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DIFFUSE NEUROMUSCULAR DISORDERS

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Acute flaccid quadriparesis or quadriplegia, chronic progressive quadriparesis and episodic weakness which worsens with exercise are 3 presenting complaints for some common diffuse neuromuscular disorders in dogs and cats.

ACUTE FLACCID QUADRIPARESIS OR QUADRIPLEGIA

Paresis which initially involves the pelvic limbs then the thoracic limbs and results in quadriparesis or quadriplegia over 24-72 hours is a common complaint in dogs, but rare in cats. In mild quadriparesis, the patellar reflexes may be the only spinal reflexes depressed. Signs can progress and animals are in lateral recumbency with loss of all pelvic and thoracic limb spinal reflexes (flaccid quadriplegia).

Cranial nerve involvement may result in weakness of eyelid closure, a change in voice and dysphasia. In severe cases, respiratory assistance may be necessary due to phrenic and intercostal nerve dysfunction.

Differential Diagnosis of Acute Flaccid Quadriparesis or Quadriplegia

1. Tick Paralysis
2. Acute Idiopathic Polyradiculoneuritis
3. Myasthenia gravis (acute crisis or medication overdose)
4. Botulism
5. Coral Snake toxicity

Tick paralysis and acute idiopathic polyradiculoneuritis are most common in dogs. Dermacenter variabilis and andersoni are 2 species of ticks that may secrete a neurotoxin which produces neuromuscular junction blockade. There may be little or no response when the nerve is electrically stimulated during an electromyographic (EMG) examination.

Since affected dogs recover from tick paralysis 24-48 hours after removal of a tick, all dogs with acute flaccid quadriparesis, should be thoroughly examined and all ticks removed. Care should be taken if a dip is applied to remove ticks as organophosphates may worsen the neuromuscular junction dysfunction.

Acute idiopathic polyradiculoneuritis also referred to as "coonhound paralysis" is difficult to differentiate from tick paralysis on the basis of the neurological examination findings. During EMG examination, fibrillation potentials and trains of positive sharp waves are often found diffusely in the limb and paravertebral musculature. These abnormal potentials are not found acutely in the other diseases on the differential diagnosis list. The nerve conduction velocity and evoked response are often normal.

If there is no clinical improvement in a dog with flaccid quadriparesis 24 hours following tick removal, acute polyradiculoneuritis is the most likely diagnosis. The dog does may not be a coonhound or be bitten by a raccoon. Affected dogs are bright and alert and may still wag their tail. Some dogs have hyperesthesia, not reported with tick paralysis or the other causes of acute flaccid quadriplegia.

An immunologic disorder of ventral nerve roots is the suspected cause of acute polyradiculoneuritis. Some clinicians administer glucocorticoids for 1 week to prevent progression to respiratory paresis and dysphagia. Assisted ventilation may be necessary for 24-72 hours. After approximately 1 week, affected dogs begin to improve and may be ambulatory within another 1-4 weeks. Once recovery is complete, reoccurrences can be seen months or years later.

Dogs and cats with myasthenia gravis have exercise induced weakness prior to the onset of a crisis of acute flaccid quadriparesis. Some myasthenic animals on excessive doses of pyridostigmine may develop a cholinergic crisis which appears the same. Animals are in lateral recumbancy with depressed myotatic reflexes. If an untreated myasthenic crisis is suspected, 0.1 mg (cats) or 0.5-1 mg (dogs) intravenous edrophonium chloride (Tensilon) will produce dramatic improvement and animals will become ambulatory for 2-5 minutes. If a cholinergic crisis is suspected, atropine is given. Diagnosis, treatment and prognosis of myasthenia gravis is discussed further with episodic weakness which worsens with

exercise.

Clostridium botulinum produces a neurotoxin which reduces neuromuscular junction transmission and causes acute flaccid quadriparesis or quadriplegia. Known or potential exposure to affected carrion or spoiled meat is often in the history. Unlike dogs with tick paralysis and acute polyradiculoneuritis, dogs with botulism are very depressed, hypothermic and have bradycardia and mydriasis. If botulism is highly suspected, treatment with penicillin and supportive care are provided. Serum from affected dogs injected intraperitoneally into healthy mice will cause flaccid quadriplegia in the mouse within 24-48 hours. In severe cases of botulism, dogs die in 24 hours. Mildly affected dogs recover in 3-4 weeks.

The neurotoxin from the coral snake also produces a neuromuscular junction blockade. Affected animals are depressed. Fatal hemolysis may also occur. There is little or no response to electrical stimulation of the nerves. Ventilatory support may be necessary. Coral snake antivenin (1-2 vials repeated in 4-6 hours if necessary) may be given immediately. If a hemolytic crisis does not occur, recovery from paraysis begins within 72 hours and animals will be normal in 1 week.

CHRONIC PROGRESSIVE FLACCID QUADRIPARESIS

Chronic progressive flaccid quadriparesis may insidiously develop in dogs and cats weeks or months. Often the pelvic limbs are weaker than the thoracic limbs. In some cases, the patellar reflexes may be the only spinal reflexes depressed or absent. In severe cases, depression or loss of all myotatic reflexes occurs. Generalized muscle atrophy of the limbs, trunk and head may also be present. Generalized muscle atrophy and depressed spinal reflexes are more characteristic of neuromuscular disease (polyneuropathy more than polymyopathy) than spinal cord disease. Involvement of cranial nerves and muscles produce problems with prehension, swallowing, phonation chewing and eyelid closure.

Differential Diagnosis of Chronic Progressive Flaccid Quadriparesis

Polyneuropathy

1. Familial polyneropathy and ventral horn cell degeneration
2. Hyperinsulinism polyneuropathy
3. Diabetes mellitus polyneuropathy
4. Hypothyroid polyneuropathy/polymyopathy
5. Toxic polyneuropathy (lead, organophosphates and drugs)
6. Paraneoplastic polyneuropathy
7. Chronic immune-mediated polyneuropathy
8. Idiopathic polyneuropathy and distal axonopathy

Polymyopathy

1. Hyperadrenocorticism
2. Polymyositis
3. Nutritional myodegeneration
4. Muscular dystrophy

If animals with chronic flaccid quadriparesis have conscious proprioceptive deficits or abnormal skin sensation, then polyneuropathy is more likely than polymyopathy. Serum muscle enzyme levels are normal in polyneuropathies, but can also be normal in some cases of chronic polymyopathy.

Positve sharp waves and fibrillation potentials and slow motor nerve conduction velocities are often found on the EMG examination in polyneuropathies. If only the ventral horn cell, ventral root or distal axon is diseased the motor nerve conduction velocity will be normal. These latter disorders can be difficult to differentiate from polymyopathies without a muscle biopsy. Grouped muscle fiber atrophy with primary involvement of type II and less involvement of type I muscle fibers are characteristic features of a muscle biopsy from chronic polyneuropathy.

If a polyneuropathy is suspected, serum glucose should be evaluated for hypoglycemia or hyperglycemia (diabetes mellitus). If hypoglycemia is present, an insulin assay should be performed and an insulin glucose ratio established to evaluate for an insulin secreting neoplasia. A thyroid stimulating hormone (TSH) test should be preformed to evaluate thyroud function. In chronic lead and

organophosphate toxicity, the serum lead and cholinesterase levels may be normal. Any other chronic drug exposure should be considered for the potential to cause polyneuropathy. Vincristine and other drugs for chemotherapy may cause polyneuropathy.

Chest and abdominal radiographs should be obtained to rule out pulmonary or other neoplasia that might have an associated paraneoplastic syndrome. Antinuclear antibody (ANA) and Coombs test may be positive in some immune mediated polyneuropathies. In cases of idiopathic chronic polyneuropathy and distal axonopathy no underlying cause of disease can be detected, but some animals may be responsive to glucocorticoids. Others have a waxing and waning disease course. For some animals, the clinical signs progress regardless of therapeutic attempts with corticosteriods and other immunosuppressive drugs.

A muscle biopsy is used to confirm the presence of muscle disease and to characterize the type of disease process. Infiltration of lymphocytes and plasma cells between muscle fibers is characteristic of polymyositis. Immune-mediated polymyositis is the most common form of polymyositis. A serum ANA may be positive. Circulating antimuscle antibody may be detected with indirect immunofluorescent studies on muscle biopsy specimens. Some cases of systemic lupus erythematosus have an associated polymyositis.

Animals with acute polymyositis often respond well to glucocorticoid therapy, but recovery may be incomplete with more chronic forms. Prednisone orally 1-2 mg/kg daily for 1-2 weeks then reduced to alternate day therapy may control the progression of the atrophy. Physical examinations and muscle enzyme determinations should be periodically reevaluated to monitor for recurrences, if glucocorticoid therapy is discontinued. Elevated muscle enzymes indicate recurrence of the disease. If severe muscle atrophy and fibrosis are present, the prognosis for recovery is poor.

Dogs with polymyositis secondary to Toxoplasma gondii have positive IgG and IgM serum titers and large numbers of organisms may be found among the granulomatous reaction seen on histologic examination of the muscle. The long term prognosis of toxoplasmosis may be poor, but treatment may be attempted with trimethoprim sulfadiazine or clindamycin. Neospora caninum can produce a severe polymyositis and polyradiculitis which results in severe muscle atrophy and fibrosis along with paraplegia in young dogs. The prognosis for recovery is grave even with antibiotic therapy.

Chronic polymyopathy secondary to hyperadrenocorticism and chronic corticosteroid therapy have both been described in dogs. Resting cortiso and levels after ACTH stimulation are evaluated to make the diagnosis. Correction of the underlying cause of hyperadrenocorticism may halt the progression of the disease process, but complete recovery depends upon the degree of muscle damage.

Nutritional myodegeneration also referred to as white muscle disease may be suspected from the muscle biopsy results, but is rare in dogs and cats. Response to vitamin E therapy may be dramatic.

Muscular dystrophy occurs as a congenital disorder in dogs and cats. Affected animals are weak and may have muscle hypertrophy instead of atrophy. The course is progressive and the prognosis poor.

There are some rare familial polyneuropathies in Golden Retriever, German Shepherd and Boxer dogs and disorders with ventral horn cell degeneration in Brittany Spanial and Rottweiler dogs which progress and have a poor prognosis.

EPISODIC WEAKNESS WHICH WORSENS WITH EXERCISE

Episodic weakness which worsens with exercise is characteristic of cardiopulmonary disorders and metabolic disorders which secondarily affect the nervous system as well as primary neuromuscular disease. The cardiovascular system should be evaluated by electrocardiography with a Holter monitor if necessary during an episode of weakness. Metabolic disorders causing weakness ie. anemia, hypoglycemia, hypocalcemia, hyperkalemia, hypokalemia and hypernatremia may be detected on a complete blood count and serum chemistry profile. If the heart and metabolic proflie is normal, neuromuscular disease should be considered. Animals are weak, but spinal reflexes are usually normal. Cats may also exhibit ventral neck flexion due to weakness.

Differential Diagnosis for Weakness which Worsens with Exercise

1. Polymyositis

2. Myasthenia gravis
3. Hypokalemic polymyopathy (cats)
4. Organophosphate toxicity
5. Hyperthyroidism (cats)
6. Hypothyroidism (dog)

Megaesophagus and dysphagia are common with polymyositis and myasthenia gravis and affected animals may be presented for aspiration pneumonia. Some dogs with hypothyroidism may have megaesophagus as well. Aspiration pneumonia is a severe and fatal complication and must be avoided if possible and treated aggressively if present.

If serum muscle enzymes are elevated, then polymyositis or hypokalemic polymyopathy are likely. The serum blood urea nitrogen (BUN) and creatinine levels will be elevated and the serum potassium will be low in cats with hyperkalemia polymyopathy. Oral supplementation with potassium gluconate (4-8 meq every 12 hours) will improve both the polymyopathy and renal dysfunction, but must be continued indefinately.

Lymphocyte infiltration observed on a muscle biopsy is used to confirm a diagnosis of polymyositis. Toxoplasma gondii organisms may be observed. Weakness due to immune-mediated polymyositis responds well to oral prednisone (1-2mg/kg). Muscle enzyme levels return to normal and may be evaluated for recurrent elevations as prednisone is reduced over the following weeks. If organisms are observed on the muscle biopsy or serum IgG and IgM are positive for toxoplasma gondii, sulfadiazine or clindimycin therapy is begun and glucocorticoids are avoided.

If serum muscle enzymes are normal or if exposure to organophosphates is possible, a serum cholinesterase level should be evaluated. Normal values are usually above 1000 IU. Neuromuscular weakness occurs when levels reach 500 IU or less. Diphenhydramine (Benedryl) intramuscularly or oral (2-4 mg/kg every 8-12 hours) may be necessary for 1-3 weeks to control the weakness. Recovery is usually complete by 3 weeks and medication can be discontinued.

Thyroid hormone levels should be evaluated to detect hypothyroidism and hyperthyroidism. Some cats with hyperthyroidism have elevated muscle enzymes.

Dogs and cats with myasthenia gravis often are presented with exercise induced weakness. The gait may become progressively stilted especially in the thoracic limbs until the animals crawl and are unable to walk. Affected animals often have weak eyelid closure. Congenital myasthenia gravis has been described in Jack Russell terriers, Smooth haired fox terriers and Springer spaniels with clinical signs manifested by 6-8 weeks of age. Acquired myasthenia gravis is seen in adult dogs and cats and is especially prevalent in German Shepherds. Dogs with acquired myasthenia gravis often have megaesophagus which can be a permanent problem.

Improved strength following intravenous edrophonium chloride (Tensilon) administration may support the diagnosis of myasthenia gravis, but cases of polymyositis and other neuromuscular disorders may appear stronger as well. Serum antibodies to acetylcholine receptors may be identified in dogs and cats with acquired myasthenia gravis. A decremental response to repetitve nerve stimulation is often seen on EMG examination in cases of myasthenia gravis. An improvement of the decrement following intravenous edrophonium chloride further supports the diagnosis of myasthenia gravis.

Myasthenia gravis is treated with oral pyridostigmine (10-15mg twice daily cats) and (15-60mg twice daily dogs) as needed to control weakness. If weakness does not improve, oral prednisone 1-2 mg/kg may be given. Aspiration pneumonia can be a severe and fatal complication. If weakness is detected and treated early and aspiration pneumonia is absent, the prognosis is good. Over the following months, the pyridostigmine dose may be reduced and then discontinued, if weakness does not return. Complete remissions and recurrences are seen.

SEIZURES AND BEHAVIORAL ABNORMALITIES IN DOGS AND CATS WITH NEUROLOGICAL DYSFUNCTION

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DIFFERENTIAL DIAGNOSIS OF SEIZURES

Seizures in dogs and cats are due to congenital disorders, meningoencephalitis, metabolic dysfunction, toxicities, trauma, vascular disorders, neoplasia and idiopathic epilepsy. Hydrocephalus, lysosomal storage disorders and lissencephaly are congenital disorders which usually cause seizures by 6 months of age.

Meningoencephalitis caused by canine distemper or feline infectious peritonitis viruses is most common in young animals, but can occur at any age. Toxoplasma gondii, cryptococcus neoformans and other organisms can also cause encephalitis at any age. Steroid responsive meningoencephalitis (SRME) and granulomatous meningoencephalitis (GME) are two common inflammations of unknown cause, which can cause seizures in adult dogs.

Hypoglycemia, hypocalcemia and hepatoencephalopathy are metabolic causes of seizures. Organophosphate and lead toxicities are common. Head injuries can cause seizures immediately or later in life. Vascular disorders are rare but vasculitis, arterio-venous malformations and arteriosclerosis have been reported in dogs. An ischemic encephalopathy of the cerebral cortex can cause seizures in cats. Primary cerebral neoplasia is rare in animals under 5 years of age except in brachycephalic breeds of dogs. Lymphosarcoma can occur at any age.

Idiopathic epilepsy can be defined as a disorder of recurrent seizures with no detectable underlying disease process. Idiopathic epilepsy can be inherited or acquired, but rarely occurs prior to 6 months of age. Inherited epilepsy occurs in pure bred dogs, 1-4 years of age and has not been reported in cats. Acquired epilepsy can occur at any age, as it is due to residual brain damage incurred from a past problem such as encephalitis or head injury.

The possibility of exposure to toxins or trauma can be ascertained from the history. Evidence of trauma or chorioretinitis (associated with meningoencephalitis) may be found on the physical examination. Serum glucose, calcium and fasting and 2 hour post prandial bile acids determinations are evaluated to detect metabolic disturbances. Serum canine distemper virus, feline infectious peritonitis virus, toxoplasmosis, and cryptococcosis titers can be evaluated to diagnose these infections. Serum cholinesterase and lead determinations can be useful to diagnose these toxicities.

Meningoencephalitis usually has increased white blood cells and protein in the cerebrospinal fluid (CSF). Along with the serum titers, CSF titers for various organisms can also be evaluated. If increased leukocytes and protein are present in the CSF, but all titers for infectious organisms are negative, SRME or GME are suspected.

Hydrocephalus can be demonstrated with ultrasound, if the fontanelle of the skull is large enough. Hydrocephalus and lissencephaly can be seen on magnetic resonance imaging (MRI) scans.

Increased protein and occasionally white blood cells in the CSF and a mass lesion on the MRI or computerized axial tomograph (CT) scan are used to diagnose neoplasia.

Idiopathic epilepsy is a diagnosis made when other causes of seizures are ruled out. Mixed breed dogs are most likely to have acquired epilepsy. Pure bred dogs could have either acquired or inherited epilepsy and breeding trials are necessary to differentiate them.

MAINTENANCE SEIZURE MANAGEMENT

Oral phenobarbital (1-2 mg/kg every 12 hours) is the routine maintenance anticonvulsant choice for dogs and cats. It is inexpensive, safe and effective. If seizures are not controlled, the dosage can be increased until serum phenobarbital levels reach 30 micrograms/ml. If the serum phenobarbital level is greater than 30 micrograms/ml, hepatopathy may develop.

Potassium bromide (KBr) 22 mg/kg once daily may be added to the phenobarbital therapy for dogs with therapeutic serum phenobarbital levels and persistent or clusters of seizures. If the dog becomes sedated on the combination of phenobarbital and KBr, phenobarbital may be reduced slightly each day until sedation disappears. If phenobarbital induced liver dysfunction is present, the phenobarbital dosage can be reduced further and KBr increased to 22 mg/kg every 12 hours. Liver function usually will improve as KBr is not metabolized by the liver, but excreted through the kidneys. KBr may also be used alone at 22 mg/kg every 12 hours to control seizures. Serum KBr levels of 1,000 - 1,500 micrograms/ml are therapeutic and reach steady state in 4 months.

In the United States, pharmaceutical grade KBr is no longer available because of toxicity to humans. Chemical grade KBr crystals are mixed in water (250 mg/ml) and clients are instructed to wear gloves and put liquid on a small amount of bread or dog food with a syringe. Once the KBr is ingesyed, the evening or morning meal is then fed. KBr is very effective for management of intractable cluster seizures in dogs. KBr has not been used in cats.

Oral diazepam is an effective anticonvulsant in cats, but not dogs. If seizures in cats are difficult to control, when serum phenobarbital levels are 30 micrograms/ml, 1-2 mg oral diazepam every 12 hours can be added to the phenobarbital. If sedation occurs, reduce the phenobarbital dosage slightly each day until sedation disappears. Oral diazepam (1-2 mg) alone may also be used in cats every 8-12 hours to control seizures.

Primidone and diphenylhydantoin are not used in cats and have become unpopular in dogs because of liver damage (Primidone) and rapid metabolism (Diphenylhydantoin). No advantage has been found over phenobarbital and potassium bromide which are safer and more effective.

Clonazepam 0.06 - 0.2 mg/kg/day divided every 6-8 hours, chlorazepate 2-6 mg/kg/day divided every 8-12 hours, valproic acid 15-200 mg/kg/day divided every 6-8 hours, carbamazepine 4-10 mg/kg/day divided every 6-8 hours and paramethadione 30-50 mg/kg/day divided every 8 hours are other anticonvulsants which have been used orally with varying success in dogs to control intractable epilepsy. These anticonvulsants are expensive and often not effective.

Acupuncture has been successful in controlling seizures in some cases that were resistant to all types of drug therapy. Referral to a certified acupuncturist is recommended. Following acupuncture, anticonvulsant doses may be reduced significantly. Acupuncture may be used instead of drug therapy for routine seizure control in dogs and cats.

Organophosphates, phenothiazine derivative tranquilizers and ivermectin should be avoided in animals with a history of intractable seizures, as these can make control difficult in individual animals.

DIFFERENTIAL DIAGNOSIS OF BEHAVIORAL ABNORMALITIES

A neurological disorder should be suspected in any dog or cat presented with dementia, aggression or other change in personality. Dementia is loss of intellectual abilities which may include training, habits and recognition of familiar objects including the owner. Aggressiveness is often due to psychological or genetic personality disorders such as dominance aggression, but can be due to neurological disease. Compulsive circling and pacing are also behaviors which may have a neurological cause.

Compulsive tail chasing and self mutilation may be a neurochemical or neuro-hormonal imbalance, but the mechanism is unknown and an underlying nervous system lesion is rarely found.

Many of the same mechanisms of disease produce behavior changes as well as seizures and an animal may have both. Congenital, inflammatory, metabolic, toxic, traumatic, vascular and neoplastic disorders of the nervous system may produce behavioral abnormalities. Animals with congenital hydrocephalus may be demented, aggressive and difficult to housebreak and train due to the severe atrophy of the cerebral cortex and diencephalon.

Lissencephaly, congenital lack of neuronal and gyral development of the cerebral cortex, results in aggression, hallucinations, dementia and unpredictable behavior in dogs and cats. Lissencephaly can be diagnosed with MRI scans.

GM_2 gangliosidosis, ceroid lipofuscinosis, neuronal glycoproteinosis and fucosidosis are lysosomal storage disorders that produce progressive behavior changes under 1 year of age. Only special centers can test for specific enzyme defects in leukocytes in GM_2 gangliosidosis and fucosidosis. Abnormalities are found on histological examination of lymph nodes in ceroid lipofuscinosis and neuronal glycoproteinosis.

As discussed above, meningoencephalitis can be caused by canine distemper virus, feline infectious peritonitis virus, Toxoplasma gondii, systemic fungi, bacteria and other organisms. GME and SRME may cause behavior abnormalities that are controlled with 2-3 mg/kg/day oral prednisone. If the behavioral abnormalities subside after 1-2 weeks, the prednisone dose is reduced over the next 2 weeks and alternate day therapy is attempted. GME eventually progresses and is confirmed at necropsy, but SRME remains controlled on alternate day prednisone, which may be eventually discontinued after 6-12 months.

Hepatoencephalopathy is characterized by episodic dementia, pacing and circling. Serum ammonia and bile acids are elevated. Hypoglycemia, hyperglycemia and uremia may produce confusion and delirium and are detected with serum glucose, blood urea nitrogen and creatinine.

Lead poisoning can cause hysterical behavior. Blood lead levels can be evaluated. Tranquilizers and stimulants produce depression and hyperactivity, respectively. The possibility of exposure to drugs should be ascertained in the history.

Head injuries cause dementia and compulsive circling that can be transient or permanent. A cerebral infarct of unknown cause described in cats can produce permanent aggressive behavior.

