PROCEEDINGS 22nd Symposium ESVN-ECVN BOLOGNA 24th–26th September 2009

Selected research communications of the 22nd Symposium of ESVN-ECVN Bologna, Italy 24th to 26th September 2009

TIMETABLE OF THE SYMPOSIUM

FRIDAY 25TH SEPTEMBER

9.00: **Welcome to participants**

9.15: **INVITED SPEAKERS SESSION – *chairperson: MARC VANDEVELDE***

Brian Summers:

"*TUMORS OF THE NERVOUS SYSTEM IN DOMESTIC ANIMALS - I INTRODUCTION*"

"*TUMORS OF THE NERVOUS SYSTEM IN DOMESTIC ANIMALS - II SELECTED CASES*"

11.15: Coffee break, poster exhibition and sponsors

12.00: **INVITED SPEAKERS SESSION – *chairperson: MARC VANDEVELDE***

Peter J. Dickinson:

"CHARACTERIZATION OF CANINE BRAIN TUMORS: A MODEL FOR HUMAN DISEASE?"

13.00 Lunch poster exhibition and sponsors

14.15: **INVITED SPEAKERS SESSION – *chairperson: LAURENT GAROSI***

Peter J. Dickinson:

"NOVEL THERAPEUTIC APPROACHES FOR CANINE BRAIN TUMORS"

Andrea Salmaggi:

"CHEMOTHERAPY AND TARGETED THERAPY IN GLIOMAS"

16.15: Coffee break, poster exhibition and sponsors

16.45: **PLATFORM PRESENTATIONS: I session – *chairperson: LAURENT GAROSI***

1) VALIDATION OF A MAGNETIC RESONANCE IMAGING COMPATIBLE, FRAMELESS STEREOTACTIC BRAIN BIOPSY SYSTEM IN THE DOG.
   AV Chen, FA Wininger, S Frey, R Comeau, RS Bagley, RL Tucker, AR Schneider, JM Gay.
2) CANCER STEM CELLS IN CANINE GLIOMAS: PRELIMINARY RESULTS IN A STUDY OF 17 CASES
   Foradada L, Vidal E, Márquez M, Fondevila D, Rabanal R, Pumarola M
3) TESTING EPIGENETIC CONCEPTS IN CANINE NEUROONCOLOGY I: METHYLATION OF DNA-REPAIR ENZYME O6-Methylguanin-DNA-Methyltransferase
   L Matiasek, J Schlegel, M Starkey, L De Risio, T Flegel, K Baiker, K Matiasek
4) TESTING EpiGENETIC CONCEPTS IN CANINE NEUROONCOLOGY II: HISTONE ACETYLATION
   K Matiasek, L De Risio, M Ortega Prieto, L Matiasek
5) A COMPARATIVE STUDY OF CANINE AND FELINE MENINGIOMA CLASSIFICATION BASED ON WHO HISTOLOGICAL CLASSIFICATION SYSTEM IN HUMANS.
   M.T. Mandara, S. Pavone, B. Brunetti, L. Mandrioli

18.00 **closing of the day**

20.00 **GALA DINNER – PALAZZO ISOLANI**

SATURDAY 26th SEPTEMBER

8.45: **PLATFORM PRESENTATIONS: II session – *chairperson: ROBERTO POMA***

6) CEREBRAL NECROSIS FOLLOWING HYPOFRACTIONATED RADIOTHERAPY FOR CANINE INTRACRANIAL TUMORS: A MAGNETIC RESONANCE IMAGING AND PATHOLOGICAL STUDY
Alejandro Luján Feliu-Pascual, Ruth Dennis, Sue Murphy, Luisa De Risio, Kaspar Matiasek.

7) SPHENOID BONE INFILTRATION IN A DOG WITH DISSEMINATED MAST CELL TUMOUR
E. Beltran, A. de Stefani, J. Stewart, L. De Risio, V. Johnson.

8) ANTEMORTEM DIAGNOSIS OF INTRACRANIAL AND OCULAR METASTASES OF A MIX MALIGNANT MAMMARY TUMOR
I. Srugo, I. Aroch, A. Berkowitz, N. Edery, O.Chai, and M.H. Shamir.

9) TYPE IV DERMOID SINUS IN AN ENGLISH BULL DOG: MAGNETIC RESONANCE IMAGING, SURGICAL AND HISTOLOGICAL FINDINGS
Fabio Stabile, Luisa De Risio, Alberta de Stefani, Kaspar Matiasek, Julien Labruyere, Andrew Holloway

10) PROGNOSTIC VALUE OF AN IMAGING SEVERITY INDEX (ISI) FOR ROSTROTENTORIAL MASS LESIONS IN THE DOG
Roberto José-López, Mark Lowrie, Inés Carrera and Jacques Penderis

11) LOW FIELD MAGNETIC RESONANCE IMAGING (MRI) IN DOGS WITH AND WITHOUT SIGNS OF DISC ASSOCIATED WOBBLER SYNDROME (DAWS): A RANDOMIZED, BLINDED STUDY.
S De Decker, IMVL Gielen, L Duchateau, J Lang, R Dennis, N Corzo-Menéndez, HJJ van Bree, I Van Soens, D Binst, T Waelbers, LML Van Ham.

12) TRANSCRANIAL MAGNETIC STIMULATION (TMS) IN DOBERMANN PINSCHERS WITH AND WITHOUT SIGNS OF DISC ASSOCIATED WOBBLER SYNDROME (DAWS): USEFUL TOOL TO DIFFERENTIATE BETWEEN CLINICALLY RELEVANT AND IRRELEVANT SPINAL CORD COMPRESSION?
S De Decker, I Van Soens, L Duchateau, IMVL Gielen, HJJ van Bree, D Binst, T Waelbers, LML Van Ham

13) TEACHING VETERINARY NEUROLOGY: EXPERIENCE WITH AN INTER-FACULTY eLEARNING ELECTIVE COURSE.
M. Koch, A. Tipold, M. Fischer, M. Vandevelde, J.P. Ehlers.

10.45: Coffee break, poster exhibition and sponsors

11.30: **Annual General Meeting (AGM) of the European Society and College of Veterinary Neurology**

13.00 Lunch poster exhibition and sponsors

14.30: **PLATFORM PRESENTATIONS: III session – *chairperson: KATE CHANDLER***

14) WEST NILE VIRUS OUTBREAK IN ITALY: CLINICAL FINDINGS IN 10 HORSES
G. Gandini, A. Gallucci, L. Mandrioli, A. Spadari, M Rosati, F. Dondi, N. Romagnoli, G. Bettini

15) WEST NILE VIRUS OUTBREAK IN ITALY: PATHOLOGICAL FINDINGS IN TWO HORSES
L. Mandrioli, G. Bettini, M. Morini, A. Gallucci, R Biserni, A. Spadari, G. Gandini.

16) MULTI-SYSTEM NEURONAL DEGENERATION IN A FAMILY OF ATAXIC NORWEGIAN BUHUND
A de Stefani, K Matiasek, O Forman, L De Risio.

17) CLINICAL AND TOPOGRAPHIC MAGNETIC RESONANCE IMAGING CHARACTERISTICS OF SUSPECTED THALAMIC INFARCTS IN 18 DOGS
R. Gonçalves, I. Carrera, L. Garosi, P. Smith, F. McConnell, J. Penderis

18) EFFECT OF MIDDLE EAR EFFUSION ON THE BRAINSTEM AUDITORY RESPONSE OF CAVALIER KING CHARLES SPANIELS
T. R. Harcourt-Brown, J. E. Parker, N. D. Jeffery

19) COMPARATIVE SEMIOLOGY OF MOTOR ACTIVITY DURING SEIZURES IN MAN AND ANIMALS: PRELIMINARY RESULTS AND PROPOSAL FOR A COLLABORATIVE STUDY
C. A. Tassinari

16.00: Coffee break, poster exhibition and sponsors

16.30: **PLATFORM PRESENTATIONS: IV session – *chairperson: MARIA TERESA MANDARA***

20) VOLUMETRIC ANALYSIS OF BRAIN PARENCHYMA WITHIN THE CAUDAL FOSSAE OF CAVALIER KING CHARLES SPANIELS.
C Driver, C Rusbridge, H Cross, HA Volk.

21) GENERALIZED MYOKYMIA AND NEUROMYOTONIA IN JACK RUSSELL TERRIERS: A CLINICAL AND ELECTROPHYSIOLOGICAL STUDY.
A.E. Vanhaesebrouck, L. Poncelet, I. Van Soens, L. Duchateau, H.C. Schenk, I. Polis, S. Bhatti, S. Diels, L. Van Ham.

22) NEUROLOGICAL DYSFUNCTION AND HYPOVITAMINOSIS A IN A 6 MONTH OLD CHEETAH (*Acinonyx jubatus*)
Luisa De Risio, Elsa Beltran, Andrew Holloway, Anthony Tropeano, Alberta de Stefani, John Lewis

23) CLINICAL SIGNS AND MRI FINDINGS OF BILATERAL PORENCEPHALY AND CAUDAL VERMIAN HYPOPLASIA IN A YOUNG DOG.
M. Rosati, M. Bernardini, P. Calò, L. Pisoni, G. Gandini

24) MULTIFOCAL ISCHAEMIC STROKES DUE TO A HYPERCOAGULABLE STATE CAUSED BY A SMALL INTESTINAL HIGH-GRADE T-CELL LYMPHOMA IN A DOG.
Meichner K, Flatz K, Schnabl E, Schulz B, Keller LJM, Janik D, Ludwig E, Fischer A.

17.45: **John Presthus and Bayer awards**

18.00: **Closing remarks**

PLATFORM PRESENTATION

VALIDATION OF A MAGNETIC RESONANCE IMAGING COMPATIBLE, FRAMELESS STEREOTACTIC BRAIN BIOPSY SYSTEM IN THE DOG. AV Chen[1], FA Wininger[1], S Frey[2,3], R Comeau[3], RS Bagley[1], RL Tucker[1], AR Schneider[1], JM Gay[1]. 1. Washington State University College of Veterinary Medicine, Pullman, WA. 2. Montreal Neurological Institute, McGill University, Montreal, Quebec. 3. Rogue Research Inc., Montreal, Quebec.

A stereotactic brain biopsy system that is MRI compatible has not been validated in dogs. The purpose of this study was to determine the mean needle placement error in canine cadaver brains using the modified Brainsight™ frameless stereotactic system.

Cadaver heads were disarticulated at C2-3, stored at 10 °C and used within 24 hours of euthanasia. Relocatable reference points (fiducial markers) were attached to the cadaver heads using a dental bite block. A T1-weighted gradient echo 3D sequence was acquired using set parameters. Fiducial markers were used to register the head to the acquired MR images in reference to a 3D position sensor. This allowed the planning of trajectory path to brain targets in real time. Coordinates (X,Y,Z) were established for each target and 0.5 ul of diluted gadolinium was injected at each target using a 26 gauge needle to create a lesion. The center of the gadolinium lesion was identified on the post-operative MR images and coordinates (X′,Y′,Z′) were established. The precision of this system in bringing the needle to target (needle placement error) was calculated using the formula: error = $\sqrt{[(X - X')^2 + (Y - Y')^2 + (Z - Z')^2]}$.

Seventeen sites were targeted in the brain. Mean needle placement error for the caudate nucleus (n = 8), thalamus (n = 6), and midbrain (n = 3) was 1.52 ± 0.87(SD), 1.70 ± 0.77, and 2.67 ± 0.65 mm, respectively. The overall mean needle placement error for all target sites(n = 17) was 1.79 ± 0.87 mm. There was no statistically significant relationship between target depth and degree of error (p = 0.54). We conclude this stereotactic system has acceptable precision and may be utilized clinically for biopsying brain lesions in dogs.

CANCER STEM CELLS IN CANINE GLIOMAS: PRELIMINARY RESULTS IN A STUDY OF 17 CASES. Foradada L[1], Vidal E[2], Márquez M[2,3], Fondevila D[1,4], Rabanal R[1,4], Pumarola M[1,2,3,4]. [1]Centre de Biotecnologia Animal i Teràpia Gènica (CBATEG); [2]Priocat Laboratory, Centre de Recerca en Sanitat Animal (CReSA), UAB-IRTA; [3]Animal Tissue Bank of Catalonia (BTAC); [4]Department of Animal Medicine and Surgery, Veterinary Faculty Universitat Autònoma de Barcelona, Bellaterra, Barcelona, Spain.

Gliomas are the most common primary neoplasms of the canine central nervous system (CNS). In veterinary medicine, gliomas are classified (WHO, 1999) according to their morphologic characteristics as astrocytic, oligodendroglial and mixed tumours. In recent years, immunohistochemical studies have added new data, including new variants of glial tumours in animals (Koestner and Higgins, 2002). In human medicine, the WHO classification (2007) includes a grading scheme that is a ''malignancy scale'' correlated to the tumours clinical prognostic.

It is unclear whether these tumours result from the differentiation of a mature glial cell or from the transformation of an immature precursor. Recently cancer stem cell hypothesis has been proposed specially to explain the origin of undifferentiated and more malignant tumours in nervous tissue (Singh et al. 2004). In veterinary literature, it has been demonstrated the presence of cancer stem cells in canine osteosarcoma (Wilson et al. 2008) and glioblastoma (Stoica et al., 2009), and some authors have pointed out the use of dogs and cats for studying this field (Pang and Argyle 2009).

We are analyzing nervous tissue tumours of our database, reclassifying them using the human WHO's criteria and, looking for the presence of adult stem cells on these tumours. Several immunohistochemistry assays are being performed to identify different tumour cell populations. Here we present the preliminary results obtained in the study of canine gliomas.

Seventeen canine gliomas diagnosed in the Veterinary Pathology Diagnostic Service of the Autonomous University of Barcelona have been re-evaluated histopathologically using a semiquantitative scoring approach of the following criteria: growth pattern, vascular proliferation, cellular density, nuclear pleomorphism, mitotic index, presence of necrosis and presence of secretions. These are the criteria used in the human WHO's nervous system tumours classification.

For immunohistochemical characterization, the following antibodies were used:

- Olig2: a transcription factor involved in the differentiation of cells of the oligodendroglial lineage. In the human brain, Olig2 is specifically expressed in oligodendrocytes (Ikota et al. 2006, Prenser et al. 2007)
- Nestin: an intermediate filament used as a marker of neural progenitor cells (Schiffer et al. 2006)
- GFAP: a filamentous protein from the astrocytes cytoskeleton.

Olig2 and Nestin had never been tested in canine CNS before. The distribution of these markers has been previously studied in normal canine brains of different ages.

The histological parameters of the human WHO classification can be used to re-classify canine gliomas. Different subtypes of glial tumours can be identified.

In oligodendrogial tumours, Olig2 expression was observed with higher intensity in low grade tumours, whereas the expression was less prominent in the more anaplastic tumours. Nestin reactive cells were scarcely observed in low grade tumours and they increased in number in higher grade tumours.

Our preliminary results indicate that low grade oligodendrogliomas express mainly markers for well differentiated oligodendroglial cells, whereas high grade gliomas express markers for undifferentiated progenitor neural cell. These results concur with those published on human gliomas (Ikota et al. 2006) confirming the presence of stem cells mainly in undifferentiated gliomas.

We are developing a more extensive panel of cell markers -especially those related with stem cells and poorly differentiated neural cells, and other techniques (primary cell cultures and neurosphere formation, cell differentiation and quantification, and experimental inoculation on mice) to confirm the presence of stem like cells in canine gliomas and the cancer stem cell hypothesis. With these new studies we will be able to use the dog as a good animal model for trial therapies and outcomes in cases of natural gliomas.

TESTING EPIGENETIC CONCEPTS IN CANINE NEURO-ONCOLOGY I: METHYLATION OF DNA-REPAIR ENZYME O6-METHYLGUANIN-DNA-METHYLTRANSFERASE. L Matiasek[1], J Schlegel[2], M Starkey[1], L De Risio[1], T Flegel[3], K Baiker[4], K Matiasek[1]. [1]Animal Health Trust, Newmarket, UK; [2]Neuropathology Laboratory, Institute of Pathology, Technical University of Munich, Germany; [3]Small Animal Clinic, University of Leipzig, Germany; [4]Neuropathology, Institute of Veterinary Pathology, Ludwig-Maximilians University of Munich, Germany

The efficacy of alkylating agents in tumour control very much depends on the activity of DNA repair enzymes that are capable of counteracting the treatment-associated damage to the tumour cell genome. In turn, silencing of O6-methylguanine-DNA methyltransferase (MGMT) by promotor methylation has been reported to increase the sensitivity of human gliomas to chemotherapy. This study for the first time evaluated whether canine brain tumours present with different MGMT methylation states.

To date, eight primary brain tumours (3 intracranial meningiomas, 3 oligodendrogliomas, and 2 astrocytomas) had been subjected to methylation-specific polymerase chain reaction after cytosine to uracil conversion by sodium bisulphite pretreatment. Specific primers were designed for methylated and modified unmethylated sequences of the canine MGMT gene.

In this first run all three meningiomas and both astrocytomas presented with silenced and unmethylated MGMT genes. In oligodendrogliomas, on the other hand, the MGMT promotor was persistently methylated.

This investigation indicated that MGMT gene transcription may be partially suppressed in certain canine meningiomas and astrocytomas. In oligodendrogliomas of tested animals, only silenced MGMT genes were detected. In accordance to previous findings in humans and cell cultures, these results lend credence to the efficacy of alkylating agents as temozolomide and nitrosourea in control of

canine oligodendroglial tumours. Methylation-specific PCR, moreover, may be a valuable adjunct to tumour profiling in advance of individualised canine tumour therapy.

TESTING EpiGENETIC CONCEPTS IN CANINE NEURO-ONCOLOGY II: HISTONE ACETYLATION. K Matiasek, L De Risio, M Ortega Prieto, L Matiasek. Animal Health Trust, Newmarket, UK

Tight nucleosomal packing of DNA represses transcription, replication and repair but also its vulnerability to anticancer drugs. Wrapping of the strands around histones is one major mechanism of chromatin condensation. It is inversely correlated to the acetylation state of the histones. Accordingly, increased histone deacetylase (HDAC) levels have been identified in different human cancers. Whether DNA packing possibly impairs the efficacy of chemotherapy in canine meningiomas is hitherto unknown.

Thus, the present pilot study investigated the nuclear expression of acetylated and deacetylated forms of the main core histones H2A, H2B, H3 and H4 in 17 canine meningiomas after immunolabelling and DAB staining. Immunopositive cells were evaluated by a purpose-written ImageJ-cell counter plugin within identical, random-sampled fields. A canine squamous cell carcinoma served as positive control.

Expression of acetylated and deacetylated histones was noted in all meningiomas and control tissues. The profiles did not correlate with the histological subtype. The ratios of acetylated and deacetylated isoforms of H2A and H3 were contradictory to the values obtained after H2B and H4 labelling. On cell level, the deacetylated forms of H2B and, to a lesser degree, H3 were more prevalent in poorly differentiated cells of the positive control and cells undergoing mitosis.

Canine meningiomas to a very individual extent express different deacetylated subtypes of core histones. Whether these dogs may have benefited from adjuvant treatment with HDAC inhibitors cannot be clarified retrospectively. Further analysis of the survival rates and treatment protocols are required to shed light on the prognostic implication of the different histones before clinical trials can be recommended.

A COMPARATIVE STUDY OF CANINE AND FELINE MENINGIOMA CLASSIFICATION BASED ON WHO HISTOLOGICAL CLASSIFICATION SYSTEM IN HUMANS. M.T. Mandara, S. Pavone, B. Brunetti*, L. Mandrioli*. Department of Biopathological Science and Hygiene of Animal and Food Productions, University of Perugia, Italy; *Department of Veterinary Public Health and Animal Pathology, Alma Mater Studiorum, University of Bologna, Italy

Meningioma is the most common primary central nervous system (CNS) neoplasm affecting brain and spinal cord of dogs, and brain of cats. This tumour shares striking similarities to human meningioma in gross and histological appearance, and biological behaviour. The current domestic animal WHO histological classification system of meningiomas categorized them into 2 major groups: benign (meningothelial, fibroblastic, transitional, psammomatous, angiomatous, papillary, granular, myxoid), and anaplastic tumours. In humans, a major component of WHO histological classification system is the grouping of tumours into 1 of 3 histological grades (benign, atypical, anaplastic). The most recent human WHO classification (2007) recognizes histological variants other than clear cell, chordoid, papillary, and rhabdoid, with a mitotic index < 4 mitoses/10 HPF, as benign meningiomas (grade I). Diagnostic criteria for atypical meningiomas (grade II) are mitotic index (≥ 4 mitoses/10 HPF); or ≥ 3 of the following criteria: patternless sheets, increased cellularity, high N/C ratio, macronucleoli, spontaneous necrosis; or brain invasion. The cytological features included in anaplastic meningiomas are a high mitotic index (> 20 mitoses/10 HPF) and a frank anaplasia (grade III). The aim of this study was to compare human and domestic animal WHO histological classification system of meningioma to identify possible critical points in applying human WHO classification to canine and feline meningiomas.

Selected paraffin embedded tissues from 57 canine and 38 feline tumours recorded as meningiomas were used in this study. Based on the current domestic animal WHO histological classification system they had been achieved as benign (38 canine, 34 feline) and malignant (19 canine, 4 feline). All these meningiomas were graded according to the criteria of the latest human WHO international histological classification of CSN tumours as benign (grade I), atypical (grade II) or anaplastic (grade III).

Based on human WHO classification system, histological grading in the dogs indicated 27/57 benign (grade I) (47.3 %), 26/57 atypical (grade II) (45.6 %) and 4/57 anaplastic (grade III) (7.0%) tumours. Eleven tumours recorded as benign meningiomas were graded as grade II, 15 malignant as grade II and 4 malignant as grade III. Two canine meningiomas were classified as chordoid type and graded as grade I. Eight canine meningiomas were classified as papillary; six of them were graded as grade I, the remaining two cases were graded as grade II. In cats histological grading identified 27/38 benign (grade I) (71.05%) and 11/38 atypical (grade II) (28.9%) tumours. In the cats, no anaplastic meningiomas were identified. Twenty-six tumours classified as benign were graded as grade I while the remaining eight benign-classified tumours as grade II, two malignant as grade I and two malignant as grade II. Nervous tissue invasion occurred in 13 cases. In one case the infiltration was the sole parameter to identify grade II. In the remaining 12 cases the infiltration was observed along with other malignancy criteria. On the other hand, all cases graded as grade II (atypical), lacking of the main atypical meningioma criteria, showed constant pattern loss. In eight cases the infiltration involved the adjacent dura mater or bone tissue, but not the nervous tissue; six of them were classified as grade I and the remaining two cases as grade II based on pattern loss, high N/C ratio, and macronucleoli.

