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Neurologic disease in the adult horse

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Goals

- Using a Case Series
  - Differential diagnosis using neuroanatomic localization
  - Early treatment of neurologic disease
  - New techniques in neurologic diagnosis

Neurologic exam

- Is this truly a neurologic disorder?
- Where is/are the sites of injury?
- What other tests do I need to diagnose disease and determine treatment?
Neurologic examination - 4 parts

1. Mentation
2. Cranial Nerves
3. Gait Analysis
   - Posture
   - Movement
   - Complicated maneuvers
4. Reflexes, sensation, body symmetry

General observation

- Advantage to observe the horse free in a stall.
  - Is he alert & aware of the environment around him?
  - Vision? Hearing?
  - How does he move in confines of stall?
  - Body posture and limb placement
- If examining at clinic horse is often excited so observation may reveal abnormalities that may not be so apparent in another, more comfortable situation.
- Often can get a great deal of information observing animal

Gait analysis

- Record what you see not what you think!
  - Exaggerated swing phase
  - Circumduction
  - Bouncing when circling
  - Foot flight path is not consistent
- Once you have recorded the info then determine if signs are consistent with ataxia, CP deficits or weakness
- *Gait analysis is not always accurate
Gait analysis

- Paresis- upper motor neuron vs. lower motor neuron
  - Lower motor neuron- difficulty supporting weight.
  - Shortened stride, bounce to weight bearing-recumbency
  - Upper motor neuron- UMN initiates the gait through recruitment of LMN.
    - Delay in onset of protraction (swing phase) of gait.
    - Spasticity to movement

- Ataxia- 3 types
  - General sensory – Proprioceptive & UMN- often affected together. Signs of loss of knowing where limb is
    - Exaggerated swing, hypermetria, scuffing of toes, spasticity
  - Vestibular ataxia- Leaning drifting or falling to one side.
    - Usually accompanied by a head tilt +/- nystagmus
    - Blindfolding animal makes signs much worse.
  - Cerebellar ataxia-
    - Stiff, spastic, hypermetric gait
    - Often more obvious in thoracic limbs
    - May swing head and neck side to side in faster gaits.
    - Intention tremors are a clue that cerebellar disease

Video
Gait analysis

- Not always clearly Sensory vs. Motor
- Many abnormalities in the gait can be due to either
- ex-toe dragging, abnormal foot flight can be due to motor deficits or sensory deficits.
- Bottom line is that these tracts run together so not as critical to differentiate the deficit as motor (weakness) vs. sensory.

Cases

Rumplestilskin - 5 year old Morgan Gelding

- July 2009 presented with a 3 week history of difficulty eating
  - Associated with new shipment of hay but was still eating grain.
  - 10 days ago decreased water intake
  - Last few days noted dropping hay and grain when eating
  - Shaking head more frequently
  - Vaccinated with EEE/EEE/Tet/WNV/INF/EHV in spring
Rumplestilskin- Physical exam

- BAR-H
- Ulceration of tongue
- Repeatedly rubbing face on walls
- Neurologic exam:
  - Difficulty masticating food
  - Tongue tone appears normal but occasionally seems to hang out of mouth
  - Mild ataxia of hindlimbs (Grade I/IV)
  - Possible mild atrophy of masseters
  - Temporal muscles appear normal
  - Some decreased sensation over mandible, muzzle and forehead and ears have normal to increased sensory response

Rumplestilskin- Location of Lesion(s)

- Focal or Multifocal?
  - Atrophy/dysfunction of muscles of mastication
  - Motor CN V
  - Sensory deficits on face?
    - Sensory CN V
  - Ataxia/Weakness hind limbs
- Multifocal disease
- Rule out list gets shorter.

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Rumplestilskin- Differential Diagnosis

- CVM
- EPM
- WNV
- EEE
- EHV
- Neoplasia/ abscess/ trauma
- Equine Degenerative Myeloencephalitis
- Equine polyneuritis equi (aka cauda equina syndrome)
- Meningitis
- P. tenuis
- Rabies
- EMND
- Others?

Rumplestilskin- Plan?

- CSF tap & cytology
  - Normal cytology protein 40 mg/dl
- WNV- IgM ELISA & Plaque reduction neutralization test
  - Why both?
  - Both negative
- EPM Western blot
  - Serum & CSF positive
- MRI- Enlarged trigeminal nerves, mild enlargement of R vestibulocochlear nerve, atrophy of masseter, temporalis muscle mass.
- Diagnostic interpretation: Likely right trigeminal and vestibulocochlear neuritis, inflammation of the left trigeminal is less evident, but possible, given the subjective mild enlargement. Neoplasia of multiple cranial nerves is extremely unlikely. Secondary muscle of mastication atrophy.

