





## NEUROLOGY

# Head Tilt in Dogs: A Clinical Approach

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Head tilt in dogs (**FIGURE 1**) is a clinical presentation that most veterinarians encounter frequently in practice. When a patient presents for evaluation of a head tilt, it can sometimes be challenging to know the best course of diagnostics to recommend and whether or what primary treatment is warranted. Differentiation of the varied clinical presentations of vestibular disease and how they relate to the generation of a list of differential diagnoses can be extremely helpful in providing appropriate primary care and being able to better predict possible outcomes.

The primary reason for a dog to exhibit a head tilt is dysfunction of the vestibular system. The vestibular apparatus is responsible for an animal's maintenance of balance and the spatial orientation of the eyes, head, trunk and limbs relative to gravity. From a clinical perspective, it is divided into two components: peripheral, which involves the vestibulocochlear nerve (cranial nerve VIII) and its receptor in the inner ear; and central, which involves the brainstem and sometimes the cerebellum.

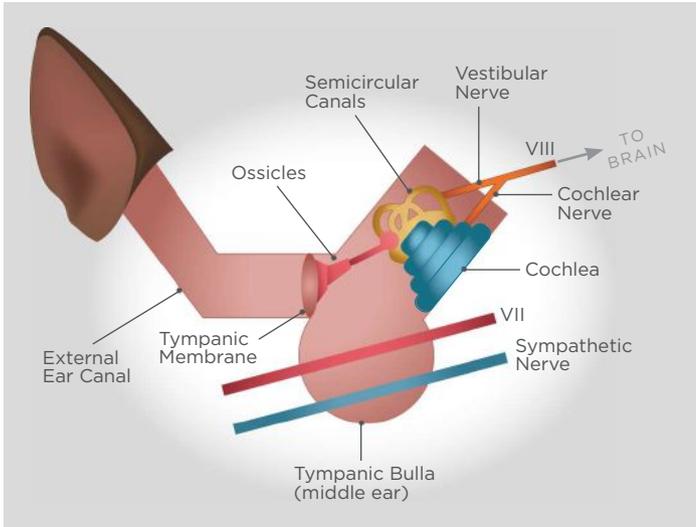
## BASIC REVIEW OF NEUROANATOMY

The receptor organ of the vestibular system is located within the petrous temporal bone in the inner ear and consists of the saccule, utricle and crista ampularis within the semicircular canal (**FIGURE 2**). Through movement of the hair cells bathed in endolymph, the position and movement of the animal as it relates to gravity is detected and then transmitted by cranial nerve VIII (vestibulocochlear nerve) to the vestibular nuclei in the brainstem.<sup>1</sup> The vestibular nuclei function to provide output to many other areas of the brain which result in:<sup>2</sup>

- Movement and position of the eyes via the medial longitudinal fasciculus and synapse on cranial nerves III, IV and VI.
- Provision of extensor tone to the muscles to support the body against gravity via the vestibulospinal tract.
- Conscious perception of position via the thalamus to the somatosensory cortex of the forebrain.

### **FIGURE 1. Evaluating a Head Tilt**

Differentiating the varied clinical presentations of vestibular disease can help in providing appropriate primary care.



**FIGURE 2.** Anatomy of the canine external, middle and inner ear.

- Coordination of movement of the eyes, head, trunk and extremities in relation to movement or static position via the caudal cerebellar peduncle to the cerebellum.
- Influence of the vomiting center in the reticular formation as a consequence of motion sickness.

## STEP 1: DETERMINE THE NEUROANATOMIC LOCALIZATION

Once a head tilt has been identified, the next step is to perform a complete neurologic examination of the patient to determine if the vestibular dysfunction is peripheral, central or paradoxical. Paradoxical vestibular disease refers to a distinct localization to the cerebellum: specifically, the caudal cerebellar

peduncle or the flocculonodular lobe<sup>2</sup> that result in slightly different clinical signs than typical peripheral or central vestibular disease. In general, patients with peripheral vestibular disease present with normal (albeit sometimes anxious) mentation, no other cranial nerve deficits (except sometimes cranial nerve VII), occasionally Horner's syndrome and normal postural reactions. On the contrary, dogs with central vestibular disease are more likely to have a more dull mentation, conscious proprioception deficits and one or more cranial nerve deficits. Postural reaction deficits are the most important indicator of central disease; and deficits in cranial nerve V to XII are the most likely.<sup>2</sup> Patients with paradoxical vestibular disease typically exhibit normal mentation, ipsilateral hypermetria, cerebellar ataxia and ipsilateral proprioceptive deficiencies but no other cranial nerve deficits (except occasionally a menace deficit); these patients exhibit a contralateral head tilt and lean opposite the side of the lesion.<sup>3</sup> A more complete guide to individual areas of testing and their neuroanatomic localization can be found in **TABLE 1**.

