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THEME ARTICLE

An update in pediatric seizure disorders

Linda P. Nelson, DMD, MScD Steven D. Ureles, DMD, MS Gregory Holmes, MD

Abstract

Recent advances in knowledge of pediatric seizure disorders, including classification of seizure types and febrile convulsions, have been the topics of major symposia. In light of these recent developments, an in-depth review is presented to aid the pediatric dentist in the treatment of these children.

Introduction

Epilepsy is so common, that it is likely that pediatric dentists will have children in their practices with the disorder. Pediatric dentists must be aware of the clinical features of this disorder and the behavior management problems that may occur in this population. This paper will review and update the behavior manifestations and current treatment of childhood epilepsy.

Seizures are among the most frequently observed neurologic disorders in children, with as many as 4% of all children having had at least one seizure during the first 15 years of life. Recurrent seizures affect about 0.5% of children (Dodson et al. 1976, Part I). By definition, a seizure is the clinical manifestation of abnormal neuronal hyperactivity, and its location determines the type of seizure. Epilepsy is defined as recurrent, unprovoked seizures. One seizure does not constitute epilepsy. Similarly, recurrent provoked seizures, i.e. febrile seizures, do not mean that the child has epilepsy. While epilepsy means recurrent, unprovoked seizures, it needn't be a lifelong disorder. Approximately 60–70% of children with epilepsy outgrow the disorder. Seizures are symptoms of underlying disease and as such can be the mode of expression for many diseases. Tables 1 and 2 (next page) give the pediatric dentist a glossary of useful terms.

The features of seizure disorders in childhood are sufficiently distinctive to deserve separate discussion. Neonatal seizures, infantile spasms, febrile convulsions, and the absence seizure syndrome are specific childhood seizure types. The etiology of pediatric seizures can be divided into two groups: symptomatic and idiopathic. In symptomatic epilepsy, an etiological factor can be identified while in the idiopathic group, no etiologic factors can be detected. Etiological agents in children differ from those in adults. Seizures in adults most frequently are caused by brain tumors, cerebrovascular accidents, and trauma, while in children, congenital brain malformations, infections, metabolic disease, hypoxic-ischemic injuries, and inherited disorders are more common. Contrary to the belief of the lay public, brain tumors rarely cause seizures in children.

