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Spinal cord disorders

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Mechanisms of disease

The localization of specific functions in the nervous system has important effects on the clinical presentation and nature of progression spinal cord disorders. Localization of function in the spinal cord causes a similar pathologic process to result in many different clinical presentations, depending on what part(s) of the spinal cord it affects. For example, a spinal cord mass located at the level of C3 vertebra may result in tetraparesis, whereas the identical mass 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 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 probably 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 the spinal cord to disease are to a degree nonspecific so that with the exception of neoplasia and congenital malformations various disorders may induce a somewhat similar histologic appearance. 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. 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 are not misdiagnosed and consequently treated inappropriately.

Diagnostic techniques

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.

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. For example, neoplastic diseases

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affecting the spinal cord 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 so many 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 if vascular factors such as infarction or hemorrhage are involved.

Physical examination

Results of this examination are used to supplement information collected in the history and may implicate involvement of body systems other than the nervous system. For example, an animal presented with a primary complaint of spinal 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. This is essential, as often there is a tendency for a clinician to focus a work-up on an obvious neurologic deficit while ignoring a related problem. A differential diagnosis list should be compiled of possible causes for each problem included in the problem list.

Minimum data base

Initial clinicopathologic tests include a complete blood count, blood chemistry profile, and urinalysis. 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 obtained 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

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 include electrophysiologic testing or advanced imaging such as computed tomography (CT) or magnetic resonance imaging (MRI).

Noncontrast vertebral radiography

Noncontrast vertebral radiography with the cat anesthetized is essential in the accurate diagnosis of a disorder affecting the spinal cord. Due to the

limitations of a neurologic examination in defining multiple lesions of the spinal cord, and for the purpose of comparison in the future, the entire vertebral column should be radiographed whenever 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.

Further diagnostic investigations may be indicated on the basis of results of noncontrast vertebral radiography. 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.

CSF analysis

CSF collection and analysis are essential in cases in which 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. However, a lumbar collection site (most often between L5 and L6 vertebrae) may be used for most 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 in which an increased intracranial pressure is suspected.

Analysis of CSF collected from a 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 and cats have been published. It should be noted that normal values differ in CSF collected from lumbar and cisternal collection sites.

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. Recently, techniques for CSF protein electrophoresis and for determination of the IgG index have been published.

Myelography

Contrast radiography should be done when results of noncontrast vertebral radiography and CSF analysis do not fully define the spinal cord disorder. 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.

Myelography is difficult to perform and 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 a precise 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 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 the extent of a spinal cord lesion. This technique is ideally done prior to completion of noncontrast vertebral radiographs as results may help to determine the exact 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 is often normal in association with disorders that primarily affect the spinal cord white matter. Furthermore, specific information regarding the cause of a myelopathy is not available by means of EMG examination.

EMG 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 measured in 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.

CT and MRI

Both these advanced imaging modalities should be considered adjunctive procedures to myelography to further delineate the extent of a lesion. Both these examinations provide for a more detailed examination of the spinal cord and surrounding tissue. CT or MRI imaging is useful for diagnosing foraminal disc extrusions as in these cases routine myelography may be normal, and in the identification of spinal cord tumors.

Clinical signs of spinal cord disorders

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.

Five groups of clinical signs 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 facilitates 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 brain stem 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 and urinary and anal sphincters (L7–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.

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–C5 spinal cord segments), cervical enlargement (C6–T2), thoracolumbar (T3–L3), and lumbar enlargement (L4–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 and lumbar enlargements.

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–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.

UMNs 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. UMN 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 asymmetric. In the majority of cases, a lesion resulting in asymmetric signs is located on the side of greater motor and sensory deficit.

Cervical (C1–C5)

Fatal respiratory paralysis resulting from interruption of descending respiratory motor pathways or damage to motor neurons of the phrenic nerve (C5–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 are usually 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 vol-

untary control of micturition. Reflex dyssynergia may also be seen. Although voluntary control of defecation may be lost, reflex defecation occurs 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 are usually depressed or absent in all limbs. Complete loss of conscious proprioception may occur 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–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 may be accomplished by doing a complete 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–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 brain stem (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–T2)

Ataxia and paresis of all four limbs are usually 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 is often 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).

