If the patient suddenly does funny things

seizures and beyond

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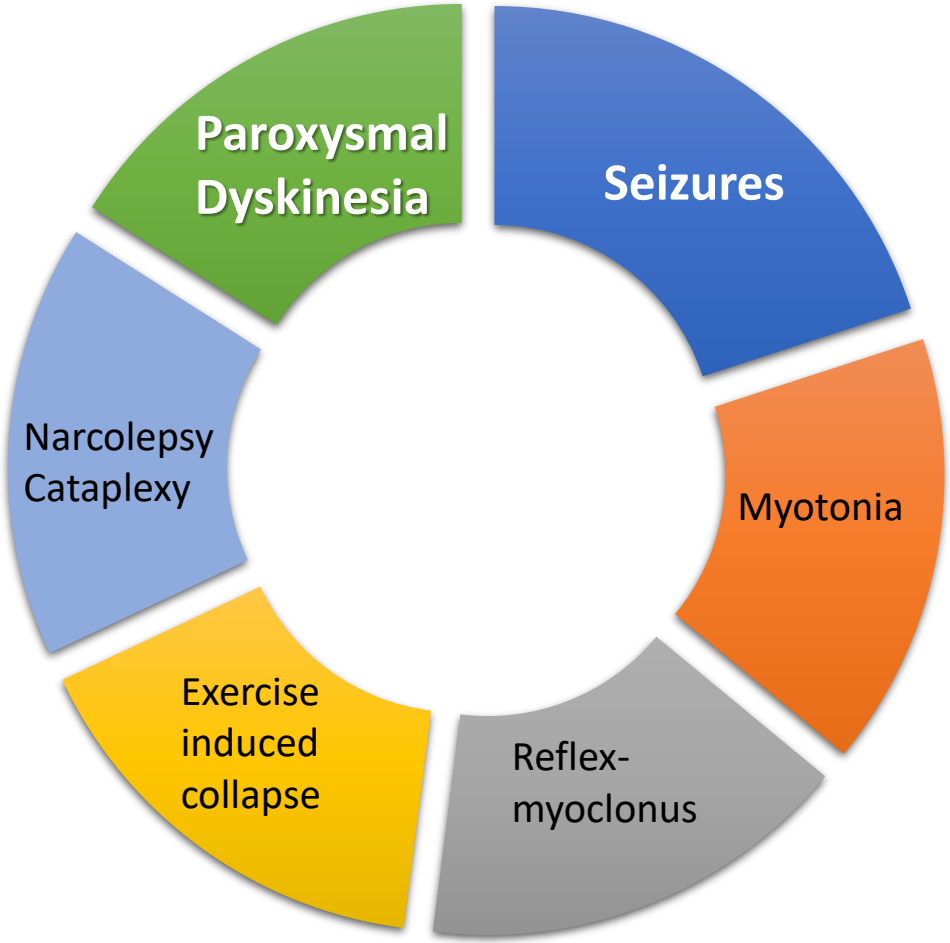
The big dilemma of sudden episodes

- it starts suddenly
- it looks funny
- it is there for a short time only
- and it disappears again



Clinical characteristics

- can start from any situation
- paroxysmal uncontrolled motor activity (increased, decreased)
- **mentation usually normal**
- **rarely autonomic signs** (salivation, urination, defecation)
- **no pre- and post ictal signs**
- **self limiting**
- **varying duration and frequency**
- **sometimes additional GI signs**

- usually starts from episodes of rest
- sudden uncontrolled motor activity (increased, reduced)
- **often abnormal mentation**
- often **autonomic signs** (salivation, urination, defecation)
- **pre and postictal signs**
- **individual pattern**
- can result in status epilepticus

Terminology of epileptic seizures

International Epilepsie Task Force consensus proposal (2015)

Epilepsy (cause)

Structural

structural brain disease:

inflammation, infection, trauma, malformation, infarct, neoplasia, storage disease

Idiopathic

no obvious brain pathology

(genetic, probably genetic, unknown reason)

Kryptogenic

you expect structural epilepsy, but you can not find it witch imaging and CSF tap

Reactive epileptic seizures

- reaction of a normal brain to a metabolic disturbance
- cause is outside of brain
- often seizures stop, when underlying pathology has been treated

Underlying pathology: < 6 months

V	infarct, hemorrhage, vascular malformation, hypertension
I	immune mediated, viral, bacterial, protozoal, fungal, rickettsial
T	traumatic brain injury (posttraumatic), toxic
A	hydrocephalus, arachnoid cysts, lissencephaly, abnormal gyration, corpus callosum agenesia
M	hypoglycemia (insulinoma), hepatoencephalopathy, severe uremia, hypocalcemia, polycytemia, hyperviscosity ...
I	idiopathic
N	primary or secondary neoplasia
D	storage disease

Underlying pathology: 6 months – 6 years

V	infarct, hemorrhage, vascular malformation
I	immune mediated, viral, bacterial, protozoal, fungal, rickettsial
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I	idiopathic
N	**primary or secondary neoplasia**
D	storage disease

Diagnostics: based on age

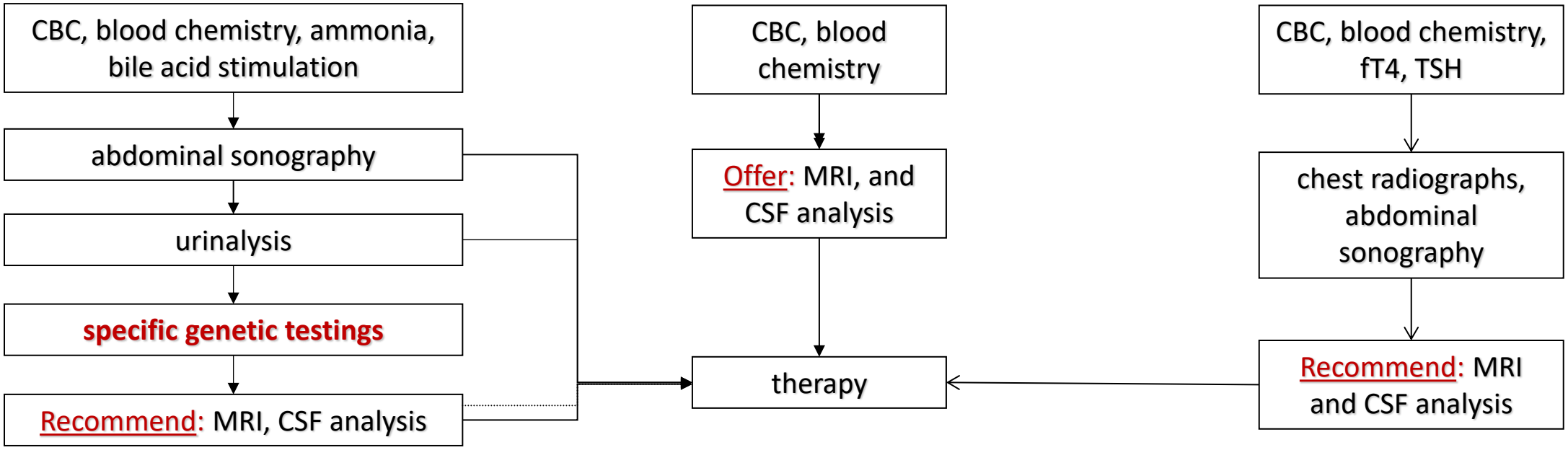
age < 6 months

age 6 months - 6 years and normal neuroexam

or seizures for years and normal neuroexam

age > 6 years

or age < 6 years and abnormal neuroexam



West Highland White Terrier, 1.5 years, female

- presented in lateral recumbency, barely responsive, intermittend excitations (seizures?)
- did have several similar episodes during the last nights: restless pacing, is biting in pillows
- ingestion of foreign material can not be excluded
- intermittent vomiting since being a puppy
- smallest puppy of all siblings
- increased frequency of urination; amount of urine not known
- referring vet expressed suspicion of gastroenteritis

Diagnostics: genetics (< 1 year)

West Highland White Terrier

Westfalen Terrier

2. Select the category and then the desired test(s)

This might interest you | Packages / Test combinations | Hereditary diseases / DLA | Coat colour / coat length | DNA profile | Breed determination | Storage

± Hereditary diseases / DLA

8294 Chondrodysplasia (CDPA) and -dystrophia (CDDY) (IVDD risk)	€62.00	8348 Craniomandibular osteopathy (CMO)	€62.00
8158 Degenerative myelopathy exon 2 (DM exon 2)	€72.00	8997 DLA typing	€98.00
8007 Globoid cell leukodystrophy (Krabbe-disease)	€62.00	8154 Hyperuricosuria (HUU/SLC)	€62.00
8062 Malignant hyperthermia (MH)	€62.00	8127 Progressive retinal atrophy (prcd-PRA)	€62.00
8015 Pyruvate kinase deficiency (PK)	€62.00	8119 von-Willebrand disease type I (vWD1)	€62.00

Symptoms of globoidcell leucodystrophy are ataxia, paresis of the hind legs and neurologic degeneration. After onset (1-3months) the disease progresses. Affected animals are usually euthanased till to the age of 10 months.

All prices include statutory VAT.
* Service is done by partner lab
* * Service is done by partner lab in bratislava

West Highland White Terrier

"Auryn Moonlight"

3 months, male

case number: 44505

Labogen

Diagnostics: blood chemistry

ammonia ↑

Glucose ↓

BUN ↓

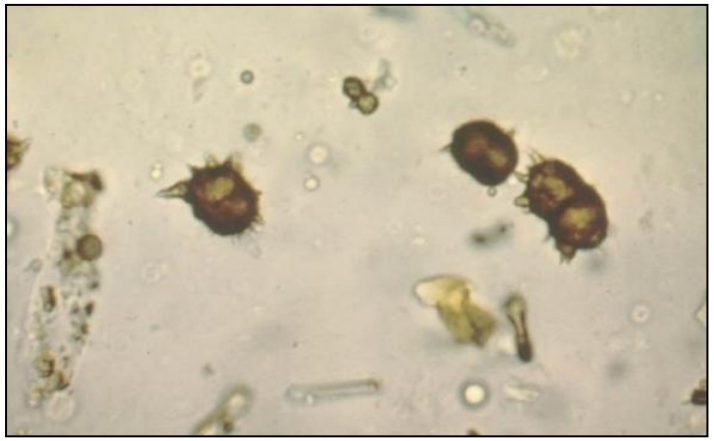
albumine ↓

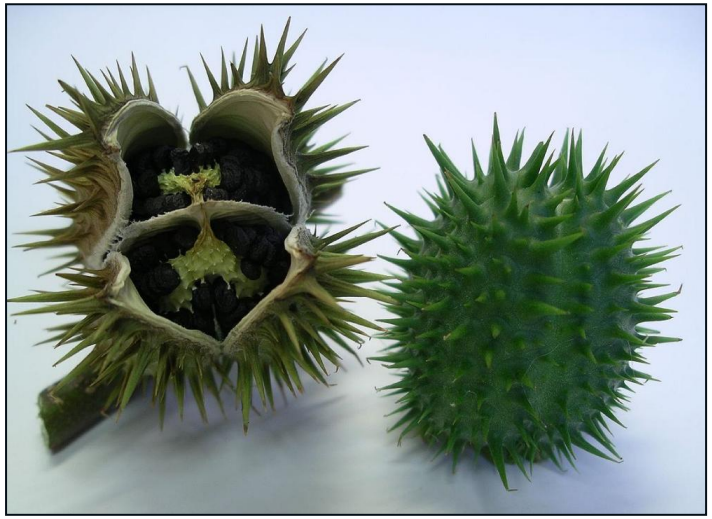
Untersuchung	Ergebnis	Einheit	Norm. min	Norm. max
NH3	**349**	umol/l	16	56
TP	66	g/l	57	78
ALP	**54**	U/l	9	53
GLU	**3,8**	mmol/l	3,9	8,2
GPT	**87**	U/l	22	84
CRE	81	umol/l	71	159
BUN	**5,31**	mmol/l	6,28	11,71
ALB	**12**	g/l	23	35
Na	153	mmol/l	147	156
K	4,6	mmol/l	3,4	4,6
Cl	114	mmol/l	107	120

Serum bile acid stimualtion test

Gallensäuren I	**165,5**	µmol/l	0	20
Gallensäuren II	**190,8**	µmol/l	0	40

Portosystemic shunt: urinalysis





thorn apple

ammonium urate crystals:

ammonia salts: ammonia ions are not converted into urea in the liver

urates: uric acid is not converted into allantoin in the liver

Portosystemic shunt: treatment

medical:

- lactulose 2.5-5 ml po TID
- lactulose enema; followed by neomycine per rectum
- antibiotics: amoxicillin, metronidazol p.o.
- feeding: easily digestable carbohydrates and proteins in frequent small portions
- anticonvulsivs: levetiracetam, propofol

feeding:

- home made: cottage cheese, yogurt (vanilla), cheese, spaghetti, rice, potataos, cooked vegetables, oat flakes

  but: it is usually not balanced!
- commercial: hepatic diet

Case 2

Ca de Bou, 6 years, female

- progressive weakness of 2 months duration
- some improvement on prednisolone
- started to have seizures 3 days ago
- had 5 generalized tonic-clonic seizures seizures since then
- is not the same anymore since seizures started

Diagnostics: blood chemistry

Untersuchung	Ergebnis	Einheit	Verlauf	Norm. min	Norm. max
NH3	36	umol/l		11	54
TP	65	g/l		50	72
ALB	**41**	g/l		26	40
ALP	67	U/l		13	83
GLU	**1,9**	mmol/l		4,1	7,1
TBIL	**1**	umol/l		2	9
IP	1,57	mmol/l		0,6	1,6
TCHO	6,06	mmol/l		2,9	8,1
GGT	**3**	U/l		5	14
GPT	44	U/l		17	78
Ca	2,77	mmol/l		2,3	3
CRE	71	umol/l		35	124
BUN	5,34	mmol/l		3,3	10,4
Na	148	mmol/l		141	152
K	**3,4**	mmol/l		3,8	5
Cl	115	mmol/l		102	117

Differentials: hypoglycemia

↑ sekretion of insulin/insulin-like growth factor	↓ Glucose production	↑ Glucose utilisation	Others
• **insulinoma**	• Addison	• sepsis	• insulin application
• extrapancreatic neoplasia	• hypopituitarism	• extreme exercise	• other drugs
• island cell hyperplasia	• hepatopathy	• pregnancy	• starvation
• xylit intoxication	• growth hormon deficits		
	• glycogen storage disease		
	• neonates		
	• toy breeds		

Diagnostics: insulinoma

17 mU/l

Insulin (CLIA)

Die Insulin-Konzentration im Serum sollte immer im Zusammenhang mit dem aus derselben Probe gemessenen Glukosespiegel interpretiert werden. Gesunde nüchterne Hunde weisen normalerweise Insulinspiegel zwischen 5 und 20 mU/l auf. Insulinkonzentrationen >20 mU/l in Verbindung mit einer wiederholten Glukosekonzentration <60 mg/dl und entsprechenden klinischen Symptomen unterstützen die Diagnose eines Insulinoms.
Die angegebenen Bereiche beziehen sich auf nüchterne Hunde mit einem Glukosespiegel <60 mg/dl.

Serum Insulin-Konzentration	
>20 mU/l	Insulinoma likely
10-20 mU/l	Insulinoma possible
5-10 mU/l	Niedriger/normaler Insulinspiegel. Insulinom unwahrscheinlich jedoch nicht ausgeschlossen.
<5 mU/l	Insulinspiegel unter der Nachweisgrenze. Insulinom sehr

Fructosamine level: ↓ Insulinom

Treatment: insulinoma

- **Acute hypoglycemia:**

  glucose: 0.5 g/kg i.v. bolus, followed by 2.5-5% glucose CRI

- **Long term therapy:**

food:	frequent feeding of small portions commercial diabetic food honey: for emergencies	
medication:	prednisolone	0.5-2 mg/kg SID
	diazoxide	5-10 mg/kg BID to TID
	streptozocin, somatostatin (octreotid), glucagon	
surgical:	partial pancreatectomy	

Case 3

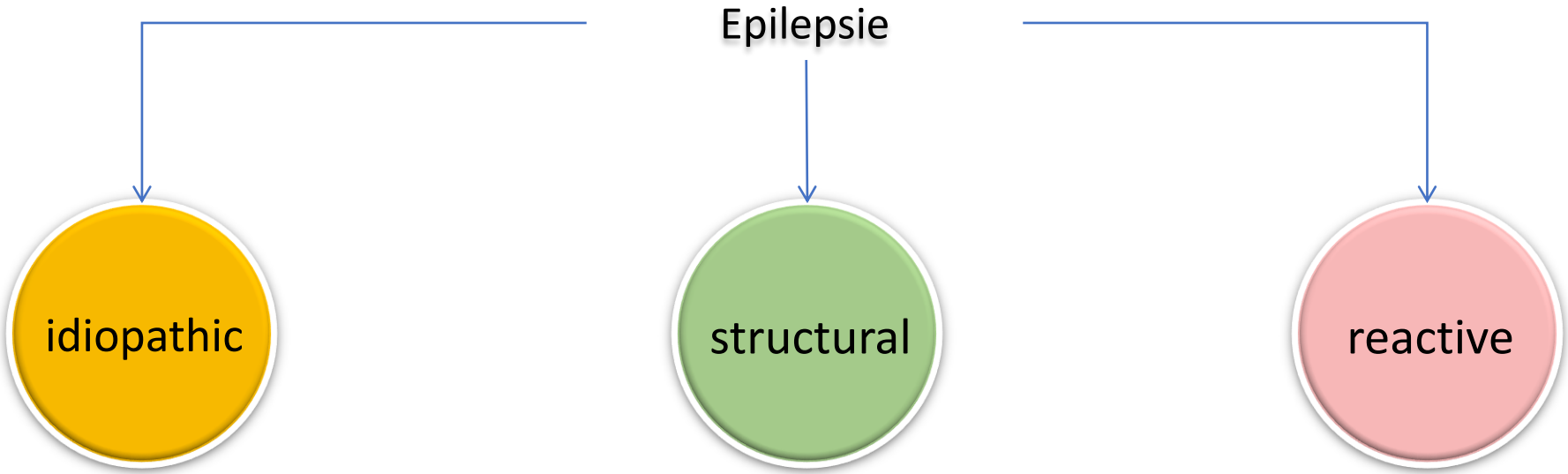
Mixed breed dog, 6 years, male

- has generalized tonic-clonic seizures for 2 years
- used to happen about once every 3 months
- 3 seizures in the last 2 days
- seems to be normal between seizures

Typical sequence

- Pre-ictal — before seizure
  - behavioral changes
  - duration: minutes – hours (days)
- Ictus — during seizure
  - duration: a few minutes (1-3)
- Post-ictal — after seizure
  - sleepy, restless, blind, ataxic, revenous
  - duration: minutes – hours (days)

Seizure treatment



- > 1 seizure in 6 months
- status or cluster
- prolonged post ictal signs

- anticonvulsives long-term

- treat underlying pathology

Plus

- anticonvulsives long-term

- treat underlying pathology

- anticonvulsives short-term

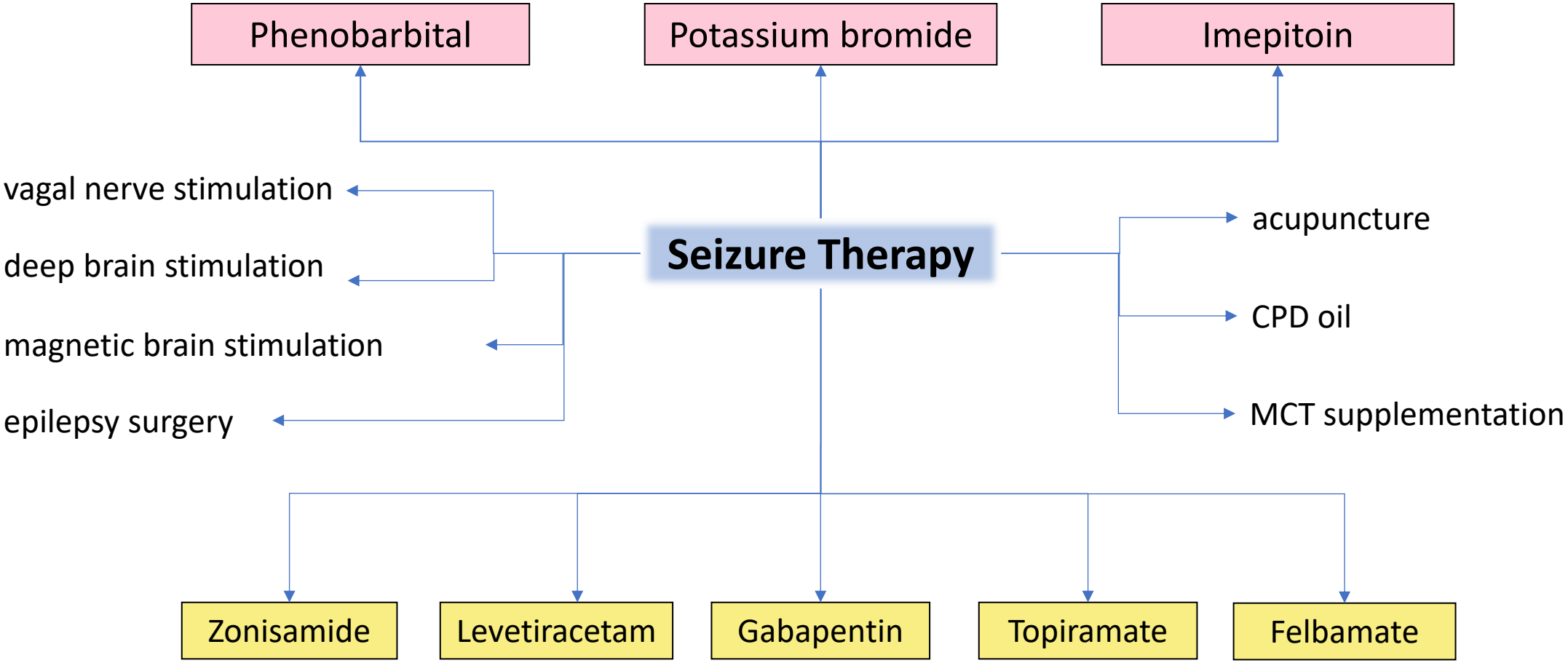
Client instructions

- therepautic goal: to reduce seizure frequency and/or severity
- complete remission in ….. % of dogs
- side effects of medication
- importance of regular medication
- need for regular recheck appoints
- influences on owner‘s personal life
- reduced live expectancy of patients
- keep seizure records

