Neurologic Examination 1 and 2 - What is important and what isn’t.
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Neurologic examination
The aim of the neurologic exam is to determine
1. Whether neurologic deficits are present or not and
2. The location of possible lesion or lesions
May be simple and may be not!! Musculoskeletal abnormalities and cardiovascular disease can mimic neurologic abnormalities and vice versa.

In broadest terms does the animal have peripheral nerve (include here neuromuscular junction or muscle) disease, spinal cord disease or brain disease or more than one of the above - and therefore is the disease process likely to be focal, multifocal or diffuse. This is important as some diseases typically produce clinical signs which indicate diffuse or generalized neurologic abnormalities (for example metabolic and toxic diseases) whereas some disease processes typically result in focal neurologic signs for example IVDD or neoplasia however there are many atypical presentations.

Lesion localisation is important in formulating a list of possible diagnoses, in making recommendations on further diagnostic tests, the likely prognosis and treatment options. Early diagnosis and assessment of prognosis are important both in providing appropriate care and advice to owners.

Neurologic Examination
Neurological examination takes time and practice. It is good practice to do a limited neurologic examination on all patients to get an idea of what is normal (fairly wide variation). A standardized neurologic exam form may be useful to make sure all parts of a neurologic examination are done. The principles are the same for all species – obviously modification is necessary for larger animals.

A neurologic examination should include
1. An observation of an animal’s level of consciousness, mentation, behaviour and interaction with its owner. History is also very important in making an assessment of an
animal’s behaviour. Abnormal behaviour at home is just as important as abnormal behaviour in a consult room. Behaviour in a consult room can be very difficult to assess in anxious or aggressive dogs and cats.

2. An assessment of the animals’ gait and posture
For many animals (large dogs, most cats, aggressive or uncooperative animals) an assessment of behaviour, mentation and gait will be the sum total of information gained on neurologic exam. However this information is the most useful part of any neurologic exam and the most often overlooked. Take the animal outside on a leash (many dogs do not like walking on linoleum) or allow free range in a large room.

3. An evaluation of postural reactions including proprioceptive paw positioning, hopping, placing to a table top, hemiwalking, wheelbarrowing (all in smaller animals) or negotiation of stairs – up (especially useful in determining animals that are weak) and down (useful especially in identifying animals with any vestibular disturbance).


5. Evaluation of spinal reflexes – tendon reflexes (patellar, biceps and triceps), flexion reflexes (thoracic and pelvic limb-withdrawal), perineal (anal), and cutaneous truncal (panniculus) reflexes.

6. palpation of the head, neck and spine

7. evaluation of nociception (pain sensation) – “superficial” (if specific peripheral nerve injuries are suspected) and “deep pain” perception if necessary (in animals without voluntary movement).

Some points to remember
- Animals with known or suspected spinal trauma should be moved as little as possible both in transport and when examined. Neurologic examination needs to be modified to gain necessary information without moving the animal and risking further spinal cord injury. Evaluation of reflexes, pain sensation, voluntary movement, etc, can be done in lateral recumbency. Similarly radiographs should be taken in lateral recumbency – with animal strapped to board or table.
- An animal with suspected atlanto-axial instability or cervical fracture should have its head and neck manipulated with care (especially ventroflexion) when examined or radiographed.

Neurologic examination
The neurologic exam can be modified depending on size and temperament of the animal.
It is easiest to do the first part of the neurologic examination standing and then examine spinal reflexes and pain sensation in lateral recumbency. If possible examine animals on flooring where they can get good footing and move around freely, eg. outside on the grass or concrete, or in large room.

Mental status, behaviour and level of consciousness
From just watching an animal and by questioning an owner on the behaviour of their animal at home you can determine whether an animals mental status is normal, behaviour is appropriate or inappropriate for the surroundings, whether it is circling, has a head tilt, can see, can hear, etc. Animals may be assessed as normal, subdued, dull, confused, altered, demented, obtunded, stuporous or comatose (unconscious).
Owner observations and neurologic examination are important in determining whether brain dysfunction is present. Some clinical signs are very obvious, eg. seizures or vestibular signs, but others are more subtle and subjective and many animals with significant forebrain disease may have no obvious neurologic deficits and a very normal neurologic exam. An owner's history of persistent change in behaviour, loss of toilet training or learned commands or “not being right” without any physical cause established may be an indication of primary intracranial disease.

**Posture**

Does the animal look painful? Does it stand with an abnormal posture? Does have a tendency to turn, fall etc to one side? Head tilt?

**Gait**

An assessment of the animal’s gait should be made. Animals can be supported by the tail or towel sling to evaluate voluntary movement and many animals that are non ambulatory on smooth flooring may be ambulatory outside on grass or paving. Assess all four limbs.

