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"Everything that shakes is not seizure" is a slogan of neurologists that strikes home in emergency departments. When confronted with abnormal movements of the body, the emergency physician must consider a differential diagnosis that is broader than seizure disorder. Movement disorders (MDs) typically are a symptom of a larger problem and may well be associated with serious illness. The emergency physician must determine whether or not the MD is a harbinger of an undiagnosed, critical condition.

Parkinsonism is one of the most common MDs. As the emergency department visits of elderly patients increase, emergency physicians will see more patients with parkinsonism. Elderly patients more often seek emergency department care for complications of parkinsonism or its therapy (such as orthostasis, causing falls) than for the symptoms of parkinsonian tremors. Other commonly seen MD drug-induced dystonias are a diagnostic challenge if the patient cannot provide a good history. Hemiballism is a MD that is important to recognize as a rare complication of stroke.

Emergency department encounters with MDs are probably more common in the daily practice of emergency medicine than genuine "seizures." However, MDs often are not considered a major consequence unless they are associated with a complication. This issue is devoted to the often overlooked neurological condition (an MD) that can indeed be a symptom of a critical underlying neurological or a non-neurological disease process.

— The Editor

Everything That Shakes Is Not a Seizure: A Primer of Movement Disorders for Emergency Physicians

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Introduction

MDs are abnormal motor activities that are not associated with primary dysfunction of the corticospinal tracts, cerebellum, sensory pathways, or peripheral nervous system. This term often is used as a synonym for basal ganglia disorders. However, some disorders that are classified as MDs, such as myoclonus and some forms of tremor, are not associated with basal ganglia pathology. MDs can be associated with an acute, primary neurological disease such as a cerebrovascular event, or a focal neurological disease such as a neoplasm. Some MDs are a manifestation of underlying systemic illness such as hepatic or renal failure or autoimmune disease. MDs, dystonia in particular, often are misdiagnosed as being hysterical or psychiatric in origin. The primary task of the emergency physician in evaluating a patient with a suspected MD is to identify the features of MD and determine if a critical underlying neurological or non-neurological condition exists that may need urgent attention. A thor-

ough history and a focused neurological examination will yield clues that will help distinguish these conditions.

Pre-Hospital Care

MDs, especially acute dystonic reactions (ADRs), easily can be confused with focal or generalized seizure activity. Pre-hospital care providers are advised against pursuing aggressive measures when the diagnosis of ongoing seizure is uncertain. It is important to differentiate generalized tonic-clonic status epilepticus from a MD or other cause of involuntary movements. For conditions other than seizures, supportive measures usually suffice as long as the airway is not compromised and vital signs are stable. Information collected by pre-hospital care providers on conditions leading to falls or other acute events is very important in evaluating patients with suspected MDs. Drug ingestion, substance abuse, and exposure to environmental toxins such as carbon monoxide can be associated with several different types of MDs.

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Emergency Department Evaluation

Assessment of vital signs and the adequacy of the airway are the first priority in the emergency department. A seizure disorder is distinguished from a MD on presentation by obtaining a thorough history and by performing a focused physical and neurological examination. *(See Table 1.)* Information from bystanders or family members is crucial for making a diagnosis of a seizure disorder. If a patient is unable to communicate, old medical records and a description of events as witnessed by pre-hospital care providers and others are most useful.

MDs usually are distinguished from seizure disorders on the basis of clinical evaluation. The patient history contributes the most to a diagnosis, with little additional information provided by laboratory and radiographic studies. Family, social, and psychiatric histories are reviewed. Use of psychotropic medications, particularly the use of antiemetics, is questioned. A careful physical examination can reveal signs of metabolic or endocrine derangements or toxic exposures.

A careful neurological examination with accurate characterization of the abnormalities will allow distinction of various types of MD. The character of the involuntary movement(s) is first assessed by observation of the patient's head, trunk, and limbs. Eye movements, tone (resistance of muscles/joints to passive manipulation), gait (casual, toe, heel, and tandem), and fine coordination (rapid finger tapping or alternating pronation and supination of the hands) are tested. Detection of neurological abnormalities other than the MD is essential in neuroanatomical localization of pathology and assists in generating differential diagnoses. Incoordination does not necessarily indicate the presence of a MD because it can result from injury to the corticospinal tracts, cerebellum, sensory pathways, or basal ganglia.

A laboratory evaluation, guided by results of the history and physical examination, can include basic serum chemistries, drug levels, and toxicological studies. If illicit drug use is suspected, urine can be tested for these substances.

The role of neuroimaging studies in the evaluation of MDs is limited. Some MDs occur acutely from focal structural lesions such as can occur from stroke. Typically, they are present in a localized body area or follow a "hemi-distribution," as in hemidystonia or hemiballism. Urgent brain imaging can be helpful following the acute onset of symptoms with a focal distribution.

Classification of Movement Disorders

MDs can be classified into four broad categories based on phenomenological features, clinical pharmacology, and neuropathology: 1) hypokinetic disorders, which are identical with the syndrome of parkinsonism; 2) hyperkinetic/choreic movement disorders; 3) tremors; and 4) myoclonus. *(See Table 2.)*

Descriptive features of individual MDs are summarized in Table 3. In some cases, differentiating specific MDs can be difficult and at times unnecessary (e.g., distinguishing mild myoclonus from chorea). Chorea, athetosis, and ballism are appropriately viewed as part of a spectrum of involuntary movements with a common pathophysiology.

Table 1. Important Historical Questions for Evaluation of a Patient with Suspected MD

1. Manner and temporal nature of symptom onset
2. Location of symptoms; body parts most affected
3. Factors that alleviate or exacerbate the symptoms
4. Whether symptoms are present at rest, with sustained posture, with movement, or only during the execution of specific tasks
5. Exposure to toxins or environmental factors and medication use
6. History of premature birth, perinatal injury, or behavioral problems

Table 2. Classification of Movement Disorders

HYPOKINETIC MD/ PARKINSONISM	HYPERKINETIC/CHOREIC MOVEMENT DISORDERS
Parkinson's disease	Chorea
Drug-induced parkinsonism	Athetosis
Parkinsonian syndromes	Ballism (Hemiballism is more common)
	Dystonia
	Tics
TREMORS	**MYOCLONUS**
Resting tremors	Generalized
Postural tremors	Segmental
Kinetic tremors	Focal
Task-related tremors	

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Hypokinetic MD (Parkinsonism)

Parkinsonism, a prototypical example of hypokinetic MD, is a syndrome caused by deficient dopaminergic effects within the striatum (caudate and putamen). Any process interfering with striatal dopaminergic function can cause parkinsonism. *(See Table 4.)* This frequently is a result of idiopathic dysfunction of dopamine innervation within the striatum, but also can occur as a result of side effects of certain drugs (e.g., phenothiazines). Parkinson's disease affects more than 1 million individuals in the United States. The incidence increases with age, resulting in a high prevalence in the elderly.

