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Veterinary Neurology made easy!

Session 1: Neurological examination: how to perform and interpret it

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NEUROLOGIC EXAMINATION AND LESION LOCALIZATION

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The **learning outcomes** of the first module of the CPD solution Neurology mini-series are become better able at:

1. Performing and interpreting the neurologic examination in dogs and cats
2. Achieving a neuroanatomic diagnosis

The neurological examination is the basic and most important tool of clinical neurology.

The neurological examination allows:

- to determine if the nervous system is affected by a disease process
- to localize the lesion within the nervous system (neuroanatomic diagnosis)
- to assess the severity of dysfunction
- to construct a list of differential etiological diagnoses (along with the information provided by the signalment, the history and the general physical examination)

These information are necessary to establish which diagnostic procedures are indicated (haematology, serum biocchemistry profile, serology for infectious disease, urinalysis, CSF analysis, survey radiography, MRI, electrodiagnostics, muscle/nerve biopsy, etc).

Performing the neuroanatomic diagnosis should always precede consideration of the differential etiological diagnoses and therefore of the diagnostic investigation plan.

Taking a thorough medical history and performing a detailed general physical examination are prerequisites to the neurological examination. This should be performed in a systematic manner and each result has to be recorded in a standard form so that no part of the examination is omitted. In certain cases, it might be necessary to modify the examination according to the chief complaint, the condition and cooperative attitude of the patient. For instance, in an animal presented with acute spinal trauma any manipulation that might exacerbate a possible vertebral instability (such as performing hopping and wheel-barrowing) should be avoided until an unstable spinal injury has been ruled out. In addition, in order to perform and interpret correctly each test, it is of paramount importance to achieve and maintain patient cooperation throughout the examination. Therefore any disagreeable or painful procedures, such as palpating a painful area, should be performed only at the end of the examination.

The neurologic examination includes assessment of mental status and behaviour, posture, gait, postural reactions, cranial nerve function, muscle tone and mass, spinal reflexes, and

pain perception. The order in which components are assessed can vary depending on the level of cooperation of the patient and the presenting concern.

Mental status and behaviour

The level of consciousness is influenced by the functional relationship between the ascending reticular activating system within the brain stem and the cerebral cortex. An animal with normal mental status is bright, alert and appropriately responsive to the surrounding environment. Obtundation occurs in animals that are awake but relatively unresponsive to normal environmental stimuli. This is not a specific sign of brain disease and may be seen in association with systemic illness. Stupor is present in an animal that sleeps except when aroused by strong stimuli. Coma is a state of unconsciousness from which an animal cannot be aroused even with noxious stimuli. Stupor and coma are commonly caused by a brain stem lesion that produces a partial or complete, respectively, disconnection of the reticular formation and the cerebral cortex. Stupor and coma can also take place with severe diffuse forebrain disease. Delirium occurs when an animal is overactive and responds inappropriately to stimuli; it indicates forebrain dysfunction.

Behavioural changes such as loss of learned habits (for instance house training), aggression, head pressing, or inability to recognize the owner suggest a forebrain disorder.

Hemi-neglect syndrome, also known as hemi-inattention syndrome, refers to an abnormal phenomenon in which an animal with a structural forebrain disease ignores sensory input from one half of his/her environment (e.g. eats only from one side of the bowl, turns to the opposite direction when called from the ignored side). Since most sensory stimuli are interpreted primarily in the somato-sensory cortex of the cerebral hemisphere contralateral to the side of the stimulus, the side that the patient ignores is contralateral to the side of the lesion.