Note:
1. Is animal lame, ataxic, paretic, hypermetric, stumbling, crossing limbs when walking, dragging or scuffing toes. Is the gait wide based or narrow based. Is the gait short or does the animal over reach with each stride.
2. If non ambulatory, is voluntary movement present – that is, movement not associated with reflex withdrawal from a noxious stimulus.
3. Is there any asymmetry ie is one side or one limb worse than the other/s.

**Paresis, Paralysis and Ataxia**

Abnormalities of gait and movement include ataxia, paresis (weakness) of one or more limbs, paralysis of one of more limbs, muscle stiffness and lameness. Lesions anywhere in the nervous system may result in gait abnormality.

Ataxia is incoordination of movement with normal muscle strength. Ataxia is a feature of abnormalities of the cerebellum, vestibular system (both peripheral and central components) and spinal cord lesions (especially cervical) which affect the spinal sensory pathways.

Dysmetria is an abnormality in the range of movement - movements may over reach the target (hypermetria) or be too short (hypometria) or appear “drunken” or staggering. Hypermetria is commonly recognized especially in the thoracic limbs and may be associated with cerebellar disease. However hypermetria /dysmetria may be seen in animals with lesions of the cervical spinal cord due to involvement of spinocerebellar pathways. The presence of ataxia of the head and neck or other signs of cerebellar disease will localise lesions to cerebellum rather than cervical spinal cord, however hypermetria of the limbs only may be due to cerebellar or spinal cord disease.

Increased muscle tone associated with upper motor neurone (UMN) lesions of the spinal cord may result in a stiff overextended gait (spasticity). A lesion may be spinal - cranial to limbs affected, or brainstem.
Paresis is weakness which may be seen as a decrease in muscle strength or as a reduction or loss of voluntary movement or both. Paresis may be seen with lesions anywhere in the nervous system caudal to the midbrain. Lesions of the spinal cord or brainstem may result in tetraparesis (all 4 limbs), hemiparesis (forelimb and hindlimb on the same side), paraparesis (both pelvic limbs) or paresis of one limb (monoparesis) only depending on the location of the lesion. Generalised abnormalities of the peripheral nervous system or muscle result in tetraparesis although clinical signs may initially be seen only in the pelvic limbs. **Muscle stiffness associated with a short strided gait and a tendency to sit frequently or reluctance to walk** is characteristic of generalised lower motor neurone disease (LMN) including disease of the peripheral nerves (polyneuropathy), muscle (polymyopathy) and neuromuscular junction (junctionopathy). Generalized LMN disease is better called Motor Unit disease. Clinical signs may be episodic especially in the case of junctionopathy.

**Paralysis** (plegia) is the absence of any voluntary movement and may be seen in one limb, both hind limbs (paraplegia), the fore and hind limb on one side (hemiplegia) or all four limbs (tetraplegia). Paralysis may be accompanied by lack of sensation (anaesthesia) in the affected limb(s).

**Lameness** (decreased or non-weight bearing on a limb) is usually a sign of musculoskeletal disease however lameness may be associated with conditions affecting the spinal nerve roots or peripheral nerves eg nerve root compression due to intervertebral disc extrusion or protrusion (nerve root signature) and initially in cases of nerve sheath tumour.

**Muscle strength /tone /atrophy** can be assessed. Are abnormalities generalized, focal or multifocal or in distribution of one nerve or nerves. Marked muscle atrophy is associated with denervation (LMN lesions) or muscle disease and less severe atrophy may be seen associated with disuse (UMN lesions). Increase in muscle tone is associated with UMN disease (spinal cord or brainstem) and hypotonia is often seen in LMN disease.

The vertebral column should be palpated carefully. Increased pressure can be applied if no response is initially seen. The neck muscles should be palpated. The neck can be extended, flexed and moved laterally to check for evidence of neck pain.

**Postural reactions**
Proprioception is the perception of the body in space. The postural reactions test both proprioception and the ability to effect a motor response. Proprioceptive paw positioning (often called conscious proprioception), hopping, hemiwalking, wheelbarrowing, placing paws onto a benchtop, all test the complex responses (not reflexes) that enable an animal to stand and move. These responses involve all components of the nervous system from peripheral nerve to cerebrum (hence the conscious) including sensory and motor pathways that enable the motor response. Proprioception also involves subcortical sensory pathways (primarily to the cerebellum) and it is very difficult to separate conscious from other “unconscious” proprioceptive mechanisms. Proprioceptive deficits are not specific for an abnormality anywhere in the nervous system but have localising value when taken in context of the rest of the neurological examination. Hopping, hemistanding, wheelbarrowing and hemiwalking are useful in revealing mild abnormalities and any asymmetry in response.
The may also be useful in assessing subtle weakness and abnormalities in voluntary motor movement.

