'Not everything that shakes is epilepsy'

The differential diagnosis of paroxysmal nonepileptiform disorders

A. DAVID ROTHNER, MD

HUGHLINGS JACKSON defined a seizure as "an occasional, sudden, excessive, rapid and local discharge of gray matter." This discharge originates in the central nervous system (CNS), is paroxysmal in nature, and results in varying clinical syndromes ranging from simple staring to automatisms with altered consciousness, to tonic-clonic jerking of all extremities with unconsciousness.[1] The present discussion focuses on disorders that are often difficult to differentiate from "true" epileptic seizures, since they are also paroxysmal in nature, and may result in altered consciousness or abnormal movements. These disorders collectively occur more frequently than epileptic seizures and are usually not responsive to antiepileptic drugs (AEDs). Identifying the specific nature of these paroxysmal nonepileptiform disorders (PNEDs) is important if we are to avoid unnecessary laboratory tests, inappropriate use of AEDs with their attendant risks and side effects, and the adverse psychosocial and economic stigma of epilepsy.

TABLE 1
PAROXYSMAL NONEPILEPTIFORM DISORDERS

Cardiovascular
Breath-holding spells
Syncope
Mitral valve prolapse syndrome
Cardiac dysrhythmias
Nocturnal/sleep-related events
Pavor nocturnus
Narcolepsy/cataplexy
Somnambulism/somniloquy
Migrainous disorders
Common migraine
Classical migraine
Complicated migraine
Ophthalmoplegic migraine
Hemiplegic migraine
Migraine variants
Paroxysmal vertigo
Confusional migraine
Basilar artery migraine
Paroxysmal torticollis
Movement disorders
Paroxysmal choreoathetosis
Tourette's syndrome/tics
Shudder attacks
Startle disease
Spasmus nutans
Psychological disorders
Pseudoseizures
Episodic rage
Munchausen's syndrome by proxy
Daydreaming/attention-deficit disorder
Hyperventilation
Gastrointestinal disorders
Gastroesophageal reflux
Recurrent abdominal pain
Cyclic vomiting

CLASSIFICATION

The international classification of the epilepsies is commonly used as a means of defining the various types of seizures.[2] No similar classification of PNEDs exists. For purposes of discussion, they are organized here into six major areas (See *Table 1*).

The classification emphasizes commonly occurring disorders and is not all-inclusive. With the newer diagnostic methods available, patients presenting with paroxysmal and recurrent spells can usually be classified and treated. Other spells may disappear or be better

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clarified with the passage of time. A child who presents with recurrent paroxysmal spells that are not easily classified should be considered for intensive video electroencephalographic (EEG) or polysomnographic monitoring. Resolution of the spells after utilization of AEDs does not confirm the diagnosis of epilepsy since many PNEDs cease spontaneously.

EVALUATION

A properly obtained history is the key to a correct diagnosis.[3] Questions should be directed to both child and parents. A young child can provide surprisingly useful information; in adolescents this may best be obtained in a private interview. During the interview the affect of the child and parents and their interactions may yield useful clues concerning depression, anxiety, and conflict.

Information concerning abnormalities of pregnancy, labor, and delivery as well as delays in development will aid in determining the presence of a static encephalopathy, in which case the occurrence of epileptic seizures would be more probable. Data concerning school function, behavior, and the psychosocial state of the family are helpful in pinpointing psychological disorders. Other medical problems and/or medications are important since the spell may be related to an underlying medical disorder or an atypical reaction to a medication. The family history may reveal a wide variety of disorders, including syncope, breath-holding spells, mitral valve prolapse, sleep disorders, migrainous conditions, and movement disorders as well as epilepsy.

Eliciting details of the illness under consideration may "paint a picture" that will be useful. Answers to the questions listed below provide a data base upon which to formulate a differential diagnosis. When possible the parents should be encouraged to videotape actual episodes for review.

