



NEUROLOGICAL EXAMINATION OF THE CAT MADE SIMPLE: Part One

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Performing and interpreting a neurological exam in cats can present a particular clinical challenge to any vet. In the first of this two part article, Jeremy Rose, Senior Clinical Training Scholar in Neurology, takes us through a step-by-step approach to the neurological examination of the cat to help diagnose those trickier feline patients.

Neurological problems are not uncommon in cats, but can present a significant diagnostic challenge. Both performing and interpreting the neurological examination can be problematic in feline patients because cats are not always cooperative during examination and findings may not always be consistent or reproducible.

If you suspect your feline patient may have a neurological problem, start by taking a full history and performing a full general clinical exam. This is essential for identification of potentially related problems with other organ systems, incidental findings and factors that may need to be taken into account when performing and interpreting the neurological exam itself. It is also important to remember that a normal neurological exam does not in itself rule out a neurological problem, for example a cat may suffer from seizures due to a neoplastic cause and have no other signs of forebrain disease on exam. In this case, a detailed history is critical in determining the nature of your patient's signs. A video of episodes recorded by the owner can also be useful in these situations.

AIMS OF THE NEUROLOGICAL EXAM

There are two main aims of the neurological exam:

1. To determine if your patient's problem is neurological
2. To localise where the lesion(s) is/are within any of eight anatomical regions (*the forebrain, the brainstem, the vestibular-cerebellar system, spinal cord segments C1-C5, C6-T2, T3-L3, L4-S3 and the neuromuscular system*). Lesions may be described as multifocal if more than one of these regions are affected.

Major abnormalities that can be associated with each of these regions are summarised in table 1, however, it is important to note that other localisations may be possible with some of these signs and that other clinical signs may be found with lesions at each location.

This table constitutes a simplistic summary and due to the limitations as such, it is recommended that more comprehensive texts (such as the BSAVA Manual of Neurology) be consulted for further information regarding localisation.

Forebrain:

Altered mentation and behaviour*, seizures*, narcolepsy/cataplexy, movement disorders, head turn*, head pressing, pacing*, circling*, hemineglect, central blindness* (decreased menace response with normal pupillary light reflex (PLR)), normal to reduced postural reactions and normal to upper motor neuron (UMN) signs in forelimbs and hind limbs.

Brainstem:

Altered mentation*, deficits in cranial nerves (CN) 3-12*, decerebrate postures, respiratory and cardiac abnormalities, gait changes* (most commonly in all four limbs), UMN signs in forelimbs and hindlimbs.

Vestibular-cerebellar system:

- Cerebellum: Intention tremor, dysmetria*, truncal ataxia*, decerebellate posture, ataxia in all four limbs*. Menace deficit with normal vision and PLR*, vestibular signs (see below*), anisocoria.
- Peripheral vestibular system: Circling*, head tilt*, ataxia (usually of all 4 limbs)*, nystagmus* (spontaneous or positional, usually horizontal or rotary with fast phase away from the side of the lesion), strabismus, facial nerve deficits (due to the course of CN through the middle ear), Horner's.
- Central vestibular system: Any of the peripheral vestibular signs above, although nystagmus can be horizontal, vertical, rotary, or variable, in addition to abnormal mentation*, deficits in any CNs (but 5-12 most common) and decreased postural reactions* (in particular paw placement and tactile placement), with normal-increased muscle tone and normal-increased spinal reflexes.

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Table 1. Summary of clinical signs associated with the eight major neuroanatomical locations.

*Abnormalities that are common signs for a particular localisation are marked with *.*

C1-C5:

Posture/gait changes (forelimbs and hindlimbs), decreased postural reactions (forelimbs and hindlimbs) with UMN signs in forelimbs and hindlimbs*. There may also be Horner's, spinal pain or loss of sensation/pain and an UMN bladder.

C6-T2: (cervicothoracic intumescence):

Posture/gait changes (forelimbs and hindlimbs), decreased postural reactions (all four limbs).

With LMN sign in forelimbs and UMN in hindlimbs*. There may also be spinal pain, UMN bladder and an absent cutaneous trunci reflex.

T3-L3:

Posture/gait changes in hindlimbs only, postural reactions decreased in hindlimbs. UMN signs in hindlimbs with normal forelimbs*. Schiff-Sherrington posture may be present (but is rare in cats).

There may also be spinal pain, reduced/absent cutaneous trunci reflex, hypoalgesia in hindlimbs, or an UMN bladder.

L4-S3 (lumbosacral intumescence):

Posture/gait changes in the hindlimbs only.

LMN signs in hindlimbs with normal forelimbs*.

There may also be tail paresis, anal sphincter dilation, hindlimb/perianal/tail hypoalgesia, spinal/lumbosacral pain and a LMN bladder.

Neuromuscular:

Deficits in any cranial nerves (CNs) (7, 9, 10 most common in generalised neuromuscular disorders), flaccid paresis OR stiff/exercise intolerance (myopathy). Normal-abnormal postural reactions in all four limbs, decreased (most common) to normal to increased muscle mass and/or muscle tone in all limbs, decreased to absent spinal reflexes in all limbs, normal to decreased sensation, muscle hyperaesthesia.

specific tests to examine your patient's reflexes and responses (details on performing tests will be published in the second part of the article).

