Status Epilepticus in the PICU: Quieting the Storm

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Abstract Status epilepticus (SE), defined by either a prolonged seizure or a prolonged period of recurrent seizures without a return to baseline in-between seizures, is a neurological emergency frequently encountered in the pediatric intensive care unit (PICU). Pediatric SE, in addition to itself leading to PICU admission, occurs commonly in critically ill children. The clinical manifestations of SE are increasingly being recognized as presenting in a widely heterogeneous manner, particularly in the PICU. Subtler presentations can include altered mental status with or without suggestive motor movements. Thus, the initial fundamental task in managing pediatric SE is astute recognition and diagnosis, which often requires both clinical acumen and electroencephalography. This, in turn, allows for prompt management with concurrent evaluation and treatment. In this article, we will review the diagnosis, evaluation, and treatment of SE occurring in the setting of the PICU, with a focus on bringing the reader up-to-date on these topics.

Keywords Electroencephalography · Status epilepticus · Intensive care units · Pediatric

Introduction

Status epilepticus (SE) is a neurological emergency defined by either a prolonged seizure or a prolonged period of recurrent seizures without a return to baseline in-between seizures. Pediatric SE, in addition to itself being a neurological emergency leading to admission and requiring intensive care, occurs commonly in critically ill children, and is therefore frequently encountered in the pediatric intensive care unit (PICU) [1]. Although the analogy of a “storm” may invoke the visual scenario of a child in a continuous generalized tonic–clonic seizure, the clinical manifestations of SE are increasingly becoming recognized to present in a widely heterogeneous manner, particularly in the PICU. Subtler presentations of SE in the PICU, due in part to the common use of sedative and neuromuscular-blocking agents in the setting of critically ill children, can include altered mental status with or without suggestive motor movements. Thus, the initial fundamental task in managing pediatric SE—“quieting the storm”—is astute recognition and diagnosis, which in the PICU often requires both clinical acumen and neurophysiological monitoring with electroencephalography (EEG). This, in turn, allows for prompt management with concurrent evaluation and treatment.

Defining Pediatric Status Epilepticus

The classical definition of SE from the International League Against Epilepsy (ILAE) [2] establishes a cut-off of a duration greater than 30 min. This temporally based definition was influenced by evidence in animal studies establishing the relationship between prolonged seizures and neuronal injury [3]. The onset of seizures, however, is

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not always directly witnessed, which often affects the precision of this definition. Furthermore, advocacy for earlier aggressive treatment has resulted in an evolving definition of SE.

Shorter durations of seizures, 5–10 min, have subsequently been advocated to be the defining standards for SE. The reasons for this are that the vast majority (>90 %) of seizures last under 2 min [4], and seizures lasting over 5–10 min are unlikely to stop without medical intervention [5]. Furthermore, the need for urgency in treatment is supported by data in adults that establishes a lower initial response rate in patients for whom treatment of SE is delayed [6]. Thus, in clinical practice, using shorter durations for defining SE frames an implied sense of urgency for more aggressive treatment. The first 10–30 min can also be referred to as early or impending SE, and 30 min as established SE. The importance of classifying seizures lasting greater than 5 min as SE is to underscore the importance of early aggressive treatment [7].

Diagnosis of Status Epilepticus

Within the PICU, children with SE generally present in one of the following scenarios: either a patient is diagnosed with SE and is being admitted to the PICU for management, or a patient admitted for an alternative clinical diagnosis develops or is diagnosed with SE while in the PICU. The recognition of SE is complicated by its varied semiological presentations. SE can occur for all seizure semiologies as defined by the ILAE [8], for example, simple complex partial SE or complex partial SE, and for generalized seizures as tonic, clonic, tonic–clonic, or myoclonic SE. In such cases, the diagnosis of SE can often be made clinically. It is also important to realize that intermittent seizures without a return to baseline in-between is also SE, as under-recognition often results in delays in medical care [9].

The use of continuous EEG monitoring is now common in intensive care units [10] and complements clinical acumen to diagnose nonconvulsive seizures (NCS) and nonconvulsive status epilepticus (NCSE). With the more common use of continuous EEG, it has been increasingly recognized that pediatric SE can present with a wide range of manifestations, from coma to milder, more intermittent presentations of altered mental status, with or without obvious motor manifestations.