PAROXYSMAL NONEPILEPTIFORM DISORDERS: USEFUL QUESTIONS

1. Is there more than one type of spell?
2. When and at what age did the spells begin?
3. Did they follow an injury, unusual life event, or illness?
4. Have they been continuously or irregularly present since their onset?
5. How often do they occur?
6. Are they becoming progressively more frequent and/or more severe?
7. Does there seem to be a warning that precedes the episode?
8. Does anything routinely precipitate them?
9. Are they related to any of the following: a) fright; b) injury; c) standing up; d) prolonged standing; e) sleep; f) stress-anxiety; g) illness; h) fever; i) specific foods; j) school; k) eating; l) fasting; m) anger and frustration; n) initiating movement; o) boredom; p) medication; q) exercise; r) travel; s) anything else?
10. Describe the spell from its beginning to its completion, including any variations.
11. Does the child cease other activity during the spell?
12. Are associated symptoms present, i.e., headache, nausea, vomiting, diaphoresis?
13. How long does it last?
14. Does anything make it go away?
15. Does (has) anyone else in the family have (had) similar spells?
16. Does anyone in the family have epilepsy?

If symptoms of increased intracranial pressure are present or if previously acquired neurologic functions have been lost (i.e., regression), the diagnostic evaluation must be accelerated.

Each organ system must be examined carefully, since it may give a clue to the underlying etiology of the spells. Specific attention should be paid to the blood pressure, supine and upright positions, cafe-au-lait spots, and cardiac murmurs. In the majority of patients with PNED the physical examination is normal, and the neurologic examination is also likely to be normal; any departure from normal requires further investigation.

The choice of laboratory tests is based upon the differential diagnosis. Routine blood studies between attacks usually are not helpful. Cardiac electrophysiologic evaluation, including electrocardiography (ECG), exercise ECG, Holter monitoring, and echocardiography are useful when the episodes are potentially cardiac in origin.[4] When epilepsy is suspected, the EEG is useful, but it must be remembered that nonspecific EEG abnormalities are frequent in normal children. Persistent focal or generalized slowing and frank epileptiform abnormalities may be significant, but again it is worth noting that benign focal epileptiform discharges are found in 2% of normal children and in 10% of children with migraine.[5] Prolonged EEG recordings, awake and during sleep, increase the likelihood of finding abnormalities. Video-EEG with recording of the spell is useful for determining the

epileptiform nature of a spell. If a sleep laboratory is available, it may be useful in evaluating nocturnal spells as well as narcolepsy. Both computed tomography and magnetic resonance imaging are safe, rapid, accurate methods of evaluating the intracranial contents. Since they are useful in a wide variety of conditions, they are definitely indicated when a structural lesion is suspected. Psychological tests may aid diagnosis of patients suspected of having learning problems and/or functional disorders.

CEREBROVASCULAR CONDITIONS

Breath-holding spells occur in 4% to 5% of children.[6] A positive family history for similar spells occurs in 25% of individuals. Two thirds of such children have cyanotic breath-holding spells, 20% have pallid breath-holding spells, and the remainder a mixture of the two. Spells occur rarely or several times daily, peaking in frequency between the first and second year of life. Precipitating factors include minor injuries, fright, anger, and frustration. Such spells cease by age 6. Twenty percent of such patients later have syncopal attacks.

In cyanotic spells, breath holding occurs after prolonged crying; the child becomes limp and consciousness is lost. If prolonged, the spells may be followed by convulsive jerks. Electrophysiologic monitoring during the spells shows that they are not primarily epileptiform in nature and are associated with tachycardia. The EEG is normal interictally. AEDs do not prevent the attacks.

In pallid breath-holding spells, patients lose consciousness following the stimulus more quickly, with a minimum of crying. They are pale, not cyanotic. Convulsive jerks are frequent as the attack ends. The interictal EEG is normal, and the ECG shows bradycardia or asystole during a spell. Youngsters with this disorder develop bradycardia or asystole when ocular compression is performed. Since ocular compression may cause bradycardia and/or asystole, this technique should not be utilized unless resuscitative facilities are at hand. The prognosis is excellent, attacks ceasing by age 6. No treatment is necessary, and AEDs are ineffective. Neither of these conditions predisposes to epilepsy. Parents must be reassured regarding the benign nature of this disorder and should not allow the spells to affect routine discipline. If the history is typical and the examination is normal, a diagnostic evaluation is not indicated.

