Involuntary, uncontrolled muscle contractions can be caused by muscle or nervous system disorders. In patients with muscle disorders, these contractions are considered myotonia. Muscle contractions caused by nervous system disorders can appear clinically as tetanus, tetany, myoclonus, or movement disorders. Myoclonus can be sporadic or repetitive. The latter predominates and can be classified as constant, action related, postural, episodic, and resting. Most constant forms are related to canine distemper encephalomyelitis. Action-related forms can be congenital or acquired. Whereas most congenital forms involve hypomyelination, most acquired forms involve toxicity or an autoimmune steroid-responsive form. Postural myoclonus involves the head and neck and has a high incidence in Doberman pinschers, English bulldogs, and boxers; the condition can also involve the pelvic limbs of older dogs and an orthostatic form in Great Danes. Episodic myoclonus is rare. Movement disorders are most commonly breed related and are often difficult to differentiate from simple partial seizures.
Disorders of the muscle cell membrane may result in myotonia, which is a persistent, repetitive muscle cell contraction without relaxation after a physiologic stimulus. It represents a muscle cell membrane disorder. There are inherited and acquired forms of myotonia. The inherited forms are often called *congenital myotonia*, but the myotonic signs are not necessarily present at birth. They are usually first observed at a few weeks of age.

From a historical viewpoint, the most well-known myotonia is the type that occurs in goats. This type of myotonia has been recognized since the late 1800s but was not diagnosed as myotonia until the mid-20th century. The sudden onset of diffuse extensor muscle rigidity after an abrupt stimulus that often caused patient collapse was incorrectly called *fainting* or *epilepsy*. This disorder is now known to be the result of a chloride-channel defect inherited as an autosomal dominant gene with incomplete penetrance. The signs first occur at a few weeks of age and do not significantly progress. The degree of myotonia is variable, and some goats remain standing during the brief episode. Unique to this caprine disorder is that after the episode of myotonia, the affected goat completely recovers and remains refractory to another episode for about 30 minutes. Descriptions in the 1800s did not define the breed or breeds involved and stated only that they were common in Tennessee and Kentucky. Today, this disorder is most common in the pygmy breed. This caprine myotonia is similar to inherited myotonia in humans, which is known as *Thomsen’s disease*. A similar myotonia has been reported in Shropshire lambs, and we have observed it in a Montadale lamb.

In dogs, the two most common breeds in which this inherited myotonia occurs are the chow chow and miniature schnauzer. Very thorough studies of the disorder in miniature schnauzers by Dr. Charles Vite at the University of Pennsylvania determined that it was caused by a chloride-channel abnormality that is inherited as an autosomal recessive gene abnormality. The offending gene has been identified, and a polymerase chain reaction test is currently in use to determine the carriers. Affected chow chows were first described in Australia and have subsequently been diagnosed worldwide with this disorder, but the physiologic and genomic bases have not been determined. In both of these breeds, similar to what happens in goats, the myotonia can be elicited by a sudden movement by the relaxed animal, especially if the animal is startled. Unlike in goats, however, dogs do not fully recover between episodes. A variable degree of limb stiffness persists in the gait of affected dogs. They also develop a variable amount of muscle hypertrophy, especially in the proximal muscle groups of the limbs.

A similar early-onset myotonia that may be inherited has been reported in dogs of other breeds, domestic shorthaired cats, and horses. Myotonia is also observed in horses with inherited hyperkalemic periodic paralysis. A form of congenital myotonia that closely resembles the goat disorder has been observed in Brazilian Murrah buffalo (like affected goats, these calves are completely normal between episodes). Muscle hypertrophy is remarkable as these calves grow.

A form of myotonia may contribute to the gait abnormality observed in a number of myopathic disorders in which weakness is the most significant clinical sign. An example of this is the dystrophinopathy inherited in male golden retrievers, which has also been seen in numerous breeds as a spontaneous mutation.

Acquired myotonia is most commonly observed in older dogs with hyperadrenocorticoicism, which can be caused by a pituitary abnormality, an adrenocortical neoplasm, or prolonged corticosteroid administration. The myopathic disorder causes a persistent characteristic gait stiffness associated with significant muscle hypertrophy. Myotonia is observed via electromyography (EMG). The pathophysiology of the muscle disorder responsible for this is not well understood. Considering the large number of dogs that have hyper-

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*MUSCLE*

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Classification of Involuntary Muscle Contractions:

- **Muscle**
  - Myotonia

- **Nervous system**
  - Tetanus
  - Tetany
  - Myoclonus
    - Sporadic
    - Repetitive
      - Constant
      - Action related (congenital or acquired)
      - Postural
      - Episodic
      - Resting

- **Movement disorders**
adrenocorticoidism, this clinical entity is relatively rare. If untreated, this myotonia can be severe enough to prevent patients from standing and walking. In addition, treatment of hyperadrenocorticoidism does not necessarily result in recovery.

