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Veterinary Made Easy!
Mini Series

Session 2: Spinal and neuromuscular disorders

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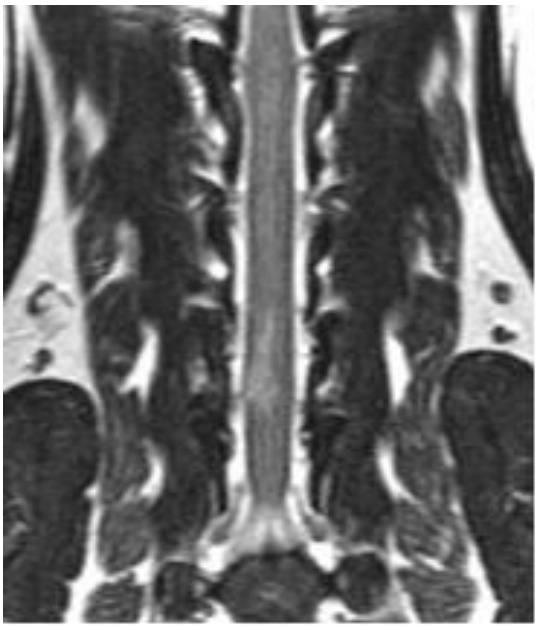

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Spinal and neuromuscular disorders
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The learning outcomes of the second module of the CPD solution Neurology mini-series are become better able at:
Develop a systematic clinical and diagnostic approach to the patient with spinal or neuromuscular neurologic signs
Recognise clinical signs of motor and/ or sensory neuropathy, myopathy or junctionopaty
Recognise clinical signs of spinal shock
Diagnose, treat and predict prognosis in dogs and cats with spinal or neuromuscular disease
Neurologic signs of spinal cord disease are detailed in the table below (subdivided by spinal cord regions, not vertebral column regions). Mentation is normal. Behaviour is normal unless affected by spinal hyperalgesia

	C1-C5	C6-T2	T3-L3	L4-S3
Posture	Normal or guarded neck posture, may have wide-base stance in all four limbs, lateral recumbence with severe lesions	Normal; may have low head carriage, guarded neck posture, wide-base stance (esp in PLs), lateral recumbence with severe lesions	Normal or kyphotic posture with painful lesions, may have wide-base stance in PLs	Normal or kyphotic posture with painful lesions
Gait	Proprioceptive ataxia, typically TL = PL), spastic (long-strided) tetraparesis/plegia Ipsilateral hemiparesis/plegia	Proprioceptive ataxia, typically PL > TL), tetraparesis/plegia Ipsilateral hemiparesis/plegia	Normal TLs Proprioceptive ataxia in the PLs Paraparesis or paraplegia	Paraparesis or paraplegia (with spinal cord lesions) Mild proprioceptive ataxia PLs (with spinal cord lesions) Lesions affecting the cauda equina nerves (L6-L7-S1 vertebrae) will only cause paraparesis without ataxia
Cranial nerves	Typically normal May see Horner's syndrome with severe or intramedullary lesions	Typically normal May see Horner's syndrome with severe or intramedullary lesions	Normal	Normal
Spinal reflexes	Normal Hyperreflexia all limbs	Hyporeflexia or absent reflexes TLs Normal to hyperreflexia in PLs	Normal Hyperreflexia PLs (transient hyporeflexia with spinal shock)	Decreased to absent reflexes in the PLs May see pseudohyperreflexia patellar reflex with sciatic lesions

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Spinal hyperesthesia	None or pain on palpation or movements	None or pain on palpation or movements	None or pain on palpation or movements	None or pain on palpation or movements
Nociception	Normal Tetraplegic animals may show decreased or absent nociception	Normal Tetraplegic animals may show decreased or absent nociception	Normal Mono/Paraplegic animals may show decreased or absent nociception caudally to the lesion	Normal Mono/Paraplegic animals may show decreased or absent nociception caudally to the lesion
Micturition	Usually normal May have detrusor areflexia-sphincter hypertonia	Usually normal May have detrusor areflexia-sphincter hypertonia	Plegic patients may have Detrusor areflexia-sphincter hypertonia	Normal or detrusor areflexia-sphincter hypotonia

