

It's Not A Tumor: Approach to Vestibular Syndrome in Old Dogs



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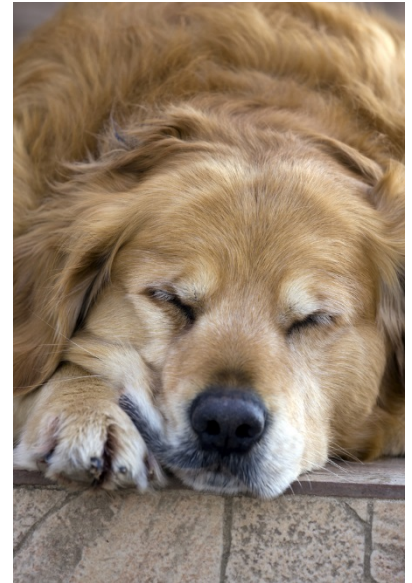
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Vestibular syndrome is one of the most common neurological clinical presentations in dogs. [Vestibular syndrome](#) refers to the collection of clinical signs caused by disruption of the normal function of the vestibular system. As with other clinical syndromes, there are specific considerations taken into account when evaluating geriatric patients.

The function of the vestibular system is to maintain balance and equilibrium. It can be broken down into peripheral and central components. The peripheral vestibular system is comprised of structures of the inner ear and the vestibular portion of cranial nerve VIII. The central vestibular system is comprised of the brainstem and cerebellum.

Classic clinical signs of vestibular syndrome include head tilt, vestibular ataxia (characterized by leaning, falling, circling, or alligator rolling), positional ventral or ventrolateral strabismus, and pathologic nystagmus. These signs occur in the direction of the lesion with the exception of the fast phase of nystagmus which is away from the lesion. The exception to this rule is paradoxical vestibular syndrome caused by certain cerebellar lesions which result in vestibular signs in the opposite direction as expected.

One of the key points to remember is that the clinical signs of the vestibular syndrome itself are identical no matter where the lesion is located. Distinction between peripheral and central disease is dependent upon evaluation for and identification of other neurological signs that could be caused by the lesion.



	Peripheral	Brainstem	Cerebellum
Additional Potential Deficits	<ul style="list-style-type: none"> • Horner's syndrome • Facial nerve dysfunction 	<ul style="list-style-type: none"> • Mentation change • Other cranial nerve deficits • Weakness • Proprioceptive deficits (postural reaction delays, proprioceptive ataxia) 	<ul style="list-style-type: none"> • Cerebellar ataxia (hypermetria, dysmetria) • Intention tremor • Truncal sway • Proprioceptive deficits (postural reaction delays, proprioceptive ataxia)

Diseases Causing Peripheral Vestibular Syndrome

[Hypothyroidism](#) is a common endocrine disorder in geriatric dogs. Approximately 7.5% of dogs with hypothyroidism will present neurological signs including peripheral vestibular dysfunction, facial nerve paralysis, or lower motor neuron para- or tetraparesis. These clinical syndromes can occur individually or together. Signs can be acute onset or insidious, progressive or non-progressive. It is important to note that many patients with neurological dysfunction secondary to hypothyroidism do not have the classic systemic clinical signs. Diagnosis and treatment is standard for hypothyroidism (measurement of thyroid hormone levels; levothyroxine 0.02 mg/kg PO q24 hours). The majority of affected dogs will respond positively to supplementation with improvement in clinical signs occurring usually over several weeks.

[Otitis media/interna](#) is one of the most common causes of peripheral vestibular syndrome in dogs. Although the proportion of affected dogs with otitis is higher in younger dogs, it still comprises a significant proportion of peripheral vestibular disease in older dogs. A vast majority of otitis media/interna cases are caused by bacterial infections which descend from the external ear canal. Clinical signs can be either acute onset or insidious, progressive or non-progressive, and can include facial nerve dysfunction and/or Horner's syndrome.

