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Neurology Survival Kit 2017 Mini Series

Session Two: Ataxia, tight jaw, dropped jaw and droopy faces

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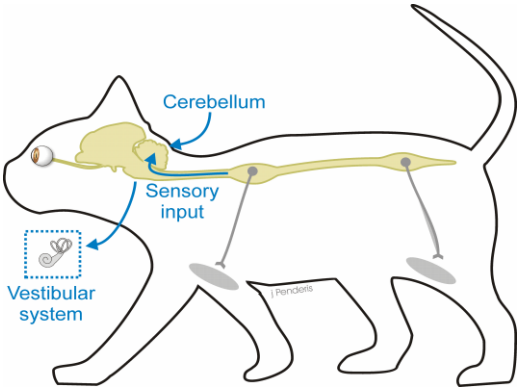


ATAXIA

Ataxia is defined as an uncoordinated gait and can arise from a lesion affecting three distinct anatomic sites in the nervous system: (1) a sensory peripheral nerve or a spinal cord lesion (general proprioceptive ataxia), (2) a vestibular lesion (vestibular ataxia), or (3) a cerebellar lesion (cerebellar ataxia) (Figure 1). Ataxia can be further divided into hypometria (shorter protraction phase of gait) or hypermetria (longer protraction phase of gait).

Understanding the origin of the ataxia

General proprioceptive (GP) ataxia reflects the lack of information reaching the central nervous system (CNS) responsible for the awareness of the movement and position of the neck, trunk and limbs in space. As a consequence, there may be a delay in the onset of protraction of the limb which may cause a longer stride than normal. The animal may walk on the dorsal part of its foot or may drag its digits. These signs often overlap with those caused by upper motor neuron (UMN) paresis. General proprioceptive (GP) ataxia results from lesions affecting the ipsilateral GP pathways in the spinal cord (fasciculus gracilis and cuneatus in the dorsal funiculus for general proprioception in the pelvic limbs and thoracic limbs respectively), ipsilateral caudal medulla oblongata, contralateral medial lemniscus in the pons, mesencephalon and thalamus, and contralateral cerebral cortex (mostly parietal lobe). Although lesions within the thalamus and cerebral cortex will cause general proprioceptive ataxia in humans, in domestic species the ataxia generated from lesions within these structures is usually too subtle to be detected on gait evaluation. The pathways of the GP sensory system are anatomically adjacent to most of the upper motor neuron (UMN) pathways necessary for gait generation. The change in the gait therefore generally reflects a combined dysfunction of both UMN paresis and GP ataxia with delayed onset of protraction of the limb and lengthened stride. From a lesion localisation point of view, UMN paresis and GP ataxia visible in the gait can occur as a consequence of lesion affecting the brainstem, or spinal cord. Compared to UMN paresis, disorders of the lower motor neurons (LMN) only cause paresis and not ataxia.

Vestibular ataxia occurs with lesions affecting either the peripheral or central vestibular apparatus. In addition to ataxia, animals will often have concurrent neurological signs that reflect a vestibular disorder, such as head tilt (unilateral vestibular disorder) or head sway (bilateral vestibular disorder), pathological nystagmus, or positional strabismus. Animals with vestibular ataxia often have a broad-based gait (especially in the pelvic limbs) with leaning towards the side of decreased vestibular tone. Some animals may have substantial swaying when walking and will occasionally fall; recumbent animals may be seen to roll. Weakness or paresis is only seen with central vestibular disease and is not a feature of peripheral vestibular disease.

Cerebellar ataxia can be seen in animals that have lesions within the cerebellar cortex. Other signs of cerebellar disease, such as intention tremors, are often present. Cerebellar ataxia is characterized by

hypermetria and dysmetria. Hypermetria associated with cerebellar ataxia consists of over-flexion during limb protraction and is therefore distinct from the over-reaching, long-strided gait noted in animals with combined general proprioceptive ataxia-upper motor neuron paresis. Dysmetria is a component of cerebellar ataxia and is manifested by a loss of synchronous limb movements.