Based on our results, we believed mitotic index ≥ 4 mitoses/10 HPF and brain invasion are sufficient criteria to identify grade II meningiomas and we suggested patternless sheets alone could be assumed as a criterion to attribute grade II to a meningioma. Despite data from human WHO classification, in this study canine papillary meningioma was considered belonging to grade I as well as grade II and canine chordoid meningioma was graded as grade I, probably due to not available data on nervous tissue invasion. Interestingly, in the cats, meningiomas of grade III were not detected, confirming the less aggressive behaviour of feline meningioma and suggesting no current grading is applicable to the feline meningioma. Moreover, our results confirmed a higher incidence of canine atypical (grade II) and anaplastic (grade III) meningioma than in humans. Because of its inherent advantages, it could be proposed that the system used in humans be adapted universally to canine meningiomas with some variations if supported by further in-depth studies determining clinical outcome and long-term prognosis.

CEREBRAL NECROSIS FOLLOWING HYPOFRACTIONATED RADIOTHERAPY FOR CANINE INTRACRANIAL TUMORS: A MAGNETIC RESONANCE IMAGING AND PATHOLOGICAL STUDY. Alejandro Luján Feliu-Pascual[1], Ruth Dennis[2], Sue Murphy[2], Luisa De Risio[2] and Kaspar Matiasek[2].. [1]Clínica Veterinaria La Merced, Calpe, Spain and [2]The Animal Health Trust, Newmarket, UK.

Radiotherapy for canine intracranial tumors is being used with increased frequency; however, cerebral necrosis as a complication of this treatment modality has been poorly documented. Moreover, information such as reason for deterioration or follow-up diagnostic imaging is lacking in many radiotherapy reports. The objective of this retrospective study was to report the Magnetic Resonance (MR) and pathological features of cerebral necrosis following radiotherapy.

The clinical and pathological records of the AHT (1994-2008) were searched for diagnosis of cerebral necrosis following radiotherapy of canine intracranial tumors. Ten cases were identified; four of them with post-mortem confirmation. In all cases pre-treatment MR images identified an intracranial mass. In three cases surgical debulking preceded external beam radiotherapy. Five weekly fractions were administered to all cases. All experienced clinical deterioration following initial improvement. Investigations included MR study (9 cases) and/or post-mortem examination

(4 cases). The original tumor volume was reduced in 8 cases and unchanged in one. A second tumor was identified in a different location within the cranial vault in two cases. Magnetic resonance imaging showed diffuse white matter hyperintensities on T2-weighted images and multifocal T2* gradient echo hypointensities in the irradiated areas in 9 cases, not restricted to the peritumoral regions. Pathological findings included extensive areas of cavitation characterized by coagulative necrosis, and multifocal hemorrhages of variable size.

Cerebral radionecrosis should be considered as a cause of deterioration following radiation therapy for canine intracranial tumors. Moreover, follow-up advance imaging should be performed in all cases to identify the underlying reason for deterioration.

SPHENOID BONE INFILTRATION IN A DOG WITH DISSEMINATED MAST CELL TUMOUR. E. Beltran, A. de Stefani, J. Stewart, L. De Risio, V. Johnson. The Animal Heath Trust, Newmarket, Suffolk, UK

Mast cell tumours are found in most organs and tissues with variable biologic behaviour in dogs. This case illustrates the clinical and magnetic resonance imaging (MRI) findings in a dog with disseminated mast cell tumour infiltrating the sphenoid bones.

A 6 year-old male neutered Greyhound presented with a three-day history of acute onset of blindness. General physical examination was normal. Neurological examination revealed mild disorientated mental status, absent menace response in both eyes, bilaterally decreased vestibulo-oculocephalic reflexes and absent pupillary light reflex, direct and consensual, in both eyes. An electroretinogram indicated normal retinal function in both eyes. A lesion involving the middle cranial fossa and particularly the optic chiasm was suspected. Haematology and serum biochemistry were normal except decreased urea (1.2 mmol/l). MRI of the head revealed increased signal intensity on T2 weighted images of the sphenoid bones and loss of their normal internal architecture. Cerebrospinal fluid analysis revealed normal nucleated cell count with the cell population consisting mostly of eosinophils. Abdominal ultrasound revealed hepatosplenomegaly and mesenteric lymphadenopathy. Fine needle aspirates were taken from the jejunal lymph node and the spleen and the results were consistent with multicentric mast cell tumour. The owner refused any treatment and the dog was euthanatised. Post-mortem examination confirmed disseminated mast cell tumour affecting multiple organs, including the sphenoid bones.

To our knowledge this is the first case describing MRI features of disseminated mast cell tumour affecting the sphenoid bones and causing acute onset of blindness in a dog.

ANTEMORTEM DIAGNOSIS OF INTRACRANIAL AND OCULAR METASTASES OF A MIX MALIGNANT MAMMARY TUMOR. I. Srugo[1], I. Aroch[1], A. Berkowitz[1], N. Edery[2], O. Chai[1], and M.H. Shamir[1]. [1]KORET school ofVeterinary Medicine, Hebrew University of Jerusalem, Israel. [2]Department of Pathology, Kimron Veterinary Institute, Bet Dagan, Israel.

A 9-year old, spayed, female cocker spaniel was presented with three weeks of progressive blindness, exophthalmos and circling. Surgical excision of a recurrent malignant mixed mammary tumor was performed two months earlier. Neurological examination revealed depression, circling and leaning to the left, head turn to the same side and ambulatory tetraparesis with mild continues proprioception deficit and hypermetria of all four limbs. Bilateral, dilated, non responsive pupils, severe corneal edema and elevated intra ocular pressure (IOP) were also recorded. Neuro-anatomic localization suggested a multi-focal lesion involving the forebrain, cerebellum and both eyes.

Post contrast computed tomography (CT) revealed enlargement of the left lateral ventricle, an intra-axial enhancement (7–10 mm) in the Diencephalon - Mesencephalon junction and cerebellar meningeal enhancement. Cerebrospinal fluid (CSF) had a markedly increased total nucleated cell count (1550 cells/μL, normal $\leq$ 3 cells/μL) and elevated protein concentration (49 mg/dL, normal $\leq$ 25 mg/dL).

Cytology of the CSF revealed high number of markedly large (20–200 microns) round to polyhedral cells of varying size, containing large nuclei with loose-chromatin and 2–5 nucleoli. These cells showed marked anisocytosis and anisokaryosis and extensive cytoplasmic vacuolation. Cytology of the aqueous humor (AH) samples showed a high celullarity of cells similar to those in the CSF. Based on these findings a tentative diagnosis of brain and ophthalmic metastases of a malignant mammary tumor was made and the dog was euthanized.

Gross pathology showed multiple nodular masses of variable size (range 0.3 cm to 2 cm) in the mammary lymph node, lungs and brain. Histopathology of all masses showed dense celullarity of neoplastic epithelial cells arranged in nodular to solid sheets that were cytokeratin-positive. The concurrent presence of these similar epithelial cells in the mammary lymph nodes, lungs, CNS and eyes with the history of a malignant mammary tumor led to the diagnosis of a malignant mammary tumor with distant metastasis. To the best our knowledge, this is the first report of an *antemortem* diagnosis of CNS mammary tumor metastases based on cytological evaluation of the CSF.

PROGNOSTIC VALUE OF AN IMAGING SEVERITY INDEX (ISI) FOR ROSTROTENTORIAL MASS LESIONS IN THE DOG. Roberto José-López, Mark Lowrie, Inés Carrera and Jacques Penderis. Faculty of Veterinary Medicine, University of Glasgow, Bearsden Road, Glasgow, UK.

The calvarium represents a fixed non-distensible volume, with the elements (including the brain) contained within having a limited adaptive capacity to increased volume (volume buffering capacity). Once this limited compensatory mechanism is exhausted (primarily mediated through CSF and blood displacement, decrease of extracellular fluid space and dura mater stretching) the intracranial contents exceed the volume of the calvarium and a rapid rise in intracranial pressure (ICP) occurs. Direct ICP measurement is difficult in dogs and indirect prediction is based on serial neurological examinations, identification of papilloedema or the modified Glasgow coma scale (GCS). While these indirect measures are useful once raised ICP is present, they are less useful in early intracranial volume increase, before volume buffering capacity is exhausted.

Identification of early changes in volume buffering capacity is possible by magnetic resonance imaging (MRI) and may be useful for prediction of patients at risk of developing raised ICP. Cases with mass lesions usually have a more gradual intracranial volume increase and therefore greater exhaustion of compensatory mechanisms. The aim of this study was to develop an imaging severity index (ISI) for rostrotentorial mass lesions and correlate this with clinical severity to assess its usefulness as an early predictor of volume buffering capacity saturation.

Sixty-six MRI studies and corresponding medical records were reviewed from dogs with solitary rostrotentorial mass lesion presented to the University of Glasgow Small Animal Hospital from November 2004 to June 2009. On the basis of the clinical abnormalities identified in rostrotentorial mass lesions in previously published studies, each dog was assigned a clinical severity score. The modified GCS was calculated for each dog. The ISI score was determined for each dog on the basis of: 1) The ratio of mass lesion volume to total intracranial volume. 2) Using a subjective scoring system: peri-lesional oedema, compression of the subarachnoid CSF spaces, presence of obstructive hydrocephalus, foramen magnum herniation and development of syringohydromyelia. 3) Using a quantitative scoring system (with normal ranges established on the basis of 30 normal canine brain MRI studies): rostral-caudal brain shift, midline brain shift, caudal transtentorial herniation and cerebellar shape change. Statistical analysis was performed using GraphPad Prism and Excel software.

Spearman's rank correlation coefficient identified highly significant correlation between the clinical severity index and modified GCS and ISI ($r = 0.7499$ and $r = 0.4618$; $P < 0.0001$ for both), with better discrimination of cases with early saturation of volume buffering capacity by ISI, while GCS correlates better with more severe clinical severity scores.

ISI strongly correlates with clinical severity and is useful in dogs with rostral fossa mass lesions for identifying exhaustion of intracranial volume buffering capacity and risk of raised ICP.

LOW FIELD MAGNETIC RESONANCE IMAGING (MRI) IN DOGS WITH AND WITHOUT SIGNS OF DISC ASSOCIATED WOBBLER SYNDROME (DAWS): A RANDOMIZED, BLINDED STUDY. S De Decker[1], IMVL Gielen[1], L Duchateau[1], J Lang[2], R Dennis[3], N Corzo-Menéndez[4], HJJ van Bree[1], I Van Soens[1], D Binst[1], T Waelbers[1], LML Van Ham[1]. [1]Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium, [2]Vetsuisse Faculty Bern, Bern University, Suisse, [3]Animal Health Trust, New Market, United Kingdom, [4]Davies Veterinary Specialists, Hertfordshire, United Kingdom.

Previous MRI studies have demonstrated spinal cord compression in clinically normal subjects. These compressions have the potential to cause false positive clinical interpretations. The purpose of this study was to investigate the intra –and interobserver variability of MRI and the prevalence of false-positive and negative interpretations.

MRI studies of dogs (n = 21) with DAWS were randomly mixed with those of age-matched clinically normal Doberman Pinschers (n = 12) and Foxhounds (n = 11) and were presented to 4 independent board-certified radiologists. MRI was performed with a 0.2 Tesla magnet. Sagittal and dorsal T1 and T2 weighted images (WI) of the entire cervical spine and transverse T1 and T2 WI from C4 to T1 were obtained. On T2 WI, disc degeneration was classified as normal, partially or completely degenerated. Ventral and dorsal compression was classified as normal, partial or complete subarachnoidal space compression and spinal cord compression. Vertebral body abnormalities were assessed on sagittal T1 WI. Abnormal intraparenchymal signal intensity (ISI) changes were classified based on the surrounding spinal cord parenchyma on T1 and T2 WI. New bone formation was described as present or absent. Finally, the observers had to judge if the MRI studies were suspected to come from a clinically affected or clinically normal dog. Kappa (κ) and weighted κ statistics were performed to assess intra- and interobserver variability.

Overall, there was very good intraobserver agreement in rating disk degeneration (κ = 0.87), disc associated compression (κ = 0.81), ISI (κ = 0.84), and vertebral body abnormalities (κ = 0.89). There was good agreement in rating dorsal compression (κ = 0.77) and new bone formation (κ = 0.73). Overall there was good interobserver agreement in rating disk degeneration (κ = 0.67) and vertebral body abnormalities (κ = 0.89). There was moderate interobserver agreement in rating disc associated compression (κ = 0.56), dorsal compression (κ = 0.51), ISI (κ = 0.55), and new bone formation (κ = 0.73). If for each dog, a consensus opinion for suspected clinical status was obtained when at least 3 of the 4 observers agreed that the severity of the MRI features would probably cause clinical signs, 2 of the 21 (9.5%) patients and 4 of the 23 (17.4%) clinically normal dogs were erroneously categorized as clinically normal and clinically affected, respectively.

The results of this study suggest that some variability exists between observers and that MRI interpretation of the cervical spine can lead to false positive and false negative results.

TRANSCRANIAL MAGNETIC STIMULATION (TMS) IN DOBERMANN PINSCHERS WITH AND WITHOUT SIGNS OF DISC ASSOCIATED WOBBLER SYNDROME (DAWS): USEFUL TOOL TO DIFFERENTIATE BETWEEN CLINICALLY RELEVANT AND IRRELEVANT SPINAL CORD COMPRESSION? S De Decker, I Van Soens, L Duchateau, IMVL Gielen, HJJ van Bree, D Binst, T Waelbers, LML Van Ham. Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

Previous work has revealed the occurrence of cervical spinal cord compression in clinically normal Dobermann Pinschers with subsequent difficulties in clinical interpretation of MRI studies. The purpose of this study was to evaluate the usefulness of transcranial magnetic motor evoked potentials (TMMEPs) to differentiate between clinically relevant and irrelevant cervical spinal cord compressions seen on MRI.

After sedation with acepromazine and morphine, TMMEPs were recorded in 33 Dobermann Pinschers from the extensor carpi radialis (ECRM) and cranial tibial (CTM) muscles. Onset latencies and peak-to-peak amplitudes were measured from the TMMEPs. Subsequently, the dogs underwent low field MRI (0.2 T) to evaluate the presence and severity of spinal cord compression. After TMS and MRI, the dogs were classified in 3 different groups; group 1, clinically normal dogs without cervical spinal cord compression on MRI (n = 11); group 2, clinically normal dogs with cervical spinal cord compression on MRI (n = 6); group 3, dogs with cervical spinal cord compression on MRI and corresponding clinical signs of DAWS (n = 16). Spinal cord compression was defined as complete compression of the subarachnoid space with deviation or distortion of the spinal cord. Severity of spinal cord compression was classified according to the degree of spinal cord deformation, displacement, and parenchymal changes into 4 gradations.

There was a significant difference in ECRM and CTM onset latencies between the 3 different groups overall and between group 3 and the two other groups separately. There was no significant difference in ECRM and CTM onset latencies between groups 1 and 2. There was a significant difference in CTM peak-to-peak amplitudes between the 3 different groups overall and between group 3 and the two other groups separately. There was no significant difference in ECRM peak-to-peak amplitudes for all the different combinations and for CTM peak-to-peak amplitudes between groups 1 and 2. There was a significant correlation between severity of spinal cord compression and ECRM onset latencies, CTM onset latencies, and CTM peak-to-peak amplitudes (r = 0.424, r = 0.418, and r = – 0.418, respectively).

The results of this study suggest that TMS is a useful diagnostic tool to differentiate between clinically relevant and irrelevant cervical spinal cord compressions seen on MRI. Especially, the recorded onset latency seems to be a reliable parameter.

TEACHING VETERINARY NEUROLOGY: EXPERIENCE WITH AN INTER-FACULTY eLEARNING ELECTIVE COURSE. M. Koch[a], A. Tipold[a], M. Fischer[b], M. Vandevelde[c], J.P. Ehlers[a]. [a]University of Veterinary Medicine Hannover, Germany; [b]University of Witten-Herdecke, Germany; [c]Vetsuisse Faculty of Bern, Suisse

eLearning is an ideal supplement to classroom education. The University of Veterinary Medicine Hannover and the Vetsuisse Faculty of Bern arranged a collaborative pilot project "Neuroimmunology" as an elective course for students. In neurology classes neuroimmunology is not taught intensively and interested students should be stimulated to learn more details.

Three case studies describing the different reaction models of the immune system in the nervous system were chosen: meningitis, encephalitis, neuritis, invasion of neutrophils, immigration of lymphocytes, molecular mimicry reaction. Typical cases with inflammatory disease were presented with videos, laboratory findings, neuropathology and pathogenesis using the CASUS®-system. Students were encouraged to find the diagnosis in an interactive way. In addition, the cases were evaluated by a group of residents in neurology and internal medicine.

Attendants to the elective course were introduced in a local kick-off meeting. In the following three weeks the three cases could be studied independently on the internet. In between discussions took place in synchronous online-meetings in a virtual classroom (netucate iLinc™) and asynchronously in a phpBB-board (www.foren4vet.de). Technical support was provided by telephone and email. The evaluation of the course was accomplished with a questionnaire. Furthermore data of the CASUS®-database (study time and success rate) were collected.

Altogether 24 students from Hannover and 14 students from Bern took part in the course. In the first meeting in the virtual classroom students could discuss questions about the cases personally with other attendants and the teacher. In the second meeting the virtual classroom and the lecture hall were combined, so students could choose their preferred medium. The evaluation results show a great acceptance from the students for the new course format (Likert scale, rates from 1 = yes, good to 6 = no, bad). The course received grades from 1,0–1,6; the cooperation between students and teachers of different universities was accepted. The success rates of the CASUS®-database varied from 70 to 85% with an average of 78,4%. In conclusion, CASUS seems to be a very efficient tool to stimulate students to self learning. Success rate is evaluated electronically and therefore not time consuming for teachers.

WEST NILE VIRUS OUTBREAK IN ITALY: CLINICAL FINDINGS IN 10 HORSES. G. Gandini[1], A. Gallucci[1], L. Mandrioli[2], A. Spadari[1], M Rosati[1], F. Dondi[1], N. Romagnoli[1], G. Bettini[2]. [1]Veterinary Clinical Dpt, University of Bologna, Italy. [2]Dpt of Veterinary Public Health and Animal Pathology, University of Bologna, Italy.

The purpose of this report is to describe the neurologic signs, and clinicopathologic abnormalities in 10 horses affected by WNV encephalomyelitis, referred to the Veterinary Clinical Department of the Faculty of Veterinary Medicine of the University of Bologna during the Italian outbreak in 2008. All horses had video recorded neurological examination, documenting the clinical signs.

Diagnosis of WND in all horses was made considering the course of the disease and the positive result of the ELISA test for the detection of antibodies (IgG) against WNV. All the horses underwent general physical and neurological examination, as well as haematobiochemical evaluation. In two more severely affected horses, cerebrospinal fluid (CSF) was analyzed and, after euthanasia, brain and spinal cord were removed for neuropathologic evaluation.

The most frequently affected breed was the standardbred (5 cases). All the horses lived in an environment severely infested by mosquitoes and they all had an acute onset of generalized illness consisting mainly in anorexia and depression. Fever was not consistently reported by the referring veterinarians. Neurologic signs varied markedly in severity from the onset to the time of the examination.

Affected horses had different patterns of Central Nervous System (CNS) signs related either to the different neuroanatomical involvement and to the degree of severity. The most striking signs were related to gait abnormalities, recorded in all horses. The two more severely affected cases had severe tetraparesis, in one case not ambulatory and leading to permanent recumbency. The other 8 horses had spinal ataxia, paresis of 2–4 limbs and central vestibular involvement in one case. Besides the recumbent horse, showing bilaterally absent menace reaction and ventral strabismus, cranial nerves tests were otherwise normal in all subjects. Change in behaviour was noticed in 5 cases and consisted mainly in hyperexcitability, depression and aggressiveness.

Different protocols of symptomatic anti-inflammatory therapy were performed in all horses (including Flunixin meglumine; corticosteroids and DMSO administration). The two more severely affected horses, despite treatment, worsened to permanent recumbency and were euthanized for humane reasons. The CSF analysis revealed in both cases a severe mononuclear pleocytosis (respectively: 190 cell/μl 97% mononuclear and 272 cell/μl, 84% mononuclear). All the other horses improved dramatically in few days and recovered without consequences.

In our experience, WND showed the same neurologic patterns as those described in the literature. Despite the limitation of the small number of cases described, apparently WND affected mainly the spinal cord. Compared to USA outbreak, muscle fasciculations were less frequently observed. Poor prognosis was related to permanent recumbency. Mild to moderate signs responded well to therapy and lead to quick improvement and complete recovery.

WEST NILE VIRUS OUTBREAK IN ITALY: PATHOLOGICAL FINDINGS IN TWO HORSES. L. Mandrioli[1], G. Bettini[1], M. Morini[1], A. Gallucci[2], R Biserni[2], A. Spadari[2], G. Gandini[2]. [1]Dpt of Veterinary Public Health and Animal Pathology, University of Bologna, Italy; [2]Veterinary Clinical Dpt, University of Bologna, Italy.

The aim of this presentation is to describe the gross and histological findings in two horses affected by West Nile Disease (WND) examined during the WND outbreak which occurred in Italy in the 2008 late summer. Both horses had been euthanized for humane reasons because of the severity of neurological signs. Complete postmortem examination was carried out immediately after euthanasia. Representative tissue samples of the CNS and major organs were fixed in 10% buffered formalin, processed for histology and stained with hematoxylin and eosin, periodic acid-Schiff, Luxol Fast blue and cresyl violet. Furthermore, samples of fresh CNS tissue were collected for virology.

