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Abstract #1

THE IMPACT OF IDIOPATHIC EPILEPSY AND ITS MANAGEMENT ON THE DOG'S QUALITY OF LIFE AND THE OWNER'S LIFESTYLE. *Y-P Chang* and T.J. Anderson, Department of Veterinary Clinical Studies, Institute of Comparative Medicine, University of Glasgow Veterinary School, Glasgow, Scotland.

When treating a dog with idiopathic epilepsy, seizure frequency has traditionally been considered a key outcome assessment tool. In the human medical literature, seizure severity and quality of life measurements in epileptic patients are also considered important in assessing outcome of management. There is a paucity of veterinary literature on the effects of other aspects of seizure management on owners and the relationship with their pets.

A retrospective questionnaire study was carried out to explore the impact of idiopathic epilepsy and its management on the dog's quality of life and the owner's lifestyle. Dogs referred to the University of Glasgow Veterinary School between 1999 and 2001 for evaluation of recurrent seizures, diagnosed with idiopathic epilepsy and treated with antiepileptic drug therapy were included in this study. Question types included open-ended questions, close-ended questions with ordered answer choices, partially close-ended questions and questions with the visual analogue scale (VAS). The VAS was applied as a 100-point measurement in this study. Patients were divided into two groups on the basis of perceived response to therapy. The Wilcoxon rank sum test was applied to analyse whether adequate seizure control would influence the impact of caring for an epileptic dog on the owner's lifestyle.

Twenty-nine dogs were included in the study. An 86% response rate was obtained. For 52% of dogs, daily activity was perceived to have decreased following the onset of seizures. Two-thirds of owners indicated the interaction between the dog and family members remained unaltered. Half of owners considered that the general quality of life in their dogs was decreased. Adverse effects of antiepileptic drugs, inadequate control of seizures and behavioural changes were given as reasons for this deterioration. The majority of owners did not consider the administration of medication as a problem. Half of owners strongly disagreed with the statement 'regular veterinary examination and blood sampling to monitor serum drug concentration is a nuisance'. One-third of owners with work commitments and two-thirds of owners without work commitments considered caring for an epileptic dog had not caused them conflicts with their work/day-to-day activity. Forty percent of owners felt caring for an epileptic dog did not affect their free time. On these two issues, no significant difference of the responses was found between dogs with adequate seizure frequency and dogs with inadequate seizure frequency.

In conclusion, given the positive attitudes expressed by the majority of owners, it is suggested that regular veterinary examination and serum drug concentration monitoring should be encouraged to achieve optimal seizure management. However, reducing the seizure frequency dose not necessarily decrease the impact of caring for an epileptic dog on the owner's lifestyle.

Abstract #2

MRI FINDINGS IN 7 DOGS WITH CONFIRMED GME. *G.B. Cherubini*, T. J. Anderson, C. Rusbridge, P. Mantis and R. Cappello. The Queen Mother Hospital for Animals, The Royal Veterinary College, London UK.

The purpose of this multicentric retrospective study was to determine the MRI findings and characteristics of GME in dogs. MRI was performed in 7 dogs with histopathological confirmed GME. In 6 dogs the GME neuroanatomical location was in the brain, in 1 dog was in the spinal cord. A single lesion was present in 3 dogs and multiple lesions were found in 4 dogs.

The following data were recorded: breed, gender, age, neurological signs and CSF analysis. Magnetic resonance images on: T1WI, T1WI post gad, T2WI, FLAIR, Gradient Echo (GE) and Proton Density (PD), were assessed. MR images were analysed looking for presence of lesion, neuroanatomical localisation, parenchyma distribution, shape, MRI intensity on T1WI, T2WI, FLAIR, GE and PD images, mass effect, presence of brain or cerebellum herniation, hydrocephalus, gadolinium enhancement, perilesional edema.

This first consistent study evaluating the MRI features in histological confirmed GME, indicates that GME has certain MRI features: multiple lesions, hyperintensity in T2WI and FLAIR, absent of herniation, tendency to be hypointense in T1WI primary macroscopically involvement of the grey matter that, thought not specific for GME, combined with the history, neurological signs and CSF analysis are helpful to increase the specificity of presumptive diagnosis of GME.

Abstract #3

RABIES: A TERRIBLE DISEASE THAT NEED NOT BE TOLERATED. Sarah Cleaveland, Centre for Tropical Veterinary Medicine, Royal (Dick) School of Veterinary Studies, University of Edinburgh, Easter Bush, Roslin, Midlothian, U.K. EH25 9RG.

Rabies is often considered an insignificant disease, but the burden of the disease is much higher than generally recognised. In Africa, the true number of human rabies deaths is estimated to be more than 100 times the number officially reported, with the burden of disease falling most heavily on children. Human rabies is totally preventable and every human death should be seen as a failure of the medical and veterinary systems. In addition to human mortality, rabies has several additional impacts; bite injuries from rabid animals can cause terrible injuries and have considerable psychological impacts; the disease exerts a high economic burden on public health and veterinary budgets due to the high costs of prevention; rabies poses an extinction threat to several endangered wild carnivore populations. This presentation discusses the causes underlying the growing problem of rabies in Africa. Options for controlling rabies are reviewed and the feasibility of control methods discussed, in terms of preventing human deaths and the local extinction of endangered wildlife population.

Abstract #4

SCRAPIE SURVEILLANCE IN ITALIAN SHEEP: A CLINICAL APPROACH TO IMPROVE THE RATE OF SUSPECTS. *A. d'Angelo*, C. Maurella, C. Bona, M. Caramelli, M.E. Careddu, A. Jaggy, G. Ru. Department of Animal Pathology University of Turin, Italy.

Since a reliable ante mortem diagnostic test for the detection of TSE is not available, the clinical diagnosis of scrapie suspects is based upon the neurological signs observed. As many other neurological diseases show the same clinical signs it is very difficult to identify an affected sheep.

The purposes of this work was: a) to identify neurological signs consistent with scrapie by visiting sheep from 19 Italian flocks in which the disease had previously been confirmed; b) to standardise a clinical protocol that is easy to handle and useful in passive surveillance of scrapie in sheep.

179 sheep were examined by applying a clinical standardized protocol. In each flock sheep were chosen according two following criteria: the availability of the historic data, and the identification clinical signs by means of flock observation. In addition, a group of sheep was chosen through convenience sampling. All sheep were submitted to post mortem rapid test analysis for scrapie. The collected data were analysed using statistical software programme (EpiInfo 6.04). Relative risks were used to identify the association between clinical signs and confirmed scrapie; moreover, predictive values were computed for single and combined clinical signs.

26 sheep were confirmed as being affected by scrapie. Most recurrent signs in scrapie positive animals were those reported in literature: conscious proprioceptive deficits of the pelvic limbs (61.5%), positive nibble reflex (53.8%), ataxia (50%) sialorrhea (34.6%) and altered mental status (30.8%). Some of these clinical signs showed high negative predictive values. Animals with a history of ''typical'' symptoms showed a larger proportion of clinical signs when compared with those chosen randomly.

Abstract #5

CLINICAL AND NEUROPATHOLOGICAL FINDINGS OF EQUINE MOTOR NEURON DISEASE IN TWO ITALIAN HORSES. Gualtiero Gandini, Elisa Brini, Claudia Salvadori, Floriana Gernone and Carlo Cantile. Department of Veterinary Clinical Sciences, Faculty of Veterinary Medicine—University of Bologna, Italy.

Two Italian saddle horses, a 10 year old gelding and a 14 year old mare, were admitted with a complain of progressive weight loss and weakness, resulting in excessive lying down on the floor of their box. Appetite was good and micturition and defecation normal. The general physical examination was normal.

The horses underwent a complete neurological examination, cell blood count (CBC), and biochemical profile. Vitamin E plasma levels were not measured because of previous supplementation. Muscle and nerve biopsies were taken from the left sacrocaudalis dorsalis medialis muscle and the ventral branch of the left spinal accessory nerve, respectively. Immediately after euthanasia for humane reasons, cerebrospinal fluid (CSF) was collected and examined and both horses had a neuropathologic examination of the brain and spinal cord. Samples of the left radial and peroneal nerves were also examined in one horse and an electromyographic (EMG) examination was performed under general anaesthesia on the second horse.

Abnormal neurological examination findings were severe generalised muscle atrophy with marked muscle tremors of the antigravitational muscles when the horses were forced to stand, and frequent shifting of the weight from limb to limb. One horse had an abnormal low carriage of the head. Gait was characterised by moderate tetraparesis with hypometria of the four limbs and exercise intolerance. There was no evidence of ataxia. BC, biochemical profile and CSF examination were normal. The EMG showed fibrillation potential in the paravertebral muscles.

Pathological findings were similar in both cases: scattered neuronal degeneration and loss in the ventral horns of the spinal cord and in the caudal brain stem. Affected neurons were swollen and chromatolytic, and occasionally showed intracytoplasmic eosinophilic inclusions. Some degenerating motor neurons were shrunken, sometimes with fragmenting nucleus. Small microglial nodules were also observed at the sites of neuronal loss. PAS-positive and Luxol fast blue-positive ceroidal lipopigment granules were observed in the cytoplasm of many degenerating neurons and some endothelial cells in the affected areas. Occasional perivascular accumulation of macrophages laden with residual ceroidal pigment was also noticed. Examination of spinal accessory, peroneal and radial nerves showed loss of large diameter fibres and axonal degeneration characterised by axonal swelling, myelin ovoids formation and phagocytosis by macrophages. Scattered angular myofibres and occasional hypertrophic fibres were observed in the sacrocaudalis dorsalis medialis muscle.

Abstract #6

TOPOGRAPHICAL MAGNETIC RESONANCE FINDINGS, RESULTS OF DIAGNOSTIC INVESTIGATION AND OUTCOME OF SUSPECTED AND CONFIRMED BRAIN INFARCTION IN 38 DOGS. *L.S. Garosi*, J.F. McConnell, S.R. Platt, V. Adams, G Barone, J.C. Baron[3], A. de Lahunta and S.J. Schatzberg—Animal Health Trust, Newmarket, Suffolk, England.

The purpose of this multicenter retrospective study was to determine the type and location of brain infarction, prevalence and type of associated medical conditions, prevalence of systemic hypertension and outcome of a population of dogs with suspected or confirmed brain infarct. Medical records of 38 dogs that presented for evaluation of acute onset, non-progressive (after 24 hours), intracranial neurological signs were reviewed [1999–2003]. All dogs had a lesion on magnetic resonance imaging (MRI) compatible with brain infarction (31/38) or post-mortem confirmation (7/38). Results of complete haematology, biochemistry, thyroid, coagulation and adrenal profile, urinalysis, thoracic and abdominal imaging and cerebrospinal fluid (CSF) analysis were available in 33/38 dogs. Arterial blood pressure was evaluated in 28/38 dogs. The topography of the brain infarcts were as follows: 10/38 telencephalic, 8/38 thalamic/midbrain, 17/38 cerebellar and 3/38 multifocal (thalamic and medulla). Telencephalic infarcts occurred within the territory of the middle cerebral (4/6), rostral cerebral arteries (2/6) and striate artery (4/11). Thalamic/midbrain infarcts occurred within the territory of perforating arteries of caudal thalamus and rostral brainstem (8/8). Cerebellar infarcts were all within the territory of the rostral cerebellar artery. The appearance of the telencephalic and cerebellar infarcts resembled territorial infarcts seen in humans, while the thalamic/midbrain infarcts resembled lacunar infarcts. All infarcts were classified as non-haemorrhagic and contrast-enhancement was observed in only 3/38 dogs, all of which were imaged more than 7 days after the onset of neurological signs. An associated medical condition was detected in 18/33 dogs. Chronic kidney disease (8/33) and hyperadrenocorticism (6/33) were the two most commonly encountered conditions. There were no significant associations between region or type of infarct and patient age and sex, occurrence of hypertension (systolic >160 mmHg and/or diastolic >120 mmHg), presence of CSF abnormalities or presence or absence of an associated medical condition. Ten of the 38 dogs were euthanized due to the lack of improvement of their neurological status (3/10) or severity of their associated medical condition (7/10). There was no association between type or region of infarct and outcome. Dogs with an associated medical condition also were significantly more likely to suffer from recurrent neurological signs due to subsequent infarcts (OR = 8.9, 95% CI = 1.0–84, $P = 0.05$).

Abstract #7

OVINE COENUROSIS ('GID'). *C.N. Hahn, G.C. Skerritt** and I.G. Mayhew. The University of Edinburgh, Royal (Dick) School of Veterinary Studies, EH25 9RG UK and the *ChesterGates Referral Hospital, Chester CH1 6LT UK.

The cestode *Taenia multiceps* is found in the small intestine of wild and domestic canidae. When the definitive host eats tissue containing coenurid cysts, protoscolices within the cyst attach to the small intestinal wall forming mature tape worms. Gravid proglottids containing the eggs detach from the end of the worm and pass out in the feces. Following ingestion of grass contaminated with eggs, hexacanth embryos are released and burrow through the intestinal wall to each a number of organs via the circulatory system. The larval tapeworms occur in large groups within a thin walled cyst known as a coenurus and have been recovered from subcutaneous or intermuscular tissues of hares, rabbits and squirrels. In sheep the embryos will only develop further in the CNS, where the larval stage is called coenurus cerebralis and the disease is known as 'gid'. Multiple cyst can occur in one sheep or goat and it takes several months for the cyst to have grown to a large enough size to cause clinical signs. Disease is generally slowly progressive, although acute coenurosis can occur in lambs when large numbers of embryos migrate simultaneously through brain parenchyma producing an acute encephalitis. Chronic disease in adult sheep presents as an insidiously progressive focal lesion of the brain, most consistently in one cerebral hemisphere. Obtundation, vestibular and cerebellar signs are most common. Softening of the frontal bone, as a consequence of a generalized increase in intracerebral pressure, may be palpable and may be a guide for localisation with a view to trephining the skull and removal of the cyst by suction. Diagnosis of gid may be made upon necropsy, or by using advanced imaging. Magnetic resonance imaging can reveal dramatic hypointense cyst on T1 weighted images and hyperintense cysts on T2 weighted images. In areas where there are few wild canids, coenurosis should be controlled by regular anthelmintic treatment of farm dogs an correct disposal of sheep carcasses.

Abstract #8

SENSORY NEUROPATHY IN GOLDEN RETRIEVERS. K. Hultin Jäderlund, E. Örvind, C.N. Hahn. Swedish University of Agricultural Sciences, Faculty of Veterinary Medicine, S-750 07 Uppsala, Sweden. [2]Neuromuscular Disease Laboratory, Royal (Dick) School of Veterinary Studies University of Edinburgh, Midlothian EH25 9RG, UK.

Peripheral neuropathies resulting in sensory signs only are rare in dogs. Breed-related sensory neuropathies have been reported in long-haired dachshunds, pointers and border collies. Clinical signs in these dogs have included self-mutilation. number of related golden retrievers with a sensory neuropathy have been seen in Sweden over the last few years. There have been not previously described and clinically are not similar to the other breed-related sensory neuropathies.

Onset of signs are been insidious and first noticed at an age of about 4–8 months. Both males and females have been affected. The clinical signs are slowly progressive over several months, but the progression seems to slow down after a while. Some dogs have again worsened at about 18 months of age, with concomitant incontinence at that stage. The predominant clinical sign is ataxia, most evident in the pelvic limbs where claws have been worn down. The dogs avoid walking on slippery floors and on stairs and have a bunny-hopping gait. Male dogs have problems lifting their hind limb while urinating. The positioning of the thoracic limbs has been somewhat abnormal, resembling a dog with weak ligamentous apparatus in the palmar aspect of the carpal joints. Owners have not reported any signs of pain related to this movement disorder.