Simple *syncope* or fainting is a common cause of loss of consciousness (LOC) in a school-age child.[7] The LOC is secondary to decreased cerebral perfusion. Common precipitants include rising from the recumbent position, prolonged standing, pain, and fright. Patients frequently experience light-headedness, pallor, and "graying" of their vision prior to LOC. The LOC is brief and in some cases is followed by convulsive movements; some children are dizzy and fatigued after the spell. No evaluation is indicated in the usual case of infrequent syncope; however, if syncope is prolonged, recurrent, severe, or associated with bradycardia, a diagnostic evaluation is indicated.[4,7] The spectrum of diseases resulting in syncope ranges from benign to life-threatening conditions. The evaluation consists of a history, physical examination, ECG, and EEG. Tilt table testing may be useful. If a cardiovascular cause is suspected, a complete cardiac evaluation is indicated. Under most circumstances, however, a simple syncope requires no treatment. The EEG is normal during an attack, and AEDs are of no value.

Mitral valve prolapse (Barlow's syndrome) is a rather common disorder occurring in 6% of the population, more frequently in women.[8] A nonejection click and late systolic murmur are present. It is difficult to associate a specific clinical syndrome with this finding. The commonest symptoms are poorly defined nonexertional chest pain, dizziness, syncope, palpitations, and fatigue. Complications have included endocarditis, transient cerebral ischemic attacks, arrhythmias, and sudden death. It is common in children, but care should be taken before associating it in a definitive sense with spells. Patients require no treatment save periodic follow-up and antibiotic prophylaxis. Some clinicians discourage participation in strenuous competitive athletics. Counseling and reassurance are essential. The syncope that may occur with this condition is not epileptiform in nature.

A variety of *cardiac dysrhythmias* cause paroxysmal dysfunction simulating neurologic disorders and seizures.[9] These dysrhythmias are being recognized with increased frequency because of increased awareness and newer diagnostic techniques. In addition to history, physical examination, and standard ECGs, most major medical centers can perform ambulatory recordings, telemetry, exercise testing, echocardiography, and intracardiac electrophysiologic studies.

Patients with bradycardia and/or decreased exercise tolerance may have a cardiac arrhythmia. Patients may also present with tachycardia and syncope. If the syncopal episodes are prolonged or the patient is

difficult to arouse, a cardiac cause should be suspected. In addition, if the patient is markedly bradycardiac on presentation, heart block should be considered, and evaluation by a pediatric cardiologist is recommended. These dysrhythmias are amenable to treatment utilizing medications and pacemakers.

NOCTURNAL SLEEP-RELATED SPELLS

Pavor nocturnus, or night terrors,[10] is a common disorder, occurring most frequently in children between ages 2 and 5. The spells occur during nonREM sleep, within two hours of falling asleep, and are characterized by fear and unresponsiveness. Children seem to awaken, open their eyes, sit up, and run or walk about, but they cannot be comforted. Sweating and tachycardia are present. The episodes last one to two minutes, and may reoccur at a later time that same night. The frequency is variable, from nightly to rarely. In most cases, the event is not recalled in the morning.

These spells must be differentiated from benign focal epilepsy of childhood (BFEC). During the night terror and interictally, the EEG is normal. AEDs are of no benefit, but when spells are severe and occurring nightly, tricyclic antidepressants and/or benzodiazepines may be useful. If the clinical presentation is characteristic, sleep studies are not indicated. Anxiety appears to increase the number of attacks, and counseling may be indicated. Parents will be reassured to hear that spontaneous resolution occurs within months in most patients.

