XVI CONGRESO ANDALUZ DE VETERINARIOS
ALMERÍA
¿Hay vida sin Resonancia Magnética?
¿Hay luz al final del túnel?
PrideVeterinaryCentre
The University of Nottingham
vetsum+
ECVN
Juan J Mínguez DVM MRCVS
ECVN Board Eligible

Soy neurólogo!! No puedo vivir sin RM!!

Soy neurólogo!! No puedo vivir sin RM!!

Por qué este tema… ?

1 No todo el mundo puede afrontar este gasto…

Por qué este tema… ?

1 No todo el mundo puede afrontar este gasto…

Por qué este tema… ?

2 La resonancia no es una máquina milagrosa!

1 No todo el mundo puede afrontar este gasto…

Por qué este tema… ?

2 La resonancia no es una máquina milagrosa!

3 La Neurología no es solo una RM

En los próximos 50 minutos:

Claramente la RM es útil…

En los próximos 50 minutos:

1 MRI limitaciones y retos…

Claramente la RM es útil…

En los próximos 50 minutos:

1 **MRI limitaciones y retos…**

Claramente la RM es útil…

2 **Podemos sobrevivir sin ella?**

En los próximos 50 minutos:

1 **MRI limitaciones y retos…**

Claramente la RM es útil…

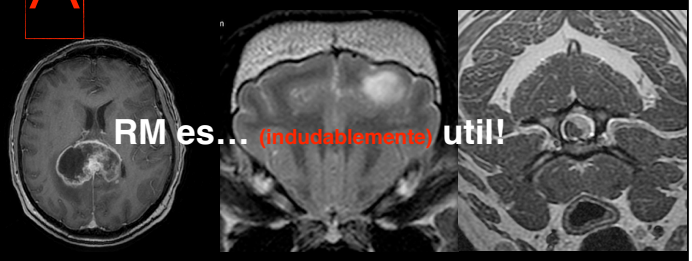
2 **Podemos sobrevivir sin ella?**

3 **Algunos ejemplos**

ACLARACIONES INICIALES

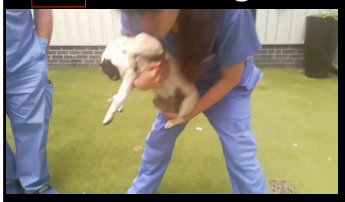
(hay que cubrirse la espalda)

A



B

No todos los casos siguen las reglas…

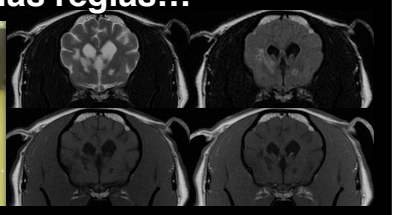


B

No todos los casos siguen las reglas…

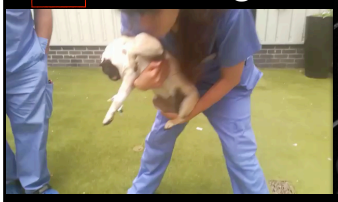
B

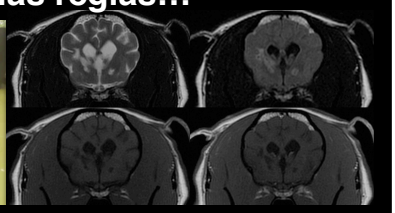
No todos los casos siguen las reglas…



B

No todos los casos siguen las reglas…





Existen discrepancias entre los signos de imagen y la clínica?

1

La RM no es perfecta…

Nadie lo es…

A RM NO ES NEUROLOGÍA!

B LESION EN RM = NO NECESARIAMENTE SIMTOMÁTICA

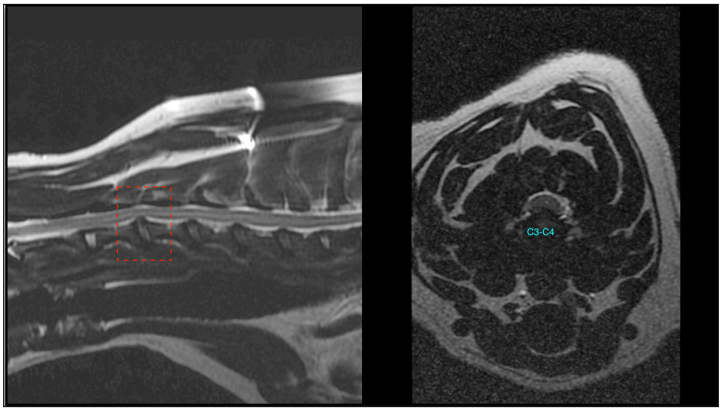
C DIFERENCIAR ENTRE ENFERMEDADES PUEDE SER DIFÍCIL…

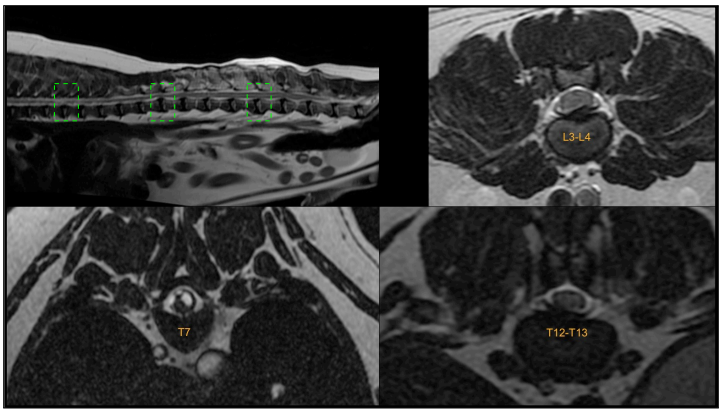
A **RM NO ES NEUROLOGÍA!**

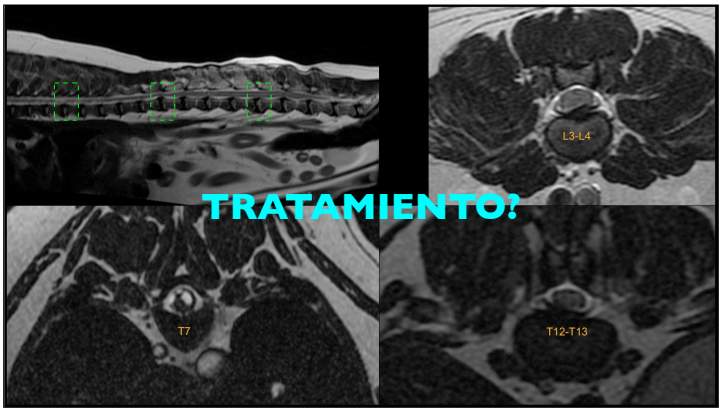
B LESION EN RM = NO NECESARIAMENTE SIMTOMÁTICA

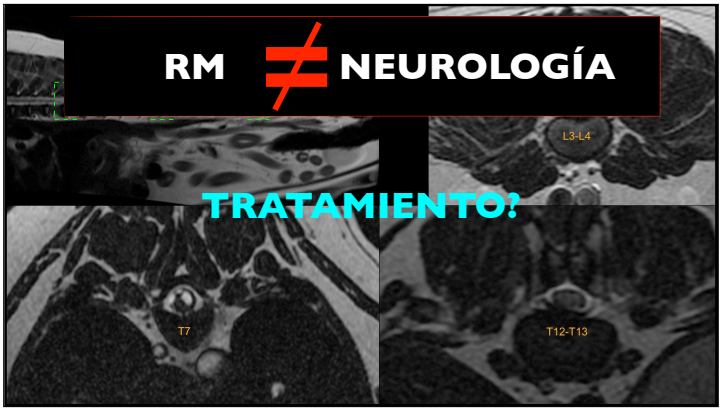
C DIFERENCIAR ENTRE ENFERMEDADES PUEDE SER DIFÍCIL…

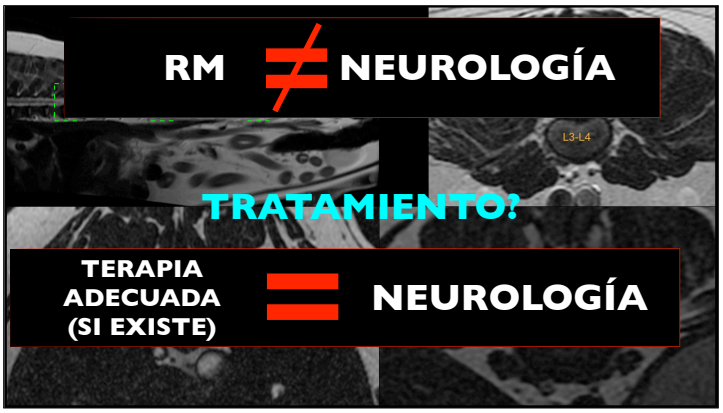
9




L3-L4
T7
T12-T13


L3-L4
TRATAMIENTO?
T7
T12-T13


RM ≠ NEUROLOGÍA
L3-L4
TRATAMIENTO?
T7
T12-T13



A RM NO ES NEUROLOGÍA!

B LESION EN RM = NO NECESARIAMENTE SIMTOMÁTICA

C DIFERENCIAR ENTRE ENFERMEDADES PUEDE SER DIFÍCIL…

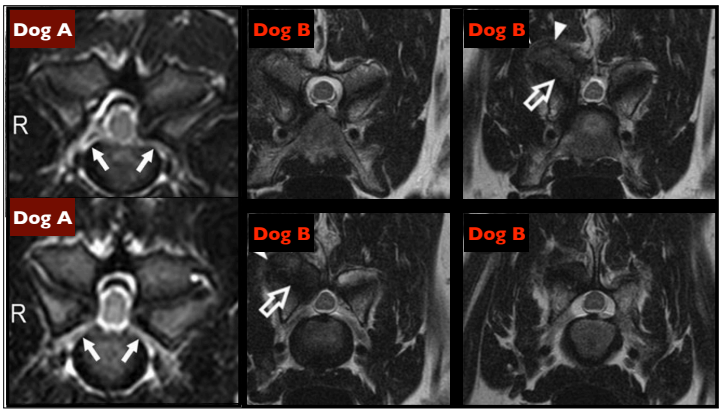
11

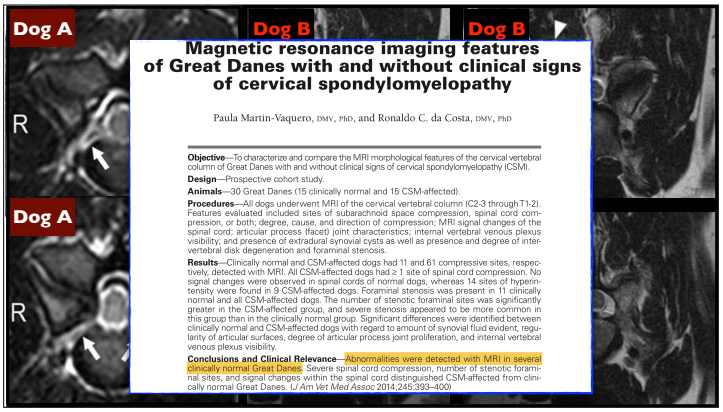
A RM NO ES NEUROLOGÍA!

B **LESION EN RM = NO NECESARIAMENTE SINTOMATICA**

C DIFERENCIAR ENTRE ENFERMEDADES PUEDE SER DIFÍCIL…

11





Magnetic resonance imaging features of Great Danes with and without clinical signs of cervical spondylomyelopathy

Paula Martin-Vaquero, DMV, PhD, and Ronaldo C. da Costa, DMV, PhD

Objective—To characterize and compare the MRI morphological features of the cervical vertebral column of Great Danes with and without clinical signs of cervical spondylomyelopathy (CSM).

Design—Prospective cohort study.

Animals—30 Great Danes (15 clinically normal and 15 CSM-affected).

Procedures—All dogs underwent MRI of the cervical vertebral column (C2-3 through T1-2). Features evaluated included sites of subarachnoid space compression, spinal cord compression, or both; degree, cause, and direction of compression; MRI signal changes of the spinal cord; articular process (facet) joint characteristics; internal vertebral venous plexus visibility; and presence of extradural synovial cysts as well as presence and degree of intervertebral disk degeneration and foraminal stenosis.

Results—Clinically normal and CSM-affected dogs had 11 and 61 compressive sites, respectively, detected with MRI. All CSM-affected dogs had ≥ 1 site of spinal cord compression. No signal changes were observed in spinal cords of normal dogs, whereas 14 sites of hyperintensity were found in 9 CSM-affected dogs. Foraminal stenosis was present in 11 clinically normal and all CSM-affected dogs. The number of stenotic foraminal sites was significantly greater in the CSM-affected group, and severe stenosis appeared to be more common in this group than in the clinically normal group. Significant differences were identified between clinically normal and CSM-affected dogs with regard to amount of synovial fluid evident, regularity of articular surfaces, degree of articular process joint proliferation, and internal vertebral venous plexus visibility.

Conclusions and Clinical Relevance—Abnormalities were detected with MRI in several clinically normal Great Danes. Severe spinal cord compression, number of stenotic foraminal sites, and signal changes within the spinal cord distinguished CSM-affected from clinically normal Great Danes. (*J Am Vet Med Assoc* 2014;245:393–400)

Animals—30 Great Danes (15 clinically normal and 15 CSM-affected).

Procedures—All dogs underwent MRI of the cervical vertebral column (C2-3 through T1-2). Features evaluated included sites of subarachnoid space compression, spinal cord compression, or both; degree, cause, and direction of compression; MRI signal changes of the spinal cord; articular process (facet) joint characteristics; internal vertebral venous plexus visibility; and presence of extradural synovial cysts as well as presence and degree of intervertebral disk degeneration and foraminal stenosis.

Results—Clinically normal and CSM-affected dogs had 11 and 61 compressive sites, respectively, detected with MRI. All CSM-affected dogs had ≥ 1 site of spinal cord compression. No signal changes were observed in spinal cords of normal dogs, whereas 14 sites of hyperintensity were found in 9 CSM-affected dogs. Foraminal stenosis was present in 11 clinically normal and all CSM-affected dogs. The number of stenotic foraminal sites was significantly greater in the CSM-affected group, and severe stenosis appeared to be more common in this group than in the clinically normal group. Significant differences were identified between clinically normal and CSM-affected dogs with regard to amount of synovial fluid evident, regularity of articular surfaces, degree of articular process joint proliferation, and internal vertebral venous plexus visibility.

Conclusions and Clinical Relevance—Abnormalities were detected with MRI in several clinically normal Great Danes. Severe spinal cord compression, number of stenotic foraminal sites, and signal changes within the spinal cord distinguished CSM-affected from clinically normal Great Danes. (*J Am Vet Med Assoc* 2014;245:393–400)

Animals—30 Great Danes (15 clinically normal and 15 CSM-affected).

Procedures—All dogs underwent MRI of the cervical vertebral column (C2-3 through T1-2). Features evaluated included sites of subarachnoid space compression, spinal cord compression, or both; degree, cause, and direction of compression; MRI signal changes of the spinal cord; articular process (facet) joint characteristics; internal vertebral venous plexus visibility; and presence of extradural synovial cysts as well as presence and degree of intervertebral disk degeneration and foraminal stenosis.

Results—Clinically normal and CSM-affected dogs had 11 and 61 compressive sites, respec-

LESIÓN EN RM ≠ ENFERMEDAD CLINICA

this group than in the clinically normal group. Significant differences were identified between clinically normal and CSM-affected dogs with regard to amount of synovial fluid evident, regularity of articular surfaces, degree of articular process joint proliferation, and internal vertebral venous plexus visibility.

Conclusions and Clinical Relevance—Abnormalities were detected with MRI in several clinically normal Great Danes. Severe spinal cord compression, number of stenotic foraminal sites, and signal changes within the spinal cord distinguished CSM-affected from clinically normal Great Danes. (*J Am Vet Med Assoc* 2014;245:393–400)

A RM NO ES NEUROLOGÍA!

B LESION EN RM = NO NECESARIAMENTE SIMTOMÁTICA

C DIFERENCIAR ENTRE ENFERMEDADES PUEDE SER DIFÍCIL…

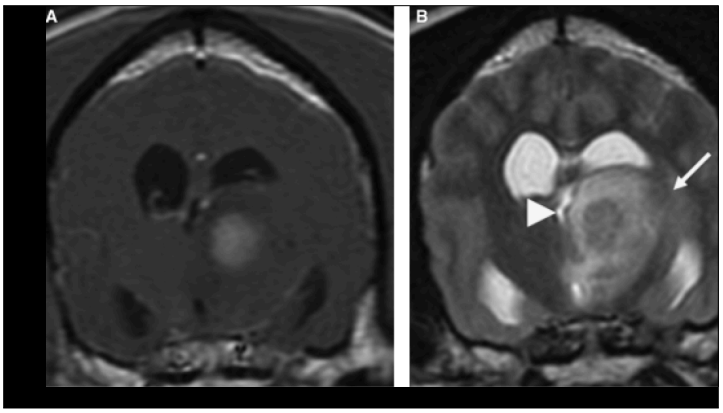
13

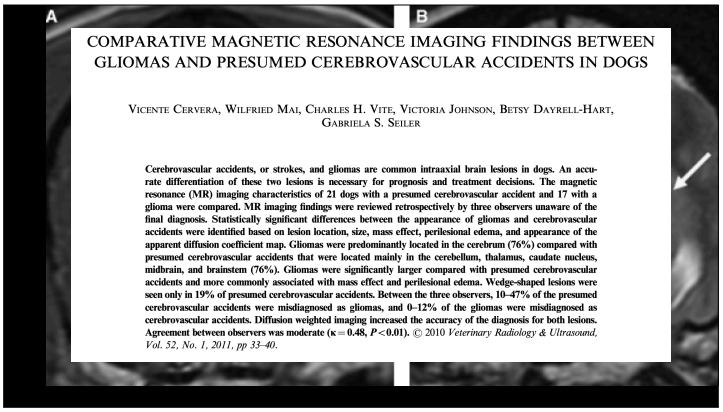
A RM NO ES NEUROLOGÍA!

