Vestibular syndrome in dogs

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Introduction

Failures of the vestibular system are among the more frequent neurological presentations in small animal practice. They can be regarded as clinically rewarding in that the neurological loss of function produces classical clinical signs, and initial recognition of most vestibular disorders usually poses no great difficulty. The next, perhaps more important, step is precise localization of the disease within the vestibular system; assessing the lesion as either peripheral (in the inner ear) or central (in the brain) may be a challenge. However, precise localization is essential, as this affects all subsequent decisions regarding diagnosis, prognosis and therapy, and the key question with "vestibular neurology" is therefore always: ear or brain? The answer is of fundamental importance for the patient, and this article aims to enable the reader to recognize vestibular disease, to distinguish between peripheral and central syndromes in a clinical setting, and to consider differential diagnoses for disorders of the peripheral vestibular system. Central vestibular disease will not be discussed in depth, as once this disorder has been recognized it is usually necessary to transfer the patient to a specialist center for MRI imaging and cerebrospinal fluid analysis.

KEY POINTS

- **Typical signs of a vestibular system disorder are generalized ataxia, nystagmus, head tilt and walking in circles.**
- **The clinician must determine if the peripheral (ear) or central (brain) section of the vestibular system is involved, as this is essential for all further diagnostic and therapeutic decisions.**
- **A combination of vestibular symptoms and unilateral facial nerve paralysis and/or Horner's syndrome without other neurological deficits invariably indicates middle or inner ear disease.**
- **Vertical nystagmus almost invariably indicates a central vestibular disorder.**
- **Peripheral vestibular disease may be diagnosed and treated by the general practitioner. Patients only require referral to a specialist for further diagnostic imaging when initial therapy fails.**

Structure and function

The vestibular system is made up of peripheral and central parts. The peripheral section consists of a receptor organ in the inner ear and the vestibular nerve. The central section, which processes information from the inner ear, is formed by the vestibular nuclei in the brainstem and certain regions within the cerebellum (the flocculonodular lobe and fastigial nucleus). Part of the cerebellum (the vestibulocerebellum) tends to have an inhibitory influence on the vestibular system, an important point when considering the pathological changes involved in vestibular disease.

These areas of the brain are linked through different pathways to their effector organs, the extraocular eye muscles and the musculature of the neck and limbs, allowing the vestibular system to fulfill the following functions:

- To respond to the effects of gravity
- To maintain balance during movement or weight transfer
- To co-ordinate head and eye movements

Two scenarios allow consideration of vestibular system physiology and facilitate understanding of the pathophysiological process:

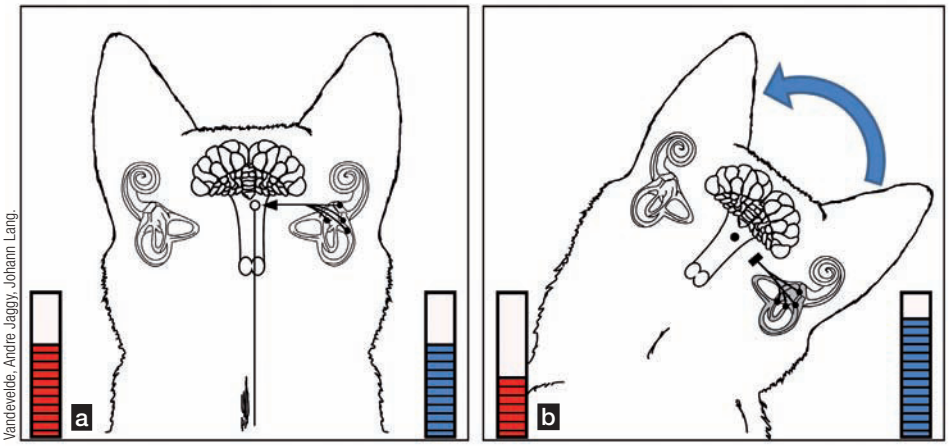

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Figure 1. Schematic diagram of the vestibular system's reaction to altered head position in a normal animal. **(a)** The vestibular system receives equal input from both inner ears when the head is upright. **(b)** When the animal tilts its head, the vestibular system receives relatively more input from the dependant side. The brain perceives this and responds by lifting the head accordingly.

