#### A VIDEO TOUR OF THE NEUROLOGICAL EXAMINATION

Simon Platt

BVM&S MFCVS DACVIM (Neurology) DECVN College of Veterinary Medicine, University of Georgia

**Abstract:** In this session, we will use video clips to demonstrate normal vs. abnormal dogs and cats being evaluated for neurological conditions. We will look at the practical way in which to evaluate all patients and determine where their lesions are localized.

Keywords: Neurological Examination, postural reactions, cranial nerves

The nervous system plays a role in nearly all body processes. Disease syndromes may affect the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system, which includes cranial nerves, spinal cord nerve roots, spinal nerves, peripheral nerve branches, and the neuromuscular junction.

Suspicion of neurological dysfunction arises from the history and physical examination. The signalment, presenting chief complaint, time course of clinical signs, and history may suggest the type of disease process or species-specific disorder. A complete neurologic examination is necessary to localize the anatomic distribution, to determine the severity of the disease process, and to assess the prognosis for patient recovery.

A neurological examination is easily integrated into a routine physical examination. The objectives of the neurological examination are to confirm if there is a neurological abnormality and to specifically localize the abnormality within the nervous system. In conjunction with the history, signalment, presenting complaint and the physical examination, the neurological lesion

localization is a piece of a jigsaw essential to creating a list of differential diagnoses for the disease. However, caution must be used as some manipulations necessary for the neurological examination could exacerbate problems such as spinal cord disease.

### • Observation

Observation of the dog or cat is essential as it allows evaluation of the mentation, posture, attitude, and gait. Changes in mentation (level and content of consciousness) are revealed by a history of personality change, change in awareness of surroundings, and inappropriate behavioural responses. Consciousness is a function of the brainstem (responsible for arousal) and the cerebral cortex (responsible for content and regulation). The evaluation of the state of consciousness can classify the patient as depressed, demented or obtunded, delirious, stuporous and comatose.

### • Palpation

The musculoskeletal system should be palpated for asymmetry, masses, tenderness and tone. A mass, tenderness, or contour change requires further investigation. The vertebral column should be palpated for deviations and pain being cautious not to apply too much pressure if suspicious of an instability. Unilateral muscle mass loss or atrophy may indicate disuse if it is chronic, or a neurogenic loss if it is acute (within 7-10 days).

# • Cranial nerves

Cranial nerves have specific functions and evaluation of these functions can help to precisely locate a neurological lesion due to their well-documented anatomy. The general functions and specific tests are summarized in Table 1.

Simplistically, cranial nerve dysfunction may indicate a central nervous system (CNS) lesion (brainstem disease) or a peripheral lesion (affecting the cranial nerves after they have exited the

2

brainstem and course through the skull). Evaluation of the cranial nerves should follow observation and palpation, with particular attention paid to normal functions of eye movement, head movement, blinking, jaw and tongue movement and general symmetry of the head. Initially an ophthalmic exam should be performed, which will assist with the evaluation of the optic (CN II), oculomotor (CN III), trochelar (CN IV), and abducens (CN VI) nerves. The following tests are essential to the evaluation of cranial nerve function:

### <u>The menace response –</u>

- i. <u>How to perform</u> obscure the vision in one eye and make slow threatening hand gesture toward the other eye.
- ii. <u>How to interpret</u> this is a learned response, not a reflex, to a perceived threat, which evaluates CNs II and VII (responsible for innervation of the orbicularis oculi muscle which closes the eyelids), as well as the central visual pathways and the cerebellum. Normal function is demonstrated by a blink or retraction of the globe in response to the threat. To localize the lesion, other cranial nerve tests would be required.

### The pupillary light reflex –

i. <u>How to perform</u> – shine a bright light in each eye to evaluate the response of the pupil.

ii. <u>How to interpret</u> – this is a reflex. Light is sensed by CN II; parasympathetic fibers of CN III cause contraction of the iris muscle with direct and indirect simulation. The pupil is also innervated by sympathetic fibres responsible for dilation, which have their origin in the thalamus and send fibres down the cervical spinal cord to the T1-T3 spinal nerve roots, before they ascend up the neck and through the middle ear. A resting inequality in pupil size is termed anioscoria; to determine which pupil is abnormal, the animal should be evaluated in the light and dark. In the dark, a sympathetic lesion will mean the affected pupil will not be able to fully dilate. In the

light, a parasympathetic lesion will mean the affected pupil will not be able to fully constrict. Animals with sympathetic lesions will often demonstrate miosis in accompaniment to third eyelid protrusion and enophthalmus; a condition called Horner's syndrome.