Cerebral neoplasia can produce progressive dementia, aggression, compulsive pacing and circling. CSF may have increased protein and on occasion increased WBC. Cerebral neoplasia can be visualized on a CT or MRI scans.

VESTIBULAR AND CEREBELLAR DISORDERS OF DOGS AND CATS

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Vestibular disorders produce disturbances of balance or equilibrium. In unilateral or asymmetrical vestbular disease, the dysequilibrium is manifested as a head tilt to one side and the animal may also lean, circle, fall, or roll to that side. Bilateral symmetrical vestibular disease is more difficult to recognize as there is no head tilt, and the dysequilibrium is associated with wide swinging head movements and the animal may weave and stagger to either side. In either unilateral or bilateral vestibular dysfunction, there may be spontaneous or positional nystagmus, rhythmic eyeball movements with a fast and slow phase. The nystagmus may be horizontal, rotary (in an arc) or vertical.

Disease of the vestibular system may be localized to 3 sites : 1. The peripheral vestibular nerve beginning with receptors in the inner ear and extending through the petrous temporal bone of the skull to the brain stem. 2. The vestibular nuclei of the brainstem in the floor of the fourth venticle of the rostral medulla oblongata. 3. The fastigial nucleus and flocculonodular lobes of the cerebellum.

Many diseases affect a region rather than a specific nerve or nucleus, so often other signs which accompany vestibular dysfunction can be used to localize a lesion. A lesion must be localized to a particular section of the vestibular system before an appropriate differential diagnosis can be established.

Lesions of the cerebellum effect muscle tone and coordination. Generalized incoordination of the head and limb movements unasssociated with weakness is the hallmark of cerebellar disease. The limbs may be hypermetric (excessively flexed) or hypometric (excessively extended) during ambulation. The head may bob and tremor as the animal attempts to eat, drink, and sniff the ground. Acute rostral cerebellar lesions, as with trauma, result in opisthotonus with the animal in lateral recumbancy, conscious, and neck and thoracic limbs hyperextended.

VESTIBULAR NERVE DISORDERS

The diseases of the vestibular system which are most frequently encountered and have the best prognosis involve the vestibular receptors and nerves in the inner ear. With unilateral disease, the head tilt is toward the lesion and if a nystagmus is present, it is horizontal or rotary and the fast phase is away from the lesion. Although there is a reduction of tone in the extensor muscles of the limbs on the same side as the lesion, which makes the animal lean or circle toward the effected side, there is no hemiparesis, conscious proprioceptive deficit (animal stands on knuckled toes) or incoordination noted when the animal ambulates.

With bilateral vestibular nerve disease the animal may stagger from side to side and have head movements with wide excursions which form a horizontal figure 8. Normal vestibular nystagmus, evidenced by a few beats of nystagmus when the head is turned from side to side, may be absent with bilateral vestibular disease.

With middle and inner ear disease, the facial nerve may also be affected and produce signs of increased or reduced lacrimation and /or salivation as well as weakness of eyelid, ear and lip movement on the same side. The sympathetic innervation to the eye may be interrupted, as it courses through the middle ear, and ptosis, miosis, and enophthalmus (Horner's Syndrome) of the eye on the same side may be seen.

In cases of bilateral inner ear disease, affected animals may have reduced or complete loss of hearing. The cochlear nerve and the vestibular nerve travel

in intimate contact and form the vestibulocochlear nerve. In most disorders of the vestibular nerve, the cochlear nerve is affected as well. During the routine neurological examination, subtle reductions of hearing often go undetected. Brainstem auditory evoked response (BAER) testing is a method of evaluating cochlear receptors and nerve function and can be useful to detect subtle hearing impairments.

On rare occasions vestibular nerve disease may be part of a cranial polyneuropathy or diffuse polyneurpathy and other cranial nerve deficits such as blindness , mydriasis, ventrolateral strabismus, dysphagia, megaesophagus, and tongue weakness or atrophy as well as weakness with depressed spinal reflexes may be seen.

A thorough history is obtained and questions concerning the onset and progression of signs, possibility of infection, injury, or toxicity, and previous medications are explored. A complete physical examination is performed, but examination of the external ear canal and pharynx should be performed under general anesthesia, if nothing is found on the initial examination.

A complete blood count(CBC) and chemistry profile is often obtained prior to anesthesia. A normal CBC does not rule out an inner ear infection. Skull radiographs and magnetic resonance imaging (MRI) scans can be useful to visualize middle and inner ear structures. A BAER test will be abnormal if the auditory portion of the vestibulocochlear nerve is involved. Once this data is collected the best diagnosis can be selected from the list below and treated accordingly.

DIFFERENTIAL DIAGNOSIS, TREATMENT, AND PROGNOSIS OF VESTIBULAR NERVE DISORDERS

1. **Idiopathic Congenital**- onset at birth or up to 12 weeks of age (Doberman Pinscher, German Shepherd, Beagle, and Smooth Haired Fox Terrier dogs and Siamese, Burmese, and Tonkanese cats); Signs may be unilateral or bilateral; Hearing may be impaired or absent; No facial or other cranial nerve abnormalities noted; No treatment; Signs associated with unilateral involvement often improve and animals compensate, but may never be normal; Signs associated with bilateral involvement may be permanent;

2. **Inner Ear Infection**- any age, any breed; Onset can be acute or chronic progressive; Signs may be unilateral or bilateral; There may be only vestibular signs; There may be facial nerve paralysis and/or Horner's syndrome; There may be impaired hearing; If the animal is treated early with an appropriate antibiotic (oral trimethoprim sulfa and cephalosporin, if no culture obtained) for 4-6 weeks, the prognosis is often very good. DO NOT DISCONTINUE THERAPY BEFORE 4 WEEKS EVEN IF SIGNS RESOLVE! DO NOT USE GLUCOCORTICOIDS! A head tilt and facial nerve paralysis (may need artificial tears) can be permanent. An inner ear infection is the most common cause of vestibular disease.

3. **Idiopathic Vestibular Syndrome**- common in adult cats and geriatric dogs; ocasionally seen in adult non geriatric dogs; Peracute onset of severe vestibular dysfunction and affected animals may be unable to ambulate due to disorientation from the severe dysequilibrium. Unilateral signs are most common; Ocasionally bilateral signs are seen; No facial nerve paralysis or Horner's Syndrome or other cranial neurpathies are present; Impaired hearing may occur in bilateral disease; Animals improve over 24-72 hours and can ambulate, although still have dysequilibrium. RULE OUT INNER EAR INFECTION! No treatment; Most animals improve over 1-3 weeks and may return to normal or retain a permanent head tilt.

4. **Trauma** -common cause of acute vestibular signs in any age or breed; Signs are most often unilateral; May find blood in external ear canal; May have facial nerve paralysis and Horner's syndrome. May give glucocorticoids if known

trauma. Signs may resolve or there can be a permanent head tilt.

5. **Neoplasia** -nasopharyngeal polyps may grow in the middle ear and produce compression or infection of inner ear, but neoplasia is rare. Neurofibroma of the vestibular nerve will produce a chronic progressive head tilt; Exploratory bulla osteotomy and attempt tumor removal; Prognosis good if a polyp removed, but poor if anything else.

6. **Hypothyroidism** -rare cause of unilateral or bilateral vestibular disease in dogs; May or not have associated facial nerve paraysis or megaesophagus; May or not have diffuse neuromuscular disease which must be differentiated from brainstem disease (see below); Do TSH stimulation on all vestibular nerve disease of unknown origin in adult dogs; Treat with thyroid replacement therapy and signs resolve.

7. **Aminoglycoside Antibiotics** -If animals are treated with gentamicin or amikacin, they should be closely monitored for dysequillibrium and hearing impairment. Historically streptomycin has caused dysequilibrium in cats and should be avoided.

8. **Cranial Polyneuropathy** -rare cause of multiple cranial nerve signs in dogs with no evidence of brainstem disease and normal thyroid function; Are glucocorticoid responsive and are thought to be an autoimmune disorder.

BRAINSTEM VESTIBULAR, VESTIBULOCEREBELLAR, AND CEREBELLAR DISORDERS

Diseases of the vestibular system in the brainstem and cerebellum are less common than vestibular nerve disease and often have a poor prognosis. If the brainstem vestibular nuclei and pathways are affected, the animal will have a head tilt toward the lesion and deficits similar to vestibular nerve disease. Nystagmus may be horizontal, rotary or vertical.

With involvement of brainstem motor pathways, hemiparesis or quadriparesis worse on the side of the lesion may be apparent. Spinal reflexes in the thoracic and pelvic limbs will be normal or hyperactive, which will differentiate the brainstem disease from a neuromuscular disorder such as hypothyroidism or some other polyneuropathy. In the latter diseases, spinal reflexes are depressed or absent.

Conscious proprioceptive deficits, evidenced by standing on the dorsum of the toes, when placed in this abnormal position, may be present due to involvement of ascending sensory pathways in the brainstem. Ataxia and hypermetria especially of the thoracic limb on the side of the lesion is commonly seen if there a is spinocerebellar tract or caudal cerebellar peduncle brainstem lesion.

If a unilateral lesion is located in the vestibular portion of the cerebellum, the head tilt is away from the side of the lesion (paradoxical head tilt). There is usually ataxia and hypermetria, especially evident in the thoracic limb on the side of the lesion; but if there is no brainstem disease then there is no hemiparesis or conscious proprioceptive deficit. Nystagmus may be horizontal, rotary, or vertical. There may be other cerebellar signs like head bobbing and tremors as well.

A diffuse or bilateral lesion of the cerebellum may produce generalized incoordination and dysequillibrium. Nystagmus may be present and is often vertical.

Many cerebellar disorders only produce incoordination of movement and head tremors with no evidence of dysequilibrium.

The patient evaluation is similar to that described for vestibular nerve disorders with the addition of cerebrospinal fluid (CSF) analysis and titers for various infectious organisms. The MRI and BAER are performed with the focus on the rostral medullary brainstem and cerebellar areas. A lesion extending from the

inner ear to the brainstem can be visualized with the MRI scan.

DIFFERENTIAL DIAGNOSIS, TREATMENT, AND PROGNOSIS OF BRAINSTEM VESTIBULAR AND VESTIBULOCERBELLAR DISORDERS

1. **Infections and Inflammations**

a. **Ascending Inner Ear Infection** -occasional in cats, but rare in dogs; Usually have a history of chronic resistant inner ear infections. CSF- degenerative neutrophilic pleocytosis, may or may not get positive CSF culture ; Treat aggressively with appropriate antibiotics as described above, but prognosis may be poor. Often avoidable if animal treated early and long enough, then monitored for recurring infections.

b. **Septicemia and Bacterial Embolization** -producing a suppurative meningoencephalitis; acute onset; animal febrile; CBC- neutrophilic leukocytosis;

CSF- degenerative neutrophilic pleocytosis, may or may not get positive CSF culture; Treat aggressively with appropriate antibiotics; Discover and treat source of infection ie. bacterial endocarditis or pyometra; Prognosis may be poor if signs severe

c. **Steroid Responsive Meningoencephalomyelitis** - any age or breed of dog; Acute onset of signs; CSF-pleocytosis (cell type varies but no degenerating neutrophils); Response to glucocorticoids (Prednisone 2.2mg/kg every 12 hours) in 72 hours; Long term prognosis can be good, but may have to be on steroids indefinately (taper dose of prednisone to lowest effective level that can be given every other day).

d. **Granulomatous Meningoencephalomyelitis** - adult dog; acute onset of signs; CSF- pleocytosis (cell type varies but mixed cells most common); May have temporary response to glucocorticoids; Long term prognosis poor; Necropsy confirms diagnosis.

e. **Distemper virus (dogs) and Feline Infectious Peritonitis virus (cats) Meningoencephalomyelitis** -may produce acute progressive signs; CSF pleocytosis (cell type varies); Serum and CSF viral titers may support the diagnosis. Treat with prednisone to reduce inflammation and antibiotics for secondary bacterial infection,but prognosis is poor. Necropsy may be needed to confirm the diagnosis.

f. **Other Infections** -include toxoplasmosis, cryptococcosis, aspergillosis and other rare organisms; Acute progressive signs; CSF pleocytosis; Serum and CSF titers for specific organisms may help confirm the diagnosis so apropriate therapy can be given; Prognosis grave.

2. **Neoplasia** -adult or geriatric dogs and cats; acute or chronic progressive signs; CSF- may or not have pleocytosis, often only elevated protein; MRI usually shows tumor; Prognosis grave; Necropsy confirms diagnosis and tumor type.

3. **Metronidazole Toxicity** -animal on metronidazole therapy may suddenly develop ataxia and nystagmus (vertical); Can go on to develop severe depression and seizures; Discontinue metronidazole therapy and control seizures; Signs often resolve if detected early.

4. **Thiamine Deficiency** -Kittens or adult cats on a deficient diet may develop signs of bilateral vestibular and cerebellar disease; Treat with 50 mg Thiamine every 12 hours until signs improve and correct the diet; Prognosis good if detected early; Rare in dogs unless on an unusual diet.

5.**Trauma** -acute onset of signs; Have history or physical evidence of trauma; treat with glucocorticoids; Prognosis varies with severity of the lesion; Can continue to slowly improve over 9-12 months.

6. **Neuroaxonal Dystrophy** -Rottweiler and Collie dogs and some cats may

develop chronic progressive incoordination, bilateral dysequillibrium, and nystagmus, which may begin at 1-2 years of age and insideously progress over the following years; No treatment; Suspected to be inherited; Animals may be acceptable pets for years, but need assitance to drink, eat, and walk later in the course of the disease.

DIFFERENTIAL DIAGNOSIS OF CEREBELLAR DISORDERS WITHOUT VESTIBULAR DYSFUNCTION

1. **Congenital Malformations** -signs of cerebellar dysfunction noticed shortly after birth; No treatment; Signs stay the same; Animals may be accetable pets if mildly affected; In cats, often due to an in-utero panleukopoenia virus infection;

2. **Cerebellar Abiotrophy, Spongiform Degeneration, Lysosomal Storage Disorders and other Cerebellar Degenerations** -Kerry Blue Terriers, Gordon Setters and other dogs may develop progressive cerebellar signs from an inherited cerebellar abiotrophy, when less than 1 year of age; No treatment; Kerry Blue Terriers are severely affected by 1 year of age, but Gordon Setters may progress more slowly and be acceptable pets for a longer period of time. Labrador Retrievers, 4-6 months of age, may develop progressive ataxia with episodes of opisthotonus from a spongiform degeneration of the cerebellum; No treatment; Poor prognosis. Several lysosomal storage disorders produce early signs of cerebellar dysfunction prior to the first year of life; No treatment; Prognosis grave. Adult dogs have been described to have progressive cerebellar signs from late onset cerebellar degeneration of unknown cause; No treatment; Signs slowly progress over time.

3. **Infections and inflammations** -infections and inflammations such as steroid responsive meningoencephalitis, granulomatous meningoencephalitis, distemper and feline infectious peritonitis viruses, and other organisms producing vestibulocerebellar disease discussed above can also only affect the cerebellum to produce generalized incoordination; The diagnosis, treatment, and prognosis are the same.

4. **Trauma** -Acute cerebellar injury may result in opisthotonus which often resolves over 4-8 weeks, but residual cerebellar incoordination may be seen.

5. **Neoplasia** -Neoplasia described with vestibulocerebellar disorders can also affect the cerebellum to produce only generalized incoordination. The diagnosis, treatment and prognosis is the same.

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EXAMEN NEUROLOGIQUE EN PRATIQUE CANINE

1. Introduction

Le système nerveux est divisé en deux parties, l'une centrale comprenant le cerveau et la moëlle épinière et l'autre, périphérique, constituée des nerfs émergeant du tronc cérébral et de la moëlle épinière. Nous survolerons en premier lieu l'anatomie de ces structures et reverrons les symptômes observés lorsque leur fonction est perturbée. Au terme de l'exposé suivant qui aura traité de l'examen neurologique, chacun devrait disposer des bases suffisantes permettant de localiser la lésion au sein du système nerveux.

2. Anatomie du système nerveux

Le cerveau est constitué anatomo-fonctionnellement de cinq parties distinctes. Il s'agit du tronc cérébral, du cervelet, du métencéphale, du cortex, du système vestibulaire. Il est important de réaliser que les symptômes neurologiques permettent de distinguer :

1) les affections du tronc cérébral (bulbe rachidien ou moëlle allongée, protubérance cérébrale, mésencéphale) d'où proviennent la plupart des nerfs crâniens,
2) les maladies du cervelet,
3) les lésions du métencéphale comprenant le thalamus et l'hypothalamus
4) les affections du cortex et des ganglions basaux
5) les troubles de l'appareil vestibulaire.

Une localisation plus précise n'est guère possible. Dans de nombreux cas neurologiques, plusieurs parties du cerveau sont atteintes. Il s'agit alors de lésions multifocales ou diffuses qui sont identifiées sur la base de signes chacun représentatif de l'atteinte d'une partie du cerveau.

2.1. Les nerfs crâniens

Les nerfs crâniens partent du tronc cérébral. Ils trouvent leur origine dans les neurones de leurs noyaux répartis entre le mésencéphale et le bulbe rachidien. Les fonctions anormales de ces nerfs s'expriment par :
- un strabisme (nerfs moteurs III, IV, VI)

- des réflexes pupillaires absents (nerf III parasympathique)
- une hypoalgésie ou une analgésie de la face (nerf V sensible)
- une paralysie de la mâchoire, syndrome de la "gueule ouverte" (nerf V moteur)
- une paralysie des muscles de la face, paralysie faciale (nerf VII moteur)
- un syndrome vestibulaire avec tête penchée, ataxie, tendance à tomber et nystagmus (nerf VIII vestibulaire)
- la surdité (nerf VIII auditif)
- des difficultés de déglutition (nerfs moteurs IX et X)
- des arythmies cardiaques et une paralysie du tractus digestif (nerf X parasympatique)
- une paralysie des cordes vocales (nerfs moteurs X,XI)
- une paralysie de la langue (nerf XII).

Le nerf accessoire (nerf XI) innerve également les muscles trapèze, brachiocéphalique et une partie du sternocéphalique. Sa lésion entraîne une atrophie de ces muscles.

Nous réalisons ainsi que deux nerfs (I,II) sont absents de cette liste. Il s'agit du nerf olfactif qui est davantage une extension du cerveau qu'un nerf proprement dit et du nerf optique (II).

Une activité anormale de ces deux nerfs se résume par :
- une anosmie (nerf I) (difficile à déterminer en médecine vétérinaire),
- une cécité périphérique (rétine, nerf II) ou centrale selon l'explication ci-dessous.

Une cécité périphérique est due à une lésion de la rétine, du nerf optique, du chiasma optique ou de la bandelette optique. Une cécité centrale est due à l'atteinte des voies visuelles situées entre la bandelette optique et le cortex occipital. La distinction entre ces deux types de cécité est basée sur la qualité des réflexes pupillaires. Lorsque les réflexes pupillaires sont absents, il s'agit d'une cécité périphérique. La présence de réflexes pupillaires est le signe d'une cécité centrale.

2.2. <u>Substance blanche de la base du cerveau</u>

La base du cerveau contient les voies afférentes et efférentes qui assurent respectivement la transmission des sensations provenant de la périphérie et celle responsable de l'activité motrice descendante. La distinction entre une atteinte des nerfs crâniens dans leur périphérie par opposition à celle de la base du cerveau est obtenue grâce aux test de maintien et de posture. Ces tests exigent l'intégrité des voies afférentes et efférentes qui traversent le tronc cérébral.

2.3 La formation réticulée

Les affections de la base du cerveau (tronc cérébral) peuvent être à l'origine d'apathie, de stupeur ou de coma. Ces signes sont généralement dus à l'atteinte de cette structure.

2.4. Résumé des affections du tronc cérébral

Une fonction anormale des nerfs crâniens associée à des tests de maintien et de posture déficients ainsi qu'à un état d'apathie sont le signe d'une maladie du tronc cérébral.

2.5 Le cervelet

Le cervelet est un organe régulateur de l'activité motrice logé dans la fosse postérieure de la boîte crânienne. Il est séparé des lobes occipitaux du cerveau par la tente du cervelet. Sa fonction majeure est de doser l'activité musculaire et de coordonner les fins mouvements du corps. Le cervelet fait également partie du système vestibulaire. Certaines lésions de la partie latéro-ventrale de cet organe peuvent être à l'origine d'un syndrome vestibulaire.

Les signes anormaux d'une affection du cervelet sont les suivants :
- posture anormale avec pattes écartées, trémor (tremblements) grossier du corps et tendance à tomber,
- ataxie généralisée sans faiblesse des pattes (ataxie tronculaire)
- mouvements trop amples (hypermétrie) en particulier des membres antérieurs
- chutes fréquentes
- les animaux se heurtent aux obstacles car ils ne régulent pas leurs mouvements
- tremblements fins de la tête au repos
- tremblements grossiers de la tête lors de mouvements volontaires (tremblements d'intention)
- spasticité des membres
- réflexe de menace absent en présence d'une vision maintenue
- syndrome vestibulaire (tête penchée, nystagmus).

La présence de lésions dans le voisinage de la base du cervelet peut, à l'occasion, provoquer des signes cérébelleux de par les lésions engendrées aux voies nerveuses afférentes et efférentes du cervelet.

2.6 Le métencéphale (thalamus - hypothalamus)

Le thalamus représente un amoncellement de noyaux dans la profondeur des hémisphères. Le thalamus est situé crânialement au mésencéphale. Cette partie du cerveau reçoit des fibres sensorielles d'origines diverses et est en

étroite relation avec les centres moteurs de la partie antérieure de la base du cerveau. Il projète les impulsions électriques principalement vers le cortex qui lui renvoie d'autres informations.

L'hypothalamus est situé en dessous du thalamus. Il est responsable des réactions viscérales tels les sentiments de faim et de soif, la régulation de la température corporelle et le degré d'excrétion de l'eau par les reins. Il produit également des facteurs réglant certaines fonctions endocrines. Certaines de ses neurones appartiennent au système végétatif (sympathique et parasympathique). Bien que l'hypothalamus ne soit pas contrôlé directement par le cortex, ce dernier influence son activité, particulièrement lors d'états émotionnels.

Les maladies de cette partie du cerveau provoquent :

- une polyurie
- une polydispie (diabète insipide)
- un appétit anormal (anorexie, polyphagie)
- un dérèglement de la température corporelle
- de l'apathie, de la stupeur ou un coma.

2.7 Le cortex, les ganglions basaux

Le cortex cérébral constitue la surface des hémisphères cérébraux.. Il est constitué d'un nombre gigantesque de cellules (substance grise) ayant entre elles des voies de communications si complexes et nombreuses qu'elles restent pour la plupart non-identifiées. Nous distinguons au plan fonctionnel des régions motrices et sensibles alors que d'autres sont responsables de phénomènes d'association indispensables à la coordination des phénomènes cérébraux. Les hémisphères cérébraux sont séparés de façon plus ou moins arbitraires en lobes frontaux pariétaux, temporaux et occipitaux. Les fibres quittant les neurones du cortex cérébral et celles responsables du transport des informations provenant de régions sous-corticales constituent la substance blanche du cerveau.