Rumplestilskin- Treatment

- Ponazuril (Marquis) 900 lb dose orally once daily 4 weeks
- Phenylbutazone- 1 gram orally twice daily tapering dose.
- Feed: Soaked pelleted diet in deep bucket, fed multiple times a day
- Update in 2 weeks.
Rumplestilskin - recheck

- Phone conversation ~ 2 weeks later
- Managing to maintain weight with soaked feeding.
- Owner thinks may be a slight improvement in neurologic deficits
- Plan to continue therapy at least 4 to 6 weeks

Rumplestilskin - January 2010

- Owner called to discuss lack of progression
- Gelding still unable to eat normally
- Reports tongue hangs out more continuously.
- Now incontinent - urine dripping continuously
- Still rubs face frequently
- Gait deficits possibly worse in hind limbs.
- Owner wishes to donate him for euthanasia and post mortem.

Video
Rumplestilskin - Post Mortem Results

The horse was markedly thin, with atrophy of the gluteal muscles, masseter muscles, and multifocal atrophy of the epaxial muscles. The extradural nerve roots of the cauda equina were yellow, severely thickened, and firm; the roots most severely affected were within the right side of the cauda equina. In the corresponding innervated muscles, normal myofibers were atrophied and replaced by pale yellow tissue. Similar severely atrophied muscles were found scattered within the right gluteal and quadriceps muscles. The lumen of the urinary bladder was distended by a large, yellow, semisolid accumulation of urine sediment (sabulous urolithiasis).

- **Diagnosis:**
  - Cauda equina: Severe, chronic, multifocal granulomatous neuritis
  - Skeletal muscle: Severe chronic multifocal atrophy of the gluteal, masseter, and epaxial muscles
  - Urinary bladder: Sabulous urolithiasis

In sections taken from the cauda equina, and lumbar spinal cord (just proximal to the cauda equina) normal extradural peripheral nerve architecture is extensively effaced and replaced by a mixed inflammatory cell infiltrate and dense fibrous connective tissue. Composed primarily of abundant aggregates of epithelioid macrophages with vacuolated cytoplasm, fewer numbers of scattered, and occasionally clustered lymphocytes, and rare multinucleated giant cells. Remnant axons are swollen and hypereosinophilic, and frequent clear vacuoles have a swollen axon (spheroid) or macrophage within the center (digestion chamber). Multifocal areas are characterized by the presence of pale staining eosinophilic material, mixed with pyknotic and karyorrhectic nuclear debris. In the section from the lumbar spinal cord, there are also moderate numbers of scattered eosinophils, and dense, band-like proliferations of Schwann cells (Bungner's bands). The white matter of the lumbar spinal cord contains occasional, scattered spheroids, and rare digestion chambers. Within a section of spinal cord taken from the cranial aspect of the cervical portion, a peripheral nerve is infiltrated by low numbers of lymphocytes.
Antemortem diagnosis of Polyneuritis equi
Muscle biopsy of tailhead revealed infiltrative macrophages and lymphocytes in nerve bundles.

Sam- 2 year old QH gelding
- Purchased in December 2010 part of a "fire sale"
- Began working with horse- noticed unusual gait behind.
- Physical exam- within normal limits
- Neurologic exam
  - Mentation- normal though quiet, young horse.
  - Cranial nerves exam
    - Possibly sluggish menace on right eye compared to left eye.
  - Gait analysis- see video
  - Sensation-
    - Cutaneous trunci normal
    - Cervico-facial reflex normal
  - Reflexes- normal
  - No evidence of muscle asymetry
Sam

Gait assessment
Grade 2 out of 4 weakness and ataxia in pelvic limbs
Hypermetric gait behind
Stabbing dysmetric gait
Grade 1 (?) out of 4 forelimbs

Sam - Differential Diagnosis

CVM
Equine protozoal myelitis
WNV
EEE
EHV
Neoplasia/abscess/trauma
Equine Degenerative Myeloencephalitis
Equine polyneuritis equi (aka cauda equina syndrome
Meningitis
P. tenuis/parasitic migration
Rabies
EMND
Others?

Plan?