If bladder dysfunction occurs, 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 is commonly observed with a spinal cord lesion of the cervical enlargement, particularly a lesion involving T1–T3 spinal cord segments or nerve roots.

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

Severe depression or loss of pain perception rarely is 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–L3)

The majority of spinal cord lesions of cats occur 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 are usually normal or exaggerated. Voluntary control of defecation may be lost. Reflex defecation occurs when the rectum is filled with feces; however, it 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 in which initiation of voiding occurs and is stopped by involuntary contraction of the urethral sphincter. The bladder can be manually expressed in some animals, but not in others due to increased tone of the urinary bladder sphincter. This is often referred to as a '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 equine ('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–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 by 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 is often 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 equine 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–Cd5).

Spinal cord diseases

Inflammatory diseases

Inflammatory diseases that affect the brain and spinal cord are among the most common neurological disorders of cats. Common causative agents are viral, protozoal, or fungal.

Feline infectious peritonitis

FIP is caused by an aberrant immune response to a mutant form of the feline enteric coronavirus, the FIP virus (FIPV). The disease has been divided into effusive (wet) and parenchymatous (dry) forms and neurological manifestations of FIP are seen mostly in cats with the dry form. Clinical disease is the result of immune-mediated host responses to macrophages infected with FIPV, and the severity of disease appears to be determined primarily by host susceptibility and immune responses, and secondarily by virus strain.

Changes in the nervous system of affected cats include pyogranulomatous inflammatory cell infiltrate in the meninges, choroid plexus, and superficial neuropil of the brain and spinal cord, and immune-mediated vasculitis resulting in vasogenic brain edema, hemorrhage, and thrombosis. Lesions may occur in any area of the central nervous system (CNS), but are particularly pronounced in the brain stem and spinal cord. Brain edema and hemorrhage may lead to herniation. Obstruction of CSF outflow within the ventricular system or through the arachnoid villa may lead to hydrocephalus.

The CNS form of FIP may occur at any age, but cats less than 3 years of age are more commonly affected. Clinical signs include seizures, nystagmus, head tilt, ataxia, paresis, and proprio-

ceptive loss. Neurologic deficits are invariably progressive. There is often ophthalmic involvement, with anterior uveitis a common finding. Most cats also have systemic signs of FIP.

The neurologic form of FIP can be difficult to diagnose antemortem. Typical results of CSF analysis are high levels of protein and a moderate to marked pleocytosis, with either lymphocytes or neutrophils predominating. These changes are not specific for FIP and should be interpreted with the history, clinical signs, and additional tests. Unfortunately serological assays including virus neutralization, enzyme-linked immunosorbent assay (ELISA), indirect immunofluorescence, Western blotting, and immunoperoxidase antibody assay are not specific for FIP. These assays detect group-specific antibody, indicating only exposure to feline coronavirus, including the relatively benign feline enteric coronavirus. Although higher antibody titers ($\geq$1:3200) are reported to occur with the noneffusive form of the disease, this is not always the case. A diagnosis of FIP also may be made by the finding of characteristic microscopic lesions in biopsy samples of abdominal organs.

The most accurate antemortem diagnostic tests appear to be a positive anti-coronavirus IgG titer in CSF, high serum total protein concentration and MRI findings suggesting periventricular contrast enhancement, ventricular dilatation, and hydrocephalus. Treatment consists of supportive care only.

Toxoplasmosis

Toxoplasmosis is caused by the protozoal organism *Toxoplasma gondii*, an obligate intracellular coccidian parasite found worldwide. Cats are the only known definitive host for the organism and cats can be infected transplacentally (rare) or by ingesting any of the three lifestages of the organism. The seroprevalence of infection varies by region, but is approximately 30% in cats in the USA. Despite this high seroprevalence, clinical disease caused by *T gondii* is rare. *T gondii* infection may cause a nonsuppurative meningoencephalitis and/or focal or disseminated myelopathy in cats. The infective organism is spread hematogenously to most organs of the body, including the CNS. Although the incidence of disease associated with *T gondii* is low; opportunistic infection in immunosuppressed animals may be more widespread than previously reported. Concurrent infection with FeLV, FIV or administration of corticosteroids may predispose

to the development of clinical signs. Toxoplasma cysts with no associated reaction are seen in the brains of some cats without neurologic deficits. In clinically affected cats, lesions are most common in lungs, liver, gastrointestinal tract, and brain. Nonsuppurative meningoencephalitis is seen histologically.

