馬頸椎狹窄性脊髓病
Equine cervical vertebral stenotic myelopathy

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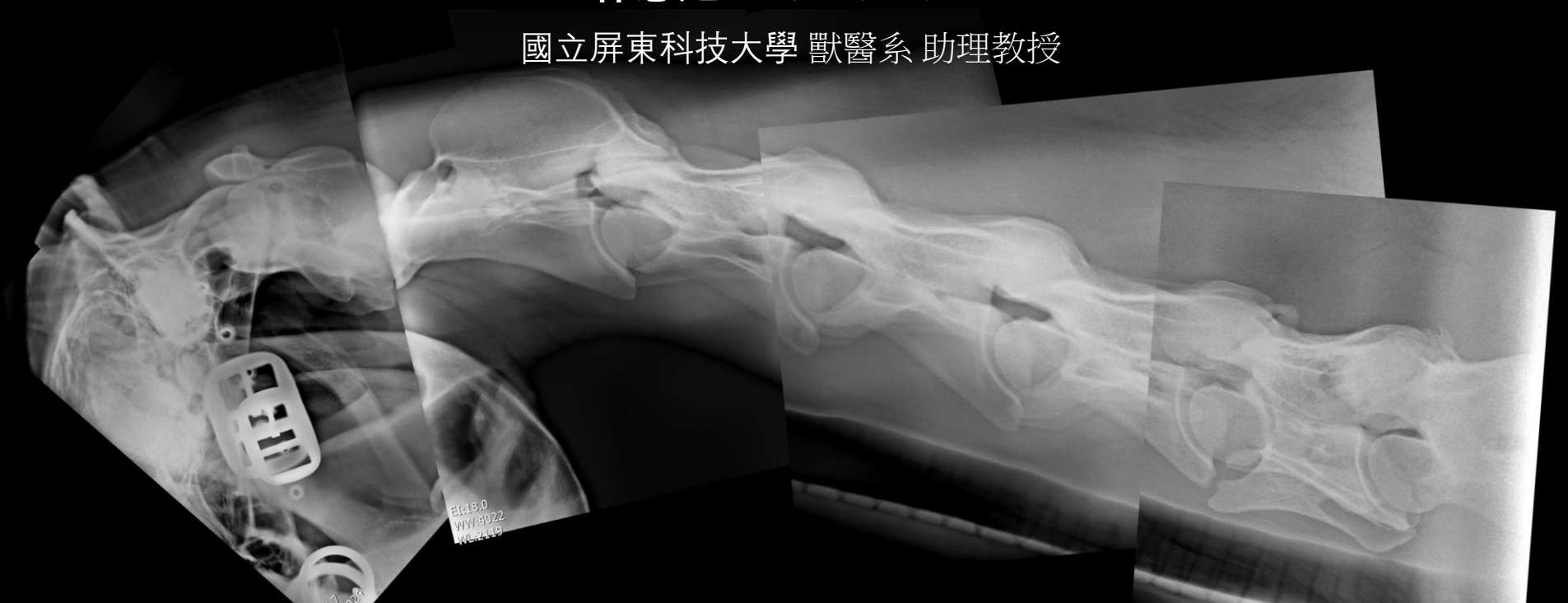
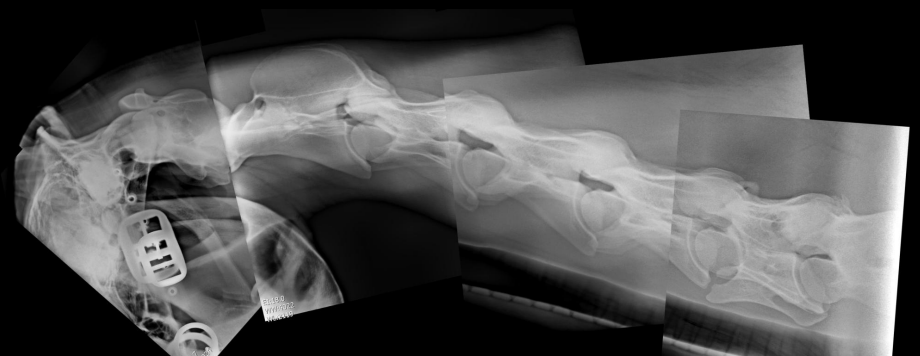


Outline

- Cervical vertebral stenotic myelopathy (CVSM)
- Types
- Clinical signs
- Diagnosis
- Treatments
- Pathological findings



Cervical vertebral stenotic myelopathy (CVSM)

- ***Malformatin of the cervical vertebrae leading to vertebral canal stenosis and subsequent spinal cord compression.***

- ***Leading cause of ataxia in horses worldwide.***

- Wobbler syndrome
- Equine sensory ataxia
- Equine incoordanation
- Cervical vertebral malformation
- Cervical stenotic myelopathy
- Cervical vertebral compression myelopathy
- Cervical vertebral stenotic myelopathy (CVSM)

Types of CVSM -- etiologies

- Development
  - Young, rapidly growing horses (1-3yo)
  - C5-C7
- Osteoarthritis or degenerative joint disease
  - Older horses, in training (>4yo)
  - C5-C7
- Congenital
  - Occipitoatlantoaxial malformation
  - Foals
  - Arabians
  - Other breeds