Les ganglions basaux sont constitués de cellules logées dans la profondeur des hémisphères. Ils bordent les ventricules latéraux. Ces ganglions sont en étroite relation l'un avec l'autre ainsi qu'avec le cortex cérébral et le thalamus par l'intermédiaire de fibres de connection.

Les lésions affectant le cortex, les ganglions basaux se résument par :

- des parésies et ataxies discrètes souvent plus marquées d'un côté du corps que l'autre
- des démarches coordonnées non contrôlées en cercle, le long d'un mur ou forcées et maintenues avec la tête poussant contre un mur
- une cécité centrale unilatérale ou bilatérale (réflexes pupillaires maintenus)

- des myoclonies, des tremblements, des crampes, des crises d'épilepsie, de narcolepsie ou de cataplexie
- des tests de maintien et de posture déficients (souvent d'un seul côté du corps).

2.6. L'appareil vestibulaire

L'appareil vestibulaire est responsable de l'équilibre spacial du corps et de la tête par rapport au sol. Il est constitué de diverses structures réparties dans le cervelet, le tronc cérébral et l'oreille interne. C'est la coordination et le bon fonctionnement de ces structures qui font que l'animal maintient l'équilibre de sa tête et de son corps.

Lors d'une atteinte vestibulaire à l'inverse, on observe des troubles de l'équilibre dont les caractéristiques les plus classiques et plus fréquentes en médecine vétérinaire sont la présence d'une tête penchée, de nystagmus, de marche en cercle, de chute ou faiblesse sur le côté affecté, et d'un strabisme ventral du côté affecté.

3. L'examen neurologique

L'examen neurologique ne doit être fait qu'au terme de l'examen général.

Le premier but recherché lors de l'examen neurologique est la localisaton de l'affection. Lorsque la lésion est localisée, les examens complémentaires tels l'examen du liquide céphalorachien (LCR), les études électrodiagnostiques et les radiographies permettent généralement de préciser ou confirmer la localisation de la lésion et de définir la nature du mal.

3.1. Espèce, race, âge, sexe

Ces informations sont primordiales tant il est vrai que certaines maladies se développent chez des sujets particuliers souvent d'âge assez bien défini.

3.2. Anamnèse

Les informaions obtenues lors de l'anamnèse sont également très importantes. On insistera sur les points suivants :

- l'évolution de la maladie (aiguë, chronique, récidivante, progressive)
- plusieurs animaux d'un élevage ou d'une famille, par exemple, sont-ils atteints du même mal ?
- quelle nourriture donne-t-on ?
- l'origine de l'animal (étranger, indigène, chenil, etc.)
- l'animal a-t-il souffert d'autres maladies ?

- les vaccins (à jour, pas à jour ?)

3.3. La locomotion

Les anomalies de la démarche peuvent être généralisées aux quatre pattes ou être limitées à une certaine partie du corps. On observera le patient pour les signes suivants :

- monoplégie, hémiplégie, paraplégie, tétraplégie
- raideur de certains membres
- mouvements anormaux (en cercle, tendance à tomber, ataxie, mouvements de manège, etc.)
- dysmétrie (généralement hypermétrie)
- raideur du cou.

3.4. Tests de maintien et de posture

Les impulsions sensitives indicatrices du degré de tension de la musculature, des tendons et des capsules articulaires proviennent de récepteurs spécifiques qui traduisent ces phénomènes mécaniques en impulsions électriques. Les potentiels électriques engendrés parcourent les nefs périphériques, la moëlle épinière et accèdent au cerveau où ils sont perçus consciemment. Une réponse motrice est alors engendrée par le cerveau. Elle assure chez les sujets sains des mouvements coordonnés et une posture adéquate.

Les tests de maintien et réflexes posturaux les plus utiles en médecine vétérinaire sont le "placé tactile" et "les sautillements de côté". D'autres, telle que la demi-marche, la marche en brouette, le placé visuel, peuvent aussi être utilisés.

Les anomalies de ces tests permettent de mieux localiser l'affection, par exemple à une ou plusieurs pattes ou d'un côté ou de l'autre du corps. Des tests de maintien et de posture anormaux suggèrent une atteinte d'une partie quelconque du système nerveux, soit :

- des nefs périphériques,
- de la moëlle épinière,
- de la base du cerveau ou
- du cortex

3.5. Les réflexes spinaux ou réflexes médullaires

Les réflexes médullaires dépendent de l'intégrité des muscles, des nefs périphériques et de la substance grise de la moëlle épinière au niveau de

l'intumescence cervicale (pour les réflexes des membres postérieurs) et de l'intumescence cervicale (pour les réflexes des membres antérieurs). La qualité des réflexes médullaires dépend aussi d'un effet inhibiteur du cerveau sur les neurones moteurs au sein de ces intumescences. Lors d'hyperréflexie (réflexes trop forts), cet effet inhibiteur est réduit ou éliminé par une lésion de la substance blanche située entre le cerveau et l'arc réflexe qui prend place au sein de ces instrumescences. Une hyporéflexie (réflexes trop faibles) ou une aréflexie (réflexes absents) sont le signe d'une lésion des nefs périphériques, des muscles ou de la substance grise de la moëlle épinière au niveau de ces intumescences. Ainsi une lésion située entre la base du cerveau et le cinquième segment cervical de la moëlle épinière provoque, même s'il s'agit d'une transsection complète de la moëlle épinière, une hyperréflexie des quatre pattes. Une lésion de l'intumescence cervicale (segments C6-T1) provoque une hyporéflexie ou une aréflexie des pattes antérieures et une hyperréflexie des pattes postérieures. Une lésion des segments T2 à L3 ne modifie pas la qualité des réflexes aux pattes antérieures, mais engendre une hyperréflexie des pattes arrières. Lorsque l'intumescence lombosacrée est lésée, nous assistons à une hyporéflexie ou une aréflexie des membres postérieurs. Lors d'atteinte des segments sacrés (S1-S3), nous observons une hyporéflexie ou une aréflexie du réflexe périnéal et du réflexe bulbo-urétral ou vulvo-urétral. Une atonie de la vessie et une incontinence urinaire due à une incompétence des mécanismes sphinctériens peuvent apparaître.

Le réflexe panniculaire est utile pour localiser les lésions de la moëlle épinière thoraco-lombaire chez le chien et le chat. Les fibres afférentes sensibles aux pincements de la peau de part et d'autre du rachis de l'animal entrent dans la moëlle épinière au niveau de celui de la stimulation. Ces fibres parcourent ensuite la moëlle épinière jusque dans les segments T1 à C8 où l'arc réflexe a lieu. Les voies post-synaptiques quittent la moëlle épinière à l'endroit de ces segments et viennent innerver le dermatome stimulé. Une hyporéflexie ou une aréflexie est généralement observée entre la lésion et la partie caudale du rachis. Parfois, une hyperreflexie est observée là où la lésion est localisée.

Les réflexes médullaires sont soit multisynaptiques et conditionnent plusieurs nerfs, tel que le réflexe de retrait du membre, ou monosynaptique, n'interessant qu'un seul nerf, tel que le réflexe du biceps brachial (membre antérieur) ou le réflexe rotulien (membre postérieur).

3.6. La sensation de douleur

L'olfaction (nerf I) est évaluée en présentant de la nourriture à l'animal (après avoir couvert ses yeux).

La vision (nerf II) est testée par le test clignement à la menace dans les différents champs de vision du patient. Il faut prendre garde à ne provoquer aucun mouvement d'air qui puisse faire fermer les paupières de l'animal.

Un strabisme (nerfs III, IV, VI) sera observé directement ou en déplaçant la tête vers le haut.

La sensation douloureuse de la face (nerf V) est évaluée en pinçant les différentes parties de la face (branche maxillaire, branche ophtalmique et branche mandibulaire).

Une paralysie des muscles de la mâchoire (nerf V) est diagnostiquée par simple observation ou lors de la manipulation de la gueule qui s'ouvre sans résistance ou reste ouverte sans se refermer ("syndrome de la mâchoire ou gueule pendante").

Une assymétrie ou une paralysie faciale (nerf VII) est observée directement : incapacité à fermer un oeil, oreille ou lèvre tombante.

Les maladies du huitième nerf (nerf acoustique) s'expriment par un syndrome vestibulaire pour la branche vestibulaire (tête penchée, nystagmus, strabisme de position , tendance à tomber, marche en cercle) ou une surdité pour la branche auditive.

Une difficulté à déglutir (nerfs IX, X) sera généralement mentionnée par le propriétaire et nous pouvons l'évaluer par administration d'eau dans la gueule ou en observant l'animal lorsqu'il se nourrit.

Une paralysie des cordes vocales (nerf X) est exprimée par une voix anormale et souvent par une dyspnée provenant des voies respiratoires supérieures et parfois par des phénomèmes de "fausses routes". L'inspection du larynx est généralement nécessaire à l'identification de cette anomalie.

Une paralysie unilatérale de la langue (nerf XII) est illustrée par une déviation de la langue vers le côté sain. Une paralysie bilatérale engendre l'incapacité à mouvoir la langue.

4. Localisation des affections nerveuses

En se basant sur tous les résultats de l'examen neurologique, nous parviendrons à localiser la lésion du système nerveux.

4.1. Anomalies de locomotion

Elles peuvent affecter une seule patte ou plusieurs membres. Elles sont parfois localisées à une moitié du corps.

4.1.1. Monoplégie

- Toujours avec hyporéflexie ou aréflexie.
- Signe de lésion d'un seul ou de plusieurs nerfs périphériques de la patte atteinte.
- Paralysie du nerf sciatique généralement due chez les petits animaux à une injection intramusculaire ou à une fracture du bassin.
- Peut-être due à une tumeur de ou plusieurs nerfs ou racines nerveuses.

4.1.2. Paraplégie

- Réflexes médullaires normaux ou exagérés : lésion de la moëlle épinière entre les segments T2 à L3.
- Réflexes médullaires abaissés ou absents : lésion de la moëlle épinière au niveau de l'intumescence lombosacrée (segments L4 à S3).

4.1.3. Hémiplégie

- Réflexes médullaires maintenus aux deux pattes lésées : lésion de la moëlle épinière cervicale (segments C1 à C5) du côté lésé.
- Réflexes médullaires diminués au membre antérieur lésé et présents ou exagérés à l'autre membre paralysé : lésion de l'intumescence cervicale (segments C6 à T1) affectant la moitié de la moelle épinière située du même côté que celui des membres paralysés.

4.1.4. Tétraplégie

- Réflexes médullaires présents à exagérés aux quatre pattes avec fonction normale des nerfs crâniens, état de conscience et comportement normaux : lésion de la moëlle épinière cervicale (segments C1 à C5).
- Réflexes médullaires diminués ou absents aux pattes antérieurs et normaux à exagérés aux pattes arrières : lésion bilatérale de l'intumescence cervicale (segments C6 à T1).
- Réflexes médullaires présents à exagérés aux quatre pattes, anomalie des nerfs crâniens, apathie, stupeur ou coma : lésion de la base du cerveau.
- Hyporéflexie ou aréflexie des quatre membres et sensation de douleur maintenue : lésions des racines nerveuses motrices.
- Hyporéflexie ou aréflexie des quatre membres et sensation de douleur diminuée : polyneuropathie périphérique.

4.1.5. Anomalie de nerfs crâniens (parmi nerfs III à XII), tests de maintien et réflexes posturaux anormaux

- Lésion du tronc cérébral,
- Les noyaux des nerfs crâniens anormaux sont atteints.
- Les voies ascendantes et descendantes responsables des tests de maintien et des réflexes posturaux qui traversent la base du cerveau sont lésées.

4.1.6. Anomalie de nerfs crâniens (parmi nerfs III à XII), tests de maintien et des réflexes posturaux normaux

- Lésion des nerfs crâniens dans leur périphérie.
- La base du cerveau est intacte (réflexes de maintien et des réflexes posturaux).

4.1.7. Cécité, réflexes pupillaires absents (cécité périphérique)

- Lésion de la rétine, du nerf optique, du chiasma optique ou des fibres prétectales qui quittent la bandelette optique et se rendent dans le mésencéphale pour provoquer le réflexe pupillaire.
- Probablement pas une lésion du mésencéphale à l'endroit de l'arc réflexe pupillaire car pas d'affection d'autres nerfs crâniens (en particulier pas de strabisme dû à une lésion du nerf III).

4.1.8. Cécité, réflexes pupillaires normaux (cécité centrale).

- Lésion du cortex optique de la région occipitale.
- Lorsque la lésion est unilatérale, l'affection se situe sur le cortex occipital opposé à celui de l'oeil aveugle.

4.1.9. Tête penchée d'un côté, paralysie faciale ainsi que miosis, ptose et enophthalmus du côté ou la tête penche, nystagmus horizontal et tests de maintien et de posture normaux

- Syndrome vestibulaire du côté où la tête penche.
- La lésion du facial ainsi que le syndrome de Horner (miosis, ptose, enophthalmus) indiquent une atteinte de l'oreille moyenne.
- Les tests de maintien et les réflexes de posture normaux en présence du syndrome vestibulaire indiquent une lésion périphérique localisée à l'oreille interne alors que le tronc cérébral n'est pas touché.
- Vraisemblablement, une otite moyenne ayant dégénéré en otite interne.

4.1.10. Tête penchée, nystagmus, tests de maintien et de postures déficients

- Syndrome vestibulaire
- Affection du tronc cérébral à l'endroit des noyaux vestibulaires (près du IVème ventricule).
- Nécessité d'examiner l'oreille interne (radiographies) et le liquide céphalo-rachidien afin d'évaluer la possibilité qu'une maladie de l'oreille interne se soit propagée à la base du cerveau.

4.1.11 Ataxie généralisée, tendance à tomber, tremor (tremblements) généralisé, et tremblement d'intention, hypermétrie.

-Symptômes représentatifs d'une maladie du cervelet.

4.1.12. Mouvements de manège en cercle vers la droite, proprioception ralentie aux membres gauches, test de sensation douloureuse à la narine gauche absente

- Les mouvements de manège en cercles sont souvent associés à une lésion corticale du côté où l'animal tourne en rond.
- Une proprioception anormale d'un côté du corps suggère une lésion du cortex contra-latéral lorsque les nerfs périphériques (réflexes normaux) et le tronc cérébral (nerfs crâniens III à XII normaux) sont sains.
- Vraisemblablement, une lésion du cortex frontal ou pariétal à droite.

4.1.13. Tétraplégie, opistotonus, pattes en extension, stupeur, nystagmus, déglutition anormale

- Les voies descendantes et ascendantes responsables de la locomotion sont lésées au cours de leur passage au sein du tronc cérébral engendrant la tétraplégie.
- L'opistotonus est observé lors de lésions graves de la base du cerveau; on parle de rigidité d'extension illustrée aussi par l'hyperextension des pattes.
- Une atteinte de la formation réticulée provoque des états d'apathie, de stupeur ou un coma.
- Le nystagmus est provoqué par des lésions du système vestibulaire périphérique ou central.
- Une déglutition anormale est due à l'atteinte des nerfs crâniens IX et X.
- Il s'agit donc d'une maladie grave de la base du cerveau.

4.1.14. Epilepsie, proprioception ralentie d'un côté du corps

- Lésion du cortex (l'épilepsie est due à une activité électrique anormale du cortex cérébral ou du thalamus).
- Vraisemblablement une lésion du cortex du côté opposé à celui où la proprioception est abaissée.

EXAMEN NEUROLOGIQUE

Propriétaire — Numéro clinique

Signalement — Date

Anamnèse

Durée des symptômes

Début de la maladie	aigu	lent
Evolution	progressive	statique
	rémissions	amélioration

Traitements

Appétit

Soif

Selles

Urine

Maladies antécédentes

Vaccinations

Antécédents familiaux

Etat psychique ou mental

Locomotion

Tests de maintien et de posture

Marche en brouette

Station sur 2 pattes ipsilatérales

Sautillement sur 2 pattes ipsilatérales

Sautillement sur 1 patte

Tests de correction

Station sur 2 pattes

Elévation de la tête

Tests du bord de la table

Réaction tonique du cou

Nerfs crâniens

Olfaction

Vision

Réflexes pupillaires

Strabisme

Sensation douloureuse (tête)

Asymétrie faciale

Désordre vestibulaire & surdité

Déglutition

Respiration

Langue

Tonus musculaire

Réflexes médullaires

Patellaire (Rotulien)

Tendon d'Achille

Triceps

Biceps

Flexion

Panniculaire

Sensation de douleur

Disorders of the Spinal Cord

Spinal cord disorders of cats or dogs are the most frequently encountered neurological diseases in small animal practice. Neurological deficits resulting from such diseases commonly affect locomotion, and therefore are easily recognized by dog and cat owners. Early diagnosis and accurate prognosis are essential for all spinal cord disorders, including those that are not amenable to treatment. Fortunately, many spinal cord diseases respond to appropriate medical and/or surgical treatment. Management of spinal cord disorders of small animals demands the ability to complete and interpret results of a neurologic examination and the ability to compile a list of differential diagnoses, and a knowledge of available diagnostic procedures and current treatment recommendations.

MECHANISMS OF DISEASE

Diseases that affect the spinal cord may be divided into two groups. The first group comprises disease processes that affect both the nervous system and other organ systems. The second group includes diseases that are unique to the nervous system, such as disorders of myelin, neurons or supporting cells (glial cells, etc.). Categories of disease that may be included in either of these two groups include: congenital and familial disorders, infections, immunologic and metabolic disorders, toxicities, nutritional disorders, trauma, vascular disorders, degenerations, neoplasia, and idiopathic disorders.

The localization of specific functions in the nervous system has important effects on the clinical presentation and nature of progression of diseases affecting the spinal cord. Localization of function in the spinal cord causes a similar pathologic process to result in many different clinical presentations, depending on which part(s) of the spinal cord it affects. For example, a spinal cord neoplasm located at the level of C3 vertebra may result in tetraparesis whereas the identical neoplasm located at the level of T13 vertebra may result in paraparesis with the thoracic limbs unaffected. Furthermore, localization of function in the spinal cord renders it inherently vulnerable to focal lesions that in other organs where function is more uniformly distributed might not result in detectable clinical signs. For example, a small infarction of the cervical spinal cord may result in tetraplegia, whereas a similar lesion occurring in hepatic parenchyma likely would not compromise liver function.

The unique susceptibility of the nervous system to a localized lesion is compounded by its strictly limited capacity to restore function in damaged tissue. The pathologic reactions of spinal cord to disease are to a degree nonspecific so that various disorders may induce a somewhat similar histologic appearance. With the exception of neoplasia and congenital malformations, most disorders of the spinal cord are characterized morphologically by the combination of a number of lesions that are not diagnostic when viewed in isolation. Certain of these lesions may be recognizable grossly, while others are seen only by means of microscopic examination and involve the cellular elements of the spinal cord. A topographic study of the lesions observed must therefore be combined with morphologic studies in order to arrive at an etiologic

diagnosis.

Clinical syndromes affecting the spinal cord may be characterized by a single focal lesion (transverse myelopathy) or by several focal lesions (multifocal disorders). Certain diseases may have a diffuse (or disseminated) distribution. Clinical differentiation of diffuse and multifocal disorders may be difficult. Myelopathies may be extrinsic, in which spinal cord dysfunction is secondary to diseases of the vertebrae, meninges, or epidural space, or may be intrinsic, in which the disease begins as an intramedullary lesion. Extrinsic myelopathies are almost always transverse myelopathies.

As the nervous system can respond in only a limited number of ways to the numerous causes of myelopathies, it is necessary to follow a systematic diagnostic approach in an animal with a spinal cord disorder. Such an approach ensures that less frequently encountered causes of myelopathy will not be misdiagnosed and consequently treated inappropriately.

DIAGNOSTIC APPROACH TO A SPINAL CORD PROBLEM

Signalment

Accurate diagnosis of a spinal cord disorder must include consideration of an animal's age, breed and sex. Diseases may be specific to certain species and breeds. Such diseases have been summarized by several authors.

History

An accurate and complete history constitutes the initial step in the diagnosis of all neurologic problems. Important aspects of history include rapidity of onset of the problem and the nature of its progression. This information may be helpful in determining the cause of a problem. For example, neoplastic diseases affecting the spinal cord most often result in focal signs that have an insidious onset and a gradual progression. In contrast, vascular disorders such as infarction or hemorrhage may produce an acute onset of focal signs without evidence of progression. Inflammatory, degenerative or metabolic disorders generally cause a diffuse distribution of signs that have an insidious onset and gradual progression. Traumatic and congenital diseases may result in either a focal or multifocal distribution of signs, most often with an acute onset and without progression, although such diseases may have a progressive course.

While careful consideration of these factors is helpful in determining the cause of a spinal cord problem, there are a sufficient number of exceptions to the general statements listed above that such information must be used with caution and should not be the sole basis for excluding a disorder from a list of differential diagnoses. For example, an acute onset of signs does not rule out neoplasia as a potential cause of myelopathy, as a neoplasm may be associated with rapid decompensation of neural tissue, particularly should vascular factors such as infarction or hemorrhage be involved.

Physical Examination

A physical examination consists of a series of observations that provide information regarding the general health of all body systems. Results of this examination are used to supplement information collected in history and may implicate involvement of body systems other than the nervous system. For example, an animal presenting with a

primary complaint of back pain may in fact have abdominal pain related to an underlying gastrointestinal or urinary system disorder.

A thorough orthopedic examination should be completed in any dog suspected of having a spinal cord disorder. Particular attention should be paid to the examination of joints for signs of effusion or abnormal motion. Disorders such as rupture of anterior cruciate ligaments bilaterally or bilateral patellar luxations may mimic paraparesis due to a neural disorder.

Neurologic Examination

A neurologic examination is an extension of a physical examination. When spinal cord disease is suspected, it is essential to complete a thorough, comprehensive and unbiased examination of the nervous system. Errors in diagnosis commonly occur when only the region of an obvious neurologic deficit is examined and more subtle alterations in other parts of the nervous system are overlooked.

The objectives of the neurologic examination are to detect the presence of and to determine the location and extent of a disorder of the nervous system.

Problem List

A complete list of problems should be compiled following completion of physical and neurologic examinations. All identified problems should be included, despite the fact that certain of the problems listed may not appear to be related directly to the presenting complaint of an animal. Problems should be listed at the most current level of understanding and should be updated, redefined or combined as more information is collected. Careful attention to maintenance of a problem list will ensure that all aspects of an animal's complaint are addressed during a workup. This is essential, as often there is a tendency for a clinician to focus a workup on an obvious neurologic deficit while ignoring a related problem. For example, a dog or cat with signs consistent with spinal cord infarction may have signs that reflect an underlying cardiovascular or endocrine problem.

Differential Diagnosis List

A list should be compiled of possible causes for each problem included on the problem list. Such a list should be exhaustive and the most probable causes should be listed first. Ranking of differential diagnoses is based on information collected in the history regarding signalment, nature of onset and progression of signs and on results of the physical and neurologic examinations.

Minimum Data Base

The minimum data base for a dog or cat with signs of spinal cord disease includes factors considered above (signalment, history etc). Initial clinicopathologic tests should include a complete blood count, blood chemistry profile and urinalysis. Results of these tests may result in addition of further problems to a problem list or may permit redefinition or combination of existing problems.

