FRIDAY 26-9-08

8.00–8.30  Registration
8.30–8.45  Welcome message from the President and the Organizing Committee-Commencement of meeting
8.45–9.30  Chairperson: Dr. Zoe Polizopoulou
Invited speaker
G. Diane Shelton – Autoimmune disorders of muscle and the neuromuscular junction
9.35–10.20  Invited lecture
Christiane Massicotte – Anatomy/physiology of the peripheral nerve at the molecular level
10.25–10.55  Coffee break – Poster exhibition
10.55–11.40  Invited speaker
Stephanie Blot – Congenital neuromuscular disease-an update on clinical and molecular data
11.45–12.45  Oral presentations
Chairperson: Dr. Jacques Penderis
11.45–12.00  Current illness variant of inflammatory demyelinating neuropathy in cats with systemic diseases-K. Matiasek, L. Matiasek, B. Wolff, S. Kuhne-Velte, A. Fischer, W. Schmahl
12.00–12.15  Development of new efficient and noninvasive tools to evaluate muscle function and the efficacy of therapy in a canine model of Duchenne Muscular Dystrophy (DMD)-C. Esciou, T. LeQuang, O. Pouyol, S. Blot, E. Viguier
12.15–12.30  Histological and ultrastructural evidence that recurrent laryngeal neuropathy is a bilateral mononeuropathy-C. Hahn, K. Matiasek, P. Nixon, I. Mayhew
12.30–12.45  Degenerative myelopathy in Chesapeake Bay retrievers-S. Long, P. Henthorn, J. Serpell, T. Van Winkle, J. Coates
12.45–14.00  Lunch break
14.00–14.45  Chairperson: Luisa De Risio
Invited lecture
G. Diane Shelton – Emerging syndromes with muscle cramps, fasciculations and collapse
14.50–16.05  Oral presentations
Chairperson: Dr. Jacques Penderis
14.50–15.05  Prognostic factors affecting survival to discharge and long-term outcome in dogs with megaesophagus-A. Thomson, I. Ramsey, E. Courcier, R. Bell
15.05–15.20  Dystrophin deficient muscular dystrophy in a family of harlequin miniature poodles-A. de Stefani, E. Beltran, L. De Risio, G.D. Shelton
15.20–15.35  Myotonia-like findings in three dogs affected by Cushing’s syndrome while on treatment with triostane-F. Gernone, F. Fracassi, E. Bianchi, L. Pisoni, C. Cantile, G. Gandini
15.35–15.50  A mitochondrially inherited disorder in golden retrievers presenting as a sensory ataxic neuropathy-K. Hultin Jaederlund, I. Baranowska, E. Oervind, K. Matiasek, R. Wibom, I. Nennesmo, N.-G. Larsson, G. Andersson, L. Andersson, A. Hedhammar
15.50–16.05  Syringomyelia in asymptomatic Cavalier King Charles spaniels does not affect sensory evoked potentials-T. Harcourt-Brown, N.D. Jeffery, N. Granger
16.05–16.30  Coffee break – Poster exhibition
16.30–18.00  Oral presentations
Chairperson: Andrea Tipold
16.30–16.45  Pathology meets artefact-diagnostic challenges in teased nerve fibres-L. Matiasek, W. Schmahl, K. Matiasek
16.45–17.00  The use of magnetic resonance imaging in pre-clinical trials of Duchenne muscular dystrophy in GRMD dogs-J.L. Thibaud, D. Bertoldi, I. Barthélymy, S. Fleury, S. Blot, P.G. Carlier.
17.00–17.15  “The robotic cats”: Slowly progressive meningoencephalomyelitis of suspected viral aetiology in 15 adult cats-L. De Risio, R. Brown, B. Tennant, L. Matiasek, A. de Stefani, A. Sparkes, K. Matiasek.
17.15–17.30  Regional brain perfusion in 12 epileptic dogs evaluated by 99mTc-ECD single photon emission computed tomography (SPECT)-V.A. Martlé, K. Peremans, K. Audenaert, S. Vermeire, S.F.M. Bhatti, I. Gielen, I. Polis, L.M.N. van Ham
17.30–17.45  Immunophenotyping of lymphocytes in canine granulomatous meningoencephalitis-C. Mariani, A. Reynolds, K. Munana, T. Brown
17.45–18.00  Neurological findings and MRI in forty-six dogs with vascular lesions in the brain-H. Gredal, M. Berendt, G. Skerritt
18.00 ESVN Annual General Meeting
21.30 Gala Dinner
SATURDAY 27-9-08
9.00–9.45 AM Chairperson: A.F. Koutinas
Invited speaker
Stephane Blot – Cell and gene therapy in muscular dystrophy
9.50–10.35 Invited speaker
Christiane Massicotte – Genetic demyelinating diseases of the peripheral nervous system
10.40–11.10 Oral presentations
  ● “Stiff-dog” syndrome in three related young male Labrador retrievers-N. Granger, T.R. Harcourt-Brown, G.D. Shelton, W. Blakemore, N.D. Jeffery
  ● Crazy cases: Meerkat-like posture in two young pug dogs-I.C. Boettcher, K. Matiasek, G. Oechtering, T. Flegel
11.10–11.40 Coffee break – Poster exhibition
11.40–13.40 Dr. Caroline Hahn
Oral presentations
11.40–11.55 Palatolingual myokymia in a hydrocephalic Maltese dog-A. Vanhaesebrouck, S.F.M. Bhatti, V. Bavegems, I. Gielen, G. Vercauteren, I. Polis, L. Van Ham
11.55–12.10 Clinical, electrodiagnostic, magnetic resonance imaging and histological findings in a dog with necrotizing myopathy-L. De Risio, F. McConnell, A. de Stefani, L. Matiasek, A. Lujan Felius-Pascual, G.D. Shelton
12.10–12.25 Clinical, electrodiagnostic, magnetic resonance imaging and pathological features of beta-mannosidosis in a Saint Bernard dog-A. Lujan Felius-Pascual, G.D. Shelton, J. Stone, A. Blunden, A. Holloway, K. Matiasek
12.40–12.55 Orthostatic tremor in a Weimaraner-P. Montoliu, L. De Risio, E. Beltran, J. Freeman
12.55–13.10 Is traditional CSF analysis the gold standard for confirming CNS inflammation? Evidence from CSF IL-6 concentrations-M. Lowrie, J. Penderis, D.F. Lappin, T.J. Anderson
13.10–13.25 Autoimmune myasthenia gravis in a ferret-H. Minh, J. Couturier, D. Boussarie, L. Cauzinille, G.D. Shelton
13.30–14.00 Closing remarks-awards
PALATOLINGUAL MYOKYMIA IN A HYDROCEPHALIC MALTESE DOG. A. Vanhaesebrouck1, S.F.M. Bhatti1, V. Bavegems1, I. Gielen2, G. Vercauteren2, T. Polis1, L. Van Ham1. 1Department of Small Animal Medicine and Clinical Biology, 2Department of Medical Imaging and 3Department of Veterinary Pathology, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

A 9-year-old male Maltese dog presented with an 8-month history of exertional dyspnea with inspiratory stridor and cyanosis. On neurologically examination, vermiform involuntary tongue and soft palate contractions, as well as a mild ataxic gait were observed. Needle EMG demonstrated myokymic discharges in the palatal and lingual muscles. Brain CT scan showed dilation of all ventricles. Since there was no history of prior radiotherapy – a regular cause of focal myokymia in humans, nor suprasellar brainstem compression, a pathogenic link between myokymia and the pituitary tumour in this case was deemed unlikely. As no other lesions were found, it was hypothesized that the hydrocephalus potentially led to a mechanical stretching of the cortico-nuclear fibres, as previously suggested in a case of facial myokymia in a hydrocephalic man. This might explain the desinhibition of the hypoglossal and/or ambiguous brainstem nuclei in our case. The primary cause of the hydrocephalus remains unexplained.

Palatal lingual myokymia is a rare condition in humans and has not yet been associated with hydrocephalus. Apart from one report of “idiopathic” facial myokymia in a puppy, this is the only other description of genuine focal myokymia in animals. Palatal lingual myokymia should be taken into consideration as an exceptional cause of upper airway complaints in dogs.

AUTOIMMUNE MYASTHENIA GRAVIS IN A FERRET. J. Couturier1, M. Huynh1, D. Boussarie1, L. Cauzinnelle1, G.D. Shetton2. 1Centre Hospitalier Vétérinaire Frégis, Arcueil, France, 2Department of Pathology, University of California, San Diego, La Jolla, CA, USA.

A 7-month-old male ferret was evaluated for episodic pelvic limb paresis of 2-weeks duration. On neurological examination, non-ambulatory flaccid tetraparesis with decreased spinal reflexes suggested a neuromuscular disease. Blood analysis, cerebrospinal fluid analysis, thoracic and abdominal radiographs and abdominal ultrasound examination were unremarkable. Electromyographic study showed subtle spontaneous activity in hind limb interosseous muscles only. On electromyography, motor nerve conduction velocities were normal and compound muscle action potential amplitude was slightly decreased in the tibial nerve. A severe decremental response of the compound muscle action potential was found on repetitive nerve stimulation (45.5% at the 3rd ulnar nerve stimulation). Intravenous neostigmine administration led to immediate remission of neuromuscular signs. Cross-reacting acetylcholine receptor (AChR) antibodies were detected in the serum (0.35 nmol/l) using a canine and feline specific muscle extract. Although a reference range has not yet been established for the ferret in this assay system, the AChR antibody titers in 5 clinically normal ferrets were all inferior to 0.06 nmol/l. Treatment with oral pyridostigmine bromide (1 mg/kg, q 8 h) resulted in a complete remission. However, the ferret was euthanized one month later because of recurrence of weakness.

To the authors’ knowledge, this is the first report of acquired MG in the ferret and the first identification of AChR antibodies in this species. Autoimmune MG should be considered in this species when generalized weakness and flaccid paraparesis suggest a neuromuscular disease. Electrophysiological studies, intravenous anticholinesterase challenge and AChR antibody titer determination are helpful for diagnosis of this condition.

CLINICAL, ELECTRODIAGNOSTIC, MAGNETIC RESONANCE IMAGING AND HISTOLOGICAL FINDINGS IN A DOG WITH NECROTIZING MYOPATHY. Luisa De Risio, Fraser McConnell, Alberta de Stefani, Lara Mattiassè, Alejandro Lujan Feliu-Pascual, G. Diane Shelton. The Animal Health Trust, Newarket, Suffolk, UK.

A 10-year-old male Old English sheepdog presented with a 5 day history of acute onset non-ambulatory tetraparesis and severe cervical hyperalgesia. General physical examination showed diffuse osteoarthrosis. Neurological examination revealed non-ambulatory tetraparesis, normal mental status and cranial nerve function. Proprioceptive positioning was delayed in all four limbs. Flexor reflexes were severely decreased in the thoracic limbs and slightly decreased in the pelvic limbs. Patellar reflex was absent bilaterally. Cutaneous trunci reflex was normal. Palpation of the caudal cervical region elicited hyperaesthesia. Serum creatine kinase (CK) was 2437 IU/L. Serology for Toxoplasma gondii and Neospora caninum was unremarkable. Needle EMG in both dogs was negative. MRI of the cervical and cervicothoracic regions showed severe

and AST, urine analysis and thoracic radiographs which were all unremarkable. Biopsy of cranial tibial muscle and peroneal nerve including ultrastructural examination in dog 1 showed no pathologic changes. In vitro electrophysiological testing for congenital myasthenia gravis was not performed. The owner of dog 2 declined any biopsy.

Symptomatic treatment with pyridostigmine resulted in improved muscular strength in both dogs. When supported by a leash dog 1 was able to walk for 20 min. Both dogs still showed the meerkat-like posture when sitting.

There are no reports on similar cases in dogs in the literature. A chipmunk-like posture in Sphynx cats with assumed muscular dystrophy is mentioned (Vet Clin Small Anim 34 (2004), 1307–1359). We suspect an unidentified neuromuscular disease responsive to pyridostigmine treatment in our dogs. Alternatively, an inherited ion channel disorder might also be considered.
changes consistent with necrosis and focal hemorrhage within the serratrs ventralis, subscapularis, supraspinatus and rhomboideus muscles. Electromyography revealed widespread fibrillation potentials, positive sharp waves, and complex repetitive discharges. Biopsies from the serratus ventralis, rhomboideus, trapezius, and biceps femoris muscles showed multifocal areas of extensive myonecrosis with the serratrs ventralis, subscapularis, supraspinatus and rhomboideus muscles, and numerous singular necrotic fibers within the other two muscles. Other than macrophages cleaning up necrotic debris, cellular infiltration was not observed.Histology for myoglobin was negative. Aerobic anaerobic bacterial culture of muscle tissue was negative. The dog recovered with nursing care and physiotherapy and was ambulatory unassisted 2 months after onset.

Necrotizing myopathy has been reported in humans following exposure to toxins such as snake and insect venoms, and drugs including haloperidol, and diabetes, and in horses due to Streptococcus equi infection, plant toxicities, and colic-associated endotoxanosis. In a review of canine biopsies received over the past 2 years (GDS), 22 cases had a histologic diagnosis of necrotizing myopathy. Many breeds were affected and ages ranged from 1–13 years. Onset of clinical signs was acute, there was no history of previous episodes, serum CK activities were moderately to markedly elevated, and most had myoglobinuria. All dogs recovered with supportive care in 2–4 weeks. As it occurred in this case, a triggering or predisposing factor for myonecrosis was not be identified based on the history and the results of diagnostic investigations. Clinicians should be alerted to the existence of this spontaneously resolving necrotizing myopathy.

**THE ROBOTIC CATS**: SLOWLY PROGRESSIVE MENGLEANCEPHALOMYELITIS OF SUSPECTED VIRAL AETIOLOGY IN 15 ADULT CATS, Luisa de Rasio, Richard Brown*, Bryn Tennant, Lara Mattiasek, Alberto de Stefani, Andy Sparkes, Kaspar Mattiasek. The Animal Health Trust, Newmarket, UK. A group of cats in Sweden, however it is still uncertain if cats reported with “staggering disease” in other geographic areas were all affected by the same agent.

Eleven cats presented with a history of slowly progressive (mean 11 months) behavioral changes, disorientation, and ataxic rigid (“robotic”) gait between 2001 and 2008. All cats had outdoors access and lived in the same geographic rural area. Mean age at onset of signs was 9 years. Thirteen cats were domestic short hair and 2 were Persian cross. Nine were male and 6 were female. Neurological examination revealed depression, disorientation, spastic and ataxic gait, stiff extended tail, increased muscle tone and decreased to absent proprioception in all four limbs. Hematology and serum biochemical analysis (including bile acids) did not reveal any specific abnormalities. Serology for feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV) was negative in all but 1 cat that was FIV positive. Serology for BDV was performed in one cat and results were negative. MRI of the brain and spine, cisternal CSF analysis, CSF PCR for Toxoplasma gondii, Neospora caninum and Canine Distemper virus, electromyography (EMG), motor nerve conduction velocity, and muscle biopsy. The affected dog and the unaffected dog underwent only a metabolic screening. MRI, CSF analysis and PCR were normal. Serum creatine kinase (CK) was markedly elevated in the 3 affected dogs. EMG revealed fibrillation potentials and complex repetitive discharges in the infraspinalus, supraspinatus and epaxial muscles of the cranial thoracic spine. Nerve conduction velocity was normal. Muscle biopsies were obtained from the infraspinalus, supraspinatus and epaxial muscles at the level of T3 vertebral body in both dogs. Biopsies consistently revealed large areas of myonecrosis with several small basophilic regenerating fibres and scattered calcific deposits. Frozen muscle biopsy sections were examined for histochemistry, immunohistochemistry, RNA-virus infection. IHC has ruled out a number of pathogens and spongiform encephalopathy could be excluded histologically. Further work is in progress to attempt identification of the causative agent.