B LESION EN RM = NO NECESARIAMENTE SIMTOMÁTICA

C **DIFERENCIAR ENFERMEDADES PUEDE SER DIFÍCIL…**

13

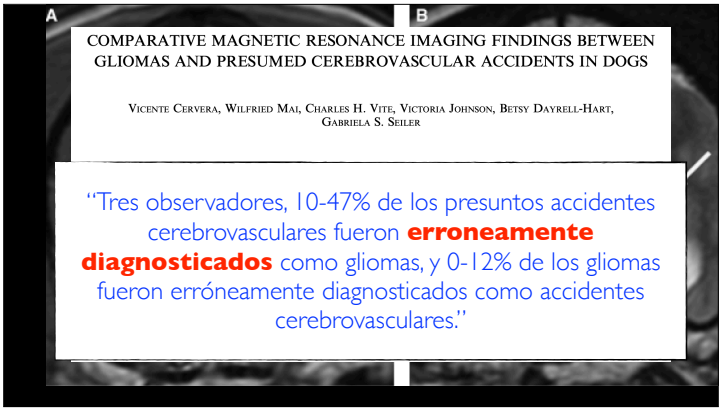




COMPARATIVE MAGNETIC RESONANCE IMAGING FINDINGS BETWEEN GLIOMAS AND PRESUMED CEREBROVASCULAR ACCIDENTS IN DOGS

Vicente Cervera, Wilfried Mai, Charles H. Vite, Victoria Johnson, Betsy Dayrell-Hart, Gabriela S. Seiler

Cerebrovascular accidents, or strokes, and gliomas are common intraaxial brain lesions in dogs. An accurate differentiation of these two lesions is necessary for prognosis and treatment decisions. The magnetic resonance (MR) imaging characteristics of 21 dogs with a presumed cerebrovascular accident and 17 with a glioma were compared. MR imaging findings were reviewed retrospectively by three observers unaware of the final diagnosis. Statistically significant differences between the appearance of gliomas and cerebrovascular accidents were identified based on lesion location, size, mass effect, perilesional edema, and appearance of the apparent diffusion coefficient map. Gliomas were predominantly located in the cerebrum (76%) compared with presumed cerebrovascular accidents that were located mainly in the cerebellum, thalamus, caudate nucleus, midbrain, and brainstem (76%). Gliomas were significantly larger compared with presumed cerebrovascular accidents and more commonly associated with mass effect and perilesional edema. Wedge-shaped lesions were seen only in 19% of presumed cerebrovascular accidents. Between the three observers, 10–47% of the presumed cerebrovascular accidents were misdiagnosed as gliomas, and 0–12% of the gliomas were misdiagnosed as cerebrovascular accidents. Diffusion weighted imaging increased the accuracy of the diagnosis for both lesions. Agreement between observers was moderate ($\kappa = 0.48$, $P < 0.01$). © 2010 *Veterinary Radiology & Ultrasound, Vol. 52, No. 1, 2011, pp 33–40.*

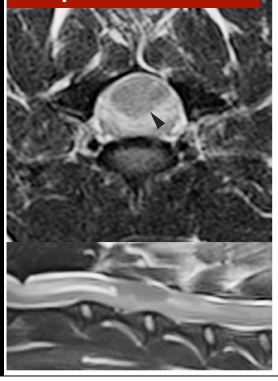


COMPARATIVE MAGNETIC RESONANCE IMAGING FINDINGS BETWEEN GLIOMAS AND PRESUMED CEREBROVASCULAR ACCIDENTS IN DOGS

Vicente Cervera, Wilfried Mai, Charles H. Vite, Victoria Johnson, Betsy Dayrell-Hart, Gabriela S. Seiler

"Tres observadores, 10-47% de los presuntos accidentes cerebrovasculares fueron **erroneamente diagnosticados** como gliomas, y 0-12% de los gliomas fueron erróneamente diagnosticados como accidentes cerebrovasculares."

Neoplasico? Inflammatorio?



Inflammatorio? Vascular?

Neoplasico? Inflammatorio?

Inflammatorio? Vascular?

Evaluation of magnetic resonance imaging for the differentiation of inflammatory, neoplastic, and vascular intradural spinal cord diseases in the dog

Amanda E. Masciarelli[1] | John F. Griffin IV[1] | Geoffrey T. Fosgate[2] | Silke Hecht[3] | Joseph M. Mankin[4] | Shannon P. Holmes[5] | Simon R. Platt[6] | Marc Kent[6] | Theresa E. Pancotto[7] | Annie V. Chen[8] | Jonathan M. Levine[2]

[1]Departments of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, TX, 77843

[2]The Department of Production Animal Studies, University of Pretoria, Pretoria, South Africa

[3]The Department of Small Animal Clinical Sciences, University of Tennessee College of Veterinary Medicine, Knoxville, TN, 37996

[4]Small Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, TX, 77843

[5]The Departments of Veterinary Biosciences and Diagnostic Imaging, University of Georgia College of Veterinary Medicine, Athens, GA, 30602

[6]Small Animal Medicine and Surgery, University of Georgia College of Veterinary Medicine, Athens, GA, 30602

[7]The Department of Small Animal Clinical Sciences, Virginia-Maryland College of Veterinary Medicine, Blacksburg, VA, 24061

[8]The Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Washington State University, Pullman, WA, 99164

Correspondence
Amanda E. Masciarelli, Departments of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77843.
Email: dr.anascicarelli@gmail.com

Abstract

Magnetic resonance imaging (MRI) is a common test for dogs with suspected intradural spinal cord lesions, however studies on diagnostic performance for this test are lacking. Objectives of this multi-institutional, retrospective, case-control study were to estimate sensitivity and specificity of MRI for: (1) distinguishing between histopathologically confirmed intradural spinal cord disease versus degenerative myelopathy in dogs; (2) categorizing intradural spinal cord diseases as neoplastic, inflammatory, or vascular; and (3) determining tumor type within the etiologic category of neoplasia. Additional aims were to (1) determine whether knowledge of clinical data affects sensitivity and specificity of MRI diagnoses; and (2) report interrater agreement for MRI classification of intradural spinal lesions. Cases were recruited from participating hospital databases over a 7-year period. Three reviewers independently evaluated each MRI study prior to and after provision of clinical information. A total of 87 cases were sampled (17 degenerative myelopathy, 53 neoplasia, nine inflammatory, and eight vascular). Magnetic resonance imaging had excellent (>97.6%) sensitivity for diagnosis of intradural spinal cord lesions but specificity varied before and after provision of clinical data (68.6% vs. 82.4%, P = 0.023). Magnetic resonance imaging had good sensitivity (86.8%) and moderate specificity (64.7–72.5%) for diagnosing neoplasia. Sensitivity was lower for classifying inflammatory lesions but improved with provision of clinical data (48.1% vs. 81.5%, P = 0.015). Magnetic resonance imaging was insensitive for diagnosing vascular lesions (25.0%). Interrater agreement was very good for correctly diagnosing dogs with intradural lesions (κ = 0.882–0.833), and good (κ = 0.726–0.671) for diagnosing dogs with neoplasia.

KEYWORDS
canine, inflammatory, MRI, neoplastic, spinal cord, vascular

Neoplasico? Inflammatorio?

Inflammatorio? Vascular?

Evaluation of magnetic resonance imaging for the differentiation of inflammatory, neoplastic, and vascular intradural spinal cord diseases in the dog

Amanda E. Masciarelli[1] | John F. Griffin IV[1] | Geoffrey T. Fosgate[2] | Silke Hecht[3] | Joseph M. Mankin[4] | Shannon P. Holmes[5] | Simon R. Platt[6] | Marc Kent[6] | Theresa E. Pancotto[7] | Annie V. Chen[8] | Jonathan M. Levine[2]

[1]Departments of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical

Abstract

"…la especificidad para lesiones intramedulares varió **entre disponer o no de los datos clínicos** (68.6% vs. 82.4%, P = 0.023)."

"La sensibilidad fue baja para clasificar lesiones inflamatorias pero **mejoró al disponer de los datos clínicos** (48.1% vs. 81.5%, P = 0.015)."

"RM resultó **insensible para el diagnóstico de lesiones vasculares** (25.0%)."

2
PODEMOS SOBREVIVIR SIN ELLA?

2
PODEMOS SOBREVIVIR SIN ELLA?
(puede que tengas que hacerlo...)

Realmente necesito una RM para este caso

Realmente necesito una RM para este caso

Pordría hacer una RM... pero puedo vivir sin ella

Realmente necesito una RM para este caso

Pordría hacer una RM... pero puedo vivir sin ella

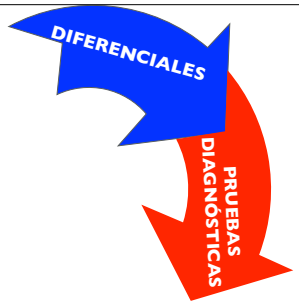
Podría hacer una RM pero sería tirar el dinero...

Realmente necesito una RM para este caso	Pordría hacer una RM... pero puedo vivir sin ella	Podría hacer una RM pero sería tirar el dinero...	No tengo ni idea de que tengo que hacer (con mi vida?)

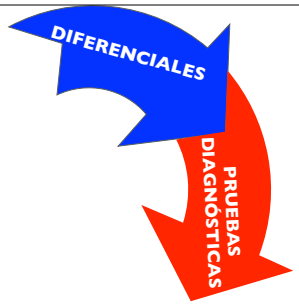
En realidad...

En realidad...

En realidad...

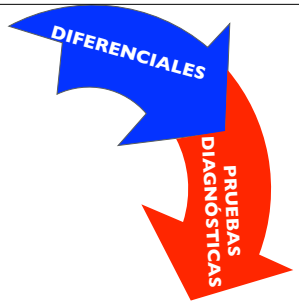


En realidad...



Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Ecogafía
Rx
TAC
RM
Cribado de ene
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

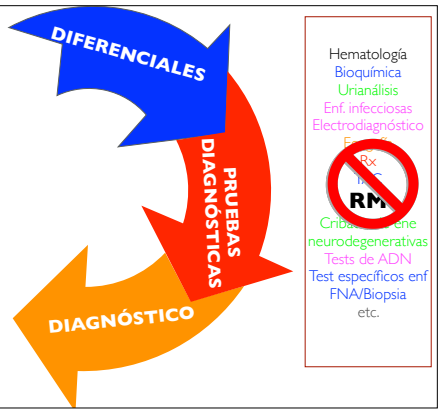
En realidad...



Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico

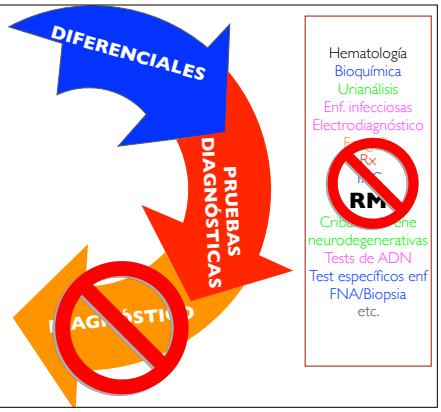
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...



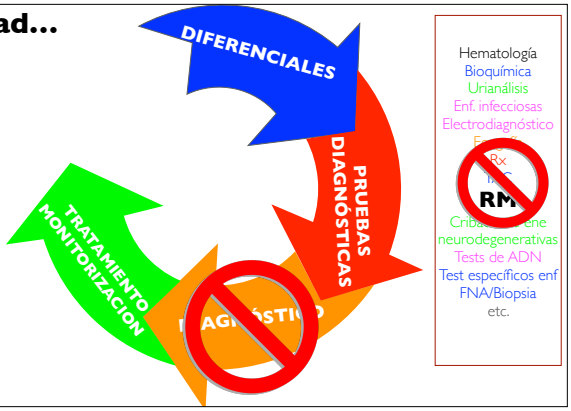
Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
RM
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...



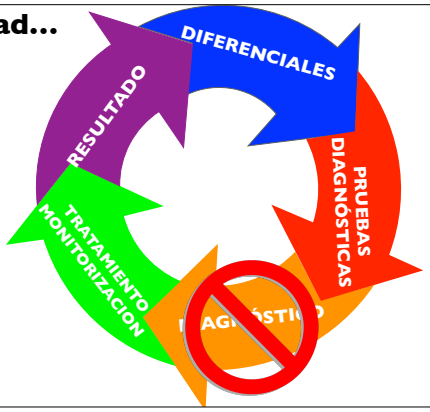
Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
RM
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...



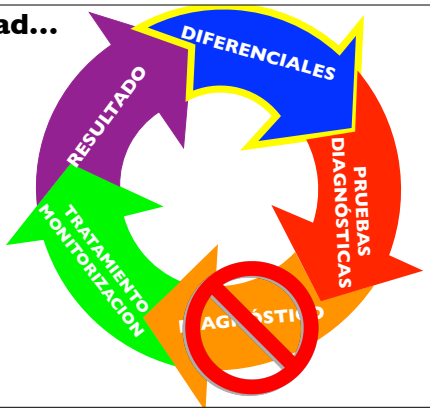
Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
RM
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...



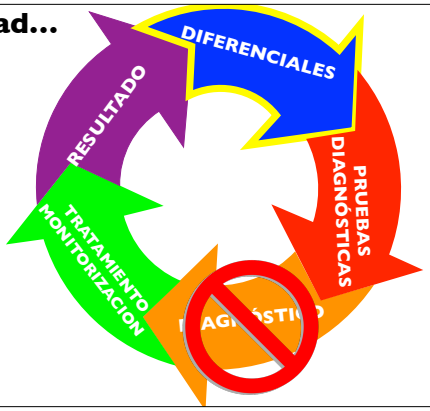
Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Rx
RM
Cribado de ene
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...



Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Rx
RM
Cribado de ene
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...

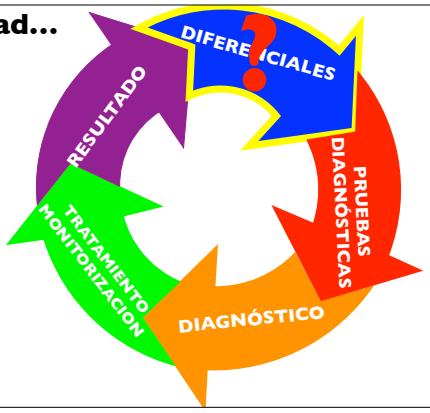


Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Ecogafía
Rx
TAC
RM
Cribado de ene
neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...

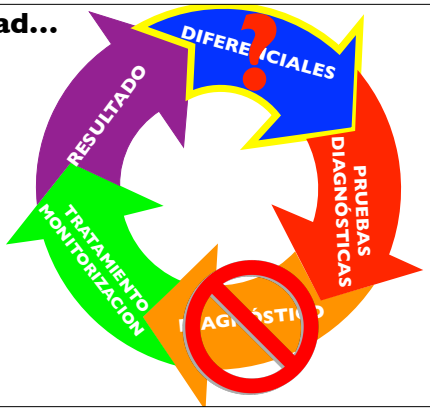
Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Ecogafía
Rx
TAC
RM
Cribado de ene neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

En realidad...

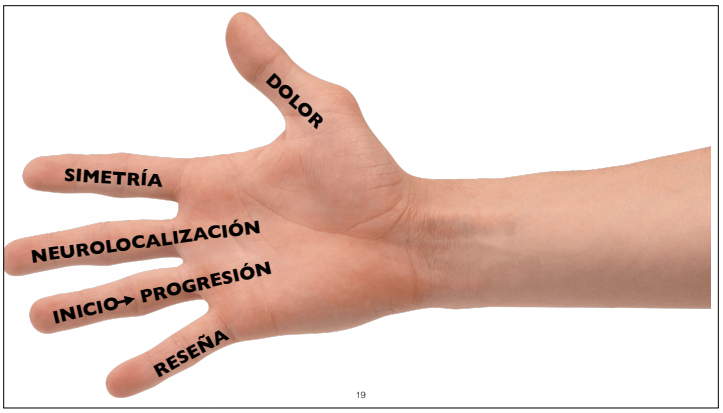


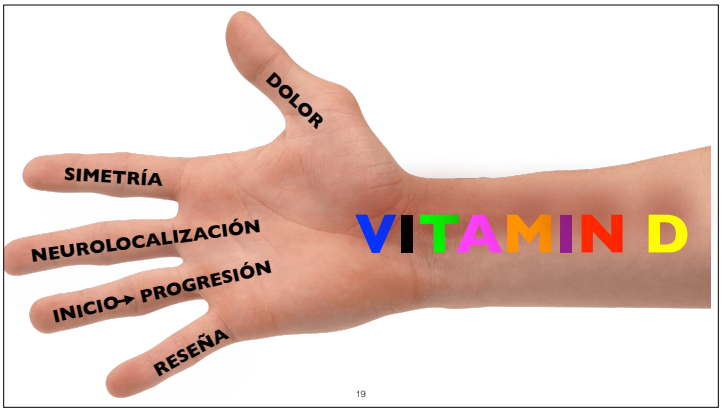
Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Ecogafía
Rx
TAC
RM
Cribado de ene neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.

