SMALL ANIMAL CASE STUDIES IN CLINICAL NEUROLOGY

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ANATOMIC DIAGNOSIS

Brain: Prosencephalon, Pons-Medulla, Cerebellum
Cranial nerves
Spinal Cord: C1-C5: UMN/GP all four limbs
C6-T2: LMN thoracic limbs, UMN/GP pelvic limbs
T3-L3: UMN/GP pelvic limbs
L4-S1: LMN pelvic limbs
S1-Cd: LMN tail, anus, perineum, excretions
Nerves: Radial, Femoral, Sciatic

GAIR PATTERN RECOGNITION

Gait is dependent on lower motor neurons (LMN), upper motor neurons (UMN) and general proprioceptive neurons (GP).

LOWER MOTOR NEURON (LMN)

LMNs innervate muscle cells and are located in the central pattern generators (CPG) for the limbs along with interneurons. For the thoracic limbs the CPG is located in the grey matter of the cervical intumescence: C6-T2 spinal cord segments. For the pelvic limbs, the CPG is located in the grey matter of the lumbosacral intumescence: L4-S2 spinal cord segments.

LOWER MOTOR NEURON (LMN) SIGNS:

In lower motor neuron disease, there is a **deficiency in the ability to support weight** which results in a short stride but no ataxia. The patient walks with a "lame" gait. The patient knows where its paws are located. Postural reactions are normal as long as the limb can be moved. The paresis is hypotonic (flaccid) with no ataxia

DIFFUSE LOWER MOTOR NEURON DISEASE (LMN): EXAMPLES

Polyradiculoneuritis
Tick Paralysis
Botulism
Myasthenia gravis

POLYRADICULONEURITIS: MOLECULAR MIMICRY

Molecular mimicry is a presumed basis for the immune-mediated inflammation in polyradiculoneuritis. The patient is exposed to an antigen that is similar to a component of either the myelin or the axon within its nerves. This would include

such components as galactocerebrosides or glycolipids. Antibodies made against the exogenous antigen attack and destroy the patient's antigen (axon and/or myelin). In humans, this polyneuritis is called the Landry-Guillain-Barre (LBG) disease. It has occurred in patients exposed to *Campylobacter jejuni*, *Mycoplasma pneumoniae*, Cytomegalovirus, Epstein Barr virus and the Zika virus. In dogs, especially coonhounds, the raccoon saliva is the presumptive source of the common antigen. The disease can be reproduced only in recovered dogs with the subcutaneous injection of raccoon saliva. This suggests that for this disease to develop, in addition to exposure to the appropriate antigen there needs to be some alteration of the patent's immune system as well. Be aware that this polyradiculoneuritis can occur in dogs without exposure to a raccoon where the nature of the common antigen is unknown.

UPPER MOTOR NEURON

The upper motor neuron cell bodies are located in the reticular formation of the brainstem especially the pons and medulla. Their axons course caudally in the spinal cord white matter to facilitate or inhibit the LMN in the CPG.
Deficit results in **hypertonic paresis**.

GENERAL PROPRIOCEPTION

General proprioceptive neurons innervate joint capsules and neuromuscular spindles. Cell bodies are located in spinal ganglia. Their axons synapse in the CPG. Axons of these second neurons project cranially in spinal cord tracts to synapse in the cerebellum and pontomedullary nuclei. The latter neurons project to the cerebrum via the thalamus.
Deficit in this system results in **ataxia**.

UPPER MOTOR NEURON (UMN) AND GENERAL PROPRIOCEPTIVE (GP) SYSTEM SIGNS

Lesions of spinal cord white matter affect both the upper motor neuron (UMN) and general proprioceptive (GP) systems together. The clinical signs reflect the involvement of both systems. These include a delay in the onset of limb movement and a prolonged stride with the joints in extension. This gives an overreaching appearance to the gait which is easier to appreciate in the thoracic limbs. Limb placement may be abnormal to either side. Postural reactions will be delayed to absent.

UMN and GP tracts are adjacent in the caudal brainstem, and spinal cord. It is not possible to clearly differentiate between UMN hypertonic paresis and GP ataxia and this is not necessary. The gait pattern will reflect the combined loss of these two systems.

When a lesion affects all 3 systems (LMN, UMN, GP), the clinical signs will reflect the loss of the LMN.

CANINE C1-C5 ANATOMIC DIAGNOSIS- PROGRESSIVE COURSE

DIFFERENTIAL DIAGNOSIS

Compressive myelopathy:

Vertebral Canal Stenosis:

Malformation/Malarticulation: congenital, acquired.

OCD may be involved in the development of the vertebral stenosis and malformation of the articular processes.

Malarticulation leads to degenerative joint disease that involves both the intervertebral disks as well as the articular processes.