Common spinal disorders include:
Fibrocartilaginous embolic myelopathy (FCEM)
Acute non-compressive nucleus pulposus extrusion (ANNPE)
Intervertebral disc herniation (IVDH)

Definitions and Pathophysiology

Fibrocartilaginous embolic myelopathy (FCEM) is a vascular disease of the spinal cord caused by embolization of spinal vasculature with fibrocartilaginous material histologically and histochemically identical to the nucleus pulposus (NP) of the intervertebral disc resulting in ischemic necrosis of dependent regions of spinal cord parenchyma. Various theories have been hypothesised to explain how the fibrocartilaginous material can enter the spinal vasculature.
Traumatic disc extrusions (TDE) refers to extrusion of either degenerated or non-degenerated intervertebral disc material following trauma to the spinal region. Occasionally, the extruded disc material (degenerated or not) can result in laceration of the dura mater and in some cases penetration of the spinal cord parenchyma.
Acute non-compressive nucleus pulposus extrusion (ANNPE) refers to the extrusion of hydrated nucleus pulposus (HNP) due to a sudden increase in intradiscal pressure during vigorous exercise (such as running and jumping) or trauma. The HNP herniates through a tear in the annulus fibrosus, contuses the spinal cord and then dissipates within the epidural space causing minimal to no spinal cord compression. This condition has also been named traumatic disc prolapse, dorsolateral intervertebral disc "explosion", high-velocity–low volume disc extrusion and Hansen type III intervertebral disc disease. However, Hansen's terminology only included two types of intervertebral disc degeneration (I and II), and Type 3 extrusions were originally described by Funquist as extension of disc material "like a carpet over several vertebrae."
Extrusion of HNP can occasionally result in spinal cord compression.
Intervertebral disc herniation (IVDH) commonly refers to the herniation of degenerated intervertebral disc (IVD) resulting in spinal cord compression. IVDH have been classified as IVD extrusion (IVDE) associated with Hansen type I IVD degeneration (chondroid metaplasia) and IVD protrusion (IVDP) associated with Hansen type II IVD degeneration (fibrous metaplasia). Hansen type I IVD degeneration (chondroid metaplasia) is characterized by a loss of glycosaminoglycans, an increase in collagen content, and a decrease in water content, resulting in a general loss of the hydroelastic properties of the disc and its ability to withstand pressure. Normal physical activity can

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result in extrusion of the degenerated and mineralised NP through the ruptured annulus fibrosus (AF), with subsequent spinal cord contusion and compression. Contusion of the spinal cord causes primary mechanical damage and initiates a chain of biochemical events that result in neuronal and glial cell necrosis and apoptosis (secondary injury) and incites and inflammatory response. The IVD material can extrude in close proximity to the affected disc space or it can disperse over several vertebral segments with or without associated haemorrhage. The extruded IVD material can be located ventrally, laterally and/ or dorsally to the spinal cord. Hansen type II IVD degeneration (fibrous metaplasia) leads to bulging of the NP within the weakened AF and ultimately dorsal IVD protrusion resulting in ventral or ventrolateral spinal cord compression. The IVD protrusion tends to occur gradually resulting in progressive chronic spinal cord injury (necrosis, and apoptosis of glial cells and neurons, ischemia, and demyelination) which can lead to spinal cord atrophy.

Signalment

Any breed of dog and cat of any age and gender can be affected by any of the spinal disorders described above.
FCEM has been reported most commonly in large and giant breed dogs of non-chondrodystrophic breeds. The reported male to female ratio in dogs ranges from 1:1 to approximately 2.5:1 in different studies. The age at diagnosis in dogs ranges from 2 months to 13 years and 5 months, with a median of 4 to 6 years in the majority of studies.
ANNPE can occur in any canine breed. Median age at diagnosis in dogs has been reported as 6.7 years (range, 2.1 to 10.7 years) and males were over-represented.
IVDEs occur most commonly in chondrodystrophic breed dogs however non-chondrodystrophic large breed dogs can also be affected. Dachshunds have been reported to be 12.6 times more likely to develop IVDE than other breeds, followed by the Pekingese, beagle and cocker spaniel, which are reportedly 10.3, 6.4, and 2.6 times more likely to develop IVDE, respectively, than other breeds. Other breeds frequently reported with IVDE include the French bulldog, shih-tzu, Lhasa apso, Jack Russell terrier, bichon frise, Maltese, and miniature poodle. The large-breed dogs most commonly reported to develop IVDE include mixed breeds, German shepherd dogs, Labrador retrievers, rottweilers, dalmatians, and Doberman pinschers.
IVDPs occur most commonly in non-chondrodystrophic dogs, although chondrodystrophic breed dogs can also be affected.
IVDH rarely occurs before 2 years of age; reaches its greatest incidence between 3 and 7 years of age in chondrodystrophic dogs and generally develops in nonchondrodystrophic dogs at a mean age of 6 to 8 years. A consistent gender predilection for IVDH has not been reported.

