The evolution of the signs should be recognized as progressive, static, improving or waxing and waning. Factors that trigger or improve the signs and previous therapy and its effect on disease course are also important to identify. After determining the chief complaint, collecting the history should end with general information regarding any previous medical or surgical conditions, current medications, family history, vaccination status, diet, previous travel history, drug reactions and the animal's environment, including the potential for toxin exposure.

AIMS OF THE NEUROLOGICAL EXAMINATION

Before rushing into the specifics of the neurological examination, attention should be focused on what questions need to be answered:
- Do the clinical signs observed refer to a nervous system lesion?
- What is the location of this lesion within the nervous system?
- What are the main types of disease process that can explain the clinical signs?
- How severe is the disease?

The first two questions are answered by performing a general physical and neurological examination with a view to defining the neuroanatomical diagnosis (location and distribution of the lesion within the nervous system). By simple observation and testing a number of reflexes and responses (see Hands-off examination [p. 17] and Hands-on examination [p. 22]), the clinician should be able to determine if the animal is neurologically sound or not.

The neurological examination aims to test the integrity of the various components of the nervous system and, if present, detect any functional deficits. Normal findings are as important as the abnormal ones in localizing the lesion. Neurological abnormalities detected on examination should be noted and added to the list of abnormal findings collected from the history. Each of these abnormal findings should then be correlated to a specific region or to specific pathways within the peripheral and/or central nervous system (CNS). An attempt should then be made to explain all of the abnormal findings by a single lesion within one of the regions of the central and peripheral nervous systems, as illustrated in **1**: (i.e. focal forebrain, brainstem, cerebellum, C1–C5 spinal cord segments, C6–T2 spinal cord segments, T3–L3 spinal cord segments, L4–S3 spinal cord segments, peripheral nerve, neuromuscular junction, muscle). Lesions within these regions result in predictable and specific neurological signs. Note that in localizing a lesion, it is not necessary for all the clinical signs referable to one location or syndrome to be present. If a single lesion cannot explain all the listed abnormal findings, the lesion localization is considered as multifocal or diffuse.

▼1 Overview of the central and peripheral nervous systems.

Forebrain
Cerebellum
Brainstem

C1–C5
C6–T2
T3–L3
L4–S3

Peripheral nerve
Neuromuscular junction
Muscle
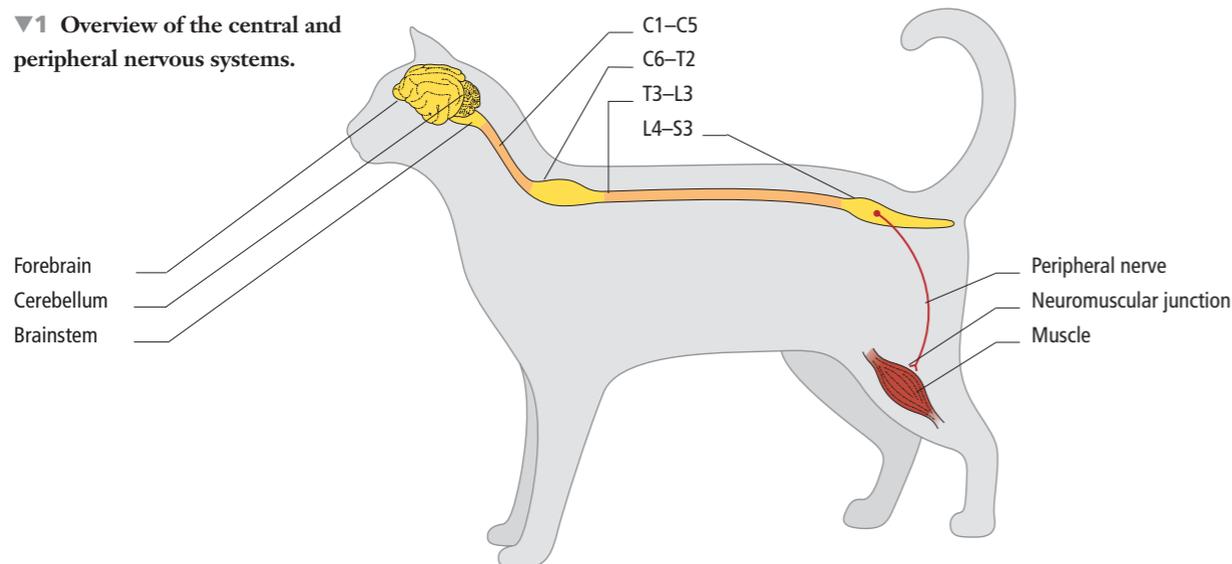
The third question is answered by compiling information on the patient signalment and history of the problem with the neuroanatomical diagnosis in order to determine the differential diagnosis list. Disease severity helps to determine the prognosis of the differential diagnoses. Diagnostic tests are then carried out to investigate the differential diagnoses. The choice and interpretation of these tests must rely on a clear knowledge of the lesion localization within the nervous system and the expected disease processes.

