European Society of Veterinary Neurology
European College of Veterinary Neurology

Research Abstracts 18th Annual ECVN & ESVN Symposium, Munich, Germany, 23rd – 24th September 2005

ABSTRACT #1

DAILY CONTROLLED PHYSIOTHERAPY INCREASES SURVIVAL TIME IN DOGS WITH SUSPECTED DEGENERATIVE MYELOPATHY. I Kathmann[1], S Cizinauskas[2], MG Doherr[3], F Steffen[4], A Jaggy[1]. [1]Department of Clinical Veterinary Medicine, Section of Neurology, University of Bern, Bremgartenstrasse 109a, 3012 Bern, Switzerland. [2]Neurology Service, Department of Clinical Veterinary Medicine, University of Helsinki, P.O. Box 57, Hämeentie 57, 00014 Helsinki, Finland. [3]Departement of Clincial Veterinary Medicine, Division of Clinical Research, University of Berne, Bremgartenstrasse 109a, 3012 Bern, Switzerland. [4]Vetsuisse Faculty, Departement for Small Animals, University of Zürich, Winterthurerstrasse 260, 8057 Zürich, Switzerland.

Degenerative myelopathy is a disease that especially affects older large breed dogs. The animals become progressively paraparetic over months until they are unable to walk and have to be euthanized. The cause is still unknown and no specific therapy exists. The purpose of this retrospective study was, firstly, to evaluate the breed distribution, age of onset and gender predisposition, as well as to describe the clinical presentation in 50 cases with degenerative myelopathy. Secondly, to evaluate whether the mean survival time was significantly affected by different means of physiotherapy performed in 22 dogs in which follow up was available. Thirdly, to look if neurological status, the anatomical localisation of the lesion or the age at onset had an influence on survival time.

The following criteria had to be fulfilled to be included in the study: 1. signalement of middle aged to older large breed dogs or mixed breed dogs over 20 kg of body weight, 2. complete history of a slowly progressive course of the disease, 3. neurological examination confirming paraparesis and ataxia in the rear in absence of spinal hyperesthesia on palpation and 4. normal results of diagnostic imaging (myelography (n = 48), CT (n = 1) or MRI (n = 1)) and normal CSF analysis (n = 44). Electromyographic examination of the muscles of all four limbs and the paravertebral muscles was performed in 23 cases.

At time of euthanasia, all dogs (n = 22) were non-ambulatory paraparetic and six dogs showed additionally urinary and fecal incontinence not lasting longer than 2–3 weeks prior to euthanasia

We found a significant ($p < 0.05$) breed predisposition for the German shepherd dog, Kuvasz, Hovawart and Bernese mountain dog. The mean age at diagnosis was 9.1 years, and both sexes were equally affected. The anatomical localisation of the lesion was spinal cord segment Th3 to L3 in 56% (n = 28) of the cases and L3 to S3 in 44% (n = 22) of the cases. Animals that received intensive (n = 9; gait exercise at least 3 to 5 times per day, additionally either 3 times per day massage and passive joint-movement or daily hydrotherapy) physiotherapy had a significant ($p < 0.05$) longer survival time (Ø = 255 days) when compared to animals with moderate (n = 6; gait exercise maximally three times per day and once a week hydrotherapy or massage; Ø = 130 days) or no (n = 7; Ø= 55,4 days) physiotherapy. In addition our results show that affected dogs that receive physiotherapy may remain ambulatory significantly ($p < 0.05$) longer than animals without physical treatment.

ABSTRACT #2

ARTICULAR PROCESS FRACTURES FOLLOWING LUMBOSACRAL DECOMPRESSIVE SURGERY. S Thiel[1], F Steffen[2], T Gödde[1]. [1]Tierärztliche Gemeinschaftspraxis Piding, Germany; [2]Tierspital Zürich, Switzerland.

L7/S1 laminectomy is widely accepted as treatment of choice for degenerative lumbosacral stenosis (DLSS) in dogs. The purpose of this retrospective study was to review clinical and radiological features of articular process fractures following lumbosacral decompressive surgery.

The records of six patients were evaluated. All dogs had been diagnosed with DLSS and/or disc protrusion L6/L7 or L7/S1 and were treated with dorsal laminectomy and dorsal fenestration of the disc. Chief complaint at the time of reevaluation in all dogs was a painful caudal lumbar vertebral column. Further work up included plain lateral and ventrodorsal X-rays of the lumbosacral junction in all dogs. The predominant radiologic finding was a radiolucent line on the base of the caudal articular process. The fracture line pointed in transversal direction and ran through the intervertebral foramen. Stress radiography with the lumbosacral joint in flexion greatly improved the visibility of the fracture line, as the fragment was subluxated in caudodorsal direction. Radiological diagnoses were unilateral or bilateral fractures of the caudal articular process of L7 or L6 in addition to a caudodorsal subluxation L6/L7 in one dog. In one dog, no pathologic radiological findings could be seen. In this dog, surgical exploration yielded the diagnosis of an unilateral fracture of the caudal articular process of L7.

Although rarely described in veterinary literature, articular process fractures should be considered as a possible complication in all patients that suffer from acute onset of pain following decompressive lumbosacral surgery. It is assumed, that L7/S1 laminectomy in combination with dorsal fenestration significantly increases the range of motion of this joint. Consequently the mechanical stress on the articular surfaces is increased. Partial surgical removal of the lamina of any vertebra removes the bony support interposed between the two articular processes. This is especially true for the caudal articular processes. Together with the ventrally situated intervertebral foramen, this leads to a point of significantly reduced mechanical strength and consequently the fractures can be located here. The fractures could not be seen on plain X-rays in all cases, which suggests that the problem may be underdiagnosed, especially if only one side is involved. To optimize postoperative stability, the authors recommend to completely spare the L7 lamina by dorsal laminectomy of S1 with extirpation of the Lig. flavum in flexed position. Furthermore it is important to spare the dorsal lamina of L7 in all patients, in which a dorsolateral foraminotomy at L7/S1 has to be performed.

ABSTRACT #3

THE CONTRIBUTION OF ELECTROSTIMULATION ON NERVE REGENERATION IN RABBITS WITH EXPERIMENTALLY INDUCED CRUSH INJURY. O Besalti[1], E Unlu[2], YS Sirin[1], I Ergin[1], T Onyay[1]. [1]Ankara University, Faculty of Veterinary Medicine, Department of Surgery, Ankara, Turkey; [2]Ministry of Health Ankara Diskapi Education Hospital, Department of Physical Therapy and Rehabilitation Ankara, Turkey.

The purpose of the study was to investigate the contribution of pulsed galvanic current stimulation on nerve regeneration in sciatic crush injury model in rabbits. Sixteen new Zealand male rabbits, allocated into two groups of equal size were subjected to this study. All rabbits were anaesthetized and their sciatic nerves were exposed, and distal motor nerve latency, compound muscle action potential recorded by the distal part of crush point (dCMAP), compound muscle action potential recorded by the proximal part of crush point (pCMAP) and nerve conduction velocity (NCV) were analyzed before and just after the crush injury. Electrostimulation was performed for 90 times every day for 3 weeks. At the 21st day all rabbits were anaesthetized and their sciatic nerve exposed and the same electrophysiological examination was carried out. Physiologic values

recorded after the sciatic nerve exposure and just before crush injury were 1.26 ± 0.14 msec, 38.57 ± 16.13mV, 43.35 ± 16.88mV, 74.18 ± 29.38m/s, and the values recorded just after the crush injury were 1.21 ± 0.12 msec, 36.28 ± 14.35 mV, 10.05 ± 7.03 mV, 58.36 ± 16.50 m/s. for dCMAP, pCMAP and NCV respectively. The values recorded at the 21th day for the experiment group were 1.29 ± 0.09 msec, 21.38 ± 11.72 mV, 21.23 ± 10.53 mV, 59.16 ± 20.60 m/s., and for the control group were 1.22 ± 0.18msec, 17.36 ± 7.05 mV, 19.79 ± 8.41 mV, 49.75 ± 7.17 m/s for distal motor latency, dCMAP, pCMAP and NCV respectively. Distal motor nerve latency was not significantly different between groups and intervals (physiologic, just after injury and after treatment). In the experiment group mean dCMAP was not significantly different between three intervals ($p < 0.05$). However in the control group, the differences between physiologic and just after crush injury mean dCMAP values were not significant but different from post treatment values ($p < 0.05$). Mean pCMAP was different at all intervals but there were no significant differences between groups ($p < 0.05$). Mean NCV was different between physiologic and crush values but there were no significant differences between groups. In conclusion the contribution of pulsed Galvanic current on nerve regeneration cannot be suggested. However, the higher level of dCMAP in experiment group at the end of the study can be accepted as a positive point for electrostimulation on nerve injury.

ABSTRACT #4

ZONISAMIDE ADD-ON THERAPY FOR CANINE REFRACTORY EPILEPSY: BENEFIT OR "HONEY –MOON EFFECT"? T Von Klopmann, D Simon, A Tipold. Dept. of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

Up to 30% of the dogs suffering from seizure activity are refractory to anticonvulsant treatment even if a combination of anticonvulsants is used. Nearly all of the newer anticonvulsive substances have a short half life in dogs so that phenobarbital and potassium bromide are still the medications of choice. An exception is the anticonvulsant zonisamide which possesses a half live of about 15 hours in dogs. The aim of the present study was to evaluate the outcome of the additional use of zonisamide in dogs with refractory epilepsy (no response to phenobarbital and/or potassium bromide treatment despite therapeutic serum levels).

14 dogs with refractory idiopathic epilepsy were treated with zonisamide as add-on therapy in a dosage of 10mg/kg body weight PO BID. The benefit of this add-on therapy was assessed over a time period of eight months. Evaluation criteria were the number of responders (reduction of seizure number of at least 50%), seizure type, seizure duration, and seizure severity. The seizure history was evaluated retrospectively for the last four months before initiation of zonisamide treatment and prospectively for the same time period under add-on therapy.

To date, 11 dogs completed the study and were evaluated. Nine of these dogs were responders with a significant reduction of the seizure frequency of 92.9%. Reduction of seizure frequency in all dogs including the non-responders was 72.4%. The median seizure frequency per month of all 11 dogs decreased from 5.75 seizure events before initiation of zonisamide therapy to 1.25 seizures per month with add-on therapy. In addition, a reduction in the median seizure duration from 2.6 minutes to one minute and a subjective decrease of the seizure severity was achieved. Three dogs became seizure free over the prospective study period of four months. Two dogs were non-responders and showed a seizure reduction of 14 and 25% only. In seven dogs a reduction of former anticonvulsant therapy was possible. Of the remaining 3 dogs one has received the add-on therapy with zonisamide for less than four months, one dog was euthanized immediately after initiation of zonisamide treatment on owner's request, and in one case no data could be obtained because of insufficient owner compliance.

The add-on therapy with zonisamide in dogs suffering from refractory epilepsy has a beneficial effect on seizure activity and seems to be a hopeful alternative in these critical cases. However, in the follow up period (> 4 months after initiation of zonisamide treatment) in three cases the seizure frequency increased again, which may represent a phenomenon known as "honey-moon effect". The use of zonisamide is cost intensive, therefore a widespread use is not to be expected. Whereas until recently zonisamide has to be imported from Japan, it is now also available in Germany and several other European countries.

ABSTRACT #5

ANAESTHESIA-TRIGGERED CEREBELLAR DYSFUNCTION IN CATS: NEUROPATHOLOGICAL FINDINGS IN ONE CASE. L Poncelet[1], M Shamir[2], O Chai[2], A Résibois[1]. [1]Anatomy/Embryology and Biochemistry/Nutrition, Free University of Brussels, Brussels, Belgium; [2]Veterinary Teaching Hospital, Koret School of Veterinary Medicine, the Hebrew University of Jerusalem, Rehovot, Israel.

A 13-year-old spayed female Domestic long haired cat was admitted for euthanasia due to incurable malignant mammary tumour. This cat was one of a series of 11 domestic long haired cats that developed acute but steady cerebellar ataxia on recovery from an uneventful general anaesthesia. The shared drug used in all cases was ketamine. In this cat the general anaesthesia was conducted for spaying 3.5 years prior to euthanasia and it was the only case available so far for necropsy.

The encephalon was removed and fixed in 10% buffered formalin. Classical stainings disclosed changes limited to the cerebellum. A severe loss of Purkinje cells was observed but granular and molecular layers were spared as were the cerebellar, pontine, lateral cuneiform and inferior olivary nuclei.

Immunostainings for calbindin pointed out that remaining Purkinje cells were mostly limited to the nodulus, uvula and floculus, where they were organized in patches. Antibody (Ab) against the neurofilament RT 97 evidenced numerous empty baskets. Anti glial fibrillary acidic protein Ab evidenced discrete gliosis of the cerebellar cortex in areas devoid of Purkinje cells. Contrasting with the situation in normal controls, remaining Purkinje cells were barely visible using the anti microtubule associated protein 2 (MAP 2) Ab, and their dendrites were not stained using the anti beta 3 tubuline Ab. Anti alpha tubuline however stained Purkinje cell main dendrite. In addition, neurons of the dorsal accessory nucleus of the inferior olive were not stained by the anti MAP 2 Ab. These features could not be retrieved in five different feline cerebellar degenerative entities, and are provisionally proposed to characterize the anaesthesia-triggered cerebellar degeneration in cats.

The origin of these changes in the immunoreactivity of two essential components of the cytoskeleton in subsets of neurones of the cerebellar system remains conjectural.

ABSTRACT #6

DANCING MALTESE TERRIER DISEASE. I Van Soens[1], SAE Van Meervenne[1], T Bilzer[2], M Tshamala[1], LML Van Ham[1]. [1]Department of Small Animal Medicine and Clinical Biology, University of Ghent, Belgium; [2]Department of Neuropathology, Heinrich-Heine University, Germany.

A 6-year-old female Maltese terrier had a 7-month history of alternately flexing and extending the pelvic limbs in a dancing motion. The dog was treated with anti-inflammatory drugs without improvement. Haematology, serum biochemistry, vertebral column radiography, myelography and electromyography were all within normal limits. On repetitive nerve stimulation (RNS), however, a decremental response of muscle action potentials was seen. A diagnosis of focal myasthenia gravis was made and the dog was treated with pyridostigmine bromide without improvement. At that time the dog was referred to our clinic for a second opinion.

General clinical examination was normal. During standing the dog was continuously flexing and extending the pelvic limbs. The dog walked in a normal manner but preferred to sit. Neurological examination revealed slightly increased patellar reflexes. Complete blood count, serum biochemistry, including creatine kinase, T4 and TSH were all within normal limits. Acetylcholine receptor antibody titers were negative. EMG showed positive sharp waves and fibrillation potentials in the M. gastrocnemius of both pelvic limbs, in the M. tibialis cranialis of the left pelvic limb and in the Mm. interossei of the four limbs. Tibial motor nerve conduction velocity (MNCV), RNS and sensory evoked potentials were all normal. In correspondence to Dancing Doberman Disease (Chrisman, 1990), a presumptive diagnosis of "Dancing Maltese Terrier disease" ("DMTD") was made.

Three weeks later the dog returned for muscle and nerve biopsy. Its condition, however, suddenly deteriorated three days before presentation to a weakness on all four limbs with still the dancing motion of the hind limbs. Neurological examination at that time revealed increased patellar reflexes and decreased withdrawal reflexes on the four limbs. On EMG examination positive sharp waves and fibrillation potentials were found in almost all muscles examined. Tibial MNCV was decreased (50, 6 m/s) and RNS was normal. At this time a presumptive diagnosis of "DMTD" complicated by an idiopathic polyradiculoneuropathy was made. Histological examination revealed severe peripheral nerve degeneration and consecutive neurogenic muscular atrophy; an axonopathy was suspected.

One month later the dog returned; the clinical symptoms of the polyneuropathy had disappeared but the dancing motion was still present. A treatment was started with gabapentine; after a few days of treatment only the dog's condition had normalised.

ABSTRACT #7

PORTOSYSTEMIC SHUNT ASSOCIATED WITH SEVERE EPISODIC WEAKNESS. A Wessmann[1], HA Volk[1], GD Shelton[2], K Chandler[1], R Cappello[1]. [1]The Queen Mother Hospital, The Royal Veterinary College, North Mymms, United Kingdom, [2]Department of Pathology, University of California, San Diego, La Jolla, CA, United States.

A two year old male neutered Yorkshire Terrier was presented to The Royal Veterinary College, London with a two month history of anorexia, weight loss, regurgitation and a period of ataxia and running movements with impaired consciousness suggestive of seizure activity for 20 minutes after food intake. Several weeks prior to presentation the dog developed exercise-induced episodes of severe weakness. The gait became short and choppy on exercise followed by ventroflexion of the neck before final collapse on all four limbs. The dog recovered fully at rest. The physical and neurological examinations in between episodes were normal. The lesion was localised to the lower motor neuron system and to the forebrain. Clinical pathology, such as haematology, biochemistry, fasted serum ammonia level, fructosamine, acetylcholine-receptor antibody, and pre-and post-exercise lactate levels were within normal limits. Pre- and post-prandial bile acids were increased and a coagulation profile revealed a prolonged activated partial thromboplastin time. An abdominal ultrasound revealed a small liver and a single extrahepatic portosystemic shunt. A barium swallow revealed a mild megaoesophagus. Electrodiagnostic studies and a nerve and muscle biopsy were unremarkable. The portosystemic shunt was partially reduced and three months later completely ligated. The weakness resolved completely following the initial ligation.

Liver diseases have been associated with lower motor neuron diseases in humans. For example peripheral neuropathy was present in 71% of human patients with end-stage liver disease (1) and portosystemic shunting had been proposed as a cause of hepatic neuropathy (2). Myasthenia gravis has also been reported in conjunction with liver diseases in humans (3). In veterinary medicine, acquired myasthenia gravis was previously diagnosed in a seven year old English setter suffering from cholangiocellular carcinoma and suspected to be a paraneoplastic syndrome (4). In this case, no apparent cause of the collapsing episodes, such as immune-mediated disease, neoplasia or cardiovascular abnormalities, were identified. To the authors' knowledge, portosystemic shunt has not previously been reported to be associated with collapsing episodes similar to myasthenia gravis or other neuromuscular junction disorders in dogs. It is possible that metabolites resulting from the abnormal shunt might theoretically have an effect on the function of peripheral nerves or neuromuscular transmission.

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ABSTRACT #8

FELINE SPONGIFORM ENCEPHALOPATHY: FIRST CASE REPORTED IN PORTUGAL. J Ribeiro[1], S Carmo[1], JF Silva[2], JJ Correia[2], L Orge[3], V Lorenzo[4]. [1]Clinica Veterinária ANI+, Lisboa, Portugal; [2]Faculdade de Medicina Veterinária, Lisboa, Portugal; [3]Laboratório Nacional de Investigação Veterinária, Lisboa, Portugal; [4]C.Veterinario Prado de Boadilla, Madrid, Spain.

Feline spongiform encephalopathy (FSE), a disease first reported in the UK in 1990 is believed to result from ingestion of food contaminated with the bovine spongiform encephalopathy (BSE) agent and subsequent accumulation of abnormal prion protein (PrP) in neural tissues. The purpose of this presentation is to describe the first case reported in Portugal, with special emphasis on clinical neurological aspects.

