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Abstract: The vestibular system is responsible for keeping an animal oriented with respect to gravity. It is a sensory system that maintains the position of the eyes, body, and limbs in reference to the position of the head. Proper interpretation of neurologic deficits and precise neuroanatomic localization are essential to diagnose and prognosticate the underlying disorder. Neurologic examination can confirm whether the vestibular dysfunction is of peripheral or central nervous system origin. Idiopathic vestibular syndrome is the most common cause of peripheral vestibular disease in dogs and, despite its dramatic clinical presentation, can improve without intervention. Central vestibular diseases generally have a poorer prognosis.

Anatomy and Physiology

This article describes the clinically relevant anatomic components of the vestibular system and the clinical signs commonly seen in vestibular system dysfunction, with particular emphasis on the question of how to distinguish central from peripheral vestibular disease. A companion article describes some common specific vestibular diseases and their diagnostic differentials.

For more information, please see the companion article, “Vestibular Disease: Diseases Causing Vestibular Signs.”

Figure 1. Schematic representation of the components of the inner ear and related structures.
Clinical Signs

It is important to recognize when an animal has vestibular disease (BOX 1). Once the presence of a vestibular disorder is confirmed, the next step is to determine whether the animal has central or peripheral disease (TABLE 1).

A head tilt is one of the more common signs indicating a vestibular disorder (FIGURE 3). It is characterized by rotation of the median plane of the head, with one ear held more ventral than the other. This abnormal posture results from a loss of tone to the antigravity muscles of the neck, either ipsilaterally or contralaterally (depending on the site of the lesion). This is different from a head turn (torticollis) in which the median plane of the head remains perpendicular to the ground but the nose is turned toward the body. A head turn usually indicates an ipsilateral forebrain lesion and is not a sign of vestibular disease, although it can be seen with syringomyelia of the cervical spinal cord.

Nystagmus is a characteristic eye movement in animals with vestibular disease, specifically jerk nystagmus, in which there is a quick phase and a slow phase. There are two major categories of jerk nystagmus: physiologic and pathologic.

Physiologic jerk nystagmus (or vestibular eye movements) can be induced in a normal animal by turning the head from side to side. Initially, a slow drift of the eye is seen away from the direction of travel, followed by a fast compensatory phase in the direction of movement of the head. A reduction or lack of vestibular eye movements is considered abnormal and can be seen with unilateral or bilateral vestibular disorders, respectively.

Pathologic jerk nystagmus is seen when the vestibular system is dysfunctional. The eyes have a tendency to spontaneously drift in the direction of the lesion (slow phase) and, via a brainstem mechanism (involving the medial longitudinal fasciculus), quickly return to their initial location (fast phase). This type of abnormal nystagmus can be seen at rest (spontaneous nystagmus) or may only occur with abnormal head positions (positional nystagmus). Horizontal, vertical, and rotary nystagmus may be present. As a general rule, if vertical nystagmus is seen, a central vestibular lesion should be suspected. Horizontal and rotary nystagmus only signify vestibular disease and do not distinguish a central from a peripheral lesion.

Strabismus results from a loss of vestibular control over maintenance of the normal eye position within the orbit. Vestibular information is projected through the medial longitudinal fasciculus to cranial nerves III, IV, and VI. If this input is abnormal, strabismus may be seen when the head position is perturbed and occasionally may be seen with the head in a normal position. Strabismus and nystagmus may be concurrent. Ventral or ventrolateral strabismus is most commonly observed ipsilaterally to the 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 spinal cord, brainstem, or peripheral nerve disorder (general proprioceptive ataxia).

Circling is not pathognomonic for vestibular disease; it is also seen in patients with forebrain lesions. If the circles are small and poorly completed, then vestibular disease is most likely. However, if they are large and complete, a forebrain disorder should be considered.

**Central or Peripheral?**

In a 10-minute consultation, it can be difficult to prioritize which neurologic deficits should be evaluated. However, the decision of whether a lesion is central or peripheral has a significant impact on the likely prognosis and outcome for the patient. In a patient with vestibular disease, the lesion lies within the brain (central disease), cranial nerve VIII, or an ear (peripheral disease). Diagnosis and management of central lesions generally require more expensive diagnostics and therapies, and the common causes of central vestibular disease are often associated with a guarded prognosis. In contrast, peripheral vestibular lesions are broadly associated with a good prognosis (with some exceptions). The following clinical signs can be used to help determine whether vestibular disease is central or peripheral in origin. These signs are also listed in **TABLE 1** and **FIGURE 4**.

