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CHAPTER 24

Disorders of the Spinal Cord

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CONGENITAL AND DEVELOPMENTAL DISORDERS

Atlantoaxial Subluxation

Definition

I. Instability or malformation of the atlantoaxial joint allows excessive flexion of the cervical (C) 1-2 vertebral joint.
II. Subsequent ventral cord compression occurs from the cranial aspect of the body of the axis.

Causes and Pathophysiology

I. It most commonly occurs in young, toy, and small-breed dogs (<2 years).
II. Subluxation is often associated with failure of normal structural development of the atlas, axis, and their supporting ligaments.
III. It can occur following minor trauma in the presence of joint instability.
IV. Clinical signs arise from concussion and compression of the spinal cord by the axis.

Clinical Signs

I. Onset is usually acute; however, signs can be slowly progressive and may wax or wane.
II. Neck pain occurs in at least 60% of cases (Beaver et al., 2000).
III. Ataxia, tetraparesis, postural reaction deficits, with normal to exaggerated spinal reflexes occur in 85% of cases (Beaver et al., 2000).
IV. Animals with tetraplegia are at risk of death from respiratory failure.
V. Occasionally, signs of brainstem dysfunction are seen.

Diagnosis

I. Confirm the diagnosis with lateral survey radiographs of the cervical spine.
II. Note an increased space between the dorsal lamina of C1 and the spinous process of the C2.
III. Use ventrodorsal views to assess most accurately the presence and size of the dens.
IV. Flex the neck carefully to confirm instability, if subluxation is not readily apparent.
V. Computed tomography (CT) can provide accurate information about the dens.
VI. Magnetic resonance imaging (MRI) can provide important information about spinal cord compression, parenchymal pathology, and ligamentous involvement.

Differential Diagnosis

I. Trauma
II. Intervertebral disc disease
III. Meningomyelitis: various forms

Treatment

I. Treat cases with acute onset of severe neurological signs with external head and neck splinting.
II. Conservative therapy may be appropriate for some dogs with mild signs.
III. Apply external splinting and restrict exercise strictly for 6 to 8 weeks.
   A. Long-term efficacy of this approach is uncertain.
   B. Dogs are always at risk of repeated injury following splint removal.
IV. Surgical management is recommended for dogs with neurological deficits.
   A. Dorsal and ventral approaches to the atlanto-axial junction have been described (McCarthy et al., 1995; Platt et al., 2004).
   B. Surgery is aimed at realignment of the vertebrae, decompression of the spinal cord, and osseous fusion of the atlantoaxial joint.
   C. Surgical complications include injury to soft tissues, laryngeal paralysis, implant failure, and death in up to 20% of cases (Beaver et al., 2000).

Monitoring of Animal

I. Nonsurgical approach is most successful in dogs that are affected for ≤30 days (Havig et al., 2005).
II. Surgical success ranges from 50% to 90% (McCarthy et al., 1995; Beaver et al., 2000).
III. Prognosis is better in young dogs (<24 months) and in those with clinical abnormalities for ≤10 months (Beaver et al., 2000).
IV. Prognosis is fair to good for those with mild to moderate neurological deficits, and guarded for those with an acute onset of tetraplegia.
V. Radiographs repeated 6 to 8 weeks after surgery may help determine the presence of osseous healing.

Congenital Vertebral Anomalies

Definition

I. Several distinct types of malformation are recognized.
II. Hemivertebrae are wedge-shaped, with the apex directed dorsally, ventrally, or medially across the midline, which often results in angulation of the vertebral column.
III. Block vertebrae appear as fusion of adjacent vertebrae, which may involve the vertebral bodies, vertebral arches, dorsal spinous processes, or entire vertebrae at any level of the vertebral column.
IV. Butterfly vertebrae have a sagittal cleft in the vertebral body.
V. Transitional vertebrae have the characteristics of, and occur at, two major divisions of the vertebral column.

Causes and Pathophysiology

I. Vertebral malformations are the result of a disturbance in embryonic development.
II. Hemivertebrae are inherited in the German shorthaired pointer, English bulldog, and Yorkshire terrier.
III. High prevalence of hemivertebrae and butterfly vertebrae occurs in the French bulldog, pug, and Boston terrier.

Clinical Signs

I. Most congenital malformations of the vertebrae cause no clinical signs.
II. Spinal cord compression can occur from canal stenosis, vertebral malalignment, or instability.
III. Hemivertebrae are most commonly associated with clinical signs.
IV. Signs are often slowly progressive, owing to the chronic compression.
V. Neurological deficits are consistent with a transverse myelopathy and include the following:
   A. Conscious proprioception deficits
   B. Ataxia
   C. Paresis or paralysis

Diagnosis

I. Confirmation of a vertebral anomaly is done by survey radiography.
II. Myelography is useful for determining the extent of associated cord compression or stenosis.
III. MRI is valuable in determining associated spinal cord compression or concurrent spinal cord anomalies.

Differential Diagnosis

I. Compression caused by traumatic or pathologic vertebral fractures
II. Vertebral neoplasia
III. Healed osteomyelitis (diskospondylitis)

Treatment

I. If clinical signs are nonprogressive, conservative management is recommended.
II. Surgical decompression combined with stabilization is recommended for progressive clinical signs from spinal cord compression.

Monitoring of Animal

I. Prognosis is good if the defect is an incidental finding; vertebral anomalies can become clinical later in life.
II. Prognosis is guarded with chronic, progressive cord compression.

Spina Bifida

Definition

I. Spina bifida is characterized by a midline cleft in the vertebral arch.
II. Spina bifida occulta indicates that the spinal cord and meninges are normal.
III. Clinical spina bifida is accompanied by a meningocele or a meningomyelocele.
IV. Meningocele is a protrusion of the meninges through the vertebral cleft.
V. Meningomyelocele is a protrusion of the meninges and spinal cord through the vertebral cleft.

Causes and Pathophysiology

I. Spina bifida is likely the result of abnormal development of the neural tube.
II. Teratogenic compounds, nutritional deficiencies, and environmental factors may be associated with spina bifida.
III. A high incidence in the bulldog and Manx cats suggests a heritable cause.
IV. In Manx cats and bulldogs, there is an association with sacrocaudal dysgenesis.
V. In the Manx, an autosomal-dominant condition leads to absence of the tail and to several sacral-related abnormalities.

Clinical Signs

I. Spina bifida is rarely associated with clinical signs.
II. When present, signs vary with the degree of spinal cord and meningeal involvement.
III. Most lesions occur in the caudal lumbar region, but can occur in any vertebra.
IV. Physical examination may reveal abnormal directions of hair growth, a skin dimple, or an open tract draining cerebrospinal fluid (CSF) at the site of the lesion.
V. Fistulous meningocele can cause hypochloremia and hyponatremia subsequent to CSF loss.

Diagnosis

I. Caution must be used when attributing clinical signs to a vertebral anomaly.
II. Dorsoventral radiographs of the vertebral column may reveal an absence of the spinous process(es).
III. CT is useful for confirmation of subtle lesions.
IV. Myelography may detect a meningocele.
V. MRI may detect specific spinal cord changes and is advised before surgical intervention.

Differential Diagnosis

I. Other congenital spinal cord anomalies
II. Meningomyelitis

Treatment and Monitoring

I. Treatment is not necessary for spina bifida occulta.
II. Meningoceles can be surgically resected in cases without concurrent spinal cord lesions.
III. Tethered spinal cord syndrome can be treated surgically by severing the filum terminale.
IV. Surgical closure of spina bifida aperta, removal of the fistulous tract, and broad-spectrum antibiotics may be necessary to reduce the incidence of bacterial meningomyelitis.
V. Prognosis is poor in cases with fecal and urinary incontinence, or severe neurological deficits.
VI. Frequent bladder and bowel expression may be necessary in some cases.

Myelodysplasia

Definition

I. Myelodysplasia refers to a variety of embryologic abnormalities of the spinal cord.
II. A specific form of myelodysplasia, spinal dysraphism, maybe hereditary in Weimaraners.

Causes and Pathophysiology

I. Myelodysplasia results from incomplete closure or development of the neural tube.
II. Anomalies of the central canal include hydromyelia, duplication, or absence of the canal.
III. Anomalies of the grey matter involve the ventral median fissure or dorsal median septum.
IV. Grey matter ectopias, chromatolysis, and loss of nerve cell bodies may also be present.
V. Syringomyelia is a fluid dilatation, usually in the dorsal funiculus of the spinal cord.
VI. With the exception of the Weimaraner, the pathogenesis of this condition is unknown.

Clinical Signs

I. The most common sign is a symmetrical, "bunny hopping" pelvic limb gait.
II. A variable transverse thoracolumbar myelopathy usually occurs in young animals (4 to 6 weeks), which is typically nonprogressive.
   A. Proprioception deficits
   B. Ataxia
   C. Paresis or paralysis
III. A classic finding is a bilateral flexor reflex in the pelvic limbs (both limbs respond to stimulation of one limb).
IV. In Weimaraners, myelodysplasia may be associated with abnormal hair "streams" on the dorsum and koilosternia.