In January 2004, a 9 year old intact siamese queen was referred with a 3 month history of behavioural changes, polydipsia, gait abnormalities and episodes of hypersalivation. The initial neurological exam revealed disorientation, vestibular ataxia, bilateral mydriasis responsive to light, and hypersensitivity especially on the head. On blood testing there were elevations of triglycerides, cholesterol and bile acids, with normal transaminase activities and ammonia content. Hematologic results were also normal. Abdominal ultrasound showed a moderately hypoechogenic liver. An intestinal formula diet was instituted and all laboratory parameters were within normal limits after 3 weeks, but the neurological signs progressed and increasing episodes of hypersalivation, episodic depression, compulsive behaviour with polyphagia, and aggressive behaviour were observed. The ataxia became more pronounced, and tremors, both intentional and at rest, were evident. MRI of the brain and CSF analysis were performed with normal results. Clinical signs progressed to tetraparesis, unresponsive mydriasis and dementia, and euthanasia was performed.

On necropsy, brain and spinal cord were grossly normal, and the liver was pale. Histologic examination revealed vacuolization of the neuropil and neuronal perikaria in several regions of the CNS. Immunohistochemical detection of resistant prion protein (PrP^{res}), using monoclonal antibodies against PrP, SAF84 and 3F4 was performed on the CNS and showed a strong, widespread granular and linear deposition of PrP^{res}. These findings are consistent with a diagnosis of FSE, a transmissible spongiform encephalopathy or prion disease in cats, this being an infectious disease with a degenerative course. To the authors knowledge this is the first confirmed case of FSE reported in Portugal.

ABSTRACT #9

CELL THERAPY OF MUSCULAR DYSTROPHY BY MESOANGIOBLASTS GRAFTING IN THE DYSTROPHIC DOGS. N Granger[1], M Sampaolesi[2], GD Antona[2], A Innocenzi[2], R Tonlorenzi[2], A Finan[2], JL Thibaud[1], G Cossu[2], S Blot[1]. [1]Unité de Neurologie, Ecole Nationale Vétérinaire d'Alfort, France; [2]Stem Cell Research Institute, HSR, Milan, Italy.

Duchenne's muscular dystrophy (DMD) is a lethal childhood disease caused by mutations of the dystrophin gene. The dystrophin protein plays a vital role in skeletal muscles. The Golden Retriever muscular dystrophic dog (GRMD) develops a clinical course that is strikingly similar to human DMD, characterized by progressive muscular atrophy, degeneration and fibrosis. Mesoangioblasts are vessel-associated stem cells that participate in post-embryonic development of the mesodermic tissues including skeletal muscles. As mesoangioblasts maintain multipotency in culture and expand indefinitely, they appear to have potential for cell therapy in dystrophic processes. We used the GRMD dog as a model of DMD in a preclinical trial to evaluate the feasability, the safety and biological and functional benefits obtained through intra-arterial delivery of canine mesoangioblasts.

Two protocols were elaborated. Dog 1 received canine wild type mesoangioblasts isolated from a donor dog (heterologous transplantation) and dog 2 received its own genetically corrected mesoangioblasts, transduced with a lentiviral vector encoding for canine micro-dystrophin, a functional truncated form of the dystrophin protein (autologous transplantation). Mesoangioblasts were isolated and characterized from biceps femoralis biopsies, which were taken at 2 weeks of age. One hundred million and 50 million mesoangioblasts were injected through the femoral artery respectively in dog 1 and dog 2. The injections were repeated monthly 3 times. An immune suppression protocol was conducted in heterologous and autologous dogs for 5 months, covering the whole protocol, to prevent rejection of the grafted cells. Muscle biopsies (sartorius, biceps femoralis, tibialis and gastrocnemius) were taken 50 days after the last injection for histological, biochemical and physiological analysis.

We detected, in muscle biopsies, 4 to 8% of dystrophin positive fibers in dog 1 (control non treated GRMD dog: 0.02% to 0.5% of dystrophin positive fibers) and 2 to 3% in dog 2. Hematoxylin-eosin stainings revealed mild improvement of the morphology of treated muscles in comparison to the controlateral ones. Western blot analysis confirmed the expression of dystrophin or micro-dystrophin in muscles. Physiological analysis of single skinned fibers isolated from the gastrocnemius muscles showed an absolute force comparable to that observed in the wild type fibers, demonstrating the benefit of mesoangioblasts treatment.

Such promising cell therapy is now being evaluated in a larger number of GRMD dogs.

ABSTRACT #10

IN VIVO DYSTROPHIN RESCUE BY EXON-SKIPPING IN A CANINE MODEL OF DMD. A Vulin[1,2], A Goyenvalle[2], JL Thibaud[1], N Granger[1], L Garcia[2], S Blot[1]. [1]Laboratoire de Neurobiologie, Ecole Nationale Vétérinaire, Maisons-Alfort, France; [2]Genethon, CNRS UMR8115, Evry, France.

Among well described animal models of Duchenne Muscular Dystrophy (DMD), the *GRMD* dog represents the best model of DMD patients in terms of size and in the pathological expression of the disease. GRMD is caused by a 3′ splice-site point mutation in intron 6, which induces the skipping of exon 7 and thus results in a frame shift that prematurely aborts dystrophin synthesis. By forced exclusion (skipping) of two exons (6 and 8) in the case of GRMD, it is possible to restore an open reading frame. Antisense oligonucleotides (AO) allow specifically to target and inhibit individual genes for the treatment of the disorder. However, since the AO

are not self-renewed, they cannot achieve long term correction. To overcome this limitation, we have introduced antisense sequences into small nuclear RNAs (snRNA) and vectorized them in AAV vectors.

To determine efficient antisense oligonucleotides (AO), we tested in particular exon splicing enhancer (ESE) motifs, which target internal exon sequences, crucial to pre-mRNA splicing. We designed different 2′O-methyl oligoribonucleotides targeting either ESE or donor or acceptor splice sites of exons 6 and 8 in GRMD. These AONs were tested first in vitro. Then we have designed AAV vector harbouring chimeric U7 snRNA carrying efficient antisense sequences against exons 6 and 8 of the dog dystrophin gene and AAV were injected in GRMD.

Among different antisense oligonucleotides tested in GRMD, the combination of 2 AONs targeting respectively ESE from exon 6 and 8 induced Δ5–9 in-frame skipping. We have shown the efficacy of these ESE sequences for exon-skipping in culture myotubes. We also detected this correct exons-skipping after intramuscular injections of the AAV-U7-ESE6/8 vector in GRMD and observed a restoration of dystrophin expression. In order to spread the benefit to the entire limb, we are currently evaluating systemic delivery of the vector.

In this study, we provide evidence that efficient skipping of two exons can be achieved in vivo after intramuscular injections through U7snRNA shuttle. Theoretically over 75% of Duchenne patients could benefit from skipping of a single exon and multiexon skipping would significantly extend this percentage to most DMD mutations.

ABSTRACT #11

INTRAVASCULAR ADMINISTRATION OF DYSTROPHIN PLASMID DNA IN A CANINE MODEL OF DUCHENNE MUSCULAR DYSTROPHY: EARLY SIDE EFFECTS AND LONG TERM BIOLOGICAL EFFICACY. JL Thibaud[1], T Huss[2], J Hegge[3], C Thioudellet[2], N Granger[1], K Gnirs[1], J Wolff[3], S Braun[2/4], S Blot[1]. [1]Laboratoire de Neurobiologie, Ecole Vétérinaire d'Alfort, Maisons-Alfort, France; [2]Transgene, Stasbourg, France; [3]Mirus Corp., Madison,Wisconsin, USA; [4]Now at AFM, Association Française contre les Myopathies, Evry, France.

Muscular dystrophy represents a formidable challenge for gene therapy. Major hurdles include the need to correct large masses of tissue with minimal damage to the already inflamed and necrotic muscles, absence of immune rejection of the therapeutic protein and sustained expression. Amongst current strategies, plasmid DNA meets many of the prerequisites. Moreover, interest has increased with a new method of intravascular delivery showing widespread transfection of limb muscles in healthy large animals including non-human primates.

The present study was aimed at assessing the efficacy and tolerance of this new intravascular route of delivery for plasmid encoding canine full dystrophin cDNA in a canine model of muscular dystrophy, the Golden Retriever Muscular Dystrophic (GRMD) dog.

In 26 young anesthetized GRMD dogs, large volumes of plasmid were injected in the femoral and/or in the brachial arteries while all blood vessels leading into and out of the limb were transiently occluded. Six young anesthetized GRMD dogs were injected in the cephalic vein up to 10 times. Care was taken to control pain and all dogs were under anticlotting regimen. Clinical and biochemical parameters were daily controlled. Biopsies were performed at 1 week, 3 and 6 months after the last injection. Iatrogenic lesions and transgene expression were quantified on histology and the effects of immunosuppressive drugs on immune responses were studied.

Moderate weight-bearing lameness occurred after intra-arterial injections but disappeared within few hours. Clinical phenotype was not affected by the procedure and few clear elevations of serum CPK and AST were observed. Transgene expression was found broadly and at high level (best results after 10 IV administrations: 22% of dystrophin-positive fibers). Dystrophin-associated glycoproteins expression was also restored. Humoral immune response against dystrophin was found but was not detrimental for persistant dystrophin expression which was observed in delayed biopsies. Cellular immune response was not found.

Given the significant levels of dystrophin transduction, and transient side effects, intra-vascular delivery of genes is particularly attractive. The simplicity of the intravenous procedure and the absence of detrimental immune responses with the use of an immunosuppressive protocol allows sequential administrations to treat different territories and to improve the level of transduction.

ABSTRACT #12

OUTCOME FOLLOWING HYPOFRACTIONATED RADIOTHERAPY IN DOGS WITH EXTRA-AXIAL MIDDLE CRANIAL FOSSA MASS LESIONS ON MRI; 44 CASES. S.R.Platt[1], V. Adams[2], J.D. Garcia[3], S. Murphy,[1]F. McConnell[1], L. Wieczorek[1], A. de Stefani[1] A. Hayes.[1] [1]Centre for Small Animal Studies, [2]Centre for Preventive Medicine, The Animal Health Trust, Newmarket, UK, [3]University of Murcia, Spain.

Intracranial middle cranial fossa mass lesions identified on MRI can be due to a limited variety of tumours. Those that are classed as extra-axial with homogenous contrast enhancement may be pituitary adenomas, meningiomas or more rarely, lymphomas. Hypofractionated radiotherapy (RT) has not been comparatively evaluated for the treatment of these mass lesions in dogs. The purpose of this study was to evaluate the effect of hypofractionated RT on survival of dogs with a middle fossa mass when compared to non-specific palliative therapy such as corticosteroids and evaluate the dogs for potential risk factors.

Dogs with forebrain signs and a single extra-axial middle cranial fossa mass on MRI which homogenously contrast-enhanced, were evaluated. Information retrieved from medical records included signalment, presence of Cushing's and / or hypothyroidism, as well as neurological examination. Absolute and relative tumour height and volume were measured from two planes of the MRIs in 44 dogs. All dogs had a normal CSF and were graded 1–3 (mild to severe) based on neurological status. Group I dogs (n=23) were not treated or were prescribed with anti-inflammatory doses of prednisone (0.5–1.0 mg/kg/day). Group II dogs (n=21) were treated with 5 once weekly doses of RT (5,8,8,8,8Gy) to a total dose of 37Gy. Survival analysis was carried out using the Kaplan-Meier and Cox proportional hazards regression procedures in SAS. Survival rates among the groups were compared using log-rank tests with significance set at $P < 0.05$.

Dogs treated with radiation (MST = 928 days) survived significantly longer than dogs treated with steroids (MST = 46 days) (P=0.0004). After controlling for the effect of neurological grades, there remained a significant treatment difference (P = 0.02). Neurologic status was also significantly associated with survival (P=0.02). Signalment, relative tumour height or volume, or endocrine abnormalities were not associated with survial. Absolute tumour volume was a significant predictor of survival (P=0.002). Tumour volume or volume ratio was not associated with treatment choice.

The study demonstrates that hypofractionated RT may be beneficial for the treatment of dogs with MRI documented extra-axial middle fossa masses and neurological signs.

ABSTRACT #13

VASCULAR ENDOTHELIAL GROWTH FACTOR AND KI-67 EXPRESSION IN CANINE INTRACRANIAL MENINGIOMAS. LA Wieczorek[1], SR Platt[1], T Scase[1], J Miller[1], K Matiasek[2], F Adamo[3], S Long[4], V Adams[1]. [1]Animal Health Trust, Newmarket, Suffolk, UK, [2]Ludwigs Maximilians University, Munich, Germany, [3]University of Wisconsin, USA, [4]University of Glasgow, Scotland.

Meningiomas are one of the most common canine intracranial neoplasms and are thought to be rather slow growing. Tumour proliferation rate can be defined by the monoclonal antibody MIB-1 to the Ki-67 antigen (MIB-1 Labelling Index). Angiogenesis is essential for the growth, invasion and metastasis of solid neoplasms. Vascular endothelial growth factor (VEGF) is one of the factors responsible for angiogenesis. In a recent study we were able to demonstrate an association between VEGF expression and survival in dogs with intracranial meningiomas. The objectives of this study were to evaluate VEGF and Ki-67 expression in canine intracranial meningiomas, to determine whether there is an association between their expressions and to investigate association of Ki-67 expression with patient outcome.

Immunohistochemical staining for VEGF and Ki-67 expression was performed on 70 cases of histologically confirmed WHO grade I intracranial meningiomas. MIB-1 Labelling Index (LI) was calculated by determining the percentage of positive nuclei from at least 3000 cells using an automated counting via Image J NIH-software. Extent, intensity and distribution of VEGF-expression were assessed by light microscopy using a semi quantitative scale. Analysis of variance and nonparametric rank correlations cross tabulations and chi-squares were performed to identify associations between VEGF expression variables and MIB-1 LI. In 15 cases dogs underwent post-surgical radiotherapy and were followed until the time of death. Overall survival was compared to expression of Ki-67. Survival analysis was carried out using a Kaplan-Meier analysis.

VEGF and Ki67 expression were not detectable in 2 and 6 cases respectively. There were no significant associations identified between any of the VEGF expression parameters and MIB-1 LI. The median overall survival time was 460 days (95% confidence interval: 217–536). MIB-1 LI was not associated with survival (P=0.7).

This study suggests that there is no association between angiogenesis and tumour proliferation as defined by MIB-1 LI in canine meningiomas, although a larger case load would be required to demonstrate this with greater confidence. Even though MIB-1 antibody is a particularly reliable marker of cell proliferation and thus possibly of tumour aggressiveness in

human meningiomas, there was no association with outcome in our canine patients. This could be based on the low number of cases included in the survival study. Based on this study, angiogenesis seems to be a more important predictor of canine meningioma activity than Ki-67 expression.

ABSTRACT #14

MULTIVARIATE ANALYSIS OF INTRACRANIAL MENINGIOMAS AND GLIAL CELL TUMOURS IN 61 DOGS. A de Stefani[1], SR Platt[1], FJ Llabres[1], V Adams[1]. [1]Animal Health Trust, Newmarket, Suffolk, UK.

Brain tumours are a commonly recognized cause of neurological dysfunction in dogs. The nature and evolution of neurological signs resulting from brain tumours depend on tumour location and size and on the rate of tumour growth.

The aims of our study were to retrospectively evaluate histopathologically confirmed meningiomas and glial cell tumours and document (i) intracranial location, (ii) tumour to total brain volume ratio, (iii) frequency of occurrence of MRI detected pathology (oedema, intra-tumoural haemorrhage and parenchymal shift) and to investigate possible association of these factors with the presence of (a) seizure activity, (b) dementia, (c) visual deficits and (d) compulsive gait. The above variables for each tumour type were also compared.

Medical records of all dogs with intracranial neoplasia presented to the Animal Health Trust between 1993 and 2004 were reviewed. Inclusion criteria for this study were complete history, neurological examination, MRI study and histopathological confirmation of tumour type by surgical biopsy or post mortem examination; 61 dogs satisfied the inclusion criteria. Logistic regression and two-sample T-tests was performed to examine the relationships among clinical signs and MRI signs and to compare the relative tumour volume for cases with and without specific clinical signs. Significance was set at $P < 0.05$ for all tests. There were significant associations between seizures and tumour location for mengingiomas ($P=0.001$) and gliomas ($P=0.004$) both separately and together ($P < 0.0001$) as well as for compulsive gait and location for gliomas ($P=0.04$) alone and also for both tumours combined ($P < 0.01$). Dogs with tumours in brainstem, cerebellum, and hypothalamus were less likely to suffer from seizures compared to dogs with tumours in the frontal/olfactory lobes or the temporal/parietal/occipital lobes. Dogs with tumours in the caudal fossa were more likely to show a compulsive gait compared to dogs with tumours in the frontal/olfactory lobes or the temporal/parietal/occipital lobes. There were no significant associations found for presence of MRI pathology and clinical signs. There were no significant differences in the tumour volume ratio between groups of cases with and without specific clinical signs for mengiomas and gliomas, either separately or together. However, tumour type was significantly associated with tumour volume ratio ($P=0.01$). Gliomas took up a significantly larger proportion of the brain on average than meningiomas (mean difference = 2.8%, 95% CI: 0.68 – 4.91). The presence of dementia ($P=0.05$) and visual dysfunction ($P=0.05$) were associated with increased tumour volume ratio in gliomas only.

ABSTRACT #15

SURVIVAL TIMES IN 50 DOGS WITH INTRACRANIAL MASSES AFTER LOMUSTINE THERAPY. SAE Van Meervenne[1], I Van Soens[1], SFM Bhatti[1], IMVL Gielen[2], I Polis[1], JP de Vos[3], LML Van Ham[1]; [1]Department of Small Animal Medicine and Clinical Biology and [2]Department of Medical Imaging, Faculty of Veterinary Medicine, Ghent University, Belgium. [3]De Ottenhorst, Clinic for Companion Animal Medicine, Terneuzen, The Netherlands.

Survival times of 50 dogs with intracranial masses, treated with lomustine in the period 1999 – 2005, were retrospectively analysed.

A presumptive diagnosis of intracranial neoplasia was made by CT-scan imaging, and exclusion of other brain disorders. Dogs were treated with lomustine (90 mg/m^2 p.o.) every 4 to 6 weeks. At the beginning of therapy, prednisolone was administered for 3 weeks. Dogs suffering from seizures also received phenobarbital orally (2,5 mg/kg b.i.d.). Myelosuppression was evaluated by a CBC 1, and if necessary 3 weeks after lomustine administration. Routine serum biochemistry determinations for liver and kidney function were performed every 6 months. In five dogs, a follow-up CT-scan was made 3 months after diagnosis.

Based on survival times, dogs could be divided into 3 groups. The first group of 19 dogs received lomustine only once. The mean and median survival times were 21 and 9 days respectively. Dogs of this group showed no clinical response to chemotherapy. The second group of 28 dogs was treated 2 – 13 times with lomustine. In this group 21 dogs were euthanized after a mean and median survival time of 7,8 and 6,6 months respectively, and 7 dogs still are alive 2 to 8 months after start of therapy. This group responded well to the lomustine protocol. The third group of 3 dogs was treated with lomustine (1 – 16 times) combined with radiotherapy. One dog of this group died soon after the radiation therapy, one dog survived for 25 months and one dog is still alive 3,5 years after start of treatment. This group, although limited in number, seemed to respond very well to this combination protocol, especially when compared to previous studies on radiotherapy alone.

In our study, multimodality therapy with a lomustine - radiation therapy protocol seems to offer the best results. A difference in sensitivity to chemotherapy for different types of brain tumours, as has been documented in human neuro-oncology, is suspected. Further investigation should confirm this conclusion. If possible, a histopathologic diagnosis of tumour type should be obtained through biopsies, before treatment planning of the different types of brain tumours.