Several recent studies have evaluated for the prevalence of NCS or NCSE among critically ill children using EEG (Table 1). The majority of these studies are retrospective, and have found either NCS or NCSE in 16–39 % of patients, and NCSE in 0–35 % of patients [11–17]. In addition, there have been three recent prospective studies, for which either NCS or NCSE was diagnosed in critically ill children using

Table 1 Nonconvulsive seizures (NCS) or nonconvulsive status epilepticus (NCSE) in critically ill children

NCS or NCSE
Retrospective data: 16–39 % [11, 15–17]
Prospective data: 7–46 % [18, 19, 20•]
NCSE
Retrospective data: 0–35 % [11–15, 17]
Prospective data: 1–19 % [18, 19, 20•]
Clinical or electrographic risk factors for NCS or NCSE:
Younger age (<24 months) [12, 17, 19, 20•]
Clinical seizure prior to EEG [11, 14, 16–19]
Epileptiform discharges on EEG [11, 16, 17]
TBI [17]
Duration of EEG to detect NCS or NCSE:
Within the first 30–60 min: 43–71 % [11, 17–19, 20•]
Within the first 24 h: 80–100 % [11, 17–19, 20•]

EEG electroencephalogram, TBI traumatic brain injury

EEG in 7 % [18], 30 % [19], or 46 % [20•], with NCSE diagnosed in 1 [18], 18 [19], and 19 [20•]. Some possible explanations for these wide variations in reported ranges include differences in ICU populations and ascertainment of EEG monitoring. For example, 90 % of the patients in the series from Abend et al. [20•] had primary brain disorders, whereas about half of the patients in the series from Shahwan et al. [18] had primary brain disorders. In terms of ascertainment of EEG monitoring, Schreiber et al. [19] used inclusion criteria of a Glasgow coma scale <12 not explained by medication or baseline chronic encephalopathy, whereas Abend et al. [20•] used differing criteria: persisting altered mental status after a convulsion, or altered mental status of unclear etiology or “disproportionate to the known medical condition”, and the clinical suggestion of seizures (vital sign changes). In the series from Saengpatrachai, 16 % were diagnosed with NCS, while no patients were diagnosed with NCSE; however, in this series continuous long-term EEG was not utilized [15]. Despite these wide ranges, it can be summarized that critically ill children are at risk to have NCS and NCSE.

Some reported risk factors for NCS or NCSE include younger age (<2 years) [12, 17, 19, 20•], and a clinical seizure prior to EEG monitoring [11, 14, 16–19]. In the series from Abend et al. [20•] for every 1-year increase in age, there was a 7 % decrease in the odds of seizure occurrence. Traumatic brain injury was identified as an independent risk factor in the series from Williams et al. [17]. In addition, in a series looking specifically at children treated with hypothermia after cardiac arrest, NCS were diagnosed with continuous EEG in 47, and 32 % with NCSE [21]. It should also be noted that NCS and NCSE can also occur in other inpatient units outside of the PICU in children with altered mental status, which may require

transfer to the PICU for management. In a recent study more than half of the patients with altered mental status who were found to be in NCSE were not critically ill [22•].

As predictors for differentiating between ill patients with NCS versus those in NCSE have not been identified, continuous EEG is needed when either NCS or NCSE are suspected. In terms of duration of EEG monitoring, one must balance the yield of diagnosing NCS or NCSE versus the resources available to support continuous EEG. The first NCS is noted within the first 30–60 min of monitoring in 43–71 % and within the first 24 h in 80–100 % [11, 17–19, 20•]. Thus extension of continuous EEG monitoring to at least 24 h improves the yield. Finally, although rare in children, the possibility of psychogenic non-epileptic seizures (PNES) resembling convulsive SE has been reported [23]. Diagnosing PNES requires EEG as part of the clinical evaluation and should be utilized in patients with an atypical history or clinical course. Continuous EEG often alters treatment [24] and is the only reliable way to diagnose NCSE in critically ill children with altered mental status. Continuous EEG and child neurology consultation should be strongly considered for patients with suspected NCS or NCSE. Once a child has been identified to be in SE, concurrent evaluation and pharmacological treatment should commence.

