Nonconvulsive Seizure Detection by Reduced-Lead Electroencephalography in Children with Altered Mental Status in the Emergency Department

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Objectives To evaluate the proportion of children presenting to the emergency department (ED) with altered mental status who demonstrate nonconvulsive seizures on reduced-lead electroencephalography (EEG), and to further investigate the characteristics, treatment, and outcomes in these patients compared with patients without nonconvulsive seizures.

Study design In this retrospective cohort study, we reviewed the database and medical records of pediatric patients (aged <18 years) in a single ED between May 1, 2016, and April 30, 2018. We first determined the proportion of nonconvulsive seizures among patients with altered mental status (Glasgow Coma Scale <15). We then compared the clinical presentation, demographic data, clinical diagnosis, EEG results, treatment, and outcomes of patients with altered mental status with nonconvulsive seizures and those without nonconvulsive seizures.

Results In total, 16.9% of the patients with altered mental status (41 of 242; 95% CI, 12.2%-21.6%) evaluated by EEG had detectable nonconvulsive seizure, equivalent to 4.4% (41 of 932) of all patients with altered mental status presenting at our hospital. More than 80% of patients monitored for nonconvulsive seizures had a previous history of seizures, often febrile. Patients with nonconvulsive seizures were older (median, 68.5 vs 36.1 months) and had a higher Pediatric Cerebral Performance Category score at presentation (median, 2.0 vs 1.0). In addition, the proportion of patients admitted to the intensive care unit was significantly higher in the patients with nonconvulsive seizures (30.3% vs 15.0%). However, total duration of hospitalization, neurologic sequelae, and 30-day mortality rate did not differ between the 2 groups.

Conclusions A relatively high percentage of pediatric patients with altered mental status in the ED experience nonconvulsive seizures. The use of reduced-lead EEG monitoring in the ED might facilitate the recognition and treatment of nonconvulsive seizures, especially among patients with a history of seizures. *(J Pediatr 2019;207:213-9).*

Children often present to the emergency department (ED) with altered mental status, which is attributed to the nonspecific manifestation of brain dysfunction and has numerous possible etiologies. ED physicians frequently face a challenge in treating patients with altered mental status because physical examination alone cannot exclude subclinical seizures.

Status epilepticus is a life-threatening prolonged seizure condition for which treatment is time-sensitive and mortality rates are high.[1-3] Early treatment of status epilepticus is critical to reduce neurologic morbidity and mortality. There are 2 types of status epilepticus: convulsive and nonconvulsive. Although convulsive status epilepticus is relatively easy to identify, nonconvulsive status epilepticus is not; a definitive diagnosis requires electroencephalography (EEG).[4]

Pediatric nonconvulsive status epilepticus is not uncommon,[3,5-13] with a significant proportion of cases diagnosed only after hospital admission, often to the intensive care unit (ICU).[9-11] Delays in obtaining an EEG in patients with nonconvulsive status epilepticus further delay its diagnosis and treatment. For this reason, a quicker, more efficient means of detecting nonconvulsive seizures before nonconvulsive status epilepticus develops is needed.

EEG is a valuable tool for objectively evaluating patients with altered mental status,[14-16]; however, it remains underused in the ED. It is unknown whether obtaining an EEG in the ED might influence therapeutic decision making and clinical outcomes.

ED	Emergency department
EEG	Electroencephalography
ICU	Intensive care unit
PCPC	Pediatric Cerebral Performance Category

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The objectives of this study were to determine the prevalence of nonconvulsive seizures among pediatric patients with altered mental status in the ED, and to examine the characteristics, treatment, and outcomes of patients with nonconvulsive seizures compared with those without nonconvulsive seizures. We used reduced-lead EEG with 4 channels across the bilateral frontal and occipital regions; this simplified EEG system enables rapid evaluation of brain activity.

Methods

This retrospective cohort study was conducted with the approval of the Ethics Committee of Hyogo Prefectural Kobe Children's Hospital, with a waiver of informed consent due to the retrospective nature of data collection. Between May 1, 2016, and April 30, 2018, 13 696 patients aged <18 years visited our hospital. Of these, 932 presented with altered mental status. A total of 246 of the 932 patients with altered mental status were monitored by reduced-lead EEG. An EEG was ordered when subclinical seizure activity was suspected by the ED physician after an initial clinical assessment. Patients in whom convulsions were detected during EEG monitoring (n = 4) were excluded. The remaining 242 patients were analyzed to determine the prevalence of nonconvulsive seizures. We excluded duplicate visits of the patients and analyzed only the first visit to compare the characteristics, treatment, and outcomes of patients with and those without nonconvulsive seizures.

