



Practical approach of traumatic spinal injuries – how to assess prognosis and which cases can I treat myself

Steven De Decker
DVM, PhD, DipECVN

Royal Veterinary College
Hatfield, United Kingdom
SDeDecker@rvc.ac.uk

Being confronted with animals with traumatic spinal disease can be overwhelming. Although spinal fracture and luxation is most commonly caused by external trauma, signs of spinal disease after trauma will not always be caused by a vertebral fracture or luxation. Other conditions, which do not require spinal surgery, should also be considered. Animals affected by trauma that do not suffer from spinal fracture and luxation, can make a complete and relative quick recovery without expensive diagnostics and treatment. It is therefore important to stay calm and try to recognise those animals that, despite severe clinical signs, might have a good prognosis without the need for referral.

INITIAL ASSESSMENT OF ANIMALS WITH ACUTE SPINAL DISEASE

It is important to follow a calm and rational approach. If an animal is presented with multiple injuries, do not immediately focus on the neurological signs. Animals will usually not die from spinal disease and you should therefore, if present, focus first on more life-threatening abnormalities.

The pathophysiology of acute spinal cord injury is complex and can be divided into primary and secondary spinal cord injury. The primary injury represents immediate physical damage at the time of injury. Examples are spinal cord laceration or rupture of blood vessels. This is followed by the secondary spinal injury, which represents a cascade of biochemical events initiated by the primary injury. The secondary spinal cord injury will cause a continuous and progressive expansion of the area of spinal cord damage. Decreased spinal cord perfusion is considered an important factor in secondary spinal injury. It is therefore important to stimulate spinal blood flow by providing adequate fluid therapy. High doses of corticosteroids have historically been considered to decrease the damaging effects of secondary spinal cord injury. Results from human and veterinary studies do however not demonstrate any benefit and suggest a higher risk of potentially life-threatening complications when administering high doses of corticosteroids in patients with acute spinal disease.

SPINAL SHOCK AND SCHIFF-SHERRINGTON POSTURE

Spinal shock and Schiff-Sherrington posture are two separate and unrelated phenomena. Although they often occur in animals with severe spinal cord dysfunction, they are not necessarily associated with a poor prognosis. Both are anatomical phenomena and can complicate neuro-anatomical localisation.

Spinal shock is characterised by transient flaccid paralysis and loss of spinal reflexes immediately after a thoracolumbar lesion has occurred. Increased muscle tone and spinal reflexes are expected in animals with thoracolumbar lesions and spinal shock can therefore be confused by a lesion affecting the L4-S3 spinal cord segments. The effect is short-lasting and spinal reflexes usually return in less than 48 hours. The neurological examination will reveal an interrupted cutaneous trunci cut-off in most animals with a lesion affecting the T3-L3 spinal cord segments. Paraplegia with absent spinal reflexes in the pelvic limbs, but a thoracolumbar cut-off of the cutaneous trunci reflex is therefore suggestive for a lesion affecting the T3-L3 spinal cord segments with spinal shock.

Schiff-Sherrington posture is characterised by spasticity of the thoracic limbs and paralysis in the pelvic limbs. This posture can occur in animals with a lesion affecting the L1-L5 spinal cord segments and can be confused with a cervical spinal cord problem. The thoracic limbs are however normal and demonstrate a normal gait, normal proprioception and intact spinal reflexes in animals with Schiff-Sherrington posture.

VERTEBRAL FRACTURE AND LUXATION

Vertebral fractures and luxations have a peracute onset of clinical signs and are most often associated with external trauma, such as a road traffic accident or fall from a height. It is important to realise that occurrence of peracute severe neurological deficits after external trauma can also be caused by non-surgical spinal conditions, such as acute noncompressive nucleus pulposus extrusion (ANNPE). This condition will be discussed in the next session. Animals with vertebral fracture and luxation are often obviously painful.

Animals with suspected vertebral fracture and luxation should be handled with great care. You should assume that they have an unstable vertebral column unless proven otherwise. Manipulating them excessively can result in further displacement of vertebral fragments with potential devastating consequences. It is therefore advised that all patients with a suspected vertebral fracture and luxation are placed and secured on a spinal board (or a rigid surface acting as a spinal board). After systemic stabilisation and evaluation of other injuries, a limited neurological assessment is performed. A complete neurological examination will often not be possible, and the patient needs to be handled with care. Most important is to localise the lesion and assess prognosis.