Client instructions

- therepautic goal: to reduce seizure frequency and/or severity
- complete remission in **20 - 30 %** of dogs
- side effects of medication
- importance of regular medication
- need for regular recheck appoints
- influences on owner‘s personal life
- reduced live expectancy of patients
- keep seizure records

Treatment options



Phenobarbital

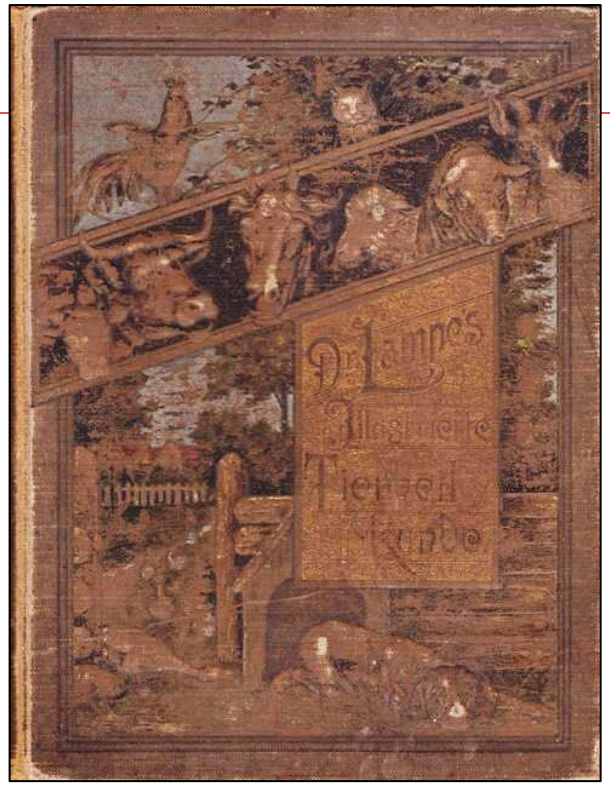
- Characteristics: effective, cheap
- Dose: 2.5 mg/kg twice daily
  (may be given 3times daily)
- Side effects: PU/PD
  potentially hepatotoxic
  rare: anemia, hyperexcitability, fear, hepatocutanous syndrome, dyskinesia, pancreatitis (0.3%)
- Blood level: 20-30 µg/ml
  when: 2 weeks, 3 months, every 6 months
  every 6 months: ALAT, albumine, glucose
  (bile acid stimulation test)
- Specifics to know: hepatic enzyme induction: AP
  interference with thyroid testings

Imepitoin

- Characteristics:
  - nearly as effective as phenobarbital
  - less side effects than phenobarbital
  - not hepatotoxic
  - anxiolytic
- Dose:
  - 20 (30) mg/kg twice daily
  - when added to PB: start with 10 mg/kg twice daily
- Side effects:
  - ataxia, vomiting, polyphagia, sleepiness, hyperactivity, listlessness, PU/PD, diarrhea, anorexia, salivation, 3rd eye lid prolaps, impaired vision and hearing
- Blood level:
  - not necessary
- Specifics to know:
  - only limited clinical data
  - not approved for clusters of seizures

Potassium bromide

- Characteristics: effective, cheap, not hepatotoxic
  very long half life time (1 month)
- Dose: 20 mg/kg twice daily
- Side effects: sedation, PU/PD, polyphagia
  rare: pancreatitis (10%), rear limb weakness
  coughing, diarrhea, vomiting
- Blood level: 2000-3000 µg/ml
  when: 3 months, every 6 months
- Specifics to know: keep chlorid intake constant
  don‘t feed together with milk products
  don‘t get disturbed by high chlorid blood levels
  loading dose: 70 mg/kg BID for 5 days



1904

Dr. Lampes Illustrierte Thierheilkunde

Cat: not recommended, severe side effects, up to 40% eosinophilic pneumonitis

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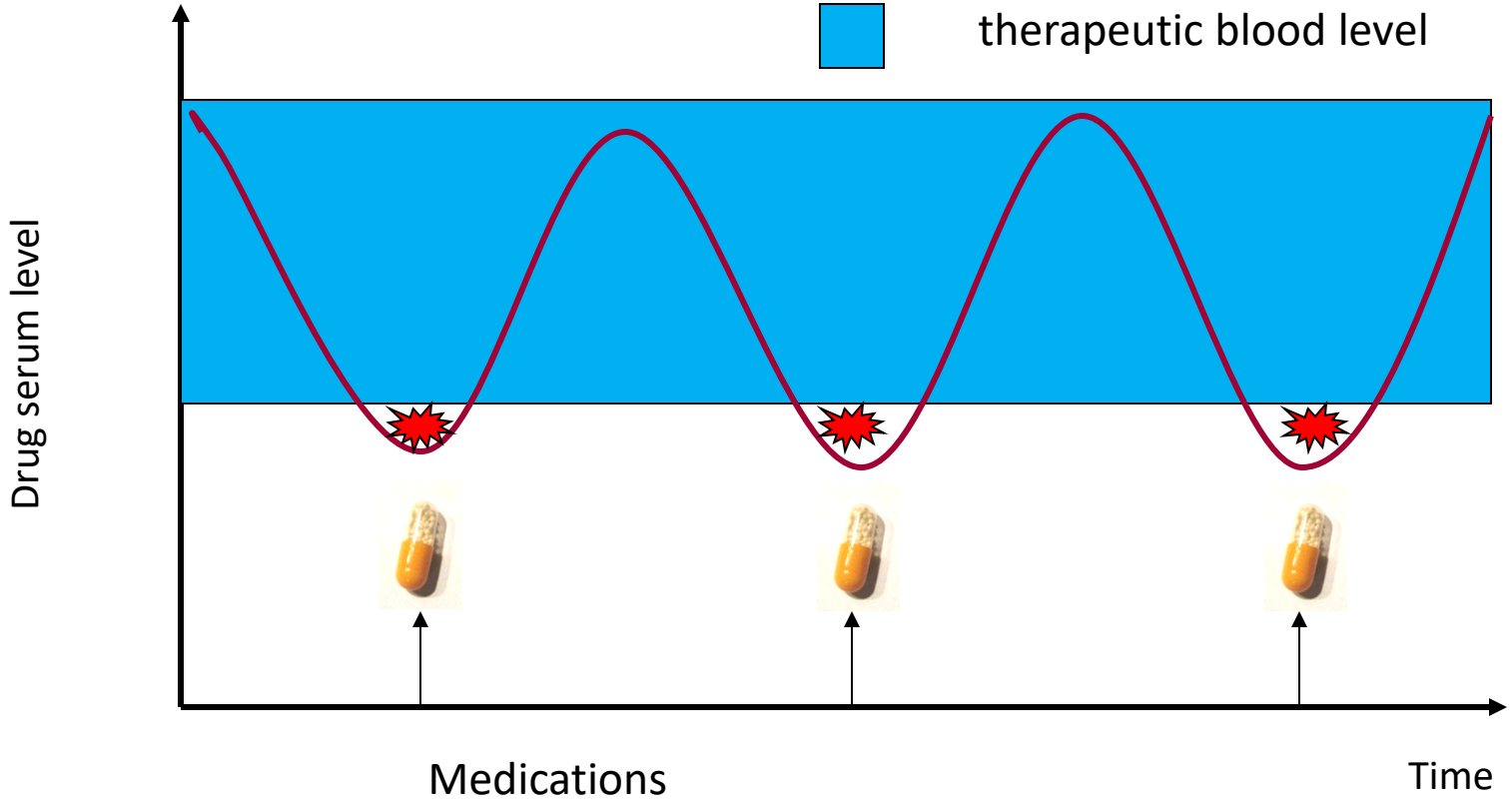
Cat: not recommended, severe side effects, up to 40% eosinophilic pneumonitis

Serum level checks (phenobarbital, potassium bromide)



Measurement of serum blood level: immediately before medication

Levetiracetam

- Characteristics: add-on to phenobarbital or potassium bromide
  may be effective as single medication in structural epilepsy (Kelly 2017)
- Dose: 10-20 mg/kg twice to three times daily
- Side effects: rare: sedation, ataxia
- Blood level: not established
- Specifics to know: well tolerated
  no hepatotoxicity
  fast onset of action (2-3 days)

A suggested treatment plan

start with phenobarbital

after 2 weeks:
check serum level

I. serum level < 20 mg/dl
increase PB
re-check in 3 and 6 months

II. serum level > 20 mg/dl
no seizures
continue as before
re-check level in 3 and 6 months

III. serum level > 25 mg/dl
still seizures
add potassium bromide or imepitoin

Serum level re-check in 3 and 6 months:

if seizure frequency is not reduced by at least 50%

increase dose if upper end of allowed serum level has not been reached

add another drug if upper end of allowed serum level has been reached

For phenobarbital: increasing dose by 50% will result in about 50% increase of serum level

Temporary medication in clusters

- rectal diazepam: 1 mg/kg once the first seizure has happened

  2 mg/kg in dogs on phenobarbital

- nasal midazolam: gel is superior to i.v.-solution 0.2 mg/kg

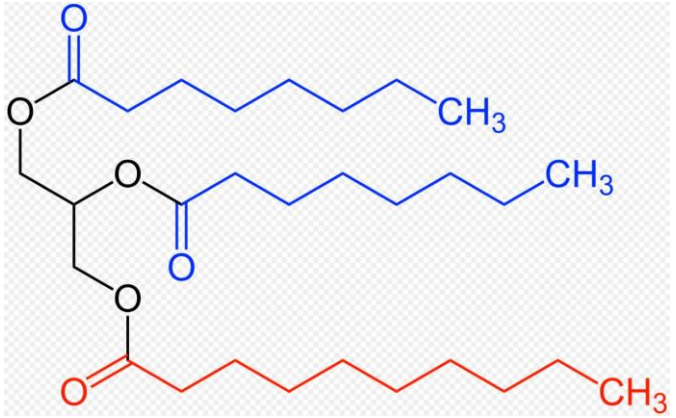
- levetiracetam: pulse therapy:

  40 – 60 mg/kg once followed by
  20 mg/kg TID until seizure free for 2 days

MCTs: medium chain fatty acid supplementation

- short – chain – FA (< 6 carbon atoms)
- medium – chain- FA (6-12 carbon atoms)

  capron, capryl, caprin, laurin acid
  in: cocos oil, palm oil
- Long – chain – FA (>12 carbon atoms)



triglycerides

Adding MCTs zu diet:
50% reduction of seizures frequency in none-responders

Law TH, Davies ESS, Pan Y et al. A randomised trial of a medium-chain TAG diet as treatment for dogs with idiopathic epilepsy. Br J Nutr 2015;114:1438-1447.

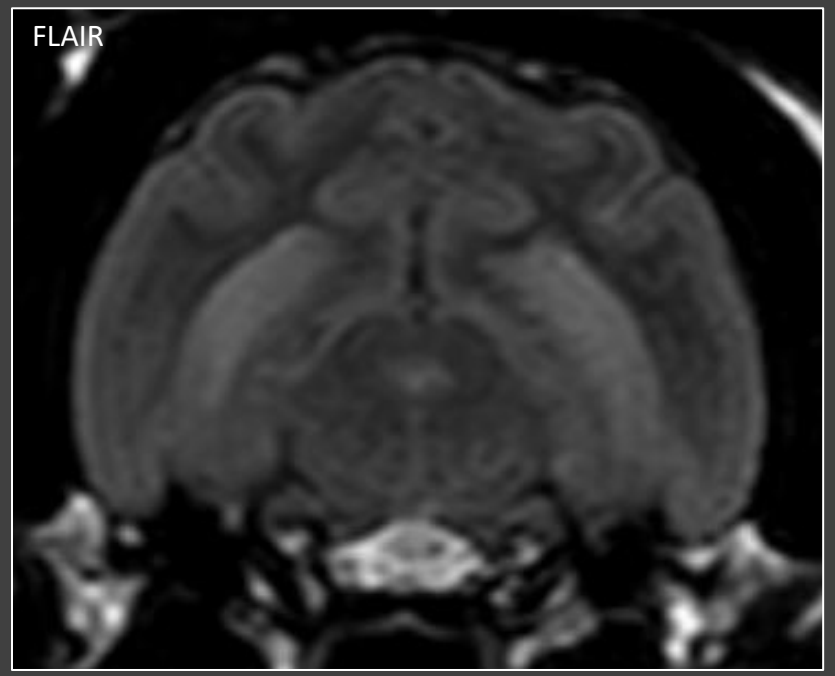
Replace 9 % of daily caloric requirement with MCT oil

Case 4

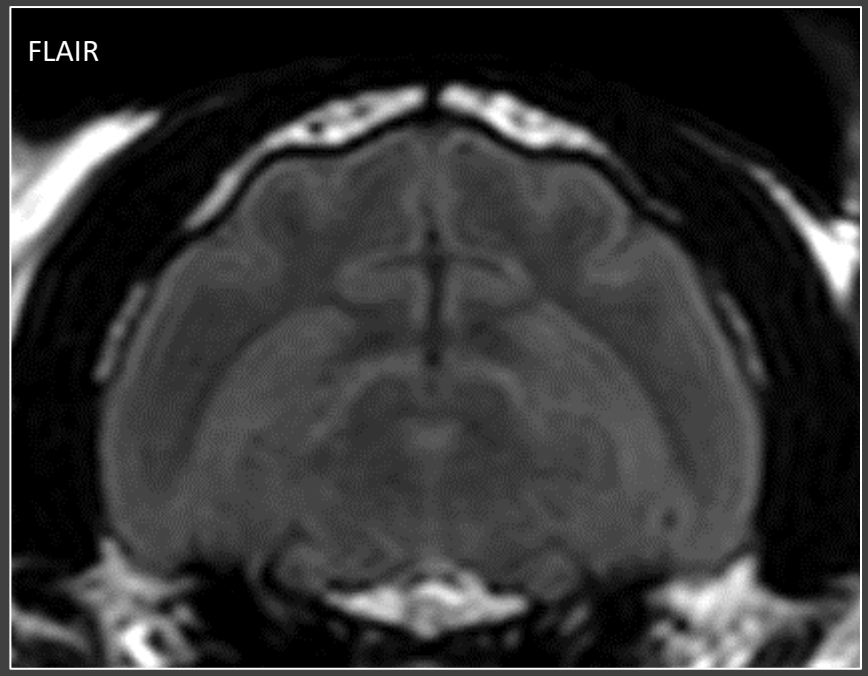
DSH cat, 2 years, female

- seizures starting 3 months ago
- cluster of epileptic seizures for 2 days
- referring veterinarian started phenobarbital as well homeopathic drugs
- at presentation seizures every hour

Specifics in cats



limbic encephalitis



normal

antibodies against voltage-gated potassium channels:

- Anti CASPR2 IgG
- Anti LGl1 IFT

Specifics in cats

Untersuchung	Ergebnis	Einheit
LAB FL Autoimmundiagnostik Katze		
Versand am:	10/2	
Material:		Serum
LGI1 IgG IFT	pos	< 1:10
CASPR2 IgG IFT	neg	< 1:10

Limbic encephalitis

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Complex partial seizures with orofacial involvement in 35 cats: MRI changes, cerebrospinal fluid analysis, voltage-gated potassium channel antibodies and survival

Thomas Flegel, Kaspar Matiasek, [...], and Johanna E König +10 View all authors and affiliations

Table 1 Numbers of cats with hippocampal hyperintensity on MRI and detection of voltage-gated potassium channel (VGKC) in 31 cats with different seizure semiologies

Type of seizures	Total number	Hippocampal T2 hyperintensity	Bilateral T2 hippocampal hyperintensity	Hippocampal contrast enhancement	Antibodies against VGKC	
					CASPR2	LGI1
CPSOFI only	20	11	11	5	1	6
CPSOFI plus generalised tonic–clonic seizures	11	7	6	6	0	2

CASPR2 = contactin-associated protein 2; CPSOFI = complex partial seizures with orofacial involvement; LGI1 = leucine-rich glioma inactivated 1

Limbic encephalitis: treatment

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Aniconvulsives:

- Phenobarbital
- alternatively Levetiracetam

Antiinflammatory medication:

- prednisolone 1mg/ kg SID for 6 months
- check Ab 6 after 6 months
- if ab negativ: slowly discontinue prednisolone
- check Ab one months after discontinuing prednisolone

Limbic encephalitis: prognosis

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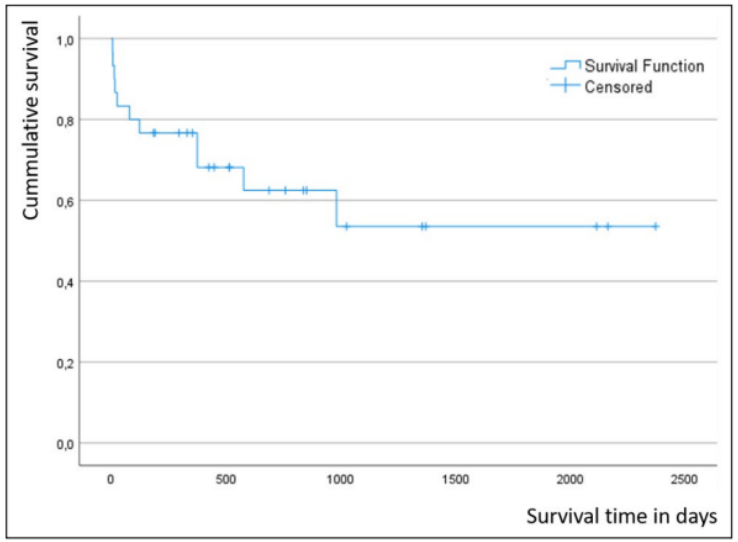


Figure 1 Kaplan–Meier survival curve of cats with complex partial seizures with orofacial involvement for which follow-up information was available (30/35). In total, 19/35 cats were censored as they were still alive at the time of writing the manuscript

Of the 33 cats that were discharged, three died or were euthanased within the first month for different reasons.

