# Neurological case studies

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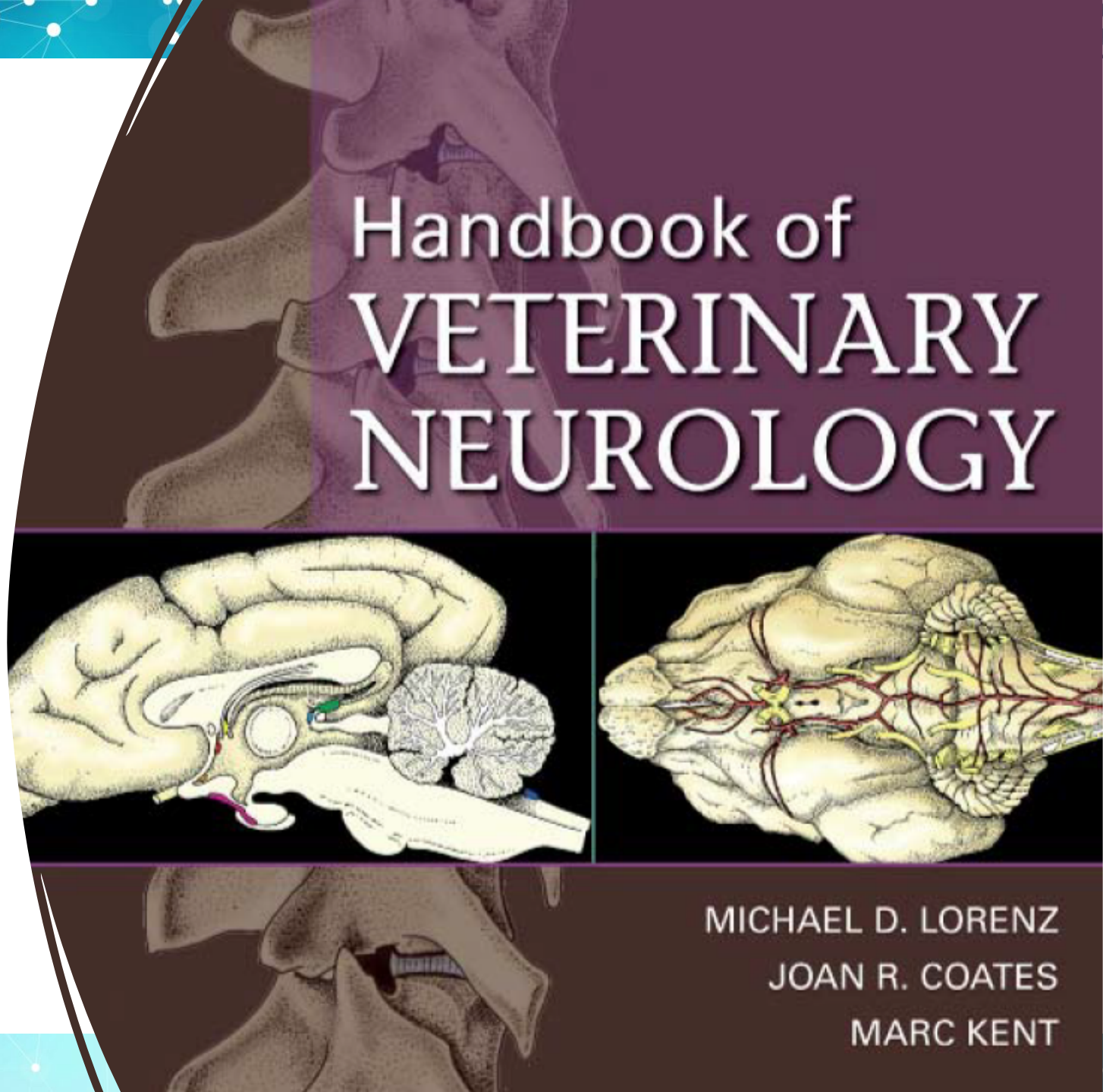
SAVA JHB May 8 2024 Webinar

# Acknowledgements


- Tomas Elvira

# Approach to the neurological (?) problem

- The minimum database
  - Minimise
  - Prioritise
  - Rare diseases last
- Screening neuro exam – is it neuro?
  - Eg syncope vs seizure; weakness vs paresis

# How to identify a problem as neurological:

- Collect **minimum database**
- Identify **problems**
- Identify one or more problems related to **nervous system**
- **Localize** level of lesion
- Estimate **extent** of lesion within that level
- List **most probable causes** (rule-outs or differential diagnosis)
- Construct **diagnostic plan** to determine cause or pathologic
- injury
- Determine **prognosis** with and without therapy

# The minimum database for a NEUROLOGICAL problem

- History
- Physical examination
- Neurologic examination
- Clinical pathology:
  - Complete blood count
  - Urinalysis
  - Chemistry profile
  - Electrolytes
  - Creatine kinase activity

# Signs, time of progression

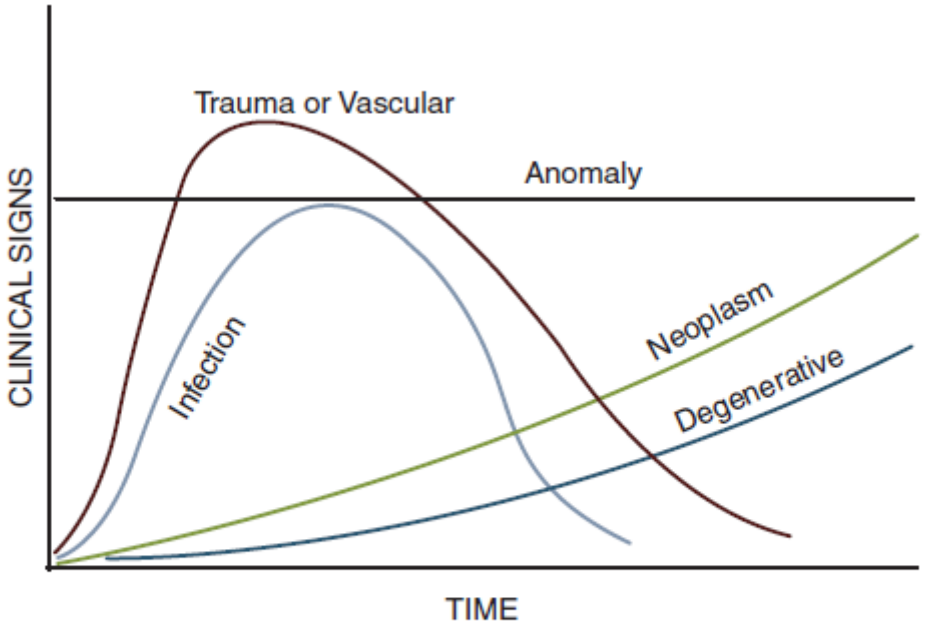
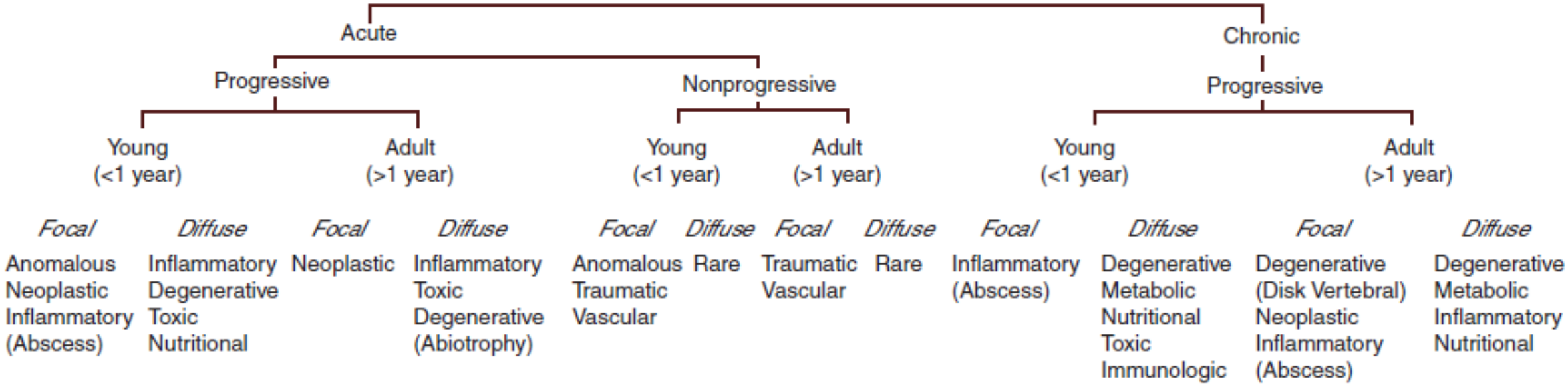



**Figure 1-1** Sign-time graph of neurologic diseases. Progression of metabolic, nutritional, and toxic diseases is variable, depending on the cause.

