90 Nonepileptic Paroxysmal Movement Disorders

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Short Description

A frequent challenge in clinical practice is to distinguish between epileptic and nonepileptic paroxysmal events. In addition, further differentiation between physiologic nonepileptic paroxysmal events and those that are psychogenic is important to provide proper treatment. Paroxysmal movement disorders are nonepileptic but may overlap with epileptic seizures both in relation to diagnosis and treatment. The former occur with much greater frequency in children than in adults. The difficulty in differentiating "spells" has been highlighted in several studies which have examined the final diagnoses among groups of children. Hindley et al. (2006) prospectively studied 380 children referred to a dedicated secondary care clinic over an 8-year period with paroxysmal events. Although 23% of the children were diagnosed with epilepsy, syncope was the most common cause (42%) identified. Bye et al. (2000) retrospectively studied children who had video EEG monitoring at a single institution over a 10 year period. Forty percent of the children who had been monitored had epileptic events while 43% had nonepileptic events emphasizing the importance of those episodes that imitate epilepsy.

The situation is further complicated by the co-existence of both epileptic and nonepileptic movement disorders in conditions such as autosomal dominant infantile convulsions and paroxysmal (dystonic) choreoathetosis (ICCA) (Guerrini 2001), Glut-1 transporter deficiency syndrome (Suls et al. 2008), and in a number of rarer syndromes. Factors leading to a misdiagnosis may include overlapping clinical features, inadequate witnessed history, the limitations of investigations and insufficient expertise in relation to the clinical features of epileptic and nonepileptic paroxysmal disorders (Ferrie 2006; Chowdhury et al. 2008).

Classification and Basic Characteristics

Since the misdiagnosis of epilepsy can lead to serious consequences including driving and employment restrictions with inappropriate antiepileptic drug treatment, correct diagnosis is important to ensure the proper approach to treatment of the missed diagnosis. *Table 90-1* lists the most common nonepileptic paroxysmal movement disorders in childhood.

Neonates and Infants

Jitteriness Neonates and young infants often present with rapid generalized tremulousness ("jitteriness"). This occurs in normal children but is more frequent in those with perinatal ischemic encephalopathy or in preterm babies with hypoglycemia or hypocalcaemia. Children whose mothers had taken sedatives during pregnancy are especially vulnerable. The jitteriness may be spontaneous or may be stimulus-triggered and can be terminated by passive flexion of the affected limb(s). Jitteriness usually resolves by 4–6 weeks post-conceptual age (Engel and Pedley 2008), though may persist for 6–12 months (Shuper et al. 1991; Kramer et al. 1994).

Startle Startle represents an exaggerated, physiological arousal response. The startle response may become pathological in situations such as in spastic cerebral palsy, where it may occur following minimal provocation, or even spontaneously. *Hyperekplexia* is a disorder characterized by exaggerated startle responses and hypertonicity that may present in the neonatal period in severe cases (Engel and Pedley 2008). While it usually occurs sporadically (Engel and Pedley 2008), an autosomal dominant form also exists, due to a mutation in the alpha 1 subunit of the glycine receptor (Sotero de Menezes 2002).

Benign Neonatal Myoclonus Benign neonatal myoclonus is described as repetitive myoclonus seen mostly during non-REM sleep (Sotero de Menezes 2002). The age of onset ranges from one to 15 days. The distribution is usually generalized, although the arms are involved more often than the legs (Fusco and Specchio 2005). Clusters of jerks usually last for a few seconds but may last up to 60 min (Daoust-Roy and Seshia 1992). If the infant awakens or the sleep phase is changed, the myoclonus stops (Fusco and Specchio 2005). Benign neonatal myoclonus usually disappears before the 6 month, although persistence to 2 years of age has been reported (McVicar and Adam 2006). By definition, the neurological examination and EEG are normal (Fejerman 2005).

