NEUROLOGICAL EXAMINATIONS: LOCALISATION AND GRADING

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MARK LOWRIE MA, VetMB, MVM, DipECVN, MRCVS in the second of a three-part article, provides a step-by-step approach to focusing on determining the severity and position of the problem

AS described in the previous article (*VT*44.20), the neurological examination has key aims and questions to answer:

• Is the problem definitely neurological?

• What is the location of this lesion in the nervous system?

• How severe is the disease?

• What are the main types of disease process that can explain the clinical signs?

Whether the problem is definitely neurological was tackled in the first article. Having determined a patient truly is neurological we can embark on a neurological examination.

This article will describe how the problem can be further localised and graded based on the other three questions. It will highlight the importance of not skipping the neurological examination and give insights about the important information that can be obtained by refining this skill. A discussion of the common diseases affecting the spinal cord will follow in a future article.

What is the location of this lesion?

The nervous system can be crudely divided into segments (Figure 1). The idea is to precisely localise the lesion to a progressively smaller area of the nervous system. By the end of the neurological examination it should be possible to locate the lesion in one or more of these areas. The examination can be divided into a series of questions that should be sequentially answered.

The neurological examination starts with watching the animal walk and observing how it interacts with its surroundings. Gait assessment is the only way of revealing certain abnormalities (for example, circling or hypermetria) although observation will also allow evaluation of posture (for example, the presence of a head tilt, wide-based stance and so on), mental status (for example, is the patient alert, obtunded, stuporous or comatose) and the presence of any abnormal behaviours and involuntary movements.

a) How many limbs are affected?

Having understood the nature of the gait abnormality (such as whether the patient is ataxic, lame or paretic) and associated signs, it is important to determine how many limbs are affected.

In many cases, particularly in patients that are non-ambulatory, this can be relatively straightforward. However, when managing ambulatory patients it can be very difficult to distinguish subtle neurological signs in potentially affected limbs. Paw positioning and hopping responses do not need to be tested if a patient is obviously dragging a limb. However, if the results of gait observation are equivocal, these procedures are extremely useful to detect subclinical neurological disease.

b) How are the reflexes in the affected limbs?

Having ascertained which limbs are affected we can slowly narrow down the location of the lesion (Figure 1). For example, a patient with only pelvic limb involvement will have a lesion caudal to T2 but a patient with all four limbs affected will have a lesion cranially to T2 or in the neuromuscular system.

To narrow this localisation further we must check the reflexes in the affected limbs and determine whether upper or lower motor neuron reflexes are present (Table 1). The patellar reflex can be considered. It is a monosynaptic reflex that evaluates the femoral nerve (L4 to L6). However, its presence and absence are unreliable, with many older dogs losing this reflex as a normal finding. With this in mind, I tend to use the withdrawal reflex (pedal or flexor reflex) as it evaluates multiple thoracic (C6 to T1) and pelvic (L6 to S1) limb nerves and seems reliable regardless of age. The importance of doing this is to determine whether the reflex is decreased or absent.

If lower motor neuron signs are present then the lesion is affecting the reflex arc and hence the

lesion will be in the L4 to S3 spinal segments (if only the pelvic limbs are affected) or the C6 to T2 spinal segments (if only the forelimbs are affected). If all four limbs are affected with lower motor neuron signs (decreased tone, decreased reflexes and atrophy) then the lesion is suspected to be involving the neuromuscular system.

If upper motor neuron signs are present to all four limbs then the lesion will be cranial to the brachial plexus (cranial to T2). If only the pelvic limbs are affected with upper motor neuron signs then the lesion will be in the T3 to L3 segments.

How severe is the disease?

The spinal cord is important in the perception of pain (performed via small diameter slow conducting neurons), in enabling voluntary movement (via descending motor fibres), and the transmission of spatial awareness (proprioception; performed via ascending large diameter fast conducting myelinated axons). These functions are lost sequentially as a spinal cord injury progresses. The large diameter fast-conducting myelinated axons are the first to be affected in spinal cord disease followed by the motor fibres. The most resilient neurons are the slow-conducting small diameter neurons involved in deep pain perception that are contained deep in the spinal cord white matter. Therefore, the first clinical sign observed in spinal cord injury would be ataxia followed by paresis. Pain perception is the last thing to be lost.

