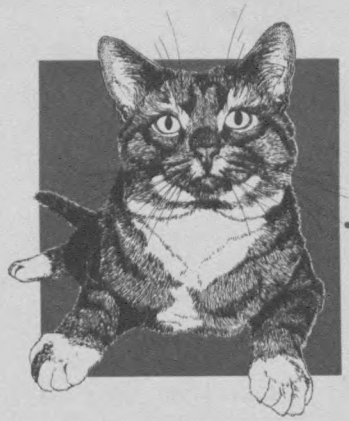

Cornell Feline Health Center
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Peripheral Vestibular Diseases in the Cat

Victoria W. Pentlarge, D.V.M.

Peripheral vestibular dysfunction (PVD) results from diseases that interfere with the vestibular division of the vestibulocochlear nerve (cranial nerve VIII). In the cat, PVD occurs frequently. It is usually characterized by ataxia, abnormal body positions and nystagmus. Anatomy, clinical signs, and differential diagnoses are discussed to aid the practitioner when encountering cats with PVD.

Anatomy

Vestibular receptors are located within the membraneous labyrinth which is encased in the bony labyrinth of the inner ear. The bony labyrinth, part of the petrosal bone, is primarily visible on the inside of the braincase. The vestibular ganglion is located within the petrosal bone. The vestibular receptors in the four communicating fluid-filled compartments of the membraneous labyrinth sense changes in head position, acceleration, and gravity. This information is transmitted to the brain via the vestibulocochlear nerve. Most of the axons terminate in the vestibular nuclei located in the medulla. Some axons continue to the cerebellum by way of the caudal peduncle.

Spinal cord, brain stem and cerebellar efferent vestibular pathways coordinate the position and activity of the eyeballs, limbs and trunk in relation to head movements and position, and the earth's gravitational field. Consequently, vestibular disturbances result in varying degrees of loss of equilibrium and alterations in position of the eyes, limbs, and trunk in reference to the head. (Refer to illustration on p. 3.)

Clinical Signs

Signs of unilateral PVD include asymmetric ataxia, head tilt, circling, falling, rolling, truncal curvature, hypertonia, hyperreflexia, abnormal nystagmus, strabismus, and vomiting. The loss of balance occurs without paresis. Postural reactions and proprioception are normal. Mild hypertonia and hyperreflexia may be present in the contralateral limbs. Stimulation of the vomiting center causes the vomiting and anorexia observed in some cats during the early stages of dysfunction.

The ventral ear in head tilts is ipsilateral to the side of the lesion. Falling, rolling, circling and lateral flexion of the trunk are toward the side of the lesion.

These signs and contralateral hypertonia and hyperreflexia can be explained by the following: vestibular neurons are tonically active and a unilateral loss of function removes facilitation of the ipsilateral extensor muscles and inhibition of the contalateral extensor muscles of the limbs and trunk[1]. The functioning contralateral vestibular system results in unopposed extensor muscle tonus which forces the trunk toward the side of the lesion.

Abnormal nystagmus is spontaneous or positional. With spontaneous nystagmus, the involuntary eyeball oscillations occur

when the head is in a normal position. If the nystagmus occurs only when the head is held in a different position, then it is called positional. The direction of the nystagmus does not change with different head positions in PVD. Nystagmus direction is defined by the direction of the oscillation's quick jerk. In PVD, the quick phase is always away from the side of the lesion and is horizontal or rotatory. Vertical nystagmus only occurs with central vestibular disturbances. Ventral strabismus of the ipsilateral eyeball may be observed when the neck is extended. Normally, the eyeball should remain in the center of the palpebral fissure for different head positions.

In cats with bilateral PVD, clinical signs include symmetric ataxia (ie.,loss of balance to either side) and wide head excursions. If there is complete bilateral receptor destruction, then normal and abnormal nystagmus is absent. Frequently, these cats are frightened and remain crouched on the ground with limbs spread apart due to their disorientation. Consequently, the ataxia may be difficult to observe if the cat is reluctant to move.

The stage of the disease will influence the clinical presentation of a cat with PVD. With time, some signs resolve or the animal learns to compensate. Vomiting, anorexia, circling, wide head excursions, and falling may decrease in intensity during the course of the disease.

