Journal of Feline Medicine and Surgery (2003) **5**, 101–108
doi:10.1016/S1098-612X(02)00124-9

jfms

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PROCEEDINGS OF THE ESFM FELINE CONGRESS, STOCKHOLM, SEPTEMBER 2002
Feline vestibular diseases—new developments

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Disorders of the vestibular system occur frequently in cats. A basic understanding of the anatomy and physiology of the vestibular system is necessary for interpretation of the clinical signs and precise lesion localization. Recognition and interpretation of the clinical signs of vestibular disease, determination of an appropriate list of differential diagnoses, completion of a diagnostic plan, interpretation of appropriate diagnostic test results, and formulation of a treatment plan are all essential for appropriate management of a cat with a vestibular disorder.

The vestibular system is the primary sensory system that maintains balance and normal orientation relative to the earth's gravitational field. The vestibular system is composed of the peripheral vestibular system located in the inner ear and the central vestibular system located in the brainstem. It has two main functions: (1) to maintain the visual image by stabilizing the eyes in space during head movements; the stabilization is performed utilizing phasic or tonic vestibulo-ocular reflexes; and (2) to stabilize the position of the head in space—thus ensuring that the position of the body is stable. The stabilization is performed utilizing phasic or tonic vestibulospinal reflexes.

Anatomy and physiology

The ear consists of three parts: (1) the external ear consisting of the pinna and the vertical and horizontal portions of the external auditory canal; (2) the middle ear, consisting of the tympanic membrane, the middle ear cavity (tympanic cavity), auditory ossicles, and associated muscles; and (3) the inner ear, consisting of the cavity within the petrosal portion of the temporal bone and the membranous labyrinth of sacs and ducts within this cavity. Vestibular dysfunction most frequently results from disorders affecting the middle and inner ear.

The middle ear consists of an air-filled tympanic cavity lined by ciliated epithelium that is connected to the nasopharynx by the eustachian tube, and separated from the external environment by the tympanic membrane. In cats, unlike dogs, the tympanic cavity is divided into a larger ventromedial and a smaller dorsolateral compartment by a thin bony septum, which arises along a line on the bulla wall running from mid-rostral to mid-lateral. The ventromedial and dorsolateral compartments communicate through a narrow fissure on the caudomedial aspect of the dorsolateral compartment, where the septum is incomplete. The smaller dorsolateral compartment is arbitrarily divided into the epitympanum dorsally and the mesotympanum.

Postganglionic sympathetic nerve fibers cross the tympanic cavity of the cat and disruption of these nerve fibers will result in an ipsilateral Horner's syndrome (miosis, ptosis, enophthalmos, and protrusion of the nictitating membrane). These sympathetic fibers arise from the cranial cervical ganglion located immediately caudal and medial to the tympanic bulla. They enter the

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1098-612X/03/020101+08 $30.00/0

tympano-occipital fissure caudal to the tympanic bulla, and then pass between the tympanic bulla and the petrosal part of the temporal bone. The nerve fibers enter the tympanic cavity at the caudal extremity of the promontory, where they branch to form the tympanic plexus. The tympanic plexus is superficially located on the surface of the promontory, and is highly vulnerable to surgical trauma at this location. Careless removal of the bony septum, or excessive curettage of the promontory, during bulla osteotomy, will result in a Horner's syndrome that may be permanent.

The inner ear consists of the bony labyrinth, a cavity within the petrosal portion of the temporal bone, and the membranous labyrinth, a delicate system of ducts and sacs within the bony labyrinth. The membranous labyrinth lies like a sleeve within the bony labyrinth. The space between the membranous labyrinth and bony labyrinth is filled with perilymph and the membranous labyrinth is filled with endolymph. The vestibulocochlear nerve (VIIIth cranial nerve) terminates in sensory areas in the membranous labyrinth.

The vestibular organ consists of interconnecting canals in the bony labyrinth, and a single large cavity to which all canals connect, called the vestibule. The vestibular portion of the membranous labyrinth consists of three curved tubes (lateral, caudal, and rostral semicircular canals) and two membranous sacs (the saccule and the utricle). The three semicircular canals each contains a dilatation (ampulla), that in turn contains a crista. Receptor cells (hair cells) are located within each crista. Receptor cells are also located within the macula of the two sac-like structures within the bony vestibule—the saccule and the utricle.