Narcolepsy is characterized by episodes of an irresistible urge to sleep, cataplexy, hypnogogic hallucinations, and sleep paralysis.[11] Excessive daytime sleepiness and cataplexy are most common. Fifty percent of narcoleptics begin having their symptoms during adolescence. The etiology is unknown, but there is a strong association with HLA-DR2. In some cases there is a positive family history. Patients exhibit excessive daytime sleepiness, with frequent naps lasting from one to ten minutes following which they feel fully refreshed. The patient may fall asleep while reading, driving, or walking. These episodes frequently are misinterpreted as absence or atonic seizures. The correct diagnosis is based upon history and confirmed by polysomnography. The multiple sleep latency test is frequently diagnostic, the onset of sleep being rapid and associated with the appearance of rapid eye movement (REM) sleep. Cataplexy is characterized by sudden loss of muscle tone and weakness, frequently following anger or fright. These spells last only seconds and are also misinterpreted as atonic seizures. Treatment of both conditions is difficult, and the clinical course is variable. The sleep attacks may respond to stimulant medications and the cataleptic attacks to tricyclics.

Two other nocturnal disturbances which may be interpreted as being epileptiform in nature include *somnambulism* and *somniloquy*.[12] Youngsters sit up in bed with a glassy facial expression, and may walk about clumsily and/or talk. Such episodes, lasting from one to ten minutes, are followed by resumption of normal sleep. There is no recollection of the event. This disorder, night terrors, and nocturnal enuresis are classified as parasomnias or nonREM dyssomnias. They occur during nonREM sleep, stages III and IV. These seem to be maturational disorders and resolve spontaneously over time. If the event is typical, an evaluation is not indicated. If the differential diagnosis is in doubt, polysomnography is usually diagnostic. Treatment is not indicated unless the spells are frequent and recurrent and present a clear and present danger to the child. Benzodiazepines and/or tricyclic antidepressants can be used for a limited time.

It is important for the clinician to recognize the relationship between sleep and epilepsy.[13] Some patients with epilepsy have seizures that are nocturnal only, such as BFEC.[14] Polysomnography and sleep EEG recordings may be necessary to confirm the diagnosis. Epileptiform discharges appear more frequently during sleep, and sleep deprivation is well known to lower the seizure threshold.

MIGRAINE SYNDROMES

Migraine, a familial disorder, is common in childhood and adolescence.[15] In Bille's study, it was noted that 1.4% of children had true migraine by the age of 7, and by age 15, the percentage increased to 5.3%.[16] The episodic nature of this disorder, its occasional association with altered consciousness, atypical movements, and other neurologic symptoms, as well as its association with EEG abnormalities, have caused it to be confused with epilepsy. The criteria for its diagnosis include recurrent headaches separated by symptom-free intervals, plus any three of the following six symptoms: 1) abdominal pain, nausea, or vomiting; 2) hemicrania; 3) a throbbing pulsatile quality to the pain; 4) complete relief after a period of rest; 5) an aura, either visual, sensory, or motor, and 6) a history of migraine headaches in one or more members of the family.[17]

The pathophysiology of the migraine attack is not completely understood. Currently it is felt that migraine is of CNS origin, resulting in secondary vasoconstriction followed by vasodilation. Changes in cerebral blood flow occur before, during, and after the migrainous attack. Accurate diagnosis depends upon the history and lack of physical findings and radiologic abnormalities.

Common migraine is the most common form of migraine in children and adolescents. The aura is nonvisual and consists of pallor, irritability, and fatigue. A generalized frontal headache is followed by nausea, vomiting, photophobia, phonophobia, and sleep. One or two attacks occur monthly.

Classic migraine occurs less frequently than common migraine. The aura is visual, and attacks are preceded by blurred vision, flashing lights, or distorted images. The headache is unilateral, and is followed by nausea, vomiting, phonophobia, and photophobia.

Epileptiform discharges can be seen in up to 10% of children with migraine.[18] This EEG abnormality, combined with the paroxysmal nature of the disorder and the associated neurologic symptoms, causes it to be confused with epilepsy. The relationship between migraine and epilepsy has recently been reviewed.[19] Treatment consists of analgesics, sedatives, abortive medication such as ergots, and preventive treatment with propranolol. There are no well-documented studies regarding the efficacy of AEDs, although many practitioners continue to utilize them.

Complex migraine, less common than the above forms, is the association of transient neurologic deficits with migraine attacks. These deficits are felt to be secondary to ischemia. Most attacks resolve completely.