Types of CVSM – imaging findings

- Dynamic
  - Compression on myelogram identified only when the neck is flexed.
  - C3-C5
  - Young horses (8-18mo)

- Static
  - Narrowing of the vertebral canal identified regardless of neck positions.
  - C5-C7
  - Older horses

Clinical presentation of CVSM

- Neurological gait deficit
- Incoordinate movement (ataxia)
- Spasticity
- Weakness, particularly in the hindlimb (most commonly missed by owners)
- Decreased proprioception
- Falling

Clinical presentation of CVSM

- Base wide
- Truncal sway at walk

Clinical presentation of CVSM

- Change in temperament

(Dyson, 2011)

Ataxic horses - ”Differential diagnosis”

- Non-infectious
  - CVSM: most common
  - Traumatic injuries: fracture, luxation (also common)
  - Vestibular or cerebellar ataxia
  - Equine degenerative myeloencephalopathy (EDM)
  - Neoplasia
- Infectious
  - Protozoan parasites: Equine protozoal myeloencephalitis (EPM)
  - Virus: EHV-1, rabies, botulism, West Nile…
  - Bacteria: Lyme disease

(Nout and Reed, 2003)

(Bedenice and Johnson, 2018)

Gold standard of a definite diagnosis?

Postmortem examination

(Image courtesy of Rossdales)

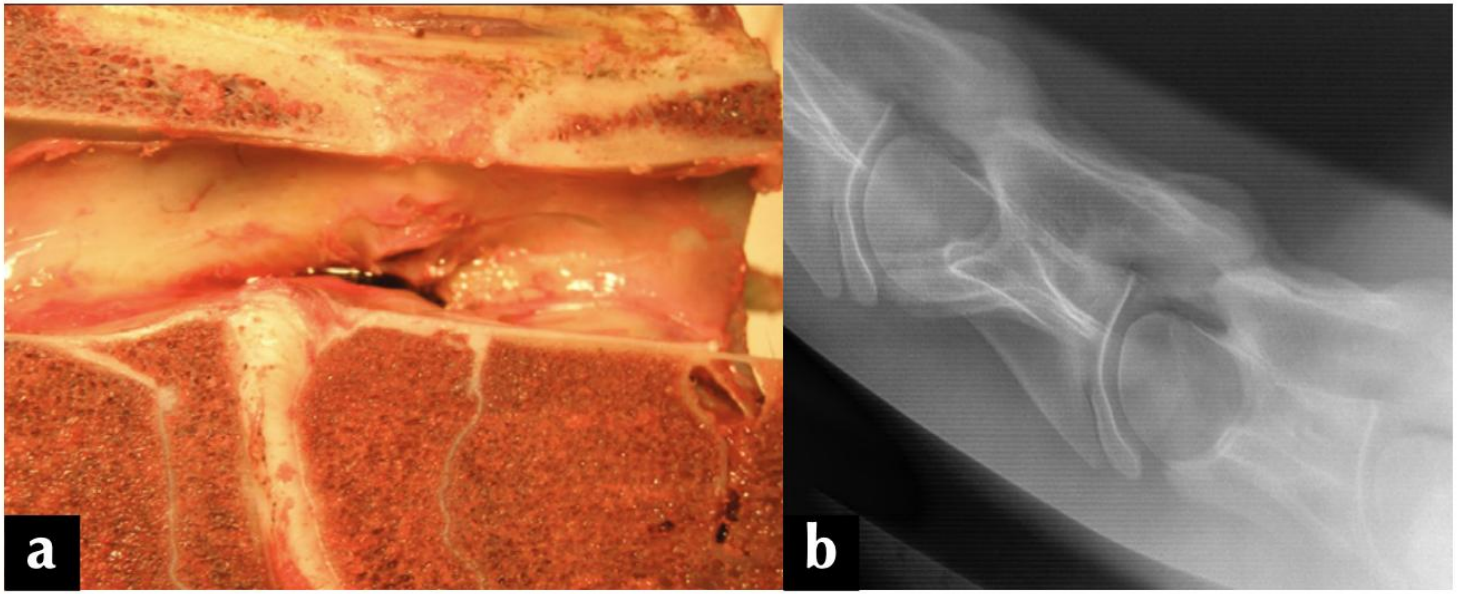


Fig 1: (a) This specimen illustrates how the spinal canal can become narrowed when the cervical vertebral bones grow in an abnormal shape. (b) The narrowing of the spinal canal is also visible in a radiograph of the same horse.

Diagnosis of CVSM:
1. Is it really spinal cord origin?

- Non-spinal cord
  - Vestibular
  - Cerebellar
  - Cerebral
  - Brainstem

- Neurological examination
  - Behaviour or mentation
  - Head and neck position
  - Posture
  - Cranial nerve examination
  - *Cervicofacial (cervicoauricular) reflex*
  - *Thoracolaryngeal reflex*
  - *Cutaneous trunci reflex*

Table 5.1. Tests of Cranial Nerve Function

Test	Nerve Tested	Abnormal Response/Interpretation
Menace	Optic, Facial	No eye blink; blindness. Must differentiate blindness from facial nerve dysfunction
Pupillary light response	Optic, Oculomotor	No response to bright light directed in eye
Horner's syndrome	Cervical sympathetic	Sweating around base of ear and eye, ptosis
Facial sensation	Facial (sensory)	Failure to respond to stimulation of facial skin
Facial symmetry	Facial (motor)	Asymmetry of muzzle, +/− ear droop, food impacted in cheek
Palpebral reflex	Trigeminal, Facial (motor)	Failure to blink
Nystagmus	Oculomotor, vestibular system	Central lesions associated with positional nystagmus; peripheral lesions are non-positional
Swallow	Glossopharyngeal, Vagus	Inability to swallow as determined by observation or passing of stomach tube
Tongue tone	Hypoglossal	Failure to withdraw tongue, or tongue weak when pulled

(Furr and Reed, Equine Neurology, 2008)

Diagnosis of CVSM:
2. Is it cervical or thoracolumbar disorders?

