Introduction
The neurologic exam is an essential component of the physical examination for patients suspected of having a neurologic disorder and is a tool to help confirm and localize nervous system problems.

Evaluating all components of the neurologic exam will help answer the following questions:
1) Does the patient have a neurologic problem?
2) Where in the nervous system is the problem located?
3) What diseases could be causing the problem?
4) What tests should be performed?

Often times, a neurologic exam is not performed because it can be intimidating for those not accustomed to performing it regularly or those who are not comfortable with the anatomical organization of the nervous system. However, with a general understanding of the components of the nervous system and how they work together, performing a neurologic exam will become easier with practice. The goal of this lecture is to review the parts of the neurologic exam.

Neurologic Exam
The neurologic exam starts as soon as the veterinarian meets the patient. There are several components to the neurologic exam which should be tested in the same order each time to enable the clinician to quickly go through the steps without skipping major parts of the exam. The parts of the neurologic exam include: History, Mentation/Behavior, Cranial Nerves, Posture/Gait/Involuntary Movements, Postural Reactions, Spinal Reflexes, Spinal Pain, and Sensory/Pain Perception.

1. History
Obtaining a thorough history is the first component of the neurologic exam. It can be the most important part of the neurologic exam because it may provide clues about the diagnosis. When dealing with a neurologic problem, the history should include the following: signalment, sign-time graph, neurologic signs observed, painful vs. nonpainful, and specific questions.
   A. Signalment: While the signalment will not give a diagnosis, it may shed light on the problem as some diseases are more prevalent in certain groups of animals.
   B. Sign-Time Graph: A sign-time graph plots the severity of clinical signs (vertical axis) against time (horizontal axis) to evaluate the course of the disease. This provides useful information about the cause of the neurologic signs. The following questions should be answered during the history in order to construct a sign-time graph:
i. What was the onset (acute, chronic)?
ii. What is the progression (static, progressive)?

**Sign-Time Graph**

C. Neurologic Signs: The description of the neurologic signs is also an important part of the history because it helps determine if the patient is exhibiting signs that are more likely to be associated with a problem in the nervous system.

D. Painful vs. Nonpainful: This information will help determine potential diseases.
   a) Painful: disk herniation/protrusion, neoplasia, inflammatory disease, diskospondylitis, trauma
   b) Nonpainful: degenerative, neoplasia, vascular, congenital

E. Specific Questions
   a) Behavior Changes: Determine if the patient has had any behavior changes that are usually seen with a neurologic problem (pacing, circling, wandering aimlessly, getting stuck in corners, urinating or defecating in the house).
   b) Triggers/Patterns: Determine if there are any triggers or patterns associated with the neurologic signs that may help decide if the problem is neurologic or not (ie. associated with activity or eating, signs are waxing/waning).

2. Mentation/Behavior
While obtaining a history from the client, the patient’s mentation and behavior can be assessed as the patient is allowed to walk around the exam room. You should observe how the patient interacts with the owner and the environment - does it respond as you would expect to respond? Abnormal mentation can be described as dull or depressed, disoriented, stuporous, or comatose.
**Definitions**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
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<tr>
<td>Depressed</td>
<td>Lethargy with decreased activity but normal mental status</td>
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<tr>
<td>Obtunded</td>
<td>Dull mentation with decreased consciousness and response to mild stimulus</td>
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<tr>
<td>Stupor</td>
<td>Decreased consciousness and response only to noxious stimulus</td>
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<tr>
<td>Coma</td>
<td>Unconscious and no response to noxious stimulus</td>
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**3. Cranial Nerves**

Cranial nerve testing evaluates the brainstem and cranial nerves. While there are 12 cranial nerves, the most clinically important cranial nerves (CN) are the optic (II), oculomotor (III), trigeminal (V), facial (VII), vestibulocochlear (VIII), hypoglossal (IX), vagus (X) and hypoglossal (XII). In general, it is easier to remember the tests that evaluate these nerves rather than list all of them.