The **mean survival time** of the remaining 30 cats was **771±646 days (range 79–2372), with 19/30 (63.6%) cats alive** at the time of writing the manuscript

Case 5

French Bulldog, 5 years, male

- seizures for 2 months
- appears to be restless for 2 weeks
- head tilt since yesterday
- intermittent stumbling

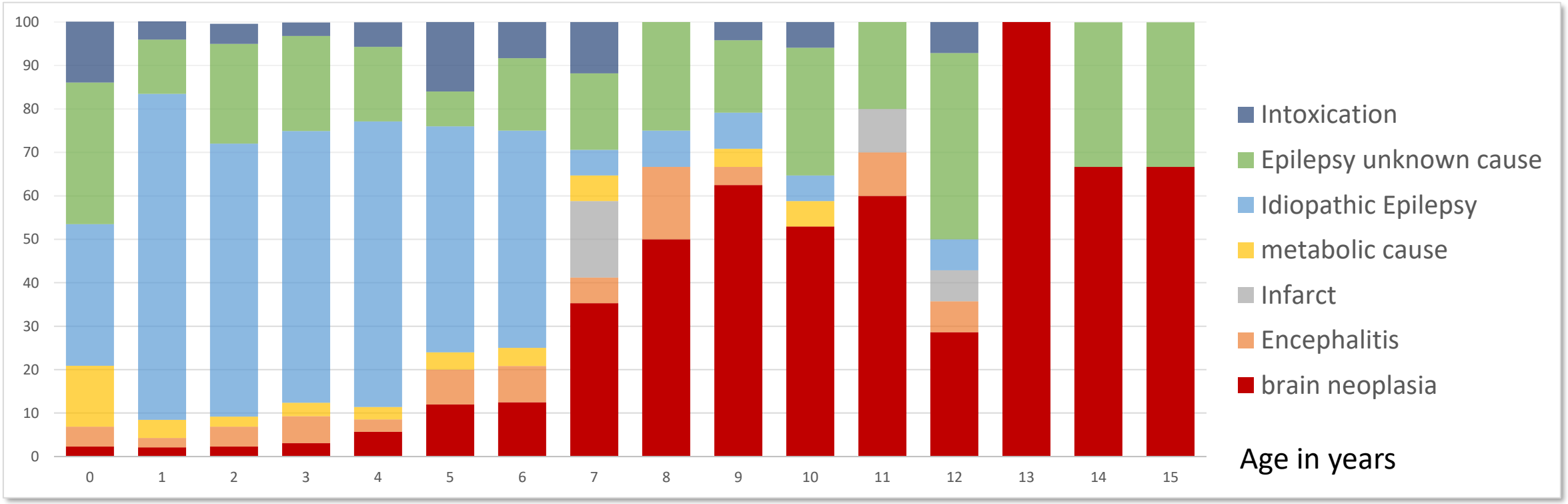
Underlying pathology: > 6 years

V	**Infarct, hemorrhage,** vascular malformation, **hypertension**
I	**immune mediated**, viral, bacterial, protozoal, fungal, rickettsial
T	traumatic brain injury (posttraumatic), toxic
A	hydrocephalus, arachnoid cysts, lissencephaly
M	**hypoglycemia (insulinoma), hepatoencephalopathy, severe uremia, hypocalcemia, polycytemia, hyperviscosity ...**
I	**idiopathic**
N	**primary or secondary neoplasia**
D	storage disease

Causes of seizures based on age

Causes of epileptic seizures in dogs depending on their age

Small Animal Department, Leipzig University, 2017 – 2021 (n=381)

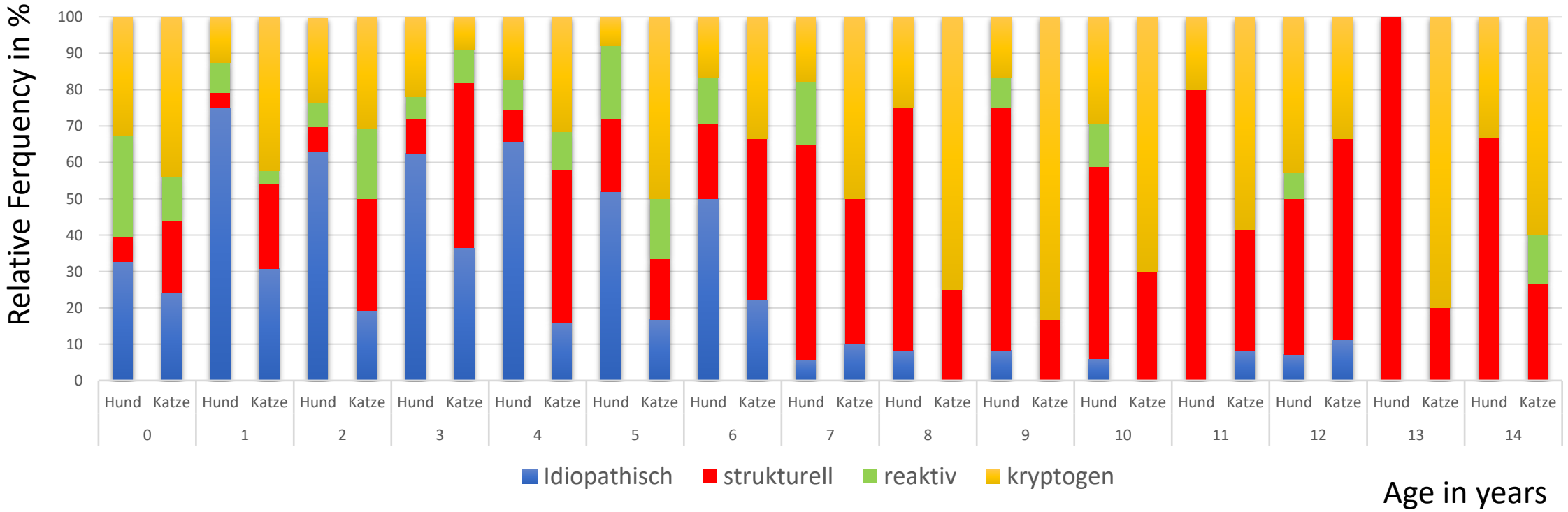


student project: Anna-Lena Holst, Olivia Kretzschmann, 2021

Causes of seizures based on age: dogs versus cats

Causes of epileptic seizures in dogs and cats based on age

Dept. Small Animals, Leipzig University, (dogs n=381; cats n=100)



Student project: Anna-Lena Holst, Olivia Kretzschmann, 2021 (Hund)
Sonja März, Kim Josephine Theysen, 2024 (Katze)

BSAVA
ORIGINAL ARTICLE
Check for updates

Prevalence of structural and idiopathic epilepsy in brachycephalic and non-brachycephalic dogs in the context of the International Veterinary Epilepsy Task Force guidelines

Journal of Small Animal Practice, 2025

n=111

Structural brain disease as cause of epileptic seizures:

brachycephalic breeds:	61,8 %
none brachycephalic breeds:	22,1%

Dogs aged 6 months – 6 years with a normal interictal neurological examination having structural brain disease:

brachycephalic breeds:	33,3 %
none brachycephalic breeds:	0%

Table 3. Comparison of the most common diagnoses for brachycephalic and non-brachycephalic patients aged 6 months to 6 years

	Brachycephalic	Non-brachycephalic
Most common diagnoses for patients aged 6 months to 6 years, *n* (%)	1. IE, 11 (50) 2. Inflammatory, 5 (22.7) 3. Intra-axial neoplasia, 4 (18.2)	1. IE, 42 (97.7) 2. Intra-axial neoplasia, 1 (2.3)

IE Idiopathic epilepsy

Treatment

Chemo-therapy

a few months
(may be 6 months)

Surgery

Meningioma
cat: > 3 years
dog: 2-3 years

Glioma dog: 0.5 – 1 year

Radiation

Meningioma
dog: 2 years

Journal of Veterinary Internal Medicine

Journal of Veterinary Internal Medicine
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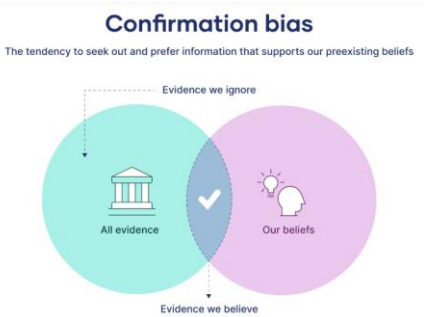
American College of Veterinary Internal Medicine



STANDARD ARTICLE OPEN ACCESS

Small Animal Internal Medicine Neurology

Comparison of Survival After Treatment of Presumed Intracranial Meningioma by Radiotherapy or Surgery in 285 Dogs

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Median survival time:
n=285

surgery:

297 days
(IQR: 99–768)

radiation:

696 days
(IQR: 368–999)

Prognosis: Meningioma following surgery

RESEARCH Open Access

Clinical presentation, diagnostic findings and outcome of dogs undergoing surgical resection for intracranial meningioma: 101 dogs

Alexander K. Forward[1*], Holger Andreas Volk[2], Giunio Bruto Cherubini[3], Tom Harcourt-Brown[4], Ioannis N. Plessas[1], Laurent Garosi[5] and Steven De Decker[6]

Table 1 Meningioma histological classification of all 101 dogs together with the median survival times

Meningioma subtype *(total number of cases: 101)*	Median survival time (days) following hospital discharge *(number of dogs)*	95% Confidence Interval	
		Lower Bound	*Upper Bound*
*Papillary (9) **	1079 (7)	134.3	2023.7
Rhabdoid (1)	572 (1)	.	.
*Angiomatous (angioblastic) (5) **	454 (5)	95.4	812.6
*Fibrous (fibroblastic) (4) **	417 (3)	.000	925.906
*Meningotheliomatous (28) **	386 (25)	184.5	587.5
Vacuolar (1)	385 (1)	.	.
*Psammomatous (12) **	363 (10)	118.2	607.8
*Transitional (mixed) (23) **	307 (20)	0.0	850.5
Unknown (10)	252 (9)	82.5	421.5
*Anaplastic (malignant) (2) **	123 (2)	.	.
Cystic (4)	99 (4)	.000	443.9
Choroid (1)	–	–	–
Atypical (1)	–	–	–

Legend: The median survival time of those dogs that survived to discharge is detailed depending on the histopathological subtype. Those with a * are those listed in the histological classification of tumours of the nervous system of domestic animals (WHO): Tumours of the meningothelial cells [8]. The others are subtypes that were later identified independently in dogs following the WHO classification system

2022

n=101

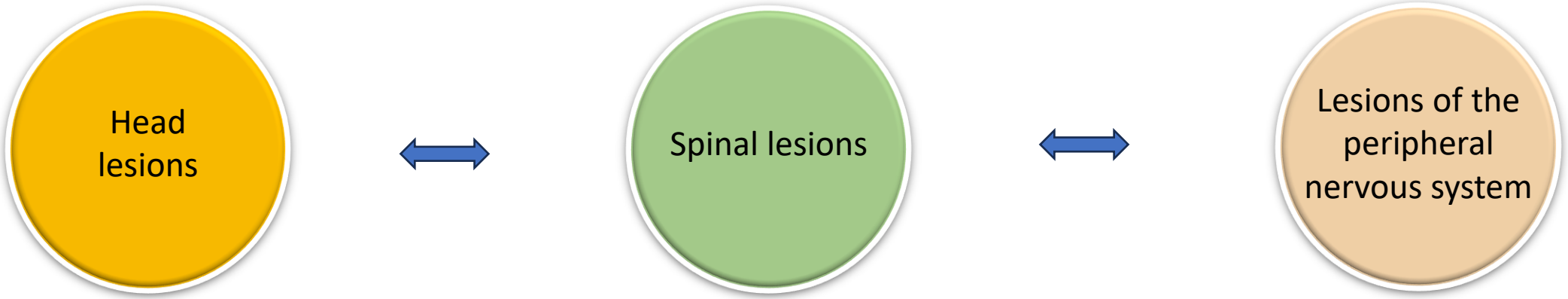
Patients with ataxia

if coordination got lost

Prof. Dr. Thomas Flegel

Dipl. ACVIM (Neurology), Dipl. ECVN
Klinik für Kleintiere, Universität Leipzig

Potential localisations



- paresis
- reduced mentation
- reduced paw placing reactions
- cranial nerve deficits
- **vestibular syndrome**
- seizures

- paresis/plegia
- reduced paw placing reactions
- reduced segmental spinal reflexes in front or rear limbs

- lack of symptomes pointing towards the head

- tetraparesis/plegia
- reduced paw placing reactions
- generalised reduced segmental spinal reflexes

Case 1

Maine Coon, 7 years, male: Cesar

- was relatively quite over the last few days
- sudden onset of clinical signs yesterday
- has a funny gait and head tilt
- continously vocalizing
- was diagnosed with diabetes mellitus one year ago, but doesn‘t get insulin, yet
- feeding: RC diabetic
- regularilly vaccinated
- indoor cat

Peripheral or Central

	Peripheral (inner ear)	Central (brain)
Mentation	normal	can be abnormal
Behavior	normal	can be abnormal
Paw positioning	normal	can be abnormal
Nystagmus	horizontal, rotatory	horizontal, rotatory, **vertical**
Cranial nerve deficits	only VII	multiple CN deficits possible
Horner‘s syndrome	can be	no

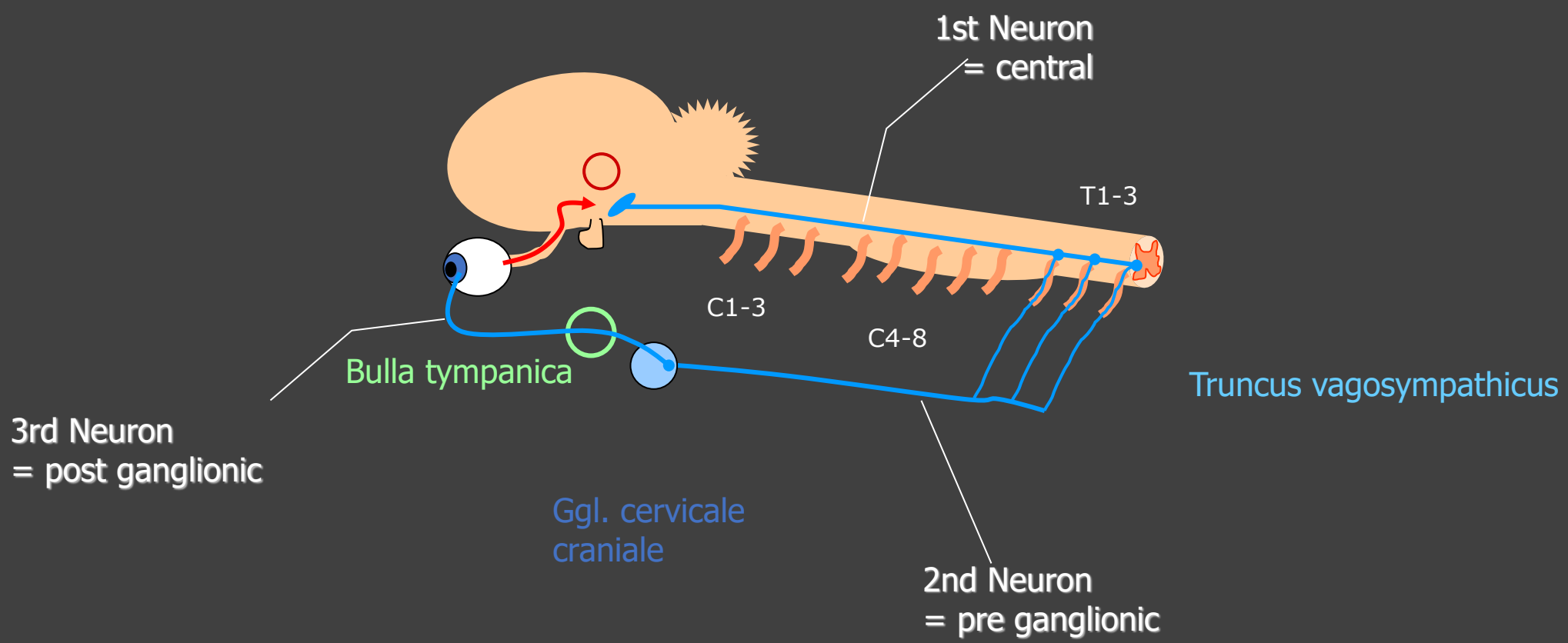
Horner's syndrome

Loss of sympathetic innervation to the eye:

- **ptosis:** **„smaller eye“**
- **miosis:** **smaller pupil**
- **enophthalmus:** **sunken in eye ball**
- **3rd eye lid protrusion**

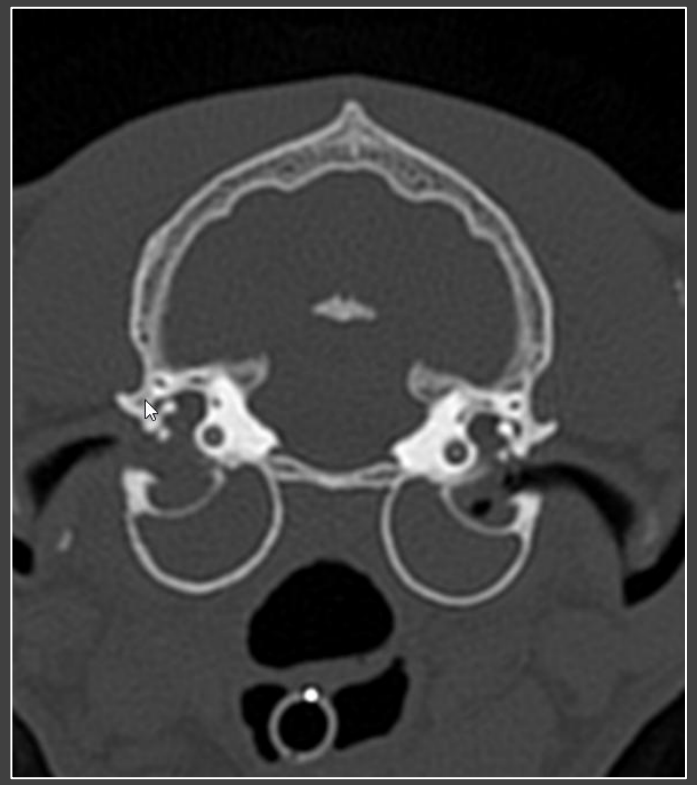
Vestibular deficits + Horner's syndrome → **peripheral vestibular lesion (inner ear)**

But: not every Horner's syndrome is caused by inner ear disease

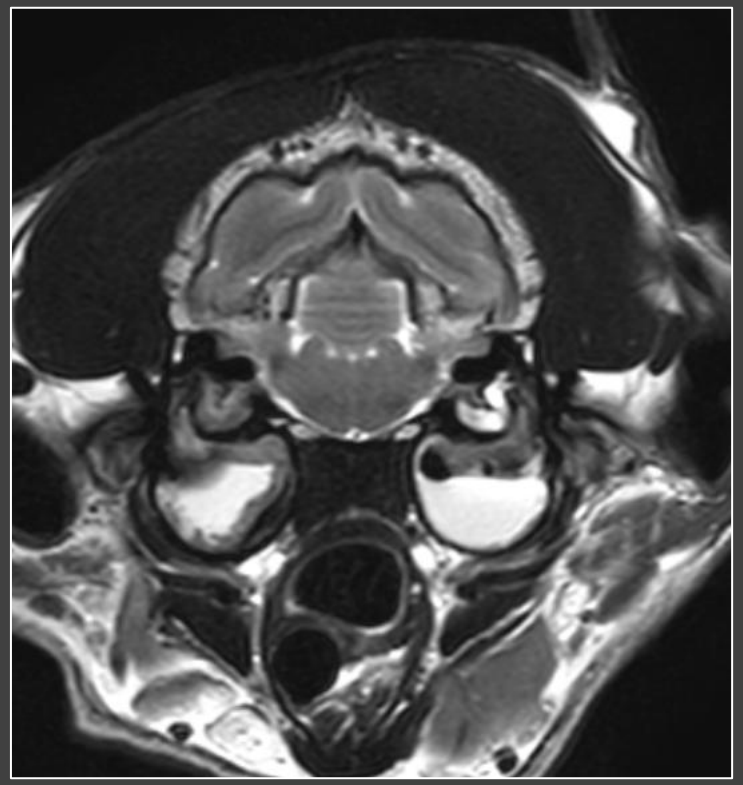


Adapted from K. Matiasek, LMU Munich

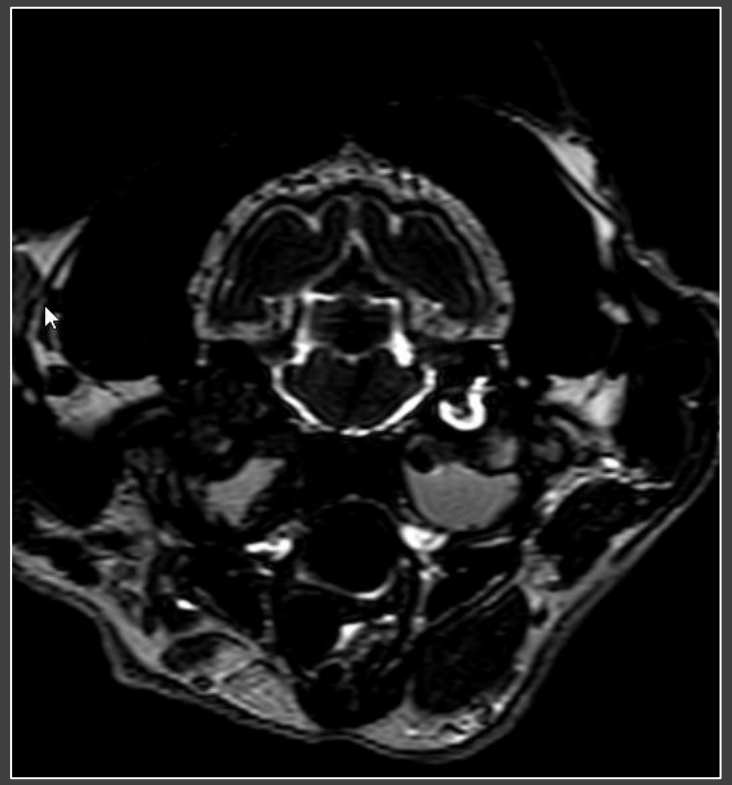
Maine Coon, 7 years, male



CT



MRI: T2



MRI: T2 cochlea sequence

Diagnosis: **bilateral otitis media, right-sided otitis interna**

Otitis media/interna CATS: systemic treatment

often ascending infection following rhinitis via tuba auditiva
+/- additional nasopharyngeal/aural polyps

Systemic conservative treatment:

- tetracyclin (i.e. doxycyclin 5 mg/kg BID) for 3 weeks OR macrolids (i.e. azithromycine 5 mg/kg SID) for 5 days
- in suspected otitis interna: lincosamide (i.e. clindamycin) or fluorchinolones (i.e. enrofloxacin) for 6 weeks
- NSAID (i.e. meloxicam) for 10 days
- inhalation using NaCl for 14 days as expectorant
- ACC/bromhexin/other expectorants for 10 days
- may be betahistin 5 mg/kg TID for 5 days
- in case of „dirty ears“: additional local treatment

in case of polyps or failure to respond to conservative treatment: surgical treatment is necesarry!!