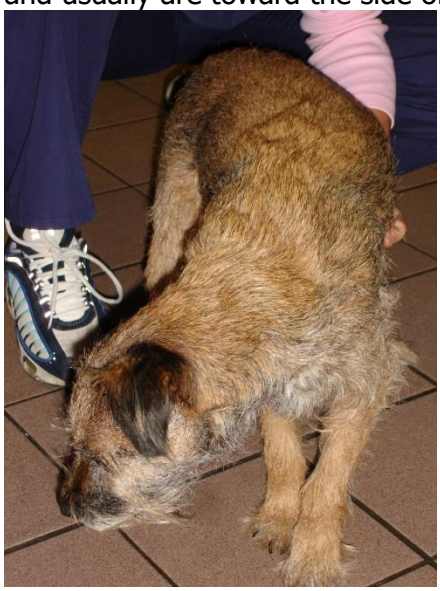
Posture

Normal posture is maintained by coordinated motor responses to sensory inputs from receptors in the limbs and the body, the visual system, and the vestibular system to the central nervous system (CNS). Posture abnormalities include:

- Wide-based stance in animals with vestibular or cerebellar disease, or with a lesion involving proprioceptive pathways between the spinal cord and the cerebral cortex. This stance may also be seen with generalised peripheral nerve disease.
- Knuckling on one or more paws while standing indicates a conscious proprioceptive deficit.

- Head tilt (one ear lower than the other) indicates unilateral vestibular dysfunction. The head is usually tilted toward the side of the lesion. Cerebellar lesions involving the flocculonodular lobe, the fastigial nucleus, or the caudal cerebellar peduncle can also produce a head tilt.

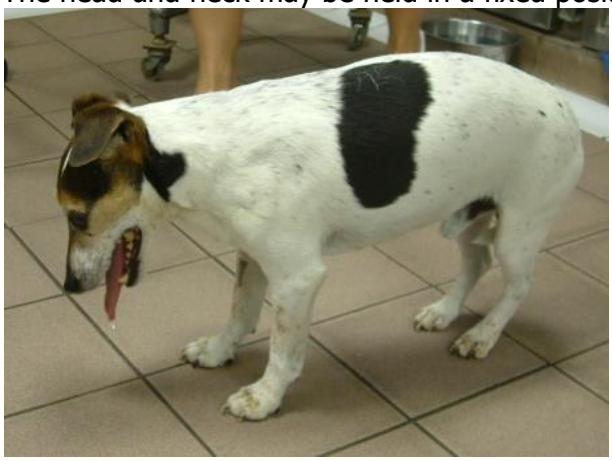
- Head turn (the ears are level, the nose is turned to one side) and Pleurothotonus (the head, neck and trunk are twisted to one side) may occur with cerebral lesions and usually are toward the side of the lesion.



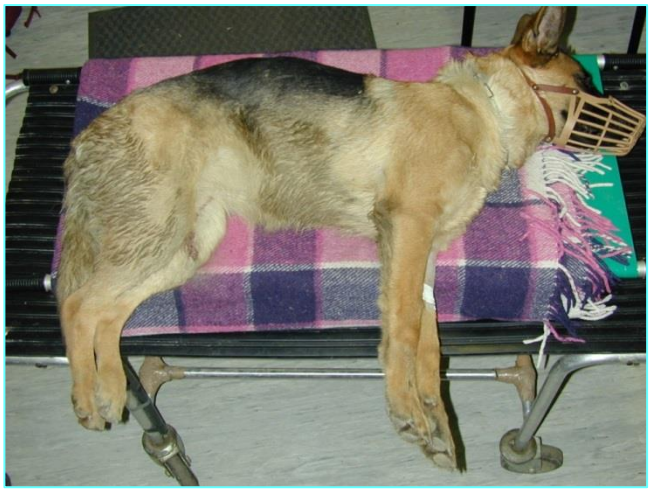
- Head and neck ventroflexion may be observed with certain myopathies (e.g. hypokalemic myopathy in cats) or junctionopaties (e.g. myasthenia gravis in cats)

- Kyphosis is often present in association with painful diseases of the thoracolumbar spine (e.g. intervertebral disc extrusion). Lordosis or scoliosis may occur in animals with vertebral column malformations (e.g. hemivertebrae).

