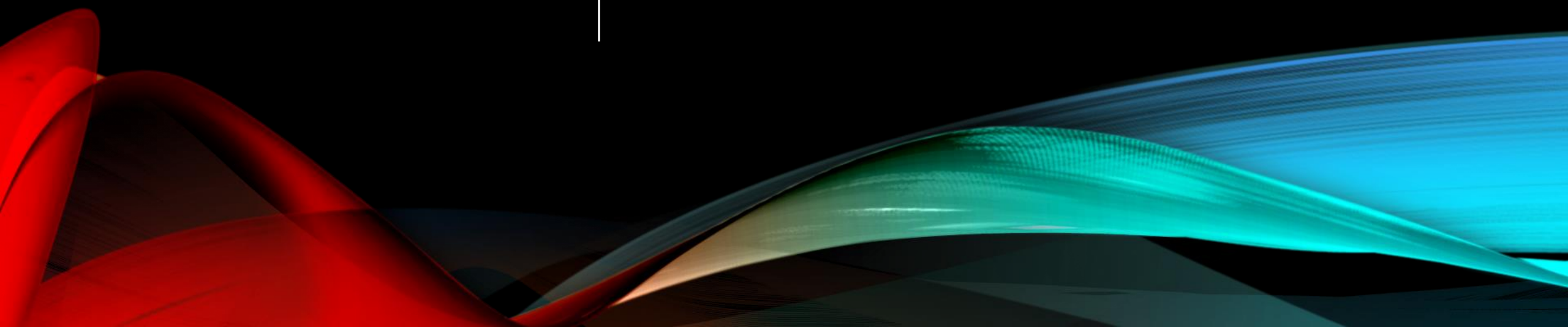


S.K. Shivapour, DVM  
10<sup>th</sup> April 2026

# THE NEUROLOGIC PATIENT



# PRESENTATION OUTLINE

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History & Five Finger Rule

---

Neurologic Examination

---

Neuroanatomic Localization

---

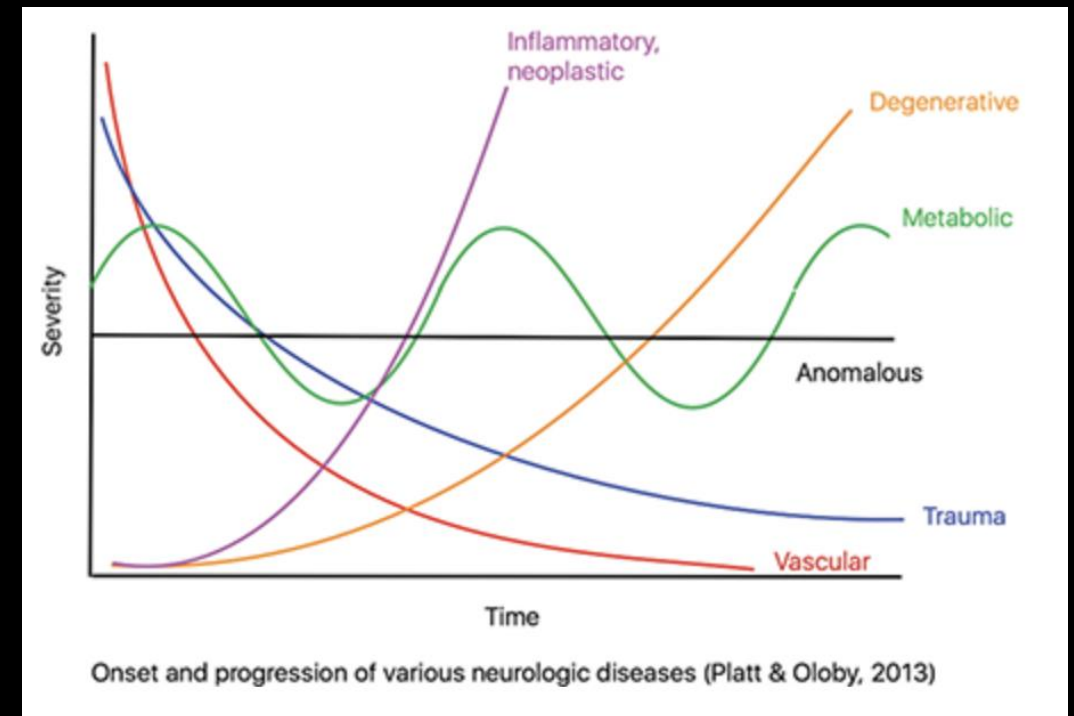
Neurologic Emergencies

---

Cases

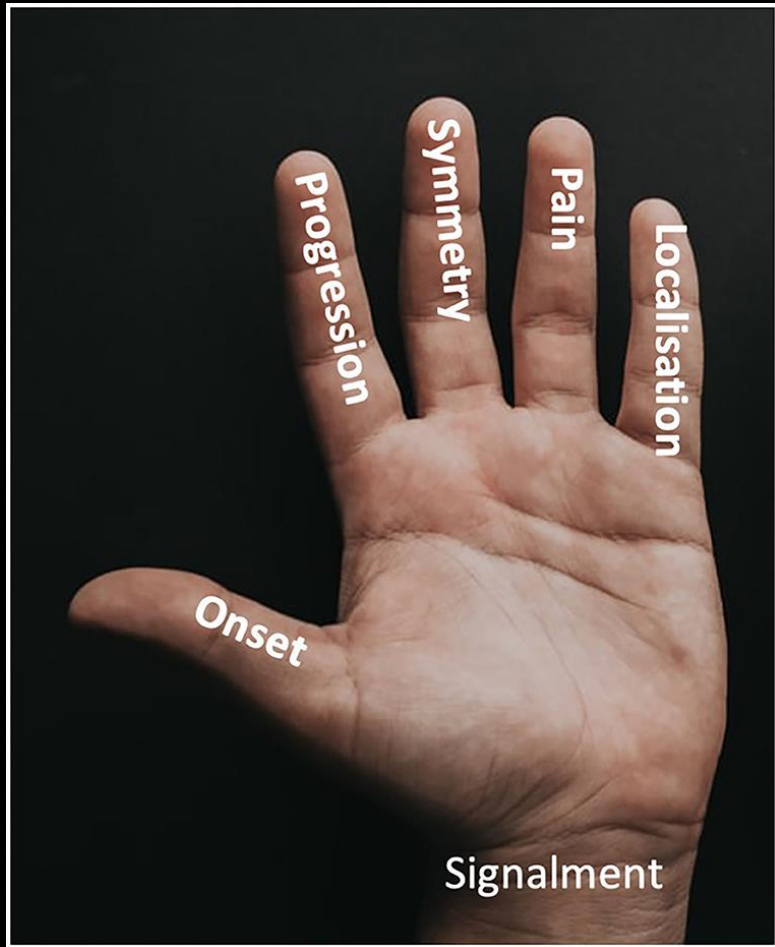
# HISTORY

- Details are critical
- Review prior medical records/imaging
- Videos of atypical episodes
- Trust, but **verify!**



# FIVE-FINGER RULE: GOALS

- Characterize neurologic problem
- Create appropriate differential list



# NEUROLOGIC EXAM: GOALS

1. Confirm presence of neurologic lesion.
2. Accurate neurolocalization.
3. Create appropriate differential list.
4. Determine diagnostic & therapeutic plan.

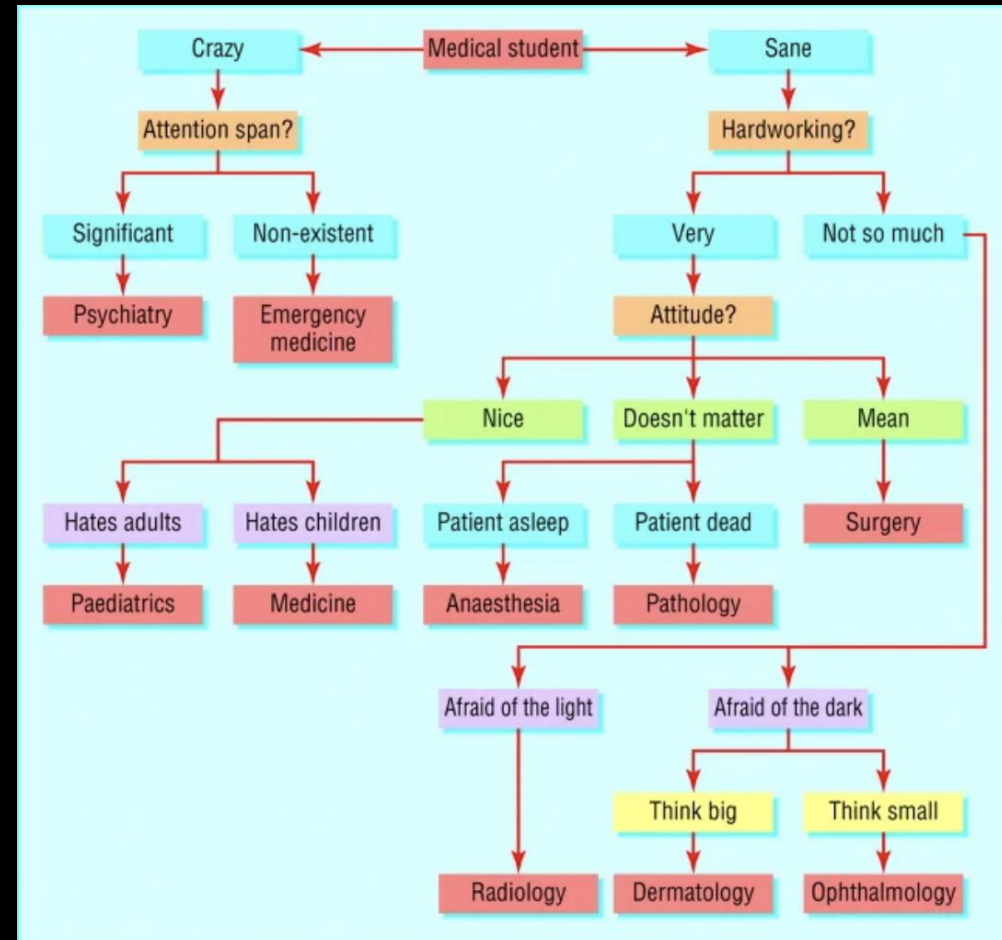
# NEUROLOGIC EXAM: BASIC COMPONENTS

## HANDS OFF

- Mentation
- Gait/Posture

## HANDS ON

- Cranial Nerves
- Postural Reactions
- Spinal Reflexes
- Palpation



# MENTATION = MENTAL STATUS + BEHAVIOR

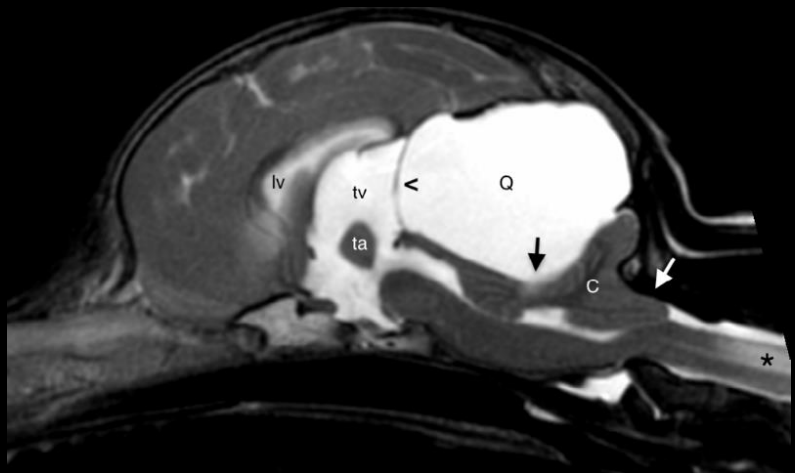
## DESCRIPTORS

- Appropriate
- Obtunded
- Stuporous
- Semi-comatose
- Comatose

## DEFECTS SEEN WITH:

- ARAS dysfunction (brainstem)
- Lesions of limbic system components of cerebrum or rostral brainstem
- Underlying systemic disease
- Post-ictal state (dysphoric, etc.)

