Cervical hyperesthesia (painful response to a noxious stimulus) is a common presentation in clinical practice. The causes are variable ranging from benign to sinister. More often, than not, these cases have a positive outcome if the underlying etiology can be identified and addressed.

First, it is important to recognize the cervical pain is not spinal cord pain. The neuroparenchyma itself has few nociceptors. Pain originates from the structures outside/adjacent to the spinal cord including the annulus fibrosis of the intervertebral disc, the meninges, the periosteum, the nerve roots (radicular), the muscle and the joints. Recognizing where the source of the hyperesthesia emanates from are important for both pathologic understanding and choice of therapeutics.

Dogs with cervical hyperesthesia may have other neurologic deficits but often do not. This is in contrast to thoracolumbar disease which frequently co-presents with proprioceptive or motor abnormalities. The predominant thought is that it relates to the larger canal/cord ratio in the neck than the back. It also speaks to why cervical disease can be much more severe and refractory to medical management when pain is the sole clinical sign.

Identifying cervical hyperesthesia is often as simple as noting a low head carriage at the walk. These dogs often have muscle fasciculations in the neck and shoulders. In more chronic cases, neurogenic atrophy can be noted in the supraspinatus muscles. Lateral motion of the neck may elicit a pain response but in the author’s experience ventroflexion and lateral palpation are most effective. The dogs may be ataxic in all limbs and postural reaction deficits may also be noted. The thoracic limbs may be spastic/hypertonic with normal reflexes in C1-6 myelopathies that have upper motor neuron disease. The limbs are flaccid/hypotonic with poor reflexes and atrophy in C6-T2 myelopathies with lower motor neuron disease. Dogs with C6-T2 myelopathies may also have poor motor component to their cutaneous trunci and Horner’s syndromes.

Several etiologies implicated in cervical hyperesthesia are to follow. Prioritizing the differential diagnosis list is based on a combination of signalment, acuteness of onset, severity of hyperesthesia and progression. From a diagnostic standpoint, cervical radiographs are often non-specific but indicated. They are useful in ruling out traumatic disease or severe discospondylitis and osteolytic neoplastic disease.

**Intervertebral disc disease (IVDD)**

With a high prevalence in chondrodystrophic breeds, cervical IVDD is nearly as common as thoracolumbar disease. Although survey spinal radiography may reveal changes suggestive of IVDD, these changes are seen in predisposed breeds and are not indicative of causal pathology. Conservative therapy is a combination of supportive care, anti-inflammatory and analgesics. The success rate associated with conservative management of dogs with neck pain only from cervical IVDD is 50–90%, but nearly half of conservatively managed dogs will have a recurrence of clinical signs. Surgical therapy has a >90% success rate and is generally considered a rapid recovery with infrequent complications.

**Atlantoaxial subluxation (AAS)**

AAS is a relatively frequent cause of C1-6 myelopathies in toy breeds of dogs, although and can occur in any age or breed. In suspected cases, care should be taken with manipulation of the neck. Developmental abnormalities of the dens (hypoplasia or aplasia) or malformations of or trauma to the supporting ligaments are responsible for the development of clinical signs, which results in instability of the atlantoaxial joint and contusion and/or compression of the spinal cord.

Radiographs are often diagnostic for AAS and on lateral projection, an increase in the space between the dorsal arch of C1 and the spinous process of C2 (> 4 mm) is noted. That said, advanced imaging is often recommended prior to surgery because of the high co-presence of craniocervical malformations such as caudal occipital malformation syndrome.

Treatment for the condition is most frequently surgical. Conservative management has a 50% success rate and the challenges of external coaptation near the mouth and around the neck. Surgery can be performed by either dorsal or ventral techniques and is most often associated with a positive outcome.

**Cervical spondylomyelopathy (CSM)-“wobblers disease”**

A bimodal disease that affects either young giant breed dogs or older large breed dogs. The giant breed dogs have an osseous stenosis that causes a dorsolateral compression. Large breed dogs, most specifically Dobermans, have a ventral “Disc associated” compressive syndrome. Both of these conditions can be constitutively present or only compressive in dynamic positions (predominantly extension). Survey radiographs are less helpful in these conditions and advanced imaging is indicated. Conservative approaches have been described but this too is often considered a surgical disease with varying reported success rates.
Meningitis/Meningomyelitis
See lecture “CSF-Who, what, when…”

Neoplasia
Any neoplasm that compresses the pain sensitive structures can result in cervical hyperesthesia. There is a high predominance of intradural tumors in the cervical spine including meningiomas and malignant nerve sheath tumors. Soft tissue sarcomas can be causal as can round cell tumors that affect bone such as plasma cell tumors. The tumors can be acute or chronic on presentation based on the affect they have to the vertebral bones. Survey radiographs are considered specific but not sensitive in that over 60% of the bone must be demineralized before radiolucency is noted. Therapies are variable and some tumors may respond to chemotherapeutic (round cell tumors) or radiation therapy (meningioma) while other don’t respond to either ancillary therapy. Surgical therapy is valuable for decompressive and diagnostic purposes but is often not curative in isolation because of the difficulty in attaining complete excision.

Key drugs, dosages and indications
Pharmacologic treatment of the patient with neck pain
Adopted from J Rossmeisl DVM, MS DACVIM (SAIM, Neurology)

<table>
<thead>
<tr>
<th>Tier 1*</th>
<th>Drug choices</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial therapy</td>
<td>Carprofen</td>
<td>2.2 mg/kg PO q 12 hrs</td>
<td>PO, SC</td>
</tr>
<tr>
<td>- Mild/moderate pain</td>
<td>Prednisone</td>
<td>0.5 mg/kg/day</td>
<td>PO</td>
</tr>
<tr>
<td></td>
<td>Acetaminophen/Codeine</td>
<td>Dose to codeine equivalent of 0.5–1 mg/kg q 6 hrs</td>
<td>PO</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tier 2</th>
<th>Drug choices</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Add to Tier 1 agent</td>
<td>Tramadol</td>
<td>2–5 mg/kg PO q 8–12 hrs</td>
<td>PO</td>
</tr>
<tr>
<td>- Moderate, severe, or persistent pain</td>
<td>Methocarbamol</td>
<td>22–44 mg/kg PO q 8 hrs</td>
<td>PO</td>
</tr>
<tr>
<td></td>
<td>Diazepam</td>
<td>0.25–0.5 mg/kg PO q 8 hrs</td>
<td>PO, IV</td>
</tr>
<tr>
<td></td>
<td>Gabapentin</td>
<td>15 mg/kg q 8hrs</td>
<td>PO</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tier 3</th>
<th>Drug choices</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Add to Tier 1 and 2 agents</td>
<td>Morphine</td>
<td>0.25–1.0 mg/kg q 4–6 hrs</td>
<td>IM, SC</td>
</tr>
<tr>
<td>- Medically refractory pain</td>
<td>Oxymorphone</td>
<td>0.1–0.2 mg/kg q 4–6 hrs</td>
<td>IM, IV, SC</td>
</tr>
<tr>
<td>Use as initial therapy for severe pain</td>
<td>Hydromorphone</td>
<td>0.1–0.4 mg/kg q 4–6 hrs</td>
<td>IM, IV, SC</td>
</tr>
</tbody>
</table>

