Objectives

• Recognition of gait deficits
• CSF analysis
• Vitamin E

Neurologic Examination

• Is there neurologic disease?
• Neuroanatomic localization

Rech and Barros Vet Clinics 2015
Neurologic Examination

- Cerebral cortex
- Brain stem
- Vestibular system
- Cerebellum
- Spinal cord
- Peripheral nerve
- A horse with no signs of brain disease or cranial nerve deficits has a lesion caudal to the foramen magnum

Gait Deficits

- Ataxia
- Paresis
- Dysmetria

- Spasticity
- Proprioception

Ataxia

- Lack of coordination of motor movements
  - Vestibular
  - Cerebellar
  - Sensory
- Description of clinical signs; not a diagnosis
- +/- spasticity or paresis
  - Sensory ataxia is often associated with paresis and often difficult to distinguish
Paresis

- Deficiency of voluntary movement arising from reduced muscular power
  - UMN:
    - Neuron
    - Axon:
      - Cerebral cortex
      - Subcortical white matter
      - Brainstem
      - Spinal cord
    - Weakness is induced by decreased activation of LMN
    - May be accompanied by spasticity
  - LMN
  - Muscle

Dysmetria

- Hypermetria:
  - Exaggerated range of motion
  - Excessive joint movement
  - Cerebellar / Spinal cord disease
- Hypometria:
  - Limb stiffness (tin soldier)
  - Decreased joint flexion
  - Exaggerated flight phase when going downhill or when head is elevated

Proprioception

- Ability to recognize the position of the limbs, body, and head in space
- Conscious: cerebral cortex
- Unconscious: cerebellum
- In horses best evaluated by dynamic observation:
  - Base-wide stance
  - Abnormal position of limbs when coming to a stop
  - Truncal sway
  - On the circle:
    - Abduction of outside limb
    - Pivoting
    - Crossing rear limbs
  - Exacerbated by head elevation
Differentiating neurogenic and musculoskeletal gait abnormalities

- Musculoskeletal disease will lead to abnormalities seen during the neurologic examination
  - In particular multiple limb / bilateral hind limb lameness
- Musculoskeletal: Regularly irregular
- Neurologic: Irregularly irregular
  - Abnormality should be apparent in all phases of examination
- NSAIDs, Nerve Blocks, Joint anesthesia

Differentiating neurogenic and musculoskeletal gait abnormalities

- NM system is composed of motor units:
  - Motoneuron or LMN
  - Its axon
  - Its axonal terminal(s)
- NM junction
- Skeletal muscle fibers (myofibers or myocytes)
Diseases

- CVCM
- EPM
- EDM

Neuroaxonal Degeneration (NAD)

- Degeneration of neurons and axons in brain and spinal cord
- Equine Degenerative Myeloencephalopathy
- Genetic
- Vitamin E

Clinical Signs

- Symmetrical ataxia
- Proprioceptive deficits
- Tetraparesis
- Hypermetria
- Occasional dull mentation

- 6 – 36 months
Diagnosis

• Post mortem
• Young age / Clinical signs
  – Stabilize
  – Slowly progressive
• Related horses affected
• Negative for EPM, WN
• Normal radiographs / myelogram

Equine Protozoal Myeloencephalitis

• ACVIM Consensus Statement (Reed et al): JVIM 2016
• Sarcocystis neurona
• Neospora hughesi
• Opossum
  – No horizontal transmission in the horse
  – No transmission to the horse from intermediate hosts
• CNS penetration
  – Endothelial cells / Leukocytes

Slide from Dr. Stephen Reed
Equine Protozoal Myeloencephalitis

- Disease Development?
  - Protozoal load
  - Protozoal genetics
  - Immune system
  - Stress

Risk Factors

- Age 1 – 5 years; > 13 years
  - 62% horses < 4 years
- S. neurona seroprevalence: 15 – 89%
- Fall (6x); Spring and Summer (3x)
- Presence of opossums
- Presence of wooded areas
- Previous EPM diagnosis
- Race horses and Show horses
Clinical Signs

- Acute / Chronic
- Insidious
- Severe
- Focal / Multifocal
- Brain, Brainstem, Spinal Cord
- Asymmetric ataxia
- Paresis
- Spasticity
- Sensory deficits
- Obtundation
- Head tilt
- Facial nerve paralysis
- Swallowing

Diagnosis

- 1) Thorough neurologic examination
  - CLINICAL SIGNS
- 2) Other disease should be ruled out
- 3) Immunodiagnostic testing
  - Serum and CSF
  - Confirm intrathecal antibody production
  - SnSAG 2 4/3 ELISA
  - NhSAG 1 ELISA

Treatment

- FDA-approved anticoccidial drugs
- a) Ponazuril (Marquis; Merial, Inc., Duluth, GA 30096, USA)
- b) Diclazuril (Protazil; Merck Animal Health, Madison, NJ, 07940, USA)
- c) Sulfadiazine/Pyrimethamine (eg, ReBalance; PRN Pharmacal, Pensacola, FL, 32514, USA)
Ancillary Treatment

- NSAIDs
- Corticosteroids
- DMSO
- Vitamin E
- (Biologic Response Modifiers – Immunomodulators)
  - Levamisole
  - Killed Propionibacterium acnes (EqstimTM; Neogen, Lansing, MI)
  - Mycobacterial wall extract (Equimune IV; Bioniche Animal Health Vetoquinol, Belville, ON, Canada)
  - Inactivated parapox ovis virus (Zylexis, Zoetis, Florham Park, NJ)
  - Transfer factor (4Life Transfer Factor, 4LifeResearch, Sandy, UT)

Vitamin E

Vitamin E deficiency on its own does not appear to reliably cause disease in horses.

No apparent clinical signs resulting from vitamin E deficiency in exercising or resting horses.

3 specific diseases that consistently have been associated with α-tocopherol deficiency:
- EMND
- NAD/EDM
- Vit E deficient myopathy

Finno and Valberg; JVIM 2012
Vitamin E

- Natural α-tocopherol vs. synthetic vitamin E?
  - CSF concentrations of α-tocopherol were significantly elevated after supplementation with the natural vitamin E, but not after supplementation of a synthetic all-rac-α-tocopherol acetate

- Natural RRR-α-tocopherol (nonacetate) form of vitamin E is currently recommended to be used to supplement deficient horses