Status Epilepticus in Children

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Status epilepticus is a common, life-threatening medical emergency in pediatric patients. Recent medical literature has focused on identifying risks and treatment options. This article highlights the epidemiology of status epilepticus, both convulsive and nonconvulsive, in children. It also reviews the recommended medications for first-line treatment of status epilepticus and refractory status epilepticus. Emphasis is placed on future pharmacotherapies and consideration of neurosurgical intervention when indicated.

Introduction

The definition of status epilepticus (SE) has a long and rich history. The first known reference to status epilepticus dates from 718–612 BC in the Sakikku cuneiform, as follows: “If the possessing demon possesses him many times during the middle watch of the night, and at the time of his possession his hands and feet are cold, he is much darkened, keeps opening and shutting his mouth, is brown and yellow as to the eyes…It may go on for some time, but he will die.”

In 1962, at the Xth Marseilles Colloquium (the tenth European electroencephalography meeting), a more contemporary definition was developed: “A condition characterized by epileptic seizures that are sufficiently prolonged or repeated at sufficiently brief intervals so as to produce an unvarying and enduring epileptic condition” [1].

The most recent revision by the International League Against Epilepsy defines SE as “a seizure that shows no clinical signs of arresting after a duration encompassing the great majority of seizures of that type in most patients or recurrent seizures without interictal resumption of baseline of CNS function” [2]. Although this definition does not specify a temporal meaning, the traditional view of the duration is greater than 30 minutes. As the molecular basis for the advantages of early seizure cessation has begun to be elucidated, more recent literature advocates that there should be a revised definition of SE as lasting no longer than 10 minutes [3] or even 5 minutes [4]. However, determining the duration of SE is not always possible because the onset may not be observed. In the setting of convulsive SE (CSE), the termination, which would be when all clinical signs of tonic, tonic-clonic, or motor activity have stopped, may be easier to recognize. Conversely, the termination of nonconvulsive SE (NCSE) may be less clear [5••]. It is likely that the various definitions will persist until the pathophysiology of CSE and NCSE is better understood.

Classification of Status Epilepticus

It is important to identify and classify the causes of CSE. The International League Against Epilepsy classification has been the standard for adult and pediatric SE and separates the etiology into acute symptomatic, remote symptomatic, idiopathic epilepsy-related, cryptogenic epilepsy-related, and unclassified [2].

Recent epidemiologic studies have argued that febrile convulsions should be a separate entity from acute CSE because they have a more favorable prognosis overall. Moreover, studies that do not separate febrile CSE from acute symptomatic CSE are likely to overstate the severity of outcome of febrile CSE, thus attenuating the severity of acute neurologic insults [6,7••]. Pediatric studies have also classified SE according to CSE, which can be further divided into generalized CSE and partial CSE and also NCSE (in which the primary symptom is the disturbance of consciousness with or without subtle motor manifestations) [8•]. Because accurate categorization of individual cases may depend on the degree of investigation, the availability of ancillary tests, and clinical data, a revised classification may still have its limitations [6].

Epidemiology

The annual incidence of pediatric SE ranges from 10 to 73 per 100,000. The highest incidence is in children less than 2 years of age, where it ranges from 135 to 156 per 100,000, with the greatest peak in the first year of life. If febrile SE is excluded, the incidence is decreased by 25% to 40% [7••,8•,9].

Prospective population-based studies have stratified the etiology of SE in children. Prolonged febrile convolution is the most common etiology (32%–46%), followed by acute symptomatic (17%–24%) and remote symptomatic (11%–28%) SE. Less common are cryptogenic or idiopathic (11%–16%) SE [5••,7••,8•]. CSE is more common
Increased risk for recurrence associated with a preexisting history of an epileptic encephalopathy or a specific epilepsy syndrome. Common in the adult population, noncompliance with antiepileptic drugs (AEDs) is not as common as NCSE, occurring in as much as 86% of patients [8•]. Table 1 lists the common etiologies of SE in children that should be considered in each case.

Between four and eight children per 1000 are expected to experience CSE before age 15 years [5••]. The most common risk factor for SE in children is a prior history of SE [10,11]. Other common risk factors for pediatric SE include a younger age, a symptomatic etiology, and a history of an epileptic encephalopathy or a specific epilepsy syndrome. Common in the adult population, noncompliance with antiepileptic drugs (AEDs) is not as common a risk factor for children [11,12]. In children who have no history of previous SE, the risk of SE decreased with increasing age. In children who have a previous history of SE, patients with symptomatic epilepsy have a higher risk. Children who have had a questionable episode of SE before the diagnosis of epilepsy are more likely to have SE during the follow-up [11]. Karasalihoglu et al. [13] suggested that SE is more likely to appear in children who have had a partial seizure evolving to a secondarily generalized seizure or in those with myoclonic seizures.