C.P. Panayiotopoulos (ed.), *Atlas of Epilepsies*, DOI 10.1007/978-1-84882-128-6_90,

Table 90-1. Paroxysmal nonepileptic events

A. Neonates and infants
• Jitteriness
• Startle
• Benign neonatal myoclonus
• Shuddering attacks
• Benign myoclonus of early infancy
• Benign paroxysmal torticollis
• Sandifer syndrome resulting from gastroesophageal reflux
• Self gratification behavior
B. Older children
• Breath holding attacks
• Syncope
• Psychogenic pseudoepileptic seizures
• Tics
• Paroxysmal dykinesias
• Sleep related disorders
• Hypnic jerks
• Rhythmic movement disorders
• Disorders of arousal
• Confusional arousals
• Sleep terrors
• Sleep walking

Table 90-2. Classification of syncope in childhood

1. Neurally mediated syncope
• Reflex syncope
• Postural orthostatic tachycardia syndrome
2. Cardiovascular causes
• Arrhythmic
• Structural
• Vascular
3. Pseudosyncopes
• Epileptic
• Psychogenic nonepileptic

Shuddering Shuddering attacks are benign nonepileptic events that typically begin in infancy. The events are brief, usually lasting no more than a few seconds, however the frequency can be up to more than a hundred events per day (Tibussek et al. 2008). The clinical event consists of rapid shivering of the head, shoulders and occasionally the trunk. The ictal EEG is normal and typically no neurological abnormalities are found. Spontaneous remission can be expected (Tibussek et al. 2008).

Benign Myoclonus of Early Infancy Benign myoclonus of early infancy usually begins in the first year of life. The attacks manifest with a series of brief tonic or myoclonic contractions involving the axial muscles and more prominently the neck. It has a self limited course with variable duration but usually disappears before 2 years of age (Pachatz et al. 1999). Attacks mostly occur during wakefulness and are frequently triggered by excitement or frustration. The EEG is normal and the long-term prognosis is excellent (Sotero de Menezes 2002).

Benign Paroxysmal Torticollis The symptoms appear during the first year of life, most often between the ages of 2 and 8 months. Twisting of the neck occurs during attacks (*Fig. 90-1*), and may be associated with vomiting, discomfort and tilting of the head to one side. The duration of the attacks is usually several hours but may be as short as 10 min or as long as 14 days (Sotero de Menezes 2002). In most cases, attacks cease by 2–3 years of age. A relationship to migraine has been suggested (Sotero de Menezes 2002).

Sandifer Syndrome Severe gastroesophageal reflux may be associated with Sandifer syndrome. Infants with Sandifer syndrome manifest abnormal movements and behaviors including irritability, crying, head and eye version, torticollis, extensor spasm and dystonic posturing (Kabukas and Kurt 2006). These are postulated to result from attempts to protect the airways from reflux or to relieve the abdominal pain caused by esophagitis (Gorrotxategi et al. 1995; Kabukas and Kurt 2006). The symptoms usually resolve with treatment of the gastroesophageal reflux, however in cases where diagnosis is late and which have accompanying complicated anatomical defects response and prognosis may be worse (Senocak et al. 1993).

Self Gratification Behavior In children, self stimulatory behavior, or masturbation, is commonly recognized to be a variant of normal behavior (Nechay et al. 2004; Yang et al. 2005). It typically begins at approximately 2 months of age and peaks at 4 years of age and again in adolescence (Yang et al. 2005). It may be difficult to recognize in infants and young children because it often does not involve manual stimulation of the genitalia (Fleisher and Morrison 1990). Instead, manifestations may include rubbing the thighs together (*Fig. 90-2*) or rocking the body or the pelvis against a hard surface.

Older Children

Breath Holding Attacks Breath holding spells are common between 6 months and 6 years of age, peaking between two and 3 years of age (McVicar and Adam 2006; Engel and Pedley 2008). Two forms have been recognized: the cyanotic and the pallid spells. Both forms may result from reflex vagal

