Vestibular disease in dogs and cats: balancing good from bad – part two

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Mark Lowrie describes the more common diseases responsible for vestibular disease in dogs and cats, followed by a systematic investigation procedure required to diagnose the underlying disease, in the conclusion of a two-part article

PART one of this article (May 3 issue) discussed the anatomy of the vestibular system and the steps we need to take to distinguish peripheral from central vestibular disease.

Having determined whether the patient has central or peripheral vestibular disease, we must then determine the likely diseases that can result in such a presentation. This article describes the more common diseases causing vestibular disease in dogs and. Having formulated a list of potential causes for vestibular disease, it is important to proceed through a systematic investigation to diagnose the underlying condition.

Diseases causing vestibular signs

Table 1 details the differential diagnoses of dogs and cats with peripheral and central vestibular disease, and highlights some of the more commonly seen diseases. I will now outline a more complete description of these common diseases:

• **Idiopathic vestibular disease.** This causes peripheral disease and occurs in both dogs and cats. It has been termed “old dog” or “geriatric vestibular disease” by some, although any adult cat or dog may develop signs. The disease is characterised by an acute onset of signs that can be severe

and crippling, including rolling, nystagmus, falling and a head tilt. Other deficits are not usually seen, so a patient with Horner's syndrome or facial nerve signs should be investigated for other causes (for example, otitis media/interna).

No treatment has proved beneficial, although occasionally symptomatic management of vomiting is advantageous. An improvement is commonly seen within the first few weeks, with the nystagmus resolving commonly within the first few days.

Most animals make a complete recovery, although severely affected animals occasionally maintain a residual head tilt.

This disease can cause bilateral vestibular disease, particularly in cats.

• **Otitis media/interna.** This is one of the most common aetiologies for peripheral vestibular disease in dogs and cats. Most commonly this is the result of:

– a descending infection from the external ear canal;

– spread of organisms from the pharynx via the auditory tube; or

– haematogenous spread.

Ipsilateral facial paralysis is a common finding in patients with ear disease, and treatment involves the administration of systemic antibiotics for a minimum of four to six weeks (the choice of antibiotics depending on culture and sensitivity results from the myringotomy). In severe cases, surgical management may be necessary. The prognosis is guarded to fair, although a residual head tilt and/ or facial paralysis is possible.

In rare cases, intracranial infection can result from extension of the middle-ear infection along the nerves and vessels of the internal acoustic meatus or by haematogenous spread. This can result in signs of central vestibular disease.

• **Meningoencephalitis of unknown aetiology.** Pathogenfree inflammatory brain disease is relatively common in dogs, yet rare in cats.

Several distinct types of inflammatory brain disease are recognised (for example, granulomatous meningoencephalomyelitis and necrotising encephalomyelitis), although a definitive diagnosis is only obtained by histopathology (which is currently rarely performed).

Clinical signs are typically acute or progressive and may involve many parts of the brain, leading to central vestibular signs in conjunction with seizures or central blindness.

A presumptive diagnosis can be made based on a consistent history, signalment (frequently young to middle-aged female terrier breeds), multifocal contrast-enhancing lesions on MRI, a mononuclear or mixed-cell pleocytosis on CSF analysis and exclusion of infectious aetiologies by serological and PCR testing.

Treatment involves immunosuppressive doses of prednisolone, frequently in combination with another immunomodulatory agent (for example, cytosine arabinoside or cyclosporine). However, an optimal regime is yet to be established.

Prognosis is extremely variable and depends, in part, on the severity of presenting signs. Despite this, it appears many dogs can do well with long-term medication, and anecdotally, many dogs can achieve full resolution of the disease after months to years of treatment.

• **Intracranial neoplasia.** Tumours can cause central (intracranial disease) or peripheral (middle-ear neoplasia and peripheral nerve tumours of VIII) vestibular signs. Central vestibular signs usually result from primary neoplasia within the caudal fossa (for example, meningiomas, choroid plexus tumours, ependymomas, gliomas and medulloblastomas) or secondary metastatic disease (for example, lymphosarcomas, haemangiosarcomas and carcinomas).

Prognosis is generally poor, although cases must be evaluated individually, as various options for management depend on the type and location of the tumour.

• **Cerebrovascular accident (CVA).** A CVA, or “stroke”, is an increasingly recognised cause for an acute onset of neurological signs in both dogs and cats. A stroke simply describes a disruption in the blood supply to the brain and is the result of an ischaemic infarction (for example, due to vessel occlusion or altered blood viscosity), or a haemorrhage.

Ischaemic infarctions are the most common manifestation of strokes in dogs and cats. They occur frequently in the cerebellum, resulting in signs of central and paradoxical vestibular disease, and are diagnosed by means of an MRI scan (Figure 1).

The term “transient ischaemic attack” (TIA) is used to describe an abrupt onset of focal neurological signs lasting less than 24 hours. In humans, these are believed to be the result of functional ischaemia, often preceding an ischaemic infarction.

The distinction of a TIA from an ischaemic infarction is that they are extremely short-lived and are not visible on MRI. Due to their transient nature and the lack of a definitive test, TIAs are poorly understood in veterinary medicine, although anecdotal reports suggest they occur.