**DYSTROPHIN DEFICIENT MUSCULAR DYSTROPHY IN A FAMILY OF HARLEQUIN MINIATURE POODLES**, Albert de Stefani, Luisa De Rasio, G. Diane Shelton, Thong LeQuang. Université Paris XI, UPSP 2007-03-135 RT12B, Ecole Nationale Vétérinaire de Lyon, Marcy l’Etoile, France. *Laboratoire de neurobiologie, Ecole Nationale Vétérinaire d’Alfort, Maisons-Alfort, France.

Dystrophic deficient muscular dystrophy is a hereditary, x-linked, recessive degenerative myopathy that typically affects male individuals. Canine X-linked muscular dystrophy (CXMD) is a homologue of human Duchenne muscular dystrophy (DMD). Numerous dog breeds with CXMD have been characterised phenotypically, however only a few have been studied at the molecular level. This report describes the clinical presentation, histopathological findings and immunohistochemical analysis in a family of harlequin miniature poodles affected with CXMD.

Four related male, miniature poodles were assessed between March 2007 and June 2008. Dogs had a histology of poor developement, reluctance to move and progressive atrophy that was particularly severe at the level of the infraspinatus and supraspinatus muscles. One dog was clinically normal. Two of the three clinically affected dogs underwent extensive investigation including metabolic screening, MRI of brain and cervical spine, CSF analysis, CSF PCR for Toxoplasma gondii, Neospora caninum and Canine Distemper virus, electromyography (EMG), motor nerve conduction velocity, and muscle biopsy. The third affected dog and the unaffected dog underwent only a metabolic screening. MRI, CSF analysis and PCR were normal. Serum creatine kinase (CK) was markedly elevated in the 3 affected dogs. EMG revealed fibrillation potentials and complex repetitive discharges in the infraspinatus, supraspinatus and epaxial muscles of the cranial thoracic spine. Nerve conduction velocity was normal. Muscle biopsies were obtained from the infraspinatus, supraspinatus and epaxial muscles at the level of T3 vertebral body. Biopsies consistently revealed large areas of myonecrosis with several small basophilic regenerating fibres and scattered calcific deposits. Frozen muscle biopsy sections were incubated with monoclonal antibodies against the rod and carboxyl terminus of dystrophin. Sarcolemm staining was absent with both antibodies supporting a diagnosis of CXMD. One dog was euthanised at 19 months of age due to liver failure. The other two affected dogs still retain reasonable quality of life.

The molecular basis of CXMD has been described only in the Golden Retriever. Rottweiler and German short-haired pointer breeds and each breed seems to have a unique mutation in the dystrophin gene. DNA has been collected from the 3 affected and 3 related normal miniature poodles. Genetic evaluation and immunoblot analysis are in progress.

**DEVELOPMENT OF NEW EFFICIENT AND NON INVASIVE TOOLS TO EVALUATE MUSCLE FUNCTION AND THE EFFICACY OF THERAPY IN A CANINE MODEL OF DMD**, Catherine Escriou, Thong LeQuang, Olivier Pouyol, Stéphane Becc, Eric Vigneron. Université Paris XI, UPSP 2007-03-135 RT12B, Ecole Nationale Vétérinaire de Lyon, Marcy l’Etoile, France. "Laboratoire de neurobiologie, Ecole Nationale Vétérinaire d’Alfort, Maisons-Alfort, France.

Duchenne Muscular Dystrophy is an always lethal X-linked muscle wasting disease. Due to its homologous phenotypic expression with human disease grmd dog is used to evaluate therapeutic strategy. To be able to produce objective evaluation, a wide battery of tests must be available to propose a functional recovery score. The aim of our study was to test a new non invasive method to determine their value for assessment of muscle function in grmd: 1) Kinetic analysis of the gait with a walkway performance platform (Gait Four®), 2) Bone density analysis (dual-energy x-ray absorptiometry). 3) Joint angles measures (manuel goniometer). 4) young adult dystrophic dogs and 2 young adult golden retriever have been tested during a 3 months period to evolution of dystrophy. A new tool type is expected for dystrophic dogs this age. Clinical score
realisation, and creatine kinase dosage, two already validated parameters, have been regularly performed to follow the dystrophic phenotype of the dogs; 2 displayed a severe phenotype and a moderate one. All biomechanic’s parameters tested demonstrated their reliability and repeatability. Gait Fourier analysis revealed specific qualitative locomotion pattern and quantitative abnormalities such as speed decrease with stride length reduction, weight displacement on forelimbs and asynchronism of the gait between forelimbs and hindlimbs for all dystrophic dogs. Walking cadency increased in dystrophic dogs with a moderate form and decreased in the severe form. Walking angles reduction was obvious only for severely affected dogs. Bone densitometry revealed significant osteopenia in dystrophic dogs. Joint angles changes were significant and discriminators for shoulder, carpal and tarsal joints. This preliminary study demonstrated the relevance of the biomechanic’s tools especially the Gait Fourier® and bone density analysis. Further studies are currently conducted to establish reference values and to evaluate their variation during the natural course of the disease.

MYOTONIA-LIKE FINDINGS IN THREE DOGS AFFECTED BY CUSHING’S SYNDROME WHILE ON TREATMENT WITH TRILOSTANE. Floriana Gernone1, F. Fracassi1, E. Bianchi2, L.fa

Purpose of this report is the description of the neuromuscular changes in three dogs affected by hypercortisolism and on treatment with Trilostane.

Typical presentation of dogs affected by Cushing’s syndrome include polyuria/polydipsia, alopecia and enlarged abdomen. A myopathy which resembles that of exogenous steroid myopathy, characterized by muscle atrophy and weakness, has been described in dogs with spontaneous hypercortisolism. Dogs affected by Cushing’s syndrome rarely develop clinical myotonia. This condition has been described in 5 out 800 dogs in one report. Hypercortisolism is considered well controlled if the clinical signs improve and serum cortisol post ACTH stimulation range between 1.0–5.0 μg/dl.

Three female dogs (two mixbreed and one Poodle) ageing between 8 and 12 years old were referred for neurologic consultation because of progressive hind limbs stiffness and difficulty to walk. All three dogs were on therapy with trilostane (dosage: 6.3, 3.3 and 5.6 mg/kg SID PO, respectively) since at least 9 months because of previously diagnosed pituitary dependent hypercortisolism (PDH).

At the time of presentation for the neuromuscular complains, the typical clinical signs of the endocrinopathy had disappeared and the Cushing’s syndrome was considered under control (post ACTH stimulation cortisol levels were 3.9, 4.4, and 2.5 μg/dl, respectively).

Neurological signs were similar in all dogs and were characterized by hind limbs stiffness, hypometria, and hypertonus that, in two dogs, prevented evaluation of the spinal reflexes and passive flexion movements of the hind limbs. Two dogs showed characteristic “bunny hopping” gait.

Normal CBC and mild abnormalities were recorded in the serum biochemical profile (mild elevated CK and SAP) of all dogs. Serology for Neospora and Toxoplasma was negative. EMG was performed in one dog and showed generalized abnormal activity consisting in complex repetitive discharges, particularly severe in the hind limbs and proximal appendicular muscles. Sciatic-tibial nerve motor conduction velocity was decreased (42.2 m/sec.). Semimembranosus and lateral vastus muscle biopsy, performed in two dogs, showed variation in fibers size with angular and atrophic fibers associated with moderate perimisial and endomisial fibrosis. Peroneal nerve biopsy showed continuous motor unit activity (of normal shape) in resting muscles (axial and proximal limbs muscles). An increase in the frequency of the continuous motor unit activity was recorded in the tibial cranial muscle during stimulation of the peroneal nerve of one conscious dog. Under general anaesthesia, EMG in two dogs was silent. Nerve conduction studies, including measurement of motor and sensory nerve conduction velocities, late potential studies (F wave) and repetitive stimulation were all normal. Frozen muscle biopsy sections, evaluated using a standard panel of histochemical stains and enzyme reactions, revealed mild reduction of type 2 fibre size. The purpose of the present study was to investigate vascular abnormalities detected on clinical or neurological examination.

Three one year old male Labrador Retrievers were presented to our clinic for abnormal gait, rigid locomotion and frequent falls that had progressed over six months. Two of the dogs had the same sire but were from different litters. All three dogs originated from the same breeder. All dogs exhibited marked stiffness at rest and when walking, with axial rigidity and marked reduction of joint flexion. The pelvic limbs were more affected than the thoracic limbs. The dogs were not able to rise from recumbency without assistance. There was increased tone and mild muscle hypertrophy, especially in the axial and proximal limb musculature but no other abnormalities were detected on clinical or neurological examination.

Conscious electromyography (EMG) revealed abnormal continuous motor unit activity (of normal shape) in resting muscles (axial and proximal limbs muscles). An increase in the frequency of the continuous motor unit activity was recorded in the tibial cranial muscle during stimulation of the peroneal nerve of one conscious dog. Under general anaesthesia, EMG in two dogs was silent. Nerve conduction studies, including measurement of motor and sensory nerve conduction velocities, late potential studies (F wave) and repetitive stimulation were all normal. Frozen muscle biopsy sections, evaluated using a standard panel of histochemical stains and enzyme reactions, revealed mild reduction of type 2 fibre size. The proportion of type I to type II fibres was similar to control muscle. Peroneal nerve biopsy in one case was normal. Finally, creative kinase activity and thyroid hormones levels were normal. Treatment trials with various drugs including diazepam, dantrolene, potassium bromide, mexiletine and phenobarbitone were unsuccessful.

These clinical investigations have led us to localise the disease to the central nervous system. One hypothesis would be a lack of inhibition of the lower motor neurons by descending motor pathways or brain extrapyramidal pathways, as described in human diseases that present with Parkinsonian syndromes. The main argument for this conclusion is the abolition of the spontaneous motor unit firing under general anaesthesia. In humans and in horses, a ‘Dystonia’ syndrome is described that shares many similarities with the clinical presentation in the Labrador Retrievers. However, affected people respond to GABAergic drugs, which wasn’t the case in our dogs.

NEUROLOGICAL FINDINGS AND MRI IN FORTY-SIX DOGS WITH VASCULAR LESIONS IN THE BRAIN. Hanne Gredal1, Mette Berendt1 and Geoff Skerritt2, 1Dept. of Small Animal Clinical Sciences, Faculty of Life Sciences, University of Copenhagen, Denmark/ChesterGates Referral Hospital, Chester, England.

Vascular lesions (infarcts, haemorrhages and subarachnoid haemorrhages) in the brain are increasingly recognised, by the use of advanced neuroimaging, as a cause of various neurological signs in dogs. The purpose of the present study was to investigate vascular accidents in the canine brain with regard to the distribution of lesions To justify the neuromuscular signs in spite of adequate therapy, three hypothesis may be raised.

One involves the possible direct muscular toxicity of the trilostane after some months of therapy. Another possible hypothesis is a pathologic action of cortisol precursors. Some studies show that in treated dogs discrete cortisol precursors (17alpha-OH-pregnenolone and 11-deoxycortisol) increase during trilostane therapy. A pathologic “cortisol-like” action of such precursors in the muscle can not be excluded. Finally, another possibility may be the inadequate control over time of the hypercortisolism state. Some studies advise to give trilostane twice daily to allow a persistent normocortisolemia state considering that the single oral dosage couldn’t actively prevent the fluctuation of cortisol plasmatic levels, particularly after several hours from administration.

Further observations are required to confirm these hypotheses.
A retrospective study of 46 dogs diagnosed with vascular accidents at ChesterGates Referral Hospital, UK (November 2004 till November 2006) was carried out. Medical records were reviewed and neurological signs and paraclinical results (e.g. blood work and cerebrosensial fluid) recorded. Magnetic resonance imaging was performed and reviewed in all cases. Statistical analysis was performed using SAS Enterprise 9.1.

Seventeen dogs (37%) were diagnosed with brain infarcts, 25 (54%) with haemorrhages, and one dog (2%) with a subarachnoid haemorrhage. In three dogs (7%) the lesion could not be categorised. Thirtyseven (80%) of the cases had lesions in the cerebrum, two (4%) in the brainstem, four (9%) in the cerebellum, one (2%) in the subarachnoid space, and two (4%) had lesions in both the cerebrum and the brain stem.

The predominant neurological signs were seizures, reported in 24 dogs (54%), followed by ataxia noted in 17 (40%). Other signs included altered mentation, inability to stand, proprioceptive deficits, dysmetria, circling, head tilt, visual deficits, nystagmus and various cranial nerve deficits.

The overall mean age was 7.15 years (Min: 0.75 years, Max: 17 years). There was no statistically significant difference in mean ages between dogs with infarcts (7.9 years) and haemorrhages (7.1 years) (p = 0.47) (t-test procedure, SAS 9.1).

In conclusion, brain infarcts and haemorrhages appear to occur in dogs at all ages. In the present referral-based study a mean age of approximately 7 years was found. Seizures and ataxia were the predominant clinical signs in this study.

A neurological syndrome presenting as a sensory ataxic neuropathy has been observed in golden retrievers. Affected dogs were related at the maternal lineage, strongly implicating a mitochondrial inheritance.

Clinical and neurologic status, electrophysiology and pathology were assessed. We also reviewed the pedigree, analysed the mitochondrial genome and performed functional analyses of mitochondria.

Clinical signs had a slowly progressive course with onset in puppyhood. Affected dogs displayed ataxia, decreased postural reactions and spinal reflexes, without apparent muscle atrophy. Blood work, spinal radiography and electrophysiology of motor systems were all within reference values. Sensory nerve conduction differed significantly from controls. Necropsy revealed a chronic progressive sensorimotor axonopathy; within the CNS the proprioceptive pathways were most severely affected and peripheral nerve damage became most obvious in large myelinated A(alpha)-fibres. Measurements of ATP-production and respiratory chain enzyme activity combined with electron microscopic assessment of muscle mitochondria detected impaired function and changes in structure. All confirmed and retrospectively suspected cases of this disease trace back to a common female ancestor, born in the 1970s. Complete resequencing of the mitochondrial genome revealed a one base-pair deletion in the mitochondrial rRNA-Tyr gene only found in affected and related dogs from the same maternal lineage. Studies of this candidate mutation are in progress.

In conclusion, a novel neurological syndrome in dogs was described. Pedigree analyses resulted in strong evidence for this disease being maternally transmitted. Further analyses of affected dogs revealed evidence of mitochondrial dysfunction and a candidate mutation has been identified in the mitochondrial rRNA-Tyr gene.