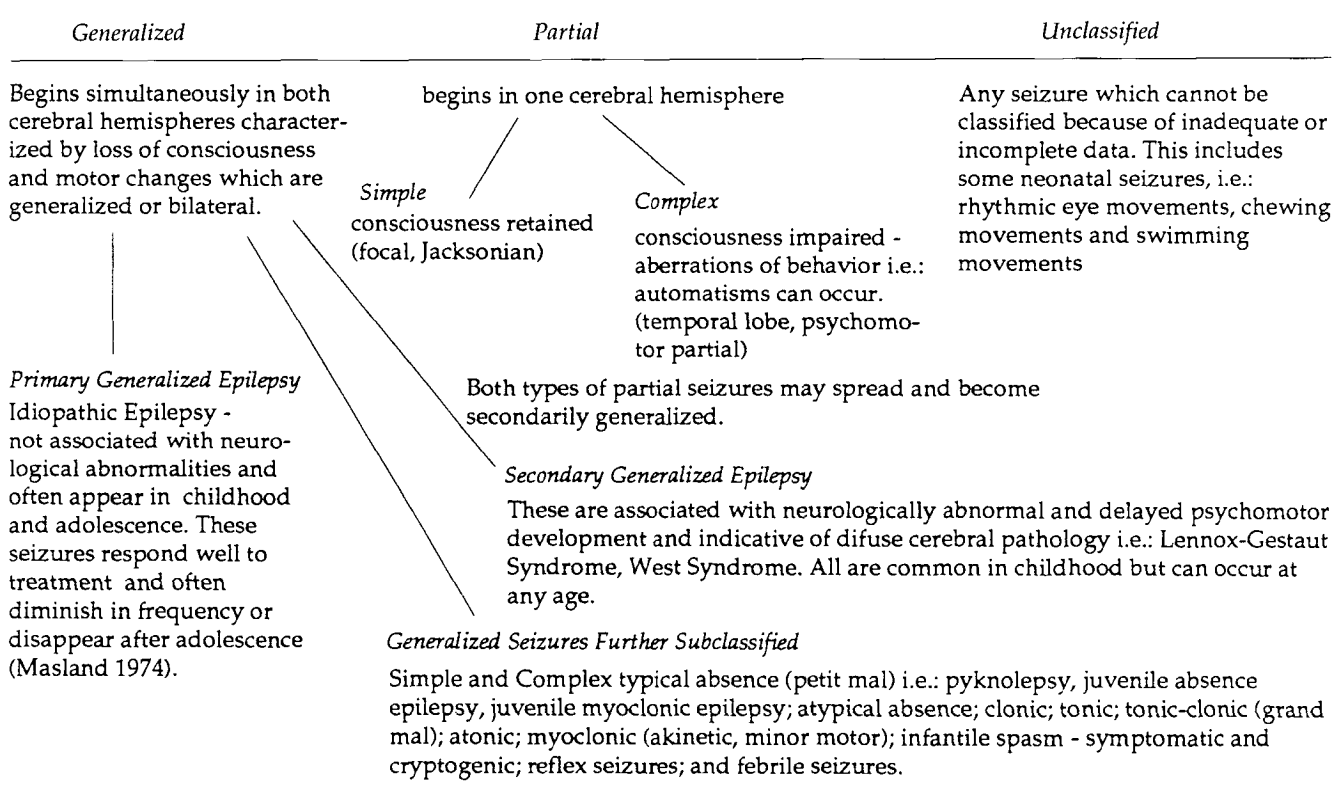
Classification

Seizures first were classified according to their clinical features. Our understanding that seizures can be symptoms of disease either within or outside the nervous system, and that seizures may reflect the location of abnormal neuronal activity, has caused a continuous reclassification of seizures and epilepsy (Farmer and Greenwood 1983). Table 2 shows an overview of the classification system. Seizures are partial, generalized, or unclassified, according to the electrophysiologic source of discharge and video monitoring techniques.

Table 1. Glossary of Terms

Term	*Definition*
Epilepsy	Recurrent unprovoked seizures
Convulsions	Seizures with motor manifestations
Myoclonic	Brief body jerks
Tonic	Continuous contraction(s)
Clonic	Alternating contraction and relaxation
Atonic	Loss of tone
Akinetic	Motionless
Typical Absences	
Simple	staring episodes with impairment of consciousness
Complex	staring episodes, impairment of consciousness, automatisms, clonic activity, changes in postural tone

Table 2. Classification of seizures (Dreifuss 1989)



Partial seizures begin in one cerebral hemisphere and are subclassified as simple (if consciousness is retained) or complex (if consciousness is altered). Generalized seizures begin simultaneously in both hemispheres and are characterized by loss of consciousness and motor changes which are generalized or bilateral. The generalized seizures are subdivided into primary and secondary generalized epilepsies. Primary generalized epilepsies, called idiopathic epilepsy, are not associated with neurological abnormalities. These seizures respond well to treatment and often diminish in frequency or disappear after adolescence (Masland 1974). Secondary generalized epilepsy is associated with neurologically abnormal and delayed psychomotor development and is indicative of diffuse cerebral pathology (i.e. Lennox-Gastaut syndrome and West syndrome). These seizures are commonly seen in childhood but can occur at any age. Generalized seizures are further subclassified as simple and complex typical absence (petit mal), atypical absence, tonic, clonic, tonic-clonic (grand mal), atonic seizures, akinetic seizures, bilateral myoclonic, and infantile spasms. The nomenclature can become very confusing, especially when a simple partial seizure progresses to a secondarily generalized seizure.

An unclassified category includes seizures which cannot be classified because of inadequate or incomplete data (Bresnan and Konkol 1987).

Generalized Seizures

Tonic-Clonic Seizures (Formerly Termed Grand Mal Seizures)

The generalized tonic-clonic seizure involves all extremities and has both tonic and clonic features. Ten per cent of adolescent and adult patients with epilepsy suffer from tonic-clonic seizures (Gastaut et al 1974). These seizures usually are not present in infants due to relatively unmyelinated neurons and reduced neuronal excitability of the CNS (Vernadakis and Woodbury 1969). Most generalized tonic-clonic seizures begin as a partial seizure and then become generalized secondarily. An aura is, in fact, a simple partial seizure. If a patient has an aura before a generalized tonic-clonic seizure it means that the seizure began partially and then generalized.

Tonic-clonic seizures usually begin with a bilateral generalized massive myoclonic muscular jerk, which lasts for several seconds and is often accompanied by a jerky cry. The person is propelled forward or backward and may fall to the ground.

The next phase is a series of both tonic and clonic motor and autonomic changes. The tonic phase consists of a sustained muscular contraction lasting 20–30 sec which usually consists of a brief phase of flexion followed by a longer period of extension. During the tonic flexion

phase, consciousness is lost abruptly. The tonic contraction of the elevator and depressor muscles of mastication occurs equally and keeps the mouth open rigidly. The extension tonic phase then begins with the mouth opening wide and snapping shut, the arms and legs extending, and a prolonged steady expiration forcing air across the spasmodic glottis as the thoracic and abdominal muscles contract. This can lead to apnea, and the patient may not begin respiration again until the postictal phase (Masland 1974).