Diagnosis

I. A tentative diagnosis is made on the basis of history, signalment, and clinical signs.
II. Radiographs, CSF analysis, CT, and MRI are usually normal.
III. MRI can detect the presence of hydromyelia and syringomyelia.

Differential Diagnosis

I. Vertebral malformation
II. Spina bifida
III. Myelitis

Treatment and Monitoring

I. No effective treatment exists.
II. Prognosis varies with severity of the clinical signs.

Syringomyelia and Hydromyelia

Definition

I. Hydromyelia is a fluid dilatation of the central canal.
II. Syringomyelia is a fluid dilatation in the spinal cord that may communicate with the central canal.
III. It is often difficult to distinguish between these two conditions.

Causes and Pathophysiology

I. Both conditions can be a secondary, long-term complication of any spinal cord disease.
II. Any condition that causes obstruction of normal CSF flow along the spinal cord can cause these abnormalities.
III. They are most commonly seen in the cervical region, but lesions can occur in any portion of the spinal cord.
IV. Cervical syringohydromyelia occurs as a component of congenital anomalies associated with caudal occipital malformation syndrome, which is most commonly reported in the Cavalier King Charles spaniel, but can be seen in other small breed dogs.
V. Occipital bone malformation causes "overcrowding" of the caudal fossa that interferes with the normal flow of CSF between the intracranial and spinal compartments.

Clinical Signs

I. Clinical signs vary with lesion location.
II. Signs are often progressive, but can be acute and can occur at any age.
III. Clinical signs do not correlate with the severity of the syringohydromyelia or with the concurrent severity of cerebellar herniation and hydrocephalus (Lu et al., 2003).
IV. Typical signs consistent with a transverse myelopathy include the following:
   A. Proprioception deficits
   B. Ataxia
   C. Paresis or paralysis
V. Additional signs include pain, paresthesia, spinal deformity (torticollis, scoliosis), and persistent flank scratching (Rusbridge et al., 2000).

Diagnosis

I. MRI is essential to document parenchymal changes and associated lesions, such as occipital malformation.
II. Survey radiographs are usually normal unless concurrent scoliosis is present.
III. Myelography may show obstruction of the flow of CSF at the foramen, but is often normal.

IV. Cisterna magna puncture is contraindicated given the likelihood of inadvertent puncture of the spinal cord.
V. Lumbar CSF analysis may show chronic inflammation, but is frequently normal.

Differential Diagnosis

I. Other developmental disorders
II. Intervertebral disc disease
III. Meningomyelitis
IV. Neoplasia

Treatment

I. Medical therapy involves antiinflammatories (prednisone 0.5 to 1.0 mg/kg PO SID and carprofen 2.0 mg/kg PO BID).
II. A diuretic, such as acetazolamide (10 mg/kg PO TID to QID), may be used concurrently on a short-term basis to reduce CSF production.
III. Gabapentin (10 mg/kg PO BID) can be used for neuropathic pain and paraesthesia.
IV. Physical therapy (e.g., massage, passive range-of-motion exercises) may be helpful.
V. Surgical therapy can be performed in cases of caudal occipital malformation syndrome.
   A. Foramen magnum decompression with dura and arachnoid excision is preferred for syringohydromyelia associated with occipital malformation syndrome (Vermeersch et al., 2004).
   B. Myelotomy with syrinx decompression and marsupialization of the dura can be used for treatment when the condition is related to other causes.

Monitoring of Animal

I. Medical therapy may be effective in mildly affected animals.
II. Surgical therapy aims to stabilize the condition rather than improve it.
III. Surgical therapy often alleviates neck pain.
IV. Recurrence of signs from reformation of the syrinx requires repeated surgery.
V. Overall prognosis depends on the severity of signs.

Spinal Intraarachnoid Cysts

Definition

I. These lesions are CSF-filled diverticuli of the arachnoid membrane rather than true cysts.
II. Synonyms include *subarachnoid cyst, meningeal cyst,* and *leptomeningeal cyst.*

Causes and Pathophysiology

I. Most commonly a congenital malformation, these lesions may be secondary to trauma, inflammation, subarachnoid hemorrhage, and neoplasia.
II. The intraarachnoid accumulation of CSF in the diverticulum results in compression of the spinal cord.
III. Typically solitary lesions occur commonly at the C2 to C3 and C5 to C6 intervertebral sites or in the caudal thoracic region; however, they can occur at any site (Jurina et al., 2004).
IV. The diverticuli are usually located dorsally over the spinal cord.

Clinical Signs

I. They can occur as an incidental finding.
II. They are most commonly seen in the cranial cervical cord of young adult, large-breed dogs, particularly rottweilers, or in the thoracolumbar spinal cord of older, smaller breeds (e.g., pug) (Rylander et al., 2002; Skeen et al., 2003).
III. Signs usually consist of a chronic and progressive transverse myelopathy.
   A. Proprioception deficits
   B. Ataxia
   C. Paresis or paralysis
IV. Neurological deficits may be asymmetrical and pain is a variable feature.
V. Dogs may have early onset of fecal or urinary incontinence (Skeen et al., 2003).

Diagnosis

I. Survey radiographs are unremarkable.
II. Myelography reveals a focal "tear-drop" accumulation of contrast medium in the subarachnoid space or an intradural filling defect.
III. Contrast-enhanced CT or MRI demonstrates the CSF-filled diverticulum within the arachnoid membrane and spinal cord compression.
IV. MRI can also identify associated parenchymal abnormalities, especially in rottweilers.
V. Surgical findings and histopathology of excised tissues confirm the diagnosis.

Differential Diagnosis

I. Cystic neoplasms
II. Meningomyelitis
III. Other developmental disorders
IV. Intervertebral disc disease

Treatment

I. Medical therapy may improve a small proportion of dogs and is only recommended in cases with mild neurological deficits.
II. Medical therapy involves antiinflammatory drugs and exercise restriction.
   A. Prednisone 0.5 to 1.0 mg/kg PO SID
   B. Carprofen 2.0 mg/kg PO BID
III. Surgical exploration is used to confirm the diagnosis and decompress the spinal cord.
IV. Complete surgical excision is usually not possible.
V. Partial excision (fenestration) and marsupialization of the dura is recommended.

Monitoring of Animal

I. Depending of the degree of signs, the overall prognosis is good following surgery.

II. Factors associated with a good outcome include an age <3 years and duration of clinical signs <4 months (Skeen et al., 2003).
III. Recurrence of signs from reformation of the diverticulum is possible.
IV. Lifelong antiinflammatory therapy has been advised in some cases.

Dermoid Sinus

Definition

I. A dermoid or pilonidal sinus is an invagination of the skin, dorsal to the spine, that extends below the skin to variable depths.
II. It can extend to the dura mater and may communicate with the subarachnoid space.

Causes and Pathophysiology

I. Sinus formation results from a failure of separation of the neural tube from the ectoderm.
II. An autosomal dominant mutation causing the presence of a dorsal ridge predisposes for dermoid sinus formation in the Rhodesian ridgeback.
III. Communication with the subarachnoid space predisposes to meningomyelitis.

Clinical Signs

I. Signs depend on location, with most occurring in the cervical region.
II. Clinical signs of meningitis and myelitis may be seen as a result of extension of infection.
III. Localized or generalized spinal pain and rigidity may occur with meningitis.
IV. Neurological deficits indicative of a transverse myelopathy may occur from myelitis.
   A. Proprioception deficits
   B. Ataxia
   C. Paresis or paralysis

Diagnosis

I. A dermoid sinus may be palpable or visualized as an opening of the skin on the dorsum.
II. Fistulography can be used to determine communication with the spinal canal.
III. CSF analysis can identify meningitis.
IV. Myelography identifies communication with CSF or compression of the spinal cord.
V. MRI defines the extent of the sinus and secondary compression of the spinal cord.

Differential Diagnosis

I. Meningomyelitis
II. Neoplasia
III. Intervertebral disc disease
IV. Other congenital or developmental anomalies of the vertebrae or spinal cord

Treatment and Monitoring

I. Antimicrobial therapy is indicated for meningomyelitis based on culture and sensitivity results.
II. Surgical excision, possibly combined with laminectomy, is warranted in symptomatic cases.
III. Prognosis is excellent in cases with no communication between the sinus and spinal cord.

Storage Disorders

Globoid Cell Leukodystrophy

See Chapter 23, Table 23-2.

Mucopolysaccharidosis

See Chapter 23, Table 23-2.