## STEP 2: DIFFERENTIAL DIAGNOSES

**TABLE 2** lists the differential diagnoses based on peripheral versus central vestibular disease. Below is a brief discussion of some of the more common diseases seen in clinical practice. In one study that evaluated 85 dogs that had MRI of the brain for the diagnosis of vestibular disease, 29% had peripheral vestibular disease, 46% had central vestibular disease and 25% had paradoxical vestibular disease.<sup>4</sup>

**TABLE 1** Clinical Signs Associated With Localization

	PERIPHERAL	CENTRAL	PARADOXICAL
Level of Consciousness	Normal	Normal or Impaired	Normal
Nystagmus	Horizontal or rotary; >60 bps	Horizontal, rotary or vertical; <10 bps	Horizontal, rotary or vertical
Head Tilt	Ipsilateral	Ipsilateral	Contralateral
Leaning/Falling	Ipsilateral	Ipsilateral	Contralateral
Postural Reactions	Normal	Ipsilateral	Ipsilateral
Cranial Nerve Abnormalities	+/- Cranial nerve VII, +/- Horner's Syndrome	Any	Uncommon, +/- menace deficit
Gait	Normal or vestibular ataxia	Vestibular +/- proprioceptive ataxia	+/- Hypermetria vestibular or cerebellar ataxia

**TABLE 2** Differential Diagnoses of Head Tilt in Dogs

	PERIPHERAL	CENTRAL
Degenerative	—	Storage disease
Anomalous	Congenital vestibular disease (rare)	Hydrocephalus, Chiari-like malformation
Metabolic	Hypothyroidism	Hypothyroidism
Neoplastic	Vestibular nerve sheath tumor, otogenic neoplasia	Intracranial neoplasia — primary vs. metastatic
Nutritional	—	Thiamine deficiency
Idiopathic	Geriatric (“old dog”) vestibular disease	—
Infectious/Inflammatory	Otitis media/interna	Viral: canine distemper virus; Bacterial: Rocky Mountain spotted fever, bartonellosis, ehrlichiosis, anaplasmosis, otogenic extension into the brain; Protozoal: toxoplasmosis, neosporosis; Fungal: cryptococcosis, coccidioidomycosis, blastomycosis, others  Non-infectious: Meningoencephalitis of Unknown Etiology (MUE/GME)
Toxic	Ototoxic drugs	Metronidazole
Trauma	Petrous temporal bone	Brainstem/cerebellum
Vascular	—	Ischemic or hemorrhagic vascular accident (brainstem/cerebellum); hypertension

## Peripheral Vestibular Disease

### Otitis media/interna

Of the peripheral vestibular diseases, otitis media/interna (OMI) is the most common diagnosis.<sup>4,5</sup> Though all breeds can be affected, in one study, the most commonly affected breeds were French bulldogs, cavalier King Charles spaniels, cocker spaniels and West Highland white terriers.<sup>5</sup> Diagnosis is based on otoscopic examination, imaging (CT or MRI is preferred<sup>4,5</sup>) and culture via myringotomy. It is important to note that an intact tympanic membrane does not rule out OMI; in fact, infection most commonly results from (1) a descending infection from otitis externa, (2) hematogenous spread of microorganisms or (3) extension of microorganisms from the pharynx via the auditory tube. A very common clinical presentation of OMI is vestibular disease with concurrent cranial nerve VII dysfunction and/or Horner's syndrome (**FIGURE 3**), due to the coursing of these pathways through the middle ear (**FIGURE 2**). Treatment is antibiotic therapy, ideally based on culture and sensitivity, and supportive care. An uncommon but severe progression of OMI is intracranial infection resulting from central extension of bacteria into the brainstem (**FIGURE 4**). Recognition

and prompt treatment of this complication can result in a good prognosis for recovery.<sup>6</sup>

### Idiopathic Vestibular Disease

Idiopathic vestibular disease is reported to be the second most common cause of peripheral vestibular disease in the dog.<sup>7</sup> Classically, no other neural deficits besides vestibular dysfunction are present (i.e., cranial nerve VII or Horner's); however recently an idiopathic syndrome of concomitant facial nerve and vestibular neuropathy was described in 16 dogs.<sup>8</sup> The average age of dogs that develop idiopathic vestibular disease is 12 to 13 years and there is no breed or sex predilection. There is typically a peracute onset with affected animals showing clinical improvement within a few days and near complete resolution within 3 to 4 weeks.<sup>7</sup> Occasionally, severely affected dogs will maintain a persistent head tilt despite otherwise complete resolution of all other vestibular signs. Though the cause of idiopathic vestibular disease is unknown, there is consideration that the cause may be due to a viral infection of the vestibular nerve.<sup>2</sup> In cases where the onset is peracute and there are no other cranial nerve abnormalities, the likelihood of idiopathic vestibular disease is high and only symptomatic treatment is

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warranted; there is no evidence to support the use of nonsteroidal anti-inflammatory drugs or corticosteroids.

