

VESTIBULAR DISEASE: PART TWO – TREATMENT AND PROGNOSIS

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Rita Gonçalves looks at the different conditions that can cause vestibular system dysfunction and the treatments available to practitioners

Summary

The vestibular system is the part of the nervous system mainly responsible for maintaining the animal's balance and orientation in response to gravity. It detects the static position of the head and its rotational movements as well as its acceleration and deceleration. It then coordinates the position of the eyes, trunk and limbs in reference to the position and movement of the head. Part one of this article reviewed the anatomy of the vestibular system and the clinical signs associated with dysfunction. Part two will now focus on reviewing the different conditions that may be underlying this, discussing the appropriate diagnostic steps, treatment options and associated prognoses.

Key words

vestibular diseases, dog, cat, otitis media/interna

AS briefly discussed in part one (*VT* 41:24), many conditions have been described in association with vestibular disease (Munana, 2005; Kent et al, 2010). To determine the most likely differential diagnosis, it is necessary to consider the onset of the clinical signs (acute or chronic) and if there has been progression of disease.

A neurological examination should allow the problem to be localised, most likely affecting the central or the peripheral vestibular systems. Once this has been done, a short list of differential diagnoses should remain ([Table 1](#)); the most common diseases are now individually reviewed.

Otitis media/interna

Reported as the most common cause of peripheral vestibular disease in both dogs and cats (Garosi et al, 2001), otitis media is more common than previously thought and occurs in more than 50 per cent of chronic otitis externa cases (Gotthelf, 2004).

It should always be considered in cases that repeatedly present for ear infections, especially if these cases are associated with neurological deficits suggestive of peripheral vestibular disease (head tilt, facial paralysis and Horner's syndrome), as discussed in part one of this article. The occurrence of ipsilateral neurogenic dry eye and nose should also raise suspicion, as this can occur when there is damage to the parasympathetic fibres of the facial nerve that innervate the lacrimal and lateral nasal glands.

It most commonly occurs from extension of otitis externa that has not been treated, inappropriately treated or that has become resistant to treatment (in cases where bacteria are involved).

Chronic otitis externa leads to thinning and weakening of the tympanic membrane and, ultimately, to erosion or rupture of the eardrum. Exudates and infectious organisms can then drain to the middle ear from the external ear canal and become trapped in the ventral portion of the bulla.

The pathophysiology of otitis is complex, but understanding it is important so that a more comprehensive treatment plan – which not only addresses the present complaint but also prevents future recurrence – can be developed (Rosser, 2004). Contributing causes of otitis externa are usually divided into predisposing, primary and perpetuating factors.

• Primary causes

These refer to the factors that directly instigate inflammation of the ear canal.

They include parasites (with *Otodectes cynotis* the most common), foreign bodies (such as grass awns), keratinisation disorders, endocrine diseases (such as hypothyroidism and hyperadrenocorticism), autoimmune diseases (for example, pemphigus foliaceus and erythematosus) and hypersensitivity disorders (atopic dermatitis and adverse cutaneous food reactions most commonly).

The latter have been shown to be some of the most important complicating factors in cases of chronic/recurrent bilateral otitis externa, with up to 50 per cent of dogs with atopic dermatitis and up to 80 per cent of dogs with food allergy having concurrent otitis externa.

• Predisposing causes

Predisposing causes are the factors that increase the risk of developing otitis and which, working in combination with the primary and perpetuating factors, result in clinical disease.

They should be recognised and addressed, when appropriate, as part of a comprehensive treatment plan. They include conformation (such as hairy, pendulous or narrow ears), iatrogenic causes (mainly overcleaning and traumatic removal of hair), excessive moisture (in dogs that swim or are bathed frequently) and obstructive ear disease (such as inflammatory polyps and neoplasia).

• Perpetuating causes

Perpetuating causes are the factors that do not participate in the initial disease process, but contribute to its continuation once this is established.

They include bacterial infection (most commonly *Pseudomonas aeruginosa*, but also *Staphylococcus pseudintermedius*, *Proteus mirabilis*, *Escherichia coli*, *Corynebacterium* species, *Enterococcus* species and *Streptococcus* species) and yeast infection – although the presence of small numbers of *Malassezia pachydermatis* can be normal, in excess they may contribute to disease.