The clonic phase consists of 30 sec of alternating rhythmic contractions and relaxations of the muscles, appearing clinically as a succession of brief and violent flexor spasms of the entire body. Tongue biting can occur. The jerks gradually subside until the end of the seizure, as the period of muscular atonia becomes progressively longer (Farmer and Greenwood 1983).

Immediate postictal features lasting several seconds to 4 min include tonic contractions of the facial muscles and muscles of mastication, producing an intense trismus and frothy saliva (Masland 1974). Postictal features include complete atonia (flaccidity) and incontinence. The patient awakens confused and then usually falls into a deep sleep, emerging with a headache and complete retrograde amnesia.

The onset of idiopathic primary generalized epilepsy is around 5 years of age, but generalized seizures caused by CNS damage may have an earlier onset. Generalized tonic-clonic seizures with focal origin usually stem from cerebral damage due to infection, birth injury, dysgenesis (defective embryonic development), metabolic and degenerative disease, or tumors (Bresnan and Konkol 1987).

No attempt should be made to move the patient who has a tonic-clonic seizure in the dental chair. The chair should be tilted back into the reclined position, and the patient's head placed to one side to prevent aspiration of saliva. Clothing around the neck should be loosened and oxygen administered if signs of anoxia occur. Although tongue biting is a concern, more damage is caused by forcing objects between the teeth; this practice is not recommended. If one is cognizant of an impending seizure, then a towel can be placed between the dental arches at the beginning of the tonic phase, when the mouth is open. If a patient bites on an instrument or rubber dam clamp lodging between the teeth, wait until the seizure subsides before attempting to remove the object. The general rule to follow if a tonic-clonic seizure occurs is to prevent the factitional injury (Braham et al 1977).

Absence Seizures (Formerly Termed Petit Mal Seizures)

Absence seizures are generalized seizures that are nonconvulsive and usually not associated with underlying structural pathology. The hallmark of the typical simple absence seizure is suppression of mental function, usually to the point of complete abolition of responsiveness, awareness, and memory (Holmes et al 1987). The seizures always begin abruptly, without an aura or warning, and generally last a few seconds. Atypical absence seizures may last a minute or more. Activity suddenly is interrupted, and the patient changes facial expression and becomes transfixed with a motionless, distant expression. The absence seizure ends suddenly, and the child returns to the gesture, sentence, or other interrupted activity. Postictal impairment never occurs although the child may be confused momentarily, due to time loss. This time loss may serve as a clue to the child that a seizure had occurred.

In simple absence seizures, the child changes facial expression, stares and has a momentary impairment of consciousness, while in complex seizures, a variety of other activities, such as, automatisms, myoclonus, and changes in postural tone occur. Complex absence seizures are much more common than simple absence seizures (Penry et al. 1975).

Absence seizures may be precipitated by hyperventilation and photic stimulation.

Atypical Absences

Atypical absences differ from typical absences by a longer duration and more changes in postural tone. The children often have other seizure types in addition to the atypical absence seizures. These seizures are much more difficult to treat than typical absences (Holmes et al. 1987).

Some of these seizures can be associated with deterioration of intellectual performance and eventual psychomotor retardation. Most children with atypical absences have the Lennox-Gastaut syndrome, characterized by mental deterioration and a mixed seizure disorder (Gastaut et al. 1966; Markand 1977; O'Donohoe 1979; Bresnan and Konkol 1987). These seizures are difficult to control with medication. Ketogenic diet using medium chain triglycerides is tried sometimes (Dodson et al. 1976).

The dental implication of absence seizures is the need to reacclimate the child to the treatment being rendered after the seizure.

Infantile Spasms

West first described infantile spasms in 1841 as massive myoclonic jerks beginning in infancy (West 1841). Infantile spasms can be divided into flexor, extensor, or mixed types. Flexor spasms are characterized by sudden flexion of the trunk, neck, hips, and arms, while in extensor spasms the legs and arms extend. In mixed spasms, elements of the flexor and extensor spasms combine. Infantile spasms are brief, lasting 1–5

sec, and usually occur in clusters. While the clusters typically consist of 10–15 spasms over several minutes, some infants can have a hundred or more spasms in a cluster. The untreated child usually has multiple clusters daily. Infantile spasms are characterized by recurring attacks and often associated with mental retardation and hypsarrhythmia (chaotic rhythm EEG, Lacy and Penry 1976). This triad of spasms, retardation, and hypsarrhythmia is known as West syndrome.

Infantile spasms may be symptomatic (occurring in infants with a history of CNS defects and disorders of prenatal, perinatal, and postnatal origin) or classified as cryptogenic (occurring in infants without neurological symptoms). Ultimately, 90% of all children with infantile spasms become mentally retarded and usually have a mixed seizure disorder of myoclonic-akinetic and other generalized seizures (Weiner et al. 1988).

