The vestibular system is the major sensory (special proprioceptive) system that, along with the general proprioceptive and visual systems, maintains balance. An individual’s sense of balance is best summarized as a normal orientation with respect to the influence of gravitational forces. The vestibular system also functions to coordinate body posture and ocular position in relation to the position or motion of the head. Considering its physiologic roles, the clinical hallmarks of vestibular dysfunction are abnormalities of the gait, head and body posture, and ocular movement.

VESTIBULAR NEUROANATOMY

For clinical purposes, the anatomic constituents of the vestibular system are functionally divided into peripheral and central components. The peripheral portions of the vestibular system are located in the inner ear and consist of the receptors, ganglion, and peripheral axons of the vestibular division of cranial nerve VIII. The central components are the vestibular nuclei in the medulla and the vestibular projections to the cerebellum, spinal cord, and rostral brainstem.

Peripheral Vestibular System

The receptors for the vestibular system are colocalized with those for the auditory system in the bony and membranous labyrinths of the petrous temporal bone (inner ear). The bony labyrinth is divided into 3 major contiguous regions: the semicircular canals, the vestibule, and the cochlea. The lumens of each of these structures are filled with perilymph.

Within the bony labyrinth is the membranous labyrinth, which contains 4, endolymph-filled, communicating structures called the (1) semicircular ducts, (2) utricle, (3) saccule, and (4) cochlear duct (see Fig. 1A). The semicircular ducts are contained within the semicircular canals, the utricle and saccule within the vestibule, and the cochlear duct within the bony cochlea. Each of the semicircular ducts is oriented at right angles to the others, thus occupying 3 planes. In one end of each of the membranous semicircular ducts is a terminal dilation called the ampulla, and on
Fig. 1. Schematic neuroanatomy of the peripheral (A) and central (B) components of the vestibular system. (Illustrations prepared by Terry Lawrence, Virginia-Maryland Regional College of Veterinary Medicine, Department of Biomedical Illustration. Courtesy of Virginia-Maryland Regional College of Veterinary Medicine, Department of Biomedical Illustration.)
one side of each ampulla are structures called cristae, each of which is lined with ciliated neuroepithelial hair cells. The ampulla and crista collectively within the terminal portion of each semicircular duct is termed the crista ampullaris. The neural activity within these hair cells is continuously tonic, such that movement of the head in any direction of angular rotation subsequently results in displacement of endolymphatic fluid, altering the tonic neural influence of the semicircular ducts by deflection of the hair cells in the corresponding crista ampullaris. Dendrites of neurons from the vestibular portion of cranial nerve VIII synapse on these hair cells, and the deflection of the hair cells stimulates vestibular neurons. The 3 cristae ampullares receptors primarily respond to acceleration, deceleration, and rotation (ie, dynamic equilibrium), but are not activated at constant velocities. The semicircular canals are organized in such a fashion that movement in a plane that activates vestibular neurons in the crista ampullaris of one semicircular duct simultaneously inhibits neurons in the synergistic duct on the opposite side of the head. This paired and reciprocal system of ductal innervation functions to instantaneously activate the appropriate postural antigravity muscles following the detection of head rotation, thus preventing the development of an abnormal posture.

The maculae are the receptors located in the membranous utriculus and saccule (see Fig. 1A). The macula of the saccule is oriented in a vertical plane, whereas the macula of the utriculus is in a horizontal plane. The surface of each macula is covered with neuroepithelial hair cells, which project cilia into an otolithic membrane that covers the neuroepithelial surface of each macula. Movement of the otolithic membrane causes deflection of the cilia of the macular hair cells, and subsequently triggers an action potential in the dendritic zone of the vestibular neurons that synapse in each macula. The macular receptors of the utricle and saccule provide continual tonic nervous input, whose net functional effect is to maintain static equilibrium (sensation of static head position relative to gravity), as well as respond to linear acceleration, which participates in the preservation of a normal, upright head and body posture.

The vestibular division of cranial nerve VIII has dendritic connections with the cristae and the maculae, and its axons project through the internal acoustic meatus. The cell bodies of bipolar vestibular axons are located in the vestibular ganglion (see Fig. 1A), which is located in the petrous temporal bone.

Central Vestibular System

After leaving the internal acoustic meatus, vestibular axons project to the lateral aspect of the medulla where the majority terminate in the vestibular nuclei (see Fig. 1B), while a smaller fraction course into the flocculonodular lobe of the cerebellar cortex and cerebellar medulla by way of the caudal cerebellar peduncle. There are 4 vestibular nuclei on either side of the midline adjacent to the lateral wall of the fourth ventricle that form the vestibular trigone. The neurons in these nuclei are interneurons that generally provide excitatory influences to local interneurons in other parts of the central nervous system. The clinically relevant central vestibular nuclear projections have 3 primary targets, which are neurons of the (1) spinal cord, (2) rostral brainstem, or (3) cerebellum.