We reviewed database and medical records of clinical presentation, demographic data, clinical diagnosis, EEG results, treatment, and outcomes were curated. Altered mental status was defined as a score of ≤14 on the Glasgow Coma Scale. This scale was designed for worldwide clinical use to assess impaired consciousness.[17] According to a previous report, Glasgow Coma Scale scores in intubated patients were found to predict linearly Glasgow verbal scores from eye and motor scores.[18,19] Therefore, among intubated patients or patients with tracheotomy, we defined E4VTM6 as a Glasgow Coma Scale score of 15. Initial neurologic symptoms were those first noted by parents or caregivers, such as seizures, disturbed consciousness, abnormal movements of the face or extremities, partial or complete paralysis, muscle weakness, partial or complete loss of sensation, or any psychiatric symptoms. Seizure onset was defined as the beginning of any neurologic symptoms, such as convulsions or eye deviation. Initial assessment was considered to be the beginning of care by an ED physician. Neurologic sequelae included loss of speech, hearing impairment, motor deficits, behavioral problems, epilepsy, or cognitive impairment. A baseline neurological assessment was performed using the Pediatric Cerebral Performance Category (PCPC) scale.[20]

General Clinical Protocol

In our hospital, at any given time, 1-3 attending physicians, 1 or 2 fellows, and 1 or 2 residents work in the ED. The neurologists are on call 24 hours a day. In general, neurologists are not immediately available in the ED. In some cases, however, the ED physicians consult with a neurologist when the patient arrives. In the present study, approximately 86% of EEGs were interpreted only by an ED physician (**Table I**). The use of EEG and administration of antiepileptic drugs were left to the discretion of the attending physicians. Neurologists at our hospital recommend that ED physicians administer antiepileptic drugs when they see 1 of 2 EEG patterns (described later), and antiepileptic drug therapy was administered on an individual basis by ED physicians according to hospital protocol.[21] The duration of EEG monitoring was at the discretion of the ED physicians and varied among patients.

Table I. Demographic data for patients with altered mental status who underwent reduced-lead EEG (N = 242)

Characteristics	Value
Age, mo, median (IQR)	43.9 (22.2-71.0)
Male sex, n (%)	124 (51.2)
PCPC on initial assessment, median (IQR)	1 (1-2)
Glasgow Coma Scale at presentation, median (IQR)	8 (5-11)*
Clinical presentation	
No seizure before ED, n (%)	38 (15.7)
Seizure before ED, n (%)	204 (84.3)
Clinical signs of seizure before ED, n (%)	
Ocular movement abnormalities	11 (5.4)
Partial motor activities in face or extremities	17 (8.3)
Generalized convulsion	176 (86.3)
Total seizure duration, min, median (IQR)	35.0 (10.0-62.3)
Time from the onset of initial neurologic symptoms to EEG monitoring, min, median (IQR)	87.0 (62.0-164.8)
Time from the initial assessment by ED physician to EEG monitoring, min, median (IQR)	20.5 (14.0-39.0)
EEG monitoring duration, min, median (IQR)	20.0 (11.0-38.8)
Antiepileptic drugs administered after EEG monitoring, n (%)	54 (22.3)
Neurologist present at ED, n (%)	35 (14.5)
Hospital admission, n (%)	192 (79.3)
ICU stay, n (%)	38 (15.7)
Clinical diagnosis, n (%)	
Febrile seizure	143 (59.1)
Epilepsy	53 (21.9)
Encephalopathy/encephalitis	23 (9.5)
Psychogenic nonepileptic seizure	10 (4.1)
Hypoglycemia	5 (2.1)
Trauma	2 (0.8)
V-P shunt dysfunction	2 (0.8)
Hyponatremia	1 (0.4)
Respiratory failure	1 (0.4)
Gastroenteritis	1 (0.4)
Unknown	1 (0.4)

V-P, ventriculoperitoneal.
*Two patients with nonconvulsive seizure and 3 patients without nonconvulsive seizure with tracheotomy were excluded from Glasgow Coma Scale assessment.

Reduced-Lead EEG Application and Interpretation

Using a portable digital EEG system (EEG-9100; Nihon Kohden, Tokyo, Japan) (**Figure**, A), digital recordings were made using 4 channels across the bilateral frontal (2 electrodes placed 2 fingerbreadths above the left and right pupils; Fp1-A1 and Fp2-A2) and occipital regions (2 electrodes placed at a similar distance on either side above the inion; O1-A1 and O2-A2), according to the International 10-20 system. We chose the frontal and occipital regions to detect abnormal waves as wide as possible. Electrodes were also placed on 3 sites: the temporal