The first horse was a 7-year-old female Selle Français with severe tetraparesis, mild changes in mental status and cranial nerves and permanent recumbency. Gross lesions were limited to the spinal cord; multifocal pinpoint hemorrhages were evident especially in the thoracic and lumbar tracts; in the lumbosacral segments the gray matter showed an asymmetric discoloration of the ventral horns, associated with haemorrhagic infarction.

Histologically, inflammatory and degenerative lesions suggestive of viral infection were evident in all the CNS tracts (brain and spinal cord). Notably, cerebral cortex, diencephalon and brain stem showed multifocal perivascular cuffs formed almost exclusively by small lymphocytes and at lesser extent by histiocytes and small foci of hemorrhages; capillaries lumina were dilated and hyperemic. The neuropil showed marked neuronal degeneration, chromatolysis and cell shrinkage; there were also multifocal microglial nodules, containing rare neutrophils admixed with glial cells. The spinal cord prompted a pattern of degenerative and inflammatory changes similar to those detected in the brain neuropil, being the thoracic tract the most severely affected: chromatolysis, cell shrinkage, neuronophagia and lymphocytic perivascular cuffing were evident in the gray matter; furthermore, there were foci of extravasated erythrocytes dissecting myelin fibers in the white matter, and frequent axonal swellings (spheroid formation). The diagnosis was severe, subacute, multifocal, not suppurative polyoencephalomyelitis. RT-PCR from CNS samples was positive for WND virus.

The second horse was a 19-year-old Appaloosa gelding with severe tetraparesis. Post mortem examination demonstrated multifocal pinpoint hemorrhages in the gray matter of thoracic spinal cord segments. The skull opening revealed some white, compact, nodular thickenings of the dura mater, ranging from 1 to 5 mm in diameter, similar to the age-related Pacchioni collagen granulations. In the extraneural tissues there were signs of moderate cardio-respiratory insufficiency. At histology, thoracic tracts of the spinal cord showed multifocal perivascular hemorrhages and focal influx of lymphocytes within the gray matter. The histological diagnosis was mild, focal poliomyelitis and multifocal spinal hemorrhages. CNS samples were positive for WND virus.

In both horses the histological lesions were suggestive of viral encephalomyelitis, and the role of WNDV was confirmed by molecular analysis. The different clinical presentation, showing intracranial involvement only in one horse, was confirmed on neuropathololgic examination. In the first case, the changes were distributed in both the brain and the spinal cord, while in the second horse only the spinal cord was affected histologically. Noteworthy, this different pattern did not match the severity of neurological signs. These findings confirm the observations from others, as for the prevalence of spinal cord lesions in WND, as for the poor correlation between the severity of clinical and histological signs.

Beside the inflammatory infiltrate, the likely basis of neurological symptoms relies on the neuronal degenerative changes; however, it is still uncertain if these changes are triggered by neurotropic cytopathic properties of the virus or by the inflammatory reaction itself.

MULTI-SYSTEM NEURONAL DEGENERATION IN A FAMILY OF ATAXIC NORWEGIAN BUHUND. A de Stefani, K Matiasek, O Forman, L De Risio. Animal Health Trust, Newmarket, Suffolk, UK.

Multi-system neuronal degenerations are familial progressive neurodegenerative disorders that selectively affect specific neuronal cell populations. Historically reported by breeders, a large proportion of Norwegian Buhund in the UK seems to suffer from a progressive and ultimately fatal form of ataxia. We are describing here the clinical and histopathological findings in 3 Norwegian Buhunds presented at our institution and diagnosed with multi-system neuronal degeneration. A litter of 5, 12-week old, Norwegian Buhunds was presented to us. One male and one female pup were showing progressive all four limbs ataxia, head tremors, truncal ataxia and occasional loss of balance. One other related pup was presented 6 years previously aged 16 weeks with identical but more pronounced clinical signs. Based on the clinical presentation a cerebellar neurolocalisation was suspected.

The 3 affected pups underwent extensive investigations including blood work, brain MR imaging, CSF analysis and PCRs for *Toxo-*

plasma gondii, Neospora caninum and Canine Distemper Virus, urinary metabolic screening and BAER. A skin biopsy was also obtained for fibroblast culture in 2 pups. No abnormalities were identified on the above tests. On breeder request the affected pups were humanely euthanised and complete *post mortem* performed. Microscopic assessment revealed mild ongoing Punkinje cell degeneration with early Bergmann's gliosis affecting the cerebellar cortex diffusely. Neuronal degeneration was also noted in the pre-cerebellar olivary nuclei, various pontine nuclei and throughout the tegmentum.

DNA was collected from all the 5 pups in the litter. Pedigrees from the affected and non-affected related dogs were also collected. Preliminary pedigree analysis suggests an autosomal recessive type of inheritance. Genetic and complete pedigree analysis is in progress.

CLINICAL AND TOPOGRAPHIC MAGNETIC RESONANCE IMAGING CHARACTERISTICS OF SUSPECTED THALAMIC INFARCTS IN 18 DOGS. [1]Rita Gonçalves, [2]Ines Carrera, [3]Laurent Garosi, [1]Peter M Smith, [1]J. Fraser McConnell, [2]Jacques Penderis. [1]Department of Veterinary Science, Small Animal Teaching Hospital, University of Liverpool, Leahurst, Chester High Road, Neston CH64 7TE, UK; [2]Institute of Comparative Medicine, Faculty of Veterinary Medicine, University of Glasgow, Glasgow, G61 1QH; [3]Davies Veterinary Specialists, Manor Farm Business Park, Higham Gobion, Hertfordshire SG5 3HR, UK

Cerebrovascular disease results from pathological processes affecting the intracranial blood supply. In human medicine, thalamic infarction is associated with well described clinical syndromes which correlate with lesions affecting defined thalamic regions. Our aim was to describe the clinical presentation and topographic magnetic resonance imaging (MRI) characteristics of suspected thalamic infarctions in the dog.

The medical records and MRI of the brain of dogs with acute-onset, non-progressive signs of brain dysfunction and MRI characteristics compatible with thalamic infarction were reviewed retrospectively.

18 dogs met the inclusion criteria. Topographically the lesions could be grouped in 4 thalamic regions: paramedian (8/18), extensive dorsal (5/18), ventrolateral (3/18) and ventromedial (2/18). Paramedian lesions resulted mainly in signs typical of vestibular dysfunction. Extensive dorsal lesions were associated with vestibular ataxia, circling and contralateral menace response deficits. Both ventromedial and ventrolateral lesions resulted in circling and contralateral proprioceptive deficits. In several patients, other regions than the thalamus were also affected: 5 extended into the midbrain; 6 extended to the internal capsule; 2 dogs had a second lesion in the cerebellum. MRI findings were similar to those previously described. Nonetheless, post-contrast T1-weighted images helped estimate the timing of infarction: lesions $\leq$ 7 days mainly presented peripheral enhancement, whilst lesions $\geq$ 8 days presented mainly parenchymal enhancement that seemed to disappear with time.

Four clinical syndromes were indentified in association with thalamic infarction although there was variation in the clinical presentation observed within these different syndromes, most likely as the lesions were not confined to specific nuclear boundaries. Understanding the likely neurological deficits associated with each should facilitate more thorough evaluation of dogs presenting with cerebrovascular disease.

EFFECT OF MIDDLE EAR EFFUSION ON THE BRAINSTEM AUDITORY RESPONSE OF CAVALIER KING CHARLES SPANIELS. T. R. Harcourt-Brown, J. E. Parker, N. D. Jeffery. Department of Veterinary Medicine, University of Cambridge, Cambridge, UK

The purpose of this study was to investigate the effect of middle ear effusion on the Brainstem Auditory Evoked Reponses (BAER) of cavalier king charles spaniels. BAER's were obtained from dogs following Magnetic Resonance (MR) imaging screening for syringomyelia. Middle ear effusion was diagnosed if the auditory bulla was completely filled with material that was isontinense to brain parenchyma on T1 weighted images and hyperintense on T2 weighted images. Dogs with otitis externa were excluded from the study.

BAER's were obtained at sound intensities ranging from 10 to 100 dB nHL (normal hearing level). The BAER threshold was determined for each ear as the last trace that showed a recognisable wave V. The latency of wave V was recorded for each intensity where it was identified and the interwave latency between waves I and V was calculated at 90 dB nHL.

Twenty three dogs were included in the study. Each dog's hearing was considered normal by their owner. The median BAER threshold was 60 dB for ears with effusion and 30 dB for those without. The proportion of ears with abnormal BAER thresholds ($>$ 30 dB nHL) was greater for ears with effusion (11/16) than those without (8/30) (Fishers exact test, $p = 0.011$). Severity of hearing loss for ears with effusion was calculated by linear regression of wave V latencies to be 23 dB (95% confidence 18 to 31 dB). The mean interwave latency between waves I and V at 90 dB for ears with and without effusion was not significantly different (Students t-test, $p > 0.05$).

These data show that middle ear effusion is associated with conductive hearing loss of 21–30 dB in affected ears.

VOLUMETRIC ANALYSIS OF BRAIN PARENCHYMA WITHIN THE CAUDAL FOSSAE OF CAVALIER KING CHARLES SPANIELS. C Driver[1], C Rusbridge[2], H Cross[1], HA Volk[1]. [1]The Royal Veterinary College, London, UK; [2]Stone Lion Veterinary Hospital, Goddard Veterinary Group, London, UK.

Chiari-like malformation (CM) and syringomyelia (SM) is a debilitating disease complex recognized in the Cavalier King Charles Spaniel (CKCS). Mesoderm insufficiency during embryogenesis has been suggested as the pathogenesis of Chiari type-I malformations in humans leading to a small posterior fossa but a normally developed hindbrain. No volumetric evidence exists regarding the role of hindbrain volume within the caudal fossa in the development of SM in dogs.

Magnetic resonance (MR) images of 59 CKCS with CM and no other systemic disease were retrospectively reviewed. T2 weighted transverse images were exported to medical imaging software (Mimics v12.0, Materialise n.v, 2008) and volumetric analysis was performed based on three-dimensional reconstruction with masks from individual slices. Volumes of hindbrain parenchyma were analyzed as percentages of caudal fossa volume and caudal fossa volume was analyzed as a percentage of total cranial cavity volume. The volume of the ventricular system was recorded as a percentage of total parenchymal volume. If SM was present, syrinx size was measured from T2 weighted MR images from the maximal dorsoventral dimension within the cervical spine.

SM was present in 40/59 (68%) dogs. All data was normally distributed. There was no significant (t-test, $p = 0.702$) age difference between dogs with (61.2 ± 33.8 months; 6.8–128.9) or without SM (57.4 ± 37.1 months; 3.9–122.8). Caudal fossa percentage of the total cranial cavity volume did not differ significantly (t-test, $p = 0520$) between dogs without ($14.4 \pm 1.5\%$) or with ($14.9 \pm 1.3\%$) SM. However, there was a significant difference ($p = 0.002$) between the two groups looking at hindbrain parenchyma percentage of the caudal fossa ($86.7 \pm 4.1\%$ and $89.9 \pm 1.67\%$ respectively). Furthermore, in the SM group a significant positive association was found between the hindbrain parenchyma percentage and syrinx size (spearman $r = 0.437$). No significant difference ($p = 0.164$) was found between the two groups for ventricular volume ($5.30 \pm 7.68\%$ and $7.73 \pm 5.36\%$ respectively), however when a syrinx was present a strong positive correlation was found between ventricular and syrinx size (spearman $r = 0.500$).

This work supports recent evidence that caudal fossa size is not associated with SM, but that overcrowding of the caudal fossa leads to SM and may be caused by mesoderm insufficiency. The association between ventricle and syrinx dimensions supports the theory that SM develops as a result of altered CSF dynamics.

GENERALIZED MYOKYMIA AND NEUROMYOTONIA IN JACK RUSSELL TERRIERS: A CLINICAL AND ELECTROPHYSIOLOGICAL STUDY. A.E. Vanhaesebrouck[1], L. Poncelet[4], I. Van Soens[1], L. Duchateau[2], H.C. Schenk[5], I. Polis[1], S. Bhatti[1], S. Diels[3], L. Van Ham[1]. [1]Dept. of Medicine and Clinical Biology of Small Animals, [2]Dept. of Physiology and Biometrics, Faculty of Veterinary Medicine, [3]Dept. of Medical Imaging and Orthopedics of Small Animals, Ghent University, Belgium; [4]Dept. of Anatomy and Embryology, Faculty of Medicine, Free University of Brussels, Belgium, [5]Dept. of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

Generalized myokymia/neuromyotonia (NMT) in Jack Russell Terriers (JRT) is characterized by vermicular muscle contractions and episodic stiffness, respectively. This entity closely resembles the peripheral nerve hyperexcitability syndrome in humans. In JRT, NMT is commonly associated with hereditary ataxia (HA).

A prospective case-control study was designed to compare clinical and electrophysiological characteristics in a group of 8 JRT cases affected by NMT with a control group of 8 healthy JRT. The anaesthetic protocol was standardized in both groups during electrodiagnostic procedures.

An extensive workup of blood and urine, including organic and amino acid profile, as well as CSF, revealed an increase of muscle enzymes in half of the affected dogs. Electromyography typically showed myokymic and/or neuromyotonic discharges in most affected dogs. Motor nerve conduction and repetitive nerve stimulation results were similar among groups. Compared with controls, brainstem auditory-evoked potentials (BAEP) showed mildly prolonged latencies, with disappearance of wave components in 3 cases. In dogs with myokymia, height- and age-adjusted mean onset latency of tibial sensory evoked potentials (SEP) was significantly delayed, when recorded at the lumbar (i.e., L6-L7, L4-L5), but not at the peripheral nerve level (i.e., trochanter major). Cord dorsum potential onset-to-peak latencies were not different between groups. These somatosensory findings reflected dorsal root (e.a. cauda equina) demyelination. Interestingly, the single affected dog without associated ataxia was the only one to show normal BAEP and SEP latencies (i.e., within the reference range of the controls).

In conclusion, we propose that BAEP and spinal SEP abnormalities shown in myokymic JRT are due to the unique association of NMT with HA. These electrophysiological findings emerge from the neurodegenerative changes characterizing HA and therefore cannot elucidate the pathogenesis of NMT.

NEUROLOGICAL DYSFUNCTION AND HYPOVITAMINOSIS A IN A 6 MONTH OLD CHEETAH (*Acinonyx jubatus*). Luisa De Risio, Elsa Beltran, Andrew Holloway, Anthony Tropeano[1], Alberta de Stefani, John Lewis[2]. The Animal Health Trust, Newmarket, UK; [1]Colchester zoo, Colchester, UK; [2]International Zoo Veterinary Group, Keighley Business Centre, Keighley, UK

A six-month-old female captive-bred cheetah presented with a three-week-history of progressive ataxia, impaired balance, and nystagmus. She was the only surviving cub of her litter and she had been hand-reared with kitten milk replacement. Since 3 months of age she was fed raw meat (horse and beef) with calcium supplementation. Neurological examination revealed cerebello-vestibular ataxia. Proprioception was difficult to assess but appeared decreased. The withdrawal reflex was normal in all four limbs. Cranial nerve examination revealed absent menace response bilaterally, constant horizontal pendular nystagmus, which became rotatory or vertical when she was lying on her back. Haematology, comprehensive biochemistry and abdominal ultrasound did not reveal any significant abnormalities. MR imaging of the brain revealed caudal fossa overcrowding and cerebellar compression and herniation through the foramen magnum. CSF analysis was normal. CSF PCRs for CDV, *Toxoplasma gondii, Neospora caninum*, FHV, Bornavirus, FeLV, FIV and Feline Coronavirus were negative. Thiamine blood level was normal. Serum Vitamin A concentration was $< 0.1\,\mu mol/l$ (reference range 1.7–4.6 μmol/l). Vitamin A oral supplementation (administered as multivitamin complex) was followed by gradual clinical improvement over several weeks. Complete recovery was observed when serum Vitamin A concentration returned to normal.

Neurological dysfunction, caudal fossa overcrowding, and cerebellar compression and herniation have been reported in young captive lions with hypovitaminosis A, but have not been reported previously in cheetahs. Vitamin A supplementation has been associated with clinical improvement in lion cubs with mild to moderate neurological dysfunction.

Nutritional aetiologies should be considered and investigated in captive-bred cheetah cubs with progressive neurological dysfunction.

CLINICAL SIGNS AND MRI FINDINGS OF BILATERAL PORENCEPHALY AND CAUDAL VERMIAN HYPOPLASIA IN A YOUNG DOG. Marco Rosati[1], Marco Bernardini[2,3], Pietro Calò[3], Luciano Pisoni[1], Gualtiero Gandini[1]. [1]Veterinary Clinical Department, University of Bologna, Italy; [2]Department of Veterinary Clinical Sciences, University of Padua, Italy; [3]Veterinary Hospital "Portoni Rossi", Zola Predosa, Italy

The present report describes a 4 months-old mixbreed female dog referred to the Veterinary Teaching Hospital of Bologna University for the suspicion of atypical seizure activity. The dog was adopted 40 days before from a kennel. After ten days, the owner noticed the occurrence of sudden-onset "attacks", described as follows: sudden tilting of the head to the right, falling towards her right side, pathologic nystagmus and opisthotonus. After approximately 30 seconds, the dog recovered completely to a normal status. Episodes occurred two to three times per day. The owner reported also episodes of tail chasing and right circling lasting 10 to 15 minutes, which tended to stop once the patient was diverted.

Physical evaluation showed an abnormal right deviation of the nasal structures. The neurological examination showed a normal mental status and behaviour, a mild right head tilt, vestibular ataxia, decreased postural reactions more pronounced on the left limbs, menace response absent on the left side and reduced contralaterally, and right ventral strabismus. Neuroanatomical localization of the lesion involved the central vestibular structures and the forebrain. Major differential diagnoses included as first anomalous disorders.

CBC and serum biochemistry were within the normal range. The dog underwent magnetic resonance imaging (MRI) of the brain. An area of low signal intensity was observed on T1-W images in the caudal cerebellar vermian region. The same region was characterized by a high signal intensity on T2-W images. These characteristics were consistent with the presence of a fluid-filled cavity in the region of the cerebellum, assumed to be due to cerebrospinal fluid accumulation. A second lesion with the same characteristics, connecting the lateral ventricle with the subarachnoid space, was observed in the left parietal lobe. A third, smaller, porencephalic area was noted in the right cerebral hemisphere. The final diagnosis, based on the clinical signs and MRI findings was congenital bilateral porencephaly and caudal vermian hypoplasia.

The vestibular episodes were considered as possible partial-complex epileptic seizures, anticonvulsant therapy with Phenobarbital was instituted and the episodes improved in frequency and entity.

The non-progressive, atypical, clinical signs in our puppy fits very well with the multiple abnormalities documented on MRI, affecting both the cranial and the caudal fossa. Caudal fossa lesions are consistent with those reported in the Dandy-Walker Syndrome (DWS). Forebrain MRI abnormalities in our puppy can be defined as asymmetric bilateral porencephaly. To the authors' knowledge, this is the first description of CNS malformation consistent with Dandy-Walker syndrome associated to porencephaly and abnormality of the face profile in the dog.

MULTIFOCAL ISCHAEMIC STROKES DUE TO A HYPERCOAGULABLE STATE CAUSED BY A SMALL INTESTINAL HIGH-GRADE T-CELL LYMPHOMA IN A DOG. Meichner K[1], Flatz K[2], Schnabl E[2], Schulz B[1], Keller LJM[1], Janik D[3], Ludwig E[3], Fischer A[1]. 1. Clinic of Small Animal Internal Medicine, Ludwig Maximilian University, Munich, Germany. 2. Clinic of Small Animal Surgery and Reproduction, Ludwig Maximilian University, Munich, Germany. 3. Institute of

Veterinary Pathology, Ludwig Maximilian University, Munich, Germany

A nine year old male pug presented with a history of chronic diarrhea, weight loss and acute syncope-like episodes. Following initial examination, the dog developed non-ambulatory tetraparesis with right-sided hemiplegia, horizontal and rotating nystagmus, and pleurothotonus to the left. Other important findings included possible left ventricular outflow tract obstruction caused by a thrombus of the septal mitral valve and a small intestinal tumour on abdominal ultrasound. Conventional and specialized magnetic resonance imaging (MRI) sequences of the brain identified multifocal ischaemic strokes. Cerebrospinal fluid (CSF) analysis showed a moderate to severe mixed-cell pleocytosis with a slight eosinophilic predominance.

Ischaemic strokes and the mitral valve thrombus were the result of a hypercoagulable state due to small intestinal high-grade T-cell lymphoma. The neoplasia caused severe protein-losing enteropathy and chronic blood loss anemia with decreased antithrombin III activity and thrombocytosis. Following resection of the intestinal tumour, a multidrug chemotherapy protocol was initiated.

Neurologic symptoms resolved almost completely within 4 to 6 weeks. To date the dog is in complete clinical remission and has attained a very good quality of life.

POSTER PRESENTATION

A T_H2-DOMINATED IMMUNE RESPONSE EXPLAINS HIGH IMMUNOGLOBULIN A AND B CELL LEVELS IN CANINE STEROID-RESPONSIVE MENINGITIS-ARTERITIS. M Schwartz[a,b], C Puff[a], VM Stein[a], W Baumgärtner[a,b], A Tipold[a,b]. [a]School of Veterinary Medicine, Hannover, Germany, [b]Center for Systems Neuroscience, Hannover, Germany

Steroid-responsive Meningitis-Arteritis (SRMA) is a systemic inflammatory disease with most prominent manifestation within the cervical meninges. Laboratory changes include increased immunoglobulin A (IgA) levels in serum and cerebrospinal fluid (CSF), which are associated with high proportions of B cells. We therefore hypothesized that the immune reaction in SRMA is characterized by a dominance of Th2 lymphocytes with production of Th2-signature cytokines interleukin (IL)-4, -5 and -10.

Samples of dogs in the acute phase of SRMA (n = 16), under glucocorticosteroid treatment for SRMA (n = 16) and with other inflammatory and neoplastic disorders of the central nervous system (CNS) (n = 19) were examined for their mRNA expression of Th2-associated cytokines IL-4, -5 and -10 and indicators for a Th1-dominated immune response IL-2 and interferon (IFN)-. Samples included peripheral blood mononuclear cells (PBMNCs) and CSF white blood cells (CSF WBCs) and quantitation of mRNA expression was performed by reverse-transcriptase real-time PCR. Values for cytokine mRNA levels were normalized to a set of 3 reference genes.

