Six of the Best - Neurological Case Headaches Mini Series

Session 2: Lecture 1: Non-Painful Spinal Disease of the Dog and Cat
Lecture 2: Painful Spinal Disease of the Dog and Cat

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NON-PAINFUL SPINAL DISEASES IN DOGS

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Fibrocartilagenous embolism (FCE)
Pathogenesis and presentation
FCE is a syndrome in which fibrocartilage identical to that found in the nucleus pulposus embolises to the spinal cord vasculature producing an area of ischaemic necrosis centered on the spinal cord gray matter. Signs are often lateralised as the embolus usually lodges in one branch of the ventral spinal artery. FCE causes peracute onset of non-painful neurologic deficits, most commonly in the lumbosacral intumesence but also in the brachial intumesence. Affected dogs are usually large, young nonchondrodystrophoid breeds of dog engaged in exercise at onset of signs, but smaller breeds such as Shetland sheepdogs, Miniature Schnauzers and Yorkshire terriers can be affected, and are more likely to have C6-T2 signs. Signs are often dramatically lateralised producing hemiparesis. Involvement of the sympathetic tracts in the cervical spinal cord can result in Horner's syndrome and vasodilation on the affected side. The vasodilation produces differential hyperthermia that can be detected by comparing the temperature in the front feet, and comparing the external pinnae (which will be flushed on the affected side).

Diagnosis
FCE should be suspected when a dog presents with peracute onset of lateralizing signs in the absence of spinal pain. Survey spinal radiographs are unremarkable, and there is no evidence of spinal cord compression on myelography, although occasionally focal swelling of the cord is detected. CSF analysis may reveal a disproportionately elevated protein and neutrophilic pleocytosis. MRI has become the standard imaging modality for the diagnosis of spinal cord ischemia and the extent of the lesion can have prognostic significance.

Treatment
Treatment centers around successful rehabilitation of the dog. Improvement can be dramatic over the first seven days and will continue for one to three months after injury.

Prognosis
The extent of recovery will depend on the extent of injury. If deep pain perception is preserved in the thoracic and pelvic limbs on the affected side, the prognosis is good. If deep pain perception is absent in one or more limbs, the prognosis is more serious, but deep pain perception should be monitored weekly and its reappearance indicates the potential for recovery.

Neoplasia - Neoplasia can cause pain and tetra-or paraparesis dependent on the location of the lesion. In dogs, extradural tumors (e.g. sarcomas and round cell tumors) account for approximately 50% of spinal tumors, intradural tumors (meningiomas and nerve root tumors) account for 35% and intramedullary tumors (e.g. gliomas and ependymomas) account for the remaining 15%. In cats the most common spinal tumor is lymphosarcoma. Diagnosis of neoplasia can be made tentatively from plain radiographs in instances when the vertebrae are affected. In the absence of bone lesions, CSF analysis, myelography, CT or MR imaging can be used to identify and characterize lesions more carefully. Biopsy or fine needle aspirate is needed to establish a definitive diagnosis in all cases. Treatment depends on the tumor type and varies from palliative treatment of pain and peritumoral edema with anti-inflammatory doses of corticosteroids and / or palliative radiation, to chemotherapy for round cell tumors, and combinations of surgical excision and radiation. Prognosis is very variable and is always guarded if neurologic deficits are severe. Prognosis for sarcomas, carcinomas, and gliomas is grave. Meningiomas are responsive to surgical resection, especially if surgery is followed by radiation and survival of over two years has been reported. Survival ranging from four to 27 months has been reported in vertebral plasma cell tumors treated with chemotherapy +/- radiation.

Syringohydromyelia
Syringomyelia is a fluid filled cavity within the spinal cord, and hydromyelia is simply dilation of the central canal. Both conditions can be a secondary long term complication of any spinal cord disease, especially those in which a large volume of spinal cord tissue becomes necrotic. Syringohydromelia also occurs in association with congenital anomalies such as Chiari-type malformations in the Cavalier King Charles spaniel and other small breed dogs like Pomeranians. Clinical signs are caused by progressive expansion of the cavities and relate to their location. A prominent sign in affected Cavalier King Charles spaniels was persistent scratching in the region of the shoulder with apparent pain in the neck, facial and shoulder region. Torticollis and scoliosis have both been reported in association with the problem. If the cavities connect with the subarachnoid space, myelography may be diagnostic, but they are most reliably visualized on MR images. Treatment may be conservative with anti-inflammatory doses of prednisone and physical therapy, or surgical if there is an underlying cause that can be addressed, such as occipital dysplasia. Prognosis depends on the severity of signs and whether an underlying cause can be addressed.