Spinal cord projections

The vestibulospinal tract is the primary spinal cord projection that descends from the vestibular nuclei in the medulla to all segments of the spinal cord in the ipsilateral ventral funiculus, and exerts the following influences over motor neurons, which are mediated via segmental interneurons: ipsilateral extensor muscles are facilitated,
ipsilateral flexor muscles are inhibited, and contralateral extensor muscles are in-
hhibited (see Fig. 1B). Therefore, the overall effect of vestibular system activation is
an ipsilateral increase in antigravity muscle tone and contralateral inhibition of tone
and stretch reflexes. These pathways contribute to coordination of motor activity
to the limbs, neck, and trunk in response to movement of the head. A vestibular lesion
that unilaterally abolishes or diminishes the normally tonic neural vestibular input
results in the unopposed stimulation of the vestibulospinal tract of the unaffected
side, which effectively causes the head and body to lean toward the side of the lesion.

**Brainstem projections**

**Medial longitudinal fasciculus** The medial longitudinal fasciculus (MLF) (see Fig. 1B)
ascends from vestibular nuclei in the medulla to synapse on lower motor neurons in
the motor nuclei of cranial nerves III, IV, and VI. This pathway provides coordinated,
conjugate ocular movements as the head changes position. The MLF is also part of the
pathway that is responsible for the observation of physiologic nystagmus that is
induced when testing the vestibulo-ocular reflex.

**Reticular formation and vomiting center** Axons from the vestibular nuclei project to
the vomiting center within the reticular formation. This pathway accounts for the vom-
iting that can be associated with motion sickness/vestibular disease. Vomiting is
uncommonly seen in veterinary vestibular diseases, when compared with human
equivalents.

**Conscious perception of balance** Conscious perception of balance and equilibrium
are obviously important, based on the verbal descriptions of abnormalities in cortical
spatial perceptions often provided by humans with vestibular disorders. The
afferent pathways for conscious perception of vestibular dysfunction are currently
poorly understood, but are believed to ascend through thalamic relay centers to the
temporal cerebral cortex.

**Cerebellar projections**

Vestibular axons from the vestibular nuclei and vestibular ganglion project to the ves-
tibulocerebellum (flocculonodular lobe and fastigial nucleus) via the caudal cerebellar
peduncle. These axons maintain coordination of the eyes, neck, trunk, and limbs in
relation to movements of the head, as well as when the head is in a static position.

**CLINICAL SIGNS OF VESTIBULAR DYSFUNCTION**

Diseases of the vestibular system cause varyingly severe balance and postural distur-
bances along with vestibular ataxia. Clinical signs may be a result of dysfunction of the
peripheral or central components of the vestibular apparatus (see Fig. 1A and B). Clin-
ical signs of vestibular dysfunction are typically reflective of a unilateral disease
process, but may occasionally be bilateral.

**Common Clinical Features of Vestibular Disease**

Diseases that affect the peripheral or central components of the vestibular system are
accompanied by a set of cardinal clinical features that are often outwardly visible or
easily evoked during the neurologic examination (Table 1).

**Head tilt**

A head tilt (Fig. 2) is the postural abnormality that results from the unilateral loss of
antigravity muscular tone in the neck region. The degree of ventral deviation of the
ear can vary from a few degrees to 45°. The ventrally deviated ear is directed toward
Nystagmus
Nystagmus is an involuntary and rhythmic movement of the eyes. Nystagmus can be physiologic or pathologic in nature. The most common forms of both inducible physiologic and pathologic nystagmus seen in veterinary practice are characterized by unequal directional eye movements and are thus termed jerk-types of nystagmus.\(^1,2\) Jerk nystagmus is characterized as having distinct fast and slow phases of ocular movement. When describing jerk nystagmus, it is conventional to define it according to the axis of movement (horizontal, vertical, rotary) of the globe as well as the direction of the fast phase.

Table 1
Clinical signs common to central and peripheral vestibular diseases

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Description and Comments</th>
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<tbody>
<tr>
<td>Head postural abnormality</td>
<td>Head tilt; deviation of one ear ventrally; the ventrally deviated ear is usually directed toward the lesion</td>
</tr>
<tr>
<td>Pathologic nystagmus</td>
<td>Jerk nystagmus present with distinct fast and slow phases; abnormal ocular movement can occur in a horizontal, rotary, or vertical direction</td>
</tr>
<tr>
<td>Vestibular strabismus</td>
<td>Positional ventral to ventrolateral strabismus (dropped globe) present ipsilateral to a vestibular lesion and usually apparent only when extending the head and neck</td>
</tr>
<tr>
<td>Vestibular ataxia</td>
<td>Flexing of the neck and trunk with the concavity toward the lesion; leaning, falling, rolling, or circling toward the side of the lesion</td>
</tr>
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the side of the vestibular lesion in most cases, except when a paradoxic central vestibular lesion is present (see later discussion).

Fig. 2. Dog with a left head tilt.
Physiologic nystagmus  In a normal animal, rotation of the head will result in the induction of a jerk nystagmus in the plane of rotation, with the fast phase occurring in the same direction as the movement. A distinct slow phase of ocular movement will occur in the direction opposite of the head rotation. The purpose of this physiologic response, which is termed the vestibulo-ocular reflex, is to preserve image stability on the retina to optimize performance of the visual system. For this system to function, afferent initiating stimuli from the semicircular canals ascend to the vestibular nuclei. The vestibular nuclei are interconnected with the somatic motor nuclei that control extraocular muscle movement in the brainstem (the oculomotor [CN III], trochlear [CN IV], and abducent nuclei [CN VI]) via the MLF (see Fig. 1B). Movement of the head results in reciprocating afferent stimuli from the paired semicircular canals in the plane of movement to the vestibular nuclei and then through the MLF in such a fashion that allows for coordinated and conjugate eye movements to occur.