- Locomotor examination
  - Walk
    - Slow: *upper motor neuron spinal cord disease can be compensated for speed in most quadrupeds*
    - Straight line
  - Figure of 8
  - Tight circles
  - Backing
  - Tail pull
  - Uphill & downhill (+ head elevation)

- ***Ataxia, paresis, spasticity, loss of proprioception***

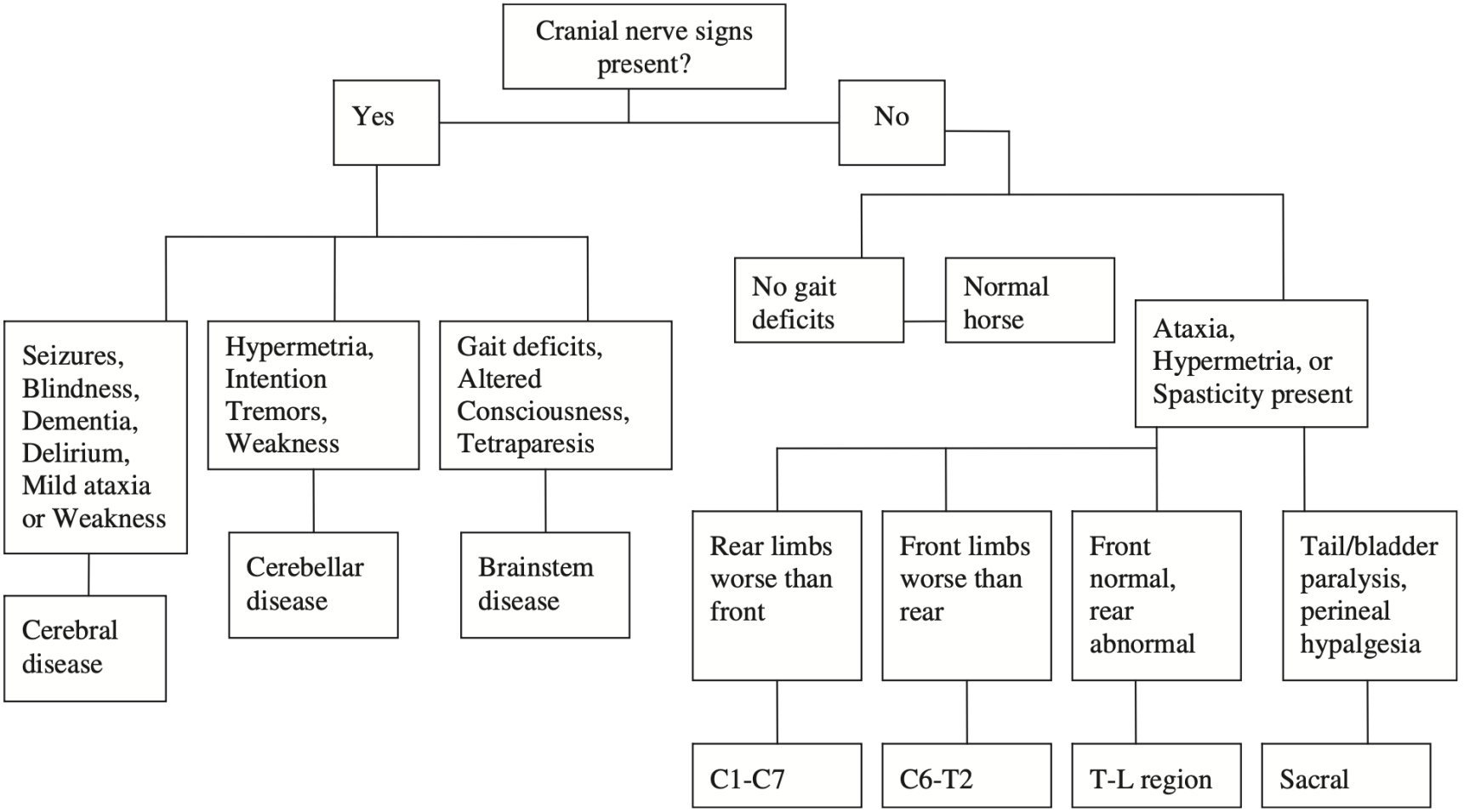


Figure 5.2. A flow chart demonstrating how the results of the neurologic examination can be used to aid the clinician in neuroanatomic localization. Ideally, the clinician should try to make one lesion explain all clinical signs. Combinations of clinical signs that cannot be explained by one site of disease imply diffuse or multifocal disease.