# ARAS DYSFUNCTION



# ABNORMAL BODY POSTURE: HEAD & NECK CARRIAGE



## FELINE CERVICAL VENTROFLEXION

- Absence of nuchal ligament
- Systemic or metabolic disease



## CANINE LOW HEAD CARRIAGE (PRAYER POSTURE)

- Seen with cervical pain
- May or may not be myelopathic
- R/O acute abdomen

# ABNORMAL BODY POSTURE: HEAD TILT VS HEAD TURN

## HEAD TILT

- Vertical axis
- Vestibular
- Ipsilateral\*

## HEAD TURN

- Horizontal axis
- Forebrain
- Pleurothotonus
- Ipsilateral



# ABNORMAL BEHAVIOUR: CIRCLING + ROLLING



## WIDE CIRCLES/COMPULSIVE PACING

- Forebrain & thalamus\*
- Circle towards lesion



## TIGHT CIRCLES/ROLLING

- Vestibular
- Ipsilateral\*\*

# ABNORMAL BODY POSTURE: RIGIDITY POSTURES OF RECUMBENCY

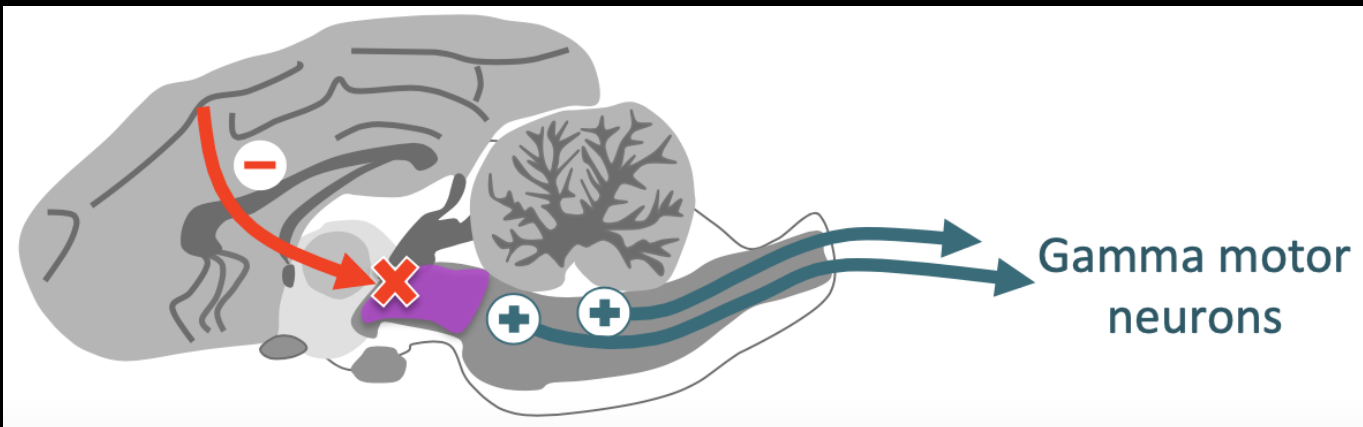




# ABNORMAL BODY POSTURE: DECEREBRATE RIGIDITY

## PRESENTATION

- Rigid extensor tone in all four limbs
- Opisthotonus
- **UNCONSCIOUS** mental status



## NEUROLOCALIZATION

- Midbrain

## PROGNOSIS

- **Poor to Grave**

# ABNORMAL BODY POSTURE: DECEREBELLATE RIGIDITY

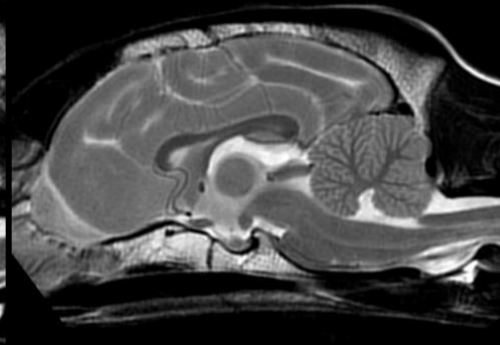
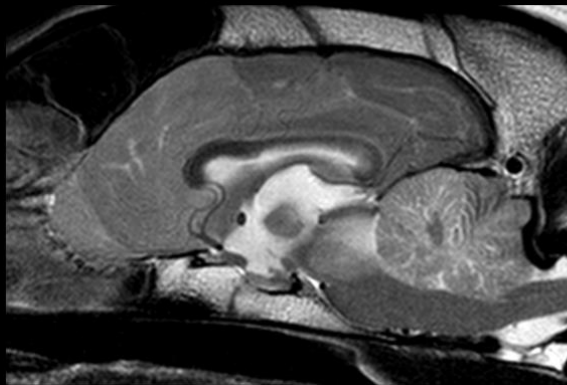
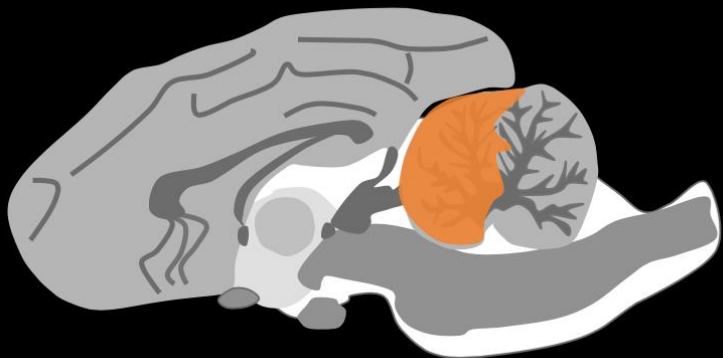


## PRESENTATION

- Rigid extensor tone in thoracic limbs
- Opisthotonus
- **CONSCIOUS** mental status

## NEUROLOCALIZATION

- Rostral cerebellum



## PROGNOSIS

- **Fair to Good**

# ABNORMAL BODY POSTURE: SCHIFF-SHERRINGTON

## PRESENTATION

- Rigid thoracic limb extensor tone in recumbency

## NEUROLOCALIZATION

- T3-L3 spinal cord injury
- Peracute lesions only



*What is the prognostic significance if seen?*

# GAIT EVALUATION

## 1. AMBULATORY vs NON-AMBULATORY

## 2. DEGREE OF MOTOR DYSFUNCTION

- **Paresis:** Reduced ability to bear-weight
- **Plegia:** Complete loss of motor function.

## 3. DISTRIBUTION & LATERALIZATION

- Mono-, Para-, Hemi-, Tetra-

## 4. PRESENCE & TYPE OF ATAXIA

- **Proprioceptive (Spinal):** Loss of limb spatial awareness
- **Vestibular:** Balance loss, head tilt, pathologic nystagmus, circling
- **Cerebellar:** Hypermetria, dysmetria



# CLASSIC NEUROLOGIC GAIT



- Limbs affected?
- Lateralization?
- Neuro-localization?
- Primary differential?

# CLASSIC CEREBELLAR ATAXIA



- Wide-based stance
- Intention tremors
- Truncal sway
- Titubation
- Hypermetria
- Dysmetria



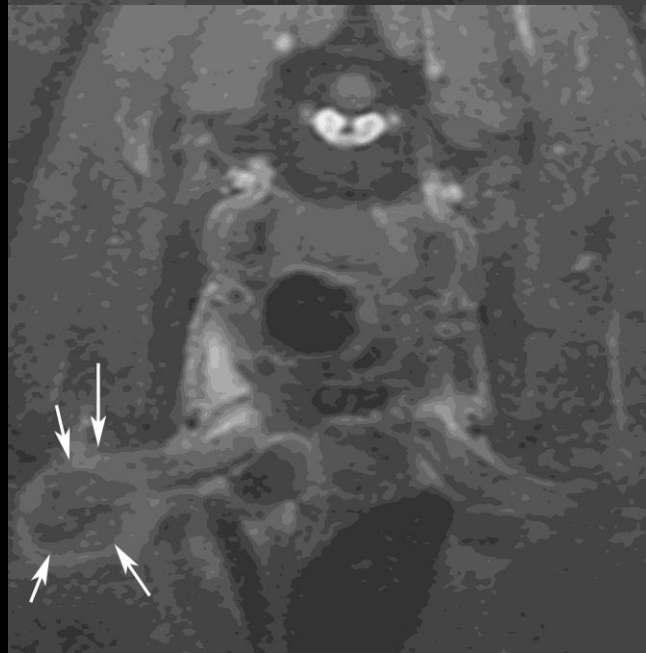
WHAT IS GOING ON HERE?



# MONOPARESIS & NERVE ROOT SIGNATURE



- **Neuro DDX**
  - Trauma
  - Foraminal IVDE/stenosis
  - Discospondylitis
  - Nerve sheath tumor
- **Non-Neuro DDX**
  - Neoplasia (OSA)
  - RCCL
  - Fracture/luxation

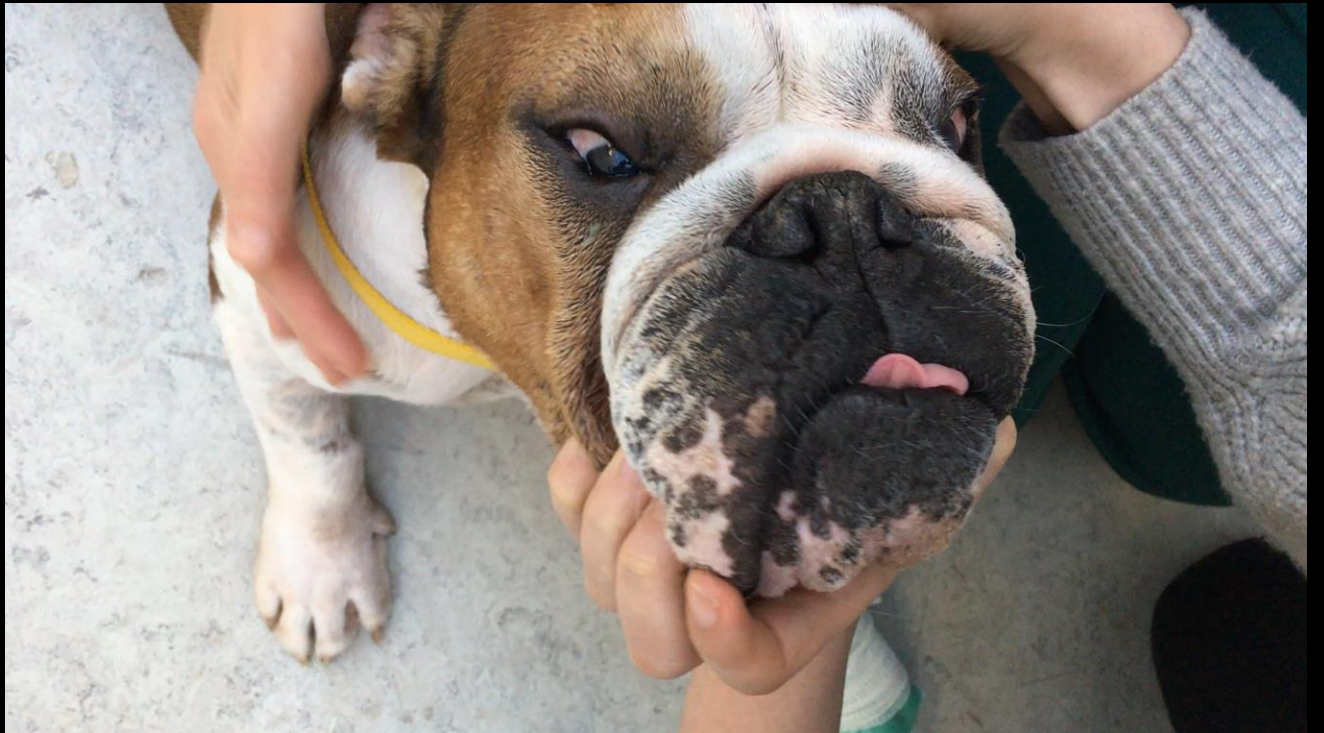


# NERVE ROOT SIGNATURE



# CRANIAL NERVES: BASIC EXAM

- Pupillary Light Reflex
- Menace Response
- Facial Sensation
- Palpebral
- Nasal Septal Response
- Oculocephalic
- Gag
- **PALPATION/POSITION**

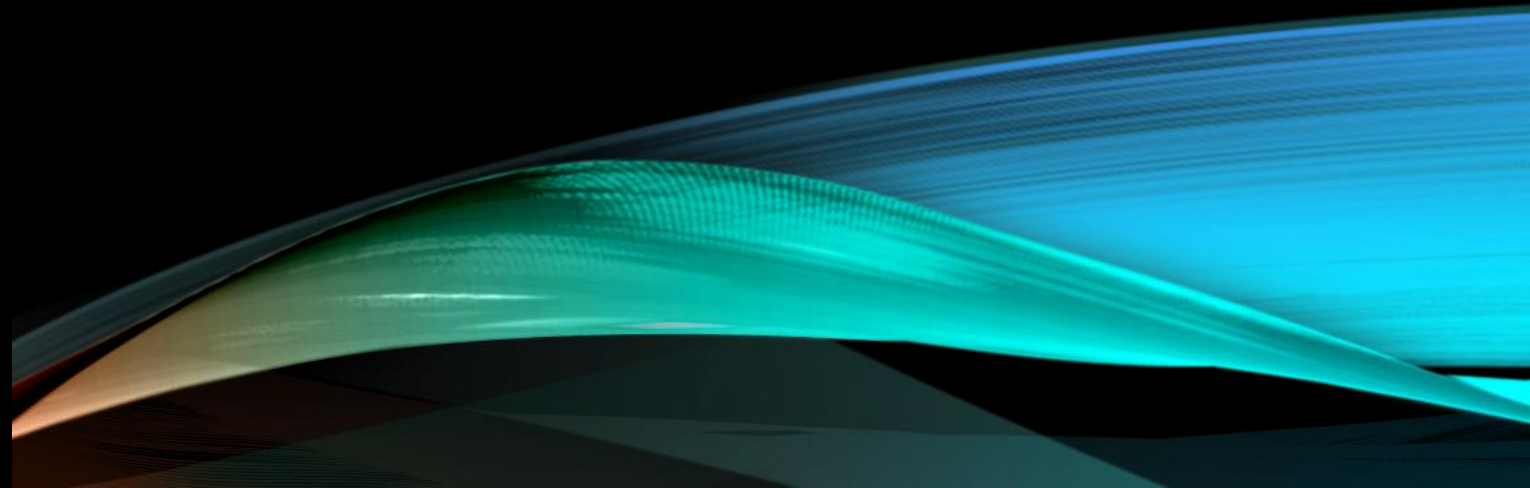




# CRANIAL NERVES

Which tests also assess forebrain function?

