

# Vestibular Diseases in Dogs - Part 1

By Dr. Filippo Adamo, DVM, Dipl. ECVN, Veterinary Neurology Specialist  
President Bay Area VNC (Veterinary Neurology Neurosurgery Consulting)  
208 Santa Clara Way, San Mateo, California, USA 944033 (608)334-3713



Fippadamo@yahoo.com

www.bayreavnn.com



Typical posture of a dog affected by vestibular disease (a). MRI pictures of dogs affected by: central vestibular disease secondary to a cerebellar infarct (b); meningioma (red arrow) with a cystic component (yellow arrow) (c); and fungal infection (d). Peripheral vestibular disease secondary to a chronic otitis media/interna (e)

## INTRODUCTION

The function of the vestibular system is to maintain balance and normal head and eyes position. Dysfunction of this apparatus cause vestibular signs. 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.

## ANATOMY

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.

## VESTIBULAR SIGNS

Head tilt, asymmetric ataxia, circling (usually tight circle), falling, leaning, drifting to one side, nystagmus (spontaneous or positional), positional strabismus (deviation of one eye in some head position, nausea, vomiting). Vestibular disease produces varying degrees of loss of equilibrium causing imbalance and ataxia. Strength is not affected, 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.

**Nystagmus.** Disturbed vestibular input to the neurons innervating extra-ocular eye muscles results in abnormal nystagmus. Nystagmus probably occurs at some time during all types of vestibular disease. Nystagmus is an involuntary rhythmic oscillation of the eyeball that nearly always affects both eyes equally. Typically, nystagmus consists of a slow phase in one direction and a fast phase in the other. It is

customary to describe nystagmus in terms of the fast phase despite the fact that in most cases the slow phase will be directed towards the affected side. Nystagmus tends to occur early in the course of peripheral vestibular disease, and to disappear later.

- Physiological nystagmus may be induced in normal animals. It occurs with normal turning of the head from side to side, or up and down (vestibular in origin), or after rotation (post-rotational nystagmus).
- Spontaneous nystagmus. If nystagmus occurs when the head is stationary. Spontaneous nystagmus is usually pathological in origin and may be horizontal, rotatory, or vertical in direction.
- Positional nystagmus. If nystagmus occurs only when the head is placed in an unusual position (e.g. laterally or dorsally), it is known as positional nystagmus.

**Abnormal Posture and Ataxia.** Loss of co-ordination between head, trunk, and limbs, results in loss of balance. This may result in a head tilt. The trunk may fall, or even roll, to one side. The trunk may be flexed laterally. Animals tend to circle. These are usually circles with a small radius. It may be possible to elicit mild hypertonia and hyperreflexia in the limbs on one side. 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 a vestibular lesion will accentuate the clinical signs.

**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 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 towards 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 towards 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.** Bilateral peripheral vestibular disease with complete loss of function is characterized by symmetrical ataxia and loss of balance of either side, with strength preserved. There is no postural asymmetry. A characteristic "side-to-side" head movement (Ray Charles-like) often accompanies these signs. Abnormal nystagmus is not observed, and with bilateral destruction of the receptor organs, normal

vestibular nystagmus cannot be elicited by head movement or caloric testing.

**Peripheral Vestibular Disease.** Peripheral lesions involve the middle and inner ear. Middle ear (bulla ossea) lesions usually produce head tilt (ipsilateral to the lesion) only, in the absence of other signs. Horizontal or rotatory nystagmus also may be seen. 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, asymmetrical ataxia. Horner's syndrome (miosis, ptosis, enophthalmos) of the ipsilateral eye may be present with either middle or inner ear disease 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.

**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 Vth cranial nerves, are also indicative of central disease. Nystagmus may be a key to differentiating central from peripheral disease. The nystagmus may vary in direction with change in head position. Vertical nystagmus in any head position is most consistent with central vestibular disease.

#### NEUROLOGICAL EXAMINATION:

The goal of the neurological examination is to differentiate a Peripheral vs a Central Vestibular diseases. This is of paramount importance since the list of the differential diagnosis (the most common causes responsible for the patient clinical signs), the diagnostic work up and the prognosis is different.