- Maintenance of balance: the left and right vestibular systems continually send impulses to the brain. If the animal is in a level, upright position, then the impulse frequency from both sides is equal. However, if the head (or entire body) is tilted to one side, then the impulse frequency from the dependent side increases, so that the brain now receives more input from this side. Based on the difference in inputs, the brain recognizes the tilt and responds by straightening the body, *i.e.,* towards the side with less input ***(Figure 1)***.

- Co-ordination of head and eye movement: co-ordinated extraocular eye muscle movements are necessary to ensure that an animal perceives its surroundings in sharp detail during head movements. If the eyes were to follow every head movement, the brain would be incapable of processing the enormous number of images generated, and a blurred visual picture (similar to that seen from the window of a moving train) would result. The body's solution for this problem is as follows: if the head moves to one side, the eyes do not immediately move with the head but rather briefly fix on an image which is "recorded" and processed, before the eyes make a quick movement to "catch up" with the head. This is repeated as necessary – the eyes frequently stopping to take single "pictures" before moving to follow the head. In this way "single impressions" are formed which can be processed by the brain. These discrete, jerky eye movements are known as physiological nystagmus, and can be tested for by moving the patient's head while the animal is stationary; smaller animals can be picked up and turned rapidly left and right in front of the examiner in order to observe the response. The question is therefore; how does the brain know that the animal has moved its head to one side? The principle of input difference from left and right inner ears also applies here. If the animal shifts its head to the left, the left inner ear is stimulated and the impulse frequency increases in comparison to the right side. This allows the brain to recognize that the head is moving to the left, and it initiates the rhythmic eye movement in the direction of the head movement, with the fast-jerk phase towards the side with the higher input and away from the side with the lower input.

Failure of the vestibular system

Clinical symptoms of a lesion in the vestibular system, regardless of whether it is peripheral or central in origin, are as follows:

- Generalized ataxia
- Nystagmus
- Head tilt
- Circling
- Falling to one side +/- rolling

Vestibular ataxia is, in contrast to ataxia caused by spinal disease, characterized by a wide-based stance. This ataxia is often accentuated on one side, whereby the animal may lunge sideways in order to steady itself. Occasionally, the ataxia becomes so pronounced that the animal is incapable of walking and falls sideways when trying to take a step; in extreme cases it may even roll over onto the floor. As most vestibular losses of function are unilateral, the animal often walks in tight circles. In addition, the animal presents with a head tilt, with the affected side lower than the healthy one. The obvious symptoms of loss of function are merely physiological reactions from pathological input into the brain. As noted above, a reduced input from one side is interpreted by the brain as a shift in the body's weight to the side with more input and it corrects this shift, so that the head tilts to the side with less input in order to re-establish balance. In pathological cases this means the head is tilted

erroneously towards the diseased side, because this is delivering less input to the brain ***(Figure 2)***.

Another major sign of all vestibular disorders is pathological nystagmus; this can be observed with the patient stationary and the head immobile. Nystagmus by definition is an involuntary rhythmic movement of the eyeballs with slow and fast phases, and is classified by the direction of the fast phase. The slow phase is generally towards the "pathological" incident, while the fast phase represents the corrective re-compensation. Nystagmus is also classified according to the direction of eye movement (horizontal, rotatory or vertical), so if a dog demonstrates fast eye movement to the left, this is known as left horizontal nystagmus.

Horizontal nystagmus can be explained similarly to head tilting and is also the brain's "normal" reaction to faulty input. The lack of input from the vestibular system on one side leads to a relatively increased input from the healthy side; this is interpreted by the brain as a "head movement" to the higher input side, so that – although the head does not in fact move at all – a "physiological nystagmus" is initiated. Note that the fast phase of the horizontal nystagmus occurs away from the lesion and towards the direction of the supposed head movement (*i.e.*, towards the side with more input). An *aide memoire* is that a normal defense mechanism is to "quickly escape from a problem".