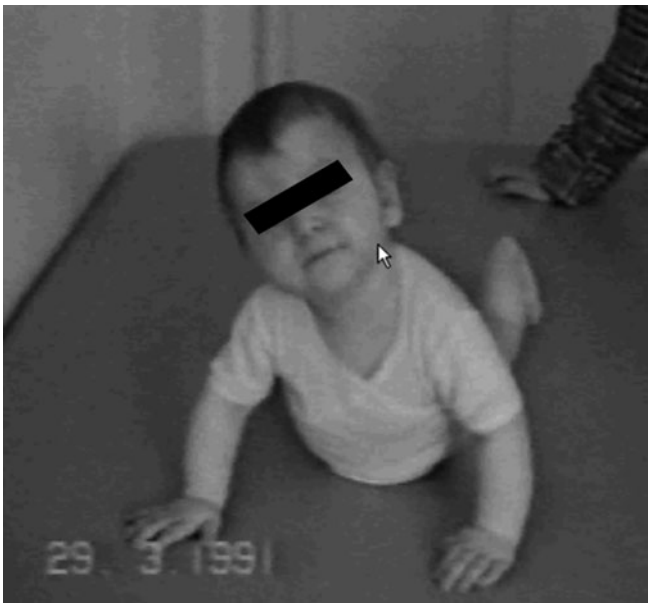

Figure 90-1. Head tilt during an attack of benign paroxysmal torticollis

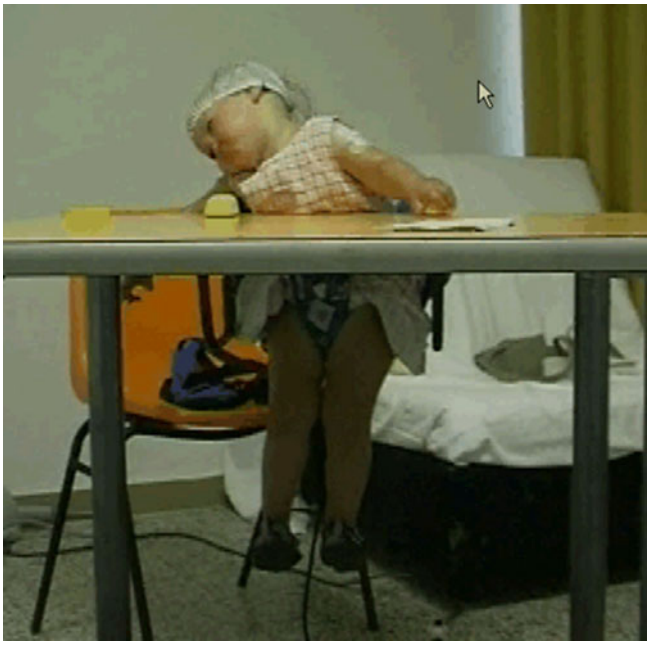

Figure 90-2. "Dystonic" posturing and tightly adducted thighs during an episode of self stimulatory behavior

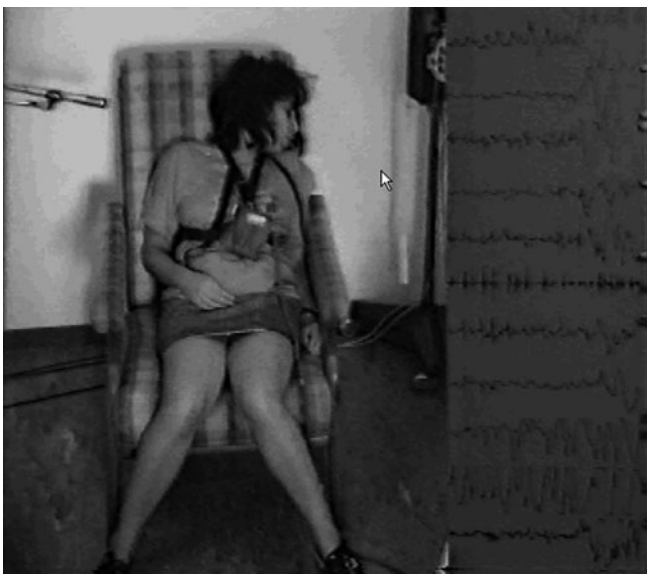

Figure 90-3. Psychogenic nonepileptic seizures (PNES) with EEG demonstrating movement artifact only during clonic movements