As general rule animals with relatively normal gait but marked proprioceptive deficits on testing are more likely to have forebrain disease. Animals with significant gait abnormality (paresis, ataxia or both) and proprioceptive deficits (often with knuckling whilst walking) are more likely to have more caudal brainstem or spinal cord disease. Animals with significant paresis (may not be able to stand or walk) associated with LMN disease (or junctionopathy or myopathy) will often have normal proprioceptive paw positioning (conscious proprioception) if supported in a standing position.

**Remember:** when testing proprioceptive paw positioning (PPP) the limb to be tested should be supported lightly in a normal standing position. If the animal is allowed to sway vestibular input will be introduced. Proprioception is tested by turning the digits of a paw over so that the dorsum of the paw is in contact with the floor. A normal response is to replace the paw to a normal weight bearing position immediately. PPP is a good measure of sensory pathways reaching the cortex and may be more sensitive in determining deficits in animals with forebrain disease than other postural reactions if the body and tested limb are not swaying when testing.

Postural reaction tests all basically do the same thing and you do not need to do all in all dogs. Big dogs are difficult to hop, wheelbarrow, etc, and placing of limbs can be just as easily evaluated by watching a dog negotiate stairs (up and down). In a room placing a piece of newspaper under a foot and sliding the newspaper laterally also tests a dog’s proprioception. A normal animal should step back to normal standing position. Placing to a table top is often less reliable than other tests of postural reactions.

**Cranial nerve examination**

Cranial nerve abnormalities may be seen associated with brainstem disease or peripheral nerve disease and cranial nerve abnormalities can help localize lesions both intracranially and extracranially. With the exception of CN1 and II the cranial nerves are essentially specialized spinal nerves arising from the midbrain (111, IV) and caudal brainstem (V-X111) and may consist of sensory and/or motor elements. Motor nerves have nuclei within the brainstem. Sensory nerves have cell bodies located in ganglia outside of the brainstem and projections/tracts located within the brainstem. Single cranial nerve abnormalities may be seen associated with intracranial or extracranial diseases. Multiple cranial nerves may be involved in multifocal brain disease or diseases causing generalized peripheral neuropathy (usually abnormalities are bilateral). Multiple cranial nerves may be affected by focal lesions if they arise from adjacent areas of the brainstem or in their course within the cranial vault (this is where some knowledge of anatomy is helpful!).

Cranial nerve examination is most easily done in standing or sitting animals. At a minimum this should include
1. Palpebral fissure size and pupil size (may be altered by abnormalities in the visual pathway including CN II, CN III, sympathetic innervation to the eye, midbrain disease, cerebellar disease or ophthalmic disease)
2. Menace response tests visual pathway (including CN II, optic nerve, thalamus, visual cortex) perception of threat (cortex), and motor response CN V11. This is a response not a reflex and non neurologic abnormalities may affect this response. Cerebellar disease may also cause menace deficit. Menace response is not present in young puppies and kittens. It is usually present consistently by 14 weeks of age.
4. Pupillary light reflexes (tests visual pathway to the level of the midbrain and parasympathetic component of CN III). The pupillary light reflex requires a strong light source to elicit in many anxious and/or frightened animals. Ocular abnormalities especially iris atrophy may alter the pupillary light reflex in animals without visual or CN111 deficits. CN111 abnormalities are uncommon. PLR is often still present in animals with significant ocular disease that may be behaviourally blind.

Dazzle reflex- A strong light will induce a blink mediated by subcortical structures.
5. Palpebral reflex  (tests CN V and CN VII function). The palpebral reflex is less consistent (and absent in normal animals) with touching the lateral canthus of the eye (maxillary branch of CNV) than the medial canthus (ophthalmic branch of CNV)
5. Corneal reflex (tests CN V ophthalmic branch and CN VI function)
6. Facial sensation (CN V) and facial muscle movement (CN VII)
7. Masticatory muscle mass, jaw tone, (CN V mandibular branch)
8. Swallowing, gag, assessment of ability to eat and drink (question owner) (CN IX and X)
   The gag “reflex” tested by putting ones finger or hand in an animal pharynx does not initiate swallowing or gagging in many normal animals. History of (or observable) dysphagia or aspiration, gagging or coughing when eating or drinking is more reliable than the gag “reflex” in assessing pharyngeal sensation and swallowing.
9. Tongue movement (CN XII)
10. Assessment of normal and any abnormal eye movements (strabismus, nystagmus) tests CN VIII, CN III, CN IV, CN VI and brainstem (especially midbrain) pathways.
11. Question owner re any change in bark/voice (CN X, recurrent laryngeal nerve)