- ❖ Menace response
- ❖ Nasal septal response



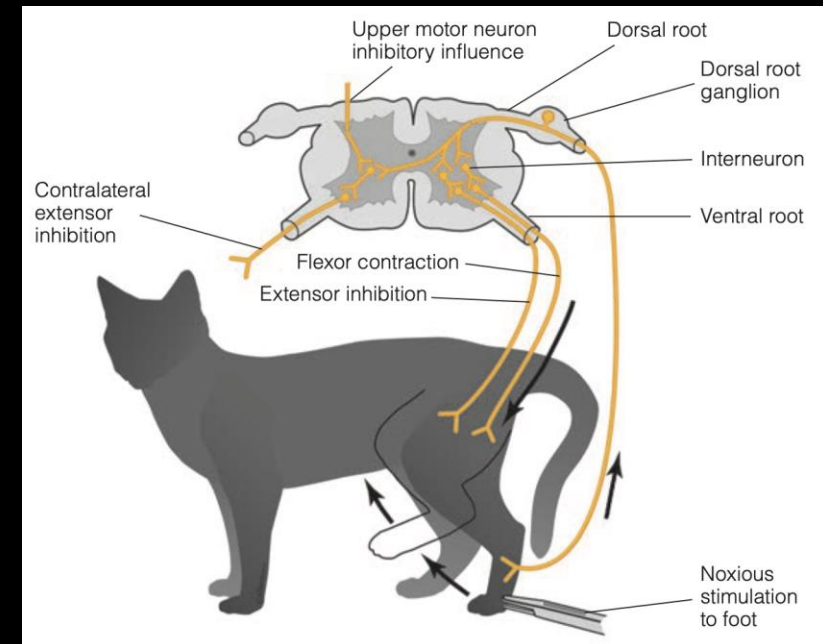
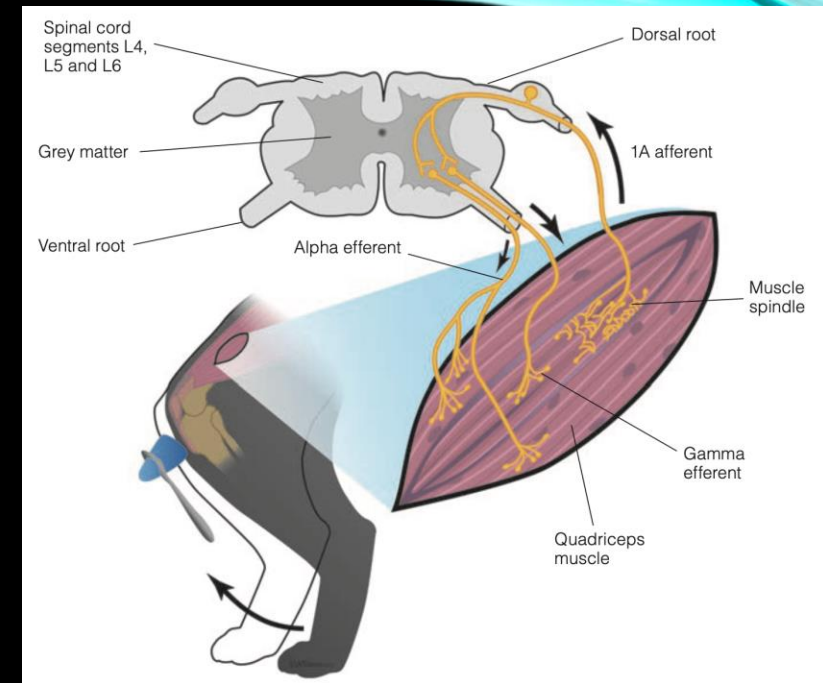
# SPINAL REFLEXES

## THORACIC LIMBS

- Withdrawal
  - Spinal Cord Segments: C6-T2
  - Spinal Nerves: Medial, ulnar, radial, axillary, & musculocutaneous nn.

## PELVIC LIMBS

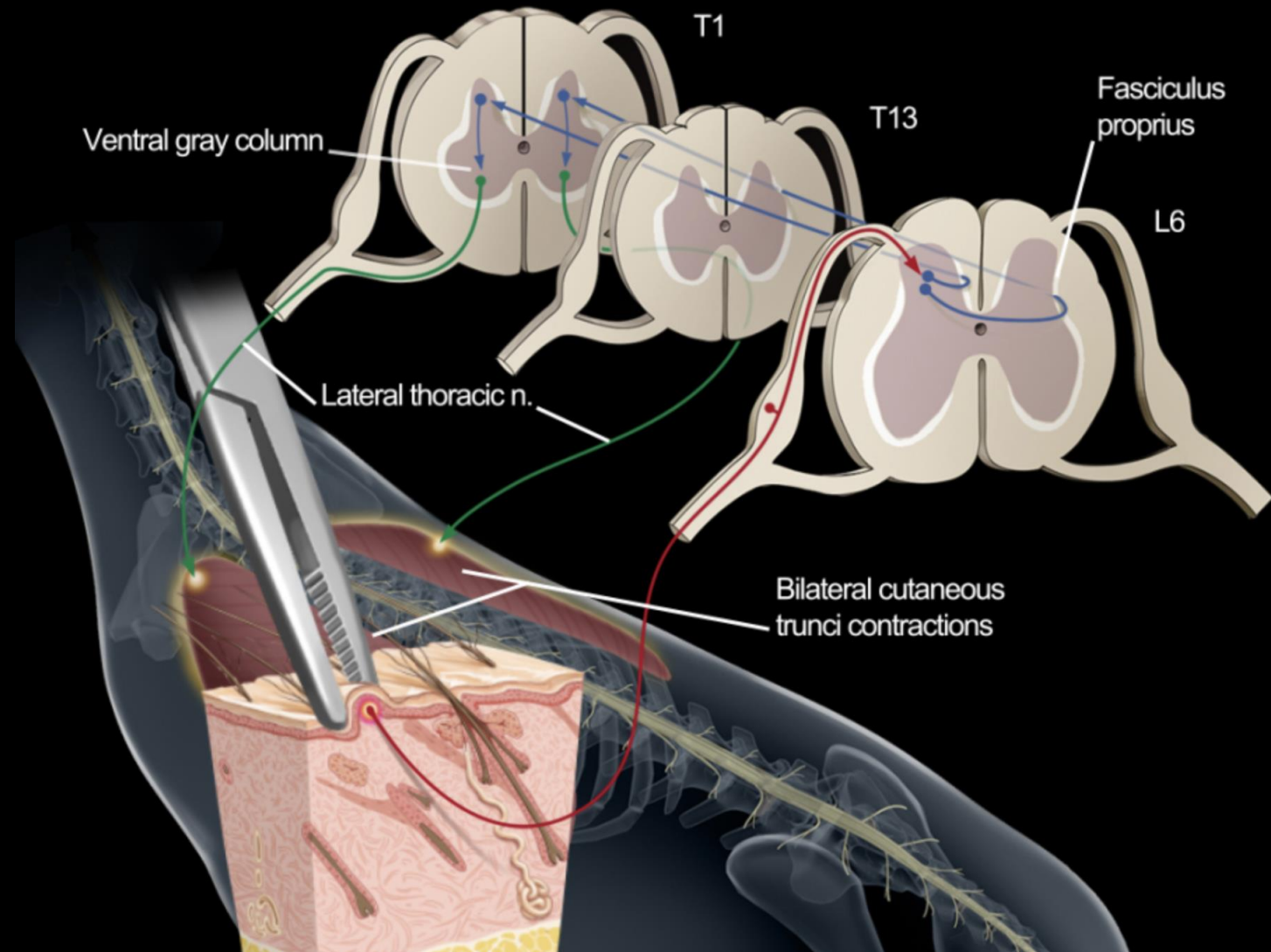
- Patellar
  - Spinal cord segments: L4-L6
  - Nerve: Femoral n.
- Withdrawal
  - Spinal cord segments: L6-S1
  - Nerve: Sciatic n.



# CUTANEOUS TRUNCI

## CUTANEOUS TRUNCI

- Pinch skin from ilial crest to scapula
- Stop once twitch elicited
- Spinal Cord Segments: C8-T1
- Nerve: Lateral thoracic n.
- Helpful for:
  - Lesion localization
  - Monitoring progression