Vestibular or cerebellar ataxias are accompanied by other signs of dysfunction of the vestibular apparatus or cerebellum respectively.

Neurological assessment of the ataxic cat and dog

It is essential to try and characterize which subclassification(s) of ataxia is contributing to the gait pattern. The presence of ataxia should suggest a lesion of the spinal cord, brainstem, cerebellum, or peripheral vestibular apparatus as discussed above; multi-focal disease with involvement of at least two of these regions should also be a consideration. Associated neurological signs are used to localise the lesion to one of these parts of the nervous system (see flowchart). Correct anatomic diagnosis is crucial in establishing a differential list as some causes of ataxia are specific to certain regions of the nervous system. Additionally, the choice of ancillary diagnostic tests is guided by lesion localization and the differential list.

The initial part of the examination is devoted to observing the animal's posture and gait. For a cat that is reluctant but able to move about in the room, enticement with a toy or laser pointer can be particularly effective. This hand-off approach should focus in detecting the following neurological clues as to neuroanatomic diagnosis:

- Head tilt: often indicate an unilateral vestibular disorder (peripheral or central). The head is usually tilted toward the same side as the lesion. Lesions affecting the cerebellar portion of the vestibular apparatus (flocculonodular lobe or cerebellar peduncle) can cause a central vestibular syndrome with a paradoxical head tilt.
- Leaning or falling to one side: indicate an unilateral vestibular disorder (peripheral or central)
- Wide excursion of the head from side to side: indicate a bilateral vestibular disorder (peripheral or central)
- Intention tremor of the head: indicate a cerebellar disorder
- Symmetrical hypermetria on all four limbs or on one side and in the absence of paresis: indicate a cerebellar disorder (or at least lesion affecting the spino-cerebellar pathways in the spinal cord in the absence of other signs of cerebellar disorder)
- Concurrent UMN paresis in limbs with no effect on the eyes or head posture: indicate brainstem or spinal cord disorder

At a minimum, the hands-on part of the examination should focus on evaluating the animal's postural reactions, segmental spinal reflexes and selected cranial nerve functions such as the menace response, pupil size and symmetry, and detecting presence of pathological nystagmus.

Postural reaction testing aim to detect subtle deficits that were not obvious on gait evaluation. These reactions reveal the animal's awareness of the precise position and movements of parts of its body, as well as the animal's ability to generate movements in the part tested. The best reactions to test in cats are the hopping response, wheelbarrowing and tactile placing as paw position testing (or "knuckling" response) can be very difficult to assess in this species. Postural reactions can be abnormal in the presence of central vestibular lesion, brainstem or spinal cord lesion. They are usually normal with peripheral vestibular lesion or cerebellar lesion (although delayed then exaggerated response may be seen with the latter lesion localisation). With gait evaluation, postural reaction testing helps to narrow down the lesion localisation as being cranial to T3 spinal cord segments (all four limbs affected or both thoracic and pelvic limb affected on the same side) or caudal to T3 spinal cord segments (both pelvic limb or only one pelvic affected). Segmental spinal reflex evaluation helps to narrow down further the lesion localisation by testing the integrity of the C6 – T2 and L4 – S3 intumescences as well as respective segmental sensory and motor nerve, that form the peripheral nerve, and the muscles innervated. Lesions at the level of these intumescences result in LMN signs in the muscles innervated (i.e. loss of segmental spinal reflexes as well as reduced muscle tone and size). Segmental spinal reflexes are best performed with the cat in dorsal recumbence between the thighs of the examiner. Withdrawal reflex and the patellar reflex are the most reliable one in cats. Other spinal reflexes (triceps, biceps, extensor carpal radialis and gastrocnemius) are more difficult to perform and to interpret.

