From head to hoof: distinguishing the neurologic horse from the lame horse

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Disclosure statement

Presenter: Sarah F. Colmer

Disclosure:
I do not have any relevant financial or non-financial relationships with industry or commerce to disclose

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Anatomy of today's discussion

- Where to start
- Summary of examination procedure
- Case examples

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Definitions

- **Lameness** – limping; abnormal gait due to musculoskeletal dysfunction
- **Ataxia** – incoordination; abnormal gait due to neurologic dysfunction

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Where to start?

- Consider the most severe abnormality (lameness vs ataxia)
- Consider the goals of the client – which is most important to them?

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Where to start?

- **Historical elements**
  - Description, duration, interventions attempted
  - Does the owner perceive pain?
  - Is the problem predictable?
  - Behavioral component?

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Behaviour issues

- "Pain vs brain" (vs learned)
- Painful stimuli
  - Orthopedic (neck, other)
  - Muscular / myopathic
  - Tack fit
- Brain dysfunction
  - Neurodegenerative disease (eNAD/EDM)
- Other
  - Vision issues


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The examination(s)

- **Depending on the history, exam or owner desires, you may be starting with neurologic or lameness evaluations**
- Our typical procedure:
  - History-taking
  - Physical examination
  - Neurological evaluation
    - Mentation, behavior
    - Cranial nerve evaluation
    - Posture, reflexes, musculature
    - Gait

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Proprioception

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The examination(s)

- **When starting with our gait evaluation:**
  - Walk in straight line
  - Trot in a straight line
- **Relevance of gaits:**
  - Neurological abnormalities best seen at a walk
  - Lameness often best seen at a trot

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Paresis and neurolocalization

- **Upper motor neuron (higher order, regulate movement and tone - inhibitory)**
- **Defects result in:**
  - Long stride
  - "Floating"
  - "Bouncy"
  - Spastic
    - Can be mistaken as cerebellar (which is much more rare)
- **Lower motor neuron (provide motor to peripheral targets)**
- **Defects result in:**
  - Short stride
  - "Choppy"
  - Weakness
  - Trembling/knuckling
  - Muscle atrophy

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The walk

- Separates out grades 1 + 2

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The trot

- Ataxic horses often have a very "bouncy" trot
  - Likely due to increased tone
- NOT a comprehensive lameness evaluation
- Identify if there is prominent, influential lameness
- **More lame or neurologic (ataxic)?**

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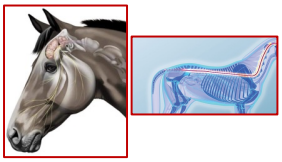
Walk vs trot

Ataxia



- **Irregularly** irregular
- Primarily evaluated at **walk**

VS

Lameness

- **Regularly** irregular
- Primarily evaluated at **trot**

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Serpentines

- Toe dragging?
- Circumduction?
- Delayed protraction?

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Head elevation

- 1. Removes horizon – relies on proprioception
- 2. Changes neck position
- Does it change the gait?
- Does it make them reluctant?

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Tail pull

- Ease of pulling off track
- Overshooting return to straight track?
- Symmetric?

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Tail pull

- Not every horse is amenable!

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Tight circles

- Delayed protraction
- Circumduction
- Interference?
- Different in one direction?

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Tight circles

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Video credit: Amy Johnson

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Walking backward

- **Normal**
  - Quick
  - Diagonal pairs
  - No toe dragging
  - No interference

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Walking backward

- **Abnormal**
  - Slow
  - "Pacing" / lateralization
  - Toe dragging
  - Interference

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....penis ataxia?

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Navigating hills

- **Toe scuffing**
- **Slipping**
- **Buckling in hind limbs**
- **Hypermetria (“floating”) in forelimbs**

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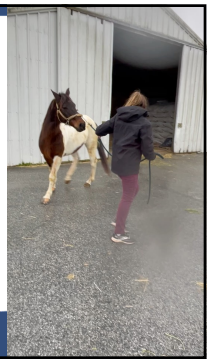
A note about footing

- Hard vs soft – may differ
  - Deep footing may worsen (or mask!)
- We watch them walk straight and circles on both surfaces