Pain perception is, therefore, the most important factor in determining prognosis. Any patient that has voluntary movement (no matter how little) in the affected limb will have retained pain perception and testing this should be reserved only for those cases that have complete paralysis of a limb or limbs.

A conscious and positive deep pain perception response is defined as the animal turning around and making some form of behavioural response that indicates they have perceived the painful stimulus, for example, whimpering or trying to bite when a pair of haemostats is applied to a digit. A withdrawal of the limb is not sufficient to declare deep pain present (**Panel 1**). An absence of deep pain perception should be considered an emergency with a prognosis of 50 per cent to 70 per cent if treatment is administered within 12 hours of losing pain perception.

Assessing severity in cervical and lumbosacral disease

Patients with cervical lesions will always have intact deep pain perception because a lesion severe enough to diminish this response would also abolish voluntary respiratory movements leading to death. Therefore, deep pain perception is a less useful indicator in cervical spinal disease.

A lesion of the lumbosacral spine will not cause paralysis to the pelvic limbs. Instead, it will cause signs compatible with damage to the nerve roots in this region (typically the sacral nerve roots; Figure 2). This is because the vertebral canal in this region contains the cauda equina (predominantly the

sacral nerve roots) rather than the spinal cord. A lesion to the cauda equina therefore has different implications to lesions involving the spinal cord. The sacral nerve roots supply the tail, anus and perineum. Complete laceration of these nerve roots would cause a flaccid tail with absent deep pain perception, a dilated anus with absent tone and loss of sensation around the perineum. In patients with laceration of these nerve roots the long-term prognosis for a return to normal continence is grave despite having intact deep pain perception.

The take-home message is loss of deep pain perception does not carry the same grave prognosis as it would for spinal cord injury because paralysis of the pelvic limbs is not an expected finding (nerve roots supplying the limbs have already exited the spinal column at this level). A traction injury to this region, however, could cause severe weakness to the pelvic limbs (for example, as is seen following tail pull injuries in cats). Furthermore, lumbosacral disease rarely affects the gait and more frequently results in pain alongside tail carriage, anal tone and perineal sensation abnormalities (which may partly manifest as faecal and/or urinary incontinence). Prognosis in these cases is determined by the presence or absence of perineal sensation, anal tone and tail base sensation.

Main types of disease process that can explain clinical signs

Different spinal cord segments are affected by different diseases. Similarly, the age, breed and speed of onset will also determine the more likely diseases. Each disease process has a typical signalment, onset and progression as well as distribution in the nervous system. For example, a middle-aged dachshund with an acute onset paraparesis is most likely to have an intervertebral disc extrusion while a young beagle with neck pain is most likely to have steroid-responsive meningitis-arteritis. Despite these patterns it is very important not to ignore other less common possibilities.

Before examination, a complete history must be taken to establish the onset and progression of the clinical signs. Did the signs occur acutely, chronically or insidiously? Is the disease progressive? This information alone can refine the list of differential diagnoses by considering the sign-time graph (Figure 3) and also allows a prognosis to be considered.

For example, a dog presenting with slowly progressive clinical signs suggests a degenerative or neoplastic condition and may be given a poorer prognosis. However, it must be emphasised to the owner that without further investigation the likely diagnoses, and associated prognoses, remain speculative. A list of potential causes can be made using the VITAMIN-D acronym (Table 2).

Based on the history, signalment and neurological localisation this list of potential causes can be narrowed further depending on the neurological segments involved (Tables 3 to 6). Therefore, at the end of the examination the clinician should be aware of the potential disease processes involved and hence enable the owner to make an informed decision as to whether further investigation to identify these causes is warranted in light of the likely prognosis.

Summary

These two articles have given a stepwise approach to the spinal patient. On presentation, a full clinical examination should first be performed to ensure the signs are not due to a non-neurological problem. A neurological assessment can then follow to include observation of the gait to ascertain the number of legs that are affected and the nature of the reflexes in the affected legs.

Finally, the severity is established, that is, is movement present or absent? If movement is absent then deep pain perception should be evaluated. Collating this information will allow an accurate neurological localisation to be determined as well as an expected prognosis. Following these simple rules and avoiding the common mistakes ensures spinal cases are managed correctly and appropriately, regardless of costs and facilities.