C2-C3 meningeal fibrosis often accompanied by syringohydromyelia. Large breed dogs, cause unknow

Diskospondylitis

Intervertebral disc extrusion

Neoplasia extraparenchymal

Myelitis

Parenchymal neoplasm

RADIAL NERVE - FEMORAL NERVE

Loss of function of these two nerves results in loss of weight support in the thoracic limb (radial nerve) or pelvic limb (femoral nerve). Protraction of these limbs is normal but when the limb is placed on the bearing surface and expected to support weight, it collapses due to inability to extend the elbow or stifle, respectively. This results in a short stride in the affected limb as the patient depends on the opposite limb for its weight support. The patient will appear to walk "lame." Nociception is compromised on the cranial surface of the antebrachium or dorsal surface of the forepaw for the radial nerve and the medial surface of the crus and hind paw for the femoral nerve where the skin is innervated by its saphenous nerve branch .

SCIATIC NERVE

Sciatic nerve dysfunction is characterized by the preservation of weight support and a protraction of the limb which is uninhibited due to the loss of hip extensor muscle function. Thus, hip flexion is brisk and the patient may exhibit a skipping form of gait. Stifle flexion is reduced and both extension and flexion of the tarsus and digits is compromised. This is evident when the patient bears weight and a passive overflexion of the tarsus ("a dropped hock") is exhibited (tibial nerve) and occasionally the patient will bear weight on the dorsal surface of the paw due to the loss of tarsal flexion and digital extensor function (fibular –peroneal nerve). Nociception will be compromised on the cranial, lateral and caudal surfaces of the crus and dorsal, lateral and plantar surfaces of the pelvic limb paw. These surface areas are innervated by the tibial and fibular branches of the sciatic nerve.

Be aware that rupture of any component of the common calcanean tendon can result in a " dropped hock" posture identical to that caused by a sciatic/tibial nerve dysfunction. However, this lesion has no effect on hip flexion.This common calcanean tendon includes the gastrocnemius tendon, the tendon of the superficial digital flexor and an extension of the tendons of the biceps femoris laterally and the semitendinosus and gracilis medially. The specific tendon rupture can be defined with MR imaging.

INHERITED POLYNEUROPATHY IN LEONBERGERS – D. SHELTON
The Leonberger dog is a product of breeding Newfoundland, Great Pyrennes and Saint Bernard dogs.
The polyneuropathy onset of clinical signs is 1 to 3 years with inspiratory dyspnea, overflexed tarsus ("dropped hocks") and walking with overflexion of the hips. Be aware that the inspiratory dyspnea may precede the evidence of a gait disorder which may influence your therapy. The gait disorder progresses over months to years to tetraparesis. This is inherited as an autosomal recessive gene but the specific genetic mutation is still being studied. This disorder is more common in males.

AORTIC VASCULAR COMPROMISE IN CATS
Caudal aortic thromboembolism only affects the blood supply to muscles and nerves of the pelvic limbs distal to the mid-thigh level. The blood supply to the lumbosacrocaudal spinal cord, tail, perineum and excretory orifices is unaffected due to the blood supply from the segmental lumbar arteries that branch from the abdominal aorta and this blood flow can bypass the aortic compromise. The spinal cord is unaffected. Muscle tone is normal in the muscles that control hip joint position and movement as well as the tail and excretory orifices. These cats can move rapidly along the ground by flexing their hips. This unique gait is characteristic of this disorder.

When cats have their abdomen compressed by the tire of a vehicle, the prolonged spasm of the lumbar arteries or possibly their thrombosis causes a poliomyelomalacia of the lumbar, sacral and caudal spinal cord segments. These cats have no muscle function or tone in their abdomen, pelvic limbs, tail, perineum or excretory orifices due to the necrosis of the spinal cord ventral grey columns. These same regions are also usually analgesic due to the necrosis of the dorsal grey columns. This same spinal cord lesion and clinical signs will result when the aorta is ligated in the region of the renal arteries.

INFRASPINATUS CONTRACTURE
This occurs primarily in hunting dogs that are in the field and thought to step in a depression or hole that causes them to overextend one thoracic limb. The distal end of the spine of the scapula is adjacent to where the tendon arises from the infraspinatus muscle. Overextension of this joint forces the distal end of the scapula into the infraspinatus muscle-tendon junction causing injury to that muscle. The affected dog walks lame for a few days, followed by a period of normal gait. After a few weeks to months, this dog will develop an unusual posture and action in that thoracic limb but without any evidence of discomfort. When the dog bears weight on standing, the humerus at the shoulder is partially rotated laterally which positions the elbow more medially under the thorax. When the dog protracts the limb to walk and the affected limb is not weight bearing, the humerus suddenly further rotates laterally causing the elbow to assume a more medial position. The paw will appear to be positioned more laterally during the stride. All of this is the result of the healing of the torn infraspinatus muscle which resulted in fibrosis and a shortening

of the muscle. This shortened muscle causes a passive but persistent excessive lateral rotation of the humerus at the shoulder joint and the characteristic gait that represents this disorder. If you stand over this affected dog when it is bearing weight on both thoracic limbs, grasp both brachia and rotate them medially and laterally, you will feel resistance to medial rotation of the humerus at the shoulder of the affected limb. Surgical removal of the fibrotic portion of this affected infraspinatus muscle should correct the abnormality.