Important historical features useful in establishing a diagnosis of parkinsonism are: difficulty with initiating or halting movement, especially getting in or out of chairs; and a history of micrographia, the tendency for letter size to become progressively smaller during handwriting.

Clinical findings in parkinsonism often are asymmetrical, with onset and preponderance of symptoms on one side of the body. Incoordination, notably with fine motor tasks, is common. A loss of facial expression (masked facies) or loss of voice amplitude (hypophonia) also are common. Examination reveals stooped posture, masked facies, saccadic pursuit eye movements, low-volume voice, reduced blinking rates, and generalized slowing of movement. Gait often is slow and shuffling, with loss of associated arm swing and the need to take several steps to turn. Muscle tone is increased, with plastic (increased resistance throughout range of motion independent of velocity) or cogwheel (ratchet-like) quality. Postural reflexes are impaired, which can lead to falls. A characteristic resting tremor often is present in the hands, legs, or chin. *(See Table 5.)*

Parkinson's disease usually has an insidious onset and is slowly progressive. Patients do not present to the emergency department for initial evaluation of parkinsonian tremors, but rather for problems that arise from some complications of Parkinson's disease and its treatment. *(See Table 6.)*

Drug therapy with dopamine replacement and/or dopamine agonists provides excellent symptomatic relief for several years. Many patients experience progression of disease that results in poor response to medication or difficult-to-manage side effects. Many patients develop marked fluctuations in response to therapy, with periods of complex involuntary movements (dyskinesias). These dyskinesias have features of both dystonia and chorea occurring in close temporal association with periods of severe bradykinesia and rigidity. These fluctuations are difficult to manage and often require judicious manipulation of medications and dosage schedules over a long period of time for optimal control of symptoms. Choreic dyskinesias tend to occur at times when the effect of dopamine replacement therapy is at its peak. Choreic dyskinesias can be improved by decreasing medication doses or lengthening the dosing interval.

Nausea is another common problem associated with the use of carbidopa/L-dopa or dopamine agonists. Taking medications at the end of a meal to slow their absorption can reduce nausea. For patients taking carbidopa/L-dopa, an adequate amount of carbidopa must be taken to block the peripheral effects of L-dopa and reduce nausea. For an average-size person, 75 mg of carbidopa usually is sufficient to reduce peripheral side effects. Other peripheral side effects include flushing and orthostatic hypotension. Orthostatic hypotension is especially troublesome, and some patients with Parkinson's disease can have autonomic insufficiency independent of drug treatment. Orthostatic hypotension can lead to syncope and falls, with their attendant consequences.

All medications used in the treatment of Parkinson's disease can cause altered mental status. Hallucinations are a relatively common side effect of carbidopa/L-dopa and dopamine agonists, and can occur with the use of anticholinergics and amantadine. These hallucinations usually are visual, typically non-threatening in character, and commonly occur in the absence of other features of delirium. However, typical delirium also can occur. Hallucinations, delirium, and other mental status changes occur most frequently in the many patients with Parkinson's disease that develop dementia. In Parkinson's patients with changes in mental status, subdural hematoma is an important diagnostic consideration due to the high incidence of falls in these patients.

Many patients with Parkinson's disease can manifest varied symptoms caused by pain, such as muscle spasms, cramps, and

Table 3. Phenomenology of Movement Disorders

MOVEMENT DISORDER	FEATURES	AREAS OF INVOLVEMENT	ANATOMIC LOCALIZATION
Parkinsonism	Bradykinesia, rigidity, often resting tremor, often postural instability, stooped posture, masked facies, hypophonia	Often asymmetric at onset, but can be generalized	Basal ganglia—Interruption of or interference with nigrostriatal dopaminergic neurotransmission
Dystonia	Sustained, spasmodic, repetitive contractions causing involuntary abnormal postures	Any voluntary muscle can be affected (usually head, neck, face, and limbs)	Presumed to be basal ganglia—Associated with putamen lesions in some cases.
Tremor	Involuntary, rhythmic and roughly sinusoidal movements: some are action-induced	Head, hands, limbs, and voice .	In parkinsonian resting tremor—Basal ganglia Most other tremors may involve cerebellar dysfunction
Chorea	Involuntary, irregular, rapid, jerky movements without a rhythmic pattern; dance-like	Generally limbs, but any body part can be affected	Basal ganglia—Striatum or subthalamic nucleus
Athetosis	Akin to chorea but with distinct "writhing" movements	Limbs, but any body part can be involved	Identical to chorea
Myoclonus	Brief, rapid, shock-like jerks	Generally involves very small muscles	Can result from dysfunction at any level of the central nervous system
Tics	Intermittent, brief, sudden, repetitive, stereotyped movements or sounds	Any body part can be affected; phonation/sounds	Presumed to be basal ganglia
Hemiballism	Uncontrollable, rapid, large amplitude flinging movements of a limb	Generally a limb	Basal ganglia—Subthalamic nucleus or striatum

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burning paresthesias.[1-4] These painful symptoms have several causes. Painful muscle spasms and uncomfortable paresthesias of uncertain etiology are common in Parkinson's disease. Many patients with complex dyskinesias have a painful dystonic component to their involuntary movements. Severe, localized limb pain, chest pain, or abdominal pain in the patient with Parkinson's disease poses a diagnostic challenge in the emergency department.

Discontinuation of dopamine replacement therapy can cause neuroleptic malignant syndrome, which is a medical emergency.

Hyperkinetic Movement Disorders

Examples of hyperkinetic MDs include dystonia, chorea, hemiballism, and tics. The distinguishing feature of hyperkinetic MDs is the overwhelming presence of involuntary movements into the normal flow of movements of specific groups of muscles. Some overlap of the different hyperkinetic MD disorders is common.

Dystonia. Any voluntary muscle group in the body can be affected by dystonia. Some muscle groups more commonly are involved than others are. Dystonia is characterized by sustained (tonic), spasmodic (rapid or clonic), or patterned or repetitive muscular contractions that frequently result in a wide range of involuntary twisting, repetitive movements, or abnormal postures. Abnormal postures, such as neck torsion, forced jaw opening, or inversion and dorsiflexion of the foot, are characteristic of dystonia.

Certain specific tasks or postures can elicit dystonia. For example, dystonia can be elicited by writing (writer's cramp) but not by other fine coordinated movements. "Spasmodic dysphonia" (a type of laryngeal dystonia), can cause difficulty with speaking but not with singing. Commonly, patients discover postures or maneuvers that reduce dystonia. The most common of these "sensory tricks" is gentle stimulation of one side of the face to reduce torticollis.