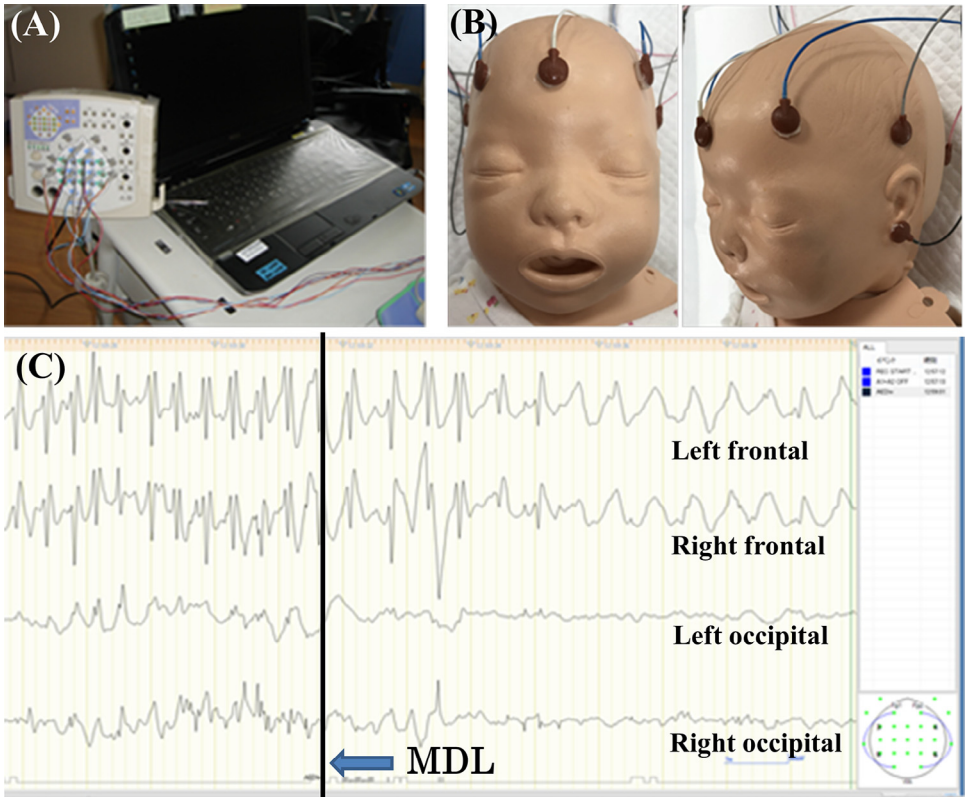

Figure. **A,** The reduced-lead EEG system used here. **B,** Electrode placement. **C,** Representative reduced-lead EEG readout showing persistent rhythmic spikes. The abnormal waves disappeared after administration of intravenous midazolam (MDL).

areas, ears, and eye bridge for reference (**Figure**, B). Application of this simplified EEG is easy, and ED physicians can quickly learn to apply this montage smoothly; thus, no special training is required. We have continued to use reduced-lead EEG without placement of the standard electrodes of the 10-20 system, because the latter is complicated and time-consuming in an ED setting. Therefore, only reduced-lead EEG data obtained in the ED were included in these analyses. EEGs were ordered when subclinical seizure activity was suspected by ED physicians after an initial clinical assessment. EEG, assessment, and treatment were then performed in the ED.

ED physicians administered antiepileptic drugs when they viewed 1 of 2 EEG patterns: (1) persistent rhythmic waves, spikes, or spikes and waves or (2) persistent, high-amplitude slow waves with rhythmical evolution. Prompt administration of antiepileptic drugs is required to treat nonconvulsive seizures, and thus we simplified the recommendation for antiepileptic drug administration, because the strict criteria for nonconvulsive seizure treatment are very complicated and warrant a neurologist's consultation. This simplified EEG interpretation does not require ED physicians to undergo any special training. Certified neurologists provided on-the-job training to the ED physicians. If the ED physicians were unable to make a decision based on persistent, rhythmic slow waves with or without evolution, they consulted a neurologist. The next day, a certified neurologist checked all archived EEGs and determined the patient's final EEG status.

The definition of nonconvulsive seizure for final EEG status was in accordance with that reported by Claassen et al.[22] In summary, rhythmic discharge or a spike and wave pattern with definite evolution in frequency, location, or morphology lasting at least 10 seconds qualified as a nonconvulsive seizure. Evolution in amplitude alone did not qualify without overt convulsive movements, such as generalized tonic-clonic seizures, grand mal seizures, convulsions, rhythmic jerking, or rhythmic twitching.

Statistical Analyses

Results are expressed as a prevalence (%), mean ± SD, or median (IQR). The Mann-Whitney *U* and Fisher exact tests were used for statistical analyses, as appropriate. A P value < .05 was considered statistically significant. Analyses were performed using GraphPad Prism 5.0 (GraphPad Software, San Diego, California).

Results

Nonconvulsive Seizure Prevalence and Population Demographics

The proportion of nonconvulsive seizure detected among ED patients who were monitored via reduced-lead EEG was 16.9% (95% CI, 12.2%-21.6%), which accounted for 4.4% of all patients with altered mental status seen in our ED. Characteristics of the study sample are presented in **Table I**. The median

patient age was 43.9 months, and patients were equally distributed by sex. The median PCPC score on initial assessment was 1, and the median Glasgow Coma Scale score was 8. Before ED arrival, 84.3% of patients had a history of seizures, with generalized convulsions the most common type (86.3%). The median total duration of seizure was 35.0 minutes.

The median intervals from the onset of initial neurologic symptoms and initial assessment by an ED physician to EEG monitoring were 87.0 minutes and 20.5 minutes (with a minimum period of 4 minutes), respectively. Fifty-four of the 242 patients (22.3%) were treated with antiepileptic drugs, based on the EEG results. Neurologists were present with the consulting ED physician in 14.5% of cases, meaning that ED physicians alone assessed EEG results in 85.5% of cases. Altered mental status was caused primarily by febrile seizures (59.1%) and secondarily by epilepsy (21.9%). Among all patients, 79.3% required hospitalization and 15.7% required an ICU stay.