The receptors located in the semicircular canals are responsive to angular (rotational) acceleration or deceleration, while the receptors located in the utricle and saccule are responsive to linear acceleration (or deceleration) and gravity. The receptor cells maintain a tonic discharge at rest. This tonic rate of discharge is either increased or decreased when the receptor cells are excited by movement. The receptor cells generate a potential in the primary afferent fibers in the vestibular portion of the vestibulocochlear nerve.

Primary afferent fibers from the vestibular receptors within the vestibulocochlear nerve pass through the skull via the internal acoustic meatus (along with the facial nerve), and enter the brain stem at the level of the rostral medulla. Many of the nerve fibers synapse in one of the four vestibular nuclei within the medulla, however, some bypass the vestibular nuclei, and enter the cerebellum via the caudal cerebellar peduncle.

There are numerous vestibular connections within the CNS. Connections descend the spinal cord via the lateral vestibulospinal tract to lower motor neurons of muscles of the neck, and thoracic and pelvic limbs. This is the largest (and thus the fastest) of all the descending pathways and is important for posture and locomotion. Interruption of input to this pathway from one side results in ipsilateral inhibition of extensor muscles and facilitation of flexor muscles, and contralateral facilitation of extensor muscles. The medial vestibulospinal tract descends in the medial longitudinal fasciculus to terminate in lower motor neurons of extensor muscles of the neck and back. Interruption of this pathway may result in flexion of the neck and trunk laterally, with the concavity directed toward the side of a vestibular lesion. Nerve fibers from all of the vestibular nuclei synapse with the lower motor neurons of the IIIrd, IVth, and VIth cranial nerves that innervate the extraocular muscles. These fibers are involved in the oculocephalic reflexes (eg, rolling of the eyes when the head is moved). There are numerous projections from vestibular nuclei into the reticular formation. Some of these are involved in the vomiting and cardiovascular reactions that may occur in vestibular disturbances.

Clinical signs of vestibular disease

Vestibular disease produces varying degrees of loss of equilibrium causing imbalance, nystagmus, and ataxia. Strength is not interfered with, and therefore paresis is not observed. As a rule, the disturbance is unilateral or asymmetrical, and the signs are those of an asymmetrical ataxia with preservation of strength. Unilateral vestibular signs may result from either central (brain stem) or peripheral (labyrinth) disease. It is important to differentiate central from peripheral disease because of the differences in treatment and prognosis. Signs of vestibular disease include: falling, rolling, tilting of the head, circling, nystagmus, positional strabismus (deviation of one eye in some head positions), and an asymmetrical ataxia.

All these clinical signs should not be expected to occur in any one case. Many of the clinical signs of vestibular dysfunction are transient, and may disappear, or be compensated for, by the time of clinical examination. In addition, many of the

isolated clinical signs result from lesions other than those of the labyrinth (eg, cerebellar lesions look similar to vestibular lesions). It should also be kept in mind that nystagmus is not pathognomonic for any syndrome. Alteration in signs of vestibular dysfunction occurs over time. Horizontal nystagmus caused by unilateral labyrinthectomy disappears after the 3rd or 4th postoperative day. Ataxia may at first seem more pronounced in the thoracic limbs, but later becomes greater in the pelvic limbs and after 3 weeks the severity of the neurologic deficits lessens tremendously (eg, only when an animal runs or jumps does it show evidence of disequilibrium).

Unilateral vestibular dysfunction

Loss of coordination and balance is reflected in a head tilt, with the more ventral ear usually directed toward the side of a vestibular lesion. Affected animals may also lean, fall, roll, or circle tightly, usually toward the side of a lesion. It may be possible to elicit mild hypertonia and hyperreflexia in the limbs on the side of the body opposite to a vestibular system lesion. Animals roll toward the side of the lesion, due to reduced extensor tone on that side and increased extensor tone on the side opposite the lesion. An animal will often fall when attempting to shake its head. Vision will assist an animal to compensate for a vestibular system deficit. Blindfolding an animal with subtle signs of vestibular disease may exacerbate signs of vestibular dysfunction, particularly a head tilt or circling. Cats with vestibular dysfunction assume a wide-based stance, and may lean or drift, usually toward the side of a lesion. Animals with acute vestibular dysfunction may also vomit, and/or 'appear to be nauseous'.

