Proceedings of the 33rd World Small Animal Veterinary Congress
Dublin, Ireland - 2008

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**Vestibular Disease in Dogs and Cats**

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The onset of a head tilt is a cardinal sign of vestibular disease. It is the most consistent sign of unilateral vestibular dysfunction, which occurs as a result of the loss of anti-gravity muscle tone on one side of the neck. Bilateral vestibular disease may also cause a head tilt if the disease process is not symmetrical but usually does not cause such a sign. The vestibular system has two functional components; the peripheral component is located in the inner ear and the central component is located in the brain stem and cerebellum. Unfortunately, a head tilt is not specific for a brainstem or a peripheral vestibular system lesion localisation, nor does it indicate a specific aetiology. Generally, central vestibular disease is associated with different aetiologies and a worse prognosis than peripheral vestibular disease. Therefore, it is essential to approach these cases aiming to primarily further localise the neurological lesion.

**Differentiation of vestibular lesions**

**Clinical signs due to disease of the peripheral vestibular system**
- Head tilt, circling, falling or rolling toward the side of the lesion
- If nystagmus is present it is horizontal or rotatory with the fast phase away from the side of the lesion
- May have concurrent facial nerve paralysis (CN VII) and Horner’s syndrome (ptosis, miosis and enophthalmos) with middle and inner ear lesions

**Clinical signs due to disease of the central vestibular system**
- Head tilt, circling, falling, or rolling toward or away from the side of the lesion
- Conscious proprioceptive deficits, hemiparesis or hemiplegia on the side of the lesion if unilateral or worse on one side if bilateral
- Nystagmus may be horizontal, rotatory or vertical if present
- May be depressed or stuporous
- May have facial paralysis (CN VII) but Horner’s syndrome is rare

**Diagnostic plan for patient with vestibular dysfunction**

1. Obtain a comprehensive history of the onset of the disease. Information regarding previous systemic disease, previous ear disease, previous medication administration (both topical and systemic), travel history, the possibility of toxin exposure and head trauma is important.
2. Perform a complete neurological examination. There are several very important components of the examination that can assist with lesion localisation. In addition to the head tilt, the other signs of vestibular disease include pathologic nystagmus, positional strabismus and ataxia characterized by a base-wide stance and swaying of the trunk and head. Pathological nystagmus can be spontaneous or positional. Spontaneous nystagmus is nystagmus that occurs when the head is not moving and is in the normal position. A unilateral acute peripheral lesion often causes spontaneous nystagmus with horizontal and rotational components. The fast phase is directed away from the side of the lesion. Spontaneous nystagmus resulting of central origin can be purely vertical, horizontal or rotational. Positional nystagmus refers to nystagmus that is present only when the head is placed in an unusual position, for example upside down. There are less specific neurological signs that may be associated with vestibular dysfunction that include: conscious proprioception deficits (indicative of brainstem disease); Horner’s syndrome (usually indicative of peripheral disease); cranial nerve deficits (multiple cranial nerve dysfunction usually indicates central disease, but facial nerve paresis can occur with central or peripheral disease); head tremor (indicating cerebellar disease); and an altered level of consciousness (indicative of central disease). The differentials for peripheral and central vestibular disease are discussed below. The next steps may be determined by the lesion localisation.
3. A minimum database should be performed on all cats which should include haematology, serum chemistry, urinalysis and thoracic radiographs. If toxicity is suspected due to antibiotic administration (metronidazole and aminoglycosides), I would recommend immediate withdrawal of the medication. In most cases, there is resolution of the vestibular abnormality. In the absence of a systemic health concern, a practical approach can be taken with peripheral vestibular disease cats.
4. Ancillary tests for patients with peripheral disease include: otoscopic examination; imaging of the tympanic bullae with radiography; computed tomography (CT), or magnetic resonance (MR) imaging; and thyroid function testing. A brainstem auditory evoked response (BAER) test may be of value.
in examining the auditory portion of cranial nerve VIII (vestibulo-cochlear nerve) as well as potentially assisting in localising the disease to the peripheral or central components. Diagnosis of central disease is based upon CT or MR imaging of the brain and analysis of cerebrospinal fluid (CSF).

Causes of peripheral vestibular disease

Idiopathic vestibular syndrome

Idiopathic vestibular syndrome is a common cause of peripheral vestibular disease in the dog and cat. The onset of the vestibular disease may be so acute that is accompanied by vomiting, but this is not specific for this aetiology. Postural reactions are normal in this disease, and cats do not have facial paresis or Horner’s syndrome. This can be only by diagnosed by ruling out the other causes; however, it is the only differential of peripheral disease that will start to improve in 72 hours with no specific treatment. If the patient is closely monitored over this time course, marked resolution of the nystagmus can be seen. This will be followed by improved gait over a 7-day period and improvement of the head tilt over a 2-month period. The head tilt may never completely resolve. If there is evidence of facial nerve involvement or Horner’s syndrome further ancillary tests should be considered.

