

Vestibular Diseases in Dogs - Part 2

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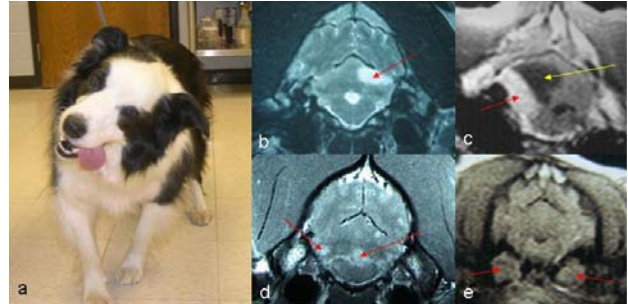
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INTRODUCTION

Please refer to Issue 3 of 2010 (pages 88-89) for Part 1.

PERIPHERAL VESTIBULAR DISEASES - continued

b) **Otitis Interna (or Labyrinthitis)** Very common : in a review of 83 cases of peripheral vestibular disease in the dog, 49% were attributed to infection. Labyrinthitis refers to inflammation of the inner ear that results in dysfunction of the membranous labyrinths. This disorder is almost always an extension of otitis media. Retrograde infection may occur via the Eustachian tubes. Another source of infection of middle ear structures is hematogenous spread. Medial extension of middle ear infection to involve meninges may occur. The majority of infections are caused by bacteria, including *Staphylococcus* spp., *Streptococcus* spp., *Proteus* spp., *Pseudomonas* spp., *Enterococcus* spp. and *Escherichia coli*. Occasionally, yeast infection (e.g., *Pityrosporum* spp. and *Candida* spp.) is observed. Rarely, fungal infection may be confined to the middle ear (e.g., *Cryptococcus* sp.). Varying degrees of vestibular dysfunction accompany labyrinthitis. Ipsilateral head tilt, nystagmus (usually rotatory), and ataxia are almost always present. Circling, falling, and rolling, may be seen in more severely affected animals. Ipsilateral facial paresis/paralysis and Horner's syndrome may occur. Because the facial nerve contains the parasympathetic preganglionic neurons that modulate lacrimal gland secretion, animals with labyrinthitis may have decreased tear production and develop ipsilateral keratoconjunctivitis sicca. Ipsilateral hemifacial spasms, resulting from "irritation" of the facial nerve, have been reported in dogs in association with otitis media. Deafness, resulting from involvement of the cochlear nerve, may accompany otitis interna. The diagnosis may be confirmed by otoscopic examination and skull radiography. Otoscopy may reveal otitis externa, and evidence of erosion or rupture of the tympanic membrane. Fluid in the middle ear may produce discoloration or bulging of the tympanic membrane. Inflammatory exudate or fluid should be submitted for culture and sensitivity testing. Fluid may be obtained by either aspiration or myringotomy. Radiographic examination of the temporal bones may reveal fluid within the tympanic cavity, or osteitis, sclerosis, or erosion of the tympanic bulla. CT or MRI images are more sensitive in outlining these alterations. Prognosis is usually favorable with prolonged oral antibiotic therapy, where selection is based on culture and sensitivity studies.



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)

Therapy: If the culture results are negative but bacterial otitis interna is suspected, then oral enrofloxacin (Baytril) 5 mg/kg every 12 hours or/and oral amoxicillin and clavulanic acid (Clavamox) 22 mg/kg may be administered. Antibiotic therapy should be continued for 8 weeks. Tear production should be monitored to detect keratoconjunctivitis sicca associated with facial nerve (CN 7) involvement, or antibiotic therapy and artificial tears administered if necessary to prevent keratitis and corneal ulceration. Aminoglycosides are contraindicated in otitis interna/media. In cases of concurrent otitis externa and media with a ruptured tympanic membrane, the external and middle ears are gently flushed with saline or a 2.5% acetic acid solution and caustic ear cleaning substances are avoided if possible. Diluted ear cleaning solutions may be necessary in some cases for debris that cannot be cleared by other means, but thorough rinsing is essential so residual cleaning solution does not irritate the vestibular labyrinth. The vestibular signs may resolve in 1-2 weeks but if antibiotics are prematurely discontinued, the clinical signs and infection recur and can be more difficult to treat. In acute cases, oral prednisone 0.25 mg/kg once-daily for 1-3 days is sometimes given to reduce inflammation and swelling of the vestibular labyrinth. In animals with recurring otitis, underlying dermatologic problems such as atopy or hypothyroidism should be investigated and treated. Ear hygiene should be monitored but care should be taken when cleaning the external ear canals. Surgical intervention: External ear canal ablation and bulla osteotomy (surgical drainage) may be required in refractory cases

- c) **Aural Cholesteatoma.** Aural cholesteatoma may accompany chronic otitis media. A cholesteatoma is a form of epidermoid cyst. It appears as a laminated structure composed of layers of keratin, and rests on a fibrous stroma of inflammatory granulation tissue. The masses may form from pockets of the tympanic membrane, which became adherent to the inflamed middle ear mucosa. Clinical signs in affected dogs included head tilt, loss of balance, deafness, and difficulty when eating or opening the mouth. Typically facial nerve involvement or Horner's syndrome is not present. Cholesteatomas may be responsible for extensive resorption and remodeling of temporal bone seen radiographically in some dogs.
- d) **Congenital Vestibular Syndrome.** Signs of peripheral vestibular disease, in the absence of deafness, have been observed in several breeds, including English cocker spaniels, German shepherd dogs, and Tibetan terriers. Severe head tilt, circling, and falling or rolling, may be noted from birth to 4 months of age. Nystagmus is rarely present. The cause is undetermined. Pathological lesions have not been demonstrated. Prognosis is guarded, as clinical signs may regress completely, re-occur, or remain static. There is no effective treatment. A congenital condition characterized by early onset of deafness and vestibular dysfunction has been reported in Doberman pinscher puppies. Signs of vestibular disease become evident between birth and 10 weeks of age. Puppies improve with age however, relapses may occur. Deafness occurs in all affected puppies. Pathological examination confirms loss of auditory sensory hair cells in the cochlea,