- The head and neck may be held in a fixed position when cervical pain is present.



- Shiff-Sherrington posture is characterised by thoracic limb extension (best appreciated with the animal in lateral recumbency) and pelvic limb paralysis. It results from an acute severe lesion of the thoracolumbar spinal cord which interrupts the ascending inhibitory impulses from the border cells of the lumbar gray matter to the extensor muscle α-motor neurons in the cervical intumescence.



- Decerebrate rigidity is due to a rostral brain stem lesion and is characterised by extension of all four limbs and sometimes opisthotonus. Affected animals are recumbent and typically have decreased consciousness (often stupor or coma).
- Decerebellate rigidity is characterized by opisthotonus, thoracic limb extension, flexion of the hips (pelvic limbs flexed forward), and normal consciousness. It is due to an acute cerebellar lesion mostly involving the rostral lobe which is especially inhibitory to the stretch reflex mechanism of antigravity muscles. If the cerebellar rostral lobe lesion involves the ventral lobules, the pelvic limbs may be rigidly extended away from the body.

Gait

The ability to stand and move requires intact proprioceptive and motor systems. Gait assessment should be performed by observing the patient walking (straight, in circles, up and down stairs) on non-slippery surface. The observer should look at the animal from the side, front and rear. The examiner must be knowledgeable of gait differences among dog breeds. Patients that are recumbent and have no disease that could be exacerbated by movement (e.g. spinal fracture/luxation) should be encouraged/helped to get up and walk if they can, or at least to show any voluntary motor activity.

Gait abnormalities include ataxia, circling, paresis, and lameness.

Ataxia is an inability to perform normal coordinated motor activity that is not caused by weakness, musculoskeletal problems, or abnormal movements. Ataxia can be classified as sensory, cerebellar and vestibular.

- Sensory or proprioceptive ataxia is caused by lesions of the general proprioceptive pathways in the peripheral nerve, dorsal root, spinal cord, brain stem and forebrain. It is characterized by a loss of the sense of limb and body position. The animal has a swinging motion of the affected limbs and scuffing of the toes while walking. It is important to remember that since proprioceptive and motor pathways are intimately associated, sensory ataxia is often compounded by weakness.
- Cerebellar ataxia is caused by cerebellar disease or selective dysfunction of the spinocerebellar tracts. It is characterized by inability to regulate the rate and range of movements with subsequent dysmetria, especially hypermetria.
- Vestibular ataxia is associated with unilateral vestibular dysfunction and is characterized by leaning, drifting, falling, rolling to one side. This type of ataxia is often accompanied by other vestibular signs such as head tilt, circling, strabismus and spontaneous nystagmus.

Circling may occur with unilateral vestibular diseases and with asymmetrical or focal forebrain disorders. The presence of other neurological deficits typical of each syndrome (vestibular versus forebrain) will help to localize the lesion.

Paresis is a partial loss of voluntary movement. Plegia or paralysis is a complete loss of voluntary movement. These two terms are combined with the following prefixes to designate the limb involved: mono (of one limb), para (of both pelvic limbs), hemi (of both limbs on one side of the body), tetra (of all four limbs). There are two types of paresis: upper motor neuron (UMN) and lower motor neuron (LMN), causing a spastic or flaccid paresis, respectively.

Lameness is usually caused by orthopaedic disorders, but it can occur also with neurological diseases affecting a nerve root (e.g. attenuation by a lateralised intervertebral disc extrusion causing the so called "root signature") or a spinal nerve (e.g. nerve sheath tumor).

Abnormal involuntary movements include:

- Tremors are involuntary rhythmic movements of a body part (or of the entire body) produced by alternating contractions of antagonistic groups of muscles. Tremors may be associated with several conditions such as cerebellar disorders (intention tremors), hypomyelination of the CNS, "shaker dog disease", toxin exposure (e.g. hexachlorophene), hypocalcemia.
- Myoclonus is a repetitive rhythmic contraction of a particular group of muscles (e.g. limb flexors, masticatory muscles) and may persist during sleep. It can be associated with encephalomyelitis, especially canine distemper.
- Myotonia is a delayed relaxation of muscle following voluntary contraction. It occurs in certain congenital or acquired muscle disorders.