Diagnostic evaluation

The diagnostic evaluation should start with otoscopy. For adequate examination of the tympanic membrane, general anaesthesia is usually required (using an endotracheal tube is advised, as flushing can cause material to drain through the eustachian tube and result in aspiration).

In severe cases, no eardrum will be present, but in some instances it may be intact, especially in recurrent cases where a ruptured eardrum may have healed, trapping bacteria and yeast in the bullae. In these circumstances, myringotomy may be necessary to obtain samples for cytology and culture.

Radiography of the bullae may help determine the presence of bone involvement and of material in the middle ear, but sensitivity can be low, especially in acute disease. Computed tomography (CT) is more sensitive in such cases ([Figure 1a](#)). Magnetic resonance imaging (MRI) will be indicated mainly when there are neurological deficits, as extension of infection to the meninges can be detected this way ([Figures 1b and 1c](#)).

Treatment of otitis media should address all the factors discussed above. Once conclusively diagnosed, samples should be obtained from the middle ear for cytology and culture, followed by gentle flushing of the bulla, at which time infusion of topical medications can be performed if appropriate. Usually, inflammation is controlled by the use of corticosteroids, and appropriate

systemic and topical antibiotics should be used based on culture and sensitivity results for six to eight weeks.

Cases that are refractory to treatment or that have recurrent clinical signs may require surgery, namely total ear canal ablation and/or bulla osteotomy.

Idiopathic vestibular disease

Along with middle/inner ear disease, idiopathic vestibular disease is one of the most common causes of peripheral vestibular disease. Also called geriatric vestibular disease, this condition occurs in both dogs and cats (Schunk et al, 1983; Burke et al, 1985).

The typical presentation in dogs is an acute onset of vestibular ataxia (incoordination with loss of balance, leaning and falling to one side and occasionally rolling), head tilt (to the side where animals fall towards) and nystagmus (horizontal or rotatory, with the fast phase away from the side of the head tilt).

Occasionally, onset of the signs involves an episode of collapse that owners interpret as a seizure event. Asking if the animal was conscious, if there were any autonomic signs typical of seizure activity (urination, defaecation and salivation) or if the owners saw nystagmus at that time (which does not occur during seizures) can help determine the nature of this initial event.

Although uncommonly reported in the literature, cases with concurrent facial nerve paralysis are often seen in the author's referral hospital (Smith et al, 2010). The signs do not progress and typically improve over the days following onset with complete resolution in most cases within three to four weeks (uncommonly there may be a mild residual head tilt).

A study evaluating vestibular disease in cats (Negrin et al, 2010) showed that a significant proportion of cats with idiopathic disease presented with an acute onset of the clinical signs, but, unlike dogs, these signs progressed over the initial two to three weeks. Most cats still made a complete recovery without treatment, although this was slower than in dogs (usually within three months).

Diagnosis

Presumptive diagnosis is often made in cases with an appropriate history (acute onset and improvement of the clinical signs without therapeutic intervention) and the neurological deficits mentioned above (no signs suggestive of central vestibular dysfunction should be identified).

Careful examination of the external ear canal should be carried out, followed by imaging of the middle ear if considered appropriate. MRI of such cases excludes other conditions and occasionally shows enhancement of the vestibulocochlear nerve, indicating local inflammation.

The prognosis for recovery is good and no treatment is necessary.

Inflammatory brain disease

This category includes both infectious and non-infectious causes of central vestibular disease. Most of these conditions tend to be acute or subacute in onset and are invariably progressive. The associated neurological deficits will reflect their distribution within the central nervous system, but they are often multifocal and asymmetrical.

Several infectious agents have been associated with the presence of central vestibular signs; the most common include canine distemper virus and protozoal disease (toxoplasmosis and neosporosis) in dogs and feline infectious peritonitis virus and toxoplasmosis in cats.

In the UK, the non-infectious causes are significantly more common. These are globally known as meningoencephalitis (inflammation of the brain and surrounding meninges) of unknown aetiology (MUAs) and most commonly affect young to middle-aged small-breed dogs (Adamo et al, 2007).