(Furr and Reed, Equine Neurology, 2008)

Diagnosis of CVSM:
3. Antemortem diagnosis of CVSM?

- Diagnostic imaging
  - *Radiography*
    - Stenosis of vertebral canal
    - Does not indicate the site of compression
  - *Myelography*
    - Specific site of compression
  - *Computed tomography*
    - Cross-sectional imaging
    - Prognosis
    - (Gold standard?)
  - Magnetic resonance imaging

- ***Other characteristic radiographic changes***

Radiography: Intra-sagittal ratio (Sagittal ratio)

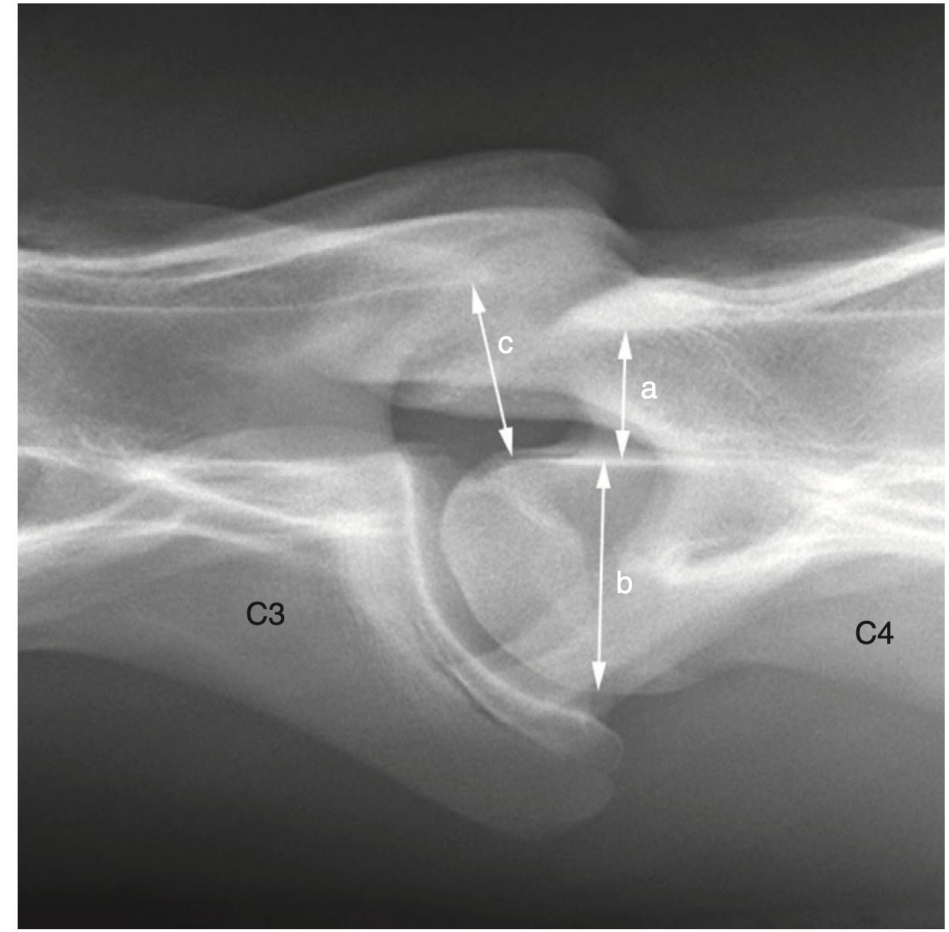


Fig 1: Lateral neck radiograph depicting the measurements made at C3–4 to determine the intra- (a/b) and the intervertebral (c/b) ratios.

(Scrivani et al., 2011)

- ***52% for C3-C6***
- ***56% for C7***

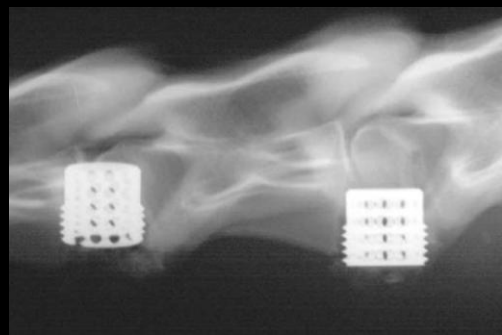
- ***Reported sensitivity & specificity 89-90%***

Treating CVSM has been “a bit” disappointing

- *Conservative management*
  - NSAIDs
  - Corticosteroids
  - VitE/Selenium supplements
  - Restricted exercise
  - Diet change

- ***30% return to work***
  - ***Mild cases***
- ***31% euthanised***

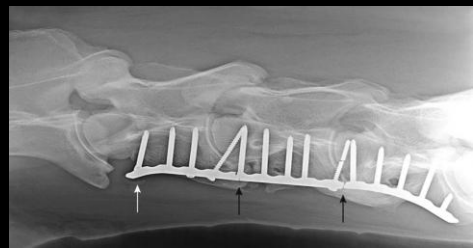
- *Surgical intercention*
  - Cervical ventral intervertebral fusion (CVIF)
    - Most commonly used technique
  - Uniportal endoscopic foraminotomy
    - Novel technique
  - Arthroscopy
    - Less common



(Nout and Reed, 2003)

Outcome of surgical intervention (CVIF)

Year	Percentage	Details
2005	37% (14/38)	Atheletic use
	18% (7/38)	Fatality (euthanised)
2018	57% (8/14)	Fatality (euthanised)
2018	13% (1/8); 2-3 years follow-up	Fatality (euthanised)
2022	20% (2/10); 1 year follow-up	Fatality (euthanised)
2023	45% (13/29)	Atheletic use
	17% (5/29)	Fatality (euthanised)
2025	41%; 1.5-6 years follow-up	Gradually became unridden
	9%	Fatality (euthanised)



Postmortem examination is the gold standard for diagnosis

- Some may argue pathological changes might not be found in dynamic CVSM.