ABSTRACT #16

MOTOR NEURON DISEASE IN A CAT: CLINICAL AND NEUROPATHOLOGICAL FINDINGS. F Gernone[1], C Salvadori[2], R Biserni[1], C Cantile[2], G Gandini[1]. [1]Department of Veterinary Clinical Sciences, Faculty of Veterinary Medicine - University of Bologna (Italy); [2]Department of Animal Pathology – Faculty of Veterinary Medicine - University of Pisa (Italy).

This report describes the clinical and pathological findings of a 6-year-old male neutered domestic cat. The cat, living in a farm with other not related, clinically normal cats, was referred because of severe tetraparesis, bilateral contracture of carpal flexors, absence of spinal reflexes and difficulty in opening the mouth. The disease quickly progressed to a permanent non-ambulatory status and in the last stage of the disease dramatic generalized muscle atrophy was observed. Cranial nerves were normal despite of severe masticatory muscles atrophy.

Biochemistry profile showed increased CK, ALT, AST. CSF revealed a mild mononuclear pleocytosis and increased proteins. Diffuse fibrillation potentials were recorded on EMG. Muscles biopsy showed neurogenic muscle atrophy and severe depletion of the intramuscular myelinated fibers with axonal fragmentation and myelin ovoid formation. On the basis of the severity of clinical signs and poor prognosis, the cat was euthanized upon request of the owners.

On necropsy, spinal cord lesions were characterized by severe loss of ventral horn neurons, accompanied with reactive gliosis. There was severe degeneration of the descending tracts of all segments of the spinal cord, whereas the ascending tracts were normal. Ventral nerve roots were markedly degenerated with axonal loss and presence of macrophages laden with myelin and axonal debris. Dorsal nerve roots were normal. In the brain stem, loss of neurons of tegmental area and nuclei of the extrapyramidal system was accompanied by severe reactive gliosis. No significant lesions were observed in other areas of the CNS and extraneural tissues.

Pathological findings were suggestive of a motor neuron disease pattern. A primary neurodegenerative condition or a toxic pathogenesis was suspected.

ABSTRACT #17

SUBOCCIPITAL CRANIECTOMY CAN SAVE A LION. Merav Shamir[1], Yael Shilo[1], Orit Chai[1], Ram Raifen[2], Alon Fridman[3], Limor Miara[4]. [1]Koret School of Veterinary Medicine the Hebrew University of Jerusalem. [2]Faculty of Agriculture The Hebrew University of Jerusalem [3]Ben-Gurion Medical School. [4]Chai-Kef Zoological garden, Rishon - Letzion.

Malformations of the skull bones, predominately the occipital bone, may cause compression of nervous tissue at the caudal fossa and may lead to herniation of the vermis, as has been reported in over 30 lions living in captivity. Such malformations are thought to be caused by deficiency of Vitamin A and among wild felides, were reported to occur only in lions.

Neurological signs of generalized ataxia, head tremor, falling, stumbling and inability to walk or stand are some of the more characteristic features of this disease that so far lead to the death of the affected lions.

We report a case of 14 months old male Barberian cross lion *(Panthera leo)* that had reduced appetite for a week before he developed neurological signs compatible with cerebellar and cranial spinal cord lesions. CT scan confirmed the diagnosis of bone malformation and thickening of mainly the occipital bone and the tentorum cerebelli applying pressure to the cerebellum, medulla and the first segment of the spinal cord. Vitamin A concentrations were measured in the serum and in the liver and were found

to be extremely low. Only 26 µg/dl of vitamin A (retinol) were found in the serum (normal range is > 75 µg/dl) and 5.6 µg/g was found in the liver (normal value 5400 IU/g). A conservative treatment of 1 mg/kg/day of steroids and vitamin A supplementation of 500000 IU/day were given for a month and were only beneficial for a short time before the neurological signs worsened as soon as the steroids were tapered down. Suboccipital craniectomy was performed using a high-speed surgairtome 5 weeks after the initial diagnosis. The surgery was aimed to enlarge the foramen magnum and expose the entire caudal aspect of the cerebellum in order to reduce the pressure over the area. This was confirmed to be accomplished by post operative CT scanning. Recovery was fast and uneventful and the lion was able to stand again the next morning and improved gradually to a point of complete normal performance 4 weeks after the surgery. Additional follow-up examinations were done every month by the same neurologist and no neurological deficits were ever recorded during the total of 7 months follow-up period.

In a search for reference values for the abnormal CT and vitamin A (retinol) levels found in our lion, 4 other lions, age 1.5 –10 years, one male and three females from two different zoological gardens were joined to the study. Blood levels of vitamin A were measured in all of them, liver biopsy was done in two and CT scan in two.

Low levels of Vitamin A in the serum were found in all lions tested (4/4) and were ranged between 16.9 to 30.86 µg/dl. Liver biopsy revealed extremely low levels of vitamin A of 1.4 and 1.744 µg/g in both lions tested. The CT scan of one young lioness showed mild changes of the same bones as was observed in the first operated lion. The other CT was completely normal.

Our findings regarding blood levels of vitamin A is in agreement with the published literature. It is known that wild felid in captivity especially lions have low serum vitamin A concentration. The normal value of 5400 IU/g in the liver referred to in the veterinary literature is only based on one lion from the wild. Liver vitamin A concentrations of healthy lions in captivity, as was done in our study, were not reported before. Low levels of vitamin A such as 0–30 IU/g was previously reported in stillbirth and lion cubs that died from the condition [1] but also in lions that died from different reasons and had no neurological abnormalities.

We assume that sub-clinical hypovitaminosis A is a very common finding in the lion population living in captivity with only a small portion of them developing clinical signs.

Whether it is genetic variation or nutritional differences that cause only some of the lions to develop clinical and pathological abnormalities it is still to be investigated. Over 60 lions were reported so far to suffer and die from hypovitaminosis A related clinical syndromes. Half of them were presented with neurological signs. This, we believe, is only a small part of the real picture. A more in-depth research is currently taking place in order to understand the real nutritional needs of vitamin A in lions and its mechanism of absorption. Cases of clinical hypovitaminosis A were reported from zoological gardens that fed the lions as recommended with vitamin A supplementation of about 9000 IU/day for a grown up lion. It may be that the vitamin supplementation powder manufactured for lions in captivity is not well given or not well absorbed in all cases.

We recommend that any case of neurological signs in a lion especially if four limb ataxia, vestibular or cerebellar signs predominate, should be CT scanned. If malformations of the occipital, tentorium cerebelli and basisphenoid bones are found to be the cause and the condition dose not respond to conservative therapy than suboccipital craniectomy can still save the lion.

ABSTRACT #18

EVALUATION OF ELECTRODIAGNOSTIC EXAMINATION TECHNIQUES IN CLINICAL PRACTICE. S Thiel[1], K Matiasek[2], A Fischer[1]. [1]Section of Neurology, Clinic of Small Animal Medicine & [2]Institute of Veterinary Pathology & Neuropathology, Ludwig-Maximilians-University, Munich, Germany.

Electrodiagnostic examination techniques are used commonly in veterinary practice as a screening tool to identify generalized neuromuscular disease and congenital deafness. Generalized or focal neuromuscular disease is identified by distribution of spontaneous electric activity within the musculature (EMG) and then further classified by evaluation of motor nerve conduction velocity, amplitude, duration and morphology of the compound muscle action potential and repetitive nerve stimulation whereas brainstem auditory evoked responses (BAER) serve to evaluate peripheral hearing and the brainstem auditory pathway.

The objective of this retrospective study was to investigate the contribution of electromyography (EMG), electroneurography (ENG) and brainstem auditory evoked responses (BAER) to the final clinical diagnosis in dogs and cats.

The medical records of 628 dogs and cats (1995–2004) with electrodiagnostic examinations were evaluated. For each indication to perform electrodiagnostic examination, the electrodiagnostic diagnosis was compared with the final clinical diagnosis. Sensitivity (S) and positive predictive value (PPW) of the electrodiagnostic examination were established for lumbosacral syndrome (n=16), generalized neuromuscular disease (n=60), and focal neuromuscular disease (n=58).

Results showed that electrodiagnostic examination was decisive for the diagnosis of congenital deafness, acquired deafness, and focal neuromuscular disease. The electrodiagnostic examination further characterized the disease and contributed to the final diagnosis in the following indications: BAER for investigation of vestibular disease, suspected brainstem disease, Jack Russell terrier hereditary ataxia and facial paralysis; EMG/ENG for further investigation of lameness, suspected lumbosacral disease, generalized neuromuscular disease, and masticatory muscle myositis. EMG/ENG was of minor value for investigation of UMN paraparesis and tetraparesis.

Sensitivity of the electrodiagnostic examination was 87.5% for lumbosacral syndrome, 98.3% for focal neuromuscular disease and 91.6% for generalized neuromuscular disease. PPW of the electrodiagnostic examination was 66.7% for lumbosacral syndrome, 100% for focal neuropathy and 85.3% for generalized neuromuscular disease. Within the latter group polymyopathies were specifically diagnosed with S= 63.6%, polyneuropathies with S=90.9%, and neuromuscular transmission disorders with S= 75 %.

In conclusion, there exists a broad range of indications to perform electrodiagnostic examination. Furthermore, this study provided objective data to assess sensitivity and positive predictive value of the electrodiagnostic examination for the specific diagnosis of a variety of neuromuscular disorders.

ABSTRACT #19

PUDENDAL NERVE TERMINAL MOTOR LATENCY IN THE DOG. GB Cherubini , R Cappello, D Brodbelt. The Queen Mother Hospital for Animals - The Royal Veterinary College, London, UK.

The Pudendal nerve (PN) derives from S1–S3 in most of the dogs. It is a mixed nerve, transmitting somatosensory impulses from the pelvic diaphragm, the external urethral and anal sphincter, the skin of the anus, perineum and genitalia and carrying motor fiber to the pelvic diaphragm, the external anal and urethral sphincters, penis, vagina, clitorides, elevator ani, and coccygeous muscles, and proximal tail muscles. The PN is also an important branch of the autonomic, parasympathetic and sympathetic system. Dysfunction of the PN is associated with urinary and faecal incontinence or retention, sexual impotence and disorders of ejaculation.

The clinical evaluation of the PN in the dog is restricted by testing: the bulbocavernous reflex that allows only a subjective evaluation and the anal-sphincter electromyography that is an indirect, muscle mediated, evaluation of the PN function. Instead in human neuro-urology and neuro-gastroenterology, the PN function can be objectively tested electrophysiologically by measuring the PN terminal motor latency (PNTML)

The PNTML is a not invasive procedure recorded with a specific glove surface electrode (St. Mark's Pudendal Electrode, London, UK) connected to the electromyography system (Dantec-Medtronic, Skovlunde, Denmark). The stimulation was applied with a finger introduced within the rectum and recorded at the anal sphincter. The objective of this research were: a) to evaluate the technical feasibility of recording the PNTML in dogs, b) to obtain the normal reference range for PNMTL in the dog, c) evaluate the relation between PNTML and sex, body weight and age. The mean PNTML (mPNTML) for each patient was calculated as the average of the left PNTML and right PNTML. Patient sex and mPNTML were compared with the Student's t test. Associations between patient age and weight and mPNTML were evaluated with linear regression, with statistical significance set at 5%. Thirty-one male and 16 female dogs were studied. The mean age of the dogs was 5.5 +/− 3.1 years, and mean weight was 29.3 +/− 10.2 kg. Border collies, Boxers, GSDs and Labrador retrievers were the most commonly represented breeds. The mPNTML for the dogs studied was 1.23 +/− 0.26 msec. There were no significant differences in mPNTML recorded in male and female patients (p= 0.62), and no association with age (p=0.52) or weight (p=0.92) and mPNTML was found. Recording the PNTML was found technically feasible and normal value were established in dogs. This can be used in the diagnostic assessment of dogs with urinary and/or faecal incontinence and with cauda equina disorders.

ABSTRACT #20

REFERENCE VALUES OF THE MOTOR NERVE CONDUCTION VELOCITY IN CALVES. HC Schenk[1], J Rehage[2], A Tipold[1]. Department

of Small Animal Medicine and Surgery[1], Clinic for Cattle[2], University of Veterinary Medicine, Hannover, Germany.

In a big clinical survey of neurological diseases of cattle in a pool of patients (n= 3580) of the clinic for cattle peripheral nerve injuries were the most frequent neurological disorder (n= 25). The animals suffered either from a paresis of the N. ischiadicus/ peroneus or the N. tibialis. Injuries of the N. radialis were quite rare.

The diagnostic tool of choice for evaluating peripheral nerve injuries is electromyography and the measurement of the motor nerve conduction velocity (mNCV). Due to a lack of reference values for mNCV in bovines, this diagnostic tool can not be used properly in this species. To obtain these values we evaluated the mNCV of the N. radialis and the N. ischiadicus/ peroneus in 20 healthy calves with ages ranging from 16 to 85 days. The nerves of each side and limb were stimulated supramaximally on a distal and proximal location and the mNCV was calculated with regard to the distance between these two points and the body temperature. The N. radialis was stimulated distally at the distal third of the humerus between the M. brachialis and the lateral head of the M. triceps and proximally in the angle between the head of the humerus and the ventral part of the scapula. The N. ischiadicus/ peroneus were stimulated distally in the popliteal fossa and proximally in the trochanteric fossa between the trochanter major and the tuber ischiadicum. The mean mNCV of N. radialis was 48.3 m/s with a standard deviation of ± 10.6 m/s. For the mNCV of the N. ischiadicus/ fibularis the mean was 83.8 m/s with a standard deviation of ± 5.9 m/s. Six of these 20 calves were measured five times on different days to evaluate the influences of interindividual (stimulated nerve, side of stimulated nerve) and environmental effects. An analysis of variance of these effects showed no significant ($p = 0.05$) effect of the day of the measurement ($p = 0.2519$) or the side of the measured nerve ($p = 0.859$) on the mNCV. The mNCV shows a highly significant ($p < 0.0001$) difference between the N. radialis and the N. ischiadicus/ fibularis. The age of the examinated calves had no significant influence on the measured mNCV.

An analysis of the variance components for the resulting mNCV of each nerve showed that the influences of calf and measurement day were greater for N. radialis (calve: 20.1, day: 7.5) than for the N. ischiadicus/ fibularis (calve: 3.6, day: 0.3). This could possibly be explained by the error caused by the shorter distance between the two stimulation points when measuring the motor nerve conduction velocity of the N. radialis in calves.

ABSTRACT #21

CUTANEOUS SAPHENOUS NERVE GRAFT TO THE SCIATIC NERVE AND ELECTROPHYSIOLOGIC FOLLOW UP IN A DOG. N Granger[1], P Moissonnier[2], L Fanchon[3], K Gnirs[1], S Blot[1]. [1]Unité de Neurologie, Ecole Nationale Vétérinaire d'Alfort, France; [2]Unité de Chirurgie, Ecole Nationale Vétérinaire d'Alfort, France; [3]Unité de médecine de l'élevage et du sport, Ecole Nationale Vétérinaire d'Alfort, France.

Traumatic events (motor vehicle accidents, fractures, bites wounds...) can cause peripheral nerve injuries. Complete discontinuity of major peripheral nerve bundles necessarily causes severe functional disorders, both sensory and motor. Defect of nerve bundles requires nerve grafts to fuse proximal and distal stumps and therefore give a chance for functional recovery.

A two-year-old, entire female Griffon Vendéen was referred to the National Veterinary School of Alfort with a one month history of right pelvic limb lameness after a traumatic event with a wild boar. Neurologic examination revealed monoplegia and anesthesia of the right pelvic limb distal to the knee, except for the saphenous cutaneous area. Electromyographic (EMG) testing revealed major spontaneous electrical activity in muscles distal to the right stifle joint. Motor nerve conduction studies failed to show tibial and peroneal compound muscle action potentials (M wave) in the tibial and interosseous muscles. Muscle contraction of the semitendinosus and semimembranosus muscles was seen with proximal sciatic nerve stimulation. Those EMG abnormalities were consistent with a severe lesion of the tibial and peroneal nerves, such as a neurotmesis, localised at the level of the stifle joint. Exploratory surgery revealed the presence of a 80 mm gap along the course of both right sciatic nerve branches thus confirming the EMG conclusions. The left cutaneous saphenous nerve (CSN) was dissected along the medial femoral area and interposed to graft the right sciatic nerve branches. A standardized protocol of joints mechanotherapy and electrophysiologic therapy was realised during the reinnervation process. Ten months after the surgery, the dog had an almost complete recovery. Mild intermittent lameness was observed at a gentle trot and neurologic examination revealed diminished flexion of the hock and digits. Control EMG testing showed no abnormal spontaneous electrical activity in the right pelvic limb muscles and small M waves were recorded in the right interosseous and tibial cranial muscles.

Without surgical treatment, neurotmesis injury results in poor return to motor and sensory functions of the affected limb, leading to amputation. If nerve defect exists, nerve grafting is the unique therapeutic option, even if surgery is largely delayed after the onset of the injury. The sensory CSN is a potential source of autologous nonvascularized peripheral nerve graft in dogs. Despite the progress of microsurgical techniques, the prognosis remains guarded but can be precised with electrophysiologic testing. Reinnervation is a long course process which needs physiologic support and full comprehension and involvement of the owner, but almost complete functional recovery is possible.

ABSTRACT #22

THIAMINE, PYRIDOXINE AND CYANOCOBALAMINE COMBINATION ON NERVE REGENERATION IN RATS WITH EXPERIMENTALLY INDUCED CRUSH INJURY. O. Besalti[1], Z Pekcan[1], I Ergin[1], E Unlu[2]. [1]Ankara Unıversıty, Faculty of Veterinary Medicine, Department of Surgery, Ankara, Turkey; [2]Ministry of Health Ankara Diskapi Education Hospital, Department of Physical Therapy and Rehabilitation Ankara, Turkey.

The goal of the study was to investigate the role of thiamine, pyridoxine and cyanocobalamine combination on nerve regeneration after experimentally induced crush injury in rats. Thirty adult, male Wistar rats weighing 220 – 270 g were allocated into two equal groups, and sciatic function index and toe spreading reflex were analyzed. All rats were anaesthetized, their sciatic nerves were exposed and standard nerve conduction study including distal motor nerve latency (DL), distal compound muscle action potentials (DCMAP), proximal compound muscle action potential (PCMAP) and nerve conduction velocity (NCV) were examined. Sciatic crush injury was induced with a smooth bulldog clamp for three minutes and the clamped area was marked with propylene suturing material in the adjacent muscle. The distal and proximal stimulation points were 1 cm above and 1cm below the injured area. The experimental group received thiamine (33 mg/kg), pyridoxine (33 mg/kg) and cyanocobalamine (0.5 mg/kg) injections intramuscularly for 3 weeks and the control group received equal volume saline injections. At the third week, the rats were evaluated as before and were euthanized. Aafter 3 weeks of treatment toe spreading reflexes were better in the experimental group than the controls ($P < 0.05$). Mean sciatic function index changed for the better with time significantly in both groups but there were no differences between the experiment and control groups after the crush injury. In the control group, normative electrophysiological values were 2.06±0.25 ms, 10.38±2.95 mv, 9.57±3.04 mv and 50.08±5.91 m/s for DL, DCMAP, PCMAP and NCV respectively in the 30 rats. At the end of the experiment in the experiment group the same values were 2.18±0.22 ms, 10.14±2.70 mv, 9.11±2.52mv and 51.47±6.78m/s. However in the control group they were 1.93±0.21ms, 10.62±3.27 mv, 10.02±3.50 mv and 48.69±4.72 m/s. respectively. There was no significant difference in regard to DL, DCMAP, PCMAP and NCV between both groups ($P < 0.05$). When the normative and post treatment values were compared, the differences were found significant ($p < 0.05$). In conclusion the beneficial effect of thiamine, pyridoxine and cyanocobalamine combination was not found in regard to the electrophysiological parameters measured. However clinical improvement was superior in the experiment group than the control group.

