Nashville, TN).

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