

# An approach to diagnosing neurological disease

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## KEY POINTS

- The key to neurological diagnosis is localization of the lesion.
- Recognition of the components of a syndrome allows localization of the lesion.
- Recognition of the components of a syndrome does not require sophisticated diagnostic techniques.
- The thoracolumbar syndrome is the most commonly seen spinal syndrome.
- The vestibular syndrome is the most commonly seen brain syndrome.

## INTRODUCTION

Unlike other body organs, the nervous system encompasses a myriad of subparts that typically have unique neuroanatomical and neurophysiological functions. Accordingly, localizing lesions within different areas of the nervous system is a challenge. This task is made easier by utilizing the clinical syndrome approach. A 'syndrome' is defined as a group of clinical signs that are usually seen together and are representative of specific organ system involvement. It is now established that specific lesions within the central nervous system (CNS), peripheral nervous system (PNS), and skeletal muscle result in predictable and specific clinical signs. It naturally follows that, by recognizing these key clinical signs (i.e., a syndrome), a lesion can be localized within any of these areas. This concept of neurological syndromes provides the basis for lesion localization, without which differential diagnosis of disease cannot logically be pursued.

## CLINICAL SYNDROMES

Fourteen neurological syndromes will be described in this article. In localizing a lesion, it is not necessary that all the clinical signs

listed for each syndrome be observed because a sufficient number of key clinical signs are usually present to permit accurate identification of the syndrome. Several diseases most commonly seen with each syndrome are listed and designated as more likely to be seen in dogs (D) and cats (C). Diseases without a designation occur in both species. For additional information on diseases known to cause any given syndrome, readers are advised to consult Braund, 1995 (1).

## Myopathic syndrome

Over the past 10 years, myopathic disorders have become quite common (1), although they are reported more often in dogs than in cats. Many myopathies are breed-related. Myopathies tend to have a bilaterally symmetrical distribution and reflexes are usually preserved, with the notable exception of Labrador Retriever hereditary myopathy, and sensory perception of pain is not impaired.

The myopathic syndrome is characterized by generalized weakness; exercise intolerance; fatigue; and a stiff, stilted gait (**Table 1**). While gait disturbance is worsened by exercise in the majority of myopathies (often with variable return of muscle strength following rest), in certain myotonic disorders, such as those reported in Chow Chows and Staffordshire Terriers, stiffness becomes less apparent with exercise. Also in these breeds, muscle mass is increased (hypertrophy), whereas in many other myopathies, muscle wasting (atrophy) tends to be a feature. In some animals muscles appear hypertrophic because of inflammation or spasms.

A temporary dimple contracture in a muscle (e.g., limb muscle or tongue) can be induced in certain myotonic myopathies, such as myotonia congenita, following a sudden tap with the hand or percussion hammer. Muscle pain, elicited by palpation, is often present in animals with polymyositis. Limited joint movement resulting from contracture is the hallmark of certain myopathies, for example, pelvic limb hyperextension in puppies with

**Table 1**  
**Myopathic syndrome**

- Generalized weakness
- Exercise intolerance
- Stiff, stilted gait
- Localized or generalized muscle atrophy
- Generalized muscle hypertrophy
- Dimple contracture
- Muscle pain on palpation
- Limited joint movement (e.g., contracture)

### *Common causes of the myopathic syndrome seen in practice*

- Polymyositis
- Masticatory myositis (D)
- Atrophic myopathy (D)
- Steroid myopathy
- Toxoplasma/neospora myositis (D)
- Labrador Retriever hereditary myopathy (D)



**Table 2**  
**Neuropathic syndrome**

*Motor neuropathy*

- Flaccid paresis/paralysis of structures innervated (e.g., limb/facial muscles, esophagus, anal sphincter)
- Neurogenic muscle atrophy
- Reduced/absent reflexes and muscle tone
- Muscle fasciculations

*Sensory neuropathy*

- Decreased pain response (hypalgesia) or sensation (hypesthesia)
- Proprioceptive deficits
- Abnormal sensation/sensitivity (paresthesia) of face, trunk, limbs
- Self-mutilation
- Reduced/absent reflexes without muscle atrophy

*Autonomic neuropathy (may be seen alone or in combination with sensorimotor neuropathies)*

- Anisocoria or dilated pupils
- Decreased tear secretion
- Decreased salivation
- Bradycardia

*Common causes of the neuropathic syndrome seen in practice*

- Idiopathic polyradiculoneuritis (D)
- Traumatic neuropathies (e.g., brachial plexus avulsion)
- Idiopathic facial paralysis (D)
- Ischemic neuromyopathy (C)
- Toxoplasma/neospora polyradiculoneuritis (D)
- Hypothyroid neuropathy (D)
- Tick paralysis
- Chronic inflammatory demyelinating neuropathy

toxoplasma/neospora myositis. Tremors and muscle fasciculations are sometimes seen in animals with myopathic disease. Some myopathies are potentially lethal, e.g., X-linked myopathy in Golden Retrievers, mitochondrial myopathies associated with lactic acidosis, and malignant hyperthermia. Junctionopathies, such as myasthenia gravis, may mimic signs of a myopathic syndrome.

### Neuropathic syndrome

The neuropathic syndrome (**Table 2**) is one of the more commonly encountered syndromes in clinical practice (1) and is frequently associated with trauma of peripheral and sometimes cranial nerves. The hallmarks of this syndrome are reduced, or absent, reflexes (hyporeflexia, areflexia), reduced or absent muscle tone (hypotonia, atonia, or flaccidity), weakness (paresis) or paralysis of limb/head muscles, and, after 1–2 weeks, neurogenic muscle atrophy.