**NERVOUS SYSTEM**

Neuronal causes of involuntary muscle contractions involve spontaneous uncontrolled discharge of motor neurons. The resulting clinical signs can be grouped into four categories: tetanus, tetany, myoclonus, and movement disorders.

**Tetanus**

Tetanus is the clinical sign of nonintermittent contraction of extensor muscles. The degree of extensor muscle contraction varies among patients and is caused by disinhibition of extensor motor neurons. This is most commonly caused by the toxin produced by *Clostridium tetani* infection. This toxin interferes with the interneuronal release of the inhibitory neurotransmitter glycine in the spinal cord and γ-aminobutyric acid (GABA) in the brainstem. By strict definition, tetanus is a clinical sign, although the term is commonly used for the disease caused by *C. tetani* infection and its production of tetanospasmin.

The clinical sign of tetanus also occurs in the thoracic limbs of Australian cattle dogs with inherited poliomecephalomyelopathy because of the degeneration of interneurons in the cervical intumescence.

**Tetany**

Tetany is the clinical sign of variably intermittent contraction of extensor muscles. In strychnine intoxication, the toxin interferes with the release of glycine from spinal cord interneurons, but the degree of extensor muscle contraction varies in affected patients. The degree decreases in relaxed, resting patients but can be exacerbated by abrupt stimulation, causing patients to move suddenly.

Inherited congenital tetany occurs in polled Hereford calves. The tetany is present at birth and causes recumbency. Affected calves always maintain a degree of structural disorder. Glycine receptor studies have not been conducted in these dogs.

A similar inherited glycine receptor abnormality is described in humans as a cause of startle disease or hyperexplexia. In these patients, an external stimulus usually induces sudden contraction of primarily anti-gravity muscles.

Presumptive inherited tetany has been published as myoclonus in Peruvian pasos, in which a deficiency of spinal cord neuronal glycine receptor function was determined. The tetanic episodes and occasionally only myoclonus are stimulus induced. Some of these foals are able to stand and walk. Glycine deficiency has been determined to be worse in more severely affected recumbent animals.

A form of congenital tetany occurs in newborn Egyptian Arabian foals that have a color dilution haircoat color. These foals are recumbent at birth with a normal sensorium, but any effort to stand elicits severe extensor rigidity of all limbs as well as the trunk and neck, producing opisthotonus. The muscles are often...
remarkably relaxed between episodes, but these foals are never able to stand. These foals have no histologic lesions in the central nervous system (CNS).

Limited studies support the case for autosomal recessive inheritance of a gene linked to haircoat color. Breeders often refer to this as *lavender Arabian foal syndrome*. It is called *coat color dilution, lethal* in the literature.

**Myoclonus**

Myoclonus is the clinical sign of a sudden contraction of a group of muscle cells followed by immediate relaxation. Sporadic and repetitive forms of myoclonus are observed.

**Sporadic Myoclonus**

Sporadic myoclonus can be benign or a form of seizure disorder. Benign sporadic myoclonus can be defined as sudden contraction of a group of muscles causing, for example, a limb to move suddenly or the facial muscles to twitch. This movement is not repeated immediately. The cause is unknown.

Sporadic myoclonus that is repeated over a period of minutes to hours may be a form of simple partial seizure caused by a prosencephalic disorder that is often (but not necessarily) structural in origin. Idiopathic forms are less common. Patients with this disorder need to be studied like those with any other seizure disorder and treated with anticonvulsants.

**Repetitive Myoclonus**

Repetitive myoclonus can be constant (i.e., during action and rest and even during sleep). Repetitive myoclonus can also be action related and observed only when the patient is awake and contracting the muscles to maintain posture or to move. Other forms of repetitive myoclonus include postural, episodic, and resting.

Constant repetitive myoclonus is a unique syndrome occurring only in dogs and most often in those infected with canine distemper virus and with some degree of encephalomyelitis. We have also seen constant repetitive myoclonus in a dog with lead intoxication. The myoclonus in this patient resolved with chelation therapy.