**SYRINGOMYELIA IN ASYMPTOMATIC CAVALIER KING CHARLES SPANIELS DOES NOT AFFECT SOMATOSENSORY EVOKED POTENTIALS.** T.R. Harcourt-Brown, N.D. Jeffery, N. Granger. Department of Veterinary Medicine, University of Cambridge, Cambridge.

Somatosensory evoked potentials (SEPs) were recorded on clinically normal Cavalier King Charles Spaniels (CKCSs) presented to our department for magnetic resonance imaging screening of the cervical spinal cord prior to breeding. The study investigate the hypothesis that syringomyelia would affect conduction through the affected area of spinal cord. 37 clinically normal CKCSs were enrolled in the study; 20 with MRI evidence of syringomyelia between C2 and C4, and 17 without. The ultrasound was stimulated and evoked potentials recorded. The evoked cerebromedullaris potential (recorded over C1) was used for analysis of wave amplitude and latency of onset. Onset latency was divided by stimulus site to provide a measure of conduction velocity. No significant difference was found between the two groups of dogs in the amplitude or velocity of the waves. Recordings from the right and left side were also made from most of the dogs and these were analyzed to see if there was any more asymmetry of conduction in affected dogs compared to unaffected dogs. No significant difference was found. The results of this study suggest that electrical conduction through the cervical spinal cord is not affected by the presence of a syrinx in asymptomatic CKCSs. Our data provide normal values for the breed and a protocol for use of SEPs in clinically affected dogs.

**DEGENERATIVE MYELOPATHY IN CHESAPEAKE BAY RETRIEVERS.** SN Long, P Henthorn, J Serpell, T Van Winkle, JR Coates, G. Division of Small Animal Clinical Sciences, University of Pennsylvania School of Veterinary Medicine, Philadelphia, USA. Department of Veterinary Medicine and Surgery University of Missouri College of Veterinary Medicine, Columbia, Missouri, USA.

Degenerative Myelopathy (DM), also known as chronic degenerative radiculomyelopathy and German Shepherd Myelopathy, has been seen in German Shepherds (GSDs) since the 1970s, and more recently in Pembroke Welsh Corgis, Rhodesian Ridgebacks and Boxers in the United States. We have also seen a number of Chesapeake Bay Retrievers (CBR) with the condition. The aims of this project were: 1) to collect and bank blood samples from affected and control CBR, 2) to investigate the mode of inheritance and an emphasis on whether other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.

Severe lesions were evident in the left as well as right recurrent laryngeal muscles, but it remains unclear if this disease is a mononeuropathy or a polyneuropathy. An understanding of the distribution of the neuropathological lesions in RLN affected horses is fundamental to studying the aetiology of peripheral nerve disease of horses, but it remains unclear if other long nerves had a similar degree of lesions as the recurrent laryngeal nerve at the same distance from their respective cell bodies.
prevalence of DM within this breed, and 3) to determine the genetic basis for DM through genome sequencing. Information including history, pedigrees, neurological examination findings, clinical investigation and histopathological examination was collected from dogs with suspected DM. This information was used to assign a stringency level of diagnosis to cases, level 1 being those cases with histopathological confirmation, level 2 being cases with MRI exclusion of compressive lesions, level 3 being cases with myelographic exclusion of compressive lesions, and level 4 being cases with consistent clinical signs and history but no imaging. Pedigree analysis was performed on pedigrees collected from affected dogs of all stringency levels and related animals. Blood was collected for DNA extraction and sequencing from affected dogs, as well as related and unrelated controls. To calculate disease prevalence, the number of affected dogs (level 1–4) was compared with all CBRs registered with the American Kennel Club for the years 1996–2000. DNA samples were submitted to the University of Missouri for genome-wide scanning using single nucleotide polymorphism (SNP)-chip analysis, followed by direct sequencing of candidate genes. Information was collected from 40 dogs with a diagnosis of DM. Of these, 16 dogs had diagnosis stringency level of 1 or 2. A mean prevalence of 0.07% per year was calculated for this breed. Pedigrees representing a total of 943 dogs were collected. Pedigree analysis revealed a disease frequency of 0.21 within affected litters, suggesting an autosomal recessive mode of inheritance without complete penetrance. Samples were obtained from 125 dogs, of which 10 affected (level 1 or 2) and 2 control samples were initially submitted for genome analysis. Using SNP-chip analysis, line-mapping techniques and gene sequencing, a missense mutation in superoxide dismutase 1 (SOD1) on canine chromosome 31 was identified in all affected dogs. This mutation was also identified in Pembroke Welsh Corgis, Boxers, Rhodesian Ridgebacks and GSDs affected with DM. Mutations in the SOD1 gene have also been identified in humans with the familial form of Amyotrophic Lateral Sclerosis (ALS). The condition appears to be autosomal recessive with incomplete penetrance in all breeds so far examined. A genetic test for DM has been developed, which will aid in developing breeding programs designed to slowly eliminate DM from the breed.

IS TRADITIONAL CSF ANALYSIS THE GOLD STANDARD FOR CONFIRMING CNS INFECTION? EVIDENCE FROM CSF IL-6 CONCENTRATIONS. M. Lowrie1, J. Penderis1, D. F. Lappin2 and T. J. Anderson3. 1Institute of Comparative Medicine, Faculty of Veterinary Medicine and The Dental School, Faculty of Medicine, University of Glasgow, UK.

The ante-mortem diagnosis of inflammatory central nervous system (CNS) disease in dogs remains challenging due to limited tissue access. Though biopsy offers the potential of high sensitivity and specificity in identifying inflammatory disease there is limited availability and issues of associated morbidity. Consequently cerebrospinal fluid (CSF) analysis is the first-line diagnostic modality in confirming and often denying an inflammatory CNS environment. CSF analysis is usually considered as total nucleated cell count (TNCC) and total protein concentration. In protracted forms of SRMA CSF parameters have been described as being unperturbed and in our experience of SRMA cases with putative relapse we have identified patients with appropriate clinical signs, non-specific evidence of inflammation (high serum concentrations of acute phase proteins) but unperturbed CSF parameters. We hypothesise that the traditional markers of CNS inflammation are not sensitive in all circumstances of inflammatory CNS disease. We propose the presence of interleukin-6 (IL-6) as an alternative marker of an inflammatory CNS environment.

Serum and CSF samples were obtained from 16 dogs with a clinical diagnosis of SRMA presented to the small animal neurology service at the University of Glasgow Small Animal Hospital between May 2006 and May 2008. Diagnosis was based on signalment, clinical signs, history, supportive CSF characteristics (increased TNCC and protein concentration), raised serum C-reactive protein and serum amyloid-A, treatment response, and exclusion of other diseases. Serum and CSF was also obtained from five dogs diagnosed with putative SRMA relapse and six dogs with non-inflammatory CNS disease (controls). IL-6 was assayed in the serum and CSF using a canine-specific IL-6 ELISA.

Traditional markers of inflammation of CSF identified all SRMA patients at presentation with inflammatory CNS disease but only identified inflammation in 2/5 of the putative relapse group.

Serum IL-6 was greater in both SRMA patients at presentation (median = 3.9 pg/ml, p = 0.19) and putative relapse (median = 7.9 pg/ml, p = 0.52) than controls (median = 0.8 pg/ml). Likewise CSF IL-6 was greater in SRMA patients (median = 1.0 pg/ml, p = 0.65) and at putative relapse (median = 0.8 pg/ml p = 1.00) than controls (median = 0.00 pg/ml). There was no significant difference between the serum and CSF IL-6 concentrations at first presentation and relapse (p = 0.97, p = 0.74).

There is a tendency towards an increase in IL-6 in both serum and CSF in SRMA at presentation and putative relapse when compared to non-inflammatory controls and no significant difference between samples representing SRMA at first presentation and relapse. This study lends credence to IL-6 being a marker of CNS inflammation and gives some support to the hypothesis that an unperturbed CSF analysis is not sensitive to all inflammatory CNS environments.

CLINICAL, ELECTRODIAGNOSTIC, MAGNETIC RESONANCE IMAGING AND PATHOLOGICAL FEATURES OF BETA-MANNOSIDOSIS IN A SAINT BERNARD DOG. Alejandro Lujan Feliz-Pascual1, G Diane Shelton1, Janet Stone2, Anthony Blunden3, Andrew Holloway1, Kaspar Matiassek1, 1Animal Health Trust, Newmarket, UK. 2Comparative Neuromuscular Laboratory, University of California San Diego, La Jolla, CA, USA. 3Biochemical Genetics Laboratory, Bristol Royal Infirmary, Bristol, UK.

A 7mo F Saint Bernard dog was presented with a three-week history of non-ambulatory tetraparesis. Physical and neurological examination showed normal mental status, cranial nerve reflexes and nociception but generalized muscle atrophy with reduced spinal reflexes and hyperextension of appendicular joints. CSF analysis showed albuminocytological dissociation with vaculated macrophages. Polyrhaphia and delayed motor conduction was revealed by electrodiagnostic studies of the peripheral nerves. Biopsy specimens from the vastus lateralis and muscle biopsy in the thigh revealed vacuolated muscle fibers and vacuolation of the Schwann cells cytoplasm and macrophages of the endoneurium and subperineurium. By electron microscopy, membrane bound vacuoles were present in Schwann cells, macrophages, endothelial cells, pericytes and fibroblasts. Following euthanasia, MRI of the brain revealed cortical atrophy and loss of distinction between gray and white matter. The activity of beta-mannosidase in fibroblasts obtained from a skin biopsy was reduced compared to a clinically normal male sibling and non-related control dogs, but normal in leukocytes. Thin layer chromatography of the urine detected an abnormal band consistent with mannosidosis. Post-mortem results showed vacuolation of macrophages throughout the body as well as vacuolation of renal tubular, bronchial and bronchiolar epithelia, pancreatic acinar cells, hepatocytes and adrenal glands. The central, peripheral and enteric nervous system showed extensive cytoplasmic vacuolation of supporting vascular cells, neurons and glial cells with regional variations. Subcortical white matter was poorly developed but the compacted myelin was spared.

Beta-mannosidase deficiency in this dog showed an intermediate phenotype between the remainant and human disorders.

IMMUNOPHENOTYPING OF LYMPHOCYTES IN CANINE GRANULOMATOUS MENINGOENCEPHALITIS. Christopher Mariani, Alex Reynolds, Karen Munana and Talmage Brown. College of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina, USA.

Granulomatous meningoencephalitis (GME) is a common inflammatory disease of dogs with an unknown etiology. Histopathologically, the disease is characterized by perivascular accumulations of macrophages and lymphocytes, which occasionally coalesce and obliterate the surrounding parenchyma. Immunophenotyping of this inflammatory infiltrate has been infrequently reported, and the few available reports are contradictory in their results and conclusions. Kiper et al. reported a predominance of
T cells in GME lesions, with very few B cells, and suggested that the condition is likely an organ-specific, delayed type hypersensitivity autoimmune response (1). Similarly, Suzuki et al. determined that T cells comprised the vast majority of lymphocytes in 2 reports, with no B cells identified in their case series (2,3). In contrast, Tipold et al. found relatively equal numbers of T and B cells in GME lesions (4), and a recent report from Vernau et al. found a predominance of B lymphocytes (5). Most of these reports examined relatively small numbers of GME cases, and the purpose of this study was to investigate T and B cell numbers in a larger case series.

Medical and pathology records were reviewed for cases of GME. Cases with a histopathologic diagnosis of GME were identified, and sections were obtained from archival, formalin-fixed, paraffin-embedded blocks for immunohistochemistry. After antigen retrieval and blocking of endogenous peroxidase, immunostaining for T cells (polyclonal rabbit anti-human CD3, Dako) and B cells (mouse anti-human CD79ac, clone HM57, Dako) was performed using a peroxidase dependent detection system. Positively stained cells within perivascular cuffs were counted and expressed as a percentage of total inflammatory cells, with a minimum of 500 cells counted.

Fourteen cases with GME involving the brainstem or forebrain have been examined to date. Inflammatory perivascular cuffs contained large numbers of lymphocytes and larger mononuclear cells, with occasional plasma cells and neutrophils. Both T cells and B cells were identified within lesions from most cases; however B cells predominated (mean 35% of total inflammatory cells, range 13–77%), while T cells were found less frequently (mean 12%, range 0–41%). In four cases, T cell staining was completely absent.

There were several potential explanations to explain the conflicting results in the currently available studies. Firstly, different primary antibodies and immunostaining protocols were employed in the studies. Secondly, there may be regional differences in the disease process underlying the trigger disease. Thirdly, GME is characterized by a stereotyped pattern pathologic pattern, but likely represents a spectrum of diseases rather than one specific disease process.

**REFERENCES**

1. Vet Pathol 1998;35:43. 2. J Vet Med Sci 2003;65:1233. 3. J Vet Med Sci 2003;65:1319. 4. Zentralbl Veterinmed A 1999;46:75. 5. J Vet Intern Med 2008;22:770 [abstract].
A total of 77600 single teased fibres with at least 5 internode length. Different fixation protocols were applied to produce chemical artefacts. Thereby, fixation in varying dilutions of glutaraldehyde was performed over certain incubation periods up to 24 hours to achieve autolytic changes, processing was delayed until 1, 2, 4, 8, 12, 24, 48, 72, 96 and 120 hours after excision. For microscopical examination samples were impregnated by osmium tetroxide and stained with an oxacarboxyamine fluorochrome.

Both, autolytic and chemical induced changes were primarily due to Schwann cell and myelin sheath alterations. In general, non-compacted myelin sheath regions were affected most severely, as observed in paranodes and incisures of Schmidt-Lantermann (SLI). Compacted myelin was less affected. Autolytic changes became visible within the first hours by infolding of the myelin tube, followed by increased widening and swelling of SLI that may simulate initial stages of Wallerian degeneration. Pronounced stages of autolysis after 72 hours, successively precluded proper evaluation of myelin sheath integrity and became indistinguishable from Wallerian degeneration stage III. Conversely, lamellar desintegration of paranodal regions as seen in models of diabetic neuropathy may be misunderstood as autolysis of at least 24 hours of duration. Protracted fixation caused amorphic swelling, undulation, vacuolization, or leopard-like spotting of the nerve sheath. These chemical artefacts were most obvious after fixation in 6.25% glutaraldehyde for 24 hours. Overfixation rather caused disclosure than mimicry of pathological conditions. Best results were obtained with 2.5% glutaraldehyde fixation for 1 to 2 hours in juvenile and 1 hour in adult animals.

Thorough knowledge of non-pathological lesions in teased peripheral nerve fibres is essential for the reliable work-up of peripheral nerve biopsies. Our study identified a spectrum of potentially misleading alterations induced by autolysis and physicochemical stimuli.

ORTHOSTATIC TREMOR IN A WEIMARANER. Patricia Montoloiu, Luisa De Risio1, E Beltran1, J Freeman1. Neurology service, Clínica Balmes, Barcelona, Spain; 2The Animal Health Trust, Newmarket, UK.