Abstract #23

RETURN OF SENSATION AFTER SECTION AND REPAIR OF THE RAT SCIATIC NERVE. Rupp A[1], Fichter A[2], Dornseifer U[3], Papadopulos N[2], Matiasek K[1]. [1]Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, LMU Munich, Germany, [2]Clinic for Plastic Surgery, Klinikum Rechts-der-Isar, Munich, Germany, [3]Department for Plastic & Reconstructive Surgery, Klinikum Bogenhausen, Munich, Germany.

Most methods used for evaluating peripheral nerve regeneration in living animals focus on motor function, whereas sensation - especially in the noci-/exteroceptive form - is important for the avoidance of harm.

In the following study 20 male Lewis rats were submitted to sectioning and repair of the sciatic nerve. All lesions had a length of 12 mm, with the distal end located 4 mm proximal to the submersion of the tibial nerve into the gastrocnemic muscle. The rats were accustomed to being handled twice daily for 5 days before the operation, and reference values were taken two days pre op. The first evaluation post op was on day (D) 5, further examinations taking place twice a week until D56. For nociceptive testing the animals were pinched with atraumatic forceps at defined points on all four sides of both feet.

Generally, hypersensitivity was noted when the foot on the operated side was pinched in reinnervated areas or those innervated by the saphenous nerve. On D5 no sensory innervation was detectable on the lateral and dorsal sides of toes 4 and 5, the whole lateral aspect of the foot, and a wedge-shaped area on the dorsal aspect of the foot extending from toes 4 and 5 towards the ankle. On the plantar aspect everything apart from the 2 medial pads and toes 1 and 2 appeared to be denervated. Some rats reacted to being pinched in the area around the heel and the plantar aspect of toe 3. The medial side of the foot and toes 1 to 3 appeared to be fully innervated throughout testing from D5 to D56 as the saphenous nerve is responsible for this area.

In the following eight weeks sensation returned at different rates for individual rats but on a set pattern. On the lateral and dorsal aspect of the foot, innervation reappeared very slowly from proximal to distal, in most animals reaching the tip of toe 4 but only the base of toe 5 by D56. The sole, however, seemed to be innervated from medial. Half the animals observed reacted to being pinched on the medial half of the sole including the heel and midline within two weeks. The lateral half as well as the base of toe 4 then followed in the same number of rats within the next three weeks. Toes 4 and 5, with their corresponding pads each being reinnervated a week later, were the last plantar areas where sensation was regained. By D56 most animals had achieved complete sensory reinnervation on the soles of their feet and toes 1 to 4.

With the proximal stump approximately 95 mm from the tip of the third toe and with a rate of regeneration of about 1.4 to 3 mm per day or even faster (since sensory nerves are involved) it is not easy to determine whether regrowth of the tibial nerve or extensive sprouting of the saphenous nerve instigates sensory reinnervation. Retrograde tracing might provide an answer to this puzzle.

ABSTRACT #24

MAGNETIC FIELD GEOMETRY DETERMINES EFFICACY OF TRANSCUTANEOUS NERVE STIMULATION. T Weyh[1], K Wendicke[1], A Rupp[2], H Zantow[1], A Fischer[3], K Matiasek[2]. [1]Heinz Nixdorf Chair for Medical Electronics, Technical University of Munich, Germany; [2]Chair of General Pathology & Neuropathology, and [3]Section of Neurology, Clinic of Small Animal Medicine, Ludwig-Maximilians-University, Munich, Germany.

The electric field induced by pulsed transcutaneous magnetic stimulation has been proven to enhance peripheral nerve regeneration after both experimental and spontaneous trauma. Beyond promotion of axonal outgrowth by so far unknown mechanisms, induced electric currents trigger muscle responses and contribute to synaptic remodelling and plasticity of central motor systems. To date, variations of nerve-muscle responses have been attributed to intrinsic factors such as heterogeneity of epineurial layers and tissue specific thresholds. On the other hand, reactions can be modulated by changes in magnetic field strength, pulse duration, field distribution and frequency. Although experimental data is missing the paradigm that the electric field gradient is the most important influence on nerve excitation has never been questioned. Studies on three-dimensional expansion of the electric fields induced by magnetic stimulation coils are sparse. Thus, spatial resolution remains a matter of controversy.

In order to investigate the spatial coupling of coil parameters and peripheral nerve fibres newly designed coils were tested on 15 laboratory rats.

In a first setting, coils with different electric field strengths but constant field gradient were tested in anaesthetized rats after surgical exploration of the sciatic nerve. In contrast to the common hypothesis peripheral nerve response recorded from interosseus muscle varied proportional to the field strength. On base of these results, another coil was designed to establish a spatial separation of maximal induced electric field and the field gradient. Now, peripheral nerve stimulation proved most effective between these two landmarks corresponding to the area of the maximal product of field strength and gradient.

Our results prove that the gradient of induced electric fields does not colocalize with the point of most effective peripheral nerve stimulation. Moreover, coils with low-level electric field strength remain insufficient to induce electric currents in peripheral nerves even if the gradient equals that of effective coils with appropriate field strength. Therefore, the field gradient is not the main influence on magnetic induced nerve excitation. The stimulation peaks adjacent to the area where the product of field strength and gradient can be calculated which, ultimately, should lead to incorporation of this parameter into advanced coil design. For more precise monitoring of spatial resolution and an assessment of optimal pulse shapes and repetition rates, magnetic stimulation of neuronal networks on cell chips provides a suitable test environment.

ABSTRACT #25

PERIPHERAL NEUROPATHIES ASSOCIATED WITH CANINE HEMANGIOSARCOMA. S Kuhne-Velte, W Schmahl, K Matiasek. Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, LMU Munich, Germany.

Even though it is a well known fact that numerous neoplasms can cause paraneoplastic syndromes, only one single case about a hemangiosarcoma being the possible cause of a peripheral neuropathy has been reported in one dog with hypothyroidism and accessory pulmonary carcinoma. This tumour type occurs quite frequently in dogs but is rarely seen in men which might be one reason for the lack of reports about its role in paraneoplastic neuropathies.

Within the scope of a large study on neoplasia-associated neuropathies in domestic animals samples of peroneal nerves from 10 dogs with hemangiosarcoma could be examined histologically and after nerve fibre teasing.

Six of ten dogs (60%) revealed a mild (1/6), moderate (4/6) or severe (1/6) chronic peripheral neuropathy. In these animals comorbidities capable of causing pathologic changes in peripheral nerves were ruled out through clinical examination, laboratory testing or necropsy. Amongst affected nerves three individuals presented an axonal neuropathy, one a demyelinating disease and two exhibited mixed neuropathies with predominance of myelin sheath pathology. Two axonal phenotypes were of atrophic, one of WD-type. Nerves with demyelinating neuropathies were characterized by myelin thickening, and, in one case, intense ballooning of the myelin sheath.

Several mechanisms have been described to cause peripheral nerve lesions in tumour patients. Apart from tumour cell infiltration, nerves can suffer from immunologic events or metabolic disturbances. Most paraneoplastic phenomenons have been traced back to autoimmune responses as a consequence to antigen shedding and molecular mimicry. In turn, autoantibodies frequently are detected in serum of human patients reacting in PNS and muscles with nuclear proteins, myelin proteins, presynaptic proteins, glycolipids, voltage-gated ion channels, receptors and muscle filaments; thus, giving rise to various axonal and demyelinating phenotypes. Whether or not histomorphological alterations found in our specimens result from similar immunological mechanisms has to be clarified by further investigations. Peripheral nerve histology did not reveal any nerve fibre selectivity and can, therefore, not predict the quality of possible deficits. This might be explained by the compensatory capability of the neuromuscular system or the poor general health condition in advanced stages of malignancy. Regardless whether clinical signs became apparent, our morphological studies prove that hemangiosarcomas in dogs are frequently associated with a peripheral neuropathy.

ABSTRACT #26

ELECTROPHYSIOLOGIC AND HISTOPATHOLOGIC STUDY OF DIABETIC NEUROPATHY IN MICE. S Añor, J Montane, A Serafin, J Martorell, F Bosch, M Pumarola. Department of Animal Medicine and Surgery, Veterinary School, Autonomous University of Barcelona. 08193 Barcelona, Spain.

Peripheral neuropathy is one of the most common complications of Diabetes Mellitus in human beings. In domestic animals, cats seem to be specially predisposed to the development of this complication, but some uncontrolled diabetic dogs may also develop clinical signs of peripheral nervous system dysfunction. In humans, the distal peripheral nervous system seems to be affected first, followed by involvement of the peripheral motor and autonomic nervous systems. Electrophysiologic studies in diabetic human beings with peripheral neuropathy show decreased motor and sensory peripheral nerve conduction velocities, as well as abnormal peripheral motor and sensory evoked potentials, consistent with a mixed, predominantly distal polyneuropathy. Histopathologically, loss of peripheral myelinated nerve fibers is the predominant morphological feature in human diabetic nerves.

The aim of the present study was to assess the function and morphology of the peripheral and central nervous system in diabetic mice, in order to determine the potential usefulness of this mice model for the sudy of diabetic neuropathy. Diabetes was induced by multiple streptotozin injections (45 mg/kg IP every day, for 5 days) in 2–3 months-old mice. One month later, diabetic animals demonstrated characteristic hyperglycemic features, such as polydypsia-polyuria, polyphagia, weakness and distended abdomen. Blood glucose measurements were performed at the beginning of the study (4 weeks after streptotozin induction), one and a half months later, and just before euthanasia 3 months later. Electrophysiological studies were performed at 16, 21, and 26 weeks after induction, under intraperitoneal ketamine-medetomidine anesthesia. Electrophysiological studies included measurement of peripheral motor and sensory nerve conduction velocities of the peroneal and tibial nerves, as well as performance of F-wave studies, measurement of cord dorsum potentials, evaluation of spinal sensory evoked potentials after stimulation of the tibial and peroneal nerves, and evaluation of spinal motor evoked potentials in the pelvic limb dorsal and plantar

interosseous muscles. Peripheral sensory and motor nerve conduction velocities showed a gradual decrease with time in diabetic animals, while showing an increase consistent with growth in control animals. No significant changes were observed in spinal motor evoked potentials during the study, and a decrease in the latency of spinal cord sensory evoked potentials was observed in control animals.

Just before euthanasia, 26 weeks after streptotozin induction, all animals were perfused with formalin. After euthanasia, samples of all body tissues and organs, including central and peripheral nervous system, were taken and processed for histological, biochemical and immunohistological studies. Histological examination of foodpads revealed significantly reduced numbers of epidermal fibers, whereas no differences were observed in sweat gland-associated autonomic axons. Myelinated fiber density and number in tibial (predominantly motor axons) and peroneal (sensory and some autonomic axons) fascicles did not seem affected by the diabetic state.

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

ABSTRACT #27

HEREDITARY MOTOR AND SENSORY NEUROPATHY TYPE II IN GREYHOUND SHOW DOGS. K Matiasek[1], A Flagstad[2], C Hahn[3], K Hultin-Jaederlund[4], K Jurina[5], D Hanzlicek[6], B Kessler[7]. [1]Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, Ludwig-Maximilians-University, Munich, Germany; [2]Department of Small Animal Clinical Sciences, The Royal Veterinary and Agricultural University, Copenhagen, Denmark; [3]Neuromuscular Disease Laboratory, Royal (Dick) School of Veterinary Studies, The University of Edinburgh, Midlothian, United Kingdom; [4]Institute of Surgery & Medicine, Swedish University of Agricultural Sciences, Uppsala, Sweden; [5]Small Animal Referral Clinic, Haar, Germany; [6]Klinika Jaggy, Brno, Czech Republic; [7]Institute of Molecular Animal Breeding & Biotechnology, Ludwig-Maximilians-University, Oberschleissheim, Germany.

Inherited neuropathies are incidentally observed in domestic animals whereas in human medicine at least 1 person out of 2500 is affected by a hereditary neuropathy with both motor and sensory deficits (HMSN). Canine HMSNs gain further attention only if they are breed-associated and appear in certain pedigrees. This study focuses the biopsy findings of a hitherto unreported polyneuropathy in greyhound show dogs that goes along with gait abnormalities, muscle wasting and respiratory distress with an onset at 4 months of age.

Peripheral nerve biopsies from 4 unrelated juvenile greyhound show dogs were taken after clinical diagnosis of a polyneuropathy characterized by progressive ataxia and tetraparesis, delayed proprioceptive placing, hyporeflexia, distal limb muscle atrophy and laryngeal involvement. Nerve samples underwent embedding in epoxy resin, and nerve fibre teasing. Thereafter, each fascicle was examined light and electron microscopically according to standardized protocols.

All dogs presented with a severe chronic axonal polyneuropathy with multifocal axonal swellings containing irregular neurofilaments and axoplasmatic organelles. Protrusion of axonal contents between paranodal lamellae resulted in enlargement of uncompacted myelin. Other myelin sheath abnormalities comprised reactive lesions with proliferation of rough endoplasmatic reticulum, multifocal hyperplasia of axon-Schwann cell networks, and Wallerian-like degeneration.

The present case series in greyhound show dogs features a chronic progressive axonal polyneuropathy characterized by both motor and sensory deficits. Peripheral nerve inspection did not only uncover an axonal phenotype. On electron microscopical level a perturbation of the fast anterograde axonal transport system could be assessed. Since mode of pathology, age of onset, exclusion of exogenous causes and intense pedigree analyses suggest a monogenetic neuropathy with an autosomal-recessive trait certain DNA regions encoding for motor proteins such as KIF have to be considered as candidate genes. Albeit axonal HMSN (type II) represent the most common clinical phenotype of inherited neuropathies in dogs, genomic background of those diseases still remains enigmatic. Like in HMSN II affected greyhounds ultrastructural investigations can help to focus further investigations as long as genetic screening tests are not available.

ABSTRACT #28

FEMORAL MONONEUROPATHY CAUSED BY A MALIGNANT SARCOMA: 2 CASES. P Montoliu, S Añor. Department of Animal Medicine and Surgery, Veterinary School, Autonomous University of Barcelona. Barcelona, Spain.

The objective of this report is to describe the clinical features, diagnostic imaging abnormalities, underlying disease and treatment of two dogs presenting for pelvic limb monoparesis caused by a femoral mononeuropathy.

Case 1 was a nine year old male poodle that was presented for a 6 month history of left pelvic limb (LPL) lameness. Physical and neurological examinations revealed muscle atrophy in the LPL, more severe in the quadriceps muscle, LPL monoparesis, absent patellar reflex and decreased hip flexion of the same limb. A lesion affecting the left femoral nerve was suspected. Abdominal ultrasound revealed a retroperitoneal mass on the left side of the abdominal cavity. On exploratory laparotomy, a 7×3 cm mass was visualised in the retroperitoneal space. The mass was attached to the left psoas muscle and was infiltrating the left lumbosacral plexus. Partial debulking of the mass was performed and histopathologic examination findings were consistent with an extra-skeletal ossifying osteosarcoma.

Case 2 was a six year old female American Staffordshire Terrier that was referred for a two year history of LPL lameness. On physical examination, severe atrophy of the left quadriceps muscle was detected. Neurologic examination revealed LPL monoparesis. The patellar reflex was absent in the LPL and decreased hip flexion was evident when assessing the withdrawal reflex in the same limb. The withdrawal reflex was mildly decreased in the RPL. A lesion involving the L4–L7 spinal cord segments, with more severe involvement of the left L4–L6 spinal cord segments, nerve roots or the left femoral nerve was suspected. Magnetic resonance imaging of the lumbar spine identified a 4×2×2.5 cm mass within the left psoas muscle, which extended dorsally and entered the spinal canal following the course of the fifth lumbar nerve root and was displacing the cauda equina to the right. A L5–L6 left hemilaminectomy was performed. The left L5 nerve root was thickened and infiltrated by a firm, whitish mass. Dissection following the left L5 spinal nerve ventrally led to the mass located in the psoas muscle, which was carefully dissected and excised. Histopathologic examination of the excised mass confirmed a diagnosis of undifferentiated sarcoma affecting the left psoas muscle and infiltrating the fifth spinal nerve and root. Pelvic limb monoparesis caused by femoral nerve neuropathy is a rare clinical entity in dogs.

To the authors knowledge, femoral nerve injuries have been reported secondary to extreme extension of the hip and to PNST infiltrating the lumbosacral plexus in dogs, but no reports have been found describing infiltrations of this nerve by surrounding malignant neoplasms. In our opinion, sarcomas and other malignant tumours growing adjacent to the femoral nerve or root should be included in the differential diagnoses of dogs with femoral nerve mononeuropathies, as they can compress or infiltrate the nerve.

ABSTRACT #29

MAGNETIC RESONANCE IMAGING AND ULTRASOUND-GUIDED FINE-NEEDLE ASPIRATE IN THE DIAGNOSIS OF BRACHIAL PLEXUS LYMPHOMA IN A CAT. A de Stefani[1], SR Platt[1], JF McConnell[1], FJ Llabres[2], LA Wieczorek[1], KC Smith[3]. [1]Animal Health Trust, Centre for Small Animal Studies, Lanwades Park, Newmarket, UK. [2]Davies Veterinary Specialists, Manor Farm Business Park, Higham Gobion, Herts. [3]Animal Health Trust, Centre for Preventive Medicine, Lanwades Park, Newmarket, UK.

A seven and a half year old male neutered domestic shorthaired cat was presented with a four-week history of left thoracic limb lameness, which had progressed to monoparesis. Based upon the neurological examination the responsible lesion was suspected to be affecting the C6 to T2 nerve roots or brachial plexus. Magnetic resonance imaging (MRI) of the left brachial plexus was perform using a 1.5 T superconducting magnet (General Electric). T2 weighted and (short tau inversion recovery) STIR sequences were obtained in dorsal and transverse planes; transverse T1 weighted images were obtained before injection of paramagnetic contrast media (MultiHance® Gadobenate dimeglumine [0.1 ml/kg]). Post contrast T1-weighted sequences were acquired in sagittal and transverse planes and repeated applying fat suppression. An s-shaped, tubular, high signal parenchymal lesion extending away from the midline and toward the left thoracic limb was detected on the dorsal STIR images. The transverse T1-weighted images post contrast demonstrated a heterogeneous pattern of enhancement. The lesion appeared to track to the vertebral canal at the level of C7 and extended to the T1 vertebral level. Ultrasound of the left axilla was performed after the MRI using a 10 MHz probe. Multiple ultrasound-guided fine needle aspirates (FNAs) of this axillary mass were performed. FNA smears were stained with H&E and found to be consistent with well-differentiated lymphoma. The cat was started on COP (Cyclophosphamide, Vincristine, Prednisolone) chemotherapy protocol. Twelve months after diagnosis the cat is still on the COP chemotherapy protocol (week 50) and is

not showing any side effects of the chemotherapy. Shortly after the start of the chemotherapy protocol the cat showed mild improvement of the neurological signs (weight bearing monoparesis) and overall he has not shown any signs of progression of the disease.

Primary neoplasms of the peripheral nerves are rarely recognized in the domestic species and appear to be of clinical relevance only in the dog. Scattered reports of peripheral nerve tumours in cats are present in the literature. Lymphoma seems to be the most commonly diagnosed tumour in these reports. Ultrasound guided FNAs are considered a sensitive and safe diagnostic aid therefore ultrasound-guided FNAs of brachial plexus masses is highly recommended before planning any invasive surgery. This is particularly advisable in cats where lymphoma, a highly chemotherapy sensitive neoplasm, is a likely diagnosis.