This syndrome relates to motor nerve dysfunction and, as such, has been called lower motor neuron disease in other texts. Chronic neurogenic atrophy may result in severe fibrosis and limited joint movement from contractures (e.g., infectious polyradiculoneuritis due to toxoplasmosis or neosporosis). A variable degree of loss of pain (hypalgesia) or sensation (hypesthesia) may be detected upon cutaneous (dermatomal) testing, since most nerves contain motor and sensory components. Tremors and muscle fasciculations (e.g., postdenervation) are sometimes seen in animals with neuropathic disease. In animals with primary sensory neuropathies (e.g., sensory ganglioradiculitis, or breed-related sensory neuropathies in Boxers, Long-Haired Dachshunds, English Pointers, and Jack Russell Terriers), the syndrome may include loss of pain (analgesia) or sensation

**Table 3**  
**Lumbosacral syndrome**

- Flaccid weakness/paralysis of pelvic limbs and tail
- Depressed pelvic limb reflexes and flaccid muscle tone
- Muscle atrophy in pelvic limbs and/or hip muscles
- Postural reaction deficits in pelvic limbs
- Dilated anal sphincter
- Depressed bulbocavernosus reflex
- Reduced sensitivity in perineal area, pelvic limbs, or tail
- Urinary incontinence
- Fecal incontinence
- ± Root signature

*Common causes of the lumbosacral syndrome seen in practice*

- Pelvic fractures and luxations
- Fibrocartilaginous embolization
- Lumbosacral stenosis (D)
- Disk disease
- Sacrococcygeal dysgenesis

(anesthesia) and/or proprioception, abnormal sensitivity about the face or trunk (paresthesia), self-mutilation (perhaps, as a result of paresthesia), and hyporeflexia/areflexia without muscle atrophy.

Peripheral neuropathies commonly involve a single nerve (i.e., mononeuropathy), such as common peroneal, radial, or facial nerves. Polyneuropathies involve several nerves, are usually bilaterally symmetrical, and are best exemplified by polyradiculoneuritis (e.g., coonhound paralysis). Other less common degenerative polyneuropathies may have a proximal limb muscle distribution (e.g., hereditary spinal muscular atrophy in Brittany Spaniels) or a distal limb muscle distribution (e.g., giant axonal neuropathy in German Shepherds or distal polyneuropathy in adult Rottweilers). Pelvic limbs are usually first affected in generalized polyneuropathies. Whereas some neuropathies may have an acute (e.g., traumatic or ischemic neuropathies) or subacute onset (e.g., polyradiculoneuritis) the majority of neuropathies are insidious in onset and have a chronic course.

Chronic, relapsing polyneuropathies are becoming more commonly observed in dogs and cats. Some of these newly recognized, relapsing conditions are self-limiting and/or steroid-responsive. While signs of autonomic nerve dysfunction (anisocoria, decreased tear secretion, bradycardia, etc.) are infrequently observed in animals with polyneuropathies, they are a feature in dogs and cats with dysautonomia. Similarly, cranial nerve dysfunction is uncommon, with the exception of facial neuropathy (cranial nerve VII) in polyradiculoneuritis and hypothyroid neuropathy, and vagus neuropathy (cranial nerve X), resulting in (a) dysphagia and megaesophagus in giant axonal neuropathy in German Shepherd dogs and (b) laryngeal paralysis and megaesophagus in young dogs (e.g. Dalmatians) and in older, large-breed dogs with laryngeal paralysis–polyneuropathy complex.

Certain disorders of the neuromuscular junction, namely botulism and tick paralysis, produce signs that mimic those observed in a diffuse polyneuropathy. Metabolic neuropathies, such as diabetic neuropathy, are now regularly diagnosed. Ill-defined peripheral nerve disorders in older dogs may be an immunological manifestation of various systemic malignant tumors – the so-called paraneoplastic neuropathies. Nerve sheath tumors are a relatively common cause of brachial plexus neuropathy.

### Lumbosacral syndrome

This syndrome represents the first of four spinal cord

**Table 4**  
**Thoracolumbar syndrome**

- Spastic weakness/paralysis of pelvic limbs
- Pelvic limb reflexes normal or brisk (clonus may be observed)
- No muscle atrophy in pelvic limbs
- Postural reaction deficits in pelvic limbs
- Increased local sensitivity (hyperesthesia) at level of lesion
- Reduced sensitivity behind level of lesion
- Urinary incontinence
- ± Schiff-Sherrington posture

*Common causes of the thoracolumbar syndrome seen in practice*

- Disk disease
- Spinal fractures
- Degenerative myelopathy
- Distemper myelitis (D)
- Diskospondylitis (D)
- Metastatic lymphosarcoma (C)
- Vertebral anomaly (e.g., hemivertebra)