It is important to complete your neurological exam as comprehensively as your patient will allow before attempting to localise your lesion in order to allow assimilation of knowledge. For example, a facial paresis in the absence of other deficits is unlikely to have a forebrain or brainstem localisation as these localisations would usually have a concurrent change in mentation.

PART 1 - OBSERVATION

This part of the exam is usually performed in a quiet room with the cat left in the opened basket on the floor. Cats should ideally be given time to habituate to the environment in which they are assessed. Observing the cat getting out of the basket is often valuable in assessing gait and posture.

Observation is a particularly pertinent part of the exam of cats, as any abnormalities identified can then be prioritised for further localisation with the hands on tests when the exam may be limited by the patient's temperament. Evaluation of the symmetry of the cat throughout the observation stage of the exam is helpful, as asymmetry (for example of the cat's features, posture or gait) can be indicative of an abnormality.

1. Assess behaviour

Abnormal behaviours include hemi-neglect syndrome (a problem with sensory input from the environment on one side, which may manifest, for example, as only eating one side of a bowl of food), persistent pacing, seizures or head pressing.

These signs are always indicative of forebrain involvement. It is important to ask the owner specifically regarding

any (and other) abnormal behaviours at home when taking a history, remember that signs may be subtle, for example, with partial seizures and must be distinguished from other neurological and non-neurological causes such as metabolic or electrolyte

disturbances, learned behaviours or other causes of myoclonus.

2. Assess the level of consciousness

Changes in consciousness indicate forebrain or brainstem involvement, however, as for behavioural changes, extracranial conditions can impact on the forebrain resulting in changes to consciousness.

Observe for mania (inappropriate exaggerated

Table 1 (continued from previous).

| Signs | Upper Motor Neuron Signs | Lower Motor Neuron Signs |
|-------------|--------------------------|--------------------------|
| Reflexes | Normal to increased | Decreased |
| Muscle mass | Normal | Normal to decreased |
| Muscle tone | Normal to increased | Decreased to absent |

Table 2: Reference to Upper Motor Neuron and Lower Motor Neuron signs.

Once you have determined the location of your lesion, it is then possible to deduce a list of sensible differential diagnoses and then select appropriate diagnostics in order to correctly determine the diagnosis, prognosis and management of each case.

PRACTICAL APPROACH

There are two main components to the neurological exam in any animal. Firstly, it is essential to observe your patient thoroughly and then secondly, perform

behavioural response to environmental stimuli), obtundation (marked inattentiveness and reduced responsiveness to environmental stimuli), stupor (unconsciousness with a significantly reduced response to environmental stimuli, but can be roused with pain), or comatose state (unconsciousness with absence of response to environmental stimuli, including pain).

3. Observe for cranial nerve abnormalities

Nystagmus (involuntary rhythmical movement of the eyeballs when the head is still). Nystagmus can be an incidental finding in some cats (e.g. congenital pendular nystagmus in Siamese, Birman and Colourpoint breeds), but is usually associated with an abnormality in the vestibular-cerebellar system.

Determine the nature of your nystagmus (vertical, horizontal or rotary) and the direction of the fast and slow phases. With a peripheral vestibular lesion, nystagmus is usually horizontal or rotary and the fast phase of the nystagmus is usually away from the site of the lesion. With a central vestibular lesion, nystagmus can be horizontal, rotary or vertical and can be towards or away from the lesion. A nystagmus that varies in direction is usually central in origin.

Anisocoria (different sized pupils under the same light conditions). Observe pupil size in both light and dark conditions, as well as assessing pupillary light response (see later) to ascertain which eye is affected and if it is a failure of dilation (miosis) or failure of constriction (mydriasis) that is present.

Abnormalities in the following structures can result in an anisocoria:

- globe (can cause miosis or mydriasis in the affected eye)
- oculomotor nerve (causes mydriasis on the ipsilateral side) sympathetic input to the eye (causing Horner's syndrome i.e. miosis, ptosis and enophthalmos ipsilaterally to the lesion) *See fig 1.*
- brainstem, including oculomotor nuclei (causes mydriasis on the ipsilateral side to the lesion)
- forebrain (causes miosis on contralateral side to the lesion)
- cerebellum (can cause mydriasis contralaterally or ipsilaterally to the lesion)

Facial droop/paralysis - observe for abnormalities in lip and ear symmetry and for inability to blink. A facial droop may indicate an abnormality in the neuromuscular system (facial musculature, facial nerve and neuromuscular junction (NMJ), the brainstem or the forebrain (rare)). If facial nerve involvement is suspected, a Schirmer tear test should be performed as part of your hands on exam as a parasympathetic branch controls tear



Figure 1. Horner's syndrome affecting the left eye.

Strabismus (visual axes of both eyes are not parallel to one another) - a very mild strabismus, without other neurological signs, may be found as an incidental finding in some cats and congenital convergent strabismus has been reported in Siamese, Birman and Himalayan breeds. However, strabismus may indicate a lesion in the neuromuscular system (including the extra-ocular musculature, the oculomotor, trochlear or abducens nerves and the NMJ), the brainstem, the forebrain or the vestibular-cerebellar system.

