Afebrile Pediatric Seizures

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KEYWORDS

- Absence seizures Neonatal seizures Infantile spasms
- Afebrile Hyponatremia Convulsions Epilepsy

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Seizures in children can be anxiety provoking for both the parent and the medical caregiver. By 14 years of age, approximately 1% of children will experience an afebrile seizure with the highest incidence being in children younger than 3 years.¹ Population-based studies reveal that there are between 25,000 and 40,000 children per year in the United States who sustain a first-time, unprovoked seizure, 70% of which are idiopathic.^{2,3} The overall recurrence rate in children with a first unprovoked afebrile seizure varies from 14% to 65%⁴; however, up to 88% of seizure recurrences occur within the first 2 years of the initial event.^{5,6} Furthermore, Shinnar and colleagues⁷ found that children who experience their first unprovoked seizure during sleep have approximately twice the recurrence rate as children whose first seizure occurred while awake. The first priority in a seizing patient is airway management and subsequent termination of the seizure.

SEIZURES THAT OCCUR IN CHILDHOOD Absence Seizures

Simple (typical) absence seizures are uncommon before 5 years of age, and are typically characterized by a sudden cessation of motor activity with an accompanying

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blank expression. Flickering of the eyelids may also be seen. The episodes last less than 30 seconds and are not associated with a postictal period. Complex (atypical) absence seizures are usually associated with myoclonic activity in the face or extremities, and are associated with an altered level of consciousness.

Lennox-Gastault

In this seizure disorder, patients experience a combination of tonic, absence, atonic, or myoclonic seizures with seizure onset between 3 and 5 years of age. Most of these children also have accompanying mental retardation and severe behavioral problems. An electroencephalogram (EEG) shows an irregular, slow, high-voltage spike pattern. While many drugs have been used to treat this condition, management is still very difficult. Valproic acid is the drug that is most commonly used; however, felbamate, top-iramate, lamotrigine, and ethosuximide have also been used as add-on therapy.^{8,9} The ketogenic diet (high fat, low protein, low carbohydrates) has been used with some success for these children. A study by Hemingway and colleagues¹⁰ revealed that 13% of patients with intractable seizures who were treated with the ketogenic diet were seizure free at 1 year. Furthermore, a decrease by up to 99% in the frequency of seizures was noted in an additional 14% of patients who were started on the ketogenic diet. It is important to remember that if glucose is given, increased seizure activity may be seen.

Benign Rolandic Epilepsy

This syndrome typically involves children between 3 and 13 years of age who experience nighttime seizures. The initial phase of the seizure involves clonic activity of the face, which can then secondarily generalize. The history is characteristic with the seizures occurring during sleep. An EEG is important in the evaluation, as a characteristic perisylvian spiking pattern can be seen. Unless these seizures are frequent, no therapy is needed as patients will usually outgrow these episodes by early adulthood. Carbamazepine and levetiracetam have been used with success in the treatment of benign Rolandic seizures.

Juvenile Myoclonic Epilepsy of Janz

As the name implies, this disorder begins in early adolescence (peak age range is 12–15 years). Patients experience myoclonic jerks typically on awakening, but may also have tonic-clonic (80%) or absence (25%) seizures. Typical inducing factors include stress, alcohol, hormonal changes, or lack of sleep. The EEG is help-ful in the diagnosis as a pattern of fast spike-and-wave discharges can be seen. Valproic acid has traditionally been the drug of choice; however, levetiracetam has shown effectiveness as both add-on and monotherapy for partial and generalized seizures.¹

Infantile Spasms (West syndrome)

Children with this syndrome typically present between 4 and 18 months of age, and males are more commonly affected than females. Up to 95% of these children are mentally retarded and there is a 20% mortality rate. Patients experience spasms, which are typically single jerking episodes in flexion or extension of the involved muscle groups. The jerking is spasmodic, often occurring in clusters, and the child often cries during the episode. Episodes rarely occur during sleep. Up to 25% of patients have tuberous sclerosis. The EEG shows the classic pattern of hypsarrhythmia (random high-voltage slow waves with multifocal spikes). Treatment with adrenocorticotropic hormone (ACTH), prednisone

vigabatrin, and pyridoxine have been used with some success. Valproic acid, lamotrigine, topiramate, zonisamide, levetiracetam, and benzodiazepines have also shown some effectiveness.^{8,9,11,12}

EMERGENCY DEPARTMENT EVALUATION OF THE FIRST AFEBRILE SEIZURE History and Physical Examination

The history should focus on the events immediately before the onset of the episode, a description of the seizure including seizure duration, cyanosis, loss of consciousness, the presence of incontinence, length of the postictal period, any postictal neurologic abnormalities, recent immunizations, change in diet or oral intake, and family history of seizures. A detailed birth history is important in new-onset seizures in neonates and infants. Home therapies and home remedies for any recent illnesses should also be determined. If the patient has a known seizure disorder, then it is important to ascertain if this seizure was different from previous seizures, the normal seizure frequency for the patient, medications the patient is on, if the patient has been compliant with the medication regimen, or if there have been any recent medication changes. The history may often help to differentiate a true seizure from a seizure mimic. Psychosocial history and recent family events are important in determining psychogenic seizures versus neurologic seizures. Patients who experience true seizures may describe an aura such as epigastric discomfort or a feeling of fear. The patient's positioning during the seizure, loss of sphincter control, duration of seizure, and the length of the postictal state should also be noted.

The physical examination focuses on the neurologic examination. In infants, measurement of head circumference may be helpful. A bulging fontanel indicates increased intracranial pressure. The eyes should be examined for papilledema and retinal hemorrhages. The presence of hepatosplenomegaly may indicate a metabolic or glycogen storage disease. The skin should also be checked for lesions such as café-au-lait spots (neurofibromatosis), vitiliginous lesions (tuberous sclerosis), and port-wine stains (Sturge-Weber syndrome). The differential diagnosis of seizures in children is listed in **Box 1**.

EMERGENCY DEPARTMENT MANAGEMENT

For the patient who is no longer seizing and whose airway is protected, little immediate management is needed beyond supportive care and continued monitoring. A bedside blood glucose reading should be obtained and hypoglycemia treated as follows: D₁₀ solution (3-10 mL/kg) for newborns and D_{25} (2-4 mL/kg) solution for children. Dextrose should not be empirically given to children on ketogenic diets, as this will break the ketogenic state and may result in seizures. Any seizure that has lasted longer than 5 minutes should be treated with a benzodiazepine.¹³ Lorazepam is an excellent choice, as it has an antiseizure duration of action of approximately 12 hours. Other options are diazepam and midazolam (Table 1). If the patient is actively seizing and intravenous (IV) access cannot be obtained, rectal diazepam, dosed at 0.3 to 0.5 mg/kg, can be administered.¹⁵ Parents of known epileptics may have already administered this medication before paramedic arrival. Although diazepam has been the agent of choice in the past, recent studies support the use of midazolam.¹⁶⁻¹⁸ The advantage of midazolam is that it can be administered by many routes including IV, intramuscular (IM), rectal, intranasal, and buccal.^{19,20} In one study, when compared with IV diazepam, IM midazolam (0.2 mg/kg) resulted in faster seizure termination due to more rapid administration rates.²¹

Box 1

Differential diagnosis of seizures in children

Benign paroxysmal vertigo

Benign myoclonus of infancy

Benign sleep myoclonus

Breath-holding spells^a

Gastroesophageal reflux

Sandifer syndrome^b

Migraine headaches

Night terrors, sleepwalking, somniloquy, narcolepsy^c

Psychological

Attention-deficit disorder

Hyperventilation

Hysteria and rage attacks

Pseudoseizures

Panic attacks

Shuddering attacks^d

Sleepwalking

Syncope

Tics-Tourette syndrome

Toxins

^a **Breath-holding spells:** Patients with cyanotic breath holding spells typically become angry, stop breathing in end-exhalation, pass out, and may have a hypoxic seizure.

