OBJECTIVES
• To review the process of performing the neurologic examination in veterinary patients
• To understand what structures of the nervous system are being evaluated for each component of the neurologic exam

KEY POINTS
Performing a proper neurologic examination is essential in any patient presenting with signs of neurologic dysfunction. The neurologic examination is also key in providing the practitioner with information on the patient’s neurolocalization in order to guide the neurological workup.

OVERVIEW
The neurologic examination consists of 6 components. They are as follows:
• Mentation
• Posture and gait
• Cranial nerves
• Postural reactions
• Spinal Reflexes
• Pain

Mentation
A level of appropriate mentation means that the interaction between the ascending reticular activating system (ARAS) and the cerebral cortex is normal. The mental status should be evaluated in terms of the level and content of consciousness. The terms typically used to describe patients’ mentation are:
• Normal (alert and responsive)
• Obtunded (dull/depressed) – “drowsiness”; aware of environment but will responds only when encouraged to do so
• Stuporous – the patient only responds to noxious stimuli
• Comatose – the patient is unresponsive, even to noxious stimuli

Behavior and degree of mental status are not synonymous! Remember, the limbic system is responsible for the generation and modulation of behavior. For example, a patient may be bright, alert, and responsive in the exam room but could be agitate, aggressive, or more docile than normal. Therefore, clinical history is key to identifying a behavioral abnormality.

Posture and gait
Gait and posture refer to how a patient walks (or fails to walk), and how they stand, sit, and position their head. Observe the patient’s head, neck, spine, and tail for abnormalities in posture.

Posture
• Head tilt = rotation of the head along the horizontal plane (one ear is closer to the ground). Head tilts are often indicative of a problem affecting the vestibular system and are most commonly ipsilateral to the lesion. The exception is paradoxical vestibular disease.
• Head turn = rotation of the nose with the transverse plane; eyes remain parallel to the ground. A head turn is often indicative of a cerebral lesion and is ipsilateral to the lesion.
• Decerebrate rigidity = indicative of a lesion in the brainstem. Opisthontonus with extensor rigidity of all limbs. Patient is often stuporous to comatose.
• Decerebellate rigidity = acute cerebellar lesion. Opisthotonus with flexion of the pelvic limbs. Patient may be responsive.
• Schiff-Sherrington posture = seen with acute T3-L3 spinal cord lesions. Due to loss of inhibitory influences in cervical intumescence. Thoracic limb extensor rigidity in lateral recumbency with perseveration of thoracic limb postural reactions. The pelvic limbs will be paretic or plegic.
• Neck guarding = often indicated cervical pain

Other postures we can see include kyphosis (arching of the back), which may also indicate pain, and lordosis (ventral or inward curve of the spine). Scoliosis is a lateral curvature of the spine and is often due to congenital defects of the spinal column.

Observe how the patient is also carrying the tail. Is it low and dragging on the ground (cats in particular)? Does the patient move it voluntarily? You can also assess the tail tone when performing your rectal exam and taking the patient’s temperature.

Lastly, remember to look at the posture of the feet for a plantigrade (dropped hocks) or palmigrade (dropped carpi) posture as they may also indicate weakness.

**Gait**
When evaluation the gait, ask yourself 3 questions:

1. Is the gait normal or abnormal?
2. If the gait is abnormal, what limbs are affected?
3. What abnormality are you seeing?

The 3 main types of gait abnormalities seen are: ataxia, paresis, and lameness. Lameness is often due to musculoskeletal diseases, although we can see it due to some neuropathic (nerve and nerve root) diseases. Also, a short-strided gait can be confused with lameness, but could actually represent weakness (particularly lower motor neuron disease). Ataxia and paresis are most commonly seen with dysfunction of the nervous system.

**Ataxia**
When a patient is thought to be ataxic, there are three types of ataxia that can be seen:

- Proprioceptive – this type of ataxia is primarily related to spinal cord disease and can be differentiated from vestibular and cerebellar ataxia by lack of head involvement (no head tilt or tremors). Proprioceptive ataxia reflects a dysfunction
of the sensory tracts that carry unconscious proprioception and can indicate a lesion anywhere in the spinal cord or the brainstem.