Results of initial blood and urine tests may support a diagnosis of a metabolic, toxic or infectious disorder that is either producing or complicating signs of spinal cord dysfunction. Additional diagnostic tests may be required to investigate disorders

suspected on the basis of results of initial screening tests. For example, hyperglobulinemia detected in a cat with signs of myelopathy may support completion of a serum feline infectious peritonitis (FIP) titer.

Thoracic or abdominal radiographs may be completed as part of the minimum data base in dogs or cats with a spinal cord disorder. This is especially necessary in older animals, in animals in which abnormalities of cardiovascular or respiratory function are suspected, or in animals in which neoplasia is included on a list of differential diagnoses.

Ancillary Diagnostic Investigations

Several diagnostic procedures aid in the differentiation of causes of a myelopathy. Certain of these procedures further aid in defining exactly the location and extent of a disorder affecting the spinal cord. The selection of an appropriate technique or techniques depends on results of physical and neurologic examinations and on the ranking of causes on a list of differential diagnoses. As a general rule, techniques should be selected to first investigate the most likely causes of a spinal cord disorder. The least invasive diagnostic tests and those with the lowest morbidity, should be completed first.

The recommended essential procedures for diagnosis of a myelopathy in advised order of completion are noncontrast vertebral radiography, cerebrospinal fluid (CSF) analysis, and myelography. Additional procedures may be added to this list, depending on the nature of the problem being investigated.

Noncontrast vertebral radiography. Noncontrast vertebral radiography is essential in the accurate diagnosis of a disorder affecting the spinal cord. A minimum requirement is that radiographs include the entire region of the vertebral column that may produce the observed clinical signs. Due to the limitations of a neurological examination in outlining multiple lesions of the spinal cord, and for the purpose of comparison should problems related to other regions of the vertebral column occur in the future, the entire vertebral column should be radiographed where possible. Correct technique, exact positioning and use of appropriate projections are essential considerations for the production of noncontrast vertebral radiographs that are of diagnostic quality. This subject has been extensively reviewed by several authors.

Further diagnostic investigations may be indicated on the basis of results of noncontrast vertebral radiography. For example, a finding of diskospondylitis may be followed by culture and sensitivity testing of blood, urine or an aspirate from an infected disk or by serologic testing for Brucella canis. Differentiation of an infectious lesion from a vertebral neoplasm may be difficult on the basis of results of noncontrast vertebral radiography. In such cases, a biopsy by means of needle aspiration or surgical excision may be indicated.

Cerebrospinal fluid. Cerebrospinal fluid collection and analysis are essential in cases where noncontrast vertebral radiographs do not fully define location, nature, and extent of a disorder affecting the spinal cord. Collection of CSF may be done by means of a cisternal or a lumbar subarachnoid puncture. It has been recommended that CSF be collected from a cisternal site should cervical spinal cord disease be suspected and from a lumbar location should a thoracolumbar disorder be involved. However, a lumbar collection site (most often between L5 and L6 vertebrae) may be used for most dogs or

cats with a spinal cord disorder regardless of the suspected location of the problem. Precautions must be used in the collection of CSF from animals where an increased intracranial pressure is suspected. Techniques for CSF collection and analysis have been discussed by other authors.

Analysis of CSF collected from a dog or cat with a spinal cord disorder should always include a total and differential white blood cell count and a quantitative estimation of protein content. Results for CSF from normal dogs have been published. It should be noted that normal values differ in CSF collected from lumbar and cisternal collection sites. Similar data for feline CSF do not exist, although it is anticipated that results for this species would be similar to those of dogs.

Results of CSF analysis may support further examination of CSF. For example, CSF may be submitted for bacterial or fungal culture and sensitivity testing, or for completion of viral titers.

Myelography. Contrast radiography should be done in those animals where results of noncontrast vertebral radiography and CSF analysis do not fully define a disorder affecting the spinal cord. Myelography is the radiographic examination of the spinal cord and emerging nerve roots following injection of contrast material into the subarachnoid space. Patterns of myelographic change can be used to differentiate intramedullary, intradural-extramedullary, and extradural space-occupying lesions.

This technique is difficult to perform, and myelography is not without undesirable consequences in all cases. Therefore, myelography should only be considered if positive findings are essential for diagnosis and prognosis or to determine precisely a site for surgery. Although myelography may be completed by means of either a cisternal or lumbar injection site, a lumbar injection site is preferred for dogs or cats with spinal cord disease at any level of the vertebral column. Use of dynamic radiographic techniques and completion of oblique projections may augment diagnostic information gained from a myelographic study.

Electrophysiology. There are several electrophysiologic techniques that may be applied to diseases affecting the spinal cord. Electromyographic (EMG) examination of paraspinal and limb musculature may be used to further define extent of a spinal cord lesion. This technique is ideally done prior to completion of noncontrast vertebral radiographs as results may help determine exactly the region or regions of the spinal cord that are affected, thus facilitating concentration of noncontrast radiographic studies to such an affected area. The role of EMG examination in the diagnosis of spinal cord disease is, however, somewhat limited. Abnormal EMG results associated with spinal cord disease are seen only when the lower motor neurons (LMNs) in the ventral horn of the spinal cord or their axons in the ventral root are affected by a pathologic process. The EMG will often be normal in association with disorders that affect primarily the spinal cord white matter. Furthermore, specific information regarding the cause of a myelopathy is not available by means of EMG examination.

Electromyographic examination may be used to define the extent of a lesion affecting the brachial or lumbar enlargement of the spinal cord. This may be accomplished by mapping the distribution of EMG abnormalities caused by denervation in

muscles of a thoracic or pelvic limb, and by correlating this information with the spinal nerve root origins of the nerves that supply the affected muscles of a limb. As it is possible by means of EMG to distinguish between disuse atrophy and atrophy that occurs secondary to denervation, such electrophysiologic findings may be valuable in precisely defining location and extent of a spinal cord lesion.

Sensory or motor nerve conduction velocities may be done on either a thoracic or pelvic limb of dogs or cats. The results of such studies may also aid in identification of nerve roots affected by a spinal cord disorder. Spinal cord potentials (cord dorsum potentials) evoked by stimulation of a peripheral sensory nerve may be used in combination with sensory nerve conduction velocity determinations to determine involvement of sensory nerve roots proximal to the dorsal root ganglia.

Percutaneously recorded evoked spinal cord potentials may provide a method for the localization of spinal cord lesions in dogs or cats as is the case in human beings. Furthermore, such studies may provide a noninvasive method for ascertaining the severity of a lesion, determining an accurate prognosis, and evaluating the response to therapy.

A variety of pressure, flow, and electrophysiologic techniques have been developed to assess the function of the lower urinary tract. Cystometry, urethral closure pressure profile recording, electromyography of urethral sphincter, uroflowmetry and evoked spinal cord potential measurements following pudendal nerve stimulation have all been invesitgated in dogs or cats. Combinations of these electrophysiologic tests may provide information regarding the functional status of spinal cord segments involved in micturition.

Additional Diagnostic Techniques. There are several additional diagnostic techniques that may be used for the accurate localization of a spinal cord disorder.

Linear tomography is a radiographic technique that may be used to cause selective blurring of images of superimposed objects, while maintaining some degree of image sharpness relative to a structure of concern. This is accomplished by coordinating tube-film movement about a central pivot point and thus requires specialized radiographic equipment. Since tomography is used to eliminate distracting shadows of superimposed structures, it is of value in assessment of complex objects such as the vertebral column. The lumbosacral junction, with overlying bodies of the ileum, and the thoracic vertebral column, with overlying ribs, are regions of the spine where linear tomography has particular application in vertebral radiography.

Other diagnostic techniques currently in use for the diagnosis of disorders affecting the vertebral column include epidurography, transosseous vertebral sinus venography, and diskography. These techniques will be discussed further under the section entitled lumbosacral vertebral canal stenosis.

X-ray computed tomography (CT) and magnetic resonance imaging (MRI) are being used with increasing frequency in the diagnosis of spinal cord disorders of dogs and cats. These techniques will aid in the accurate determination of location and extent of spinal cord diseases in the future.

CLINICAL SIGNS OF SPINAL CORD DISEASES

Complete assessment of an animal's gait and posture, postural reactions, spinal reflexes, cranial nerve function, and state of consciousness is essential in determining the presence or absence of spinal cord disease, the most likely location(s) of a spinal cord lesion, and whether a focal, multifocal, or disseminated disease process is present involving the spinal cord and/or other parts of the nervous system.

Bailey and Morgan (1983) describe five groups of clinical signs that are seen to a varying degree in all animals that have a disease affecting the spinal cord. These clinical signs are: depression or loss of voluntary movement, alteration of spinal reflexes, changes of muscle tone, muscle atrophy, and sensory dysfunction. Careful assessment of each of these groups of clinical signs in an animal suspected to have a disease affecting the spinal cord will facilitate lesion localization and diagnosis. Neurologic disorders that result in either loss of voluntary movement alone or sensory dysfunction alone are unlikely to be spinal cord disorders, as the majority of spinal cord diseases do not affect selected tracts while sparing anatomically adjacent pathways.

Diseases of the spinal cord may also result in dysfunction of bladder, urethral sphincter and anal sphincter, and in loss of voluntary control of urination and defecation. This may be due to interruption of spinal cord pathways connecting brainstem and cerebrum to bladder and rectum that are important in normal detrusor reflex function and voluntary control of micturition and defecation, or may be due to interruption of the parasympathetic nerve supply to the bladder, urinary and anal sphincters (L7 to S3 spinal cord segments and spinal nerves). Spinal cord diseases also indirectly interfere with excretory functions by impairing the ability of an animal to assume the posture necessary for normal defecation or urination.

Voluntary movement. Loss of voluntary movements due to interruption of motor pathways at any point from cerebrum to muscle fibers is referred to as paralysis (plegia). Lesser degrees of motor loss are referred to as paresis (or weakness). The terms tetraplegia (or quadriplegia) and tetraparesis (or quadriparesis) refer to abscence of voluntary movements in thoracic and pelvic limbs and weakness of movements in thoracic and pelvic limbs respectively. The terms paraplegia and paraparesis describe absence of voluntary movements and depression of voluntary movements in only the pelvic limbs. Hemiplegia and hemiparesis refer to paralysis or motor dysfunction of a pelvic limb and a thoracic limb on the same side.

Voluntary movements must be differentiated from reflex movements on the basis of neurologic examination findings and general observations.

Ataxia (incoordination) is seen in association with paresis, and probably occurs due to interference with both ascending and descending spinal cord pathways. Many ascending spinal cord tracts contribute to the transmission of sensory information to the cerebrum for coordination of voluntary movements, however interference with the spinocerebellar tracts probably causes a large part of the ataxia seen in association with spinal cord disease in animals. It must be remembered that observation of gait is the only means available for clinical testing of these pathways.

Spinal Reflexes. Spinal reflexes are stereotyped involuntary responses that occur independently of the brain, and can be elicited regularly by specific stimuli. The central nervous system (CNS) components of spinal reflex arcs are located entirely within the spinal cord. Disturbance of spinal reflexes occurs in almost all animals with spinal cord disease. A spinal reflex may be normal, depressed (hyporeflexia) or absent (areflexia), or exaggerated (hyperreflexia). Classification of spinal reflexes into one of these three categories is essential for localization of a spinal cord lesion.

Depression of a spinal reflex in association with spinal cord disease most frequently occurs as a result of involvement by a pathologic process of spinal cord segments mediating the reflex. It must be remembered that involvement of motor nerves arising from, or sensory nerves travelling to, such spinal cord segments, or abnormalities of the effector organ (muscle), may also result in depression of spinal reflexes.

Exaggeration of a spinal reflex in association with spinal cord disease occurs when a lesion affects the spinal cord cranial to segments that mediate a reflex. Neural mechanisms that result in spinal reflex exaggeration are not completely understood. The concept that reflex exaggeration simply results from interruption of descending inhibitory pathways is useful in the exercise of lesion localization, however other factors are likely to be involved, such as collateral axonal sprouting or the development of denervation supersensitivity. It is important to remember that reflex exaggeration may result from a brain lesion as well as from a spinal cord lesion.

Spinal cord lesions that affect both gray and white matter may result in depression of spinal reflexes mediated by spinal cord segments involved in a pathologic process, and in exaggeration of spinal cord reflexes mediated by spinal cord segments caudal to a lesion. This is useful in lesion localization, particularly for a lesion that affects the brachial enlargement (C6 to T2 spinal cord segments), where thoracic limb hyporeflexia and pelvic limb hyperreflexia may be present.

Interpretation of reflex abnormalities must be approached with the knowledge that two (or more) lesions located within the same anatomical division of the spinal cord may result in reflex changes identical to those produced by a single lesion. For example, two lesions between T3 and L3 spinal cord segments will cause hyperreflexia in the pelvic limbs that is indistinguishable from that resulting from a solitary lesion in this location. Further, hyporeflexia produced by one spinal cord lesion may mask hyperreflexia that would otherwise result from a second lesion in a more cranial location. For example, a lesion in the lumbar enlargement (L4 to S3 spinal cord segments) will cause hyporeflexia in the pelvic limbs that will mask the hyperreflexia that would otherwise result from a second lesion cranial to the L4 spinal cord segment.

Depression of spinal reflexes caudal to a lesion may be seen for several days following spinal cord injury in persons or primates. This phenomenon is called spinal shock. Sudden withdrawal of suprasegmental facilitation has been stated to be the likely cause. It is apparent that spinal shock occurs in quadrupeds, however it is too brief in duration to be of clinical significance. Hyporeflexia observed immediately following spinal cord injury should be attributed to damage to spinal cord segments mediating the reflexes, or to other systemic complications such as hypovolemic shock, that frequently accompany spinal cord trauma.

Muscle Tone. Maintenance of normal muscle tone is a function of spinal reflexes (tonic muscle stretch reflexes). Alterations of muscle tone are therefore interpretted in a similar fashion to spinal reflexes described above. Abnormal muscle tone may be depressed (hypotonia) or absent (atonia), or exaggerated (hypertonia), depending on the location of a spinal cord lesion.

Muscle Atrophy. Two types of muscle atrophy may occur in association with a spinal cord disease: 1. Denervation atrophy is seen when alpha motor neurons (LMNs) that innervate a muscle are damaged by a lesion affecting their spinal cord segment(s) of origin. Denervation atrophy is evident within a week of injury, usually is severe, and is associated with EMG abnormalities. 2. Disuse atrophy may be seen in muscles innervated by LMNs caudal to a spinal cord lesion. Disuse atrophy usually is slower in onset and progression than denervation atrophy, most often is less severe in character, and is not associated with EMG alterations.

Sensory Dysfunction. Abnormalities of sensory (ascending) pathways of the spinal cord contribute to the ataxia of spinal cord disease, however specific clinical tests of their function do not exist. Perception in animals must be inferred from certain behavioral responses that indicate ascending sensory signals have reached the cerebral cortex (eg, aversive response to a noxious stimulus). Interruption of sensory signals at any point between (and including) sensory receptors in the periphery and cerebral cortex may depress or obliterate normal sensory function. Therefore, results of clinical examination for signs of sensory dysfunction alone may not be of value in localizing a lesion to the spinal cord. However, when combined with results of other parts of a neurologic examination, signs of sensory dysfunction can provide important diagnostic and prognostic information.

Conscious proprioception (perception of body position or movement), and pain perception should be tested during a neurologic examination. Conscious proprioception is a sensitive indicator of spinal cord function, and depression or loss of conscious proprioception frequently are the signs first produced by any myelopathy.

Pain perception may be normal, depressed (hypesthesia) or absent (anesthesia), or exaggerated (hyperesthesia). Two types of pain perception are sometimes distinguished in animals. Cutaneous ("superficial") pain perception is manifested by a response to pricking or pinching of the skin, and deep pain perception is manifested by reaction to pinching the toes or tail across bone with hemostatic forceps. Areas of decreased or absent cutaneous pain perception may aid in identification of specific nerves, nerve roots, and spinal cord segments involved in a pathologic process. This technique of cutaneous mapping is especially useful in lesions that affect the cervical or lumbar enlargements.

Deep pain perception appears to be the sensory function that is most resistant to a spinal cord disease, and is the last spinal cord function to disappear in myelopathies of all types. An animal with a complete bilateral loss of deep pain perception due to a transverse myelopathy is necessarily paralyzed caudal to the lesion. Therefore, loss of deep pain perception is a very grave prognostic sign. Hyperesthesia in association with a spinal cord disease may indicate nerve root or spinal nerve involvement, or may be consistent with meningeal irritation. A focal area of hyperesthesia over the vertebral column may indicate the location a spinal cord lesion.

LOCALIZATION OF SPINAL CORD DISEASES

Motor, sensory, reflex and sphincter abnormalities may be used to determine the location of a lesion within one of four major longitudinal divisions of the spinal cord. The divisions are cervical (C1 to C5 spinal cord segments), cervical enlargement (C6 to T2), thoracolumbar (T3 to L3), and lumbar enlargement (L4 to Cd5). It is essential to remember that these divisions refer to spinal cord segments, not vertebrae, and that spinal cord segments do not correspond exactly with vertebrae of the same number. Some variations may be encountered due to slight differences between animals in segments that form cervical or lumbar enlargements. The diseases most commonly associated with neurologic signs referrable to each of these four regions are listed in Tables 1 through 4.

A disorder of each of the four regions of the spinal cord results in a combination of neurologic signs that is specific for the region involved. Recognition of a characteristic group of clinical signs therefore allows accurate localization of a spinal cord lesion. This concept of neurologic syndromes as a basis for lesion localization has been recommended by several authors. The presence of neurologic deficits indicative of involvement of more than one region of the spinal cord is highly suggestive of multifocal or disseminated spinal cord disease.

The functional differences between upper motor neurons (UMNs) and LMNs may be used to localize lesions to one of the functional regions of the spinal cord.

Cell bodies of spinal cord LMNs are located in the spinal cord gray matter. Their axons leave the spinal cord via the ventral nerve roots to become part of a peripheral nerve, and to terminate on a muscle. The LMNs of the thoracic limb have their cell bodies in C6 to T2 spinal cord segments that form the cervical enlargement, while LMNs of the pelvic limb arise from the L4 through S1 spinal cord segments of the lumbar enlargement. Anal and urethral sphincter LMNs originate from S1 through S3 spinal cord segments. Signs of LMN dysfunction, which in diseases affecting the spinal cord reflect damage to the spinal cord segment(s) from which LMNs originate, are: depression or loss of voluntary motor activity, depression or loss of muscle tone, and rapid, severe atrophy of an affected muscle due to denervation.

Upper motor neurons arise from cell bodies located in the brain. Their axons form descending pathways of the spinal cord, and terminate on interneurons that in turn synapse with LMNs. Lesions affecting UMNs result in UMN signs. These UMN signs result from an increase in the excitatory state of LMNs. Upper motor neuron signs include: depression or loss of voluntary motor activity, normal or exaggerated segmental spinal reflexes, appearance of abnormal spinal reflexes (eg, crossed extensor reflex), increased muscle tone, and muscle atrophy due to disuse.

Unilateral signs resulting from spinal cord disease are unusual, however signs frequently are asymmetrical. In the majority of cases, a lesion resulting in asymmetrical signs will be located on the side of greater motor and sensory deficit.

Cervical (C1 to C5). Fatal respiratory paralysis resulting from interruption of descending respiratory motor pathways or damage to motor neurons of the phrenic nerve (C5 to C7 spinal cord segments) occurs in a complete transverse myelopathy. Lesions

that are less than complete may not affect respiration, and in such cases other signs may be detectable.

Ataxia and paresis of all four limbs usually are seen. Tetraplegia rarely is seen, as lesions of sufficient severity to cause tetraplegia also produce respiratory paralysis. Hemiparesis occasionally may be present in association with a cervical lesion. Lesions of the cervical spinal cord may result in paraparesis with minimal neurologic deficits in thoracic limbs. The reasons for this are poorly understood.

Spinal reflexes and muscle tone are intact in all limbs, and may be normal or exaggerated. Muscle atrophy generally is not present, however disuse atrophy may develop in cases that have a chronic course. Anal reflexes are intact and anal tone usually is normal. Bladder dysfunction may occur due to detrusor muscle areflexia, with normal or increased urinary sphincter tone, and loss of voluntary control of micturition. Reflex dyssynergia may also be seen. Although voluntary control of defecation may be lost, reflex defecation will occur when feces are present in the rectum.

Horner's syndrome (ptosis, miosis, and enophthalmos) rarely may be present in an animal with a severe destructive, cervical lesion.

Conscious proprioception and other postural reactions usually are depressed or absent in all limbs. It should be remembered that complete loss of conscious proprioception may be present without detectable loss of pain perception.

Cervical hyperesthesia ("spasms", apparent pain on palpation, cervical rigidity, and abnormal neck posture) may be seen in some animals with cervical myelopathy. Occasionally an animal may hold a thoracic limb in a partially flexed position, a posture that may be consistent with C1 to C5 nerve root or spinal nerve entrapment ("root signature"), although this posture is seen more commonly with a disorder of the cervical enlargement.

Disorders that affect the cervical region of the spinal cord must be differentiated from brain lesions that result in tetraparesis. This can be accomplished by doing a comlete neurologic examination, however occasionally this distinction can be difficult. In most circumstances a cervical lesion does not result in neurologic deficits attributable to involvement of the medulla oblongata, however there are several notable exceptions to this rule. Positional strabismus resulting from loss of the vertebral joint proprioceptive input to the attitudinal reflexes, may be seen in association with a cranial cervical lesion (C1 to C3 spinal cord segments). A cranial cervical lesion may also cause facial hypesthesia as a result of involvement of the spinal nucleus and tract of the trigeminal nerve. Cranial cervical trauma often results in clinical signs referable to injury to the caudal brainstem (head tilt, pharyngeal paresis, facial paresis) or cerebellum.

The Schiff-Sherrington sign (syndrome or phenomenon) consists of hypertonicity of thoracic limb muscles and hyperextension of the neck, and is seen in association with spinal cord lesions caudal to the cervical enlargement. It is essential to differentiate this sign from thoracic limb hypertonicity caused by a cervical lesion.

Cervical Enlargement (C6 to T2). Ataxia and paresis of all four limbs usually are present. Occasionally paresis of thoracic limbs and paralysis of pelvic limbs may be seen.

Spinal reflexes and muscle tone may be normal or depressed in thoracic limbs, and normal or exaggerated in pelvic limbs. The nature of thoracic limb reflex alterations depends on the exact craniocaudal location of a lesion within this region. Muscle atrophy often is severe in thoracic limbs. Panniculus reflex may be depressed or absent unilaterally or bilaterally due to interruption of the LMNs involved in this reflex (C8 and T1 spinal cord segments).

Should bladder dysfunction occur it is similar to that observed with a lesion in the cervical region, with loss of voluntary control of urination. Anal reflexes and anal tone most often are normal although voluntary control of defecation may be absent.

Unilateral Horner's syndrome commonly is observed with a spinal cord lesion of the cervical enlargement, particularly a lesion involving T1 to T3 spinal cord segments or nerve roots.

Conscious proprioception and other postural reactions usually are depressed in all four limbs. Alterations in these functions may be more pronounced in the pelvic limbs than in thoracic limbs. Occasionally conscious proprioception will be absent only in a thoracic and pelvic limb on the same side.