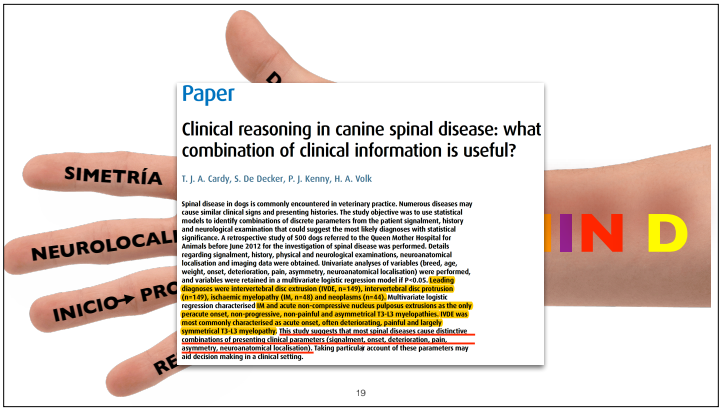
En realidad...



Hematología
Bioquímica
Urianálisis
Enf. infecciosas
Electrodiagnóstico
Ecogafía
Rx
TAC
RM
Cribado de ene neurodegenerativas
Tests de ADN
Test específicos enf
FNA/Biopsia
etc.







Paper

Clinical reasoning in canine spinal disease: what combination of clinical information is useful?

T. J. A. Cardy, S. De Decker, P. J. Kenny, H. A. Volk

Spinal disease in dogs is commonly encountered in veterinary practice. Numerous diseases may cause similar clinical signs and presenting histories. The study objective was to use statistical models to identify combinations of discrete parameters from the patient signalment, history and neurological examination that could suggest the most likely diagnoses with statistical significance. A retrospective study of 500 dogs referred to the Queen Mother Hospital for Animals before June 2012 for the investigation of spinal disease was performed. Details regarding signalment, history, physical and neurological examinations, neuroanatomical localisation and imaging data were obtained. Univariate analyses of variables (breed, age, weight, onset, deterioration, pain, asymmetry, neuroanatomical localisation) were performed, and variables were retained in a multivariate logistic regression model if $P<0.05$. Leading diagnoses were intervertebral disc extrusion (IVDE, n=149), intervertebral disc protrusion (n=149), ischaemic myelopathy (IM, n=48) and neoplasms (n=44). Multivariate logistic regression characterised IM and acute non-compressive nucleus pulposus extrusions as the only peracute onset, non-progressive, non-painful and asymmetrical T3-L3 myelopathies. IVDE was most commonly characterised as acute onset, often deteriorating, painful and largely symmetrical T3-L3 myelopathy. This study suggests that most spinal diseases cause distinctive combinations of presenting clinical parameters (signalment, onset, deterioration, pain, asymmetry, neuroanatomical localisation). Taking particular account of these parameters may aid decision making in a clinical setting.

Spinal disease in dogs is commonly encountered in veterinary practice. Numerous diseases may cause similar clinical signs and presenting histories. The study objective was to use statistical models to identify combinations of discrete parameters from the patient signalment, history and neurological examination that could suggest the most likely diagnoses with statistical significance. A retrospective study of 500 dogs referred to the Queen Mother Hospital for Animals before June 2012 for the investigation of spinal disease was performed. Details regarding signalment, history, physical and neurological examinations, neuroanatomical localisation and imaging data were obtained. Univariate analyses of variables (breed, age, weight, onset, deterioration, pain, asymmetry, neuroanatomical localisation) were performed, and variables were retained in a multivariate logistic regression model if $P<0.05$. Leading diagnoses were intervertebral disc extrusion (IVDE, n=149), intervertebral disc protrusion (n=149), ischaemic myelopathy (IM, n=48) and neoplasms (n=44). Multivariate logistic regression characterised IM and acute non-compressive nucleus pulposus extrusions as the only peracute onset, non-progressive, non-painful and asymmetrical T3-L3 myelopathies. IVDE was most commonly characterised as acute onset, often deteriorating, painful and largely symmetrical T3-L3 myelopathy. This study suggests that most spinal diseases cause distinctive combinations of presenting clinical parameters (signalment, onset, deterioration, pain, asymmetry, neuroanatomical localisation). Taking particular account of these parameters may aid decision making in a clinical setting.

Reseña

Reseña

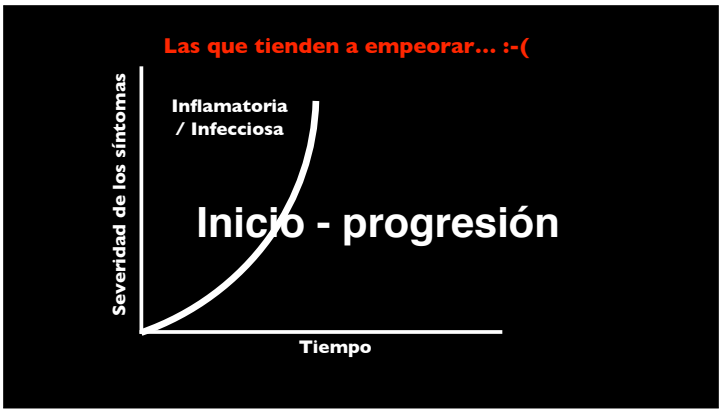
Reseña

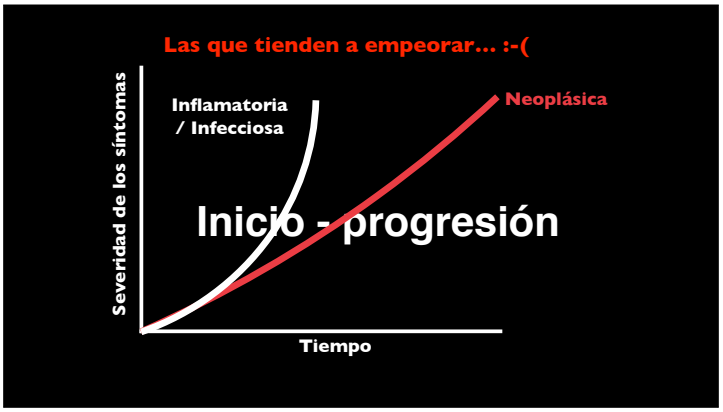
INICIO - PROGRESIÓN

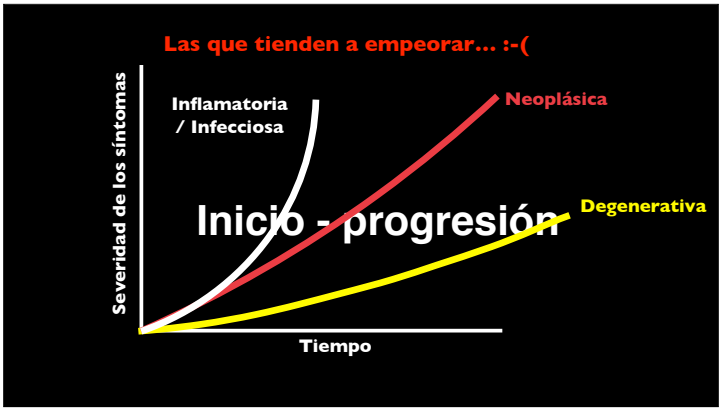
Severidad de los síntomas
VITAMIN D
Inicio - progresión
Tiempo

Severidad de los síntomas
Inicio - progresión
Tiempo

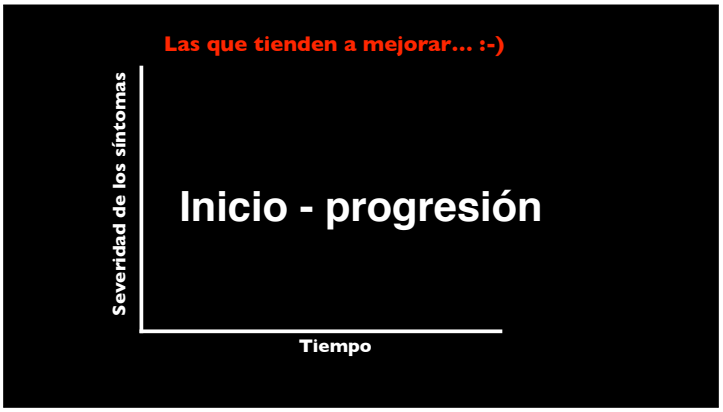
Las que tienden a empeorar... :-(
Severidad de los síntomas
Inicio - progresión
Tiempo

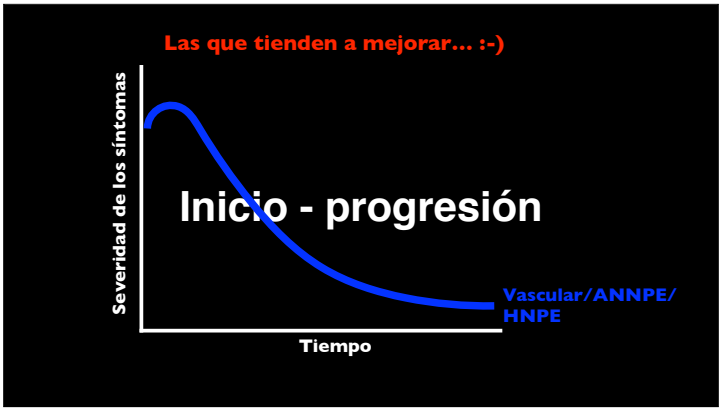

Las que tienden a empeorar... :-(
Severidad de los síntomas
Inflamatoria
/ Infecciosa
Inicio - progresión
Tiempo

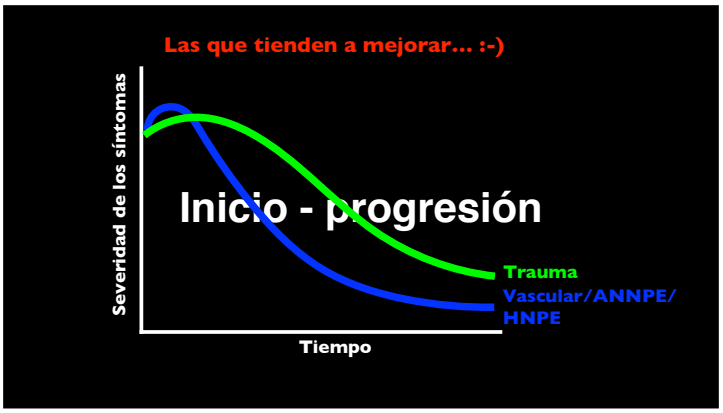

Las que tienden a empeorar... :-(
Severidad de los síntomas
Inflamatoria
/ Infecciosa
Neoplásica
Inicio - progresión
Tiempo

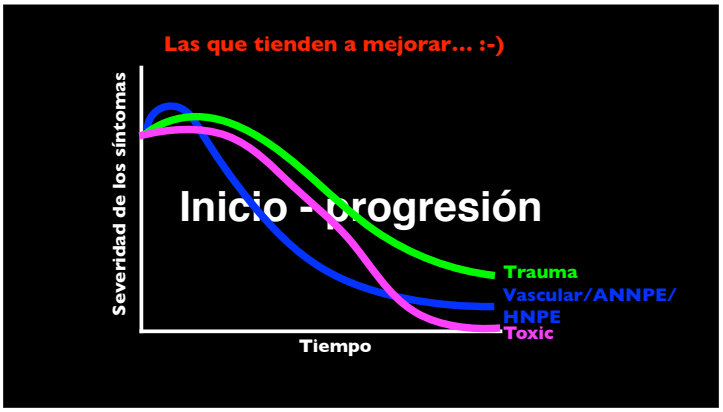

Las que tienden a empeorar... :-(
Severidad de los síntomas
Inflamatoria
/ Infecciosa
Neoplásica
Degenerativa
Inicio - progresión
Tiempo

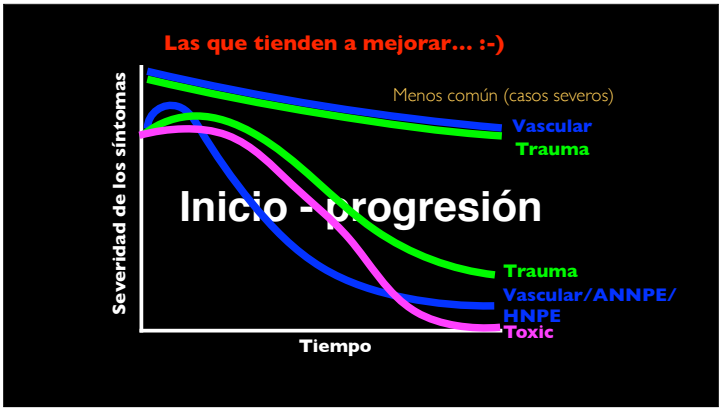
Severidad de los síntomas
Inicio - progresión
Tiempo

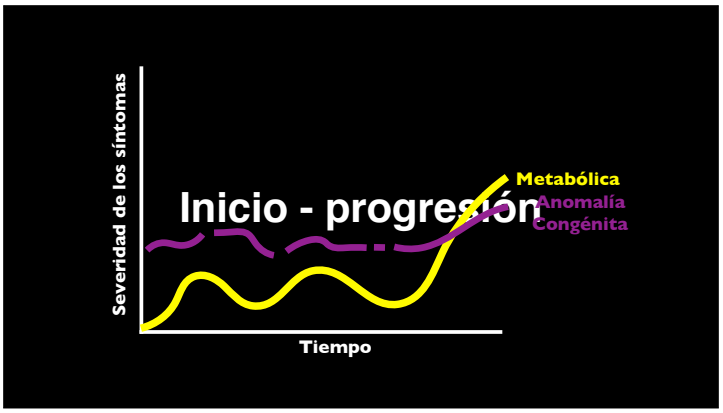

Las que tienden a mejorar... :-)
Severidad de los síntomas
Inicio - progresión
Tiempo

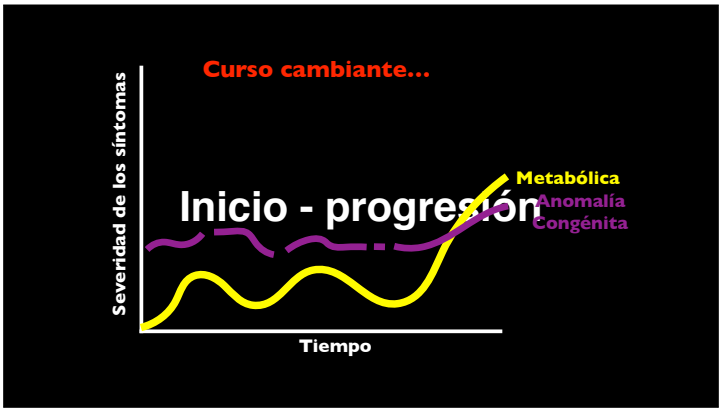

Las que tienden a mejorar... :-)
Severidad de los síntomas
Inicio - progresión
Vascular/ANNPE/
HNPE
Tiempo


Las que tienden a mejorar... :-)
Severidad de los síntomas
Inicio - progresión
Trauma
Vascular/ANNPE/
HNPE
Tiempo


Las que tienden a mejorar... :-)
Severidad de los síntomas
Inicio - progresión
Trauma
Vascular/ANNPE/
HNPE
Toxic
Tiempo


Las que tienden a mejorar... :-)
Severidad de los síntomas
Menos común (casos severos)
Vascular
Trauma
Inicio - progresión
Trauma
Vascular/ANNPE/
HNPE
Toxic
Tiempo





FOCAL	MULTIFOCAL	GENERALIZADA difusa
Vascular	**Inflammatorio/Infeccioso**	**Intoxicación**
Neoplasia	Neurodegenerativa	**Metabolica**
Degenerativa (IVDD)	Trauma	Neurodegenerativa
Trauma	Neoplasia	Inflammatoria/Infecciosa
Inflammatorio/Infeccioso	Anomalía	
Anomalía	Vascular	
	Degenerativa (IVDD)	

VITAMIN D

Neurolocalización

FOCAL	MULTIFOCAL	GENERALIZADA difusa
Vascular	**Inflammatorio/Infeccioso**	**Intoxicación**
Neoplasia	Neurodegenerativa	**Metabolica**
Degenerativa (IVDD)	Trauma	Neurodegenerativa
Trauma	Neoplasia	Inflammatoria/Infecciosa
Inflammatorio/Infeccioso	Anomalía	
Anomalía	Vascular	
	Degenerativa (IVDD)	