# Classification of neurological diseases in order of frequency

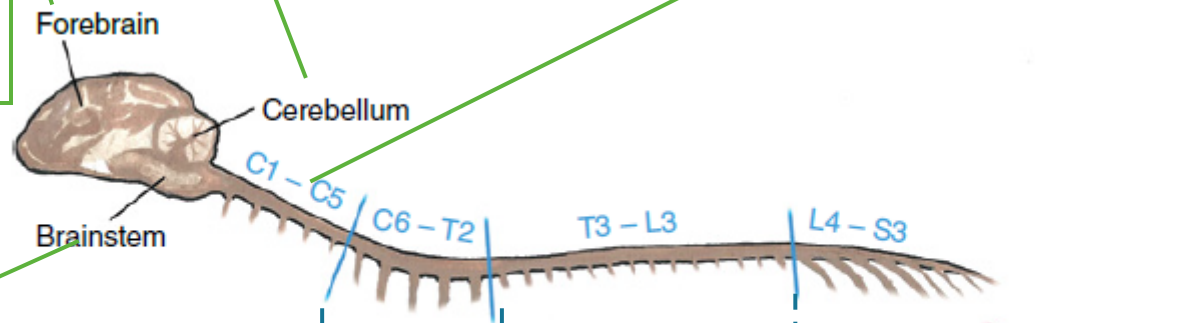


# Neuroanatomical regions & typical signs

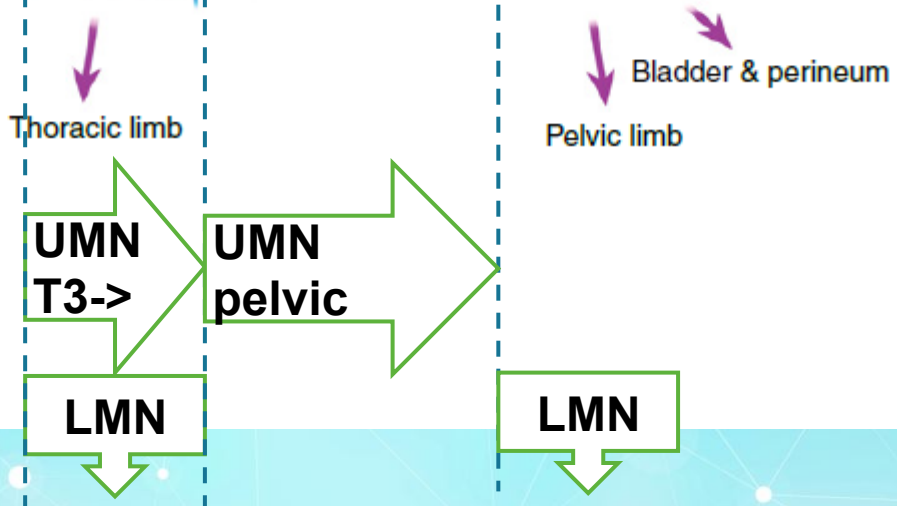
**Prosencephalon**
Telencephalon = Cerebral hemispheres +
Diencephalon = Hypothalamus + Thalamus
**CX**: Behaviour change, central blindness, seizures

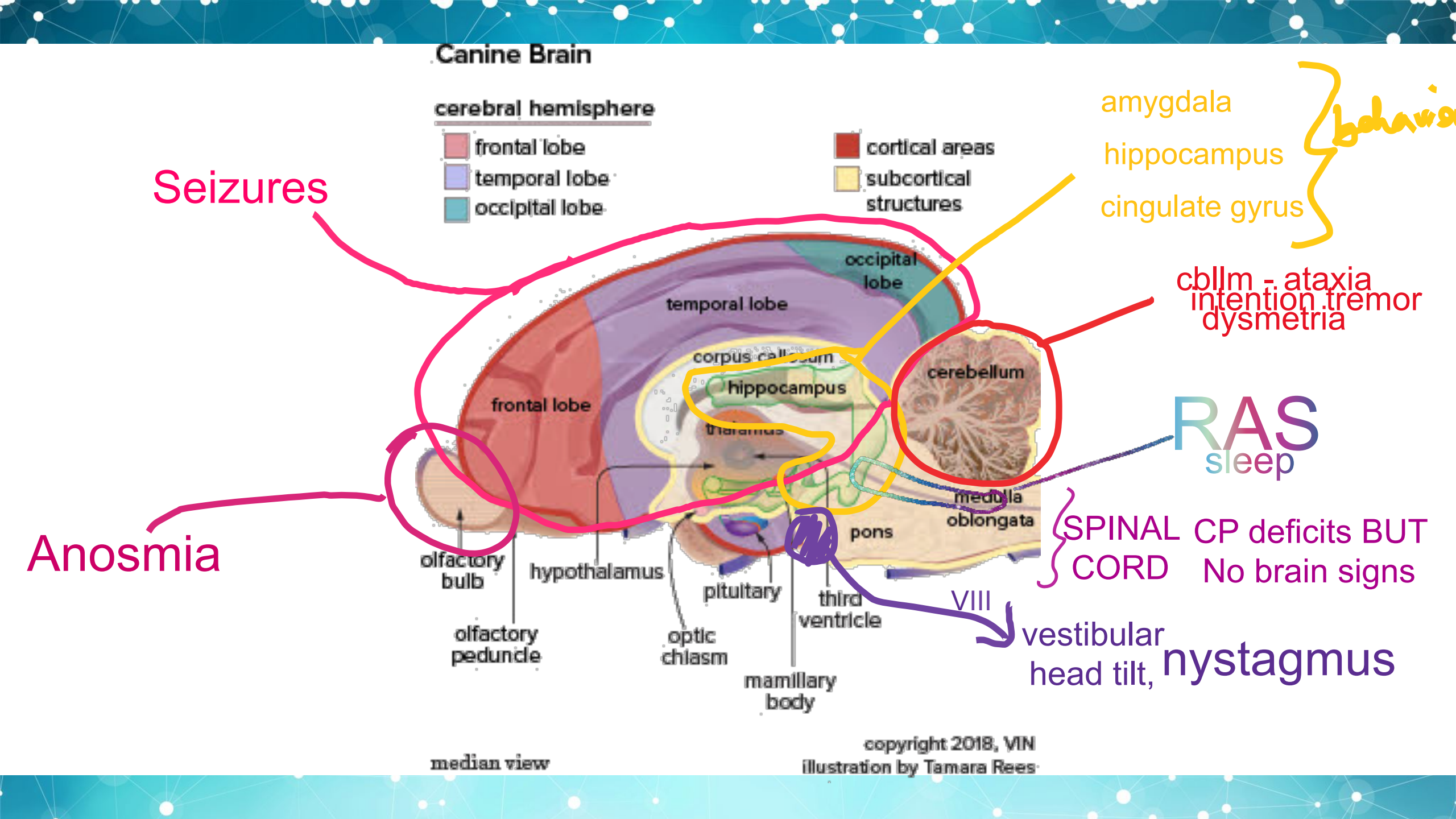
**Cerebellum** (dorsal metencephalon)
**CX:** hypermetria, intention tremor

**C1-C5**
Only UMN
All 4 limbs, lateralised




**Midbrain** (mesencephalon) +
**Pons** (ventral metencephalon) +
**Medulla** (myelencephalon)
**CX**: proprioceptive deficits, ataxia, cranial nerve and vestibular signs, mentation (RAS)




Canine Brain
cerebral hemisphere
frontal lobe
temporal lobe
occipital lobe
cortical areas
subcortical structures
Seizures
amygdala
hippocampus
cingulate gyrus
behavior
cbllm - ataxia
intention tremor
dysmetria
occipital lobe
temporal lobe
corpus callosum
hippocampus
thalamus
frontal lobe
cerebellum
RAS
sleep
medulla oblongata
pons
Anosmia
SPINAL CORD
CP deficits BUT
No brain signs
olfactory bulb
hypothalamus
pituitary
third ventricle
VIII
olfactory peduncle
optic chiasm
mamillary body
vestibular
head tilt,
nystagmus
median view
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# Case #1

Young male neutered cat

Acute onset, then slowly progressive over few days

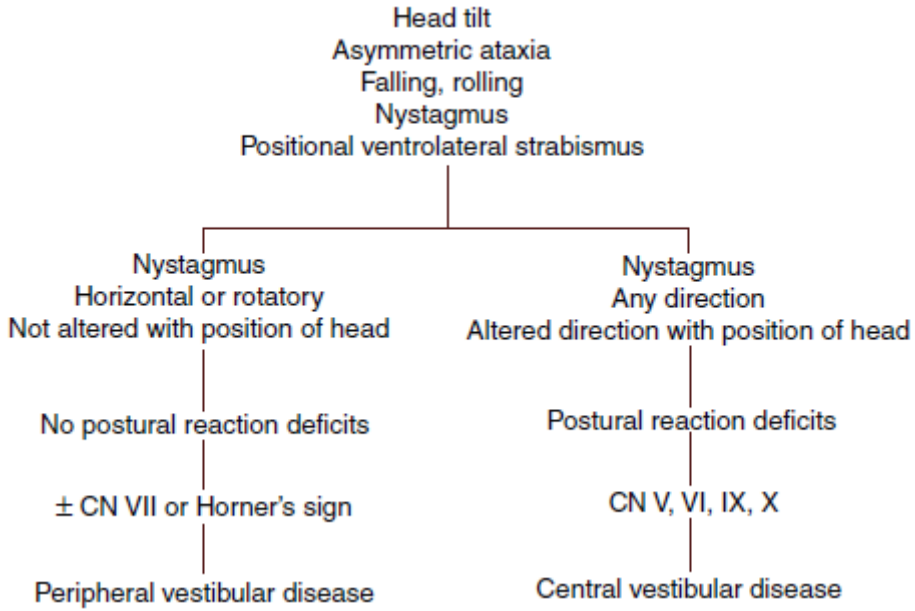
Characterise – general neurolocalisation – DDx – MDB?