Dystonia is a primary neurological disorder or a prominent manifestation of a neurological disorder due to metabolic derangement as occurs in Wilson's disease, Lesch-Nyhan syndrome, and mitochondrial cytopathies. Dystonia also is known to result from structural injury to the central nervous system (CNS). *(See Table 7.)*

With liberal use of phenothiazines in the emergency department, drug-induced dystonia probably is more common than is generally recognized. Drug-induced dystonia also is the most commonly observed dystonia in the emergency department. Many patients treated with phenothiazines report feeling "jittery" and "uneasy." Overt manifestations of a dystonic reaction, such as bizarre movements and postures, may not always be present.

Table 4. Forms of Parkinsonism

IDIOPATHIC PARKINSONISM	Involves basal ganglia but no discernible degenerative conditions
DRUG-INDUCED PARKINSONISM	Neuroleptics, phenothiazine, haloperidol, tricyclic antidepressants, methyldopa, lithium, metoclopramide
NEURODEGENERATIVE DISORDERS	*(Clinically indistinguishable from other forms of parkinsonism.)* Involve basal ganglia. Discernible degenerative conditions

Table 5. Cardinal Features of Parkinsonism

BRADYKINESIA	Slowness of movement with a paucity of normal spontaneous movements such as arm swing when walking
RIGIDITY	Form of increased resistance to passive manipulation in which the increased tone has a "plastic" (constant resistance to passive manipulation) quality or "cogwheel" rigidity (in which resistance has a ratchet-like characteristic)
TREMOR	Typically a "resting tremor" of the hands/arms, legs, or chin that improves with use of the affected body part
IMPAIRMENT OF POSTURAL REFLEXES	Manifested by falls or near falls, and in difficulty in maintaining a stable stance when displaced gently backward on examination

Dystonia frequently can be misinterpreted as a psychiatric or hysterical condition because of several reasons, including:[5]

1) Bizarre movements and postures;
2) The finding of "action-induced dystonia" (the exacerbation of symptoms with stress and improvement with relaxation);
3) Diurnal fluctuations; and
4) Frequent effectiveness of various sensory tricks.

Selected Examples of Dystonia. Idiopathic torsion dystonia (dystonia musculorum deformans) is a familial (more common in Ashkenazi Jews) neurological disorder that has an autosomal dominant trait with variable penetrance.[6] Childhood-onset of primary dystonia is common. In the early stages, the abnormal movements are characterized by "action dystonia" and commonly start in one leg. With progression of the disease, dystonia often becomes generalized and is present at rest.

Focal dystonia refers to the involvement of a specific part of the body. A primary dystonia that begins in adulthood usually is focal (e.g., spasmodic torticollis). Torticollis can mimic a variety of orthopedic and neurological disorders that are important to recognize in the emergency department. *(See Table 8.)*

Blepharospasm, involuntary, periodic blinking of eyelids, is the second most common focal dystonia that is either isolated or associated with oromandibular dystonia, and is more common in women than in men. Approximately 15% of patients become functionally blind due to tonic closure of the eyelids. Blepharospasm can respond to sensory stimulation as occurs with talking, singing, and yawning.[7]

Oromandibular dystonia is characterized by forced mouth opening, occasionally with tongue protrusion, or involuntary jaw clenching that can result in mutilation of the lips and teeth. Blepharospasm-oromandibular dystonia syndrome commonly is referred to as Meige syndrome.

Spasmodic dysphonia is a form of laryngeal dystonia that causes spasm of the vocal cords. Patients generally are asymptomatic except for abnormalities of voice.

Writer's cramp is a focal "action dystonia," and is described as task-specific. As suggested by the name, dystonia of the hand and arm occurs only when attempting to write. A change in handwriting can be the presenting complaint. "Muscle ache" and dystonic spasms of the forearm musculature are common complaints in these patients.

Secondary dystonia is a term reserved for dystonia that results from identifiable metabolic disorders, CNS degenerative processes, or structural lesions of the CNS. There are no distinguishing clinical features of secondary dystonia. However, sudden onset, presence of dystonia at rest, rapid progression, or an unusual distribution such as hemidystonia in an adult suggests secondary dystonia. A thorough neurological examination usually reveals dysfunction of other parts of the CNS, including the cranial nerves, pyramidal system, cerebellar system, or the higher cortical functions. Hemidystonia suggests a focal lesion such as a mass, infarction, or hemorrhage of the basal ganglia. Secondary dystonia can have delayed onset of weeks to years following a cerebral injury such as stroke. The most frequent causes of delayed-onset dystonia are perinatal trauma or hypoxia.

Torticollis refers to dystonia-producing abnormal neck postures and it merits special attention in the emergency department because it has a more complicated differential diagnosis. Potentially life-threatening etiologies of torticollis, such as atlantoaxial subluxation or a posterior fossa tumor, must be considered before declaring dystonia as the cause of torticollis.[8] Causes of torticollis other than dystonia are listed in Table 8. Direct or indirect trauma to the neck suggests atlantoaxial subluxation. Gradually progressive extremity paresthesias and weakness suggest a herniated cervical disc. Visual disturbance and headaches can be caused by a posterior fossa tumor. Cervical adenopathy can cause torticollis in children. Associated neck dystonia with an impaired level of consciousness or other symptoms suggests the possibility of seizures. Dystonic torticollis can produce neurological complications such as cervical myelopathy or radiculopathy due to persistent abnormal neck postures.

Evaluation and Management of Dystonia. The goal of emergency department evaluation is to identify 1) "secondary dystonia," which may have a treatable cause; and 2) complications of

Table 6. Common ED Presentations of the Parkinsonian Patient

COMPLICATIONS OF PARKINSONISM

1. Falls due to impaired postural reflexes (consider subdural hematoma in a patient with mental status changes)
2. Orthostatic hypotension from autonomic instability resulting in syncope and falls
3. Painful muscle spasms
4. Paresthesias
5. Severe localized limb pain, chest pain, or abdominal pain[1-4]

COMPLICATIONS OR SIDE EFFECTS OF DRUG THERAPY FOR PARKINSONISM

1. Nausea: Common with carbidopa/L-dopa or dopamine agonists
2. Flushing and orthostasis resulting from the therapy
3. Mental status changes, particularly hallucinations, delirium, and dementia
4. Neuroleptic malignant syndrome can result from discontinuation of dopamine replacement therapy

conditions responsible for primary dystonia. It is important to distinguish dystonia from focal seizures. Recent-onset twisting and repetitive abnormal movements in an adult that respond to sensory stimuli or abnormal movements that can be suppressed voluntarily favor the diagnosis of dystonia.

Management of most dystonias is difficult, and symptomatic therapy generally is prescribed. High doses of anticholinergic medications frequently are successful in relieving some symptoms of dystonia. The higher doses of phenothiazines are better tolerated in children than in adults. Specific drug therapy is available for Parkinson's disease and Wilson's disease and should be prescribed in consultation with the neurologist. Patients with blepharospasm, oromandibular dystonia (especially jaw closing), spasmodic torticollis, spasmodic dysphonia, and cases of focal limb dystonia should be referred for botulinum toxin therapy.

Table 7. Etiologies of Selected Dystonias

DYSTONIA DUE TO DEGENERATIVE DISORDERS OF CNS	DYSTONIA DUE TO NON-DEGENERATIVE DISORDERS OF CNS
Parkinson's disease	Traumatic brain injury
Huntington's disease	History of perinatal anoxia
Progressive supranuclear palsy	Kernicterus
Other degenerative disorders of the basal ganglia and midbrain	Stroke (cerebral infarction)
Wilson's disease	Arteriovenous malformation
Storage diseases	Encephalitis
GTP cyclohydrolase deficiency	Toxins (e.g., manganese)
Lesch-Nyhan disease	Brain tumors
Mitochondrial disorders	Multiple sclerosis
Leigh's syndrome	Drugs
	Peripheral trauma

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Chorea. Involuntary irregular, rapid, jerky movements without a rhythmic pattern, that are randomly distributed with a flowing "dance-like" quality, characterize chorea, the Greek term for dance. Chorea generally involves multiple body parts. Athetosis (writhing movement) and ballism are part of the spectrum of chorea, and appear to share a common pathophysiology, usually involving the striatum or subthalamic nucleus.

Many neurological and non-neurological disorders are associated with the development of chorea.[9] *(See Table 9.)* The non-neurological conditions capable of causing chorea include certain immunological, infectious, metabolic, degenerative, and drug- and toxin-induced disorders.

The use of the medication L-dopa, a commonly prescribed therapy for parkinsonism, is associated with the development of chorea and probably is the most commonly encountered chorea in the emergency department.[8] Titration of L-dopa dosing can minimize the choreiform movements often seen in patients with parkinsonism.

Autoimmune causes of chorea include systemic lupus erythematosus (SLE) and primary antiphospholipid antibody syndromes.[10,11] However, only 2% of patients with SLE have chorea.[10] The cause of chorea in autoimmune disorders is not known but autoimmune-mediated injury to the basal ganglia has been postulated.[12] Chorea from autoimmune disorders can last from days to years and can be episodic and recurrent; this makes sufferers more likely to seek emergency care. Other neurological findings in SLE include migraine, stroke, seizures, cognitive impairment, peripheral neuropathy, and transient ischemic attacks. Antiphospholipid antibody syndrome is associated with recurrent vascular thrombosis, recurrent spontaneous abortions, and stroke. An antiphospholipid antibody titer is obtained in cases of chorea associated with these clinical situations. Imaging studies typically are normal.[12]

Structural lesions from cerebral infarctions involving the basal ganglia and thalamus can produce chorea. Stroke is likely the most common cause of hemichorea-hemiballismus.[13-15] Other causes include arteriovenous malformations, venous angiomas, metastatic tumors, or primary CNS neoplasms.

Thyroid dysfunction is a rare cause of chorea. Interestingly, both hyperthyroidism and hypothyroidism are known to be associated with chorea.[16] The pathophysiology is not well understood but is likely due to altered function of the basal ganglia, particularly the striatum.

Sydenham's chorea is a form of autoimmune chorea preceded by group A streptococcus infection, typically rheumatic fever. Unlike other manifestations of rheumatic fever, Sydenham's chorea occurs several months after the onset of acute streptococcal infection, usually affects patients between ages 5 and 15 years, and develops in girls more frequently than boys.[19] There appears

Table 8. Disorders Simulating Dystonic Torticollis (Cervical Dystonia)

NEUROLOGICAL DISORDERS

- Posterior fossa tumor
- Focal seizures
- Bobble-head syndrome (third ventricular cyst)
- Syringomyelia
- Congenital nystagmus
- Extraocular muscle palsies
- Arnold-Chiari malformation

MUSCULOSKELETAL/ STRUCTURAL

- Herniated cervical disc
- Rotational atlantoaxial subluxation
- Congenital muscular or ligamentous absence, laxity, or injury
- Bony spinal abnormalities: Degenerative; neoplastic; infectious
- Cervical soft-tissue lesions: Adenitis, pharyngitis
- Labyrinthine disease
- Abnormal posture in utero

Adapted from: Wiener W, Lang A. *Movement Disorders: A comprehensive Survey.* Mount Kisco, NY: Futura Publishing Co.; 1989.

Table 9. Differential Diagnosis of Chorea

HEREDITARY CHOREAS

- Huntington's disease (classic choreiform movement)
- Neuroacanthocytosis
- Wilson's disease
- Benign familial chorea
- Inborn errors of metabolism
- Porphyria
- Ataxia-telangiectasia
- Tuberous sclerosis

METABOLIC CHOREAS

- Hyper- and hypothyroidism
- Hyper- and hypoparathyroidism
- Hypocalcemia
- Hyper- and hyponatremia
- Hypomagnesemia
- Hepatic encephalopathy
- Renal encephalopathy

INFECTIOUS OR IMMUNOLOGICAL CHOREAS

- Sydenham' chorea (post rheumatic fever)
- Chorea gravidarum
- Systemic lupus erythematosus
- Polycythemia vera
- Multiple sclerosis
- Sarcoidosis
- Viral encephalitis
- Tuberous meningitis

CEREBROVASCULAR CHOREAS

- Basal ganglia infarction
- Arteriovenous malformation
- Venous angiomata
- Polycythemia

STRUCTURAL CHOREAS

- Posttraumatic
- Subdural and epidural hematoma
- Tumor (primary CNS or metastatic)

DRUGS/MEDICATIONS

- Phenytoin
- Phenothiazines
- Lithium
- Amphetamines
- Oral contraceptives
- Levodopa

TOXINS

- Mercury
- Carbon monoxide

INFECTIONS

- Neurosyphilis
- Lyme's disease
- Subacute sclerosing panencephalitis

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to be a familial prevalence, suggesting hereditary susceptibility. It tends to occur abruptly, worsens over 2-4 weeks, and usually resolves spontaneously in 3-6 weeks. It occurs more commonly in children who lack appropriate antibiotic care. Outbreaks of Sydenham's chorea have occurred in the United States and other developed countries. Measurement of antistreptolysin-O titers can help detect recent streptococcal infection. Since Sydenham's chorea can occur as late as six months after the streptococcal infection, measurements of antistreptolysin-O and antistreptokinase antibody concentrations obtained later may not be useful.[17]

Chorea gravidarum refers to choreiform movements associated with pregnancy. Approximately one-third of patients with chorea gravidarum have had Sydenham's chorea, suggesting that previous injury to the basal ganglia predisposes to chorea when estrogens and progesterone levels are elevated.[17,18] The use of oral contraceptives in women is associated with the development of chorea, especially in patients with a history of Sydenham's chorea. Chorea also is associated with the use of numerous other medications.