VITAMIN D

Simetría

Simetrica	Asimetrica
Intoxicación	**Vascular**
Metabolica	Neoplasia
Neurodegenerativa	
Anomalía	
Inflammatoria/Infecciosa	
Trauma	

VITAMIN D

Piel

Tejido subcutáneo

Grasa

Tendones músculos

Ligamentos

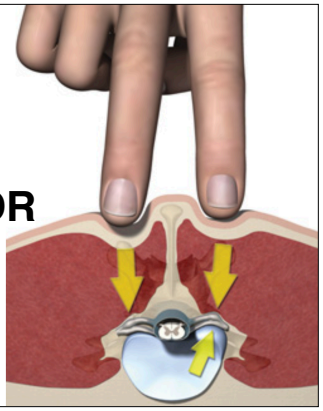
Huesos

Articulaciones

Disco

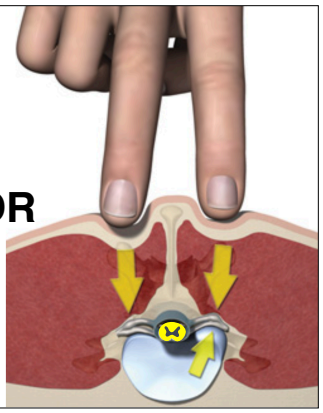
Nervios espinales | **Meninges**

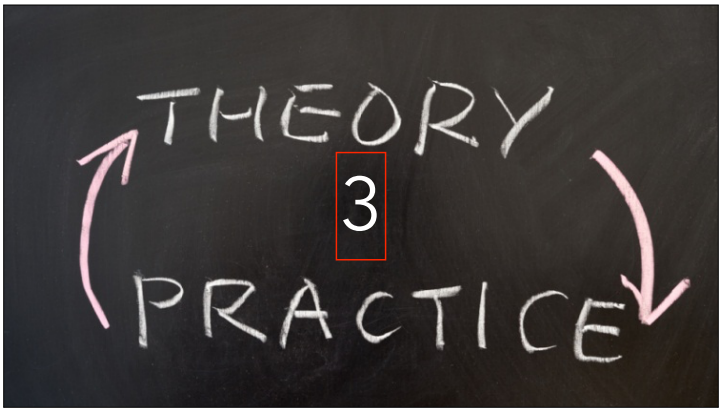
DOLOR



Piel
Tejido subcutáneo
Grasa
Tendones músculos
Ligamentos
Huesos
Articulaciones
Disco
Nervios espinales / Meninges

DOLOR





Los que no deberíamos meter en la máquina…

A

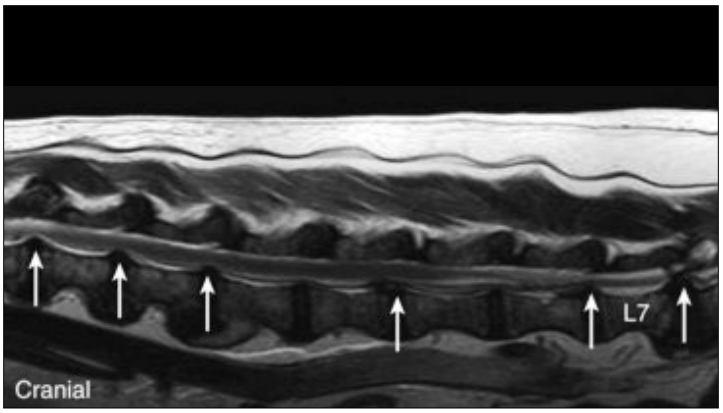
Los que no deberíamos meter en la máquina…

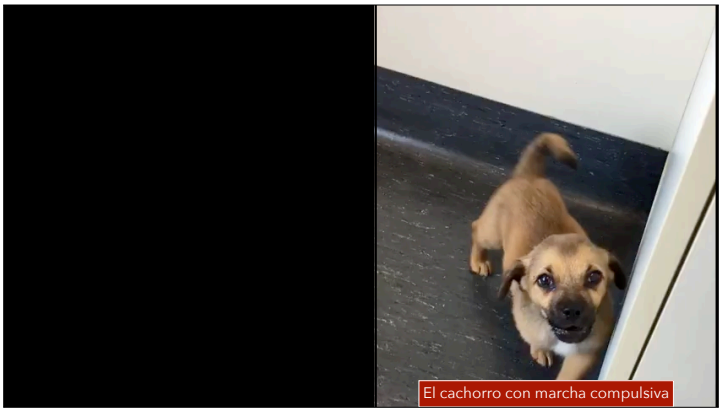
A

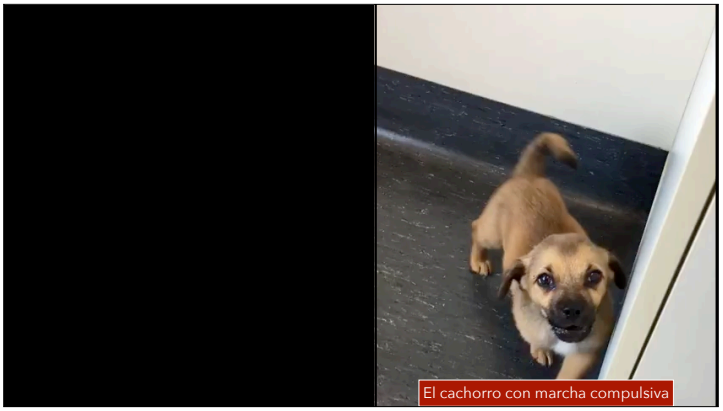
El Pastor Aleman débil de los MMPP…

NEUROMUSCULAR
El Pastor Aleman débil de los MMPP…

NEUROMUSCULAR
El Pastor Aleman débil de los MMPP…


L7
Cranial

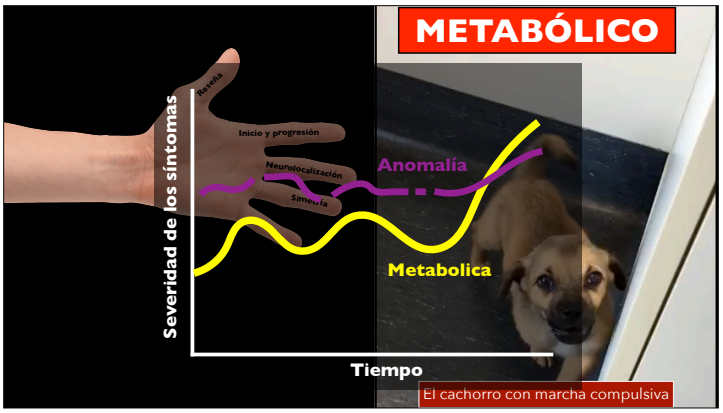

El cachorro con marcha compulsiva

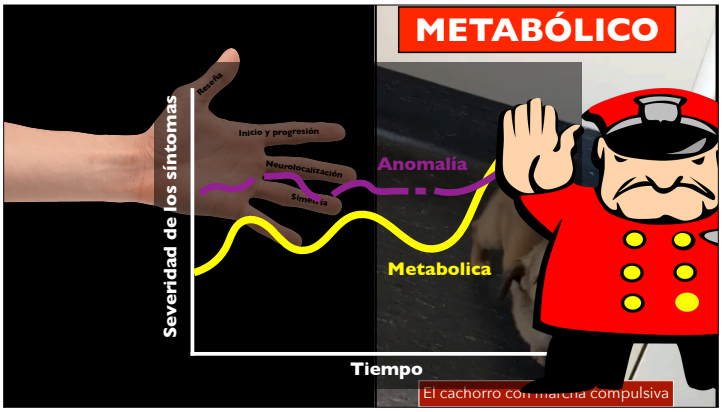

El cachorro con marcha compulsiva

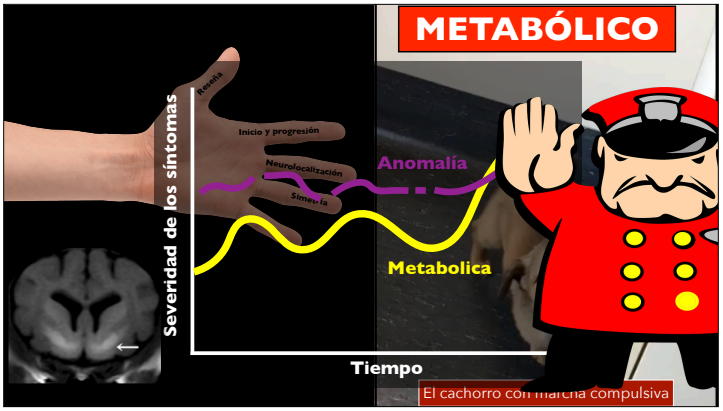
Reseña
Inicio y progresión
Neurolocalización
Simetría
Dolor
El cachorro con marcha compulsiva

METABÓLICO
Reseña
Inicio y progresión
Neurolocalización
Simetría
Dolor
El cachorro con marcha compulsiva

METABÓLICO
Severidad de los síntomas
Reseña
Inicio y progresión
Neurolocalización
Simetría
Dolor
Tiempo
El cachorro con marcha compulsiva


METABÓLICO
Reseña
Inicio y progresión
Neurolocalización
Síntesis
Anomalía
Metabolica
Severidad de los síntomas
Tiempo
El cachorro con marcha compulsiva


METABÓLICO
Reseña
Inicio y progresión
Neurolocalización
Síntesis
Anomalía
Metabolica
Severidad de los síntomas
Tiempo
El cachorro con marcha compulsiva


METABÓLICO
Reseña
Inicio y progresión
Neurolocalización
Síntesis
Anomalía
Metabolica
Severidad de los síntomas
Tiempo
El cachorro con marcha compulsiva

El gato con ventroflexión

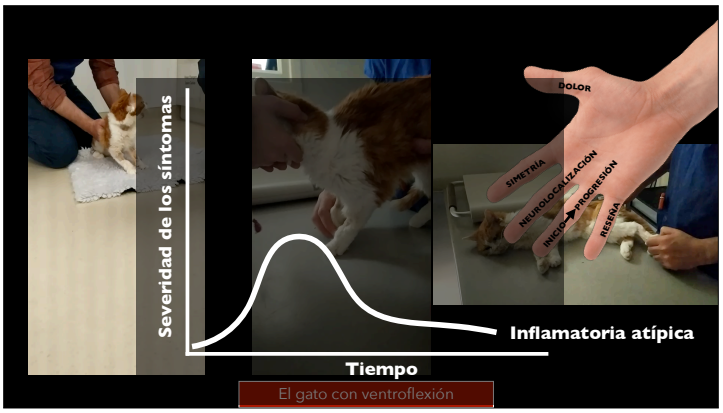
El gato con ventroflexión

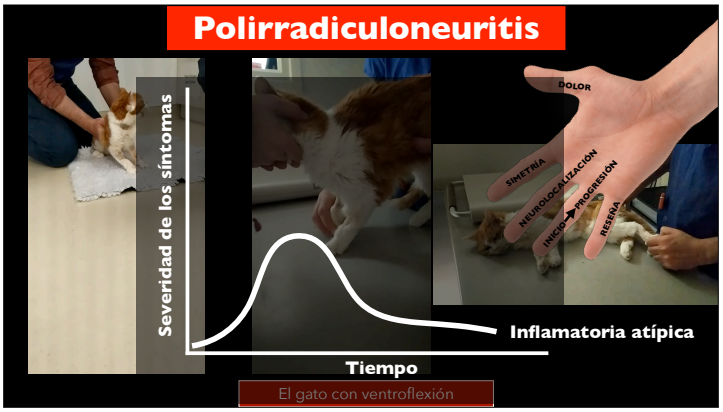
El gato con ventroflexión

El gato con ventroflexión

DOLOR
SIMETRÍA
NEUROLOCALIZACIÓN
INICIO
PROGRESIÓN
RESEÑA
El gato con ventroflexión

Severidad de los síntomas
Tiempo
DOLOR
SIMETRÍA
NEUROLOCALIZACIÓN
INICIO
PROGRESIÓN
RESEÑA
El gato con ventroflexión


Severidad de los síntomas
Tiempo
Inflamatoria atípica
DOLOR
SIMETRÍA
NEUROLOCALIZACIÓN
INICIO→PROGRESIÓN
RESEÑA
El gato con ventroflexión


Polirradiculoneuritis
Severidad de los síntomas
Tiempo
Inflamatoria atípica
DOLOR
SIMETRÍA
NEUROLOCALIZACIÓN
INICIO→PROGRESIÓN
RESEÑA
El gato con ventroflexión

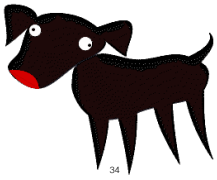
Polirradiculoneuritis
Severidad de los síntomas
Tiempo
Inflamatoria atípica
DOLOR
El gato con ventroflexión

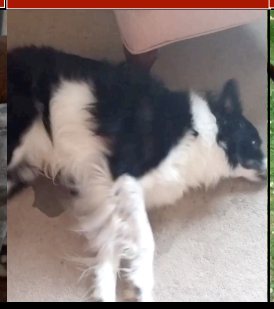
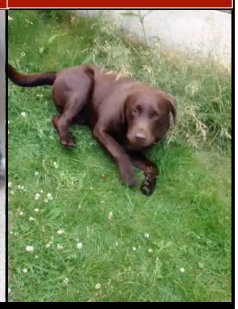
B

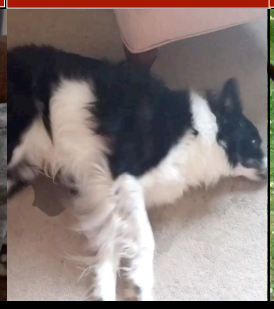
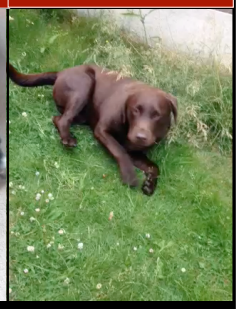
Algunos casos de síntomas episódicos…

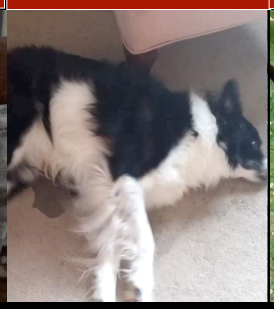
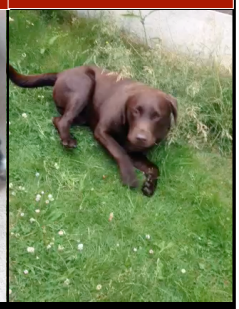
B

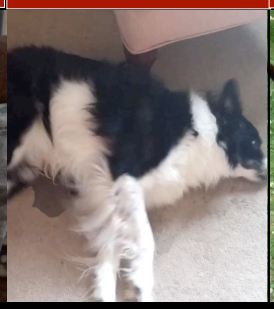
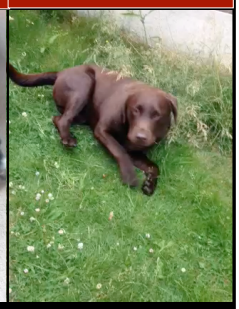
Algunos casos de síntomas episódicos…



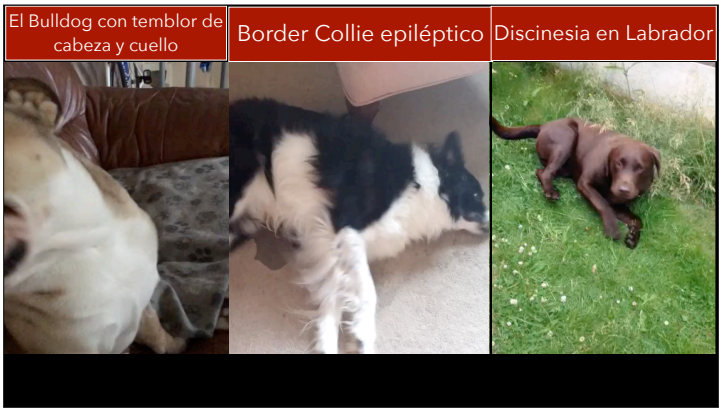
El Bulldog con temblor de cabeza y cuello	Border Collie epiléptico	Discinesia en Labrador
		

El Bulldog con temblor de cabeza y cuello	Border Collie epiléptico	Discinesia en Labrador
		

El Bulldog con temblor de cabeza y cuello	Border Collie epiléptico	Discinesia en Labrador
		

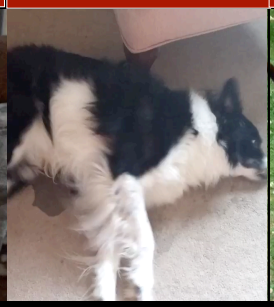
El Bulldog con temblor de cabeza y cuello	Border Collie epiléptico	Discinesia en Labrador
		

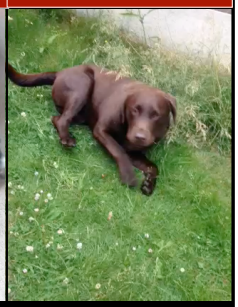
El Bulldog con temblor de cabeza y cuello
Border Collie epiléptico
Discinesia en Labrador
Como sabemos de qué se trata?