# What is the problem?

- Ataxia
  - What kind?
    - Spinal?
    - Cerebellar?
    - Vestibular?
  - Why?
- Vestibular ataxia, because of head tilt
- DDx?
  - Otitis media
  - Trauma
- Tests?
  - Cranial nerves
  - Otoscopy

# Vestibular disease – the hallmarks

| Central (cranial medulla) | | Peripheral |
|---|---|---|
| Normal – depressed | **Mentation** | Normal |
| Head tilt, falling (us. Toward lesion) | **Posture** | Head tilt, **circling**, rolling, falling |
| Ipsilateral hemiparesis<br>Vestibular ataxia | **Movement** | **No paresis**<br>SEVERE vestibular ataxia |
| Ipsilateral **deficits** | **Postural reactions** | Normal |
| Cr N VIII (+/- **V,** VII, nystagmus) | **Cranial nerves** | Cr N VIII (+/- VII, nystagmus, Horner's) |


Head tilt
Asymmetric ataxia
Falling, rolling
Nystagmus
Positional ventrolateral strabismus
Nystagmus
Horizontal or rotatory
Not altered with position of head
No postural reaction deficits
± CN VII or Horner's sign
Peripheral vestibular disease
Nystagmus
Any direction
Altered direction with position of head
Postural reaction deficits
CN V, VI, IX, X
Central vestibular disease

# Case #2

Middle aged rottweiler

Chronic, progressive over weeks


(c) Anthony Zambelli 2011

# What is the problem?

- Ataxia
  - What kind?
    - Spinal?
    - Cerebellar?
    - Vestibular?
  - Why?
- Spinal ataxia – no head tilt, no intention tremor or hypermetria
- DDx?
- Tests?

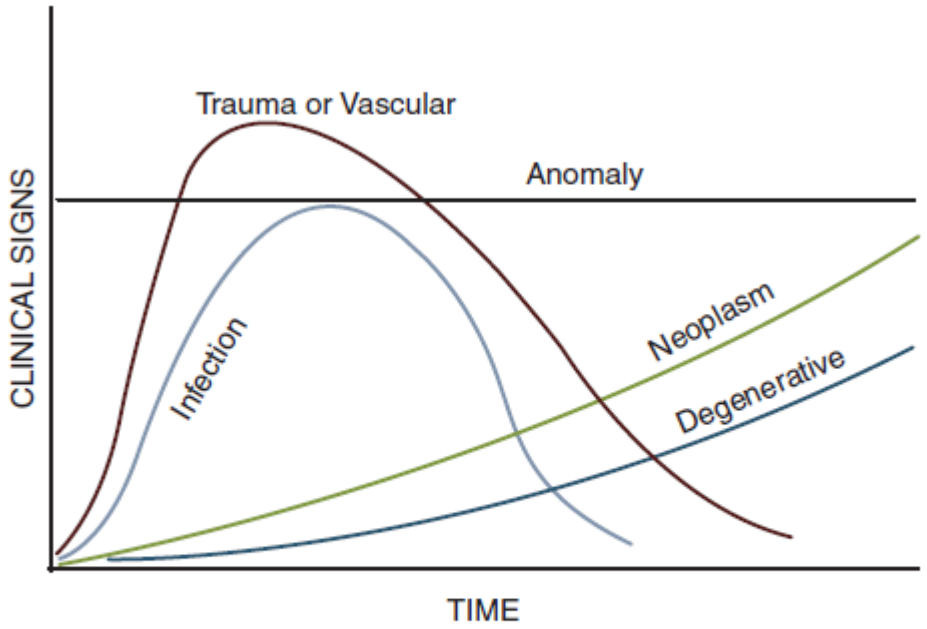
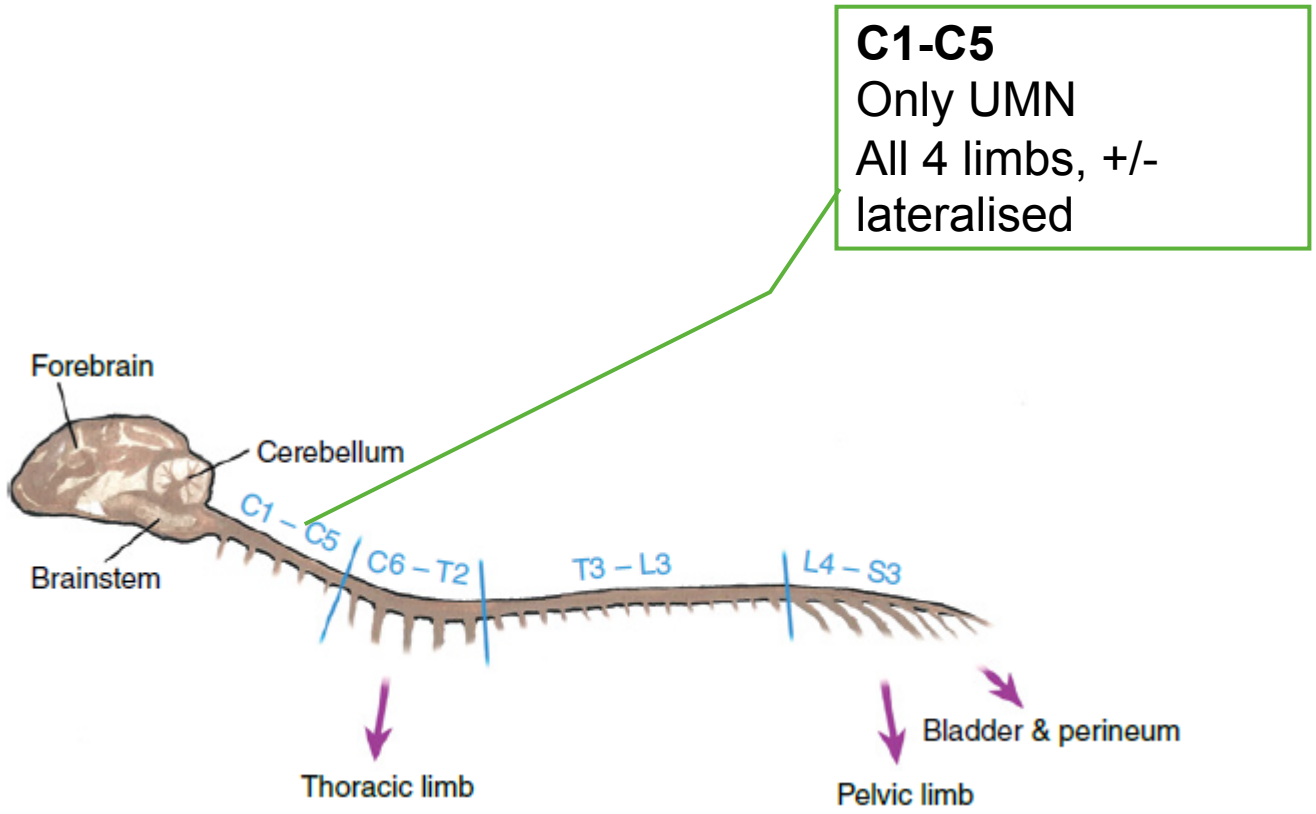
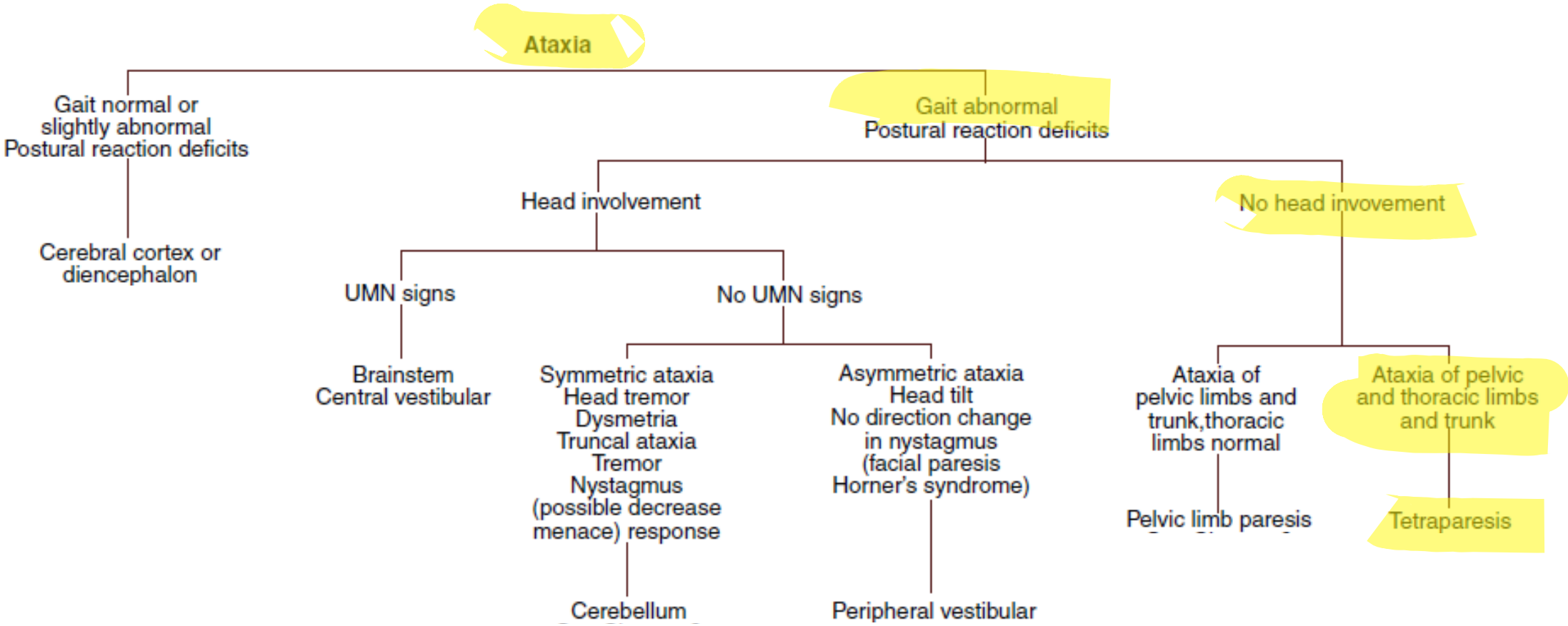



Figure 1-1 Sign-time graph of neurologic diseases. Progression of metabolic, nutritional, and toxic diseases is variable, depending on the cause.