<table>
<thead>
<tr>
<th>Test</th>
<th>Afferent CN</th>
<th>Efferent CN</th>
<th>Response</th>
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<tbody>
<tr>
<td>Palpebral reflex</td>
<td>5</td>
<td>7</td>
<td>Blink eye when medial or lateral canthus touched</td>
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<tr>
<td>Nasal sensation</td>
<td>5</td>
<td>11</td>
<td>Withdraw head by touching nasal mucosa</td>
</tr>
<tr>
<td>Lip curl</td>
<td>5</td>
<td>7</td>
<td>Pull back lip by pinching lip</td>
</tr>
<tr>
<td>Corneal sensation</td>
<td>5</td>
<td>6, 7</td>
<td>Retract globe and blink when cornea touched</td>
</tr>
<tr>
<td>Menace response</td>
<td>2</td>
<td>7</td>
<td>Blink eye with a menacing gesture</td>
</tr>
<tr>
<td>Vestibulo-ocular reflex</td>
<td>8</td>
<td>3, 4, 6</td>
<td>Physiologic nystagmus induced by turning head</td>
</tr>
<tr>
<td>Pupillary light reflex</td>
<td>2</td>
<td>3</td>
<td>Pupil constricts by shining light in eye</td>
</tr>
<tr>
<td>Gag reflex</td>
<td>9, 10</td>
<td>9, 10</td>
<td>Contraction of pharynx elicited by palpation</td>
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- **Facial sensation (CN 5):** The trigeminal nerve is responsible for facial sensation, and all branches (eyelids, lips, jaw) should be evaluated for response to touch (Nasal Stimulation). This sensation is separate from facial expressive muscle evaluation (CN 7).
- **Facial symmetry (CN 5, 7):** The trigeminal nerve’s maxillary branch innervates the muscles of mastication. Injury to this nerve can result in severe atrophy of the temporals and masseter muscles or cause a “dropped jaw” from inability to close the mouth. The facial nerve innervates muscles of facial expression, and injury to it can result in drooping of the lip or eyelid on the affected side.
- **Facial expression (CN 7):** The facial nerve innervates the muscles that move the muzzle and eyelids. Pinching the lip of the muzzle near the canine tooth should produce a “snarl” response.
(Lip Curl). Touching the lateral and medial canthus of the eye should result in a blink (Palpebral Reflex).

- **Menace response (CN 2, 7):** Not a true reflex but a learned response. This test evaluates an animal’s ability to respond (see and blink) to a menacing or threatening gesture. It tests the optic nerve, cerebrum, cerebellum, and facial nerve. It may not be present in animals less than 16 weeks old.

- **Pupillary light reflex (CN 2, 3):** Light is shined in one eye and pupillary constriction is assessed in both eyes. It evaluates the retina, optic nerve, brainstem pathways, and parasympathetic fibers of the oculomotor nerve.

- **Ocular position (3, 4, 6, 8):** Strabismus is when the eyes do not point in the same direction. It occurs when cranial nerves 3, 4, 6, or 8 have a lesion resulting in a change of eye position. Loss of the oculomotor nerve input to skeletal muscles of the eye causes ventrolateral strabismus. The pupil will appear to look down and out to the side of the head (“down and out”). Loss of the trochlear nerve to the dorsal oblique muscle of the eye causes a dorsomedial rotation of the pupil that is difficult to see in dogs (round pupils). This is a rare problem but can be noted in cats (oblong pupil) or with retinal vessel examination in dogs. The pupil will appear to rotate so the top is turned away from the bridge of the nose. Loss of abducent innervation to the lateral rectus muscle can cause medial deviation of the eye. This will make a patient look “cross-eyed.” Loss of vestibular input to CN 3, 4, or 6 can result in a ventrolateral strabismus.

