ACUTE HEAD TILTS IN DOGS AND CATS

Simon R. Platt

BVM&S FRCVS DACVIM (Neurology) DECVN

College of Veterinary Medicine, University of Georgia

Abstract: In this session we will review the signs of vestibular dysfunction and how to determine if the cause is affecting the peripheral or central nervous system. We will review the causes and describe diagnostic and therapeutic approaches to these cases.

Keywords: Vestibular, head tilt, nystagmus, ataxia, idiopathic

The vestibular system is essential in maintaining balance and preventing the animal falling over by keeping and adapting the position of the eyes, head and body with respect to gravity. It is therefore not surprising that disease of the vestibular system results in some of the most dramatic and distressing neurological signs. Head tilt, falling, rolling, leaning, circling, abnormal nystagmus and ataxia commonly result. Clinical signs of vestibular disease may be a result of lesions involving either the receptor organs in the inner ear or the vestibular portion of the eighth cranial nerve (i.e., peripheral vestibular disease) or lesions involving the brainstem vestibular nuclei or vestibular centers in the cerebellum (i.e., central vestibular disease). This session reviews the clinical approach of an animal with a vestibular disorder.

VESTIBULAR DISORDERS: CLINICAL SIGNS

Vestibular disorders (VD) are common in dogs and cats and may result in any or all of the following clinical signs: head tilt, falling, rolling, leaning, circling, abnormal nystagmus, positional strabismus, and ataxia.

- **Head tilt** often indicates a VD. This abnormal head posture is characterized by a rotation of the median plane of the head (one ear is held lower than the other). It occurs in VD as a result of the loss of antigravity muscle tone on one side of the neck. It must be differentiated from a head turn where the median plane of the head remains perpendicular to the ground but the nose is turned to one side. Such head turn is usually associated with a body turn. A head turn does not indicate a vestibular disorder and is usually toward the side of a forebrain lesion.
- **Circling** may occur in conjunction with VD as well as an asymmetrical or focal lesion in the forebrain. Tight circles are usually but not exclusively associated with a VD, while wide circles are often associated with a forebrain lesion.
- **Nystagmus** is an involuntary rhythmic movement of the eyeballs. Physiologic nystagmus is nystagmus that occurs in normal animals while pathologic nystagmus reflects an underlying VD. The direction of the nystagmus is typically defined by the direction of the fast phase. Physiologic nystagmus can be induced in the normal animal by rotating the head from side to side (oculo-vestibular reflex). It is characterized by a slow phase in the opposite direction of the head movement and a fast compensatory phase in the same direction as the head rotation. Physiologic nystagmus can be depressed in animal with unilateral VD or absent in animal with bilateral VD. Pathologic nystagmus can be either spontaneous (observed when the head is in a normal position at rest) and/or positional (that which occurs, or is altered in character, intensity or direction, with alteration in the position of the head, for example by placing the

animal upside down on its back). Nystagmus is usually classified on the basis of its direction and may be horizontal, vertical or rotatory and may change in direction on changing position of the head. The fast phase of pathologic nystagmus is typically directed away from the side of the vestibular lesion.

- **Strabismus** refers to an abnormal position of the globes. Strabismus can be seen in VD when the head is placed in an abnormal position (extended dorsally or the animal placed upside down on its back). VD often causes a ventral or ventrolateral positional strabismus in the eye on the same side as the vestibular lesion.
- **Ataxia** is defined as an uncoordinated gait and can be caused by a vestibular disorder (vestibular ataxia), a cerebellar disorder (cerebellar ataxia) or a peripheral nerve, spinal cord or brainstem disorder (proprioceptive or sensory ataxia). Vestibular lesions often cause ataxia characterized by swaying of the trunk and head, base-wide stance, leaning, falling and rolling to one side with unilateral lesion. With bilateral VD, affected animals tend to fall to both sides and often show wide excursion of the head from side to side.