Otitis media/interna DOGS: systemic treatment

usually secondary to otitis externa perforating ear drum (except brachycephalics: impaired drainage through tuba auditiva)

- perform always cytology from ear canal (cocci rods, yeast, neutrophiles, epithelial cells) and bacterial culture (antibiogram from ear often unreliable!)

- **systemic conservative treatment:**
  - ß-Lactam-antibiotics (i.e. cephalosporines [cefalexin] or aminopenicillines [amox/clav]) for 2 weeks
  - in otitis interna: lincosamide (i.e. clindamycin) or fluorchinolones (i.e. enrofloxacin) for 6 weeks
  - NSAID (i.e. meloxicam, robenacoxib) for 10 days
  - may be betahistin 5 mg/kg TID for 5 days
  - expectorants and inhalation usually not heplful
  - allways: careful ear flushing using NaCl followed by local ear treatment
- **in failure to respond to conservative treatment: surgical treatment (bulla osteotomy) is necesarry!!**

Otitis media/interna DOGS and CATS: local treatment

CAVE: be aware that the ear drum may be perforated; avoid local application of ototoxic substances!

- ear cleaning using Triz EDTA buffer BID
- 30 minutes following cleaning: local application of the following „ear mixture“:

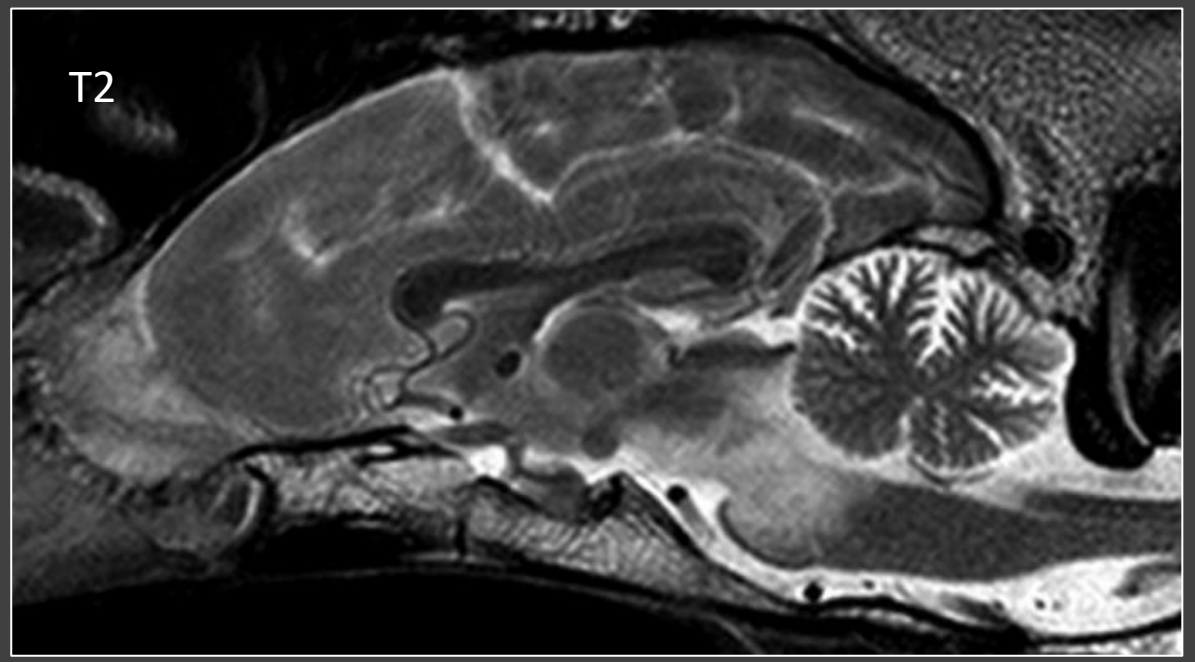
  6 ml Baytril 2,5% + 3 ml dexamethasone (4 mg/ml + 11 ml aqua ad inj.)
- treat for 2 to 3 weeks using ear cleaning + „ear mixture“
- after discontinuing „ear mixture“: gradually reduce ear cleaning; otherwise excessive production of cerumen

Case 2

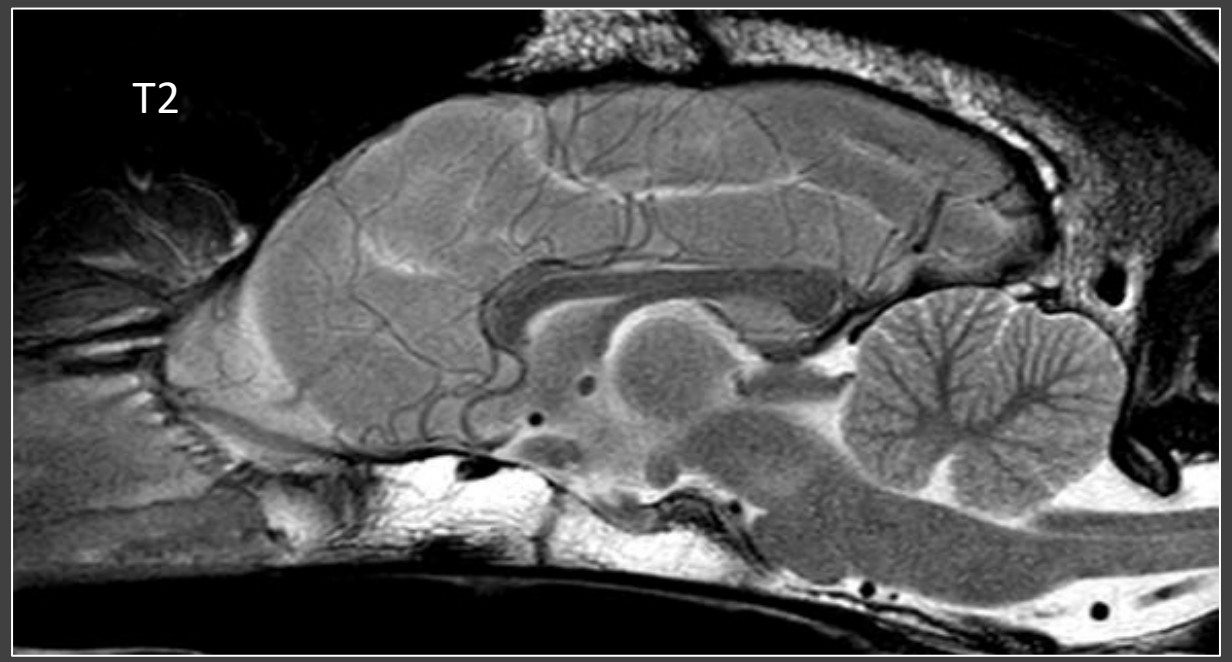
Mixed breed dog, 3 year, female: **Chiara**

- has been obtained 2 months ago
- has been rescued from Romanian streets
- neurologically abnormal since then
- neurological deficits were more obvious initially: lack of coordination

Mixed breed dog, 3 year, female



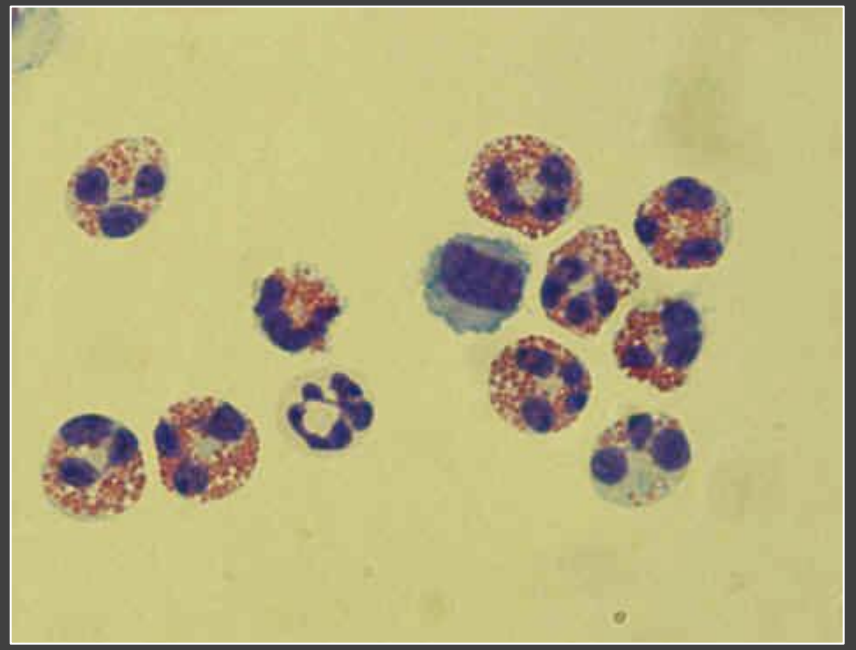
our patient



normal image for comparison

Mixed breed dog, 3 year, female

Untersuchung	Ergebnis	Einheit
Liquoranalyse inkl. Punktion		
Material im Frost		
Serum	ja	
Liquor	250 + 1500 µl	
Entnahme	cisternal	
Farbe	farblos	
Transparenz	trüb +	
Pandy	schwach positiv	
kernhaltige Zellen	8832/3 (2944)	
Erythrozyten	0	/µl
Eiweiß	0,49	g/l
SEDIMENT	3)	
aktivierte Lymphozyten	0	%
Lymphozyten	0	%
Monozyten	14	%
Makrophagen	1	%
eosinophile Granulozyten	78	%
neutrophile Granulozyten	7	%
Zytologische Diagnose: Eosinophile Pleozytose		



147155

Normal:

number of nucleated cells: < 6/µl

protein concentration: < 0.30 g/l

Eosinophilic meningoencephalomyelitis

J Vet Intern Med 2009;23:275–281

Cerebrospinal Fluid Eosinophilia in Dogs

R.C. Windsor, B.K. Sturges, K.M. Vernau, and W. Vernau

Background: Marked eosinophilic meningitis or meningoencephalomyelitis (EME) is rarely reported in dogs and the cause is usually undetermined. Long-term prognosis for dogs with cerebrospinal fluid (CSF) eosinophilia is variable.

Animals: Twenty-three client-owned dogs.

Methods: Retrospective case series. Dogs with eosinophilic CSF, defined as total nucleated cell count (TNCC) >3 cells/μL with >20% eosinophils, were identified by a computerized search of all dogs having cisternal and/or lumbar CSF analyzed as part of the diagnostic workup between 1992 and 2007.

Results: TNCC in CSF ranged from 4 to 4,740 cells/μL (median 84 cells/μL, reference range ≤3 cells/μL), with 22 to 95% (median 78%) eosinophils in the differential count. An infectious agent was identified on necropsy in 4 of 23 (17%) dogs (*Cryptococcus neoformans* [n = 2], *Neospora caninum* [n = 1], and *Baylisascaris procyonis* [n = 1]). Each of these dogs had progressive neurologic deterioration. Sixteen dogs had idiopathic EME. Magnetic resonance imaging (MRI) findings were abnormal in 7 of 13 dogs with EME; 2 dogs had focal lesions and 5 dogs had multifocal lesions. Clinical signs in 12 of 16 (75%) dogs with idiopathic EME resolved with prednisone treatment. Three dogs with acute intervertebral disc herniations recovered after decompressive surgery alone.

Conclusions: Idiopathic EME is a common cause of eosinophilic pleocytosis in dogs. MRI findings are variable. Infectious causes of EME were less common and had a poor prognosis.

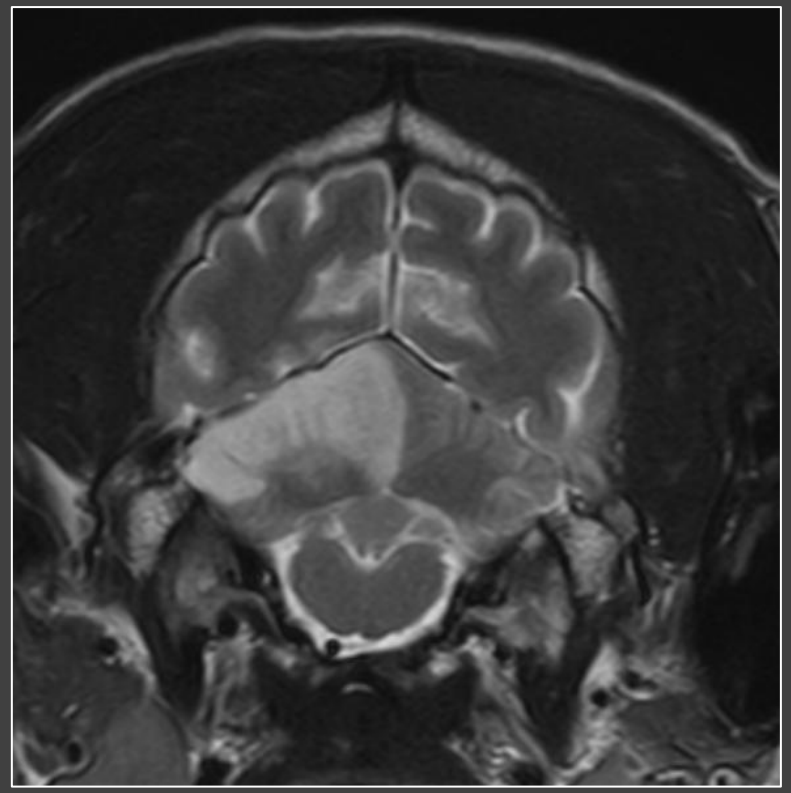
Key words: CSF analysis; Eosinophil; Meningitis; MRI.

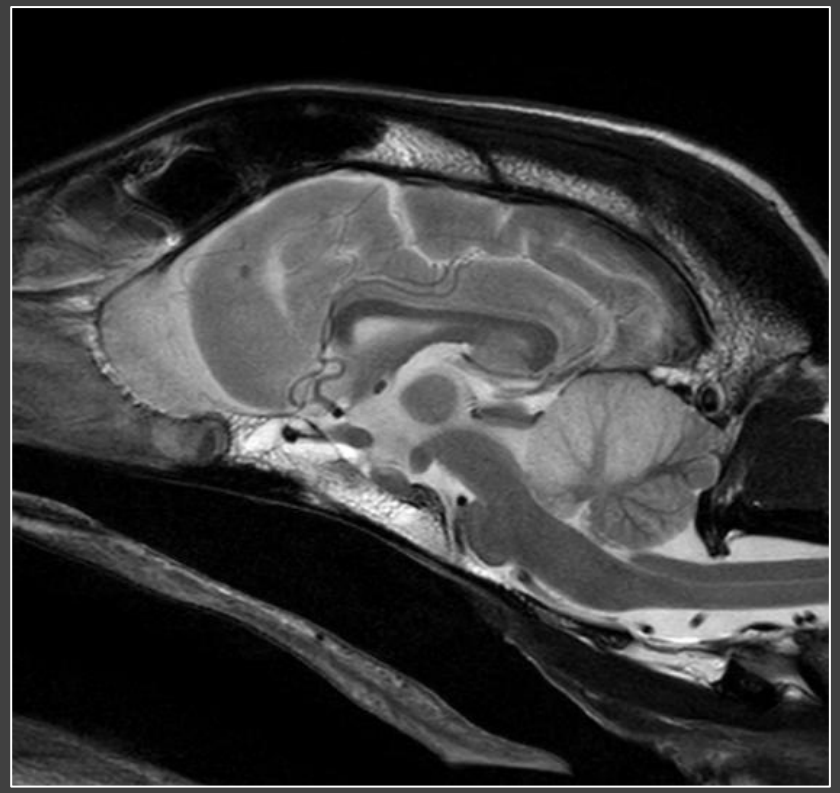
Mainly idiopathic

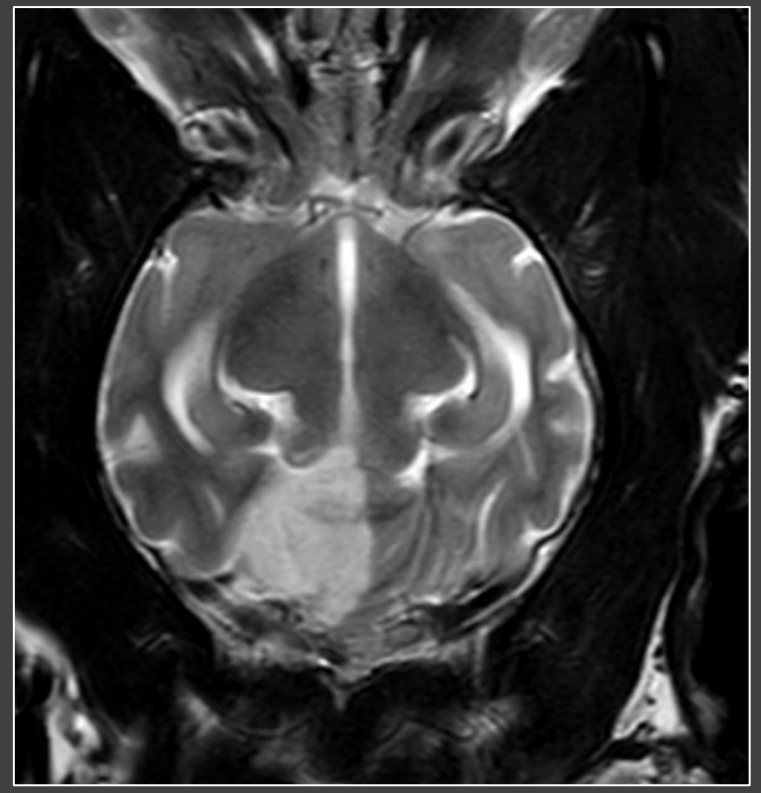
But can be:

- toxoplasmosis
- neosporosis
- baylisascaris
- angyostrongylus
- drug induced

Infarct: rostral cerebellar artery



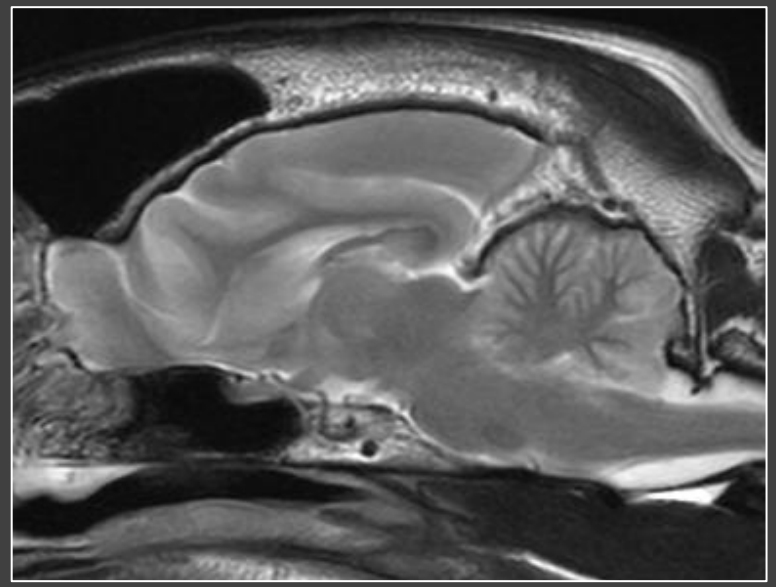




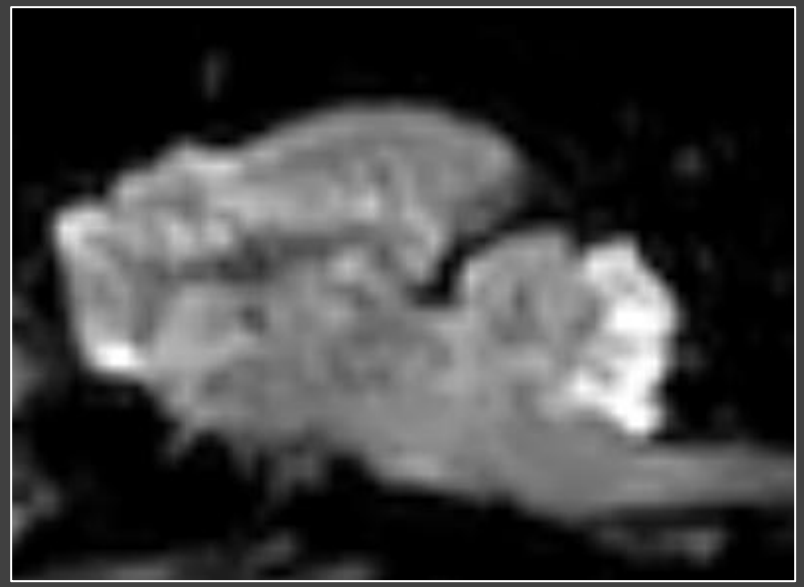
T2

Cocker Spaniel, 14 years

Infarct: caudal cerebellar artery



T2



DWI

ADC

125147

British shorthair cat, 15 years

Infarct: clinical signs in relation to neuroanatomical localisation

Journal of Veterinary Internal Medicine, 2026, Volume 40, Issue 1

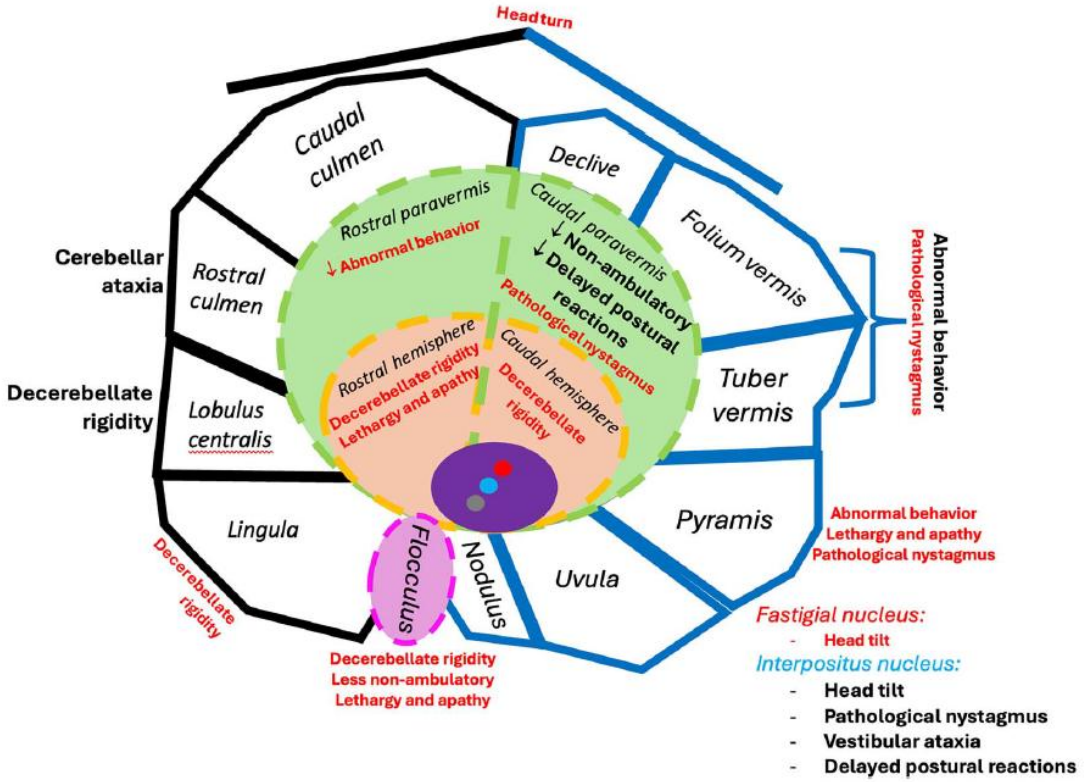
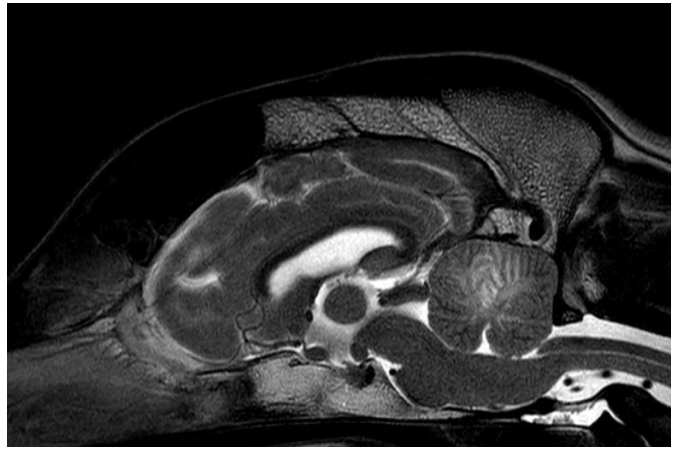
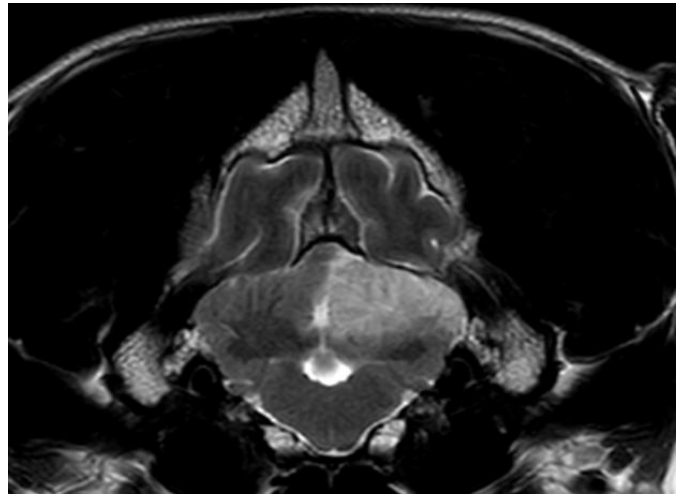


Figure 4 Associations between clinical sign and lesion topography. The significant associations observed in the multivariable analysis are bolded. Associations observed on the univariable analysis only are highlighted in red.

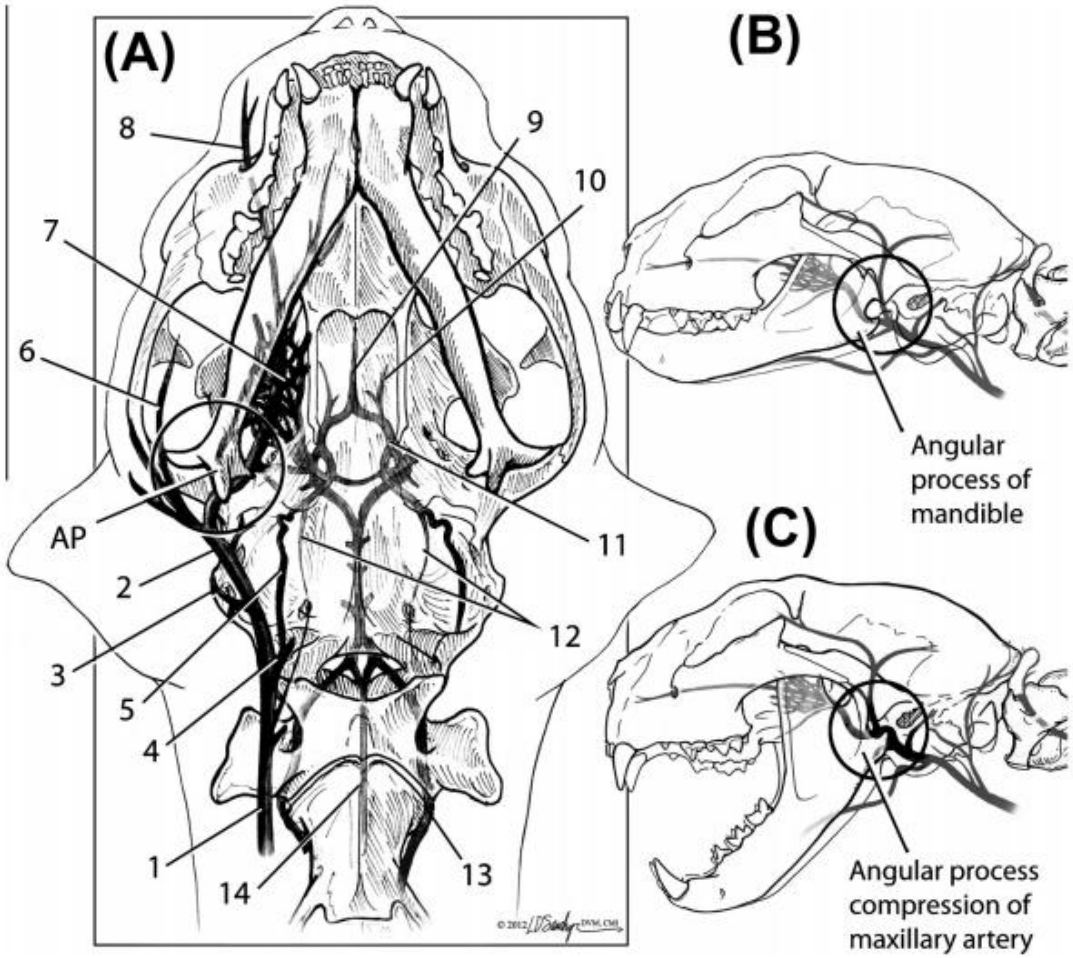




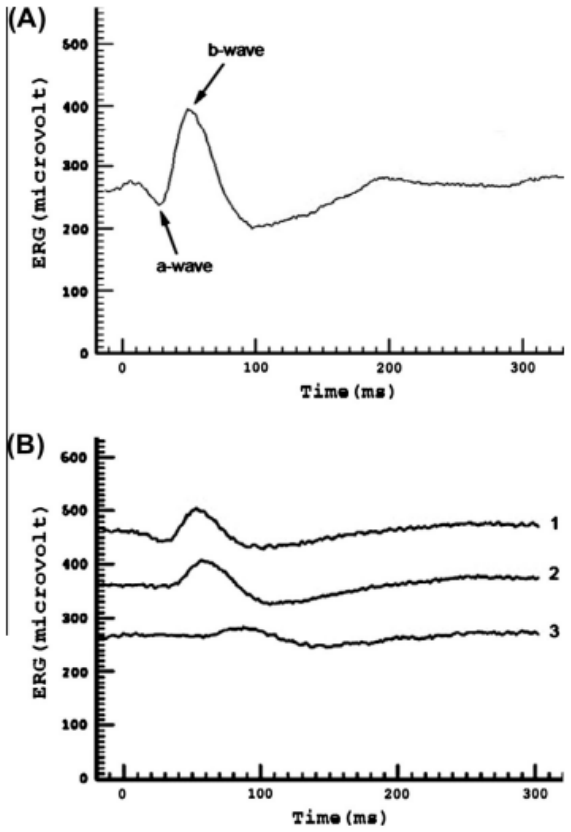
Villalonga et al. 2026

Postanesthetic blindness in cats

A.L. Barton-Lamb et al./The Veterinary Journal 196 (2013) 325–331



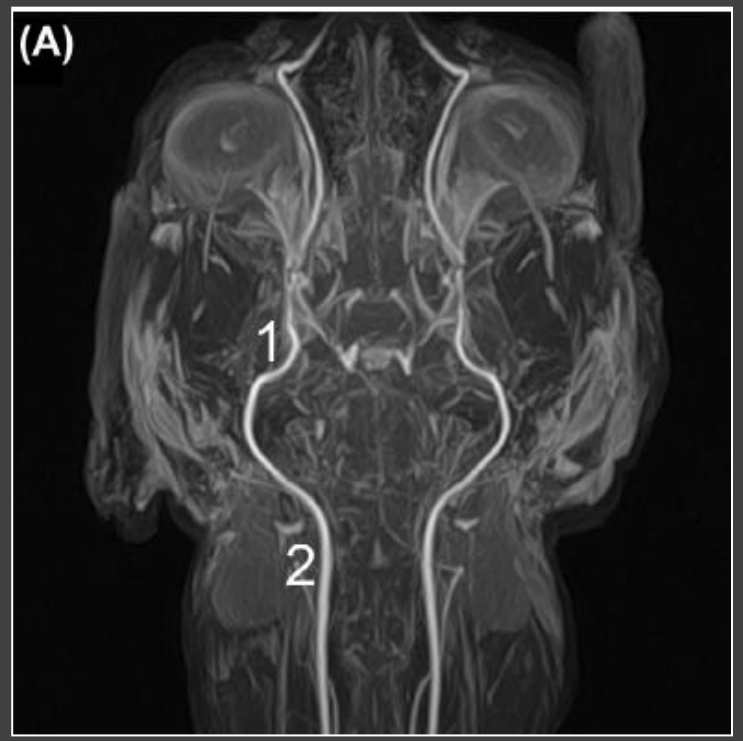
electroretinogram



under anesthesia
mouth closed

under anesthesia
mouth open

Postanesthetic blindness in cats



DSH cat, years
mouth closed

DSH cat, 2 years
mouth opened with a gag

Evaluation of maxillary arterial blood flow in anesthetized cats with the mouth closed and open

A.L. Barton-Lamb, M. Martin-Flores, P.V. Scrivani, A.J. Bezuidenhout, E. Loew, H.N. Erb, J.W. Ludders

Vet Journal 2013

Time of flight (TOF)

Outcome of ischemic infarcts

Survival and clinical outcome of dogs with ischaemic stroke

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Cerebrovascular accident
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ABSTRACT

The objectives of the present study were to investigate survival time, possible predictors of survival and clinical outcome in dogs with ischaemic stroke. A retrospective study of dogs with a previous diagnosis of ischaemic stroke diagnosed by magnetic resonance imaging (MRI) was performed. The association between survival and the hypothesised risk factors was examined using univariable exact logistic regression. Survival was examined using Kaplan–Meier and Cox regression.

Twenty-two dogs were identified. Five dogs (23%) died within the first 30 days of the stroke event. Median survival in 30-day survivors was 505 days. Four dogs (18%) were still alive by the end of the study. Right-sided lesions posed a significantly increased risk of mortality with a median survival time in dogs with right-sided lesions of 24 days vs. 602 days in dogs with left sided lesions ($P = 0.006$). Clinical outcome was considered excellent in seven of 17 (41%) 30-day survivors. Another seven 30-day survivors experienced new acute neurological signs within 6–17 months of the initial stroke event; in two of those cases a new ischaemic stroke was confirmed by MRI. In conclusion, dogs with ischaemic stroke have a fair to good prognosis in terms of survival and clinical outcome. However, owners should be informed of the risk of acute death within 30 days and of the possibility of new neurological events in survivors. Mortality was increased in dogs with right-sided lesions in this study.

died: **23 %**
(within 30 days)

excellent: **41%**
(of 30 day survivors)

Infarct: further diagnostics

J Vet Intern Med 2005;19:725-731

Results of Diagnostic Investigations and Long-Term Outcome of 33 Dogs with Brain Infarction (2000–2004)

L. Garosi, J.F. McConnell, S.R. Platt, G. Barone, J.C. Baron, A. de Lahunta, and S.J. Schatzberg

Medical records of 33 dogs presented for acute onset, nonprogressive, intracranial dysfunction that had a magnetic resonance imaging diagnosis of brain infarction were reviewed. Postmortem confirmation of brain infarction was available in 10 dogs. All dogs were evaluated by CBC, serum biochemistry, thyroid and adrenal testing, urinalysis, thoracic and abdominal imaging, and cerebrospinal fluid analysis. Results of coagulation profile and arterial blood pressure were available in 32/33 and 28/33 dogs, respectively. On the basis of the imaging findings, infarcts were classified depending on their type (territorial or lacunar) and location within the brain (telencephalic, 10/33; thalamic/midbrain, 8/33; cerebellar, 15/33). No marked associations among location or type of infarct and patient age and sex, occurrence of systemic hypertension, and the presence or absence of a concurrent medical condition were identified. Small breed dogs (≤15 kg) were significantly more likely to have territorial cerebellar infarcts, whereas large breed dogs (>15 kg) were significantly more likely to have lacunar thalamic or midbrain infarcts. A concurrent medical condition was detected in 18/33 dogs with brain infarcts, with chronic kidney disease (8/33) and hyperadrenocorticism (6/33) being most commonly encountered. Of 33 dogs, 10 were euthanized because of the severity and lack of improvement of their neurologic status or the severity of their concurrent medical condition. No association was identified between type or location of infarct and patient outcome. Dogs with concurrent medical conditions had significantly shorter survival times than those with no identifiable medical condition and were significantly more likely to suffer from recurrent neurologic signs because of subsequent infarcts.

Key words: Cerebrovascular; Hypertension; Lacunar; Magnetic resonance imaging; Thrombosis.

30 – 50 % of patients have a concurrent medical condition …

… that could have caused the infarct.

Treatment of ischemic infarcts

Voltaire:

“The art of medicine consists of amusing the patient while nature cures the disease.”

1694 - 1778

We rely on:

- survival of penumbra
- re-vascularisation
- brain plasticity

Propentofylline

Case 3

Germanic Bearhound, 6 years, male: **Odin**

- somehow abnormal gait for 3 – 6 months
- appears to be slightly progressive
- was diagnosed with laryngeal paralysis 3 years ago

Germanic Bearhound, 6 years, male

Leonberger

2. Select the category and then the desired test(s)

This might interest you | Packages / Test combinations | Hereditary diseases / DLA | Coat colour / coat length | DNA profile | Breed determination | Storage

± Erbkrankheiten / DLA

8294	Chondrodysplasia (CDPA) and -dystrophia (CDDY) (IVDD risk)	€62.00	8158	Degenerative myelopathy exon 2 (DM exon 2)	€72.00
8997	DLA typing	€98.00	8154	Hyperuricosuria (HUU/SLC)	€62.00
8685	Larynxparalysis with polyneuropathy type 3 (LPPN3)	€62.00	8487	Leonberger polyneuropathy 1 (LPN1)	€62.00
8494	Leonberger polyneuropathy 2 (LPN2)	€62.00	8283	Leukoencephalomyelopathy (LEMP)	€62.00
				Typical symptoms of LEMP are coordination and movement disorders. Only a few months after the first symptoms, the affected dogs will not be able to stand up or to walk any more. The age of onset is about 1 – 3 years.	
8062	Malignant hyperthermia (MH)	€62.00	8127	Progressive retinal atrophy (prcd-PRA)	€62.00
8119	von-Willebrand disease type I (vWD1)	€62.00			

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Ergebnis: Genotyp N/N

Interpretation: Das untersuchte Tier ist reinerbig (homozygot)
für das Wildtyp-Allel. Es trägt somit nicht die ursächliche
Mutation für LPPN3 im CNTNAP1-Gen.

Erbgang: autosomal-rezessiv

Eine Korrelation zwischen dieser Mutation und der Erkrankung
wurde bisher bei folgenden Rassen beschrieben: Bernhardiner, Labrador

Leukoenzephalomyelopathie (LEMP) - PCR

Ergebnis: Genotyp N/N

Interpretation: Das untersuchte Tier ist reinerbig (homozygot)
für das WildtypÄAllel. Es trägt somit nicht die ursächliche
Mutation für LEMP im NAPEPLDÄGen.

Erbgang: autosomalÄrezessiv

Eine Korrelation zwischen dieser Mutation und der Erkrankung
wurde bisher bei folgenden Rassen beschrieben: Leonberger

Polyneuropathie (LPN1) - PCR

Ergebnis: Genotyp LPN1/LPN1

Interpretation: Das untersuchte Tier ist reinerbig (homozygot)
für die ursächliche Mutation für LPN1 im ARHGEF10-Gen.