PANEL 1

Deep pain perception is not the same as the withdrawal reflex

It is important not to confuse the withdrawal reflex with the conscious perception of pain. The withdrawal reflex is useful only in localising lesions whereas deep pain perception is only useful in establishing a lesion’s severity.

If a lesion does not affect the reflex arc then the withdrawal reflex may be intact even if deep pain perception is lost due to a spinal cord lesion situated more cranially. Pain perception is tested by pinching the digits. If there is no conscious response then the nail beds and digits are also tested with haemostats. If there is still no response then forceps are applied to the tibia.

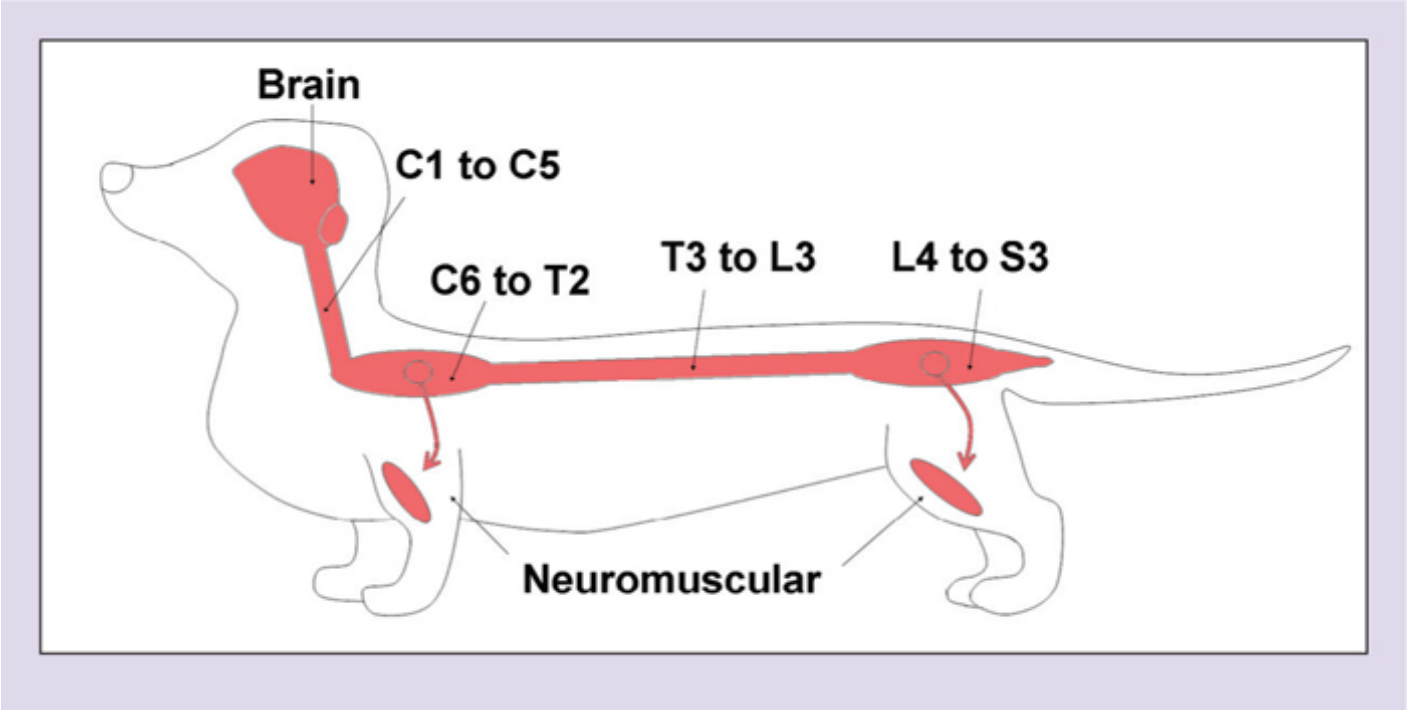

Figure 1. Schematic representation of the components of the nervous system and how they are divided. Lesions at C6 to T2 and L4 to S3 (and the neuromuscular system) cause lower motor neuron signs. Lesions in the region of C1 to C5 and T3 to L3 cause upper motor neuron signs.

