



Six of the Best - Neurological Case Headaches Mini Series

Session 3: Lecture 1: Cranial Nerve Dysfunctions of the Dog, Lecture 2: Vestibular Diseases of the Dog and Cat

**Professor Simon R Platt BVM&S, MRCVS, Dipl. ACVIM
(Neurology) Dipl. ECVN
RCVS Specialist in Veterinary Neurology
College of Veterinary Medicine, University of Georgia**



Common Cranial Nerve Syndromes in Dogs

Simon R. Platt BVM&S MRCVS DACVIM (Neurology) DECVN
College of Veterinary Medicine, University of Georgia, Athens, GA

Generally, the underlying causes of neurological abnormalities of the head and face are similar whichever nerve (or nucleus) is affected, but, in addition, there are a few conditions that are specific to, or more commonly affect, certain nerves.

Neurodiagnostic investigation

A complete neurological examination is a prerequisite for investigating suspected neurological disease of the head and face. The neurological examination will help identify whether the cranial nerve dysfunction originates from a central nervous system or peripheral nervous system origin, which helps with determination of the differential diagnosis and the prognosis. Several features of the investigation may require particular attention and are described below.

a) History

A thorough history is essential because certain neurological disorders of the head and face may produce signs that cannot be observed by the veterinarian during a consultation. For instance, if an animal is reported to be vomiting, it is crucial to determine whether it is 'true' vomiting or regurgitation. The owner of any animal in which generalized weakness or exercise intolerance is reported should be questioned about the animal's vocalisation, since dysphonia is commonly associated with laryngeal dysfunction.

b) Observations

Before conducting a 'hands on' examination, several observations may aid in recognizing or classifying disorders of the head and face. Firstly, as the animal walks around a room the position of the head and neck can be observed, looking in particular for a head tilt, or generalized tremors (which may also affect the head). Examination for *symmetry* provides important clues regarding the functioning of nerves innervating musculature of the head and face; this examination thus focuses on the relative sizes of the masticatory muscle mass, facial expression, palpebral fissures and pupils. The ability to close the jaw (dependent on the motor component of the trigeminal nerves) and maintain a normal tongue position (mediated by the hypoglossal nerves) is also apparent from simple observation.

c) Neurological examination

Important functions for assessment of innervation of the head and face include the menace response, blink (or palpebral) reflex, vestibulo-ocular reflex and gag reflex. Several other tests may be of value for detecting neurological abnormalities of the head and face:

- Masticatory muscle tone is evaluated by opening the jaws
- Tongue strength is evaluated by attempting to hold the tongue; normal animals can easily withdraw their tongues (since it is wet and slippery).

d) Ancillary diagnostic tests

Hearing can be tested crudely by observing responses to noise; however, a more sophisticated and objective test is the brainstem auditory evoked response (BAER).

Schirmer tear test can be used to detect deficits in tear production.

Pharmacological testing with adrenergic agonists can be used to investigate Horner's syndrome.

Measurement of antibodies to the acetylcholine receptor can be used to substantiate the diagnosis of immune-mediated myasthenia gravis.

Measurement of antibodies to type 2M muscle fibres can be used as an aid to diagnosis of masticatory muscle myositis.

MRI or CT scanning to detect CNS lesions, especially tumors

Cisternal CSF analysis to detect inflammatory disease of the CNS or nerve roots.

Electrodiagnostics (EMG, NCV) to objectively diagnose generalised peripheral neuropathies, or to assess specific cranial nerve function such as that of the facial and trigeminal nerves.

Measurement of antibodies against various infectious agents, including Toxoplasma, Neospora, Rickettsia, Leishmaniasis.

Specific neurological syndromes of the head and face

Laryngeal paresis / paralysis

Inspiratory stridor and dysphonia are the classical clinical signs associated with laryngeal paresis or paralysis, although severe inspiratory dyspnoea, cyanosis and collapse can occasionally be identified. These signs may be sufficiently mild that animals will only present for treatment following particular periods of respiratory stress, such as prolonged exercise or extreme hot weather, at which time they may present in a hyperthermic crisis. Affected animals may also cough, especially when drinking water, and can develop clinical signs related to the secondary development of aspiration pneumonia (after they have inhaled food).