RATIONALE AND PRINCIPLES OF LESION LOCALIZATION

The purpose of the neurological examination is to determine the neurologic abnormalities and, based on that, the location of the lesion or lesions responsible for causing these abnormalities. The location is the anatomical diagnosis. Narrowing down which part(s) of the nervous system may be affected can present a number of advantages:
- From a diagnostic point of view, the differential diagnosis is very dependent on the anatomical diagnosis.
- Aside from determining which part of the nervous system is affected, localizing the lesion also involves determining if the problem is focal, multifocal (i.e. affecting multiple parts of the nervous system) or diffuse (i.e. affecting globally and symmetrically one or more parts of the nervous system). Such information can then be used to further narrow down the differential list (see How to establish a differential diagnosis list, p. 33).
- A number of disease processes may only be diagnosed by exclusion of other causes mimicking a similar clinical history and presentation. This process of exclusion implies evaluating the correct part of the nervous system in order confidently to rule out these similar clinical diseases. Failure to localize the lesion, the interpretation of negative diagnostic test results (as seen with some vascular or degenerative diseases of the CNS) or findings incompatible with the clinical history can end up creating a significant challenge for the clinician.
- Running a limited number of investigations aimed at narrowing down the differential list will result in less cost for the owners and less time spent reaching a diagnosis for the clinician.

SYSTEMATIC APPROACH TO LOCALIZING THE LESION

The neurological examination can be divided into two main parts: hands-off examination and hands-on examination.

Hands-off examination
State of consciousness, awareness and behaviour
The first step in the neurological examination should focus on evaluating the animal's state of consciousness, awareness of its environment and response to being handled.

Disturbances of level or quantity of consciousness are classified in order of severity as obtundation, stupor (semicoma) and coma. Stupor and coma both represent a state of unconsciousness. While a stuporous animal can be roused by a painful stimulus, a comatose animal will fail to respond to any environmental stimulus, including pain. As a rule, altered states of consciousness relate to either a diffuse lesion or widespread multifocal lesions of both cerebral hemispheres or a focal lesion affecting the ascending reticular activating system (ARAS) of the brainstem. The latter functions to arouse the cerebral cortex and maintains the state of wakefulness. Acute coma usually results from extensive brainstem lesions or diffuse forebrain lesions secondary to intoxications or a metabolic disorder.

Common changes in quality of awareness and behaviour include disorientation, aggression, vocalizing, circling, compulsive walking or head pressing. Alterations in the patient's level of awareness and behaviour reflect disturbances in the ARAS and limbic system components of the cerebrum or rostral brainstem.

Circling can be caused by a lesion in the vestibular system as well as by an asymmetrical or focal lesion in the forebrain. Tight circles are usually but not exclusively associated with a vestibular disorder, while wide circles are often associated with a forebrain lesion. With vestibular disease, circling is associated with other signs of vestibular dysfunction (head tilt, nystagmus, positional strabismus and/or falling) and is usually ipsilateral to the lesion (except with lesions affecting the caudal cerebellar peduncle, fastigial nucleus and flocculonodular lobes of the cerebellum). Circling is usually towards the side of a focal or asymmetrical forebrain lesion.