This myoclonic syndrome is usually limited to one or two limbs, occasionally affects the jaw, and less often affects the whole body. The group of involved muscles does not change in affected dogs. The muscle contractions occur rhythmically (1 to a few seconds apart) and are most obvious in resting animals. They occur during activity but are often masked by the action involved. The contractions continue in the recumbent resting state and often during sleep. It is hypothesized that this is a functional disturbance in the environment of the lower motor neurons, innervating the myoclonic muscles, and is caused by some form of pacemaker mechanism that results in rhythmic stimulation of the participating lower motor neurons.
In many affected dogs, myoclonus can be stopped with intravenous lidocaine or oral procainamide. When these drugs are stopped, the myoclonus returns. Histologic lesions in the environment of these lower motor neurons are very mild or absent. What role this viral infection has in inducing this syndrome is unknown. Dogs with this myoclonus syndrome usually also have other clinical signs of neurologic deficits because of the destructive effect of this virus. In the older literature, this repetitive myoclonus was most commonly called canine chorea, which is a misnomer. In chorea, the myoclonic muscle groups continually change in affected patients. This is described in the section on movement disorders.

**Action-related repetitive myoclonus** usually diffusely affects skeletal muscle and is very rapid (i.e., many contractions per second), producing what is best described as a tremor. The more active the patient is and thus the more lower motor neuron stimulation recruited, the more rapid the myoclonus or tremor. This myoclonus disappears when the patient is totally relaxed or sleeping. Action-related myoclonus is sometimes referred to as intention tremors and is thought to be related to a cerebellar disorder. However, if these tremors are caused by a cerebellar disorder, obvious signs of cerebellar ataxia will be present, and the myoclonus will be limited to the head and neck. A diffuse, whole-body, action-related myoclonus cannot be produced with a lesion limited to the cerebellum. In our experience, this requires a diffuse disorder that can be structural (affecting myelin or neurons) or functional (caused by toxicosis or a neurotransmitter disorder). Congenital and acquired forms of repetitive action-related myoclonus have been observed.

**Congenital action-related myoclonus** (congenital tremors) is most commonly caused by a diffuse abnormality of CNS myelination (hypomyelination or dysmyelination). The tremors are observed at birth or as soon as the animal can stand and walk. They are action related and are not present in resting or sleeping animals. These congenital myelin disorders have been studied most extensively in pigs in which viral (hog cholera, swine fever, circa), inherited (Landrace, British saddleback), and toxic (trichlorfon from 43 to 65 days’ gestation) causes have been identified. In sheep and cattle, in utero infection with strains of the bovine virus diarrhea virus causes hypomyelination. In sheep, the fleece is abnormal, and these lambs are called hairy shakers. On occasion, affected calves and lambs “grow out of” the problem, presumably by eventually producing sufficient myelin to allow normal conduction.

In many breeds of dogs, although there have been numerous reports of hypomyelination-causing congenital tremors, no viral cause has been identified. In some breeds, an inherited basis has been documented (i.e., sex-linked recessive in male springer spaniels, autosomal recessive in Samoyeds, in which the disorder is lethal). However, in most descriptions, the inherited basis is presumed but not confirmed. We have observed a congenital coarse tremor in dalmatian puppies that produces a bouncing movement primarily in the pelvic limbs and trunk. Recovery occurred in a few weeks. The family incidence suggested an autosomal recessive inheritance. We have recently seen a similar disorder in a litter of golden retrievers.

Action-related myoclonus (congenital tremors) may accompany disturbances of neuronal function. A congenital diffuse central axonopathy has been described in quarter horse foals. This axonopathy causes coarse tremors that are most pronounced in the pelvic limbs and trunk, creating a bobbing, bouncing action similar to that seen in the dalmatian puppies already discussed. We have also observed a similar central axonopathy and coarse action-related tremor in newborn Holstein calves. These foals and calves need assis-

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**Rapid action–related myoclonus is a tremor that has many possible causes.**
Late-onset oligodendroglial dysplasia occurs in bullmastiffs and produces progressive spinal cord signs at a few months of age, reflecting a C1 through C5 anatomic diagnosis. These dogs also develop a mild diffuse whole-body tremor that distinguishes this diffuse myelin disorder from the more common focal cervical spinal cord lesions.