Degenerative Disorders

Intervertebral Disc Disease

Definition

I. Intervertebral disc disease (IVDD) implies degeneration of the intervertebral disc structures and subsequent herniation of disc material into the vertebral canal.
II. Extrusion of the nucleus pulposus through the annulus fibrosis is a Hansen type I lesion.
III. Protrusion of the annulus caused by shifting of nucleus pulposus is a Hansen type II lesion.
IV. Explosive disc disease that occurs peracutely, without compression, is a type III or high-velocity low-volume disc herniation.

Causes and Pathophysiology

I. Type I disc disease commonly affects chondrodysplastic breeds (e.g., dachshund, beagle, Pekingese, Lhasa apso, shih tzu) and chondrodystrophic-like breeds (e.g., American and English cocker spaniel, miniature poodle) (Cherrone et al., 2004).
   A. Type I disc disease is most commonly associated with chondroid disc degeneration.
   B. Disc degeneration occurs in 75% to 100% of all discs by 1 year of age in chondrodystrophoid breeds (Morgan and Miyabayashi, 1988).
   C. Type I disc disease is associated with an acute onset of signs.
   D. Often there is mineralization of the nucleus pulposus.
II. Type II disc disease commonly affects older, large-breed, achondrodystrophic dogs.
   A. Type II disc disease is associated with degeneration characterized by fibrous metaplasia of the nucleus pulposus.
   B. Type II disc disease causes slow, progressive spinal cord compression.
III. Although uncommon in cats, type I disc disease occurs most frequently (Munana et al., 2001).

Clinical Signs

I. Type I disc disease typically occurs in dogs between 3 and 5 years of age.

II. Type II disc disease typically occurs in dogs between 6 and 9 years of age.
III. On average, affected cats are 10 years of age (Munana et al., 2001).
IV. Signs reflect the region of the affected spinal cord.
V. Cervical disc disease accounts for 15% of cases (Coates, 2000).
VI. Thoracolumbar (T-L) disc disease accounts for 85% of cases (Coates, 2000).
VII. Thoracolumbar disc disease typically occurs between T11 to L3 vertebrae.
VIII. Severity of signs ranges from pain, paresis and ataxia, loss of motor abilities, or loss of nociception (deep pain perception).
IX. IVDD most commonly causes a transverse myelopathy.
  A. Proprioception deficits
  B. Ataxia
  C. Paresis and paralysis
X. Asymmetrical clinical signs are often seen.
XI. Cervical disease usually results in less-severe clinical signs than thoracolumbar disease.
  A. Apparent neck pain is the most common clinical sign with cervical disc disease.
  B. Nonambulatory tetraparesis is more common in large-breed dogs with cervical disease than small-breed dogs (Cherrone et al., 2004).
  C. Lower motor neuron (LMN) deficits or monoparesis can be seen with caudal cervical disc disease.
XII. LMN deficits may be seen in the pelvic limbs with herniations caudal to L3 that cause compression of the lumbosacral spinal cord or cauda equina.
XIII. Progressive hemorrhagic myelomalacia occurs in up to 11% of dogs that have lost nociception (Olby et al., 2003).
  A. The lesion may ascend, resulting in cranial progression of analgesia, and ultimately cause tetraplegia and respiratory failure.
  B. The lesion may descend, resulting in LMN signs in the pelvic limbs, as well as urinary and fecal incontinence.

Diagnosis

I. Spinal radiographs may be suggestive of disc herniation or be normal.
  A. Characteristic findings include a narrowed or wedged intervertebral disc space, small intervertebral foramen with narrowed articular processes, or mineralized disc material in the vertebral canal.
  B. Type I disease is not associated with spondylosis, although spondylosis may be associated with type II disease (Levine et al., 2006).
II. Myelography is important for determining the site (or sites) of disc herniation before surgery.
  A. Characteristic findings include extradural spinal cord compression or diffuse spinal cord swelling.
  B. Myelography may be normal when there is lateral or intraforaminal extrusion.
III. CSF analysis is needed before myelography.
IV. CT can accurately identify the site and lateralization of type I disc herniation, without the need for concurrent myelography (Olby et al., 2000).
V. MRI is extremely sensitive in localizing disc disease, extradural compressions, and associated parenchymal disease (Ito et al., 2005; Besalti et al., 2006; Chang et al., 2006)

Differential Diagnosis

I. Type I disc disease
  A. Trauma
  B. Diskospondylitis
  C. Atlantoaxial subluxation
  D. Fibrocartilaginous embolic myelopathy
  E. Meningomyelitis
II. Type II disc disease
  A. Neoplasia affecting the spinal cord
  B. Degenerative myelopathy
  C. Myelitis
  D. Orthopedic disease

Treatment

I. Medical therapy
  A. Medical therapy is reserved for cases with only pain or cases with mild, nonprogressive neurological deficits.
  B. Medical therapy consists of corticosteroid or nonsteroidal antiinflammatory therapy.
    1. Prednisone 0.5 to 1.0 mg/kg PO, IV SID, tapered over 1 to 2 weeks
    2. Carprofen 2.0 mg/kg PO BID
  C. Consider concurrent use of centrally acting muscle relaxants for 1 to 2 weeks (diazepam 0.25 mg/kg PO TID).
  D. Strict exercise restriction is of equal importance and is done concurrently for 4 to 6 weeks.
II. Surgical therapy
  A. Surgery is considered for cases with severe motor dysfunction or recurrent, refractory spinal pain.
  B. Rapid decompression is undertaken to avoid further damage to the spinal cord from continued compression.
  C. Cervical lesions are decompressed via a ventral slotting technique for ventral midline lesions.
  D. Lateralized cervical disc herniation can be treated via a dorsal or modified lateral laminectomy (Rossmeisl et al., 2005).
  E. Thoracolumbar lesions cranial to L4 are decompressed via a dorsolateral hemilaminectomy.
  F. Lesions caudal to L4 are decompressed via a dorsal laminectomy.
  G. Postoperatively, exercise restriction is continued for 4 to 6 weeks.
  H. Bladder care, such as manual expression, catheterization, and pharmacological manipulation is crucial to reduce the potential for detrusor muscle atony and secondary urinary tract infections.
III. Methylprednisolone sodium succinate therapy
  A. High doses of methylprednisolone sodium succinate *(Solu-Medrol)* have been used in acute disc disease as a component of medical or surgical therapy.

B. Contradictory data exist on its efficacy when administered within 8 hours of the onset of the disc extrusion (Olby et al., 1999).
C. The recommended dose is 30 mg/kg IV followed 2 and 6 hours later by 15 mg/kg IV and continued QID for a maximum of 48 hours.
D. Potential side effects include pancreatitis, gastrointestinal hemorrhage, diarrhea, and colonic perforation.

Monitoring of Animal

I. Recovery of nonambulatory dogs varies according to the time interval from onset of signs to surgery, initial severity of neurological dysfunction, and speed of onset of signs.
II. The prognosis is good for dogs with mild to moderate sensory and/or motor deficits.
III. Success rates following cervical disc surgery range from 87% to 100%, whereas success following thoracolumbar disc surgery range from 58% to 95% (Coates, 2000).
IV. Recovery rates with medical therapy in nonambulatory dogs with thoracolumbar disc disease range from 43% to 51% (Coates, 2000).
V. Paraplegic dogs with loss of nociception have a 69% chance of recovering the ability to walk and a 58% chance of recovering nociception, if treated within 48 hours of onset of signs (Olby et al., 2003).
VI. The return of nociception within 2 weeks of surgery is a good prognostic indicator for recovery (Olby et al., 2003; Laitinen and Puerto, 2005)
VII. Recurrence rates after surgery range from 2.7% to 41.7% with thoracolumbar IVDD (Coates, 2000).
VIII. Recurrence rates after surgery range from 10% to 33% with cervical IVDD (Cherrone et al., 2004).
IX. Recurrences usually (96%) develop within 3 years of the initial event (Mayhew et al., 2004).
X. Recurrence rates of 34% to 40% have been reported following conservative therapy, with an average interval to recurrence of 1.7 years (Coates, 2000).

Cervical Spondylomyelopathy

Definition

I. Cervical vertebral malformation or malarticulation results in compression of the cervical spinal cord segments.
II. Synonyms include wobbler syndrome, caudal cervical malformation-malarticulation, cervical spondylopathy, cervical vertebral instability, and cervical vertebral stenosis.

Causes and Pathophysiology

I. The etiology remains undetermined.
II. It commonly affects male, large to giant breeds of dogs.
III. Genetic factors are suspected for the Doberman pinscher and Great Dane.
IV. Clinical disease occurs most frequently in young Great Danes (<2 years) and middle-aged Doberman pinschers (3 to 9 years).
V. The C5-C6 and C6-C7 vertebrae and discs are affected most commonly.
VI. Compression of the spinal cord can be static or dynamic.
A. Great Danes most commonly have a static compression from dorsal or dorsolateral osseous malformation (Abramson et al., 2003).
B. Dobermans most commonly have a ventral dynamic compression, and possibly dorsal soft-tissue hypertrophy.
1. Dorsal compression occurs from hypertrophy of the ligamentum flavum and joint capsules.
2. Ventral compression occurs from annulus hypertrophy and dorsal longitudinal ligament pathology.