In cases in which infarcts are suspected, underlying hypertension, hyperadrenocorticism, hypothyroidism and cardiac or renal disease should be investigated. An underlying cause is found in approximately 50 per cent of dogs suffering an infarct. The prognosis for patients in which a

predisposing medical condition is identified has been found to be much poorer than those in which a cause is not found. Given time and supportive care, many animals with infarcts will improve.

• **Hypothyroidism.** This is a canine disease that can manifest as peripheral or central disease. However, the pathogenesis for these two presentations is different, as outlined below:

– Polyneuropathy: causing peripheral vestibular signs. The pathogenesis of this phenomenon is unknown, but treatment of the endocrinopathy can result in improvement of vestibular signs. Hypothyroidism can cause bilateral vestibular disease.

– Myxoedematous disease: causing peripheral or central vestibular signs. Myxoedematous deposits are thought to develop alongside cranial nerves as they exit the skull foramina, causing compression and a neuropathy.

Diagnosis of hypothyroidism is by demonstration of a low-serum thyroxine (T4) and elevated thyroid hormone supplementation (TSH).

TSH usually results in improvement within a few months.

• **Metronidazole toxicity.** This can affect dogs and cats, resulting in central vestibular disease, and is usually seen in animals receiving high doses of the medication.

However, due to its hepatic metabolism, animals with liver dysfunction may develop signs of vestibular disease despite appropriate doses of metronidazole. Therefore, the history of any patient presenting with vestibular signs should be thoroughly reviewed to ensure it has not received this medication.

Treatment with diazepam has been reported to speed recovery, although the exact mechanism of action is uncertain. Prognosis is very good if early intervention is achieved.

Thiamine deficiency. This is a rare cause of central vestibular disease in dogs and cats. Most cases are the result of inappropriate food preparation, inadequate dietary concentration or feeding diets high in thiaminases (found in high concentrations in raw fish). Liver and gastrointestinal disease can also result in deficiency due to decreased absorption or metabolism. The pathological changes are symmetrical and focal, causing selective destruction of certain brainstem nuclei. Therefore, bilateral vestibular disease is one possible presentation for this disease without causing significant changes in mentation.

Often, a presumptive diagnosis is made based on the history, neurological examination and MRI findings. Urinary organic acid screening and transketolase activity in red blood cells gives a more definitive diagnosis. Most affected animals respond rapidly to oral thiamine supplementation.

• **Inflammatory polyps.** Polyps in cats are the most common benign pharyngeal and external/middle-ear masses observed in this species. They can occur at any age, although are typically seen in cats less than two years old.

Polyps may be unilateral or bilateral. Clinical signs depend on the location of the polyp, with upper respiratory signs resulting from polyps in the nasopharyngeal region or vestibular signs when a polyp is affecting the middle ear. Therefore, a cat presenting with respiratory signs preceding a peripheral vestibular episode should increase the suspicion of an inflammatory polyp.

Most polyps originate in the Eustachian tube and consequently block drainage from the middle ear, leading to clinical signs of ear disease and hence peripheral vestibular signs. Diagnosis can be made using radiography or advanced imaging (Figure 2).

Treatment involves surgical removal of the polyp when it is located in the middle ear, either by gentle traction or ventral bulla osteotomy. When middle-ear involvement is present, tissue and/or fluid samples should be submitted for bacterial culture and sensitivity as a bacterial otitis media may also be present.

Prognosis for this condition after surgical removal is excellent when the polyp has been completely removed.

Investigation

A full haematology and biochemistry panel should be performed in all cases, regardless of whether a central or peripheral lesion is suspected.

In dogs, inclusion of serum T4 and endogenous thyrotropin TSH is helpful. In an older patient, chest radiographs and an abdominal ultrasound scan are performed to exclude the possibility of systemic conditions that can spread to the nervous system (for example, a paraneoplastic syndrome or metastatic disease).

Brainstem auditory response (BAER) testing may be abnormal if the brainstem, peripheral nerve (VIII) or ear are involved, and it is occasionally possible to extrapolate the location of the lesion from the results of this test.

If peripheral vestibular disease is suspected, a thorough examination of the external ear canal should be performed under anaesthesia with the aim of visualising the tympanic membrane. However, the presence of an intact tympanic membrane does not eliminate the possibility of disease affecting the middle ear. If the tympanic membrane is ruptured, swabs of the middle ear may be taken for culture and sensitivity.

If the tympanic membrane is intact, but appears abnormal, a 20-gauge spinal needle can be used

to puncture the membrane to obtain samples for culture and sensitivity. Lavage of the middle ear may also be required. However, this must only be done with warm saline, as the toxins chlorhexidine and aminoglycosides cause vestibular disease, and will make an animal far worse if used to clean the ear.

Radiographic evaluation of the tympanic bullae has been reported, but the sensitivity of this procedure is low – as a normal radiograph does not rule out middle-ear disease – and, whenever possible, it is now recommended that at CT or MRI scan are performed.

Patients with central vestibular disease would usually require advanced imaging (CT or MRI). Depending on the results of the imaging, further tests may be performed, including cerebrospinal fluid analysis (nucleated cell count, cytology and total protein concentration) and infectious titres for various infectious organisms such as *Toxoplasma gondii, Neospora caninum*, canine distemper and feline infectious peritonitis (Figure 3).

If a specific cause is suspected (for example, thiamine deficiency or a cerebrovascular accident), then further tests may be warranted to confirm the diagnosis or to investigate underlying causes for this problem (refer to the previously described individual diseases for specifics).