Samples containing PBMNCs of dogs in the acute phase of SRMA showed increased IL-4 levels whereas IL-2 and IFN- gene expression was low. Messenger RNA encoding for IL-5 and -10 could be recorded in PBMNCs, levels, however, did not differ from those of the remaining disease categories. The IL-4:IL-2 ratio in the acute phase of SRMA, which was used as a measure for the Th2:Th1 ratio, was similar in PBMNCs and CSF WBCs. Contrasting this, IL-10 mRNA levels were significantly higher in CSF WBCs when compared to PBMNCs. Th2:Th1 ratios remained increased in a number of dogs with SRMA under glucocorticosteroid treatment.

The presented data indicate that SRMA is associated with a Th2-dominated immune response characterized by a pronounced IL-4 production. These results may well explain previous findings of increased B:T cell ratios and high IgA levels in serum and CSF. Some dogs showed a persistent Th2-dominance under glucocorticosteroid treatment, which is in line with the finding that IgA levels remain elevated in a number of these individuals. Similar Th2:Th1 ratios in peripheral blood and CSF indicate that no selective entry into or proliferation within the CSF occurs of either Th2 or Th1 lymphocytes. Up-regulation of the immunomodulatory IL-10 may prevent progression of the inflammatory response to the CNS parenchyma and may as well induce the transition of symptomatic phases into clinically silent periods in-between.

MMP-2 AND -9 EXPRESSION OF LEUKOCYTES INVADING THE SUBARACHNOID SPACE IN THE ACUTE PHASE OF CANINE STEROID-RESPONSIVE MENINGITIS-ARTERITIS. M Schwartz[a,b], C Puff[a], VM Stein[a], W Baumgärtner[a,b], A Tipold[a,b]. [a]School of Veterinary Medicine, Hannover, Germany, [b]Center for Systems Neuroscience, Hannover, Germany

The hallmark of steroid-responsive Meningitis-Arteritis (SRMA), the most frequently occurring inflammatory disease of the canine central nervous system (CNS), is the migration of large amounts of neutrophils into the subarachnoid space. Measurements of the IgG index suggest that intrathecal immunoglobulin production is accompanied by a loss of blood-cerebrospinal fluid (CSF)-barrier integrity. Matrix metalloproteinases (MMP)-2 and -9 are gelatinases that degrade extracellular matrix and thus facilitate leukocyte extravasation and also mediate blood-CSF-barrier permeabilization. We therefore hypothesized that leukocytic up-regulation of MMP-2 and -9 occurs in SRMA and also investigated whether counter-regulation with tissue inhibitor of metalloproteinases (TIMP)-1 or -2 expression is initiated.

Cell pellets of dogs in the acute phase of SRMA (n = 16) were examined for gene expression of MMP-2/-9 and TIMP-1/-2. Quantification of mRNA expression was performed by reverse-transcriptase real-time PCR and values were normalized to a set of 3 reference genes. Results were compared to those of dogs under glucocorticosteroid treatment for SRMA (n = 16) and dogs with other inflammatory and neoplastic diseases of the CNS (n = 19). Samples included mononuclear (PBMNCs) and polymorphonuclear cells (PBPMNs) of the peripheral blood and cerebrospinal fluid white blood cells (CSF WBCs).

Matrix metalloproteinase-2 and -9 and TIMP-1 and -2 expression could be detected in CSF WBCs of dogs in the acute phase of SRMA. MMP-2 mRNA levels in CSF WBCs were significantly up-regulated in comparison to PBMNC expression levels ($p < 0.01$) and MMP-9 expression in PBPMNs of dogs in the acute phase of SRMA was significantly higher than in PBPMNs of dogs under glucocorticosteroid treatment ($p < 0.05$).

Presence of mRNA encoding for MMP-2 and -9 in CSF WBCs of dogs in the acute phase of SRMA supports a contribution of these cells to the overall content of MMPs in the CSF of affected dogs. Up-regulation of MMP-2 in CSF WBCs in comparison to PBMNCs suggests that MMP-2 production is relevant for PBMNC migration into the subarachnoid space. Higher MMP-9 mRNA levels in PBPMNs of dogs in the acute phase of SRMA in comparison to dogs under treatment indicate a contribution of MMP-9 to the pathogenesis of the marked neutrophilic pleocytosis in SRMA. Counter-regulation with TIMP-1 and -2 production by CSF WBCs may represent a mechanism that prevents damage of the CNS parenchyma.

REACTIVE SEIZURES IN DOGS: A RETROSPECTIVE STUDY OF 96 CASES C. Brauer[a], M. Jambroszyk[b], A. Tipold[a]. [a]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany; [b]Small Animal Practice Dr. Ehrhardt & Ehrhardt, Castrop-Rauxel, Germany

Reactive seizures can be elicited by a wide variety of metabolic and toxic disturbances. Dysfunction of virtually any organ system can lead to this kind of seizure. Depending on the underlying disease, most of these conditions are reversible which is important for planning of treatment regimens.

Patient records of 877 dogs presented for seizure disorders to the University of Veterinary Medicine Hannover from 2004 to 2008 were reviewed. Out of these 96 dogs (11%, 96/877) were identified as cases with an underlying metabolic or toxic etiology including intoxications by varying substances, hypoglycemia, electrolyte disorders, hepatic encephalopathy, hypothyroidism, uremic encephalopathy, hypoxia and hyperglycemia.

Intoxication was the most frequent underlying cause for reactive seizures (39%, 37/96). Metaldehyde intoxication was identified in seven (19%, 7/37), organophosphate/carbamate intoxication in six dogs (16%, 6/37). In the latter, the mean cholinesterase serum level was 354 U/L (reference range 1500–3000 U/L). Hypoglycemia caused seizures in 31 dogs (32%, 31/96) and was related to neoplasias in 21 dogs (68%, 21/31). Mean age of dogs with neoplasia

induced hypoglycemia was 10 years (range 7–16 years). Hypoglycemia in five young dogs (16%, 5/31) was due to starvation, gastrointestinal parasites or disturbances. These dogs had a mean age of 3.4 months. Mean blood glucose concentration of hypoglycemic dogs was 2.19 mmol/L (reference range 3.9–6.1 mmol/L). Electrolyte disorders were responsible for seizures in ten dogs (10%, 10/96). In five of these marked hypocalcemia with mean ionized calcium concentrations of 0.61 mmol/L was identified (reference range 1.25–1.47 mmol/L).

Metabolic and toxic disturbances were responsible for seizures in a significant number of dogs and should therefore always be included as an important differential diagnosis. This is especially important since these dogs require different treatment strategies than dogs suffering from idiopathic epilepsy.

COMPUTED TOMOGRAPHIC FINDINGS IN CATS WITH SUSPECTED PERIPHERAL VESTIBULAR SYNDROME. AM Hernández-Guerra[1], F Liste[1] and ML Ortiz[1]. [1]Hospital Clínico Veterinario. Universidad CEU-Cardenal Herrera, Moncada, Valencia, Spain.

The aims of this study were to describe the clinical signs and CT scan findings of 15 cats with peripheral vestibular syndrome (PVS). Medical records were obtained from feline patients with PVS that underwent CT scan of the head. Clinical history and follow up were recorded. Cats showing one or more signs compatible with vestibular syndrome of central origin (altered mental status, proprioceptive deficits, vertical nistagmus, and cranial nerve deficits other than facial nerve palsy) or abnormal CSF analysis were excluded. CT scan was evaluated using bone and soft tissue windows. Thickening and bone sclerosis of tympanic bullae walls and presence of soft tissue/fluid inside the middle ear cavities was assessed. CT scans were independently reviewed by the three authors.

Six cats showed CT signs of bilateral otitis media (bone thickening and sclerosis and/or soft tissue density inside tympanic bullae) (40%), 4 of the 15 cats showed no abnormalities in the CT scans (27%), 2 cats signs of right otitis media (13%), 2 cats signs of left otitis media (13%) and 1 cat tympanic bullae fracture (7%).

From among cats showing signs bilateral otitis media on CT, 2 showed clinical bilateral vestibular syndrome and 4 displayed signs of unilateral vestibular disease (2 right-sided, 2 left-sided).

All five cats with unilateral CT findings (otitis media, fracture) showed corresponding ipsilateral clinical signs.

Cats with no CT abnormalities were considered as to have idiopathic vestibular syndrome (IVS). No age, sex or breed predisposition were observed. 3 cats affected with IVS showed an acute onset of signs but all of them recovered uneventfully without any medication within one month. In one cat showing a normal CT scan, some head tilt still persisted one month later.

The results of this study confirm that the main causes of PVS are either idiopathic (40%) or related to otitis media (53%). The percentage of otitis media is slightly superior to other studies' percentage. This may be due to the arguably superior capacity to detect bony changes. On the other hand, probably due to its limitation in imaging soft tissues in caudal fossa compared to magnetic resonance, no CNS inflammatory o degenerative conditions were diagnosed.

CLINICAL SIGNS IN ASSOCIATION WITH RATHKE'S POUCH CYST IN 11 DOGS. U. Michal Altay, L. Motta, J. Woolley, G.C. Skerritt. ChesterGates Referral Hospital, Chester UK

Rathke's pouch cysts (RPC) are thought to be remnants of the embryological precursor of the adenohypophysis; they are epithelium-lined cysts containing mucoid material. Four dogs with this condition have been reported previously.

Dogs with RPC (n = 11) were studied. The findings of clinical evaluation, laboratory analysis, magnetic resonance imaging (MRI), histopathology and outcome with or without surgical treatment are reported. A pharyngoscopy was carried out and confirmed a cyst in two cases.

Males were overrepresented (72%). The median age at presentation was 3.5 years (23–83 months). All dogs presented with neurological clinical signs including acute generalised seizures with altered mentation, new development of aggression (mainly in association with seizures), respiratory noise in association with seizures, episodic behavioural changes and exercise intolerance in one case. Polyuria and polydipsia were reported in two cases.

Neurological examination was normal in all cases apart from reduced visual ability in one case. Laboratory changes were mild and nonspecific in all cases. Additional serological testing revealed no evidence of Toxoplasmosis, Neosporosis, hypothyroidism or liver dysfunction. An ACTH stimulation test was carried out and was normal in three cases. The dog with exercise intolerance was investigated for neuromuscular disease with no diagnostic results. Cerebrospinal fluid was evaluated and normal in five cases. On MRI cysts were mostly isointense on T1 (6/11), hyperintense on T2 (8/11) and enhancing after gadolinium in one case. Cyst diameter was 3 to 6 mm (median 4 mm) at time of first presentation.

Surgical removal of the cysts were carried out by a transpalatine approach in seven dogs, whereas four dogs were treated conservatively. Immediate postoperative recovery was uneventful. No complication apart from mild nasal discharge in the immediate postoperative time was seen. After surgery, one dog was showing no more seizures; five dogs continued to have seizures at the original severity and two of these were euthanased within three months; the dog with exercise intolerance displayed unchanged clinical signs.

In dogs that did not undergo surgery, two dogs did not show further seizures, seizures were well-controlled on phenobarbitone in one dog, and one dog was lost for follow-up.

Inflammation of cysts was confirmed on histopathology in four cases. This includes the dog with contrast enhancement on MRI. Squamous metaplasia was found in the dog with progressive seizures and behavioural changes. This histopathological feature has been associated with cyst recurrence postoperatively in humans.

Our case group showed a male predisposition. In humans, women are predisposed. The main clinical presenting signs in dogs with Rathke's pouch cyst were seizures and aggression. Persistence of clinical signs post surgery could be due to underlying primary epilepsy, incomplete cyst removal or a cyst of different origin (ie. cranial pharyngeal duct remnant or migrated respiratory epithelium). Incidental RPC have been reported in humans but not in veterinary medicine. Cysts were presumed incidental in 6/7 and in 3/4 cases and the authors are currently evaluating further cases as the incidence in dogs is not known, considering particularly cyst size and histopathological changes in relation to clinical signs.

THERAPY AND FOLLOW-UP IN 7 CATS WITH SUSPECTED HIPPOCAMPAL NECROSIS. Pakozdy Akos[1], Kneissl Sibylle[2], Gruber Andrea[3], Leschnik Michael[1], Partej Michaela[1], Magloczky Zsofia[4], Halasz Peter[5], Hassan Jasmin[2], Thalhammer Johann G[1]. [1]Clinic for Internal Medicine and Infectious Diseases, [2]Clinic of Diagnostic Imaging, [3]Institute of Pathology and Forensic Veterinary Medicine, University of Veterinary Medicine, Vienna, Austria [4]Institute of Experimental Medicine, Hungarian Academy of Science, Budapest, Hungary, [5]Pázmány Péter Catholic University, Faculty of Information Technology, Budapest, Hungary

Hippocampal necrosis (HN) is reported to be a cause for seizures in cats[1,2,3]. Although ante mortem tentative diagnosis is possible with Magnetic Resonance Imaging (MRI), no data are available about treatment and follow-up[3]. The aim of the study was to analyse the effect of treatment on the clinical course and follow-up. Included in this study are cats with the following criteria: (1) seizures in the medical history, (2) HN, suggested by advanced diagnostic imaging or confirmed by pathohistological examination, (3) antiepileptic drug therapy, (4) minimum follow-up of thirty days after the first seizures available. Seven cats met the inclusion criteria. All cats were indoor, European shorthair. The age of onset was 2–11 years (mean 6.14). The following abnormalities were detected by physical and neurological examination: decreased menace response (4), elevated body temperature (4), aggression (3), mild decreased proprioception (2), paraparesis (2), confusion (1), circling (1). MRI of 4 cats showed bilateral hippocampal T1 hypo/isointensity and T2 hyperintensity. Computer tomography (CT) revealed mildly in-

creased hippocampal and meningeal contrast enchancement in one cat. CSF analysis was unremarkable in all 7 cats. The following treatment was administered: phenobarbital (7), gabapentin (5), supportive fluid therapy (5), diazepam (3), prednisolone (3), amoxicillin-clavulanic acid (2), thiamin (2), dexamethasone (1), marbofloxacine (1), midazolame (1). One cat was euthanised due to lack of remission 52 days after the first seizures and 4 days after start of treatment. Another cat was euthanised 111 days after the first seizure and 109 days after the start of treatment, because of recurrent seizures.

The histopathological examination of these 2 cats showed marked bilateral hippocampal neuronal loss, accompanied by severe gliosis with proliferation of microglia and gemistocytes, proliferation of capillaries and mild non suppurative perivascular inflammatory infiltrates. The mean survival time for all 7 cats was 322 days (range 52–730 days). Five cats were still alive at the time this manuscript was submitted. Three of them remained seizure free with long-term phenobarbital therapy (for 61, 240, 730 days) and two cats showed second cluster seizure period 6 and 8 months after the first event, and regular seizure activity during the monitoring period. The owners of the five surviving cats reported good quality of life. Repeated neurologic examination showed paraparesis in 2 cats. In contrast to the previously published 44 cases on feline HN that suggested unfavourable prognosis as all cases died or were euthanised, our study demonstrates survival of 5/7 patients – although most of these were unconfirmed[1,2,3]. We also observed poor initial response to antiepileptic therapy, which may result in euthanasia in some cases and lead to the conclusion, that this disease cannot be treated. We observed that cats became frequently seizure-free after antiepileptic drugs and supportive therapy for several days (3–8 days). In these animals normal behaviour gradually returned after 7–90 days. However, the lack of histopathological confirmation in the surviving cases limits the conclusion. The use of corticosteroids in acute stage of the disease may have positive effects, because of the mild hippocampal inflammation and fever in some cases.

REGULATION OF SURFACE MOLECULES ON CANINE MICROGLIA DEPENDS ON THE LOCALISATION IN THE CNS. E.-M. Ensinger, T.M. Boekhoff, R. Carlson, A. Tipold, V.M. Stein. Department of Small Animal Medicine and Surgery, School of Veterinary Medicine Hannover, Germany

Microglial cells are the resident immune effector cells of the central nervous system (CNS). Their principal function is to control and to sustain the integrity of tissue in the CNS. By secreting neurotrophic substances on the one hand they seem to have neuroprotective qualities. On the other hand due to the production of potentially toxic substances they might have negative influences on tissue regeneration. According to these dual qualities microglia may play an important role in degeneration and regeneration. Differences in the regulation of surface molecule expression on microglia might give important insights for the understanding of predilection sites of some diseases within the CNS. Therefore, the aim of this study was the evaluation of immunophenotype and morphology of canine microglial cells in relation to different regions in the CNS.

In an effort to answer this question microglial cells from 22 healthy three-month-old SPF-Beagles were isolated ex vivo from three localisations in the CNS, brain, cervical, and thoracical spinal cord, and stained with 16 different antibodies, including CD11b, CD1c, ICAM-1, B7-1, B7-2, MHC I and II. The isolation protocol comprised a mechanical and enzymatic dissociation of spinal cord and brain tissue, and two consecutive density gradient centrifugation steps. The isolated and labelled cells were measured by flow cytometry.

Surface marker expression revealed physiological regional differences in the immunophenotype of canine microglial cells. Specifically, surface markers responsible for co-stimulation of T-cells, for leukocyte adhesion and aggregation and for lipid or glycolipid presentation are expressed differently between the brain, the cervical and thoracical spinal cord. These differences are statistically significant for B7-1 +, CD14 + and CD44 + cells and for the integrins CD18, CD11b, CD11c and CD1c. A certain tendency of higher expression of CD45 was seen in the thoracical spinal cord.

Our data of microglial immunophenotypical and morphological characterization represent an important tool for comparative studies with diseased animals. Only microglia from specific localisations in the CNS can serve as reference values. The dog seems to be an ideal model to further investigate neuroimmunological aspects for instance in spinal cord trauma. The current findings encourage speculation regarding predilection sites for CNS diseases.

ENHANCED FUNCTIONAL ACTIVITY OF CANINE MICROGLIA ISOLATED FROM THE SPINAL CORD. T.M. Boekhoff, E.-M. Ensinger, R. Carlson, A. Tipold, V.M. Stein. Department of Small Animal Medicine and Surgery, School of Veterinary Medicine Hannover, Germany.

Microglial cells are known as resident immune effector cells of the central nervous system (CNS). They play an important role for the balance of the homoeostasis and in the host defence against invading pathogens. In case of pathogenic stimuli the microglial cell develops different functions such as processing and presenting of antigens, phagocytosis, modulation of the T-cell response, production and release of cytokines, chemokines, reactive oxygen species (ROS) and nitrogen species. Due to these skills microglial cells can support neuronal regeneration as well as be responsible for the development of secondary damage after trauma.

The purpose of this study was to isolate canine microglial cells from brain, cervical and thoracical spinal cord and to make them available for *ex vivo* characterizations of their functions. Functional characterization was assessed by phagocytosis assay and ROS-generation test. In the course of this examination the functions of the microglial cells isolated from the three different regions of the CNS were compared to each other. Microglia of 22 3-month-old Beagles was isolated by mechanical and enzymatical dissociation of the CNS tissue, followed by density gradient centrifugation. The isolated cells were characterized morphologically and functionally using flow cytometry.

It could be shown that microglial functional activity demonstrated physiological regional differences. Phagocytosis as well as generation of ROS were more active in cervical and thoracical spinal cord than in the brain, which was statistically significant ($p < 0.05$). There was even a certain tendency of higher phagocytosis in the cervical than in the thoracical spinal cord. Microglia of the spinal cord seems to be more active either because this part of the CNS is more exposed to lesions or contrary the brain is better protected and ROS production reduced to a minimum. The results of this study can be used for evaluation of microglial phagocytosis and ROS generation in dogs with spinal cord trauma. It is important to know that microglia from specific regions needs to be compared to region specific reference values. The canine spinal cord serves as an ideal model for investigating different regions of the CNS having enough material for gaining microglia.

MEDIASTINAL PARAGANGLIOMA WITH EXTRADURAL INVASION IN A DOG. D. Sanchez[1,2], J. Closa[1], A. Font[1], A. Zamora[2], M. Pumarola[3], J. Mascort[1]. [1]Hospital ARS Veterinaria, Barcelona, Spain. [2]Centre de la Imatge Veterinaria IMAGOVET, Sant Joan Despi, Barcelona, Spain [3]Universitat Autonoma de Barcelona, Bellaterra, Barcelona, Spain

Mediastinal paragangliomas are a group of tumors that arise from paraganglia associated with the pulmonary artery and aortic arch or from the segmental paraganglia located in the autonomic chain of the thoracic region. Based on the anatomic location, these tumors can be further classified into 3 groups: pheochromocytomas, extra-adrenal pheochromocytomas, and chemodectomas.

An 11 year-old, 25-kg, male intact, Chow-chow dog was evaluated because of weakness, lethargy and anorexia of four weeks duration. During physical and neurological examination, the dog was lethargic and thin, had a stiff pelvic limb gait, with slightly delayed postural reactions mainly on the left side, and bilaterally increased segmental spinal cord reflexes on both pelvic. Nocicepcion was intact. Examination of the thoracic limbs and cranial nerves

was considered normal. The lesion was therefore localized to the T3-L3 spinal cord segments.

Hematology, biochemistry and urianalysis results were within reference ranges. Abdominal ultrasounds were unremarkable. Thoracic radiographs showed a soft tissue opacity dorsal to the lung fields in the left lateral projection. On ventrodorsal projection the trachea was displaced to the right.

The dog underwent a Magnetic Resonance (MR) study of the cervical and thoracic spine with an Esaote 0.25T unit. T1-weighted, T2-weighted and FLAIR sequences were obtained in different planes. These revealed a mediastinal mass ventral to the spine, extending from the first to fifth thoracic vertebra and measuring approximately seven centimetres in the craniocaudal direction. There was invasion of the vertebral canal by the mass through the left T3-T4 vertebral foramen resulting in compression of the spinal cord. Postcontrast T1-weighted sequences showed a heterogeneous contrast enhancement. Based on the imaging findings, differential diagnoses included a neoplasm of the paraspinal soft tissues or a large peripheral nerve sheath tumor.

A left-side thoracotomy at the level of the fifth intercostal space was performed in order to remove the mediastinal neoplasia which was followed by a left T3-T4 hemilaminectomy to decompress the spinal cord. One week after the surgery, the dog suffered an acute respiratory distress and sudden death. Necropsy was not allowed by the owners but a pulmonary thromboembolism was suspected. The dog was still recumbent by the time of death.