Clinical Signs

I. Clinical signs reflect chronic compression of the cervical spinal cord.
II. Onset of clinical signs is gradually progressive over several months or years; however, acute onset is occasionally seen.
III. Initial signs usually begin in the pelvic limbs and progress to tetraparesis.
IV. Signs usually reflect a C1-C5 or C6-T2 myelopathy.
V. Neck pain is uncommon, although the dogs may resist movement of the neck.

Diagnosis

I. Survey radiography may be normal or show a variety of pathologic changes.
II. Radiographic changes include malalignment remodeling and sclerosis of the vertebrae, narrowing of the intervertebral disc (IVD) space, degenerative changes of the articular facets, and spondylosis.
III. Myelography is essential to determine neural involvement, and to identify static and dynamic lesions.
IV. CT or MRI can identify spinal cord compression and atrophy, as well as parenchymal pathology secondary to chronic compression.

Differential Diagnosis

I. Intervertebral disc disease
II. Neoplasia affecting the spinal cord
III. Myelitis
IV. Trauma
V. Diskospondylitis

Treatment

I. Therapy for dogs with mild clinical signs is conservative.
A. Prednisone 0.5 to 1.0 mg/kg PO SID
B. Carprofen 2.0 mg/kg PO BID
C. Exercise restriction
D. Dietary protein restriction in young Great Danes
II. Surgical therapy is often used for moderately to severely affected dogs.
III. The primary objectives of all surgical procedures are to decompress the spinal cord and/or stabilize of the affected vertebrae.
IV. Available surgical techniques include ventral slotting; ventral distraction and fusion, with or without ventral slotting; or continuous dorsal laminectomy (De Risio et al., 2002).

V. The high potential for morbidity and postoperative complications must be considered and discussed with the owner.

Monitoring of Animal

I. Most affected dogs require surgical therapy for long-term relief.
II. The prognosis with surgery depends on the number of sites affected, chronicity, and neurological status of the dog; it can vary from 20% to 80%.
III. Recurrence can occur from implant failure or adjacent disc disease (domino effect).

Spondylosis Deformans

Definition

I. Spondylosis deformans is a chronic, degenerative, noninflammatory disease characterized by the production of osteophytes on the spine that result in the formation of spurs or complete bony ridges across the intervertebral disc space.
II. Ankylosing spondylosis and ankylosing spondylitis are sometimes used synonymously; however, ankylosis is uncommon and the condition is not inflammatory.

Causes and Pathophysiology

I. Osteophyte production occurs in response to degenerative changes in the intervertebral discs.
II. The changes may be secondary to aging and trauma.
III. The disease has been reported in dogs >2 years of age, with 75% of dogs affected to some extent by 9 years of age (Levine et al., 2006).
IV. Because of the high prevalence in female boxers, it is possibly an inherited disease; however, all dog breeds can be affected.
V. The caudal thoracic, lumbar, and lumbosacral spinal segments are most frequently affected in dogs, and the highest incidence occurs at the level of T7-T8 in cats.
VI. An association may exist between radiographically apparent spondylosis and type II disc disease (Levine et al., 2006).

Clinical Signs

I. Compression of the cord or spinal nerves from osteophytic projections into the spinal canal is rare.
II. Rarely, compression of neural tissue may result in a transverse myelopathy or neuropathy.

Diagnosis

I. Diagnosis is based on radiographic identification of osteophyte formation on the ventral surface (in the region of the metaphysis) of the vertebral body or bodies.
II. Osteophytes may occur at either normal or narrowed disc spaces.
III. Myelography can detect associated spinal cord compression and its cause.
IV. CT or MRI helps identify spinal cord compression or foraminal stenosis.

Differential Diagnosis

I. Diskospondylitis
II. Trauma
III. Intervertebral disc disease

Treatment and Monitoring

I. Treatment is usually not necessary.
II. Analgesia and exercise restriction may help dogs that exhibit only discomfort.
III. Surgical decompression may be necessary in cases with clinical signs.
IV. If clinical, the prognosis is guarded owing to the high risk of recurrence.

Spinal Synovial Cyst

Definition

Spinal extradural synovial cysts arise from the articular facets and surrounding connective tissues of the cervical and thoracolumbar vertebrae of dogs.

Causes and Pathophysiology

I. The cysts commonly occur in the cervical spine of young, large-breed dogs and in the thoracolumbar spine of older, large-breed dogs (Dickinson et al., 2001b).
II. Occurrence has been associated with degenerative disc disease and trauma.
III. Increased mechanical stress and joint motion may predispose the thoracolumbar junction to osteoarthritis and synovial cyst formation.
IV. Histopathology of the cyst reveals fibrous connective tissue with a synovial cell lining.

Clinical Signs

I. Clinical signs are consistent with a transverse myelopathy at the site of the lesion.
II. Signs include proprioception deficits, ataxia, paresis or paralysis, often accompanied by paraspinal hyperesthesia.

Diagnosis

I. Degeneration and remodeling of the articular processes are seen at the site of the lesion.
II. Myelography demonstrates spinal cord compression.
III. CT and MRI better define the lesion.

Differential Diagnosis

I. Spinal stenosis
II. Intervertebral disc disease
III. Cervical spondylomyelopathy
IV. Neoplasia

Treatment and Monitoring

I. Surgical decompression of the spinal cord with cyst removal is indicated in dogs with neurological deficits or refractory pain.
II. Recurrence rates are unknown, but surgery usually provides long-term resolution of signs (Dickinson et al., 2001b).

Spinal Stenosis

Definition

I. Spinal stenosis indicates a narrowing of the vertebral canal that may be focal, segmental (affecting several adjacent vertebrae), or generalized (present throughout the vertebral column).
II. Bony impingement on neural elements may be congenital, developmental, acquired, or idiopathic.
III. Compression of neural tissue occurs by nonosseous components of the vertebral canal.
IV. Hypertrophy of the dorsal, longitudinal ligament and the ligamentum flavum may be involved.
V. Disc extrusion or protrusion may occur.

Causes and Pathophysiology

I. Congenital stenosis may occur as a primary lesion or may be seen in association with other congenital vertebral anomalies.
II. Segmental vertebral stenosis occurs in the cranial thoracic spine of several dog breeds (e.g., Doberman pinscher).
III. Developmental stenosis may result from inborn errors of skeletal growth in dogs.
IV. Hypertrophy of the nonosseous components of the vertebral canal has been reported in rottweilers secondary to ligamentous proliferation at C2-C3.

Clinical Signs

I. Clinical signs reflect the location of the lesion, regardless of the precise cause.
II. Onset of signs is usually insidious and progressive.

Diagnosis

I. Diagnosis can be made by survey radiography.
II. Myelography is essential for precise localization of the spinal stenosis.
III. CT or MRI may aid in identification of the location and the extent of soft tissue and parenchymal involvement (Abramson et al., 2003).

Differential Diagnosis

I. Other congenital anomalies of the vertebrae
II. Intervertebral disc disease
III. Cervical spondylomyelopathy
IV. Spinal synovial cysts

Treatment and Monitoring

I. Conservative therapy may be appropriate in mildly affected cases.
   A. Prednisone 0.5 to 1.0 mg/kg PO SID
   B. Carprofen 2.0 mg/kg PO BID
   C. Exercise restriction
II. Decompressive surgery is indicated in animals with persistent pain or progressive neurological deficits.

Osteochondromatosis

Definition

I. A skeletal osteochondroma is a cartilage-capped exostosis arising from the surface of a bone formed by endochondral ossification.
II. Synonyms include multiple or solitary cartilaginous exostoses, hereditary multiple exostoses, multiple osteochondromatosis, diaphyseal aclasis, dyschondroplasia, and hereditary deforming chondrodysplasia.

Causes and Pathophysiology

I. Outgrowths are related to the metaphysis of growing bones in the appendicular and axial skeleton, particularly affecting vertebrae.
II. Feline osteochondromatosis is seen in mature cats (2 to 4 years) in association with feline leukemia virus (FeLV) and feline sarcoma virus.
III. Canine osteochondromatosis is usually seen before 18 months of age and may have an hereditary basis.
IV. Malignant transformation has been reported.

Clinical Signs

I. Clinical signs reflect the location of any vertebral exostosis producing a transverse myelopathy (if there is associated cord compression).
   A. Proprioception deficits
   B. Ataxia
   C. Paresis or paralysis
II. Lesion may result in asymmetrical signs.
III. Pain may be the only clinical sign.

Diagnosis

I. Radiographically, vertebral lesions tend to be circular and smooth with sclerotic borders and are often radiopaque, with radiolucent areas.
II. Myelography is necessary to demonstrate associated spinal cord compression.
III. CT may better define the extent of the bony proliferation within the vertebral canal.
IV. Surgical biopsy is necessary to confirm the diagnosis.

