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“Gait Assessment in Cats”

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WHAT CAN YOU LEARN FROM THE GAIT IN CATS?

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A normal gait requires intact function of the brainstem, cerebellum, spinal cord and sensory and motor peripheral nerves, neuromuscular junction and muscles. The cerebrum’s contribution to the gait is less important in cats compared to primates. Evaluation of the gait should be done with the aim of determining if the cat is ataxic (uncoordinated), paretic (weak) or lame (from either neuromuscular disease or an orthopaedic disorder) and which limb(s) are involved. Ataxia is defined as an uncoordinated gait and can arise from a peripheral nerve or spinal cord lesion (general proprioceptive ataxia), a vestibular lesion (vestibular ataxia or a cerebellar lesion (cerebellar ataxia). Paresis is defined as a loss of ability to support weight (lower motor neuron disease) or inability to generate a gait (upper motor neuron disease). The term paresis implies that some voluntary movement is still present as compared to paralysis that refers to a more severe paresis with complete (-plegia) loss of voluntary movement.

WHAT ARE YOU TRYING TO ACHIEVE?

The aims of the neurological evaluation of any companion animal are to answer the following questions:

1. Do the clinical signs observed refer to a nervous system lesion?
2. What is the location of this lesion within the nervous system?
3. What are the main types of disease process that can explain the clinical signs?
4. How severe is the problem?

The first two questions are answered by performing a general physical and neurological examination and aim to determine the anatomical diagnosis (location and distribution of the lesion within the nervous system). The third question is answered by compiling the information on the patient signalment and history of the problem with the anatomic diagnosis to determine the differential diagnosis. Disease severity helps to determine prognosis of the differential diagnoses. Diagnostic tests are then carried out to investigate the differential diagnosis. The choice and interpretation of these tests must rely on a clear knowledge of the anatomical diagnosis and the expected disease processes.

DO THE CLINICAL SIGNS REFER TO A NERVOUS SYSTEM LESION?

A number of non-neurological conditions may mimic a nervous system lesion. Orthopaedic problems, cardiorespiratory diseases or metabolic disturbances, to name a few, can easily mimic some common neurological presentations such as gait abnormality, neuromuscular weakness or collapse. Furthermore, some inflammatory, infectious or neoplastic diseases of the nervous may also affect other body system. A detailed clinical examination should therefore be performed before embarking on the neurological
examination. Orthopaedic examination and evaluation of femoral pulses are particularly important when evaluating a cat with abnormal gait. Ophthalmic (and in particular retinal) examination can be particularly useful when evaluating a cat suspected of neurological form of feline infectious peritonitis, lymphoma, systemic arterial hypertension or Toxoplasmosis.

WHY SHOULD YOU ATTEMPT TO LOCALISE THE PROBLEM?

The purpose of the neurologic 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 anatomic diagnosis. Narrowing down to which part(s) of the nervous system may be affected can undeniably present a number of advantages.

From a diagnostic point of view, the differential diagnosis is entirely dependent on the anatomic diagnosis. Aside for determining which part of the nervous system is affected, localising the lesion also involve 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 narrow down even further the differential list (see section how to establish a differential diagnosis list).

Furthermore, 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 to confidently rule-out these mimics. Failure to localise the lesion, the interpretation of any diagnostic test results can end up a very challenging task for the clinician in the face of negative findings (as seen with some vascular or degenerative disease of the central nervous system) or findings that do not match the clinical history.

Finally, running a limited number of investigations aimed at narrowing down the differential list to a specific part of the nervous system can only result in less cost for the owners and less time spent to reach a diagnosis for the clinician.

WHAT ARE THE PRINCIPLES OF LESION LOCALISATIONS?

Before rushing into the specifics of the neurological examination, attention should be focused on what questions are aimed to be answered:

1- Is there any neurological abnormality detected?
2- Which part(s) of the nervous system may be involved to explain these abnormalities?
3- Is the lesion localisation focal, multifocal or diffuse?

The first question does not require any detailed knowledge of neuroanatomy or neuroanatomic pathways. By simple observation and testing a number of reflexes and responses (see section hands-on and hands-off approach), the clinician should be able to determine if the cat is neurologically sound or not.