Severe depression or loss of pain perception rarely are seen in association with a lesion of the cervical enlargement, except in intrinsic myelopathies (eg, ischemic myelopathy). There may be hyperesthesia at the level of a lesion of the cervical enlargement, thoracic limb lameness or apparent neck pain.

Thoracolumbar (T3 to L3). The majority of spinal cord lesions of dogs or cats occurs in this region. Typically thoracic limb gait is normal, and paresis and ataxia, or paralysis, are seen in pelvic limbs.

Thoracic limb spinal reflexes are normal. Pelvic limb spinal reflexes and muscle tone are normal to exaggerated, depending on the severity of the lesion. Muscle atrophy is not seen in thoracic limbs. Pelvic limb muscle atrophy, if present, is the result of disuse and is seen in animals with a severe, chronic lesion.

Anal reflexes and anal tone usually are normal or exaggerated. Voluntary control of defecation may be lost. Reflex defecation will occur when the rectum is filled with feces, however may not be at an appropriate time or place. Degree of bladder dysfunction varies depending on the severity of a spinal cord lesion. There may be loss of voluntary control of urination, detrusor muscle areflexia with normal or increased urinary sphincter tone, or reflex dyssynergia where initiation of voiding occurs and is stopped by involuntary contraction of the urethral sphincter. The bladder may be manually expressed in some animals, and not in others due to increased tone of the urinary bladder sphincter. This is often referred to as an "UMN bladder". Although "overflow" incontinence may occur with lesions of the spinal cord in this region secondary to overfilling of the bladder, detrusor muscle tone and urinary sphincter tone

are present, distinguishing this type of incontinence from that due to lesions of the lumbar enlargement and cauda equina ("LMN bladder").

Conscious proprioception and other postural reactions are normal in the thoracic limbs, and depressed or absent in the pelvic limbs.

Pain perception is normal in the thoracic limbs and may be normal, depressed or absent in the pelvic limbs. Panniculus reflex may be reduced or absent caudal to a lesion. In the lumbar region the panniculus reflex may be present in lesions caudal to L3 due to the pattern of cutaneous innervation of lumbar spinal nerves. There may be an area of hyperesthesia at the level of a lesion.

The Schiff-Sherrington sign may be seen with a lesion in this region. Usually it is an indication of an acute and severe spinal cord lesion, although such a lesion may be reversible.

Lumbar Enlargement (L4 to Cd5) and Cauda Equina. Involvement of this region by a pathologic process results in varying degrees of pelvic limb paresis and ataxia, or paralysis, and is often accompanied by dysfunction of bladder, and paresis or paralysis of anal sphincter and tail. Thoracic limb function is normal.

Pelvic limb reflexes and muscle tone are reduced or absent. Muscle atrophy often is present in pelvic limbs. Conscious proprioception and other postural reactions are reduced or absent in pelvic limbs.

Anal tone and anal reflexes are reduced or absent. The rectum and colon may become distended with feces, and fecal incontinence, with continual leakage of feces, is often seen. Constipation may result from the inability to void feces. Paresis or paralysis of the urethral sphincters and detrusor muscle result in overfilling of the bladder and "overflow" incontinence. Affected animals have a large residual volume of urine in the bladder, and the bladder is easily expressed manually.

The Schiff-Sherrington sign occasionally may be seen with an acute lesion affecting this region of the spinal cord.

The term cauda equina is used to describe the lumbar, sacral, and caudal nerve roots and spinal nerves as they extend caudally from the caudal tip (conus medullaris) of the spinal cord within the vertebral canal. Lesions that affect cauda equina result in clinical signs that are indistinguishable from lesions that affect the spinal cord segments from which the nerves of the cauda equina arise (L6 to Cd5).

Table 1: Diseases affecting the cervical region (spinal cord segments C1–C5)

Category of disease		Disease
Anomalous (Hereditary/congenital)	*	Atlantoaxial subluxation
	*	Congenital vertebral anomalies
		Spina bifida
		Myelodysplasia
		Syringomyelia/hydromyelia
		Globoid cell leukodystrophy
		Hereditary ataxia
		Pilonidal sinus/epidermal cyst
		Calcium phosphate deposition disease in great Dane dogs
Degenerative	*	Intervertebral disk disease
	*	Cervical spondylomyelopathy
		Demyelinating myelopathy of miniature poodles
		Leukoencephalomyeopathy of rottweilers
		Neuroaxonal dystrophy of rottweilers
		Spondylosis deformans
		Dural ossification
Inflammatory/infectious	*	Diskospondylitis
	*	Corticosteroid-responsive meningitis
	*	Granulomatous meningoencephalomyelitis/ reticulosis
	*	Distemper myelitis
	*	FIP meningitis/myelitis
		Bacterial/fungal/Rickettsial/protothecal meningitis/myelitis
		Protozoal myelitis
		Pyogranulomatous meningoencephalomyelitis
		Feline polioencephalomyelitis
		Spinal nematodiasis
Neoplastic	*	Neoplasia
Traumatic	*	Spinal cord trauma
Vascular	*	Ischemic myelopathy
		Necrotizing vasculitis
		Progressive hemorrhagic myelomalacia
		Hemorrhage
		Vascular malformations and benign vascular tumors
Nutritional		Hypervitaminosis A in cats
Idiopathic		Spinal arachnoid cysts
		Multiple cartilaginous exostoses

* Common causes of spinal cord disease

Table 2: Diseases affecting the cervical enlargement (spinal cord segments C5-T2)

Category of disease		Disease
Anomalous	*	Congenital vertebral anomalies
		Spina bifida
		Myelodysplasia
		Syringomyelia/hydromyelia
		Hereditary myelopathy of Afghan hounds
		Calcium phosphate deposition disease of great Dane dogs
		Globoid cell leukodystrophy
		Pilonidal sinus/epidermoid cyst
Degenerative	*	Intervertebral disk disease
	*	Cervical spondylomyelopathy
		Spondylosis deformans
		Dural ossification
Inflammatory/infectious	*	Diskospondylitis
		Distemper myelitis
		FIP meningitis/myelitis
		Protozoal myelitis
		Bacterial/fungal/Rickettsial/prototheca meningitis/myelitis
		Feline poliocencephalomyelitis
		Spinal nematodiasis
		Granulomatous meningoencephalomyelitis/ reticulosis
Neoplastic	*	Neoplasia
Traumatic	*	Spinal cord trauma
Vascular	*	Ischemic myelopathy
	*	Progressive hemorrhagic myelomalacia
		Necrotizing vasculitis
		Hemorrhage
		Vascular malformation and benign vascular tumors
Nutritional		Hypervitaminosis of cats
Idiopathic		Multiple cartilaginous exostoses
		Spinal arachnoid cysts

* Common causes of spinal cord disease

Table 3: Diseases affecting the thoracolumbar region (spinal cord segments T3-L3)

Category of disease		Disease
Anomalous	*	Congenital vertebral anomalies
		Spina bifida
		Myelodysplasia
		Syringomyelia/hydromyelia
		Mucopolysaccharidosis
		Globoid cell leukodystrophy
		Hereditary myelopathy of Afghan hounds
		Pilonidal sinus/epidermoid cyst
Degenerative	*	Intervertebral disk disease
	*	Degenerative myelopathy
		Hound ataxia
		Spondylosis deformans
		Dural ossification
Inflammatory/infectious	*	Diskospondylitis
	*	Distemper myelitis
	*	FIP meningitis/myelitis
		Bacterial/fungal/Rickettsial/protothecal meningitis/myelitis
		Protozoal myelitis
		Feline polioencephalomyelitis
		Spinal nematodiasis
		Granulomatous meningoencephalomyelitis/ reticulosis
Neoplastic	*	Neoplasia
Traumatic	*	Spinal cord trauma
Vascular	*	Ischemic myelopathy
		Progressive hemorrhagic myelomalacia
		Hemorrhage
		Necrotizing vasculitis
		Vascular malformations and benign vascular tumors
Idiopathic		Multiple cartilaginous exostoses
		Spinal arachnoid cysts

* Common causes of spinal cord disease

Table 4: Diseases affecting the lumbar enlargement (spinal cord segments L4-Cd5, or cauda equina)

Category of disease		Disease
Anomalous	*	Spina bifida
	*	Sacrocaudal dysgenesis
		Congenital vertebral anomalies
		Myelodysplasia
		Syringomyelia/hydromyelia
		Globoid cell leukodystrophy
		Pilonidal sinus/epidermoid cyst
Degenerative	*	Intervertebral disk disease
	*	Lumbosacral vertebral canal stenosis
		Spondylosis deformans
		Dural ossification
Inflammatory/infectious	*	Diskospondylitis
	*	Protozoal myelitis
		Distemper myelitis
		FIP meningitis/myelitis
		Bacterial/fungal/Rickettsial/prototheca meningitis/myelitis
		Feline polioencephalomyelitis
		Spinal nematodiasis
		Granulomatous meningoencephalomyelitis/ reticulosis
Neoplastic	*	Neoplasia
Traumatic	*	Spinal cord trauma
Vascular	*	Ischemic myelopathy
	*	Progressive hemorrhagic myelomalacia
		Hemorrhage
		Necrotizing vasculitis
		Vascular malformations and benign vascular tumors
Idiopathic		Multiple cartilaginous exostoses
		Spinal arachnoid cysts

* Common causes of spinal cord diseases

Intervertebral Disk Disease

Etiology and Pathogenesis

Degeneration of intervertebral disks may result in protrusion or extrusion of disk material into the spinal canal resulting in spinal cord compression and clinical signs ranging from apparent pain to complete transverse myelopathy. Degenerative changes may occur in any of the intervertebral disks (C2-3 to L7-S1) however disk protrusion or extrusion occurs most commonly in the cervical, caudal thoracic and lumbar spine. The intervertebral disks between T1 and T11 are stabilized dorsally by the intercapital ligaments that join opposite rib heads across the floor of the spinal canal over the dorsal anulus fibrosus of the intervertebral disk. These ligaments are closely associated with the dorsal longitudinal ligament located on the floor of the spinal canal and the dorsal anulus fibrosus of each disk. As a result, disk protrusion or extrusion is less likely in this region.

Two types of disk herniation (type I and type II) have been reported to occur in dogs by Hansen. Type I disk herniation occurs with degeneration and rupture of the dorsal anulus fibrosus and extrusion of nucleus pulposus into the spinal canal. Type I disk extrusion is most commonly associated with chondroid disk degeneration. Type II disk protrusion is characterized by bulging of the intervertebral disk without complete rupture of the anulus fibrosus. Type II disk protrusion is most commonly associated with fibroid disk degeneration.

Chondroid metaplasia of the nucleus pulposus and Type I disk extrusion occur most commonly in chondrodystrophoid breeds including Dachshund, beagle, Pekinese, Llasa apso, Shih tzu, and breeds with chondrodystrophoid tendencies including miniature poodle and cocker spaniel. Chondroid disk degeneration and type I disk extrusion may occur in any breed, including large breeds of dog. These authors have noted an unusually high incidence of type I disk extrusion in Doberman pinscher dogs.

Recent studies have demonstrated differences between the vertebral canal and spinal cord mensuration of Dachshunds and German shepherd dogs. The spinal cord of Dachshunds was found to terminate further caudally than that of German shepherd dogs. Further, the ratio of spinal cord to vertebral canal heights in the lumbar region was notably greater in Dachshunds than in German shepherd dogs. The smaller lumbar epidural space in Dachshunds may explain the occurrence of severe clinical signs seen in this breed in association with apparently small amounts of extruded disk material. It is also possible that the larger epidural space present in large breeds of dog may account for the fact that small amounts of extruded disk material within the spinal canal in these breeds may not cause spinal cord compression and associated clinical signs.

Chondroid degeneration of disks is characterized by an increase in collagen content of the disk, alteration of specific glycosaminoglycan concentration of the nucleus pulposus, and a decrease in water content of the disk. The normally gelatinous nucleus pulposus becomes progressively more cartilaginous and granular and eventually may mineralize (calcify). Extrusion of degenerative nucleus pulposus occurs through fissures

in, or rupture of, the anulus fibrosus. Hansen has reported that in chondrodystrophoid breeds of dog 75 to 100 per cent of all disks undergo chondroid metaplasia by one year of age. Recently, the radiographic pattern of degenerative changes that occur with aging in the vertebral column and intervertebral disks of beagles has been described. This study aids in the differentiation of clinically insignificant degenerative changes and pathologic changes that may produce clinical signs.

Fibroid disk degeneration occurs in older dogs of all breeds but is most often recognized as a clinical problem in older, large breed, nonchondrodystrophoid dogs and is characterized by fibrous metaplasia of the nucleus pulposus. An increase in the noncollagenous glycoprotein content of intervertebral disks occurs in nonchondrodystrophoid breeds of dog with aging. Calcification of the disk may occur, but is rare. Protrusion of the disk occurs with a bulging of the anulus fibrosus due to partial rupture of the anular bands. Rupture of the anulus fibrosus and extrusion of nucleus pulposus (characteristic of type I disk extrusion) uncommonly is seen in association with type II disk protrusion. Intervertebral disk protrusion or extrusion may occur in a ventral, dorsal or lateral direction. In most instances, only dorsal protrusions or extrusions are of clinical significance as meningeal irritation, nerve root and/or spinal cord compression may occur. Occasionally a lateral disk protrusion or extrusion may result in nerve root or spinal nerve compression with associated clinical signs.

The cause of intervertebral disk degeneration is unknown. Trauma does not appear to play a major role in chondroid degeneration but may be a factor in acute disk extrusion. Mechanical and anatomic factors are probably important, as disk extrusions are most common in the cervical and T11 to L3 regions of the vertebral column. Genetic factors probably have a role in the accelerated degeneration of disks in chondrodystrophoid breeds but the exact influence of these factors is not known. Hypothyroidism and autoimmune disease have also been proposed as contributing factors.

Type I disk extrusion often results in more severe clinical signs than type II protrusion although the mechanical distortion and compression of the spinal cord caused by type II protrusion may be greater. Nucleus pulposus is most often extruded into the spinal canal acutely (minutes to hours) or subacutely (days) from disks undergoing chondroid degeneration, whereas slowly progressive spinal cord compression most often accompanies protrusion of disks undergoing fibroid degeneration as the bulging fibrous mass increasingly enlarges within the spinal canal. The spinal cord changes seen in acute versus chronic spinal cord compression differ, and are reflected in the difference in clinical signs and response to treatment seen in these different types of intervertebral disk disease. The severity of spinal cord injury depends on the velocity at which the compressive force is applied, the degree of compression, and the duration of the compression. Vascular factors, as well as mechanical distortion of the spinal cord as a result of herniated disk material, are important in the pathogenesis of resulting spinal cord lesions. Severe spinal cord lesions may be found in spinal cord that does not have evidence of compression, presumably as a result of vascular changes.

Hemorrhage, edema and necrosis of both spinal cord grey and white matter is characteristic of acute spinal cord injury associated with acute type I disk extrusion. Hemorrhage and edema are not a major feature of chronic spinal cord compression where white matter changes such as demyelination, focal malacia, vacuolization and loss of axons are seen. Type I disk extrusions often are associated with rupture of vertebral

venous sinuses, and hemorrhage into the epidural space may increase the degree of spinal cord compression. Pulmonary emboli arising from the nucleus pulposus have been described in three chondrodystrophoid dogs with acute thoracolumbar transverse myelopathies as a result of type I disk extrusions, presumably as a result of disk material entering the vertebral venous sinuses. Nucleus pulposus may also penetrate the dura mater. Traumatic rupture of the anulus fibrosus and extrusion of normal nucleus pulposus may occur, resulting in spinal cord compression and an acute onset of clinical signs indicative of a transverse myelopathy.

Degenerative disk disease also occurs in cats, although the incidence of clinical signs associated with disk protrusion is low. Degenerative changes and distribution of disk protrusions is similar to type II disk protrusions in nonchondrodystrophoid dogs. Clinical signs seen usually are indicative of a slowly progressive transverse cervical or thoracolumbar myelopathy. Type I disk extrusion associated with calcification of intervertebral disks and an acute onset of neurologic deficits rarely has been reported in cats. Diagnosis and treatment are similar to that described for dogs.

Clinical Findings

Chondroid degeneration and type I disk extrusion most commonly occur in dogs three years of age and older, but may occur in younger animals. Fibroid degeneration and type II disk protrusion most commonly occur in dogs older than five years of age. There does not seem to be a sex predisposition for intervertebral disk disease.

Clinical signs seen with intervertebral disk disease vary depending on whether type I or type II disk herniation is present, the location of the lesion and severity of the spinal cord lesion. Clinical signs seen in association with type I disk extrusion include apparent pain and/or motor and/or sensory deficits. These clinical signs usually develop rapidly within minutes or hours of disk extrusion. However, clinical signs may progress slowly over several days or manifest periods of improvement and subsequent worsening over weeks or months. These findings are probably associated with extrusion of small amounts of disk material into the spinal canal over a period of time.

Clinical signs associated with type I disk extrusion in the cervical spine usually are less severe than those associated with extrusions in the thoracolumbar region. Although large amounts of disk material may be extruded in the cervical region, the vertebral canal in this region is larger in diameter in relation to the spinal cord than is the case in the thoracolumbar region. Apparent neck pain is the most common clinical finding in dogs with cervical disc extrusion. Affected dogs often hold the head and neck rigidly, cry out when moved and may show spasms of cervical musculature. Neurological deficits indicative of a cervical myelopathy such as proprioceptive deficits, tetraparesis or tetraplegia are seen less commonly.

Ipsilateral Horner's syndrome and hyperthermia have been described in cases of acute, severe, dorsolateral cervical disk extrusions. Lower motor neuron deficits in the thoracic limbs may be seen in caudal cervical disk extrusions. Thoracic limb lameness may also be seen in caudal cervical disk extrusions as a result of nerve root compression, particularly from lateral disk extrusions where disk material enters an intervertebral foramina.

Clinical findings in animals with thoracolumbar type I disk extrusion depend on the severity of spinal cord injury, and range from apparent back or abdominal pain to complete paraplegia and loss of deep pain perception. Neurologic deficits usually are indicative of a transverse myelopathy between T3 and L3, as most disk extrusions in this region occur between T11 and L3. Lower motor neuron signs may be seen in the pelvic limbs if disk extrusion occur caudal to L3 as a result of compression of the lumbosacral spinal cord or nerves of the cauda equina. Lower motor neuron signs also may be seen in paraplegic animals with progressive hemorrhage myelomalacia (PHM). The clinical signs and diagnosis of PHM will be discussed separately.

The panniculus reflex may be depressed or absent caudal to the site of disk extrusion. The site of a lesion is usually one or two vertebral spaces cranial to the loss or depression of panniculus reflex. The Schiff-Sherrington sign may be seen in animals with acute type I disk extrusion caudal to T2.

Clinical signs seen in both cervical and thoracolumbar type I disk extrusion may be asymmetrical, especially if extrusion occurs dorsolaterally within the spinal canal. Apparent pain associated with disk extrusions results from inflammation and/or ischemia caused by compression of meninges and/or spinal nerve roots. Extruded disk material initiates an extradural inflammatory reaction that results in fibrous adhesions between the dura mater and extruded disk material. Pain may also arise from stimulation of sensory nerve endings in the anulus fibrosus and dorsal longitudinal ligament. The nucleus pulposus of each disk does not contain nerve fiber endings.

Clinical signs associated with type II disk protrusion generally are slowly progressive over a period of months. Clinical signs, however, may develop acutely over days in some animals. Neurologic deficits usually are indicative of a cervical or thoracolumbar myelopathy. Paraparesis or tetraparesis, depending on the site of the lesion, are the most common clinical findings and deficits may be asymmetrical. In the cervical spine type II protrusions most commonly occur in caudal cervical disks. In some cases, caudal cervical type II disk protrusion may be part of the spectrum of abnormalities associated with cervical spondylomyelopathy. Apparent neck or back pain may or may not be a feature of type II disk protrusion.

Diagnosis

A tentative diagnosis of type I disk protrusion or extrusion may be made on the basis of age, breed, history and clinical signs, however other causes of transverse myelopathy or apparent pain should be considered in the differential diagnosis. It must be remembered that apparent spinal pain is seen in animals with meningitis. Dogs with thoracolumbar disk extrusions may show apparent abdominal pain, and in such animals causes of abdominal pain such as pancreatitis and peritonitis must be considered in the differential diagnosis.

The differential diagnosis in animals with type II disk protrusion includes all other causes of progressive transverse myelopathy, the most likely being neoplasia or degenerative myelopathy.

Spinal radiographs, and in almost all cases, CSF analysis and myelography are necessary to confirm a diagnosis of disk extrusion or protrusion. General anesthesia is

required to achieve the precise positioning needed to obtain radiographs of diagnostic value. Foam wedges or sandbags are usually needed to align the vertebral column parallel to the table top for lateral projections. Care must be taken however when anesthetizing and positioning for radiographs animals that have acute type I disk extrusions, as further extrusion of disk material and further spinal cord compression may occur with manipulation and movement of the spine.

Calcification of the nucleus pulposus is best seen on lateral radiographic views and usually is seen in one or more disks of most chondrodystrophoid dogs greater than one year of age. Calcified disks also may be seen in older nonchondrodystrophoid breeds of dog. The radiographic density of such disks varies from a slight haziness to that equal to the density of the vertebral body. Calcified material within the nucleus pulposus is indicative of disk degeneration, but alone is not of clinical significance.

The disk space of an extruded disk may be narrower than adjacent disk spaces and may be wedge shaped with a decrease in the width of the disk space dorsally. However, positioning is important as some disk spaces (C7-T1, T9-10 or T10-11 and L7-S1) are normally narrower than adjacent spaces and cervical and lumbosacral disks are normally wedge-shaped on hyperextension and flexion of the spine. "Spikes" of calcified material suggestive of disk extrusion may extend dorsally from a disk. Calcified material may be present within the vertebral canal but often is difficult to visualize due to overlying vertebral articular processes or ribs. Intervertebral foraminae are larger in the lumbar spine and calcified material often is easily visualized in the spinal canal in this region. Disk material within the spinal canal may appear as a hazy, indistinct shadow or as a dense mass with distinct margins. The former pattern often is seen associated with explosive disk extrusion and dissemination of disk material along the spinal canal whereas the latter pattern usually is associated with a slower extrusion of disk material over a longer time with dessication, fibrosis and possibly further mineralization of the disk material within the spinal canal. In many cases of disk extrusion calcified material is not visualized within the spinal canal, as disk material is probably not sufficiently mineralized to be visible on radiographs. Ventrodorsal views, and in some cases oblique views, are important in determining laterality of any visible mineralized material within the spinal canal. Vertebral osteophytes and vertebral end plate sclerosis may be seen associated with chronic disk degeneration and extrusion or in cases of chronic disk degeneration without disk extrusion or protrusion.

Type II disk protrusion may be associated with narrowing of the disk space, osteophyte production and end plate sclerosis. Calcification of disc material rarely is seen in association with type II disk protrusion. In some animals with type I or type II disk herniation obvious abnormalities are not seen on noncontrast vertebral radiography.