Histological findings showed a highly cellular mass. Neoplastic cells were uniform medium-sized, round or polyhedral in shape, with moderately hyperchromatic round nuclei and distinct nucleoli. The cytoplasm was finely granular and eosinophilic. Several large areas of hemorrhage and necrosis were also present in the submitted sections. Tumor cells showed no reaction to synaptophysin or chromogranin. Histological diagnosis was a malignant mediastinal paraganglioma.

Extra-adrenal paragangliomas are rare neoplasm in dogs, and spinal cord invasion of a primary mediastinal paraganglioma has only been reported in 3 dogs previously. However no surgery had been attempted. In human medicine, surgical resection is recommended with o without adjuvant therapy.

Presumptive diagnosis is based on a combination of history, physical examination, and imaging studies. Definitive diagnosis can only be reached following histopathological confirmation. Immunohistochemical analysis may be useful to confirm whether the tumor has neuroendocrine origin, however further subclassification may not be possible in cases of undifferentiated tumors.

A mediastinal extra-adrenal paraganglioma should be included as a differential diagnosis for dogs with progressive paraparesis and an intrathoracic neoplasm.

ASSOCIATION OF NEUROLOGICAL SIGNS AND PRE- AND POST-TRACTION MAGNETIC RESONANCE IMAGING FINDINGS IN 25 DOBERMANS WITH CAUDAL CERVICAL SPONDILOMYELOPATHY. Fabio Stabile[1], Marco Bernardini[2,4], Livio Corain[3], Giovanni Bevilacqua[4], Alberta de Stefani[1], Luisa De Risio[1]. [1]The Animal Health Trust, Newmarket, UK; [2]Department of Veterinary Clinical Sciences, University of Padua, Italy; [3]Department of Management and Engineering, University of Padua, Italy; [4]Clinica Veterinaria "Portoni Rossi", Bologna, Italy

Determining the presence of a dynamic component in caudal cervical spondylomyelopathy (CCSM) is important to plan the most appropriate treatment. Predictive criteria for traction-responsive lesions on pre-traction magnetic resonance imaging (MRI) have not been reported in dogs with CCSM. The purpose of this prospective study was to assess the association between neurological signs and findings on pre and post linear traction MRI of the cervical spine in Dobermans with CCSM.

Dobermans diagnosed with CCSM that underwent physical and neurological examination, routine laboratory tests, and MRI of the cervical spine pre and post linear traction (as described by Penderis 2004), between January 2006 and December 2008, were included in the study. The following MRI findings were assessed: vertebral body abnormal shape and tipping, intervertebral disc degeneration and protrusion, ligamentum flavum hypertrophy, presence and features of intramedullary hyperintensity (above the affected intervertebral disc space) on T2-weighted images. Statistical analyses were performed by Non-Parametric Combination tests. The level of significance was set at $p < 0.05$.

Twenty-five Dobermans met the inclusion criteria. Twelve were male and 13 were female. Median age was 6.8 years. A traction-responsive lesion was identified in 15 dogs. Neurological examination findings were not associated with the presence of traction-responsive lesions. Tipping of C7 vertebral body ($p = 0.044$), degeneration of C7-T1 intervertebral disc ($p = 0.041$) and presence of a well-defined intramedullary hyperintensity on T2-weighted images ($p = 0.042$) were associated with traction-responsive lesions.

These results suggest that certain findings on pre-traction MRI can help to predict a dynamic component of the lesion in Dobermans with CCSM.

CANINE STATUS EPILEPTICUS DUE TO ACUTE INTOXICATIONS. Romina Zimmermann, Velia-Isabel Hülsmeyer, Andrea Fischer. Section of Neurology, Clinic of Small Animal Medicine, Ludwig-Maximilians-University Munich, Germany

Seizure disorders are the most common neurological diseases in dogs such as in humans. Status epilepticus (SE) represents a special form of epileptic seizures (ES). This life-threatening condition requires urgent and adequate treatment depending on the underlying disease condition.

The purpose of this study was to evaluate type of toxin, clinical presentation and outcome of dogs with status epilepticus (SE) due to acute poisoning presented to a large referral veterinary hospital.

Medical records of dogs that were admitted to a veterinary teaching hospital (January 1, 2002 to April 30, 2008) because of SE were screened for entry in this study and evaluated retrospectively. The general inclusion criterion was initial presentation in SE caused by acute intoxication.

Fourteen dogs with SE due to acute intoxication were identified. Toxicological analyses detected poisonings with carbofurane, crimidine, paraoxone, metaldehyde, strychnine, zinc phosphide and diazinon. All dogs were neurologically normal up to day of presentation. Dogs were hospitalized for two to ten days (median five days). The survival rate was 85.7%. None of the surviving dogs experienced any subsequent seizures. Among dogs presenting with SE with no prior history of seizure disorder, 20.9% suffered from poisoning.

One-fifth of dogs presenting SE as their first seizure suffered from acute intoxication. Ancillary to the acute clinical presentation, preliminary reports (possible uptake of poisonous material) and an inconspicuous medical history may suggest a tentative diagnosis. Managed adequately, these patients can have a high survival rate and should never experience seizures again. Clinicians should also keep uncommon intoxications in mind. Organophosphate and carbamate poisonings require additional treatment with atropine.

PORENCEPHALY AND HYDRANENCEPHALY IN FOUR DOGS WITH SEIZURES AND ABNORMAL MENTATION. Emma Davies, Laurent Garosi, Sebastian Behr, Holger Volk, Brian Summers, Alexander de Lahunta. From: Davies Veterinary Specialists, Higham Gobion, England (Behr, Garosi); Royal Veterinary College, London, England (Davies, Summers, Volk), Rye, New Hampshire (de Lahunta)

Porencephaly and hydranencephaly are cerebral cavities which frequently communicate with the subarachnoid space and/or lateral ventricles following fetal or perinatal brain destruction. These are rarely reported in dogs.

We present case history, clinical signs, imaging and post mortem findings of four dogs with either unilateral porencephaly (1,2 & 4) or hydranencephaly (3). The dogs presented with forebrain disorders, either seizures (dog 1 and 2), abnormal mentation (dogs 2, 3 & 4) and circling (dog 3 & 4) at an age range of 6 months to 3 years. The seizure frequency in dog 2 was increasing and did not respond to standard antiepileptic medication.

The physical examination and skull conformation were normal (n = 4). Magnetic resonance (MR) imaging of all dogs revealed a cavity communicating with the lateral ventricles. The fluid within the cavities had the same MR characteristics as CSF. Secondary asymmetry of the ipsilateral thalamus and midbrain was also noted (n = 4). MR imaging was repeated after 3 months in dog 1, which revealed an enlargement in the size of the abnormal ventricle, with loss of periventricular parenchyma. A ventriculoperitoneal shunt was placed in dog 4 which three months post-operatively continued to have an improved mentation and greatly reduced circling frequency. Dog 2 was euthanized following diagnostic imaging. The post mortem examination revealed a complete loss of the right frontal lobe, with meningeal fibrosis and dural osseous metaplasia; thickened meninges extended into the defect to the lateral ventricle. The caudate nucleus was malformed on the right side and the thalamus, internal capsule and crus cerebri were small on the right compared to the left. A single blood vessel was occluded by an organised collagenous thrombus. These changes suggest that the lesions had occurred either intrauterine or perinatally.

In humans these lesions are most commonly considered to be secondary to ischemic events. A vascular compromise is suspected to be the cause of the cerebral malformations in these dogs.

AN IMPROVED PROTOCOL FOR CULTIVATION OF CANINE MYOBLASTS. I.S. Kiesewetter[a], A. Tipold[a], D. Ragancokova[b], K. Krampfl[b], H.C. Schenk[a]. [a]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Germany; [b]Department of Neurology, Division Molecular Neurophysiology, University of Medicine Hannover, Germany

The pathophysiology of many neuromuscular diseases in dogs is still not understood. Skeletal muscle cell culture is an important tool to discover the molecular background of these diseases. The aim of the current study was to improve an existing protocol to extract and cultivate canine myoblasts. Using different enzymes for tissue digestion, the contamination of fibrocytes should be reduced and more myoblasts should be gained after a shorter cultivation period. In addition, the influence of the transport time to the laboratory on numbers of gained myoblast cells was assessed.

During routine surgery twenty-one muscle biopsies (n = 21) from healthy dogs were taken and divided in two portions of 0.2 g. Samples were mechanically trimmed, enzymatically dissociated using either Protease or Trypsin and cultured under identical conditions for 168 hours. For differentiation of myoblasts and fibroblasts an immunocytochemical staining of the muscle cell specific intermediate filament desmin was performed and the number of both cell types was evaluated. In addition, eight biopsies (n = 8) were used to analyse the influence of shipping time. These samples were cultured on the first, second and third day after surgery.

Using Protease as the digesting enzyme recovered a significant higher amount of myoblasts (P = 0.0102). After digestion with Protease the average percentage of myoblasts was 78.96% and 54.68% using Trypsin. Furthermore, Protease digestion revealed a better proliferation of cells during morphological evaluation. The duration of the transport (1–3 days) did not show any significant changes (P = 0.798) in the results for the cultured myoblasts. This is the basic requirement to send muscle biopsies of dogs with neuromuscular diseases from clinics to a specialised laboratory.

In conclusion, the use of the digestion enzyme Protease is a significant improvement for the cultivation of canine myoblasts. It produces purer cultures and therefore improves the initial conditions for further investigations of myoblasts such as patch clamp techniques or examination of specific muscle proteins.

AGREEMENT AND REPEATABILITY OF LINEAR VERTEBRAL BODY AND CANAL MEASUREMENTS USING COMPUTED TOMOGRAPHY (CT) AND LOW FIELD MAGNETIC RESONANCE IMAGING (MRI). S De Decker[1], IMVL Gielen[2], L Duchateau[1], I Polis[1], HJJ van Bree[2], LML Van Ham[1]. [1]Department of Small Animal Medicine and Clinical Biology, [2]Department of Medical Imaging, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

CT and MRI are increasingly used for the diagnosis of disorders affecting the vertebral column and spinal cord. These advanced imaging modalities have been used for morphometric measurements in different anatomic planes to provide information about the pathogenesis, diagnosis, clinical decision-making, presurgical planning, and prognosis of different disorders affecting the vertebral column and spinal cord; however, little is known about the intra- and interobserver agreement of measurements using different imaging modalities or about the agreement between CT and MRI derived measurements. Our purpose was to evaluate agreement and repeatability of vertebral column measurements using CT and low field MRI.

In 18 client-owned dogs and 3 dog cadavers, 5 measurements of the fifth cervical vertebra were performed on CT and MRI: vertebral body length (VBL), vertebral canal height (VCH), vertebral body height (VBH), vertebral canal width (VCW), and vertebral body width (VBW). Measurements were performed independently twice by 2 observers. Bland-Altman plots were created to evaluate agreement. After imaging, the cervical spinal column of the 3 cadavers was defleshed to measure the actual dimensions.

The largest discrepancy between CT and MRI measurement was for VBL (mean difference ± SD = 1.262 mm ± 1.245; $P < .001$), with the difference for all the other variables being acceptable. The 1st measurement was significantly higher than the 2nd only for VBL using CT (mean difference = 0.476 mm ± 1.120; $P = .009$), with all other variables having acceptable differences. Mean difference for all measurements between 2 observers was small, except for VBL using CT (mean difference = 0.762 mm ± 1.042; $P < .001$). Only the difference for VBL between CT and actual cadaver specimens was statistically significant.

Our results suggest high repeatability and good agreement for most vertebral measurements of interest. VBL measurement using CT was considered problematic with a clinically important and consistent overestimation combined with the highest intra –and interobserver variability. Provided limitations are understood, linear measurements of vertebral dimensions from CT and MRI images can be used clinically.

RHOMBENCEPHALOMYELITIS DUE TO *LISTERIA MONOCYTOGENES* IN A CAT IN SWITZERLAND. K. Raith[1], T. Müntener[2], M. Vandevelde[1], A. Oevermann[3]. [1]Department of Clinical Veterinary Medicine, Section of Neurology, [2]Institute of Animal Pathology, and [3]Neurocenter, Department of Clinical Research and Veterinary Public Health, Vetsuisse Faculty Bern, Switzerland

Listeria monocytogenes is a ubiquitous zoonotic bacterium, which causes a variety of infections, including septicemia, abortion, meningitis, and encephalitis. Humans and ruminants are most commonly affected, and very few infections in carnivores have been reported. Moreover, brainstem encephalitis seen in ruminants and humans has not been previously reported in small animals.

A stray cat was presented with multifocal neurologic deficits, considered localized to the brainstem and cervical cord. A FeLV-antigen test was positive and because clinical signs did not improve the cat was euthanized. Necropsy revealed a multifocal rhombencephalomyelitis with positive immunohistochemical staining (polyclonal rabbit anti-listeriolysin O antibody) for *Listeria monocytogenes*. The particular distribution of lesions, including local extension from the cervical intumescence to the midbrain, indicates that - as it is suggested for ruminant and human listeric rhombencephalitis - CNS infection followed centripetal axonal migration through nerve roots from the brachial plexus and spread subsequent rostral and caudal spread within the CNS.

This is the first observation of listeric rhombencephalitis in the cat, which was previously thought highly resistant to infection, indicating that infectious pressure of *L. monocytogenes* strains with neurotropism may be increasing. We conclude that listeriosis – a zoonotic and frequently fatal infection - should be considered a differential diagnosis for multifocal CNS disorders in cats.

ENCEPHALOMALACIA DUE TO VASCULITIS OF PROBABLE INFECTIOUS ORIGIN IN HORSES. A. Oevermann[1], F. Del Piero[3], D. Tewari[4], P. Moore[5], M. Vandevelde[2]. [1]Neurocenter, Department of Clinical Research and Veterinary Public Health and [2]Department of Clinical Veterinary Medicine, Division of Clinical Neurology, Vetsuisse Faculty Bern, University of Bern, Bern, Switzerland, [3]Department of Pathobiology and [4]Department of

Clinical Studies and PADLS, School of Veterinary Medicine, University of Pennsylvania, New Bolton Center, USA, [5]Department of Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California at Davis, Davis, USA.

Here we describe clinical signs, neuropathological findings and ancillary results of a detailed etiological investigation in seven cases of equine cerebral vasculitis in Switzerland.

The age of affected horses (5 mares, 2 stallions) ranged between 4 and 13 years. There was neither a temporal nor a geographical relationship between cases. Clinical signs included somnolence, head tilt, circling, pushing forward, ataxia, lateral recumbency, blindness, and cranial nerve deficits. In all cases, significant pathological findings were restricted to the brain and centered on the basal nuclei, thalamus, midbrain, and brainstem. Gross lesions included swelling and yellowish discoloration, occasionally associated with small hemorrhages. Histopathology consistently revealed lymphohistiocytic vasculitis with fibrinoid vessel wall necrosis and perivascular hemorrhages. Small and medium-sized arteries were mainly affected. Secondary lesions included severe edema and necrosis of the surrounding white and grey matter. Brain tissue for further investigations was available from 4 horses. Paraffin sections were stained with Gram, PAS and Grocott's methenamine silver. Indirect immunohistochemistry (IHC) was performed using antibodies against Equine Herpesvirus 1 (EHV-1), Equine Arteritis virus (EAV), Equine Eastern Encephalitis alphavirus (EEEV) and West Nile flavivirus (WNV). Polymerase chain reactions (PCR) were performed for EHV-1, EHV-2, 5 and other gamma-herpesviruses, EEEV, and WNV. Infectious agents could not be detected either with special stains or with molecular techniques. PCR amplification of the variable and joining regions of immunoglobulin genes and T-cell receptor genes was not able to detect the presence of clonal lymphocytic populations.

In conclusion, this cerebral vasculitis appears to be a sporadic disease of horses of yet unknown origin. We assume an infectious agent to be the cause. Common agents of vasculitis including EHV-1 and EAV and a neoplastic process were excluded. The absence of leukocytoclasia and systemic lesions makes an immune-mediated event such as equine purpura hemorrhagica very unlikely. The presence of *Trypanosoma evansi*, which has been recently reported to cause similar brain lesions in horses of Brazil, is currently being investigated.

MUTATIONS IN THE TRANSLATED REGION OF *KCNA1* AND *KCNA2* GENES ARE NOT THE CAUSE OF PERIPHERAL NERVE HYPEREXCITABILITY IN JACK RUSSELL TERRIERS. A.E. Vanhaesebrouck[1], L. Peelman[2], H.C. Schenk[3], S. Bhatti[1], L. Van Ham[1]. [1]Department of Small Animal Medicine and Clinical Biology, [2]Department of Animal Nutrition, Genetics, Breeding and Ethology, Faculty of Veterinary Medicine, Ghent University, Belgium; [3]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

Peripheral nerve hyperexcitability (PNH) describes a group of disorders characterized by vermicular contractions (myokymia) and/or muscle stiffness (neuromyotonia). In Jack Russell Terriers (JRT) generalized myokymia/neuromyotonia, formerly called continuous muscle fibre activity, is commonly associated with a hereditary ataxia as previously described in this breed. The objective of this study is to determine whether the genes *KCNA1*, associated with inherited PNH and episodic ataxia in humans and mice, and *KCNA2*, encoding a target protein of autoimmune PNH in humans, were also involved in the JRT breed with PNH. These genes encode the voltage-gated potassium channels Kv1.1 and Kv1.2, co-expressed in the peripheral nerve. Consequently, mutations in these channels can lead to a decreased potassium channel activity and a delay in repolarisation, resulting in a 'hyperexcitable' phenotype.

The exonic and splice site regions of the canine *KCNA1* and *KCNA2* genes were analyzed by direct DNA sequencing in 6 JRT with PNH and 6 normal control JRT. All affected dogs, except one, suffered additionally from hereditary ataxia. PNH was confirmed in all affected dogs by characteristic electromyographic findings.

Direct sequencing of the coding sequences in affected dogs revealed no pathogenic sequence changes in comparison with control dogs.

These results rule out a mutation in the translated region of the *KCNA1* and *KCNA2* genes as the cause of PNH and hereditary ataxia in the JRT breed. However, mutations in regulatory and non-coding regions or other genes encoding ion channels cannot be excluded. Therefore, further research is ongoing.

SEGMENTAL MYELITIS IN A CAT CAUSED BY *TOXOPLASMA GONDII*. Lisa Alves, Daniela Gorgas, Marc Vandevelde, Diana Henke. University of Bern, Switzerland.

Toxoplasma gondii is a protozoan that can cause disease in a wide variety of animals. It is most common in dogs and cats and has been described in man. The incidence of clinical signs is very rare. Immunosuppressed individuals are more susceptible to develop this disease.

This case report describes clinical signs, magnetic resonance imaging (MRI), and histopathological findings in a cat with segmental myelitis as a single lesion due to *T.gondii* infection.

A 4-year-old, female domestic shorthair cat was examined for a 1 month history of progressive gait abnormalities. The cat displayed a non ambulatory paraparesis. Postural reactions were decreased in both pelvic limbs. The segmental spinal reflexes of all 4 limbs were normal and cutaneus trunci response was absent bilaterally. The cat displayed severe pain on palpation of the thoracic spine. The lesion was localized at T3-L3. Differential diagnoses included neoplastic, inflammatory, infectious, and degenerative diseases.

Results of complete blood work, serum biochemistry profile, urinalysis, FeLV/FIV testing, and thoracic radiographs were unremarkable. Analysis of the cerebrospinal fluid (CSF), taken from the cisterna magna, revealed 96 white blood cells/μL, a positive Pandy test, and 30 mg/dL albumin. Differential cell count revealed 51% lymphocytes, 38% neutrophiles, 7% monocytes, and 4% macrophages.

On MR images a segmental intramedullary lesion at the level of T6-T9 was identified, which was irregularly hyperintense on T2-weighted and STIR images, and irregularly hypointense on T1-weighted images. There was strong contrast uptake in T1-weighted post contrast images. The normal hyperintense signal around the spinal cord was lacking. Based on MRI and CSF findings an inflammatory or neoplastic disease was suspected.

Serum immunofluorescence antibody (IFAT) for *T.gondii* revealed a mildly positive IgG (1:64) and negative IgM titer. The owners decided to euthanize the cat.

Complete necropsy was unremarkable. Macroscopically the spinal cord appeared swollen slightly and discolored grayish at the level of vertebra body T5-T10. On transverse sections, normal structure of spinal cord parenchyma was lost. Histologically, lesions consisted of a marked inflammatory process with necrosis of large areas within gray and white matter. The lesion was lateralized. Severe mononuclear meningitis, prominent perivascular cuffs with lymphocytes and plasmacells, parenchymal infiltration of the necrotic areas with lymphocytes and macrophages, and diffuse astrogliosis were present. Within the lesion few protozoal cysts were detected. Other regions of CNS were normal except of sporadic, small glial nodules. Immunohistochemistry for *T.gondii* was positive.

This is the first described case with MRI findings of a cat with segmental myelitis caused by *T.gondi* without immunosuppression. Although rare, this disease should be considered as a possible cause for segmental myelitis in cats.

INTRATHECAL Gd-DTPA ENHANCED MR IMAGING IN DOGS WITH TRAUMATIC SPINAL LESIONS. Valentina Lorenzo[1], Isidro Mateo[1], Jerónimo Martínez[1], Alberto Muñoz[1,2]. [1]Resonancia Magnética Veterinaria. Madrid, Spain. [2]Radiology Department, Faculty of Medicine, Complutense University of Madrid, Spain

We report the intrathecal use of gadolinium-dethylenetriamine-pentaacetic acid (Gd-DTPA) for MR imaging in 4 dogs with spinal cord trauma, in order to identify potential CSF leakages and a meningocele. Although conventional MR imaging usually demonstrates

well the CNS anatomy, there are some pathological conditions in which intrathecal CSF contrast enhancement may be beneficial. These include lesions that alter CSF flow (ie: hydrocephalus, post-surgical adhesions), cyst masses bordering CSF pathways, and postraumatic CSF meningoceles and leakages. Intrathecal Gd-enhanced MR has been studied in animal models, showing high margins of tolerance and no significant physiologic or neurohistological effects, and has been used in humans, in selected cases in which standard MR was not definitively diagnostic.