Diagnosis is usually confirmed by spinal radiographs. Computed tomography (CT-scan) is however more sensitive and subtle fractures and mild luxations can be missed on spinal radiographs. Care should therefore be taken when trying to exclude the presence of a vertebral fracture and luxation using radiographs. Although the combination of lateral and ventrodorsal increases your diagnostic accuracy, great care should be taken when manipulating your patient. Orthogonal views should therefore only be taken if necessary or by using a horizontal beam if available.



In contrast to dogs with acute intervertebral disc extrusion, severity of clinical signs will not be the most important factor when considering medical or surgical treatment. The most important factors when considering if surgery is necessary are 1) the presence of vertebral instability and 2) the anatomical region.

Assessment of vertebral instability can be challenging and can be performed in different ways. The most common method is the 3-compartment method in which the vertebral column is divided into a ventral, middle and dorsal compartment. When lesions are present in more than one compartment, you can assume that instability is present. Surgical stabilisation is indicated in these cases. Medical management can be considered when vertebral instability is not present (even if dogs have severe neurological deficits).

Thoracic vertebral fractures can often be treated medically because the ribcage offers good additional stability. Luxations in the cranial cervical and caudal lumbar vertebra column can also be associated with large displacement with relative mild neurological deficits. These locations can also respond favourably to medical management. A fracture of the dens of the axis is common fracture and is also a good candidate for medical management.

Medical management consists of strict rest for 6 weeks in combination with analgesia and supportive care. An external splint can be considered in selected cases. Not every fracture and luxation can however be treated with an external splint and adaptations are necessary for specific locations. A poorly applied external splint can have the opposite effect and decrease the likelihood of recovery.

The prognosis of vertebral fracture and luxation is in general somewhat worse than for acute intervertebral disc extrusion. Prognosis is good as long as animals have intact pain perception and receive appropriate treatment (i.e., surgical stabilisation if indicated). Animals that lost deep pain perception usually do not recover, even if they are treated surgically immediately after the traumatic incident. We do not recommend surgery in these cases. The prognosis is hopeless when loss of pain perception is seen in combination with more than 100% displacement of the vertebral fragments. As mentioned above, a large displacement of fragments can be seen in the cranial cervical and caudal lumbar regions of animals with relative mild neurological deficits.

ACUTE NON-COMPRESSIVE NUCLEUS PULPOSUS EXTRUSION

Acute non-compressive nucleus pulposus extrusion (previously also referred to as traumatic intervertebral disc extrusion, high-velocity low-volume disc extrusion and even Type III intervertebral disc disease) is non-surgical cause of acute paralysis, which can be caused by external trauma..

This condition should not be confused by degenerative intervertebral disc disease (Type I and Type II intervertebral disc disease) and should be considered an unrelated disorder with a completely different pathophysiology. The normal, healthy and non-degenerate intervertebral disc consists of a central part, the nucleus pulposus, and a peripheral part, the anulus fibrosus. The healthy nucleus pulposus has a very high-water content and has a liquid gelatinous consistency.

When for some reason the pressure in the intervertebral disc becomes excessively high, the (healthy) intervertebral disc can suddenly rupture and the liquid nucleus pulposus can extrude with considerable force into the vertebral canal. This can cause substantial spinal cord contusion and peracute onset of severe clinical signs. The liquid nucleus pulposus will however dissipate quickly and spinal cord compression will therefore NOT be present. Surgery is therefore not indicated in this condition.

Clinical signs can occur after external trauma such as a road traffic accident or during excessive activity, such as running for a ball. In dogs, ANNPE is associated with excessive exercise in 60% of cases and by external trauma in 40% of cases. In cats, ANNPE is even more often seen after external trauma. ANNPE is therefore an important differential diagnosis for vertebral fracture and luxation. It is easy to understand that the treatment and prognosis for both conditions is very different. Animals affected ANNPE experience a peracute onset of clinical signs and can be mildly painful in the first 24 hours after the onset of clinical signs. Pain should however not be present more than 24 hours after onset of clinical signs. Clinical signs can be obviously lateralised (one limb much worse affected than the opposite) and clinical signs should not be progressive after 24 hours. Treatment consists of intense physiotherapy and hydrotherapy. Strict cage rest is not necessary. Prognosis is good if deep pain perception is still present. Initial improvement is most often seen a few days after making a diagnosis.