The clinical presentation and diagnostic imaging findings can help differentiating among the spinal disorders described above.

Clinical presentation

The clinical presentation findings that can help differentiating among the spinal disorders described above have been summarised in this table. Type and severity of neurological signs will depend on the site and degree of spinal cord injury.

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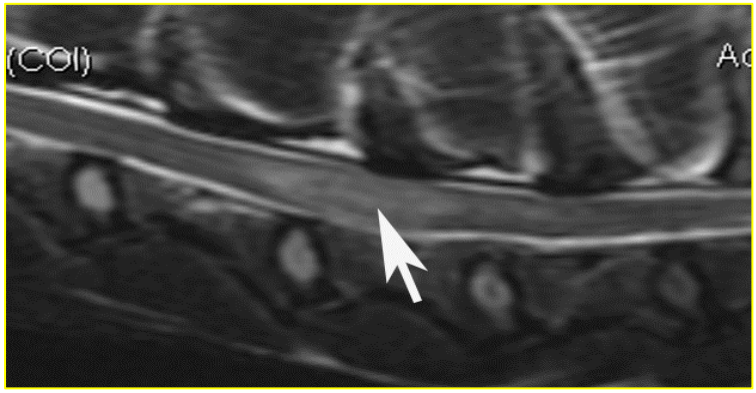
	FCEM	ANNPE	IVDE	IVDP
Onset of neurological signs	Peracute, frequently associate with physical activity	Peracute, frequently associate with trauma or vigorous physical activity	Peracute to subacute, sometimes history of previous episodes of spinal pain +/- neuro deficits	Subacute to chronic
Progression of neurological signs	Non progressive after 24-48 hours of onset	Non progressive after 24-48 hours of onset	Progressive deterioration can occur	Progressive deterioration can occur
Lateralization of neurological signs	Common (53-87%) and marked	Common (62%) and marked	Can occur, but very rarely as marked as for FCEM and ANNPE	Can occur, but not as marked as for FCEM and ANNPE
Spinal hyperalgesia	Absent 24-48 hours after onset	Common	Common	Can occur

Diagnostic imaging

MRI is the diagnostic imaging modality of choice in supporting the antemortem diagnosis of FCEM and ANNPE.
The MRI features suggestive of FCEM include a focal, relatively sharply-demarcated intramedullary and often lateralized lesion (edematous infarcted tissue), predominantly involving the grey matter that is hyperintense to normal grey matter on T2-weighted fast spin echo (FSE) and FLAIR images and iso- or hypointense to normal grey matter on T1-weighted FSE images. Postcontrast T1-weighted FSE images may show mild and heterogeneous enhancement of the affected area, generally on the fifth to seventh day of disease.
MRI imaging performed 24 to 72 hours after onset of neurologic signs may reveal no intraparenchymal signal intensity changes in dogs with FCEM. Diffusion weighted (DW) MRI may increase the sensitivity and specificity for diagnosis of spinal cord infarction in the early stages of the disorder. However, DW MRI of the spine is technically challenging in small animals.
Figure 1a and 1b (T2W MRI). FCEM in the cervical spine of a 5 year old border collie with peracute onset right-sided severe hemiparesis.