Detailed Characteristics of nonconvulsive seizure Cases

Table II (available at www.jpeds.com) displays characteristics of all nonconvulsive seizure cases. EEG findings in nonconvulsive seizure cases were nonspecific. However, delta activity with rhythmical evolution was the most common finding, appearing in 28 patients (68.3%). Thirty-eight patients were treated with antiepileptic drugs. During the 30-day study period, 33 patients developed no sequelae, 3 developed neurologic sequelae, 1 remained intubated and exhibited refractory hypertonia, 1 developed epilepsy, and 1 developed incomplete upper-right extremity paresis. Two patients among those followed died, 1 from acute encephalopathy caused by influenza B and the other from hypoglycemia. Three patients were lost during the follow-up period.

EEG Analysis by ED Physicians

In 5 of the 41 patients with nonconvulsive seizures, EEGs were analyzed in the ED by a neurologist (**Table II**). Two of these patients developed neurologic complications (epilepsy or refractory hypertonia). The other 36 patients were monitored with EEG, and all cases of nonconvulsive seizures were detected by ED physicians. Thirty-three patients were treated exclusively by an ED physician, and 3 patients were not given antiepileptic drugs because their nonconvulsive seizures disappeared spontaneously. Three of 36 patients developed complications, including 1 who developed neurologic sequelae (incomplete right upper extremity paresis), and 2 who died, as noted above. Of all 242 patients assessed, 13 (5.3%) were diagnosed with nonconvulsive seizures by ED physicians, and a neurologist identified patients without nonconvulsive seizures by retrospective EEG analysis (**Table III**; available at www.jpeds.com). All patients but 1 (who was lost to follow-up) did not develop sequelae. Nonconvulsive seizures were most often mistakenly diagnosed when delta activity was observed (92.3%).

ED physicians did not identify any patients without nonconvulsive seizures who were diagnosed with nonconvulsive seizures by a neurologist.

Characteristics of Patients with and without Nonconvulsive Seizures in the ED

To compare the characteristics of patients with and without nonconvulsive seizures in the ED, we excluded patients with duplicate visits and included only the first visit for 242 patients (36 cases were revisits). Thus, we compared 33 patients with nonconvulsive seizures and 173 patients without nonconvulsive seizures. The demographic data of patients with and without nonconvulsive seizures seen in the ED are summarized in **Table IV**. Age at presentation was significantly older in patients with nonconvulsive seizures than in those without nonconvulsive seizures (median, 68.5 vs 36.1 months), and PCPC scores on initial assessment were significantly higher in patients with nonconvulsive seizures (median, 2.0 vs 1.0). There were no significant differences in the Glasgow Coma Scale score at presentation, clinical presentation, or clinical diagnosis between the 2 groups. EEG assessment, antiepileptic drug administration, and outcomes for the 2 groups are presented in **Table IV**. The interval from initial assessment by an ED physician to the initiation of EEG monitoring was significantly shorter in patients with nonconvulsive seizures than in those without nonconvulsive seizures (median, 15.0 vs 22.0 minutes). In addition, the rate of antiepileptic drug administration before EEG monitoring was lower in patients with nonconvulsive seizures (42.4% vs 64.7%). The proportion of patients admitted for an ICU stay was significantly greater in the group with nonconvulsive seizures (30.3% vs 15.0%); however, hospital admission rates and total length of hospital stay were not statistically significantly different between the groups (87.9% vs 82.7% and 3.0 vs 2.0 days, respectively). Two patients with nonconvulsive seizures and 32 patients without nonconvulsive seizures were lost during the follow-up period. Thus, 31 patients with nonconvulsive seizures and 141 patients without nonconvulsive seizures were ultimately analyzed for short-term outcomes. Neurologic sequelae and mortality rate at 30 days did not differ significantly between the 2 groups (9.7% vs 5.7% and 3.2% vs 0%, respectively).

Discussion

Our data show a 16.9% incidence of nonconvulsive seizures in pediatric patients with altered mental status detected via reduced-lead EEG in the ED, accounting for 4.4% of all patients with altered mental status (Glasgow Coma Scale <15) presenting to our hospital during the study period. The 95% CI was 12.2%-21.6%, implying that reduced-lead EEG monitoring might result in treatment of 1-2 out of 10 patients for nonconvulsive seizures. This estimate is quite high and indicates the usefulness of EEG observation in the ED. Nonconvulsive seizures and nonconvulsive status epilepticus are well-defined entities that produce altered mental status,[23] which is not rare in pediatric cases. Topjian et al reported that electrical seizures were detected in 42% of critically ill children.[3] Abend et al reported further that 59% of pediatric patients in the ICU underwent EEG monitoring, which led to specific clinical interventions, and in a prospective observational study, 46% of critically ill pediatric patients presented with electrographic

Table IV. Demographic data, EEG assessment, antiepileptic drug administration, and outcomes in patients with and without nonconvulsive seizures in the ED