Congenital laryngeal paralysis is considered to account for up-to 30% of the cases seen in clinical practice and has been proven or suspected in several breeds including Bouvier des Flandres, Siberian Huskies, Husky crosses, Dalmatians and Bull Terriers (see manual appendix); it may be accompanied by a generalised peripheral neuropathy, when it has been termed laryngeal paralysis peripheral neuropathy complex (LPPC). In Bouviers and Huskies there is evidence of degeneration of neuronal cell bodies within the nucleus ambiguus in the medulla oblongata with subsequent Wallerian-like degeneration of the laryngeal nerves; whereas in Dalmatians and Rottweilers it occurs as part of a generalised 'dying back' axonal degeneration. The onset of clinical signs in these dogs can be as early as 4 to 6 months of age, at which time the condition can be unilateral or bilateral. Electromyographic examination can reveal variable bilateral denervation of the laryngeal abductor muscles usually correlating with laryngoscopic examination. Surgery is necessary to relieve airway obstruction, but will only be a short-term measure in those breeds with concurrent progressive tetraparesis. As the disease is inherited as an autosomal dominant trait in Bouviers, selective breeding should be employed.

Most commonly, laryngeal paralysis occurs as an acquired disease in middle-aged and older large breed dogs, notably Labradors. Although laryngeal paralysis in mature animals is often considered 'idiopathic' there is some evidence that it commonly occurs as one, particularly prominent, component of a generalised polyneuropathy. Since the recurrent laryngeal nerve is especially long, it has been proposed that it will be more susceptible than other peripheral nerves to axonal 'dying back' diseases, in which transport of vital substances to the tip of the axon is compromised. Therefore it is important to consider investigation for systemic disease that can cause polyneuropathies.

Acquired laryngeal paralysis occasionally occurs as a consequence of trauma to the vagus nerve during cervical surgery, lead and organophosphate toxicity, retropharyngeal infection and neoplasia in the vicinity of the recurrent laryngeal nerve; the most commonly incriminated tumour type is thyroid carcinoma. Laryngeal paresis / paralysis can occasionally be seen to be due to brainstem disease; in these cases there are usually other cranial nerves involved and there is often a profound alteration of the dog's consciousness.

Diagnosis

Laryngeal paralysis is usually diagnosed simply by examination of the movement of the vocal folds, either directly through the mouth when the animal is lightly anaesthetised, or by ultrasonography during consciousness. In equivocal cases, EMG examination is helpful, not only in objectively defining denervation of the larynx, but also in detecting more generalised disease both of cranial structures (especially the pharynx) and throughout the entire body. Palpation, ultrasonography and biopsy

readily diagnose thyroid tumours.

Treatment and prognosis

Treatment of laryngeal paralysis requires surgical lateralisation of the arytenoid cartilage regardless of the underlying cause as it is usually a permanent defect. Nevertheless, surgical lateralisation of the arytenoids should not be undertaken lightly in cases in which deglutition is compromised, since it will dramatically increase the risk of food inhalation. Some thyroid tumours can be excised surgically but others are extremely locally invasive, implying that other anti-cancer treatment modalities should be considered.

Megaesophagus

Clinical signs

The characteristic sign of megaesophagus is regurgitation, usually involving both food and fluid; during initial investigation it is of course critical to differentiate regurgitation from true vomiting. Because of regurgitation there may be several other associated clinical signs such as weight loss (or failure to gain weight) and coughing because of inhalation / aspiration pneumonia.

Pathogenesis

There are both congenital and acquired forms of megoesophagus. The underlying cause of congenital megaesophagus is unknown, but recent studies have suggested that afferent function may be aberrant rather than denervation or muscle anomalies. Congenital megoesophagus can also arise as a secondary consequence of a persistent right aortic arch or as one aspect of congenital myasthenia gravis (although not all affected breeds develop megoesophagus). Acquired megoesophagus can result from a wide range of lesions including both brainstem and systemic disease, most notably myasthenia gravis, in which there is immune-mediated destruction of the acetylcholine receptor.