Evaluation of Status Epilepticus

It is important to understand the etiologies of pediatric SE, as this influences the clinical evaluation. The etiology of pediatric SE is classified most commonly into the following categories:

- Febrile SE is provoked by fever in the absence of a central nervous system infection, and typically occurs within the age range of 6 months to 5 years.
- Acute symptomatic SE occurs in the setting of an acute provoking factor, excluding prolonged febrile convulsions. Examples include central nervous system infections, trauma, and stroke.
- Remote symptomatic SE occurs in patients with a prior neurological abnormality without an acute provoking factor. Examples include cerebral dysgenesis and remote brain injury.
- Cryptogenic SE occurs in the absence of a definable cause.
- Idiopathic SE occurs in patients with an idiopathic genetic predisposition for epilepsy.
- SE due to progressive encephalopathy occurs in neurodegenerative disorders.

In prior retrospective studies evaluating for etiologies of pediatric SE [25, 26], the most common etiology is febrile

SE, occurring in about a third of patients. This is followed by remote symptomatic (12–28 %), acute symptomatic (17–18 %), with the remainder classified as either idiopathic or cryptogenic (11–12 %), or progressive neurological (4 %) [25, 26]. The age of the patient influences the etiology, as younger children are more prone to present with febrile SE [27] and due to acute symptomatic etiologies, in contrast to older children who tend to present in SE from cryptogenic or remote symptomatic etiologies [28].

About 10 % of children presenting in SE have no history of seizures [5, 29•]. A recent study from Singh et al. [29•] specifically evaluated the scenario in which a child who has no history of seizures presents in SE, using prospectively obtained data. Singh et al. found that one-third of these cases were due to febrile SE, but cryptogenic SE made up the majority of cases, at 42 %, with remote symptomatic at 18 %, acute symptomatic at 16 %, and idiopathic at 4 % of cases. [29•] Although the authors defined SE at 20 min and also excluded patients with prolonged periods of intermittent seizures without return to baseline, these exclusions probably do not alter the clinical utility of their data.

Thus, based upon known classifications and using age-related trends as guidance, an evidence-based evaluation can proceed for the child in SE. In 2006, the quality standards subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society released a practice parameter to guide the diagnostic assessment of the child with SE, excluding febrile SE [30]. Their review of relevant literature found that lab abnormalities (sodium, glucose, calcium) were present in 6 %, low antiepileptic drug (AED) levels in 32 % (although not necessarily the cause of SE), toxicological evidence of ingestion in 3.6 %, evidence for an inborn error of metabolism (IEM) in 4.2 %, and neuroimaging abnormalities were found in 8 % [30]. It is notable, however, in the series from Singh et al. using more recent data including high quality magnetic resonance imaging (MRI), that imaging yielded a diagnosis in 30 %, and directed acute management in 24 % [29•].

For a patient *without* a known history of epilepsy, evaluation should include basic serum chemistries, including bedside glucose determination and a comprehensive electrolyte panel. Urgent neuroimaging should be performed, preferably MRI, but if unavailable, then with computed tomography. Toxicology and evaluation for IEM is based upon clinical suspicion. However, for patients *with* known epilepsy who are presenting in SE, repeat neuroimaging may not be needed, unless there are atypical features to the clinical presentation. However, routine electrolyte and AED levels are reasonable.

When a previously normal child in SE has a concurrent fever, the first task is to evaluate and treat for the potential of a primary central nervous system (CNS) infection. If the

patient proves to not have a CNS infection with a complete evaluation failing to reveal another cause, and they are between 6 months and 5 years old, they fall under the classification of febrile SE. Any febrile illness can provoke a child to have a prolonged febrile convulsion. However, human herpes viruses (HHV) may be an important cause. In a recently published prospective study, of 169 children with febrile status epilepticus, 32 % had serologic evidence of HHV-6 and 7.1 % had serologic evidence of HHV-7 infection [31]. It is not known if high fever caused by these infections or other factors from the virus may cause the SE in susceptible patients, but HHV-6 and HHV-7 appear to have a prominent role for febrile SE in children.

Another special clinical circumstance is SE in the young infant. Consideration of underlying treatable metabolic diseases and vitamin deficiencies should prompt vitamin trials with concurrent EEG, which can be both diagnostic and also therapeutic. Although rare, the early treatment and recognition of pyridoxine dependency, pyridoxal 5'-phosphate dependency, and folinic-acid-dependent seizures are critical for their successful outcome. A proposed algorithm can consist sequentially of pyridoxine 100 mg IV, then (if needed) pyridoxal 5-phosphate (10 mg/kg/dose $\times$ 2, 2 h apart), and then folinic acid (5 mg $\times$ 2, 6 h apart) [32•].