A four-year-old, male neutered, Weimaraner was presented with a nine month history of tremors in the pelvic limbs, which had progressed to involve also the thoracic limbs. On clinical examination the dog was noticed to exhibit gross muscular tremors in all four limbs while weight bearing. Tremors were worse in the pelvic limbs and became more pronounced after the dog had been standing for a few seconds. When the dog was walking, tremors disappeared from the non-weight bearing limb during the swing phase of the gait cycle. Tremors disappeared in all limbs when the dog was lying down. Mental status, postural reactions, spinal reflexes and cranial nerve assessment remained normal. Mild and inconsistent discomfort was observed on palpation of the lumbarosacral spine and manipulation of the tail. Serum biochemistry was normal besides increased creatine kinase (2011U/L). Hematology, total T4, free T4, TSH, MRI of the lumbarosacral region, routine CSF analysis, CSF PCR for canine distemper virus, Toxoplasma gondii, and Neospora caninum, centric nerve EMG (performed under general anaesthesia), MNVC, and muscle and nerve biopsies were all normal. Conscious surface EMG recordings of extensor and flexor limb muscles, obtained whilst the patient was standing, disclosed spontaneous discharges of irregular amplitude caused increased frequency (19 Hz). These discharges were not recordable when the tested limb was lifted off the ground or when the dog was lying down. Treatment with phenobarbitone produced a marked, although incomplete, improvement.

Orthostatic tremor (OT) is a condition manifested by high-frequency tremors (13–20 Hz) of the leg/limb muscles that occur whilst the patient is standing or performing an isometric contraction. This condition has been reported in humans, 2 Great Danes and in a Scottish deerhound. The diagnosis is made based on the characteristic clinical signs (jumping and conscious surface electromyographic recordings. OT is considered to be an idiopathic disorder in the majority of cases as a primary cause cannot be identified despite extensive investigations. OT is thought to be driven by a unique supraspinal tremor generator however the exact pathophysiology of OT still remains understudied.

AQUIRED MOTOR NEURON DEGENERATION IN A CAT WITH SEVERE PELVIC LIMB CONTRACTURE1, Cecilia Rohdin1, Erika Karlstam2, Robert J Higgins1 and G Diane South3, 1Albano Small Animal Hospital, Stockholm, Sweden; 2Department of Pathology and Wildlife Diseases, National Veterinary Institute, Uppsala, Sweden; 3Department of Pathology, Microbiology and Immunology, University of California, Davis.

This report describes a young cat with paraplegia from extensor muscle contractions of the pelvic limbs. The cat was admitted as a stray at approximately 12 weeks of age with an unknown past history. On physical examination, the kitten was paraplegic with severe extensor contracture of the pelvic limbs. The contractures caused severe deformities of the stifle and hock joints. It was not possible to flex the pelvic limbs, even with force under general anesthesia. Both pelvic limbs were equally affected. The kitten showed no signs of pain. The spinal reflexes in the pelvic limbs were impossible to evaluate due to the severe contractures. Pain perception was intact in both pelvic limbs and anal reflex was normal. Pulses were good bilaterally to the pelvic limbs with appropriate color and warmth. The proximal part of the tail also showed extensor rigidity. The thoracic limbs, mental status and cranial nerve function was normal. Focal tremors was considered and the cat was treated with metronidazole. Biochemistry profile showed normal creatine kinase (CK), alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities. Serum antibody titers for retroviruses (FeLV and FIV) and protozoal infections (Toxoplasma gondii and Neospora caninum) were all negative. The spine, hips and pelvic limbs were radiographed and, with the exception of the contractures and deformities of the pelvic limb joints, no abnormalities were detected. CSF obtained from a lumbar puncture showed a normal cell count and protein concentration. Spontaneous fibrillation potentials were identified in the pelvic limbs with no abnormalities found in the thoracic limbs. Nerve conduction velocity studies were not performed. A necropsy was done three weeks after initial admittance. Lesions were limited to the lumbar spinal cord, peripheral nerve roots and nerves, and skeletal muscles. In the ventral gray matter horns of the lumbar cord, a striking, bilateral, symmetrical loss of a subpopulation of large motor neurons with prominent vascularity was found. Axonal degeneration in the nerve roots and peroneal nerve, and neurogenic muscle atrophy, were most likely secondary to neuron loss. An acquired loss of motor neurons and not a congenital condition was suspected with a possible vascular, metabolic or toxic etiology.

THE USE OF MAGNETIC RESONANCE IMAGING IN PRE-ClinICAL TRIALS OF DUCHENNE MUSCULAR DYSTROPHY IN GRMD DOGS. J.L. Thibaud, D. Bertoldi, I. Barthélemy, S. Blot, P.G. Carlier, National Veterinary School of Alfort NMR Laboratory Institute of myology AIN-CEA, France.

Magnetic resonance imaging (MRI) is being increasingly used in neuromuscular disorder patients, mainly to determine precisely the degree of pathological involvement of the different muscle groups. More recently descriptions of inflammatory muscles diseases in dogs have been published. The use of MRI in pre-clinical trials of neuromuscular diseases in dogs has never been reported. The aim of this study was to demonstrate the ability of MRI to define and follow over time quantitative indices that differentiate dystrophic form healthy muscles and their use in pre-clinical trial in Golden Retriever muscular dystrophy (GRMD) dogs.

Six two-month old control and 6 GRMD dogs were examined at 4 T. Five control and 5 GRMD dogs were examined at 3 T at the age of 2.4 and 6 months. Standard and fat-saturated T1-weighted images were acquired, followed by T2-weighted images. After Gad-DTPA injection, the time-course of muscle enhancement was monitored with fat-saturated T1-weighted imaging during 2 hours. Extensor carpi radialis and flexor carpi ulnaris were studied. Indices were calculated as follows: T2w/T1w signal ratio (SR = T2w Signal / T1w Signal) and T2w heterogeneity (H = (SD−SD)/noise(0.655)). An exponential decay was fitted to the signal decrease post Gad-DTPA injection: maximal relative enhancement (RE) and time-constant of decay were compared. A three-way analysis of variance was performed. We also evaluated muscles of three GRMD dogs 3 months after

1Albano Small Animal Hospital, Stockholm, Sweden; 2Department of Pathology, Microbiology and Immunology, University of California, Davis; 3Department of Pathology, University of California, San Diego.
intramuscular injection of adeno-associated virus type 1 for exon skipping therapy.

T2w/T1w SR, H2 and RE were found significantly increased in dystrophic muscles at 4 T. These findings were confirmed at 3 T on standard and fat-saturated sequences, at all ages. Standard and fat-saturated H1 were also found significantly increased at 3 T in dystrophic muscles at 4 and 6 months of age. The fat-saturated T1-and T2 heterogeneity indices were efficient to detect a significant evolution of the dystrophic muscles between 4 and 6 months.

Evaluation of treated dogs revealed a trend to improvement for T2w/T1w SR and H2. This finding was corroborated by a similar evolution of strength measurement after electrical stimulation of the peroneal nerve. Moreover, immunohistological evaluation of treated muscle revealed a high percentage of dystrophin-positive fibres. Several characterization indices differentiated dystrophic from normal muscles, in two different imaging conditions and over a range of ages. The fat-saturated T1-and T2 heterogeneity indices showed a significant progression with age and might be relevant to monitor muscle response to treatment. Some of these indices have induced a swelling discharge that was collected from cases and assumed to be a titiatively dystrophic muscle after exon skipping therapy in GRMD dogs.

PROGNOSTIC FACTORS AFFECTING SURVIVAL TO DISCHARGE AND LONG-TERM OUTCOME IN DOGS WITH MEGAESOPHAGUS. A. Thomson, I. Ramsey, E. Courcier, R. Bell. Faculty of Veterinary Medicine, University of Glasgow, Glasgow, Scotland.

Megaesophagus is uncommon in dogs and has a variable, uncertain prognosis. This study aimed to identify, in dogs with megaesophagus, factors at the first visit associated with death prior to discharge and which affected survival following discharge.

Patients were identified from radiological and clinical databases at University of Glasgow Veterinary School (UGVS) from 1996 to 2007. Dogs with oesophageal dilation seen only under general anaesthesia were excluded. Patient data were reviewed for signalment, historical, clinical, radiological, and treatment information. Patients were grouped into those that did or did not survive to discharge. Data concerning discharge was collected from cases notes and telephone interviews. The relationship of 38 variables to survival was evaluated. Categorical data were analysed using Kaplan-Meier and log rank methods and continuous variables using Cox’s proportional hazards logistic regression analyses. Significance was assumed at p < 0.05.

Eighty-six cases were included. German Shepherds (15), Golden retrievers (6), Labrador and Great Danes (5) were most commonly affected. Mean age at presentation was 5.2 y (range 2 m–13 y). In 64 cases, megaesophagus was the only clinical sign, 13 cases had signs of oesophageal dilatation at presentation and 9 cases of central nervous system disease. Secondary megaesophagus was most commonly attributed to myasthenia gravis (12) and vascular ring anomalies (7). Fifty-nine dogs (68.6%) survived to discharge. Patients that did not survive to discharge had decreased duration of clinical signs before referral (P = 0.004) and were more likely to have; pyrexia (P = 0.04), abnormal lower respiratory tract noise (P = 0.049), dyspnoea (P = 0.045) and an alveolar infiltrate (P = 0.011). 14 non-survivors (52%) had pyrexia and a significantly lower survival (median 3.0 months, interquartile range 1.4-7.7 months) compared to survivors (median 12.1 months, interquartile range 3.1-25.1 months, P = 0.001).

Acute inflammatory demyelinating polyneuropathy (AIDP) is one of the most important immune-mediated diseases of the peripheral nervous system in humans. Despite extensive research, the aetiology yet is poorly understood, and experimental animal models fail to display all facets of the spontaneously arising disease. Therefore, this study was aimed to characterise the immunopathological phenotype of an avian inflammatory demyelinating polyradiculoneuropathy (AvIDP) and to evaluate its possible relevance to the human disease.

This investigation enrolled peripheral nerves and spinal nerve roots from 40 female chickens, aged 6–9 weeks, comprising 20 histologically confirmed cases of AvIDP and 20 healthy controls. Immunohistochemistry (IHC) was performed on cryostat sections, in order to phenotype the cellular composition of the infiltrates, and later-on accomplished by immunofluorescence analysis of possible immunoglobuline M and G (IgG) deposition. Additionally, immune cells were harvested from spinal nerve roots and characterised via fluorescence-activated-cell-scanning (FACS). Furthermore, mRNA expression profiles concerning B- and T-cell markers were assessed in affected spinal ganglia by qRT-PCR.

IHC showed that inflammatory infiltrates consisted of nearly equal numbers of T- and B-lymphocytes with some interspersed macrophages. Both T-helper (Th) cells and cytotoxic T-cells were involved with a slight predominance of the former, as indicated by FACS-data. Cytokine analysis sorted most of Th-cells in the Th2-category which was in line with rather low IFNγ values. Induced humoral responses were highlighted by significant expression of B-cell activation markers. Moreover, affected nerves presented with intramyelinic IgG deposition. Furthermore, Th2 and T-cell markers were assessed in affected spinal ganglia by qRT-PCR.

The evidence of IgG deposition, increased B-cell markers and Th2 cytokines, in combination with low IFNγ expression are indicative of a Th2-guided immune response. Th2-features replace Th1-driven events at the late stages of AIDP and experimental allergic neuritis and they coincide with amelioration of the clinical pheno-
type in these species. Because of these striking similarities, the clinical phase of AVDP in future may offer new insights into the immunobiology underlying the Th1-to-Th2 transition observed in mammalian inflammatory neuropathies.

ASSOCIATION OF MATRIX METALLOPROTEINASES-9 AND -2 WITH PERITUMOURAL OEDEMA AND TUMOUR VOLUME IN CANINE INTRACRANIAL MENINGOMIAS. E. Beltran, L.A. Mattiasek, L. De Risto, A. De Stefani, A. Lujan Feliu Pascual, K. Mattiasek. The Animal Health Trust, Newmarket, Suffolk, UK.

Peritumoural oedema (PTO) is a frequent and variable finding in canine intracranial meningioma (CIM). The oedematogenesis of meningiomas has not been elucidated fully and is likely to be multifactorial. Matrix metalloproteinases (MMP) are proteolytic enzymes that break down basal membranes and other components of the extracellular matrix. The most widely studied MMPs are MMP-9 and MMP-2. They are important factors for tumour invasion, and, in particular, the expression of MMP-9 is known to play a role in oedematogenesis in human intracranial meningioma. MMP-9 and MMP-2 expression has been previously described in CIM. The aim of this investigation was to assess the expression of MMP-9 and MMP-2 in CIM and to investigate their association to tumour volume (Vo) and PTO.

MRI and tissue samples of 22 dogs with histologically confirmed intracranial low-grade meningiomas were retrospectively assessed. MR images were imported into a commercial software (Able software 3D-doctorTM) to measure Vo and oedema volume (Vo). The oedema index (OI) was calculated as the ratio of Vo to Vo. The tumoral expression of MMP-9, latent MMP-2 (proMMP-2) and active plus latent MMP-2 (α/ proMMP-2), was semiquantitatively assessed by different anti sera, assigning immunoreactivity scores (IRS), and subsequently compared to the imaging data.

The Vt ranged from 0.03 cm³ to 8.94 cm³ (mean 4.83±0.07 cm³, median 4.66 cm³), OI ranged from 0 to 5.38 (mean 0.24±0.27; median 0.11). IRS of MMP-9 were high (5.95±0.99) in all tumours. IRS of proMMP-2 were high in all but one tumour (5.14±1.59). IRS of α/ proMMP-2 were much low (3.00±1.56) and present only in nine tumours. None of the IRS was statistically linked to Vo, OI or expression of any of the other MMPs.

Even though a statistically significant association was not identified between IRS and OI or Vo, there was a tendency in all meningiomas with severe PTO to reveal a high expression of MMP-9, which is in agreement with previous reports in humans. However, the major player in meningioma associated brain oedema has not yet been clarified. In contrast to a previous study on CIM, expression of MMP-9 and MMP-2 did not correlate. Controversial immunohistochemistry results indicate that immunohistochemical MMP-2 assessment in dogs remains to be validated by enzymological investigations.

NEUROMUSCULAR ELECTRODIAGNOSTIC STUDIES IN FERRETS. E. Bianchi, D. Callegari, M. Ravera, M. Dondi. University of Parma, Italy.

Neurologic diseases are frequently reported in the domestic ferret (Mustela putorius furo), but experiences with most neurodiagnostic tests is limited. Especially lacking are data on electrodiagnostic procedures. Object of this study was to establish the normal values of motor nerve conduction studies (MCNS), supramaximal repetitive nerve stimulation (SRNS), F waves and corder dorsum potentials (CDP) of tibial nerve in the domestic ferret.

The tests were performed on 10 sedated domestic ferrets (6 males and 4 males) of ages ranging from 8 months to 2 years while rectal temperature was maintained above 36.5 °C. All subjects had no history of neurologic problems and unremarkable physical and neurological examinations. The sciatic-tibial nerve was stimulated at the level of the femoral neck (MCNS) and at the tarsus (MCNS, SRNS, F waves, CDP), and the muscular potentials were recorded in the plantar interosseous muscles (MCNS, SRNS, F waves). Trains of 10 stimuli with a rate of 2 Hz were used for SRNS. The late responses (F waves) were obtained recording at least 20 supramaximal stimuli. CDP were recorded from the L4-L5 intervertebral space after averaging 250–500 stimulations. Only one tibial nerve for each subject was evaluated. Descriptive statistics were calculated for the main parameters.