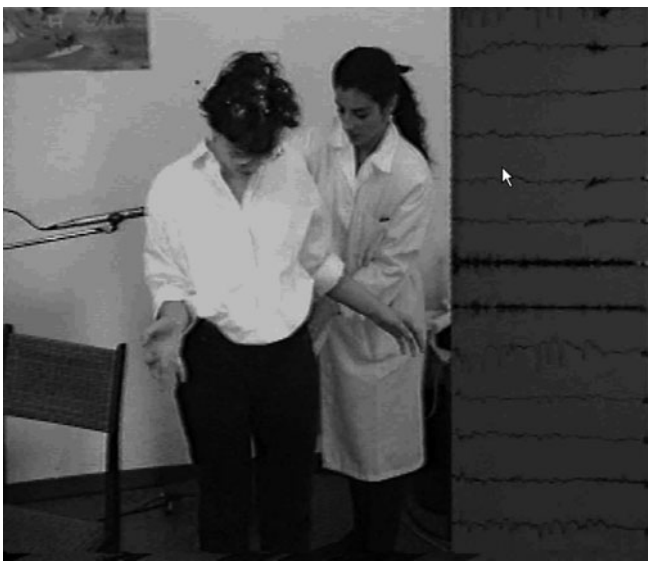

Figure 90-4. Dystonic posturing of the right-hand in a patient with kinesigenic dyskinesia, provoked by the movement of rising from the chair

changes that produce bradycardia and decreased blood flow (Engel and Pedley 2008). In the more common cyanotic form, the child generally cries in response to anger, frustration or minor trauma. Breathing ceases suddenly, often during expiration. Cyanosis appears immediately and is followed by loss of consciousness and limpness. Consciousness rapidly returns after one to 2 min with resumption of normal activities. Although the pallid form may also follow trauma or surprise, crying may be minimal or absent. Profound bradycardia or even asystole may occur during events (Engel and Pedley 2008). The attacks may be longer and, in fact, represent reflex anoxic seizures (Fejerman 2005). Brief clonic jerks of eyes or extremities may occur although in rare cases generalized or prolonged epileptic seizures may follow the tonic episodes (Battaglia et al. 1989).

Treatment of the breath holding spells most often comprises behavioral modification of the parents' responses to the episodes (McVicar and Adam 2006; Engel and Pedley 2008). Cyanotic breath holding spells may be associated with iron deficiency and may respond to iron supplementation (Mackay 2005; Engel and Pedley 2008). In the case of pallid attacks, treatment with atropine-like agents or even cardiac pacing may rarely be necessary (McVicar and Adam 2006).

Syncope Syncope is an abrupt, transient and self limited loss of consciousness associated with loss of postural tone (Crompton and Berkovic 2009) and is the antithesis of a movement disorder. In childhood, syncope can be broadly divided into three groups (*Table 90-2*).

Neurally mediated syncopes are a heterogeneous group of autonomic disorders which result in orthostatic intolerance.

Reflex syncopes are transient disturbances in autonomic control of heart rate and blood pressure. Neurocardiogenic syncope is the most common form (McLeod 2003). It occurs when activation of the autonomic nervous system causes reflex slowing of the heart with sinus bradycardia and blood pressure charges (McVicar and Adam 2006). Common triggers include micturition, coughing, swallowing and vasovagal phenomena. With a prolonged duration and subsequent cerebral hypoxia, the child may have a reflex anoxic seizure (Crompton and Berkovic 2009).

The most common cause of *cardiogenic syncope* is a cardiac electrophysiological conduction defect (McVicar and Adam 2006). Cardiac dysrhythmias such has prolonged QT syndrome may predispose to malignant ventricular arrhythmias and cardiac arrest (Crompton and Berkovic 2009). Exercise is a known precipitant of cardiogenic syncope (McLeod 2003; Fejerman 2005).

The *pseudosyncopes* can be broadly divided into the epilepsies and the psychogenic pseudoepileptic seizures. The latter will be discussed in more detail, below.