- **Ocular movement (CN 3, 4, 6, 8):** Nystagmus is involuntary, rhythmic, oscillations of the eyes that occurs when the head is rotated in order to keep the eyes focused on the visual field. Nystagmus can either physiologic or pathologic. Physiological nystagmus can be observed when turning the head from side to side (known as the vestibulo-ocular reflex). The pupils will appear to flicker, and the fast phase should be in the direction of the head turn. Any loss of this normal movement should be noted. Pathologic nystagmus occurs with a lesion in the vestibular system. Pathologic nystagmus can be either spontaneous or positional. Spontaneous nystagmus occurs when the head is at a normal postural position. Positional nystagmus occurs when the animal is placed in an abnormal position, such being placed on its back or elevating the head. Vertical (pupils move up and down) implies a central nervous system location of the vestibular lesion. Horizontal (pupils move side to side) and rotary may be seen with peripheral or central vestibular disease.

- **Gag reflex (9, 10, 12):** A finger or tongue depressor is placed at the base of the tongue/throat and the animal is evaluated for the ability to gag/swallow (vagus and glossopharyngeal nerves). Most animals will lick after the gag reflex which allow the symmetry of the tongue and the ability to move it (hypoglossal nerve) to be evaluated. Use caution in animals with potential for rabies exposure or questionable vaccination history. Dysphagia, regurgitation, a voice change, or inspiratory stridor may also indicate a CN 9,10 lesion.

**4. Posture/Gait/Involuntary Abnormal Movement**

Gait abnormalities are one of the most common reasons why clients bring in their pet for evaluation. A normal gait requires an intact brainstem, cerebellum, spinal cord, and sensory/motor nerves. The cerebrum is not a major contributor to gait in dogs and cats. The animal’s posture and gait can be assessed while the patient is walking into the exam room or by taking it
outside before the animal is handled physically. The animal’s gait is observed walking in a straight line, from the side, turning in circles, and walking up and down steps. The goal of gait evaluation is to determine if the animal is ataxic (abnormality in coordination), paretic (abnormality in strength of movements), or lame (orthopedic or peripheral nerve problem). Other abnormalities that may be detected include a heat tilt, head turn, wide based stance, body lean, circling, spinal curvature, tremors and myoclonus.

Ataxia is defined as an uncoordinated gait. When an animal is ataxic, it is important to classify the ataxia as sensory, cerebellar, or vestibular to help determine what part of the nervous system is affected. Sensory ataxia is caused by a lesion affecting the long tracts in the cerebrum, brainstem, spinal cord, or the sensory part of the peripheral nerve resulting in conscious proprioceptive deficits (crossing the feet and scuffing the toes). Cerebellar ataxia is caused by a lesion in the cerebellum resulting in unconscious proprioceptive deficits (wide-based stance, intention tremor, dysmetric gait - hypometria, hypermetria). Vestibular ataxia is caused by a lesion in the vestibular system resulting in vestibular signs (leaning, falling, head tilt, nystagmus).

Paresis is defined as weakness (voluntary movement is still present) which can be due to an inability to generate a gait (CNS paresis- cerebrum, brainstem, spinal cord) or an inability to support weight (Neuromuscular paresis - peripheral nerve, neuromuscular junction, muscle). Plegia refers to complete loss of voluntary motor (paralysis). Paresis and plegia can be described as affecting one limb (mono), limbs on one side (hemi), all limbs (tetra), or both pelvic limbs (para). CNS paresis usually causes stiff and spastic movements. Neuromuscular paresis causes difficulty in weight bearing which can range from a short, choppy gait to complete inability to support weight. Muscle tremors can also be seen when the animal is standing.

Lameness typically indicates limb pain and is most often associated with an orthopedic problem but can be seen with a neurologic problem. With an orthopedic problem, the animal will bear less weight on the limb or may not bear any weight on the limb, but it has motor function in limb. The stride of the lame limb is usually shortened. In contrast, when a neurologic problem is causing the lameness there may also be proprioceptive deficits, and the animal may drag the limb or scuff the nails. However, the exception to this rule is the sign known as a "root signature". This sign is caused when a spinal nerve is compressed, entrapped, or inflamed and results in referred pain down the limb. The animal will carry the limb in flexion and appear to have a lameness. A lateralized disk herniation and a nerve sheath tumor are the two most common causes of “root signature”. A thorough orthopedic exam and neurologic exam are necessary to help differential between the two. With “root signature”, animals often have spinal pain and/or reflex changes in the limb.