Ophthalmoplegic migraine produces orbital pain and a complete or incomplete third nerve palsy. Headache precedes the ophthalmoplegia, the pain being severe and uniocular. LOC is not common. Associated dysfunctions include ptosis, strabismus, diplopia, and mydriasis. The neurologic deficit may last several days to weeks after the headache has disappeared.

Hemiplegic migraine is recurrent hemiparesis with headache in a known migraine patient. The hemiparesis may precede, accompany, or follow the attack. Underlying causes for the hemiparesis must be ruled out. The episodic nature of the disorder, the associated neurologic symptoms, and the abnormal EEG findings cause confusion with epilepsy. Although some clinicians have advocated the use of anticonvulsants to prevent these attacks, supportive data concerning the efficacy of prophylactic treatment for these disorders are sparse.

Migraine variants are most frequently misdiagnosed as epilepsy because they are paroxysmal, associated with altered consciousness, neurologic abnormality, and EEG changes.

Acute confusional migraine consists of an altered sensorium, agitation, and headache. It must be distinguished from drug intoxication and nonconvulsive status epilepticus. The confusional state lasts several hours and is associated with a communicative disorder. The diagnosis of migraine is made retrospectively. In most cases the EEG during the attack shows slowing over the dominant hemisphere. The patient recovers within six to 12 hours.

In *basilar migraine*, recurrent attacks of neurologic dysfunction are referable to the brain stem and cerebellum. Vasoconstriction in the distribution of the basilar artery is the suspected cause. Episodes occur suddenly and usually clear completely. Symptoms are variable and include occipital headache, vertigo, nausea, vomiting, tinnitus, facial pain, ataxia, alternating hemiparesis, and LOC. This latter feature causes confusion with epilepsy. The disorder may be associated with occipital epileptiform discharges.

Paroxysmal vertigo occurs in younger children between ages 2 and 7. The episodes are sudden and brief and are associated with nystagmus. Consciousness is retained, but the youngsters are fearful and ataxic. *Paroxysmal torticollis* is a rare disorder in younger children consisting of episodic headache and torticollis. Included in the differential diagnosis is gastroesophageal reflux.

MOVEMENT DISORDERS

Paroxysmal choreoathetosis is a rare involuntary movement disorder, usually beginning in children between the ages of 5 and 16.[20] Attacks occur spontaneously or are induced by movement, startle, or anxiety. The movements may be tonic, dystonic, or choreoathetotic; the course is nonprogressive. A typical attack includes posturing of the elbow, wrist, head, and leg, sometimes accompanied by the inability to speak. The attack lasts less than a minute and may occur three or four times per week. EEGs are normal. Response to AEDs is excellent. The most common misdiagnoses include hysteria and focal epilepsy. The pathophysiology of the disorder is unknown.

Tics are common in childhood, occurring in 20% of

children. They consist of sudden, brief, involuntary rapid repetitious movements or utterances which are purposeless and stereotyped. Motor tics usually involve the face, neck, and shoulders. They are less frequent during sleep and are exacerbated by stress. The spectrum ranges from simple tics to multiple motor tics and to *Tourette's syndrome (TS)*. This familial disorder is a clinical diagnosis with vocal and motor tics in association with behavioral and learning problems.[21] Beginning under the age of 10 with mild brief manifestations, the motor tics of TS come and go until they become more intense and more frequent; then vocal tics become apparent as do behavioral and learning problems. TS may be confused with myoclonic seizures. The EEG is within normal limits. Current treatment includes special education, psychological support, and medication such as haloperidol.

Shuddering attacks are less common and have been confused with epilepsy.[22] These attacks of shivering with flexion of the knees, elbows, and body are seen in young infants and children and are thought to be secondary to monosodium glutamate or to be the precursor of essential tremor. The attacks become less frequent and remit during the latter part of the first decade. The EEG is normal. Recognition of the syndrome is important to avoid unnecessary investigation and treatment.

Startle attacks or hyperexplexia are attacks of sudden jumping movements secondary to unexpected stimuli.[23] These exaggerated startle responses can be confused with myoclonic seizures. The EEG is normal. The disorder does not respond to anticonvulsant medication.