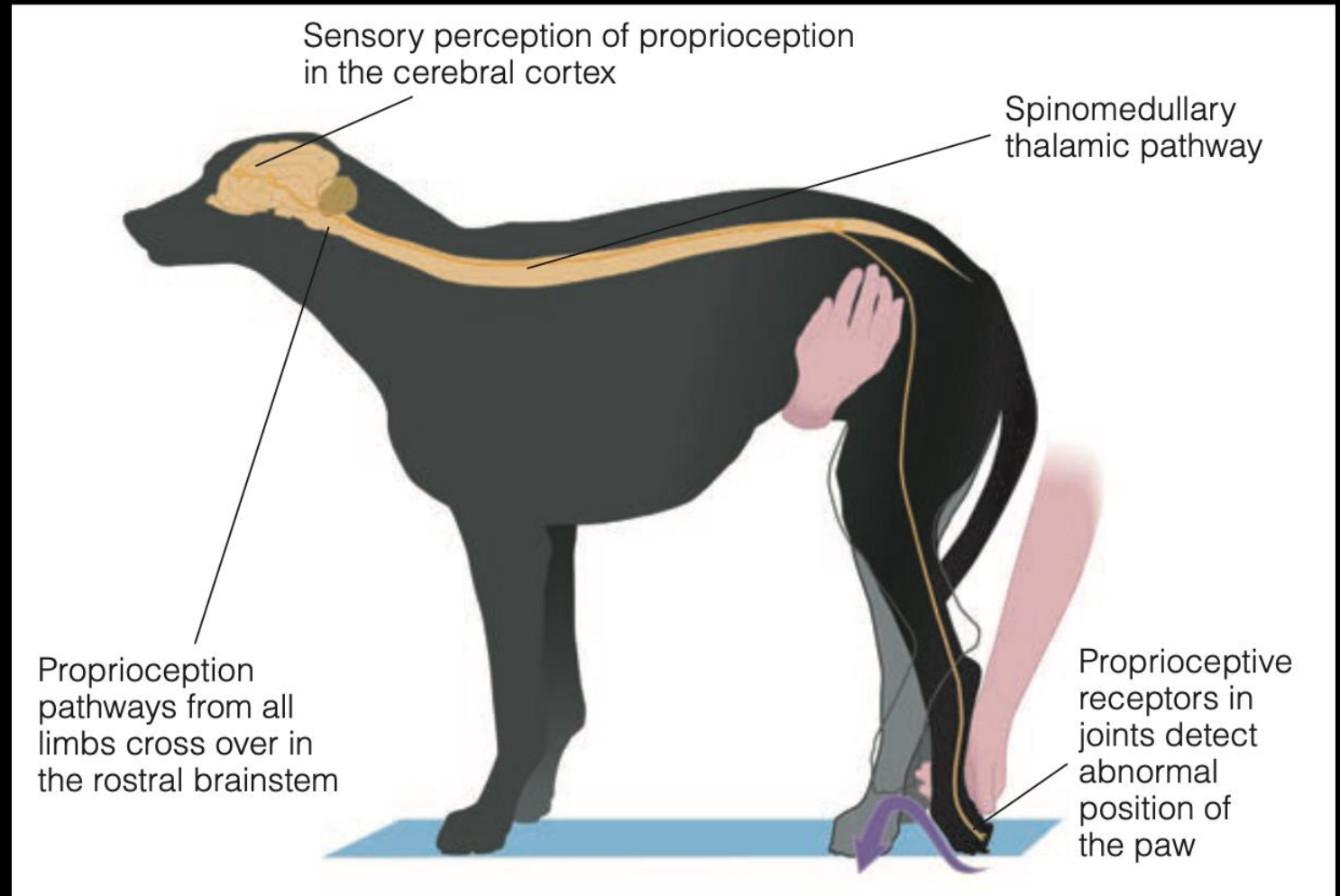
# SPINAL REFLEXES & SPINAL SHOCK

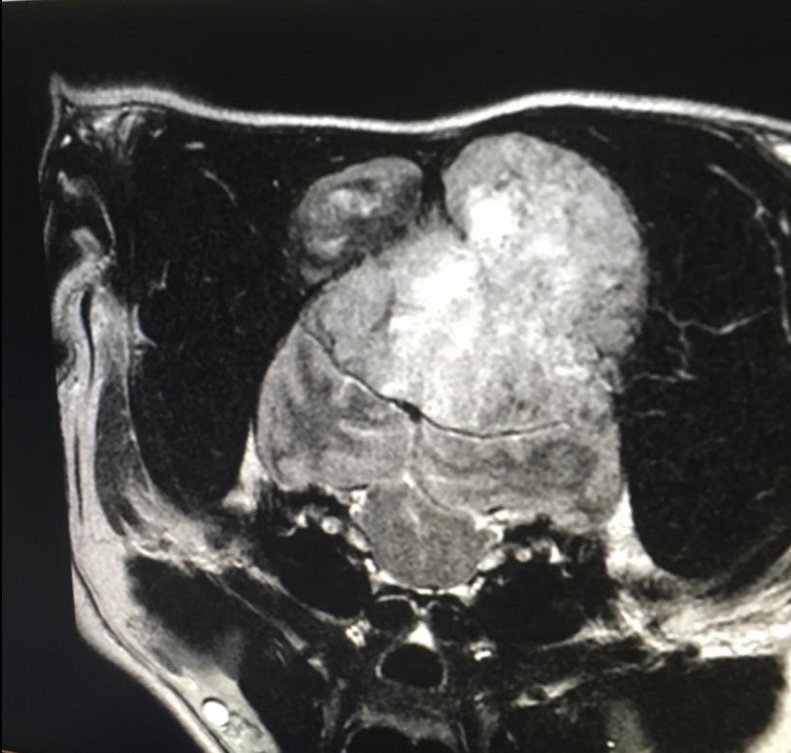
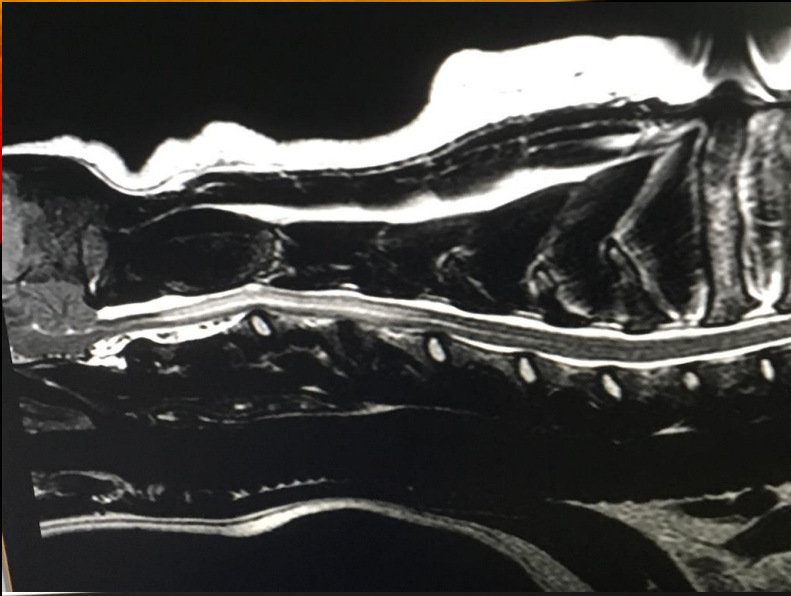
## SPINAL SHOCK

- Reduced spinal reflexes & muscle tone caudal to spinal cord injury
- Clinical variables seen in T3-L3 myelopathy + spinal shock
  - Acute onset (<24 hours)
  - Lower body weight (<10 kg)
  - Paraplegia (vs paresis)
- Associated diagnoses: Acute IVDE (56-90%), FCE (2-33%), ANNPE (8-11%)
- Return of function: anal tone -> patellar -> withdrawal
  - Often within 48 hours

# POSTURAL REACTIONS

- Paw Replacement
- Hopping
- Extensor Postural Thrust
- Wheelbarrow





# PALPATION

- Asymmetry
- Muscle atrophy
- Extensor Tone
- Pain\*\*\*

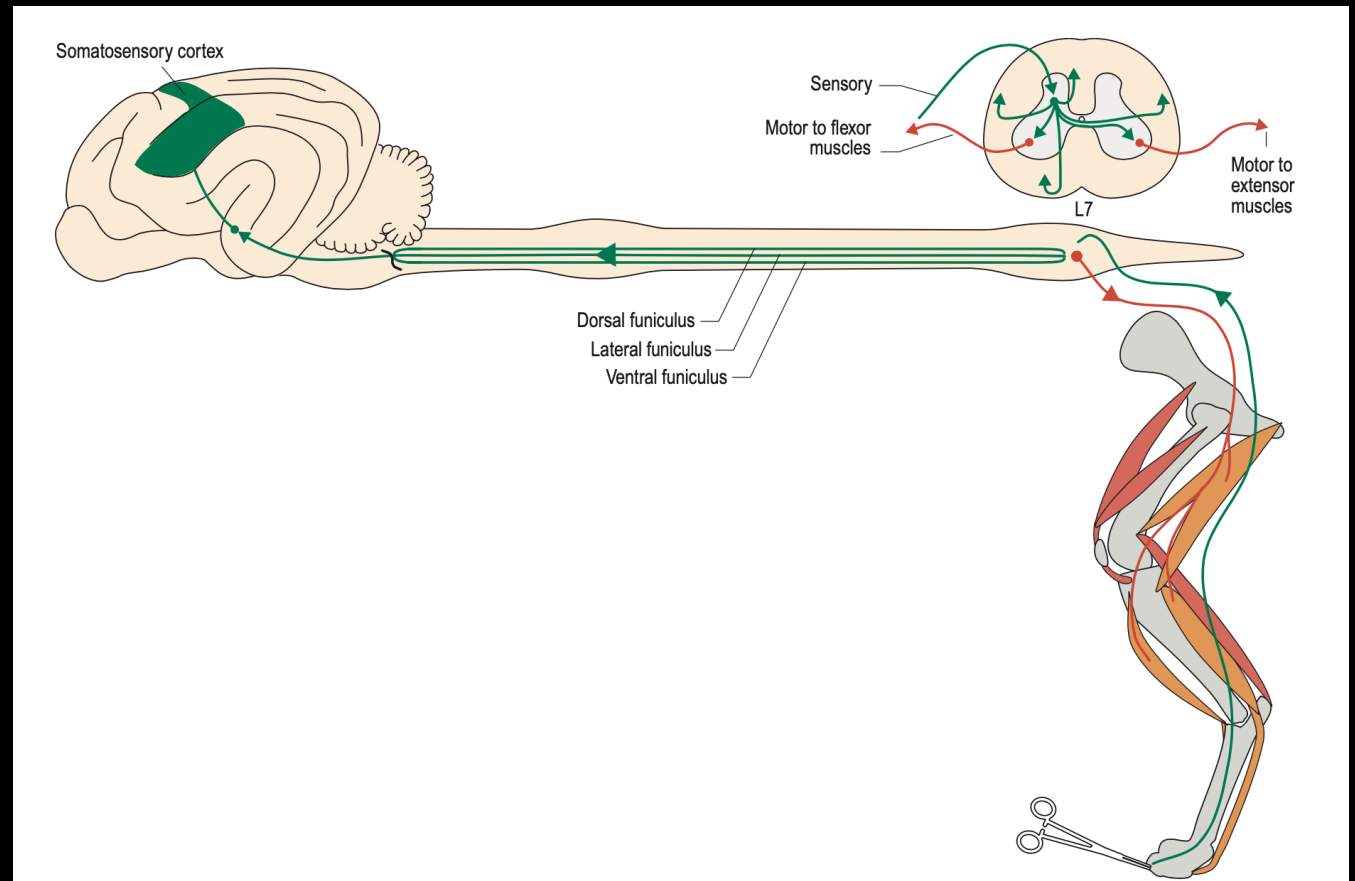
# NOCICEPTION (AKA DEEP PAIN PERCEPTION)

## WHEN TO TEST:

- ONLY in plegic patients

## HOW TO PERFORM:

- Apply force directly over bone in toe
- Look for *RESPONSE* to painful stimulus



# NEUROANATOMIC LOCALIZATION

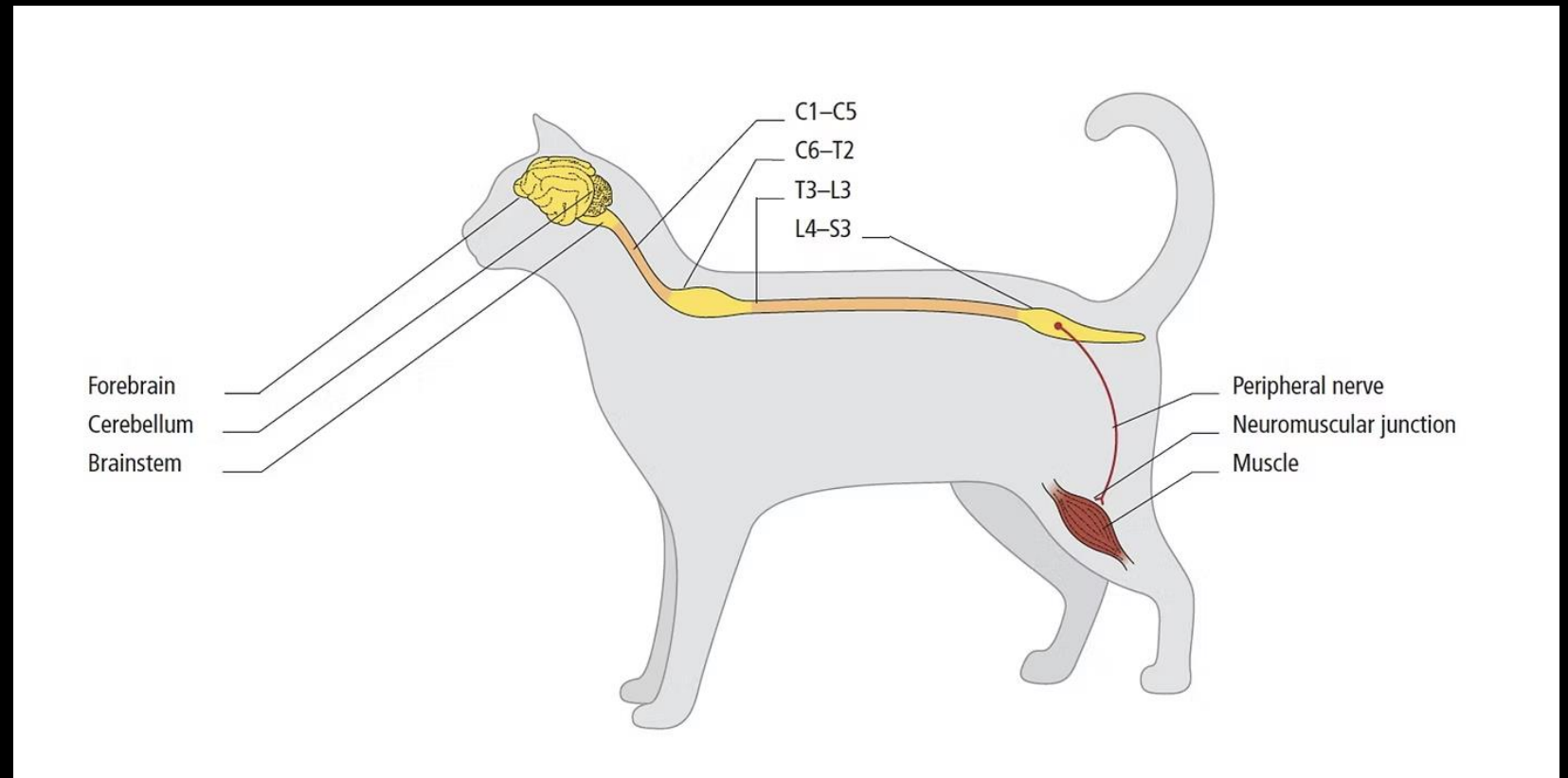
## INTRA-CRANIAL

- Forebrain
- Brainstem
- Cerebellum

## SPINAL CORD

- C1-C5
- C6-T2
- T3-L3
- L4-S3

## NEUROMUSCULAR



# NEUROLOCALIZATION: INTRA-CRANIAL

FINDINGS	FOREBRAIN	BRAINSTEM	CEREBELLUM
MENTATION	Depression, confusion, behavior changes	Stupor/Coma	Normal
CRANIAL NERVES	Contralateral perceptual deficits (absent menace, nasal septal)	Ipsilateral deficits (CN III-XII)	<i>Ipsilateral</i> menace deficit with normal vision
GAIT/POSTURE	<b>NORMAL GAIT</b> Abnormal posture/movement - head pressing, pacing, aimless circling, pleurothotonus	Tetraparesis or hemiparesis/hemiplegia +/- decerebrate rigidity	Intention tremors, hypermetria, truncal ataxia, broad-based stance, decerebellate rigidity
POSTURAL REACTIONS	Deficits in <i>contralateral</i> limbs	Deficits in <i>ipsilateral</i> limbs (or all limbs)	Delayed initiation with exaggerated response
SPINAL REFLEXES	<i>Intact</i>	<i>Intact</i>	<i>Intact</i>
PALPATION	Facial hypoalgesia, +/- cervical pain	Potential cervical pain	Unaltered
OTHER	<b>SEIZURES</b> , cervical pain, hemi-neglect syndrome	<b>Respiratory &amp; cardiac abnormalities (ARAS)</b>	Increased urinary frequency