Erbgang: autosomal-rezessiv

Eine Korrelation zwischen dieser Mutation und der Erkrankung
wurde bisher bei folgenden Rassen beschrieben: Leonberger
ACHTUNG: Es gibt in dieser Rasse weitere ursächliche Mutationen

Leonberger Polyneuropathie 2 (LPN2) - PCR

Ergebnis: Genotyp N/N
```

Cased based work-up

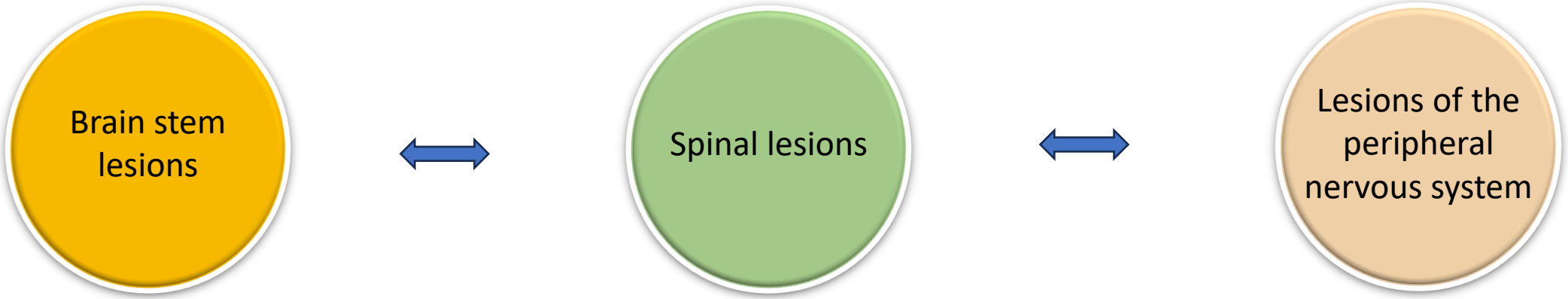
Patients with Paresis/Paralysis

Thomas Flegel

Dipl. ECVN, Dipl. ACVIM

European Specialist in Veterinary Neurology

Potential localisations



- paresis
- reduced mentation
- reduced paw placing reactions
- cranial nerve deficits
- **vestibular syndrome**
- seizures

- paresis/plegia
- reduced paw placing reactions
- reduced segmental spinal reflexes in front or rear limbs

- lack of symptomes pointing towards the head

- tetraparesis/plegia
- reduced paw placing reactions
- generalised reduced segmental spinal reflexes

Case 1

Mixed breed dog, 4 years, male: **Rudi**

- suddenly abnormal gait in the rear since yesterday
- can‘t jump on the sofa anymore
- radiographs performed by referring vet: normal
- no improvement under pain medication (NSAID)
- completely unable to walk since this morning

Mixed breed dog, 4 years, male: **Rudi**

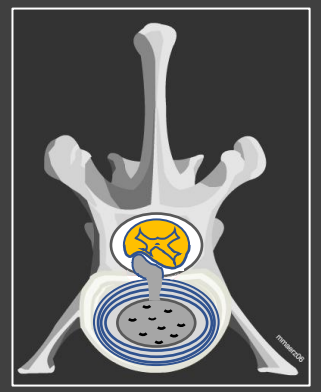
Dept. for Small Animals, Leipzig University

Mixed breed dog

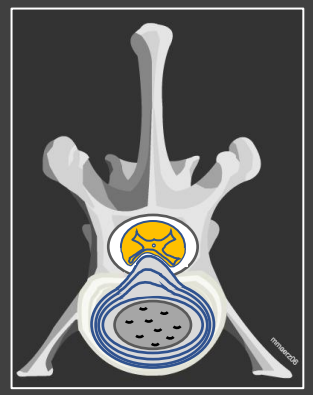
4 years, male

Types of intervertebral disc disease

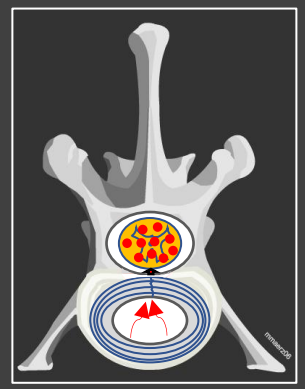
Type I
Extrusion



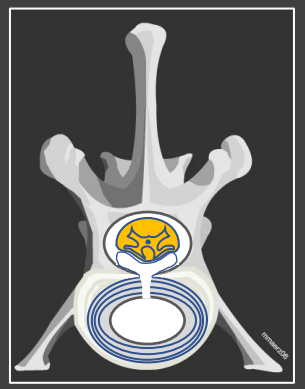
Type II
Protrusion



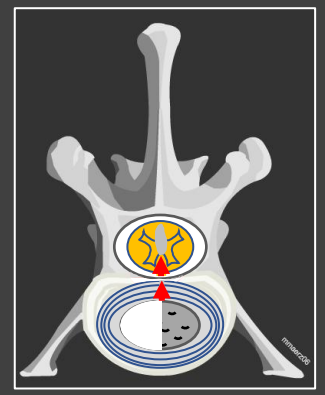
„Type III"
ANNPE



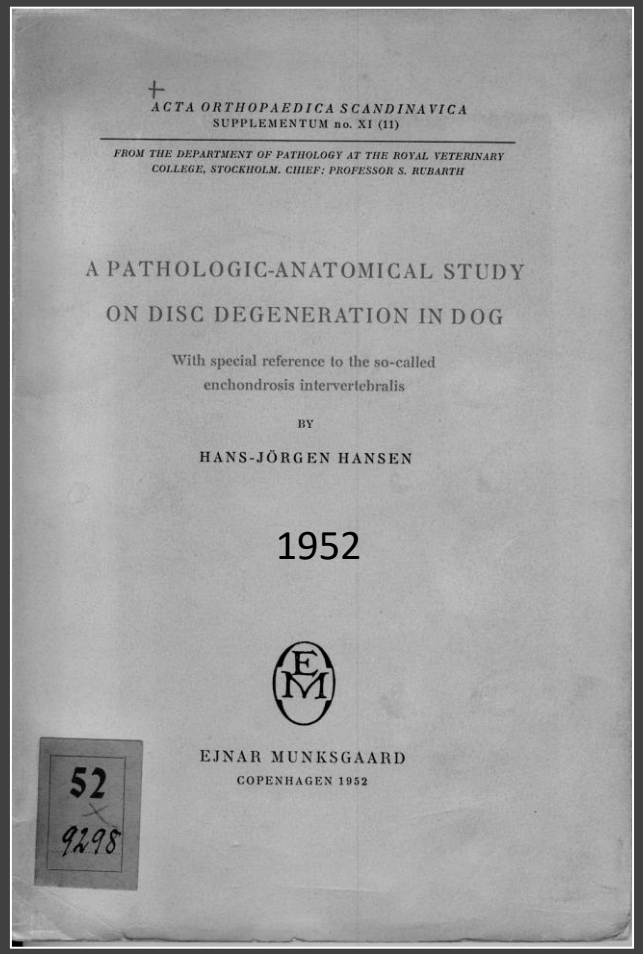
„Type IV"
HNPE



„Type V"
IIVDE



Where everything started…



ACTA ORTHOPAEDICA SCANDINAVICA
SUPPLEMENTUM no. XI (11)

FROM THE DEPARTMENT OF PATHOLOGY AT THE ROYAL VETERINARY COLLEGE, STOCKHOLM. CHIEF: PROFESSOR S. RUBARTH

A PATHOLOGIC-ANATOMICAL STUDY
ON DISC DEGENERATION IN DOG

With special reference to the so-called enchondrosis intervertebralis

BY

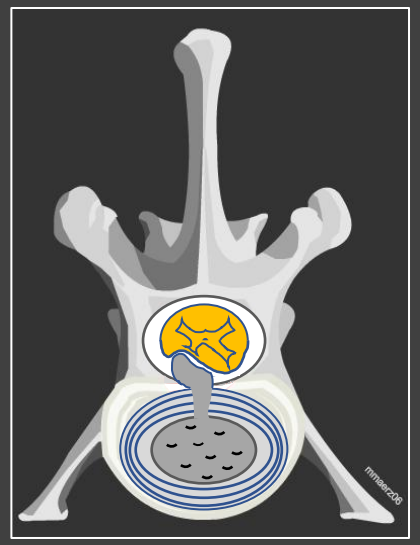
HANS-JÖRGEN HANSEN

1952

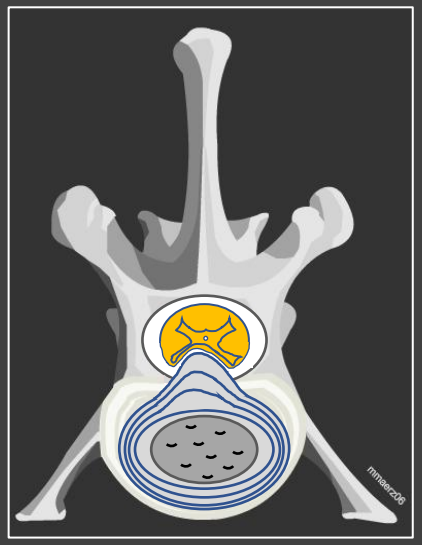
EJNAR MUNKSGAARD
COPENHAGEN 1952

52
9298

Type I



Type II



Hansen type I: extrusion

PLOS One 2013

How Long and Low Can You Go? Effect of Conformation on the Risk of Thoracolumbar Intervertebral Disc Extrusion in Domestic Dogs

Rowena M. A. Packer[1], Anke Hendricks[2], Holger A. Volk[2], Nadia K. Shihab[3], Charlotte C. Burn[1]*

1 Department of Production and Population Health, Royal Veterinary College, University of London, Hertfordshire, United Kingdom, **2** Department of Clinical Science and Services, Royal Veterinary College, University of London, Hertfordshire, United Kingdom, **3** Southern Counties Veterinary Specialists, Unit 6, Forest Corner Farm, Ringwood, Hampshire, United Kingdom

Abstract

Intervertebral disc extrusion (IVDE) is a common neurological disorder in certain dog breeds, resulting in spinal cord compression and injury that can cause pain and neurological deficits. Most disc extrusions are reported in chondrodystrophic breeds (e.g. Dachshunds, Basset Hounds, Pekingese), where selection for 'long and low' morphologies is linked with intervertebral discs abnormalities that predispose dogs to IVDE. The aim of this study was to quantify the relationship between relative thoracolumbar vertebral column length and IVDE risk in diverse breeds. A 14 month cross-sectional study of dogs entering a UK small animal referral hospital for diverse disorders including IVDE was carried out. Dogs were measured on breed-defining morphometrics, including back length (BL) and height at the withers (HW). Of 700 dogs recruited from this referral population, measured and clinically examined, 79 were diagnosed with thoracolumbar IVDE following diagnostic imaging ± surgery. The BL:HW ratio was positively associated with IVDE risk, indicating that relatively longer dogs were at increased risk, e.g. the probability of IVDE was 0.30 for Miniature Dachshunds when BL:HW ratio equalled 1.1, compared to 0.68 when BL:HW ratio equalled 1.5. Additionally, both being overweight and skeletally smaller significantly increased IVDE risk. Therefore, selection for longer backs and miniaturisation should be discouraged in high-risk breeds to reduce IVDE risk. In higher risk individuals, maintaining a lean body shape is particularly important to reduce the risk of IVDE. Results are reported as probabilities to aid decision-making regarding breed standards and screening programmes reflecting the degree of risk acceptable to stakeholders.

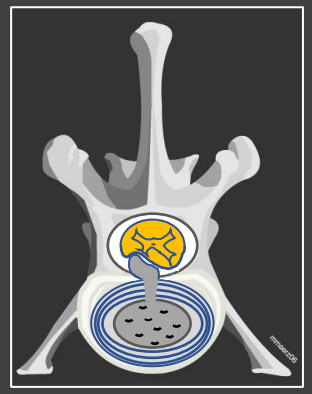
Ration back length to height at withers:

1,1	risk of IVDD:	30%
1,5	risk of IVDD:	68%

Hansen type I: extrusion

Type I

Extrusion



- chondrodystrophic breeds
- young adult to middle aged
- acute symptomes

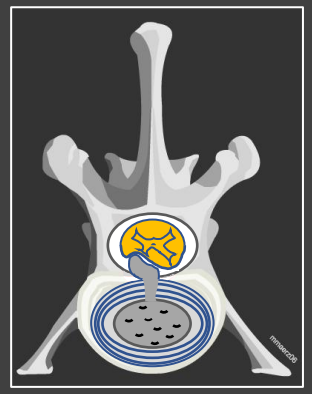
- Dachshund
- Havanesee
- Pekingese
- Maltese
- Shih Tzu
- Lhasa Apso
- Basset
- Welsh Corgi
- Tibetan Spaniel
- Cavalier King Charles Sp.

- Beagle
- American Cocker Spaniel
- Miniature Schnauzer
- Engl. Bulldog
- French. Bulldog
- Bichon Frisé

Hansen type I: genetics

Type I

Extrusion



- chondrodystrophic breeds
- chondroid metaplasia
- young adult to middle aged
- acute symptoms

Retrogen FGF4 (fibroblast growth factor):

Dachshund: chromosome 12, 18

French Bulldog: chromosome 12

Odds ratio to suffer from intervertebral disc disease:

chromosome 12: 51 x (44 x; homozygot)

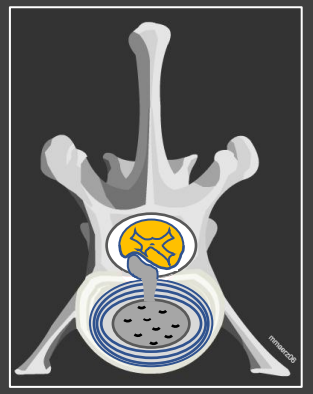
Brown et al. 2017

Dickinson and Banasch 2020
Banasch et al. 2022

Hansen type I: extrusion

Type I

Extrusion

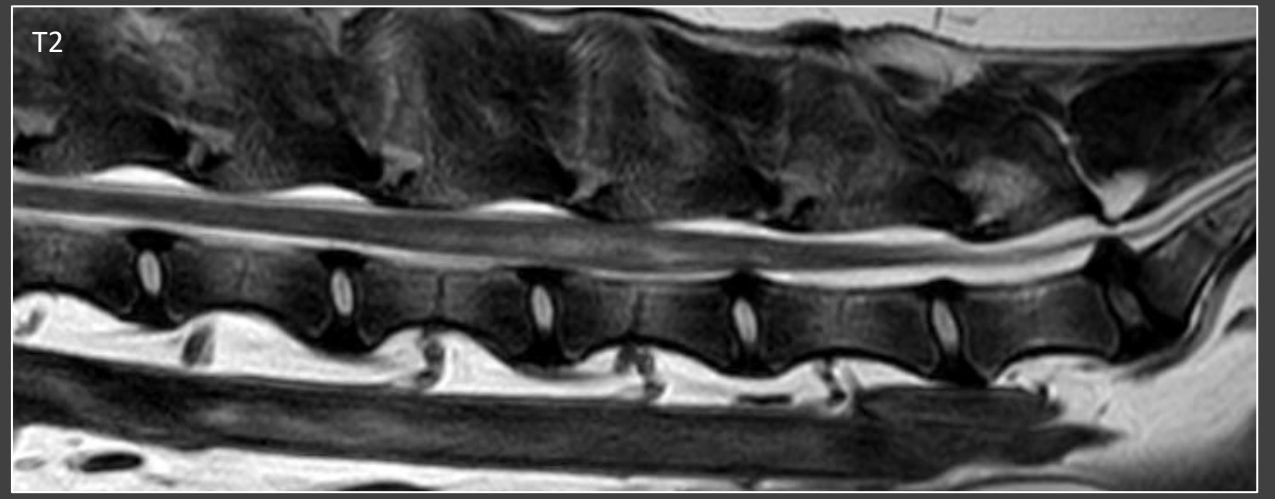


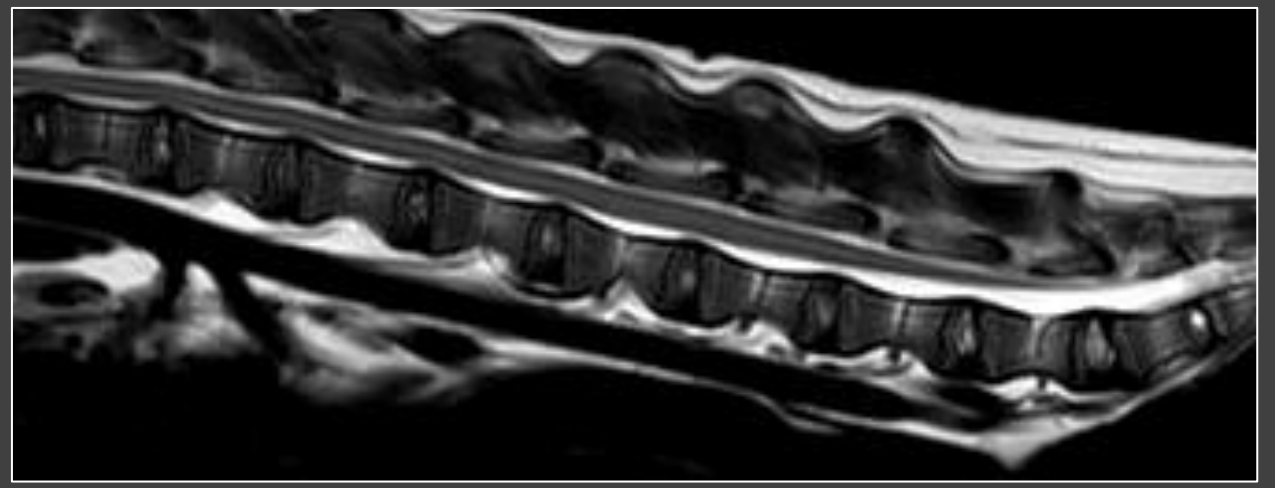
- chondrodystrophic breeds
- young adult to middle aged
- acute symptomes

non-chondro-dystrophic breed
3 years

Dachshund
5 months

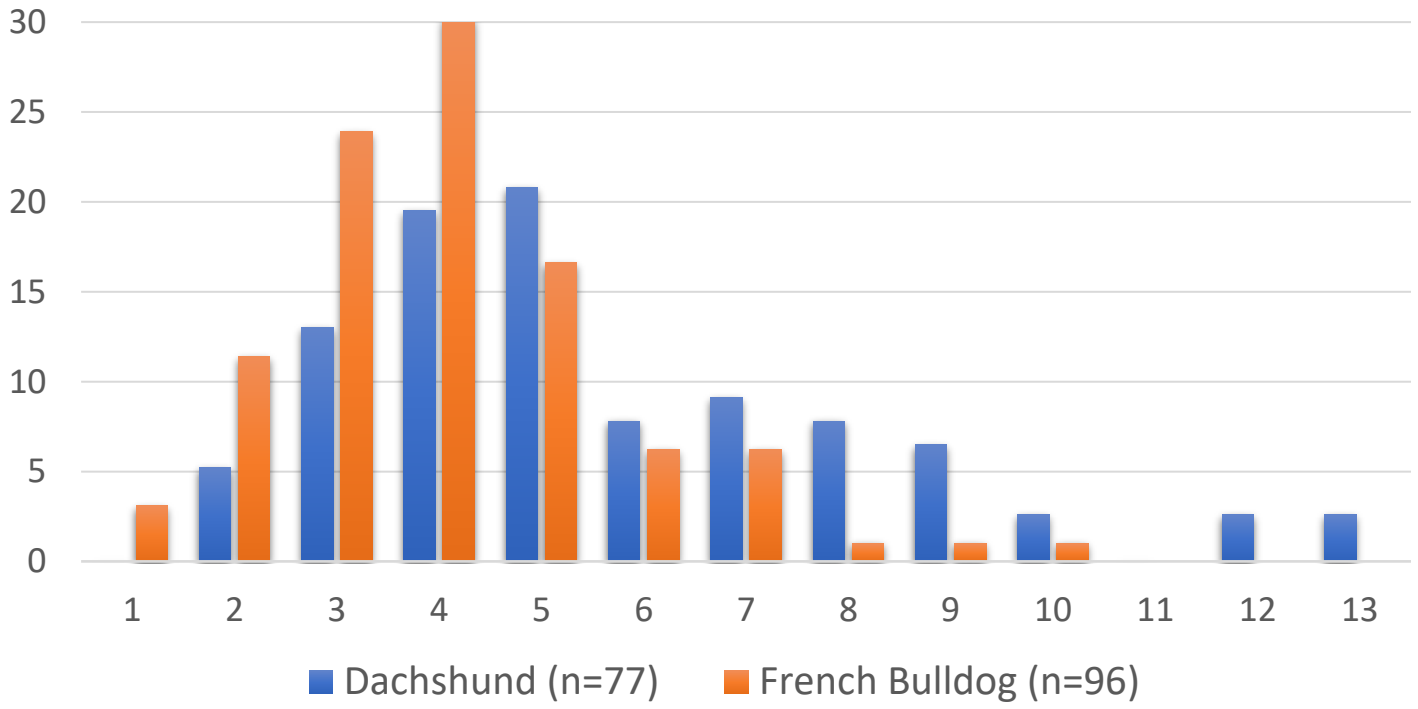
two - white





Hansen type I: Dachshund – French Bulldog