### Morbidity and Mortality

The outcome of pediatric SE is based upon its morbidity and mortality. Among children, the risk of recurrence of SE at 1 year was 11% in one study [14] and 16% in another [7••] and was 18% at 2 years [14]. Both studies included children with a first febrile episode of SE and reported an increased risk for recurrence associated with a preexisting neurologic abnormality. A Finnish study included only patients with afebrile SE and found that more than half of those with SE experienced recurrent SE [12]. The risk for recurrent afebrile SE in this study was 13.1% at 1 year and 16.9% at 2 years [12]. In a Minnesota population-based study combining all ages, one-third of individuals experienced a second episode of SE, with the risk doubled for those with progressive symptomatic etiology and for female patients [15•].

Future epilepsy, focal neurologic deficits, cognitive impairment, and behavioral problems may be associated with CSE. However, specific risk factors in the pediatric population have not yet been reported [6]. In patients with cryptogenic SE, a significant portion of infants went on to develop West syndrome or mental deficiency, most because of an underlying neurologic disorder [8•].

A devastating epileptic encephalopathy, which begins with intractable SE evolving over days to weeks and persists as bilateral temporal lobe pharmaco-resistant epilepsy, has become increasingly recognized in children. The main features include 1) onset in previously healthy school-aged children; 2) an initial febrile context but no evidence of intracranial infection; 3) onset with prolonged intractable SE followed by intractable epilepsy without any latent period in between; 4) clinical and electroencephalogram (EEG) features of focal seizure onset during both SE and follow-up, primarily in the perisylvian areas; 5) bilateral mesial temporal dysfunction on neuropsychological tests with atrophy and/or in hypersignal on MRI; and 6) frontal lobe involvement in half of the cases. The morbidity of this devastating encephalopathy, more commonly known as fever-induced epileptic encephalopathy in school-aged children (FIRES), consists of severe memory, language, and behavior troubles with permanent cognitive sequela [16••].

In a population-based study of adults and children, the rate of fatalities ranged from 7.6% to 22% and showed a bimodal distribution, with young children and elderly patients experiencing the greatest mortality from CSE. Age was the only significant predictor of in-hospital death following generalized CSE [9]. The mortality rate in pediatric SE ranges from 0% to 7% [5••,7••,12]. Children with acute or remote symptomatic CSE are associated with the highest mortality during hospitalization [7••]. Death due to SE is most often due to the underlying cause, such as central nervous system infection or severe neurologic disabilities. Some have associated SE as a direct cause of death, regardless of the underlying etiology [12,15•]. The relatively low morbidity and mortality in pediatric SE is likely multifactorial, being helped by advances in childhood medical and nursing practices with acute life support, critical care management, and evolving AED therapy [5••].

### Table 1. Common etiologies of status epilepticus in children and incidences from population-based studies

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Incidence</th>
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<tr>
<td>Acute</td>
<td></td>
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<tr>
<td>Acute symptomatic (17%–52%)</td>
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<tr>
<td>Acute CNS infection (bacterial meningitis, viral meningitis, encephalitis)</td>
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<tr>
<td>Metabolic derangement (hypoglycemia, hyperglycemia, hyponatremia, hypocalcemia, anoxic injury)</td>
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<tr>
<td>AED noncompliance or withdrawal</td>
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<td>AED overdose</td>
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<td>Non-AED/drug overdose</td>
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<tr>
<td>Prolonged febrile convulsion (23%–30%)</td>
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<td>Influenza</td>
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<td>Exanthem subicum</td>
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<td>Remote (16%–39%)</td>
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<tr>
<td>Cerebral migration disorders (lissencephaly, schizencephaly)</td>
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<tr>
<td>Cerebral dysgenesis</td>
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<td>Perinatal hypoxic-ischemic encephalopathy</td>
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<tr>
<td>Progressive neurodegenerative disorders</td>
<td></td>
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<tr>
<td>Idiopathic/cryptogenic (5%–19%)</td>
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AED—antiepileptic drug; CNS—central nervous system.
30 minutes without improvement in clinical state or return to a preictal EEG pattern between seizures. In 49 admissions of 43 patients in a neurointensive care unit, 23 patients were found to be in NCSE [17]. Seventy-six percent of patients had a history of a clinical seizure preceding NCSE, and 25% were de novo [17]. Mortality of NCSE in this combined adult–pediatric study was 57% and was strongly linked to duration of NCSE and delay of diagnosis of NCSE [17]. In a large hospital-based series, 92% of patients with nonconvulsive seizures were in NCSE (for an overall incidence of 17% in those monitored) [18]. Risk factors in this study included comatose state on neurologic examination, age younger than 18 years, a past medical history of epilepsy, and a convulsive seizure prior to monitoring [18].