Circling is another abnormality that can be seen when evaluating gait. Circling can be caused by a lesion in the vestibular system or a focal lesion in the forebrain. Tight circles are usually associated with a vestibular lesion, while wide circles are often associated with a forebrain
lesion. Animals will usually circle towards the side of the lesion (ex. right forebrain lesion will cause circling to the right).

Involuntary abnormal movements can be seen when the patient is at rest or moving and can be intermittent or continuous. Frequently recognized movement disorders include myoclonus and tremors. Myoclonus is a coarse, jerking, shock-like contraction a muscle group and is associated with canine distemper encephalomyelitis. Tremors are caused by alternating contractions of opposite groups of muscles. Tremors can be caused by both neurologic (hypomyelination, dysmyelination, steroid responsive tremor syndrome, toxicities) and non-neurologic problems (fear, chilling, fatigue, drug reactions, primary muscle disease). An intention tremor (also called an action tremor) is the most common neurologic tremor and is caused by a cerebellar disease. The tremor is produced when the initiates a movement and is not present at rest.

Definitions

<table>
<thead>
<tr>
<th>Plegia</th>
<th>Complete absence of motor function</th>
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<tr>
<td>Paresis</td>
<td>Motor weakness (mono: one limb; hemi: ipsilateral thoracic and pelvic limbs; para: both pelvic limbs; tetra: all 4 limbs)</td>
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<td>Ataxia</td>
<td>Loss of coordination</td>
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<tr>
<td>Hypermertia</td>
<td>Gait abnormality seen with cerebellar disease (abnormal length of movement); “Goose-stepping”</td>
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<tr>
<td>Opisthönus</td>
<td>Posture dominated by increased extensor tone of the limbs and extension of neck</td>
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<tr>
<td>Kyphosis/Lordosis</td>
<td>Dorsal deviation of the spine/Ventral deviation of the spine</td>
</tr>
<tr>
<td>Scoliosis</td>
<td>Lateral curvature of the spine</td>
</tr>
<tr>
<td>Torticollis</td>
<td>Neck twisted or turned to one side</td>
</tr>
<tr>
<td>Decerebrate rigidity</td>
<td>Extensor rigidity of all limbs; secondary to severe cortical/midbrain lesion; patient usually comatose</td>
</tr>
<tr>
<td>Decerebellate rigidity</td>
<td>Extensor rigidity of the thoracic limbs with flexion of pelvic limbs and dorsal extension of the head; associated with acute cerebellar lesions including herniation</td>
</tr>
<tr>
<td>Schiff-Sherrington</td>
<td>Extension of thoracic limbs with paralysis of pelvic limbs; caused by acute thoracic or lumbar spinal cord lesions (T3-L3 segments)</td>
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5. Postural Reactions

Postural reactions are normal responses that help an animal maintain its upright position when weight is shifted among the limbs. The main value of testing postural reactions is that they can detect subtle deficits that may not be detectable when observing the gait. Postural reactions involve both sensory and motor pathways in several parts of the nervous system (peripheral nerves, spinal cord, brainstem, and cerebrum). Therefore, while they are a sensitive indicator of neurologic dysfunction, postural reactions do not localize a lesion to one part of the nervous system. However, it is uncommon to see postural reaction deficits with a peripheral nerve lesions. Peripheral nerve diseases usually need to be quite severe to cause postural reaction deficits (ie. peripheral nerve avulsion). Postural reactions deficits are most commonly seen with a central nervous system lesion. Postural reaction deficits are ipsilateral to peripheral nerve, spinal cord, and most brainstem lesions. Postural reaction deficits are contralateral with a cerebral lesion.

The following postural reactions are routinely tested: Proprioceptive Positioning (conscious proprioception), Hopping Reaction, Wheelbarrowing, Extensor Postural Thrust, Hemiwalking, and Placing Reaction.

Postural reactions are also helpful in distinguishing between a neurologic and orthopedic problems. Postural reactions are intact with an orthopedic problem. If an animal has severe orthopedic disease, its weight must be supported adequately while testing postural reactions.