Diagnosis

Megoesophagus is readily diagnosed by radiography, especially if food mixed with barium mixed is given shortly before. Determining the cause for megoesophagus relies on detecting and investigating other signs of systemic or brainstem disease. For instance, routine blood analysis of haematology and blood chemistry can suggest hypoadrenocorticism, myositis or systemic lupus erythematosus and hypothyroidism and myasthenia gravis can be detected by specialist blood tests. If neurological examination detects generalised peripheral nerve diseases, botulism or generalised peripheral neuropathies may be considered, whereas if nearby cranial nerves show deficits then brainstem disease may be suspected and diagnosed using advanced imaging techniques or CSF analysis. In cats, the possibility of dysautonomia should be considered (dogs do not develop megoesophagus with dysautonomia because they have striated muscle throughout the length of the oesophagus). If all of these conditions can be ruled out then idiopathic disease must be assumed.

Treatment and prognosis

For many acquired cases of megoesophagus treatment of the underlying disease is possible – most often by immunosuppressive therapy using corticosteroids or other drugs. In congenital myasthenia gravis, although the disease can be treated using anticholinesterases it can be difficult to achieve the correct dose to restore satisfactory swallowing function. Similarly, unless persistent aortic arches are surgically treated early the megoesophagus will not resolve adequately.

No matter what the cause, postural feeding (*i.e.* feeding the animal with the head directly above the stomach) and feeding a diet that is of a gelatine-like consistency is highly recommended to aid passage of food from mouth to stomach and avoid inhalation.

The prognosis for animals affected with megoesophagus is guarded because of the very high incidence of inhalation pneumonia, which is life-threatening; many animals succumb within a few weeks to months of starting therapy. It has been suggested that since spontaneous remission can occur in myasthenia gravis it may be preferable to avoid immunosuppressive therapy because it may promote respiratory tract infection.

Idiopathic trigeminal nerve palsy

Clinical signs

Motor deficits caused by trigeminal nerve palsy are recognised in the syndrome of 'dropped jaw', in which the patient (almost always a dog) cannot close the mouth or prehend food properly. Other cranial nerves are not affected but there can be a variable degree of sensory loss (sensory trigeminal distribution) and some animals display Horner's syndrome.

Pathogenesis

The cause is thought to be an idiopathic neuritis, although older texts suggested an unsubstantiated link with dogs carrying heavy loads in their mouths. It is important to recognise that the condition must be bilateral to produce the clinical signs and therefore must affect the peripheral nerves and not the brainstem.

Treatment and prognosis

Treatment is simply supportive – helping the animal to eat by offering boluses of soft food and assisting with drinking or the placement of a temporary feeding tube. It has been suggested that holding the mouth shut with a muzzle may aid recovery. Most animals recover rapidly – so that they can eat unassisted within three weeks.

Facial paralysis

Facial nerve dysfunction may be due to disease of the peripheral facial nerve (CN VII) due to otitis media-interna, trauma, hypothyroidism, neoplasia of the middle/inner ear or polyneuropathies. Disease of the facial nerve nucleus in the medulla of the brainstem can be due to any of the diseases affecting the CNS. The most common cause of peripheral facial nerve paralysis has been reported to be idiopathic (75% of dogs and 25% of cats with facial paralysis).

Idiopathic facial paralysis

Idiopathic facial nerve paralysis or palsy may be unilateral or bilateral, but usually occurs in the absence of other neurological deficits (sometimes affected animals also develop idiopathic vestibular syndrome). Exposure keratitis occurs commonly subsequent to the improper lubrication of the cornea. Clinical signs are usually maximal in 7 days and recovery can take 3-6 weeks if at all. Since unilateral facial nerve palsy can also develop in association with CNS diseases it is essential to consider the possibility of CNS lesions as a cause – typically these will be associated with abnormalities of the postural reactions. In early reports on idiopathic facial nerve palsy, biopsies revealed marked depletion of large diameter myelinated axons.

Diagnosis

All possible causes of facial paresis/paralysis should be excluded before this specific diagnosis can be made. A thorough investigation for ear disease should take place in addition to blood tests for hypothyroidism and electromyography to rule out polyneuropathies.