Hemi-neglect syndrome, also known as hemi-inattention syndrome, refers to an abnormal behaviour in which an animal with structural forebrain disease ignores sensory input from one half of its environment (e.g. eating from only one half of the food bowl, turning in the wrong direction in response to sound). This syndrome indicates a diencephalic lesion contralateral to the side ignored by the animal.

Posture and body position at rest

The posture and body position at rest should be evaluated and determined as being normal or abnormal. With reference to lesion localization, a number of characteristic abnormal postures can be encountered in the evaluation of the emergency neurology patient:

• **Head tilt.** This abnormal head posture is characterized by a rotation of the median plane of the head along the axis of the body, resulting in one ear being held lower than the other one. A head tilt indicates a vestibular disorder (peripheral or central) and occurs as a result of the loss of anti-gravity muscle tone on one side of the neck (**2**).

• **Head turn.** Compared with a head tilt, the median plane of the head remains perpendicular to the ground, but the nose is turned to one side (**3**). A head turn is often associated with a body turn (pleurothotonus) and circling. These signs (called adversive syndrome) are usually towards the side of a forebrain lesion.

• **Decerebrate rigidity.** This posture is observed as a result of a rostral brainstem lesion (between the colliculi of the midbrain). It is characterized by rigid extension of all limbs and opisthotonus (extension of the head and neck) associated with a stuporous or comatose mental status (**4**).

• **Decerebellate rigidity.** The rostral part of the cerebellum is inhibitory to the stretch reflex mechanism of antigravity muscles (extensor muscle tone). Lesions at this level can result in opisthotonus, with the forelimbs extended (decerebellate posture). Compared with decerebrate posture, the hips may be flexed by the increased tone in the iliopsoas muscle and mentation remains normal (**5**). This posture is often caused by an acute cerebellar lesion and can sometimes be episodic.

• **Schiff–Sherrington posture.** This posture is observed with an acute severe thoracic or cranial lumbar spinal cord lesion. Such a lesion may interfere with inhibitory ascending neurons that project from the lateral grey matter of the cranial lumbar spinal cord segments cranially to inhibit the forelimb extensor muscles. This posture consists of an extensor hypertonia of the forelimbs, with retention of normal conscious proprioception, voluntary movements and a flaccid paralysis of the hindlimbs (despite the fact that the paralysis is caused by direct interference with the upper motor neuron). This posture is present only in severe and acute lesions, but it does not have prognostic significance.

• **Wide-based stance.** This posture is characteristic of a balance disorder indicating diseases particularly affecting the cerebellum or vestibular apparatus.

Identification of abnormal involuntary movements

• **Myoclonus.** Myoclonus is the clinical sign of sudden contraction followed immediately by relaxation of a specific muscle group. It can be sporadic or repetitive. Sporadic myoclonus can be benign and idiopathic or be a form of simple focal seizure due to a forebrain disorder. Repetitive myoclonus can be constant (often as a result of encephalitis or myelitis caused by distemper virus in dogs), action related (congenital and most commonly caused by a diffuse abnormality of CNS myelination or acquired [e.g. idiopathic generalized tremor syndrome]), postural (head bobber) or episodic (myokymia).

• **Tremor.** With rapid and repeated cycles, repetitive myoclonus demonstrates as a tremor. Tremors can affect all or part of the body and be classified as resting tremors or action-related tremors (also known as kinetic). A resting tremor is present only during rest. An action-related tremor occurs following initiation of voluntary movement. It worsens with increasing levels of activity and disappears with rest. An action-related tremor can be classified in veterinary patients as postural or kinetic.