Dorsal

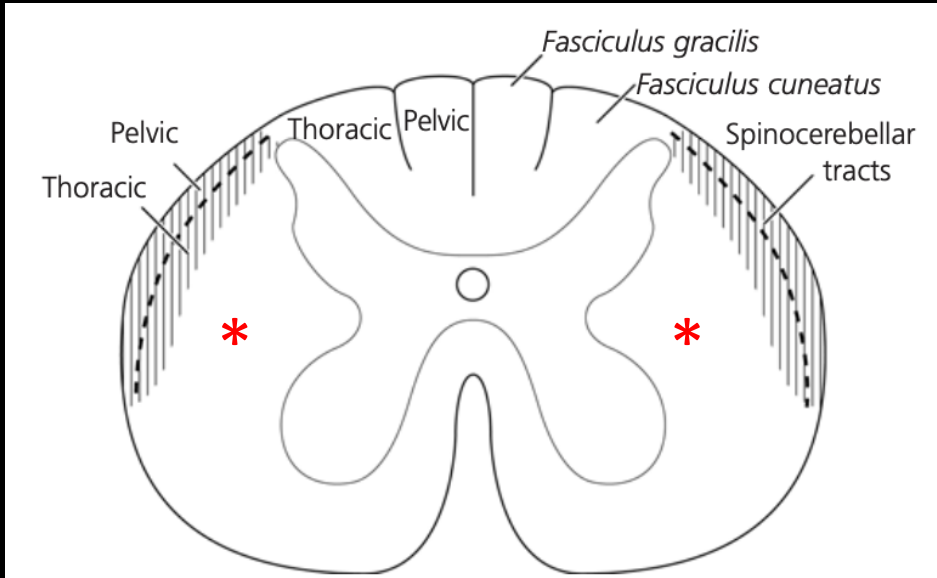
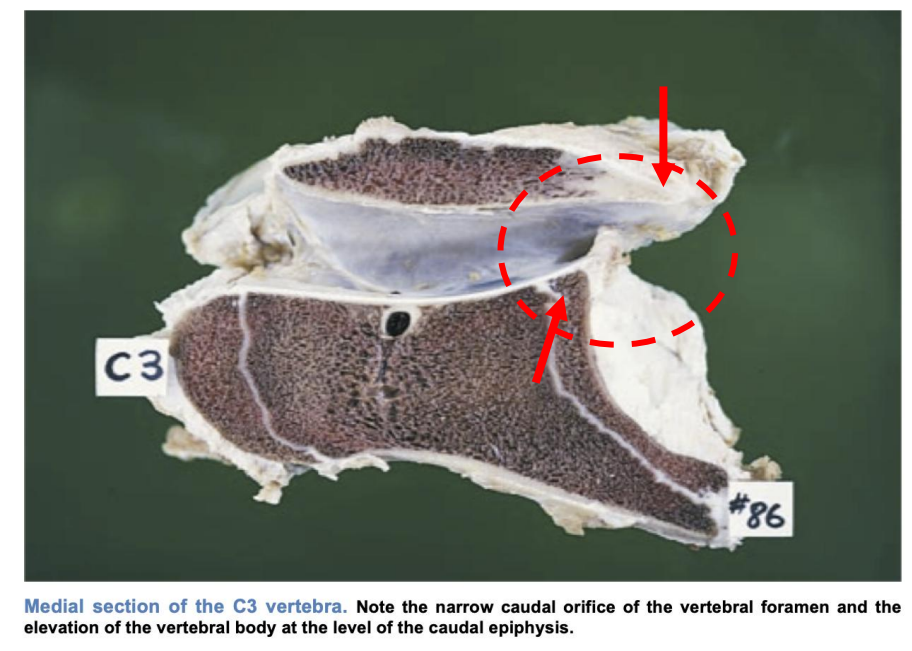


Fig 5: Cross-section of the cervical spinal cord showing the affected proprioceptive tracts during spinal cord compression.