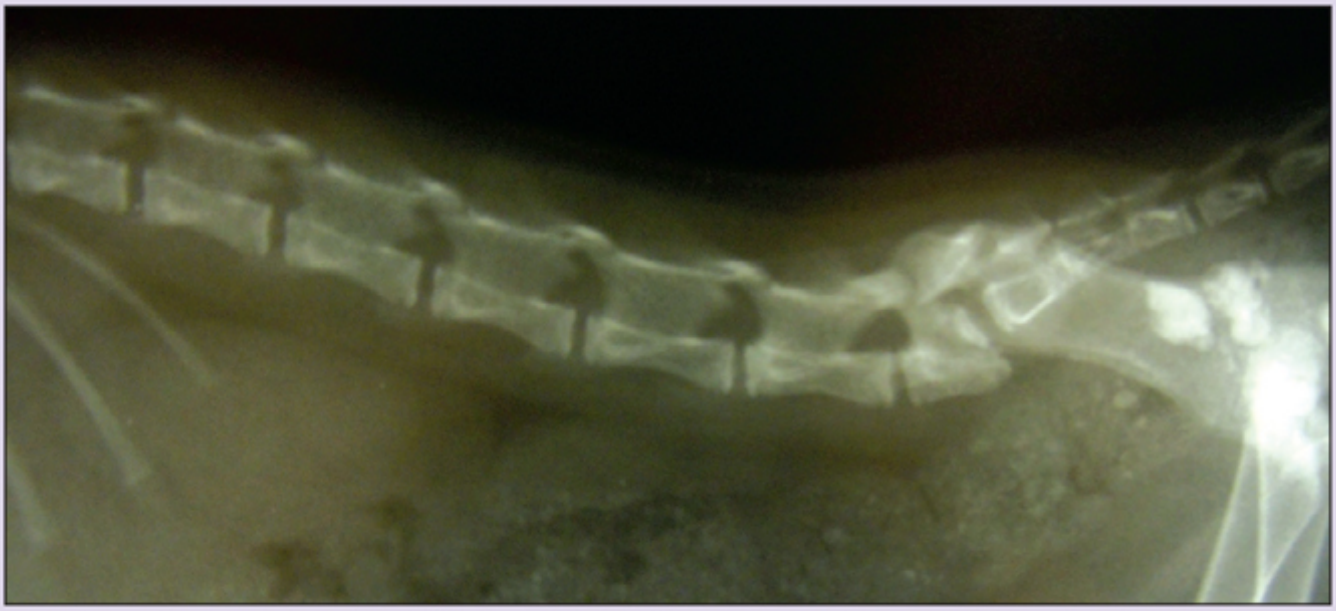

Figure 2. A 12-week-old domestic shorthair cat presented after being accidentally trodden on by the owner. The neurological presentation was a flaccid tail with no deep pain perception, and absent anal tone and perineal sensation. The cat was still able to walk, although it exhibited mild paraparesis with no ataxia, most probably due to transient traction of the nerve roots supplying the pelvic limbs at the time of trauma. A displaced fracture of the lumbosacral region is seen on this lateral radiograph. This was causing instability of the lumbosacral region. The fracture was stabilised and, long term, the cat returned to normal mobility (as would be expected) as well as regaining normal tail function, and urinary and faecal continence.