**Acquired action-related myoclonus** is most commonly observed as an acute-onset, diffuse, whole-body tremor that is nonprogressive and usually responds in a few days to a few weeks to immunosuppressive levels of corticosteroids. Continuous alternate-day therapy may be needed to prevent recurrence. On occasion, mild vestibular or cerebellar ataxia or other neurologic signs accompany the diffuse tremor. This disorder is most common in small white canine breeds (e.g., Maltese, West Highland white terrier), which is the basis for the name “white shaker syndrome”; however, this disorder can occur in any breed of any color. The larger canine breeds are less commonly affected. Imaging studies are usually normal. On occasion, we have seen evidence of mild meningoencephalitis via magnetic resonance imaging (MRI). Cerebrospinal fluid (CSF) may be normal or contain a slight elevation of lymphocytes and protein. The few histologic studies of the CNS show a very mild, nonsuppurative meningoencephalomyelitis consisting of a few scattered lymphocytic perivascular cuffs in the leptomeninges, parenchyma, and choroid plexus with no associated structural parenchymal lesions. The nature of the lesion and the response to immunosuppressive therapy suggest that this is an autoimmune disorder. The involved epitope may be a neurotransmitter or its cell membrane receptor. There is a precedent for an autoimmune reaction directed against a biochemical compound. The “stiff man syndrome” in humans is an autoimmune disease directed against glutamic acid decarboxylase, which is an enzyme necessary for the synthesis of GABA, an inhibitory neurotransmitter. In this syndrome, progressive persistent muscle spasms of the pelvic limbs are caused by central neuronal disinhibition.

Toxicosis is the most common condition initially resembling this autoimmune inflammation that needs to be differentiated from it. Many neurotoxins initiate a diffuse whole-body action-related tremor as the first clinical sign of intoxication. Depending on the toxin and amount of exposure, the patient may recover or progress to other CNS signs, including seizures and coma followed by death. These toxins include metaldehyde (snail bait), pyrethrins, lead, hexachlorophene, chlorinated hydrocarbons, organophosphates, and numerous mycotoxins. A common intoxication that causes acute onset of severe diffuse tremors is ingestion of penitrem-A—a mycotoxin produced by *Penicillium* spp of mold that grow on contaminated bread products or refrigerated products, such as cottage cheese. In this mycotoxicosis, multiple dogs in a household may develop similar signs after ingesting the same contaminated food product. Macadamia nuts contain a tremorgenic toxin that produces diffuse whole-body tremors in animals that ingest them.

**Postural repetitive myoclonus** involves muscle activity and therefore could be considered action related; however, this form appears to be limited to the postural muscles involved in weight support and is absent during voluntary movements. It primarily occurs in two forms: The first affects the head and neck postural muscles of relatively young dogs. The other occurs in the pelvic limbs of aged dogs. In addition, a unique severe form of postural myoclonus is pronounced in all the postural muscles of young adult Great Danes.

Episodic, rapid, repetitive myoclonus occurs most commonly in young adult (i.e., 6 months to a few years of age) Doberman pinschers, English and French bulldogs, and boxers; primarily involves the neck muscles; and causes a rapid head and neck tremor. The movement can be vertical or horizontal and appears to be present only when the head and neck are held in a supporting position. It disappears when the dog is distracted by a toy or food, during eating or any intentional activity, and when the dog lies down so that the head and neck are resting on a supporting surface. The tremor appears to depend on a specific degree of muscle tension in the neck before it occurs, suggesting that it involves some physiologic disturbance of the stretch reflex mechanism. This disorder is not progressive and is not associated with the development of any other neurologic signs. For unknown reasons, these tremors often occur sporadically for 1 week to a few weeks and then may recur after a few weeks to months. There is no pattern to their occurrence.
In this condition, the results of CSF and MRI studies of the head and neck are normal. There are no reports of electrodiagnostic testing, muscle and nerve biopsies, or CNS histologic studies. No studies have been conducted on the possible inheritance of this disorder. It can occur in any breed or mixed breeds but certainly predominates in Doberman pinschers, English bulldogs, and boxers, in which the term *head bobbers* is often used to describe the condition. This tremor syndrome may have some similarity to a benign postural tremor in humans referred to as *essential tremors*. The cause of this human disorder is poorly understood, but an abnormality of the stretch reflex mechanism has often been invoked. No well-designed results of drug studies have been published on the canine disorder. However, in our experience, anticonvulsants, including phenobarbital and potassium bromide, are unsurprisingly ineffective in treating affected patients.