Diseases that damage the vestibular division of cranial nerve VIII can also damage other nearby nerves (ie., the cochlear division of cranial nerve VIII, facial nerve, and sympathetic axons that innervate the eyeball). Part of the facial nerve in the facial canal of the petrosal bone is separated from the middle ear by only a small amount of connective tissue. Therefore, middle ear diseases can interfere with facial nerve function. Impaired facial nerve function results in ipsilateral facial paresis or palsy. Postganglionic sympathetic axons pass through the middle ear cavity on their course to the ocular and periocular smooth muscles. Disruptions to these sympathetic axons causes ipsilateral enophthalmia, miosis, protrusion of the third eyelid, and a narrowed palpebral fissure (ie.,Horner's Syndrome). Damage to the cochlear division results in deafness which is difficult to clinically detect in cats with unilateral PVD.

Differential Diagnoses

The differential diagnoses for PVD in the feline includes congenital disorders, trauma, receptor degeneration, neoplasia, idiopathic-vestibular neuropathy, and otitis media-interna[1]. Neoplasia, trauma, and otitis media-interna can also cause functional disturbances of the facial nerve and sympathetic axons that innervate the eyeball[1-5].

Congenital disorders have been described in Siamese and Burmese kittens[1]. Common signs include head tilt and ataxia. A hereditary basis is suspected and therefore, affected cats should not be bred.

Cornell Feline Health Center
Veterinary News

A publication for veterinary professionals

The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere, by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

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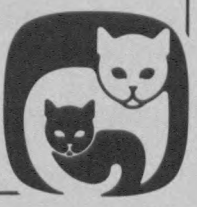