# Where is the lesion?

- Lateralising?
  - No
- Fore + Hind or only Hind?
  - All 4
  - Cranial to C6 (C1-C5)




Ataxia
Gait normal or slightly abnormal Postural reaction deficits
Cerebral cortex or diencephalon
Gait abnormal Postural reaction deficits
Head involvement
UMN signs
Brainstem Central vestibular
No UMN signs
Symmetric ataxia Head tremor Dysmetria Truncal ataxia Tremor Nystagmus (possible decrease menace) response
Cerebellum
Asymmetric ataxia Head tilt No direction change in nystagmus (facial paresis Horner's syndrome)
Peripheral vestibular
No head invovement
Ataxia of pelvic limbs and trunk,thoracic limbs normal
Pelvic limb paresis
Ataxia of pelvic and thoracic limbs and trunk
Tetraparesis

# Brain vs forebrain vs spine

- Brainstem coordinates, forebrain only reinforces
- Forebrain = altered mental status/seizures/blindness
  - but normal gait once walking
  - Contralateral postural deficits = NOT paresis

*Small Animal C1-T2 Spinal Cord Diseases: Differential Diagnosis Based on Clinical Course and Etiologic Categories**

| Etiologic Category | Acute Nonprogressive | Acute Progressive | Chronic Progressive |
|---|---|---|---|
| Degenerative | None | Hansen type I IVDD (6,7)<br>Ascending descending myelomalacia (6)<br>Myelinolysis (6) | Hansen type II IVDD (6,7)<br>Cervical spondylomyelopathy (7)<br>Spondylosis deformans (6)<br>Neuronopathies (7)<br>Myelinolysis (6)<br>Dysmyelinating diseases (7)<br>Axonopathies (7)<br>Lysosomal storage diseases (8,15)<br>Extradural synovial cysts (7) |
| Anomalous | None | None | Spinal cord malformation (6)<br>Vertebral column malformation (6)<br>Atlantoaxial subluxation (7)<br>Atlantooccipital malformation (7)<br>Chiari-like malformation (7)<br>Intra-arachnoid cyst/diverticulum (6) |
| Neoplastic (6) | None | Primary<br>Hematopoietic<br>Skeletal<br>Metastatic | Primary<br>Hematopoietic<br>Skeletal<br>Metastatic |
| Nutritional | None | None | Hypervitaminosis A (cats) (15) |
| Inflammatory | None | Distemper myelitis (15)<br>Bacterial myelitis (15)<br>Rickettsial myelitis (15)<br>Protozoal myelitis (15)<br>Mycotic myelitis (15)<br>Diskospondylitis (6)<br>Epidural empyema (6)<br>Vertebral physitis (6) | Feline infectious peritonitis (15)<br>Granulomatous meningoencephalomyelitis (15)<br>Steroid-responsive meningitis arteritis (15) |
| Traumatic | Fractures (6)<br>Luxations (6)<br>Contusions (6)<br>Hansen type III disk extrusion (6) | Ascending descending myelomalacia (6) | None |
| Vascular | Fibrocartilaginous embolism (6,7)<br>Vascular malformations (7)<br>Embolic myelopathy (6) | None | None |

# Tests?

- History
- Imaging
  - Radiographs
  - CT / MRI
- Nerve/muscle biopsy
- T4, glucose, cortisol assay

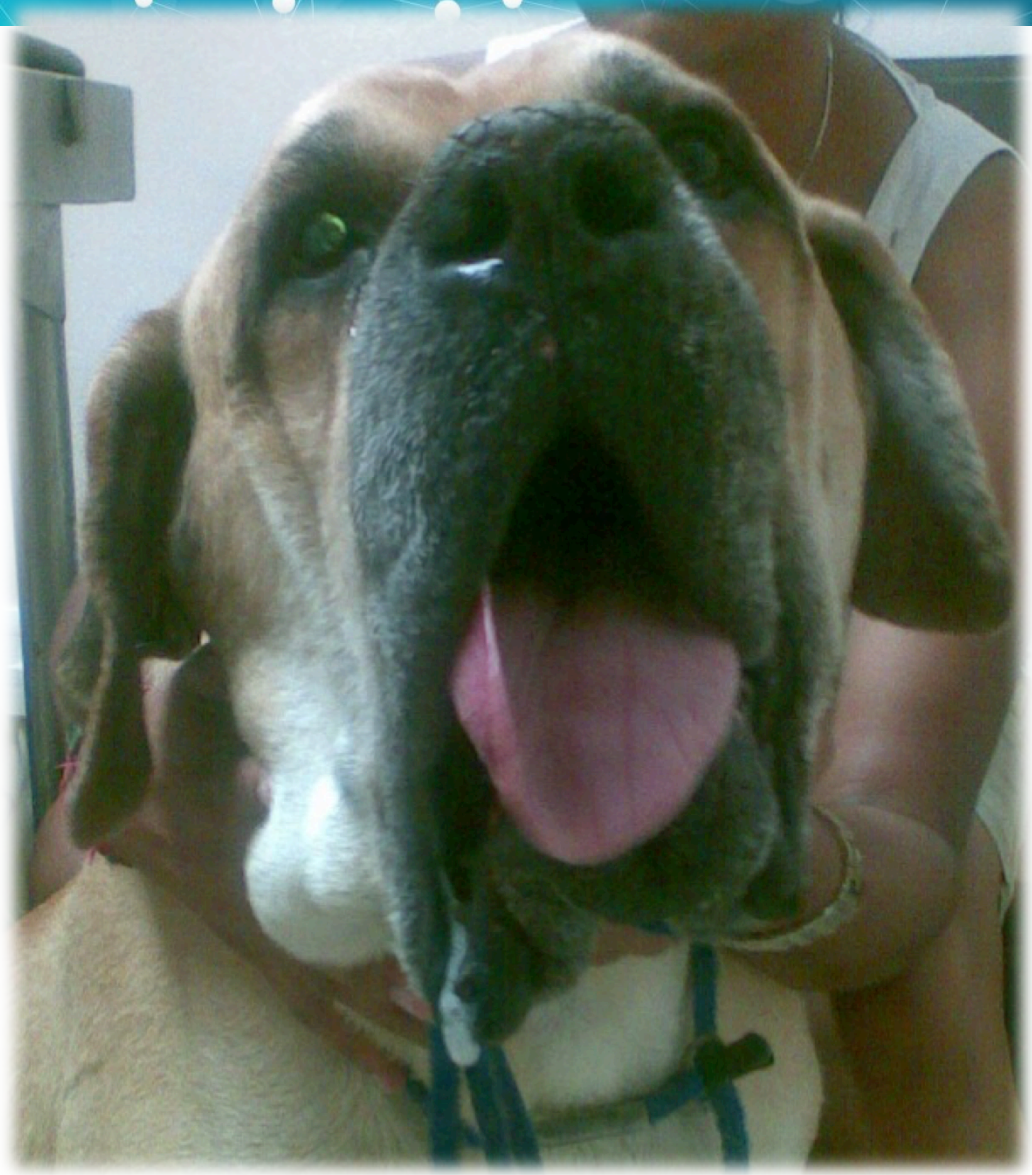
- Results
  - History of organochlorine tick dip exposure (sold by vet) 4-5m prior
  - Demyelination of spinal axons