Differential Diagnosis

I. Benign bone tumors: osteomas
II. Neoplasia
III. Osteomyelitis

Treatment

I. Asymptomatic lesions do not require treatment.
II. Surgical excision is indicated if spinal cord compression is causing neurological signs.

Monitoring of Animal

I. For dogs, prognosis varies with severity of signs.
II. The prognosis for dogs having reached skeletal maturity is better than for immature dogs.

III. The prognosis for cats concurrently infected with FeLV is guarded because of increased risk of recurrence.

Degenerative Myelopathy

Definition

I. It is a slowly progressive noninflammatory disease of the spinal cord consisting of axonal degeneration and demyelination.
II. It is synonymous with *chronic degenerative radicular myelopathy.*

Causes and Pathophysiology

I. The etiology remains unknown.
II. Immune-related degeneration has been proposed.
III. German shepherd dogs may have a genetic predisposition.
IV. Pathologic changes have been identified throughout the spinal cord, as well as in the red, lateral vestibular, and dentate nuclei (Johnston et al., 2000).
V. Lesions are most prominent in thoracic segments of the spinal cord.

Clinical Signs

I. Signs are usually nonpainful, insidious, progressive ataxia and paraparesis of the pelvic limbs that ultimately leads to bladder incontinence and paraplegia over 6 to 12 months.
II. It occasionally (10% to 20%) causes reduced patella reflexes from dorsal nerve root involvement (Averill, 1973).
III. Nociception (deep pain perception) is usually unaffected.
IV. It occurs most frequently in the German shepherd dog, and is also recognized in the Pembroke Welsh corgi, boxer, and other breeds.
V. It is rare in cats.
VI. Average age of affected dogs is 9 years old; dogs younger than 5 years are rarely affected (Longhofer et al., 1990).

Diagnosis

I. Tentative antemortem diagnosis is based on classic clinical signs and the exclusion of other diseases.
II. An increased protein level may be found in lumbar CSF.
III. CT and myelography reveal spinal cord atrophy (Jones et al., 2005).

Differential Diagnosis

I. Intervertebral disc disease
II. Neoplasia
III. Myelitis
IV. Orthopedic disease

Treatment and Monitoring

I. Effective treatment has not been reported.
II. Medical treatments that have been advised but not proven include the following:
   A. Aminocaproic acid 500 mg PO TID
   B. N-acetylcysteine 70 mg/kg PO TID for 2 weeks then TID every other day
III. Long-term prognosis is uniformly poor.

Leukoencephalomyelopathy of Rottweilers

Definition

I. It is a demyelinating disorder of the brain and spinal cord.
II. It is reported in rottweilers in United States and Europe.

Causes and Pathophysiology

I. Bilateral symmetrical demyelination occurs in the white matter of the spinal cord, brainstem, and cerebellum.
II. Lateral and dorsal funiculi are commonly affected in the cervical and thoracic spinal cord.
III. The precise etiology is unknown, but the condition may be heritable.

Clinical Signs

I. Onset is at 18 to 42 months of age.
II. Slow progressive clinical signs develop over a 6- to 12-month period.
III. Clinical signs are consistent with a C1-C5 myelopathy and include tetraparesis, hypermetria, and exaggerated segmental reflexes.

Diagnosis

I. Tentative diagnosis is based on signalment, clinical signs, and exclusion of other diseases.
II. Definitive diagnosis is made with histopathology.

Differential Diagnosis

I. Myelitis
II. Neoplasia
III. Cervical spondylomyelopathy
IV. Other degenerative diseases of the spinal cord

Treatment and Monitoring

I. No effective treatment exists.
II. Progression of the disease frequently leads to recumbency and euthanasia.

INFLAMMATORY AND INFECTIOUS DISORDERS

Diskospondylitis/Vertebral Osteomyelitis

Definition

I. Diskospondylitis is infection of the intervertebral disc and adjacent vertebrae.
II. Vertebral osteomyelitis is infection of only the vertebra.
III. Vertebral physitis is infection restricted to the physis.

Causes and Pathophysiology

I. Infection of the intervertebral disc is most commonly associated with *Staphylococcus intermedius*.
II. Other less commonly identified organisms include *Streptococcus* spp., *Escherichia coli*, *Actinomyces* spp., *Brucella canis*, and *Aspergillus* spp.
III. Young German shepherd dogs are predisposed to aspergillosis.

IV. Young basset hounds may be predisposed to diskospondylitis associated with systemic tuberculosis.
V. Infection may arise from hematogenous spread from distant foci of infection, extension of a paravertebral infection, penetrating wounds, surgery, or plant material (grass awn) migration.
VI. Infection causes extradural spinal cord or cauda equina compression.
VII. It is infrequently seen in cats.

Clinical Signs

I. It occurs most commonly in intact male, middle-aged, large- and giant-breed dogs.
II. Single or multiple sites can be infected.
III. The L7-S1 IVD space is most commonly affected.
IV. The thoracolumbar spine is more commonly affected than the cervical spine.
V. Clinical signs reflect the location of the lesion.
VI. Spinal pain is the most frequent initial clinical sign.
VII. Approximately 30% of dogs have signs of systemic illness (e.g., fever, weight loss) (Thomas, 2000).
VIII. Clinical signs may be present for several weeks or months before diagnosis.

Diagnosis

I. Diskospondylitis should be considered in animals with spinal pain or pyrexia.
II. Imaging of the entire spine is done to look for foci of infection.
III. Hematological changes are not usually present unless there is concurrent, systemic infection.
IV. Urinalysis may reveal bacterial or fungal agents.
V. Aerobic, anaerobic, and fungal cultures of blood and urine are positive in up to 75% and 50% of cases, respectively (Thomas, 2000).
VI. Serology for brucellosis is positive in 10% of cases and is performed in all dogs suspected of having diskospondylitis, because of its zoonotic potential (Thomas, 2000).
VII. Definitive diagnosis is usually made with spinal radiography.
    A. Radiographic changes include narrowing of the IVD space and lysis of the vertebral endplates, which are surrounded by sclerosis.
    B. The entire spine must be evaluated.
    C. Radiographs also are used to monitor response to therapy (Shamir et al., 2001).
    D. Radiographic changes often lag behind clinical improvement.
VIII. Myelography is indicated in animals with substantial neurological deficits.
IX. CSF analysis may be normal or may have an increased white blood cell (WBC) count and/or protein content.
X. CT can identify subtle endplate erosion and paravertebral soft-tissue swelling.
XI. MRI can identify inflammatory lesions within the disc space and adjacent soft tissues (Gonzalo-Orden et al., 2000; Cherubini et al., 2004).
XII. Percutaneous, fluoroscopic-guided needle aspiration and culture of the disc space may confirm the etiology in up to 75% of cases (Thomas, 2000).
XIII. Surgical biopsy of the lesion may be warranted in refractory cases.

Differential Diagnosis

I. Spondylosis deformans
II. Vertebral neoplasia
III. Intervertebral disc disease
IV. Meningomyelitis
V. Myositis
VI. Polyarthritis

Treatment and Monitoring

I. Treatment consists of long-term use of an antimicrobial that is effective against the causative organism, as determined by culture and sensitivity testing.
II. If an organism is not cultured, direct empirical therapy against *Staphylococcus* spp. is started.
    A. Cephalexin 20 to 30 mg/kg PO TID
    B. Cefazolin 20 mg/kg IV, IM, SC QID
    C. Amoxicillin 20 mg/kg PO BID
III. Intravenous antimicrobials are given to animals with severe neurological deficits.
IV. Antimicrobials are provided for a minimum of 6 to 8 weeks.
V. Resolution of signs usually occurs within 2 weeks of instituting therapy; however, neurological deficits can persist.
VI. Continued pain is indicative of active disease.
VII. Failure to respond to first-line antimicrobials requires the addition of a second antimicrobial.
    A. Enrofloxacin 5 to 11 mg/kg PO BID (dogs)
    B. Doxycycline 25 mg/kg PO SID (dogs); may cause vomiting
    C. Trimethoprim-sulfadiazine 15 mg/kg PO BID
VIII. Surgical exploration for internal decompression and possible stabilization may be necessary in refractory cases (Auger et al., 2000; Kinzel et al., 2005).
IX. Fluconazole 2.5 to 5 mg/kg PO BID is recommended for *Aspergillus* spp. infections.
X. Ideally, fungal infections are treated based on sensitivity testing.
XI. Nonsteroidal antiinflammatory drugs can be used to alleviate pain.
    A. Carprofen 2 mg/kg PO BID
    B. Ketoprofen 1 mg/kg PO SID for 5 days
XII. The prognosis is very good unless there is an associated endocarditis or a fungal etiology.

Steroid-Responsive Meningitis-Arteritis

Definition

I. It is a noninfectious, inflammatory disease affecting the meninges and associated vasculature.
II. Synonyms include necrotizing vasculitis, juvenile polyarteritis syndrome, corticosteroid-responsive meningitis/

meningomyelitis, aseptic suppurative meningitis, and pain syndrome.

