NEUROLOGICAL EXAMINATIONS: HOW TO DETERMINE CONDITION

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Categories: Vets

Date: May 19, 2014

MARK LOWRIE MA, VetMB, MVM, DipECVN, MRCVS provides practical guidance on how to determine whether a patient has a neurological condition using traditional methods in the first of a three-part series

MANY veterinary practitioners consider neurology one of the hardest disciplines. This apprehension is heightened when they are faced with an uncooperative patient, whether it is a fractious feline or a catapulting canine. These circumstances frequently result in the clinician neglecting the neurological examination in favour of making assumptions on the most likely cause for the symptoms – with no regard as to the aspect of the nervous system affected or even if a neurological problem is present. This enticement may be heightened if the veterinarian has easy access to CT, MRI or myelography.

It is important to emphasise these tools are not a replacement for the traditional approach to a neurological patient and their absence should not preclude the practitioner from performing a targeted neurological examination to gain a better understanding of a patient’s problem. In these days of austerity, training, experience, clinical skills and a rational approach to a specific complaint assume even more importance, and “shotgun” diagnostics become even less justifiable.

Old-fashioned way is always best

The traditional or conventional method relies primarily on establishing if and where a lesion is in the
nervous system and then drawing up a list of possible causes for that neurological lesion.

This list of differential diagnoses is based on the history, signalment and neuroanatomical diagnosis. The clinician can then carefully select the correct diagnostic procedures to investigate this short list of diseases. The interpretation of the test results relies on a clear understanding of the neuroanatomical diagnosis and the expected disease processes involved. For example, a normal MRI scan of the T3 to L3 spinal segments in a patient with thoracolumbar disease can be diagnostic if certainty exists the test has been performed appropriately. It is the response of a clinician to these negative findings that defines his or her ability to correctly diagnose neurological patients.

The aim of this series of articles is to provide the veterinarian with the necessary clinical tools to tackle the neurological spinal patient, with particular emphasis on ataxia and paresis. The emphasis will be very much on the clinical skills and approach all can learn and develop without the need for expensive diagnostics.

This article will focus on determining whether a patient has a neurological condition or some other cause for the clinical signs.

**Aims of a neurological examination**

Before beginning an examination, it is important to be familiar with the aim of a neurological evaluation, which is to answer the following questions:

- Is the problem definitely neurological?
- What is the location of this lesion in the nervous system?
- What are the main types of disease process that can explain the clinical signs?
- How severe is the disease?

**Is it neurological?**

Spinal cases can present with a wide spectrum of severity – ranging from relatively vague symptoms that may progress gradually with time, to more acute, severe symptoms indicative of spinal cord damage that potentially require emergency management. The majority of patients will have an excellent outcome with the appropriate management.

Although advanced imaging is helpful in confirming a diagnosis, a lot can be ascertained from the clinical and neurological examination. This includes information about the likely diseases causing the clinical signs and the prognosis; that is, is it worth pursuing further investigation? Before
undertaking a neurological examination, it is important to ask “is the patient neurological?”

This can be a very straightforward or challenging question, depending on the individual case. Alterations in ability to ambulate, weakness, altered mental status, apparent pain and paroxysmal events are common presenting signs in animals with neurological disease. However, these signs are not exclusive to neurological conditions.

Recognition of neurological versus non-neurological disease is essential for appropriate diagnostic planning. Inappropriate diagnostics, such as cerebrospinal fluid collection – which require general anaesthesia and entail potential risk – should be avoided when a neurological localisation has not been achieved. Similarly, a neurological localisation is important if expensive procedures such as MRI are to be correctly performed. For example, a dog with referred neck and back pain due to a brain tumour may have normal spinal imaging, but an abnormal brain on MRI (Figure 1). Correct localisation is essential.

Pain manifestations

Apparent pain is often a difficult clinical sign to localise. Abdominal, pelvic, soft tissue and orthopaedic (particularly joint) pain may often manifest with signs such as cervical rigidity, an arched back (kyphosis) and abnormal gait – signs frequently and mistakenly assumed to be associated with neurological disease. Examples of this include bilateral cruciate disease, aortic thromboembolism and polyarthritis, so it highlights why a general clinical examination is so important before a full neurological examination is embarked on.