syndromes. Lesions involving (a) spinal cord segments L4 and L5 through S1 through S3 (+ coccygeal segments) or (b) lumbosacral nerve roots that form the cauda equina (including femoral, obturator, sciatic, pudendal, pelvic, and coccygeal nerves) will result in a lumbosacral syndrome (**Table 3**). The lumbosacral syndrome reflects various degrees of involvement of the pelvic limbs, bladder, anal sphincter, and tail. Clinical signs will range from flaccid weakness to paralysis of pelvic limbs and tail. Patellar and withdrawal reflexes (as well as gastrocnemius and cranial tibial reflexes) may be depressed or absent in pelvic limbs, as may be perineal (anal) and bulbocavernosus (in male dogs) reflexes. Tone in pelvic limb muscles may be reduced or absent. After 1–2 weeks of clinical signs, segmental muscle atrophy due to denervation will be observed. ‘Segmental’ refers to the particular spinal cord segment involved in the lesion (e.g., segmental atrophy may develop in the iliopsoas, quadriceps, and sartorius muscles following an injury to the L4 through L6 spinal cord segments). Pain perception in pelvic limbs, tail, and perineum may be reduced or absent and pelvic limb postural reactions such as hopping and placing may be depressed. Thoracic limb function is normal. The anal sphincter may be flaccid and dilated, resulting in fecal incontinence. The bladder is frequently paralyzed, resulting in urine retention and passive overflow incontinence. With this syndrome, the flaccid bladder is easily evacuated manually.

In some animals with lumbosacral disk extrusion, one pelvic limb may be held in partial flexion or a repetitive ‘stamping’ motion may be observed. These animals frequently show considerable pain on manipulation of the limb and lumbosacral spine. This combination of signs is termed ‘root signature’ and is believed to be associated with nerve root compression or entrapment by a fragment of extruded disk material. It is important to note that some animals with the lumbosacral syndrome will be paretic or paralyzed in the pelvic limbs, with reduced reflexes and muscle tone, but will have normal anal sphincter function. In other animals, anal sphincter and bladder dysfunction may be the principal clinical signs, with only mild pelvic limb weakness. Both groups of animals have a lumbosacral syndrome, but the lesion occurs at slightly different levels of the lumbosacral spinal cord or lumbosacral nerve roots.

### Thoracolumbar syndrome

A spinal cord lesion located between cervical and lumbar enlargements (intumescences), that is, between T3 and L3 cord

segments, will produce a thoracolumbar syndrome (**Table 4**). This is the most commonly encountered spinal cord syndrome in dogs and cats. The thoracolumbar syndrome is characterized by spastic weakness or paralysis of pelvic limbs – spasticity is associated with increased muscle tone, especially in extensor muscles. Ataxia may be observed in ambulatory animals (e.g., crossing of the pelvic limbs when walking, knuckling, or abnormal abduction or protraction of the pelvic limbs). Pelvic limb reflexes are intact (normal or increased). However, postural reactions such as hopping and placing are depressed in pelvic limbs. In some animals, reflex testing may induce clonus (spasms in which contraction and relaxation of limb muscles alternate in rapid succession). Flexor reflex testing may also induce prolonged, repetitive flexion of the limb being tested in the absence of repeated stimuli (2). A crossed extensor reflex may be observed. Thoracic limb function is normal.

Animals with thoracolumbar disk disease may keep their backs slightly arched (‘kyphosis’). Often, there is reduced cutaneous sensation along the dorsal spine behind the lesion site, but sensation is increased at, or immediately above, the level of the lesion. In dogs with thoracolumbar disk disease, digital pressure on the spine at the level of disk extrusion will usually elicit back pain. Animals are usually incontinent, with a characteristic inadequate spurting urination of brief duration (‘spastic bladder’). It is difficult (and may be dangerous) to express the bladder manually because of hypertonia (spasticity) of the external urethral sphincter.

Segmental muscle atrophy is *not* a feature of the thoracolumbar syndrome. However, atrophy caused by disuse can occur in animals with long-term or permanent paralysis. Such atrophy is usually generalized and involves all muscles of the spine caudal to the level of the spinal cord lesion, as well as muscles of the pelvic limbs.

An acute, compressive lesion of the thoracolumbar spinal cord occasionally may be accompanied by a Schiff-Sherrington posture, which is observed as rigid extension of the thoracic limbs with the animal in lateral recumbency. However, voluntary movement (with support) and postural reactions, such as wheelbarrow and hopping, are normal in the thoracic limbs. The wheelbarrow reaction is particularly useful for testing thoracic limb function. It is usually depressed in animals with cervicothoracic or cervical syndromes. It should be noted that this test is manipulative and should not be performed on animals with vertebral column injuries.

### Cervicothoracic syndrome

The cervicothoracic spinal cord segments that extend from C6 through T2 form an enlarged area of the cord known as the cervical intumescence. The gray matter of these segments gives rise to various nerves (e.g., suprascapular, musculocutaneous, axillary, radial, median, and ulnar nerves) supplying thoracic limb muscles. A lesion in this region of the spinal cord produces signs of a cervicothoracic syndrome. The hallmarks of the cervicothoracic syndrome (**Table 5**) are weakness or paralysis in both thoracic limbs, in all four limbs (i.e., tetraparesis or tetraplegia), in limbs on the same side of the body (i.e., hemiparesis or hemiplegia), or in only one thoracic limb. Ataxia may be observed in ambulatory animals. Other signs include depressed or absent reflexes (tricipital, bicipital, withdrawal) and decreased or flaccid muscle tone in one or both thoracic limb(s). As with a lumbosacral syndrome, segmental muscle atrophy due to denervation is usually observed in the thoracic limb(s) of animals 1 to 2 weeks after spinal injury. In pelvic limbs, reflexes are intact and may be increased (brisk), but there is no atrophy. Postural reactions, such as hopping and placing, may be depressed in all limbs, especially in the thoracic. In some instances, animals will clumsily propel themselves on their chins using their pelvic limbs, with thoracic limbs drawn to their flanks.