# Case #3

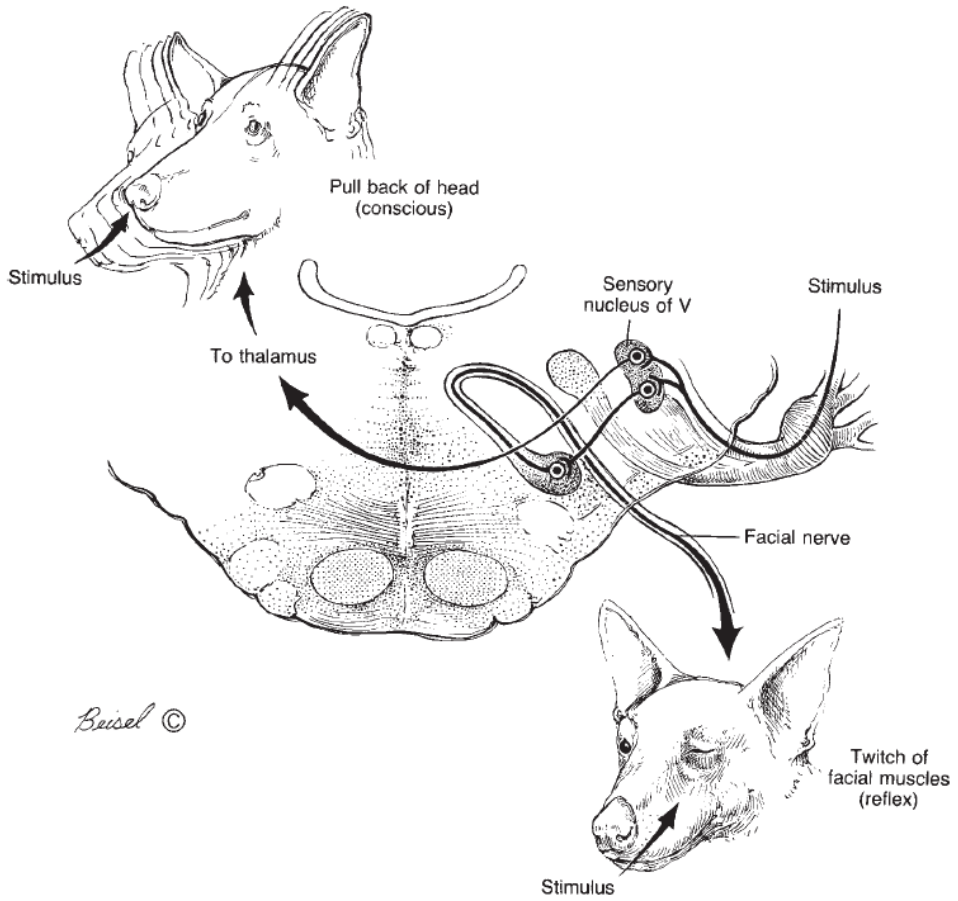
# Presenting signs

- Panting a lot
- Lower lips sagging
- No pupillary response
- All else WNL on Physical

- Next step?



# Was there a muscle response to stimulation?




Not on the right, reduced on the left

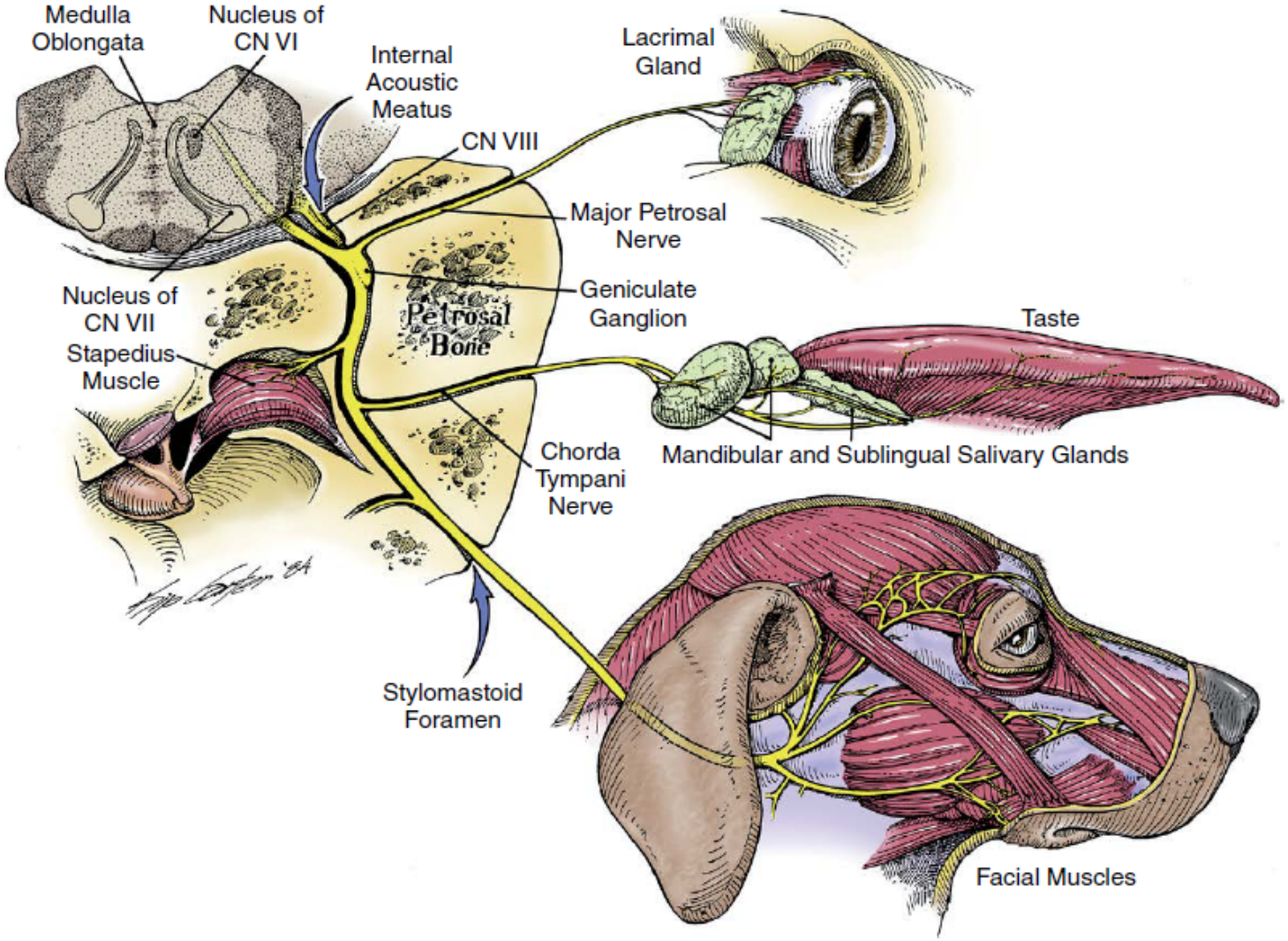
FACIAL NERVE

Dropped jaw

TRIGEMINAL NERVE

# Was there facial muscle tone?

- Not on the right



# Neuro exam and bloods

- Neuro
  - Oculomotor (III), Trigeminal (V) AND Facial (VII) paralysis
- Chem abnormalities
  - ALT 577
  - ALP 554
  - GGT 43
  - CRP 4.0
  - Cholesterol 9.9
- What else?

# T4, TSH

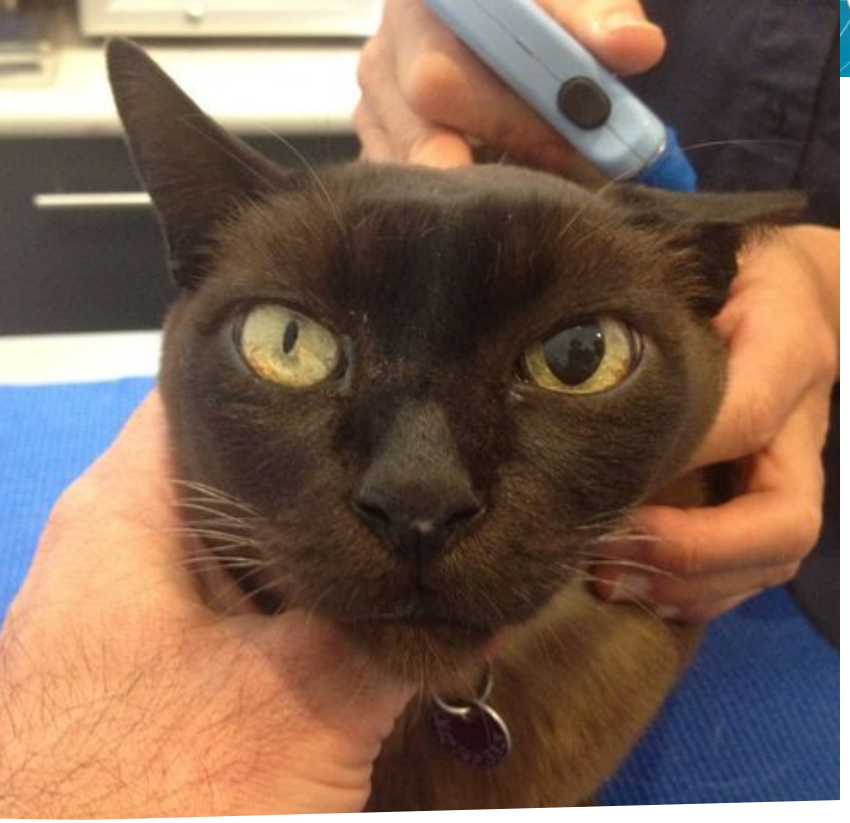
- T4 = 8 nmol/L
  - LOW
  - Euthyroid sick syndrome?
- Verification
  - cTSH
  - 1.16 ng/ml
  - HIGH