ABSTRACT #30

MAGNETIC RESONANCE IMAGING OF THE INTRATEMPORAL FACIAL NERVE IN IDIOPATHIC FACIAL PARALYSIS IN THE DOG. V Lorenzo,[1] A Muñoz,[1,2] ASP Varejão,[3] [1]Resonancia Magnética Veterinaria, Madrid, Spain. [2]Neuroradiología, Hospital 12 de Octubre, Madrid, Spain. [3]Department of Veterinary Sciences, CETAV – University of Trás-os-Montes e Alto Douro, Vila Real, Portugal.

The most common cause of peripheral facial nerve paralysis in dogs, in the absence of otitis media, is thought to be idiopathic. Gadolinium-enhanced MRI (Gd-MRI) has been used for at least 15 years to study peripheral facial weakness in humans, with a wide variety of pathology, including Bells Palsy, the clinical equivalent of idiopathic facial nerve paralysis in dogs, in which Gd-MRI may demonstrate abnormal enhancement of the intratemporal facial nerve. The aim of this study is to outline the role of the Gd-MRI in dogs with idiopathic facial nerve paralysis, with regard to pattern of Gd-MRI enhancement, and to search for prognostic information.

Five dogs with unilateral peripheral facial nerve paralysis, followed between 2004 and 2005, were included in this study. All dogs underwent a complete physical and neurological examination as well as diagnostic tests to screen for potential underlying causes of facial nerve paralysis, including routine haematology, serum biochemistry, thyroid screening and cerebrospinal fluid analysis. No significant abnormalities were found except for the facial paralysis. MRI imaging was performed using a 0.5-T system (Gyroscan T5-II, Philips, Eindhoven, The Netherlands). A quadrature head coil was employed in all examinations. MRI images were obtained on transverse, dorsal and sagittal planes using the following sequences and parameters: SE T1 (TE 14–16, TR 500), SE T2 (TE 100, TR 4000), and T1-weighted postcontrast examination immediately after IV injection of gadopentetate dimeglumine (0.2 mmol/kg). In addition, T1 STIR images were obtained in all dogs. The sequences were performed with a slice thickness of 2–3 mm with no intersection gap. The time interval between the onset of the clinical signs and the MRI examination and the progress of the disease were noted for each dog. The following four segments of the facial nerve were analysed: internal auditory canal (IAC), labyrinthine segment/geniculate ganglion (Lab/GG), tympanic segment (Tymp), and mastoid segment (Mast).

Contrast enhancement in at least one segment of the facial nerve was found in three dogs (60%): in 1 dog in all segments and in 2 dogs in one segment. In the 3 dogs with enhancement, 1 recovered completely in 8 weeks and 2 have not recovered completely. Dogs without enhancement (40%) recovered completely in an average time of 4 weeks.

In conclusion, Gd-MRI was useful in the study of idiopathic facial nerve paralysis. We were able to rule out other diseases and to detect nerve enhancement. Dogs with enhancement had a poorer prognosis, with longer course (1 case) or permanent deficits (2 cases), on the contrary dogs without enhancement had a clinical remission, and in a shorter time (2 cases). Although the number of cases of this study is low, results are similar to what is found in humans and seem to indicate that Gd-MRI may also have a prognostic value in dogs with idiopathic facial nerve paralysis. It has been stated that the increased capillary permeability to gadolinium is likely due to breakdown of the blood – peripheral nerve barrier, most probably as the result of Wallerian degeneration.

ABSTRACT #31

INTRACRANIAL EPIDERMOID CYST IN A DOBERMAN PINSCHER. T Steinberg[1], A Brühschwein[2], K Matiasek[3], A Fischer[1]. [1]Section of Neurology, Clinic of Small Animal Medicine, [2]Diagnostic Imaging Center, Clinic of Small Animal Surgery & [3]Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, Ludwig-Maximilians-University, Munich, Germany.

A 4 year-old male intact Doberman Pinscher was referred to the Small Animal Teaching Hospital of Munich with a 3-month history of slowly progressive behavioural changes, intermittent ataxia and falling to the right side. On presentation, the dog was excited and aggressive. He presented with a severe ataxia with tight circling and head tilt to the right side. Cranial nerve examination showed positional horizontal nystagmus with the fast phase to the left. There were mild hopping deficits on the right limbs. Based upon both behavioural changes and abnormal postural reactions the lesion was assessed as a right-sided central vestibular syndrome. Haematology, blood chemistry, liver enzymes, liver function test and urine analysis were within normal limits.

The dog underwent magnetic resonance imaging (MRI) with a Siemens Magnetom Open, 0.2 Tesla. T1- and T2-weighted sequences in dorsal, transverse and sagittal orientations were performed. Additional T1 post-contrast studies were obtained after intravenous injection of 0.1 mmol/kg Gadolinum-DTPA. The MRI demonstrated a normal parenchyma of the cerebrum with normal lateral ventricles and third ventricle. Adjacent to the cerebellum was a round cyst-like lesion located in the right cerebellopontine angle with severe displacement of the cerebellum. Results of cerebrospinal fluid examination were within normal limits.

Based on the MRI features, differentials included epidermoid or dermoid cysts, brain abscess, neoplasia, and parasitic cyst. The dog was euthanised due to progressive and severe behavioural changes and neurological impairment. On gross necropsy of the brain there was a spheroidal encapsulated mass located paramedian within the fourth ventricle severely compressing the adjacent brain stem, cerebellar roof and ventral aspects of the vermis. Results of the histological examination were consistent with an epidermoid cyst.

Epidermoid cysts present an extremely rare developmental disorder in both man and dog. The case described above demonstrates that MRI is an excellent imaging modality to diagnose this type of intracranial lesion. Epidermoid cysts should be considered as a differential for a focal compressive mass, especially in the cerebellopontine angle in dogs.

ABSTRACT #32

CEREBELLAR INFARCT IN A CAT: MRI & HISTOPATHOLOGICAL FINDINGS. GB Cherubini[1], R Cappello[1], M Peris[2], P Mahoney[1], S Schoeniger[1], B Singh[1]. [1]The Royal Veterinary College, London, UK.[2] Barton Lodge Veterinary Centre , Hemel Hampstead, UK.

A 10 years old female neutered domestic short hair cat was referred with a hyper-acute onset of neurological signs consistent with ''cranial cerebellar syndrome''.

The MRI study done approximately 6 hours after the onset of the clinical signs showed a single, focal cerebellar lesion that was hyperintense on T2, T2 flair and T2*, isointense on T1, with no uptake of contrast. There was sharp demarcation between the lesion and adjacent normal cerebellum, and there was no apparent mass effect. On the transverse images, the lesion was centred on the midline, radiating to the dorsal surface of the cerebellum. The extension of the lesion involving grey and white matter, the rostral part of the cerebellar hemisphere and vermis can be justified by an ischemic/embolic lesion of the rostral cerebellar arteries arising from the caudal cerebral arteries. Due to the short post-infarction time before the MRI study, vasogenic edema and secondary mass effect were not visible on T1 post Gad., T2 and T2*. However early lesion, consistent with cytotoxic edema, was visible on both T2 and T2* and no ''fogging'' effect was detected.

A diagnostic work-up to rule out possible causative etiological disorder (CSF analysis, CBC, biochemistry, electrolytes, T4, basal Cortisol, PT-APTT, ECG, non-invasive arterial blood pressure, chest radiographs and abdominal ultrasound) was unremarkable.

No macroscopic lesions were visible on gross examination. On histopathological examination, the cerebellar vermis (gray and white matter) contained a focal extensive, well-demarcated area of pale staining and disruption of the parenchyma (acute infarct). Within the infarcted area, gray and white matter had presence of multiple confluent clear spaces, fragmentation of neuropil and presence of swollen astrocytes. Less disrupted areas of white matter contained distended myelin sheets with eosinophilic debris, rare axonal spheroids and swollen oligodentrocytes. Rare gemistocytes and macrophages and very few neutrophils were observed. Vessels were lined by swollen endothelial cells and contained numerous intraluminal monocytes. The absence of macroscopic cerebellar lesions together with observed microscopic findings are consistent with an acute cerebellar infarct of approximately 8h duration. The left cranial lung lobe contained a mass, which was histologically identified as pulmonary adenocarcinoma with angioinvasion.

MRI findings of histopathological confirmed cerebellar infarct have not yet been reported in cats; thus cerebellar vascular accident should be considered in the differential diagnosis in cats with hyperacute onset of cerebellar signs.

ABSTRACT #33

IMAGING FINDINGS IN 12 CATS WITH MENINGO-ENCEPHALITIS. A Negrin, CR Lamb, GB Cherubini, R Cappello. Department of Veterinary Clinical Sciences, The Royal Veterinary College, University of London, London, UK.

Pathogenesis and CSF characteristics in cats with intracranial inflammatory conditions have been largely described, while MRI and CT findings of inflammatory diseases have not been reported in detail. From the medical record, 12 cats (11 cats had MRI and 1 cat CT) with inflammatory CSF, positive CSF antibody titres or post mortem examination compatible with a CNS intracranial inflammatory diseases were retrospectively evaluated. To facilitate unbiased interpretation, MRI images of cats with inflammatory CNS diseases were mixed with 24 cats that had MRI of the brain and non-inflammatory CFS, as control group.

Six cats were diagnosed with Feline Infectious Peritonitis (FIP), 2 with toxoplasmosis, 1 with bacterial meningitis extending from an inner ear infection, 1 with neutrophilic meningoencephalitis, 1 with lymphocytic meningoencephalitis, and 1 with histiocytic encephalitis.

The MRI was performed in 5 cats with FIP. T1-weighted images with contrast revealed multiple lesions with the main lesion being a diffuse uptake in the ependymal layer of the ventricular system in 2 cats, multifocal uptake in the brain and meningeal involvement in 2 other cats. Only in 1 case there was a multifocal hyperintensity in T2-weighted images. In one cat the MRI did not reveal brain lesions; however, in this case there was a frontal sinus inflammation. In the cases with ependyma uptake there was generalized ventricular system dilation. In one cat with FIP a CT study revealed multifocal contrast uptake. The MRI investigation in the 2 cats with toxoplasmosis revealed: multifocal hypointense lesions in T1-weigthed images with contrast uptake that were hyperintense lesions in T2-weighted images. In 1 case there was also a meningeal contrast uptake.

The MRI in the cat with bacterial meningitis showed contrast uptake in T1-weighted images and mild hyperintensity in T2-weighted images. The lesion was in the pons with focal meningeal involvement. The MRI in the cats with neutrophilic meningitis and lymphocytic meningitis revealed multifocal contrast uptake and multifocal hyperintensity in T2-weighted images. The lesions were distributed in the pons and meninges. The MRI in the cat with histiocytic encephalitis was unremarkable. MRI detected intracranial lesions in 8/11 (73%) cats. The MRI appearances of feline CNS inflammatory diseases were more frequently characterised by isointense signal in T1-weighted and hyperintense in T2-weighted. Contrast enhancement was present in all 8 cases. The MRI findings of FIP involve meninges and ependyma associated with hydrocephalus. No typical and specific distribution of lesions was present in cats with toxoplasmosis, which appeared multifocal. The MRI findings in all cases were consistent with the histological lesions.

ABSTRACT #34

MAGNETIC RESONANCE IMAGING OF DEMYELINATION IN CANINE DISTEMPER. A Bathen-Nöthen[1], VM Stein[1], A Gerdwilker[1], C Puff[2], W Baumgärtner[2], A Tipold[1]. [1]Department of Small Animal Medicine and Surgery, [2]Institute of Pathology, University of Veterinary Medicine Hannover, Foundation, Germany.

Demyelination is the prominent histopathological hallmark in the acute stage of canine distemper virus (CDV) infection. In this stage of the disease there are no manifestations of inflammation, since perivascular infiltrations usually are lacking.

Magnetic resonance imaging (MRI) has become an important diagnostic tool in human medicine to determine myelination and demyelination of the brain, for example in multiple sclerosis (MS). Changes within the myelin sheath lead to aberrant T1- and T2- relaxation times. In T1- weighted images, demyelinating lesions appear hypointense, whereas in T2-weighted images they are hyperintense relative to grey matter.

The purpose of our study was to evaluate whether demyelinating lesions due to CDV infection could be visualized by use of MRI (Magnetom Impact Plus, 1.0 Tesla, Siemens, Erlangen, Germany). A standard brain protocol was used including T1 (TR/TE, 330/12) - and T2 (3458/96) -weighted images in sagittal, transverse and dorsal planes. In one dog fluid attenuated inversion recovery (FLAIR) images were obtained.

Four dogs with clinically suspected CDV infection were subjected to MRI and euthanized on request of the owners for histopathological diagnosis.

MRI revealed hyperintensity in areas of the lobus temporalis in all dogs in T2- weighted images. Furthermore increased signal intensities were observed periventricularly in the thalamus (n = 1), hypothalamus (n = 1), lobus parietalis (n = 1), and lobus frontalis (n = 1) in T2. These lesions appeared hypointense or isointense in T1. Lesions visible in FLAIR were similar to that in T2.

In two dogs there was a loss of contrast of white and gray matter in the arbor vitae of the cerebellum. Contrast medium (Gd-DTPA) was given in three dogs, and contrast enhancement was observed in one. Histopathological examination revealed signs of myelin swelling and demyelination with gemistocytes and gitter cells in the brain of all dogs, especially in the classical predilection sites in the cerebellum.

MRI-findings correlated well with the results of histopathological examination. MRI seems to be a valuable tool for the visualization of in vivo myelination defects in dogs. Specific MRI techniques such as magnetization transfer and diffusion tensor imaging could offer further advantages in establishing the diagnosis of dysmyelination, enabling the veterinarian to distinguish more precisely between demyelination, vasogenic edema, and/or inflammation.

ABSTRACT #35

MRI OF THE HIPPOCAMPUS IN CANINE EPILEPSY. CS White, A Wessmann, H Volk, GB Cherubini, R Cappello, KE Chandler. The Queen Mother Hospital, the Royal Veterinary College, North Mymms, United Kingdom.

Temporal lobe epilepsy (TLE) is the most common form of epilepsy in adult humans, is frequently associated with abnormalities on magnetic resonance imaging (MRI) and a typical pathological change known as hippocampal sclerosis (HS) (Mathern et al, 1996, Epilepsy Research 26 151–161). In this study, we asked if there was evidence of subtle hippocampus abnormalities visible on MRI in dogs with recurrent seizures, similar to MRI findings in the human TLE.

MRI scans were assessed subjectively in 44 dogs with recurrent seizures and 11 controls (dogs with peripheral vestibular signs). Symmetry in size, shape and signal intensity were assessed blindly by three neurologists on T1 and T2 weighted scans and T1 plus gadolinium. The cross-sectional area of the left and right hippocampi were measured from T1- weighted scans, by tracing the scans by hand onto paper. An imaging program (www.scion-corp.com) was then used to calculate the areas and the ratio was calculated for each patient.

No significant difference in hippocampal ratios was found between dogs with recurrent seizure and controls (ratio in control dogs = 0.96; ratio in epileptic dogs = 0.93, p =0.096, Mann Whitney U test). However, the hippocampus was hyperintense on T2 and FLAIR images in 10 dogs from the seizure group, but none in the control group.

We therefore conclude that there are subtle changes in signal intensity of the hippocampus in some dogs with recurrent seizure disorders, although a significant change in hippocampal ratio was not observed. The presence of hippocampal sclerosis in dogs will need to be confirmed using histopathological techniques.

ABSTRACT #36

ASSOCIATION BETWEEN CLINICAL SIGNS AND MAGNETIC RESONANCE IMAGING IN DOGS WITH DEGENERATIVE LUMBOSACRAL STENOSIS. L. Perez-Klein[1], S.R. Platt[1], R. Dennis[1], F. Llabres-Diaz[1], F. McConnell[1], Katherine Rogers[2], V. Adams[2] [1]Centre for Small Animal Studies, [2]Centre for Preventive Medicine, Animal Health Trust. Newmarket. UK.

Degenerative lumbosacral stenosis (DLSS) is reported to be the most common disease of the cauda equina in large-breed dogs. It is characterized by combinations of intervertebral disk degeneration, intervertebral disk protrusion, subluxation of the facet joints, and thickening of the joint capsule and ligamentum flavum. Magnetic resonance imaging (MRI) is the most advanced imaging technique available to investigate the lumbosacral region, being the preferred method for diagnosis of lumbar disc herniations in humans. However, the sensitivity of the images of the region may lead to over diagnosis of disease. The aim of this study was to evaluate the degree of neural compression seen on MRI examinations in a series of normal dogs and dogs with DLSS in order to test the hypothesis that severity of clinical signs is directly related to the severity of cauda equina compression.

Dogs included in this study were medium to large, non-chondrodystrophoid dogs undergoing lumbosacral spinal MRI using a spine coil. Sixty-two dogs were included; 23 of them were normal dogs and 39 dogs presented with clinical evidence of LS disease. All dogs underwent a neurological exam and were placed into one of five clinical categories (0–4; normal-severe neurologic deficits). Sagittal and transverse T2-weighted 3 mm thick images of the LS area were obtained in all dogs included in this study. The dogs were placed into one of five MRI categories (0–4; normal-severe vertebral stenosis).

Cross tabulations and chi-square tests of association were performed to examine the relationship between clinical score and MRI score. Significance

was set at P=0.05 for all tests. Clinical score and MRI score were significantly associated (N=62, χ^2=38.8, 16 df, P=0.001). Dogs with less severe clinical scores had less severe MRI scores. The same was not true when only the LS cases were used (N=39, χ^2=7.2, 9 df, P=0.7). Combining MRI scores into 2 categories resulted in the association almost reaching statistical significance in the LS cases (N=39, χ^2=3.55, 1 df, P=0.06)

The most important finding of the study was the presence of an association between clinical signs and MRI findings. While there may be a significant imaging difference in clinically affected dogs between those with just pain and those with additional neurological signs, a larger study would be needed to show this. Based on the findings of this study, a total of at least 60 LS cases would be needed to show a statistically significant association. This study provides important new information about the imaging diagnosis of DLSS in dogs.

ABSTRACT #37

PREVALENCE OF AND RISK FACTORS FOR STATUS EPILEPTICUS OR CLUSTER SEIZURES WITH CANINE IDIOPATHIC EPILEPSY. R. Monteiro,[1] S. R. Platt,[1] V. Adams,[2] L. Wieczorek,[1] A. De Stefani[1].[1]Centre for Small Animal Studies & [2]Centre for Preventive Medicine, Animal Health Trust, Newmarket, Suffolk, UK.

Status epilepticus (SE) represents a continuous series of two or more discrete seizures lasting at least 5 minutes between which there is incomplete recovery of consciousness. SE affects dogs of all signalments and is associated with a variety of underlying diseases. Cluster seizures (CS) represent two or more seizures within a 24 hour period. The aims of this study were to investigate the prevalence of SE and CS in dogs with idiopathic epilepsy, whether there is any relationship between these two events and what risk factors exist for these events.

Medical records of dogs presenting with seizure disorders (2000–2004) were evaluated. All dogs included in the study were confirmed to have idiopathic generalised tonic-clonic seizures based upon (i) owner description of seizure events, (ii) normal physical and neurological examinations, (iii) normal blood work, (iv) normal thoracic and abdominal imaging, (v) normal cerebrospinal fluid analysis and (vi) normal cerebral magnetic resonance imaging. Variables reported for each dog included signalment, onset of seizure activity, anticonvulsant therapy where given, in addition to the occurrence of SE and/or occurrence of CS based upon the aforementioned definitions.

The proportion of cases with SE and the proportion of cases with CS were reported with 95% confidence intervals (CI). Logistic regression was carried out to examine the relationship between the occurrence of SE and CS with potential explanatory variables (age, gender, neuter status and treatment). Variables were selected for inclusion in the final model if they significantly improved the fit (likelihood ratio χ^2 statistic $P < 0.05$). Significance was set at $P < 0.05$ for all final models.