VESTIBULAR DISORDERS: LOCALIZING THE LESION

Clinical signs of VD may be a result of lesions involving the receptor organs in the inner ear or the vestibular portion of the eighth cranial nerve running in the petrous part of the temporal bone (i.e., peripheral VD) or lesions involving the brainstem vestibular nuclei (i.e., central VD).

- **Peripheral or central?**

Most lesions affect a region, rather than a specific nerve or nucleus, so accompanying neurologic abnormalities can often be used to localize the lesion to the peripheral or central vestibular

system (Table 1). Both peripheral and central VD can cause a head tilt, horizontal or rotatory nystagmus, and ataxia. Facial paralysis and Horner's syndrome can be seen with peripheral VD due to the proximity of cranial nerve VII (facial nerve) and the sympathetic nerve supply to the eye to the vestibular nerve in the region of the petrous temporal bone. Correctly identifying central VD requires identification of clinical signs that cannot be attributed to diseases of the peripheral vestibular system. Lesions that affect the central vestibular system typically have additional clinical signs suggestive of brainstem involvement. Such lesions often involve the reticular formation as well as ascending and descending motor and sensory pathways to the ipsilateral limbs. Therefore, abnormal mental status, ipsilateral paresis, and conscious proprioceptive deficits are commonly associated with central VD. Deficits of cranial nerves V through XII can also be associated with central VD. The presence of spontaneous or positional jerk nystagmus indicates vestibular dysfunction but does not further localize the lesion to the peripheral or central vestibular system. However, vertical nystagmus and nystagmus that changes in direction on changing position of the head are a feature of central vestibular lesions. Rate of nystagmus (number of beats per minute with the head in a neutral position as well as with the animal in dorsal recumbency) can further assist with differentiation between central VD from peripheral VD. Median rate of resting and positional nystagmus appears to be significantly faster for dogs with peripheral VD with a resting nystagmus ≥ 66 beats per minute providing the highest combined sensitivity and specificity in diagnosing peripheral VD. With peripheral and central VD, the head is usually tilted in the direction of the lesion. With paradoxical VD, however, the head is usually tilted opposite to the direction of the lesion.

	Peripheral	**Central**

Head tilt	Ipsilateral	Ipsilateral (or contralateral in case of paradoxical VD)
Asymmetrical ataxia	Ipsilateral	Ipsilateral
Nystagmus • Horizontal • Rotatory • Vertical • Positional	 Ipsilateral Ipsilateral No No (except during the early phase of recovery)	 Ipsilateral or contralateral Ipsilateral or contralateral Yes Yes
Postural reaction deficit	No	Yes (always ipsilateral to the lesion)
Abnormal mental status	No	Possible
Circling	Ipsilateral	Ipsilateral or contralateral

Table 1. Clinical findings associated with peripheral and central vestibular disease

Occasionally, intracranial lesions can result in signs suggestive of a peripheral lesion. Although animals with a peripheral VD have a normal level of consciousness and no evidence of weakness or postural reaction deficits, the absence of these signs does not rule-out the presence of a central VD. If in doubt about the localization of the lesion, the clinician should evaluate the animal for central VD as well as peripheral VD. In very rare cases, VD may be part of a diffuse polyneuropathy or cranial polyneuropathy. Other cranial nerve dysfunction such as dysphagia,

tongue weakness, jaw weakness and/or facial paralysis as well as limb weakness with depressed segmental spinal reflexes may be seen. Note also that lesions of the thalamus and/or extrapyramidal basal nuclei may also cause abnormal head posture and signs of central VD.

- **Ipsilateral, contralateral or bilateral lesion?**

With both central and peripheral VD, the head tilt, circling and nystagmus typically occur ipsilateral to the side of the lesion. Less frequently, lesions affecting the caudal cerebellar peduncle, the fastigial nucleus, or the flocculonodular lobes of the cerebellum can cause central VD with a resulting paradoxical head tilt. This syndrome is called paradoxical because the head tilt and circling occur contralateral to the side of the lesion. Bilateral VD is characterized by head sway from side to side, loss of balance on both sides and symmetrical ataxia with a wide-based stance. A physiological nystagmus usually cannot be elicited and a head tilt is not observed.