Postural reactions assess the same neurologic pathways that are involved in gait, that is, the proprioceptive and motor systems. The main value of postural reaction testing is to detect subtle deficits that may not be obvious during the analysis of locomotion (e.g. in animals with forebrain disease). Postural reactions are sensitive indicators of neurologic dysfunction,

but, by themselves, do not provide a specific localization of the lesion within the nervous system. Postural reaction deficits typically occur ipsilateral to peripheral nerve, spinal cord, medulla oblongata and pontine lesions, and contralateral to forebrain and midbrain lesions. Postural reaction tests include the paw placement response (proprioceptive positioning), tactile and visual placing, hopping, hemiwalking, wheelbarrowing, and extensor postural thrust. The occurrence of deficits during two or more of these tests in any limb indicates a significant deficit.

Cranial nerve function

The cranial nerve examination should be performed when the patient is in the most cooperative attitude. A detailed description of the functional neuroanatomy of each cranial nerve can be found in the references. Here, the procedure for cranial nerve examination will be described indicating in parenthesis the specific cranial nerve/s being evaluated. The head is observed for any evidence of a head tilt (vestibular VIII), facial asymmetry related to weakness/ spasm of the muscles of facial expression (VII) or to atrophy of the masticatory muscles (motor V). To elicit the menance response (II-VII) one eye of the patient is covered and the opposite eye is menanced with a threatening gesture of the hand, being careful to avoid mechanical stimulation of the vibrissae; the normal response is a blink (VII) and some degree of head withdrawal. The menance response is a learned response and may not occur until 10 to 12 weeks of age in puppies and kittens. The response may be normally less obvious in brachiocephalic dogs with large eye globes. The palpebral and corneal reflexes are elicited by touching the medial/lateral canthus of the eye and the cornea (sensory V), respectively; the normal response is a blink (VII) and a retraction of the globe (VI). Facial sensation (sensory V) is assessed over the distribution of the three branches: ophthalmic, maxillary, and mandibular of the trigeminal nerve (V). If a sensory deficit is suspected, the most sensitive area to test with a blunt object is the mucosa of the nasal septum inside each nostril, the normal animal will immediately withdraw the head. The temporalis and masseter muscles are palpated to detect any swelling, atrophy, or asymmetry (motor V). The eyes are observed for evidence of strabismus or spontaneous nystagmus (III, IV, VI, vestibular VIII). By moving the head from side to side and up and down the vestibulo-ocular reflex (physiological nystagmus) can be elicited (III, IV, VI, vestibular VIII). The size and symmetry of the pupils (II, parasympathetic III, sympathetic innervation) and their response to light (II-parasympathetic III) are assessed. By opening the mouth we can evaluate jaw tone (motor V), position, movements and symmetry of the tongue (XII), and elicit the gag reflex (IX, X).

Ventrolateral strabismus in the right eye

Muscle tone and mass

Muscle tone is maintained by the muscle stretch reflex (muscle stretch receptor- Ia afferent neurons- spinal cord- lower motor neuron- muscle). Decreased muscle tone can result from injury to this reflex arc or intrinsic disease of the muscle itself. Increased muscle tone can result from a lesion of the upper motor neurons that originate in the brain and have an inhibitory influence on the lower motor neurons. Muscle mass and tone are evaluated with the animal standing (try to have the patient bearing the same amount of weight on the two limbs that are being compared) and in lateral recumbency. Abnormalities include atrophy (early and severe with LMN diseases, chronic and mild with UMN disorders), hypo/atonia (LMN signs), and spasticity (UMN sign). Generalized or localized muscle hypertrophy may be associated with myotonic myopathies or with muscular dystrophies.