Treatment and prognosis

There is no effective treatment for the underlying disease but it is important to ensure that the cornea is adequately lubricated (either by endogenous production, or eye drops). The prognosis for idiopathic facial palsy varies – many animals make a gradual recovery (weeks to months) but some will be left with permanent deficits, which may progress to produce muscle contracture and deform the facial expression permanently. This can be mistakenly interpreted as an uncommon syndrome in dogs and cats of hemifacial spasm.

vii) Multiple cranial nerve problems

Various combinations of unilateral or bilateral cranial nerve deficits are sometimes detected; almost any combination of signs is possible, depending on the underlying cause (see below). In some instances there will be concomitant depression in limb postural reactions and some animals may exhibit stupor.

Pathogenesis

It is important to recognise the significance of bilateral cranial nerve disorders and postural reaction deficits: bilateral cranial nerve deficits suggest that the lesion is not in the CNS (a lesion in the brainstem large enough to cause bilateral deficits would likely be fatal), and postural reaction deficits in combination with cranial nerve deficits suggest that the lesion *is* in the CNS.

Generalized peripheral neuropathies can cause any combination of cranial nerve deficits; therefore it is of the utmost importance to examine all peripheral nerve reflexes in any animal in which there is a cranial nerve disorder. Occasionally, the most prominent signs are confined to the cranial nerves, for instance there are uncommon idiopathic diseases that can affect multiple cranial nerves – most notably: i) feline dysautonomia, in which pupillary dilatation and megaoesophagus are characteristic signs and ii) polyganglioneuritis, which has been reported as a cause of multiple somatic cranial nerve deficits.

Brain tumors may occasionally cause multiple cranial nerve signs, especially if they develop within the caudal part of the brainstem; in most instances these signs would be accompanied by depression in limb postural reactions. A more confusing presentation is that associated with skull-base tumors (*i.e.* cavernous sinus syndrome) in which there may be multiple cranial nerve deficits, often bilaterally, but without evidence of brainstem involvement. Such animals are often also very depressed because of elevated intracranial pressure.

VESTIBULAR SYSTEM DISORDERS IN CATS AND DOGS

Simon R. Platt BVM&S MRCVS DACVIM (Neurology) DECVN
College of Veterinary Medicine, University of Georgia

The vestibular system is essential in maintaining balance and preventing the animal falling over by keeping and adapting the position of the eyes, head and body with respect to gravity. It is therefore not surprising that disease of the vestibular system results in some of the most dramatic and distressing neurological signs. Head tilt, falling, rolling, leaning, circling, abnormal nystagmus and ataxia commonly result. Clinical signs of vestibular disease may be a result of lesions involving either the receptor organs in the inner ear or the vestibular portion of the eighth cranial nerve (*i.e.*, peripheral vestibular disease) or lesions involving the brainstem vestibular nuclei or vestibular centers in the cerebellum (*i.e.*, central vestibular disease). This session reviews the clinical approach of an animal with a vestibular disorder.

VESTIBULAR DISORDERS: CLINICAL SIGNS

Vestibular disorders (VD) are common in dogs and cats and may result in any or all of the following clinical signs: head tilt, falling, rolling, leaning, circling, abnormal nystagmus, positional strabismus, and ataxia.

- **Head tilt** often indicates a VD. This abnormal head posture is characterized by a rotation of the median plane of the head (one ear is held lower than the other). It occurs in VD as a result of the loss of antigravity muscle tone on one side of the neck. It must be differentiated from a head turn where the median plane of the head remains perpendicular to the ground but the nose is turned to one side. Such head turn is usually associated with a body turn. A head turn does not indicate a vestibular disorder and is usually toward the side of a forebrain lesion.
- **Circling** may occur in conjunction with VD as well as an asymmetrical or focal lesion in the forebrain. Tight circles are usually but not exclusively associated with a VD, while wide circles are often associated with a forebrain lesion.
- **Nystagmus** is an involuntary rhythmic movement of the eyeballs. Physiologic nystagmus is nystagmus that occurs in normal animals while pathologic nystagmus reflects an underlying VD. The direction of the nystagmus is typically defined by the direction of the fast phase. Physiologic nystagmus can be induced in the normal animal by rotating the head from side to side (oculo-vestibular reflex). It is characterized by a slow phase in the opposite direction of the head movement and a fast compensatory phase in the same direction as the head rotation. Physiologic nystagmus

can be depressed in animal with unilateral VD or absent in animal with bilateral VD. Pathologic nystagmus can be either spontaneous (observed when the head is in a normal position at rest) and/or positional (that which occurs, or is altered in character, intensity or direction, with alteration in the position of the head, for example by placing the animal upside down on its back). Nystagmus is usually classified on the basis of its direction and may be horizontal, vertical or rotatory and may change in direction on changing position of the head. The fast phase of pathologic nystagmus is typically directed away from the side of the vestibular lesion.