Four dogs with neurological signs after spinal cord trauma were studied: a Chow-Chow with thoracolumbar signs (case 1) a Jack Russell with right brachial plexus signs (case 2), and a Golden Retriever and a Yorkshire Terrier with lumbosacral signs (cases 3 and 4). MR imaging was performed using a superconducting 0.5T system. T2-weighted fast spin echo (FSE) (4000/110/16; TR/TE/echo train) images of the spine were acquired in sagittal and transverse planes and T1-weighted postcontrast SE images (500/14; TR/TE, Gd-DTPA dose was 0.1 mMol/kg, IV) were also acquired in the transverse plane. After conventional imaging, 0,5–1 mL of cerebrospinal fluid (CSF) was withdrawn by atlanto-occipital puncture, mixed with Gd-DTPA and re-injected intrathecally *via* the same puncture to achieve a dose of 3–3,5 μmol Gd-DTPA/g of brain tissue. T1-weighted spin echo (SE) transverse images and fat-saturated T1-weighted transverse images (SPIR) using the same acquisition parameters as before were obtained.

In all of the dogs intramedullary T2-hyperintense lesions related to the clinical findings were identified in conventional MR imaging. In case 1, a well-circumbscribed dorsal extramedullary lesion, hypointense on T2 sequences, compatible with haematoma or traumatic meningocele, was also found. In the remaining cases, a diffuse T2-hyperintensity involving the paravertebral soft tissue was present. Imaging after intratecal Gd-DTPA allowed the identification of a dural pouch filled with intrathecal contrast, compatible with postraumatic meningocele in case 1, and extravasation of intrathecal contrast in the remaining three cases, supporting the diagnosis of spinal dural tears.

Intrathecal Gd-DTPA was useful to identify potential CSF leakages and a meningocele. Diagnosis of dural tears is relevant because the defects increase the risk of meningitis and also may entrap nerve roots. Besides, a dural rent can develop into a postraumatic meningocele that may act as a chronic focus for nerve root entrapment. Intrathecal Gd-DTPA use is not currently approved by the US Food and Drug Administration and is used off-label, so its clinical use should be considered carefully, having in mind that this procedure is potentially the most accurate and less risky in the diagnosis of CSF leaks. Although the number of cases is limited, the results in our cases and the reported results in other species makes this technique to be a promising diagnostic tool in selected cases.

STABILIZATION OF A TRAUMATIC CERVICAL INSTABILITY WITH DORSAL AND VENTRAL FIXATION IN A DOG, Ozak A., Nisbet H.O., Yardımcı C. and Sirin Y.S. Department of Surgery, Faculty of Veterinary Medicine, Ondokuz Mayıs University, Samsun - TURKEY

This case study reports the outcome of dorsal and ventral stabilization of traumatic cervical instability in a dog. A two year old, male pointer dog was admitted to the Department of Surgery, Faculty of Veterinary Medicine, Ondokuz Mayıs University, following a motor vehicle accident.

Clinical examination revealed non-ambulatory tetraparesis, severe neck pain, and upper motor neuron changes of the thoracic and pelvic limbs. Deep pain response was present. Subluxation of C2-C3 and fractures of the dorsal spinous process and lamina of C2 were observed in the radiographic examination. Ventral stabilization was performed with screws and bone cement (polymethylmethacrylate). For dorsal fixation of the fractures, screws and cerclage wire was used.

The dog stood up independently after a month, was able to walk 1.5 months postoperatively and had recovered completely one year following surgery.

We conclude that the combined stabilization techniques are effective in this kind of cervical fractures in which dorsal, middle and ventral structures of the vertebra are severely disrupted.

IMAGING AND OUTCOME OF SPINAL TUMORS IN 11 DOGS AND 2 CATS. Besalti O, Caliskan M., Sirin YS, Pekcan Z., Ankara University Faculty of Veterinary Medicine Department of Surgery, Ankara – Turkey; Kırıkkale University Faculty of Veterinary Medicine Department of Surgery, Kırıkkale –Turkey; Ondokuz Mayıs University Faculty of Veterinary Medicine Department of Surgery, Samsun –Turkey

The purpose of the study is to present magnetic resonance imaging findings and surgical outcomes of spinal tumors in 11 dogs and two cats. Medical records of the cases admitted for spinal disorders were reviewed, and cases having spinal tumors which were diagnosed by MRI between November 1997 and April 2008 were included in the study. T1 and T2 weighted and contrast enhanced T1 weighted (post Gd-DTPA administration) images were taken in transverse, coronal (dorsal) and sagittal sections, and interpreted to evaluate tumors. The following features were taken into account to reach to the diagnosis in MRI: anatomic site, T1 weighted and T2 weighted features, edema, and shape. Predictive diagnosis of tumors by MRI was as follows: Meningioma (n: 4, [three dogs, one cat]), ependymoma (n = 2), Schwannoma (n = 2 [one dog, one cat]), glioma (n = 1), epidurally invading mesenchymal neoplasia (n = 2) and metastatic (n = 2). At surgery the mass was gross totally removed in 2 cats and 4 dogs. The results of surgical removal were favorable except for one dog which had malign mesenchymal tumors and died 2 months after operation. Histopathological confirmation was carried out after operation as ependymoma (n = 1), Schwannoma (n = 1), and meningioma (n = 3), epidurally invading malign mesenchymal neoplasia (n = 1), and after euthanasia epidurally invading rhabdomyosacoma (n = 1), meningioma (n = 1), glioma (n = 1).

In conclusion, spinal neoplasia should be considered in cases with progressive neurological signs, and MRI is a satisfactory diagnostic tool to diagnose spinal tumors. Operative management can be suggested for intradural – extramedullary spinal tumors according to the surgical outcomes,

MAGNETIC RESONANCE FEATURES OF CENTRAL NERVOUS SYSTEM LYMPHOMA IN DOGS & CATS. V Palus[1], HA Volk[2], CR Lamb,[2] GB Cherubini[1]. [1]Dick White Referrals, Six Mile Bottom UK, [2]The Royal Veterinary College, University of London, UK.

The magnetic resonance (MR) imaging features of central nervous system (CNS) lymphoma are well documented in humans; however there are few descriptions of such lesions in animals.

The aims of this study were to describe the MR imaging features of CNS lymphoma in a series of dogs and cats. Medical records for the period 2000–2009 were searched for animals with cytological or histopathological diagnosis of CNS lymphoma (multicentric, metastatic or primary) that had brain and/or spinal cord MR imaging (1.5 or 0.4 Tesla). MR imaging features including lesion location, signal intensity, lesion site, margins, oedema and mass effect were evaluated.

Twelve cases (8 dogs and 4 cats) fulfilled the criteria for inclusion. Five animals had primary CNS lymphoma and 7 had multicentric lymphoma with CNS involvement. Mean (range) age was 7.6 years (4–15 years. There were 7 males and 5 females. Onset of clinical signs was insidious. Neurological signs varied according to location of the lesion. On the basis of MR images, lesions were considered extra-axial in 7 cases, intra-axial in 3 cases and appeared to have both intra- and extra-axial components in 2 cases. Lesions affected the meninges (8 cases), spinal cord (4), frontal lobe (2), occipital lobe (1), temporal lobe (1), hippocampus (1), hypothalamus (1), metencephalon (1) and mesencephalon (1). Two animals with intracranial lymphoma had signs of local extracranial extension and 4 had retropharyngeal adenopathy.

Lesions were hypointense in T1-weighted (7/12), hyperintense in T2-weighted (12/12) and hyper/isointense in FLAIR (9/9) images compared to white matter. Margins were indistinct in T2-weighted images in 10/12 instances. Increased signal after gadolinium administration was evident in all lesions (12/12). Meningeal contrast uptake was found in 8 cases. Perilesional oedema was present in 7 cases. Mass effect was considered mild in 7 cases, moderate in 4 and marked in 1.

Lymphoma may affect a wide range of structures within and adjacent to the CNS. Lesions are hyperintense in T2-weighted images,

tend to have indistinct margins and signs of perilesional oedema, and consistently enhance after gadolinium administration. Although not specific, when combined with the history and neurological signs, these features aid presumptive diagnosis that should be confirmed by cytology or histopathology.

PERSISTENCE OF CANINE DISTEMPER VIRUS IN THE CNS IS ASSOCIATED WITH EFFICIENT DIRECT VIRAL CELL TO CELL SPREAD DIRECTED BY THE VIRAL ATTACHMENT PROTEIN IN THE ASTROCYTIC SYNCYTIAL NETWORK. Gaby Wyss-Fluehmann*, M. Vandevelde*, A. Zurbriggen°, P. Plattet°. *Department of Clinical Veterinary Medicine, Division of Neurology and °Department of Clinical Research and Veterinary Public Health, Vetsuisse Faculty, University of Bern, Switzerland

The mechanism of viral persistence, the driving force behind the chronic progession of inflammatory demyelination in canine distemper virus (CDV) infection, a model for human MS, is poorly understood. We monitored infection by a fluorescent wild CDV in living primary canine brain cell cultures by time lapsed confocal microscopy and videomicroscopy. The culture system closely mimics the white matter infection in vivo and preserves virulence of the agent. We show that CDV induces a non cytolytic infection of astrocytes with expression of nucleocapsids and surface proteins, which spreads from cell to cell along cell processes in a selective manner. Titration and agar overlay experiments as well as electronmicroscopy showed that spread is not mediated by infectious particles. Moreover, PCR showed that SLAM, the only known receptor for wild CDV, is not expressed in astrocytes. However, transmission of CDV infection clearly requires the viral attachment protein, since H knock out CDV variants were completely unable to spread. This implies the existence of an hithertoo unrecognized CDV receptor in the CNS. While binding of H to its receptor is known to activate the fusion protein, which we found to be amply expressed in infected astrocytes, syncytia, visible evidence of cell-cell fusion, were not detected in infected cultures. However, SLAM transfection of CDV infected brain cell cultures showed that viral H-F fusogenic complexes are expressed at the cell surface.

In view of previous reports, showing that fusion in virulent CDV is strictly controlled at multiple levels, we concluded that only very limited cell-cell fusion occurs, sufficient to allow transfer of infectious material. This conclusion is strongly supported by the demonstration of nucleocapids in cell processes connecting infected and target cells. Viral transfer occured from the tip of astrocytic processes to the cell body of target cells, frequently over long distances, bridging large uninfected areas of the culture. This selective pattern of spread strongly suggests that CDV uses pre-existing cell connections, known as the astrocytic syncytial network. Cell to cell spread of viruses evolved to escape immunedetection. In the case of distemper, immunedetection eventually does occur with formation of inflammatory demyelinating plaques.

The ability of CDV to spread efficiently in compact white matter rapidly covering long distances allows the virus to « outrun » the intrathecal antiviral immuneresponse and thus to persist in the CNS, continuously eliciting new lesions.

DETERMINATION OF IGA LEVELS IN SERUM AND CEREBROSPINAL FLUID: ASSESSMENT OF ITS DIAGNOSTIC VALUE FOR CANINE STEROID-RESPONSIVE MENINGITIS-ARTERITIS A. Maiolini[b], R. Carlson[a], M. Schwartz[a,b], G. Gandini[c], A. Tipold[a,b]. [a]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany; [b]Center for Systems Neuroscience, Hannover, Germany; [c]Veterinary Clinical Department, Faculty of Veterinary Medicine, University of Bologna, Italy

Steroid-responsive meningitis-arteritis (SRMA) is a systemic inflammatory disease of young dogs resulting from a dysregulation of the immune system. Previous neuroimmunological studies suggested that concomitant elevation of IgA levels in both serum and CSF are specific for SRMA throughout the different stages of the disease and also during long-term treatment with glucocorticosteroids. Other recent studies, however, raised concerns over the value of this test.

The purpose of this study was therefore to verify our previous results in a large number of cases and to determine the diagnostic value of IgA level testing in paired cerebrospinal fluid (CSF) and serum samples. We compared IgA levels of dogs with SRMA with dogs affected by various other diseases and calculated sensitivity and specificity of the test.

IgA content of 1050 canine CSF and serum samples were evaluated. Paired samples derived from 311 dogs with SRMA and 214 dogs with other diseases such as other central nervous system (CNS) inflammatory diseases (n = 34), neoplasia of the CNS (n = 46), idiopathic epilepsy (n = 42), intervertebral disc disease (n = 46) and diseases not affecting the CNS (n = 46).

Serum IgA levels were significantly higher in dogs in the acute form of SRMA in comparison to dogs with other diseases. This was also found evaluating IgA levels in the CSF, with the exception of the inflammatory CNS diseases, in which the IgA content did not differ significantly from those with SRMA. The sensitivity for simultaneous elevation of IgA levels in serum and CSF was 91% with a specificity of 78%, respectively.

Analyses of a large number of samples confirmed that IgA production is higher within the group of dogs with SRMA when compared to the remaining disease categories examined. Calculation of the diagnostic value of the determination of IgA confirmed that this test is still an important tool and highly sensitive to confirm the diagnosis of SRMA. Testing paired CSF and serum samples for IgA is recommended for the diagnostic work-up in suspected cases of SRMA, particularly in those animals that received glucocorticosteroids prior to CSF puncture. However, since the specificity is not high, other diseases causing neck pain have to be ruled out in the protracted form.

NEUROLOGICAL, IMAGING AND PATHOLOGICAL FEATURES OF SPINAL CORD HAEMANGIOMA IN TWO DOGS. P Jull[1], GL Walmsley[1], N Wenzlow[1], EL Rayner[1], BA Summers[1], GB Cherubini[2], S Schöniger[1], HA Volk[1]. [1]The Royal Veterinary College, London, UK. [2]The Dick White Referrals, Six Mile Bottom, UK.

Primary vascular neoplasia causing a myelopathy is rare in animals and man. The typical appearance on magnetic resonance (MR) imaging has been documented in humans; however, few descriptions exist in the veterinary literature. Here we present the clinical and imaging features of two canine cases diagnosed *post mortem* as capillary and cavernous haemangioma.

A 7 year old, female spayed German Shepherd dog (GSD) and a 4 year old, female spayed Labrador Retriever (LR) were presented with progressive T3-L3 myelopathy over 7 months and 10 days respectively. In both cases MR imaging of the spinal cord revealed a focal intra-medullary, spherical mass situated over the middle of the vertebral body of T7 or T5 (GSD and LR respectively). The capillary haemangioma (GSD) was iso-hyperintense to the spinal cord parenchyma on T2 and T1-weighted images with a strong contrast-enhancement on T1-weighted images. The cavernous haemangioma in the LR was predominantly hyperintense on T1 and T2-weighted images but had a clear ring, which was strongly hyperintense on T1 and hypointense on T2-weighted images consistent with prior haemorrhage. There was minimal enhancement following contrast administration. The LR was euthanized after imaging. The GSD was treated with lomustine 80 mg/m^2 every 5 weeks and physiotherapy for 15 months prior to euthanasia. A repeat MR imaging scan was performed after 4 months and the appearance of the lesion was unchanged. At gross *post mortem* examination, lesion identified on MR imaging corresponded to a focal, expansile, well-demarcated intra-medullary mass. Histopathologically both dogs had non-encapsulated masses composed of discrete small-calibre (GSD) or large sized, cavernous (LR) vascular channels, consistent with a capillary and a cavernous haemangioma respectively. Neoplastic endothelial cells of both tumours stained positive for von Willebrand factor. The capillary haemangioma contained also smooth muscle actin positive cells, pericytic or mycocytic components.

Haemangiomas are scarely mentioned in the literature as a differential for a myelopathy. MR imaging findings of a focal, spherical mass situated over the mid-vertebral body may be suggestive; cavernous haemangiomas in particular may also have a characteristic ring of hypointensity on T2 and hyperintensity on T1-weighted images consistent with blood breakdown products. These lesions have a predilection for the cervical and thoracic spinal cord in man and both cases presented here were in the mid thoracic spine.

CHRONIC TRAUMATIC BRAIN INJURY IN A DOG. J. Hordeaux[1], J. L. Thibaud[2], S. Laurent[1], F. Delisle, S. Blot[2] and M. A. Colle[1]. [1]Unité d'Anatomie Pathologique, UMR 703 INRA/ENVN, Ecole Nationale Vétérinaire de Nantes, France. [2]Unité Médicale et Chirurgicale de Neurologie, Laboratoire de Neurobiologie, Ecole Nationale Vétérinaire d'Alfort, France.

Chronic Traumatic Brain Injury is rarely encountered in humans and has not previously been documented in dogs. This report describes the case of a 2-year-old female American Staffordshire terrier that was referred for several episodes of four-limb ataxia, decreased vigilance and disorientation following repeated aggressions and physical abuses from its owner. A diffuse cortical lesion was suspected. CSF analysis revealed a neutrophilic pleocytosis and computed tomography showed a widening of cerebral sulci and a bilateral ventriculomegaly. At the owners' request, the dog was humanely euthanized. Necropsy revealed narrowing of the cerebral cortical gyri and a consequent widening of the sulci, consistent with bilateral diffuse cortical atrophy.

Microscopically, there were chronic subarachnoid hemorrhages and the cortical subpial layer displayed spongiosis, capillary hyperplasia, astrocytosis, microgliosis, and frequent neuronal necrosis occuring in a characteristic laminar pattern. This histopathological pattern of damage differs significantly from what had previously been described in people suffering from repeated traumatic brain injuries over a long period of time. This may be due to the important masticatory muscle mass at the top of dogs' heads which may prevent bone lesion. To the authors' knowledge, this is the first report of clinical features and pathological lesions of canine Chronic Traumatic Brain Injury.

ELECTROPHYSIOLOGICAL AND HISTOPATHOLOGICAL CHANGES OF THE EAR AND AUDITORY PATHWAYS IN THE MUCOPOLYSACCHARIDOSIS VII DOG. J. Hordeaux[1], S. Laurent[1], J. Amiaud[1], P. Costiou[2], Y. Cherel[1], and M-A Colle[1]. [1]INRA UMR 703, Ecole Nationale Vétérinaire, Nantes, France; [2]Anatomie des animaux domestiques, Ecole Nationale Vétérinaire, Nantes, France.

Mucopolysacharidosis type VII (MPSVII) is a lysosomal storage disease in which a genetic defect of β-glucuronidase induces a multisystemic glycosaminoglycan accumulation, singularly affecting the central nervous system. Children with mucopolysaccharidosis commonly have profound and poorly understood hearing loss including both conductive and neurological components. The purpose of this study is to describe the electrophysiological and histopathologic changes of the ear and the auditory brain pathway in MPS VII dogs in order to clarify the pathogenesis of hearing dysfunction.

For that purpose, pathological changes in middle and inner ears, and brain auditory pathways were assessed in 4 MPSVII beagle dogs ranging from 2 to 5.6 months old. Moreover, brainstem auditory evoked potentials (BAEPs) were recorded in one of those dogs. Epoxy embedded semi-thin sections and paraffin sections of middle and inner ears and brainstem were examined. We investigated middle and inner ear lesions as well as neuronal lysosomal storage and white matter lesions in the cochlear nucleus, the trapezoid body, the olivary complex, the lateral lemniscus, the inferior colliculus and the medial geniculate nucleus.

We observed a mild to moderate otitis media. Lysosomal storage was detected within cells of tympanic membrane, ossicles, bulla tympanica and cochlear bones, spiral limbus, spiral ligament, stria vascularis, basilar and Reissner membrane, endothelial cells of vessels, perivascular macrophages, neurons in the spiral ganglion, and support cells in the organ of Corti. No lesion was observed within the hair cells. We noted a slight to moderate neuronal storage in auditory nuclei and axonal spheroids in the trapezoid body and the lateral lemniscus. BAEPs analysis showed a mixed conductive and retrocochlear hearing loss that correlated with middle ear and brainstem axonal lesions. This mixed hearing impairment corresponds to what is described in patients; we therefore conclude that the dog is a good model to investigate mucopolysaccharidosis related deafness.

FUCOSIDOSIS IN A DOMESTIC SHORT HAIRED CAT. Arrol **LP and Smith PM**. University of Liverpool, Small Animal Teaching Hospital, Leahurst, Chester High Road, Neston, Wirral CH64 7TE, UK

A 2 year old female neutered domestic short-haired cat was presented for investigation of acute onset incoordination and tremor around 10 days previously, with little change in clinical signs since this time. A general physical examination revealed a slightly irregular left kidney but was otherwise unremarkable; neurological examination showed a generalized tremor and a wide-based stance at rest, with ataxia and hypermetria when walking. Proprioceptive positioning was normal in all four limbs but hopping responses were impaired; spinal and cranial nerve reflexes were intact but the menace responses were impaired in both eyes. A retinal examination proved unremarkable.

These findings indicated cerebellar dysfunction. An MRI scan of the brain showed a diffuse hyperintensity of the white matter throughout the brain on T2-weighted scans, with poor distinction between grey and white matter. On T1-weighted scans, there were bilaterally symmetrical hyperintense regions in the rostral thalamus and in the mesencephalon. A cerebrospinal fluid sample taken at the same time was normal. Aspirates of lymph nodes, which were not enlarged, showed a proportion of lymphocytes to contain small, clear vacuoles; similar vacuoles were seen in lymphocytes identified in a blood smear. An ultrasound scan of the abdomen was normal with the exception of the kidneys, which showed heterogeneous echogenicity throughout parenchymal tissue and very poor distinction between cortex and medulla. Biopsies of the liver and kidney showed diffuse vacuolation of the renal tubular epithelium and of hepatic parenchymal cells and hydropic degeneration of some hepatocytes; vacuoles were often periodic acid Schiff positive. A white cell lysosomal enzyme screen was then performed on a blood sample, revealing negligible alpha-fucosidase activity compared with reference values; activity of a variety of other lysosomal enzymes was not significantly different from control values. Whilst it seems likely that most currently recognized lysosomal storage diseases will eventually be recognized in domestic animals, fucosidosis has not been described previously in cats.

BRAIN ABSCESS FROM EMBOLIC PSEUDOMONAS AERUGINOSA ENDOCARDITIS IN A DOG Espino L, Rodríguez D, Schmeckenbecher1 C, Barreiro D, Failde D, Bravo A, Santamarina G. Departamento de Ciencias Clínicas Veterinarias. USC, Lugo, Spain.