* Drug choices in this tier should not be used in combination.
Cerebrospinal fluid is an ultrafiltrate of blood that bathes the neuroparenchyma. The CSF is created predominantly by the choroid plexi in the ventricles of the brain. The fluid circulates through the ventricular system and into the subarachnoid space around the spinal cord. The CSF is predominantly drained through the arachnoid villi. Because of its proximity to the neuroparenchyma and meninges, cytology can reveal regarding infectious/inflammatory and neoplastic conditions of the central nervous system. CSF changes may also be seen when nerve roots are inflamed even if there is no direct CNS involvement, because the proximal nerve roots are encased by the meninges. The common clinical example of this is polyradiculoneuritis, which can be idiopathic or due to infectious agents such as *Neospora* or *Toxoplasma*.

In cases where meningoencephalitis or –myelitis is thought likely; CSF collection should be considered. CSF collection can be performed in isolation but often follows advanced imaging, specifically MRI. The reason it is often performed after imaging is related to its primary contraindication. Space occupying masses or other causes of elevated intracranial pressure can predispose to brain shift and herniation with CSF acquisition. Further, the process of collecting CSF itself can cause later imaging artifacts. Other contraindications include atlantoaxial subluxation or severe overlying pyoderma.

CSF collection is easier to perform at the cisterna magna and less likely to result in blood contamination. However, the risk of iatrogenic injury is higher. Lumbar CSF acquisition is more challenging and can be performed at the L6-7 space in dogs/ L7-S1 space in cats. There is consideration for increased safety of lumbar punctures in cases of high intracranial pressure as there is more compliance between the sight of puncture and elevated pressure.

With both techniques, CSF should be allowed to drop freely into the collection tubes. CSF needs to be analysed or preserved ideally within 1 hour of collection.

General CSF cytology includes total protein count, nucleated cell count and nucleated cell differential. The total red blood cell count is noted as is general fluid color and presence of foreign material. Other parameters occasionally measured include creatinine kinase and glucose.

CSF cytology is considered a sensitive but non-specific indicator of CNS disease. It is rare that CSF is pathognomonic for any singular condition (<2%). Specific etiologies that could be identified include neoplasms (lymphoma or choroid plexus tumors) or infectious disease (Cryptococcus). More often than not, cell differentials give non-definitive hints of the underlying etiology.

A neutrophilic pleocytosis is most suggestive of bacterial meningitis or steroid responsive meningitis arteritis. Bacterial meningitis is often associated with pyrexia, vomiting, inappetance, severe hyperesthesia and poorly localizing neurologic signs. It is rare and difficult to diagnose as bacteria can contaminate cytology and yet are difficult to culture from the CSF. Ribosomal RNA PCR has been performed to identify bacterial subunits but is rarely performed. Treatment is with antimicrobials that cross the blood brain barrier. SRMA is a more commonly encountered disease most frequently found in Beagles, Boxers, Pointers, Bernese Mountain dogs and Nova Scotia Duck Tolling Retrievers. They frequently present for cervical hyperesthesia and although they may have pyrexia are more frequently systemically normal. As the name implies they are respond well with high doses of immunosuppressive steroids. In cats, a neutrophilic pleocytosis is frequently associate with feline infectious peritonitis.

A lymphocytic/plasmacytic pleocytosis is more suggestive of viral or the meningoencephalitides of unknown etiology (MUE). Distemper, rabies and other viral etiologies should always be considered, particularly in unvaccinated young animals. Serum titers are often coupled with CSF and are often paired with known viruses vaccinated for. Other non-viral infectious agents include rickettsial agents which are typically identified through serology. The MUE is a class of immune mediated diseases that includes the frequently seen granulomatous meningoencephalitis and the lesser seen necrotizing polyoencephalitis and leukoencephalitis. The MUE are frequently encountered in small breed female dogs, though they are seen in a variety of breeds and both sexes. These conditions are more challenging to treat and often involve a combination of steroids in conjunction with other immunomodulators.

An eosinophilic pleocytosis is most suggestive of protozoal, fungal or immune mediated etiologies. Toxoplasmosis and Neosporosis are most easily identified through serologic testing. Fungal agents, with the exception of Cryptococcus which has sensitive and specific serologic testing, are often identified by urine antigen testing.

Albuminocytologic dissociation is a term characterizing an elevation in CSF protein with a normal cell count. A very non-specific finding, this is often seen in cases of Generalized Tremor Syndrome; White Shaker Dog syndrome: These dogs present with diffuse, fine, whole body tremor. Dogs with white hair coats (Maltese terrier, West Highland White terrier) are more commonly affected, however, dogs with other coat colors are also affected. Additional neurological abnormalities include nystagmus, menace response abnormalities, proprioceptive deficits, and seizures. No underlying etiologic cause has been identified but these dogs are also responsive to steroid/immunomodulatory therapy.
Dizzy Now?
Vestibular Syndrome
Fred Wininger, VMD, MS, DACVIM
Veterinary Specialty Services
Manchester, MO

Function of the vestibular system
The vestibular apparatus is a sensory system essential for maintaining posture and balance relative to gravity and movement. The system has its sensory receptors in the inner ear, its processing center in the brainstem, and its output caudally through the spinal cord and rostrally towards the eyes. It receives regulatory input/feedback from the cerebellum. It is clinically divided into peripheral and central components, both because of the ability to separate neurolocalization based on clinical signs and because of the implications these localizations have on differential diagnosis.

Peripheral vestibular anatomy
The membranous labyrinth deep within the inner ear and the vestibular portion of the vestibulocochlear nerve make up the peripheral vestibular system. Within these labyrinths are cavities that contain endolymph fluid. The receptors (crista ampularis) detect the movement of this fluid and in turn detect angular acceleration. Angular acceleration is essentially rotational movement of the head from its resting state. Other receptors (the utricle and saccule; the maculae) have small rocks (otoliths) which give the receptors mass and make them capable of detecting the exerted force of gravity. This is known as linear acceleration. These mechanoreceptors give afferent information along with auditory inputs to the vestibular ganglion and then to the brainstem. The proximity of CN VIII to CNVII is highest within the middle ear and can have clinical consequences. The sympathetic innervation to the face also runs adjacent to these nerves.