If an animal shows nystagmus which does not alter with changes in the head position, this is known as static pathological nystagmus. Positional nystagmus presents only with a change of posture (*e.g.*, when the animal is rolled onto its back) or if the type of nystagmus changes with an alteration in posture. A ventral or ventrolateral positional strabismus ***(Figure 3)*** may also be observed if the clinician extends the dog's neck. In cases of mild vestibular losses of function, this strabismus may be the only conspicuous finding on neurological examination (1).

It should be emphasized that vertical nystagmus invariably (and positional nystagmus frequently) indicates a central vestibular dysfunction.

If there is bilateral involvement of the vestibular system, one-sided functional abnormalities such as walking in circles and head tilt are not noted. Affected animals demonstrate searching, weaving head movements and physiological nystagmus is absent, *i.e.*, there is no rhythmic eye movement when the head moves; instead

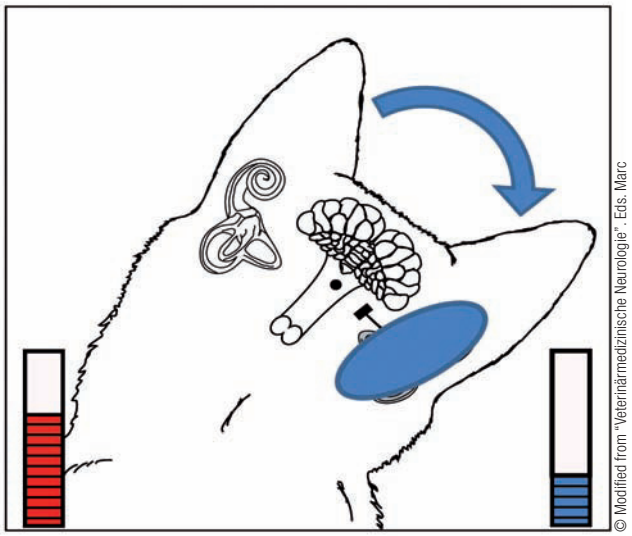

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Figure 2. Schematic diagram of the pathophysiology of head tilt. In diseases of the inner ear, the brain receives more input from the healthy side, which it falsely interprets as a head tilt towards the healthy side. The brain reacts to this apparent tilt by correcting the head so that the head is repositioned towards to the other, diseased side. Therefore the head tilt always occurs towards the diseased side.

the eyes move at the same time as the head. Bilateral vestibular syndrome in patients that retain normal levels of awareness and maintain the ability to walk are, as a general rule, peripheral in nature.

Central or peripheral – brain or ear?

In diseases of the peripheral vestibular system, the brain is not affected and so no additional neurological deficit is found on examination that would indicate involvement of the brain; patients show normal awareness, unimpaired vision, normal cranial nerve function, normal proprioceptive paw positioning and no limb paresis. There are two exceptions to this, as two nerves (the facial nerve and the sympathetic nerve) may be involved with a peripheral vestibular disorder. Parts of both nerves run through the middle ear or rest against the wall of the middle ear, and can be implicated in inner and middle ear disease. Involvement of the facial nerve, which runs through a channel in the wall of the middle ear (2) and which is only partially separated from the middle ear lumen by a thin membrane, causes paralysis of the facial musculature, visualized by drooping of the ear and lip ***(Figure 4)***, an enlarged palpebral fissure, a narrowed nostril, loss of the palpebral reflex and menace response. Involvement of the sympathetic fibers (which run through the middle ear to the eye) causes Horner's syndrome: ptosis (reduced eyelid opening), miosis (small pupil), nictitating membrane prolapse and enophthalmos. Other than signs related to

these two nerves, no other cranial nerve deficits should be observed with a peripheral vestibular syndrome. The inverse also applies: a vestibular syndrome with simultaneous facial paralysis and/or Horner's syndrome without other neurological deficits almost always indicates a peripheral lesion. If further cranial nerve signs develop, or there are other neurological deficits present which can be localized to the brain (limited awareness, limb paresis, diminished proprioceptive paw positioning, tremors, other cranial nerve deficits...), then this points to a central lesion.