Otitis media-interna

Up-to 50% of cases of peripheral vestibular disease are due to otitis media-interna. Infection of the middle ear can cause vestibular disease due to the production and spread of bacterial toxins into the inner ear. The infection can directly damage the inner ear with spread to the labyrinth. The bacteria commonly responsible for this disease are *Staphylococcus* spp., *Streptococcus* spp., *E. coli* and *Pseudomonas* spp. If the culture results are negative but bacterial otitis interna is suspected, then oral enrofloxacin 5 mg/kg every 12 hours for dogs or cats or a combination of oral trimethoprim sulfadiazine (TMP-SDZ) 15 -30 mg/kg orally every 12 hours and oral cephealexin 22 mg/kg every 8 hours may be administered. Antibiotic therapy should be continued for 6-8 weeks. Tear production should be monitored to detect sicca associated with facial nerve (CN 7) involvement or antibiotic therapy and artificial tears administered if necessary to prevent keratitis and corneal ulceration. Aminoglycosides are contraindicated in otitis interna/media. In cases of concurrent otitis externa and media with a ruptured tympanic membrane, the external and middle ears are gently flushed with saline or a 2.5% acetic acid solution and caustic ear cleaning substances are avoided if possible. Diluted ear cleaning solutions may be necessary in some cases for debris that cannot be cleared by other means, but thorough rinsing is essential so residual cleaning solution does not irritate the vestibular labyrinth. Topical treatment of the external ear canal with small amounts of low residue non-irritating antibiotic solutions for a few days may be given but the accumulation of greasy ointments in the middle ear should be avoided. The vestibular signs may resolve in 1-2 weeks but if antibiotics are prematurely discontinued, the clinical signs and infection recur and can be more difficult to treat. Corticosteroids are usually not required and are avoided if osteomyelitis is present. In acute cases, oral prednisone 0.25 mg/kg once-daily for 1-3 days is sometimes given to reduce inflammation and swelling of the vestibular labyrinth. In animals with recurring otitis, underlying dermatologic problems such as atopy or hypothyroidism should be investigated and treated. Ear hygiene should be monitored but care should be taken when cleaning the external ear canals.

Surgical intervention: External ear canal ablation and bulla osteotomy in refractory cases.

Nasopharyngeal polyps

Polyps are pedunculated masses which arise from the lining of the tympanic cavity, eustachian tube, or nasopharynx. Non-septic otitis media/interna may occur secondary to occlusion of the eustachian tube due to a nasopharyngeal poly and polyps may occur as a result of chronic middle ear infection or from ascending infection from the nasopharynx. Diagnosis may require advanced imaging if physical examination and skull radiographs are not helpful.

Neoplasia

Neoplasia of the structures of the ear includes squamous cell carcinoma, ceruminous gland adenocarcinoma and lymphoma.

Ototoxicity

There are many drugs listed as being ototoxic and can potentially cause both vestibular dysfunction and deafness. The deafness caused by such drugs is often permanent whereas the vestibular disease may resolve or at least the dog may compensate for the abnormality. The most common topical drug implicated in this scenario is chlorhexidine.

Trauma

Head trauma may cause the onset of vestibular disease, which may be peripheral or central depending upon the severity of the trauma. Middle ear haemorrhage subsequent to a trauma may cause peripheral vestibular disease seen with or without facial paresis and Horner’s syndrome.

Congenital disease

Peripheral vestibular disease may be evident in young animals and attributed to a congenital malformation or degeneration of the inner ear structures. If the abnormality is bilateral, these animals may not have a head tilt or nystagmus; however, they will frequently
have a symmetrical ataxia, a wide-based stance and a side-to-side movement of the head in the horizontal plane. They may also be deaf.
Numerous breeds have been associated with congenital vestibular disease (German shepherd, Doberman, English cocker, beagle, Siamese, Burmese, Tonkinese). Clinical signs usually begin around 3-4 weeks of age (when the animal begins to ambulate) and may consist of a head tilt, nystagmus, strabismus, ataxia, circling, falling, rolling, and abnormal head movements. Many learn to compensate by 2-4 months of age but some will remain permanently affected. Recurrence can occur. With bilateral disease, there is usually no abnormal nystagmus and normal nystagmus cannot be elicited.

Causes of central vestibular disease

**Inflammatory disease**
The common infectious diseases responsible for inflammation of the brain and its structures are canine distemper, feline infectious peritonitis (FIP), *Toxoplasma*, bacteria and *Cryptococcus*. Whilst all the above infections can cause vestibular disease, they often have a multifocal central nervous system distribution and may also cause profound systemic abnormalities. The prognosis for each patient not only depends on the infectious etiology but also on the severity of the presenting signs, neurological as well as systemic. There are several inflammatory diseases of unknown aetiology and presumed immune-mediated. These include granulomatous meningoencephalomyelitis (GME) and necrotizing meningoencephalitis and can both affect the central vestibular system as part of a multifocal disease.

**Neoplasia**
The most common tumours to cause central vestibular disease in cats are meningiomas and lymphomas located at the cerebello-medullary pontine angle. Tumors of the forebrain can also cause central vestibular disease due to caudal transtentorial herniation with secondary compression of the brain stem.

**Metronidazole toxicity**
Doses greater than 30 mg/kg/day can result in vestibular disease. The onset is acute and usually occurs when animals receive high doses for a long duration (e.g., after being on high doses for 7 to 12 days). Dose reductions need to be made in patients with liver and kidney disease as the drug is metabolized and excreted by these organs, respectively. Clinical signs may include generalized ataxia, nystagmus, anorexia, and vomiting. In severe cases, altered mental status, seizures, and opisthotonus may be present. Removal of the drug and supportive care (IV fluid diuresis) usually results in quick recovery. Occasionally, deficits are permanent.

**Cerebrovascular disease**
Cerebrovascular disease (CVD) may cause a peracute onset of central vestibular signs. Ischemia may be embolic or thrombotic and is the most common form of CVD; its causes may be due to sepsis, neoplasia, endocrine disease, hypertension or may commonly be idiopathic. In some areas of the country this can result from aberrant parasite (cuterebra) migration through the nervous system. Non-traumatic hemorrhage, the other form of CVD, is bleeding into the parenchyma of the brain that may extend into the ventricles. Recovery from this disorder can be complete but can depend on the underlying disorder.