Central vestibular anatomy
The four vestibular nuclei are in the most dorsal portion of the medulla, directly under the cerebellum. This proximity manifests clinically in that diseases causing herniation of the cerebellum though the foramen magnum can compress these cell bodies. There is a direct connection between these nuclei and the “vestibulocerebellum” made up of the flocculonodular lobe. The primary outputs of the vestibular system are to the extraocular muscles of the eyes and caudally through the spinal cord. The medial longitudinal fasciculus sends outputs to CN III, IV and VI, maintaining the globe’s position in the orbit and the ability to maintain gaze in the face of head rotation. The outputs to the spinal cord are through the vestibulospinal system, essential an anti-gravity tract facilitating mostly extensor muscles to the neck, trunk and limbs.

Clinical signs of vestibular disease
Certain clinical signs are inherent to any form of vestibular disease and are in some cases pathognomonic for the neurolocalization. They include

1. Head tilt
2. Nystagmus (resting or positional)
3. Vestibular Ataxia
4. Ventrolateral strabismus

These clinical signs emerge because of imbalance of vestibular input. In the normal patient, the vestibular system is bilaterally providing input that when processed together yields the precision system of balance. When one side is diseased and fails to provide normal input, the non-diseased side produces unilateral information and thus clinical signs often reflect the non-diseased side rather than the diseased side. Many of the clinical signs of vestibular disease would be absent in the animal affected bilaterally. The precedent for this is seen mostly in cats with bilateral otitis media/interna. They have no nystagmus, head tilt or ataxia. Clinical signs in these patients include snake like movements of the head and a lack of normal vestibulocular function best assessed by evaluating the physiologic nystagmus (vestibulo-ocular reflex).

Differentiating central vs. vestibular disease
As previously stated, differentiating these diseases are essential for differential diagnosis creation and prognostication. The differences are inherent to the adjacent structures that would be affected. The peripheral component is in close proximity to CNVII and the sympathetic nerve to the face. Thus facial paresis and Horner’s syndrome are commonly seen with this neurolocalization. Central disease is in proximity to the sensory proprioceptive systems, the arousal centers and other cranial nerves. Therefore, proprioceptive deficits, mental dullness and cranial nerve dysfunction (other than CNVII) are common with this localization. Although simple in theory, distinction of central vs. peripheral disease can be difficult in practice as the clinical signs are rarely clear and completely present. Should the vestibular cerebellum become diseased, these signs would point to a central vestibular localization, but often with conflicting sign sidedness. This is known as paradoxical vestibular disease. Postural reaction deficits are used to determine sidedness, as they are consistently ipsilateral to the lesion.
<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Peripheral</th>
<th>Central</th>
<th>Paradoxical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head tilt or circling</td>
<td>Toward lesion</td>
<td>Toward lesion</td>
<td>Away from lesion</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Horizontal or Rotary, Non-positional</td>
<td>Horizontal, rotary or vertical, +/- positional</td>
<td>Horizontal, rotary or vertical, +/- positional</td>
</tr>
<tr>
<td>Mentation</td>
<td>Alert</td>
<td>+/- Mentally inappropriate.</td>
<td>+/- Mentally inappropriate.</td>
</tr>
<tr>
<td>CP Deficits</td>
<td>No</td>
<td>Ipsilateral</td>
<td>Ipsilateral</td>
</tr>
<tr>
<td>CN deficits</td>
<td>+/- CN VII</td>
<td>+/- CNs V-XII</td>
<td>+/- CNs V-XII</td>
</tr>
<tr>
<td>Horner's syndrome</td>
<td>+/- Ipsilateral</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

**Differential diagnosis of vestibular disease**

Creation of a differential diagnosis list is best approached by using the VITAMIN D scheme. Common differentials for peripheral disease include infectious (otitis media/interna) and idiopathic, commonly seen in the geriatric patient. More common central vestibular differentials include neoplasia, infectious/inflammatory (meningoencephalitis), vascular, and toxic (metronidazole toxicosis).

Cross sectional imaging is a mainstay of diagnosing the cause of vestibular disease. MRI has supplanted CT as the diagnostic modality of choice. CT can adequately identify disease of the peripheral system and most specifically tympanic bulla changes. However, beam hardening artifact limits the modality in evaluating the caudal fossa (central vestibular system). MRI can image the entire system with superior contrast resolving ability. Other ancillary diagnostics may include myringotomy/culture and brain auditory evoked potentials to evaluated the auditory component of CN VIII. Treatment and prognosis are largely contingent on the etiology itself.
I’ve Fallen and I Can’t Get Up: The Acute Lower Motor Neuron Patient
Fred Wininger, VMD, MS, DACVIM
Veterinary Specialty Services
Manchester, MO

Cases of acute lower motor neuron disease present diagnostic challenges, but when managed correctly can have rewarding results. The cases can often be diagnosed by tests clinically available to the savvy general practitioner without need for referral.

The lower motor neuron unit is comprised of four parts:
1. The motor neuron cell body or soma
2. The neuron/axon
3. The neuromuscular junction
4. The muscle.

These four entities are lumped together clinically as they serve as a functional unit, and disease of one part appears similar to disease in another. Though most frequently evaluated in the appendicular skeleton (nerve and muscles of the limbs), it is important to remember that the motor unit is located and can be clinically investigated in other places, namely the face/head, neck, trunk and perineal region. Many of the cranial nerves have lower motor neuron function (ie, cranial nerve VII-the facial nerve innervates the somatic muscles of facial expression).

Classic lower motor neuron signs include decreased tone, decreased reflexes and neurogenic atrophy with is considered acute and severe. This is distinguished from disuse atrophy which takes longer, is less severe, and frequently affects muscle groups of different innervation. In most cases of lower motor neuron disease, sensory function remains intact, but certain disease processes can affect both motor and sensory systems. Many of the lower motor neuron diseases also affect the autonomic system, or more specifically the parasympathetic system. This is not surprising as they share a common neurotransmitter, acetylcholine. The difference lies in their receptors, with the neuromuscular junction comprised of nicotinic receptors and the parasympathetic post-ganglionic synapse being comprised of muscarinic receptors.

Lower motor neuron disease can affect a single group of motor units or be generalized. It can also be classified as acute or chronic. Chronic LMN disease can be caused by a myriad of etiologies and is best evaluated using the VITAMIN D differential approach. Fortunately, acute LMN disease has a shorter list of differentials. They include idiopathic polyradiculoneuritis, myasthenia gravis, botulism and tick paralysis. Acute LMN paralysis is often associated with a good prognosis with early diagnosis and the capacity for excellent supportive care when the patients reach their highest level of affectedness.

Acute myasthenia gravis (MG)
Myasthenia gravis typically causes exercise-induced weakness and can present similarly to the other acute LMN diseases. The primary difference is that in cases of MG, spinal reflexes tend to be preserved. MG often causes facial muscle weakness and depressed palpebral reflexes. In cats, ventro-flexion of the head is a frequent clinical sign. Megaesophagus is particularly prominent in this disease with regurgitation as a secondary consequence.