Myelography is almost always necessary to confirm that disk material has herniated into the spinal canal resulting in spinal cord compression. Myelography is most important in determining the site (or sites) of disk herniation, and lateralization of disk material within the spinal canal prior to surgical decompression. Myelography should not be done solely as a means of confirming a diagnosis of likely type I disk herniation in animals with signalment, history, clinical signs and radiographs that are highly suggestive of disk extrusion, where surgical decompression is not anticipated. Myelography, however, is necessary for diagnosis in most cases of type II disk protrusion as a means of

distinguishing disk protrusion from other causes of slowly progressive transverse myelopathy such as spinal neoplasia and degenerative myelopathy.

Cerebrospinal fluid should be collected and analyzed prior to myelography to rule out inflammatory or infectious disease of the spinal cord and/or meninges. Clinical signs in animals with GME, distemper myelitis, FIP, spinal lymphoma and other disorders may mimic those of cervical or thoracolumbar disk disease.

The characteristic myelographic findings in both type I and type II disk herniation into the spinal canal are extradural compression of the spinal cord with displacement of the spinal cord and narrowing of the subarachnoid space on lateral and/or ventrodorsal views, depending on the location of the compressive mass. Type II, and most type I, disk herniations result in a ventral or ventrolateral epidural mass that causes dorsal displacement of the spinal cord. Disc material may extend over more than one vertebral segment in type I extrusions and may result in deviation or narrowing of contrast columns over more than one vertebral length. Disk material may completely encircle the spinal cord. Acute type I disk extrusions often are accompanied by spinal cord edema and swelling. The spinal cord may be widened over several spinal cord segments and the myelographic appearance is similar to that of an intramedullary mass, making precise determination of the site of disk extrusion difficult. In some animals disk material is scattered along the spinal canal without obvious mechanical distortion of the spinal cord.

Rarely, in the cervical region, type I disk extrusion may occur laterally or intraforaminally resulting in neck pain or thoracic limb pain due to nerve root compression. In such cases myelography may be normal, however, increased density associated with calcified disk material may be visualized intraforaminally on ventral oblique radiographs of the cervical spine.

Traumatic disk protrusion is usually associated with narrowing of the intervertebral disk space on radiographs. Other abnormalities such as vertebral fracture, luxation or instability also may be seen. Myelography is useful in determining the presence or abscence of spinal cord compression in such cases, and therefore whether surgical decompression is indicated.

Treatment

Type I disk extrusion. The appropriate treatment for animals with type I disk extrusion depends on the animal's neurologic status. Each animal should be evaluated individually. Medical treatment directed at decreasing spinal cord edema by means of corticosteroids is indicated in all animals with an acute onset of neurologic deficits. The recommended dose is as for spinal cord trauma. The use of corticosteroids in dogs with type I disk extrusion has been associated with pancreatitis, gastrointestinal bleeding or colonic perforations. The incidence of these complications may be reduced by using lower doses of corticosteroids and administering potent injectable corticosteroids for as short a time as possible (maximum of one to two days).

Nonsurgical (medical or conservative) treatment is recommended for animals with apparent pain only or animals that have mild neurologic deficits but are ambulatory and have not had previous clinical signs associated with disk disease. These animals should be strictly confined to a small area as much as a hospital cage in a quiet place away from

other pets for at least two weeks and walked (on a leash or harness) only to urinate and defecate. The objective of confinement is to allow fissures in the anulus fibrosus to heal, thus preventing further extrusion of disk material, and allowing resolution of the inflammatory reaction caused by small amounts of extruded disk material. Confinement cannot be accomplished effectively by the majority of dog owners.

Use of analgesics, muscle relaxants and antiinflammatory drugs such as corticosteroids is not recommended in most cases as it is believed their use encourages animals to exercise and risk further disk extrusion. Very cautious use of analgesics or antiinflammatory agents occasionally may be indicated, however strict confinement followed by a period of restricted exercise is imperative. Owners should also be warned that an animal's neurologic status may deteriorate due to extrusion of further disk material despite this treatment and that an animal should be observed very carefully. Should worsening of neurologic status occur, an animal's treatment should be re-evaluated. Owners should also be warned that a recurrence of clinical signs is very common due to further disk extrusion at the same or a different site and subsequent episodes may be more severe, especially in the thoracolumbar spine.

Animals with severe cervical pain frequently do not respond to cage rest. These dogs often have large amounts of disk material within the spinal canal and dogs that do not show improvement after seven to 10 days of confinement should be evaluated further by means of radiographs and possibly myelography, and ventral cervical decompression should be considered.

Surgical disk fenestration has been recommended as a prophylactic measure to prevent further extrusion of disk material into the spinal canal. Fenestration of the disks most likely to herniate (C2-3 through C6-7 in the cervical spine and T11-12 through L3-4 in the thoracolumbar spine) is recommended in animals that have had one or more episodes of apparent neck or back pain and have evidence of intervertebral disk disease on radiographs. Various surgical techniques have been described. Fenestration of disks does not remove disk material from the spinal canal and therefore is not recommended as the sole surgical procedure in dogs that have evidence of disk material within the spinal canal and spinal cord compression on radiographs and myelography. Disk fenestration should be done with care in animals showing evidence of pain, as disk material may be forced into the spinal canal resulting in a worsening of neurologic status.

The role of disk fenestration in the management of intervertebral disk disease is controversial. Most authors feel fenestration of cervical disks is of value in preventing further disk extrusion and recurrence of apparent neck pain. In the thoracolumbar region some investigators report that disk fenestration does prevent recurrence of disk extrusion at the same or different sites, while others report that only spinal cord decompression at the site of initial disk extrusion is indicated, as the low incidence of recurrence at other sites makes disk fenestration unnecessary.

Disk fenestration in the thoracolumbar region is not easily done and complications such as scoliosis, pneumothorax and hemorrhage may occur. Disk fenestration in the cervical region is achieved more easily and rarely is associated with such complications. Fenestration does not prevent recurrence of disk extrusion in all animals. The effectiveness of fenestration depends largely on the amount of nucleus pulposus removed.

Completion of disk fenestration is recommended at the time of spinal cord decompression. This author recommends fenestration in either the cervical or thoracolumbar regions of the vertebral column for dogs that have recurrent bouts of apparent pain resulting from type I intervertebral disk disease.

Animals with neurologic deficits such as paresis or paralysis with deep pain perception intact, animals with recurrent bouts of apparent back or neck pain, or animals with apparent back or neck pain (or mild neurologic deficits) that are unresponsive to strict confinement should be evaluated by means of spinal radiographs, CSF analysis, and myelography. Surgical decompression of the spinal cord and removal of disk material from the spinal canal should be considered. Although many dogs with moderate or severe paresis will improve neurologically if treated with corticosteroids and cage rest, 269 neurologic recovery is often more rapid and more complete in animals following surgical decompression of the spinal cord. In addition the neurologic status of some dogs with type I disk extrusion, especially in the thoracolumbar spine, suddenly worsens over a period of hours or days despite medical treatment. Such deterioration usually results from further disk extrusion that may result in irreversible spinal cord damage and permanent paralysis. This progression of signs always is a risk with medical treatment of animals with thoracolumbar disk disease. As such progression is impossible to predict on the basis of history, clinical signs or radiography, owners should be made aware of treatment options and offered the opportunity of referral to an appropriate surgical facility when animals are initially presented. Surgical decompression should be done as soon as possible to prevent further spinal cord damage incurred as a result of sustained compression or further extrusion of disk material. In addition, if surgery is delayed two to three weeks, disk material hardens and becomes adherent to dura mater, and becomes difficult or impossible to remove from the spinal canal.

Prognosis for neurologic recovery in animals that retain deep pain perception postsurgically is fair to very good. The major factors that correlate with the degree of neurologic improvement seen postsurgically are the animal's neurologic status prior to surgery, the rapidity of onset of clinical signs, and the time interval between onset of clinical signs and surgical decompression. Animals that have severe neurologic signs, a rapid onset of clinical signs (hours) and a long period of time before surgery generally have a prolonged recovery period and may have varying degrees of permanent neurologic deficit.

The incidence of recurrence of clinical signs due to disk extrusion is greater in nonsurgically than surgically treated dogs. One author found that one third of dogs with type I disk herniation that were treated nonsurgically had a recurrence of clinical signs, and generally showed greater severity of neurologic deficits at the time of recurrence. Another author reported a recurrence rate of 40% in nonsurgically treated dogs.

The advantages and disadvantages of various techniques for spinal cord decompression have been discussed. Surgical treatment is not without risks. Anesthesia is necessary, and surgery occasionally results in further spinal cord damage due to surgical manipulation. Nonsurgical treatment should be attempted in animals that are poor anesthesia or surgical candidates or if surgical treatment is not possible financially.

In animals with clinical signs of a complete transverse myelopathy, without deep

pain perception for a period of greater than 24 hours, the prognosis for return of spinal cord function is very poor despite medical or surgical treatment. A small percentage of these animals may improve neurologically if given sufficient time, however, surgical treatment does not appear to increase the probability of improvement and usually is not recommended. In cases in which deep pain perception has been absent for less than 24 hours the prognosis for return of spinal cord function is guarded to poor, however surgical treatment may increase the likelihood of neurologic improvement in this group. Use of evoked spinal cord potential monitoring may aid in deciding whether or not spinal cord function remains and whether improvement is likely.

Regardless of whether medical or surgical treatment is instituted, animals that are paretic or paralyzed require intensive nursing care. Neurologic improvement may take weeks or months and this requires owner cooperation and enthusiasm regarding care and physical therapy. Manual expression, intermittent catheterization and/or indwelling catheterization of the bladder is often required to ensure emptying of the bladder. Weekly urinalysis, especially in animals that do not have voluntary control of micturition, is important in monitoring for urinary tract infection. It is also important to keep animals well padded, clean and dry to prevent formation of pressure sores, and to ensure caloric and water intake is adequate. Physical therapy will not result in neurologic improvement but helps prevent disuse muscle atrophy associated with paraplegia or tetraplegia. Physical therapy should not be attempted in animals treated medically for at least the first two weeks following onset of signs, as further extrusion of disk material may occur.

Type II Disk Protrusion. Treatment with corticosteroids may result in neurologic improvement for variable periods of time in animals with type II disk protrusion. However, corticosteroid therapy is not curative. The reason for this improvement is not clear as intramedullary hemorrhage and edema seen in cases of acute spinal cord injury are not a feature of chronic spinal cord compression. In the thoracolumbar spine surgical removal of protruded disk material is generally impossible without causing further spinal cord damage. Surgical decompression without removal of protruded disk material may result in improvement, however the neurologic status of some dogs is worsened permanently despite very careful surgical technique. The reasons for this are not known but increased vascular permeability has been described in the spinal cord associated with release of chronic spinal cord compression and this probably plays a role in this phenomenon. Ventral decompression in the cervical spine allows removal of protruded type II disk material and neurologic improvement may occur, however some dogs, especially those with moderate to severe neurologic deficits prior to surgery, may manifest temporary or permanent worsening of clinical signs postoperatively. Neurologic improvement may take several months and is believed to be primarily due to remyelinization of axons in the white matter of the spinal cord.

Chemonucleolysis

Injection of the proteolytic enzyme chymopapain into the nucleus pulposus of intervertebral disks to cause discolysis has been used infrequently in veterinary medicine. The precise mechanism by which chymopapain causes dissolution of the nucleus pulposus is unknown. In one study in dogs, dissolution of the nucleus pulposus was demonstrated histologically in all cervical intervertebral disks injected with chymopapain via a ventral surgical approach. Similar pathologic findings were found in lumbar disks of dogs injected with chymopapain transcutaneously under fluoroscopic guidance via a lateral

approach. Significant postoperative clinical complications were not seen. Radiographic narrowing of the intervertebral disk spaces was found in both the cervical and lumbar spine, beginning on the second day after injection of chymopapain. Cervical injection resulted in a more noticeable narrowing than lumbar injection. However, successful injection as determined histologically was not always detected radiographically. These studies have described only the acute response to chymopapain. However, another study has shown chymopapain results in progressive dissolution of nucleus pulposus and eventual regeneration of nuclear ground material.

Chemonucleolysis may be of benefit in animals with intervertebral disk disease when the nucleus pulposus is still contained within an intact or partially ruptured anulus fibrosus. Dissolution of nucleus pulposus in these cases may relieve pressure of the protruding disk on the spinal cord and nerve roots. Chemonucleolysis may also be useful as a prophylactic measure in animals with evidence of intervertebral disk degeneration to prevent acute Type I disk extrusion.

Chemonucleolysis is not indicated in cases of Type I disk extrusion, as the enzyme is unable to reach sequestered nucleus pulposus within the spinal canal. Chemonucleolysis has been used in the treatment of Type II disk protrusion in the cervical spine of large breeds of dog. The majority of dogs in one study improved clinically despite persistence, or only slight decrease, in the degree of spinal cord compression on myelography. Injection of chymopapain via a surgical approach to the intervertebral disks is recommended to prevent inadvertent intrathecal injection or accidental penetration of the vertebral arteries, spinal arteries or spinal nerve roots. Further evaluation of the effect of chemonucleolysis in dogs with intervertebral disk disease is needed, however it seems likely that this technique may have advantages over the presently used methods for surgical disk fenestration.

Table: Summary of indications for therapy for Type I intervertebral disk extrusion of dogs.

Type of therapy		Indications
1.	Medical	Apparent back pain only - 1st episode or Mild ataxia and paresis - 1st episode or Paralysis with absent deep pain perception for greater than 24 hours
2.	Surgical	
	a. Fenestration only	Apparent back pain only - 2nd or multiple episodes
	b. Decompression (and fenestration)	Mild ataxia and paresis - 2nd or multiple episodes or Moderate/severe ataxia and paresis, or paralysis - 1st episode or Deterioration of neurologic status despite adequate medical therapy or Paralysis with absent deep pain perception for less than 24 hours

Techniques de neuro-radiologie

L'utilisation de l'imagerie en médecine vétérinaire en temps que moyen diagnostique s'est beaucoup développée ces dernières années. L'imagerie utilise aujourd'hui la tomodensitométrie, la résonance magnétique, l'échographie, la scintigraphie, qui sont des techniques de haute technologie. Ces procédures diagnostiques sont maintenant utilisables en médecine vétérinaire mais vont-elles supplanter la radiologie conventionnelle ? L'utilisation de la radiologie en neurologie n'a un intérêt que si l'examen neurologique et son interprétation ont été réalisés. En neurologie, l'imagerie permet de confirmer l'existence d'une lésion, de préciser son étendue, d'avancer un pronostic, et de proposer un traitement adapté si il en existe. Cette première partie comprend les notions de base de la neuro-radiologie de la colonne, à savoir sa technique, son interprétation, et ses indications.

Conseils généraux:

La radiologie est une technique d'imagerie des structures anatomiques normales ou pathologiques. Elle permet de détecter et d'interpréter des changements qui peuvent être présents. En neuroradiologie il est nécessaire d'avoir une parfaite connaissance de l'anatomie du segment vertébral que l'on veut imager, des divers types de lésions qui peuvent atteindre ce segment, et des avantages et des limites des techniques utilisées. D'autres paramètres à considérer lors du choix de la radiologie comme étape diagnostique sont l'aspect financier et l'équipement dont on dispose d'autant plus lorsque l'utilisation de produit de contraste suit les radiographies dites sans préparation.

La radiologie est une technique d'imagerie rapide et simple. Elle doit être la première étape diagnostique lors de manifestations cliniques médullaires afin de confirmer ou d'infirmer les pathologies les plus courantes de la colonne. C'est une aide au diagnostique et n'autorise en rien d'éviter l'examen neurologique qui permet la localisation de la lésion. Les indications classiques sont les douleurs axiales, les palpations anormales ou mal alignements vertébraux, et les déficits neurologiques dont l'origine est médullaire. Le principal avantage de la radiologie, est que c'est un moyen de détection rapide des lésions osseuses. Par contre une de ses limites est le manque de contraste des tissus mous sains ou lésés qui empêche la visualisation médullaire, celle des racines nerveuses, des disques intervertébraux, et des ligaments vertébraux. Ces structures peuvent être indirectement évaluées en les comparant aux structures osseuses avoisinantes (diminution de taille de l'espace intervertébral, augmentation du diamètre d'un foramen, calcification dégénérative). L'existence d'une superposition osseuse des structures vertébrales irrégulières (facettes

articulaires, processus latéraux, apophyses dorsales), les variations anatomiques d'un animal à l'autre, et la présence de changements dégénératifs qui n'ont aucune incidence clinique (spondylose, calcification méningée) sont d'autres limites.

Considérations techniques:

L'équipement nécessaire à la neuroradiologie est un peu plus important que celui nécessaire à la radiographie courante. Des générateurs puissants sont souvent nécessaires (jusqu'à 100 kV et 300 mA). Le temps d'exposition doit être long (0,1 à 0,3 seconde) ce qui permet d'utiliser une grille sur des animaux anesthésiés de préférence. Cela donne une échelle de contraste plus petite, donc plus de blanc et de noir. Un bon diaphragme est souhaitable. Il permet de diminuer les rayons diffractés et obliques. Le rayon le plus perpendiculaire doit être le plus central. Comme beaucoup de lésions sont très subtiles, des écrans de haute résolution sont nécessaires. Pour une épaisseur inférieure à 9 cm, une cassette comprenant un écran peut s'utiliser en direct. Au delà, une grille fixe permet de limiter la dispersion des rayons. L'écran utilisé doit être adapté aux capacités de l'équipement et au type de film. Les cassettes et écrans doivent être très propres et d'excellente qualité. Des coussins radiotransparents et des sacs de lestage pour assurer un bon positionnement sont indispensables.

Les artefacts dus aux mouvements et aux malpositions rendent l'interprétation des radiographies difficile voir impossible. Une anesthésie générale ou une tranquilisation profonde est nécessaire afin de positionner l'animal parfaitement. Elle permet aussi de régler l'appareil avec un temps d'exposition assez long sans que l'animal ne bouge. Parce que bon nombre de lésions vertébrales sont discrètes et de faible densité tissulaire, la technique doit être aussi parfaite et répétitive que possible. L'anesthésie garantit un bon résultat, une bonne reproductibilité et une minimisation des frais et des risques d'exposition aux rayons.

Positionnement:

Deux clichés perpendiculaires l'un à l'autre (une vue latérale et une vue ventro-dorsale) sont nécessaires pour l'évaluation d'un segment vertébral. Les segments cervicaux, cervico-thoraciques, thoraciques, thoraco-lombaires, lombaires, et lombo-sacrés doivent être examinés séparément car leur épaisseur est différente. D'autre part, comme le faisceau de rayons dévie crânialement et caudalement, l'incidence n'est vraiment perpendiculaire qu'au centre de la radiographie. La colonne est positionnée parallèlement à la table à l'aide de matériel radiotransparent (serviette éponge, coin de mousse) de façon à ce que les rayons incidents soient

perpendiculaires aux corps vertébraux et parallèles aux espaces intervertébraux. Ce matériel radiotransparent est placé sous le milieu du cou et de la région lombaire de façon à ce que les laminas droite et gauche de chaque vertèbre se superposent. Ceci facilite l'évaluation des espaces intervertébraux, des facettes articulaires au niveau cervical, et des foramens au niveau thoraco-lombaire. Une cale est placée sous le museau et les membres afin de superposer les processus latéraux. La position ventro-dorsale place la colonne très proche du film ce qui permet de conserver un maximum de détails et assure un minimum de distorsion. L'animal doit être parfaitement symétrique, et pour ce, on utilise un berceau radiotransparent ou des cales latérales. Le rayon le plus perpendiculaire doit être centré directement au dessus de l'endroit suspect. La divergence latérale du rayon rend la lecture des bords latéraux de la radiographie difficile. Pour éviter la superposition des plaques articulaires des corps vertébraux, plusieurs petits films doivent être utilisés. Ainsi deux radiographies (centrées sur T13-L1 et L4-L5) peuvent suffire sur un petit chien pour imager la région thoraco-lombaire et lombaire, alors que trois ou quatre peuvent être nécessaires pour un chien de grande taille (centrées sur T9-T10, T13-L1, L3-L4, et L7-S1).

Aspect pratique:

1- Jonction Occipito-atlanto-axiale

latérale droite
latérale en hyperflexion
oblique
ventro-dorsale bouche ouverte

Une vue en hyperflexion a un intérêt diagnostique en cas d'instabilité atlanto-axiale ou atlanto-occipitale. Cette vue est à réaliser avec beaucoup de précaution parce qu'une hyperflexion forcée peut entraîner une lésion médullaire. Une vue latérale oblique de 20o de l'articulation C1-C2 ou ventro-dorsale bouche ouverte peut permettre de diagnostiquer une agénésie, une mal angulation ou une fracture de la dent de l'axis.

2- Colonne cervicale

latérale droite
ventro-dorsale
C5-C7 (chez les grands chiens), latérale et ventro-dorsale
oblique (x2)
latérale en flexion
latérale en extension
latérale en traction

L'intérêt de vues en hyperflexion et hyperextension des vertèbres cervicales, sans injection de produit de contraste, pour évaluer une éventuelle instabilité est probablement très réduit. Par contre, une vue en traction peut mettre en évidence une instabilité vertébrale dynamique. Des vues cervicales obliques (de 45o par ce que les apophyses latérales sont perpendiculaires aux laminas) permettent de visualiser alternativement les foramens droits ou gauches.

3- Colonne Thoraco-lombaire

latérale droite
ventro-dorsale
jonction thoraco-lombaire
oblique (x2)

Au niveau thoraco-lombaire, les apophyses latérales étant ventro-obliques, elles ne se superposent pas aux foramens lors de vues latérales. Des vues obliques au niveau thoraco-lombaire sans utilisation de produit de contraste sont donc sans intérêt.

4- Colonne lombo-sacrée

latérale droite
ventro-dorsale
latérale en flexion
latérale en extension

Des vues en hyperflexion et hyperextension de la jonction lombo-sacrée peuvent être intéressantes pour mettre en évidence une instabilité dynamique.

Développement et lecture:

Le développement automatique non seulement est plus rapide mais il permet une meilleur standardisation des résultats. Lire un film humide est source d'erreur. Ne pas utiliser un négatoscope en est une autre. Il est toujours souhaitable de disposer les radiographies dans la même position sur le négatoscope de façon à utiliser sa mémoire photographique qui avec l'habitude, porte le regard sur ce qui n'est pas normal. Les lésions sont plus facilement misent en évidence sur des radiographies qui ne montrent que la colonne que sur des radiographies qui prennent l'ensemble de l'animal. Certaines images peuvent paraître bizarres mais être tout à fait normales: Une région très peu radiodense est visible au niveau du canal vertébral de C5 et C6; les espaces intervertébraux C2-C3, C7-T1 et T10-T11 sont toujours plus réduits que les autres; les faces ventrales des corps vertébraux de L3 et L4 apparaissent peu définies du fait de l'attache diaphragmatique.

Les signes radiographiques de lésion vertébrale sont des changements de forme, de taille, d'alignement, de densité. Les changements de forme, de taille et d'alignement ont des origines congénitale ou traumatique; les changements de densité (sclérose, néoformation, lyse) ont des origines infectieuse, dégénérative ou néoplasique.

Conclusion:
Avec un équipement optimal, la radiographie conventionnelle, surtout si elle est suivie d'une technique de contraste est une procédure diagnostique très utile en neurologie, surtout en ce qui concerne les lésions de la colonne. Mais encore une fois, elle est inintéressante sans un bon examen neurologique.

