The Neuro Exam: An introduction

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What’s the point?
The purpose of our neurologic examination is to help us answer two major questions:
1) Is there a neurologic issue?
2) If so, what part of the nervous system is being affected? (i.e. localization)

But remember- some neurologic signs will only occur intermittently and not be apparent during a neurologic exam. These include seizures or seizure-like (paroxysmal) episodes, some neuromuscular signs, and events related to physiologic changes that may not always be present, like low blood glucose or high blood ammonia. **Just because your neurologic exam is normal does not mean that your patient is neurologically normal!**

A good history
So before you even begin your neuro exam, a very good history is important. If there are “episodes” occurring, some of these questions are helpful:
1) What does a typical episode look like?
2) Have any of the episodes looked different than that?
3) How many episodes have occurred total?
4) How far apart were the episodes?
5) Did any episodes occur in clusters (> 1 in 24 hours)
6) What is your pet doing when an episode begins?
7) Is there a time of day where the episodes tend to occur?
8) Do the episodes occur before or close after eating?
9) Do you think your pet is mentally normal during the episode?
10) Can you interrupt an episode (make it stop) by feeding/petting?
11) Can you think of anything your pet has eaten near the time of an episode that is not a normal pet food/treat? Can you think of any potentially toxic substances that your pet could have been exposed to either directly or indirectly inside or outside? Are there any oral or topical medications in the home that your pet could’ve been exposed to?

How do these questions help? Some of these questions can help you determine if a pet is likely experiencing seizures. Descriptions of altered mentation, tonic-clonic movements of the face/jaw or limbs, episodes that tend to begin when the pet is at rest, and episodes that look very similar to one another (“stereotyped”) leads you to think seizures are more likely. Episodes that tend to occur shortly after eating could indicate hepatic encephalopathy, often from a liver
shunt, or an insulinoma. Signalment helps you from there! Insulinomas may also cause neurologic episodes if a pet isn’t eating frequently enough. Syncopal episodes are more likely associated with excitement or exercise and tend to cause the pet to become flaccid rather than having increased muscle tone. Cataplexy episodes can be triggered during eating or other excitement, and cause a pet to become flaccid while they are otherwise still awake/aware.

The neuro exam: the overview

1) Mentation
   a. Appropriate? Aware? Active?

2) Posture/Gait
   a. Head/neck/body tilt or turn?
   b. Abnormal head/body/limb carriage?
   c. Is there ataxia present?
   d. Is there paresis present?
   e. How many of the limbs are affected?

3) Postural reactions
   a. How many limbs are abnormal and which limbs are the most affected?

4) Cranial nerve exam
   a. CN II (Optic n.)
   b. CN III (Occulomotor n.)
   c. CN IV (Trochlear n.)
   d. CN V (Trigeminal n., ophthalmic, maxillary and mandibular branches)
   e. CN VI (Abducens n.)
   f. CN VII (Facial n.)
   g. CN VIII (Vestibulocochlear n.)
   h. CN IX (Glossopharyngeal n.)
   i. CN X (Vagus n.)
   j. CN XI (Accessory n.)
   k. CN XII (Hypoglossal n.)

5) Spinal reflexes
   a. Thoracic limb
      i. Triceps, biceps and withdrawal
   b. Pelvic limb
      i. Patellar, gastrocnemius, withdrawal
   c. Back
      i. Cutaneous trunci
   d. Perineal region
      i. Perineal, tail clamp

6) Palpation
   a. Muscle mass/tone
   b. Pain
      i. Muscles of limbs/trunk
      ii. Paravertebral
      iii. Skull
iv. Jaw

**How do abnormalities on the neurologic exam help me localize?**
Our goal is to find a **SINGLE** lesion that can explain any neurologic abnormalities, if possible. But first, we have to determine where neurologic deficits **COULD** localize to- then we can narrow it down.

**The neuro exam: What localizes where?**

**Mentation: Appropriate? Aware? Active?**

Ask yourself these three questions about the patient (and ask the owners, too). Are they acting mentally appropriate for the situation? Hyper-reactivity, inappropriate vocalization or reactions to stimuli, or abnormal behaviors at the hospital or at home may mean an **INappropriate** mentation. Is your patient aware of the environment, or do you get the feeling they’re “checked out” to some degree. Is your patient actively interested in the environment, or do they seem very subdued/non-interactive?

If your patient displays inappropriateness, decreased awareness or decreased mental activity as you observe them or based on what an owner reports, we would consider that an alteration of mentation. Altered mentation = brain. We can be even more specific, though. In appropriate mentation implies a problem in the conscious parts of the brain that take in, interpret and react to the outside world. Therefore, inappropriate mentation = forebrain (cerebral hemispheres and thalamic structures). However, an abnormal mentation that is simply less active/aware in the environment could be related to either the forebrain or the brainstem, due to the reticular activating system that runs through the brainstem to the thalamus and is responsible for “waking up” the cerebral hemispheres. **Note: The cerebellum is NOT involved in mentation.**

What’s the terminology? It varies a little, but I often describe an inappropriate mentation (i.e. vocalizing constantly), and attach a label to levels of diminished awareness/mental activity:

1) **Obtunded-** Diminished mentation. Very wide range, and includes non-neurologic conditions (i.e. obtundation because you are ill and have a fever).