The gold standard for diagnosis of MG is serologic identification of antibodies against the acetylcholine receptor. The antibodies cause the junctionopathy and this test is extremely sensitive and specific. False negatives are rare and are associated with lack of circulating antibodies or rare forms of the disease. Edrophonium (tensilon) testing is a more immediate means of diagnosing MG. However this test is non-specific with other acute LMN diseases, which can also improve with this anticholinesterase therapy. For this reason results should be validated with the gold standard. At the time of this proceeding Edrophonium is no longer commercially available. Electrodiagnostic testing is available for MG (supramaximal repetitive stimulation and single fiber EMG), but is often not performed because of the serologic test and because the tests require anesthesia which can exacerbate the disease. Chest radiographs are a good ancillary study to evaluate for megaesophagus, aspiration pneumonia, and a cranial mediastinal mass, which is rarely reported as a causative feature.

Treatment is generally a longer acting anti-cholinesterase and supportive care. Cholinesterases are available in oral, and IV forms for cases with significant regurgitation. Immune modulation is often used but caution must be exercised in the face of potential pneumonia. Prednisone and Mycophenolate Mofetil are often used because of their effectiveness and quick onset of action. The prognosis is good if the dogs do not succumb to aspiration pneumonia.

Idiopathic polyradiculoneuritis (IPRN)
The disease is often referred to as Coonhound paralysis because the disease has been associated with an immune-mediated reaction to antigen present in the saliva of raccoons. However, most reported cases are not associated with animals that had any exposure to raccoons. Polyradiculoneuritis is considered the counterpart of Guillian-Barré syndrome (GBS) in people, a similar immune mediated neuropathy that causes paresis and hyperesthesia/allodynia.
In this disease, the ventral nerve root become inflamed through immune mediated attack and a severe lower motor neuron clinical picture ensues, usually in an ascending pattern. We have seen multiple cases that present more spastic/upper motor neuron initially but quickly become lower motor neuron in 3-4 days after the onset of signs. One clinically unique feature compared to other causes of acute LMN is hyperesthesia of the distal limbs. Often these animal will react to light touch with an exaggerated pain response, but this sign is only present in some cases. The hyperesthesia may be due to inflammatory affects on the adjacent sensory dorsal nerve root. Rarely, these dogs will develop cranial nerve dysfunction as well with facial paresis and decreased gag reflexes reported.

Diagnosis of is made on clinical impression coupled with cerebrospinal fluid analysis. Of the acute LMN diseases, this is the only one that will cause CSF changes, most commonly albuminocytologic dissociation (elevated protein with a normal cell count). The reason is that this disease affects the ventral nerve root which is intradural and bathed by the CSF. A more definitive diagnosis can be made by an F-Wave electrodiagnostic study performed by a veterinary neurologist, which evaluates nerve function of the proximal nerve segment.

Dogs will often recover within 3-5 weeks with physical rehabilitation and supportive care. Similar to GBS syndrome in people, corticosteroid treatment appears not to influence recovery rate. That said, some dogs do not recover, either because of too severe a clinical onset or incomplete disease resolution. There is evidence that intravenous immunoglobulin (IVIG) and plasmapheresis can shorten the duration of clinical disease. Unfortunately, these treatments are often cost limiting.

**Botulism**

Botulism is a disease caused by toxins produced by Clostridium botulinum. These bacteria are anaerobic, Gram-positive, and spore-forming. Several subtypes have been identified, based on the presynaptic protein they effect. All canine cases to date have been caused by toxin type C. Ingestion of the preformed toxin is known as food-borne-botulism and is the most common form of intoxication. Bacteria residing in a wound or in the gut can produce botulinum toxin, but this method of intoxication is not seen in the domestic animal. The toxin’s pathology is based on disruption of pre-synaptic proteins that prevent the exocytosis of acetylcholine into the neuromuscular junction. Botulism is difficult to diagnose, with the gold standard being the mouse bioassay. Ruling out the other acute LMN diseases makes the clinical diagnosis. Electrodiagnostic testing of dogs with botulism is poorly specific and usually not useful. Botulism is self-limiting and many animals will recover within three weeks. Treatment is supportive and spontaneous recovery will occur in many dogs if the amount of ingested toxin is low. If dogs succumb to the disease it is usually because of respiratory failure or aspiration pneumonia. The level of autonomic affectedness likely plays a role in prognosis as well. Antitoxin is generally contraindicated as the toxin is already internalized and bound. The prognosis is generally good with supportive care.

**Tick paralysis**

Tick paralysis is a common cause of acute LMN disease in warmer climates. The cause is a neurotoxin from an adult dermacentor tick in the US, which in Australia the nymph form of ixodes are more commonly described. The neurotoxin disrupts calcium channels at the presynaptic membrane that prevent exocytosis of acetylcholine into the synaptic cleft. The site of the tick bite seems to dictate the severity of clinical signs, with neck and trunk bites being more severe than those on the appendages. Clinical signs are usually only somatic in nature. Diagnosis is made by the identification of a single large female tick or response to quick acting acaracids. Dogs respond to tick removal quickly with resolution within 24 hours. The clinical signs are much more severe in Australian cases than those in the US.

<table>
<thead>
<tr>
<th>Weakness/Gait</th>
<th>Myasthenia Gravis</th>
<th>Idiopathic Polyradiculo-neuritis</th>
<th>Tick Paralysis</th>
<th>Botulism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Segmental Reflexes</td>
<td>WNL, Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Cranial Nerve Involvement</td>
<td>Dysphonia, Megaesophagus, CN VII</td>
<td>Dysphonia, mild CN VII</td>
<td>Dysphonia</td>
<td>Dysphonia, Megaesophagus, Autonomic</td>
</tr>
<tr>
<td>Condition</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Hyperesthesia</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Big Mama Tick</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSF</td>
<td>WNL</td>
<td>Increased Protein</td>
<td>WNL</td>
<td>WNL</td>
</tr>
</tbody>
</table>
MRI- What’s the Big Deal?
Fred Wininger, VMD, MS, DACVIM
Veterinary Specialty Services
Manchester, MO

Imaging the nervous system is a difficult task because of its residence in the hard bone of the skull and vertebral column, as well as its opaque parenchyma and minimal variation in tissue density. The introduction of computerized tomography (CT) and then magnetic resonance imaging (MRI) revolutionized veterinary neurology because of the ability to detect structural disease despite these obstacles. Cross sectional imaging has the advantage of improved resolution over 2-dimensional imaging by eliminating summation and permitting partial thickness intra-cranial reconstruction. Further, techniques that utilize protons rather than density take advantage of the more robust chemical differences present in neuroparenchyma. CT first and now MRI are the gold standard in structural imaging of the brain and spinal cord. That said, each technique has indications for different presentations and choosing the correct modality can mean the difference in arriving at a successful diagnosis.