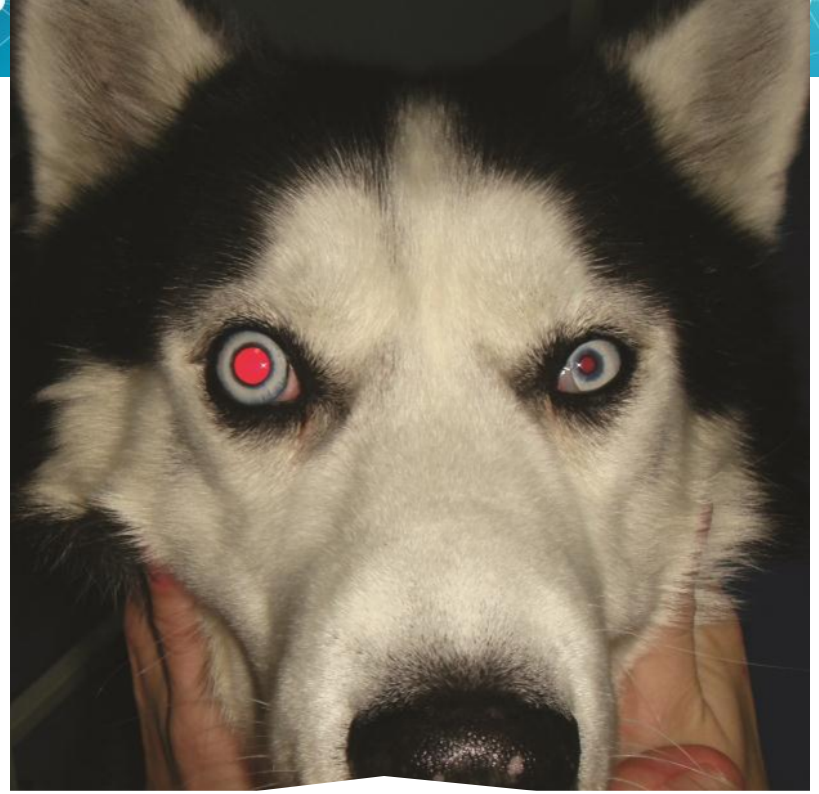
- HYPOTHYROID NEUROPATHY

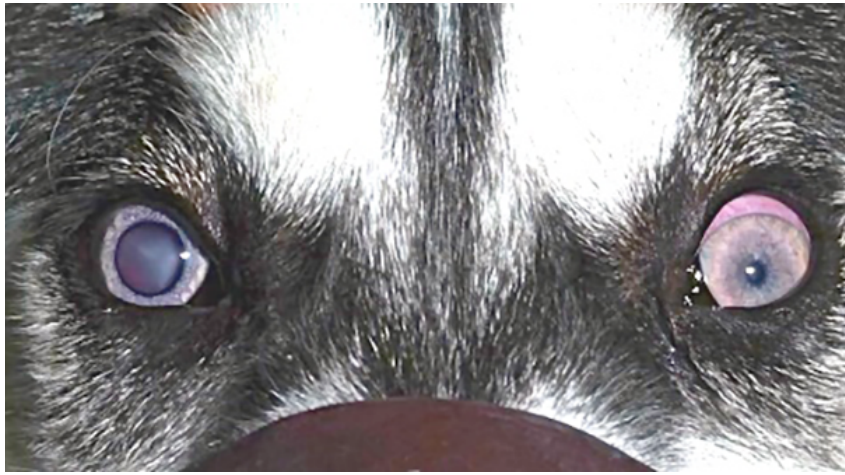
# Management

- Leventa 0.45ml q24h (for 23kg)
  - Checked in 3 weeks
    - T4 16
  - Clinical response within 14 days
  - Aim at 25 nmol/L (mid range)
  - By 1 June, clinical normalisation

# Case #4

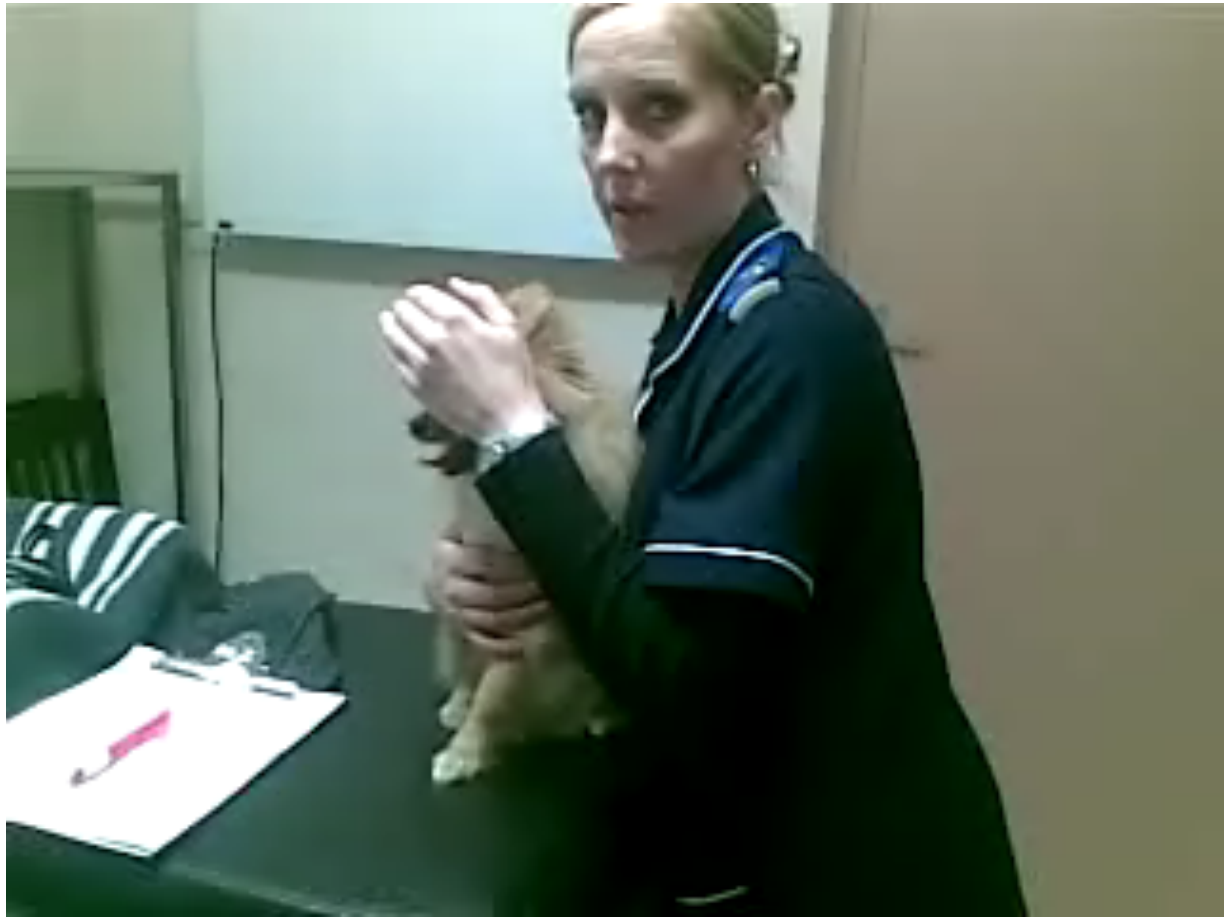




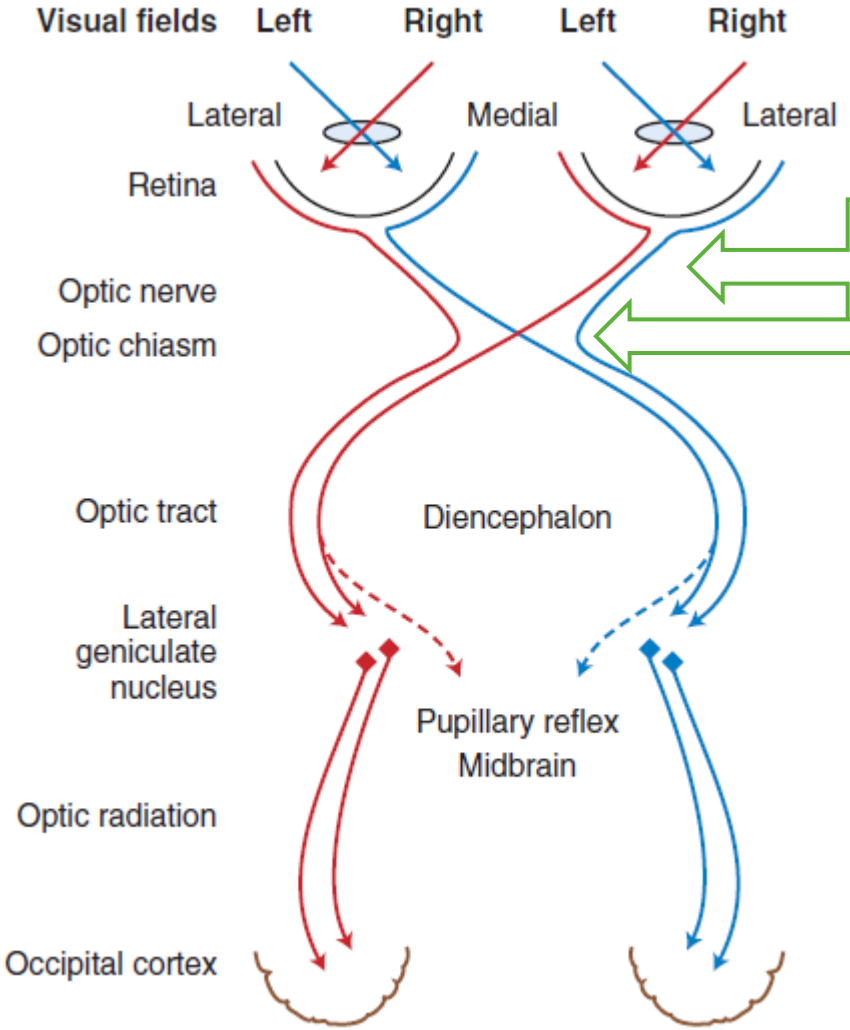


# Failure to constrict or dilate?

- Failure to constrict = loss of parasympathetic (Cr N III, Oculomotor)
- Failure to dilate = sympathetic failure (SNS)



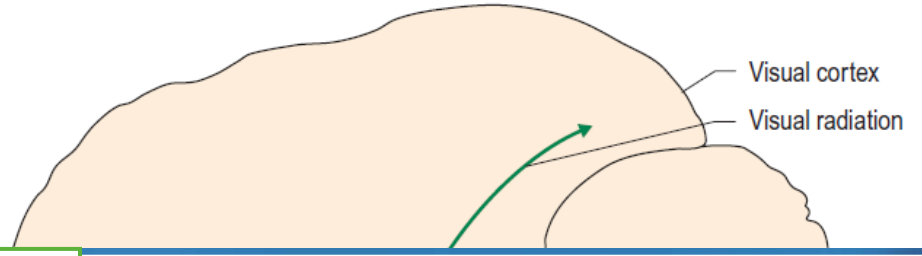
# Little pupil or big pupil – which is the problem?