The cutaneous trunci reflex, mediated by the lateral thoracic



**Table 5**  
**Cervicothoracic syndrome**

- Weakness/paralysis in:
    - all four limbs (i.e., tetraparesis/tetraplegia)
    - limbs on the same side of the body (i.e., hemiparesis/hemiplegia)
    - only one thoracic limb (i.e., monoparesis/monoplegia)
  - Depressed reflexes and flaccid muscle tone in thoracic limb(s), muscle atrophy after 1–2 weeks
  - Normal/increased reflexes and muscle tone, without atrophy, in pelvic limb(s)
  - Postural reaction deficits in one thoracic limb, in limbs on the same side, or in all limbs
  - Increased local sensitivity at level of lesion
  - Reduced sensitivity behind level of lesion
  - Cutaneous trunci reflex depressed or absent (unilaterally or bilaterally)
  - Horner's syndrome
    - Miosis
    - Enophthalmos
    - Ptosis
    - Protrusion of third eyelid
- Common causes of the cervicothoracic syndrome seen in practice*
- Brachial plexus avulsion
  - Brachial plexus sheath tumors (e.g., neurofibromas)
  - Fibrocartilaginous embolization
  - Wobbler syndrome (D)
  - Disk disease

nerve that originates in cord segments C8 through T2, may be depressed or absent unilaterally or bilaterally, depending on the extent and location of the lesion. Animals with lesions in cord segments T1 through T3 may have signs of a Horner's syndrome – miosis (small pupil), ptosis (upper lid droop), enophthalmos (sunken globe), and prolapse of the third eyelid.

One condition that mimics the cervicothoracic syndrome is traumatic avulsion of the brachial plexus. Animals with this disorder may show evidence of areflexia, muscle atrophy, and weakness/paralysis of one thoracic limb together with signs of a partial Horner's syndrome in which only miosis is observed. The miosis will be ipsilateral – that is, on the same side as the paralyzed thoracic limb. In animals with brachial plexus avulsion, postural reactions will be depressed in the affected limb but normal in all other limbs.

### Cervical syndrome

A lesion between C1 and C5 spinal cord segments produces the cervical syndrome (**Table 6**). As with the thoracolumbar syndrome, clinical signs reflect disruption of white matter pathways rather than gray matter involvement (as seen in lumbosacral and cervicothoracic syndromes). With a cervical syndrome, clinical signs may range from weakness to spastic paralysis of all four limbs or of limbs on the same side of the body. Ataxia may be observed in ambulatory animals. Postural reactions are usually depressed or absent in all limbs. Dorsal and lateral compressive lesions of the cervical spinal cord may result in signs being more severe in the pelvic limbs, perhaps because of the more superficial location of the ascending proprioceptive pathways from the pelvic limbs. In contrast, a ventral median compressive lesion may produce more severe signs in the thoracic limbs, perhaps because of the more medial location of descending motor tracts projecting to the cervical intumescence (3).

**Table 6**  
**Cervical syndrome**

- Spastic weakness/paralysis in:
    - all four limbs, or
    - limbs on the same side of the body as the lesion
  - Normal/increased reflexes and muscle tone, in all limb(s) ± clasp-knife extensor rigidity in limbs on the same side as the lesion or in all limbs
  - Postural reaction deficits in limbs on the same side as the lesion or in all limbs
  - Cervical muscle spasms, pain and/or rigidity (animals may resist neck flexion/extension)
  - Root signature
    - ± Respiratory difficulty
    - ± Horner's syndrome
- Common causes of the cervical syndrome seen in practice*
- Cervical trauma
  - Disk disease
  - Wobbler syndrome (D)
  - Meningitis
  - Atlantoaxial subluxation
  - Diskospondylitis (D)
  - Fibrocartilaginous embolization

A more centrally located lesion within the spinal cord (e.g., a centrally expanding intramedullary tumor or central necrosis secondary to acute spinal trauma) may produce more severe signs in the thoracic limbs because motor tracts of the thoracic limbs lie more centrally than do those of the pelvic limbs (4). Reflexes and muscle tone are intact or increased in all limbs. In some animals with a severe cervical cord lesion, muscle tone may be increased to the point of pronounced extensor rigidity that may be clasp-knife in character (in which a rigidly hyperextended limb suddenly gives way to forced flexion). There is no evidence of segmental muscle atrophy in any of the limbs. Affected animals may experience variable loss of pain perception in all limbs and in the neck caudal to the level of the lesion. However, it is unusual to detect complete loss of pain sensation, since spinal cord injury of such magnitude would most likely be accompanied by respiratory failure.

Cervical muscle spasms, pain on palpation or manipulation, and cervical rigidity due to splinting of the neck muscles will be present in some animals (e.g., dogs with cervical disk disease). These dogs strenuously resist flexion and extension of their necks and they may assume an abnormal posture with the nose held close to the ground and the back arched. In some dogs with cervical disk disease, one thoracic limb may be held in partial flexion, or a repetitive 'stamping' motion may be observed. These animals frequently show considerable pain on manipulation of the limb and neck. This combination of signs is termed 'root signature' and is believed to be associated with nerve root compression or entrapment by a fragment of extruded disk material.

Occasionally, an animal may manifest a variable degree of respiratory difficulty. Rarely, an ipsilateral Horner's syndrome may be present in an animal with a severe destructive lesion in the cervical cord (e.g., infarction secondary to fibrocartilaginous embolization).

### Pontomedullary syndrome

Diseases involving the pons and medulla oblongata can produce the pontomedullary syndrome (**Table 7**). This syndrome is characterized by the presence of multiple cranial nerve deficits in an animal showing signs of ipsilateral hemiparesis/hemiplegia or

Table 7

**Pontomedullary syndrome**

- Spastic weakness/paralysis in:
    - all four limbs, or
    - limbs on the same side of the body as the lesion
  - Normal/increased reflexes and muscle tone in all limb(s)
  - Postural reaction deficits in limbs on the same side as the lesion or in all limbs
  - Multiple cranial nerve deficits
    - Jaw paralysis, decreased facial sensation (cranial nerve V)
    - Depressed palpebral reflex (cranial nerves V, VII)
    - Facial paralysis (cranial nerve VII)
    - Head tilt, falling, rolling, nystagmus (cranial nerve VIII)
    - Pharyngeal/laryngeal paralysis (cranial nerves IX, X)
    - Tongue paralysis (cranial nerve XII)
  - Irregular respiration
  - Mental depression
- Common causes of the pontomedullary syndrome seen in practice*
- Cranial trauma
  - Distemper encephalitis (D)
  - Granulomatous meningoencephalomyelitis (reticulosis)
  - Rabies
  - Pseudorabies
  - Feline infectious peritonitis (C)
  - Choroid plexus papilloma

Table 8

**Cerebellar syndrome**

- Spastic, goose-stepping gait in all limbs, especially thoracic, with preservation of strength
  - Truncal ataxia
  - Intention tremors of head, eyes
  - Broad-based stance
  - Postural reactions delayed, with exaggerated responses
  - Menace deficit (ipsilateral), with normal vision
  - ± Opisthotonos (rare)
  - ± Vestibular signs (rare)
  - ± Anisocoria (contralateral pupil dilated)
- Common causes of the cerebellar syndrome seen in practice*
- Cerebellar malformation (hypoplasia)
  - Canine distemper encephalitis (D)
  - Hypomyelinogenesis
  - Feline infectious peritonitis (C)
  - Cerebellar degeneration
  - Choroid plexus papilloma

tetraparesis/tetraplegia. Ataxia may be observed in ambulatory animals. Postural reactions can be depressed in all limbs or in limbs on one side only, ipsilateral to the side of the lesion. Reflexes are intact in all limbs. Heightened muscle tone may cause limb spasticity, similar to that seen in animals with a cervical syndrome.

Pontomedullary lesions involving cranial nerve nuclei and/or cranial nerves may produce a variety of clinical signs, including jaw paralysis, masticatory muscle atrophy, decreased facial sensation, and depressed palpebral reflex (cranial nerve V; trigeminal); medial strabismus (cranial nerve VI; abducent); inability to close eyelid(s), lip paralysis, ear droop, facial spasms (cranial nerve VII; facial); head tilt, rolling, nystagmus (cranial nerve VIII; vestibular); pharyngeal paralysis resulting in dysphagia and depressed gag reflex (cranial nerve IX; glossopharyngeal); laryngeal/esophageal paralysis resulting in cyanosis, dysphonia, inspiratory distress, megaesophagus (cranial nerve X; vagal); and tongue paralysis (cranial nerve XII; hypoglossal). The respiration of animals with severe pontomedullary lesions may be irregular and apneic (i.e., apneustic respiration); rapid and shallow (i.e., central neurogenic hyperventilation); or shallow, slow, but regular (i.e., central alveolar hypoventilation) (5).

Mental depression may be observed as a consequence of disruption of the ascending reticular activating system (e.g., in animals with intracranial injury). Certain primary tumors, such as medulloblastomas, choroid plexus papillomas, and epidermoid cysts, as well as some inflammatory diseases, including canine distemper encephalitis, rabies, and the disseminated form of granulomatous meningoencephalomyelitis, have a predilection for this area of the brain stem.

**Cerebellar syndrome**

This is one of the most readily recognizable syndromes in veterinary practice. The cerebellum is a reinforcing and coordinating organ that plays an important role in harmonizing muscle contraction. Cerebellar disease results in an inability to regulate the rate, range, and force of a movement (i.e., dysmetria). Clinical signs

of the cerebellar syndrome (**Table 8**) include an exaggerated limb response when a movement is initiated, such as 'goose-stepping' (hypermetria) when walking, or a delay followed by an exaggerated response during postural reaction testing, such as hopping and placing or overshooting a food bowl when attempting to eat.

Limb movements are typically spastic, clumsy, faltering, and jerky. The animal assumes a broad-based stance at rest, and swaying of the trunk (i.e., truncal ataxia) may be observed when the animal is walking. Initiation of movement is delayed and often accompanied by tremors (i.e., intention tremors). Tremors are especially noticeable involving the head. Intention tremors disappear at rest. Fine, pendular, or oscillatory eye movements may also be present. A bilateral menace deficit may be noted, although vision is not affected. If the lesion involves only one side of the cerebellum, the menace deficit will be ipsilateral. Anisocoria is sometimes detected in animals with cerebellar lesions. Usually the pupil contralateral to the side of the lesion will be slightly dilated. Both pupils respond normally to light directed into either eye.

Infrequently observed signs associated with specific areas of the cerebellum include opisthotonos (e.g., when a lesion involves the rostral lobe of the cerebellum) and vestibular signs (e.g., when a lesion occurs in the flocculonodular lobe or fastigial nuclear area of the cerebellum) (6).