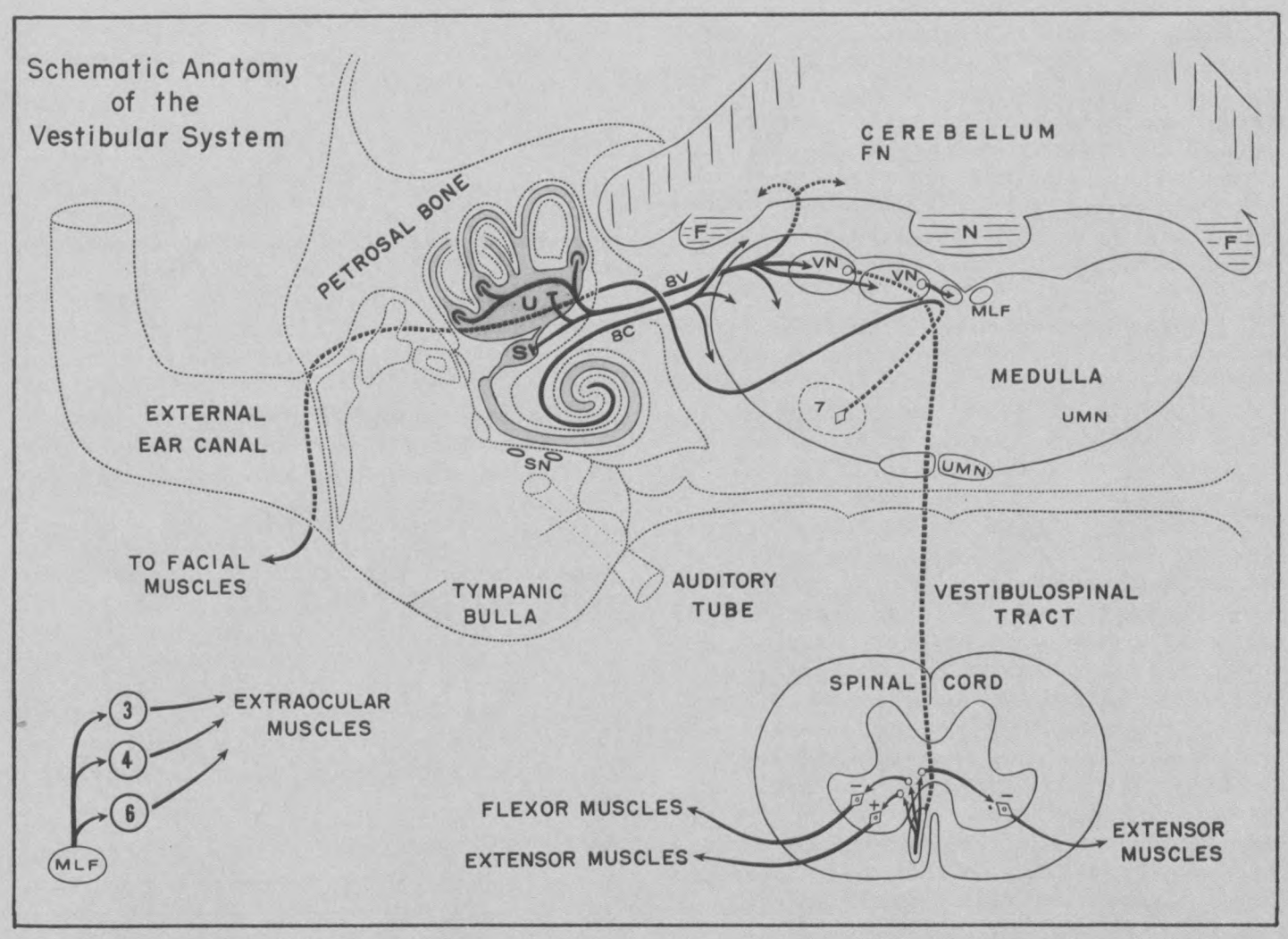

Schematic anatomy of the vestibular system. N: nodulus; F: flocculus; FN: fastigial nucleus; UMN: upper motor neuron; MLF: medial longitudinal fasiculus; VN: vestigular nucleus; 8V: cranial nerve VIII, vestibular portion; 8C: cranial nerve VIII, cochlear portion; U: utricle; S: saccule; SN: sympathetic neurons; 3: oculomotor nucleus; 4: trochlear nucleus; 6: abducent nucleus; 7: facial nucleus.

Illustration was reprinted from the second edition of "Veterinary Neuropathy and Clinical Neurology" by Dr. deLahunta with permission from the W.B. Saunders Co.

Traumatic fracture of the bony labyrinth may damage the membraneous labyrinth thereby causing PVD. The most common causes of head trauma in cats are car accidents and falling from multistory buildings. Skull and bulla radiographs are useful to ascertain the extent of bony damage. Treatment may include rest, local and systemic antibiotics, glucocorticoids, and maintenance of fluid, electrolyte, nutrient, and caloric requirements.

Neurofibromas of the vestibulocochlear nerve can produce PVD[1]. As the neurofibroma grows, brain stem signs may develop due to brain stem compression. Peripheral vestibular disturbances caused by middle and inner ear squamous cell carcinoma, ceruminous gland carcinoma, and fibrosarcoma have been reported[2-5]. Neoplasia should be suspected whenever there are destructive lesions radiographically.

Aminoglycoside antibiotics can cause degeneration of the vestibular and/or cochlear labyrinth receptors. In cats, streptomycin is more likely to damage the vestibular system receptors whereas the other aminoglycosides usually affect the auditory receptors[1]. If the drug is withdrawn early, the PVD may resolve. However, hearing usually does not return.

Idiopathic-vestibular neuropathy is also known as feline idiopathic vestibular disease. The pathogenesis is unknown but intoxication of the vestibulochochlear nerve has been suggested based on the absence of necropsy lesions[2]. The disease primarily occurs in the summer and early fall in the northeast. All ages of cats are susceptible. It is characterized by an acute onset of severe peripheral vestibular signs. The remainder of the physical and neurologic examination, including the otoscopic examination, is normal. Marked improvement of clinical signs occurs within one week with continued improvement over the next 2-3 weeks. Specific therapy is not indicated. Glucocorticoids do not seem to alter the course of the disease. A head tilt may be a persistent sign. Recovered cats, if stressed, may develop ataxia.

Otitis media-interna causes peripheral vestibular signs when the inflammation impairs the function of the membraneous labyrinth. The inflammation may be due to bacterial, fungal, protozoal, and/or parasitic infections. Otitis media-interna usually develops secondarily from otitis externa. In addition, the source of the infection may be blood borne or via extension from the nasopharynx by way of the auditory tube which communicates with the middle ear. Characteristic findings during a diagnostic workup may include otoscopic evidence of otitis externa and/or radiographic changes of the bulla (ie., middle ear) and possibly of the bony labyrinth. If possible, exudate should be obtained for cytology and cultures. Treatment of bacterial otitis media-interna requires prolonged topical and systemic antibiotics. A bulla osteotomy may be indicated in all types of otitis media-interna for obtaining culture and biopsy samples and for drainage.