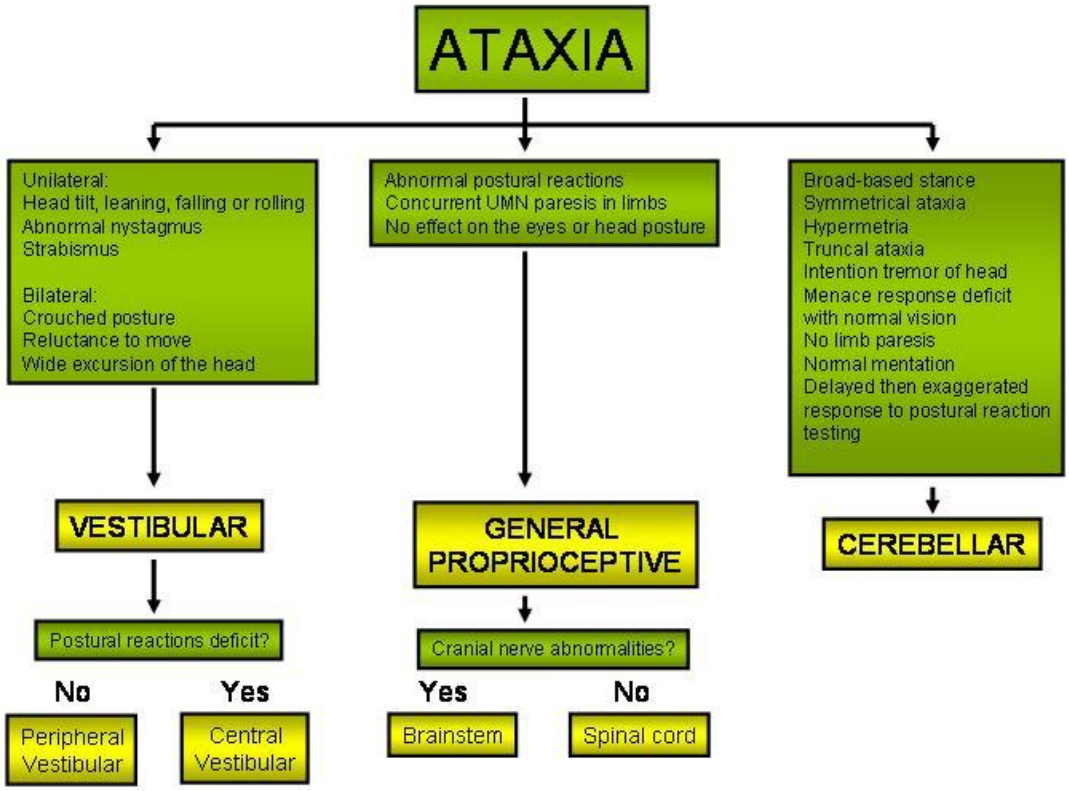
The menace response is elicited by making a threatening gesture at the eye tested while the other eye is blindfolded. The expected response is a closure of the eyelid. It is absent in very young cats and dogs (<10-12 weeks). This response tests the retina, optic nerve (cranial nerve II = CN II), contralateral optic tract and contralateral forebrain, ipsilateral cerebellum and facial nerve (CN VII). The visual placing response requires intact visual and motor pathways and can be useful in assessing visual function in a cat or small dog where the menace response is ambiguous. It is tested by carrying the animal towards a tabletop. On approaching the surface the animal will reach out to support itself on the table before the paw touches the table. In the context of an ataxic animal, the menace response may be abnormal with cerebellar lesion (absent on the same side as the lesion with intact visual placing) or with multifocal CNS disease process (inflammatory, infectious or metastatic disease). Evaluation of pupillary size and equality in ambient light as well as in darkness is also an important part of the evaluation of an ataxic animal. Normal pupils should be symmetrically shaped and equal to each other in size. Horner's syndrome (manifesting as miosis, enophthalmia and protrusion of third eyelid) can be associated with peripheral vestibular lesion, especially in animals with otitis media/interna, nasopharyngeal polyps and middle ear tumour. The presence of

spontaneous or positional nystagmus: indicate vestibular disorder. Vertical or nystagmus which changes direction with different positions of the head indicate a central vestibular disorder.