**Vestibular syndrome**

The vestibular syndrome (**Table 9**) is another commonly recognized syndrome in clinical practice. Clinical signs may be caused by (a) central lesions involving the vestibular nuclei located on either side of the medulla oblongata beneath the floor of the fourth ventricle (7) or (b) peripheral lesions involving the vestibular portion of the eighth cranial nerve or, more commonly, the vestibular receptors in the membranous labyrinth located within the petrous portion of the temporal bone. Vestibular disease results in loss of equilibrium. Peripheral vestibular diseases are more common than central disorders. Clinical signs of peripheral vestibular disease include ipsilateral head tilt, falling, rolling, nystagmus, or walking in tight circles. There may be exaggerated extensor tone of the contralateral limbs, accompanied by decreased tone in ipsilateral limbs (8). Strength is preserved in peripheral vestibular disease. Nystagmus is present in the acute stages of most peripheral vestibular diseases and is usually jerking in nature with fast and



**Table 9**  
**Vestibular syndrome**

	<i>Central vestibular disease</i>	<i>Peripheral vestibular disease</i>
Loss of balance	Yes	Yes
Head tilt	Yes	Yes
Falling/rolling	Yes (greater tendency to roll)	Yes
Nystagmus	Yes	Yes
horizontal	Yes	Yes
rotatory	Yes	Yes
vertical	Yes	No
positional	Yes	No
Strabismus (ventrolateral)	Yes	Yes
Cranial nerve deficits	Possible V, VI, VII	Possible VII
Horner's syndrome	No	Possible
Cerebellar signs	Possible	No
Mental depression	Possible	No
Hemiparesis with ipsilateral postural reaction deficits	Possible	No

*Common causes of the vestibular syndrome seen in practice*

- Peripheral labyrinthitis (i.e., otitis media/otitis interna)
- Feline and canine idiopathic vestibular disease
- Drug ototoxicity
- Congenital vestibular disease
- Central granulomatous meningoencephalitis
- Choroid plexus papilloma
- Distemper encephalitis (D)
- Toxoplasmosis
- Cryptococcosis

slow components. The quick phase of horizontal nystagmus is in a direction away from the side of the lesion and it is the compensatory phase of the eye movement. Sometimes in animals with vestibular disease, nystagmus can be initiated by moving and holding the head in a different position (i.e., positional nystagmus).

Normal, physiological nystagmus can be induced by rapid head movements in vertical or horizontal planes. The fast phase of the nystagmus is in the direction of the head movement. This response may be depressed or absent in animals with vestibular disease when the head is moved toward the side of the lesion. A ventrolateral strabismus (abnormal position of the eyeball) may be elicited in affected animals by extending the head. The strabismus is ipsilateral. Horner's syndrome and facial paralysis are frequently observed with peripheral vestibular diseases that are associated with otitis media/otitis interna, since both facial and sympathetic nerves pass through the middle ear. Righting reactions are diminished or absent in affected animals.

Central vestibular disease is suggested by the presence of ipsilateral vertical, horizontal, or rotatory nystagmus, which may change direction with different positioning of the head. Additionally, there may be altered mental status, hemiparesis and/or ataxia, ipsilateral postural reaction deficits, a greater tendency to roll in one direction, and clinical evidence of other cranial nerve dysfunction, such as involvement of the trigeminal nerve (e.g., ipsilateral jaw weakness, decreased facial sensation, depressed palpebral reflex) and/or abducent nerve (e.g., ipsilateral medial strabismus).

Infrequently, mass lesions located in (a) the caudal cerebellar peduncle (containing axons connecting vestibular and cerebellar nuclei) or (b) the flocculonodular lobe of the cerebellum (where the vestibulocerebellar fibers largely terminate) may cause head tilt,

**Table 10**  
**Midbrain syndrome**

- Spastic weakness/paralysis in:
    - all four limbs, or
    - limbs on the contralateral side of the body
  - Increased reflexes and muscle tone in limbs on the contralateral side or in all limbs (all limbs may be held in rigid extension)
  - Postural reaction deficits in limbs on the contralateral side or in all limbs
  - Mental depression or coma
  - Ipsilateral deficits of cranial nerve III (oculomotor)
    - ventrolateral strabismus
    - dilated pupil unresponsive to light, with normal vision
    - drooping of upper eyelid (ptosis)
  - Hyperventilation
  - ± Bilateral miosis
  - ± Obstinate progression/head pressing (C)
- Common causes of the midbrain syndrome seen in practice*
- Thiamine deficiency (C)
  - Cranial trauma with midbrain compression and/or hemorrhage
  - Distemper encephalitis (D)
  - Granulomatous meningoencephalomyelitis

body tilt and strabismus on the side opposite that of the lesion (9). This has been termed paradoxical vestibular disease (10, 11). The lesion is on the side that has the postural reaction and proprioceptive deficits. Mass lesions include neoplasms, especially choroid plexus papillomas, and the focal form of granulomatous meningoencephalomyelitis.

**Midbrain syndrome**

This is a relatively uncommon syndrome (**Table 10**). Animals may be depressed or comatose, and there may be rigid extension of all limbs. If the lesion is located on one side of the midbrain, limbs on the contralateral side will show signs of hemiparesis or hemiplegia. Ataxia may be observed in ambulatory animals. If the oculomotor nucleus and/or nerve is involved, animals will have a ventrolateral strabismus, a widely dilated pupil that is unresponsive to light stimulation in either eye, and ptosis (drooping) of the upper eyelids. These signs may be ipsilateral or bilateral, depending on the location and extent of the lesion. Vision is usually normal. Rarely, visual impairment and menace deficit contralateral to the side of the lesion may be noted in animals with lesions involving the lateral geniculate body. Hyperventilation is seen in some animals.