Potential pitfalls

When determining whether a patient is neurological, we should usually look for some sort of alteration in movement. It is important to remember an animal unwilling to move may look similar to an animal unable to move. Therefore, when a patient is presented with an inability to move, some form of encouragement for it to walk should be given.

Performing a neurological examination on a patient with non-neurological disease can cause very confusing results. A dog with severe orthopaedic pain will be reluctant to replace its paw when testing conscious proprioception. A very weak patient with a severe medical condition would also be unable to stand and walk, and may also give the illusion of having delayed paw positioning responses. Therefore, it is imperative a complete general clinical assessment is performed in a patient before consideration is given to a neurological examination, so as to prevent misleading results.

Non-neurological conditions mimicking neurological disease
A classic example of neurological disease being mimicked by non-neurological conditions is dogs and cats with aortic thromboembolism.

These patients will present with some degree of paresis or paralysis to one or both back legs.

If the problem is acute and severe, a femoral pulse deficit is usually obvious. However, some of these patients, particularly dogs, may present with an insidious and chronic onset of a pelvic limb gait abnormality or exercise intolerance. A femoral pulse deficit is expected, although this may only be reduced as opposed to absent. If this is not detected on general clinical examination, the neurological examination can be misleading and difficult to interpret – leading to an erroneous localisation.

**Observe the gait**

Not all dogs that are “off their legs” have neurological disease. Gait evaluation is one of the best ways to determine whether a problem is neurological, and the presence of ataxia (incoordination) would usually imply a neurological condition is present. In a clinic it is very common for animals that are “off their legs” to be presented in the back of a car or nestled tightly in an owner’s arms, as the problem has occurred acutely and owners are reluctant to move pets and simply want to offer them comfort. As a consequence, this commonly leads to the pitfall of examining the patient without first observing the gait.

If a patient is carefully supported and encouraged to walk, it is often surprising how well it can ambulate. A sling under the back legs and a harness around the chest will provide an acceptable means to evaluate which limbs are affected and whether movement is present in these affected limbs.

**Basic neuroanatomy**

Unfortunately, neuroanatomy plays a small role in understanding whether a patient is neurological. Spinal cord tracts can be divided into afferent (ascending or sensory) and efferent (descending or motor) pathways.

**Sensory (ascending/ afferent) function**

Lesions affecting sensory function (that is, lesions affecting the peripheral sensory nerve, the ascending proprioceptive pathways in the dorsal spinal cord and the sensory areas of the brain) will result in ataxia and can be assessed by:

- observation of gait to allow detection of ataxia; and
- paw position response – testing conscious proprioception.
Motor (descending/efferent) function

Lesions affecting motor function (that is, lesions affecting the motor centres of the brain, the descending motor pathways in the ventral spinal cord, and the peripheral motor nerve and muscles) will result in weakness, paresis or paralysis (depending on the severity) and can be assessed by:

• Observation of gait to allow detection of weakness and determine if movement is present in the affected limbs.

• Hopping – my preferred method of assessing motor function, as it can be done in any animal regardless of size. It allows each individual limb to be assessed for weakness and allows for an easy comparison between limbs (Figure 2).

Physiology of gait generation

The nervous system obtains sensory information from receptors, allowing identification of changes in an environment (processing), resulting in transmission of information to effectors to make an appropriate response. Therefore, in terms of proprioception, stretch receptors in the muscles, joints and tendons convey sensory information regarding limb position in peripheral sensory nerves to the spinal cord, where it ascends (in the spinocerebellar, spinothalamic and dorsal tracts) to the brain.

The vestibular system provides similar information, allowing balance and posture to be maintained regarding the head and trunk, which is then organised in the cerebellum to moderate activity in the descending motor neurons and correct changes in body orientation and posture. Information also reaches the forebrain for integration into the consciousness.

A motor response is generated by connections from these processing centres to motor nuclei in the brainstem. Descending motor tracts then deliver this coordinated information via the spinal cord to the effector organs (that is, skeletal muscles) via lower motor neurons. Two types of gait abnormality are possible with spinal cord disease that may occur alone or in combination – ataxia and paresis.