In older dogs, benign, rapid, postural repetitive myoclonus tremor may occasionally develop in the pelvic limbs. All four limbs are rarely affected. This tremor is evident only when the dog is standing. It disappears or is completely masked during voluntary movement and when the dog is recumbent and, therefore, not supporting weight. This postural myoclonus can be elicited in a dog that is resting in lateral recumbency by applying pressure to the plantar surface of the paw. As in the head bobbbers described previously, this tremor appears to require a certain degree of tension in the limb muscles, suggesting a role of the stretch reflex mechanism in this disorder. No physiologic or pathologic studies have been published on this disorder. Although the intensity of the tremor may progress slightly with age, there is no indication that it causes discomfort, and it does not interfere with the dog’s function; therefore, it does not require therapy.

Orthostatic postural myoclonus (tremor) occurs in young adult Great Danes. It is observed only when the dog stands at rest; attempts to lie down; or postures to drink, eat, or excrete. The tremor becomes very severe while the dog lays down, prolonging the time it takes for this activity and causing the dog to constantly move and shift its body weight between its limbs. As soon as the dog is recumbent and relaxed, the tremor disappears. There is no evidence of a tremor when the dog walks or runs, and the dog does not show fatigue. The tremor disappears when the standing dog is picked up to avoid all weight support. The neurologic examination is normal, as are all the imaging studies, CSF studies, and muscle and nerve biopsies. EMG recordings in awake, standing dogs show a constant frequency of 13 to 16 Hz that disappears when the dog lies down. This is the basis for the diagnosis of orthostatic postural tremor in humans, which is thought to be a functional CNS disorder involving a supraspinal generator. On limited observation, these tremors slowly increase in intensity with time. An inherited basis is suspected in Great Danes.

*Postural tremors are benign and absent during voluntary movement.*
Danes. Drug therapy studies are limited and have been inconclusive to date. Phenobarbital and gabapentin may provide some relief.

**Episodic, nonpostural repetitive myoclonus** is a rare disorder in dogs that also classifies as a form of movement disorder. This is a poorly understood event that has generated numerous terms, the most common of which is myokymia. In humans, continuous muscle fiber activity is a more recent term applied to a group of hereditary and acquired conditions that all have continuous involuntary muscle contraction of peripheral nerve origin.\(^{36,37}\) Myokymia is the most common clinical sign and is defined as undulating vermiform movements of the overlying skin caused by contraction of small bands of muscle fibers. With EMG, the individual motor unit potentials fire at a rate of 5 to 150 Hz. In limited observations in dogs, these myokymic events clinically result in stiffness of the limbs followed by collapse into lateral recumbency with rigid limbs and delayed muscle relaxation. In affected dogs, these events are stimulated by exercise or excitement. Hyperthermia has been commonly observed. These episodes can last from a few minutes to a few hours, between which the dog is normal.

In humans, continuous muscle fiber activity is considered to represent hyperactivity of peripheral nerve axons not usually associated with any recognizable neuropathy. The term neuromyokymia has been used by some to implicate the role of the neuronal axon in this disorder.\(^{38}\) An axonal channel defect may be responsible because many humans have circulating antibodies to voltage-gated K channels of the peripheral nerve axons.

As veterinarians gain more experience with this rare canine disorder, its classification may need to be altered. Treatment options are limited, with procainamide and mexiletine being reasonable choices.

**Resting myoclonus** is present only during rest, as occurs in humans with degeneration of the substantia nigra from Parkinson’s disease. The condition has not been recognized in domestic animals. Horses that ingest the toxin present in yellow star thistle develop acute degeneration of the substantia nigra and globus pallidus but do not show a resting tremor. No resting tremor occurs as part of the clinical syndrome in Kerry blue terriers and Chinese crested dogs with cerebellar abiotrophy and degeneration of the substantia nigra and caudate nuclei.\(^{39,40}\)
Movement Disorders

A movement disorder is a sudden, spontaneous contraction of a group of skeletal muscles in a conscious patient with a normal sensorium during rest or activity. Various terms have been used for the different forms of these paroxysmal movements: *Chorea* is abrupt, non-sustained contraction of different groups of muscles; *dystonia* is sustained, involuntary contraction of a group of muscles; *athetosis* is prolonged contraction of trunk muscles, causing bending or writhing; and *ballism* is abrupt contraction of limb muscles, causing flailing of the limb.

In humans, the pathogenesis of many of these movement disorders is unknown. Some well-defined movement disorders are related to diseases of specific extrapyramidal nuclei, such as the caudate nuclei in Huntington’s chorea and resting myoclonus with substantia nigra lesions in Parkinson’s disease. Other disorders are unassociated with any recognizable CNS lesion, are often considered to represent some disorder of neuronal ion channels, and are referred to as ion channelopathies.