A definitive diagnosis of otitis media/interna requires advanced imaging of the head (CT or MRI), myringotomy, deep ear flush, and culture of middle ear, although a strong presumptive diagnosis can be made in some cases. Otic examination and otic cytology are critical diagnostic tools in these cases allowing for visualization of an inflamed or bulging tympanic membrane, ruptured tympanic membrane, or purulent material within the ear as well as cytologic evidence of infection. Treatment of otitis media/interna involves systemic antibiotic therapy for total of 6 to 8 weeks. Ideally antibiotic choices are based on culture of the middle ear; however, Clavamox (16 - 20 mg/kg PO Q 12 hours) is a good empirical choice. Cases in which a full diagnostic workup is strongly recommended include dogs that do not respond to empirical antibiotic therapy, patients that were successfully treated but experienced a recurrent infection, or patients with chronic ear disease that has been managed long-term with multiple rounds of antibiotic therapy. These patients are much more apt to have resistant infections. Given that most affected dogs also have [otitis externa](#), topical therapy is also important for management of current signs and future risk of recurrence. It is critical to avoid products with otic toxic ingredients (aminoglycoside antibiotics and chlorhexidine).

Prognosis for otitis media/interna is good and most dogs will show improvement in clinical signs within one week of starting appropriate antibiotic therapy although it can take several weeks for them to reach maximal neurological improvement. Many affected dogs will have residual neurological signs, most commonly a mild head tilt +/- facial nerve dysfunction.

Idiopathic vestibular disease, also known as old dog vestibular disease or geriatric vestibular disease, is the most common cause of peripheral vestibular syndrome in geriatric dogs. Clinical signs are classically peracute onset and regressive. There is no directed treatment for dogs with idiopathic vestibular disease; treatment is centered on supportive care such as anti-nausea medications, anti-anxiety medications, +/- IV fluids, and nursing care. Prognosis with idiopathic vestibular disease is generally good with most patients starting to show signs of improvement after 2 to 3 days and more complete recovery occurring in 2 to 3 weeks.

Diseases Affecting the Central Vestibular System

Primary brain tumors account for 85% of diagnosed intracranial neoplasms with the remaining 15% being metastatic cancers. Clinical signs can be acute although are more often insidious and are almost always progressive. Unfortunately, diagnosis of intracranial neoplasia does require advanced imaging. Otherwise, we are limited to a presumptive diagnosis based on patient age and clinical history. Treatment options for intracranial neoplasia is highly dependent upon the tumor location and type. Anti-inflammatory steroids (prednisone 1mg/kg/day) are the only empirical therapy option and work to reduce perilesional edema in the brain which often temporarily improves neurological symptoms. When taking all brain tumor types and locations into account, the median survival time with palliative treatment with steroids alone is approximately 2 to 4 months. However, infratentorial location, which would account for the majority of dogs relevant to this discussion, has been identified as a negative prognostic indicator with a lower median survival time of one month. Definitive therapies for brain tumors include [chemotherapy and radiation therapy](#) which can afford many more months or even years of survival, although these treatments do require definitive diagnosis.

Ischemic strokes occur as a result of occlusion of a blood vessel supplying an area of the brain. Classically, clinical signs are peracute onset and almost always regressive with spontaneous improvement occurring over several days to several weeks. Strokes can cause a variety of neurological signs which are referable to the area of the brain affected, although in dogs the rostral cerebellar artery is most commonly affected, resulting in vestibulocerebellar symptoms.

In dogs, approximately 50% of ischemic strokes are associated with some type of underlying risk factor. Risk factors can include hypertension, hypothyroidism, Cushing's disease, chronic kidney disease, protein losing disease (PLN, PLE), diabetes mellitus, hyperviscosity syndromes, hypercoagulability disorders, or tumor embolus. The remaining 50% of strokes are considered idiopathic. Although definitive diagnosis of a stroke itself requires advanced imaging, oftentimes a fairly strong presumptive diagnosis can be made based on clinical history. Investigation for underlying risk factors is critical, and the minimum diagnostic evaluation should include CBC, chemistry panel, thyroid hormone panel, urinalysis, UPC, and blood pressure. Additional diagnostic testing that may be indicated depending on the specific patient as well as the results of the initial diagnostic testing may include VCM or TEG (measures of coagulation and methods of evaluating for hypercoagulability), chest x-rays, abdominal ultrasound, and/or ACTH stimulation test or low dose dexamethasone suppression test.

There are no directed treatments for an ischemic stroke. Treatment centers around supportive care similar to what was discussed for idiopathic vestibular disease as well managing any underlying causes in an effort to prevent recurrence. Prognosis for a stroke is generally good with the majority of patients showing spontaneous regression of clinical signs within the first few days, although speed of recovery is extremely variable (days to weeks).