Summary

In conclusion, PVD is usually characterized by ataxia and abnormal body positions and nystagmus. Concurrent involvement of the facial nerve and sympathetic axons localizes the lesion to the middle and inner ear. The different etiologies of PVD in the cat include congenital disorders, trauma, receptor degeneration, neoplasia, idiopathic-vestibular neuropathy, and otitis-media interna. These different causes of PVD influence therapy and prognosis.■

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Victoria W. Pentlarge received her DVM degree from the University of California at Davis. Her internship was in small animal medicine and surgery and her residency was in small animal medicine at Cornell University.

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Understanding Coronaviral Serology Titers

Healthy cats are often needlessly euthanized because their serum has been found to be "FIP-positive." It is assumed that the cats have feline infectious peritonitis which is usually considered to be a fatal disease. Unfortunately, one cannot predict the development of FIP or definitively diagnose FIP on the basis of such positive tests.

The Disease

FIP is a chronic, progressive, and usually fatal immunologically-mediated disease caused by a coronavirus. Feline infectious peritonitis virus (FIPV) is closely related to several other animal coronaviruses, including transmissible gastroenteritis virus (TGEV) of swine, canine coronavirus (CCV), and human respiratory coronavirus (HCV 229E). In addition, there are coronaviruses which infect cats but do not cause FIP.

The Tests

FIP tests detect coronavirus antibodies in cat serum; they do not detect the actual coronavirus. They are therefore quite different from the FeLV tests which actually detect feline leukemia virus in cat blood. Detection of coronavirus antibodies indicates only prior exposure to a coronavirus. Since exposure to FIPV, feline coronaviruses which do not cause FIP, and coronaviruses of other species can result in positive test results, these tests should really be referred to as "coronavirus antibody tests." Coronavirus antibody tests cannot determine whether or not a cat is presently infected, is a virus carrier/shedder, has the disease, or, if seropositive, which coronavirus was responsible.

There are three basic serologic techniques used to detect coronavirus antibodies: indirect immunofluorescence assay (IFA), virus neutralization, and enzyme-linked immunosorbent assay (ELISA). The IFA is most widely used by clinical and diagnostic laboratories, although exact procedures vary greatly between laboratories. To further complicate matters, the tests are either homologous or heterologous, depending on the source of the antigen which is used to detect the antibody. Homologous tests use FIPV; heterologous tests use the serologically cross-reactive CCV and TGEV.

The numerous tests employed by different laboratories and the lack of standardized testing protocols compound the difficulties in interpreting coronavirus antibody titers. Conflicting titer results should be expected when a serum sample is tested by different laboratories using different serologic techniques, or even by different laboratories using the same technique.

Role of Recent Vaccination

Titer intrepretation has been further complicated by the discovery that recent vaccination can cause false-positive coronavirus antibody results in those assays which are performed without the benefit of proper controls. Cats can produce antibodies against bovine serum components in feline vaccines, and these antibodies can react with similar bovine serum components in the cell cultures used in the various assays. Serum samples for antibody testing should therefore be drawn no sooner than three to four months after the most recent vaccination.

Interpretation of Test Results

Serologic surveys using homologous and heterologous IFAs have shown that exposure of cats to coronavirus(es) is much more widespread than was once believed. In the general healthy feline population (excluding cats in catteries, multiple-cat households, and cats with FIP) approximately 10 to 40% will be seropositive for

coronavirus antibodies. Titers in breeding catteries are either completely absent or present in 80-90% of animals. However, FIP is a relatively uncommon disease in nature, even in crowded catteries. The vast majority of coronavirus antibody-positive cats will never develop lethal FIP.

The presence of coronavirus antibodies in any cat is indicative only of prior exposure to a coronavirus, possibly FIPV, but not necessarily. A positive coronavirus antibody titer therefore does not diagnose clinical or latent FIP. Also, a positive titer does not necessarily infer any degree of protection against the development of FIP. The only conclusive way to make a positive diagnosis of FIP is by histopathological examination of biopsy or necropsy samples.