Although cross sectional imaging has changed veterinary neurology for the better with improved diagnostic power, it is important to remember structural assessments of the nervous system cannot replace the functional evaluation. The primary functional assessment of the nervous system is a thorough physical and neurologic examination. Other functional assessments include electrophysiological techniques (EMG, EEG, ERG, BAER) and newer hemodynamic techniques (fMRI and Infrared spectroscopy). Often structural disease is not accompanied with functional disorders (ie. Chiari malformations or hydrocephalus). The inverse is also true in that functional disease may not have a structural correlate (idiopathic epilepsy). For this reason these functional and structural assessments complement each other but cannot be evaluated in isolation.

The term tomogram refers to any cross sectional modality. These techniques include CT and MRI, but also ultrasound and advanced metabolic imaging techniques such as SPECT or ultrasound.

CT principles
CT uses the simple techniques of 2D radiography and through complex computations is able to reconstruct these images in cross sectional and 3D studies. Images are acquired by rapid rotation of an x-ray tube 360 degree around a patient. The transmitted radiation is then measured by a ring of radiation sensitive detectors on the opposite side of the subject. An image is generated from these measurements on the premise that the density of the structures within the cylinder can be calculated when evaluated in combination. Complex mathematical algorithms allow each voxel to be assigned a numerical value based on the level of attenuation occurring within that volume of tissue. These units are known as Hounsfield units (HU).

Hounsfield units range from -1000 for air and +3095 for the densest object than can be imaged by CT (compact bone). Soft tissues of clinical interest generally range from -100 to +100. In neuroparenchyma, the grey matter is +35HU and the white matter +45HU, explaining why the technique has difficulty discerning the contrast of these structures. However, adjacent structures such as the ventricles can be easily imaged (HU=0).

The human eye can perceive 32 shades of grey. Therefore, for highest contrast resolution the goal is to maximize those shades to evaluate the tissue of interest. Window level defines the middle of the tissue of interest. Window width describes the range of HU displayed. The larger the window, the more HU represented by each shade of grey, the lesser the contrast. Soft tissue windows are smaller because the range of densities is lower when compared to bone. In other words, a shade of grey in a soft tissue window may represent 3 different HU, while in bone a shade may represent 100HU or more.

Contrast resolution is defined as the ability to display an image of an object that differs only slightly in attenuation compared to its surrounds. CT has 10x better contrast resolution over radiographs. However, the contrast resolution of CT for discerning neuroanatomical structures from each other and disease is poor.

MRI principles
MRI is different from other imaging modalities in that it utilizes protons (predominantly water content) to create images rather than density. This is particularly of value in the brain and spinal cord, where density differences are minor compared to differences in water content.

At rest in the normal environment, the protons of the body are moving towards entropy in a random arrangement. MRIs created large magnetic fields, which cause these protons to align along an axis. Though aligned, these protons are still rotating around their aligned axis, a process called precession. The speed at which they precess is the same for all protons of the same valence as defined by the larmor equation. Though precessing at the same speed, the protons are all out of phase with each other. When a radiofrequency wave is applied to the protons, they are jarred out of alignment together and all develop the same phase. With time, they return to their resting state in the magnetic field. The speed at which this return to rest is dependent on the chemical characteristics in which the proton resides and can be measured.

- The speed at which the proton returns to its resting axis is known as either longitudinal, spin-lattice or T1 relaxation.
- The speed at which the proton comes out of phase with the adjacent protons around it is known as transverse, spin-spin or T2 relaxation.

The ability to weight the images on these and other proton characteristics allows for interrogation of the nervous system, to make the tissue or lesion of interest more conspicuous.

**CT indications compared to MRI**

Although MRI has become the gold standard in neuro-imaging, there are several scenarios in which CT is indicated and perhaps preferable. CT has greater spatial resolving abilities when imaging bone. Because of this and its ability to create very thin slices, fractures can be easily identified. 3D reconstructions are superior with CT and allow for more global imaging of the bone as well as aid in surgical planning. CT is also excellent at imaging hemorrhage, which will appear as hyper-attenuating 3 hours after a bleeding event and remain so for 3 weeks as the HU is directly proportional to the PCV of the bleed. CT is inherently less expensive to purchase and maintain which translates to less expense and greater availability to the client. Although MRI has become quite fast, acquisition times are shorter with CT which is essential for critical patients or those that can not tolerate anesthesia. CT is less susceptible to metal artifacts compared to MRI, which can be important in projectile/shrapnel wounds.

CT has some inherent deficiencies aside from its poor contrast resolution. CT requires relatively large doses of ionizing radiation; Not frequently a concern in veterinary medicine. CT is poor at imaging the caudal aspect of the skull/brainstem because of beam hardening artifacts. In this case, the low energy photons do not pass through the petrous temporal bone proportionally to the high-energy photons and the computer gets conflicting information casting dark streaks across the brainstem.

**Low field vs. high field MRI**

Magnets what produce a field 1T or greater are generally considered high field, with low fields being anywhere between 0.2-0.5T. High field MRI have a superconducting magnet, which requires a cryogen to keep the system cooled and maintain low electrical resistance in the coil. These magnets requires special housing systems both to eliminate radio interference but also for protection from their attractive properties. This size restriction and cost of maintenance limits their availability. Low field MRIs are less costly and cheaper to maintain.

The main advantage to High field MRIs over their low field counterparts is the time required for image acquisition. The high magnetic field promotes faster proton relaxation and therefore multiple signals and averages are acquired in minutes. High field MRI images have more information and higher resolution with smaller voxels. Theoretically, low field MRI could create similar quality diagnostic images, but with acquisition times nearing hours to days! There have been few studies in veterinary medicine comparing these different fields, but the human field literature demonstrated the poor sensitivity and specificity of low field MR over two decades ago and it is rarely used for neural imaging. However, low field imaging maintains a role in extremity imaging for humans, as it does not require a closed bore system. (An advantage for the claustrophobic patient). This advantage does not translate to the anesthetized veterinary patient.

CT and MRI have their own inherent advantages and struggles. Our experience is that they often work best in tandem or when one modality fails. Future imaging techniques with MRI and PET are embracing the idea of combined structural and functional imaging.
Neurolocalization is the ultimate goal of the neurologic examination, piecing the puzzle of reflexes and responses together to an anatomic segment. Once accomplished, the practitioner is able to assign a rank list of differential diagnosis for this neuroanatomic site based on the patient’s age, breed, clinical onset/progression and the presence of spinal hyperesthesia. Whereas this list can easily be found in any neurology text, the ability to interpret findings and combine them to fit a single lesion site requires practice.