Function and clinical signs associated with dysfunction of the cranial nerves

<table>
<thead>
<tr>
<th>Cranial Nerve</th>
<th>Sensory Function</th>
<th>Clinical Signs</th>
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<tbody>
<tr>
<td>Olfactory (I)</td>
<td>Sensory-olfaction</td>
<td>Anosmia (rarely recognised)</td>
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<tr>
<td>Optic (II)</td>
<td>Sensory-visual</td>
<td>Visual deficits</td>
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<td>Cranial Nerve</td>
<td>Function</td>
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<td>Occulomotor (III)</td>
<td>motor - extraocular muscles ventrolateral stabismus</td>
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<td></td>
<td>-levator palpebrae ptosis</td>
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<td>normal vision</td>
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<td></td>
<td>parasympathetic to iris dilated unresponsive pupil</td>
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<tr>
<td>Trochlear (IV) recognised</td>
<td>motor-extraocular muscle mild strabismus (rarely recognised)</td>
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<td>Trigeminal (V) muscles</td>
<td>motor - muscles of mastication atrophy of masticatory</td>
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<tr>
<td></td>
<td>sensory to face, cornea, mouth, tongue decreased sensation</td>
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<td>Abducens (VI) retract globe</td>
<td>motor-extraocular muscles medial strabismus, inability to retract globe</td>
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<tr>
<td>Facial (VII) muscles</td>
<td>motor- muscles of facial expression paresis or paralysis of facial</td>
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<td></td>
<td>tear production dry eye</td>
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<td></td>
<td>sensory - taste</td>
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<td>Vestibulococchlear (VIII) sensory - hearing</td>
<td>deafness</td>
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<td>sensory - equilibrium disequilibrium, ataxia, head</td>
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<td>tilt positional strabismus.</td>
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<td></td>
<td>nystagmus</td>
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<tr>
<td>Glossopharyngeal (IX) sensory to pharynx ,taste</td>
<td>motor to pharyngeal muscles dysphagia</td>
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<tr>
<td>Vagus (X) sensory to larynx, pharynx and viscera</td>
<td>motor to pharynx, dysphagia</td>
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<td></td>
<td>motor to larynx laryngeal paralysis, change</td>
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<td>motor to oesophagus megaoesophagus</td>
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<td></td>
<td>parasympathetic to viscera GIT signs, bradycardia, tachycardia</td>
<td></td>
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<td>Accessory (XI) muscle(rarely seen)</td>
<td>motor-trapezius muscle atrophy trapezius</td>
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<td>Hypoglossal (XII) motor to the tongue</td>
<td>paresis, paralysis, atrophy of the tongue</td>
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Sympathetic innervations to the eye –is not a cranial nerve but is included here.

**Horner's syndrome** is due to loss of sympathetic innervation to the smooth muscle structures of the eye, orbit and head and is characterised by miosis, enophthalmos and
third eyelid prolapse, ptosis and vasodilation. The tortuous course of the sympathetic innervation to the eye makes Horner’s syndrome possible with disease in a number of different locations including:

1. Brain and Cervical spinal cord – severe cervical myelopathy due to acute trauma, IV disc extrusion or FCE
2. T1-T3 spinal segments or spinal nerve roots - same as above and brachial plexus avulsion injuries and spinal nerve sheath tumours
3. Cranial sympathetic trunk within the thorax - intrathoracic tumours or other mass lesions
5. Middle ear-infection, neoplasia, inflammatory polyps (cats)
6. Region of the cavernous sinus (intracranial) - very unusual as other neurologic abnormalities are more likely to be seen and Horner’s Syndrome rarely recognised. Tumour most common cause.
7. Retrobulbar - neoplasia, trauma – usually associated with other neurologic abnormalities as in close apposition to other ocular nerves.

Spinal reflexes
The examination of spinal reflexes is important as it can help in localizing lesions to particular segments of the spinal cord which mediate these reflexes or to one or more peripheral nerves.

Spinal reflexes and pain sensation are best examined in lateral recumbency.
In lateral recumbency muscle tone and muscle mass can again be evaluated. Each spinal reflex has a sensory and motor (LMN) component and each reflex is influenced by descending motor (UMN) pathways with the spinal cord. The most reliably interpreted spinal reflexes are the patellar (quadriceps) reflex and the flexor (withdrawal) reflexes in the thoracic and pelvic limbs. The biceps and triceps tendon reflexes are also elicited in most animals but decreased or absent reflexes are not always abnormal. Patellar reflexes may not be present in normal animals. Other reflexes are much less reliable and not really useful clinically.

