Vet Education Pty Ltd Presents
The 2013 Equine Mini Symposium

The Neurological Horse

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INTRODUCTION

The neurologic examination should be performed only after a thorough general clinical examination. Neurology is unique in that the clinical signs alone allow the clinician to predict the location of the lesion with fair accuracy. It is appropriate to first attempt to explain the clinical signs with a lesion in a single location: if this is not possible, then a multifocal lesion is likely. Once a neuro-anatomical site for the lesion(s) has been discerned, further diagnostics aimed at characterising the lesion (extent/nature of the damage and aetiology) can be performed.

It is convenient and logical to approach the examination functionally, dividing the neurologic system into:

- Cerebrum
- Cerebellum
- Brainstem
- Cranial nerves
- Spinal cord
- Peripheral nerves

1. **Distant exam: evaluation of mental status**

Watching the horse from a distance first will allow for the best evaluation of mental status (cerebral & brainstem function). Observe the horse in the stall, without interference from people. Subtle behaviour changes are often first noted by the owner and may or may not be indicative of neurologic disease. More severe signs (seizures, head pressing, central blindness, bizarre behaviour) are seen with significant cerebral dysfunction. Also observe the posture – leaning is typical of vestibular disease, a wide-based stance is typical of a cerebellar lesion, and unusual postures may be seen with cerebral dysfunction.

2. **Cranial nerve (CN) examination**

   *Evaluate each nerve by first thinking of its function.*

**CN I: Olfactory**

- Smell difficult to evaluate, lesions very rare

**CN II: Optic**

- Vision
- Evaluation:
  o Menace response (also requires CNVII)
  o PLR (also requires CNIII). Remember that the PLR should still be present with cortical (central) blindness, but not if there is CNII dysfunction.
  o Direct visualisation (ophthalmoscopic exam)

**CN III: Oculomotor**
- Eye movement (all muscles except dorsal oblique (CNIV) and lateral rectus (CNVI))
- Pupillary constriction (dilation via sympathetic autonomic innervation)
- Evaluation:
  o PLR (also requires CNII)
  o Eye position – strabismus may also be caused by CN IV, VI, or VIII dysfunction

**CN IV: Trochlear**
- Innervates dorsal (superior) oblique orbital muscle
- Evaluation
  o Eye position – strabismus may also be caused by CN III, VI, or VIII dysfunction

**CN V: Trigeminal**
- Sensation to most of face, motor to muscles of mastication
- Evaluation
  o Test facial sensation (pen or finger inside each nostril will usually evoke a reaction)
  o Visually evaluate and palpate masseter & temporal muscles for symmetry

**CN VI: Abducens**
- Innervates lateral rectus orbital muscle
- Evaluation:
  o Eye position – strabismus may also be caused by CN III, IV, or VIII dysfunction

**CN VII: Facial**
- Motor to muscles of facial expression. Supplies lacrimal glands
- Evaluation
  o Observe facial symmetry (particularly muzzle, ears and upper eyelids). Muzzle will deviate away from the side of the lesion
CN VIII: Vestibulocochlear
- Balance and hearing
- Evaluation
  - Unilateral hearing loss hard to evaluate in horses
  - Unilateral vestibular disease results in:
    - Head tilt (poll deviated towards the side of the lesion)
    - Lean, circling towards the side of the lesion
    - Loss of balance
    - Spontaneous nystagmus:
      - Fast phase away from side of lesion
      - Look for absence of normal (physiologic) nystagmus
      - Ventral strabismus (of eye on same side as lesion)
        - Particularly noticeable if head held out straight and parallel with ground
    - Blindfolding worsens subtle lesions (removes visual compensation)
    - Central lesion (nucleus) may have associated paresis, nystagmus not horizontal (rotary or random, varied with head position) vs peripheral lesion.

CN IX: Glossopharyngeal
&
CN X: Vagus:
- Laryngeal and pharyngeal muscular function (deglutition)
- Evaluation
  - Observe the horse eating and drinking to assess ability to swallow
  - Endoscopy allows direct visualization of the pharynx, larynx, and swallowing ability and may be useful if dysfunction is suspected.