El Bulldog con temblor de cabeza y cuello
Border Collie epiléptico
Discinesia en Labrador

El Bulldog con temblor de cabeza y cuello
Border Collie epiléptico
Discinesia en Labrador
Que hay de las enfermedades estructurales cerebrales?

El Bulldog con temblor de cabeza y cuello	Border Collie epiléptico	Discinesia en Labrador





CORRESPONDENCE Open Access

International veterinary epilepsy task force consensus proposal: diagnostic approach to epilepsy in dogs

Luisa De Risio[1*], Sofie Bhatti[2], Karen Muñana[3], Jacques Penderis[4], Veronika Stein[5], Andrea Tipold[6], Mette Berendt[8], Robyn Farqhuar[9], Andrea Fischer[8], Sam Long[10], Paul J.J. Mandigers[11], Kaspar Matiasek[12], Rowena MA Packer[13], Akos Pakozdy[15], Ned Patterson[14], Simon Platt[15], Michael Podell[16], Heidrun Potschka[17], Marti Pumarola Batlle[18], Clare Rusbridge[19,20] and Holger A. Volk[21]

Abstract

This article outlines the consensus proposal on diagnosis of epilepsy in dogs by the International Veterinary Epilepsy Task Force. The aim of this consensus proposal is to improve consistency in the diagnosis of epilepsy in the clinical and research settings. The diagnostic approach to the patient presenting with a history of suspected epileptic seizures incorporates two fundamental steps: to establish if the events the animal is demonstrating truly represent epileptic seizures and if so, to identify their underlying cause. Differentiation of epileptic seizures from other non-epileptic episodic paroxysmal events can be challenging. Criteria that can be used to make this differentiation are presented in detail and discussed. Criteria for the diagnosis of idiopathic epilepsy (IE) are described in a three-tier system. Tier I confidence level for the diagnosis of IE is based on a history of two or more unprovoked epileptic seizures occurring at least 24 h apart, age at epileptic seizure onset of between six months and six years, unremarkable inter-ictal physical and neurological examination, and no significant abnormalities on minimum data base blood tests and urinalysis. Tier II confidence level for the diagnosis of IE is based on the factors listed in tier I and unremarkable fasting and post-prandial bile acids, magnetic resonance imaging (MRI) of the brain (based on an epilepsy-specific brain MRI protocol) and cerebrospinal fluid (CSF) analysis. Tier III confidence level for the diagnosis of IE is based on the factors listed in tier I and II and identification of electroencephalographic abnormalities characteristic for seizure disorders. The authors recommend performing MRI of the brain and routine CSF analysis, after exclusion of reactive seizures, in dogs with age at epileptic seizure onset <6 months or >6 years, inter-ictal neurological abnormalities consistent with intracranial neurolocalisation, status epilepticus or cluster seizure at epileptic seizure onset, or a previous presumptive diagnosis of IE and drug-resistance with a single antiepileptic drug titrated to the highest tolerable dose.

This consensus article represents the basis for a more standardised diagnostic approach to the seizure patient. These recommendations will evolve over time with advances in neuroimaging, electroencephalography, and molecular genetics of canine epilepsy.

Keywords: Dog, Seizure, Epilepsy, Idiopathic epilepsy, Diagnosis

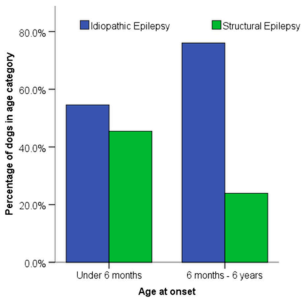


Figure 1 Proportion of dogs with idiopathic and structural epilepsy stratified by age at epileptic seizure onset (< 6 months versus 6 months to 6 years)

Epilepsy Task Force. The aim of this consensus proposal is to improve consistency in the diagnosis of epilepsy in the clinical and research settings. The diagnostic approach to the patient presenting with a history of suspected epileptic seizures incorporates two fundamental steps: to establish if the events the animal is demonstrating truly represent epileptic seizures and if so, to identify their underlying cause. Differentiation of epileptic seizures from other non-epileptic episodic paroxysmal events can be challenging. Criteria that can be used to make this differentiation are presented in detail and discussed. Criteria for the diagnosis of idiopathic epilepsy (IE) are described in a three-tier system. Tier I confidence level for the diagnosis of IE is based on a history of two or more unprovoked epileptic seizures occurring at least 24 h apart, age at epileptic seizure onset of between six months and six years, unremarkable inter-ictal physical and neurological examination, and no significant abnormalities on minimum data base blood tests and urinalysis. Tier II confidence level for the diagnosis of IE is based on the factors listed in tier I and unremarkable fasting and post-prandial bile acids, magnetic resonance imaging (MRI) of the brain (based on an epilepsy-specific brain MRI protocol) and cerebrospinal fluid (CSF) analysis. Tier III confidence level for the diagnosis of IE is based on the factors listed in tier I and II and identification of electroencephalographic abnormalities characteristic for seizure disorders. The authors recommend performing MRI of the brain and routine CSF analysis, after exclusion of reactive seizures, in dogs with age at epileptic seizure onset <6 months or >6 years, inter-ictal neurological abnormalities consistent with intracranial neurolocalisation, status epilepticus or cluster seizure at epileptic seizure onset, or a previous presumptive diagnosis of IE and drug-resistance with a single antiepileptic drug titrated to the highest tolerable dose.
This consensus article represents the basis for a more standardised diagnostic approach to the seizure patient. These recommendations will evolve over time with advances in neuroimaging, electroencephalography, and molecular genetics of canine epilepsy.

Keywords: Dog, Seizure, Epilepsy, Idiopathic epilepsy, Diagnosis

CORRESPONDENCE Open Access

International veterinary epilepsy task force consensus proposal: diagnostic approach to epilepsy in dogs

Luisa De Risio[1*], Sofie Bhatti[2], Karen Muñana[3], Jacques Penderis[4], Veronika Stein[5], Andrea Tipold[6], Mette Berendt[8], Robyn Fanghua[9], Andrea Fischer[8], Sam Long[10], Paul JJ. Mandigers[11], Kaspar Matiasek[12], Rowena MA Packer[13], Akos Pakozdy[14], Ned Patterson[14], Simon Platt[15], Michael Podell[16], Heidrun Potschka[17], Marti Pumarola Batlle[18], Clare Rusbridge[19,20] and Holger A. Volk[21]

Abstract

This article outlines the consensus proposal on diagnosis of epilepsy in dogs by the International Veterinary Epilepsy Task Force. The aim of this consensus proposal is to improve consistency in the diagnosis of epilepsy in the clinical and research settings. The diagnostic approach to the patient presenting with a history of suspected epileptic seizures incorporates two fundamental steps: to establish if the events the animal is demonstrating truly represent epileptic seizures and if so, to identify their underlying cause. Differentiation of epileptic seizures from other non-epileptic episodic paroxysmal events can be challenging. Criteria that can be used to make this differentiation are presented in detail and discussed. Criteria for the diagnosis of idiopathic epilepsy (IE) are described in a three-tier system. Tier I confidence level for the diagnosis of IE is based on a history of two or more unprovoked epileptic seizures occurring at least 24 h apart, age at epileptic seizure onset of between six months and six years, unremarkable inter-ictal physical and neurological examination, and no significant abnormalities on minimum data base blood tests and urinalysis. Tier II confidence level for the diagnosis of IE is based on the factors listed in tier I and unremarkable fasting and post-prandial bile acids, magnetic resonance imaging (MRI) of the brain (based on an epilepsy-specific brain MRI protocol) and cerebrospinal fluid (CSF) analysis. Tier III confidence level for the diagnosis of IE is based on the factors listed in tier I and II and identification of electroencephalographic abnormalities characteristic for seizure disorders. The authors recommend performing MRI of the brain and routine CSF analysis, after exclusion of reactive seizures, in dogs with age at epileptic seizure onset <6 months or >6 years, inter-ictal neurological abnormalities consistent with intracranial neurolocalisation, status epilepticus or cluster seizure at epileptic seizure onset, or a previous presumptive diagnosis of IE and drug resistance with a single antiepileptic drug trialed to the highest tolerated dose.

This consensus article represents the basis for a more standardised diagnostic approach to the seizure patient. These recommendations will evolve over time with advances in neuroimaging, electroencephalography, and molecular genetics of canine epilepsy.

Keywords: Dog, Seizure, Epilepsy, Idiopathic epilepsy, Diagnosis

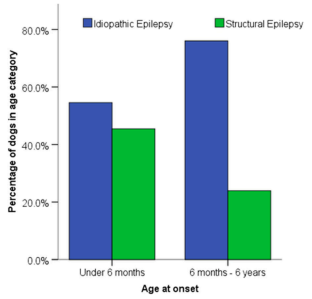


Figure 1 Proportion of dogs with idiopathic and structural epilepsy stratified by age at epileptic seizure onset (< 6 months versus 6 months to 6 years)

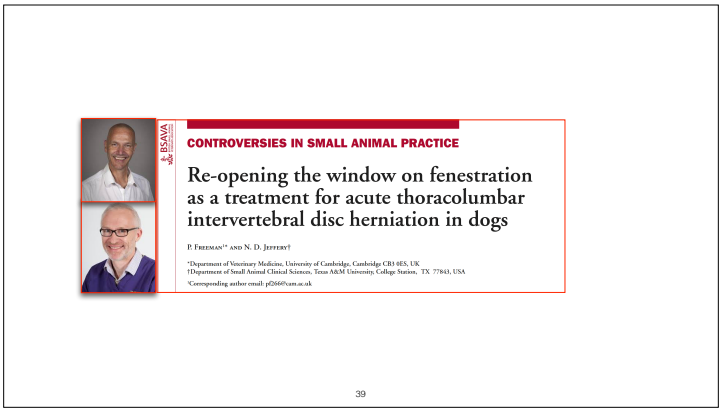
C **Otra columna...**

C **Otra columna...**

Teckel 4 años
Dolor lumbar que progresa a paraplejía aguda
Mielopatía T3-L3
Simetrico
Dolor leve
El salchicha parapléjico

Teckel 4 años
Dolor lumbar que progresa a paraplejía aguda
Mielopatía T3-L3
Simetrico
Dolor leve
El salchicha parapléjico

Teckel 4 años
Dolor lumbar que progresa a paraplejía aguda
Mielopatía T3-L3
Simetrico
Dolor leve
Reseña
Inicio y progresión
Neurolocalización
Simetría
Dolor
El salchicha parapléjico



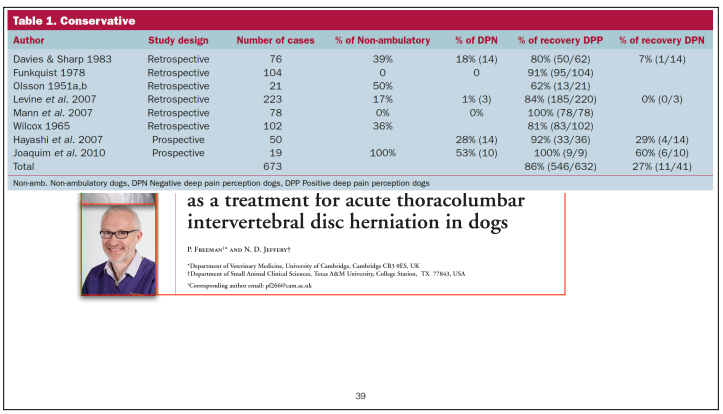


Table 1. Conservative

Author	Study design	Number of cases	% of Non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Davies & Sharp 1983	Retrospective	76	39%	18% (14)	80% (50/62)	7% (1/14)
Funkquist 1978	Retrospective	104	0	0	91% (95/104)	
Olsson 1951a,b	Retrospective	21	50%		62% (13/21)	
Levine *et al.* 2007	Retrospective	223	17%	1% (3)	84% (185/220)	0% (0/3)
Mann *et al.* 2007	Retrospective	78	0%	0%	100% (78/78)	
Wilcox 1965	Retrospective	102	36%		81% (83/102)	
Hayashi *et al.* 2007	Prospective	50		28% (14)	92% (33/36)	29% (4/14)
Joaquim *et al.* 2010	Prospective	19	100%	53% (10)	100% (9/9)	60% (6/10)
Total		673			86% (546/632)	27% (11/41)

Non-amb. Non-ambulatory dogs, DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Table 1. Conservative

Author	Study design	Number of cases	% of Non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Davies & Sharp 1983	Retrospective	76	39%	18% (14)	80% (50/62)	7% (1/14)
Funkquist 1978	Retrospective	104	0	0	91% (95/104)	
Olsson 1951a,b	Retrospective	21	50%		62% (13/21)	
Levine *et al.* 2007	Retrospective	223	17%	1% (3)	84% (185/220)	0% (0/3)
Mann *et al.* 2007	Retrospective	78	0%	0%	100% (78/78)	
Wilcox 1965	Retrospective	102	36%		81% (83/102)	
Hayashi *et al.* 2007	Prospective	50		28% (14)	92% (33/36)	29% (4/14)
Joaquim *et al.* 2010	Prospective	19	100%	53% (10)	100% (9/9)	60% (6/10)
Total		673			86% (546/632)	27% (11/41)

Non-amb. Non-ambulatory dogs, DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Table 2. Nonlaminectomy

Author	Study design	Number of cases	% of non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Brown et al. 1977	Retrospective	167	76%	17% (32)	63%	67% (15/32)
Loughin et al. 2005	Retrospective	48	100%	100%		73% (35/48)
Tarlowski et al. 2009	Retrospective	23	100%	13% (3	91%	33% (1/3)
Dosch et al. 2007	Prospective	51	100%	0%	80%	
Davis & Brown 2002	Retrospective	112	100%	0%	90%	
Hazlehn et al. 2005	Retrospective	30	93%	27%	86%	50% (4/8)
Mehta 1982	Retrospective	38	100%	7% (3)	100%	0% (0/3)
Scott 1987	Retrospective	40	78%	8% (3)	80%	66% (2/3)
Fenoies et al. 2002	Retrospective	71	100%	0%	86%	
Abana et al. 2012a	Retrospective	855	69.50%	25% (211)	90%	52% (133/211)
Ruddle et al. 2006	Retrospective	250	100%	10% (25)	92%	72% (17/25)
Olby et al. 2003	Retrospective	64	100%	103% (66)		58% (37/64)
Mar et al. 1995	Retrospective	44	95%	23% (11)	83%	30% (5/11)
Lubbers & Puerto 2005	Retrospective	48	100%	100% (46)		41% (19/46)
Obeck 1998	Retrospective	28	68%	3% (1)	100%	100% (1)
Schulman & Lipscott 1987	Retrospective	91	94%		90%	
Davis 1994	Retrospective	34	100%	38% (29)	93%	62% (18/29)
Scott & McKee 1999	Retrospective	24	100%	100%		62% (20/34)
Knecht 1972	Retrospective	09	100%		77%	
Jeffery et al. 2018	Prospective	178	100%	100%		58% (45/78)
Total		2279			90% (1486/1650)	56% (335/600)

Nonamb. Nonambulatory dogs,DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

t for acute thoracolumbar
disc herniation in dogs

sity of Cambridge, Cambridge CB1 0ES, UK
ces, Texas A&M University, College Station, TX 77843, USA
ak

Table 1. Conservative

Author	Study design	Number of cases	% of Non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Davies & Sharp 1983	Retrospective	76	39%	18% (14)	80% (50/62)	7% (1/14)
Funkquist 1978	Retrospective	104	0	0	91% (95/104)	
Olsson 1951a,b	Retrospective	21	50%		62% (13/21)	
Levine *et al.* 2007	Retrospective	223	17%	1% (3)	84% (185/220)	0% (0/3)
Mann *et al.* 2007	Retrospective	78	0%	0%	100% (78/78)	
Wilcox 1965	Retrospective	102	36%		81% (83/102)	
Hayashi *et al.* 2007	Prospective	50		28% (14)	92% (33/36)	29% (4/14)
Joaquim *et al.* 2010	Prospective	19	100%	53% (10)	100% (9/9)	60% (6/10)
Total		673			86% (546/632)	27% (11/41)

Non-amb. Non-ambulatory dogs, DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Table 2. Nonlaminectomy