Nystagmus

Nystagmus is defined as involuntary rhythmic oscillations of the eye. It may occur with dysfunction of the CNS (cerebellum or brain stem), the eye, labyrinth (inner ear), or it may be induced in normal cats, where it is termed physiological nystagmus (eg, when moving the head from side to side, or following rotation of the head, or by irrigation of the external ear canals with either warm or cold water). Should nystagmus occur when the head is held still and there is no rotation or movement of the surroundings, it is called spontaneous nystagmus. Spontaneous nystagmus is usually pathological in origin. Spontaneous nystagmus may be horizontal, rotatory, or vertical in direction. If nystagmus occurs only when the head is placed in an unusual position (eg, laterally or dorsally), it is known as positional nystagmus.

Although the oscillations of the eye may occur with equal movements in each direction (pendular nystagmus), typically, nystagmus consists of a slow phase in one direction and a fast phase in the other. It is customary to describe nystagmus clinically in terms of the fast phase, despite the fact that in most cases the slow phase will be directed toward the affected side. It should be noted that a cat may have a nystagmus in one direction early in a disease, and later may develop a nystagmus in the opposite direction. This change of direction is explained by the probability of an early disease process acting as an irritating lesion to produce ipsilateral nystagmus, and the progression of the lesion later resulting in a destructive process producing contralateral nystagmus. Nystagmus tends to occur early in the course of peripheral vestibular disease, and to disappear later. Pendular nystagmus is usually associated with visual pathway deficits in primarily Siamese cats.

Abnormal nystagmus is a sign of disturbed vestibular input to the neurons that innervate extraocular eye muscles. Nystagmus probably occurs at some time during all types of vestibular disease and nearly always affects both eyes equally.

Nystagmus is described in terms of: (1) its direction (horizontal, rotary, or vertical); (2) whether it is non-positional (constant direction in all head positions) or positional (nystagmus that occurs only when the head is placed is an unusual position (eg, laterally or dorsally), or changes direction with altered head position); and (3) whether it is conjugate (same direction in each eye) or dysconjugate (different direction in each eye). In some cats with severe acute vestibular dysfunction, there may be a head oscillation or eyelid contraction that corresponds to the rate of nystagmus.

Postural reactions—strabismus

When the head is extended in a tonic neck reaction, the eyeballs should remain in the center of the palpebral fissure in dogs and cats. This often fails to occur on the side of a unilateral vestibular disturbance, and results in a ventrally deviated eyeball. Occasionally, in vestibular disease, an

eyeball is noticed to deviate ventrally or ventrolaterally without extension of the head and neck. This appears as an LMN strabismus, and can be corrected by moving the head into a different position or by inducing the patient to move its eyeballs to gaze in different directions. This is referred to as vestibular strabismus. The ventrally deviated eyeball is on the side of the vestibular lesion. Occasionally, the opposite eyeball will appear to be deviated dorsally.

Paradoxical central vestibular syndrome

Unilateral lesions of the peripheral vestibular system produce a head tilt toward the side of the lesion. With few exceptions, the same occurs with lesions of the central components of the vestibular system. Exceptions to this rule are therefore termed 'paradoxical'. Some unilateral lesions of the central vestibular pathways, especially unilateral involvement of the flocculonodular lobe of the cerebellum or the supramedullary part of the caudal cerebellar peduncle, produce a head tilt and ataxia directed toward the side opposite to the lesion, and a nystagmus with the fast component toward the side of the lesion. Such lesions are usually space-occupying lesions. Usually these lesions will produce postural reaction deficits or additional cranial nerve abnormalities on the affected side, which aid in determining on which side a lesion is located.