▲ 2 **Left-sided head tilt and facial paralysis in a 6-year-old Boxer with otitis media/interna.**

▲ 3 **Left-sided head and body turn (pleurothotonus) in a 4-year-old West Highland White Terrier with a left forebrain lesion caused by granulomatous meningoencephalitis.**

▲ 4 **Decerebrate posture in a 7-month-old Weimaraner with head trauma following a road traffic accident.**

▲ 5 **Decerebellate posture in a 2-year-old Hungarian Vizsla with a rostral cerebellar artery infarction.**

• **Myokymia.** Myokymia is defined as undulating vermiform movements of the overlying skin due to contraction of small bands of muscle fibres.

• **Myotonia.** Myotonia is a sustained repetitive contraction of a muscle or group of muscles without relaxation following physiological stimulus. It occurs in certain congenital and acquired muscle cell membrane disorders.

• **Epileptic seizure.** Epileptic seizures are the clinical manifestation of excessive and/or hypersynchronous electrical activity in the cerebral cortex. They can be focal or generalized. An epileptic seizure refers to a forebrain disorder. Its cause may originate from outside (extracranial causes) or inside (intracranial causes) the brain.

- **Movement disorder.** A movement disorder is defined as an episodic sudden involuntary contraction of a group of skeletal muscles in a conscious patient, with a normal sensorium during the activity. Various terms have been used to describe clinical observations in affected animals (dyskinesia, dystonia, chorea, athetosis, ballism; see Chapter 13).

Evaluation of the gait

Examination of the gait should be done in a place where the patient can be allowed to move freely. This is best accomplished by having the owner walk the animal over a non-slippery surface. If the animal is not making any attempt to walk, body support (e.g. a sling or harness) should be provided as necessary so that any subtle voluntary movement can be detected. A normal gait requires intact function of the brainstem, cerebellum, spinal cord and sensory and motor peripheral nerves, neuromuscular junctions and muscles. The cerebrum's contribution to the gait is less important in dogs and cats when compared with primates.

Evaluation of the gait should be done with the aim of determining if the animal is ataxic, paretic or lame (from either peripheral nerve disease or an orthopaedic disorder) and which limb(s) is/are involved.


Gait generation

Gait generation requires the interaction between two motor systems: the upper motor neuron (UMN) system and the lower motor neuron (LMN) system (**6**).

The UMN system is the motor system that is confined to the CNS. It is responsible for the initiation and maintenance of normal movements and for the maintenance of tone in the extensor muscles in order to support the body against gravity. Its cell body lies predominantly within the brainstem. It travels through the brain and/or spinal cord white matter and synapses indirectly (via an interneuron) with an LMN to modulate its activity (essentially via inhibition).

The LMN system is the motor system connecting the CNS with the muscle to be innervated. Its cell body lies within the ventral horn of the spinal cord grey matter or within the cranial nerve nucleus of the brainstem. Its axon leaves the CNS by the ventral nerve roots to join, successively, a spinal nerve and a peripheral nerve before it synapses with an effector muscle. The LMN is the last neuron in the chain of neurons that produces the muscular contraction necessary to maintain posture, support weight and provide the gait (i.e. it is the final common pathway to the effector).

The UMN pathways are responsible for stimulating the appropriate LMN that induces the postural and protraction phases of locomotion.