^b Gastrointestinal reflux: Infants with Sandifer syndrome present with crying, vomiting, esophagitis, and writhing and arching movements of the neck and back that may be confused with seizure activity.

^c **Sleep disorders:** Night terrors occur when the child sits up suddenly during sleep, cries, screams, and is unresponsive to consoling attempts. The patient then returns to sleep and has no recollection of the event the next morning.

^d **Paroxysmal movement disorders:** Shudder attacks are episodes of shivering activity that are not associated with any change in mental status. Spasmus mutans typically occurs in infants between 4 and 12 months of age, with the child experiencing head nodding, head tilt, and nystagmus.

If benzodiazepines do not terminate the seizure, the next agent of choice is either phenytoin or fosphenytoin because they do not cause central nervous system or respiratory depression. Fosphenytoin can be given 3 times as quickly as phenytoin (3 mg/kg/min vs 1 mg/kg/min) and reaches therapeutic serum concentrations within 15 minutes (phenytoin takes 25 minutes).²²

LABORATORY TESTING

In patients who are on anticonvulsant medications, a drug level should be obtained. The practice of obtaining an electrolyte panel, and calcium and magnesium levels on every patient with a short seizure has been called into question in the child who

Table 1 Drugs used in the management of seizures				
Diazepam	0.2–0.3 mg/kg IV, 0.5 mg/kg rectal			
Lorazepam	0.05–0.1 mg/kg IV			
Midazolam	0.1 mg/kg IV, 0.2 mg/kg IM			
Phenytoin	15–20 mg/kg IV (no faster than 1 mg/kg/min)			
Fosphenytoin	15–20 mg/kg IV or IM (can be given 3 mg/kg/min IV)			
Phenobarbital	18–20 mg/kg IV, then 5–10 mg every 10 min (1 mg/kg/min) (maximum 50–60 mg/kg)			
Levetiracetam	IV 50 mg/kg (maximum 2.5 g) Oral treatment should be initiated with a daily dose of 20 mg/kg in 2 divided doses (10 mg/kg bid). The daily dose should be increased every 2 weeks by increments of 20 mg/kg to the recommended daily dose of 60 mg/kg (30 mg/kg bid) ¹⁴			
Pyridoxine	50–100 mg IV			
Lidocaine	1–2 mg/kg IV, then 4–6 mg/kg/h for refractory seizures			

Abbreviations: bid, twice a day; IM, intramuscular; IV, intravenous.

is alert, interactive, and back to his or her baseline level of functioning. The Report of the Quality Standards Subcommittee of the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society recommends that laboratory testing only be ordered based on clinical circumstances such as vomiting, diarrhea, dehydration, or failure to return to baseline level of consciousness.²³ Furthermore, a toxicology screen should be performed if there is any suspicion of drug exposure or abuse.

Newborns and children younger than 6 months have been found to be at greater risk for electrolyte abnormalities because of underlying metabolic abnormalities,²⁴ specifically hyponatremic seizures due to increased free water intake from formula overdilution. Farrar and colleagues²⁵ studied 47 patients younger than 6 months with seizures, and reported that the median seizure duration was longer (30 minutes vs 17 minutes, P = .007) in patients with hyponatremia, with a greater incidence of status epilepticus, 73% versus 36% (P = .02). Furthermore, emergency intubation was performed more often in hyponatremic patients than in normonatremic patients (P = .009). Median temperature was lower in hyponatremic infants (35.5°C vs 37.2°C, P = .0001). Temperature less than 36.5°C was the best predictor of hyponatremic seizures in infants younger than 6 months.

In a retrospective review of 149 infants younger than 12 months, Scarfone and colleagues²⁶ studied 214 visits for seizures, 80 of which were classified as febrile seizures. All laboratory results were reviewed: 19 of 80 febrile seizures were tested and none had abnormal electrolytes or calcium or magnesium levels: 67 of 134 non-febrile infants were tested, with 13% having abnormal chemistry results. However, patients with abnormal laboratory results were more likely to be actively seizing in the emergency department (ED), have hypothermia (temperature <36.5°C), or be younger than 1 month.