Causes and Pathophysiology

I. The etiology and pathogenesis of this condition are not well understood.
II. The disease may be triggered by an environmental factor that leads to dysregulation of the immune system (Tipold et al., 1999).
III. Immunoglobulin (Ig) A may play a role in the pathogenesis (Tipold et al., 1995).

Clinical Signs

I. It is reported in the beagle, Bernese mountain dog, boxer, German short-haired pointers; it may occur in other breeds.
II. Affected dogs are often young, large-breed dogs between 7 and 18 months old.
III. Signs occur acutely or may follow a waxing and waning course over weeks to months.
IV. Dogs are usually febrile, anorexic, and hyperesthetic, with cervical rigidity.
V. Concurrent immune-mediated polyarthritis or glomerulonephritis may be present.
VI. Neurological deficits, weakness, and ataxia can be seen in the chronic cases.

Diagnosis

I. A marked neutrophilia with a left shift is often seen on a CBC.
II. CSF analysis reveals severe neutrophilic pleocytosis and protein elevation.
   A. Cell counts >100 cells/μL are common.
   B. Neutrophils are nondegenerate.
   C. Marked elevations of protein are common (40 to 350 mg/dL).
   D. Infectious agents are not identified.
III. Blood and CSF cultures, serology, and polymerase chain reaction (PCR) analysis are negative for infectious agents.
IV. Nonspecific elevations of IgA are detected in the CSF and serum (Tipold et al., 1995).

Differential Diagnosis

I. Infectious meningomyelitis
II. Granulomatous meningoencephalomyelitis
III. Diskospondylitis
IV. Polyarthritis, polymyositis
V. Intervertebral disc disease

Treatment and Monitoring

I. Treatment consists of long-term immunosuppressive doses of corticosteroids.
   A. Prednisone 4 mg/kg PO, IV SID for 2 days, *then*
   B. Prednisone 2 mg/kg PO, IV SID for 14 days, and if clinical signs have improved, *then*
   C. Prednisone 1 mg/kg PO SID for 28 days, and if clinical signs have resolved, *then*
   D. Prednisone 0.5 mg/kg PO SID for 2 months, then stopped if dog remains normal
II. Approximately 50% of dogs have a recurrence after discontinuation of the corticosteroids.
III. CSF analysis should be normal before stopping the prednisone.
IV. Additional immunosuppressive therapy may be necessary in some cases (see Chapter 23).

Granulomatous Meningoencephalomyelitis

See Chapter 23.

Distemper Myelitis

Definition and Cause

I. Distemper myelitis is infection of the canine spinal cord with canine distemper virus (CDV).
II. CDV is a Morbillivirus of the family of Paramyxoviridae.
III. Infection of the CNS can cause focal or diffuse lesions in both the grey and white matter.

Pathophysiology

I. Focal or diffuse demyelination can occur in the white matter of the spinal cord.
II. Resultant lesions depend upon host immunity, age, and duration of infection.
   A. Acute polioencephalomyelopathy with glial and neuronal necrosis occurs in immature or immunodeficient dogs.
   B. Chronic leukoencephalomyelopathy with demyelination occurs in older or immunosuppressed dogs.
   C. Demyelination is more frequent in the chronic stages of the disease.
   D. The mechanism by which demyelination occurs may be a primary effect of the virus on glial cells or may occur secondary to immunological mechanisms (Vandevelde and Zurbriggen, 2005).
   E. The white matter of cerebellum, cerebellar peduncles, optic nerves, optic tracts, and spinal cord are most frequently affected.

Clinical Signs

I. Distemper myelitis can occur in any age or breed of dog and vaccination does not always confer protection.
II. The CNS may be the only system affected.
III. Spinal cord signs depend on the location of the lesion, with T3-L3 segments frequently involved.
IV. Signs may be acute or chronic, progressive or relapsing, and occur bilaterally, with occasional asymmetry (Vandevelde and Zurbriggen, 2005).
V. In addition to a transverse myelopathy causing paraparesis and/or plegia, several other signs have been associated with this infection.
   A. Self-mutilation of the limbs and tail
   B. Paraphimosis and/or urinary incontinence
   C. Infrequent paraspinal hyperesthesia from pia arachnoid inflammation
   D. Myoclonus of the limbs that persists during sleep

Diagnosis

I. A tentative antemortem diagnosis is based on history and compatible clinical signs.
II. Diagnosis is often based on exclusion, as a definitive antemortem diagnosis is often difficult to obtain.
III. Hematological and CSF findings are nonspecific.
IV. Positive immunofluorescent assays for CDV antigen on conjunctival tissue, CSF, urine, skin, or blood can facilitate a diagnosis.
V. Analysis of CSF-specific IgG levels and determining the CSF:serum IgG ratio can be used to detect chronic CDV infections.
VI. PCR analysis of CSF and urine is a sensitive method for detecting infection (Amude et al., 2006; Saito et al., 2006).

Differential Diagnosis

I. Infectious meningomyelitis
II. Congenital, inherited neurodegenerative diseases
III. Degenerative myelopathy
IV. Type II disc disease
V. Neoplasia affecting the spinal cord

Treatment and Monitoring

I. Specific antiviral therapy is not available.
II. Administration of modified-live virus vaccines is only effective if given before clinical signs appear.
III. Short-duration prednisone therapy (0.5 to 1.0 mg/kg PO SID to BID for 1 to 3 days) may provide some relief.
IV. The prognosis for recovery is poor.

Infectious Meningomyelitis

Definition

I. Meningitis is inflammation of the meninges.
II. Myelitis is inflammation of the parenchyma of the spinal cord.

Causes

I. Viral causes include feline infectious peritonitis (FIP), FeLV, rabies, CDV, and canine adenovirus.
II. Bacterial causes include *Staphylococcus* spp., *Pasteurella* spp., *Actinomyces* spp., and *Nocardia* spp.
III. Protozoal causes include *Toxoplasma gondii* (dogs, cats), *Neospora caninum* (dogs), and *Sarcocystis* spp. (dogs, cats) (Dubey et al., 2006).
IV. Rickettsial causes include *Rickettsia rickettsii* (Rocky Mountain spotted fever [RMSF]) and *Ehrlichia canis*.
V. Fungal causes include *Cryptococcus neoformans*, *Blastomyces dermatitidis*, *Histoplasma capsulatum*, and *Coccidioides immitis*.

Pathophysiology

I. FIP causes granulomatous inflammation of the meninges, ependymal cells, and choroid plexus.
II. FeLV-associated myelopathy has been reported in chronically infected cats (Carmichael et al., 2002).
III. Bacteria and fungal diseases can infect the spinal cord through extension, penetration, and hematogenous routes.
IV. *T. gondii* and *N. caninum* can infect the spinal cord, peripheral nerves, and muscles.
V. Rickettsial disease affects the spinal cord through immune-mediated vasculitis.

Clinical Signs

I. The hallmark of infectious diseases is multifocal clinical signs.
II. Neurological signs reflect the region of the spinal cord affected.
III. Signs include paresis and ataxia.
IV. Often there are concurrent, systemic signs of inflammation.
V. Viral diseases may produce the following:
   A. FIP
      1. Cats can be affected at any age but are often between 6 months and 5 years of age.
      2. Signs are insidious and may reflect multiorgan involvement.
      3. The CNS signs may be focal, diffuse, or multifocal.
      4. Intracranial signs are more common than spinal signs.
      5. Clinical signs reflect the location of the pathology.
   B. FeLV causes hyperesthesia and progressive paraparesis to paralysis.
VI. Bacterial and fungal diseases
   A. Signs reflect the location and severity of the pathology.
   B. Neurological signs are often acute and rapidly progressive, but occasionally fungal infections can be slowly progressive.
   C. Fever occurs intermittently and is more likely with concurrent bacteremia or disseminated fungal infection.
VII. Protozoal diseases are characterized by the following:
   A. *T. gondii*
      1. Affected animals usually have signs of progressive multifocal disease.
      2. A focal transverse or diffuse myelopathy may be seen.
      3. Neurological signs reflect the location of the infection.
      4. In dogs <1 year of age, a syndrome of progressive paralysis and rigid extension of one or both pelvic limbs may be seen, as well as muscle contracture of the affected limbs.
      5. Concurrent signs of systemic infection include fever, lymphadenopathy, pneumonia, gastrointestinal disease, and chorioretinitis.
   B. *N. caninum*
      1. Puppies are more severely affected than adult dogs.
      2. Young dogs develop an ascending paralysis.
      3. Concurrent signs include dysphagia and jaw paresis.
VIII. Rickettsial disease may affect dogs of all ages.
   A. Mental depression is the most common clinical finding.
   B. Concurrent, systemic signs include fever, anorexia, lymphadenopathy, dyspnea, diarrhea, vomiting, hemorrhagic diatheses, and joint pain.