The incidence of NCSE in pediatric patients undergoing long-term monitoring in the intensive care setting ranges from 16% to 32% [19–20,21••]. Tay et al. [19•] found that half of these children had preexisting epilepsy, with common causes including an exacerbation of the underlying metabolic disorder or an acute infection. The overall mortality rate of NCSE was 26% [19•]. Median duration lasted from 48 to 56 hours. More than one-fourth of the pediatric patients were less than 1 year of age [19•,21••].

Most studies in adults have shown that NCSE is often due to an acute or remote central nervous system insult (eg, stroke, anoxic-ischemic encephalopathy, intracerebral hemorrhage). However, most children with NCSE are previously healthy and have no preexisting disease [19•,21••]. The availability of prolonged EEG monitoring has allowed increased recognition of this condition. Recommendations for monitoring include any patient with unexplained or protracted impairment of consciousness, comatose state on neurologic examination, or altered behavior after a recognized seizure [17].

EEG Findings in Status Epilepticus

Treiman et al. [22] initially described five identifiable EEG patterns that occurred in a predictable sequence during the course of secondarily generalized CSE in adults. The stages were 1) EEG changes of discrete seizures with interictal slowing; 2) merging seizures with waxing and waning ictal discharges; 3) continuous ictal discharges; 4) continuous ictal discharges with “flat” periods; and 5) periodic epileptiform discharges on a “flat” background [22]. In a primarily adult retrospective review, focal interictal epileptiform discharges were most common during routine and digital video EEG, with the latter more commonly detecting electrographic seizures. Other patterns included periodic lateralized epileptiform discharges, burst suppression, and generalized spike-and-wave discharges [23]. Generalized background abnormalities were also common in patients with CSE [13]. After the resolution of SE, epileptiform discharges were significantly more common in the remote symptomatic and cryptogenic groups than in the acute symptomatic group [8•].

EEG findings in NCSE are more variable, including typical and atypical spike and wave, multiple or polyspike discharges, and a rhythmic sharp delta pattern [19•]. Abnormal EEG patterns such as periodic lateralized epileptiform discharges, generalized periodic epileptiform discharges, and burst suppression patterns were seen in those patients with NCSE who required prolonged EEG monitoring [18]. In a recent pediatric study, most ictal EEGs showed a focal distribution of discharges [19•].

Treatment of Status Epilepticus

Acute status epilepticus

Benzodiazepines are recommended as the initial drug of choice for treating SE. Intravenous (IV) diazepam was developed first, followed by IV lorazepam. IV diazepam has a more rapid onset of activity, whereas IV lorazepam has a longer duration of antiseizure effect. In the past, when IV diazepam was administered as the first-line medication, a second AED was often required because breakthrough seizures would develop. With the development of rectal diazepam, there seems to be less risk of breakthrough seizures. However, there are no published pediatric head-to-head comparison trials of the benzodiazepines.

Most studies for the acute treatment of SE have been in the adult population. The Veterans Affairs Status Epilepticus Cooperative Study [24], a multicentered adult trial, evaluated four different treatment modalities for SE: 1) diazepam then phenytoin, 2) lorazepam, 3) phenobarbital, or 4) phenytoin. Of the 570 patients who met inclusion criteria, 395 (69%) were in SE. In a pairwise comparison, lorazepam was significantly superior to phenytoin but not to the combination of diazepam then phenytoin or phenobarbital alone. Among the 134 patients with a verified diagnosis of NCSE, there were no significant differences among the treatments [24].

