Imagine you are trapped inside of a clothes dryer - spinning and rolling, trying to get a bearing on which way is up, and all while feeling like you’re about to vomit. This is what our patients experience when their vestibular system isn’t working properly. Trying to localize, treat and prognosticate these patients can be enough to make your head spin (pun intended), but with a few simple tricks, you’ll be right-side-up in no time - and so will your patients!

You can think of the vestibular system like the gyroscope in your smart phone. When you turn your phone horizontally, the gyroscope senses its position in space and adjusts the screen to accommodate. In animals, this “gyroscope” is the vestibular system, which can be separated into two basic components; the peripheral vestibular system (PVS) and the central vestibular system (CVS). The PVS is comprised of the inner ear and cranial nerve VIII (CNVIII), which senses the position of the eyes in relation to the head. The CVS includes pathways within the brainstem and cerebellum (medulla oblongata and flocculonodular lobes), which process sensory information from the PVS and send signals to the rest of the body on how to stay upright.

Anything interrupting these pathways can cause the combination of clinical signs we commonly see that are collectively referred to as Vestibular Syndrome:

**HEAD TILT**: A head tilt is described as a rotation of the head about the atlas bone, resulting in one ear being held lower than another. Typically, the head is tilted towards the lesion.

**ATAXIA & FALLING**: Vestibular patients are ataxic because their body is not properly communicating how to fight gravity. If your friend runs up and shoves you, your body feels you begin to fall and instructs your limbs on how to catch yourself. In vestibular patients, this function isn’t working correctly, so they are off balance and may fall. Typically, they are falling or stumbling towards the lesion.

**NYSTAGMUS**: An involuntary, rhythmic movement of the eyeballs is termed nystagmus. In vestibular patients, there is commonly a slow, drifting of the eyes to one direction (the “slow-phase”), and there is not a consistent change in anatomy, it is incorrect to refer to it as a disease. It is referred to it as a syndrome - a collection of clinical signs often observed together; or simply “vestibular dysfunction”.

**Vestibular Disease**

“Vestibular Disease” is actually a misnomer. There are many diseases that can cause vestibular dysfunction, but since there are many causes (or in some cases a cause cannot be found), and there is not a consistent change in anatomy, it is incorrect to refer to it as a disease. It is referred to it as a syndrome - a collection of clinical signs often observed together; or simply “vestibular dysfunction”.

This is easy to interpret if the nystagmus is along a horizontal plane, however, sometimes the eyes can jerk along a vertical plane or even in a rotary fashion. In these cases, it is more likely to be a central lesion since the deficit is not lateralized, although bilateral peripheral lesions can cause this as well.
HORNER’S SYNDROME AND FACIAL PARALYSIS: Due to the close proximity of CNVIII to the sympathetic innervation of the eye, patients with vestibular dysfunction may also display Horner’s Syndrome. This is characterized by constriction of the affected pupil, sinking of the globe into the orbit, protrusion of the third eyelid and drooping of the eyelids.

CNVII, which controls the muscles of facial expression, also runs along a similar path. Dysfunction of CNVII can cause paralysis of the ear, eyelids, nose, cheeks and lips.

These deficits would appear on the same side as the lesion.

CONSCIOUS PROPRIOCEPTION (CP): You might be asking yourself what conscious proprioception is doing in this paper, seeing as it is mostly a test of spinal cord function, and you’d be right! No part of the PVS would affect proprioception, however, a lesion within the CVS may. If you find proprioceptive deficits in a vestibular patient the lesion is likely central, or your patient is unlucky enough to have a concurrent spinal cord issue.

Paradoxical Vestibular Dysfunction is when your clinical signs are not lateralized. This “paradox” of clinical signs localizes to one specific location within the CVS, the cerebello-medullary junction. An example of this would be a right head tilt and falling to the right, fast-phase left nystagmus and CP deficits on the left, everything points to a right-sided lesion except for the CPs. In this situation, the lesion will be on the side of the CP deficits. It’s safe to assume that paradoxical vestibular patients, and vestibular patients with CP deficits, have a central lesion.

To diagnose the lesion, a minimum database should first be performed. Hypothyroidism has been known to cause vestibular dysfunction in dogs, so a T4 and TSH should also be performed. While the middle ear cannot be visualized on external examination, evidence of infection, polyps or masses may be detectable in the external canal. Oblique skull radiographs and CT may allow good visualization of the osseous bulla, and if a middle ear infection is suspected a myringotomy can be performed to obtain a cytology and culture of the fluid. Ultimately, MRI is considered the gold standard for visualizing the inner ear and brain at the same time to determine the location and cause of the lesion.

In some instances, diagnostics do not identify a lesion. In these cases, Idiopathic Vestibular Syndrome (sometimes referred to as “Old Dog Vestibular Syndrome”) is the diagnosis of exclusion. These patients typically are suddenly and severely affected, but often have a good prognosis and show improvement within 2-3 days.
If a lesion can be identified, treatment is aimed according to the lesion. This may involve antibiotics or surgery for a middle ear infection, immunoregulators for inflammatory diseases, surgical or chemotherapeutic intervention for neoplasia, surgical removal for polyps or managing factors that may contribute to a stroke (renal failure, hypertension, cardiac disease) or metabolic cause (hypothyroidism, Thiamine deficiency, metronidazole toxicity) or just supportive care for idiopathic cases. Prognostically, neoplasia tends to carry the worst prognosis, followed by inflammatory, infection and metabolic. Prognosis for stroke varies on the underlying cause, but if future stroke events can be avoided patients commonly recover slowly on their own. It is important to note that severity of signs does not necessarily correlate to a poorer prognosis. Some patients may have a lingering head tilt and become less oriented on car ride or while swimming long term. Until clinical signs improve, supportive care is very important and can be challenging for these patients. Meclizine or Cerenia can be used to treat motion sickness and diazepam to help with anxiety and distress. Patients tend to lose orientation when lifted, and should be securely supported on the side they are falling to. Patient’s may prefer to lay on the side they are falling to, making it difficult to rotate sides. Extra care should be taken to prevent bad sores, including 3-4 inches of foam bedding and attempting to keep patients propped in sternal recumbency. Patients may need assistance eating and drinking until they become more oriented. This can be accomplished in hospital or at home depending on client comfort.

Myringotomy

A surgical procedure in which a needle is inserted into the inner ear through the eardrum (if present) to relieve pressure caused by excessive buildup of fluid and to collect and drain fluid from the middle ear.

REFERENCES