#### DIAGNOSTIC STEPS

A minimum database should be performed on all dogs which should include haematology, serum chemistry, urinalysis and thoracic radiographs. If toxicity is suspected due to antibiotic administration (metronidazole and aminoglycosides), immediate withdrawal of the medication is recommended. In most cases, there is resolution of the vestibular abnormality. Ancillary Test for patients with peripheral disease include: otoscopic examination (usually under sedation or general anesthesia); Radiography of tympanic bulla (under sedation or general anesthesia), however there are many false negative when using radiography; computed tomography (CT), or magnetic resonance (MR) imaging; and thyroid function testing. If abnormal material is seen within the tympanic bulla, this could be collected through myringotomy (needle aspirate through the ear drum) and submitted for cytology and bacteriology analysis. Diagnosis of central vestibular disease is based upon CT or MR imaging of the brain and analysis of cerebrospinal fluid (CSF).

#### I) PERIPHERAL VESTIBULAR DISEASES

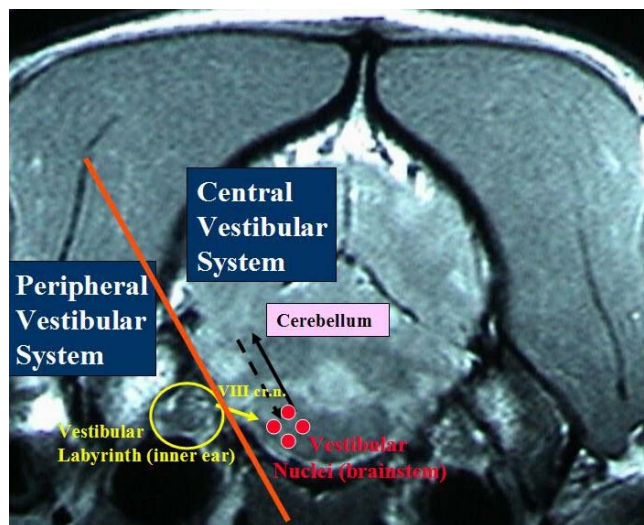
a) **Idiopathic Vestibular Disease.** Very common: in a review of 83 cases of peripheral vestibular diseases in dogs 39% were idiopathic. This is an acute vestibular syndrome of older dogs. There is no evidence of inflammatory disease in affected animals. Affected dogs have signs of peripheral vestibular involvement. Vomiting is occasionally seen. The signs appear suddenly, and often result in severe dysfunction and inability to stand and walk. 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. 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, in the presence of normal tympanic membranes, and normal radiographs of the temporal bones, further support this diagnosis. The cause of this idiopathic disorder remains undetermined.

**Prognosis:** for spontaneous recovery is good; however, recovery may require 2-4 weeks. Re-occurrence may be seen, especially in dogs, either on the same or the opposite side. There is no evidence that treatment of any type alters the course of the disease.

**Therapy:** Supportive care and Meclizine Hydrochloride or Cerenia during the first 24-48 hrs to control dizziness and vomiting

**Pathophysiology:** unknown, suspected osmolality disturbance of the fluid within the vestibular labyrinth.



Transverse MRI of the dog brain showing the schematic representation of the anatomic structures of the peripheral and central vestibular system.

Part 2 of this article will be in the next magazine (Issue 4 of 2010).

**About the author:** Dr. Adamo is European Board Certified Veterinary Neurologist and Neurosurgeon. During the last 20 years, he built up his neurological experience in both academia and private practice in both Europe and USA. Dr. Adamo has been a Board Certified Neurologist since 1997. Prior to moving to California, Dr. Adamo served from 2002 to 2007 as Clinical Assistant Professor in Neurology at the School of Veterinary Medicine, University of Wisconsin. He then moved with his family to California where he served as Chief of Neurology at the Bay Area Veterinary Specialists in San Leandro until July 2009. As a chief of neurology at the University of Wisconsin, he developed many years of experiences in research and clinical neurology/neurosurgery. Dr. Adamo developed an alternative cyclosporine medical treatment for Granulomatous meningoencephalomyelitis (GME) in dogs, designed a frameless guided stereotactic CT guided brain biopsy, investigated alternative medical therapy for brain meningioma in dogs and cats, and developed and tested the first artificial disc for the canine cervical spine. He has lectured and published extensively in the United States and Europe. His main areas of interest are brain inflammatory diseases, and brain and cervical spinal surgery. When not spending time with his son, and their dog (Pancio), Dr. Adamo enjoys playing Brazilian rhythm music and Aikido.