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Neck range of motion

- “Carrot stretches”
- Later (right and left)
- Dorsoventral

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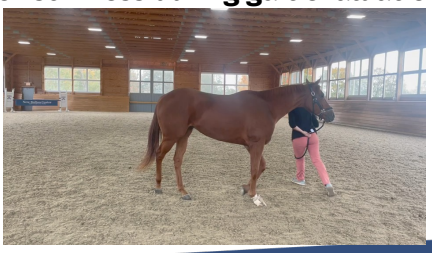
Neck stiffness during gait evaluation



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Grazing

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Gait evaluation overview

- Standing position
- Walk – straight line
- Trot – straight line
- Serpentines
- Head elevated on flat ground
- Tail pull
- Tight circles in either direction
- Walk backward
- Walk up and down a hill – neutral
- Walk up and down a hill – head elevated
- Tight circles on hard surface
- Neck range of motion
- Grazing

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Summarize findings

- Neurologically normal or abnormal?
- In how many limbs is the horse abnormal?
- Type of ataxia?
- Upper or lower motor neuron?
- If spinal ataxia, what grade?
  - Modified Mayhew ataxia scale
- **Neurolocalization?**

- *Then* formulate ddx and diagnostic plan

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Key points for "neuro vs lame"

- **Stride length**
  - **Lame**: short and choppy
  - **Ataxic**:
    - UMN: long, floaty
    - LMN: short, choppy
- **Hind end drifting**
  - **Lame**: away from lame leg
  - **Ataxic**: toward affected leg / bi-directional
- **Foot placement**
  - **Lame**: regularly irregular
  - **Ataxic**: irregularly irregular

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What now?

- **Is it lame, neurologic or both?**
  - Which is more severe?
  - Which is client priority?

- **When it's a combination and/or not entirely clear:**
  - Consider diagnostic analgesia followed by re-evaluation
    - Do deficits lessen/disappear?
  - Consider anti-inflammatory trial then re-evaluate
  - Consider evaluating a different day

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Common things happen commonly

- CVSM / Wobblers
- eNAD/EDM
- EPM (for us!)

- Myopathies
  - PSSM (1+2)
  - MFM
  - Vitamin E responsive myopathy
  - Exertional rhabdomyolysis
- Peripheral neuropathies
- Movement disorders

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	CVSM	EPM	EDM
Chronic tripping or stumbling	*		*
Rapid worsening	*	**	
Improvement with NSAIDs	*		
Improvement with steroids	*	*	
Improvement with rest	*	(worse)	(worse)
Neck pain/stiffness	*		
Thoracic limb lameness	**	*	
Pelvic limb lameness		**	
Dullness or depression		*	*
Bad or unpredictable behavior	*		**
Focal or asymmetric atrophy		**	
Diffuse, symmetric atrophy	*		*
Cranial nerve deficits		**	

The "big 3" (in our cases in the US)

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Pursuit of diagnostics

- **Lameness**
  - Diagnostic analgesia
    - Non-blockable forelimb lameness +/- neck stiffness
  - NSAID trial
  - Imaging (radiography, nuclear scintigraphy, cross-sectional)
- **Neurologic disease**
  - Imaging (cervical radiographs, myelography)
  - CSF analysis
- **Myopathies**
  - Muscle biopsies
- **Other**

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"Bramble"

- 16-year-old Hanoverian gelding
- Neck stiffness and gait abnormalities, acute worsening

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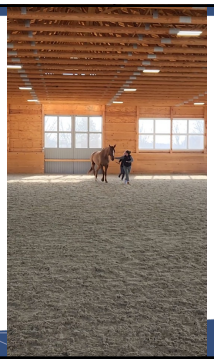
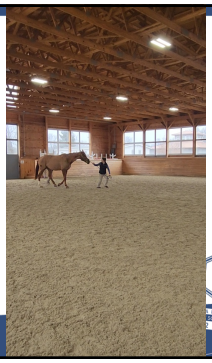

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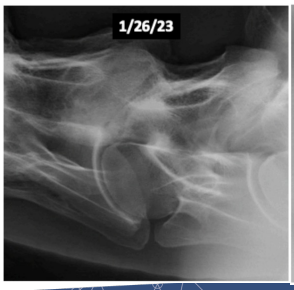