Localising the neuroanatomic lesion in the ataxic cat and dog

The above neurological assessment should help to test the integrity of the various components of the nervous system which may be involved in an ataxic animal (i.e. general proprioceptive, vestibular and cerebellar system) and detect any functional deficit(s) present. Normal findings are as important as abnormal ones in localising a lesion. Neurological abnormalities detected on examination should be added to the list of abnormal findings collected from the history.

Attempts should be made to explain all the abnormal findings by a single lesion within one of the following specific regions of the nervous system: peripheral or central vestibular system, brainstem, spinal cord or cerebellum. Lesions within these regions of the nervous system result in predictable and specific neurological signs (flowchart ataxia). Note that in localising a lesion, it is not necessary that all the clinical signs referable to one location or syndrome are present. If a single lesion cannot explain all the abnormal findings identified, the lesion localisation is considered as being multifocal or diffuse.



How to establish a differential diagnostic list?

Differential diagnosis should be established based on the neuroanatomic origin of ataxia. The differential diagnosis list can be developed by taking into account the patient signalment, historical data (mode of onset and pattern of development of the condition) and neurological findings (neuro-anatomic diagnosis as peripheral or central vestibular, cerebellar or general proprioceptive ataxia). Disease processes that can affect the nervous system are traditionally classified according to the 'VITAMIN D' mnemonic. Each category has a typical signalment, onset and progression which helps to narrow down the differentials.

With a clear knowledge of the region of the nervous system involved, and a differential list reduced to no more than three or four disease processes, consideration should be given only to those diagnostic tests that will help to narrow down the list further. These tests should ideally be run in succession from least invasive through to more invasive.

Disease mechanism	Peripheral vestibular disease	Central vestibular disease
Vascular		Brain infarct Brain hemorrhage
Inflammatory/ Infectious	Otitis media/interna Nasopharyngeal polyps	Infectious encephalitis (Toxoplasma, Neospora, Viral, Bacterial, FIP) Meningo-encephalitis of unknown etiology (presumed immune-mediated)
Trauma	Head trauma	Head trauma
Toxic	Aminoglycosides, topical chlorhexidine	Metronidazole
Anomalous	Congenital vestibular disease	Intracranial intra-arachnoid cyst, dermoid/epidermoid cyst
Metabolic		
Idiopathic	Idiopathic vestibular disease	
Neoplastic	Middle and/or inner ear tumour	Primary or metastatic brain tumour
Nutritional		Thiamine deficiency
Degenerative		Neurodegenerative disease

Causes of vestibular disorder

Vascular	Cerebellar cerebrovascular accident
Infectious	Feline Infectious Peritonitis (FIP), Feline spongiform encephalopathy, Fungal diseases, Parasitic encephalomyelitis, Toxoplasmosis, Neosporosis, Viral
Toxic	Metronidazole
Trauma	Trauma
Neoplastic	Brain tumours
Degenerative	Lysosomal storage diseases, Cerebellar abiotrophy
Developmental	Cerebellar Hypoplasia (Feline Panleukopaenia Virus), Intracranial intra-arachnoid cysts