- **Vestibular** – vestibular ataxia is the easiest to recognize. The hallmark of vestibular ataxia is asymmetry and often patients have a concurrent head tilt.
- **Cerebellar** – cerebellar ataxia is characterized by dysmetria (inability to control the rate and rhythm of movement), most often seen as hypermetria. Patients may also have intention tremors of the head and titubation (truncal swaying).

*Be aware that all three types of ataxia can be present in one patient!

**Paresis**
Paresis means partial loss of motor function, usually manifested as weakness. Paralysis (plegia) refers to complete loss of motor function. When assessing a patient with paresis/paralysis, additional prefixes such as tetra-, para-, hemi-, or mono- are used to indicated which limb(s) are involved. Remember, it is also important to distinguish between that is a patient is ambulatory or non-ambulatory. Additionally, using descriptive terminology such as barely, severely, moderately, mildly ambulatory can be helpful when reevaluating a patient (or reevaluating an associate’s patient you’ve have never seen before!).

- **Upper motor neuron paresis** – result of loss of UMN tracts within the spinal cord or brainstem resulting in long-striding in the affected limbs. *Think inability to generate movement
- **Lower motor neuron paresis** – occurs when LMNs at the cervical or lumbosacral intumescence are lost resulting in a short-strided and choppy gait. *Think inability to support weight.

**Cranial Nerves**
Most of the cranial nerve tests are reflexes and involve an afferent (sensory) nerve and an efferent (motor) nerve with a central pathway within the brainstem. Therefore, understanding the neuroanatomy of the cranial nerves can help determine if the patient has disease in just one or multiple cranial nerves.

- **Menace response**: The menace response is an exception to the reflex “rule” of the cranial nerves. The menace is a learned response (not seen in puppies and kittens until 4 months of age). The menacing gesture is made in the ipsilateral eye and the response carried in the optic nerve (CNII) and travels to the contralateral cerebral cortex, where the patient recognizes this menacing gesture. The response is the patient closing the eye; motor to the eyelid is mediated by the facial nerve (CN VII). But, the patient also moves their head away from this menacing gesture. Movement of the head away is coordinated by the cerebellum (ipsilateral).

- **Pupillary light reflex (PLR)**: tests the sensory (afferent) function of the optic nerve (CN II) and the parasympathetic function (efferent) of the oculomotor nerve (CN III). A light source is shone directly into one eye to observe maximal pupillary constriction (direct PLR); then observe the opposite eye to also ensure pupil constriction (indirect or consensual PLR). Be sure to use a good light source, which is often the cause for a poor PLR. Be sure to evaluate PLR in an ambient and dark environment.
- **Fundic exam:** We often will perform the fundic exam when testing the PLR.
- **Pupil size:** With anisocoria (pupils of unequal size), it is important to ask which eye is abnormal. The causes of anisocoria can be due to a primary ophthalmic lesion (commonly iris atrophy). Neurogenic causes include lesions in the optic nerve (CN II), oculomotor nerve (CN III) or sympathetic nerve (causing Horner’s).
- **Vestibulo-ocular reflex (occulocephalic reflex, Doll’s eye, physiologic nystagmus):** When moving the head or spinning a patient, the vestibular nerve (CN VIII) provides the afferent input. This response is processed centrally in the brainstem (via the vestibular nerve cell body and medial longitudinal fasciculus) and cerebellum (floculonodular lobe). Motor input to the eye is mediated by the oculomotor (CN III), trochlear (CN IV) and abducens (CN VI) nerves. Following the VOR, the head is often tilted upward to look for a positional ventral strabismus, which can be indicative of vestibular dysfunction.
- **Palpebral reflex:** This test is performed by touching the medial and lateral aspects of the eye as well as the base of the ear. The sensory (afferent) function is mediated by the trigeminal nerve (CN V) and the motor (efferent) function (closure of the eyelid) is mediated by the facial nerve (CN VII).
- **Facial symmetry** evaluates the function of the facial nerves (CN VII). Stand back from the patient and observe their face for any asymmetry such as drooping of an upper lip, eyelid, or ear.
- Palpation of the **muscles of mastication** for any asymmetry evaluates the motor function of the trigeminal nerve (CN V).
- **Nasal sensation:** Evaluates the integrity of the sensory components of the trigeminal nerve (CN V; maxillary and ophthalmic branches) and the ability of the cerebrum to respond to noxious stimuli. This test is performed by covering the patient’s eyes and gently probing the nasal philtrum either with a hemostat or cotton tipped applicator.
- **Gag reflex:** Evaluates the integrity of the glossopharyngeal (CN IX) and vagus nerve (CN X). Additionally, the accessory nerve (CN XI) provides some motor to this region. This test is often performed in patients with a history of dysphagia, dysphonia, or stertorous breathing.
- The tongue is evaluated for any asymmetry/atrophy as well as normal movement (CN XII; hypoglossal nerve).