Cervical radiographs
CSF tap
EHV-1 PCR nasal swab
Myelogram?
EPM western blot analysis
Cervical radiographs

- Mild osteoarthritis C6-C7
- Sagittal ratios:
  - 58% C3
  - 56% C4 (> 52%)
  - 55% C5 (> 52%)
  - 57% C6 (> 52%)
  - 58% C7 (> 56%)
- Unlikely to have CVM what next?

Plan?

- CSF cytology
- EPM testing
  - Western blot vs. IFAT vs. sag ELISA
    - Western blot and IFAT both have good sensitivity and specificity.
    - sagELISA- poor sensitivity (i.e. lots of false positives) good specificity
CSF tap

- New technique described at MSU
- Actually old technique that was rediscovered!
- Standing tap at AO-C1 region.
- Horse sedated with Detomidine/butorphanol/morphine
- Ultrasound guidance - spinal cord easily visualized.
- Approach is at an ~135 degree angle
- Allows collection of CSF rostrally with little risk of blood contamination and without anesthesia.
Sam-

- **CSF cytology**-
  - 10 RBC/ul
  - 2 cells/ul consisting of mononuclear cells only
  - Microprotein 25 mg/dl
  - Specific gravity: 1.005
- **EPM Western blot**
  - Serum: positive
  - CSF: negative
Sam

- Owner declined further workup at this time.
- Discharged
- Treated with high doses of Vitamin E ~ 1 year
- No improvement in neurologic signs
- Owner requested euthanasia & postmortem evaluation.

The brain was microscopically normal, except for accumulation of lipofuscin in small numbers of scattered brainstem neurons. In all sections of the spinal cord, there was mild to moderate attenuation of myelin in the peripheral white matter tracts of the dorsolateral aspects of lateral funiculi and the ventromedial aspects of ventral funiculi. A few neurons (no more than 2-3 per section) in the intermediate gray matter or medial aspect of the dorsal gray horns had rather homogeneous eosinophilic cytoplasm and lacked discernible Nissl substance. Occasional swollen eosinophilic axons (spheroids) were present in the vicinity. Small numbers of scattered spinal neurons contained lipofuscin. In a few spinal cord sections, there was mild gliosis of the gray matter, with the increased number of glial cells predominantly around neurons (satellitosis).

Neuroaxonal dystrophy in Quarter Horses

- Indistinguishable from Equine Degenerative Myelitis
- Seen in humans, sheep, cats and dogs & other breeds of horses
- Recently described on a large cutting horse farm
  - 59% of horses on farm were affected
- Signalment—typically young (<2 years) no sex or breed predispositions
- Association with low Vitamin E level.
EDM- Clinical Signs

- Most develop clinical signs in 1st year of life
- Most cases have slow progression of signs
- Clinical signs:
  - Progressive ataxia and weakness hind > fore
  - Base wide stance at rest
  - Abnormal circling is most apparent
  - Dull mentation may be noted
  - Sluggish menace may be seen
  - Decreased cutaneous trunci sensation & cervico-facial slap test response may be seen.
  - Differentiates from CVM
  - Toe stabbing gait, particularly going up hill.

EDM- Pathologic findings

- NO GROSS Lesions
- Neuroaxonal dystrophy in brain stem nuclei:
  - cuneatous, gracilis, lateral cuneate
- Equine degenerative myelitis
- Diffuse neuroaxonal dystrophy of spinal cord
- Most prominent in spinocerebellar tracts in cervicothoracic region
- Axonal swelling (spheroids) pigment accumulation & astrogliosis
- Grey matter lesions in spinal cord most marked in spinocerebellar tracts
- Pathologic findings are consistent with some of the experimental Vitamin E deficiency in monkeys

NAD/EDM

- Familial inheritance shown by breeding studies for EDM
- Two STB stallions’ offspring had a 40% incidence compared to other stallions on farm.
- Inheritance likely polygenic mode or has variable expression
- Supplementation with Vitamin E decreased incidence in offspring
- Supplementation with Vitamin E in affected offspring (at 6 months of age) resulted in some improvement.
- Genetic counseling resulted in a decline in incidence from 59% to 10% in next foal crop in the QH farm
EDM- Risk factors

- Questionnaire- sent out to 235 cases (56 affected with EDM)
- Exposure to insecticides
- Exposure to treated wood
- Time spent on dirt lots
- Appaloosa Breeding Study (EDM)
  - Deliberately avoided above risk factors- still saw high incidence
  - Likely multifactorial with genetic predisposition
- Vitamin E deficiency

Vitamin E deficiency?