Author	Study design	Number of cases	% of non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Brown et al. 1977	Retrospective	167	76%	17% (32)	63%	67% (15/32)
Loughin et al. 2005	Retrospective	48	100%	100%		73% (35/48)
Tarlowski et al. 2009	Retrospective	23	100%	13% (3	91%	33% (1/3)
Dosch et al. 2007	Prospective	51	100%	0%	80%	
Davis & Brown 2002	Retrospective	112	100%	0%	90%	
Hazlehn et al. 2005	Retrospective	30	93%	27%	86%	50% (4/8)
Mehta 1982	Retrospective	38	100%	7% (3)	100%	0% (0/3)
Scott 1987	Retrospective	40	78%	8% (3)	80%	66% (2/3)
Fenoies et al. 2002	Retrospective	71	100%	0%	86%	
Abana et al. 2012a	Retrospective	855	69.50%	25% (211)	90%	52% (133/211)
Ruddle et al. 2006	Retrospective	250	100%	10% (25)	92%	72% (17/25)
Olby et al. 2003	Retrospective	64	100%	103% (66)		58% (37/64)
Mar et al. 1995	Retrospective	44	95%	23% (11)	83%	30% (5/11)
Lubbers & Puerto 2005	Retrospective	48	100%	100% (46)		41% (19/46)
Obeck 1998	Retrospective	28	68%	3% (1)	100%	100% (1)
Schulman & Lipscott 1987	Retrospective	91	94%		90%	
Davis 1994	Retrospective	34	100%	38% (29)	93%	62% (18/29)
Scott & McKee 1999	Retrospective	24	100%	100%		62% (20/34)
Knecht 1972	Retrospective	09	100%		77%	
Jeffery et al. 2018	Prospective	178	100%	100%		58% (45/78)
Total		2279			90% (1486/1650)	56% (335/600)

Nonamb. Nonambulatory dogs,DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Table 3. Fenestration

Author	Study Design	No. Cases	% Non-amb.	% DPN	% Recovery DPP	% Recovery DPN
Padilho Filho & Selmi 1999	Retrospective	29	45%	17% (5)	100%	80% (4/5)
Burzykowski 2008	Prospective	26			88%	
Olsson 1951a,b	Prospective	28			89%	
Nakama et al. 1993	Retrospective	66	74%	39% (26)	85%	65% (17/26)
Flo 1975	Retrospective	67		17% (12)	95%	75% (9/12)
Ranzel et al. 2005	Retrospective	331		46% (152)	89%	38% (58/152)
Knapp et al. 1990	Retrospective	160	30%	0-6 (18	98%	
Denny 1978	Retrospective	30	80%	30% (9)	100%	67% (6/9)
Butterworth & Denny 1991	Retrospective	100	44%	6%	90%	33% (2/6)
Funkquist 1978	Retrospective	80		0	99%	
Davies & Sharp 1983	Retrospective	34	47%	0	91%	
Total		951			94% (694/742)	45% (95/209)

Nonamb. Nonambulatory dogs, DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Table 1. Conservative

Author	Study design	Number of cases	% of Non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Davies & Sharp 1983	Retrospective	76	39%	18% (14)	80% (50/62)	7% (1/14)
Funkquist 1978	Retrospective	104	0	0	91% (95/104)	
Olsson 1951a,b	Retrospective	21	50%		62% (13/21)	
Levine *et al.* 2007	Retrospective	223	17%	1% (3)	84% (185/220)	0% (0/3)
Mann *et al.* 2007	Retrospective	78	0%	0%	100% (78/78)	

Table 4. Percentage recovery

	Deep pain positive	Deep pain negative
Conservative	86% (546/632)	27% (11/41)
Decompression	90% (1486/1650)	56% (335/600)
Fenestration	94% (694/742)	45% (95/209)

Table 2. Nonlaminectomy

Author	Study design	Number of cases	% of non-ambulatory	% of DPN	% of recovery DPP	% of recovery DPN
Brown et al. 1977	Retrospective	167	76%	17% (32)	63%	67% (15/32)
Loughin et al. 2005	Retrospective	48	100%	100%		73% (35/48)
Tarlowski et al. 2009	Retrospective	23	100%	13% (3	91%	33% (1/3)
Dosch et al. 2007	Prospective	51	100%	0%	80%	
Davis & Brown 2002	Retrospective	112	100%	0%	90%	
Hazlehn et al. 2005	Retrospective	30	93%	27%	86%	50% (4/8)
Mehta 1982	Retrospective	38	100%	7% (3)	100%	0% (0/3)
Scott 1987	Retrospective	40	78%	8% (3)	80%	66% (2/3)
Fenoies et al. 2002	Retrospective	71	100%	0%	86%	
Abana et al. 2012a	Retrospective	855	69.50%	25% (211)	90%	52% (133/211)
Ruddle et al. 2006	Retrospective	250	100%	10% (25)	92%	72% (17/25)
Olby et al. 2003	Retrospective	64	100%	103% (66)		58% (37/64)
Mar et al. 1995	Retrospective	44	95%	23% (11)	83%	30% (5/11)
Lubbers & Puerto 2005	Retrospective	48	100%	100% (46)		41% (19/46)
Obeck 1998	Retrospective	28	68%	3% (1)	100%	100% (1)
Schulman & Lipscott 1987	Retrospective	91	94%		90%	
Davis 1994	Retrospective	34	100%	38% (29)	93%	62% (18/29)
Scott & McKee 1999	Retrospective	24	100%	100%		62% (20/34)
Knecht 1972	Retrospective	09	100%		77%	
Jeffery et al. 2018	Prospective	178	100%	100%		58% (45/78)
Total		2279			90% (1486/1650)	56% (335/600)

Nonamb. Nonambulatory dogs,DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Table 3. Fenestration

Author	Study Design	No. Cases	% Non-amb.	% DPN	% Recovery DPP	% Recovery DPN
Padilho Filho & Selmi 1999	Retrospective	29	45%	17% (5)	100%	80% (4/5)
Burzykowski 2008	Prospective	26			88%	
Olsson 1951a,b	Prospective	28			89%	
Nakama et al. 1993	Retrospective	66	74%	39% (26)	85%	65% (17/26)
Flo 1975	Retrospective	67		17% (12)	95%	75% (9/12)
Ranzel et al. 2005	Retrospective	331		46% (152)	89%	38% (58/152)
Knapp et al. 1990	Retrospective	160	30%	0-6 (18	98%	
Denny 1978	Retrospective	30	80%	30% (9)	100%	67% (6/9)
Butterworth & Denny 1991	Retrospective	100	44%	6%	90%	33% (2/6)
Funkquist 1978	Retrospective	80		0	99%	
Davies & Sharp 1983	Retrospective	34	47%	0	91%	
Total		951			94% (694/742)	45% (95/209)

Nonamb. Nonambulatory dogs, DPN Negative deep pain perception dogs, DPP Positive deep pain perception dogs

Vet J. 2017 Feb;220:7-16. doi: 10.1016/j.tvjl.2016.12.008. Epub 2016 Dec 16.

Proportion recovery and times to ambulation for non-ambulatory dogs with thoracolumbar disc extrusions treated with hemilaminectomy or conservative treatment: A systematic review and meta-analysis of case-series studies.

Langerhuus L[1], Miles J[2].

Author information

Abstract

Thoracolumbar intervertebral disc extrusion is a common cause of spinal cord dysfunction in dogs. Peer-reviewed studies reporting treatment of predominantly chondrodystrophic dogs with disc extrusion with loss of ambulation with either hemilaminectomy or conservative treatment (rest, analgesics and anti-inflammatories) were evaluated in a systematic review of the literature. Generally, the level of evidence available was low with no controlled studies and only case series available. In the meta-analysis, there was a clear trend to a greater proportion of dogs recovering and returning faster to ambulation for dogs treated with hemilaminectomy than for conservatively treated dogs. The mean proportions that recovered for neurological grades 3, 4 and 5 were 93, 93 and 61% for those treated with hemilaminectomy, and 79, 62 and 10% for those treated conservatively (Grade 3 - non-ambulatory paraparetic dogs; grade 4 - paraplegic dogs with intact deep pain perception; grade 5 - paraplegic dogs without intact deep pain perception). Due to the use of case series, these results represent between-study comparisons, thereby increasing the risk of selection bias and other biases. Data presented in this review support the current recommendations for surgical management of non-ambulatory dogs with disc-extrusion, but controlled clinical studies comparing outcomes are necessary to confirm these findings.

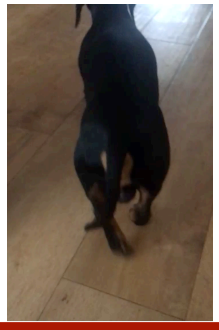
Vet J. 2017 Feb;220:7-16. doi: 10.1016/j.tvjl.2016.12.008. Epub 2016 Dec 16.

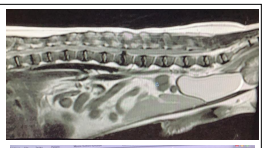
Proportion recovery and times to ambulation for non-ambulatory dogs with thoracolumbar disc extrusions treated with hemilaminectomy or conservative treatment: A systematic review and meta-analysis of case-series studies.

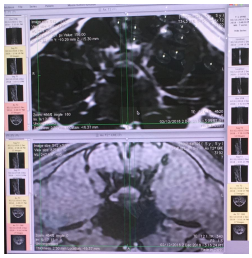
Langerhuus L[1], Miles J[2].

Author information

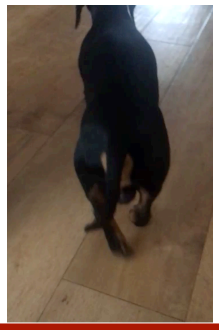
	Grade 3	Grade 4	Grade 5
Conservador	79%	62%	10%
Hemilaminectomía	93% t recuperación 10 d	93% t recuperación 15 d	61% t recuperación 38 d

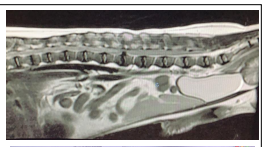


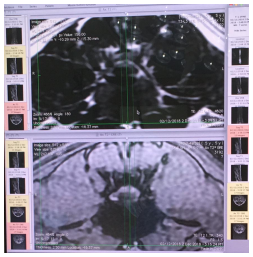




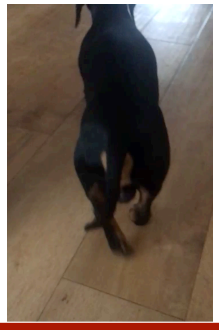
Cuando es tu propio perro…

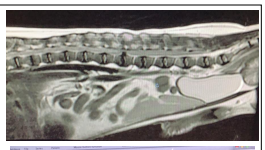


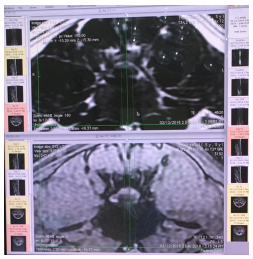




Cuando es tu propio perro…







Y sale del quirófano sin SSP…

MARCHA

Journal of Veterinary Internal Medicine

ACVIM

Open Access

Standard Article

J Vet Intern Med 2017;31:492–497

Acquisition of Involuntary Spinal Locomotion (Spinal Walking) in Dogs with Irreversible Thoracolumbar Spinal Cord Lesion: 81 Dogs

A. Gallucci, L. Dragone, M. Menchetti, T. Gagliardo, M. Pietra, M. Cardinali, and G. Gandini

Background: Spinal walking (SW) is described as the acquisition of an involuntary motor function in paraplegic dogs and cats without pain perception affected by a thoracolumbar lesion. Whereas spinal locomotion is well described in cats that underwent training trials after experimental spinal cord resection, less consistent information is available for dogs.

Hypothesis: Paraplegic dogs affected by a thoracolumbar complete spinal cord lesion undergoing intensive physical rehabilitation could acquire an autonomous SW gait under field conditions.

Animals: Eighty-one acute paraplegic thoracolumbar dogs without pelvic limb pain perception.

Methods: Retrospective study of medical records of dogs selected for intensive rehabilitation treatment in paraplegic dogs with absence of pain perception on admission and during the whole treatment. Binary regression and multivariate logistic regression were used to analyze potential associations with the development of SW.

Results: Autonomous SW was achieved in 48 dogs (59%). Median time to achieve SW was of 75.5 days (range: 16–350 days). On univariate analysis, SW gait was associated with younger age ($P = .002$) and early start of physiotherapy ($P = .024$). Multivariate logistic regression showed that younger age (≤60 months) and lightweight (≤7.8 kg) were positively associated with development of SW ($P = .012$ and $P < .001$, respectively). BCS, full-time hospitalization, and type and site of the lesion were not significantly associated with development of SW.

Conclusions: Dogs with irreversible thoracolumbar lesion undergoing intensive physiotherapic treatment can acquire SW. Younger age and lightweight are positively associated with the development of SW gait.

Key words: dog; pain perception; paraplegia; physiotherapy.

42

ESPINAL

Acquisition of Involuntary Spinal Locomotion (Spinal Walking) i
Dogs with Irreversible Thoracolumbar Spinal Cord Lesion: 81 Dog

A. Gallucci, L. Dragone, M. Menchetti, T. Gagliardo, M. Pietra, M. Cardinali, and G. Gandini

Background: Spinal walking (SW) is described as the acquisition of an involuntary motor function in paraplegic dogs and cats without pain perception affected by a thoracolumbar lesion. Whereas spinal locomotion is well described in cats that underwent training trials after experimental spinal cord resection, less consistent information is available for dogs.

Hypothesis: Paraplegic dogs affected by a thoracolumbar complete spinal cord lesion undergoing intensive physical rehabilitation could acquire an autonomous SW gait under field conditions.

Animals: Eighty-one acute paraplegic thoracolumbar dogs without pelvic limb pain perception.

Methods: Retrospective study of medical records of dogs selected for intensive rehabilitation treatment in paraplegic dogs with absence of pain perception on admission and during the whole treatment. Binary regression and multivariate logistic regression were used to analyze potential associations with the development of SW.

Results: Autonomous SW was achieved in 48 dogs (59%). Median time to achieve SW was of 75.5 days (range: 16–350 days). On univariate analysis, SW gait was associated with younger age (P = .002) and early start of physiotherapy (P = .024). Multivariate logistic regression showed that younger age (≤60 months) and lightweight (≤7.8 kg) were positively associated with development of SW (P = .012 and P < .001, respectively). BCS, full-time hospitalization, and type and site of the lesion were not significantly associated with development of SW.

Conclusions: Dogs with irreversible thoracolumbar lesion undergoing intensive physiotherapic treatment can acquire SW. Younger age and lightweight are positively associated with the development of SW gait.

Key words: dog; pain perception; paraplegia; physiotherapy.

Acquisition of Involuntary Spinal Locomotion (Spinal Walking) i
Dogs with Irreversible Thoracolumbar Spinal Cord Lesion: 81 Dog

A. Gallucci, L. Dragone, M. Menchetti, T. Gagliardo, M. Pietra, M. Cardinali, and G. Gandini

Background: Spinal walking (SW) is described as the acquisition of an involuntary motor function in paraplegic dogs and cats without pain perception affected by a thoracolumbar lesion. Whereas spinal locomotion is well described in cats that underwent training trials after experimental spinal cord resection, less consistent information is available for dogs.

Hypothesis: Paraplegic dogs affected by a thoracolumbar complete spinal cord lesion undergoing intensive physical rehabilitation could acquire an autonomous SW gait under field conditions.

Animals: Eighty-one acute paraplegic thoracolumbar dogs without pelvic limb pain perception.

Methods: Retrospective study of medical records of dogs selected for intensive rehabilitation treatment in paraplegic dogs with absenc ... riate logistic regression w

Results: ... days (range: 16–350 days ... hysiotherapy (P = .024). ... re positively associated w ... e and site of the lesion w

Conclusi ... acquire SW. Younger ag

Key words: dog; pain perception; paraplegia; physiotherapy.

"Grado 5. Marcha espinal 59% en un tiempo medio de 75.5 días. **Mejores candidatos < 7.8 kg, < 5 años e inicio rápido de fisioterapia.**"

D

D

D

44

SCHNAUZER 6 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
Mielopatía C6-T2
Asimetría (izquierda)
NO dolorosa
44

Labrador7 años M

Monoplejia **aguda** con mejoría

Mielopatía T3-L3

Asimetriía (izquierdo)

NO doloroso

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

Mielopatía C6-T2

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

Mielopatía T3-L3

Asimetría (izquierdo)

NO doloroso

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

Mielopatía C6-T2

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

Mielopatía T3-L3

Asimetría (izquierdo)

NO doloroso

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

Mielopatía C6-T2

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

Mielopatía T3-L3

Asimetría (izquierdo)

NO doloroso

46

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

Mielopatía C6-T2

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

Mielopatía T3-L3

Asimetría (izquierdo)

NO doloroso

46

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

Mielopatía C6-T2

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

Mielopatía T3-L3

Asimetría (izquierdo)

NO doloroso

46

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

MIELOPATÍA FOCAL

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

MIELOPATÍA FOCAL

Asimetría (izquierdo)

NO doloroso

46

SCHNAUZER 6 años M

Inicio agudo de hemiparesia / hemiplejía. Mejoría

MIELOPATÍA FOCAL

Asimetría (izquierda)

NO dolorosa

Labrador7 años M

Monoplejia **aguda** con **mejoría**

MIELOPATÍA FOCAL

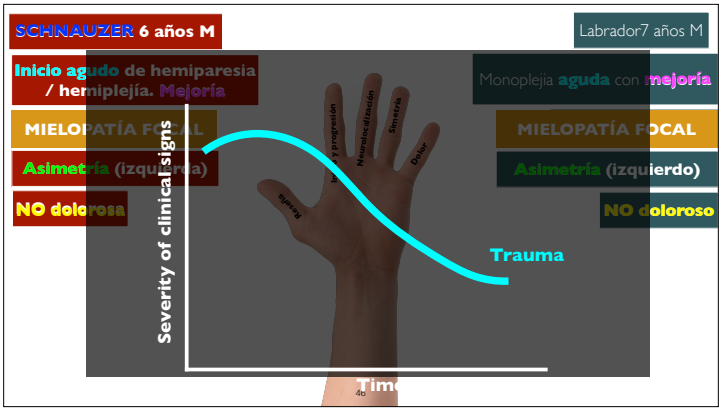
Asimetría (izquierdo)

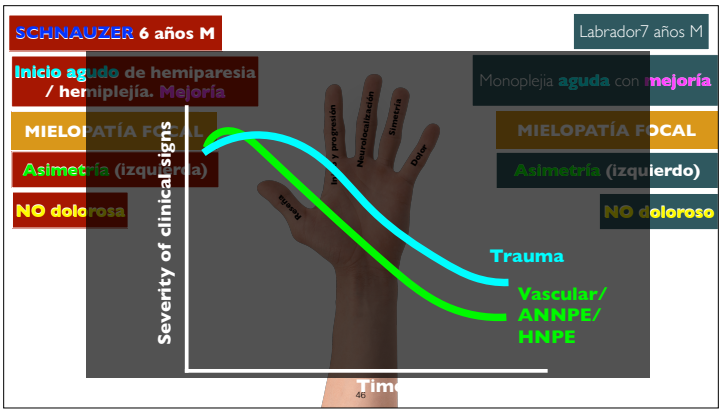
NO doloroso

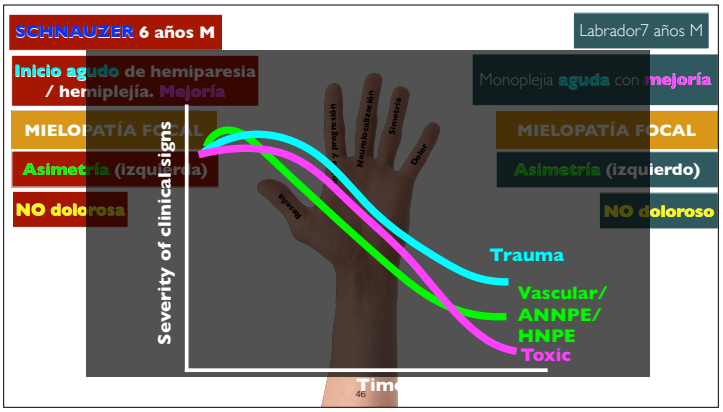
46

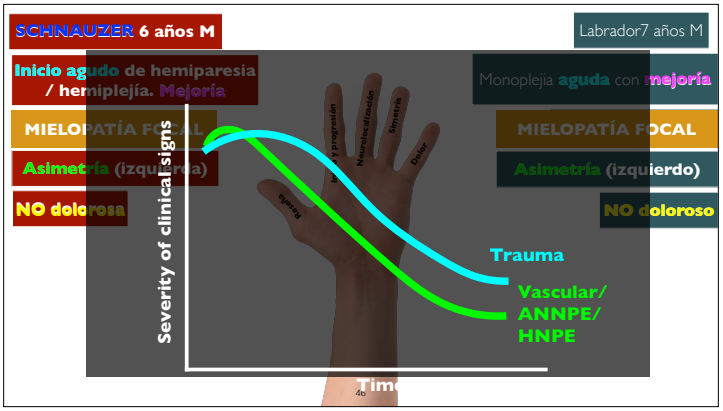
SCHNAUZER 6 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Labrador7 años M
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Inicio y progresión
Neurolocalización
Simetría
Dolor
Reacción
46

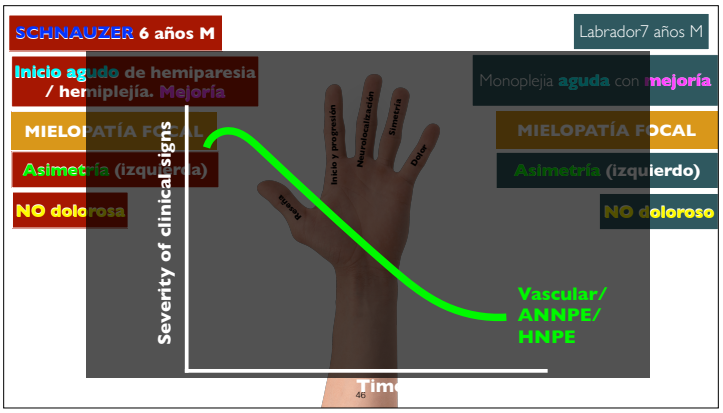
SCHNAUZER 6 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Labrador7 años M
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Severity of clinical signs
Time
46


SCHNAUZER 6 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Labrador7 años M
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Severity of clinical signs
Trauma
Time
46


SCHNAUZER 6 años M
Labrador7 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Severity of clinical signs
Trauma
Vascular/ ANNPE/ HNPE
Time

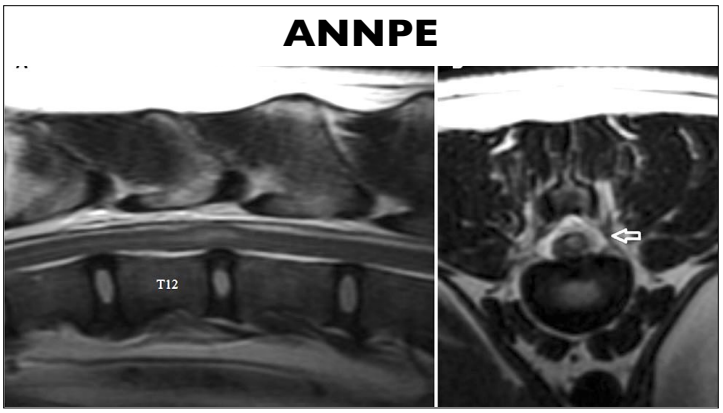

SCHNAUZER 6 años M
Labrador7 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Severity of clinical signs
Trauma
Vascular/ ANNPE/ HNPE
Toxic
Time

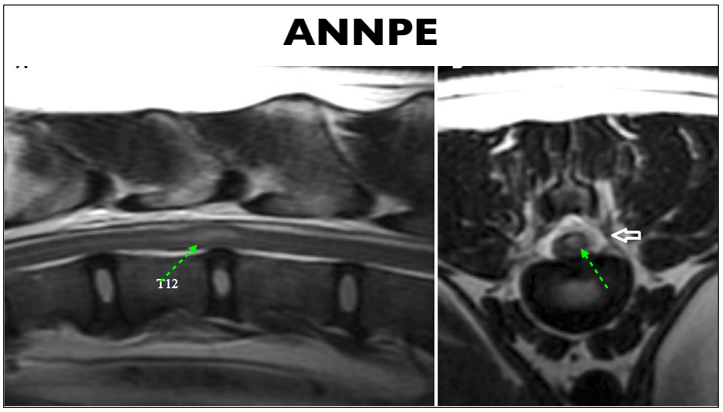

SCHNAUZER 6 años M
Labrador7 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Severity of clinical signs
Trauma
Vascular/ ANNPE/ HNPE
Time

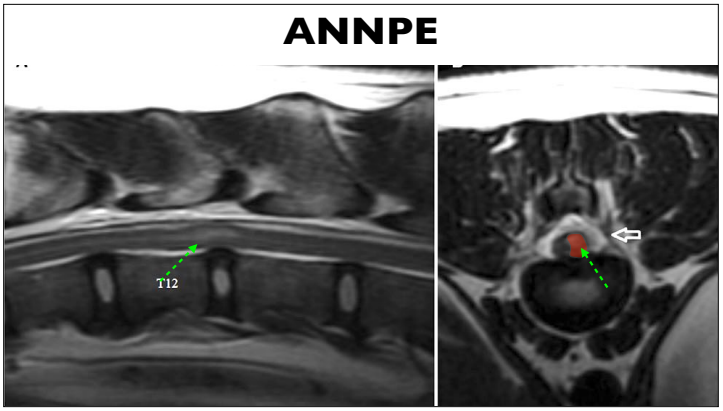

SCHNAUZER 6 años M
Inicio agudo de hemiparesia / hemiplejía. Mejoría
MIELOPATÍA FOCAL
Asimetría (izquierda)
NO dolorosa
Labrador7 años M
Monoplejia aguda con mejoría
MIELOPATÍA FOCAL
Asimetría (izquierdo)
NO doloroso
Severity of clinical signs
Inicio y progresión
Neurolocalización
Simetría
Dolor
Reflejos
Vascular/ ANNPE/ HNPE
Time
46

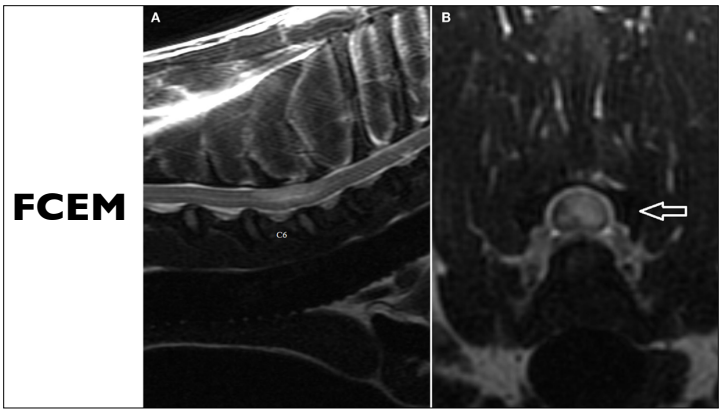
MI (EFC)
Vascular/ ANNPE/ HNPE

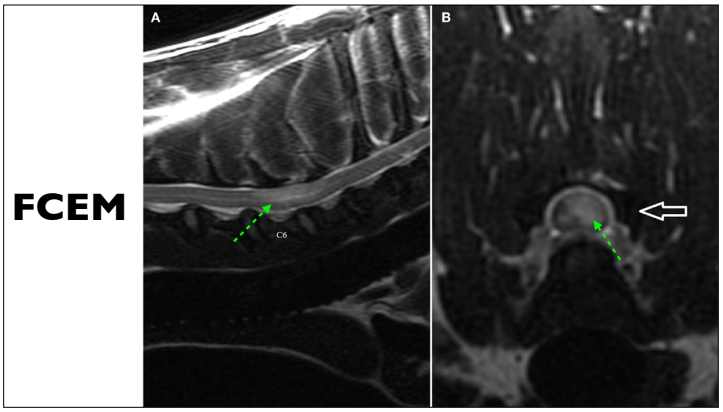
MI (EFC)
ANNPE HNPE
Vascular/ ANNPE/ HNPE

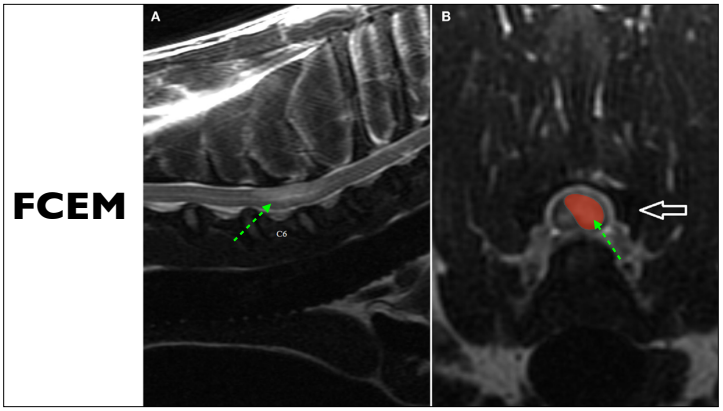

ANNPE
T12


ANNPE
T12


ANNPE
T12

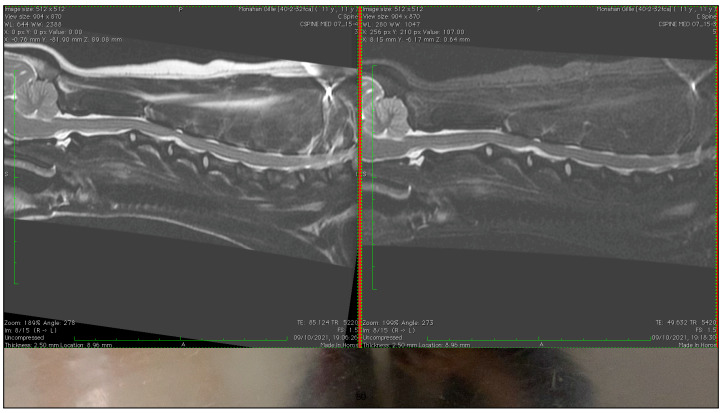

FCEM
A
B
C6

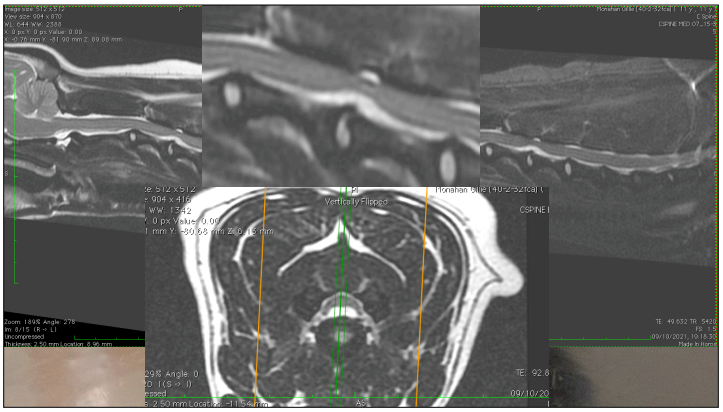

FCEM
A
B
C6

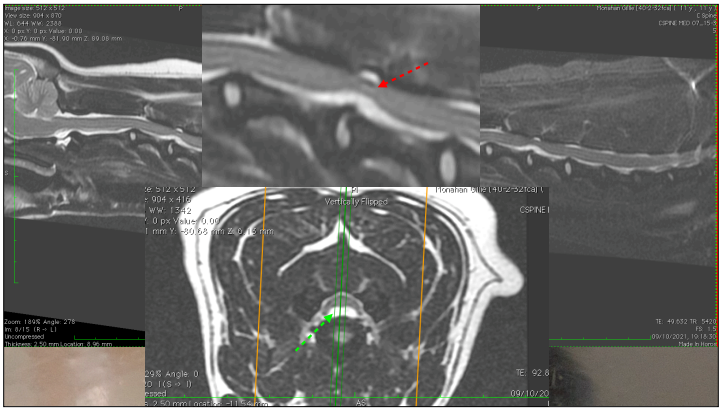

FCEM
A
B
C6

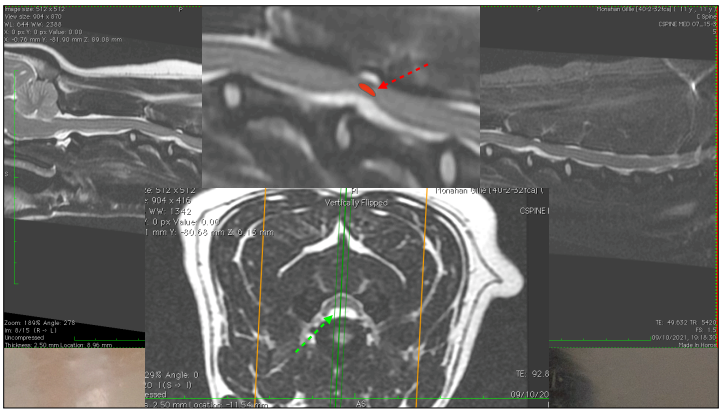
49

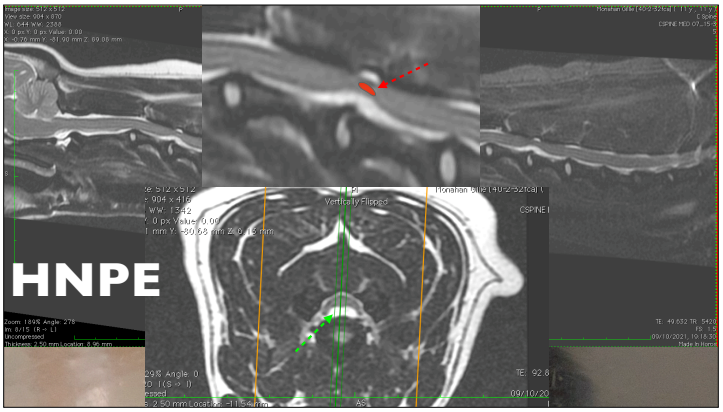
49




Vertically Flipped


Vertically Flipped





AL GRANO

PAPER

Clinical and magnetic resonance imaging features of canine compressive cervical myelopathy with suspected hydrated nucleus pulposus extrusion

Paper

Comparison of medical and surgical treatment for acute cervical compressive hydrated nucleus pulposus extrusion in dogs

Paper

Clinical characteristics of canine fibrocartilaginous embolic myelopathy (FCE): a systematic review of 393 cases (1973–2013)

Paper

Outcome comparison in dogs with a presumptive diagnosis of thoracolumbar fibrocartilaginous embolic myelopathy and acute non-compressive nucleus pulposus extrusion

51

PAPER

Clinical and magnetic resonance imaging features of canine compressive cervical myelopathy with suspected hydrated nucleus pulposus extrusion

Paper

Comparison of medical and surgical treatment for acute cervical compressive hydrated nucleus pulposus extrusion in dogs

LA MAYORÍA DE LOS PACIENTES MEJORAN

NO EXISTEN DIFERENCIAS SIGNIFICATIVAS EN RESULTADO ENTRE MI EFC y ANNPE

NO HAY DIFERENCIAS SIGNIFICATICVAS ENTRE TTO QUIRÚRGICO O CONSERVADOR PARA HNPE (?)