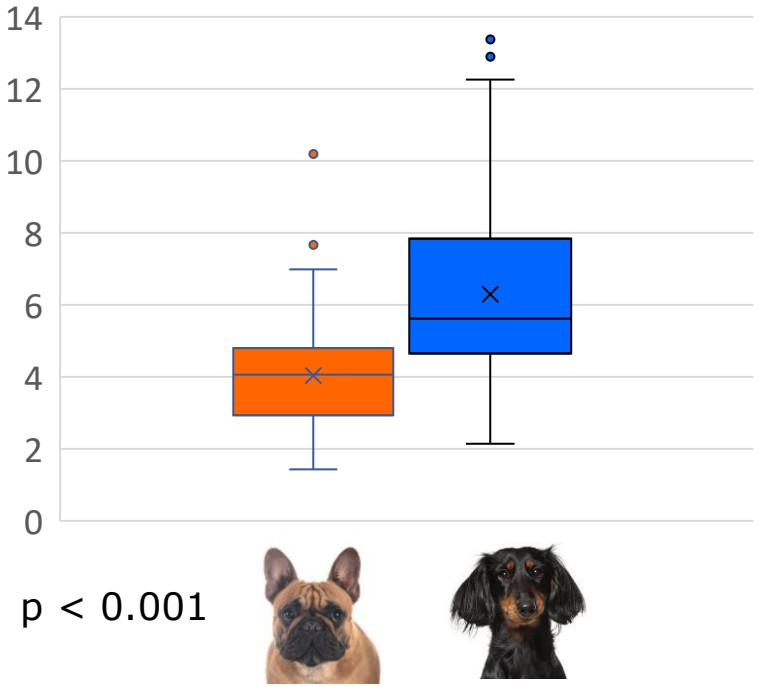
Age distribution (Leipzig University, relative frequency)



Mean age in years:

Dachshund: **6.2**

Fr. Bulldog: **4.0**



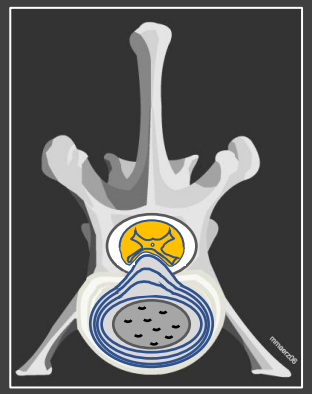
$p < 0.001$

Sarah Feiner, Lea Proske
Dept. For Small Animals, Leipzig University

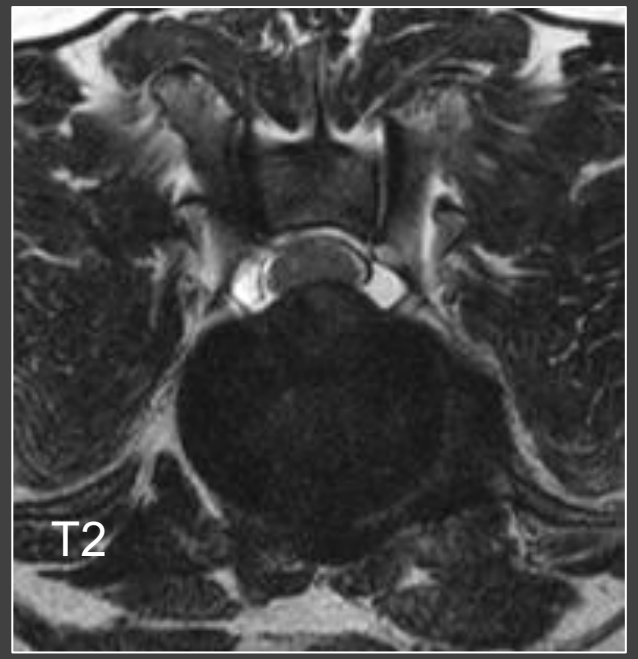
Hansen type II

Type II

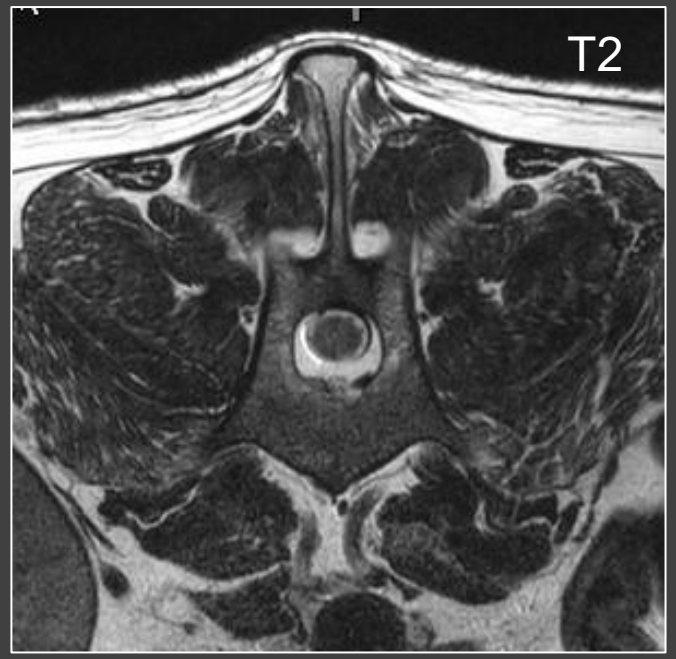
Protrusion



- none-chondrodystrophic breeds
- older dogs
- chronic progressive signs



Typ II extrusion

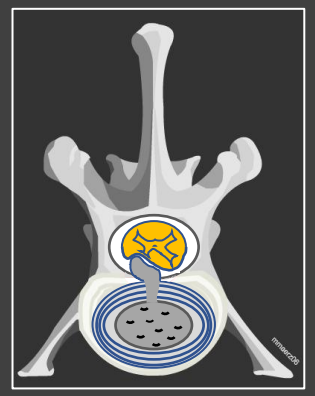


normal

Typ III disc disease

Type I

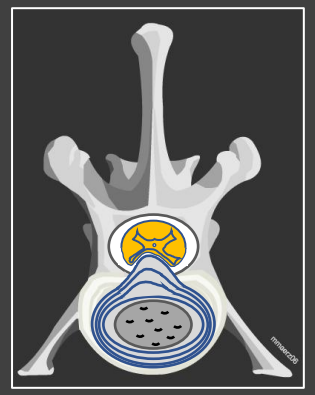
Extrusion



chondrodystrophic breeds
young adult to middle aged
acute symptomes

Type II

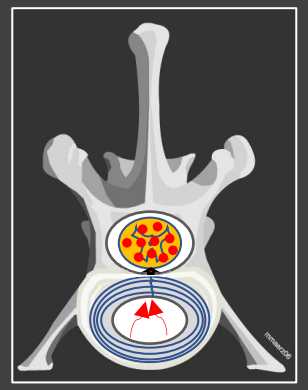
Protrusion



non-chondrodystrophic breeds
older dogs
chronic progressive signs

„Type III"

ANNPE

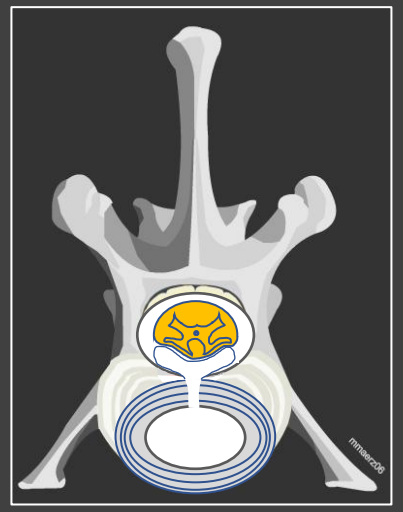


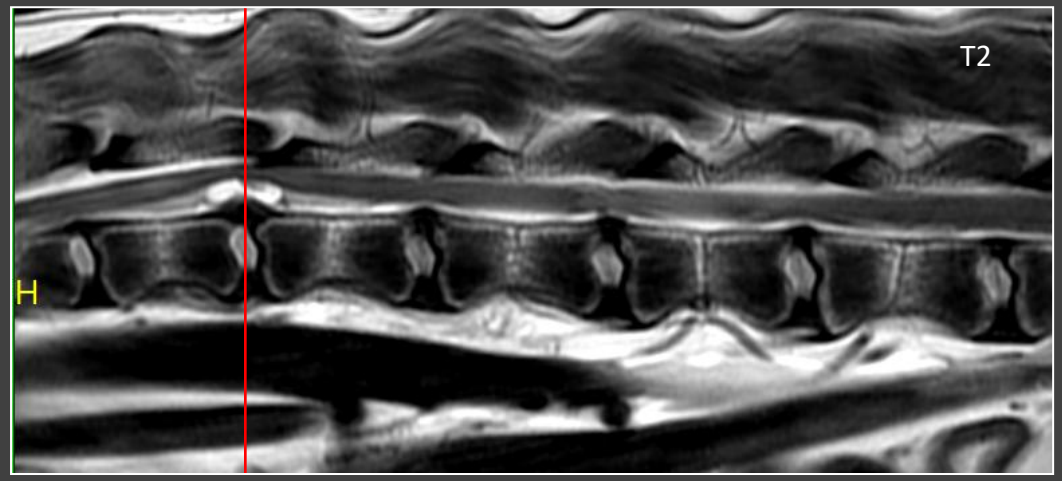
- non-chondrodystrophic breeds
- peracute signs
- spinal cord contusion

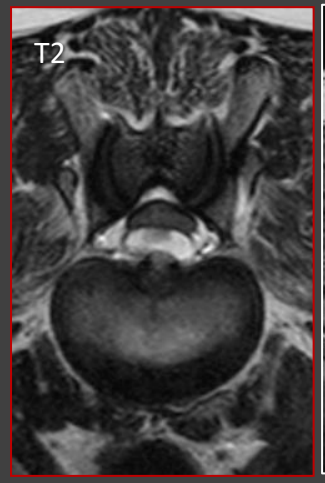
Type IV: hydrated nucleus pulposus extrusion (HNPE)

„Type IV"

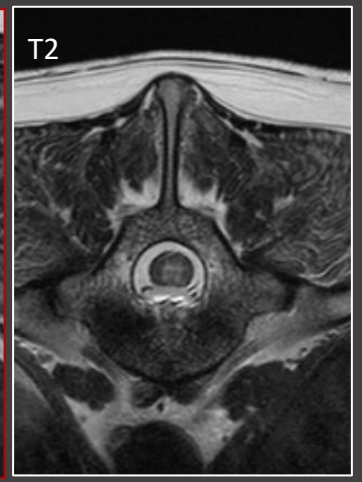
HNPE



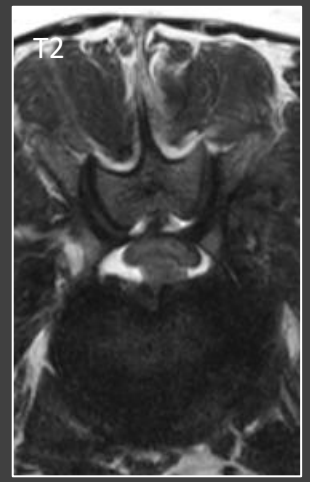




Type IV



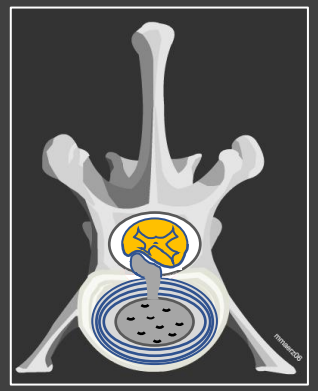
normal



Type II

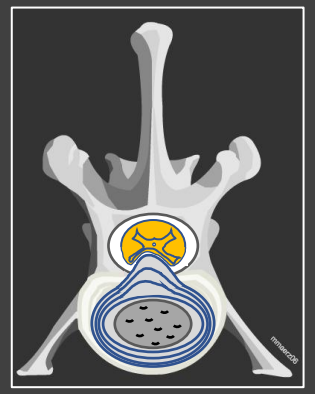
Type V: Intradural/intramedullary intervertebral disc extrusion (IIVDE)

Type I
Extrusion



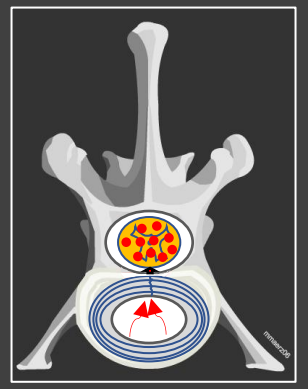
chondrodystrophic breeds
young adult to middle aged
acute symptomes

Type II
Protrusion

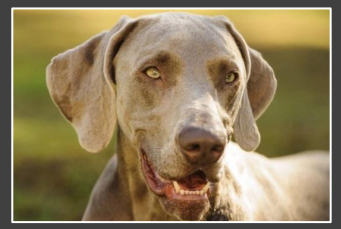


non-chondrodystrophic breeds
older dogs
chronic progressive signs

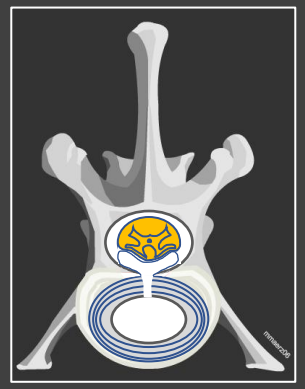
„Type III"
ANNPE



non-chondrodystrophic breeds
peracute signs
spinal cord contusion

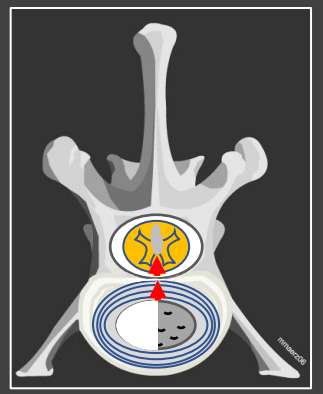


„Type IV"
HNPE



„discal cysts"
non-chondrodystrophic breeds
spinal cord compression by gelationous material
peracute signs

„Type V"
IIVDE



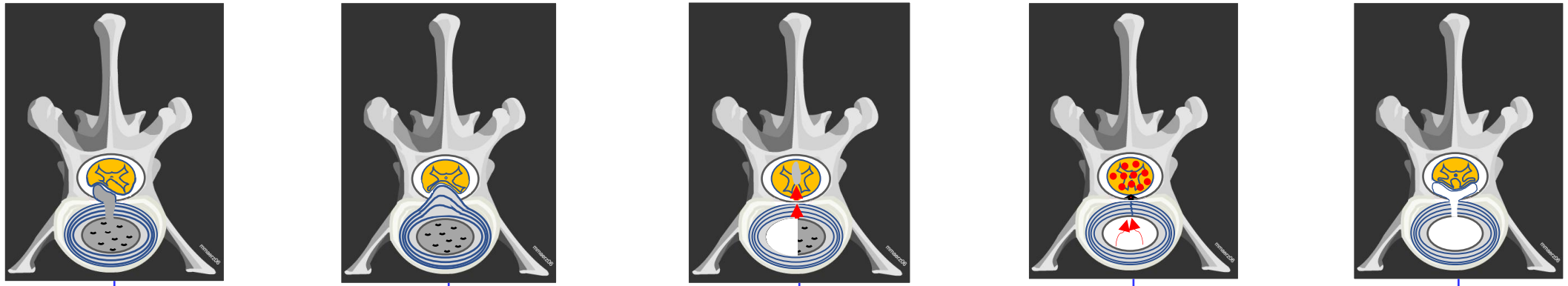
- disc material extrudes with high velocity and penetrates dura mater
- chondrodystrophic and non chondrodystrophic breeds

Intervertebral disc disease: acute versus chronic

Disc disease secondary to disc degeneration

Disc disease in healthy (non-degenerated) disc

Type I	Type II	„Type V“	„Type III“	„Type IV“
Extrusion	**Protrusion**	**IIVDE**	**ANNPE**	**HNPE**



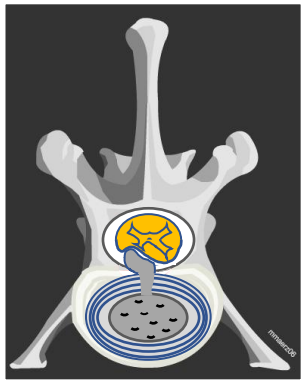
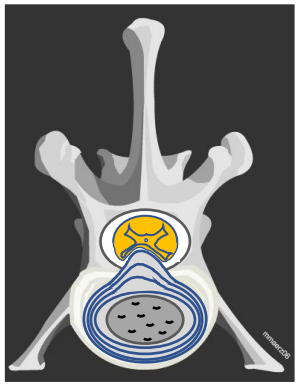
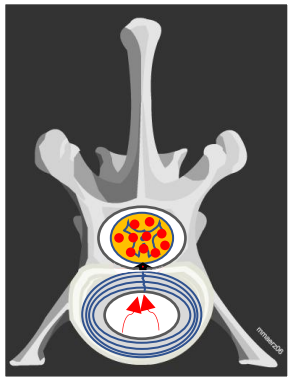
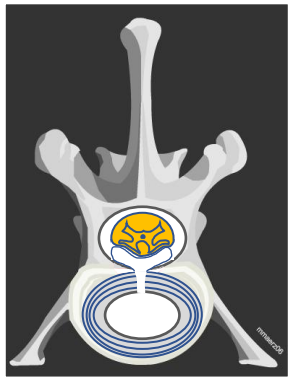
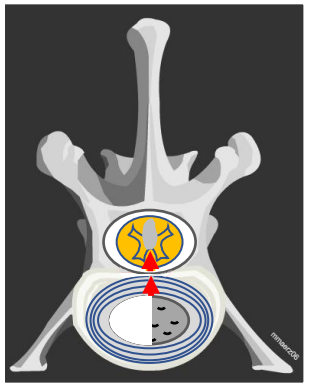
chronic signs

acute signs

Intervertebral disc disease: compressive versus non-compressiv

Compressive disc disease

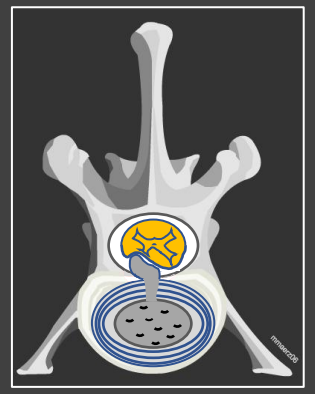
None-compressive disc disease

Type I	Type II	„Type III“	„Type IV“	„Type V“
Extrusion	**Protrusion**	**ANNPE**	**HNPE**	**IIVDE**
				
surgery	surgery	conservative	surgery	???

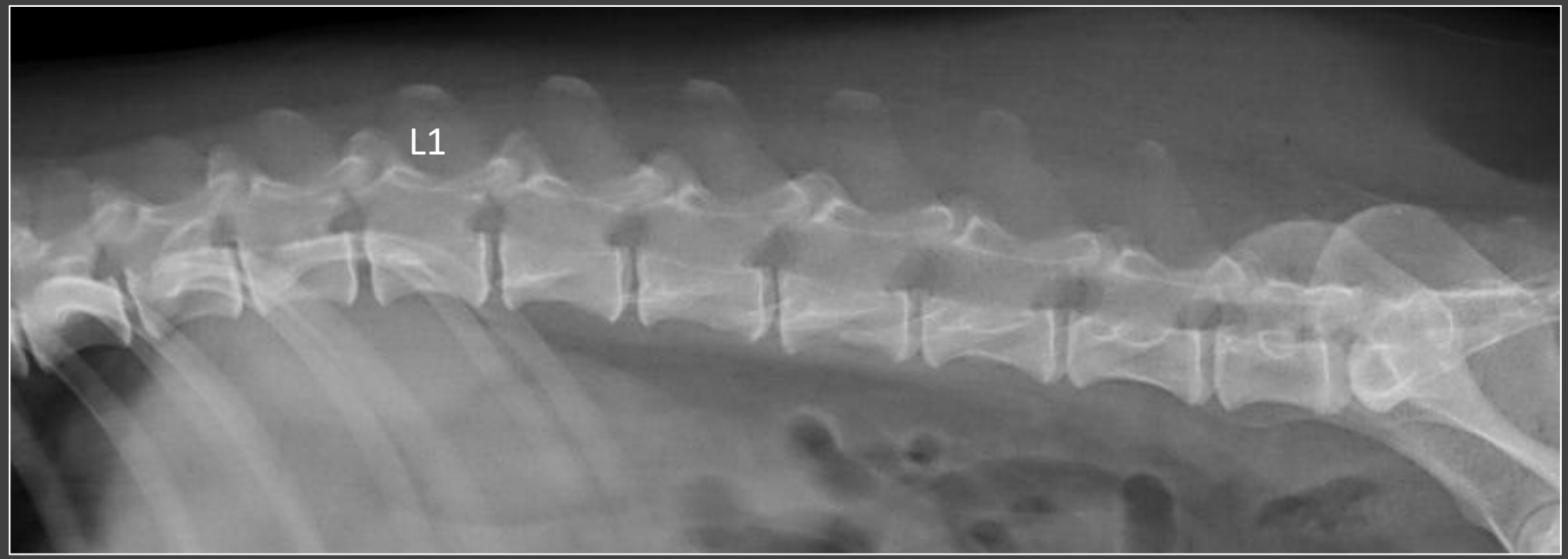
Hansen type I: diagnostics

Type I

Extrusion



Does it have to be the CT or MRI all the time??

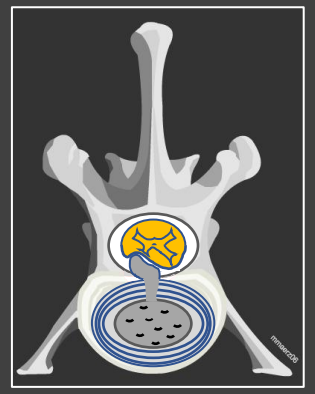


- **narrowed intervertebral disc space**
- wedge-shaped intervertebral disc space
- opacity in the intervertebral foramen

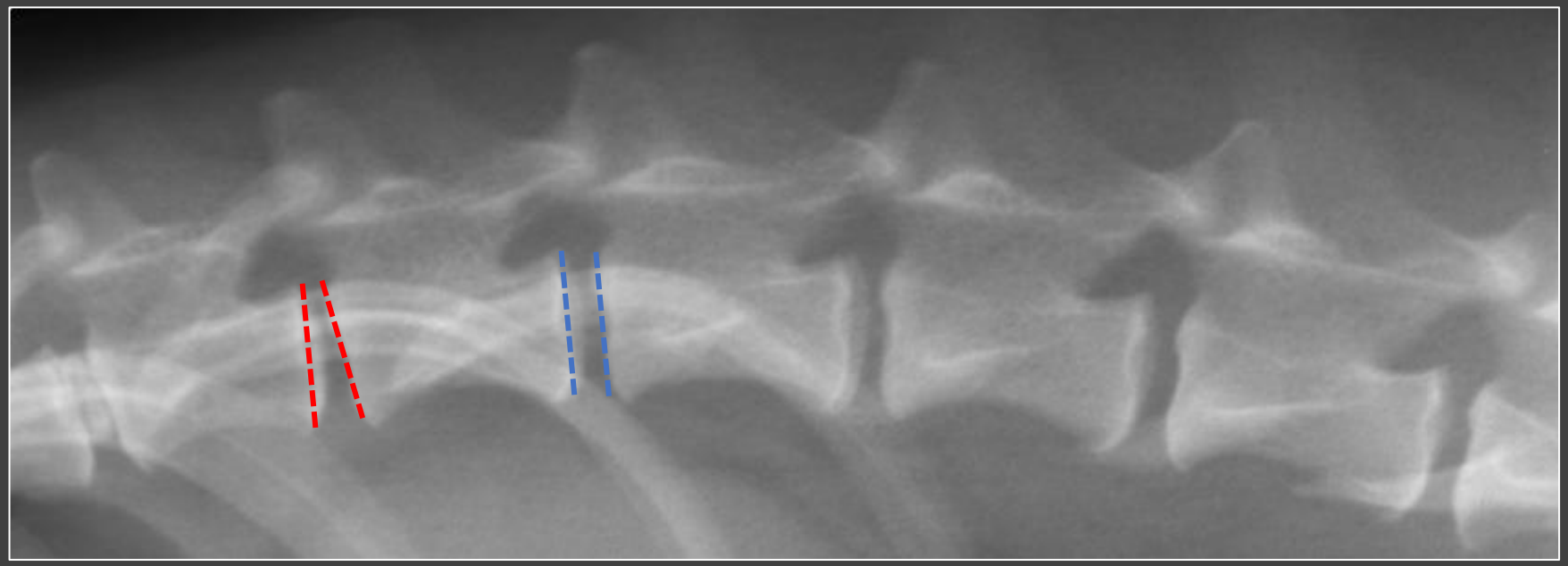
Hansen type I: diagnostics

Type I

Extrusion



Does it have to be the CT or MRI all the time??

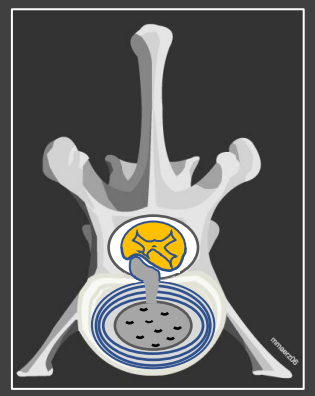


- narrowed intervertebral disc space
- **wedge-shaped intervertebral disc space**
- opacity in the intervertebral foramen

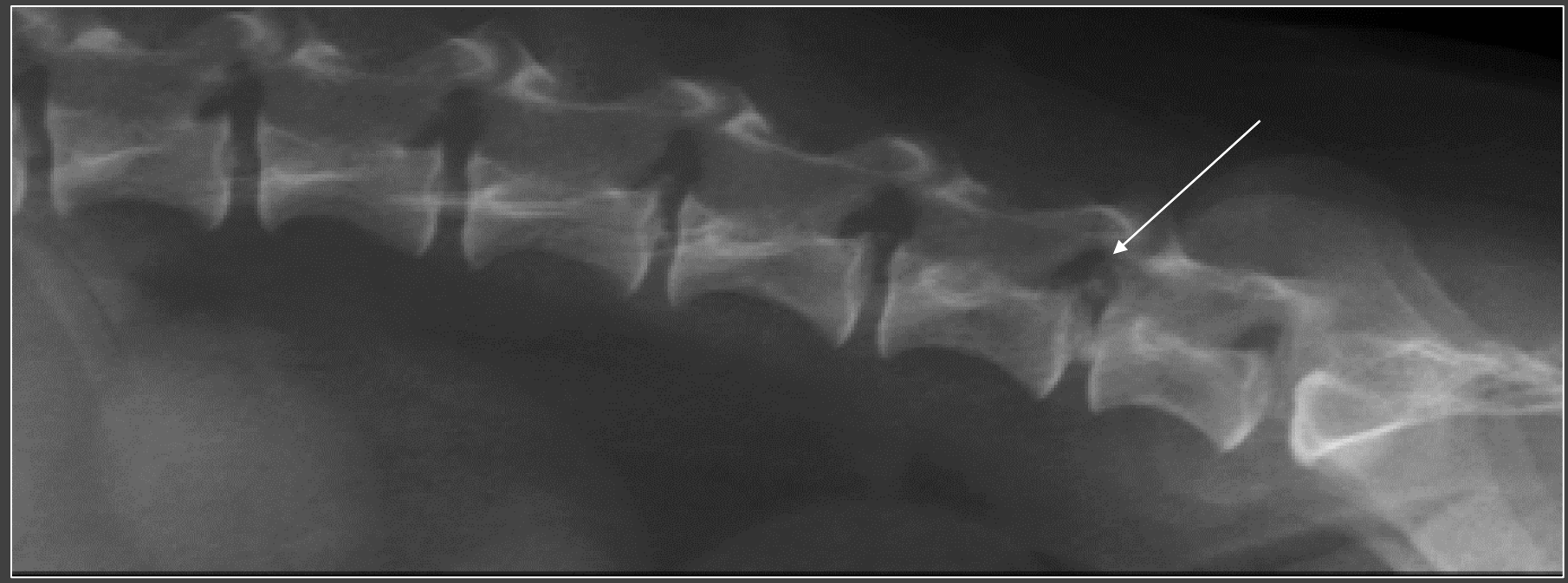
Hansen type I: diagnostics

Type I

Extrusion



Does it have to be the CT or MRI all the time??



- narrowed intervertebral disc space
- wedge-shaped intervertebral disc space
- **opacity in the intervertebral foramen**

BUT ....

Hansen Type I: diagnostics

… a plane radiograph will never lead to a definite diagnosis of intervertebral disc extrusion.

To cut or not to cut … that is here the question

- much less expensive
- no anesthetic risk
- no risk of surgery associated deterioration

500 – 1 000 €

conservative ⟷ surgical

4 000 – 6 000 €

- no spinal cord decompression
- higher risk of relaps

Diagnostic and therapeutic decision

- **patient is still ambulatory:**
  - ataxia
  - reduced paw positioning
  - mild ambulatory paresis
- financial limitations, but intact deep pain perception

- **patient is non-ambulatory**
- severe spinal pain
- no improvement on conservative treatment

Loss of deep pain sensation:

chance to recover ambulation: **about 60%**

success rate: **about 50%**
relaps rate: **about 50%**

Conservative treatment

Exercise restriction

- „cage rest“
- no jumping on furniture nor going stairs
- short leash walks only
- duration: 3 weeks

Pain treatment

- NSAIDs
- no Librela
- < 1 week prednisolone 0.5 mg/kg SID (not in combination with NSAID)
- neuro pain medication:
  gabapentine 10 mg TID
  pregabaline 2-4 mg/kg BID
- acupuncture

Urination

- urinary tract infection 20 – 40%
- urinary catheter versus bladder expression
- alfuzosin 0.5 mg/kg SID
- be restictive with antibiotics
- UTIZen

Physio-therapie

- professional
- magnetic resonance therapy
- Laser therapy

Conservative treatment: prognosis type I (extrusion)

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STANDARD ARTICLE

Recovery of ambulation in small, nonbrachycephalic dogs after conservative management of acute thoracolumbar disk extrusion

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Abstract

Background: Currently, low-level evidence suggests loss of ambulation associated with acute thoracolumbar disk extrusion is best treated by decompressive spinal surgery. Conservative management can be successful, but the proportion of dogs that recover and the fate of herniated material are uncertain.