In the general healthy feline population, seropositive cats usually have IFA titers ranging from 1:25 to 1:400. Titers of 1:400 and above are often consistent with, but not diagnostic of FIP. A greater antibody titer is generally proportional to the chronicity of the disease process (i.e., cats with non-effusive FIP tend to have higher titers than do cats with the more acute, effusive form).

There are two primary situations where the determination of coronavirus antibody titers can be useful for the practitioner and cat owner/breeder:

- As a screening test, to determine the presence or absence of antibody in a previously untested household, and to detect potential virus carriers/shedders when introducing new cats into coronavirus antibody-negative households or catteries.
- As an aid in the clinical diagnosis of a symptomatic cat with signs suggestive of FIP.

Conclusion

Available serology tests do not differentiate actively diseased from seropositive cats or identify the exact coronavirus(es) to which the cat may have been exposed. Therefore, there is no known medical reason for destroying healthy seropositive cats. ■

This article was adapted from:

Barlough JE, Jacobson RH, and Scott FW: Feline coronaviral serology. Feline Practice, 13(3): 25-35 (1983).

Barlough JE and Stoddart CA: Feline infectious peritonitis. Cornell Feline Health Center Information Bulletin, Vol. 6 (1984).

You will soon be receiving our new scientific bulletin on feline infectious peritonitis which provides more detailed and up-to-date information on this complex and frustrating disease.

Clinic Clips

A client brings a blue-smoke Persian cat into your clinic for a routine checkup. During the examination you note that the cat's eyes have a yellowish appearance, cataracts are developing and there is spontaneous nystagmus. After taking a blood sample, the venipuncture site continues to bleed beyond the normal clotting time. Other than these few signs the cat appears to be healthy.

Your lab technician reports that the blood smear, stained with Wright's stain, has unusual granules in the cytoplasm of the neutrophils. (Refer to photo.)

What is your diagnosis? (For a complete explanation see page 8.)

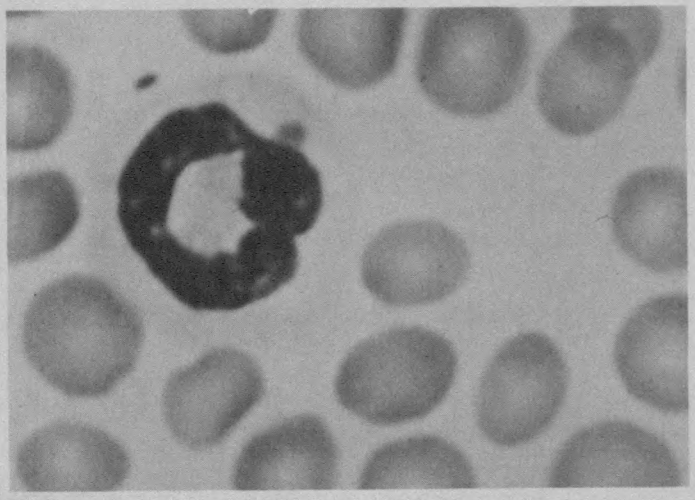

Photo courtesy of Dr. John W. Kramer, Washington State University.

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Clinic Clips *(continued from page 6)*

The clinical signs presented along with the results from the blood smear indicate that the cat is affected with a genetic disorder known as the **Chediak-Higashi syndrome.**

The blood smear and microscopic examination of hair can confirm a diagnosis based on clinical signs. The blood smear reveals large primary granules in mature and immature neutrophils. Hair shafts show large, elongated, irregular clumps of melanin.

Problems associated with the disease are not severe enough to warrant treatment. The bleeding tendency does not appear to be life-threatening, but hemostasis may be a problem if any surgical procedures are performed on an affected cat.

To date this syndrome has only been documented in blue-smoke Persians of an inbred bloodline. Other species also affected are man, mink, mice, cattle and the killer whale. In most of these species the disease is inherited as an autosomal recessive trait. The same seems to be true in cats, but this remains unproven. Suggestions about the nature of a genetic disease having such a widespread distribution have included:

1. The defect could have been carried as a genetic load throughout the phylogenetic history of the animals involved and is only occasionally expressed in the homozygous state.

2. It represents a labile genetic locus which has periodically undergone mutation possibly because of some enviornmental influence.

3. The genetic locus of CHS is not more labile than other loci, but that when the condition is expressed in the homozygous state it can be readily detected by the untrained eye because of the partial albinism.

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