(Nout and Reed, 2003)

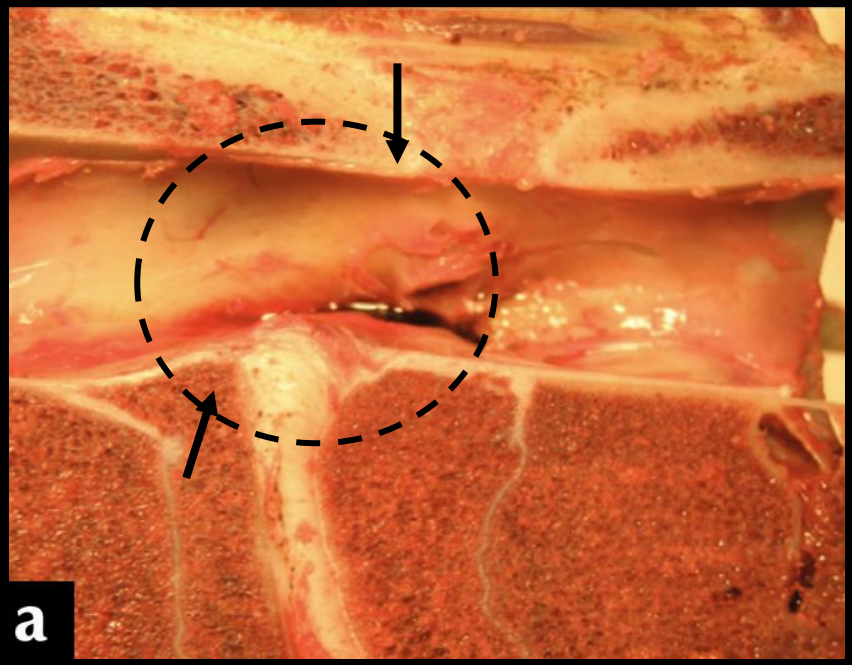
Ventral

- Narrowed caudal orifice of the vertebral canal.
- Enlargement and dorsal protrusion of the caudal epiphysis.
- Caudal extension of the dorsal laminae.



Medial section of the C3 vertebra. Note the narrow caudal orifice of the vertebral foramen and the elevation of the vertebral body at the level of the caudal epiphysis.

(Pradier and Muylle, 2014)



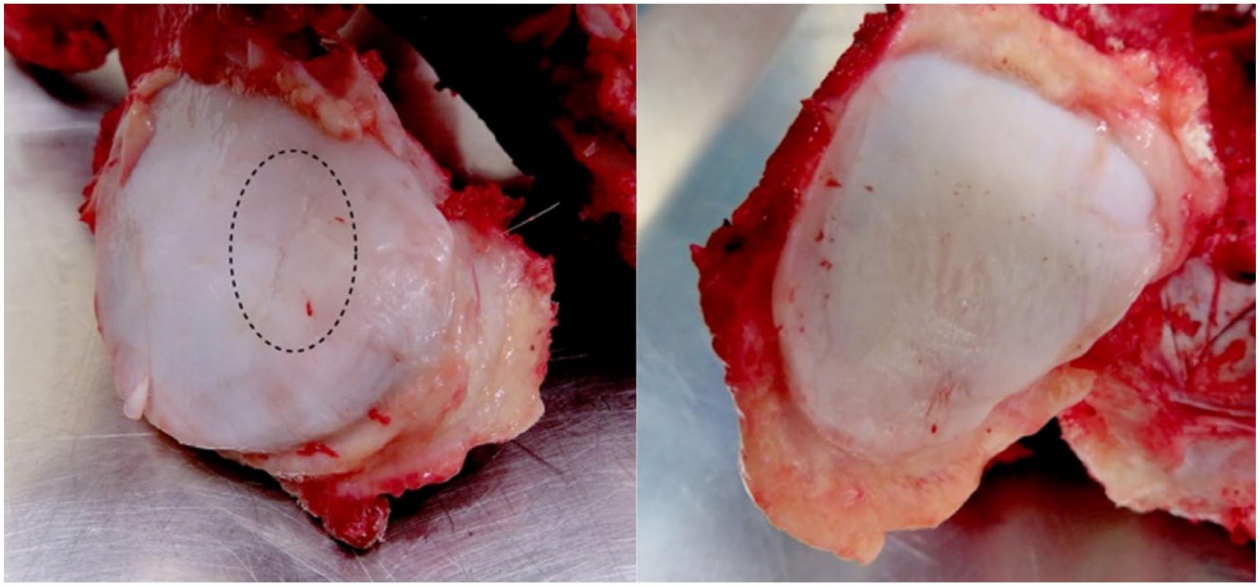
(Image courtesy of Rossdales)

- Multiple congested and enlarged blood vessels on the dorsal surface of the spinal cored.

Fig 2: Dorsal view of spinal cord acquired after excision from the spinal canal at the level of C4/C5 (the nerve roots of the fifth spinal nerve are visible biaxially as they leave the spinal cord) showing engorged blood vessels consistent with a compressive lesion.

(Bailey et al., 2021)

- Linear branching fissure in the cartilage.
- Generalised irregular and fibrillated appearance to the cartilage.



(Bailey et al., 2021)

Fig 3: Post-mortem appearance of the right caudal articular process of C4 (left) and the right cranial articular process of C5 (right) showing a branching fissure of the cartilage (circled) of C4 and irregular and fibrillated appearance to the cartilage on C5.