- Inadequate dietary intake?
- Reduced absorption of Vitamin E?
  - lack of bile acids
  - Intestinal malfunction
- Failure to make chylomicrons in enterocytes?
- Absence of transfer protein in liver?
  - chylomicrons to lipoproteins
- Lack of adequate cellular vitamin E receptors?
- Increased utilization or excretion of Vitamin E?

Vitamin E supplementation?

- Probably not necessary if:
  - On pasture
  - Fed well cured recently stored hay
- Risk factors for deficiency:
  - Feeding old hay or pelleted feed
  - Long winters
  - Exposure to insecticides or creosote-painted fences
  - Horses with neurologic disease
- Current recommendations
  - Natural Vitamin E much more readily absorbed than synthetic
  - Current supplementation
    - Requirement – 2,000 iu/day
    - For treatment of disease- 5-10,000 iu/day
Almighty Thor
June 21, 2007

Signalment & History

- 3 year old STB gelding
- Raced 3 weeks ago
- Training problems developed
  - Hocks/ Shoulders injected
  - Given an “anticoccidial” for several weeks
- Circling in stall 7 days prior to admit.
  - Admitted to rDVM practice 4 days later.

- Treated by rDVM with DMSO, Bute, Marquis, B-vitamins, Dexamethasone
- Nasal swab submitted for EHV-1
- Vaccinated for WNV & EHV-1 only
- Appetite decreased at rDVM
- Facial nerve dysfunction
- Circling compulsively to the right at rDVM's
Admission
- Very ataxic off the trailer.
- Collapsed on initial admission into stall
- Multiple lacerations on body
- Left eyelid swollen
- Normal temperature elevated HR (60)

Neurologic exam - CN exam
- + menace in both eyes
- Normal PLR on R, left pupil constricted
  - Corneal ulcer on left eye
- Nystagmus variable
  - Horizontal w/ fast phase to right, then left.
  - At times vertical nystagmus noted.
- Left ear droop, muzzle pulled to right, menace, retracts left globe.
- Able to chew and swallow, normal tongue tone

Neurologic exam
- Head tilt to left, circles to right
- Leans to left predominantly but occasionally found with neck twisted to right (see video).
- Mentation - depressed, obtunded but hyperesthetic
- Normal anal & tail tone, defecating & urinating normally
- Reaction to stimulus varies from normal to extremely violent.
- Intention tremors noted when offered feed.
Neurologic exam - Gait analysis

- Extremely unsteady, falls with little provocation
- Hypermetric gait in all 4 limbs
- Sway to trunk when standing
- Leaning to left when standing but circles to right when walking.
- Grade 4/4 weakness all 4 limbs
- Grade 4/4 ataxia all 4 limbs

Site of lesion

- Ear droop and muzzle deviation on left side
- Nystagmus varies from horizontal to vertical
- Left pupil miotic
- Hypometric
- Intention tremors
- Mentation depressed
- Extremely hypermetric
- Severe weakness and ataxia
- Circling to right
- Head tilt to left
- Left sided facial nerve paralysis
- Miotic pupil likely response to corneal ulcer
- Vestibular signs - central, paradoxical
- Gait abnormalities - cerebellar, spinal cord, brain stem
- Intention tremors - cerebellar
- Mentation abnormalities - cerebral, RAS

Location of lesion
- Likely multifocal due to multiple deficits involving central brain stem & cerebrum
DDx?

- EPM
- Meningitis/abscessation
- EHV-1
- Parasitic migration
- EEE, WNV, Rabies
- Metabolic
- Trauma
- Temporohyoid osteopathy
- Cervical Stenotic myelopathy

Diagnostic plan

- CSF tap & cytology & culture?
- EPM testing
- EHV-1 PCR on CSF, nasal swab & buffy coat
- Skull films, CT scan, MRI?
- Post mortem…?

Post Mortem Exam

- Grossly normal CNS
- Rabies negative
- EEE IFA on brain tissue negative
- WNV PCR negative
- EHV-1 PCR negative
- Histopathology: Focally extensive tracts of marked rarefaction of neuropil with inflammatory infiltrates (neutrophils and multinucleated giant cells) from caudal brain stem to hippocampus involving both white and grey matter.
- Diffuse gliosis, neuronal degeneration, necrosis and astrocytosis.
- Perivascular cuffing with lymphocytes, plasma cells, eosinophils and fewer neutrophils.
- IFA positive for EPM
EPM

- USDA study 14 cases/10,000 horses/year
- Competition/racing much higher incidence compared to pleasure & breeding
- TB, QH, STB & young horses over represented
- Parasite dose, strain, immune function and physiologic stress (including strenuous exercise) thought to play a role in pathogenesis
- Common experimental model- long-distance transport supports role of stress.
- 50-60% seroprevalence in the USA.