Spasmus nutans is a benign disorder of infants usually beginning between the fourth and twelfth month of life.[24] The three cardinal features include head nodding, nystagmus, and torticollis. Head nodding is the first symptom and precedes the nystagmus. The movements of the head are not synchronous with the nystagmus, which may be monocular. The etiology is unknown, and no treatment is necessary once other more serious neurologic disorders have been excluded. The EEG is within normal limits, and the disorder does not respond to AEDs.

PSYCHOLOGICAL DISORDERS

Minor as well as severe psychological disorders are common in childhood; they are frequently associated with unusual mannerisms and atypical behavior. If they occur in a paroxysmal fashion, and coexist with nonspecific EEG abnormalities, the diagnosis of "seizure equivalent" is frequently and erroneously made.

Pseudoseizures are a not uncommon disorder in adolescents and may be quite difficult to diagnose.[25] The primary disorder may be hysteria with a dissociative reaction or a conversion reaction. Ten percent may have associated epileptic seizures; and associated behavioral disorders commonly coexist. Females are more frequently affected. Details suggesting this diagnosis include occurrence at specific times or when specified individuals are present, gradual onset, lack of injury during the seizure, lack of fecal or urinary incontinence, lack of postconvulsive stupor, and presence of bizarre or atypical convulsive movements. Their duration is variable, and their pattern changes; stressful situations exacerbate their frequency. The neurologic examination and the interictal EEG are normal, but personality testing (e.g., MMPI) may demonstrate significant abnormalities. When possible, a seizure should be induced or recorded spontaneously during video-EEG monitoring. During these pseudoseizures the EEG remains normal, confirming the diagnosis. If anticonvulsant medication has been prescribed, it should be discontinued, and psychological counseling provided. The overall outlook for these patients is variable, since many are resistant to therapy.

Episodic rage may be caused by a neurologic disease or a psychological disorder.[26] The rage is paroxysmal and results in altered behavior for which the patient may be amnestic. Epilepsy is then considered in the differential diagnosis. With little or no provocation except frustration, these patients may suddenly shift from a state of calm to wild rage with a serious impulse to violence and destruction. Subduing such patients may be difficult, since they appear to be out of contact with reality and do not respond to reasoning. Directed rage is rarely a manifestation of seizure activity. In most situations episodic rage represents a psychological deviation.

Daydreaming is a disorder involving a lapse of vigilance and inability to maintain attention. It probably represents boredom or the attention deficit disorder (ADD).[27] All children daydream at times. If episodes occur frequently and are noted by parents or teachers, they may be misdiagnosed as absence or petit mal seizures. In ADD, daydreaming coexists with physical hyperactivity and learning disability (LD). If a diagnosis of absence is suspected, an EEG with hyperventilation is necessary, and three minutes of hyperventilation will produce a characteristic absence seizure. In patients who are daydreaming, the EEG is normal. If

daydreaming is part of the ADD-LD syndrome, stimulant medication may be effective in selected cases. AEDs are of no value.

The *hyperventilation syndrome* is characterized by a constellation of symptoms occurring mainly at rest, including dizziness, light-headedness, vertigo, focal or generalized paresthesias, visual disturbances, headache, dyspnea, and LOC not accompanied by convulsive movements.[28] Neurologic symptoms during the attack may be asymmetrical. Although onset may be in the first decade, the disorder appears frequently in adolescence. The majority of patients are girls. The symptomatology is aggravated by the psychological state of the patient, who may have other psychological symptoms between attacks. The paroxysmal nature of the symptoms in association with neurologic dysfunction often results in an erroneous diagnosis of epilepsy; symptoms may be present for months to years before the correct diagnosis is reached. The neurologic examination is normal, as is the EEG. Other diagnostic information of value may be obtained from a psychological profile (MMPI) and reproduction of the symptoms during hyperventilation. Treatment involves rebreathing from a paper bag, reassurance, and counseling.