Common CNS clinical signs include seizures, ataxia, behavioral changes, and paralysis. Animals with CNS toxoplasmosis may, or may not have other clinical signs indicative of systemic infection (fever, lymphadenopathy, pneumonia, apparent muscle pain, gastrointestinal tract disease, uveitis iritis, or chorioretinitis).

Antemortem diagnosis of CNS toxoplasmosis in cats is difficult, and may be tentatively based on all of the following: (1) serological evidence of infection, (2) clinical signs of disease referable to toxoplasmosis, (3) exclusion of other common causes of these clinical signs, and (4) positive response to appropriate treatment. Although results of routine hematologic and biochemical tests may be abnormal in cats with acute systemic toxoplasmosis, such results reflect only the organ systems involved and are not specific for toxoplasmosis. CSF may be normal, or may have an elevated white blood cell count with a mixed mononuclear pleocytosis, and an elevated protein concentration. Radiography or ultrasound of the thorax or abdomen of cats with acute disease may demonstrate effusion, pneumonia, or abdominal masses. *T gondii* organisms may be identified in cytologic preparations of thoracic or peritoneal effusions, or in biopsies of lymph node or muscle examined by conventional histopathologic techniques or by other methods such as immunoperoxidase staining. However, demonstration of *T gondii* organisms does not confirm toxoplasmosis as the cause of a clinical disease.

The presence of a positive IgG antibody titer in a single serum sample indicates only exposure to *T gondii*, not recent or active disease. The demonstration of an increasing IgG titer in paired serum samples collected 2–3 weeks apart may indicate recent or active disease, however, this is seldom possible, as most animals with clinical toxoplasmosis have chronic low-grade clinical signs and are not evaluated serologically until their IgG antibody titers have reached their maximum values.

T gondii-specific IgM is detectable in serum by ELISA in approximately 80% of subclinically ill cats within 2–4 weeks following experimental induction of toxoplasmosis. Generally these titers are negative within 16 weeks after infection.

Serum IgM titers >1:256 are commonly a marker of recent or active infection.

Currently it is recommended that for serologic diagnosis of toxoplasmosis in cats a single serum sample should be submitted for *T gondii*-specific IgG and IgM determinations, and for calculation of levels of circulating antigen to *T gondii*. In cats suspected of having toxoplasmosis, both FeLV and FIV titers should be determined. Polymerase chain reaction (PCR) to detect *T gondii* DNA in biological samples such as aqueous humor from infected cats has been found to correlate with clinical disease in some, but not all cats. PCR may provide a more sensitive tool for antemortem diagnosis of toxoplasmosis in the future.

Fecal examination for oocysts is the most practical method for determining the public health risk of a cat suspected to have toxoplasmosis. Oocysts are shed in feces of infected cats for only a short time (5 days to 2 weeks postinfection).

Supportive care is provided as needed. Available drugs are effective in CNS tissues only against actively proliferating forms of the organism, and are not active against encysted forms. Clinical signs of ocular and CNS toxoplasmosis respond more slowly to therapy. Clindamycin can be given orally at a daily dose of 25 mg/kg divided q12 or q8 h for 4 weeks. If vomiting occurs in the first few days the drug should be discontinued for 24 h and then started at a lower dosage, ultimately increasing the dose to 25 mg/kg. The effectiveness of clindamycin hydrochloride in penetrating CNS tissues of cats has not been determined, although clinical success in a limited number of cases has been reported. An alternative to clindamycin is sulfadiazine or triple-sulfas given orally at a daily dosage of 22 mg/kg divided ql2 h. Addition of pyrimethamine at a daily dosage of 0.11– 0.22 mg/kg permits reduction of the sulfadiazine dosage by half. Hematologic monitoring for bone marrow suppression is essential for cats placed on this therapeutic regimen.