Huntington's disease (HD), commonly associated with choreiform movement, is an autosomal dominant neurodegenerative disorder. In addition to chorea, athetosis, dystonia, dementia, and psychiatric problems are common in patients with HD. Neurobehavioral disturbances, such as personality changes, agitation, apathy, depression, obsessive-compulsive disorders, social withdrawal, and sometimes, features of psychosis can precede choreiform movements. Symptoms and signs of HD begin at any age, but commonly present in the fourth and fifth decades. Life expectancy is approximately 15-20 years after diagnosis.[8]

Patients with HD seek emergency care for complications caused by their underlying disease process. Swallowing dysfunction can lead to poor nutrition and/or aspiration pneumonia, and sometimes asphyxia. Falls are common. Cerebral atrophy associated with HD places these patients at a higher risk for subdural hematomas. Severe dysarthria, dysphagia, dementia, and loss of ambulation occur in the final stages of the disease. Psychiatric disorders are associated with a high rate of suicide.[8]

Assessing the underlying cause of chorea is important. Medications that reduce dopaminergic neurotransmission can lessen the severity of chorea. The dopamine receptor antagonist haloperidol is the medication most frequently used to achieve this effect. Dopamine-depleting agents, such as reserpine or tetrabenazine, also can be effective. In many patients, impairments of coordination or mentation result from the doses of dopamine antagonists needed to reduce chorea significantly. Management of chorea in the emergency department is providing supportive care. Chorea does not require emergent treatment unless it interferes with function.

Hemiballism. Hemiballism, a hyperkinetic MD, is considered to be an extreme form of "hemichorea." Uncontrollable, rapid,

Table 10. Causes of Hemiballism

CAUSES	SUBTYPES
Cerebrovascular accidents	Ischemic, hemorrhagic Arteriovenous malformation Subarachnoid hemorrhage
Space occupying lesions	Metastatic cancer Subthalamic nucleus cyst
Infections	Tuberculous meningitis
Cerebral trauma	
Metabolic disorders	Non-ketotic hyperosmolar state
Multiple sclerosis	
Drugs	Phenytoin toxicity Oral contraceptives and estrogens Levodopa
Complications of stereotactic surgery	

Adapted from: Wiener WM J, Lang Anthony L, eds. Movement Disorders: A Comprehensive Survey. Mount Kisco, NY: Futura Publishing Co.; 1989.

Table 11. Etiological Classification of Tics

PRIMARY TIC DISORDERS

Tourette's syndrome
Various chronic tic disorders

SECONDARY TIC DISORDERS

Inherited: Huntington's disease
Neuroacanthocytosis
Torsion dystonia
Chromosomal abnormalities
Other

Acquired: *Drugs:* Neuroleptic, anticonvulsants, levodopa, stimulants
Trauma
Infections: Encephalitis, Creutzfeldt-Jakob disease, Sydenham's chorea
Developmental: Mental retardation, static encephalopathy, autism, pervasive developmental disorder
Stroke
Degenerative: Parkinsonism, progressive supranuclear palsy
Toxic: Carbon monoxide

Adapted from: Kurlan R, ed. *Treatment of Movement Disorders.* Philadelphia, PA: J.B.Lippincott Co.; 1995.

large-amplitude proximal flinging movements of a limb characterize hemiballism. Hemiballism refers to unilateral involvement, whereas rare bilateral involvement is called biballism. Typically, the face is not affected.[13] Hemiballism formerly was attributed solely to lesions of the subthalamic nucleus. It is now known that hemiballism can occur from lesions in other parts of the basal ganglia and the thalamus.

Stroke, generally a lacunar infarct in the subthalamic nucleus, is the most common cause of hemiballism. Hemiballism occurs most frequently in individuals older than 60 years of age who also have risk factors for stroke. Other causes of hemiballism are listed in Table 10. Common predisposing factors include hypertension, diabetes, thrombocytosis, or vasculitis.

Appropriate measures are taken to prevent injuries caused by violent hemiballistic movements. Disabling hemiballism requires immediate symptomatic relief even when the cause is not known. A neuroleptic medication such as haloperidol is most effective. Following a focused history and physical examination, ancillary tests should be directed toward diagnosing metabolic disorders, particularly a nonketotic hyperosmolar state. A history of medication use, including estrogens, oral contraceptives, phenytoin toxicity, and levodopa is sought. CT imaging may reveal evidence of a stroke.

Tics. Most common of all the MDs, tics are characterized by intermittent, sudden, repetitive, stereotyped movements (motor tics) or sounds (vocal tics). Tics can result from contraction of just one group of muscles, causing simple tics, which are brief, jerk-like movements or single, meaningless sounds. Complex tics result from a coordinated sequence of movements. Complex vocal tics can include linguistically meaningful utterances. Patients often admit that the tic occurs as an unavoidable but purposeful performance of the movement or sound. Tics can be suppressed temporarily and often wax and wane in type, frequency, and severity.

Tics can vary from a mild, transient disorder to a potentially devastating neurobehavioral disorder. Simple tics are extremely common and many people have some form of them. Tics rarely require emergent therapy. Several neurological and non-neurological disorders associated with tics are listed in the Table 11. Tics can be associated with stroke, head trauma, encephalitis, post-encephalitic syndrome of encephalitis lethargica, brain tumors, and carbon monoxide poisoning. They can occur as a result of long-term neuroleptic use (i.e., tardive tics).

Transient tic disorders (TTD) are tic disorders that are present in childhood for less than one year. They are extremely common among school-aged children, with an estimated prevalence of 5-24%.[19] An example of a TTD is a "chronic cough" that has not responded to medications.

Gilles de la Tourette's syndrome is the best known of all tic disorders. Tourette's syndrome (TS) is a disorder characterized by childhood onset of motor and vocal tics. Obsessive-compulsive disorder (OCD) and attention deficit hyperactivity disorder (ADHD) are strongly associated with TS. The established criteria for diagnosis of TS are onset before age 21 years, multiple motor tics, one or more vocal tics, and a fluctuating course and presence of tics for longer than one year. Males are affected more frequently than females, and there is a substantial genetic component.[20,21] Non-genetic factors such as maternal life stressors during pregnancy, gender of the child, and severe hyperemesis gravidarum are known to influence the form and severity of TS. The precise neuroanatomical location of a pathological lesion in TS is not known, although striatal abnormalities are hypothesized. The biochemical basis of TS is likely an increased activity of the

Table 12. Conditions that Can Enhance Physiologic Tremor

- **Mental state:** Anger, anxiety, stress, fatigue, excitement
- **Metabolic:** Fever, thyrotoxicosis, pheochromocytoma, hypoglycemia
- **Drugs and toxins**
- **Miscellaneous:** Caffeinated beverages, monosodium glutamate, nicotine

Adapted from: Weiner W, Lang A. In: Movement Disorders: A Comprehensive Survey. Mount Kisco, NY: Futura Publishing Company, Mount Kisco; 1989.