Data for 407 cases were included in the analysis. The mean (SD) age at diagnosis was 4 (2.6) years (range 2 months – 14 years and 5 months). There were a total of 10 cases with SE (2.5%, 95% CI: 1.0 – 4.0) and 166 cases with cluster seizures (41%, 95% CI: 36 – 46). There was no association between SE and cluster seizures (χ^2= 0.55, 1 degree of freedom, P=0.46). Treatment was significantly associated with the occurrence of SE and CS, with dogs on PB and KBr being more likely to suffer from SE and CS. Entire dogs were 1.9 times more likely than neutered dogs to suffer from CS. There were no breed influences on the occurrence of either SE or CS.

This study confirms a relatively low prevalence of SE and moderately high prevalence of CS in dogs with idiopathic epilepsy, which is important information for owners. Seemingly neutering may reduce the risk of CS in male dogs.

ABSTRACT #38

EPILEPTIC SEIZURES ASSOCIATED WITH INTRACRANIAL TUMORS IN CATS: RETROSPECTIVE STUDY OF 61 CASES. A. Tomek[1], S. Cizinauskas[3], M. Doherr[2], G. Gandini[4], A. Jaggy[1].[1]Department of Clinical Veterinary Medicine, Division of Animal Neurology, and [2]Division of Clinical Research, Vetsuisse Faculty, University Bern, Switzerland, [3]Small Animal Hospital AISTI, Vantaa, Finland, [4]Department of Veterinary Clinical Studies, University Bologna, Italy.

The purpose of this study was a retrospective analysis of feline population with intracranial neoplastic diseases referred to our institution, and the documentation of seizure patterns in these animals. In addition, groups of cats with and without seizure(s) within the population with intracranial neoplasia were compared. Special emphasis was given to the evaluation of tumor type, localization and size of the intracranial lesion and the correlation with seizure prevalence.

Sixty-one cats with histopathological diagnosis of intracranial tumor were identified. Fourteen cats (23%; Group A) had history of seizure(s). Forty seven cats (77 %; Group B) with neoplastic brain disease had no history of seizure(s). Generalized tonic-clonic seizures were seen in eight cats (57%). Both generalized and partial seizures were observed in two cats (14%), and three cats suffered exclusively partial complex seizures. One cat had partial complex seizures with secondary generalization. Clusters of seizures were observed in 6 cats. Status epilepticus was observed in one patient. The mean age of cats within group A was 7.9 years and within group B 9.3 years. The cats with lymphoma within group B and also within both groups A+B were significantly younger than cats with meningioma. In both groups meningioma and lymphoma were confirmed as most frequent tumor type, following by glial cells tumors. The prevalence of the seizures was 26.7% (4/15) in glial cell tumors and 26.3% (5/19) in lymphomas, followed by cases with meningiomas (15%; 3/20). In forty-two (69%) cases were the tumors localized in forebrain (supratentorial). All but two tumors from group A were localized just in forebrain. Parietal lobe and basal ganglia were mostly affected in group A, tumors in parietal and frontal lobe and in diencephalon were most frequent within group B. The prevalence of the seizuring animals with the neoplasia histopathologicaly localized just in forebrain was 28.6% (12/42). A positive association between localization of the tumor in the cerebrum and seizures occurrence was documented.

Results of our study indicate that gliomas and lymphomas have a tendency to cause seizures more frequently than meningiomas. The likelihood that cat will suffer seizures in addition to other neurological signs is when the tumor is located in basal ganglia or parietal lobe. None of the cats had suffered seizures if the tumor was localized in cerebellum or the infratentorial brainstem.

ABSTRACT #39

MAGNETIC RESONANCE IMAGING FINDINGS IN FINNISH SPITZ DOGS WITH FOCAL EPILEPSY R. Viitmaa R[1], Cizinauskas S[1], Bergamasco L-A[2], Kuusela E[1], Pascoe P[3], Teppo A-M[1], Jokinen TS[1], Kivisaari L[4], Snellman M[1]. [1]Department of Clinical Veterinary Sciences, University of Helsinki, Finland; [2]Department of Veterinary Morphophysiology, University of Turin, Italy; [3]Department of Surgical and Radiological Sciences, University of California, Davis, USA; [4]Department of Radiology, Helsinki University Central Hospital, Finland.

The purpose of this study was to describe magnetic resonance imaging (MRI) changes in a group of epileptic Finnish Spitz (FS) dogs with focal seizures in order to define the contribution of the MRI examination in diagnosing idiopathic and symptomatic epilepsy.

Two groups of dogs were studied: three healthy FS dogs that served as control animals, and 11 known epileptic FS dogs. Presence of at least two focal seizure episodes, complete seizure history and clinical data, absence of changes in general physical and neurological examinations, and normal blood examination, urinalysis and performed electroencephalography (EEG) were the minimum criteria for inclusion of dogs in the study. All control dogs underwent an identical work-up. The EEG was recorded under medetomidine (Domitor) sedation (14-channel monopolar montage). A 1.5 T MRI-machine was used to obtain T1-weighted (T1W) and T2-weighted (T2W) images in all three standard planes (sagittal, transverse, dorsal) during propofol (Rapinovet) infusion. Multiplanar reconstructions (MPR) were used instead of T1W images in four epileptic and all control dogs. Fluid-attenuated inversion recovery (FLAIR) sequence was performed in these four dogs in the dorsal plane. T1W images or MPR were repeated after IV contrast medium – gadolinium (Magnevist) injection. Cerebrospinal fluid (CSF) was collected after MRI examinations. Scans were reviewed independently by two radiologists (M.S. and L.K.). Special emphasis was given to seizure history to determine any correlations between seizure intervals and MRI findings.

CSF analyses of all dogs were within reference range. On EEG focal epileptic activity was found in 7/11 dogs (64 %) and generalized epileptic activity in 4/11 dogs (36 %). MRI detected changes in one epileptic dog. Mild contrast enhancement after gadolinium injection was identified in this dog's right parietal cortex. No such changes were observed in repeated magnetic resonance images. Two of the dogs were euthanised. No changes in brain histopathology were found.

Our results indicate that our FS dogs suffer from focal idiopathic epilepsy and have no detectable findings on MRI or pathology. MRI showed poor sensitivity in detecting epileptogenic areas in our patients with focal seizures. Reversible MRI changes in one dog could have been caused by seizures.

ABSTRACT #40

THE USE OF A QUESTIONNAIRE TO COMPARE CLINICAL CHARACTERISTICS AND TYPE OF SEIZURE OCCURRING IN 3 DOG BREEDS WITH IDIOPATHIC EPILEPSY. G. Holt[1], H.A. Volk[1] and K. Chandler[1]. [1]Department of Veterinary Clinical Sciences, Neurology, The Royal Veterinary College, Hatfield, UK.

The clinical presentations of seizures from 35 dogs previously diagnosed with idiopathic epilepsy by a UK referral hospital were recorded with a retrospective telephone questionnaire aimed at owners of epileptic dogs. The results were then classified according to a modified version of the International League Against Epilepsy (ILAE) seizure classification system. The three most commonly presented breeds were selected; Labrador Retrievers, Border Collies and German Shepherd dogs. All dogs had an extensive diagnostic work-up including MRI scans of the brain. Our results agreed with other recent studies; focal seizures were more common than generalized seizures, with over 60% of dog owners reporting signs of a presumed pre-ictal phase lasting a few minutes. Tonic-clonic was the most common presentation for generalized seizures. No correlation could be found between the age of onset of seizures and the type of seizure reported. Marked variation in the presentation of seizures was seen between breeds and our results suggest that Labrador Retrievers experience less severe seizures and respond better to anti-epileptic treatment, in comparison to Border Collies or German Shepherd Dogs. An unusual finding was that several of the Labradors Retrievers experiencing generalized seizures were recorded as being conscious throughout the event. Some evidence of audiogenic or 'mental stress' trigger factors were recorded in some cases. Our study highlights the usefulness of detailed questionnaires in gathering phenotypic information in epilepsy. Future plans involve developing the questionnaire to allow dog owners to complete it online and to develop a version for use in first opinion veterinary practices.

ABSTRACT #41

CRANIAL THORACIC DISC PROTUSIONS IN 3 GERMAN SHEPHERD DOGS. Lluis Gaitero, Sònia Añor. Servei de Neurologia i Neurocirurgia, Departament de Medicina i Cirurgia Animals, Facultat de Veterinària,Universitat Autònoma de Barcelona, 08193 Bellaterra, Barcelona, SPAIN.

Degenerative intervertebral disc disease is one of the most common neurological problems in dogs. Disc disease can result in extrusion (Hansen I) or protusion (Hansen II) of disc material into the spinal canal resulting in spinal cord compression. Classically, Hansen type II intervertebral disc disease is most commonly recognized in older, non-chondrodystrophic, large-breed dogs. Althought degenerative disc changes can occur at any spinal level, cervical and thoracolumbar intervertebral discs are more commonly affected. The presence of the intercapital ligament between rib heads (except for T1, T12 and T13), which lies under the dorsal longitudinal ligament, provides additional resistance and stability to the thoracic spinal column. Thus cranial thoracic intervertebral disc protrusions and extrusions are extremely rare.

The cases reported here describe the clinical, radiographic, and surgical findings, as well as the postoperative outcome in 3 German Shepherd dogs with T2-T3 disc protusions. The affected dogs (2 males, 1 female; ages from 7 to 9 years), were admitted for chronic progressive paraparesis. Duration of the neurologic dysfunction ranged from 2 to 6 months. General physical examinations were normal in all cases. On neurologic examination, two dogs were severely paraparetic, but still ambulatory. The third one was ambulatory and showed a severe, hypermetric truncal ataxia in the pelvic limbs. Conscious propioception was severely depressed in the pelvic limbs in all three dogs. Spinal reflex and cranial nerve examination were normal. Mild cranial thoracic hyperesthesia was elicited on deep palpation of the spine in one dog. Neuroanatomic localization was consistent with a T3-L3 myelopathy in all three patients. Lumbar myelography and post-myelographic computerized tomography revealed ventrolateral, extradural spinal cord compressions over the T2-T3 intervertebral disc, two on the right side and one on the left side. A T2-T3 hemidorsal laminectomy was performed in the three cases, allowing visualization of chronic disc protusions in all dogs. The dorsal annulus of the T2-T3 intervertebral disc was sectioned and excised in each case. All animals experienced immediate postoperative worsening of their neurologic status, progressing to non-ambulatory paraparesis, and one of them developed a unilateral Horners syndrome, ipsilateral to the laminectomy side. However, all were ambulatory 7–15 days post-surgery and the Horners syndrome resolved completely. One month after surgery, two of the dogs were mildly paraparetic and had mild conscious propioceptive deficits. The third dog returned to the previous preoperative neurological status two months after surgery.

Cranial thoracic disc herniations are rare in veterinary and human medicine. There are only 3 cases reported in the veterinary literature in different breeds of dogs. To the authors knowledge, this is the first cases series describing T2-T3 disc protusions in German Shepherd dogs.

ABSTRACT #42

COMPRESSIVE MYELOPATHY DUE TO SUBFASCIAL SEROMA FOLLOWING CERVICAL DORSAL LAMINECTOMY IN A ROTTWEILER. L.A. Wieczorek, S.R. Platt, A de Stefani, R. Dennis, A. Petite. CSAS, Animal Health Trust, Newmarket, Suffolk, UK.

Complications following an initially successful dorsal spinal cord decompression leading to compressive myelopathy include haematoma, empyema/ abscess formation, vertebral (sub)luxation, fractures and laminectomy membranes. Even though subcutaneous seromas are known to occur post surgically, this is, to the authorŝ knowledge, the first case description of a subfascial seroma, subsequent to cervical dorsal laminectomy, causing a compressive myelopathy.

An 11-month old male Rottweiler was presented with chronic progressive pelvic limb ataxia. Neurological examination was consistent with a C1-C5 neurolocalisation. Magnetic resonance imaging (MRI) revealed laterolateral and dorsolateral spinal cord compression at C2-C3 due to facet enlargement. A dorsal laminectomy at C2-C3 was performed and laminectomy edges were undercut, decompressing the cord. Post surgical recovery was uneventful, and the dog was ambulatory the following day. He exhibited tetraparesis 24 hours later, which progressed to tetraplegia within the next two days. No apparent swelling of the surgical site was present. Repeat MRI revealed marked disruption of the normal organisation of the soft tissue plane dorsal to the upper cervical spine. Several pockets with material of high signal intensity on T2-weighted images and low signal intensity on T1-weighted images dissected the fascial planes at the surgery site. A mixed intensity material was found at the laminectomy site compressing the spinal cord dorsally. Gradient echo images revealed a small area of extradural haemorrhage. MRI was consistent with dorsoventral compression of the spinal cord due to post-surgical seroma, admixed with haemorrhage and soft tissue components. Surgical revision was performed. When the suture of the deep fascia overlying the nuchal ligament was removed a large amount of fluid was relieved under seemingly great pressure. The laminectomy site was exposed and the spinal cord appeared of normal colour and shape. Before closure, a closed suction drain was placed. Culture of the seroma fluid revealed no bacterial growth. The dog recovered well and was discharged seven days later.

Compressive myelopathy due to subfascial seroma should be considered as a possible complication when the spinal cord is approached dorsally, in particular in the region of the cervical spine, where multiple soft tissue layers are dissected. Young, lively dogs might be at an increased risk for seroma formation. MRI is a sensitive method in detection of this complication. Surgical revision and drainage is indicated, which may result in quick recovery. Interestingly, in humans a suction drain is routinely placed between the muscle planes and the dura after cervical laminectomy, an approach worth considering in dogs at high risk for seroma formation.

ABSTRACT #43

BITE WOUNDS TO THE SPINE. Orit Chai, Merav H. Shamir. Koret School of Veterinary Medicine, The Hebrew University of Jerusalem Israel.

Bite wounds are mentioned in the literature as a possible cause of spinal injury but very little information is available regarding treatment considerations and prognosis. Performing meticulous debridement to all affected soft tissue and muscles as recommended for treatment of bite wound has to be weighted against leaving as much soft tissue and muscles around the fractured vertebrae as recommended for treatment of back fractures. This dilemma presents a medical challenge.

A retrospective study was done on dogs and cats presented with bite wounds that caused fractured vertebrae between the years 2002–2005. Information obtained from the medical records included signalment, neurological status, additional injuries, radiographic evaluation, medical treatment, method of stabilization used and outcome.

Five dogs and two cats, aged 3 months to 6 years, were included. All weighted less than 10 kg. Bite wounds were found in the neck (5/7) and in the lumbar region (2/7). Additional injuries included pneumothorax (1/7), tracheal tear (1/7) and tarso-metatarsal sub-luxation (1/7). Neurological status varied between quadriparesis (5/7), paraparesis (1/7) and paraplegia (1/7). Radiographic finding included vertebral fracture with displacement, endplate fractures and narrowed disc space. Therapy included antibiotics (7/7), Methyl prednisolone (4/7) surgical debridment of the wounds (7/7), stabilization of the spine using Scotch-cast ® cast (5/7) and a cage rest (2/7).

Five cases regain normal gait, one remained paraparetic and one died from organ failure. The time for full recovery was 14–45 days (5/7) but the ability to walk was regained much sooner (< 24 days).

We concluded that bite wounds to the spine present a unique pathological entity combining contaminated wounds with vertebral fractures. The use of more conservative surgical debridement with broad spectrum antibiotics, external coaptation and methyl prednisolone in the first 24 hours is shown to give a good to excellent prognosis in most cases (6/7).

ABSTRACT #44

10 CLINICAL CASES OF ATLANTOAXIAL SUBLUXATION IN DOGS. J. Jeserevics[1,2], P. Srenk[1], J. Beranek[1], S. Cizinauskas[2]. [1]Referral Small Animal Clinic JAGGY, Brno, Czech Republic; [2]Referral Animal Neurology Clinic AISTI, Helsinki.

Atlantoaxial subluxation (AAS) is an inherited or acquired disease mostly described in small and miniature breed dogs. Veterinary surgeons tend to use ventral stabilisation techniques more frequently in the latest reports. Procedures often performed are: 1) transarticular pinning with K-wires, 2) transarticular pinning and placing of cortical screws with polymethylmetacrylate reinforcement and 3) transarticular cortical screw placing. Complications are most often caused by misplacement or migration of implants. Purpose of this article is to present 10 cases of AAS treated with 1.5 mm transarticular cortical screw placing through the atlantoaxial articulations and to compare this technique with other ventral stabilisations techniques.

10 miniature breed dogs with confirmed diagnosis of AAS were treated surgically between year 2000 and 2005. Data collected for each case were signalement, anamnesis as well as results of the neurological examination. First clinical signs were noted within a first year of life in all dogs but one. Diagnosis in all dogs was confirmed after radiological examination. Increased distance between arch of atlas and dorsal process of axis was noted in all cases. Arthrodesis of atlantoaxial articulation was made and two 1.5 mm cortical screws were used for stabilization of C1 – C2. In radiographs made postoperatively distance between arch of atlas and dorsal process of axis was found to be normal in all cases. Follow–up information was obtained through the phone one month and 2 – 32 months after surgery. Follow-up condition of seven surviving dogs was defined as ''good'' - without clinical abnormalities, ''favourable'' - rare episodes of rigidity, pain and ''poor'' - status similar or worse as before surgery.

Four dogs did not have postoperative complications and continuous recovery was observed. Three dogs get worse after external trauma 1, 20 and 53 days after surgery respectively. Conservative therapy leads to improvement of clinical condition of these dogs. Two dogs get worse two and five days after surgery and owners decided for euthanasia. One dog had cardiopulmonary arrest and died 5 hours after surgery. Follow-up condition was ''good'' in four dogs and ''favourable'' in three dogs.

Surgical treatment is usually necessary for fixation of AAS. Stabilisation of AAS with transarticular cortical screws is a relatively safe and easy technique. Success rate in present study is comparable to the other published techniques. Reduced activity for approximately 4 – 6 weeks are of paramount importance in order to provide time for adequate bone fusion between the first and the second cervical vertebrae.

ABSTRACT #45

HIGH RESOLUTION PROTEIN ELECTROPHORESIS OF CANINE CEREBROSPINAL FLUID AND SERUM IN INFLAMMATORY DISEASES OF THE NERVOUS SYSTEM: RETROSPECTIVE STUDY OF 61 CASES. S. Behr[1], C. Trumel[2], L. Cauzinille[1], F. Palanché[3], J.-P. Braun[3]. [1]Clinique Frégis, Arceuil, France; [2]Département des Sciences Cliniques des Animaux de Compagnie et de Sport, Ecole Nationale Vétérinaire de Toulouse, France; [3]Département des Sciences Biologiques et Fonctionnelles, Ecole Nationale Vétérinaire de Toulouse, France.

This study was performed to investigate the diagnostic relevance of cerebro-spinal fluid (CSF) High Resolution Electrophoresis (HRE) in inflammatory diseases of the nervous system.

This laboratory technique was realized on 61 samples of CSF and serum, on the same analytical run as recommended in human medicine. According to the final diagnosis, cases have been classified in two categories central nervous system infectious diseases (n=14) and inflammatory non-infectious diseases (INI, n=47). The INI group has been subdivided into encephalitis of unknown origin (n=20), aseptic suppurative meningitis (n=18), acute idiopathic polyradiculoneuritis (n=6) and other (n=3). The final diagnosis was based on the classic epidemiological, clinical, laboratory or pathology findings of these diseases.