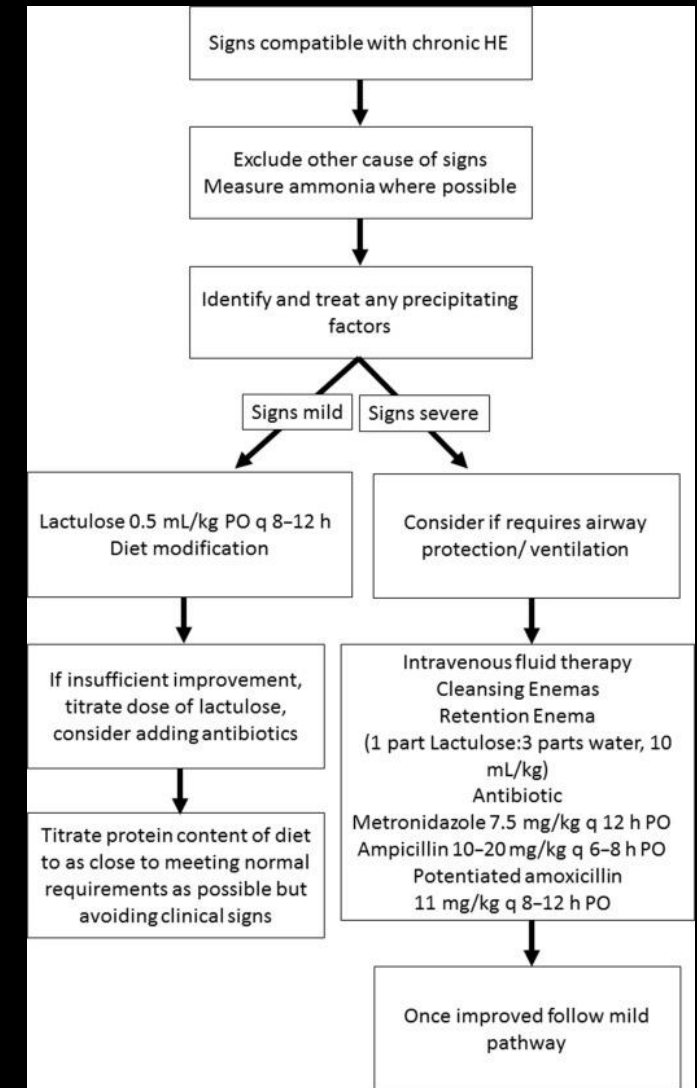
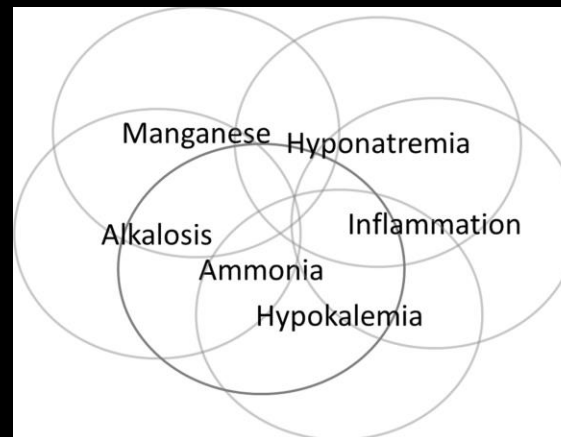
# CLASSIC FOREBRAIN DYSFUNCTION

- DIFFERENTIAL DIAGNOSES?
  - Neoplasia
    - Primary
    - Metastatic
  - Hypertension
    - Renal
    - Hyperthyroidism
  - Infectious
    - FIP, fungal/bacterial/parasitic meningoencephalitis
  - Metabolic/Structural
    - Thiamine deficiency
    - Hepatic encephalopathy



# HEPATIC ENCEPHALOPATHY

- Neurologic dysfunction due to:
  - Liver disease
  - Portosystemic shunt
- Acute HE
  - Acute liver failure
  - Cerebral edema → Increased ICP
  - Grave prognosis
- Chronic HE
  - Most common (CPSS)
  - Diagnostics:
    - Ammonia
    - AUS or CT sca



# ABNORMAL EPISODES: IS IT A SEIZURE?

## DIFFERENTIALS

- Syncope
  - Vestibular event
  - Paroxysmal dyskinesia
  - Narcolepsy/cataplexy
  - Tremorigenic activity
  - Myasthenia gravis
  - Cervical pain
- 
- MAKING THE DISTINCTION
    - Careful history
    - Defined ictal & post-ictal
    - Autonomic signs
    - Lack of consciousness



# WHEN TO BEGIN ANTI-EPILEPTIC DRUG THERAPY

- IVETF Guidelines for AED Initiation

- **Interictal period of  $\leq 6$  months** (i.e. 2 or more epileptic seizures within a 6 month period)
- **Status epilepticus or cluster seizures**
- **The postictal signs are considered especially severe** (e.g. aggression, blindness) **or last longer than 24 hours**
- **The epileptic seizure frequency and/or duration is increasing and/or seizure severity is deteriorating over 3 interictal periods**

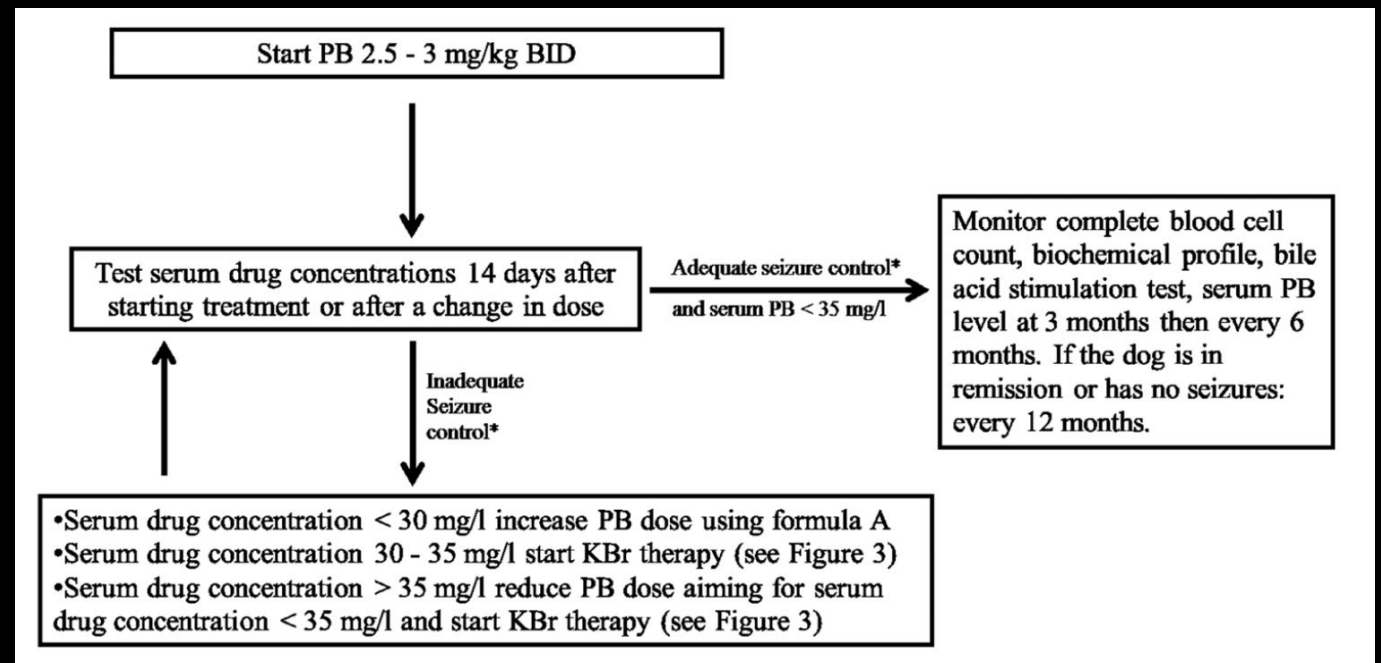


# FIRST-LINE AED THERAPY

- AED selection is multi-factorial:
  - Drug: Adverse effects, administration frequency
  - Patient: Seizure etiology, comorbid disease
  - Client: Lifestyle, finances

- First-Line AED Choices:
  - Phenobarbital
    - 85% seizure free for 6 mo
  - Potassium Bromide
    - 52% seizure free for 6 mo

- Client Education is CRITICAL!
  - Lifelong AED therapy
  - Expected adverse effects
  - Monitoring schedules & cost



$$\left( \frac{\text{Desired concentration}}{\text{Actual concentration}} \right) \times \text{total mg phenobarbital per day} = \text{Oral daily dose of phenobarbital (mg)}$$

# AED THERAPY: ADJUNCT TREATMENT OPTIONS

- LEVETIRACETAM

- 20-30 mg/kg q 8-12 hr
- Few adverse effects, no monitoring required
- Used as adjunct, monotherapy, & rescue
- Pulse therapy in cluster seizures

- ZONISAMIDE

- 5 mg/kg PO q 12 hr
- Used as adjunct and monotherapy
- Adverse effects: Vomiting, ataxia, inappetence
- Rare toxic idiosyncratic reactions
  - Renal tubular acidosis, acute hepatopathy

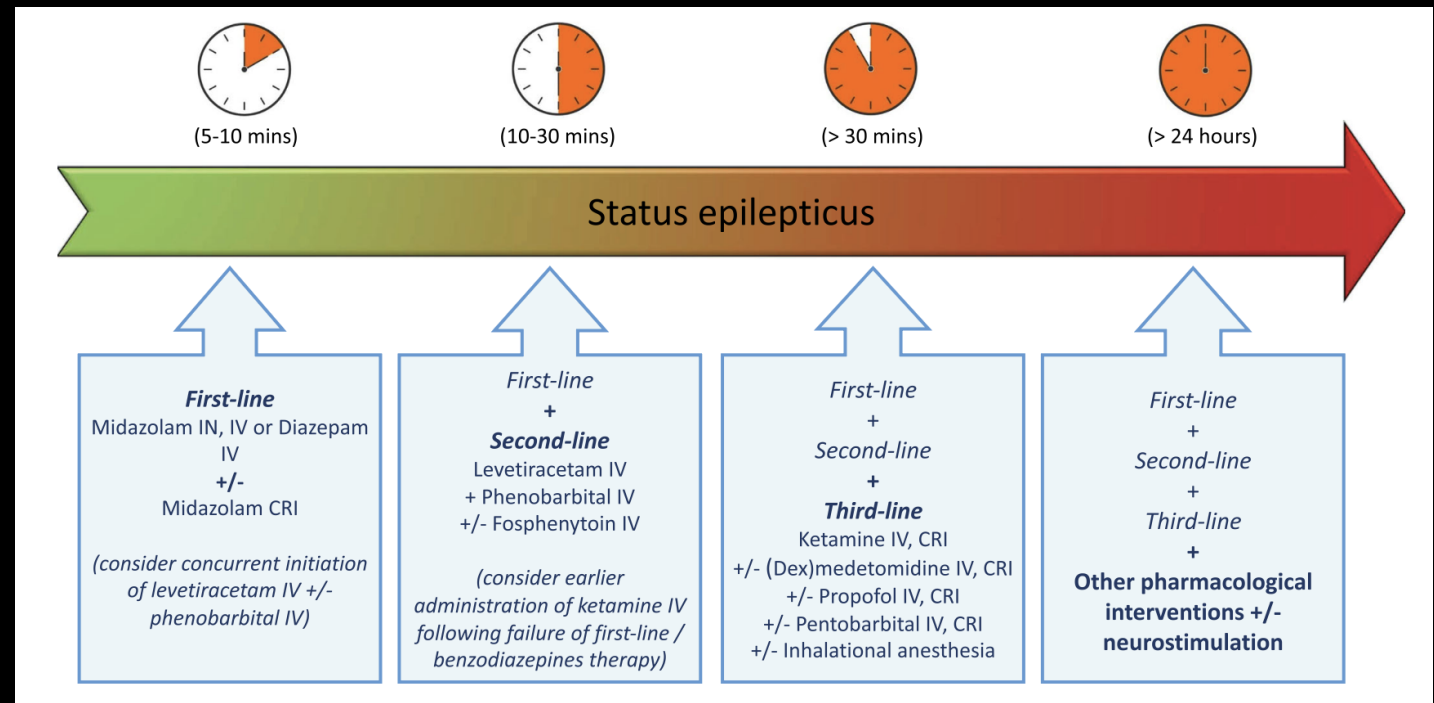
# EMERGENCY SEIZURE MANAGEMENT

- **STATUS EPILEPTICUS:**

- Seizure  $\geq 5$  minutes
- Overall mortality: 25-38.5%
- 20-60% of IE dogs exhibit SE
- Immediate care required
  - Risk permanent brain damage

- **CLUSTER SEIZURES:**

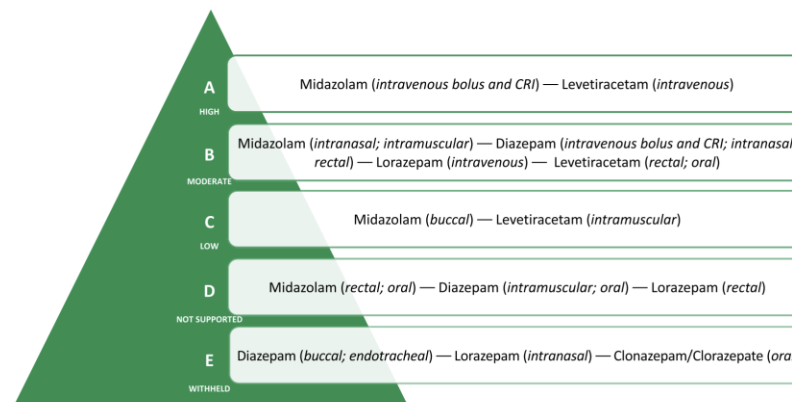
- $\geq 2$  seizures in 24 hours
- Incomplete recovery between episodes