Degenerative myelopathy
Clinical signs:
Dogs with degenerative myelopathy (DM) show an insidious, progressive ataxia and paresis of the pelvic limbs ultimately leading to paraplegia and euthanasia. If pelvic limb hyporeflexia is observed, reflecting nerve root involvement, the disease is termed canine degenerative radiculomyelopathy (CDRM). Although the German Shepherd Dog is the most commonly affected breed, DM has been reported in many other pure- and mixed-breeds. There is no sex predilection. In most breeds, the mean age of onset of neurological signs is 9 years.

Progressive, asymmetric UMN paraparesis, pelvic limb GP ataxia and lack of paraspinal hyperesthesia are key clinical features of DM. The clinical course of DM can vary after the presumptive diagnosis. Pet owners usually elect euthanasia when the dogs can no longer support weight in their pelvic limbs and need walking assistance. Smaller breed dogs can be cared for by the pet owner over a longer time. The median disease duration in the Pembroke Welsh Corgi (PWC) was 19 months. As a result of a longer survival time, affected PWCs often have signs of thoracic limb paresis at the time of euthanasia.

Early Disease. The earliest clinical signs of DM are GP ataxia and mild spastic paresis in the pelvic limbs. Worn nails and asymmetric pelvic limb weakness are apparent upon physical examination. Asymmetry of signs at disease onset is frequently reported. At disease onset, spinal reflex abnormalities are consistent with UMN paresis localized in the T3 to L3 spinal cord segments. Patellar reflexes may be normal or exaggerated to clonic; however, hyporeflexia of the patellar reflex has also been described in dogs at similar disease stage. Flexor reflexes may also be normal or show crossed extension (suggestive of chronic UMN dysfunction). Most large breed dogs progress to nonambulatory paraparesis within 6 to 9 months from onset of clinical signs. Often dogs progress to nonambulatory paraparesis and are euthanized during this disease stage.

Late Disease. If the dog is not euthanized early, clinical signs will progress to LMN paraplegia and ascend to affect the thoracic limbs. Flaccid tetraplegia ultimately occurs in dogs with advanced disease. The paresis becomes more symmetrical as the disease progresses. LMN signs emerge as hyporeflexia of the patellar and withdrawal reflexes, flaccid paralysis, and widespread muscle atrophy beginning in the pelvic limbs as the dogs become nonambulatory. Widespread and severe loss of muscle mass occurs in the appendicular muscles in the late stage of DM. Most reports attributed loss of muscle mass to disuse but flaccidity in dogs with protracted disease suggests denervation.. Cranial nerve signs include swallowing difficulties and inability to bark. Urinary and fecal continence usually are spared until when the dog becomes paraplegic.

Pathogenesis: For many years the aetiology of degenerative myelopathy remained unknown. Immunologic, metabolic or nutritional, oxidative stress, excitotoxic and genetic mechanisms have been explored as underlying the pathogenesis of DM. The uniformity of clinical signs, histopathology, age and breed predilections suggested an inherited basis for DM. Segregation of DM in families has been reported in the Siberian Husky, Pembroke Welsh Corgi and Chesapeake Bay Retriever . Awano et al.

(2009) used genome-wide association and determined a missense mutation in the superoxide dismutase 1 (*SOD1*) gene. Mutations in *SOD1* are an underlying cause for some forms of human ALS (Lou Gehrig's disease), an adult onset fatal paralytic neurodegenerative disease. The disease derives its name from the combined degeneration of upper and lower motor neurons projecting from the brain and spinal cord. The Greek derivation of amyotrophy means, "muscle without nourishment." Lateral is the location within the spinal cord of axonal disease and sclerosis refers to 'hardening' with axons being replaced by astrogliosis. Canine DM associated with this *SOD1* mutation resembles an upper motor neuron onset form of human ALS. Dogs testing homozygous for the mutation are *at risk* for developing DM. Some dogs are homozygous for the mutation but remain free of clinical signs which suggest age-related incomplete penetrance.