As noted above, a vertical nystagmus invariably, and a positional nystagmus frequently, indicates a central lesion, whereas horizontal or rotatory nystagmus does not allow differentiation between a peripheral or central lesion. However, nystagmus frequency may assist with lesion localization; high frequencies (≥ 66 beats/minute) are more common with lesions of the peripheral vestibular system (3). Remember that (as noted above), an animal with bilateral peripheral vestibular disease can walk without circling and will show no physiological nystagmus or head tilt, only ataxia and searching head movements.

A paradoxical vestibular syndrome, which may occur with lesions of the central vestibular system, is also recognized. Here the classic symptoms of head tilt, circling and nystagmus do not correlate, in that an animal may circle, have a head tilt to the right, and show left horizontal nystagmus (which all suggest that the right side is affected), whilst diminished or absent proprioceptive paw positioning in both fore and hind left legs suggests a left-sided lesion. This paradox can be explained pathophysiologically as affected animals have a lesion in the angle between the caudal cerebellum and the brainstem, so that brainstem function (responsible for the proprioceptive paw positioning), the part of the cerebellum that influences the vestibular system (the flocculonodular lobe), and/or its connections to the brainstem (the caudal cerebellar peduncle) are affected. Remember that the cerebellum's output, which influences the vestibular system, is almost always inhibitory; if this inhibitory influence fails on the diseased side, this side now paradoxically gives a higher input into the brain, which is misinterpreted as the healthy side delivering less input. With a paradoxical vestibular disorder the head tilt and nystagmus are misleading, and it is the absent proprioceptive reflexes which accurately indicate the diseased side. The diagnostic importance of a paradoxical vestibular syndrome is that it is always caused by a lesion within the brain. The differential signs are summarized in ***Table 1***.

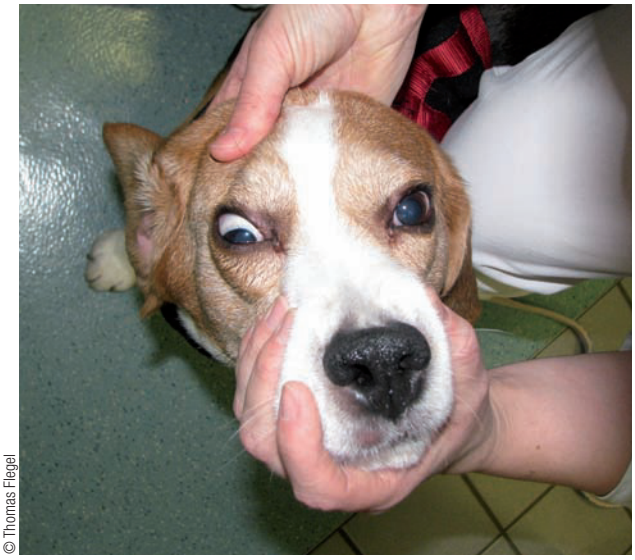
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Figure 3. A Beagle with a ventral strabismus of the right eye caused by vestibular syndrome; the strabismus is best seen by extending the neck.