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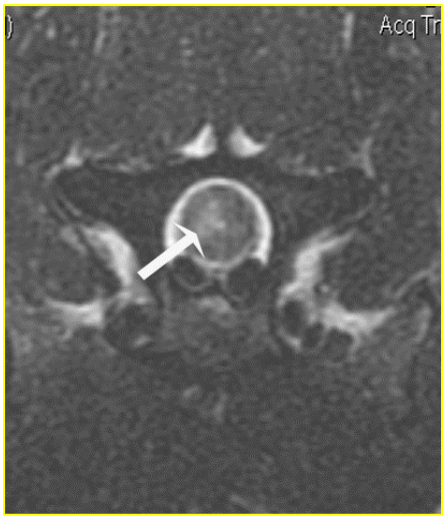
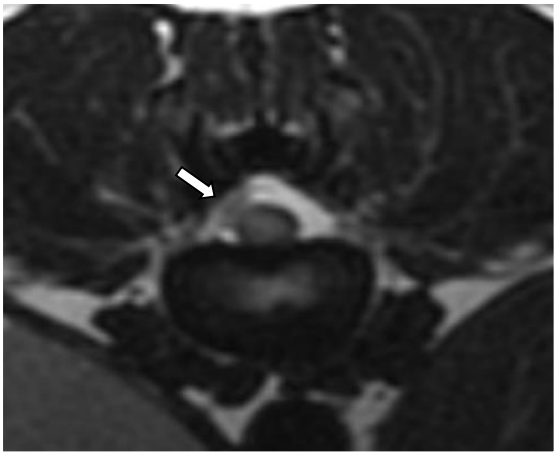


The MRI findings of ANNPE include a focal area of T2W hyperintensity within the spinal cord overlying a narrowed intervertebral disc space, with absent or minimal spinal cord compression. There is decreased volume and signal intensity of the affected nucleus pulposus on T2-weighted images. Extraneous material or signal change may be evident in the epidural space dorsally.
Figure 2 (T2W MRI). ANNPE in a 5 year old, male, boxer with peracute onset paraparesis (paresis was marked on the right pelvic limb and mild on the left pelvic limb). Note the signal change (arrow) in the epidural space above the affected IVD and the spinal cord intramedullary hyperintensity.



In dogs with traumatic disc extrusion resulting in laceration of the dura mater, myelography or CT-myelography may show extradural leakage of iodinated contrast medium or focal accumulation of contrast within the spinal cord. Traction of the cervical spine may result in penetration of myelographic contrast medium through a defect in the dura and annulus. MRI allows visualization of disc material herniated within the spinal cord parenchyma and associated spinal cord edema and/ or hemorrhage, as well as narrowing of the underlying intervertebral disc space. A communicating tract extending from the intervertebral disc into the spinal cord parenchyma can sometimes be seen.

Myelography, CT (for mineralised IVDE), CT myelography and MRI are imaging modality for diagnosing IVDH. The reported accuracy of myelography for IVDH localization ranges from 72% to 97% and its accuracy for lateralization of the lesion ranges from 53% to 100%. Combined lateral, ventrodorsal and oblique projections maximise accuracy of IVDE site and side identification on myelography. One study reported that the agreement of myelography, CT, and helical CT with surgical findings was 94.7%, 100%, and 94.7%, respectively, for lesion localization and 78.9%, 87.4%, and 85.3%, respectively, for lesion localization and lateralization. Similarly another study reported an accuracy of CT and myelography to determine lateralization of the lesion of 95.6% and 91.7%,

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respectively. MRI has been reported to be more accurate than myelography for determining the site and side of IVDE. Complete agreement between MRI and surgical findings has been reported with regard to the affected thoracolumbar IVD and lesion lateralization in two studies.
MRI findigns have been shown to have a prognostic value in dogs with FCEM, ANNPE, and IVDE.

Figure 3 (T2W MRI). IVDE at L2-3 in a 4 year old, male neutered, dachshund with severe thoracolumbar hyperalgesia and mild proprioceptive dysfunction of 2 days duration.