The neurological examination aims to test the integrity of these various components of the nervous system and, if present, detect any functional deficit. Normal findings are as important as the abnormal ones in localising the lesion. Neurological abnormalities detected on examination should be listed 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. Attempts should then be made to explain all the abnormal findings by a single lesion within one of the following regions of the nervous system: focal forebrain – brainstem – cerebellum - [C1 – C5 spinal cord segments] – [C6 – T2 spinal cord segments] – [T3 – L3 spinal cord segments] - [L4 – L6 spinal cord segments] – [L7 – S3 spinal cord segments], peripheral nerve - neuromuscular junction - muscle. Lesions within these regions of the nervous system result in predictable and specific neurological signs. Note that in localizing a lesion, it is not necessary that all the clinical signs referable to one location or syndrome be present. If a single lesion cannot explain all the listed abnormal findings, the lesion localisation is considered as multifocal or diffuse.

**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 cat 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. Cats with vestibular ataxia often have a broad-based gait (especially in the pelvic limbs) with leaning towards the side of decreased vestibular tone. Some cats 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 cats 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**

It is essential to try and characterise 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 cat'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 cat’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 cat’s awareness of the precise position and movements of parts of its body, as well as the cat’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 (<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 where the menace response is ambiguous. It is tested by carrying a cat towards a tabletop. On approaching the surface the cat will reach out to support itself on the table before the paw touches the table. In the context of an ataxic cat, 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 cat. 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 cats 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**

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

**Causes of vestibular disorder**

<table>
<thead>
<tr>
<th>Vascular</th>
<th>Cerebellar cerebrovascular accident</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Feline Infectious Peritonitis (FIP), Feline spongiform encephalopathy, Fungal diseases, Parasitic encephalomyelitis, Toxoplasmosis</td>
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<tr>
<td>Toxic</td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Trauma</td>
<td>Trauma</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Brain tumours</td>
</tr>
<tr>
<td>Degenerative</td>
<td>Lysosomal storage diseases, Cerebellar abiotrophy</td>
</tr>
<tr>
<td>Developmental</td>
<td>Cerebellar Hypoplasia (Feline Panleukopaenia Virus), Intracranial intra-arachnoid cysts</td>
</tr>
</tbody>
</table>
Causes of cerebellar disorder

<table>
<thead>
<tr>
<th>Causes</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular</td>
<td>Haemorrhage, Fibrocartilaginous embolism</td>
</tr>
<tr>
<td>Infectious</td>
<td>Feline Infectious Peritonitis (FIP), Fungal diseases, Parasitic</td>
</tr>
<tr>
<td></td>
<td>encephalomyelitis, Toxoplasmosis, Epidural abscess</td>
</tr>
<tr>
<td>Trauma</td>
<td>Trauma</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Spinal cord tumours, Vertebral tumours</td>
</tr>
<tr>
<td>Nutritional</td>
<td>Hypervitaminosis A</td>
</tr>
<tr>
<td>Degenerative</td>
<td>Intervertebral disc disease</td>
</tr>
<tr>
<td>Developmental</td>
<td>Spinal intra-arachnoid cysts, Congenital spinal anomalies, Feline Chiari malformation</td>
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Causes of sensory (spinal) ataxia

PARESIS

Neuroanatomical basis of the paretic cat

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

The upper motor neuron (UMN) system is the motor system that is confined to the central nervous system (CNS). It is responsible for the initiation and maintenance of normal movements and for the maintenance of tone in the extensor muscles to support the body against gravity. Its cell body lies within the cerebral cortex, basal nuclei, brainstem or spinal cord. It travels through the brain and/or spinal cord white matter and synapses indirectly (via an interneuron) with a LMN to modulate its activity (essentially inhibitory).