Bacterial endocarditis in dogs and humans can cause thromboembolic disease affecting multiple organ systems. Bacterial brain abscesses developing by haematogenous spread from a remote source of infection are very rare in dogs. This report describes the clinical presentation and pathological features in a dog that developed a brain abscess associated with a bacterial endocarditis due to *Pseudomonas aeruginosa*.

An 8 year old male Griffon was referred to our hospital with a history of apathy of a week of duration and two generalized seizures the day before. Four months ago the dog had been treated from a fracture of os penis during a hunting trip. On presentation the dog was depressed and moderately dehydrated. A grade IV/VI systolic murmur was auscultated over the left cardiac apex. A severe leukocytosis (WBC 50.61 K/mL) with neutrophilic predomination (36.97 K/mL) was evident on a complete blood count. In addition

the biochemical panel developed a mild elevation in total proteins (8.5 g/dL) and alkaline phosphatase (373 U/L). Thoracic radiograph showed a mild enlargement of the cardiac silhouette and the ECG demonstrated a sinus rhythm of 120 bpm and noted evidences of both left atrial and ventricular enlargement. An echocardiograph study demonstrated endocarditis of the mitral valve and enlargement of left heart. After 12 hours of stabilization, the neurological exam noted depressed mental status, left hemiparesis and the head turned to the right, absent propioception in the left side and absent of the flexor reflex in the left anterior leg. These signs located the lesion in the right supratentorial area. At this moment, the owner rejected to continue with therapy and chose to have the dog euthanized. Necropsy revealed severe, suppurative and vegetative endocarditis of the left atrioventricular valve with numerous intralesional gram-negative bacilli and an abscess in the right cerebral hemisphere. Bacterial culture of a sample of both tissues yielded growth of *Pseudomonas aeruginosa*.

Intraparenchymal brain abscess formation is very rare in dogs. Infection can occur following penetrating injury, through extension of infection from adjacent structures or by haematogenous spread. Central nervous system (CNS) thromboembolic disease secondary to bacterial endocarditis in dogs has been rarely reported. Commonly implicated bacteria in canine endocarditis include *Streptococcus spp*., *Staphylococcus spp*. and *E. coli*. In this case, *Pseudomonas aeruginosa* was demonstrated the causal agent, being one of the few cases reported of this microorganism causing endocarditis and one of the few with a brain abscess as a complication.

BACTERIAL DISKOSPONDYLITIS IN MINK (MUSTELA VISON): A RETROSPECTIVE STUDY OF 10 CASES. Espino L[1], Fernández-Antonio R[2], Nieto JM[1], López-Peña M[1], Astorga J[3], Fidalgo LE[1], Barreiro A[1]. [1]Departamento de Ciencias Clínicas Veterinarias. USC, Lugo, Spain. [2]NUPE S.L., A Coruña, Spain. [3]Clínica Veterinaria Santa Cruz, A Coruña, Spain.

Diskospondylitis is an inflammatory condition of the intervertebral disk and the associated end plates of the adjacent vertebrae.[1,2] This condition is relatively uncommon in farmed mink with a yearly incidence from 0.17% to 2%.[7] Although a general description of the etiology and pathogenesis of the disease was published in a previous report, there is little information about the radiology and clinical findings of this disease.[7] The purposes of this study were to describe the clinical features and the contrast radiographic and CT findings of minks with bacterial diskospondylitis.

The animals that were studied included a total of 10 mink, 8 males and 2 females, all of which were between 8 and 14 weeks of age. Dorsoventral and lateral radiographs of the entire spine were taken from all minks. Lumbar mielography and computed tomography examination were performed in 3 animals. Routine bacterial cultures of liver, kidney, intestine, urine, blood and the disk material of the affected intervertebral site were done on all the animals. The most common systemic clinical signs included anorexia and loss of general body condition. Fever was observed only in one case. Neurological deficits were noted in all the animals and included propioceptive deficits (10 animals), hind limb paresis (7), tetraparesis (2), hind limb paralysis with no deep pain perception (1) and urinary incontinence (3). Radiographic findings included focal loss of a definable intervertebral disk space with collapse of the immediately adjacent vertebral bodies, accompanied by moderate lytic and proliferative bony changes. Affected vertebral bodies were shortened and in 2 cases vertebral subluxation and dorsal deviation of the spine was observed (Figure 1). The majority of the animals had a single lesion (7 of 10). The distribution of lesions was as follows: C2-C3 (1), C6-C7 (5), T2-T3 (1), T5-T6 (1), T6-T7 (1), T7-T8 (2), and T9-T10 (1). Two minks had multiple lesions limited either to the thoracic or to the cervical region. One mink had lesions in both the cervical and the thoracic regions. Contrast radiographic studies showed that compression of the spinal cord was mainly soft tissue in nature, but subluxation of the adjacent vertebrae and bone formation also contributed to these lesions (Figure 2). Computed tomographic findings included marked lytic lesions with irregular active bone proliferation along the ventral aspects of the affected vertebrae (Figure 3). Samples from biopsy specimens of affected disks yielded microbial growth in 7 of 10 samples. Streptococcus spp (4) and Sthapylococcus spp (3) were the microbial agents isolated.

Infections of vertebral column tissues may include vertebral physitis, spondylitis, diskitis and diskospondylitis.[2] Diskospondylitis is an inflammatory condition of the intervertebral disk and the associated end plates of the adjacent vertebrae.[1,2] Diskospondylitis primarily affects large and giant-breed dogs, and affected dogs are most often middle-aged.[1,2,6] In contrast, diskospondylitis in minks is usually diagnosed in young animals and appears to be more similar clinically and anatomically to juvenile diskitis[3] seen in humans than to diskospondylitis observed in dogs.[1,2] Severe neurologic deficits are frequent in this disease due to the presence of vertebral instability and subluxation. Late recognition of a problem by farmers results in minks having more advanced disease at the time of initial examination, compared with the stage of disease typically seen in small animals when first examined. Any intervertebral disk can be affected, although cervical area is most often involved. This finding differs from results of a previous report;[7] however, because radiographs of the entire spine were not available in the previous report, it is possible that the number of cervical disks affected was higher than reported. Diskospondylitis is most commonly caused by Streptococcus spp and Staphylococcus spp in the mink as occurs in human and canine diskospondylitis.[2,4,5,8] Hematogenous spread of organisms from a primary focus of infection to vertebral endplates and disks is thought to be the cause of autogenous diskospondylitis. Incriminated sources of infection are the urogenital tract, skin infections, dental diseases and endocarditis. However, in our study, the source of infection was not identified.

ANATOMICAL DISTRIBUTION OF SYRINGOMYELIA IN CAVALIER KING CHARLES SPANIEL WITH CHIARI-LIKE MALFORMATION. S Loderstedt[1], L Benigni[1], K Chandler[1], C Lamb[1], C Rusbridge[2], HA Volk[1]. [1]Royal Veterinary College, London, UK; [2]Stone Lion Veterinary Hospital, Goddard Veterinary Group, London, UK.

Chiari type 1 malformation in humans and, the comparable disease in Cavalier King Charles Spaniels (CKCS), Chiari-like malformation (CM) have been associated with the development of syringomyelia (SM). The relationship between CM and the development of SM is not fully understood, and there remains a lack of data about the prevalence and anatomical distribution of SM along the spinal cord.

The objective of this study was to evaluate the prevalence and anatomical distribution of SM in clinically-affected CKCS. It was hypothesised that (1) SM is not restricted to the cervical spine, (2) the maximal syrinx diameter can occur anywhere along the spinal cord, (3) there is an association between cervical syrinx diameter and distribution and SM in other regions of the spinal cord.

Thirty-seven CKCS with clinical evidence of SM were studied prospectively. Dogs with evidence of other types of myelopathy, ear disease or heart failure were excluded. Magnetic resonance (MR) imaging of the brain and the entire spinal cord of each dog were performed at 1.5 Tesla. SM was defined based on intramedullary foci with signal compatible with cerebrospinal fluid. The maximal dorsoventral dimension of SM in T1-weighted images was measured over each vertebral body and divided by the depth of the body of C3 to produce an index of syrinx diameter applicable to dogs of differing size.

SM was not limited to the cervical region. SM was present in the region of the C1-C4 vertebral bodies in all dogs (100%), C5-T1 in 31/37 (84%), T2-L2 in 29/37(78%) and L3-L6 in 21/37 (57%). Maximal SM diameter occurred at C1-C4 in 19/37 (51%) dogs, at C5-T1 in 2/37 (5%), at T2-L2 in 15/37 (41%) and at L3-L6 in 1/37(3%) dogs, respectively. There was no significant difference between the mean syrinx diameter at the region C1-C4 (0.33 ± 0.17), C5-T1 (0.27 ± 0.24) and T2-L2 (0.31 ± 0.26), but mean syrinx diameter was less in the region L3-L6 (0.08 ± 0.11) compared to the aforementioned regions ($p < 0.05$). Mean syrinx diameter at C1-C4 was positively correlated with mean syrinx diameter at C5-T1 (Spearman $r = 0.78$), T2-L2 (Spearman $r = 0.38$) and L3-L6 (Spearman $r = 0.35$) respectively.

Many CKCS with a cranial cervical syrinx also have a syrinx affecting more caudal regions of the spinal cord. Dogs with a large cranial cervical syrinx tend to also have a large syrinx more caudal. MR imaging restricted to the cervical region will underestimate the extent of the syrinx and the severity of the disease process in the majority of dogs.

NEUROLOGICAL, IMAGING AND PATHOLOGICAL FEATURES OF A LEPTOMENINGEAL INFLAMMATORY PSEUDOTUMOR IN A MALTESE TERRIER. S Loderstedt[1], GL Walmsley[1], BA Summers[1], R Casppello[2], HA Volk[1]. [1]The Royal Veterinary College, London, UK. [2]North Down Referrals, Bletchingley, UK.

Well recognized in human medicine, inflammatory pseudotumour (IPT) is a controversial focal mass lesion that has acquired several names; originally viewed as inflammatory, it is now thought to be a plasma cell, lymphocyte and eosinophil rich myofibroblastic tumor (WHO, 2002). IPTs have been infrequently described in veterinary medicine. IPT causing progressive myelopathy has not been reported in dogs and is rare in humans. Here we present the clinical, imaging and pathological features of a spinal IPT in a 5 year 8 month old, male entire Maltese Terrier.

The patient was presented with a three-week history of progressive pelvic limb gait abnormality. Neurological examination revealed an ambulatory paraparesis and pelvic limb ataxia, decreased postural reactions in the pelvic limbs, normal to increased spinal segmental reflexes and focal pain response on palpation over the thoracolumbar junction. Neuroanatomical localisation was T3-L3 spinal cord segments. The myelogram showed a golf-tee sign at the dorsal contrast column at the level of T13/L1, most consistent with an intradural extramedullary mass. The magnetic resonance (MR) imaging revealed a spherical, intradural extramedullary mass lesion hypointense to the spinal cord parenchyma on T2 weighted images, isointense on T1 weighted images and enhancing homogenously after contrast administration on T1 weighted images with the evidence of a dural tail. Lumbar cerebrospinal fluid (CSF) analysis revealed a mixed white blood cell pleocytosis 33/µl (< 5/µl) and an increase in protein 0.64 g/l (0.35 g/l). Quantitative PCR for *Toxoplasma gondii, Neospora caninum* and canine distemper virus of CSF was negative. A T13/L1 dorsal laminectomy, durotomy and excisional biopsy of the mass were performed. Histopathological examination showed congested, cellular tissue with infiltration of a dense mixture of lymphocytes, macrophages and neutrophils. Mature collagen, suggesting meningeal origin was seen in a trichrome stain. Grocott and PAS stains did not reveal fungal hyphae, parasite larvae, plant material or vasculitic changes. The pathological diagnosis of an inflammatory pseudotumor was made. Prednisolone 1 mg/kg twice a day was prescribed and tapered 50% every 4–6 weeks over a total of 5 months. The patient showed continuous improvement and fully recovered neurologically function. Repeated MR 5.5, 8 and 21 months post-surgery showed a moderate kyphosis at the thoracolumbar junction, but no signs of regrowth of the IPT.

IPT is a very rare cause of myelopathy in dogs, which could be considered as a differential for a focal intradural, extramedullary lesion with pleocytosis and elevated CSF protein content and might be treated successfully by surgical excision combined with immunomodulatory treatment.

BEHAVIOURAL CHANGES IN DOGS ASSOCIATED WITH THE DEVELOPMENT OF IDIOPATHIC EPILEPSY. N Shihab[1], J Bowen[1,2], HA Volk[1]. [1]The Royal Veterinary College, London, UK. [2]Imperial College, London, UK

Idiopathic epilepsy is the most common chronic canine and human neurological disorder. In human medicine and in animal models, epilepsy has been associated with a variety of neurobehavioural abnormalities. The current study was designed to test the hypothesis that behavioural changes are seen in canine patients with the development of idiopathic epilepsy. An additional aim was to identify seizure and patient phenotypes that might be associated with behaviour change.

Clinical records at a UK referral hospital were searched for canine patients with a recurrent seizure disorder. Inclusion in the study required the dogs to be diagnosed with idiopathic epilepsy on the basis of unremarkable inter-ictal neurological examination, haematology, biochemistry, magnetic resonance imaging of the brain and cerebrospinal fluid analysis. Each owner was contacted individually and was asked a modified and validated behavioural questionnaire (Hsu and Serpell, 2003). Additional data was collected with the use of a second questionnaire designed to obtain a consistent, unbiased patient history as formerly reported (Holt et al, 2005). Data collected included: age of dogs at first seizure, seizure type, frequency, and anti-seizure medication.

Eighty dogs met the inclusion criteria for the study and had complete records. The mean age was 63.75 ± 3.5 months (12–142), 52 dogs were male (29 neutered) and 28 female (21 neutered). Principal axis factoring was used to explore any latent structure within the behavioural questionnaire data, and identified several factors. In the drug-naive group (n = 26) post onset scores were significantly increased ($p < 0.05$), compared to pre-onset, for the following factors: fear/anxiety ($\bar{x}_{pre} = 0.27 \pm 0.09$; $\bar{x}_{post}$ 0.42 ± 0.13), defensive aggression ($\bar{x}_{pre} = 0.30 \pm 0.09$; $\bar{x}_{post}$ 0.39 ± 0.11) and perception changes ($\bar{x}_{pre} = 0.04 \pm 0.02$; $\bar{x}_{post}$ 0.14 ± 0.06). In the group receiving anti-seizure medication (n = 54) significant differences were still present in fear/anxiety ($\bar{x}_{pre} = 0.18 \pm 0.05$; $\bar{x}_{post}$ 0.37 ± 0.09) and perception ($\bar{x}_{pre} = 0.13 \pm 0.04$; $\bar{x}_{post}$ 0.51 ± 0.07). Additional significant differences ($p < 0.05$) recorded in this group were: abnormal reactivity ($\bar{x}_{pre} = 0.46 \pm 0.09$; $\bar{x}_{post}$ 0.79 ± 0.12), attachment disorders ($\bar{x}_{pre} = 0.26 \pm 0.06$; $\bar{x}_{post}$ 0.48 ± 0.08), dementia ($\bar{x}_{pre} = 0.08 \pm 0.03$; $\bar{x}_{post}$ 0.42 ± 0.07) and apathy ($\bar{x}_{pre} = 0.11 \pm 0.04$; $\bar{x}_{post}$ 0.27 ± 0.05). These differences were irrespective of seizure type or sex, and there was no significant relationship between seizure frequency and behavioural traits apart from a positive association with defensive aggression (Spearman r = 0.2778).

We conclude that behaviour changes, such as an increase in fear/anxiety, altered perception and an increase in defensive aggression, may be associated with the development of epilepsy in dogs. The possibility of this co-morbidity should be considered when treating dogs with epilepsy.

CLINICAL MANIFESTATIONS OF IDIOPATHIC EPILEPSY IN BORDER COLLIES. V. Huelsmeyer[1], R. Zimmermann[1], C. Brauer[2], C. Sauter-Louis[3], A. Fischer[1]. [1]Section of Neurology, Clinic of Small Animal Medicine, Ludwig-Maximilians University, Munich, Germany; [2]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Germany; [3]Clinic for Ruminants, Ludwig-Maximilians University, Munich, Germany

There is a lack of data on idiopathic epilepsy (IE) in Border Collies (BCs) in the current literature. Hypothesis of this study was that IE occurs in BCs and manifests often with severe clinical signs and poor response to medical treatment.

Forty-nine BCs diagnosed with IE were identified based on the review of medical records, age at onset and detailed questionnaires fulfilled by the owners. Subsequent phenotypic case classification was performed by evaluation of seizure history and treatment data (active epilepsy, remission; mild, moderate or severe clinical course; pharmacoresistant, not pharmacoresistant). Possible predictors for the development of a certain phenotype were statistically analyzed. Pedigrees were matched for the appearance of common ancestors.

Clinical manifestations were dominated by moderate (33%) and severe clinical courses (49%) defined by the occurrence of cluster seizures or status epilepticus, respectively. Pharmacoresistance was apparent in 71% of 24 dogs treated with ≥ 2 antiepileptic drugs. The epilepsy remission rate was 18%. Dogs in remission showed a significantly higher median age at onset and a significantly lower initial seizure frequency compared to dogs with active epilepsy. Survival time was significantly reduced in dogs aged < 2 years at seizure onset and in dogs with severe clinical courses. Pedigree analyses indicated a strong genetic founder effect in the appearance of epilepsy, resembling autosomal recessive inheritance.

In conclusion, IE occurs in BCs and is frequently associated with severe clinical signs and pharmacoresistance. While further genetic research is required, the results of this study suggest a substantial hereditary (disease) component.

THREE CASES OF SPINAL NEPHROBLASTOMA IN YOUNG DOGS. M le Chevoir[1], JL Thibaud[1], P Moissonnier[1], F Deslisle[2], P Devauchelle[2], F Crespeau[1], C Escriou[1], A Uriarte[1], S Blot[1]. 1: Ecole Vétérinaire d'Alfort; 2: CCV, 94700 Maisons Alfort, France.

Spinal Nephroblastoma or "thoracolumbar spinal cord tumor of young dogs" is an uncommon neoplasia usually found in young dogs, especially large breed ones. In the last 5 years, three cases of canine spinal nephroblastomas have been operated in our institu-

tion. Informations pertaining clinical signs, MRI description, treatment and long term follow up are described here.

Three dogs (25–30 kg), from 17- to 27-months old, were presented for symmetric (dog 1) or asymmetric (dogs 2 and 3) ambulatory paraparesis deteriorating from 1 to 5 months. MRI examination disclosed a hyperintense T2-weighted images (WI) and T1-WI intradural/extramedullar (dog 1) and an intramedullar (dogs 2 and 3) lesion between T10 and L1.

This lesion had T1WI contrast enhancement after gadolinium delivery. Moreover an ill defined intramedullar T2-hyperintense area surrounding the tumor was registered in dogs 1 and 3. This ill defined intramedullar T2-hyperintense area was also present in dog 2 and localized cranially to the lesion. Following anamnesis and MRI results spinal nephroblastoma was suspected in each case.

Surgical approach included dorsal laminectomy and durotomy. Cleavage plane between tumor and normal spinal cord tissues was found successfully in dogs 1 and 2. Histological examination revealed a "triphasic pattern" of mesenchymal, epithelial and blastemal cells. At that time a diagnosis of spinal nephroblastoma was established. Dogs 1 and 3 were ambulatory at 1 and 5 days after surgery respectively. Dog 2 did not recover deep pain and was euthanized 12 days after surgery. Three months after surgery, dog 1 relapsed and was re-operated. A successful recovery was achieved until he died 15 months after the first surgery due to a suspected pulmonary thromboembolism. Due to the unsatisfactory exeresis in dog 3, radiation therapy was initiated 4 weeks after surgery. Neurological and MRI examinations undergone at 3, 5, 9 and 13 months (dog 3) did not exhibited any tumor recurrence.

Little information is available for MRI characteristics of canine spinal Nephroblastoma. This report describes a new feature that is the spontaneous hyperintensity in T1-WI. Moreover, we describe the first surgical excision followed by radiation therapy with long clinical and MRI follow up. Published data confirm the similar histopathological features between canine spinal nephroblastoma and Wilm's tumor in children which are known to be responsive to radiation therapy. Such treatment is in particularly recommended in extrarenal Wilm's tumor. Moreover, Wilm's tumor is known to be chemosensible. Consecutively, in order to reduce radiation side effects, chemotherapy could be of interest for the treatment of such tumors.

In conclusion, surgical approach of canine spinal nephroblastomas can lead to good long term outcome especially if it is followed by radiation therapy. Nevertheless more cases are needed in order to confirm this hypothesis.

POST SURGICAL EVOLUTION OF MRI FINDINGS IN DOGS WITH ACUTE THORACOLUMBAR DISC DISEASE AND CORRELATION TO CLINICAL STATUS. Thomas Dayer[1], Daniela Gorgas[1], Diana Henke[2], Patrick Kircher[1], J. Lang[1] and Franck Forterre[3]. Division of [1]Clinical Radiology, [2]Neurology and [3]Surgery, Department of Clinical Veterinary Medicine, Vetsuisse faculty Bern, Switzerland.

In acute cases of thoracolumbar intervertebral disc disease in chondrodystrophic dogs MRI is helpful to assess exact location of herniated disc material, degree of spinal cord compression and to detect the character and extent of intraparenchymal spinal cord disease. Recent studies investigated the potential of MRI to provide prognostic factors in dogs with disc extrusion by evaluating the pre surgical MRI and the evolution of the neurologic deficits. However, the correlation between spinal cord changes on MRI and clinical signs is still under debate. The aim of the present prospective study was to assess possible correlations between pre and post surgical MRI findings and clinical status and to describe the evolution of changes within the spinal cord.

Dogs were included if thoracolumbar disc extrusion with compression of the spinal cord was confirmed with MRI and followed by hemilaminectomy. A second MRI examination a minimum of 4 weeks after surgery was performed and images were evaluated concerning extent of compression and changes of signal intensity (SI) within the spinal cord in T2 weighted images. Increase of SI was assessed by drawing regions of interest (ROI) on transverse images using Osiris (Osiris version 4.19 (Digital Imaging Unit (UIN) HUG; University of Geneva; Switzerland)). ROIs were set over the spinal cord at the level of compression and cranial to the lesion. The average intensity of the pixel values was determined.