Conduction velocity, proximal and distal CMAP amplitudes of MCNS of sciatic-tibial nerve were respectively 63.25+/−7.6 m/sec, 10.79+/−2.7 mV, and 13.02+/−3.4 mV. In all ferrets for each train of SRNS were obtained decrements of less than 8% of CMAP amplitudes and areas between the first and the following waves. The late potentials (F waves) had a minimum latency of 8.49+/−0.65 ms and a “F” ratio of 1.92+/−0.160. CDP onset latency was 1.99+/−0.03 ms, onset to peak latency difference was 1.85+/−0.36 ms and peak amplitude was 4.92+/−2.93 μV.

The waveforms recorded in ferrets were not dissimilar from those found in other species. Late potentials could be elicited only with supramaximal stimuli, therefore they were probably constituted mainly by F waves, but a contamination by the H reflex can’t be excluded. Latencies of these F waves and of CDP were stable among subjects as expected for the lack of limb length variability of this species. At the authors’ knowledge this is the first report on normal values of neuromuscular electrodiagnostic studies in domestic ferrets. More studies including other electrodiagnostic tests and nerves are needed to further increase the information available for investigation of disorders affecting the peripheral nervous system of ferrets.

CHRONIC TRIGEMINAL NEURITIS IN A DOG. Bonaldi M1, Cantile C, Cozzi F2 and Lombardo R3. 1University of Milan, School of Veterinary Medicine, Milan, Italy2University of Pisa, School of Veterinary Medicine, Pisa, Italy3Private Practitioner, Milan, Italy.

The purpose of this study is to describe the case of a dog showing clinical signs and magnetic resonance imaging findings of trigeminal nerves involvement with a 5-year follow-up and histological confirmation of bilateral chronic trigeminal neuritis. A 5-year-old spayed female Rottweiler was examined; the presenting complaint was masticatory muscles atrophy and mandibular plosis that had been noticed a few days before. Physical examination confirmed bilateral masseter and temporalis muscles atrophy, partial mandibular plosis and reduced jaw tone, and revealed mild depression, decreased gag reflex, bilateral corneal and nasal anesthesia. Hematology and chemistry profile were within normal reference, included freeT4 and TSH. Serum titers for Leishmania and Toxoplasma were both negative. Electromyography of the masseter and temporalis muscles on both sides was within normal limits. A cervical cerebrospinal fluid tap was performed and cell count and cytology were within normal limits, total proteins were 66 mg/dl. At magnetic resonance imaging trigeminal nerves were enlarged,ointense on T1- and T2-weighted images and with presence of homogeneous contrast enhancement. The suspected diagnosis was an infiltrative lesions such as bilateral nerve sheath tumor or lymphoma. The dog survived 5 years with nursing care, clinical signs progressed slowly during that time, then the dog was euthanized at the owner’s request. Macroscopical examination revealed marked enlargement of the left trigeminal nerve with adhesion of the epinevrium to the basal dura. Histopathology revealed marked epineural and endoneural fibrosis with extensive loss of nerve fibers. Moderate fibrosis and fiber loss of the contralateral nerve was also observed. Inflammatory cell aggregates were seen, and only occasionally observed. In the trigeminal ganglia there was ganglioncic cells loss, fibrosis and presence of Nagotte nodules, bilaterally. The lesions were interpreted as the result of a chronic bilateral trigeminal neuritis and ganglionitis. In conclusion, based on our experience, chronic neuritis should be taken into consideration in differential diagnosis of a bilateral trigeminal nerve disease, with signs of enlargement of the nerves.

PHOTIC STIMULATION AND HYPERVENTILATION IN ELECTROENCEPHALO-GRAPHIC RECORDINGS IN TEN HEALTHY BEAGLE DOGS UNDER PROPOFOL ANAESTHESIA. C. Brauer, S.B.R. Kästner, H.C. Schenk, J. Tünsmeyer, A. Tipold. Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

The purpose of this study was to evaluate the effect of photic stimulation and hyperventilation on the electroencephalogram.
CAUSED BY MYCOBACTERIUM AVIUM IN AN EUROPEAN MINK.

The mink was initially treated with clindamycin. After two weeks nothing was observed on clinical examination or myelography. Therefore a second treatment with clindamycin was given for two more weeks. After this treatment interval of three minutes without stimulation the dogs were hyperventilated and the endtidal CO₂ (EtCO₂) concentration was measured continuously. Hyperventilation was performed for at least 180 seconds and until an EtCO₂ concentration of 25 mmHg was reached. Post hyperventilation recording lasted for another three minutes.

Visual analysis did not reveal differences in background activity between periods with and without stimulation methods of any kind. Paroxysmal discharges did not occur. Muscle artefacts vanished within seconds after rocuronium administration and did not reappear during periods with stimulation. Quantitative analysis of the EEG recordings with Fast Fourier Transformation (FFT) showed presence for slow delta rhythms in all derivations. Means for relative power data of delta activity ranged from 57.06 to 69.67%. Frequency bands (δ, θ, α, β) were evaluated between several points of stimulation but no obvious increase or decrease occurred during periods with and without stimulation.

EEG in veterinary neurology is limited because anaesthesia is necessary for artefact free recordings. Photic stimulation and hyperventilation are frequently used in human medicine to increase the occurrence of paroxysmal discharges and therefore to improve the diagnosis of epilepsy. Further studies will show if the described method is capable of inducing interictal paroxysmal discharges in dogs suffering from epilepsy. The results of the current study will help to compare EEG data of healthy dogs and dogs suffering from epilepsy, since healthy dogs seem not to develop paroxysmal discharges during periods with photic stimulation and hyperventilation.

CLINICAL EVALUATION OF 51 DOGS TREATED CONSERVATIVELY FOR DISC ASSOCIATED WOBBLER SYNDROME. S De Decker, SFM Bhatti, L Duchateau, I Van Soens, SAE Van Meervenne, JH Saunders, LML Van Ham. 1Department of Small Animal Medicine and Clinical Biology, 2Department of Physiology and Biometrics and 3Department of Medical Imaging, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

Disc associated wobbler syndrome (DAWS) is the most typical and most presenting wobbler syndrome. Little is known about the results and risk factors associated with non-surgical treatment of this specific wobbler syndrome.

Medical records of dogs with DAWS treated conservatively were retrospectively analyzed. Diagnosis was confirmed by myelography in all dogs. Dogs were considered to have conservative treatment if no surgery was undertaken during the study period. Fifty-one dogs were included in the study. Follow-up information was obtained by recheck examinations or telephone questionnaire. An outcome score of 1 to 11 was given and a successful outcome was defined as an outcome score of 9 or higher. This definition included only dogs that did not show worsening of clinical signs after DAWS was diagnosed. The following potential risk factors were evaluated: age, sex, type and results of treatment prior to diagnosis, severity of clinical signs, number of protruded intervertebral discs (IVDs), and additional radiographic and/or myelographic abnormalities. Statistical analysis was performed using the Fischer exact test, the Wilcoxon rank sum test and the proportional odds model. Significance was established at a P ≤ 0.05.

A successful outcome was achieved in 45% (23/51) of the dogs. This included dogs with an unchanged (n = 1) or improved (n = 22) neurological status. None of the dogs improved to a symptom free clinical status (outcome score of 11). Median follow-up period was 18.5 months and median survival time was 47 months. Eighty-five percent of the dogs, who had to be euthanized due to DAWS, were so in the first year after diagnosis. There was no significant effect of age (P = 0.8), number of protruded IVDs (P = 0.3), use (P = 0.06), type (P = 0.3) and results (P = 0.07) of prior treatment to diagnosis on the outcome scores of dogs treated conservatively for DAWS. Severity of neurological signs (P = 0.05) and additional abnormalities on radiography and/or myelography (P = 0.03) were significantly associated with lower outcome scores.

A result of this study is to suggest that conservative treatment of DAWS can be considered in mildly affected cases without additional radiographic and/or myelographic abnormalities and that the first year after diagnosis is a critical period to evaluate the outcome.

MAGNETIC RESONANCE IMAGING AND HISTOPATHOLOGICAL CHARACTERISTICS OF INTRACRANIAL AND SPINAL MENINGIOANGIOMATOSIS - 2 CASES. Rui Gonçalves, Pamela Johnston, Annette Wessmann, Jacques Penderis. Faculty of Veterinary Medicine, University of Glasgow, Glasgow, Scotland.

Meningioangiomatosis is a rare proliferative disorder of the central nervous system, only sporadically reported in the veterinary

minectomy and durotomy from C4–C6 was performed to obtain tissue biopsies. Since a moderate malacia of the spinal cord was seen during surgery, the mink was euthanized with the owner’s consent. Histopathological examination revealed moderate, multifocal, subacute, non-suppurative, encephalitis with multifocal areas of malacia within the cerebrum, brainstem and cerebellum. Abnormalities at the cervical spinal cord included severe, multifocal, granulomatous myelitis with extensive malacias and intramedullary basillary microcorticis. Bacterial culture as well as immunohistochemistry for CDV and toxoplasmosis were negative. PCR for Mycobacterium avium species on spinal cord tissue showed a positive result.

Central nervous system (CNS) infections caused by Mycobacterium avium have been described previously, especially in humans, dogs, and cats. This is the first report of an isolated CNS disease in an European mink, should be considered in these species and is an important differential diagnosis to granulomatous myelitis of unknown origin in minks.
literature as the cause of progressive brainstem or cervical spinal cord deficits. The present report aims to describe two further cases of this rare condition, their imaging and histopathological findings and for the first time to document the presence of a meningioangiomatosis lesion in the thoracolumbar spinal cord.

Case 1 – A 4-year old male boxer presented for investigation of progressive paraparesis with concurrent facial and urinary incontinence. Neurological examination localised the lesion to the T3-L3 spinal segment. Magnetic resonance imaging (MRI) was performed using a 1.5 T unit. This revealed an intramedullary lesion at the level of T12, hyperintense with a hypo-intense centre on T2WI and with marked homogenous contrast enhancement following administration of gadolinium. At the owner’s request, the dog was euthanatised and on gross postmortem examination a triangular area of discolouration was seen in the thoracolumbar cord. Histopathology of this lesion revealed a locally extensive well demarcated unencapsulated lesion composed peripherally of high numbers of proliferating small blood vessels lined by flattened endothelial cells admixed with spindle cells. No mitotic figures were seen. Centrally within the lesion vessels and spindle cells were separated by increased amounts of eosinophilic material (collagen). Masson’s Trichrome confirmed the increasing amounts of collagen towards the centre of the lesion. Immunohistochemistry with von Willebrands Factor and Vimentin, confirmed the presence of endothelial cells and spindle mesenchymal cells respectively. Remaining neoproliferated demonstrated degeneration with loss of normal architecture, vacuolation within the white.

Case 2 – A 5-month old female Labrador presented with a 3 month history of progressive central vestibular signs. MRI of the brain revealed the presence of a lesion affecting the brainstem and the thalamus that was hyperintense on T2WI and FLAIR, isointense on T1WI and had only mild patchy contrast enhancement. The owners refused further investigation and the dog was euthanatised. Histopathology of this mass revealed the same characteristics as those of case 1, although mineralisation was present in the meningeal plaque in this case (previously unreported).

Meningioangiomatosis should be considered in the differential diagnosis of brainstem, cervical and also thoracolumbar spinal cord signs. MRI characteristics of these lesions vary, most likely depending on the degree of vascularisation and collagen deposition.

THE EVOLUTION IN DIAGNOSTIC IMAGING TECHNIQUE OF CHOICE IN DOGS WITH THORACOLUMBAR SPINAL CORD INJURIES AND ITS INFLUENCE ON OUTCOME (2000-2007)

D. Henke, M. Vandevelde, J. Lang, A. Oevermann, ‘University of Bern, 3012 Bern, Switzerland.

This report describes an eosinophilic granulomatous encephalitis of unknown origin in two unrelated 3 (dog 1) and 6 (dog 2) month-old Belgian Tervueren dogs that were presented both with a one week history of progressive tetraparesis and proprioceptive deficits. Neurological examination in dog 1 revealed a non-ambulatory tetraparesis with severely increased muscle tone on the left side. Postural reactions were absent. Clinical signs progressed to decreased consciousness and gag reflex. Dog 2 revealed an ambulatory tetraparesis, hypermetric gait, severe dorsoflexion of head and neck, mildly decreased postural reactions and decreased to absent menace response and palpebral reflex and developed behavioural changes within 2 days. On magnetic resonance imaging (MRI), bilateral extensive diffuse intra-axial lesions in the cerebral hemispheres of the frontal, parietal and temporal lobes were observed. The changes were mainly located in the white matter without obvious mass effect. However in the thalamus, the grey matter of parietal and temporal lobes was affected as well. The lesions were hyperintense on T2-weighted and FLAIR images, with multifocal small (0.3-0.5 cm) round regions with irregular margins, which were isointense to normal brain tissue. These lesions were slightly hypointense on T1-weighted images with mild to moderate inhomogeneous contrast enhancement. Additionally, there was extensive meningeal enhancement in the frontal and parietal lobes in dog 1. Cerebrospinal fluid (CSF) analysis revealed 31 (dog 1) and 14 (dog 2) white blood cells/μl, 300 mg/dl albumin and a positive pandy test in dog 1. Differentiation revealed 27% (dog 1) and 24% (dog 2) eosinophil granulocytes in the CSF, respectively. An eosinophilic meningoencephalitis was suspected. Serum antibody titer for Toxoplasma gondii were positive with 1:320 in dog 2, but negative in dog 1. Serum antibody titer for Neospora canis (dog 2) and CSF antibody titer for Borrelia (dog 2) were negative. Dogs were euthanatised by request of the owners. Neuropathology revealed multiple, randomly distributed grayish-tan foci up to 1 cm in diameter in brain (dog 1 and 2) and spinal cord (dog 1). Histologically, these areas consisted of a necrotic central zone surrounded by numerous epithelioid macrophages and eosinophils and a smaller number of lymphocytes, plasma cells and neutrophils. Causative infectious agents could not be detected neither in H&E and special stains nor with immunohistochemistry. Both deso 5 grading were of the same breed, sex and age and showed striking similarities in clinical signs, MRI, CSF and neuropathological findings. Canine eosinophilic meningitis and meningoencephalitis have been described in protozoal infections and as an idiopathic form. The histopathology of the latter two conditions does not resemble the lesions in our dogs. Focal areas of necrosis surrounded by intense eosinophilic inflammation may be equally distributed between grades 2 to 5 for myelography patients. In contrast, the majority of MRI patients (53.85%) were grade 2, reflecting intermittent MRI availability and therefore its greater use in patients in which a delay to imaging was not considered clinically detrimental. Consistent with this the duration of clinical signs prior to imaging was greater than seven days in only 12% of the myelography cases, but over 37% of MRI cases. For patients that had radiography only performed 75% of these patients were either grade 2 or 5. Between 2000 and 2007, the use of myelography decreased from 63.6% to 23.3% and radiography from 31.8% to 10%. MRI increased from 0% to 63.3% of cases. Comparison of outcome could demonstrate no statistical difference when the grade and speed of recovery was compared between the MRI and myelography patients.
suggestive of migrating parasitic larvae but no parasitic fragments could be observed in the lesions and granulomas were not found in other organs. Worthy of note is the histopathological similarity to the eosinophilic granulomas of yet unknown origin that have been previously reported in intestine, liver, lungs and oral cavity of Siberian Huskies, cavalier King Charles spaniels and occasionally other breeds. The lesions in the present two cases differ vastly from any of the so called canine breed specific encephalitides. It cannot be excluded that they represent yet another form of immune-mediated, tissue damage perhaps specific for the Belgian Tervueren shepherd breed.