Horner syndrome (miosis, enophthalmos, protrusion of the third eyelid, ptosis of the upper eyelid) is usually seen only with peripheral vestibular disease. The sympathetic nerve supply to the eye is near the vestibular nerve between the petrous temporal bone and tympanic bulla (**FIGURE 1**). Facial paralysis may occur in either central or peripheral disease, as cranial nerve VII (the facial nerve) follows a similar course to the vestibular nerve as it runs through the petrous temporal bone (**FIGURE 1**).

Lesions affecting the central vestibular system typically produce clinical signs suggestive of brainstem and/or cerebellar involvement in addition to the potential signs of peripheral disease. The reticular formation is integrally associated with the brainstem, as are the ascending and descending motor and sensory pathways (i.e., the long tracts) to the limbs. Therefore, abnormal mentation, hemiparesis, and postural reaction deficits are commonly associated with central vestibular disease (**FIGURE 4**). Note that when testing conscious proprioception in an animal with a vestibular disorder, it can be difficult to balance the patient’s weight evenly, so false-positive reactions may be seen. If the animal is small, then tactile placing responses are useful; however, in a larger animal, this is not practical, so hopping and paw placing should be evaluated by supporting the animal's weight evenly.

Deficits of cranial nerves V to XII can also be associated with a central vestibular lesion due to the association of these nerves with the brainstem. A general rule to follow is that if two or more cranial nerves are affected (in addition to cranial nerve VIII), a central lesion should be suspected. However, an exception to this rule does exist (see below).

The absence of specific central signs (**TABLE 1**) does not rule out a central vestibular lesion. However, their presence makes central disease far more likely.

Occasionally, patients with peripheral vestibular disease have polyneuropathies. These animals have normal mentation with single or multiple cranial nerve deficits, such as dysphagia (cranial

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**Table 1. Clinical Signs That Distinguish Central and Peripheral Vestibular Disease**

<table>
<thead>
<tr>
<th>Clinical Sign(s)</th>
<th>Peripheral</th>
<th>Central</th>
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<tbody>
<tr>
<td>Head tilt</td>
<td>Toward the lesion</td>
<td>Toward the lesion (or away from the lesion with a paradoxical head tilt)</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Horizontal or rotary</td>
<td>Horizontal, rotary, or vertical</td>
</tr>
<tr>
<td>Postural reaction deficits</td>
<td>Normal</td>
<td>Deficits on the same side as the lesion</td>
</tr>
<tr>
<td>Horner syndrome</td>
<td>Possible on the side of the lesion (postganglionic)</td>
<td>Not as common as with peripheral vestibular disease (preganglionic)</td>
</tr>
<tr>
<td>Consciousness</td>
<td>Normal mentation (disorientation possible)</td>
<td>Altered mentation</td>
</tr>
<tr>
<td>Cranial nerve deficits</td>
<td>Cranial nerve VII, on the side of the lesion</td>
<td>Involvement of multiple cranial nerves (most commonly, cranial nerves V and VII, although others may be involved)</td>
</tr>
<tr>
<td>Cerebellar signs</td>
<td>No</td>
<td>On the same side as the lesion</td>
</tr>
</tbody>
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*Signs in bold are only seen in central vestibular disease*
Vestibular Disease: Anatomy, Physiology, and Clinical Signs

nerve IX and X), tongue weakness (cranial nerve XII), jaw weakness and masticatory muscle atrophy (cranial nerve V), and facial paralysis (cranial nerve VII). Paresis of the limbs is rarely present, although decreased tone and reduced segmental spinal reflexes may be observed.