Eighteen dogs were included in the study. MRI findings and neurological grade were not correlated between corresponding exams. However, high SI of the spinal cord at the preoperative examination was correlated with lower neurological grade in the post surgical examination. Overall, the SI changes within the spinal cord did not change significantly over time despite clinical improvement.

High SI within the spinal cord seems to be associated with outcome and did not disappear within weeks after surgery. Because of the inability to differentiate chronic and acute parenchymal pathologies of the spinal cord with MRI, interpretation of high SI weeks or months after a spinal cord injury is difficult and should not or not necessarily be interpreted as sign of acute spinal cord injury.

F-WAVES OF THE ULNAR NERVE IN CLINICALLY NORMAL CATS. Ravera M., Melis G., Bianchi E., Dondi M. Animal Health Department, Faculty of Veterinary Medicine - University of Parma, Italy.

F waves are late responses that reflect the integrity of the entire motor fibre from the motor neuron to the terminal branch. Evaluation of F waves is an important step of a thorough electrophysiologic assessment. Object of this study was to establish the normal values of F waves parameters for the ulnar nerve in the cat.

Twelve clinically normal cats (7 females and 5 males) 10 months to 5 years old, anesthetized for neutering procedures, were evaluated in this study. F waves were recorded in the palmar interosseus muscles by stimulation of the ulnar nerve at the carpus. Rectal temperature was maintained above 36.5° C during the entire procedure. Two trains of 11 stimuli were recorded in all the cats. Parameters evaluated included latency, maximum amplitude, frequency and duration of F waves. Other parameters measured were F ratio, calculated by the formula (latency F wave – latency M wave) – 1/2 X latency M wave, and F wave conduction velocity, calculated by the formula ulnar nerve length X 2/latency F wave – latency M wave – 1. Only one ulnar nerve for each subject was studied. Descriptive statistics were calculated for the parameters evaluated. The study design was approved by the University of Parma Ethics Committee for the protection of animals used for experimental purposes and data were collected with owner informed consent.

F waves of all the nerves tested in the study were recorded. The mean F wave latency was 8.66 ± 0.47 ms, the mean maximum amplitude of F waves was 1.84 ± 0.90 mV, the mean F wave frequency was $94.09 \pm 4.90\%$, the mean F wave duration was 5.63 ± 3.16 ms. The mean F ratio was 1.98 ± 0.38, while the mean F wave conduction velocity was 90.30 ± 10.74 m/s.

To the authors' knowledge there is only one previous paper from Knecht and colleagues (1985) on feline ulnar F waves. The parameters evaluated in that report were F wave latency and velocity. The further data provided by the present study may assure a more complete and accurate electrodiagnostic evaluation of peripheral motor disorders. Evaluation of F waves is an important part of the electrodiagnostic examination of small animals. This test precisely assesses motor nerve conduction and is especially used to evaluate the ventral nerve root and the most proximal segment of the nerve. In fact, these structures can't be investigated with routine motor nerve conduction studies. Furthermore F waves frequency reflects the motor neuron excitability. Therefore, the availability of normative values for all the nerves commonly used for clinical neurophysiology investigations is essential to reach a correct interpretation of electrodiagnostic findings. This is especially true in generalized neuromuscular disorders in which the contralateral limb of the same patient can't be used as a normal control. More cats, and subjects of different ages should be tested to further improve the normative data available for the neurologist.

INTRANEURAL PERINEURIOMA IN A DOG: CLINICAL AND DIAGNOSTIC FEATURES. Cornelis Ine[1], Chiers Koen[1], Kramer Martin[2], Ducatelle Richard[1], D'Herde Katharina[3], De Decker Steven[1], Van Ham Luc[1]. [1]Faculty of Veterinary Medicine and [3]Faculty of Medicine and Health sciences, Ghent University, Merelbeke, Belgium; [2]Justus-Liebig University, Giessen, Germany

A perineurioma is a rare benign peripheral nerve sheath neoplasm composed exclusively of perineurial cells. It represents only 1% of

all nervous system tumors in humans and has been described only once in dogs (Higgins et al., 2006). Although elektron microscopy is the diagnostic modality of choice, histopathology and immunohistochemistry are important diagnostic tools. In this report, the clinical and diagnostic features of a second case of canine intraneural perineurioma are presented.

A 4 year old male Leonberger was presented with a 2 year history of left thoracic limb lameness. The physical and orthopedic examinations demonstrated a very painful palpation of the mediopalmar side of radius and ulna. Survey radiographs of the affected limb were unremarkable. Neurologic examination revealed left thoracic limb lameness with pronounced muscle atrophy, mainly over the scapula. Ultrasonography demonstrated an echogenic homogenous thickening of the median nerve. Electromyography revealed spontaneous activity consisting of fibrillation potentials and positive sharp waves restricted to the flexor carpi radialis muscle. Peripheral magnetic stimulation showed no physical interruption of the affected nerve. Surgical exposure revealed a distally thickened median nerve. The lesion was excised and submitted for histopathological examination.

Histological examination of HE-sections revealed an irregularly enlarged, hypercellular nerve fascicle. The neoplastic tissue consisted of spindle-shaped cells arranged in pseudo-onion bulb-like whorls around axons. This pattern is typical and distinguishes the intraneural form from the three other types of perineurioma. Electron microscopy showed several cells with a discontinuous basal membrane and some containing pinocytotic vesicles, confirming their perineural nature.

Immunohistochemically, neoplastic cells were negative for S-100 and positive for laminin and claudin-1. Claudin-1 is a specific marker for perineurial tissue. More canine perineuriomas should be examined to confirm the consistency of claudin-1 expression.

AN ORIGINAL METHOD FOR THE VALIDATION OF ANTIBODY USE IN IMMUNOHISTOCHEMISTRY ON DOMESTIC ANIMAL ARCHIVAL MATERIAL. Ruel H and Poncelet L. Free University of Brussels, Brussels, Belgium

Possible epitope differences among species and epitope alteration during sample processing make antibody use validation mandatory. Cell cultures are species-specific and are amenable to controlled biological process alterations. A method for processing cultured cells of feline origin in the same way as histological material (formalin fixation, paraffin embedding) has been designed and results were compared to those observed in feline tumour tissues for three antibodies useful in cell survival/death processes study.

Crandell feline kidney cells were cultured in a routine manner either on cover slips or in culture bottles. Cultures were treated either with thapsigargine or staurosporine or covered with serum free medium, inducing unfolded protein response, apoptosis and autophagy respectively; untreated cultures served as control. Coverslips were paraformadehyde fixed while bottle-cultured cells were harvested and pelleted. The pellets were formalin fixed, entrapped in an agarose gel, routinely processed for paraffin embedding and cut at 5 μm.

Sections were rehydrated and antigen retrieval was performed through microwave intermittent boiling in selected buffers. An indirect, peroxydase-antiperoxydase, diaminobenzidin immunostaining method was conducted using antibodies against immunoglobulin heavy chain-binding protein (BiP, 1/100), cleaved caspase 3 (1/1000, both from Cell Signaling) and microtubule associated protein light chain (LC3, 1/1000, Sigma).

Caspase 3, BiP and LC3 antibodies gave a cytoplasmic staining (the expected cellular localization) in many cells treated with their inductors (staurosporine, thapsigargine and serum starvation, respectively). The untreated cells remained negative. Similar stainings could be observed on archival feline material. Comparison with the better preserved morphology of coverslip cultured cells was important.

This method of agarose gel cell culture pellet entrapment allowed generating on purpose-designed control material.

NEUROTROPIC T-CELL LYMPHOMA AND NEUROLYMPHOMATOSIS IN A CAT. [1]Mandrioli L.,[2] Gandini G.,[1] Morini M.,[1] Bacci B.,[2] Bersan E.,[2] Biserni R.,[3] Calzolari C.,[2] Gentilini F.,[1] Bettini G. [1]Dpt of Veterinary Public Health and Animal Pathology, University of Bologna, Italy. [2]Veterinary Clinical Dpt, University of Bologna, Italy. [3]Genefast Lab, Castelnuovo Rangone, Modena, Italy

A 5 year old Domestic Short Hair spayed female cat was referred to the Veterinary Teaching Hospital of the Bologna University for a history of sudden onset of monoplegia of the left forelimb associated to jaw paralysis, not responsive to antibiotics and corticosteroid treatment. On neurological examination, the cat showed a severely depressed mental status, ambulatory tetraparesis and plantigrade stance of the pelvic limbs. Multiple cranial nerves deficits were registered. Neuroanatomical localization was consistent with a multifocal syndrome. Serologic test for FeLV was positive. CSF examination showed severe mononuclear pleocytosis and elevated protein concentration. On cytology the CSF specimens were highly cellular and almost exclusively composed by large, monomorphic lymphocytes with high N:C ratio, diffuse chromatin pattern and prominent nucleoli; mitoses were also readily evident.

Due to poor prognosis, the cat was euthanized. At necropsy, whitish soft 3–5 mm multilobulated masses were evident at the skull base, surrounding oculomotor nerves and semilunar ganglia, and around roots of thoracic spinal nerves. No other changes were grossly apparent.

Histology revealed a monotonous proliferation of medium-to-large lymphocytes, with scarce cytoplasm, round to oval nuclei and prominent central nucleoli infiltrating the nervous tissue, with multifocal leptomeningeal involvement, occasional perivascular cuffings, focal neuropilar infiltration. In the peripheral nervous system (PNS) there were marked degeneration and atrophy of ganglionic perikaria and of axons. Immunohistochemistry revealed strong and diffuse CD3 immunophenotype, demonstrating a T-cell lymphoma. Fragment analysis (GeneScanning) of feline TCR gene rearrangements evidenced an oligoclonal pattern with few peaks of similar height. Integrated Feline Leukemia provirus was detected by PCR.

The main feature of this case of T-cell lymphoma was the prevalent PNS involvement and this prompted similarities with a human entity called neurolymphomatosis. At our knowledge, feline neurotropic T-cell lymphoma with neurolymphomatosis has never been reported. This prompts the need to consider neurotropic lymphoma with neurolymphomatosis a potential differential diagnosis in cats with peripheral neuropathy.

CYCLOOXYGENASE-2 (COX-2) EXPRESSION IN FELINE INTRACRANIAL MENINGIOMAS. Mandrioli L.[1], Mandara M.T.[2], Brunetti B.[1], Bacci B.[1], Pavone S.[2], Gandini G.[3], Bettini G.[1] [1]Department of Veterinary Public Health and Animal Pathology, Alma Mater Studiorum University of Bologna, Italy; [2]Department of Biopathological Science and Hygiene of Animal and Food Productions, University of Perugia, Italy; [3]Veterinary Clinical Department, Alma Mater Studiorum University of Bologna, Italy.

Cyclooxygenase (COX, also called prostaglandin H synthase) is a bifunctional enzyme that catalyzes the conversion of arachidonic acid into prostaglandins. Currently, three COX isoenzymes are known: the COX-2 isoform is inducible and is expressed in normal cells, such as macrophages, in response to pro-inflammatory stimuli and in multiple types of cancer cells. Recent studies have demonstrated COX-2 expression in a variety of canine and feline tumors. Furthermore, strong COX-2 expression has been identified as a negative prognostic factor in several human neoplasms, as well as in canine and feline mammary tumors and in canine osteosarcoma.

In human neurology, COX-2 is frequently expressed in the brain during different pathologic conditions. Numerous data show the presence of COX-2 in the glioma-affected brain and has been observed also in the majority of meningiomas, although the increasing immunohistochemical COX-2 expression was not constantly associated to malignancy grade. Recently, COX-2 expression has been found also in 21 out of 24 canine intracranial meningiomas, with no significant association to tumor grade. COX-2 expression has not been investigated yet in feline meningiomas.

The aim of this study was to evaluate by immunohistochemistry the expression of COX-2 in a series of 35 intracranial feline men-

ingiomas (20 biopsy and 15 post mortem samples) classified histologically according to the WHO system. Nineteen were transitional, seven fibroblastic, four psammomatous, four malignant, one meningothelial. The histological grade was I in 24 and II in 11 cases. Eight cases were also investigated with MRI, and moderate peritumoral edema was evident in only one of them (transitional histotype, grade I). COX-2 immunohistochemistry was carried out using a rabbit polyclonal antibody (Cayman Chemical) and prompted immunopositivity in 15 out of 35 feline meningiomas (43%). The immunoreactivity was cytoplasmic with occasional perinuclear reinforcement, and spread multifocally throughout the sections in meningothelial nests and small cellular aggregates. Pearson's chi-square test did not show any correlation between COX-2 expression, tumor histotype and tumor grade.

These findings suggest that COX-2 is expressed in feline meningiomas at low levels. Further investigations are required to elucidate the role of COX-2 in the biological behavior of feline meningioma and the possible perspectives of COX-2 inhibitory treatment strategies.

USE OF FALX DEVIATION ON MRI AS A PROGNOSTIC INDICATOR FOR SURVIVAL IN NON-INFECTIOUS CANINE MENINGOENCEPHALITIS: 27 CASES. JM White, HL Barnes Heller. VCA Aurora Animal Hospital, Aurora, IL.

The aim of this study was to determine if deviation of the falx on MRI in non-infectious canine meningoencephalitis was a prognostic indicator for survival. Medical records of patients diagnosed with immune-mediated meningoencephalitis at VCA-AAH between December 1, 2005 and February 29, 2008 were examined. Criteria for inclusion in this study consisted of a positive spinal tap (protein greater than 21 mg/dL and/or WBC greater than 5 cells/uL), negative infectious disease titers and a brain MRI at the time of diagnosis. In 5 cases, elevations of Lyme titers were observed without additional clinical signs. Vaccine-associated increase was considered most likely, therefore these patients were included. Deviation of the falx from midline was measured (using iPACS viewer) on the axial T2 weighted image at the level of the interthalamic adhesion.

Twenty-seven patients met the inclusion criteria, with a 15:12 female:male ratio. Patients were separated into two groups: Group A, those not exhibiting a midline falx deviation (n = 13) and Group B, those exhibiting a midline falx deviation (n = 14). Patients were initially treated with oral prednisone (initial mean and median dosages for Group A: 2.06 and 2.04 mg/kg/day respectively with a range of 1.75–2.27 mg/kg/day; initial mean and median dosages for Group B: 2.04 and 2.06 mg/kg/day respectively with a range of 1.53–2.39 mg/kg/day). Adjunctive treatment with Cytosar was administered to one patient in each group when the response to oral prednisone was incomplete. In Group A, mean and median CSF protein levels were 119.0 and 44.5 mg/dL respectively; CSF mean and median WBC counts were 57.9 and 8.0 cells/uL respectively. In Group B, mean and median CSF protein levels were 84.7 and 88.0 mg/dL respectively; CSF mean and median WBC counts were 115.0 and 22.0 cells/uL respectively. No statistical difference existed between the groups with respect to CSF WBC or protein. A Kaplan Meier survival curve was performed (using XLSTAT) comparing survival between Group A and Group B. Patients were censored at time of final analysis (6/30/09). No statistical difference in survival was found between Group A and Group B ($p = 0.518$), however, Group A shows a trend towards prolonged survival when compared to Group B. This may indicate a statistical association given a larger sample size. Variables such as owner compliance administering medications and tolerance for clinical signs may have affected results.

OCCURRENCE OF CANINE HERPESVIRUS-1 IN THE CANINE VESTIBULAR LABYRINTH AND GANGLION. B Parzefall[1], A Fischer[2], A Blutke[1], W Schmahl[1], K Matiasek[3]. [1]Institute of Veterinary Pathology and [2]Section of Neurology, Clinic of Small Animal Medicine, Department of Veterinary Clinical Sciences, Ludwig-Maximilians University of Munich, Germany; [3]Animal Health Trust, Newmarket, UK

Reactivation of herpesviruses within the human vestibular ganglion (VG) is suspected to cause inflammation and sensory dysfunction that manifest in transient vestibulopathies such as Ramsay Hunt syndrome, benign paroxysmal vertigo, vestibular neuritis and Ménière's disease. Herpesviruses also have been considered to contribute to the aetiopathogenesis of canine idiopathic vestibular syndrome. This study was aimed to assess the overall prevalence of canine herpesvirus-1 (CHV-1) DNA in the VG and vestibular labyrinth (VL) using PCR.

DNA-extraction was performed on the VL, VG and trigeminal ganglion (TG), from 52 dogs, followed by amplification of a fragment of the canine GAPDH gene and the CHV-1 glycoprotein gene B. Clinical and pathological records were studied for clinical diseases, with special regards to vestibular dysfunction and vaccination status.

CHV-1 DNA was detected either unilaterally or bilaterally in the VL of 17% and the VG of 19% of 52 dogs. Furthermore, 12% of the dogs with available TG, showed a bilaterally infected TG. In two dogs CHV-1 DNA was detected in all investigated compartments. One pup presented with fatal CHV-1 infection whereas the other dogs suffered from systemic diseases not related to CHV-1 infection. Five dogs had presented clinically with vestibular dysfunction. Three of them showed CHV-1 DNA in the VL and/or VG whereas the remaining two dogs were tested negative. Vestibular signs of the CHV-1 positive dogs were attributed to intracranial neoplasia. None of the infected dogs had previously been vaccinated against CHV-1.

This study indicates that subclinical CHV-1 infection is common amongst the dog population and reactivation of the virus theoretically might be involved in transient vestibular dysfunction. However, presence of CHV-1 DNA does not shed light on the relevance of an infection as long as in situ techniques have not proven the a compromise of the host cells metabolism and gene expression.

NEUROOPHTHALMOLOGICAL CONSEQUENCES OF ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY (AVIDP) IN CHICKENS M Matas[1], S Bader[2], R Korbel[3], W Schmahl[2], B Kaspers[4], D Donaldson[1], K Matiasek[1]. [1]Animal Health Trust, Newmarket, UK; Institutes of [2]Veterinary Pathology, [3]Avian Diseases and [4]Veterinary Physiology, Ludwig-Maximilians University of Munich, Germany

Neuroophthalmological complications are common in the axonal subtype of the immune-mediated human Guillain Barré syndrome. Demyelinating neuropathies, on the other hand, rarely affect the eye in mammals whereas in chicken with AvIDP, oculomotor nerve damage is a striking feature. This study was aimed to provide further details on the involvement of cranial nerves III, IV and VI and their clinical relevance in AvIDP-affected birds. The investigation enrolled ten chicken suffering from the paralytic stage of AvIDP. Ten healthy animals were used as controls. A full ophthalmological examination was performed, including biomicroscopy, funduscopy and pupillary oscillometry. Postmortem examination was conducted on the central and peripheral nervous system in order to confirm type and stage of nerve involvement. Upon enucleation, the globes were immersed in Davidsoń s fixative followed by routine processing and sectioning in a sagittal plane. Epoxy embedded nerve sections, teasing preparation and electron microscopy was performed on the trunks of CN III, IV and VI. All histological changes of the nerves and eyes were recorded and graded. Clinically, all animals performed successfully in a maze test and showed normal menace responses. They were able to move their eyes voluntarily. Only the affected group showed occasional pupillary oscillations, but there was no significant anisocoria or impairment of pupillary light reflexes. Ophthalmological examination was unremarkable in all cases.

Histologically, all AvIDP-affected birds showed an inflammatory demyelination in at least two of the cranial nerves III, IV and VI. Furthermore, intraocular nerve involvement was seen in all paralytic chickens. It affected the iridal and ciliary intramuscular nerve branches moderately to markedly. Choroidal fascicles and those segments penetrating the sclera were mildly affected. Notably all but one control animal presented with mild, lymphoplasmocytic and histiocytic infiltration surrounding the uveal blood vessels without other, segregating lesions. Both extraocular and intraocular branches of the oculomotor nerve are consistently affected in AvIDP. Impairment of the eye movements is incomplete and depends on the concurrent involvement of CN IV and VI. Intraocular neuritis affects the myelinated fibres supporting ciliary Crampton and

Brücke muscles, pupillary sphincter and dilator. Thereby, pupillomotor dysfunction manifests as increased oscillations rather than anisocoria or impaired pupillary light reflexes. Involvement of the ciliary muscle innervation is supposed to affect the focussing ability. The antigenetic profile that renders the myelin sheath of these cranial nerves particularly attractive to the autoimmune attack remain to be determined in order to identify and combat the immunological triggers.

CAN GENE EXPRESSION PROFILING DISCRIMINATE BETWEEN RADIOSENSITIVE AND RADIORESISTANT MENINGIOMAS? Mike Starkey, Celine Courtay-Cahen, Luisa De Risio, Kaspar Matiasek, Simon Platt*. Animal Health Trust, Newmarket, UK; *University of Georgia, College of Veterinary Medicine, Athens, GA, USA

Meningiomas are the most common primary canine brain tumour. Surgery followed by radiotherapy represents an effective modality for dogs with intracranial meningiomas, but approximately one third of animals are insensitive to such therapy. In the absence of a test which can predict whether a dog will respond to adjuvant radiotherapy, we conducted a pilot study to investigate whether gene expression profiles may discriminate between radiosensitive and radioresistant meningiomas.

Meningioma samples were collected from dogs that underwent cytoreductive surgery and subsequently received a hypofractionated megavoltage radiotherapy protocol. Each tumour was categorised as exhibiting either a 'good' or 'poor' response to radiotherapy according to whether the progression-free survival time of the dog bearing the tumour was above/below the median progression-free survival time of dogs that receive the hypofractionated megavoltage radiotherapy protocol following surgery. A canine whole genome microarray was used to measure gene expression in 3 meningiomas that displayed a 'good response', and 3 meningiomas that displayed a 'poor' response, to surgery and radiotherapy.

We have identified genes that display statistically significant differential expression between radiosensitive and radioresistant meningiomas. Amongst the genes whose differential expression makes conceptual sense are Prostate apoptosis response 4 protein (Par-4), upregulated ($p < 0.001$) in the radiosensitive meningiomas, and Caspase 6 (CASP6) which is downregulated ($p < 0.001$) in the radioresistant meningiomas. Par-4 expression is associated with radiosensitivity in prostate cancer, whilst decreased CASP6 expression is associated with radioresistance in oesophageal cancer cell lines.

These preliminary results suggest that radiosensitive and radioresistant meningiomas may exhibit some consistent differences in gene expression.