Pathologic nystagmus  Pathologic nystagmus that can be observed when the head is at rest or in a neutral position is termed spontaneous or resting nystagmus. Pathologic nystagmus may also be inducible in the absence of resting nystagmus when the head is moved into certain positions, such as placing the animal in dorsal recumbency; this is termed positional (pathologic) nystagmus. Pathologic nystagmus results from a unilateral disturbance in the normal bilaterally tonic influences provided by vestibular neurons to the motor nuclei of the extraocular muscles (CN III, IV, VI). Spontaneous nystagmus may be a very short-lived abnormality, as it can often be rapidly compensated for by voluntary visual fixation, especially when the spontaneous nystagmus is the result of a peripheral vestibular lesion.

Vestibular (positional) strabismus  Vestibular strabismus is a positional phenomenon that manifests as a ventral to ventrolateral deviation of the globe, resulting in an increased exposure of the sclera dorsally when the head and neck are extended during testing of the tonic neck reaction. Vestibular strabismus subsequently resolves when the head is returned to a neutral position. Vestibular strabismus occurs on the side ipsilateral to a vestibular lesion.

Vestibular ataxia  The hallmark of vestibular ataxia is its asymmetric nature. Affected animals tend to lean, fall, roll, or circle toward the side of the lesion. Vestibular dysfunction typically results in circling characterized by a tight turning radius. The head and trunk may sway, and animals can assume a base-wide stance. The asymmetry of vestibular ataxia results from the altered physiologic influences normally provided via the vestibulospinal tract, as described earlier (see Fig. 1B).

Clinical Differentiation of Central and Peripheral Vestibular Lesions  Once any of these common features has been identified (head tilt, nystagmus, vestibular ataxia, positional strabismus), the priority of the examining clinician should be to attempt to identify the origin of the problem in either the peripheral or central components of the vestibular system (Table 2). Definitive diagnosis and management of central lesions will, in general, require more expensive and aggressive diagnostics and therapies, and the common causes of central vestibular disease are often associated with guarded prognoses. With the exception of malignant aural neoplasms, peripheral vestibular lesions are usually associated with good prognoses, and can be usually be diagnosed with equipment and techniques that are available and familiar to veterinary practitioners.
Clinical signs of peripheral vestibular disease

Peripheral vestibular disease does not affect strength or general proprioception; thus, peripheral disease results in an asymmetric ataxia and loss of balance, in the notable absence of detectable paresis or proprioceptive deficits. Spontaneous or positional horizontal or rotary jerk nystagmus can occur with peripheral vestibular lesions, and the fast phase will be away from the side of the lesion. Any pathologic nystagmus noted will not change direction as the head position is changed. Although debatable, it is the general consensus that vertical nystagmus is rarely (or never) associated with peripheral vestibular disease.2,7 Peripheral vestibular lesions can also affect the facial nerve and postganglionic sympathetic innervation (Horner syndrome) to the head. Both of these neural structures are closely associated with the inner ear and vestibular receptors.

Bilateral Peripheral Vestibular Disease

Bilateral peripheral vestibular disease is occasionally seen, and is clinically characterized by the absence of a head tilt and pathologic nystagmus, and an absent vestibulo-ocular reflex due to bilateral interruption of input from vestibular receptors. Affected animals will usually crouch low to the ground, walk tentatively, and may fall to both sides. The animal also will usually display wide lateral excursions of the head from side to side in an attempt to maintain visual fixation. This condition occurs more commonly in cats, and they often will have little apparent disturbance in equilibrium.1,2

Clinical Signs of Central Vestibular Disease

Lesions in the pontomedullary region most often exert a regional affect rather than being limited to a specific nerve or nucleus. Thus, lesions in the area of the vestibular nuclei also incorporate the reticular formation, which includes ascending general proprioceptive (GP) and descending upper motor neuron (UMN) white matter tracts, the reticular activating system (RAS), and cranial nerve V-XII lower motor neurons. Therefore, vestibular signs associated with a depressed level of consciousness (RAS), spastic hemiparesis (descending UMN tract deficit), cranial nerve V-XII deficits, or general proprioceptive deficits (ascending GP tracts) on the same side as the vestibular deficits should be considered to indicate a central vestibular disorder.2,7 Identification of hemi- or tetraparesis in an animal with vestibular signs is the most

| Table 2 | Differentiating clinical features of peripheral and central vestibular disease |
|---|---|---|
| Clinical Sign | Peripheral Vestibular Lesion | Central Vestibular Lesion |
| Head tilt | Toward lesion | To either side |
| Pathologic nystagmus | − Direction not altered by head position | − Direction may change with head position |
| | − Horizontal or rotary | − Horizontal, rotary, or vertical |
| | − Fast phase away from lesion | |
| Postural reactions | Normal | Deficits ipsilateral to lesion |
| Conscious proprioception | Normal | Deficits ipsilateral to lesion |
| Cranial nerve deficits | ± Ipsilateral CN VII | ± CNN V-XII ipsilateral to lesion |
| Horner syndrome | ± Postganglionic | ± Preganglionic (rare) |
| Consciousness | Normal | Normal to comatose |
| | • Disorientation if acute | |
reliable indicator of the presence of a central vestibular lesion.\textsuperscript{2,7} In addition, spontaneous vertical nystagmus or pathologic nystagmus that changes direction (ie, from horizontal to vertical on changing head position) indicates the presence of central vestibular disease.\textsuperscript{1,2}