Diagnosis: Tentative antemortem diagnosis is presently based upon ruling-out other diseases causing progressive myelopathy. Common differentials include intervertebral disc disease, inflammatory disease and spinal cord neoplasia. Hip dysplasia and degenerative lumbosacral stenosis (LSS) often can be confused with the diagnosis of DM although the neurological findings are different if a careful examination is performed. It is not uncommon for DM affected dogs to have coexisting neurological and orthopaedic diseases. Diagnostic testing is typically performed in the early disease stage. The lack of abnormal findings of electrodiagnostic testing, and myelography or cross sectional imaging (MRI, CT) along with characteristic disease pattern for progression of neurologic signs support a presumptive diagnosis of DM. Later in the disease course, electromyography and nerve conduction studies will show abnormalities associated with denervation atrophy and axonopathy / demyelination.

Definitive diagnosis of DM is determined post-mortem by histopathological examination of the spinal cord. In general, neuropathological lesions involve the spinal cord myelin and axons in all funiculi but most severe in the dorsal portion of the lateral and medial portion of the dorsal funiculi. Histopathologic changes include degeneration and neuronal fiber loss of ascending sensory and descending motor pathways that are most severe in the mid- to caudal thoracic spinal cord. In dogs with advanced DM, nerve specimens show fiber loss resulting from axonal degeneration and secondary demyelination. Muscle specimens have changes typical of denervation atrophy.

Although the clinical signs, disease progression and genetic analysis are provocative for considering DM as a canine model of ALS, there remain significant pathological and clinical differences. These include the absence of any evidence of neuronal cell body degeneration or loss in the ventral horn of the spinal cord and the diffuse nature of the axonopathy that involves the proprioceptive pathways as well as the UMN tracts. Studies are underway to further describe the neuronal cell body and spinal nerve root and nerve pathology associated with DM.

Treatment and prognosis: Treatment regimens have been empiric with lack of evidence-based medicine approaches. Moreover, there exists no prophylactic or curative treatment for human ALS. Aminocaproic acid, an antiprotease agent, has been advocated for long-term management of DM; however, there have been no published clinical data to support drug efficacy. While treatment of vitamin deficiencies can resolve neurological disease in some animals, therapy with parenteral cyanocobalamin or oral alpha-tocopherol did not affect neurological progression in a study of DM affected dogs. Physical rehabilitation regimens have been advocated for management of DM. Overall, long-term prognosis is considered poor.

Hansen type II Disc Disease
Hansen type II intervertebral disc disease (IVDD) is annular protrusion caused by shifting of central nuclear material and is commonly associated with fibroid disc degeneration.

This type of disc degeneration is more common in older non-chondrodystrophoid dogs and has a slow progressive onset and is frequently non-painful but it can be associated with acute onset of pain on an intermittent basis. However, it can be seen in chondrodystrophoid dogs, again having a slow progressive course. Spinal cord damage is usually most severe at the site of the intervertebral disc protrusion. Pathologic changes are characterised as compressive myelopathy with demyelination of the ventral, lateral, and dorsal funiculi. Wallerian degeneration occurs in the spinal cord segments above and below

the lesion in the ascending and descending pathways, respectively. An inflammatory reaction consisting primarily of macrophages and fibrous astrocytes develops as a result of the neurodegenerative process.
As for disc extrusion, the treatment for disc protrusion is often determined by multiple factors including; neurologic status of the dog, delay in presentation for veterinary attention, concurrent medical treatment, concurrent vertebral instability, previous episodes of spinal pain, method of diagnosis of the protrusion, lesion localisation and economics. Again, this means that criteria for such treatment need to remain 'fluid' and therefore specific treatments may differ in similar patient circumstances.