Based on the results of these studies, it is reasonable to obtain laboratory studies on pediatric patients with prolonged seizures, age less than 6 months, history of diabetes or metabolic disorder, dehydration or history of excess free water intake, and with an altered level of consciousness. Routine lumbar puncture in patients who are alert and oriented after a first afebrile seizure is not indicated. However, a spinal tap should be

performed in patients with altered mental status, signs of meningitis/encephalitis, prolonged postictal period, or immunocompromised state, or if there is any suspicion of subarachnoid hemorrhage.

NEUROIMAGING

Magnetic resonance imaging (MRI) has superior resolution and is more sensitive than head computed tomography (CT) for the detection of low-grade tumors and heterotopic gray matter. A recent study of MRI findings in children with a first recognized seizure found at least one abnormality in 31% of children. Abnormalities defined as significant or related to seizures occurred in only 14%.²⁷ MRI is not readily available in all EDs and may be best performed on an outpatient nonemergent basis. MRI also lacks the radiation risks of CT scans.²⁸ Children are at greater risk than adults from a given dose of radiation because they are more radiosensitive and because they have more remaining years of life during which a radiation-induced cancer could develop.²⁹

Although many providers routinely perform CT scans on every patient with new-onset seizures, this practice has been called into question. In 1997, Warden and colleagues³⁰ recommended that emergent neuroimaging be reserved for patients with a prolonged postictal period, status epilepticus, age less than 6 months, new-onset focal neurologic defects, recent head injury, patients with ventriculoperitoneal shunts, or other neurocutaneous disorders. In 1998, Garvey and colleagues³¹ conducted a retrospective analysis of 107 neurologically normal patients who underwent neuroimaging in the ED for "first seizure": 8 of 107 had nonepileptic events (gastroesophageal reflux, syncope, and rigor). Of the remaining 99 patients, 49 had provoked seizures and 50 had unprovoked seizures: 19 of 99 patients had CT abnormalities, and 9 of 49 with provoked seizures had abnormalities on CT, but none required intervention (mild hydrocephalus, angioma, asymmetry, periventricular leukomalacia). Ten of 50 patients with unprovoked seizures had CT abnormalities, with 7 receiving further investigation or interventions: 2 had tumors, 3 had vascular abnormalities, 1 had cysticercosis, and 1 had obstructive hydrocephalus. In this study, CT scan abnormalities requiring treatment or monitoring were more often seen in children with unprovoked seizures (P<.01) and in children with focal seizures or focal neurologic findings (P<.04).

Sharma and colleagues³² reviewed 500 patients with new-onset seizures, of whom 475 underwent neuroimaging. The mean patient age was 62 months (range 0–21 months). Focal seizures were present in 33% of patients. Risk factors for neuroimaging abnormalities included the presence of a predisposing condition or a focal seizure in children younger than 33 months. Predisposing conditions were sickle cell disease, bleeding disorders, cerebral vascular disease, malignancy, human immunodeficiency virus (HIV), hydrocephalus, travel to areas with cysticercosis, closed head injury, or the presence of hemihypertrophy. Clinically significant findings were present in 8% (38/475) of patients who underwent neuroimaging. Twenty-six percent (32/121) of high-risk patients had abnormalities versus 2% (6/354) of the low-risk patients. The investigators concluded that emergent imaging should be performed only in patients with high-risk criteria. Furthermore, they advised that if follow-up can be obtained, low-risk patients can be discharged without immediate ED imaging.

A practice guideline written jointly by the Quality Standards Subcommittee of the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society concur that there is insufficient evidence to support the routine performance of neuroimaging in children with a first unprovoked nonfebrile seizure.²³

Bedside brain ultrasonography is often the imaging study of choice in neonatal seizures, due to challenges of transporting critically ill neonates to CT or MRI. The

disadvantage of ultrasonography is the poor detection of cortical lesions and subarachnoid blood.