- **Strabismus** refers to an abnormal position of the globes. Strabismus can be seen in VD when the head is placed in an abnormal position (extended dorsally or the animal placed upside down on its back). VD often causes a ventral or ventrolateral positional strabismus in the eye on the same side as the vestibular 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 peripheral nerve, spinal cord or brainstem disorder (proprioceptive or sensory ataxia). Vestibular lesions often cause ataxia characterized by swaying of the trunk and head, base-wide stance, leaning, falling and rolling to one side with unilateral lesion. With bilateral VD, affected animals tend to fall to both sides and often show wide excursion of the head from side to side.

VESTIBULAR DISORDERS: LOCALIZING THE LESION

Clinical signs of VD may be a result of lesions involving the receptor organs in the inner ear or the vestibular portion of the eighth cranial nerve running in the petrous part of the temporal bone (i.e., peripheral VD) or lesions involving the brainstem vestibular nuclei (i.e., central VD).

- **Peripheral or central?**

Most lesions affect a region, rather than a specific nerve or nucleus, so accompanying neurologic abnormalities can often be used to localize the lesion to the peripheral or central vestibular system (Table 1). Both peripheral and central VD can cause a head tilt, horizontal or rotatory nystagmus, and ataxia. Facial paralysis and Horner's syndrome can be seen with peripheral VD due to the proximity of cranial nerve VII (facial nerve) and the sympathetic nerve supply to the eye to the vestibular nerve in the region of the petrous temporal bone. Correctly identifying central VD requires identification of clinical signs that cannot be attributed to diseases of the peripheral vestibular system. Lesions that affect the central vestibular system typically have additional clinical signs suggestive of brainstem involvement. Such lesions often involve the reticular formation as well as ascending and descending motor and sensory pathways to the ipsilateral limbs. Therefore, abnormal mental status, ipsilateral paresis, and conscious proprioceptive deficits are commonly associated with central VD. Deficits of cranial nerves V through XII can also be associated with central VD. The presence of spontaneous or positional jerk nystagmus indicates vestibular dysfunction but does not further localize the lesion to the peripheral or central vestibular system. However, vertical nystagmus and nystagmus that changes in direction on changing position of the head are a feature of central vestibular lesions. Rate of nystagmus (number of beats per minute with the head in a neutral position as well as with the animal in dorsal recumbency) can further assist with differentiation between central VD from peripheral VD. Median rate of resting and positional nystagmus appears to be significantly faster for dogs with peripheral VD with a resting nystagmus ≥ 66 beats per minute providing the highest combined sensitivity and specificity in diagnosing peripheral VD. With peripheral and central VD, the head is usually tilted in the direction of the lesion. With paradoxical VD, however, the head is usually tilted opposite to the direction of the lesion.

	Peripheral	Central
Head tilt	Ipsilateral	Ipsilateral (or contralateral in case of paradoxical VD)
Asymmetrical ataxia	Ipsilateral	Ipsilateral
Nystagmus <ul style="list-style-type: none"> • Horizontal • Rotatory • Vertical • Positional 	Ipsilateral Ipsilateral No No (except during the early phase of recovery)	Ipsilateral or contralateral Ipsilateral or contralateral Yes Yes
Postural reaction deficit	No	Yes (always ipsilateral to the lesion)
Abnormal mental status	No	Possible
Circling	Ipsilateral	Ipsilateral or contralateral

(Table 1.) Clinical findings associated with peripheral and central vestibular disease

Occasionally, intracranial lesions can result in signs suggestive of a peripheral lesion. Although animals with a peripheral VD have a normal level of consciousness and no evidence of weakness or postural reaction deficits, the absence of these signs does not rule-out the presence of a central VD. If in doubt about the localization of the lesion, the clinician should evaluate the animal for central VD as well as peripheral VD. In very rare cases, VD may be part of a diffuse polyneuropathy or cranial polyneuropathy. Other cranial nerve dysfunction such as dysphagia, tongue weakness, jaw weakness and/or facial paralysis as well as limb weakness with depressed segmental spinal reflexes may be seen. Note also that lesions of the thalamus and/or extrapyramidal basal nuclei may also cause abnormal head posture and signs of central VD.