### Hypothyroidism

Hypothyroidism has been implicated as a cause for both peripheral and central vestibular disease in dogs. The pathogenesis of hypothyroid-associated vestibular disease has been described as a component of polyneuropathy or as myxedematous disease causing nerve compression (cranial nerve VIII and sometimes cranial nerve VII) as they exit the skull through the internal acoustic meatus.<sup>2,9</sup> Accordingly, patients that present with cranial nerve VII and VIII dysfunction should have their thyroid function evaluated. Thyroid hormone supplementation typically results in improvement within a few months.

### Ototoxicity

Peripheral vestibular signs may develop if the ear has been flushed with any routine cleaning solution if the



**FIGURE 3.** Photograph of canine Horner's syndrome in the right eye. Note the elevated third eyelid, miosis and mild ptosis. Enophthalmos, which is often present, is not appreciated in this photograph.

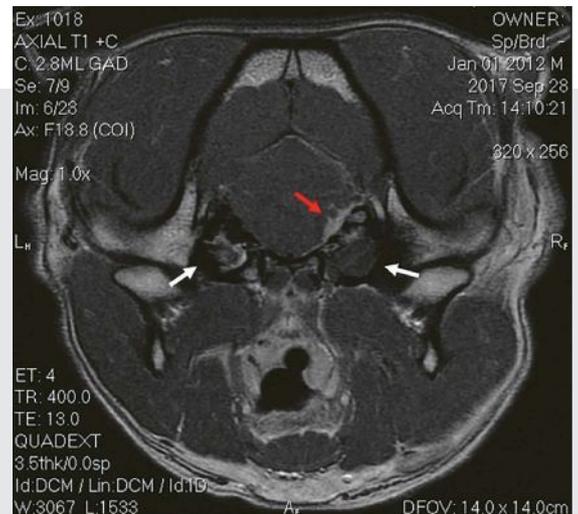
tympanum is ruptured. Many drugs/agents are potentially ototoxic; dogs may develop signs of peripheral vestibular disease or deafness. Clinical signs of vestibular disease may develop rapidly (as soon as 10 minutes). Within 3 days to 3 weeks, central compensation results in gradual improvement of clinical signs; the compensation is maximal after about 3 months. The head tilt, however, may still be obvious and permanent.

Chlorhexidine solutions can significantly contribute to ototoxicity and should be avoided if the status of the tympanum is unknown. Aminoglycoside antibiotics (parenteral or topical) can affect the hair cells, flocculonodular lobe and fastigial nucleus; streptomycin tends to have vestibular effects, whereas neomycin, gentamycin, kanamycin, and vancomycin tend to have more effect on the auditory system. In addition, loop diuretics, cisplatin and propylene glycol have also been implicated as potentially ototoxic.

### Central/Paradoxical Vestibular Disease

#### Meningoencephalitis of Unknown Etiology (MUE)

MUE encompasses several presumed idiopathic noninfectious inflammatory diseases in dogs; other terms that fall into this grouping include:



**FIGURE 4.** Post-contrast axial image of the canine brain at the level of the caudal brainstem. Note the contrast enhancement of the right lateral aspect of the medulla (red arrow). The tympanic bullae (white arrows) show isointense signal and heterogenous contrast enhancement depicting a soft-tissue/high protein type infiltrate in the normally air-filled bullae.

Left: Simon R. Platt

granulomatous meningoencephalomyelitis, necrotizing meningoencephalitis and necrotizing leukoencephalitis. MUE should be considered in patients that are suspected to have central vestibular disease with a multifocal intracranial localization; for example, vertigo with seizure activity, multiple cranial nerve deficits and/or concurrent spinal cord dysfunction or pain. Female, small breed dogs 4 to 8 years of age are predisposed, and diagnosis is based on MRI and, cerebrospinal fluid analysis, with or without infectious disease testing. Treatment involves immune suppression with glucocorticoids plus a chemotherapy drug (cytarabine arabinoside, procarbazine) or immunomodulating drug (cyclosporine, leflunomide). Aggressive long-term treatment is necessary, and the prognosis is guarded.<sup>10</sup>

A cerebrovascular accident (CVA) should be considered high on the differential list in patients that have acute onset, focal and nonprogressive central or paradoxical vestibular signs.