51

E

Inflamatoria y aun sin RM…?

52

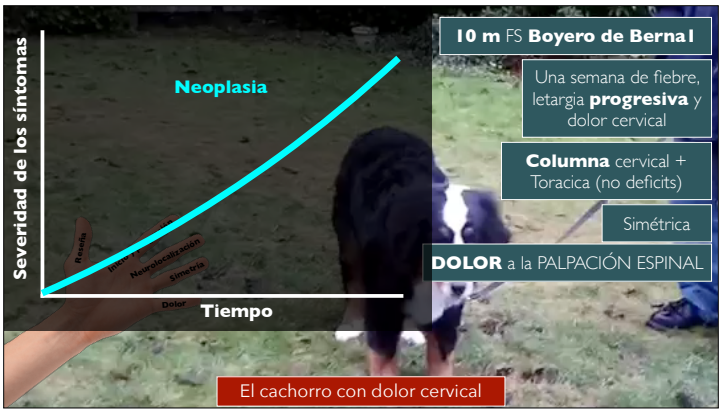
E

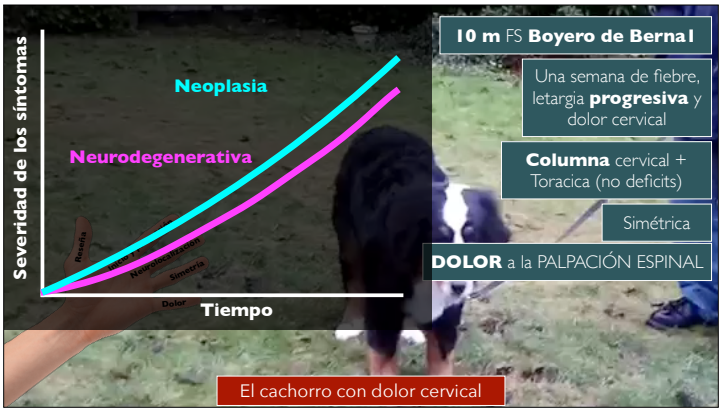
Inflamatoria y aun sin RM…?

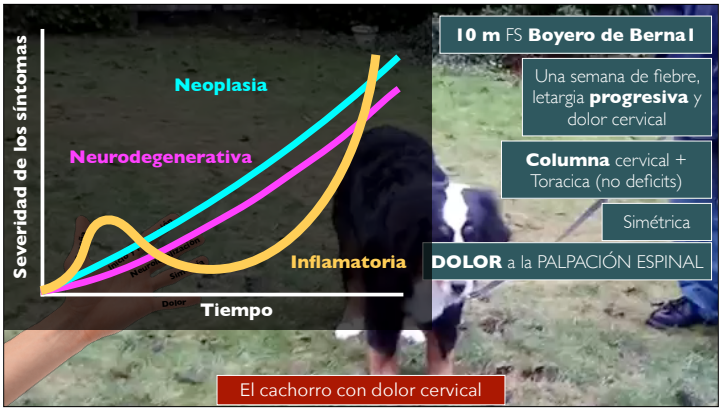
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

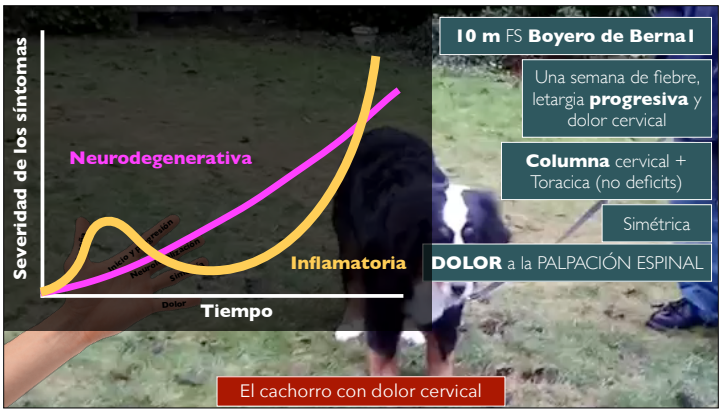
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
Reseña
Inicio y progresión
Neurolocalización
Simetría
Dolor
El cachorro con dolor cervical

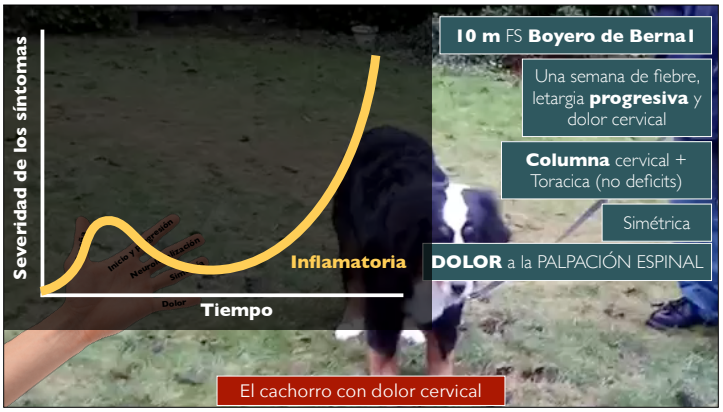
Severidad de los síntomas
Tiempo
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

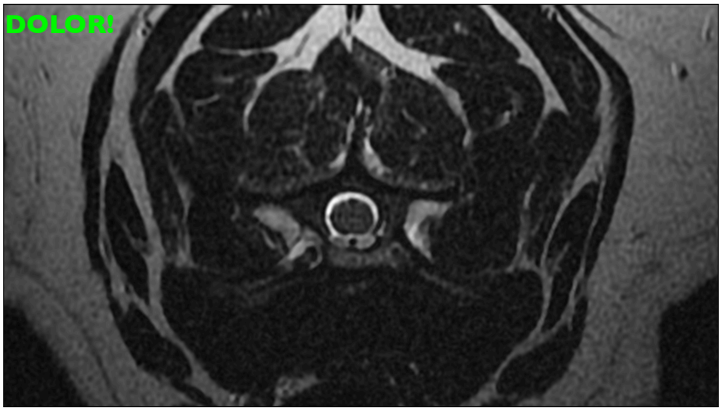

Severidad de los síntomas
Neoplasia
Tiempo
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

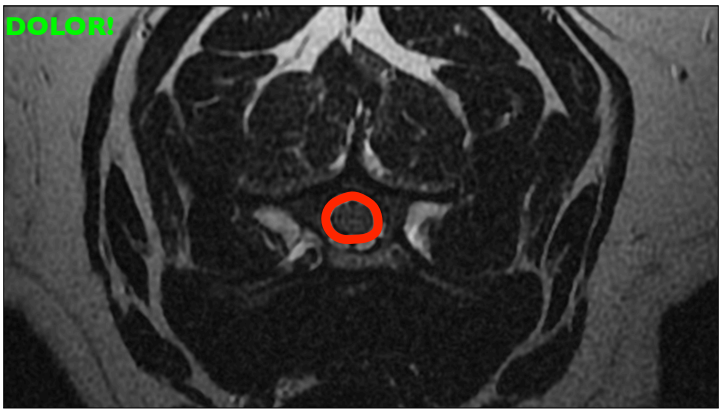

Severidad de los síntomas
Neoplasia
Neurodegenerativa
Tiempo
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

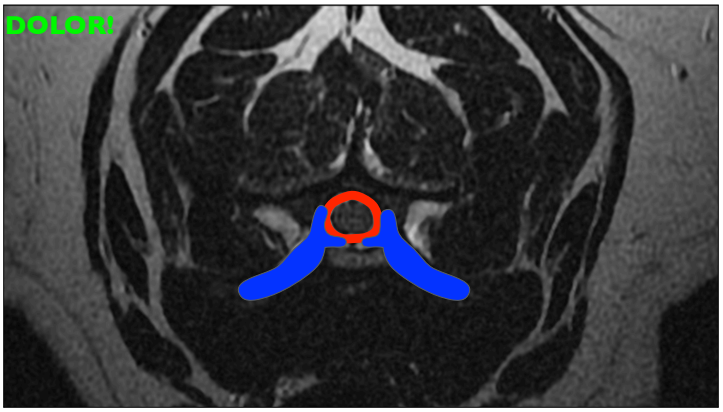

Severidad de los síntomas
Neoplasia
Neurodegenerativa
Inflamatoria
Tiempo
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

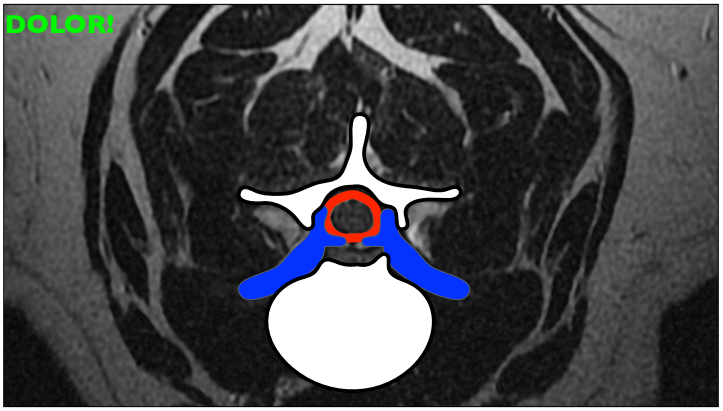

Severidad de los síntomas
Neurodegenerativa
Inflamatoria
Tiempo
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

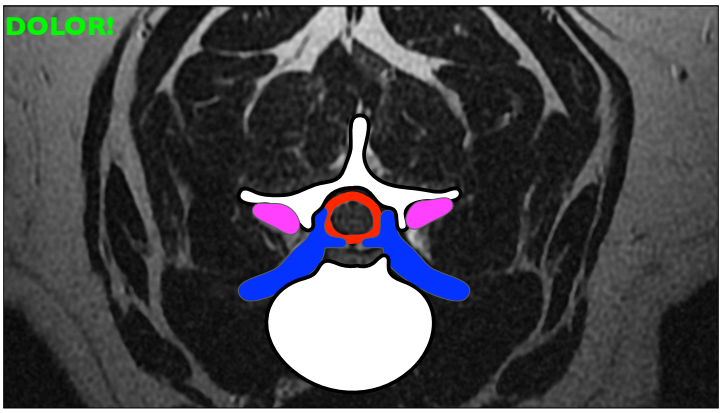

Severidad de los síntomas
Inflamatoria
Tiempo
10 m FS Boyero de Bernal
Una semana de fiebre, letargia progresiva y dolor cervical
Columna cervical + Toracica (no deficits)
Simétrica
DOLOR a la PALPACIÓN ESPINAL
El cachorro con dolor cervical

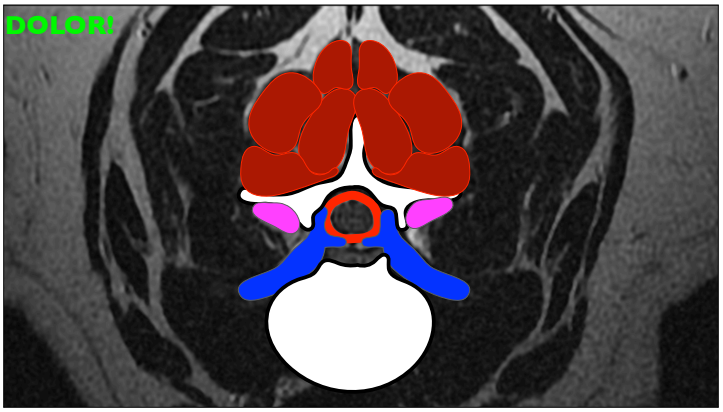

DOLOR!

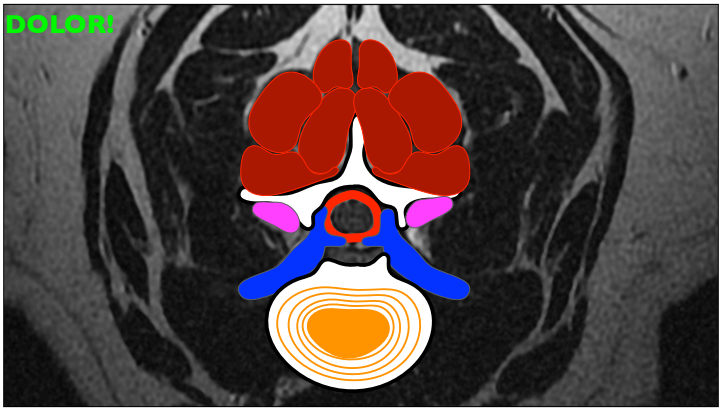

DOLOR!

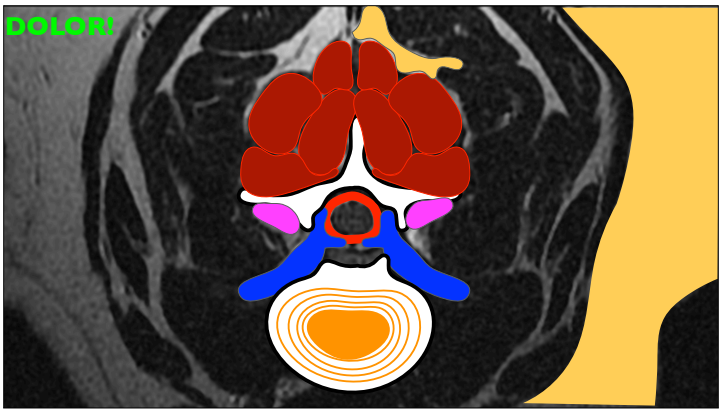

DOLOR!

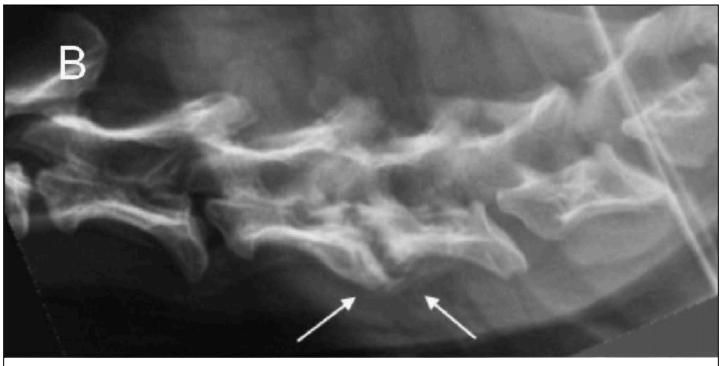

DOLOR!


DOLOR!

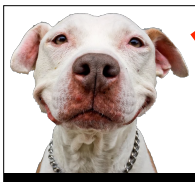

DOLOR!


DOLOR!


DOLOR!

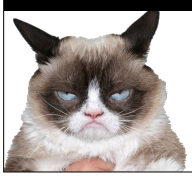

B

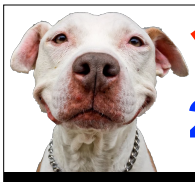
MENSAJES PARA CASA



1 La RM es útil... pero NO es una máquina milagrosa!

MENSAJES PARA CASA

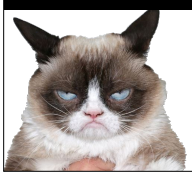


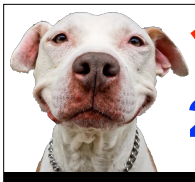


1 La RM es útil... pero NO es una máquina milagrosa!

2 Utiliza razonamientos clínicos simples para obtener buenos diferenciales.
La Neurología NO es solo RM!

MENSAJES PARA CASA

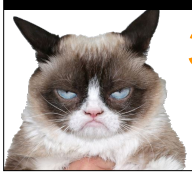




1 La RM es útil... pero NO es una máquina milagrosa!

2 Utiliza razonamientos clínicos simples para obtener buenos diferenciales.
La Neurología NO es solo RM!

MENSAJES PARA CASA

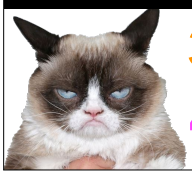


3 Metabolicos-Toxicos-Neuromusculares!
NO todos los casos necesitan una RM.

1 La RM es útil... pero NO es una máquina milagrosa!

2 Utiliza razonamientos clínicos simples para obtener buenos diferenciales.
La Neurología NO es solo RM!

MENSAJES PARA CASA



3 Metabolicos-Toxicos-Neuromusculares!
NO todos los casos necesitan una RM.

4 La Neurología tiene sentido hasta que deja de tenerlo!
NO todos los casos siguen las reglas pero...
el tiempo siempre cuenta la verdad de los casos!