DEVELOPMENT OF A TAIL PARALYSIS MODEL IN THE YUCATAN MINIPIG. JH Lim, JA Piedrahita, T Ghashghaei and NJ Olby. College of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina, USA.

Research into developing transplantation strategies to treat spinal cord injury is performed in rodents but there are significant limitations to this approach. For example, the distances over which an axon needs to regenerate in a rat are much smaller than would be needed in a dog or human. A large mammalian model of severe spinal cord injury is needed but animal welfare and practical considerations mean that producing hind limb paralysis is undesirable. To determine whether it was possible to induce upper motor neuron paralysis of the tail in Yucatan minipigs, we investigated the surgical anatomy and the behavioral and histological effect of complete spinal cord transection at the level of first caudal (cd1) segment. Pigs were trained before surgery for behavioral analysis and videotaped for quantitative measurement of changes of tail deviation and movement. Following dorsal laminectomy and stimulation of the nerves of the cauda equina, the first caudal spinal cord segment was identified and transected. Pigs were monitored for one month post-operatively and then euthanized, perfused and analyzed histopathologically.

The normal pig tail was neutrally positioned horizontally with a curly shape and responded to stimuli by wagging, ventroflexion or dorsiflexion. After complete transection, the tail maintained a consistent ventroflexed position except when urinating or defecating. Bowel and bladder function and hind limb gait remained intact. Quantitative evaluation revealed permanent changes of tail deviation in the vertical plane. On histological evaluation, a complete spinal cord transection at the level of cd1 segment was present. We conclude that cd1 spinal cord transection in pigs produces persistent deficits in tail motor function without urinary and bowel impairment. This paralysis model can be used to investigate transplantation strategies following severe spinal cord injury.

NEUROMUSCULAR SYNDROME WITH UNUSUAL CLINICAL COURSE IN YOUNG SNOWSHOE CATS. L. Matiassek, A. Lujan Feliu-Pascual, G. D. Shelton, L. De Riso, K. Matiassek. 1. Institute of Veterinary Pathology, Chair of General Pathology and Neuropathology, and 2. Section of Neurology, Department of Small Animal Medicine, Ludwig-Maximilians University, Munich, Germany. 3. The Animal Health Trust, Newmarket, Suffolk, UK.

Feline herpesvirus 1 (FHV-1) is an alpha-herpesvirus which causes acute upper respiratory tract and ocular diseases in cats. Prevalence is more than 80% of cats become latent carriers of the virus after FHV-1 infection in spite of a specific immune response. The primary site of latency is supposed to be the trigeminal ganglion. Corticosteroid administration, lactation, pregnancy, re-housing and other types of distress can lead to reactivation and shedding of the virus. FHV-1 had been detected in neural and non-neural tissue by polymerase chain reaction (PCR). This is the first study, however, that specifically investigates the feline vestibular apparatus and ganglion for the presence of FHV-1 using PCR.

The study enrolled 97 temporal bones and 60 trigeminal ganglia of altogether 50 cats that were presented for post-mortem examination. All clinical records were screened for vestibular abnormalities and other neurological deficits. The vestibular apparatus, the superior/inferior vestibular ganglion, and the trigeminal ganglion were harvested, snap frozen in liquid nitrogen, and stored at −80°C. DNA extraction was performed with a commercial available kit according to the manufacturer’s description. Published primers of the FHV-1 thymidine kinase gene were used to amplify a 383 base pair product. The reaction products were electrophoresed through a 2% agarose gel with 0.5 TAE buffer, and stained with ethidium bromide.

No FHV-1 DNA was detected in the vestibular apparatus of any cat. In 14% of the 50 cats FHV-1 DNA was detected in the vestibular ganglion with a unilateral distribution in 86% and bilateral affection in 14% of the cats. Thirty two percent of the examined cats were bilaterally positive in the trigeminal ganglion. Twenty two percent of the trigeminal ganglion positive cats also harboured FHV-1 DNA in the vestibular ganglion. One of seven cats with vestibular FHV-1 infection had presented with contralateral vestibular deficits due to suppurative otitis media and interna. One further cat showed unilateral neurological findings due to tumour brain oedema. Inflammatory CNS changes were not recognised.

FHV-1 DNA was detected via PCR in the vestibular ganglia of a considerable number of cats. Infection of the vestibular ganglion always was associated with FHV-1 DNA in the trigeminal ganglia. As reported in other species, vestibular infection does not necessarily lead to dysfunction and clinical signs. Whether or not presence of FHV-1 poses an increased risk of developing vestibular disease in immunosuppressive situations remains to be further elucidated. However, transient vestibular signs rarely give rise to euthanasia thereby precluding the invasive approach, necessary to confirm an infection of the respective inner ear ganglion.
NEURONAL VACUOLATION AND SPINOCEREBELLAR DEGENERATION IN TWO ROTTWEILER SIBLINGS, Z.Ş. Polizopoulou1, N. Soğbı̈sılın1, A. Taşgıdı̈l1, A. Giannakopoulou2, G. C. Papadopoulos2, A. Oevermann2, A.F. Koutinas2. Clinic of Companion Animal Medicine and Laboratory of Anatomy and Histology, Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece. 2NeuroCenter, Department of Clinical Veterinary Medicine, University of Bern, Switzerland.

Neuronal vacuolation and spinocerebellar degeneration, a rare idiopathic degenerative disease that has been sporadically reported in purebred Rottweiler puppies, is described in this report. A 3-month-old female Rottweiler was initially admitted by referring veterinarian with acute ataxia and stridor, both of which resolved after treatment of the animal in the intensive care unit. Subsequent neurological examination revealed ataxia, spastic tetraparesis, hypermetria and proprioceptive deficits that were more pronounced in the posterior limbs. One month later its littermate, a four-month-old male was also admitted with ataxia and weakness as the main complaints. Mild inspiratory stridor, ataxia, spastic tetraparesis, hypermetria, positional strabismus and nystagmus were detected upon neurological examination. A unilateral laryngeal paresis was diagnosed with laryngoscopy. In both cases, cognition and behavioral and spinal reflexes were normal. The sire of these puppies was a direct offspring of the dam. Motor signs worsened progressively over the next two months in both puppies, finally leading to severe tetraparesis. Such deterioration was not observed in laryngeal function and no further dyspneic episodes were seen.

Due to the severely incapacitating signs and poor prognosis the puppies were eventually euthanized but necropsy was permitted only for the male littermate. Sections of the brainstem and cervical segments of the spinal cord were cut and stained with Kluver-Barrera and/or hematoxylin/eosin following in vivo fixation with intracardiac perfusion of 10% neutral buffered formalin. Clear vacuoles of varying size were observed within neurons and the neuropil of several brainstem nuclei with bilateral-symmetrical distribution. These lesions were associated with gliosis. The cervical spinal cord was affected by bilateral-symmetrical axonal degeneration and myelin loss, most prominent in the lateral spinocerebellar tract. Furthermore, loss of large calibre fibres and predominance of small calibre fibres was observed in the cranial laryngeal nerve. Similar cases have been described in young Rottweilers in which vacuolated neurons were visualized within dorsal root and autonomic ganglia, cerebellar nuclei and other parts of the CNS. Axonal degeneration and/or necrosis in the cervical spinal cord and peripheral nerves have also been reported in those cases.

EPIDEMIOLOGIC FEATURES, CLINICAL FINDINGS AND LONG-TERM FOLLOW-UP OF 33 DOGS WITH PRESUMPTIVE IDIOPATHIC VESTIBULAR DISEASE. Sergio Rodenas, Marti Pumarola, Sónia Añor. Departament de Medicina i Cirurgia Animals, Facultat de Veterinària, Universitat Autònoma de Barcelona, 08193 Bellaterra, Barcelona, SPAIN.

Idiopathic vestibular disease is a well recognized condition in dogs. The classic form affects most commonly older dogs and clinical signs reflect unilateral or bilateral involvement of the peripheral vestibular system. Diagnosis is based on the presence of acute peripheral vestibular signs and lack of abnormal findings in any diagnostic test. The condition usually resolves spontaneously within 2–4 weeks, although residual head tilt and ataxia may persist. Simultaneous occurrence of idiopathic peripheral vestibular disease and facial nerve dysfunction has also been reported. Medical records of 33 dogs with suspected idiopathic vestibular disease were investigated retrospectively in order to investigate (1) the epidemiological and clinical findings, (2) the diagnostic aids, (3) facial nerve dysfunction, (3) the outcome, including de presence of residual signs and the time needed for complete resolution. All dogs in the study had: (1) acute or per-acute onset of peripheral vestibular signs, (2) complete physical and otoscopic exams, (3) neurological examination consistent with peripheral vestibular disease, (4) normal complete CBC, serum biochemistry and thyroid profiles, (5) unremarkable brain MRI, (6) and lack of previous administration of corticosteroids. CSF analysis was normal when performed (16 dogs). The most commonly represented pure breeds were the Boxer (8/33; 24.24%) and the German Shepherd (5/33; 15%). Twelve dogs were large breed dogs (body weight >15 kg) and 4 were small breed dogs (body weight <15 kg). Median age at presentation was 10 years (range 6–15 years). Twenty-one dogs were male, and 12 were female. CSF analysis was performed in 16 dogs and results were within normal limits in all cases. Normal BAER were recorded in one dog. Regarding the onset of clinical signs, no correlation with a particular season could be found. Head tilt was present in all dogs and ataxia was identified in 31/33 dogs. Sixteen dogs had moderate ataxia, 6 dogs had mild ataxia and 8 dogs were severely ataxic. Rolling and falling towards the side of the head tilt was observed in 5 dogs. Circling to the side of the head tilt was present in two dogs. Positional ventrolateral strabismus was observed in 29/33 dogs. Spontaneous nystagmus was identified in 18/33 dogs and positional nystagmus was observed in 5/33 dogs (rotary in 8 dogs and horizontal in 15 dogs). Ten dogs were vomiting or had a history of vomiting at the time of presentation. Two dogs developed bilateral vestibular syndrome two and three weeks after the initial presentation, respectively. Facial nerve dysfunction was observed in 12 (36%) dogs, and 7 of these were Boxers. Long term follow-up (1–18 months) was obtained for 31 dogs. Two dogs died within 1 month after presentation due to unrelated causes. Histological examination in these dogs was unremarkable. Residual signs were observed in 12 dogs (36%). Four dogs suffered episodic head tilt and mild ataxia, and 4 dogs had a mild permanent head tilt. Mild facial nerve dysfunction was observed in four dogs. Twenty-one dogs did not have a residual sign. In these dogs, the mean time to resolution of the head tilt was 18 and 43 days, respectively. The results of this study suggest that idiopathic facial dysfunction is a relatively common finding in dogs with idiopathic peripheral vestibular disease, and that there is an increased risk for these syndromes in Boxers. Necropsy was permitted in a high percentage of dogs. Mean time for resolution of clinical signs in the dogs of this study was similar to that reported in previous studies. However, our results suggest that the time for resolution of facial nerve dysfunction is longer than that needed for resolution of ataxia and head tilt.

MAGNETIC RESONANCE AND HISTOPATHOLOGICAL FINDINGS IN 2 DOGS WITH A VASCULAR CAVERNOUS MALFORMATION OF THE BRAIN. Sergio Rodenas, Marti Pumarola, Sónia Añor. Departament de Medicina i Cirurgia Animals, Facultat de Veterinària, Universitat Autònoma de Barcelona, 08193 Bellaterra, Barcelona, SPAIN.

Vascular malformations of the brain are an infrequent cause of neurological dysfunction in both humans and animals. This paper reports the MR and histopathological findings in two adult dogs with cavernous malformations in the brain. A 12-year-old, female mixed breed dog was presented with a history of disorientation of 3 days duration and generalized cluster seizures of one day duration. Neurological examination was consistent with a right cerebral hemispheric lesion. Serum biochemistry, CBC, thoracic radiographs and abdominal ultrasound were unremarkable. Magnetic resonance imaging was performed one day after presentation. On T1 weighted images (WI), a heterogeneous and hyperintense lesion in the right cerebral hemisphere was causing compression of the right lateral ventricle and a midline shift to the left. Contrast enhancement was mild and heterogeneous. On Flair and T2WI, the lesion was heterogeneous and hypointense, with several areas of different signal intensity. Marked perilesional edema was also observed. The dog’s neurological condition worsened progressively and death occurred two days after presentation. At necropsy, a focal hemorrhagic lesion was observed in the right parietal lobe. A 13-year-old, female mixed breed dog was presented with a history of disorientation and generalized cluster seizures of 3 days duration by the owner. Neurological examination was consistent with a right cerebral hemispheric lesion. Serum biochemistry, CBC, thoracic radiographs and abdominal ultrasound were normal. Magnetic resonance imaging of the brain displayed a heterogeneous and mildly hyperintense lesion in the right cerebral hemisphere on T1WI. Compression of the right ventricle and a midline shift to the left were also observed, as well as moderate and heterogeneous contrast enhancement. On T2WI, the lesion was heterogeneous and hypointense, and a moderate amount of perilesional edema was also observed. The dog was euthanized one week later because persistent neurological dysfunction. At necropsy, a hemorrhagic mass was found within the right frontal and parietal lobes.
Histological findings were similar in both cases, demonstrating the presence of non-encapsulated masses, filled by blood cells surrounded by small endothelium-covered cavities. A capillary proliferation infiltrated the adjacent brain parenchyma. The blood vessels had accumulations of collagen fibers (Masson’s trichrome), but lacked elastic fibers (Verhoff-Van Gieson). A few GFAP-positive astrocytes were found surrounding the vascular proliferations. Histological diagnosis in both cases was cavernous angioma of the brain.

To the authors knowledge, there are only a few reports of cavernous malformations in the brain of dogs, and there is only one case describing the MR findings of such lesions. Cavernous malformations in human beings appear on MR images as sharply lobulated masses, often without perilesional edema, but with different signal intensity areas which give them a popcorn-like appearance. A hypointense peripheral rim representing hemosiderin on T2WI is often found in cavernous angiomas. A T1 hyperintense perilesional signal is also a common feature of cavernous malformations. The MR appearance of intracranial hemorrhages depends in part on the time elapsed from the onset of the bleeding episode and the time of imaging. In the dogs of this report, the presence of hypertensive lesions on both T1WI and T2WI was consistent with late subacute hematomas. Magnetic resonance imaging features were suggestive of intracranial hemorrhage in both dogs, although an underlying cause for the bleeding could not be determined. Vascular malformations should be included in the differential diagnostic list for dogs with acute onset of cerebral signs and MR images consistent with recent bleeding of undetermined cause.