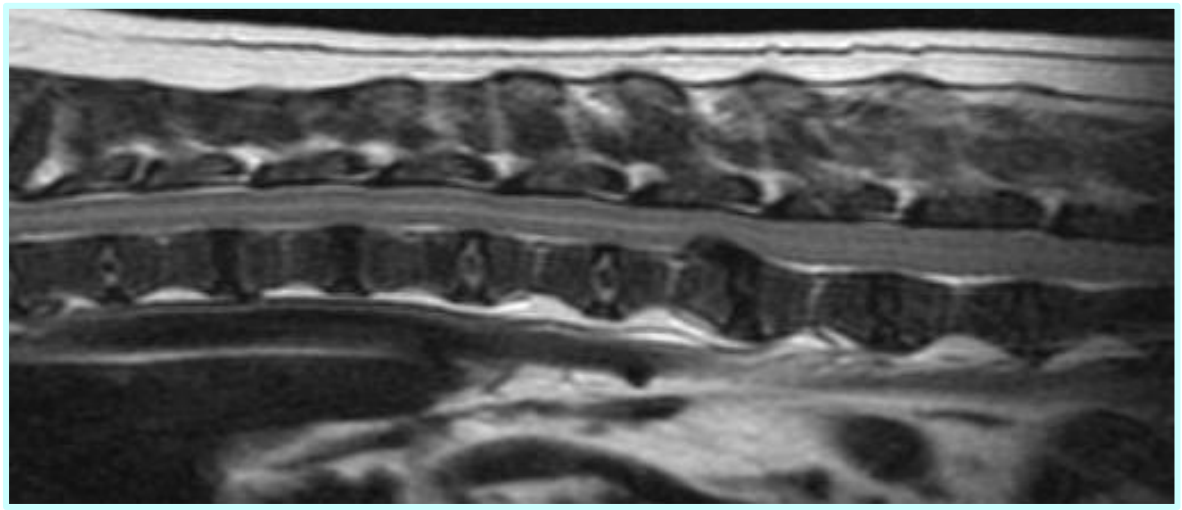
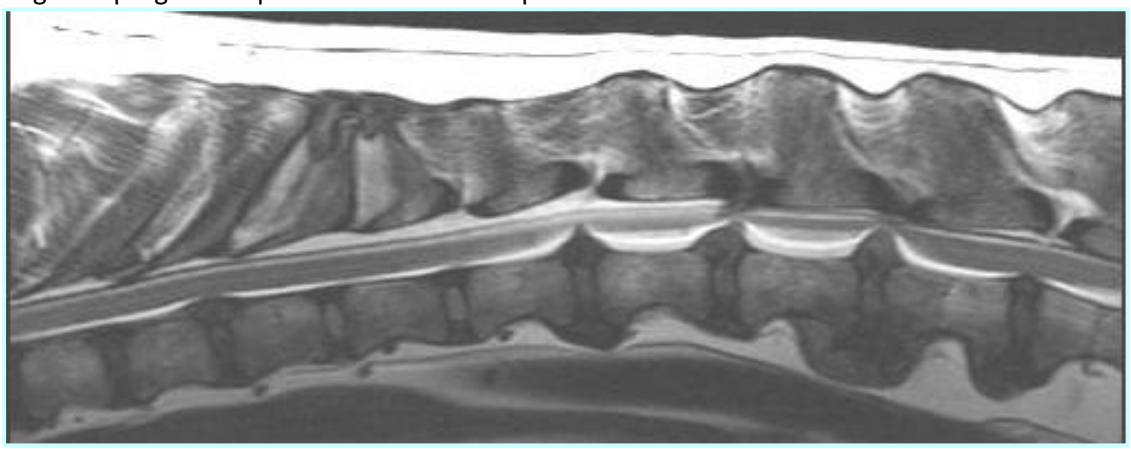


Figure 4 (T2W MRI). Multiple IVDP from T12 to L2 in a 7 year old, male neutered, German shepherd dog with progressive pelvic limb ataxia and paresis.