It is generally thought that early diagnosis and treatment leads to a better prognosis. A normal infant with a normal head size who begins having spasms has the best prognosis. Although the incidence is low, 0.24/1000 live births (Nelson 1972), an early diagnosis is extremely important, as prompt treatment with intramuscular long acting adrenocorticotropic hormone (ACTH) may prevent permanent encephalopathy, severe retardation, and generalized seizures (Gastaut 1974). Infantile spasms continue to be one of the more resistant forms of epilepsy to antiepileptic therapy.

Febrile Convulsions

Febrile convulsions are seizures that occur with fever and infections. Within the first 5 years of life, 7% of all children will have a seizure, and 50% or more of these will be febrile seizures (Bresnan and Konkol 1987). These seizures typically occur between 6–24 months of age. Ninety per cent of these seizures are generalized, of short duration (15 min or less) and predominantly tonic. As the temperature exceeds 102°F (39°C), there is an increased risk for convulsion. A positive family history of convulsions and genetic studies suggest autosomal dominant inheritance with incomplete penetrance (Bresnan and Konkol 1987).

Three factors increase the risk of subsequently developing epilepsy after febrile seizures:

1. Duration longer than 15 min, focal in character, or more than one seizure in 24 hr
2. Preexisting neurological abnormality
3. Family history of afebrile seizures (epilepsy in parents or siblings).

Two of these risk factors present a 10% chance of developing epilepsy. The recurrence of simple febrile seizures also increases the risk of subsequent afebrile seizures to 4% (Nelson and Ellenberg 1978). The overall recurrence rate of simple febrile seizures is 30–40% (Emerson 1981), but age of onset appears to be an important factor in determining the frequency of recurrences. Fifty per cent of children who have a seizure by one year of age will have a another one (Dreifuss 1983; Weiner et al 1988).

The goal of treatment is to prevent recurrence of febrile seizures. Typical management of a febrile seizure includes antipyretics to decrease temperature and fluid replacement therapy if the patient is dehydrated. Most febrile seizures do not need medication, but anticonvulsant therapy is considered if there is an abnormal neurological exam, prolonged focal seizure (greater than 15 min), or association with a neurological deficit. Phenobarbital taken daily prevents recurrent febrile seizures. However, it has been demonstrated recently that phenobarbital lowers the IQ in children treated for febrile seizures, and is ineffective in preventing recurrences of febrile or afebrile seizures (Farwell et al. 1990).

Status Epilepticus

Most investigators define status epilepticus as continued seizure activity lasting longer than 30 min and resulting in a fixed epileptic condition. Status epilepticus is an emergency situation, regardless of its clinical form. If it lasts untreated for an hour, the patient may never regain consciousness (Gastaut 1970; Dodson et al. 1976; Abramawicz 1983). Diazepam, diazepam with simultaneous administration of phenytoin, or phenobarbital are first-line medications in treatment.

Partial Seizures

Simple Partial Seizures

Partial seizures with simple symptoms (focal seizures) consist of motor movements or sensory phenomena limited to one limb, or they may involve both limbs on one side of the body (Dreifuss 1983). There may be a spread of motor activity (jacksonian march). A jacksonian seizure may begin, for example, with twitching of the thumb followed by twitching of the hand, forearm, upper arm, and shoulder. The most common causes are focal damage (cerebral palsy), tumor, arteriovenous malformation, and abscess. In neonates and infants, metabolic seizures, may occasionally be focal (Hoekelman et al. 1987).

Complex Partial Seizures

This focal seizure disorder may or may not be secondary to a lesion in the temporal lobe. Complex partial seizures frequently begin in the temporal lobe, but may start in frontal, occipital, or other cortical regions. Causes include birth trauma, prolonged febrile convulsions, tumor, and hamartomas. Partial seizures as a part of a mixed seizure disorder secondary to a diffuse fixed encephalopathy are probably the most common. There

frequently is an aura, often described by children as "funny feelings," fearful feelings, "butterflies," or abdominal pain. The seizure may take the form of automatisms, automatic movements and tasks such as swallowing or lip smacking, a repetitive motion with the hands (picking at one's clothes), behavior abnormalities (walking to another part of the room), or lack of responsiveness. Amnesia, confusion, and drowsiness accompany the episodes. Complex partial seizures may become generalized to a tonic-clonic seizure (Weiner et al. 1988; Bresnan and Konkol 1987).