Neurological examination have revealed reduced proprioceptive placing reactions and generally depressed spinal reflexes. The patellar reflexes have been completely lost or severely decreased. Cranial nerves have not been affected.

Sections of brain, spinal cord, spinal nerves and muscle were examined. Routine histopathological changes were surprisingly mild and consisted of Schwann cell proliferation in the spinal cord dorsal roots, which may be due to replacement of axons with astrocyte processes. Rare macrophages containing myelin debris and spheroids were evident in spinal cord dorsal funiculi. Peripheral nerves showed very occasional digestion chambers, however semi-thin sections of the same nerves subjectively suggested a significant loss of large caliber axons. Sections of muscle showed single fibre atrophy but no group atrophy, excessive fibre size variation and rare single anguloid fibres which may indicate low grade involvement of some motor axons.

Results from neurological examination together with electrophysiology and histopathology findings point to a sensory neuropathy. A familiar occurrence is indicative of an inheritance for the sensory neuropathy described.

Abstract #9

CEREBELLAR CORTICAL DEGENERATION IN THREE LAGOTTO ROMAGNOLO DOGS. *Jokinen* T.S., Rusbridge C. Viitmaa R, Syrjä P, de Lahunta A, Snellman M., Cizinauskas S. Department of Clinical Veterinary Sciences, P.O. Box 57 (Hameentie 57), 00014 University of Helsinki, Finland.

Abiotrophy means premature tissue degeneration in absence of an exogenous cause. Cerebellar cortical abiotrophy has been reported in many dog breeds with differences in the age of onset and the progression of signs. Usually the most obvious finding histologically has been depletion of Purkinje cells. This study describes three cases of cerebellar cortical degeneration in Lagotto Romagnolo Dog.

All three dogs (two females and one male) had gait abnormalities since seven they were 13 weeks old but otherwise they were bright and alert. The physical examination was normal. The neurological examination showed generalized ataxia, hypermetria, decreased proprioception in all four limbs and bilaterally decreased menace reaction. The disease was localized to the cerebellum in all three patients. The list of differential diagnoses included a cerebellar malformation (hypoplasia), abiotrophy, inflammatory/infectious diseases, storage diseases and neoplasia.

Complete blood cell count, blood biochemistry profile and cerebrospinal fluid examination were normal in one of the patients. Magnetic resonance imaging revealed decreased size of the cerebellum.

All three dogs were euthanized because of progressive neurological signs. The only pathological changes were found in the cerebellum in all three dogs. One of the dogs had decreased size of the cerebellum (7% of the total brain weight). One dog had clear depletion of cerebellar granular cell layer and the other two dogs had degenerative changes in Purkinje cells.

Cerebellar abiotrophy has not been reported in this dog breed previously. Cerebellar cortical abiotrophy was diagnosed in three Lagotto Romagnolo Dogs based on the history, histopathological findings and in the absence of an exogenous cause to these degenerative changes. An early onset of clinical signs was observed in all three dogs. The correlation between the histopathological changes and clinical signs appears not to be complete. Primary depletion of the granular cell layer is a more rarely encountered form of cerebellar cortical degeneration but was noticed in one of the dogs.

Abstract #10

SURGICAL TREATMENT OF COENUROSIS IN SHEEP: A RETROSPECTIVE STUDY AND FOLLOW UP OF 1,130 CASES (1996–2004). A.Th. Komnenou, S. Argyroudis, *Z.S. Polizopoulou*, N. Giadinis and A. Dessiris. School of Veterinary Medicine, Aristotle University of Thessaloniki, Thessaloniki, Greece.

Coenurosis, a fatal disease of young sheep caused by the larval stage (coenurus) of *Taenia multiceps*, is a common problem in Greece and a cause for considerable annual losses in breeding livestock, which can reach 80% in heavily infested regions. This report describes the clinical features, the surgical procedure for cyst removal and the follow up in 1,130 cases that were exhibiting signs of neurological dysfunction and were referred to the School of Veterinary Medicine, Aristotle University of Thessaloniki during the years 1996 to 2004. The age of the animals that belonged to a variety of local breeds, ranged from 6 months to 3 years, the majority of them (622–55%) being younger than 1 year. A complete history was taken in all cases with emphasis on general flock management, previous health problems, as well as duration of signs in affected sheep. Neurological examination, using a standard protocol outlined for large animals, aimed in localizing the lesion in the cerebrum, cerebellum, rostral and caudal brainstem or spinal cord. Main differential diagnoses included listeriosis, louping ill, visna, scrapie, cerebrocortical necrosis or brain abscess and were ruled out based on epidemiology, clinical and clinicopathological findings and lack of response to symptomatic treatment. Lesion localization was one of the cerebral hemispheres in 938 cases (83%), the cerebellum in 6 (68%) and the brainstem in 2 (23%). In the remaining 101 cases it was impossible to locate the affected site clinically. Election of the surgical site was done according to the findings of neurological examination. After exposure of the cyst, its contents were aspirated using a plastic catheter with an attached syringe, followed by gentle removal of the cyst wall with a hemostatic forceps. Postoperatively, dexamethasone and broad spectrum antibiotics were administered for 2 and 7 days, respectively. Surgery was successful in 994 cases (88%), with cysts found in the cerebrum in 845 (85%), the cerebellum in 50 (5%) and the brainstem in 20 (2%) animals. Seventy-nine (8%) sheep proved to have multiple cysts, since their condition improved temporarily after the removal of the most prominent cyst, to deteriorate subsequently. In those cases the existence of multiple lesions was noted during necropsy. In the remaining 136 sheep (8%) it was impossible to locate parasitic cysts during surgery; at postmortem examination those were found in the cerebellum (103–76%) and the brainstem (33–24%), respectively. Intraoperative uncontrolled bleeding after cyst removal accounted for the loss of 11 (1%) sheep. From the 983 animals that finally were able to return in their flocks, 660 (73%) showed immediate and 235 (26%) gradual improvement, while 9 (1%) developed bacterial meningoencephalitis and died. In this study, surgical treatment of coenurosis proved to be effective and with a relatively high success rate, since finally 965 sheep (85%) could return in their flocks with minimal or no residual neurological deficits.

Abstract #11

ASSESSMENT OF HEART RATE VARIABILITY ANALYSIS IN THE DIAGNOSIS OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES IN SHEEP. *T Konold*, R. Sayers and S. Bellworthy. Neuropathology, Veterinary Laboratories Agency Weybridge, UK.

The resting heart rate varies in response to respiration via cardioexcitatory (sympathetic) and cardio-inhibitory (parasympathetic) neural influences. Measurement of heart rate variability (HRV) is a useful method for assessing sympathetic and parasympathetic autonomic effects on the heart. Diseases involving the brainstem in humans can lead to autonomic dysfunction resulting in abnormalities of HRV. Transmissible spongiform encephalopathies in ruminants are characterised by neuronal vacuolation and accumulation of proteinase-resistant prion protein (PrPres) in the brainstem. The parasympathetic nucleus of the vagus nerve provides efferent fibres to the heart and is generally affected. An increased parasympathetic tone resulting in bradycardia has already been demonstrated in cattle suffering from bovine spongiform encephalopathy (BSE).

This study tested the hypothesis that transmissible spongiform encephalopathies (TSE) in sheep result in abnormalities of heart rate variability that are distinguishable from healthy sheep. Electrocardiograms (ECGs) were recorded for 5 min in 7 adult sheep affected by natural scrapie and 6 adult sheep orally inoculated with the BSE agent with a postmortem diagnosis of TSE. All had clinical signs of the disease. These were compared with ECGs from 11 clinically healthy adult sheep born on farms in the UK known to be free of scrapie and which had no postmortem TSE changes (control group).

Heart rate, low frequency power (LF, 0–0.16 Hz), high frequency power (HF, 0.16–1.2 Hz) and LF/HF ratio were calculated by Fourier transform on tachygrams of instantaneous frequency (frequency domain analysis).

Statistically significant differences among the three groups were found only for the heart rate means ($P = 0.037$, ANOVA on log transformed values) but not for the other variables. Further examination of the heart rate differences using t-tests with separate variance estimates for each group revealed that the heart rate mean of the control group was significantly lower than that for BSE ($p = 0.036$) and the difference from the scrapie group was nearly significant ($P = 0.068$).

Contrary to BSE in cattle, bradycardia was not a feature of TSEs in sheep. Frequency domain analysis of ECGs of 5 min duration was not useful to distinguish diseased sheep from normal sheep in this study.

Abstract #12

EVALUATION OF TELOMERASE STAINING IN CANINE BRAIN TISSUE AND ASSOCIATION WITH MALIGNANCY IN BRAIN TUMOURS. S. *Long*, C. Botteron, G. Rutteman, S.Platt, N. Olby, D.J, Argyle and L. Nasir. Institute for Comparative Medicine, University of Glasgow Veterinary School, Glasgow, UK.

Due to the 'end-replication problem', it is impossible for linear chromosomes to be fully replicated during mitotic division. As a result, the terminal portion of chromosomes, known as telomeres, shorten by 50–200 bp with every cell division. Accumulated telomere shortening ultimately triggers cellular senescence and is therefore thought to represent an important aging mechanism for cells. Telomerase is a cellular reverse transcriptase which maintains telomere length, and its presence is associated with the ability of cells to bypass senescence and thus achieve immortalisation. Telomerase activity is present in fetal tissue and stem cells but is inactivated in the vast majority of somatic cells. Additionally, telomerase activity has been detected in up to 90% of human brain tumours, making it a useful diagnostic tool. The enzyme is composed of two principal subunits: TR, or TERC (TElomerase RNA Component), an intrinsic RNA component, and TERT (TElomerase Reverse Transcriptase), a reverse transcriptase catalytic subunit. TERT is only present in telomerase-positive cells, and its introduction into telomerase negative fibroblasts is sufficient to convey telomerase activity, suggesting that TERT is essential for telomerase activation.

Our group has shown the presence of telomerase activity in canine tumours, with little or no activity in most somatic tissues. To assess telomerase activity in canine brain tumours, immunohistochemical staining using a human anti-TERT antibody was evaluated in normal canine brain and a panel of 54 brain tumours. The correlation between TERT staining and malignancy, as defined by conventional histological grading schemes and proliferation index, was examined. In order to evaluate each stain, positive controls, telomerase-positive canine cell lines CMT7 and D17 and canine testicular tissue samples were examined. Normal canine fibroblasts and primary antibody omission provided negative controls. Overall, the presence of TERT staining was associated with malignancy in the tumours examined, with 11% of benign tumours possessing telomerase ac-

tivity, compared with 67% of malignant tumours. A trend for telomerase activity to be associated with malignancy was also found in each tumour subtype, although this was not statistically significant. Specific TERT staining was also found in particular cell types in the normal brain, particularly neuronal cell bodies. These results suggest that telomerase activity, as assessed by TERT staining, may provide a useful prognostic indicator in the evaluation of canine brain tumours. However, the role of TERT staining in normal brain remains to be elucidated.

Abstract #13

AN APPRAISAL OF GREAT DANE MYOPATHY. *A. Luján Feliu-Pascual*, M. Targett, G.D. Shelton, K.C. Chang, S.N. Long, E. Comerford, T.J. Anderson. Institute of Comparative Medicine, University of Glasgow Veterinary School, Scotland.

Centrally located, structurally distinct disorganised accumulations of cytoskeletal elements in canine skeletal muscle have been identified in two case reports concerning a myopathy in growing Great Danes. The descriptive term Central Core Myopathy (CCM) has been used to refer to this unusual disease. However, the description of the ultrastructure of the affected muscles differs between the two reported cases. Anecdotal evidence suggests that the disease occurs in the British Great Dane population with the most recent cases confirmed earlier this year.

Clinical, histopathological and pedigree information from 21 young Great Danes with CCM was reviewed. All cases were presented with a progressive exercise intolerance, muscle tremor, progressive muscle atrophy, weakness and gait abnormality. The majority of cases were less than 10 months of age and fawn colour. Both male and females were affected in similar proportions. Examination revealed generalised muscle atrophy, particularly of temporal, caudal thigh, quadriceps and shoulder muscles; stiff, short-stride gait, generalised body tremor, exaggerated during walking; and collapse after a few metres. Standing posture was characteristic with pelvic limbs tucked under the abdomen and extended tail carriage. Myalgia was not encountered in any case. Increases in serum creatine kinase (CK) at presentation up to 26-fold (range 292–3925IU/L) with variable level during the course of the disease were noted. Serum AST and ALT are also elevated in most cases. Electromyography performed in 12 cases was consistent with myopathy/neuropathy. Pedigree information was available for 20 cases, all linked to a common ancestor. Histopathologically confirmed, clinically affected but not confirmed, and normal siblings were observed in the same litter. Analysis of the family tree was consistent with autosomal recessive inheritance; however, autosomal incomplete dominance or polygenic could not be ruled out. Muscle biopsies show distinct pale central areas in myofibres using haematoxylin/eosin stainings. With periodic acid-Schiff (PAS), the cores are dark suggesting accumulation of carbohydrates. There is myofibre size variation and the cores tend to be located in type I fibres. Succinate dehydrogenase and cytochrome oxidase staining suggests high oxidative enzymatic activity in the cores. Electron microscopy (EM) shows disruption of the normal myofibre architecture, accumulation of glycogen and mitochondria within the cores.

These findings are in contrast with Central Core Disease (CCD) in humans, where the cores are devoid of mitochondria and composed of disrupted myofibrils. The human condition is caused by mutations in the skeletal ryanodine receptor (RYR1) gene. Mutations in this gene are also responsible for malignant hyperthermia in both human and canine individuals. Due to the dissimilarities at immunohistochemical and EM level, RYR1 is unlikely to be a candidate gene for the canine condition. Studies are being carried out to characterise the disease at molecular level.

Abstract #14

WEST NILE VIRUS ENCEPHALOMYELITIS: WHAT TO EXPECT DURING THE COMING EUROPEAN EPIZOOTICS. *R.J MacKay*, College of Veterinary Medicine, University of Florida, Gainesville, FL, USA.

The neuroinvasive flavivirus responsible for WNVE of horses and humans is endemic to Africa, Asia, and Australia and associated with sporadic epizootics in Europe and eastern Mediterranean countries. The virus is propagated through mosquito-bird transmission and periodically spreads to terminal hosts such as horses and humans. Outbreaks of equine neurologic disease were first reported in France in the early 1960s. More than 30 years later, new outbreaks have been reported from Morocco (1996), Italy (1998), France (2000), and Israel (2000). Almost all cases occurred from August through October. In the fall of 1999, West Nile virus suddenly emerged in New York, killing thousands of native and exotic birds and causing deaths in humans and horses. This virus was almost identical to a highly virulent strain isolated in Israel the previous year from domestic geese and migrating storks. In 1999, there were 25 equine cases reported in the US with 9 deaths. Since then, the virus has spread rapidly to the south and west with 14,717 reported cases in horses in 2002 in 40 states. Last year was the first that saw an overall decrease in reported cases in horses (5,181) and humans (2,866), although the virus' range has continued to expand to include all states except Oregon, Hawaii, and Alaska. In 2003, West Nile virus activity also was reported in Canada, Mexico, central America, and the Caribbean. The virus has notably decimated corvid bird (eg, crow, raven, jay) populations in the US, while affecting more than 110 other bird species and numerous other mammals, amphibians, and reptiles. As is the case in Europe, West Nile virus has been isolated from many different culicine and anophelene mosquitoes, with *Culex pipiens* likely important in transmission among birds. There is a suggestion that ''hybrid'' *C. pipiens* in North America are competent to transfer the virus from birds to mammals, whereas European *C. pipiens* tend to feed exclusively on either birds or mammals.