Ataxia

Ataxia is a vague and non-specific term for an abnormality in the gait that originates from the ancient Greek words “a” meaning “without” and “taxis” meaning, “order”. Ataxia is due to a defect in proprioception, that is, the ascending pathways collecting and processing information regarding movement perception and spatial orientation. In simple anatomical terms, lesions of the sensory nerve, ascending pathways of the spinal cord and processing centres within the brain cause ataxia. It is not due to defects in descending motor tracts (paresis). Disturbances in these ascending pathways cause the nervous system to fail to initiate conscious and reflex motor activity,
resulting in an ataxic gait (that is, one where the positional placement of the paws is inappropriate with each stride, resulting in a different placement of the limb; Figure 3).

Ataxia can be divided into three types – cerebellar, proprioceptive and vestibular. The three forms of ataxia arise from lesions at different points in the ascending pathway.

- Vestibular ataxia describes defects in the vestibular system and associated tracts. Clinical signs most commonly manifest as a head tilt with abnormalities in eye position (for example, strabismus and nystagmus) and difficulty in maintaining balance (falling and leaning to either side).

- Cerebellar ataxia is characterised by an inability to regulate the rate, range and force of a movement. No direct pathways run from the cerebellum to the spinal cord, meaning cerebellar lesions do not result in paresis. Instead, the loss of inhibitory control of the descending motor tracts from cerebellar disease results in uncontrolled and exaggerated spastic movements, for example, dysmetria and hypermetria.

  Postural reaction testing (for example, hopping and placing) may yield a mildly delayed, followed by an exaggerated, response. Further evaluation of a dog with cerebellar ataxia may also reveal a decreased menace response and an intention tremor of the head and body.

- Proprioceptive ataxia is caused by lesions in the ascending sensory pathway alone and is not due to disturbances in the descending motor tracts; hence true “proprioceptive” ataxia is not accompanied by spasticity, paresis or involuntary movements. However, due to the anatomical proximity of the descending motor tracts to the ascending sensory tracts in the spinal cord, it is rare to have a spinal cord lesion resulting in ataxia without some degree of paresis. Therefore, the term paraparesis is preferred terminology in the context of pelvic limb ataxia.

Proprioceptive fibres are larger in diameter and are situated in the periphery of the spinal cord, while the motor fibres are smaller and located deeper in the cord.

As a result, proprioceptive ataxia is often the first neurological sign to be observed in patients suffering spinal cord compression followed by motor deficits, that is, paresis. In practice, this distinction is rarely evident.

**Paresis**

Paresis is defined as weakness or an inability to generate a gait. Patients with paresis appear weak, with no power to the affected limb(s), although the placement of the paws is in the correct position with no incoordination (Figure 4).

The term paresis implies some voluntary movement is present as compared to paralysis (or plegia), in which complete loss of voluntary movement is observed. Depending on which limbs are
involved, paresis/paralysis can be subdivided into four groups.

• Tetraparesis/plegia is paresis or paralysis of all four limbs, and results from a lesion located cranial to the T2 spinal cord segment or from a generalised lower motor neuron disorder.

• Paraparesis/plegia is paresis/paralysis of the pelvic limbs caused by a lesion caudal to T2.

• Monoparesis/plegia refers to paresis/lysis of one limb caused by a lesion of the lower motor neuron innervating the affected limb.

• Hemiparesis/plegia is paresis/lysis of the limbs on one side of the body due to a lesion located cranial to T2 is known as hemiparesis or plegia. It is ipsilateral to a lesion located between T2 and the mid-brain, but contralateral to a lesion located in the rostral mid-brain or cerebrum.

Paresis can affect the upper or lower motor neuron. This results in two types of paresis – upper motor neuron and lower motor neuron paresis.

The upper motor neuron system is confined to the central nervous system (within the brain and spinal cord) and is responsible for the initiation and maintenance of movement and the maintenance of tone in extensor muscles.