EPM

- Clinical signs commonly relate to spinal cord and brain stem infection
- Cerebral disease uncommon
- Slowly progressive asymmetric gait deficits with focal muscle atrophy and/or cranial nerve deficits most common.

EPM Diagnosis

- Currently no perfect test for EPM
- All immunologic tests have varying sensitivity and specificity
- Antibody can move passively across BBB making interpretation of CSF titers difficult.
  - Need to be interpreted in light of serum values if possible.
- Modified Western blot
- IFAT
- Sag1 ELISA
- PCR
- What about N hughesi?
Western blot

- First test available
- Serum/CSF from affected horse is incubated with antigens from S. neurona
- Secondary reaction to identify binding or presence of antibody
- Modified Western Blot (MSU)
  - Attempt to decrease false positive results by cross reacting first with other Sarcocystis spp. Antigens
  - Sensitivity & Specificity 100% & 98% @ MSU 89% & 69% in another report

snSAG ELISA(s)

- snSAG ELISA's
  - Surface antigens of S. neurona
  - snSAG-2 ELISA offered the best sensitivity & specificity
  - snSAG-1 very poor sensitivity in multiple studies
  - Important to know what test is being used!

- IFAT
  - Sensitivity & specificity 92.3 & 89.7% on CSF
  - Cut-off values 1:80 serum 1:5 CSF

CSF indices

- Goal is to assess likelihood that titer is due to passive transfer of protein across BBB vs. intrathecal production.
- Calculation of Albumen index & C-value
  - AI 71% sensitivity and 100% specificity
  - C-value sensitivity of 86% & specificity of 100%
  - Blood contamination did not affect these results until very high blood contamination (100,000 RBC/microliter CSF)
Current recommendations

- Know your test!
- Ask for sensitivity and specificity data
- Current recommendations
  - If clinical signs consistent with EPM and
  - Serum positive
  - CSF is recommended to improve accuracy.

Vinnie- 3 year old TB gelding

- Came home from trainers noted to “not be engaging behind”
- ~ 4 months later was given 1 month course of Marquis
- Improvement noted continued on with TMS & pyrimethamine
- Presented for neurologic evaluation
- Neurologic exam:
  - No cranial nerve deficits
  - Normal mentation (3 year old!)
  - Gait- ataxia all 4 limbs, spastic in hind limbs, proprioceptive deficits all 4 limbs (floats leg over curb). Stumbles when asked to transition downward.
- Summary- Grade 3 out of 4 ataxia and weakness in hind limbs
  Grade 2 out of 4 in front limbs

Vinnie- Cervical radiographs

- Moderate osteoarthritis C5-C6 & C6-C7
- C5-C6 sagittal ratio 44%
- Myelogram
  - Cord compression at C5-C6 consistent with Cervical Vertebral Malformation.
3 year old thoroughbred with CVSM - myelography

Facet joint OA between C5/C6

Dorsal dye column interrupted

Vinnie

- Owner’s elected to take home on anti-inflammatories and rest.
- Wanted to consider surgical stabilization of CVM.
- Several months later, Vinnie donated to MSU CVM.

At the time Cervical Vertebral Canal Endoscopy project was


Meningeal spaces

- Subarachnoid space
- Epidural space

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ENDOSCOPY

- Flexible video-endoscope
  - Olympus GIF N-180
  - 110 cm working length
  - 4.9 mm external diameter
  - 2-way angulation capability
  - 2 mm working/biopsy channel

Approach – AO space

- RL recumbency, 15cm dorsal midline incision
- Midline dissection (excl. nuchal ligament)
- Incision of the dorsal AO membrane
  - Epiduroscopy
  - 20º reverse Trendelenburg
- Incision of the dura mater
  - Pre-placed sutures & rubber stints
  - Myeloscopy

Radiographic control

When nerve roots were seen:
1. Lateral radiograph
2. Distance marker on the endoscope

Allows accurate identification of individual nerve roots in the epidural and subarachnoid space.
3 year old thoroughbred with CVSM

Myeloscopy: Intervertebral space between C5/C6 (left) and C6/C7 (right), endoscope dorsal to the spinal cord, looking caudally

Vinnie- Post Mortem

- CVM compression of spinal cord at C6-C7 (not C5-C6)
- In the future this technique may be more sensitive for detecting location of cervical cord compression


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Rocky

- July 2011
- Aged Morgan gelding
- Presented for 2 day history of ADR
- Fever morning of admission
- Toxic mm, dehydrated, depressed, inappetant.
- Manure normal, decreased amounts
- Received 500 mg Flunixin meglumine prior to admission
- Vaccinated against EEE, WEE, Tet, WNV, Rabies, EHV-1, INF one month earlier
Changes in Mentation

- Viral encephalitis
  - Rabies
  - EEE
  - WNV
- Primary hyperammonemia
- Mycotic encephalopathy
- Parasitic encephalitis
- Meningitis
- Cerebral abscess
- Moxidectin
- Others?