DIFFERENTIAL DIAGNOSIS OF VETSIBULAR DISORDERS

A lesion must be localized to a particular section of the vestibular apparatus before an appropriate differential diagnosis can be established and further test conducted. The formation of a differential diagnosis list is essential in choosing and interpreting any diagnostic test. Diseases affecting the nervous system are classically classified in disease processes using the mnemonic VITAMIN D.

Disease mechanism	**Peripheral vestibular disease**	**Central vestibular disease**
Vascular		Brain infarct

		Brain hemorrhage
Inflammatory/ **I**nfectious	Otitis media/interna Nasopharyngeal polyps	Infectious encephalitis Meningo-encephalitis of unknown etiology
Trauma	Head trauma	Head trauma
Toxic	Aminoglycosides, topical chlorhexidine	Metronidazole
Anomalous	Congenital vestibular disease	Intracranial intra-arachnoid cyst, hydrocephalus, Chiari-like malformation
Metabolic	Hypothyroidism	
Idiopathic	Idiopathic vestibular disease	
Neoplastic	Middle / inner ear tumour	Primary or metastatic tumor
Nutritional		Thiamine deficiency
Degenerative		Neurodegenerative disease

Idiopathic vestibular syndrome is common in adult cats and dogs (often geriatric). Clinical signs are usually peracute and initially severe with affected animal appearing extremely disable in the first 48 to 72 hours. If facial nerve paralysis or Horner's syndrome (miosis, enophthalmia, protrusion of third eyelid, ptosis of upper eyelid) is also present then other differentials should be considered. Diagnosis is based on the presence of compatible history and exclusion of other causes of peripheral VD. Most animals tend to improve over 1 to 3 weeks period and often return to normal. However, some animal may be left with a permanent head tilt or episodic ataxia. No treatment has proved beneficial and recurrence is possible.

Otitis media/interna can be secondary to otitis externa, oropharyngeal infection (spreading via the auditory tube) or hematogenous spreading. Therefore, the absence of sign of otitis externa does not rule-out the presence of otitis media/interna. Clinical course can be acute or progressive. Signs of VD caused by otitis media/interna are often associated with ipsilateral facial nerve paralysis and/or Horner's syndrome. Diagnosis is made by otoscopic examination and imaging studies (bulla radiographs, CT or MR scan) and/or exclusion of other causes of peripheral VD. If fluid is visualized within the middle ear then attempt should be made to obtain a sample via myringotomy for cytology and bacterial culture. Treatment of otitis media/interna consists of systemic antibiotic for a minimum of four to six weeks (oral amoxicillin/clavulanate, fluoroquinolone or cephalosporin if no culture can be obtained following myringotomy). Surgical drainage and debridement via bulla osteotomy should be considered in case of failure of medical treatment. Prognosis is guarded to fair as some animal may be left with permanent head tilt and/or facial paralysis.

Tumors of the caudal fossa and meningo-encephalitis of unknown etiology (MUE) such as granulomatous meningo-encephalitis (GME) are the two most common causes of central VD. Common types of tumors found in the caudal fossa include meningioma and choroid plexus tumor both which have a tendency to arise at the level of the emergence of the vestibulo-cochlear nerve at the cerebellomedullary angle. Less common tumors include glioma, ependymoma or medulloblastoma. Signs of VD associated with these tumors are often slowly progressive. Presumptive diagnosis is made by advance imaging (CT or MRI) but the exact type of tumor can only be confirmed histologically (either by surgical tissue biopsy or post-mortem). Prognosis is fair for surgically accessible cerebellomedullary angle meningioma. Prognosis is more guarded for other tumors.