### Cerebrovascular Disease

Head tilt is the second most common clinical sign in dogs with cerebellar vascular infarcts.<sup>12</sup> A cerebrovascular accident (CVA) should be considered high on the differential list in patients that have acute onset, focal and nonprogressive central or paradoxical vestibular signs. Cerebrovascular disease can initially present as a transient ischemic attack (TIA), which is defined as a temporary, focal brain deficit secondary to vascular disease that completely resolves in <24 hours. In a recent study, 22% of the dogs investigated had episodes consistent with a TIA prior to a CVA or stroke.<sup>12</sup> Diagnosis of cerebrovascular disease is based on history, clinical signs and MRI of the brain; most CVAs are easily recognized on MRI; however many cases of TIA are undetectable even with high field MRI.<sup>13</sup> Following diagnosis of a CVA or suspicion of TIA, an investigation for the underlying cause should ensue. In one study of 33 dogs with brain infarcts,

18 cases had the following comorbidities identified: hypertension (29%), chronic kidney disease (24%), hyperadrenocorticism (18%) and single cases of aortic stenosis, diabetes mellitus, hemangiosarcoma and pheochromocytoma.<sup>14</sup> Treatment for stroke patients is supportive and many patients (approximately two-thirds) will recover with time; risk of recurrence is significantly higher in those patients in which a predisposing medical condition is identified.<sup>14</sup>

### Metronidazole Toxicity

Metronidazole administration can cause central vestibular or cerebellar dysfunction in dogs, typically following chronic administration of doses that exceed 60 mg/kg q24h. Diagnosis is based on history and clinical signs. Treatment includes cessation of metronidazole therapy and supportive care. It has been shown that diazepam administration (0.5 mg/kg PO q8h for 3 days) significantly reduces recovery time in affected dogs.<sup>11</sup>

## STEP 3: RATIONAL DIAGNOSTICS AND INITIAL TREATMENT STRATEGY

The diagnostic plan, treatment and prognosis can vary to a large extent based on whether the patient has peripheral or central vestibular disease. A careful history should be taken, including rate and duration of onset, whether the signs have progressed since onset, medications administered, diet, travel history, and existence of any other comorbidities. Physical exam should include a complete otoscopic exam and complete neurological examination to determine if the patient localizes to peripheral or central disease. A minimum database, including complete blood count, serum chemistry, blood pressure and thoracic with or without bullae radiographs, are recommended. If systemic infectious disease is suspected, serum titers or polymerase chain reaction for likely organisms (TABLE 2) can be performed. In patients where peripheral vestibular disease is suspected, additional diagnostic considerations may include myringotomy with culture and sensitivity and a complete thyroid panel (thyroxine, free T4 and thyroid-stimulating hormone) based on clinical suspicion. For patients that localize to central vestibular disease or those that are not improving with initial therapy, advanced imaging (ideally MRI) of the head and possible cerebrospinal fluid analysis are highly recommended.



Symptomatic treatment is often beneficial as many patients that have vestibular disease experience varied degrees of severity of clinical signs including nausea, anorexia, anxiety, recumbency, circling, falling and rolling. For dogs that are mildly affected, meclizine, an antiemetic and antivertigo drug, is indicated. Other antiemetics such as maropitant, ondansetron or chlorpromazine can also be considered. Patients more severely affected will benefit from parenteral fluid therapy and intensive nursing care in an inpatient setting. During periods of severe vertigo (i.e., continuous rolling), these patients should be sedated to the point of relaxation; I prefer intermittent dosing or constant rate infusion of dexmedetomidine at a low initial starting dose (62.5 mcg/m<sup>2</sup> IV). In addition, these patients need to be situated in as much of a “normal” posture as possible while still rotating the patient every 4 to 6 hours. Following the acute phase of vertigo, physical rehabilitation appears to aid in the recovery of ambulation and is recommended.

## SUMMARY

Head tilt in dogs is generally mediated through dysfunction of the vestibular or cerebellovestibular system. Careful attention to history, progression of disease and a complete neurological examination, especially postural reaction testing and a cranial nerve examination, is paramount. Localization of whether the vestibular disease is of peripheral or central origin is essential to the accurate formation of a differential diagnoses for the patient. The underlying cause may be simple or more complex, with peripheral disorders typically carrying a better overall prognosis. **TVP**

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An Auburn University veterinary school graduate, Dr. Carnes interned at University of Missouri-Columbia. She completed her neurology/neurosurgery residency and received a Master's degree in biomedical science from Auburn University. In 2009, she received her ACVIM board certification in neurology and completed a canine rehabilitation course at University of Tennessee in Knoxville and advanced neurosurgical certification course in Las Vegas. She has presented research globally and is a founding member of the Veterinary Neurosurgical Society. Carnes serves as a reviewer for the *American Journal of Veterinary Research*. In 2009, Dr. Carnes and her husband, Eric, co-founded Florida Veterinary Neurology & Imaging Centers, which owns Specialists in Companion Animal Neurology (SCAN).