Equine hepatic encephalopathy

**Common Causes**
- PA toxicity
- Theiler's disease
- Tyzzer's disease (foals)
- Hyperlipemia
- Cholangiohepatitis
- Cholilithiasis
- Alsike clover

**Uncommon Causes**
- Moldy corn
- Alfatoxins
- Portal caval shunts
- Iron toxicity

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Hepatic encephalopathy

- Most common cause of neurologic disease with mentation abnormalities.
-Often misdiagnosed as colic on initial exam
  - Further evaluation will reveal mentation abnormalities, blindness, ataxia, head pressing, severe obtundation etc...
- Clinical signs of liver failure
  - Icterus most common
- Severe elevation in liver enzymes!
- History- treatment with plasma, TAT or vaccination within last 60-90 days is typical of Serum sickness or Theiler's disease.
Hepatic encephalopathy- treatment

- Tranquilize if needed
- Minimize stress
- Feed grass hay or soaked beet pulp, grains high in BCAA (corn & sorghum)
- IV fluid therapy
- Anti-inflammatories
- Neomycin sulfate - 4-8 mg/kg q 8 hours orally to try to decrease ammonia producing bacteria
- Lactulose? 0.1-0.2 ml/kg po q8-12 hours

HE treatment- unknown?

- Metronidazole 25 mg/kg po q 12-24 hours
- Pentoxyfilline 7.5 mg/kg po q 24 hours
- S-adenosylmethionine 10 mg/kg antioxidant
- Prednisolone (1 mg/kg) for chronic progressive hepatitis
- Vitamin B complex
- Vitamin E
- Avoid sunlight (secondary photosensitization)

Primary Hyperammonemia

- Relatively recently recognized cause of acute neurologic signs.
- Acute neurologic signs in horses with colitis or colic
- Diagnosis
  - History of GI disturbance
  - Laboratory values- hyperglycemic, metabolic acidosis & elevated blood ammonia (>150 umol/l)
- Prognosis- guarded to poor
- Treatment
  - supportive care (IV fluids to correct lactic acidosis)
  - Sedation (CRI may be best but difficult to manage)
  - Protect animal from injury
  - Neomycin orally?
Wyatt

- Middle aged QH gelding
- Presented for acute onset (2-3 days) unsteady in pasture.
- Trail horse, no new horses, no recent travel
- Vaccinated in spring- Core vaccines

Temporohyoid Osteopathy

- All ages and breeds affected
- Sudden onset of vestibular disease and facial nerve paralysis -
- Head shaking / ear rubbing
- Cause – unknown
  - Some cases may have inner/middle ear infection/inflammation that leads to osteoarthritis of the TH joint.
  - Some cases associated with recent dentistry
Facial (VII) and Vestibulocochlear Deficits

Temporohyoid Osteopathy

**Diagnosis**
- Clinical signs
  - Acute onset of unilateral CN 7 & 8 deficits.
- Endoscopy - guttural pouches
- Radiographs
- Sensitive to palpation at base of ear

vestibular and facial nerves
**Temporohyoid Osteopathy**

**Temporohyoid Osteopathy Treatment**
- **Medical**
  - Antimicrobials
  - NSAIDs
- **Surgery**
  - Cut stylohyoid bone
  - Remove ceratohyoid bone
- **Prognosis**
  - Favorable for life
  - Corneal ulcers are common
  - Guarded to poor for performance due to loss of balance and pain associated with tongue head movement.
Ceratohyoidectomy performed on Wyatt

- Ceratohyoidectomy performed on Wyatt 5 days after admission.
- Approximately 3 hours after recovery started cribbing on stall door!
- Later discussion with owner revealed he had stopped cribbing for several weeks.
  - Become very difficult in the bridle
  - Jumped out of stocks during application of a dental speculum.
- Discharged 2 days later
- Two week follow up - owners thought he appeared normal.

Questions?