SPINAL PAIN DISORDERS IN DOGS
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Chiari-like malformation and Syringomyelia (CM/SM):
Chiari-like malformation (CM) and syringomyelia (SM) often occur together, although both may occur independently of the other. Syringomyelia is a condition characterized by the presence of a fluid filled cavity (syrinx) or cavities within the parenchyma of the spinal cord. SM is secondary to abnormal cerebrospinal fluid movement and is usually associated with Chiari-like malformation, although it may be associated with other conditions such as congenital malformations, trauma, inflammation, and neoplasia. Chiari-like malformation is defined as a decreased caudal fossa volume with herniation of the cerebellum and often brainstem into or through the foramen magnum. In people, this condition is referred to as Chiari malformation, which has several types.

The term syringomyelia is accepted to describe fluid accumulation within the spinal cord, whether it be secondary to central canal dilation (hydromyelia) or secondary to fluid accumulation within the spinal cord parenchyma (syringomyelia or syringhydromyelia). It is difficult to determine the location of the fluid using Magnetic Resonance Imaging (MRI) and these cavities often communicate with each other. Syringomyelia frequently occurs with Chiari-like malformation in dogs and the terms Chiari-like malformation and syringomyelia (CM/SM) have been adopted to describe the canine condition.

Clinical signs: Onset of signs may be acute or chronic in dogs ranging from 6 months to 10 years of age. The most common sign of CM/SM is pain, predominately isolated to the cervical region, occurring in 35% of affected dogs and 80% of people with the similar condition. Syrinx width, measured by MRI, has been shown to be the strongest predictor of pain in dogs where a wider syrinx was significantly associated with discomfort. Additionally, the location of the syrinx within the dorsal aspect of the spinal cord affecting the dorsal horn is thought to be one mechanism behind the development of pain, specifically neuropathic pain. Neuropathic pain is secondary to disordered processing of sensory information within the nervous system and results in spontaneous pain, paresthesia, dysthesia, allodynia, or hyperpathia. As a result, dogs may dislike touch to the skin of their neck or they may scratch with or without making contact to the skin on their neck. This "phantom scratching" has frequently been described in affected dogs. Pain may also be to CM alone as seen in dogs without SM secondary to compression of the brainstem or first cervical nerve.

Other clinical signs depend on the location of the syrinx, although the cervical spinal cord is predominately affected. These clinical signs include scoliosis and neurological deficits relating to cervical spinal cord dysfunction. Intracranial signs, such as facial paresis and vestibular dysfunction, have also been reported. However, dogs may also be asymptomatic for CM/SM.

Pathogenesis: The human classification of Chiari type I, which is the most similar to the canine condition, necessitates elongation and caudal displacement of the cerebellar tonsils (vermis and paravermal lobes) through the foramen magnum into the cranial cervical vertebral canal. A similar condition has been documented in dogs particularly affecting toy or small breed dogs. Predisposition to CM/SM has been seen in Cavalier King Charles Spaniels (CKCS) and Brussels Griffon dog. In CKCS, CM/SM is a hereditary condition, possibly autosomal recessive with incomplete penetrance. CM is caused by congenital hypoplasia of the supraoccipital bone resulting in overcrowding of the structures within the caudal fossa. As a result, the cerebellum herniates through or into the foramen magnum, the medulla becomes kinked, and the dorsal subarachnoid space at the craniocervical junction is obstructed. The flow of CSF through the foramen magnum is disrupted as a result. Many theories behind why syringomyelia develops as a result of this obstruction have been postulated although a

single theory has not been proven.

Diagnosis: These structural abnormalities are best diagnosed with MRI, but they may be clinically silent; therefore, their significance must be carefully considered when such abnormalities are discovered.

Treatment and prognosis: Treatment may not be necessary in asymptomatic dogs or dogs with mild nonprogressive signs. Dogs exhibiting pain, more severe neurological deficits, or progressive signs can be treated either medically or surgically. Typically, medical therapy is pursued initially involving the use of analgesics and drugs that reduce CSF formation. Furosemide (1-2 mg/kg orally q12h) and prednisone (0.5-1 mg/kg orally 24h, tapering dose) are frequently used. Treatment of neuropathic pain with drugs such as Gabapentin (10 mg/kg PO q 8 h) is also an important aspect of therapy.