- Axonal swelling (spheroid), axonophagia, and axonal dropout

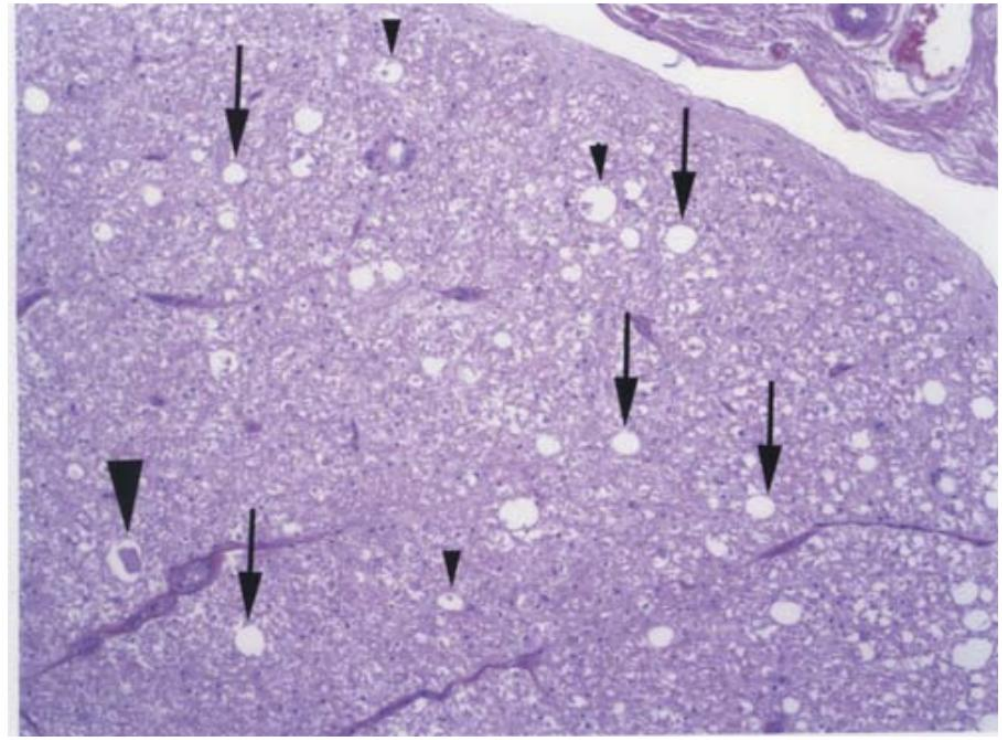


Fig 2: Haematoxylin and eosin stained cross-section of the cervical spinal cord from the horse in Figure 1 viewed at x40 magnification. *This is an image of the ventral funniculus at the level of C3–C4. In the* ***top right*** *corner the ventral median fissure is present. This cross-section demonstrates axonal swelling (****large arrow head****), axonophagia (****small arrow heads****) and axonal dropout (****arrows****).*

(Nout and Reed, 2003)

- Wallerian degeneration characterised by dilayed myelin sheaths containing glassy or eosinophilic, swollen axons or necrotic debris (ellipsoids) mixed with foamy macrophages.
- Multifocal spheroids