The patellar reflex is mediated by the femoral nerve (sensory and motor, spinal cord origin L4-L6). The pelvic limb flexion reflex is mediated by both the sciatic (predominant) and femoral nerves (spinal cord origin L4-S1). The biceps and triceps reflex are mediated by the musculocutaneous and radial nerves predominantly (spinal cord origin C6-T1). The thoracic limb flexion reflex is mediated by all major nerves of the thoracic
limb (spinal cord origin C5-T2). When testing spinal reflexes be aware of the presence of any abnormal reflexes eg crossed extensor reflex, extensor thrust. Crossed extensor reflexes when a withdrawal reflex is elicited in one limb the contralateral limb extends are not generally seen in normal animals. These reflexes are seen as an UMN abnormality. They are most commonly seen in animals with chronic UMN lesions rather than acute spinal cord lesions. Spinal reflexes should be assessed as normal, hyporeflexic (poor), hypereflexic (exaggerated or clonic) or absent. There is a wide variation in a normal patellar reflex.

The perineal or anal reflex (mediated by the pudendal nerve spinal cord origin S1-S3) should be examined and perineum evaluated for faecal soiling. The tail should be examined and voluntary movement and muscle tone assessed. Perineal sensation should also be assessed in animals with poor anal tone. The bulbocavernosus reflex tests the same reflex arc as the anal reflex.

The cutaneous trunci (panniculus) reflex is most easily tested in a standing or upright position. It can aid in determining the site of a thoraco-lumbar lesion as sensory input is from each cutaneous sensory nerve (1 per segment) of the thoracic and cranial lumbar spinal cord. A sensory “cut-off” may help localize a spinal lesion. It is normally a bilateral reflex ie stimulating one side will elicit a bilateral response. The cutaneous sensory dermatomes do overlap and are located more caudally than the spinal foramen from which the cutaneous nerve arises in the caudal thoracic and lumbar region ie the L2 or L3 dermatome may extend to the ilial wing. An absent cutaneous trunci reflex unilaterally or bilaterally localises a lesion to the C8-T1 vertebral segments (esp FCE), spinal nerve roots (may seen in avulsion injuries of the spinal nerve roots of the brachial plexus) or spinal nerves (nerve sheath tumours) which contribute to the lateral thoracic nerve (efferent of this reflex). This reflex is not normally seen caudal to the wings of the ilium.

The bladder should be palpated. Urinary incontinence or the inability to urinate normally is often associated with spinal disorders.

A large flaccid bladder which is easy to express and “overflow” incontinence is often seen with disorders of the sacral spinal cord segments or spinal nerve roots (cauda equina) (often referred to as an LMN bladder).

A large tense bladder which is difficult to express or dribbles a small amount of urine before increased urinary sphincter tone stops the flow of urine is often seen with lesions cranial to the sacral segments (often referred to as an UMN bladder).

Absent or decreased spinal reflexes are indicative of an abnormality in the spinal cord segments or spinal nerve roots or peripheral nerves that mediate the reflex.

An absent or reduced spinal reflex is often referred to as a Lower Motor Neurone (LMN) sign.

Exaggerated or clonic reflexes or the presence of abnormal reflexes such as the crossed extensor reflex are indicative of interference with the normal inhibition of the spinal reflex by descending pathways within the spinal cord cranial to the segments mediating the reflex arc. Exaggerated reflexes are often referred to as an Upper Motor Neurone (UMN) sign.

Points to note:
- Very tense animals may have exaggerated tendon reflexes that are not abnormal.
• It may also be difficult to elicit any tendon reflexes in animals with increased muscle tone.
• An exaggerated patellar reflex is usually associated with lesions cranial to L₄ as the reflex is dependent on normal L₄ – L₆ spinal cord segments. However, an exaggerated reflex may be seen with lesions caudal to L₆ due to denervation of muscles innervated by the sciatic nerve. These muscles generally counteract the kick of the patellar reflex. This is often called a pseudoexaggerated reflex.
• Old dogs may have decreased or absent patella reflexes and no other signs of spinal cord or peripheral nerve disease. This is probably due to an age-related change in the dorsal root ganglia.
• Many animals with spinal cord disease have normal spinal reflexes regardless of the location of the lesion. Exaggerated reflexes and crossed extensor reflexes are seen most commonly in chronic spinal cord lesions. Absent or decreased reflexes are most commonly seen with grey matter destruction in the cervical or lumbosacral intumescences, for example FCE or trauma rather than more chronic diseases predominantly affecting spinal cord white matter such as caudal cervical spondylomyelopathy (wobbler syndrome).
• Acute spinal cord lesions (e.g., trauma/acute IV disc extrusion) may be associated with reduced spinal reflexes caudal to lesion when normal to increased reflexes would be expected due to “spinal shock” - thought not to occur in dogs but may in the first 24 hours or so post injury.