Approximately 70% of patients show some improvement, but it is rarely complete. If medical therapy does not alleviate the clinical signs, surgical decompression of the foramen magnum has been suggested (suboccipital craniectomy) and is the treatment of choice in people. Foramen magnum decompression has been performed in dogs with a success rates reported at about 80%; however, recurrence is common and neuropathic pain may persist requiring continued medical therapy. Additionally, multiple surgeries may be required if scar tissue develops at the surgical site obstructing CSF flow; although, cranioplasty may reduce the likelihood of this complication. Improvement may not be a result in the reduction of syrinx size, which usually persists.

STEROID RESPONSIVE MENINGITIS-ARTERITIS

A severe form of steroid responsive meningitis-arteritis (SRMA) has been reported in Beagles, Bernese Mountain Dogs, Boxers, German Short-Haired Pointers, and sporadically in other breeds. This condition has a worldwide distribution and represents one of the most important inflammatory diseases of the canine CNS. Beagles, especially but not exclusively those in laboratory-bred colonies, appear at risk. In the Beagles, the condition has been termed Beagle pain syndrome, necrotizing vasculitis, polyarteritis, panarteritis, juvenile polyarteritis syndrome, and primary periarteritis. In other breeds, this condition previously appears under the terms necrotizing vasculitis, corticosteroid-responsive meningitis, aseptic suppurative meningitis, and corticosteroid-responsive meningomyelitis. This plethora of terminology reflects not only the dearth of knowledge about this condition but also highlights important clinical signs such as pain, improvement following corticosteroid medication, and histologic involvement of the meninges and blood vessels.

Affected animals usually are most commonly young adults between 8 and 18 months of age, although the age range may extend from 4 months to 7 years. The clinical course is typically acute with recurrences. A more protracted form of the disease may be seen following relapses and inadequate treatment. Signs include recurring fever, hyperesthesia, cervical rigidity, and anorexia. There may be a creeping gait, arching of the back with head held down, and crouched posture. Some dogs with protracted disease may show clinical signs of parenchymal involvement such as ataxia, paresis, tetraparesis or paraplegia. Hematological studies often reveal a peripheral neutrophilia with a left shift, increased erythrocyte sedimentation rate, and in some cases, an elevated a - 2-globulin fraction. CSF studies indicate increased protein and neutrophilic pleocytosis.

The cause of SRMA remains unknown. To date, no bacterial or viral infectious agents have been identified, although activated T cells have been found in some dogs indicating these cells have had contact with some unidentified antigen.

The prognosis is guarded to favorable, especially in dogs with acute disease that are treated promptly using immunosuppressive doses of corticosteroids. Untreated dogs tend to have a remitting and relapsing course. Tipold recommends the following long-term therapy (e.g., for at least 6 months), especially in any dog that has had a relapse: prednisolone at 4 mg/kg/day, PO or IV initially. After 2 days, the dose is reduced to 2 mg/kg daily for 1 to 2 weeks, followed by 1 mg/kg daily. Dogs are re-examined, including CSF analysis and hematology, every 4 to 6 weeks. When signs and CSF are normal, the dose can be reduced to half of the previous dosage until a dosage of 0.5 mg/kg every 48 to 72 hours is attained. Treatment is stopped 6 months after clinical examination, CSF, and blood profiles are normal. In refractory cases, other immunosuppressive drugs such as azathioprine (at 1.5 mg/kg PO every 48 hours)

may be used in combination with steroids (e.g., alternating each drug every other day). Antibiotics are ineffective. Results of a long-term treatment protocol (up to 20 months) involving 10 dogs with SRMA have been recently published. Eight of the 10 dogs were without clinical signs up to 29 months after the treatment was terminated. Long-term glucocorticosteroid treatment resulted only in mild clinical side effects, such as polyuria/polydipsia, polyphagia and weight gain, which were reversible after the therapy was discontinued. It was noted that elevated serum and CSF IgA levels did not decrease to normal values during prednisolone treatment and were still slightly increased after the therapy was discontinued. Monitoring of CSF cell count in dogs with this condition was a sensitive indicator of success of treatment. In addition, older dogs with high IgA levels in the CSF and frequent relapses seemed to require a longer duration of therapy and had a less favorable prognosis long term.

Note that Akitas, Bernese Mountain dogs, and other breeds with immune-mediated polyarthritis may show similar clinical signs as animals with SRMA and have concurrent meningitis.

