Neurological examination and neurolocalization in the paretic/paralysed patient (C1-S3)

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Paresis/paralysis is a common neurologic presentation in dogs and cats. To establish if the animal is suffering from neurologic disease, or not, is usually not difficult but to identify the neuroanatomical localization of this problem can sometimes pose a challenge to the more unexperienced clinicians.

The reasons for performing a neurologic examination is solely to answer these two questions:

- Does the animal have a neurologic problem?
- If there is a neurologic problem, where is it located?

When examining a patient suspected for a spinal problem we are mostly looking for a focal lesion but it is important to realize that sometimes the lesions can be very extensive and thereby affecting more than one region of the spinal cord, or even be multifocal. In addition, many patients presenting with paresis/paralysis may not have a spinal problem at all but may be affected by a diffuse generalized neuromuscular disorder or a peripheral neuropathy. Therefore, one of the most important aspects is to identify if the patient is affected by a spinal or a peripheral neuromuscular problem. This may sound straight forward but can pose quite a challenge in some patients and it is therefore important to pay attention to this question while obtaining the patient history and during the neurological examination.

One of the most important aspects of the neurological examinations in the paretic/ataxic patient is the gait analysis. By simply observing the animal from a distance it is often possible to determine which limbs are involved and if those limbs have an upper motor neuropathy (UMN) or a lower motor neuropathy (LMN).

An animal with a UMN paresis/paralysis will have a delayed protraction of the affected limb(s) and thereby longer, uncoordinated steps. They will often scuff their paws when walking and thereby their nails are often worn down, even on the dorsal aspect if it is a long-standing problem.

Hallmarks for upper motor neuron (UMN) spinal cord disease:

- Paresis and proprioceptive ataxia (incoordination/inability on hopping, not correcting paw placement)
- Delayed protraction of the affected limb(s) and thereby longer, uncoordinated steps
- No or mild muscle atrophy
- Spastic paresis/paralysis with normal or increased muscle tone
- Normo- to hyperreflexia
- Abnormal spinal reflexes – crossed extensor or myoclonus

Hallmarks for lower motor neuron (LMN) spinal cord disease and peripheral neuromuscular disease:

- Weakness (no ataxia if only motor neurons are affected – sensory neurons can be intact)
- Muscle atrophy (occurs quickly, within days, and is often profound)
- Flaccid paresis/paralysis with reduced to absent muscle tone
- Hypo reflexia to absent reflexes
- Proprioception is generally intact (if the animal is strong enough to weightbare)
- The gait pattern is characterized by a short stepping gait (as if the animal is walking on broken glass)
- Weakness – generally flexor muscles
- Bunny hopping if both hind limbs are affected
- Tremors while standing still

Once we have asserted that it is indeed a spinal patient (and not a peripheral neuromuscular problem) we try to localize the problem into one of the following regions:

- C1-C5 UMN signs in both thoracic and pelvic limbs.
- C6-T2 It should be LMN signs in the thoracic limbs and UMN signs in the pelvic limbs but often we see UMN signs in both thoracic and pelvic limbs.
- T3-L3 Normal thoracic limbs and UMN signs in the pelvic limbs.
- L4-S1 Normal thoracic limbs and LMN signs in the pelvic limbs.
- S1-S3 Normal thoracic and pelvic limbs with signs of LMN urinary and fecal incontinence. Sometimes there can be mild LMN signs in the pelvic limbs.

Low Cervical lesions - Wobbler gait
Low cervical lesions tend to cause a very typical gait pattern called "Two engine gait". The reason for this typical gait pattern are the short, stiff strides in the fore limbs (LMN signs) and the long, ataxic strides with the hind limbs (UMN signs). Sometimes the lesion is not severe enough to cause loss of proprioception in front limbs and it is only obvious in the hindlimbs. It is therefore crucial to always evaluate if patients presenting with only paresis posterior have neck pain and not always assume that they have a T3-L3 lesion. Sometimes a fore limb lameness (nerve root signature) is the most prominent sign of a low cervical spinal cord lesion.

Shiff-Sherrington Syndrome
This is seen in animals with an acute spinal cord injury T2-L4.
They will present with an increase muscle tonus of fore limbs and a flaccid paralysis of the hind limbs. This may seem paradoxical but it is caused by loss of cranial inhibitory innervation derived from "border cells" which are located in the grey matter of the spinal cord T2-L4 which send inhibitory signals to the motor neurons of the fore limbs. This syndrome is usually only seen in the acute phase and is associated with severe spinal cord injury but is not reported to hold prognostic value.

Spinal shock
This is a paradoxical syndrome only seen in case of an acute, severe spinal cord trauma/injury where an UMN lesion (T3-L3) causes temporary flaccid paralysis and loss of spinal reflexes in the pelvic limbs. Even the pain perception can be temporary absent. It only occurs in the acute phase and mostly only lasts for up to 24 hours in dogs and cats. It is believed that this phenomenon is caused by sudden loss of all communication between the vestibular/cerebellar centres and the LMN of the pelvic limbs which results in hyperpolarisation of these lower motor neurons, rendering them unresponsive to any stimulation.

Lumbosacral disease
As the spinal cord stops in the area of L5-L6 in most dogs, a lumbosacral spinal lesion will not cause spinal cord compression but only compression of the cauda equina. These patients will present with a mild kyphosis and a short stepping (LMN) gait of the pelvic limbs. A lumbosacral lesion will generally only result in pain and rarely in proprioceptive deficits. If there are clear proprioceptive deficits of the pelvic limbs it is almost never caused by a lumbosacral hernia and the cause should be looked for further cranially.

Determining if a patient has deep pain sensation in his limbs is the most prognostically important aspect of the entire neurologic examination and therefore crucial that it is performed and interpreted correctly. You should pinch the most sensitive part of the digits, which are the nail beds (and NOT pinch in the web between the toes which is a reasonably unsensitive area). Initially use your fingers and pinch hard over the nail bed. If there is no clear response, then use a pair of haemostats/artery forceps and pinch hard. The lateral and medial toes, on both thoracic and pelvic limbs, are innervated by different nerves - hence it is important that one pinches the most medial toe if there is no reaction when pinching lateral toe and vice versa.
The fact that an animal has an intact withdrawal reflex must not be confused with having deep pain sensation, withdrawal reflex is a spinal reflex and can be intact in patients with a severed spinal cord. To verify that a patient has an intact deep pain sensation, the animal must show a cortical response (vocalizing, turning his head etc.) when the digit is pinched.
The difference in prognosis for recovery of ambulation, between intact deep pain sensation (80-90% of the animals recover their ambulation if operated) and absence of deep pain sensation (around 50% of the animals recover if operated within 24 h.) is very significant which is why this part of the examination is of vital importance.