Psychogenic Nonepileptic Seizures Psychogenic nonepileptic seizures (PNES) can be defined as episodes of altered movement, sensation or experience that mimic epilepsy but are related to a psychological process (Crompton and Berkovic 2009). They most commonly occur in adolescents (Fejerman 2005; Mackay 2005) and may be a common cause of pseudo-resistant epilepsy. Estimates of the prevalence of comorbid epilepsy and PNES have a wide range reported (Gene-Cos and Ring 2005) depending upon the rigor of the study.

A number of clinical features have been suggested as being useful in the differentiation between epileptic and PNES including the lack of stereotypic patterns, avoidance of painful stimuli, the uncommon occurrence of urination and limited post-ictal confusion though these features are not absolute (Fejerman 2005). Although a number of predisposing/ precipitating factors for the development of PNES have been proposed, the patient population is heterogeneous and ubiquitous (Gene-Cos and Ring 2005; Reuber et al. 2007).

Psychogenic nonepileptic seizures are unassociated with paroxysmal epileptiform discharges in the brain (Crompton and Berkovic 2009) (*Fig. 90-3*). Video-EEG monitoring is required to confirm the diagnosis (Sotero de Menezes 2002; Mackay 2005; Crompton and Berkovic 2009).

Tics Tics are intermittent, repeated stereotyped movements or sounds. Simple tics occur in up to 20% of children and most commonly involve the muscles of the face and neck (McVicar and Adam 2006). Children with complex tics may have multiple types of tics, or tics involving several muscle groups. Tics may be exacerbated by stress or increased mental concentration. They tend to subside within weeks to months but may recur.

Paroxysmal Dyskinesias The "paroxysmal dyskinesias" are uncommon disorders characterized by episodic hyperkinetic movements that occur without impairment of consciousness (Zorzi et al. 2003; Crompton and Berkovic 2009). There is increasing evidence of the role of ion-channelopathies in many of these disorders (Crompton and Berkovic 2009). Various clinical subtypes have been delineated including kinesigenic, nonkinesigenic and exertion-induced dyskinesias. Correct identification is important because of the treatment implications.

Paroxysmal kinesigenic dyskinesia is the most frequent form of paroxysmal dyskinesia (Zorzi et al. 2003). Attacks are triggered by sudden voluntary movement (*Fig. 90-4*), but are brief, lasting less than 1 min (Crompton and Berkovic 2009). Although attacks may occur multiple times per day, they often respond dramatically to treatment with low-dose anticonvulsant medication, such as carbamazepine (Crompton and Berkovic 2009). Paroxysmal nonkinesigenic dyskinesia is less common and attacks are longer, although less frequent (Crompton and Berkovic 2009). The attacks can be provoked by a number of triggers including caffeine, alcohol, sleep deprivation and stress (Crompton and

Berkovic 2009). Paroxysmal exercise-induced dyskinesia is characterized by attacks which tend to be provoked by prolonged exertion, fasting or stress, and can range from less than 1 min to 2 h (Crompton and Berkovic 2009). This form is sometimes associated with epilepsy (Guerrini 2001; Guerrini et al. 2002), while benign familial infantile seizures may occur in association with paroxysmal kinesigenic dyskinesia (Crompton and Berkovic 2009).

Chorea refers to "irregular changing movements that flow between body areas" (Wolf and Singer 2008). Sydenham's chorea is the neurological sequel of Rheumatic fever and represents the prototypical autoimmune disorder. The precipitating Group A streptococcal infection occurs 1 week to 6 months before the onset of neurological disease (Dale 2005). In recent years a number of other movement disorders have also been attributed to antibodies produced as the result of an immune response to Group A streptococcal surface proteins. These nonepileptic paroxysmal movement disorders include tics, chorea, parkinsonism (Dale 2005), paroxysmal dyskinesias (Senbil et al. 2008), and restless legs syndrome (Matsuo et al. 2004). The term "PANDAS" was coined by Swedo et al. to describe 50 patients with recurrent, acute fulminant exacerbations of tics and obsessive-compulsive symptoms that had a temporal association with Group A streptococcal infections (Swedo et al. 1998). The existence of this disorder remains controversial with continuing arguments both for (Snider and Swedo 2004; Dale 2005; Mell et al. 2005; Feitosa de Oliveira 2007) and against (Wolf and Singer 2008).