WNVE in horses is a nonsuppurative polioencephalomyelitis affecting particularly the ventral horns of thoracic and lumbar spinal cord segments. Encephalitic lesions are largely restricted to the brainstem. Acute neurologic signs in horses have included limb ataxia and weakness, behavioral hyperresponsiveness or depression, muscle fasciculations, especially around the face, and flaccid muzzle, lips, and tongue. Severity has ranged from very mild (a few twitches around the face) to rapid recumbency and death. Recovery takes from several weeks to several months and is not always complete. Evidence of acute West Nile infection is an IgM ELISA titer $\geq$400. Serum virus neutralization tests are not particularly useful as they can be affected by prior vaccination. Treatment is supportive although the use of interferon α and hyperimmune serum or plasma is under evaluation. A killed vaccine has been available since August 2001 and is apparently highly effective. More recently, a recombinant vaccine has been introduced.

Abstract #15

EQUINE PROTOZOAL MYELOENCEPHALITIS (EPM): UPDATE (EVERYTHING YOU NEED TO KNOW TO PASS BOARDS). *R.J MacKay*, College of Veterinary Medicine, University of Florida, Gainesville, FL, USA.

EPM is endemic among horses in the Americas. Horses are infected when they ingest sporocysts of the causative agent, *Sarcocystis neurona. S. neurona* is propagated in nature by passage between opossums (definitive host) and intermediate hosts such as 9-banded armadillos, striped skunks, raccoons, sea otter, Pacific harbor seals, and domestic cats. The horse is presumed to be a dead-end host for *S. neurona.* A sporadic form of EPM is associated with *Neospora hughesi* infection.

Signs of spinal cord involvement are more common than signs of brain disease. Typically, there is asymmetric or symmetric weakness and ataxia of one to all limbs, sometimes with obvious muscle atrophy. The most common signs of brain disease in horses with EPM are depression, head tilt, and facial paralysis. Without treatment, EPM often progresses to cause recumbency and death after a course of hours to years.

Diagnosis is by demonstration of parasites in H&E or immunostained histologic sections of the CNS. Schizonts, in various stages of maturation, or free merozoites commonly are seen in the cytoplasm of neurons or mononuclear phagocytes of the CNS. In horses with neurologic signs, demonstration of specific antibody in CSF by immunoblot is suggestive of EPM. A positive immunoblot test in serum indicates only exposure to *S neurona* whereas a negative result, in either serum or CSF, tends to exclude EPM.

Two anticoccidial agents are approved by the FDA for EPM treatment: ponazuril (Marquis) and nitazoxanide (Navigator). Each is a dial-by-weight paste formulation that is given for 28 days. An alternative is the combination of pyrimethamine and sulfonamide, given orally for 3–6 months. Toxic effects of sulfa/pyrimethamine include bone marrow suppression, glossitis, and congenital defects. Adverse effects are rare with ponazuril but depression, fever, and colitis may be seen in horses treated with nitazoxanide. With any of these treatments, $>$60% of horses improve clinically, but $<$50% recover completely. Relapses are common in horses that remain positive on immunoblot and rare in those that become negative.

No proven preventative is available. A conditionally-approved vaccine is marketed and its efficacy continues to be evaluated. Most prevention efforts focus on protection of food and water sources and control of *S. neurona* host species (especially opossums).

Abstract #16

HYPOVITAMINOSIS A IN A LION (*PANTHERA LEO*). P.J.J. Mandigers. Veterinary Specialist Centre ''De Wagenrenk'' Keijenbergseweg 18, 6705 BN Wageningen, The Netherlands.

An eight months old male lion was referred with clinical signs of mild ataxia and a possible impaired vision. The lion was kept in Circus together with 8 other large cats. Dietary, breeding and clinical histories of this and all other cats were according to the owner normal. At the time of referral the signs had been present

for a minimum of 2 months. A neurological examination, performed without any restraints, revealed a mild ataxia of both front as well as hind legs. Visus appeared to be normal as were all other cerebral reflexes. However apparent 'looking up' was present. Postural and spinal reflexes could not be evaluated. After a medetomidine-ketamine based premedication, propofol induction the animal was intubated and kept on oxygen-air mixture and approximately 1.5% of isoflurane. A full red blood & white blood cell count, biochemistry, vitamin B1, B6 & B12 analysis revealed no abnormalities. FIP, FIV, FeLV status was negative. Spinal x-rays of the complete vertebral column as well as skull were normal as well. After sending out an email for help to ACVIM-neurology, ECVN diplomats as well as Zoo vets two responses suggested that the case was a 'stargazer' due to a possible vitamin A deficiency. After a third consultation the meat preparation appeared not to be in order. Animals in a lower social hierarchy appeared to eat less vitamin supplements that those in a higher order. After changing the preparation method (enrich each piece properly) clinical signs disappeared within three weeks.

Abstract #17

TETRAPARALYSIS IN AN AFRICAN LION (*PANTHERA LEO*). P.J.J Mandigers. Veterinary Specialist Centre ''De Wagenrenk'' Keijenbergseweg 18, 6705 BN Wageningen, The Netherlands.

A twelve months old female white lion was in the process of being introduced into a (1–4) African lion group. On the first day she was introduced to the leading male and was attacked by the male and another female. The injury the animal was able to lift her head but was recumbent. At that time the animal was isolated, treated with flunixine 1 mg/kg s.i.d. and amoxycillin/clavuculanic-acid 12 mg/3 mg/kg b.i.d. The next day the lion stayed recumbent, was able to lift her head but was not able to drink or eat. At referral the lion had been recumbent for 1.5 days. Because of zoo policy the neurological examination had to be performed at a distance of at least one meter. The animal was vocal, appeared to have good vision and showed no abnormal cerebral reflexes. Postural reflexes could not be evaluated but the spinal reflexes appeared to be exaggerated in both thoracic and pelvic limbs. It was concluded that there was at least a spinal lesion at the level of C1-C6. After a medetomidine (30mg/kg) and ketamine (1 mg/kg) based premedication, the animal was taken into the clinic. The lion was further anesthetized using propofol (based on effect approximately 2 mg/kg) and after intubation maintained on 2 litre of oxygen, 1 litre of nitrogen and approximately 1.5% of isoflurane. The lion was put on lactated ringers solution at a rate of 10 ml/kg per hour. Cervical radiographs revealed a fracture of part of the wing of C1 and a large part of the spinous process of C2. The latter pointed ventrally towards the spinal cord. Despite the bad prognosis it was decided to perform surgery by means of a dorsal approach. A large part of the dorsal cervical musculature had been torn into pieces. The bone fragments appeared to be dislocated towards the spinal cord. The dura mater had been punctured and a fragment appeared to have entered the spinal cord. Although it was possible to remove all fragments and close the surgical area the lion died. It is concluded that the correct neuroanatomic diagnosis had been made. Prognosis of this type of lesion is most likely grave and the type of animal as well as the time between injury and referral worsened the prognosis further.

Abstract #18

HAZARDOUS HEDGES—NEUROPATHOLOGY OF PRIVET POISONING IN TWO HORSES. *K. Matiasek*, W. Ranner, C. Deeg, L. Wieczorek, M. Pumarola and W. Schmahl. Chair of Neuropathology & General Pathology, Institute of Veterinary Pathology, Faculty of Veterinary Medicine, LMU Munich, Germany.

Sporadic reports on privet poisoning in herbivore animals indicate that the consequences of uptake can be either acute to peracute colic, hypotension and renal failure, or severe persistent proprioceptive deficits. Differences in course, symptoms and outcome may be dose-dependent or due to preferences of ingesting berries, leaves or wooden parts of the plant, each of them harbouring a distinct cocktail of toxic agents.

Two female standardbred yearlings could be observed right after spontaneously having ingested leaves and branches of *Ligustrum vulgare*. Acute clinical signs in terms of severe abdominal colic and bradycardia were overcome successfully by immediate emergency care comprising shock prophylaxis, analgesia and elimination therapy. Thereafter, however, both animals developed a severe monophasic and progressive proprioceptive tetra-ataxia with an emphasis on rear limbs. After a period of seven months both horses were euthanized because of poor prognosis.

Histologic and electron microscopic examinations were carried out on CNS, pre- and paravertebral ganglia and peripheral nerves. Most strikingly, histopathology uncovered multiple axonal spheroids of tubulovesicular or mixed type within the long spinal tracts, as well as numerous axosomatic synaptic buttons in vestibular, accessory cuneate and cerebellar roof nuclei. Peripheral nerves were affected mildly by a chronic axonal non-regenerative mixed-fiber polyneuropathy.

According to the pathological findings, the single ingestion of toxic plant products culminated in a marked neuroaxonal dystrophy predominantly affecting the proprioceptive system. Enlargement of preterminal/presynaptic axonal segments is based on an accumulation of irregular axoplasmatic material and suggests a perturbation of the intraaxonal transport as one mechanism of disease. More sophisticated immunohistochemical and molecular analyses will help to specify the compromised branch and fraction of this bidirectional system, and to identify the molecular targets tackled by the privet poison.

ABSTRACT #19

EPISODIC NEUROLOGIC SYNDROMES OF HORSES. I.G Mayhew. Royal (Dick) School of Veterinary Studies, EBVC, Roslin, Midlothian, Scotland.

Truly episodic syndromes wherein the patient is essentially normal between episodes are considered in this paper. Although many of these syndromes have recognised predisposing factors those associated with exercise such as ERS, HYPP, AMG, SHS and Head Shaking will not be covered here.

A seizure, ictus or convulsion can be regarded as abnormal behaviour and is the physical expression of dysrhythmic discharges in forebrain neurons that reach the motor areas and initiate spontaneous, paroxysmal, involuntary movements in any part of or the entire body. The syndrome of recurrent seizures, no matter what the cause, is referred to as epilepsy. In the adult horse seizures more frequently occur in conjunction with other signs of brain disease, even though these may be subtle. Inherited, persistent (true) epilepsy with an adult onset and no active underlying disease process probably has not been demonstrated in horses.

The term sleep disorders probably covers better the spectrum of episodic syndromes wherein the otherwise totally normal patient appears to be in a state of sleep at unusual times or intermittently, fully or partly collapses. This covers the disorders of Narcolepsy, Cataplexy and the more common Idiopathic Hypersomnia.

Fainting or syncope is collapsing because of a non-traumatic, temporary lack of oxygen delivery to the brain most often due to hypotension or cardiac blockade. It probably is non-existent in the horse in the absence of overt heart failure.

Stereotypical behaviour refers to unacceptable activities that are repeated; thus anthropomorphically referred to as vices or obsessive-compulsive disorders. They particularly result from an unsatisfactory environment and the identifiable environmental derangements are referred to as stressors.

Various intermittent and sometimes spectacular attacks of unusual behaviour are referred to as 'panic attacks', 'freeze attacks', and 'startle attacks'. There can be triggering cues involved such as saddling up. Although many of these episodes reflect prominent but physiological responses, there is reason to believe that some of the syndromes represent forms of epilepsy, others of hypersomnia and still others as resulting from a painful focus somewhere in the body because the syndrome is attenuated with the use of anti-inflammatory drugs.

Abstract #20

GRASS SICKNESS. B. McGorum, Royal (Dick) School of Veterinary Studies, University of Edinburgh.

Equine grass sickness (EGS) is a multi-systems neuropathy of equids that is characterised by degeneration of neurons within the autonomic, enteric and somatic nervous systems. The disorder has an extremely high mortality rate and carries serious welfare, emotional and financial implications. Interestingly, an identical disorder occurs in wild and farmed rabbits, hares, dogs and cats. EGS is described as acute, sub-acute or chronic, according to the severity and duration of clinical signs. These categories probably reflect the severity of autonomic dysfunction, particularly enteric neuronal dysfunction. Clinical signs include gastrointestinal stasis, dysphagia, colic, salivation, gastric and small intestinal distension, secondary impaction of the large bowel, reduced faecal output, tachycardia, sweating, bilateral ptosis, rhinitis sicca, muscular tremors, base narrow stance and cachexia. While the acute and sub-acute forms of the disorder are, by definition, invariably fatal, a small proportion of horses with mild chronic EGS may survive. As there is no specific treatment for the disorder, intensive nursing is the mainstay of the treatment. Interestingly, horses that recover from EGS may have a persistent and marked depletion of enteric neurons, despite having no clinical evidence of gastrointestinal dysmotility. There is an increasing body of evidence, both historic and recent, to suggest that EGS is a toxicoinfectious form of botulism, caused by production of botulinum toxin(s) within the gastrointestinal tract. However, definitive proof of this is currently lacking. Prior to accepting the botulinum hypothesis, the author considers that two issues must be resolved. Firstly, while the clinical features of EGS and botulism have some overlap (including dysphagia, salivation, bilateral ptosis, abdominal discomfort, muscular tremors and weight loss), there are important clinical differ-

ences (only botulism causes mydriasis and profound myasthenia, while only EGS causes rhinitis sicca, marked tachycardia and large bowel impactions). These differences mean that the two disorders are readily differentiated by experienced veterinarians simply on clinical grounds. Secondly, horses with neuromuscular botulism do not have the neuronal pathology which characterises EGS. It is possible that EGS may be caused by botulinum toxins other than the neurotoxins, or by an organism related to botulism. Current research is directed at confirming the role of this organism and clarifying which of the botulinum subtypes and toxins are involved, with the aim of developing a vaccine to control this disorder.

Abstract #21

TRANSCRANIAL MAGNETIC STIMULATION IN THE HORSE. *H. Nollet*, L. Van Ham, G. Vanderstraeten and P. Deprez. University of Gent, Department of large animal internal medicine, Merelbeke, Belgium.

The standardization of the technique in horses is described. To prevent anxiety in some horses, the influence of a sedative combination, detomidine and buprenorphine, on magnetic motor evoked potentials (MMEPs) was examined in six horses. No significant differences could be observed for both onset latency (OL) and peak-to-peak amplitude (AMP) measurements before and after sedation. Also the influence of the coil position, current direction and stimulation intensity on MMEPs recorded in the extensor carpi radialis (ECR) and cranial tibial (CT) muscles was evaluated in seven horses. The median of the forehead seems to be the optimal coil position for recording MMEPs in both the ECR and CT muscle. The direction of the current flow in the coil had no influence on the OL of the MMEPs.

84 normal horses of different heights were stimulated transcranially. Hence normal values for OL and AMP of MMEPs recorded in the ECR and CT muscles were recorded in order to formulate a 95% prediction interval by which values obtained in clinical patients can be judged as normal or abnormal. A left-to-right difference in OL and AMP was not observed. In contrast to OL, AMP showed a very large intra- and inter-individuals variability, even in the same muscle. No significant effects of gender were observed on OL and AMP. The age of the horse had only a small but significant effect on AMP, with larger responses in older horses. Height at the withers and weight of the horse, parameters that strongly correlate with the size of the horse, had an important and significant influence on OL but not on AMP.

The usefulness of TMS was evaluated in clinical patients with spinal cord lesions. A first clinical group consisted of 65 horses with tetraplegia or ataxia in all four limbs. Only in two of these 65 horses were normal MMEPs found, probably indicating a pure sensory ataxia in those animals. In all other horses, MMEPs recorded in the ECR as well as in the CT muscles had prolonged onset latencies. In most horses the AMP was smaller. Polyphasic pattern MMEPs were observed in many of the horses. In only one of the 18 that were re-evaluated after a period of clinical improvement, had MMEPs normalised, demonstrating that the technique is also able to detect lesions in horses with subtle clinical symptoms of incoordination. The second clinical group consisted of 10 patients with bilateral hind limb ataxia. By recording MMEPs in the ECR and CT muscles, the patients could be divided in two groups: 4 horses suspected of having cervical spinal cord lesions (abnormal MMEPs in both ECR and CT muscles) and 5 horses and one donkey suspected of having a lesion of the spinal cord caudal to Th2 (abnormal MMEPs only in CT muscles).

We can conclude that the TMS is a valuable ancillary test to assess the integrity of the spinal cord in horses.