Benign Rolandic Epilepsy

Simple partial seizures are unusual in childhood. One notable exception is Benign Rolandic Epilepsy (BRE), also known as Sylvian seizures and midtemporal spikes, or benign epilepsy of childhood with centrotemporal EEG foci. These seizures tend to begin late in childhood and disappear at or just after adolescence. BRE makes up 16–24% of childhood seizures (Cavazzuti 1980). They are quite recognizable by the clinical characteristics of onset during sleep of brief facial clonus, salivary pooling, and anarthria (loss of power of articulate speech). It has been concluded that BRE is the result of autosomal dominant inheritance with an age-dependent penetrance. Almost all children outgrow these seizures with age (Bresnan and Konkol 1987; Farmer and Greenwood 1983).

Neonatal Seizures

Neonatal seizures are a major prognostic indicator of children who will later have both severe mental retardation and cerebral palsy (Nelson and Broman 1977). Seizures with an onset during the neonatal period have different characteristics, causes, treatment, and prognosis than seizures that occur in an older infant. This is related to the immaturity of myelination and synaptic development. Seizures are seen in fewer than 1% of full-term and in more than 5% of premature neonates, and are the most common manifestation of neurologic difficulties during the neonatal period. These seizures are related to underlying abnormalities as listed in Table 3.

These seizures are confusing because of difficulty in distinguishing them from jitteriness. Jitteriness differs from seizures by being stimulus sensitive, ceasing when the extremity is held, and not being associated with abnormal eye movements. The most effective treatment of neonatal seizures is prevention. Prenatal and intrapartum monitoring and appropriate resuscitation following delivery may prevent brain damage. Once seizures start, intravenous solutions are started to correct any metabolic disorder. If anticonvulsants are necessary, phenobarbital is the drug of choice in the neonate. The prognosis is good. In the follow-up of the Collaborative Perinatal Project, 70% of the 181 who survived following neonatal seizures were developmentally normal at age 7 years (Myers and Cassady 1983; Volpe 1977).

Table 3. Common etiologies of neonatal seizures

Day 1
Hypoxia
Hypoglycemia
Hyperglycemia
Drug withdrawal (from mother)
Pyridoxine dependency
Infection (meningitis, sepsis, TORCH)
Trauma, severe
Days 2-3
Infection (sepsis, meningitis)
Drug withdrawal
Developmental malformations
Intracranial hemorrhage
Metabolic disorders (urea cycle, hyperglycinemia)
Hyperglycemia
Hypoglycemia
Hyponatremia or hypernatremia
Day 4 on
Developmental malformations
Hypocalcemia and hyperphosphatemia (dietary)
Hyponatremia
Metabolic (urea cycle or amino acid disorders, hyperglycinemia)
Infection (sepsis, meningitis, herpes simplex)
Drug withdrawal

Diagnostic Aids for the Pediatric Dentist To Evaluate Patients With Seizures

Physical Exam

The pediatric dentist rarely has the opportunity to observe a seizure at the time of onset; so a careful history is important. Included in the review of past and present medical history should be a discussion concerning family history of seizures, complications at birth, prewarning signs of seizures (auras) experienced, the manner in which seizure progresses, whether consciousness is lost, postictal behavior, the age seizures began, how often seizures occurs, whether seizures have changed in character, the medication currently used, and its effectiveness.

If the child has no seizure history and has one seizure in the dental office, the child should be examined by a

physician following the seizure. The physical examination should include a thorough neurological exam and evaluation for injury. These children often will require an electroencephalogram (EEG), and the pediatric dentist should understand its usefulness. The EEG is a diagnostic aid for patients with suspected severe seizure disorders because it frequently shows evidence of a paroxysmal electrical dysrhythmia (Farmer and Greenwood 1983). The EEG can reveal the nature and location of brain cell overactivity, identify structural abnormality, provide clues as to the clinical seizure type, and may predict recurrences (1985). A normal EEG does not rule out epilepsy, and many children with a normal waking EEG can have an abnormality induced by hyperventilation, photic stimulation, or a spontaneous arousal during natural sleep or sedation. Almost all children, with the exception of children with typical absence seizures, will have computerized tomography (CT) or magnetic resonance imaging (MRI) scans obtained. The "yield" of the MRI over CT is about 10%, making it a superior test that eventually will replace the CT.

Pharmacotherapy

Children are not placed on antiepileptic drugs until they have two or more unprovoked seizures. Frequently, a child will only have a single seizure, and treatment with potentially harmful drugs is not required. Some neurologists do not treat children with more than two seizures if they are brief, non life-threatening, and occur at a frequency of less than two per year. The chief reason for treating a child after a second unprovoked seizure is to prevent the associated emotional and physical injury. Children who have frequent or prolonged seizures require anticonvulsant therapy.