CN XI: Accessory
- Innervates trapezius and sternocephalicus
- Evaluation:
  - Muscular symmetry – lesions very rare

CN XII: Hypoglossal
- Motor to the tongue
- **Evaluation:**
  - Open mouth and remove the tongue to assess the horse’s tongue strength and symmetry of tongue muscle

3. **Gait analysis**

The difficulty in testing most limb reflexes and placement tests in the standing horse means that gait analysis is the major means by which abnormalities of motor neuron function and proprioception are tested.

**Technique:**

- walk horse in straight line
- trot horse in straight line (unless severely ataxic)
  - in mildly ataxic horses, trotting then stopping suddenly may allow better assessment of limb placement (proprioception)
- walk horse in serpentine or figure of 8
- walk horse with head elevated
- walk horse while pulling tail in each direction
- spin horse in tight circles
- walk horse on uneven terrain (back and forth over curb, up and down hill).

If the horse appears ataxic, use the gait characteristics to decide on the “type” of ataxia it is displaying: cerebellar, vestibular, or spinal (general proprioceptive). Also decide if the horse demonstrates paresis, defined as deficiency in the generation of gait or ability to support weight, which can be characterized as upper motor neuron (UMN) or lower motor neuron (LMN).

**Gait characteristics of:**

**Cerebellar disease:**

- Hypermetria, exacerbated by lifting the head
- Gait is quite spastic, similar to cervical spinal ataxia
- Strength is maintained with cerebellar disease (there is no paresis as would be seen with cervical spinal ataxia)
- Look also for head tremors, loss of menace response, and a wide-based stance to support a diagnosis of cerebellar disease.
**Vestibular disease:**

- Head tilt and loss of balance with a tendency to lean or fall to one side.
- Recumbent horses often have a strong preference to lie on the affected side.
- Ambulatory horses with peripheral vestibular disease tend to have
  - wide-based stance and are hesitant to walk but show no proprioceptive deficits.
  - Facial nerve deficits may be evident on the ipsilateral side.
- Ambulatory horses with central vestibular disease often show
  - Proprioceptive deficits and paresis
  - Obtundation (due to interference with the ascending reticular activating system),
  - Multiple cranial nerve deficits (facial nerve deficits, dysphagia, etc.).
- In the acute stage of vestibular disease (first 12-24 hours) horses often show nystagmus, which may then disappear.

**Spinal lesions:**

- Proprioceptive deficits
  - Toe scuffing/dragging
  - Delayed protraction
  - Knuckling
  - Crossing over
  - Stepping on itself
  - Pivoting or circumduction when circling
  - Uneven/irregular stride length
- **Upper motor neuron** lesions
  - Spastic gait
  - Will usually have characteristics of proprioceptive deficit (toe scuffing/dragging, delayed protraction etc.)
- **Lower motor neuron** lesions
  - **Characterised by flaccid paralysis/severe paresis**
  - Short, choppy stride
  - Base-narrow stance and muscle tremors
  - Pronounced muscle atrophy often accompanies lower motor neuron lesions.
**Upper vs Lower motor neuron dysfunction: clinical characteristics**

<table>
<thead>
<tr>
<th>LMN</th>
<th>UMN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flaccid</td>
<td>Spastic</td>
</tr>
<tr>
<td>Decreased tone</td>
<td>Increased tone</td>
</tr>
<tr>
<td>Reduced reflex response</td>
<td>Exaggerated reflex response</td>
</tr>
<tr>
<td>Profound muscle atrophy</td>
<td>Minimal muscle atrophy</td>
</tr>
<tr>
<td>Fasciculations</td>
<td>No Fasciculations</td>
</tr>
</tbody>
</table>

**Spinal cord disease: localisation to a segment**

<table>
<thead>
<tr>
<th>Spinal Segment</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical C1-C5</td>
<td>Spastic gait, proprioceptive deficits, paresis <strong>worse in hind limbs</strong> +/- Horner’s syndrome</td>
</tr>
<tr>
<td>Cervico-thoracic C6-T2</td>
<td>Proprioceptive deficits <strong>may be worse in forelimbs.</strong> Spastic gait in hind. Paresis &amp; muscle atrophy in forelimbs. +/- Horner’s</td>
</tr>
<tr>
<td>Thoracolumbar T3-L3</td>
<td>Normal forelimbs. Proprioceptive deficits, spasticity and paresis in <strong>hind limbs.</strong></td>
</tr>
<tr>
<td>Lumbosacral L4-S5 + cauda equina</td>
<td>Urinary incontinence, fecal retention, reduced sensation perineal region. Variable degrees of <strong>hind limb</strong> paresis &amp; proprioceptive deficits</td>
</tr>
<tr>
<td>Coccygeal</td>
<td>Reduced tail tone. Normal fore &amp; hind limbs</td>
</tr>
</tbody>
</table>
### Grading ataxia