**Paradoxic (Central) Vestibular Disease**

A head tilt and balance loss will occasionally be appreciated in a patient that simultaneously has postural reaction deficits that are contralateral to the direction of the head tilt. When these specific clinical signs are noticed, the lesion must involve the caudal cerebellar peduncle or the flocculonodular lobe of the cerebellum on the side of the body opposite that of the head tilt.\textsuperscript{2,6} This condition is called paradoxic vestibular disease, and it is always indicative of central vestibular dysfunction. Because the head tilt does not fit the expected pattern for central disease, it is a paradox.

**VESTIBULAR DISEASES OF THE DOG AND CAT**

**Peripheral Vestibular Disorders**

In addition to a detailed history and neurologic examination, diagnostics that are helpful to assess the peripheral vestibular apparatus include otoscopic examination, bulla radiographs, bulla ultrasound, microbiology, myringotomy, fine-needle aspirate, serology, and biopsy procedures (Fig. 3). Performance of these diagnostics is greatly facilitated by heavy sedation or general anesthesia. Computed tomography (CT) and magnetic resonance imaging (MRI) are also valuable for the diagnosis and morphologic characterization of peripheral vestibular diseases,\textsuperscript{8–13} but most disorders that cause peripheral vestibular dysfunction can be identified and managed without these imaging techniques. Table 3 provides a summary of common origins of peripheral vestibular dysfunction.

![Fig. 3. Diagnostic algorithm for peripheral vestibular disease. CT, computed tomography; DDx, differential diagnoses; MRI, magnetic resonance imaging; OMI, otitis media interna; US, ultrasonography.](image-url)
Congenital vestibular disease

Congenital vestibular dysfunction has been reported in multiple purebred dogs, including Dobermans, Beagles, Cockers, Akitas, and primarily oriental breeds of cats such as Siamese, Tonkanese, and Burmese.\textsuperscript{14,15} Clinical signs are usually apparent at birth or develop within the first few weeks of life, the cause is usually unknown, and there is no therapy. Bilateral vestibular dysfunction has been occasionally reported in some breeds. Signs resolve spontaneously in some animals, whereas others may have residual and permanent head tilts. Affected animals are usually able to compensate well for the vestibular dysfunction. This condition is variably associated with deafness or other congenital malformations. Therefore, performance of brainstem auditory evoked responses (BAER) to evaluate hearing may be indicated in these cases.

Hypothyroidism

Hypothyroidism has been implicated as a possible cause of peripheral cranial mononeuropathies that affect CN VIII, and often CN VII concurrently.\textsuperscript{16,17} Affected dogs may also have accompanying signs of flaccid limb weakness, suggestive of a more generalized polyneuropathy. Hypothyroidism may result in peripheral cranial neuropathies as a result of myxomatous compression of cranial nerves as they exit their respective skull foraminae. The onset of hypothyroid implicated vestibular disease may be acute or chronic in nature.\textsuperscript{16} Diagnosis is based on documentation of low T4, free T4, and elevated thyroid-stimulating hormone (TSH) concentrations. Thyroid hormone supplementation usually results in improvement within a few months.

Aural neoplasia

Primary aural neoplasms can arise from constituents of the pinna, external canal, or middle and inner ear. Aural neoplasms may cause peripheral vestibular disease via direct compression or infiltration of the labyrinthine or neural components of the peripheral vestibular systems, or are indirectly associated with the inflammatory responses they initiate. Ceruminous adenoma/adenocarcinoma, sebaceous adenoma/adenocarcinoma, carcinomas of undetermined etiology, squamous cell carcinoma, and feline lymphoma are the most common primary aural tumors of small animals that are associated with peripheral vestibular dysfunction.\textsuperscript{18,19} Vestibular neurofibromas (schwannomas) may also primarily arise from the vestibulocochlear

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Common causes of peripheral vestibular disease by species</th>
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<tbody>
<tr>
<td>DAMNIT Category</td>
<td>Specific Diseases</td>
</tr>
<tr>
<td>Anomalous</td>
<td>Congenital vestibular disease</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Primary aural neoplasia</td>
</tr>
<tr>
<td></td>
<td>Vestibular neurofibroma</td>
</tr>
<tr>
<td>Infectious/Inflammatory</td>
<td>Otitis media interna (OMI)</td>
</tr>
<tr>
<td></td>
<td>Naso- and otopharyngeal polyps</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Idiopathic vestibular disease (vestibular neuronitis)</td>
</tr>
<tr>
<td>Trauma</td>
<td>Inner ear trauma</td>
</tr>
<tr>
<td>Toxic</td>
<td>Ototoxic drugs (systemic and topical)</td>
</tr>
</tbody>
</table>