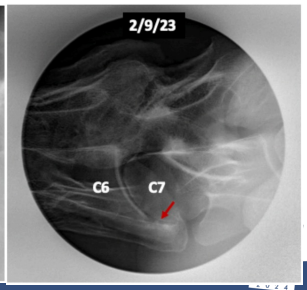

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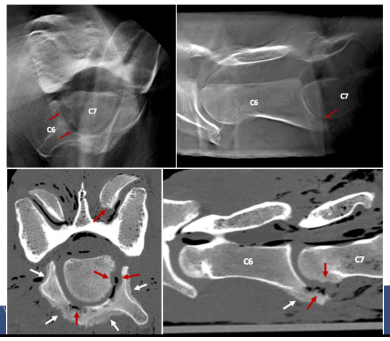

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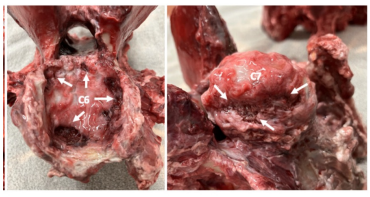

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"Sun"

- **History:**
  - 11-day history non-weight-bearing lameness, left hind limp
  - Intermittent history of stumbling and falling, 2 weeks
  - Became cast; sensitive to palpation of left semis, unable to extend on hard ground

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”Sun” - admission

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”Sun”- progression

- Unable to weight-bear LH
- Did not block out up to low-six-point block
- Considered neurologic differentials
  - EPM
  - Trauma
  - Melanoma

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S. neurona and Neospora hughesi serum:CSF ratio

Combined SAG 2,3,4 Titer on serum:
1:4000

Positive, specific antibodies were detected. This result indicates exposure to S. neurona, a causative agent of equine protozoal myelitis (EPM). It does not confirm clinical disease. Serum titers range from <1:250 (negative) to >1:4000 (high).

Combined SAG 2,3,4 Titer, CSF:
1:10240

Positive, antibodies were detected. CSF titers range from <1:2.5 (negative) to >1:40 (high). With CSF titers of ≥1:20 active disease caused by S. neurona is probable in the presence of accompanying neurological signs. A serum to CSF titer ratio is recommended.

Combined SAG 2,3,4 serum to CSF titer ratio:
0.39 Ratio is <100

A serum:CSF titer ratio of <100 is highly diagnostic of clinical EPM, since this is indicative of intrathecal antibody production.

Serum Albumin:
3321 mg/dl Elevated **Reference Range** 1000 - 3000 mg/dl

CSF Albumin:
50.4 mg/dl Normal **Reference Range** 15 - 70 mg/dl

Specific Index Calculation:
168.42 >1.0

Elevated, the S.neurona specific index is elevated. This supports an EPM diagnosis.

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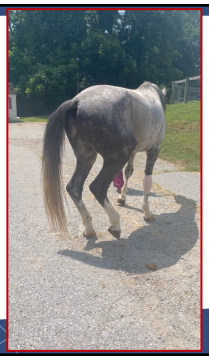

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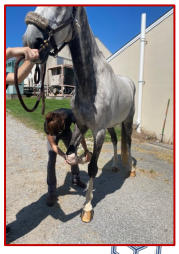

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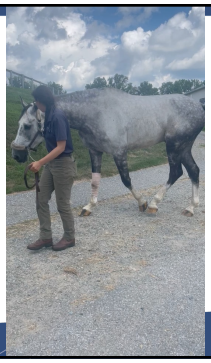

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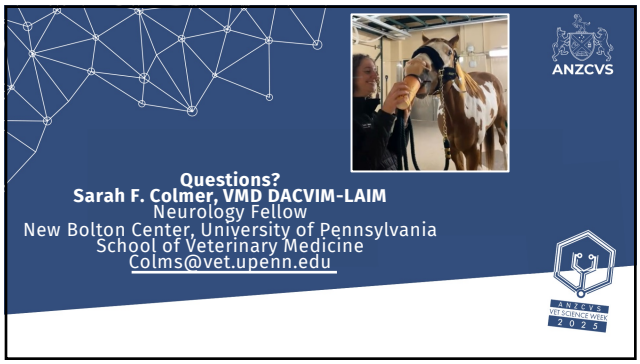
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