Abstract #22

''STIFF HORSE SYNDROME''?! *H. Nollet*, L. Van Ham, G. Vanderstraeten, P. Deprez. University of Gent, Department of Large Animal Internal Medicine, Merelbeke, Belgium.

During the last 6 years, 8 horses were presented in our clinic with intermittent stiffness and spasms in the axial muscles of the back and in the muscles of the pelvic limbs. The contractures were typically precipitated by voluntary movements (going to the manger, eating, coming out of the box,. . .), fright (startle) or sounds. There was no weakness or muscular atrophy, rather a muscle hypertrophy. After the first steps the movements became more relaxed and once the horse was walking or trotting no spasms were evident. In one horse (horse 8) there were also facial and labial contractions. In this horse there was also unilateral atrophy of the gluteus muscle and displacement of the pelvis due to a sacro-iliac problem after foaling. The disease has many similarities with an entity described in human medicine, namely the Stiff Man Syndrome (SMS).

In 7 of the 8 horses electromyographic findings showed persistent motor unit activity in the axial and gluteal musculature, indistinguishable from normal voluntary motor activity, that persisted for minutes before tapering off gradually. These electromyographic findings are strongly suggestive of SMS. In one horse (horse 8) no continuous motor activity was evident, but denervation fibrillation potentials and complex repetitive discharges were found in several muscles.

Administration of diazepam to these horses greatly abated clinical signs in 7 of the 8 horses and a moderate improvement in horse 8. In 5 of the 8 horses prednisolone was administered and in 2 of the 5 cases this therapy was beneficial. However, when reducing the dose, the clinical signs worsened again. Phenytoïne resulted in no improvement in any of the horses.

In human medicine an auto-immune etiology of this disorder is proposed: autoantibodies against glutamic acid decarboxylase (GAD) are found in about 60% of the patients. Therefore, serum samples were collected and examined for these autoantibodies. Only in 2 of the 8 horses were elevated GAD-antibodies found when compared to values measured in normal horses.

In 2 horses (one of the 7 similar cases and horse 8) muscle biopsies were examined. In the first horse no abnormalities were found, as described in SMS. In horse 8, however, chronic primary myopathy was diagnosed.

We can conclude the clinical findings and EMG activities in 7 of the 8 horses resemble SMS. This clinical syndrome in horses does not appear to have been described by other authors.

Abstract #23

MAGNETIC RESONANCE SPECTROSCOPY AND IMAGING FEATURES OF CANINE FUCOSIDOSIS. *J.Penderis,* J.F.McConnell, M. Jackson, A. de Stefani, and N. Holmes. Dunning, A. Lakatos, R.J.M. Franklin Animal Health Trust, Newmarket, UK.

Introduction: Fucosidosis is a lysosomal storage disease that has only been demonstrated in humans and English springer spaniels. The magnetic resonance imaging (MRI) features of fucosidosis are limited to a few human case reports and have never been described in veterinary patients. Magnetic resonance spectroscopy (MRS) is of use in the investigation of metabolic diseases where, while it is usually of insufficient sensitivity to measure the specific chemical responsible for the disorder, it can detect the secondary chemical pathological changes. In some situations these secondary chemical changes may be specific for the disorder, including Canavan's disease (aspartoacyclase deficiency), phenylketonuria and Niemann-Pick disease type C. The aims of this study are therefore to describe the MRI and MRS features of fucosidosis in two dogs.

Material and methods: Fucosidosis was confirmed in an English springer spaniel by PCR amplification of the mutated region of the α-L-fucosidase gene using published primer sequences in order to confirm a 14-base pair cDNA deletion as compared to normal controls. Fucosidosis was diagnosed in a terrier-cross dog by the demonstration of absent α-fucosidase activity (with normal β-galactosidase activity) in cultured skin fibroblasts. A normal dog cell line and 3 human cell lines were assayed simultaneously. MRS and MRI of the brain were performed under general anaesthesia using a 1.5-tesla superconducting magnet and an extremity coil. MRS was performed using a PRESS technique with long echo time (TE = 144ms) for ^{1}H in the two affected and seven control dogs, with the volume of interest defined within the thalamic grey matter.

Results: MR imaging features were similar in both dogs and demonstrated diffuse, bilaterally symmetrical hyperintensity of both the grey and white matter, but particularly the white matter of the cerebral hemispheres, with reduction of the grey-white matter interface. The white matter hyperintensity was apparent on both T_1- and T_2-weighted sequences. Subtle mass effect was evident with slight swelling and flattening of the cerebral gyri and reduction of sulci size. No abnormal contrast enhancement was evident following intravenous administration of gadobenate dimeglumine (Multihance, Bracco, Milan). MR spectroscopy demonstrated similar spectra in both affected dogs, with the predominant features being elevation of the N-acetyl aspartate:total creatinine (Cr) peak area ratio (affected dogs: 3.51 and 3.63; control dogs: mean = 1.55, SE = 0.062, range = 1.39 to 1.8), reduction of the choline:Cr peak area ratio (affected dogs: 1.48 and 1.32; control dogs: mean = 1.63, SE = 0.045, range = 1.5 to 1.83) and the presence of significant lactate production, evident as the characteristic lactate peak doublet at 1.33ppm, inverted at TE = 144ms.

Conclusion: MRI and MRS are highly useful in the preliminary diagnosis of fucosidosis and their features may allow it to be differentiated from other neurometabolic diseases.

Abstract #24

MAGNETIC RESONANCE IMAGING EVAULATION OF SPINAL CORD COMPRESSION DUE TO HANSEN TYPE I THORACOLUMBAR INTERVERTEBRAL DISC DISEASE AND ITS ASSOCIATION WITH OUTCOME IN 67 DOGS. V. Penning, S.R.Platt, V. Adams, R. Dennis, L.S. Garosi, J. Penderis and R Capello. The Queen Mother Hospital for Animals, The Royal Veterinary College, University of London.

Hansen type I intervertebral disc disease implies herniation of the nucleus pulposus through the annular fibres and extrusion of nuclear material into the spinal canal, which often results in spinal cord compression. Magnetic resonance (MR) is a superior, high-resolution, multiplanar imaging modality, which can

accurately assess the degree of spinal cord compression. The aim of this study was to determine if there is an association between the degree of MR detected transverse spinal cord compression following T3-L3 Hansen type I intervertebral disc disease, in addition to its rate of onset and its duration and (i) the presenting neurological status and (ii) the post-operative outcome.

Dogs included in this study were surgically confirmed to have type I intervertebral disc disease localised to the T3-L3 spinal cord segments. All dogs underwent a spinal MR examination prior to surgery. The rate of onset and duration of disease were recorded, in addition to the pre- and 1-month post-surgical neurological grades and the use of intravenous steroids. The percentage spinal cord compression was determined on transverse T2-weighted images at the maximal point of compression. Linear regression was used to examine the association between spinal cord compression and each of the possible above-mentioned variables. Significance was set at $p<0.05$.

Sixty-seven dogs (mean age = 6yrs) were included in the study with a mean spinal cord compression of 47% (SD = 20; range 15%-86%). Eight-five percent of the dogs were chondrodystrophoid. There was no statistical evidence to suggest that the degree of spinal cord compression was related to neurological grade at presentation, duration of dysfunction, rate of onset of clinical signs, post-surgical outcome, or any combination of these parameters. No significant difference was apparent when any of the results were compared between chondrodystrophoid dogs and non-chondrodystrophoid dogs. The use of steroids resulted in a significantly worse post-surgical outcome.

Although the degree of spinal cord compression identified with the aid of MR imaging in human intervertebral disc disease can be associated with prognosis, it was not shown to be useful in our study. The use of steroids as a medical treatment for intervertebral disc disease in dogs cannot be supported from this study.

Abstract #25

MAGNETIC RESONANCE IMAGING: A DIAGNOSTIC AID FOR INFLAMMATORY MYOPATHIES *S.R.Platt,* F. McConnell, L.S. Garosi, J. Ladlow and G.D. Shelton. Centre for Small Animal Studies, The Animal Health Trust, Newmarket, Suffolk, England.

The magnetic resonance imaging (MRI) signal intensity of normal muscle is intermediate between that of fat and cortical bone. The relative signal intensities of muscle are similar on T1-weighted and T2-weighted pulse sequences. In humans affected with polymyositis, regions of high signal intensity are found on T2-weighted images of muscles. Although electromyography (EMG) is more practically useful for muscle disease evaluation, and a muscle biopsy is the only manner in which a definitive diagnosis can be made, MRI has proven useful if a specific anatomic localisation is difficult. Three cases of focal polymyositis in dogs, diagnosed with the assistance of MRI, are discussed.

Case 1 was a 2-year-old male Boxer, which presented for a 2-week history of lumbar spinal pain. No other physical or neurological abnormalities were detected. Lumbar cerebrospinal fluid (CSF) was considered normal. An EMG examination was normal. An MR study of the lumbar spine revealed focal hyperintensities within the epaxial muscles on T1 and T2-weighted images; marked enhancement was noted in these muscles after contrast administration. Epaxial muscle biopsy confirmed an inflammatory myopathy. Case 2 was a 7-year-old male Boxer, which presented for dysphagia of 7 days duration. Physical examination was unremarkable as was the neurological examination apart from poor tongue movement. A cerebellomedullary cistern CSF was normal. An MR of the brain and brainstem was within normal limits. However, on T1-W and T2-W images, diffuse poorly marginated hyperintensity was visible in the major muscles over the cranium as well as in the tongue. Marked enhancement was visible after contrast administration. A subsequent EMG examination was abnormal. Temporalis, biceps femoris and tongue muscle biopsies confirmed an inflammatory myopathy. Type 2M antibody titers were negative. Case 3 was a 2-year 6 month-old female Briard which presented for a 3 month history of dysphagia. Contrast radiography suggested a pharyngeal mass; an MRI subsequently performed revealed a symmetrical increase in signal intensity on T1 and T2-W images within the muscles of the pharynx and larynx, with uniform enhancement after contrast administration. A 2M antibody titre was negative. Laryngeal and triceps muscle histopathology confirmed an inflammatory myopathy.

In conclusion, there was a good correlation between uniform hyperintensity on T1 and T2-W images showing enhancement after contrast administration and muscle inflammation identified histopathologically. MRI studies may be a useful adjunctive diagnostic procedure for difficult to diagnose cases of polymyositis.

Abstract #26

HEADSHAKING IN HORSES: IS REMODELLING OF THE CAUDAL OCCIPITAL BONE SIGNIFICANT? P.J. Pollock and H. McAllister. Department of Veterinary Surgery, Faculty of Veterinary Medicine, University College Dublin, Dublin, Republic of Ireland.

Headshaking in horses is a frustrating condition for both owners and veterinary surgeons. This is mainly due to the poor understanding of the condition, its multifactorial aetiology,[1] and often poor response to treatment for many horses presented with this condition. Of the variety of causes described, periosteal new bone formation on the caudal occipital surface of the skull, although documented as potentially associated with horses that head shake,[2] has yet to be fully investigated and a true incidence reported.

The medical records of horses presented at the University Veterinary Hospital, Dublin for the investigation of headshaking between 1995 and 2002 where reviewed. Horses which had new bone formation or fracture of the caudal occiput as the only clinical findings were selected. Caudal skull radiographs of all non-headshaking horses presented to the clinic were also reviewed for evidence of changes to the caudal occiput.

Of the horses presented as headshakers during this time, seven horses had new bone formation on the caudal occiput. These horses had no evidence of other potential causes of headshaking. Infiltration of the site of radiographic changes with local anaesthetic had been performed in five of these horses resulting in temporary remission of the headshaking.

Of the 20, non-headshaking, equine caudal skull radiographs reviewed, only five showed similar radiographic changes.

This preliminary study demonstrates the importance of radiographic examination of the caudal skull of horses with clinical signs of headshaking that have no evidence of the other reported causes of this condition. At this time the pathogenesis of the headshaking can only be speculated and further investigation is warranted.

Abstract #27

ARTHROCENTESIS OF EQUINE CERVICAL FACETS. S.M. Reed. The Ohio State University, Columbus, OH.

Arthrocentesis of equine cervical facets is indicated for diagnosis and treatment of some horses that present for pain or stiffness of the cervical vertebral column. Osteoarthritis or osteochondrosis of the cervical articular facets has been reported in approximately 50% of clinically normal horses at the $C_{6,7}$ cervical vertebrae. Temporary relief of the pain and/or stiffness may be achieved by injection of corticosteroids into the articular process joints.

The procedure can be performed with the horse in either a sedated standing or recumbent position. To locate the site of injection radiographs of the cervical vertebral column are essential to determine which site(s) have pathology. Palpate the location for injection; start with C_1 and progress caudally assuming each vertebra is one hand width. Orient the ultrasound transducer parallel to the long axis of the horse's neck. The articular facets are the most dorsal bony structures in the neck and they have a characteristic hyperechoic appearance. The lamina of the cranial vertebra abuts the raised pedicle of the caudal vertebra forming the appearance of a ''chair''.

End or side firing ultrasound probes can be used 7.5 MHz, 5 MHz or 3.5 MHz transducer. Higher frequency transducers are recommended because image resolution is better. The site should be clipped, blocked with local anesthetic and prepared with a surgical scrub. Sterile ultrasound gel/alcohol, sterile gloves and ultrasound probe cover are necessary. To inject we utilize spinal needles (18 or 20 gauge, 3 1/2 inch), Depomedrol 40mg/site, along with ½ cc of Amikacin.

The procedure involves location of the site(s) for injection, clip and sterile preparation of the injection site(s), and injection of local anesthetic at the injection site(s). Prepare dose and sterilely prepare ultrasound transducer and sonographer. Relocate articular facets, position joint in the middle of the image. Keeping the needle (stylet in place) parallel to the ultrasound beam, direct the tip of the needle underneath the ultrasound transducer and advance needle to into the joint. Remove stylet and aspirate joint fluid. In some instances joint fluid is not recovered. As long as the sonographer is confident that they are in the joint, the dose is injected.

Abstract #28

TREATMENT OF LAFORA DISEASE (INHERITED MYOCLONIC EPILEPSY) IN DOGS. *C. Rusbridge,* S.N. Fitzmaurice, H. Lohi, E.J. Young and B.A. Minassian. Stone Lion Veterinary Centre, Wimbledon, London, UK.

Lafora disease is a progressive myoclonus epilepsy characterised by neurotoxic starch-like polyglucosan cellular accumulations and is seen in a number of dog breeds most notably the Miniature Wire Haired Dachshund, Bassett Hound and Beagle. Age of onset is generally between 5 and 8 years. Clinical signs are typified by myoclonus with or without generalised tonic clonic seizures. The myoclonus is characterised by retropulsive jerks due to contractions of the neck and limb muscles. In addition, the jaw snaps shut or chatters and the eyelids blink rapidly. The myoclonus occurs spontaneously and in response to noise, flickering light, and sudden movement in the visual field. Hypnic (sleep related) myoclonus may also occur. The disease is not generally fatal in the dog however

the myoclonus and seizures may worsen with time and older dogs may become blind and ataxic compelling the owner to euthanasia. Treatment of Lafora disease is limited. The seizures can be managed to a certain extent with anti-epileptic drugs such as bromide, phenobarbitone and slow release phenytoin. The myoclonus does not appear to respond to these drugs although novel anti-epileptic drugs like levetiracetam may prove more effective. Both levetiracetam and zonisamide are useful for treating the myoclonus in human Lafora disease.

Therapy of human Lafora disease has recently focused on diet. Lafora disease is caused by mutations in the *EPM2A* or *EPM2B* (*NHLRC1*) genes encoding laforin starch-binding phosphatase and malin E3 ubiquitin ligase, respectively. Early data suggests that *EPM2A* and *EPM2B* genes work together to safeguard neurons against accumulating too many carbohydrates and work has focused on therapy with low carbohydrate and ketogenic diets. Limited studies suggest that the course of the canine disease is affected by diet. For example starchy/sugary treats may aggravate the condition. The authors have found that myoclonus is reduced if the dogs are exclusively fed a proprietary antioxidant rich diet (Hills b/d ™). Owners of dogs that responded to the diet reported that the myoclonus reduced very quickly, sometimes on the second feed. Hills b/d ™ protects nerve cells against oxidative damage however this may not be the mechanism by which it is effective for Lafora disease. The same effect was not observed in one dog maintained on the existing diet with supplementary antioxidants (Aktivate; Vet Plus). Hills b/d ™ has a relatively high carbohydrate content of 51.1% from a mostly maize and wheat source (i.e. starch). Dry extruded dog food typically contains 30–60% carbohydrate (mostly starch). Sugar levels in commercial pet foods are typically low (higher in some diets with gravy). Further studies are required particularly in elucidating how the canine genetic defect affects nerve cell function.

Abstract #29

EQUINE WOBBLER SYNDROME. A. C. Schütte, B. D. Grant, and George Bagby. Bonsall, CA.

Approximately 100 horses with various neurological deficits present yearly to the San Luis Rey Equine Hospital in California for evaluation. The diagnosis of cervical vertebral malformation is made after thorough evaluation. Treatment of CVM using either a bone graft or stainless steel perforated cylinder (''Bagby Basket'') has been done since 1979. About 30 horses underwent ventral cervical fusion for treatment of their cervical vertebral malformation yearly. In our study we evaluated 126 cases from 2000–2003 to compare initial condition, improvement and long-term results after surgery using a modified implant called ''Seattle Slew Implant''.

Affected horses would show a wide variety of neurological signs. Typically locomotor weakness especially of the hind limbs and ataxia was observed. Performance horses would sometimes show personality changes due to their frustration of their proprioceptive loss and incoordination leading to difficulties to do their job. Neurological examination includes tests of cranial nerve functions, pain response to palpation of the temporo mandibular joint, panniculus testing, evaluation of walk and trot on a straight line, walk up- and downhill with natural and elevated head carriage, limb placement, tight circles and manipulative tests like back withdrawal, neck mobility and tail sway. For grading a neurological scale introduced by Mayhew was used, ranging from 0 (no neurological deficits) to 5 (unable to walk). Most of our cases were graded 1/5–3/5 on this scale. After Radiographs of the skull and cervical area, myelogram and CSF analysis are obtained, cord compression is diagnosed when both the dorsal and ventral dye columns are 50% reduced in width when compared to the neutral position. Prognosis and treatment recommendations were made according to the neurological grading and diagnostic findings. In general, horses with short duration of clinical signs and neurological grades less than 3/5, one compression site and a good recovery from anaesthesia for the myelogram are given the best prognosis.

After cervical fusion was performed approximately 80% of the patients had improved one grade from their initial exam at a 60 days post Op re-check. Complication rates for this procedure were 6% for fatal complications. Long term results were evaluated by personal examination of the horse or information obtained by the owner or referring veterinarian. We found that 60% were successfully ridden at different levels, of which many were even competing at shows again, 20% were used for breeding and 14% were retired for different reasons. We learned that there were no direct correlations to be established between severity or kind of clinical signs and the severity or numbers of cervical cord compression sites. Especially if surgery was chosen at a favourable early time the outcome in most of the cases strongly recommends surgical treatment for cervical vertebral malformation.

Abstract #30

NEUROLOGICAL EVALUATION AND ANALYSIS OF OUTCOME IN DOGS PRESENTED WITH LARYNGEAL PARALYSIS. *P. Smith*, K. Talbot and N. Jeffery. Department of Clinical Veterinary Medicine, University of Cambridge, CB3 0ES, England.

The long-term prognosis for old large-breed dogs that suffer laryngeal paralysis is uncertain and may depend on other disabilities caused by a putative associated generalised neuropathy. This study was undertaken to investigate the outcome in dogs with laryngeal paralysis following surgery, with the aim of identifying pre-operative prognostic indicators.

Retrospective analysis of sixteen operated cases revealed that three had died soon after surgery; five had considerably reduced exercise tolerance and a further three exhibited milder locomotor disability.

Twelve dogs (seven Labradors) were subsequently examined prospectively, though it was not possible to carry out all tests in all cases. Neurological examination was abnormal in seven of eight evaluated dogs and nerve conduction velocity was reduced in the ulnar nerve (mean 38.5 m/s) in seven of eight dogs and in the sciatic nerve (mean 43.7 m/s) in eight of eight dogs; tibial nerve residual latency was prolonged in three of five dogs in which it was measured. Eight of nine dogs had no evidence of hypothyroidism and seven of seven dogs had no evidence of immune-mediated myasthenia gravis. Two dogs died soon after surgery because of aspiration pneumonia, three showed persistent exercise intolerance but four were improved; the remainder were unavailable for follow-up. Four dogs had evidence of multiple cranial nerve dysfunction, of which two died post-operatively. The three animals showing persistent exercise intolerance all had prolonged tibial nerve residual latencies.

We conclude that generalised peripheral neuropathy complicates the response to surgery in approximately 50% of cases. Prolonged tibial nerve residual latency may predict those dogs in which exercise intolerance will persist.

Abstract #31

BSE AND CJD: WHAT DOES THE FUTURE HOLD? *R.G.Will.* National CJD Surveillance Unit, Bryan Matthews Building, Western General Hospital, Crewe Road, Edinburgh, EH4 2XU.

The epidemic of bovine spongiform encephalopathy (BSE) in the UK has been in decline for some years. The active testing programme introduced in continental Europe now suggests that the incidence of BSE is now decreasing in most countries. However, international concern about BSE continues following the identification of cases in Japan, Canada and the USA, underlining the view of the joint WHO/FAO/OIE consultation in 2001 that BSE risk has a global dimension.

There is now good evidence that BSE is the cause of variant Creutzfeldt-Jakob disease (vCJD) but the initial fears of a large human epidemic have not been borne out. In the UK, the country with the largest number of vCJD cases (and the greatest risk of human exposure to BSE) there has been a decline in the number of cases of vCJD over the past few years. There are continuing concerns about future waves of vCJD occurring for example in individuals with different codon 129 genotypes, or through iatrogenic transmission. The identification of possible transmission of vCJD through blood transfusion has underlined the importance of public health measures designed to minimise the chances of onward transmission of infection from person to person.

In response to the public health concerns raised by BSE, a range of legislative measures have been introduced in the UK, other European countries and elsewhere. The costs of these measures are considerable and an important challenge for the future is to decide whether these measures remain necessary and whether they are proportionate to the changing level of risk as BSE epidemics decline.

Abstract #32

WHAT'S UP DOC? NEUROLOGY OF THE RABBIT. P. Boydell. Animal Medical Centre Referral Services. Manchester/Yorkshire, UK.

The rabbit has become an increasingly frequent patient, making up some 10% of active patient lists at some veterinary practices in the UK. The majority of these are now indoor companion animals and are much less likely to be neglected children's pets languishing at the bottom of the garden. As such, the average owner's awareness of the pet and its health has improved dramatically, and the pet-owner bond has developed concurrently. As a result there is a much greater demand for advanced veterinary care and referral services.

The commonest neurologic presentation involves vestibular signs. These may be peripheral or central associated with otitis media/interna and encephalitis. The role of *Pasteurella multocida* and *Encephalitozoon cuniculi* as specific infectious agents is commonly suspected but proven with far less frequency.

Spinal trauma may be a result of a direct blow to the back, or, more commonly struggling during handling. Such presentations may be reducing as more rabbits have a better plane of nutrition and exercise allowing satisfactory bone strength.

Other presentations are less common but include many of the conditions that the small animal neurologist might expect to see in other species and it is generally a pleasure to deal with these animals and their well-informed, caring owners.

Abstract #33

THE IMPACT OF IDIOPATHIC EPILEPSY AND ITS MANAGEMENT ON THE DOG'S QUALITY OF LIFE AND THE OWNER'S LIFESTYLE. *Y-P Chang* and T.J. Anderson, Department of Veterinary Clinical Studies, Institute of Comparative Medicine, University of Glasgow Veterinary School, Glasgow, Scotland.

When treating a dog with idiopathic epilepsy, seizure frequency has traditionally been considered a key outcome assessment tool. In the human medical literature, seizure severity and quality of life measurements in epileptic patients are also considered important in assessing outcome of management. There is a paucity of veterinary literature on the effects of other aspects of seizure management on owners and the relationship with their pets.

A retrospective questionnaire study was carried out to explore the impact of idiopathic epilepsy and its management on the dog's quality of life and the owner's lifestyle. Dogs referred to the University of Glasgow Veterinary School between 1999 and 2001 for evaluation of recurrent seizures, diagnosed with idiopathic epilepsy and treated with antiepileptic drug therapy were included in this study. Question types included open-ended questions, close-ended questions with ordered answer choices, partially close-ended questions and questions with the visual analogue scale (VAS). The VAS was applied as a 100-point measurement in this study. Patients were divided into two groups on the basis of perceived response to therapy. The Wilcoxon rank sum test was applied to analyse whether adequate seizure control would influence the impact of caring for an epileptic dog on the owner's lifestyle.

Twenty-nine dogs were included in the study. An 86% response rate was obtained. For 52% of dogs, daily activity was perceived to have decreased following the onset of seizures. Two-thirds of owners indicated the interaction between the dog and family members remained unaltered. Half of owners considered that the general quality of life in their dogs was decreased. Adverse effects of antiepileptic drugs, inadequate control of seizures and behavioural changes were given as reasons for this deterioration. The majority of owners did not consider the administration of medication as a problem. Half of owners strongly disagreed with the statement 'regular veterinary examination and blood sampling to monitor serum drug concentration is a nuisance'. One-third of owners with work commitments and two-thirds of owners without work commitments considered caring for an epileptic dog had not caused them conflicts with their work/day-to-day activity. Forty percent of owners felt caring for an epileptic dog did not affect their free time. On these two issues, no significant difference of the responses was found between dogs with adequate seizure frequency and dogs with inadequate seizure frequency.

In conclusion, given the positive attitudes expressed by the majority of owners, it is suggested that regular veterinary examination and serum drug concentration monitoring should be encouraged to achieve optimal seizure management. However, reducing the seizure frequency dose not necessarily decrease the impact of caring for an epileptic dog on the owner's lifestyle.

Abstract #34

MRI FINDINGS IN 7 DOGS WITH CONFIRMED GME. G.B. Cherubini, T.J. Anderson, C. Rusbridge, P. Mantis and R. Cappello. The Queen Mother Hospital for Animals, The Royal Veterinary College, London UK.

The purpose of this multicentric retrospective study was to determine the MRI findings and characteristics of GME in dogs. MRI was performed in 7 dogs with histopathological confirmed GME. In 6 dogs the GME neuroanatomical location was in the brain, in 1 dog was in the spinal cord. A single lesion was present in 3 dogs and multiple lesions were found in 4 dogs.

The following data were recorded: breed, gender, age, neurological signs and CSF analysis. Magnetic resonance images on: T1WI, T1WI post gad, T2WI, FLAIR, Gradient Echo (GE) and Proton Density (PD), were assessed. MR images were analysed looking for presence of lesion, neuroanatomical localisation, parenchyma distribution, shape, MRI intensity on T1WI, T2WI, FLAIR, GE and PD images, mass effect, presence of brain or cerebellum herniation, hydrocephalus, gadolinium enhancement, perilesional edema.

This first consistent study evaluating the MRI features in histological confirmed GME, indicates that GME has certain MRI features: multiple lesions, hyperintensity in T2WI and FLAIR, absent of herniation, tendency to be hypointense in T1WI primary macroscopically involvement of the grey matter that, thought not specific for GME, combined with the history, neurological signs and CSF analysis are helpful to increase the specificity of presumptive diagnosis of GME.

Abstract #35

AORTIC THROMBOEMBOLISM IN PEDMONTESE CALVES, A. D'Angelo, C. Bellino, G.L. Alborali, M.T. Capucchio, C. Casalone, M.I. Crescio, G.L. Mattalia, V. Vittone, A. Jaggy. Department of Animal Pathology University of Turin, Italy.

This report describes the clinical, pathological and etiological aspects of aortic thromboembolism in two calves. The first case, an 11 day old female Pedmontese calf, was referred to the Teaching Hospital of the Faculty of Veterinary Medicine of Turin for an acute onset of paraplegia two days previously. One day before the animal had showed diarrhea that had been treated and controlled with antibiotics, non steroidal ant inflammatory drugs and fluid therapy.

At presentation the calf was slightly depressed, dyspnoic and had pale mucous membranes. Perineum and pubis were dirty with urine. Neurological examination showed flaccid paralysis and absent spinal reflexes in the pelvic limbs. The anal reflex was decreased, the tail was flaccid and the bladder easy to void by manual expression. Both pelvic limbs were cold and there was no femoral pulse or conscious perception of pain. The animal could maintain sternal recumbency. Cranial nerves as well as spinal reflexes in the thoracic limbs were normal. The calf's condition deteriorated and it died into two hours. Necropsy was performed. A cisternal cerebrospinal fluid sample, collected soon after death, showed an increase of protein (54,2 mg/dl). The haematological profile indicated neutrophilia (57,3%), elevated AST (2750 U/L), CK (>3000), BUN (115 mg/dl) and creatinine (9,54 mg/dl). Protein electrophoresis showed hypoalbuminemia (1,5 g/dl) and β globulin increase (3,9 g/dl). Total proteins were normal (7,4 g/dl) while albumin globulin rate resulted strongly decreased (0.26).

The second case a male, eight day old Pedmontese calf from a different herd, was referred for the same clinical signs Unfortunately this animal died during transport and only necropsy was performed. In both case one and two multifocal hemorrhages, edema, necrosis, variation in fiber sizes and inflammatory cell infiltrates were observed in the muscles of the hind limbs. The kidneys showed large hemorrhages associated to multifocal areas of necrosis. Venous thrombi were present in the lungs associated to focal areas of necrotic bronchopneumonia. Voluminous thrombi were observed at the trifurcation of the abdominal aorta. The sacral spinal cord showed moderate axonal and neuronal degeneration associated to gliosis. No lesions were observed in the brain and in the heart. An hemolytic E. coli strain was isolated from several affected tissues and long bones.

An *E. coli* strain isolation in both these two cases could be the etiological cause for thrombus formation. Circulation of antigen-antibody complexes or of bacterial endotoxins could represent the pathogenic mechanism in thrombogenesis both by platelet aggregation and by vascular endothelium damage.

Abstract #36

MEDULLOBLASTOMA IN A DALMATIAN PELICAN (*PELECANUS CRISPUS*). M. Friess, N. Robert, W.Haefeli, C. Botteron. NeuroCenter, Institute of Animal Pathology, Vetsuisse Faculty, University Bern.

A 2-year-old Dalmatian pelican (*Pelecanus crispus*) of a colony in a Swiss animal park showed acute loss of balance (falling on his back), head tilt, hypertonus of the legs and tachypnoea. Radiographic examination of the body did not show any abnormalities. The animal was treated with antibiotics, dexamethason and vitamin B, but the clinical signs did not improve and the animal died suddenly overnight. At necropsy a poorly demarcated pale mass was found in the caudal portion of the cerebellum. The tumor was examined histologically by H&E and using different immunohistochemical stains (S-100, GFAP, NSE, neurofilament and synaptophysin).