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>Neurological deficits only just detected at normal gait. Deficits worsened by backing, tight circling, head elevation. Generally not detected by layperson.</td>
</tr>
<tr>
<td>2</td>
<td>Neurological deficits easily detected at walk (by clinician). Usually detected by layperson. Deficits worsened by backing, tight circling, head elevation.</td>
</tr>
<tr>
<td>3</td>
<td>Neurological deficits prominent at walk, may be postural deficits at rest. Usually detected by layperson. Tendency to buckle or fall during backing, tight circling, head elevation.</td>
</tr>
<tr>
<td>4</td>
<td>Severe neurological deficits, with spontaneous stumbling and falling at the walk</td>
</tr>
<tr>
<td>5</td>
<td>Recumbent and unable to rise</td>
</tr>
</tbody>
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### 4. Spinal reflexes and muscle evaluation (tone and size)

Tendon and withdrawal reflexes are not usually performed in ambulatory horses. Reflexes that the practitioner should check in the standing horse include cervicofacial/auricular and cutaneous trunci (panniculus). Examine the horse carefully for any muscle atrophy. Check tail tone and anal tone. Look for any loss of skin sensation (hypalgesia or analgesia), increased sensitivity (hyperesthesia), or abnormal sweating (sympathetic denervation).
Neuroanatomical localisation summary

<table>
<thead>
<tr>
<th>Anatomical Region</th>
<th>Predominant Clinical Signs</th>
</tr>
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<tbody>
<tr>
<td>Cerebrum</td>
<td>Altered mentation, seizures, blindness</td>
</tr>
<tr>
<td>Brain Stem</td>
<td>Multiple CN deficits, ataxia, paresis</td>
</tr>
<tr>
<td>Vestibular System</td>
<td>Head tilt, nystagmus, ataxia, postural deficits</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>Intention tremors, wide-based stance, hypermetria</td>
</tr>
<tr>
<td>Spinal cord UMN</td>
<td>Ataxia, dysmetria, spasticity, paresis (+ associated proprioceptive deficits)</td>
</tr>
<tr>
<td>Spinal cord LMN (or PN)</td>
<td>Flaccid paresis, ataxia, muscle atrophy (focal with peripheral nerve)</td>
</tr>
</tbody>
</table>

Ancillary Tests

Cerebrospinal fluid (CSF) collection:
- LS space (standing)
- AO space (GA)

Normal CSF characteristics:
- Leukocytes < 5/uL
  - All mononuclear
- RBCs < 50/uL
- Protein < 100 mg/dl

Diagnostic imaging:
- Plain radiography
  (head, neck, lumbosacral region in adults)
• Myelography (C1-T1 only in horses, generally only lateral-medial possible)
• MRI
• CT (head & cr neck)
• Scintigraphy

**Cerebral dysfunction**

Important to first consider intra- vs extra-cranial causes

• **Extra-cranial causes**
  – Hepatic encephalopathy
    • PA toxicity & fatty liver (ponies) most common
  – Intestinal hyperammonemia
  – Severe hyponatremia
    • diarrhea
  – Uraemic encephalopathy (rare)

• **Intra-cranial causes**
  – Viral encephalitis
    • Hendra virus
    • Japanese encephalitis
    • EEE, VEE, WEE (exotic)
    • West Nile Virus (exotic)
    • Rabies (exotic)
    • EHV 1 (usually lumbosacral syndrome)
  – Trauma
  – Abscess (S Equi, R Equi)
  – Neonatal encephalopathy
  – Neoplasia
  – Verminous encephalomyelitis
  – Toxic
    • Mouldy corn, Swainsona etc.
EPM (exotic)

Diagnostics

- CBC & chemistry
  - Sodium
  - Liver function
    - Bile acids, ammonia, bilirubin (= function)
    - GGT, AST, SDH/IDH
  - Signs of inflammation?
  - Hendra virus PCR
  - Serum titres – EHV & EEE, VEE, WEE, WNV