Table 13. Well-Known Causes of Tremor

PHYSIOLOGIC

PATHOLOGIC

Essential tremor
- Parkinson's disease
- Wilson's disease
- Midbrain tremor
- Peripheral neuropathy
- Multiple sclerosis
- Cerebellar infarction
- Cerebellar degenerative disorders

PSYCHOGENIC TREMORS

DRUGS AND TOXINS

Neuroleptics
- Lithium
- Adrenocorticosteroids
- Beta-adrenergic agonists
- Theophylline
- Ethanol
- Calcium channel blockers
- Valproic acid
- Thyroid hormone
- Caffeine
- Nicotine
- Tricyclic antidepressants

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dopaminergic system. TS frequently has a variable course, with waxing and waning of tics over several years. Tics tend to worsen in adolescence and abate in adulthood.

Haloperidol, a dopamine receptor antagonist, is most effective for control of tics. Haloperidol is used in doses ranging from 0.25 to 2.5 mg/day. Higher doses can be used in acute disorders. Clonidine, an alpha$_2$-adrenergic receptor agonist, can be useful in treating TS. New, atypical antipsychotics, such as risperidone, might have a role in the management of TS. Selective serotonin reuptake inhibitors, such as fluoxetine, are used widely to treat OCD, which frequently is associated with TS. Because of the possibilities of developing a tardive MD and other complications of neuroleptic use, these agents are reserved for disabling tics. Initial treatment with clonidine is preferred.

Tremors

Tremors are defined as involuntary, rhythmic, and roughly sinusoidal movements.[22] Tremors can be characterized as resting, postural, kinetic, or task-related. Resting tremor refers to tremor while a body part is relaxed without the influence of gravity. Postural tremor occurs during maintenance of steady body posture against gravity, which usually can be assessed by asking patients to extend their arms in front of them. Kinetic tremor occurs during goal-directed movements such as finger-to-nose testing. Task-related tremor occurs only during the performance of a specific task (e.g., a primary writing tremor). Intention tremor is an imprecise term generally used to describe wide oscillations that occur when a limb approaches a precise destination.

Selected Examples of Tremors. Physiological tremor is considered to be a normal phenomenon. Anxiety, fatigue, or stress exacerbates it. *(See Table 12.)* Hypoglycemia, hyperthyroidism, and pheochromocytoma all can enhance physiological tremors. Normal and enhanced physiological tremors are minimal at rest, present with posture, and worse with use of the affected limb. Many medications can cause tremor (see Table 13), likely by exacerbating physiological tremor.

Essential tremor (ET) is a distinct neurological syndrome characterized by postural and kinetic tremor of the hands; isolated head tremor; and voice tremor with no identifiable cause, such as drugs or toxins; or other focal neurological findings. ET can begin at any age; however, it is more common in the elderly. The tremor of parkinsonism usually is a resting tremor, and patients with ET do not have other features of parkinsonism. Emotional stress, anxiety, thyrotoxicosis, caffeine, and other stimulants exacerbate ET. The pathophysiology of ET is unknown but is likely due to alterations in cerebellar function.

Propranolol in a dose of 240-320 mg/day is a widely used treatment for ET. Primidone and benzodiazepines have been used for ET with variable success.

Task-related tremor occurs during specific motor tasks. The most common is primary writing tremor. Benzodiazepines can be useful in treating these unusual tremors.

Orthostatic tremor is a rare but frequently misdiagnosed condition. It occurs more frequently in women, and the onset is typically in the sixth decade. It manifests as tremor of the legs that is triggered by standing.[23] Orthostatic tremor should be distinguished from ataxia, which is unrelated to orthostasis.

Cerebellar tremor is a common consequence of injury to the cerebellum or its outflow pathways. This type of tremor can have resting, postural, and kinetic components. It is commonly described as affecting proximal muscles and invariably is associated with ataxia, dysmetria, and other signs of cerebellar dysfunction.

Psychogenic tremor is the typical hysterical MD. Marked fluctuation of the tremor is the hallmark of this tremor. Patients

Table 14. Features of Psychogenic Tremor

1. History of many undiagnosed conditions
2. History of multiple somatization
3. Absence of significant finding on physical examination or imaging study
4. Presence of secondary gain (pending compensation or litigation)
5. Spontaneous remissions and exacerbations
6. Employment in the health care delivery field
7. History of psychiatric illness

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demonstrate marked tremor that improves significantly when they are distracted. Other signs of functional illness are nonphysiological sensory deficits, tunnel vision, and bizarre gait disturbance. *(See Table 14.)*

Myoclonus

Myoclonus are brief, very rapid, sudden, and shock-like jerks that involve very small muscles or the entire body. Hiccup is a good example of "physiological myoclonus" that is called diaphragmatic myoclonus. Myoclonus is a descriptive term and not a diagnosis. Myoclonus does not indicate a specific neurological etiology.[24] These movements can be caused by active muscle contractions (positive myoclonus) or lapses in posture or muscle contractions (negative myoclonus or "asterixis"). Each jerk or sudden movement is a discrete, separate movement, in contrast to chorea, where dance-like, continual flow of movement occurs from one body part to another without interruption. Myoclonus differs from "tic syndromes" in that tics are stereotypic in quality and anatomical distribution, and generally can be suppressed with conscious effort by the patient.[25,26]

The four broad categories of myoclonus are 1) physiological; 2) essential or idiopathic; 3) epileptic; and 4) symptomatic.

Physiological Myoclonus. Physiological myoclonus occurs in normal people and includes sleep (hypnic) jerks, anxiety-induced myoclonus, exercise-induced myoclonus, and hiccup.

Essential Myoclonus. Essential myoclonus is a rare, possibly autosomal dominant hereditary disorder, which begins at a young age and generally has a benign course.

Epileptic Myoclonus. Epileptic myoclonus, as the term suggests, occurs in the setting of a chronic seizure disorder, and is a component of several different epileptic syndromes. Myoclonus can occur as a component of a seizure or as the sole manifestation of a seizure.