**Postural reactions**
Postural reaction testing evaluates proprioception, cerebrocortical integration, and motor system responses. The purpose of these tests is to detect subtle deficits or asymmetries that may not be noted during the gait assessment. The two most commonly used tests are proprioceptive positioning and hopping.

**Proprioceptive positioning** (placing, conscious proprioception) is performed by supporting the animal’s weight and placing the paw on its dorsum. Normal animals will almost immediately correct the paw. *If an animal pulls the paw away (foot shy), this DOES NOT mean they have normal placing!*

**Hopping** is performed by placing the entire weight of the animal one limb and pushing the patient in the direction of the limb being tested. When performing hopping, we evaluate the speed, coordination, and strength of the response.
Other less commonly used tests include visual and tactile placing, wheelbarrow, and extensor postural thrust.

Spinal Reflexes
Spinal reflexes test the integrity of the sensory and motor components of the reflex arc. In the pelvic limbs, I tend to assess the patellar and the withdrawal reflex and only perform the withdrawal reflex in the thoracic limb. The reason for this is that the other reflexes (cranial tibial, cranial tibial, gastrocnemius, extensor carpi radialis, biceps, and triceps) may not be present in a normal dog.

*Pelvic limbs:* The **patellar reflex** is a monosynaptic reflex mediated by the femoral nerve (L4-L6). The **withdrawal reflex** (flexor) is polysynaptic and is mostly mediated by the sciatic nerve (L7-S1). Additionally, when I am evaluating the pelvic limb reflexes, I will also perform the **perineal reflex**. This reflex is mediated by the pudendal nerve (S1-S3) and is performed by stimulating the perineum with either a cotton-tipped applicator or hemostats.

*Thoracic limbs:* The **withdrawal reflex** in the thoracic limbs is mediated mostly by the musculocutaneous nerve (C6-C8) but if a patient has a decreased withdrawal reflex, we will generally localize them to a C6-T2 myelopathy.

The **cutaneous trunci** in a polysynaptic reflex with afferents mediated via the fasciculus proprius in the spinal cord and its efferent projections in the lateral thoracic nerve (C8-T1).

Pain
*Paraspinal palpation* is performed to detect any focal or diffuse spinal pain. Initially, light pressure is applied first followed with more firm pressure on subsequent palpation. When palpating the thoracolumbar spine, gentle pressure is applied adjacent to the dorsal spinous processes. When palpating the cervical spine, gentle pressure is applied ventrally to the transverse processes. Manipulation of the cervical spine (extension, flexion, excessively turning the head) is NOT RECOMMENDED, especially in patients where there is concern for instability (AA lux, Wobbler’s, etc).

Nociception: Testing the presence or absence of deep pain perception (nociception) is ONLY performed in those patients suspected to be plegic. This is performed by using hemostats on the metatarsals or nail bed and clamping them. **It is important to not confuse the withdrawal reflex with the presence of deep pain.** As such, the patient is monitored for a reaction (whining, trying to bite, moving away, turning head, pupil dilation, etc).

REFERENCES:
