Granger, N., & Neeves, J. (2015). Developing objective measures of gait abnormalities in dogs with degenerative myelopathy. *Veterinary Record*, 176(11), 290. <https://doi.org/10.1136/vr.h1344>

Peer reviewed version

Link to published version (if available):
[10.1136/vr.h1344](https://doi.org/10.1136/vr.h1344)

[Link to publication record in Explore Bristol Research](#)
PDF-document

University of Bristol - Explore Bristol Research

General rights

This document is made available in accordance with publisher policies. Please cite only the published version using the reference above. Full terms of use are available:
<http://www.bristol.ac.uk/red/research-policy/pure/user-guides/ebr-terms/>

Degenerative myelopathy in dogs: the need for objective measures of gait abnormalities.

Nicolas Granger DVM PhD Dip.ECVN FHEA MRCVS, Jacob Neeves BVSc 3rd year, MSc student

Correspondence:

Please email:

Dr Nicolas Granger: nicolas.granger@bristol.ac.uk

or

Mr Jacob Neeves: jn1216.2011@my.bristol.ac.uk

We would like to bring to the attention of colleagues a research project we are conducting in the School of Veterinary Sciences, University of Bristol, on degenerative myelopathy in dogs and ask for contribution with affected cases (diagnosed by exclusion of compressive and inflammatory spinal cord diseases on MRI and CSF analysis plus with genetic testing).

Degenerative myelopathy (DM) in dogs has long since been characterised as an insidious and progressive neurodegenerative disease affecting the brain and spinal cord, initially described in German Shepherds in the seventies (1). The disease leads to pelvic limb ataxia and paresis, and eventually paralysis. Nowadays it is a disease identified in an ever increasing number of breeds (cases from fifty-six breeds were recently reported (2) including Corgis, Boxers, Cavalier King Charles Spaniels, Chesapeake Bay Retrievers, Rhodesian Ridgebacks, Pugs) and whilst the clinical picture is well recognised, there is no treatment for this condition. We possess an increasing understanding of DM pathology and genetics, but to date we are lacking objective measures to quantify the severity of the disease. Researcher in the University of Missouri, College of Veterinary Medicine, have played a key role in advancing our knowledge in DM. The Canine Genetic Diseases Network provides an overview of the disease (<http://www.caninegeneticdiseases.net/DM/basicDM.htm>).

Affected dogs are typically presented to veterinarians with owners describing toe dragging and crossing over of pelvic limbs. Affected dogs can be either sex, usually around eight years of age. On neurological examination, affected individuals usually show an asymmetrical pelvic limb ataxia and paresis with preserved spinal reflexes in the pelvic limbs (i.e. affecting the 'upper motor neuron' within the T3-L3 spinal cord segments). The disease is non-painful. The clinical presentation is non-specific, but the insidious onset, slow progression and lack of spinal pain can orientate the differential diagnosis towards a degenerative disease, and a presumptive diagnosis of DM.

A more accurate diagnosis relies on exclusion of compressive and inflammatory diseases of the spine and spinal cord (using magnetic resonance imaging and cerebrospinal fluid analysis). Unfortunately, definitive diagnosis relies upon post-mortem examination, which shows axonal degeneration and secondary demyelination within the white matter funiculi. Genetic research has helped in recent years because a mutation of the *super-oxide dismutase 1* gene (*SOD1*) has been linked to the disease (3). Cytoplasmic inclusion bodies containing SOD1 antigens are found on pathological specimens. However, whilst *SOD1* mutation homozygosis is a predisposing risk factor for the disease, some affected animals will not bear the mutation and dogs with the mutation might not develop the disease.

We have identified a need for objective outcome measures to allow quantification of the gait abnormalities in affected individuals. This has a dual purpose: (i) being able to grade affected cases; and (ii) being able to assess the efficacy of new therapies that might arise in the near future for this condition. We propose to record the gait of affected dogs using a

previously developed kinematic system for gait analysis in dogs. This simple and non-invasive method consists of walking the dog on a canine treadmill and recording the position of limbs in space via reflective markers placed on the fur and filmed with infrared cameras. Whilst this is providing extremely precise data, it also provides the dogs with a form of motor training on the treadmill. There are some evidences, although limited, that hydrotherapy and training prolong survival of affected cases. The project was reviewed and accepted by an ethical committee and was given the veterinary investigation number VIN/14/035.

We would be grateful if colleagues could contribute to our project by contacting us if they are presented with a case of DM (we are happy to discuss suitability of cases) in any breed, or if they could direct to us the owners or breeders owning animals suspected to be affected by DM.

We are grateful to the Kennel Club Charitable Trust who provided support for the purchase of our kinematic equipment.

References

- (1) Averill DR. Degenerative myelopathy in the aging German Shepherd Dog: Clinical and pathologic findings. *J Am Vet Med Assoc* 1973;162:1045-51.
- (2) Zeng R, Coates JR, Johnson GC, Hansen L, Awano T, Kolicheski A, Ivansson E, Perloski M, Lindblad-Toh K, O'Brien DP, Guo J, Katz ML, Johnson GS. Breed distribution of SOD1 alleles previously associated with canine degenerative myelopathy. *J Vet Intern Med* 2014;28(2):515-21.
- (3) Awano T, Johnson GS, Wade CM, Katz ML, Johnson GC, Taylor JF, Perloski M, Biagi T, Baranowska I, Long S, March PA, Olby NJ, Shelton GD, Khan S, O'Brien DP, Lindblad-Toh K, Coates JR. Genome-wide association analysis reveals a *SOD1* mutation in canine degenerative myelopathy that resembles amyotrophic lateral sclerosis. *Proc Natl Acad Sci USA* 2009;106:2794-99.