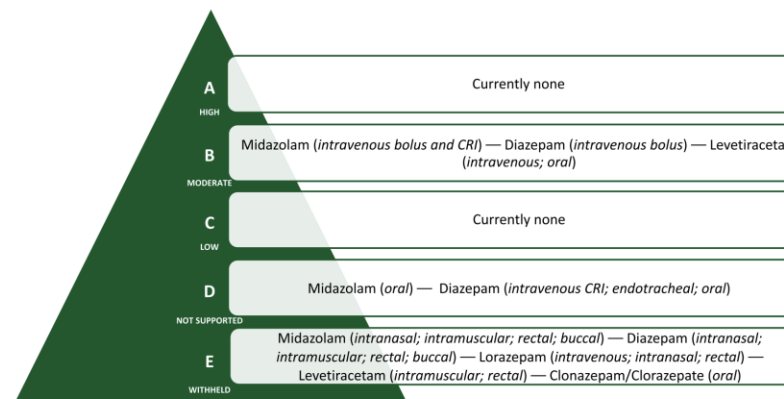
# TREATMENT OF CLUSTER SEIZURES



ACVIM pyramid of hierarchy regarding antiseizure therapy recommendations for **cluster seizures in dogs**



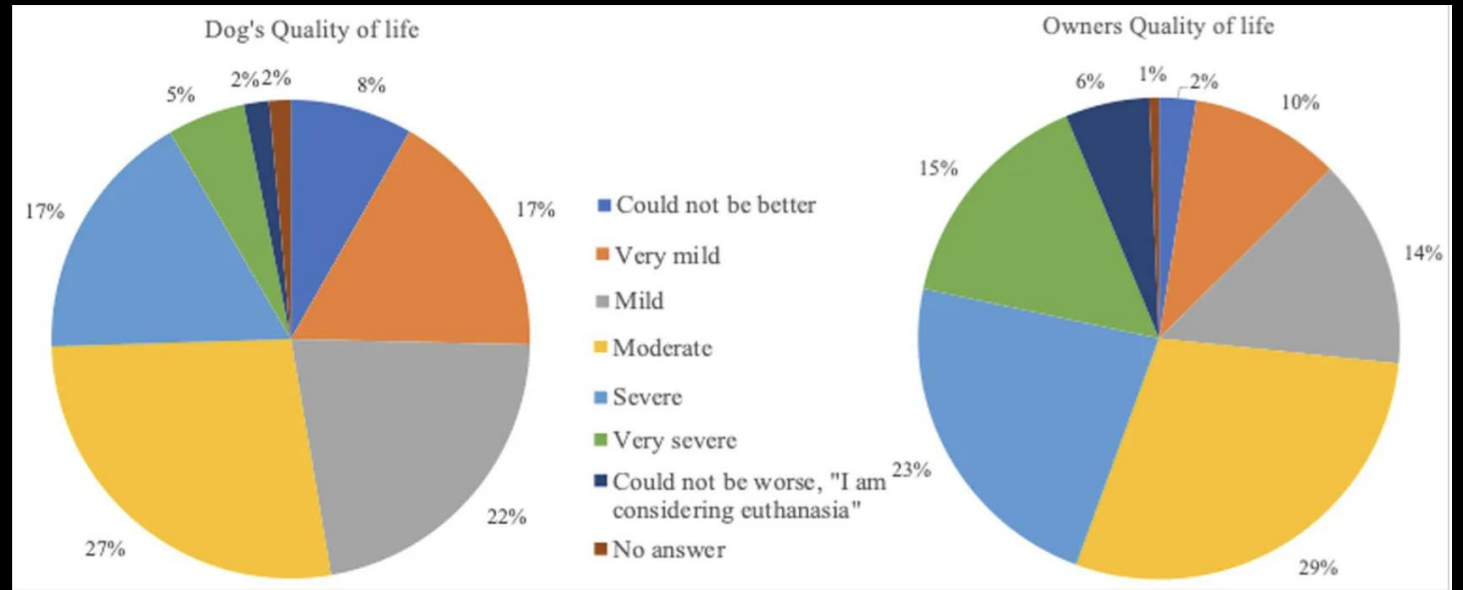
ACVIM pyramid of hierarchy regarding antiseizure therapy recommendations for **cluster seizures in cats**



# SEIZURE MANAGEMENT & QUALITY OF LIFE

## • TREATMENT GOALS

- Significant reduction in
  - Seizure frequency
  - Episode severity
- Prevent cluster seizures & status epilepticus
- Improve patient & family quality of life

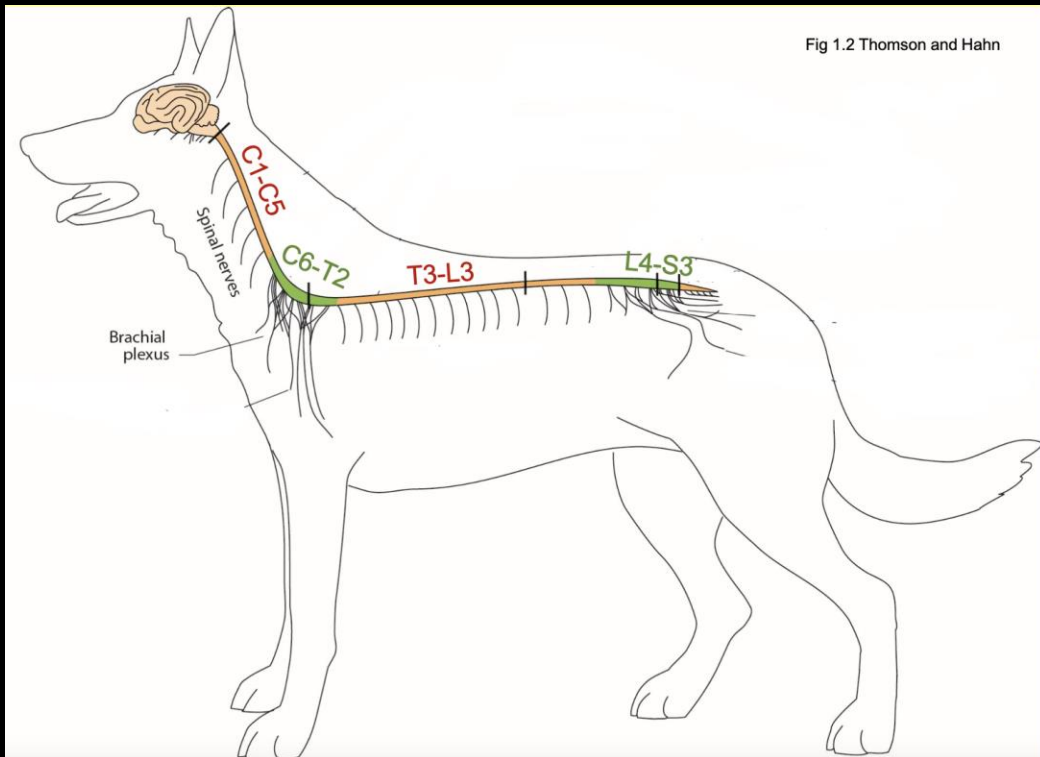


# BRACHYCEPHALICS & SEIZURES

- Brachycephaly = Risk factor for structural epilepsy
- MRI & CSF analysis recommended
- Harris et al. 2025
  - Structural Lesions on MRI
    - 34.2% of dogs
      - 61.8% brachycephalics
      - 22.1% non-brachycephalics
  - Structural Epilepsy (6 mo to 6 yo dogs)
    - 33.3% brachycephalics
    - 0% non-brachycephalics



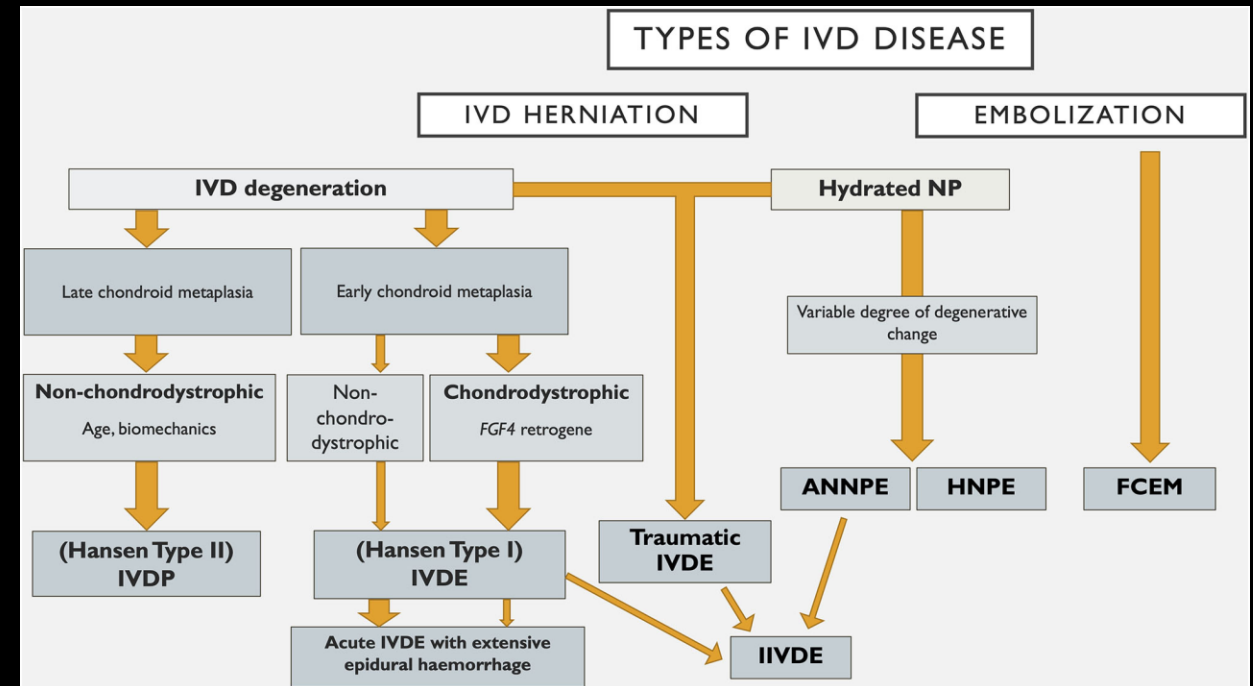
# NEUROLOCALIZATION: SPINAL CORD SEGMENTS



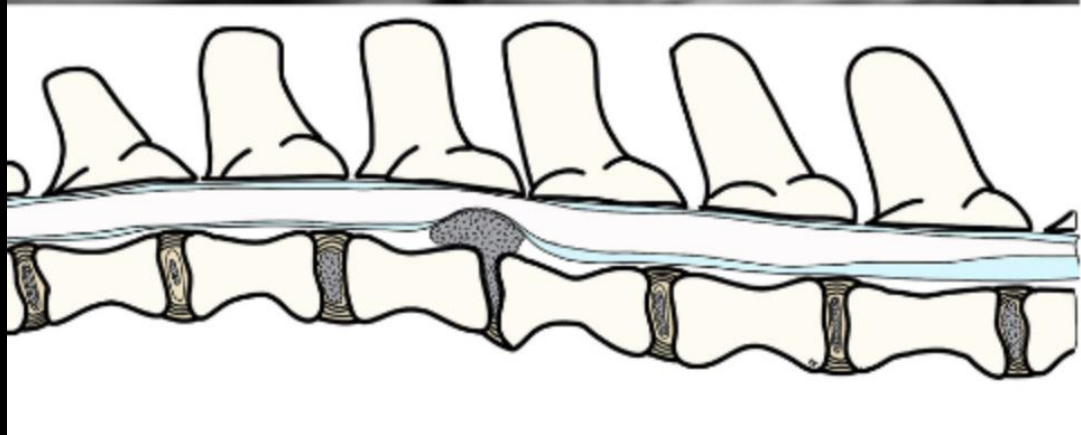
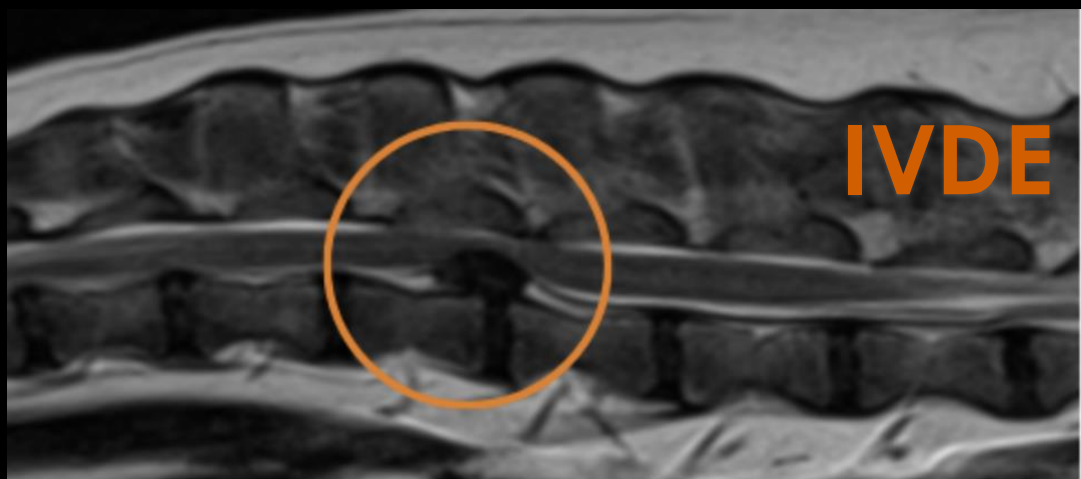
EXAM FINDINGS	C1-C5	C6-T2	T3-L3	L4-S3
Gait/Posture	Tetraparesis /plegia	Tetraparesis/plegia	Paraparesis /plegia	Paraparesis /plegia
Spinal Reflexes	INTACT	REDUCED/ ABSENT (TL)	INTACT	REDUCED/ ABSENT (PL)
Postural Reactions	Delayed to absent x 4	Delayed to absent x 4	Delayed to absent (PL)	Delayed to absent (PL)
Palpation	+/- Cervical pain	+/- Cervical pain	+/- TL pain	+/- TL pain
Muscle Tone, Other	Potential neck rigidity	Reduced extensor tone +/- ipsilateral Horner's	Increased extensor tone	Reduced extensor tone +/- flaccid tail & anus