Characteristic	NCS(+) (N = 33)	NCS(–) (N = 173)	*P* value
Age, mo, median (IQR)	68.5 (41.4-94.5)	36.1 (18.9-61.2)	.0002*
Male sex, n (%)	18 (54.5)	91 (52.6)	.8518
PCPC on initial assessment, median (IQR)	2 (1-3)	1 (1-1)	.0005*
Glasgow Coma Scale at presentation, median (IQR)	9 (6-11.5)	8 (6-11)	.4736†
Clinical presentation			
No seizure before ED, n (%)	8 (24.2)	27 (15.6)	.2171
Seizure before ED, n (%)	25 (75.8)	146 (84.4)	.2171
Clinical signs of seizure before ED, n (%)			
Ocular movement abnormalities	1 (4.0)	7 (4.8)	1.00
Partial motor activities in face or extremities	1 (4.0)	11 (7.5)	1.00
Generalized convulsion	23 (92.0)	128 (87.7)	.7417
Total seizure duration, min, median (IQR)	25 (10-46)	35 (10-62)	.5217
Clinical diagnosis, n (%)			
Febrile seizure	22 (66.7)	112 (64.7)	1.00
Epilepsy	5 (15.2)	28 (16.2)	1.00
Encephalopathy/encephalitis	5 (15.2)	13 (7.5)	.1763
Hypoglycemia	1 (3.0)	4 (2.3)	.5862
Psychogenic nonepileptic seizure	0 (0)	9 (5.2)	.3600
V-P shunt dysfunction	0 (0)	1 (0.6)	1.00
Trauma	0 (0)	2 (1.2)	1.00
Gastroenteritis	0 (0)	1 (0.6)	1.00
Hyponatremia	0 (0)	1 (0.6)	1.00
Respiratory failure	0 (0)	1 (0.6)	1.00
Unknown	0 (0)	1 (0.6)	1.00
Neurologist present in ED, n (%)	5 (15.2)	25 (14.5)	1.00
Time from onset of initial neurologic symptoms to EEG monitoring, min, median (IQR)	88.0 (68.0-300.0)	92.0 (63.0-178.0)	.4578
Time from initial assessment by ED physician to EEG monitoring, min, median (IQR)	15.0 (12.0-25.0)	22.0 (15.0- 44.0)	.0308*
AED administration before EEG monitoring, n (%)	14 (42.4)	112 (64.7)	.0196‡
AED administration after EEG monitoring, n (%)	31 (93.9)	13 (7.5)	<.0001‡
Hospital admission, n (%)	29 (87.9)	143 (82.7)	.6114
Hospital length of stay, d, median (IQR)	3 (2-7)	2 (1-5)	.0936
ICU stay, n (%)	10 (30.3)	26 (15.0)	.0451‡
Neurologic sequelae at 30 d, n (%)	3 (9.7)	8 (5.7)	.4195
30-d mortality, n (%)	1 (3.2)	0 (0)	.1802

AED, antiepileptic drug; *NCS*, nonconvulsive seizure.
*The Mann-Whitney *U* test identified significant differences in age, PCPC on initial assessment, and time from the initial assessment by ED physicians to EEG monitoring.
†Two patients in the NCS(+) group and 3 patients in the NCS(-) group with tracheotomy were excluded from Glasgow Coma Scale assessment.
‡The χ^2 test identified significant differences in AED administration before and after EEG monitoring and ICU stay.

seizures.[9,10] Using continuous video EEG for monitoring pediatric patients in ICU, Schreiber et al reported that 18% exhibited nonconvulsive status epilepticus.[11] In the ED setting, Alehan et al reported that 23.8% of nonconvulsive status epilepticus cases (5 of 21 patients) among pediatric patients in the ED were detected by EEG.[24] Thus, our results and previous work report a relatively high percentage of patients with nonconvulsive seizures/nonconvulsive status epilepticus with altered mental status. Therefore, the use of EEG in the ED should contribute to the prompt recognition and treatment of nonconvulsive seizure. In our study, of all 242 patients, 22.3% were actually treated with antiepileptic drugs, as indicated by EEG results.

Several studies have demonstrated the efficacy of monitoring via EEG in the ED. Kothare et al reported that in a cohort of 32 children, 30 (94%) demonstrated emergent EEG results, which were used in the clinical decision making process.[25] Alephan et al reported that 84% of the results of emergent EEG contributed to a further diagnosis in a population of 56 children.[24] Thus, EEG in children with altered mental status in the ED should be considered to promote earlier intervention and treatment.[7] Nonetheless, few studies have reported on the utility of EEG in the ED, in part because the use of EEG in ED requires significant time and may require a trained technician or neurologist to apply 20 or more electrodes. The time taken to obtain a standard EEG in the US has been reported to be approximately 3 hours on average.[26] In addition, only approximately 2% of US EDs are equipped with EEG equipment or have a technologist who can properly apply EEG electrodes.[23] These situations and others may render an EEG unavailable after hours or on weekends in many hospitals, possibly leaving ED physicians in a position where no diagnostic test is available, despite a clinical suspicion of nonconvulsive status epilepticus.