Vestibular Disease

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nerve itself, but are uncommon. The majority (85%) of aural neoplasms in cats represent malignant phenotypes, whereas approximately 60% of canine aural tumors are malignant. Diagnosis of these neoplasms is often obvious on visual inspection of the ear or otoscopic examination, appearing as irregular, pedunculated, polypoid, or ulcerated masses on the pinna or within the external ear canal or tympanic bulla (Fig. 4). The neoplasms will also occasionally result in visible and palpable soft tissue swellings external to the ear (see Fig. 4A and B). Otoscopic-assisted biopsy will confirm the diagnosis. CT and MR imaging may be considered to determine the extent of the lesion before cytoreductive surgery or radiation therapy, as these neoplasms may locally invade the adjacent soft tissues of the head (Fig. 5), skull, or brainstem. Diagnostic imaging features of lytic bone disease involving the bulla or petrous temporal bone is more often associated with aural neoplasia than inflammatory disease. Thus, if osteolysis is present on radiographs, CT, or MRI, aural neoplasia should be a primary differential diagnostic consideration. Aggressive surgical excision of aural neoplasia is the treatment of choice, although primary or adjunctive radiotherapy may also be of benefit. Prednisone (0.5–1 mg/kg/d by mouth) may transiently palliate some of the clinical signs.

Otitis media interna
Otitis media interna (OMI) is the most common cause of peripheral vestibular disease seen in dogs and cats, and may account for nearly 50% of all cases of canine peripheral vestibular disease. It is important to recognize that otitis media alone will not result in vestibular signs. If deficits compatible with peripheral vestibular dysfunction are detected, inner ear involvement is confirmed. OMI is the most common cause of combinations of simultaneously occurring ipsilateral deficits of the peripheral portions of cranial nerves VII, VIII, and the postganglionic sympathetic neuron (Horner syndrome) to the head. In animals with OMI, peripheral vestibular signs may also be accompanied or preceded by nonneurologic signs referable to infection of the external or middle ears, such as head shaking, temporomandibular pain, bulla pain, or otic discharge. Otitis media has been shown to be a common complication of chronic otitis externa, occurring in 50% to 80% of dogs with chronic otitis externa.

A thorough otoscopic examination, bulla imaging, and myringotomy are the primary tools used to diagnose OMI. Otoscopic diagnosis of OMI can be complicated by
chronic remodeling of the external ear canal (hyperplasia, stenosis) that impedes visualization of the tympanum and sampling of the middle ear cavity. In addition, the presence of an intact or grossly normal tympanic membrane or normal appearing external ear canal does not exclude the possibility that OMI may be present. It has been reported that 70% of dogs with OMI had an intact tympanum.23 Bulla radiographs, CT, and MRI provide supportive diagnostic information by revealing fluid or soft tissue accumulations within the bullae, and often secondary reactive or remodeling changes of the middle and external ear (Fig. 6A and B; sclerosis, thickening, or lysis of the bullae, calcification or stenosis of external ear canal), depending on the chronicity of the lesion.10–12 CT imaging has been reported to be more sensitive than radiography for evaluation of the bullae in cases of OMI.10,12,13 When performing a radiographic bulla series, obtaining rostrocaudal open-mouth and oblique views in addition to

Fig. 5. Postcontrast, axial (A) and dorsal planar (B) T1-weighted MR images from the level of the tympanic bullae from a cat with right peripheral vestibular signs and a palpable soft tissue swelling at the base of the right ear (arrows). Both the material in the right tympanic bulla and the periaural soft tissues demonstrate contrast enhancement. Biopsy of these lesions revealed lymphosarcoma.
standard lateral and dorsoventral views is helpful. Ultrasonographic imaging techniques that are effective in the identification of fluid within the bullae have also been described. Ultrasonography offers advantages over radiography, CT, and MRI in that the bullae can usually be satisfactorily imaged without anesthesia.

Myringotomy samples should be submitted for cytologic evaluation and culture. Commonly cultured organisms include staphylococcal species, *Pseudomonas*, *Streptococcus*, *Proteus*, *Malassezia*, and *Candida*. The emergence of multidrug-resistant *Pseudomonas* and staphylococcal species in veterinary medicine has confirmed the importance of culture and sensitivity in the management of chronic or recurrent OMI. The presence of anatomic conformational defects, otic foreign bodies, keratinization disorders, ectoparasites, and allergic disease may predispose the animal to otitis externa and therefore OMI. A primary secretory otitis media (PSOM) has been also described, primarily in Cavalier King Charles Spaniels, to commonly cause vestibular signs. In PSOM, the debris in the middle ear consists of a viscous mucous plug.
Medical treatment of OMI and PSOM consists in thorough cleansing and flushing of any exudates and debris from the affected ear under anesthesia, 4 to 8 weeks of broad-spectrum, systemically administered antimicrobial therapy, ideally based on culture and sensitivity, identification and treatment of predisposing factors, and anti-inflammatory (topical or systemic) therapy. When cleansing the ear, exercise caution when considering the instillation of any solution or drug that is potentially ototoxic. Sterile physiologic (0.9%) saline or sterile water are readily available, nontoxic, inexpensive, and sufficient for most ear-cleansing applications.