In veterinary medicine, movement disorders have been described in a number of breeds. For many years, a muscle contraction disorder called *Scottie cramps* has been recognized in Scottish terriers. In our opinion, this is a movement disorder. These involuntary movements are usually precipitated by exercise or stress. They consist of a combination of chorea and dystonia in one or more limbs. No histologic lesions have been found in the nervous systems of affected dogs. One study implicated a deficiency of serotonin activity in the spinal cord gray matter. This disorder is presumed to be genetically inherited in an autosomal recessive manner.

Other possibly familial movement disorders have been recognized in cavalier King Charles spaniels (tetany, hypertonicity, episodic falling, deer stalking), Bichon frises, soft-coated wheaten terriers (personal observation), and Norwich terriers. A classic severe form of movement disorder was described in two unrelated litters of boxers in which the movements were defined as paroxysmal dystonic choreoathetosis. MRI studies were normal in these dogs.

In addition, we have seen what we believe are forms of movement disorders in individual dogs of numerous breeds. In some instances, when the involuntary movement is repeated in the same group of muscles, it may be difficult to differentiate a movement disorder from a simple partial seizure disorder. This is particularly true for so-called Chinook seizures and the Border terrier episodes called *Spike disease.* If imaging and CSF studies are normal, an electroencephalographic study (when reliably available) or an anticonvulsant drug trial may be necessary to help differentiate between these disorders.

In our opinion, when the diagnosis of a movement disorder is suspected, MRI is warranted to determine the possible presence of extrapyramidal nuclear lesions. To date, we are not aware of publication of an article on such a relationship. A neurotransmitter or ion channel disorder would not be visible on an MRI scan.

What has been described in cavalier King Charles spaniels as tetany (i.e., sudden contraction of the extensor muscles of the pelvic limbs or all four limbs) may just as well be a form of dystonia. The latter is more likely because these episodes can be very brief or prolonged for a few minutes but are followed by complete recovery. This bears some resemblance to hyperexplexia in humans with glycine-receptor abnormality.

CONCLUSION

It is obviously difficult to classify all uncontrolled involuntary movements into specific entities. This classification is an attempt to provide some order where we believe there is a considerable lack of understanding. We hope this stimulates more accurate diagnosis of these disorders and provides a basis for improving our diagnostic and therapeutic capabilities as more scientific studies are conducted. We encourage readers to share their experiences with us and to offer constructive criticism of this classification.

REFERENCES


2. Myotonia is not a feature of
   a. hyperadrenocorticoidism.
   b. chloride-channel deficiency.
   c. muscular dystrophy.
   d. the loss of glycine inhibition.
   e. any breed of cattle.

3. Chow chows have an inherited disorder that causes
   a. tetany.
   b. myotonia.
   c. myoclonus.
   d. postural tremors.
   e. dystonia.

4. Thomsen’s disease in humans causes
   a. dystonia.
   b. myoclonus.
   c. postural tremors.
   d. myotonia.
   e. tetany.

5. Sporadic myoclonus may be a
   a. seizure disorder.
   b. stretch-reflex disorder.
   c. glycine-receptor deficiency.
   d. potassium-channel abnormality.
   e. form of myokymia.

6. Penitrem-A commonly causes
   a. glycine inhibition.
   b. tetanus or tetany.
   c. constant repetitive myoclonus.
   d. postural myoclonus.
   e. acquired action-related myoclonus.

7. The myoclonus observed with hypomyelinogenesis is
   a. sporadic.
   b. constant.
   c. action related.
   d. postural.
   e. episodic.

8. Congenital repetitive myoclonus is not seen as a sign of
   a. hypomyelinogenesis.
   b. glycine-receptor deficiency.
   c. neuronal storage disorder.
   d. globoid-cell leukodystrophy.
   e. bovine virus diarrhea viral infection.

9. Doberman pinschers, English bulldogs, and boxers are at risk for
   a. congenital myotonia.
   b. congenital tetany.
   c. dystonic chorea.
   d. acquired action-related repetitive myoclonus.
   e. postural repetitive myoclonus.

10. Hyperexplexia in humans is similar to
    a. muscular dystrophy in golden retrievers.
    b. congenital myotonia in miniature schnauzers.
    c. congenital tetany in Hereford calves.
    d. cramps in Scottish terriers.
    e. tetanus in Australian cattle dogs.