In light of the limitations discussed here, a reduced-lead EEG consisting of only 4 electrodes was introduced to our hospital more than 10 years ago. This simple and easily configured EEG enables ED physicians to rapidly detect nonconvulsive seizures/ nonconvulsive status epilepticus. In the present study, electrode placement was limited to 4 sites, shortening the median time for EEG setup to only 20.5 minutes (with a fastest time of only 4 minutes) after patient arrival in the ED. The

efficacy of reduced-lead EEG was also reported by Brenner et al in their comparison of a 23-lead EEG and reduced-montage EEG, similar to that used in the present study, in adults admitted to the ED. They reported similar findings for the 2 EEG modalities.[27] Young et al reported sensitivity and specificity of 68% and 98%, for 4-channel recordings.[28] These results support the use of reduced-lead EEG in the ED.

Although the studies noted here have reported the utility of EEG in the ED, none has associated nonconvulsive seizure/nonconvulsive status epilepticus diagnosed by EEG with clinical outcomes. Nonconvulsive status epilepticus is associated with mortality and poor outcomes in critically ill children. Topjian et al reported an association between nonconvulsive status epilepticus and an increased risk of mortality (OR, 5.1) and worse PCPC scores (OR, 17.3).[3] Therefore, prompt performance of EEG to diagnose nonconvulsive seizures in the ED is a reasonable clinical decision. Our study went further to compare the detection of nonconvulsive seizures and outcomes, and found no differences in the rates of any neurologic sequelae or mortality at 30 days between patients with and without nonconvulsive seizures. Moreover, although 57.6% of patients with nonconvulsive seizures did not receive any antiepileptic drugs before EEG monitoring, all patients with nonconvulsive seizures who needed antiepileptic drugs were treated, at a median interval of 15 minutes. This suggests that the early treatment of nonconvulsive seizures in the ED might contribute to improved outcomes in surviving patients. Further prospective studies are needed, however.

The common clinical features of nonconvulsive status epilepticus have been previously documented and include agitation, lethargy, confusion or delirium, and abnormal movements.[24,29] In the present study, more than 80% of the children monitored with reduced-lead EEG had a previous history of seizures. The most common reason for pursuing EEG monitoring was a prolonged disturbance in consciousness, and additional symptoms in monitored patients included muscle weakness, apnea, hypertonia, nystagmus, and changes in vital signs, such as tachycardia (data not shown). These symptoms were not exclusive to patients with nonconvulsive seizures, however; those without nonconvulsive seizures also had similar symptoms. In addition, clinical features and clinical diagnoses did not distinguish patients with and without nonconvulsive seizures. Therefore, the accurate identification of nonconvulsive seizures requires EEG monitoring.

In the present study, antiepileptic drug therapy was started in all patients with nonconvulsive seizures detected by reduced-lead EEG assessed by ED physicians. However, 5.4% of patients without nonconvulsive seizures were also treated in this way by ED physicians. Intermittent or irregular delta activity was most often mistaken as indicating nonconvulsive seizures (92.3%), possibly because these activities in deep sleep are difficult to distinguish from brain dysfunction or damage. However, consultation with a neurologist in these cases takes time and delays antiepileptic drug administration. In our study, all but 1 patient without nonconvulsive seizures treated with antiepileptic drugs (lost to follow-up) did not develop neurologic sequelae. Of note, our data show only a 5.4% false-positive rate and a 0% false-negative rate, with no adverse events following treatment for the false-positive events. Therefore, ED physicians should consider using reduced-lead EEGs to quickly establish antiepileptic drug treatment.

We have presented a series establishing the value of reduced-lead EEG and estimating the proportion of nonconvulsive seizures in the pediatric population presenting with altered mental status to the ED, as well as the characteristics and short-term outcomes of nonconvulsive seizures. This study has some limitations, however. The most important limitation is that EEG application was not based on a specific protocol, but was rather at the ED physician's discretion. Therefore, a selection bias could exist, and the rates of nonconvulsive seizures found in our study groups are not representative of likely nonconvulsive seizures in all pediatric patients with altered mental status encountered in the ED. Indeed, >80% of the children undergoing reduced-lead EEG had a history of seizures, often febrile. Second, because of the simplicity of the EEG monitoring system used in this study, it is possible that EEG abnormalities were missed, especially in unmonitored areas of the brain. Third, the EEGs used here only recorded tens of minutes of brain activity. It is not known whether such a short period of EEG monitoring is sufficient, although Bauista et al reported that 5 minutes of recording in the ED was adequate for monitoring patients with altered mental status.[30] Therefore, additional studies are required to confirm whether longer periods of continuous EEG monitoring are required in the ED. An additional limitation is that we evaluated the results of EEG only in the ED, and thus nonconvulsive seizures occurring after hospitalization were not assessed. Finally, we were unable to assess the long-term outcomes in our patient sample. Despite these limitations, our study might lead to improved ED care by supporting the use of reduced-lead EEG.