The management of the child with a seizure disorder can best be summarized by "attempt to cause no harm." Anticonvulsant therapy should preserve vital functions while controlling seizure activity. If possible, treatment of the underlying disease is a goal of therapy while preventing damage to the brain. Therapeutic approaches to convulsive seizures are summarized in Table 4. In the majority of the children with seizure disorders, no specific cause is found. Difficulties of drug treatment include poor prognosis, prolonged multidrug treatment,

Table 4. Therapeutic approaches to convulsive seizures

Major Drug	*Type of Seizure*	*Toxic Side Effects*	*Doses*	*Drug Interactions of Interest to the Pediatric Dentist*
Carbamazepine (Tegretol) (drug of choice)	partial seizures	Leukopenia hepatic dysfunction rashes	≤ 8 years 100 mg BID or TID v ≥ 8 years 100-200 mg *TID or QID* Therapeutic levels in 15–30 hr.	Erythromycin will elevate tegretol levels and thus its use must be monitored carefully.
Valproic acid (Depakote)	absence mal	hepatotoxicity pancreatitis	15–60 mg/kg/day medication administered every 6–12 hr to maintain blood levels. Therapeutic levels in 6–15 hr.	Valproic acid may cause prolonged bleeding times.
Clonazepam (Klonopin)	atypical absences infantile spasms	behavioral changes hyperactivity, irritability, aggressive behavior in 15% of patients. Thick speech, salivation, and bronchial hypersecretion	≤ 10 years (30 kg) 0.05 mg/kg/day then increased by 0.25 to 0.5 mg/kg/day to a maximum of 0.2 mg/kg/day in 3 divided doses.	Do not stop Clonazepam therapy suddenly or status epilepticus may be precipitated.
Phenobarbital	febrile seizures (although rarely treated)	behavioral side effects may interfere with cognition hyperactivity	30 mg BID (children) 60 mg BID (teenagers) Therapeutic levels in 1-3 weeks.	Sedatives and phenobarbital can lead to over-sedation. Also true for primidone (mysoline).
Phenytoin (Dilantin)	tonic-clonic or focal seizures	maculo pappular rash, Stevens-Johnson syndrome, hirsutism, gingival hypertrophy	6 mg/kg/day up to 30kg in children 200–300 mg/day in teenagers Therapeutic levels in	Increased incidence of cleft lip/palate in children of mothers taking phenytoin.

chronic toxicity, and uncertainty of the relative efficacy and toxicity of individual anticonvulsants. Compliance is a major problem. Because of the number of drugs prescribed, their unwanted side effects, and the prolonged treatment, some patients omit some or all of their medications (Reynolds 1978).

Numerous anticonvulsant medications are available, but multidrug regimens (polypharmacy) are usually avoided if possible. In children, carbamazepine is the current drug of choice of pediatric neurologists for partial seizures and valproic acid for primarily generalized seizures.

Phenobarbital is used widely by pediatricians. Phenobarbital is no longer the first drug used in the treatment of seizures in children by most neurologists although pediatricians, by habit, continue to use phenobarbital as their drug of choice. Although safe, phenobarbitol's main disadvantages are behavioral side effects and interference with cognition (Farwell 1990).

Phenytoin is another effective anticonvulsant. A maculopapular rash (resembling measles) is a side effect which may lead to Stevens-Johnson syndrome if medication continues. Other side effects include hirsutism, gingival hypertrophy, blood dyscrasias secondary to bone marrow toxicity, megaloblastic anemia (folate deficiency), and rickets or osteomalacia (low calcium and high alkaline phosphatase). Although nystagmus on lateral gaze is a good clinical sign in teenagers that they are taking the medication, it is not present with regularity in children. Ataxia of gait is a common manifestation of toxicity in all age groups (Weiner et al. 1988). The method of action of phenytoin is probably related to preventing the spread of seizure activity rather than abolishing the primary focus of seizure discharges (Farmer and Greenwood 1983).

Carbamazepine is becoming increasingly popular for partial seizures and generalized seizures because of its lack of side effects. Toxic side effects include leukopenia, hepatic dysfunction and rashes. The pediatric dentist should be aware that erythromycin will elevate carbamazepine levels (Weiner et al. 1988).

Valproic acid is most effective in absence, myoclonic, tonic, atonic and primary generalized tonic-clonic seizures. The pediatric dentist should be aware that valproic acid may lead to bleeding abnormalities (Browne 1980; Bresnan and Konkol 1987; Weiner et al. 1988).

Clonazepam is primarily used in atypical absences and infantile spasms. Hyperactivity, irritability, and aggressive behavior occur in about 15% of patients. Other toxic symptoms include thick speech, salivation, and bronchial hypersecretion. It is important that the pediatric dentist does not suddenly stop clonazepam therapy as status epilepticus may be precipitated (Browne 1978; Weiner et al. 1988).