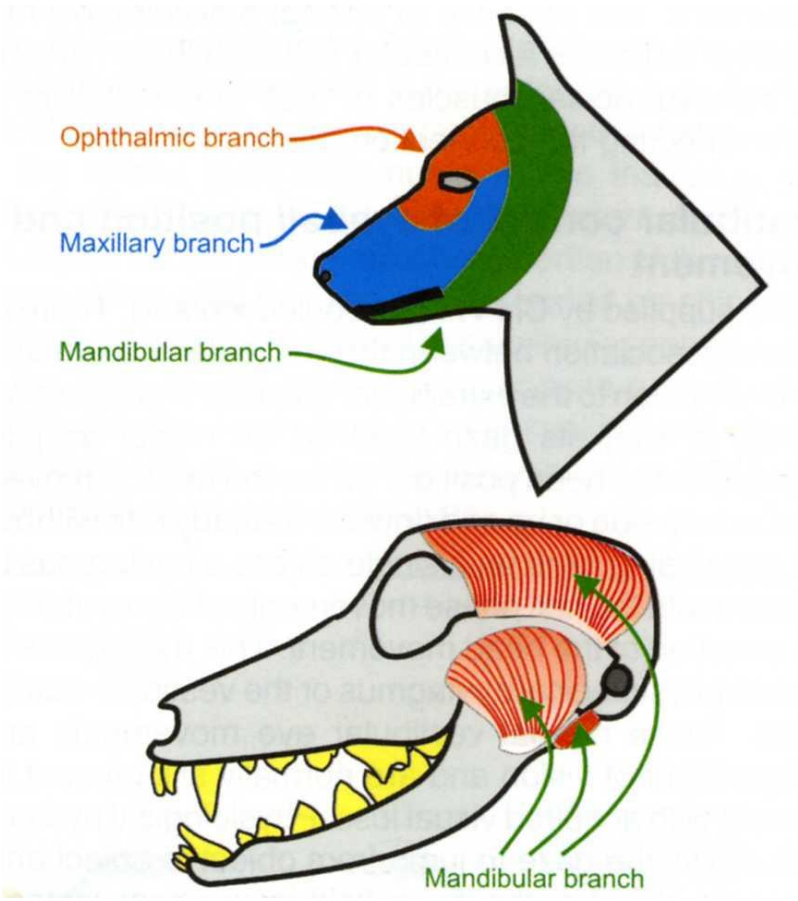
Causes of cerebellar disorder

Vascular	Haemorrhage, Fibrocartilaginous embolism
Infectious	Feline Infectious Peritonitis (FIP), Fungal diseases, Parasitic encephalomyelitis, Toxoplasmosis, Neosporosis, Viral, Epidural abscess
Inflammatory	Presumed immune-mediated myelitis/meningo-myelitis
Trauma	Trauma
Neoplastic	Spinal cord tumours, Vertebral tumours
Nutritional	None
Degenerative	Intervertebral disc disease
Developmental	Spinal intra-arachnoid cysts, Congenital spinal anomalies, Chiari malformation and syringomyelia

Causes of sensory (spinal) ataxia

TRISMUS, DROPPED JAW AND DROOPY FACE

The trigeminal nerve (CN V) provides motor innervation of the masticatory muscles (temporalis, masseter, medial and lateral pterygoid and rostral part of the digastric muscles) and sensory innervation of the face (including cornea, mucosa of the nasal septum and mucosa of the oral cavity). It consists of three branches: ophthalmic, maxillary and mandibular. The mandibular branch serves both a motor and sensory function while the other two have only sensory function. The motor function of CN V is assessed by evaluating the size and symmetry of the masticatory muscles and testing the resistance of the jaw to opening the mouth.



TRISMUS

Trismus is defined as difficulty opening the jaw. Neurological conditions responsible for trismus include masticatory muscle myositis, muscular dystrophy, polymyositis, extraocular myositis (referred jaw pain) and tetanus. Non-neurological causes comprise of craniomandibular osteopathy, retrobulbar abscess and temporomandibular joint disease including luxation/subluxation. Complete physical and neurological examination is important to try distinguishing there different conditions. Patients should be closely examined for evidence of trauma that could have resulted in temporomandibular joint luxation/subluxation. Thorough oral and ophthalmic examination should be performed. Retrobulbar

masses often cause a visible swelling or drainage behind the carnassial teeth. Animal with trismus caused by tetanus often show a characteristic facial expression ('risus sardonicus') resulting from an increase in facial muscle tone.

Masticatory muscle myositis

Masticatory muscle myositis (MMM) is an auto-immune, focal inflammatory myopathy with clinical signs restricted to the muscles of mastication (masseter, temporalis, pterygoid and rostral digastricus) which are innervated by the mandibular branch of the trigeminal nerve. Masticatory muscles contain a unique muscle fiber type (type 2M) that differs both histochemically and biochemically from fiber type present in limb muscles (types 1A and 2A). Biopsies of dogs with MMM are characterised by intense multifocal lymphocytic and plasmacytic perivascular infiltration, occasional eosinophils, necrosis and phagocytosis of type 2M myofibers. Circulating auto-antibodies against masticatory muscle type 2M fibers (fiber type-specific auto-antibodies) can be detected in more than 80% of dogs with MMM and are the basis of serology testing for this condition. Despite many hypotheses proposed to explain the formation of auto-antibodies directed specifically against type 2M fibers including molecular mimicry, the primary initiating factor to this auto-immune disorder is unknown.