Bilateral vestibular disease

Cats with bilateral peripheral vestibular disease with complete loss of vestibular function do not have postural asymmetry (eg, head tilt), nor do they have nystagmus. A characteristic 'side-to-side' head movement often accompanies these signs. Normal vestibular nystagmus cannot be elicited by head movement or caloric testing. Normal oculocephalic reflexes are absent, (ie, the eyes do not move when the head is moved), and affected cats usually have a symmetrical ataxia and may fall to either side.

Peripheral vestibular disease

Clinical signs of peripheral vestibular dysfunction result from disorders of the middle and inner ear, that involve the receptors in the labyrinth and the vestibular nerve. Vestibular dysfunction does not occur with disease of the middle ear alone, however, many middle ear diseases extend to involve the inner ear, and result in signs of peripheral vestibular disease. Middle ear (bulla ossea) lesions usually produce head tilt (ipsilateral to the lesion) only, in the absence of other signs. Horizontal or rotatory nystagmus may be seen occasionally. Inner ear disease, which actually involves the receptors and vestibular nerve within the petrosal bone, usually produces other signs in addition to the ipsilateral head tilt—falling, rolling, circling, nystagmus, positional strabismus, and asymmetrical ataxia.

Horner's syndrome (miosis, ptosis, enophthalmos) of the ipsilateral eye may be present with either middle or inner ear disease in dogs and cats, because the sympathetic trunk passes through the middle ear in close proximity to the petrosal bone. The facial nerve may be affected in inner ear disease, as it courses through the petrosal bone in contact with the vestibulocochlear nerve. The primary characteristics of unilateral peripheral vestibular disease are: asymmetrical ataxia without deficits in postural reactions, and a horizontal or rotatory nystagmus that does not change in direction with different head positions. The fast phase of the nystagmus is directed away from the affected side. Mental status is normal, and paresis or deficits of proprioceptive positioning should not be present.

Central vestibular disease

Any signs of brainstem disease in association with vestibular signs indicate that central involvement is present. The most frequent differentiating feature is a deficit in postural reactions, as central vestibular lesions most often result in paresis or loss of conscious proprioception. Alterations in mental status, or deficits in Vth or VIIth cranial nerves, are also indicative of central disease. Nystagmus may be a key to differentiating central from peripheral disease. Nystagmus occurs in most central syndromes, and appears to be a permanent deficit. It is a positional nystagmus; therefore, it may be present in some head positions (with respect to gravity), but not in others and may also be dysconjugate. Also the nystagmus may vary in direction with change in head position. Vertical nystagmus in any head position is most consistent with central vestibular disease.

Affected cats usually have a head tilt, ventrolateral strabismus, and asymmetrical ataxia. Other clinical signs of brain stem disease (eg, ipsilateral proprioceptive positioning deficits, hemiparesis, reduced mental status), and other cranial nerve deficits (particularly cranial nerve V) or signs of cerebellar disease, (eg, dysmetria and

intention tremors), may be present. Clinical signs (eg, seizures) reflecting involvement of other areas of the CNS, seen in association with clinical signs of central vestibular dysfunction, may be apparent in animals with multifocal disease.

Diagnostic approach to vestibular disease

A complete history and a thorough physical and neurological examination, including ophthalmoscopic and otoscopic examinations, are essential in the management of a cat exhibiting clinical signs of vestibular dysfunction. A minimum database, (including a complete blood count, serum biochemistry profile, urinalysis, thoracic radiographs, and abdominal ultrasound or radiography) should be obtained. Results of these examinations may provide evidence of multisystemic or concurrent disease.

Results of a neurological examination usually permit localization of a vestibular disorder to either the peripheral or central parts of the vestibular system. Since many different disorders located in the same area of the vestibular system will produce similar clinical signs, additional diagnostic testing is required in order to make a diagnosis. Generally, the least invasive diagnostic tests are done before the more invasive tests.

Peripheral vestibular disease

General anesthesia is recommended in order to thoroughly examine the pharynx and ears, and radiograph the skull. Skull radiographs (lateral, dorsoventral, open mouth, and left and right oblique projections) and, when available, computed tomography (CT), aid in the evaluation of the tympanic cavity and bulla, and petrosal portion of the temporal bone. Myringotomy (needle aspiration through the ventrocaudal part of the tympanic membrane) allows collection of fluid from the tympanic cavity for cytological examination and microbial culture and sensitivity testing. Abnormal tissue within the external auditory canal or tympanic cavity may be biopsied for cytological or histopathological examination.