**INVESTIGATING THE ROLE OF PERISYNAPTIC SCHWANN CELLS IN ACUTE PARALYTIC PERIPHERAL NERVE DISORDERS – ESTABLISHMENT OF BASELINE VALUES AT THE MURINE NEUROMUSCULAR JUNCTION.** A. Rusbridge1, S. Knowler1, L. Pieterse2, M. Saito, A. Fukui, M. Muto and M. Inoue2.

The results of this preliminary study indicate, that in correspondence with other studies which have described an increased loss of NMJ stability with age, the cellular composition of the NMJ also changes over time and an increased diversity can be observed between individual animals. These observations need to be kept in mind when conducting repeated *in vivo* studies on NMJs of the sternomastoid muscle in the mouse.

**CHIARI-LIKE MALFORMATION IN THE Griffon Bruxellois, C. Rusbridge1, SP Knowler1, L. Pieterse2.** Stone Lion Veterinary Centre, Wembley, UK. Statusque Griffon Bruxellois, Sydney, Australia.

The purpose of this study was to describe Chiari-like malformation (CM) and syringomyelia (SM) in the Griffon Bruxellois (GB) and to establish if skull radiographs could be used to predict the disease.

A MRI scan of the brain and neck was obtained in 56 BG dogs and assessed for CM, SM, spinal cord central canal dilation (CCD) and ventricular dilatation (VD). Lateral skull radiographs were obtained in 33 dogs and two rostrocaudal and two ventrodorsal measurements were made. Ratios of these measurements were compared between 1) male and female; 2) with and without CM; 3) with and without SM; 4) CM and SM. Differences with p < 0.01 were considered significant.

In this selected population, 61% had CM, 47% had SM ≥ 2 mm wide (9% SM only, 38% CM/SM) and 18% had CCD or SM < 2 mm wide. All dogs with CCD or SM had ventriculomegaly with 94% (34 of 36) having moderate or severe VD. 4 dogs had clinical signs of CM/SM; all had an asymptomatic CM/SM affected dam. There was evidence of hereditary predisposition to both CM and SM. There may be a “carrier” state for CM as normal dogs may have affected offspring.

The radiographic study demonstrated that using one of the rostrocaudal measurements in a ratio with any of the lengths could be used to predict CM. The most useful was to divide the first rostrocaudal measurement by the second. Dogs with CM tended to have a value of less than 2.7. This technique may be useful as a low cost screening test for CM in the GB until such time that a genetic test is available. The skull radiographic measurements could not be used to predict gender or SM. The radiographic study also suggested that CM in the GB is characterised by shortening the basicranium (skull base) and lengthening of other bones in the cranial vault especially the parietal bone. It is hypothesized that the shortening is due to craniosynositis and the lengthening is compensatory, allowing the developing forebrain to be accommodated. However, because growth of the bony caudal fossa parallels the cranial fossa and is in advance and independent of cerebellar growth there is no compensatory increase in size of the caudal fossa resulting in overcrowding of the neural structures i.e. CM. It is further hypothesized that the bony changes in brachycephalism and/or CM could result in intracranial hypertension due venous narrowing at the jugular foramina which could be a contributory factor in the pathogenesis of both ventriculomegaly and SM.

In conclusion, CM in the GB is characterised by a shortening of the basicranium and a compensatory lengthening of other bones in the cranial vault especially the parietal bone. We describe a technique for quantifying these bony changes which could have use as a screening test for CM. SM in the GB most commonly occurs in association with CM but it may occur without the bony defect. Ventriculomegaly is also common especially in conjunction with SM.

**ESOPHAGEAL COMPOUND MUSCLE ACTION POTENTIALS ELICITED BY REPEETITIVE NERVE STIMULATION IN DOGS.** M. Saito, A. Fukui, M. Muto and M. Inoue. Azabu University, Sagamihara, Kanagawa, Japan.

The aim of this study was to evaluate whether compound muscle action potentials (CMAPs) can be detected in the esophageal muscle following repetitive, subcutaneous electrical stimulation of the vagal nerve, and to investigate the feasibility of this as a test for neuromuscular transmission disorders of the esophagus.
Normal beagles were used in the study. The esophagus and vago-
sympathetic trunk were surgically exposed in anesthetized dogs 
(n≈3). Wire recording electrodes were inserted into the esophageal 
muscle. The vago-sympathetic trunk was electrically stimulated us-
ing a forceps-electrode to confirm that CMAPs could be obtained 
from the esophagus. The surgical site was closed. All the electrodes 
were replaced subcutaneously, and the procedure was then re-
peated. Subcutaneous, supramaximal repetitive stimulation of the 
vaso-sympathetic trunk was performed in additional dogs before 
(n≈10) and after (n≈6) pancuronium bromide administration. 

Polyphasic esophageal CMAPs were recorded both surgically and 
subcutaneously. Repetitive stimulation with low frequency produced 
< 10% decrement in all dogs. Repetitive stimulation after pancuronium bromide revealed a decremental response in 5 
of 6 dogs. No significant complications were associated with the 
procedure.

This study suggests that this technique may be useful in evaluat-
ing dogs with suspected neuromuscular transmission disorders of 
the esophagus.

**RECOVERY PROGNOSIS VALUES OF RHEOBASE, CHRONAXY, FISHGOLD QUOTIENT IN A CANINE MODEL OF DENERVATED MUSCLE.** Serge G Savaya*, Catherine Whitmann*, Claude Carozzo, Catherine Escriot. UPSS 2007- 
03-135 RT12B, Ecole Nationale Vétérinaire de Lyon, – France. 
*Biom’Up – Labouratoire des Biomate´riaux, Université Claude 
Bernard, Lyon, France.

Electrophysiological stimulation parameters (ESP) as the rheo-
base (Rh), chronaxy (Chr) or Fishgold quotient (FQ) are considered 
as traditional basic parameters in human neuromuscular electrodi-
agnostic and electrotherapy, especially when dealing with muscle 
denervation. Rh is the intensity threshold necessary to obtain motor 
fiber depolarization with a infinitely long rectangular current (gal-
vanic threshold). Chr is the pulse duration needed to obtain motor 
fiber depolarization with an intensity value twice as large as the 
rheobase. FQ is the ratio between the faradic threshold and the gal-
vanic threshold (slightest intensity necessary to obtain the slightest 
muscle contraction with a 1ms duration rectangular current). Thus 
ESP reflect muscle ability to react to stimulation and are considered 
as good markers of muscle functionnality. Moreover these measure-
ments can be done without any sedation or anesthesia. However poor 
datas are available on dog’s ESP. The aim of our study was to 
evaluate the relevance of ESP measurements in the follow-up and 
prediction of muscle functional recovery in a canine model of den-
ervated muscle compared to the reference tool used (conventional 
electromyography (EMG)). Nine female beagle dogs, 6 months old, 
experienced muscle denervation after section of the fibular nerve. 
Group A (n=3) received an autograft (sectioned fibular nerve su-
tured back to front); Group B (n=4) received a commercial artificial 
nerve conduit and group C (n=2) was the control group. Neurolog-
ic examination, EMG and ESP measurements were performed at 
J0 (day of surgery), J0+1week and then at least every 2 month until 
7,5 months post-surgery. Eight days after surgery, conventional 
electromyography revealed tibialis cranialis denervation and ab-
sence of motor or sensitive conduction for fibular nerve. 

Reinnervation was obvious for all the dogs from A and B groups 
5,5 months post surgery. No recovery was observed for the 2 dogs of 
the control group. One week after surgery ESP were dramatically 
modified for all the dogs. This negative evolution reached its peak 2 months post-surgery and then began to elevate favorably for 
groups A and B dogs. On the 5,5th month, the mean parameters of 
A and B groups were nearly normal and for the C group it wasn’t 
possible to measure any parameter because of the severe muscle fi-
brosis. The results of EMG study and of ESP measurements were 
correlated together and with the clinical evolution (functional re-
covery of the tibialis cranialis assessed by the capacity of the dog to 
flex his tarsal joint, and the increasing in muscle size for group A 
and B, no recovery for group C). This study assess the interest of 
ESP in the follow-up of muscle denervation and reinnervation in the 
dog. Further studies especially on traumatic nerve injuries fre-
quently observed in the dogs are necessary to determine the real 
prognosis value and sensitivity of this method.

**COMPARISON OF SEVERAL HIND LIMB MUSCLES CHRONAXIES IN HUMAN DOG AND HORSE.** Serge G. 
SAWAYA1, Estelle MEALLIER1, Delphine COMBET1, Jean-Jacques 
THIEBAUT1, Guillaume CHANOIT3, David LEVINE3, Denis J. 
MARCELLIN-LITTLE1, 1Unité de Physiothérapie-Rééducation-
Ostéopathie, UPSS 2007-03-135 RT12B ; GREMEREZ.2Unité de 
Pharmacologie-Thérapeutique – Ecole Nationale Vétérinaire de Lyon-
France. 3Department of Physical Therapy, University of Tennessee, 
Chattanooga, TN, USA. 4Department of Clinical Sciences, College of 
Veterinary Medicine, North Carolina State University, Raleigh, NC, 
USA.

Hind limb muscles electrical stimulation (EMS) is often used in 
rehabilitation programs in dogs and horses, to strengthen atrophied 
muscles, enhance active joint stability or treat denervated muscles. 
The optimal stimulation, which is comfortable for the animal and 
produces efficient contractions, depends particularly on the choice 
of pulse duration. The ideal pulse-duration value, sufficient to stimu-
late the motor-neuron without exciting the nociceptive fibers, 
corresponds to the chronaxy of the muscle to stimulate. In this 
study the motor chronaxy of 9 clinically relevant hindlimb muscles 
in dogs and horses were measured, then compared to chronaxies 
values published in human literature. 10 beagles and 11 horses, 
healthy and adult, were enrolled. The tests were realized with a de-
vice equipped with programs for quick chronaxy measurement, and 
density/pulse-duration I=f(t) graph creation. First, the rheobase 
was determined (Rh) as the minimal intensity value necessary to 
obtain the slightest muscular contraction visible or palpable, with 
a long duration monopolar rectangular current (500ms). Secondly, 
the chronaxy was measured as the minimal pulse duration value 
necessary to obtain the same slightest muscular contraction, with 
twice the value of the rheobase (2Rh). For each muscle, the mean 
chronaxy value and its confidence interval in microsecond were cal-
culated (see Table 1).

Hamstrings and distal caudal limb muscles chronaxies are much 
higher in man than in dog and especially than in horse. Compared

| Muscle                          | Dog         | Horse       | Human       |
|---------------------------------|-------------|-------------|-------------|
| Gluteus superficialis           | —           | 152±64 (114–190) µs | 80–160 µs   |
| Gluteus medius                  | 179±69 (136–221) µs | 137±32 (118–156) µs | 80–160 µs   |
| Quadriceps femoris (vastus lateralis) | 233±73 (181–282) µs | 134±37 (110–156) µs | 80–160 µs   |
| Gluteo femoris                  | —           | 105±13 (96–113) µs | —           |
| Biceps femoris                  | 207±63 (167–246) µs | 126±32 (105–145) µs | 440–720 µs  |
| Semi-tendinosus                 | 166±25 (150–181) µs | 102±14 (95–111) µs | 440–720 µs  |
| Tibialis cranialis              | 230±111 (160–314) µs | —           | 200–360 µs  |
| Extensor digitorum longus       | —           | 160±45 (131–187) µs | 200–360 µs  |
| Flexor digitorum pedis lateralis| —           | 199±38 (174–223) µs | 440–720 µs  |
to humans, pelvic limb muscles of dogs and horses have a higher proportion of fast twitch fibers which have shorter chronaxy. We conclude from this project that the optimal pulse duration required for hamstrings and distal limb muscles electromyostimulation is lower in horses compared to calves. By adapting pulse duration for each muscle it makes possible to improve EMS treatment efficiency and comfort, and adapts the rehabilitation protocol for each patient.

MORPHOMETRIC PARAMETERS OF PERIPHERAL NERVES IN CALVES IN CORRELATION TO CONDUCTION VELOCITY. Henning Christian Schenk1,2, Kirsten Haastert1,2, Claudia Grothe2,3, Jürgen Rehage1, Michael Fehr1, Jan Bokemeyer1, Carl Rohn1, Andrea Tipold1,2,3.
1Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany; 2Department of Neuroanatomy, School of Medicine, Hannover, Germany; 3Clinic for Cattle, University of Veterinary Medicine, Hannover, Germany

In the current study the electrophysiological and morphometric reference values of peripheral nerves in calves were determined. The motor nerve conduction velocity (mNCV) of the N. radiialis and the N. ischiadicus/fibularis was measured in 20 healthy calves (age 16 to 85 days). The mean value of the mNCV of the N. radiialis was 48.3 m/s (mean/standard deviation 48.3 ± 10.6 standard deviation (SD), while the mean value of mNCV of the N. ischiadicus/fibularis was 83.8 m/s ± 5.9 SD. Variance analysis revealed that the mNCV of the N. ischiadicus/fibularis is significantly (p < 0.0001) faster than the N. radiialis. Nerve biopsies from a group of six calves (age 14 to 30 days) were taken to correlate the obtained electrophysiological data with morphological parameters. Biopsies were epon embedded and semi-thin transverse sections were stained with 1% paraphenylenediamine solution. Digitized, cross-sectioned slices were manually evaluated with the Analy-SIS ProR software. The fascicle area, the total number of fibres per fascicle, fibre density, fibre area, fibre diameter, axon area, axon diameter, myelin sheath thickness and the g-ratio were obtained in the N. fibularis. The average fibre diameter (mean value 8.40 µm ± 2.80 SD, range 1.98 µm to 17.90 µm) and the average g-ratio (0.61 ± 0.04 SD) of the calves were used for comparison with other species. In conclusion, the established reference values of the mNCV in calves correlate well with the evaluated morphometric parameters. Due to comparably fast mNCV and the high fibre diameters juvenile calves appear to be much more mature individuals than dogs, horses, sheep, cats and humans.

TWO DOGS WITH IATROGENIC DISKOSPONDYLITIS CAUSED BY METICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS (MRSA). M Schwartz1,2, IC Boettcher1, S Kramer2, A Tipold1,2,3.
1School of Veterinary Medicine, Hannover, Germany, 2Center for Systems Neuroscience, Hannover, Germany

Steroid-responsive Meningitis-Arteritis (SRMA) is a systemic inflammatory disease of juvenile to young adult dogs. It shows a relapsing course and most prominently manifests in the cervical meninges. The most characteristic laboratory finding is a marked neutrophilic pleocytosis. Integrin (CD11a, b, c) expression on polymorphonuclear cells (PMNCs) was quantified by immunophenotyping and subsequent flow cytometric measurements. Values were determined for peripheral blood (PB) in the acute phase of SRMA (n = 14) as well as during glucocorticosteroid treatment (n = 16). Results were compared to those from dogs with other neurologic diseases (n = 49) and healthy individuals (n = 7). Integrin expression was also investigated on PMNCs deriving from cerebrospinal fluid (CSF) of dogs in the acute phase of SRMA (n = 14). In a second part of the study PMNCs of healthy dogs were incubated with sera of dogs in the acute phase of SRMA (n = 12). The influence on integrin expression was studied and results were compared to those after incubation with pooled sera of dogs suffering from idiopathic epilepsy (n = 3).