Intervertebral disc disease

Acute disc extrusion has been reported in cats. Although disc extrusion resulting in clinical signs of a myelopathy in cats is an infrequent problem, it does occur and therefore must be considered as a differential diagnosis in cats presenting with a myelopathy. Surgical removal of extruded disc material in healthy cats appears to result in good neurologic and functional recovery. Even cats in which deep pain perception was (apparently)

absent on initial examination, if diagnosed and treated promptly may have a very good recovery.

Spinal cord trauma

Spinal cord injuries in cats result most frequently from direct physical trauma. Following injury, the spinal cord may undergo sustained compression, distraction, or both. The severity of a spinal cord injury, as determined by the eventual quality of recovery, is related to three factors: the velocity with which the compressive force is applied, the degree of compression (transverse deformation), and the duration of the compression.

Cats with spinal cord injury frequently have serious injuries of other organ systems. At the time of presentation, the animal should be placed in lateral recumbency and should remain in that position during subsequent clinical and radiographic examinations. A thorough assessment of the animal's general condition must be made, looking for major problems such as hemorrhage, shock, airway obstruction, or limb fractures.

A neurologic examination is performed to localize the site of the injury and determine its severity. Careful palpation of the vertebral column may aid in identification of a vertebral fracture or luxation. Administration of tranquilizers or analgesic should be delayed until completion of the neurologic examination, as these agents may alter a cat's responses. A neurologic examination should be done with minimal movement of the animal in order to prevent further injury resulting from vertebral instability. The extent of a spinal cord injury usually can be assessed accurately at the time of initial examination, as most spinal injuries are nonprogressive, and as spinal shock (a phenomenon resulting in loss of physiologic functions caudal to a spinal cord injury) is not of clinical significance in subprimates.

Several aspects of the neurologic examination are of special importance in assessing a cat with a spinal injury. Attention given to the animal's posture may aid in determination of location and severity of a lesion. For example, Schiff–Sherrington syndrome, which results from thoracolumbar spinal cord injury, is of great localizing value and must be differentiated from other postures such as decerebrate rigidity and decerebellate rigidity. Motor function, muscle tone, and spinal reflexes must be assessed carefully. These functions are utilized to localize a lesion to one of four major regions of the spinal cord: cervical (C1–C5), brachial enlargement (C6–T2), thoracolumbar (T3–L3), and lumbar enlargement (L4–Cd5).

Pain perception should be assessed by applying a painful stimulus and observing the animal for a brain-mediated response. The stimulus applied to a foot may result in withdrawal of the limb by a spinal reflex mechanism (the flexion reflex), even though the spinal cord may have been severed. It is essential to distinguish these spinal reflex movements from brain-mediated responses. Two types of pain perception are sometimes distinguished in cats. 'Superficial' pain perception is manifested by a response to pricking or pinching the skin, and 'deep' pain perception is manifested by response to pinching the toes or tail with hemostatic forceps. When multiple spinal fractures occur, the clinical signs of a more caudal lesion may mask those resulting from a second lesion located further cranially. For example, the LMN signs caused by a lesion at L5–L6 may mask the hyperreflexia that would be caused by a second injury at L1.

Diagnosis Results of the neurologic examination are usually extremely revealing in determining the site and severity of spinal cord injury subsequent to trauma. Radiographs of the vertebral column are essential. Ventrodorsal radiographs are best done by means of a horizontal beam, in order to avoid further spinal injury. The entire vertebral column must be radiographed, and proper positioning is required. A logical approach is to anesthetize the animal as soon as possible after completion of the physical and neurologic examinations. This will, of course, be influenced by the severity of non-neural injuries in many cases. Anesthesia permits precise positioning and allows for the completion of CSF analysis and myelography. The major values of radiographic studies are in precise lesion localization, outlining unsuspected lesions, and in assessing the need for surgery and the procedure to be used.