C. Clinical signs of spinal disease may be accompanied by those of intracranial disease and reflect the location of the lesion.
D. Cervical rigidity and pain may occur with RMSF.

Diagnosis

I. Viral diseases
  A. FIP
    1. Confirmation antemortem is difficult.
    2. Biopsy of affected tissue is necessary for definitive diagnosis.
    3. CSF analysis reveals a neutrophilic pleocytosis and markedly elevated protein levels.
    4. Indicators of disease are a positive coronavirus titer or PCR analysis in CSF, a high serum total protein concentration, and findings on imaging that include CNS periventricular enhancement, ventricular dilation, and hydrocephalus.
  B. FeLV-associated disease can only be definitively confirmed postmortem.
II. Bacterial and fungal diseases
  A. A diagnosis of bacterial or fungal meningomyelitis is made by isolating a causative organism in CSF.
  B. CSF cytology may demonstrate the presence of organisms.
    1. Classically bacterial diseases produce marked neutrophilic pleocytosis, and fungal diseases are associated with a mixed mononuclear and polymorphonuclear pleocytosis frequently with eosinophils.
    2. Aerobic, anaerobic, and fungal cultures are performed in suspected cases.
  C. Serology may be useful for diagnosis of fungal infections.
III. Protozoal diseases
  A. Tentative antemortem diagnosis is based upon serological evidence, compatible clinical signs, and positive response to treatment.
  B. CSF analysis shows a mixed-cell or mononuclear pleocytosis, and elevated protein levels.
  C. PCR of the CSF may confirm the diagnosis (Schatzberg et al., 2003).
IV. Rickettsial diseases
  A. CSF analysis may be normal or show mild increases in protein and pleocytosis (predominantly lymphocytes).
  B. Serology can confirm the diagnosis.

Differential Diagnosis

I. Steroid-responsive meningitis-arteritis
II. Diskospondylitis
III. Polyarthritis, polymyositis
IV. Cervical disc disease
V. Neoplasia

Treatment and Monitoring

I. Viral diseases
  A. Treatments include immunosuppression and prevention of secondary bacterial infections.
    1. Give prednisolone 2 to 4 mg/kg PO, IV SID.
    2. Cyclophosphamide 2 mg/kg PO SID for 4 consecutive days of each week has also been used.
    3. Give broad-spectrum antibiotics for 2 to 3 weeks.
  B. Despite aggressive therapy, the prognosis for FIP infection is poor.
II. Bacterial and fungal diseases
  A. Use high-dose, IV bactericidal drugs for at least 4 to 6 weeks in bacterial infections.
    1. Penicillin and penicillin derivatives in high doses (10 to 30 mg/kg IV, IM, PO BID to QID) are recommended for gram-positive infections for the first week.
    2. Most cephalosporins penetrate the CNS poorly.
      a. Several third-generation cephalosporins may reach therapeutic levels in the CNS and are advisable for gram-negative infections.
      b. Drugs to consider include cefotaxime 30 to 40 mg/kg IV, IM, SC TID to QID and ceftazidime 20 to 50 mg/kg IV BID to TID.
    3. Use metronidazole 10 mg/kg IV, PO TID for the treatment of anaerobic infections.
    4. Trimethoprim-sulfadiazine 15 mg/kg SC, PO BID penetrates the CNS effectively.
  B. Surgical drainage of an abscess may be necessary.
  C. Prognosis for bacterial infections is guarded.
  D. Several antifungals may be tried.
    1. Amphotericin B
      a. Dose is 0.15 to 0.5 mg/kg IV three times a week.
      b. When the total dose reaches 4 to 6 mg/kg, a maintenance dose of amphotericin is used (0.15 to 0.25 mg/kg IV once monthly), and rifampin 10 to 20 mg/kg PO TID is started.
    2. Ketoconazole (poor CNS penetration)
      a. Initial dose is 15 to 20 mg/kg PO BID for at least 2 to 3 months or until remission
      b. Maintenance dose is 10 mg/kg PO SID.
    3. Fluconazole
      a. Option 1: 2.5 to 5.0 mg/kg PO, IV BID
      b. Option 2: 5 to 10 mg/kg PO, IV SID for 56 to 84 days
  E. Fungal infections are difficult to eliminate from the CNS, and the prognosis is poor.
III. Protozoal diseases
  A. Early treatment for 2 to 4 weeks is essential.
  B. Trimethoprim-sulfadiazine 15 mg/kg SC, PO BID or ormetoprim-sulfadimethoxine 15 mg/kg PO BID, and clindamycin 15 mg/kg IV, PO for 28 days may improve clinical signs.
  C. Pyrimethamine 0.25 to 0.5 mg/kg PO SID for 28 days can be used adjunctively, but bone marrow suppression can occur in young animals.
  D. Prognosis is poor with rapidly progressive disease, pelvic limb hyperextension, and disease chronicity.
IV. Rickettsial diseases
  A. Give tetracycline 22 to 30 mg/kg PO TID for at least 14 days.
  B. Doxycycline 10 to 20 mg/kg PO BID is often used because of its excellent CNS penetration.
  C. Chloramphenicol 15 to 30 mg/kg PO TID for 7 days has good CNS penetration and is indicated in young (<6 months) dogs to avoid dental staining.

VASCULAR DISORDERS

Ischemic Myelopathy

Definition

I. Ischemic myelopathy involves vascular compromise of the spinal cord that often progresses to local infarction.
II. The term is often used synonymously with fibrocartilaginous embolic myelopathy (FCEM), which denotes a specific cause of the ischemia.

Causes and Pathophysiology

I. Acute spinal cord infarction can be secondary to FCEM, neoplastic emboli, and intravascular coagulation.
II. FCEM is characterized by acute spinal cord infarction from embolization of fibrocartilage identical to that of the nucleus pulposus.
   A. Many theories exist as to the pathophysiology of the embolization, but none are proven.
   B. Fibrocartilaginous emboli occlude arteries and/or veins of the leptomeninges and spinal cord parenchyma.
   C. Achondrodystrophic, medium- to large-breed dogs are predisposed.
   D. There is an increased incidence in miniature schnauzers.
   E. It is infrequently reported in cats (Mikszewski et al., 2006).

Clinical Signs

I. Classically, clinical signs are peracute in onset, nonprogressive, nonpainful, and often asymmetrical.
II. Transverse myelopathy occurs, with signs compatible with the location of the infarction.
   A. Proprioception deficits
   B. Ataxia
   C. Paresis or paralysis
III. Thoracolumbar signs are more common than cervicothoracic, but signs can occur in any region and often involve the intumescence.
IV. Clinical onset is frequently associated with trauma or exercise.
V. Maximal neurological deficits develop within 12 hours and are then nonprogressive.
VI. Severe signs are accompanied by loss of nociception (deep-pain perception).

Diagnosis

I. Tentative diagnosis is based upon history, signalment, and compatible clinical signs.
II. Myelography may demonstrate intramedullary spinal cord swelling in the early stages (Gandini et al., 2003).
III. MRI documents parenchymal pathology (Abramson et al., 2005; Mikszewski et al., 2006).
IV. CSF may reveal mild pleocytosis or a normal cell count with an elevated protein level (albumino-cytological dissociation).

Differential Diagnosis

I. Trauma of the spinal cord
II. Intervertebral disc disease
III. Myelitis
IV. Neoplasia affecting the spinal cord
V. Hemorrhage of vessels in or around the spinal cord

Treatment and Monitoring

I. There is no specific medical therapy for FCEM; however, the use of high-dose corticosteroid therapy has been considered.
   A. The dose of methylprednisolone recommended is 30 mg/kg IV followed 2 and 6 hours later by 15 mg/kg IV and continued QID for a maximum of 48 hours.
   B. Potential side-effects include pancreatitis, gastrointestinal hemorrhage, diarrhea, and colonic perforation.
II. Clinical improvement depends on the severity of sensorimotor dysfunction.
III. A poor prognosis has been correlated with lack of improvement within 14 days, involvement of the intumescences, and a lack of deep pain perception.
IV. Supportive care, physiotherapy, and hydrotherapy may aid in recovery.

NEOPLASIA

Vertebral and Spinal Tumors

Definition and Causes

I. Primary and secondary tumors can affect the vertebrae, meninges, and spinal cord.
II. Feline lymphoma may be associated with FeLV infection.
III. Neoplasia can be classified as extradural, intradural-extramedullary, or intramedullary.
   A. Extradural tumors can be primary or secondary.
      1. Primary vertebral tumors: fibrosarcoma, osteosarcoma, chondrosarcoma, hemangiosarcoma, myeloma
      2. Secondary vertebral tumors: mammary, prostatic, thyroid carcinomas, malignant melanoma, metastatic osteosarcoma
      3. Epidural tumors: lymphoma, metastatic tumors
   B. Intradural extramedullary tumors include meningioma, peripheral nerve sheath tumor, lymphoma, and nephroblastoma.
   C. Intramedullary tumors include astrocytoma, oligodendroglioma, ependymoma, and metastases.
IV. Primary and secondary extradural tumors are more common than intradural extramedullary tumors; intramedullary tumors are rare.
V. Spinal neoplasia accounts for 27% of all spinal disease in cats (Marioni-Henry et al., 2004).
   A. Epidural lymphoma is the most common spinal tumor of cats.
   B. Meningioma is the most common benign, nonlymphoid tumor of cats (Rossmeisl et al., 2006).
   C. Osteosarcoma is the most common malignant, nonlymphoid tumor of cats.