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Neuromuscular disorders

Neurologic signs of neuromuscular disease include:
Normal mental status and behaviour.
Clinical signs vary depending on the lesion location, e.g. cranial or spinal nerve, sensory or motor component, neuromuscular junction, or muscle.
Peripheral nerve dysfunction generally is characterized by flaccid paresis or paralysis, decreased to absent postural reactions and spinal reflexes, and muscle hypotonia and atrophy.
Cranial nerve involvement results in various clinical signs depending on the affected CN (see cranial nerve examination).
Nociception may be decreased or absent in the dermatome of the affected nerve. Hyperalgesia or paraesthesia can occur. Some neuropathies are exclusively or primarily characterized by motor dysfunction, others by sensory dysfunction and some by a combination of both motor and sensory dysfunction. With mononeuropathies, deficits are restricted to regions innervated by the affected nerve. Polyneuropathies may affect multiple spinal or cranial nerves or both.
Muscle disorders are often characterized by weakness, fatigability and stiff, stilted gait. A postural tremor may be observed. Masticatory muscles, muscles of facial expression or pharyngeal and laryngeal muscles may also be involved. Regurgitation can occur with oesophageal skeletal muscle involvement. Muscle tone may be normal, increased or decreased. Spinal reflexes are usually normal, but may be weak or fatigable, or difficult to evoke in animals with severe muscle atrophy and fibrosis. Palpation may reveal muscle atrophy or less commonly hypertrophy, and sometimes hyperaesthesia. Sensations (including proprioception and nociception) are normal.
Animals with *neuromuscular junction disorders* may have a normal neurologic examination following rest, however various degrees and duration of exercise often results in fatigability and stiff, stilted gait which improves or resolves following rest. Postural reactions are normal, although profound weakness may affect performance. Spinal reflexes are usually normal but may be fatigable or weak. Muscles of facial expression, pharyngeal, laryngeal, oesophageal skeletal muscles may also be involved resulting in facial paresis, dysphonia, dysphagia and regurgitation.

An extensive presentation of neuro muscular disorders in dogs and cats can be found in neurology textbooks. Clinical features of selected neuromuscular disorders are presented below. Further details can be found in the recommended references.

Myasthenia gravis

Myasthenia gravis (MG) is a disorder of neuromuscular transmission in which muscle weakness results from an autoantibody mediated depletion of acetylcholine receptors (AChRs) at the neuromuscular junction. MG can be acquired or hereditary.
Congenital myasthenic syndromes are hereditary disorders of neuromuscular transmission resulting in structural or functional defects of the neuromuscular junction.
Acquired MG is an autoimmune disease in which antibodies are formed against the nicotinic ACh receptors, resulting in decreased numbers of receptors on the postsynaptic sarcolemmal surface, decrease in normal neuromuscular junction transmission and skeletal muscle weakness. While both dogs and cats can be affected by acquired MG, there are distinct differences in clinical presentations and frequency of spontaneous remission.
In dogs and cats, a bimodal age of onset has been identified. Cats are typically affected between 2 and
3 yrs of age or between the ages of 9 and 10 yrs. In dogs, this bimodal age of onset occurs in young dogs between 4mos and 4 yrs (average, 3 yrs) and in older dogs between 9 yrs and 13 yrs of age (average, 10 yrs).
Acquired MG can present in the dog and cat as one of three different clinical syndromes:

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focal MG (weakness of esophageal, pharyngeal, laryngeal, and facial muscles in the absence of generalized appendicular muscle weakness)
generalized MG
acute fulminating MG
Acquired MG has been associated with other diseases (including hypothyroidism, thymomas, thymic cysts, nonepitheliotropic cutaneous lymphoma, cholangiocellular carcinoma, anal sac adenocarcinoma, osteogenic sarcoma, oral sarcoma, and methimazole therapy in cats) and these should be taken into consideration when planning the diagnostic evaluation of a case and determining the prognosis.
In dogs and cats with MG, the neurologic examination is usually normal when performed prior to the induction of exercise.
Presenting clinical signs in cases with:
Focal MG include:
regurgitation secondary to megaesophagus
dysphagia due to pharyngeal muscle weakness
dropped jaw
diminished or absent palpebral reflexes
voice changes (dysphonia) due to laryngeal and/or palatal muscle weakness.
.
Generalized form of MG is characterized by:
appendicular muscle weakness that may be induced or exacerbated by exercise (usually a few minutes) but following rest the animal regains muscle strength and can return to activity for a short period before a relapse of the muscular weakness
the clinical signs described in the focal MG form
Retrospective studies reported that generalized weakness with or without megaesophagus was present in 57–64% of dogs and 80% of cats with acquired MG.
Cats may frequently demonstrate cervical ventroflexion as a clinical sign of generalized weakness.

Acute fulminating MG is a severe and rapidly progressing form of generalized MG. Affected animals are often unable to sand up and walk. Weakness of the skeletal muscles eventually affects the intercostal muscles and/or diaphragm, at which stage affected animals demonstrate severe respiratory distress. The prognosis for fulminating MG is poor.