**BLIND** ipsi
**Dir – Cons -**

geniculate

**BLIND** ipsi
**Dir – Cons -**



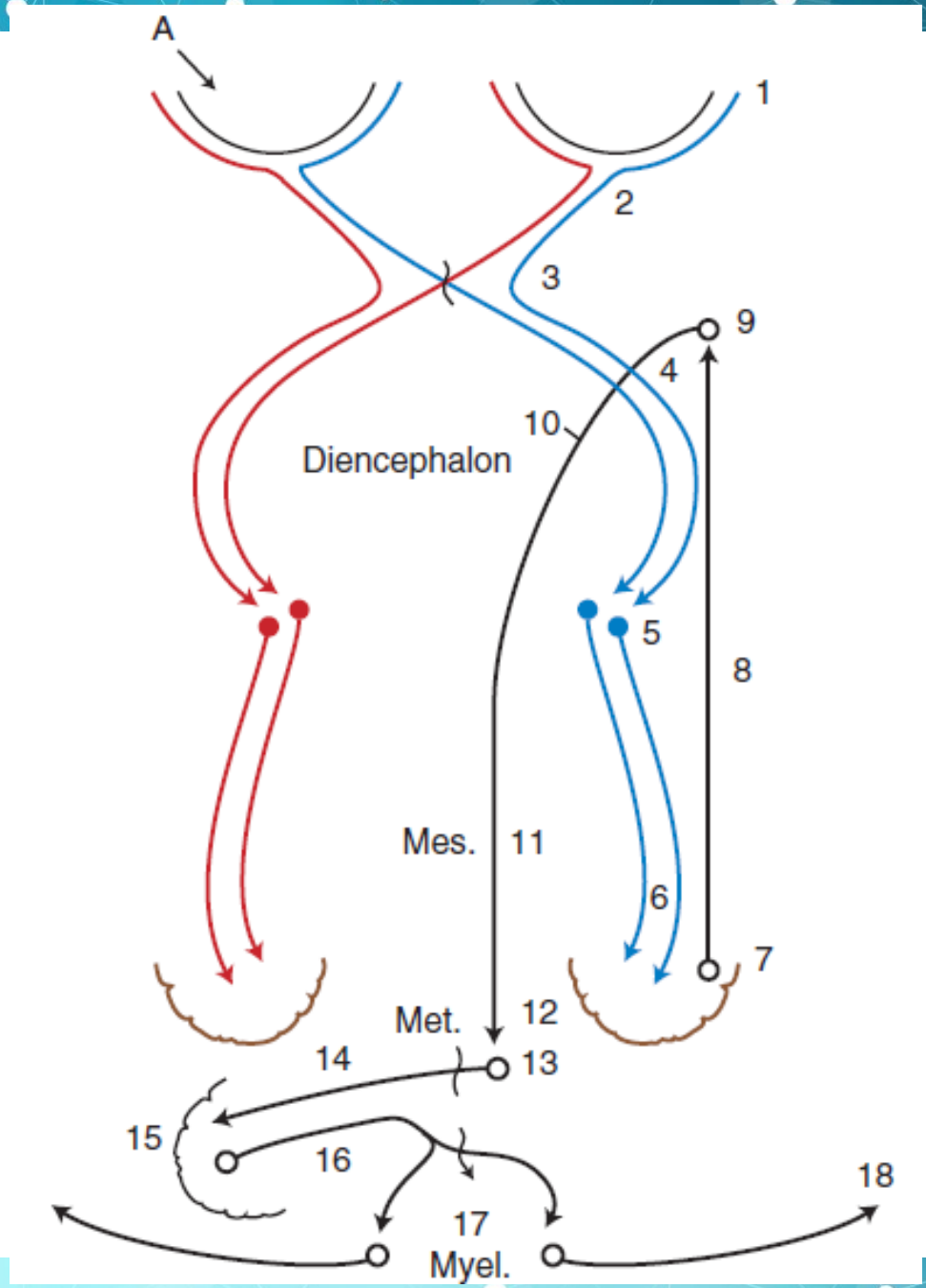

*l and Pupillary Light Reflex Pathways*

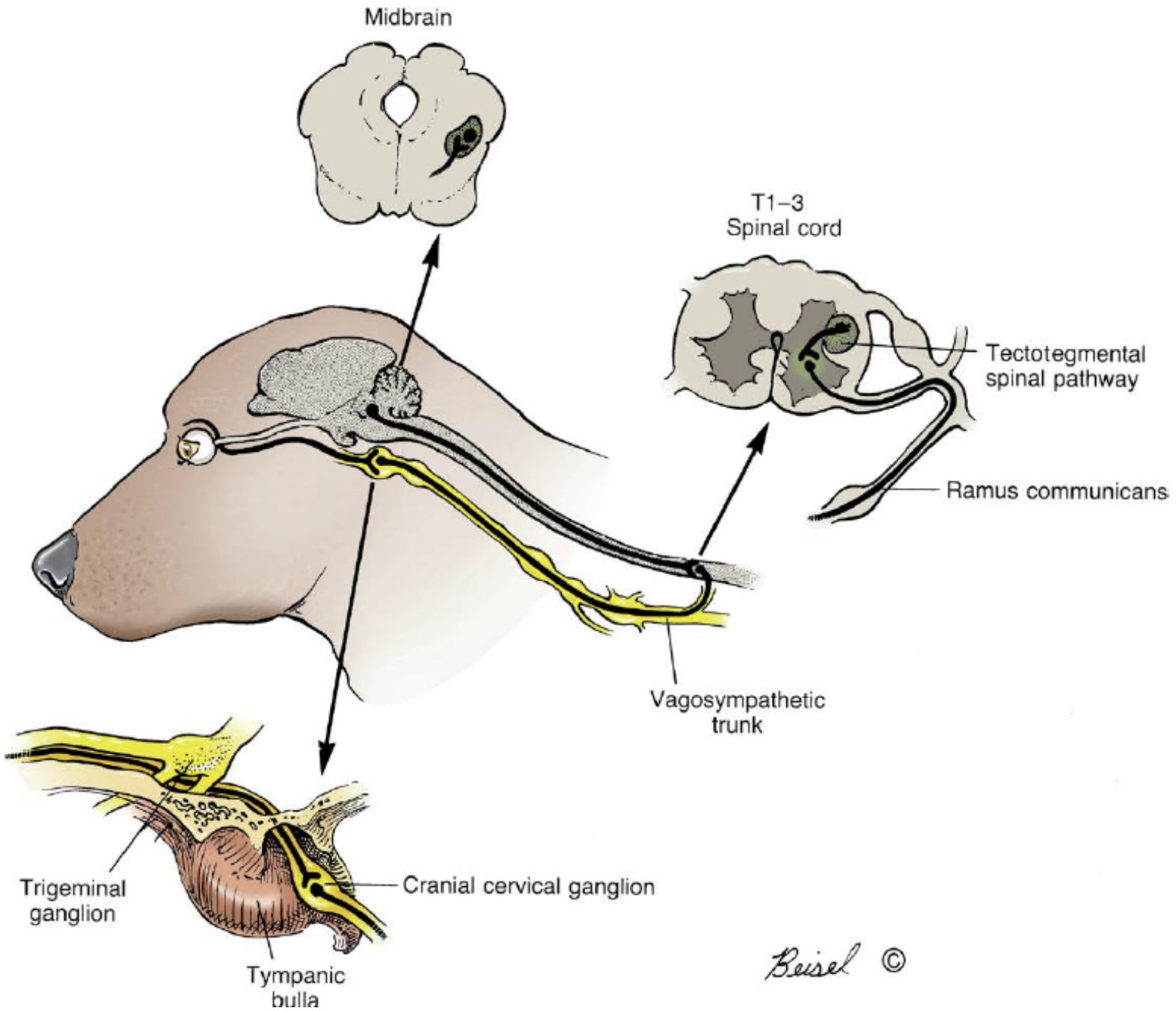
| | ision/Menace Response | Pupil Size | Pupillary Light Reflexes |
|---|---|---|---|
| lesion) | bsent on affected side | Usually mydriasis of the affected side* | *When testing affected eye*<br>Direct PLR—reduced to absent<br>Consensual—reduced to absent<br>*When testing the unaffected eye*<br>Direct PLR—normal<br>Consensual—reduced to absent |
| Bilateral retina/optic nerve/optic chiasm | Absent in both eyes | Bilateral mydriasis | Absent in both eyes |
| Unilateral optic tract, lateral geniculate nucleus, optic radiation, occipital cortex | Absent in contralateral eye‡<br>Normal in ipsilateral eye | Normal in both eyes | Normal in both eyes |
| Midbrain (bilateral) parasympathetic nucleus of CN III† | Normal in both eyes | Bilateral mydriasis | Absent in both eyes |
| Unilateral CN III | Normal in both eyes | Mydriasis of the affected side<br>Normal in contralateral eye | *When testing affected eye*<br>Direct PLR—reduced to absent<br>Consensual—normal<br>*When testing the unaffected eye*<br>Direct PLR—normal<br>Consensual—reduced to absent |
| Sympathetic innervation to the eye | Normal in both eyes | Constricted on affected side and does not dilate in dark<br>Normal on contralateral side and dilates in dark | Normal in both eyes |

ulus
s
spinal tract
l connections
ement.