Spinal reflexes

Examination of the spinal reflexes tests the integrity of the sensory and motor components of the spinal reflex arch and the influence of descending UMN pathways on this reflex. Spinal reflexes include muscle stretch reflexes (such as extensor carpi radialis and patellar reflexes), thoracic and pelvic limb flexor reflexes, perineal and cutaneous trunci reflexes. Accurate evaluation of spinal cord reflexes, and particularly muscle stretch reflexes, requires a relaxed patient, a muscle placed in optimum position and the application of an adequate stimulus. Spinal reflexes are decreased to absent with LMN disorders and normal to increased with UMN disease. It is important to evaluate muscle tone and spinal reflexes along with the gait abnormality. For instance, in dogs with myasthenia gravis we might observe severe neuromuscular paresis with normal muscle tone and reflexes.

Pain perception (nociception)

Perception of a noxious stimulus should be assessed with the animal relaxed in a quiet environment. It is important to remember that pain is the subjective response of the patient to a noxious stimulus and varies between individuals.

Superficial pain sensation is assessed by grasping and lifting a small fold of skin at the test site with a blunt haemostat. The force of pinch is gradually increased until a response is elicited.

Deep pain sensation is tested by pinching the digits with the fingers or an haemostat. Always apply the minimum stimulus that allows eliciting a response. Normal responses to superficial or deep pain testing include:

- a reflex flexion of the limb or a skin twitch indicating that peripheral nerves, nerve roots (motor and sensory) and spinal cord segments are intact
- a behavioural response, (such as turning the head towards the stimulus, crying or biting), which indicates that peripheral nerves, nerve roots, spinal cord segments and the ascending pain pathways in the spinal cord and brain stem to the forebrain are intact.

The pain pathways that carry superficial and deep pain sensation are different. The latter ones are more resistant to damage than other pathways including those responsible for proprioception, motor function, and superficial pain. Therefore testing deep pain perception is necessary only if superficial pain is absent. In patients with severe spinal cord injuries, the presence or absence of deep pain perception is important in assessing prognosis for recovery. It is very important NOT to confuse reflex flexion of the limb with conscious perception of the noxious stimulus (look for a behavioural response!). Altered states of consciousness (e.g. following trauma and shock) and certain drugs such as analgesics and sedatives may alter results of pain sensation testing.

Superficial pain assessment is particularly useful to map the distribution of a sensory loss in cases of peripheral nerve and brachial plexus injuries. Thorough knowledge of dermatomes and autonomous zones is required.

Finally the spine is palpated in order to detect any areas of hyperalgesia.

The abnormalites recorded during the neurological examination form the "building blocks" of the neurological syndromes. These include: forebrain, midbrain, cerebellar, vestibular, pontomedullary, cervical, cervicothoracic, thoracolumbar, lumbosacral, and multifocal syndromes. The neurological syndrome approach helps performing correct lesion localization within the nervous system. The neurologic signs that can be associated with different neurological localisations are detailed below:

Neurologic signs of **forebrain** dysfunction include:

- Mental status: Normal, obtunded, demented, stupor (less likely)
- Behavior: Normal, hemi-inattention, wandering, vocalizing

- Seizures: Present or absent
- Posture: Normal, head turn (ipsilateral to the side of unilateral/lateralised lesions), pleurothotonus (ipsilateral to the side of unilateral/lateralised lesions), head-pressing
- Gait: Normal, circling (ipsilateral to the side of unilateral/lateralised lesions), movements with lack of purpose
- Cranial nerve evaluation: Normal, decreased or absent menace response, decreased or absent nasal/facial sensation
- Postural reactions: postural reaction deficits (contralateral to the side of unilateral/lateralised lesions)
- Motor function: normal to hemi-tetraparesis
- Spinal reflexes: Intact
- Spinal hyperesthesia: Present or absent, especially in the cervical spine
- Pain perception: Usually normal; may see mild contralateral sensory loss, particularly on the face
- Micturition: May show inappropriate urination and/or defecation