Brain stem auditory-evoked potential (BAEP) testing may be used to estimate peripheral and central auditory function. Since the auditory and vestibular parts of the vestibulocochlear nerve are in close proximity, BAEP testing may provide useful information regarding the functional status of the peripheral portions (and central pathways) of the VIIIth cranial nerve.

Diagnostic approach for central vestibular disorders

Magnetic resonance imaging (MRI) is the preferred modality for imaging the central vestibular system, although skull radiographs and CT may be useful in demonstrating bony abnormalities or signs of acute trauma such as hemorrhage. MRI is more sensitive than CT for imaging brain stem structures, and is not susceptible to the artifacts (such as beam hardening artifact) that may occur with CT examination of the caudal fossa and brain stem. Intravenous contrast should always be administered, as some lesions are not apparent without it.

Cerebrospinal fluid (CSF) analysis may be helpful in making a diagnosis of a cause of central vestibular system dysfunction. Results of CSF analysis usually are supportive, rather than diagnostic, of a disease process, unless organisms (eg, *Cryptococcus* spp) or tumor cells (eg, malignant lymphocytes) are present. CSF may also be submitted for serological assessment (eg, *Toxoplasma gondii* titer) or anaerobic and aerobic microbial culture and sensitivity testing.

In cats with mass lesions, biopsies (either open or CT-guided) should be considered, and the tissue submitted for cytological examination (conventional or crush preparation) and/or histopathology. As with lesions located elsewhere in the body, a definitive diagnosis may only be possible following biopsy.

Disorders of the peripheral vestibular system

Feline idiopathic vestibular disease

This is an acute vestibular syndrome of cats of all ages particularly in the summer months. The signs appear suddenly, and often result in severe dysfunction and inability to stand and walk. Cats develop an acute onset of severe peripheral vestibular dysfunction in the absence of facial paralysis, Horner's syndrome, or CNS involvement. Clinical signs are often preceded by, or occur concurrently with, upper respiratory tract disease. In a few days the affected animals tend to stabilize and improvement continues for several weeks. Residual deficits such as mild head tilt may persist, and blindfolding or darkness will cause a re-occurrence of signs well after apparent recovery has occurred. It is important to distinguish this idiopathic benign disorder, which resolves spontaneously without therapy, from otitis media-interna, which requires vigorous

therapy and may produce recurrent or persistent signs.

Diagnosis is based on the exclusion of other causes of vestibular disease. The idiopathic disease is characterized by a peracute onset of head tilt, asymmetrical ataxia, and horizontal or rotatory nystagmus, in the absence of facial paresis, Horner's syndrome, or signs of CNS involvement. An absence of otitis externa, normal tympanic membranes, and normal radiographs of the temporal bones further supports this diagnosis. The cause of this idiopathic disorder remains undetermined although it has been speculated that migration of *Cuterebra* larvae through the inner ear may be a cause in some cats. Prognosis for spontaneous recovery is good; however, recovery may require 2–4 weeks. Recommended therapy is supportive care, and prognosis for recovery is excellent.

Otitis interna (or labyrinthitis)

Labyrinthitis refers to inflammation of the inner ear that results in dysfunction of the membranous labyrinths. It is a common cause of peripheral vestibular dysfunction in cats. Usually otitis interna is bacterial in origin, and secondary to otitis media, which may occur secondary to otitis externa. Otitis media may also be caused by extension of infection from the pharynx to the tympanic cavity via the auditory tube, or by hematogenous dissemination. Other causes of otitis media/interna include yeast, fungi (eg, *Cryptococcus* spp), parasites, foreign bodies (eg, grass awns), and inflammatory polyps or neoplasms. Extension of infection to involve the brain stem by means of the internal auditory meatus may occur.