RADIOLOGIE . INTERPRETATION DU FILM SANS PREPARATION

L'appareil de radiographie

Il convient de disposer d'un générateur de 100 milliampères et de 100 kilovolts au minimum.Ceci autorise un temps d'exposition relativement court ,inférieur au 1/10 eme de seconde,malgré la grande épaisseur des tissus à traverser .
L'appareil doit posséder un collimateur afin de permettre une bonne focalisation du rayonnement,et la possibilité d'incliner le rayon principal par rotation transversale du tube.

Les films

Si l'utilisation des films sans écrans est théoriquement possible à partir d'une installation de 300 mA,le temps d'exposition relativement long et la perte de certaines images de tissus de faible densité en limite l'usage .
Le film avec écran renforçateur est le type le plus couramment employé.
La qualité de l'écran entre alors en ligne de compte.
L'écran renforçateur standard à grain moyen suffit si le générateur est assez puissant et si le système anti -diffusion est de bonne qualité. Cependant les progrés en cette matière permettent de disposer d' écrans à haute définition à grain trés fin et à vitesse rapide (écrans aux terres rares).Ces écrans associés à des films orthochromatiques autorisent l'utilisation de générateurs de faible puissance.

Le système anti diffusion

Le système anti-diffusion a pour but de supprimer le rayonnement secondaire lié à l'effet Compton .Celui ci devient important lorsque l'épaisseur du sujet à radiographier dépasse 9 cm ,ce qui est généralement le cas .
On utilise une grille anti diffusion soit fixe soit mobile.
Une grille fixe est soit focalisée soit non focalisée .On la choisira aussi fine que possible .
Une grille mobile est montée sur un système à déplacement longitudinal appelé POTER BUCKY ou POTER .

l'anesthésie

La plupart des lésions discales sont de petite taille et leur densité radiologique est parfois faible.Il est donc absolument indispensable qu'aucun mouvement parasite ne vienne créer un "flou radiologique".L'immobilité du sujet se révèle indispensable,car la rapidité du temps d'exposition est égale ou seulement un peu inférieure au 1/10 eme de seconde Il est même fortement recommandé de supprimer à l'instant du cliché les mouvements respiratoires ,ceux -ci étant particulierement important à la jonction thoraco-lombaire la zone la plus souvent incriminée.
On utilise dans ce but des anesthésiques fixes -Penthiobarbital- des associations d'anesthésiques fixes (Xilazine , Kétamine);l'anesthésie gazeuse Ils permettent en outre de maintenir l'animal dans la position idéale pour le cliché

PROTOCOLE ET POSITIONS RADIOLOGIQUES

Par sa longueur,sa complexité anatomique,la diversité des éléments qui la constitue,la colonne vertébrale nécessite l'emploi d'incidences multiples.C'est à ce niveau que se vérifie le mieux les grandes lois de l'optique radiologique.

Tout d'abord en raison de sa longueur il ne saurait etre question de radiographier le rachis en entier.
Aux distances focales habituellement employées (< à 1 m),pour obtenir nettement la projection d'un espace inter vertébral, le foyer du tube doit se trouver dans le plan de séparation de 2 vertèbres et ce plan doit contenir le rayon principal .Cette condition ne peut se réaliser sur plus de 2 ou 3 vertèbres .Au delà des déformations importantes se produisent dues à la projection conique .
Il est donc indispensable de multiplier les incidences
Ensuite en raison des courbures de la colonne vertébrale.
Il est important que le rayonnement soit perpendiculaire à l'axe vertébral.

Les deux positions radiologiques de base sont :
le décubitus latéral droit ou gauche et le
décubitus dorsal ou ventro-dorsal

Dans le cas du décubitus latéral la colonne va subir des déformations passives dans le plan longitudinal (scoliose) ou dans le plan sagittal (rotation)
dans le plan longitudinal on rétabli la rectitude par la mise en place de coussins radio-transparents (billes de polystirène)ou de coins de mousse dans les zones en dépression (creux lombaire ,région scapulaire .....)
Dans le plan sagittal on corrige la rotation en surélevant les membres profonds.

Suivant le segment à radiographier on modifie légèrement les positions standarts

Rachis cervical
Deux positions standards ,une position complémentaire

Décubitus latéral.(incidence latérale)
C'est de loin la position la plus intéressante .
L'animal en décubitus latéral la tete en légère extension sur l'encolure les membres antérieurs tirés en arrière .
La rectitude est assurée par des supports radio-transparents ou mieux encore par l'opérateur lui meme qui réalise en meme temps une traction modérée ce qui a pour effet d'écarter les espaces intervertébraux normaux

Décubitus dorsal (incidence ventro - dorsale)
L'animal sur le dos ,la tete dans la continuité de l'encolure ,les membres tirés en arrière .
Dans ce cas le rayon principal doit faire un angle de 5 à 10[avec la verticale dans la direction craniale ceci afin d'etre parallele à l'axe de l'espace inter-vertébral.

Position complémentaire
Le décubitus oblique ventro-latéral droite
ventro-latéral gauche

A partir de la position dorsale on fait subir une rotation à gauche ,puis une rotation à droite d'environ 30[.cette position fait passer le rayon principal dans l'axe des trous de conjugaison permettant ainsi de visualiser une éjection discale latérale .

Nombre d'incidences par position :

Deux suffisent . A la hauteur de C3 et à la hauteur de C6

Rachis thorace-lombaire

Deux positions : décubitus latéral
décubitus ventro-dorsal

Décubitus latéral
Dans la position latérale on veille à la correction de la scoliose entrainée par le décubitus et à la correction de la rotation .

Nombre d'incidences ,il est de trois .

A la hauteur de T10/T11 ,à la hauteur de T13/L1 ,et à la hauteur de L3/L4 .

Décubitus ventro-dorsal
Le décubitus ventro-dorsal n'a pas de caractère particulier.Le nombre d'incidence est limité à une incidence à la hauteur de l'espace considéré comme pathologique sur la radiographie en décubitus latéral.

En règle générale la position ventro-dorsale confirme une pathologie décelée sur une vue latérale .Si aucune lésion n'est visible sur le cliché en vue latérale ,il y a peu de chance pour que la vue dorsale apporte un élément positif

LECTURE DU CLICHE

La première condition d'une bonne lecture est de disposer de radiographies sèches .Aucune lecture ne doit etre faite sur un cliché humide
La deuxième condition est de disposer d'une source lumineuse adaptée à cet examen (négatoscope).
La troisième est de bien connaître l'anatomie radiologique normale et de réaliser un examen analytique dont le déroulement sera toujours identique .

Eléments de lecture

Rappelons les éléments d'anatomie radiologique impliqués dans la hernie discale Ces éléments sont situés à la périphérie de l'espace intervertébral.Il s'agit :

du rebord cranial et caudal des vertèbres ou PLATEAU VERTEBRAL
de l'espace délimité par ces deux plateaux ou ESPACE INTER VERTEBRAL
du plancher du canal vertébral
du trou de conjugaison
des apophyses articulaires

On commmence l' examen par les zones préférentiellement atteintes :
au niveau du rachis cervical on examine l'espace C2 C3 puis les espaces plus postérieurs .
Au niveau du rachis thoraco-lombaire on commence par l'espace T13 L1 puis on s'éloigne de part et d'autre de cet espace .

La lecture s'effectue en comparant la zone suspecte à celles immédiatement postérieure et immédiatement antérieure .On compare:
-la distance séparant les deux plateaux vertébraux
-le parallélisme de ces deux plateaux
-la transparence de l'espace inter-vertébral
-l'aspect du plancher
-le diamètre du trou de conjugaison
-la distance séparant les apophyses articulaires
-la radio-transparence ou radio-densité des plateaux vertébraux

Espace intervertébral normal

Lorsque l'espace inter-vertébral est normal il doit posséder au niveau du rachis thoraco-lombaire les carectères suivants .

-Il est sensiblement égal au précédent et au suivant .En raison de la loi de déformation conique ,l'espace va en diminuant de façon progressive au fur et à mesure que l'on s'éloigne de part et d'autre de l'axe du rayon principal
-Il est de meme largeur en tout point, à bord parallèle
-Il est transparent .L'annulus fibrosus et le nucleus pulposus étant à l'état normal parfaitement transparent aux rayons X
-Il ne présente pas de densité à l'extrémité supérieure ou dorsale .Le plancher du canal vertébral ne recèle aucun éléments radio-opaque .
-Le trou de conjugaison qui surmonte l'espace inter-vertébral doit avoir un diamètre sensiblment égal au trou de conjugaison précédent ou suivant.
-L'espace articulaire inter apophysaires est régulier et égal à celui qui le suit et à celui qui le précède.
- la radio transparence ou radio densité des plateaux vertébraux est identique

En ce qui concerne le rachis cervical il est nécéssaire de faire les remarques suivantes .
On distingue tout d'abord l'espace C2 C3 des autres espaces intervertébraux.
L'espace C2 C3 est un espace à direction ventro-caudale plus prononcée,discrètement triangulaire ,à grande base ventrale .Cet espace est le seul à etre surmonté d'un trou de conjugaison ,de forme triangulaire, radiologiquement visible ,il est donc le seul à posséder une

A partir de C3 C4 l'espace retrouve son parallélisme et le trou de conjugaison n'est plus visible ,il est recouvert par la silhouette de l'apophyse articulaire craniale de la vertèbre immédiatement postérieure .La lecture du cliché cervical est donc un peu plus délicate et la maneuvre que nous avons décrite précédemment prend toute sa valeur.

Espace intervertébral pathologique

Lorsqu' un disque intervertébral est expulsé et que la hernie de ce disque est à l'origine d'une compression ,au moins deux des conditions précédentes ne sont pas respectées.
Il est souvent possible de les retrouver toute ensembles .
Il est important de souligner que l'espace anormal doit être retrouvé sur toutes les radiographies pour que la suspicion se confirme.

Dans le cas d'une hernie discale au niveau cervical ou thoraco-lombaire

- l'espace inter-vertébral pathologique est plus étroit que le précédent ou le suivant
-les bords ne sont plus parallèles ,il apparait une légère triangulation à grande base inférieure ,l'espace est dit "pincé" .Il est toutefois à noter que l'espace T10 T11 est souvent à l'état normal d'aspect un peu triangulaire ,il convient donc d'interpréter en fonction d'autres signes .
-l'espace pathologique peut rester transparent ,mais généralement il présente des éléments denses ,radio-opaques ,qui sont les restes du disque calcifié.
-on peut trouver à l'extrémité supérieure ou dorsale des éléments calcifiés ,fragments du disque inter-vertébral
-le trou de conjugaison qui surmonte l'espace pathologique est d'un diamètre plus petit que celui qui le précède et qui le suit
-l'espace articulaire inter apophysaire est collabé
- la radio densité est augmentée au niveau des plateaux vertébraux adjacents (en raison probablement de l'inflammation.)

Insistons sur le fait qu'un disque calcifié n'est pas forcément à l'origine d'une compression .
Un disque calcifié ,homogène et en place n'est pas responsable d'une compression ,celle ci doit être recherchée en examinant les espaces précédents ou suivants

diagnostic différentiel

Deux phénomènes pathologiques sont susceptibles de donner des images un peu similaires :
les spondylodiscites ou discospondylites
les tumeurs de la zone intervertébrale - rares-

- Les spondylodiscites se traduisent au début par une perte de densité des plateaux vertébraux puis par une image de destruction de ces plateaux, en fin d'évolution par l'apparition d'une spondylose ventrale et d'une densification scléreuse des plateaux vertébraux.
- Les tumeurs de l'espace intervertébral sont extèmement rares et se traduisent par des lésions essentiellement lytiques, similaires à celles des spondylodiscites .

La myélographie : Pourquoi ? Comment ?

Dr Philippe Moreau

La myelographie est une technique d'imagerie qui consiste à radiographier la colonne vertébrale après avoir introduit un matériel de contraste dans l'espace sous arachnoïdien. Cette technique est utilisée fréquemment en médecine vétérinaire pour délimiter précisement les contours de la moelle épinière qui ne sont pas visibles clairement lors des clichés standards.

Il est trés fréquent lors de processus compressifs de la moelle que la localisation de la lésion soit peut claire et incertaine avec des clichés conventionnels sans contraste. Le risque d'erreur de localisation de la lésion est donc grand et ne doit pas être toléré lorsqu'on est amené à une exploration chirurgicale décompressive. Les pathologies compressives de la moelle les plus fréquemment rencontrées chez le chien et le chat comprennent les hernies discales, les sténoses du canal vertébral, les tumeurs (extra et intra médullaires) les hématomes, et les déplacements par luxation ou fracture de la colonne.

Les liquides de contraste qui sont aujourd'hui disponibles, fond que cet examen est devenu une routine car les produits sont moins toxiques qu'auparavant. Les risques d'effets secondaires (convultions, choc, coma et mort) sont donc, fortement diminués et de plus le coût des produits et leur présentation (flacons multi-usage), les rendent d'autant plus attrayants pour un usage fréquent.

Les produits de référence sont aujourd'hui :

1. Le iopamidol et l'iohexol. Ces produits iodés non ioniques ont remplacé le métrizamide (amipaque) considéré comme trop toxique. Nous utilisons le Iopamidol (Iopamiron 300) à la dose de 0,3ml/kg (le produit se présente sous forme de flacon multi emploi) afin de remplir l'espace sous-arachnoïdien de façon suffisante (Iopamiron 300, Laboratoire Scherring, 300mg d'iode/ml flacon de 50ml= 152,00Frs).

Les techniques d'injection sont simples, mais demandent une certaine expérience et une bonne connaissance de l'anatomie de la colonne vertébrale si on veut éviter les accidents et obtenir de bons résultats.

Nous préférons l'injection haute (dans le trou occipital) et n'utilisons les myélographies basses (lombaires), lorsque les contrastes et images obtenues par l'injection haute sont de mauvaise qualité. Si on suspecte une lésion basse lombaire ou même lombo-sacrée, une myélographie haute suivie d'un positionnement immédiat de l'animal en position verticale (ou le plus verticale) permet d'obtenir un écoulement de la colonne de contraste jusqu'au bout de la colonne vertébrale. Il est aujourd'hui très rare que nous ayons recours à une injection lombaire. Certains préfèrent toutefois cette méthode qui est plus difficile au premier abard, mais sans doute moins dangeureuse car une ponction de la moelle lombaire n'engendre pas d'effets secondaire, alors qu'en position haute (trou occipital) le risque existe malgré tout, de toucher des zones critiques.

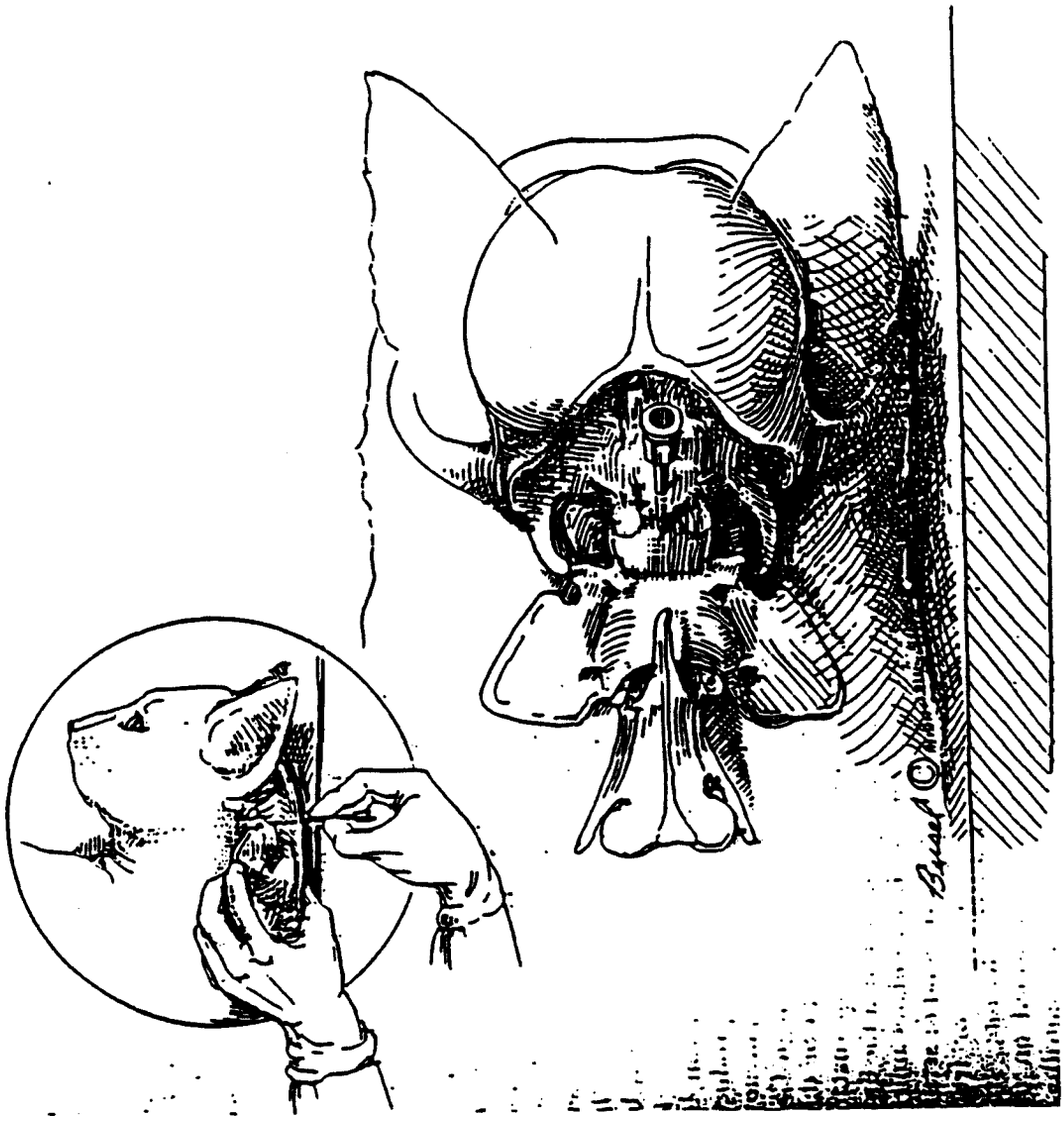
L'examen doit être pratiqué sous anesthésie générale profonde. L'animal est placé en décubitus latéral -quelque soit la technique utilisée (haute ou basse)-. Le site de ponction est préparé chirurgicalement et la

technique de ponction et d'injection sera strictement stérile afin d'éviter toute contamination de l'espace sous-arachnoïdien. Les schémas de ponction sont repris sur les figures de cet article. On utilisera de préférence des aiguilles ou trocard à ponction lombaire, qui ont la particularité d'avoir un mendrin interne.

Il est conseillé de retirer un volume de LCR équivalent (ou légèrement inférieur), au volume de contraste que l'on va injecter. Le LCR collecté peut être gardé pour examen cytologique et biochimique et ne devrait pas être jeter avant que l'on soit sûr qu'il sera inutile pour le diagnostic.

Lors de ponction souillée par du sang ou lorsqu'on n'obtient pas de LCR, il n'est pas conseillé d'injecter et il vaut mieux reponctionner. Toute injection de produit dans la moelle peut avoir des conséquences graves, alors qu'une insertion d'aiguille ne laisse généralement pas de séquelles.

Les soins post-examen sont généralement sans particularité si ce n'est, qu'on veillera à maintenir la tête de l'animal surélevée et on s'assurera que la respiration et le réveil de l'anesthésie sont normaux. Certains utilisent systématiquement le Valium en pré et post-médication, lors de myélographie, afin de prévenir les convulsions éventuelles. Depuis que nous utilisons le Iopamidol, l'incidence des convulsions est devenue quasi-nulle. Nous continuons à prémédiquer les patients au Valium, mais aucun traitement particulier n'est donné après.



EXAMEN DU LCR EN PRATIQUE CANINE : UN EXAMEN DE ROUTINE
QUAND - POURQUOI - COMMENT
TECHNIQUES ET RESULTATS

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Physiologie

Le liquide céphalorachidien est généré principalement par les plexus choroïdes qui filtrent le sang. Ce liquide s'accumule dans les ventricules cérébraux et coule en direction caudale, quitte le système ventriculaire cérébral au niveau du quatrième ventricule et accède à l'espace sous-arachnoïdien. Il baigne ainsi le cerveau et la moelle épinière. Il disparait ensuite dans le sang et la lymphe, étant résorbé par les villosités arachnoïdiennes, les parois des ventricules et les enveloppes des nerfs spinaux. Le contact étroit entre le LCR et le système nerveux implique que de nombreuses modifications pathologiques du système nerveux central modifient sa composition. Par conséquent l'analyse du LCR revêt une importance majeure en neurologie clinique.

Ponction du liquide céphalorachidien

Lors de la ponction de liquide céphalorachidien on a recours à des aiguilles à biseaux courts munis d'un mandrin. Le LCR peut être obtenu par ponction sous-occipitale ou lombaire effectuées chez le patient anesthésié.

Ponction sous-occipitale : l'animal est placé sur le côté droit lorsque le clinicien tient l'aiguille dans la main droite. La peau de la région occipitale est rasée et désinfectés. Un aide place la tête en flexion forcée. L'opérateur identifie l'aile de l'atlas grâce à son index et à la protubérance occipitale avec son pouce. Il imagine ainsi le triangle rejoignant la protubérance occipitale aux bords crânio-latéraux des ailes de l'atlas. Il introduira alors l'aiguille au centre de ce triangle. Il restera attentif à la consistance des différents tissus pénétrés : la peau, les muscles (sans guère de résistance), les méninges (avec éventuellement une résistance élastique). Lorsque l'aiguille pénètre dans l'espace sous-arachnoïdien la résistance des tissus disparaît subitement.

Lors du contact de l'aiguille avec la dure-mère, le patient contracte parfois la musculature de la région occipitale. Ce phénomène n'est pas dangereux et nous indique que la position de l'aiguille est adéquate. On laissera couler le LCR sans l'aspirer. Lorsque du sang est mélangé au LCR (légère hémorragie due à la ponction), on prendra soin d'analyser l'échantillon le plus propre. Ceci implique qu'il faille toujours recueillir plusieurs échantillons séparés lors d'hémorragies car la quantité de sang diminue à mesure que le LCR coule.

Ponction lombaire : le patient est placé sur le côté. On identifie ensuite l'apophyse épineuse de la 6ème vertèbre. L'aiguille est alors introduite devant et parallèlement à cette apophyse jusqu'à ce qu'elle touche le toit de la vertèbre. La pointe de cette aiguille sera alors déplacée vers l'avant jusqu'à ce qu'elle pénètre l'espace intervertébral avant de la retirer petit à petit si le LCR ne coule pas au terme de l'insertion initiale. Le danger d'hémorragie contaminant le LCR est plus grand lors de la ponction sous-occipitale. La quantité de liquide obtenu est souvent bien moindre qu'avec la ponction occipitale.