The most common clinical signs associated with MMM are inability to open the jaw, jaw pain, and masticatory muscle atrophy. Some dogs may present with pyrexia, mandibular lymphadenopathy, trismus, swollen and painful masticatory muscles, and bilateral exophthalmos from swelling of the pterygoid muscles during the acute phase of the condition. Many owners however do not recognise a problem until the chronic phase when marked muscle atrophy and enophthalmos because of atrophied pterygoid muscles are present.

MMM can be seen in any breed of dog with no apparent gender predilection. The average age of onset is 3 years, although dogs as young as 4 months of age with MMM have been reported. Diagnosis can be confirmed by detection of significant levels of anti-type 2M muscle fiber antibodies in the serum of suspected dogs. False negative results may occur if corticosteroids have been administered before sampling. Serum creatinine kinase (CK) levels are modestly elevated in some dogs in the acute phase of MMM. EMG can help to confirm the selective involvement of masticatory muscles and differentiate MMM from polymyositis. However, EMG may be normal in dogs with end-stage disease because of severe fibrosis and myofiber depletion. Evaluation of muscle biopsy taken from the masticatory muscles can also provide diagnostic confirmation of the disease as well as prognostic information by determining the stage of the disease.

Immunosuppressive doses of corticosteroids (prednisolone 1 to 2 mg/kg every 12 hours orally) comprises the cornerstone of treatment of MMM. This dose should be maintained until jaw function and serum CK level (when initially elevated) have both returned to normal. Dosage of prednisolone is then slowly decreased over a few months to the lowest every-other day dose that keeps the clinical signs at bay. Other immuno-suppressive agents such as azathioprine (1 to 2 mg/kg every 24 hours

orally) are indicated in dogs that failed to respond to corticosteroids treatment or that relapse when the dose is tapered. Short-term prognosis is usually good however many dogs that are treated for insufficient period of time will experience relapses. Life-long treatment is occasionally necessary. The prognosis of dogs in the more chronic phase of the disease (gradual replacement of myofibers by fibrous tissue) is guarded. Persistent muscle atrophy is a common manifestation of the disease.

DROPPED JAW

An inability to close the mouth (also described as 'dropped jaw') is a commonly encountered presentation in dogs. Affected animals frequently present as well with difficulty eating and drinking, and hypersalivation. The most common cause for this presenting sign is idiopathic trigeminal neuropathy (also called trigeminal neuritis, trigeminal neurapraxia or trigeminal nerve palsy). Other neurological causes for bilateral paralysis of the mandibular branch of the trigeminal nerve include multicentric lymphosarcoma (neoplastic lymphoid cell infiltration), myelomonocytic leukaemia, idiopathic hypertrophic chronic pachymeningitis, disseminated, non-suppurative ganglioradiculoneuritis and rabies. Non-neurological causes are usually responsible for mechanical obstruction and include bilateral luxation of the temporomandibular joints, fracture of the mandible or oral foreign body.