PMNCs in PB of dogs in the acute phase of SRMA exhibited higher values of CD11a expression when compared to dogs under treatment and to control groups, whereas CD11b and c expression was comparable among the different groups. In the acute phase of SRMA CD11b expression on PMNCs in CSF was increased in comparison to that in PB. Incubation with SRMA sera caused a stronger upregulation of CD11a than did pooled epilepsy sera in 9/12 cases whereas an upregulation of CD11b and c was observed in single cases only.

High CD11a expression on PMNCs in PB appears to be an important factor in the pathogenesis of SRMA. This integrin is known to be essential for adhesion of PMNCs within the neutrophilic recruitment cascade and therefore might mediate the enhanced invasion of neutrophils into the subarachnoidal space eventually resulting in meningitis and clinical signs. Since sera of dogs suffering from SRMA selectively induce an upregulation of CD11a it can be suspected that this fluid contains one or multiple factors that are responsible for this.

SELECTIVE RECRUITMENT OR STRONG INTRATHECAL PROLIFERATION OF B CELLS IN DOGS WITH STEROID-RESPONSIVE MENINGITIS-ARTERITIS. M Schwartz1,2, PF Moore1, A Tipold1,2,3, R Carlson1, A Tipold1,2,3.
1School of Veterinary Medicine, Hannover, Germany, 2Center for Systems Neuroscience, Hannover, Germany

Steroid-responsive Meningitis-Arteritis (SRMA) is a systemic inflammatory disease of the juvenile to young adult dog with a relapsing course. Manifestation of SRMA is most prominent in the cervical meninges. In addition to a marked neutrophilic pleocytosis the simultaneous increase of immunoglobulin (Ig) A levels in serum and cerebrospinal fluid (CSF) is an important laboratory finding.
To explain this IgA production, immunophenotyping and flow cytometric measurement of lymphocytes in peripheral blood (PB) and CSF was performed in the acute phase of SRMA (n = 12) as well as during glucocorticosteroid treatment (n = 10). Values were compared to those from dogs with other neurologic diseases (n = 63) and healthy individuals (n = 7).

In dogs with SRMA the CD4/CD8 ratio in PB was shifted towards T helper cells. In addition, low T:B cell ratios were detected in 414 2009 ESVN Abstracts der glucocorticosteroid treatment. The T:B cell ratio in CSF was significantly lower than in PB indicating that either a selective recruitment of B cells or, alternatively, their strong intrathecal proliferation takes place.

Results suggest that a T helper-mediated immune response occurs in SRMA. This defence mechanism typically serves to eliminate extracellular pathogens but is also found in autoimmune diseases. Further more SRMA seems to be a model for the investigation of the compartmentalization of immune responses and for studies on differences in local central nervous system and systemic immune responses.

HYPERTENSIVE ENCEPHALOPATHY IN TWO CATS WITH CHRONIC RENAL FAILURE. N. Soubas1, Z.S. Polizopoulou1, A. Oevermann2, C. K. Koutinas1, A.F. Koutinas1, A. Oevermann2, C. K. Koutinas1, A.F. Koutinas1, 1Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece2NeuroCenter, Department of Clinical Veterinary Medicine, University of Bern, Switzerland.

This report describes hypertensive encephalopathy, a well-known sequela of arterial hypertension in humans, in two aged cats with chronic renal insufficiency. In the first cat (case 1), a 20 year old male Siamese, renal disease has been diagnosed three years ago, was evaluated as compensated and managed only with administration of the appropriate prescription diet. One week prior to hospitalization the cat had exhibited generalized motor seizures in clusters. Upon clinical examination the animal was depressed, demented, tetraplegic and found to have marked arterial hypertension (200–220 mmHg) and bilateral retinal detachment. The second case involved a 5 year old male castrated Persian cat, diagnosed with renal insufficiency six years ago when acute blindness associated with arterial hypertension ensued. Treatment included the administration of amlodipine in combination with dietary modification and had apparently controlled the signs with the exception of blindness that was irreversible. One month prior to admission the cat developed multiple generalized motor seizures along with progressive loss of appetite. On presentation the animal was semicomatose and had an arterial blood pressure of 180-200 mmHg. In both cases initial clinicopathological evaluation revealed anemia, moderate azotemia, but proteinuria was seen only in case 1. Thyroid function tests were normal in both cats, which were also serologically negative for FeLV and FIV infection. The condition of first cat (case 1) continued to deteriorate despite intensive care treatment for one week and finally euthanasia was performed upon the owner’s request. The second cat developed intractable seizures and died during an episode of status epilepticus. Permission for necropsy was granted in both cases.

ENHANCED MONOCYTE FUNCTION IN CANINE DISTEMPER VIRUS INFECTION. V. M. Stein1, N. M. S. Schreiner1, P. F. Moore2, M. Vandevelde3, A. Zürbinger4, A. Tipold5. 1Department of Small Animal Medicine and Surgery, 2University of Veterinary Medicine, Hannover, Germany, 3VM Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California, Davis, USA, 4Institute of Animal Neurology, University of Berne, Switzerland.

Canine distemper virus (CDV) infection is characterized by a multifocal demyelination in the central nervous system (CNS) in the acute immunosuppressive stage of the disease. The pathogenesis of demyelination is unknown. It is thought that the resident macrophages of the CNS, the microglia, as well as invading monocytes may play a crucial role in the demyelinating process.

In an effort to evaluate the role of monocytes their immunophenotype was characterized in dogs during the course of an experimental CDV infection. Twenty SPF-Swiss-Beagle dogs were infected with the virulent CDV strain A75/17. The dogs were regularly examined physically and blood samples were taken every 3–4 days. Monocytes were isolated from peripheral blood by discontinuous density gradient centrifugation and stained against the surface molecules CD11b, CD18, CD1c, B7-1 (CD80), CD44, CD45, MHC I. The murine mAb D110 was used for detection of an epitope of the CDV nucleocapsid protein. Staining against CD3, CD4, CD8a, and CD21 served to exclude contamination with lymphocytes. Measurement was performed by flow cytometry.

According to the clinical signs after remission of initial disease and results of histopathological examination of the CNS dogs were assigned to three examination groups: group I (n = 7) without any clinical signs and no lesions in the CNS, group II (n = 6) with mild clinical signs and no lesions in CNS, group III (n = 7) with severe clinical signs and demyelinating lesions of the CNS. Rectal temperatures were higher in group III than in the other groups, and all dogs of group III showed up to 33.3% CDV-positive monocytes during the course of the infection. The absolute monocyte cell count was not grossly altered in any of the dogs. The percentage of monocytes expressing CD18, CD11b, CD45, CD44, B7-2, and MHC I did not significantly change in the course of the infection. CD14, CD1c, and B7-1 were increasingly expressed by monocytes in all three examination groups. The intensity of expression was enhanced for CD1c, CD11b, B7-2, and MHC I peaking at the end of the third week post infection (p<0.05) followed by a gradual down-regulation. Peak levels of MHC I, CD11b, B7-2, CD1c, and CDV-antibody staining in monocytes were preceded by or concurrent with elevations of body temperature in several animals.

In conclusion, all the molecules examined play an important role in the host’s immune response particularly antigen presentation and cell adhesion. Therefore, an upregulation in certain surface markers reflects enhancement of macrophage functions. As dogs with demyelination showed highest values of CDV-infected monocytes these cells might be crucial in the invasion and spread of the virus in the CNS.

THE DEVELOPMENT OF PART TASK TRAINERS FOR LUMBAR AND CISTERNAL CSF SAMPLING IN THE DOG. M. P. Targett, G.B Cherubini, and L. Mossop. School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, Loughborough, UK.

This study was undertaken to develop part task trainers for lumbar and cerebrospinal fluid sampling in the dog. A part task trainer is a device that permits selected aspects of a task to be practiced independently of other elements of the task. Once developed these models were evaluated for their usefulness in undergraduate teaching as an adjunct to assist in the teaching of spinal anatomy.

Two part task trainers were developed using canine bones with prosthetic materials to model the soft tissue structures. The bones were coated with a series of contact surfaces which allowed a real time readout of the position of the sampling needle when in contact with bones. This readout was displayed on a control box. Evaluation of the usefulness in an undergraduate curriculum was achieved by introducing these models into practical anatomy teaching alongside more classical teaching techniques and cadaver CSF sampling from the cisternal site. Student feedback was obtained from questionnaires at the time of teaching and a subsequent focus group session.

The two part task trainers model needle placement for cisternal and lumbar CSF sampling in the dog. The trainers accurately model the skeletal landmarks for CSF sampling and the sensation of needle placement. The models provide an indication of correct placement of the needle tip against bone the location of the needle when in contact with bone and the correct position of the needle. Student feedback from the use of the trainers was extremely positive. In the student focus group the models were considered to have aided in the assimilation of anatomical knowledge. On the questionnaire 96% of students agreed or strongly agreed that their anatomical knowledge had been improved by the teaching sessions with 90% agreeing or strongly agreeing that the training model enhanced their learning experience.
The new part task trainers fulfilled their design brief and appear to be a useful adjunct to more classical anatomy teaching in the veterinary undergraduate curriculum. The trainers will now be evaluated in a clinical post graduate teaching scenario.

MUSCLE-FORCE PEAKS UNDER MAGNETIC FOUR-WAVE BURST-STIMULATION. D. Thines1, C. Altenhöfer2, A. Fischer2, T. Weyh2, W. Schmidt1, J. Schmidt1, K. Mattasek1, 'Chair of General Pathology & Neuropathology, Institute of Veterinary Pathology, and 2Section of Neurology, Department of Small Animal Medicine, LMU Munich, Germany; 3Heinz Nixdorf Chair of Medical Engineering, TU Munich, Germany; 4Department of Comparative Medicine, Helmholtz Zentrum Munich, Neuherberg, Germany; 5The Animal Health Trust, Newmarket, Suffolk, UK.

Magnetic stimulation (MS) serves as a valuable diagnostic and therapeutic tool for evaluation of the integrity and excitability of the nervous system. MS scarcely evokes pain and therefore is well tolerated by the patients. Compared to commonly used single pulses, burst stimulation achieves a more effective and longer lasting effect on the CNS. Thus, this study, now, was aimed to evaluate the influence of burst and single pulses on limb muscle contraction, induced by magnetic stimulation of peripheral motor-axons.

Measurements were performed in ten healthy anaesthetised beagle dogs. Burst- and single-pulse stimulation of the femoral nerve was applied with a frequency of 7.5 Hz and a stimulator intensity of 60%. Burst stimuli were composed of two to six consecutive sine waves with a frequency of 14 kHz. One single sine wave had a pulse duration of 70 μs. MS was conducted with a figure-of-eight coil. Throughout all stimulations, the optimal coil-position was maintained by a coil-clamp. Muscle strength was measured in force units (FU) through a purpose-built angle-force sensor connected to an USB interface. Isometric quadriceps muscle contractions were obtained through fixation of the knee at 120° joint flexion. Stimulations were discontinued after the muscle contraction force reached a maximum.

In both forms of stimulation a superposition of muscle contractions led to an incline of muscle force. Single pulses, in general, evoked weaker contractions (AFU 0.15) than bursts. Amongst different bursts, an incline of muscle force was observed from two to four consecutive waves with mean FU at 0.82, 1.17 and 1.25 respectively. Instead, five and six consecutive waves resulted in less effective muscle contraction (both 1.06 FU) than four waves. Four consecutive sine waves, furthermore, evoked the fastest isometric contraction (mean 0.52 FU/sec).

In summary, burst-stimulation showed a stronger effect on the evoked muscle contraction force than single pulses. Despite the incline of muscle contraction strength with every wave up to four consecutive sine waves, the force drops when additional waves are applied. Thus, the facilitating effect on neuromuscular coupling, that appears to be triggered by magnetic bursts, reaches its maximum after application of four waves whereas longer stimulation results in fatigue. Hence, pulse types and duration have to be carefully planned when evaluating muscle forces in rehabilitation, or designing an MS-based physiotherapy.

FOREIGN-BODY INDUCED NEURITIS MASQUERING AS A CANINE BRACHIAL PLEXUS TUMOUR. Q. L. Walmsley1 E. Scrucell2, B. A. Summers2, V.A. Penning3, K. Chandler4 and H. A. Volk5, 1Department of Veterinary Clinical Sciences, Royal Veterinary College, London, UK; 2Department of Veterinary Pathology, Royal Veterinary College, London, UK.

A 9-year-old male entire Cocker Spaniel presented with a 3 week history of lethargy, inappetance and progressive right thoracic limb lameness. General physical examination showed a 5/10 thoracic limb lameness. No significant orthopaedic disease was detected. Neurological examination found reduced withdrawal reflex on the right thoracic limb and atrophy of the spinatus musculature. Pain was elicited on palpation and manipulation of the neck and right shoulder. Neuroanatomic localisation was the right brachial plexus. Routine haematology, biochemistry and radiographs of the cervical spine were essentially unremarkable. A magnetic resonance (MR) imaging study of the cervical spine, incorporating images optimised for evaluation of the brachial plexus and associated nerves, revealed no significant abnormalities. Electromyography found spontaneous activity (positive sharp waves and fibrillation potentials) in the right thoracic limb musculature.

One week later clinical signs had progressed to include a right-sided miosis. Surgical exploration of the right brachial plexus was performed via a cranialateral approach. No discrete masses could be found however roots supplying the caudal part of the plexus appeared thickened. Evaluation of biopsies found evidence of Wallerian degeneration. A proximal malignant peripheral nerve sheath tumour (MPNST) was suspected and the owner requested euthanasia.

On post-mortem examination, an irregular, firm, 3×0.7×1 cm, mottled purple-white mass was discovered associated with the right brachial plexus. Histopathological examination revealed abundant granulation tissue admixed with areas of granulomatous inflammation, haemorrhage and fibrosis which surrounded and entrapped multiple nerves. The moderate foreign body-type giant cells often surrounded small fragments of refractile, honecomb-like and Periodic acid-Schiff-positive material consistent with plant material. Within occasional nerves, there was disruption of the
perineurium accompanied by Wallerian degeneration and Schwann cell proliferation.

Foreign body reactions secondary to migrating plant material are not uncommon in dogs and a variety of clinical syndromes have been reported. Vertebral involvement is most commonly restricted to the cranial lumbar region and it is thought that this propensity reflects diaphragmatic migration of inhaled grass awns or penetration of the bowel by swallowed seeds with migration in the mesentery. Involvement of the cervical region is uncommon however extradural compression of the cervical spinal cord following a pharyngeal stick injury and intramedullary granuloma at the level of the first cervical vertebra secondary to a plant foreign body have been reported. To the authors’ knowledge, this is the first case of nerve root signature due to inflammatory disruption of the nerves following migration of foreign plant material within the axillary region.