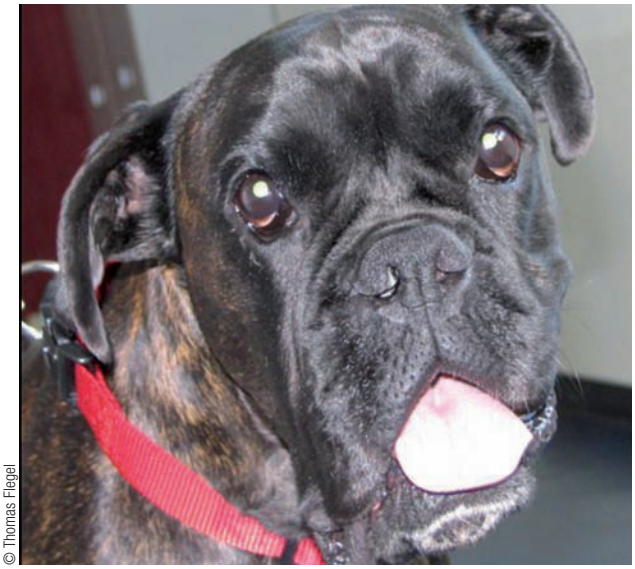
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Figure 4. A Boxer with right-sided peripheral vestibular syndrome. Note the head tilt to the right, signs of right-sided facial nerve paresis (drooping lip and ear), and an enlarged palpebral fissure.

Differential diagnoses of peripheral vestibular disease

The above section should enable the veterinarian to distinguish a peripheral lesion of the vestibular system from a central one. ***Table 2*** lists the most frequent differential diagnoses of peripheral and central causes of vestibular

Table 1. Differentiation of central (brainstem, cerebellum) or peripheral (inner ear) vestibular syndrome.

Sign	Peripheral	Central
Behavior	normal	abnormal
Awareness	normal	abnormal
Nystagmus	horizontal or rotatory	horizontal, rotatory or vertical
Nystagmus frequency	higher	lower
Cranial nerve deficits	facial nerve deficits Horner's syndrome	multiple cranial nerve deficits
Proprioceptive reflexes	normal	reduced
Motor function	normal	paresis, plegia
Paradoxical signs	not observed	possible
Ventral/ventrolateral strabismus	possible	possible

Table 2. Common etiologies of peripheral and central vestibular syndromes, in order of frequency.

Peripheral vestibular syndrome
• Otitis interna • Idiopathic vestibular syndrome • Hypothyroidism • Traumatic perforation of the eardrum with secondary otitis media/interna • Toxins: *e.g.*, - disinfectants: chlorhexidine - antibiotics: aminoglycosides, fluoroquinolones - heavy metals • Neoplasia • Head injury with fracture of the petrosal bone • Congenital vestibular syndrome (Akita Inu, Beagle, Cocker Spaniel, German Shepherd, Doberman, Tibetan Terrier) • Neuritis of the vestibulocochlear nerve
Central vestibular syndrome
• Encephalitis (non-infectious, infectious, expansion of otitis media/interna into the brain) • Infarction • Neoplasia • Intoxication (*e.g.*, metronidazole) • Skull-brain trauma (hemorrhage, contusion) • Enzyme storage disease

syndrome. All general practitioners should be able to diagnose peripheral vestibular disease, and a short discussion on the three most common etiologies of peripheral vestibular syndrome is worthwhile. It is the author's opinion that animals with a central lesion should always be referred to a specialist.

Otitis interna

Otitis media/interna is the underlying etiology in approximately 50% of patients with peripheral vestibular disease (4). Otitis media by itself does not cause loss of vestibular function, but the anatomical proximity of middle and inner ear means that otitis media is often associated with otitis interna. By comparison, it is rare to have otitis interna without involvement of the middle ear, so when otitis media occurs with simultaneous peripheral vestibular failure, this indicates that the inner ear is involved in the inflammatory process. Note that it is not inevitable that otitis externa will also be present, since infection can reach the middle and inner ear through hematogenous spread or by ascending from the pharynx through the Eustachian tube into the middle ear.

Aside from peripheral vestibular failures, otitis interna/media can, as noted above, involve two other nerves; the facial nerve, lying within the wall of the tympanic bulla, and fibers of the sympathetic nerve, running through the middle ear to the eye. Hence an otitis media/ interna can lead to a facial paralysis and Horner's syndrome; other cranial nerves should not be affected.

On the basis of the frequent causal relationship between middle and inner ear inflammation as well as otitis