Meningo-encephalitis of unknown etiology (MUE) is often attributed to GME, the diagnosis of which can only be confirmed on histopathology. Clinical signs can be acute or progressive in onset. Neurolocalisation often suggests multifocal involvement but can occasionally be focal. A presumptive diagnosis can be made based on a consistent history, clinical signs, signalment (frequently young to middle-aged female terrier breeds), multifocal, contrast-enhancing lesions on MRI, CSF analysis (pure mononuclear pleocytosis or a mixed cell population) and exclusion of infectious aetiologies on serological or PCR tests (mostly Distemper, Toxoplasma and Neospora). Immunosuppressive doses of corticosteroids have been the mainstay of treatment for presumptive GME. Other immunomodulatory drugs such as azathioprine, procarbazine, cytosine arabinoside and cyclosporine as sole agent or as an adjunctive treatment with prednisone have been reported to be effective in some dogs. Overall, the prognosis is guarded but survival times range from weeks to years.

NEURODIAGNOSTIC INVESTIGATIONS OF VESTIBULAR DISORDER

The choice of neurodiagnostic tests in patient with VD depend essentially on where the lesion is suspected on the basis of the neurological examination. If in doubt about the localization of the lesion, the animal should be evaluated for both peripheral and central VD.

- **Peripheral Vestibular Disease**

Diagnostic plan for patients with signs suggestive of peripheral VD include at least otoscopic and pharyngeal examination, imaging of the tympanic bullae with radiographs, computed tomography (CT) or magnetic resonance imaging (MRI) and thyroid function testing. Middle ear pathology should be suspected if the tympanic membrane is ruptured, bulging, cloudy or red in color on otoscopy. If the tympanic membrane is ruptured, swabs for cytology and culture (aerobic, fungal and yeast) can be taken directly from the middle ear. If the tympanic membrane

is intact but bulging or of an abnormal colour, a small hole (myringotomy) can be made in the tympanic membrane with a 20-gauge spinal needle to obtain samples for cytology and culture. Additionally, the middle ear cavity can be flushed by attaching a 10 to 20 cc syringe of warm saline to the spinal needle. Warm water is flushed into the tympanic cavity and gently suctioned. The resulting fluid can then be submitted for cytology and culture. Radiographic evaluation of the tympanic bullae requires general anesthesia to allow adequate positioning. Four radiographic projections are classically used (dorsoventral, latero-lateral, latero 20° ventral-laterodorsal and rostro 30° ventral-caudodorsal open-mouth projection). Although positive radiographs can be seen as highly specific in the diagnosis of middle ear disease, negative radiographs do not rule out the presence of middle ear disease. CT and MRI are more sensitive than radiographs in detecting middle ear pathology. Brainstem auditory evoked response test may be abnormal if the cochlea, vestibulocochlear nerve or auditory brainstem pathways are involved and can sometime be used to differentiate central from peripheral VD. Finally, electromyography (EMG) and motor nerve conduction study are indicated in patients suspected of multiple cranial nerve neuropathy or of a more diffuse polyneuropathy.

- **Central Vestibular Disease**

Evaluation of patients suspected of central VD include in first instance the use of advance imaging (CT or MRI), cerebrospinal fluid (CSF) analysis (nucleated cell count and cytology, total protein concentration), serum and CSF titers (serology and/or PCR) for various infectious organisms (toxoplasma gondii, neospora caninum, canine distemper, coronavirus, fungal agents...). Further investigations may be require in cases suspected of brain tumour (tissue biopsy by surgical or stereotactic biopsy, thoracic and abdominal imaging to investigate

metastatic disease), thiamine deficiency (urinary organic acids excretion screening or transketolase activity in fresh erythrocytes) or cerebrovascular accident (routine hematology and serum biochemistry, clotting profile, evaluation of arterial blood pressure, thyroid, kidney, adrenal and heart function).