Analyse du liquide céphalorachidien

Différents paramètres peuvent être évalués lors de l'exemen du LCR y compris :

- la pression intracrânienne
- l'aspect physique
- la culture bactérienne
- la protéinorachie
- l'examen cytologique
- la numération cellulaire
- la glycorrachie
- les enzymes

1. La pression intracrânienne.

Celle-ci peut-être mesurée facilement au moment de la ponction en utilisant un manomètre stérile que l'on adapte à l'aiguille en veillant de ne pas bouger celle-ci. Les résultats obtenus en fonction de la pression intracrânienne, mais aussi de l'anesthésie utilisée. Les variations peuvent-être grandes chez des individus normaux et, il est donc recommandé à chacun d'établir ses normes en fonction de la méthode utilisée.

En plus de la pression, on peut estimer également le rythme de production du LCR, pour cela on mesure la pression et on enlève 1ml de LCR puis on remesure la pression et on détermine le temps nécessaire pour

retrouver la pression initiale. Les normes étant voisines de 0.04 + ou - 0.005 ml/minute.

2. L'aspect physique.

Le LCR normal doit-être clair et sans odeur ("eau de roche"). On peut estimer la clarté de l'échantillon en lisant des caractères d'imprimerie noirs au travers du LCR en graduant de 0 (normal) à 4+ suivant le degré de turbidité observé.

Un échantillon souillé par du sang doit-être interprété avec réserves . Si du sang apparait lors de la ponction il est conseillé de laisser couler quelques gouttes de LCR contaminé avant de recueillir le liquide une fois clarifié. La présence de sang est souvent iatrogène, mais peut-être le signe d'une hémorragie sous-arachnoïdienne récente. Pour distinguer une contamination iatrogène d'une présence de sang pathologique, il suffit de centrifuger l'échantillon. Lors de contamination iatrogène, le surnageant devient clair, lors d'hémorragie pathologique un certain degré de pigmentation plus ou moins jaunâtre (xanthochromie) persiste dans le surnageant.

Un augmentation du contenu cellulaire (> 500 cell/ml) ou de la protéinorachie peut-être aussi responsable de la turbidité du LCR.

3. La culture bactérienne

Lorsque une infection bactérienne est suspectée (LCR trouble, présence de nombreux GB type polynucléés, protéinorachie important), il est indiqué de conserver une quantité suffisante de LCR pour en faire la culture bactérienne et obtenir éventuellement un antibiogramme.

4. La protéinorachie

Le protéinorachie normale chez l'animal de compagnie doit-être inférieure à 0.25 g/l. Les protéines sont essentiellement de l'albumine, molécule apte de par sa taille à passer la barrière céphalo-méningée (BCM). Lors de la lésion de la BCM, certaines molécules plus importantes (globulines) se retrouvent dans le LCR. Lors d'inflammation la perméabilité capillaire peut-être altérée laissant passer ces éléments du sang dans le LCR. Les globulines peuvent refléter également une production locale d'anticorps.

Une estimation grossière de la protéinorachie peut-être obtenue en utilisant les bandelettes urinaires. Une autre méthode particulièrement fiable et attractive consiste à faire une estimation qualitative des protéines totales du LCR par la méthode de Pandy. Cette technique est à la fois simple, rapide et peu couteuse. Elle consiste à faire tomber une à deux gouttes de LCR dans une solution d'acide carboxylique saturé (solution de Pandy) et d'observer

l'apparition d'un nuage blanchâtre plus ou moins intense (graduation de 0 (normal) à 4+) dans la transparence du liquide.

On peut également avoir recours à une mesure quantitative exacte par spectrophotométrie dans n'importe quel laboratoire d'analyse. Une électrophorèse des protéines du LCR peut-être également demandée et permet de distinguer les quantités respectives de l'albumine et des différentes globulines.

5. L'examen cytologique

Celui-ci est important et requiert une analyse quasi-immédiate après prélèvemant (30 minutes) car les cellules dégénèrent rapidement après la ponction. Les cellules du LCR sont classifiées et reconnuespar la nomenclature hématologique et histopathologique classique. Les cellules que l'on peut rencontrer comprennent les lymphocytes, les monocytes, les macrophages (monocytes phagocytaires), les neutrophiles, les éosinophiles, les plasmocytes, et les cellules tumorales. La distinction de ces différentes cellules demande un oeil entrainné et avertti et il est sage de confier cet examen à des cytologistes confirmés (dans chaque hopital).

La population cellulaire normale du LCR comprend essentiellement des lymphcytes et quelques moncytes. Lorsque le nombre de monocytes dépassent celui des lymphos, ce phénomène est compatible avec une irritation du SCN. Les neutrophiles et éosinophiles ne doivent pas etre retrouvés dans un LCR normal et sont le signe le plus souvent d' un processus pathologique.

Les modifications de la composition cytologique du LCR reflètent les changement du SCN et sont d'autant plus significatives que le nombre total de cellules est augmenté. Il faut savoir que le temps est compté (30 à 40 minutes) entre le moment de la ponction et de l'analyse car les cellules sont nombreuses et rapidement lysées dans le LCR receuilli. Les méthodes qui semblent offrir les résultats les plus satisfaisants et qui sont également peu couteuses sont la sédimentation et le filtre millipore. La cytocentrifugation peut également convenir mais ne donne pas nécessairement de meilleurs résultats et demande un équipement trés couteux. L' étalement direct du LCR n'est pas utile, vu le faible nombre de cellules, cette technique n' apporte rien.

6. La numération cellulaire.

Cette étape importante de l'analyse du LCR permet d'établir s'il y a une modification significative qualitative et quantitative des cellules présentes dans le LCR. La numération des érythrocytes (GR) et des cellules nuclées (GB) est rapide et simple et peut-être faite à l'aide d'un hémacytométre (chambre à

numération). On compte le nombre de GR et GB dans les 9 carrés de la chambre. En multi'liant par le nombre de cellules comptées, on obtient le nombre de cellules par millilitre.

Les normes quantitatives des GR et GB du LCR sont de 0 à 8 par mm3 chez le chien et le chat. Une augmentation de la cellularité s' appelle une "pléocytose". Le degré de pléocytose est variable et dépend d'un certain nombre de facteurs y compris la gravité de la maladie, sa cause, et le type de communication de la lésion du SNC avec l' espace sous arachnoidien.

7. La glycorrachie.

le taux de glucose du LCR varie entre 60 et 80% du taux sanguin de l'animal. Il faut 1 à 3 heures avant que des modifications significatives de la glycémie ne se répercute sur la concentration en glucose du LCR, car cette molécule passe lentement la barriére céphalo méningée. Lors de mesure du glucose dans le LCR, il est improtant de faire mesurer la glycémie en même temps. Il est intéressant de savoir que la technique laboratoire de mesure est identique.

une hypoglycorrachie se rencontre dans les méningites bastériennes (infections pyogénes). Cette analyse n'offre que peu d'intéret en pratique courante.

8. Les enzymes.

Certaines lésions ou destructions tissulaires (y compris du tissu nerveux) peuvent introduire une libération de certains enzymes dans le LCR. Les enzymes que l'on peut rencontrer et mesurer dans le LCR comprennent l'alanine aminotransférase (ALT), l'aspartate aminotransférase (AST), la lactate déshydrogénase (LDH), et la phosphokinase (CPK). Ces enzymes ne passent pas la barriére céphaloméningée dans des conditions normales car leur taille est trop importante. On peut mesurer simultanément ces mêmes enzymes dans le sérum et comparer avec les taux obtenus dans le LCR afin d'établir si la lésion est propre au tissu nerveux proprement dit (lésion de structure) ou s'il s'agit plutot d'une lèsion de la barriere oèphlo-méningée (lésion fonctionnelle).

chez l'homme l'interprétation des taux enzymatiques dans le LCR est plus "affinée" dans la; mesure ou on peut faire certaines corrélations plus précises (exemple: augmentation des CPK: maladie démyélinisante). De plus, la mesure d'un isoenzyme CPK1 spécifique de la CPK montre une bonne corrélation avec le degré lésionnel du SNC et semble être un bon paramètre

pronostic chez l'homme. Ces types d'interprtétation de l'enzymologie du LCR restent encore à être démontrés chez l'animal.

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CLASIFICACIÓN DE LOS TUMORES NERVIOSOS DEL PERRO Y DEL GATO

La clasificación histológica de los tumores del tejido nervioso de los animales domésticos se basa en la realizada por la WHO (Fankhauser et al.) en 1974, y en trabajos posteriores, como el de Braund (1987), o el de Cordy (1990).

Sin embargo, para una clasificación más precisa recurriremos a la clasificación de Russell-Rubinstein (1989) para la especie humana.

Para establecer la clasificación de los tumores del tejido nervioso procederemos a hacerlo desde diferentes puntos de vista.

* CLASIFICACIÓN HISTOLÓGICA, los divide en:
- **Primarios**, aquellos tumores que se originan a partir de células neuroectodérmicas, ecto y mesodérmicas, presentes normalmente en el tejido nervioso o asociadas al mismo. Si la célula neoplásica pertenece al tejido nervioso o no nos determinará tumores:
    - **Parenquimatosos**: Glioblastomas, Astrocitomas, Meduloblastomas
    - **Extraparenquimatosos**: Meningiomas, Adenoma de hipófisis, Schwannomas

- **Secundarios**, aquellos tumores que se originan a partir de las estructuras circundantes (hueso, músculo) al tejido nervioso, o llegadas al mismo por via hematógena desde órganos lejanos.

* CLASIFICACIÓN TOPOGRÁFICA, según la localización del tumor: Intracraneales, Intraespinales, del Sistema Periférico, etc..

A continuación realizamos una clasificación de los tumores dels sistema nervioso más corrientes en perro y gato:

1. Tumores de neuronas
- Ganglioneuromas
- Neuroblastomas
    - N. Cerebelar (Meduloblastoma)
    - N. Periférico

2. Tumores de células gliales
- Astrocitomas
- Oligodendrogliomas
- Gliomas mixtos o indiferenciados
- Ependimomas
- Papiloma de los plexos coroideos

3. Tumores de células mesenquimatosas
- Meningiomas
- Tumores vasculares
- Linfoma histiocítico (Reticulosis neloplásica)

4. Tumores congénitos

Craniofaringioma
Teratomas o teratocarcinomas
Quistes epidermoides
Cordomas

5. Tumores del SNP

Schwannomas
Neurofibromas
Neuromas
Tumor de células granulares
Paragangliomas

6. Metástasis tumorales

Carcinomas (mama, pulmón, próstata, gl. salivar)
Sarcomas de tejidos vecinos (Osteosarcoma, Condrosarcoma, Hemangiosarcoma, Fibrosarcoma)
Linfomas

Su incidencia varía según la especie raza y edad por lo que en general podemos afirmar que:

PERRO

Entre los tumores intracraneales predominan :

- Meningiomas en razas dolicocéfalas (Pastor alemán, Collie)
- Gliomas en razas braquicéfalas (Boxer, Bulldog inglés, Boston terrier)

En la médula espinal predominan los tumores óseos malignos (Osteosarcoma, Fibrosarcoma, y Hemangiosarcoma) primarios y extradurales, o sus metástasis. También puede aparecer el Neuroepitelioma, intradural-extramedular, en Pastor alemán, localizado en general a nivel T10-L1.

GATO

Los tumores intracraneales más frecuentes son los Meningiomas (gatos machos castrados y mayores de 10 años), únicos o múltiples, localizados en la tela coroidea del tercer ventrículo y en las meninges supratentoriales.
Tanto en el encéfalo como en la médula destaca el Linfoma tanto extradural como parenquimatoso, primario o secundario.

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NEUROONCOLOGÍA DEL PERRO Y DEL GATO

1. INCIDENCIA Y FACTORES DE RIESGO

La incidencia de tumores en el Sistema Nervioso Central (SNC) es baja (1-3% del total de los tumores en el perro), seguramente debido a la escasez de trabajos realizados en este sentido. Su conocimiento actual se fundamenta especialmente en los estudios llevados a cabo en la especie humana; su clasificación también ha seguido el mismo modelo.

Según su localización los podemos dividir en :

TUMORES INTRACRANEALES:
- Supratentoriales
- Infratentoriales
- Intermedios

TUMORES INTRAESPINALES, independientemente del nivel de la médula espinal afectada pueden ser:
- Extradurales (epidurales)
- Intradurales (subdurales)
  - . Extramedulares
  - . Intramedulares

TUMORES DEL SISTEMA PERIFÉRICO
- Nervios periféricos
- Sistema Autónomo y Paraganglionar

1.1 TUMORES INTRACRANEALES

Algunos autores, como Vandevelde, sugieren para los tumores intracraneales una incidencia de 14,5 por 100.000 perros y de 3,5 por 100.000 gatos (9,2 por 100.000 en la especie humana).

Por edad se aprecia una incidencia más alta a partir de los 5 años (de 6 a 11 años), sin embargo pueden presentarse en perros muy jóvenes (Meduloblastoma o los tumores ventriculares).

Por razas se observa que los tumores de células gliales y de hipófisis son más frecuentes en perros de razas braquicéfalas, mientras que los Meningiomas ocurren con mayor frecuencia en las dolicocéfalas. La Reticulosis o el Limfoma histiocítico se presentan con mayor frecuencia en Terriers, Caniches y otras razas de pequeño tamaño.

El gato aparentemente resulta menos afectado que el perro. El Meningioma es el tumor más frecuente en gatos adultos, siendo en muchos casos de presentación múltiple. El resto de tumores son raros en el gato si exceptuamos el Linfoma.

HOMBRE: presentación bimodal, en niños (Astrocitoma cerebelar, Ependimoma ventricular) y en adultos a partir de 50 años (Gliomas representan el 25%).

1.2 TUMORES INTRAESPINALES

En el perro, los tumores extradurales representan el 50%, los intradurales extramedulares el 35% y los intramedulares el 15% del total de tumores espinales.

Por edades, en perros adultos el más frecuente es el Meningioma, sobretodo en el área cervical; en perros jóvenes (6 meses a 3 años) se observa el Neuroepitelioma.

En el gato el Linfoma es el tumor observado con mayor frecuencia, muchas veces relacionado con la infección por FeLV o con FIV.

1.3 TUMORES DEL SISTEMA PERIFÉRICO

En el perro, la incidencia más alta corresponde al Schwannoma, tanto afectando a los nervios raquídeos como a los pares craneales. La Neurofibromatosis también ha sido descrita en el perro.

En el gato los Schwannomas son raros, mientras que la Neurofibromatosis no ha sido aún descrita. Sin embargo se observan con frecuencia metástasis en nervios periféricos de gatos con Linfomas.

2. PATOLOGÍA TUMORAL

Señalaremos aquí los cambios estructurales derivados de los tumores y de su presencia en el tejido nervioso. Podemos agruparlos en:

- Cambios focales:
1. **Cambios en la masa tumoral**, el aumento de tamaño del tumor suele ser lento y progresivo. Sin embargo la cavitación, con formación de quistes, o la presencia de hemorragias en el tumor pueden provocar cambios bruscos en su tamaño.
2. **Cambios en el parénquima adyacente**, debidos a compresión, con desplazamiento del parénquima adyacente, o infiltracion y destrucción del mismo.

- Cambios regionales:
1. **Edema cerebral**, por alteración de la vascularización.
2. **Cambios en la cirulación del LCR**: hidrocéfalo.
3. **Hernias cerebrales o cerebelares**, según la localización del tumor podemos observar:
   * Tumores supratentoriales:
     - Hernia cerebral subfalcial (*falx cerebri*)
     - Hernia cerebral central
     - Hernia cerebral externa
   * Tumores infratentoriales
     - Hernia cerebelar superior (*tentorium cerebelli*)
     - Hernia cerebelar tonsilar (*foramen magnum*)

3. CUADRO CLÍNICO Y EVOLUCIÓN

La sintomatología asociada a los tumores del sistema nervioso dependerá de su localización, de los centros y vías nerviosos afectados por su presencia así como de los efectos secundarios (aumento de la presión intracraneal) que genera el tumor. Como signos clínicos generales podemos destacar:

* T. **Intracraneales**: Convulsiones, cambios de conducta, marcha en círculo, apoyo o presión de la cabeza contra un obstáculo, cambios en el estado mental, alteraciones motoras asociadas, signos de pares craneales, etc.
* T. **intraespinales**: signos de compresión medular progresiva, problemas vasculares como isquemia, necrosis y hemorragia sobretodo en t. intramedulares, signos de mielopatía transversa (lesion de la médula espinal craneal y caudalmente al punto afectado).
* T. **en nervios periféricos**: déficits nerviosos progresivos, signos de NMI, complicaciones medulares, S. de Horner, etc.

Los criterios de **benignidad** y **malignidad** son muy relativos a la hora de hablar de tumores del sistema nervioso. Tal vez resulta más interesante hablar de conceptos como rapidez de crecimiento, o grado de invasividad, a la hora de valorarlos y establecer un pronóstico adecuado.

Las **recidivas** son raras en tumores benignos y se realacionan con extirpaciones quirúrgicas incompletas (Schwannomas). Los tumores malignos, sin embargo, tienen mayor tendencia a las recidivas.

Los tumores nerviosos primarios **metastatizan** de forma excepcional, y si lo hacen utilizan la via hematógena. También pueden colonizar por continuidad tejidos extraños que se encuentren adyacentes a ellos. En algunos casos el LCR (Líquido cefaloraquídeo) puede ser también una via de difusión sobre todo para los tumores localizados cerca de los ventrículos o en los plexos coroideos (Meduloblastomas, Germinomas).

4. TÉCNICAS DE DIAGNÓSTICO

El examen físico y el neurológico nos permiten dirigir el diagnóstico hacia la posible presencia de un tumor. Será necesario disponer además de un hemograma completo, un perfil bioquímico y el análisis de orina.

Examen radiológico, aporta poca información ya que los tumores nerviosos no son en general observables mediante el uso de rayos X. Podemos observar lesiones relacionadas con ellos como una osteolisis, una hiperóstosis, o incluso calcificaciones dentro del tumor; también nos permitirá identificar tumores no nerviosos que desde órganos vecinos invaden al sistema nervioso.

El **estudio del LCR** no resulta de carácter diagnóstico pero puede aportar información muy valiosa. En general, la presencia de tumores en el parénquima nervioso se asocia a aumento de la presión del LCR, aumento de su contenido de proteínas y del número de células de la línea blanca (>50 células/μl).

Mielografía, permite la identificación y clasificación de los tumores de la médula espinal siendo de gran importancia para detrminar la pauta terapéutica a seguir.

Técnicas de diagnóstico mediante el **uso de contrastes**: Ventrículografia, Angiografía cerebral, Venografía de los senos cavernosos, Cisternografía basal, etc. pueden ser útiles para demostrar la presencia de lesiones que ocupan espacio, así como sus límites y su relación con los tejidos adyacentes.

Tomografía Axial Computerizada (TAC) y **Resonancia Magnética** (RMN) permiten visualizar la presencia de los tumores, su forma, tamaño dimensiones, grado de infiltración, y, en muchos casos, su identificación. Podemos identificar masas de hasta 0,5cm de diámetro. El uso de contraste iodado intravenoso permite, en la especei humana, descubrir hasta un 90% de los tumores nerviosos. Estas técnicas resultan fundamentales para la cirugía.

Electromiografía (EMG), muy importante para el diagnóstico de los tumores de nervios periféricos.

La **biopsia** resulta diagnóstica a la hora de identificar el tumor y establecer una pauta terapéutica.

5. TRATAMIENTO

El objetivo principal será el de reducir el tamaño del tumor o, si es posible, erradicarlo. Para ello podemos aplicar diferentes tipos de tratamiento:

* **Farmacológico**:
  - Córticoesteroides (Dexametasona en fase aguda, Prednisona, Prednisolona para mantenimiento, etc), para reducir el edema peritumoral y el crecimiento del tumor
  - Anticonvulsivantes: Phenobarbital
  - Reguladores de la homeostasis

* **Quirúrgico**: dependerá de la localización del tumor y de su invasividad. Puede ser útil para la extracción de biopsias y su estudio. Trataremos de reducir el tamaño del tumor, para poder continuar con otro tipo de tratamiento, o su total eliminación.
* **Radioterapia**, indicado para todo tipo de tumores, tanto primarios como secundarios obteniéndose buenos resultados.
* **Quimioterapia**, como tratamiento paliativo y en experimentación. Se aplican compuestos como BCNU, CCNU (nitrosourea) y Procarbazina (inhibidor de la monoaminoxidasa) para los tumores intracraneales. Para los Schwannomas se recomienda una combinación de Vincristina, Doxorubicina y Ciclofosfamida.
Los linfomas seguirán la misma pauta de tratamiento que en los casos sistémicos.
* **Inmunoterapia**, para modificar la respuesta inmune del paciente y poder eliminar el tumor inmunológicamente. En perros se han realizado estudios experimentales mediante cultivo de linfocitos autólogos incrementado su número y efectividad citotóxica.

6. PRONÓSTICO

La supervivencia de los perros y gatos a los tumores nerviosos se halla muy discutida y depende de muy diversoos factores. Las cifras pueden variar desde 10 a 307 dias para los tumores intracraneales.

Los primeros estudios evidencian que la radioterapia, sola o combinada con otros tipos de tratamiento, asegura una supervivencia más larga, observándose una disminución del tamaño del tumor, así como su mejor delimitación. Si se practica la eliminación quirúrgica del tumor, lo más completa posible, y una radioterapia posterior, la supervivencia se alarga (hasta 571 dias).

En el caso de los tumores de nervios periféricos si la excisión es radical el pronóstico resulta favorable.

7. COMPLICACIONES NEUROLÓGICAS DE LAS NEOPLASIAS SISTÉMICAS

En la especie humana hasta un 20% de las neoplasias sistémicas desarrollan síntomas neurológicos. Estas complicaciones también han sido descritas en el perro y en el gato si bien no se dispone de estudios completos sobre este apartado.

Estas complicaciones las podemos agrupar en:
- **Metástasis al sistema nervioso**, en la especie humana se describen que hasta un 25% de los tumores provocan metástasis intracraneales. La médula espinal, las leptomeninges y los nervios son el segundo lugar en importancia (5-10%).

Aparte de las metástasis nos podemos encontrar con otro tipo de complicaciones, en general reversibles, pero que pueden comprometer la vida del animal:
- **Encefalopatía metabólica**, cursa con cambio de conducta. Es debida a un fallo del metabolismo encefálico por cambios en el equilibrio electrolítico general, el metabolismo hepático, renal, una hipoxia, una sepsis, etc.
- **Infecciones** debidas a la alteración de los mecanismos inmunes o a la neutropenia que genera la quimioterapia, los tumores de médula ósea o de los vasos.
- **Problemas cerebrovasculares** debidos a trombocitopenia o a déficits en los mecanismos de coagulación.
- **Efectos adversos derivados del tratamiento**, por el uso de fármacos neurotóxicos (Fluouracilo, Methotrexato, Cisplatina, alcaloides de la vinca) que actúan directamente sobre el sistema nervioso o indirectamente, sobre otros órganos.
- **Síndrome paraneoplásico**, incluye mecanismos poco claros que repercuten sobre el sistema nervioso: reacciones autoinmunes, infecciones víricas, toxinas tumorales, desnutrición, etc. Se reconocen efectos paraneoplásicos como la Polineuropatía sensitivo-motora, la Myasthenia gravis associada a timoma y la Polimiositis.