# Evaluation of strabismus

- i. <u>How to perform</u> observe the animal's head in a normal position for a deviation of one or both globes in the orbit(s).
- <u>How to interpret</u> cranial nerves III, IV and VI aid vision by maintaining the globe in a central position. Deviation of the globe from its central axis indicates dysfunction in one or more of these nerves: ventrolateral CN III, dorsolateral CN IV, and medial CN VI.

# The palpebral reflex

- i. <u>How to perform</u> touch the medial canthus of the normal eyelid and watch response.
- ii. <u>How to interpret</u> the normal eyelid should close. Cranial nerve V (trigeminal nerve) is responsible for facial sensation, whereas the motor response to facial sensory stimulation is generally provided by the facial nerve (CN VII). Facial paresis presents as a drooping of the facial muscles, most notably the lips and the eyelids. It may also be detected as a reduction or absence in the blink response.

# Evaluation of jaw tone

- i. <u>How to perform</u> observe patient for a dropped lower jaw and / or an inability to eat. Assess the strength of the jaw safely by manually opening the mouth and evaluating the resistance to opening.
- <u>How to interpret</u> the mandibular branch of CN V provides motor function to the jaw. A dropped lower jaw or the inability to chew can indicate damage to CN V.

## The oculocephalic reflex / physiological nystagmus

- i. <u>How to perform</u> move the head from side to side in a horizontal plane and observe the resulting movement of the eyes.
- ii. <u>How to interpret</u> in normal animals, a physiological nystagmus will be induced, with the fast phase in the direction of head movement. This reflex tests the integrity of CN VIII (vestibulocochlear nerve), which is the sensory arm of this reflex, and CNs III, IV and VI, which are responsible for the motor movement of the eyes. Clinical signs of peripheral vestibular disease are manifest after damage to the inner ear or vestibular branch of CN VIII, which effectively gives unbalanced input to the intact central vestibular system. In the absence of head motion, spontaneous horizontal nystagmus is consistent with CN VIII damage, with the fast component away from the side of the lesion. Unilateral peripheral disease may cause a head tilt and circling to the side of the lesion.

# • Postural reactions

The postural reactions are complex, requiring intact sensory and motor pathways throughout the nervous system as well as unimpaired processing and integration in the brain. The complexity of the postural reactions allows detection of minor deficits in any key component of the pathway. Postural deficits are seen caudal to or at the level of the lesion. Additional testing must be performed to use the postural deficit to help localize the lesion within the pathway of the deficit.

i. <u>How to perform</u> – a leg is placed in an abnormal position and a correcting response by the animal is observed. Knuckling the toes over whilst supporting the body can be done to evaluate how long it takes for the animal to correct. Alternatively, a piece of paper may be placed under each foot and slowly moved sideways, to see if the animal returns its foot to the standing

position. Other postural reactions include wheelbarrowing, hopping, hemistanding and extensor postural thrust.

ii. <u>How to interpret</u> – conscious proprioception is the patient's awareness of limb position and movement without visual information. When the knuckling test is performed, an abnormality is indicated by a delay or absence of the response. The sensory branch of proprioception is carried from the skin, muscle and joints of the leg through the spinal cord and brainstem to the sensory motor cortex, where the brain responds by sending messages back to the lower motor neuron for motor function, resulting in a rapid correcting foot placement. Ascending sensory pathways are located in the outermost regions of the spinal cord and are very sensitive to compression. With minor spinal cord injury, proprioceptive deficits may be present because of disrupted sensory pathways, while motor function persists because the deeper motor tracts are unaffected. Both visual and tactile placing reactions require an intact motor cortex and intact motor pathways to the involved limb. A cortical lesion may produce deficits in the contralateral limb, whereas lower lesion produces deficits in the ipsilateral limb.

## Spinal reflexes

It is rare to have any reflex abnormalities if the animal has no evidence of gait abnormality, muscle mass loss or conscious proprioceptive deficits. In these cases, a complete reflex examination is unlikely to be helpful. Completion of a reflex requires an intact sensory nerve that provides transmission to the spinal cord and an intact motor nerve that elicits function from the innervated muscle. The reflex arc itself does not involve the brain or the remainder of the spinal cord. Lesions in the motor arm of the reflex arc, termed lower motor neuron (LMN), may cause a decreased or absent reflex (hyporeflexia or areflexia). An exaggerated response (hyperreflexia) results from an interruption in proximal motor pathways that modulate the reflex, termed upper motor neuron (UMN); however, stress or anxiety may cause an apparent increased reflex response, so it should not be considered too important without other evidence of neurological disease. Lower motor neuron signs indicate damage to one or more components of the reflex arc. Upper motor neuron signs indicate damage anywhere between the reflex arc and the brain (Table 2). The most reliable reflex is the flexor withdrawal in the thoracic and pelvic limbs. The other reflexes can appear to be present in small dogs just because the limbs will move when struck with a reflex hammer irrespective of reflex function.