Treatment of Status Epilepticus

The successful treatment of SE in the PICU begins prior to patient arrival, by both having in place and being familiar with an established scientifically based and practical treatment plan [33]. Having this plan as a protocol promotes the intensive care team's ability to stay one step ahead in the preparation and administration of medications in order to urgently abort SE. This sense of urgency is justified and appropriate, as the time between seizure onset and first-line treatment initiation is inversely correlated with treatment response for both adults [6] and also in children [34]. Delays in initiation of treatment past 30 min have been shown to be particularly detrimental, although in the PICU, the majority of patients have received treatment prior to their arrival. There is evidence to suggest that the underlying pathophysiology of this is due, at least in part, to decreased sensitivity to gamma-aminobutyric acid (GABA)-A with time due to the down-regulation and internalization of GABA receptors [35].

Furthermore, during the entire continuum of care for the patient with SE, the basics of systemic medical care should be attended to, starting with the maintenance of the patient's airway and breathing, and support of the circulation to include blood pressure. The need for intravenous volume support, the need for antibiotics or antiviral

medications for those with suspected infection, and the need for stress steroids for those at risk for adrenal insufficiency should all be assessed simultaneously.

How and What to Treat SE

Although Class I evidence for pediatric SE is scarce, clinical practice is generally extrapolated from adult Class I data, to include the superiority of lorazepam and diazepam over placebo [36]. The treatment options for pediatric SE are listed in Table 2, which assumes that by the time a patient is in the PICU, intravenous (IV) access has already been established. Furthermore, when patients are transferred into the PICU for treatment of SE, they may have already been treated with first-line medications either in the community or from another inpatient unit, as community treatment has been shown to decrease seizure duration and decrease seizure recurrence [37]. Patients should be quickly assessed as to where along the treatment plan they have progressed, with the next available treatments given as urgently as possible for those continuing to convulse.

Early or Impending SE, First-line

In the initial stages of SE (5–10 min), first-line therapy includes benzodiazepines, which primarily act as GABA receptor agonists. In particular, those benzodiazepines that can be given in an IV formulation to include lorazepam, diazepam, and midazolam—are preferred for the treatment of SE. However, if IV access is difficult to obtain, alternative methods of administration can be used: for example, intramuscularly, intranasally, or rectally. Due to its longer anticonvulsant effect, lorazepam (12–24 h) is preferred over diazepam and midazolam.

However, if a patient is on chronic maintenance AED for which an IV form is available and for which a recent serum level is available, then this agent may be used to give a partial bolus to increase serum levels as part of first-line therapy. Examples of anticonvulsants in which an IV form is available with serum levels that can be obtained rapidly are phenytoin and valproate. This scenario can also be considered for those patients in which a history can be obtained of missed dosing or admitted noncompliance.

In addition, some children with medically refractory epilepsy may have a vagal nerve stimulator (VNS), for which a magnet can be used to give an additional stimulation. A patient's magnet is often located on their wrist, or attached to a personal item, such as a backpack or wheelchair. When available, the magnet can be swiped slowly once across the VNS which is normally located in the subcutaneous tissue of the left upper chest. As most VNS's are programmed to stimulate for 60 s with a single swipe, quick repetitive swipes are not necessary, and furthermore

Table 2 Treatment options for pediatric status epilepticus (SE)

Timing	Treatment	Suggested dosing
Early or impending SE		
First-line	Lorazepam	0.05–0.1 mg/kg IV, repeat $\times$ 1
	Diazepam	0.2–0.5 mg/kg IV, repeat $\times$ 1
	If VNS, swipe with magnet	
	Fosphenytoin	20–25 PE/kg IV
	Phenobarbital	20 mg/kg IV
Second-line	Levetiracetam	20–30 mg/kg IV
	Valproate	20 mg/kg IV
Refractory SE		
First-line	Midazolam	Load: 0.1–0.3 mg/kg IV, maintenance: 0.05–0.4 mg/kg/h IV
	Pentobarbital	Load: 2–10 mg/kg IV, maintenance: 1–5 mg/kg/h IV
	Phenobarbital	“High dose”, see text
	Lacosamide	Uncertain pediatric dosing, see text
Second-line	Propofol	Load: 2 mg/kg IV, maintenance: 2–5 mg/kg/h
	Topiramate	Load: 5–10 mg/kg bolus NG, maintenance 5–15 mg/kg day NG
	Other	
	Electroconvulsive therapy	
	Hypothermia	
	Ketogenic diet	
	Immunomodulation	
	Surgery	
	Vitamin trials	

holding the magnet over the VNS is also not recommended, as this will turn the VNS generator off.