- CSF
  - Lymphocytic pleocytosis common with viral causes
  - Increased protein also indicative of inflammation within CNS
  - Usually normal with extra-cranial causes
  - Submit also for:
    - Antibody titres for viral encephalitidies
    - Culture

- Imaging
  - Radiography/CT
    - Skull fractures
  - MRI
    - Brain imaging

Treatment

- Depends on inciting cause – see relevant notes

For head trauma:

- Anti-inflammatory
  - Corticosteroids – MPSS acutely
  - DMSO?
- NSAIDs - analgesia
  - Mannitol (not if active intracranial haemorrhage)
  - Anticonvulsant therapy
  - Broad spectrum antibiotics (esp neonates!)
    - Sinus communication
    - Gut. Pouch communication

**Prognosis**
- Difficult to maintain adult horses with severe deficits for prolonged periods
- Safety a concern
  - During treatment
  - Upon recovery if persistent deficits
- Response to treatment best guide
- MRI may provide better information

**Approach to the horse with seizures/collapse**
- True seizures
  - Rare in adults
    - Same DDx as more consistent cerebral dysfunction if acute
    - Recurrent seizures – “cryptogenic epilepsy”
  - Foals
    - Benign juvenile epilepsy (Arabians)
    - Neonatal encephalopathy
- Differentiate from other causes of collapse
  - CV- syncope
  - Narcolepsy
  - Cataplexy
  - Sleep deprivation
  - HYPP
• Additional diagnostic tests
  – Additional tests
  – Holter ECG
  – EEG
  – Video monitoring
• Treatment of true seizures
  – Immediate management
    • Diazepam, midazolam
    • Phenobarbital
    • Alpha-2 agonists
  – Maintenance
    • PO phenobarbital
    • Potassium bromide

Cervical Stenotic Myelopathy (CSM) = “Wobbler” syndrome

• = CVM (cervical vertebral malformation)
• Focal compression of the spinal cord caused by disturbances of growth &/or articulation of cervical vertebrae
• Young, fast-growing horses affected (often males)
• Thoroughbreds commonly affected
• Common cause of ataxia in horses
• Form of developmental orthopaedic disease
• There may be a genetic predisposition
• Mineral imbalance?
  • Copper supplementation of juveniles may reduce incidence
• Overfeeding, faulty bone metabolism?
• Usually no pain on manipulation of neck & palpation of cervical vertebrae
• Clinical diagnosis often accurate
• Usually no changes on CSF (may be mild inflammatory response)
• Young horses <3 years
  • Often C3-6 compression
  • Often dynamic compression
• Older horses: >10 years
  • DJD dorsal articular facets (C5-6, C6-7)
  • Usually static compression
• Differential diagnosis for (cervical) spinal cord disease
  • Trauma, fracture
  • EHV1
  • EDM (equine degenerative myelopathy)
  • Verminous myelitis
  • Discospondylitis
  • Neoplasia
  • Equine Protozoal Myelitis (EPM) (exotic)
• Diagnostic tests
  • Plain radiography
    • Can appreciate narrowing of vertebral canal, but not stenosis of the cord
    • Sagittal ratios <50% suggestive
    • Rule out fracture
    • Detect osteoarthritis
    • Detect misalignment (care with interpretation)
  • Myelography (C1-T1)
    • Narrowing of dorsal and ventral dye columns at opposed site confirms cord compression
    • Requires general anaesthesia and AO puncture
    • CSF sample taken simultaneously
• Treatment
  • Signs usually do not resolve, and often progress
- Euthanasia for severely affected horses
- Medical therapy
  - NSAIDS, DMSO, corticosteroids
  - Rest
  - restrict feed
  - Unlikely to improve, though may halt progression in young (<12 months)
- Surgical therapy – stabilisation of vertebrae or decompression
  - May improve 1 grade
  - Not often performed in Australia

**Verminous myelitis/encephalitis**

- Caused by aberrant migration of parasitic worm larvae through the CNS
- Any age can be affected
- In Australia *Strongylus vulgaris* seems most common species involved
- Larvae cause irreparable damage
- History
  - Sudden onset of signs
  - Often good worming history
- Clinical signs
  - Can affect any part of CNS – therefore variable, though usually progressive
  - May be asymmetrical
- Diagnosis
  - Can be difficult antemortem
  - CSF analysis may reveal an increased total cell count, with increased eosinophils and or neutrophils, increased protein and xanthochromia
- Treatment:
  - Poor prognosis for recovery
  - Supportive therapy & anti-inflammatories indicated
• Anthelmenthics? – larvae are often long gone on histopathology sections. Killing them in situ may cause more damage