6. Spinal Reflexes

A spinal reflex requires an intact sensory neuron, motor neuron, and varying number of interneurons within the spinal cord. They evaluate peripheral nerve function and local spinal cord segments. Although spinal reflexes are influenced by the cerebrum and brainstem, they are needed to generate a reflex. Reflexes do not involve consciousness and can be elicited even if the region of the spinal cord responsible for its presence is completely isolated from the brain.

Segmental spinal reflexes are graded on a scale of 0–4.
- 0 is used to describe an absent reflex (Areflexia)
- 1 indicates a reflex that is present but markedly decreased (Hyporeflexia)
- 2 indicates a normal reflex (Intact/Normal)
- 3 indicates an increased or exaggerated reflex (Hyperreflexia)
- 4 indicates clonic activity (continual repetitive response to a mild stimulus)

Thoracic limb (C6-T2 Spinal Cord Segments):
- Biceps reflex: Evaluates the musculocutaneous nerve (C6-C8)
- Triceps reflex: Evaluates the radial nerve (C7-T2)
- Extensor Carpi Radialis reflex: Evaluates the radial nerve (C7-T2)
- Flexor withdrawal: Evaluates multiple nerves - musculocutaneous, axillary, radial, median, ulnar nerves (C6-T2)
Pelvic limb (L4-S2 Spinal Cord Segments)

- Patellar reflex: Evaluates the femoral nerve (L4-6)
- Cranial tibial reflex: Evaluates the peroneal nerve (L6-S1)
- Gastrocnemius reflex: Evaluates the tibial nerve (L6-S2)
- Flexor withdrawal: Evaluates the sciatic nerve (L6-S2)

The patellar and withdrawal reflexes are the most reliable reflexes in the limbs. Other reflexes evaluated include the perineal reflex and cutaneous trunci reflex (panniculus). The perineal reflex involves stimulation of the anus or perianal region and evaluates the pudendal and perineal nerves as well as the S1-3 spinal cord segments. The normal response is contraction of the anal sphincter and flexion of the tail. Anal sphincter tone can also be assessed while taking a patient’s rectal temperature. The cutaneous trunci reflex starts over the lumbosacral region and proceeds cranially. It involves pinching the skin on both sides of the paralumbar and parathoracic region (T1-L4) with fingernails or hemostats to elicit a bilateral skin twitch. The cutaneous trunci reflex evaluates the spinal cord, the C8-T1 spinal cord segments, and the ipsilateral lateral thoracic nerve. A cutoff point along the spine suggests a spinal cord lesion 1-4 spinal cord segments cranial to the cutoff level. Note that the cutaneous trunci reflex is absent in the neck and sacral regions.

7. Spinal Pain
Hyperesthesia is a painful response to an innocuous stimulus. This is elicited by lightly and deeply palpating the paraspinal muscles, spinous processes, articular processes, transverse processes, and ribs. All region of the spine should be evaluated including the tail base. Some animals will tense their abdomen when they have spinal pain, so one hand should be lightly placed on the abdomen to detect tensing of the abdominal muscles as the spine is palpated. The cervical and lumbosacral spine can also be flexed and extended to check for pain. Compressive, inflammatory/infectious, or neoplastic spinal lesions can cause spinel pain by stimulating sensory nerve endings in the bone, meninges, nerve roots or annulus fibrosis. Spinal palpation is used to evaluate conscious awareness of sensation.

8. Pain Perception
Superficial and deep pain of the limbs can be assessed by pinching the toes (superficial sensation) or the pad/bone (deep sensation) and obtaining a conscious response. It is important to recognize that the animal may flex or pull away (withdraw) the limb because the withdrawal reflex is stimulated. This does not indicate deep pain sensation. Conscious recognition of deep pain includes turning the head, vocalizing, or showing pupillary dilation and elevated heart rate in animals that cannot turn or vocalize. Severe spinal cord lesions may cause loss of deep pain sensation caudal to the site of the lesion. Absence of deep pain is a grave prognostic sign because of these pathways are more resistant to damage than other pathways and the fact that these pathways are diffuse and bilateral within the spinal cord.

References available upon request.