2) **Stuporous (Semi-coma)-** The patient is sleeping/will not respond to stimuli except a noxious stimulus.

3) **Comatose-** The patient is non-responsive/unrousable via any stimuli, including noxious.

**Posture/Gait:**

1) **Head tilt vs. head turn:** Deviation of the axis of the head.
   a. Head tilt = ears no longer on the same level.
      i. **Vestibular system!**
         1. Peripheral = middle/inner ear, or vestibular n.
             a. Tilt toward the side of the lesion
         2. Central = nuclei of vestibular n. (brainstem), small part of cerebellum
             a. Tilt usually toward side of the lesion
b. Head turn = nose deviated from midline.
   i. Forebrain, usually
      1. Turn toward the side of the lesion

2) Abnormal limb position
   a. Are the limbs bearing weight normally? Are all of the joints extended normally?
      i. Over-flexion of the hock most frequent
         1. Sciatic nerve distribution
      ii. Overt standing on dorsum of paw
         1. Usually proprioceptive dysfunction
         2. In pelvic limb, can be fibular n. deficit (br. of femoral n.)

3) Is there ataxia present?
   a. Proprioceptive ataxia
      i. Incoordination caused by loss of understanding of where the limbs are in space. When steps are taken, the limb/paw is often placed in an abnormal position. This leads to a “wobbly” gait where steps may be intermittently too narrow, too wide, or even crossing over midline.
         1. Proprioceptive system extends from specialized receptors in the limbs/trunk/neck, up the spinal cord, through the brainstem and into the cerebellum and forebrain
            a. Localization: **Anywhere- BUT most frequent:**
               i. Brainstem
               ii. Spinal cord
   b. Cerebellar ataxia
      i. Incoordination caused by loss of normal rate/range of movements
         1. Hypermetria/dysmetria most common
            a. Cerebellar localization
   c. Vestibular ataxia
      i. Incoordination caused by loss of balance
         1. Leaning/falling, very off-balance
            a. Peripheral or central vestibular systems

4) Is there paresis present?
   a. Upper motor neuron paresis (“spastic paresis”)
      i. The upper motor neurons exist in the forebrain/brainstem and tell the lower motor neurons in the spinal cord how to function in order to generate a gait
      ii. Slow communication between upper motor neurons and lower motor neurons leads to a delay in generation of the gait
         1. Delayed “swing” phase of gait
         2. Often prolonged movement before completion of step
            a. Leads to a “long and slow” appearance to the limb movements
            b. As there is more and more upper motor neuron dysfunction, paresis will increase
Gradual loss of motor function, less and less ability to generate a limb movement

3. Localization is ANY SEGMENT cranial to the affected limbs because the communication pathway is disrupted
   a. However, in our patients most of the upper motor neurons that generate gait exist in the brainstem
      i. Brainstem localization
      ii. Spinal cord localization

b. Lower motor neuron paresis (“flaccid paresis”)
   i. The lower motor neuron includes the direct limb innervation from the spinal cord, the nerves that innervate the limb, the neuromuscular junction and the muscles being innervated. These are recruited by the upper motor neurons in order to generate the gait.
   ii. Lower motor neurons dysfunction usually changes the actual ability for the limb to support weight, maintain tone and perform movements.
      1. Over-flexed joints in limb = decreased weight bearing/tone
      2. Shaking of limbs while bearing weight
      3. Shorter, choppier steps = less ability to support weight/perform movement of limb
         a. As there is greater lower motor neuron dysfunction, there will be less ability to bear weight or move limbs
            i. Localization MUST INCLUDE the lower motor neuron system to that limb
               1. Spinal cord nuclei of spinal nerves
               2. Spinal nerves innervating limbs
               3. Neuromuscular junction
               4. Muscles of the limbs

Postural reactions:
1) Tests of the brain’s ability to understand and correct a limb that is out of position
   a. Multitude of tests that can be performed
   b. Testing “conscious” and “unconscious” proprioception
   c. Sensitive but not SPECIFIC tests
      i. Localization can be ANYWHERE cranial to the abnormal limbs = disrupted communication of proprioceptive tracts
      ii. Localization is to the same side of the dysfunctional limb all the way cranial UNTIL the forebrain, which has contralateral control