The lower motor neuron (LMN) system is the motor system connecting the central nervous system 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 central nervous system 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 produce muscular contraction necessary to maintain posture, support weight and provide the gait (final common pathway to the effector). The UMN pathways are responsible for stimulating the appropriate LMN that induce the postural and protraction phases of locomotion.
Paresis is defined as a loss of ability to support weight (lower motor neuron disease) or inability to generate a gait (upper motor neuron disease). The term paresis implies that some voluntary movement is still present as compared to paralysis that refers to a more severe paresis with complete (-plegia) loss of voluntary movement. Depending which limbs are affected, the terms paresis/paralysis can be further defined as tetraparesis/plegia (all four limbs affected), paraparesis/plegia (pelvic limbs affected), monoparesis/plegia (only one limb affected), hemiparesis/plegia (limbs on one side affected). Two qualities of paresis can be distinguished: UMN and LMN paresis. UMN paresis causes a delay in the onset of protraction, which is the swing phase of the gait. Lesions at many different levels of the CNS can produce the same set of UMN clinical signs. Due to their close anatomic relationship within the caudal brainstem and spinal cord, most gait abnormalities involving the UMN pathways necessary for gait generation also caused some degree of general proprioceptive (GP) ataxia. From a lesion localisation point of view, upper motor neuron paresis and GP ataxia visible in the gait can occur as a consequence of lesion affecting the brainstem, or spinal cord. Aside for lesion caused by acute disease processes (i.e., infarct, haemorrhage and head trauma), lesion affecting the forebrain cause contralateral paresis that is so mild that it is usually not apparent in the gait. LMN paresis affects the gait with lesions in the peripheral nerves, neuromuscular junction and muscles. Motor deficit observed are ipsilateral to the lesion. LMN paresis reflects degrees of difficulty in supporting weight and varies from a short stride to complete inability to support weight, causing collapse of the limb whenever weight is placed on it. Care must also be taken as many cats can have an apparent plantigrade stance (crouched posture) in a hostile environment such as a consultation room. Compared to UMN paresis, disorder of the LMN does not cause ataxia but only paresis.

Neurological evaluation of the paretic cat

The finding of paresis or paralysis in the absence of intracranial signs indicates spinal cord disease or generalized lower motor unit disease. Brainstem injury is unlikely to cause paralysis without obvious additional signs such as altered states of consciousness, vestibular ataxia, cerebellar ataxia, or cranial nerve signs. Aside for lesion caused by acute disease processes (i.e., infarct, haemorrhage and head trauma), lesion affecting the forebrain cause contralateral paresis that is so mild that it is usually not apparent in the gait. The next goal for the clinician is to further localize the problem as diagnostic approaches, differential diagnoses and treatment options differ for each possible disease location.

STEP 1: Gait evaluation

Initial evaluation of an animal with a gait abnormality should be done with the aim of determining if this cat is ataxic, paretic or lame (from either neuromuscular disease or an orthopedic disorder) and which limb(s) are involved. The locomotor status should be evaluated on a non-slick surface with support if needed. Asymmetry should be assessed and graded. Assessing weakness can be done by describing whether the cat can rise, stand or walk unassisted. Other semi-quantitative descriptors could include how far it can walk without falling, how much support the animal needs to rise or stand, and how much support is needed for
purposeful ambulation. Noting the involvement of thoracic limbs is important as some cats with tetraparesis may have marked weakness in the pelvic limbs and minimal weakness in the thoracic limbs.

In ambulatory cats, gait analysis can help differentiate lower motor neuron from upper motor neuron paresis. Lower motor neuron dysfunction typically results in a short-strided gait and decreased ability to support weight. Dysfunction of the descending upper motor neuron system results in a long-strided, reaching and stiff gait. Cats with a caudal cervical lesion are often described as having a “two-engine gait” with a short, choppy gait in the thoracic limbs and a long-strided, floating gait in the pelvic limbs.

STEP 2: Postural reaction testing

Postural reactions should be evaluated in all cats presenting for paresis and paralysis. Each limb should be evaluated and the results noted on the neurological examination form. The aim of postural reaction testing is primarily to detect subtle abnormalities that were equivocal or not obvious on gait evaluation. The results of gait evaluation and postural reaction testing help to identify which limbs are involved. Muscle tone and segmental spinal reflexes are then tested on each abnormal limb to determine if the lesion is UMN or LMN in nature.

Paw position testing can be very difficult to assess in cats. Other postural reaction testing such as the hopping response, weelbarrowing and tactile placing are preferred in this species. If the patient is reluctant to hop, the cat should be held with three limbs restrained and lowered suddenly to the ground surface with the limb to be tested extended. As soon as the paw strikes the ground the cat should be move laterally to force it to hop on that limb.