The albumin quotient (AQ) highest median value was found in the aseptic suppurative meningitis group but no statistical differences were found between this group and the others. The AQ, calculated with this technique, did not bring more information. Moreover, the electrophoretic profiles were not characteristic of any disease. Even if HRE gave sharper bands than normal electrophoresis, a visual detection of an abnormal CSF electrophoretic profile compared to the serum was too subjective. This is indicative of the imprecision of HRE to detect an intrathecal secretion of immunoglobulin. In conclusion, this study did not confirmed that High Resolution Electrophoresis on paired CSF and serum samples was a valuable ancillary diagnostic tool for canine CSF protein complementary analysis in inflammatory diseases of the nervous system.

ABSTRACT #46

EVALUATION OF PCRs FOR THE DIAGNOSIS OF *NEOSPORA* AND *TOXOPLASMA* INFECTION IN CANINE CEREBROSPINAL FLUID IN A CLINICAL SETTING. I.C. Boettcher[1], A.M. Tenter[2], R. Carlson[1], A. Tipold[1]. [1]Dept. of Small Animal Medicine and Surgery, [2]Institute for Parasitology, University of Veterinary Medicine Hannover, Germany.

The clinical diagnosis of infectious diseases is frequently cumbersome, as antibody (ab) detection might be unspecific in serum and cerebrospinal fluid (CSF). The detection of a specific antigen in the CSF is thought to be more helpful. In the present study our experience with PCR testing for *Neospora* and *Toxoplasma* DNA in the CSF of 28 dogs is described.

As controls, 3 dogs with histopathological diagnosis were used. One positive control consisted of a case with meningoencephalitis due to *Neospora* infection, in which the diagnosis was additionally confirmed by infection of mice. Two of these cases served as negative controls (intracranial lymphoma and fibrocartilaginous infarction). The other 25 dogs with a presumptive clinical diagnosis were divided in 2 groups: 8 of them served as presumptively negative samples (idiopathic epilepsy, facial paralysis, degenerative spinal cord disease, steroid responsive meningitis-arteriitis, discospondylitis). In the remaining 17 cases, a clinical diagnosis of meningoencephalomyelitis of unknown origin (n=13) or intracranial neoplasia (n=4) was made after CSF examination and MRI findings. For *Neospora*, a fragment of the Nc5 gene was amplified according to a standard protocol using the NP6+/21+ primer pair. For *Toxoplasma*, two newly developed real-time PCRs were used, one based on the B1 gene and another based on a 529 bp repeat unit with a high copy number in the *Toxoplasma* genome. Antibodies were determined in CSF and serum by an indirect immunofluorescent antibody test (IFAT).

Of the 10 presumptively negative samples, 7 dogs had positive PCR results (2 for *Neospora*, 4 for *Toxoplasma*, 1 for both) and 3 were negative. The dog with histopathologically confirmed *Neospora* infection was PCR positive for both *Neospora* and *Toxoplasma* in CSF as well as in brain tissue. In the remaining 17 dogs with a clinical diagnosis of meningoencephalomyelitis or neoplasia, PCRs were positive in 9 animals (1 for *Neospora*, 3 for *Toxoplasma*, 5 for both) and 8 dogs were negative. In only one of these cases good response to treatment and high serum ab titer of 1:40960 and CSF ab titer of 1:5120 against *Neospora* supported our PCR findings. The other dogs had serum ab titers from 1:20 to 1:640 for *Toxoplasma* and from 1:20 to 1:40 for *Neospora*. CSF and serum IgA and IgG levels, CSF cell count and CSF total protein, immunosuppressive and antiparasitic treatment or travelling to southern countries did not influence PCR results.

In the current study, PCR amplicons were found in presumptively negative controls and in patients with a parasitic encephalitis as differential diagnosis. We excluded technical problems using several controls for the PCRs. In conclusion, there is a high suspicion that latent infections exist in the central nervous system and the use of PCR to diagnose an encephalitis due to *Neospora* or *Toxoplasma* infection has to be newly discussed.

Abstract #47

MENINGEAL CELL OCCURRENCE IN CEREBROSPINAL FLUID SPECIMENS. A. Wessmann[1], H.A. Volk[1], K. Tennant[1], K. Chandler[1]. [1]The Queen Mother Hospital, The Royal Veterinary College, North Mymms, United Kingdom.

Cerebrospinal fluid (CSF) cytology is an important tool for diagnosing neurological diseases. The significance of meningeal cells in canine CSF is uncertain. A CSF analysis of a dog with a meningioma in the cervical spinal cord revealed meningeal cells (1). However, a retrospective study on 77 dogs

with primary brain tumours including 16 meningiomas revealed no neoplastic cells in the CSF (2). Meningeal cells are reported as normal findings in canine CSF and were believed to be associated with the sampling technique via pneumoencephalography or ventricular puncture (3).

This study investigates the hypothesis that the occurrence of meningeal cells is not related to any specific underlying disease. Dogs undergoing investigations at The Royal Veterinary College between 2002 and 2005 involving a CSF analysis were recruited and 411 canine CSF samples were obtained. The presence of meningeal cells was related to disease groups with and without suspected meningeal involvement. Meningeal involvement could not be excluded in all cases with primary inflammatory CNS disease (group 1), compressive or traumatic lesions of the meninges including disc herniation, trauma, malformation (2) and neoplasia (3). Meningeal involvement was not suspected in idiopathic, metabolic, vascular, neuromuscular and in non-neurological diseases (4). Meningeal cells were identified by their morphological characteristics during cytological examination and occurred in 28/411 dogs (7%). Meningeal cells occurred up to 7% in diseases without suspected meningeal involvement (17/240 dogs). Eight per cent (3/36 dogs) of the CSF specimens taken from dogs with neoplastic CNS disease showed meningeal cell occurrence but no confirmed meningiomas showed meningeal cells in the analysed CSF. Eight per cent (7/87 dogs) of dogs having compressive lesions (group 2) and only 0.2% (1/48 dogs) of dogs having inflammatory CNS disease had meningeal cell occurrence in the CSF. In this study, there was no significant evidence that the presence of meningeal cells is related to disease in the central nervous system in the studied population.

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Abstract #48

A COMPARATIVE STUDY OF SMEAR CYTOLOGY AND HISTOLOGY IN THE DIAGNOSIS OF CANINE AND FELINE NERVOUS SYSTEM LESIONS.

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Intra-operative cytological evaluation of smear preparations from Nervous System lesions is a common and well established diagnostic procedure in human medicine and an accurate method that can also be used to determine the presence of neoplasia in the brain of dogs and cats.

42 nervous System lesions taken from 33 dogs and 9 cats affected by neurological deficits were collected during craniotomy or laminectomy or during necropsy. Cytological samples were prepared using the squash technique, then air dried and stained in automatic slide stainer with the May-Grünwald Giemsa method. All histological specimens were paraffin embedded and routinely stained with H&E. Cytologist's and pathologist's evaluations were then separately performed. When considered appropriate, special and immunohistochemical stainings were used. Cytological diagnostic consistence with the histological diagnosis was checked at 95% confidence interval.

In 32 cases (76.2%) the cytological diagnosis fell into group a), i.e. *completely correct cytological diagnoses,* when it was completely in accordance with final histological diagnosis. Seven (16.6%) and three cases (7.2%) fell into group b), i.e. *incompletely correct cytological diagnoses*, and group c), i.e. *incorrect cytological diagnosis*, respectively. Group b) included cytological diagnoses that were only partially in accordance with the final histological diagnosis, while group c) included those that did not correspond to the general histological lesion. Cytological diagnoses from group a) and group b) were considered satisfactory and totaled 39 out of 42 cases (92.8% of total cases).

18/18 meningiomas, 4/7 peripheral nerve sheath tumors (PNSTs), 3/4 sarcomas, 2/3 carcinomas, 1/1 lymphoma, 1/1 ependymoma, 1/1 plasmocytoma and 2/2 astrocytomas fell into group a), 3/7 peripheral nerve sheath tumors, 1/4 sarcoma, 1/1 benign mesenchymal tumor, 2/2 choroid plexus carcinoma fell into group b) and the remaining 1 carcinoma from nasal cavities, 1 granulation tissue and 1 vascular hamartoma fell into group c).

Cytological misinterpretations may especially occur owing to the heterogeneity of the primitive tumor cell population and the high degree of anaplasia or the overlapping of morphological features from neoplastic and not neoplastic lesions. Therefore, diagnostic data provided by cytology should always be supported by accurate clinical and radiological investigations.

ABSTRACT #49

LATE ONSET CEREBELLAR CORTICAL DEGENERATION IN TWO ADULT PITBULL TERRIERS.

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Two adult male Pitt Bull littermates, from a litter of eight puppies, were examined for hypermetria and a wide based stance. Dog 1 – The first dog evaluated was four years old. The neurological signs had progressed over a 6-month period. On initial gait analysis the dog frequently stumbled and had general difficulty maintaining balance. The gait was hypermetric in all limbs and a mild intention tremor of the head was present. Postural responses were present but they were considered to be exaggerated in all limbs. Examination of the cranial nerves was normal. All the myotatic reflexes were brisk but not considered clonic. A bilateral vertical nystagmus was present. These signs were compatible with a vestibulo-cerebellar disorder. Neoplasia and encephalitis were considered as differential diagnoses but the slow, steady rate of progression was also suggestive of late-onset cerebellar degeneration such as abiotrophy or storage disease. Results of haematology, serum biochemical analyses, urinalysis and analysis of CSF were within reference ranges. A presumptive diagnosis of cerebellar cortical degeneration was made. Due to the poor prognosis, euthanasia and necropsy were performed.

Dog 2 - Two months later a 5-year-old littermate was examined with the same signs. The owner first noticed trembling of the pelvic limbs, which had progressed over several months. An intention tremor of the head was detected when the dog was at rest as was a bilateral nystagmus of variable direction. Postural responses were exaggerated. Initial work-up was as for dog 1 and the results were within reference ranges. A diffuse atrophy of the cerebellum was identified in both dogs using magnetic resonance imaging. Both dogs were euthanised at the owner's request. Grossly, the cerebellum appeared reduced in size and subjectively the folia appeared narrowed. Microscopically, there was a diffuse reduction of the molecular and granular layers. Purkinje cells were markedly reduced in number, and the remaining Purkinje cells were shrunken, dark and eosinophilic. There was no evidence of storage product in any cell, and the pattern of degeneration was atypical for any documented toxin. The final diagnosis was cerebellar cortical atrophy.

Due to the lack of an identifiable aetiology and the similarity to other breeds with heritable cerebellar abiotrophy, the Pitbulls described in this report may represent a familial adult onset variant. Additional cases are necessary to prove heritability.

ABSTRACT #50

DEVELOPMENT OF WHITE MATTER ABNORMALITIES FOLLOWING ACUTE SPINAL INJURY IN DOGS AND CATS.

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Experimental models have shed light on the physiological changes that follow spinal injury and have identified ways in which damage might be minimised and recovery enhanced. Few of these interventions, however, have made a significant impact on the treatment of clinical cases. The aim of the current study was to examine the histological changes that occur following naturally occurring spinal injury in dogs and cats, to provide insights into the reason that function is lost following injury and to assess the extent to which experimental models mimic real life injuries.

Seven paraplegic animals were euthanased at the request of their owners, between 24 hours and 3 months following natural spinal injury, and were immediately perfused with fixative. At early time points, there was haemorrhage and oedema scattered throughout the spinal cord, with widespread axonal swelling and degeneration of myelin sheaths. At later time points, much of the myelin debris was removed by macrophages and morphologically normal axons could be identified alongside both demyelinated and remyelinated axons. Axons were relatively well preserved in the dorsal part of the cord, distant from the site of compression, and around the periphery adjacent to the pial surface. Very few axons larger than 5μm in diameter were seen in any of the cases, indicating that smaller diameter axons were better preserved than larger ones.

Ultrastructural analysis of the lesions confirmed the presence of demyelinated axons and also revealed axons that were remyelinated by both oligodendrocytes and Schwann cells; remyelination was extensive in the case examined 3 months following injury. Paranodal myelin abnormalities were observed in sagittal sections in a number of cases, usually in axons in which remaining myelin appeared normal. In several animals, a cystic cavity was present at the centre of the lesion, containing a network of interconnecting cell processes. Detailed examination revealed that these processes were predominantly astrocytic and that clusters of small diameter unmyelinated axons were also present.

The presence of remyelinated axons is an important finding, since this is the first report to describe extensive remyelination following naturally occurring spinal injury. We also identified small diameter axons at the epicentre of the lesion and paranodal demyelination in axons that appeared otherwise normal. These findings might have important functional implications for both animals and humans and are important findings to consider when examining the spinal cord from individuals involved in therapeutic trials.

ABSTRACT #51

DIFFERENTIAL EXPRESSION OF CD45 AS SIGN OF MICROGLIAL GRADED RESPONSE TO DISTURBANCES IN THE CNS. V. M. Stein[1], W. Baumgärtner[2], S. Schröder[1], A. Zurbriggen[3], M. Vandevelde[3], A. Tipold[1]. [1]Department of Small Animal Medicine and Surgery; [2]Institute of Pathology; University of Veterinary Medicine, and Centre for Systems Neuroscience, Hannover, Germany; [3]Institute of Animal Neurology, University of Berne, Switzerland.

CD45 also named leukocyte common antigen (LCA) is a molecule on the surface of nearly all white blood cells and serves as co-stimulator for lymphocytes. In the brain CD45 is used to distinguish microglial cells with a characteristic phenotype of CD11b/c^{+} and CD45^{low} or even absent CD45 expression for the dog from other CNS macrophages which show an expression of CD11b/c^{+} and CD45^{high}. In pathological conditions in rodents microglia is known to upregulate expression of various surface molecules, such as CD45.

The expression of CD45 on microglia of 42 dogs was examined by means of flow cytometry. The dogs were classified in two examination groups according to histopathological diagnosis of the CNS. Microglial cells of dogs without changes in the CNS (group I; n = 22) showed absent or extremely low expression of CD45. In contrast, in 1/3 of the dogs with different intracranial diseases (group II; n = 20) a high percentage of CD45^{+} microglial cells could be detected with a small subpopulation expressing CD45 with the same intensity such as canine monocytes. Dogs with an expression of CD45^{high} suffered from long-lasting CNS diseases with seizure activity, however no specific disease could be related to this phenomenon. Most of these dogs were young.

Microglial differential expression of CD45 is therefore regarded as a sign for graded response of this cell population to a given challenge in the CNS with higher expression in severe lesions most probably to enhance immune defence mechanisms.

ABSTRACT #52

INTRACRANIAL MENINGIOMA IN THE DOG: IMMUNOHISTOCHEMICAL ANALYSES OF PROLIFERATIVE POTENTIAL AND TUMOR SUPPRESSORS p53 AND PTEN. D. Janik[1], B. Stierstorfer[1], W. Schmahl[1]. [1]Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, Ludwig-Maximilians-University, Munich, Germany.

Intracranial meningioma is the most frequent tumor of the canine central nervous system. Due to its advantageous anatomical localization and the generally low grade of malignancy, this tumor is more and more the target of therapeutic approach. Labeling indices (LI) of proliferating cell nuclear antigen (PCNA) and Ki-67 are routinely used in human meningioma patients to estimate recurrence and survival.

One aim of this research was to determine PCNA and Ki-67 LI on 18 intracranial canine meningiomas and to critically compare the applicability of WHO histological classifications of humans and domestic animals to these cases. The tumors were classified into three groups according to the grading-system exclusively established in human medicine. The cases of group I comprised common meningiomas (transitional [n=6], fibrous [n=3], angiomatous [n=1], granular cell [n=1], meningothelial [n=1]) and group III anaplastic meningioma [n=1]. In all tumors PCNA LI was higher than Ki-67 LI. The meningiomas with atypical features (group II) showed mean indices between those of group I and III. Statistical analyses exhibited significant correlation with die mitotic index, indicating that PCNA and Ki-67 are appropriate markers for evaluation of the proliferative potential in canine meningiomas.

Somatic mutations of tumor suppressor *p53* are among the most common genetic alterations reported in human cancer. p53 was detected by immunohistochemistry using the polyclonal p53 antibody CM1. The p53 LI was determined and compared with histological subtype and proliferative activity. Common and atypical meningiomas generally exhibited low p53 LI while the anaplastic case was highly p53 immunoreactive. p53 LI was found to be significantly correlated to PCNA LI or Ki-67 LI.

Alterations of tumor suppressor gene *phosphatase and tensin homolog deleted on chromosome ten (PTEN)* have been found in a wide variety of human cancers, including tumors of the brain. In this study immunohistochemistry for PTEN was performed in meningioma for the first time. PTEN immunoreactivity in most of the tumors was homogeneous and involved the cytoplasmic and nuclear compartment. The anaplastic meningioma exhibited an irregular staining pattern with focal loss of PTEN expression.

A growing understanding of mechanisms fundamental to canine meningeal tumor development allows consideration of treatment modalities from human medicine and optimizing cancer therapy of dogs.

ABSTRACT #53

LONG-TERM ASSESSMENT OF A CURRENTLY RECOMMENDED THERAPEUTIC PROTOCOL IN 12 DOGS WITH TENTATIVE DEGENERATIVE MYELOPATHY: A RETROSPECTIVE STUDY. Z.S. Polizopoulou, A.F. Koutinas, M.N. Patsikas, N. Soubasis, T. Danourdis. School of Veterinary Medicine, Aristotle University of Thessaloniki, Stavrou Voutyrastr. 11, GR 546 27 Thessaloniki, Greece.

Degenerative myelopathy (DM), a disease of unknown yet presumed immune-based etiology, is the most common cause of spinal cord dysfunction in medium and large breed dogs older than 5 years of age. Clinical signs are typical of a chronic, progressively worsening, non-painful thoracolumbar (T3-L3) myelopathy and a tentative diagnosis is based on signalment, clinical signs and exclusion of other spinal cord disorders (CSF analysis, diagnostic imaging studies). An integrated management of DM involving administration of medications, dietary supplementation and supportive measures has been proposed, however the progression of the disease is usually not altered. Long-term prognosis is poor, with most dogs euthanized within 6–12 months due to severe paresis or plegia. The objective of this study was to assess the long-term efficacy of the currently recommended therapeutic protocol in 12 dogs with tentative degenerative myelopathy (DM) that were followed up for 6–12 months.

Twelve dogs with a history of progressive posterior body ataxia and/or paraparesis, of 2 to 12-month duration, were referred for evaluation and were included in the study. Physical, neurological and clinicopathological (CBC, serum biochemistry, urinalysis, CSF analysis) examinations were performed in all cases. The assessment of locomotor dysfunction was based on a scoring system ranging from I to V (I-mild ataxia, II- moderate paraparesis and ataxia, III-severe paraparesis and ataxia, IV-paraplegia, V-paraplegia and loss of deep pain sensation). In all cases diagnostic imaging studies included survey radiographs of the entire spinal column and lumbar myelography, whereas in 4 dogs further investigation of the spinal cord with MRI was also pursued.

The therapeutic protocol applied included the daily administration of ?-aminocaproic acid (EACA, 500 mg TID PO) and acetylcysteine (25 mg/kg BW TID PO for 2 weeks), plus vitamin B, C and E supplementation. Prednisolone, at an anti-inflammatory dose of 1 mg/kg BW SID PO, was also given for the first 2 weeks post-admission and upon worsening of neurological signs, as a rescue drug. The owners were further instructed to exercise their dogs on a daily basis, either by walking or swimming, starting from 10–15 and gradually increasing in duration up to 30 to 45 minutes. During the follow-up period that lasted from 6 to 12 months, the dogs were being reexamined every month, unless there was worsening of neurological signs that prompted otherwise.