14-year Cocker with peripheral vestibular syndrome secondary to old dog idiopathic vestibular disease

and otoconial abnormalities or absence in maculae. The disorder in Doberman pinschers may have an autosomal recessive mode of inheritance. Congenital nystagmus in the absence of vestibular disease occurs sporadically in puppies. The nystagmus is usually pendulous, and resolves spontaneously. It has also been seen in Belgian shepherds with incomplete development of the optic chiasm.

- e) **Metabolic:** (Polyneuropathy-neuritis). Unilateral or bilateral signs of peripheral vestibular disease may occur in association with facial paresis or paralysis, in mature dogs without evidence of otitis media-interna. Some affected dogs are hypothyroid. Thyroid hormone-replacement therapy has proved ineffective.
- f) **Trauma.** Cranial trauma may result in signs of peripheral vestibular disease secondary to fractures of the temporal bone or tympanic bulla. Facial paralysis may accompany petrosal bone injury.
- g) **Toxic.** Prolonged systemic or topical therapy with aminoglycoside antibiotics may result in degeneration of the labyrinth receptors of the vestibular or auditory systems, or both.
- h) **Neoplasia.** Neoplasms involving the temporal bone may produce peripheral vestibular disease, often with facial paralysis or paresis. Fibrosarcoma, osteosarcoma, chondrosarcoma, and squamous cell carcinoma have been reported. Squamous cell carcinoma and ceruminous gland adenocarcinoma may involve adjacent soft tissues.

II) CENTRAL VESTIBULAR DISEASES

- a) Neoplasms of the brainstem or at the cerebellomedullary angle affect the vestibular system. Neoplasms may be located at the surface of the parenchyma (e.g., meningioma, neurofibroma, medulloblastoma, choroid plexus papilloma or malignant lymphoma), or may be located within the parenchyma (e.g., glioma, or granulomatous meningo-encephalomyelitis). Neoplasms at these locations occur in animals of all ages; however, young dogs appear to be susceptible.
- b) Nutritional. Thiamine deficiency may produce a mild vestibular ataxia as the earliest sign of degeneration. Bilateral and symmetrical hemorrhagic necrosis in the brainstem. Clinical signs: neck ventroflexion, Vertical nystagmus
- c) Inflammatory/ Infectious: Any cause of meningo- encephalitis may result in involvement of central vestibular structures. Reported causes include canine distemper, toxoplasmosis, cryptococcosis and granulomatous meningo-encephalomyelitis. Aberrant parasitic migration may produce severe signs of vestibular disturbance.
- d) Latrogenic. Secondary to ear cleaning. Chorexidine in a ruptured tympanic membrane.
- e) Toxic: Metronidazole toxicity: Dosages greater than 30 mg/kg/day can result in vestibular disease. The onset is acute and usually occurs when animals receive high doses for a long duration (e.g., after being on high doses for 7 to 12 days). Dose reductions need to be made in patients with liver and kidney disease as the drug is metabolized and excreted by these organs, respectively. Clinical signs may include generalized ataxia, nystagmus, anorexia, and vomiting. In severe cases, altered mental status, seizures, and opisthotonus may be present. Removal of the drug and supportive care (IV fluid diuresis) usually results in quick recovery. In severe cases diazepam administration with IV bolus (0.5mg/kg) and the diazepam orally (0.5mg/kg) for 3 days speed up neurology recovery. Prognosis: signs improves within 24-72 hrs of drug discontinuation but some time it may takes 1-2 weeks for a full neurologic recovery. Occasionally, deficits are permanent.
- f) **Metabolic:** hypothyroidism. Unilateral signs of central vestibular disease may also occur in mature dogs affected by hypothyroidism. Thyroid hormone-replacement therapy has proved to be effective.
- g) **Cerebrovascular Disease.** Cerebrovascular disease (CVD) may cause a peracute onset of central vestibular signs. Ischemia may be embolic or thrombotic and is the most common form of CVD; its causes may be due to sepsis, neoplasia, endocrine disease, hypertension or may commonly be idiopathic. In some areas of the country this can result from aberrant parasite (Cuterebra) migration through the nervous system. Non-traumatic hemorrhage, the other form of CVD, is bleeding into the parenchyma of the brain that may extend into the ventricles. Recovery from this disorder can be complete but can depend on the underlying disorder.

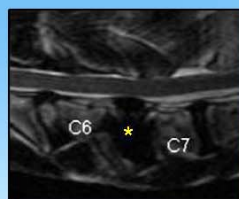
NEW TREATMENT OPTION FOR WOBBLER SYNDROME CERVICAL ARTIFICIAL DISC PLACEMENT



MRI - DIAGNOSIS
SPINAL CORD COMPRESSION
SECONDARY TO DISC HERNIATION
(ARROW)



POST OP RADIOGRAPHS
TITANIUM ARTIFICIAL
DISC IMPLANTED



MRI - 2 YEAR FOLLOW UP
ARTIFICIAL DISC IN PLACE (*)
WITH NO SIGNS OF SPINAL
CORD COMPRESSION AT THE
TREATED AND AT THE
ADJACENT DISC
SPACES