Histologically the mass was densely cellular and showed a infiltrative growth. The neoplastic cells were arranged in short interwoven bundles, were elongated to oval, had indistinct cell borders, and a scant to moderate amount of eosinophilic cytoplasm. The nuclei were small, round to elongated, with dense chromatin. Scattered cells had a large amount of eosinophilic cytoplasm and a round to oval nucleus with a single distinct nucleolus (neuronal differentiation). The mitotic rate was high and ranged from 5 to 10 per high power field. The neoplastic cells were arranged radially around blood vessels, forming pseudorosettes. Multiple small necrotic areas consisting of cellular debris and various numbers of gitter cells were distributed throughout the tumor. Moderate to severe lymphocytic infiltrates were found in the meninges adjacent to the tumor and around blood vessels, mostly at the tumor margins.

The diagnosis of medulloblastoma was based on the localization of the tumor, the young age of the animal (pelicans may live over 20 years) and the typical histological and immunochemical findings.

Medulloblastomas are primitive neuroectodermal tumors which are believed to originate from cells of the outer germinal layer (fetal granular layer of Obersteiner) and are always located in the cerebellum. These highly malignant brain tumors (WHO grade IV) are common in young children. In young animals these tumors are rare and were only described in domestic animals of the class mammalia such as calves, puppies, kittens and piglets. This is the first report of a medulloblastoma in a bird.

Abstract #37

DETECTION OF *NEOSPORA* TACHYZOITES IN CANINE CSF. L. Gaitero, M. Pumarola, P. Montoliu and S. Añor. Department of Animal Medicine & Surgery, Veterinary School, Autonomous University of Barcelona. 08193 Barcelona, Spain.

A four-year-old female Labrador Retriever was referred to the Veterinary Teaching Hospital of the Autonomous University of Barcelona for progressive generalized ataxia, depressed mental status and generalized seizures of 7–10 days duration. On referral, no abnormalities were detected on physical examination. Neurologic examination identified a stuporous mental status, decerebrate rigidity and non-ambulatory tetraparesis. Abnormalities on cranial nerve examination included absent oculocephalic movements, absent bilateral menace responses, anisocoria (left eye mydriatic) and absent pupillary light reflexes. Neuroanatomical lesion localization was diffuse or multifocal (forebrain and brainstem). Emergency treatment consisted of mannitol, dexamethasone, ranitidine and fluid therapy.

Complete minimum data base was unremarkable. MRI images demonstrated brain stem meningeal thickness with diffuse supra and infratentorial meningeal contrast enhancement, including the cranial cervical segment, and a left parietotemporal focal lesion causing mass effect over the left lateral ventricular wall. These findings were considered characteristic of a diffuse pachymeningitis with a probably secondary left cortical ischemic focal lesion. Cerebrospinal fluid (CSF) collected from the cerebellomedullary cistern showed a severe mixed, mostly mononuclear pleocytosis, and elevated protein concentration. Cytologic CSF examination revealed the presence of numerous protozoal tachyzoites. They were seen intracellularly within inflammatory cells and as free extracellular tachyzoites. CNS neosporosis was diagnosed and treatment with clindamycin was started. The patient's status deteriorated despite treatment and death due to cardiorespiratory arrest occurred 5 days after admission. Post-mortem examination demonstrated a severe protozoal meningoencephalomyelitis with multifocal encephalomalacia and presence of bradyzoite cysts. Immunohistochemical techniques performed in brain histological sections were positive for *Neospora caninum*. A diffuse meningoencephalomyelitis due to *Neospora caninum* infection was the final diagnosis.

This report describes the case of an adult dog with progressive multifocal neurological signs caused by N. caninum. To the authors knowledge, this is the first clinical case report in which cytologic detection of *N. caninum* in the CSF of a dog is described. In humans, *Toxoplasma gondii* organisms can be identified on cytologic examination of CSF. However, direct identification of *T.gondii* in the CSF of human beings is extremely rare and only 3 cases have been documented to date. Based on the CSF findings in this dog, a diagnosis of canine CNS neosporosis can be made by identification of tachyzoites in CSF samples.

Abstract #38

ELECTRICALLY INDUCED CUTANEOUS TRUNCI REFLEX IN HORSES. J.M. Gorraiz, L. Monreal and S. Añor. Hospital Veterinari Canis, Gerona, Spain.

The cutaneous trunci reflex is a contraction of the cutaneous trunci muscle in response to a cutaneous stimulus. In healthy horses, gentle pricking of the skin over the trunk, particularly in the lateral aspects of the body wall, causes a contraction of the cutaneous trunci muscle, which is seen as a flicking of the skin over the trunk. The sensory stimulus travels to the spinal cord in the thoracolumbar spinal nerves at the level of the site of stimulation. The stimulus is then transmitted cranially in the spinal cord through deeply located pathways to C8-T1, where synapse with the lower motor neuron cell bodies of the lateral thoracic nerve occurs. Stimulation of the lateral thoracic nerve causes contraction of the cutaneous trunci muscle. Lesions anywhere along this pathway may cause suppression of the reflex. Clinically, spinal cord disorders affecting the thoracolumbar segments cause decrease or absence of the reflex 1 to 2 segments caudal to the lesion. The reflex is not affected by common equine cervical spinal cord disorders such as cervical stenotic myelopathy, but it can be depressed in horses with equine degenerative myeloencephalopathy (EDM) or other conditions affecting the thoracolumbar spinal cord.

The aim of this study was to assess the cutaneous trunci reflex electrophysiologically, in order to assess reproducibility and to obtain normal reference values for the muscle reflex responses obtained, to further apply the test to potential EDM cases. To elicit the reflex, the 16th left thoracic sensory nerve was electrically stimulated in 11 Andalusian adult horses using needle electrodes placed just caudal to the 16th rib, with the cathode approximately 1cm dorsal to the anode. The evoked reflex muscle responses were recorded using a concentric needle electrode inserted in the cutaneous trunci muscle, about 15 cm caudal to the ipsilateral elbow. Stimuli were square-wave pulses of 0.1 ms duration and supramaximal intensity. A ground electrode was placed subcutaneously between the stimulating and the recording electrodes. Twenty recordings were obtained for each horse. Means and standard deviations (SD) for each parameter studied were calculated using the values of all responses. The reflex muscle responses obtained after stimulation were polyphasic muscle potentials of variable duration, amplitude and area, which were followed in some cases by a second potential, probably related to pain sensation, as seen in man. Mean latency values $\pm$ SD of the evoked muscle potentials were 53.74 $\pm$ 10.18 ms. Mean amplitude values were 29.26 $\pm$ 10.31 mV. Mean values of the area of the muscle potentials were 288.54 $\pm$ 120.57 ms.mV, and mean duration values were 52.13 $\pm$ 23.79 ms. Results of this study demonstrate that the cutaneous trunci reflex can be electrically induced and recorded in horses.

Further studies are necessary to demonstrate the potential usefulness of the test for the pre-mortem diagnosis of EDM or other disorders affecting the equine thoracolumbar spinal cord.

Abstract #39

ENDOSCOPIC BIOPSY OF ESOPHAGEAL TONSILS IN A *BOA CONSTRICTOR IMPERATOR*: A NEW DIAGNOSIS APPROACH OF INCLUSION BODY DISEASE. N. Granger, G. Breuil, J.L. Thibaud, L. Schilliger and S. Blot. Unité de Neurologie, Ecole Nationale Vétérinaire d'Alfort, France.

Inclusion Body Disease (I.B.D.) is caused by a Retroviridae and primarily affects snakes of the family Boidae. Snakes originating from both private and zoological collections in the United States, Africa, and Europe have been diagnosed with this disease. The route of natural transmission is unknown but all the body secretions are supposed to be infectious, leading to considerable loss in reptile collections. The growing number of cases diagnosed for 10 years has increased the need of a direct and antemortem diagnosis.

A 6 year old male *Boa constrictor imperator*, was presented with a 2 week history of progressive postural abnormalities and behavioral changes. The Boa was originating from the United States. Husbandry conditions were in agreement with conditions required for boid snakes. The owner reported a 5 month period of anorexia and regurgitations. Neurological examination reveals loss of righting reflex, spiral convolution and, when the snake was stimulated, a ''star gazing'' posture. Encephalitis caused by bacterial, mycotic and viral infection (I.B.D. or paramyxovirus) was primary suspected. Other conditions such as metabolic diseases or neoplasia may cause postural abnormalities. Routine analysis (biochemistry, blood cytology and fecal analysis) were unremarkable. As the index of suspicion for I.B.D. increases, the snake was anaesthetized (0.1 mg/kg IM of chlorhydrate medetomidine and 50 mg/kg IM of chlorhydrate ketamine) for endoscopic biopsy of esophageal tonsils. Histologic examination revealed eosinophilic intracytoplasmic inclusions in epithelial cells, confirming the diagnosis of I.B.D.. Considering the infectious risk and the poor prognosis associated with I.B.D., the snake was euthanized. At necropsy, inclusion body were also found in cerebral and hepatic tissues.

Serologic tests are currently researched. To date, only liver biopsy can lead to an antemortem diagnosis of I.B.D.. Esophageal tonsils biopsy for I.B.D. diagnosis have only been reported once in snakes. This easier and less invasive procedure seems to be as sensitive as liver biopsies, but need to be extended to a larger number of boids infected by I.B.D. to precise its capacities. As the number of infected boids is increasing and often affect collections of snakes, this approach could be proposed as an antemortem screening procedure for I.B.D. carrier animals.

Abstract #40

PARAPARESIS IN A MINIATURE GOAT—DIAGNOSIS BY MAGNETIC RESONANCE IMAGING. M. Gygi, Kathmann, M. Konar, S. Rottenberg, A. Jaggy and M.. Meylan. Department for Clinical Veterinary Medicine, Large Animal Clinic, University of Berne, Switzerland.

A four-year-old miniature goat was presented at the large animal clinic, Vetsuisse-Faculty of the University of Berne, Switzerland with the history of a chronic progressive paraparesis. The clinical anatomical localisation was between spinal cord segments thoracal 3 and lumbar 3 because of moderate paraparesis, absent proprioception, normal to increased spinal reflexes and hypertonicity of the muscles in the hind limbs.

Plain radiographs of the vertebral column showed a slight deviation of the dorsal spinal process of the third lumbar vertebral body to the left. A Magnet Resonance Imaging (MRI) of the thoraco-lumbar vertebral column was performed. A 14 x 6 x 7 cm inhomogeneous space occupying lesion was seen extending ventrally and on the left side of the lumbar vertebrae at the level of L 3 to 6, invading the vertebral canal through the neuroforamina L3/4, L4/5 and L5/6. The most extensive compression of the spinal cord was at the level of L4. The differential diagnosis of the lesion given the signal behaviour in the different sequences were lymphosarcoma, rhabdomyosarcoma and sarcoma of other origin, and the owner decided to euthanize the animal. The macroscopic pathologic examination revealed further masses at the level of the proximal thoracic vertebral column, proximal to the thoracic aperture and in the thorax. The histopathologic examination of the mass showed a proliferation of pleomorphic round cells which where immunohistochemically positive for CD 79a and CD 3 and therefore classified as B-cells. The spinal cord showed multifocal degeneration of axons with swelling and fragmentation of the myelin sheets, as well as an infiltration by macrophages. No infiltrating neoplastic cells were detected. The histopathologic diagnosis was a B-cell lymphosarcoma.

MRI appears to be a good diagnostic tool for use in small ruminants with spinal cord disease, especially in those, like in this case, which are considered as pets and therefore costs are of less importance.

Abstract #41

CONJUNCTIVAL BIOPSIES AS A DIAGNOSTIC AID IN ASSESSMENT OF LATE-TYPE NEURONAL CEROID LIPOFUSCINOSIS IN TIBETAN TERRIERS AND POLISH OWCZAREK NIZINNY DOGS. K. Matiasek1, R. Brahm and W Schmahl. Chair of Neuropathology & General Pathology, Institute of Veterinary Pathology, Faculty of Veterinary Medicine, LMU Munich, Germany.

The term neuronal ceroid lipofuscinosis (NCL) refers to a group of inherited storage diseases characterized by extensive intralysosomal accumulation of autofluorescent lipid droplets, and neurodegeneration. Amongst plenty affected canine breeds, autosomal-recessive traits could be demonstrated in English setters, border collies and Tibetan terriers whereas sporadic cases have been described in Dalmatians, Polish owczarek nizinny (PONs) dogs and others. In humans the 8 clinical NCL-subtypes have been traced back to more than 151 mutations of the 8 candidate genes CLN 1 to 8. They can give rise to both enzyme deficiency and structural protein dysfunction. Up to now, all attempts to identify mutations of candidate genes in dogs have failed. Therefore, the definite diagnosis of canine NCL still is based on post mortem analysis. Within a period of several months four adult Tibetan terriers and two adult PONs with disorientation and abnormal behaviour had been presented to one of us. All dogs exhibited various degrees of mental and cognitive impairment, gait abnormalities with ataxia and paresis, and visual deficits. A complete ophthalmological examination including ophthalmoscopy, biomicroscopy and electroretinography (ERG) was performed on two Tibetan terriers and both PONs. One of the latter presented the typical NCL-associated fundus abnormalities and ERG changes which are known to occur in this breed. In the remaining dogs no ocular abnormalities could be observed. Based on both the clinical findings and the data obtained from pedigree analysis, diagnosis of NCL in all six animals had been proposed. In consequence, conjunctival biopsies were taken and processed for electron microscopy. All of them revealed abundant electron dense osmiophilic, granular, membranous and/or finger-print-like lipofuscinic inclusions within endothelial cells, vascular smooth muscle cells, pericytes, perineurial cells, Schwann cells and axons, when compared to age-matched controls. The diagnosis of NCL was offered and later confirmed for one Tibetan terrier which had to be euthanized after rapid deterioration of its neurological state. In this dog NCL could be proven histologically, histochemically and by electron microscopy.

Taken together, post mortem neuropathology is prerequisite for definite diagnosis of NCL in dogs that do not exhibit typical alterations of the retina. Unless specific biochemical and genetic tests will be available for clinical settings, abundant lipopigment accumulation in conjunctival biopsies is the most conclusive sign indicating late-onset NCL in living Tibetan terriers and PONs.

Abstract #42

CONJUNCTIVAL BIOPSIES AS A DIAGNOSTIC AID IN ASSESSMENT OF LATE-TYPE NEURONAL CEROID LIPOFUSCINOSIS IN TIBETAN TERRIERS AND POLISH OWCZAREK NIZINNY DOGS. K Matiasek, R. Brahm and W Schmahl. Chair of Neuropathology & General Pathology, Institute of Veterinary Pathology, Faculty of Veterinary Medicine, LMU Munich, Germany.

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Taken together, post mortem neuropathology is prerequisite for definite diagnosis of NCL in dogs that do not exhibit typical alterations of the retina. Unless specific biochemical and genetic tests will be available for clinical settings, abundant lipopigment accumulation in conjunctival biopsies is the most conclusive sign indicating late-onset NCL in living Tibetan terriers and PONs.

Abstract #43

HISTOLOGICAL STUDY OF 32 CASES OF MENINGIOMA IN DOGS. P. Montoliu, M.. Pumarola, E. Vidal and S. Añor. Department of Animal Medicine & Surgery, CRESA Foundation Veterinary School, Autonomous University of Barcelona. 08193 Barcelona, Spain.

Thirty-two cases of canine meningioma diagnosed through surgical biopsies or necropsies were reviewed using routine light microscopy methods. Of the 32 dogs, 17 were male (58%), 12 were female (41%) and three dogs were of unknown gender. Mean age was 9.5 years (2–15), and 53 % of dogs were 10 years or older. Nine breeds of dogs were represented. German Shepherd and mixed breed dogs were the most common, accounting for 21% and 34 % of the cases, respectively. Tumor location was recorded for 30 of the 32 tumors. Twenty-five meningiomas were located intracranially and of these 22 were supratentorial (88%), 3 were infratentorial tumors (12%), 2 were retrobulbar and arising from the optic nerve, and 3 were located in the spinal canal.