Pathophysiology

I. Spinal tumors occur most commonly in animals >5 years old (Dernell et al., 2000).

A. Dogs with spinal nephroblastoma are usually 4 to 38 months old (Summers et al., 1988).
B. Median age for cats with spinal lymphoma is 4.5 years (Marioni-Henry et al., 2004).

II. Any breed can be affected.
A. All classes of spinal neoplasia occur more frequently in large-breed dogs.
B. German shepherd dogs and golden retrievers may be predisposed to spinal nephroblastoma.

III. Spinal neoplasia occurs throughout the spinal cord and vertebral column.
A. Meningiomas most commonly occur in the cervical spine.
B. Nephroblastoma occurs most commonly between T10 and L2 vertebrae.
C. Feline lymphoma has a predilection for the thoracic and lumbar spinal cord.

Clinical Signs

I. Clinical findings depend on the location of the tumor.
II. Pain is the most common initial clinical signs.
III. The most common sign is a transverse myelopathy that is often bilateral, but can be asymmetrical.
A. Proprioception deficits
B. Ataxia
C. Paresis and paralysis
IV. Signs may progress over 1 week to 1 year.
V. An acute onset may occur from pathologic fractures, hemorrhage, or ischemia.

Diagnosis

I. Tentative diagnosis is based on clinical, radiographic, and CSF analysis, and on advanced imaging findings.
II. Thoracic radiography (three views) and abdominal ultrasonography are performed to identify primary or secondary neoplastic lesions.
III. Radiography can identify bony lesions of the vertebrae.
A. Bone lysis is most common finding associated with a vertebral tumor.
B. Punched-out lytic lesions in multiple vertebrae commonly occur with multiple myeloma.
C. Usually only one vertebra is involved (unless secondary).
IV. CSF analysis often reveals nonspecific changes.
A. The CSF may be normal or may have elevated protein levels.
B. Mild to moderate neutrophilic pleocytosis may occur with tumors affecting the leptomeninges.
C. Tumor cells are rarely identified, except with lymphoma.
V. Advanced imaging may be useful.
A. Myelography helps to differentiate intramedullary, intradural-extramedullary, and extradural lesions.
B. CT is used to identify bony lesions.
C. MRI is the best imaging modality for spinal neoplasia.
1. MRI allows specific determination of extramedullary or intramedullary lesions (McConnell et al., 2003).
2. Intravenous contrast administration helps determine the soft-tissue and osseous extent of the tumor.
3. MRI determines extent of disease and assists with surgical planning (McDonnell et al., 2005).

VI. Definitive diagnosis requires histopathologic interpretation of biopsy specimens.

Differential Diagnosis

I. Intervertebral disc disease
II. Diskospondylitis, osteomyelitis
III. Meningomyelitis
IV. Degenerative myelopathy
V. Spinal trauma

Treatment and Monitoring

I. Long-term prognosis is poor, with survival time often inverse to the severity of neurological deficits (Dernell et al., 2000).
II. Medical therapy consists of the following:
A. Prednisone 0.5 to 1.0 mg/kg PO SID can be used palliatively.
B. Chemotherapy may be tried for certain tumors.
1. Tumors amenable to chemotherapy include lymphoma and multiple myeloma.
2. In cats with spinal lymphoma, remission rate is 50%, with complete remission duration of 14 weeks, and partial remission duration of 6 weeks (Spodnick et al., 1992).
3. Long-term control of solitary plasmacytomas can be achieved with chemotherapy and radiation (Rusbridge et al., 1999).
C. Radiation therapy may be tried as primary, adjunctive, or palliative therapy (Dickinson et al., 2001a).

III. Surgical therapy involves surgical decompression of the spinal cord and debulking of the tumor.
A. Most vertebral tumors are not surgically respectable.
B. The median survival time of dogs with vertebral tumors was 135 days following multimodality therapy that included surgical resection (Dernell et al., 2000).
C. Surgical resection of meningiomas in dogs may lead to remissions of >6 months, and adjunctive radiotherapy may increase remission time to approximately 15 months (Levy et al., 1997).
D. Cytoreductive surgery of malignant, nonlymphoid spinal tumors in cats provides a median survival time of 110.5 days (Rossmeisl et al., 2006).
E. Cytoreductive surgery of benign, nonlymphoid spinal tumors in cats provides a median survival time of 518 days (Rossmeisl et al., 2006).
F. Intramedullary tumors are often difficult to excise without damage to the surrounding parenchyma (Sanders et al., 2002).

TRAUMATIC DISORDERS

Spinal Cord Trauma

Definition

I. Injury to the spinal cord may be caused by endogenous (intervertebral disc disease) or exogenous (vehicular trauma) factors.

II. Direct trauma to the spinal cord results in primary and secondary injuries.
III. Direct trauma to the spinal cord (concussion) may be followed by sustained compression, distraction, or both.

Causes

I. Type I intervertebral disease
II. Vertebral fractures and luxations
III. Fibrocartilaginous embolism
IV. Spinal instability: atlantoaxial subluxation

Pathophysiology

I. Acute spinal cord injury is often caused by a combination of events that can include concussion, compression, ischemia, and laceration (primary injuries).
II. Each of these primary injuries can lead to secondary injury, which is a series of biochemical and metabolic events that expand the primary zone of tissue necrosis.
III. Most secondary injuries occur within 24 hours and contribute to clinical deterioration.

Clinical Signs

I. Neurological examination is performed (carefully) to localize the site of the trauma before any sedation or analgesia is done.
II. Concurrent trauma involving other organ systems is frequently present and must also be assessed.
III. Neurological signs reflect the site of injury.
IV. Progressive hemorrhagic myelomalacia is suspected with continued deterioration.

Diagnosis

I. The diagnosis is made based on history and supportive clinical signs.
II. Thorough evaluation of the entire animal is essential to identify concurrent abnormalities.
III. Survey spinal radiography commonly delineates traumatic luxation and/or subluxation and fractures of the vertebral column.
   A. Lateral survey radiographs are taken of the whole spine before manipulation of the animal.
   B. General anesthesia maybe necessary for accurate positioning, but is delayed until the animal is stabilized.
   C. Sedation or analgesia may assist with positioning, but increases the risk of neurological deterioration secondary to paravertebral muscle relaxation.
IV. Advanced imaging is required to assess nervous tissue.
V. Myelography assists in assessing the degree of associated spinal cord compression.
VI. CT is invaluable in identifying bony defects that may not be apparent on survey radiography.
VII. MRI provides information about spinal cord compression, extradural hemorrhage, and parenchymal structure, but may not provide much detail about fractures and luxations.

Differential Diagnosis

I. Type I intervertebral disc disease
II. Pathologic fracture secondary to neoplasia
III. Fibrocartilaginous embolic myelopathy
IV. Ischemic neuromyopathy

Treatment and Monitoring

I. Initiate stabilization of the cardiovascular and respiratory systems before assessing and treating the spinal injury.
II. Immobilize the spine to prevent further vertebral displacement.
III. Delay administration of analgesia or sedation until the initial assessments, neurological examination, and diagnostics have been performed.
IV. Medical treatment is indicated for animals with mild clinical signs and no evidence of vertebral instability.
   A. Methylprednisolone sodium succinate
      1. The dose recommended is 30 mg/kg IV followed 2 and 6 hours later by 15 mg/kg IV and continued QID for a maximum of 48 hours.
      2. Potential side effects include pancreatitis, gastrointestinal hemorrhage, diarrhea, and colonic perforation.
   B. Cage rest for 6 weeks.
V. If spinal instability is detected but the owner declines surgery, an external splint is required to immobilize the area for 6 to 8 weeks.
VI. Surgical treatment is indicated in cases with instability, vertebral malalignment, or spinal cord compression.
   A. Laminectomy ± durotomy are necessary for decompression.
   B. Realignment and fixation can be achieved using Steinmann pins, screws, and polymethylmethacrylate, or vertebral body plates.
   C. Postoperative care involves exercise restriction, analgesia, soft bedding, and management of bladder evacuation.
   D. Physiotherapy and hydrotherapy may also help recovery.
   E. Prognosis depends on the degree of sensorimotor loss and chronicity of lesion at time of surgery, in addition to severity of any systemic disorders.

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