Objectives: Determine the proportion of nonambulatory dogs with conservatively treated acute thoracolumbar disk extrusion that recover ambulation and measure the change in spinal cord compression during the first 12 weeks after presentation.

Animals: Seventy-two client-owned nonambulatory dogs with acute thoracolumbar intervertebral disk extrusion.

Methods: This is a prospective cohort study. Enrolled dogs underwent magnetic resonance imaging at presentation and owners were provided with conservative management recommendations. Imaging was repeated after 12 weeks. Recovery of ambulation was defined as 10 consecutive steps without falling. Spinal cord compression was determined from the cross-sectional area of the vertebral canal and extradural compressive material at the lesion epicenter. The association between recovery and change in compression over the 12-week observational period was examined.

Results: Forty-nine of fifty-one (96%; 95% confidence interval [CI], 87%-99%) of deep pain-positive and 10/21 (48%; 95% CI, 28%-68%) of deep pain-negative dogs recovered ambulation within the 12-week period. The median time to ambulation was 11 and 25 days for deep pain-positive and -negative dogs, respectively. Reduction in spinal cord compression varied among individuals from minimal to complete and apparently was unrelated to the recovery of ambulation.

Conclusions and Clinical Importance: A high proportion of conservatively treated dogs recovered ambulation after conservative management of acute thoracolumbar disk herniation. Recovery was not dependent on the resolution of compression.

Recovery of unsupported ambulation in non-ambulatory dogs:

deep pain positive:	**96 %**
deep pain negative:	**48 %**

Duration until unsupported ambulation in non-ambulatory dogs:

deep pain positive:	**11 days**
deep pain negative:	**25 days**

Therapeutic decision

- **patient is still ambulatory**
- **Acute clinical signs** (< 1 week)
- financial limitatios (if deep pain sensation is preserved)

- **none ambulatory**
- **severe pain unresponsive to treatment**
- **no improvement on conservative treatment**

Loss of deep pain sensation:

Chance of recovery of unassisted ambulation:

about 60%

Surgical treatment: prognosis type I (extrusion)

Therapeutic goal:

- ambulatory
- none painful
- controlled urination/defecation

prognosis after surgery

• parese (ambulatory or none-ambulatory):	**95%**
• pleagia with intact deep pain sensation:	**90 - 95%**
• plegia with loss of deep pain sensation (How long?):	**60%**

Relapse rate: **15-20%**

Case 2

West Highland White Terrier, 13 years, male: **Willi**

- problems in both rear limbs for about 2 weeks
- progressive
- no improvement on antiinflammatory medication (NSAIDs)

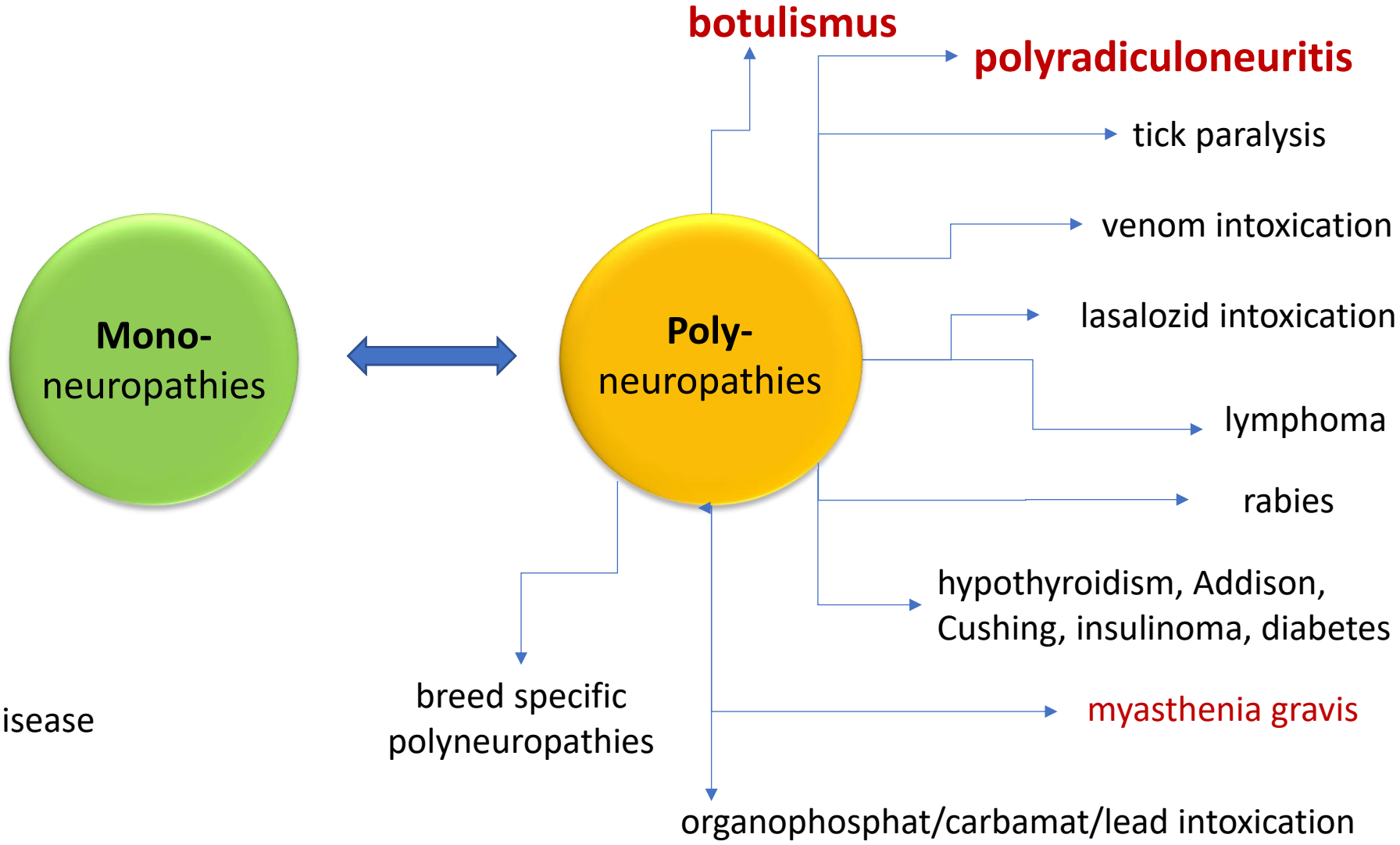
Differentials

cranial nerves:

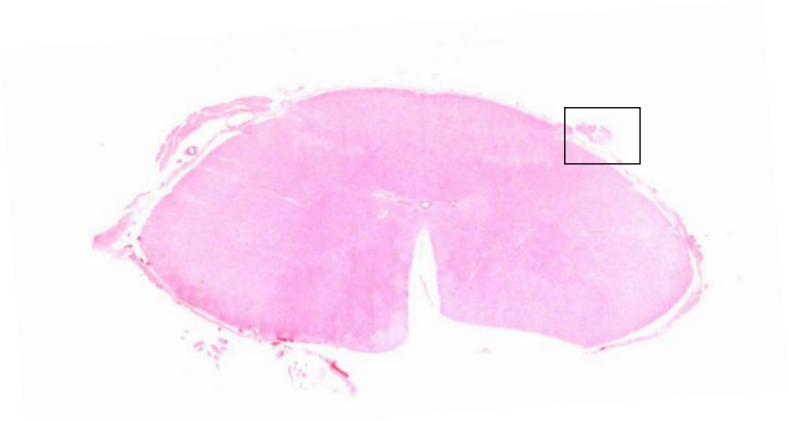
- optic (?)
- oculomotor
- trigeminal
- facial

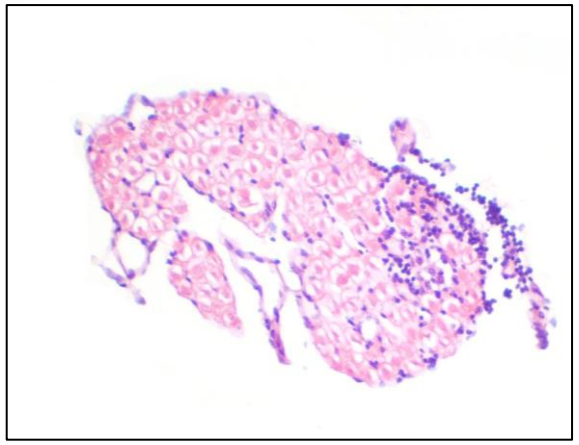
appendicular nerves:

- brachial plexus
- lumbar plexus
- orthostatic tremor
- Dancing Doberman disease



Polyradiculoneuritis





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inflammation of multiple nerve roots (sometimes involving cranial nerves as well)

potential involvement of campylobacter jejuni

„Coonhound paralysis“

- acute paraparesis/plegia
- progression to front limbs with 3 days
- tail motor function nd control of micturition are maintained
- less commonly cranial nerve deficits
- initially: hyperesthesia/allodynia possible
- rarely megaesophagus

Polyradiculoneuritis: treatment

Clinical Course of Acute Canine Polyradiculoneuritis Following Treatment with Human IV Immunoglobulin

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ABSTRACT

Treatment of dogs with acute canine polyradiculoneuritis (ACP) is restricted to physical rehabilitation and supportive care. In humans with Guillain-Barré syndrome, the counterpart of ACP, randomized trials show that IV immunoglobulin (IVIg) speeds recovery. The authors of the current study hypothesized that dogs with ACP would tolerate IVIg well and recover faster than dogs managed with supportive treatment only. Sixteen client-owned dogs with ACP were treated with IVIg, and 14 client-owned dogs served as a retrospective control group. Diagnosis was confirmed using clinical features, electrodiagnostics, cerebrospinal fluid analysis, and muscle/nerve biopsies. The duration of the initial progressive phase, the time from IVIg administration until the dogs were ambulating without assistance, and the duration of the complete episode were evaluated. Adverse reactions (anaphylaxis, mild hematuria) were observed in two dogs. Dogs treated with IVIg were ambulating without assistance after a median of 27.5 days (range, 15–127 days) from onset of clinical signs. The control group was ambulatory without assistance at a median of 75.5 days (range, 5–220 days). Even though this result is not statistically significant, there is a clear trend toward faster recovery in dogs treated with IVIg. (*J Am Anim Hosp Assoc* 2012; 48:299–309. DOI 10.5326/JAAHA-MS-5651)

Median time to ambulation:

without immunoglobulines:

75 days (5- 220)

with immunoglobulines:

28 days (15 – 127)

Relapse rate: 15 – 20 %

Botulism or Polyradiculoneuritis

	Botulism	Polyradiculoneuritis	Myastenia gravis
Symptomes	flacid tetraparesis/plegia starting in the rear cranial nerve involvement megaesophagus none painful	flacid tetraparesis/plegia starting in the rear less common cranial nerve no megaesophagus paresthesia, allodynia	exercise induced weakness more pronounced in rear limbs cranial nerve involvement possible
Diagnostics	bacteria in faeces toxin detection in faeces Ab detection in serum chest radiographs Electrodiagnostics	Electrodiagnostics ⇑ protein in CSF muskel/nerve biopsy	„tensiolon test“ acetylcholin receptor-Ab repetetive nerve stimulation
Treatment	supportive may be metronidazol p.o.	supportive gapapentine, pregablin immunoglobulines?	pyridostigmine may be prednisolone