Varying degrees of vestibular dysfunction accompany labyrinthitis. Ipsilateral head tilt, nystagmus (usually rotatory), and ataxia are almost always present. Circling, falling, and rolling, may be seen in more severely affected animals. Ipsilateral facial paresis/paralysis and Horner's syndrome may occur. Because the facial nerve contains the parasympathetic preganglionic neurons that modulate lacrimal gland secretion, animals with labyrinthitis may have decreased tear production and develop ipsilateral keratoconjunctivitis sicca. Deafness, resulting from involvement of the cochlear nerve, may accompany otitis interna.

Diagnosis of otitis media/interna is based on otoscopic examination and imaging. Otoscopy may reveal otitis externa, and evidence of erosion or rupture of the tympanic membrane. Fluid in the middle ear may produce discoloration or bulging of the tympanic membrane and a sample may be obtained by myringotomy. Inflammatory exudate or fluid should be submitted for culture and sensitivity testing. Imaging (skull radiographs, CT, or MRI) may reveal a fluid or soft tissue density in the tympanic cavity, or sclerosis and lysis of tympanic bulla and adjacent bones. Skull radiographs may be normal in acute infections.

Prognosis usually is good when long term oral antibiotic therapy is initiated on the basis of results of culture and sensitivity testing. Although clinical signs may improve within 1–2 weeks, antibiotic therapy should be continued for at least 6 weeks. In more chronic cases, unresponsive to medical therapy, surgical drainage of the tympanic cavity may be necessary by means of a lateral or ventral bulla osteotomy. It must be kept in mind that the tympanic bulla in cats is divided into two compartments by an incomplete bony septum and that the communication between the dorsolateral and ventromedial compartments may be obstructed by exudate, and both compartments should be surgically drained.

Congenital peripheral vestibular disease

Congenital vestibular disorders have been reported in Siamese and Burmese kittens. In affected Siamese kittens, clinical signs develop at 3–4 weeks of age and usually kittens demonstrate clinical improvement by 3–4 months of age. Clinical signs of vestibular dysfunction may be accompanied by deafness. Clinical signs in Burmese kittens develop at or shortly after birth, and are non-progressive. Lesions are not apparent on pathological examination. Although a hereditary problem is suspected in both breeds, it has not been proven. Congenital deafness has been reported in white cats. Diagnosis of congenital peripheral vestibular disease is based on history, and results of serial neurological examinations and BAEP testing.

Trauma

Cranial trauma may result in signs of peripheral vestibular disease secondary to fracture of the petrosal part of the temporal bone or tympanic bulla. Facial paralysis may accompany petrosal bone injury. Fractures of the petrosal portion of the temporal bone may predispose cats to infection from organisms ascending from the

tympanic cavity or ear canal. Diagnosis is based on history and imaging (skull radiographs or CT). Treatment consists of supportive care. Broad spectrum antibiotic administration should be considered.

Toxicity

Therapy with ototoxic agents may result in degeneration of the vestibular and/or auditory receptors, usually resulting in permanent dysfunction. Bilateral vestibular dysfunction may result from ototoxic drug administration. Ototoxicity may occur as a result of oral, parenteral, or topical drug therapy. Although ototoxicity from topical drug therapy to the external ear occurs more commonly if the tympanic membrane is ruptured, it may occur in the presence of an intact tympanic membrane.

All aminoglycoside antibiotics may have a toxic effect on either the peripheral vestibular or auditory system, or both. Prolonged therapy with aminoglycoside antibiotics may result in degeneration of the labyrinth receptors of the vestibular or auditory systems, or both. Gentamicin and streptomycin are reported to affect primarily the vestibular system, whereas neomycin, kanamycin, tobramycin, and amikacin affect primarily the auditory system.

Loop diuretics such as ethacrynic acid, bumetanide, and furosemide are ototoxic. Furosemide is the least ototoxic and its effects are reported to be reversible if used for a short period of time.

Ear canal cleansers and vehicles in otic preparations such as propylene glycol, chlorhexidine, and cetrimide also are ototoxic, particularly if the tympanic membrane is ruptured.

In the southeastern USA, cats may develop acute peripheral vestibular disease from ingestion of the tail of the five-lined skink.