Equine Herpesvirus Myeloencephalopathy

• **EHV-1** – causes a vasculitis within the neurological system
• All ages can be affected
• Outbreaks in winter/early spring
  – Crowded areas – track/breeding farms
• Neurological EHV1 is **NOTIFIABLE**
• EHV-1 most often associated with respiratory disease & abortion
• Clinical signs
  – Progressive symmetrical ataxia & tetraparesis
    • Worse in hind limbs
  – May be severe hind limb weakness/paralysis in some cases
    • “dog sitting”
  – Urinary incontinence (Spastic, UMN bladder)
  – faecal retention
  – recumbency
  – FEVER
  – Generally do not progress after 48 h
  – Vestibular signs occasionally
• **DDx**
  – Same as CSM for tetraparesis & ataxia
  – Other things to consider for lumbosacral syndrome
    • Trauma
    • Polyneuritis equi
    • Aortoiliac thrombosis
• Diagnosis
  – Clinical signs
  – EHV-1 viral PCR
    • Blood (buffy coat, EDTA tube)
    • Nasopharyngeal swabs
  – CSF analysis –
    • XANTHOCHROMIA
    • Increased protein
  – Paired EHV1 serologic titres

• Treatment:
  – Supportive nursing care
  – Anti-inflammatory medication
    • Corticosteroids
    • Aspirin
  – Antiviral therapy – acyclovir may be beneficial
  – Improvement after 48 hours
  – May require weeks-months for full recovery
  – Some horses have permanent deficits

Equine Protozoal Myeloencephalitis (EPM)
  – Exotic, though important differential for neurologic disease in horses imported from North America
    • Recrudescence common
    • Unclear incubation period
  – Caused by Sarcocytis neurona
  – Less commonly Neospora spp.
  – Can cause lesions anywhere in CNS
  – Treatment with antiprotozoal drugs may be successful (Ponazuril & others) although horse that recover may be left with some permanent deficits
Botulism

- Horses most sensitive species of all to *Clostridium botulinum* toxin
- Poisoning circumstances in horses:
  - Adults
    - Ingestion of preformed toxin in feed
  - Foals
    - Toxico-infectious (proliferation in GIT)
- Botulinum toxin:
  - Inhibits Ach release at motor end plates of NMJ, causing:
    - Progressive symmetrical tetraparesis
    - Loss of tongue tone, eyelid tone, anal tone
    - Reduced GIT motility
    - May progress to respiratory paralysis & death
- DDx
  - EHV, ionophore toxicosis, EMND
  - other causes of dysphagia
- Diagnosis
  - Demonstration of toxin in horse rarely possible
  - May be able to demonstrate spores or toxin in feed source
  - Clinical signs: botulism generally a clinical diagnosis
- Treatment
  - Nursing care
  - Tube feeding may be necessary
  - Multivalent antitoxin stops progression ($$)
    - Currently not available in Australia
  - Ventilation in foals – can do well even if severe & recumbent ($$)
- Prognosis
Equine motor neuron disease (EMND)

- Associated with lack of dietary vit E intake
  - Oxidant stress
  - Motor neuron body & axonal degeneration
- Clinical signs reflect denervation of skeletal muscles
  - Weakness & atrophy
  - Acute onset (rarely insidious)
  - Fasciculations, “horse on a ball” stance
  - Reluctant to stand still
- Diagnosis
  - *Sacrocaudalis dorsalis medialis* muscle biopsy
  - Serum vit E levels may be low
  - EMG
- Treatment
  - Vit E supplementation
  - 40% recover, 40% mortality
  - 20% chronic disease