A concept integral in localization is that of the Upper Motor Neuron (UMN) and Lower Motor Neuron (LMN). The Upper Motor Neurons are essentially the long tracts from the brain that instruct the lower motor neurons what to do. The LMN consists of the motor neuron cell body within the spinal cord, the nerve itself, the neuromuscular junction and the muscle itself. In cases of UMN disease, the lower motor neuron works without instruction, known as a loss of inhibition. It is classically hypertonic, with normal to increased reflexes and minimal atrophy. In cases of LMN disease, the upper motor neuron is irrelevant, because the LMN cannot respond to its instruction. Thus hypotonicity, decreased reflexes and severe neurogenic atrophy are noted.

The spinal cord can be divided into four areas. The LMN of the limbs are located in swellings of the spinal cord known as intumescences. When these intumescences are the sight of disease, LMN signs are notable. When the lesion blocks the long tracts between the limb the brain and the LMN, UMN signs emerge. Thus the spinal cord can be divided into the following segments.

<table>
<thead>
<tr>
<th>Lesion location</th>
<th>Thoracic limbs</th>
<th>Pelvic limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1-C5</td>
<td>UMN</td>
<td>UMN</td>
</tr>
<tr>
<td>C6-T2</td>
<td>LMN</td>
<td>UMN</td>
</tr>
<tr>
<td>T3-L3</td>
<td>Normal</td>
<td>UMN</td>
</tr>
<tr>
<td>L4-S3</td>
<td>Normal</td>
<td>LMN</td>
</tr>
</tbody>
</table>

The neurologic examination is comprised of multiple parts for accurate localization.

**Gait**

Ataxia is defined as an uncoordinated gait and is frequently classified as “the drunken sailor walk”. It is often classified as

1. Proprioceptive/spinal-a.k.a The true drunken sailor walk, often coupled with a spastic/long strided gait suggesting UMN dysfunction. Patients are often thought of as overstepping or “floating”
2. Vestibular-Characterised by imbalance often manifest as “wall walking, coupled with a head tilt and nystagmus
3. Cerebellar lesions –Characterized by hyper/dysmetria (Goose stepping), truncal swaying, and intention tremors.

Paresis is weakness of the gait, reduced voluntary movement, whereas paralysis is complete loss of voluntary movement. Both paresis and paralysis ( -plegia) can be used to describe the deficits in only one limb (monoparesis/plegia), in the pelvic limbs (paraparesis/plegia), in all four limbs (tetraparesis/plegia) or on one side of the body (hemiparesis/plegia).

**Proprioception**

The ability of a patient to identify the location of its limbs in space. A subjective but integral component to the examination, these tests confirm the presence of a neurological disorder and can detect subtle dysfunction, helping identify which limbs are affected. This includes paw position, hopping and placing responses.

**Spinal reflexes**

Reflexes are quite different than responses, such as proprioception, in that they do not require forebrain input. This is an important distinction

The easiest and most reliable are the patellar reflex in the pelvic limbs and the withdrawals in both the pelvic and the thoracic limbs. Don’t forget to examine the tail and anus (perineal reflex). Reduced reflexes in a limb identify a LMN lesion in that limb, whilst normal or increased reflexes localize the lesion to the UMN

Cutaneous trunci reflex (panniculus)--helps narrow down lesion localization in the thoracolumbar region. After pinching the skin, the sensory information enters the spinal cord approximately two vertebral spaces cranially, ascends the spinal cord to the level of C8-T1 where bilateral synapse occurs with the motor neurons of the lateral thoracic nerve, which then course through the brachial plexus and innervate the cutaneous trunci muscle, resulting in bilateral contraction of these muscles. Normally, this reflex is present from T2 to about L4-L5 and a cut-off in this region suggests a spinal cord lesion just cranial to the cut-off level. Loss of the cutaneous trunci reflex can also be due to a brachial plexus lesion, in which case it will be completely absent on the side of the lesion and normal on the contralateral side.
**Pain sensation**

The spinal pathways that carry pain sensation are located deep in the spinal cord so only a severe lesion will impair pain perception (making this an important prognostic factor). For conscious perception of pain, manifested by vocalization and/or turning the head and trying to bite, the information needs to be recognized by the sensory nerve, travel up the entire spinal cord cranial to that area and be interpreted by the brain. It is important to differentiate a pain response from a local withdrawal reflex (which should be present if both the peripheral nerve and spinal cord segment of the stimulated peripheral nerve are intact), in which case the limb will be retracted but no signs of conscious awareness of the pain will be evident. Pain sensation is tested by applying heavy pressure with haemostats to the bones of the digits (don't forget to test different digits) or to the long bones of the limbs.

**Spinal palpation**

Looking for areas of hyperesthesia or deformities. Pressure is applied to the spinous and transverse processes of the vertebrae in all spinal segments. Manipulation of the cervical spine in all directions is performed.

**References**

Seizure Disorders are extremely common in dogs with an incidence estimated as high as one percent of the pet population. The cause of these seizures is generally classified as structural, metabolic or idiopathic/heritable. Idiopathic epilepsy is by far and away the most common cause, representing 30% of all epileptics. Despite large advances in anatomic imaging, genetic characterization and surgical care, the understanding of the cause of idiopathic epilepsy is still in its infancy. However, many novel anti-seizure medications are entering the clinical arena with potential use in patients unresponsive to traditional medications such as phenobarbital and bromide.

Managing seizure disorders presents a major challenge to the veterinarian, especially when a dog does not respond to standard (i.e., phenobarbital, bromide) therapy. Such refractory cases account for between 25–30% of all epileptics. It is very important for the clinician to inform the pet owner that most epileptic dogs do not reach seizure-free status; success is typically considered a reduction in the frequency and duration of seizures. Nonetheless, the goal of anticonvulsant therapy should be to eliminate seizure activity in the patient, or come as close to this goal as possible, without subjecting the patient to unacceptable side effects of drug therapy or the client to unreasonable financial burden.

**Phenobarbital**

**MOA**
- Primary mechanism of action is by decreasing seizure onset via enhanced GABA activated chloride conductance
- Secondary mechanism of action is by decreasing seizure spread via reduced current through calcium channels and reduce glutamate-mediated excitation

**T ½**
- 24-40 hours

**Metabolism/Excretion**
- Majority metabolized by the liver, with 1/3 excreted unchanged in the
- Phenobarbital will induce hepatic microsomal enzymes (p450 enzymes) and it can be expected that elimination half-lives will decrease with time with concomitant reductions in serum levels

**Side Effects**
- Behavioral: hyperexcitability, restlessness, sedation. Normally this is seen for the first few weeks of treatment
- Immune mediated neutropenia or thrombocytopenia or anemia (these reversible blood dyscrasia occurs within the first 6 months of dosing)
- Idiosyncratic hepatic reactions: RARE. Evidenced by a rapid elevation in ALT and abnormal bile acids – phenobarbital should be stopped immediately and another AED should be loaded and started
- With chronic dosing, PU/PD is common and psychogenic polydipsia may develop. The most common serum biochemical changes include elevated alkaline phosphatase