from Small Animal Spinal Disorders Sharp and Wheeler 2nd edn

“Pain” sensation - nociception
Pain sensation should be evaluated in all limbs and tail. Generally speaking if an animal can voluntarily move a limb, it can feel it, so when testing pain perception, apply a less noxious stimulus (“superficial”) first – pinching the skin rather than crushing toes. If there is no behavioural response to a superficial stimulus, deep pain is tested by squeezing across a digit (bone) or nail bed with haemostats or needle holders. Note well – reflex withdrawal is not an indication of pain perception. If the segments of the spinal cord necessary for reflex withdrawal of a limb are intact, the limb will move (the contralateral limb may also move as part of a crossed extensor reflex and the tail may also move reflexly when stimulating a pelvic limb). This doesn’t mean the spinal cord is intact cranial to these segments. A behavioural response has to be seen to indicate perception of a painful stimulus (turning of the head, crying out, etc). Very stoic
dogs may just show a change in respiratory rate but the response needs to be repeatable and consistently seen with painful stimulus. If in doubt apply a stimulus cranial to suspected lesion (eg a forelimb if a TL lesion is suspected) and note the response. If a lesion is suspected in the cervical or lumbar enlargements (C6 – T2, L4 – Cd5) the autonomous sensory zones of each peripheral nerve needs to be tested. Lesions of the spinal cord segments and spinal nerve roots contributing to peripheral nerves can produce abnormalities in the sensory distribution of those nerves. Autonomous sensory zones are also important in assessing the severity of peripheral nerve injuries.

**Loss of deep pain sensation is a poor prognostic sign** and indicative of severe spinal cord or nerve damage. It is therefore the presence or absence of deep pain perception is of major importance prognostically regardless of the cause of the spinal cord lesion. Tracts in the spinal cord necessary for noxious perception are located throughout the cord, the fibres smaller in diameter, myelinated and unmyelinated and more resistant to injury than more superficial, larger diameter sensory neurones such as those necessary for conscious proprioception and other postural reactions.

**Increased sensitivity** to pain (hyperesthesia) may be generalised or detected in a focal area. It probably results from irritation of nerve roots or meanings (which have their own sensory receptors).

**Pain** on palpation of the spine or neck, or focal areas of hyperesthesia may help in localising spinal lesions especially extradural compressive lesions (eg IV disc extrusion), diseases of the meninges or vertebrae (discospondylitis, vertebral tumours). Diffuse or multifocal spinal pain is highly suggestive of meningitis and/or myelitis but may also be seen in animals with polyarthritis, polymyositis or multifocal spinal metastases. **Animals with lumbosacral pain** may have a hunched posture, show reluctance to jump or go up or down stairs, stiff pelvic limb gait and thoracolumbar spinal hyperesthesia. **Animals with thoracolumbar pain** may have a hunched posture, show reluctance to jump or go up or down stairs, stiff pelvic limb gait and thoracolumbar spinal hyperesthesia. **Cervical lesions commonly cause neck pain**. Cervical pain is often severe and dogs will hold the head and neck rigidly, cry when trying to move and spasms or fasciculations of the cervical muscles are commonly seen or palpated when the animal is examined. Animals with less severe neck pain may tend to turn their whole body laterally rather than turn head and neck only.

**Abdominal pain** may be presenting sign in dogs with thoracolumbar spinal lesions and chondrodystrophoid dogs (with a higher incidence of intervertebral disc disease) should be observed closely and treated as a possible IV disc extrusion until cause of abdominal pain is established.

**On the basis of the findings on neurologic examination one should be able to determine if neurologic abnormalities are present. If neurologic abnormalities are present can they be localized to the brain, spinal cord or one or more peripheral nerves (motor unit). Are the abnormalities attributable to a focal abnormality or are multifocal or more diffuse neurologic abnormalities present.**

**Localising lesions within the nervous system** can be difficult but a localization to the brain, spinal cord or peripheral nerves is the first step –may be more than one.
Lesions within the brain may be localized to the forebrain (essentially the cerebral hemispheres, thalamus and hypothalamus (structures in front of the tentorium cerebelli in the rostral and middle cranial fossae) or the caudal brainstem and/or cerebellum (structures within the caudal fossa or hindbrain) or to the midbrain (the bit in the middle). Lesions may be very focal however larger lesions may cause obstruction of CSF pathways or vascular alteration or increases in intracranial pressure that may produce more diffuse neurologic abnormality. Localisation of lesions to explain clinical abnormalities seen is imperative when considering further diagnostic tests to determine the likely cause. Advanced imaging is not useful if the wrong bit is imaged.