Otogenic infections arising from the external or middle ears can extend into the calvarium, causing brain abscessation and bacterial meningoencephalitis. Clinical signs in these cases indicate central vestibular lesions, but may be preceded by peripheral vestibular signs. Aggressive surgical debridement and parenteral antibiotic therapy are required in these cases.

Bulla osteotomy or total ear canal ablation procedures should be considered in animals that do not respond to medical treatment, experience a relapse of clinical signs despite appropriate therapy, or have chronic, end-stage remodeling of the ear anatomy. In general, animals with OMI that are successfully treated will compensate for residual vestibular dysfunction and recover, but facial paresis may be permanent and can be a complication of surgery.

Naso- and otopharyngeal polyps

Inflammatory polyps arise from the mucosal lining of the tympanum, auditory tube, or pharynx, and are much more common in cats than dogs. Inflammatory polyps are usually unilateral, and are typically seen in young cats (1–5 years of age). Vestibular signs may be preceded by signs of chronic upper respiratory, pharyngeal, or otic disease. Polyps are usually easily diagnosed with otoscopic and oral examinations (Fig. 7). Radiographs, CT, or endoscopy may occasionally be required for diagnosis, or to document the presence of middle ear involvement when planning treatment. Removal of the polyp via traction polypectomy through the mouth or external ear canal

Fig. 7. Intraoral photograph from a cat with peripheral vestibular signs from an otopharyngeal polyp (arrows). The soft palate is retracted with a spay hook.
is usually successful and sufficient if there is no involvement of the tympanic cavity, but is associated with a 30% to 40% recurrence rate. Surgical removal via a bulla osteotomy/ear canal ablation has a recurrence rate of less than 10%. Vestibular signs, Horner syndrome, and facial nerve paresis, which are usually transient, can occur as sequelae of surgical polypectomy.

**Canine and feline idiopathic peripheral vestibular disease; geriatric vestibular disease; vestibular neuritis**

Canine idiopathic peripheral vestibular disease is the second most common cause of peripheral vestibular dysfunction in dogs, and is a common etiology for unilateral peripheral vestibular dysfunction of peracute onset (head tilt, ataxia, horizontal or rotary nystagmus) in dogs and cats. Although this disease may be seen in any aged dog, geriatric canines appear to be predisposed, and it is very atypical to be seen in dogs younger than 5 years old. In both dogs and cats, idiopathic peripheral vestibular disease results in clinical signs referable to dysfunction of the peripheral vestibular system only; affected animals do not have concurrent facial nerve paralysis or postganglionic Horner syndrome. In the acute setting the clinical signs can be severe (rolling, falling) and some animals may vomit.

Feline idiopathic peripheral vestibular disease differs slightly in that it can occur in cats of any age, has a higher incidence in outdoor cats in the summer and fall months in the northeastern and mid-Atlantic regions of the United States, and will occasionally result in bilateral peripheral vestibular signs. Idiopathic vestibular disease is diagnosed by excluding other causes of peripheral vestibular dysfunction. Diagnostic imaging studies of the peripheral vestibular apparatus are usually normal in animals with this disease. The cause of this disease is unknown, although it is often compared with vestibular neuronitis in humans, which may be triggered by viral antigens.

Diazepam can be administered for its anxiolytic effects. An empiric course of systemic broad-spectrum antibiotic therapy is reasonable to treat occult OMI. Initial signs of improvement occur within 3 to 5 days, and recovery is noted within 2 to 3 weeks. A residual head tilt may persist. Therapy is mainly supportive, and compensation for vestibular dysfunction is also greatly accelerated in animals that are encouraged and assisted to walk. There is no evidence that symptomatic medical therapy, such as anti-inflammatory treatment with corticosteroids, nonsteroidal anti-inflammatories, or antihistamine motion sickness drugs expedites or influences recovery from this disease process. Nausea and vomiting associated with vestibular disease can be treated as necessary. This condition may occasionally recur.

**Ototoxicity**

Numerous therapeutic substances, including aminoglycoside antibiotics, furosemide, platinum-containing antineoplastic agents, salicylates, and many detergents and alcohol-based solutions have been demonstrated to have ototoxic potential when administered parenterally or topically in the presence of a compromised tympanic membrane. The ototoxicity of most compounds results from induction of damage to or death of the neuroepithelial (hair-cell) receptors within the membranous labyrinth. The clinical manifestations of ototoxicity are drug dependent and wide ranging in severity, and can include both sensorineural deafness and vestibular dysfunction. In the majority of cases, deafness that results is permanent, while vestibular signs may improve or resolve. It is prudent to recognize that the majority of commercially marketed otic antimicrobial and cleansing solutions approved for topical applications contain one or more potentially ototoxic ingredients. Any therapeutic agent with ototoxic potential should be avoided in cases in which the tympanum is known or
suspected to be perforated. It may be necessary to perform a BAER to confirm acquired sensorineural deafness.