Early or Impending SE, Second-line

If first-line therapies are not successful, then an anticonvulsant with IV formulation for longer-term therapy should be initiated. Fosphenytoin, which acts primarily upon the sodium channel, is used most often in children. It is dosed in phenytoin equivalents (PE), and is usually loaded at 20–25 PE/kg IV, without a maximum dosing. In a recent randomized controlled trial in children, a diazepam–phenytoin combination was just as successful in aborting SE as lorazepam [38]. Although phenytoin is often used in adult SE, in children the prodrug fosphenytoin is preferred. Fosphenytoin administration can be performed at a faster rate than phenytoin (fosphenytoin 150 PE/min versus phenytoin 25–50 mg/min), and fosphenytoin comparatively has lower risks of local caustic effects on tissue, and less risks for hypotension and cardiac dysrhythmias.

Other second-line options include phenobarbital, levetiracetam, and valproate. Phenobarbital is a barbiturate that can be dosed at 20 mg/kg IV, or in increments of 5–10 mg/kg to total 20 mg/kg to start. It is important to be

ready to secure a patient’s airway when using higher doses of phenobarbital, especially when used following benzodiazepine administration. However, with a secure airway, higher serum levels can be achieved.

Levetiracetam, which has an uncertain mechanism of action, can be loaded at 20–30 mg/kg IV, and has the advantage of minimal drug–drug reactions and minimal liver metabolism. However, the evidence supporting its use in pediatric SE is confined to case reports and case series. Levetiracetam has been reported to have efficacy in cases of refractory SE [39–41] and also in cases of NCSE [39, 42, 43]. However, in some series, levetiracetam is reported to have poor efficacy for SE [44].

Valproate, which has multiple mechanisms of action, can be loaded at 20 mg/kg. Caution should be practiced in patients with liver disease, and in younger patients (<2-year-old) especially those with an undiagnosed etiology for their developmental delay. Valproate can exacerbate illness in patients with certain abnormalities in energy metabolism such as mitochondrial dysfunction. Retrospective series have found IV valproate to be safe and effective for SE of a wide spectrum of seizure types [45, 46], and a randomized controlled trial in children found valproate to be as effective as diazepam (80 % for valproate vs. 85 % for diazepam), but with less side effects (none for valproate

versus need for mechanical ventilation in 60 % and hypotension in 50 % for diazepam) [47].

Refractory SE

Refractory status epilepticus (RSE) occurs when initial attempts to abort SE fail. RSE can be defined as SE lasting over 60 min, which occurs in about 30 % of pediatric SE [29, 34]. However, clinically, RSE can be defined as SE not responding to at least two anticonvulsant medications. When using a suppressive agent, such as versed or pentobarbital, EEG should be utilized to monitor medication effectiveness with the goal of EEG burst-suppression. In addition, patients at this stage are at least partly encephalopathic, and NCS and NCSE may be difficult to differentiate clinically from a post-ictal state without EEG support.

Refractory SE, First-line

The most commonly used medications for RSE are continuous infusions of either midazolam or pentobarbital [48, 49]. Although both can be used effectively [50], pentobarbital has higher rates of adverse effects including hypotension [51]. High-dose phenobarbital can also be used with case series reporting median serum levels of 144 [52] and daily dosing of up to 80 mg/kg [53].

Refractory SE, Second-line

Second-line medications for RSE include lacosamide, propofol, and topiramate. Lacosamide enhances the slow activation of sodium channels, and has an IV preparation; however its use and dosing in pediatric SE is not established. A dosing of 25 mg twice a day has been used to successfully treat an 8-year-old boy with RSE [54]. Topiramate, which has multiple proposed mechanisms of action, does not come in an IV preparation, and thus if used in pediatric SE, is administered via nasogastric (NG) tube. Evidence in pediatric RSE consists of case reports and series, and includes loading and maintenance dosing regimens ranging from 5–10 mg/kg day [55–58]. Continuous propofol infusions, although not uncommonly used in adult SE, are used less in children due to the higher risks of propofol-infusion syndrome in children. Safe and effective use of propofol for RSE has been reported in children [59], although caution and close monitoring should still be used while avoiding higher dosing (over 5 mg/kg/h), and using for the shortest duration possible.