# SCI: DIFFERENTIAL DIAGNOSES

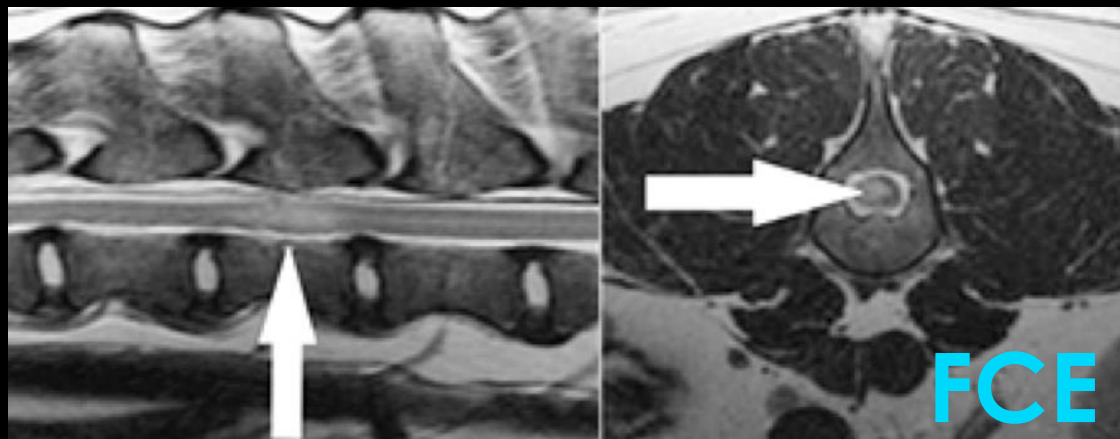
- **DEGENERATIVE**
  - IVD Extrusion/Protrusion
- **ISCHEMIC**
  - Fibrocartilagenous Embolism (FCE)
  - Acute Non-Compressive Nucleus Pulposus Extrusion (ANNPE)
- **TRAUMA**
  - Vertebral fracture/luxation
  - Pathologic fracture (OSA)
- **ANOMALOUS**
  - Atlantoaxial Instability
  - Cervical Spondylomyelopathy (Wobbler's)
  - Congenital Vertebral Malformations
    - Pugs, Frenchies
- **INFLAMMATORY/INFECTIOUS**
  - Discospondylitis
  - Steroid-responsive meningitis-arteritis (SRMA)



# SO WHAT DOES IT ALL MEAN?



**SURGICAL**



**NON-SURGICAL**



# IVDE: CERVICAL VS THORACOLUMBAR

- **CERVICAL HYPERAESTHESIA**

- SEVERE neck pain
- Cervical muscle fasciculations
- Low head carriage +/- kyphosis
- Spontaneous vocalization
- May have NO neurologic deficits

- **THORACOLUMBAR MYELOPATHY**

- Pain on spinal palpation
- Some degree of myelopathy:
  - Postural reactions
  - Paraparesis/plegia
  - +/- Spinal reflexes (L3-S3)

# PUTTING IT INTO PRACTICE (NO MRI REQUIRED)

- ANNPE:
  - *PERACUTE* onset, lateralized (90% dogs)
  - Develops during exercise or trauma
  - Post-trauma in cats (75%)
  - NON-PROGRESSIVE after first 24 hr
  - Thoracolumbar region most affected
  - Mild to NO PAIN on spinal palpation
- FCE:
  - *PERACUTE* onset, lateralizing signs
    - Medium/large breed during exercise (50%)
    - Non-chondrodystrophic breeds
  - NON-PROGRESSIVE after 24 hr
  - Cervical or thoracolumbar localization
  - Giant breeds, plegic w/absent DPP → GRAVE



Full neurologic exam **BEFORE** analgesia  
Drugs may impact findings!

# FCE/ANNPE: WHEN TO HOSPITALIZE/REFER

- Severity of myelopathy
  - Inability to stand/walk → Inability to void
  - ALL non-ambulatory patients require:
    - Bladder management q 8h
    - Recumbency care
    - Physiotherapy
- Degree of Discomfort
  - If >48 hr, reconsider DDx
- Monitor neurologic progression
- Advanced Imaging
  - Diagnostic/Prognostic Value

# FCE/ANNPE PROGNOSIS

- TIMELINES DIFFICULT TO PREDICT
  - Some recumbent patients are walking in 24-48 hrs
  - Others require weeks to months of physical therapy
- MRI PROGNOSTIC INDICATORS
  - Smaller lesion → more rapid recovery from ANNPE
  - Lesion > 67% (FCE) or 90% (ANNPE) cross-sectional SC area
    - Grave prognosis for return to function
- BEST PROGNOSTIC INDICATOR = NOCICEPTION

# NOCICEPTION IN FCE/ANNPE: PROGNOSTIC VALUE

Grade		Ambulatory paraparetic	Non-ambulatory paraparetic	Paraplegic with DPP	Paraplegic with NDPP
ANNPE (22, 23)	Amb	100% (n = 84)	100% (n = 105)	100% (n = 40)	Unknown
	UC	96.9% (n = 65)	91.1% (n = 90)	82.1% (n = 28)	Unknown
		100% (n = 19)	100% (n = 15)	92% (n = 12)	
	FC	92.3% (n = 65)	75.7% (n = 90)	46.4% (n = 28)	Unknown
		100% (n = 19)	93% (n = 15)	58% (n = 12)	
	FCEM/ ischemic myelopathy (24–26)	Amb		87.5% (n = 301)	
UC			99% (n = 40)		Unknown
				70.4% (n = 51)	
FC			97% (n = 40)		Unknown
			59.3% (n = 51)		

ANNPE, acute non-compressive nucleus pulposus extrusion; FCEM, fibrocartilagenous embolic myelopathy; Amb, ambulatory; UC, urinary continence; FC, fecal continence; NDPP, no deep pain perception; DPP, deep pain perception.

# NOCICEPTION IN IVDE: PROGNOSTIC VALUE

Grade	Overall recovery with conservative management (%)	Overall recovery with surgery (%)	Recovery at 2 w (%)	Recovery at 4–6 w (%)	Recovery at 3 m (%)	Development of PMM (%)
Ambulatory paraparetic	72.5 (n = 116)	98.4 (n = 318)	84	92	93.9	0
Non-ambulatory paraparetic	79.8 (n = 74)	93 (n = 341)	77.8	88.9	92.8	0.6
Paraplegic with DPP	56 (n = 77)	93 (n = 548)	70.8	78.2	83.2	2.7
Paraplegic NDPP	22.4% (n = 48)	61 (n = 502)	26.5	42.3	53.8	13.9

w, weeks; m, months; PMM, progressive myelomalacia; NDPP, no deep pain perception; DPP, deep pain perception (10, 12–21).

# NOCICEPTION & MYELOMALACIA

Fatal spinal cord necrosis

Estimated 10-15% of paraplegic dogs with absent nociception

No definitive ante-mortem diagnostic test

## CLINICAL SIGNS

- Flaccid paralysis
- Ascending cutaneous trunci
- Respiratory paralysis

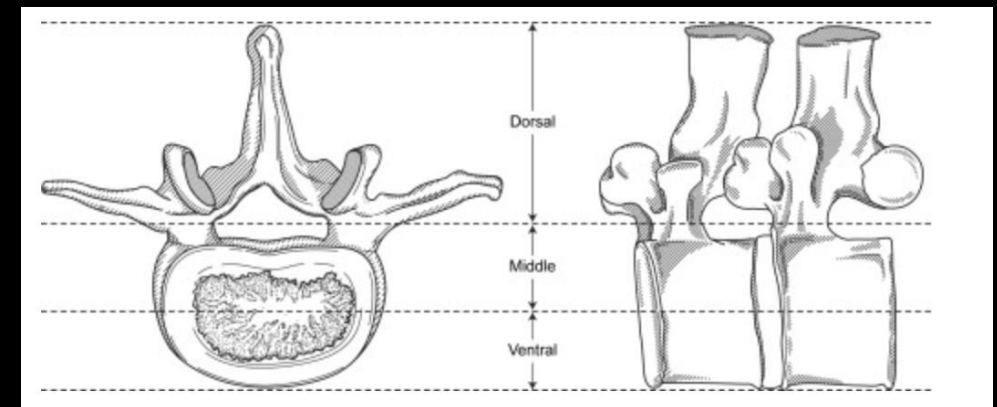
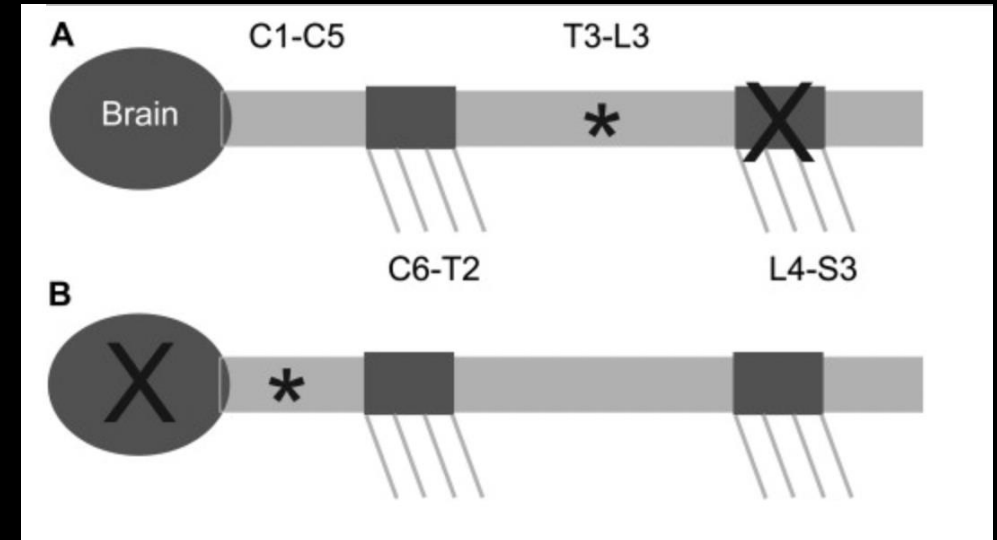
**TABLE 9** | Association of spinal cord hyperintensity detected on T2 weighted MRI in dogs with intervertebral disc extrusion and outcome.

Study	Design and population	Magnet strength	MR imaging changes	Conclusion	Comments
Ito et al. (79)	Retrospective 77 dogs Dogs paraplegic Blinded study Morphologic and morphometric	0.3T	Only dogs with T2W SC hyperintensity equal to L2 vertebra	Hyperintensity better predictor of outcome than absence of DPP	Large variation between onset signs and MRI—median 7d
Levine et al. (53)	Retrospective 129 dogs with follow-up Ambulatory and non-ambulatory dogs Blinded study Morphologic and morphometric	1.0T for the majority of cases	Any degree of T2W SC hyperintensity	Direct association between the length ratio of hyperintensity with long-term functional outcome	The large variation in injury severity makes comparison with other studies challenging
Boekhoff et al. (80)	Retrospective 63 dogs All dogs paraplegic Blinded study Morphologic and morphometric	1.0T	SC T2W hyperintensities ranging from half of L2 vertebra to >2 times L2	Association between extent of T2 hyperintensity with delayed ambulation, not statistically significant	Large variation between onset signs and MRI—62% between 2 and 7 days
Wang-Leandro et al. (81)	Prospective 35 dogs All dogs paraplegic Blinded study Morphologic and morphometric	3.0T	SC T2W hyperintensities assessed in sagittal plane using L2 vertebra as reference	Length of SC T2 hyper-intensity had no association with motor functional recovery	Only 2 dogs were enrolled with >7 days of onset of signs
Otamendi et al. (82)	Retrospective 47 dogs	3.0T	SC T2W hyperintensities assessed in sagittal plane using L2 vertebra as reference	No association between T2 hyperintensity and recovery of motor function or PMM	Abstract only

SC, spinal cord; PMM, progressive myelomalacia.