Central Vestibular Disorders

Clinical localization of the lesion to the central vestibular system is generally an indication for the performance of more aggressive and invasive diagnostics (intracranial cross-sectional imaging such as MRI and CT, CSF analysis, serologic and genetic assays, and BAER). MRI is the preferred diagnostic imaging modality for patients with central vestibular dysfunction. In a retrospective review of canine vestibular disease, brain morphologic abnormalities were detected in 100% of dogs with clinical evidence of central vestibular dysfunction in which MRI was performed.8 With few exceptions, many of the common causes of central vestibular disease (Table 4) can be associated with rapid and severe neurologic deterioration or death if not identified and treated promptly.

Hypothyroidism

Central vestibular and vestibulocerebellar signs can rarely be associated with canine hypothyroidism.30,31 Many dogs (70%) with central vestibular complications of hypothyroidism have no other extraneural clinical evidence of hypothyroidism.30 However, serum biochemical profiles from affected dogs commonly demonstrate hypercholesterolemia or hypertriglyceridemia. The cause of hypothyroid-associated central vestibular disease is likely multifactorial, and includes ischemic infarction associated with atherosclerotic vascular disease and central nervous system (CNS) demyelination.30,32 Intracranial imaging studies from these dogs may be normal or reveal evidence of infarction. Diagnosis is based on documentation of low T4, free T4, and elevated TSH concentrations, excluding other possible causes of central vestibular

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<thead>
<tr>
<th>Table 4</th>
<th>Central vestibular diseases of the dog and cat</th>
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</thead>
<tbody>
<tr>
<td><strong>DAMNIT Category</strong></td>
<td><strong>Specific Diseases</strong></td>
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<tr>
<td>Anomalous</td>
<td>Quadrigeminal arachnoid-like cysts</td>
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<td></td>
<td>Caudal occipital malformation syndrome</td>
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<td></td>
<td>Hydrocephalus</td>
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<tr>
<td>Metabolic</td>
<td>Hypothyroidism&lt;sup&gt;a&lt;/sup&gt; (± infarction)</td>
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<tr>
<td>Nutritional</td>
<td>Thiamine deficiency</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Primary intracranial neoplasms&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>• Meningioma, glioma, medulloblastoma, choroid plexus tumors, lymphoma</td>
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<tr>
<td></td>
<td>Metastatic neoplasms</td>
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<tr>
<td>Infectious/Inflammatory</td>
<td>Viral—Canine distemper virus, feline infectious peritonitis</td>
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<tr>
<td></td>
<td>Bacterial—Abscess, Rocky Mountain spotted fever, ehrlichiosis, bartonellosis</td>
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<td></td>
<td>Protozoal—Toxoplasmosis, neosporosis</td>
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<td></td>
<td>Mycotic—Cryptococcosis, blastomycosis, others</td>
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<tr>
<td></td>
<td>Noninfectious meningoencephalitides—Granulomatous meningoencephalitis, necrotizing meningoencephalitides</td>
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<tr>
<td>Trauma</td>
<td>Brainstem trauma</td>
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<tr>
<td>Toxic</td>
<td>Metronidazole&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>Vascular</td>
<td>Cerebrovascular disease&lt;sup&gt;a&lt;/sup&gt;</td>
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<sup>a</sup> Discussed in this article.
dysfunction. Thyroid hormone supplementation results in rapid improvement within a few days.

**Intracranial neoplasia**

Meningiomas, which are the most common primary intracranial tumor in both dogs and cats, have a propensity to develop along the lateral and ventral surfaces of the cerebellopontomedullary region.\(^{33}\) Choroid plexus tumors also commonly develop at the cerebellopontomedullary angle and within the fourth ventricle.\(^ {34}\) Gliomas may develop anywhere within the brainstem parenchyma. In these cases, central vestibular signs are common, and may develop as a result of increased intracranial pressure, compression or invasion of vestibular nuclei, obstructive hydrocephalus, or a brain herniation due to a neoplasm causing mass effect from any location within the brain.

The preferred method of presumptive antemortem diagnosis of intracranial neoplasia is MRI, as CT causes beam-hardening artifact that may preclude visualization of small lesions in the cerebellum, pons, and medulla. The MRI characteristics of common canine and feline intracranial neoplasms have been well defined (Fig. 8), and often allow for accurate and noninvasive prediction of the histologic tumor type.\(^ {35-37}\) However, definitive diagnosis of intracranial neoplasia requires tumor biopsy. Although analysis of CSF typically reflects nonspecific abnormalities, exfoliated neoplastic cells may be detected in animals with choroid plexus carcinomas and CNS lymphoma.\(^ {34}\)

In cases of infratentorial neoplasia, the prognosis is likely dependent on the histologic type of the tumor, the severity of tumor-associated neurologic dysfunction, the neuroanatomic location and extent of the neoplasm, and the type of treatments administered. Although there are few evidence-based data in the literature that provide objective prognostic information pertaining to infratentorial tumors, the prognosis is generally considered unfavorable compared with that of supratentorial tumors. Intra-axial tumors (gliomas) are typically associated with a worse prognosis than extra-axial tumors (meningioma, choroid plexus tumor; Fig. 9), and the severity of neurologic dysfunction is also considered to be negatively correlated with outcome.