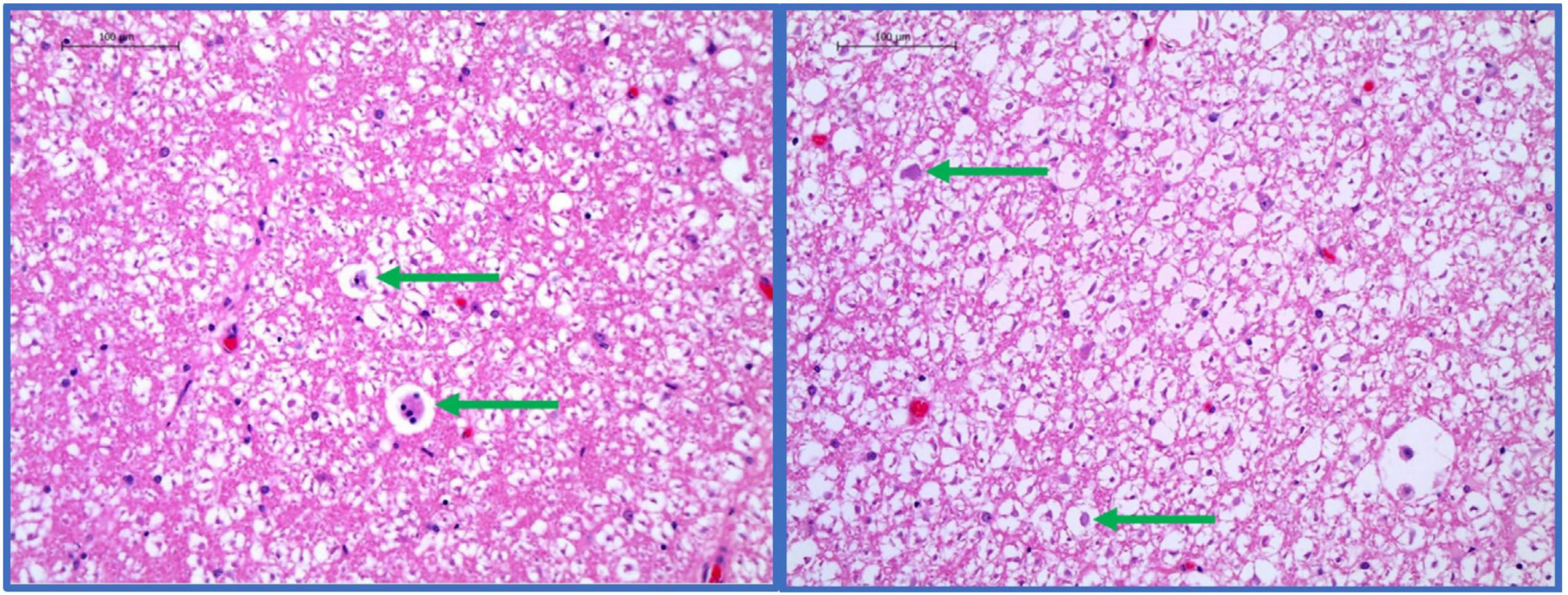


Fig 4: left—H&E-stained section of spinal cord showing Wallerian degeneration in ascending white matter tracts (dorsolateral funiculus) cranial to the lesion (green arrows show an ellipsoid and a digestion chamber), right—H&E-stained section of spinal cord showing Wallerian degeneration of descending tracts (ventral funiculus) caudal to compressive lesion (Green arrows: spheroids, large cavity bottom right: digestion chamber) scale bars measure 100 um.

(Bailey et al., 2021)

- Spinal cord, dorsal medial fasciculi: Necrosis, focally extensive, asymmetric with lymphohistiocytic and eosinophilic perivasculitis and lepto-meningitis, quarterhorse, *Equus caballus*

2-1. Cervical spinal cord, horse.

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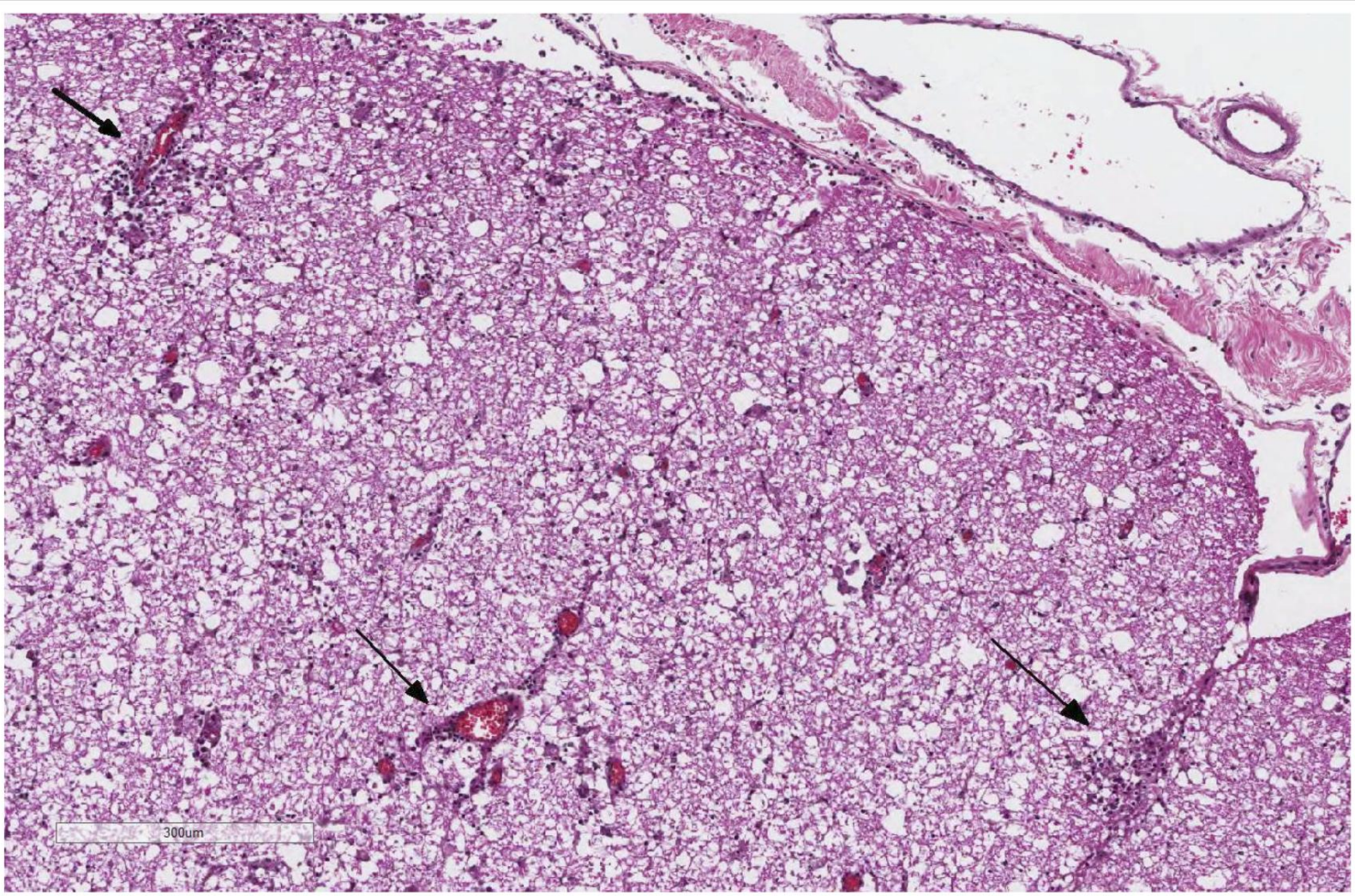
- Dorsal funiculus: moderate numbers of dilated myelin sheaths and a lymphoplasmacytic and eosinophilic perivascular infiltrate



2-2. Cervical spinal cord, horse.

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Question?