Nine of the dogs were males and 3 were females and they all belonged to large breeds, of which 9 were German shepherds. The age of the animals ranged from 5 to 12 years and they were current on vaccinations. Initial physical and clinicopathological evaluation was unremarkable. In 7 cases asymmetry of neurological signs with only one hind limb being initially affected, was reported to occur. Upon neurological examination the lesions were localized at the thoracolumbar spinal cord segments (T3-L3) in all 12 dogs, which demonstrated hind limb proprioceptive deficits of various degrees. Motor deficits were also noticed in 9/12, further classified as mild (grade II) in 7/12 and moderate (grade III) in 2/12 animals. Both survey radiographs and lumbar myelograms failed to show evidence of spinal cord compression or space-occupying lesions. Four dogs exhibited a transient worsening of their neurological status immediately following contrast studies, which however resolved upon initiation of prednisolone administration and within 48 hours. In 4/12 cases, which were further investigated by MRI, demyelinating lesions compatible with those of DM were amply demonstrated. During the follow-up period the progression of neurological dysfunction was slow but relentless in all cases, resulting to spastic paraplegia (grade IV) in 7/12 or severe paraparesis (grade III) in 5/12 dogs. Euthanasia was done in 6 paraplegic and 3 severely paraparetic dogs upon their owners' request, however permission for necropsy was denied, thus making the histopathological confirmation of clinical signs impossible.

The results of this study showed that the currently recommended therapeutic protocol may not be as beneficial, as it has been suggested,

since the progression of neurological dysfunction was not halted or even slowed in dogs with DM, admitted with only spinal ataxia or motor deficits.

ABSTRACT #54

NEW ASPECTS ON HEREDITARY ATAXIA IN SMOOTH HAIRED FOX TERRIERS. C. Kronberg[1], L. Lüdtke[2], K.H. Jäderlund[3]. [1]Albano Small Animal Hospital, Stockholm, Sweden, [2]National Veterinary Institute, Uppsala, Sweden and [3]Swedish University of Agricultural Sciences, Uppsala, Sweden.

Clinical course, histopathological changes in the spinal cord, and genetic aspects on hereditary ataxia in smooth haired fox terriers was described from Sweden in the ′50s and ′60s. Since then, a similar clinical condition in Jack Russell Terriers (JRT), with comparable histopathological changes, has been described. Since the fox terrier breed was involved in the development of the JRT, the disease was suspected to be the same in the two breeds. However, JRTs with hereditary ataxia also have clinical signs relating to degenerative lesions detected in the brain, which was not the case in smooth haired fox terriers with hereditary ataxia.

Recently in Sweden, in a fox terrier litter of seven puppies, three puppies developed clinical signs consistent with hereditary ataxia. Several ancestors to this litter were closely related to formerly affected dogs. Two of the ataxic puppies were clinically and neurologically examined at the Swedish University of Agricultural Sciences at an age of 7 and 10 months, respectively. One of those was euthanized and autopsied.

At examination, neurological deficits were prominent and mainly in accordance with earlier descriptions. Additionally, both puppies expressed severely depressed to absent menace responses bilaterally, a neurological sign not possible to explain by spinal cord involvement. At necropsy, degenerative changes of myelin sheets and axons were found in white matter of the spinal cord, consistent with histopathologic lesions in hereditary ataxia. Degenerative changes were also found in mesencephalon, cerebellum, medulla oblongata, obex and a lumbar peripheral nerve. When compared to the described histopathology of hereditary ataxia in JRTs, this foxterrier seemed to have the same kind of degenerative changes, although milder than and not fully as widespread as in the JRTs. This finding could reflect either another stage of a pathological process, or different diseases in the two breeds.

In conclusion, smooth haired fox terriers with hereditary ataxia may have neurological signs arising from lesions in the brain, as well as histopathologic lesions in the brain.

ABSTRACT #55

NEURAL TISSUE MOBILISATION IN THE DOG: ANATOMICAL BASIS AND POTENTIAL USE IN LUMBAR SPINAL NERVE DISORDERS. F.I. Grünenfelder, A. Boos, M. A. Mouwen, and F. Steffen. Neurology, Clinic for Small Animals and Department of Veterinary Anatomy, Vetsuisse Faculty, University of Zurich, Winterthurerstrasse 260, 8057 Zurich, Switzerland; Email: e-mail: fsteffen@vetclinics.unizh.ch, e-mail: fgruenenfelder@vetclinics.unizh.ch and e-mail: boosa@vetanat.unizh.ch

Neural tissue mobilisation has not become a standard procedure in small animal clinical neurology so far, but has gained increased attention in the rehabilitation of people with spinal and lumbar nerve disorders. The first aim of the study was to establish passive physical exercises such as the straight leg raising (SLR), the passive knee bending (PKB) and the dural stretch. The second aim was to evaluate the measurable anatomical effect of the applied physical exercises from nerve root L4 to L7 and the dura at the level Th 13 L1 in ten healthy beagles.

Fifteen beagles between 1 and 8 years old and between 7 – 14 kg BW were euthanised with Thiopental i.v. and bled. The dogs were immediately frozen at −25° for conservation. The dogs were defrosted during 24 hours before surgical preparation. Preparation of the nerve roots L4 to L7 and a Th13/L1 laminectomy were performed with surgical standard procedures. Nerve roots and Dura were marked with resin paint in neutral position of the hind limb. For scaling the region of interest, a Schirmer -Tear -Test strip was applied next to the mark. The SLR, the PKB and the dural stretch were established with the first five beagles. To obtain constant results, the angles of the different parts of the exercises were controlled goniometrically. The angles were selected under the following criteria: the applied forces had to be tolerated in live animals and the nerve root was mobilised to a maximal degree. The exercise was divided into three parts.

Following angles were selected for the PKB: In the neutral position the pelvic limb is hold parallel to the table with an angle of 90° between trochanter major and the lumbal spine. The first part is an extension of the hip from 90° to 150° while keeping the leg parallel to the table. The second part is an abduction of the pelvic limb of 80° while keeping the leg in extended position. This was defined as the pretension position. The third part is a continuous movement from 40° flexion to 180° extension of the knee. This is considered to be the therapeutic part during which the nerve root glides back and forward at the level of the foramen intervertebrale.

Following angles were selected for the SLR: In the neutral position the pelvic limb is hold parallel to the table with an angle of 90° between trochanter major and the lumbal spine. The first part is a flexion of the hip from 90° to 35° while keeping the leg parallel to the table. The second part is an outward rotation of the hip of 30° while keeping the leg in the flexed position. This was defined as the pretension position. The third part is a continuous movement from 40° flexion to 180° extension of the knee. Palpable anatomic landmarks such as the trochanter major, the lumbal spine the condylus lateralis and the maleolus lateralis were used for measuring the angles.

The dural stretch: The dog's spine is flexed until the chin gets in contact with the pelvic limbs. This was defined as the pretension position. The second part is a continuous maximal flexion and maximal extension of the atlantooccipital joint. All exercises were performed with the animal lying in lateral position. Distances between neutral and maximally mobilised position of the nerve roots were digitally photographed and processed with Madena $X_{©}$ medical viewer.

The mean maximal gliding distances in PKB for L4 was 0.73 mm with a standard deviation (SD) of ±0.45 mm and a standard error of mean (SEM) of ±0.15 mm, for L5 1.54 mm with a SD of ±0.52 mm and a SEM of ±0.17 mm, for L6 1.16 mm with a SD of ±0.41 mm and a SEM of ±0.14mm. The mean maximal gliding distances in SLR for L7 was 0.94 mm with a SD of ±0.4 mm and a SEM of ±0.13 mm. The dural stretch exercise showed a mean maximal gliding distance of 0.66 mm with a SD of ±0.42mm and a SEM of ±0.15 mm. The accomplished gliding distance between hip extension and additional rotation as well as the extension and flexion under pretension was significant with a $p < 0.05$. No correlation was found between gliding distances and crown rump length or bodyweight. However, correlations between these parameters could be present in very large or small dogs. Compared with human studies, gliding distances in these dogs are up to ten times smaller[1]. The exercises are designed to support the recovery of dogs after lumbosacral surgery with L7 nerve root entrapment, to prevent tethered cord syndrome after thoracolumbar spine surgery, to reduce radicular pain by reduction of intraneural edema and to enhance axonal regeneration by inducing axonal retrograd and antegrad flow[2]. The results of this study are intended to form a basis for their application in clinical trials in dogs.

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Research Abstracts 17th Annual ECVN & ESVN Symposium, Glasgow, UK, 23rd – 25th September 2004

ABSTRACT #1

MRI FINDINGS IN NEOPLASTIC AND NON-NEOPLASTIC BRAIN LESIONS IN DOGS AND CATS. G. B. Cherubini, P.Mantis, C.R. Lamb, T. Martinez, R. Cappello. The Queen Mother Hospital for Animals, The Royal Veterinary College, London UK.

The aim of this study was to identify MRI signs that aid differentiation of neoplastic versus non-neoplastic brain diseases in dogs and cats.

MRI of 36 dogs and 13 cats with histopathological diagnosis of intracranial disease (30 primary and 3 metastatic brain tumours, 11 infectious/inflammation diseases, 3 vascular lesions, 1 degenerative disease

and 1 development malformation) were reviewed retrospectively. Accurate diagnosis of neoplastic versus non-neoplastic lesion on the basis of these features was not always possible because of common MRI appearance.

However, upon univariate analysis of 24 MR signs, there were 7 that had a significant association with neoplasia: single lesion ($p = 0.004$), regular shape ($p = 0.0015$), dural contact ($p = 0.040$), dural tail ($p = 0.005$), bone lesions ($p = 0.008$), mass effect ($p = 0.002$), and enhancement after administration of gadolinium ($p = 0.025$). Age was also found to be statistically significant ($p = 0.0001$). MRI features of non-neoplastic brain diseases in dogs and cats were more variable and less specific than those of brain neoplasia.

Tissue biopsy remains necessary for a definitive diagnosis of intracranial diseases.

ABSTRACT #2

A NOVEL, PROGRESSIVE, SCLEROSING, PANENCEPHALITIS IN A HORSE. J. M. Swain, N. P. H. Hudson and I. G. Mayhew. University of Edinburgh, Easter Bush Veterinary Centre, Roslin, Midlothian, Scotland, UK.

Sclerosing panencephalopathies have not been recorded in domestic animals to the authors' knowledge. In humans, two diseases that result in sclerosing encephalitis include; subacute sclerosing panencephalitis (SSPE), a rare but serious progressive degenerative disease of the central nervous system caused by a persistent measles virus infection and progressive multifocal leucoencephalopathy (PML), a rare demyelinating disease of the central nervous system caused by a neurotropic papovavirus.

We describe a case of progressive neurological disease in a horse, the pathological findings of which were a novel diffuse sclerosing panencephalalitis.

A 12 year old cob gelding was presented for investigation of an acute onset of blindness of four weeks' duration. Ophthalmoscopic examination failed to detect any abnormalities. Neurological examination revealed intact direct and consensual pupillary light responses. There was a poor right-sided menace response and a negligible left-sided menace response. The horse had a hypermetric (bilaterally 1+), plodding forelimb gait but no other neurological abnormalities were evident. A re-examination eight weeks later revealed additional neurological signs including; ptosis and drooping of the ear and lower lip on the left, ptyalism and inability to completely close the jaws at rest, protrusion of the tongue between the incisors despite good tongue tone, decreased facial sensation and cervico-facial responses and an intermittent left head tilt. The gait was hypermetric and paretic and intermittent episodes of stupor were seen. The horse was euthanased sixteen weeks following intitial presentation. Gross post mortem revealed focal areas of malacia (2–10 mm diameter) and diffuse congestion of the grey and white matter. Multiple petechial haemorrhages and pale tan 1–10 mm firm foci were present within all cut surfaces of the corona radiata. Histopathologically a non-suppurative meningitis was present and lymphocytic perivascular infiltration with prominent astrocytosis and collagen deposition (sclerosis) and asymmetric, patchy myelin loss were widespread in the cerebral, cerebellar and thalamic white matter. The histopathological findings were consistent with a novel, active, progressive panencephalitis with sclerosis and microcavitation. Although some clinical and pathological similarities exist with both SSPE and PML in humans evidence for an association with a paramyxo or papova or any other virus is lacking in this case. An extensively diffuse astrocytoma, as reported in humans was also considered as a differential diagnosis, however to the authors' knowledge only localised astrocytomas affecting the optic disc have been reported previously in the CNS of horses.

ABSTRACT #3

A CASE SERIES OF OTITIS MEDIA/INTERNA IN DAIRY CALVES: CLINICAL ASPECTS, CT IMAGING AND SURGICAL OSTECTOMY. Jérôme Van Biervliet, Gillian A. Perkins, Brett Woodie, Alessandra Pelligrini-Massini, Thomas J. Divers, Alexander de Lahunta. Cornell University Hospital for Animals, Ithaca, NY.

We observed difficult mastication, spontaneous regurgitation, dysphagia and obtundation, in addition to facial and vestibulochochlear nerve dysfunction in dairy calves. Otitis media/interna commonly complicates bacterial bronchopneumonia, especially caused by *Mycoplasma spp*.

Bacterial meningitis, as a local extension of petrous temperal osteomyelitis, was thought to cause obtundation. In 2 cases, CSF cell count was abnormal in the atlantooccipital CSF and normal in the lumbosacral CSF, indicating that meningeal inflammation was localized, that there was dilution of cells or progression of the meningitis over time. Endoscopic examinations revealed a general loss of pharyngeal tone, dorsal displacement of the soft palate and esophageal hypomotility and hypotonicity, leading to passive regurgitation of forestomach content. This suggests a lesion involving CN IX and/or X. These may be affected by meningitis, as both travel through the subarachnoid space and leave the cranial vault through the jugular foramen in close proximity to the internal acoustic meatus. Another hypothesis is that CN IX and X are affected as they pass over the medial, resp. caudal, outer surface of the tympanic bullae. Pain elicited by jaw movement was thought to cause difficult prehension and mastication, as the inflamed bullae are close proximity to the temporo-mandibular joint and there was no evidence for a CN V deficit.

CT imaging was used to obtain detailed information in these calves. Although oblique radiographs have been reported to be helpful, CT evaluation is likely more sensitive than conventional radiography, because the bony partitions in the bovine tympanic bulla decrease the contrast between normal and fluid filled bullae. Our CT findings corresponded well to the reported postmortem findings, which include fluid or exudate accumulation in enlarged tympanic bullae, mucosal thickening as a result of proliferation of a fibrous inflammatory tissue, osteolysis of the septa and osteomyelitis of the petrous temporal bone. Wall thickness should be interpreted with caution, as it may appear artifactually thicker in fluid-filled bullae. Contrast enhancement is expected due to the degree of lysis.

Otitis media/interna in calves has a guarded prognosis (estimated mortality of 50%). The disease is often detected in late stages when infection is already firmly established and involves surrounding bone. Long-term intravenous administration of antimicrobials aimed at Mycoplasma spp. and other respiratory pathogens may be required. Poor response to medical therapy may be due to poor antibiotic penetration or antimicrobial resistance, as many strains of M. bovis show in vitro resistance. In one of our refractory cases, surgical bulla ostectomy, curettage and drainage resulted in rapid clinical improvement and long-term survival. Weight loss is prominent in these cases. Pan-feeding of milk from the ground may avoid significant aspiration in calves with partial dysphagia, but partial parenteral nutrition may be of great value, especially in inappetant calves.

ABSTRACT #4

ACQUIRED CERVICAL SCOLIOSIS IN 6 HORSES ASSOCIATED WITH DORSAL GREY COLUMN CHRONIC MYELITIS. Jérôme Van Biervliet*, A de Lahunta*, D. Ennulat', M. Oglesbee#, B. Summers*. *College of Veterinary Medicine, Cornell University, Ithaca, NY; #College of Veterinary Medicine, Ohio State University, Columbus, Ohio; `College of Veterinary Medicine, University of Pennsylvania, Kenneth Square, Pennsylvania.

A syndrome of mild-to-severe cervical scoliosis (see figure), mild spastic hemiparesis and ataxia was studied in six horses of both sexes and various breeds, presenting between 5 months and 3 years of age. Clinical signs developed over approximately 1–3 weeks, resulting in permanent, pronounced scoliosis.

Consistent microscopic findings at necropsy were a continuous, unilateral, chronic myelitis centered on one dorsal gray column of the cervical and cranial-to-caudal thoracic spinal cord. This lesion occurred on the convex side of the deformity. Some animals had secondary damage to the articular facets in the midcervical region. The topography and character of the spinal cord lesion were most compatible with a parasite migration, and in one case, an intralesional nematode was identified, presumed to be *Parelaphostrongylus tenuis*. *P. tenuis* is a common parasite of white-tail deer (*Odocoleus virginianus*) that commonly infects small ruminants and camelids, in which the parasite causes extensive lesions.

The development of scoliosis was thought to relate to a sensory denervation of paraspinal muscles and ligaments of the neck, caused by the loss of their general proprioceptive afferents from neuromuscular spindles that synapse in the dorsal gray column. This pathophysiology has been elaborated in humans and experimental animals. We have seen scoliosis of neurogenic origin associated with this pathophysiology in other animal species, such as dogs with syringomyelia secondary to Arnold-Chiari malformations or a calf with post-anesthetic myelomalacia (unpublished observations). The loss of the general somatic afferent nociceptive neurons resulted in an ipsilateral hypalgesia while the ipsilateral mild spastic hemiparesis and ataxia reflected a lesion in the adjacent dorsal and lateral funiculi.

We speculate that spinal nerves may serve as route of access to the dorsal gray column and that selective parasite migration in the dorsal gray columns may reflect a tropism for synaptic neurotransmitters released there.

ABSTRACT #5

RECORDING OF EVOKED OTO-ACOUSTIC EMISSIONS IN CATS.

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Evoked oto-acoustic emissions (OAE) are acoustic signals generated by the outer hair cells in response to an acoustic stimulus and mechanically transmitted via the middle-ear and eardrum to the external auditory canal, where, provided the middle-ear is in proper working order, the responses can be recorded. Transient oto-acoustic emissions (TEOAE) are elicited by wide-band clicks and depend on intact hair cell function over a wide range of frequencies. Distortion-product otoacoustic emissions (DPOAE) are elicited by two stimulus tones of differing frequencies which generate a so-colled distortion effect in a third frequency which the microphone picks up. Any malfunction of the cochlea limits signal-feedback or renders such in-conclusive therefore facilitating frequency-specific testing. In human medicine, this procedure is followed to make an objective frequency-specific examination of the functional integrity of the cochlea. In veterinary medicine, the same method could complement the established concept of brainstem auditory-evoked response testing for evaluation of hearing.

The purpose of this study was to examine whether measurement of evoked oto-acoustic emissions could be applied to veterinary practice.

73 ears from 51 randomly selected, anaesthetized cats were examined otoscopically and tested for the presence of TEOAE and DPOAE. OAE were recorded with standard human medical equipment (Hortmann) with the cats under general anesthesia. For TEOAE a repetitive series of wide-band clicks with a frequency spectrum of 0.5 to 5 kHz were used. DPOAE were measured over nine different frequencies ranging between 1 and 6 kHz.

Results showed that OAE testing in cats required shorter latency settings than in humans. TEOAE and DPOAE were recorded from 88 % and 92 % of normal cats' ears, respectively. TEOAE were absent and DPOAE were recorded in only 8 % of the ears of six cats with either severe otitis externa or deafness suspected by the owners. TEOAE and DPOAE could not be recorded from the ears of two cats examined shortly after being euthanatized.

This study established that it is feasible to measure evoked oto-acoustic emissions under veterinary practice conditions.