ELECTROENCEPHALOGRAPHY

An EEG is rarely needed in the ED setting, except for patients with refractory seizures or in patients in whom the diagnosis of nonconvulsive status epilepticus is being considered. Well-appearing children who have experienced a first-time afebrile seizure can be managed as outpatients, with an EEG arranged by the primary care physician. In idiopathic and cryptogenic seizures, the EEG has been found to be the most important predictor of recurrence, with a 2-year recurrence rate of 58% in patients with an abnormal EEG compared with a 28% seizure recurrence rate in patients with a normal EEG.³³ Of note, a normal EEG does not rule out a seizure disorder or other underlying neurologic disorder.

Portable EEG monitoring is now available in many EDs and pediatric intensive care units, and can be used to determine treatment response.

ANTIEPILEPTIC AGENTS

In a critical review of the literature, the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society found that although anticonvulsant agents decrease the incidence of second seizures, they do not decrease the long-term risk of developing epilepsy.²³ Therefore, they recommend that for stable, well-appearing children who have experienced a single seizure that did not require emergent anticonvulsant therapy, maintenance medications are not initiated. Patients who experience recurrent seizures should be started on an antiepileptic medication. The decision to initiate long-term anticonvulsant therapy should be made in conjunction with a pediatric neurologist or the patient's primary care physician. The most common agents used to treat afebrile seizures are listed in **Table 2**. Felbamate is not commonly used because of the availability of safer antiepileptic agents, and is indicated only for cases of severe refractory seizures (Lennox-Gastault syndrome) where the benefit clearly outweighs the risk of liver toxicity and aplastic anemia.

NEONATAL SEIZURES

It is often difficult in the newborn to differentiate between a seizure from other conditions, especially because newborn seizures can present in a variety of different ways including apnea, subtle eye deviations, or abnormal chewing movements. In addition, associated autonomic system findings seen with older patients with seizures may not be seen. Useful tips in differentiating between a newborn with seizures and a "jittery baby" are that true seizures cannot be suppressed by passive restraint and seizures cannot be elicited by motion or startling.

The most common cause of a seizure in the first 3 days of life is perinatal hypoxia or anoxia. Approximately 50% to 65% of newborn seizures are due to hypoxic-ischemic encephalopathy.³⁴ Intraventricular, subdural, and subarachnoid hemorrhages account for 15% of newborn seizures, and an additional 10% are caused by inborn errors of metabolism, sepsis, metabolic disorders, and toxins (**Box 2**).³⁵ Pyridoxine deficiency is an autosomal recessive disorder that is a rare cause of seizures, and usually presents in the first 1 to 2 days of life.³⁶

Benign familial neonatal convulsions and benign idiopathic neonatal convulsions are 2 types of neonatal seizures that are benign and carry a favorable prognosis.

102

Table 2 Common anticonvulsant agents				
Drug	Type of Seizure	Side Effects	Maintenance	
Carbamazepine (Tegretol)	Generalized tonic-clonic, partial, benign Rolandic seizures	Rash, liver disease, diplopia, aplastic anemia, leukopenia	10–40 mg/kg divided bid or tid	
Clonazepam (Rivotril, Klonopin)	Myoclonic, akinetic, infantile spasms, partial, Lennox-Gastault	Fatigue, behavioral issues, salivation	0.05–0.3 mg/kg/d divided bid or tid	
Ethosuximide (Zarontin)	Absence	GI upset, weight gain, lethargy, SLE, rash	20–40 mg/kg/d divided qd or bid	
Gabapentin (Neurontin)	Partial and secondarily generalized seizures	Fatigue, dizziness, diarrhea, ataxia	20–70 mg/kg/d	
Lamotrigine (Lamictal)	Complex partial (atypical absence), Lennox-Gastaut, myoclonic, absence, tonic-clonic	Headache, nausea, rash, Stevens- Johnson syndrome, lymphadenopathy, diplopia, GI upset	10–12 mg/kg/d if given as monotherapy, 2–5 mg/kg/d if given with valproic acid	
Levetiracetam (Keppra)	Partial-onset seizures in children >4 y, generalized tonic-clonic seizures in children >6 y, juvenile myoclonic seizures in children >12 y	Dizziness, somnolence, headache	20 mg/kg/d divided bid. Daily dose increased every 2 wk by increments of 20 mg/kg to the recommended daily dose of 60 mg/kg (30 mg/kg bid)	
Phenobarbital (Luminol)	Generalized tonic-clonic, partial	Sedation, behavioral issues	2–6 mg/kg/d divided qd or bid	
Phenytoin (Dilantin)	Generalized tonic-clonic, partial	Gingival hyperplasia, hirsutism, rash, Stevens-Johnson syndrome, lymphoma	4–8 mg/kg/d divided bid, tid, or qhs	