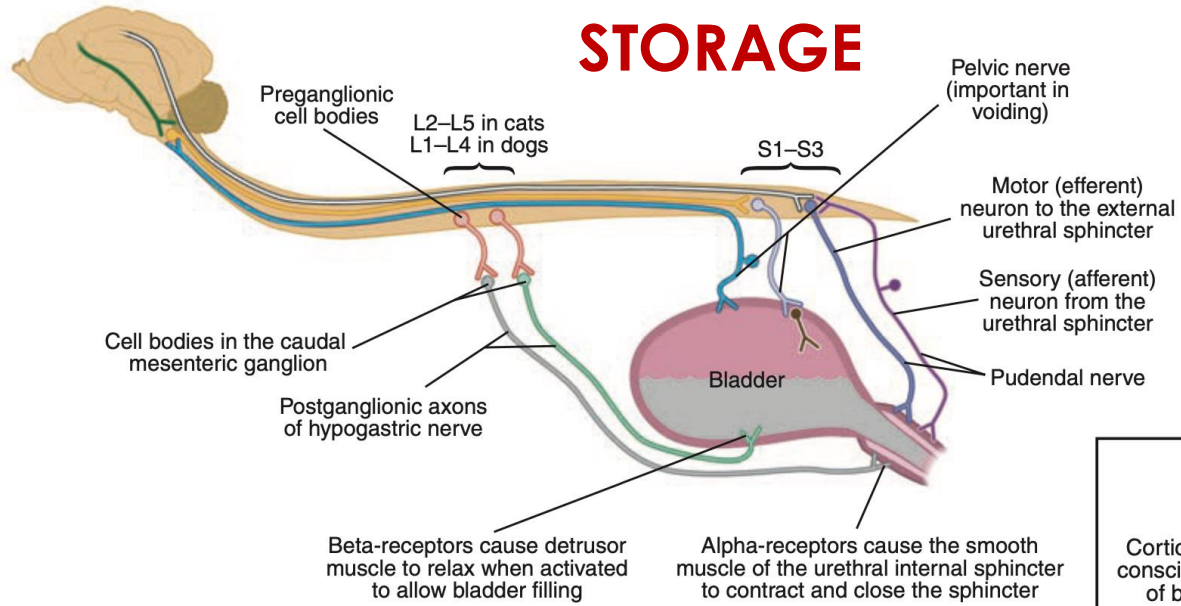
# SPINAL FRACTURE & NOCICEPTION

- History of trauma
- Neuro Exam
  - Often multi-focal signs
  - T10-L2 in 50%
- Spinal X-rays
  - Lateral spinal survey
  - V/D of any suspected fracture
  - Extreme care w/sedation
- Treatment: Surgery vs Conservative
  - Depends on vertebral instability
- Plan for safe transport
  - Strap to spinal board
- **Absent nociception → Grave prognosis**
  - Euthanasia often advised

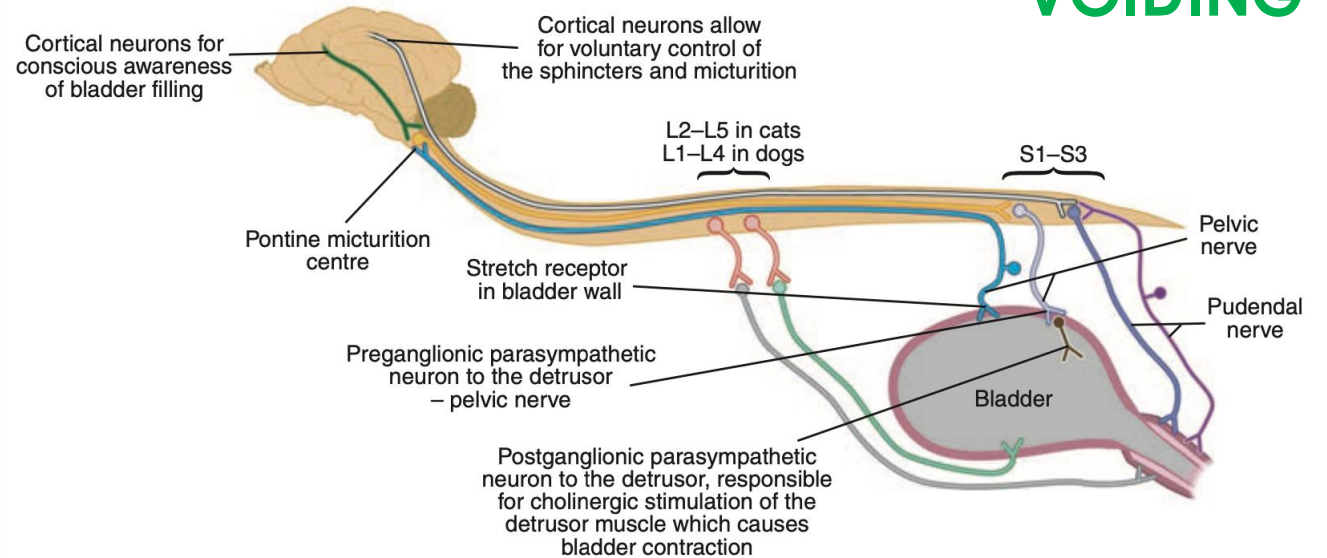


# SPINAL CORD INJURY & BLADDER FUNCTION

## STORAGE



## VOIDING



# SPINAL CORD INJURY & BLADDER FUNCTION

Lesion localization	Conscious voiding attempts	Bladder expression/size	Residual urine volume	Perineal reflex	Type of micturition dysfunction
Cerebral cortex to brainstem	Absent	Difficult/small	Small	Present	Inappropriate urination
Cerebellum	Normal; increased frequency	Difficult/small	Small	Present	Detrusor muscle hyperreflexia
Brainstem to L7 <b>UMN</b>	Absent; dyssynergia	Difficult/large; tapering stream; small spurts of urination	Moderate to large	Present	Failure to eliminate urine; reflex incontinence; overflow incontinence
Sacral spinal cord <b>LMN</b>	Absent; may attempt but limited success	Easy; leakage; may have some resistance/large	Large	Reduced to absent	Urethral incompetence; detrusor muscle atony; overflow incontinence
Disruption of tight junctions to the detrusor muscle	Absent	Some resistance; bladder is flaccid/large	Large	Present	Overflow incontinence

# NEUROLOCALIZATION: NEUROMUSCULAR OVERVIEW

## **RADICULOPATHY**

- Nerve root signature, limb paresis/paralysis, generalized weakness, focal/diffuse muscle atrophy, dysphonia
- Ex: Polyradiculoneuritis, brachial plexus avulsion

## **PERIPHERAL NEUROPATHY**

- Paresis/paralysis, severe muscle atrophy, muscle fasciculations, dysphonia, reduced/absent reflexes
- Ex. Leonberger polyneuropathy (LPN), GOLPP

## **JUNCTIONOPATHY**

- Abnormal neurotransmission at the neuromuscular junction
- Ex: Myasthenia gravis (acquired, congenital, fulminant), tick paralysis, botulism, toxins (OP, carbamate)

## **MYOPATHY**

- Generalized or focal weakness, stiff/stilted gait, muscle atrophy, dysphonia, trismus, usu normal reflexes
- Ex: Centronuclear myopathy, masticatory myositis

# POLYRADICULONEURITIS



# MYASTHENIA GRAVIS





ANY  
QUESTIONS?

# RECOMMENDED READING & RESOURCES

**SEIZURE MANAGEMENT:** International Veterinary Epilepsy Task Force

- [ivetf.org](http://ivetf.org)

**INTERVERTEBRAL DISC DISEASE:** Canine Spinal Cord Injury Consortium (CANSORT – SCI)

- <https://www.frontiersin.org/research-topics/11439>

**FUNCTIONAL NEUROANATOMY:** University of Georgia College of Veterinary Medicine Clinical Neurology & Functional Neuroanatomy Website

- <https://vmmerc.uga.edu/CranialNerves/index.html>

**NEUROLOGY CASE VIDEOS:** de Lahunta's Veterinary Neuroanatomy & Clinical Neurology Case Videos

- <http://www.neurovideos.vet.cornell.edu/>

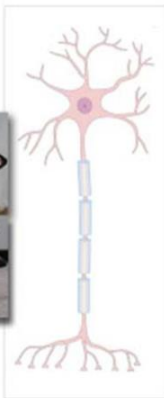
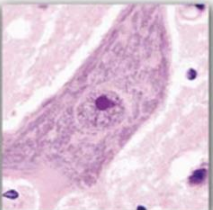
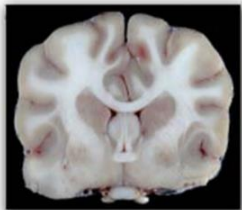
# RECOMMENDED READING & RESOURCES



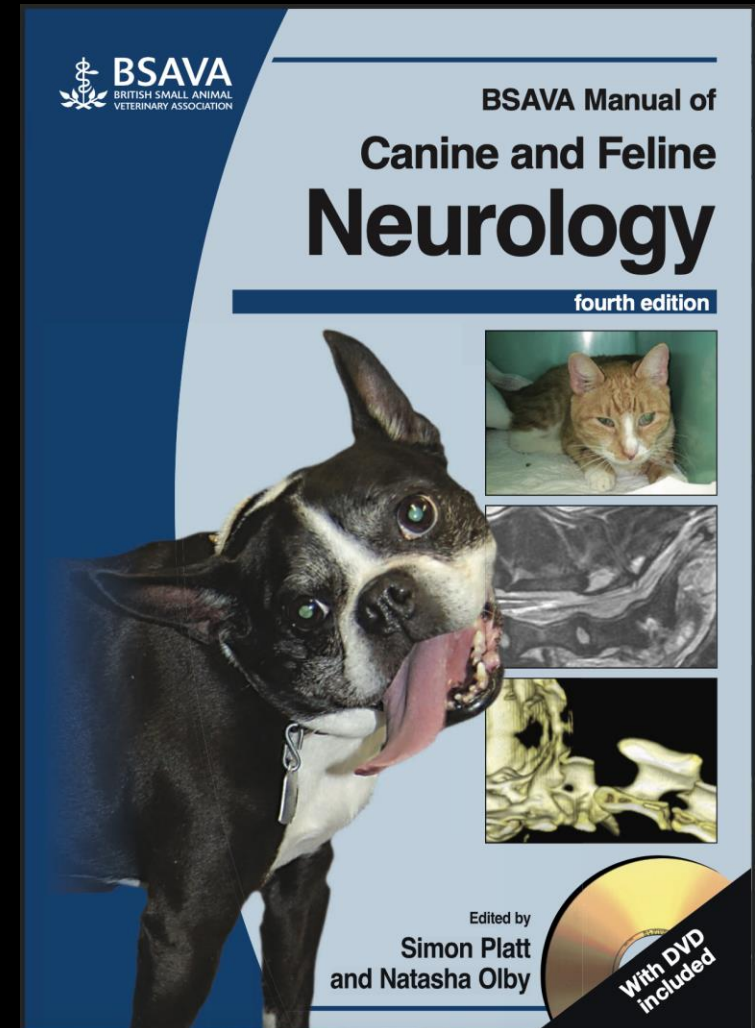
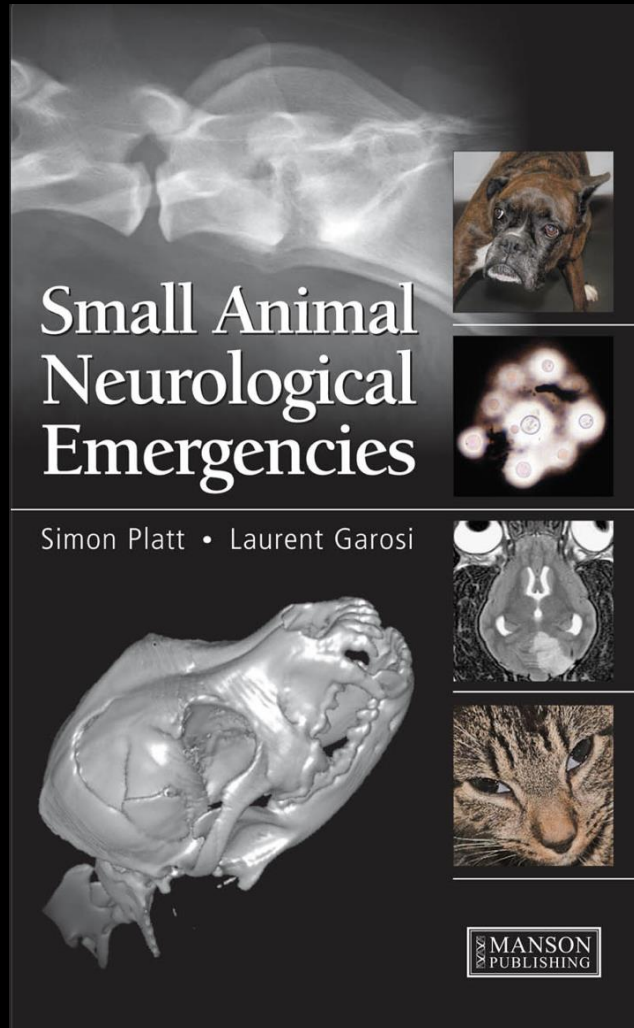
## Veterinary Neuroanatomy

A Clinical Approach

Christine Thomson  
Caroline Hahn



SAUNDERS  
ELSEVIER



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