Other Options

Other treatment options for RSE, for which evidence consists mostly of case reports, that can be considered

when initial treatments are ineffective include the ketogenic diet (KD) [60], moderate hypothermia [61], immunomodulation, and electroconvulsive therapy [62]. The KD is a high-fat, low-carbohydrate, adequate protein diet that can be administered as a liquid via NG tube. In a recently published case series of five RSE patients initiated on the KD, after 1 month two patients became seizure-free and the remaining three patients had significant decreases in seizure activity, allowing tapering of other anticonvulsant medications and weaning from prolonged ventilation [60]. Immunomodulation can consist of steroid therapy or intravenous immunoglobulins, and is typically reserved for specific immune-mediated etiologies.

Finally, when medical therapy fails, epilepsy surgery may be considered for carefully chosen candidates. Candidates can include those with focal cortical dysplasia or patients with lateralized lesions, such as Rasmussen's encephalitis [63–65]. In addition, implantation of a vagal nerve stimulator has been reported in the preceding days prior to EEG normalization and being able to wean from suppressive medications in a 6-year-old with RSE [66].

Prognosis

The goal of urgently treated SE is improved prognosis. In aggressively treated SE, mortality can be low, ranging from 0–9 % [25, 26, 67, 68], but prognosis is highly dependent upon the etiology [67, 68], with deaths occurring mostly within acute symptomatic and progressive neurological etiologies. Morbidity in aggressively treated SE is also low with new neurological deficits occurring in 1–13 % [25, 67, 68]. Increased morbidity can be associated with increased duration of seizures [67], however in some series this is only for acute symptomatic etiologies [68]. This is consistent with high rates of mortality (30 %) [69], and resultant epilepsy (100 %) [70], in some series of severe prolonged pediatric RSE [71], especially for acute symptomatic etiologies. Overall 11–16 % at 1 year and 18 % at 2 years will have a recurrence of SE [26, 72], but recurring especially in those patients who were not neurologically normal at baseline [72].

Morbidity and mortality, however, are especially rare for febrile SE, with no deaths and no new neurological deficits among a series of 180 patients [73]. However, in a recent study of prospectively obtained data on 199 children with febrile SE, 11.5 % of patients had MRI findings of abnormal increased T2 signal in one of the hippocampi, which was not present in the controls [74]. This finding had been reported previously [75], but is now substantiated in a prospective study. Thus it does appear that prolonged febrile seizures can cause focal neuronal injury. However, it is not clear whether these MRI findings are purely due to

the febrile SE, or whether there was a preexisting risk of injury, as developmental abnormalities, mainly hippocampal malrotation, was noted in 10.5 % of patients with febrile SE versus 2.1 % in the simple febrile seizure control group. The clinical relevance of these findings will hopefully be elucidated with long-term follow-up of these patients; however this data is supportive of the need to aggressively and urgently treat pediatric SE.

An unanswered question at the present is how the diagnosis and treatment of NCS and NCSE in children affects morbidity, mortality, and long-term outcome. Worse outcomes in adults are associated with NCSE [76], however it is not clear if this is influenced more by etiology than as a primary result of NCSE. When NCS or NCSE are suspected, however, expedited diagnosis using EEG and treatment are strongly recommended, realizing that the effect on prognosis remains to be fully understood.

Conclusion

Status epilepticus is commonly encountered in the PICU. Although most cases manifest obvious clinical signs, PICU patients in SE can sometimes show only subtle signs, necessitating diagnostic support with EEG. Once diagnosed, concurrent evaluation and treatment should be accomplished urgently, and the health-care team should be prepared with a standardized evidence-based plan for treatment. Although outcome is strongly influenced by etiology, urgent treatment is prudent to ensure the best long-term outcome possible. Future long-term prospective studies of the varied types of SE in children encountered in the PICU are needed to help us understand the outcomes of effective diagnosis and treatment of pediatric SE, to include both convulsive and nonconvulsive forms.

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