Cranial nerve exam:
1) The cranial nerves (CN) originate from the brain in order, so dysfunction of them localizes either to the nerve itself or the nucleus within the brain, extending rostral to caudal
   a. CN II (optic) = thalamus
   b. CN III (occulomotor), IV (trochlear) = midbrain
c. CN V (trigeminal) = pons
d. CN VI (abducens), VII (facial), VIII (vestibulocochlear), IX (glossopharyngeal), X (vagus), XI (accessory), XII (hypoglossal) = medulla oblongata

2) Tests we do: **Note: “response” indicates there is some cerebral cortex involvement, which will be contralateral; otherwise CN dysfunction is to the same side of the lesion**

a. Palpebral reflex
   i. CN V (trigeminal) for sensory
   ii. CN VII (facial) for blink
   iii. CN VI (abducens) for eye retraction in most dogs if they can’t blink

b. Menace response
   i. CN II (optic) for vision
   ii. Other areas of cerebrum for vision
   iii. CN VII (facial) for blink
   iv. CN VI (abducens) for eye retraction in most dogs that can’t blink

c. Pupillary light reflex
   i. CN II (optic) for vision
   ii. CN III (occulomotor) for pupil constriction

d. Facial sensation responses
   i. CN V (trigeminal)
      1. Medial canthus, cornea, +/- inside nares = ophthalmic branch
      2. Lateral canthus and maxillary area = maxillary branch
      3. Mandibular area = mandibular branch

e. Physiologic nystagmus (“doll’s eye reflex”)
   i. CN VIII (vestibulocochlear) for sensory
   ii. CN III, IV, VI (occulomotor, trochlear and abducens) for motor of eye muscles

f. Static or positional strabismus
   i. Positional strabismus = CN VIII (vestibulocochlear)
   ii. Static strabismus = CN III, IV, VI (occulomotor, trochlear, abducens)

g. Facial appearance
   i. CN V (trigeminal), mandibular branch
      1. Normal muscling around head (“masticatory muscles”)
   ii. CN VII (facial)
      1. Facial expression
         a. Normal lip/cheek/ear positions and expressions

h. Gag reflex
   i. CN IX, X (glossopharyngeal, vagus) for sensory of pharyngeal area
   ii. CN X, XI, XII (vagus, accessory, hypoglossal) for motor of pharyngeal m. and tongue

Spinal reflexes
Reflexes can help identify/confirm lower motor neuron dysfunction if reduced/absent. In the thoracic limb, the spinal cord segments that could be affected are C6-T2. In the pelvic limb, the spinal cord segments that could be affected are L4-S1. If the perineal reflex is affected,
S1-S3 cord segments may be involved, and if tail reflex/tone is abnormal the caudal cord segments could be abnormal. The cutaneous trunci reflex acts as the “limb” reflex for the T3-L3 spinal cord segments.

1) Thoracic limb
   a. Biceps, triceps reflexes
      i. Not consistent in all patients, use in conjunction with muscle tone/weight bearing
   b. Withdrawal reflex
      i. Ensure normal range of motion of limb first
      ii. Can test strength of withdrawal, too, by holding onto the limb which may be sensitive for lower motor neuron dysfunction if poor

2) Pelvic limb
   a. Patellar reflex
      i. If absent, weight bearing should be affected d/t femoral n. dysfunction
   b. Withdrawal reflex
      i. Hip flexion is controlled by the femoral n., but flexion of the other joints is from the sciatic n. distribution.
      ii. Can test strength of withdrawal by holding limb

3) Perineal reflex/tail clamp
   a. Can combine with anal tone palpation via rectal
   b. Both the anal and urethral sphincters are innervated by the pudendal n.

4) Cutaneous trunci
   a. Useful to locate a T3-L3 lesion based on the “cut off” of the reflex
      i. Cut-off is always 1-2 vertebrae caudal to the lesion
   b. Can be absent if there is a lesion C8-T1, as the reflex originates there in the spinal cord

Palpation

1) Muscle mass/tone
   a. Sudden and severe loss of muscle mass can indicate lower motor neuron dysfunction, especially loss of innervation
   b. Decreased tone usually indicates lower motor neuron dysfunction while increased tone usually indicates upper motor neuron dysfunction

2) Pain
   a. Muscle pain occurs in some lower motor neuron diseases of the muscle
   b. Joint pain = could mimic lower motor neuron diseases d/t short/choppy gait
   c. Paravertebral pain
      i. Direct palpation
         1. Signs of discomfort may be subtle, keep hand on abdomen
      ii. Range of motion (cervical)
         1. Do not perform in toy breeds or if trauma is suspected
   d. Skull pain
      i. Can squeeze temporal bones and bullae
         1. May indicate intracranial lesion
Final thoughts

So much of the neurologic exam is about observation. Don’t miss the forest for the trees! Spend at least 1/3 of the total exam time observing the pet’s mentation, posture, gait, facial symmetry and muscling. That information will give you most of what you need to localize broadly, and you can use the rest of the exam to fine-tune!