Primidone is a drug which must be introduced in small increments to avoid oversedation. It is felt that primidone has little to offer over phenobarbital alone. Primidone is metabolized to a large extent to phenobarbital; thus, the combination of barbiturates and primidone can lead to oversedation. Diazepam plus primidone may also cause oversedation (Weiner et al. 1988).

Ethosuximide is the drug of choice for absence seizures. A lupus-like syndrome can occur as a toxic side effect. (Bresnan and Konkol 1987; Weiner et al. 1988).

Acetazolamide is a useful adjunct medication for absence and myoclonic seizures, and occasionally in generalized tonic-clonic seizures. ACTH is a useful adjunct medication for infantile spasms.

A ketogenic diet has been advocated to control myoclonic, tonic, atonic, atypical absence, and generalized tonic-clonic seizures. This diet is designed to produce ketosis with a high fat intake, thus raising the convulsive threshold (Dodson et al. 1976).

In general, children who have not had a seizure for three to four years, who are of normal intelligence, and who have normal findings on EEG and neurologic examination have a 74% chance of being free of seizures with discontinuation of medication. Fifty per cent of relapses after discontinuation of medication are likely to occur within three months, and 80% within one year (Emerson et al. 1981).

It is important for the pediatric dentist to be aware of lidocaine-induced seizures. Seizures from oral viscous lidocaine prescribed for herpetic gingivostomatitis are documented (Hess and Walson 1988). As well, toxic doses of lidocaine given for dental procedures can induce seizure activity (Hellström-Westas et al. 1988).

Social Aspects

The majority of people with epilepsy are intelligent, and historically have included great political leaders, artists, musicians, and scientists. Patients with seizures encompass a variety of people; ranging from successful businesspeople, to wheelchair-bound people with mental retardation and quadriplegia. However, all share the common fear of an unpredictable seizure ending with public embarrassment and humiliation (Niedermeyer 1990).

Epilepsy is a common disorder, but patients have great difficulty being accepted in schools, communities, and social activities. Their problems of acceptance can be attributed to ignorance, false illusions of what seizures are, and superstitions. "Epilepsy" is derived from the Greek word epilambanein: to take, to seize. In other words, an evil spirit "takes over" or "seizes" the patient (Niedermeyer 1990).

In the past, people with epilepsy were institutional-

ized and labelled mentally retarded, but today, they cannot be hospitalized against their will merely because they have epilepsy. People with epilepsy are still discriminated against. Obtaining health, life, or automobile insurance continues to be a major problem, and if coverage is available at all, the premiums are too high for most people to afford. Driving restrictions for these patients vary from state to state but most states require medical verification of seizure control and usually require a seizure-free period ranging from six months to two years. Some people deny their condition and do not seek medical care (Epilepsy Foundation 1987).

At present, few laws protect patients with epilepsy against employment discrimination. The Rehabilitation Act of 1973 only requires an employer who is a federal agency, or contracts with the federal government for over $2500, or is a recipient of federal financial assistance not to discriminate against people with seizures. The accident rate for people with epilepsy in industry is as low as that of other employees, but few corporations or companies appreciate this data (Epilepsy Foundation 1987). Education of the public and participation in public activities by those with seizure disorders can change the misconceptions and attitudes toward the disorder.

Drs. Nelson and Ureles are associates in pediatric dentistry, Children's Hospital, and clinical instructors in pediatric dentistry, Harvard School of Dental Medicine, Boston, MA. Dr. Holmes is director, Neurophysiology Laboratory, Children's Hospital and associate professor of neurology, Harvard School of Medicine, Boston, MA. Reprint requests should be sent to Dr. Linda P. Nelson, Dental Division, Children's Hospital, 300 Longwood Ave., Boston, MA 02115.

Abramowicz M: Drugs for epilepsy. Med Let Drugs Ther 25:81–84, 1983.

Braham RL, Casamassimo PS, Nowak AJ, Posnick WR, Steinberg AD: The dental implications of epilepsy. US Department of Health, Education, and Welfare. Rockville, MD: DHEW Publication No. (HSA) 78–5217, 1977, pp 1–13.

Bresnan MJ, Konkol RJ: Seizure disorders, in Primary Pediatric Care. Hoekelman RA, Blatman S, Friedman SB, Nelson NM, Seidel HM eds. St. Louis: CV Mosby Co, 1987, pp 1467–77.

Browne TR: Clonazepam. N Engl J Med 299:812–16, 1978.

Browne TR: Valproic acid. N Engl J Med 302:661–65, 1980.

Dodson WE, Prensky AL, DeVivo DC, Goldring S, Dodge PR: Management of seizure disorders: selected aspects. Part I. J Pediatr 89:527–40, 1976.