- **Ipsilateral, contralateral or bilateral lesion?**

With both central and peripheral VD, the head tilt, circling and nystagmus typically occur ipsilateral to the side of the lesion. Less frequently, lesions affecting the caudal cerebellar peduncle, the fastigial nucleus, or the flocculonodular lobes of the cerebellum can cause central VD with a resulting paradoxical head tilt. This syndrome is called paradoxical because the head tilt and circling occur contralateral to the side of the lesion. Bilateral VD is characterized by head sway from side to side, loss of balance on both sides and symmetrical ataxia with a wide-based stance. A physiological nystagmus usually cannot be elicited and a head tilt is not observed.

DIFFERENTIAL DIAGNOSIS OF VESTIBULAR DISORDERS

A lesion must be localized to a particular section of the vestibular apparatus before an appropriate differential diagnosis can be established and further test conducted. The formation of a differential diagnosis list is essential in choosing and interpreting any diagnostic test. Diseases affecting the nervous system are classically classified in disease processes using the mnemonic VITAMIN D.

Disease mechanism	Peripheral vestibular disease	Central vestibular disease
Vascular		Brain infarct Brain hemorrhage
Inflammatory/ Infectious	Otitis media/interna Nasopharyngeal polyps	Infectious encephalitis (Distemper, Toxoplasma, Neospora, Fungus, Bacterial, FIP) Meningo-encephalitis of unknown etiology (GME, necrotizing, idiopathic)
Trauma	Head trauma	Head trauma
Toxic	Aminoglycosides, topical chlorhexidine	Metronidazole
Anomalous	Congenital vestibular disease	Intracranial intra-arachnoid cyst, dermoid/epidermoid cyst, Dandy- Walker syndrome, Chiari-like malformation
Metabolic	Hypothyroidism	
Idiopathic	Idiopathic vestibular disease	
Neoplastic	Middle and/or inner ear tumour	Primary or metastatic brain tumor
Nutritional		Thiamine deficiency
Degenerative		Neurodegenerative disease

Idiopathic vestibular syndrome is common in adult cats and dogs (often geriatric). Clinical signs are usually peracute and initially severe with affected animal appearing extremely disable in the first 48 to 72 hours. If facial nerve paralysis or Horner's syndrome (miosis, enophthalmia, protrusion of third eyelid, ptosis of upper eyelid) is also present then other differentials should be considered. Diagnosis is based on the presence of compatible history and exclusion of other causes of peripheral VD. Most animals tend to improve over 1 to 3 weeks period and often return to normal. However, some animal may be left with a permanent head tilt or episodic ataxia. No treatment has proved beneficial and recurrence is possible.

Otitis media/interna can be secondary to otitis externa, oropharyngeal infection (spreading via the auditory tube) or hematogenous spreading. Therefore, the absence of sign of otitis externa does not rule-out the presence of otitis media/interna. Clinical course can be acute or progressive. Signs of VD caused by otitis media/interna are often associated with ipsilateral facial nerve paralysis and/or Horner's syndrome. Diagnosis is made by otoscopic examination and imaging studies (bulla radiographs, CT or MR scan) and/or exclusion of other causes of peripheral VD. If fluid is visualized within the middle ear then attempt should be made to obtain a sample via myringotomy for cytology and bacterial culture. Treatment of otitis media/interna consists of systemic antibiotic for a minimum of four to six weeks (oral amoxicillin/clavulanate, fluoroquinolone or cephalosporin if no culture can be obtained following myringotomy). Surgical drainage and debridement via bulla osteotomy should be considered in case of failure of medical treatment. Prognosis is guarded to fair as some animal may be left with permanent head tilt and/or facial paralysis.

Tumors of the caudal fossa and meningo-encephalitis of unknown etiology (MUE) such as granulomatous meningo-encephalitis (GME) are the two most common causes of central VD.

