Organisation

Local Organising Committee:

Mette Berendt  (Chair)
Hanne Gredal  (Co-Chair)

Bodil Cathrine Koch
Maria Søndergaard Thøfner

31st Annual Symposium of the ESVN-ECVN

31st ESVN-ECVN Symposium in Copenhagen
20 - 22 September 2018

www.vetneuro2018.org
# Programme Overview

## Thursday 20 September

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Welcome

Dear participants,

Dear colleagues,

It is a great pleasure to welcome you to the 31st ECVN-ESVN Annual Symposium in Copenhagen.

During the symposium days, you will experience a diverse programme that includes plenary sessions, oral presentations, flash presentations and poster sessions. You will get a unique opportunity to meet experts within the field and get updated on the latest research happening across the globe.

Furthermore, we have organised some exciting social events, so you will get the chance to experience the cultural side of the city.

We hope you will enjoy the conference and your stay in Copenhagen!

Kind regards

Mette Berendt
Conference Chair

Hanne Gredal
Conference Co-Chair
## Detailed Programme

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| 08.30-09.15 | **Keynote:** The Plastic Brain in clinical practice  
Troels Wesenberg Kjaer |
| 09.15-10.00 | **Keynote:** Induced pluripotent stem cells for unraveling neurodegeneration in man and dog  
Poul Hyttel |
| 10.00-10.30 | Coffee break – Exhibition - Poster session  
In exhibition area |
| 10.30-11.40 | **Oral presentations**  
Chair: Thomas Flegel |
| O1 | Salbutamol sustains the clinical improvement of pyridostigmine in myasthenia  
An Vanhaesebrouck |
| O2 | Novel PCR assay using CSF for the diagnosis of intra-spinal Spirocercosis and BBB disruption  
Marco Ruggeri |
| O3 | Collection in EDTA versus plain tubes does not affect the cerebrospinal fluid analysis in dogs  
Bodil Cathrine Koch |
| O4 | Canine neuropathies associated with mutations in the N-myc downstream regulated gene 1  
Fredrik Skedsmo |
| O5 | Functional effect of olfactory ensheathing cells genetically modified to express chondroitinase abc on fore-paw reaching in rats after spinal cord injury  
Jon Prager |
| O6 | Congenital myopathy with phospholipid inclusions in European Shorthair cats  
Nina Meyerhoff |
| O7 | The prognostic role of postoperative magnetic resonance imaging in dogs with surgically treated intervertebral disc extrusion: a prospective study  
Federica Tirrito |
| 11.45-12.30 | **Keynote:** The competing nervous systems – the gut versus the brain, who is influencing whom?  
Holger Volk |
| 12.30-13.45 | Lunch - Exhibition - Poster session  
In exhibition area |
| 13.45-14.30 | **Keynote:** Personalized medicine in veterinary neurooncology - Where are we going?  
Peter Dickinson  
Chair: Holger Volk |
| 14.30-15.30 | **Flash presentations**  
Chair: Holger Volk |
| FP1 | Characterisation of early onset clinical deterioration in dogs following hemilaminectomy for thoracolumbar intervertebral disc extrusion  
Thomas Cardy |
| FP2 | An in vitro assay of canine olfactory ensheathing cell proliferation and migration on substrates inhibiting spinal cord regeneration  
Daisuke Ito |
| FP3 | A polysulfone tailor-made implant for the surgical correction of a frontoparietal meningoencephalocele in a cat  
Alba Farré Mariné |
| FP4 | Congenital lafora disease in the chihuahua breed | Daniel Sanchez Masian |
| FP5 | First clinical results regarding the use of a noninvasive intracranial pressure (ICP-NI) monitoring in dogs | Monica Vicky Bahr Arias |
| FP6 | Hippocampal expression of the CB1 receptor in canine epilepsy | Draginja Kostic |
| FP7 | Prognosis of non-ambulatory dogs with cervical intervertebral disc herniation after single versus multiple ventral slot decompression | Shanshan Guo |
| FP8 | Approaching phantom complex after limb amputation in cats: a preliminary study | Marika Menchetti |
| FP9 | Investigation of clinical, clinicopathological, magnetic resonance imaging findings, treatment and outcome in 14 dogs with idiopathic eosinophilic meningoencephalomyelitis (2009-2017) | Theophanes Liatis |
| FP10 | Clinical and electrodiagnostic findings and quality of life of dogs and cats with brachial plexus avulsion | Marika Menchetti |
| FP11 | Prevalence and clinical characteristics of phenobarbitone-associated adverse effects in epileptic cats | Giulia Corsini |
| FP12 | Inherited polyneuropathy in Border Collie: a motor and sensory polyneuropathy? | Hélène Vandenberghe |
| FP13 | Eosinophilic cerebrospinal fluid pleocytosis associated with neural Angiostrongylus vasorum infection in a dog | Emili Alcoverro Balart |
| FP14 | Venous sinus aneurysm in a Scottish Deerhound | Lucia Vicens |
| FP15 | Presence of Probst Fibres Suggest White Matter Remodelling in a Dog with Corpus Callosum Hypoplasia and Dysplasia | Katrin Beckmann |

15.30-16.00 **Coffee break - Exhibition - Poster session**

In exhibition area

16.00-17.45 **Annual General Meeting**

Thomas Flegel (President ECVN)

19.30-24.00 **GALA DINNER**

Jacobsen Brewhouse and Bar (Carlsberg)
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<td>Adriana Kaczmarska</td>
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<td>Epileptic seizures in dogs with meningitis of unknown origin</td>
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<td>Johanna Forsgård</td>
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<td>O10</td>
<td>Seizure-precipitating factors in dogs with idiopathic epilepsy</td>
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<td>Marjo Hytönen</td>
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<td>O11</td>
<td>Deletion in mitochondrial peptidase results in lethal juvenile brain disorder with status epilepticus in dogs</td>
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<td>Nova scotia duck tolling retrievers with degenerative encephalopathy are homozygous for a rare nonsynonymous rb1cc1 variant</td>
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<td>Development of a neurological examination protocol for harbor seal (Phoca vitulina richardii) pups</td>
<td>Christine Thomson</td>
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<td>HPA axis dysregulation associated with chronic idiopathic epilepsy in dogs</td>
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<td>Sofie Van Meervenne</td>
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<td>Detecting sacral nerve signals for artificial bladder control in spinal cord injury: a new cuff electrode design</td>
<td>Nicolas Granger</td>
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<td>Prevalence of congenital sensorineural deafness in a population of client-owned pure-breed kittens in the uk</td>
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<td>Unusual lactic acidosis in a 5 year old dog with glycogen storage myopathy due to muscle phosphorylase a deficiency</td>
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12.50-13.15 Presentation of ESVN-ECVN Annual Symposium 2019
Marcin Wrzosek, Poland

13.15-14.15 Lunch break – Exhibition - Poster session
In exhibition area

14.15-15.00 Keynote: Canine brain tumor treatment trials - are we lost in translation?
Simon Platt
Chair: Mette Berendt

15.00-15.45 Oral presentations
Chair: Mette Berendt

O16 Short peptide binding GM-CSF interferes with glioma-microglia environment and inhibits glioblastoma progression
Maria Pasierbińska

O17 The prognostic value of the Koret CT score of dogs with traumatic brain injury
Kira Rapoport

O18 Fractures of the second cervical vertebra in 66 dogs and 3 cats: a multicentric retrospective study
Fenella Davies

O19 Cerebrospinal fluid pulsation within canine spinal arachnoid diverticulae as measured on cine balanced fast field echo MRI
Sara Shivapour

15.45-16.15 Coffee break - Exhibition - Poster session
In exhibition area

16.15-17.15 Oral presentations
Chair: Mette Berendt

O20 Treatment of canine gliomas with systemic oncolytic virus delivered by allogeneic mesenchymal stem cells
Isidro Mateo

O21 Expression of the cysteine/glutamate transporter SLC7A11 in canine tumour associated epilepsy
Marco Rosati

O22 Canine gliomas immune evasion: immunohisto-chemical study of programmed death ligand 1 expression and tumour-infiltrating lymphocytes
Roberto Jose-Lopez

O23 Investigating the short-term effects of medium-chain triglycerides (MCT) supplement on canine epilepsy in drug-non responders
Benjamin-Andreas Berk

O24 Prognostic factors for recovery of vision in dogs with optic neuritis of unknown aetiology: 21 cases (2003 - 2018)
Christoforos Posporis

O25 Quantitative sensory testing after gabapentin with or without meloxicam in dogs with neuropathic pain
Helene Ruel

17.15-18.00 Awards & closing remarks
Thomas Flegel & Mette Berendt
Invited Speaker Abstracts
THE PLASTIC BRAIN IN CLINICAL PRACTICE

Troels Wesenberg Kjaer, MD, PhD, Chief Physician
Professor at the Department Clinical Medicine, University of Copenhagen, Denmark

Brain plasticity is pivotal to how we learn, develop and compensate for disease. It can be observed at several scales from regulation of biochemical pathways, to cortical reorganization with change of synapse and neuronal networks to change of behavior.

Brain plasticity may be secondary to injury (injury-dependent) or appear as a normal process caused by behavior, cognition or external stimuli (activity-dependent). The plastic processes play a major role in learning and memory (learning plasticity), in normal development (developmental plasticity) and recovery from brain damage (compensatory plasticity).

All of us use our brain every day. However, most of us do the same type of actions day after day. Do you remember to take regular steps out for your comfort zone? We are all aware of keeping our body fit, but do you remember to challenge the brain in new ways every day? A number of examples and exercises will be introduced.

There is evidence that early plasticity makes the brain more robust against neurodegenerative diseases like Alzheimer’s disease and other types of dementia. On the other hand, cortical plasticity is also harmful in other conditions like phantom limb pain and in dystonia. Thus, the usefulness of cortical plasticity depends on the clinical issues as well as types of task. For the clinician it is important to be aware of these phenomena in the new area where a fit brain is at least as important as a fit body.
Induced pluripotent stem cells (iPSCs) are somatic cells reprogrammed into an embryonic stem cell-like state, which are capable of differentiating into all cell types of the mammalian body. Patient-derived iPSCs hold great potentials for disease modeling as their differentiated progeny mimic the disease of the patient. In the frame of the stem cell center, BrainStem, we model human neurodegeneration with particular focus on Alzheimer’s disease (AD), Parkinson’s disease and frontotemporal dementia (FTD). Fibroblasts, grown from skin biopsies from the patients, are reprogrammed into iPSCs by a non-integrating plasmid-based technology and differentiated into neural cell subtypes that serve as in vitro cell models or “micro brains” expressing patient-specific disease phenotypes. In case of familial neurodegeneration, the disease-causing mutations are corrected by CRISPR/Cas9 gene editing creating isogenic control cell lines with exactly the same genomic background as the patient cells except for the disease-causing mutations. We have generated faithful in vitro cell models for FTD and AD, in which phenotypes related to dysfunction of mitochondria, endosomes and iron homeostasis have been documented. In dogs, a condition known as canine cognitive dysfunction exhibits similarities with human AD. In a recently initiated project, we are deriving iPSCs from dogs affected by CCD in order to compare the molecular disease phenotypes in neural in vitro cell models with such from human patients suffering from AD. At the moment, we are struggling to reprogram skin fibroblasts from the affected dogs as well as healthy control individuals into iPSCs.
THE COMPETING NERVOUS SYSTEMS – THE GUT VERSUS THE BRAIN, WHO IS INFLUENCING WHOM?

Holger A. Volk, DVM, PGCAP, PhD, DipECVN, FHEA, MRCVS

Professor of Veterinary Neurology and Neurosurgery, Royal Veterinary College, University of London, United Kingdom

Have you ever eaten something which made you feel different? And vice versa have you ever been stressed or felt anxious and realised that this did effect your gut function and your appetite on certain type of food? The brain-gut or gut-brain axis describes a bidirectional communication system between the gastrointestinal (GI) tract, the enteric and the central nervous system (CNS). Both systems are connected via the vagal nerve, but also communicate via the substances released into the blood stream. In recent years the microbiota has been shown to be one of the main players which can influence the gut-brain axis. Microbes release quorum-sensing molecules to communicate to each other, but also influence their environment. These molecules can be recognised by the cells of the host organism and have the potential to influence enteroendocrine cells, immune cells and nerve endings in the gut. Metabolites reported to be produced by gut microbiota who cannot only have an effect locally but also in the brain are bile acids metabolites, specific fatty acids, tryptophan pre-cursors and metabolites, serotonin, catecholamines and GABA. Serotonin and GABA are interesting molecules for neurologists which have been associated with effects on the GI tract (Gastric, pancreatic and intestinal secretion, GI motility and tone), Behaviour (visceral pain, emotion, stress response, appetite, addiction, sexuality) and CNS (Motor control, circadian rhythm, cerebellar regulation, body temperature, vascular tone). The kynurenine pathway which metabolises tryptophane is also important to consider as it influences NMDA response in the brain through the formation of kynurenic acid (KYNA) or quinolinic acid (QUIN). KYNA is an NMDA receptor antagonist at the glycine site, whereas QUIN is an NMDA agonist. These aforementioned neurotransmitters do play a central role in diseases such as epilepsy, cognition and neurobehavioural disorders and highlight the role microbiota play in physiology and disease.

The more obvious role of the GI tract is to ensure the body receives sufficient nutrients. The brain is dependent on a constant supply of glucose. The brain accounts only for 2% of the body weight of an adult human being but it uses up to 20% of total glucose-derived energy expenditure. Under physiological conditions, glucose utilisation is tightly correlated with neural activity, allowing imaging techniques such as positron emission tomography (PET) scans using radiolabeled glucose analogs to visualize brain activity. PET scanning techniques have been widely used to study diseases such as epilepsy, where an increase in seizure activity is associated with an increase in glucose metabolism. However, in the interictal period the epileptic focus is usually characterised by a glucose hypometabolism. PET scans of dogs with idiopathic epilepsy have shown areas in the brain characterised by interictal glucose hypometabolism which could indicate epileptic foci. Glucose hypome-
tabolism cannot only be associated with the epileptic focus but also have been suggested to contribute to functional deficits in cognition and behaviour in people with epilepsy. Apart from epilepsy, also normal ageing and cognitive dysfunction is associated with decrease in brain glucose uptake and metabolism. Glucose hypometabolism has been associated with mild cognitive dysfunction but also with canine cognitive dysfunction. In a study of ageing beagle dogs glucose brain metabolism was decreased by as much as 25% by the age of 6 years, which could be attributed to mitochondrial dysfunction. An alternating energy source is therefore needed to improve energy supply of the brain and mitochondrial function. Although it needs to be noted that other energy sources can only be seen as supplementation as glucose cannot be entirely replaced.

Ketogenic diets (KDs) have shown their efficiency in improving cognition and reducing seizure frequency in people with epilepsy and animal models of epilepsy. Ketone bodies (acetone, acetoacetate, and β-hydroxybutyrate) can support 60% of the brain’s energy requirements and have been shown to be increased in the brain of patients consuming a KD. Changing brain metabolism has been one explanation why KDs can improve brain function and seizure control. The original ketogenic diet, characterized by its high fat and low carbohydrate content, has been used for many years successfully in children with drug-resistant epilepsy, even allowing reduction or cessation of AED in some patients. The diet is also efficacious in adult patients but compliance to the traditional KD is poor due to the high fat and low carbohydrate content of the diet. The original human KD can induce ketosis in people, but not as easily in dogs. Its effect in dogs were therefore questionable. A traditional high fat low carbohydrate/protein KD failed to improve seizure control in dogs. A more promising KD is based on medium chain triglycerides (MCT) which improved seizure control in the majority of cases. MCTs have a high ketogenic yield which can improve brain metabolism. Furthermore, valproic acid, an AED, is an MCT and it is thought that its metabolites and other MCTs might have a similar antiepileptic effect. There is also now robust evidence that the MCT decanoic acid (capric acid; C10) has anti-seizure effects, with a recent ground-breaking study revealing its mechanism of action. Decanoic acid was found to be a non-competitive AMPA receptor antagonist at therapeutically relevant concentrations, in a voltage- and subunit-dependent manner, that results in direct inhibition of excitatory neurotransmission, and thus has an anticonvulsant effect. This is especially interesting, as most AEDs used in veterinary medicine work on increasing the function of the inhibitory brain pathways, which can also explain the side effects frequently seen such as sedation and ataxia. Decanoic acid has been shown to readily pass the blood brain barrier with 60-80% of its serum concentration arriving in the brain. Interestingly, in experimental seizure models in which the direct seizure reducing effect of decanoic acid has been shown to be effective, high concentration of acetone or beta-hydroxybutyrate have no effect. This could suggest that the effect on the AMPA receptor is the main mechanism of action for an MCT diet. Another interesting potential mechanism could be explained by decanoic acid regulating mitochondrial proliferation and therefore protecting against mitochondrial dysfunction, which can be seen with intensive seizure activity.

A MCT enriched diet was tested in a 6-month prospective, randomized, double-blinded, placebo controlled crossover dietary trial in chronically antiepileptic drug treated dogs with IE. The dogs were randomised to either start on the MCT or placebo diet and were switched over to the other diet after a 3-months period respectively. Seizure frequency, severity, physical and neurological examination findings, drug serum concentrations and clinical pathology data were recorded and analysed for all dogs with IE completing the study. The overall seizure frequency was significantly reduced by 13% on the MCT diet in
comparison to placebo diet; 71% of dogs showed a reduction in seizure frequency, 48% of dogs showed a 50% or greater reduction in seizure frequency and 14% of dogs achieved cessation of seizures. As many dogs experienced cluster seizures, the number of seizure days was also assessed which also significantly decreased on MCT diet. The MCT diet resulted in significant elevation of blood beta-hydroxybutyrate concentrations in comparison to the placebo diet. In addition to the demonstrated benefits of MCTs on seizure frequency, there are potentially beneficial effects on the behavioral comorbidities seen in canine epilepsy. A pilot study in children with autism showed an improvement in some of the social interaction, behavioral, and cognitive insufficiencies seen in these patients. In dogs, diets have been reported to modify certain types of behaviors, for example certain types of aggression can improve on a low protein diet. Interestingly, a similar MCT diet as used in the aforementioned epilepsy trial in dogs has previously been shown to support cognitive health of ageing dogs. The authors hypothesized that the improvement in cognitive function can be explained by the diet providing the aged brain with a more effective energy source. A significant reduction in chasing behaviour (a potential indicator of canine ADHD-like behaviour) was documented during the MCT diet period compared to the placebo diet phase, and a reduction in stranger-directed fear, which may indicate anxiolytic properties of the MCT. During the congress we will show that we were able to replicate the MCT study by using an MCT oil added to a dog’s diet (see presentation Berk et al., 2018). We have recently also reported that dogs with epilepsy have cognitive dysfunction, especially affecting their spatial memory. A similar diet has been shown to also improve cognitive function in dogs with canine cognitive dysfunction and it would be interesting to see how this comorbidity will respond to a dietary intervention.
References

PERSONALIZED MEDICINE IN VETERINARY NEUROONCOLOGY - WHERE ARE WE GOING?

Peter J. Dickinson, BVSc, PhD, Diplomate ACVIM (Neurology)

Professor of Neurology/Neurosurgery, UC Davis
Veterinary University School, United States

The detailed molecular genetic phenotyping of a vast array of human cancer types has paved the way for “personalised”, “precision” or “targeted” therapeutic interventions that have promised to revolutionise cancer medicine. The targeted tyrosine kinase inhibitor Imatinib (Gleevec) in “gene addicted” cancers such as BCR-ABL positive myelogenous leukaemia is the poster child of personalized medicine, and beneficial advances have been seen with a variety of targeted approaches for other cancers including BRAF/MEK +ve melanoma and EGFR +ve breast cancer. However, resistance and disease progression are generally the rule, and many authors have questioned the true value of “personalized” medicine with respect to overall outcome analysis, and the financial cost of individualized approaches. Targeting of networks rather than individual pathways is the rationale progression of personalized medicine, however combinations of drugs and pathways present additional issues including increased toxicity. Immunotherapy may be personalized in terms of utilizing patient-tumor specific “vaccine approaches” and also by defining tumor mutational load and tumor specific neoantigens, both of which have been associated with efficacy of the recently developed drugs targeting immune checkpoint inhibitors such as CTLA-4 and PD-1 amongst others.

In it’s purest sense personalized medicine is based on molecular genetic definition of an individual patient’s tumor in terms of cytogenetic, mutational, and epigenetic characteristics, followed by a tailored choice of an agent/agents based on these data. This approach is being offered at an increasing number of human oncology centers, and its feasibility has been demonstrated in veterinary oncology. At a broader level than this, the detailed molecular genetic profiling of large numbers of tumors, including gliomas, has allowed for the definition of discrete subsets of tumors with implications for therapy and prognosis that can subsequently be applied at a broader level based on defined abnormalities rather than genome wide assessment.

As “genomic/personalized” therapy develops and advances in human neurooncology, it will be even more critical that comprehensive characterization of canine brain tumors is established to define tumor subsets and allow rationale therapeutic targeting and selection of cases for translational investigations. Biologically and clinically relevant information relating to tumor behavior and patient prognosis are likely to be defined during this process as has been the case in human medicine.
Even in the era of the $1,000 genome, clinically/translationally relevant data will need to be generated from hundreds of canine tumors, likely necessitating a broad, multicenter approach to provide both the financial and sample generating resources required. The development of the NIH/NCI Comparative Brain Tumor Consortium (CBTC) is one example of a multicenter approach to try and address this issue.  

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THE GENETIC LANDSCAPE OF VETERINARY NEUROLOGY - IMPLICATIONS FOR THE FUTURE

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The list of inherited neurological disorders in companion and production animals is ever expanding. There are over 120 known genetic variants for neurological disorders in dogs alone, and with advances in molecular genetic technology and consistently decreasing costs, the list is continuing to expand at a rapid rate. Many of these disorders are associated with breed specific syndromes and have a relatively “localized” effects on the health of the overall dog population. The vast phenotypic diversity within domesticated dogs is the result of selection for genetic variants that define key traits such as skeletal size, body size, skull shape, snout length, coat color, leg length and other breed defining characteristics. Beyond the “desirable” morphological traits, undesirable “disease” syndromes may be associated with these genetic loci due to either multiple phenotypic sequelae of specific variants, or associated genetic variants carried within long regions of linkage disequilibrium. As causative variants are defined, the veterinary profession is facing ethical decisions where the primary mission of the profession (“...prevention and relief of animal suffering...”) may sometimes conflict with dog breeders’ phenotypic goals. This is most problematic when disease causing genes define key characteristics of the breed e.g. head shape and leg length resulting in the variant being essentially fixed in a majority if not all animals within certain breeds. However, many morphological characteristics have been shown to result from multiple genetic loci including body size, leg length and skull shape, that may allow for selective breeding against specific disease-causing genes with preservation of the general breed phenotypes. Skeletal dysplasia, chondrodystrophy and breed associated intervertebral disc disease in the context of breed defining leg length provides an example where such a scenario may exist.

Long term advancement of health within breeds harbouring morphological/disease associated variants may be achieved through careful selective breeding where segregation of alleles is present. Comprehensive evaluation of disease phenotypes and genetic associations is essential, particularly when less palatable recommendations for outbreeding may be necessary in specific cases where disease associated alleles are fixed within breeds.

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A number of preclinical rodent models have been developed in an effort to recapitulate injury mechanisms and identify potential therapeutics for traumatic brain injury (TBI), and stroke which are both major causes of death and long-term disability worldwide. However, the lack of restorative treatments for these brain injuries has led to considerable criticism of current preclinical therapeutic development strategies, namely the translatability of widely-used rodent models to human patients. The use of large animal models, such as the pig, with more comparable brain anatomy and physiology to humans may enhance the translational capacity of current preclinical animal models. Recent work has attempted to develop and characterize a graded pig TBI model with quantitative pathological features at the cellular, tissue, and functional level that become more prominent with increasing TBI severity. The success of this will be discussed in this session. The vast majority of cerebral stroke cases are caused by transient or permanent occlusion of a cerebral blood vessel (“ischemic stroke”) eventually leading to brain infarction. The final infarct size and the neurological outcome depend on a multitude of factors such as the duration and severity of ischemia, the existence of collateral systems and an adequate systemic blood pressure, aetiology and localization of the infarct, but also on age, sex, comorbidities with the respective multimedication and genetic background. Thus, ischemic stroke is a highly complex and heterogeneous disorder. It is immediately obvious that experimental models of stroke can cover only individual specific aspects of this multifaceted disease. A basic understanding of the principal molecular pathways induced by ischemia-like conditions comes already from in vitro studies. One of the most frequently used in vivo models in stroke research is the endovascular suture or filament model in rodents with occlusion of the middle cerebral artery (MCA) in large animal models, which causes reproducible infarcts in the MCA territory. Direct transient or permanent occlusion of cerebral arteries represents an alternative approach but requires craniectomy. Closeness to reality has its price and goes along with higher variability of infarct size and location as well as unpredictable stroke onset in spontaneous models versus unpredictable reperfusion in experimental models. These issues will be reviewed in this session.
Spontaneous animal models of cancer, such as dogs with brain tumours, offer an opportunity to apply a ‘comparative’ perspective with translational applications for new diagnostic, drug and other therapeutic trials. Several ‘companion’ animal species have successfully contributed to investigations of cancer biology and drug development, including cats, horses, ferrets and dogs. Dogs have major anatomical and physiological similarities to humans; additionally, a high prevalence of pet dogs diagnosed and managed with cancer each year, has turned attention on this species as a translational model. Dog owners are highly motivated to seek out new options for brain tumour treatment in their pets and are interested in participating in clinical trials when conventionally available treatments do not meet their goals. Again, such factors have contributed to the feasibility of utilizing pet dogs as pre-clinical animal models for the mutual benefit of veterinary and human medicine. Investigations of cancer in dogs have been pursued for over 40 years, but dogs have been under-utilized as a cancer model. In comparison to laboratory animals, dogs are more genetically out-bred and have an intact immune system. The canine genome provides evidence of strong similarities with humans, particularly with respect to the gene families associated with cancer, which are significantly closer than the relationship between a mouse and a human. These factors combined suggest that pet dogs with cancer can be a viable model for pre-clinical human cancer research, although the limitations of dogs in translational brain tumour research must also be understood in order to best address the needs of both veterinary and human medicine. Many of the naturally occurring canine brain tumours exhibit the same pathology, molecular and cytogenetic abnormalities, as well as neuro-imaging characteristics as their human counterparts. While chemo- and radio-sensitivity similarities are not as well documented in dogs as compared to humans, conventional radiation therapy and stereotactic radiosurgery (SRS) can prolong survival for a few months when compared to palliative treatment or surgery alone in both species, indicating that traditional treatments can likely be of use in dogs to serve as a control therapy in clinical trials. However, the lack of ‘gold standard’ treatments permits acceptable early testing of novel therapies, allowing pet dogs to be utilized ethically as a pre-clinical model. However, a major hurdle in advancing new diagnostic and treatment regimens for central nervous system (CNS) neoplasia is determining how much pre-clinical evidence is required before a clinical human trial is warranted. A significant part of this decision depends on how well data collected in ‘tumour bearing models’ such as dogs can help us predict what might happen in human beings. Unfortunately, many human CNS tumour treatment trials have failed because the pre-clinical animal models do not sufficiently parallel the human disease. The question we will address in this session is just how much the use of the dog as a clinical model will advance the successful investigation of novel therapies for human brain tumours?
Oral Abstracts
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[O1] SALBUTAMOL SUSTAINS THE CLINICAL IMPROVEMENT OF PYRIDOSTIGMINE IN MYASTHENIA

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Pyridostigmine is commonly used in myasthenia, but it looses its effectiveness over time. Salbutamol (β2-adrenergic agonist) is effective in a cohort of human patients with congenital myasthenia, namely in those with specific gene mutations resulting in disruption of postsynaptic structure.

We investigated whether adding salbutamol to pyridostigmine would prevent apparent loss in effectiveness of pyridostigmine.

The 12-week trial consisted of 4 groups of 10 mice with congenital myasthenia, receiving no treatment, pyridostigmine, salbutamol, or pyridostigmine to which salbutamol was added mid-trial.

Following an initial improvement in strength, a gradual decline in the effect of pyridostigmine was observed in mice treated with pyridostigmine alone. Salbutamol, as an add-on drug, significantly counteracted this decrease in strength. Decrement and endplate potential studies showed an improvement in mice treated with salbutamol and pyridostigmine, compared to pyridostigmine alone. Postsynaptic areas were smaller in pyridostigmine treated mice than in untreated mice. Salbutamol, as an add-on drug, counteracted this decrease in neuromuscular junction size. Muscle fiber diameters and fiber type compositions were similar between groups.

This study demonstrates that salbutamol can counteract the long-term loss in efficacy of pyridostigmine, by counteracting the disruption of postsynaptic structure induced by pyridostigmine. This study indicates that the β2-adrenergic receptor has a crucial role in stabilizing postsynaptic structure.
Intra-spinal *Spirocerca lupi* (ISSL) aberrant migration is an important cause of severe acute myelitis with high morbidity in endemic countries. Tentative diagnosis is based on characteristic clinical presentation and cerebrospinal fluid (CSF) analysis.

We used a high-resolution melt quantitative PCR for diagnosis of ISSL and looked for evidences of blood brain barrier (BBB) disruption around the migrating nematode through which Avermectins can enter the spinal cord parenchyma.

Ten CSF samples from 7 dogs with clinically suspected ISSL were examined (three dogs had both cervical and lumbar samples). The spinal cords of 2 of these dogs that died were prepared for histological and immunofluorocence staining of local and remote inflammatory changes: nematode tissue was harvested from paraffin embedded spinal cord segments and used to confirm the diagnosis and compare sequences to those of the CSF.

Four out of 10 CSF samples available, were positive (sensitivity 40%), and were confirmed by sequences of public available *S. lupi* DNA (specificity 100%). PCR of nematode tissue from the spinal cord parenchyma (n=2) were positive. Immunofluorocent staining revealed sever BBB disruption around the nematode but not in remote spinal cord segments.

Due to low sensitivity, CSF PCR can provide an important ante-mortem definitive positive diagnosis but cannot be used to rule out the disease.

Avermectins administered systemically are expected to diffuse through the spinal parenchyma around the nematode but not elsewhere in the cord. Therefore, early diagnosis followed by administration of the drug is recommended in order to kill the nematode and prevent further damages.

Ethical permission was obtained for this study.
O3] COLLECTION IN EDTA VERSUS PLAIN TUBES DOES NOT AFFECT THE CEREBROSPINAL FLUID ANALYSIS IN DOGS

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Cerebrospinal fluid (CSF) can be collected into EDTA or plain plastic tubes. The EDTA content presumably contributes to a better cell preservation, however, EDTA is reported to cause a false elevation in the total protein concentration and also dilute the CSF sample, thereby affecting the diagnostic interpretation. To the authors’ knowledge, no validated studies support this view. The aim of this study was therefore to determine if the choice of tube (EDTA or plain) influences the CSF analysis.

The study was completed at the University Hospital for Companion Animals, University of Copenhagen, and was approved by the Local Administrative and Ethics Committee. CSF samples were collected prospectively from dogs presenting for diagnostic purposes or dogs presenting for euthanasia for varying reasons. Paired samples (EDTA and plain) were obtained from the cerebellomedullary cistern. The CSF analysis was performed within 30 minutes of collection and included a macroscopic evaluation, semi-quantitative protein measurement, manual RBC and WBC counts, and a differential cell count (neutrophils, lymphocytes and monocytes).

32 paired samples were included in the study. There was no statistically significant difference in the semi-quantitative protein concentration when comparing EDTA and plain tube samples (P>0.999). The WBC count did not significantly differ between EDTA (median=2/µL, Q1-Q3=1-8/µL) and plain tube samples (median=2/µL, Q1-Q3=1-7/µL) (P=0.847). There were no significant differences in the differential cell counts between the two tubes.

According to our findings, the collection of CSF into EDTA tubes should not influence the result of the traditional CSF analysis.
[O4] CANINE NEUROPATHIES ASSOCIATED WITH MUTATIONS IN THE N-MYC DOWNSTREAM REGULATED GENE 1

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Mutations in the N-myc downstream regulated gene 1 (NDRG1) may cause polyneuropathy in humans, dogs and rodents. Additionally, NDRG1 is dysregulated in several cancers. A definite cellular role for NDRG1 remains to be established. Studies of NDRG1 by immunohistochemistry, immunofluorescence and western blot in tissues and cell cultures from neuropathic Alaskan malamute dogs and controls, as well as the ultrastructural nerve pathology of affected dogs, might provide information about the function of the protein and its role in the pathogenesis of both canine and human diseases. No ethical approval was necessary for this study.

In non-neuronal tissues from control dogs, NDRG1 is primarily found in epithelial cells, predominantly localized basolaterally. In canine peripheral nerves, NDRG1 is found in the cytoplasm of myelinating Schwann cells. High levels of phosphorylated NDRG1 appear in distinct sub-cellular localizations of the Schwann cells, suggesting signaling-driven re-routing of the protein. The ultrastructural changes in affected canine nerves are characterized by demyelination and accumulation of material in the adaxonal Schwann cell cytoplasm.

Overall, the tissue distribution of canine NDRG1 is similar to that of humans and rodents. Our data from peripheral nerves and primary Schwann cell cultures suggest that cell biology of NDRG1 is highly dynamic and probably influenced by signaling events leading to reversible phosphorylation of the protein. The pathologic similarities between this canine polyneuropathy and the human Charcot-Marie-Tooth type 4D suggest that further studies of canine tissues and cells might be valuable in the understanding of NDRG1-associated neuropathies, as well as cancers, in several species.
[05] FUNCTIONAL EFFECT OF OLFACTORY ENSHEATHING CELLS GENETICALLY MODIFIED TO EXPRESS CHONDROITINASE ABC ON FORE-PAW REACHING IN RATS AFTER SPINAL CORD INJURY

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Canine olfactory ensheathing cells (cOECs) and chondroitinase ABC (ChABC) have both, separately, improved walking ability after spinal cord injury (SCI) in clinical trials in companion dogs. However, variable and unpredictable responses are seen. We previously reported that cOECs can be genetically modified to express ChABC; in this study we tested if this modification provides functional benefit.

We used a crush lesion to the dorsal corticospinal tracts at C3 in rats, which results in fore-paw grasping deficits. Immediately after injury we transplanted either: (i) cOECs expressing ChABC (cOEC-ChABC), (ii) cOECs expressing green fluorescent protein (cOEC-GFP), or (iii) media control. All experiments were performed under Home Office regulations. Primary outcome measure was fore-paw pellet retrieval on ‘staircase’ behaviour apparatus. Secondary outcome measures included accuracy of pellet retrieval on Whishaw reaching task, and number of foot slips crossing a horizontal ladder. Surgery, transplant and behavioural testing were performed blinded, and animals were randomised.

Rats transplanted with cOEC-ChABC showed a trend towards retrieving an increased number of pellets, and increased accuracy of pellet retrieval, on staircase testing over 8 weeks and had a significantly higher accuracy of pellet retrieval at 6 weeks after injury on Whishaw reaching task (p<0.01) compared to cOEC-GFP and media transplanted animals. No difference was seen between cOEC-GFP and media transplanted animals. No difference was seen between any groups on ladder testing.

These results suggest that cOEC-ChABC may improve accuracy of fore-paw reaching in rats after SCI. cOEC-ChABC may therefore be a functionally relevant novel delivery method of ChABC.
[O6] CONGENITAL MYOPATHY WITH PHOSPHOLIPID INCLUSIONS IN EUROPEAN SHORTHAIR CATS

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This case series describes the diagnostic work up of a so far unknown myopathy in a family of European Shorthair cats.

A female cat and her 8 month old kitten (two females and one male) were presented with chronic non-progressive, not exercise-related gait abnormalities. Clinical examination revealed a waxing and waning mildly stiff stilted gait with intermittent hypermetria, mild ataxia and intention tremor. A muscular or diffuse white matter disease was suspected.

The blood work-up was unremarkable except for mildly increased lactate levels in two cats. MRI of brain and spinal cord as well as cerebrospinal fluid of all cats were unremarkable. Diffusely mild to moderate pathologic spontaneous activity occurred in electromyographic examinations. Motor and sensory nerve conduction studies were normal in peroneal, fibular, ulnar and radial nerves, but repetitive motor nerve stimulation with 1 and 2 Hz revealed a decrement of up to 73%. Brainstem auditory evoked responses showed physiological wave I, III, V latencies. Muscle biopsies revealed mild myofiber diameter changes and diffusely increased nuclear internalization, mitochondrial prominence and clustered myofibres with decreased COX and NADH-TR reactivities. On electron microscopy, all cats displayed conspicuous sarcoplasmic phospholipid membrane inclusions. Levels of organic acids in urine, and aminoacids and acylcarnitin in serum were comparable to healthy age matched control cats.

Phospholipid inclusions have been identified in various human disorders such as mucolipidosis type IV. As the mother and her entire litter have been affected, an inherited myopathy seems most likely. Search for associated mutations using whole genome sequencing is underway.
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[07] THE PROGNOSTIC ROLE OF POSTOPERATIVE MAGNETIC RESONANCE IMAGING IN DOGS WITH SURGICALLY TREATED INTERVERTEBRAL DISC EXTRUSION. A PROSPECTIVE STUDY

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Decompressive surgery is a common treatment for intervertebral disc (IVD) herniation in dogs.

Inadequate spinal decompression is a reported cause of neurological signs recurrence. The utility of postoperative imaging has been poorly investigated in thoracolumbar surgeries, while no studies have been conducted in cervical IVD herniation.

The purpose of this study was to assess the accuracy of surgeons’ perception in determining the efficacy of spinal decompression compared to postoperative magnetic resonance imaging (MRI) findings in a population of dogs with IVD extrusion; to evaluate whether postoperative MRI findings had a better association with outcome or recovery time as compared to surgeons’ evaluation; to investigate the relationship of MRI intramedullary changes with outcome and recovery time.

A prospective study was conducted in 68 dogs with cervical (13) or thoracolumbar (55) IVD extrusion; medical records, pre- and postoperative MRI findings, surgery reports, outcome and recovery time were collected.

The agreement between surgeons’ evaluation and postoperative MRI findings regarding the efficacy of spinal decompression was fair. Severe preoperative spinal compression was associated with an increased chance of inadequate decompression.

Postoperative MRI findings of adequate spinal decompression were associated with successful outcome and reduced recovery time, while intraoperative evaluation was associated only with recovery time.

Intramedullary T2-weighted hyperintensity in preoperative (17.6%) and postoperative (33.8%) MRI scans was not associated with outcome and recovery time.

Results support the use of postoperative MRI in patients with IVD herniation to objectively evaluate the degree of spinal decompression obtained and better predict the outcome.
EPILEPTIC SEIZURE FREQUENCY AND PATTERN IN IDIOPATHIC EPILEPTIC DOGS AFTER INITIATION OF PHENOBARBITAL OR IMEPITOIN MONOTHERAPY.

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The aim of this study was to evaluate changes in epileptic seizure (ES) frequency and pattern in antiepileptic-drug (AED)-naïve dogs with idiopathic epilepsy (IE) after initiation of phenobarbital or imepitoin monotherapy.

This is an observational prospective cohort study. Inclusion criteria were: TIER II diagnosis-confidence-level of IE according to IVETF-criteria in AED-naïve dogs and a detailed ES-diary. Exclusion criteria were: occurrence of cluster seizures (CS) or status epilepticus prior to treatment initiation and concurrent disease and/or treatment.

Thirty-one dogs were started on imepitoin 10-20mg/kg/12h and 30 dogs were started on phenobarbital 2.5-3.3mg/kg/12h. AED dosage was increased overtime (up-to imepitoin 30mg/kg/12h and phenobarbital 5.2mg/kg/12h). All dogs experienced generalised-tonic-clonic-seizures. In the imepitoin group: pre-treatment median-ES-frequency was 1.50 ES/m (range 1-4 ES/m); post-treatment median-ES-frequency was 0.95 ES/m (range 1 ES/6m-3 ES/m); 21/31 dogs (67.7%) developed CS 1-18m after treatment initiation, 7/31 dogs (22.6%) developed unacceptable side effects in the first month of treatment which required switching to an alternative AED, 3/31 dogs (9.7%) did not develop CS with a 3 year follow-up. In the phenobarbital group: pre-treatment median-ES-frequency was 2.46 ES/m (range 1-7 ES/m); post-treatment median-ES-frequency was 0.36 ES/m (range 0 ES/3years-1 ES/m); 11/30 dogs (36.7%) developed CS 12-25m after treatment initiation. Nineteen/30 dogs (63.3%) did not develop CS with a 3 year follow-up, 3 of these were seizure free.

CS developed in a greater proportion of dogs on imepitoin than on phenobarbitone. SE did not develop in any dogs.
[O9] EPILEPTIC SEIZURES IN DOGS WITH MENINGOENCEPHALITIS OF UNKNOWN ORIGIN: CLINICAL FEATURES, RISK FACTORS AND LONGTERM OUTCOME.

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Patients with meningoencephalitis can experience seizures acutely, but also have an increased risk of developing unprovoked seizures following resolution of the acute phase of the illness. The aim of this retrospective study was to report the prevalence of epileptic seizures in dogs with meningoencephalitis of unknown origin (MUO), and to define their risk factors and prognosis.

The clinical records and magnetic resonance imaging (MRI) studies of dogs diagnosed with MUO were reviewed. Dogs with prior neurologic insult or history of seizures were excluded. Seizures were classified as early symptomatic (ESS) if they occurred within 3 months of the acute episode of meningoencephalitis. Post-encephalitic epilepsy (PE) was defined as recurrent seizures following that initial period with no other clinical signs of possible relapse.

75 dogs met the inclusion criteria, among them 40 had ESS and 10 PE. Three dogs developed drug resistant epilepsy. Dogs with ESS were younger, more frequently presented with abnormal mental status and had significantly shorter survival times. They also showed cortical involvement (in particular temporal and parietal lobes), features of increased intracranial pressure and meningeal contrast enhancement more frequently on MRI when compared with dogs without seizures. On multivariate regression analysis the factors associated with PE were presence of ESS (p=0.034) and MRI lesions in hippocampus (p = 0.014) and temporal lobe (p = 0.026).

These results demonstrate that dogs with MUO and ESS are at higher risk of PE. MRI findings may provide valuable prognostic information and could help determine which patients require long-term antiepileptic treatment.
Seizure-precipitating factors have not been thoroughly studied in epileptic dogs, although they are frequently reported in human epilepsy patients. In humans, common seizure-precipitating factors include for example stress, sleep deprivation and infectious diseases. We hypothesized that seizure-precipitating factors are common in dogs with idiopathic epilepsy, and the occurrence of these factors associate with the patients’ signalment, personality, and epilepsy-related factors. Furthermore, we hypothesized that most dogs with idiopathic epilepsy have protective factors for seizures.

In this cross-sectional observational study, we collected 50 dogs with idiopathic epilepsy from the hospital populations of the University Veterinary Teaching Hospital of University of Helsinki and privately-owned Referral Animal Hospital Aisti. The owners were interviewed about their dogs’ possible seizure-precipitating factors and protective factors according to a predefined questionnaire.

In our study population the prevalence of seizure-precipitating factors was 74% and the most frequently reported factors included stress-related situations, sleep deprivation, weather, and hormonal factors. Almost all of the studied dogs had both precipitated and unprecipitated seizures. Most commonly the seizure occurred within 24 h of exposure to the precipitating factor. In dogs with focal onset seizures, the number of precipitating factors was 1.9 times higher compared to dogs with generalized seizures. Additionally, 60% of owners recognized at least one protective factor for seizures.

In conclusion, seizure-precipitating factors are common in dogs with idiopathic epilepsy and the nature of these factors is consistent with those reported among human patients. By acknowledging and avoiding patients’ seizure-precipitating factors, veterinarians could achieve better treatment outcomes.

**[O10] SEIZURE-PRECIPITATING FACTORS IN DOGS WITH IDIOPATHIC EPILEPSY**

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We investigated a canine brain disorder with epileptic seizures beginning at 6 to 12 weeks of age. After onset, the disease progresses rapidly to status epilepticus and death.

Detailed clinical investigations and necropsy were carried out and tissues collected for pathology and functional studies. DNA samples were obtained from 7 affected dogs and ~400 unaffected dogs by owner’s consent and with ethical permission. Homozygosity mapping and exome sequencing was performed to identify the genetic defect. Candidate variants were validated by Sanger sequencing and TaqMan assay.

We discovered a novel rapidly progressing recessive juvenile mitochondrial encephalopathy resulting in lethal status epilepticus. Pathology showed a diffuse necrotizing panpolioencephalopathy with extensive neuronal mitochondrial crowding in two of the affected dogs. Genetic analyses revealed a recessive in-frame 6-bp deletion in a gene coding for a mitochondrial peptidase.

We have characterized a novel canine brain disorder, juvenile mitochondrial encephalopathy with status epilepticus, and identified the genetic cause. The gene codes for a mitochondrial matrix peptidase, that degrades mitochondrial targeting sequences cleaved from imported proteins. Mutations in this gene have been associated with a slowly progressing neurodegeneration in humans. In contrast to the human disease, the canine condition is rapidly progressing after birth leading to status epilepticus and death. Further functional and pathological studies aim to uncover pathophysiological details of the disease and functions of the identified gene.
TIME COURSE AND PROGNOSTIC VALUE OF SERUM GFAP, PNF-H AND S100BETA CONCENTRATIONS IN DOGS WITH COMPLETE SPINAL CORD INJURY

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Prediction of outcome following acute, severe spinal cord injury in dogs is difficult due to variable recovery. Serum biomarkers show promise as prognostic tools but their concentrations change over time. The purpose of this study was to describe the time course of serum concentrations of glial (glial fibrillary acidic protein, GFAP, and S100β) and neuronal (phosphorylated neurofilament heavy chain, pNF-H) biomarkers after severe spinal cord injury in dogs. We hypothesized that combinations of these biomarkers would predict outcome in deep pain negative (DPN) dogs with surgically treated thoracolumbar intervertebral disc extrusion (TL-IVDE).

Paraplegic, DPN dogs with surgically treated TL-IVDE were recruited, blood samples were banked at presentation, and on days 1-3, 14, 28 and 56. Outcome was established at 6 months. Serum levels of GFAP, S100β and pNF-H were measured with ELISA tests. The association between outcome and serum levels was examined using regression tree and ROC curve analysis.

Thirty-one dogs were included, 19/31 (62%) recovered independent ambulation by 6 months. GFAP and S100β concentrations peaked within 3 days and rapidly decreased. PNF-H peaked at 14 days and was still detectable at 8 weeks. A GFAP concentration of <0.31ng/mL within 72 hours of onset of paralysis predicted outcome with an accuracy of 0.84.

Severe spinal cord injury causes ongoing axonal degeneration that produces prolonged elevation of serum pNF-H while astrocytic injury resolves rapidly. Serum GFAP at time of presentation is a useful prognostic indicator for spinal cord injury and a rapid bedside test for this protein would be clinically valuable.
[O13] EVALUATION OF A PREOPERATIVE IMPLANT PLACEMENT PLANNING METHOD FOR CANINE ATLANTOAXIAL STABILISATION

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Canine atlantoaxial stabilisation is a challenging procedure associated with relatively high mortality rate. Yet, little data are available on implant placement accuracy obtained with these surgeries. The aims of this retrospective study were to 1- describe a practical method of preoperative implant placement planning and 2- report implant placement results in a group of affected dogs.

Dogs included in the study were treated with ventral cemented constructs using 2 cortical screws in the atlas and 3 in the axis. Intended implant positions and relevant anatomical landmarks were preoperatively identified using computed tomography (CT) images. Postoperative CT images were also obtained in all cases, allowing to grade screw positions based on the amount and location of breach outside the intended bone corridor.

Nine dogs met the inclusion criteria for a total of 45 implants positioned. Immediately after surgery, all constructs were considered unlikely to impair neurovascular structures. Upon retrospective review, 35 screws were graded as optimal, 7 protruded outside the intended bone corridor but not within the vertebral canal (VC) and 3 actually caused VC violation. The cranial articular surface of the axis had the highest rate of optimal implant placement (17/18). Monocortical screws placed in the sagittal plane of the axis often penetrated C2-3 intervertebral disc space (6/9). VC violation occurred most commonly when aiming for atlantal lateral masses (2/18).

These results suggest the proposed method can achieve acceptable implant positioning although further precautions may be beneficial to improve the procedure accuracy, especially when targeting atlantal lateral masses.
NOVA SCOTIA DUCK TOLLING RETRIEVERS WITH DEGENERATIVE ENCEPHALOPATHY ARE HOMOZYGOUS FOR A RARE NONSYNONYMOUS RB1CC1 VARIANT

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The purpose of this study was to identify the genetic variant responsible for an autosomal recessively inherited progressive degenerative encephalopathy that affects Nova Scotia Duck Tolling Retriever (NSDTR) dogs. The clinical characteristics of this disease include degeneration of the caudate nuclei and development of a REM sleep behavior disorder (Barker, 2016), features that distinguish the NSDTR disease from previously described canine encephalopathies.

A 20-fold average coverage whole genome sequence (WGS) of an affected NSDTR generated using Illumina NextGen sequencing technology was aligned to CanFam 3.1. Variant alleles were filtered for those that were: (1) predicted to alter the primary structure of the gene product, (2) homozygous in the affected NSDTR dog, and (3) absent from the WGSs of 105 dogs with other disease phenotypes. Fifteen variant alleles met these criteria, including a C>T transition at 29:4451005 which was homozygous in all 10 NSDTR with a confirmed diagnosis that were available to us at the time. This transition predicted a p.Gly1503Arg change in the encoded RB1CC1 protein (FIP200). FIP200 is involved in a number of cellular pathways including the induction of autophagy. We were unable to demonstrate major changes in autophagy markers in cultured fibroblasts from affected NSDTR. Thus, the p.Gly1503Arg amino acid substitution may affect one of the other pathways in which FIP200 participates such as apoptosis.

We have now genotyped 1767 privately owned NSDTR for 29:4451005C>T. The frequency of the variant 29:4451005T allele in this tested cohort is 15%.
[O15] DEVELOPMENT OF A NEUROLOGICAL EXAMINATION PROTOCOL FOR HARBOR SEAL (PHOCA VITULINA RICHARDII) PUPS

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Neurological examination (NE) protocols are well developed for different terrestrial vertebrates. However many NE tests are not appropriate for free-ranging pinnipeds, i.e. animals anatomically and behaviorally adapted to aquatic locomotion.

The Marine Mammal Center (Sausalito, California) admits an average of 88 stranded, harbor seal pups (HSP) each year for rehabilitation. Neurological diseases in HSP are an increasing concern; observational scoring and MRI of brain lesions have been reported. Mercury-induced developmental anomalies are possible as excessive mercury levels have been identified in harbor seals from California. We aimed to develop a NE protocol that could identify and localize neurological abnormalities, including those associated with mercury exposure.

Neurological examinations were performed in one week periods in 2017 (24 NE on 20 HSP – four repeats) and 2018 (40 NE – 20 HSP, each animal was examined twice). In 2017, we developed a draft protocol that included tests specific for mercury-induced anomalies recorded in other mammals; this protocol was optimized in 2018.

We had to develop new tests to assess neurological function in this species. New tests include: incline plane and handstand (proprioception and motor function), dripline (cutaneous sensation), grasp and flipper fan (spinal reflexes and limb strength), and fish head (visual field assessment). Utilizing this NE protocol, three animals with neurological deficits were identified (hydrocephalus, unilateral porencephaly, cerebellar hypoplasia); their neuroanatomical localization was confirmed by MRI and necropsy.

By observing normal neurological function in this marine-adapted animal, a species-specific NE was developed that will continue to evolve and be adaptable for other aquatic quadrupeds.
SHORT PEPTIDE BINDING GM-CSF INTERFERES WITH GLIOMA-MICROGLIA ENVIRONMENT AND INHIBITS GliOBlastoma PROGRESSION.

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Glioblastoma (WHO grade IV, GBM) is a malignant, primary brain tumor which despite many years of research remains incurable. Tumor microenvironment plays an important role in growth, metastasis and response to treatment. Glioma cells overexpress and secrete protein - granulocyte macrophage colony-stimulating factor (GM-CSF) that reprogram microglia accumulated in GBM into cells which potentiate tumor invasion and growth and suppress antitumor immunity.

The aim of the study was to design and identify a humanized peptide that selectively binds to GM-CSF, blocks its binding to respective receptors on microglia and inhibits activation of the receptors and downstream signaling pathways resulting in inhibition of glioma invasiveness.

We identified peptide binding GM-CSF using peptide microarrays, enzyme-linked immunosorbent assay (ELISA) and a technique based on surface plasmon resonance (SPR). Subsequently, we selected peptide (G7) with most potent capacity for inhibition of human glioma cell invasiveness in the presence of human and mouse microglia cell line using the Matrigel Matrix invasion assay. We confirmed that this peptide blocks binding of GM-CSF to its receptor using methods based on SPR and Ligand Tracer technology. Antitumor activity of G7 in vivo was confirmed in orthotopic xenograft mouse model using in vivo fluorescence imaging system and microscopic histological measurements.

We designed and identified G7 peptide which binds GM-CSF and blocks GM-CSF binding to its receptor. G7 has a potent capacity for inhibition of glioma cell invasiveness induced by the presence of microglia and exhibits antitumor activity in vivo. Study was approved by the Local Ethics Committee.
[O17] THE PROGNOSTIC VALUE OF THE KORET CT SCORE OF DOGS WITH TRAUMATIC BRAIN INJURY

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Traumatic brain injury (TBI) is common in dogs and can be life-threatening. However, determining the prognosis upon presentation is difficult. Recently, we reported the development of a scoring system, dubbed KCTS, which may aid in predicting prognosis in dogs following head trauma. This 0-7 point scale is based on abnormal head CT findings. The aim of the current study was to validate the prognostic value of this score.

The study included dogs admitted to the Koret School Veterinary Teaching Hospital with TBI within 72 hours of injury that underwent CT examination of the head. Obtained data included signalment, medical history, physical and neurologic examination findings and short-term outcome (up to 14 days). All CT scans were reviewed by neurologist and radiologist blinded to the clinical status and outcome; then, KCTS was calculated for each dog.

Fourteen dogs met the inclusion criteria, of which 12 (85%) survived. CT scans revealed cranial vault fractures, parenchymal abnormalities, or both in 10/14 dogs (71%). ROC analysis for prediction of survival by the KCTS yielded an area under the curve of 0.896 (95% confidence interval, 0.726-1). A KCTS score of <3 points was associated with survival with 83% sensitivity and 100% specificity.

These preliminary results show the high potential of the KCTS to predict prognosis in dogs with TBI. Further testing with larger cohorts is needed to increase the statistical power of the results.
In human medicine, fractures of the second cervical vertebra have been studied elaborately and categorized in detail, which has not been done so far in veterinary medicine.

It was the aim of this multi-institutional retrospective case series to describe clinical features of 66 dogs and three cats with a fractured axis and to assess potential predictors for the type of axis fracture and for functional motor recovery.

In the dog population, crossbreeds and Labrador Retrievers were the most represented. Mean age was 2.75 years. Motor vehicle accident was the most common inciting cause, followed by frontal collision (running into an object). Neurological symptoms ranged from mere cervical pain with or without mild ataxia (n=22), to tetraparesis (n=28) and tetraplegia (n=11). Neither weight nor age, type of trauma, length of the axis (cm), or degree of clinical symptoms had an impact on the type of axis fracture seen. Concerning treatment, 54% of patients underwent surgical fracture stabilisation, 39% received conservative therapy and 7% were immediately euthanized. Functional motor recovery was achieved in 90% of all treated cases. Due to the low number of cases lacking functional motor recovery, no reliable predictive factors for outcome were detectable.

Fractures of the axis most commonly occur in young dogs. In many cases, neurological symptoms are relatively mild. Generally, animals with a fractured axis have a very good prognosis for functional motor recovery, irrespective of whether they undergo surgery or conservative treatment. The risk of perioperative mortality seems to be much lower than previously reported.
Oral Abstracts

[O19] CEREBROSPINAL FLUID PULSATION WITHIN CANINE SPINAL ARACHNOID DIVERTICULAE AS MEASURED ON CINE BALANCED FAST FIELD ECHO MRI

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Canine spinal arachnoid diverticulae (SAD) are characterized by focal cerebrospinal fluid dilatations within the subarachnoid space, commonly resulting in nonpainful paresis and ataxia secondary to chronic compressive myelopathy. Numerous imaging techniques have been described in SAD diagnosis, including myelography, computed tomography myelography, and magnetic resonance imaging (MRI). The present retrospective study investigated the utility of cine balanced fast field echo (bFFE) MRI sequences in measuring pulsatile flow within twelve canine SAD. A secondary aim included characterization of the prevalence and location of syringes in relation to the SAD, as the co-occurrence of these conditions has not been previously reported.

The degree of pulsatile flow within each SAD was calculated as the change in area per cardiac cycle on sagittal (12/12) and transverse (7/12) cardiac-gated cine bFFE MRI sequences. Syringes were identified as any intra-parenchymal fluid-filled lesion >2mm width on T2W transverse sequence, and classified as rostral or caudal to each SAD when present.

Pulsation was identified on all cine sequences, with a median ratio of change in SAD area from minimum to maximum of 0.14 (Range 0.10 – 0.27; N = 12) on sagittal cine bFFE and 0.22 (Range 0.05 – 0.53; N = 7) on transverse cine bFFE. A high prevalence of syringomyelia was identified (11/12; 92%) on T2W sequences, occurring both rostral (5/12; 42%) and caudal (7/12; 58%) to the SAD. Results support the ability of cine bFFE sequences to identify abnormal CSF pulsation within SAD, and identify a high co-occurrence of SAD and syringomyelia in the study population.

Study submitted for ethical approval to SSRERB at authors’ affiliated institution.
The purpose of the study is to describe the clinical presentation, disease progression, imaging and pathological findings in 10 dogs with single prosencephalic oligodendrogliomas treated with weekly systemic infusions of allogeneic mesenchymal stem cells (MSC) infected with a canine oncolytic adenovirus (ICOCAV17), designed to selectively replicate in tumoral cells, due to MSC homing capacity towards the tumour.

All dogs had sudden onset of CNS signs, primarily seizures or behavioural changes that led to MRI evaluation which revealed a single prosencephalic intra-axial mass with classical radiological features of glioma. Tumor size was measured using transverse post-gadolinium T1W image sequences (or transverse T2W images when the lesion did not enhance). MRI was repeated in the 7 patients alive after 8 weeks of treatment, which revealed a decrease in tumour size in 5 of them. Nevertheless according to the response assessment in veterinary neuro-oncology (RAVNO) system, the disease was considered progressive in 2 patients, stable in 3 and partially responsive in 2 of them. All dogs died or were euthanized at the owner’s request due to disease progression with an overall median survival time (MST) of 116 days (range 43 – 199 days) since beginning of treatment and of 144 days (range 47 – 239 days) since beginning of clinical signs. All tumours were classified as high grade (anaplastic oligodendroglioma grade III or grade IV glioblastoma multiforme). All of them showed moderate to severe peri- or intra-tumoural inflammatory infiltrates.

Treatment with MSC infected with oncolytic viruses produces an inflammatory response associated with tumour. MST was not considered superior to other treatment modalities.
Oral Abstracts

[O21] EXPRESSION OF THE CYSTINE/GLUTAMATE TRANSPORTER SLC7A11 IN CANINE TUMOUR ASSOCIATED EPILEPSY

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Tumor-associated epilepsy (TAE) is a common neurological sequela in glioma patients. The pathogenesis of TAE is poorly understood but recently neurotoxic concentrations of glutamate, released by tumor cells, have been advocated as an epileptogenic key factor. The cystine/glutamate transporter SLC7A11 is considered a major pathway of glioma glutamate release. As it can be pharmacologically targeted, we investigated its expression and its association to TAE in a population of dogs affected by neuroglial tumors.

Altogether, 24 glioma patients with (15/24) or without TAE (9/24) were included in the study. SLC7A11 expression was visualized immunohistologically and semiquantitatively rated within (1) the tumour, (2) the peritumoural and (3) remote brain parenchyma using standard algorithms.

SLC7A11 was successfully traced in all brains. Intratumoural expression was clearly higher in oligodendroglioma than astrocytoma but it did not correlate to seizure prevalence or tumour grade. In epileptic dogs, however, the peritumoural and/or remote expression fraction was significantly higher than in non-epileptic glioma patients.

Canine TAE brains show an increased SLC7A11 expression in the perilesional and remote brain parenchyma. It may therefore be a valuable target for tailored TAE treatment. Being a surrogate of hyperexcitability, it furthermore substantiates the concept of colocalizing and distant ictal-onset zones. The hypothesis requires to be further addresses by coregistration studies implementing glutamate spectroscopy.
Human gliomas appear to exploit immune inhibitory mechanisms to maintain an immunosuppressive microenvironment and evade immune eradication. Binding of programmed cell death protein 1 (PD-1) to its ligand (PD-L1) promotes activated T-cell exhaustion and apoptosis. Tumour cells (TCs) evade host’s immune attack by expressing PD-L1 and stimulating PD-1 expression on tumour-infiltrating lymphocytes (TILs). High-grade and astrocytic gliomas express higher levels of PD-L1 and immunotherapeutic targeting of the PD-1-PD-L1 pathway is intensely studied; however, clinical evidence supporting these strategies’ efficacy is lacking.

Little is known about whether canine gliomas share this immunomodulating pathway. We analysed immunohistochemical expression of PD-L1 and PD-1 in a retrospective series of canine gliomas.

Formalin-fixed paraffin-embedded tissue sections of 18 gliomas including four grade IV glioblastomas, six grade III anaplastic oligodendrogliomas and two each of grade III anaplastic astrocytoma, oligoastrocytoma, and grade II diffuse astrocytoma and oligodendroglioma were immunostained for PD-L1, PD-1, CD3, and CD20.

All tumours contained PD-L1-expressing cells (>1% positive-TCs), mainly in the periphery and infiltration zone of the tumour. Mean expression increased with grade and was considerably higher in astrocytic tumours. Intratumoural and/or infiltration zone PD-1+ TILs were observed in 16 cases; however, only three tumours with high PD-L1 expression and CD3+ TIL density were considered positive for PD-1 (>1% positive-TILs). All high-grade gliomas contained PD-1+ TCs.

This is the first study to immunohistochemically characterise the PD-1-PD-L1 axis in canine gliomas, demonstrating that spontaneous canine gliomas model immune evasion in its human counterpart. This supports the use of canine gliomas in translational human studies.
O23] INVESTIGATING THE SHORT-TERM EFFECTS OF MEDIUM-CHAIN TRIGLYCERIDES (MCT) SUPPLEMENT ON CANINE EPILEPSY IN DRUG-NON RESPONDERS

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The influence of diets have been studied extensively in human epilepsy, however, there is a lack of comparative data in veterinary medicine. It has been shown that a medium-chain triglyceride (MCT) enriched diet has a positive effect on seizure control and behaviour in dogs with idiopathic epilepsy. However, it is unknown whether MCTs administered in the form of an add-on dietary supplement (DS) to a variable base diet will show similar positive effects.

A 6-month multi-centre, prospective, randomised, double-blinded, placebo-controlled cross-over trial was completed, comparing a MCT-DS with a standardised placebo-DS in a population of dogs with idiopathic epilepsy, chronically treated with antiepileptic drugs and reaching tier-2 diagnostic certainty. A 9%-MCT or placebo oil was added to the dogs’ diet for three months, followed by the alternative oil for a further three months. Twenty-eight dogs completed this study and were included in further analysis. Seizure frequency was significantly lower when dogs were fed MCT-DS (median 2.51/month, 0–6.67/month) in comparison with the placebo-DS (2.67/month, 0–10.45/month; \( P=0.0147 \)). Seizure day frequency was also significantly lower during MCT-DS phase (1.68/month, 0–5.60/month vs. 1.99/month, 0–7.42/month, \( P=0.0101 \)). Two dogs achieved seizure-freedom, three additional dogs had ≥50% reduction and 12 had <50% reduction in seizure frequency. Eight dogs showed no response or a slight increase (8%).

In summary, these data show antiepileptic properties associated with MCT-DS when compared to a placebo and support former evidence for the efficacy of the MCTs as a nutritive, therapeutic option for a subpopulation of drug-non responsive epileptic dogs.

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Optic neuritis of unknown aetiology (ONUA) is an accepted condition, yet factors influencing recovery of vision are currently unknown. The aim of this study was to investigate prognostic factors for recovery of vision in dogs with ONUA.

Medical records of dogs diagnosed with isolated ONUA or with meningoencephalitis of unknown aetiology (MUA)-associated ON were reviewed. Statistical analysis investigated the association between recovery of vision and the duration of blindness prior to immunosuppression, presence/absence of PLR, presence/absence of menace response, and CSF results. Datasets were tested for normality using the Shapiro-Wilk test. All data was non-parametric. The datasets were compared using the Mann-Whitney and the Kruskall Wallis test.

Twenty-one dogs (10 male, 11 female) with a mean age of 4.14 years (range 0.58-11) met the inclusion criteria. Follow-up period ranged from 33 to 1685 days. Eight dogs (38%) achieved complete recovery of vision, 4 (19%) did not recover and 9 (43%) improved partially. Fifteen dogs (71%) were diagnosed with MUA-associated ON and 6 (29%) were diagnosed with isolated ONUA. Presence/absence of PLR (p<0.048) and CSF protein concentration (p<0.011) showed a significant association with complete recovery of vision.

Our study suggests that presence/absence of PLR and CSF protein concentration may influence complete recovery of vision in dogs with ONUA. A larger cohort of dogs is required to determine whether our findings are robust and whether any additional measurable parameters enable accurate prognostication for recovery of vision in dogs with ONUA.
[O25] QUANTITATIVE SENSORY TESTING AFTER GABAPENTIN WITH OR WITHOUT MELOXICAM IN DOGS WITH NEUROPATHIC PAIN

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This study evaluated the effects of gabapentin alone (G) or in combination with meloxicam (GM) on electrical (ENT) and mechanical (MNT) nociceptive threshold in dogs with neuropathic pain.

Thirty-one client-owned dogs [6.7 (0.6-12.9 years); 26.9 ± 18 kg (22 males/9 females)] with naturally occurring neuropathic pain were included. Diagnosis was confirmed via magnetic resonance imaging. Dogs were randomly allocated to receive medical treatment with G/placebo/GM or GM/placebo/G. Duration of each treatment was 7 days and included resting. The ENT and MNT testing were performed on initial presentation (day 0), days 7, 14 and 21. Stimulation was applied to the dorsal aspect of the metacarpus and the plantar aspect of the metatarsus in a randomized order until a behavior response was observed or a cut-off reached. For ENT, transcutaneous electrical stimulation (TENS) was provided using two adhesive electrodes. For MNT, an increasing pressure was applied with an algometer. Tests were performed twice (60 seconds apart). Statistical analysis included mixed linear model (p < 0.05).

Mean ± SEM ENT (mA) was significantly higher on initial presentation (49 ± 3.2) than after G (38 ± 3.3; p = 0.008) and GM (40 ± 3.3; p = 0.03) but not placebo (42 ± 3.3; p = 0.09). Mean ± SEM MNT (N) was not significantly different after any treatment [10.1 ± 0.5 (initial presentation); 10.2 ± 0.5 (G); 10.1± 0.5 (GM); 10 ± 0.5 (placebo).

Changes in ENT showed that gabapentin with or without meloxicam improved somatosensory function in dogs with neuropathic pain.
Poster Flash Abstracts
Poster Flash Abstracts

[FP1] CHARACTERISATION OF EARLY ONSET CLINICAL DETERIORATION IN DOGS FOLLOWING HEMILAMINECTOMY FOR THORACOLUMBAR INTERVERTEBRAL DISC EXTRUSION

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Surgical treatment for canine intervertebral disc extrusion (IVDE) commonly involves hemilaminectomy to decompress the spinal cord. Early onset clinical deterioration (EOCD) occurs in some dogs after surgery. A retrospective study was performed to characterize EOCD causes within 90 days of hemilaminectomy and evaluate outcomes following a second surgery. Included dogs had complete medical and MRI records and repeated neurological assessments. EOCD was categorized as Group 1: 0-7 days, Group 2: 8-30 days and Group 3: 31-90 days.

Thirty dogs were included (Group 1: n=13, Group 2: n=8, Group 3: n=9). EOCD occurred in 2.0% of hemilaminectomies in the study period (2010-2017). Four causes of EOCD were identified: further extrusion of nucleus pulposus at original IVDE site (n=15), extrusion of an adjacent disc (n=6), haematoma at original surgical site (n=5) or extrusion of an unrelated disc (n=4).

Dogs in Group 1 were heavier (p=0.02, OR: 6.5) and more often had EOCD due to haematoma (p=0.017, OR:16.4). Dogs in Group 3 more often had extrusion of an adjacent disc (p=0.02, OR:3.4) and a quicker time to discharge (3.3 days) compared to Group 1 (6.8 days, p=0.049) or Group 2 (7.6 days, p=0.03). At discharge following second surgery there was no significant difference in median neurological grade between groups (Group 1: 4, Group 2: 4, Group 3: 3).

Preliminary results suggest that EOCD is an uncommon complication with a limited number of causes. Prognosis for recovery following second surgery appears good in this small cohort of dogs, although further research is required.
[FP2] AN INVITRO ASSAY OF CANINE OLFACTORY ENSHEATHING CELL PROLIFERATION AND MIGRATION ON SUBSTRATES INHIBITING SPINAL CORD REGENERATION

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Chondroitinase ABC (ChABC) has improved walking after spinal cord injury (SCI) in a clinical trial in companion dogs. ChABC digests the chondroitin sulphate proteoglycans (CSPGs) found in the glial scar that forms after SCI, and are inhibitory to axonal growth. Olfactory ensheathing cells (OECs) given in combination with ChABC has shown improved locomotor outcomes in experimental animals. We have recently demonstrated that canine OECs (cOECs) can be genetically modified to express ChABC (cOEC-ChABC). To characterise any synergistic interaction between ChABC and cOECs, we designed an in vitro assay of cOEC proliferation and migration.

We used an IncuCyte Zoom Live Cell Imaging System (Essen Bioscience) to take time-lapse images of cOEC-ChABC and cOECs expressing green fluorescent protein (cOEC-GFP) in culture on three concentrations of CSPGs. Using ImageJ, we automatically quantified cell number at each time-point and manually tracked cOEC migration in each condition. IncuCyte ‘NeuroTrak’ software was used to quantify cOEC process length at each time-point. We demonstrated a significantly increased cell number and process length of cOEC-ChABCs compared to cOEC-GFPs on all concentrations of CSPGs, but no difference when cultured on poly-L-lysine (PLL; control, non-inhibitory substrate). Preliminary investigation of cOEC migration suggests a higher distance and speed of migration for cOEC-ChABC compared to cOEC-GFP on CSPG but not PLL.

This assay has shown an interaction between cOECs and ChABC in vitro, suggesting changes to cOEC proliferation and migration with ChABC that may impact on their function when transplanted in vivo, and will allow us to explore mechanisms of this interaction.
A 6-week-old entire female domestic shorthair cat was presented for evaluation of a soft bulge and a palpable skull defect on the frontal area of the head since adoption. The neurological examination revealed absent menace response bilaterally and apparent blindness localizing the lesion to the occipital cortex. Main differential diagnoses were meningocele/meningoencephalocele (MC/MEC). Surgical repair was proposed once the cat reached the adult size. Meanwhile, the patient developed seizures and treated with anticonvulsant therapy. When 8-month-old a CT confirmed a frontoparietal MEC with associated porencephaly. Based on a 3D printed skull mold, a polysulphone implant was created. The meninges were dissected from the skin, a durectomy was performed and samples of the protruding brain tissue were obtained. Part of the cerebrospinal fluid was drained until the size of the protruding brain decreased enough to be included below the implant that was anchored with cerclages within predrilled holes. The histopathology confirmed the presence of aberrant meningeal and neural tissue. Three months later, the cat is partially blind, but otherwise neurologically normal and seizure free since the surgery.

There are few reports of MEC in dogs and cats, and only two of surgical management never using a polysulfone tailor-made implant. Although a recent study suggested that medical management could be an option for dogs with MC/MEC and mild neurological signs, in human medicine, surgical intervention is the treatment of choice. This case highlights a new implant option for surgical correction of MEC with good results.
Lafora disease (LD) is a fatal neurodegenerative disorder caused by abnormal glycogen metabolism. Clinical signs are similar in both humans and dogs, including spontaneous myoclonic epilepsy, hypnic jerks and generalised tonic-clonic epileptic seizures. LD is most frequently diagnosed in the Miniature Wirehaired Dachshunds, Basset Hound and Beagles. Single cases have been reported in the miniature and Standard Poodle, Pointer and Corgi. The aim of this study is to describe the clinical presentation and causative defect in one affected Chihuahua dog.

A seven year old, male, castrated Chihuahua presented for further investigations of acute onset generalised tonic-clonic epileptic seizures with a progressive increase in frequency. Additionally, the dog exhibited frequent sudden head and body movements characterised by myoclonic jerks which could be triggered by bright light, loud noises and by approaching towards his head. Comprehensive blood tests (haematology, biochemistry, electrolytes and BAST) revealed no significant abnormalities. Magnetic resonance imaging of the brain and cerebrospinal fluid analysis were largely unremarkable.

As per previous studies, LD genetic testing was performed by Southern blotting and membrane pre-hybridisation specific for the specific DNA fragment to EPMB2. A population of six Chihuahua dogs were also genetically tested and used as a control group. All blood samples were obtained in accordance to the University of Liverpool Committee on Research Ethics (VREC589). Genetic testing revealed one homozygous/affected case and within the control group three carriers and three cases with wild type alleles. This is the first description of genetically confirmed Lafora disease in a Chihuahua dog.
[FP5] FIRST CLINICAL RESULTS REGARDING THE USE OF A NONINVASIVE INTRACRANIAL PRESSURE (ICP-NI) MONITORING IN DOGS

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Invasive and noninvasive intracranial pressure (ICP) measurements are rarely done in dogs, due to complications and limitations. A new ICP-NI monitoring method developed by Braincare Inc. (Brazil) has been used in research with rats and humans with good results. It uses a sensor that detect changes induced by intracranial pressure waves. Our objective is to describe the first results with this new device in dogs.

ICP pulse was monitored using Braincare BcMM/2000 monitor. The device was initially tested in three normal anesthetized dogs submitted to procedures not related to the study, to verify the location where the sensor better captured the waves, which was defined as the parietal region, with dogs in lateral recumbence. The waveform had three characteristic peaks, meaning that the cerebral complacency was normal. Monitoring was then performed in three dogs with central nervous system disorders. In a puppy with hydrocephalus ICP was monitored before and after withdrawal of cerebrospinal fluid by ventriculocentesis. In the second patient, with spinal affection, the ICP was monitored before, during and after injection of contrast in the subarachnoid space for myelography. In a patient with brain injury, ICP was monitored after application of mannitol and one hour later. In all cases, it was possible to observe that the P2 peak was higher than the P1 peak at times of ICP increase, indicating decreased brain complacency.

This novel ICP-NI method is capable of monitoring dynamics of ICP in dogs, but more studies are needed to implement the technique in clinical practice.
**[FP6] HIPPOCAMPAL EXPRESSION OF THE CB1 RECEPTOR IN CANINE EPILEPSY**

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In epilepsy, the endocannabinoid system could be involved in the pathomechanism through the cannabinoid receptor type 1 (CB1). The CB1 receptor is known to control neuronal activity on the synaptic level by negative feedback mechanism. Endocannabinoids are elevated in cerebrospinal fluid in epileptic dogs. Therefore, the aim of this study was to describe CB1 receptors in epileptic patients and quantitatively compare them to healthy dogs.

CB1 receptor expression was evaluated by immunohistochemistry of the brains of seven controls, seven dogs with structural and five with idiopathic epilepsy (CB1, Abcam ab23703). Hippocampal density and intensity of CB1 receptor expression were analyzed in cornu ammonis (CA) 1, CA3, dentate gyrus (DG) and hilus using ImageJ software (U.S. National Institutes of Health).

CB1 receptor expression was confirmed in the hippocampus of all dogs. In CA1 the area expressing CB1 receptor was smaller in patients with idiopathic as compared to patients with structural epilepsy (p<0.0001) or healthy dogs (p<0.001). In the same region, intensity of the CB1 receptor staining was stronger in dogs with idiopathic than structural epilepsy (p<0.001). Statistically significant group differences with smaller area and higher intensity of CB1 receptor expression in idiopathic epilepsy dogs in comparison to structural epilepsy occurred in DG, CA3 and hilus.

Our results give evidence for a downregulation of CB1 receptor expression in idiopathic canine epilepsy in sub-regions of the hippocampus. The disease seems to cause redistribution of CB1 receptors proving that the endocannabinoid system is a major component of epileptogenesis which could enable therapeutic approach.
[FP7] PROGNOSIS OF NON-AMBULATORY DOGS WITH CERVICAL INTERVERTEBRAL DISC HERNIATION AFTER SINGLE VERSUS MULTIPLE VENTRAL SLOT DECOMPRESSION

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No previous study has been conducted to compare the outcome in dogs which required single (SVSD) versus multiple ventral slot decompression (MVSD) as a treatment of cervical intervertebral disc herniation.

Medical records of 185 dogs, which had undergone single or multiple VSD based on clinical signs (non-ambulatory tetraparesis/plegia) and MRI results between January 2010 and September 2016 at a single veterinary practice were reviewed. Data about signalment, preoperative neurological status, intervertebral disc(s) involved, outcome (good: ambulatory, fair: ambulatory but with perceivable neurological deficit, poor: non-ambulatory or perioperative death), time to recovery (TTR), and neurological status at the ‘last seen’ date were collected.

59.5% of all dogs experienced a good outcome, 36.7% had a fair and 3.8% a poor outcome. Median TTR was 3 days.

66.5% of dogs required SVSD and 33.5% MVSD. There was a significant difference between those two groups in age (SVSD: M=112.6 months and MVSD: M=133.2 months; t-test, p<0.001) and breed size (χ² test, p=0.006), small breeds were more likely to require MSVD than medium or large breeds. No significant differences were found for sex, neuter status, outcome (χ² tests: p=0.158, p=0.573, and p=0.816, respectively) and TTR (Mann-Whitney-U, p=0.166).

Controlling for age and breed, belonging to the SVSD or MSVD group was not associated with the outcome.

We concluded that, although the two groups differed in age and breed size, the chances of a full recovery were not affected by the need for single or multiple VSD, and that MVSD is as safe and effective as SVSD.
Phantom Complex (PC) is a human multifaceted syndrome that includes stump pain, phantom limb sensation and phantom limb pain. Early description of behavioural changes following limb amputation has been reported in cats, but there are no data on time-related onset and semiology of pain manifestations. The aim of the study was to record behavioural changes, signs of discomfort, their onset, duration and overall QoL in a population of owned cats after limb amputation.

Owners completed a survey comprising 71 questions evaluating medical conditions, pain and manifestations related to PC before and after amputation and QoL (ethical approval ID 664/2016).

27 cat owners completed the survey. The main reason for amputation was trauma (63%) followed by cancer (29%). In 45% of the patients, pain was reported before and after amputation. Out of these, 91% of cats started experiencing pain 24 hours-1 week after the amputation. 18% of owners perceived the cat was in pain for more than 1 year after amputation. On the whole population, 85% of the owners noticed behavioural changes possibly related to pain/d after the amputation. In 39% of cats those signs lasted for more than 1 year, while 17% started showing these signs between 1 month and 1 year after amputation.

Signs related to PC are common in amputated cats and the first week after amputation requires special care for pain monitoring and treatment. Delayed onset of PC likewise warrant greater awareness and specific owner education. Investigation of larger group is however necessary to confirm preliminary results.

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Idiopathic eosinophilic meningoencephalomyelitis (IEME) is a rare form of idiopathic meningoencephalomyelitis in human, dog and cat, characterised by CSF eosinophilic pleocytosis in the absence of infectious agents.

This is a retrospective study of 14 dogs with EME during 2009-2017. Inclusion criteria consisted of cases with full records (including MRI), CSF eosinophilic pleocytosis and negative infectious tests.

Fourteen dogs were included; males (10/14), females (4/14); small (7/14), medium (4/14), large (3/14) breeds. Median age was 3.7 years (range 0.5 – 12 years). Depression (7/14) and ataxia (5/14) were the main presenting complaints. Neurological examination revealed spinal hyperaesthesia (7/14), ataxia (7/14), cranial nerve (6/14) and proprioceptive (5/14) deficits. Neuroanatomical localisation was multifocal/diffuse (7/14), forebrain (4/14), facial & vestibulocochlear nerves (1/14), T3-L3 (1/14), L4-S3 (1/14). Peripheral eosinophilia was present in 4/14. MRI revealed ill-defined patchy T2 hyperintensities in: forebrain (6/14) and L4-L5 (1/14). These hyperintensities did not suppress on T2 FLAIR in: forebrain (4/14) and meninges (2/14). Contrast enhancement was present in: meninges (6/14), temporal muscle (1/14), facial/vestibulocochlear nerve (1/14). MRI was considered negative in 3/14. Treatment was based on dexamethasone: as monotherapy (2/14), in association with CCNU (2/14), Cytarabine (2/14), Ciclosporin and CCNU (2/14), Ciclosporine and Cytarabine (2/14), Cytarabine and CCNU (1/14); and Prednisolone and Cytarabine was used in (3/14). Antiepileptics were administered in 2/14. All dogs survived on discharge and 11/14 at least one year after initial treatment.

IEME varies in clinical manifestations, with a variety of MRI findings. IEME can have short-termed positive response to combination of steroids and chemotherapy treatment.
Brachial plexus avulsion (BPA) is a common clinical circumstance in small animal practice. BPA can be associated with neuropathic pain affecting the quality of life (QoL) of human patients. The aim of the study was to describe the clinical and electrodiagnostic (EDX) findings and the outcome of a cohort of dogs and cats with BPA and to investigate their QoL, the presence of signs of pain and the owners’ perspective.

The clinical records of 40 dogs and 23 cats with BPA were retrospectively reviewed. Specific attention was put on the evaluation of EDX findings (35/40 dogs; 11/23 cats) and telephon ic interview results (26/40 dogs; 15/23 cats) (ethical committee approval is pending).

The most common neurological condition was inability to bear weight and absence of deep pain perception on the affected limb (65% dogs; 52% cats). Radial and ulnar motor nerve conduction (MNC) were absent respectively in 47% and 62% of dogs and 55% and 50% of cats. In dogs, absence of radial (P=0.02) and ulnar (P=0.007) MNC was significantly associated to the amputation of the affected limb. The owners described signs of pain/discomfort in 73% of dogs and 53% of cats. Despite QoL was considered excellent in 34% of dogs and 80% of cats, it was described as poor in 8% of dogs and 7% of cats. Owners perceived BPA affecting their daily activities (29% dogs; 14% cats) and social life (25% dogs 14% cats).

BPA represents a common entity and its possible association to neuropathic pain must be considered in order to improve the QoL.
[FP11] PREVALENCE AND CLINICAL CHARACTERISTICS OF PHENOBARBITONE-ASSOCIATED ADVERSE EFFECTS IN EPILEPTIC CATS

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The purpose of this study was to investigate the prevalence and clinical characteristics of phenobarbitone-associated adverse effects in epileptic cats.

The medical records of two referral veterinary clinics were searched for cats diagnosed with epilepsy and treated with phenobarbitone (2007-2017). Follow-up information was obtained from the primary veterinarian and referral institution medical records and a questionnaire completed by the cats’ owners.

Seventy-seven cats, 58 with idiopathic epilepsy and 19 with structural epilepsy, met the inclusion criteria. One or more of the following adverse effects were reported in 47% (36/77) cats: sedation 89% (32/36), ataxia 53% (19/36), polyphagia 19% (7/36), polydipsia 6% (2/36), polyuria 6% (2/36) and anorexia 6% (2/36). Median phenobarbitone dosage at the time of adverse effect onset was 3 mg/kg (range 1.25-5 mg/kg) BID. Adverse effects were known to resolve following achievement of steady state or following dose reduction in 20 cats. Logistic regression analyses revealed a significant association between adverse effect occurrence and phenobarbitone dose and administration of a second antiepileptic drug. For each increment of 1 mg/kg BID, the risk of ataxia increased 2.7 times and the risk of sedation increased 3.4 times. Epilepsy aetiology was not associated with adverse effect occurrence. Idiosyncratic adverse effects, characterised by severe neutropenia and severe granulocytic hypoplasia, were diagnosed in only one cat. These resolved following phenobarbitone discontinuation.

In this study, phenobarbitone dose and administration of a second antiepileptic drug were the most significant risk factors for the development of the most commonly encountered adverse effects (sedation and ataxia).

This study was approved by the Ethics Committee of the Animal Health Trust, England (approval number AHT62-2016).
Degenerative sensory polyneuropathy, has been described in the Border collie. It has recently been associated with a mutation of the \textit{FAM134B} gene, thought to be critical in the survival of sensory nerve cells. This case series aims to provide a detailed clinical and electrophysiological description of the disease in a cohort of 6 dogs.

Border Collies with clinical signs of polyneuropathy of juvenile onset were retrospectively included in the study. Clinical, electrophysiological and histological data were reviewed.

Six dogs aged from 4 to 19 months were presented with a chronic history of progressive proprioceptive ataxia. Decreased nociception and loss of withdrawal reflex were seen in all dogs. Self-mutilation was reported in 2/6 dogs. Muscle atrophy was reported in 5/6 dogs.

EMG showed fibrillation potentials in the interosseous muscles of the pelvic limbs and the thoracic limbs in respectively 6/6 and 3/6 dogs and in other muscles in 2 dogs. CMAP amplitudes and MNCVs were decreased in at least one nerve in 5 and 2 dogs respectively. Compound sensory action potentials were elicited but never obtained. Histological analyses of muscle biopsies were normal in 3 dogs and showed myofiber atrophy consistent with chronic denervation in 3 dogs.

This study suggests phenotypical heterogeneity in sensory neuropathy in Border Collie, with partial motor involvement in all dogs, as described in human patients affected by \textit{FAM134B} associated polyneuropathy.
[FP13] EOSINOPHILIC CEREBROSPINAL FLUID PLEOCYTOSIS ASSOCIATED WITH NEURAL ANGIOSTRONGYLUS VASORUM INFECTION IN A DOG

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A 1-year-old, female Pug dog was presented with a month history of progressive truncal swaying, ataxia, right-sided head tilt, altered mentation, right eye exophthalmia, and conjunctival hyperaemia. Neurological examination revealed obtunded mentation, mild ambulatory tetraparesis and vestibulo-cerebellar ataxia, occasional truncal swaying with the tendency to fall on both sides and generalised whole body tremors. The postural responses were markedly delayed to absent in all limbs. Menace response was absent bilaterally. The findings indicated a multifocal intracranial lesion affecting the central vestibular system (cerebellum and brainstem) and forebrain. Magnetic resonance imaging (MRI) detected multiple haemorrhagic lesions in the forebrain, thalamus, mesencephalon and metencephalon. Cerebrospinal fluid (CSF) collection was not performed due to the increased risk of bleeding. Baermann test and zinc sulfate flotation with centrifugation performed on faecal samples were positive for lungworm larvae and an antigenic test confirmed *Angiostrongylus vasorum* infection. Anthelmintic treatment was started with consequent marked clinical improvement. Seventy days later the dog was clinically normal and no larvae were detected on Baermann test. Repeat MRI of the brain revealed marked improvement of the haemorrhagic lesions. CSF then showed marked eosinophilic pleocytosis and anthelmintic treatment was restarted. Follow up CSF analysis 4 months after first presentation revealed resolution of the eosinophilic pleocytosis. This is the first case report of marked eosinophilic pleocytosis associated with neural *Angiostrongylus vasorum* infection in a dog. CSF eosinophilic pleocytosis persisted for several weeks after treatment, even in absence of concurrent clinical signs and with negative Baermann test for *A. vasorum*.
Vertebral venous system abnormalities have been identified on MRI in 12% of all sight-hounds in a recent study but the underlying cause is so far unknown. This case report presents the histopathological findings of a 3-year-old, male neutered Scottish Deerhound with 3 weeks history of severe neck pain. MRI of the cervical spine showed severe dilatation of the vertebral venous sinus, mainly on the right side causing mild to moderate spinal cord compression. The dog died from an aspiration pneumonia shortly after the MRI and was subjected to a post mortem examination.

The macroscopic findings showed severe dilatation of the venous sinus on the right side causing significant compression of the spinal cord and nerve roots. Histopathologic examination showed mild changes in the spinal cord with some spheroids and digestion chambers. In the dilated venous sinus there were severe degenerative changes, with multifocal areas of hyalinization within the tunica media with infiltration of macrophages, siderophages and neutrophilic granulocytes. Also within the tunica adventitia there were small bleedings and hemosiderin filled macrophages. The endothelium was irregular and even completely missing in some parts of the vessel. The findings indicate an aneurysm with secondary dissection through the vessel wall.

Aneurysms in humans have been described as having a variety of causes including high blood pressure, atherosclerosis, tumors, infection, trauma, abnormal blood flow and hereditary anomalies. Given that this phenomenon is most commonly seen in sighthounds and only on rare occasions in other breeds, indicates that it is likely to have a hereditary origin in these breeds.
Corpus callosum abnormalities (CCA) occur rarely in dogs and are clinically related with hypo/adyspic hypernatremia and seizures. Hypoplasia and dysplasia of the corpus callosum with concomitant lobar holoprosencephaly is the most commonly reported variant. It is currently uncertain if MRI findings in canine CCA reflect a failure of commissural fibres to develop or to cross the hemispheres.

Diffusion tensor imaging (DTI) was performed in a 4-year-old Staffordshire mix breed dog with CCA. Tractographies of the whole brain, corpus callosum, and supracallosal cingulate fibres were performed. Fibre tracts derived from regions of interest were compared to those of a healthy beagle, which were acquired using the same DTI protocol.

Tractography of the dog affected with CCA depicted only white matter tracts corresponding to the temporal callosal fibres. Longitudinal supracallosal axonal bundles of the dog with CCA appeared increased in number, with unorganized architecture in comparison to the control dog, extending into the ipsilateral cerebral cortex.

The supernumerary axonal bundles strongly suggest homology to Probst fibres reported in humans with CCA. Similar as in humans, presence of Probst fibres in canine CCA could represent compensatory neuroplasticity-mediated networking and could be a contributing factor to previously reported fair prognosis of affected dogs.
[FP16] HPA AXIS DYSREGULATION ASSOCIATED WITH CHRONIC IDIOPATHIC EPILEPSY IN DOGS

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A bidirectional relationship exists between seizures and stress; stress is the most common seizure precipitant in people with epilepsy, and recent research has revealed the acutely stressful nature of generalised seizures in dogs with idiopathic epilepsy (IE). Although significant spikes in salivary cortisol are observed immediately post-seizure, the effect of repeated seizures on the hypothalamic-pituitary-adrenal (HPA) axis is not yet known. This study aimed to investigate the effect of chronic seizure activity on hair cortisol concentrations (HCC), a chronic stress measure in dogs.

Owners of Border Collies (BCs; n=135) reported their dog’s clinical history and collected a 0.5 g hair sample from their dog’s dorsal neck region. Dogs were included in the IE group if they met International Veterinary Epilepsy Task Force tier I criteria, and in the control group if they had no history of specified health conditions. HCC was determined using established methods with a commercial cortisol assay kit (Salimetrics). Samples from 46 BCs with IE and 89 control BCs were analysed; HCC was significantly lower in dogs with IE compared to controls (p=0.02). Within the IE group, total number of seizures was negatively correlated with HCC (r=-0.31, p=0.04).

Despite acute increases in salivary cortisol observed following seizures, HCC was lower in dogs with IE compared to controls, and lower in dogs that had experienced a greater number of seizures. Chronic stress with frequent HPA-axis activation may, over time, lead to HPA axis dysregulation, as seen in chronic psychiatric disorders in people, including depression and post-traumatic stress disorder.
[FP17] COMPARISON OF IMMULITE 2000® VERSUS CATALYST DX® INSTRUMENTS FOR THE ANALYSIS OF SERUM CONCENTRATION OF PHENOBARBITAL IN DOGS.

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Therapeutic drug monitoring is recommended for dogs with seizures treated with phenobarbital. Dose adjustments are calculated based on a formula using the actual and desired serum concentration. Monitoring of serum levels of phenobarbital also provide an indication for tolerance and risk of hepatotoxicity.

In house testing is getting more common, as it is both advantageous for the clinician to obtain test results quicker and from an economic point of view. Most of these instruments have been validated by correlation studies as an indicator of agreement but the differences between the test results have not been studied.

The aim of this study was to compare serum concentrations of phenobarbital in dogs between two different instruments: Catalyst Dx® (Idexx) and Immulite 2000® (Siemens).

Blood samples of 30 client owned dogs were collected and analysed with Catalyst Dx® (Idexx) and Immulite 2000® (Siemens). Serum concentrations from both analytic instruments were statistically compared by a paired t-test. The significance level was set at $p \leq 0.05$.

Serum levels of phenobarbital analysed by Catalyst Dx® (103.983 ± 20.472µmol/L) were significantly higher compared to the same samples analysed by Immulite 2000® (84.323 ± 19.301 µmol/L), $p < 0.0005$.

These results should alert the clinician to check which analytic instrument is used when comparing levels of phenobarbital in a dog to detect development of tolerance to the drug, when interpreting results to judge for the risk of hepatotoxicity and when calculating dose adjustments.
[FP18] THE ASSOCIATION BETWEEN DURATION OF ANESTHESIA AND OUTCOME IN DOGS WITH ACUTE SEVERE SPINAL CORD INJURY SECONDARY TO THORACOLUMBAR INTERVERTEBRAL DISC EXTRUSION UNDERGOING DECOMPRESSIVE HEMILAMINECTOMY

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Exploratory research identified a possible relationship between duration of surgery and outcome in severely affected dogs treated surgically for thoracolumbar intervertebral disc extrusion (TL-IVDE). The objective of this study was to investigate this relationship further. We hypothesized that increased duration of surgery is associated with poor outcome in deep pain negative (DPN) dogs treated surgically for TL-IVDE.

Medical records of 5 institutions were retrospectively reviewed. Inclusion criteria were DPN status, surgical management of TL-IVDE and >6 weeks post-operative outcome (ambulatory: yes or no). Patient data, outcome, and surgery and total anaesthesia duration were retrieved.

342 dogs were included, with 118/310 (38.1%) ambulatory at 6 weeks and 184/297 (62.0%) ambulatory at 1 year. 74 dogs (21.6%) were euthanized due to ascending-descending myelomalacia. Among the dogs that failed to recover, duration of surgery and anaesthesia data demonstrated a marked positive skew towards several extreme outliers. Median anaesthesia duration in dogs that regained ambulation within 1 year of surgery (4.04hrs, interquartile range [IQR] 1.91) was significantly shorter than those that did not (4.46hrs, IQR 1.91, P=0.017). Multivariable logistic regression demonstrated a significant association between ambulation at 1 year and duration of surgery (coefficient=-0.205, P=0.010) and total anaesthesia (coefficient=-0.294, P=0.002) when controlling for body weight and number of disc spaces operated on.

Our findings support a negative association between increased duration of anesthesia and outcome in this group of dogs. However, this effect was driven by several marked outliers and does not necessarily imply a causal relationship. Further study of these outliers is indicated.
Spinal cord injury can lead to paralysis and urinary incontinence in people and animals. Supra-sacral injuries lead to bladder over-activity characterised by frequent involuntary emission of urine and lack of bladder distension awareness. Currently, no satisfactory method exists to monitor the detrusor activity, that could be used to either block the onset of ‘reflex bladder contractions’ (i.e. neuromodulation) or to ‘inform’ the patient of the level of bladder fullness.

Past research has used hook or microchannel electrodes on teased sacral nerves in anaesthetised rodents, but faced major obstacles in large animals: (i) afferent bladder signals were masked by electrical noise and bioelectric interference from other organs; and (ii) electrode implantation led to nerve damage and high impedance, preventing recordings. We have designed a new nerve interface, multi-electrode cuff (from silicone with steel electrodes), connected to a Cooper cable for signal extraction. The cuff design is based on the anatomy of sheep sacral nerves. Using this implant, we have conducted impedance measurements following stimulation of explanted pig vagal nerves. This showed impedance values of ~5kΩ for frequencies ranging 100-1,000Hz, which fulfils theoretical requirements for afferent bladder sacral nerve recording. This cuff contains up to 10 electrodes to exploit the method of velocity selective recording, a powerful technique to discriminate various afferent fibres based on their conduction velocity. In particular, we plan to use this device in a sheep model, in which bladder afferents coding for pressure and volume of the urinary bladder are similar to man, ~38 and 41 m/s respectively.
The purpose of this study is to describe the prevalence of congenital sensorineural deafness (CSD) in a client-owned population of white pure-breed kittens and non-white littermates in the UK.

Full litters of kittens with at least one white kitten, aged between 6 and 21 weeks at the time of brainstem auditory evoked response test were included (193 cases - 56 retrospectively and 137 prospectively). There were 132 solid white kittens. CSD was diagnosed only in solid white kittens. The overall prevalence of CSD in solid white kittens was 30.3% (15.9% bilateral, 14.4% unilateral). The prevalence of CSD was significantly higher in white kittens with either one (44.4%) or two (50%) blue eyes than in white kittens without any blue eyes (22.2%). Kittens with at least one blue eye were 3.2 times more likely to have CSD than kittens without blue eyes. Prevalence of CSD did not differ significantly between white cats with one or two blue eyes. The prevalence of CSD (in solid white kittens of each breed) was 46.7% (7/15) in Turkish Vankedisi, 44.0% (8/18) in Main coon, 43.9% (18/41) in Norwegian forest, 27.0% (3/11) in British shorthair, 16.7% (2/12) in Devon rex, 8.3% (1/12) in Persian, 4.8% (1/21) in Russian and 0% (0/2) in Sphinx.

This study identified a high prevalence of CSD in a population of client-owned pure-breed white kittens in the UK. Differences in breed-specific prevalence of CSD were of particular interest and should be investigated in larger populations.
[FP21] VENTRICULOMEGALY IN DOGS TRAINED FOR FMRI STUDIES: FINDINGS AND FUTURE OBJECTIVES

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The purpose of the study was to investigate how the brain ventricles’ volume changes with age in a unique sample, i.e. dogs trained to lay awake in an MRI during scanning.

Structural MRI data were obtained at least twice (1-6 years between scans) from 21 family dogs (Mage = 3.81 years, range 1.1-11.4; F = 7, M = 14) trained to lay motionless (movements below 1.00 mm for each translation direction, and below 0.01 degree for each rotation direction) in the MR scanner (Philips Ingenia 3.0 T), without any sedation or physical restrain, for 6-8 minutes (ethical permissions were obtained). Repeated T1-weighted images were acquired, inter-scan range varied between 1-6 years, number of scans obtained between 2 and 4. Separate left and right lateral ventricular volumes were calculated with FSL and FEI Amira 6.5 softwares.

Preliminary results (N = 7) indicated that the range of ventricular increase was between 20% and 63% (median = 37%) for the left ventricle, and between 16% and 172% (median = 58%) for the right ventricle. None of the dog owners reported significant changes in the behavior of their dog during the given period, and despite the ventricular enlargement the dogs were able to perform the trained task fulfilling above mentioned strict criteria.

This is the first longitudinal study investigating MRI signs of brain ageing in awaken and healthy dogs. The technique provides unique opportunities for the study of the canine brain, while avoiding the risks and ethical considerations associated with general anesthesia.
Glutamate is the most important epilepsy-associated excitatory neurotransmitter and renders brain regions as the hippocampus particularly vulnerable to excitotoxicity. Recently increased expression of the cysteine/glutamate transporter SLC7A11 has been identified to correlate with seizure incidence in structural epilepsy due to glutamate induced excitotoxicity and subsequent neuronal injury.

As there appear to exist essential neurobiological differences regarding hippocampal involvement in epilepsy and the existence of temporal lobe epilepsy in different species, we investigated the immunohistochemical expression pattern of SLC7A11 within the hippocampi of rats, cats, dogs and subhuman primates.

SLC7A11 indeed presents with distinct expression patterns in each species. The hippocampal expression in the rat was in general very weak while staining intensity suggests predominant neocortical expression. In contrast, clear bands of SLC7A11 expression were seen in molecular layer and alveus of the feline hippocampus. Canine brains displayed more widely distributed immunoreactivity in molecular layer, stratum oriens and stratum radiatum up to CA4-CA2 with subsequent involvement of the entire pyramidal cell layer in the remaining regions up to subiculum. In non-human primates, the molecular layer of dentate gyrus is spared, while stratum oriens and radiatum of CA3 and CA4 stain strongly immunopositive.

This expression study highlights essential interspecies differences regarding the distribution of SLC7A11 in the non-epileptic hippocampus. Its neurobiological role in epilepsies of the respective species has to be further investigated.
Different glycogen storage diseases have been described in dogs with or without muscle involvement but this is the first time that glycogen storage disease type V has been identified in a dog. This disease is caused by a deficiency of muscle phosphorylase A corresponding to McArdle disease.

A five year old, neutered female, cross breed dog was presented with a three months history of exercise intolerance. On presentation, the dog had a mildly elevated CK (358 U/L) and a severely elevated lactate (30 mmol/l) after only a few minutes of exercise.

Muscle histology revealed moderate chronic myopathic changes diastase-sensitive polysaccharide inclusions. Biochemical/metabolic testing showed a decrease of muscle phosphorylase A as seen in human McArdle disease/glycogen storage disease V.

The late onset of the McArdle disease is similar to what is noted in humans where the disease is often not diagnosed until the patient is 30-40 years old. Treatment in people consists of exercise avoidance and high oral glucose supplementation just before any physical activity. That way human patients can live to an old age. Unusual for McArdle patients, the dog also suffers lactic acidosis with yet unclear relation to the enzyme deficiency according to lactic acidosis type B. Thus, the outcome in this particular patient is more uncertain.
SENSORY NERVE CONDUCTION MEASUREMENTS AND DIAGNOSTIC APPLICABILITY IN HORSES WITH SYMPTOMATIC AND IDIOPATHIC HEADSHAKING

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Sensory nerve conduction (SNC) threshold of the infraorbital nerve was reported to be decreased in horses with idiopathic headshaking (IHS) with a stimulation threshold of ≤5mA in comparison to healthy horses.

In the current study, a prospective ongoing trial, horses were examined using standardized diagnostic procedures to differentiate symptomatic headshaking (SHS) from IHS. In all patients, SNC of the infraorbital nerve was measured bilaterally under general anesthesia. Sensory nerve conduction velocity, amplitude of the sensory nerve action potential (SNAP) and stimulation threshold were recorded. The technique described by Aleman et al. 2013 had to be modified to minimize artefacts. All examinations were performed with written owner consent according to university guidelines.

Twelve horses with equine headshaking were included: two with SHS and 10 with IHS. In total, 22 measurements were obtained. In the horses with SHS, SNAP were evoked at 15mA (left side) and 20 mA (right side), as well as at 7.5mA bilaterally. In horses with presumed IHS, the median threshold was 10 mA (5 mA - 15 mA). Despite small subdermal hemorrhage no major side effects or deterioration of clinical signs were detected after the measurements.

In conclusion, measurement of SNC of the infraorbital nerve is a feasible and safe method to further support the diagnosis of IHS in horses, although the displayed threshold might vary depending on the electrodiagnostic laboratory and laboratory specific thresholds have to be determined.
[FP25] THE UTILITY OF A RAPID, IN HOUSE METHOD OF CSF ANALYSIS INVOLVING SEDIMENTATION AND CYTOLOGICAL EVALUATION DIRECT FROM THE SPINAL NEEDLE

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The project aims to assess the utility of a novel method of CSF analysis, to determine whether it can provide a rapid preliminary result in the diagnosis and treatment of conditions leading to pleocytosis, such as Steroid Responsive Meningitis Arteritis (SRMA) and Meningoencephalitis of Unknown Origin (MUO).

Inclusion criteria were cases presented to the Queen’s Veterinary School Hospital, Cambridge and a private referral centre for investigation of neurological signs in which CSF analysis was performed. In addition to standard laboratory processing, an additional slide was prepared by everting the spinal needle used to take the sample and standing on a plain glass slide, leaving for a minimum of one hour to allow sedimentation of cells, then processing with a rapid stain for cytological evaluation. Pleocytosis was defined as an average of >5 cells per 50x high power field.

55 samples were included in the study, with sensitivity and specificity found to be 89.4% and 94.4% for detecting laboratory defined pleocytosis (determined using a haemocytometer). Sensitivity and specificity for detecting a neutrophilic pleocytosis were 100% and 97.8% respectively, and for a mononuclear pleocytosis, 50% and 90.9%.

This simple and rapid method of CSF analysis appears to have high sensitivity and specificity for the detection of pleocytosis and may be advantageous in cases requiring rapid treatment or where access to laboratory facilities is lacking.
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Poster Abstracts
[P1] PRELIMINARY VOLUMETRIC ASSESSMENT OF THE LATERAL TEMPORAL LOBES IN DOGS WITH IDIOPATHIC EPILEPSY

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Epilepsy is one of the most common neurological diseases in humans and dogs. In humans, there is some evidence that temporal lobe epilepsy (TLE) is characterized by abnormalities in the hippocampus, but also in brain regions that extend beyond it (amygdala, entorhinal cortex, parahippocampal gyrus). To date, volumetric assessment of the temporal structures has not been performed in dogs with idiopathic epilepsy (IE). Hence, the aim of the study was to carry out a preliminary volumetric assessment of the temporal lobes in dogs with IE.

The study was carried on 19 Border collies divided into two groups: A; dogs with confirmed IE (n=10) and B; dogs with no history of seizures and normal brain MRI (n=9). Forty 3D-T1-weighted MRI images (TR 25; TE 4.8; FS 1.5; voxel size 0.75x0.75x0.375mm) of the brain were assessed and the volume of the left and right temporal lobe (ROI, cm³) was segmented using a semi-automatic method (OsiriX 8, Switzerland).

A statistical analysis carried out using the student t-test revealed no significant differences in the lobe volumes between the groups (p>0.05). The interhemispheric lobe ratio was calculated (smaller volume/larger volume) in all the animals and a mean was calculated for group B. Nine of the 10 animals in group A had a larger lobe ratio than the mean value of group B.

These preliminary findings form the basis for a wider study aimed at determining whether the lateral temporal lobes undergo structural changes and have a role in epileptogenesis in TLE.
[P2] TRANSTHORACIC VERTEBRAL REALIGNMENT AND FIXATION FOR TREATMENT OF THORACIC HEMIVERTEBRAE IN DOGS

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Congenital vertebral malformations are common findings in screw-tailed brachycephalic breeds that can be treated conservatively or surgically depending on its severity and progression when causing neurological signs.

We describe six dogs (from six months to four years old) with clinical thoracic hemivertebrae excluding other concomitant myelopathies, surgically corrected with the same technique of vertebral realignment and fixation, through a transthoracic approach (2013-2018). Diagnosis was achieved with myelography(2), myeloCT(1), MRI(1), MRI and CT(1), MRI and myelography(1). A right CASPAR cervical distractor system (Aesculap Implant Systems, Germany) was used for the vertebral distraction and realignment, and cortical screws placed one to two vertebrae cranial and caudal to the malformation and embedded in polymethylmethacrylate. Clinical improvement without post-op deterioration was achieved in all cases with two cases becoming neurologically normal. No complications were reported during the follow-up period (7 months to 4.5 years).

Most surgical techniques for the correction of clinical hemivertebrae in dogs use a dorsal approach with decompression and/or fixation. However, the vertebral canal height does not differ between dogs with and without thoracic hemivertebrae; therefore, realignment of the vertebral canal might be sufficient in compressive cases.

The technique used in these case series has been described in detail by its author Baroni (personal communication) but no clinical studies on the long-term outcome have been published.

This case series suggests that vertebral realignment and fixation with cortical screws and PMMA through a transthoracic approach for the treatment of clinical hemivertebrae is a feasible technique with promising results.
[P3] TREATMENT OUTCOME OF SURGICALLY REINTERVENED RECURRENT INTRACRANIAL GLIOMA IN 4 DOGS

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Glioma includes several types of primary nervous system tumors further categorized by grade on the basis of histologic assessment and biological characteristics. Treatment options for confirmed gliomas include radiotherapy, surgery, chemotherapy or combinations with the former providing longer survival time (255 days) compared to surgery alone (66 days) probably due to incomplete resection. To the authors’ knowledge there are no reports of reintervining intracranial gliomas in dogs. Herein, we report our clinical outcome in 4 cases.

Four dogs with histopathologically confirmed glioma previously resected with curative intent followed by adjuvant temozolamide developed tumour recurrence and underwent reintervention. Gross total resection of tumor recurrence was performed using the same previous approach. After the second resection, temozolomide was replaced by toceranib (2.5-2.8 mg/kg 3 times weekly) except in one dog (case 1) treated with lomustine after her third surgery (70mg/m²/3 weeks).

Neurological deterioration (<24h) after reintervention was minimal except for case 4 euthanized 24h after the second surgery for respiratory complications. In the remaining three dogs euthanasia was due to presumed (n=2) or radiologically confirmed (n=1) tumour recurrence. Survival times were 778, 241, 422 and 472 days following imaging diagnoses. Cases 1, 2 and 4 lived 176, 73 and 136 additional days from imaging diagnosis of recurrence. Case 1 was reintervened twice and lived 183 additional days after the third surgery.

In human medicine reoperation of recurrent gliomas when feasible improves outcomes. Our case series suggests that reintervention in canine recurrent gliomas should be considered as an option in their treatment.
[P4] FUNCTIONAL NEUROREHABILITATION SCALE FOR DOGS WITH THORACOLUMBAR SPINAL CORD INJURY WITHOUT DEEP PAIN SENSATION

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Functional Neurorehabilitation (FNR) is an area of restorative neurology, which aims to optimize neuromodulation and neural reorganization for functional recovery. Dogs with sensorimotor complete spinal cord injury that do not recover pain perception after the injury undergo extensive changes in the spinal cord below the injury level that may be manipulated to produce functional recovery. The purpose of this work was develop a scale to reliably quantify the recovery of function in dogs with chronic severe spinal cord injury.

Ten dogs that were paraplegic with no pain perception underwent serial neurological examinations. Assessments of sensory perception, reflexes, muscle tone, motor function and coordination and used to develop ordinal scores. Reliability was assessed by 2 observers examining the dogs.

The scale captured 4 different levels of function in each category to give a total score of 20. Dogs scoring 0 had no movement, no sensation, reduced reflexes and muscle tone while dogs scoring more than 15 could walk independently. The complete assessment took approximately 2 minutes. Interobserver reliability was 82.4%.

The scale was easy to use and captured changes in different functional categories. It provides a useful tool to quantify recovery and modify rehabilitation protocols in dogs undergoing FNR.
[P5] USE OF NOCITA FOR PAIN MANAGEMENT AFTER HEMILAMINECTOMY IN DOGS

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We demonstrate that NOCITA\textsuperscript{®} is safe and effective for pain management after hemilaminectomy in dogs following IVDD.

Post-op opioid sparing techniques are being emphasized in human medicine. Modern anesthesia is shifting to a reliance on topical anesthetics, NSAIDs and NMDA antagonists to achieve adequate post-operative analgesia. Such protocols decrease medication related side effects, shorten hospitalization and enhance quality of post-operative recovery. The use of local anesthetics (LAs) has the most evidence-based support as a perioperative analgesic in veterinary medicine. Liposomal bupivacaine has been developed as long acting LA to provide extended release action over 72 hours. Extensive studies have validated the local and systemic safety of NOCITA\textsuperscript{®} in dogs: It has been shown to not migrate from its site of application and has been documented to be safe and efficacious following epidural, perineural, and intraneural injections. NOCITA\textsuperscript{®} has the potential to provide valuable and safe opioid sparing post-operative analgesia following spinal surgery.

Thirty-eight retrospective cases were collected from June 2017 to March 2018. Dogs undergoing a thoracic or lumbar hemilaminectomy for IVDD correction, that received a NSAID post-operatively, NOCITA\textsuperscript{®} on the epaxial muscles during surgical closure, and a 0.5mg/kg/hour ketamine CRI for 24 hours were collected. Pain score, clinical and neurological examination as well as side effects were documented. Successful pain control with no or minimal opioid rescue therapy and no side effects related to the NOCITA\textsuperscript{®} injection was observed.
Th17 cells are a special type of T helper cells playing an important role during inflammation and autoimmune disease. To investigate the role of these cells in diseases of the nervous system in dogs in a clinical setting, methods for fast identification had to be identified.

In earlier studies an ELISA test measuring IL17 in cerebrospinal fluid (CSF) samples and an ELISpot technique were successfully applied. However, these techniques cannot be performed on a daily routine basis. Therefore, Th17 cells should be measured using flow cytometry. Since Th17 cells are only a minor population of lymphocytes, stimulation is necessary for better visibility. The measurement protocol time takes about 15 hours being the main difficulty for routine analysis. For this reason, results of 10 blood samples divided in two portions were compared: half of the blood sample was processed immediately and the other half was stored overnight for 15-20 hours. Cells were stimulated with PMA, Ionomycin and Brefeldin A for 6 hours and Th17 cells were intracellularly stained with biotinylated IL17A (Dendritics, France) and measured by flow cytometry.

No significant difference between stored and freshly measured cells occurred (p= 0.7144). Stimulated Th17 cells account for approximately 8-20 percent of canine lymphocytes (n=150).

In conclusion, storing blood samples overnight offers an efficient way to measure Th17 cells by flow cytometry in canine neurological diseases. The study was conducted in accordance with the ethical guidelines of the University of Veterinary Medicine Hannover (experiment number 33.8-42502-05-18A290).
[P7] CERVICAL DISTRACTION-FUSION IN SMALL DOGS

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The purpose of this study was to retrospectively evaluate the outcome of cervical distraction-fusion (CDF) in small breed dogs <15kg with chronic intervertebral disc protrusions, between 01/2013 and 01/2018.

8 dogs met the inclusion criteria and underwent CDF using an intervertebral traction device (Fits Intervertebral Traction Screw; FITS) with positive threaded pins and polymethylmethacrylate bone cement. Data collected from electronic patient records included patient signalment, duration of clinical signs, findings on neurologic examination, MRI findings, implants utilised and clinical outcome. Long-term outcome was assessed via telephone interview (5.5-45months). A distraction index was calculated to assess for evidence of vertebral subsidence comparing immediate post-op and long-term post-op imaging. A paired sample t-test was used to assess for significance.

Various breeds were represented with median weight 9.3kg (range 5.95-14.3) and age 8.75years (range 3-11.5). The median duration of clinical signs was 28days (range 5-183). 7/8 dogs failed conservative therapy prior to surgery. The commonest clinical sign was ambulatory tetraparesis (4/8). Diagnostic investigations consisted of MRI in all cases and CT in 4/7 cases. The intervertebral spaces most commonly affected were C3/4 and C5/6 (3/8 cases). Long term, 5/8 cases demonstrated an improvement in clinical signs. There was no significant difference in distraction index immediately post-op and at 6-8 weeks.

CDF appears to be an effective treatment for intervertebral disc protrusions in small breed dogs. This technique may have advantages over ventral slot decompression where disc collapse, vertebral instability or dynamic protrusion is suspected.
Melanoma is a common neoplasia in dogs, accounting for 7% of all malignant tumors and 3.3% intracranial metastatic neoplasia. However, primary leptomeningeal melanomatosis originating from leptomeningeal melanocytes is a rare malignancy in both human and veterinary medicine.

Here we presented a 13 years old spayed female Labrador retriever who was referred to neurology service for rapidly progressed neurologic signs of non-ambulatory tetraparesis and right head tilt over 2 weeks. MRI showed diffuse T1W isointense, T2W hyperintense and strongly enhanced leptomeningeal lesion extending from the cervical spine into the cranium, mainly right side of the brainstem, cerebellum and longitudinal fissure. Nodular formation and invasion into spine and cerebral parenchyma were also noticed. CSF analysis indicated elevated protein level and pleocytosis with mostly neoplastic round cells and some melanocytes. The patient continued to deteriorate drastically and died 30 days after the neurologic signs was first noted. At necropsy, the dog’s brain and entire spinal cord were covered with thick and dark tissue. No evidence of a primary melanoma was found in other parts of the body. Histopathologic findings and immunohistochemical results against Melan-A supported the diagnosis of presumed primary leptomeningeal melanomatosis. Its MRI features in dogs have not been reported before but similarities with human were found in our patient. Unlike the low diagnostic value in human, CSF cytology provided this dog imperative antemortem diagnostic information.
[P9] INCIDENCE AND CLINICAL FEATURES OF SUSPECTED POSTICTAL CHANGES IN EPILEPTIC DOGS

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Post-ictal changes (PC) detected on brain magnetic resonance imaging (MRI) are postulated to be a consequence of excitotoxicity associated to seizures, and are recognized in human and veterinary medicine. In veterinary literature, detailed description of these findings is limited to one case-series report.

The purpose of this retrospective study was to determine the incidence, imaging characteristics and clinical associations of PC observed on T2-weighed MRI sequences in a population of epileptic dogs.

Medical records and imaging findings of dogs with diagnosis of epilepsy and brain MRI (0.2T or 1.5T) performed between January 2016 and March 2018 were retrospectively reviewed. Signalment, seizure type (focal/generalised), aetiology (idiopathic/structural/unknown origin), presence of clusters or status epilepticus (SE), time between last seizure and MRI, detection of structural lesion and presence and localisation of suspected PC were assessed.

Eighty-nine dogs met the inclusion criteria. Lesions interpreted as PC were observed in 12 dogs (13,5%). Brain areas affected included piriform lobes and/or hippocampus, cingulate gyrus, and more diffuse cortico-subcortical areas. PC were bilateral in 6 dogs. Three dogs suffered SE, 6 dogs had clusters and 3 dogs isolated seizures. Mean time between last seizure and MRI was lower in dogs with PC. Prevalence of idiopathic and structural epilepsy was similar in both affected and unaffected dogs.

Results show that distribution of presumptive PC can be variable in dogs. As described in humans, PC were more frequently but not exclusively observed after clusters or SE, and they can be observed in structural and idiopathic epilepsy.
[P10] SURGICAL RESOLUTION OF SEVERE DISCOSPONDYLITIS AND SPINAL EMPYEMA IN A DOG DUE TO FOREIGN BODY

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The aim of this case report is to describe the surgical resolution in a dog with spinal epidural empyema (SEE), severe discospondylitis and removal of a retroperitoneal foreign body (FB) through decompressive surgery with stabilization and laparotomy for removal of the FB. To the authors’ knowledge both approaches simultaneously have not been reported.

A four years-old male German Shorthaired Pointer was referred for a 4-month history of progressive fever, paraparesia and severe lumbar pain. Neurological examination was consistent with a severe T3-L3 myelopathy. Thoracic radiographs were unremarkable. Abdominal ultrasound revealed an abscess in the retroperitoneal cavity. Survey radiographs of the lumbar spine revealed changes consistent with discospondylitis at L3-L4. Computed tomography and MRI of the lumbar spine revealed SEE causing severe compression of the spinal cord at that L3-L4 and severe osteolytic changes in both vertebrae suggesting instability. Moreover, a retroperitoneal fistulous path was identified.

Hemilaminectomy at L3-L4 was performed, removing the purulent content, and stabilization with pins and methacrylate was performed. After that, an exploratory laparotomy was performed and a FB (grass/plant material of approximately 5 cm) was removed. A culture of the purulent content identified Pseudomonas aeruginosa. Four days after surgery there was improvement of the clinical signs. Five months later the dog was clinically normal.

The aim of this case report is to demonstrate a successful outcome of the two surgical procedures above mentioned in the same anesthetic time for treatment of severe SEE and discospondylitis secondary to a retroperitoneal FB.
The purpose of this case report was to report a skeletal osteosarcoma with central nervous system metastasis.

A 9-year-old, male neutered Old English Sheepdog was presented with a ten-day history of altered mentation that had progressed to impaired vision and partial hemineglect syndrome. During the five months prior to referral, the dog had also suffered from intermittent discomfort on the right forelimb. Radiographs of the right forelimb and thorax revealed the presence of an aggressive solitary bone lesion affecting the right humerus with no evidence of pulmonary metastatic disease.

Neurological examination was consistent with a multifocal anatomical neurolocalisation. Magnetic resonance (MR) imaging of the brain was performed with a 1.5 Tesla scanner. This showed multifocal intra-axial lesions, which had a solid appearance, stippled mineralization, marked contrast enhancement and no signs of intraparenchymal haemorrhage. Marked perilesional oedema and mass effect were present. Metastatic disease to the brain from the previous diagnosed humeral lesion was suspected.

Owing to the poor prognosis the owners elected to euthanize their dog and gave consent for submission of the brain and biopsies of the humeral lesion for histopathology. Osteosarcoma of the humerus and brain osteosarcoma metastases were confirmed on histopathology.

Metastases from primary bone osteosarcoma to the brain have rarely been reported in veterinary medicine. To the author’s knowledge, this is the first case report describing the MR Imaging features of osteosarcoma metastases in the brain.
We describe the surgical treatment of an epidural haemorrhage due to SRMA and the use of CSF D-dimers in confirmation of the diagnosis.

A one-year-old male mixed breed dog was presented due to acute non-ambulatory tetraparesis, neck pain and pyrexia. 3 Tesla MRI of the cervical spine revealed multifocal T1- and T2-hypointense, extradural masses with mild contrast enhancement, and severe spinal cord compression at the level of C6. A signal void was seen in T2*. This was indicative for an extradural haemorrhage. CSF showed increased protein and erythrocytes. D-dimers were elevated in CSF (3680 ng/ml) and serum (3430 ng/ml). Based on these findings SRMA was suspected. Partial dorsal laminectomy at the level of C6 was performed. Postsurgical treatment included pain medication, antibiotics, gastroprotection, and tranexamicacid in combination with metoclopramide. Corticosteroid therapy was not started because of its side effects on wound healing, instead treatment with cytosine arabinoside was started. The dog returned to ambulation and was discharged 1 week after surgery. An increased immunoglobulin A concentration in CSF (>0.2 µg/ml) confirmed the presumptive diagnosis.

CSF D-dimer concentration in this case might be elevated because of the inflammatory process due to bleeding. It was previously described as an additional diagnostic marker for SRMA.
Intramedullary intervertebral disc extrusion (IMDE) and acute noncompressive nucleus pulposus extrusion (ANNPE) have both been described as causes of acute spinal cord dysfunction in dogs, with distinct magnetic resonance imaging (MRI) findings. The aims of this study were to compare the clinical signs and short-term outcome of dogs diagnosed with presumptive IMDE to dogs with presumptive ANNPE.

In this retrospective descriptive study, 41 MRI scans of dogs presenting with peracute onset spinal cord dysfunction were assessed by three reviewers. Interobserver agreement was calculated with reported MRI characteristics used to diagnose presumptive ANNPE and presumptive IMDE. Only cases with agreement on diagnosis were included in further clinical comparisons. Signalment, activity and possible vocalisation at time of onset, progression of clinical signs, degree of neurological deficits at the onset, IVD affected and four-week follow up were compared between the two groups. Ethical approval was granted (URN: M2016 0083).

Intraclass correlation coefficient of the imaging diagnosis was 0.82. Interobserver agreement was reached in 13 dogs with IMDE and 19 with ANNPE. The only statistically significant difference between the two groups was days of hospitalisation, which was longer in dogs with IMDE (P = 0.028; median and IQR 3 (1-5) vs 8 (1-15).

These findings suggest that a presumptive diagnosis of IMDE based on MRI features may not be associated with more severe neurological signs at time of presentation or with a longer time until ambulation compared to ANNPE. Further studies are needed to evaluate long-term outcome on these cases.
A 1-year-old Labrador presented with acute onset lethargy, neck pain and pyrexia. Six months earlier, a subtotal pericardiectomy had been performed for neutrophilic pericarditis and focal pyogranulomatous epicarditis, from which the dog recovered uneventfully. No abnormalities were detected on pre-referral thoracic radiographs. Cervical hyperaesthesia and quiet mentation were present on physical examination; there were no neurological deficits. Steroid-responsive meningitis-arteritis (SRMA) was suspected. Moderate left-shifted neutrophilia, hyperglobulinemia and increased C-reactive protein (CRP) were documented. Infectious disease (Toxoplasma gondii, Neospora caninum, Dirofilaria immitis, Anaplasma spp., Ehrlichia spp., Borrelia burgdorferi and Angiostrongylus vasorum) testing was negative.

Computed tomography (CT) was performed, revealing a large, non-contrast enhancing mass in the craniodorsal mediastinum with contrast-pooling in several intra-lesional cavitations. This was consistent with a large haematoma. Blood culture was sterile. Coagulation profile and echocardiography were normal. Cerebrospinal fluid analysis revealed neutrophilic pleocytosis. A diagnosis of SRMA was made. Immunosuppressive treatment with prednisolone resulted in resolution of the clinical signs.

Three weeks following referral, the dog was clinically normal, and the CRP within normal limits. Repeat CT scan revealed almost complete resolution of the previously reported haematoma. On three-month follow-up the dog was clinically normal and the CRP within normal limits.

Clinical signs of SRMA result from a combined meningitis and arteritis of leptomeningeal vessels. The arteritis has also been reported to involve the vessels of the heart, mediastinum and thyroid glands. Given its resolution with treatment for SRMA the mediastinal haematoma reported in this case was suspected to represent a form of arteritis.
[P15] IVORY-LIKE VERTEBRA SIGN IN A DOG

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The ivory vertebra sign is defined as uniform sclerosis of a vertebral body, with no alterations in size or contours and no change in the adjacent intervertebral disks. In human medicine, the most common cause is sclerotic metastatic bone disease (prostate, breast and lung cancer). Other differential diagnoses are Paget’s disease, lymphoma or osteomyelitis. To the authors’ knowledge, this is the first report of a dog with changes in the vertebra similar to ivory vertebra in humans causing spinal compression in a dog.

A 11 years-old female spayed Cocker Spaniel was referred for a two months history of ambulatory paraparesis and thoracic hyperesthesia. Neurological examination was consistent with a severe T3-L3 myelopathy.

Survey spinal radiographs showed hyperdensity of the vertebral body T9. Thoracic radiographs and abdominal ultrasound as well as CT of the abdomen and thorax did not reveal a primary neoplasia. Computed tomography of the thoracic spine revealed bone overgrowth and increase density of the vertebral body of T9. The abnormal vertebra was excessively calcified with not osteolytic changes and was invading the vertebral canal causing severe spinal cord compression.

Dorsal laminectomy at T9-T10 with partial corpectomy was performed two months later and bone was submitted for histopathological evaluation which revealed a proliferative lesion affecting spongy osseous tissue with no evidence of malignant neoplasia. Three months after surgery the dog was ambulatory. The main difference with the Ivory vertebra sign reported in humans in this case was the overgrowth of the bone causing spinal cord compression.
[P16] MAGNETIC RESONANCE IMAGING FINDINGS AFTER METALDEHYDE INTOXICATION IN A DOG

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Our purpose is to describe unusual Magnetic Resonance Imaging findings after metaldehyde intoxication in a dog.

A 3 year old female Golden Retriever was referred after a sudden onset of tonic clonic seizures. The dog showed a stuporous state on admission and the neurological examination was consistent with a multifocal intracranial neurolocalization. Blood exams were unremarkable while the MRI showed symmetrical bilateral T2 hyperintense lesions in the caudate nuclei and girus cinguli. The lesions were isointense in T1W images and showed a mild contrast enhancement and a mild mass effect. Cerebrospinal fluid analysis did not show significant abnormalities. The gastric content was toxicologically tested and found to be positive for metaldehyde screening. Thus, a diagnosis of metaldehyde intoxication was made. The dog progressively recovered to normal within 7 days with antiepileptic and supportive therapy. A 2 month-follow up MRI showed small, symmetrically well defined bilateral lesions in the caudate nuclei with a hyperintense signal in T2W images and a hypointense signal in T1W images, which were interpreted as cavitated lesions. The girus cinguli lesions were not detected on the follow up MRI and were thought to be postictal lesions.

To our knowledge, bilateral caudate nuclei lesions detected on MRI have not been reported after metaldehyde intoxication in a dog. Such lesions are described only in a few canine reports due to carbon monoxide intoxication or metabolic and degenerative disorders.

We suggest that metaldehyde intoxication should be included among the differential diagnosis of symmetrical bilateral caudate nuclei lesions detected on MRI.
[P17] ACTINOBACILLUS PLEUROPNEUMONIAE OSTEOMYELITIS LEADING TO VERTEBRAL FRACTURE IN A WEANER

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An eight-week-old female weaner was presented due to a non-ambulatory tetraparesis. The neurological signs were consistent with a lesion in the C6-T2 spinal cord segments.

Radiography and computed tomography of the cervical vertebral column revealed a collapse of the seventh vertebral body, magnetic resonance imaging (MRI) showed extradural well demarcated heterogeneous space-occupying material in the ventral and right-sided aspect of the vertebral canal severely compressing and dislocating the spinal cord. A diskitospondylitis was suspected as underlying cause of the lesion. Post-mortem examination identified a chronic abscess of the seventh cervical vertebral body with subsequent pathological fracture and compression of the spinal cord with associated meningitis and neuritis. In the microbiological analysis, Actinobacillus (A.) pleuropneumoniae was demonstrated using PCR and DNA sequence analysis.

Many terms are used to describe this condition since the pathological findings are highly variable, such as vertebral abscess, vertebral osteomyelitis or spinal abscess. Osteomyelitis is relatively frequent in young pigs and a few bacterial species have been postulated to be the causative agents. To the authors’ knowledge, this is the first report that describes MRI findings in vertebral osteomyelitis in a pig potentially caused by A. pleuropneumoniae.
External hydrocephalus in infants is a benign clinical entity in which macrocephaly is associated with an increase in volume of the subarachnoid space because of excessive accumulation of cerebrospinal fluid (CSF). It is considered a self-limiting condition and is therefore rarely treated.

Only a few cases have been published in veterinary medicine and all but one were surgically treated. And the only canine case reported of external hydrocephalus was diagnosed with computed tomography (CT) images only.

A 4 month-old, male Chihuahua was presented for a month history of behavioral changes, right circling and tetraparesis that had acutely worsened to severely depressed mental status and non-ambulatory tetraparesis following a traumatic event. An evident dome-shaped calvarium was noticed on physical examination at presentation. Magnetic resonance imaging (MRI), CT and ultrasound of the brain demonstrated a generalized, moderate widening of the subarachnoid space between the skull and the neocortex and a moderate enlargement of the lateral ventricles. A small CSF sample obtained from the open fontanel was xanthochromic and displayed mild mononuclear pleocitosis with eritrophagocytosis, consistent with a previous haemorrhage. No organisms were apparent on cytopathological evaluation. The dog improved with medical treatment only.

Treatment consisted of supportive care and one week of antibiotics and nonsteroidal anti-inflammary drugs for the traumatic lesions. Five months after the diagnosis the owner reported that the animal has suddenly died. Necropsy was not allowed.

To the author’s knowledge this is the first report of the MRI characteristics of a suspected benign external hydrocephalus in a dog.
Poster Abstracts

[P19] THE MISERY OF INSUFFICIENT TREATMENT GUIDELINES IN POSTTRAUMATIC EPILEPSY - A CASE REPORT

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Posttraumatic epilepsy (PTE) is well described in human and veterinary medicine. Immediate, early and late PTE are known in men and dogs.

The recommended treatment period with antiepileptic drugs (AEDs) after head trauma is about seven days in human medicine including immediate and early PTE. Up to now standardized protocols for treatment to prevent PTE in dogs are not available.

The current case report underlines the importance of early onset of treatment in immediate and early PTE and the difficulty of preventive treatment of late PTE.

Two dogs were presented after traumatic brain injury (TBI), a Jack Russell Terrier (JRT) kicked by a horse and a Magyar Vizsla (MV) hit by train.

Both dogs developed seizures immediately after the trauma and were successfully treated for one month (JRT) with Imepitoin and for half a year (MV) with Phenobarbital.

Both dogs had no seizures during the whole treatment period. However, after discontinuing the application of AEDs by the owners both dogs developed seizures, leading to death of the JRT in status epilepticus. The MV was treated again with AEDs (Phenobarbital, Imepitoin, Levetiracetam, Potassium bromide), but continued to have cluster seizures every two weeks.

In conclusion, long-term treatment with AEDs in dogs with seizures after TBI is recommended.
[P20] IMMUNE-MEDIATED PYOGRANULOMATOUS GANGLIONEURITIS IN A CAT

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A 7-year-old male DSH was presented with an acute episode of dysuria and plantigradism of two weeks duration. Neurological examination revealed plantigradism, pelvic limb muscle atrophy, ambulatory paraparesis with delayed postural reactions in both pelvic limb, decreased withdrawal reflexes and lumbosacral pain suggesting a L6-S3 lesion. Toxoplasma spp. IgG antibodies were 1/80 and IgM were positive suggesting exposure. Myelo-CT showed a left extradural medullar compression at the level of L6-L7 intervertebral space. Cerebrospinal fluid analysis from a lumbar puncture showed proteinocytologic dissociation. An exploratory left-side hemilaminectomy revealed a left L6 spinal nerve enlargement of which biopsies were taken. The results of histopathology showed a pyogranulomatous ganglioneuritis and radicular edema and Toxoplasma spp. immunohistochemistry was negative. A diagnosis of immune-mediated pyogranulomatous ganglioneuritis was reached. Treatment with prednisone was initiated at 1mg/kg BID and was tapered following clinical remission for eight months. Motor function and muscular atrophy recovery has been almost complete ten months after diagnosis with only slight paresthesia managed with pregabalin remaining.

Common causes of ganglioneuritis and root enlargement in small animals are neoplastic, infectious or immune-mediated; therefore, it is paramount to achieve a definitive diagnosis through the analysis of histopathological samples. In our case, the biopsy revealed a pyogranulomatous inflammation without evidence of infectious agents suggesting an immune-mediated process, and reinforced by the response to immunosuppression. Immune-mediated pyogranulomatous ganglioneuritis should be considered in the differential diagnoses of enlarged spinal ganglia in cats.
[P21] THE PRESENCE OF ADDITIONAL DISC MATERIAL WITHIN THE VERTEBRAL CANAL FOLLOWING FENESTRATION OF INTERVERTEBRAL DISCS DURING HEMILAMINECTOMY

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Intervertebral disc extrusion is a common disease affecting chondrodystrophic dogs. It has previously been reported that fenestration of intervertebral discs can reduce recurrence of disc extrusion and is associated with a low rate of complications. This study aims to ascertain if, and with what frequency, additional disc material may be introduced into the vertebral canal by fenestration of the affected disc following decompressive surgery.

Thirteen dogs presenting to the Queens Veterinary School Hospital, Cambridge that underwent hemilaminectomy and disc fenestration for treatment of intervertebral disc extrusion had intraoperative assessment of the vertebral canal before and after fenestration by a board certified neurologist. The spinal cord was first decompressed by hemilaminectomy and removal of all visible extruded disc material within the vertebral canal. Once no further material was visible manual fenestration of the affected disc was performed. The vertebral canal was then inspected again, and the presence or absence of additional material was noted and included in the surgery report.

Three dogs showed the presence of “new” disc material in the canal post fenestration.

This preliminary study shows that additional disc material can be forced into the vertebral canal by fenestration following decompressive surgery, with a frequency of 23%. This illustrates the importance of checking the vertebral canal after fenestration of an extruded intervertebral disc.
EFFICACY OF RECTAL LEVETIRACETAM AS ADD-ON TREATMENT IN DOGS AFFECTED BY CLUSTER SEIZURES OR STATUS EPILEPTICUS

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The purpose of the study was to evaluate the efficacy of Levetiracetam (LEV) administered per rectum as add-on to a standard protocol of treatment in preventing the onset of further seizures in dogs referred for Cluster Seizures (CS) or Status Epilepticus (SE).

Patients were divided in a control group and a rectal LEV group, and were monitored for 24h after admission for further epileptic seizures. Dogs were defined as “responders” if no further seizures occurred during the 24-h observation period; “non-responders” were dogs that experienced an additional seizure despite treatment.

Fifty-seven patients were included in the study. Dogs assigned to the control group (n = 36) were treated with a standard protocol, comprising rectal administration of diazepam (1–2 mg/kg if dogs were seizuring at presentation); followed by IV administration of Phenobarbital (4–5 mg/kg q8h). Patients included in the rectal LEV group (n = 21), received 40 mg/kg of LEV per rectum in addition to the aforementioned treatment protocol.

The responder rate was 90% (19/21) in the rectal LEV group, compared with 41% (15/36) for the control group (p < 0.001; OR =12.7, Fisher test).

Suspected/confirmed idiopathic epilepsy accounted for 16 patients in the control group (44%) and 12 dogs in the rectal LEV group (57%). A structural etiology was suspected/confirmed in 20 patients (56%) in the control group and 9 dogs (43%) in the rectal LEV group.

Based on the results, rectal LEV seems to offer a potentially useful add-on to the treatment of seizure activity in dogs.

Ethical permission was obtained for the study.
A four-month-old cat was presented due to an acute onset of paraplegia after receiving an injection of cefovecine and dexamethasone in dorsal thoracic area, during which it suddenly became uncooperative.

One day after the injection, the neurological examination revealed a paraplegia without deep pain sensation and reduced segmental spinal reflexes in the pelvic limbs, suggesting either a L4-S1 myelopathy, or a T3-L3 myelopathy with spinal shock.

The magnetic resonance imaging showed swelling of the spinal cord from Th9 to L1 combined with a heterogeneous T2-weighted intramedullary hyperintensity and multifocal, centrally located signal void on T2*-weighted images. These changes were compatible with the suspected traumatic intraspinal injection.

The cat was treated symptomatically for four days without improvement and was consequently euthanized. The findings of the post mortem examination were consistent with a severe segmental hemorrhage and myelomalacia. Histopathology of the spinal cord after hematoxylin and eosin staining revealed a cavity containing some eosinophilic droplets at the edges that could be compatible with an injection trajectory. A necrotic area in the grey matter was also detected. The spinal cord changes were compatible with a traumatic lesion. However, in the surrounding structures no further evidence of blunt trauma, vertebral fracture, disk extrusion or injection was present.

To the authors’ knowledge, this is the first report of a putative intraspinal injection causing myelomalacia. The traumatic myelopathy may be the consequence of either a contusion or - most likely - an intraspinal injection causing vascular rupture.
Fibrocartilaginous embolism within the spinal cord is commonly reported in dogs and occasionally in cats. Fibrocartilaginous embolism affecting the brain has rarely been reported in people and there is only one case report each in a dog and a lamb.

A 12 year old male castrated Siamese cat was evaluated for a one-day history of neurologic signs. Abnormalities on neurologic examination included a decerebellate posture, non-ambulatory tetraparesis, mild obtundation, reduced menace response and palpebral reflex in the right eye, and absent physiologic nystagmus. Postural reactions were absent in the right limbs. Neuroanatomic localization was multifocal CNS including the right cerebellum and brainstem.

A complete blood count revealed a mild lymphopenia, marked elevation in AST, and mild elevation of creatine kinase (CK). A urine culture was negative. Differential diagnoses included vascular disease, neoplasia, and infectious or inflammatory disease.

Nine hours after admission, the cat became agonal and went into cardiopulmonary arrest, and attempts at resuscitation were unsuccessful.

Post-mortem examination revealed multiple fibrocartilaginous emboli (FCE) within the cerebellum and the meningeal vessels ventral to the brainstem.

This is the first reported case of fibrocartilaginous embolism causing an encephalopathy in a cat. While this is a rare disease, it should be considered as a differential diagnosis in cases of acute encephalopathies in cats.
[P25] THE CLINICAL FEATURE OF NEUROLYMPHOMATOSIS IN TWO CATS.

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Neurolymphomatosis is a rare manifestation of lymphoma and is characterized by neoplastic infiltration of the peripheral nervous system.

Case 1: An 8-year-old neutered female Domestic Shorthair cat was presented for a month history of progressive non-ambulatory monoparesis of left thoracic limb. MRI revealed an enlarged the left brachial plexus and the swelling the left C7 and C8 spinal nerves. Histopathology, neoplastic lymphocytes involved the C8 spinal nerve. Immunohistochemically, the neoplastic lymphocytes were positive for CD20. The cat was treated with the UW-25 chemotherapy protocol and the clinical symptoms were improved. However, on the 77th day from the chemotherapy starting, the thoracic limbs paraparesis occurred. A co-administration of nimustine and irradiation was started. Although these therapies were effective, the owner did not request further therapies. The cat died the 100th day. A necropsy was not permitted.

Case 2: A 9-year-old neutered male Scottish Fold was presented for a month history of progressive non-ambulatory monoparesis of left pelvic limb. MRI revealed swelling the left C8 and L7 spinal nerves. The owner requested the palliative treatment with prednisolone and the clinical symptoms were improved. However, on the 107th day from the MRI examination, the thoracic limbs paraparesis occurred. The cat died the 231th day. At necropsy, both the left C8 and the right T1 spinal roots were swollen. Histologically, neoplastic lymphocytes extensively involved not only the spinal cord, but also the left frontal lobe of the cerebrum and the peripheral spinal nerves. Immunohistochemically, neoplastic lymphocytes were positive for CD20.

Further consideration of the management strategy for feline neurolymphomatosis is needed.
[P26] AN INVESTIGATION INTO THE ASSOCIATION BETWEEN BODY CONDITION SCORE AND OUTCOME IN MINIATURE DACHSHUNDS UNDERGOING HEMILAMINECTOMY FOR TREATMENT OF THORACOLUMBAR INTERVERTEBRAL DISC EXTRUSION

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Miniature Dachshunds are at a high risk of developing thoracolumbar intervertebral disc extrusion (TL-IVDE). Predisposing factors including body condition score (BCS) have been identified, but the effect of BCS on outcome has not been evaluated. The aim of this study was to evaluate the effect of BCS on rate of recovery and outcome of miniature Dachshunds having undergone hemilaminectomy surgery for treatment of TL-IVDE.

This was a prospective observational study including 37 miniature Dachshunds diagnosed with surgical TL-IVDE. Along with patient variables, a BCS (1-9) and open field score (OFS, 0-14) was recorded preoperatively for each dog. Postoperatively OFS was recorded for each day of hospitalisation, as well as at 4-week re-examination. Outcome was recorded as overall OFS change over 4 weeks, OFS change per day of hospitalisation and number of days hospitalised. Institutional ethical approval was received.

Median recorded BCS was 6, with 30 dogs (81%) having a BCS of between 5 and 7. Median preoperative OFS was 4 (range 0-13), median OFS change per day hospitalised was 0.7 (range 0.0-2.0) and median final OFS was 11 (range 0-14). No significant association was found between BCS and any of the recorded outcome measures in this study population.

These findings suggest that BCS may not have a significant effect on outcome and rate of recovery in miniature Dachshunds undergoing surgical management of TL-IVDE. The statistical power of the analysis was limited by the low variation in BCS observed and therefore low number of cases in several of the BCS groups.
[P27] EVALUATION OF PROGNOSTIC FACTORS FOR CATS WITH SACROCAUDAL LUXATION

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The aim of this retrospective study was to evaluate potential prognostic factors for cats with sacrocaudal luxation or ‘tail pull injury’.

Medical records and radiographs were reviewed for cats diagnosed with sacrocaudal luxation. Information obtained from the clinical records included signalment, clinical presentation, treatment details, outcome and survival time. Severity of neurological signs was graded from 1 to 5, based on previous grading systems for cats with sacrocaudal luxation. Degree of sacrocaudal displacement was calculated on survey radiographs. Outcome was collected from serial neurological examinations and interviews with referring veterinary surgeons and owners. Cats had to be given a minimum of 30 days to regain urinary function to be included in this study.

Seventy cats were included. Fifty-five of 61 cats (90%) for which this information was available, regained voluntary urinary function. Higher neurological grade was significantly associated with poor urinary outcome ($P<0.01$) and longer time to regain voluntary urinary control ($P=0.0003$). No significant associations were found between urinary outcome and age, sex, anal tone, perineal sensation, tail base sensation, degree of craniocaudal or dorsoventral sacrocaudal displacement, concurrent orthopaedic injury, tail amputation, faecal function at diagnosis, and survival. Patients that could defaecate voluntarily had longer survival times than patients that lacked voluntary control over defaecation ($P=0.03$).

In agreement with previous studies, neurological grade was the most reliable prognostic indicator, which may also provide useful information when advising owners on the expected time frame for recovery. Faecal incontinence may be a more important predictor for long-term survival than previously suspected.
[P28] CONGENITAL MALFORMATIONS OF THE LUMBOSACRAL VERTEBRAL COLUMN IN NEUROLOGICALLY NORMAL FRENCH BULLDOGS, ENGLISH BULLDOGS AND PUGS AND THEIR ASSOCIATION TO TAIL MALFORMATION.

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The aims of this study were to evaluate the prevalence and anatomical characteristics of lumbosacral congenital vertebral malformations (CVM) in French bulldogs, English bulldogs and Pugs presenting for problems unrelated to spinal disease. Furthermore, a possible association of these CVMs to the degree of tail malformation was investigated.

In this retrospective cross-sectional study, computed tomography scans of the caudal lumbar, sacral and coccygeal vertebral column were reviewed for presence and location of different types of CVMs (hemivertebrae, block vertebrae, transitional vertebrae and spina bifida) and degree of tail malformation.

In 84 (56.4%) of the 149 included dogs (53 French, 37 English bulldogs and 59 Pugs) at least one type of CVM was found, with lumbosacral transitional vertebrae being the most common (34.2%). Pugs demonstrated significantly more often lumbosacral transitional vertebrae (54.2%) and significantly less often hemivertebrae (1.7%) compared to English (13.5% and 24.3% respectively) and French bulldogs (26.4% and 32.0% respectively). Tail malformation was significantly more severe in dogs with evidence of hemivertebrae.

As previously described, CVMs are a common finding especially in the thoracic vertebral column of these three breeds, but this also seems to be true for the lumbosacral vertebral column. These anatomical variances need to be considered carefully when planning for neurosurgical and neurodiagnostic procedures. Furthermore, this study suggests a possible link between degree of tail malformation and presence of hemivertebrae. The clinical relevance of this needs to be further investigated and this could potentially be used to establish breeding guidelines to reduce the prevalence of hemivertebrae.
Paroxysmal dyskinesias (PD) are characterised by recurrent episodes of sudden involuntary abnormal movements or muscle tone and preserved consciousness, with multiple breed-specific conditions reported in dogs. The aim of this study was to document a novel breed-specific PD in the Welsh Terrier, characterise the phenotype and clinical course, and estimate the prevalence.

Welsh Terrier owners were invited online to complete a questionnaire to determine the clinical characteristics, precipitating factors and clinical course of a suspected paroxysmal dyskinesia in this breed. Institutional ethical approval was obtained.

Questionnaires were completed for 177 Welsh Terriers in the study. Paroxysmal episodes were reported in 76 (42.9%), of which 44 (24.9%) were classified as PD based on owner description and video footage where available. Episodes were characterised by limb dystonia (n=37, 84%), writhing movements with abnormal body posture (n=17, 39%) and occasionally abnormal head posture (n=4, 9%), with preserved consciousness. Median age at onset of signs was 57 months (range 8-156), with no sex predisposition (P=0.60). Median frequency of episodes was 2 per month (range 0.1-30). Stress, excitement or exercise was the most frequent suspected precipitating factor (n=8, 18%), although most occurred at rest (n=31, 70%) and owners could not predict them. Of owners that could comment, most dogs displayed a stable (40%) or improving (32%) clinical course, with owner-perceived quality of life typically unaffected (n=28, 66%).

The findings of this study describe a previously unreported paroxysmal dyskinesia in the Welsh Terrier. Further studies are required to explore a potential genetic basis and underlying aetiology.
[P30] MRI CHARACTERISTICS IN 20 FRENCH BULLDOGS WITH NECROTIZING ENCEPHALITIS

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Description of MRI features of French Bulldogs with necrotizing encephalitis is restricted to case reports or scattered within studies involving several breeds. We retrospectively evaluated MRI characteristics of 20 French Bulldogs.

Dogs were suspected to have encephalitis with inflammatory CSF and one or more MRI lesions (n=11) or normal CSF, but multiple small MRI lesions (n=2). Additionally, 7 dogs diagnosed by brain biopsy were included.

Of dogs with suspected encephalitis, 12/13 dogs had multifocal MRI lesions. Forebrain was involved in 11/13, brainstem in 8/13, thalamus in 7/13, cerebellum in 4/13 and optic nerve in 1/13. Lesions involved grey and white matter in 10/13. Midline shift was present in 3/13.

Of dogs with brain biopsy, 3/7 had a focal MRI lesion. Forebrain was affected in 7/7, thalamus in 2/7 and cerebellum in 2/7. Lesions involved grey and white matter. 2/7 had a midline shift and 1/7 showed foraminal herniation. CSF was non-inflammatory in 7/7 and 2/7 showed albuminocytologic dissociation.

All lesions were T2 and FLAIR hyperintense, T1 iso- to hypointense and contrast enhancement varied from none to strong, being inhomogenous and slight in most cases.

Forebrain lesions were consistently present (90%). About 50% had additional lesions in thalamus and brainstem. Interestingly, cerebellum was involved in 30%. Without brain biopsy, 3 dogs with a focal lesion and normal CSF would have been more suspicious for neoplasia than for encephalitis. Brain biopsy here showed that immune-mediated encephalitis can also present with focal lesions, without CSF changes or albuminocytologic dissociation only.
[P31] SAROLANER INTOXICATION IN A YORKSHIRE TERRIER PUPPY

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Sarolaner (Simparica™) is a novel oral isoxazoline for the control of flees, ticks and mites in the dog. We report about a 3-month-old male Yorkshire Terrier presenting with signs of intoxication affecting the neurologic and pulmonary system after drug overdose.

The owner administered a 80 mg tablet (considered for 20-40 kg BW) for flea control to the puppy with a body weight of 2.5 kg. Five hours later, the dog started to show fasciculations and tremor. Eight hours after ingestion, the dog showed the first of two generalized seizures. Permanent salivation and respiratory distress occurred. The dog was presented 19 hours after ingestion.

Upon presentation the puppy showed pale mucous membranes, prolonged capillary refill time, sinus bradycardia of 80/min, salivation, dyspnea, a respiratory rate of 60/min, wheezing and muffled heart sounds. Neurologic exam was unremarkable except for a negative menace response. Initial diagnostic tests showed mild neutrophilic leukocytosis without left shift and hypokalemia. Diagnostic imaging showed alveolar lung patterns and air bronchograms dorsal to the hilus, suspected to be either edema or pneumonia.

The dog received oxygen, theophylline, furosemide, atropine, intravenous fluids, potassium substitution, diazepam, levetiracetam and antibiotics. One more seizure occurred in the following 24 hours. The dog was discharged neurologically unremarkable after 4 days with regressive lung patterns and normal respiration.

This is the first report about Sarolaner intoxication in a dog. During licensing procedures, selflimiting tremors and convulsions occurred. This case additionally presented with neurologic lung edema or excessive bronchial secretion. Seizures occurred for 40 hours after ingestion, possibly due to the elimination half-life of 12 days.
Zinc phosphide is known to affect various organ systems, including the gastrointestinal tract, the respiratory tract, the cardiovascular system and the central nervous system. In most cases of intoxication, only one or two of those are affected.

It is a report of a 10 year old female German Spitz, which was observed while ingesting zinc phosphide containing mouse bait. It experienced clinical signs within thirty minutes of ingestion, starting with severe vomiting. The following six hours, additional symptoms as reduced mentation, generalized seizure activity and coughing developed. The patient was not responsive to its environment during the following twelve hours.

Initial diagnostic tests including complete blood count, coagulation profile and blood gas analysis did not reveal any significant abnormality, while blood chemistry showed mild elevation of alkaline phosphatase, cholesterol and glutamate-pyruvate transaminase. On thoracic radiographs a megaesophagus and decreased transparency in the cranial lung tissue was seen. The latter was consistent with non-cardiogenic toxic edema or aspiration pneumonia.

The patient remained on the ICU for forty-eight hours, receiving intravenous fluid therapy, phenobarbital and levetiracetam, activated charcoal and omeprazole per os. Gastric lavage was not performed since toxin ingestion happened hours ago. The patient recovered completely within two days and was discharged without detectable neurological deficits. There were no abnormal findings in the control radiographs of the thorax.

This case is the first to describe a megaesophagus in a dog with zinc phosphide intoxication and one of the few cases in which more than one organ system was affected.
A 9 year-old, intact male Shih tzu dog was presented with systemic weakness and a per-acute onset of tetraplegia. On neurological examinations, tetraplegia with lower motor neuron signs was notified. Myasthenia gravis was ruled out based on the normal findings of supramaximal repetitive nerve stimulation with electromyography and neostigmine administration. Initial 0.3 T-magnetic resonance imaging (MRI) findings included relatively sharply demarcated intramedullary lesions at C2 to C6, mainly involving grey matter, which appeared hypointense on T1-weighted images (WI) and hyperintense on T2-WI and FLAIR. There was no enhancement on post-contrast T1-WI. Neutrophilic pleocytosis was observed in cerebrospinal fluid analysis. No clinical responses were observed for the treatment with immunosuppressive dosage of prednisolone. Six-day follow-up 3-T MRI demonstrated hyperintensity on diffusion-weighted image (DWI) and decreased apparent diffusion coefficient (ADC) value of the spinal lesions. Through histological examination, fibrocartilaginous embolism (FCE) was definitively confirmed.

This report firstly describes an ischemic spinal injury visualized by DWI with high-field MRI in a chondrodystrophic dog with FCE.
Orthostatic tremor (OT) is a rare movement disorder that causes instability and psychological stress in humans. A tremor with a 13–18 Hz frequency that occurs in the limbs when standing is pathognomonic and part of the definition of OT. When documented in conjunction with other movement disorders, the term ‘orthostatic tremor plus’ is coined. Here are reported two cases of OT plus in geriatric Jack Russell Terriers.

Dog 1 (16-year-old) displayed very rapid tremors in all limbs in a standing position. Dog 2 (12.5-year-old) displayed very rapid tremors in hind limbs when standing. Both dogs also displayed a hypermetric gait, most apparent in the pelvic limbs. Tremors ceased upon movement or when extensor muscles were not loaded. No intention- or head tremor was noted. Electrophysiological testing in dog 1 revealed a tremor frequency of 13 Hz consistent with OT. Oral administration of gabapentin resulted in reduction of tremor severity.

These are the first two reported ‘OT plus’ cases in canines. OT has been reported in young giant breed dogs. In humans, age of onset varies, but most patients are presented around 60 years of age. In older dogs, a benign, idiopathic, rapid postural tremor (BIRPT) or ‘senile tremor’ of pelvic limbs is reported. This was considered in both cases reported here, but not deemed an appropriate diagnosis due to clinical findings. Indeed, BIRPT cases might encompass OT cases. Especially since diagnostic tests are not often performed in many of those older patients and the term ‘idiopathic’ is not substantiated.
[P35] AN ASSESSMENT OF THE CUTANEOUS TRUNCI REFLEX IN NEUROLOGICALLY NORMAL CATS

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The cutaneous trunci reflex (CTR) is an important part of the neurological examination and yet it can be very inconsistent in cats. Although there are studies investigating the CTR in dogs, there is a gap in the feline literature. We aimed to evaluate the reliability of the CTR in neurologically normal cats, and investigate if stress or age had any influence. This study has welfare and ethical approval.

Inclusion criteria were neurologically normal cats older than 6 months with a normal hydration status and no history of neurological or orthopedic disease. Cats underwent a subjective assessment of stress level and were categorised into ‘relaxed’, ‘stressed’ or ‘very stressed’, with the latter group being excluded. An ECVN boarded neurologist assessed the cats and performed a neurological examination including assessment of mentation, gait, postural reactions, patellar reflex, and CTR. Any cat showing neurological abnormalities other than CTR abnormalities was excluded. Age, stress level and CTR results were recorded. CTR was defined as normal, if easy to elicit and present bilaterally, or abnormal if difficult to elicit or absent.

Of the 26 cats, only 9 were classified as normal. Five cats had bilateral absent CTR. No statistical significance was found when correlating abnormality with age or stress level. However 4 of the 5 cats with absent CTR reflex were classified as “stressed” and 4 were older than 10yrs.

The presence of the CTR in our cat population was inconsistent and preliminary results suggest an influence of external determinants.
An 8 years old male English Setter dog was presented to the neurology unit of Policlinico Veterinario Roma Sud for evaluation of a one year history of progressive neck pain.

At neurological examination gait, postural reactions and spinal reflexes were normal but severe neck pain was evident, consistent with C1-C5 neurolocalization. The most likely causes were intervertebral disc herniation, neoplasia and inflammatory diseases.

Cervical MRI showed, at the level of the occipital-atlanto-axial joint, a multilobulated T2 and STIR hyperintense and T1 hypointense mass with a well-defined capsule extending from the caudal aspect of the occipital bone to the mid third of the vertebral body of C2. Morphology and distribution of the mass reflected that of the occipito-atlanto-axial synovial membrane. This mass showed a marked capsular enhancement and enhancement of the adjacent muscles.

Biopsy of the lesion displayed a multiloculated, poorly cellular, proliferation of large stellate cells embedded in abundant myxoid matrix. CD18 immunohistochemistry highlighted a neoplastic proliferation of type A synoviocytes that together with growth pattern was consistent with synovial myxosarcoma.

Myxosarcomas are rare neoplasms of fibroblastic origin that were previously reported in middle age or older dogs affecting the heart, the eye and brain, the stifle, tarsus, carpus and spine. The prognosis for myxosarcoma in dogs is poor because of a high rate of local recurrence and metastasis.

Nine months after diagnosis the dog is still alive.

The purpose of this report is to describe the first case of myxosarcoma affecting the composite occipito-atlato-axis joint in a dog.
[P37] CEREBELLAR DEGENERATION ASSOCIATED WITH PRIMARY CNS LYMPHOMA IN A YOUNG CAT

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Cerebellar degeneration (CD) or abiotrophy is the premature degeneration of the cerebellar cell population, caused by a probable intrinsic metabolic abnormality and is considered a rare neurologic condition in domestic cats. Lymphoma is mentioned as the second most frequent intracranial neoplasm in young cats. In humans, CD is reported as a rare paraneoplastic syndrome in children with Hodgkin’s lymphoma. CNS Lymphoma associated with CD has not been reported in small animals previously. The present report includes an 8-months-old male, shorthair domestic cat presented with clinical signs of altered consciousness, symmetric ataxia, vertical positional nystagmus, mydriasis, strabismus, intention tremor of the head, and increased patellar reflexes. Neuroanatomic diagnosis indicated a diffuse cerebellar lesion. Blood count analysis showed thrombocytopenia and lymphopenia. Cerebrospinal fluid analysis indicated mononuclear and neutrophilic pleocytosis. Contrast CT imaging revealed multiple hypodense heterogeneous areas in both cerebral hemispheres, mild ventriculomegaly at the level of the caudal fossa, and a circular sharply marginated, homogeneously hyperdense mass occupying the right cerebellar hemisphere. Post-mortem study indicated a 5 mm x 1 mm mass in the right cerebellar hemisphere close to the vermis. Histopathological analysis showed diffuse and severe loss of Purkinje cells with a decrease in the density of granular cells and moderate gliosis. Additionally, numerous neoplastic lymphoid cells were observed in the infiltrated mass consistent with a diagnosis of lymphoma. Immunohistochemistry revealed expression of CD20 and a B-cell immunophenotype. This report is the first described case of possible paraneoplastic cerebellar abiotrophy associated with CNS lymphoma in a cat.
[P38] CHARACTERIZATION OF NEUROLOGICAL ABNORMALITIES IN 97 DOGS WITH SUSPECTED PITUITARY DETECTABLE MASSES

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Pituitary tumors (PT) may cause neurological dysfunction by compressing/invading the adjacent central nervous system structures. However, consistent data regarding the detailed neurological presentation of dogs with PT are lacking. Hence, the purpose of the study is to provide a detailed description of the neurological abnormalities in a large population of dogs with a suspected pituitary detectable mass and relate them with the size of the mass using the pituitary height/brain area ratio.

Dogs were retrospectively included if they had a magnetic resonance imaging (MRI) showing a lesion compatible with a pituitary detectable mass (PDM) and a detailed neurological examination (AHT 10-2015). The neurological signs were evaluated in relation to the pituitary height/brain. In addition, neurological signs were correlated with the presence or absence of MRI signs of brain compression.

The study included 97 dogs. All dogs showed at least one neurological abnormality, the most common being abnormal mentation (79%), abnormal gait (61%) and cranial nerves deficits (44%). Out of these, abnormal postural and proprioceptive reactions (48%), head and cervical pain and/or hyperesthesia (25%) and postural abnormalities (21%) have not been previously reported. The majority of dogs with a pituitary detectable mass had signs of brain compression. The presence of an high pituitary height/brain area and brain compression represented a risk factor for developing mental status abnormalities.

Our results show that dogs with PDM have a variety of neurological abnormalities which should be adequately considered in order to plan a correct diagnostic and therapeutic work-up.
[P39] LEVETIRACETAM AS FIRST-LINE THERAPY IN DOGS WITH SUSTAINED STATUS EPILEPTICUS: A PRELIMINARY STUDY

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Diazepam (DZ) is the standard first-line treatment for Status Epilepticus in dogs. Sustained epileptic activity causes a sharp decline in DZ therapeutic efficacy which worsened over 30 minutes probably due to the SE-associated subunit-specific trafficking of GABA₆. Levetiracetam (LEV) is a new antiepileptic drug, whom mechanism of action is not related to GABA₆ receptors. Hence, the aim of this study was to evaluate the therapeutic efficacy of LEV and compare that with DZ in dogs with SE established for ≥30 minutes (ID 817-2017).

10 dogs were prospectively enrolled in the study. 5 were treated with LEV (60 mg/kg IV; group A) and 5 were treated with DZ (1-2 mg/kg IV; group B). Efficacy was defined as cessation of seizures soon after the bolus administration.

3/5 of dogs treated with LEV showed a complete cessation of seizure activity whereas only 1/5 of dogs treated with DZ was responsive. 4/5 of dogs treated with LEV were seizure-free during hospitalization. 1/5 of dogs in group B died because of complications related to the SE. Of the remaining dogs in group B, 3/4 had further seizures during hospitalization. To obtain 24h-seizure-free status, a mean of 5h (±7h) was necessary in the group A and a mean of 24h (±22h) was necessary in group B.

This study suggests LEV can be therapeutically more effective than DZ in dogs with sustained SE. Further studies are necessary in order to confirm this preliminary observation.
[P40] NEUROLOGICAL AND ELECTRODIAGNOSTIC EVALUATION IN A DOMESTIC CHICKEN (GALLUS GALLUS DOMESTICUS) WITH TRAUMATIC ISCHIATIC NERVE INJURY

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We report the neurological exam, electrodiagnostic findings, treatment and outcome of a case of traumatic ischiatic neuropathy in a domestic chicken.

A 1 year-old hen was referred for a two-weeks acute and non progressive history of abnormal gait in the left limb after being entangled in a wire fence. Neurological examination revealed ambulatory left limb monoparesis with dragging of the foot on the dorsal aspect. Postural reactions were absent in the left hindlimb and there was a marked reduction/absence of the left withdrawal, cranial tibial and gastrocnemius reflexes. Neurolocalization was left lumbosacral spinal cord segment, spinal nerve roots, spinal nerve and sciatic nerve. The asymmetry of the clinical signs and the history of trauma strongly suggested a left ischiatic nerve lesion.

The hen was put under general anesthesia and a needle electromyography performed; there was diffuse spontaneous activity in the left ischiatic nerve distribution muscles.

In order to limit self-trauma the left distal limb was bandaged with the toes extended. The patient’s clinical conditions progressively improved and a complete recovery of the motor function was obtained in two months. The hen recently died for unrelated cause: it was killed by a fox.

To author’s knowledge this is the first description of neurological evaluation and electrodiagnostic findings of traumatic ischiatic nerve injury in a chicken. In our opinion, a favorable prognosis must be considered in this patients and the electrodiagnostic test can confirm the clinical suspicions also in avian species.
In humans, multiple autonomic nervous system disturbances (ANSd) compound the course of Guillain-Barré syndrome (GBS). In ACIP, ANSd refer mainly to the laryngeal function and are little data regarding the effect over cardiovascular function.

A 5-year-old male Siberian Husky, with a five-days history of progressive acute nonambulatory flaccid tetraparesis, preserved perianal reflex and urinary continence and no impairment of cranial nerves was presented to the VTH-Iasi. Blood tests showed a mild leukocytosis, increased CK (3454 U/L) and 1.46 albumin/globulin ratio. CSF proteins and cells were in normal limits. Fibrillation potentials and positive sharp waves in both pelvic and thoracic limb musculature, reduction in MCS, SCS and F wave latency with normal repetitive stimulation (sciatic and tibial nerve) were found on electrodiagnostic studies. Complete cardiological examination revealed idiopathic arterial hypertension (AHT) (SAP 214±19 mmHg) only and no previous cardiovascular history was reported. ANS evaluation through heart rate variability analysis showed withdraws of vagal tone with preserved of the sympathetic one. A definitive diagnosis of ACIP and AHT was established and amlodipine treatment and physiotherapy were started. The arterial pressure decreased but remained constantly above the 150 mmHg, until the dog became ambulatory (day 51). Amlodipine was stopped 2 weeks after the dog recovered the ambulatory status. Blood pressure remains in normal range until the last follow-up (22 weeks amlodipine free) sustaining the hypothesis that AHT was ACIP induced.

To our knowledge this is the first report which describes AHT as a clinical presentation of ACIP and resembles similarities with GBS-AHT.
Degenerative lumbosacral stenosis (DLS) in dogs is similar to human degenerative lumbar spinal disease, as both affect the cauda equina and can cause neurological deficits and lower back pain. Surgical procedures aim to decompress and/or stabilize the lumbosacral joint. Lumbar interbody fusion technique, by using a cage is the most indicated in human medicine, but few studies exist about cage implantation between these vertebrae in veterinary medicine.

Cadaveric lumbosacral spines from ten adult large dogs (20-35kg), with and without radiographic/tomographic signs of DLS, that died for reasons unrelated to this study were used. It was performed an anatomical, radiographic and tomographic evaluation on the lumbosacral joint of the spine of these dogs, to determine whether a human cage model had adequate dimensions for this joint and to develop a cage prototype for dogs. The vertebral body dimensions and the L7-S1 intervertebral space occupied by the intervertebral disc were measured by lateral and ventrodorsal radiographs and by computed tomography in the dorsal, sagittal, and transverse views. Measurements were also taken of the anatomical specimens in the sagittal and transverse planes.

The human lumbar cage models from LDR, Baumer Orthopedics, Stryker, Synthes, and Vertebal Technologies Inc. brands and cervical cages from B-Braun and Stryker brands were evaluated and were found to be unsuitable for large dogs. Thus, two cage models with different shapes were developed using 3D modelling software. Both models had similar measurements and were designed for insertion via dorsal laminectomy and discectomy in the dorsolateral portions of the intervertebral space.

Approved - Institutional Ethics Committee on Animal Use, protocol 9360.2016.64
[P43] ANATOMICAL, RADIOGRAPHIC, TOMOGRAPHIC AND VOLUMETRIC STUDY OF THE SKULL AND CRANIOCERVICAL JUNCTION OF NEUROLOGICALLY NORMAL DOGS WITH AND WITHOUT DORSAL NOTCH IN THE FORAMEN MAGNUM

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The skull, brain and craniocervical junction of small dogs may have anatomical abnormalities, which can cause neurological diseases. Caudal occipital malformation syndrome is characterized by foramen magnum cerebellar herniation, which was suggested to be a consequence of overcrowding of the neural structures within the caudal fossa, that could cause mechanical pressure bone resorption of the supraoccipital bone.

The objective of this study was the post mortem evaluation of twelve small dogs without neurological disorders presenting (n=6) or not (n=6) foramen magnum alteration (occipital dysplasia). Radiographic, tomographic and anatomical study of the skull, brain, cervical spine and craniocervical junction were performed. The total volume of the cranial cavity and caudal cranial fossa volume and the ratio between them were also calculated.

The values of cranial volume, caudal fossa and ratio between them were the same in both groups, and these values showed a positive correlation with the weight of the dogs. It was not observed syringohydromyelia and the only differences between both groups were the presence of the dorsal notch in the foramen magnum as well anatomical variation in the shape of the occipital bone, external occipital prominence, external sagittal crest and nuchal crest, differences resulting from the skull morphology. In dogs with the dorsal notch in the foramen magnum it was observed a fibrous membrane covering it and there was no cerebellar herniation in both groups.

In dogs with or without alterations in the foramen magnum size, with normal volume of the caudal fossa, we did not observe cerebellar herniation.

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It has been reported that findings of X-ray myelography in dogs with intervertebral disc diseases (IVDD), especially deficits of cerebrospinal fluid (CSF) line due to swelling of the spinal cord, would associate with prognosis. Similar myelographic images could be obtained using MR myelography (MRM). The purpose of this study is to evaluate MRM findings in dogs with IVDD would associate with severity of neurological signs.

Thirty-four dogs with IVDD were included in this study. MRM were obtained after conventional MRI scan. In all dogs, area of the spinal cord and extruded disc material at lesion site were measured on T2-weighted transverse images and neurological severity were obtained from medical record. The length of CSF line deficits was measured on MRM and L2 ratio was calculated from the length of deficit and L2 vertebrae. Relation between deficits of the line and severity, and correlation between the areas and severity were statistically analyzed.

There were significant relations between length of CSF line deficits and severity (P=0.002). However, no significant correlation between area of the spinal cord and length of CSF line deficit, and area of the disc material and the length were observed. In two dogs with progressive myelomalacia, L2 ratio were 20.7 and 20.5, longer than those in parapregic dogs without symptom of the myelomalacia (mean 5.47).

The length of CSF line deficit on MRM would associate with severity of clinical signs in dogs with IVDD and deficits of CSF line for long distance might indicate progressive myelomalacia.
[P45] MRI FEATURES OF SUBACUTE LAMINAR CORTICAL NECROSIS IN A DOG FOLLOWING SUSPECTED ANAESTHESIA-RELATED BRAIN HYPOXIA

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Laminar cortical necrosis is a consequence of severe hypoxic, ischaemic or hypoglycaemic event in the brain, occurring in the most vulnerable cerebral cortical layers. In humans, MRI features follow a consistent temporal course. In the late subacute stage, lesions in the cortex are of increased T2-signal-intensity (SI) and of increased T1-SI due to accumulation of denatured proteins or lipid laden macrophages. The lesions undergo variable contrast enhancement. Limited information reporting MRI findings is available in dogs affected by laminar cortical necrosis.

A 3-year-old Belgian shepherd dog was referred 8 days after sudden onset of blindness following general anaesthesia for a dental procedure. Neurological examination showed bilateral central blindness and mild ataxia. A 3-Tesla MRI examination of the brain revealed bilateral non-symmetrical areas of T2 and FLAIR increased SI within the occipital, parietal, temporal and frontal cortex, involving grey and white matter. Furthermore, linear T1-hyperintense lesions were found in the cerebral cortex of the same areas undergoing moderate, heterogeneous contrast enhancement. Presence of increased SI in DWI with low ADC values demonstrated restricted diffusion in the affected cortical grey matter.

The lesions were compatible with subacute laminar cortical necrosis with corresponding cytotoxic oedema suspected to be secondary to anaesthesia-related brain hypoxia. 3-Tesla MRI enabled clear identification laminar pattern of the cortical lesion and associated restricted diffusion.
USE OF PCR FOR ANTIGEN RECEPTOR REARRANGEMENTS TO DISTINGUISH LYMPHOMA AND INFLAMMATORY PROCESSES IN FOUR CASES WITH LYMPHOCYTIC CSF PLEOCYTOSIS IN DOGS

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PCR for antigen receptor rearrangements (PARR) of lymphocytes gained from tissue aspirates or body fluids is considered to be a useful test to differentiate lymphoma and inflammatory disease and for immunophenotyping lymphoma cells.

In four dogs with progressive neurological signs, intracranial or spinal MRI-alterations and severe lymphocytic pleocytosis, PARR was used to detect clonality in native CSF samples. In dog 1 (CSF: 14000 cells/3µl, 60% lymphocytes), PARR of CSF revealed monoclonal B-cell proliferation. Cytology of a concurrent perianal mass indicated an extraneural component of lymphoma. The dog was euthanized four weeks after diagnosis of stage V lymphoma. In dog 2, CSF pleocytosis (2530 cells/3µl, 100% lymphocytes) with biclonal T-cell proliferation and an extramedullary lumbar spinal mass indicated T-cell lymphoma. After 7 months with palliative therapy, the dog was euthanized due to neurological deterioration. Dog 3 was diagnosed with meningoencephalomyelitis of unknown origin after detection of multifocal lesions in MRI affecting brain and spinal cord and pleocytosis (6300 cells/3µl, 87% lymphocytes) in CSF. PARR revealed polyclonal B- and T-cell populations. The dog improved under immunosuppression (prednisolone, ciclosporine) and is stable 6 months after diagnosis. In dog 4, diagnosis was idiopathic pachymeningitis. In CSF, pleocytosis (85 cells/3µl, 86% lymphocytes) and polyclonal T-cell population in PARR occurred. Oral prednisolone was administered for 7 months. The dog is clinically normal apart from unilateral central blindness 15 months after diagnosis.

In conclusion, PARR seems to be a promising additional CSF examination tool in dogs with lymphocytic pleocytosis and can assist to confirm the diagnosis.
[P47] MRI FEATURES OF EXERTIONAL MYOPATHY IN A GREYHOUND

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We describe the case of a 4 year old, female neutered greyhound, presented for investigation of thoracolumbar hyperalgesia one week after extreme exertion. MRI of the thoracolumbar spine revealed muscle changes consistent with exertional myopathy. The MRI features of this disease are previously unreported.

This dog’s exercise was exclusively lead walking, but she escaped and ran for four miles, developing severe pain, unresponsive to carprofen, tramadol and prednisolone, but responsive to gabapentin. Serum creatinine kinase was markedly elevated. The dog was presented to us one week after the incident, and pain was elicited on palpation of the thoracolumbar spine.

On MRI, diffuse T2W and STIR signal hyperintensity extended bilaterally over the longissimus and iliocostalis muscles from the T5 to L7 vertebrae. These were mildly hyperintense on T1W and strongly contrast enhancing. Gradient echo images showed no signs suggestive of haemorrhage. The muscles were markedly swollen. No other muscle groups were affected. The changes were consistent with muscle oedema, attributed to exertional myopathy. Myoglobinuria was not present. This dog made a full recovery following 2 weeks of rest and analgesia, and remained normal with a follow-up period of 8 months.

Key points from this case are that exercise induced myopathy causes marked, diffuse hyperintensities on T2W and STIR images, with strong contrast enhancement. In cases with focal pathology and without myoglobinuria, an excellent outcome can be achieved with medical management.
The Dandy-Walker malformation complex refers to a group of congenital central nervous system malformations that primarily involve the cerebellum and adjacent tissues. This malformation consists of complete or partial agenesis of the cerebellar vermis, cystic malformation of the fourth ventricle and an enlargement of the caudal fossa. In some cases, substantial enlargement of the caudal fossa is not observed and is referred to as Dandy-Walker variant. To the author’s knowledge this kind of malformation has not been reported yet in cats.

A 8-month-old male Domestic Shorthair cat was referred for an uncoordinated gait seen by the owners since birth. General physical examination was unremarkable. Neurological examination revealed generalized ataxia with hypermetria of all four limbs, wide-based standing, and intermittent intention head tremors. Neurological examination was consistent with a cerebellar lesion. Hematology, full biochemistry and FeLV/FIV serology did not reveal abnormalities.

MRI sequences of the brain showed a large retrocerebellar fluid accumulation associated with an enlarged fourth ventricle. These findings were associated with cerebellar hypoplasia and partial atrophy/absence of the caudal portion of cerebellar vermis. CSF analysis was performed and was unremarkable. PCR analysis of Toxoplasma and Coronavirus was negative in CSF. After 3 months of diagnosis, clinical signs were similar with a good quality of life. Similar MRI findings have been reported in Dandy-Walker malformation in dogs or inferior cerebellar hypoplasia in purebred Eurasier dogs.
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Traumatic skull fractures are relatively frequent in dogs and cats, but there is little information regarding their epidemiological, clinical and advanced imaging features. The aim of this comparative, retrospective, multi-institutional study was to describe the neurological and computed tomographic (CT) features of a large cohort of dogs and cats with traumatic skull fractures.

Ninety-four dogs and 101 cats with traumatic skull fractures identified on CT were included. Cause of trauma, signalment and presence of neurological deficits were recorded. Fractures were classified according to their location (affected bone and region: skull vault, skull base or face) and evidence of fragmentation, displacement and depression.

Road traffic accident (RTA) was the most common cause of trauma in both dogs and cats. In dogs, mean age was 3.5 years, the most commonly affected breeds were Chihuahua and Crossbreed with 55% being males, and 54% having neurological deficits. The frontal bone was the most affected, 55% had fractures in a single region with the cranial vault being overrepresented. Fractures were fragmented in 51.6%, displaced in 32.8% and depressed in 29.1%.

In cats, mean age was 4.9 years, Domestic Shorthair was the most common breed with 65% being males, and 46% having neurological deficits. Sixty-nine percent of fractures affected the face, and 78.4% had more than one region affected simultaneously, with the maxillary and pterygoid bones most commonly affected. Fractures were fragmented in 52% cases, displaced in 27.8% and depressed in 20.5%.

Skull fractures have different features in dogs and cats.
[P50] CHRONIC INFLAMMATORY DISTAL POLYNEUROPATHY WITH AN ODD PSEUDOHYPERMETRIC GAIT IN A MINIATURE PINSCHER

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Peripheral neuropathies are the most challenging nervous system disorders as they require specific diagnostic investigation and may remain elusive regarding the underlying etiology, even when a disease entity is confirmed histopathologically. The main pathological changes observed are axonal degeneration and demyelination, which may occur in combination.

An eight-year-old, male intact, client-owned vaccinated Miniature Pinscher was admitted because of chronic progressive gait abnormality that appeared six months prior to referral. Neurological examination revealed a palmigrade stance, distal tetraparesis more severe to the thoracic limbs, a gait abnormality including hypermetria and laxity of both carpal and tarsal joints and bilateral distal muscle atrophy of thoracic limbs (flexor carpi ulnaris and extensor carpi radialis) and pelvic limbs (tibialis anterior). Orthopedic examination, routine clinicopathological testing, including serology for protozoal diseases and cerebrospinal fluid analysis were unremarkable. Electromyographic testing revealed focal spontaneous electrical activity limited to the extensor carpi radialis muscle. Histopathological examination of biopsies showed diffuse, chronic, severe muscle atrophy and diffuse, chronic-active, moderate to marked, lymphocytic-plasmocytic neuritis/perineuritis and interstitial myositis. The dog is being re-examined every six months for two years, showing a slowly progressive clinical deterioration, even though it still remains ambulatory.

Clinical and histopathological findings were compatible with inflammatory distal polyneuropathy of the chronic progressive or relapsing motor and sensory peripheral neuropathy groups. These diseases have been associated with inflammatory changes in the spinal nerve roots, the cranial or the more peripheral regions of the appendicular nerves. The slowly progressive disease course and the distinctive gait abnormalities were the most prominent features in this case.
Osteopetrosis is a genetically determined bone disease that develops as a result of malfunction of osteoclastic activity leading to excessive deposition of immature bone, thickening of cortical bone and narrowing of the medullary cavities.

An 18 month-old male Cavalier King Charles Spaniel presented for evaluation of cervical hyperesthesia. Brain and spinal MRI revealed a thickening and sclerosis (markedly hypointense signal on T1 sequences) of the calvaria with complete absence of bone marrow in the bones of the skull and vertebral bodies. There was severe Chiari-like malformation with herniation of the cerebellum and medulla into the foramen magnum. The occipital bone was markedly dysplastic and was compressing the caudal aspect of the cerebellum. The medulla was compressed by the cerebellum dorsally and by the dens of the axis ventrally. Spinal MRI revealed syringomyelia within the C2-C3 and C5-L3 segments. The maximal width of a syrinx (4.8mm) occurred at the level of T4, at which level very little spinal cord parenchyma remained. Radiography confirmed thickening of the cortices and narrowing of the medullary cavities of the appendicular skeleton. Haematology confirmed a non-regenerative anaemia consistent with myelophthisis.

We hypothesise that a mass effect from the calvarial thickening leads to reduced volume of the caudal fossa and cerebellar herniation. The absence of osteoclastic activity may lead to abnormal growth of the occipital bone resulting in a smaller caudal fossa. This is the first case report where MR findings in a patient with osteopetrosis associated with Chiari-like malformation are described in veterinary medicine.
[P52] CLINICAL, MAGNETIC RESONANCE IMAGING, AND HISTOPATHOLOGIC FEATURES OF A HYPOGLOSSAL MALIGNANT PERIPHERAL NERVE SHEATH TUMOR IN A MALTESE DOG

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A three-year old male Maltese dog presented with a one-week history of occasional neck pain and progressive tetraparesis. On neurologic examination the dog showed a depressed mental status, left pleurothotonus, vestibular ataxia, left proprioceptive deficits, left eye ventrolateral strabismus and left lingual hemiatrophy with ipsilateral deviation.

Magnetic resonance imaging of the brain showed a well defined extra-axial mass on the left side of the caudal fossa with extracranial extension along the course of the hypoglossal nerve. The mass was hyperintense on the T2-weighted images, isointense on the T1-weighted images, and markedly enhanced after contrast administration, with a core of presumed necrotic tissue.

The owner elected for euthanasia. The removal of the brain revealed the presence of an ovaloid firm mass of approximately cm. 1.5x1.2x0.6, compressing the left ventral surface of medulla oblongata. The mass extended into a widened hypoglossal canal with a tubular, pink to grey appearance along the course of the XII CN.

At histological examination the hypoglossal nerve was diffusely infiltrated by neoplastic elongate cells arranged in Antony A and Antony B patterns. The most solid pattern was characterised by hypercellularity of pleomorphic spindle cells showing marked nuclear atypia and up to 4 mitotic figures/HPF. Large areas of necrosis were present. Neoplastic infiltration did not occur neither in the brainstem, nor in the tongue. Based on histological findings a diagnosis of MPNST was made.

To our knowledge this is the first report describing a hypoglossal MPNST in a dog.
[P53] CERVICAL SYRINGOMYELIA SECONDARY TO SINGLE SPACE-OCCUPYING INTRACRANIAL LESIONS IN DOGS: MAGNETIC RESONANCE IMAGING FINDINGS AND RISK FACTORS

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The purpose of this study is to investigate potential risk factors for the development of secondary cervical syringomyelia (SCSM) in dogs affected by single intracranial space-occupying lesions.

This is a retrospective single-cohort study. The database of two referral hospitals was searched for dogs undergoing brain and cervical spinal cord MRI for investigation of intracranial neurological signs. Dogs diagnosed with a single intracranial space-occupying lesion were included and divided in two groups based on the presence or absence of SCSM. Breed, age at the time of MRI, and cranial morphology were compared between the two groups. The MR studies of both groups were reviewed by a single operator, and the following parameters were assessed and compared: mass localization and volume, perilesional edema volume, mass effect, ventriculomegaly, and presence and severity of cerebellar herniation.

Four significant risk factors for development of SCSM were identified: mass volume (p < 0.0001), mass effect (p 0.031), ventriculomegaly and presence and severity of cerebellar herniation (p < 0.0001). Additionally, dogs affected by intracranial lesions having a relative volume higher than 0.028 were 2.01 more likely to develop SCSM.

This study has identified risk factors for the development of SCSM in dogs with a single space-occupying intracranial lesion. When one or more of these risk factors are identified, it is advisable to extend the MRI study to the cervical spine, to investigate the presence of SCSM. Conversely, in dogs with SCSM and no concurrent cervical or congenital caudal fossa disease, the MRI study should be extended to the brain to investigate for a potential mass lesion.
[P54] IDENTIFICATION OF OWNER-PERCEIVED SEIZURE TRIGGERS AND BEHAVIOURAL PREDICTORS PRIOR TO SEIZURE ACTIVITY IN DOGS WITH IDIOPATHIC EPILEPSY

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Epilepsy is the most common chronic brain disease in dogs. Currently, seizures are considered spontaneous due to lack of understanding of the transition between the inter-ictal and the ictal stages. In humans, prodromal activity (e.g. change in behaviour or mood) and seizure triggers have been reported, however this is yet to be explored in dogs. A better understanding of these factors may improve our understanding of epilepsy and enhance management options.

We designed an online questionnaire-based study to identify whether owners believe they can predict forthcoming seizure activity in their dog, to report observed pre-seizure changes and seizure triggers. Owners of 228 dogs with Tier I idiopathic epilepsy completed the survey, with 136 (60%) reporting that they could predict an upcoming seizure. The top three reported prodromal activities were increased clingingness, restlessness and signs of fear behaviour. The majority of owners detected these clinical signs at least 5 minutes before seizure activity, and up to 1 week before. No significant association was found between reported prodromal behaviour and breed or age. Triggers were less frequently reported, with stress, food and excitement being the most commonly reported. No significant association was found between triggers and breed or age.

In conclusion, many owners of dogs with epilepsy perceive both a prodromal phase in their dogs and seizure triggers. Investigation of reported behavioural and physiological changes is required to further identify pending seizure activity and whether a sufficient window of time exists to allow use of therapeutics to prevent an upcoming seizure.
Degenerative Lumbosacral Stenosis (DLSS) is an acquired multifactorial condition affecting dogs. Different treatments modalities are available with surgery appearing to provide a better outcome. Lumbosacral epidural-steroid injection (ESI) has been described in a single previous study in dogs. ESI provides a more targeted therapy than oral medication, being applied closer to the affected nerve roots avoiding systemic effects. These advantages allied with rapid response to treatment, gives ESI the potential of being utilised as a predictive test for subsequent surgical management outcome.

A prospective study was undertaken of 9 dogs presented with DLSS over one year. All patients underwent advanced imaging including CT and MRI for confirmation of a diagnosis of DLSS and an ESI before surgical management. Decompressive surgery included a combination of uni/bilateral foraminotomy and dorsal laminectomy. Seven out of 9 patients improved following ESI, and 3/9 had a complete resolution of clinical signs. In all patients improvement was short-lived, with recurrence within 3 to 18 days. After decompressive surgery all patients improved, of which 4/9 had a complete resolution of clinical signs.

Improvement following epidural injection does not seem to predict short-term outcome following decompressive surgery. All cases that improved followed ESI also improved following surgery, with the 3 cases that failed to respond to ESI responding to surgical management.

This study confirms that ESI is a safe technique producing short-term good responses in cases of DLSS. However, ESI appears not to be useful as a predictor of short-term post-surgical outcome in dogs suffering from DLSS.
Intradural-extramedullary spinal tumours and extradural osteosarcomas are both rare entities. We describe a case of canine primary intradural-extramedullary osteosarcoma of the cervical spine.

An 11-year-old male neutered crossbreed dog presented with progressive left forelimb lameness and proprioceptive deficits in the left fore- and pelvic limb over 4 weeks. Neurological examination revealed ambulatory proprioceptive ataxia in all four limbs and decreased withdrawal reflex in the left forelimb consistent with a C6-T2 neurologicalisation.

A CT-scan of the neck revealed a large quantity of left-sided mineralised material within the dorsolateral vertebral canal at C6-C7. An MRI scan confirmed this lesion as a heterogeneous mixed signal-intensity on T2-weighted images with minimal contrast enhancement. An extradural lesion was suspected with a disc extrusion or tumour being more likely.

The dog underwent left-sided dorsolateral hemilaminectomy between C6 and C7. The lesion was found to be intradural and so was debulked via durotomy and sent for histopathological examination. The dog recovered well from surgery and was ambulatory within 3 weeks. The diagnosis from histopathology was an osteosarcoma. Chemotherapy consisting of 10mg/kg of carboplatin intravenously every three weeks was started post-operatively. The dog remains ataxic six months post-operatively but with a good quality of life.

Primary intradural-extramedullary osteosarcomas of the spine are extremely rare. This is the first case described in a dog. In human medicine there are very few cases described and no established therapeutic approach has been determined. This patient responded well to a debulking surgery and chemotherapy.
[P57] MORPHOLOGICAL EVALUATION OF THE VESTIBULE ON MR CISTERNOGRAPHY IN DOGS WITH IDIOPATHIC VESTIBULAR DISORDER

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Although endolymphatic hydrops of the internal ear and vestibular neuritis are suspected, the cause of idiopathic vestibular disorder (IVD) in dog is unknown. In human patients with Meniere's disease (MD), which is thought similar to IVD, extended reuniting duct of the vestibular was observed in affected side on MR cisternography. The purpose of this study is to evaluate the morphology of the vestibular structures on MR cisternography in dogs with IVD which did not show any abnormality on conventional MR images.

Nine dogs with IVD and six dogs with idiopathic epilepsy were included in this study. In each dog, structures of the vestibular was assessed and the volume was measured blindly on MR cisternography using commercially available software.

In seven dogs with IVD, swelling of the reuniting duct were observed in affected side, while those structure were thought to be normal in unaffected side. In all dogs with idiopathic epilepsy, those structure were assessed as normal. The sensitivity of swelling of the reuniting duct on MR cisternography in IVD was 77.8% and specificity was 81.0%. There was no significant differences in the volume of the vestibular structure between affected and unaffected side.

This study suggested that morphological changes in the vestibular structures had occurred in dogs with IVD similar to human MD because abnormal swelling of the reuniting duct was observed. Measured volume of the vestibular was not significantly different between lesion and unaffected side because the structure was too small to precisely measure the volume.
[P58] OUTCOME OF ACUTE SPINAL SUBPERIOSTEAL HEMATOMA IN THREE GRAYHOUNDS AFTER CONSERVATIVE TREATMENT

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The purpose of this study is to describe spinal subdural hematoma (SSDH) in three Greyhounds treated conservatively. SSDH is a vascular disorder causing compression of the spinal cord and acute onset of neurological symptoms. In veterinary medicine this pathological condition is poorly documented. One case of SSDH in a Greyhound has been published.

A seven-year old male neutered Greyhound was presented for acute paraparesis followed by deterioration in the subsequent hours. Neurological examination revealed paraparesis grade IV, hind limbs proprioception deficit and normal spinal reflexes. no discomfort on palpation of the spine and paraspinal muscles was observed. The neuroanatomical localization T3-L3 myelopathy. MRI imaging findings were compatible with subperiosteal hematoma at L2 vertebra.

A four-year old male neutered Greyhound had a similar history with acute paraparesis at onset with rapid deterioration within few hours. The neuroanatomical localization was again T3-L3. MRI findings were compatible with dorsal extradural haemorrhage at T11-T12.

The third Greyhound was a four-year old male neutered and the clinical signs were similar. In this dog MRI imaging described a dorsal extradural lesion at T11-T12.

The MRI imaging revealed subperiosteal extradural mass causing lateral displacement of the spinal cord, homogeneous hyperintensity on T2W and STIR, intermediate hyperintensity on T1W sequences. Although surgical treatment has been documented, all our patients had conservative management including NSAIIDs(one dog) and physiotherapy(three dogs). All dogs had a good functional recovery 4-5 weeks after onset of clinical signs.
[P59] PERIPHERAL VESTIBULAR PAROXYSM IN A DOG: IS IT A NEW ENTITY?

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Vestibular syndrome in humans can be acute, chronic or episodic; the latter can be triggered (benign paroxysmal positional vertigo, central paroxysmal positional vertigo, vestibular paroxysmia) or spontaneous (vestibular migraine, Meniere’s disease, transient ischaemic attack). Vestibulogenic/vertiginous epilepsy is another under-recognised non-lesional pharmacoresponsive condition caused by lesions in the vestibular representation in the frontal cortex. In young dogs, congenital vestibular disease has been reported, however it is not episodic.

A four-month-old male entire client-owned vaccinated Hungarian Vizla-cross was referred for paroxysmal episodes consisting of wide-based stance, right compulsory trunk sway, right head tilt, disorientation, generalised muscle tremor and spontaneous horizontal nystagmus without post-ictal changes. No relation with the head position was reported.

Physical examination revealed bilateral otitis externa (treated with topical medication), whilst neurological examination was unremarkable. Clinicopathologic evaluation and arterial blood pressure were unremarkable. Neurolocalisation was compatible with right peripheral vestibular system. Despite the resolution of otitis externa, the episodes frequency increased up to once a week. Further investigations included head CT and CSF analysis and were normal. Levetiracetam, Phenobarbital and Imipetoin were ineffective. After three years the dog still experiences daily vestibular paroxysms.

In this case, congenital vestibular disease, ear disease or structural encephalopathy are considered unlikely due to their permanent nature. Vestibulogenic seizure is unlikely due to lack of pharmacoresponsiveness. Vestibular migraine and transient ischaemic attacks are of central origin, whilst benign paroxysmal vertigo is related with the head posture. Thus, the dog may experiences a spontaneous episodic (peripheral) vestibular syndrome similar to the human condition called Meniere’s disease.
Assessment of cats with suspected spinal disease can be daunting for first opinion practitioners. Although successful treatment is only possible after reaching an accurate diagnosis, it is not always possible to refer feline spinal patients for advanced diagnostics. The aim of this retrospective study was to evaluate if a combination of easily identifiable clinical characteristics can be used to obtain a reliable differential diagnosis in cats with spinal disease.

221 cats referred for further evaluation of spinal disease were included and categorised into the following disease categories: neoplasia (n=44), intervertebral disc disease (IVDD) (n=42), fracture/luxation (n=34), ischaemic myelopathy (n=22), feline infectious peritonitis virus myelitis (n=18), lymphoma (n=16), vertebral canal stenosis (n=11), acute non-compressive nucleus pulposus extrusion (n=11), traumatic spinal cord contusion (n=8), spinal arachnoid diverticula (n=7), lumbosacral stenosis (n=5) and spinal empyema (n=3). Information retrieved from the medical records included signalment, clinical history and clinical presentation. Univariate analyses of variables (clinical history, breed, age, gender, general physical examination findings, onset, deterioration, spinal hyperaesthesia, asymmetry and neuroanatomical localisation) were performed, and variables were retained in a multivariate logistic regression model if \( P < 0.05 \).

Multivariate logistic regression revealed that IVDD most often occurs in middle-aged, purebred cats with a normal general physical examination and an acute onset of painful and progressive clinical signs. Ischaemic myelopathy occurs most often in older cats with a stable or improving, non-painful, lateralising, C6-T2 myelopathy.

This study suggests that using easily identifiable clinical characteristics can assist in obtaining a preliminary differential diagnosis when evaluating cats with spinal disease.
[P61] SURGICAL TREATMENT OF THORACIC KYPHOSIS BY DISCECTOMY, VERTEBRAL DISTRACTION AND STABILIZATION USING A POLIAXIAL (PAX) LOCKING PLATE SYSTEM IN A PUG.

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Kyphosis is a commonly detected condition in the thoracic spine of screw tails dog breeds. These malformations might lead to instability, spinal cord compression and secondary neurological signs. Surgical management of this condition include segmental spinal stabilization using vertebral body pin and polymethylmethacrylate or interlocking plate fixation which might be associated to decompression of the impinged spinal cord.

A seven months-old, female, pug dog was presented to our hospital with a 2-weeks history of paraplegia and normal deep pain perception of the pelvic limbs. MRI scan revealed T7 hemivertebra with kyphosis at T6-T7 leading to canal stenosis and spinal cord compression. A left seventh intercostal space thoracotomy was performed. After a discectomy of the T5-T9 segment, a pair of blunt Gelpi retractors was placed at T5 and T9 ribs’ head which allowed spine distraction and subsequently reduction of the kyphosis. A 6 holes 2,4 PAX plate with four locking screws was used to stabilize the T5-T9 spinal segment. Three-months after surgery the dog presented a paraparetic ambulatory gait and continued improving since the surgery.

Discectomy, vertebral distraction and stabilization of the affected spinal segment with a locking plate system is a promising alternative for stabilization of thoracic kyphosis in dogs with secondary neurological signs. Main advantages of this technique include the less invasiveness of the approach compared with bilateral approaches, the natural redimensioning of the spinal canal after reducing the kyphosis and the use of a titanium, versatile implant system which allow screw angulation for an accurate implant placement.
Spontaneous regression of intervertebral disc extrusion (IVDE) has been reported in people particularly in the lumbar region. Regression of IVDE has also been reported in dogs after variable periods of medical treatment.

A four-year-old female neutered French Bulldog was presented for repeated episodes of neck pain. Neuroanatomical localisation was to a C1 – T2 myelopathy. Magnetic resonance (MR) imaging revealed a C4 – C5 intervertebral disc herniation. The dog was hospitalised for a week on cage rest and then discharged for a further three weeks of cage rest and analgesia. Her owners reported complete resolution of her pain episodes over this time. Neck pain recurred one week later while on medical management and the dog was re-presented. A neurological examination localised once more to a C1 – T2 myelopathy. A second MR scan was performed which revealed a new IVDE at the level of the C3 – C4 intervertebral disc space. The initial herniation at C4 – C5 had regressed. The dog had ventral slot decompressive surgery performed at the level of C3 – C4 and at a re-examination one month later was neurologically normal.

This report supports the concept that regression of cervical IVDE can occur rapidly after a period of medical management, but notably illustrates that a further herniation is possible even during a period of strict cage rest.
[P63] THE MENACE RESPONSE: DIAGNOSTIC UTILITY

Will Humphreys¹, Lorenzo Golini¹, Massimo Mariscoli¹, Luca Motta¹

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The menace response has long formed part of the neurological examination in human and veterinary patients. The aim of this study was to provide an evidential basis to quantify the diagnostic utility of the response as part of the neurological examination and make an assessment of how this varies with neurolocalisation.

This retrospective study correlated radiologist reported MRI findings for 405 veterinary patients presented to referral hospital with the presence or absence of the menace response as reported during neurological examination at admission. Anonymous data analysis was carried out to characterise the diagnostic test.

The prevalence of intracranial disease in the examined population was 65.7% (266/405 cases). The overall sensitivity of the menace response was 34.6% (28.9-40.6% p = 0.05). The overall specificity of the test was 76.3% (68.3-83.1% p = 0.05). Subset analysis of test results in patients with intracranial lesions at MRI outlined how test characteristics change with neurolocalisation. Likelihood ratio analysis indicates that the menace response possesses little to no diagnostic value for lesions in the myelencephalon. It is of highest utility for multifocal lesions, and lesions in the forebrain, whereby a positive result is a useful indicator of a possible intracranial lesion. The post-test probability of forebrain and multifocal lesion diagnosis is increased by 15-20%.

We propose that an absent menace response is a practical indicator of intracranial disease, but that a normal menace response does not equivocally exclude intracranial pathology. We also propose that the diagnostic utility of the test result varies with intracranial neurolocalisation.
General Information

Conference website  www.vetneuro2018.org

Conference venue  Hotel Scandic Copenhagen
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1601 Copenhagen V, Denmark

Badges  The conference name badges must be worn at all times during the conference. Access to the conference venue will not be granted without the name badges issued by the conference organisers.

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Conference language  The conference will be held in English.

Information for Speakers  Please bring your presentation to the auditorium at least two hours before your presentation starts. A technician will help you upload the presentation to the computer. Please bring your presentation on a USB stick. We do not allow the use of personal laptops for presentations. At the end of the conference, all presentations will be deleted in order to secure that no copyright issues will arise.

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Social Events

WELCOME RECEPTION
Thursday, 20 September 2018
19.00 - 21.30
Tycho Brahe Planetarium
Gammel Kongevej 10
1610 Copenhagen V

ESVN-ECVN 2018 GALA DINNER
Friday, 21 September 2018
19.30 - 24.00
Visit Carlsberg
Gamle Carlsberg Vej 11
1799 Copenhagen V
General Information

Information for poster presenters

The poster sessions are held in the lunch and coffee breaks:

Friday 21 September
10.00-10.30, 12.30-13.45 & 15.30-16.00.

Saturday 22 September
10.00-10.30, 12.55-14.15 & 15.45-16.15.

Please be present at your poster to answer questions from fellow participants during these times.

Please note that we expect all posters to be set up before 10.00 on Friday 21 September and taken down between 16.15-18.30 on Saturday 22 September.

Programme & Abstract book

You will receive a printed programme and abstract book when you arrive at the conference, but you will also be able to download the book from the conference website from 20 September 2018.

Exhibition

In the breaks, please visit the exhibition, where all the companies who have supported the organization of the conference will be showcasing their activities, research and products. The exhibition is located in the areas surrounding the main auditorium.
Floor Plan

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