Acute idiopathic polyradiculoneuritis

Acute idiopathic polyradiculoneuritis is an idiopathic inflammatory disorder primarily involving both axons and myelin of ventral nerve roots in dogs. It is probably the most common polyneuropathy in this species. Although much less common, an analogous polyneuropathy has been described in cats. A similar disorder in people is Guillain–Barr´e syndrome (GBS).
Although the pathogenesis is uncertain, an autoimmune process is suspected in canine acute idiopathic polyradiculoneuritis. It is possible that an infectious process and secondary molecular mimicry or the production of autoantibodies against axolemmal components is the underlying cause for this disease. In humans, GBS is most commonly temporally associated with gastrointestinal Campylobacter jejuni infections.
The typical clinical presentation for canine acute idiopathic polyradiculoneuritis involves a rapidly developing LMN paresis/plegia, usually initially in the pelvic limbs, and subsequently involving the thoracic limbs. Most affected animals will progress to being either non-ambulatory tetraparetic or tetraplegic within 3-10 days of the initial onset of clinical signs. Loss or change of voice is common, and some patients will also exhibit facial weakness. Spinal reflexes are typically absent Life-threatening respiratory paralysis can develop, especially in the more rapidly developing cases.

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Skeletal muscles are hypotonic, and neurogenic atrophy develops quickly in recumbent patients. Proprioceptive placing reactions are normal in those animals that still have enough motor ability to perform the efferent component of these tests. The ability to eat and drink, urinate and defecate, is maintained although manual support is required. Pain sensation also remains intact. In fact, these animals often seem hyperesthetic upon limb manipulation, which may reflect the inflammatory nature of the disease.
Diagnosis is typically based upon characteristic clinical signs of a rapidly progressive motor polyneuropathy. Other potential causes of polyneuropathy should be ruled out. Electrodiagnostic tests and muscle/nerve biopsies will also support the diagnosis of a polyneuropathy. Protein levels may be increased on CSF analysis.
There is no specific therapy for this disease. Nursing care, physical therapy, and proper nutrition are essential for recovery. Glucocorticoids have been suggested, but there is no evidence of efficacy. IV immunoglobulin, although expensive, have shown promising results to achieve and accelerate recovery.
The prognosis for full recovery is often favourable, but it is typically prolonged, usually taking several weeks to several months. Some patients develop life-threatening respiratory paresis/paralysis in the acute phase of the disease (usually those dogs whose signs progress rapidly over 72 hrs) and may need to be mechanically ventilated.

Periodic hypokalaemic polymyopathy

Periodic hypokalaemic polymyopathy is a genetic disease of Burmese cats and related breeds such as the Bombay, Tonkinese and Tiffanie that has been reported in Australasia, Europe and South Africa. Affected cats usually present with signs of muscle weakness and muscle pain during the first year of life. Characteristic clinical features include ventroflexion of the head and neck, however not all cats display these signs. Weakness is generally periodic or episodic, but occasionally it is persistent. Signs in affected cats have been reported to wax and wane, possibly in response to changes in dietary factors and stress. During an episode of muscle weakness, serum potassium concentration is generally but not always low. Therefore serial serum potassium concentration determinations, testing of serum creatine kinase activity and exclusion of other potential causes of muscle disease in cats (including muscular dystrophies, Toxoplasma myositis, immune-mediated polymyositis, and organophosphorus intoxication) were needed to diagnose this condition until the genetic test become commercially available. Recent molecular genetics research has identified a single nonsense mutation in the gene (WNK4) coding for lysine-deficient 4 protein kinase, an enzyme present primarily in the distal nephron. The underlying pathophysiology seems related to a potassium wasting nephropathy, as lysine-deficient 4 protein kinase is involved in complex sodium/potassium exchange mechanisms in the kidney. The diagnosis of Burmese (and related breeds) hypokalaemia is now straightforward, as an inexpensive PCR test can identify affected homozygous individuals, as well as carriers. The elimination of this condition from the Burmese breed, and also from pedigree cats infused with Burmese lines, such as the Bombay, Tonkinese and Tiffanie breeds, should therefore be possible (Malik 2015).

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