Idiopathic trigeminal neuropathy

Idiopathic trigeminal neuropathy is the most common cause of 'dropped jaw'. Onset is usually acute. Horner's syndrome, some degree of sensory loss in the sensory distribution of the trigeminal nerve and facial nerve paralysis can occasionally be associated with the 'drop jaw'. It is a diagnosis of exclusion and cannot be confirmed by any antemortem test. The only tests that showed abnormal results in most dogs are electromyography (EMG) and CSF. EMG often reveals positive sharp wave and/or fibrillation potentials in the masticatory muscles unless the dog is tested to soon after the onset of mandibular paralysis (up to 7 days after the onset). CSF analysis can reveal a mild mononuclear pleocytosis, often with normal or mildy elevated protein content or can be normal. The etiology remains unknown. A non-suppurative inflammatory neuritis in motor branches of the trigeminal nerve and ganglion has been confirmed in some cases, however it is unknown whether this inflammatory process occur in all cases. Some report postulated a link with dogs carrying heavy object which was hypothesize to stretch the mandibular nerve from hyperextension of the jaw. Treatment is mainly supportive, helping the animal to eat and drink. Corticosteroid administration appears not to affect the clinical course of the disease. Use of tape muzzles has been recommended to improve ingestion of food as these dogs are unable to grab food but can swallow normally. Mean time for recovery ranges from 2 to 10 weeks. Dogs with longer recovery frequently show marked atrophy of the masticatory muscle caused by prolonged denervation.

Chronic polyradiculoneuritis

Although rare, unilateral atrophy or bilateral paralysis of muscles of mastication can be caused by chronic polyradiculoneuritis in which neural involvement is largely focused in the trigeminal nerves. Syndromes that may be classified as chronic polyradiculoneuritis include chronic relapsing polyradiculoneuritis, hypertrophic neuropathy, polyradiculoneuritis, and chronic polyneuritis. The etiology and pathogenesis of these syndromes are largely undertermined. Very little is known about the treatment and prognosis of these conditions.

Idiopathic hypertrophic chronic pachymeningitis

Idiopathic hypertrophic chronic pachymeningitis is a recently recognised cause of multiple cranial nerve deficits. 'Dropped jaw' is the most common complain for this condition, however, most dogs also show associated other cranial nerve deficits. No long tract signs (limb weakness, postural reaction deficit, ataxia) are usually observed as this condition mainly affect the meninges without brain parenchymal involvement. Lurcher and Greyhound seem to be predisposed. This condition is characterised by diffuse thickening of the dura mater caused by fibrosing inflammatory process that involves the dura mater. The etiology is unknown. The CSF in most cases showed inflammatory changes but can be normal. Neuroimaging studies revealed diffuse or localized thickening of the dura. MR imaging is key to diagnosis of this disorder with increased meningeal thickening and diffuse or localized hyperintensity in T2W and FLAIR images and severe contrast enhancement. Treatment consists in using immunosuppressive doses of corticosteroids (prednisolone 1 to 2 mg/kg every 12 hours orally until remission then at decreasing dosage). The addition of other immunosuppressant such as cytosine arabinoside seems to help getting a better control of the disease. Although most dogs get into clinical remission, cure is more difficult to obtain.

Neoplastic cell infiltration of the trigeminal nerve

Dogs with 'dropped jaw' that present with multiple cranial nerve or other neurological deficits are more at risk of having malignant disease and further diagnostic test to rule-out neoplastic or infectious disease should be considered. Both multicentric lymphosarcoma and myelomonocytic leukaemia have been identified as possible causes of 'dropped jaw' in dogs primarily via neoplastic infiltration of the trigeminal nerve. Lymphosarcoma in dogs can take a number of different forms and can involve the CNS, the peripheral nervous system (PNS), or both. Dogs with CNS involvement frequently present immature lymphoid pleocytosis on CSF analysis while dogs with PNS involvement may have normal CSF. Treatment options are limited and include the use of corticosteroids, lomustine (CCNU), carmustine (BCNU), cytosine arabinoside and radiation therapy. Regardless of treatment modality used, the long-term survival for dogs with multicentric lymphoma is guarded to poor with most patient showing only a partial or brief remission of clinical signs.