An anatomic pathway for the **menace response**. *Mes.*, Mesencephalon;
*Met.*, metencephalon; *Myel*., myelencephalon. indicates axons crossing the midline of the brain. **A lesion in the left cerebellar hemisphere** would prevent a response to a menace directed at the left eye from its left visual field (*A*) because 65% to 90% of the optic nerve axons cross in the optic chiasm.

1. Retina
2. Optic nerve
3. Optic chiasm
4. Optic tract
5. Lateral geniculate nucleus
6. Optic radiation
7. Visual cortex
8. Internal capsule association fiber
9. Motor cortex
10. Internal capsule projection fiber
11. Crus cerebri
12. Longitudinal fibers of the pons
13. Pontine nucleus
14. Transverse fibers of the pons and middle cerebellar peduncle
15. Cerebellar cortex
16. Efferent cerebellar pathway
17. Facial nuclei
18. Facial muscles–orbicularis oculi






# Sympathetic innervation of eye

- Dilation of pupil

- Failure = MIOSIS
- Ie Horner's Syndrome

# Case #5

# 8yo Airedale, Acutely Progressive

# What to decide?

- Forelimbs and hindlimbs?
- Only hindlimbs?
- If tetraparetic, C5+
- If hindlimb UMN and forelimb LMN, C6-T2
- If hindlimb UMN, T3-L3
- If hindlimb LMN, L4-S2
- BUT

# What if hindlimb LMN, ascending and stopping?

- History
- Vaccination
- Trauma
- Hyperpathic zone
- Localise
- Careful repeat neuro
- Other tests? Imaging? CSF? Bloods? Titres?

# Why isn’t it…

- A disc
  - Because no pain
  - Not UMN in h/4
  - Ascending
- A tumour
  - Imaging negative
- Rabies
  - No cranial nerve involvement
- FCE
  - Progressive and ascending

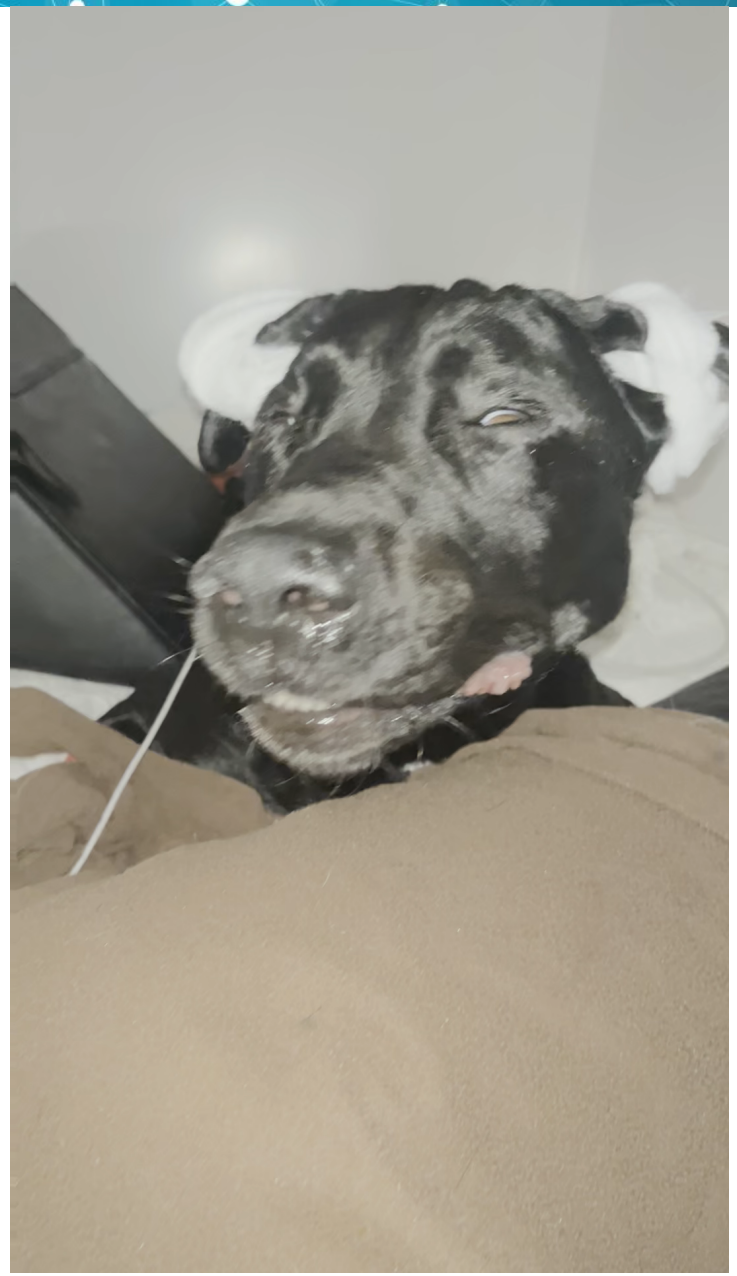
# Diagnosis

- Progressive, ascending, lower motor neuron
  - Polyradiculoneuritis

- DDx
  - Myelitis
  - Myelomalacia
  - Axonopathies
  - Cysts
  - Thromboembolism?

# Cases #6 and #7

Compare and contrast

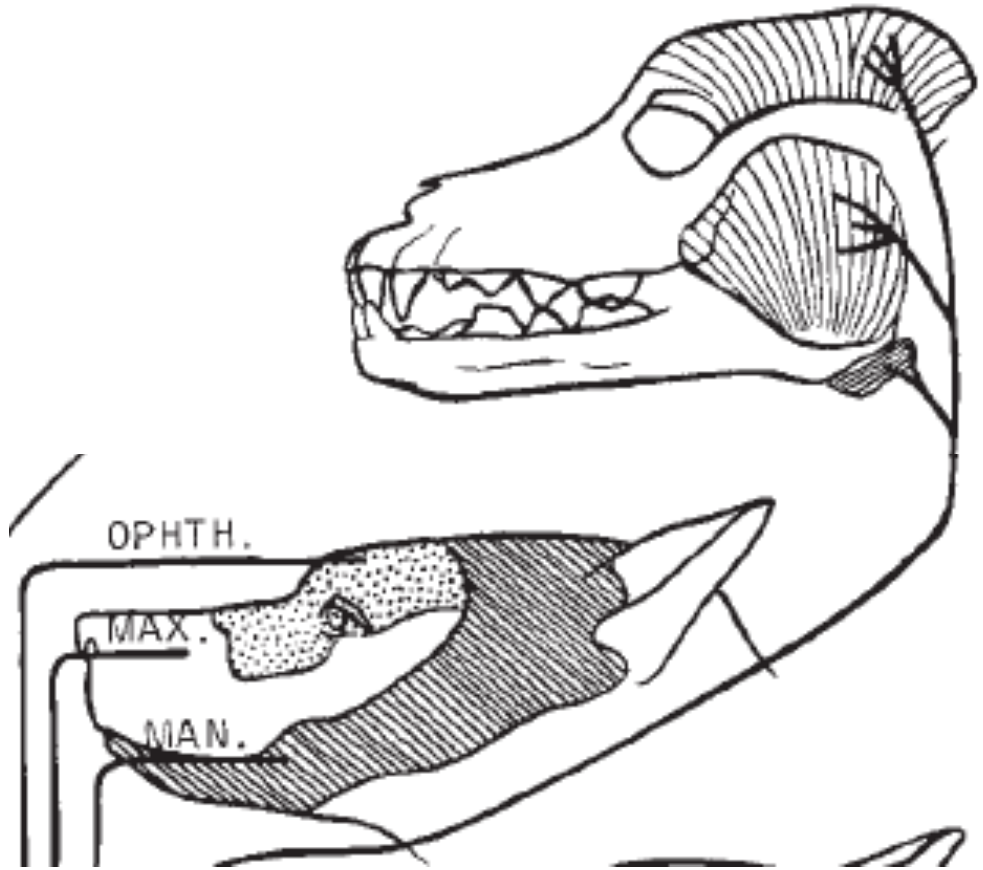


Courtesy TOMAS ELVIRA-RODRIGUEZ - ECVN Resident - Neurology [LIN]

# Are these just opposites or truly different?

- **TRISMUS**
  - Locked jaws
  - Articular or nonarticular
    - Infection (tetanus, parotid sialoadenitis, meningitis, tonsillitis)
    - Fracture (jaw, TMJ)
    - Tumours (jaw, parotoid, pharyngeal)
    - Fibrosis (eg after myositis, radiation)
    - Autoimmune (myositis, SLE)
- **DROPPED JAW**
  - Trigeminal nerve, bilateral
    - DDx fracture, lymphoma, leukaemia, brainstem trauma

Trigeminal MOTOR




Trigeminal SENSORY