Munchausen's syndrome by proxy is a term used to describe a condition in which the parents of children consistently give fraudulent clinical histories and fabricate signs causing needless harmful medical investigations of the child, hospital admissions, and treatment over periods of time ranging from months to years.[29] "Symptoms" include bizarre neurologic symptoms, hematuria, bacteriuria, recurrent diarrhea, bloody stools, drowsiness, urticaria, fever of unknown origin, episodic bleeding, lethargy, etc. The psychodynamics of this situation are complex. In some instances, children have died under circumstances implying parental involvement. Investigation of unusual medical conditions must take into consideration this unique form of child abuse. Unnecessary procedures and treatment must be avoided. Separation from the parents is often necessary, as are mobilization and coordination of support services, including psychiatric, social, and legal services. The parents must be confronted with any well-founded suspicions. Family and individual therapy may be useful.

GASTROINTESTINAL DISORDERS

Gastroesophageal reflux or Sandifer's syndrome is a disorder that affects both normal and neurologically impaired children.[30] It can produce vomiting, failure to thrive, anemia, esophagitis, pulmonary aspiration, asthma, and episodic posturing simulating epilepsy. Some patients have significant contortions of the neck and assume abnormal postures leading to the misdiagnosis of dystonia as well as epilepsy. The etiology of this disorder is unknown, but impairment of the neurologic control of the gastroesophageal sphincter is suspected. Diagnosis is made utilizing such procedures as an upper GI series, mean resting lower esophageal sphincter pressure measurements, esophageal intraluminal pH measurement, endoscopy, and gastroesophageal scintiscan. Treatment depends on severity of symptoms and includes upright positioning, bland diet, antacids, and medications to decrease gastric secretions. In patients with neurologic disorders, surgical treatment may be necessary.

Recurrent abdominal pain usually begins at about 5 years of age and ends within 3 to 4 years.[31] The frequency is weekly in about half the cases but may be daily. The pain lasts several hours and does not show a seasonal preference. It most frequently occurs in the morning before school. After a negative GI workup, an EEG is frequently requested to evaluate "abdominal epilepsy." If the EEG shows abnormalities, it may erroneously be considered an epilepsy variant. These patients should be questioned concerning headache and a family history of migraine. Many of them will later be shown to have migraine, although some have milk allergy and others psychological causes as the basis for their abdominal pain. In the absence of alteration of consciousness or epileptiform seizures, a diagnosis of epilepsy should not be made.

Cyclic vomiting usually begins at 2 to 3 years of age and disappears within 3 to 4 years.[32] Paroxysmal episodes of severe nausea, vomiting, and abdominal pain, occurring most frequently in the morning, may continue on a daily to weekly basis, resulting in acidosis, dehydration, and eventual hospitalization. A complete diagnostic evaluation is usually negative. Many of these patients develop migraine in later years and others are found to have psychological disturbances. If there is no associated loss of consciousness, the diagnosis of epilepsy is unlikely.

SUMMARY

Many infants, children, and adolescents exhibit unusual mannerisms, behaviors, and spells. These events cause concern to parents and are frequently

brought to the attention of physicians. If the spells are infrequent and do not interfere with function, no intervention may be necessary. If they are severe and recurrent, however, diagnostic evaluation is necessary. This article has reviewed the most common spells that can be misdiagnosed as epilepsy by pediatricians. Other spells such as head banging, head rolling, body rocking, enuresis, nightmares, bruxism, obsessions, compulsions, self-injurious behavior, self-stimulating behavior, and stereotypies are reviewed elsewhere.[33] Emphasis is placed on the importance of a thorough history, a complete general physical and neurologic examination, and the judicious use of laboratory testing. The diagnostic methodologies currently available, including cardiac evaluation, video-EEG monitoring, and polysomnography, will frequently result in a specific diagnosis, and allow appropriate treatment. In many cases, however, even after the above diagnostic tests have been utilized, no diagnosis is forthcoming, but reassurance and follow-up can be of help to the young patient and the family.

A. DAVID ROTHNER, MD
Section of Pediatric Neurology
The Cleveland Clinic Foundation
One Clinic Center
9500 Euclid Avenue
Cleveland, Ohio 44195

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