In animals with severe cranial trauma that diffusely involves the midbrain, bilateral pupillary miosis may be seen initially, with a gradual change to fixed, dilated pupils. Lesions located in the ventral midline (i.e., interpeduncular area) in cats can produce signs of obstinate progression in which cats propel themselves forward until meeting an obstacle and continue to push against it (head pressing) (12).

**Hypothalamic syndrome**

The hypothalamus is the most ventral portion of the diencephalon and is intimately involved in autonomic visceral body functions, including appetite, sexual activity, sleep-wake cycle, body temperature, blood pressure regulation, and emotions (13). It also regulates much of the body's endocrine activity. While the hypothalamic syndrome (**Table 11**) is relatively uncommon in dogs and cats, it is most often associated with pituitary tumors. Animals

**Table 11**  
**Hypothalamic syndrome**

- Normal gait
- Altered mental status (disorientation, lethargy, or coma)
- Change in behavior (aggression or hyperexcitability)
- Abnormal movements/postures (trembling, pacing, wandering, hiding, tight circling, or head pressing)
- Bilateral deficits of cranial nerve II (optic) at the level of the optic chiasm (visual impairment, dilated pupils, depressed pupillary reflexes)
- Abnormal temperature regulation (hyperthermia, hypothermia, or poikilothermia)
- Abnormal appetite (hyperphagia/obesity or anorexia/cachexia)
- Endocrine disturbances (diabetes insipidus, diabetes mellitus, hyperadrenocorticism, or acromegaly/excess growth hormone)
- Seizures

*Common causes of the hypothalamic syndrome seen in practice*

- Pituitary tumors
- Granulomatous masses (e.g., toxoplasmosis) and mycotic diseases (e.g., cryptococcosis, blastomycosis)

may show signs of altered mental status (e.g., disorientation, lethargy, or coma) and/or behavior changes (e.g., aggression, hyperexcitability, pacing, wandering, hiding, tight circling, head pressing, and trembling). Seizures may be noted. Gait is usually normal.

Abnormal temperature regulation may be manifested as hyperthermia, hypothermia, or poikilothermia. Abnormalities in appetite are seen as hyperphagia and obesity, or anorexia and cachexia. Vision is frequently impaired if the lesion extends to involve the optic chiasm, in which case pupils may be dilated and weakly or not responsive to light stimulation. Endocrine disturbances most often include diabetes insipidus or hyperadrenocorticism.

### Cerebral syndrome

This commonly occurring syndrome (**Table 12**) is often characterized by abnormal movement, such as circling (usually to the same side as the lesion), continual pacing, or head pressing into a wall or cage. In some animals, the head and trunk may be twisted (i.e., pleurothotonos) toward the side of the lesion. Altered behavior and mental status are frequently observed: apathy, depression or stupor, disorientation, failure to recognize the owner or environment, loss of trained habits (e.g., house training), and sometimes aggression or hyperexcitability. Vision may be impaired (e.g., bumping into objects, depressed menace reflex) on the side opposite the lesion, although pupillary light reflexes are normal.

Seizures and papilledema may be observed. Seizures may be (a) generalized, with loss of consciousness and uncontrolled autonomic activity (e.g., salivation, urination, defecation, pupillary dilation, and chewing movements) and abnormal motor function (e.g., muscular rigidity, followed by running and paddling movements of the limbs), or (b) partial, where there is no loss of consciousness and where signs may indicate the location of the seizure focus, for example, motor cortex head turning, spasms in one limb, tail chasing; visual cortex light or fly biting; or limbic system confusion, viciousness, screaming, attacking inanimate objects, fearful behavior, etc. Partial seizures may spread to become generalized seizures. While animals may have a normal gait, postural reactions such as hopping, placing, and hemiwalking are usually depressed in the contralateral limbs. In comatose animals, breathing may be characterized by waxing and waning of the depth of respiration, with regularly recurring periods of apnea (i.e., Cheyne-Stokes respiration).

**Table 12**  
**Cerebral syndrome**

- Normal gait
- Altered mental status (apathy, depression, disorientation, lethargy, coma)
- Change in behavior (loss of trained habits, failure to recognize owner, aggression or hyperexcitability)
- Abnormal movements/postures (pacing, wandering, circling, or head pressing), twisted head and trunk (pleurothotonos)
- Postural reaction deficits in contralateral limbs
- Visual impairment (e.g., bumping into objects, contralateral menace deficit) with normal pupillary reflexes
- Seizures
- ± Papilledema
- ± Irregular respiration

*Common causes of the cerebral syndrome seen in practice*

- Cranial trauma
- Hydrocephalus, distemper encephalitis (D)
- Rabies
- Neoplasia (e.g., meningiomas in cats and dogs)
- Gliomas (astrocytomas, oligodendrogliomas in brachycephalic dogs)
- Hepatic encephalopathy
- Feline ischemic encephalopathy

### Multifocal syndrome

In all the preceding syndromes, a single lesion is presumed to account for the clinical signs. However, a situation may arise in which an animal has signs that reflect two or more syndromes, for example, cerebral and lumbosacral syndromes. This indicates a 'multifocal syndrome' (**Table 13**), in which more than one lesion site is present.

Multifocal syndromes are usually seen in animals with infectious diseases of the nervous system. Multifocal syndromes also tend to be the hallmark of the rare, degenerative storage diseases (e.g., gangliosidosis, globoid cell leukodystrophy, etc.), which, in the majority of cases, result from a genetically determined enzyme defect with subsequent accumulation and storage of substrates within various areas of the nervous system.