Adult studies have also evaluated out-of-hospital treatment for SE. In a randomized double-blinded trial, patients were given either 5 mg of diazepam repeated to a maximum of 10 mg, 2 mg of lorazepam repeated to a maximum of 4 mg, or placebo. Primary outcome was termination of SE by the time of arrival to the emergency department. Secondary outcome measures included out-of-hospital complications, complications during treatment, duration of SE prior to arrival at the hospital, and neurologic outcome at discharge. The study found IV benzodiazepines to be safe and effective when administered by paramedics. Lorazepam and diazepam were both more effective than placebo, with a trend favoring lorazepam over diazepam. However, between 41% and 57% of patients who received active therapy were still in SE upon arrival to the hospital [25]. In 45 episodes of generalized CSE in 38 children, prehospital treatment with diazepam was associated with a shorter duration of SE (32 minutes vs 60 minutes) compared with lorazepam, with a decreased risk of recurrent seizures in the emergency department (58% vs 85%) [26].
In a retrospective pediatric study in the United Kingdom evaluating pre-intensive care unit treatment of SE, either diazepam or lorazepam was the most commonly administered first-line AED, but the dose was frequently lower than that recommended. Children who had prehospital treatment were more likely to receive more than two doses of benzodiazepine, and those who had prehospital treatment were also more likely to have respiratory depression [27].

Traditionally, based on long-term clinical experience and case-control studies, IV phenytoin has been used as the second-line drug for SE [28••]. A dose of 18 mg/kg of phenytoin given over 20 minutes through an IV peripheral line is recommended, with additional bolus to maximize the total dose to 25 mg/kg [24]. Fosphenytoin is a prodrug of phenytoin (150 mg of fosphenytoin is equivalent to 10 mg of phenytoin) with 100% bioavailability. Although there are limited adult and pediatric efficacy data on the use of fosphenytoin, the drug’s practical benefits include a reduced risk and incidence of serious extravasation reactions, hypotension, and cardiac dysrhythmias. Although it is more expensive and not routinely available in most hospitals worldwide, it may eventually replace phenytoin as the long-acting anticonvulsant of choice [29].

Phenobarbital may also be used as a second-line agent or as an alternative to phenytoin when a benzodiazepine has failed. The initial dose for SE may range from 15 to 20 mg/kg in children, with a maintenance dosage of 3 to 4 mg/kg per day. However, the dose of phenobarbital to be recommended is not as precisely determined as other AEDs. Very high-dose phenobarbital, without reference to a predetermined maximum dose or serum level, has also been used to treat refractory SE. Adverse effects of the higher dose include a marked and prolonged sedative effect, with depression of respiratory drive and hypotension at higher doses [30].

Combined adult and pediatric randomized trials have suggested the favorability of valproic acid over phenytoin. In a randomized trial of age- and sex-matched groups comparing IV valproic acid with IV phenytoin, IV valproic acid was found to be as effective as IV phenytoin and was more tolerable [28••]. Similarly, Misra et al. [31•] looked at seizure control with either loading phenytoin at 18 mg/kg at an infusion rate of 50 mg/min or valproic acid at 30 mg/kg over 15 minutes. Valproic acid was more effective than phenytoin in controlling CSE, both as the first (66% vs 25%) and as the second choice (79% vs 25%). VPA as a second choice terminated SE more frequently (79%) than phenytoin (25%) [31•]. However, this study did not compare the maximum dose of valproic acid to the maximum equivalent dose of phenytoin (25 mg/kg).

Valproic acid has also been effective in pediatric retrospective studies. In a retrospective review of 40 patients loaded with valproic acid, 20 of whom had SE, rapid loading at 25 mg/kg at a rate of 3 mg/kg per minute stopped seizures in 18 (90%) patients with SE within 20 minutes [32]. All 18 patients regained baseline mental status within 1 hour of seizure cessation. Among the other 22 patients with acute repetitive seizures, only one had further seizures after valproic acid infusion, with transient tremor as an adverse effect in one patient [32]. In a relatively large study of intravenous valproic acid in children in SE, 78% of the patients who were refractory to initial standard therapy responded to valproic acid, and in most cases the response was very rapid [33]. Sixty-five percent of these patients responded immediately (within 2–6 minutes). The remaining 10% responded within 3 to 10 minutes. Loading doses ranged from 20 to 40 mg/kg, with the highest success rate being from 30 to 40 mg/kg [33]. Valproic acid, along with the newer anticonvulsants discussed in the following text, may eventually move to replace phenytoin as second-line therapy for SE. Table 2 summarizes suggested medications for acute and refractory SE.

**Refractory status epilepticus**

More than 90% of all convulsive seizures end spontaneously within the first 5 minutes. As the time of initiation of therapy for SE with benzodiazepines increases, the efficacy of these medications decreases [12]. The most accepted definition of refractory SE is when a seizure continues despite the administration of two or more first-line medications [5••]. Others have characterized the duration to refractory SE (RSE) as lasting greater than 60 minutes [34,35••,36•].