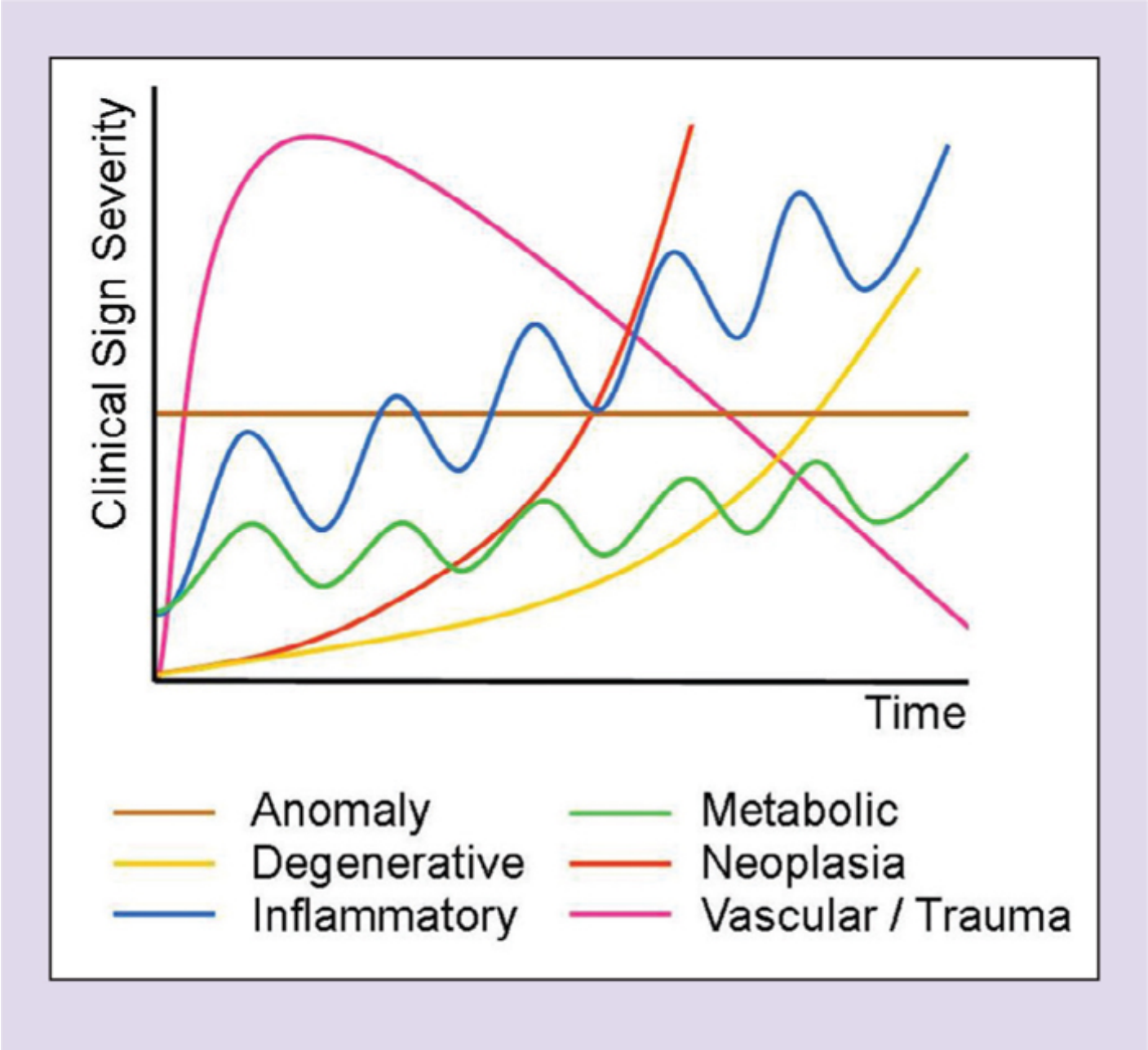

Figure 3. A sign-time graph to show the course of different categories of neurological disease over time.

	Upper motor neuron signs	Lower motor neuron signs
Reflexes	Normal or increased	Decreased or absent
Atrophy	Late and mild (due to disuse)	Rapid and severe (neurogenic)
Tone	Normal or increased	Decreased or absent
Location	Occur with brain, C1 to C5, and T3 to L3 lesions	Occur with C6 to T2, L4 to S3 and neuromuscular lesions

Table 1. Clinical signs that may be expected with upper motor neuron and lower motor neuron signs

	Process	Disease
V	Vascular	Ischaemic myelopathy (such as fibrocartilaginous embolism). Intradural/extradural haemorrhage/ haematoma formation.
I	Inflammatory	Immune-mediated meningomyelitis. Steroid responsive meningitis-arteritis.
	Infectious	Infectious meningitis. Discospondylitis. Empyema.
T	Traumatic	Acute non-compressive nucleus pulposus extrusion (ANNPE). Spinal fractures, instabilities and luxations. Atlanto-axial instabilities.
A	Anomalous	Arachnoid diverticulae (previously termed cysts). Atlanto-axial malformations resulting in instability. Hemivertebra resulting in instability.
M	Metabolic	Not applicable.
I	Idiopathic	Not applicable.
N	Neoplastic	Extradural spinal tumours. Intradural spinal tumours. Intradural extramedullary spinal tumours.
D	Degenerative	Intervertebral disc disease (Hansen Type I and Type II). Lumbosacral stenosis (cauda equina syndrome). Cervical spondylomyelopathy. Degenerative myelopathy (previously termed chronic degenerative radiculomyelopathy – CDRM).