STEP 3: Evaluation of muscle tone and segmental spinal reflexes

Muscle tone should be assessed by flexing and extending the limb and joints. Although increased resistance is indicative of UMN signs, it can also be seen in fractious, excitable or painful animals, as well as in association with LMN paresis affecting the flexor system. Diminished tone is a hallmark of LMN signs. Although many spinal reflexes are described, the most reliable one in cats are the withdrawal reflex and the patellar reflex. Other spinal reflexes (triceps, biceps, extensor carpal radialis and gastrocnemius) are more difficult to perform and to interpret. The withdrawal reflex is performed with the cat in dorsal recumbence between the thighs of the examiner. A noxious stimulus is applied to the tested limb by pinching the nail bed or digit with the fingers or haemostat. This stimulus causes a reflex contraction of the flexor muscles and withdrawal of the tested limb. If this withdrawal reflex is absent, individual toes can be tested to detect if specific nerve deficits are present. It should be stressed that the withdrawal reflex in the thoracic or pelvic limbs does not depend on the animal’s conscious perception of noxious stimuli (nociceptive function). The withdrawal reflex is a segmental spinal cord reflex that only depends on the function of the local spinal cord segments. The patellar reflex is elicited by hitting the patellar ligament and observing a reflex contraction of the quadriceps muscle and extension of the stifle joint. It is performed again with the cat in dorsal recumbence between the thighs of the examiner. Evaluation of the cat extensor tone on the pelvic limb can also be used as a control in cats with ambiguous patellar reflex as it involves the same neuro-anatomical
components (femoral nerve and quadriceps muscle). The patellar reflex evaluates the integrity of spinal cord segments L4 to L6 (and associated nerve roots) as well as the femoral nerve. A weak or absent patellar reflex indicates a lesion of the L4 to L6 spinal cord segments or the femoral nerve.

**STEP 4: Evaluation of tail, bladder and anal sphincter**

Tail function is evaluated by assessment of voluntary tail movement, tail tone and sensation. Bladder and urethral function are evaluated by assessment of bladder size, resistance to manual expression, and presence of urine dribbling. The bladder is often large, firm and difficult to express with UMN lesions (i.e. lesions cranial to S1 spinal cord segment); while large, flaccid and easily (but incompletely) expressed with LMN lesions. The anal sphincter is evaluated by assessment of anal tone on digital rectal palpation, presence of anal reflex and sensation.

**STEP 5: Sensory testing**

Sensory evaluation is the final component of the neurological examination. Nociception testing is performed with a small hemostat systematically over the surface of the affected limbs. Application of pressure should only be escalated when the initial stimulus fails to elicit a behavioral response such as turning of the head, vocalizing or an escape behavior. Withdrawal of the limb is only the flexor reflex and should not be taken as evidence of pain sensation.

**STEP 6: Cutaneous trunci reflex and spinal palpation/manipulation**

The cutaneous trunci reflex enables accurate localisation within the T3 to L3 UMN spinal cord segments, and additionally assesses the C8 to T1 region of the brachial plexus (efferent arm of the reflex) via the lateral thoracic nerve from spinal cord segments C8 or T1. The cutaneous trunci reflex can be decreased or absent caudal to a lesion anywhere in this pathway. In the occasional normal animal the cutaneous trunci reflex is either unreliable or totally absent. This test is conducted by stimulating the skin with a pinprick or by pinching with a pair of haemostats, starting at the iliac crest, about one inch lateral to the midline. This should result in a bilateral contraction (or twitch) of the cutaneous trunci muscles. In the absence of such muscle contraction, the point of skin stimulation should be moved cranially until a normal reflex is observed (i.e. cut-off point).

Paraspinal palpation is performed near the end of the examination to ensure the continued cooperation of the patient. The clinician should palpate down the spine feeling for focal pain, muscle spasm and heat. If the dog does not react to light palpation then moderate pressure is applied. The neck is flexed from side-to-side, dorsally and ventrally. The lumbosacral junction is flexed and extended while trying not to flex other joints. A behavioural reaction to what should be an innocuous stimulation can be interpreted as pain. Concurrent administration of anti-inflammatory or analgesic...