High-dose pentobarbital, or pentobarbital coma, has commonly been used in the intensive care unit for RSE, and this requires continuous EEG for monitoring burst suppression. It is generally given as a bolus dose of 5 to 15 mg/kg followed by a maintenance infusion of 1 to 5 mg/kg per hour. Although it is effective in terminating seizures, pentobarbital administration is associated with hypotension, myocardial depression, and low cardiac output. It is also a potent respiratory depressant, often requiring endotracheal intubation and mechanical ventilation [35••].

More recently, midazolam has been suggested as a first-line therapy in RSE. In a retrospective multicenter study, 76% of patients treated with midazolam achieved clinical seizure control within 30 minutes of treatment initiation [35••]. Midazolam was eventually successful in treating 15 seizure episodes (88%). Relapse after discontinuation of therapy occurred in one patient (6%), and there were no significant adverse effects [35••]. Morrison et al. [36•] recently developed a high-dose midazolam algorithm with an emphasis on rapid seizure control. The bolus dose was 0.5 mg/kg followed by an infusion of 2 μg/kg per minute up to 24 μg/kg per minute and reaching as high as 32 μg/kg per minute. Of 17 patients admitted to the pediatric intensive care unit who followed this algorithm, 15 patients were controlled and remained seizure free for 12 to 24 hours on midazolam prior to weaning. The remaining two patients could not be controlled with midazolam therapy and required the administration of sodium thiopentone [36•].

Brevoord et al. [37] retrospectively reviewed four different treatment modalities in the cessation of generalized
CSE in children: 1) midazolam bolus and infusion, 2) phenytoin bolus, 3) phenytoin bolus followed by a versed continuous infusion, or 4) phenobarbital/pentobarbital bolus and infusion. Most of the patients (68%) had initially received rectal diazepam. Cessation of epileptic activity was achieved with midazolam only in 58 patients (48%); with midazolam and phenytoin in 19 patients (15%); with midazolam, phenytoin, and continuous midazolam in 32 patients (26%); and with additional barbiturates in 13 patients (11%) [37].

Anesthetic treatment of refractory status epilepticus

Various anesthetics have been administered in adult and pediatric SE. Propofol has been administered for RSE and has been shown to terminate clinical seizures, but the quality of burst suppression has been variable [38•]. However, case reports of propofol infusion syndrome in children, characterized by metabolic acidosis, lipemic serum, and bradyarrhythmia leading to progressive myocardial failure, make this a less desirable option [39]. Within minutes of initiating therapy, the inhalational anesthetic isoflurane,
accompanied by desflurane when necessary, stops epileptic discharges with sustained burst suppression [40]. In a pediatric retrospective study, propofol was more safe and effective than thiopental in controlling RSE (64% vs 55%, respectively) [41]. In the past and in certain cases, other anesthetics, including paraldehyde and lidocaine, along with the ketogenic diet, have been used in treating RSE.

**Emerging pharmacotherapy**

Newer studies suggest that both levetiracetam and topiramate may have promise in RSE in the future. Since 1999, levetiracetam has been prescribed worldwide as adjunctive therapy for partial-onset seizures in adults and in children older than 4 years of age. Most analyses have been in the adult population. Patel et al. [42•] found that in six patients (including children and adolescents) with SE refractory to at least two AEDs, adjunctive levetiracetam proved effective in several types of SE, including generalized, focal, and NCSE. Effective doses of levetiracetam ranged from 500 to 3000 mg/d, and seizure control was achieved within 12 to 96 hours [42•]. Rosetti and Bromfield [43] found that levetiracetam administered together with other AEDs represented 10% of the cohort of patients with SE. As its pharmacokinetic profile is very promising, levetiracetam may represent a valuable alternative to the existing treatment of SE [43]. In 2007 the parenteral form of administration became available, allowing earlier administration. The injectable formulation of levetiracetam has proven to be well tolerated and exhibits similar pharmacokinetics to oral formulations, broadening its clinical application [44].

Topiramate, a sulfamate-substituted monosaccharide, is a broad-coverage antiepileptic drug available only in the parenteral form. Topiramate administered via nasogastric tube at doses between 300 and 1600 mg/d in six adults effectively terminated RSE in generalized CSE and NCSE [45]. In the case of infantile spasms, topiramate may be rapidly titrated or loaded without severe adverse effects [46]. Kahrman et al. [47] retrospectively reported on three children with pediatric RSE who responded to rapidly titrated nasogastric topiramate at a daily dose of 5 to 6 mg/kg. Topiramate was initiated at 2 to 5 mg/kg per day and titrated up to 22 to 25 mg/kg per day in children with partial SE refractory to phenobarbital, phenytoin, and midazolam. Rapid titration to a very high total daily dose via nasogastric tube was well tolerated and allowed for the successful taper to other AEDs [48].