Sleep-Related Disorders

Hypnic Jerks Hypnic jerks (or "sleep starts") represent a physiological motor phenomenon (Mantagna 2004; Fusco and Specchio 2005). They consist of brief, bilateral body jerks during the transition from wakefulness to sleep. They are often accompanied by the subjective impression of falling. Hypnic jerks do not usually require any treatment, however, when particularly severe, can cause a sleep-onset insomnia (Mantagna 2004; Fusco and Specchio 2005).

Rhythmic Movement Disorders Rhythmic movement disorders occur at sleep onset and during brief arousals or transitions to lighter stages of sleep. They may comprise head banging or rolling and/or body rocking. Rhythmic movement disorders usually begin in the first year of life and end by 4 years of age in normal children. In autistic or mentally retarded individuals, they may persist during wakefulness and into adulthood (Fusco and Specchio 2005).

Disorders of Arousal The disorders of arousal all arise from non-REM sleep. They may be due to incomplete maturation of the system governing the sleep-wake cycle and arousal (Derry et al. 2006).

Box 90-1

(a) *Confusional arousals*

Confusional arousals are characterized by sudden arousal with disorientation and confusion, sometimes associated with semi-purposeful behavior. Vocalization, and sometimes coherent speech, is common. The events may last for 10 min or more, during which the child is difficult or slow to awaken (Derry et al. 2006).

(b) *Sleep terrors*

Sleep terrors affect between 1 and 6% of prepubertal children with a peak incidence between five and 7 years of age (Tinuper et al. 2007). The child suddenly arouses from sleep with variable degrees of motor activity including screaming, incoherent vocalizations, inconsolable crying and extreme emotional and autonomic activation. The episodes generally last 1–5 min but may last up to 20 min.

(c) *Sleep walking*

During sleep walking affected children may display simple or complex motor behaviors such as moving objects, talking, dressing, eating or drinking (Derry et al. 2006). The episodes may last from a few minutes to 30 min and usually end with the child returning to bed and to sleep. Some sleepwalkers will respond to a command to return to bed (Derry et al. 2006).

Adult Movement Disorders

In adults, movement disorders that imitate seizures have different etiologies, often not encountered in the younger population. Tics, torticollis, paroxysmal dyskinesias and myoclonus have been discussed and may abate or persist into the adult years. Some motor imitators of epilepsy not uncommonly pose a question of epilepsy in adults (Fisher and Blum 1994) (see list below).

Box 90-2

- Ballism
- Complex tic disorders
- Dystonia
  - Head and neck (blepharospasm, Meige's syndrome, and torticollis)
  - Body (nocturnal paroxysmal dystonia)
- Paroxysmal (kinesiogenic) choreathetosis
- Psychogenic
- Myoclonus and clonus
- Spasms (hemifacial spasm)
- Tremor

Ballism is a violent movement with characteristics similar to chorea. Wild, rapid, involuntary, flinging of an extremity that is usually an arm is usually unilateral (hemiballism) and is often associated with subcortical infarction of the basal ganglia that progressively wanes over weeks to months. Drugs and metabolic abnormalities are infrequent but may

occur. Unlike focal seizures, ballism and chorea are proximal motor movements and consciousness is always intact.

Tics as previously mentioned are most prevalent in childhood. When they are complex they may be associated with other clonic or dystonic movements that mimic epilepsy such as upward eye deviation or lateralized movements of the head and neck, that can become confused with epileptic seizures. Despite their complexity, their chronicity usually reveals their nonepileptic origin in adults, unless co-morbid epilepsy exists. The causes are diverse and treatment is with dopaminergic drugs such as haloperidol or pimozide.