DROOPY FACE (FACIAL PARALYSIS)

The ear serves as the organ of hearing and balance in vertebrates. It is divided into three distinct anatomic and functional components: the outer, middle and inner ear. The external ear serves as a sound-gathering structure by receiving air vibrations, then concentrating and transmitting them to the tympanic membrane. Most of the middle ear is composed of the air-filled tympanic cavity. Sound vibrations in the ear canal are transmitted to the tympanic membrane, and in turn are transmitted through the ossicular chain to the membrane of the oval window, which divides the middle from the inner ear. The inner ear structures are made up of the bony and membranous labyrinths housed within the temporal bone. Their main function is to receive the stimuli that result in hearing and equilibrium via the semicircular canals and the cochlea. Diseases affecting the ear can be broadly divided into inflammatory and non-inflammatory conditions. Otitis and neoplasia represent the main differential diagnoses. Because the facial and sympathetic nerves course near the middle ear, a facial nerve paralysis and/or Horner's syndrome may occur on the same side as the middle ear disease. The clinical signs of inner ear disease in the dog and cat are usually those of a peripheral vestibular syndrome, which reflects injury to the vestibular nerve or receptor organs. Although deafness may also be caused by inner ear disease, it may not be noticeable clinically with unilateral involvement.

The facial nerve (CN VII) is motor to the muscle of facial expression and sensory (providing the sense of taste) to the rostral two thirds of the tongue and palate. Its parasympathetic component innervates the lacrymal gland, the mandibular and sublingual salivary glands. Neurons innervating the muscles of facial expression are located in the facial nucleus in the rostral medulla oblongata. The axons pass in the internal acoustic meatus of the petrosal bone on the dorsal surface of the vestibulocochlear nerve and leave the skull through the stylomastoid foramen. The facial nerve courses through the middle ear before branches are distributed to the muscles of facial expression (ear, eyelids, nose, cheeks, lips) as well as caudal portion of the digastricus muscle.

The motor function of CN VII is primarily assessed by observation of the face for symmetry (position of the ears and lip commissure on each side within the same plane, symmetry of the palpebral fissure), spontaneous blinking and movement of the nostrils. It also is the motor response (efferent part) of following tests: palpebral reflex, corneal reflex, menace response, pinching of the face. The parasympathetic supply of the lacrymal gland associated with CN VII can be evaluated by the Schirmer tear test strips. This later test quantitatively assesses the tear flow by measuring the amount of wetting on a filter paper inserted in the lower conjunctival fornix at the outer half of the palpebral fissure. In normal dog and cats, the wetting of the tear test paper ranges from 10 to 25 mm in one minute.

Salivation can be subjectively assessed by examining the mouth for a moist mucosa.

Motor involvement of CN VII produces the following signs: drooping and inability to move the ear and lip, drooling, widened palpebral fissure, absent spontaneous and provoked blinking, absent abduction of the nostril during inspiration, deviation of the nose toward to the normal side due to the unopposed muscle tone on the unaffected side. With chronic denervation, the lips are retracted further than normal and the nostril is deviated to the affected side as a result of muscle fibrosis . Unilateral involvement can be seen in the asymmetry of the ears, eyelids, lips and nose. Lesion of the individual branches of the facial nerve long their course produces paresis or paralysis to the specific muscle they innervate. Involvement of the parasympathetic supply of the lacrymal gland produces keratoconjonctivitis sicca.

Differential diagnosis of facial nerve paralysis using the mnemonic mean VITAMIN D should include :

- **I**nfectious and inflammatory disorder: middle ear infection, viral/bacterial/protozoal and immune-mediated meningoencephalitis and inflammatory/immune-mediated neuromuscular disorders such as polyradiculoneuritis, myasthenia gravis
- **T**raumatic disorder: iatrogenic injury to the peripheral facial nerve, head trauma
- **M**etabolic: hypothyroid polyneuropathy
- **I**diopathic: acute unilateral or bilateral paralysis may be seen
- **N**eoplasm: CNS neoplasm located to the caudal fossa, middle ear neoplasm

Compared to with middle/inner ear disease where facial nerve paralysis may be associated with Horner ‘s syndrome and peripheral vestibular disorder, diseases of the facial nerve nucleus in the medulla oblongata are usually associated with brainstem signs such as ipsilateral paresis and postural reaction deficit, decreased mentation, central vestibular signs and other cranial nerve deficits. With generalised neuromuscular disorders, any combination of cranial nerve deficit as well as limb signs of neuromuscular weakness (intermittent or permanent weakness, decreased segmental spinal reflexes and muscle tone associated or not with muscle atrophy).