The goal of management of injury of the spinal cord is to reduce the resultant neurologic deficit and prevent any additional loss of neurologic function. In a patient with a suspected bony injury of the spine, the spine must be immobilized to prevent neural injury and the anatomy of the injury must be analyzed as a first step in providing effective treatment.

Many cats with spinal trauma have resultant vertebral fractures or luxations that are evident radiographically. These fractures or luxations often occur adjacent to areas of rigid stability such

as the sacroiliac articulation. Fractures usually occur transversely from the intervertebral foramen cranioventrally through the vertebral body or through the vertebral epiphysis in younger animals. Alternatively, the vertebral body may be compressed (compression fracture). Luxations occur through the intervertebral disk space, with the caudal segment or vertebra usually being displaced cranioventrally. Other animals with spinal trauma and significant neurologic deficits have no radiographic evidence of vertebral fractures or luxations. In some of these cases, vertebral displacement may have occurred at the time of trauma, with immediate return to normal alignment. More commonly, perhaps, these cats have spinal cord contusion analogous to that occurring in the brain as a result of trauma. The associated intramedullary hemorrhage and edema may cause myelographic evidence of spinal cord swelling at the site of injury.

Medical therapy The aim of medical therapy is to control the chemical and vascular changes that result in secondary injury. Numerous drugs have been described for the treatment of spinal cord injury, however, the efficacy of these drugs remains controversial. Despite extensive studies the role of dexamethasone in the treatment of traumatic spinal cord injuries remains poorly defined. Due to the frequency of adverse side effects and uncertainty about its efficacy, the use of dexamethasone for treating acute spinal cord injury is not recommended.

Methylprednisolone (MP) has been studied intensely in both the research and clinical setting for the past 10 years. The drug must be administered as soon as possible after injury and the effective dose range and time after injury when the drug may be given are very narrow. While some experimental and clinical studies have found an improved recovery in research animals and people treated with MP following spinal cord injury, a number of other studies have not found any significant improvement. In people the actual functional differences are relatively small and benefit upper body function as opposed to actual locomotion.

Surgical therapy A decision regarding surgical treatment must be made as soon as non-neural injuries have been treated and medical management instituted. Ideally, this is within 2 h of the time of injury. Surgical decompression by laminectomy is beneficial when there is myelographic evidence of sustained extradural compression of the spinal cord. In cases where extradural compression is not present and spinal cord swelling is the major source of compression, durotomy or myelotomy may be combined with laminectomy.

Alignment and stabilization of the vertebral column is indicated in animals that demonstrate severe displacement or instability of a fracture or luxation. Most fractures or luxations must be considered unstable, even though all such injuries will not result in sustained spinal cord compression or distraction. Surgical management of spinal cord injury in animals must be considered in all cases, as it provides the best opportunity for rapid and complete recovery in cases of sustained compression or instability and facilitates post-injury care. However, conservative therapy, including strict confinement for 4–6 weeks, may be efficacious in animals with minimal neurologic deficits and little vertebral displacement.

Numerous surgical procedures have been utilized in stabilizing the spine in cats. The use of Steinmann pins and polymethylmethacrylate provides a method that is applicable to all levels of the vertebral column.

Regardless of the form of internal stabilization used for vertebral fractures, the stabilizing device may fail if there is excessive motion during the initial postoperative phase. For this reason, strict confinement is recommended for 2 weeks after surgery.

Complications Potential complications encountered with spinal cord trauma and associated paralysis include urinary tract infections, urine scalding, and decubitus.

Prognosis Prognosis for an animal with spinal cord injury depends on numerous factors, with results of the neurologic examination being the major determinant. The most important factor in neurologic prognosis is the presence or absence of pain perception. The loss of 'superficial' pain perception occurs with less severe lesions than those resulting in loss of 'deep' pain perception. In general, animals in which any degree of pain perception is detected with certainty can be expected to be restored to locomotion. The more severe the depression of pain perception, the longer the recovery period. In severe injuries, where pain perception has been absent for a brief period, recovery may require months and residual ataxia and paresis may persist. Most animals in which pain perception has been absent for 24 h or more cannot be restored to locomotion, although occasional recoveries have been seen in such cases.

Further reading

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