▼ **6** **Upper motor neuron (UMN) and lower motor neuron (LMN) systems.**

UMN
C6–T2
LMN

UMN
L4–S3
LMN
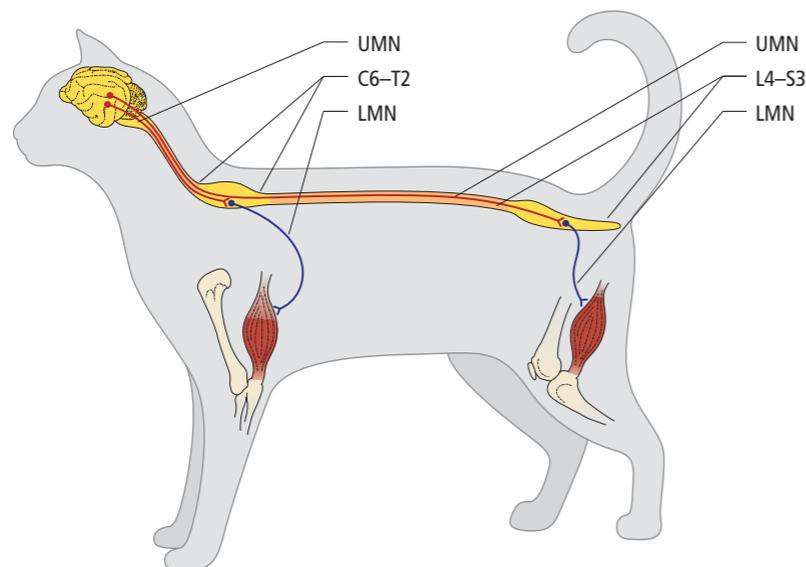
Ataxia

Ataxia is defined as an uncoordinated gait and can arise from either a sensory peripheral nerve or a spinal cord lesion (general proprioceptive ataxia), a vestibular lesion (vestibular ataxia) or a cerebellar lesion (cerebellar ataxia). Ataxia can be further divided into hypometria (shorter protraction phase of gait) or hypermetria (longer protraction phase of gait). General proprioceptive (GP) ataxia reflects the lack of information reaching the 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 patient may walk on the dorsal part of its foot or may drag its digits. These signs often overlap with those caused by UMN paresis (see below). Vestibular or cerebellar ataxias are accompanied by other signs of dysfunction of the vestibular apparatus or cerebellum, respectively (see Chapter 9).

Paresis

Paresis is defined as a loss of ability to support weight (LMN disease) or inability to generate a gait (UMN disease) (see *Table 2*). The term paresis implies that some voluntary movement is still present as compared with paralysis, which refers to a more severe paresis (plegia) with complete loss of voluntary movement. Depending on which limbs are affected, the terms paresis/paralysis can be further defined as tetraparesis/plegia (all four limbs affected, caused by a lesion located cranial to T3 spinal cord segment or a generalized LMN disorder), paraparesis/plegia (hindlimbs affected, caused by a lesion caudal to T2), monoparesis/plegia (only one limb affected, caused by a lesion of the LMN innervating the affected limb) and hemiparesis/plegia (limbs on one side affected due to ipsilateral lesion located between T2 and the caudal midbrain or contralateral lesion located in the rostral midbrain) (see Chapter 10).

Based on whether a lesion affects the UMN or LMN system, two types of paresis can be distinguished: UMN paresis and LMN paresis (*Table 2*):

- UMN paresis causes a delay in the onset of protraction (swing phase of the gait), with the resultant stride being longer than normal and with a stiff quality of movement. Lesions of the UMN system typically result in release of the inhibitory effect that the UMN system has on LMNs located caudal to the level of the injury (dysinhibition). This dysinhibition effect is usually more apparent on the extensor muscles, which results clinically in a spastic paresis/paralysis. Lesions at many different levels of the CNS can produce the same set of UMN clinical signs. Due to their close anatomical relationship within the brainstem and spinal cord, most gait abnormalities

Table 2 **Lower motor neuron paresis/upper motor neuron paresis differentiation criteria**		
CRITERION	**LOWER MOTOR NEURON PARESIS**	**UPPER MOTOR NEURON PARESIS**
Posture	Difficulty supporting weight. Crouched stance as a result of overflexion of the joints	Often normal (unless the animal is paralysed). Abnormal limb position (knuckling, abducted, adducted or crossed over)
Gait	Short strides. Tendency to collapse	Stiff and ataxic strides. Delayed protraction
Motor function	Flaccid paresis/paralysis	Spastic paresis/paralysis
Segmental reflexes	Decreased to absent	Normal to increased
Resting muscle tone	Decreased resistance	Slight resistance
Passive limb flexion/extension	Decreased resistance	Slight resistance
Muscle atrophy	Early and severe neurogenic atrophy	Late and mild disuse atrophy

involving the UMN pathways necessary for gait generation are also associated with some degree of GP ataxia. From a lesion localization point of view, UMN paresis and GP ataxia visible in the gait can occur as a consequence of lesions affecting the brainstem or spinal cord. Apart from lesions caused by peracute disease processes (i.e. infarct, haemorrhage and head trauma), lesions affecting the forebrain cause such a mild contralateral paresis that it is usually not apparent in the gait.