In conclusion, the present study demonstrates a relatively high incidence of nonconvulsive seizures among pediatric patients with altered mental status presenting to the ED. The reduced-EEG method used is easily administered by ED physicians, even in the absence of a neurologist, and could aid in the assessment and treatment of nonconvulsive seizures. Based on our findings, reduced-lead EEG in the ED may enable the prompt recognition and treatment of nonconvulsive seizures in pediatric patients with altered mental status, especially in those children with a history of seizures, often febrile. Thus, the results of this study may stimulate additional research into the use of reduced-lead EEG in multiple clinical contexts. ■

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Table II. Characteristics of patients with nonconvulsive seizures (N = 41)

Patient	Age, mo	Clinical diagnosis	Initial neurologic symptom	Total duration of preceding clinical seizure, min	Time from the onset of initial neurologic symptom to EEG monitoring, min	Time from initial assessment by ED physician to EEG monitoring, min	Time from the onset of initial neurologic symptom to AED administration, min	Time from AED administration according to EEG findings to electrical seizure remission, min	EEG findings*	AEDs after EEG	Neurologist in ED	Outcome on day 30
1	141.6	Febrile seizure	Generalized convulsion	5	88	39	92	<1	Bilateral frontal 1- to 2-Hz rhythmic delta activity	DZP	No	No deficit
2	60.0	Febrile seizure	Left hemiconvulsion	30	65	14	94	3	Bilateral frontal 2-Hz rhythmic delta activity	DZP	No	No deficit
3	209.7	Febrile seizure	Generalized convulsion	89	144	15	149	4	Diffuse polyspikes	MDL, fPHT	No	No deficit
4	22.8	Febrile seizure	Generalized convulsion	15	68	8	70	4	Bilateral frontal 2- to 3-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
5	94.5	Febrile seizure	Generalized convulsion	22	470	23	476	6	Diffuse 3-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
6	43.8	Febrile seizure	Generalized convulsion	67	81	34	82	75	Diffuse 1-Hz rhythmic delta activity	MDL, fPHT	Yes	No deficit
7	51.1	Febrile seizure	Generalized convulsion	45	88	14	90	<1	Diffuse 2-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
8	97.8	Febrile seizure	Generalized convulsion	23	42	12	55	<1	Bilateral frontal 1- to 2-Hz rhythmic delta activity	MDL	No	No deficit
9	48.9	Febrile seizure	Generalized convulsion	10	68	14	72	27	Bilateral frontal 1-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
10	103.4	Febrile seizure	Generalized convulsion	45	87	25	95	13	Bilateral frontal 2-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
11	35.6	Febrile seizure	Generalized convulsion	2	110	25	118	15	Diffuse 4-to 5-Hz rhythmic theta activity	MDL, fPHT	No	No deficit
12	80.2	Febrile seizure	Disturbed consciousness	—	54	20	60	4	Bilateral frontal 2-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
13	82.5	Febrile seizure	Disturbed consciousness	—	515	128	582	2	Bilateral frontal 3-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
14	99.7	Febrile seizure	Generalized convulsion	25	51	11	58	13	Bilateral frontal 1-Hz rhythmic delta activity	MDL	No	No deficit
15	79.0	Febrile seizure	Generalized convulsion	13	78	73	120	<1	Right hemisphere 2-Hz spike and waves	MDL	No	No deficit
16	33.7	Febrile seizure	Generalized convulsion	1	254	24	260	<1	Bilateral frontal 2- to 3-Hz rhythmic delta activity	MDL	No	No deficit
17	43.8	Febrile seizure	Generalized convulsion	30	120	10	122	19	Left occipital 1-Hz rhythmic delta activity	MDL	No	No deficit
18	31.7	Febrile seizure	Generalized convulsion	46	60	12	72	15	Diffuse 2-to 3-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
19	38.0	Febrile seizure	Generalized convulsion	144	410	24	418	2	Diffuse 2-to 3-Hz rhythmic delta activity	MDL	No	No deficit
20	48.3	Epilepsy	Disturbed consciousness	—	80	23	81	4	Diffuse frontal dominant 2-Hz rhythmic delta activity	MDL, fPHT	No	No deficit

(*continued*)