Primidone (Mysoline)	Generalized tonic-clonic, partial	Rash, ataxia, behavioral issues, sedation, anemia	10–25 mg/kg/d divided bid, tid, or qid
Topiramate (Topamax)	Refractory complex partial seizures, infantile spasms, adjunctive therapy for temporal lobe epilepsy	Fatigue, nephrolithiasis, ataxia, headache, tremor, GI upset	1–9 mg/kg/d
Tiagabine (Gabitril)	Adjunctive therapy for refractory complex partial (focal) seizures	Decreased attention span, tremor, dizziness, anorexia	Average dose 6 mg/d
Valproic acid (Epicene, Epical)	Generalized tonic-clonic, absence, myoclonic, partial, akinetic, juvenile myoclonic epilepsy of Janz, infantile spasms	Gl upset, liver involvement, alopecia, sedation	10–60 mg/kg/d divided tid or qid
Vigabatrin (Sabril)	Infantile spasms, adjunctive therapy for refractory seizures	Weight gain, agitation, depression, behavioral changes, visual field constriction, optic neuritis	30–100 mg/kg/d divided qd or bid
Oxcarbazepine (Trileptal)	Partial/focal seizures	Hyponatremia, hepatic or blood dyscrasias	20–40 mg/kg/d
Zonis amide (zonegran)	Adjunctive therapy for partial or general seizures	Rash, renal calculi, photosensitivity	Begin with 1–2 mg/kg/d

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Abbreviations: bid, twice a day; GI, gastrointestinal; SLE, systemic lupus erythematosus; tid, 3 times a day; qd, every day; qhs, every bedtime; qid, 4 times a day.

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Congenital anomalies	Hypertension		
	Congenital anomalies		
Developmental brain disorders	Developmental brain disorders		
Benign idiopathic neonatal seizures	Benign idiopathic neonatal seizures		

The etiology is unknown. Benign familial neonatal convulsions typically present in the first 3 days of life, and there is a strong family history of epilepsy or neonatal seizures. These seizures resolve by 1 to 6 months of age. Benign idiopathic neonatal convulsions, also known as "fifth-day fits," occur on the fifth day of life

105

and cease on day 15 of life.³⁶ These diagnoses should be ones of exclusion. The workup should be tailored to the patient but may include cranial ultrasonography, a head CT or MRI scan, electrolytes, glucose, calcium, magnesium, toxicology screen, urinalysis and culture, complete blood count and culture, and cerebrospinal fluid (CSF) studies. If an inborn error of metabolism is suspected, then blood for amino acids, lactate, pyruvate and ammonia levels should be obtained, as well as urine for organic acids.