**Neurosurgical interventions for status epilepticus**

Rasmussen’s encephalitis is a rare childhood disease characterized by the triad of the development of intractable focal seizures with epilepsy partialis continua, progressive hemiparesis, and increasing intellectual impairment with inflammation of the brain and progressive atrophy of one cerebral hemisphere. Hemispherectomy is often considered to control the seizures and prevent the progressive cognitive decline that accompanies the disease. Neurosurgical intervention may also be considered in the rare case of medically refractory SE. Emergent epilepsy surgery in 10 children with focal epileptogenesis successfully stopped SE, and all patients showed significant improvement during follow-up [49]. In a retrospective analysis of patients receiving epilepsy surgery, 20% were in continuous SE or intermittent SE. All of these patients had extratemporal localization of the epilepsy, and most (86%) of these patients underwent hemispherectomy [50]. In a 4-year period at two tertiary referral epilepsy centers, five pediatric patients were treated successfully with neurosurgery for SE. The seizures were fully controlled in four patients, and in the fifth patient the seizure frequency was reduced by more than 90% [51••].

**Conclusions**

SE is a common medical emergency in pediatrics. Advances in prolonged EEG monitoring have allowed for increased recognition of NCSE in children. Because there is increasing evidence that SE may be treated better with earlier intervention, the definition of SE is evolving, with an emphasis on shortening the duration of seizure activity. A significant percentage of patients who present with their first seizure may present in SE. In the pediatric population, the prolonged febrile convulsion should be identified distinctly from the acute symptomatic group because of the distinct differences in prognosis and neurologic outcomes. Patients at greatest risk, especially those with a history of prior SE, should be identified early. Rectal diazepam should be made available to caregivers for out-of-hospital treatment of SE. Benzodiazepines are still viewed as a first-line medication, with phenytoin, fosphenytoin, and phenobarbital following as second-line medications. Valproic acid and midazolam loading and maintenance doses may be tolerable and more efficacious at higher concentrations. Newer pharmacotherapies, including topiramate and levetiracetam, may prove more advantageous for acute and refractory SE in the future. Although neurosurgical treatment is never viewed as a first- or second-line therapy, it should be considered in patients with RSE, but only in those with focal epileptogenesis or epilepsy partialis continua. The future of successful SE treatment lies within evidence-based guidelines derived from out-of-hospital and in-hospital therapies, along with controlled studies in pediatric populations that target specific etiologies. Future studies should focus on the need to standardize the treatment of convulsive emergencies to shorten the duration of in-hospital or intensive care unit stay, which will ultimately decrease morbidity and mortality.

**Disclosures**

No potential conflicts of interest relevant to this article were reported.
References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

• Of importance
•• Of major importance


4. Lowenstein DH, Bleck T, MacDonald RL: It’s time to revise the definition of SE. Epilepsia 1999, 40:120–122.


This is a comprehensive retrospective review on the epidemiology and outcome of patients presenting in SE.


This is the first prospective population-based study of pediatric SE.


This is the first report of the incidence of SE in Japan, where the incidence is much higher than that reported in whites.


The most common risk factor for SE is a history of prior SE.


This article emphasizes the increased need to recognize devastating epileptic encephalopathy in school-aged children as a cause of SE with resulting severe morbidity after a fever.


This study shows that nonconvulsive SE in children occurs in previously healthy children with EEG characteristic of focal discharges.


The authors of this study found that nonconvulsive seizures are not uncommon in pediatric patients with an altered state of consciousness, and they characterized common EEG patterns in nonconvulsive SE.


This randomized study showed IV valproic acid to be as effective as IV phenytoin patients with SE, with better tolerability.


This randomized controlled study suggested favorability of IV valproate over IV phenytoin, although the maximum doses were not equally compared.


The authors of this study developed an algorithm for high-dose midazolam for seizure control.

This study showed that propofol may terminate clinical seizures, but the quality of EEG burst suppression may vary.


This study showed that high-dose levetiracetam can be beneficial as adjunctive therapy in refractory SE.


This study showed a high success rate in surgical intervention for medically refractory SE.