Symptomatic Myoclonus. Symptomatic myoclonus refers to myoclonic syndromes associated with an identifiable underlying neurological or non-neurological disorder. This is the most common cause of non-physiologic myoclonus. Associated neurological deficits include encephalopathy, dementia, ataxia, and pyramidal or extrapyramidal signs as dominant features of the illness. When recognized, clinical disorders responsible for this group of myoclonus may be treatable. Posthypoxic myoclonus resulting from global cerebral hypoxia from any cause is a well-known clinical entity.[27,28]

Symptomatic myoclonus resulting from metabolic derangements such as uremia, hepatic coma, hypercapnia, and hypoglycemia usually produces multifocal, arrhythmic myoclonic jerks predominantly affecting the face and proximal musculature. Changes in mental status are characteristic. The myoclonus caused by metabolic encephalopathy resolves as the encephalopathy is corrected. No specific therapeutic measure is required.

Asterixis, or negative myoclonus, was described originally in patients with hepatic encephalopathy, but also can occur in other metabolic or toxic disorders. Asterixis can occur in the recovery phase of general anesthesia, with sedative or anticonvulsant drug administration, and in normal drowsy individuals.[29-34]

Although rare, intractable myoclonus (as in viral encephalitis) can cause hyperthermia, hyperkalemia, hyperuricemia, systemic hypotension, and renal failure secondary to rhabdomyolysis.[35] Myoclonus can be a manifestation of serious underlying disease processes such as toxic or metabolic encephalopathies, or chronic epileptic disorders requiring urgent medical attention.

The focus of the examination in the emergency department is to determine possible correctable causes of the underlying illness causing myoclonus. Serum glucose levels, electrolytes, hepatic and renal function tests; drug and toxin screens; brain imaging; and urgent EEG can assist in diagnosing the most common metabolic and neurological derangements. Advanced studies such as evoked potentials, determination of enzyme activities (for storage disorders), DNA tests, tissue biopsy (for storage disorders and mitochondrial disease), or copper studies (for Wilson's disease) require referral to a neurologist.

Management of myoclonic movements in the emergency department is directed to specific management of the underlying illness in cases of symptomatic myoclonus. Valproic acid and clonazepam are effective for treating symptomatic myoclonus in many individuals.[36,37] Physiological myoclonus does not require specific treatment. Reassuring the patient is helpful. Standard antiepileptic drug (AED) therapy is used for myoclonus that is a component of an epileptic syndrome.

Movement Disorders Caused by Commonly Used Drugs

MD caused by the use of various medications is more common than generally is recognized. The cause-and-effect relationship between the drug and the MD is poorly understood, but preexisting CNS pathology likely predisposes to the development of MDs. Many MDs improve after the offending medication is discontinued. The following groups of commonly prescribed medications are known to cause MDs: antiepileptics; neuroleptics; CNS stimulants; oral contraceptives; calcium channel blockers; antihistaminics and anticholinergics; and antidepressants.

Table 15. Neuroleptic Medication-Induced MD

1. Acute dystonic reaction (ADR)
2. Akathisia
3. Drug-induced parkinsonism
4. Neuroleptic malignant disorder
5. Tardive disorders

Antiepileptics. Nystagmus, dysarthria, and ataxia commonly are associated with toxic levels of phenytoin and carbamazepine. Asterixis and spontaneous myoclonic jerks are common in the toxicity of phenytoin, phenobarbital, primidone, and carbamazepine. Chorea and dystonia are known to occur with the use of AEDs.[38,39] Chorea generally is associated with the chronic use of multiple antiepileptics. Initial use of an AED rarely results in chorea or dystonia. However, one exception is the development of chorea and dystonia with intravenous administration of phenytoin for status epilepticus.[40] This effect resolves gradually as the peak drug levels decrease.

Valproic acid is known to cause postural tremor (similar to benign essential tremor or enhanced physiological tremor) in approximately 20-25% of patients taking the medication.[41] Severity of tremor does not directly correlate with serum drug levels of valproate, but symptoms subside with decreasing drug levels.

Neuroleptics. The five major categories of MDs associated with the use of neuroleptic medications are listed in Table 15.

The time of onset of MD has some bearing on the type of MD seen with the use neuroleptic medications. ADR, akathisia, and parkinsonism generally occur early after treatment with neuroleptic medications is begun. Tardive disorders occur with prolonged use of neuroleptics. Neuroleptic malignant disorder (NMS) can occur at any time. The dopamine-blocking effects of neuroleptic medications likely are the pharmacological basis for the development of these MDs.

Acute Dystonic Reaction (ADR). Parenteral administration of phenothiazines is more likely to cause ADR than oral preparations, and the risk of ADR increases with the size of the dose. The risk of causing ADR after administration of phenothiazine is approximately 2-5%. ADR usually occurs at the initiation of therapy; 95% of ADR episodes occur within 96 hours of receiving the offending medication.[42] ADR is more common in children and young males. Females between the ages of 12 and 19 years are more prone to metoclopramide (Reglan)-induced ADR.[43] A history of ADR with neuroleptic therapy is an indicator for future risk of development of a MD.[44] Cocaine abuse increases the risk of a neuroleptic-induced ADR.[45] ADR typically involves cranial or truncal musculature. Children tend to have more generalized involvement, particularly in the trunk and extremities. Adults have a more restricted involvement of cranial, neck, and upper limb musculature. ADR is the most common cause of "oculogyric crisis," which consists of forced conjugate eye deviation upward or laterally, often accompanied by extension or lateral movements of the neck, mouth opening, and tongue protrusion. Blepharospasm, grimacing, trismus, forceful jaw opening, and tongue twisting are examples of involvement of other cranial musculature. Milder forms of muscle involvement can present as muscle cramps or tightness of jaw and tongue, leading to difficulty chewing, swallowing, and speaking. Respiratory stridor with resultant cyanosis can occur in patients with severe ADR.[46] ADR can result in extremely disabling dysarthria, dysphagia, jaw dislocation, compromised extremity function, and abnormal gait. ADR typically follows a varied course, with symptoms lasting from minutes to hours. ADR can be difficult to diagnose in the emergency department because abnormal movements can subside or fluctuate spontaneously, and can improve with reassurance of the patient.

The risk of developing ADR increases with the potency of the neuroleptic drugs and occurs more frequently with parenteral neuroleptics than with oral medications. The duration of symptoms depends on the half-life of the drug. Symptoms of ADR can be controlled quickly by parenteral administration of anticholinergics such as benztropine (Cogentin) or biperiden. The initial dose of benztropine is 2 mg given intravenously, with a maintenance dose of 1-2 mg orally twice daily for 7-14 days to prevent recurrence. Alternatively, diphenhydramine, which has antihistaminic and anticholinergic properties, can be given in a dose of 25-50 mg parenterally for rapid control of symptoms, and a maintenance dose of 25-50 mg orally 3-4 times daily for a few days. Some neurologists prescribe prophylactic use of amantadine for young males requiring neuroleptic therapy.