In neonates who are actively seizing, treatment includes attention to the airway, breathing, and circulation. Benzodiazepines are often given as the first line of treatment, followed by phenobarbital, fosphenytoin, or phenytoin, all of which have risks and benefits.³⁴ Benzodiazepines have been associated with serious adverse effects such as hypotension and respiratory depression in preterm and term infants, and therefore should be used with caution.³⁷ Phenytoin is not the preferred initial agent, as it has a depressive effect on the newborn myocardium and has an unpredictable rate of metabolism due to immature hepatic function.³³ Pyridoxine (50-100 mg IV) or lidocaine may be used if refractory seizures are present (see Table 1).³⁶ If the seizure is a result of an electrolyte abnormality such as hyponatremia, hypocalcemia, or hypomagnesemia, these abnormalities should be rapidly identified and treated. In patients who are actively seizing, hyponatremia can be treated with normal saline (0.9%) boluses or alternatively with 5 to 10 mL/kg IV of 3% saline (this can be administered as a bolus given over 10 minutes). If the patient has hypocalcemia (plasma calcium <7.0 mg/dL), then 100 to 300 mg/kg of IV 10% calcium gluconate should be infused. In cases of hypomagnesemia (serum magnesium <1 mEq/L), 0.1 to 0.3 mL/kg of 50% magnesium sulfate should be given IV IV or IM. Ampicillin and cefotaxime should be initiated in any patient considered to have sepsis. Acyclovir (20 mg/kg every 8 h IV) should also be administered if there is a positive maternal history of herpes, if the patient has a vesicular rash, focal neurologic findings, or a CSF pleocytosis or elevated CSF protein without organisms on Gram stain.³⁸ Patients should be admitted to a monitored bed for further observation and evaluation.

FUTURE DIRECTIONS

Future research needs must focus on:

- Neuroimaging safety and guidelines for imaging
- Safe antiepileptic drugs for children
- Improved access to pediatric neurologic evaluation for the subset of children with difficult to manage and refractory afebrile seizures.

SUMMARY

Afebrile seizures in children are common and often recur. Fortunately, most children with childhood epilepsy have a favorable long-term prognosis. In particular, patients with idiopathic etiology usually reach remission.³⁹ There are specific types of afebrile seizure disorders that emergency physicians should be aware of, with absence seizures being the most common. Newborn seizures are often difficult to diagnose, and are evaluated and treated more aggressively than afebrile seizures in older infants and children. Children that present to the ED often have a known seizure disorder, are taking medications for their disorder, and usually are in a postictal state on arrival. Seizures lasting longer than 5 minutes should be treated initially with a benzodiazepine and standard advanced life support protocols. Laboratory studies are needed only in children younger than 6 months, in patients with

prolonged seizures or altered level of consciousness, or in patients with history of a metabolic disorder or dehydration. Routine neuroimaging is not recommended in children with a first unprovoked afebrile seizure, although imaging studies should be considered in children younger than 3 years with a predisposing condition or focal seizures. Most well-appearing children can be managed as outpatients after a first afebrile seizure, with instructions for an outpatient EEG and follow-up by the primary care physician. Anticonvulsant drugs do not decrease the long-term incidence of epilepsy and are therefore not usually recommended after a first afebrile seizure. New anticonvulsant drugs continue to be investigated, but it is important to recognize that no anticonvulsive agents decrease the long-term incidence of epilepsy and are therefore not usually recommended after a first afebrile seizure. Adjunct nonpharmacologic therapies such as vagal nerve stimulation are also being used in patients with severe epilepsy. Intermittent electrical stimulation is delivered to the cervical vagus nerve. The lead is usually located on the left side of the neck, and the generator is implanted in the chest wall. The emergency provider should keep abreast of new technologies and emerging trends in pharmacologic antiepileptic management.

KEY CONCEPTS

- An EEG should be performed as soon as possible on patients with an apparent first unprovoked seizure.
- Electrolyte testing is not routinely necessary on well-appearing children older than 6 months.
- Emergent neuroimaging of children with first-time seizures should be performed on patients with the following risk factors: focal seizures, prolonged postictal period, status epilepticus, sickle cell disease, immunocompromise, head injury, age less than 6 months to 1 year, ventriculoperitoneal shunts, recent travel to an area endemic for cysticercosis, bleeding disorders, cerebral vascular disease, neurocutaneous disorders, malignancy, HIV, or hydrocephalus.
- Well-appearing children who have experienced a first unprovoked seizure and are in the low-risk category do not need emergent neuroimaging if they have close outpatient follow-up.
- Children on ketogenic diets should not be given dextrose empirically.

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