The clinical signs seen with lesions of the forebrain are:
1. Disturbances of consciousness
2. Abnormal behaviour - head pressing, getting stuck in corners, pacing, restlessness
3. Circling towards the side of the lesion in cases of unilateral lesions. Circling is a tendency rather than compulsive. Head turn may also be seen towards the side of lesion.
4. Postural reaction deficits contralateral to the side of the lesion. Mild contralateral hemiparesis may be seen but relatively normal gait.
5. Visual deficits contralateral to the side of the lesion with normal pupillary light reflexes. Note the menace response may be lost (contralateral) without associated loss of vision, with some lesions in the sensorimotor cortical area as menace is really a motor response to visual stimuli not a reflex.
6. Seizures - partial or generalised. Partial seizures with involuntary movements in a localised area, eg facial muscles or one leg may spread to one half of the body (contralateral to lesion) or may be followed by secondary generalisation.

Endocrine and/or autonomic disturbances - can be seen with hypothalamic involvement. These include polyuria and polydipsia (central diabetes insipidus), hyperphagia or anorexia, poor temperature regulation or altered sleep patterns. The pituitary gland lies in the hypophyseal fossa ventral to the hypothalamus and thalamus. Macroadenomas or pituitary adenocarcinomas can result in neurologic signs of thalamic and/or cerebral dysfunction with or without clinical signs of hyperadrenocorticism.

The characteristic abnormalities of midbrain disease are:
1. alterations in level of consciousness - varying degrees
2. compulsive circling or head turn usually towards the side of the lesion
3. postural reaction deficits - contralateral or ipsilateral
4. lack of normal eye movements (physiologic nystagmus or oculovestibular reflexes)
5. CN III abnormalities - ventrolateral strabismus and/or mydriasis
6. respiratory pattern abnormalities

Cerebellar disease is characterised by:
1. ataxia (uncoordinated movements with normal strength);
2. dysmetria (abnormality in range of movement - too long or too short) especially hypermetria;
3. wide based stance
4. intention tremor. Some degree of abnormal head movement should be seen to differentiate cerebellar disease from spinal cord disease as cervical lesions can produce signs of cerebellar ataxia in fore or hindlimbs due to involvement of spinocerebellar tracts.
5. Cerebellar disease may be associated with menace deficit. Vision and palpebral reflexes are normal. The mechanism of this abnormality is not clear.
6. Lesions of the flocculonodular lobe and/or cerebellar peduncles can cause signs of vestibular disease which may be contralateral (paradoxical vestibular disease). Lesions in this area (cerebello-pontine angle) are not uncommon - both inflammatory and neoplastic.

Acute cerebellar lesions which can occur usually as a result of trauma present very differently. Decerebellate posturing is characterised by opisthotonus and extension of the thoracic limbs, either extension of flexion of the pelvic limbs and either extension or flexion of the pelvic limbs.

Lesions in the caudal brainstem (pons and medulla oblongata) are characterised by:
1. abnormal gait
2. abnormal postural reactions -ipsilateral
3. ipsilateral hemiparesis or tetraparesis.
4. Alterations in level of consciousness due to involvement of the brainstem reticular formation is common.
5. Respiratory pattern abnormalities are seen in severe brainstem lesions (trauma).
6. Cranial nerves V-XII arise from the pons and medulla (numbered from rostral to caudal). Changes in function of any of these cranial nerves associated with any of the above is the best evidence for brainstem disease.

Cranial nerve abnormalities without other signs of brainstem disease are more likely to be due to peripheral neuropathy rather than brainstem disease.

If a lesion can be localized to the spinal cord which segment or segments of the spinal cord is/are affected?
Lower motor neurone deficits versus Upper motor neurone deficits
Lower motor neurone deficits indicate an abnormality of the lower motor neurones that is the spinal cord segments containing the neuronal cell bodies, their ventral spinal nerve roots and peripheral nerves therefore LMN deficits can be seen in some spinal cord lesions and are also seen in neuromuscular disorders.

Lower motor neurone deficits include:
- paresis or paralysis
- reduction or loss of muscle tone
- normal, decreased or absent spinal reflexes
- muscle atrophy - early onset (neurogenic).

Upper motor neurone deficits are indicative of an abnormality in transmission by the upper motor neurones that is descending neurones within the CNS - brain, brainstem and spinal cord - that synapse on LMNs and initiate voluntary movement and influence muscle tone, reflexes and involuntary motor functions.