Diagnosis is based on history, and results of otoscopic examination and BAEP testing. Treatment consists of cessation of the drug therapy and initiation of supportive care. Prognosis for recovery from the vestibular dysfunction is good, particularly when the cause is recognized early, however, some permanent deficits may remain and deafness may be permanent.

Feline nasopharyngeal polyps

Feline nasopharyngeal polyps are well vascularized soft tissue growths lined by epithelium. These have been seen within the auditory tube, middle ear, external ear canal, or nasopharynx of cats. Definitive etiology for the polyps is not known, however, chronic inflammation, infection with calicivirus, and congenital or familial factors have been hypothesized as causes. Nasopharyngeal polyps usually affect cats less then 3 years of age, however, they have been reported in older cats. Polyps may grow into the middle ear, nasopharynx, or external ear canal, and clinical signs occur secondary to obstruction of the nasopharynx or ear canal. Associated clinical signs include: dysphagia, voice change, inspiratory stridor, sneezing, rhinitis, otitis externa, and otitis media/interna, with or without associated peripheral vestibular disease. Diagnosis is made on the basis of an otoscopic examination, nasopharyngeal examination, and imaging (skull radiographs, CT, or MRI). Treatment is surgical removal of the polyp by means of ventral bulla osteotomy. Prognosis following surgical removal is excellent, however, the polyp may return.

Neoplasia

Neoplasms that involve the temporal bone may produce peripheral vestibular disease, often in association with facial paralysis or paresis. Malignant tumors reported to occur include fibrosarcoma, osteosarcoma, chondrosarcoma, squamous cell carcinoma, ceruminous gland adenocarcinoma, lymphoma, and sebaceous gland adenocarcinoma. Squamous cell carcinoma appears to be the most frequently occurring tumor affecting the middle ear of cats. Rarely, middle ear tumors may extend directly into brainstem. Neurofibromas of the vestibulocochlear nerve usually cause signs of unilateral vestibular disturbance prior to compression of brainstem. Inflammatory polyps have been observed within the middle ear of cats. Resection is recommended, however, the polyp may re-occur.

Diagnosis is based on the finding of destructive bony lesions on skull radiographs or CT, and on biopsy results. Prognosis varies with the invasiveness of the neoplasm, however, is generally considered poor.

Disorders of the central vestibular system

Any cause of meningo-encephalitis may result in involvement of central vestibular structures. Reported causes include feline infectious peritonitis, toxoplasmosis, and cryptococcosis.

Aberrant parasitic migration may produce severe signs of vestibular disturbance. Neoplasms of the cerebellomedullary angle affect the vestibular system. Neoplasms may be located at the surface of the parenchyma (eg, meningioma, neurofibroma, medulloblastoma, choroid plexus papilloma, or malignant lymphoma), or may be located within the parenchyma (eg, glioma or granulomatous meningo-encephalomyelitis). Neoplasms at these locations occur in animals of all ages; however, young dogs appear to be susceptible.

Thiamine deficiency may occur in cats fed raw fish exclusively, following chronic anorexia without vitamin therapy, and in cats fed highly processed foods without thiamine added. Nervous tissue has a high metabolic rate and thiamine necessary for energy pathways. Initial clinical signs of thiamine deficiency include anorexia, followed by vestibular ataxia, which progress rapidly to cervical ventroflexion, mydriasis, recumbency, seizures, and coma. Pathological findings in thiamine deficiency are bilaterally symmetrical hemorrhagic necrosis of the brain stem periventricular gray matter.

Metronidazole toxicity, resulting in clinical signs of acute CNS dysfunction, has been reported in three cats. The cats were treated with metronidazole at doses ranging from 48 to 62.5 mg/kg/day for 5 days to 10 months prior to presentation. Although diffuse neurological abnormalities were present in these cats (2/3 cats also had seizures), signs of ataxia and disorientation were reported in all three cats. Vertical nystagmus is a consistent finding in dogs with metronidazole toxicity, and could potentially occur in cats. History and clinical signs are the key to diagnosis. Treatment consists of supportive care and withdrawal of the drug. Prognosis for recovery is good unless the clinical signs are severe.

Further reading

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