Dystonia is a slow and usually sustained abnormal posture that may be focal or segmental and may be confused with epilepsy. Causes may be symptomatic or cryptogenic and they may be genetically determined. *Spasmodic torticollis* is a focal dystonia with slow regular repetitive neck movements that may be associated with a jerk or spasm imitating partial seizures with a more favorable prognosis in children. *Paroxysmal nocturnal dystonia* is often difficult to differentiate from frontal lobe epilepsy with nocturnal occurrence, a tendency to cluster, brief ("hypermotor") patterned attacks, bizarre posturing, normal EEG, and response to AEDs such as CBZ noted. Video-EEG monitoring may help to clarify and stratify those patients with movement disorders and those with epileptic seizures though final impressions are ultimately based upon the clinical patterns to distinguish movements from seizures. *Blepharospasm* with or without oromandibular dystonia (*Meige's syndrome*) may manifest unilaterally or commonly bilaterally with forced eye closure and is an infrequent mimic of epileptic seizures though commonly occurs in adulthood.

Muscle spasms or cramps typically occur due to peripheral nervous system involvement and are usually seen in normal individuals. Stiff-person syndrome and amyotropic lateral sclerosis are often patient fears that are readily dispensed with neurological evaluations aided by laboratory tests and electromyography. *Hemifacial spasm* has movements that may involve the face and neck and persist into sleep like seizures. Eye blinking may give rise to unilateral facial dystonia that mimics a focal seizure. Consciousness is intact and a structural cause compressing the fifth or seventh cranial nerves should be sought. Treatment is medical with baclofen, AEDs, or botulinum toxin or with neurovascular decompression if an aneurysm or foreign tissue lesion is encountered in the cerebellopontine angle of the brainstem.

Psychogenic movement disorders are being recognized with increasing prevalence and are most common in adults. It has been suggested that up to 50% of patients seen in specialty clinics or seeking confirmatory medical advice are psychogenic though epidemiological studies lack confirmatory diagnostic testing. Certain features are common to many different types of psychogenic movement disorders including abrupt onset (often triggered by a minor injury), rapid progression to maximum disability, or peak severity shortly after onset, a static course, previous remissions, and paroxysmal exacerbations (Bye et al. 2000; Lang 2006). Psychogenic movements are incongruous with their organic counterparts and demonstrate inconsistencies historically, during examination, or over time. Nonphysiological features on exam include give-way weakness, and nonanatomic sensory dysfunction. Myoclonus and tremor may be amenable to neurophysiologic documentation though clinical aspects are most useful. Clues that a movement disorder may be psychogenic are listed in the table (adapted from Bye et al. 2000).

Box 90-3

Historical
1. Abrupt onset, static course, spontaneous remissions
2. Young female, multiple somatic complaints, psychiatric disturbance
3. Onset after minor trauma, litigation pending, secondary gain

Clinical
1. Inconsistent amplitude, frequency, distribution, selective disability
2. Paroxysmal, decrease with distraction, ability to trigger or stop movement through suggestion techniques (sounds/lights, tuning fork, "main nerve")
3. Deliberate slowness of movements, false weakness or sensory complaints
4. Functional disability out of proportion to exam findings
5. Movement abnormality that is bizarre, multiple, or difficult to classify

Treatment
1. Unresponsive to appropriate medication
2. Response to placebo or remission with psychotherapy

No single symptom exists for adults with movement disorders or with seizures. Most movements resolve with sleep yet nocturnal paroxysmal dystonia will be activated and some forms of myoclonus (palatal) and dystonia (hemifacial spasm) will continue. Tremor and clonus will usually resolve and cramps or spasms will be associated with pain allowing distinction between motor movements and seizures. The natural course will often provide information over time and video-EEG monitoring may be helpful to exclude the stereotypy and epileptiform discharges that serve to underlie a diagnosis of epilepsy.

Conclusion

Although many nonepileptic conditions share semiologic features with epilepsy, and epilepsy and nonepileptic movement disorders may sometimes co-exist, careful history taking often allows for the correct diagnosis to be made. In

those situations where the diagnosis remains unclear, review of a video of the event, or, in selected cases, video EEG monitoring, generally allows for definitive characterization.

Related Topics

► Mimics of Epileptic Seizures in Neonates and Young Infants

► Nonepileptic Paroxysmal Events Occurring During Sleep and Sleep Disorders

► Parasomnias in Early Childhood that Mimic Epileptic Seizures

► Psychogenic Nonepileptic Events Imitating Epileptic Seizures

► Syncopal Attacks

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