The direction of the strabismus is important and may offer clues as to the origin. Lesions to the oculomotor nerve (or associated nuclei which are located in the brainstem) may cause a ventrolateral strabismus. Lesions to the trochlear nerve or nuclei may cause a dorsolateral strabismus and lesions to the abducens nerve or nuclei may cause a medial strabismus.

Decreased masticatory muscle mass - may indicate a lesion in the neuromuscular system (including temporal, masseter, digastric or pterygoid muscles, trigeminal nerve or the NMJ) or the brainstem.

Inability to close the mouth - inability to close the mouth in the absence of a non-neurological cause, such as oral obstruction, retrobulbar lesion or orthopaedic problem, may indicate a lesion in the neuromuscular system (including the local muscles, bilateral trigeminal nerve deficit (uncommon) or NMJ) or the brainstem.

4. Observe gait and posture

Look specifically for the presence of the following:
Head tilt (*See figure 2 - rotation of the head about the axis of the median plane of the skull i.e. one ear or eye is lower than the other*). A head tilt usually reflects a vestibular-cerebellar lesion (and can include the brainstem), but can rarely be seen in forebrain disease and high cervical lesions (C1-C3). A head tilt is usually towards the side of the lesion, except in a paradoxical vestibular lesion, which occurs secondary to a central vestibular-cerebellar disorder.



Figure 2. Severe left-sided head tilt in a kitten with a middle ear polyp.

Head turn (nose turning to one side of the body but the median plane of the head remaining perpendicular to the ground). A head turn indicates a forebrain lesion and is usually toward the side of the lesion.

Circling - can indicate a forebrain or vestibular-cerebellar disease. Circling is usually towards the side of the lesion (except in central vestibular-cerebellar disease, where it can be either towards or away from the side of the lesion). Smaller circles are often associated with vestibular lesions and wider circles with forebrain disease, but this is variable.

Specific postures (decerebrate, decerebellate and Schiff-Sherrington).

- Decerebrate posture, characterised by extension of forelimbs and hindlimbs with opisthotonus and a stuporous or comatose mental state, is due to a brainstem lesion and carries a poor prognosis.
- Decerebellate posture, characterised by extension of forelimbs with possible flexion or extension of the hips, opisthotonus and a normal mental state, is due to a rostral cerebellar lesion (usually acute i.e. vascular) and does not necessarily indicate a poor prognosis.
- Schiff-Sherrington posture, characterised by extension of forelimbs and possible opisthotonus with a normal mental state, is associated with a T3-L3 spinal cord lesion and is not a prognostic indicator. It is usually worsened by lateral recumbency and patients have normal proprioception in the forelimbs.

Ataxia and paresis – Ataxia is incoordination caused by a sensory deficit and may be of three types: general proprioceptive, vestibular or cerebellar. Paresis is weakness caused by a motor deficit and may be of two types: upper motor neuron paresis or lower motor neuron paresis. Observe the cat walking (on a non-slip surface in an area with sufficient space). It can be difficult to distinguish between ataxia and paresis (for example

in cats affected by a C1-C5 myelopathy) and this is often unnecessary but, it is important to decide which limbs are affected to help with neurolocalisation. If you are able to identify your patient as ataxic or paretic, you can try to determine which type is present.

With **general proprioceptive ataxia** (*loss of awareness of where the limbs are in space*), there is commonly a delay in the onset of protraction (the swing phase) of the limb, excessive flexion, adduction or abduction and the presence of ‘knuckling’ the paw(s) over onto their dorsal surface.

Vestibular ataxia (loss of balance) may be observed as a tendency to lean, drift, fall or roll to a particular side and is often accompanied by an abnormal nystagmus, strabismus and head tilt. Bilateral vestibular lesions often result in a low crouched posture with wide head excursions from side to side and no head tilt.

Cerebellar ataxia (inability to modulate gait generating systems) usually results in limb movements that appear to lack control, are abrupt in onset, show overflexion on protraction and have an abnormal site of limb placement (i.e. hypermetric gait). Cerebellar lesions often result in a wide-based stance, truncal sway, intention tremors and menace deficits in the presence of normal visual tracking and PLRs. Cerebellar ataxia may be accompanied by vestibular signs due to significant components of the central vestibular system being located in close proximity to the cerebellum. Several congenital cerebellar conditions have been reported in cats, including cerebellar abiotrophy, cerebellar degeneration and cerebellar hypoplasia. Note that purely cerebellar lesions have normal mentation and no paresis.

Lower motor neuron (LMN) paresis is usually observed as difficulty to support weight, which may cause a short-stride length (and therefore mimic lameness), a tendency to collapse, trembling and/or neck flexion. Upper motor neuron (UMN) paresis commonly causes a delay in the onset of protraction and a longer stride with a variable degree of spasticity.

To see a video of a neurological exam being performed, please visit: www.felineupdate.co.uk

The next article, discussing the hands on neurological examination, will be published in the next edition of the Feline Update.

References

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