Another, more common example of a multifocal syndrome is progressive, diffuse hemorrhagic myelomalacia, which can develop secondary to an explosive intervertebral disk extrusion. With this disorder, an initial thoracolumbar syndrome may be followed by a lumbosacral syndrome and then by a cervicothoracic syndrome, as the lesion descends and ascends the spinal cord. In addition, multifocal syndromes are commonly encountered in animals with intoxications (e.g., in tetanus and strychnine poisoning, tetanic spasms usually involve multiple areas of the nervous system).

The presence of constant tremors in animals is also usually indicative of a diffuse disturbance of the CNS. Tremors are typically intensified by voluntary movement. Coarse tremors of the head and body may be first seen in young animals beginning to walk, as a result of congenital/hereditary disorders, such as hypomyelination of the CNS and spongiform encephalopathies. Similar tremors may occur suddenly in young mature dogs, often of small white breeds. Coarse whole body tremors can also be caused by toxins such as hexachlorophene. In conjunction with other neurological signs, tremors in dogs and cats may also be seen in a variety of diseases, including cerebellar disorders, lysosomal storage diseases, metabolic diseases (e.g., hypocalcemia, hypoglycemia, and uremia encephalopathy), and following ingestion of certain toxins such as metaldehyde (snail bait), chlorinated hydrocarbons,

**Table 13**  
**Multifocal syndrome**

*Presence of clinical signs that reflect two or more syndromes*  
*Common causes of the multifocal syndrome seen in practice*

- Distemper (D)
- Granulomatous meningoencephalomyelitis
- Feline infectious peritonitis
- Meningitis
- Mycotic diseases
- Hemorrhagic myelomalacia (D)
- Shaker dog disease
- Organophosphate/carbamate toxicity
- Lead poisoning
- Tetanus
- Strychnine poisoning

**Table 14**  
**Paroxysmal syndrome**

- |   |   |
|---|---|
| <ul style="list-style-type: none"> <li>● Episodic seizures</li> <li>● Episodic sleep</li> <li>● Episodic syncope</li> <li>● Episodic muscle cramping</li> <li>● Episodic weakness</li> <li>● Episodic tetany</li> <li>● Episodic myoclonus</li> </ul> | <p><i>Common causes of the paroxysmal syndrome seen in practice</i></p> <ul style="list-style-type: none"> <li>● Idiopathic epilepsy</li> <li>● Myasthenia gravis</li> <li>● Hypoglycemia (D)</li> <li>● Muscle cramping (D)</li> </ul> |
|---|---|

strychnine, and organophosphate/carbamate compounds. Tremors may also accompany muscle weakness associated with CNS disorders, peripheral neuropathies, or primary myopathies.

### Paroxysmal syndromes

The term 'paroxysmal syndromes' refers to a group of sporadically occurring disorders that usually have no structural lesions within the nervous system. Each paroxysmal syndrome (**Table 14**) tends to manifest distinctive clinical signs and the animal is typically alert and responsive (i.e., without neurological deficits) between episodes. The various syndromes include:

- **Episodic seizures:** The causes of this syndrome include idiopathic epilepsy and insulinoma-induced hypoglycemia. The actual seizure episode is called the ictus. A preictal aura may occur seconds or minutes before the seizure, during which time the animal may appear apprehensive and restless and may seek out the owner or hide and act fearful. The postictal period is the interval during which recovery occurs, often manifested as depression, visual impairment, protracted sleep, or other behavioral disturbances. It may last from an hour to a day or more.
- **Episodic sleep (narcolepsy/cataplexy complex):** In people, episodic sleep, or narcolepsy, is characterized by excessive sleepiness. In dogs and cats, however, the dominant clinical sign is cataplexy, which is characterized by sudden, paroxysmal attacks of flaccid paralysis (muscle atonia) with conservation of consciousness, which may last from a few seconds to more than 20 minutes, and with sudden termination of signs.
- **Episodic syncope:** Syncope, or fainting, is a sudden loss of consciousness resulting from paroxysmal episodes of cerebral deprivation of oxygen or glucose. It is usually associated with cardiopulmonary disease that leads to decreased cerebral perfusion.

- **Episodic muscle cramping:** Episodic muscle cramping, also known as Scotty cramp, and episodic falling in Cavalier King Charles Spaniels are seen in young dogs and are usually precipitated by exercise. The signs of episodic cramping include either a bounding pelvic limb gait, in which the limbs may be abducted and appear stiff, or a bunny-hopping gait, with arching of the spine, and frequent falling and curling into a ball. There is no loss of consciousness.
- **Episodic weakness:** This disorder is usually induced by exercise or excitement (e.g., congenital and acquired forms of myasthenia gravis, hypoglycemia, hyperkalemia, or hypokalemia). It may also occur as an idiopathic condition in Burmese cats in association with head nodding and neck ventroflexion.
- **Episodic tetany:** Episodic tetany is characterized by abrupt onset of intermittent muscle spasms, muscle fasciculations, ataxia, episodic rigidity, and falling. Tonic-clonic spasms of the limbs may be seen in animals with hypocalcemia, sometimes accompanied by nervousness, panting, and pacing.

Rarely, a paroxysmal episode may develop into a prolonged state (e.g., status epilepticus occurring after an episodic seizure attack). Episodic myoclonus has been reported in Labrador Retriever puppies, in which stimulus-sensitive contractions occur in muscles of the face, jaw, trunk, and limbs, resulting in pronounced extensor rigidity. Affected puppies are bright and alert, but remain in lateral recumbency.

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