Akathisia is a subjective sensation of restlessness commonly associated with the inability to remain seated.[47] Abnormal limb sensation; inner restlessness, dysphoria, and anxiety are the commonly described symptoms associated with akathisia. This disabling condition can be mistaken for psychiatric illness such as agitation, hyperactivity, or anxiety in patients with agitated depression or schizophrenia.[48] Symptoms abate when the responsible medication is withheld, but management of this disorder often is very difficult.

Drug-induced parkinsonism (DIP) is associated with the use of neuroleptic medications, anti-nausea medication (metoclopramide), and antihypertensive agents (reserpine). The features of DIP generally are indistinguishable from those of idiopathic parkinsonism. A rhythmic, perioral, and perinasal tremor mimicking a rabbit chewing, termed rabbit syndrome, is typical of DIP.[49] The risk of developing DIP is higher in females than in males. Other risk factors include the dose and potency of neuroleptic medications. Anticholinergics and amantadine frequently are used to treat DIP, and have variable success.

Tardive disorder occurs following prolonged use of neuroleptic medications in about 20% of patients treated with these drugs.[50] Tardive disorder often is precipitated or worsened when the dose of the neuroleptic medication is reduced or the drug is withdrawn. Increasing age increases the risk for developing tardive dyskinesia,[51] and the probability of spontaneous remission

declines with advancing age. Involuntary stereotypical movements involving orofacial, neck, trunk, and axial muscles constitute the typical tardive dyskinesia. Patients commonly demonstrate pursing, smacking, chewing with frequent tongue protrusion, or pushing the tongue into the inner cheek.

Stimulants. Dextroamphetamine, methylphenidate (Ritalin), pemoline, and cocaine are all stimulant (dopaminomimetic) drugs with peripheral and central actions. Acute and chronic use of these drugs can result in chorea, orofacial dyskinesia, stereotyped movements, dystonia, and tics. Of these, stereotyped movements, comprising compulsive and complex activities, occur most often.

Oral Contraceptives. Chorea is the most frequently experienced MD caused by the use of oral contraceptives in otherwise healthy young females. It typically develops in a nulliparous woman who has been taking the contraceptive for nine weeks.[18] A unilateral distribution of chorea suggests the possibility of preexisting basal ganglia pathology. Symptoms generally abate within a few weeks following discontinuation of the contraceptive.

Antihistaminics and Anticholinergics. The use of chlorpheniramine and brompheniramine is associated with the development of orofacial dyskinesia, blepharospasm, tic-like movements, dystonia, and involuntary, semi-purposeful movements of the hands.[48] ADR with the use of diphenhydramine (Benadryl) has been reported.[52] The use of H_2-receptor blockers cimetidine and ranitidine is associated with the development of postural and action tremor, dystonic reactions, parkinsonism, confusion, and cerebellar dysfunction.[53] The movement abnormalities induced by these agents are generally short-lived and resolve after the responsible medication is discontinued.

Antidepressants. Although not common, tricyclic antidepressants such as amitriptyline, imipramine, and nortriptyline are known to cause choreiform movements, particularly orofacial dyskinesia.[54,55] The anticholinergic effects of tricyclic antidepressants are considered to be responsible for the development of chorea. The use of monoamine oxidase (MAO) inhibitors is associated with tremors and less often with myoclonic jerks.[56] As with MAO inhibitors, an overdose of tricyclic antidepressants is associated with myoclonus.

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Physician CME Questions

33. All the following statements regarding movement disorders resulting from an acute event such as a stroke are correct *except:*
    A. MDs are commonly manifested in a localized body area.
    B. MDs may follow a "hemidistribution," as in hemidystonia or hemiballism.
    C. Urgent brain imaging can be helpful following the acute onset of symptoms.
    D. MDs can be associated with an acute, primary neurological disease such as a cerebrovascular event.
    E. MDs are commonly believed to result from injury to the cerebral cortex.

34. Parkinson's disease:
    A. is caused by deficient serotonin within the caudate and the putamen.
    B. can result from medication use, such as phenothiazine.
    C. has a high prevalence in the middle age population.
    D. is an example of hyperkinetic MD.

35. Which of the following is *incorrect*?
    A. Seizures are common in Parkinson's disease.
    B. Falls in a patient with Parkinson's disease can be due to impaired postural reflexes.
    C. Prescribed medications for patients with Parkinson's disease can cause mental status change.
    D. Many patients with Parkinson's disease manifest varied pain symptoms such as painful muscle spasms, cramps, and burning paresthesias.
    E. In the parkinsonian patient, severe, localized limb pain, chest pain, and abdominal pain pose a diagnostic challenge in the emergency department.
    F. The most common chorea evaluated in the emergency department is probably L-dopa-induced chorea in a patient with parkinsonism.

36. All of the following are correct *except:*
    A. Torticollis can result from "focal dystonia," producing abnormal neck postures.
    B. Torticollis can be due to orthopedic and other neurological conditions.
    C. Neuroleptic medications and phenothiazines are responsible for most of the acute dystonic torticollis evaluated in the emergency department.
    D. Focal seizures can manifest as a torticollis.
    E. Dystonias do not respond to sensory tricks.

37. Which of the following statements about chorea is *incorrect*?
    A. Chorea involves a single body part.
    B. Chorea can be associated with the chronic use of numerous antiepileptic drugs.
    C. Chorea can have immunological or infectious etiologies.
    D. About one-third of patients with chorea gravidarum have Sydenham's chorea.
    E. Sydenham's chorea can occur several months after the onset of acute streptococcal infection.

38. All of the following are correct *except*:
    A. Transient tic disorder is common in children, with an estimated prevalence of 5-24% in school-age children.
    B. Conditions such as chronic cough or behavioral disorders can mimic tics in the emergency department.
    C. Tics can be associated with stroke, head trauma, and carbon monoxide poisoning.
    D. Tourette's syndrome has its onset in the third or fourth decade of life.
    E. Haloperidol and clonidine are used in the management of tics.

39. Physiological myoclonus:
    A. occurs in normal people.
    B occurs in the setting of a chronic seizure disorder.
    C. is an autosomal dominant hereditary disorder.
    D. has an identifiable underlying neurological or non-neurological disorder.

40. Which of the following is correct?
    A. Tardive disorder occurs following prolonged use of neuroleptic medications in about 20% of patients treated with these drugs.
    B. Tardive disorder often is precipitated when the dose of the neuroleptic medication is reduced or the drug is withdrawn.
    C. Tardive disorder often is worsened when the dose of the neuroleptic medication is reduced or the drug is withdrawn.
    D All of the above

In Future Issues: Urinary Tract Infection