**Dose**
- 2.5mg/kg PO BID as a starting dose, with all future adjustments based on serum drug concentrations in conjunction with clinical assessment

**Blood levels**
- Serial serum phenobarbital concentrations should be evaluated at 14, 45, 90, 180 and 360 days, and 60 days thereafter
- Therapeutic range: 20-40mg/dl

**Bromide**

- **MOA**
  - Primary mechanism of action is by decreasing seizure onset via enhanced GABA activated chloride conductance
- **T ½**
  - 20-46 days
- **Metabolism/Excretion**
  - Bromides are principally excreted by the kidneys
- **Side Effects**
  - PU/PD, lethargy and mild ataxia
  - Pancreatitis and gastrointestinal intolerance have been reported but are infrequent
- **Dose**
  - 40-60mg/kg PO SID
- **Blood levels**
Zonisamide
- **MOA**
  - Primary mechanism of action is by decreasing seizure spread via reduced current through calcium channels
  - Secondary mechanism of action is by decreasing the seizure onset via enhanced sodium channel inactivation
- **T1/2**
  - 15-20 hours
- **Metabolism/Excretion**
  - Most of the drug is excreted via the kidneys into the urine, but about 20% is metabolized, primarily in the liver
- **Side Effects**
  - Sedation, dry eye, ataxia, inappetence and vomiting – patients with a history of sulfonamide hypersensitivity should NOT be prescribed this medication
  - Metabolic acidosis and liver dysfunction has been reported in dogs
- **Dose**
  - 5-10mg/kg PO BID
- **Blood Levels**
  - Currently, zonisamide levels are able to be evaluated at Auburn University. This is a new medication and at this time we aim to obtain a blood level close to 20ug/ml (10-40ug/ml). It has been reported that stable plasma concentrations are achieved within 3-4 days with oral administration of zonisamide

Levetiracetam (Keppra)
- **MOA**
  - Binding with a specific synaptic vesicle protein (SV2A) in the brain.
  - No directly affect common neurotransmitter pathways (e.g., GABA, NMDA) or ion channels (e.g., sodium, T-type calcium).
- **T ½**
  - 3–4 hours
- **Metabolism/Excretion**
  - 70% excreted in urine
  - no hepatic metabolism
- **Side Effects**
  - Lethal dose is 100 times the recommended dose. Dosing can be increased several fold in attempts to increase efficacy.
  - No side effects of note
- **Dose**
  - 20 mg/kg PO TID
- **Blood levels**
  - Not typically performed because dose-efficacy ratio not direct.
- **Other notes**
  - neuroprotective properties, and may ameliorate seizure-induced brain damage.
  - Prevents further seizures (anti-kindling)
  - Injectable-Possible emergency drug for SE
  - Honeymoon effect
  - More effective in cats with limited side effects and no noted honeymoon effect

Felbamate
- **MOA**
  - Positive modulator of GABA_A receptors
  - Possible NMDA antagonist of the MR2B subunit
- **T ½**
  - 5-6 hours
- **Metabolism/Excretion**
  - 70% excreted in urine
  - Remainder hepatic metabolism
- **Side Effects**
  - No sedation
  - Possible hepatotoxicity
- **Blood dyscrasia and KCS**
  - **Dose**
    - 15-20 mg/kg PO TID with dose escalation permissible
  - **Blood levels**
    - The author has not performed drug levels of Felbamate
  - **Other notes**
    - Expensive

### Gabapentin
- **MOA**
  - binding to the α2δ subunit of voltage-gated neuronal calcium channels. This binding decreases intracellular calcium influx, leading to decreased synaptic release of excitatory neurotransmitters.
- **T ½**
  - 3-4 hours
- **Metabolism/Excretion**
  - **Urine excretion**
  - **Hepatic metabolism**
  - 30–40% of the orally administered dose of gabapentin undergoes hepatic metabolism to N-methyl-gabapentin
- **Side Effects**
  - **Dose**
    - 15–60 mg/kg PO TID or QID
  - **Blood levels**
    - Suspected blood levels are 4–16 mg/L. As this drug has questionable AED efficacy, I rarely have blood levels checked.
  - In recent years, the efficacy of gabapentin as an anti-convulsant has come into question, particularly with the other previously mentioned tertiary agents.

### Pregabalin (Lyrica)
- **MOA**: Same as gabapentin
- **T1/2**
  - 7 hours (11 hours in cats)
  - The half-life of elimination of pregabalin in dogs is about
- **Metabolism/Excretion**
  - **Dose**
    - 2-4mg/kg PO BID

### Topiramate
- **MOA**
  - Mechanisms of action include
    - decreasing seizure onset via both enhanced sodium channel inactivation and enhanced GABA activated chloride conductance
    - decreasing seizure spread via reduced glutamate-mediated excitation
- **T1/2**
  - 2-4 hours
- **Metabolism/Excretion**
  - Low rate of hepatic metabolism
  - Both unchanged topiramate and its metabolites are excreted mainly by the kidneys
- **Side Effects**
  - Sedation and weight loss
  - In recent studies elevations in liver enzymes were appreciated, however, these patients were also receiving phenobarbital (in conjunction with the topiramate) so it is unknown if the liver elevations were secondary to the phenobarbital or the topiramate
- **Dose**
  - 5-10mg/kg PO BID
- **Blood Levels**
  - Not established at this time
Animals with neurological disease often present for problems walking, moving, and standing. This discussion will focus upon the clinical diagnosis of these abnormalities in dogs and cats.

**Neuroanatomy of gait**

Gait is commonly defined as a regularly repeating series of leg movements during walking or running. The nervous system controls the actions of the muscles, bones, joints and associated connective tissue important for walking. Normal walking is produced by the recruitment of stepping reflexes, which alternate between the extensor and flexor muscles. The vestibulospinal and reticulospinal tracts are facilitatory to the extensor muscles, important for maintenance of the body tone against gravity (the stance or propulsive phase). The corticospinal and the rubrospinal tracts are facilitatory to the flexor muscles, important for the protraction (flight) phase of limb movement.

Locomotion is thought to be controlled at the level of the brain stem; however, a discrete anatomic gait center (nucleus) has not been identified. Supratentorial (forebrain) structures are important for voluntary initiation of movement. The cerebellum, while not necessary for the initiation of movement, is important for coordination of movement. Cerebellar influences coordinate and smooth body movements by controlling rate, range, and force of limb motion. The cerebellum helps to "smooth" movements.

**Clinical evaluation of abnormalities of gait**

Clinical evaluation of gait usually involves observation of the animal's movements during walking, and, when indicated, running. This is best accomplished by having a handler walk the animal over a flat, non-slippery area (such as concrete or carpet). An overall assessment is made of how the animal moves and clues are obtained to abnormalities present.