Table II. Continued

Patient	Age, mo	Clinical diagnosis	Initial neurologic symptom	Total duration of preceding clinical seizure, min	Time from the onset of initial neurologic symptom to EEG monitoring, min	Time from initial assessment by ED physician to EEG monitoring, min	Time from the onset of initial neurologic symptom to AED administration, min	Time from AED administration according to EEG findings to electrical seizure remission, min	EEG findings*	AEDs after EEG	Neurologist in ED	Outcome on day 30
21	46.7	Epilepsy	Disturbed consciousness	15	87	7	89	2	Diffuse polyspikes and waves	MDL	No	No deficit
22	38.4	Epilepsy	Right upper extremity convulsion	65	73	27	—	—	Left hemisphere polyspikes	None	No	No deficit
23	184.0	Epilepsy	Disturbed consciousness	—	300	42	353	1	Bilateral frontal sharp waves	MDL	No	No deficit
24	74.6	Epilepsy	Disturbed consciousness	35	82	6	91	<1	Right frontal 2-Hz spike and waves	MDL	Yes	No deficit
25	44.8	Epilepsy	Generalized convulsion	50	57	10	64	15	Left hemisphere 1-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
26	47.4	Epilepsy	Disturbed consciousness	74	75	12	79	18	Left hemisphere 1-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
27	59.8	Epilepsy	Generalized convulsion	20	72	24	89	11	Diffuse 2-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
28	41.4	Epilepsy	Generalized convulsion	2	380	12	420	2	Bilateral frontal 4- to 5-Hz rhythmic theta activity	MDL	No	No deficit
29	101.3	Epilepsy	Generalized convulsion	6	69	14	70	<1	Right hemisphere 1-Hz spike and waves	MDL, fPHT	Yes	No deficit
30	68.5	Epilepsy	Disturbed consciousness	—	38	8	—	—	Left frontal 1-Hz rhythmic delta activity	None	No	No deficit
31	142.0	Meningitis	Disturbed consciousness	—	358	13	914	24	Diffuse 3-Hz rhythmic delta activity	MDL, fPHT	No	No deficit
32	37.0	Acute encephalopathy	Disturbed consciousness	—	329	24	330	3	Diffuse 2-Hz rhythmic delta activity	MDL	No	No deficit
33	73.2	Acute encephalopathy	Disturbed consciousness	—	170	5	177	21	Bilateral frontal sharp waves	MDL, fPHT	No	No deficit
34	44.8	Acute encephalopathy	Generalized convulsion	640	839	15	844	82	Left occipital sharp waves	MDL, thi	Yes	Intubated, hypertonia
35	8.2	Acute encephalopathy	Generalized convulsion	267	274	205	275	22	Right hemisphere 1-Hz spike and waves	MDL, thi	Yes	Epilepsy
36	33.6	Febrile seizure	Generalized convulsion	65	75	7	79	31	Left hemisphere 2-Hz spike and waves	MDL, fPHT	No	Incomplete right upper extremity paresis
37	48.0	Hypoglycemia	Disturbed consciousness	—	350	110	—	—	Right occipital 1-to 2-Hz rhythmic delta activity	None	No	Death
38	71.2	Acute encephalopathy	Disturbed consciousness	93	100	25	102	9	Diffuse 2-to 3-Hz rhythmical delta activity	MDL, fPHT	No	Death
39	85.5	Febrile seizure	Generalized convulsion	3	63	13	78	4	Bilateral frontal 2-Hz rhythmic delta activity	MDL	No	Lost to follow up
40	80.4	Febrile seizure	Generalized convulsion	20	69	17	70	15	Bilateral frontal 1-Hz rhythmic delta activity	MDL	No	Lost to follow-up
41	54.3	Febrile seizure	Generalized convulsion	7	50	15	55	31	Bilateral frontal 1- to 2-Hz rhythmic delta activity	MDL	No	Lost to follow-up

AED, antiepileptic drug; *DZP*, diazepam; *fPHT*, fos-phenytoin; *MDL*, midazolam; *thi*, thiamylal.
Convulsive seizure and a sequence of intermittent seizures are included.
*All rhythmic activity had the evolution to meet criteria for seizure.

Table III. Characteristics of patients without nonconvulsive seizures administered antiepileptic drugs by ED physicians

Patient	Age, mo	Clinical diagnosis	EEG findings	AEDs after EEG monitoring	Outcome on day 30
1	43.5	Acute encephalopathy	Right hemisphere irregular 1- to 2-Hz delta activity	fPHT, Thi	No deficit
2	45.4	Acute encephalopathy	Left occipital 1- to 2-Hz delta activity	MDL	No deficit
3	51.9	Febrile seizure	Diffuse irregular 2- to 3-Hz delta activity	DZP	No deficit
4	22.9	Febrile seizure	Bilateral frontal intermittent 1-Hz delta activity	MDL	No deficit
5	17.1	Febrile seizure	Left occipital intermittent 1-Hz delta activity	MDL	No deficit
6	29.6	Febrile seizure	Bilateral frontal irregular 1-Hz delta activity	fPHT, Thi, PB	No deficit
7	46.1	Febrile seizure	Diffuse irregular 1- to 2-Hz delta activity	MDL	No deficit
8	15.9	Febrile seizure	Left occipital intermittent 1-Hz delta activity	DZP	No deficit
9	18.9	Febrile seizure	Bilateral frontal irregular 1-Hz delta activity	MDL, fPHT	No deficit
10	22.2	Febrile seizure	Diffuse irregular 1- to 3-Hz delta activity	MDL, fPHT	No deficit
11	109.4	Febrile seizure	Intermittent left hemisphere 1- to 2-Hz delta activity	MDL	No deficit
12	53.4	Respiratory failure	Diffuse periodic sharp waves	MDL	No deficit
13	44.9	Febrile seizure	Left hemisphere frontal dominant irregular 1- to 2-Hz delta activity	MDL	Lost to follow-up

PB, phenobarbital.