Tetanus

- *Cl tetani* wound infection
  - Anaerobic environment – proliferation, death & liberation of toxins
- Tetanospasmin travels to the CNS via the hemolymphatic system and via peripheral motor nerves.
- The toxin exerts its effect on presynaptic inhibitory interneurons in the ventral horn of the spinal cord.
• Differential diagnosis
  – Laminitis
  – Hypocalcaemia (check plasma iCa2+)
  – Rhabdomyolysis
  – Rabies
  – Myotonia (v rare!)
• Diagnosis
  – Clinical syndrome
  – Rule out hypocalcaemia
  – Growth of Cl tetani from wound site (only useful in retrospect)
• Treatment
  – **Tetanus antitoxin**: 100-200 U/kg IV or IM (single dose) will bind circulating toxin.
    • Consider intrathecal administration of 50 ml TAT (20-30 ml in foals)
  – **Penicillin G** (potassium or sodium): 22000-44000 IU/kg IV q6h for 7-10 days
  – Muscle relaxation / sedation
    • Acepromazine
    • Phenobarbital
  – Local infiltration of the wound with procaine penicillin and/or tetanus antitoxin (3000-9000 IU).
  – Vaccination with tetanus toxoid
    • **clinical disease does not result in a sufficient immune response.** Use separate injection site to antitoxin.
• Nursing Care
  • Confine to a quiet, dark stall with deep bedding.
  • Minimize auditory stimulation with ear plugs.
  • Padded walls and/or a padded helmet to minimise injury.
  • Frequent turning of recumbent horses (q 2-4 hours)
  • Recumbent horses that are unable to rise may benefit from slinging.
  • Manual rectal evacuation and/or urinary catheterisation may be necessary
• Expected Course and Prognosis
- Horses that are recumbent and unable to rise have a grave prognosis
- Horses that retain the ability to stand and ambulate have a fair prognosis.
- The clinical signs may persist for weeks, however survivors will generally stabilise after 7 days and begin to show improvement after 2 weeks.
- Recovery may take as long as 6 weeks, but is usually complete
- The attitude of the individual horse affects outcome.
- The overall mortality rate in horses is reported to be 50-80%.

**Prevention/Avoidance**

- Initial vaccination with 2 doses of tetanus toxoid 3-4 weeks apart.
- Annual toxoid booster thereafter
- Tetanus toxoid should be administered in the case of a wound if there has not been vaccination within the last 6 months
- Pregnant mares should be given a toxoid booster 4-6 weeks prior to expected parturition

**Peripheral neuropathies**

**Stringhalt**
- Classical stringhalt – aetiology unknown
- Australian stringhalt – associated with grazing pastures contaminated with *Hypochaeris radicata* (Cat’s ear), Dandelions, & other species
- Unilateral or bilateral uncontrollable flexion of the hindlimb(s) occurs due to an axonopathy of long large-diameter nerves
- Mild cases need to be differentiated from upward fixation of the patella
- Left laryngeal hemiplegia can be present
- Cases can spontaneously resolve – may take weeks to a year
- Anti-inflammatory therapy, phenytoin & surgery to transect lateral extensors have been used with variable success
Approach to the horse with vestibular (VIII) and/or facial (VII) cranial nerve dysfunction

- Facial & vestibular often occur together
  - Proximity of nuclei in brainstem
  - Nerves run together in petrous temporal bone
  - If dysphagia, suggestive of CN IX, X, XII
- DDx
  - Brainstem
    - Abscess
    - Neoplasia
    - Verminous myelitis
    - EPM (exotic)
  - Peripheral
    - Trauma
    - Temperohyoid osteoarthropathy
- Diagnostics
  - CBC & Chem WNL
  - Endoscopy
  - Radiography
- Treatment and prognosis
  - Dependent on cause

Temperohyoid osteoarthropathy

- Inflammation and degeneration of the temporohyoid joint
- Bony proliferation of the tympanic bulla, stylohyoid and petrous temporal bones

- Etiology?
  - Otitis media/interna extension - infection
  - DJD

- Presentation
  - Clinical signs of VII, VIII dysfunction +/- IX & X
- Age of onset: 10.8 ± 5.5 years
- Uni- or Bi-lateral
- Breed predisposition:
  - 51% QH
  - 18% TB
- Pathogenesis
  - Abnormal proliferation of bone
  - Fusion of temporohyoid joint
  - Pathologic fracture due to normal motion of tongue and larynx
    - Petrous, basisphenoid, occipital, stylohyoid
    - NG tube placement may precipitate
  - Acute neurologic dysfunction
- Treatment
  - Medical
    - Prolonged Antimicrobial > 30 days
    - NSAIDs
    - Corticosteroids
  - Surgical
    - Partial stylohyoidostectomy
    - Ceratohyoidectomy

Further reading: