European Society of Veterinary Neurology
European College of Veterinary Neurology

Program of the 19th Annual ESVN & ECVN Symposium
Barcelona, 29th–30th September, 2006

Index of Abstracts

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ABSTRACT # 1

IS C-REACTIVE PROTEIN A VALUABLE TOOL IN DIAGNOSIS AND TREATMENT CONTROL IN DOGS WITH STEROID RESPONSIVE MENINGITIS-ARTERIITIS? A. Bathen-Nöthen[1], R. Carlson[1], A. Tipold[1], [1]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, GERMANY.

Steroid responsive meningitis-arteriitis (SRMA) is characterized by meningeal inflammation and arteriitis, and in addition has features of a systemic disease. The diagnosis and treatment control in dogs with SRMA is based on the examination of cell count, cell differentiation and IgA level in the cerebrospinal fluid (CSF) in addition to clinical features and IgA level in serum. The puncture of CSF is performed in general anesthesia. The aim of the current study was to evaluate C-reactive protein (CRP) for its value as diagnostic tool and for treatment control without the necessity of CSF examination in SRMA.

Recently, CRP was established as a major acute phase protein in dogs with inflammatory diseases. In preliminary studies, we could show that CRP seems to be more sensitive in diagnosing inflammation than macroglobulins and albumin. In addition, CRP values are helpful to differentiate between bacterial and viral meningitis in human medicine.

CRP was determined in 129 paired serum and CSF samples using an ELISA (Tridelta Development Ltd., UK): 27 dogs with SRMA, 13 with other meningo encephalitides, 32 with intervertebral disk disease and degenerative lumbosacral stenosis, 24 with tumors of the CNS, 25 with idiopathic epilepsy and 8 healthy dogs as negative control. Samples from 6 dogs with sepsis served as a positive control. In 21 dogs with SRMA treatment monitoring was performed (serum- and CSF-CRP values, CSF cell count and serum and CSF IgA levels). In addition, in all dogs further parameters were evaluated, such as blood cell count, total protein, albumin, liver enzymes, serum IgA, IgA in the CSF, CSF cell count, cell differentiation, and CSF total protein.

Serum-CRP was significantly higher in dogs with SRMA and sepsis than in dogs with other neurological diseases ($p<0,001$). In addition, serum-albumin levels were significantly lower in dogs with SRMA ($p<0,01$) compared to dogs with other neurological diseases. In the SRMA group, serum-CRP levels correlated with serum alkaline phosphatase ($r=0,49$, $p<0,01$), which - as CRP - seems to be induced by inflammatory mediators. CRP values in the CSF were nearly undetectable in other diseases than SRMA. In dogs with SRMA CSF-CRP values correlated with CSF cell count ($r=0,49$, $p<0,01$). During treatment controls CRP-values decreased in the same amount as the number of CSF cells.

In the present study we could show, that CRP in serum and CSF are markedly elevated in dogs with SRMA, supporting the hypothesis that SRMA is a systemic disease. Measurement of CRP serum levels might be useful to support the diagnosis of SRMA and to monitor treatment of this disease. However, it still remains necessary to exclude other systemic inflammatory diseases as a differential diagnosis.

ABSTRACT # 2

OXIDATIVE STRESS IN DOGS WITH STEROID RESPONSIVE MENINGITIS-ARTERIITIS (SRMA). M. Beiner[1], R. Carlson[1], H.-P. Sallmann[2], A. Tipold[1]. Dept. of Small Animal Medicine and Surgery[1], Biochemistry[2], University of Veterinary Medicine, Hannover, GERMANY.

The pathogenesis of steroid responsive meningitis-arteriitis (SRMA) in dogs is not completely understood. Immunopathological mechanisms seem to play an important role, because of high levels of IgA in CSF and serum and the benefit of glucocorticosteroid treatment. Because of high values of neutrophils in CSF and blood the production of oxygen radicals is feasible and may lead to the phenomenon of oxidative stress. Therefore, the aim of the current study was to elucidate the role of oxidative stress in the pathogenesis of SRMA. In pretrial studies HPLC to measure malondialdehyde (MDA) as an indicator for the harmful influence of oxidative stress towards unsaturated fatty acids in the canine CNS was found to be the best method in regard of sensitivity and reproducibility.

MDA was determined in 92 paired CSF and serum samples: from dogs with SRMA ($n=12$) and dogs with SRMA under treatment ($n=13$). These MDA levels were compared to those of dogs with epilepsy ($n=13$), with discopathy ($n=14$), with CNS-neoplasia ($n=16$), with CNS-inflammation ($n=20$) and with canine distemper encephalitis ($n=4$). In addition the following parameters were measured: IgA and leucocytes in CSF and blood samples and protein levels in CSF. Characteristic pleocytosis and elevated levels of IgA in serum and CSF could be determined in SRMA compared to other diseases.

MDA-levels in CSF of all groups were very low and elevated only in chronic diseases. However, compared to other neurological diseases MDA levels in serum of dogs with SRMA were significantly higher (up to 4 - 7 fold) than in dogs with other diseases ($p=0,01$ SRMA versus epilepsy; $p=0,02$ SRMA versus discopathy). A correlation between MDA-levels in serum and CSF was realized only in SRMA-patients (SRMA $p=0,003$; SRMA under treatment $p=0,05$). There was no correlation between MDA, IgA and leucocytes in serum as well as in CSF of SRMA-patients. MDA levels in serum decreased after application of glucocorticosteroids in dogs with SRMA.

The current findings suggest that oxidative stress has an influence on the pathogenesis of SRMA and supports the hypothesis that SRMA starts as a systemic disease and expands later into the meninges. In respect to clinical signs and treatment of SRMA the current results let suggest the application of glucocorticosteroids in SRMA patients leads to a reduction of oxidative stress and most probably prevents the transition of the acute in the chronic form of SRMA either through preventing damage of CNS vessels or of genetic material and development of autoantigens.

ABSTRACT # 3

CEREBRAL ANGIOGRAPHY TO EVALUATE EFFECTS OF SIMVASTATIN AND CYCLOSPORINE ON CEREBRAL VASOSPASM IN A CANINE MODEL OF SUBARACHNOID HEMORRHAGE. Coates JR, Bulsara KR, Agrawal VD, Eifler DM, Wagner-Mann C, Durham ED, Toft K. Department of Veterinary Medicine – College of Veterinary Medicine, Division of Neurosurgery – School of Medicine, University of Missouri, Columbia, MO, U.S.A.

Potential consequences of subarachnoid hemorrhage (SAH) include cerebral vasospasm. SAH can result in impaired endothelium-dependent relaxation mediated by nitric oxide and an intense inflammatory response that parallels the time course for cerebral vasospasm. We proposed that simultaneous upregulation of nitric oxide synthase with simvastatin and suppression of inflammation with cyclosporine would ameliorate cerebral vasospasm to a greater extent than simvastatin alone.

A double subarachnoid hemorrhage model was induced in dogs by 2 injections of autologous blood into the cerebellomedullary cistern 24 hours apart. Dogs were assigned to one of three groups: Control-untreated ($n=5$); simvastatin (Zocor, Merck Inc.; 20 mg/kg SID PO) only ($n=4$); simvastatin (20 mg/kg SID PO) and cyclosporine A (Sandimmune, Sandoz Inc.; 6 mg/kg SID PO) ($n=4$). Medications were administered 24 hours after the second injection for 10 days. Cerebral angiography was performed on days 0, 3, 7, and 10 post injection. An iodinated contrast agent was injected into the

vertebral artery, and lateral and ventrodorsal views were captured. Measurements of the basilar artery diameter were taken from just distal to the confluence of the vertebral arteries to just proximal to the bifurcation of the caudal cerebral arteries. The basilar artery was divided into 10 approximately equal segments in length of which individual vessel diameter measurements were obtained using units and scale of pixels in the Photoshop™ program. A mean average of the narrowest regions of the basilar artery was taken for the 40%, 50% and 60% data points. Data were analyzed within groups using the paired t-test or one-way repeated measures ANOVA and among groups using a two-way repeated measures ANOVA. ($p<0.05$) On day 10, dogs were euthanized. Neurologic examination was normal in all dogs throughout the study. Scleral hemorrhage was noted in most dogs within 24 hours after second injection which resolved in most dogs by day 10. Decreased basilar artery diameter was seen on day 3 in control and simvastatin/cyclosporine groups. A return to baseline diameters was seen by day 7. An increase from baseline diameter was seen in the simvastatin group at day 10. Cyclosporine may interfere with the vasodilatory effects of simvastatin. Vasodilation above baseline is seen at day 10 in the simvastatin only group. Combination of simvastatin and cyclosporine does not ameliorate cerebral vasospasm in a canine model to a greater extent than simvastatin alone.

ABSTRACT # 4

EFFECTS OF SIMVASTATIN AND CYCLOSPORINE ON CEREBROSPINAL FLUID ANALYSIS IN A CANINE MODEL OF SUBARACHNOID HEMORRHAGE INDUCED VASOSPASM DOCUMENTED BY CEREBROVASCULAR ANGIOGRAPHY. DM Eifler[1], JR Coates[1], KR Bulsara[2], C Wagner-Mann[2], AB Royal1, LM Berent[1]. University of Missouri[1]. [1]College of Veterinary Medicine and 2School of Medicine, Columbia, MO

Cerebral vasospasm due to subarachnoid hemorrhage (SAH) leads to morbidity and mortality in humans suffering from stroke and may play a role in veterinary patients with cerebrovascular disease. Impaired nitric oxide release and inflammation may contribute to development of vasospasm. Simvastatin, which upregulates nitric oxide synthase and cyclosporine, which suppresses inflammation may decrease vasospasm. The purpose of this study was to investigate effects of simvastatin and cyclosporine on basilar artery vasospasm using cerebral angiography in a proven canine model of SAH and to examine relationships between CSF changes and vasospasm. Serial evaluations of CSF have not previously been described using this model.

Three mls of autologous blood was injected into the cerebellomedullary subarachnoid space 24 hours apart. CSF was collected from the cerebellomedullary cistern before each injection and on days 3, 7, and 10. Angiography was performed prior to initial injection and on days 0, 3, 7, and 10. Routine CSF analysis was performed. Thirteen dogs were studied: untreated control dogs (n=5), dogs treated with simvastatin and cyclosporine (SC) (n=4), dogs treated with simvastatin (S) alone (n=4). Drug treatment was initiated 24 h after the second injection. Statistical analyses were performed using one and two-way analysis of variance for repeated measures or the paired t-test.

Angiography demonstrated basilar artery vasospasm on day 3 only in controls and Group SC. This resolved in both groups by day 10.Vasodilation was observed in group S on day 10. CSF analysis revealed significantly elevated total protein (TP) and red blood cell (RBC) count in all groups 24 hours post initial injection (day 1), returning to near baseline levels by days 7–10. Total Nucleated Cell Count (TNCC) was significantly elevated in controls and Group SC on day 3. All groups had inflammatory CSF by day 3 which declined to near baseline levels by day 10. A Mixed or neutrophilic pleocytosis was most commonly observed.

In conclusion, simvastatin alone prevented vasospasm to a greater degree than treatment with both simvastatin and cyclosporine, compared with controls. This corresponded with the decreased CSF inflammatory cell response observed in dogs treated with simvastatin alone compared to other groups. SAH in all instances resulted in elevations in CSF RBC count, TP, and TNCC in all groups. Despite observed trends, variability and low number of dogs may have obscured statistical significance in some instances. RBC count, TP, and TNCC declined in all groups to near original values within 10 days. Clinically, it is important to recognize time for normalization of RBC count, TP, and TNCC after SAH as this could otherwise interfere with interpretation of CSF analysis.

ABSTRACT # 5

AN *IN VITRO* INVESTIGATION INTO THE EFFECT OF VENTRAL SLOT SURGERY ON VERTEBRAL COMPRESSIONAL STABILITY. Ian Lowery, Edward Draper, Rodolfo Cappello. The Royal Veterinary College, University of London, UK.

Ventral slot surgery is a common method of cervical spinal decompression. Despite the plethora of published papers investigating distraction and stabilisation following ventral slot surgery, there is still no strong biomechanical evidence to establish whether or not such stabilisation is required. This study aims to test the hypothesis that: ventral slot surgery is associated with a change in functional spinal unit (FSU) stiffness, yield point load or energy to yield point. This was done by analysing the load-deformation curves obtained during axial compression of intact and ventrally slotted greyhound FSUs.

FSUs consisting of C3–C4 and C5-C6 were harvested from 17 ex-racing greyhounds. The spines were randomly assigned into two groups and dissected free of surrounding musculature. One group of FSUs remained intact as a control whilst ventral slots were performed on the other group of FSUs. The slot size measured 7 mm in width and 15 mm in length. The FSUs were tested to destruction in a materials tester with 1 mms^{-1} axial compression. Measurements of load and deformation were recorded at a frequency of 0.1kHz. A Load-deformation graph was plotted for each FSU and the linear portion gradient, yield point load and energy to yield point recorded.

Ventral slot surgery did not have any consistent effect on the average linear portion gradient, a measure of stiffness. The average yield point load were not significantly different between the control and ventral slot groups ($p=0.102$). The average energy to failure of the control group was 7329J (747.47N) compared to an average energy to failure of only 2685J (273.84N) in the ventral slot group. This difference was found to be significant using a two-way ANOVA ($p=0.006$)

Ventral slot surgery significantly decreases the energy required to reach the FSU's yield point. To date there are no studies on the physiological energy applied to the disc however, human studies have revealed that the mean compression forces on the C4-C5 motion segment ranged to 1164N. We conclude that the energy required to produce intervertebral disc failure following ventral slot could be less that the normal physiology energy applied to the disc.

This study suggests that ventral slot surgery reduces the energy required to collapse the intervertebral disc space in vitro, predisposing to foraminal compression and bulging of any residual annulus fibrosus and dorsal longitudinal ligament into the vertebral canal. Further studies on disc function and the effect of different surgical techniques are necessary.

ABSTRACT # 6

SAFE CORRIDORS FOR IMPLANT INSERTION IN CAUDAL CERVICAL SPONDYLOMYELOPATHY: A COMPUTED TOMOGRAPHY STUDY IN AFFECTED DOBERMANN PINSCHERS. Daniele Corlazzoli, Clinica veterinaria Roma Sud, Roma, ITALY.

The purpose of this study is the evaluation of the safety of the proposed angle of insertion for bicortical implants in Dobermann Pinchers affected by the dynamic form of Caudal cervical spondylomyelopathy (CCSM) trough CT images analysis.

Nine Doberman Pinschers patients with a myelographic diagnosis of traction responsive ventral compression of the C5-C6 or C6-C7 or both intervertebral spaces (IVS), underwent a Ct evaluation with a third generation Ct scan (GE Ct Max) or with a spiral CT SCAN (GE Hyspeed CTE). Digital images were elaborated with a commercial photographic software (Adobe Photoshop CS) in order to:

- evaluate the safety of implant insertion from the midline with 30°, 35° and 40° of inclination by drawing a line simulating the implant
- evaluate the correct positioning of the patients in surgery by superimposing a grid to the pictures. The degree of malpositioning was measured.

Insertion of the implant with 30° of inclination would have caused violation of the spinal canal in 15% of the areas considered. In 30% of the areas considered, insertion of the implant with 30° of inclination would have been safe but with a high risk of vertebral canal violation. In the remaining 55% of the areas insertion with 30° of inclination was considered safe.

Insertion of the implant with 35° of inclination would have caused violation of the spinal canal in 11% of the areas considered. In 10% of the areas considered insertion of the implant with 35° of inclination would have been safe but with an high risk of vertebral canal violation. In 79% of the areas considered insertion with 35° of inclination would have been safe.

Insertion of the implant with 40° of inclination would have caused violation of the spinal canal in none of the areas considered besides the caudal third of C7. Four patients out of nine were positioned with the spine perfectly parallel to the gantry. In five patients out of nine the spine was rotated from −9° to + 10° along the long axis.

Ct evaluation can aid in planning surgery in Dobermann Pinscher affected by CCSM.

The result of this study suggests that bicortical implants in the caudal cervical area in Dobermann Pinschers affected by CCSM carry a high risk of vertebral canal violation but clinical studies are needed to demonstrate this hypothesis in clinical settings.

ABSTRACT # 7

PROGNOSTIC VALUE OF MAGNETIC RESONANCE IMAGING IN DOGS WITH PRESUMPTIVE ISCHAEMIC MYELOPATHY. Davies ESS, Cherubini GB, Brodbelt DC, Volk HA, Lamb CR. Department of Veterinary Clinical Sciences, The Royal Veterinary College, University of London, UK

Ischaemic myelopathy as a result of fibrocartilagenous embolism is a common cause of acute spinal signs in the dog. Definitive diagnosis of ischaemic myelopathy requires histopathology. Presumptive ante mortem diagnosis may be based on results of magnetic resonance (MR) imaging. It is unclear if MR imaging can be used for prognosis.

The aims of this study were to assess if the lesion intensity or extent of spinal cord involvement in MR images is associated with outcome.

MR images from 25 dogs with presumptive ischaemic myelopathy were reviewed. Diagnosis was based on clinical signs (acute, non-painful, non-progressive myelopathy), lack of inflammatory cerebrospinal fluid, and hyperintense lesions on T2-weighted MR images with minimal gadolinium uptake on T1-weighted images. Axial T2-weighted images were ranked according to a blinded subjective assessment of lesion signal intensity and maximal proportion of the cross-sectional area of spinal cord affected. The association of lesion signal intensity and maximal cross sectional area with neurological grade at presentation, whether they recovered or not, time to recovery, and time to voluntary urination were assessed using Fisher's exact test and the logrank test as appropriate.

Dogs with less intense lesions tended to be more likely to recover than those with most intense lesions (Odds Ratio (OR) 9.4, 95% Confidence Interval (CI) 0.8-472.1, $p=0.073$). There was a trend suggesting that dogs with lesions occupying $<50\%$ cross-sectional area of the spinal cord were more likely to recover than those with lesions affecting the majority of the cord (OR 3.9, CI 0.5-49.4, $p=0.20$). There was a tendency for dogs with less intense lesions to recover walking faster than those with more intense lesions ($p=0.13$)

On the basis of these results, it appears that lesion signal intensity and maximal proportion of the cross-sectional area of spinal cord affected might aid prognosis in dogs with presumptive ischaemic myelopathy. A larger scale study is merited.

ABSTRACT # 8

CONTRAST ENHANCED FLUID ATTENUATED INVERSION RECOVERY Vs CONTRAST ENHANCED SPIN ECHO T1 WEIGHTED IMAGING AT LOW FIELD STRENGTH: EVALUATION OF 46 CASES WITH DIFFERENT BRAIN DISORDERS. Cristian Falzone[1], Massimo Baroni[1], Maurizio Calistri[1], Massimo Tranquillo[2]. [1]Valdinievole Veterinary Clinic, Monsummano Terme (PT), Italy; [2]Experimental Zooprofilattic Institute of Lombardia and Emilia Romagna, Brescia, ITALY.

In human medicine contrast enhanced fluid attenuated inversion recovery (CE-FLAIR) imaging plays an important role in detecting brain diseases. The aim of this study was to define the clinical utility of CE-FLAIR by comparing results to those with contrast enhanced Spin Echo T1 weighted imaging (CE-SET1WI) in dogs and cats with different brain disorders.

46 patients (41 dogs and 5 cats) with clinical suspicion of brain disease and 30 normal animals (25 dogs and 5 cats) were evaluated using an ESAOTE Vet-MR scanner (0.2T permanent magnet). Prior to contrast injection, SE T1WI, spin echo T2 weighted imaging (SE T2WI) and T2W-FLAIR sequences were performed on three planes. SE T1WI and T2W-FLAIR were repeated after gadolinium (Magnevist) injection. Sensibility in detecting (number), delineating lesions (location and margins) and enhancement pattern and rate were evaluated. Lesion location was considered as forebrain, brainstem and cerebellum. Lesion margins were classified as demarcated or not demarcated. The enhancement pattern (degree and distribution of enhancement) was visually graded from 1–4: 1-good and regular; 2-good and irregular; 3-low and regular; 4-low and irregular. We finally measured the enhancement rate defined as the lesion to background signal intensity ratio. Statistical analysis were performed by R software and $P<0.05$ was considered significant.

In the normal animals no lesions were seen. In the affected animals, CE-SET1WI detected 48 lesions in 34 patients whereas CE-FLAIR detected 81 lesions in 44 patients; in 2 cases both sequences failed to identify contrast enhancement. The difference between the two sequences in detecting lesions was highly significant ($P<0.01$) but there was no difference in the location or margins of the identified lesions ($P=0.50$). There was also no difference in the enhancement patterns ($P=0.11$), though there was a tendency for lesions to be classified as grade 1 or 3 using CE-FLAIR. The enhancement rate was significantly superior ($P<0.01$) on CE-FLAIR.

The results of this study suggest a superiority of CE-FLAIR on CE-SET1WI in detecting and enhancing brain lesions. We speculate that this is due to the ability of FLAIR to highlight T2-hyperintense lesions usually undetectable on routine SE T1WI and to the T1 and T2 effects summation in revealing lesions. Based on the data collected and the limitations of this study (heterogeneous diseases examined and probability of confusion in establishing lesion margins on CE-FLAIR) we conclude that CE-FLAIR could be a valuable adjunct to routine brain MRI protocols but further investigations are needed.

ABSTRACT # 9

CLINICAL AND MAGNETIC RESONANCE IMAGING FINDINGS IN FELINE VESTIBULAR DISEASES. A.Negrin[1], G.B.Cherubini[1], C. Lamb[1], L. Benigni[1], S.R. Platt[2]. The Royal Veterinary College, University of London, UK. [2]Animal Health Trust, Newmarket, UK.

The aims of this study were to investigate the clinical and MRI findings of cats affected by vestibular disorders and associate the results of MRI with clinical data. Medical records were searched for cats with vestibular signs that had MRI of the head. Clinical data, CSF results, follow up and, when available, histological results were recorded. MRI criteria including signal intensity, lesion site, margins, ventricular dilatation, oedema and mass effect were evaluated reviewing T1- and T2-weighted images, T1-weighted images after contrast administration and, where available, FLAIR images. A total of 103 cats with vestibular disorders were included in this study; 77 of them had a complete MRI study which was reviewed by two boarded radiologists. Clinical localisation was consistent with lesion site observed on MRI in 95% of cases. Forty cats (52%) were affected by central vestibular syndrome (CVS), which was part of a multifocal disease in 17 (42.5%). Only one cat presented with a paradoxical vestibular syndrome due to cerebellopontine-angle meningioma. Most frequent cause of CVS were inflammatory conditions (18 cats; 45%) with prevalence of infectious aetiology, including bacterial inflammation as intracranial extension of inner ear otitis (5 cats; 12.5%), feline infectious peritonitis (3 cats; 7.5%) and toxoplasmosis (2 cats; 5%). Neoplasia (11 cats; 27.5%) was the second most common cause of CVS{lymphoma, meningioma, glioma, schwannoma and squamous cell carcinoma}. Two cats (2.6%) were suspected of thiamine deficiency from MRI findings and improvement following vitamin B1 supplementation. In the 37 cats (48%) affected by peripheral vestibular syndrome (PVS), two main conditions have been recorded: idiopathic vestibular syndrome, suspected in 16 (43%), and otitis media or interna, observed in 16 (43%). The remaining 5 cats (17%) with PVS presented inflammatory polyp (1), meningitis (1), thiamine deficiency (1) and vascular event (2). Cats affected by idiopathic vestibular syndrome did not show any breed or age predisposition, with a mean age of 5y 2m (range 6m-13y). Onset was most frequently acute, although 4 cats presented with chronic clinical signs; 3 of these cats, however, improved dramatically without medication. Most of the cats (9 cases; 56%), in which idiopathic vestibular syndrome was suspected, showed rapid and complete recovery, while head tilt remained in 3 cats. No underlying systemic diseases were observed in these cases and recurrence was recorded in just one cat (6.3%). Five cats (13.5%) showed signs compatible with bilateral vestibular syndrome, in 4 due to bilateral otitis media/interna, and in 1 due to thiamine deficiency. This study demonstrates that clinical localisation of cats with VS is consistent with MRI findings in 95% of cases; idiopathic and infectious causes of PVS are equally common and inflammatory aetiology is frequent cause of CVS. Statistical evaluation of the clinical findings and patient outcome are underway.

ABSTRACT # 10

SUPERPARAMAGNETIC IRON OXIDES FOR TRACKING NEURONAL STEM CELLS BY A CLINICAL 1.5 TESLA MAGNETIC RESONANCE SCANNER. K. Matiasek[1,5], A. Beer[2], G. Piontek[3,5], J Schlegel[3,4,5]. [1]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, LMU Munich, Germany; [2]Department of Radiology, TU Munich, Germany; [3]Institute of Pathology, GSF Federal Research Centre of Environment & Health, Neuherberg, Germany; [4]Institute of Pathology, TU Munich, Germany; [5]*Task Force on Life Cell Imaging* of the Universities of Munich and the GSF Research Centre for Environment & Health, Neuherberg, Munich, GERMANY.

Understanding of stem cell biology in therapeutic trials severely hampers from lack of accessible imaging techniques suitable for non-invasive tracking of cell migration, trafficking, homing, differentiation, maturation and death. Thus, the present study was aimed to evaluate the suitability of commercially available and FDA-approved tracers for extra-corporal labelling of neuronal stem cells (NSC) and subsequent screening by a 1.5 T clinical scanner after stereotaxic application.

NSC were developed from a permanent murine TBV2 cell line by sorting upon nestin-expression and treatment with growth factors and neurotrophins. Prior to intracranial injection, the NSC were labelled by two supraparamagnetic iron oxides (SPIOs) – namely Resovist® and Endorem® - via fluid-phase endocytosis. Thereafter, extracellular particles of the supernatant were removed while positively labelled cells were sorted and divided into aliquots containing $5x10^4$ NCS each. Stereotaxic cell transfer was performed in accordance to HCNR guidelines. Per tracer, eight syngenic mice received injections into the cerebellum, neocortex, thalamus and basal ganglia. A same number of animals received unlabelled NSC and cell-free phosphate-buffered saline. Magnetic resonance (MR) images of the brain were obtained at post-operative days D0, D3, D7 and D14 under general anesthesia using a 1.5 T scanner and a birdcage coil. Applied sequences comprised 2D multislice T2-weighted spin echo-sequences and T2*-weighted gradient echos. Imaging parameters provided a matrix size of 2562 pixels, a spatial resolution of 200 µm, 500 µm slice thickness and a field of view of 100x100 mm. The animals were euthanized at D14 and their brains were processed for histopathological investigation.

Cellular uptake of both SPIOs was highly effective and did not compromise viability of the NSCs throughout the experiment. All animals survived until D14 without overt signs of neurological impairment. On MR images, both SPIOs presented with strong signal extinction in T2*-gradient echos, superior to that of intraoperative hemorrhage, and it did not attenuate until D14. Inspite of only mesoscopical resolution, the sites of cell injection could be readily identified in all mouse brains. Given the higher number of NSC used in therapeutic trials and a signal amplification factor close to that of paramagnetic microspheres, the detection threshold of these commercial SPIOs is likely to allow tracking of NSC by routine MR scanners in the CNS of larger animals.

ABSTRACT # 11

ASSOCIATION OF PERITUMOURAL OEDEMA WITH VASCULAR ENDOTHELIAL GROWTH FACTOR, KI-67 EXPRESSION AND SURVIVAL IN CANINE INTRACRANIAL MENINGIOMAS. L A Matiasek[1], S R Platt[1], T Scase[1], J Miller[1], K Matiasek[2], V Adams[1]. Animal Health Trust, Newmarket, Suffolk, UK, [2]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, Ludwig Maximilians University, Munich, GERMANY.

Peritumoural oedema (PTO) occurs variably with meningiomas and can adversely affect the clinical course. A positive correlation of vascular endothelial growth factor (VEGF) and Ki-67 expression with the amount of PTO has been described in humans. The aim of this study was to investigate whether the amount of PTO with canine intracranial meningioma can predict survival, and to assess the role of VEGF and Ki-67 expression in oedema formation.

Magnetic resonance imaging (MRI) of brain and tissue samples of histologically confirmed WHO grade I meningiomas from 26 dogs were included. The volume of PTO was defined by using a commercial software program, which allowed the estimation of three-dimensional volume on the basis of MR images. The volume of oedema (Vo) was measured according to the high signal intensity area around the tumour in T2-weighted images. The volume of the tumour (Vt) was measured in gadolinium enhanced T1-weighted images. The oedema index (OI) was calculated using the ratio of Vo and Vt. Immunohistochemical staining for VEGF and Ki-67 was performed. To quantify Ki-67 expression, MIB-1 Labelling Index (LI) was calculated by determining the percentage of positive nuclei using automated counting via Image J NIH-software. Extent, intensity and distribution of VEGF-expression were assessed by light microscopy using a semi-quantitative scale. Spearman's rank correlation coefficient and Goodman and Kruskal's gamma coefficient were used to identify associations between OI and VEGF and also MIB-1LI. All dogs underwent post-surgical radiotherapy and were followed until the time of death. Overall survival was compared to OI. Survival analysis was carried out using a Kaplan-Meier procedure with log-rank tests.

VEGF expression was detectable in 23 of 26 dogs . MIB-1 LI was measured in 19 dogs, and in 18 of them MIB-1 LI >0.00. OI was >0.0 in 24 of 26 cases. There was no association between OI and VEGF staining parameters or MIB-1LI. Median survival time was 425 days (95% CI: 225-625). OI was not associated with survival.

This study suggests that the amount of peritumoural oedema in canine intracranial meningioma is not predictive for malignancy as determined by Ki67 index. Other factors than VEGF must play an important role in enhancement of vascular permeability and formation of vasogenic oedema. The amount of peritumoural oedema does not seem to be of predictive value for survival of dogs with intracranial meningiomas treated with surgery and radiation.

ABSTRACT # 12

L-2-HYDROXYGLUTARIC ACIDURIA: CHARACTERISATION OF THE MOLECULAR DEFECT IN A SPONTANEOUS CANINE MODEL. Jacques Penderis[1,2]; Jacqui Calvin[3]; Carley Abramson[2]; Cornelis Jakobs[4]; Louise Pettitt[2]; Matthew Binns[2]; Nanda Verhoeven[4]; Simon Platt[2]; Eamonn O'Driscoll[3]; Cathryn Mellersh[2]. [1]Institute of Comparative Medicine, Faculty of Veterinary Medicine, University of Glasgow; [2]Animal Health Trust, Newmarket; [3]Biochemical Genetics Unit, Department of Clinical Biochemistry, Addenbrooke's NHS Trust, Cambridge; [4]Metabolic Unit, Department of Clinical Chemistry, VU University Medical Centre, Amsterdam

L-2-hydroxyglutaric aciduria (L-2-HGA) is a neurometabolic disorder producing neurological deficits in human patients which include psychomotor retardation, progressive cerebellar dysfunction, learning disability and seizures. L-2-HGA is characterised by elevated levels of L-2-hydroxyglutaric acid (L-2-HG) in a variety of tissues and fluids, including CSF, plasma and urine. Mutations within the gene *L2HGDH* (Entrez Gene ID 79944) encoding L-2-hydroxyglutaric dehydrogenase have recently been shown to cause L-2-HGA in human patients. L-2-HGA has been identified within the Staffordshire bull terrier (SBT) and West Highland white terrier (WHWT), with affected dogs presenting between 6-months and 1-year of age (but up to 7-years) with ataxia, muscular stiffness at exercise or excitement, altered behaviour or epileptic seizures.

21 SBTs and 2 WHWTs with L-2-HGA and 127 closely related normal SBTs were enlisted from an outbred population of pet dogs. 2-HG accumulation was confirmed by urine gas chromatography mass spectrometric (GCMS) analysis. Differentiation between L-2-HG and D-2-HG was performed by stable-isotope-dilution GCMS. MR imaging studies of the brain were performed in 14 affected dogs. In order to determine whether the gene *L2HGDH* was responsible for canine L-2-HGA we identified, amplified and fractionated four microsatellite markers adjacent to the canine homologue of *L2HGDH* (*Canis familiaris* Ensembl Gene ID: ENSCAFG00000014237). The ten exons and 120 base pairs of the flanking 5′- and 3′- intron region of canine *L2HGDH* were sequenced in their entirety. Pedigree analysis was performed in all dogs with available pedigree information.

The study indicates: 1) Affected dogs have dramatic L-2-HG elevation in urine and characteristic brain MRI changes. 2) Microsatellite homozygosity mapping indicates a mutation within *L2HGDH* is responsible for canine L-2-HGA. 3) Sequencing of canine *L2HGDH* demonstrates a two base pair substitution in exon 10 of affected SBTs and the introduction of a premature stop codon in exon 8 of affected WHWTs, significantly altering the encoded amino acid sequence. 4) The 14 affected and 34 carrier SBTs with pedigree information all trace back to a common ancestor, supportive of a common founder effect.

Canine L-2-HGA is caused by a mutation in canine *L2HGDH* and is a true homologue of human L-2-HGA. We have developed a genetic screening test, allowing eradication of canine L-2-HGA.

ABSTRACT # 13

ASSOCIATION BETWEEN SPINAL CORD DORSAL INVOLVEMENT AND PAIN IN SYRINGOMYELIA SECONDARY TO CANINE CHIARI MALFORMATION. C Rusbridge[1]. H. Caruthers[1], Marie-Pierre Dubé[2], M Holmes[3], N.D. Jeffery[3]. [1]Stone Lion Veterinary Centre, Wimbledon, London, UK [2]Department of Medicine, Université de Montreal, and The Montreal Heart Institute, Canada [3]Department of Veterinary Medicine, University of Cambridge, Madingley Road, Cambridge, CB3 OES

This study was designed to test the hypothesis that pain associated with syringomyelia in dogs is related to spinal cord dorsal horn damage.

Syrinx dimensions and precise location within the spinal cord were determined by masked observers from magnetic resonance images of 55 cavalier King Charles spaniels (CKCS) with syringomyelia. After removal of masking, syrinx parameters were compared between the cohort of dogs that exhibited pain with those that did not.

Maximum syrinx width was the strongest predictor of pain in dogs with syringomyelia. Syrinx width was also correlated with scratching behaviour and scoliosis. Syrinx width was strongly correlated with dorsal horn

involvement. Dogs with pain were also more likely to have extensive dorsal grey column damage.

Large syrinxes with dorsal horn damage are associated with persistent pain which may have implications for the success of surgical and medical management. Our results suggest that the pain behaviour expressed by this group of patients is likely to be 'neuropathic pain', resulting from disordered neural processing in the damaged dorsal horn. As such it is likely that conventional analgesic medication may be ineffective.

ABSTRACT # 14

MAGNETIC RESONANCE IMAGING EVALUATION OF HEAD TRAUMA IN 32 DOGS; ASSOCIATIONS WITH MODIFIED GLASGOW COMA SCORE AND PATIENT OUTCOME. S.R.Platt, V. Adams, F. McConnell, L. Matiasek, A. de Stefani, A. Lujan Feliu-Pascual, L. De Risio. The Animal Health Trust, Newmarket, UK.

The role of magnetic resonance imaging (MRI) in human head trauma offers distinct advantages over computed tomography in the recognition of parenchymal lesions, but its use has not been evaluated in veterinary medicine.The aims of this study were to investigate whether MRI assessment of head trauma is associated with severity of neurolgical dysfunction and could be predictive of patient survival. Dogs presenting with evidence of head trauma and which were imaged with a 1.5T MRI were retrospectively evaluated. Criteria necessary for inclusion in the study were (i) imaging performed within 7 days of the trauma (ii) neurological examination at time of MRI enabling a modified Glasgow coma score (MGCS) to be estimated (iii) survival at 1 and 6 months after MRI (iv) T1, T2, T2* gradient echo and FLAIR weighted images. All images were blindly evaluated for (i) extra-axial haemorrhage (ii) intra-axial haemorrhage (iii) fractures (linear, comminuted, compound and/or depressed) (iv) degree of parenchymal shift (mm) (v) single or multiple parenchymal lesions and (vi) MRI grade of severity (I-IV) modified from establised criteria in human head trauma imaging.

The MRI parameters were individually evaluated with the estimated MGCS and survival at 1 and 6 months. Cross-tabulations and Fishers exact tests or Goodman and Kruskal's gamma were performed to identify associations between MRI characteristics and outcome and MGCS. The effect of the MGCS on outcome was examined using Wilcoxon rank sum tests.

Thirty-two dogs fulfilled the criteria. The median MGCS was 15 (range 7–18). A linear trend was demonstrated between survival at 1 and 6 months and the MGCS ($P=0.01$ and 0.0002). Nineteen of 32 dogs (59%) had an abnormal MRI which was not associated with outcome at 1 or 6 months. MRI grade was significantly associated with outcome at 1 and 6 months ($P=0.04$); a higher class was associated with a reduced probability of survival. The presence of intra- or extra-axial haemorrhage was not associated with outcome at 1 or 6 months. Dogs with no midline shift were more likely to survive to 1 month than dogs with any evidence of midline shift ($P=0.02$). Whether a dog had a skull fracture or not and type of fracture were not associated with survival.

The MGCS was not associated with the presence of extra-axial haemorrhage, skull fractures or fracture types. There were significant associations between the MGCS and abnormal MRI, MRI grade, presence of intra-axial haemorrhage and the degree of midline shift ($P<0.0001$ in each case).

The use of the MGCS in conjunction with MRI can help determine prognosis in dogs with head trauma. This study may provide MR indicators for surgical therapy.

ABSTRACT # 15

EEG MONITORING FOR THERAPY OF STATUS EPILEPTICUS. Raith K*, Steinberg T*, Matiasek K**, Fischer A *Section of Neurology, Department of Small Animal Medicine, Ludwig-Maximilians-University, Munich, **Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, Ludwig-Maximilians-University, Munich, Germany

In human medicine it has been suggested that continuous electroencephalographic (EEG) monitoring should be part of the management of status epilepticus (Anaesthesia 2004; Oct;59(10):1033-4). The EEG is considered a diagnostic tool to evaluate efficacy of pharmacological treatment of seizures, depth of anaesthesia and the point of tapering the medicaments. There is still disagreement whether clinical or electrophysiological seizure termination or a burst suppression pattern should be intended. Also, for the time of tapering different recommendations exist in humans. Depending on the author general anaesthesia should be obtained for at least twelve to 48 hours.

Ten patients (7 dogs, 3 cats) presented in status epilepticus (2004–2005) from various reasons (3 probable idiopathic epilepsy, 3 intoxications, 4 unknown etiology) and were monitored continuously with the NicoletOne Modular Neurodiagnostic System (Viasys Healthcare). EEG was recorded from five stainless steel needle-electrodes (LO, RO, LF, RF, V; eight channels, bipolar derivations) inserted subcutaneously with the patients under general anesthesia. Patients were initially treated with diazepam followed by either propofol (n=3) or pentobarbital (n=7) continuous infusion.

In all patients clinical seizures stopped after induction of general anesthesia. However, the EEG still showed distinct epileptiform patterns (spikes, polyspikes) in all patients indicating insufficient depths of anesthesia. Paroxysms disappeared with an increased infusion rate of either pentobarbital or propofol. In some patients (n=4) epileptiform activity reappeared when trying to taper the dose after a minimum of six hours thus indicating an insufficient duration of general anesthesia. A burst-suppression pattern was only achieved in five patients. The outcome of the patients (6 recovered, 3 were euthanized, 1 died) did not depend on depth of anaesthesia but on the underlying cause for the status epilepticus.

It was concluded that EEG is an essential tool for therapeutic monitoring of status epilepticus in dogs and cats. It can guide the anesthetic depth and duration of anesthesia necessary to manage status epilepticus successfully. Further investigation to define therapeutic endpoints is warranted.

ABSTRACT # 16

THE CANINE "QUADRIGEMINAL CYST"- AN INCIDENTAL FINDING? CLINICAL AND DIAGNOSTIC IMAGING CHARACTERISTICS IN 28 CASE. L A Matiasek, SR Platt, R Dennis, S Shaw, Animal Health Trust, Newmarket, Suffolk, UK

Clinical significance of cysts of the quadrigeminal cisterna (QC) is controversial. Seizures and ataxia are documented in dogs, and incidental QC are described. The aims of this study were to evaluate the prevalence of QC in dogs, assess breed and gender predisposition, age at the time of diagnosis, document magnetic resonance imaging (MRI) abnormalities and to investigate association between MRI findings and clinical signs.

Diagnosis of a QC was based on 1) supracollicular localisation, 2) same signal intensity as cerebrospinal fluid (CSF) and 3) no contrast enhancement. CSF results were included where obtained. Signalment, clinical signs and MRI abnormalities were recorded. QC associated mass effect was calculated. The percentage displacement of brain tissue was compared to the appearance of relevant clinical signs using un-paired t-tests.

In 4100 canine brain scans, 28 QC were diagnosed in 18 toy, 7 small breed and 3 other dogs. Fifteen dogs were brachycephalic. There were 18 males and 10 females. Median age was 4 years. Routine CSF examination was normal in 15 of 20 dogs. In 4 cases, changes were linked to other CNS disease. In 9 cases, cerebellum and occipital lobes were displaced. The cerebellum or the occipital lobes were displaced in 11 and 4 cases, respectively. In 4 cases there was no mass effect. Clinical signs assumed to be associated with parenchymal displacement were focal and generalised seizures in 5 cases and cerebellar signs in 6 cases. In one dog the QC caused obstructive hydrocephalus and seizures. Mean occipital lobe displacement was 5.8% (SD=7.2), and there was a statistically significant difference for the percentage displacement between clinically affected and normal dogs ($p=0.04$). Compression greater that 14% was always associated with clinical signs. Mean cerebellar displacement was 13% (SD=12.2); there was no association of compression and clinical signs ($p=0.42$). Three different phenotypes of QC could be characterized: Fourteen appeared to have an anatomical relationship to the 3rd ventricle, 4 to the 4th ventricle, and 10 seemed bilocular.

The prevalence of QC in dogs is low. Small, brachycephalic, male dogs are over represented. QC do not seem to alter CSF. Occipital lobe displacement is more likely to cause clinical signs than cerebellar compression. However, 57% of our patients had an incidental QC despite parenchymal displacement. Therefore, clinical signs, metabolic status and CSF analysis must be considered when assessing the clinical significance of this neuroimaging finding. Until the heterogeneous MRI appearance of QC is further characterised by MR flow studies, it is the author's opinion that the term ''supracollicular fluid accumulation'' is more appropriate.

ABSTRACT # 17

CHRONIC CANINE HYPERTROPHIC NEURITIS MIMICKING NERVE SHEATH TUMOURS ON ADVANCED IMAGING; 3 CASES.

S.R.Platt[1], F. McConnell[1], R. Dennis[1], A. DeLahunta[2] [1]The Animal Health Trust, Newmarket, UK. [2]College of Veterinary Medicine, Cornell University, Ithaca , New York USA

Localised or diffuse enlargment of peripheral nerves may be produced by a variety of inflammatory, inherited or reactive processes as well as neoplastic diseases. The present study aims to describe the clinical, imaging and histopathological findings in three dogs with hypertrophic neuritis. Case 1- A 12-year-old neutered male cross breed presented with chronic, progressive, pelvic limb weakness and ataxia, lateralised to the right. Neurological examination localised a painful T3-L3 lesion. Thoracolumbar spinal magnetic resonance imaging (MRI) was performed using a 1.5T unit. A 2.5x0.5x0.5cm intradural mass was identified at the level of T5 which exited the enlarged associated right-sided foramen and compressed the spinal cord by over 75%. The mass was isointense to the cord on T1-W images, hyperintense on T2-W images and uniformly enhanced following gadolinium administration. A dorsal laminectomy enabled resection of an intradural-extramedullary nerve root mass via a durotomy and rhizotomy. Histopathology confirmed extensive destruction of the nerve fascicles with abundant deposition of myxoid stroma, collagen, Bungner's bands and vascular sprouting, consistent with a focal, chronic hypertrophic neuritis. The dog recovered well from surgery with no recurrence of signs in the following 3 years. Case 2 - A 5-year-old female Labrador retriever presented with a chronic, progressive, left thoracic limb paresis and Horner's. Neurological examination localised a painful C6-T2 extramedullary lesion. Cervicothoracic spinal MRI was performed using a 1.5T unit. Marked and extensive thickening of the left C8 spinal nerve exiting the foramen without spinal cord involvement was identified. The lesion extended ventrally to the axilla with the same signal characteristics as for case 1. A dorsal laminectomy enabled durotomy and rhizotomy achieving mass removal. Histopathology confirmed significant loss of myelinated fibres, replacement with columns of Bunger's bands, and lymphocyte and neutrophil accumulation, consistent again with a focal chronic neuritis. The dog was successfully managed on prednisone for a further 21 months. Case 3 – An 8-year-old neutered male Golden retriever presented with chronic, progressive, left thoracic limb paresis. Neurological examination localised a painful C6-T2 extramedullary lesion. A contrast-enhanced computed tomographic (CT) examination revealed an intradural-extramedullary mass over the body of C6 which was resected via a dorsal laminectomy.

Histopathologic examination was similar to the above cases and consistent with chronic hypertrophic neuritis. The dog did not progress for a year before it was lost to follow up.

Focal lesions affecting the spinal nerves identified with MRI or CT may be inflammatory rather than neoplastic in aetiology.

ABSTRACT # 18

SUSPECTED CHANNELOPATHY OF THE SKELETAL MUSCLES IN A FAMILY OF CATS.

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Channelopathies are hereditary disorders of the resting membrane potential of muscle cells or cells in the central nervous tissue. These functional disorders are recognized in humans as well as in animals. In this case report a suspected potassium aggravated myotonia in a family (n = 12) of cats is described. Potassium aggravated myotonia is a well known syndrome in humans but has never been described in cats.

Puppies of one cat family showed exercise and stress induced episodes of muscle spasticity beginning with an age of 2 months. Initially these episodes were characterized by a hypermetric and ataxic gait after extensive movement while playing. The muscle tonus increased resulting in a status of spastic recumbency, when the physical effort was not discontinued. The animals did not lose consciousness during these episodes and recovered spontaneously after several minutes, when they were brought to a quiet and familiar place. The threshold for the cats to reach the status of total spasticity was lowered by infections and changes in the environment, such as noise or irregular feeding. The diagnostic work up included a clinical-neurological examination, a complete blood cell count, a pre- and post exercise serum analysis of electrolytes, glucose, lactate, creatinkinase and blood gas parameters. All parameters were within the reference ranges and showed no pathological shift from pre to post exercise. Further diagnostics such as magnetic resonance imaging, electromyography, motor nerve conduction velocity, repetitive nerve stimulation and a metabolic screening for storage diseases showed no pathological changes in these cats. Moreover, the histopathological examination (semi-thin slices and special staining techniques) of 5 sacrificed cats revealed no relevant findings to explain the clinical signs.

Two further clinical tests were performed: A cold environment (+ 4°C) had no influence on the episodes of the cats, whereas a potassium-rich diet caused an aggravation of the clinical signs. These findings suggested a sodium-channelopathy, which is described in humans as potassium aggravated myotonia. Clinical and laboratory findings only lead to a suggestion of such a disease, since efficient methods to diagnose this kind of functional disorders are missing in the veterinary medicine. In further studies we attempt to establish a method to measure the functionality of ion-channels on the surface of muscle cells with help of the patch-clamp technique.

ABSTRACT # 19

IMAGING FINDINGS AND SURGICAL TREATMENT OF HEMIVERTEBRAE IN THREE DOGS.

Catherine Talbot, Peter Smith, Nick Jeffery (Department of Veterinary Medicine, Cambridge University, Cambridge, UK)

The use of magnetic resonance (MR) images to devise an individually tailored approach to spinal cord decompression and vertebral stabilisation in animals affected by hemivertebrae.

Three dogs affected by hemivertebrae were presented to Cambridge University Veterinary School Hospital for treatment of pelvic limb paresis. Radiographs and MR imaging located the veretebral canal stenosis associated with hemivertebrae. Each case underwent decompressive surgery plus vertebral stabilisation.

In all cases, radiographs showed marked kyphosis of the spine in the mid thoracic region. In 2 dogs MR images showed ventrodorsal spinal cord compression caused by gross shortening of the pedicles and in 1 dog ventrolateral compression from a hyperintense mass on the right side. The surgical approach was determined by the origin of the compressive lesion; 2 dogs had a dorsal laminectomy and 1 dog had a hemilaminectomy. Spinal fixation was chosen according to the stabilisation required after the different decompressive techniques. All the dogs have had a satisfactory outcome following surgery.

In conclusion, MR images provide cross sectional information on the type and location of spinal cord compression associated with hemivertebrae of sufficient superiority to plain and contrast radiography to permit reliable outcomes following decompressive surgery.

ABSTRACT # 20

ASSOCIATION OF CLINICAL, MAGNETIC RESONANCE IMAGING FINDINGS AND OUTCOME IN 52 DOGS WITH SUSPECTED ISCHAEMIC MYELOPATHY.

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The magnetic resonance imaging (mri) features of ischaemic myelopathy (im) have been described in the human literature and in a small number of cases in the veterinary literature.

the purposes of this study were: 1) to assess the association of mri findings (site, lateralization and extent of the lesion) with the timing of imaging and with presenting neurological deficits (localization, lateralization and severity of signs) in dogs with a presumptive diagnosis of im, and 2) to associate the outcome (successful/ unsuccessful) with clinical and mri findings (site, lateralization and severity/extent of the lesion) at presentation.

the medical records and mr images of 78 dogs with a presumptive diagnosis of im (2000–2006) were reviewed. Inclusion criteria were: acute (< 24 hours) onset of non-progressive and non-painful myelopathy, 1.5t mri of the spine performed within 7 days of onset, clinical and mri findings consistent with non-traumatic im, complete medical records and follow-up. All mr images were reviewed to a consensus by two boarded radiologists blinded to the clinical findings. Fifty-two dogs met the inclusion criteria. There was no breed predisposition. Median age was 6 years. Fifteen dogs were female and 37 were male. Neurolocalization was c6-t2 in 15 dogs, t3-l3 in 10 dogs, t3-l3 with a decreased withdrawal reflex in one or both hind limbs in 4 dogs, l4-s3 in 23 dogs. Forty-five dogs had asymmetric neurological signs. Csf analysis was done in 32 cases. Time interval between onset of signs and mri was less than 24 hours in 24 cases, 25 to 48 hours in 15 cases and 49 to 168 hours in 13 cases. Mri was normal in 11 cases. Lack of mri changes has been reported in humans in the acute stage of im. Mri was abnormal (focal intramedullary hyperintensity in t2w and hypo-isointensity in t1w images) in 41 cases. The presence of mri abnormalities was not associated with timing of imaging ($p=0.3$). The presence of mri abnormalities was associated with ambulatory status on presentation ($p=0.04$). In the 41 cases with abnormal mri the site of the lesion identified by mri agreed with clinical neurolocalization in 32 cases. Of the remaining 9 cases mri identified the lesion in either the c1-c5 (2 dogs) or the t3-l3 (7 dogs) spinal cord segments while clinical examination revealed decreased withdrawal in the front limbs (2 dogs) or in the hind limbs (7 dogs), respectively. Mri findings regarding

lateralization of the lesion agreed with clinical findings in all but 3 cases whose clinical lateralization was very subtle. Severity of signs on presentation was associated with extent of the lesion on mri ($p=0.02$). Median follow-up was 583 days. Outcome was successful in 42 dogs and unsuccessful in 8. The severity of clinical signs on presentation was associated with outcome ($p=0.02$). No association was found between the site of the lesion and outcome ($p=0.1$). In addition symmetry/asymmetry of the lesion was not associated with outcome ($p=0.5$). The extent of the lesion on mri associated with outcome ($p<0.01$).

ABSTRACT # 21

FDG-PET IN NORMAL AND EPILEPTIC FINNISH SPITZ DOGS. Viitmaa R[1] , Haaparanta-Solin M[2], Snellman M[1], Cizinauskas S[3], Kuusela E[1], Jokinen TS[1,3] , Jeserevics J[3], Bergamasco L-A[4], Tukia E[1], Metsahonkala L[5], [1]Department of Clinical Veterinary Sciences, University of Helsinki, Finland [2] PET-Centre, University of Turku, Finland [3]The Referral Animal Neurology Hospital ''Aisti'', Vantaa, Finland [4]Department of Veterinary Morphophysiology, University of Turin, Italy [5]Epilepsy Unit, Hospital for Children and Adolescents, Helsinki Univ Centr Hospital, Finland

Magnetic resonance (MR) is usually not able to detect brain changes in dogs with idiopathic epilepsy, and knowledge about electroencephalographic (EEG) changes in dogs is not solid. Therefore other functional methods can be needed to better understand pathomechanisms of this disease. The aim of this study was to correlate changes in cerebral glucose metabolism utilizing 2-[^{18}F]fluoro-2-deoxy-D-glucose (FDG) uptake in brain as measured with positron emission tomography (PET)-scans of normal and epileptic dogs with comparison of EEG changes.

Eleven epileptic and 8 healthy Finnish Spitz (FS) dogs underwent PET-examinations with high resolution research tomography. Epileptic dogs had focal seizures and epileptogenic activity on EEG examination with no changes in MRI of brain. EEG was not performed to one epileptic FS dog. MRI was performed to 7/8 and EEG to 1/8 control dogs.

A bolus of FDG was administrated iv 15 min after medetomidine, butorphanol and midazolame sedation. Scanning (47 minutes) started 55 minutes after the tracer injection. PET images with co-registration of MRI were visually analyzed independently by 3 examiners, blinded for dogs' healthiness status.

Glucose uptake abnormalities/asymmetries appeared in 9 epileptic dogs (80%), in the occipital (5 dogs), lateral temporal (2 dogs) and frontal cortical areas (1 dog), in the caudal colliculum (1 dog) and in the cingulate gyrus (1 dog) or in the whole hemisphere (1 dog). In healthy dogs, asymmetries appeared in the parietal (3 dogs), lateral or medial temporal (3 dogs) cortices and caudal colliculum (2 dogs). Even though it is not possible to evaluate the absolute hypometabolism/hypermetabolism based on the visual analysis, the abnormalities were most often considered to be as hypometabolic. 4/8 healthy and 2/11 epileptic dogs did not have any findings. EEG detected focal paroxysmal discharges in 7/10 and generalized in 3/10 of epileptic dogs.

In comparison of FDG-PET and EEG findings 3/10 epileptic dogs had a perfect agreement of these two methods, in 6/10 dogs findings were not in clear disagreement. In 1/10 dog FDG-PET and EEG findings disagreed. Several FS-dogs with focal idiopathic epilepsy had a hypometabolic cortical lesion in a similar manner as known from previous human studies. Most of the hypometabolic lesions were temporo-occipital. FDG-PET had similar sensitivity in detection of possible epileptogenic cortical areas compared with EEG.

ABSTRACT # 22

FDG-PET IN EPILEPTIC LAGOTTO ROMAGNOLO DOGS. Jokinen TS[1,2], Viitmaa R[1], Haaparanta-Solin M[3], Grönroos T[3], Cizinauskas S[2], Jeserevics J[2], Snellman M[1], Metsä-Honkala L[4]. [1]Department of Clinical Veterinary Sciences, University of Helsinki, Finland [2]The Referral Animal Neurology Hospital ''Aisti'', Vantaa, Finland [3]PET-Centre, University of Turku, Finland [4]Epilepsy Unit, Helsinki University Central Hospital, Finland

For example, regional cerebral metabolism and blood flow can be measured nonivasively with positron emission tomography (PET). Interictal PET imaging has been useful in identifying the epileptic focus as an area of hypometabolism in cerebral grey matter in humans.

The aim of this study was to identify changes in cerebral glucose metabolism in epileptic Lagotto Romagnolo (LR) dogs compared to healthy LR dogs and to evaluate changes in glucose metabolism with age.

PET examination was performed in 10 LR dogs of different ages; 5 epileptic and 5 healthy controls. PET-scanning was performed with a high resolution research tomography (HRRT) under medetomidine, butorphanol and midazolame sedation. ^{18}Fluoro-2-deoxyglucose (FDG) was injected intravenously 55 minutes before PET-scanning (emission scan 40 minutes and transmission scan 7 minutes).

Zones of hypometabolism were identified by visual analysis of the FDG tomographic images. Focal abnormality was defined as an area of decreased accumulation of FDG if it could be recognized on three or more adjacent slices. Three investigators (blinded to the clinical history of the dogs) analysed the images independently. In addition to visual analysis, multi-angular regions of interest (ROI) were manually drawn to different areas of the brain with the help of a commercial software (Imadeus Academic Programm) and regional absolute accumulation values as well as relative uptake values (SUV) for FDG were calculated.

The cortical areas, caudate nuclei, caudal colliculi and in adult dogs also the cerebellar vermis appeared to have the highest glucose-uptake. Visual analysis revealed areas of hypometabolism in three epileptic LR dogs. Four healthy control dogs had normal PET scanning but one showed hypometabolic area. The relative glucose uptake values did not differ between the epileptic LR dogs and healthy controls. A change in relative cerebral glucose uptake with age comparable to humans was noticed so that the glucose uptake decreased after puppyhood.

FDG-PET scanning seems to be an informative tool in revealing areas of hypometabolism in epileptic patients. Also, in dogs a decrease in cerebral glucose metabolism with age can be noticed.

ABSTRACT # 23

WHY IS MELAS-MUTATION CAUSING MITOCHONDRIAL ENCEPHALOPATHIES IN HUMANS BUT NOT IN DOGS ? K. Baiker[1,3], S. Hofmann[2], M.F. Bauer[2,3], A. Fischer[4], W. Schmahl[1], K. Matiasek[1]. [1]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, LMU Munich; [2]Institutes of Diabetes Research and [3]Clinical Chemistry, Molecular Diagnostics & Mitochondrial Genetics, Academic Hospital Munich-Schwabing, Munich; [3]Section of Neurology, Department of Small Animal Medicine, LMU Munich, Germany

In humans, MELAS mutation refers to an A-G nucleotide transition in the mitochondrial tRNA gene for leucine$^{(UUR)}$. This specific pathogenic mutation evokes several neurological phenotypes like the MELAS syndrome (Mitochondrial Encephalopathy, Lactic Acidosis with Stroke-like episodes), MERRF (Myoclonus Epilepsy with Ragged Red Fibers), CPEO (Chronic Progressive External Ophthalmoplegia) and Leigh syndrome.

In a previous molecular genetic study, we identified 4 dogs (3 Yorkshire terriers, 1 Alaskan husky) out of 11, affected by canine Leigh syndrome known as subacute necrotizing encephalopathy (SNE), but none of 11 control dogs to be carriers of an A > G transition at nucleotide position 2691 within the canine tRNA-leu$^{(UUR)}$ gene. This mutation corresponds exactly to the A3243G mutation in the humans. We, therefore, erroneously concluded the A2691G mutation to be causative for SNE in dogs.

Extension of our control group (n = 42), however, revealed an A2691G transition in three healthy dogs pointing towards a non-pathogenic polymorphism which was in accordance to recent updates in the gene data bank of Shahid et al., 2005, who found the A2691G mutation to be a neutral polymorphism. Nonetheless, the mutation is remarkably less prevalent in healthy dogs than in our SNE affected group (36.3% (4/11) vs. 7.1% (3/42)).

The following haplotype lineage analysis, tackling the mtDNA D-loop of all affected and non-affected dogs, sorted all G-carriers into one specific subgroup indicating that A2691G mutation is a phylogenetically old neutral polymorphism.

It remains unclear whether the higher prevalence of A2691G within the SNE group versus our control group is due to an epigenetic susceptibility factor. Moreover, the question why the same evolutionally high conserved mutation, that leads to severe neurological disorders in humans, does not fulfil a pathogenic role in dogs has to be addressed. Comparison of secondary and tertiary structure of the human and canine tRNA-leu$^{(UUR)}$ shows a clearly more unstable human tRNA, with only two Watson-Crick pairs in the D-stem, which facilitates a dimerization of two mutated tRNAs and therefore a loss of function. In contrast, the canine tRNA D-stem is stabilized by two additional Watson-Crick pairs and probably retains its normal tertiary structure, even in presence of A2691G, because of the strengthened D-stem. These structural characteristics may provide an explanation for the different impact of the tRNA-leu$^{(UUR)}$ point-mutation in humans and dogs.

ABSTRACT # 24

TRANSIENT TETRAPARESIS IN A YORKSHIRE TERRIER WITH CYTOCHROME C OXIDASE DEFICIENCY. K.Baiker[1,4], T. Flegel[2],

S.Hofmann[3], M.F.Bauer[3,4], W. Schmahl[1], K. Matiasek[1]. [1]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, LMU Munich; [2]Small Animal Clinic, Veterinary Faculty, University Leipzig; Institutes of [3]Diabetes Research and [4]Clinical Chemistry, Molecular Diagnostics & Mitochondrial Genetics, Academic Hospital Munich-Schwabing, Munich, GERMANY

Cytochrome c oxidase (COX) or complex IV catalyzes the terminal step in the electron transport chain, which results in the reduction of molecular oxygen to water. COX deficiency leads to primary respiratory chain dysfunction with ATP depletion and represents a heterogeneous group of disorders that predominantly affect tissues with high-energy demand such as brain, skeletal muscle and heart. This case report features a transient neuromuscular syndrome in an adult Yorkshire terrier with significantly reduced COX activity. A 10 year old male Yorkshire terrier was presented with subacute onset of weakness and exercise intolerance that started in the hind limbs and progressed to severe tetraparesis within two weeks. At time of presentation the animal showed non-ambulatory tetraparesis, delayed postural reactions and reduced segmental reflexes at all limbs. EMG revealed mild denervation pattern while NCS exhibited a slightly reduced nerve conduction velocity and an M-wave dispersion. Based on these findings, a generalized lower motor neuron disorder was diagnosed. Subsequently, nerve and muscle biopsies were harvested. Examination of the left common peroneal nerve exhibited a moderate axonal neuropathy, predominantly affecting large myelinated Aα-fibers. Apart from mild neurogenic atrophy with fiber grouping, the tibialis cranialis muscle showed increased sarcoplasmatic lipid droplets and ragged red fibers suggestive of a mitochondrial myopathy. Therefore, additional muscle samples were processed for ultrastructural analysis and biochemical testing. Electron microscopy revealed subsarcolemmal accumulation of dysmorphic mitochondria with abundant paracrystalline inclusions. Biochemical screening for respiratory chain enzymes revealed a marked decrease in COX activity (0.7 U/UCS; reference range 1.7–2.4 U/UCS). Blood and liquor parameters were within normal limits. Biopsy findings and COX deficiency were consistent with the diagnosis of a multisystemic mitochondriopathy (MP). While waiting for the laboratory results the dog was sent home without treatment. During the following three weeks a complete remission of the clinical signs could be observed and there was no relapse until now (4 months). This is the first report of an adult-onset MP in a dog. Since exposure to toxic agents could be ruled out, this COX defect most likely results from a mutation with mild phenotypic penetrance. Sudden onset of clinical signs may be explained by transient increase in energy requirements due to performance or stress. A relapse of clinical signs is to be expected as congenital MPs worsen more rapidly durisenescence. A supportive therapy with radical scavengers or coenzyme Q10 might be helpful for prolongation of the asymptomatic interval.

ABSTRACT # 25

MALFORMATION OF CORTICAL DEVELOPMENT ASSOCIATED WITH REFRACTORY EPILEPSY. Cantile C., Salvadori C., Arispici M. and Gandini G. *Department of Animal Pathology – University of Pisa; *Department of Veterinary Clinical Sciences – University of Bologna (ITALY).

Reports of seizure activity due to malformation of cortical development (MCD) in animals, as well as descriptions of such neural malformation, are very rare. Here we describe the clinical and neuropathological features of a dog and a cat affected with seizure activity refractory to therapy.

A 3-month-old mixed breed male dog was referred because of severe seizure activity starting one month before. Seizures were characterized by tonic-clonic generalized activity including autonomic signs. Frequency of seizures activity was progressively increasing and, at presentation, was represented by daily cluster of 6 to 10 seizures. Blood parameters and CSF were normal. Phenobarbital therapy (3 mg/kg BID), instituted after the neurological examination, was ineffective. The dog was euthanized upon owner's request a few days later.

A 2.5-month-old domestic shorthaired male cat presented a rapidly progressive seizure activity, which in a few days reached the frequency of one fit every 15 minutes. The other littermates were normal and no history of toxin exposure was reported. Administration of benzodiazepine was ineffective and the cat was euthanized upon owner's request.

In both cases, neuropathological examination revealed anomalies of the cortical hemispheres. In the dog, there was loss of the normal distinction between gray and white matter, shallow sulci and mild enlargement of the ventricular cavities. Histologically, there was disorganization of cortical lamination with presence of large, randomly located cytomegalic neurons with abundant pale staining cytoplasm, often expressing phosphorylated neurofilaments. The histopathological pattern was consistent with a form of cortical dysplasia of Taylor type.

In the cat brain, gyri and sulci appeared roughly developed, giving a bumpy appearance of the cortical surface. There was absence of the corona radiata, irregularly thickened cortex, loss of sulci and broadening gyri. Histologically, the cortical architecture was highly disordered with no recognizable lamination and no preservation of the molecular layer. Often, thin white matter tracts intermingled with the most superficial cortex. The macroscopic and histopathological pattern was consistent with a form of type II lissencephaly.

Although there is no direct evidence that MCD in our cases were actually accountable for epileptogenesis, the severe clinical form, the young age of the animals and the neuropathological pattern of our cases are likely consistent with a congenital brain disorder. We suggest that – although rare – MCD may be responsible of the development of seizure activity in young animals and that, at least in some instances, the application of MRI techniques might facilitate the in vivo identification of cortical malformations in animals with epilepsy.

ABSTRACT # 26

PERMETHRIN INTOXICATION IS STILL RELEVANT IN CATS. I.C. Boettcher, H.C. Schenk, A. Tipold. Dept. of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Germany.

In the 90ies, several letters to the editors and some publications emphasized feline permethrin intoxication. Cats can get intoxicated when highly concentrated permethrin products are inappropriately applied. Obviously, pet owners are not yet sufficiently educated by veterinarians since this intoxication is still frequently observed.

Of 41 cats presented with history of intoxication at the Dept. of Small Animal Medicine and Surgery of the University of Veterinary Medicine Hannover between Jan 2002 and May 2005, ten patients showed clinical signs after contact with permethrin. Age, breed, sex, body weight, dose of permethrin, route, clinical manifestation, time of first signs and duration were retrospectively evaluated. In addition, laboratory changes, treatment and outcome were recorded.

Nine domestic shorthair cats and 1 siamese cat with a median age of 4.75 years (range 2.5–10.4) and a median body weight of 5 kg (range 3.2–5.7) were affected. All 6 males and 4 females were castrated. All cats had received a dermal application of a spot-on product designated for dogs. The amount varied between 2 drops to a full ampule. The owners noticed first signs after 2 to 24 hours (median 6.5). The clinical manifestation lasted between 12 hours and 4 days (median 30 hours). The most frequently observed signs were tremor (n=8), mydriasis (n=7) and generalized seizures (n=6). When tremor and seizures were present, tachycardia (n=5), panting (n=1), ventricular extrasystoles (n=3) and hyperthermia (n=4) occurred. In addition, ataxia (n=3), apathy (n=3), unconsciousness (n=2), salivation (n=1), proprioceptive deficits (n=1), reduced menace response (n=1), reduced pupillary light reflexes (n=1) and absent palpebral reflex (n=1) were recorded. The neurological examination revealed a diffuse intracranial lesion that comprised forebrain and brainstem. Laboratory changes included hemoconcentration (n=2), azotemia (n=2), leukocytosis with left shift (n=2), slight hyperglycemia (n=2), hyperkalemia (n=1) and acidosis (n=5). In all cats, cholinesterase activity was normal. The treatment was based on decontamination, control of tremor and seizures and symptomatic therapy. One owner elected euthanasia after 24 hours. All other cats were discharged without neurologic symptoms after 2 to 9 days (median 4.5).

The clinical signs of the 10 presented cats are similar to the literature. The route of intoxication was exclusively via dermal application. In other studies, 7% to 37% of affected cats are euthanized or die. Except of one euthanized patient, all cats in this study survived. With early and consequent treatment, prognosis in permethrin intoxication is good. Despite permethrin intoxication has not been mentioned in the literature in recent years, we want to emphasize that owners still inappropriately use permethrin products on cats and should be informed about the potentially lethal intoxication in their pets.

ABSTRACT # 27

QUANTITATIVE EVALUATION OF SPINAL DECOMPRESSION AFTER VENTRAL SLOT SURGERY FOR CERVICAL INTERVERTEBRAL DISC DISEASE USING COMPUTED TOMOGRAPHY. P. Böttcher, T. Flegel, K. Schmerbach, E. Ludewig, I. Kiefer, V. Grevel. Department of Small Animal Medicine, University of Leipzig, Leipzig, GERMANY.

The ventral slot procedure is the preferred surgery for ventral spinal cord compression caused by intervertebral disc disease in the cervical spine.

Limited access and visualization of the spinal cord usually prevent objective assessment of achieved decompression. The objective of this study was to quantify the degree of spinal cord decompression achieved by ventral slot procedure for cervical none-dynamic intervertebral disc disease. Nine dogs of different breeds were included in this ongoing prospective study. All patients showed a none-dynamic ventral extradural spinal lesion between C1 and Th2 on myelography. In addition spinal computed tomography (CT) of the cervical region was performed immediately after myelography and after finishing the ventral slot procedure. For quantitative analysis of spinal decompression both CT-myelographic image sets were matched using intensity-based image-registration algorithms, in order to assure measurements at exactly the same anatomical position pre- and postsurgically. Compression of the spinal cord was classified at five defined anatomical positions based on visual inspection of aligned and axially reconstructed images as severe, moderate, slight or absent. Furthermore the area of the spinal cord was outlined by hand in both image sets within a digital image-editing program and measured in mm^2. In case in which no contrast column could be clearly identified no judgment or measurement were made. Visual and quantitative evaluation of spinal compression and decompression was made at: 1. axial image centred over the affected intervertebral disc 2. axial images at 25% and 50% of the length of the adjacent vertebral bodies away from the first measuring point in both directions. Quantitative measurement of spinal decompression produced unreliable results. Due to the high variability within the manually outlined spinal cord areas, statistical interpretation of the results was not possible. In contrast, subjective evaluation of the degree of preoperative spinal compression and achieved postoperative decompression was straight forward and highly reproducible. In all patients at least some degree of decompression was seen on post-surgical images. However only in three of the nine cases complete decompression could be demonstrated along all five measurement points. Two cases with moderate preoperative compression showed an almost unchanged degree of compression after the ventral slot procedure.

CT-myelography is a valuable tool investigating spinal decompression after ventral slot procedure. However the predictive value of the degree of postoperative decompression on short- and long-term outcome has to be investigated in the future. Quantitative assessment of spinal decompression by CT-myelography is technically challenging and has to be substantially improved before further recommendations can be made.

ABSTRACT # 28

THE PROGNOSTIC SIGNIFICANCE OF INTRAMEDULLARY MRI FINDINGS IN CANINE INTERVERTEBRAL DISK DISEASE. C. Bull[1], A. Gerdwilker[1], M. Beyerbach[2], A. Tipold[1]. Department of Small Animal Medicine and Surgery[1], Institute of Biometry, Epidemiology and Information Processing[2], University of Veterinary Medicine, Hannover, Germany

Today intervertebral disk disease is frequently diagnosed using magnetic resonance imaging (MRI). In several dogs an area of hyperintensity can be found in the spinal cord at the site of compression, sometimes accompanied by an enlargement of the central canal. Nevertheless, these alterations are not seen in all dogs with disk disease. Therefore, the question of this study was to determine whether intramedullary MRI findings can be used as prognostic factors for clinical outcome in dogs with intervertebral disk disease.

Medical records of 63 dogs presented with neurological deficits resulting from cervical or thoracolumbal intervertebral disk disease were reviewed. MRI was performed using a 1.0 TESLA system (Magnetom Impact Plus, Siemens, Erlangen, Germany) with T2-weighted images in sagittal (TR 4700 ms, TE 112 ms) and transversal (TR 3458 ms, TE 96 ms) planes. The dogs were classified on the basis of severity of clinical signs at the time of initial examination and anamnestic treatment with corticosteroids. The MR images revealed an intramedullary hyperintensity in 28 of the 63 dogs (in 26 dogs up to the length of one and in 2 dogs two times the length of the vertebral body) adjacent to the area of extruded disk material. 15 of these dogs additionally had an enlargement of the central canal. The remaining 35 dogs showed a normal signal intensity of the spinal cord. 79% of the alterations were found in the thoracolumbal area (T11-L3). The time span from onset of neurological signs, the anamnestic treatment with corticosteroids and the neurological status had no significant ($p>0.05$) influence on these MRI findings. However, in dogs without deep pain perception a normointense spinal cord never occurred. 55 dogs regained the ability to walk unaidedly with or without having slight proprioceptive deficits and ataxia after decompression. MRI findings had no significant influence on this outcome ($p>0,05$). Nevertheless, the 2 dogs revealing a hyperintensity extending the length of two vertebral bodys showed poor functional results. There was a significant ($p<0.05$) longer recovery time for dogs with severe neurological deficits. In addition there seems to be an influence of the intramedullary changes on this value (Ø= 11 days for dogs with a normointense spinal cord, Ø= 19 days for dogs showing a hyperintense spinal cord and Ø= 25 days for dogs being diagnosed with a combination of spinal cord hyperintensity and enlargement of the central canal), but these differences were not significant ($p>0.05$).

The results of the present study let suggest that dogs with intervertebral disk disease, in which MRI shows an area of hyperintensity of the spinal cord up to the length of one vertebral body, with or without an enlargement of the central canal, do not have a poor prognosis for functional outcome, but these findings may have a negative effect on the recovery time.

ABSTRACT # 29

EMERGENCY EXTERNAL VENTRICULAR DRAIN IN A CASE OF ACUTE HYDROCEPHALUS. Rodolfo Cappello, Holger Volk, Kate Chandler, Dan Chan. The Royal Veterinary College, University of London, UK

A 13 month old, male entire, Akita, was referred to the Queen Mother Hospital for Animals with a history of progressive ataxia, circling to the left, drooling and vomiting. The dog following received a dose of steroids and ampicillin from the referring veterinarian. The following day the dog developed episodes of opisthotonus and nystagmus. At arrival, the dog was in decerebellate posture, had positional nystagmus, and had a depressed mental status. The dog deteriorated overnight: the mental status became stuporous, the tongue showed fasciculation, there was a dorsolateral strabismus, severely reduced oculo-vestibular response and the pupils were pin-point. The clinical signs were consistent with a severe brainstem compression due to a cerebellar herniation. Complete blood count, biochemistry and urine analysis were within the normal limits. The following day the dog underwent to a magnetic resonance imaging (MRI) examination of the head which revealed a large hydrocephalus involving the lateral ventricles, quadrigeminal cistern and 3rd ventricle. The cerebellum appeared displaced caudally and a large portion of the vermis was herniated under the occipital bone causing a severe deformation and compression of the brainstem. The cerebellum showed some areas of hyperintensity on T2 weighted images suggesting a parenchymal oedema. The diagnosis was of acute hydrocephalus and an emergency external ventriculotomy was performed. The position of the ventriculotomy was estimated on the images and was performed through the parietal bone, midway from the superior margin of the orbit and the occipital crest and 1 cm from the dorsal midline. The skin was surgically prepared and incision on the skin was performed with a number 10 blade. A 18G Jamshidi needle for bone biopsy was used to create the hole in the parietal bone. Once the bone defect was obtained an 18G IV catheter was inserted. The catheter was connected to a closed drainage system and cerebrospinal fluid (CSF) was collected for analysis. The CSF pressure was monitored and when the pressure was over 10 mmHg the CSF was drained. The catheter and relative connection were dressed before the anaesthetic gas was turned off. The dog recovered from the anaesthesia uneventfully and already showed signs of improvement. The dog was sent to the intensive care unit (ICU) and CSF pressure and clinical signs were monitored for any signs of worsening. Interestingly, the clinical signs worsened when the CSF pressure was above 12 mmHg, the CSF was drained until the pressure normalised. The night following the emergency ventriculotomy, the dog was stable and showed signs of improvement however, in the early hours of the morning the catheter was occluded (due to kinking) and the clinical signs started worsening again. The following morning a permanent ventriculoperitoneal shunt was placed by routine surgical technique described by various authors. The dog progressively recovered from the surgical ventriculo-peritoneal shunt and was normal 3 days late

Conclusion: Emergency external ventriculotomy can be an effective temporary treatment for acute hydrocephalus. The technique described is relative easy and of low cost. This technique allowed us to acquire the correct permanent ventricular shunt and plan the placement adequately. The authors would like to stress the importance of diagnostic imaging to reach the diagnosis and the important role of intensive critical care in this case. Six months later, the dog is conducting a normal life.

ABSTRACT # 30

NEUROBLASTOMA IN THE FOURTH VENTRICLE OF A HORSE. F Caporelli[1], S Añor[2], I Tarancón[1], M Pumarola[3], L Monreal[1]. [1]Servei de Medicina Interna Equina, [2]Servei de Neurologia i Neurocirurgia & [3]Unitat de Neuropatologia, Facultat de Veterinària, Universitat Autònoma de Barcelona, Barcelona, Spain.

The purpose of this report is to describe the clinical signs, progression and histopathologic features of an intracranial neuroblastoma in a horse.

A two year-old Andalusian-bred stallion was referred for evaluation of an acute onset of ataxia, leaning to the left and prolonged recumbency periods, probably related to a previous head trauma episode. On admission, the horse was in left lateral recumbency and obtunded. Physical exam, CBC, biochemistry and blood gasses were within normal limits. Neurological examination performed on sternal recumbency revealed left pleurotonus, decreased left pupillary and palpebral reflexes, and absent left menace response. CSF analysis revealed xantocromia. Based on the deficits found, the neuroanatomic localization of the lesion was considered to be diencephalic-rostral brainstem. No signs of trauma that could explain the neurological signs were found. The horse's neurologic condition worsened rapidly, showing a a great impairment of mental status and presence of signs of increased ICP (bradycardia, spontaneous horizontal nystagmus). Treatment at this time included intranasal oxygen, intravenous mannitol and fluid therapy, DMSO, anti-convulsive/sedative medication (phenobarbital) and antibiotics. Due to the poor prognosis and fast worsening of clinical signs, the horse was euthanized at the owner's request. At necropsy, a 4 cm diameter mass was found in the fourth ventricle, compressing the cerebellum dorsally and the medulla oblongata ventrally. Histopathological and immunohistochemical studies (GFAP, Cytokeratin, NSE) confirmed that the mass was a primitive neuroectodermal tumor (PNET) with neuronal differentiation.

Brain tumors are extremely rare in horses, with the exception of pituitary adenomas. Isolated reports of other primary brain tumors in horses (choroid plexus papilloma, oligodendroglioma, ependymoma) can also be found in the literature. On the other hand, PNETs have been described in the eye (malignant medulloepitheliomas) and pineal gland (pinealoblastomas) of horses, but there are no reports of this type of tumors affecting the CNS in this species. PNETs are reported to occur commonly in young cattle and dogs, and have been sporadically observed in pigs and cats but, to our knowledge, this is the first report of a PNET growing within the fourth ventricle and affecting the brainstem and cerebellum of a horse.

ABSTRACT # 31

MAGNETIC RESONANCE IMAGING FEATURES OF TRAUMATIC INTERVERTEBRAL DISC EXTRUSION IN DOGS. [1]Yapei Chang; [2]Ruth Dennis; [2]Simon R. Platt; [1]Jacques Penderis [1]Institute of Comparative Medicine, Division of Companion Animal Sciences, Faculty of Veterinary Medicine, University of Glasgow; [2]Animal Health Trust, Newmarket

Intervertebral disc disease in the dog was first classified by Hansen as type I, where herniation of the nucleus pulposus occurs through the annular fibres of the disc into the spinal canal, and type II, where annular protrusion into the spinal canal is caused by shifting of the nucleus pulposus material. Both Hansen type I and type II disc disease are always preceded by disc degeneration. Traumatic disc extrusion may also occur where trauma causes rupture of a disc that demonstrated no obvious preceding degenerative changes, resulting in spinal cord injury with no or minimal residual compression. Because the nucleus pulposus material is not demonstrating degenerative changes and is normally hydrated it diffuses within the epidural fat, leaving only the secondary changes attributable to acute spinal cord contusion and in some cases subtle spinal cord compression.

This retrospective study evaluated the magnetic resonance (MR) imaging features of traumatic disc extrusion in 11 dogs. Clinical databases at the University of Glasgow Veterinary School and Animal Health Trust were searched retrospectively for dogs presenting with clinical and MR imaging features suggestive of traumatic disc extrusion. Only cases that met all of the following criteria were included: 1) focal neurological deficits localised to the cervical, thoracic or lumbar spinal cord, 2) acute, non-progressive onset of neurological deficits coinciding with a traumatic event, 3) no obvious preceding disc degeneration at the lesion site on MR imaging, 4) reduced disc space, 5) minimal to no spinal cord compression, and 6) parenchymal changes within the spinal cord adjacent to the affected disc space. The size and signal intensity of the nucleus pulposus and the width of the disc space of the affected disc were compared to adjacent normal discs on mid-line sagittal T_2-weighted MR images using image analysis software (Image-Pro® Plus, Media Cybernetics, Silver Spring). Statistical analysis was performed using the paired t-test.

The MR imaging findings included reduction in volume and signal intensity of the nucleus pulposus, focal hyperintensity within the overlying spinal cord on T_2-weighted MR images and subtle spinal cord compression, extraneous material or signal change within the vertebral canal. The maximal area of spinal cord hyperintensity was located directly over or close to the affected disc space, appeared asymmetrical and in the majority of cases the extent was less than one vertebral length. Parenchymal spinal cord haemorrhage was identified in four dogs. Vacuum phenomena, evident as a signal void in the centre of the disc, were identified in two dogs.

The MR imaging appearance is distinct from that reported for other causes of spinal cord dysfunction and is useful in the ante mortem diagnosis of traumatic disc extrusion.

ABSTRACT # 32

MAGNETIC RESONANCE IMAGING FEATURES OF SPINAL EPIDURAL EMPYEMA IN FIVE DOGS. A. de Stefani[1], L.S. Garosi[2] , J.F. McConnell[1], F.J. Llabres Diaz[2], S.R.Platt[1]. [1]The Animal Health Trust, Newmarket, UK. [2]Davies Veterinary Specialists Higham Gobion, UK.

Spinal epidural empyema (SEE) is defined as an extensive accumulation of purulent material in the epidural space of the vertebral canal. SEE should be considered as a differential diagnosis in dogs presenting with pyrexia, spinal pain and a rapidly progressing myelopathy. The specific diagnosis of SEE requires a careful evaluation of the complete clinical spectrum including history of potential risk factors, laboratory data and results of imaging studies. Because of its minimally invasive nature and its high anatomical detail MRI is the imaging test of choice in human medicine. The aims of this study are to describe the MRI features of 5 dogs with confirmed SEE.

Inclusion criteria were a complete medical history, clinical signs compatible with spinal cord neurolocalisation, MRI of the spine and surgically confirmed diagnosis of SEE by way of histology and/or culture. MRI of the spine was acquired using a 1.5Tesla scanner in 3 cases and a 0.4Tesla scanner in 2 cases. Each MRI examination was reviewed by a board-certified radiologist.

The following information was recorded: location of the lesion, cranio-caudal length of the lesion expressed as a ratio relative to L2, enhancement pattern of the abnormal epidural tissue, presence of abnormal signal in the spinal cord, severity of the neural structures' compression and presence of abnormal signal within the vertebrae adjacent to the lesion and/or within the paraspinal soft tissues. All dogs presented with severe back pain. Two dogs were pyrexic and 3 dogs showed leucocytosis. Three dogs were paraparetic of which one was non-ambulatory, two dogs were paraplegic but maintained nociception in both pelvic limbs. Lower motor neuron signs were present in three dogs. The lesion was dorsally located in two cases and ventrally located in three cases. Average cranio-caudal length of the lesion expressed as a ratio relative to L2 range from 1.8 to 5.4. All patients had narrowing of the vertebral canal by over 50%. MRI depicted the epidural lesions as high or mixed signal masses in T2W images. Increased signal within the spinal cord grey matter at the site of the lesion was detected in T2W images in all cases. Two patterns of enhancement were detected on post contrast T1W images. Mild to moderate peripheral enhancement was described in three cases and a diffuse pattern of enhancement was present in one case. Discospondylitis was clearly demostrated in 3 cases on T1W post contrast images.

Decompressive spinal surgery was carried out in all cases. One dog was euthanised oneweek after surgery. The remaining dogs improved neurologically after surgery and were hospitalised for a mean of 8 days. Cultured bacteria from the abnormal epidural tissue included, Enterobacter cloacae, coagulase positive Staphylococci, Pasteurella multocida and Escherichia.-coli. One dog had negative culture. MRI clearly identified the epidural lesion in all five cases in this study, allowing prompt diagnosis and appropriate treatment planning.

ABSTRACT # 33

TREATMENT OF POLYRADICULONEURITIS WITH HUMAN INTRAVENOUS IMMUNOGLOBULIN IN FIVE DOGS. A Fischer[1], K Jurina[2], T Steinberg[1], K Hirschvogel[1], L Matiasek[1], K Matiasek[3] [1]Section of Neurology, Department of Small Animal Medicine, Ludwig-Maximilians-University, Munich, [2]Small Animal Hospital Haar, [3]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, Ludwig-Maximilians-University, Munich, Germany

Treatment of polyradiculitis in dogs is usually restricted to physical rehabilitation and supportive care. Many times, dogs will recover within three weeks without therapy but prolonged courses up to three months are also possible. Glucocorticoids may ameliorate clinical signs in chronic demyelinating polyradiculoneuritis but efficacy in acute polyradiculitis has been questioned. In human neurology intravenous immunoglobulins (IVIG) and plasmapheresis are widely used for treatment of Guillain-Barrè syndrome, the human counterpart of acute canine polyradiculoneuritis.

Acute polyradiculitis was diagnosed in four dogs based on acute onset of non-ambulatory lower motor neuron tetraparesis, distal paresthesias, and widespread spontaneous electric activity on needle electromyography with normal to reduced nerve conduction velocities (n=5), low amplitude CMAPs and absent F-wave recordings and supported by results of CSF analysis (n=4) and muscle/nerve biopsies (n=3).

Human IVIG (0.5-1g/kg) was infused intravenously in an attempt to shorten recovery time and effort for intensive care for the five paralyzed dogs.

A positive response to treatment with IVIG was noted in each dog. Steady improvement following infusion of IVIG was evident in each dog. Three dogs walked without support within ten days. Improvement of motor funtion was noted in the other two dogs, but when the dogs were still non-ambulatory two weeks later IVIG infusion was repeated. Both dogs walked

without support within one week. One dog experienced recurrences twelve and 14 months following the initial episode, and two dogs within one and two months, respectively. Each time treatment with IVIG was repeated and rapid long-term improvement of motor function was observed.

It was concluded that human IVIG infusion may speed recovery in acute canine polyradiculoneuritis. A prospective blinded treatment study is warranted.

ABSTRACT # 34

MAGNETIC RESONANCE IMAGING IN A LABRADOR RETRIEVER WITH LEUKOENCEPHALOPATHY. K.M.Flatz[1], A.Bruehschwein[1], I.Holz[1], I.Foltin[2], K. Matiasek[3], U.Matis[1]. [1]Department of Veterinary Surgery, University of Munich, Germany, [2]Praxis Dr. Rieden, Heidelberg, Germany, [3]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, University of Munich, GERMANY.

Magnetic resonance imaging (MRI) is a well-established method of diagnosing leukoencephalopathy in human medicine. Leukoencephalopathy has also been described in the veterinary literature.

A 12-year-old spayed female Labrador retriever was presented with episodes of progressive head tremor and front limb weakness. At the time of presentation, the dog was bright, alert and responsive. However, the patient had difficulty rising and stumbled while walking. Postural reactions were normal in the front limbs and delayed in both hind limbs. The patellar reflexes were delayed. All other reflexes were normal. The cervical spine was slightly painful on extension. The results of haematological and biochemical analyses were within normal ranges. After a neurological examination, MRI of the brain and cervical spinal cord was carried out at 1.5 Tesla using a Siemens Magnetom Symphony MR scanner. T1- and T2-weighted sequences in transverse, sagittal und dorsal orientations were obtained. Moreover, a T2-weighted FLAIR sequence was done. Additional T1 post-contrast studies were obtained after intravenous injection of 0.1 mmol/kg gadolinium-DTPA.

The T2 TSE and FLAIR sequences showed bilaterally symmetric hyperintensities within the periventricular white matter of the parietal and occipital lobes. No abnormal contrast enhancement was seen on T1-weighted images. The spinal cord showed no abnormal signal intensities. Based on the MRI findings, the differential diagnosis included leukoencephalopathy, edema and multilobar CNS tumours, such as gliomatosis cerebri. The neurological signs worsened during the following year. Although the dog received a low dose of prednisolone daily, it was unable to rise on its own and knuckling was observed in the front limbs. At clinical presentation, all postural reactions were delayed. A second MRI examination, using the same sequences, was carried out eleven months later. No significant changes in size and signal intensities were detected in the hyperintense areas that were seen in the T2-weighted images the year before. As well, there was no contrast enhancement. The lateral ventricles were larger than in the previous images, probably due to loss of periventricular parenchyma (hydrocephalus e vacuo). Because of the severity of the clinical signs, the dog was euthanased and underwent a postmortem examination. The tentative diagnosis of a bilateral leukoencephalopathy was confirmed by histological examination. Leukoencephalopathies are rare in dogs, but should be considered in the differential diagnosis when there is a bilaterally increased signal intensity of cerebral white matter in T2-weighted magnetic resonance images.

ABSTRACT # 35

CEREBRAL CHOLESTEROL GRANULOMA IN A CAT. Gaby Fluehmann[1], Martin Konar[2], André Jaggy[3], Alexandra Nicolier[4], Marc Vandevelde[5]. [1]Dr.med.vet. Gaby Fluehmann, Resident ECVN, Department of Clinical Veterinary Medicine, Division of Animal Neurology, University of Bern, Länggasstrasse 128, 3012 Bern, Switzerland. [2]Dr.med.vet. Martin Konar, Resident ECVDI, Department of Clinical Veterinary Medicine, Division of Radiology, University of Bern, Länggasstrasse 128, 3012 Bern, Switzerland. [3]Prof.Dr.med.vet, André Jaggy, DECVN, PhD, Department of Clinical Veterinary Medicine, Division of Animal Neurology, University of Bern, Länggasstrasse 128, 3012 Bern, Switzerland. [4]Dr.med.vet. Alexandra Nicolier, DECVP, Department of Clinical Veterinary Medicine, Division of Animal Neurology, University of Bern, Länggasstrasse 128, 3012 Bern, Switzerland. [5]Prof.Dr.med.vet. Marc Vandevelde, DECVN, Department of Clinical Veterinary Medicine, Division of Animal Neurology, University of Bern, Länggasstrasse 128, 3012 Bern, Switzerland.

A 13-year-old, female, spayed, domestic shorthair cat was presented with apathy, disorientation, rigid head and neck posture, compulsive walking, generalised sensory ataxia and proprioceptive deficits of the left hind and front limbs. The menace response on the left side was decreased and the cervical vertebral column appeared painful on palpation. The MRI showed an extensive space-occupying lesion in the area of the falx cerebri. It compressed both hemispheres and deformed the third and lateral ventricles, as well as the thalamus. The lesion showed a peripheral irregularly shaped hypointense rim in all sequences (FSE T2, FE T2*, FE 3D T1w, FLAIR, STIR, FE 3D MPR), but was most obvious in the T2*-weighted sequence. The inner parts of the mass showed inhomogeneous, in T2* and T1 mainly hypointense, in STIR, FLAIR, and T2 mainly hyperintense signal intensity. The inhomogeneous, mainly hypointense signal intensity in T1 and T2* was interpreted as hemorrhage, calcification, or cholesterol. The differential diagnosis included meningioma or cholesterol granuloma. On pathological examination the space-occupying lesion consisted of a well-demarcated encapsulated mass located in the longitudinal cerebral fissure dorsal to the corpus callosum. The lesion was entirely extracerebral and completely separated from the nervous parenchyma by the meninges. The histopathological findings were consistent with a diagnosis of cholesterol granuloma. Cholesterol granulomas have been reported in human medicine at different sites throughout the body. In veterinary medicine, they have mostly been described in horses and only sporadically in other species. In the majority of cases they were found in the choroids plexus of the lateral or fourth ventricle. This is the first report of an intracranial cholesterol granuloma in a cat.

ABSTRACT # 36

CANINE CUTANEOUS PERIPHERAL NERVE SHEATH TUMORS: A HISTOPATHOLOGICAL AND IMMUNOHISTOCHEMICAL STUDY. Lluís Gaitero, Sònia Añor, Dolors Fondevila, Martí Pumarola, Departament de Medicina i Cirurgia Animal, Facultat de Veterinària, Universitat Autònoma de Barcelona, Bellaterra, Barcelona, SPAIN.

In veterinary medicine, the term peripheral nerve sheath tumor (PNST) is usually restricted to neoplasms closely associated with an identified nerve. However, a subgroup of cutaneous PNSTs suspected to arise from unidentified small peripheral nerves has been considered. In addition, clearly established diagnostic criteria do not exist for PNSTs due to their inconsistent immunohistochemical features. The aim of the present study was to retrospectively evaluate the histological features and analyze the immunohistochemical staining pattern of canine subcutaneous spindle cell tumors resembling peripheral nerve sheath tumors.

The study included archived samples from 29 canine cutaneous tumors previously classified as spindle cell tumors consistent with schwannomas from biopsy samples received from 1999 to 2004 by the Veterinary Pathology Service of the Universitat Autonoma de Barcelona. Five canine cutaneous fibrosarcomas were also included as controls. All tumors were classified by several morphologic criteria after human and canine literature review. Their immunohistochemical expression of vimentin, S-100 protein, glial fibrillary acidic protein (GFAP), neuron specific enolase (NSE) and protein gene product 9.5 (PGP 9.5; a broad neural marker not previously reported in any canine neoplasm to date) was also studied.

Microscopic examination showed masses predominantly not-well encapsulated and heterogeneous appearance. Three different histological patterns could be identified in the same tumor: a) Dense areas of spindle-shaped cells often arranged in interlacing bundles and fascicles (resembling Antoni A schwannoma pattern); b) Less cellular areas with more pleomorphic cells (resembling Antoni B schwannoma pattern); c) Whorled pattern around collagen. Small nerve fibers could be ocasionally identified within tumors (4/29). Vimentin was uniformly and strongly positive in all cases. S-100 was expressed in 13/29 of the selected tumors; when present, the pattern of expression was always scattered and patchy. PGP 9.5 staining was displayed in 28/29 tumors of the selected cases and in 4/5 fibrosarcomas.

In conclusion, the morphologic and immunohistochemical findings of these canine cutaneous spindle cell tumors were consistent with PNSTs. Some histological features suggest they should be classified mostly as low-grade malignant PNST. Immunohistochemical staining with vimentine, S-100, PGP 9.5, GFAP and NSE showed limited value in classifying them definitively as PNSTs,. According to the human classification, we suggest that at least some of them, specially those with characteristic Antoni A and Antoni B growth pattern and S-100 protein expression, might be classified as ''cutaneous Schwannomas''.

ABSTRACT # 37

A RETROSPECTIVE STUDY OF AORTIC THROMOBEMBOLISM IN 11 DOGS. Rita Goncalves[1], Jacques Penderis[1], Yai Pei Chang[1], John Mosley[2] T. James Anderson[1]. [1]Institute of Comparative Medicine, Faculty

of Veterinary Medicine, University of Glasgow, Glasgow; [2]Division of Veterinary Clinical Studies, University of Edinburgh, Roslin

Aortic thromboembolism (AT) is a common disorder in cats typically characterised by an acute onset of paraparesis or paraplegia, absent femoral pulses, cyanotic nail beds and swollen musculature. AT in cats is primarily associated with underlying cardiac disease. In contrast to cats a variety of underlying causes are reported in dogs, with the most common including cardiac disease, neoplasia, hyperadrenocorticism, disseminated intravascular coagulation and sepsis. The condition in dogs is also considered rare and is poorly documented with no detailed description of the neurological presentation.

The medical records of 11 dogs presented as neurology referral cases and that were diagnosed with aortic thromboembolism as the cause for the clinical signs were reviewed. There was an apparent male predisposition (8/11) and the Cavalier King Charles Spaniel (CKCS) breed was over-represented (5/11). Mean age of presentation was 8.3 years (range 6–10.9 years). The onset was acute in 4 dogs, chronic in 5 dogs (with all of these presenting as exercise intolerance) and chronic with acute deterioration in 2 dogs. In 7 dogs there was progression of the clinical signs. In 6 dogs the onset was associated with the presence of pain. Mean duration of clinical signs was 40 days (range 3–240 days), with 3 dogs presenting during the first week and 7 dogs after the first month. The locomotor deficits included exercise intolerance with pelvic limb weakness (5/11), pelvic limb ataxia (1/11), monoparesis (1/11), paraparesis (2/11) and non-ambulatory paraparesis (2/11). The locomotor deficits were symmetrical in 7 dogs, asymmetrical in 3 dogs and only affected one pelvic limb in one dog. Neurological examination revealed conscious proprioception deficits in the hind limbs in 5 dogs and reduced spinal reflexes in 4 dogs. Pulse quality was reduced in 7/11 dogs and absent in 4/11. The thromboembolism was identified in all dogs by abdominal ultrasound and confirmed by post-mortem examination in 3 dogs. Systolic blood pressure measurements revealed hypertension in 1/8 dogs (SBP $\geq$ 200 mmHg). Urinalysis revealed proteinuria in 3/9 dogs. Echocardiography revealed endocardiosis in 5/8 dogs. Thoracic radiographs revealed evidence of metastatic disease in 1/10 dogs. Serum muscle enzyme levels were normal or mildly increased: mean creatine kinase was 291 U/l (range 86-1222U/l) and mean alanine aminotransferase was 37.5 U/l (range 19-436U/l). Suspected underlying disease processes identified were cardiac disease (3/11), protein losing nephropathy (2/11), protein losing enteropathy (1/11) and neoplasia (1/11). Four dogs are still alive and have improved (in 1 the thrombus has dislodged), 5 dogs were euthanased (in 6–450 days), 1 dog died within the first 12 hours and 1 dog was lost to follow-up.The clinical presentation of AT in dogs appears to segregate into two major groups according to the speed of onset of the clinical signs. Dogs with an acute onset of the clinical signs tend to be more severely affected, while dogs with a chronic onset predominantly present with the exercise intolerance. In both circumstances, AT should be considered as a differential diagnosis and conclusively ruled out.

ABSTRACT # 38

THE USE OF MASKING WHITE NOISE IN THE BRAINSTEM AUDITORY-EVOKED RESPONSE IN DOGS. [1]Rita Goncalves, [2]Julia Freeman, [1]Jacques Penderis. [1]Institute of Comparative Medicine, Faculty of Veterinary Medicine, University of Glasgow, Glasgow; [2]Animal Health Trust, Newmarket

The brainstem auditory-evoked response (BAER) is one of the most commonly used tests for assessing auditory function in dogs and is also useful as a site-of-lesion diagnostic tool. The BAER allows assessment of conductive auditory pathways in the outer and middle ears, sensory auditory structures in the inner ear, cranial nerve VIII, auditory parts of the brainstem and higher neural structures involved in auditory perception. Currently the BAER is the main validated technique for assessment of congenital sensorineural deafness in canine patients. All dog breeds may be affected by congenital sensorineural deafness, but the Dalmatian has the highest reported incidence of 18% in the UK (13% unilateral and 5% bilateral deaf) and 30% in the USA (22% unilateral and 8% bilateral deaf). While masking noise in the non-test ear is routinely performed in human auditory assessment and is sometimes used in canine auditory assessment, its clinical relevance has never been evaluated in dogs.

The BAER was evaluated in 15 Dalmatian puppies with confirmed unilateral deafness. The active electrode was placed on the vertex, reference electrode rostral to the tragus of the test ear and ground electrode on the dorsal neck midline. The BAER was elicited by 512 unilaterally applied 0.1ms click stimuli generated by earphones manually held against the opening of the external ear canals. The initial stimulus intensity in the deaf ear was 80 decibels normal hearing level (dBnHL) and subsequently increased to 100 dBnHL. The 100 dBnHL stimulus was then repeated with simultaneous application of 80 dBnHL white masking noise to the non-test (normal) ear.

At the 80 dBnHL intensity stimulus in the affected ear, 3 dogs had no distinguishable BAER waveforms but in 12 dogs a wave V was recognized in the BAER. Median wave V amplitude was 1 μV (range 0.5–1.3 μV) and median latency was 5 ms (range 4.8–5.7 ms). At the 100 dBnHL intensity stimulus, all dogs had a discernible wave V in the BAER of the deaf ear. Median wave V amplitude was 1.25 μV (range 0.5 2.3 μV) and median latency was 4.7 ms (range 4.3 5.4 ms). Following white masking noise at 80 dBnHL, no BAER waveforms could be identified in the affected ears.

The abolition of the wave V recognised in the BAER of the affected ear by the introduction of white noise masking at 20dB below the intensity of the click stimulus would suggest that this wave is the result of the crossover effect. In the crossover effect the click stimulus directed at the test ear stimulates the cochlea of the non-test ear, either as a result of noise leakage to the non-test ear or the click stimulus resulting in vibration of the skull and thereby of the contralateral cochlea. The results obtained support the need of the use of masking noise in the non-test ear in cases of asymmetrical hearing loss in order to eliminate the crossover effect, with the use of white masking noise at 20dB below the intensity of the click stimulus effective in abolishing this crossover effect.

ABSTRACT # 39

HYDRANENCEPHALY IN A DOMESTIC SHORTHAIR CAT: CT DIAGNOSIS. Hernández-Guerra, AM[1], López-Murcia MM, Ortega J[2]. [1]Departamento de Medicina y Cirugía Animal. Universidad Cardenal Herrera-CEU. Moncada. Valencia. Spain; [2]Joaquín Ortega. Departamento de Atención Sanitaria, Salud Pública y Sanidad Animal. Universidad Cardenal Herrera-CEU. Moncada. Valencia. Spain

Hydranencephaly is a rare congenital malformation involving total or near total absence or cerebrum, but with intact cranial vault and meninges. The cerebral hemispheres are replaced by cerebrospinal fluid (CSF)-filled sacs lined by leptomeninges, a glial membrane, and ependymal remnants. Dogs and cats may be affected. The pathogenesis of this anomaly is not always certain. In people, is thought to be due to vascular occlusive insult, post infectious vasculitis, or other trauma a fetal cerebrovascular accident. In animals, the most common cause is *in utero* viral infection. In cats has been linked with vaccine-induced intrauterine feline panleukopenia / parvovirus infections. Microphthalmia and lost of nerve fibres and reduced myelin staining in optic nerves has been reported. Other structures including the brainstem and cerebellum may or may not be affected. A three month old male DSH was presented from a local animal shelter for behavioural alteration. The cat did not interact with other cats and reacted aggressively against shelter staff, and was indifferent to the environment. The cat was able to eat, drink, urinate and defecate normally. The cat had difficulty walking. On neurological exam the cat was blind, proprioceptive deficits were present on four legs, oculocephalic and pupillary reflexes were absent, with a miotic and rotated inwards pupils. Rest of cranial reflexes were normal. CT examination was performed that showed absence of cerebral tissue and a remarkable increase of CSF. No cerebral mantle was observed and cerebral falx was present. This gave us a CT diagnosis of hydranencephaly. Differential diagnoses of increased CSF are extreme hydrocephalus, alobar holoprosencephaly, and porenchepaly. However, the presence of cerebral falx, the complete absence of cerebral mantle and the presence areas of normal cerebral parenchyma, made hydranencephaly the most likely diagnosis. The cat was euthanized for humane reasons. Postmortem examination confirmed the diagnosis, with a sac filled with CSF liquid instead of cerebral parenchyma, and a patency of cerebral falx. Hydranencephaly is a rare disease. It has been describe previously, usually associated to virus infections (including Akabane, Bluetongue and Rift Valley fever) in large animals, and linked with vaccine-induced intrauterine feline panleukopenia / parvovirus in cats. To our knowledge, this is the first CT description of a hydranencephaly in the veterinary literature. As in human medicine, its major differential is hydrocephalus. Clinically are difficult to distinguish, however in the human literature hydranencephaly usually presents with normocephaly rather than macrocephaly and normal facial appearance. This cat presented both signs, without a doom-shaped cranium and a ''sunset sign'' eyes. In human medicine it is normally detected sonographically before birth. It has also bedescribed a prenatal sonographic diagnosis in a Chihuahua. Hydranencephaly, unlike hydrocephalus, has no treatment options and therefore, has a poorer prognosis.

ABSTRACT # 40

COMPARISON OF FLAIR, T2-WEIGHTED, T1-WEIGHTED AND T1-WEIGHTED POST CONTRAST MAGNETIC RESONANCE IMAGES IN THE DETECTION OF INTRACRANIAL INFLAMMATORY AND

INFECTIOUS DISEASES IN THE DOG. S. Howson[1], S. Platt[2], E. Baines[1], R. Dennis[2], D. Brodbelt[1], G.B. Cherubini[1]. [1]Department of Veterinary Clinical Sciences, The Royal Veterinary College, University of London, UK. [2]Centre for Small Animal Studies, Animal Health Trust, Newmarket, UK.

Intracranial inflammatory and infectious diseases are common neurological conditions in dogs. Magnetic resonance (MR) has been considered the imaging modality of choice in the examination of the central nervous system (CNS).

The aim of the project was to compare the sensitivity of T1-weighted, T2-weighted, T1-weighted post administration of gadolinium and fluid-attenuated inversion-recovery (FLAIR) images in the detection of intracranial inflammatory and infectious diseases in dogs.

Medical records at The Royal Veterinary College and at the Animal Health Trust were searched for dogs that had had cerebrospinal fluid (CSF) analysis findings consistent with inflammatory disease (73 patients). Control patients were selected that had CSF findings negative for inflammatory disease and MR images not compatible with neoplasia (19 patients). The MR images were reviewed blindly by two boarder radiologists and each sequence was reviewed in isolation from the others. Magnetic resonance sequence sensitivities of CSF positive patients were compared with the Chi-squared test and logistic regression accounting for clustering at the patient level. Statistical significance was set at the 5% level.

FLAIR images were found to have the highest sensitivity (83.6% 61/73), followed by T2-weighted images (63.0% 46/73), T1-weighted post contrast images (61.6% 45/73) and then by T1-weighted images (23.3% 17/73) ($P<0.001$). In the logistic regression model after adjusting for clustering at the patient level, FLAIR images were 106.1 times (95% confidence interval (95% CI) 25.2 – 447.5) more likely to correctly identify CSF positive patients than T1-weighted images, 6.4 times (95% CI 2.2 – 18.2) more likely than T1-weighted post contrast images and 5.8 times (95% CI 2.0-16.4) more likely than T2-weighted images.

It was concluded that FLAIR was the most sensitive MR sequence for detecting intracranial inflammatory and infectious disease of the CNS in dogs.

ABSTRACT # 41

EEG FINDINGS IN EPILEPTIC AND HEALTHY FINNISH SPITZ DOGS. Janis Jeserevics, DVM, Ranno Viitmaa, DVM, Tarja Susanna Jokinen, DVM, Sigitas Cizinauskas, DVM, DECVN, Luciana Bergamasco, DVM, PhD

The aim of study was to describe visual and quantitative electroencephalography (EEG) parameters in epileptic Finnish Spitz dogs and compare them with healthy dogs of the same breed.

Fifteen epileptic Finnish Spitz dogs (FSE) and 16 healthy Finnish Spitz dogs (FSN) were examined. EEG recordings were performed in medetomidine (Domitor) sedation. A 14 channel monopolar montage was used to record bio–electrical activity. A method of standardized placement of EEG electrodes, similar to the 10–20 international system for the humans, was used. EEG recording was continued for 20 minutes.

At the visual examination paroxysmal activity or EEG burst-suppression were observed in FSE. Focal epileptic activity (10 FSE) was characterized by volleys of spikes and sharp waves with propensity to spread out contralaterally or generalize. Generalized epileptic activity (6 FSE) was characterized by volleys of polyspikes and wave complexes and synchronous and bilateral sharp waves. Seven FSN exhibited in their EEG pseudoepileptic activity characterized as phantom waves spikes, small sharp waves and occipital spikes. Therefore we made two subgroups: FSN-healthy dogs and FSPS-healthy dogs with pseudoepileptic EEG pattern. FSE showed significantly more beta and theta activity of their EEG as FSN and FSPS. FSPS have significantly more beta and theta activity as FSN. FSN had significantly more alfa and delta activity as the other groups.

Number of explanations for pseudoepileptic EEG pattern could be mentioned: (i) conditions described in humane medicine as pseudoepilepsy, (ii) subclinical epilepsy, (iii) drug related changes.

Focal idiopatic epilepsy seems to be more frequent in examined Finnish Spitz dogs group according to their EEG recordings.

Increased beta and theta activity is characteristic for FSE EEG.

ABSTRACT # 42

CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY WITH HYPERTROPHY OF CERVICO-THORACAL NERVE ROOTS IN A DOG. Iris Kathmann[1], Irene Christine Böttcher[2], Thilo von Klopmann[3], Axel Gerdwilker[4], Andrea Tipold[5]. [1]Dr. med. vet. Iris Kathmann, DECVN. Department of Clinical Veterinary Medicine, Section of Neurology, University of Bern, Länggassstrasse 128, 3012 Bern, Switzerland. [2]Dr. med. vet. Irene Christine Böttcher, Clinic for small animals, Stiftung Tierärztliche Hochschule, Bischofsholer Damm 15, 30173 Hannover, Germany. [3]Dr. med. vet. Thilo von Klopmann, Clinic for small animals, Stiftung Tierärztliche Hochschule, Bischofsholer Damm 15, 30173 Hannover, Germany. [4]Dr. med. vet. Axel Gerdwilker, Clinic for small animals, Stiftung Tierärztliche Hochschule, Bischofsholer Damm 15, 30173 Hannover, Germany. [5]Prof. Dr. med. vet. Andrea Tipold, DECVN, Clinic for small animals, Stiftung Tierärztliche Hochschule, Bischofsholer Damm 15, 30173 Hannover, Germany

A unusual case of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) in a Magyar Vizsla dame, 7 months of age, is described. The neurological deficits such as movement disorders, hyporeflexia and muscle atrophy, were limited to the front legs. The hypertrophied cervicothoracal nerve roots could be shown by magnetic resonance imaging (MRI). The diagnosis was additionally based on clinical findings, the relapsing course, the good response to therapy with prednisolone, the results of electrodiagnostic workup and muscle and nerve biopsy.

This case report showed that CIDP should also be considered as a differential diagnosis in premature animals with slowly progressive neurological deficits which are consistent with a lesion in the peripheral nervous system.

Additionally this is the first description of hypertrophic nerve roots in a dog shown by MRI. In human medicine MRI is used for diagnosis and for follow up in CIDP with hypertrophic neuropathy. We think that also in veterinary medicine MRI is a great diagnostic aid in CIDP as well as to document the hypertrophied nerve roots, especially in cases with suspicion of spinal cord involvement.

ABSTRACT # 43

LOW-FIELD MAGNETIC RESONANCE IMAGING IN DIAGNOSING INTERVERTEBRAL DISC PROLAPSE IN THREE CATS AND OUTCOME OF SURGERY. Diane Lu, Mongkok, HONG KONG

Three cats, (Case 1, 2 Persian cats, Case 3 domestic short hair cat) were presented with progressive paraparesis. Spinal pain was not a common feature (only in 1 cat). The neurological signs were suggestive of a upper motor neurone disease of the hindlimbs, i.e., a T3-L3 spinal cord segment lesion localisation was made in each cat. Advanced diagnostic imaging was used to investigate the spinal problem, by a 0.23-Tesla (low-magnetic field) open magnetic resonance machine. Sequences used include T1-weighted (pre- and post-gadolinium in selected cases) and B-FFE. An extradural compression could be identified over T3/4 (Case 1), T6/7 (Case 2), and T2/3 (Case 3) intervertebral disc space. In addition, the T2/3 appeared subluxated in Case 3. Interestingly, there was enhancement of the extradural compression in Case 1 and 2. No contrast study was performed in Case 3. Hemilaminectomy of the thoracic spine was performed in all three cats. In the first two cats a large piece of disc material confimed histologically, was removed from the ventral aspect of the vertebral canal. In Case 3 the prolapsed disc was only partially removed, and part of the vertebral body was curetted to decompress the cord. No internal fixation was applied as manipulation of the spine during surgery was considered to be stable. There was no epidural fat in the compressed region in all three cats and the cord appeared swollen to a certain degree. Case 1 deteriorated following surgery and became paraplegic, but gradually improved and regained ambulation. Case 2 improved following surgery and recovered, only very mild weakness was noticed two months postoperatively. Case 3 was stable following surgery, and gradual improvement was observed. The outcome was satisfactory in all three cats following spinal decompression. Low-field MR is capable of achieving good imaging studies in small spinal patients.

ABSTRACT # 44

IATROGENIC BRAINSTEM INJURY DURING CSF COLLECTION: CLINICAL AND MRI FINDINGS IN FOUR DOGS. Alejandro Luján Feliu-Pascual*, Laurent Garosi^, Simon Platt* and Ruth Dennis*. *The Animal Health Trust, Newmarket, UK; ^Davies Veterinary Specialists, Hitchin, UK

The purpose of the study is to characterize the clinical and MRI findings of caudal brainstem trauma caused by CSF collection from the cerebellomedullary cistern in four dogs.

Case 1, a 9-year-old male neutered Shih-tzu, presented as an emergency referral after a failed attempt to obtain CSF. On examination the dog was recumbent, with moderate obtundation, left pleurothotonus, weak gag reflex and tongue movements, postural opisthotonus and cranial cervical pain.

MRI study 27-hours after the failed CSF tap revealed a linear T2W hyperintense, T1W isointense and GE hypointense lesion in the myelencephalon with no contrast enhancement, consistent with a needle tract. The dog improved gradually with supportive treatment and was ambulatory three days later. Sudden deterioration with opisthotonus developed on day 12 and the dog was euthanased at the local practice. Case 2, a 6-year-old male WHWT, presented with sudden onset tetraplegia after repeated CSF collection attempts. On examination, he was stuporous, tetraparetic with decreased facial sensation and motor function, positional vertical nystagmus, reduced oculocephalic reflex, bilateral miosis and reduced gag reflex. MRI performed 24-hours after CSF attempt revealed two linear lesions in the myelencephalon similar to that seen in case 1, consistent with two needle tracts. The dog died a few hours after the MRI study of a respiratory arrest. Case 3, a 3-year-old male neutered Great Dane, presented with non-ambulatory tetraparesis, obtundation, left sided head tilt, positional nystagmus, increased spinal reflexes in the pelvic limbs and decreased on the thoracic limbs after a cervical myelogram was performed. MRI performed 36-hours after myelography showed a T2W hyperintense lesion extending from C2 spinal cord segment to the caudal cerebellar peduncle on the left side. GE and T1W sequences were not obtained. The dog was euthanased due to concurrent aspiration pneumonia. Case 4, a 4-year-old male Yorkshire Terrier, developed acute onset paraplegia after a fall. Attempts to perform a cervical myelogram were unsuccessful. Examination at the time of referral disclosed paraplegia and mild left thoracic flaccid paresis. Cranial nerve examination was normal. MRI performed four days after myelography revealed a linear lesion in the left myelencephalon which was hyperintense on both T2W and GE, consistent with a needle tract. The dog made a functional recovery.

In conclusion, the development of cranial nerve deficits after iatrogenic needle trauma carried a guarded prognosis in our series. Conspicuous T2W hyperintense linear lesions on midsagittal planes compatible with haemorrhage were evident in the most severe cases; these lesions were hypointense with GE sequences in two of the three cases.

ABSTRACT # 45

TUMORS AFFECTING THE SPINAL CORD OF CATS. K. Marioni-Henry[1], T. J. Van Winkle[2], S. H. Smith[3], C. H. Vite[4]. [1]Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, Knoxville, TN, USA. [2]Laboratory of Pathology and Toxicology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA, USA. [3]Department of Veterinary Pathology, Royal (Dick) School of Veterinary Studies, University of Edinburgh, Easter Bush, Midlothian, UK. [4]Department of Clinical Studies-Philadelphia, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA, USA.

Lymphosarcoma is the most common tumor affecting the spinal cord of cats; other types of spinal tumors are uncommon and are often described in the veterinary literature as single case reports or small case series. We conducted a retrospective study on tumors that affect the spinal cord of cats to determine the prevalence of lymphosarcoma and other types of tumors. Our objective was to relate specific tumors affecting the spinal cord to signalment, history, and clinical findings to improve the chances of an antemortem diagnosis.

Case material was reviewed from the biopsy and necropsy services of the School of Veterinary Medicine at the University of Pennsylvania and the University of Tennessee. We identified 85 cases with a definitive diagnosis that was confirmed by histopathology. In 53 cases a complete post-mortem exam was available. In 32 cases, tissue samples collected during a surgical procedure or at the post-mortem were examined. The cases were divided based on the cell type presumed to originate the tumor.

The lymphosarcoma group was the most numerous with 35 cats (41% of total cases). Osteosarcoma was the second most common tumor affecting the spinal cord with 14 cats. Other cats presented tumors of glial origin (7 cats), meningiomas (6 cats), fibrosarcomas (5 cats), peripheral nerve sheath tumors (4 cats), primitive neuroectodermal tumors (4 cats), histiocytic tumors (3 cats), plasma cell tumors (2 cats), undifferentiated sarcomas (2 cats), metastatic tumors (2 cats), and one chondrosarcoma. Cats with lymphosarcoma had a younger average age at death (5.9 years), other information such as FeLV status (52% of the cats with lymphosarcoma were FeLV positive) and presence of malignant cells outside the nervous system were also useful to obtain an antemortem diagnosis of lymphosarcoma.

ABSTRACT # 46

BRAINSTEM ABSCESS DUE TO VEGETAL FOREIGN BODY: CLINICAL SIGNS, MAGNETIC RESONANCE IMAGING AND HISTOPATHOLOGICAL FINDINGS. I.Mateo[1], V. Lorenzo[1], A. Muñoz[1, 2], M. Pumarola[3]. [1]Resonancia Magnética Veterinaria, Madrid, Spain. [2]Neurorradiología, Hospital 12 de Octubre, Madrid, Spain. [3]Dept. Patología Animal, Universitat Autónoma de Barcelona, Barcelona. Spain.

A 7-month old male Dachshund was referred with a 1-month history of progressive ataxia and depression with further stabilization. Blood test showed mild thrombocytopenia, neutrophilia and increased globulins. Serum antigen for *D.immitis,* antibodies for *E.canis* and *B.burgdorferi,* and PCR for Canine Distemper virus were all negative. The dog was treated with prednisone.

At the time of presentation, no abnormalities were detected on physical examination. Abnormalities on neurological examination included: depressed mental status, ambulatory tetraparesis worse on pelvic limbs, absent hopping reaction on the thoracic limbs, decreased extensor postural thrust, depressed menace reaction on both sides, and bilateral decrease on palpebral reflex.

Neuroanatomical lesion localization was considered in the brainstem.

Under general inhalatory anesthesia magnetic resonance imaging (MRI) study of the brain and cranial cervical spinal cord was performed, using a superconductive magnet operating in 0.5 T. MRI study included T1-weighted spin echo (SE) sequences (500/14; TR/TE), T2-weighted fast spin echo (FSE) sequences (4000/110/16; TR/TE/echo train), and gadolinium-dimeglumide (0.1 mmol/kg IV) enhanced T1-weighted SE sequences. The study revealed the presence of swelling and increased T1 and T2 relaxation rates in the caudal brainstem. An intraaxial round mass localized in the centre of the caudal brainstem. The lesion induced changes related to vasogenic edema, and it enhanced heterogeneously in the periphery on T1-weighted postcontrast images.

Differential diagnosis included neoplasm (mainly diffuse glioma) an abscess. Due to the poor prognosis the animal was euthanized.

On gross examination of the brain no abnormalities were found. Microscopic examination of brain sections stained with hematoxylin and eosin (HE) showed areas of malacia with a severe inflammatory reaction, localized in the medulla oblongata. This inflammation, associated with proliferation of collagen, formed a thick wall surrounding a vegetal material that was acting as a foreign body. Masson's trichromic stain showed a capsule of connective tissue around the vegetal material.

Final diagnosis was caudal brainstem granuloma due to migrating vegetal foreign body.

Only a few cases (1 cat, 3 dogs) of brain neurological lesions due to migrating vegetal foreign bodies have been described in small animals. To our knowledge, this the first case of an intracranial vegetal foreign body granuloma localized in the medulla oblongata in a dog, and also the first case in which MRI images have been described.

ABSTRACT # 47

CLINICAL PHENOTYPE, MAGNETIC RESONANCE IMAGING AND RADIOLOGICAL FINDINGS ASSOCIATED WITH BETA MANNOSIDOSIS IN A CAT. L A Matiasek[1], S R Platt[1], A Sparkes[1], J F McConnell[1], A Blunden[1], K Matiasek[2]. [1]Animal Health Trust, Newmarket, Suffolk, UK, [2]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, Ludwig Maximilians University, Munich, Germany

Beta mannosidosis has been described as a neurodegenerative lysosomal storage disease in goats, cattle and humans as a result of inherited beta mannosidase deficiency. In contrast to the relatively mild course in humans, the disease in animals is more severe involving neurological and skeletal abnormalities. This case report is, to the authors' knowledge, the first description of clinical and imaging findings in confirmed feline beta mannosidosis.

An 8-month old male Persian was presented with subacute onset of tetraparesis. An abnormal pigmented fundus was present on clinical examination. Neurological examination revealed non-ambulatory tetraparesis with absent and reduced postural reactions on the pelvic and thoracic limbs, respectively. Spinal reflexes, cranial nerves and mentation were unremarkable. There was hyperaesthesia on palpation of the neck. MRI of the cervical spine showed lateral stenosis of the spinal canal, most prominent at C2/C3. On gradient echo images the vertebrae and calvarium had a slightly increased signal intensity suggesting bone loss or demineralisation. There was occipital bone dysplasia with overcrowding of the caudal fossa and cerebellar herniation. Throughout the brain there was mild, diffuse grey matter swelling and slight changes in signal intensities of the white matter resulting in decreased grey matter-white matter contrast. Radiographs of the skull, spine and limbs revealed marked generalised osteopenia, most evident in cancellous bone. The main differential diagnosis at this point was a degenerative disorder such as storage disease. Normal parathyroid hormone levels excluded osteopenia due to secondary hyperparathyroidism. Urine, plasma and a skin biopsy for fibroblast cultures were submitted for investigation of intrinsic metabolic diseases. Plasma beta mannosidase

activity was 15% of that found in control cats. Enzyme activity in cultured fibroblasts was also low. The cat was euthanased. Main post mortem findings were osteomalacia and severe white matter degeneration due to compressive myelopathy within the cervical spinal cord. Electron microscopy revealed mild vacuolation of neurons, pericytes and macrophages, in particular in the temporal lobe and throughout the hippocampus. Hypomyelogenesis as described in ruminants was not found.

Beta mannosidosis should be considered as a differential diagnosis in young cats with skeletal and neurological abnormalities. Diagnosis can be based on low enzyme activities in plasma and fibroblasts. Pedigree studies and genetic screening are the next steps for further characterizing this newly identified feline storage disease.

ABSTRACT # 48

PRELIMINARY RESULTS OF A 'FAST' CT PROTOCOL FOR ACUTE THORACOLUMBAR DISC EXTRUSIONS IN DOGS. Susanne Medl[1], Chiara Tartarelli[2], Andrea Margini[2], Gian Luca Rovesti[2], [1]Babenhausen, Germany, [2]Cavriago, Italy.

Non contrast Computed Tomography (CT) is able to detect the presence of mineralized Hansen type I disc herniation. Moreover degenerated disc material mixed with hemorrhage has been reported to be slightly more attenuating than the spinal cord. Patients affected by acute disc herniation often require fast surgical decompression. Prolonged anesthesia time enhances the risk of anesthetic accidents and infection. The purpose of this prospective study was the evaluation of the reliability of a 'fast' CT protocol for the diagnosis of acute disc extrusions. 28 dogs with hyperacute (<1 hour) and acute onset (1 to 24 hours) of neurological signs compatible with thoracolumbar or lumbar disc disease were included. Grading was as follows: pain only (grade 1), ambulatory paraparesis (grade 2), non-ambulatory paraparesis (grade 3), paraplegia with deep pain perception (dpp) (grade 4), paraplegia without dpp (grade 5). Using a third generation CT scanner, a single transverse image (3mm slice thickness) in the middle of each disc space from the tenth thoracic to the first sacral vertebra was acquired (total of 11 slices) with the gantry angled parallel to each intervertebral disc space. If one or more extruded discs were found, continuous transverse images were acquired at 3 mm intervals (slice thickness 3mm) from the centre of the cranial adjacent vertebral body to the centre of the caudal adjacent vertebral body. If CT-imaging was negative or not conclusive, a cisternal CSF tap, myelography and myelo-CT or an MRI of the suspicious area were performed. The onset of clinical signs was hyperacute in 5 patients and acute in 23 patients. The grade was 1 in 2 dogs, 2 in 10, 3 in 8, 4 in 6, 5 in 2. The neuroanatomical localisation was T3-L3 in 25 dogs, and L4-S3 in 3 dogs. The average duration of this CT protocol was16 minutes (8–35 min.). Of the 28 dogs I ncluded in the study 24 were of chondrodystrophic breeds: in 18 of these dogs a hyperdense area representing extruded mineralized disc material was found at one or more (four dogs) disc spaces and confirmed by surgery in 17 dogs. In two dogs the extruded disc material was only mildly hyperdense. Myelo-CT and surgical findings confirmed the diagnosis in both patients. In 3 chondrodystrophic dogs (2 grade 1, 1 grade 2) CT showed only very small hyperdense areas. In one dog (grade 1) CT showed no compression at all. In these dogs MyeloCT or MRI findings were consistent with the CT results. In all nonchondrodystrophic large breed dogs the extruded disc material was isodense or only very mildly hyperdense in relation to the spinal cord on the CT. Myelo-CT confirmed the location for the surgery. From these preliminary results we can conclude, that this protocol can be completed in a reasonable short time , which is desirable in patients, who shall undergo an urgent surgery. It worked well in chondrodystrophic breeds with mineralized disc extrusions, concerning the affected disc spaces as well as the lateralization. In the six dogs, in which isodense or only mildl hyperdense material was compressing the spinal cord, the Myelo-CT always confirmed the suspected disc space. These dis extrusions are more difficult to recognize, however with increasing experience this may change. In none of the dogs a disc extrusion was overlooked.

However, if CT doesn't show a disc extrusion with this protocol a different protocol or imaging technique certainly is required to achieve an exact diagnosis.

ABSTRACT # 49

DETECTION OF ANGIOSTRONGYLUS VASORUM IN THE CSF OF A PUG DOG. A. Negrin[1], G.B. Cherubini[1], E. Steeves[2]. [1]Department of Veterinary Clinical Sciences, The Royal Veterinary College, University of London, UK. [2]Department of Veterinary Pathology, The Royal Veterinary College.

Infestation with Angiostrongylus vasorum, a metastrongylid helminth parasite of domestic dogs and related canids, has a worldwide distribution and within Europe is recognised to be endemic in France, Ireland and Denmark, while it is of increasing importance in UK. The clinical signs associated with angiostrongylosis may be different, however two clinical syndromes are predominant: the respiratory disease, caused by migrating larvae, and diffuse haemorrhages. In literature, presence of CNS signs in dogs affected by Angiostrongylus vasorum have been reported to be secondary to the CNS haemorrhages with diagnostic tests, including MRI and CSF analysis, compatible with haemorrhagic disease. A 11-months-old, male entire Pug dog was referred to The Royal Veterinary College, with a 2-weeks-history of progressive depression, ataxia, loss of balance, lethargy and associated mild respiratory signs. The neurological examination detected depression, intentional head tremor, severe generalised ataxia and body tremor. Cranial nerve examination revealed bilaterally absent of menace response. Haematology showed mild non-regenerative normocytic normocromic anemia and mild thrombocytopenia, while biochemistry, PT, APTT and BMBT were unremarkable. Chest radiographs showed peripheral alveolar pattern, more severe in the dorsal-caudal region of the caudal lobe of both lungs, consistent with lungworm infestation. Angiostrongylus vasorum infestation was confirmed by parasitology on faecal and endotracheal flush samples. MRI investigation was consistent with severe meningitis, mainly around the cerebellum, characterised by multifocal post gadolinium meningeal enhancement in T1WI, and meningeal and submeningeal hyperintensity in FLAIR and in T2WI. No neuroparenchyma invasion or haemorrhages were observed on T2starWI. CSF analysis detected increased protein (0.89 g/l) and WBC count (8 cells/mm^3) characterised by mixed pleocytosis with presence of reactive macrophages and eosinophils, and parasitic larval lung-worms. Fenbendazole at 50 mg/kg/SID and Prednisolone at 1 mg/kg/SID was started. Mild ataxia and mild head tremor were still present when discharged, 6 days after the presentation. The neurological examination after 50 days from the presentation detected just mild stiffness on his right hind limb. CSF analysis can be useful in CNS infectious diseases, detecting different types of inflammation, but rarely CSF has been reported to detect the aetiology, especially parasites. This is the first case of detection of Angiostrongylus vasorum in CSF. Moreover, CNS infestation of parasites, generally, is consistent with a poor prognosis; in this case, the dog dramatically improved to normality; this could be explained by absence of neuroparenchyma invasion. The present study presents a dog with CNS signs secondary to A. vasorum worm migration, diagnosed by detection of worm larvae in CSF, and the results of the MRI of his brain, which were compatible with meningitis, rather than haemorrhagic process.

ABSTRACT # 50

FIVE NEW CASES OF DYSAUTONOMIA IN ENGLAND: AN EMERGING DISORDER? S.J.M. Niessen[1], J. Eastwood[1], J.B.A. Smyth[2], G.B. Cherubini[1]. [1]Department of Veterinary Clinical Sciences and [2]Department of Pathology and Infectious Diseases, The Royal Veterinary College, University of London, UK.

Canine dysautonomia (CD) is a degenerative polineuropathy characterized by neuronal degeneration within the autonomic, somatic, central peripheral and / or enteric system causing multi systemic effects. CD seems to be rare in Europe compared to the high numbers reported in the USA. The only English case of CD was reported 23 years ago, although 2 cases were reported in Scotland 4 years ago.

Five dogs (2 Labradors, 1 Weimaraner, 1 Rottweiler, 1 Beagle) were referred over a one-and-a-half year period (June 2004–October 2005, none at the end of winter or beginning of spring) to the Royal Veterinary College, University of London.

CD was definitively diagnosed in four dogs, aged eight, ten, fifteen months and eight years, via histopathology, and was strongly suspected in one dog, aged fifteen months, on the basis of clinical physical and neurological findings, diagnostic imaging features and autonomic function testing.

Four dogs originated from urban areas. No direct contact with horses, farm animals, cadavers or toxins was reported. Acute-onset chronic progressive vomiting, diarrhoea, depression and inappetance were the most common presenting clinical signs.

Lethargy and depression, tetraparesis, reduced or absent anal tone, dysuria, bradycardia, absence of pupillary light reflexes with intact vision, mydriasis, decreased corneal sensitivity and third eyelid protrusion were amongst the most frequent neurological findings.

All dogs were eventually euthanased due to lack of response to treatment and the poor prognosis. Histopathology displayed marked chromatolysis of ganglion cell bodies in the celiac, mesenteric and trigeminal ganglia, bladder and brainstem.

The previously proposed scoring system for canine dysautonomia was further validated using these cases.

This report emphasises that, dysautonomia should be suspected when presented with a dog with compatible clinical signs and physical examination findings in England. Autonomic function testing and imaging should be performed to substantiate the suspicion. Unfortunately, post mortem remains the only method of confirming the disease.

ABSTRACT # 51

SWAINSONINE INTOXICATION IN A HORSE: FIRST CASE REPORTED IN EUROPE. H Nollet[1], K. Panter[2], K. Vanschandevijl[1], L. Van Ham[1], P. Deprez[1]. [1]Faculty of Veterinary Medicine, UGent, Merelbeke, Belgium [2]ARS Poisonous Plant Research Laboratory, Logan, Utah, USA

A 20-years-old male horse was referred with behavioural changes (cycles of excitement-depression), slight hypermetric gait, trembling and fright reactions when touched and mild intention tremor, since a few hours. Blood analysis upon arrival showed metabolic alkalosis, very mild elevation of blood urea nitrogen (BUN), creatine phosphokinase and very mild decrease of potassium (P). Creatinine was at the upper limit and urine analysis showed an elevated γ–GT level. During the following hours the horse became very excited and showed generalized trembling. Also the ataxia and hypermetria became more severe. Subsequent blood analyses showed a steady decrease of P and increase of BUN. A treatment with polyionic isotonic perfusions with extra added P was started, but 6 hours later the horse still had a small bladder and had not urinated. Dopamine was administered to increase renal blood flow. To calm the horse, diazepam was given. After a few days the horse seemed to be less stressed and showed only short episodes of excitement and longer periods of depression. Also the oliguria was converted and the administration of perfusions and diazepam was discontinued after 4 days. Five days later a small relapse of P decrease has to be corrected. The urinary γ–GT level had decreased but not normalised. During the following days P levels remained stable and clinical signs stabilized to a level of mild ataxia. EMG and TMS did not reveal any abnormalities. The horse returned home where clinical signs progressively disappeared and now the horse is ridden again. Four months after admission urinary γ–GT had normalised.

The neurological symptoms together with the renal tubular lesion can be induced by the toxin swainsonine. The toxin acts by inhibiting the lysosomal enzyme alpha-mannosidase resulting in a lysosomal storage disease, similar to genetic mannosidosis, described in humans, Angus cattle and cats. The toxin is present in locoweeds, which are also endemic in Belgium. The horse was fed ensilated grass-hay and spent a few hours per day on pasture. The serum sample of the horse taken on admission (ca. 12 h after the onset of the signs) was found positive for swainsonine (154 ng/ml) by a competitive binding assay with Jack Bean alpha mannosidase. Three other samples of the same horse (a sample taken one day later, one week later and 4 months later respectively) were all below the detectable limits (less than 30 ng/ml). This can be explained by the short half-life of swainsonine in the serum (16–20 hours), which means that the toxin has to be detected in a blood sample taken within 2 days of eating locoweed. To our knowledge this is the first report of a swainsonine intoxication in a European horse. No exact explanation for the low incidence can be given but maybe due to the change in the environmental management, whereby less herbicides are used, an increase in described cases of poisoned horses can be expected.

ABSTRACT # 52

IDIOPATHIC EPILEPSY: THERAPY, MONITORING TIME, SURVIVAL TIME, CAUSE OF DEATH: A RETROSPECTIVE STUDY OF 100 DOGS. A. Pakozdy, M. Leschnik, J.G. Thalhammer. Clinic of Internal Medicine and Infectious Diseases, University of Veterinary Medicine, Vienna, Austria

The goal of this study was to investigate retrospectively therapy, outcome, monitoring time and the cause of death or reason for euthanasia in dogs with idiopathic epilepsy (IE). Idiopathic epilepsy was considered in dogs with recurrent seizures when the neurological status was within normal limits for at least 1 year after seizure occurred or routine serum biochemical, haematological testing, CSF analysis, computed tomography (CT) or magnetic resonance imaging (MRI) did not reveal any underlying cause. In the study 100 dogs of 28 different breeds with idiopathic epilepsy were included. Fifty-four dogs were considered belonging to large breeds (>=25kgs) and 46 dogs to small breeds (<25 kgs). The mean bodyweight was 24,57 kg (range 3 to 74). Thirteen patients were not treated because of low frequency of seizures. Seventy dogs were treated with phenobarbital alone of those 49 dogs were well-controlled. Seventeen dogs were treated with phenobarbital and potassium bromide as add-on therapy. The median monitoring time from the first seizure to the end of the study was 1130 days (range 60 to 3600 days). Eighty-one dogs were still alive at the time of completing the study. Three dogs were euthanised or died due to complication of status epilepticus, three were euthanised because of refractory epilepsy and severe adverse effects of treatment. Further two dogs developed hepatic failure due to hepatic cirrhosis. These two patients were treated with phenobarbital and potassium bromide previously because of severe seizures. Both dogs survived 1800 days after the first seizure. One of these two patients received primidon therapy additionally. Two patients died from complication of histopathologically confirmed acute haemorrhagic necrotizing pancreatitis. They were treated with phenobarbital for 700 and 1500 days. Further causes of death or reasons for euthanasia were not connected with seizures or anticonvulsant therapy in 9 dogs. Out of 100 dogs with idiopathic epilepsy 71% can be categorized as having a good quality of life, because either no therapy was necessary or seizures could be well-controlled by therapy. Hepatotoxicity of phenobarbital has been already reported, this complication was 2% in our investigation. The frequency of fatal pancreatitis in our clinic was with 2% higher as the previously reported 0.3% in dogs with long term phenobarbital treatment. We concluded that a high percentage (71%) of dogs with IE has a good quality of life. Fatal consequences of IE were observed in 10 % of cases. Median survival time was 931 days (60 to 1800 days) in this group.

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ABSTRACT # 53

SPINAL SYNOVIAL CYST ASSOCIATED WITH THE ANNULUS FIBROSUS IN A ROTTWEILER. V Penning[1], L Benigni[1], E Steeves[2], R Cappello[1]. [1]Queen Mother Hospital for Animals, Royal Veterinary College, University of London. [2]Department of Veterinary Pathology, Royal Veterinary College

A six year old male neutered Rottweiler had chronic episodic signs of thoracolumbar pain and inability to stand that did not improve after rest and non-steroidal anti-inflammatory medication. The neurological examination revealed a mild pelvic limb ataxia and pain on palpation of the thoracolumbar region. All reflexes were normal. The lesion was localized to T3-L3 spinal segments. Magnetic resonance imaging (Gyroscan NT, Philips Medical Systems,) revealed a focal oblong well marginated extradural mass between the spinal cord and the T13/L1 intervertebral disc on the dorsal aspect of the intervertebral disc. The lesion had a low signal intensity rim that enhanced slightly after gadolinium administration (Dotarem, Guerbet, Milton Keynes MK5 6LB, UK)and contents with similar signal intensity to cerebrospinal fluid. On T2w images and FLAIR there was a region of hyperintensity within the cord from T11–T13. The mass in these sequence had a signal intensity equal to CSF. These images were suggestive of a spinal synovial cystic lesion causing cord oedema as a result of compression. CSF from the cerebromedullary cistern was consistent with cytoalbinodissociation with an elevated protein of 0.77g/l. The lesion was removed surgically via a right sided hemilaminectomy over the T13-L1 intervertebral disc space by severing its attachments to the dorsal aspect of the annulus fibrosus, the cyst burst on excision and the wall was submitted for histopathology. The histologic diagnosis was synovial cyst. The dog was neurologically normal and was discharged 6 days after surgery. Ten months after surgery the dog was suffering from an unrelated medical condition. All previously reported spinal synovial cysts in dogs are associated with the true intervertebral joints and impinge on the dorsal or lateral aspects of the cord, hence the lesion in this dog had an atypical location. The pathogenesis of the formation of synovial cysts is unknown in this case the cyst could have formed due to the presence of ectopic tissue or metaplasia of the annulus fibrosus.

ABSTRACT # 54

MAGNETIC RESONANCE IMAGING EVALUATION OF SPINAL CORD SWELLING DUE TO HANSEN TYPE 1 INTERVERTEBRAL DISC DISEASE AND ITS ASSOCIATION WITH CLINICAL DATA IN 67 DOGS. V. Penning[1], S.R.Platt[2], V. Adams[3], [1]The Queen Mother Hospital for Animals, The Royal Veterinary College, University of London, [2]Centre for Small Animal Studies, Animal Health Trust, Newmarket, Suffolk, England; [3]Centre for Preventive Medicine, Animal Health Trust, Newmarket, Suffolk, England.

Hansen type I intervertebral disc disease results in compression and contusion of the cord and initiates secondary spinal cord injury mechanisms after extrusion of the nucleus pulposus through the annulus fibrous into the

spinal canal. Magnetic resonance (MR) can be used to evaluate the spinal cord parenchyma. The ratio of length of spinal cord swelling to L2 on myelography was shown to affect the prognosis in dogs with no deep pain with the ratios over 5 leading to a worse prognosis. The length over which hyperintensity was visualised on MRI in the spinal cord correlated with a worse prognosis. The aim of this study was to determine if there is an association between the ratio of length of spinal cord swelling on sagittal MR images compared to the length of the second lumbar vertebra following T3-L3 Hansen type I intervertebral disc disease and clinical data. Dogs included were surgically confirmed to have type I intervertebral disc disease localised to the T3-L3 spinal cord segments. All dogs underwent a spinal MR examination prior to surgery. The rate of onset and duration of disease were recorded, in addition to the pre- and 1-month post-surgical neurological grades. Spinal cord swelling was inferred due to the lack of signal from the epidural fat over the region of disc extrusion. The mid line sagittal T2 weighted image was used and the minimum length over which the fat signal was missing was measured. The length of the second lumbar vertebra (L2) was also measured to allow estimation of the ratio of spinal cord swelling (sc) to length of L2 to account for variation in size of dog. A final neurological score was recorded 34–180 days post surgery for each dog using the same grading system or whether the dogs died due to the disease. The association between potential explanatory variables and SC swelling was analysed using non parametric tests for ordered variables (Kruskal-Wallis ANOVA and post hoc pairwise Wilcoxon rank sum tests or Goodman and Kruskal's gamma coefficient). Sixty-seven dogs with a mean age of 6yrs (sd=2.4, range 1.5–11.3) were included in the study and had a mean sc swelling: L2 ratio of 0.74 (sd = 0.52; range 0.10–0.86). There was an association between sc swelling and presenting neurological grade (P=0.03), change in neurological grade (P=0.048) and final outcome (P=0.03). There was no association between sc swelling and spinal cord compression (P=0.1), speed of onset (P=0.2 or duration of clinical signs (P=0.8). The results from this study indicate that spinal cord swelling could be used as a prognostic indicator with a greater amount of spinal cord swelling being predictive of a worse prognosis.

ABSTRACT # 55

ADVANCED IMAGING FINDINGS IN TWO DOGS WITH CEREBRAL TOXOPLASMOSIS. Z.S. Polizopoulou[1], V. Souftas[2], A.F. Koutinas[1], M.N. Patsikas[1], E. Kaldrymidou[1], N. Soubasis[1]. [1]Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece. [2]Medical School, Democritus University of Thrace, Greece.

The CT or MRI imaging characteristics of two elderly dogs with cerebral toxoplasmosis, confirmed immunohistologically, are described in this report. The first case was a 7 year-old intact female mongrel, admitted with a 2-month history of dementia and unprovoked aggression and compulsive walking and circling. The other dog was an 12 year-old intact female Miniature Poodle-cross with a 1-month history of dementia and generalized motor seizures. Both dogs were current on vaccinations and kept strictly indoors. Initial laboratory work-up revealed mild neutrophilia and increased alkaline phosphatase activity only in the first dog; the latter abnormality was probably due to prior therapy with glucocorticosteroids and phenobarbital by the referring veterinarian. In the first dog, CT demonstrated the presence of multiple focal lesions of variable size, extending over the supratentorial area and cerebellar hemispheres and diffusely enhancing after the administration of ioparmirol. In the second case, MRI scanning showed that the pattern was also multifocal, involving the cerebral and cerebellar hemispheres and displaying a ring-like configuration after the enhancement with gadodiamide. The CT or MRI appearance in both animals would bear resemblance to that of metastatic brain disease or granulomatous encephalitis.

Both dogs were euthanized because of the rapid and relentless progression of the disease and permission for post mortem examination was granted. Brain histopathology showed a non-suppurative meningoencephalitis dominated by lymphoplasmacytic perivascular infiltrations, focal necrosis and microgliosis, along with the presence of protozoal tachyzoites that were proved to belong to *Toxoplasma gondii* by ensuing immunocytochemistry.

As in human cerebral toxoplasmosis, CT and MRI findings are variable and depend on the stage and distribution of the lesions within the brain and the degree of host response to infection; histopathology of surgical or postmortem biopsies remains the only means for a definite diagnosis.

ABSTRACT # 56

CANINE COGNITIVE DYSFUNCTION AND ALZHEIMER'S DISEASE: A COMMON NEURODEGENERATIVE PROCESS. M. Pugliese[1], J. Mascort[2], I. Ferrer[3] and Nicole Mahy[1]. [1]Unitat de Bioquímica, Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Facultat de Medicina, Universitat de Barcelona, Barcelona, Spain. [2]Servei de Neurologia, Ars Veterinaria, Barcelona, Spain. [3]Institut de Neuropatología, Servei de Anatomia Patològica, IDIBELL-Hospital Universitari de Bellvitge, Hospitalet de Llobregat, Spain.

The principal hallmarks of Alzheimer's disease (AD) are the formation and deposition of amyloid-beta (Ab) protein in the form of senile plaques and cerebral amyloid angiopathy and the hyperphosphorylation of tau. Combined clinical and neuropathological studies in AD have shown a strong relationship between cognitive impairment and load of neuropathological hallmarks.

The aim of the present study was to assess the no-housed dog as a promising model for examining behavioral, cellular and molecular processes involved in early phases of human brain aging and AD. Twenty five dogs, eight females and seventeen males of different breeds and weight, and ranging from 1 to 16 years, were examined at the veterinary hospital *Ars Veterinaria*, Barcelona, Spain and used for this study. To characterized the global and progressive deterioration of memory, cognition and personality of all 25 dogs, a new cognitive test was used in collaboration with the pet owners. Analysis of cerebrospinal fluid (CSF) parameters related to brain energy metabolism and post-mortem histological and immunohistochemical studies of sections of prefrontal cortex corresponding to area 8a on the proreal gyrus were performed in all dogs. Based on the results of the cognitive test, dogs were categorized as young control (YC), i.e. with no signs of behavioral disorder, light cognitive deficits (LCD), and severe cognitive deficits (SCD). Starting at the age of 8 years, and increasing with age and with cognitive deficit severity, Ab immunohistochemistry revealed the presence of delicate and more compact diffuse Ab deposits throughout all cortical layers of the cerebral cortex in a characteristic four-stage distribution. Tau hyperphosphorylation, as revealed with phospho-specific antibodies to tauThr181 and tauSer396, increased with age and with the degree of cognitive dysfunction in individual neurons. Moreover, the subcellular pattern shifted from perinuclear localization to granular cytoplasmic and nuclear distribution with age. In all situation, the distribution of phospho-tau immunoreactivity lacked any spatial relation with Ab deposition. CSF analysis determined the correlation between energy consumption, ion balance, Ab deposition, and the cognitive deficit.

Finally, our data demonstrate that, despite their variety in breeds and life conditions, dogs can serve to study the cascade of first events in human brain aging and early stages of AD, ruling out the need of a specific strain to be housed during years in controlled conditions.

ABSTRACT # 57

TWO EXAMINATIONS, TWO RESULTS: ELECTROPHYSIOLOGIC AND MORPHOMETRIC ASSESSMENTS AFTER DOUBLE SCIATIC NERVE TRANSSECTION AND REPAIR IN THE RAT. A Rupp1, U Dornseifer2, A Fischer[3], P Gais[4], K Rodenacker[5], U Jütting[5], A Fichter[6], A Wilson[6], S Leichtle[6], N Papadopulos[6], K Matiasek1* [1]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, LMU Munich, Germany, [2]Department for Plastic, Reconstructive & Hand Surgery, Hospital Bogenhausen, Munich, Germany, [3]Clinic for Veterinary Internal Medicine, Ludwig-Maximilians-Universität Munich, Germany, [4]Institute of Pathology, GSF Forschungszentrum, Neuherberg, Germany, [5]Institute of Biometrics und Biomathematics, GSF Forschungszentrum, Neuherberg, Germany, [6]Department of Plastic & Reconstructive Surgery, Klinikum rechts der Isar, Munich, Germany

Electrophysiologic tests are occasionally applied in experimental nerve regeneration studies in addition to other methods of evaluation in order to assess the regenerating nerve and corresponding muscles individually.In the following study 20 male Lewis rats were submitted to excision of a 14 mm segment from the sciatic nerve and its subsequent repair through bio-compatible guidance interponation, with the distal end of the defect located 4 mm proximal to where the tibial nerve enters the gastrocnemius muscle. The extent of regeneration was assessed eight weeks later by means of electrophysiologic and morphometric studies and compared to the contralateral healthy side. Morphometric assessments of transverse sections of the complete interponate revealed abundant regeneration (>10,000 regenerating myelinated fibres per section) in seven rats, moderate to low regeneration (< 8,000 myelinated fibres per section) in nine rats and no regeneration at all (0 fibres per section) in four rats. In all animals compound muscle action potentials (CMAPs) could be recorded in the gastrocnemius muscle after stimulation of the sciatic nerve proximal and distal to the operation site. Motor nerve conduction velocities (NCVs) ranged between 26 m/s and 46 m/s (mean: 34.0 ± 5.5 m/s). However, no correlation could be found between the number of myelinated fibres and the NCVs in the individual animals. Myelinated fibre numbers (mean 7944 ± 772 per section) and NCV (mean 55.6 ± 6.4 m/s) of the contralateral side corresponded well

to values reported in literature. It has been estimated that in primates the presence of 4,000 to 5,000 myelinated axons greater than 5 μm is necessary for recording a nerve action potential. No information is available, however, about eliciting CMAPs by direct stimulation of the corresponding nerve. In rats exhibiting no or only very little regeneration the source of muscle stimulation can only be surmised and requires further investigation. Either aberrant innervation of the gastrocnemius muscle or technical problems pose the most probable options since sprouting of the proximal stump had been prevented by drawing each stump deeply (3mm) into the interponate tube before securing it. The discrepancy between regained impulse conduction and nerve fibre counts does however underline the need for functional tests in sciatic nerve regeneration studies to be supplemented by morphometric assessments in order to avoid overestimating the degree of anatomical restoration.

ABSTRACT # 58

MR IMAGING IN DOGS WITH AN IMPLANTED MICROCHIP. M. Saito, Y. Une, S. Ono, H. Kayanuma, M. Honnami, T. Kageyama, and M. Muto. Azabu University, Sagamihara, Kanagawa, Japan.

A microchip, implantable electronic identification devise for animals, contains metal materials. MR imaging in the presence of a metallic implant raises concern for the potential complications of implant migration, heat injury, and MR image distortion caused by the metallic implant. The purpose of this study was to determine the effect of implanted microchip on MR imaging and patient's tissue due to the interaction of microchip with strong radiofrequency magnetic field produced by MR scanner. We also examined effectiveness of microchip after exposing to a MR environment.

Five healthy adult beagle dogs were implanted with microchip (LIFECHIP®, Digital Angel Corp.) in the subcutaneous space, just dorsal to the cervicothorax junction with the microchip aligned on the axial plane. One month after the implantation, three dogs underwent 1.5T MR scanning (Toshiba ViSART) (*MR Group*). The cervicothorax area were imaged for 90 minutes using a surface coil. T1WT, T2WT, T2*, FLAR, and T1WT contrast study were performed in all planes (i.e. transverse, sagittal and dorsal). After imaging was completed the microchips were removed surgically together with adjacent skin and subcutaneous tissue under the same general anesthesia. In the remaining two dogs, which were not scanned, the microchips and the adjacent skin and soft tissue were removed surgically in the same fashion (*Control Group*). The microchip was removed from the harvested soft tissue specimen, and the specimen was submitted for histopathological examination. The removed microchip was scanned to determine its record function. The MR images were analyzed to calculate the size of the artifact using commercially available software (Photoshop®).

There was a significant image distortion around where the microchip was located on all MR imagings. The nature of the distortion was consistent with metallic artifact. The affected area was most extensive in T2* on all planes. The percentage of the distorted area to the cervical area was 67% in maximum (T2*, horizontal) and 22% in minimum (FLAIR, transverse) of all images obtained and the artifacts rendered the affected area undiagnostic. Histopathologically, there was no considerable difference in the tissue of dogs between MR and Control groups. All microchips were read correctly with the reader.

The results of this study suggest that the effect of the implanted microchip on MR imaging may be substantial depending on the size of the animals and region of interest, although the dog with a implanted microchip may be scanned safely on 1.5T MRI.

ABSTRACT # 59

CANINE AND FELINE MUSCLE CELL CULTURE-A NEW APPROACH TO ELUCIDATE FUNCTIONAL NEUROMUSCULAR DISEASES IN DOMESTIC ANIMALS. HC Schenk[1,3], K Krampfl[2,3], A Tipold[1,3]. Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany[1], Department of Neurology, Division Molecular Neurophysiology, University of Medicine, Hannover, Germany[2], Centre of Systems Neuroscience[3], Hannover, Germany

In domestic animals several functional neuromuscular disorders are described, which result in muscular weakness (Myasthenia gravis) as well as in muscular stiffness and spasticity (Myokymia of Jack Russell Terrier, Myotonia of several breeds in cats and dogs). In some of these diseases the underlying pathomechanisms are not known. Functional diseases do not cause pathomorphological changes and histopathological examination remains normal.

Function disorders of the neuromuscular system can be studied by electrophysiological tools like the patch clamp technique. To provide enough tissue material for this in vitro method we have established a cell culture method (harvesting, proliferation, differentiation, storage and re-cultivation of muscle cells) for biopsies of skeletal muscles of healthy dogs and cats. The biopsy (size 1 cm3) from M. longissimus dorsi or M. quadriceps is taken during routine surgical procedures. The piece of muscle designated for the cell culture is digested with trypsin after mechanical dissociation and centrifuged several times to harvest satellite cells from the supernatant. The harvested satellite cells are resuspended in proliferation medium (basic Fibroblast Growth Factor, Epidermal Growth Factor, Fetuin, Insulin, Dexamethason, Fetal Calf Serum) to seed them in a cell culture flask. The cultures are incubated at 37°C in a 5% CO_2-enriched, humid atmosphere. The medium is changed every other day. Until the cells have reached a confluence up to 80% they are kept under these conditions (around 10 to 20 days). To characterize the cultured cells, we used immune-fluorescence staining for the muscle specific intermediate filament desmin. Part of the cells are frozen and stored at - 80°C and the rest is exposed to differentiation medium (Insulin, Horse Serum) for the induction myotube-forming.

Under these specific conditions we could show that myotubes with cross-striation and spontaneous contractions are developing. In addition, the cultured cells displayed electrical activity measured by patch clamp experiments, indicating the expression of functional Acetylcholine receptors on the cell membranes of the myotubes. The possibility of cryopreservation and the successful re-cultivation of the proliferating satellite cells will allow us to establish a muscle tissue bank of biopsies from animals suffering from various neuromuscular diseases. The described cell culture system with the subsequent patch clamp studies could be a new approach to elucidate the functional pathophysiology of neuromuscular diseases in animals and will help to find treatment strategies in vitro.

ABSTRACT # 60

NEUROANATOMY OF FORMALIN-FIXED NEONATAL CALF BRAINS AS REVEALED BY HIGH-RESOLUTION MAGNETIC-RESONANCE-IMAGING. Schmidt, Martin Jürgen[1]; Pilatus, Ulrich[2]; Wigger, Antje[1]; Oelschläger, Helmut[3]; Kramer, Martin[1]. [1]Small Animal Clinic - Surgery, Justus Liebig-University, Giessen, Germany. [2]Brain-Imaging-Center, Neuroradiology, Johann Wolfgang Goethe-University, Frankfurt a. M., Germany. [3]Institute of Anatomy, Johann Wolfgang Goethe-University, Frankfurt a. M., Germany.

Magnetic resonance imaging (MRI) has become a standard diagnostic method in veterinary neurology and has even raised interest for neurobiological research groups, since brain tissue can be repeatedly analyzed in situ without artifacts due to sectioning of the specimen. Although MRI has been widely used to visualize brain pathologies in veterinary neurology, there is no study that could provide more detailed Magnetic-Resonance (MR)-images of the physiological brain structure to date. The development of high resolution digital atlases or maps of the different mammalian species can be used for comparative studies to investigate their evolutionary relation conditions with minimum time, effort and cost. The heads of two neonatal Holstein-Frisian calves were obtained at the Clinic for Obstetrics, Gynecology and Andrology of the Justus-Liebig University of Giessen. After initial flushing of the dissected heads with 0.9 % saline via the internal carotid artery, the samples were perfused with 4% formaldehyde in phosphate buffer and post fixed in this solution. Imaging was performed after 6 month on a Siemens 1.5 tesla scanner. Optimum contrast was achieved using a 3D-sequence with a flip angle of 30°, TR of 20ms and TE of 6.8 ms. To achieve a sufficient signal-to-noise ratio (SNR) 32 averages were accumulated overnight (approximately 8 h) obtaining slices of 2mm thickness with 256 × 256 mm field of view at 512 × 512 matrix size. After acquisition each dimension was additionally extrapolated by a factor 2 resulting in an image size of 1024x1024 voxels with 0,25 mm in plane resolution and 1mm slice distance. This study presents the first MR-images of the bovine brain in high image quality and resolution. Since contrast is not limited to white and gray matter structures the visualisation of even delicate substructures within these main contrasting tissues is possible in situ. Mesencephalic nuclei and fiber tracts can be clearly identified as well as the fine layers of the optic tectum or the olfactory bulbs. The high resolution images allow the determination of brain features from various planes, preserving the gross morphological and internal structures of the brain. There are no artifacts due to exenteration, sectioning or staining of the specimen.

Studies like the present one will help to establish a reference base of animal brain MR-topography to scan selected parts of the organ during a clinical or experimental approach. However, although the anatomic structures can be clearly visualized, the signal intensities of formalin-fixed specimen differ from in-vivo imaging. Hyperintensities are found in the thalamic nuclei, the optic tract and the pituitary gland. No explanations have been found for this increase in signal intensity so far. Further studies of

the magnetic-resonance signal changes during the fixation process with formalin will help to explain this phenomenon.

ABSTRACT # 61

NECROSIS OF THE HIPPOCAMPUS AND PIRIFORM LOBE IN CATS - MRI AND HISTOPATHOLOGICAL FINDINGS. O Schmied, U Michal, F Ehrensberger, D Weishaupt and F Steffen. Vetsuisse-Faculty University of Zurich, Switzerland

Necrosis of the hippocampus and piriform lobe is an important cause for feline seizures in Europe. This disease of unknown etiology was first described in a group of 38 cats with either generalised or complex partial seizures of acute onset and rapid progression of neurological and behavioural signs (Fatzer et al, 2000). Neuronal necrosis restricted to the hippocampus and occasionally the piriform lobe was found histopathologically. Ante-mortem diagnosis was not possible in these cases, as results of CSF and CT are usually normal (Gandini et al. 2004). It is the purpose of this poster to describe the clinical, neuropathological and magnetic resonance findings in cats with necrosis of the hippocampus and piriform lobe.

Four cats with epileptic seizures and interictal abnormal behaviour were investigated. All of them had a history of an acute onset of generalised or complex-partial seizures as well as behavioural changes including excessive aggression, salivation, polyphagia and disorientation. Results of CBC, biochemical analysis, urinalysis and CSF examination were normal in all cats. High-field MRI scans (1.5T) were performed in all 4 cases using a human extremity coil (Signa Horizon, GE Medical systems). Characteristic abnormalities were detected in all cases. T2-weighted sequences revealed a hyperintense signal involving the entire area of the hippocampus (3 cats) and parts of the piriforme lobe (1 cat). In T1-weighted images of the same region, a hypointense signal was detected. In 3 cats, T1-weighted postcontrast (Magnevist, Schering AG) was performed: mild to intense enhancement of the affected regions was observed. In 2 cats, T2-weighted FLAIR images provoked a hyperintense signal in the entire hippocampus and focal hyperintensity in the lobus piriformis (1 cat). Based upon the MRI findings, a tentative diagnosis of necrosis of the hippocampus was made. Two cats were euthanized and necropsied within a week because of absent response to anticonvulsive treatment (Phenobarbital and Diazepam) and progressive worsening of their neurological and behavioural status. The remaining 2 cats lived for 3 and 4 months respectively and then were euthanized because of persisting behavioural and neurological signs. Only one of these cats was available for histopathological examination. The histological findings correlated well with the results of the MRI abnormalities in all cases. Pathologic changes were restricted to the hippocampus and one cat also had abnormalities in parts of the capsula externa and gyrus parahippocampalis. The main findings included a severe, diffuse, bilateral necrosis and malazia of the hippocampus and neuronal degeneration. Other parts of the brain were not affected.

In conclusion, MRI seems to be a valuable tool for antemortem diagnosis of cats with necrosis of the hippocampus. Larger series of affected cats have to be examined to allow definite statements about the diagnostic sensitivity of MRI, especially early in the course of the disease.

ABSTRACT # 62

CLINICAL PRESENTATION AND OUTCOME OF CATS WITH SEIZURE DISORDERS. S Schriefl[1], T Steinberg[1], S Walter[1], K Matiasek, A Fischer[1]. [1]Section of Neurology, Department of Small Animal MedicineC [2]Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, Ludwig-Maximilians-University, Munich, Germany

Epileptic seizures can occur as reactive seizures due to metabolic/toxic disorders, symptomatic seizures because of structural brain disease or idiopathic epilepsy. Incidence and prognosis of idiopathic epilepsy in cats is yet not defined.

The medical records of cats (n=164) presented because of epileptic seizures (2000–2004) were evaluated for signalement, age at first seizure, seizure type, seizure frequency, and results of diagnostic investigations. Outcome was assessed by phone contact to the owners. Seizures were classified as either reactive, symptomatic, or idiopathic. A presumptive clinical diagnosis of idiopathic epilepsy was made by a history of recurrent seizures with normal findings on interictal neurological examination, laboratory evaluation, CSF analysis (n=13), and brain imaging (n=11). Survival times were graphically displayed as Kaplan-Meier curves and differences between groups were assessed by log-rank test.

An underlying disease was identified in 91 cats: 23 reactive (16 metabolic, 4 toxic, 3 cardiac), 45 symptomatic seizures (20 inflammatory, 17 neoplastic, 3 traumatic, 3 vascular, 2 hippocampal and pyriform lobe necrosis) and 23 cats with idiopathic epilepsy (25.3%). There was no difference in seizure type between groups. Cats with idiopathic epilepsy lived longer than cats with either reactive ($p=0.003$) or symptomatic seizures ($p<0.001$), and cats with reactive seizures lived longer than cats with structural brain disease ($p=0.018$). The 1-year probability to survive was 0.8 in cats with idiopathic epilepsy vs. 0.4 and 0.18 in cats with reactive and symptomatic seizures, respectively.

The present study demonstrates that a good prognosis should be given to cats with idiopathic epilepsy.

ABSTRACT # 63

HISTOPATHOLOGIC CHARACTERIZATION OF DIABETIC NEUROPATHY IN A TRANSGENIC MICE RIP/IFNβ. Beatriz García[1], Dolors Fondevila[1,2], Anna Serafín[1,2], Fatima Bosch[1] and Martí Pumarola[1,2]. [1]Department of Animal Medicine and Surgery. [2]CBATEG. Universitat Autònoma de Barcelona. Bellaterra (Barcelona), Spain

Peripheral neuropathy is one of the most common complications of type I Diabetes Mellitus. Loss of myelinated nerve fibres is the most distinct morphological abnormality found in human diabetic nerves, affecting motor, sensory, or autonomic, or a combination of any of these fibres.

The aim of the present study was to assess the morphology of the peripheral nervous system in diabetic transgenic RIP/IFNβ mice, in order to determine the potential usefulness of this mouse model for the study of diabetic neuropathy. After 12 weeks of streptotozin diabetes induction, physical examination, biochemical, histological and immunohistochemical studies of the peripheral nerves were performed in diabetic and non-diabetic animals.

Diabetic animals demonstrated characteristic hyperglycemic features, such as polydypsia, polyuria, and polyphagia. Blood glucose measurements were performed every two weeks. All animals were euthanized after four month oh diabetes induction. Samples of all body tissues and organs, including central and peripheral nervous system, were taken and processed for histological, biochemical and immunohistological studies. Histological examination of foodpads revealed significantly reduced numbers of epidermal fibers, whereas no differences were observed in sweat gland-associated autonomic axons. A decreased density and number of myelinated fibers in tibial fascicles were observed in the diabetic state.

Our findings indicate that immunohistochemical analysis of the peripheral nervous system in RIP/IFNβ-diabetic mice have potential to serve as a model system for investigations of functional, biochemical and morphological changes in a mouse model of human diabetic neuropathy.

ABSTRACT # 64

NEUROLOGICAL AND RADIOLOGICAL EVALUATION OF THE LUMBOSACRAL JUNCTION IN 33 WORKING GERMAN SHEPHERD DOGS: A PROSPECTIVE STUDY OVER 3 YEARS. F Steffen, K Hunold, G Scharf, M Roos and M Flueckiger, Vetsuisse Faculty, University of Zurich, Switzerland

Degenerative lumbosacral stenosis (DLS) is widespread among working German Shepherd dogs (GSD) and an important cause for premature drop out and restricted duty. While the prevalence of the disease has been reported based upon necropsy data, prevalence and natural history of DLS based on clinical and radiographic signs are not available so far. Radiographic changes have been reported not to equate with clinical signs of DLS . However, other trends may become apparent on a prospective basis allowing to predict the disease. The purpose of this study was to evaluate the correlation between clinical and radiological findings at the lumbosacral junction after a 3 years interval and to assess progression of clinical and radiological signs of DLS.Thirtythree working German Shepherd Dogs (GSD) operating at a Swiss Police unit were available for a prospective evaluation of their lumbosacral articulation during a three year period. A detailed questionnaire focusing on signs related to lumbosacral problems was answered by the owners and their veterinarians. All dogs underwent a physical, neurological and orthopedic examination and plain radiographs of the lumbosacral junction were taken in all dogs at the beginning and the end of the study. Presence of DLS was confirmed by myelography, epidurography or MRI in 9 dogs. In the remaining dogs, diagnosis was based upon neurological findings, radiographic exclusion of orthopaedic and spinal disorders that may mimick DLS and repeated neurological examinations. Differential diagnosis such as nerve root tumors or the lower motor

neuron form of degenerative myelopathie would have become overt during the 3 year period of surveillance. Radiographic parameters that could potentially predict development of DLS were analysed. On the 2nd neurological examination signs of lumbosacral disease were found in 15 dogs and 18 dogs were normal. 22 dogs were able to perform unrestricted duty and 4 dogs were used for restricted duty. The reason for the restriction was DLS in 3 dogs. 7 dogs had been excluded from active duty during the period of surveillance, 6 because of DLS. Of the 15 clinically affected dogs, 13 showed radiological abnormalities in the lumbosacral area. Of the 18 dogs without clinical symptoms, 14 showed radiographic abnormalities of their lumbosacral junction. Altough the progression of both clinical and radiological parameters was statistically significant, correlation analysis showed no association between clinical and radiological findings.

In conclusion, the prevalence of DLS-affected dogs in this uniform population of working GSDs was found to be 45%. Drop-out rate including those dogs, that were not fully deployable was 29%. However, mildly to moderately affected GSDs may be used in active duty for months to even years. Radiographic signs that might predict development of DLS were not found in this study.

ABSTRACT # 65

UNUSUAL CASE OF DISTEMPER ENCEPHALITIS MIMICKING A CEREBRAL NEOPLASIA. T Steinberg[1], K Matiasek[2], K Jurina[3], K Flatz[4], A Fischer[1]. [1]Section of Neurology, Department of Small Animal Medicine, Ludwig-Maximilians-University, Munich, [2]Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, Ludwig-Maximilians-University, Munich, [3]Small Animal Hospital Haar [4]Diagnostic Imaging Center, Clinic for Small Animal Surgery, Ludwig-Maximilians-University, Munich, Germany

Subject Canine distemper is a viral disease causing most notably multifocal lesions in the grey and white matter of the central nervous system. Inflammatory diseases of the CNS presenting as a single mass lesion are infrequently described in veterinary medicine. They should be taken into account as a differential diagnosis for intracranial neoplastic lesions.

Material and Methods A 1.5 year-old female intact Saint Bernard dog was referred because of an acute onset of generalised seizures. On presentation the dog appeared slightly depressed but clinical and neurological examination was otherwise normal. Laboratory work-up including pre- and postprandial bile acids, abdominal ultrasound and thoracic radiographs was unremarkable. Due to the seizures the lesion was localized to the forebrain.

The dog underwent magnetic resonace imaging (MRI) of the brain with a Siemens Magnetom Symphonie 1.5 Tesla unit. Pre- and post-contrast T1W, T2W, CISS and FLAIR sequences were obtained in transverse, dorsal and sagittal orientations. Cerebrospinal fluid (CSF) was taken from the atlantooccipital site.

Results A well-defined broad-based and round-shaped lesion with a maximal expansion of 1×1×1 cm was demonstrated post-contrast on T1W sequences. Pronounced dural tail sign was noticed. The lesion appeared hypo- to isointense on pre-contrast T1W sequences. On both, T2W and FLAIR sequences a strong hyperintense signal was notable extending caudally into the pyriform lobe. Thus, marked surrounding edema was suspected. CISS sequences implicated an extraaxial location of the lesion. CSF analysis was normal. Based on MRI appearance of the lesion differentials included menigioma, brain metastasis or local inflammation. The dog was euthanized because of severe postictal aggressive behaviour. Histological examination of the brain revealed disseminated perivascular lympho-histiocytic infiltrates and a focal marked degenerative and edematous area within the right pyriform lobe with lymphocytic infiltrates, reactive astrogliosis and activation of microglia. Immunohistochemistry demonstrated the presence of intraneuronal canine distemper virus antigen.

Conclusions MRI appearance of focal distemper encephalitis can resemble intracranial neoplasia. In young dogs, distemper encephalitis should be considered as a possible underlying cause of a focal contrast-enhancing lesion on MRI.

ABSTRACT # 66

EVALUATION OF BRAIN MATURATION IN LABRADOR PUPPIES WITH MAGNETIC RESONNANCE IMAGING. JL. Thibaud[1], I. Barthelemy[1], F. Delisle[2], S. Blot[1]. [1]Neurology Unit, Veterinary School of Alfort, [2]Scan-Vet. Maisons-Alfort, France

Development of the mammalian brain is not completely achieved at birth. Myelination is a major biological event during post-natal brain development. MRI offers the unique opportunity to document developmental features non-invasively and accurately. This study describes MRI evolution of the canine brain between 1 and 6 months of age.

Three Labrador retriever puppies were examined at 1, 2, 3 and 6 months of age. MRI scans were performed using a knee coil in a 0.2 Tesla system. Axial T1-and T2-weighted images were obtained, then dorsal T1-, T2- and Fluid Attenuated Inversion Recovery (FLAIR)-weighted images. T1-weighted images were acquired with parameters as follows: echo time (ET)= 12ms, repetition time (RT)= 380ms (axial) and 300ms (dorsal), field of view (FOV)= 16 cm, matrix =192x192. T2-weighted image parameters were: ET=100ms, RT=5250ms (axial) and 4000ms (dorsal), FOV=16cm, matrix: 192x192 (axial) and 224x192 (dorsal). FLAIR-weighted image parameters were: ET=108ms, RT=5600ms, inversion time=1400ms, FOV=18cm, matrix 192x192. Section thickness was 3,5mm (axial) and 3mm (dorsal).

Our results have associated:

- a disappearance of the relative T1-hyperintensity of the white matter (WM), seen first in the brainstem (1m) then in the internal capsule (2m), at 3 months of age,
- an increase in the gray/white matter contrast on T2 imaging which seems normal at 3 months, even in the frontal lobes,
- a decrease of ventricles and sulci size, comparable to the adult size at 6 months of age.

On T2-weighted images, the cortical WM seems hyperintense at 1 month, hypo- to isointense at 2 months. On FLAIR-weighted images, gray/white matter contrast appears at 2 months.

These features are also described in human patients. The increase of the gray/white matter contrast is due to myelination and increase in saturated long chain fatty acids. Before myelination, the lipid and water components are similar in gray and white matter. Myelination induces a decrease in the water content and an increase in lipids and proteins. The decrease in the free water fraction and the myelin interaction are responsible for the relative T2-hypointensity of the WM. The decrease in ventricle and sulci size could be explained by the increase in dry weight (about 50%) due to an increase in number and size of glial cells. This qualitative MRI evaluation is transposable in routine clinical conditions for the monitoring of puppies with suspected brain disease. Knowledge of normal maturation is a prerequisite to the evaluation of puppies with abnormal or delayed myelination. However this study should be completed with MRI scans of dogs from smaller and taller breeds. Quantitative studies could also be performed and would be more accurate to determine more subtle changes.

ABSTRACT # 67

A SPONTANEOUS INTRAMEDULLARY HAEMATOMA IN A DOG: PRE- AND POST OPERATIVE CLINICAL AND MAGNETIC RESONANCE IMAGING FOLLOW-UP. JL. Thibaud[1], A. Hidalgo[2], G. Benchekroun[3], F. Crespeau[4], F. Delisle[5], S. Blot[1]. [1]Neurology Unit, [2]Surgery Unit, [3]Medical Unit, [4]Pathological Unit, Veterinary School of Alfort, [5]Scan-Vet. Maisons-Alfort, France

Intramedullary haematomas are rarely described in humans as in veterinary neurology. Increasing use of MRI currently allows to identify pre-mortem intraparenchymatous haematomas. This case illustrates the clinical and MRI features due to a spontaneous intramedullary haematoma in a dog before and after surgical treatment.

A 4-year-old male Jack Russel Terrier was presented for a right biped lameness with episodic neck pain, degrading since 6 months. General examination was normal but neurological examination showed conscious proprioceptive deficit on the right limbs and increased muscle tone. Reflexes were normal. A severe neck pain was elicited. Steroids transiently improved the animal. CSF analysis showed increased protein (0,39g/l) and normal cell count without erythrophagia. CBC, prothrombin time and activated partial thromboplastin time were within normal limits. Cervical spine radiograph and computed tomodensitometry were normal. On MRI examination, a right intramedullary lesion was identified at the level of the 4th cervical vertebra. A T1-hyperintense and T2-hypo/isointense lesion was surrounded by a T1- and T2-hyperintense lesion. There was no contrast enhancement. Two months later, the dog was suddenly tetraparetic and developed a right partial Horner's syndrome. Pain could not be relieved even with opioïds. MRI was repeated. The spinal cord was markedly enlarged and a T1-hyperintense and T2-hypointense lesion was surrounded by a T1-hypointense and T2-hyperintense lesion, consistent with a subacute bleeding. A dorsal myelotomy was performed. A fluid-surrounded haematoma was removed and analysed as well as a medullary biopsy. The subacute haematoma was histologically confirmed and no vascular malformation or neoplastic cells were seen. After surgery the dog was in pain and tetraplegic. Five months later, only a conscious proprioceptive deficit was elicited on the right

forelimb. Two MRI examinations were performed, 2 weeks and 5 months after surgery. The former revealed a T2-hyperintense and a mixed T1-hypo/hyperintense lesion with mild contrast enhancement. The latter revealed a more focal T2-hyperintense and T1-isointense lesion with no contrast-enhancement. To our knowledge, this is the first case of canine spontaneous intramedullary haematoma. No evidence of a predisposing cause of bleeding was identified: primitive or secondary coagulopathy, medullary tumor, vascular malformation. However spinal angiography, faecal analysis and Von Willebrand factor evaluation were not performed. The evolution of the MRI signal was useful to identify the iterative bleedings without any other lesions. Moreover, the post-operative MRI controls revealed a transient gadolinium enhancement as previously described by Walker and al. in 2004. Knowledge of this phenomenon is essential for accurate assessment of residual enhancement.

ABSTRACT # 68

HYPERINHIBITION: A FURTHER CAUSE OF MOTOR NEURON DEGENERATION?! D. Thinnes[1], L. Becker[2], L. Matiasek[1], A. Rupp[1], A. Fischer[3], H. Weyer[2], W. Schmahl[1], K. Matiasek[1]. [1]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, LMU Munich; [2]Institute of Diabetes Research, Academic Hospital Munich-Schwabing, Munich; [3]Section of Neurology, Department of Small Animal Medicine, LMU Munich, Germany

Glycine is the most important inhibitory transmitter in the spinal cord and lower brain stem. The Glycine-Receptor (GlyR) represents a ligand-gated ion channel and consists of a pentamer comprising 3 α- and 2 β-subunits. The β-subunit is linked to gephyrin, both anchoring the receptor to the postsynaptic membrane, while the α-subunit is the ligand-binding site. Contact with glycine leads to chloride-influx and hyperpolarisation of the neuron.

All recently reported subunit-mutations resulted in GlyR-hypofunction, which causes neuronal overexcitability. The clinical picture is that of hyperekplexia, also called stiff-baby-syndrome or startle-disease, which has been noted in several mammalian species. Amongst murine mutants *spastic*-mice exhibits a decreased transcription of the GlyRβ-subunit and, thereby, reduced numbers of functional GlyRs, while lines *spasmodic* and *oscillator* both have a point-mutation of the α_1-subunit. *Spasmodic* mice show a decreased affinity to glycine whereas *oscillators* suffer from complete loss of receptor-function. All these mouse-lines display exaggerated startle-response, stimulus-induced muscle rigidity, delayed righting-time and tremor.

In contrast to these studies, the impact of excessive glycinergic action on the nervous system is largely unknown. Thus, the present study was aimed to investigate overexpression of wildtype GlyRα_1-subunit in genetically engineered mice. Therefor we examined 29 GlyRα_1-transgenic mice according to the SHIRPA-protocol and compared them to 30 age-matched controls. In a second step, 20 animals per group were evaluated through EMG and NCS followed by histopathological and morphometrical investigation of the spinal cord, peripheral nerves and limb muscles.

Within 3 weeks of age all transgenic mice developed abnormal clenching of the hind limbs when lifted by the tail, delayed contact-righting, severe ataxia and paresis of the hind limbs, muscle-atrophy, and a kyphotic backline. Symptoms worsened with increasing age, albeit the defect remained sublethal. Electrophysiological testing revealed fibrillation potentials and positive sharp waves in all hind limb muscles. Moreover, F-waves could not be elicited in transgenic animals. Histopathology showed ongoing motor neuron degeneration associated with fibre-loss in peripheral nerves, and neurogenic muscle atrophy.

Taken together, excessive glycinergic inhibition induces ALS (amyotrophic lateral sclerosis)-like motor neuron degeneration in mice. This effect seems to be partially mediated by employment of the BDNF (brain derived neurotrophic factor) system and provides a novel target for therapeutic tools in motor neuron diseases.

ABSTRACT # 69

EFFECTS AND MONITORING OF SEDATION AND ANESTHESIA FOR TRANSCRANIAL MAGNETIC MOTOR EVOKED POTENTIALS IN DOGS. I. Van Soens[1], M. Struys[2], I. Polis[1], M. Tshamala[1], L. Van Ham[1]. [1]Department of Small Animal Medicine and Clinical Biology. [2]Department of Anaesthesia. Ghent University, Belgium.

Transcranial magnetic motor evoked potentials (TMMEP) may be a clinically useful, non-invasive and painless tool to asses motor pathway integrity. In veterinary medicine, sedation or anesthesia are necessary for the use of TMMEP. However, most anesthetic regimens result in a depression of the evoked responses. We tried to standardize type and depth of anesthesia for TMMEP monitoring.

In the present study four anesthesia protocols were used: acepromazine (0,01mg/kg) (Placivet)-methadone (0,1mg/kg) (Mephenon) and propofol (2mg/kg) (Rapinovet); acepromazine (0,01mg/kg)-methadone (0,1mg/kg) and etomidate (1,5mg/kg) (Hypnomidate); medetomidine (4 µg/kg) (Domitor) and propofol (2mg/kg); medetomidine (4 µg/kg) and etomidate (1,5mg/kg). Sedation and anesthesia was monitored using Bispectral analysis index (BIS) and midlatency auditory evoked potentials index (AAI).

Six Beagle dogs were anesthetized four times with a different sedation-anesthesia protocol following a cross-over study (at least 1 week interval). TMMEP was performed during sedation and during general anaesthesia (every three minutes until the dogs were too awake to perform the procedure). Anesthesia was continuously monitored using BIS and AAI.

TMMEP could be evoked during sedation and anesthesia. TMMEP recorded during general anesthesia demonstrated a significant decrease in peak-to-peak amplitude but no significant changes in onset-latency. Propofol depressed peak-to-peak amplitude more then etomidate.

BIS and AAI correlated well with clinical appearance during sedation in this study. After injection of the anesthetic, a marked decrease in BIS and AAI was seen. However, changes in BIS and AAI did not statistically correlate with changes in onset latency and peak-to-peak amplitude. These results indicate that BIS and AAI could not be used to objectively monitor anesthesia for TMMEP.

Conclusion: sedation with medetomidine and acepromazine-methadone could both be used to perform TMMEP. Nevertheless, medetomidine offers more practical advantages in comparison to acepromazine-methadone. General anesthesia with etomidate is more feasible and reliable for TMMEP than propofol. BIS and AAI can be used as 'hypnosis' monitor during sedation and anesthesia for the used sedative and anesthetic products.

ABSTRACT # 70

CONTRIBUTION OF CUTANEOUS INPUTS FROM THE HINDPAW TO THE CONTROL OF LOCOMOTION IN RATS. Artur S.P. Varejão[1], Vítor M. Filipe[2],José E. Pereira[1], Luís M. Costa[1], Pedro A. Couto[2], António Cunha[2], Raul Morais[2], Pedro Melo-Pinto[2], José Bulas-Cruz[2]. [1]Department of Veterinary Sciences, [2]Department of Engineering, CETAV, University of Trás-os-Montes e Alto Douro, Vila Real, Portugal

The study of locomotion in rats is used extensively in spinal cord injury research. The central pattern generators are capable of inducing stepping patterns in the absence of supraspinal input. The goal of these experiments was to define the contribution of hindpaw cutaneous feedback in the control of locomotion. The question we ask here is whether gait deficits become apparent also in intact animals after removing these cutaneous inputs.

To evaluate this question, we conducted a detailed hindlimb kinematic analysis in animals who suffered complete loss of thermal sensibility. Two-dimensional hindlimb kinematics, temporal and spatial measurements, and walking track analysis were performed in six rats before and after hypothermic anesthesia, during locomotion.

The walking velocity, duration of the step cycle and stance phase, and stride length were statistically indistinguishable between the two testing conditions. Swing phase duration was significantly decreased after sensory loss. Analysis of angular motion revealed an increased hip and knee extension and an ankle joint with increased flexion during the step cycle under plantar anesthesia. Also after plantar cooling, the hip and knee angular velocity was significantly affected along the step cycle. The remarkably geometric similarity of the angle-angle plots obtained in our experiments reflected an interjoint coordination; however the interpretation of the cyclogram perimeter revealed a larger excursion by the ankle and hip in their respective joint spaces in rats deprived of sensation. Examination of the horizontal position of the ankle with respect to the hip during the step cycle and the extension before toe-off revealed no major changes, whereas there was a slight decrease in distance of the hip to the ground after sensory loss. Also, the walking tracks revealed a significant functional deficit following reduced cutaneous information of the plantar aspect of the hindpaw.

We conclude that sensory feedback from the hindpaw is important in the maintenance of normal rat locomotion. The present investigation provides further insights into the control mechanisms of gait in spinal cord injured animals, where it seems likely that initiation of adaptive and compensatory mechanisms after injury will use the information from cutaneous receptors.

ABSTRACT # 71

RETROSPECTIVE STUDY OF HISTOLOGICALLY CONFIRMED BRAIN TUMOURS IN DOGS. Pandy Vidhya[1], Simon Platt[2], Kate

Chandler[1], David Brodbelt[1], Rodolfo Cappello[1]. [1]The Royal Veterinary College, University of London, UK. [2]The Animal Health Trust, Newmarket, UK

Traditionally neuroepithelial tumours (oligodendrogliomas and astrocytomas) and pituitary tumours are associated with brachycephalic breeds and meningiomas with dolichocephalic breeds. In a comprehensive study of 237 dogs with brain tumours, oligodendrogliomas were found to be the most common tumours in brachycephalic dogs such as Boxers, Boston terriers and Bull dogs. This retrospective study of histologically confirmed brain tumours was conducted to explore the correlation between skull morphology and affinity for a particular brain tumour. Signalment and tumour classification were evaluated in 224 dogs (1995–2005) from the Animal Health Trust (AHT) and the Royal Veterinary College (RVC). Histopathological examination of the brain was performed by necropsy or via craniotomy removal of the affected tissues by the respective pathology departments at AHT and RVC. The World Health Organisation (WHO) tumour classification for domestic animals was employed and where appropriate, immunohistochemistry was used for classification. Of the 224 cases analysed, 87 tumours of the neuroepithelial tissues, 84 tumours of the meninges and 53 other types were diagnosed. Analysis of the type of tumour revealed that the prevalence of gliomas was significantly higher (P=0.003) in brachycephalic dogs than dolichocephalic breeds and brachycephalic dogs are 3.89 times more likely to develop gliomas than meningiomas. A comparison of brachycephalic dogs against the combination of dolicho- and mesocephalic dogs showed similar results with brachycephalic dogs 3.21 times more likely to develop gliomas than non-brachycephalic dogs (P=0.001). The neuroepithelial tumour subtypes in brachycephalic dogs were distributed in oligondendroglioma (56%) astrocytoma (20%) and other types of neuroepithelial tumour (26%). Among the 55 breeds investigated in this study, Boxers had the highest incidence of tumours (21%; n=48) and represented 77% of all brachycephalic dogs. Other breeds with a high incidence of tumours were, Cross breeds (13%; n=29), German Shepherd dogs (6.6%; n=15), Labrador Retrievers (7.5%; n=17) and Golden Retrievers (5.3%; n=12). In the 84 meningiomas, 46 occurred in females and 38 in males with the highest prevalence in spayed females (45%). Of the 87 neuroepithelial tumours, 49 occurred in entire males and 10 in neutered males with the combined prevalence being 68%. Analysis of type of tumour and age group showed that the younger dogs (<6 years of age) have a higher frequency(51%) of neuroepithelial tumours than tumours of other cellular origin.

Elaboration of this retrospective study with particular attention to certain brachycephalic breeds and brain morphology could lead to better understanding of the pathogenesis of canine glioma. Further gene expression studies exploring the possible genetic predisposition of brachycephalic breeds to gliomas are warranted in the future

ABSTRACT # 72

MAGNETIC RESONANCE IMAGING OF FOCAL RADICULOPATHY. Walmsley G, Mantis P and Cappello R. The Royal Veterinary College, University of London, UK.

Nerve root tumours commonly affect the spine in dogs, however the magnetic resonance imaging (MRI) appearance of such lesions is poorly documented in the veterinary literature. The aim of this retrospective study is to present the MRI findings of a series of nerve root lesions (neoplastic and non-neoplastic) and attempt to define distinguishing features.

Eight cases were seen at the Queen Mother Hospital for Animals and diagnosed with spinal nerve root lesions based on histopathology. Five cases were diagnosed with peripheral nerve sheath tumours, in one case appearance and immunostaining were suggestive of a mesenchymal tumour and two cases had non-neoplastic lesions: lymphocytic ganglioneuritis and mature, reactive fibrovascular tissue surrounding the nerve root and ganglion. Scans were retrieved and the MRI studies were reviewed by two examiners who had to reach an agreement.

Nerve root lesions were visible on MRI in all cases. In 2 MRI interpretation underestimated the extent of the lesion compared with gross inspection. Neoplastic lesions were spread throughout the spine with C6-T2 and T3-L3 cord segments more commonly affected while both non-neoplastic lesions affected the L4-S3 segment. Almost all lesions extended into the spinal canal causing cord or cauda equina involvement (either compression or invasion) in 5 of 6 neoplastic lesions and 2 of 2 non-neoplastic lesions. The majority of lesions had distinct margins however 2 of 6 neoplastic lesions invaded both spinal cord and vertebrae. Lesion intensity was variable and all cases showed contrast enhancement, however only neoplastic lesions (2 of 6) had a peripheral pattern of enhancement. In all cases the lesion appeared as a focal enlargement, which was 2.3 ± 0.7 (mean ± SD) times larger than the contralateral nerve in neoplastic lesions and 1.5 and 2.5 times larger in non-neoplastic lesions. Atrophy of surrounding musculature was obvious in 2 of 6 neoplastic lesions and one non-neoplastic lesion. Degenerative spinal disease was associated with both non-neoplastic lesions: extrusion of the L6/7 disc and lumbosacral disease, this was not the case with the neoplastic lesions.

In conclusion, non-neoplastic lesions may be indistinguishable from neoplastic lesions. Features of malignancy suggested by these results include very large or invasive masses and those with signs suggestive of central necrosis however nerve root neoplasms can be of small diameter and the majority are non-invasive. Interestingly, both non-neoplastic lesions were associated with diseases affecting the surrounding tissues - these could be primary, secondary or co-incidental. Finally, we suggest that inflammatory and hyperplastic lesions should be considered as differentials for focal enlargements of nerve roots.

ABSTRACT # 73

ELECTRODIAGNOSTIC STUDIES OF A CASE OF CANINE GLOBOID CELL LEUKODYSTROPHY. O. Zeira[1], E. Bianchi[2], D. Callegari[2], A.M. Cantoni[2], R. Di Lecce[2], M. Dondi[2]. [1]Department of Veterinary Surgery, University of Camerino, Italy. [2]Animal Health Department, University of Parma, Italy.

Globoid cell leukodystrophy (GLD) is a rare hereditary lysosomal storage disease that results in progressive degeneration of white matter in CNS and PNS. The disease is caused by a deficiency of the enzyme galactosylceramidase which leads to the accumulation of the lipid psychosine, that is highly toxic to oligodendrocytes and Swann cells. This disorder is caused by mutations of the gene encoding galactosylceramidase and has been reported in humans, dogs and other species. This report describes a case of GLD in a puppy with particular regard to clinical neurophysiology results. A 5 months old, male, West Highland white terrier dog was referred with a history of progressive hindlimb ataxia and paresis. Physical examination was unremarkable. Findings on neurologic examination were intention tremors, hypermetria and ataxia of the hindlimbs and paraparesis. Postural reactions and spinal reflexes were reduced in both hindlimbs. A complete blood cell count, urinalysis and serum biochemistry panel with fasting and postprandial bile acids were unremarkable. CSF analysis showed a slightly increased cell count and a moderate elevation in protein concentration. Serology tests for Neospora and Toxoplasma and PCR test for Distemper virus in the CSF were negative. Electromyography revealed mild spontaneous activity in appendicular muscles. Motor and sensory nerve conduction studies showed reduced conduction velocities, polyphasia and temporal dispersion. Minimum F wave latencies were increased, while supramaximal repetitive nerve stimulation was normal. Brainstem auditory-evoked potentials (BAEP) tracings were characterised by prolonged absolute and interpeak latencies of the waves with normal thresholds. Somatosensory evoked potentials (SSEP) showed prolonged central conduction times and delayed cord dorsum potential latencies. These electrodiagnostic findings were suggestive of peripheral and central demyelination with only mild axonal involvement. A progression of the myelinopathy was evident from the electrodiagnostic tests performed after 20 days. These findings increased the diagnostic suspect of GLD, that was confirmed by genetic testing and pathologic findings. A definitive antemortem diagnosis of GLD can be achieved only with genetic testing or measuring galactosylceramidase activity in leukocytes. Nevertheless clinical neurophysiology tests represent sensitive diagnostic tools especially in the first stages of the disease in detecting early signs of demyelination. Moreover they provide objective data on function of the tested structures. Therefore these tests can be used to monitor the progression of the disease and verify the efficacy of new therapies.

ABSTRACT # 74

FINDINGS OF MAGNETIC RESONANCE IMAGING IN SUSPECTED CANINE FIBRO-CARTILAGINOUS EMBOLIZATION. V.M. Stein[1], F. Wagner[1,2], C. Bull[1], A. Gerdwilker[1], F. Seehusen[3], W. Baumgärtner[3], A. Tipold[1]. Department of Small Animal Medicine and Surgery[1], Institute of Pathology[3], University of Veterinary Medicine, Hannover, Germany, Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, The Netherlands[2]

Fibrocartilaginous embolization (FCE) of the spinal cord is a common cause of acute onset paresis or plegia of thoracic and/or pelvic limbs. The diagnosis was formerly achieved as one of exclusion of compressive diseases by myelography and/or computed tomography. With the advent of magnetic resonance imaging (MRI) in veterinary medicine a new potential diagnostic tool for the intra vitam diagnosis of FCE is available.

In an effort to assess the MRI appearance of FCE the clinical records of dogs (from Febr. 2004 to Febr. 2006) with acute to peracute onset of paresis/

plegia of thoracic and/or pelvic limbs, frequently with asymmetry of signs were retrospectively evaluated. Dogs with compressive diseases and tumors were excluded from the study. Twentysix dogs met the inclusion criteria. Twentytwo were suspected to suffer from FCE, six of these dogs were euthanized and FCE was confirmed histopathologically. The remaining 4 of the 26 dogs suffered from explosive intervertebral disc herniation as confirmed by intraoperative or histopathological findings.

The MRI (Magnetom Impact Plus, 1.0 Tesla, Siemens, Erlangen, Germany) revealed an intramedullary hyperintensity in T2-weighted turbo spinecho (TSE, TE 112 ms, TR 4700 ms, Flip angle 180°) images in 18 of the 26 dogs whereas in the remaining 8 dogs the spinal cord was normointense. T1-weighted spinecho (SE, TE 12 ms, TR 330 ms, Flip angle 90°) images of the corresponding T2-lesions showed a hypointensity or were normointense to the surrounding spinal cord. Single pre-treatment with glucocorticosteroids could not be correlated with presence or absence of a T2-hyperintensity, the severity of neurological signs tended towards being milder in dogs with normointense spinal cord and the outcome was better in these dogs. However, the time lapsed since clinical signs began and the MR imaging as well as the size and localization of the embolized blood vessels may have an important impact on the appearance of FCE in the MRI.

In conclusion, our study describes for the first time the occurrence of histopathologically confirmed FCE without signs of T2-hyperintensity in the MRI. And MRI is not omniscient: explosive intervertebral disc herniations can produce MRI findings indistinguishable from FCE. Therefore, the absence of an intramedullary hyperintensity does not exclude the existence of FCE, and in reverse the detection of a hyperintensity in T2-weighted images does not necessarily confirm the existence of FCE. Characteristic clinical signs in line with the history, and in combination with the findings in MRI can lead to the diagnosis of FCE whereas explosive intervertebral disc herniation still might be an important differential diagnosis.