Clinical signs were recorded in 25 cases. Seizures was the most common clinical sign recorded, affecting 60% of dogs with intracranial meningioma. Other clinical signs in dogs with intracranial meningioma were lethargy (30%), central vestibular signs (20%), paresis (15%), behavioral changes (5%) and circling (5%). One dog with an optic nerve meningioma presented for blindness in the left eye, and the other one had exophthalmos of the right eye. Dogs with spinal cord meningiomas presented for different degrees of paresis.

Nine types of meningioma were diagnosed: transitional (8/32; 25%), meningothelial (7/32; 21%), psammomatous (4/32; 12.5%), anaplastic (4/32; 12.5%), fibroblastic (3/32: 9%), angioblastic (2/32; 6%), microcystic (1/32; 3%) and papillary (1/32; 3%). Optic nerve meningiomas were considered a separate entity. Meningothelial and transitional meningiomas were more common than any other type. Four tumors were classified as anaplastic meningiomas based on the presence of, at least, four of the following features: increased cellularity, infiltrative growth, extensive necrosis, haemorrhage, cellular atypia, nuclear pleomorphism and high mitotic index. Other meningiomas showed one or more malignant criteria also, but this was not the predominant pattern. One case displayed a predominant microcystic pattern (clear and vacuolated tumor cell cytoplasm) and was classified as a microcystic meningioma according to the WHO human classification of meningiomas. Six of the tumors in this series displayed one or more types of mesenchymal metaplasia (cartilaginous, osseous or myxoid), and among these there were the two optic nerve meningiomas and the three spinal cord meningiomas.

The Domestic Animal classification of meningiomas (WHO, 1999) was used to classify most of the tumors in our series. Nevertheless, several tumors included in this retrospective study do not fit exactly within this classification. The human WHO classification has been used to classify the mycrocystic meningioma due to its similarities with the human variant. In our opinion, optic nerve meningiomas should be included in the domestic animal classification as a separate entity due to their distinct and typical morphologic features.

Abstract #44

CLINICAL SURVEY OF NEUROLOGICAL DISEASES IN SMALL RUMINANTS. H.C. Schenk, A. Gerdwilker, M. Ganter, W. Baumgärtner and A. Tipold. Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover.

In recent years small ruminants become more and more important to their owners as companion animals. There is a rising interest in advanced diagnostics of CNS disorders in the individual animal due to the BSE-crisis and the fight against TSE, especially Scrapie. Only a few studies on the importance and diagnosis in neurological diseases of small ruminants are available.

During a big survey of neurological diseases in TSE susceptible species, 87 small ruminants with neurological signs where examined. Clinical exams and additional tests such as blood work, cerebrospinal fluid (CSF), analysis, diagnostic imaging including radiography, CT, MRI and electrodiagnostics were performed. The animals which did not recover (n = 30) were necropsied and examined histopathologically to correlate these findings to the clinical data. The pathological results correlated with the clinical findings in 22 cases (73.33%).

As shown in the table below the majority of animals suffered either from metabolic–toxic diseases like hypocalcaemia and vitamin E/ selenium deficiency (n = 45) or inflammatory/ infectious diseases (n = 29) such as listeriosis or other bacterial infections. The cell counts in the CSF were elevated in both of these disease categories (cell count in inflammatory diseases 4–2679 cells/μl, in metabolic/toxic diseases 0.1–190 cells/μl). MRI and CT scans were most valuable in characterising intracranial and spinal lesions. These advanced techniques were used in 13 cases and could confirm the diagnosis in 10 of these animals.

Electrodiagnostics like EMG and NCV in 7 cases supported the localisation of the lesion in the peripheral nervous system and the muscles. Due to the wide variation of lesions and diseases in small ruminants it is necessary to use several special investigation techniques, as is state of the art in companion animals.

	Small ruminants		
Disease categories	Total n = 87	Sheep n = 68	Goats n = 19
Vascular	1	—	1
Inflammatory/infectious	29	19	10
Trauma	8	7	1
Anomaly	1	1	—
Metabolic—toxic	45	38	7
Idiopathic	3	3	—

Abstract #45

AUTOANTIBODIES AGAINST STRUCTURES OF THE CENTRAL NERVOUS SYSTEM IN STEROID-RESPONSIVE MENINGITIS-ARTERITIS IN DOGS. K. Schulte, R. Carlson and A. Tipold. Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

Steroid-responsive meningitis-arteritis (SRMA) is a well known disease of dogs. A combined evaluation of IgA in serum and cerebrospinal fluid (CSF) seems to be an important diagnostic tool. It is suspected that immunpathological mechanisms are involved in the pathogenesis of SRMA because of the marked response to steroids. Although the etiology of SRMA is still unknown, excessive production of IgA seems to play a central role in its pathogenesis. One reason for the uncontrolled IgA synthesis in SRMA might be an immune reaction to self-antigens of the central nervous system (CNS).

To test this hypothesis, we analyzed cerebrospinal fluid (CSF) samples from 55 dogs with SRMA using the western blot method. After blotting canine brain tissue IgA, IgM and IgG of the CSF samples were tested for their affinity to CNS structures. We also evaluated CSF samples from 45 dogs with other brain diseases, including encephalitides and intracranial tumors, and from healthy dogs as controls.

Positive reactions were observed in the CSF samples from dogs with SRMA, encephalitides and brain tumors (a total of 8% positive samples). IgA reacted more frequently with CNS proteins than did the other CSF immunoglobulins. The occurrence of autoantibodies against CNS structures was significantly higher in the control group ''other brain diseases'' than in the SRMA group ($P = 0.0135$). There was no significant difference in the number of positive samples between dogs with SRMA and the negative control group (healthy dogs, $p = 0.1535$). Despite the small number of positive samples, only dogs with abnormal findings in the CSF analysis also had autoantibodies in the CSF. There was no significant correlation between the occurrence of autoantibodies and levels of IgA and cell counts in the cerebrospinal fluid, nor was there any significant correlation between autoantibodies and protein content of the CSF. However, there was a certain trend toward positive reactions in CSF samples with high protein content.

Positive antigen-antibody reactions were found with CNS proteins of different sizes (between 10 and 130 kDa). Because of the small number of positive reactions, antigens were not defined in the current study. However, a reaction with glial fibrillary acidic protein (GFAP) or with heat shock protein (HSP60) could be possible. The present study did not substantiate the hypothesis that SRMA is caused by an autoimmune reaction. The occurrence of autoantibodies in dogs with SRMA thus seems to be an epiphenomenona rather than the cause of the disease.

Abstract #46

MAGNETIC RESONANCE IMAGING FEATURES OF LUMBOSACRAL OSTEOCHONDRITIS DISSECANS IN THREE DOGS. A. de Stefani, L.S. Garos, S.R. Platt, J. Penderis, F.J. Llabrés-Díaz and R Dennis. Animal Health Trust, Lanwades Park, Newmarket, Suffolk, England, UK.

Introduction: Lumbosacral osteochondritis dissecans (OCD) is an increasingly recognised disease that is over-represented in young male German Shepherd Dogs (GSD) and is of significance in the pathophysiology of cauda equina syndrome (CES). The radiographic features of the OCD lesion have been well characterised and the disease process has been further described in the past using a variety of imaging techniques, including myelography, epidurography and CT. Besides reference to the use of magnetic resonance imaging (MRI) in one case, the MRI features of lumbosacral OCD have never been characterised and the aim of this presentation is therefore to describe the MRI features of lumbosacral OCD and to present the advantages of this imaging technique compared to other imaging techniques.

Material and methods: Three dogs were included in this study. Inclusion criteria were radiographic and MRI evidence of sacral OCD and clinical signs compatible with CES. All three dogs underwent complete physical and neurological examination prior to imaging. Neutral and flexed lateral and ventrodorsal survey radiography was performed in all cases and in one case an additional ventrodorsal oblique view was performed. MRI of the lumbosacral area was performed under general anaesthesia using a 1.5-Tesla superconducting magnet (1.5 Tesla Signa Echospeed system, General Electric Medical Systems, Waukesha, Wisconsin) and a spine coil.

Results: The sacral end plate defect, subchondral sclesosis and/or triangular free bony fragment diagnostic for OCD was evident on the sagittal plane MR images in all cases. In two cases T2 and/or T1-weighted sagittal images were diagnostic, while in the third case a sagittal gradient echo was required to demonstrate the osseous fragment. The lack of superimposition of surrounding structures made MRI superior to survey radiography to identify and localise the bone lesions. Moreover, the presence of intervertebral disc changes, secondary cauda equina compression and foraminal stenosis was not evident on survey radiography but could readily be identified with MRI.

Discussion: Imaging modalities used in the past to investigate lumbosacral OCD possess severe limitations, particularly the superimposition of surrounding structures during radiography and the requirement for subarachnoid or epidural contrast medium injections in order to evaluate the presence or absence of cauda equina compression. Both CT and MRI avoid the superimposition of neighbouring structures, but MRI is superior to conventional CT because of its multiplanar capabilities and superior inherent soft tissue contrast. Moreover no epidural or subarachnoid contrast injection is needed to identify the presence of compressive lesions with MRI.

Conclusion: MRI allowed detailed and accurate visualisation of the lumbosacral OCD lesions in this cases series, as well as demonstrating the associated effects on the cauda equina.

Abstract #47

EQUINE HERPESVIRUS 1 INDUCED OPTIC NEURITIS AND ENCEPHALITIS IN A LLAMA. B Stierstorfer, K Matiasek, A Neubauer and W Schmahl. Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, Faculty of Veterinary Medicine, LMU Munich, Germany.

Four llamas (*Lama glama*) showed progressive loss of vision over one week until all animals became blind. Body temperatures were normal and the animals were alert. Serum samples were evaluated for antibodies against a panel of infectious diseases causing neurologic or ophthalmic disease (e.g. Borna disease virus, leptospirosis) with negative result. One pregnant female llama was euthanatized and complete necropsy was carried out.

Macroscopic investigation of the carcass revealed no gross pathological findings whereas histological examination showed moderate to severe nonsuppurative optic neuritis with vasculitis and perivascular accumulations of lymphocytes and macrophages. Axonal degeneration was occasionally observed. Both optic nerves were involved along their entire length. In the brain moderate nonsuppurative encephalitis and meningitis with abundant lymphocytic perivascular aggregates and glial reaction were visible, mainly adjacent to the optic tracts. Additionally, a large area of malacia was localized in the lateral geniculate nuclei. Staining for myelin did not show significant myelin deficits or accumulated myelin breakdown products. No histopathologic changes were evident in the left eye whereas a lymphocytic uveitis and retinal detachment was obvious in the right eye. Polymerase chain reaction (PCR) on DNA extracted from formalin fixed and paraffin embedded tissue samples of brain and optic nerve was successful in detecting Equine Herpesvirus 1 (EHV-1).

Equine Herpesvirus 1 is an alphaherpesvirus associated with abortion, respiratory disease and myeloencephalopathy mainly in horses. Infection is acquired through the mucosal epithelium of the upper airway, after contact with virus-containing secretions from the nasopharynx or reproductive tract of an infected horse or from an aborted fetus. Evidence suggests that EHV-1 is also able to cause disease in alpacas and llamas. According to the majority of published cases, blindness seems to be the predominant clinical sign in this species with only few reported cases of neurological deficits others than those associated with vision (e.g. nystagmus, head-tilt and/or paralysis). Affected llamas and alpacas appear to be dead-end-hosts, because virus usually can not be recovered from secretions or blood after the acute stage of the disease.

The most likely sources of infection with EHV-1in this case are the horses of the circus, which were housed in close contact with the llamas. EHV-1 is enzootic in most domestic horse populations and the epizootiological reservoir seems to be the large pool of latently infected, intermittently-shedding carrier horses, in which dorsal root neurons in the trigeminal ganglia serve as the cellular hosts by providing shelter from recognition and destruction by the immune system.

Abstract #48

DYSPLASTIC GANGLIOCYTOMA OF THE CEREBELLUM IN A HORSE. B. Stierstorfer, K. Matiasek and W. Schmahl. Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, Faculty of Veterinary Medicine, LMU Munich, Germany.

A 4-year-old female Frisian horse was presented for clinical evaluation of severe movement disorders. The mare was reported to have a severely ataxic gait which progressed over a period of two weeks. The horse was euthanised because of a poor prognosis.

Macroscopic inspection of the brain revealed a marked asymmetric enlargement of the cerebellum with intense convoluted and thickened folia. Normal configuration appeared only in some minor parts of the lateral hemisphere. Hematology, CSF cytology and clinical chemistry of CSF and serum were inconclusive of systemic disease. After routine fixation in 7% neutral-buffered formalin and embedding in paraffin, the cerebellar tissue was sectioned at 5 micrometers. Sections were conventionally stained with H&E, cresyl-violet and Schroeder-Woelcke-staining. Immunohistochemical examination included PCNA, GFAP and synaptophysin.

Histopathological examination of the widened folia exhibited a disorganized cerebellar architecture in which Purkinje cell bodies were rarely recognized and the border between the molecular and granular layers could not be discerned. The granular cell layer was thickened and contained large ganglion cells with clear nuclei and prominent nucleoli. The broadened molecular layer was thickened and irregularly myelinated tracts extended from the abnormal neurons in the granular layer. Severity of the findings varied from a recognizable granular cell layer including occasional large dysplastic neuronal cell bodies, to an unrecognizable granular layer occupied by a population of large neurons between the molecular layer and deep white matter. The myelinated axis of the cerebellum was atrophic and cystified. Mitoses were absent; staining with PCNA marked only some vascular endothelial cells and gitter cells. Synaptophysin indicated axosomatic synapses peripheral of dysplastic ganglion cells. GFAP stained radial Bergmann glia.

Neuropathologic findings closely resembled lesions described in human cases of Lhermitte-Duclos disease (LDD; dysplastic gangliocytoma of the cerebellum). This disease occurs in early adulthood, arises from the cerebellar cortex and is characterized by an asymmetric dysplastic enlargement of the cerebellum without clear demarcation of diseased and normal tissue and highly enlarged and distorted folia. The lesion causes progressive mass effects and is commonly associated with cerebellar dysfunction and signs of increased intracranial pressure. Only about 100 human cases have been documented and there is only one reported case in a horse. The pathogenesis is incompletely understood but 40% of LDD cases in humans are associated with Cowden's syndrome, a rare autosomal dominant multiple hamartoma syndrome. Linkage analysis has determined that a single locus within chromosome 10q23 is likely to be responsible for Cowden's disease and probably for LDD.

Abstract #49

MORPHOMETRICAL ANALYSIS OF THE EQUINE ACCESSORY NERVE. J. Tanck1, P. Gais, U. Juetting, K. Rodenacker and K. Matiasek. Chair of Neuropathology & General Pathology, Institute of Veterinary Pathology, Faculty of Veterinary Medicine, LMU Munich, Germany.

Examination of peripheral nerve biopsies is the gold standard for diagnosing peripheral neuropathies in vivo. In addition to morphological assessments, histometry has proved successful in quantification of the peripheral nerve composition and adding correlatives to clinical and electrophysiological data. Up to now, the lateral palmar nerve has been favoured as being the most suitable for morphometric investigations in the horse. The accessory nerve, however, is the preferred target for in vivo-diagnosis of equine motor neuron disease. This study was designed to test the suitability of the equine accessory nerve for quantitative assessment of PNS parameters.