![Fig. 8. Axial T1-weighted, postcontrast MRI scan obtained from a dog with left head tilt, vertical nystagmus, and left hemiparesis. A uniformly enhancing, extra-axial mass lesion is present in the left ventrolateral aspect of the medulla (arrows). A transitional meningioma was confirmed at necropsy.](image-url)
In the infratentorial region, cytoreductive surgery is usually limited to cases with extra-axial neoplasia. Primary or postoperative adjunctive external beam radiotherapy (fractionated or stereotactic) has been shown to be beneficial for improving the quality of life and prolonging survival in animals with brain tumors.\textsuperscript{38} Palliative treatment with corticosteroids (0.5–1.0 mg/kg/d by mouth) may temporarily improve clinical signs.

**Meningoencephalitis**

Multiple infectious agents and noninfectious inflammatory diseases (see Table 4) can involve the central vestibular system. Depending on the causative agent, central vestibular signs may be the predominant clinical manifestation, part of a multifocal CNS presentation, or a component of a polysystemic clinical disease. The pathogenesis, diagnosis, and management of the meningoencephalitides are covered in detail in this issue and elsewhere.\textsuperscript{1,2,39}

**Metronidazole toxicosis**

Metronidazole administration can cause central vestibular disease or vestibulocerebellar signs, particularly in dogs.\textsuperscript{40,41} Neurologic toxicity has usually been reported to occur following subacute to chronic administration of metronidazole doses that exceed 60 mg/kg/d.\textsuperscript{40–43} However, individual animal susceptibilities to the toxic effects of this drug are apparently variable, as toxicity has been observed at lower doses in both dogs and cats. Felines may commonly present with neurologic signs referable to forebrain dysfunction, such as seizures, blindness, or alterations in consciousness.\textsuperscript{42,43} The exact mechanism of toxicity is unknown, but is theorized to be modulated by \( \gamma \)-aminobutyric acid receptors in the vestibulocerebellum.\textsuperscript{41} Diagnosis is based on an appropriate history of exposure and clinical signs. Treatment should include cessation of metronidazole therapy and supportive care. The recovery time with nonspecific, supportive therapies is 1 to 2 weeks. It has been demonstrated that administration of diazepam (0.5 mg/kg intravenous once; then 0.5 mg/kg by mouth every 8 hours for 3 days) greatly expedited improvement and recovery from metronidazole toxicity in dogs.\textsuperscript{41} Dogs treated with diazepam recovered in 1.5 days versus 11 days for dogs receiving supportive care only.

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**Fig. 9.** Axial, T1-weighted, postcontrast MRI scan obtained from a dog with paradoxic central vestibular signs consisting of a right head tilt, rotary nystagmus, and left hemiparesis. An extra-axial, hyperintense mass (choroid plexus tumor) is present in left cerebelloponmotedullary region (arrow).
Cerebrovascular disease
Ischemic infarctions and transient ischemic attacks (TIAs) have been increasingly recognized as a cause of acute-onset, focal, and nonprogressive central and paradoxical vestibular signs in dogs, and to a lesser extent, cats. TIAs are characterized by an abrupt onset, brief (<24 hours), focal neurologic disturbance that results from functional ischemia. TIAs may precede infarctions visible on imaging studies. Central vestibular dysfunction resulting from ischemic infarcts can result from infarction of the medullary components of the central vestibular apparatus or the vestibulocerebellum. Cerebellar ischemic infarcts typically appear wedge-shaped and are hypointensifying on CT images. With MRI, ischemic infarcts demonstrate T1 iso- to hypointensity, T2 and fluid-attenuated inversion recovery hyperintensity, and mild to absent contrast enhancement depending on the timing of imaging in relation to the onset of clinical signs. Cerebellar infarctions often topographically appear as territorial lesions that occur within the distribution of the rostral cerebellar artery. Diagnosis of infarctions is greatly supported by performing diffusion-weighted and T2* gradient-echo images. Spaniels and spaniel-crosses may be predisposed to cerebellar infarctions. In cases in which infarcts are suspected, the animal should be evaluated for underlying hypertension, hyperadrenocorticism, hypothyroidism, and cardiac or renal disease. Many animals with infarcts in this area will improve with time and supportive care. The future risk for infarction and neurologic-associated mortality is significantly higher in dogs with infarcts in which a predisposing medical condition is identified.

SUMMARY
The vestibular system is the primary sensory modality that participates in the maintenance of balance. Clinical signs of vestibular disease include asymmetric ataxia, head tilt, and pathologic nystagmus. Neuroanatomic localization of observed vestibular signs to either the peripheral or central components of the vestibular system is paramount to the management of the patient with vestibular dysfunction, as the causes, diagnostic approaches, and prognoses are dependent on the neuroanatomic diagnosis. This article reviews functional vestibular neuroanatomy, as well as the diagnosis and treatment of common causes of small animal vestibular disease.

REFERENCES