# The anal sphincter reflex

<u>How to perform</u> – pinch the anal sphincter with haemostats and watch for a wink-like contraction of the external sphincter muscles and tail flexion.

<u>How to interpret</u> – this reflex reveals information regarding the pudendal nerve and caudal segments of the spinal cord. A flaccid unresponsive anus indicates LMN damage to the pudendal nerve or its spinal roots. A hypertonic, hyperresponsive anal sphincter indicates UMN damage at any point cranial to the pudendal nerve.

## The pedal flexor reflex

<u>How to perform</u> – apply a pinch stimulus to each foot and evaluate the response of the ipsilateral and contralateral limb.

<u>How to interpret</u> – this is a withdrawal reflex in which stimulation of sensory receptors in the toes elicits contraction of flexor muscle groups in the leg. Presence of a withdrawal reflex requires an intact sciatic nerve (sensory and motor) and an intact spinal segment at the

lumbosacral plexus, but does not require transmission along the spinal cord to the brain. Absence of the withdrawal reflex in the pelvic limb denotes extensive lower motor neuron damage involving the lumbosacral spinal cord segements (L6-S2) as well as the nerve roots and the lumbosacral plexus; in the thoracic limb it denotes damage to the cervical spinal cord segments (C6-T2), the spinal nerve roots and the brachial plexus.

### The patella reflex

<u>How to perform</u> – a tap stimulus should be applied to the straight patella tendon and the response of the limb should be evaluated. Reflex hammer size must be adapted to patient size for improved accuracy.

<u>How to interpret</u> – this is a myotactic (stretch) reflex that effectively stretches the quadriceps muscle. This stretch stimulates the femoral nerve (L4-L5), which generates muscular contraction to extend the stifle. Upper motor neuron lesions cause hyperreflexia and should be accompanied by weakness and poor weight bearing. Disease in the L4-L5 spinal cord segments or nerves causes hyporeflexia.

## **Cutaneous Sensation and Pain**

Cutaneous sensation testing provides information regarding the location and severity of a spinal cord or peripheral nerve lesion.

Evaluation of nociception (deep pain perception) is reserved for those animals showing evidence of spinal cord disease based on abnormalities in gait, proprioception and spinal reflexes. Lack of deep pain sensation is a poor prognostic factor as it indicates severe nervous system damage. Nociception requires cerebral perception of painful or injurious stimuli. It is important to remember that a withdrawal reflex is not an indicator of pain perception and may be elicited in an animal whose spinal cord has been transected cranial to the segment responsible for that reflex arc.

Hyperpathia is the sensation of pain produced by an innocuous stimulus such as palpating the vertebrae. All of the cervical and thoracolumbar vertebra should all be palpated to detect focal points of hyperpathia which may help localize the neurological lesion and will help with the differential diagnosis.

Cranial Nerve	Nerve Function	Applicable Tests	
I Olfactory	Smell	Blindfold the animal and monitor behavioural response to food placed near nose; loss of smell usually due	
		to nas	al disease rather than
		neuro	logical disease.
II Optic	Vision	i.	Menace response
		ii.	Pupillary light reflex
		iii.	Obstacle course
		iv.	Dropping cotton wool balls
			in front of each eye
III Oculomotor	Extrinsic and intrinsic	i.	Eyeball position
	ocular muscles / upper	ii.	Pupil size (mydriatic in
	eyelid muscle		disease)

 Table 1. Cranial Nerves: Function and Applicable Tests

		iii. Physiological nystagmus
		iv. Pupillary light reflex
IV Trochlear	Extrinsic ocular muscles	i. Eyeball position
V Trigeminal	Facial sensation / jaw	i. Palpebral reflex
	movement	ii. Jaw & masticatory muscle
		palpation
VI Abducens	Extrinsic ocular muscles	i. Eyeball position
		ii. Physiological nystagmus
VII Facial	Muscles of facial	i. Palpebral response
	expression /	ii. Evaluation of facial symmetry
	parasympathetic supply	ii. Shirmer tear test
	to lacrimal glands	
VIII	Hearing and balance	i. Oculocephalic reflexes
Vestibulocochlear		i. Assessment for head tilt
IX	Muscles of pharynx &	i. Gag reflex
Glossopharyngeal	larynx	
X Vagus	Muscles of larynx,&	i. Gag reflex
	pharynx	
XI Accessory	Superficial neck muscles	None applicable
XII Hypoglossal	Muscles of tongue	i. Tongue grab / inspection
N		-