Upper motor neurone deficits include
- paresis or paralysis
- normal or increased muscle tone
- normal or exaggerated spinal reflexes
- no or mild muscle atrophy - late onset (disuse)

Clinical signs of spinal cord disease
- Gait abnormality – paresis or paralysis and/or ataxia
- Postural reaction deficits dysfunction of both sensory (proprioceptive) and motor pathways (UMN +/or LMN)
- One or more limbs depending on location of the lesion
- spinal reflex abnormalities depending on lesion location
- abnormalities in pain perception
- spinal pain
- bladder abnormalities and/or faecal incontinence
The spinal cord can be divided into four regions based on limbs affected and reflex abnormalities expected in each region. However, spinal reflexes may be normal in lesions C6-T2 and L4-S1 and often it is difficult to be as precise re likely location of a spinal cord lesion.

**In animals with spinal cord disease**

**Remember**

- more than one lesion may be present and a caudal lesion may be masked by a more cranial lesion.
- some lesions may be very asymmetric especially fibrocartilagenous embolism. The lesion is usually on the side of the greatest deficit.
- animals with cervical lesions may have a disparity in the severity of deficits in the thoracic and pelvic limbs (especially true of caudal cervical lesions). Generally deficits are more severe in the pelvic limbs however may be much worse in the thoracic limbs as spinal tracts to thoracic and pelvic limbs are located separately within the spinal cord.
- Spinal reflexes are often normal in animals with spinal cord disease regardless of where the abnormality is. Not all animals with spinal cord disease can be neatly categorised as textbooks suggest. The presence of normal spinal reflexes does not rule out a lesion in the C6-T2 or L4-Cd segments. The lack of exaggerated reflexes does not rule out a lesion in the C1-C5 or T3–L3 spinal cord segments.
- LMN deficits in the thoracic limbs are uncommon in caudal cervical lesions except in lesions which affect the gray matter of the spinal cord for example fibrocartilagenous embolism or spinal nerve roots for example nerve sheath
tumours. Animals with chronic caudal cervical lesions especially caudal cervical spondylomyelopathy (Wobbler syndrome) often have a wide based ataxic pelvic limb gait with variable paraparesis and a stiff short forelimb gait (as does any lesion at C6C7) with no appreciable ataxia or paresis of the forelimbs.

- Lesions of the brainstem can be difficult to distinguish from lesions of the cervical spinal cord and may only be separated by the presence of other abnormalities indicative of brainstem disease such as alterations in consciousness and/or cranial nerve deficits.
- Cranial cervical trauma may also be associated with clinical signs indicative of caudal brainstem or cerebellar injury (eg. head tilt, pharyngeal paresis).
- Neck pain although most commonly associated with disease of cervical spinal cord may also be seen in animals with brainstem or forebrain disease and may be the only presenting complaint. Neurologic deficits indicative of intracranial disease may or may not be present.
- Generalised LMN disease including polyneuropathy, polymyopathy and junctionopathy can also be difficult to distinguish from cervical spinal cord disease. Tetraparesis (progressive or episodic) is typical of generalised LMN disease. Hyporeflexia is typical of polyneuropathy however normal spinal reflexes may be seen and normal spinal reflexes are typical of polymyopathy and junctionopathy. Postural reactions (eg conscious proprioception are usually normal unless animals are severely paretic). Loss or change of voice, dysphagia or megaoesophagus may be seen in LMN disease.
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**Generalised LMN disease** including polyneuropathy, polymyopathy and junctionopathy is characterised by

- tetraparesis that may be progressive or episodic. Weakness may be ascending-seen in pelvic limbs then thoracic limbs.
- Proprioceptive paw positioning is generally preserved if animal is supported until severe paresis evident.
- generalised LMN deficits
- spinal reflexes generally decreased or absent in animals with polyneuropathy but may be normal initially and in animals with polymyopathy or junctionopathy (eg myasthenia gravis) spinal reflexes are usually normal.
- spinal pain generally not seen however acute polyradiculoneuritis may cause generalised hyperaesthesia initially and polymyopathy especially polymyositis may result in significant muscle pain.

Animals with generalised LMN disease may hold head and neck low probably due to cervical weakness, have a short stiff gait and tire easily.
Loss of bark and vocalisation is common due to paresis of recurrent laryngeal nerves or involvement of laryngeal muscles in myopathy.

Decreased, absent or abnormal sensation (parasthesia) is indicative of sensory neuronal involvement. Sensory deficits are more commonly seen in neuropathies affecting one or multiple peripheral nerves in one limb.