Lesions of the forebrain often cause behavioural change, altered mental status (e.g. obtundation), seizure activity, compulsive walking in circles (usually in the direction of the lesion), and head pressing. Decreased menace response and decreased facial sensation may also be detected.
Hemi-inattention syndrome," or "hemineglect syndrome," refers to a phenomenon in which a patient with a lateralised or unilateral structural forebrain lesion ignores input from one-half of his or her environment. Since most sensory stimuli are interpreted primarily in the cerebral hemisphere contralateral to the stimulus side, the side that the patient ignores is contralateral to the side of the lesion. These patients may eat from only one-half of the food bowl, turn the opposite direction when called by name (i.e. when called from the ignored side), and ignore or have difficulty localizing nociceptive stimuli when applied contralateral to the side of the brain lesion.
Patients with diencephalic lesions may also exhibit sings of endocrine dysfunction (e.g. PU/PD), abnormal eating patterns, and problems with temperature regulation.

Neurologic signs of **brain stem** dysfunction include:

- Mental status: obtunded, stupor, coma
- Posture: normal, head tilt (ipsilateral/contralateral), wide-base stance; recumbent patients may manifest decerebrate or decerebellate rigidity
- Gait: normal, spastic paresis
- Cranial nerves: abnormalities in cranial nerves III to XII may occur uni or bilaterally, central vestibular signs can occur
- Postural reactions: postural reaction deficits in four limbs or ipsilateral to the side of unilateral/lateralised lesions
- Spinal reflexes: Normal to increased
- Spinal hyperaesthesia: absent or can have cervical hyperaesthesia

Lesions from the midbrain through the medulla are more likely to produce severe disturbances of consciousness (stupor, coma) due to impairment of the ARAS

Neurologic signs of unilateral **vestibular** dysfunction include

- Head tilt
- Asymmetric ataxia
- Falling, rolling
- Nystagmus

- Positional ventrolateral strabismus

In dogs with peripheral vestibular disease the nystagmus is generally horizontal or rotatory and not altered with position of head, there are no postural reaction deficits and facial paralysis and/or Horner's signs can occur
In dogs with central vestibular disease the nystagmus can be in any direction, it can change direction when the position of the head is altered, there are postural reaction deficits and cranial nerves V, VI, IX, X can be affected

Neurologic signs of **cerebellar** dysfunction include:

- Mental status: normal
- Posture: head tilt (ipsilateral/contralateral), wide-base stance
- Gait: spastic ataxia, dysmetria, intention tremor, and no obvious signs of weakness
- Cranial nerves: nystagmus may occur but is usually more of a tremor of the globe than the slow-quick (jerk) movements
- associated with vestibular disease
- Postural reactions: postural reaction deficits in four limbs or ipsilateral to the side of unilateral/lateralised lesions
- Spinal reflexes: Normal to increased

The cerebellum coordinates movements. It controls the rate and range of movements without actually initiating motor activity. Head intention tremors are uncoordinated movements that become much worse as the animal initiates an activity, such as eating or drinking. Acute injury to the cerebellum can cause a decerebellate posture, typically extensor hypertonus in the thoracic limbs, flexion in the pelvic limbs, and opisthotonos. Lesions of the flocculonodular lobes of the cerebellum produce signs similar to those of vestibular disease.

