10th BVNS Symposium Proceedings

"Exploring sleep disorders and the science of sleep"

British Veterinary Neurology Society

Wednesday 19th March 2025

Manchester Central Convention Centre

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Welcome

Dear colleagues and friends,

On behalf of the BVNS Committee members, it is our great pleasure to welcome you to the 10th British Veterinary Neurology Society Annual Symposium.

We are pleased to offer exciting keynote lectures from the human fields of sleep science, as well as recent progress in the field of veterinary sleep disorders and management, and recent research developments. We hope these talks will continue to spark the ongoing interest in advancing translational medicine for the benefit of our patients.

Our society is centred on sharing knowledge and research ideas. Thus, it is our pleasure to not only offer an exciting programme but to also have the opportunity to connect and collaborate with each other, exchange our knowledge and experiences, and to have fun together.

We would like to thank our sponsors once again who make this meeting possible.

Yours truly,

BVNS Committee

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10th BVNS Symposium Programme 2025

"Exploring sleep disorders and the science of sleep"

Wednesday 19th March 2025

08:30-09:00 **Registration & Coffee**

09:00-09:15 **BVNS President - Welcome & Introduction**

09:15-10:00 **Professor Derk-Jan Dijk PhD, FRSB, FMedSci**
Professor of Sleep and Physiology, University of Surrey
"Sleep regulation and sleep disorders in humans and other animals"

10:00-10:30 **Dr Carrie Tooley BSc BVetMed MSc PGCVE DipECAWBM(BM) CCAB FHEA MRCVS**
Behavioural Referrals veterinary practice, Chester
"Sleep deprivation – Why should we care about poor sleep in our patients?"

10:30-10:45 **Sponsors presentations**

10:45-11:15 **COFFEE BREAK**

11:15-12:00 **Dr Mkael Symmonds BM BCh PhD FRCP**
Consultant in Neurology and Clinical Neurophysiology, University of Oxford NHS Trust
An overview of neurological sleep disorders in humans

12:00-12:45 **Dr Susana Monforte DVM MSc CertAVP PGCertVPS AFHEA DipECVN MRCVS**
Clinical Neurologist, University of Cambridge
'BOAS, the canine 'obstructive sleep apnoea' effect on the brain - The BBB study'

12:45-14:00 **LUNCH**

14:00-14:30 **Clinical case discussion with Prof Dijk and Dr Symmonds**

14:30-15:00 **Dr Carrie Tooley BSc BVetMed MSc PGCVE DipECAWBM(BM) CCAB FHEA MRCVS**
Behavioural Referrals veterinary practice, Chester
A practical toolkit for assessing and treating poor sleep in veterinary species

15:00-15:30 **COFFEE BREAK**

15:30-16:45 Clinical Research Abstracts

Short communications (10 min)

A. Karpozilou: CERVICAL VENTROFLEXION IN CATS: 86 CASES (2003-2024)

I. Clemot: DYNAMIC MRI OF THE LUMBOSACRAL SPINE IN NEUTRAL AND FLEXED POSITION FOR PRE-SURGICAL ASSESSMENT OF CLINICALLY AFFECTED DOGS WITH DEGENERATIVE LUMBOSACRAL STENOSIS

A. Patel: A RETROSPECTIVE STUDY IN 65 DOGS TO CHARACTERISE SURGICAL TREATMENT OF EARLY POST-OPERATIVE NEUROLOGICAL DETERIORATION FOLLOWING HEMILAMINECTOMY

O. Chan: MAGNETIC RESONANCE IMAGING-BASED MORPHOLOGICAL ASSESSMENT OF THE THALAMUS AND CAUDATE NUCLEI IN DOGS WITH IDIOPATHIC EPILEPSY

Flash presentations (3 min)

G. Kaemper: A CASE REPORT OF SUSPECTED ACQUIRED NARCOLEPSY IN A DOG WITH STEROID RESPONSIVE MENINGITIS ARTERITIS.

G. Kaemper: DELAYED ONSET CONGENITAL NARCOLEPSY FOLLOWING ANAESTHESIA IN A DACHSHUND

J. Poacher: SUSPECTED DISCOGENIC PAIN IN A SPRINGER SPANIEL DOG

I. Molina: VERTEBRAL FIBROUS DYSPLASIA IN A CAT

A. Lande: SEVERE CONGENITAL HYDROCEPHALY AND SYRINGOMYELIA IN CREAM, LONG-HAIRED MINIATURE DACHSHUND PUPPIES.

F. Decrop: ACUTE GLOSSITIS SECONDARY TO A CAUDAL LINGUAL ABSCESS IN A DOG WITH MENINGOENCEPHALOMYELITIS OF UNKNOWN ORIGIN TREATED WITH IMMUNOMODULATORY THERAPY.

J. de Frias: MONOCULAR NYSTAGMUS IN A DOG DIAGNOSED WITH PRESUMPTIVE MENINGOENCEPHALITIS OF UNKNOWN ORIGIN

R. Hall: LONG-TERM OUTCOMES IN LGI1-AUTOANTIBODY ASSOCIATED FELINE AUTOIMMUNE ENCEPHALITIS: PRELIMINARY FINDINGS FROM AN OWNER QUESTIONNAIRE STUDY

M. Mees: L7 TRANSVERSECTOMY FOR TREATMENT OF ‘FAR-OUT SYNDROME’ CAUSED BY A CONGENITAL VERTEBRAL BODY MALFORMATION IN A DOG

A. Fischer: FEASIBILITY OF ACTICAL® ACCELEROMETERS FOR MONITORING SLEEP AND REST PERIODS IN INDIVIDUAL DOGS

16:50-17:00 Closing remarks and AGM

17:00 A drink together at the Peaky Blinders

Invited Speakers

Professor Derk-Jan Dijk PhD, FRSB, FMedSci

Professor of Sleep and Physiology, Director Surrey Sleep Research Centre
University of Surrey

Derk-Jan Dijk PhD, FRSB, FMedSci is Distinguished Professor of Sleep and Physiology and Director of the Surrey Sleep Research Centre at the University of Surrey. He is Group Leader for Improving Sleep and Circadian Disruption in the UK Dementia Research Institute.

Dr Dijk has more than 40 years of experience in clinical sleep research. His current research interests include: the interaction of sleep homeostasis and circadian rhythmicity in the regulation of sleep and cognition; identification of novel-biomarkers for susceptibility to the negative effects of sleep loss; sleep and circadian disruption in dementia and the validation and implementation of new technologies for monitoring sleep and circadian rhythms in dementia.

Dr Dijk has published more than 300 research and review papers in the area of sleep and circadian rhythms. Dr Dijk is invited frequently to speak at international sleep meetings and he has given opening and plenary lectures for the joint meeting of the Canadian Sleep Society, American Academy of Sleep Medicine and Sleep Research Society, The European Sleep Research Society and the Hong Kong Sleep Medicine Society.

Dr Dijk has served as an Associate and Deputy Editor to SLEEP and Editor of the Journal of Sleep Research. He also serves as consultant to the pharmaceutical industry. He was a Royal Society-Wolfson Research Merit Award holder and a recipient of the Distinguished Scientist Award from the Sleep Research Society (USA).

"Sleep regulation and sleep disorders in humans and other animals"

Sleep is widespread in the animal kingdom and similarities in behavioural and electrophysiological aspects across species, in particular mammals and birds, have been investigated and demonstrated in many studies. This implies that many aspects of sleep regulation and function in humans can easily be compared to sleep in other animals and vice versa. It also implies that knowledge about the pathophysiology and aetiology of sleep disorders in humans may be relevant to the understanding of sleep disorders in small animals and large domestic animals such as cattle. Examples include narcolepsy and sleep apnoea. Biologists have been particularly interested in the sleep of species living in extreme environmental conditions such as marine mammals, migrating birds or animals living at extreme latitudes with associated exposure to constant darkness or light during winter and summer respectively. These studies have revealed surprising adaptations to these environments and even more surprising findings related to negative consequences, and absence thereof, of sleep deprivation. Large scale studies of sleep in humans living in the community or animals living in their natural habitat require technologies that are unobtrusive and can collect data over longer periods of time. In this presentation I will illustrate these comparative aspects of sleep regulation using examples from both human and animal sleep research.

Dr Carrie Tooley
BSc BVetMed MSc PGCVE DipECAWBM(BM) CCAB FHEA MRCVS
RCVS Advanced Practitioner, Companion Animal Behaviour
Behavioural Referrals veterinary practice, Chester

Carrie qualified from the Royal Veterinary College in 2012 and spent three years in mixed general practice before pursuing her interest in behavioural medicine. She completed an MSc in Clinical Animal Behaviour followed by a behavioural medicine residency. She sees canine and feline clinical cases at Behavioural Referrals Veterinary Practice and has a particular interest in the role of sleep in the management of physiological disease and behavioural presentations. Carrie is an ESRS (European Sleep Research Society) and BSS (British Sleep Society) member.

"Sleep deprivation – Why should we care about poor sleep in our patients?"

Humans spend around a third of our lives asleep, and our veterinary patients frequently a much higher proportion. But why, what is the function of this *readily reversible state of reduced responsiveness to, and interaction with, the environment* (Bear et al, 2001, 594)?
Sleep is essential for emotional health and emotional regulation, and has direct impacts on many aspects of physiological health including cardiac, endocrine, healing and repair, pain and a variety of neurological conditions.
Normal sleep must be understood before abnormal sleep can be assessed. Dogs and cats "cycle" through the same phases of sleep that humans do, experiencing light slow wave sleep, deep slow wave sleep and REM (rapid eye movement)/paradoxical sleep. Typically the cycles last for 20 minutes for dogs and 30 minutes for cats. There are variable durations for daily sleep published in the literature, with agreement that lower durations correlate with increased behavioural problems. Dogs have been reported to show sleep disorders akin to REM sleep behavioural disorder and sleep obstructive apnoea but there are a myriad of sleep disorders reported in humans which are yet to be recognised in veterinary patients.

Reference

Bear, M.F., Connors, B.W. and Paradiso, M.A. (eds.) (2010) *Neuroscience, Exploring the Brain.* Baltimore: Lippincott Williams & Wilkins.

Dr Mkael Symmonds BM BCh PhD FRCP
Consultant in Neurology and Clinical Neurophysiology
University of Oxford NHS Trust

Dr Mkael Symmonds is a dual accredited Consultant Neurologist and Clinical Neurophysiologist based at the John Radcliffe Hospital in Oxford.
He attended medical school in Oxford and undertook postgraduate specialist training in Clinical Neurophysiology, Neurology and Sleep Medicine in London and Oxford. He currently leads the supra-regional Neurology Sleep Disorders service for the south-central region and his specialist interests include non-respiratory sleep disorders and epilepsy.

"An overview of neurological sleep disorders in humans"

The talk will give a broad overview of common and rarer sleep disorders seen in a typical adult neurology sleep clinic, including hypersomnias, dyssomnias, and parasomnia disorders. The aim is to outline the methods used to investigate sleep disorders in humans, and give a systematic approach and framework for sleep assessment, before discussing the diagnostic and treatment approaches for a variety of complaints including central disorders of hypersomnolence, insomnia, sleep related movement disorders, and REM and non-REM parasomnias.

Dr Susana Monforte DVM MSc CertAVP PGCertVPS AFHEA DipECVN MRCVS
Clinical Neurologist, University of Cambridge

Susana graduated from the University of Porto, Portugal in 2010. She spent one year working in a teaching hospital in Portugal, followed by a Junior Clinical Training Scholarship in Small Animal Studies at the University of Cambridge. Susana then spent 5 years as a general practice small animal vet before returning to Cambridge in 2018, first as a Junior Neurologist, followed by a Residency in Neurology and Neurosurgery. Susana is a Diplomate of the European College of Veterinary Neurology and works as an Associate Teaching Professor and Clinical Neurologist at the QVSH - University of Cambridge. She combines teaching and clinical work with research into the association between the reduction of brain oxygenation and development of brain-associated signs in brachycephalic dogs, the main focus of her ongoing PhD at the University of Cambridge, and the genetics of spongiform leucoencephalomyelopathy in Border Terriers.

"BOAS, the canine 'obstructive sleep apnoea' effect on the brain - The BBB study"

Brachycephalic dogs (p.e, Pugs, French bulldogs and English bulldogs) have a high prevalence of brachycephalic obstructive airway syndrome (BOAS). This respiratory conformational disease has been associated with several other disorders in affected dogs, numerous of which are believed to be associated with a chronic state of intermittent, systemic hypoxia. One of these conditions is sleep disordered breathing, which is linked to severe forms of BOAS and can improve with its medical or surgical management. The respiratory condition in dogs has been used since the 1980's as a naturally occurring animal model for obstructive sleep apnoea (OSA) in humans. OSA has been found to be associated with a multitude of brain-related disorders, including epilepsy, Alzheimer's disease, cerebrovascular accidents and, most recently, development of malignant brain tumours. Interestingly, in some patients with OSA and brain conditions, treatment of the respiratory sleep disorder can lead to a partial reversibility of the brain dysfunction and structural brain damage as seen in neuroimaging studies. Preliminary data from Cambridge has found the presence of suspected brain-related signs in brachycephalic dogs to be associated with more severe BOAS phenotypes and the perception of sleep disordered breathing by their owners. It is possible that the same pathophysiological mechanisms behind brain dysfunction in humans with OSA, ranging from chronic hypoxemia, neuroinflammation and sleep deprivation, could also cause functional and/or structural damage to the canine brain. Alternatively, or concurrently, brain dysfunction could be associated with central sleep apnoea and deterioration of the OSA by weakening the muscles of the upper airway.

Dr Carrie Tooley BSc BVetMed MSc PGCVE CCAB FHEA MRCVS
RCVS Advanced Practitioner, Companion Animal Behaviour
Behavioural Referrals veterinary practice, Chester

Carrie qualified from the Royal Veterinary College in 2012 and spent three years in mixed general practice before pursuing her interest in behavioural medicine. She completed an MSc in Clinical Animal Behaviour followed by a behavioural medicine residency. She sees canine and feline clinical cases at Behavioural Referrals Veterinary Practice and has a particular interest in the role of sleep in the management of physiological disease and behavioural presentations. Carrie is an ESRS (European Sleep Research Society) and BSS (British Sleep Society) member.

"A practical toolkit for assessing and treating poor sleep in veterinary species"

Currently, a widely-adopted or validated method for assessing sleep in clinical veterinary patients does not exist. Research techniques are, broadly speaking, impractical to employ on an individual patient level, especially if the patient is presenting for a complaint unrelated to their sleep and we are simply trying to ascertain if poor sleep is an additional factor.
Taking a strategic history is therefore important, with a focus on caregiver estimation of duration, caregiver observation of REM/paradoxical sleep movements, typical postures and locations (often most efficiently assessed from pictures) and potentially the use of data from wearable devices recommended. We discuss the SNoRE 3.0 questionnaire (Mondino et al, 2023).
Addressing poor quality sleep in veterinary patients involves 1) providing sufficient sleep opportunities (or optimising "sleep hygiene" to borrow a term from the human sleep medicine field) and 2) treating underlying causes of poor sleep.
Optimising sleep hygiene includes consideration for the comfort and accessibility of rest places (including thought for the thermoneutral zone of the species in question), reducing environmental and social disturbances, educating caregivers as to how to best facilitate sleep in their pets, optimising inter-pet relationships (or providing for each pets' needs where there is tension in a relationship) and manipulation of the daily routine of the patient where appropriate.
Treating the underlying causes of poor sleep involves optimising each aspect of the health triad (Heath, 2010) to ensure physical, cognitive and emotional needs are met.
Understanding of medications for the treatment of true sleep disorders in veterinary species are in their infancy, with more work required to form accurate diagnoses and rule out other causes of poor sleep before we reach for sleep-enhancing or sleep-inducing medications.

References

Heath; Sink analogy from the Heath Model of Emotional Health© Sarah Heath MRCVS 2010
Mondino, A., Ludwig, C., Menchaca, C., Russell, K., Simon, K.E., Griffith, E., Kis, A., Lascelles, B.D.X., Gruen, E.E. and Olby, N.J. Development and validation of a sleep questionnaire, SNoRE 3.0, to evaluate sleep in companion dogs. *Scientific Reports* 13 (2023).

Clinical Research Abstracts

CERVICAL VENTROFLEXION IN CATS: 86 CASES (2003-2024)

Athina Karpozilou,[1] Alberta De Stefani,[2] Theofanis Liatis[1,2]

[1] *Southern Counties Veterinary Specialists, Ringwood, UK*
[2] *Royal Veterinary College, Hatfield, UK*

Cervical ventroflexion is a clinical sign of weakness of the neck, particularly dramatic in cats due to the absence of nuchal ligament and its suspensory properties. Despite its frequent occurrence, no retrospective studies have been conducted to investigate this characteristic clinical sign in cats. The objective of this study is to describe cervical ventroflexion and diseases that are associated with its occurrence.

This is an observatory retrospective two-centre study conducted at two referral hospitals in the United Kingdom spanning the period from 2003 to 2024. Inclusion criteria consisted of complete medical records, presence of cervical ventroflexion upon admission, haematology and serum biochemistry including electrolytes and a final diagnosis. Statistical analysis was performed using standard statistical software (SPSS Statistics 26, IBM Corporation, Armonk, New York). The cat population was divided into two groups: hypokalaemic group (including cats with serum potassium < 3.5 mmol/L at the time of presentation) and normokalaemic group (including cats with serum potassium at 3.6-4.6mmol/L) for univariate analysis,

A total of 86 cats were included in the study. The most common diagnoses associated with cervical ventroflexion in cats were feline hypokalaemic myopathy (42/86, 48.8%), hyperthyroidism (10/86, 11.6%), thiamine deficiency (9/86, 10.5%), immune-mediated polyneuropathy (6/86, 7%), cervical ischaemic myelopathy (5/86, 5.8%), acquired myasthenia gravis (3/86, 3.5%) and FIP meningoencephalomyelitis (3/86, 3.5%). Absence of other accompanying neurological signs was significantly associated with hypokalaemia ($p<0.001$) compared to normokalaemic group. All but two cats (59/86, 68.6%) that received treatment depending on the diagnosis, showed rapid improvement of the clinical sign of cervical ventroflexion at discharge. These results can provide clinically useful information for clinicians when evaluating cats with cervical ventroflexion.

DYNAMIC MRI OF THE LUMBOSACRAL SPINE IN NEUTRAL AND FLEXED POSITION FOR PRE-SURGICAL ASSESSMENT OF CLINICALLY AFFECTED DOGS WITH DEGENERATIVE LUMBOSACRAL STENOSIS

Irenka Baldó Clemot[1], Chiara Briola[2], Abel Bulamu Ekiri[3], Rodolfo Cappello[4, 5], Riata Marinelly[1, 5], Josep Brocal[6, 7], Alice Prodger[6, 8], Lorenzo Mari[1].

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[2]Diagnostic Imaging Department, The Ralph Veterinary Referral Centre, Marlow, United Kingdom
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[4] South East Veterinary Referrals, Sevenoaks, United Kingdom
[5]North Downs specialist Referrals, part of Linnaeus Veterinary Limited. Bletchingley, United Kingdom.
[6]AndersonMoores Veterinary Specialists, part of Linnaeus Veterinary Limited. Winchester, United Kingdom
[7]Veterinary Specialty Hospital of Hong Kong, Wan Chai, Hong Kong.
[8]Department of Surgical and Radiological Sciences, College of Veterinary Medicine, University of California, Davis, California, USA

Ethical approval was not necessary for this study as entirely retrospective based only on clinical records, with no additional procedure performed on the animals nor contact with the carer made for the sole purpose of the study.

The aim of this retrospective, comparative, multicenter study was to compare diagnostic findings in flexed and neutral magnetic resonance imaging (MRI) of the lumbosacral joint (LSJ) performed for pre-surgical assessment in dogs with clinically suspected, diagnostically confirmed canine degenerative lumbosacral stenosis (DLSS), and to asses if these findings support the need for decompressive dorsal laminectomy/partial discectomy and/or foraminotomy in combination with distraction stabilization techniques.

Dogs with clinically suspected, MRI confirmed DLSS that underwent dynamic LSJ imaging were included. Medical records and MRI findings of included cases from three referral hospitals between 2020 and 2024 were reviewed. Quantitative and qualitative assessments of the LSJ were compared in neutral and flexed positions, including LSJ angle, degree of IVD protrusion and protrusion ratios, degree of IVD degeneration, spondylosis, ventral bulging of the ligamentum flavum (VBLF) and foraminal compression. Interindividual agreement was assessed among three observers.

A total of twenty-four dogs met the inclusion criteria. Quantitative and qualitative assessments of the LSJ were compared in neutral and flexed positions, including LSJ angle, degree of IVD protrusion and protrusion ratios, degree of IVD degeneration, spondylosis, ventral bulging of the ligamentum flavum (VBLF) and foraminal compression. Interindividual agreement was assessed among three observers.

The degree of IVD protrusion and protrusion ratios, the degree of foraminal stenosis and of VBLF were significantly reduced in flexion compared with neutral position ($p<0.001$ for all comparisons). No dogs had persistent compression of the cauda equina or completely occluded foramina in flexion. The response of IVD protrusion to flexion was statistically significantly directly correlated to the degree of IVD degeneration ($p = 0.004$) but not of spondylosis.

In conclusion, in a flexed position, IVD protrusions, VBLF and foraminal stenoses improved in all cases, with resolution of all compression sites. These results suggest that in LSJ distraction-stabilization techniques the need for concurrent decompressive dorsal laminectomy/partial discectomy or foraminotomy should be questioned, unless performed for IVD spacer placement. Surgical case-control studies are required to investigate this further.

A RETROSPECTIVE STUDY IN 65 DOGS TO CHARACTERISE SURGICAL TREATMENT OF EARLY POST-OPERATIVE NEUROLOGICAL DETERIORATION FOLLOWING HEMILAMINECTOMY

Patel, R.P.[1], Crawford, A.H.[2], Cardy, T.J.C[3]

[1]Anderson's Veterinary Surgery, Bromley North, UK, [2]Royal Veterinary College, Hatfield, UK, [3]Cave Veterinary Specialists, Wellington, UK

Surgical treatment of thoracolumbar intervertebral disc extrusion (IVDE) commonly involves spinal cord decompression using various techniques (with or without disc fenestration) with hemilaminectomy and mini-hemilaminectomy being performed most frequently. Recovery rates for surgically treated dogs are good with a return to ambulation of 93% to 98.5% in dogs with nociception, and 61% to 63% in dogs with absent nociception. However, complications can occur in the days to weeks following surgery. Causes of early post-operative neurological deterioration (EPOND) are surgical site infection, haematoma, further nucleus pulposus extrusion at the affected disc, myelomalacia or iatrogenic trauma from the surgical procedure. Studies of the causes of EPOND are limited, with small sample sizes, differing imaging modalities and varying durations of follow-up after surgery. The objectives of our study were to characterise the clinical presentation and outcomes of surgically treated dogs experiencing EPOND within 90 days of hemilaminectomy for thoracolumbar IVDE.

Digital records between 2007 and 2023 from the Small Animal Referral Hospital at the Royal Veterinary College and 2019 to 2023 from Cave Veterinary Specialists were reviewed. Dogs that experienced EPOND within 90 days of initial hemilaminectomy and underwent repeat MRI and a second surgery were included. Multiple metrics were collected for each case including neurological grade (Modified Frankel Score) which was recorded before and after first and second surgeries. Neurological outcome was based on the difference between grades prior to surgery and following surgery. Outcomes were considered positive if dogs had a stable/improved neurological grade following surgery and negative if they had a worse grade. All potential causes of EPOND were confirmed by repeat MRI and during the second surgery by a board certified neurologist or supervised ECVN resident. Statistical analysis was performed using GraphPad Prism.

Sixty-five dogs were included in the study, median age was 64 months (42-126 months), median weight was 8.1kg (4.0-35.0kg). Common breeds were Dachshunds (n=30), Cross Breeds (n=6), Cocker Spaniels (n=6), French Bulldogs (n=4) and Shih Tzu (n=3). Reasons for EPOND were: extrusion of further nucleus pulposus at the site of the initial IVDE ('Same Disc', n=24, 36.9%), extrusion of nucleus pulposus at an immediately adjacent site to the original IVDE ('Adjacent disc', n=20, 30.8%), extrusion of nucleus pulposus at a non-adjacent site ('Non-adjacent disc', n=11, 16.9%) and haematoma at the site of original surgery ('Haematoma', n=10, 15.4%). Time to EPOND varied significantly by cause: Haematoma (median=2 days, 2-3), Same Disc (6 days, 1-89), Non-Adjacent Disc (31 days, 2-84) and Adjacent Disc (55 days, 2-88). Older dogs (median=89.5 months, p=0.04) and heavier dogs (median=13.9kg, p=0.002) were more likely to experience EPOND from haematoma formation. Median time to discharge following second surgery was 5 days (2-15). Over 91% (n= 54) of dogs had a positive outcome after second surgery and 47.7% were ambulatory at discharge.

The current study highlighted four different causes of EPOND occurring within different timeframes following initial surgery. Regardless of cause most dogs had a good outcome following a second surgery and regained ambulatory status. These findings provide key information to make informed treatment decisions of EPOND following hemilaminectomy.**A**

MAGNETIC RESONANCE IMAGING-BASED MORPHOLOGICAL ASSESSMENT OF THE THALAMUS AND CAUDATE NUCLEI IN DOGS WITH IDIOPATHIC EPILEPSY

Chan, T.Y.[1], Israeliantz-Gunz, N[1], Madden, M.E.[1]

[1] *Royal (Dick) School of Veterinary Studies, University of Edinburgh, Midlothian, EH25 9RG, Scotland.*

Epilepsy affects 0.6-0.75% of the general canine population with idiopathic epilepsy (IE) being the most common cause. A preliminary diagnosis of IE is made in dogs developing recurrent epileptic seizures between the ages of 6 months and 6 years, with normal neurological examination and bloodwork results. Further investigations, e.g. brain magnetic resonance imaging (MRI) and cerebrospinal fluid analysis, are often performed to rule out structural causes of epilepsy and strengthen the diagnosis. Human MRI studies have documented structural changes in the thalamus and basal nuclei (BN) of epileptic patients. However, studies that specifically investigate whether equivalent structural changes exist in dogs with IE have not yet been performed.

This retrospective study aimed to investigate the morphological differences in the BN and thalamus of dogs with IE (n=40) compared to controls (n=38) using MRI studies. Ethical approval was granted by the Veterinary Ethical Review Committee (reference: 101.23). Limited by the difficulties in accurately delineating BN, which is composed of multiple nuclei, the caudate nucleus (CN) was the only BN analysed in this study. Measurements of the interthalamic adhesion (ITA) thickness and area, thalamic area and volume, and CN area and volume, were made to compare the size and symmetry of these structures between groups. All measurements were normalised to the individual's forebrain height, area or volume to account for breed-related variability in brain size and body weight (BW). Further statistical analysis was also performed to evaluate whether factors other than IE (i.e. age, weight, sex, and neuter status) had a statistically significant effect on the measurements.

Results indicated the thalamic area ($p<0.0001$), thalamic volume ($p<0.0001$), and CN volume ($p=0.0126$) were significantly smaller in the IE group. Both epileptic status and BW had a significant impact on the thalamic area (BW: $p=0.0418$; IE status: $p<0.0001$) and volume (BW: $p=0.0169$; IE status: $p<0.0001$). Meanwhile, IE dogs had a significantly larger ITA thickness ($p<0.0001$) and area ($p<0.0001$). Analysis suggested age and epileptic status had a significant effect on the ITA thickness (age: $p=0.000410$; IE status: $p=0.000139$) and area (age: $p=0.000994$; IE status: $p=0.00187$). There were no significant differences in the CN area or thalamic and CN symmetry between groups.

Considering no significant differences in BW, sex, neuter status and skull shape distribution between groups were found, their influence on the observed differences is likely minimal. However, the groups differ in age. While the impact of IE remained significant after accounting for age in statistical analysis, future studies comparing brain morphology between dogs with IE and controls should incorporate age stratification or create age-matched groups to eliminate the effect of age as a confounding factor.

In conclusion, our findings indicate a smaller thalamic and CN size is associated with canine IE. Different BN-thalamo-cortical circuits have been shown to contribute to movement disorders in dogs and epilepsy in humans. It is reasonable to hypothesise the involvement of BN-thalamo-cortical circuits in canine IE based on our findings. Therefore, future research should explore the involvement of BN-thalamo-cortical circuits in IE pathophysiology using advanced imaging and histopathological techniques.

A CASE REPORT OF SUSPECTED ACQUIRED NARCOLEPSY IN A DOG WITH STEROID RESPONSIVE MENINGITIS ARTERITIS.

Kaemper, G.[1], Stalin, C.[1], Gutierrez Quintana, R.[1], Kaczmarska, A.[1]

[1] *University of Glasgow Small Animal Hospital, School of Biodiversity, One Health and Veterinary Medicine, Glasgow, United Kingdom.*

No ethical approval was obtained for this study.

Narcolepsy is an uncommon sleep disorder characterised by an inability to regulate sleep-wake cycles. It features excessive daytime sleepiness, cataplexy, and abnormal REM sleep events such as sleep paralysis, sleep onset REM periods and hypnagogic hallucinations. However, this last phenomenon is only observed in humans. In veterinary patients, typical clinical signs include the sudden onset of REM sleep and generalized cataplexy, a pathognomonic peracute collapse caused by muscle atonia.

Congenital narcolepsy has been well described in Dachshunds, Dobermans and Labradors with a *canarc-1* gene mutation. A small number of case reports describe acquired narcolepsy in dogs secondary to infectious and immune-mediated encephalitides, other extra-neural inflammatory conditions, and pituitary macroadenoma. The objective of this case report was to review available narcolepsy literature and describe a clinical presentation of a case with suspected narcolepsy associated with steroid-responsive meningitis arteritis (SRMA).

A 12-month-old male entire Cavalier King Charles Spaniel cross presented with a two-month history of collapse episodes, characterised by acute flaccid collapse with loss of consciousness followed by up to 24-hours of hypersomnolence, occurring every 1-3 weeks. Episodes occurred at rest and play. Video recording provided by the owner showed the dog asleep and unresponsive with loss of neck muscle tone. Physical and neurological examination between episodes were normal. During an episode witnessed in the hospital the dog exhibited somnolence, a tendency to sleep when unstimulated, and partial cataplexy characterised by slight drooping of the head, eyelid closure, knee bending, truncal sway resulting in gradual collapse and brief sleep.

The dog had been diagnosed with SRMA two months prior based on cervical hyperaesthesia, pyrexia, inflammatory cerebrospinal fluid (CSF), and elevated C-reactive protein (CRP). The first collapse episode coincided with the onset of SRMA but was not investigated at the time. Investigations into hypersomnolence included haematology, biochemistry, echocardiography, magnetic resonance imaging (MRI), CSF cytology and hypocretin, and urine organic acids. No abnormalities were detected except for moderate methylmalonate aciduria suggesting suboptimal cobalamin status despite normal serum cobalamin levels. CSF was non-diagnostic for hypocretin due to insufficient sample volume. The dog was started on cobalamin supplementation and showed moderate improvement in clinical signs, with hypersomnolent episodes decreasing in frequency from once weekly to every 8-weeks.

Acquired narcolepsy has not been described with SRMA, but it has been linked to inflammatory conditions in dogs. An autoimmune co-aetiology is recognized in human narcolepsy and characterised by low hypocretin CSF levels due to hypothalamic neuronal destruction. Although CSF hypocretin was non-diagnostic in this case, we suspect that the dog suffers from acquired narcolepsy, secondary to SRMA or its immune trigger. The role of cobalamin is unclear, but cobalamin may alleviate neuroinflammation, potentially aiding recovery. On the other hand, most previously reported dogs with inflammatory associated narcolepsy recovered over time. Amongst these cases, only one had CSF hypocretin tested which was normal. Interestingly, in human narcolepsy, inflammation has been proposed as a neurodegenerative mechanism, which may explain the lack of spontaneous recovery. In contrast, recovery observed in dogs suggests a different pathomechanism of acquired narcolepsy.

DELAYED ONSET CONGENITAL NARCOLEPSY FOLLOWING ANAESTHESIA IN A DACHSHUND

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No ethical approval was obtained for this study.

Narcolepsy is an uncommon sleep disorder characterised by an inability to regulate sleep-wake cycles. It features excessive daytime sleepiness, cataplexy, and abnormal REM sleep events such as sleep paralysis, sleep onset REM periods and hypnagogic hallucinations. However, this last phenomenon is only observed in humans. In veterinary patients, typical clinical signs include the sudden onset of REM sleep and generalized cataplexy, a pathognomonic peracute collapse caused by muscle atonia. Cataplexy is commonly induced by emotional events such as feeding.

This report describes a case of suspected congenital narcolepsy in which clinical signs were induced by general anaesthesia. A 7-year-old male neutered wire-haired Dachshund presented for acute-on-chronic progressive non-ambulatory paraparesis and spinal pain. A T12-T13 intervertebral disc extrusion was diagnosed on magnetic resonance imaging and the dog was treated by means of a left T12-T13 hemilaminectomy. Whilst hospitalised post-operatively, the dog was noted to collapse when fed. In these episodes, there was flaccid atonia of skeletal muscles and apparent loss of consciousness, consistent with cataplexy. The dog recovered fully within 30 seconds, with no changes in mentation or muscle tone between episodes. The owner reported no prior history of collapse or any other episodes. Genetic testing for the *canarc-1* gene was performed (results pending).

Familial narcolepsy has been reported in Dachshunds, Labradors and Dobermans that are *canarc-1* positive. These dogs have a short interspersed nucleotide element (SINE) insertion variant in the *hypocretin receptor 2* (*Hcrtr2*) gene. The disease has an autosomal recessive inheritance with full penetrance. The onset of clinical signs is typically at birth or within a few months of age, with progression until one year of age, then gradual resolution by five years old.

Similar to this case, De Lahunta[1] reported cataplexy in several adult dachshunds following anaesthesia for hemilaminectomy. Clinical signs self-resolved after one week. Genetic testing was not performed, but a *canarc-1* gene variant was suspected that caused narcolepsy only after central nervous system exposure to general anaesthetics. Narcolepsy has also been induced experimentally in subclinical heterozygous Dobermans following exposure to medications that act on the cholinergic and monoaminergic systems. In humans, the most common subtype of narcolepsy is multifactorial, with clinical signs occurring only with a combination of the DQw6 gene variants and environmental factors. We posit that delayed onset narcolepsy in Dachshunds may represent either a clinical subtype of the *canarc-1* variant or a heterozygous genotype.

In conclusion, this case report provides further evidence of delayed onset congenital narcolepsy following general anaesthesia. To the authors' knowledge, it is the first described case to undergo genetic testing.

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SUSPECTED DISCOGENIC PAIN IN A SPRINGER SPANIEL DOG

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Discogenic pain is a recognised major component of low back pain in humans, often occurring independently of overt disc herniation due to inflammation, nerve sensitisation and biomechanical instability within the intervertebral disc (IVD). It has previously been considered unlikely in canine patients because of lack of innervation to their intervertebral discs. This case highlights the potential role of discogenic pain in intervertebral disc disease (IVDD).

A nine-year-old neutered male English springer spaniel presented with acute cervical pain and reluctance to exercise. Neurological examination revealed cervical hyperaesthesia without neurological deficits. Haematology and biochemistry were unremarkable. MRI showed mild, non-compressive disc protrusions at C2-3 and C3-4, deemed not clinically significant, along with degenerative changes affecting all cervical discs. Cerebrospinal fluid analysis was unremarkable. Synovial fluid analysis from the carpi and left stifle revealed a mild neutrophilic inflammation, raising suspicion for Immune-mediated polyarthritis (IMPA). The dog was initially hospitalised; however, due to good clinical response to analgesics and uncertainty surrounding the IMPA diagnosis, it was discharged on gabapentin (10 mg/kg PO TID), meloxicam (0.1 mg/kg PO SID), paracetamol (20 mg/kg PO TID), and amantadine (5mg/kg PO SID).

Despite treatment for suspected IMPA, the dog's condition worsened, progressing to ambulatory tetraparesis with postural reaction deficits in the pelvic limbs while the thoracic limbs remained normal. A 'two-engine' gait was also observed, localising to a C6-T2 myelopathy. Due to financial constraints, repeat MRI was not performed, and medical management was continued, with prednisolone (0.5mg/kg PO SID) with a view to increase the dose if infectious disease testing was negative. Repeat haematology and biochemistry were unremarkable, and serology for Neospora (IFAT 1:50) and Toxoplasma (IFAT IgM 1:20) were within normal reference ranges.

Due to poor clinical response, the dog was euthanised five days later, and a post-mortem examination was performed. This identified a Hansen type II disc protrusion at C6-7 and associated Wallerian degeneration in the spinal cord parenchyma, consistent with the neurological signs that developed but were not evident on the initial MRI. It is presumed that the C6-7 intervertebral disc was already degenerated at presentation, causing discogenic pain, and subsequently herniated, resulting in further deterioration and associated neurological changes. This finding aligns with the concept of discogenic pain, which is well-documented in humans. It is also possible that one of the other cervical discs identified as degenerate may have been the cause of the pain.

We believe this case report is important because it challenges the current understanding of pain associated with intervertebral disc disease, which has traditionally been considered to arise only from meningeal or nerve root inflammation, or from compression caused by disc herniation.

No formal ethical approval was required for this case report. The owner gave permission for the case to be used for teaching and research purposes, having been referred to the university teaching hospital.

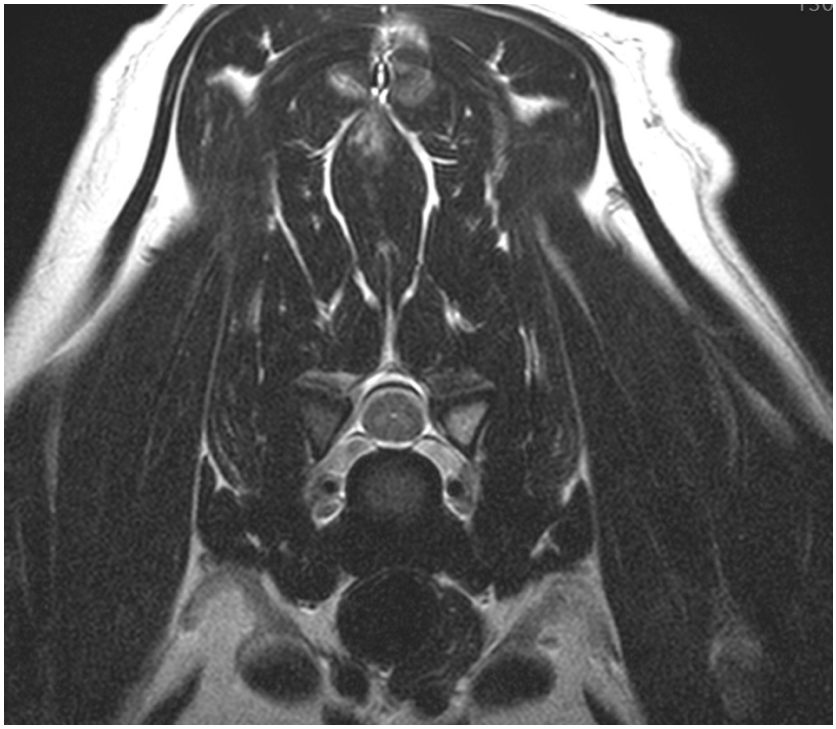

Figure 1: T2W transverse at the level of C6-C7

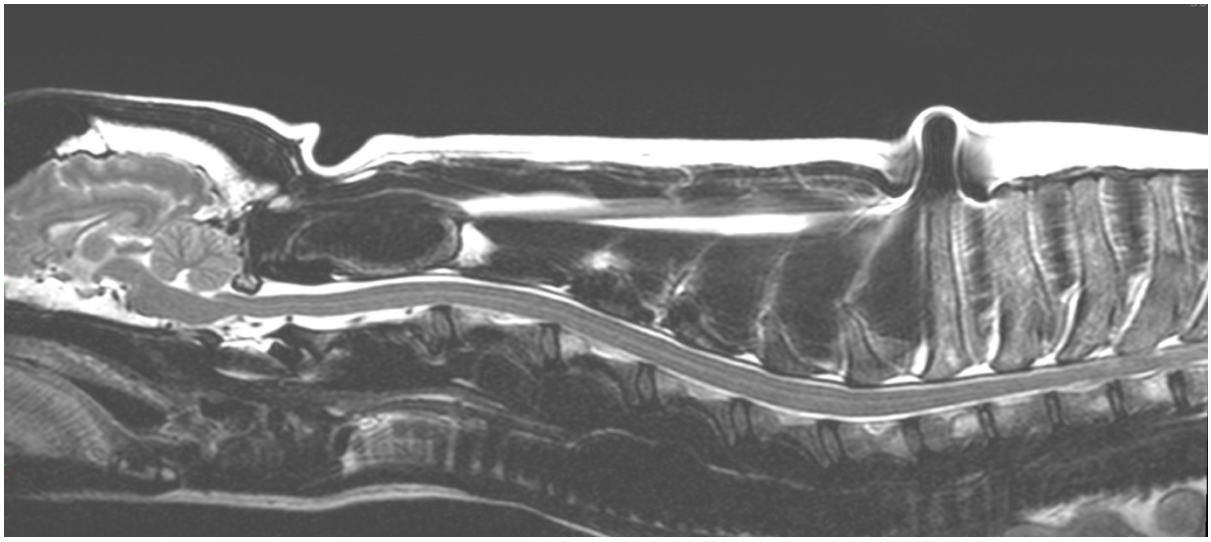

Figure 2: T2W sagittal of the cervical spine of the patient

VERTEBRAL FIBROUS DYSPLASIA IN A CAT

Irene Strelitzia Garcia Molina, Tom Shaw

Eastcott Veterinary Referrals

A 4-year-old female domestic shorthair presented to our hospital with a 6-month history of progressively worsening paraparesis. On examination, the cat was ambulatory paraparetic with moderate pelvic limb ataxia, reduced pelvic limb postural reactions (slightly worse on the right) and was equivocally reactive to thoracolumbar spine palpation. MRI and CT of the T3-L3 spine revealed a large osteolytic and osteoproliferative extradural vertebral mass lesion within the caudal lamina of L1, that was invading the vertebral canal and severely compressing the spinal cord. On MRI, the mass had well defined margins, was T1 isointense and T2 hypointense, STIR heterogeneously hyperintense, and markedly and homogeneously contrast enhancing. On CT the mass was homogeneously soft tissue attenuating on plain imaging and was markedly and homogeneously contrast enhancing. No other significant findings were made on contrast CT of the thorax and abdomen. The imaging report considered primary neoplastic disease such as plasma cell tumour, lymphoma, or giant cell tumour to be most likely. Fibrous dysplasia was not mentioned as a possibility.

After 1 month of medical treatment with prednisolone, surgical excision of the mass was performed. A dorsal approach and right-sided modified hemilaminectomy was performed at the level of L1-L2. A postoperative CT confirmed that all radiographically visible parts of the mass were excised. The histopathology performed revealed the nature of the mass to be fibrous dysplasia. Two months later, the cat had completely recovered, and no neurological deficits were found on examination. A repeat CT scan showed no evidence of regrowth of the mass.

Fibrous dysplasia is characterized by replacement of normal cancellous bone in the medullary canal with immature fibrous tissue. It is a rare condition, mainly seen in young animals, and it can be either monostotic or polyostotic. It can be challenging to diagnose based solely on imaging features as its appearance can vary from case to case. Although it has been previously described in the skull of cats and dogs, to our knowledge this is the first report in veterinary medicine of fibrous dysplasia in a cat vertebra.

No ethical approval was required for this case report.

SEVERE CONGENITAL HYDROCEPHALY AND SYRINGOMYELIA IN CREAM, LONG-HAIRED MINIATURE DACHSHUND PUPPIES.

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Retrospective case series - No ethical approval

Four cases of congenital hydrocephalus and syringomyelia were presented to two referral hospitals in south England in 2024 over the course of five months. All dogs were cream coloured, long-haired miniature Dachshunds aged 12-18 weeks with a domed shaped head. There were two males and two females. Two pups were from the same breeder, though from different litters. No relationship could be ascertained between them. The most common presenting complaints were dull mentation, ataxia in all four limbs, circling, failure to thrive and pain.

Cases 1, 2 and 3 were dull but ambulatory. Case 1 displayed a low-head carriage and severe neck pain. Cases 2 and 3 were ataxic with moderately delayed postural reactions in all limbs. Case 4 was stuporous and non-ambulatory tetraparetic, with absent left-sided postural reactions. All dogs had normal spinal reflexes. Variable deficits were noted on cranial nerve examination including absent menace response, delayed oculocephalic reflex and resting strabismus.

Magnetic resonance imaging in cases 1, 2 and 3 documented severe ventriculomegaly of the lateral, third and fourth ventricles, dilation of the mesencephalic aqueduct and central canal and a large cervical syrinx. There was also evidence of sub-occipital herniation in case 3. Computed tomography of case 4 documented similar changes of severe, diffuse dilation of the ventricular system and syringomyelia. These findings suggested a possible obstruction or absence of the lateral apertures.

In case 4, lack of response to medical therapy and lack of funds for surgery led to euthanasia. Surgery was performed in cases 1, 2 and 3 which, following intraventricular pressure measurement, consisted of placing a ventriculoperitoneal shunt (Medtronic® medium pressure valve or Surgiwear Chhabra® hydrocephaly system medium) and performing a sub-occipital craniectomy.

In dogs, absence of the foramen of Magendie, (a median communication between the caudal aspect of the fourth ventricle and the cisterna magna) is reported (Coben, 1967). In an attempt to restore CSF flow, sub-occipital craniectomy and incision of the caudal medullary velum was performed, creating an aperture for communication of the fourth ventricle and subarachnoid space, therefore bypassing the lateral apertures.

All three dogs recovered well from surgery and showed marked improvement which continues at 2, 4 and 7 months post-operatively.

Although we acknowledge that these cases may just be sporadic, this series could indicate a genetic basis for hydrocephalus and syringomyelia in the Dachshund breed, particularly cream, long-haired Dachshunds. Three miniature Dachshunds have been previously reported in the literature (Hasewaga et al., 2005 & Kitagawa et al., 2008). Some genes in humans are clearly linked to hydrocephaly, in particular the autosomal recessive MPDZ variant (Liu et al., 2024) which has been predicted to cause deleterious effects on protein function in nine vertebrate species including dogs (Rozman & Kunej, 2018). Elucidating a genetic basis of hydrocephaly may lead to a better understanding of the disease. We wish to alert colleagues to this severe form of congenital hydrocephaly and syringomyelia and call for similar cases that could form the basis of a genetic study.

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ACUTE GLOSSITIS SECONDARY TO A CAUDAL LINGUAL ABSCESS IN A DOG WITH MENINGOENCEPHALOMYELITIS OF UNKNOWN ORIGIN TREATED WITH IMMUNOMODULATORY THERAPY.

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A 7-year-old female French Bulldog presented with an acute onset of lethargy, dysphagia, and worsening right-sided head tilt with vestibular ataxia and rolling behaviour. The patient had been receiving treatment for meningoencephalomyelitis of unknown origin (MUO), including a 200 mg/m² cytarabine infusion over 8 hours and prednisolone (1 mg/kg twice daily), administered 12 days prior following advanced diagnostic investigations.

Physical examination revealed mild dehydration (5%) and a body temperature of 39°C. Neurological assessment was largely unchanged from the initial presentation, except for a reduced gag reflex. Blood tests, including haematology and biochemistry, were unremarkable.

The patient was hospitalised and started on fluid therapy, dexamethasone (0.14 mg/kg twice daily), and cyclosporine (5 mg/kg twice daily) was added for suspected deterioration of MUO. After 36 hours, the dog acutely developed severe glossitis, preventing tongue movement. Ultrasound of the mandibular region revealed a structure consistent with an abscess at the base of the tongue. Cytology and culture of a purulent sample confirmed *Pasteurella multocida* infection. Treatment was adjusted by initiating intravenous amoxicillin-clavulanate (20 mg/kg three times daily), discontinuing cyclosporine, and reducing prednisolone to 1 mg/kg once daily. The dog showed significant improvement, with resolution of glossitis within 48 hours. Antibiotic therapy was continued at home for 2 weeks with complete resolution of related signs.

Lingual abscesses are rarely reported in dogs and should be considered a differential diagnosis in cases of dysphagia followed by acute glossitis, particularly in immunosuppressed patients.

MONOCULAR NYSTAGMUS IN A DOG DIAGNOSED WITH PRESUMPTIVE MENINGOENCEPHALITIS OF UNKNOWN ORIGIN

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A 3-year-old, male neutered toy Chinese Crested powderpuff dog was presented with an acute onset obtundation that progressed to status epilepticus. On presentation, neurological examination was consistent with a right forebrain lesion. Bizarre episodes, consisting of disconjugate nystagmus of the left eye, medial strabismus of the right eye with convergent-retraction movements in both eyes, were recorded. Head magnetic resonance imaging revealed intra-axial multifocal lesions in the right fronto-temporal regions and dorsal paramedian thalamus. Cerebrospinal fluid analysis revealed a marked mononuclear pleocytosis. Electroencephalographic recordings shown right temporal recurrent medium-amplitude spikes. A presumptive diagnosis of meningoencephalitis of unknown origin was made. Despite treatment, the dog deceased.

This is the first report of epileptic monocular nystagmus in veterinary medicine, a rare clinical sign in human patients.

No ethical approval from a board was obtained due to nature of study (case report)

LONG-TERM OUTCOMES IN LGI1-AUTOANTIBODY ASSOCIATED FELINE AUTOIMMUNE ENCEPHALITIS: PRELIMINARY FINDINGS FROM AN OWNER QUESTIONNAIRE STUDY

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Autoimmune encephalitis (AE) associated with autoantibodies against the leucine-rich glioma inactivated 1 (LGI1) protein is increasingly recognised in domestic cats as a cause of seizures. Here, we describe preliminary results from a longitudinal study exploring the long-term outcomes of affected cats with the aim of correlating these against outcomes in human patients with this disease. This study was performed with Royal Veterinary College ethical approval (URN 2020 1957-2).

Questionnaires were distributed to English- and German-speaking** owners of cats that were positive for LGI1-autoantibodies on the Oxford LGI1 feline-specific live cell-based research assay. Questions assessed ongoing seizure frequency, behavioural outcomes, and quality-of-life (QoL) outcomes. Twenty owner-reported behaviours were mapped onto three key domains identified in human LGI1-AE long-term outcome studies: fatigue, cognition, and changes in mood or personality. QoL outcomes were assessed via three methods: an overall owner-assessed QoL rating, degree of impairment across five QoL domains (general demeanour, energy and mobility, interaction with their owner and the environment, physical appearance, and appetite), and reduction in QoL-associated behaviours.

Data from completed questionnaires for twenty cats were analysed. Eighteen cats were alive, with a median follow-up time of 15.5 months (range 3-55 months) from diagnosis. Two cats were euthanised due to AE 5 months and 35 months post-diagnosis. Nine of eighteen surviving cats were reported seizure-free with median follow-up of 21 months, compared to 6 months for the cats with ongoing seizures. Five cats remained on immunosuppressive treatment at follow-up, including ciclosporin (5/18), prednisolone (3/18), and cytarabine (1/18). Twelve cats were continuing to take at least one antiseizure medication, including phenobarbital (11/18), levetiracetam (5/18), imepitoin (1/18) and topiramate (1/18).

Fifteen of eighteen cats alive at follow-up displayed owner-reported behaviours correlating to impairment in at least one assessed outcome domain. Twelve displayed behaviours indicative of fatigue: increased lethargy (9/18), reduced playfulness (8/18) and weakness (5/18). Ten displayed behaviours suggestive of impaired cognition: difficulty navigating stairs or furniture (7/18), episodes of confusion (5/18), and going missing or getting lost (2/18). Ten were perceived to have had mood or personality changes compared to pre-diagnosis, including decreased affection (5/18), increased affection (4/18), irritability (4/18), aggression (3/18), and anxiety (3/18). Other reported changes included weight gain (9/18), increased vocalisation (9/18), increased appetite (8/18), changes to toileting habits (7/18) or sleeping habits (7/18), sleep disturbance (5/18) and reduced appetite (4/18).

Fourteen of eighteen cats alive at follow-up had owner-rated QoL of ‘good’ or ‘excellent’; the remaining four were rated as ‘acceptable’. Despite this, ten owners reported that their cat exhibited impairments correlating to at least one of five QoL domains: energy and mobility (6/18), general wellbeing (3/18), physical appearance (3/18), owner interaction (1/18), and appetite (1/18), while eleven cats reportedly showed reduction in at least one QoL-associated behaviour. Neither ongoing

seizures, nor any behavioural outcome measure, were associated with any QoL metric (Fisher's exact tests).

Further work is needed to characterise long-term outcomes and QoL in cats with LGI1-AE, with the aim to improve one-health understanding of this condition and contribute towards the development of evidence-based treatment protocols.

***translation of materials from English to German performed by Friederike Eldin MRCS MITI MIOL*

L7 TRANSVERSECTOMY FOR TREATMENT OF 'FAR-OUT SYNDROME' CAUSED BY A CONGENITAL VERTEBRAL BODY MALFORMATION IN A DOG.

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Eastcott Veterinary Referrals

A 2-year-old female spayed Cocker Spaniel was referred to our neurology service for evaluation of a 9-month history of right pelvic limb lameness, accompanied by reluctance to jump and climb stairs. On examination, there were no neurological deficits, but intermittent right pelvic limb lameness was observed during gait assessment, along with a bunny-hopping gait when ascending stairs. Palpation of the right lower paralumbar fossa elicited a painful response. Electrodiagnostic testing of the right tibial nerve indicated a proximal sciatic or L6-S1 neuropathy or radiculopathy. MRI and CT revealed a right sided, ventrally and laterally proliferative malformation of the L6-L7 vertebral bodies, causing extraforaminal compression of the right L6 spinal nerve against the L7 transverse process.

Surgery was performed to release the affected nerve. A right sided dorsolateral approach was made and an L7 transversectomy was performed at the junction with the vertebral body to expose the thickened and oedematous L6 spinal nerve. Postoperative CT confirmed complete removal of the proximal two thirds of the transverse process. The dog was neurologically normal at the time of discharge, and at the one-month follow-up appointment, the dog was normal on examination and the owners reported a complete resolution of clinical signs. At a two-month follow-up phone call, her owners reported no recurrence of clinical signs despite returning to normal exercise levels and performing activities such as vigorous running and jumping.

This case shares notable similarities with 'Far-Out Syndrome'; a condition in humans that is typically caused by degenerative osteophytosis in the lumbar spine, leading to compression of the L5 spinal nerve between the sacrum and the L5 transverse process, or occasionally, compression of a lumbar spinal nerve between a vertebral body osteophyte and the transverse process of an adjacent vertebra. To our knowledge, this is the first documented instance of the diagnosis and treatment of an extraforaminal osseous nerve compression in the lumbar spine of a dog.

No ethical review was required for this case report.

FEASIBILITY OF ACTICAL® ACCELEROMETERS FOR MONITORING SLEEP AND REST PERIODS IN INDIVIDUAL DOGS

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Monitoring of sleep and rest periods is often overlooked in clinical settings, yet it has the potential to provide valuable insights into the well-being and health of dogs. Accelerometers typically measure sleep efficiency of dogs, but the underlying proprietary algorithms are not disclosed. There is a need for a simple and reliable tool that can be used in clinical and research environment providing access to raw data.

The aim of this study was to evaluate a triaxial accelerometer which provides raw data to document sleep and rest periods in dogs. The study was conducted with ethical approval (133-13-07-2018). Ten privately owned dogs were fitted with a collar and a triaxial accelerometer (Actical®; Philips Respironics Inc., Pennsylvania, USA). In total, 32 sleep and resting periods were monitored by veterinarians or veterinary technicians while the dogs rested during the afternoon or evening in their home environment. Movements and behavioral state (apparent sleep, rest) were documented for each minute of the observation time including the time shortly before, during, and after the resting and sleep periods.

Results showed that the accelerometer effectively captured moderate and distinct movements of head and neck. Movements restricted to the paws or face did not interfere with the counts. In detail, the accelerometer documented the sleep and resting periods with a sensitivity of 94.0% and specificity of 96.1% but could not differentiate between sleep and rest with eyes open or closed.

We concluded that the device could be considered a useful clinical tool for documentation of sleep and rest periods and their interruptions in individual dogs. Further evaluation in long-term studies is warranted.