- LMN paresis reflects the degree of difficulty in supporting weight and varies from a short stride, choppy gait to a complete inability to support weight, causing collapse of the limb whenever weight is placed on it. When standing, affected limbs may exhibit a tremor in the muscles. LMN paresis affects the gait with lesions in the peripheral nerves, neuromuscular junction and muscles. Motor deficits observed are ipsilateral to the lesion. Compared with UMN paresis, dysfunction of the LMN does not cause ataxia.

Lameness
Lameness usually presents with a short stride on the affected limb and a long stride on the contralateral limb. Lameness is usually associated with pain from orthopaedic disease. Additionally, it can be associated with nervous system dysfunction referred to as nerve root signature (referred pain down a limb causing lameness or elevation of the limb, resulting from entrapment of the spinal nerve, usually due to a lateralized disc extrusion or nerve root tumour).

Hands-on examination
Postural reaction testing
The primary aim of postural reaction testing is to detect subtle deficits that were not obvious on gait evaluation. In a patient that is recumbent with tetraplegia or paraplegia, evaluation of the postural reactions in the affected limbs is redundant. However, evaluation of the forelimb postural reactions in a paraplegic patient is important in order to detect an abnormality that could suggest a focal cranial thoracic lesion or a multifocal disorder. The postural reactions test the animal's awareness of the precise position and movements of parts of its body, especially the limbs, as well as the animal's ability to generate movement in the part tested.

The postural reactions commonly tested are:
- **The paw replacement reaction**, which is evaluated by placing the paw in an abnormal position (turned over so that the dorsal surface is in contact with the ground) and determining how quickly the animal corrects the paw position (**7**). The majority of the animal's weight should be supported when undertaking this test in order to improve test sensitivity and reduce the interference introduced by orthopaedic disease. Paw replacement reaction can be very difficult to assess in cats that resent having their feet handled. Other postural reaction tests, such as the hopping response, wheel-barrowing and tactile placing, are preferred in this species and should be considered in animals in which the paw replacement reaction is equivocal or difficult to interpret.
- **Hopping response**, which is tested by holding the patient so that the majority of its weight is placed on one limb while the animal is moved laterally (**8**). Normal animals hop on the tested limb in order to accommodate a new body position as their centre of gravity is displaced laterally.
- **Wheel-barrowing**, where the animal's hindlimbs are lifted off the ground by supporting the animal under the abdomen and forcing it to walk forwards (**9**). Abnormal animals may scuff their digits, drag their paws or cross their limbs.

▲ **7** The paw replacement reaction is elicited by gently placing the dorsal part of the foot on the floor. A normal animal should immediately replace its foot in a normal position. This cortically-mediated response tests the conscious awareness of limb position (proprioception).

- **Tactile placing response**, where the animal is lifted and the distal part of the forelimb is brought into contact with the edge of a table. When the dorsal surface of the paw makes contact with the edge of the surface, the animal should immediately place its foot on the surface.

All the components of the peripheral nervous system and CNS that affect the limb tested are needed in order for the animal to perform postural reactions (**10**). These responses are complex in their pathways, but generally involve an afferent arm and an efferent arm. The afferent arm consists of a joint proprioceptor, peripheral sensory

◀ **8** The normal dog responds to hopping by quickly replacing the limb under the body as it moves laterally. The hopping movement should be smooth and fairly rapid and not irregular or excessive. One forelimb should be carefully compared with the other.

▼ **9** Wheel-barrowing is performed with the neck extended and the hindlimbs elevated.

Sensory perception of proprioception in contra-lateral cerebral cortex

Spinothalamic pathway

Decussation in caudal brainstem

Proprioceptive receptors in joints detect abnormal position of paw

▶ **10** Proprioceptive (paw) positioning response in the hindlimb.