Atlantoaxial subluxation

Presentation and pathogenesis

The atlas (first cervical vertebra) and axis (second cervical vertebra) are bound together by ligaments that run from the dens of the axis to the atlas and the skull, over the dens binding it to the floor of the atlas (the transverse ligament) and between the dorsal lamina of the atlas and the dorsal spinous process of the axis. The dens is a boney projection from the cranial aspect of the body of the axis and develops from a separate growth plate. Subluxation of the atlantoaxial junction is a relatively common problem and usually results from a failure of ligamentous support. Toy and small breeds of dog such as the Chihuahua and Yorkshire terrier are at highest risk of the problem as a result of failure of development of the dens (congenital absence or hypoplasia of the dens). Fracture of the dens and rupture of the soft tissues maintaining the stability of the atlantoaxial junction can occur in any dog or cat as a result of trauma. Onset of signs in dogs with the congenital form of the disease usually occurs in young dogs (less than two years of age), although problems can develop at any age. Signs can develop acutely or gradually, and waxing and waning of signs is often reported; presumably a reflection of instability at the atlantoaxial junction causing repeated injury to the spinal cord. Signs include neck pain (variably present), ataxia, tetraparesis, and postural reaction and conscious proprioceptive deficits with normal to increased muscle tone and myotatic reflexes in all four legs. In severe cases, animals can present with tetraplegia and difficulty breathing and they may die acutely as a result of respiratory failure.

Diagnosis

Atlantoaxial subluxation can be diagnosed from survey radiographs of the cervical spine although extreme care must be taken when restraining and moving dogs in which this disease is suspected. If the animal is sedated or anesthetized, the head and neck should be supported in slight extension to avoid further spinal cord injury. On lateral radiographs an increased space can be seen between the dorsal lamina of the atlas and the dorsal spinous process of the axis. In severe cases, malalignment of the bodies of the atlas and axis is clearly visible. The presence and size of the dens can be evaluated most accurately on VD views. If there is no evidence of subluxation on the lateral views, the neck can be carefully flexed to see if there is instability (the space between the dorsal lamina of the atlas and the dorsal spinous process of the axis should be evaluated). It is preferable to do this with fluoroscopy so that the movement can be monitored to prevent accidental iatrogenic subluxation. Recently, MRI evaluation of the spine and cord has proved beneficial in both the diagnosis and prognosis.

Treatment
Conservative

Dogs with mild signs can be treated conservatively by placing an external splint for at least 6 weeks. The splint must immobilize the atlantoaxial junction and so must come over the head cranial to the ears and go back to the level of the chest. The aim is to stabilize the junction while the ligamentous structures heal. The splint should be checked daily for signs of pressure sores by the owner and checked weekly by the veterinarian, with regular bandage changes if necessary. While often effective in the short term, the long-term efficacy of this approach is not known and dogs treated in this way will always be at risk of repeated injury.

Surgery

Surgery is recommended in dogs with neurological deficits, although it can be associated with high perioperative morbidity and mortality. Dorsal and ventral approaches to the atlantoaxial junction have been described, but dorsal approaches are associated with a greater risk of causing spinal cord injury during surgery, and a higher incidence of implant failure. Using ventral approaches, subluxation is reduced and the atlantoaxial articular surfaces are curetted to promote boney fusion. The two bones are fused using transarticular screws or Kirschner wires and a cancellous bone graft placed over the junction. In case of a traumatic injury or poor bone purchase, screws or Kirschner wires are placed in the body of the atlas and axis and the junction stabilized with polymethylmethacrylate cement . A neck splint is placed postoperatively while fusion occurs. This is a problematic area to repair surgically; bone quality is often poor, the bones are small, movement of the vertebrae may cause additional injury to the spinal cord, and the pharynx and larynx can be damaged during retraction. There is a risk of respiratory arrest and death in the perioperative period as a result of additional spinal cord injury, or inflammation of the upper airways secondary to retraction.