Table 2. Some of the more common diseases affecting the spinal cord of the dog classified according to the VITAMIN-D acronym.

Disease	Typical breeds	Age	Onset	Neurological signs	Spinal pain
Atlantoaxial Instability (Subluxation)	Mainly toy or small; Yorkshire terrier, Poodle	Typically young, <2yrs	Acute or chronic	Common, obvious ataxia and paresis	Present in most cases
Hansen Type I disc disease	Any, mainly small breeds	>2yrs	Acute	Mild or absent	Severe
Hansen Type II disc disease	Any, mainly large breeds	>2yrs	Chronic	Mild to moderate	Mild to moderate
Cervical spondylomyelopathy (bone-associated)	Great Dane, other giant breeds	<4yrs	Usually chronic	Common, obvious ataxia and paresis	Mild
Cervical spondylomyelopathy (disc-associated)	Dobermann, other large breeds	>2yrs	Usually chronic	Common, obvious ataxia and paresis	Mild
Fibrocartilaginous embolism	Any	Any	Acute	Common, can be asymmetrical	None
Spinal trauma	Any	Any	Acute	Common	Common
Steroid responsive meningitis-arteritis	Boxers, beagles, any	Young, <2yrs	Acute or chronic	None	Severe
Acute non-compressive nucleus pulposus extrusion	Any	Any	Acute	Common, can be asymmetrical	None

Table 3. The common diseases affecting the canine cervical spine (C1 to C5 spinal segments) and cervical intumescence (C6 to T2 spinal segments).

Disease	Typical breeds	Age	Onset	Neurological signs	Spinal pain
Degenerative myelopathy	GSD, corgi, boxer mainly large breeds	>5yrs	Chronic (months)	Always, progressive	None
Fibrocartilaginous embolism	Any	Any	Acute	Common, can be asymmetrical	None
Hemivertebra resulting in instability	Pugs, screw-tailed breeds	Young, <1yr	Chronic	Common, can be progressive though may be static	Rare
Hansen Type I disc disease	Any, mainly small breeds	>2y	Acute	Moderate to severe	Moderate to severe
Hansen Type II disc disease	Any, mainly large breeds	>2y	Chronic	Mild to moderate	Mild to moderate
Meningomyelitis	Any	Any	Subacute	Variable, can be asymmetrical	Variable, can wax and wane
Spinal trauma	Any	Any	Acute	Common	Common
Tumours affecting the spinal cord	Any	Any	Acute or Subacute	Common	Common
Acute non-compressive nucleus pulposus extrusion	Any	Any	Acute	Common, can be asymmetrical	None

Table 4. The common diseases affecting the canine thoracolumbar spine (T3 to L3 spinal segments).

Disease	Typical breeds	Age	Onset	Neurological signs	Spinal pain
Degenerative myelopathy	GSD, corgi, boxer mainly large breeds	>5yrs	Chronic (months)	Always, progressive	None
Fibrocartilaginous embolism	Any	Any	Acute	Common, can be asymmetrical	None
Hansen Type I disc disease	Any, mainly small breeds	>2yrs	Acute	Moderate to severe	Moderate to severe
Hansen Type II disc disease	Any, mainly large breeds	>2yrs	Chronic	Mild to moderate	Mild to moderate
Meningomyelitis	Any	Any	Subacute	Variable, can be asymmetrical	Variable, can wax and wane
Spinal trauma	Any	Any	Acute	Common	Common
Tumours affecting the spinal cord	Any	Any	Acute or Subacute	Common	Common
Acute non-compressive nucleus pulposus extrusion	Any	Any	Acute	Common, can be asymmetrical	None

Table 5. The common diseases affecting the canine caudal lumbar spine (L4 to L7 spinal segments).

Disease	Typical breeds	Age	Onset	Neurological signs	Spinal pain
Lumbosacral stenosis (cauda equina syndrome)	Mainly large breeds, GSD most common	>5yrs	Chronic	Typically mild to moderate, may be lame or weak but no ataxia	Often present but hard to elicit
Spinal trauma	Any	Any	Acute	Common	Common
Tumours affecting the spinal cord	Any, usually large breeds	Any	Acute or subacute	Common	Variable
Discospondylitis	Any, usually large breeds	Any	Acute or chronic	Usually not present	Severe pain

Table 6. The common diseases affecting the canine lumbosacral spine (L7 to S3 spinal segments).