This term is used to describe cases with meningoencephalitis (ME) in which an infectious agent cannot be identified and an immune-mediated cause is suspected; it includes patients with the granulomatous form (GME) and the more aggressive necrotising form (NME).

Diagnosis

Diagnosis is usually through analysis of the cerebrospinal fluid (CSF), which shows increased total nucleated cell counts and protein content, combined with advanced imaging – typically showing multifocal areas of oedema. The diagnostic investigation should also include serology for infectious diseases and CSF culture when appropriate.

Treatment options depend on the underlying aetiology – antibiotics for protozoal and bacterial diseases or immunosuppressive drugs in cases of non-infectious disease (prednisolone alone or in combination with cytosine arabinoside, ciclosporin, azathioprine or lomustine). Combination treatment appears to be associated with longer survival times and fewer side effects from the use of high doses of corticosteroids.

Prognosis

Prognosis appears to vary, most likely reflecting the severity of the underlying disease. A review of the current literature suggests dogs that respond well to treatment initially tend to have long survival times and, sometimes, even complete resolution of the disease. The ones that don't respond or that respond partially to treatment (with residual neurological deficits) are significantly more prone to relapses and often have shorter survival times.

Cerebrovascular disease

This refers to an abnormality of the brain caused by a disturbance in the blood supply (Garosi et al, 2006).

A cerebrovascular accident, also called a stroke, is the sudden onset of neurological dysfunction resulting from an intracranial vascular event. There are two main types of stroke: ischaemic (caused by arterial or venous obstruction) and haemorrhagic (caused by the rupture of intracranial vessels).

The clinical signs are variable and will reflect their localisation within the neuroaxis. For the purposes of this review, patients would present with signs of central vestibular disease associated with brainstem, cerebellar or thalamic dysfunction (Garosi et al, 2006; Gonçalves et al, 2010). The onset of the clinical signs is typically acute and they are non-progressive after the initial 24 hours, although occasionally there is mild worsening of the neurological deficits within the first few hours. When the clinical signs last less than 24 hours, the event is called a transient ischaemic attack (TIA).

Diagnosis

Diagnosis requires the use of MRI, which shows well-defined, sharply demarcated lesions limited to the territory of a main cerebral or perforating artery ([Figures 2a](#) and [2b](#)).

A concurrent medical condition is identified in approximately half of cases, with chronic kidney disease and hyperadrenocorticism among the most common. Certain breeds, such as the cavalier King Charles spaniel and the greyhound, appear to be over-represented in the literature (Garosi et al, 2005).

Baseline diagnostic investigation should include haematology, serum biochemistry and coagulation profiles, urinalysis (including a urine protein: creatinine ratio), thyroid and adrenal function tests and blood pressure measurements.

Echocardiography and blood cultures may be indicated if a septic embolus is suspected. Thoracic radiography and abdominal ultrasound are valuable in excluding neoplastic disease. Faecal analysis may also be performed to rule out *Angiostrongylus vasorum*.

Treatment is mainly supportive and directed at the underlying disease in cases where this is identified. Prognosis is fair to good, with most dogs making a full recovery within weeks. However, cases with concurrent medical conditions have shorter survival times and are more likely to suffer recurrences.

Neoplasia

Tumours can cause signs of either peripheral or central vestibular disease depending on their location (Snyder et al, 2006, 2008).

Neoplasia of the ear canal and tympanic bulla is often associated with signs of chronic otitis that does not respond or that only responds partially to treatment. Signs may be seen such as a head tilt, facial paralysis, Horner's syndrome, neurogenic dry eye and pain opening the mouth. Possible types of tumour include squamous cell carcinomas, fibrosarcomas, osteosarcomas ([Figure 3a](#)) and adenocarcinomas (of the ceruminous or sebaceous glands). Diagnosis usually requires imaging and histopathology. Prognosis is usually poor, but if diagnosed early, radical resection may be effective.

Neoplasia causing signs of central vestibular disease is usually located either in the brainstem or the cerebellum and the signs will reflect the location, as discussed in the previous article. Most common types of tumour include meningiomas ([Figure 3b](#)), gliomas and choroid plexus tumours.

Prognosis is also poor in these cases, as surgical access to this region is limited, with the main therapeutic option being radiotherapy.

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