Central Vestibular Disease With a Paradoxical Head Tilt
A head tilt and balance loss are occasionally appreciated in a patient that has proprioceptive deficits that are opposite to the direction of the head tilt (FIGURE 4). These specific clinical signs are seen when a lesion involves the vestibular apparatus of the cerebellum (i.e., the caudal cerebellar peduncle or flocculonodular lobe) on the side of the body opposite to that of the head tilt. The reason for this is that the cerebellar output is largely inhibitory, so it decreases activity within the ipsilateral vestibular nuclei (FIGURE 4). The head tilt in any vestibular syndrome is always to the side of least activity within the vestibular nuclei. If this inhibitory influence is lost (e.g., due to a severe cerebellar lesion), then activity in the ipsilateral vestibular nuclei is increased, causing the head to tilt to the side opposite the lesion. Clinically, the recognition of proprioceptive deficits on the opposite side to a head tilt should alert the observer to the possibility of a central vestibular lesion.

Bilateral Vestibular Disease
Bilateral vestibular disease (BVD) is rare and is most commonly peripheral in origin, although it can, theoretically, occur in very small focal and symmetric central lesions (e.g., due to metabolic, nutritional, or toxic causes such as thiamine deficiency). Diffuse, large central lesions are unlikely to cause BVD as they would severely disrupt the reticular formation, resulting in coma or death. Animals with BVD are reluctant to walk, exhibiting vestibular ataxia and wide head excursions, swinging their head from one side to the other. It is uncommon to see a head tilt or pathologic nystagmus in these patients, although normal physiologic nystagmus is absent.

References
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
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| 1. The peripheral components of the vestibular system | a. are not closely associated with the sympathetic trunk.  
| | b. include the vestibular nuclei in the medulla.  
| | c. are contained within the inner ear (the petrosal portion of the temporal bone).  
| | d. none of the above  |
| 2. In a dog with vestibular disease, the characteristic head tilt results from | a. loss of tone to the antigravity muscles of the neck.  
| | b. fluid in the tympanic bulla.  
| | c. increased intracranial pressure.  
| | d. none of the above  |
| 3. In a dog with vestibular disease, which statement describes the characteristic head tilt? | a. The ventrally deviated ear is usually on the opposite side from the lesion.  
| | b. The ventrally deviated ear is usually on the same side as the lesion.  
| | c. It is difficult to differentiate from torticollis in a dog with a central vestibular lesion.  
| | d. none of the above  |
| 4. Which statement is true regarding pathologic jerk nystagmus in a dog with vestibular disease? | a. Vertical nystagmus can indicate a central vestibular lesion.  
| | b. The slow phase is usually toward the side of the lesion.  
| | c. The nystagmus may resolve if the head is held in normal position.  
| | d. all of the above  |
| 5. Which statement is true regarding physiologic jerk nystagmus? | a. It can be induced in a normal animal by rotating the head to simulate a head tilt.  
| | b. If the head is turned from side to side, a slow drift of the eye is seen toward the direction of travel followed by a fast compensatory phase in the opposite direction of movement of the head.  
| | c. A reduction or loss of physiologic jerk nystagmus is abnormal and can indicate pathology.  
| | d. all of the above  |
| 6. Which statement is true regarding Horner syndrome? | a. It is characterized by ptosis, miosis, enophthalmos, and nystagmus.  
| | b. It is usually not seen with central vestibular disease.  
| | c. It is associated with a poor prognosis if it occurs with vestibular disease.  
| | d. all of the above  |
| 7. Which clinical finding(s) is/are normally associated with central vestibular disease? | a. abnormal mentation  
| | b. hemiparesis and postural deficits  
| | c. deficits involving two or more cranial nerves (in addition to cranial nerve VIII)  
| | d. all of the above  |
| 8. Which clinical finding(s) might characterize a dog with vestibular signs associated with ear disease? | a. normal mentation  
| | b. facial paralysis  
| | c. Horner syndrome  
| | d. all of the above  |
| 9. Deficits of cranial nerve(s) ______ are more likely to occur in a dog with central vestibular disease. | a. III  
| | b. IV and VI  
| | c. V, VII, and VIII  
| | d. VIII  |
| 10. Which statement is false regarding bilateral vestibular disease? | a. It is most commonly associated with central vestibular disease.  
| | b. Ataxia may be observed.  
| | c. A head tilt is uncommonly observed.  
| | d. Pathologic nystagmus is unlikely to be observed.  |