Dreifuss FE: Pediatric Epileptology: Classification and Management of Seizures in the Child. Boston: John Wright, PSG, Inc, 1983, pp 129–44.

Dreifuss, FE: Seizure disorders. Classification of epileptic seizures and the epilepsies. Pediatr Clin North Am 36(2):265–79, 1989.

Emerson R, D'Souza BJ, Vining EP, Holden KR, Mellits ED, Freeman JM: Stopping medication in children with epilepsy. N Engl J Med 304:1125–29, 1981.

Epilepsy Foundation of America. The Legal Rights of Persons with Epilepsy: An Overview of Legal Issues, Federal Laws, and State Laws Affecting Persons with Epilepsy. 5th ed. Landover, MD, 1987, pp 1–89.

Farmer TW, Greenwood RS: Paroxysmal disorders, in Pediatric Neurology 3rd ed. Farmer TW ed. Philadelphia: Harper and Row, 1983, pp 205–63.

Farwell JR, Lee YJ, Hirtz DG, Sulzbacher SI, Ellenberg JH, Nelson KB: Phenobarbital for febrile seizures — effects on intelligence and on seizure recurrence. N Engl J Med 322:364–9, 1990.

Gastaut H: Clinical and electroencephalographical classification of epileptic seizures. Epilepsia 11:102–19, 1970.

Gastaut H, Broughton R, Roger J, Tassinari CA: Generalized convulsive seizures without local onset, in Magnus O, Lorentz de Haas Am eds: Handbook of Clinical Neurology, Vol. 15. The epilepsies. New York: American Elsevier Publishing, 1974, pp 107–29.

Gastaut H, Roger H, Soulayrol R et al: Childhood epilepsia emphalopathy with diffuse slow spike waves (otherwise known as "petit mal variant") or Lennox syndrome. Epilepsia 7:139–79, 1966.

Hauser WA: Evaluation of the newly identified seizure patient. New York: Columbia University, 1985.

Hellström-Westas L, Westgren U, Rosén I, Svenningsen NW: Lidocaine for treatment of severe seizures in newborn infants. I. Clinical effects and cerebral electrical activity monitoring. Acta Paediatr Scand 77:79–84, 1988.

Hess GP, Walson PD: Seizures secondary to oral viscous lidocaine. Ann Emerg Med 17: 725–27, 1988.

Holmes GL, McKeever M, Adamson M: Absence seizures in children: clinical and electroencephalographic features. Ann Neurol 21:268–73, 1987.

Lacy JR, Penry JK: Infantile spasms. New York: Raven Press, 1976, pp 1–124.

Markand ON: Slow spike-wave activity in EEG and associated clinical features: often called 'Lennox' or 'Lennox-Gastaut' syndrome. Neurology 27:746–57, 1977.

Masland RL: The classification of the epilepsies: a historical review. In Magnus O, Lorentz de Haas Am eds. Handbook of Clinical Neurology, Vol. 5, The Epilepsies. New York: American Elsevier Publishing, 1974, pp 1–24.

Myers GJ, Cassady G: Neonatal seizures. Pediatr in Rev 5:67–72, 1983.

Niedermeyer E: The Epilepsies: Diagnosis and Management. Baltimore, MD: Urban and Schwarzenberg, Inc, 1990, pp 1–7.

Nelson KB: Discussion, Alter M, Hauser WA eds. In The Epidemiology of Epilepsy: A Workshop NINDS monograph, No. 14. Washington, DC: U.S. Government Printing Office, 1972.

Nelson KB, Broman SH: Perinatal risk factors in children with serious motor and mental handicaps. Ann Neurol 2:371–77, 1977.

Nelson KB, Ellenberg JH: Prognosis in children with febrile seizures. Pediatrics 61:720–27, 1978.

O'Donohoe NV: Epilepsies of childhood. London: Butterworths, 1979.

Penry JK, Dean JC, Riela AR: Juvenile myoclonic epilepsy: Long-term response to therapy. Epilepsia 30:(Suppl 4) S19–27, 1975.

Reynolds EH: Drug treatment of epilepsy. Lancet 2:721–25, 1978.

Vernadakis A, Woodbury DM: Maturational factors in development of seizures. In Jasper HH, Ward AA, Pope A eds. Basic Mechanisms of Epilepsies. Boston: Little, Brown and Co, 1969, pp 535–41.

Volpe JJ: Neonatal seizures. Clin Perinatol 4:43–63, 1977.

Weiner HL, Urion DK, Levitt LP: Pediatric Neurology for the House Officer, 3rd ed. Baltimore, MD: Williams and Eilkens, 1988, pp 33–61.

West WJ: On a peculiar form of infantile convulsions. Lancet 1:724–25, 1841.