Upper motor neuron paresis results from a lesion in these descending motor pathways in the brain or spinal cord, causing a spastic paresis (Table 1) with 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 upper motor neuron typically result in a release of inhibition on lower motor neurons located caudal to the level of the injury. This effect is most profound on the lower motor neurons to the extensor muscles, resulting in a spastic paresis or paralysis. Lesions at many different levels of the central nervous system will produce the same upper motor neuron clinical signs and may also result in some proprioceptive ataxia in view of the close relationship between the ascending proprioceptive and descending motor pathways in the spinal cord and brainstem.

The lower motor neuron system connects the central nervous system with the muscle to be innervated.

It consists of a motor neuron with the cell body located in the grey matter of the spinal cord or in the cranial nerve nucleus of the brainstem. The axons leave the central nervous system coursing to the effector muscle. The lower motor neuron is the final part of the pathway involved in producing muscular contraction, supporting weight and generating gait.

Lesions of the lower motor neuron produce a flaccid paresis/paralysis (Table 1). Depending on the
severity, affected patients will struggle to support their own weight and collapse on the affected limbs when attempting to stand. Lower motor neuron paresis affects the gait with the lesion being in the peripheral motor nerve, neuromuscular junction or muscles.

As previously mentioned, upper motor neuron paresis may be seen with ataxia due to the close proximity of the two pathways. In contrast, however, dysfunction of the lower motor neuron very rarely results in ataxia. The exception is with the neuropathies affecting the motor and sensory nerves, which can present with ataxia and lower motor neuron paresis. However, thankfully these conditions are extremely rare.

Summary

The presence of ataxia implies a lesion within the central nervous system or sensory nerves. If ataxia is observed then the suspicion of a neurological condition is increased, provided no sedative drugs (for example, opioids) have just been administered.

Ataxia would certainly rule out an orthopaedic condition, for example. If paresis is present, this symptom does not quite as definitively rule in a neurological process.

Many diseases can result in profound secondary weakness, from orthopaedic to medical conditions. Therefore, the general clinical examination is even more important to determine whether the paretic patient is neurological.
Figure 1a. A six-year-old beagle presented with the primary complaint of yelping and severe neck pain. On initial presentation, the dog had a low head carriage and obvious signs of neck pain based on the arched back and low head carriage. However, subtle neurological deficits were present on a full examination that suggested a forebrain lesion was present.
Figure 1b. A sagittal (A) and transverse (B) T2-weighted MRI scan of the dog in Figure 1a. The dotted line in A shows the level at which the transverse image B is taken. This confirms the presence of a large forebrain mass compatible with a forebrain neoplasm.
Figure 2. The hopping reaction is tested by holding the patient so the majority of its weight is on one limb while the animal is moved laterally. Normal animals hop on the tested limb to accommodate a new body position, as their centre of gravity is displaced laterally. An equal response should be seen on both sides. Subtle ataxia or weakness of one limb may be detected with this test, resulting in the dog losing balance and falling on one side.
Figure 3. Screenshots (A-D) of a four-year-old Jack Russell terrier with an acute onset of ambulatory proprioceptive ataxia in the pelvic limbs (paraparesis). Notice the crossing of the back legs and severe swaying of the trunk. The presence of weakness in the left pelvic limb (E) manifests by a delayed paw positioning response. This patient had a T3 to L3 localisation and was diagnosed with an acute disc herniation (Hansen Type I).
**Figure 4.** Screenshots (A-H) of a five-year-old fox terrier examined because of an acutely progressive history of non-ambulatory paresis affecting all four legs (tetraparesis). Notice the profound weakness to all four limbs and the requirement for support. However, placement of these limbs is appropriate with no incoordination. The patient had a neuromuscular localisation and was diagnosed with idiopathic polyradiculoneuritis, making a complete recovery with nursing care and rehabilitation alone.

<table>
<thead>
<tr>
<th>Upper motor neuron signs</th>
<th>Lower motor neuron signs</th>
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<tbody>
<tr>
<td>Reflexes</td>
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<tr>
<td>Atrophy</td>
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<tr>
<td>Tone</td>
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<tr>
<td>Location</td>
<td>Occur with brain, C1 to C5, and T3 to L3 lesions</td>
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**Table 1. Clinical signs that may be seen with upper and lower motor neuron signs**