**General abnormalities of gait**

**Ataxia**

Ataxia literally means lack of an axis, and is sometimes described as incoordination. Ataxia can result from a variety of anatomic lesions within the nervous system, most commonly of the cerebellum, vestibular system and spinal cord sensory pathways. Ataxia, without motor involvement (paresis), usually implies cerebellar or cerebellar pathway disease. Sensory ataxia due to loss of joint position sense is often made worse by blindfolding the animal, effectively preventing visual compensatory mechanisms.

**Dysmetria**

Dysmetria is improper estimation of distance during muscular activity. Dysmetria includes both hypometric and hypermetric. With hypermetria, voluntary muscular movement overreaches the intended goal; with hypometric, voluntary movement falls short of the intended goal. Hypermetria is more commonly recognized than hypometria. Both of these abnormalities are most often associated with lesions of the cerebellum or cerebellar pathways. In the instance of hypermetria, for example, the loss of cerebellar input, which normally stops the flexion phase of gait, results in the exaggerated movement.

**Spasticity**

Spasticity is a state of increased muscle tone and commonly results from upper motor neuron (UMN) lesions. Spasticity is observed in the gait as a lack of normal flexion or floating (failure to adequately flex the limbs during gait).

**Stiffness**

Stiffness associated with decreased step length is commonly seen with diseases of the peripheral neuromuscular apparatus [lower motor neuron (LMN) cell body, nerve roots, peripheral nerve, neuromuscular junction and muscle]. Dogs with neuromuscular disease may also have a stiff, stilted, choppy gait due primarily to muscle weakness. These abnormalities may be episodic and occur, as in the case of myasthenia gravis, as the level of exercise increases. A similar appearance may occur in dogs with pain, primarily from musculoskeletal disease.

**Paresis**

Paresis is derived from the Greek word for relaxation and suggests neurological weakness without complete paralysis (implies that some voluntary motion remains). Varying degrees of paresis can occur with some animals retaining the ability to walk while others are unable to support their own weight and stand. Paresis may be observed at gait as dragging of the toes or feet. Abnormal toenail wear may suggest underlying paresis.

Paresis at gait first occurs with lesions in the midbrain caudal to the level of the red nucleus. The severity of the gait impairment increases as the lesion occurs progressively more caudally in the central nervous system. For example, supratentorial lesions may
result in significant hemiparesis when postural reactions are tested; however, gait remains relatively normal. With brain stem or spinal cord lesions, the associated paresis usually results in obvious gait impairment.

**Lameness**

Lameness [decreased or non-weight bearing on a limb(s)] is usually associated with pain of the limb from musculoskeletal disease. A similar abnormality can also occur with nervous system dysfunction (and presumably pain), referred to as nerve root signature. This abnormality often occurs in a single thoracic limb due to cervical spinal compressive disorders (intervertebral disk extrusion). The same phenomenon may affect a pelvic limb. Often, the affected limb may appear painful upon manipulation, mimicking an orthopedic problem.

**Clinical assessment**

Important clues as to the cause of the gait abnormality can be obtained by assessing the step distance (i.e., the distance between where both thoracic or both pelvic limbs are placed in relation to each other). This determination is best made when viewing the animal from a lateral direction and when the animal is perpendicular to the examiner's line of sight.

Generally, animals with UMN neurological disease have normal or increased step lengths, while dogs with orthopedic or neuromuscular disease have shortened step lengths. Animals with UMN disease may appear irregular and uncoordinated, having a tendency to sway from side to side; some may fall to the side. Their feet may contact the ground with increased force. Doberman pinchers with cervical vertebral malformation - malarticulation, for example, may overflex the hock joints during weight-bearing, presumably the result of motor or sensory weakness.

Dogs with LMN disease may hold the head low (possibly because of neck muscle weakness) and take short ("choppy") steps. Some have a kyphotic posture. They may tire easily and appear unwilling to perform. Animals with orthopedic disease may have a similar gait abnormality wherein the limb step length is short. Normal conscious proprioception in the presence of a shortened step distance should suggest underlying orthopedic disease or neuromuscular weakness. Poor limb perfusion, such as from a partial bilateral iliac arterial thrombus or from a right-to-left PDA may also result in a short, choppy pelvic limb gait. The clinical signs may worsen with exercise (similar to myasthenia gravis) and differential cyanosis may be seen with the latter disease. Dogs with vertebral pain may also be short-strided and reluctant to move.

Depending upon where within the nervous system the lesion occurs, gait may be altered differently. Supratentorial disease will often not affect gait. Wide circling toward the side of a unilateral forebrain lesion is common. Occasionally, spasticity may be noticed, with the limb (primarily a thoracic limb) appearing stiff, floating and over-reaching. Also, apparent hypermetria may be found, usually in the thoracic limb opposite a supratentorial lesion.

Lesions of the brain stem and cervical spinal cord will often have dramatic effects on gait, frequently impairing the ability to stand and generate a gait. If the lesion occurs unilaterally, ipsilateral hemiparesis is noted. If pathways are affected bilaterally, tetraparesis will be present. Spinal reflexes should reflect the UMN nature of the lesion.

**Neurologic abnormalities resulting in abnormal gait**

**Intracranial disease**

Depending upon where within the nervous system the lesion occurs, gait may be altered differently. Supratentorial disease will often not affect gait. Wide circling toward the side of a unilateral forebrain lesion is common. Occasionally, spasticity may be noticed, with the limb (primarily a thoracic limb) appearing stiff, floating and over-reaching.

Lesions of the brain stem and cervical spinal cord will often have dramatic effects on gait, frequently impairing the ability to stand and generate a gait. If the lesion occurs unilaterally, ipsilateral hemiparesis is noted. If pathways are affected bilaterally, tetraparesis will be present. Spinal reflexes should reflect the UMN nature of the lesion.

**Spinal cord abnormalities**

Animals are commonly presented for problems involving the spine and spinal cord. Often, these clinical signs are reflected as problems with walking or moving (limb motion). If there is abnormal function of a limb, the first determination made is whether there is, in fact, a neurologic problem. If a neurologic problem is encountered, then a determination of the location of lesion or lesions is made. Once the location of the lesion is determined, an appropriate differential diagnosis and diagnostic plan can be formulated.

The spinal cord can be functionally divided based upon the spinal segments and associated clinical signs of disease affecting these segments. The following is a summary of the clinical signs associated with lesions of selected spinal segments.

The functional division of the spinal cord is primarily dependent on the presence of either upper motor neuron (UMN) or lower motor neuron (LMN) clinical signs to the limbs. The presence or absence of these characteristic signs is based upon the concept of local reflex (does not require conscious control) function and the normal control of these reflex functions from higher nervous system centers.

**Lower motor neuron disease**

Technically, the lower motor neuron includes the motor neuron cell body, the motor (efferent) peripheral nerve, the neuromuscular junction, and the muscle.