Similar to the peroneal nerve, which is one of the most commonly used nerves for morphometry in small animals, the accessory nerve contains all different fiber classes, has a constant location, is easily accessible for surgical exploration and can be excised at a sufficient length. Samples of the ventral cervical accessory nerve branch were taken from 8 horses and compared to peroneal counterparts. Thereafter, digital image analysis was performed on p-phenylenediamine stained cross sections of each fascicle. All histograms of the myelinated nerve fiber (MF) diameters, fiber areas, axon areas and the myelin sheath thickness show a large similarity between the two different nerves investigated. The MF diameters of both nerves revealed a characteristic bimodal distribution with one upper peak at 4–6 μm and one lower peak at 10–12 μm. The mean MF diameter ranged from 5.01 ± 1.69 to 8.83 ± 3.95 μm. These values are comparable to those of the lateral palmar nerve reported by Wheeler (1987). The percentage of fibers larger than 7 μm in diameter varied from 11.8 % to 67.2 % in both the accessory and the peroneal nerve. The mean g-ratio per horse, which indicates the overall relationship between myelin thickness and axonal diameter, varied individually from 0.47 ± 0.05 to 0.63 ± 0.02. Thereby, the peroneal nerve seems to exhibit slightly lower values than accessory nerves. The mean myelin sheath thickness ranged from 2.18 ± 0.42 to 2.71 ± − 0.68 μm in both nerves and when plotted on histograms it shows an unimodal distribution with a peak between 1.8 and 2.2 μm.

Consistent with recent observations in other species, a tight correlation between MF diameter and myelin sheath thickness is lacking. This study proves that the equine accessory nerve is suitable for morphometric assessment, contains a representative fiber population comparable to that of the peroneal nerve, and, moreover, presents some characteristics of the lateral palmar nerve. Amongst all morphometric features MF diameters and their histograms, the histograms of fiber and axon areas, the MF density per fascicle and the g-ratio as well as the g-ratio plotted against the fiber diameter turned out to be most reliable and informative. Based on these results the accessory nerve can be highly recommended for systematic morphometric studies in the horse.

Abstract #50

''ACCORDION'' ANOMALY OF THE CERVICAL VERTEBRAE IN AN ALPACA: CLINICAL, RADIOLOGICAL AND PATHOLOGICAL FINDINGS. A. Tomek, S. Martig, C. Botteron, A. Oevermann, M. Zulauf, J. Lang and A. Jaggy. Department of Clinical Veterinary Medicine, Animal Neurology Section, Vet Suisse-Faculty of Bern, Switzerland.

A one-year-old, male alpaca was presented with an acute non-progressive onset of tetraparesis. The animal had been kept at pasture and the owner reported that there had been a lighting storm during the night before presentation. The llama was found lying on its side in the morning and the owners thought that their animal was hit by a falling tree. Initial treatment by the referring veterinarian included non-steroidal anti-inflammatory drugs. There was no improvement over the next three days and the animal was referred.

On clinical examination no abnormalities were noted. The animal was lying in sternal recumbency, but was not able to stand up without help. Severe tetraparesis was noted and proprioceptive positioning was absent in all four limbs. Hypertonicity of extensor muscles and increased patellar reflexes were evident. The flexor reflexes appeared decreased in the left thoracic limb. The neck was stiff and painful during palpation.

The neuro-anatomical localisation of the lesion appeared to between in C6 and T2 spinal cord segments. A traumatic origin of the clinical signs was placed on the top of the rule/out list including compressive lesions such as subluxation, luxation or fracture of the cervical vertebral bodies, disk protrusion or extramedullary hemorrhage. Developmental abnormalities of the cervical spinal cord and vertebrae, including subarachnoidal cysts, syringomyelia or other anomalies could not be excluded. Inflammatory/infectious diseases such as abscesses, bacterial osteomyelitis or myelitis were also included in the rule/out list.

The diagnostic work-up included a plain lateral radiographs of the cervical area. The results were compatible with multiple malformations of the vertebral bodies. Myelography confirmed dorsal and ventral compression of spinal cord at the level of C2/3, C4/5 and C6/7 and dorsal compression at C3/4. Cerebrospinal fluid examination was normal. The final diagnosis was complex malformations of the cervical vertebral bodies with acute involvement of the spinal cord.

Because of the bad prognosis and financial constraints the animal was euthanized on the owner request. A post-mortem examination confirmed the diagnosis: on histopathology, swelling of axons in all spinal cord funiculi at the level at C2 through C7 were observed. In addition, proliferation of macrophages, glial cells, capillaries and loss of neurons as well as gliosis of the gray mater confirmed an acute myelomalacia with secondary compression of the spinal cord.

Abstract #51

DANDY-WALKER LIKE SYNDROME IN AN ADULT CAT AND A KITTEN. I. Van Soens, S.F.M. Bhatti, I.M.V.L. Gielen and L.M.L. Van Ham. Department of Small Animal Medicine and Clinical Biology, Ghent University, Belgium.

Dandy-Walker syndrome in humans refers to the morphological triad of aplasia or hypoplasia of the cerebellar vermis, cystic dilatation of the fourth ventricle and hydrocephalus. Other entities are described that are related to this syndrome but do not represent the key components as listed above; they are called Dandy-Walker variants. Although onset of symptoms occurs in the majority of human patients in childhood; there are also reports of adult onset. In human patients symptoms of elevated pressure dominate, but cerebellar signs can be present. Diagnosis is based on CT images and MRI. Many surgical treatments are described: cyst excision, ventriculo-peritoneal and/or cysto-peritoneal shunting or ultrasound-guided puncture of the cyst; all with variable outcome.

In veterinary medicine Dandy-Walker syndrome is described in many species, all representing young animals varying from newborn to 6 months of age. These reports all describe the typical key components of the Dandy-Walker malformation: hydrocephalus, cystic dilatation of the fourth ventricle and hypoplasia of the cerebellar vermis. These animals were stillborn or euthanized shortly after the diagnosis had been made because of the severe progressive, neurological signs.

The present report describes a Dandy-Walker variant in an adult cat and a kitten. The cats were both presented with cerebral signs; the adult cat with involvement of the cerebellum. Diagnosis was made through CT-scan. These images showed clearly a hydrocephalus and a large fluid-filled cyst communicating with the fourth ventricle. In both cats there was no appearance of hypoplasia of the cerebellar vermis. The relevant presence of the cerebellar vermis can be missed on a transverse slice; a more definitive diagnosis should be made on MRI and necropsy.

Central nervous system signs of these cats improved with a short medical treatment with steroids. The adult cat survived four-and-a-half years after onset of clinical symptoms; the kitten is four years after diagnosis still alive with no neurological deficits.

The two cats of this report were diagnosed as Dandy-Walker variants. The CT images showed some features of Dandy-Walker syndrome but not the typical triad; more advanced imaging techniques are needed for more precise description of the anomaly in these animals.

Previously the malformation had not been reported in adult animals. As seen in these cases, the malformation is not always progressive; a long term survival is possible without the need of life-long treatment or invasive surgery.

Abstract #52

OCCURRENCE OF EUTHYROID SICK SYNDROME IN DOGS WITH IDIOPATHIC EPILEPSY PRIOR TO TREATMENT WITH ANTIEPILEPTIC DRUGS. T. von Klopmann, I.C. Boettcher, A. Rotermund, A. Tipold. Dept. of Small Animal Medicine and Surgery, School of Veterinary Medicine, Hannover, Germany.

As shown in former studies, treatment with antiepileptic drugs (AEDs) may lead to subnormal plasma thyroid hormone levels in spite of normal thyroid function. The aim of the present study was to identify an influence of idiopathic epilepsy (IE) itself on thyroid hormone levels. For this, plasma thyroid hormone values were measured in dogs with seizures prior to the administration of any anti-epileptic medication. Reference values (see table below) were obtained from a former study.

Forty-eight dogs with seizures were divided into 3 groups: IE without pretreatment (group A, n = 26), IE with pretreatment (group B, n = 14) consisting of AEDs or steroids and secondary epilepsy (group C, SE, n = 8). Diagnostic work up consisted of complete blood work, T4 and cTSH level measurement, cerebrospinal fluid (CSF) analysis and magnetic resonance imaging (MRI). IE was diagnosed in dogs in which clinical and neurological examination, blood and CSF analyses and MRI were within normal limits. Seizure history was reviewed concerning time range between last seizure and thyroid hormone level measurement.

Patients' median age was 3.5 years (range 0.5–13 years). Median time from last seizure to T4/cTSH measurement was 48.5h (range 2–1224h). The highest percentage of euthyroid sick patients was found in the pretreated IE group (43%, see table below). In IE without any treatment euthyroid sick syndrome was found in 11.5% of the cases. However, low T4 levels suspicious of euthyroid sick syndrome occurred in half of the cases. All cases diagnosed as euthyroid sick or suspicious euthyroid sick had normal cholesterol values and showed no clinical signs of hypothyroidism. The period between the last seizure and the thyroid hormone sampling as well as the age of the dogs seemed to have no influence on thyroid level distribution in the 3 groups. The two IE groups did not differ statistically significantly concerning the T4 levels ($P>0.05$). In summary, euthyroid sick syndrome was documented prior to antiepileptic treatment in this presented group of epileptic dogs. It must be considered that epileptic activity alone can influence thyroid hormone levels. Therefore, the incidence of the euthyroid sick syndrome in epileptic dogs due to AED administration may be lower than expected.

	A	B	C
Euthyroid sick (T4: $<1.1\mu$g/dl, cTSH $<$ 0.3ng/ml)	3/26	6/14	2/8
Suspicious euthyroid sick (T4: 1.1–1.6μg/dl, cTSH $<$ 0.3ng/ml)	13/26	2/14	1/8
Hypothyroid (T4: $<1.1\mu$g/dl, cTSH $>$ 0.3ng/ml)	0/26	0/14	1/8
Normal (T4: 1.7–4.5 μg/dl, cTSH $<$ 0.3ng/ml)	10/26	6/14	4/8

Abstract #53

BRAIN AND SPINAL CORD HAEMORRHAGE ASSOCIATED WITH *ANGIOSTRONGYLUS VASORUM* INFECTION IN FOUR DOGS. A. Wessmann, D. Lu, C.R. Lamb, B. Smyth, P. Mantis, K. Chandler, G.B. Cherubini and R. Cappello. The Royal Veterinary College, Hertfordshire, United Kingdom.

Angiostrongylus vasorum (French heartworm) is a metastrongyloid nematode parasite that infects the pulmonary arteries and the right ventricle of wild and domestic canids, in particular, dogs and foxes. The infection can be asymptomatic or proceed to severe disease or death caused by a combination of coagulopathy, respiratory and cardiac dysfunction. Central nervous system (CNS) signs are common. Haemorrhage or embolism in the spinal cord or in the brain are usually identified at post mortem examination.

Multifocal CNS haemorrhage associated with *Angiostrongylus vasorum* infestation was diagnosed in 4 dogs by magnetic resonance imaging (MRI). A 2 year old male neutered Staffordshire bull terrier (dog 1) was presented with an 8-day history and a 13-month-old male Scottish terrier (dog 2) with a 5 day history of progressive paresis and spinal pain. An 8 year old Labrador retriever (dog 3) presented with neck pain and seizures and a 9 month old male entire Bulldog (dog 4) showed head pressing and depression. Haemorrhagic lesions were found in the lumbar and the cervicothoracic spinal cord and in the brain respectively. Erythrophagocytes and marked elevation of protein were seen in the spinal fluid of 3 dogs suggesting cerebrovascular disease. Faecal analysis by the Baermann method revealed 60, 11 and more than 2500 *A. vasorum* larvae respectively, but was not performed in the 4th case. Histological examination of the CNS performed in dog 2 and 4 correlated well with the lesions seen on MRI and confirmed multifocal haemorrhage caused by *A. vasorum* infection.

MRI was very sensitive in imaging CNS haemorrhage, in particular the gradient echo (GRE) sequences. Recent haemorrhage contains deoxyhaemoglobin, a paramagnetic substance, causing low signal intensity in GRE images and therefore a hypointense appearance. The olfactory bulb was affected in dogs 2, 3 and 4 and may be a predilection site for intracranial haemorrhage caused by *A. vasorum.* Initial myelographic investigation of the spinal lesions in dog 1 and 2 determined the intramedullary localisation of the lesion. Intramedullary contrast accumulation as seen in dog 1 was suggestive of myelomalacia. This study emphasizes that MRI appears to be superior to myelography and can distinguish haemorrhage from necrosis, since GRE images show a signal void in haemorrhagic lesions indicating a magnetically susceptible substance. This information is helpful for both diagnosis and prognosis. If only myelography is available, the veterinary surgeon should consider that haemorrhage due to *A. vasorum* infestation is a differential diagnosis for intramedullary lesions.

A. vasorum infestation should be considered as a differential diagnosis in haemorrhagic CNS lesions. Magnetic Resonance imaging, in particular, the GRE sequence, is a useful tool to diagnose haemorrhagic lesions in the CNS due to *A. vasorum* infestation.

Abstract #54

DYSTROPHIN-DEFICIENT MUSCULAR DYSTROPHY IN AN OLD ENGLISH SHEEP DOG. L.A. Wieczorek, L.S. Garosi and G.D. Shelton. Animal Health Trust, Lanwades Park, Newmarket, Suffolk, England, UK.

Muscular dystrophies (MD) are a heterogeneous group of inherited, degenerative mostly non-inflammatory disorders characterized by progressive muscle weakness and wasting. In companion animals, dystrophies associated with an absence of or abnormality in dystrophin, sarcoglycans, and laminin $\alpha 2$ have been recognized. Dystrophin-deficient MD and mutations of the dystrophin gene is the most common and best-studied MD in dogs, and has been reported in several breeds. To the authors' knowledge, this is the first case of dystrophin-deficient MD reported in an Old English Sheep Dog.

A-three-months old, male Old English Sheep Dog was presented with a few weeks history of difficulty eating, excessive drooling, mild exercise intolerance and impaired growth. Physical examination confirmed markedly undersize and underweight body condition, with atrophy of the temporalis, paraspinal and distal limb muscles. Marked swelling of the tongue and pharyngeal region was evident. Neurological findings included a stiff gait with an increased muscle tone of both hind limbs, and decreased to absent withdrawal reflexes in all four limbs. Tongue movement was decreased, and the gag reflex was absent. Haematology and serum biochemistry were normal apart from a marked elevation of serum creatine kinase activity. Serum antibody-titres for *Toxoplasma gondii* and *Neospora caninum* were negative. Needle electromyography was abnormal on all four limbs, paraspinal, masticatory, tongue and laryngeal muscles. Pathological changes within muscle biopsy specimens were dystrophic in nature. Immunohistochemical staining for dystrophin was absent. Considering the rapidly progressing deterioration, the poor prognosis, and the absence of a specific therapy for dystrophin-deficient MD, the owners elected for euthanasia.

Dystrophin deficient muscular dystrophy in dogs is X-linked and comparable to Duchenne muscular dystrophy in humans. Isolated non-inherited cases may occur as a result of new mutations, since the dystrophin gene is exceptionally large and, therefore, likely to be target for mutations. Littermates and parents of this dog appeared to be unaffected, nevertheless, no genetic analysis have been performed. The question of whether or not this dog was an inherited or isolated case of dystrophin-deficient muscular dystrophy remains open.

Abstract #55

MAGNETIC RESONANCE IMAGING IN THE DIAGNOSIS OF UNILATERAL LAMENESS IN A CHICKEN (*GALLUS DOMESTICUS*) . J. Woolley, G. Skerritt and I. Elliott. Cranmore Veterinary Services and VetMRI, 140 Chester Road, Childer Thornton, South Wirral, CH66 1QN, UK.

A three-year old Speckled Sussex hen was presented for investigation of a gradual but progressive onset of unilateral paresis of the right leg. The intimate association between the lumbosacral plexus and the kidney is an indication for the application of a study by magnetic resonance imaging. Multiple nodules of solid tissue were revealed within both kidneys together with a large lesion in the musculature of the thigh. Samples of the abnormal tissues were obtained post mortem and histopathological examination showed them to be lymphosarcomatous, suggestive of a diagnosis of Marek's disease.