Prognosis

This is a serious disease but dogs with mild deficits treated surgically have an excellent prognosis if they survive the 48-hour perioperative period. Although reported surgical success rates range from 50% to 90%, the majority report a mortality rate in the region of 20% with the majority of deaths occurring either during or immediately after surgery. As with all spinal cord diseases, prognosis is worse in animals with severe and chronic neurological deficits. It has also been shown that prognosis is better in young dogs (<24 months).

Cervical disc disease

Presentation and pathogenesis

Cervical disc disease is a common problem in chondrodystrophoid breeds of dog such as Dachshunds, Shih Tzus, and Pekingese. It also occurs frequently in Beagles and Cocker spaniels and can occur sporadically in almost any breed. Although thoracolumbar disc herniations have been reported in cats, cervical disc herniations are extremely rare. The intervertebral disc is composed of an outer fibrous portion (the anulus fibrosus) and a gelatinous center (the nucleus pulposus). With normal ageing the nucleus is slowly replaced by fibrocartilage, but in chondrodystrophoid breeds the nucleus ages prematurely and the nucleus matrix degenerates and mineralizes. As a result of these degenerative changes, affected dogs are prone to extrusion of the mineralized nucleus pulposus into the spinal canal, (Hansen type 1 disc herniations) causing spinal cord concussion and compression. The C2/3 disc is most commonly affected, with incidence decreasing further caudally in the cervical spine.

Onset of signs can occur from eighteen months of age, with a peak incidence between three and seven years of age. It is very unusual for a disc herniation to occur in dogs less than two years of age as the predisposing degenerative changes have not occurred. The most common presenting sign is severe neck pain as there is enough space within the cervical vertebral canal for herniation of disc material without compression of the spinal cord. The dog may adopt a stance with the head held down, neck rigid and back arched as the weight is shifted to the pelvic limbs. Entrapment of nerve roots can cause a nerve root signature (holding up a thoracic limb and lameness). The neck pain is so severe that dogs avoid moving their head, and spasm and rigidity of the cervical musculature are easily palpable. Neurological deficits are less common but can occur when the spinal cord is sufficiently compressed and include tetra or hemiparesis or -plegia, ataxia, and conscious proprioceptive and postural reaction deficits.

Diagnosis

Survey radiographs should be taken to identify degenerative changes typical of a disc herniation and to rule out other causes of the signs. Changes indicative of a disc herniation include narrowing of the intervertebral disc space, narrowing of the intervertebral foramen and the presence of mineralized material within the vertebral canal and disc space. However, a definitive diagnosis cannot be reached with survey radiographs alone with adequate accuracy for surgery to be undertaken and MRI, computed tomography or myelography are used to identify the site of spinal cord compression. CSF analysis is performed concurrently to rule out an inflammatory disorder.

Treatment
Conservative
Dogs can be managed conservatively with strict cage rest for four weeks combined with pain relief using anti-inflammatory drugs, opiates and/or muscle relaxants. Judicious use of anti-inflammatory doses of corticosteroids combined with appropriate cage confinement can be attempted if the pain is not responsive to non-steroidal anti-inflammatory drugs. Muscle spasm can also be responsive to gentle massage and hot packing of the neck. Administration of an H2 blocker such as famotidine may help to prevent the development of gastric ulceration. The aim of cage rest is to allow defects in the annulus fibrosus to heal, and resolution of pain does not mean that confinement should be discontinued. If this approach is successful, gradual reintroduction to controlled exercise can be attempted and the owners should be cautioned to prevent their pet from activities that involve jumping in the long term. Dogs should be monitored weekly and if the pain is unresponsive to conservative therapy, recurs, or neurological deficits develop, surgery should be recommended.

Surgery
Indications for surgery include unremitting or severe pain, recurrent pain, or neurological deficits. Once the site of disc herniation has been confirmed, a ventral slot is performed to remove the herniated disc material. Adjacent discs are fenestrated to prevent recurrence of the problem. Post operatively dogs are provided with pain relief and confined for four weeks (two weeks of strict confinement and then if doing well, two weeks of increasing controlled exercise). Dogs are then gradually re-introduced to normal activity. If the dog has neurological deficits, post-operative care includes performing passive range of motion exercises, massage, hydrotherapy and controlled exercise.

Prognosis
Prognosis for dogs treated conservatively is unknown. Prognosis for dogs treated surgically is excellent unless neurological deficits are severe.