Neurologic signs of **spinal cord** disease are detailed in the table below (subdivided by spinal cord regions, not vertebral column regions). Mentation is normal. Behaviour is normal unless affected by spinal hyperalgesia

	C1-C5	C6-T2	T3-L3	L4-S3
Posture	Normal or guarded neck posture, may have wide-base stance in all four limbs, lateral recumbence with severe lesions	Normal; may have low head carriage, guarded neck posture, wide-base stance (esp in PLs), lateral recumbence with severe lesions	Normal or kyphotic posture with painful lesions, may have wide-base stance in PLs	Normal or kyphotic posture with painful lesions
Gait	Proprioceptive ataxia, typically TL = PL), spastic (long-strided) tetraparesis/plegia Ipsilateral hemiparesis/plegia	Proprioceptive ataxia, typically PL > TL), tetraparesis/plegia Ipsilateral hemiparesis/plegia	Normal TLs Proprioceptive ataxia in the PLs Paraparesis or paraplegia	Paraparesis or paraplegia (with spinal cord lesions) Mild proprioceptive ataxia PLs (with spinal cord lesions) Lesions affecting the cauda equina nerves (L6-L7-S1 vertebrae) will only cause paraparesis without ataxia
Cranial nerves	Typically normal May see Horner's syndrome with severe or intramedullary lesions	Typically normal May see Horner's syndrome with severe or intramedullary lesions	Normal	Normal
Spinal reflexes	Normal Hyperreflexia all limbs	Hyporeflexia or absent reflexes TLs Normal to hyperreflexia in PLs	Normal Hyperreflexia PLs (transient hyporeflexia with spinal shock)	Decreased to absent reflexes in the PLs May see pseudohyperreflexia patellar reflex with sciatic lesions
Spinal hyperesthesia	None or pain on palpation or movements	None or pain on palpation or movements	None or pain on palpation or movements	None or pain on palpation or movements
Nociception	Normal Tetraplegic animals may show decreased or absent nociception	Normal Tetraplegic animals may show decreased or absent nociception	Normal Mono/Paraplegic animals may show decreased or absent nociception caudally to the lesion	Normal Mono/Paraplegic animals may show decreased or absent nociception caudally to the lesion
Micturition	Usually normal May have detrusor areflexia-sphincter hypertonia	Usually normal May have detrusor areflexia-sphincter hypertonia	Plegic patients may have Detrusor areflexia-sphincter hypertonia	Normal or detrusor areflexia-sphincter hypotonia

Neurologic signs of **neuromuscular disease** include:

- Normal mental status and behaviour.
- Clinical signs vary depending on the lesion location, e.g. cranial or spinal nerve, sensory or motor component, neuromuscular junction, or muscle.

Peripheral nerve dysfunction generally is characterized by flaccid paresis or paralysis, decreased to absent postural reactions and spinal reflexes, and muscle hypotonia and atrophy.

Cranial nerve involvement results in various clinical signs depending on the affected CN (see cranial nerve examination).

Nociception may be decreased or absent in the dermatome of the affected nerve. Hyperalgesia or paraesthesia can occur. Some neuropathies are exclusively or primarily characterized by motor dysfunction, others by sensory dysfunction and some by a combination of both motor and sensory dysfunction. With mononeuropathies, deficits are restricted to regions innervated by the affected nerve. Polyneuropathies may affect multiple spinal or cranial nerves or both.

Muscle disorders are often characterized by weakness, fatigability and stiff, stilted gait. A postural tremor may be observed. Masticatory muscles, muscles of facial expression or pharyngeal and laryngeal muscles may also be involved. Regurgitation can occur with oesophageal skeletal muscle involvement. Muscle tone may be normal, increased or decreased. Spinal reflexes are usually normal, but may be weak or fatigable, or difficult to evoke in animals with severe muscle atrophy and fibrosis. Palpation may reveal muscle atrophy or less commonly hypertrophy, and sometimes hyperaesthesia. Sensations (including proprioception and nociception) are normal.

Animals with *neuromuscular junction disorders* may have a normal neurologic examination following rest, however various degrees and duration of exercise often results in fatigability

and stiff, stilted gait which improves or resolves following rest. Postural reactions are normal, although profound weakness may affect performance. Spinal reflexes are usually normal but may be fatigable or weak. Muscles of facial expression, pharyngeal, laryngeal, oesophageal skeletal muscles may also be involved resulting in facial paresis, dysphonia, dysphagia and regurgitation.

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