Common types of tumors found in the caudal fossa include meningioma and choroid plexus tumor both which have a tendency to arise at the level of the emergence of the vestibulo-cochlear nerve at the cerebellomedullary angle. Less common tumors include glioma, ependymoma or medulloblastoma. Signs of VD associated with these tumors are often slowly progressive. Presumptive diagnosis is made by advance imaging (CT or MRI) but the exact type of tumor can only be confirmed histologically (either

by surgical tissue biopsy or post-mortem). Prognosis is fair for surgically accessible cerebellomedullary angle meningioma. Prognosis is more guarded for other tumors.

Meningo-encephalitis of unknown etiology (MUE) is often attributed to GME, the diagnosis of which can only be confirmed on histopathology. Clinical signs can be acute or progressive in onset. Neurolocalisation often suggests multifocal involvement but can occasionally be focal. A presumptive diagnosis can be made based on a consistent history, clinical signs, signalment (frequently young to middle-aged female terrier breeds), multifocal, contrast-enhancing lesions on MRI, CSF analysis (pure mononuclear pleocytosis or a mixed cell population) and exclusion of infectious aetiologies on serological or PCR tests (mostly Distemper, Toxoplasma and Neospora). Immunosuppressive doses of corticosteroids have been the mainstay of treatment for presumptive GME. Other immunomodulatory drugs such as azathioprine, procarbazine, cytosine arabinoside and cyclosporine as sole agent or as an adjunctive treatment with prednisone have been reported to be effective in some dogs. Overall, the prognosis is guarded but survival times range from weeks to years.

NEURODIAGNOSTIC INVESTIGATIONS OF VESTIBULAR DISORDER

The choice of neurodiagnostic tests in patient with VD depend essentially on where the lesion is suspected on the basis of the neurological examination. If in doubt about the localization of the lesion, the animal should be evaluated for both peripheral and central VD.

- **Peripheral Vestibular Disease**

Diagnostic plan for patients with signs suggestive of peripheral VD include at least otoscopic and pharyngeal examination, imaging of the tympanic bullae with radiographs, computed tomography (CT) or magnetic resonance imaging (MRI) and thyroid function testing. Middle ear pathology should be suspected if the tympanic membrane is ruptured, bulging, cloudy or red in color on otoscopy. If the tympanic membrane is ruptured, swabs for cytology and culture (aerobic, fungal and yeast) can be taken directly from the middle ear. If the tympanic membrane is intact but bulging or of an abnormal colour, a small hole (myringotomy) can be made in the tympanic membrane with a 20-gauge spinal needle to obtain samples for cytology and culture. Additionally, the middle ear cavity can be flushed by attaching a 10 to 20 cc syringe of warm saline to the spinal needle. Warm water is flushed into the tympanic cavity and gently suctioned. The resulting fluid can then be submitted for cytology and culture. Radiographic evaluation of the tympanic bullae requires general anesthesia to allow adequate positioning. Four radiographic projections are classically used (dorsoventral, latero-lateral, latero 20° ventral-laterodorsal and rostro 30° ventral-caudodorsal open-mouth projection). Although positive radiographs can be seen as highly specific in the diagnosis of middle ear disease, negative radiographs do not rule out the presence of middle ear disease. CT and MRI are more sensitive than radiographs in detecting middle ear pathology. Brainstem auditory evoked response test may be abnormal if the cochlea, vestibulocochlear nerve or auditory brainstem pathways are involved and can sometime be used to differentiate central from peripheral VD. Finally, electromyography (EMG) and motor nerve conduction study are indicated in patients suspected of multiple cranial nerve neuropathy or of a more diffuse polyneuropathy.

- **Central Vestibular Disease**

Evaluation of patients suspected of central VD include in first instance the use of advance imaging (CT or MRI), cerebrospinal fluid (CSF) analysis (nucleated cell count and cytology, total protein concentration), serum and CSF titers (serology and/or PCR) for various infectious organisms (toxoplasma gondii, neospora caninum, canine distemper, coronavirus, fungal agents...). Further investigations may be require in cases suspected of brain tumour (tissue biopsy by surgical or stereotactic biopsy, thoracic and abdominal imaging to investigate metastatic disease), thiamine deficiency (urinary organic acids excretion screening or transketolase activity in fresh erythrocytes) or cerebrovascular accident (routine hematology and serum biochemistry, clotting profile, evaluation of arterial blood pressure, thyroid, kidney, adrenal and heart function).